

ONCOLOGY

Effect of Radiation from Personal Computer Monitor on Free-Radical Processes in Rats

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Radiation from a personal computer monitor reduces antioxidant activity and the contents of diene conjugates and Schiff bases in rat serum and has no effect on chemiluminescence and the content of carbonyl derivatives. The radiation causes no significant changes in blood contents of nitrites and ceruloplasmin and activity of Cu,Zn-superoxide dismutase. The results obtained indicate that radiation from a monitor produces potentially hazardous biological effects.

Key Words: *monitor; lipid peroxidation; antioxidant systems*

The problems associated with biological effects of radiation from a personal computer monitor (PCM) have been discussed in the literature [1,8,13]. A PC user is exposed to electromagnetic field (EMF), visible and ultraviolet light, radio-range waves, and low-frequency fields (50 Hz) and their harmonics [8]. Preliminary experiments showed that EMF of varied frequency and intensity activates some oncogenes, suppresses the immune system, causes neurologic disorders, impairs the function of hypothalamo-hypophyseal system, and increases the risk of tumorigenesis [7,13]. The incidence of premature births and infants born with pathologies and the risk of brain tumors are higher for PC operators [13]. The PCM radiation stimulates the growth of urethane-induced lung tumors in mice [2]. Modification of free-radical processes in the organism may be a key factor in the mechanism of biological effects of PCM radiation.

These processes play an important role in age-related pathologies, atherosclerosis, immunodepression, and tumorigenesis. As we are aware, the effects of PCM radiation on free-radical oxidation and antioxidant systems have not been investigated. The present study is an attempt to examine these effects.

MATERIALS AND METHODS

Experiments were performed on 40 male 3-month-old LIO rats bred at the N. N. Petrov Institute of Oncology. The rats were maintained in polypropylene cages (55×36×15 cm, 5-6 animals) at 21-23°C and 12:12 h light regime and had free access to standard vivarium chow and water. A Litron Professional Co. PC/AT-286 computer (Singapore) with an EGA color monitor (230 V, 50 Hz, 0.4 A, Taiwan) was used as a source of radiation (Fig. 1). A text file was on the monitor during irradiation (1 h during a 5-day period). Control rats were subjected to the same manipulations without irradiation. The doses of PCM radiation components were measured as previously [2].

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The parameters of free-radical processes were measured in serum. Rats were sacrificed on day 6 of experiment in the morning after an overnight starvation. Blood was centrifuged for 15 min at 1500 rpm; serum was collected and stored at -20°C. The intensity of chemiluminescence initiated with 2% H₂O₂ was measured at 37°C as light sum for 2 min and expressed in arb. units/mg protein [4]. Lipid peroxidation was assessed by serum contents of primary (diene conjugates) and final (Schiff bases) products of lipid peroxidation (LPO) [4,7]. Peroxidation of serum proteins was assessed by serum content of carbonyl derivatives of amino acids [8]. Serum antioxidant activity was determined by the intensify of riboflavin chemiluminescence [14], and activity of Cu,Zn-superoxide dismutase (SOD) was assayed by suppression of nitro blue tetrazolium oxidation [5,6]. The content of ceruloplasmin was determined by its oxidizing activity toward p-phenylenediamine [14]. The content of nitrites was measured using the Griss reaction after precipitation of proteins with 5% zinc sulfate [9]. The results were processed using Student's *t* test and Wilcoxon—Mann—Whitney test.

RESULTS

Dosimetry showed that radiation levels in the laboratory and vivarium were practically the same: intensity of ultralow-intensity alternate magnetic field was 0.4 mT and illumination was 20-80 lx [2]. In comparison with the parameters outside the zone of exposure to PCM, in the exposure zone the intensity of ultralow-intensity alternate magnetic field and the intensities of high-frequency EMF and static electric fields were higher, while illumination and intensities of low-frequency alternate magnetic field and X-ray radiation [2] were practically the same.

Radiation from PCM had no effect on the intensity of serum chemiluminescence and slightly reduced the content of CO-derivatives in proteins

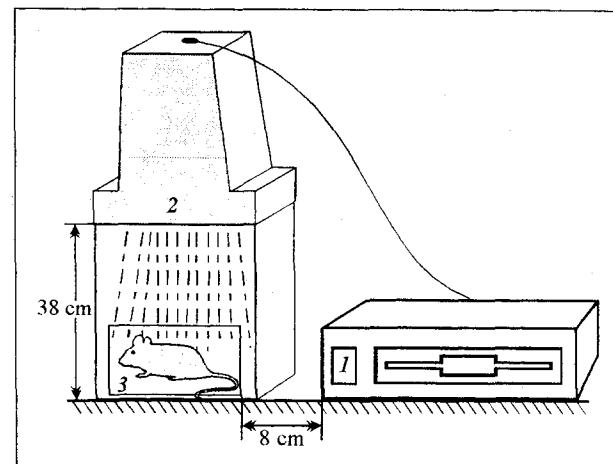


Fig. 1. Installation for irradiation of animals with a personal computer monitor. 1) computer; 2) monitor; 3) cage with rat.

during the 1st week. At the same time, it decreased serum content of diene conjugates by 53% and that of Schiff bases by 22% (Table 1). Suppression of LPO processes correlated with suppression of serum total antioxidant activity (23%, $p<0.05$) and had no effect on SOD activity and ceruloplasmin concentration remained unchanged. There was a tendency toward an increase in the total content of nitrites, which can be regarded as an indirect indication of the involvement of nitric oxide in free-radical oxidation induced by PCM radiation.

A decrease in blood content of LPO products was observed in chronic intoxication [3] and at the early stages of tumorigenesis [5,6]. This decrease may result from suppression of oxidation-reduction processes in blood serum and liver as well as from nonspecific adaptive modifications of lipids, specifically, a decrease in the content of unsaturated fatty acids. Taken together with the literature data, our results are consistent with the concept that suppression of LPO at the initial stages of stress reflects immediate adaptive reaction of an organism to the

TABLE 1. Effect of PCM Radiation on Free-Radical Oxidation in Rat Serum¹

Parameter	Control (n=7)	PCM (n=6)
Chemiluminescence intensity, arb. units/mg protein	1.05±0.11	1.05±0.04
Carbonyl derivatives, mmol/mg protein	0.85±0.07	0.76±0.09
Diene conjugates, nmol/mg protein	0.099±0.012	0.047±0.007**
Schiff bases, arb. units/mg protein	0.487±0.036	0.380±0.014*
Nitrites, pmol/mg protein	16.4±1.7	19.2±2.9
Ceruloplasmin, mg/dl	34.7±3.9	33.2±1.9
SOD, U/mg protein	1.93±0.03	2.02±0.27
Antioxidant activity, arb. units/mg protein	0.82±0.03	0.63±0.04*

Note. ¹Serum from 2-3 rats was used for one determination. * $p<0.05$, ** $p<0.01$ compared with the control.

stressful factor. Investigation of the effects of longer exposure to PCM radiation on LPO may provide more information regarding this issue.

Our results indicate that radiation from a PCM may produce adverse effects on the health of users who ignore safety measures [1]. Protective filters reduce the hazard of PCM radiation.

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REFERENCES

1. V. N. Anisimov, *Priroda*, No. 2, 78-85 (1995).
2. V. N. Anisimov, I. G. Popovich, M. A. Zabeyzhinskii, *et al.*, *Vopr. Onkol.*, **42**, No. 1, 77-81 (1996).
3. S. O. Burmistrov, O. N. Mashek, and I. I. Stepanova, *Eksp. Klin. Farmakol.*, **55**, No. 5-6, 55-60 (1992).
4. I. D. Stal'naya, in: *Modern Methods in Biochemistry*, V. N. Orekhovich (Ed.) [in Russian], Moscow (1977), pp. 63-66.
5. S. Chevari, T. Andryal, K. Benke, and Ya. Shtrenter, *Vopr. Med. Khimii*, **38**, No. 5, 4-5 (1992).
6. S. Chevari, I. Chaba, and Y. Sekei, *Lab. Delo*, No. 11, 678-681 (1985).
7. K. S. Chiao and A. L. Tappel, *Biochemistry*, **8**, 2821-2827 (1969).
8. *Electromagnetic Fields (300 Hz to 300 GHz)*. *Environmental Health Criteria* 137, Geneva (1993).
9. J. P. Kehrer, *Crit. Rev. Toxicol.*, **23**, 21-48 (1993).
10. R. L. Levine, D. Garland, C. N. Oliver, *et al.*, *Methods Enzymol.*, **186**, 464-485 (1990).
11. F. Madueno and M. G. Guerro, *Anal. Biochem.*, **198**, 200-202 (1991).
12. J. J. Moreno and W. A. Prior, *Chem. Res. Toxicol.*, **5**, 425-431 (1992).
13. G. Pinholster, *Environ. Health Perspect.*, **101**, 292-297 (1993).
14. H. A. Ravin, *J. Lab. Clin. Med.*, **58**, 161-168 (1968).
15. B. H. Strekler and C. S. Soup, *Arch. Biochem. Biophys.*, **47**, 8-15 (1953).
