Effects of GSM-Frequency Electromagnetic Radiation on Some Physiological and Biochemical Parameters in Rats

E. E. Khirazova, A. A. Baizhumanov*, L. K. Trofimova*, L. I. Deev*, M. V. Maslova, N. A. Sokolova, and N. Yu. Kudryashova

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 153, No. 6, pp. 791-794, June, 2012
Original article submitted March 30, 2011

Single exposure of white outbred rats to electromagnetic radiation with a frequency 905 MHz (GSM frequency) for 2 h increased anxiety, reduced locomotor, orientation, and exploration activities in females and orientation and exploration activities in males. Glucocorticoid levels and antioxidant system activity increased in both males and females. In addition to acute effects, delayed effects of radiation were observed in both males and females 1 day after the exposure. These results demonstrated significant effect of GSM-range radiation on the behavior and activity of stress-realizing and stress-limiting systems of the body.

Key Words: *electromagnetic radiation; behavior; antioxidant protective system; glucocorticoids*

The safety of GSM-range electromagnetic radiation (EMR) emitted by cell phones becomes now an urgent problem. It has been previously shown that GSM-range EMR induces accumulation of lipid peroxidation products, reduced activities of superoxide dismutase, glutathione peroxidase, and catalase with simultaneous activation of xanthine oxidase and adenosine deaminase in the brain tissues and other organs of experimental animals [8,12]. These changes can induce CNS dysfunction and affect behavioral activity of animals, which prompted us to study the effects of GSM-range EMR on behavioral reactions.

Here we investigated the main behavioral patterns and biochemical parameters characterizing stress-realizing and stress-limiting systems in laboratory animals (white rats) exposed to EMR.

MATERIALS AND METHODS

The experiments were performed on 10-12-week-old male and female white outbred rats weighing 260-350 g (n=10 for each group). The study was carried out in accordance with ethical principles on animal welfare and regulating documents of European Science Foundation and Declaration of Helsinki.

The test cell phone GSM 900 (model GF337, Ericsson) was used as the source of EMR [6]. The animals were exposed to 905-MHz EMR (pulse power 2 W); the specific absorbed dose for rats was 1.67 W/kg. Static magnetic field during the experiments varied from day to day in the range of 50-60 μT, alternating low-frequency magnetic field did not exceed 50 nT. The signal included standard GSM modulations during operation in discontinuous transmission mode.

The experimental animals were placed into a plastic container and exposed to EMR for 2 h. The control animals spent 2 h under the same conditions but without irradiation.

In experimental series I, we evaluated acute (group 1) and delayed (group 2) effects of EMR on behavior. Registration of rat behavioral activity in the open field test was started 5 min (group 1) or 24 h (group 2) after exposure. Rat behavior was recorded over 15 min in complete silence under a 15 W red lamp. The registered parameters allow assessment of locomotor, orientation, and exploratory activities and anxiety level [3].

Department of Human and Animal Physiology, *Department of Biophysics, Biological Faculty, M. V. Lomonosov Moscow State University, Russia. ***Address for correspondence:*** lidia.trofimova@gmail.com. L. K. Trofimova

0007-4888/12/1536-0817

In II experimental series, the state of the antioxidant defense system (ADS) was assessed by blood levels of non-peptide thiols [1], SOD-like [7] and catalase [10] activities in blood hemolysate, and hemoglobin content in the blood [9]. In addition, we measured SOD-like activity of blood plasma [7], plasma levels of ceruloplasmin [1], lipid hydroperoxides [4], total protein [1], and thiobarbituric acid-reactive substances [1]. These parameters allow assessment of total level of ADS system activation in the body and the level of oxidative stress. SOD-like plasma activity was expressed in arb. units/mg protein, SOD-like hemolysate activity – in arb. units/g hemoglobin (50% inhibition of adrenaline oxidation was taken for 1 arb. units). In addition, plasma level of glucocorticoids reflecting activity of the hypothalamic—pituitary—adrenal axis was measured [2].

The results were statistically processed using Microsoft Excel and Statistica 6.0 software and nonparametric (Mann-Whitney *U* test, exact Fisher test) and parametric (Student's *t* test) tests.

RESULTS

In experimental series I, evaluation of EMR effects on behavioral activity showed that acute effects (5 min after exposure) were oppositely directed in males and females. In males, EMR exposure increased orientation, exploratory, and locomotor activities and reduced anxiety (as measured by vertical motor activity, number of steps out from walls, and center visits). Females demonstrated reduced locomotor, orientation, and exploratory activities (by vertical motor activity, number of steps out from walls, and center visits) and increased anxiety (by the number of defecations and total freezing time; Table 1).

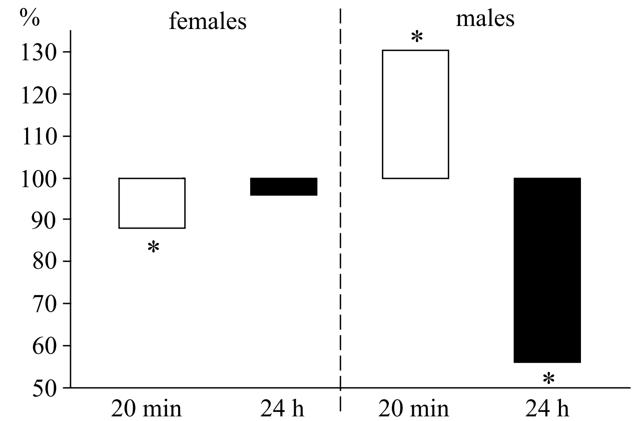

Fig. 1. Dynamics of glucocorticoid plasma levels in males and females during the acute and delayed periods after EMR exposure (% of control). *p<0.05 in comparison with the control.

In group 2 (24 h after training), females retained EMR effect on anxiety level: freezing time and defecation number were significantly increased. However, delayed consequences of EMR in males appeared to be opposite to the acute ones: the number of center visits and steps out from the walls were significantly decreased, which attested to reduced locomotor, orientation, and exploratory activities and increased anxiety (Table 1).

These findings suggests that EMR induced both acute and delayed changes in locomotor, orientation, and exploratory activities and in anxiety level in females and males; moreover, these changes were oppositely directed in males and co-directed in females.

TABLE 1. Changes in Open-Field Behavior of Male and Female Rats after Exposure to EMR (*M*±*m*)

Group	Parameter	Horizontal motor activity, arb. units	Vertical motor activity, arb. units	Steps out form walls and center visits	Freezing time, sec	Number of defecations, arb. units
			Acute effects (5 min after exposure to EMR)			
Males	control	60.4±18.2	7.9±2.7	1.2±0.6	394.1±67.4	1.4±0.7
	EMR	68.9±12.1	19.1±3.6*	4.5±1.0*	275.9±69.0	0.5±0.3
Females	control	133.2±20.3	45.1±8.6	15.2±4.1	87.1±46.5	0.5±0.1
	EMR	56.0±14.2*	11.5±5.4*	2.2±0.9*	338.8±82.5*	2.3±0.9*
			Delayed effects (24 h after EMR)			
Males	control	105.6±15.3	27.3±4.4	10.9±2.2	251.9±72.7	3.0±0.6
	EMR	95.8±14.1	26.3±4.1	3.9±0.5*	264.6±62.9	2.0±0.5
Females control	110.0±7.4	31.8±4.2	9.6±0.9	144.2±19.2	0.4±0.2	
	EMR	137.3±18.9	25.7±5.6	9.3±2.0	343.5±103.3*	3.8±1.1*

Note. *p<0.05 in comparison with the control.

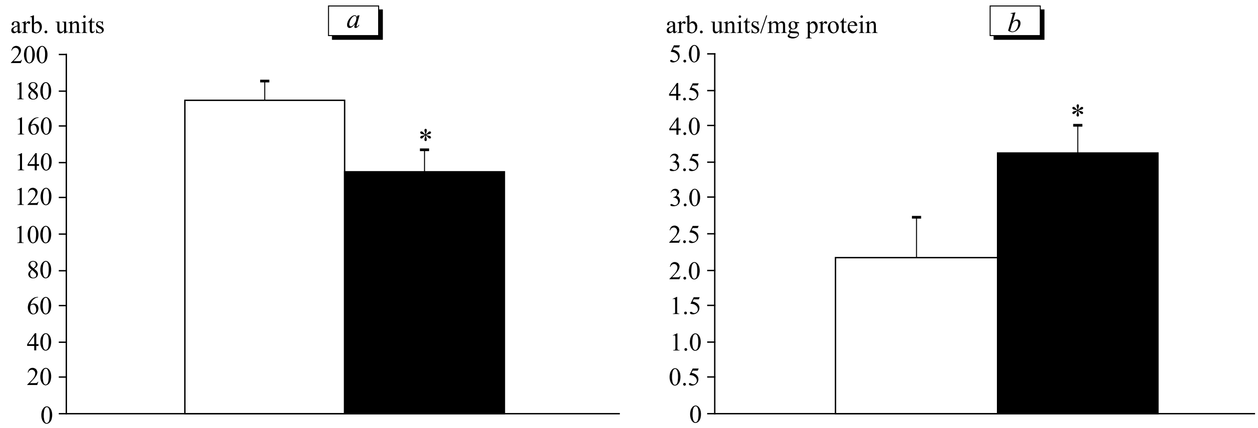

Fig. 2. Dynamics of plasma levels of lipid hydroperoxides during the acute period (*a*) and SOD-like activity in delayed period after exposure to EMR (*b*) in females. Light bars: control, dark bars: EMR. **p*<0.05 in comparison with the control.

The observed acute and delayed effects of EMR on behavioral parameters can be stipulated by changes in activity of dopaminergic, glutamatergic and GABA-ergic brain structures described previously in rats exposed to GSM-range EMR [11].

Another possible cause of the above-described changes in behavioral patterns can be reduced activity of the hypothalamic—pituitary—adrenal axis in exposed animals. Analysis of plasma levels of glucocorticoids revealed co-directed shifts in the acute period (increase in hormone levels) in males and females. In the delayed period, the effects of EMR were seen only in males and manifested in decreased glucocorticoid levels (Fig. 1).

Since the effects of EMR on plasma level of glucocorticoids in males and females during the acute period were co-directed and manifested in increased hormone level, the decrease in glucocorticoid concentration in males 1 day after the exposure probably resulted from negative feedback in the hypothalamic—pituitary—adrenal axis. Thus, increased blood hormone levels provoke reduction of corticotropin-releasing factor and adrenocorticotropic hormone production, which leads to suppression of the adrenal cortex activity and reduces glucocorticoid release [5]. It cannot be excluded that unchanged level of glucocorticoids in females 24 h after the exposure is maintained by the same mechanisms, but these reactions develop with different velocity in comparison with the males. This assumption is speculative and requires experimental verification.

Evaluation of the effects of EMR on ADS parameters has shown that the levels of lipid hydroperoxides decreased in males 20 min after irradiation in comparison with the control (Fig. 2, *a*). In females, increased SOD-like plasma activity was detected 24 h after EMR (Fig. 2, *b*). No other changes in ADS parameters were noted at these times.

This data attest to ADS response to EMR exposure and possible gender differences in the rate of this reaction development in experimental animals. The mechanisms of the observed sings of ADS stimulation against the background of increased activity of the stress-realizing system during the acute period after EMR exposure remain unclear. This effect can be compensatory in nature and stipulated by mobilization of endogenous antioxidants, glutathione peroxidase activation and by other ADS components (not evaluated in our study) in response to intensification of lipid peroxidation and generation of reactive oxygen species during the exposure.

Our findings suggest that single exposure to GSM-range EMR with 905 MHz frequency exerts a long-term (at least for 24 h) substantial influence on locomotor, orientation, and exploratory activities and anxiety, as well as on activity of the stress-realizing and stress-limiting systems in rats. The direction of some effects depends on animal gender.

REFERENCES

1. M. Ya. Akhalaya, A. A. Baizhumanov, N. A. Brazhe, *et al.*, *Dokl. AN.*, **415**, No. 6, 826-828 (2007).
2. L. K. Trofimova, A. A. Baizhumanov, E. N. Goncharenko, *et al.*, *Izv. Ross. Akad. Med. Nauk. Ser. Biol.*, No. 1, 101-106 (2011).
3. L. K. Trofimova, I. A. Surova, M. V. Maslova, *et al.*, *Neirokhimia*, **26**, No. 3, 220-224 (2009).
4. A. Alberti, L. Bolognini, D. Macciantelli, and M. Caratelli, *Res. Chem. Intermed.*, **26**, No. 3, 253-267 (2000).
5. A. Armario, *CNS Neurol. Disord. Drug Targets*, **5**, No. 5, 485-501 (2006).
6. I. Y. Belyaev, C. B. Koch, O. Terenius, *et al.*, *Bioelectromagnetics*, 27, No. 4, 295-306 (2006).
7. Y. Fu and L. L. Ji, *J. Nutr.*, **133**, No. 11, 3603-3609 (2003).
8. A. Iehan, A. Gurel, and F. Armutcu, *Clin. Chim. Acta.*, **340**, No. 1-2, 153-162 (2004).

9. A. Karsan, I. Maclaren, D. Conn, and L. Wadsworth, *Am. J. Clin. Pathol.*, **100**, No. 2, 123-126 (1993).
10. B. Kula, A. Sobczak, and R. Kuska, *J. Occup. Health*, **44**, 226-229 (2002).
11. A. L. Mausset-Bonnefont, H. Hirbec, X. Bonnefont, *et al.*, *Neurobiol. Dis.*, **17**, No. 3, 445-454 (2004).
12. F. Oktem, F. Orgunez, H. Mollaogly, *et al.*, *Arch. Med. Res.*, **36**, No. 4, 350-355 (2005).