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THE RELATIONSHIP OF STRESS FACTORS AND THYMUS

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The article discusses the problem of the interrelationships of the nervous, endocrine and immune systems on the basis of literature and own data. In this case, the thymus is considered not only from the standpoint of the immune system, but also as an endocrine gland that allows for neuroimmunoendocrine interactions. Thymus polypeptides have an effect not only on the immune system, but also have a stress-protective effect.

Key words: stress, lymphocytes, neuroimmunoendocrine system, thymus peptides.

Currently, the problem of stress is of concern not only to specialists, but also to society as a whole. In modern conditions, stress is understood as a condition that occurs when the body is exposed to emergency or pathological stimuli called stressors, and leads to stress of its nonspecific mechanisms [1, 2]. The hypothalamus, pituitary gland, adrenal glands and the autonomic nervous system (ANS) are among the mandatory participants in stress [4].

The body responds to stress with a triad of symptoms: an increase in the cortical layer of the adrenal glands with an increase in its activity, a decrease in the thymus and lymph nodes, the appearance of spot hemorrhages, erosions and ulcers on the mucous membrane of the stomach and intestines. If the stressor is not strong enough, and the body's resistance is high, then these pathological changes may not be detected [4]. With prolonged exposure to a stressor, an adaptive reaction of the body occurs, called by G. Selye the general adaptation syndrome, which stereotypically proceeds in three stages: anxiety, resistance and exhaustion [3].

The first stage (anxiety) develops within 6 hours and lasts 1-2 days. At this stage, due to the involvement of the hypothalamic-pituitary-adrenal system in the process, urgent adaptation mechanisms are triggered. The highest regulator coordinating the system of endocrine organs and metabolism with the work of the ANS and integrative emotional and behavioral reactions is the hypothalamic-pituitary neurosecretory complex. There is every reason to consider the hypothalamus as the main element coordinating biologically expedient behavior; it is here that the affective component of sensations and reactions is formed. In the lateral parts of the hypothalamus, near the centers of hunger and thirst, there is a center of rage (anger), the irritation of which

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provokes an aggressive emotional and motor reaction and severe stress. Not far from it, in the periventricular nuclei, there is a punishment center, the irritation of which provokes fear, displeasure, causes severe pain and an active avoidance reaction; with prolonged exposure, debilitating stress occurs, which can lead to the death of the body. The punishment center transmits activating effects to the center of rage, which makes possible an active external response of the animal – defense and avoidance. If the external motor-emotional response coordinated by the anger center turns out to be impossible or limited, then this leads to pathological changes in internal organs such as exhaustion or "adaptation disease" [5]. There is also a pleasure center (or reward, satisfaction) in the hypothalamus. It is located in the ventromedial and lateral nuclei along the central bundle of nerve fibers and is represented by noradrenergic fibers. The pleasure center calms stress. It is closely related to the centers of satiety and sexual desire. Having the opportunity to self-stimulate this center, animals feel an irresistible attraction to it [4]. Under stress, the hypothalamus, activated by factors acting both humorally (interleukins, etc.) and cholinergic and serotonergic influences emanating from the limbic and other parts of the central nervous system, already at the beginning of the anxiety stage leads to an increase in corticoliberin production. The latter, entering the adenohypophysis through the local portal system, stimulates the production of ACTH. In turn, corticoliberin and a short peptide from ACTH stimulate the center of anxiety and fear in the limbic system, creating a characteristic emotional background. ACTH stimulates the adrenal gland, promoting the production of catecholamines – hormones of fight and flight [4]. However, along with them, a large amount of glucocorticosteroids (GCS) enters the bloodstream, and the metabolism of RNA, proteins and other substances increases in the central nervous system. At this stage, the body's energy consumption is largely due to the excitation of the central nervous system and the sympathoadrenal system. Compared to the energy consumption during physical work, energy consumption during stress is significantly higher. The stage of anxiety is characterized by pronounced emotions (more often negative); dissimilation processes prevail in metabolism [1, 2]. The next stage, the resistance stage, is one of the types of long–term adaptation to a constantly acting stressor. It is based on cellular mechanisms of adaptation and formation of the corresponding functional system. The duration of the resistance stage can be calculated in days and weeks. The elements of increasing resistance under the prolonged action of a stressor are an increase in the synthesis of RNA and some proteins, mobilization of the immune system (IS), an increase in the production of GCS stimulating gluconeogenesis [2]. The stage of resistance may be absent in the presence of functional insufficiency of the hypothalamic-pituitary or sympathoadrenal systems (for example, in persons with lymphatic-hypoplastic diathesis). With the reversible nature of hormonal and metabolic shifts occurring at the stage of resistance without severe exhaustion, stress

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may develop along the path of normalization [1]. With the incessant effect of a stressor on the body, adaptation is inhibited, and the last stage begins – the stage of exhaustion. It is not detected in all cases, but only when exposed to a very strong or prolonged effect of a weak stimulus that exceeds the functional reserves of the neuroendocrine apparatus of the individual. In case of termination of the stressor's effect on the body, the outcome of this phase is normalization of the condition, with continued exposure, the most likely outcome is death [1, 2]. Thus, when studying stress, attention was drawn to the fact that stress is associated with homeostasis mechanisms, and corticotropin plays a leading role in the implementation of this process. To date, the activities of the cardiovascular system, respiratory system, and gastrointestinal tract have been well studied in the implementation of stress, but clearly not enough attention is paid to IS. At the same time, from the very beginning of the stress study, G. Selye, along with an increase in the adrenal glands, observed a decrease in the mass and size of the thymus [3]. Here, however, it should be noted that 30 years before the work of G. Selye, the German morphologist J. Hammar described a decrease in the thymus in children under the influence of infections, calling this condition accidental, i.e. accidental (Latin accident – chance), thymus involution [6]. Subsequently, J. Hammar himself and other researchers observed the phenomenon of thymus involution both in infectious diseases and as a result of a number of effects on humans and animals (injuries, starvation, hypothermia, physical exertion, strong emotions, metabolic disorders, hypoxia, therapy with GCS and cytostatics, X-ray irradiation, etc.) [7-14]. In the end, it became clear that this process does not develop by chance, but naturally, and only the reason that caused the thymus involution is accidental. This thymus reaction is most pronounced in children. It should be noted that at the time when the theory of adaptation syndrome began to develop, there was still no information about the thymus as the central organ of IP. Currently, no one denies the fact that the thymus is an organ in which the reproduction and maturation of T–lymphocytes - cells that carry out cellular immunity reactions - takes place. In the second half of the twentieth century, it was firmly established that lymphocytes are a heterogeneous group of cells, including B, T and NK cells. All of them and their subpopulations differ from each other only with the help of special (immunological) research methods. However, despite the intensive development of immunology and immunological research methods, peripheral blood tests with the determination of the main types of leukocytes in stained smears, introduced into practice back in the 30s of the twentieth century, still remains relevant. This affordable, cheap and minimally invasive method can be used as a screening test to determine the indications for a special immunological examination of the patient. However, the lymphatic system is directly connected to the venous bed, and therefore the peripheral blood reflects the changes occurring in the system of lymphoid organs. This makes it possible to regard peripheral blood as an indicator of the level of

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lymphocytes in the body. Summarizing the available information allows us to present this process in the form of a sequential change of 5 phases. The 1st phase was initially presented as a resting thymus, however, by the works of M.P. Yelshanskaya [8], N.A. Yurina and L.S. Rumyantseva [9], as well as a number of other researchers, have shown that she is characterized by an increase in organ mass compared to the age norm by 50% or more [11, 12, 14]. This is associated with the accumulation of a significant number of lymphoblasts in the subcapsular zone of the thymus (which is probably due to antigenic stimulation). This condition can be regarded as an attempt to compensate for the incipient organ dysfunction. In addition, at the very beginning of this process, increased blood filling of the thymus and its edema occur. All this taken together leads to an increase in the organ. Phase 2 marks the beginning of involutive changes and is characterized by the migration of macrophages into the cortical zone of the thymus, which carry out phagocytosis of T-lymphocyte breakdown products. Electron microscopically, this is documented by the detection of macrophages in the cytoplasm, fragments of destroyed cells, phagolysis and accumulation of RAS-positive and sudanophilic inclusions in it; at the light level, the adhesion of lymphocytes to the surface of macrophages is detected [12]. The occurrence of such phenomena in the thymus indicates the 2nd phase of accidental involution. Light microscopy in this phase reveals a picture of the so-called "starry sky" [9, 12, 19, 20]. In general, phase 2 is characterized by the onset of extensive involutive changes, but the mass of the thymus does not noticeably decrease during this phase. The latter is associated with the not yet completely extinguished process of lymphocyte proliferation, increased influx of macrophages, as well as with edema of the stroma and pronounced fullness of the organ. Such changes occur, for example, in children suffering from a lightning-fast form of meningococcemia, acute fast-flowing pneumonia, sepsis, who die in the first 24-48 hours of the disease [12]. The 3rd phase of accidental involution is documented by the processes of increasing involution. The most characteristic feature of this phase is the inversion of layers, resulting from a significant emptying of the thymus cortex, and an increase in the number of thymic bodies, which are localized not only in the brain area, but also spread beyond it. In phase 4, not only the cortical, but also the medullary zone is emptied; at the light-optical level, this phase is characterized by the disappearance of the inversion of layers. Thymic lobules take the form of homogeneous formations consisting of epithelial cells. Only a few lymphocytes remain, which probably belong to the cortisol-resistant population. Within the intra-lobular perivascular spaces, the number of B lymphocytes and plasma cells increases. In the 5th phase, coarsening and collagenization of the stroma increase; the capsule and large vessels, as a rule, are sclerosed. Lipomatosis develops in the interlobular stroma. In general, the 4th and 5th stages are close and differ in the degree of sclerosis of the thymus stroma and the reversibility of the process [12].

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Moreover, it has been found that emotional stress can induce genetic damage in the reproductive and somatic cells of mammals, similar to those after physical and chemical influences [35, 36]. Comparing the achievements of immunology with the theory of general adaptation syndrome under stress, N. Dourov proposed the following concept of the formation of accidental thymus involution [37]. From the point of view of this researcher, the leading role in the development of accidental involution is played by GCS, which cause an increasing alteration of cortisol-sensitive lymphocytes, which make up 70-80% of the total number of thymocytes. These primarily include subpopulations of small immature T lymphocytes localized in the subcortical zone of the thymus in "nurse" cells. Under the influence of GCS, certain enzyme systems are activated in such lymphocytes, which leads either to the death of most of the lymphocytes by apoptosis, or to rapid differentiation and a decrease in sensitivity to steroid and thymic hormones [38-41].

One of the first was the publication of Yu.I. Zimin [42], who established an increased propensity of animals exposed to stressors to diseases caused by Herpes simplex viruses, polio, Coxsackie B, polioma and pathogens of other infections. Moreover, these animals showed an elongation of exposure to delayed hypersensitivity reactions and a decrease in resistance to tumor growth. This indicates that stress is accompanied by the formation of an individual's immunodeficiency. In most cases, immunosuppression under stress is associated with an increase in the concentration of GCS in the blood and the redistribution of lymphocytes [43]. The information available to date on the relationship between the adrenal cortex and the thymus suggests that a decrease in the body's resistance to various stressors is associated not so much with an increase in the level of GCS, but with a lack of functional activity of the thymus. The GCS and the functional capabilities of the thymus are in an antagonistic relationship.

In acute stress in the anxiety stage, increased proliferation of myeloid cells and an increase in the number of neutrophils per unit volume of blood are noted. This is due to the entry of mature bone marrow cells into the bloodstream, as well as as a result of neutrophils entering the bloodstream from the parietal pool located in the vessels [46]. Lymphocytopenia in acute stress is usually relative [47]. Neutrophilic leukocytosis in acute stress can also develop due to other causes. It was stated above that the function of the adrenal glands is controlled by ACTH. This pituitary hormone, intensively synthesized under stress, stimulates the adrenal glands, leading to an increase in the concentration of both catecholamines and GCS in the blood. The latter contribute to the apoptosis of cortisol-sensitive lymphocytes.

There are two groups of factors that can cause stress-induced immune suppression. Firstly, such a situation can occur with direct activation of the neuroendocrine axis due to an increase in the production of corticotropin-releasing hormone (CRH) by the hypothalamus. Exposure to toxic substances and emotional

stress on the body belongs to this group. Secondly, indirect activation of the neuroendocrine axis may occur, which, for example, can occur in infectious diseases and unbalanced nutrition [50]. Of the toxic stress effects, the effect of opiates has been studied better than others.

Thus, monitoring the level of lymphocytes can contribute to the diagnosis of stress in general and its stage in particular. As a screening test, it is advisable to evaluate the leukocyte formula at least on the first, 2-3 and 7-10 days after exposure to a stressor, and subsequently determine the number and ratio of CD4/ CD8 lymphocytes. The detection of gross violations can probably be corrected with the help of thymus polypeptides.

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