The American Journal of Medical Sciences and Pharmaceutical Research
(ISSN – 2689-1026)
VOLUME 04 ISSUE 02 Pages: 33-37
SJIF IMPACT FACTOR (2020: 5. 286) (2021: 5. 64)
OCLC – 1121105510 METADATA IF – 7.569
Publisher: The USA Journals

Journal Website: https://theamericanjournals.com/index.php/tajmspr

Research Article

HORMONAL FUNCTION OF ADENOHYPOPHISIS

Submission Date: February 10, 2022, Accepted Date: February 20, 2022,
Published Date: February 28, 2022 |
Crossref doi: https://doi.org/10.37547/TAJMSPR/Volume04Issue02-08

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ABSTRACT

This article discusses hormonal function of adenohypophysis, which produces a number of hormones that regulate the growth of the child. The production of adenohypophysis hormones, in turn, depends on liberins and statins, hormones of the hypothalamus that enter the pituitary portal system.

KEYWORDS

Adenohypophysis, adenohypophysis hormones, hormonal function, growth of the child, liberins, statins.

INTRODUCTION

The secretion of liberins and statins is controlled by adrenergic, cholinergic and dopaminergic neurons of higher nerve centers [1]. In addition, the secretion of certain hormones of the adenohypophysis and liberins is inhibited by the hormones of the peripheral endocrine glands according to the principle of negative feedback. Thus, hormones of the hypothalamus, adenohypophysis, and peripheral endocrine glands, the targets of adenohypophyseal hormones, are involved in growth regulation [2]. 7 hormones were isolated from the extract of the anterior pituitary gland: growth hormone, or somatotropic hormone,

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thyroid-stimulating hormone, follicle-stimulating hormone, luteinizing hormone, luteotropic hormone, prolactin (lactogenic) and adrenocorticotropic hormone (ACTH). All hormones of the anterior lobe are of a protein nature and are obtained in a purified form, some of them, such as growth hormone and lactogenic, are isolated in crystalline form, others are synthesized (for example, ACTH). Thyroid-stimulating and gonadotropic hormones are produced by basophilic cells, which, in accordance with this, are divided into two types: the so-called. thyrotrophs and gonadotrophs. Oxyphilic cells produce growth hormone and prolactin. The question of cells producing ACTH has not been resolved; it is probably formed by basophils [3].

MATERIALS AND METHODS

A growth hormone. STG family. It includes growth hormone and prolactin, as well as a hormone formed in the placenta - placental lactogen. All these hormones consist of one non-glycosylated polypeptide chain and are characterized by a significant similarity of the primary structure.

STH is synthesized in somatotropic cells, has a molecular weight of 22,000 and contains 191 amino acids. The physiological effects of STG are usually divided into direct and indirect. Direct effects of growth hormone: stimulation of the synthesis and secretion of IGF in the liver and other organs and tissues, stimulation of lipolysis in adipose tissue and stimulation of glucose production in the liver. The indirect effects of GH are its growth-promoting and anabolic effects. These effects are mediated by IGF-I. The main source of IGF-I is the liver. IGF-I stimulates the growth of bone, cartilage and soft tissues. The indirect effects of growth hormone are suppressed by glucocorticoids.

This peptide hormone is produced in somatotropic cells of the adenohypophysis. Synthesis and secretion of growth hormone are controlled by two hypothalamic hormones: somatoliberin and somatostatin. Somatoliberin stimulates, and somatostatin suppresses the secretion of growth hormone and blocks the stimulating effect of somatoliberin. It has been established that somatostatin is produced not only in the hypothalamus, but also in other parts of the nervous system, as well as in the gastrointestinal tract. Somatostatin suppresses the secretion of many hormones, including insulin, glucagon, and gastrin [72]. The level of GH secretion depends on the ratio of somatoliberin and somatostatin concentrations. Once in the blood, GH interacts with a GH-binding protein homologous to the extracellular domain of the GH receptor.

The growth-stimulating effect of GH is mediated by IGF - hormones that are formed under the influence of GH in the liver and other tissues. Two types of IGF have been identified: IGF-I and IGF-II. These are single-chain proteins similar in structure to proinsulin. IGF-I and IGF-II are present in serum mainly in the form of complexes with binding proteins. The most common IGF-binding protein type 3. IGF-I and IGF-II affect target cells in different ways. This is due to differences in the interaction of IGF with receptors. Both IGF-I and IGF-II bind to IGF-I receptors, but the similarity of IGF-I to IGF-I receptors is much greater than that of IGF-II. Both IGFs are involved in fetal development; in the postembryonic period, IGF-I plays a major role in growth regulation. It stimulates the proliferation of cells of all tissues, primarily cartilage and bone. The physiological role of IGF-II in the development of a child and in an adult organism has not yet been elucidated. As well as growth hormone, both IGFs act on the hypothalamus and adenohypophysis on the feedback

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principle, controlling the synthesis of somatoliberin and somatostatin and the secretion of growth hormone. Surgical removal of the pituitary gland in a young animal results in growth arrest. Injection of a pituitary extract containing growth hormone into such animals restores their normal growth. The introduction of growth hormone into young growing animals sharply stimulates growth and leads to gigantism (giant ambistoma, rats, dogs, and other animals were obtained in the experiment): in humans, excessive secretion of growth hormone causes a disease with symptoms of gigantism or acromegaly. Decreased secretion of growth hormone causes dwarf growth.

Prolactin is synthesized in lactotropic cells, has a molecular weight of 22,500 and contains 198 amino acids. The main target of prolactin is the mammary glands. Prolactin stimulates the growth of the mammary glands during pregnancy and lactation after childbirth. During pregnancy, the lactogenic effect of prolactin is blocked by estrogens and progesterone. Prolactin receptors are found in the hypothalamus, liver, testicles, and ovaries, but the effect of prolactin on these organs has not been studied enough. Hyperprolactinemia depresses the hypothalamic-pituitary-gonadal system and is a common cause of infertility in women. It has recently been shown that prolactin receptors are present on T-lymphocytes and that prolactin influences immune responses.

The family of glycoprotein hormones includes the adenohypophyseal hormones LH, FSH, and TSH, as well as the placental hormone hCG. These hormones consist of two highly glycosylated polypeptide chains (subunits) - alpha and beta. All hormones have identical alpha subunits: they include 92 amino acids arranged in the same sequence. In contrast, the amino acid sequences in the beta subunits differ. It is these differences that determine the specificity of the action of glycoprotein hormones on target tissues. The molecular weight of LH, FSH, TSH, and CG is not the same and depends primarily on the number of carbohydrate residues [4].

LH and FSH are synthesized in gonadotropic cells. In both hormones, the beta subunit includes 115 amino acids, and the molecular weight is 29400 and 32600, respectively. LH and FSH regulate the synthesis and secretion of sex hormones and gametogenesis [5]. In the ovaries, LH stimulates ovulation and secretion of progesterone. LH and CG receptors are present on the cells of the outer shell and granular layer of the follicles and on the interstitial cells. FSH stimulates estrogen secretion, growth and maturation of follicles. FSH receptors are present only on the cells of the granular layer. In the testicles, LH stimulates the secretion of testosterone. LH and CG receptors are present only on Leydig cells. FSH does not affect androgen synthesis, but is required for spermatogenesis. FSH receptors are found only on Sertoli cells.

Follicle-stimulating, luteinizing and luteotropic hormones. Atrophy of the reproductive system that occurs after the removal of the pituitary gland can be prevented by the introduction of gonadotropic hormones. In infantile animals, the administration of these hormones causes precocious puberty. Injection of a pituitary extract containing gonadotropic hormones to frogs causes spawning and spermatogenesis in them in autumn and winter; normal tadpoles develop from eggs after fertilization. LH and FSH. These are glycoprotein hormones secreted by gonadotropic cells of the adenohypophysis. The production of LH and FSH is regulated by GnRH. From the beginning of puberty, LH and FSH regulate the synthesis and secretion of sex hormones and gametogenesis. LH stimulates the secretion of progesterone in the ovaries and

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testosterone in the testicles. FSH stimulates the secretion of estrogen in the ovaries. Estrogens and testosterone determine the development of secondary sexual characteristics, pubertal growth acceleration and closure of the epiphyseal growth zones of long bones. Like other hormones of the peripheral endocrine glands, estrogens and testosterone, by the principle of negative feedback, inhibit the secretion of GnRH, LH and FSH.

Follicle-stimulating hormone regulates the growth of follicles in the ovaries and spermatogenesis. In females, luteinizing hormone causes premature follicular growth, ovulation, and the formation of a corpus luteum, and in males, secretion of the male sex hormone by interstitial testicular cells, i.e., Leydig cells. Luteotropic hormone supports the function of the corpus luteum; in some animals (rat, sheep) this hormone causes lactation. Prolactin (lactogenic hormone). Participates in the regulation of the process of milk secretion. Removal of the anterior pituitary in lactating females stops milk secretion; the introduction of prolactin restores lactation.

Thyroid-stimulating hormone. TSH is synthesized in thyroid-stimulating cells, has a molecular weight of 30,500; the beta subunit contains 112 amino acids. The main role of TSH is to stimulate the synthesis of thyroid hormones. TSH controls almost all stages of synthesis, including the addition of inorganic iodine to thyroglobulin and the formation of T3 and T4 from mono- and diiodotyrosine.

This glycoprotein hormone is produced in the thyroid-stimulating cells of the adenohypophysis. Synthesis and secretion of TSH are controlled by thyroliberin. TSH stimulates the synthesis and secretion of thyroid hormones (T3 and T4) - the most important growth regulators of all body tissues. By inhibiting the synthesis of thyroliberin and the secretion of TSH, T3 and T4 close a negative feedback loop in the hypothalamic-pituitary-thyroid system. Removal of the anterior pituitary gland causes atrophy of the thyroid gland and, as a result, a decrease in basal metabolism. Injections of a pituitary extract containing thyroid-stimulating hormone cause an increase in the thyroid gland and an increase in its function.

RESULTS AND DISCUSSIONS

A family of proopiomelanocortin derivatives. Corticotropic cells of the adenohypophysis secrete several hormones: ACTH, alpha and beta MSH, beta and gamma lipotropins, and endorphins. All these hormones contain the heptapeptide Met-Glu-Gis-Phen-Arg-Trp-Gly and are formed from a large precursor molecule, proopiomelanocortin (molecular weight 31,000).

ACTH has a molecular weight of 4500 and contains 39 amino acids. ACTH stimulates the synthesis of hormones in the adrenal cortex, primarily the synthesis of glucocorticoids in the fascicular and reticular zones. The release of ACTH from corticotropic cells or the administration of a large dose of ACTH can cause a short-term rise in aldosterone levels. Another effect of ACTH is the stimulation of melanin synthesis in melanocytes. Apparently, this is the cause of hyperpigmentation in Nelson's syndrome and primary adrenal insufficiency.

The functions of other derivatives of proopiomelanocortin are less well understood. It has been established that alpha-MSH stimulates the synthesis of melanin in melanocytes, and gamma-MSH stimulates the synthesis of aldosterone in the adrenal cortex. In experiments on cell cultures of the adrenal cortex, it was shown that beta-lipotropin stimulates the synthesis of corticosteroids, and the effect of beta-lipotropin is mediated by ACTH receptors.

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The synthesis of ACTH in the corticotropic cells of the adenohypophysis and the secretion of ACTH are controlled by corticoliberin. Acting on the cells of the adrenal cortex, ACTH stimulates the synthesis and secretion of cortisol, a hormone with a wide spectrum of action. In vitro and in vivo studies have shown that low concentrations of cortisol are necessary for cell growth, but even a small excess of this hormone inhibits cell proliferation. ACTH stimulates the activity of the adrenal cortex and the release of corticosteroid hormones by it, and also restores the gland atrophied as a result of the removal of the pituitary gland. The influence of the anterior pituitary gland on metabolism is carried out through growth hormone, ACTH and other hormones.

The middle lobe of the pituitary gland produces the intermediate hormone, or melanocyte-stimulating hormone, which affects the color of the skin of fish and amphibians. The physiological significance of this hormone in birds and mammals is unclear.

The posterior pituitary gland is involved in the regulation of blood pressure, urination (hormone vasopressin) and the activity of the muscles of the uterus (hormone oxytocin). Vasopressin and oxytocin are produced in the paraventricular and supraoptic nuclei of the hypothalamus, from where they enter the posterior pituitary gland. Both hormones are synthesized.

CONCLUSION

The functions of the pituitary gland depend on environmental conditions. From experiments carried out on birds and mammals, it was established that light regulates the gonadotropic, thyrotropic and adrenocorticotropic functions of the pituitary gland; The action of light on the pituitary gland is carried out through the central nervous system. It has also been proven that the endocrine functions of the pituitary gland are under the control of the hypothalamus, in which special neurohumoral substances of a peptide nature are produced - the so-called. releasing, or releasing factors, humorally stimulating the secretion of pituitary hormones. [6]

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