

Effect of Low-Intensity Extremely High Frequency Radiation on Reproductive Function in Wistar Rats

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The exposure to low-intensity extremely high frequency electromagnetic radiation during spermatogenesis was accompanied by pathological changes, which resulted in degeneration and polymorphism of spermatozoa. The number of newborn rats increased in the progeny of irradiated animals.

Key Words: *spermatogenesis; extremely high frequency radiation; Wistar rats; polymorphism; centriole*

The exposure to low-intensity nonthermal radiation ($<10 \text{ mW/cm}^2$) of the extremely high frequency (EHF) range (30-300 GHz) has several sanogenic and pathological effects in mammals [2,3]. They manifest in the impairment of mitosis and meiosis leading to reproductive dysfunction.

Here we studied the effect of EHF radiation on spermatogenesis in Wistar rats. We also evaluated the delayed effect of irradiation in the offspring of these animals.

MATERIALS AND METHODS

In series I, we studied the influence of EHF electromagnetic radiation (EMI) on spermatogenesis. The conditions of treatment (power $>0.3 \text{ mW/cm}^2$, irradiation time $t=30$) were similar to those of EHF therapy in clinical practice [3]. The experiments were performed on 10 adult Wistar rats with normal reproductive function. The degree of sperm degeneration was estimated throughout the experiment.

The results were analyzed 7 days after irradiation. The minimum total time of exposure (t_e^{max}) was 31.5 h (63-day irradiation). The period of

exposure ($0, t_e^{\text{max}}$) was divided into 10 sessions. The testicles were isolated from narcotized animals by the end of each session (including the basal state). The samples were fixed. Microsamples were prepared, routinely stained with hematoxylin and eosin, and examined under a NIKON ESLIO SE-400 microscope (maximum magnification 6×600). Degenerative changes were studied by counting normal and abnormal cells at the stage of late spermatids. Particular attention was given to polymorphism of sex cells, which resulted from aberrations in cell differentiation and served as the source of degeneration.

In series II, each pair of males was exposed to EHF EMR of similar duration (first pair, $1t_s=3.5 \text{ h}$; second pair, $2t_s=7 \text{ h}$; etc.; $t_s^{\text{max}}=17.5 \text{ h}$). The testicles were obtained weekly from one of the males and subjected to a morphological study. Another male was coupled with a female rat. This male was removed with the appearance of the first signs of pregnancy in the female (11th day after coupling). Newborn rats were examined and counted to evaluate the delayed effect of EHF EMR. We selected 1 male and 2 females of generation F1 from the male parent exposed to EHF EMR. Further experiments were performed with generation F2, etc. We studied mutations, reproductive function, number of newborns in the litter, etc.

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TABLE 1. Birth Rate in Control Wistar Rats

Experiment, No.	Date of coupling	Date of labor (days after coupling)	Number of newborns per litter
1	9.04.05	3.05.05 (on day 24)	9
2	23.04.04	19.05.05 (on day 26)	12
3	18.05.05	9.06.05 (on day 22)	8
4	27.05.05	24.06.05 (on day 28)	9
5	3.06.05	27.06.05 (on day 24)	9

TABLE 2. Birth Rate in Experimental Group Wistar Rats (Series II)

Experiment, No.	Date of coupling with female	t_{Σ} of EHF EMR	Date of removal of male	Date of labor (days after coupling)	Number of newborns per litter
1	9.04.05 (day 7)	3.5 h	26.04.05 (after 17 days)	7.05.05 (on day 28)	14
2	23.04.04 (day 14)	7 h	4.05.05 (after 11 days)	17.05.05 (on day 24)	10
3	18.05.05 (day 21)	10.5 h	27.05.05 (after 11 days)	13.06.05 (on day 26)	14
4	27.05.05 (day 28)	14 h	7.06.05 (after 11 days)	20.06.05 (on day 24)	14
5	3.06.05 (day 35)	17.5 h	20.06.05 (after 17 days)	25.06.05 (on day 22)	14

Note. Start of the study March 31, 2005.

RESULTS

Microscopy in series I showed that the degree of sperm polymorphism in experimental animals is low under basal conditions. Morphological abnormalities were not found in 85% sex cells (normal spermatogenesis). The percentage of abnormal spermatozoa was 35 and 42% on days 7 ($t_{\Sigma}=3.5$ h) and 14 ($t_{\Sigma}=7$ h) after the start of irradiation. The percentage of abnormal spermatozoa increased on days 21-28 (from 48 to 55%) and 35-63 (up to 98%, $t_{\Sigma}^{\max}=31.5$ h).

Degenerative changes in spermatozoa manifested in deformation of the head and appearance of omegapyrene and apyrene spermatozoa with doubled filaments or without filaments. Diploid spermatozoa with 2-3 filaments were detected on days 49-63. The observed morphological changes indicate that low-intensity (nonthermal and bio-informational) EHF EMR impair the interaction between centrioles. They move aside during normal cell division and act synergistically in filament formation. These centrioles are split under conditions of abnormal development, which results in duplication of the proximal part in filaments. Two or more filaments are formed when the centrioles develop into filaments.

Apart from pathological changes in centrioles, the formation of abnormal spermatozoa was probably related to an imbalance in chromosome appa-

ratus. Enlargement and deformation of the spermatozoon head reflected an increase in DNA content in the head of diploid spermatozoon. These changes attest to abnormal conjugation during meiosis and impairment of stage II of sex cell maturation.

In series II, the offspring was obtained from all adult Wistar rats exposed to EHF EMR (Tables 1 and 2). The onset and duration of pregnancy in females corresponded to normal [1]. However, the number of newborn animals in the litter of primiparous females exceeded the normal (6-10 rat pups). It was associated with the presence of abnormal spermatozoa in males of the EHF EMR group.

Our results and published data [2,3] indicate that nonthermal EHF EMR is a pathogenic factor for mammalian spermatogenesis, which determines the number of newborns in irradiated males.

Over the last 10-15 years a wide use of EHF therapy for medical rehabilitation of patients of reproductive age was in conflict with medical ethics.

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