Module 4:Hormone-Behaviour Relationship

Lecture 25:Adrenal medulla & adrenal cortex

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Adrenal medulla

Adrenal medulla secretes epinephrine (also called adrenaline) and norepinephrine (also called noradrenaline). They affect metabolism, heart rate, breathing, etc. preparing the individual for fight-flight situations. Anxiety and mood disorders affect our sympathetic nervous system, limbichypothalamic-pituitary-adrenal (LHPA) axis and the serotonin system. As a consequence, behavioural response to stress and emotional regulation are affected. You are aware of the fight-flight or freeze reaction. It is marked by increase in heart rate, blood pressure, and metabolic rate. This is caused due to increase of epinephrine level and SNS activity. The animation given below demonstrates how stress affects our brain and body.

As seen in the animation above, in a stressful event the locus coeruleus stimulates the amygdale. In the first unit we have already discussed that amygdala is involved in the regulation of emotion. Stimulation of amygdala stimulates the hypothalamus to release corticotrophin-releasing hormone (CRH) which, in turn, stimulated the pituitary to secrete adrenocorticotropin (ACTH). Amygdala also stimulates dopaminergic inputs to the medial prefrontal cortex whereas CRH also stimulates the cortex. The ACTH stimulates the adrenal gland to release cortisol. Cortisol triggers the negative feedback channel thus inhibiting the hypothalamus, pituitary, and hippocampus activities. Suppression of the LHPA axis restores the baseline cortisol level. This helps contain the stress response thus establishing homeostasis. As you can see here, CRH works as a hormone as well as a neurotransmitter.

The dopaminergic input to prefrontal cortex is sensitive to stress. Increase in the level of dopamine in the prefrontal cortex affects attention and other cognitive processes. Hence, chronic stress results into very high level of dopamine in the prefrontal cortex, thus impairing functions controlled by it. This might result into inattention, hypervigilance, and learning problems. It might induce psychotic symptoms developing children. Serotonin is important for mood regulation. Low level of serotonin has

been associated with aggressive behaviour and suicidal tendency.

Animal studies of unpredictable and uncontrollable stress prove that decrease of serotonin the amygdala, medial prefrontal cortex, nucleus accumbens, and lateral hypothalamus leads to learned helplessness. Further, maternal deprivation and maternal stress affects the development of LHPA axis of the offspring.

Norepinephrine: Norepinephrine is a catecholamine produced by dopamine ß-hydroxylase. It is either secreted as a hormone from the adrenal medulla into the blood or released as a neurotransmitter in the brain. Learning and memory processes change the synaptic strength and norepinephrine plays an important role in this. Emotionally salient events activate locus coeruleus subsequently followed by release of norepineprine in the brain. This results into enhancement of memory. The norepinephrine further activates ß-adrenoreceptors thus facilitating synaptic transmission. This mechanism involves rise in the intracellular cAMP concentration and new protein synthesis. This mechanism facilitates acquisition and maintenance of memory.

Norepinephrine also promotes the expression of Arc protein, thus influencing the mechanisms of synaptic plasticity and memory formation.

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Release of norepinephrine in the amygdala facilitates encoding and retention of emotionally significant events. Hippocampus is another important locus of norepinephrine activity that facilitates learning and memory. It also regulates locomotor activity, mechanisms of pain, sleep and vigilance. Insufficient activity of the norepinephrine system can induce depressive syndrome. Increased activity of the serotonin system and decreased activity of the dopamine and norepinephrine systems of the brain are held responsible for emotionally negative states. The reverse is true for emotionally positive states, that is, increased dopamine and norepinephrine and decreased serotonin activities.
Animal studies show that when norepinephrine is injected to various regions of the brain while memory encoding takes place or shortly after the behavioural training it could enhance memory performance. Epinephrine-norepinephrine ratio is relatively high in fight than in flight condition. Serotonin weakens fight type aggression.

Adrenal cortex

Adrenal cortex secretes corticosteroids such as glucocorticoids (examples— cortisol, cortisone and corticosterone) and mineralocorticoids (example— aldosterone). Corticosterone affects metabolism and prepares for response to stress and exercise Hypersecretion can cause Cushing's syndrome which is characterized by changes in carbohydrate and protein metabolism. Aldosterone regulates sodium, potassium and pH balance.
Congenital adrenal hyperplasia is a genetic disorder caused by 21-hydroxylase deficiency in most cases. It affects adrenal cortex resulting into inadequate synthesis of glucocorticoids and mineralocorticoids. This results into excessive secretion of androgens and the corticotrophin-releasing hormone. Congenital adrenal hyperplasia affects the cognitive profile of the patient such as variations in intelligence quotient. High level of androgens during early developmental stage increases cognitive masculinization in females that gets reflected in the form of decreased interest in maternal behavior, intense aggressive behaviour, and less concern for infants.
The memory of stressful events is better compared to events devoid of emotions. Glucocorticoid level consolidates memory-encoding in both animals and human beings. Low dose of glucocorticoid enhance memory consolidation whereas high dose impairs it.

Prostaglandins

Prostaglandins are local hormones that act on or near their site of synthesis. It is synthesized almost in every tissue of the body, but the prostaglandin system of the kidney is considered the most active systems. It can impair memory. It mediates depression-like behaviour and reduces tendency of social exploration. Besides mediating certain physiological functions, prostaglandins also reconcile sickness behaviour. Hennessy et al. (2007) have reported that the injection of indomethacin to maternally separated guinea pig pups also reduced their passive behaviour. Indomethacin is an inhibitor of prostaglandin synthesis.

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