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Topic: Adrenal gland

Adrenal gland

Adrenal gland, also called suprarenal gland, either of two small triangular endocrine glands one of which is located above each kidney. In humans each adrenal gland weighs about 5 grams (0.18 ounce) and measures about 30 mm (1.2 inches) wide, 50 mm (2 inches) long, and 10 mm (0.4 inch) thick.

The adrenal glands have a rich blood supply and experience one of the highest rates of blood flow in the body. They are served by several arteries branching off the aorta, including the suprarenal and renal arteries.

The adrenal gland consists of an outer cortex of glandular tissue and an inner medulla of nervous tissue. The cortex itself is divided into three zones: the zona glomerulosa, the zona fasciculata, and the zona reticularis. Each region secretes its own set of hormones.

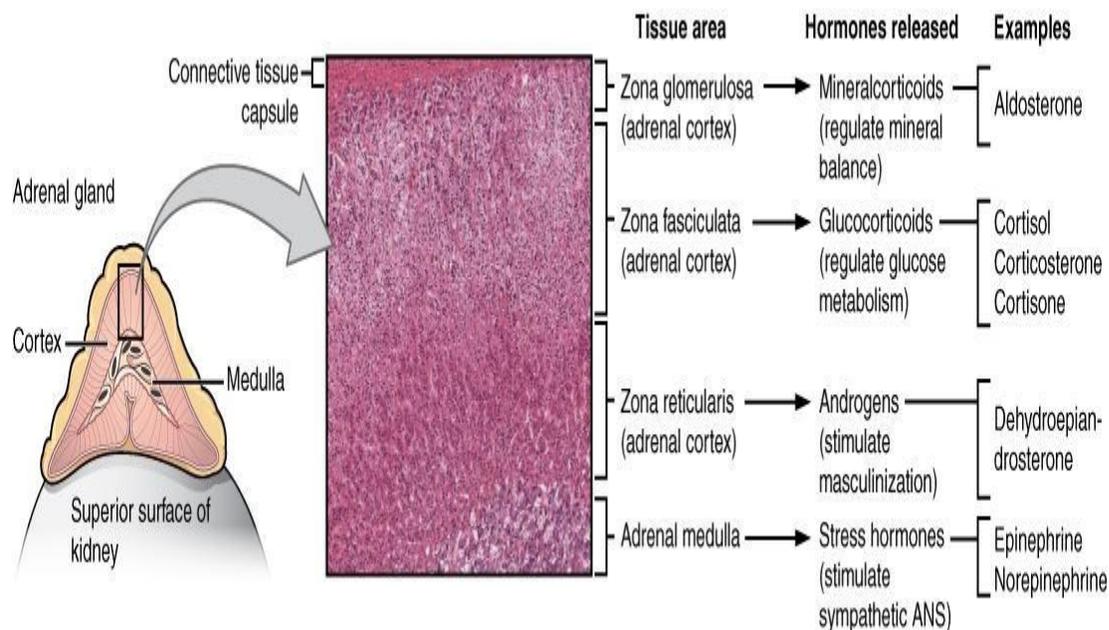


Fig. Adrenal glands

The HPA axis involves the stimulation of hormone release of adrenocorticotrophic hormone (ACTH) from the pituitary by the hypothalamus. ACTH then stimulates the adrenal cortex to produce the hormone.

The adrenal medulla is neuroendocrine tissue composed of postganglionic sympathetic nervous system (SNS) neurons. It is really an extension of the autonomic nervous system, which regulates homeostasis in the body.

The sympathomedullary (SAM) pathway involves the stimulation of the medulla by impulses from the hypothalamus via neurons from the thoracic spinal cord.

The medulla is stimulated to secrete the amine hormones epinephrine and norepinephrine.

One of the major functions of the adrenal gland is to respond to stress. Stress can be either physical or psychological or both. Physical stresses include exposing the body to injury, walking outside in cold and wet conditions without a coat on, or malnutrition.

Psychological stresses include the perception of a physical threat, a fight with a loved one, or just a bad day at school.

The body responds in different ways to short-term stress and long-term stress following a pattern known as the general adaptation syndrome (GAS).

Adrenal Cortex

The adrenal cortex consists of multiple layers of lipid-storing cells that occur in three structurally distinct regions. Each of these regions produces different hormones.

Hormones of the Zona glomerulosa

The most superficial region of the adrenal cortex is the zona glomerulosa, which produces a group of hormones collectively referred to as mineralocorticoids because of their effect on body minerals, especially sodium and potassium. These hormones are essential for fluid and electrolyte balance.

Aldosterone is the major mineralocorticoid. It is important in the regulation of the concentration of sodium and potassium ions in urine, sweat, and saliva.

For example, it is released in response to elevated blood K^+ , low blood Na^+ , low blood pressure, or low blood volume. In response, aldosterone increases the excretion of K^+ and the retention of Na^+ , which in turn increases blood volume and blood pressure.

Its secretion is prompted when CRH from the hypothalamus triggers ACTH release from the anterior pituitary.

Aldosterone is also a key component of the renin-angiotensin-aldosterone system (RAAS) in which specialized cells of the kidneys secrete the enzyme renin in response to low blood volume or low blood pressure.

Renin then catalyzes the conversion of the blood protein angiotensinogen, produced by the liver, to the hormone angiotensin I. Angiotensin I is converted in the lungs to angiotensin II by angiotensin-converting enzyme (ACE).

Angiotensin II has three major functions:

1. Initiating vasoconstriction of the arterioles, decreasing blood flow
2. Stimulating kidney tubules to reabsorb NaCl and water, increasing blood volume

3. Signaling the adrenal cortex to secrete aldosterone, the effects of which further contribute to fluid retention, restoring blood pressure and blood volume.

Hormones of the Zona fasciculata

The intermediate region of the adrenal cortex is the zona fasciculata, named as such because the cells form small fascicles (bundles) separated by tiny blood vessels. The cells of the zona fasciculata produce hormones called glucocorticoids because of their role in glucose metabolism. The most important of these is cortisol, some of which the liver converts to cortisone.

A glucocorticoid produced in much smaller amounts is corticosterone. In response to long-term stressors, the hypothalamus secretes CRH, which in turn triggers the release of ACTH by the anterior pituitary. ACTH triggers the release of the glucocorticoids.

Their overall effect is to inhibit tissue building while stimulating the breakdown of stored nutrients to maintain adequate fuel supplies.

In conditions of long-term stress, for example, cortisol promotes the catabolism of glycogen to glucose, the catabolism of stored triglycerides into fatty acids and glycerol, and the catabolism of muscle proteins into amino acids.

Hormones of the Zona reticularis

The deepest region of the adrenal cortex is the Zona reticularis, which produces small amounts of a class of steroid sex hormones called androgens.

During puberty and most of adulthood, androgens are produced in the gonads. The androgens produced in the Zona reticularis supplement the gonadal androgens. They are produced in response to ACTH from the anterior pituitary and are converted in the tissues to testosterone or estrogens.

In adult women, they may contribute to the sex drive, but their function in adult men is not well understood. In post-menopausal women, as the functions of the ovaries decline, the main source of estrogens becomes the androgens produced by the zona reticularis.

Adrenal Medula

The adrenal cortex releases glucocorticoids in response to long-term stress such as severe illness. In contrast, the adrenal medulla releases its hormones in response to acute, short-term stress mediated by the sympathetic nervous system (SNS).

The medullary tissue is composed of unique postganglionic SNS neurons called chromaffin cells, which are large and irregularly shaped, and produce the neurotransmitters epinephrine (also called adrenaline) and norepinephrine (or noradrenaline).

Epinephrine is produced in greater quantities—approximately a 4 to 1 ratio with norepinephrine—and is the more powerful hormone.

Because the chromaffin cells release epinephrine and norepinephrine into the systemic circulation, where they travel widely and exert effects on distant

cells. Derived from the amino acid tyrosine, they are chemically classified as catecholamines.

The secretion of medullary epinephrine and norepinephrine is controlled by a neural pathway that originates from the hypothalamus in response to danger or stress (the SAM pathway).

Both epinephrine and norepinephrine signal the liver and skeletal muscle cells to convert glycogen into glucose, resulting in increased blood glucose levels.

These hormones increase the heart rate, pulse, and blood pressure to prepare the body to fight the perceived threat or flee from it. In addition, the pathway dilates the airways, raising blood oxygen levels.

It also prompts vasodilation, further increasing the oxygenation of important organs such as the lungs, brain, heart, and skeletal muscle. At the same time, it triggers vasoconstriction to blood vessels serving less essential organs such as the gastrointestinal tract, kidneys, and skin.

Regulation of Adrenal Hormone Secretion

The secretion of cortisol and aldosterone is regulated by different mechanisms. The secretion of cortisol is regulated by the classical hypothalamic-pituitary-adrenal feedback system.

The major determinant that controls the secretion of cortisol is corticotropin (adrenocorticotropin; ACTH).

Hormones of the Adrenal Glands

Adrenal gland	Associated hormones	Chemical class	Effect
Adrenal cortex	Aldosterone	Steroid	Increases blood Na ⁺ levels
Adrenal cortex	Cortisol, corticosterone, cortisone	Steroid	Increase blood glucose levels
Adrenal medulla	Epinephrine, norepinephrine	Amine	Stimulate fight-or-flight response

In normal subjects there is both pulsatile and diurnal (referred to as a circadian rhythm) secretion of corticotropin, which causes pulsatile and diurnal secretion of cortisol.

Variations in the secretion of corticotropin are caused by variations in the secretion of corticotropin-releasing hormone by the hypothalamus and by variations in serum cortisol concentrations.

An increase in serum cortisol concentrations inhibits the secretion of both corticotropin-releasing hormone and corticotropin.

Conversely, a decrease in serum cortisol concentration results in an increase in the secretion of corticotropin-releasing hormone and corticotropin, thereby restoring the secretion of cortisol to normal concentrations.

However, if the adrenal glands are unable to respond to stimulation by corticotropin, decreased serum cortisol concentrations will persist.

Severe physical or emotional stresses stimulate the secretion of corticotropin-releasing hormone and corticotropin, resulting in large increases in serum cortisol concentrations.

However, under these circumstances, increased serum cortisol concentrations do not inhibit the secretion of corticotropin-releasing hormone or corticotropin and thereby allow large amounts of cortisol to be secreted until the stress subsides.

Aldosterone secretion is regulated primarily by the renin-angiotensin system. Renin is an enzyme secreted into the blood from specialized cells that encircle the arterioles (small arteries) at the entrance to the glomeruli of the kidneys (the renal capillary networks that are the filtration units of the kidney).

The renin-secreting cells, which compose the juxtaglomerular apparatus, are sensitive to changes in blood flow and blood pressure, and the primary stimulus for increased renin secretion is decreased blood flow to the kidneys.

A decrease in blood flow may be caused by loss of sodium and water (as a result of diarrhea, persistent vomiting, or excessive perspiration) or by narrowing of a renal artery.

Renin catalyzes the conversion of a plasma protein called angiotensinogen into a decapeptide (consisting of 10 amino acids) called angiotensin I.

An enzyme in the serum called angiotensin-converting enzyme (ACE) then converts angiotensin I into an octapeptide (consisting of eight amino acids) called angiotensin II.

Angiotensin II acts via specific receptors in the adrenal glands to stimulate the secretion of aldosterone, which stimulates salt and water reabsorption by the kidneys, and the constriction of arterioles, which causes an increase in blood pressure.

Diseases of the Adrenal Glands:

Diseases of the adrenal glands may be divided into those of the medulla and those of the cortex.

The only known disease of the adrenal medulla is a tumor known as a pheochromocytoma. Pheochromocytomas secrete excessive quantities of epinephrine and norepinephrine.

Many patients with these tumours have periodic episodes of hypertension (high blood pressure), palpitations of the heart, sweating, headaches, and anxiety, whereas other patients have persistently high blood pressure.

High blood pressure and other symptoms can be treated with drugs that block the action of epinephrine and norepinephrine; however, the most effective treatment is surgical removal of the tumour.

Diseases of the adrenal cortex may be manifested as hyperfunction (excessive secretion of adrenocortical hormones) or hypofunction (insufficient secretion of these hormones), also known as Addison disease.

Adrenocortical hyperfunction may be congenital or acquired. Congenital hyperfunction is always due to hyperplasia (enlargement) of both adrenal glands, whereas acquired hyperfunction may be due to either an adrenal tumour or hyperplasia.

Congenital adrenal hyperplasia, also known as adrenogenital syndrome, is a disorder in which there is an inherited defect in one of the enzymes needed for the production of cortisol.

Excessive amounts of adrenal androgens must be produced to overcome the block in cortisol production. In female infants this results in masculinization with pseudohermaphroditism (anomalous development of genital organs), whereas in male infants it results in premature sexual development (sexual precocity).

Acquired adrenocortical hyperfunction is manifested by either cortisol excess (Cushing syndrome), androgen excess, or aldosterone excess (primary aldosteronism).

Cushing syndrome is characterized by obesity, rounding and reddening of the face, high blood pressure, diabetes mellitus, osteoporosis, thinning and easy bruising of the skin, muscle weakness, depression, and, in women, cessation of menstrual cycles (amenorrhea).

The major causes of Cushing syndrome are a corticotropin-producing tumour of the pituitary gland (known as Cushing disease), production of corticotropin by a nonendocrine tumor, or a benign or malignant adrenal tumour.

All these disorders are treated most effectively by surgical removal of the tumour. Androgen excess in women is characterized by excessive hair growth on the face and other regions and amenorrhea; in contrast, androgen excess has few effects in men.

The major causes of adrenal androgen excess in adults are late-onset congenital adrenal hyperplasia and adrenal tumours.

Primary aldosteronism: is characterized by high blood pressure, caused by increased retention of salt and water by the kidneys, and low serum potassium concentrations (hypokalemia), caused by excess excretion of potassium in the urine.

The symptoms and signs of aldosterone excess include not only hypertension but also muscle weakness and cramps and increased thirst and urination.

Primary aldosteronism is usually caused by a benign adrenal tumour (adenoma), but some patients have hyperplasia of both adrenal glands.

Successful removal of the adrenal tumour usually results in reduction in blood pressure and cessation of potassium loss; patients with bilateral adrenal hyperplasia are treated with antihypertensive drugs.

