



Evidence of oxidative stress after continuous exposure to Wi-Fi radiation in rat model

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Abstract

Exposure to electromagnetic radiation (EMR) is rapidly increasing in everyday environment, consequently conferring potential health effects. Oxidative stress is emerging as a mechanism implicated in pathophysiology and progression of various diseases. To our knowledge, no report has been made on the status of antioxidant redox systems after continuous exposure to radiofrequency radiation emitted from a Wi-Fi access point in animal model so far. Therefore, we aimed to continuously subject rats in the experimental group to radiofrequency (RF) radiation emitted from a commercially available Wi-Fi device. Male Wister rats were exposed to 2.45 GHz RF radiation emitted from a Wi-Fi for 24 h/day for 10 consecutive weeks. In order to assess the change in antioxidant redox system of plasma after continuous exposure to a Wi-Fi device, the total antioxidant capacity of plasma, level of thiobarbituric acid reactive substances, concentration of reduced glutathione (GSH), and activity of different enzymatic antioxidants, e.g., superoxide dismutase [SOD], catalase [CAT], glutathione peroxidase [GSH-Px], and glutathione S-transferase [GST], were measured. In the Wi-Fi exposed group, a significant decrease was detected in total antioxidant capacity of plasma and the activities of several antioxidant enzymes, including CAT, GSH-Px, and SOD ($P < 0.05$). Meanwhile, the GST activity was significantly increased in this group ($P < 0.05$). However, no significant changes were found in GSH and TBARS levels following exposure to RF radiation. According to the results, oxidative defense system in rats exposed to Wi-Fi signal was significantly affected compared to the control group. Further studies are needed to better understand the possible biological mechanisms of EMR emitted from Wi-Fi device and relevant outcomes.

Keywords Wi-Fi device · Oxidative stress · Antioxidant capacity

Introduction

Currently, electromagnetic radiation-emitting devices, particularly wireless access points, are widely used in household, industrial, medical, military, and scientific settings. Mutually, these wireless radiation-emitting devices have raised safety concerns regarding the side effects of their radiations on human health (Vignera et al. 2012). Several publications revealed the various

reversible or irreversible impairments caused by exposure to radiofrequency (RF) radiation, including cell cycle arrest, alteration of gene expression pattern, and consequently, induced cell death (Li et al. 2007, Naziroğlu et al. 2013). It is suggested that some of these impairments occur due to disturbance of the balance between the production of reactive oxygen species (ROS) and cellular antioxidant defense system in favor of ROS (Akdag et al. 2007, Repacholi & Greenebaum 1999). According to the literature, increased production of ROS induces oxidative damage of various biomolecules, including carbohydrates, lipids, proteins, and nucleic acids. ROS-induced DNA damage has been proposed as an underlying molecular mechanisms of carcinogenesis in humans (Ongel et al. 2009).

Strictly controlled through the counteracting activity of cellular antioxidants, ROS levels are critical in cellular homeostasis. Enzymatic antioxidants protect body by detoxification or scavenging of excess ROS. In this regard, the most important enzymatic antioxidants are catalase (CAT), glutathione

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reductase (GR), glutathione peroxidase (GSH-Px), superoxide dismutase (SOD), and glutathione S-transferase (GST). On the other hand, vitamin C, vitamin E, and reduced glutathione (GSH) are the major non-enzymatic antioxidants in oxidative defense system, which neutralize the ROS in biological system (Pham-Huy et al. 2008).

Despite numerous studies showing the adverse effects of EMR originated from mobile phones, little data is available on the health hazards of Wi-Fi devices, which also emit electromagnetic radiation (Atasoy et al. 2013, Avendano et al. 2012). It might be hypothesized that radiation-emitting Wi-Fi devices may be able to induce harmful changes in various organs. The involvement of oxidative stress in metabolic disorders due to exposure to static magnetic field has been previously shown in animal model (Elferchichi et al. 2010, Ghodbane et al. 2011). However, previous studies on the health-threatening effects of RF-EMR have reported contradictory results. To date, the biological effects of Wi-Fi signal and its mechanism of action remain unclear. Some studies on experimental animals have shown that exposure to EMR from mobile phones could induce overproduction of ROS, which leads to the depletion of tissue antioxidants (Celik et al. 2016, Özorak et al. 2013, Salah et al. 2013). Nevertheless, others have suggested that EMR had no significant effect on antioxidant defense system due to unchanged oxidative stress markers (e.g., MDA) (Atasoy et al. 2013). Researchers have proposed that the antioxidant status and damage level depend on the levels of ROS produced after exposure to EMR (Brydon et al. 2001). With this background in mind, this study aimed to determine whether continuous exposure to Wi-Fi signal can cause oxidative stress in animal model.

Materials and methods

Chemicals

In the present research, analytical grade chemicals and organic solvents were used and purchased from Merck (Germany) and Sigma–Aldrich (St. Louis, MO, USA) corporations. All solutions, with the exception of sodium or potassium phosphate buffers, were prepared and measured daily.

Animals

In total, 20 male rats were randomly selected and divided equally into two non-exposed (control) and exposed (experimental) groups (ten samples per group). All animals were maintained in clean polypropylene cages during 14/10 h light/dark cycles and acclimated to the pathogen-free laboratory conditions 3 days before experiments. The samples with initial body weight of 160 ± 10 g and age of 3 months were included in the study. At the end of the research, the body weight of the rats was 248 ± 28.8 g.

Exposure setup

After a short adaptation period, samples of the experimental group were continuously exposed to EMR originated from a Wi-Fi device. In this study, one ADSL2+ wireless gateway with dipole antenna was applied as a signal generator. Multiple functions were supported by router for the wireless interface. The schematic design of the device and the position of rats used for radiation exposure in this study are shown in Fig. 1. The Wi-Fi device was placed on the platform and kept 30 cm far away from the cages of subjects. The exposure system was designed in a way as to apply equal condition to all ten animals at the same time during the experiment. It is notable that continuous exposure to Wi-Fi signal was performed 24 h/day for 10 consecutive weeks. On the other hand, rats in the control group were kept under identical environmental conditions with no exposure to EMR. There was no movement restriction for rats in cages during the study. At the end of the 10 weeks, blood samples were drawn from the inferior vena of all animals after anesthesia. The samples were collected in tubes containing ethylene diaminetetraacetic acid (EDTA) and were centrifuged at 3500g for 10 min to obtain plasma.

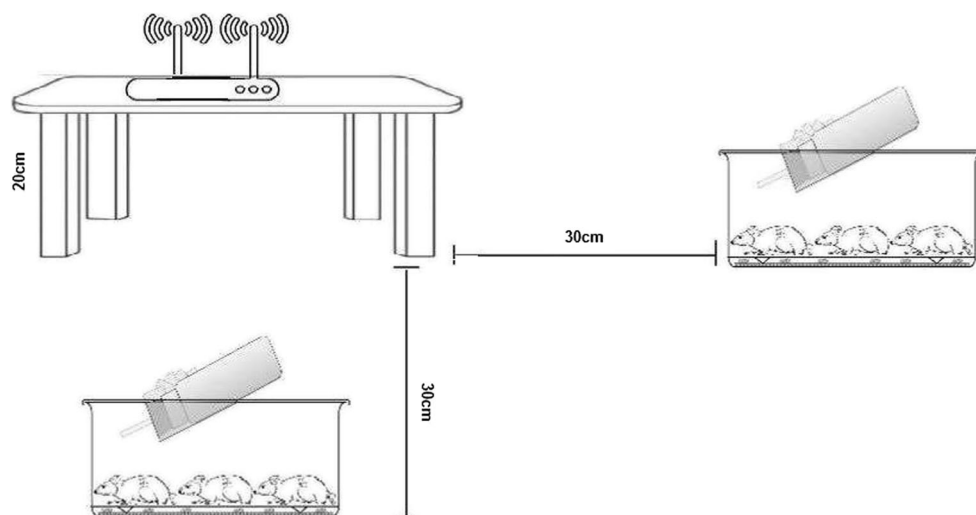
Measurement of total antioxidant capacity

Total antioxidant capacity of plasma was estimated by the Ferric Reducing Ability of Plasma (FRAP) assay according to the method described by Benzie and Strain (1996). The working solution was freshly made by adding acetate buffer (300 mM, pH 3.6), 2,4,6-tris(2pyridyl)-s-triazine (40 mM dissolved in 40 mM HCl), and ferric chloride (20 mM in water) in a 10:1:1 ratio. The FRAP working solution was prepared daily and warmed at 37 °C for 10 min before use. In addition, serial dilution of ferrous sulfate (2000 μ M) prepared in deionized water was used as standard. Briefly, 50 μ l of the sample, water blank, or standards was added to 1000 μ l freshly prepared FRAP reagent and incubated for 10 min at 37 °C. Finally, absorbance of mixtures was measured at 593 nm against the blank. Moreover, the antioxidant capacity of plasma was calculated using a calibration curve of ferrous sulfate (100–2000 μ M).

Measurement of thiobarbituric acid reactive substances

As an end product of lipid peroxidation, plasma thiobarbituric acid reactive substances (TBARS) were measured by the method described by Buege and Aust (1978). Briefly, 50 μ l of the plasma was mixed with 1950 μ l thiobarbituric acid reagent (0.375% w/v thiobarbituric acid, 15% w/v trichloroacetic acid, and 0.25 N hydrochloric acid). Following that, the solution was incubated at 100 °C in a water bath for 15 min. In

Fig. 1 The experimental setup for irradiation of rats to a Wi-Fi device



the next stage, the incubated mixture was cooled and centrifuged at $2000\times g$ for 10 min, and the absorbance of the supernatant of each sample was measured at 535 nm against blank using spectrophotometer. TBARS level of each sample was estimated based on the molar extinction coefficient ($\varepsilon = 156 \text{ mM}^{-1} \text{ cm}^{-1}$) of product, expressed as nanomoles per milliliter in plasma.

Measurement of catalase

The activity of catalase was assayed based on the method of Aebi (1984) using H_2O_2 as a substrate. In brief, 50 μl of the plasma was added to reaction mixture of potassium phosphate buffer (200 mM, pH 7), which contained 10 mM H_2O_2 as substrate. Absorbance change of samples was read at 240 nm every 30 s for 2 min. Furthermore, catalase activity was calculated using molar extinction coefficient of H_2O_2 at $39.4 \text{ M}^{-1} \text{ cm}^{-1}$. Accordingly, decomposition of 1 μmole of H_2O_2 per minute at pH 7.0 was considered one unit of catalase.

Measurement of GSH-Px activity

At this stage of the research, the Wendel method was used to analyze glutathione peroxidase activity based on the rate of *t*-butyl hydroperoxide decomposition (Wendel 1981). Basically, the oxidation rate of the reduced glutathione was coupled to reduced NADPH concentration after adding glutathione reductase to the reaction mixture, as demonstrated by decrease in absorbance at 340 nm. Briefly, 100 μl plasma was added to reaction mixture, including 2.7 ml of PB buffer (50 mM, pH 7.0 containing 15 mM EDTA, and 3 mM GSH), 100 μl NaN_3 (30 mM), 100 μl NADPH (6 mM), and 5 μl glutathione reductase (1 unit). After adding 10 μl of *t*-butyl hydroperoxide, the initiation of enzymatic reaction was observed by decrease in absorbance at 340 nm, which was

recorded for 3 min at 30 s. Glutathione peroxidase activity was calculated from absorbance changes using molar extinction coefficient of $6.22 \times 10^6 \text{ M}^{-1} \text{ cm}^{-1}$ for NADPH. One unit of glutathione peroxidase was expressed as nanomoles of NADPH oxidized per minute.

Measurement of reduced glutathione level

Reduced glutathione content was measured spectrophotometrically using 5,5'-dithiobis (2-nitrobenzoic acid), known as Ellman's reagent, which reacts with reduced glutathione to yield a reduced yellow chromophore (Ellman 1959). Concisely, 100 μl plasma was mixed with 100 μl of 10% trichloroacetic acid and was centrifuged at $12000\times g$ for 3 min at 4 °C. Afterwards, the supernatant was separated and used to calculate reduced glutathione. To this end, 0.1 ml of the supernatant was added to 2 ml of phosphate buffer (pH 7.5) containing 6 mM of Ellman's reagent. The absorbance of yellow product was recorded at 412 nm using a spectrophotometer. Moreover, the standard curve was constructed by pure reduced glutathione with different concentrations, used to estimate the concentration of reduced glutathione in all samples.

Measurement of SOD activity

Using the riboflavin-methionine method of Giannopolitis and Ries, assay of superoxide dismutase was carried out spectrophotometrically (Giannopolitis & Ries 1977). In brief, reaction mixture contained 50 μl of plasma and 2.95 ml of phosphate buffer pH 7.8 (10 mM) containing EDTA (0.01 M), methionine (13 mM), riboflavin (20 μM), and nitro blue tetrazolium chloride (75 μM). Reaction mixture was kept under two 20-W fluorescent lamps, which blue color formazan developed upon reduction of nitro blue tetrazolium. Following that, the absorbance of reaction mixture was measured at 560 nm against the

reagent blank. One unit of the SOD was the amount that was required to inhibit reduction of NBT by 50% per minute.

Measurement of glutathione S-transferase

In this research, the glutathione S-transferase activity was measured based on the protocol presented by William H. Habig using GSH and 1-chloro-2,4-dinitrobenzene (CDNB) as substrates (Habig et al. 1974). Basically, glutathione was transferred by GST to 1-chloro-2,4-dinitrobenzene, which resulted in the formation of glutathione 2,4-dinitrobenzene conjugate measured by spectrometrically at 340 nm. Briefly, reaction mixture was composed of 50 μ l of plasma and 2.9 ml of potassium phosphate buffer pH 6.5 (100 mM), 1 mM glutathione, and 1 mM CDNB. Formation of glutathione 2,4-dinitrobenzene conjugate was measured by changes in absorbance of reaction mixture at 340 nm for 3 min.

Statistical analyses

All data were expressed as means $\pm$ standard deviation (SD), and results were analyzed using the Statistical Package for the Social Sciences software (SPSS) statistical program (version 16.0). In addition, differences of each parameter among two study groups were analyzed by Student's *t* test.

Results

In this research, the Wi-Fi signal significantly decreased the FRAP value, as the index of total antioxidant capacity (949.21 ± 89.00 vs. 1142.2 ± 119.82 μ M, $P = 0.001$; Fig. 2a). However, exposure to radiation caused no significant difference between the study groups in terms of the MDA level, as an index of lipid peroxidation (16.33 ± 1.85 vs. 17.53 ± 1.67 nM, $P > 0.05$; Fig. 2b). The plasma catalase in the

experimental group was significantly reduced, compared to the control group (28.50 ± 4.23 vs. 17.77 ± 2.39 U/l, $P < 0.001$; Fig. 3). Similarly, RBC catalase activity significantly decreased, compared to the control group (79.37 ± 14.57 vs. 50.41 ± 11.24 U/gr Hb, $P < 0.001$; Fig. 3).

The activity of SOD in the control and experimental groups was found at 120.97 ± 13.79 and 90.45 ± 9.17 U/gr Hb, respectively (Fig. 3). According to the results of the present study, the SOD activity in the experimental group significantly decreased compared to the control group ($P < 0.001$). The activity of GSH-Px in the research groups was found to be 55.09 ± 3.15 U/ml and 50.61 ± 2.32 U/ml, respectively (Fig. 4). Statistical analysis showed a significant reduction in the activity of GSH-Px in the experimental group compared to the control group ($P < 0.01$).

The mean GST activity in the control group and the rats exposed to Wi-Fi signal is shown in Fig. 4. In this regard, a significant increase was detected in the activity of GST in rats exposed to EMR, compared to the control group (51.98 ± 20.07 vs. 83.73 ± 15.26 U/l, $P = 0.001$). While there was an increase in the GST activity of the samples in the experimental group, no significant differences were found between the research groups regarding the reduced glutathione concentration (9.33 ± 4.22 vs. 10.67 ± 3.67 μ M, $P > 0.5$; Fig. 4).

Discussion

Long-term exposure to EMR is rapidly increasing day by day in all human habitats. While numerous harmful biological effects were detected after exposure to EMR, potential hazards of wireless devices on the emergence of oxidative damage are still not fully covered. Therefore, the blood oxidative stress markers and antioxidants were evaluated in the current research to confirm the oxidative damages caused by RF radiation emitted from commercial wireless devices. According to

Fig. 2 FRAP value (a) and MDA levels (b) in the experimental and control groups. *Significantly different compared to the control group

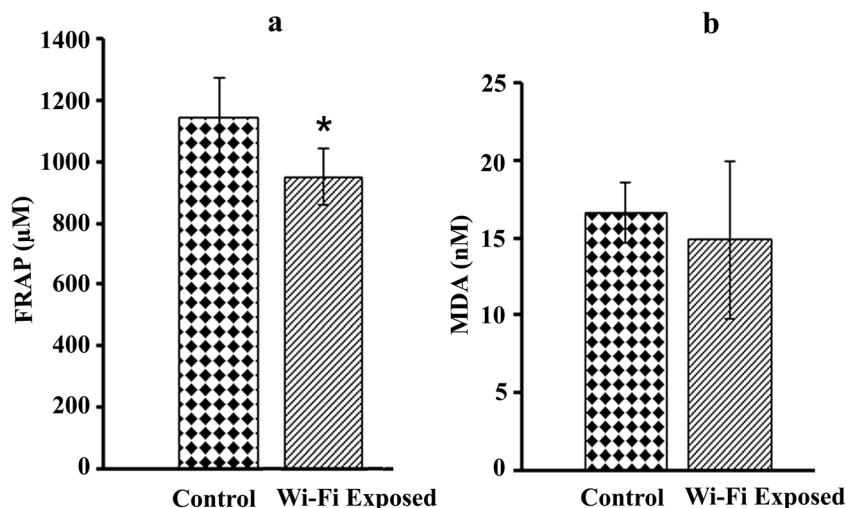
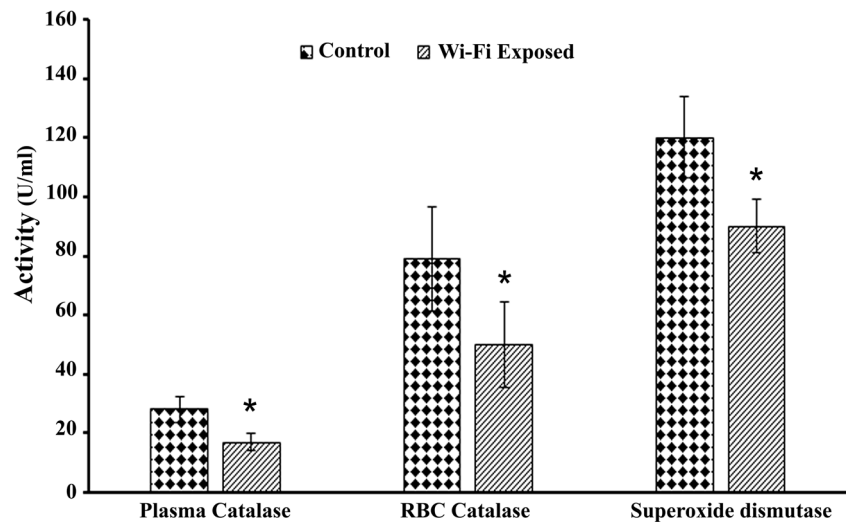


Fig. 3 Plasma catalase, RBC catalase, and superoxide dismutase activities and reduced glutathione levels in the experimental and control groups
*Significantly different compared to the control group



the results of the present study, continuous exposure to Wi-Fi signal was able to induce oxidative stress. In addition, this type of exposure resulted in the depletion of total antioxidant ($P < 0.05$). In the current study, there was a significant decrease in the activity of catalase in both plasma and RBC of rats exposed to EMR, compared to the control group. Rats exposed to radiation showed a significant decreased in the activity of glutathione peroxidase. Furthermore, the activity of SOD significantly decrease in rats continuously exposed to RF-EMR emitted from a wireless device. In our study, there was no change in the concentration of GSH in the experimental group, whereas a significant increase was detected in the activity of glutathione S-transferase in the same group.

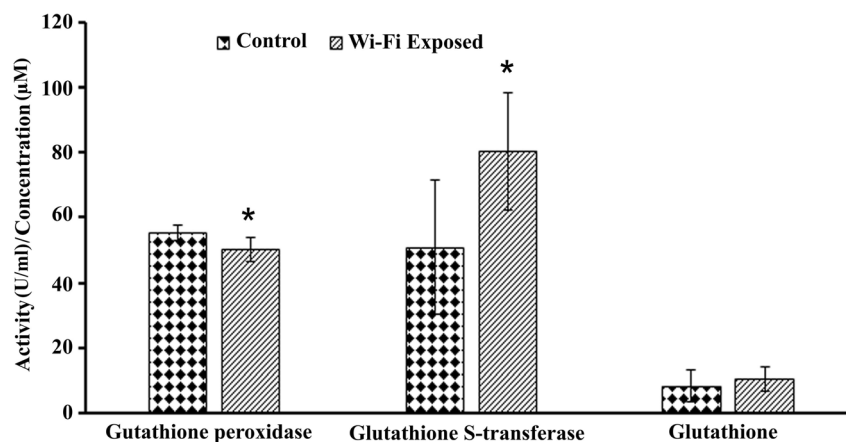
The potential harmful effects of such radiations depend on the duration of exposure, radiation dose, body posture, and age (Dimbylow & Findlay 2010). Cytogenetic damage induced by RF-EMR at 2.45 GHz in human peripheral blood lymphocytes was investigated by Zotti-Martelli et al., who reported that long-term exposure to radiation caused cytogenetic damage to human blood lymphocytes (Zotti-Martelli et al. 2000). Moreover, Ozlem Sangun et al. reported the

adverse effect of long-term exposure to RF-EMR emitted from wireless internet devices on the growth and puberty in animal model (Sangun et al. 2015).

Recent reports have indicated that EMR originated from Wi-Fi devices and mobile phones can cause oxidative stress due to the overproduction of oxygen-derived free radicals (Naziroğlu et al. 2013). In turn, it was hypothesized in the present study that increased generation of ROS due to long-term or continuous exposure to such radiation also caused depletion of major component of antioxidant defense system.

As such, decreased level of plasma total antioxidant may be indicative of overproduction of ROS under continuous exposure to EMR originated from Wi-Fi devices, which is in line with the results obtained by Saygin et al. indicating the emergence of testicular inflammatory biomarkers after exposure to RF-EMR at 2.45 GHz in young rats (Saygin et al. 2015). According to the mentioned researchers, the total antioxidant status of the testes tissue significantly decreased in the EMR-exposed group. They also found an increase in the level of TBARS in the testes tissue. Furthermore, Gumral et al. (2009) found similar results on the lipid peroxides status in rats after

Fig. 4 Glutathione peroxidase and glutathione S-transferase activities and reduced glutathione levels of the subjects in the experimental and control groups
*Significantly different compared to the control group



exposure to RF-EMR at 2.45 GHz (Gumral et al. 2009). However, there was no significant difference between the research groups in terms of plasma level of TBARS. Contradictory results on TBARS level in the current study might be due to methodological differences in the periods of exposure to Wi-Fi signal, compared to other studies. Whole body exposure to wireless devices and continuous exposure were applied in this experiment, which was methodologically different from that of the previous studies. In this respect, our findings are in congruence with the report of other studies, which indicated no significant differences in the TBARS level in plasma of rats exposed to RF-EMR (Gürler et al. 2014, Yuksel et al. 2016). Given the lack of difference between the groups regarding the TBARS levels, it was proposed that other biomarkers of lipid peroxidation (e.g., isoprostanes or HNE) may contribute to the pathophysiology of continuous exposure to EMR.

This experiment is unique in the sense that the oxidative status of individuals continuously exposed to RF-EMR from conventional wireless device was investigated. Atasoy et al. (2013) evaluated the harmful effects of EMR originated from conventional wireless devices on the reproductive system of growing rats. These researchers applied a continuous RF-EMR at 2.45 GHz for 20 weeks. According to their results, no significant difference was found in lipid peroxidation, whereas a significant decrement was reported in the catalase and GSH-Px activities of rats' testes. Since hydrogen peroxide in its higher concentration is neutralized by catalase, the hazardous effects of this reactive oxidant on body could be prevented by catalase. Atasoy et al. marked that rats exposed to Wi-Fi signal experienced a significant decrease in CAT activity of testes (Atasoy et al. 2013). The significant decrease in CAT activity, an important enzyme in the cellular defense mechanism, may be observed at excess superoxide radical and hydrogen peroxide production (Kono & Fridovich 1982, Pigeolet et al. 1990). In a previous study, Gumral et al. (2016) exposed female Wistar Albino rats to 2450 MHz wireless devices and evaluated their blood antioxidant status. According to the results of the mentioned research, no significant difference was observed in lipid peroxidation, glutathione level, and GSH-Px activity of samples.

Both glutathione peroxidase and catalase are well distributed in plasma and red blood cells (Sureta et al. 2005). Therefore, lower activity of glutathione peroxidase and catalase may be indicative of oxidative stress occurrence. This decreased catalase and GSH-Px activities caused by elevated level of free radicals would possibly cause oxidative damage to macromolecules.

In addition, the decreased SOD activity may occur either via direct oxidative damage of the SOD or by altered pattern of SOD gene expression by oxidative stress, or both (Klaunig et al. 2010, Scandalios 2005). However, decreased SOD activity after continuous exposure to Wi-Fi signals may

subsequently lead to the generation and accumulation of superoxide anion. In this respect, our findings are consistent with the results obtained by Salah et al. (2013), who reported a significant decrease in catalase, glutathione peroxidase, and superoxide dismutase activities. In the mentioned research, subchronic exposure to RF-EMR at 2.45 GHz from a Wi-Fi device affected the oxidant–antioxidant balance in rat tissues. Effects of Wi-Fi frequencies on the various antioxidants of newborn rat's kidney and testis were assessed by Özorak et al. (2013), who reported no change in the GSH level in testes but a significant decrease significantly in kidney after 6 weeks of exposure of newborn rats to Wi-Fi signals at 2.45 GHz.

In addition to a role in xenobiotic detoxification, glutathione S-transferase plays a role in the removal of endogenous hydroperoxides produced in body through oxidative stress-induced lipoperoxidation processes (Danielson & Mannervik 1985, Hayes & McLELLAN 1999). This is in accordance with our findings, which demonstrated that increased GST activity in the experimental group led to no significant changes in MDA level. The result of an in vitro study showed the sensitivity of GST to hydrogen peroxide treatment (Neeffjes et al. 1999). Other studies have documented that GST inhibition in some conditions is associated with oxidative stress in tissues (Goel et al. 2005, Mansour & Mossa 2009). On the other hand, it was proposed that induction of GST gene expression caused resisting of cells against hydrogen peroxide-induced apoptosis, especially in liver (Yang et al. 2001). In the present study, it was proposed that increased activity of GST was an adaptive response to oxidative stress, which is important in the removal of the lipid peroxides and protection against cellular damages.

Overall, our experimental data clearly indicated that exposure to Wi-Fi radiation led to the generation of ROS, which suggests the possible effect of Wi-Fi signals on induced oxidative stress as a consequence of elevated ROS after continuous exposure. In this regard, the depletion of antioxidants or alterations in gene expression of various antioxidants and/or inhibition of enzymatic antioxidant activity may be responsible for persistence of oxidative stress under continuous exposure to EMR emitted from wireless devices. However, further experiments are required to demonstrate the underlying mechanism of EMR with different frequencies on various tissues.

Conclusion

According to the results of the current research, long-term exposure to EMR emitted from wireless devices had adverse effects on the antioxidant potential of blood. Therefore, to protect individuals from harmful effects of Wi-Fi signals, it is advised to limit the use of such devices for household and occupational activities, if possible. This study may stimulate future helpful research in the development of new protective

or therapeutic approaches. In addition, it is recommended that the target tissues of EMR emitted from wireless devices and the level of other mediators be investigated to understand the exact molecular mechanism and site of action upon continuous exposure to such radiations.

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