

# Effects of acute exposure to the radiofrequency fields of cellular phones on plasma lipid peroxide and antioxidant activities in human erythrocytes

Yasser M. Moustafa <sup>a</sup>, Randa M. Moustafa <sup>c</sup>, A. Belacy <sup>c</sup>,  
Soad H. Abou-El-Ela <sup>b,\*</sup>, Fadel M. Ali <sup>d</sup>

<sup>a</sup> Department of Pharmacology and Toxicology, Faculty of Pharmacy, Suez Canal University, Ismailia 41522, Egypt

<sup>b</sup> Department of Biochemistry, Faculty of Pharmacy, Suez Canal University, Ismailia 41522, Egypt

<sup>c</sup> Department of Physiology, Benha Faculty of Medicine, Zagazig University, Benha, Egypt

<sup>d</sup> Department of Biophysics, Faculty of Science, Cairo University, Cairo, Egypt

Received 18 November 2000; received in revised form 8 March 2001; accepted 11 March 2001

## Abstract

Radiofrequency fields of cellular phones may affect biological systems by increasing free radicals, which appear mainly to enhance lipid peroxidation, and by changing the antioxidant activities of human blood thus leading to oxidative stress. To test this, we have investigated the effect of acute exposure to radiofrequency fields of commercially available cellular phones on some parameters indicative of oxidative stress in 12 healthy adult male volunteers. Each volunteer put the phone in his pocket in standby position with the keypad facing the body. The parameters measured were lipid peroxide and the activities of superoxide dismutase (SOD), total glutathione peroxidase (GSH-Px) and catalase. The results obtained showed that the plasma level of lipid peroxide was significantly increased after 1, 2 and 4 h of exposure to radiofrequency fields of the cellular phone in standby position. Moreover, the activities of SOD and GSH-Px in human erythrocytes showed significant reduction while the activity of catalase in human erythrocytes did not decrease significantly. These results indicate that acute exposure to radiofrequency fields of commercially available cellular phones may modulate the oxidative stress of free radicals by enhancing lipid peroxidation and reducing the activation of SOD and GSH-Px, which are free radical scavengers. Therefore, these results support the interaction of radiofrequency fields of cellular phones with biological systems. © 2001 Elsevier Science B.V. All rights reserved.

**Keywords:** Radiofrequency fields; Free radical; Cellular phone; Superoxide dismutase; Glutathione peroxidase; Catalase; Lipid peroxide; Oxidative stress; Antioxidases

## 1. Introduction

Oxidative stress is a cellular or physiological condition of elevated concentrations of reactive

\* Corresponding author. Tel.: +20-12-2571203; fax: +20-64-355741.

E-mail address: soad@workmail.com (S.H. Abou-El-Ela).

oxygen species that cause molecular damage to vital structures and functions [1]. Several factors influence the susceptibility to oxidative stress by affecting the antioxidant status or free oxygen radical generation. These factors can be divided into those of endogenous, e.g. exercise and psychological stress or of exogenous origin, e.g. food, alcohol, cigarette smoke, environmental pollutants and radiation [1].

Recently, people have become especially concerned about the safety of wireless communication, e.g. cellular phones that are rapidly gaining popularity and yet little is known about the health hazards of the radiofrequency fields of these cellular phones. There are several cellular phone systems in use, which allow many users to communicate via the system simultaneously. The dominant access technique in Egypt is the so-called time division multiple access (TDMA), which is used in global system for mobile communication system (GSM). The carrier frequency bands allocated for this service is set mainly in the spectrum regions of 800–900 MHz [2].

The question of whether the radiofrequency fields of cellular phones affect oxidative stress and the antioxidase activities are of considerable interest to biomagnetics and biochemistry. Thus, the present study was designed to evaluate the effect of acute exposure to radiofrequency fields of commercially available cellular phones on some parameters indicative of oxidative stress (lipid peroxide, superoxide dismutase (SOD), glutathione peroxidase (GSH-Px) and catalase) in human blood.

## 2. Materials and methods

The study was conducted on 12 adult male volunteers (aged 20–25 years) and weighed  $71.33 \text{ kg} \pm 2.013$  (S.D.). They were non-smokers, normotensive, took no medication and were of the same socioeconomic status. Each volunteer placed the cellular phone (Ericsson GH 688, Ericsson Mobile Communication, AB, Sweden) in his pocket in standby position, emitting a 900 MHz radiofrequency field, with the keypad of the phone facing the body. After exposure to the

cellular phones for 1, 2 and 4 h, blood was collected on EDTA to obtain plasma and erythrocytes according to the method described by Parogzai et al. [3]. Blood was also collected from the volunteers before their exposure to the cellular phones as a control (zero time of exposure). Plasma was kept in  $-80^\circ\text{C}$  until analyzed for lipid peroxide using the method of Yagi [4] based on the formation of malondialdehyde (MDA) and measured by the thiobarbituric acid method. Erythrocytes were washed with isotonic solution of NaCl and centrifuged at 3000 rpm for 10 min where erythrocyte pellets were separated and resuspended in cold distilled water to obtain the erythrocyte lysates, which were used immediately for the enzymatic assays. SOD activity was determined using the xanthine and xanthine oxidase method as described by Nebot et al. [5].

Total GSH-Px was determined using reduced glutathione and cumene hydroperoxide as ascertained by Barja de Quiroga et al. [6]. Catalase activity was measured using hydrogen peroxide as a substrate according to the method of Aebi [7]. Protein concentration was measured as described by Lowry et al. [8].

The statistical significance of the results was calculated using Student's *t*-test.

## 3. Results

Table 1 shows that acute exposure to 900 MHz radiofrequency fields of cellular phones for 1, 2 and 4 h significantly increased the human plasma level of lipid peroxide. On the other hand, human erythrocyte SOD activity was significantly decreased after 1 and 4 h of exposure to radiofrequency fields of cellular phones whereas an insignificant decrease was observed after 2 h of exposure. GSH-Px activity in human erythrocytes was significantly decreased after 1 and 2 h of exposure but did not decrease significantly after 4 h of exposure. Human erythrocyte catalase activity did not change significantly after acute exposure to radiation of cellular phones.

#### 4. Discussion

Free radicals are very reactive and unstable molecular species that can initiate chain reactions to form new free radicals. Although formed as a result of a wide range of normal biochemical processes, they are potentially damaging. Several mechanisms are in place to neutralize their effects, which include a system of nutritional and endogenous enzymatic antioxidant defenses that generally hold the production of free radicals and prevent oxidant stress and subsequently tissue damage [9]. The balance between the oxidants and the antioxidants may be disturbed by an increase in free radical production or by a reduction in antioxidants [10]. This imbalance between the oxidants and the antioxidants can lead to 'oxidative stress' i.e. a series of peculiar and potentially damaging biochemical reactions [11]. Particularly susceptible to oxidative damage by free radicals are the polyunsaturated fatty acid acyl chains of phospholipids, which lead to lipid peroxidation. Uncontrolled lipid peroxidation is a toxic process resulting in the deterioration of biological membranes [1,11]. Lipid peroxidation products e.g. malondialdehyde has been taken as a biomarker to oxidative stress in biological system [12]. The antioxidant enzymes include, SOD, which removes superoxide anion, as well as catalase and GSH-Px that scavenge hydrogen peroxide and prevent accumulation of the hydroxyl radical.

Radiofrequency waves are a very important part of electromagnetic spectrum with respect to their applications and possible health consequences. Epidemiological studies remain inconclusive with regard to the health effects of prolonged exposure to electromagnetic fields [13–26]. Wire-

less communication especially cellular phones are rapidly gaining popularity knowing little about the effect of the radiofrequency fields of these phones on human health. The present study showed that acute exposure for 1, 2 and 4 h to 900 MHz radiofrequency fields of cellular phones increased the plasma level of lipid peroxidation and decreased the activities of SOD and GSH-Px in human erythrocytes. Both enzymes are important in scavenging the superoxide anion and hydrogen peroxide, respectively, and prevent accumulation of the hydroxyl radical as well as lipid peroxidation. The fluctuating activity of SOD and GSH-Px and the insignificant decrease in the activity of catalase in addition to the consistent increases in the level of lipid peroxides after all the times of exposure may be attributed to disruption in the antioxidant mechanisms that neutralize free radicals [9,10]. Erythrocytes are particularly susceptible to oxidative stress because they encounter higher oxygen tension than any other cells in the body, with the exception of lung cells [27]. Enzymes and proteins in mature erythrocytes are potential targets for oxidative damage and that inevitably results in loss of cell function.

Earlier studies [28–31] on the effects of electromagnetic fields on free radicals and antioxidants utilized frequencies, intensities and times of exposure different from those concerned with the electromagnetic field of the cellular phone. In addition, their experiments involved in vitro and animal studies and did not involve humans. Zheng et al. [28] reported that exposure to 50 MHz fields for 3 h per day and continued for 30 days significantly increased the lipid peroxidation in brain tissue of mice but did not change the

Table 1

Effects of acute exposure to radiofrequency fields of cellular phones on parameters indicative of oxidative stress in human blood

Parameters	Control (0 time)	1 h	2 h	4 h
Plasma lipid peroxides (nmol/ml)	2.087 ± 0.058	2.262 ± 0.053 <sup>a</sup>	2.279 ± 0.058 <sup>a</sup>	2.320 ± 0.049*
Erythrocyte SOD activity (U/mg protein)	61.26 ± 0.697	56.84 ± 1.080 <sup>a</sup>	59.36 ± 0.900	57.04 ± 1.056 *
Erythrocyte GSH-Px activity (U/mg protein)	1.869 ± 0.023	1.698 ± 0.037 <sup>a</sup>	1.712 ± 0.019 <sup>a</sup>	1.796 ± 0.038
Erythrocyte catalase activity (U/mg protein)	40.69 ± 0.728	39.12 ± 0.903	39.73 ± 0.701	39.53 ± 1.042

Values are mean ± S.E., *n* = 12.

<sup>a</sup> Significantly different from control using Student *t*-test (*P* < 0.05–0.0005).

activities of SOD and GSH-Px. Moreover, the in vitro study of Iwsaka and Ueno [29] showed no effect of magnetic fields on the reaction of SOD, peroxidase and xanthine oxidase using a spectrophotometric system with an external optical cell room in a super conducting magnet. However, the study showed an effect on the reaction of catalase with 50 mM of hydrogen peroxide without affecting the catalytic activity of the enzymes itself. Other studies [30,31] reported that in vitro exposure to magnetic fields generated free radical species such as superoxide anion and hydrogen peroxide that could affect the biological system. These studies did not report the effect of magnetic field exposure on the free radical scavengers.

## 5. Conclusion

The data presented here suggested that radiofrequency fields of cellular phones generated free radicals that increased peroxidation in human plasma and decreased the activities of the antioxidant system, SOD and GSH-Px in humans erythrocytes. Whenever the balance of antioxidants is outweighed by prooxidizing factors as shown by the radiofrequency of cellular phones, oxidative stress may develop in cells. Therefore, these results support the interaction of radiofrequency fields of the cellular phones with biological systems.

## References

- [1] P. Moller, H. Wallin, L.E. Knudsen, *Chemico-Biol. Interact.* 102 (1996) 17–36.
- [2] L. Verschaeve, A. Maes, *Mutat. Res.* 410 (1998) 141–165.
- [3] M. Parogzai, E. Roth, G. Matos, G. Temes, J. Lantos, E. Karpati, *Pharmacol. Res.* 33 (1996) 327–336.
- [4] K. Yagi, *Methods Enzymol.* 105 (1984) 328–331.
- [5] C. Nebot, M. Moutet, P. Huet, J.Z. Xu, J.C. Yadan, J. Chaudiene, *Anal. Biochem.* 214 (1993) 442–451.
- [6] G. Barja de Quiroga, P. Gil, M. Lopez-Torres, *J. Comp. Physiol.* B158 (1988) 583–590.
- [7] H. Aebi, *Methods Enzymol.* 105 (1984) 121–126.
- [8] O.H. Lowry, N.J. Roenbrough, A.L. Farr, R.J. Randall, *J. Biol. Chem.* 196 (1951) 265–275.
- [9] B. Hallwell, *Lancet* 344 (1994) 721–724.
- [10] B.M. Winkhofer-Roob, *Acta Paediatr. (Suppl.)* 395 (1994) 49–57.
- [11] A. Pompella, *Intl. J. Vit. Nutr. Res.* 67 (1997) 289–297.
- [12] J. Laval, *Pathol. Biol.* 44 (1996) 14–24.
- [13] D.A. Savitz, J.E.M. Kleckner, *Am. J. Epidemiol.* 131 (5) (1990) 763–773.
- [14] E.P. Washburn, M.J. Orza, J.A. Berlin, W.J. Nicholson, A.C. Todd, H. Frumkin, T.C. Chalmers, *Cancer Causes Control* 5 (4) (1994) 299–309.
- [15] S. Preston-Martin, W. Navidi, D. Thomas, P.J. Lee, J. Bowman, J. Pogoda, *Am. J. Epidemiol.* 143 (2) (1996) 105–119.
- [16] J.G. Gurney, B.A. Mueller, S. Davis, S.M. Schwartz, R.G. Stevens, K.J. Kopecky, *Am. J. Epidemiol.* 143 (2) (1996) 120–128.
- [17] M. Coleman, V. Beral, *Int. J. Epidemiol.* 17 (1) (1988) 1–13.
- [18] B.M. Matanoski, E.A. Elliot, P.N. Breyse, M.C. Lynberg, *Am. J. Epidemiol.* 137 (6) (1993) 609–619.
- [19] G. Theriault, M. Goldberg, A.B. Miller, B. Armstrong, P. Guenel, J. Deadman, E. Imbernon, T. To, A. Chevalier, D. Cyr, *Am. J. Epidemiol.* 139 (10) (1994) 1053.
- [20] D.A. Savitz, D.P. Loomis, *Am. J. Epidemiol.* 144 (2) (1996) 205.
- [21] B. Floderus, S. Tornqvist, C. Stenlund, *Cancer Causes Control* 5 (2) (1994) 189–194.
- [22] S.J. London, D.C. Thomas, J.D. Bowman, E. Sobel, T.C. Cheng, J.M. Peters, *Am. J. Epidemiol.* 137 (3) (1991) 381.
- [23] J.H. Olsen, A. Nielsen, G. Schulgen, *Br. Med. J.* 307 (6909) (1993) 891–895.
- [24] G.H. Schreiber, G.M. Swaen, J.M. Meijers, J.J. Slangen, F. Sturmans, *Int. J. Epidemiol.* 22 (1) (1993) 9–15.
- [25] P.K. Verkasalo, E. Pukkala, M.Y. Hongisto, J.E. Valjus, P.J. Jarvinen, K.V. Heikkila, M. Koskenvuo, *Br. Med. J.* 307 (6909) (1993) 895–899.
- [26] M. Feychting, A. Ahlbom, *Am. J. Epidemiol.* 138 (7) (1993) 467–481.
- [27] W.G. Siems, O. Sommerburg, T. Grune, *Clin. Nephrol.* 53 (2000) 59–67.
- [28] B.Y. Zheng, G.D. Yao, L.H. Xie, Y. Lin, D.Q. Lu, H. Chiang, *Proceedings of the Second World Congress for Electricity and Magnetism in Biology and Medicine.* <http://infoventures.com/emf/meetings/bems/97/343.html> 1997.
- [29] M. Iwsaka, S. Ueno, *Proceedings of the Second World Congress for Electricity and Magnetism in Biology and Medicine.* <http://infoventures.com/emf/meetings/bems/97/321.html> 1997.
- [30] S. Roy, Y. Noda, V. Eckert, M.G. Traber, A. Mori, R. Liburdy, L. Packer, *FEBS Lett.* 376 (1995) 164–166.
- [31] J.C. Scaiano, F.L. Cozens, N. Montal, *Photochem. Photobiol.* 62 (1995) 818–829.