The results obtained suggest that substitution therapy with thyroid hormones in the treatment of autoimmune thyroiditis has a significant effect on the improvement of indicators and stabilization of patients' health status. This method of treatment allowed to regulate thyroid hormone levels in the blood to physiological parameters and to lower the antibodies to thyroperoxidase level to a minimum level. When 50 μg of the drug "L-thyroxine" was taken, antibodies to thyroperoxidase were present in the blood, with an increase in the dose of up to 75 μg of these antibodies in the blood was not detected.

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MITOGEN-INDUCED PROLIFERATION OF MEMORY T-CELLS IN PATIENTS WITH MULTIPLE SCLEROSIS AND PARKINSON'S DISEASE

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There have been active studies of memory T-cells and their subsets to determine the properties, functional activity and role in maintaining of autoimmune and neurodegenerative reactions in humans.

Keywords: Parkinson's disease, multiple sclerosis, neurodegeneration, neuroinflammation, memory T-cells, central nervous system.

Neurodegenerative diseases (NDD) are a group of the nervous system diseases characterized by neuronal loss that leads to severe neurological symptoms [1].Two most common NDD are the Parkinson's disease (PD), a progressing disease, characterized by the destruction and death of dopaminergic neurons; and multiple sclerosis (MS), a demyelinating disease driven by autoimmune inflammation [2]. In the formation of neurodegeneration the key role belongs to neuroinflammation and adaptive immune reactions, including memory T-cells, study the functions of which may lead to understanding of diseases' pathogenesis.

The aim. To characterize the functional potential of memory T-cells subsets in patients with MS and PD.

Materials and methods. The material was whole peripheral venous blood obtained from 21 MS patients (10 men and 11 women, 29.5 [23.0 ÷ 33.0] y.o.), 6PDpatients(4 men and 2 women, 61.2 [57.0 ÷ 65.0] y.o.) and 14 healthy donors (8 men and 6 women, 29.0 [24.0 ÷ 45.0] y.o.). The level of disability on EDSS scale in MS patients was 3.0 [2.0 ÷ 3.5]scoresand on the Hoehn and Yahr scale in PD patients – 2.5 [2.0 ÷ 3.0] scores. The proliferation of T-cells subpopulations was determined by flow cytometrymethod using monoclonal antibodies CD3-PC7, CD4-PC5, CD8-FITC, CD45RO-ECD, CCR7-PE and flow cytometer CytomicsFC500. Mitogen-induced proliferation of lymphocytes was assessed using CFSE-method and 2.5 μg/ml of phytohemagglutinin (PHA).The main subpopulations of memory T-cells were identified as central memory $CD3^+CCR7^+CD45RO^+$T-cells(TCM), effector memory $CD3^+CCR7^-CD45RO^+$T-cells (TEM) and terminally differentiated memory effector $CD3^+CCR7^-CD45RO^-$T-cells (TEMRA). Statistical processing of data was carried out using the standard Statistica8.0.

Results. MS and PD patients showed a tendency to increase the number of memory T-cells in peripheral blood compared to the control group ($p<0.05$), while the total number of $CD3^+$T-cells and their major subsets did not change statistically. In both groups the relative amount of TEM was increased compared to ones in healthy donors ($p<0.05$). Moreover, the number of TEM exceeded TCM count in MS patients(18.9 (13.3÷22.8)*vs* 14.7 (11.8÷25.1))as well as in PD patients(17.4 (13.1÷22.2)*vs* 14.8 (8.5÷22.6)), while in the group of healthy donors a subpopulation of TCM was prevailed ($p<0.05$)(12.6 (7.2÷20.3) TEM *vs* 19.2 (13.4÷26.0) TCM).After 6 days of cultivation the up-regulation of spontaneous and PHA-stimulated TEM (8.0 (6.1÷13.2) vs. 59.2 (55.2÷63.4)and TEMRA (5.9 (4.9÷7.2) vs. 65.9 (46.8÷84.6))proliferation were established in PD patients– mainly due to $CD4^+$ and to a less extent of $CD8^+$T-cells subsets ($p<0.05$) as well as in MS patients (25.6 (15.6÷35.7) *vs*89.3 (88.0÷90.5) TEM and 32.3 (23.5÷41.2) *vs*90.0 (89.3÷90.75) TEMRA) – conversely due to $CD8^+$ and to a less extent of $CD4^+$T-cells subsets ($p<0.05$) compared to healthy donors in which the basic composition wasdetermined by TCM.Moreover, in NDD patients the tendency in differentiation of memory T-cells toTEMRA was registered.

Conclusion. The differences in alterations of memory T-cells subsets in neurodegenerative diseases are revealed: MS and PD patients have increased number of TEM with prevalence of TEMRA cellswhile healthy donors are dominated by TCM. The results of this study confirm the pathogenic role of TEM and TEMRA cells in NDD patients and what may be used as laboratory criteria for the development of the neurodegeneration as well as autoimmune reactions and monitoring the effectiveness of NDD treatment.

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STUDY OF GENETIC MARKERS OF RADIOSENSITIVITY AS A WAY TO IMPROVE HUMAN SAFETY IN CONTACT WITH IONIZING RADIATION

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Now, genetic markers of radiosensitivity acquire great urgency. They allow assessing the resistance of the organism at the cellular level. In general, they can used for setting norms in cases of forced contact with radioactive radiation.

Keywords: radiation, radiosensitivity, genes, organisms, risk, emergence of, role, human, impact, activities, individual, ionizing, research, cells.

As known, the problem of radiosensitivity is one of the central problems of radiobiology. Radiosensitivity is the susceptibility of organisms to the effects of ionizing radiation, the ability to react to it in a certain way. It is an individual feature of the body and depends on many factors. This problem is multifaceted and can be considered at both micro and macro levels.

According to the literature, the most promising areas of radiation genetics is the study of individual human radiosensitivity at the cellular level.

The strength and duration of ionizing effects play a key role in the occurrence of effects in the human body.

Thus, in individuals with chronic exposure, the risk of malignant tumors increases, the number of cells with a cell cycle block increases, and TCR mutations increase, and the frequency of chromosomal aberrations increases.

Radiation-induced instability of the genome can lead to the emergence of distant effects such as the occurrence of genetic changes in descendants of irradiated cells.

There are DNA repair systems in the human body that serve as a protective mechanism for radiation exposure. It used correctly; these systems can prevent malignant transformation of cells.

Not one work is devoted to the topic of genetic markers of radiosensitivity. Among the candidate radiosensitivity genes, according to various studies, there are: reparation genes (RAD51, RAD52, XRCC4, XRCC1, XRCC5, XPG, XPD, OGG1, BRCA1, BRCA2, LIG4, PRKDC, DCLRE1); cell cycle and apoptosis control genes (TP53, ATM, ATR, Nbc1, NF-kB, c-jun, Erg-1); gene responsible for the metabolism of nitric oxide and the induction of mechanisms of radio protection (NOS), genes for detoxification of xenobiotics (CYP, GST, NAT).

Research of the role of these genes in the realization of radiosensitivity is important in connection with the increasing role of radio emission in our daily lives.

The greatest danger of radiation damage exists for people who are directly exposed to radiation because of their professional activities. Radiation risk caused by the activity of enterprises of the nuclear industry and energy, the functioning of radiation research laboratories, medical institutions of radiation diagnostics and treatment, etc.