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EFFECTS OF NONIONIZING ELECTROMAGNETIC RADIATION  
(FOUO 1/80) 1 OF 1

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8 February 1980

# USSR Report

BIOMEDICAL AND BEHAVIORAL SCIENCES

(FOUO 1/80)

Effects of Nonionizing Electromagnetic Radiation



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USSR REPORT  
BIOMEDICAL AND BEHAVIORAL SCIENCES  
(FOUO 1/80)

EFFECTS OF NONIONIZING  
ELECTROMAGNETIC RADIATION

This serial publication contains articles, abstracts of articles and news items from USSR scientific and technical journals on the specific subjects reflected in the table of contents.

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CONTENTS	PAGE
Possible Mechanisms Behind the Bioeffects of Low Frequency Electromagnetic Fields (G. I. Yevtushenko, F. A. Kolodub; VOZMOZHNYE MEKHANIZMY BIOEFFEKTOV ELEKTROMAGNITNYKH POLEY NIZKIKH CHASTOT, 1979)..	1
Bioeffects in Response to a Low Intensity Pulsed 9,400 MHz Microwave Electromagnetic Field (Yu. D. Dumanskiy, et al.; BIOEFFEKTY PRI DEYSTVII MALIONTENSIVNOGO IMPUL'SNOGO ELEKTROMAGNITNOGO POLYA MIKHOVOLN CHASTOTY 9400 MGTs, 1979) .....	5
Biological Action of an Industrial Frequency (50 Hz) Electric Field (M. G. Shandala, et al.; BIOLOGICHESKOYE DEYSTVIYE ELEKTRICHESKOGO POLYA PROMYSHLENNOY CHASTOTY (50 GTs) 1979).	16
Immunological Effects of Low Microwave Exposure (M. G. Shandala, et al.; IMMUNOLOGICHESKIYE EFFEKTY VOZDEYSTVIYA MALIONTENSIVNOGO MIKROVOLNOGOGO OBLUCHENIYA, 1979)	26

- a - [III - USSR - 21A S&T FOUO]

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CONTENTS (Continued)	Page
Dynamics of Changes in an Organism's Behavioral Reactions Induced by Microwave Radiation (M. I. Rudnev, M. I. Navakatikyan; DINAMIKA IZMENENIY POVEDENCHESKIKH REAKTSIY ORGANIZMA, INDUSIROVANNYKH MIKROVOLNOVOY RADIATSIYEV, 1979) .....	31
The Effects of Injury and Restoration of the Organism of Rats Under Microwave Irradiation (2400 MHz) (V. S. Tikhonchuk; BYULLETEN' EKSPERIMENTAL'NOY BIOLOGII I MEDITSINY, No 7, 1979).....	41
Kinematic Description of a Helical Frame of Reference System in the Special Theory of Relativity .....	46
Relativistic Kinematic Equations and the Theory of Continuous Media .....	47

- b -

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POSSIBLE MECHANISMS BEHIND THE BIOEFFECTS OF LOW FREQUENCY ELECTROMAGNETIC FIELDS

Unknown VOZMOZHNYE MEKHANIZMY BIOEFFEKTOV ELEKTROMAGNITNYKH POLEY NIZKIKH CHASTOT in Russian 1979 pp 1-6

[Preprint of article by G. I. Yevtushenko and F. A. Kolodub, Khar'kov Scientific Research Institute of Labor Hygiene and Occupational Diseases]

[Text] The biological activity of solenoid-generated low frequency (7 and 70 kHz) and industrial frequency (50 Hz) electromagnetic fields (LF and IF EMF's) of different intensities (10-72,000 amps/meter), exposure times (up to 6 months) and generation modes (continuous, pulsed) was studied in experiments on male white rats.

It was established that at certain EMF intensities and exposure times, depending upon frequency and generation mode, distinct changes arise in the functional state of the central nervous and cardiovascular systems. Morphological changes develop in the CNS, the heart, the liver, the kidneys, and endocrine glands (adenohypophysis, adrenal glands, thyroid, testes), and the morphological blood picture and the body's immunobiological reactivity change.

The observed functional and morphological changes are the product of dissociation of carbohydrate-energy, nitrogen, and nuclein metabolism.

The frequency characteristics of the EMF and its generation mode have an effect on the direction taken by the changes. Thus a decline in the intensity of anaerobic glycolysis was typical of the continuous generation mode, while its activation was typical of the pulsed mode. Pulsed EMF's also cause more expressive changes in nitrogen and nuclein metabolism.

The question arises as to what is responsible for these differences.

It appears that they lie in the different influences EMF's have on the kinetics of enzymatic reactions catalyzing individual links of the metabolic processes.

It should be kept in mind that the activity of an enzyme is governed by many factors, and that it depends on the quantity of the enzymatic protein itself

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and on presence of factors altering the rate of the enzymatic reaction (substrates, coenzymes, activators, inhibitors, apoenzyme conformation, and so on). In our research, we observed a decline in the quantity of amide and sulfhydryl groups in the protein molecules in response to an LF EMF. It may be hypothesized that one of the possible causes of change in enzyme activity might be change in conformation of some enzymatic proteins.

Changes in the ratio of free to bound water in organs and tissues play a certain role in alteration of the catalytic activity of enzymes; evidence of this was found in change in the dielectric permeability of tissues.

Change in the structural rigidity of intracellular water could be elicited by change in the kinetics of different enzymatic reactions, and it could cause conformational changes in a number of proteins and enzymes. These circumstances could be responsible for the numerous deviations noted in enzymatic activity under the influence of LF EMF's in continuous and pulsed generation modes. EMF's play the role of activators in relation to some enzymes (cardiac hexokinase, hepatic and cardiac glucose-6-phosphate dehydrogenase, cerebral, hepatic, cardiac, and muscle adenylate deaminase, cerebral ribonuclease and adenosine triphosphatase, and hepatic and testicular deoxyribonuclease). They are unique inhibitors in relation to other enzymes (hepatic glutamate dehydrogenase, and cerebral cytochrome oxidase, creatine kinase, glutamine synthetase, and deoxyribonuclease). EMF's do not generally have a significant influence on the activity of enzymes such as cerebral, hepatic, and testicular lactate dehydrogenase, cerebral hexokinase, hepatic cytochrome oxidase, and cerebral and hepatic glutaminase.

[One line not reproduced] (for example glutamate dehydrogenase, cytochrome oxidase, glutamine synthetase, and deoxyribonuclease) EMF's varied in their influence depending on the organ to which the enzyme belonged. There should be no difficulties in explaining this fact, since we know that enzymes catalyzing the same reaction but located in different organs often differ in amino acid composition. These differences are governed by optimum medium pH and other factors. Hence it becomes clear that when the state of free water changes by the same order of magnitude, the degree of changes in enzymatic activity and their direction would vary, due to a difference in the degree of conformational changes in enzyme proteins.

A typical feature of the action of EMF's of the studied frequency ranges is their capability for changing the intensity of oxidative processes. Pulsed 7 kHz EMF's and continuous and pulsed 50 Hz fields altered the degree to which oxidation and phosphorylation were linked. A consequence of this was a decline in the level of macroergic compounds in the tissues (ATP and creatine phosphate), and change in the ratio of the oxidized and reduced forms of nicotinamide adenine dinucleotide and flavoproteins.

It would be difficult to unambiguously answer the question as to what sort of concrete mechanisms are responsible for dissociation of oxidation and phosphorylation in response to EMF's. It follows from the provisions of

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quantum mechanics that the energy of a low frequency EMF is insufficient to cause any sort of molecular alteration, even at the highest intensities we studied (72 kA/meter). Theoretical computations confirm the possibility of arisal of induction currents in the body. Their magnitude in the presence of magnetic field intensities greater than 10 kA/meter may be commensurate with those having a pronounced biological effect on certain cell components. One such effect might be disturbance of the structural integrity of mitochondrial membranes. It is precisely this circumstance which dominates in the mechanisms responsible for dissociation of oxidation from phosphorylation.

In order to test the suggested hypotheses, we performed *in vitro* experiments to study the effect EMF's have on the structure and functional activity of mitochondria and the activity of some enzymes that undergo change when the entire organism is exposed to an EMF.

It was established that neither irradiation of enzymatic extracts of cerebral and hepatic lactate dehydrogenase and cytochrome oxidase, cerebral, hepatic, and testicular ribo- and deoxyribonuclease (pulsed EMF, 72 kA/meter), cerebral creatine kinase, and cerebral and hepatic adenosine triphosphatase in rats (LF EMF, 70 kHz), nor the effect of continuous and pulsed LF's applied at the moment of formation of enzyme-substrate complexes changed the activity of the studied enzymes. These data provide the grounds for suggesting that changes in enzyme activity arising *in vivo* in response to EMF's are not the consequence of the direct influence of the fields on the conformation and catalytic properties of the enzyme; instead, they obviously stem from disturbances in the regulatory systems. Confirmation of this hypothesis can be found in an analysis of data from research on the structural and functional activity of mitochondria. It was established that the optical density and functional activity of hepatic mitochondria changed only when the entire organism was exposed to a pulsed electromagnetic field (72 kA/meter). The field did not have significant influence upon actively metabolizing hepatic mitochondria *in vitro*.

These data persuade us that disturbances in regulatory systems, particularly the neuroendocrine regulatory systems, play the main role in manifestation of the field's effects upon biochemical processes in the entire irradiated organism.

We demonstrated the EMF's have a significant influence on the functional state of the medullary and cortical layers of the adrenal glands, manifesting itself as a decline in the level of epinephrine in the adrenal glands in response to the action of pulsed EMF's and 70 kHz EMF's, and its increase in response to LF and IF EMF's, and by enlargement of the concentration of 11-oxycorticosteroids in blood plasma in response to 70 and 7 kHz EMF's.

We know that epinephrine and 11-oxycorticosteroids (glucocorticoids) are powerful regulators of carbohydrate metabolism in the body. The possibility is not excluded that changes of different directions in the body's carbohydrate-energy metabolism stem precisely from the different influences EMF's of the studied frequency ranges have on the hormone concentrations.



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According to present ideas steroid hormones, androgens in particular, have an important role in regulation of interstitial metabolism. We demonstrated that an LF EMF reduces the concentration of testosterone in blood draining from the testes from  $1.32 \pm 0.35$   $\mu\text{g}$ -percent in control animals to  $0.23 \pm 0.01$   $\mu\text{g}$ -percent in rats exposed to pulsed EMF's.

It may be hypothesized that the body's testosterone deficiency could have influenced the intensity and direction of biochemical processes. To test this hypothesis we determined a complex of biochemical indicators that change in response to pulsed EMF's; the test objects included rats exposed to pulsed EMF's and rats simultaneously receiving testosterone. Injection of testosterone propionate (1 mg/kg) into animals exposed to the pulsed EMF did not influence the glycogen level in any of the analyzed organs, but it did cause an adjustment in the concentration of lactic acid and ammonium, and it increased the level of RNA in the liver. The obtained data confirmed presence of a direct relationship of disturbances in the hypophysis-gonad system to arisal and development of certain metabolic disorders in the body. Other hormones may also cause disturbances in metabolic processes.

We know that thyroid hormones have the greatest dissociating effect. We hypothesized that dysfunction of the thyroid may lead to dissociation of oxidative phosphorylation, since 7 and 70 Hz EMF's produced morphological signs of thyroid hyperfunction. But when animals were given mercazolyl at doses of 0.8-3.2 mg/kg (thyrostatic doses) in conjunction with irradiation, dissociation of oxidation from phosphorylation was averted.

Thus changes occurring in the neuroendocrine regulatory systems doubtlessly have a part in the development of numerous metabolic deviations arising in response to LF, IF, and SHF EMF's.

It appears from our research and from the published data that metabolic shifts observed in response to EMF's of industrial and low frequencies are obviously mediated by a complex of changes arising at different levels: molecular (Zeeman effect), subcellular, cellular, and tissue (change in structural rigidity of water and in transport of ions and ionized molecules, and arisal of induction currents), and organismic (owing to development of adaptive reactions or the disadapting influence of endocrine systems in the case of their dysfunction).

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BIOEFFECTS IN RESPONSE TO A LOW INTENSITY PULSED 9,400 MHZ MICROWAVE ELECTRO-MAGNETIC FIELD

Unknown BIOEFFEKTY PRI DEYSTVII MALIONTENSIVNOGO IMPUL'SNOGO ELEKTROMAGNITNOGO POLYA MIKROVOLN CHASTOTOY 9400 MGTs in Russian 1979 pp 1-8

[Preprint of article by Yu. D. Dumanskiy, N. G. Nikitina, I. P. Los', L. A. Tomashevskaya, F. R. Kholyavko, Yu. I. Vasilenko, L. G. Andriyenko, S. A. Lyubchenko, S. V. Zotov, and N. P. Gordynya, Kiev Scientific Research Institute of General and Communal Hygiene imeni A. N. Marzeyev]

[Text] As we know from world practice, industrial devices and apparatus utilizing the energy of a microwave electromagnetic field (EMF) have been extensively introduced into various areas of man's economic activity today. Consequently man has naturally begun encountering microwave electromagnetic fields more and more frequently. In this connection researchers in different countries of the world are becoming increasingly more interested in how significant this factor is to the body and in what its consequences are.

It was with this purpose in mind that we studied the bioeffects of pulsed microwave radiation with a frequency of 9,400 MHz ( $\lambda = 3$  cm).

The research was conducted in experimental conditions, in an echoless chamber in which we simulated the distribution pattern of an electromagnetic field in open space (Figure 1) [figures not reproduced]. The internal surface of the chamber was made from radioabsorptive material having an electromagnetic energy reflection factor not greater than 3 percent. An oscillator with adjustable output power working stably at 9,400 MHz was used as the microwave energy source. Electromagnetic energy was fed from the generator along a waveguide to an emitting antenna located at the top of the echoless chamber. This antenna location made it possible to sensibly use the chamber area and achieve a relatively uniform distribution of microwave EMF's at the places where experimental animals were to be irradiated.

Four groups of animals in radiotranslucent containers were placed beneath the emitter (the antenna) within the zone of the formed field--that is, at a distance of not less than

$$d = \frac{2L^2}{\lambda},$$

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from the antenna, where  $L$  is the larger dimension of the horn antenna and  $\lambda$  is wavelength. EMF nonuniformity was not more than 10 percent.

We used 1,200 mongrel white rats as the experimental animals, which were subdivided into six experimental and two control groups depending on the level of the factor under analysis. The experimental and control groups of animals were maintained in identical conditions (food ration, temperature, humidity, light-dark schedule, and so on).

Two series of experiments were conducted in our research on the biological action of microwave EMF's.

The first series involved power flux densities (PFD's) of 115, 60, and 5  $\mu\text{w}/\text{cm}^2$ .

The second series involved power flux densities of 40, 25, and 10  $\mu\text{w}/\text{cm}^2$ .

Each series of experiments lasted 6 months (4 months exposure time to the factor, and 2 months aftereffect period), during which time the experimental animals were exposed to the factor 12 hours each day.

An integrated approach foreseeing the use of physiological, biochemical, immunological, and other analysis methods was used to study the nature of the biological action of a microwave electromagnetic field.

The physiological analysis methods were: determination of the electrocutaneous sensitivity threshold from the magnitude of the summational threshold index (STI); determination of static working capacity (the time during which the animals could maintain their position on a tilted plank); the study of conditioned reflex activity using a combined motor-dietary method.

Analysis of the results of research on the electrocutaneous sensitivity threshold indicates a statistically significant increase in the summational threshold index in response to PFD's of 115 and 60  $\mu\text{w}/\text{cm}^2$  at all exposure times, and 40  $\mu\text{w}/\text{cm}^2$  following 90 days of irradiation (see Table 1). This index did not return to normal until 30 days after irradiation was terminated. Lower PFD's (25-5  $\mu\text{w}/\text{cm}^2$ ) did not have an influence on this index.

Thus these data show that microwaves (9,400 MHz) noticeably reduce electrocutaneous sensitivity, which indirectly indicates a possible influence of this factor on the functional state of the central nervous system. This is confirmed to a certain extent by the results of research on the conditioned reflex activity of the animals.

We assessed the conditioned reflex activity of the animals from the nature of formation and reinforcement of a positive conditioned reflex to an acoustic stimulus, from the length of the latent period of the conditioned reflexes, from the strength of the motor reaction, and from the number of times the conditioned reflexes failed to occur.

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Data from these studies, presented in Table 2, showed that at as low a PFD as  $40 \mu\text{w}/\text{cm}^2$  an increase in the latent period of conditioned reflexes (up to  $5.92 \pm 0.35$  sec, as compared to the initial  $4.07 \pm 0.47$ ,  $t = 3.27$ ), a decrease in the magnitude of the motor reaction to the positive conditioning stimulus ( $53.9 \pm 3.0$ , as compared to the initial  $74.5 \pm 4.7$ ,  $t = 3.74$ ), and extinction of a positive conditioned reflex to a bell occur in the first month. The most pronounced changes in the indices arose in the fourth month of exposure to the factor. The length of the latent period in the reaction to a differentiated stimulus (a buzzer) did not exhibit significant differences in rats of the control and experimental groups ( $10$  and  $25 \mu\text{w}/\text{cm}^2$ ). A statistically significant difference from control was observed in this index only for the group of animals exposed to  $40 \mu\text{w}/\text{cm}^2$ . The strength of motor food acquisition conditioned reactions did not exhibit regular directed changes in either the experimental animals or the control group. These data and the results of a functional test of conditioned reflex extinction and recovery showed that changes arise in the functional state of the central nervous system in response to low intensity microwaves.

Changes in the central nervous system reflected to a certain extent upon the working capacity of the animals as well. The research data showed that PFD's of  $115\text{--}60 \mu\text{w}/\text{cm}^2$  reduce static working capacity by an average of two times. In this case the earlier and more expressive changes were observed in response to a PFD of  $115 \mu\text{w}/\text{cm}^2$  (see Table 1). Lower intensities of the factor (below  $40 \mu\text{w}/\text{cm}^2$ ) did not elicit statistically significant changes.

Thus our analysis of the physiological research results permits the conclusion that a pulsed microwave electromagnetic field ( $9,400$  MHz) does have an influence on the functional state of the central nervous system when the PFD is above  $25 \mu\text{w}/\text{cm}^2$ .

These studies were supplemented by an investigation of some aspects of metabolic processes occurring in the body. In particular we established a phasal nature for cholinesterase activity in the presence of microwave EMF's. Thus the research results showed that a PFD of  $115 \mu\text{w}/\text{cm}^2$  elicits a noticeable increase in blood cholinesterase activity in the first month of exposure, and that in later periods of the experiment it gradually declines (see Table 3). Cholinesterase activity also changes in response to PFD's of  $60$  and  $40 \mu\text{w}/\text{cm}^2$ .

The obtained data permit the hypothesis that change in cholinesterase activity elicited by microwaves apparently has an influence on biochemical reactions supporting normal occurrence of nervous processes in the animal body, which is possibly one of the causes behind the disturbances in the conditioned reflex activity of the animals described earlier.

Further research on metabolic processes allowed us to determine the influence of microwaves on other indicators of enzymatic activity as well.

Thus we established that electromagnetic energy with a PFD of  $115 \mu\text{w}/\text{cm}^2$  elicits a decline in the cytochrome oxidase activity of hepatic and cerebral mitochondria in all periods of the experiment. At lower PFD's ( $60$ ,  $40$ , and

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Table 1. State of Some Behavioral Reactions of Animals Exposed to Pulsed Microwave EMF's (9,400 MHz)

(1) Показатели	Период (2): экспериментальный		(3) ПМЭ мкВт/см <sup>2</sup>				Контроль (4)
	I-воздей-ствие ЗМП-последей-ствие, сутки	II-5	60	40	25	Контроль (4)	
(6) Порог электроочувствительности, V	фон (5)						
I 30	37,9 ± 1,29	37,8 ± 1,08	37,3 ± 1,08	37,7 ± 0,97	37,6 ± 0,86	37,7 ± 0,75	
60	54,3 ± 1,26*	41,7 ± 1,51*	37,4 ± 1,08	38,9 ± 1,19	37,9 ± 0,64	37,9 ± 0,64	
90	54,4 ± 2,81*	41,8 ± 1,29*	37,5 ± 1,29	37,9 ± 0,75	37,8 ± 1,08	37,9 ± 0,64	
120	56,5 ± 3,24*	41,4 ± 1,29*	37,4 ± 1,19	41,8 ± 1,08*	38,2 ± 0,86	37,9 ± 0,75	
II 30	57,8 ± 2,81*	41,8 ± 1,19*	37,5 ± 1,29	42,0 ± 0,86*	39,0 ± 0,97	37,8 ± 0,86	
60	43,6 ± 1,51*	37,9 ± 1,29	37,4 ± 1,51	37,7 ± 0,75	38,0 ± 1,19	37,9 ± 0,64	
	38,6 ± 1,08	38,0 ± 1,19	37,5 ± 1,51	-	-	-	
(7) Статистическая работоспособность, сек.	фон (5)						
I 30	9,1 ± 0,75*	9,3 ± 0,75	9,8 ± 0,75	8,7 ± 0,43	8,6 ± 0,43	8,6 ± 0,86	
60	5,8 ± 0,99*	7,5 ± 1,08	9,9 ± 0,86	7,8 ± 0,54	8,7 ± 0,64	8,4 ± 1,08	
90	5,1 ± 0,64*	6,7 ± 1,25	10,5 ± 1,08	8,0 ± 0,64	9,2 ± 0,86	8,8 ± 1,08	
120	4,2 ± 0,43*	4,3 ± 0,43*	10,0 ± 0,64	7,7 ± 0,43	8,0 ± 0,43	8,9 ± 0,97	
II 30	4,1 ± 0,43*	4,1 ± 0,43*	9,5 ± 0,43	7,5 ± 0,54	7,7 ± 0,43	8,9 ± 0,97	
60	6,0 ± 0,64*	6,4 ± 0,75*	9,2 ± 0,64	-	-	-	
	6,3 ± 0,64	6,6 ± 0,64	9,3 ± 0,75	-	-	-	

\*--Statistically significant changes in the indices

- Key:
- Indices
  - Experimental period: I--EMF exposure, II--aftereffect, days
  - PFD, μW/cm<sup>2</sup>
  - Control
  - Background
  - Electrocuteaneous sensitivity threshold, volts
  - Static working capacity, sec

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Table 2. Conditioned Reflex Activity Indices for White Rats Exposed to a 9,400 MHz Pulsed EMF

(1) Показатели	(2) ППЗ, мкВт/см <sup>2</sup>	(3) Сроки эксперимента, сутки		
		воздействие фактора		
		60	90	120
(4) Величина латентного периода, сек.	40	0,97 ± 0,05	1,23 ± 0,17	1,17 ± 0,09*
	25	0,98 ± 0,09	1,32 ± 0,23	1,01 ± 0,07*
	10	1,07 ± 0,02	0,92 ± 0,05	0,89 ± 0,03
	(5) контроль	1,23 ± 0,18	0,99 ± 0,06	0,83 ± 0,02
(6) Величина двигательной реакции, усл.ед.	40	83,8 ± 2,7	93,6 ± 1,0*	96,7 ± 1,2
	25	87,8 ± 1,6	90,7 ± 2,2	94,5 ± 1,1
	10	89,9 ± 1,5	87,8 ± 1,82	95,9 ± 1,2
	(5) контроль	88,2 ± 1,3	87,0 ± 2,0	95,3 ± 1,2
(7) Величина латентного периода дифференцировочного торможения, сек.	40	1,27 ± 0,19*	-	4,9 ± 1,2*
	25	2,5 ± 0,6	-	5,65 ± 1,3
	10	2,25 ± 0,6	-	7,01 ± 1,10
	(5) контроль	3,5 ± 0,9	-	8,08 ± 1,03
(8) Величина двигательной реакции дифференцировочного торможения, усл.ед.	40	79,9 ± 3,4	-	53,0 ± 13,2
	25	78,7 ± 5,6	-	40,0 ± 12,7
	10	74,7 ± 6,2	-	39,7 ± 13,2
	(5) контроль	70,3 ± 9,1	-	24,8 ± 12,9

Key:

- |  |   |
|--|---|
| 1. Indices                                     | 5. Control  |
| 2. PFD, $\mu\text{w}/\text{cm}^2$              | 6. Magnitude of motor reaction, arbitrary units                 |
| 3. Experiment time, days of exposure to factor | 7. Latent period of differentiated inhibition, sec              |
| 4. Latent period, sec                          | 8. Motor reaction of differentiated inhibition, arbitrary units |

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Table 3. State of Enzymatic Metabolic Processes in the Bodies of Animals Subjected to Long-Term Irradiation by a 9,400 MHz Pulsed EMF

Показатели (1)	ПДЭ мкВт/см <sup>2</sup> (2)	(3) Сроки эксперимента, сутки				(5) последствие		
		воздействие фактора (4)				I20	I30	
		I5	30	60	90			
(6) активность холинэстеразы на ЮСмл крови	II5	*144,57±6,65	*98,71±9,64	*108,55±5,96	II0,54±8,04	II6,84±9,99	II8,43±3,02	II8,57±4,86
	60	II86,29±6,65	II18,43±8,01	II06,77±7,4	II2,57±8,16	II16,84±4,99	II23,71±4,84	II25,29±5,74
	контроль (7)	II19,87±3,6	II21,0±2,36	II21,7±3,8	II18±5,4	II28,5±4,8	II21,7±2,7	II29±4,2
(8) активность цитохромк- сидазы, ΔЕ на 1 мг бел-ка митохондрий	II5	*2,15±0,14	*2,08±0,12	*2,07±0,12	I,97±0,12	I,89±0,09	I,51±0,09	I,53±0,08
	60	*2,06±0,1	*1,97±0,11	*1,94±0,11	I,96±0,11	I,88±0,08	I,51±0,08	I,63±0,11
	контроль (7)	I,68±0,09	I,67±0,08	I,7±0,1	I,68±0,09	I,67±0,08	I,6±0,10	I,61±0,1
	II5	I,8±0,15	I,91±0,23	I,9±0,19	I,9±0,19	I,91±0,1	I,85±0,12	I,9±0,1
	II0	I,87±0,18	I,84±0,21	I,75±0,18	I,84±0,21	I,75±0,18	I,76±0,15	I,8±0,15
	контроль (7)	I,75±0,136	I,8±0,16	I,75±0,16	I,84±0,16	I,8±0,16	I,8±0,15	I,82±0,16
(9) содержание церулоплазмина в сыворотки крови усл. ед.	II5	*0,71±0,023	*0,7±0,08	*0,67±0,04	*0,63±0,04	*0,66±0,044	*0,54±0,03	*0,54±0,03
	60	*0,65±0,04	*0,66±0,02	*0,63±0,03	*0,62±0,02	*0,64±0,02	*0,56±0,027	*0,54±0,02
	контроль (7)	0,54±0,012	0,52±0,01	0,51±0,02	0,52±0,01	0,52±0,02	0,54±0,03	0,55±0,03
	II40	0,59±1,4	0,56±1,62	0,58±1,51	0,6±1,73	0,56±1,62	0,55±1,5	0,57±1,2
	контроль (7)	0,55±1,94	0,56±1,73	0,54±1,83	0,57±1,73	0,56±1,73	0,57±0,09	0,56±0,07

\*--Statistically significant values

- Key:
- Indices
  - PFD, μw/cm<sup>2</sup>
  - Experiment time, days
  - Time of exposure to factor
  - Aftereffect period
  - Cholinesterase activity, μg/min, per 100 ml blood
  - Control
  - Cytochrome oxidase activity, ΔE per mg mitochondrial protein
  - Blood serum ceruloplasmin concentration, arbitrary units

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25  $\mu\text{w}/\text{cm}^2$ ) the decline in the activity of this enzyme was less pronounced, and it was observed not from the first days of exposure to the factor, as was the case with 115  $\mu\text{w}/\text{cm}^2$ , but only 30 days after the animals began to be irradiated.

Concurrently we studied the state of metalloenzymes, which play a significant role in oxidation-reduction and hormone processes. As we know, ceruloplasmin and transferrin are such metalloenzymes. We established that microwave electromagnetic fields with PFD's of 115 and 60  $\mu\text{w}/\text{cm}^2$  elicit an increase in the activity of ceruloplasmin in the blood serum of experimental animals throughout the entire period of their irradiation. The same PFD's elicited an increase in iron saturation of transferrin after 90 days of exposure of the animals. At lower intensities of the factor (40-5  $\mu\text{w}/\text{cm}^2$ ) changes were not observed in these two indicators.

The changes noted in metalloenzymes are apparently the result of redistribution of microelements in the body in response to the microwave electromagnetic field.

Among the metabolic processes having vitally important significance, protein metabolism offers special interest in connection with the action of microwaves.

Considering this, we studied the concentration of the end components of protein metabolism, urea and nitrogen residues, in the blood of the animals. The research results showed that a microwave electromagnetic field (PFD's of 115 and 60  $\mu\text{w}/\text{cm}^2$ ) caused an increase in the urea concentration in all periods of observation (see Table 4). At PFD's of 40-5  $\mu\text{w}/\text{cm}^2$  changes were not observed. In the aftereffect period the urea concentration in the blood of the animals was within the limits of the control values.

The concentration of residual nitrogen in the blood serum of the animals increased only in response to an electromagnetic field with a PFD of 115  $\mu\text{w}/\text{cm}^2$ , and in all of the other cases--that is, at lower intensities (60-5  $\mu\text{w}/\text{cm}^2$ ) this index did not exhibit any changes (see Table 4).

The data from these studies were supplemented by an investigation of carbohydrate metabolism, which was assessed from the concentration of glycogen in the liver of experimental animals. The research results showed that the liver glycogen level of the animals declines in response to the action of microwave EMF's with PFD's of 115-60  $\mu\text{w}/\text{cm}^2$ . Lower EMF intensities (40-5  $\mu\text{w}/\text{cm}^2$ ) did not elicit statistically significant changes in this index. The noted decline in the liver glycogen level attests to limitation of energy resources, and it is obviously one of the causes of functional disturbances resulting from the action of microwave EMF's. However, we should not forget that such changes may also occur as a compensatory reaction of the body to disturbances in the body's oxidative processes caused by an EMF.

In addition to performing the research described above, we studied the effect microwave EMF's have on the hypothalamus-hypophysis-adrenal system, the



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Table 4. Characteristics of Protein and Carbohydrate Metabolism in Animals Exposed to a Pulsed Microwave EMF (9,400 MHz)

(1) Изученные показатели	(2) ППС, мВт/см <sup>2</sup>	(3) Сроки эксперимента, сутки					(5) последствие
		15	30	60	90	120	
(8) Содержание мочевины, мг.% в сыворотке крови	II5	41,7 ± 3,17*	37,5 ± 3,05*	37,0 ± 2,12*	35,75 ± 2,38*	37,5 ± 2,57*	29,14 ± 1,51
	60	34,35 ± 2,49	35,38 ± 2,78*	33,25 ± 1,85	34,14 ± 2,12*	35,00 ± 2,72	28,86 ± 1,81
	контроль (11) 40	30,7 ± 1,08	29,8 ± 1,65	28,3 ± 1,9	29,6 ± 1,7	29,7 ± 1,2	29,0 ± 1,51
(9) Остаточный азот, мг.% в сыворотке крови	II5	30,51 ± 1,5*	29,86 ± 1,59*	28,5 ± 1,06*	27,88 ± 1,19*	28,79 ± 1,28	24,57 ± 0,76
	60	27,16 ± 1,24	27,62 ± 1,39	26,5 ± 1,06	26,79 ± 1,06	27,5 ± 1,36	24,0 ± 0,9
	контроль (11) 40	25,4 ± 0,7	24,15 ± 0,5	24,9 ± 0,5	24,8 ± 0,45	24,7 ± 0,4	24,15 ± 0,8
(10) Содержание гликогена в печени, мг.%	II5	885 ± 131,2*	852 ± 93,2*	950 ± 64,3*	979 ± 43,8*	1001 ± 52,9*	1231 ± 45,4
	60	1138 ± 79,8	1033 ± 73,2*	1006 ± 29,8*	1002 ± 60,8*	999 ± 55,9*	1191 ± 60,5
	40	1422 ± 79	1486 ± 71,4	1318 ± 81,2	1163 ± 75,7	1250 ± 89,2	1300 ± 77,9
(11) контроль	1270 ± 57	1310 ± 81,1	1305 ± 54	1472 ± 92	1265 ± 54	1223 ± 88,7	

\*--Statistically significant changes in the index

- Key:
- Indices studied
  - PPD, μw/cm<sup>2</sup>
  - Experiment time, days
  - Time of exposure to factor
  - Aftereffect period
  - Protein metabolism
  - Carbohydrate metabolism
  - Urea concentration, mg-percent, in blood serum
  - Residual nitrogen, mg-percent, in blood serum
  - Liver glycogen concentration, mg-percent
  - Control

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Table 5. Concentration of Catecholamines and ACTH in the Organs and Blood of Rats Exposed to a Pulsed Microwave EMF (9,400 MHz)

ППЗ, (1) мкВт/см <sup>2</sup>	Сроки (2) воздействия, сутки	(3) Адреналин		(6) Норадреналин		АКТГ в плазме крови (7)
		надпочечни- ках (4)	в ткани мозга (5)	в надпочечниках (4)	в ткани мозга (5)	
40	60	0,35 ± 0,02	0,153 ± 0,009	0,224 ± 0,016	0,203 ± 0,013	25,1 ± 1,77
	120	0,37 ± 0,012*	0,158 ± 0,006*	0,214 ± 0,018	0,18 ± 0,015	23,01 ± 2,07
25	60	0,34 ± 0,013	0,15 ± 0,007	0,245 ± 0,012	0,21 ± 0,01	26,7 ± 1,8
	120	0,36 ± 0,007*	0,155 ± 0,006*	0,228 ± 0,013	0,20 ± 0,015	24,8 ± 2,7
10	60	0,33 ± 0,013	0,13 ± 0,007	0,234 ± 0,013	0,208 ± 0,013	26,28 ± 2,12
	120	0,32 ± 0,01	0,14 ± 0,009	0,24 ± 0,012	0,195 ± 0,012	26,5 ± 1,8
(8) контроль	60	0,33 ± 0,015	0,14 ± 0,01	0,23 ± 0,015	0,20 ± 0,013	26,57 ± 2,12
	120	0,32 ± 0,016	0,13 ± 0,009	0,235 ± 0,012	0,21 ± 0,012	25,7 ± 0,81

\*--Statistically significant changes in the index

Key:

1. PFD, μw/cm<sup>2</sup>
2. Time of exposure, days
3. Epinephrine
4. In adrenal glands
5. In cerebral tissue
6. Norepinephrine
7. ACTH in blood plasma
8. Control

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Table 6. State of Immune Reactions of Animals Exposed to a Pulsed Microwave EMF (9,400 MHz)

Изученные показатели (1)	ППЭ, мВТ/см <sup>2</sup> (2)	(3) Сроки эксперимента, сутки					(5) последнее действие
		15	30	60	90	120	
(6) Титр компонента	II5	0,08 ± 0,004	0,11 ± 0,004	0,1 ± 0,004	0,13 ± 0,009	0,16 ± 0,01	0,1 ± 0,004
	60	0,1 ± 0,004	0,1 ± 0,004	0,11 ± 0,006	0,11 ± 0,004	0,11 ± 0,006	0,11 ± 0,004
(7) контроль		0,11 ± 0,006	0,1 ± 0,004	0,11 ± 0,004	0,11 ± 0,004	0,11 ± 0,004	0,11 ± 0,006
Дегрануляция базофилов	II5	10,7 ± 1,4	12,1 ± 2,26	14,1 ± 3,02*	13,5 ± 3,00*	17,3 ± 3,02*	9,3 ± 0,75
	60	6,4 ± 1,5	6,4 ± 2,2	22,1 ± 3,4*	22,1 ± 3,0*	15,7 ± 2,3*	14,5 ± 2,2
(7) контроль		7,1 ± 1,5	7,8 ± 2,3	8,5 ± 1,3	9,3 ± 1,3	11,2 ± 1,4	10,7 ± 1,5
	40	10 ± 2,16	18,5 ± 2,16*	16,0 ± 2,7*	15,0 ± 2,16*	21,0 ± 1,6*	10 ± 2,16
(7) контроль		9 ± 1,62	9,5 ± 1,62	8,0 ± 1,62	10,5 ± 1,08	13,0 ± 1,62	10,5 ± 1,08
Динамика образования	II5	2,6 ± 0,38	2,8 ± 0,35	2,7 ± 0,33	4,8 ± 0,7*	6,9 ± 1,17*	3,3 ± 0,34
	60	2,4 ± 0,3	2,8 ± 0,5	3,4 ± 0,21*	3,7 ± 0,42*	2,9 ± 0,36*	2,8 ± 0,48
(7) контроль		2,8 ± 0,2	2,2 ± 0,39	2,3 ± 0,3	2,4 ± 0,4	2,1 ± 0,3	2,5 ± 0,5

Key:

1. Indices studied
2. PFD, μW/cm<sup>2</sup>
3. Experiment time, days
4. Time of exposure to factor
5. Aftereffect period
6. Complement titer
7. Control
8. Basophil degranulation
9. Plaque formation dynamics

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state of which was assessed from the quantitative concentration of catecholamines (epinephrine and norepinephrine) in the adrenal glands and in brain tissue, from the activity of adrenocorticotrophic hormone (ACTH) in blood plasma, and from the concentration of ascorbic acid in the adrenal glands. The research results showed that microwave EMF's ( $40-25 \mu\text{w}/\text{cm}^2$ ) caused an increase in the epinephrine level in cerebral tissues and the adrenal glands after 120 days.

In this case the ACTH activity of plasma and the ascorbic acid level did not differ significantly from the control values. Such a response by the body attests to participation of the hypophyseoadrenal system, with specific humoral links taking part.

We turned a certain amount of attention in our experimental research to an integral index of the body's functional state--immunological reactivity, which was evaluated on the basis of some nonspecific and specific immunity reactions. The results of this research showed that under the influence of a microwave EMF (with a PFD of  $115 \mu\text{w}/\text{cm}^2$ ), the complement activity of blood serum in experimental animals exhibited phasal changes--stimulation of function alternated with its inhibition, which may be an indication of a certain functional stress placed upon the humoral link of natural immunity.

The simultaneous increase in basophil degranulation and plaque formation (see Table 6) permits the hypothesis that a microwave EMF with a frequency of 9,400 MHz has a sensitizing action on the body, resulting from development of autoallergic processes. Of special interest in this case is the earlier development of autoallergy in animals exposed to a low intensity EMF ( $40 \mu\text{w}/\text{cm}^2$ ). The biological experiment ended with pathomorphological analysis of internal organs; the results revealed insignificant changes in the microstructure of some organs in response to microwaves with a PFD of  $115 \mu\text{w}/\text{cm}^2$ . In particular we noted moderate plethora of the tissues of the brain and internal organs, and dystrophic changes in the liver.

Thus the research demonstrated that a pulsed 9,400 MHz microwave EMF is a biologically significant factor which, with systematic and lengthy exposure at an intensity above  $25 \mu\text{w}/\text{cm}^2$ , elicits changes on the part of physiological, biochemical, and immunological indicators of the body's functional state.  
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BIOLOGICAL ACTION OF AN INDUSTRIAL FREQUENCY (50 Hz) ELECTRIC FIELD

Unknown BIOLOGICHESKOYE DEYSTVIYE ELEKTRICHESKOGO POLYA PROMYSHLENNOY CHASTOTY (50 GTs) in Russian 1979 pp 1-6

[Preprint of article by M. G. Shandala, Yu. D. Dumanskiy, Ye. V. Prokhvatilo, I. P. Los', L. A. Tomashevskaya, L. G. Andriyenko, S. A. Lyubchenko, I. S. Bezdol'naya, and Yu. I. Vasilenko, Kiev Scientific Research Institute of General and Communal Hygiene imeni A. N. Marzeyev]

[Text] High and superhigh tension power transmission lines are one of the main sources of industrial frequency (50-60 Hz) electromagnetic field radiation. The quantity of such lines is increasing with every year in all countries of the world. This naturally means that man is being subjected more and more frequently to the action of industrial frequency electric fields. This is why this factor is doubtlessly interesting from the standpoint of its biological action.

Considering this, we studied the influence an industrial frequency electric field has on the animal body with the purpose of determining the biological significance of short-term exposure to this factor.

We devoted special attention in our experimental studies on animals to experimentally modeling the real conditions under which man is subjected to an electric field created by power transmission lines. The following had to be true to satisfy these conditions:

The electric field vector ( $E$ ) had to be oriented vertically in relation to the biological object;

electric contact between the animals and "ground" had to be reliable;

the distribution of the electric field within the zone in which the experimental animals were located had to be uniform.

These conditions were satisfied with a plane-parallel capacitor, the upper plate of which was under a potential and the lower plate of which was grounded. The distance between the capacitor plates was chosen with a consideration for

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the actual ratio between the height of the human body and the height at which power transmission line conductors are suspended. Basing ourselves on this, and considering that white rats were used as the experimental biological model, the distance chosen between the plates was 430 mm.

Field intensity ( $E$ ) was determined with the formula:

$$E = \frac{U}{d}, \text{ volts/meter,}$$

where  $U$  is the voltage applied to the upper plate of the capacitor and  $d$  is the distance between the capacitor's upper and lower plate.

The capacitor plate dimensions were selected such that the distribution of the electric field within the zone in which the experimental animals were located was practically uniform. Plates having 950 x 450 mm dimensions corresponded to these conditions.

Ten to thirteen experimental animals were placed in dielectric plastic containers with a metallic bottom; these containers were set on the lower "grounded" plate of the capacitor in such a way that contact between the experimental animals and the "ground" was reliable (see Figure) [figures not reproduced].

The experimental research was performed in a chronic experiment lasting 6 months (4 months exposure to the factor, and 2 months aftereffect period); the 400 white rats employed were subdivided into a number of experimental groups depending on voltage and the exposure conditions. Table 1 shows the experimental design.

Discontinuous exposure of experimental animals to the factor was achieved with the help of an automated system based on an automatic experimental program control block. The following indicators of body functional states were studied in relation to all experimental animals, including the controls:

physiological--summational threshold index, emotional excitability, static endurance, conditioned reflex activity (latent period and magnitude of motor reaction); biochemical--concentration of urea, residual nitrogen, and glucose in blood serum, of glycogen in the liver, and of ascorbic acid in the adrenal glands, and activity of enzymes in blood and mitochondria (cholinesterase, ceruloplasmin, transferrin, succinate dehydrogenase, and cytochrome oxidase); immunological--basophil degranulation (Shelley reaction), plaque formation ((Iyerne-Klemparskaya) reaction), and complement, lysozyme, and hemagglutinin titers; hematological--concentration of hemoglobin, erythrocytes, leukocytes, eosinophils, and reticulocytes, and the leukocyte formula.

At the end of the exposure period and during the aftereffect period the animals were killed, and the internal organs and brain were subjected to pathomorphological study.

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Table 1. Experimental Design

Серия (1)	Напряженность кВ/м (2)	Экспозиция мин. (3)	Продолжительность паузы, мин (4)	Общая продолжительность воздействия, мин/сутки (5)
I	20	5	120	20
	15	5	120	20
II	20	5	25	80
	15	5	25	80
	10	5	25	80
III	20	20	30	180
	15	20	30	180
IV	20	80	30	300
	15	80	30	300
	10	80	30	300

Key:

1. Series
2. Voltage, kv/m
3. Exposure time, min
4. Interval length, min
5. Total exposure time, min/day

An analysis of the results established that 15 and 20 kv/m industrial frequency electric fields elicit, under some exposure conditions, statistically significant changes in the functional state of the body. Thus the indicated intensities of the factor as a rule led to an increase in the summational threshold index and a decline in emotional excitability and static endurance in the third to fourth months when total daily exposure time was 300 minutes (Table 2).

In addition changes occurred in the pattern of conditioned reflex activity. In the first month of exposure these changes manifested themselves as an increase in the latent time of a positive conditioned reaction; it manifested itself in the third and fourth months as difficulty in differentiation (Table 2).

The summational threshold index also changed at an exposure time of 80 minutes ( $E = 20$  kv/m), beginning with the second month of exposure.

It should be noted that the observed changes in physiological indicators disappeared following 1 month of recovery--that is, 1 month after exposure was terminated. Statistically significant differences from control were not noted in relation to other analyzed parameters of the factor. Our study of the biochemical indicators of body functional state afforded a possibility

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Table 2. Physiological Indicators of the Functional State of White Rats Following Short-Term Exposure to an Industrial Frequency Electric Field ( $\bar{x} \pm Sx$ )

(1) Показатели	(2) Параметры ЭП E, кВ/м; t, мин.	(3) До(5) воздей- ствия	(6) В период воздействия через				(9) Последней С.г.ж(9) 1 месяц (7)
			(7) 1 месяц	(8) 2 месяца	(8) 3 месяца	(8) 4 месяца	
Суммарно-поро- говый показатель (10)	20 300 15 300 (11) контроль	38,0 ± 1,40 37,8 ± 1,51 37,9 ± 1,29 37,7 ± 1,08	44,6* ± 2,48 38,9 ± 1,29 38,0 ± 1,08 37,7 ± 1,08	44,8* ± 1,51 46,2* ± 1,19 42,1* ± 1,41 37,8 ± 0,97	51,6* ± 1,73 56,5* ± 2,38 47,8* ± 1,20 37,7 ± 0,64	38,2 ± 1,83 38,1 ± 1,19 38,0 ± 1,29 37,8 ± 0,86	
Статимическая выносливость, мин. (12)	20 15 300 (11) контроль	9,8 ± 0,97 9,8 ± 0,97 9,7 ± 0,97	8,3 ± 0,97 9,2 ± 1,08 10,0 ± 0,97	6,5* ± 0,86 7,5 ± 0,64 9,2 ± 0,75	5,8* ± 0,97 6,5* ± 0,86 9,4 ± 0,97	8,4 ± 0,86 8,2 ± 0,86 9,6 ± 0,86	
Латентный период условных рефлек- сов, сек (13) - на звонок(4) - на зуммер(15)	20 15 300 (11) контроль	3,4 ± 0,30 3,5 ± 0,20 3,0 ± 0,30	0,9 ± 0,15 2,0 ± 0,40 1,18 ± 0,30	0,65* ± 0,09 1,1 ± 0,31 1,27 ± 0,20	0,6* ± 0,18 0,98* ± 0,12 1,55 ± 0,13	0,7 ± 0,09 1,0 ± 0,16 0,77 ± 0,10 4,9* ± 0,50 5,2 ± 0,51 6,10 ± 0,60	
Величина двигатель- ных реакций, (16) условные единицы - на зуммер (15)	20 15 300 (11) контроль	8,3 ± 0,60 9,1 ± 0,60 7,9 ± 0,60	8,1 ± 0,50 7,7 ± 0,50 7,1 ± 0,60	8,7* ± 0,50 7,7 ± 0,50 6,3 ± 0,50	8,9* ± 0,30 3,2* ± 0,40 6,3 ± 0,70	4,0 ± 0,70 4,8 ± 0,70 4,6 ± 0,70	

Note: Asterisks denote statistically significant changes

[Key on following page]

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## Key:

- |                              |   |
|------------------------------|---|
| 1. Indicators                | 10. Summational threshold index, volts            |
| 2. Electric field parameters | 11. Control                                       |
| 3. $E$ , kv/m                | 12. Static endurance, min                         |
| 4. $t$ , min                 | 13. Latent time of conditioned reflexes, sec      |
| 5. Before exposure           | 14. To a bell                                     |
| 6. During exposure, after:   | 15. To a buzzer                                   |
| 7. Month                     | 16. Magnitude of motor reactions, arbitrary units |
| 8. Months                    |   |
| 9. Aftereffect period        |   |

for establishing a number of changes in the pattern of metabolic processes (Tables 3 and 4). Changes in blood biochemistry were typified by enlargement of the concentration of glucose, urea, and residual nitrogen, reduction of cholinesterase activity, and growth in ceruloplasmin and transferrin activity. In parallel we noted a decline in the hepatic glycogen concentration and change in the concentration of ascorbic acid in the adrenal gland and in the activity of cytochrome oxidase and succinate dehydrogenase in cerebral and hepatic mitochondria.

The noted changes in biochemical indicators were observed throughout the entire time of exposure to electric fields with intensities of 15 and 20 kv/m, where total daily exposure times were 80, 180, and 300 minutes, and they were typified by reversibility following termination of exposure.

Table 5 presents data on the immunological resistance of white rats subjected to short-term exposure to an industrial frequency electromagnetic field; these data show that this factor has an influence on some immunological indicators. In particular at electric field intensities of 15 and 20 kv/m (180 and 300 minute exposure times) a statistically significant increase is noted in the percentage of degranulated basophils (Shelley reaction) and in autoimmune hemolysis plaque formation ((Yyerne-Klemparskaya) reaction). We did not concurrently reveal significant changes on the part of the lysozyme and hemagglutinin titers following immunization, which was used as a functional load.

The decrease we established in complement titer (only on the seventh day following vaccination) (see Table 5) is probably associated with the effect of the electric field, and it may attest to a certain stress placed upon the nonspecific immunity of animals in a 15 kv/m field (300 minute exposure time).

Concurrently with studying the body's immunological resistance we studied the composition of peripheral blood which, as the experimental results showed, exhibited changes of different directions in the quantitative concentration of hemoglobin, eosinophils, and leukocytes (Table 6). However, these shifts were not beyond the limits of physiological variations, and therefore we obviously cannot interpret them as being biologically significant. An exception to

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Table 3. Protein and Carbohydrate Metabolism Indicators for White Rats Subjected to Short-Term Exposure to an Industrial Frequency Electric Field ( $\bar{x} \pm Sx$ )

Показатели	(1) : Параметры, ЭП(2) : E, кв/м ; t, мин ; (3)	(6) В период воздействия через				Последствие (7)
		1 месяц(7)	2 месяца (8); 3 месяца(8)	4 месяца(8)	1 месяц (7)	
Мочевина крови, мг % (10)	20	29,2 ± 1,40	33,5 ± 1,29**	38,4 ± 1,70**	38,2 ± 1,73**	38,6 ± 1,50**
	15	28,4 ± 1,60	33,6 ± 1,60*	38,5 ± 1,84**	4,08 ± 0,97**	39,5 ± 1,95**
	(11) контроль	28,1 ± 1,4	27,8 ± 1,19	28,7 ± 1,40	29,3 ± 1,20*	30,0 ± 1,50
	20		27,4 ± 0,82	37,2 ± 1,95**	35,3 ± 1,80*	34,3 ± 1,85*
	15		28,2 ± 0,60	36,1 ± 2,05**	37,0 ± 2,26*	36,5 ± 1,95**
(11) контроль	20	29,3 ± 0,72	38,3 ± 1,44**	36,0 ± 2,05*		31,2 ± 2,40
	15	28,5 ± 1,13	36,2 ± 1,74**	36,1 ± 2,46		31,0 ± 2,30
	20	28,7 ± 1,20	28,3 ± 1,23	29,0 ± 1,75		
	15		99,1 ± 5,70**	99,6 ± 3,57**	104,2 ± 4,12**	104,2 ± 4,11**
	20	76,2 ± 2,27	77,9 ± 3,78	102,1 ± 3,13**	104,8 ± 3,25**	86,2 ± 4,76
Глюкоза крови, мг % (12)	15	76,5 ± 2,92	78,5 ± 3,59	77,0 ± 2,16**	77,1 ± 2,70*	75,4 ± 2,70
	(11) контроль		75,2 ± 3,08	99,8 ± 6,47**	99,7 ± 5,34*	86,7 ± 4,72
	15		79,9 ± 2,80	100,1 ± 6,17*	86,7 ± 5,60	72,4 ± 2,5
	15		76,3 ± 2,8	104,3 ± 5,24*	90,5 ± 5,50*	87,8 ± 6,8
	20	78,0 ± 2,70	72,3 ± 2,36	72,9 ± 2,98		71,2 ± 2,80**
Гликоген печени, мг % (11) контроль (13)	80			1170,7 ± 76,3	1554,3 ± 86,2	
	20			4580,8 ± 8,16	1505,7 ± 63,5	
	300			1139,0 ± 42,21**	1990,0 ± 58,44	

Key: 1. Indicators 9. Aftereffect period  
 2. Electric field parameters 10. Blood urea, mg-percent  
 3. E, kv/m 11. Control  
 4. t, min 12. Blood glucose, mg-percent  
 13. Liver glycogen, mg-percent

\* P < 0,05 \*\* P < 0,01

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Table 4. State of Enzymatic-Metabolic Processes in the Body of White Rats Subjected to Short-term Exposure to an Industrial Frequency Electric Field ( $\bar{x} \pm \text{Sc}$ )

Показатели (1)	Параметры ЭП (2)		До (3) До (4) До (5) До (6) До (7) До (8) До (9) До (10) До (11) До (12)				Последние (13)		
	E, кВ/м (3)	t, мин. (4)	1 месяц (5)	2 месяца (6)	3 месяца (8)	4 месяца (9)	1 мес (11)	1 мес (12)	
Аскорбиновая кислота надпочечников, мг % (10) (11) контроль	20	80	263,2±19,74	117,2±3,03	144,1* ±4,76	105,5* ±5,80	105,5* ±3,68	110,5* ±4,87	124,3±3,57
Активность холинэстеразы крови, мкг/мин. (12) (11) контроль	20	300	162,6 ±3,18	121,6* ±5,96	124,6* ±5,55	128,1* ±5,14	156,2±4,3	148,7±5,96	158,0 ±3,59
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	15	80	152,4 ±6,37	124,7* ±5,55	124,0* ±5,24	126,4* ±5,55	148,7±5,96	158,0 ±3,59	158,0 ±3,59
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	20	80	0,52±0,021	0,63* ±0,027	0,62* ±0,019	0,66* ±0,02	0,63* ±0,017	0,53±0,016	0,52±0,022
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	15	80	0,50±0,021	0,60* ±0,02	0,61* ±0,024	0,61* ±0,019	0,62* ±0,027	0,52±0,022	0,52±0,022
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	20	300	0,55 ± 0,02	0,44* ±0,023	0,63* ±0,025	0,61* ±0,024	0,57±0,025	0,57±0,025	0,57±0,025
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	20	80	0,56 ± 0,021	0,53 ±0,027	0,54 ±0,021	0,54 ± 0,023	0,54 ± 0,023	0,54 ± 0,023	0,54 ± 0,023
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	15	80	0,114±0,003	0,13* ±0,005	0,13* ±0,005	0,12±0,005	0,13* ±0,006	0,12±0,003	0,12±0,003
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	20	300	0,12 ±0,004	0,12* ±0,006	0,14* ±0,006	0,13±0,005	0,12 ± 0,005	0,12±0,011	0,12±0,011
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	20	80	0,112±0,003	0,113 ±0,003	0,113 ±0,005	0,116±0,003	0,113± 0,003	0,115±0,003	0,115±0,003
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	20	80	1,23* ±0,038	1,61 ±0,01	1,48* ±0,043	1,59 ±0,048	1,51* ±0,053	1,27 ±0,03	1,44* ±0,048
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	20	300	1,48* ±0,043	1,59 ±0,048	1,51* ±0,053	1,27 ±0,03	1,44* ±0,048	1,53 ±0,045	1,30 ±0,03
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	20	80	1,51* ±0,053	1,27 ±0,03	1,44* ±0,048	1,53 ±0,045	1,30 ±0,03	1,30 ±0,03	1,30 ±0,03
Активность церулоплазмина крови, оп-тические единицы (13) (11) контроль	20	300	1,48* ±0,043	1,59 ±0,048	1,51* ±0,053	1,27 ±0,03	1,44* ±0,048	1,53 ±0,045	1,30 ±0,03

- Key:
1. Indicators
  2. Electric field parameters
  3. E, kv/m
  4. t, min
  5. Before exposure
  6. During exposure, after:
  7. Month
  8. Months
  9. Aftereffect period
  10. Adrenal gland ascorbic acid, mg-percent
  11. Control
  12. Blood cholinesterase activity, µg/min
  13. Blood ceruloplasmin activity, optical units
  14. Blood transferrin saturation by iron, arbitrary units
  15. Cytochrome oxidase activity, E550 per mg protein
  16. Succinate dehydrogenase activity, E600, per mg protein, in cerebral mitochondria

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Table 5. Immunological Indicators for White Rats Subjected to Short-Term Exposure to an Industrial Frequency Electric Field ( $x \pm St$ )

Показатели (1)	Э, кв/м (3)	В период воздействия через (8)				Последствия (6)	
		1 месяц (5)	2 месяца (7)	3 месяца (7)	4 месяца (7)		
Дегрануляция базофилов (реакция Шелли), % (9)	20	9,3 ± 1,40	12,27 ± 1,49	19,0 ± 1,08	14,0 ± 1,08	12,1 ± 2,27	
	80	11,4 ± 3,02	13,18 ± 1,49	20,5 ± 1,08	13,0 ± 1,08	11,4 ± 2,27	
	15	10,0 ± 1,50	9,5 ± 0,99	11,5 ± 2,16	10,0 ± 1,08	11,4 ± 1,50	
	(10) контроль	20	19,5 ± 2,16	19,5 ± 2,18	22,0 ± 2,16	20,5 ± 1,68	24,2 ± 2,65
Бляшкообразование (реакция Йерне-Клемпарской), % (11)	20	2,95 ± 0,35	3,84 ± 0,46	7,04 ± 0,84	6,04 ± 0,85	5,63 ± 0,72	
	80	3,12 ± 0,53	3,63 ± 0,58	7,01 ± 1,09	5,31 ± 0,47	6,65 ± 1,48	
	15	3,48 ± 0,58	3,06 ± 0,54	3,04 ± 0,58	2,91 ± 0,35	2,92 ± 0,57	
	(10) контроль	20	2,87 ± 0,40	3,16 ± 0,35	2,88 ± 0,38	3,86 ± 0,40	4,28 ± 0,53
Титр компонента после иммунизации, мл (12)	20	3,05 ± 0,50	2,83 ± 0,44	4,28 ± 0,59	4,34 ± 0,52		
	15	3,05 ± 0,38	2,94 ± 0,40	2,94 ± 0,36	2,77 ± 0,33		
	(10) контроль	15	0,068 ± 0,004	0,068 ± 0,004	0,068 ± 0,004	0,068 ± 0,004	
	20	0,070 ± 0,004	0,070 ± 0,004	0,070 ± 0,004	0,070 ± 0,004		

Key:

1. Indicators
2. Electric field parameters
3. E, kv/m
4. t, min
5. During exposure, after:
  6. Month
  7. Months
  8. Aftereffect period
9. Basophil degranulation (Shelley reaction), percent
10. Control
11. Plaques formation (Yverne-Klemparskaya) reaction, percent
12. Complement titer following immunization, ml
13. Seventh day following first vaccination

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Table 6. Composition of Peripheral Blood in White Rats Subjected to Short-Term Exposure to an Industrial Frequency Electric Field ( $\bar{x} \pm St$ )

Показатели (1)	E, кВ/м; t, мин (3)	Доза (2)	В период воздействия через (6)				Последнее значение (7)	
			1 месяц (7)	2 месяца (8)	3 месяца (8)	4 месяца (8)		
Гемоглобин, г % (10)	20	20	13,8 ± 0,28	13,7* ± 0,17	12,5 ± 0,17	12,5* ± 0,19	14,0* ± 0,08	12,8 ± 0,11
	80	80	14,3 ± 0,05	13,8* ± 0,07	13,7* ± 0,13	12,8 ± 0,05	14,1* ± 0,08	13,1 ± 0,11
	180	180		13,7 ± 0,20	13,4 ± 0,12	12,1* ± 0,16	13,3 ± 0,15	
	300	300		13,3 ± 0,18	14,0* ± 0,06	13,6 ± 0,21	11,9* ± 0,23	
(11) контроль			13,5 ± 0,26	13,2 ± 0,14	12,9 ± 0,18	12,9 ± 0,28		
Ретикулоциты (12)	20	180	62,0* ± 2,57	68,0 ± 8,98	32,0* ± 2,38	72,4* ± 4,33		
	20	300	69,0* ± 6,80	66,0 ± 5,74	60,3* ± 1,51	87,6* ± 3,17		
	15	300	68,0* ± 3,17	57,0 ± 2,38	65,0* ± 3,71	91,6* ± 2,91		
	10	300	65,0 ± 6,80	64,0 ± 6,36	73,0* ± 3,57	84,6* ± 3,05		
(11) контроль			51,0 ± 2,27	66,0 ± 4,41	48,0 ± 4,59	56,0 ± 2,29		
Эозинофилы (13)	20	20	100,6 ± 4,14	117,9 ± 4,06	94,6* ± 2,49	106,3 ± 4,73	110,6 ± 2,71	114,1* ± 3,78
	15	80	116,1 ± 1,84	119,8 ± 2,03	119,6 ± 0,89	106,3 ± 4,09	100,0* ± 4,73	125,9* ± 3,74
	15	180		116,1 ± 1,84	117,0* ± 3,72	112,0 ± 5,20	98,2 ± 3,79	
(11) контроль			117,0 ± 4,65	100,0 ± 4,41	107,0 ± 5,51	108,3 ± 4,41		
Лейкоциты (14)	20	20	7200 ± 66,2	7830 ± 184,0	7672* ± 120,1	7640* ± 183,9	7630 ± 97,4	6771 ± 211,6
	15	20	8037 ± 278,0	7760 ± 86,6	7745* ± 170,0	7395* ± 43,1	7710 ± 173,2	7057 ± 28,7
	(11) контроль		7950 ± 159,0	7500 ± 141,0	8415 ± 229,6	6660 ± 176,2	7566 ± 183,9	7214 ± 196,5

Note: Asterisks denote statistically significant changes

- Key:
1. Indicators
  2. Electric field parameters
  3. E, kv/m
  4. t, min
  5. Before exposure
  6. During exposure, after:
  7. Month
  8. Months
  9. Aftereffect period
  10. Hemoglobin, gm-percent
  11. Control
  12. Reticulocytes
  13. Eosinophils
  14. Leukocytes

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this may be found only with reticulocytes, the concentration of which in animals exposed to electric fields with intensities of 10,15, and 20 kv/m, where the exposure times were 180 and 300 minutes per day, increased noticeably toward the end of the experiment.

The experimental research culminated with histological study of individual tissues and organs taken from experimental and control animals. Data obtained both at the end of the exposure time and during the aftereffect period did not reveal any sort of changes significantly different from control.

Thus the research results showed that at exposure times of from 80 to 300 minutes per day, electric fields with intensities of 15 and 20 kv/m had an influence on the functional state of experimental animals, which was manifested as change in their physiological, biochemical, and immunological indicators. However, the observed changes were weakly expressed, and as a rule they were not beyond the bounds of physiological norms, for which reason they cannot be interpreted as pathological.  
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IMMUNOLOGICAL EFFECTS OF LOW MICROWAVE EXPOSURE

Unknown IMMUNOLOGICHESKIYE EFFEKTY VOZDEYSTVIYA MALIONTENSIVNOGO MIKROVOLNOVOGO OBLUCHENIYA in Russian 1979 pp 2-9

[Preprint of an article by M. G. Shandala, M. I. Rudnev, G. I. Vinogradov, N. G. Belonozhko, and N. M. Gonchar, Kiev Scientific Research Institute of General and Communal Hygiene imeni A. M. Marzeyev]

[Text] Study of the influence a microwave electromagnetic field has on the body's immune system, the normal operation of which insures genetic constancy of the internal environment, has important significance to the overall complex of research on the biological action of this form of radiation.

Though few in number, data have nevertheless been already published in the Soviet Union on the influence a low intensity electromagnetic field has on the body's immunological reactivity in experiments on animals. Thus Zalyubovskaya and Kiselev (2) showed that exposure of SVA line mice to millimeter radio waves at an intensity of  $1,000 \mu\text{w}/\text{cm}^2$  for 15 minutes in a period of 20 days causes a decrease in the leukocyte count of peripheral blood, a decline in the blood serum lysozyme and complement titers, a decline in the phagocytic activity of neutrophils, and inhibition of the skin's bactericidal properties. These data show that microwave irradiation of the body may disturb its nonspecific defenses. The authors also showed that the animals consequently exhibit a decline in resistance to infections and intoxications. The latter could be interpreted as a manifestation of disturbed specific immunity.

Research on the immunological effects of exposure to low intensity microwaves, conducted by the Kiev Scientific Reserach Institute of General and Communal Hygiene imeni A. M. Marzeyev, showed that at a current density of [one line not reproduced] months, and at intensities of 10, 50, and  $500 \mu\text{w}/\text{cm}^2$  for one month.

Recovery of altered functions was observed for 3 months after irradiation.

A load--repeated one-time irradiation of the animals at a PFD [power flux density] of  $500 \mu\text{w}/\text{cm}^2$  for 7 hours--was applied at the end of the aftereffect period with the objective of analyzing the compensatory and adaptive possibilities of the body's immune system, and the functional completeness of recovery.

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The immunological analyses were performed with animals in the experimental and control group both during the time of irradiation and following it. Analysis of the resulting data showed the following.

The group of white rats exposed to microwaves with a PFD of  $500 \mu\text{w}/\text{cm}^2$  exhibits significant inhibition of the capability lymphocytes have for being stimulated by phytohemagglutinin (PHA) (Figure 1) [figures not reproduced]. Thus by as early as the third day of exposure a certain decline in the quantity of blast cells was noted ( $29.0 \pm 3.78$  in experiment, as opposed to  $34.7 \pm 3.44$  in control). Next, after 7 days of irradiation the percentage of blasts declined to  $24.0 \pm 3.06$ , after 2 weeks it declined to  $8.9 \pm 1.78$ , and by the end of the third week it was  $8.8 \pm 1.3$ . The maximum decline in the quantity of blast-forming cells is observed at the end of the period of irradiation-- $7.9 \pm 1.85$  in experiment, and  $37.0 \pm 3.53$  in control. A certain rise in the blast transformation indicator is noted in the aftereffect period, but the percentage of stimulated lymphocytes never reached normal, even by the end of the recovery period: The number of blasts in the experimental group remains significantly lower than that in the control group of animals ( $27.8 \pm 2.52$  and  $34.3 \pm 3.31$ ).

A tendency for the number of macrophages to decline is noted throughout almost all of the stages of the reaction, but significant differences from intact animals were not observed in this regard.

The percentage of blast formation in a culture not containing PHA exposed to the same level of SHF energy is also low, though such changes were also observed in control.

The load applied at the end of the experiment showed that when greater requirements were imposed on the body (when the additional influence was applied), the experimental animals revealed fuller suppression of the activity of T-cells in comparison with the intact animal group ( $14.7 \pm 2.62$  in experiment,  $29.0 \pm 3.68$  in control).

Similar studies were conducted on animals exposed to microwave energy with a PFD of  $50 \mu\text{w}/\text{cm}^2$  (Figure 2). An analysis of the obtained data reveals that a significant decline in blast formation is observed in the experimental group, in comparison with the control group, by as early as on the third day of irradiation:  $17.6 \pm 3.44$ , as opposed to  $35.7 \pm 4.71$ . The percentage of blasts continues to decline toward the end of the first week of irradiation ( $14.6 \pm 2.81$ ), while in the control group it remains at its previous level. Following an exposure time of 2 weeks the quantity of blast cells declined significantly to  $13.6 \pm 2.78$ , a level which persists even after 3 weeks of irradiation ( $15.6 \pm 3.23$ ). At the end of the exposure period the percentage of blast-forming cells remains practically at the same level ( $14.5 \pm 3.29$ ). An increase in the percentage of blast-forming cells ( $21.3 \pm 2.98$ ) is observed after 30 days of the recovery period. Further recovery of the percentage of blast cells is noted after 2 months. Thus it increases to as much as  $30.3 \pm 3.64$  in the experimental group; compare this with the level in the control group-- $37.4 \pm 4.76$  ( $p < 0.05$ ). However, full recovery occurs only toward the end



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of a 3-month period. Moreover the load we used revealed this recovery to be functionally incomplete, since repeat exposure elicits a sharply pronounced [one line not reproduced] in irradiated animals, in comparison with the intact group.

A certain decline is observed in the percent concentration of cells in the macrophage series; however, these changes are not significant, remaining within the limits of physiological variation. Interesting data were obtained in an analysis of the indicators for blast transformation of lymphocytes in the peripheral blood of animals exposed to microwaves with a PFD of  $10 \mu\text{w}/\text{cm}^2$  (Figure 2). The experimental group did exhibit a decline in the quantity of blast-forming cells in comparison with control during irradiation, though the decrease was to a lesser degree. However, toward the end of the second month of the recovery period the blast transformation indicators returned to normal, and after 3 months of the aftereffect period the indicators remained at practically the same level. The load we used, which took the form of one-time exposure to SHF energy with a PFD of  $500 \mu\text{w}/\text{cm}^2$ , demonstrated complete recovery of the indicators; specifically, the data obtained following the load exhibited no significant differences between the experimental group and the control group ( $25.5 \pm 2.65$  and  $29.3 \pm 2.92$ ).

The results of research on the action of microwaves with PFD's of 1 and  $5 \mu\text{w}/\text{cm}^2$  in an exposure period of 3 months showed that such low intensities do not have a significant influence on the capability small lymphocytes have for being stimulated.

The additional load demonstrated that changes in the quantities of blast cells in experimental and control groups do not differ statistically. This indicates that microwaves of these intensities do not produce latent changes in immunocompetent cells responsible for the state of cellular immunity (Figure 3).

As was noted earlier, we also employed the spontaneous reaction of rosette formation in the thymus and spleen to evaluate the T-system of immunity. A comparison of the data obtained from comparing the lymphocyte blast transformation reaction and the rosette formation reaction would show that the changes occurring in the body's immune system in response to microwaves proceed in the same direction. The significant changes observed include statistically significant inhibition of the function of immunocompetent cells, the extent of this inhibition being directly dependent on the power flux density.

Thus microwave radiation with intensities of  $500$  and  $50 \mu\text{w}/\text{cm}^2$  causes arisal of immunological deficiency in the thymus-dependent lymphocyte population. Changes arising at a PFD of  $10 \mu\text{w}/\text{cm}^2$  can be classified as compensatory, since we observe full recovery in the course of 3 months following irradiation. Microwaves with intensities of  $5$  and  $1 \mu\text{w}/\text{cm}^2$  do not elicit significant disturbances in the reactions of cellular immunity.

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These data agree with our previous research (7), and they permit the hypothesis that when the T-system of immunity is deficient, B-cells begin to react to tissue antigens, producing antibodies which promote development of an auto-immune state in response to microwave radiation with PFD's of  $50 \mu\text{w}/\text{cm}^2$  and higher.

We attach important significance, in an integrated assessment of the influence of environmental factors on the body's immunological reactivity, to the functional state of white blood cells participating in immune defenses.

It was in this connection that we undertook research aimed at studying the biologically active components of neutrophils, which play an important role in cell metabolism, immunogenesis, and adaptation. This research was conducted on white mongrel rats, in the experimental conditions described above. In our assessment of the concentration of glycogen and alkaline phosphatase we consider the mean cytochemical factor (MCF) and the percentage of positively reacting cells. The obtained data indicate that electromagnetic SHF fields with PFD's of 10 and  $50 \mu\text{w}/\text{cm}^2$  cause intensification of energy metabolism in neutrophils during the first 3 weeks of irradiation. Evidence of this can be found in the significant decline in concentration of alkaline phosphatase and glycogen in cells (figures 4, 5). The changes occurring in this period are obviously compensatory.

Further exposure causes a decline in the concentration of alkaline phosphatase, which is obviously the product of the inhibitory action of the SHF field on the activity of this enzyme following long-term exposure.

Microwaves with an intensity of  $500 \mu\text{w}/\text{cm}^2$  activate glycolysis in neutrophils, which leads to a decline in the concentration of glycogen in the cells by the end of the fourth week of irradiation.

Electromagnetic fields with PFD's of 1 and  $5 \mu\text{w}/\text{cm}^2$  do not influence the energy balance of white blood cells; all they do is alter phosphatase activity insignificantly (decrease it) in the first 30 days (figures 6, 7). Analysis of the concentration of glycogen and alkaline phosphatase in the neutrophils of white rats during a 3-month recovery period showed that at microwave energy levels of 1, 5, 10, and  $50 \mu\text{w}/\text{cm}^2$  the indicators do undergo recovery. It is only at a PFD of  $500 \mu\text{w}/\text{cm}^2$  that we observe a decline in the glycogen concentration and in phosphatase activity.

Considering the results of previous research on the action of low intensity superhigh frequency energy on carbohydrate metabolism in organs and tissues (5,6), and comparing these results with data from our own previous research, we can say that the changes exhibited by glycogen are associated with disturbances in enzymatic processes, and that they are a manifestation of an adaptive-compensatory reaction in response to disturbed oxidative phosphorylation. In this case the electromagnetic field plays the role of an uncoupling agent, preventing activation of enzymes responsible for glycogen synthesis and breakdown.

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Thus on generalizing the results of the research we can say that low intensity microwave radiation with PFD's of 500 and 50  $\mu\text{w}/\text{cm}^2$  is injurious to the body's immunological reactivity. Evidence of this can be found in inhibition of the thymus-dependent immunity system in response to these exposure conditions, manifested as arisal of secondary immunological deficiency. Lower intensities (10, 5, and 1  $\mu\text{w}/\text{cm}^2$ ) do not elicit such effects. The changes observed in this case are handled by immune defense mechanisms.

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DYNAMICS OF CHANGES IN AN ORGANISM'S BEHAVIORAL REACTIONS INDUCED BY  
MICROWAVE RADIATION

Unknown DINAMIKA IZMENENIY POVEDENCHESKIKH REAKTSIY ORGANIZMA, INDUTSIROVANNYKH  
MIKROVOLNOVOY RADIATSIYEV in Russian 1979 pp 1-13

[Preprint of article by Dr Med Sci M. I. Rudnev and Jr Scientist M. I.  
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[Text] The significance of behavioral reactions as the most sensitive indicators reflecting the state of the body as a whole is universally recognized today in research on the effects of unfavorable environmental factors. Most of the research has been performed in this regard on the action of superhigh frequency fields of thermal and subthermal intensities (6,7,10,12,15-20), presence of which can be said to be firmly established. At the same time a number of papers have been published attesting to possible behavioral influences by fields of lower intensities (1,3,5,11,14); however, the nature of changes occurring during irradiation and especially in the aftereffect period has not been clarified.

It was in this connection that we undertook research on the chronic influence of a low intensity SHF field (2,375 MHz) on animal behavior.

The research was conducted in three series of experiments on white mongrel rats 4-5 months old at the beginning of irradiation. Animals in series one were irradiated by SHF energy with a PFD of 500  $\mu\text{w}/\text{cm}^2$  for 1 month. Animals in the second series were exposed to PFD's of 50 and 10  $\mu\text{w}/\text{cm}^2$  for 3 months. In the third series the animals were exposed to PFD's of 5 and 1  $\mu\text{w}/\text{cm}^2$  for 3 months. The duration of daily irradiation was 7 hours during daylight hours. The animals were irradiated as groups within a field created inside an echoless chamber. Aftereffects were monitored for 3 months.

Methods

Defensive conditioned reflexes were studied in a maze of our own design. The device consists of three chambers located at the apexes of an equilateral triangle. Three corridors connect the chambers into a closed system with two

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exits in each chamber. The floor of the entire device is made from metallic rods (diameter--4 mm, intercenter spacing--1 cm). Pedals at the ends of the corridors turn switches on as the animal runs by. The walls of the device are made from white organic glass with a matte finish. The ceilings are transparent. Lamps (10 w) are installed outside the outer walls of the chambers, and a loudspeaker used to transmit the conditioning signals is installed in the center. Chamber height is 21 cm; the lengths of the side walls of the chamber are 24 and 30 cm. The length of the corridor's inner wall is 32 cm, and that of the outer wall is 23.5 cm; corridor breadth is 8 cm, and its height is 10 cm. The following conditioning procedure was employed: Upon transmission of the conditioning signal the animal had to run from the "start" chamber to either one of the other two. In the next trial that chamber in which the rat was located at this moment became the start chamber. If the rat failed to complete its run within 3.0 sec, a voltage was applied to the "start" chamber and to all corridors (100 Hz, pulse duration 1 msec, stabilized voltage). The applied voltage was 20 volts higher than the threshold inducing the animal to run. After the animal's run, the voltage was left on until the next conditioning signal only in the corridors. The conditioning signal consisted of a light beyond the wall of the chamber in which the rat was resting, and a sound (75 db, 500 Hz). Both stimuli were applied together for 6 sec. The interval between conditioning signals was 40 sec. The animals were exposed to radiation until five successive appearances of conditioned reflexes (attainment of a safe chamber in less than 3 sec), or for up to 90 trials a day.

To evaluate conditioned reflex activity we considered the mean latent time of the conditioned reactions ( $L_C$ ); the average running time along the corridor during performance of conditioned reactions ( $R_C$ ); the number of trials prior to appearance of five conditioned reflexes in succession ( $N_C$ ); the number of intersignal reactions occurring per trial (IR). An intersignal reaction was defined as a run from one chamber to another occurring not in response to transmission of a conditioning signal.

At each recording point we recorded indices twice, with an interval of a day, in an open field test (using a square 1 x 1 meter field containing 25 squares) consisting of three 1-minute trials with an interval of 15-20 sec. At the beginning of each trial the rat was placed in an opaque cube in the center of the field. The total number of squares crossed by the rat was recorded. In the first day of recording we interpreted this value as exploratory activity (EA) and in the second day we called it motor activity (MA).

The threshold of sensitivity to electrocutaneous stimulation (T) was determined in a cage with a floor consisting of metallic rods (diameter--2 mm, intercenter spacing--7 mm) from the amount of voltage (100 Hz, square pulse duration--1 msec) causing the rat to withdraw its forepaws from the floor.

To evaluate the aggressiveness of the animals we ascertained the outcome of a fight (the fight ended when one of the rats assumed a submissive posture)

32

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provoked by electric current in a chamber with a floor of metallic rods. Rats from the experimental group fought rats in the control group.

The parameters of the unconditioned dietary reaction we recorded included its latent time ( $L_d$ ) and the magnitude of the reaction ( $R_d$ ) (the quantity of food eaten in a 20-minute trial).

Dynamic working capacity ( $D_w$ ) was determined from the time the rat could maintain its position in a rotating cylinder (14 rpm; diameter--4.3 cm; lined with fabric). The time the rat could maintain its position on fabric-lined planks secured at a 30° angle to the vertical, 50 cm above the floor, was used as the magnitude of static working capacity ( $S_w$ ). Plank length was 40 cm, and its cross section was 1.5 x 1 cm, and 2.8 x 1.7 cm following the 90th day of the experiment. Trial time in the presence of a field with a PFD of 500  $\mu\text{w}/\text{cm}^2$  was 15 min, and after the 90th day it was 10 min. If the animals did not fall, the working capacity index was said to be 15 or 10 min correspondingly.

In all three series of experiments we selected out a group of females to be used in the open field test and in the threshold test, and a group of males for all the other tests. Another group of males was selected out to study conditioned reflexes in the presence of fields with PFD's of 500, 50, and 10  $\mu\text{w}/\text{cm}^2$ . Each series of experiments included a parallel control located in the same space in which the experimental animals were subjected to radiation. The groups contained 8-15 animals.

## Index Recording Schedule

All indices were recorded before irradiation, and the animals were redistributed to create homogeneous groups.

In the first series of experiments the conditioned reflex parameters were recorded on the 10th and 30th days of irradiation and on the 30th, 60th, and 90th days after irradiation. Parameters of all other tests were recorded on the 10th, 20th, and 30th days of irradiation and on the 15th, 30th, 45th, 60th, 75th, and 90th days after irradiation. In the second series we recorded conditioned reflexes at a monthly interval; unconditioned reflexes were recorded on the 10th, 20th, and 30th days of irradiation, and subsequently at a monthly interval. (The number of aggressiveness recording points was decreased, since the method was found to be insensitive). In the third series the conditioned defensive reflex, dietary behavior, and static working capacity indices were recorded at a monthly interval. The threshold and exploratory activity were additionally recorded on the 10th and 20th days of irradiation. Dynamic working capacity and aggressiveness were determined on the 90th day of irradiation and on the 90th day of the aftereffect period.

The time of daily irradiation was delayed by 3-4 hours in connection with the recording session, and those animals that were developing conditioned reflexes were exempted from irradiation.

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## Treatment of the Experimental Data

Prior to the experiment the animals were redistributed into groups in such a way that they did not differ from the control group by more than

$$\sqrt{M_c^2 + M_e^2}$$

where  $M_c$  and  $M_e$  are the mean errors for the control and experimental groups prior to irradiation. If we were unable to satisfy this condition in relation to all parameters, the figures for the particular rats were discarded, and thus the conditions of the statistical tests applied were met for all parameters in relation to all recording points.

Prior to statistical treatment, all deviating trials were discarded with the use of special tables (2,8). Inasmuch as indices were recorded in the experimental and control groups at the same time by alternating the animals, our analysis made use of a variant of the sign test in which the signs were related in time. We used the  $U$  test (Wilcoxon-Mann-Whitney) to evaluate conditioned reflex activity; Fisher's formula for a four-field table was used in the analysis of working capacity. Wherever the text makes reference to a test for differences, we used Student's  $t$ -test. Differences were said to be significant at  $p < 0.05$ .

## Research Results

## Series I

Analysis of changes in exploratory activity demonstrated that this index was highly sensitive to an EMF with a PFD of 500  $\mu\text{w}/\text{cm}^2$ . Exploratory activity was found to be inhibited on the 20th and 30th days of irradiation (according to the sign test). Inhibition was superceded in the aftereffect period by activation (arousal) of exploratory activity (a significantly higher level was noted in the experimental group on the 30th, 45th, 60th, and 90th days after irradiation). Relative deviations ( $\Delta M\%$ ) of the experimental group from the control group and the error of these deviations ( $m\%$ ) were computed with the following formulas in order to gain a more graphical impression of the parameter's changes:

$$\Delta M\% = \frac{M_e - M_c}{M_c} \times 100\%$$

$$m\% = \frac{\sqrt{(m_e^2 + m_c^2)}}{M_c} \times 100\%$$

where  $M_e$  and  $M_c$  are the means for the experimental and control groups, and  $m_e$  and  $m_c$  are the mean errors. Using these values, we can easily compute parameter  $t$  to evaluate differences in the means with Student's test:

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$$t = \Delta M\%/m\%$$

Data on relative change in exploratory activity are shown in Figure 1 [figures not reproduced].

In contrast to exploratory activity, significant change (growth) in motor activity was not noted until the 90th day after irradiation.

The electrocutaneous stimulation threshold was found to be one of the most sensitive parameters. During the period of irradiation, we recorded a significant decline in the threshold on the 30th day and an increase on the 20th day. In the aftereffect period we recorded a decrease on the 30th day (according to the sign test) and on the 60th day. The dynamics of the process are shown in Figure 2, which is set up in the same way as Figure 1.

Feeding behavior exhibited significant signs of inhibition both in the irradiation period (enlargement of  $L_d$  and reduction of  $R_d$  on the 10th day) and after irradiation (enlargement of  $L_d$  on the 15th and 30th days, and reduction of  $R_d$  on the 60th day after irradiation).

The aggressive behavior of the animals and the conditioned reflex activity parameters hardly changed at all. A significant decline was noted in the proportion of intersignal reactions on the 10th day of irradiation, and  $R_c$  was noted to decline on the 90th day after irradiation. Data describing the dynamics of the rate of conditioned reflex development and intersignal reactions are presented in figures 3 and 4.

The working capacity of the animals changed (decreased) only in response to irradiation: Dynamic working capacity declined on the 10th and 20th days, and static working capacity declined on the 10th and 30th days.

#### Series II

The functional state of the central nervous system also underwent inhibition when rats were irradiated with fields having PFD's of 50 and 10  $\mu\text{w}/\text{cm}^2$ .

A significant decline in exploratory activity was noted with both intensities on the 30th and 60th days of irradiation. Although there were no significant differences in the aftereffect period, the average value for exploratory activity of the irradiated groups attests to a certain amount of excitation of the central nervous system (see Figure 1). No changes were recorded in the motor activity parameter.

Changes occurring in the sensitivity threshold indicate presence of a central nervous system arousal phase in the initial period of irradiation. A decline was noted in the threshold on the 10th day at a PFD of 50  $\mu\text{w}/\text{cm}^2$  and on the 10th and 20th days at a PFD of 10  $\mu\text{w}/\text{cm}^2$  (see Figure 2). At the end of the exposure period the threshold was significantly higher with a PFD of 50  $\mu\text{w}/\text{cm}^2$ --that is, the central nervous system was inhibited.

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Dietary behavior underwent inhibition during irradiation, and it was reactivated following it. At a PFD of  $50 \mu\text{w}/\text{cm}^2$  the  $L_d$  decreased on the 30th day of the aftereffect period; at a PFD of  $10 \mu\text{w}/\text{cm}^2$  it increased on the 30th day of irradiation. On the 30th day of irradiation the  $R_c$  decreased in the presence of both intensities, and it decreased on the 30th day of the aftereffect period at a PFD of  $50 \mu\text{w}/\text{cm}^2$ .

$D_w$  was high on the 60th day of irradiation at a PFD of  $50 \mu\text{w}/\text{cm}^2$ . The  $S_w$  of the same group increased on the 90th day of irradiation and on the 30th day of the aftereffect period; it was lower on the 60th day of the aftereffect.  $S_w$  increased for the group irradiated with a PFD of  $10 \mu\text{w}/\text{cm}^2$  on the 60th day of irradiation and the 30th day of the aftereffect period.

The described intensities of the SHF field elicit clearly pronounced inhibition of conditioned reflex reaction, which persisted even after irradiation. At a PFD of  $50 \mu\text{w}/\text{cm}^2$  the  $N_c$  parameter was significantly larger on the 60th (according to the  $U$  test) and 90th days of irradiation, and on the 30th and 90th (according to the  $U$  test, for the range below 23 trials) days of the aftereffect period. The PFD of  $10 \mu\text{w}/\text{cm}^2$  reduced the rate of reflex development (it increased parameter  $N_c$ ) on the 90th day of irradiation. The number of intersignal reactions also changed in parallel. When the rats were irradiated with a field having a PFD of  $50 \mu\text{w}/\text{cm}^2$ , IR decreased on the 90th day of irradiation, and on the 30th and 60th days following irradiation. At the PFD of  $10 \mu\text{w}/\text{cm}^2$  IR declined on the 30th and 90th days of irradiation and on the 30th day of the aftereffect period. In addition  $L_c$  increased on the 60th day of irradiation with a field of the same intensity (see figures 3 and 4).

The rest of the parameters did not change significantly.

## Series III

The intensities of SHF energy used in this series had a less pronounced effect on behavior; an SHF field with a PFD of  $5 \mu\text{w}/\text{cm}^2$  inhibited exploratory activity on the 90th day and motor activity on the 60th day of irradiation. It reduced the threshold on the 90th day following irradiation,  $S_w$  on the 90th day, and  $L_d$  on the 30th day of irradiation. Conditioned reflex activity was inhibited ( $N_c$  increased) on the 60th and 90th days of the period of irradiation; by the 60th day after irradiation, meanwhile, the corridor running time ( $R_c$ ) decreased.

Very few changes occurred in response to a PFD of  $1 \mu\text{w}/\text{cm}^2$ . On the 30th day of irradiation the latent time of the conditioned reflex decreased; on the 60th day the exploratory activity declined and  $N_c$  increased. By the 30th day after irradiation the number of intersignal reactions exhibited an increase.

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## Discussion

Data on significant differences are summarized in Figure 5 in order to provide a better impression of the overall nature of behavioral changes occurring in response to all three intensities of the SHF field. This figure shows five zones corresponding to each intensity. A parameter symbol located above the day on which the index was recorded means a significantly larger value than control, and a parameter symbol below the recording day means a decline relative to the level for the control group.

As we can see, the number of significant differences decreases as the field intensity drops. However, if we look at individual parameters, we find that the extent of these changes (the significant ones) hardly depends at all on field intensity.

In our experiments even very low field intensities elicited some changes in behavioral reactions. Such results might have been mistrusted not that long ago. However, we have now accumulated evidence of the possibility of such influence upon the body, both in the USSR (1,3,5) and in the USA (13), where changes in the aggressive behavior of animals were noted during irradiation by pulsed microwaves with an average PFD of  $5 \mu\text{w}/\text{cm}^2$ .

An analysis of the distribution of significant differences reveals certain features in the nature of the field's actions at different periods of time. If we assume growth in latent times and thresholds and reduction of activity, working capacity, rate of reflex development, the number of intersignal reactions, and reaction magnitudes to be signs of overall inhibition of the central nervous system, and the opposite changes to be signs of arousal, then, utilizing these concepts, we can isolate a number of periods (stages) in the state of the central nervous system.

Thus at a PFD of  $500 \mu\text{w}/\text{cm}^2$  almost all changes occurring in the irradiation period attest to inhibition of the central nervous system, while those occurring after irradiation indicate arousal. An exception to this can be found in the decline in thresholds on the 30th day of irradiation, the increase in  $L_d$  on the 50th and 30th days after irradiation, and the decrease in  $R_d$  on the 60th day after irradiation. At intensities of  $1-50 \mu\text{w}/\text{cm}^2$  certain signs of arousal appear at the beginning of irradiation. However, the only significant changes in threshold occur with PFD's of 10 and  $50 \mu\text{w}/\text{cm}^2$ , since this index exhibits a regular decline in the presence of an aroused central nervous system at a PFD of  $500 \mu\text{w}/\text{cm}^2$ . The direction of changes in  $L_d$  is often not associated with the state of the central nervous system, and  $L_c$  changed so rarely in other cases that it would be better not to make any hasty conclusions concerning the presence of an arousal phase on the 30th day in the presence of PFD's of 5 and  $1 \mu\text{w}/\text{cm}^2$ .

Signs of arousal were revealed in the aftereffect period for PFD's of 5 and  $1 \mu\text{w}/\text{cm}^2$ , and, in relation to unconditioned reflex indices, for PFD's of 50 and  $10 \mu\text{w}/\text{cm}^2$ . An exception is the decrease in  $S_w$  on the 60th day at a PFD of

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50  $\mu\text{w}/\text{cm}^2$ . Concerning the groups of animals which underwent development of conditioned reflexes and which were irradiated by fields having intensities of 50 and 10  $\mu\text{w}/\text{cm}^2$ , their central nervous system activity was clearly inhibited. Apparently the test requiring compulsory development of a defensive reflex in the irradiation period was so difficult for the nervous systems of these animals, weakened by irradiation, that their conditions deteriorated, higher nervous activity failed, and signs of arousal did not appear in these groups.

Thus we can distinguish a phase of central nervous system arousal after irradiation, an inhibition phase developing toward the end of irradiation, and an arousal phase in the initial period of irradiation. Earlier recording of indices at the 500  $\mu\text{w}/\text{cm}^2$  intensity would also have indicated presence of an arousal phase. Presence of a certain number of exceptions can obviously be explained by the fact that this factor may influence both the parameter being recorded itself and the mobility of the animal. Its influence may increase the working capacity of an animal, but due to simultaneous growth in mobility the animal may fall from the plank earlier, thus creating the appearance of reduced working capacity.

Presence of phases in reactions of the central nervous system to effects of different intensities and durations was observed by many authors (9), and hypotheses have been suggested (4) on the role of these changes in non-specific adaptive reactions, one of them being Selye's stress reaction.

We also believe that at least some of the changes in functional state of the central nervous system have such a mechanism behind them.

Figure Legends [figures not included]

Figure 1. Action of SHF Energy on Exploratory Activity.

Figure 2. Effect of an SHF Field on the Electrocutaneous Threshold.

Figure 3. Effect of an SHF Field on the Number of Trials Required to Develop a Reflex ( $M \pm m$ ): Number of animals is shown inside the columns. Above: First columns--control, second columns--exposure to a PFD of 500  $\mu\text{w}/\text{cm}^2$ . In the middle: First columns--control; second columns--exposure to 10  $\mu\text{w}/\text{cm}^2$ ; third columns--50  $\mu\text{w}/\text{cm}^2$ . Below: First columns--control; second columns--1  $\mu\text{w}/\text{cm}^2$ ; third columns--5  $\mu\text{w}/\text{cm}^2$ .

Figure 4. Effect of SHF Energy on Intersignal Reactions. Indices are plotted as in Figure 3.

Figure 5. General Nature of the Action of an SHF Field.

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THE EFFECTS OF INJURY AND RESTORATION OF THE ORGANISM OF RATS UNDER MICROWAVE IRRADIATION (2400 MHz)

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[Article by V. S. Tikhonchuk, Biophysics Institute, USSR Academy of Sciences, Moscow]

[Key Words: injury; restoration; species sensitivity; microwave irradiation.]

[Text] The pathophysiological shifts arising in rats under intensive microwave irradiation leading to their deaths have already been the subject of experimental investigation (1, 2, 6, 7, 9). The influence of the irradiation schedule on the time of onset of the lethal outcome has been established (8).

The present investigation further examines the general tendencies of the formation of the processes of injury and restoration and the correlation between them in rats and compares these data with results previously obtained on mice (3, 5).

#### Methods of Investigation

Two thousand seventy-two mongrel female rats with an average mass of 220±12 g were used in the experiments. The animals were irradiated in an anechoic chamber with microwaves (2400 MHz) at a power density (PD) of 60 to 800 mW/cm<sup>2</sup> and an ambient temperature of 20-22 degrees C. The nonuniformity of the experimental microwave field was no greater than 2 dB. The death of the animals was studied. The experimental distributions obtained were expressed algebraically.

#### The Results of Investigation

Within the experimental range of the PD (800-60 mW/cm<sup>2</sup>) the experimental distributions of the effects of the death of the rats from the time of microwave irradiation (fig 1) are sufficiently well described by an equation in the form (test samples):  $Y=10.5566 \cdot X+6.0070$ ;  $Y=20.3105 \cdot X+2.0128$ ;

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$Y=69.7311 \cdot X+20.7204$ ;  $Y=20.4726 \cdot X-6.6568$ ;  $Y=15.6766 \cdot X-8.6621$ ;  $Y=15.2329 \cdot X-14.1035$ ;  $Y=18.1849 \cdot X-22.7886$ , where  $Y$ =the effect of death (test samples),  $X$ =log of the irradiation time (in minutes). As in mice (5) with an increase in PD the time of balanced effect increases and the distribution flattens out. The presence of these two tendencies determines the exponential character of the functional dependences between PD and the time of microwave irradiation for balanced effects, for example: 0.1, 50, 99.9 percent:  $\log Y=2.6338-0.6918 \log X$ ;  $\log Y=2.7790-0.6741 \log X$ ;  $\log Y=2.9257-0.6549 \log X$ , respectively, where  $\log Y$  is PD (in  $\text{mW}/\text{cm}^2$ ) and  $\log X$  is the time of microwave irradiation (in minutes). Analysis of the functional dependences obtained indicates the close correlation of the frequency characteristics of the effects of injury and restoration, their dependence on PD and the time of microwave irradiation.

The presence of the restorative reaction with an increase in the time interval between single irradiations was previously demonstrated in mice (3) and rats (4). For rats, with a PD equal to 800, 500, 300, 200 and 100  $\text{mW}/\text{cm}^2$  the dependence between the lethal effects and the interval between single irradiations in minutes was interpolated by means of the following equation (test samples):  $Y=8.4830 X-5.0486$ ;  $Y=5.4680 X-0.1803$ ;  $Y=6.9626 X+0.1669$ ;  $Y=6.8968 X+0.4743$ ;  $Y=7.2205 X+0.9657$ ; respectively where  $Y$  is the survival rate ( $v$  probitakh) and  $X$  is the log of the irradiation time (in minutes).

At the compared levels of injury and restoration effects, for example 0.1 percent injury and 99.9 percent restoration, 50 percent injury and 50 percent restoration, the correlation of the rate of development of these tendencies is a function of the PD of the microwave irradiation and can be described by an equation of the exponential function (fig 2):  $\log Y=2.7613+1.7690 \log X$  and  $\log Y=3.7714+1.8749 \log X$ , respectively. As in mice, this correlation is greater in absolute value the greater the PD and the lower the level of injury; the distinction is the parallelism of lines 1 and 2 (see fig 2).

When the rates of the processes of injury and restoration are equal ( $\log (v \text{ injury}/v \text{ restor})=0$ ), the value of PD (the projection of points A and B on the abscissa, see fig 2) is equal to 102 and 36.4  $\text{mW}/\text{cm}^2$ . In the latter case, the death rate of the animals did not exceed 0.1 percent (was practically equal to zero). Thus, in rats at values of PD equal to or less than 40  $\text{mW}/\text{cm}^2$  the correlation of the rates of injury and restoration should satisfy the condition:  $\log (v \text{ injury}/v \text{ restor} \leq 0) =$  the projection of points C, D, E, F, G on the ordinate (see fig 2; when PD equals 10, 1,  $10^{-1}$ ,  $10^{-2}$ ,  $10^{-3}$   $\text{mW}/\text{cm}^2$ ). This indicates the increasing significance of the restorative processes compared to the effects of injury when the PD of the microwave irradiation decreases and formally explains the data previously obtained (8).

The results earlier obtained (2-5) and the experimental facts reported in the present work make it possible to evaluate and compare in first approximation some general tendencies of the formation of the processes of injury and restoration and the correlation between them in two species of animals--mice and rats.

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For each of these species it is possible to determine the threshold values of PD and time of microwave irradiation at which the lethal effect does not exceed 0.1 percent. The dependence between these two values may be interpolated from an exponential function of the form:  $\log Y = 2.4333 - 0.7408 \log X$  (for mice) and  $\log Y = 2.7613 + 1.7690 \log X$  (for rats). At values of PD greater than  $40 \text{ mW/cm}^2$  and irradiation time values exceeding the threshold values, a function of the type: effect (death) = irradiation time (when PD = const) is revealed. Analysis of the curves of equal effect (death) formally indicates the presence of the restorative reactions during microwave irradiation, a fact which has been confirmed experimentally (3, 4). The rates of formation of injury and the restorative reactions are of a diverse nature when the effects of injury and restoration are comparable. When correlated together they are a function of the PD of the microwave irradiation. Mice are the more sensitive animals when compared with rats with respect to the time parameters of the onset of equal effects of injury and restoration.

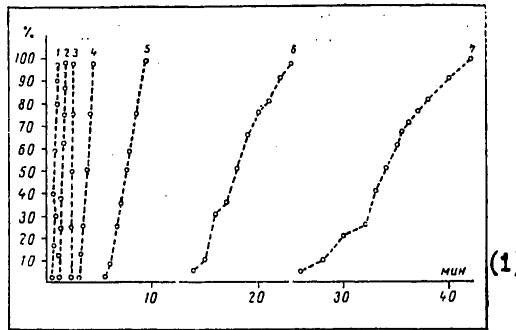


Figure 1. Death of Rats as Function of Irradiation Time  
 Abscissa=irradiation time (in minutes); ordinate=death of rats (in percentage).  
 1-7= experimental distributions where PD equals 800, 500, 300, 200, 100, 80 and 60  $\text{mW/cm}^2$ , respectively.

Key:  
 1. minutes



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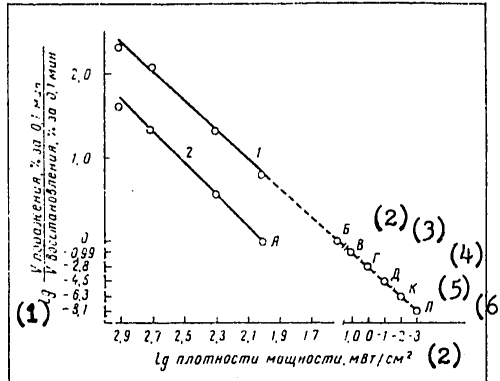


Figure 2. The Relation of the Rate of Injury to the Rate of Restoration as a Function of the PD of the Microwave Irradiation  
 1,2= levels of injury and restoration equal to 0.1 and 99.9 percent and 50 and 50 percent, respectively.

- Key:
1.  $\log \frac{V \text{ injury, percent in 0.1 minutes}}{V \text{ restoration, percent in 0.1 minute}}$
  2.  $\log \text{ power density, mW/cm}^2$
  3. B
  4. C
  5. D
  6. E
  7. F
  8. G

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KINEMATIC DESCRIPTION OF A HELICAL FRAME OF REFERENCE SYSTEM IN THE SPECIAL THEORY OF RELATIVITY

Tomsk IZVESTIYA VYSSHIKH UCHEBNIKH ZAVEDENIY - FIZIKA in Russian No 12, 1978 p 126 article deposited at VINITI, No 3213-78, 1978

IVANOV, V. G. and LAPKOVSKIY, A. K.

[Text] This work, which uses relativistic kinematic equations to a great extent, gives kinematic description of a frame of reference system  $\Sigma$  in the special theory of relativity in which the reference bodies projected on Euclidean subspace move along helical lines (with a pitch  $h$ ). Such a frame of reference system  $\Sigma$  may be realized by noninteracting charges in a permanent magnetic field (without taking into account the force of friction). At  $h = 0$  we achieve the rotating reference system, which is of great interest. All the invariant forms and the absolute invariants of 4 velocities of the reference bodies are found; their geometric interpretations are given.  
[132-P]

CSO: 1840-P

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UDC 530.12;531.51

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RELATIVISTIC KINEMATIC EQUATIONS AND THE THEORY OF CONTINUOUS MEDIA

Tomsk IZVESTIYA VYSSHIKH UCHEBNYKH ZAVEDENIY - FIZIKA in Russian No 6, 1978  
p 155 article deposited at VINITI, No 553-78, 1978

Lapkovskiy, A. K.

[Text] The new approach to the theory of continuous media in the special theory of relativity presented in the paper is based on relativistic kinematic equations (obtained on an algebraic basis). The discussion is carried out in a nonholonomic attached reference frame with rotation. Problems of relativistic rotation and relative velocities and accelerations are analyzed and a rigorous basis is provided for the concept of a small particle of the medium. The passage in the limit to Newtonian theory is carried out.  
[131-P]

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