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Topic: Function of Adrenal gland

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.

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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	2Steroid	Increase blood glucose levels
Adrenal medulla	Epinephrine, norepinephrine	Amine	Stimulate fight-or-flight response

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).

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.