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The Effect of Stress on the Morphology of the Thyroid Gland

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Abstract: the article writes about the morphological changes that occur in the thyroid gland when the body is exposed to short-term and long-term stress. Under the influence of cold, the thyroid mass of rats increased. In the thyroid tissue, the volume of follicular epithelium increased and the volume of colloid decreased. Structural transformations of the follicles were accompanied by an increase in the volume of the vascular bed and the number of degranulated mast cells in the connective tissue stroma of the thyroid gland. It was concluded that under the influence of extremely low temperatures, the functional activity of the thyroid gland in rats increases, despite the depletion of the body's adaptive reactions.

Key words: stress, thyroid gland, thyroid hormones, absorption and organization of iodine, deiodinases.

Relevance. Stress is an integral part of human life these days. However, this does not make the consequences of chronic stress on our body any less dire. Stress affects all organs and systems, and one of its main targets is the thyroid gland, an organ that regulates most metabolic processes in our body. How exactly stress affects the thyroid gland and how to prevent serious health consequences is discussed in the article. As soon as we encounter stress, the "stress system" is activated in our body. From this moment on, it is its mechanisms that begin to regulate the functioning of organs and systems in the body, including the organs of the endocrine system. The first thing to change is the functioning of the endocrine glands. The brain gives a command to increased synthesis of stress hormones: Glucocorticoids in the adrenal cortex - the hormones cortisol and corticosterone; Catecholamines in the adrenal medulla are the hormones adrenaline and norepinephrine. With short-term stress on the body, the thyroid gland is stimulated by adrenaline and norepinephrine. Such exposure does not lead to the development of pathologies: it is only aimed at mobilizing the body's internal resources. With chronic stress, a completely different picture emerges. If stress persists for a long time, adrenaline and norepinephrine are replaced by cortisol, which gradually begins to suppress the function of the thyroid gland. Finally, prolonged exposure to stress on the thyroid gland can cause the development of inflammatory processes in the thyroid tissue. The immune system begins to mistakenly perceive this inflammation as a threat, and its cells begin to actively attack the thyroid tissue, causing autoimmune thyroiditis (AIT). In recent years, the number of research studies devoted to the study of the effects of stress on thyroid function and peripheral metabolism of thyroid hormones has increased significantly. This may be due, apparently, to several components. On the one hand, the growth of thyroid pathology in many countries of the world against the background of improved iodine prophylaxis and the search for factors that modify iodine metabolism in the thyroid gland. On the other hand, one cannot help but note the increased tension in the life of the individual and

society as a whole (psychological, social, environmental and other types of stress). In fact, social development has created a new habitat for humans with an increased level of stress factors. The consequences of its impact are both adaptation of the body and maladaptation and irreversible pathological changes. According to the definition of the founder of the science of stress (from the English stress - tension) Hans Selye, "Stress is a nonspecific reaction of the body to any strong irritation." It is the consequence of stress exposure that leads to the development of a general adaptation syndrome and the body's resistance to stress. The first studies on the effects of stress on thyroid function date back to the 50s of the last century and are devoted to the influence of stress on the morphology of the thyroid gland, glucocorticoid regulation of the GGT axis at the level of the hypothalamic and pituitary structures, and the study of thyroid status. Today, the study of the role of glucocorticoid hormones in the regulation of key stages of thyroid cell metabolism, as well as the mechanisms of development of stress-induced thyroid pathology and its prevention, remains relevant. Thyroid hormones take an active part in the formation of the body's adaptive response to the action of various environmental factors. Studies have shown that the pituitary-thyroid system is involved in the stress response already in the early stages and, according to this author, plays an important role in the development of the general adaptation syndrome described by G. Selye. At the same time, the hormonal activity of the thyroid gland (TG) is largely determined by the nature, nature and intensity of the effect. However, the question of the patterns of changes in the functional reserves of the thyroid gland depending on the specifics of the current stress factor remains unresolved. It has been shown that short-term immobilization causes a significant increase in the secretion of thyroid hormones by the thyroid gland, but already one day after the onset of immobilization stress, according to morphometry data in animals, a decrease in the synthetic activity of thyrocytes is detected. Under conditions of long-term expectancy stress, in rats of various strains selected for the excitability of the nervous system, the hormone-synthetic activity of the thyroid gland decreases and signs of its hypofunction appear with the restoration of the morphofunctional state 2 weeks after the end of the neurotic influence. A morphological study of the thyroid gland in rabbits with parallel measurement of the level of hormones in the blood under conditions of prolonged emotional-painful stress with increasing intensity of effects showed that with stress depletion of the body's adaptive reactions, a parallel decrease in the secretory activity of the thyroid gland is observed.

Purpose of the study is to study the patterns of adaptive morphogenesis of the thyroid gland and its functional pattern under the conditions of the experimental model "Thyroid gland-stress".

To achieve this goal, the following tasks were identified:

- study the micromorphology of the thyroid parenchyma of the thyroid gland in rats of the study groups;
- study the histology of the thyroid parenchyma of the thyroid gland in rats of the study groups;

Materials and research methods

The object of the study was the thyroid gland of clinically healthy white outbred male rats (weighing 300+50 g) at the age of 3 months. All animals were kept in the same conditions, received a standard vivarium diet, and had access to food and drinking water ad libitum. All laboratory animals were obtained from the same vivarium and were carried out on albino rats aged 4-5 months. Adult (4-5 month old) white outbred rats were kept in standard vivarium conditions with relative humidity (50-60%), temperature (19-22°C) and light regime (12 hours of darkness and 12 hours of light). In order to prevent and ensure the absence of infectious diseases in the vivarium, laboratory animals were quarantined for 21 days and observed during these days, their temperature was taken and their weight was checked several times during these days. The growth was tracked.

During this period, they did not experience any symptoms of the disease, the temperature was within normal limits (38.5-39.5°C), appetite disturbances and other external changes were not detected. For the experiment, a control and two experimental groups were formed. Hypothyroidism was experimentally induced in rats of experimental groups I and II. To simulate hypofunction of the thyroid gland, the classic thyreostatic agent "Tyrozol®" (H03BB02 - Thiamazol) was used. The drug was diluted with water for injection and administered intragastrically using a tube at a dose of 2.5 mg per 100 g of animal weight for 28 days. On the 10th day, the animals were removed from the experiment under ether anesthesia in compliance with the rules of euthanasia, and autopsy material was collected for subsequent histological examination (thyroid gland). Autopsy material was marked, fixed in 10% buffered formalin, and subjected to histological examination using standard histological techniques. Quantitative (morphometric) analysis of the studied samples was carried out using specialized software. To study the morphological parameters of the organs of laboratory animals, research methods widely used in experimental studies (anatomical dissection) were used. All histological preparations were viewed using a trinocular microscope HL-19 (China) with software. The rats of the second experimental group were additionally simulated thermal stress, for which the cages with the animals were irradiated with an IR lamp (K3K250W) with an exposure of 5 hours. per day.

Result and discussions

There is literature data: the effects of repeated and chronic stress on thyroid function are largely consistent with the effects of acute stress and are characterized by multidirectional changes in the levels of T4 and T3 in the blood. A 60-day unavoidable stress induced an increase in the concentration of T3 and a decrease in T4 in male rats; with chronic stress, a decrease in the concentrations of T3 and T4 was noted. Keeping animals under conditions of unavoidable, as opposed to avoidable, stress, exposure to prolonged intense stress caused a decrease in T3 levels, as shown by the authors using morphometric and biochemical methods of analysis. Activation of thyroid function (increased T4 concentration) was observed after daily one-hour immobilization of cats for 7 days, mild chronic stress in white outbred rats (increased levels of total T4 and T3 in serum), long-term transportation of male cattle (increased levels of total and free iodothyronines. Chronic stress during pregnancy in rats caused multidirectional changes in thyroid function: a decrease in the mother and an increase in thyroid status in the offspring.

In the control group, the clinical condition of the rats was characterized by the presence of stable indicators. In animals when modeling hypofunction of the thyroid gland, no sharp deviations from the clinical parameters of the control group were detected. With a gradual increase in signs of hypothyroidism and the concomitant effect of stress factors in rats of experimental group II. apathy, depression, decreased appetite, and an increase in temperature by 0.3-0.5°C were noted. The histoarchitecture of the thyroid gland of rats in the control group is organotypic. The capsule is well developed, loose, moderately injected with blood, the parenchyma of the gland is divided into lobules by trabeculae. Follicles vary in shape. In the peripheral lobules of the gland they are large, often irregular in shape, while in its central lobules the follicles are smaller and spherical in shape. The colloid in the follicles is homogeneous, pale pink in color. The interfollicular component is represented by few epithelial cells; Ashkenazi cells are not visualized. The shape of the thyrocyte is from low-prismatic to cubic, the cell nuclei are round-oval in shape. The architecture of the gland in experimental hypothyroidism is heteromorphic. The thyroid capsule is thinned and poorly vascularized. The follicles are significantly deformed, their shape ranges from round to ellipsoidal. The colloid in hypofunctional follicles is red-pink, layered, and in smaller follicles it is fragmented (due to large-scale zones of lysis). In some cases, the lumens of the

follicles are empty. The interfollicular component is small-celled, high density, and in some places forms continuous fields. Some capillaries are deserted, but the number of functioning microvessels is sharply increased. Thyrocytes vary in shape and size - from low prismatic to flattened, their cytoplasm is weakly oxygenic, vacuolated, the nuclei are basophilic, clear, in individual thyrocytes nuclei are found at different stages of lysis.

Conclusions

The effects of stress are determined by its duration, characterized by activation of the pituitary-thyroid system in the acute period and suppression during long-term and chronic stress. Stress induces a decrease in T3 concentration and an increase in rT3 production through activation of deiodinase DIII. Glucocorticoids stimulate TSH-mediated activation of iodide uptake, inhibit thyroid peroxidase in the thyroid gland and, consequently, iodide organization. The body's resistance to stress depends on the thyroid status, namely, with hypothyroidism, adaptive capabilities are reduced, which is confirmed by a decrease in the concentration of cortisol in the blood serum. In animals of the experimental group, under the combined influence of a stress factor and hypothyroidism, there was an increase in the concentration of thyroid-stimulating hormone by 14.2%, which had a stimulating effect on nuclear and cytoplasmic synthesis in thyrocytes (an increase in NPO by 1.2 times), while fluctuations in cortisol concentration with an average value, but exceeding the norm.

Consequently, the results obtained confirm the body's desire for a euthyroid state. Against the background of hypothyroidism and exposure to stress factors, despite an increase in the protective-adaptive reaction of the body, changes in the histological structures of the organ were detected in the thyroid gland, causing a decrease in the synthesis of thyroid hormones in the blood serum.

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