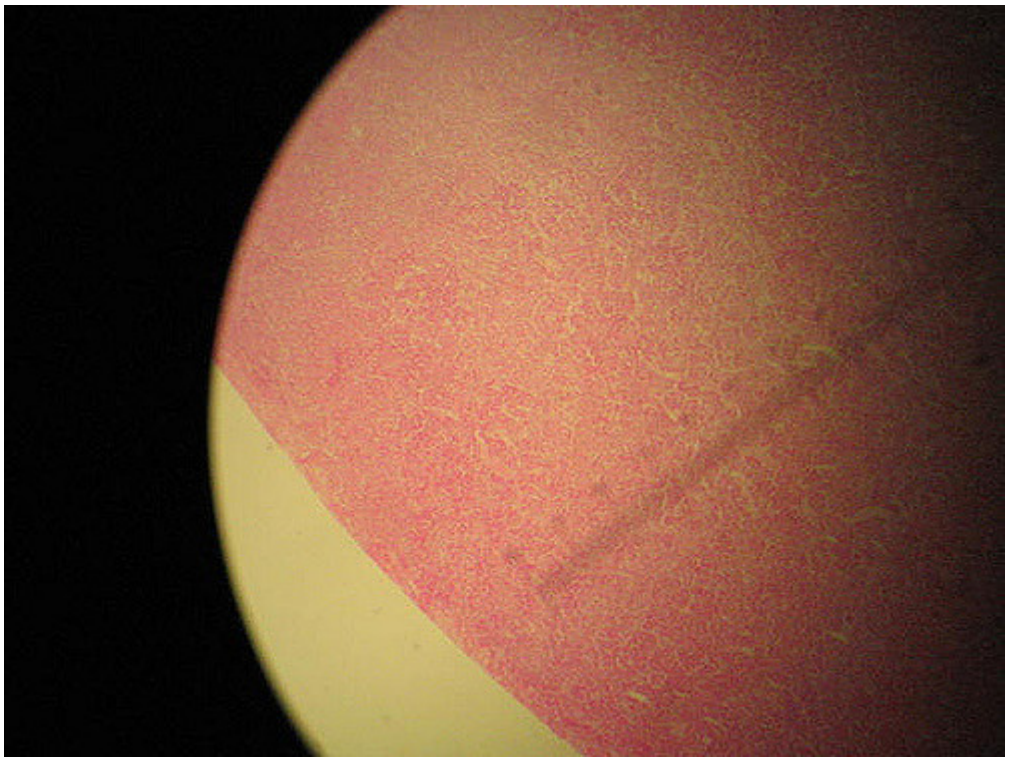
Pituitary Hormones: Posterior Pituitary & Anterior Pituitary

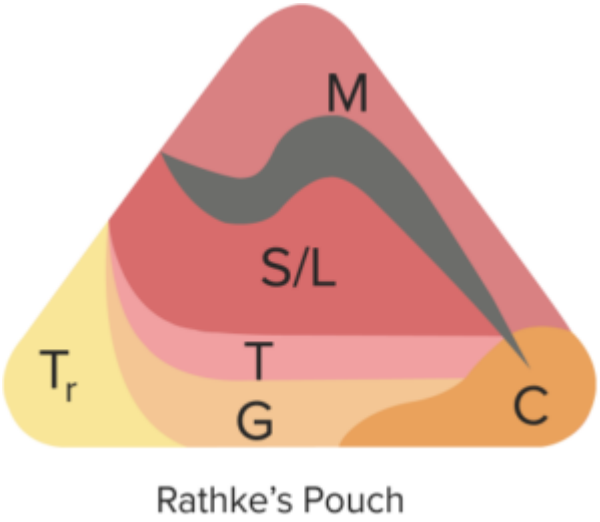
See online here

The pituitary gland, also known as the hypophysis, is a pea-sized gland measuring 1 cm in diameter and weighing 0.5–1 gram. The pituitary gland lies in a small bony cavity situated at the base of the brain, known as sella turcica. The pituitary stalk (infundibulum) maintains a connection between the pituitary gland and the hypothalamus. The secretion of hormones by the pituitary gland is under the influence of the hypothalamus through various hormonal and neuronal signals. Between the posterior and anterior pituitary lies a zone, which is relatively avascular and less-developed in humans, known as pars intermedia.



Anterior Pituitary Cell Types: Development and Histology

The image alongside shows the Rathke’s pouch; the cell types are listed below.



Rathke's Pouch

- Corticotropes (C) are at the posterior tip
- Thyrocytes (Tr) are at the corner, opposite to C
- The most ventral cells are the gonadotropes (G), followed by the main thyrotropes (T), the somatotropes and lactotrophs (S/L), and the melanotropes (M)

Hormones Secreted by the Posterior Pituitary

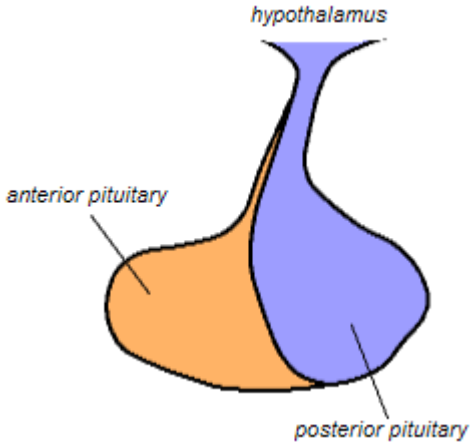


Image: "Pituitary Gland" by Diberri. License: CC BY-SA 3.0

The posterior pituitary (or **neurohypophysis**) is made up of modified glial cells (pituicytes) and axonal processes of neurons that extend from nerve-cell bodies located in the supraoptic and paraventricular nuclei of the hypothalamus. The posterior pituitary secretes two peptide hormones, namely, **arginine vasopressin** (AVP), formerly called ADH (antidiuretic hormone), and **oxytocin.**

Both hormones are synthesized by the cell bodies of the **supraoptic and paraventricular nuclei** in the hypothalamus. They are released in response to the action potential that travels from the neuronal cell bodies in the hypothalamus to the nerve terminals in the posterior pituitary, where they are stored. Both hormones of the posterior pituitary are synthesized as pre-pro-hormones, processed as nonpeptides, and released directly into the systemic circulation.

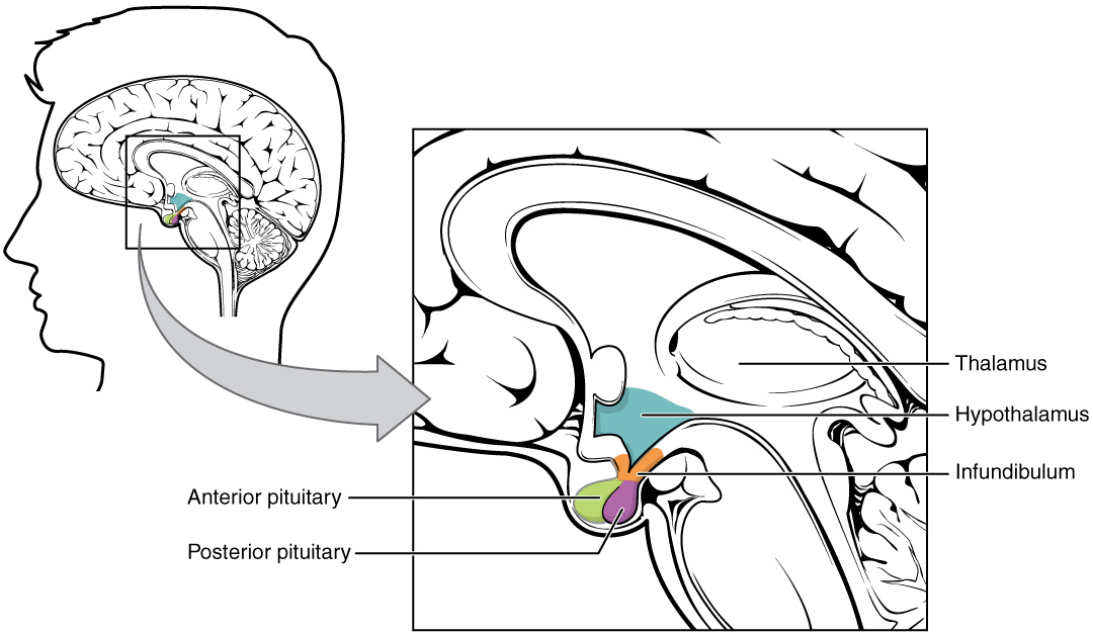


Image: “Hypothalamus-Pituitary Complex” by Phil Schatz. License: CC BY 4.0

ADH (vasopressin)

ADH is predominantly produced in the **supraoptic nucleus.** It controls the rate of excretion of water into the urine and thus regulates the concentration of body fluids. ADH effectively controls osmolarity, extracellular fluid (ECF) volume, and water excretion.

Functions of ADH

ADH plays an important role in the regulation of fluid volume and serum osmolality, sodium homeostasis, and possibly learning and memory modulation. Three receptor subtypes that mediate the actions of ADH have been identified, namely, V1A, V1B, and V2.

The primary function of ADH is to increase water reabsorption (causing water retention) in the distal tubules and collecting ducts of the kidneys. This effect is mediated by the V2 receptors via the action of cAMP leading to an increased expression of aquaporins in the distal tubules and collecting ducts.

ADH also brings about the V1 receptor-mediated generalized vasoconstriction of the vascular smooth muscle.

Regulation of ADH release

Factors that increase ADH release include **increased osmotic pressure** in the ECF/increased plasma osmolality (sensed by the hypothalamic osmoreceptors), **decreased blood volume** (sensed by the cardiac baroreceptors), **decreased blood pressure**, stress, hypoglycemia, nausea, emotional stress, pain, and physical trauma. On the other hand, alcohol consumption, decreased plasma osmolality, and the release of atrial natriuretic peptide (ANP) and alpha-agonists are among the factors that decrease ADH release.

Disorders in ADH secretion

Syndrome of inappropriate ADH release (SIADH)

SIADH is characterized by an **overproduction** or **abnormally high levels** of circulating ADH, which may arise as a result of drug therapy after surgery and brain trauma or from vasopressin-secreting tumors such as bronchial carcinoma. Patients with SIADH usually produce highly concentrated urine; water retention decreases plasma osmolality and leads to hyponatremia (sodium depletion).

Diabetes insipidus

Lack of ADH results in diabetes insipidus, which is characterized by the inability to produce concentrated urine. Polyuria, polydipsia, and increased plasma osmolality are the hallmark of diabetes insipidus, which can be categorized into two types. **Central diabetes insipidus** arises as a result of head injuries or tumors that damage the hypothalamus or posterior pituitary gland and can be effectively treated using ADH (desmopressin). **Nephrogenic diabetes insipidus** arises due to the inability of the collecting ducts of the kidney to concentrate urine and results from an impaired response to ADH.

Oxytocin

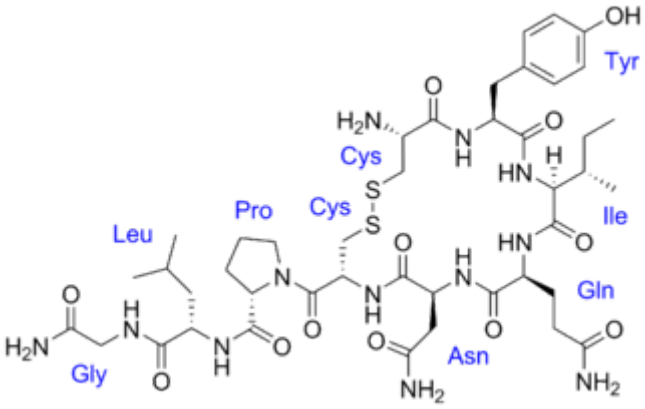


Image: “Chemical Structure of Oxytocin with Labeled Amino Acids” by Edgar181. License: Public Domain

Oxytocin is predominantly secreted by the neurosecretory cells of the hypothalamic paraventricular nucleus and helps during childbirth and breastfeeding.

Functions of oxytocin

Oxytocin is responsible for the release of milk from the breast in response to suckling via contraction of the myoepithelial cells of the alveoli and alveolar ducts. Oxytocin also helps in childbirth at the end of gestation by promoting uterine contractions and increasing the sensitivity of the myometrium (uterine smooth muscles) to spasmogenic agents. Effective smooth muscle contractions caused by oxytocin help deliver the baby and placenta during labor.

Hormones Secreted by the Anterior Pituitary

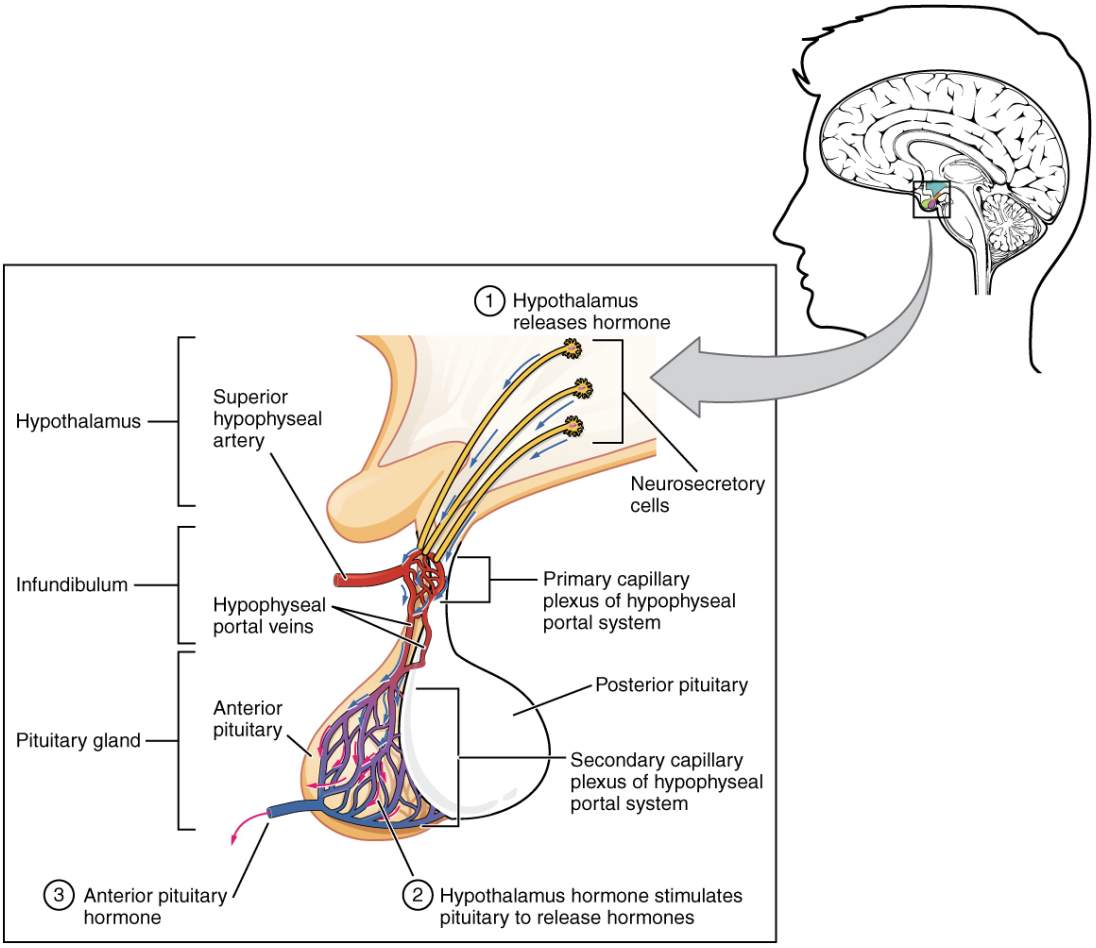


Image: "The Anterior Pituitary Complex" by OpenStax College. License: CC-BY 3.0

The anterior pituitary (or **adenohypophysis**) secretes the following six peptide hormones that are crucial in controlling the metabolic functions of the body:

1. **Growth hormone (GH)**
2. **Thyroid-stimulating hormone (TSH)**
3. **Adrenocorticotropic hormone (ACTH)**
4. **Prolactin (PRL)**
5. **Follicle-stimulating hormone (FSH)**
6. **Luteinizing hormone (LH)**

The anterior pituitary has several cell types responsible for the synthesis and secretion of anterior pituitary hormones. Histologically, five basic cell types can be identified. The cell types and the hormone secreted by each are listed below.

1. Somatotropes: growth hormone (GH)
2. Thyrotropes: thyroid-stimulating hormone (TSH)
3. Corticotropes: adrenocorticotropic hormone (ACTH)
4. Lactotropes: prolactin (PRL)
5. Gonadotropes: follicle-stimulating hormone (FSH) and luteinizing hormone (LH)

Hypothalamic control of pituitary secretion

A system of blood vessels known as the **hypothalamohypophysial portal system** is present between the hypothalamus and anterior pituitary. The hypothalamus regulates the secretion of the pituitary gland by the release of hypothalamic hormones into the hypophyseal portal blood. A negative feedback mechanism operates within the

hypothalamic-pituitary axis and target tissues to regulate endocrine and metabolic functions. The hypothalamic ‘releasing hormones’ are thought to enhance the secretion of anterior pituitary hormones, with the exception of prolactin, whose secretion is mainly regulated by the inhibitory effect of dopamine.

Hypothalamic factors or **releasing hormones** that control the secretion of anterior pituitary hormones include the following:

- Thyrotropin-releasing hormone (TRH)
- Gonadotrophin-releasing hormone (GnRH)
- Growth hormone-releasing hormone (GHRH)
- Corticotropin-releasing hormone (CRH)
- Dopamine (DA) or prolactin inhibitory factor (PIF)
- Somatostatin (SS)

Growth hormone (GH)

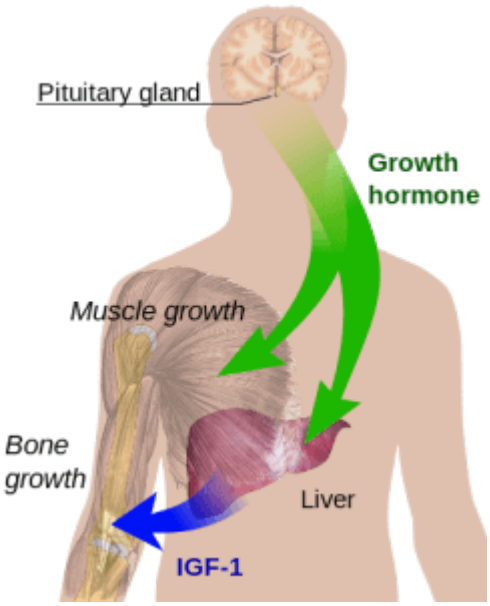


Image: “Main Pathway in Growth Regulation by the Endocrine System Mediated by Growth Hormone and Insulin-like Growth Factor-1 (IGF-1)” by Mikael Häggström . License: Public Domain

The secretion of GH is regulated primarily via hypothalamic GHRH stimulation and somatostatin inhibition. The GH targets almost all body tissues and exerts negative feedback via the hypothalamic-pituitary axis when released from the anterior pituitary.

Actions of GH

- Stimulation of growth and metabolism of almost all body tissues
- Production of insulin-like growth factor-1 (IGF-1) in the liver
- Maintenance of tissues and promoting linear growth during childhood and adolescence (acting indirectly via IGF-1)
- Increases protein synthesis and lipolysis
- Decreases glucose uptake in cells, which results in increased blood glucose levels (diabetogenic action)

Disorders of GH Production

Hypersecretion of GH during early life leads to **gigantism,** which is characterized by the symmetrical enlargement of body tissues leading to an overgrowth of long bones, connective tissue, and visceral organs.

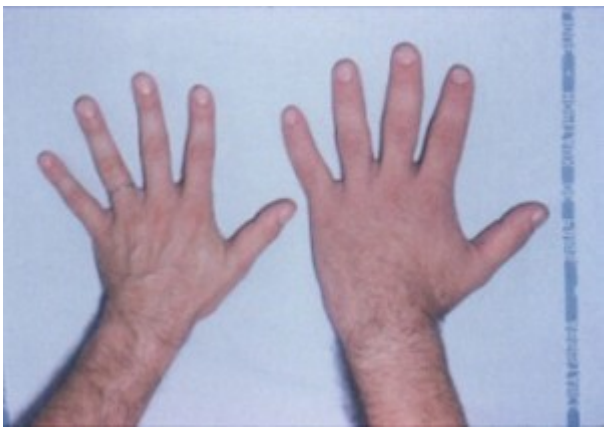


Image: “As compared to the hand of an unaffected person (left), the hand of a patient with acromegaly (right) is enlarged, the fingers widened, thickened and stubby, and the soft tissue thickened.” by Philippe Chanson and Sylvie Salenave. License: CC-BY 2.0

However, if hypersecretion of GH occurs after complete body growth, it results in **acromegaly,** characterized by asymmetrical growth and overgrown cancellous bones, leading to a protruding jaw, thickening of phalanges, and abnormally enlarged soft tissues and visceral organs.

Thyroid-stimulating hormone (TSH)

TSH stimulates the secretion of thyroid hormones from the thyroid gland. TSH secretion, in turn, is stimulated by TRH from the hypothalamus and inhibited by the negative feedback of circulating triiodothyronine.

In Grave’s disease, thyroid autoantibodies bind to the TSH receptor and mimic the action of TSH, which leads to the persistent stimulation of the thyroid and increased levels of thyroid hormones.

Adrenocorticotropic hormone (ACTH)

ACTH is produced in the anterior pituitary by the proteolytic processing of pre-pro-opiomelanocortin (also known as POMC) and other related neuropeptide products including β- and ɣ-lipotropin, β-endorphin, and α-melanocyte-stimulating hormone (α-MSH). CRH, ADH, stress, and hypoglycemia stimulate the release of ACTH from the anterior pituitary. ACTH stimulates the adrenal cortex and causes the release of glucocorticoid and steroid hormones and is also a key regulator of the stress response.

Overproduction of ACTH from the anterior pituitary causes **Addison’s disease,** which is characterized by a lack of cortisol production by the zona fasciculata of the adrenal glands. This results in the loss of negative feedback suppressing ACTH release; consequently, ACTH overproduction causes various effects including hyperpigmentation.

Prolactin (PRL)

Prolactin is the only hormone of the anterior pituitary, which is primarily controlled by the

hypothalamus by means of a negative feedback loop, with dopamine (or PIF) suppressing its release. TRH and oxytocin stimulate the release of prolactin. Prolactin **is responsible for the development of mammary glands and lactogenesis**. It is known to inhibit ovulation by blocking the effects of GnRH. Its function is not well-defined in men; however, it is suggested to be involved in the development of Leydig cells in prepubertal men.

Deficiency of PRL is not a usual clinical concern; however, its over-production may lead to the amenorrhea-galactorrhea syndrome.

Follicle-stimulating hormone (FSH) and luteinizing hormone (LH)

FSH and LH are produced by the gonadotropes in the anterior pituitary. GnRH from the hypothalamus stimulates the synthesis and secretion of these gonadotropic hormones, particularly LH. FSH secretion is mainly controlled by inhibin, which is produced by testes and ovaries.

FSH **stimulates the growth and maturation of ovarian follicles** (oocytes) in women and is responsible for the **production and maturation of sperm** in men. LH, on the other hand, triggers ovulation and enhances estrogen and progesterone secretion in women; in men, it stimulates testosterone production.

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Notes