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Morphological Indicators of the Thyroid Gland After Hormonal Therapy of Experimental Processes of Intestinal Scar Formation

Kibriev Bekhruz Abdurakhmonovich
Bukhara State Medical Institute

Thyroid function is a complex system of interconnected processes that reflect at different levels both the specificity and strength of the hormonal signal and the sensitivity of the responding tissues. Based on the analysis of literary sources, it was established that glucocorticoids affect all links of the thyroid function: 1) biosynthesis and secretion of hormones by the gland; 2) their transport by blood; 3) interaction with target organs; 4) implementation of biological action; 5) metabolism and excretion of hormones. Biosynthesis and secretion of ITG by the thyroid gland: hydrocortisone (intraperitoneally at a dose of 10 mg/100 g) - 5-60 minutes after a single injection, the morphology of thyrocytes and the activity of thyroid peroxidase in them, which catalyzes the iodination of tyrosine residues of thyroglobulin and the fusion of iodotyrosines in the process of synthesis of thyroxine (T4) and triiodothyronine (T3), did not change. However, after a 7-day administration, the activity of thyroid peroxidase decreased, as did the number of secretory elements in the endocrine parenchyma of the thyroid gland due to the transformation of thyrocytes into non-functioning "light" cells. Transport of ITG in blood: hydrocortisone: 1) in vitro experiments (incubation of cell line and primary cultures of rat choroid plexus epithelial cells in 10, 100, 1000 nM solutions) – increase in expression of the ITG transporter transthyretin after 12 and 18 h in all samples, after 24 h – only when using 100 nM solution. In the cell line, the effect was also observed after 36 h of incubation, but only when using 10 and 100 nM solutions. The effect of hydrocortisone (incubation of the choroid plexus cell line in 100 nM solution for 12 h) was suppressed by glucocorticoid (1.16 μM mifepristone solution) and mineralocorticoid (1 μM spironolactone solution) receptor antagonists; 2) in vivo experiments (to increase the level of corticosteroids in the blood of male and female rats, they were exposed to acute (for 24 hours) (an increase of approximately 6 and 4 times) and chronic (for 9 weeks) (an increase of 2 and 2.5 times) psychosocial stress, placing 9 individuals in polypropylene cages measuring 480 × 375 × 210 mm (floor area 166 cm2/animal) - an increase in the expression of transthyretin and its mRNA in the liver and choroid plexus, and the expression of transthyretin in the cerebrospinal fluid [4]; methylprednisolone (incubation of B-lymphocytes obtained from healthy donors in a 5.34 μM solution for 4, 24 and 48 hours) - expression of immunoglobulin M, which belongs to the thyroxine-binding transport proteins of blood plasma [5], on the surface of the indicated cells decreased by 16, 58 and 68%. There are data proving the functional relationship between the hypothalamic-pituitary-adrenal system and the thyroid system. Glucocorticoids are involved in the regulation of iodine metabolism in the thyroid gland [8]: in adrenalectomized rats, there is no

inhibitory effect of high doses of potassium iodide on iodine uptake by thyrocytes and the synthesis of thyroid hormones after a single administration (1000 daily doses), and hyperthyroidism develops after repeated administration (from 1 to 500 daily doses for 14 days). On the other hand, iodine-induced blockade of the thyroid gland (8 μg potassium iodide / 100 g body weight for 5 days) leads to a short-term increase in the concentration of cortisol in the blood of rats, which indicates stimulation of adrenal function under these conditions [9]. A similar relationship has been found in stress. Experimental hypothyroidism (2.5 mg/100 g body weight tyrosol, 28 days) causes a lower concentration of cortisol in the blood of rats under temperature exposure.