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

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Abstract: Thyroid function is a complex system of interrelated 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 found that glucocorticoids affect all links of the thyroid function. At the same time, it remains relevant for researchers to study the morphometric and immunohistochemical indices of the thyroid gland, which is sensitive to homeostasis disorders, after hormonal therapy of cicatricial processes in the experimental intestine.

Keywords: intestine, cicatrization, hormones, thyroid gland, experiment.

Introduction

Regarding surgical recommendations for patients with adhesive bowel disease at high risk of intestinal obstruction, the use of high doses of betamethasone or dexamethasone is recommended. The benefits of this treatment are well documented: decreased neonatal mortality even in underdeveloped countries, without increasing the rate of maternal infections [1]. However, there is increasing evidence that high doses of these glucocorticoids may be detrimental to the health of children at other stages of life. In a literature review [Astiz], the authors showed that the use of glucocorticoids during pregnancy brings short-term benefits to the fetus, but, throughout life, they can have harmful effects on various body systems. In animal models, glucocorticoid exposure during pregnancy can lead to central and peripheral changes in the levels of ACTH, cortisol, growth hormone, IGF-1, thyroid hormones, FSH and LH, gonadotropic hormones, and pancreatic hormones. These changes can disrupt responses to stress, leading to the development of psychological diseases, metabolic diseases and reproductive dysfunction.

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 found that glucocorticoids affect all links of 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.

Methodlogy

The effect of glucocorticoids on thyroid function.

Biosynthesis and secretion of ITG by the thyroid gland: hydrocortisone (intraperitoneally at a dose of 10 mg/100 g) – 5–60 min 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 during the synthesis of thyroxine (T4) and triiodothyronine (T3), did not change. However, after 7 days of 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 [3].

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% [6].

Interaction of ITG with receptors in target organs: hydrocortisone (incubation of mixed (containing neurons, oligodendroglia and astroglia) and neuron-enriched cultures of rat brain cells with 0.3 μM hormone solution) - an increase in the level of TRα1 mRNA (TR - thyroid receptor: thyroid hormone receptor) by 5 times on the 13th day of incubation [3]; dexamethasone (intraperitoneally 50, 125 and 250 μg/100 g of body weight every 12 hours for 48 hours) – an increase in the level of TRβ1 by 52% in the liver cells of adrenalectomized rats when using a dose of 250 μg/100 g, an increase in the content of TRβ1 mRNA by 43 and 74% – at 125 and 250 μg/100 g, an increase in the rate of transcription by 255% – at 250 μg/100 g, associated with stimulation of the transcriptional activity of the TRβ1 promoter, induction of protein binding to the DNA sequence of the TRβ1 promoter site [4]. Biological action of ITG: dexamethasone (incubation of embryonic cerebrocortical cells of intact mice and mice with deficiency of thyroid hormone receptor TRa1 in 10 nM solution of the hormone for 48 h) – increase in mRNA (polymerase chain reaction) of genes, the expression of which in the brain is regulated by thyroid hormones, Klf9 (Krupfel-like transcription factor 9 or Basic Transcription Element Binding Protein) and Aldh1a1 (aldehyde dehydrogenase 1a1, a gene sensitive to hypothyroidism induced by blockade of thyroid hormone formation, but not to hypothyroidism as a result of inactivation of the Dio2 deiodinase gene and the gene encoding the synthesis of thyroid hormone transporters Mct8). Dexamethasone and T3 (1 nM solution) had a synergistic effect on stimulation of the expression of these genes. These results indicate the interaction of thyroid hormones and glucocorticoids during the development of the nervous system [5].

Results and discussion

ITG metabolism: dexamethasone (via the umbilical vein in female rats 1-5 μg/g) - a decrease in the activity of deiodinase D1 (provides up to 30-40% of the rapid extrathyroidal production of T3) in the liver and kidneys of 20-day-old fetuses, as well as the activity of deiodinase D3 (deiodinates the inner ring and catalyzes the conversion of T4 to rT3 and T3 to T2 (both inactive metabolites), rT3 to rT2) in the liver and, at the same time, an increase in the activity of deiodinases D3 and D2 (the latter provides up to 70% of the slow extrathyroidal production of T3) in the brain. In 5-day-old rats, the activity of deiodinase D3 in the liver and kidneys and D2

in the brain increased, while the activity of deiodinase D3 in the brain decreased. In 12-day-old rats, as in 5-day-old rats, the activity of deiodinase D3 in the liver and kidneys increased, but the activity of D2 in the brain decreased.

Thus, glucocorticoids stimulate the activity of thyroid hormones in the brain only during a short period of animal development [1]; dexamethasone (intramuscularly 12 mg to pregnant ewes, twice at 24-hour intervals) – an increase in the activity of deiodinase D1 in the liver and a decrease in the activity of D3 in the kidneys of fetuses and in the placenta of sheep; dexamethasone (injection of 50 μg to chicken embryos on the 18th day) – an increase in the expression and activity of mRNA of deiodinase D2 in the brain [2]; dexamethasone (incubation of the stromal-vascular fraction of rat brown adipose tissue cells in a 50 nM solution overnight) – a decrease in the activity and level of mRNA of deiodinase D3 [3]. The above-described changes in thyroid function under the influence of glucocorticoids are associated with the influence of the latter on its regulation by trans- and parapituitary. According to S.A. Kashchenko (2019), after the introduction of methotrexate, a decrease in the absolute weight of the thyroid gland was observed compared to the control group already from the 1st day. - by 2.44%, on the 7th day the difference was 8.50%. Similar changes were also observed in the linear dimensions of the thyroid gland lobes: the length of the right lobe on the 7th day of observation was less than the control group by 10.13%, the width - by 8.58%, the thickness - by 12.05%. The thyroid gland volume in animals exposed to methotrexate also decreased compared to the control group of animals: on the 1st day of observation, the difference was 10.43%, on the 7th day - 27.38%.

Conclusion

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 thyroid blockade (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, indicating stimulation of adrenal function under these conditions. A similar relationship was found under 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 (irradiation with an infrared lamp for 5 hours). An increase in the serum level of corticosteroids after emotional and pain stress correlates with a decrease in the iodine content in the thyroid gland of rats [E. A. Gusakova, 2019].

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