Exposure to Low-Intensive Superhigh Frequency Electromagnetic Field as a Factor of Carcinogenesis in Experimental Animals

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 139, No. 2, pp. 211-214, February, 2005
Original article submitted April 26, 2004

Inbred albino mice and C57Bl/6 mice were exposed to nonthermal radiation of 37 GHz frequency in order to detect delayed effects caused by repeated irradiation. The detected pathomorphological changes and the dynamics of their formation suggest that these factors are responsible for delayed formation of immunodeficiency provoking mutagenic and carcinogenic effects.

Key Words: *electromagnetic radiation; mutagenesis; carcinogenesis; immunodeficiency; thyroid glang*

The effects of bioactive agents in supralow doses on biochemical and physiological processes have been described [2]. Biological objects react to peptides, hormones, and toxins in concentrations below 10^{-12} M. Results of experiments in magnetobiology, a science actively developing during the latest 15-20 years [1,3-8], indicate that living organisms are no less sensitive to low-intense electromagnetic radiation (EMR), even as low as countable quanta, *i. e.* energy flow surface density of $P \sim 10^{-20}$ W/Hz/cm^2, which is comparable to the intensity of connate basal radiation of distant airspace [6,7].

Several concepts of the efficiency of exposure of biological objects to low-intensive EMR are put forward by the present time [1,2,4,5], but no unambiguous solution of the problem is yet offered. Our position consists in initiation of a "chain reaction" in the biochemical processes of vital activity, stochastic [6] and chiral [3] resonance, and evolutional immanence of live organisms to natural supralow intensity EMR, including that of airspace origin [7].

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This problem is interesting not only from the natural science viewpoint, but practically as well: the methods of superhigh frequency (SHF) therapy, that is, exposure to EMR of 30-300 GHz, are widely, we may even say, on an overall scale, introduced in clinical practice [5-7]. EMR with $P<10$ mW/cm^2 (below threshold thermal exposure) are used.

Positive therapeutic effects gave grounds for the use of SHF EMR in cardiology, neurology, endocrinology, gastroenterology, dentistry, and many other spheres of practical medicine [5-7]. Fine mechanisms of SHF EMR interactions at the levels of cellular and subcellular structures, including cellular enzymatic systems, regulator proteins, and nucleic acid molecules directly responsible for programming and translation of bioinformative processes, remain unknown. Delayed effects of SHF therapy are virtually not studied.

We studied the delayed effects caused by multiple exposure of animals to SHF EMR in modes adequate to SHF therapy procedures.

The aim of our study was to prove that the biological effects of SHF EMR should not be regarded as unambiguously positive; selection of the parameters of SHF EMR should be well-grounded and the patients for this therapy should be selected more stringently.

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MATERIALS AND METHODS

Experiments for detection of delayed effects of SHF EMR exposure were carried out on adult C57Bl/6 mice and inbred albino mice. The study was aimed at clearing out the effects of only one environmental factor, and hence, the control group consisted of intact animals not taken into experiment. The animals were exposed to SHF EMR of 37 GHz and $P<10$ mW/cm^2 (nonthermal nonionizing radiation). All mice were repeatedly exposed to SHF EMR with these parameters for 1 month, this simulating SHF therapy procedures in patients [8]. The total duration of exposure was 3.5 h for all groups. The methodology of the experiment was aimed at detection of delayed effects caused by multiple exposure to SHF EMR, specifically, at evaluation of dynamic pathomorphological changes associated with the formation of immunodeficiency, leading to negative carcinogenic and mutagenic effects.

The study was carried out on specially irradiated animals and animals exposed to SHF EMR in previous experiments.

RESULTS

Macroscopic and morphological changes were evaluated during delayed period after repeated exposure of adult mice and their progeny to SHF EMR.

General degenerative changes and red bone marrow hypoplasia, leading to death of 80% animals, were detected 6-8 months after exposure of C57Bl/6 mice to SHF EMR.

Mutation effects were registered in the first generation descending from survived parents; the life span of the progeny did not surpass 5-10 days. The life span of the parents later did not surpass 6-8 months. The results of this stage of experiment were regarded as pathological prerequisites for the formation of oncological processes, which prompted further observation of mice survived after exposure and their progeny.

As irradiated C57Bl/6 mice gave no viable progeny and died early, further studies were carried out on inbred albino mice and their progeny.

The animals were observed for 1 year starting from the beginning of the experiment. The following specific features were detected in animal development. The mice gave viable progeny without visual pathological changes during 10 months after the exposure, but after 8-10 months the first generation, similarly as their parents, developed tumors of the neck and ileac area. Macroscopically the tumors looked like nodes of irregular shape from 1.5×3 to 2.5×3.5 cm in size (Fig. 1). The tumor nodes had no clear-cut interface and were tightly fixed to the adjacent tissues. Dissected tumor was light-gray with degradation areas.

Microscopic study of preparations from paraffin blocks showed numerous fragments of glandular cancer, whose elements were situated in the derma (Figs. 2, 3).

The tumor structure was solid, cystic alveolar, and cribrous; at some sites the structure was similar to that of adenocystic cancer (cylindroma). Foci of hemorrhages and small necrotic foci were seen. Immunohistochemical study with antibodies to thyroglobulin, calcitonin, and human chromografin *A* showed negative reaction of tumor cells. Staining of the stromal component of the tumor, necrotic and hemorrhagic foci was slightly positive, while staining of amorphous homogeneous substance filling some cystic cavities (in reaction with antibodies to thyroglobulin and chromografin *A*) was sharply positive. The antibodies used in the study cross-reacted with the above-mentioned structures of the tumor and the homogeneous substance filling the cysts.

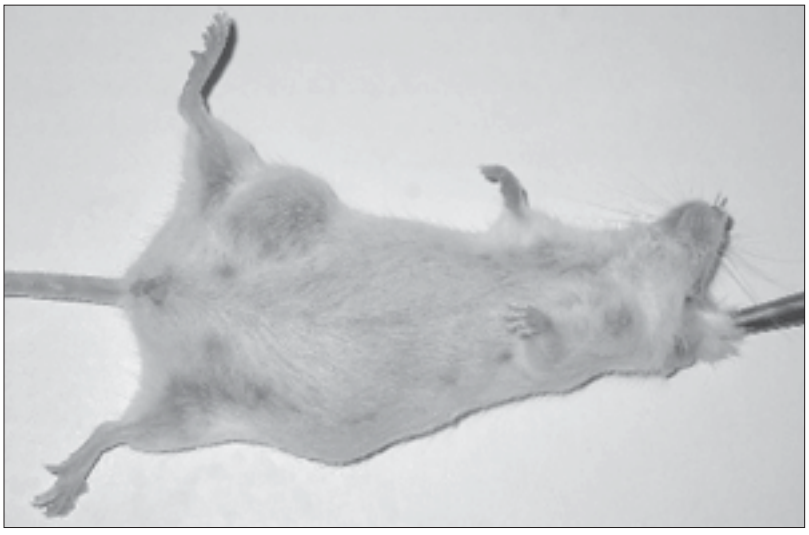

Fig. 1. Tumor node (macroscopic picture).

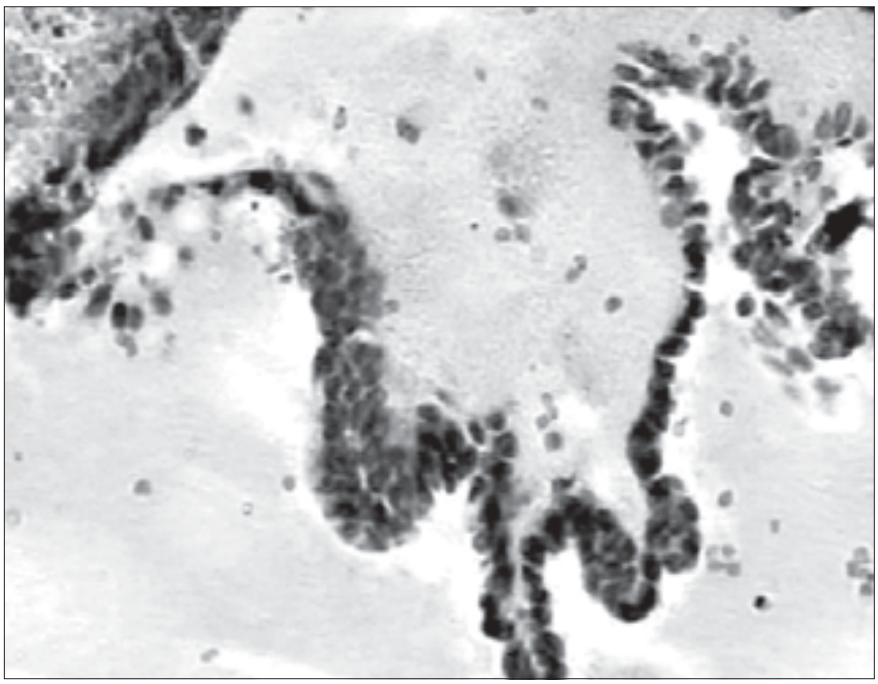

Fig. 2. Papillary thyroid cancer (microscopic picture of tumor tissue), ×300.

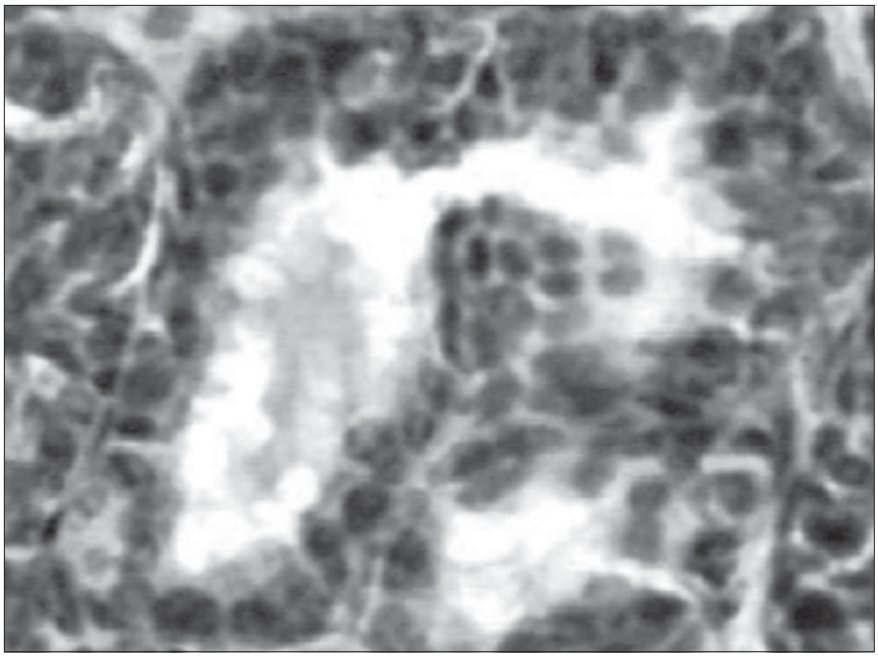

Fig. 3. Fragment of tumor tissue formed by atypical alveolar and cribrous structures, ×600.

Morphological studies were carried out at Pathology Department, Tula Regional Hospital, and Medical Radiology Research Center, Russian Academy of Medical Sciences, in Obninsk.

Pathomorphological changes and the time course of their formation indicate that exposure to SHF EMR of the above-mentioned parameters is a factor causing delayed formation of immunodeficiency, provoking mutagenic and carcinogenic effects. The authors cannot rule out the situation of induced carcinogenesis at the level of cell genome. Presumably, DNA analysis will give a positive answer.

It is noteworthy that the damaging effects of SHF EMR were observed in previous experiments [6], *e. g.* morphological changes of compensatory adaptive type, stimulation of mitotic activity of the cells, delayed negative dysplastic changes, *etc.*

The conclusions drawn from these results are the most important for clinical SHF therapy [6,8]. By no means excluding SHF therapy from the armory of nonmedicamentous methods of treatment, it is important to pay more attention to choice of its parameters, primarily EMR frequency and radiation power. It seems that this latter value can be recommended at the level

of $P<0.10$-0.01 μW/cm^2, and the therapy should include an active feedback system [6]. The use of SHF therapy for patients of reproductive age should be limited.

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