Model Answer

M. Sc. I Sem 2014-15

Paper LZT:303 Endocrinology

Q 1.

Answer

(i) d	(ii) d	(iii) c	(iv) b	(v) b
(vi) d	(vii) b	(viii) d	(ix) b	(x) c

Q 2.

Answer

The corticosteroids are cholesterol-derived endocrine hormones synthesized and secreted from the adrenal cortex. There are two major classes of corticosteroids:

- Glucocorticoids
- Mineralocorticoids

Weak androgens are also secreted from the adrenals.

The range of physiological effects of the glucocorticoids is remarkably diverse and widespread. Among others, they include effects on carbohydrate, protein and lipid metabolism, the stress response, aspects of nervous system and cardiovascular function, skeletal muscle and, notably, the immune system. Mineralocorticoids, on the other hand, have strictly defined functions, namely to regulate plasma (and hence extracellular) sodium and potassium levels. In either case, the corticosteroids are essential to life-sustaining biological systems and, consequently, their

synthesis and secretion are tightly regulated via the hypothalamic-pituitary-adrenal axis.

Glucocorticoids

Glucocorticoids are synthesized from cholesterol in a series of reactions carried out in the zona fasciculata of the adrenal cortex via distinct P450 enzymes. In humans, the resultant major glucocorticoid is cortisol (hydrocortisone). In many other mammals (eg. rodents) corticosterone is usually the major glucocorticoid. Both are extensively bound to plasma proteins and have fairly short durations of action.

Biological actions of Adrenocorticoids -

Total loss of adrenocortico secretion usually causes death. Without mineralocorticoids, K concentration of ECF rises markedly. The Na^+ and Cl^- conc. decrease and total extracellular fluid volume and blood volume also become greatly reduced. Because glucocorticoids are absent water, carbohydrate and protein metabolism become abnormal and thus death follows.

I- Physiological effects of Glucocorticoids -

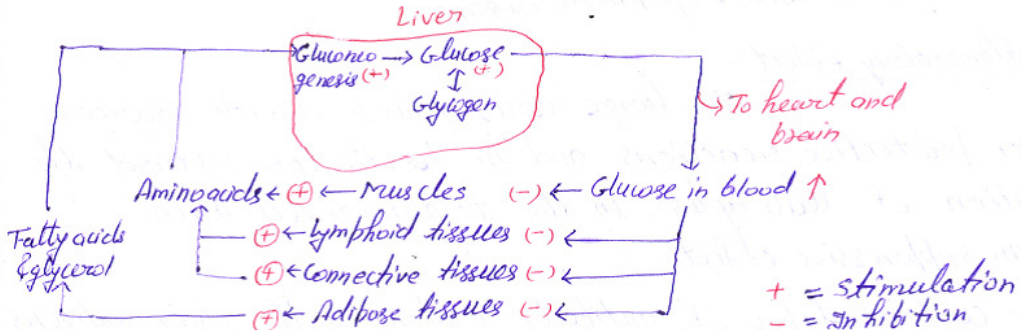


Fig. Action of Glucocorticoids on Intermediary Metabolism

- In peripheral tissues glucocorticoids are catabolic.
- Protein synthesis is depressed. In adipose tissues glucocorticoids increase lipolysis.
- In liver, gluconeogenesis, glycogen deposition and urea production are increased.
- Glucocorticoids, thus exert an antiinsulin action and make diabetes worse. However, brain and heart are spared and extra glucose supply is ensured to these vital organs.

II - Permissive action of glucocorticoids -

- Small amount of glucocorticoid must be present for a number of metabolic reactions to occur.
- Permissive actions include the requirement of glucocorticoides to be present for glucagon and catecholamines to exert their calorigenic effects.

Mechanism of action: activate heteromeric receptors resident in the cytoplasm of cells, the ligand binding component of which dimerizes and translocates to the nucleus and, in turn, activates the glucocorticoids response element (GRE) present on certain genes, leading to altered gene expression (positive and negative) Physiological effects:

Many 'permissive' effects - profound alteration in absence of glucocorticoid,

but minimal change with increases over normal levels. Example: diminished responsiveness of vascular smooth muscle to catecholamines in the absence of circulating cortisol; little effect of

supranormal levels of cortisol (though secondary hypertension may result from mineralocorticoid action of supraphysiological levels of glucocorticoids)

Metabolic effects:

- stimulate gluconeogenesis when fasting
- increase glucose production from protein
- increase lipolysis (counteracted by increased insulin secretion, which stimulates lipogenesis)
- stimulate RNA and protein synthesis in the liver

Anti-inflammatory and immunosuppressive effects:

leukocyte number and prostaglandin production:

- greatly reduced inflammatory responses

lymphocyte (interleukin-2 production) and macrophage function:

- greatly reduced immune response

Antibody production is usually unaffected at low to moderate doses of glucocorticoids

Mineralocorticoids

Aldosterone is also synthesized from cholesterol in the adrenal cortex , but in different cells (zona glomerulosa) than those that produce glucocorticoids and, consequently, it is not controlled by ACTH. Its synthesis and release from the adrenal cortex are largely regulated by extracellular K+ levels and, most notably, angiotensin II.

Mechanism of action:

act on distinct mineralocorticoid receptors resident in cytoplasm of distal tubule cells in the kidney, resulting in signaling similar to that found for the glucocorticoid receptor

Physiological effects:

promotes reabsorption of sodium in the distal tubule (and 'collecting' ducts) of the kidney, leading to increase water retention (osmotic effect) and hence in an increased internal volume

promotes potassium and hydrogen secretion in the distal tubules.

Q 3.

Answer

The renin-angiotensin aldosterone system (RAAS) plays an integral role in the homeostatic control of arterial pressure, tissue perfusion, and extracellular volume. It functions as an unusual endocrine axis in which the active hormone, angiotensin (Ang) II, is formed in the extracellular space by sequential proteolytic cleavage of its precursors. This pathway is initiated by the regulated secretion of renin, the rate-limiting processing enzyme.

Components of system

The renin-angiotensin aldosterone hormonal cascade begins with the biosynthesis of renin by the juxtaglomerular cells (JG) that line the afferent arteriole of the renal glomerulus. Renin is synthesized as a preprohormone, and mature (active) renin is formed by proteolytic removal of prorenin, the proenzyme or renin precursor. Mature renin is stored in granules of the JG cells and is released by an exocytic process involving stimulus-secretion coupling into the renal and then the systemic circulation. Active renin secretion is regulated principally by 4 interdependent factors:

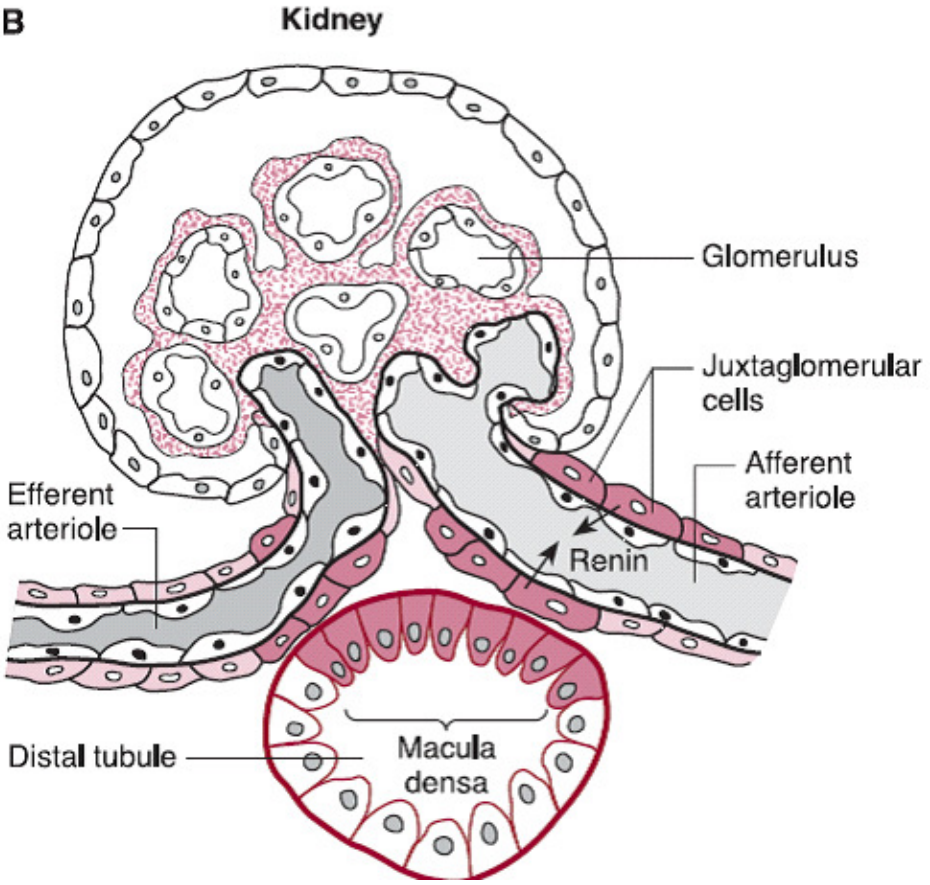
(1) a renal baroreceptor mechanism in the afferent arteriole that senses changes in renal perfusion pressure,

(2) changes in delivery of NaCl (sensed as changes in Cl- concentration) to the macula densa cells of the distal tubule (which lie close to the JG cells and, together, form the "JG apparatus"),

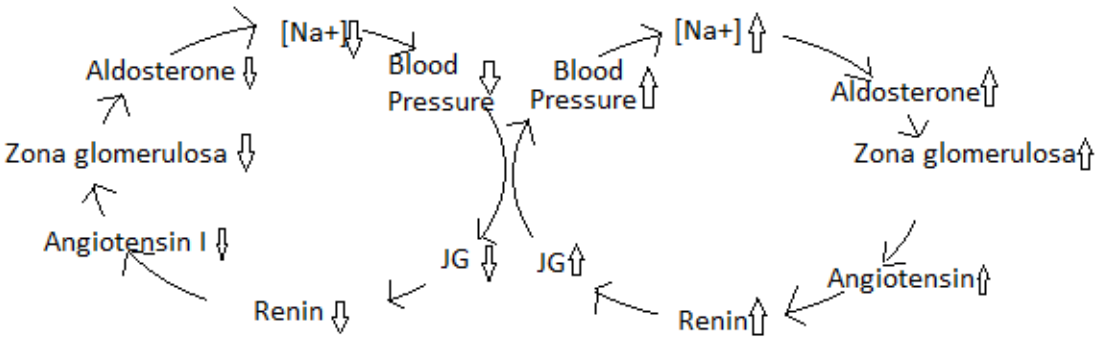
(3) sympathetic nerve stimulation via beta-1 adrenergic receptors, and

(4) negative feedback by a direct action of Ang II on the JG cells

Control of renin secretion is a key determinant of the activity of this system. Renin regulates the initial, rate-limiting step by cleaving the N-terminal portion of a large molecular weight globulin, angiotensinogen, to form Angiotensin I. The primary source of systemic circulating angiotensinogen is the liver. The inactive Angiotensin I is hydrolyzed by angiotensin-converting enzyme to form the Angiotensin II.



Renal juxtaglomerular apparatus



Renin-Angiotensin system and Na^+ homeostasis

An atrial natriuretic factor (ANF) inhibits tubular reabsorption

ANF is secreted from atria of heart. ANF (28 amino acid) is derived from a 126 amino acid precursor (pro-ANF). ANF affects dieresis and natriuresis by several physiological actions-

- It inhibits aldosterone production by adrenal glomerulosa cells.
- It inhibits release of rennin.
- It inhibits vasopressin secretion from the pituitary, as well action of vasopressin at the level of kidney.
- It causes relaxation of blood vessels.

All these ANF actions effectively reduce the retention of Na^+ and water.

Q 4

Answer

- **Cytoplasmic/nuclear receptor**

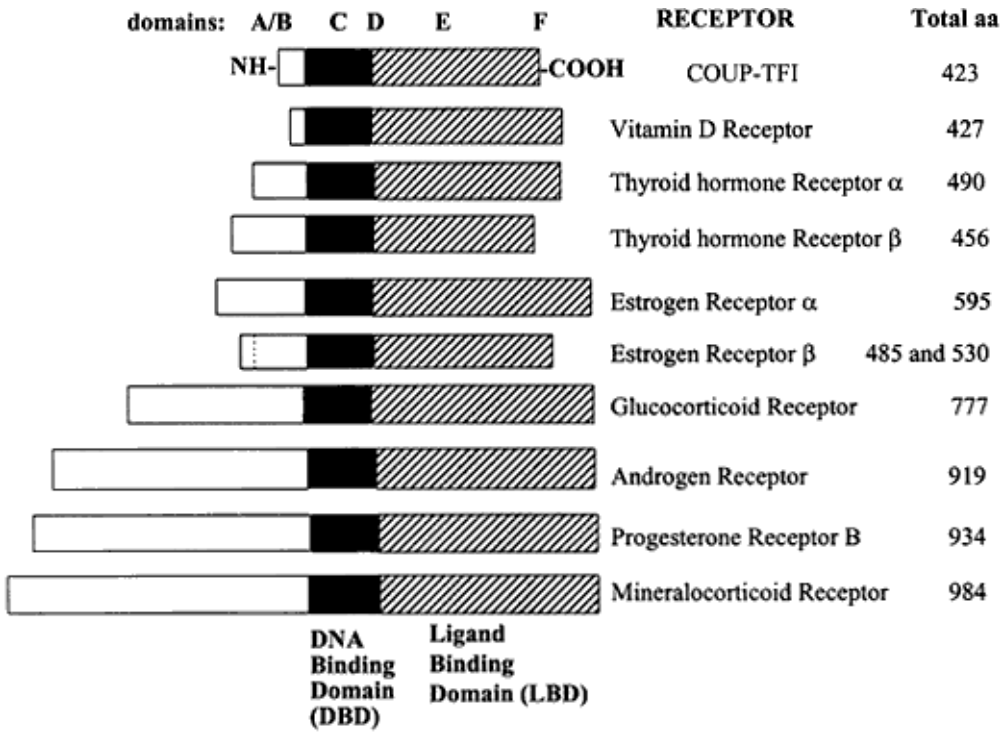
Steroid receptors of the nuclear receptor family are all transcription factors. Depending upon the type of receptor, they are either located in the cytosol and move to the cell nucleus upon activation, or remain in the nucleus waiting for the steroid hormone to enter and activate them. This uptake into the nucleus is facilitated by nuclear localization signal (NLS) found in the hinge region of the receptor. This region of the receptor is covered up by heat shock proteins (HSPs) which bind the receptor until the hormone is present. Upon binding by the hormone the receptor undergoes a conformational change releasing the HSP, and the receptor together with the bound hormone enter the nucleus to act upon transcription.

Members of the steroid receptor superfamily share direct amino acid homology and a common structure. Receptors in this superfamily contain several key structural elements which enable them to bind to their respective ligands with high affinity and specificity, recognize and bind to discrete response elements within the DNA sequence of target genes with high affinity and specificity, and regulate gene transcription. The receptors contain three major functional domains that have been shown experimentally to operate as independent cassettes within the molecule. The three major functional domains of the receptor are-

- A variable N-terminus domain that confers immunogenicity and modulates transcription in a gene and cell-specific manner through its N-terminal Activation Function

- A central DNA-binding domain (DBD), comprised of two functionally distinct zinc fingers through which the receptor physically interacts directly with the DNA helix

- The ligand-binding domain (LBD) that contains Activation Function



Structure of Steroid hormone receptor

The receptor superfamily is sub-divided into three classes-

- Class 1 is the steroid receptor family, and includes the progesterone receptor (PR), the estrogen receptor (ER), the glucocorticoids receptor (GR), the androgen receptor (AR) and the mineralocorticoid receptor.

- Class 2, or the thyroid/retinoid family, includes the thyroid receptor (TR), vitamin D receptor (VDR), the retinoic acid receptor (RAR) and the Peroxisome proliferator

activated receptor (PPAR)

- Class 3 nuclear receptor is known as the orphan receptor family. This class of nuclear receptor comprises a set of proteins sharing significant sequence homology to known nuclear receptors, but for which the ligands have not yet been identified.

- **Protein hormones**

Peptide and protein hormones are products of translation. They vary considerably in size and post-translational modifications, ranging from peptides as short as three amino acids to large, multisubunit glycoproteins. Peptide hormones are synthesized in endoplasmic reticulum, transferred to the Golgi and packaged into secretory vesicles for export. They can be secreted by one of two pathways:

· Regulated secretion: The cell stores hormone in secretory granules and releases them in "bursts" when stimulated. This is the most commonly used pathway and allows cells to secrete a large amount of hormone over a short period of time.

· Constitutive secretion: The cell does not store hormone, but secretes it from secretory vesicles as it is synthesized.

Most peptide hormones circulate unbound to other proteins, but exceptions exist; for example, insulin-like growth factor-1 binds to one of several binding proteins. In general, the half-life of circulating peptide hormones is only a few minutes. Several important peptide hormones are secreted from the pituitary gland. The anterior pituitary secretes:

· Luteinizing hormone and follicle stimulating hormone, which act on the gonads.

· prolactin, which acts on the mammary gland,

· adrenocortiotrpic hormone (ACTH), which acts on the adrenal cortex to regulate the secretion of glucocorticoids, and

· growth hormone, which acts on bone, muscle and liver.

The posterior pituitary gland secretes:

· antidiuretic hormone, also called vasopressin, and

· oxytocin.

Peptide hormones are produced by many different organs and tissue including:

· the heart (atrial-natriuretic peptide (ANP) or atrial natriuretic factor (ANF))

· pancreas (insulin and somatostatin),

· the gastrointestinal tract cholecystokinin, gastrin, and

· fat stores (leptin)

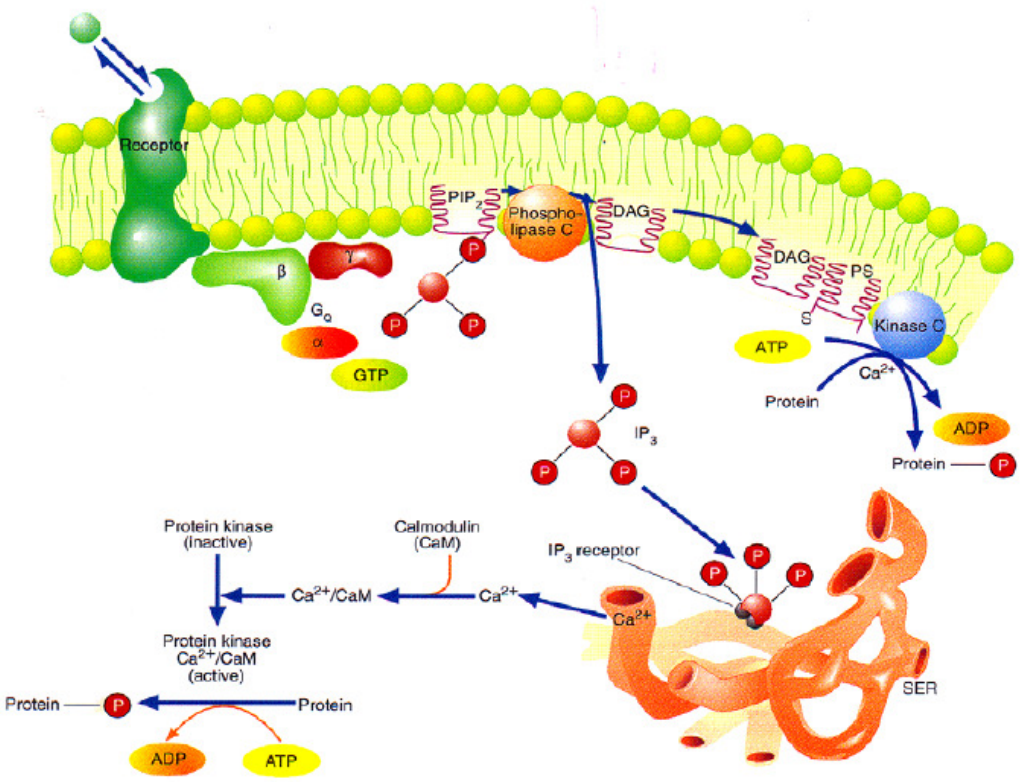
- **IP_3 and DAG**

Hydrolysis of PIP2 by PLC generates second messengers IP3 and diacylglycerol (DAG). The binding of vasopressin to the receptor induces the associated G protein to exchange GDP for GTP causing the Gαq subunit to dissociate from the $G_{\beta\gamma q}$ dimer. The $G_{\alpha q}$ subunit with GTP bound associates with phospholipase C activating the lipase. The activated lipase hydrolyzes the phosphodiester bond linking the phosphorylated inositol to the diacylglycerol. The cleavage of this phosphodiester bond produces 2 second messengers, inositol 1,4,5- triphosphate (IP_3) and diacylglycerol (DAG). DAG diffuses laterally in the lipid membrane while IP_3 is water soluble and diffuses into the cytosol of the cell.

Inositol 1,4,5 triphosphate is a second messenger that binds to IP_3-gated channels located in the membrane of the endoplasmic reticulum. The binding of IP_3 promotes the rapid release of Calcium ions. Calcium ions are yet another second messenger which triggers muscle contractions in muscle tissue, exocytosis and glycogen breakdown. Inositol 1,4,5 triphosphate has a short half life. It is rapidly converted into derivatives such as inositol or inositol 1,3,4,5 tetrakisphosphate that do not open the calcium channels.

Diacylglycerol (DAG) is another second messenger. Diacylglycerol activates a number of protein effectors for example protein kinase C (PKC). This is a kinase that uses ATP to phosphorylate serine and tyrosine residues of many target proteins. Before it is activated by DAG, this protein is free in the cytosol. When phosphatidylinositol diphosphate is hydrolyzed by the activated phospholipase C, DAG is generated in the lipid membrane. PKC binds to the DAG creating an attachment of PKC to the membrane. When PCK is anchored to the membrane the psuedosubstrate sequence interacts with head groups of the phospholipids of the membrane. This activates the kinase activity of this enzyme. PKC also requires Calcium ions for activity (remember that IP3 induces the release of Ca2+ from the endoplasmic reticulum and the calciosomes). The two second messengers DAG and IP3 work in tandem to activate PKC. DAG also has a short half life. It is phosphorylated to form phosphatidic acid or it

is hydrolyzed to its glycerol and fatty acid components.

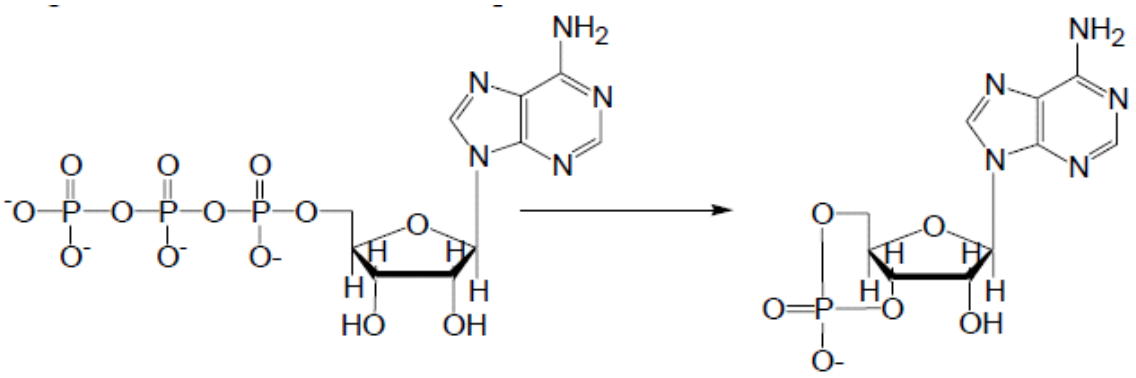


Signaling involving IP_3 and DAG

Q 5

Answer

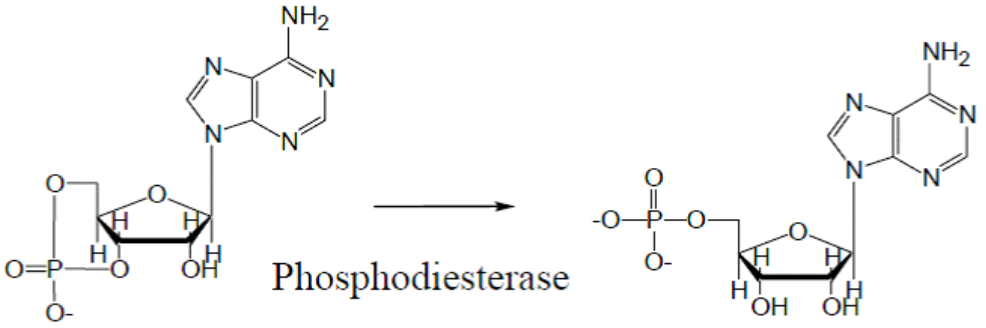
G-proteins are intermediaries in signal transduction. They are called G-proteins because they containing binding sites for guanosine nucleotides. In the case of the β-adrenergic receptor, the resting G-protein is a heterotrimer consisting of α, β and γ subunits. The α subunit contains the guanosine nucleotide binding site. In the resting state, the α subunit has GDP bound and associates with receptors such as the glucagon receptor or the β-adrenergic receptor. The binding of the hormone to the receptor produces allosteric conformational changes that cause the α subunit to release GDP and bind GTP. The binding of GTP is a switch which causes the α subunit to dissociate from the Gβγ dimer. The GTP bound α subunit diffuses laterally through the membrane until is associates with adenylate cyclase. The association of these two proteins activates adenylate cyclase which then starts producing cAMP. A single hormone bound to its receptor can activate 100s of Gα molecules.



Adenylate Cyclase

ATP **cAMP**

Second messengers need to have short half lives so that the response can be rapidly terminated. Phosphodiesterase hydrolyzes the phosphodiester bond to convert cAMP to AMP. The G proteins ultimately need to reset themselves. The Gα subunit has an intrinsic GTPase activity. The bound GTP will slowly be hydrolyzed into GDP and Pi. This GTPase activity is like a built in clock that spontaneously resets the Gα subunit after a short period of time. After the Gα subunit has hydrolyzed GTP it tightly binds the GDP. When the Gα subunit had GDP bound it dissociates from adenylate cyclase turning this enzyme off and reassociates with the Gβγ dimer to reform the heterotrimer. This requires the hormone to be bound to the receptor to keep adenylate cyclase active.

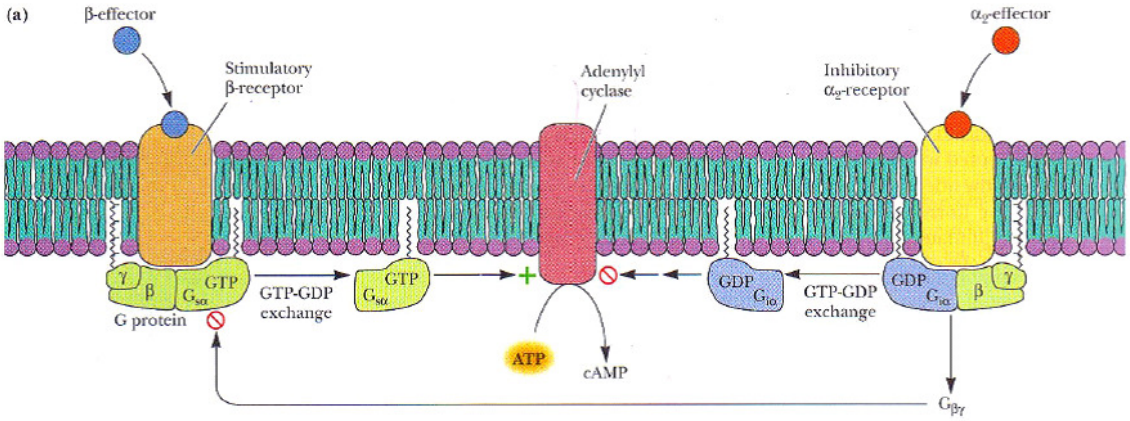


cAMP **AMP**

The typical G-proteins are heterotimers consisting of α, β and γ subunits in the resting state. The binding of the hormone to the receptor causes the exchange of GDP for GTP in the α subunit. The Gα subunit then dissociates from the $G_{\beta\gamma}$ dimer and associates with an effector protein. Eventually the Gα subunit hydrolyzes the GTP, binds the GDP tightly, dissociates from the effector protein and reassociates with the $G_{\beta\gamma}$ dimer to reform the heterotrimer. Glucagon and epinephrine bind to different receptors yet activate the same G protein which in turn activates the same effector protein, adenylate kinase. Other effector proteins activated by other G proteins are phospholipase C, phospholipase A2, potassium channels, sodium channels, calcium channels. There are more than 20 different G-proteins discovered to date. A few are listed to the left.

The hormone-receptor mediated processes regulated by G proteins may be stimulatory as in the example of the epinephrine, β-adrenergic receptor, or inhibitory. Each G-protein interacts with a stimulatory G-protein denoted $G_{\alpha s}$ or with an inhibitory G protein denoted $G_{\alpha i}$. Epinephrine also binds to a α-adrenergic receptor. The α-adrenergic receptor associates with a $G_{\alpha i}$ protein. The binding of epinephrine to the aadrenergic receptor causes the exchange of GDP for GTP causing the $G_{\alpha i}$ to dissociate from the $G_{\beta\gamma i}$ dimer. The inhibition comes from either the Gαi subunit associated with adenylate cyclase to directly

inhibit the cyclase, or by the action of $G_{\beta\gamma i}$ which associates with the Gαs subunit when it has GTP bound. The Gαi thus competes with adenylate cyclase for Gαs.



Signaling involving G protein and cAMP

Q 6

Answer

Tyrosine hydroxylase catalyzes the rate-limiting step in catecholamine synthesis

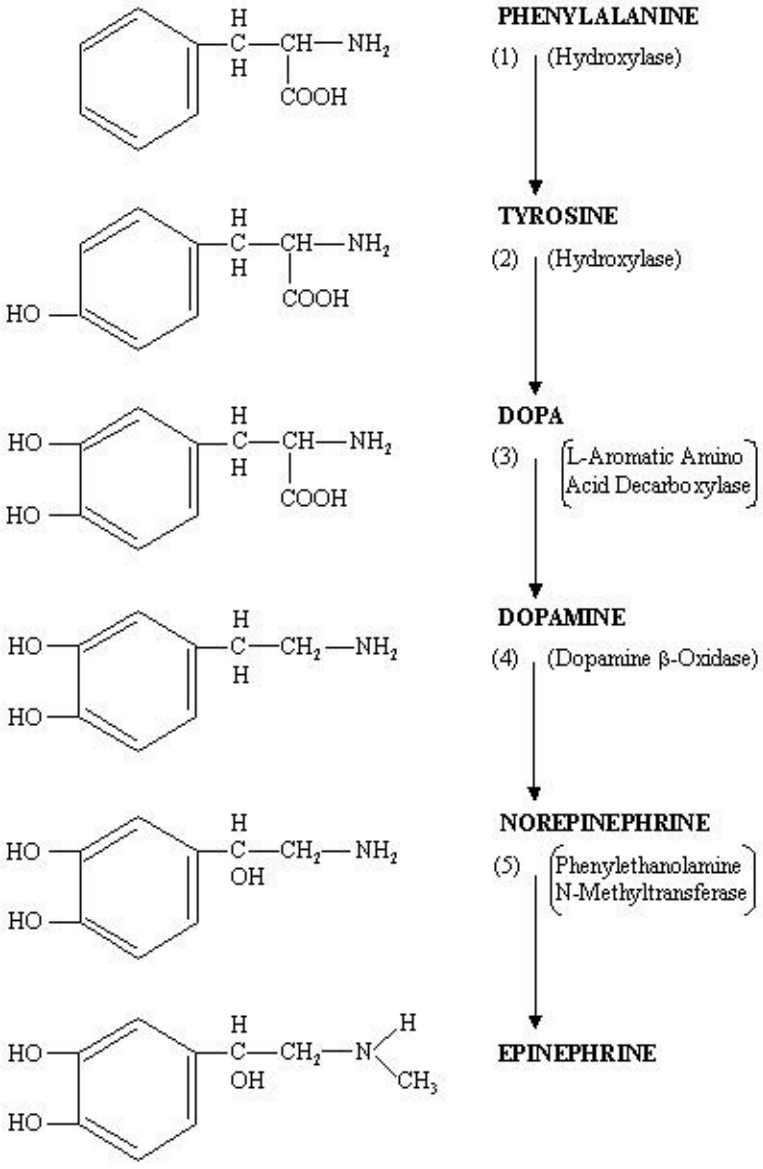
The amino acid, tyrosine –> DOPA –> Dopamine –> Norepinephrine

Tyrosine is obtained from dietary protein and is transported from the blood into the brain.

Each step in the formation of catecholamines depends on a specific enzyme that acts as a catalyst

(an agent that increases the rate of a chemical reaction) for that step.

Tyrosine hydroxylase is the rate limiting enzyme in the pathway because it determines the overall rate of dopamine and norepinephrine formation.


PHENYLALANINE
(1) (Hydroxylase)
TYROSINE
(2) (Hydroxylase)
DOPA
(3) L-Aromatic Amino Acid Decarboxylase
DOPAMINE
(4) (Dopamine β-Oxidase)
NOREPINEPHRINE
(5) Phenylethanolamine N-Methyltransferase
EPINEPHRINE

Pathway of catecholamine biosynthesis

The pathways of catecholamine biosynthesis within CNS, sympathetic post ganglionic neuron and adrenal chromaffin tissues appear to be identical. The number of steps, however, depends on the definitive product i. e. dopamine, norepinephrine or epinephrine to be secreted.

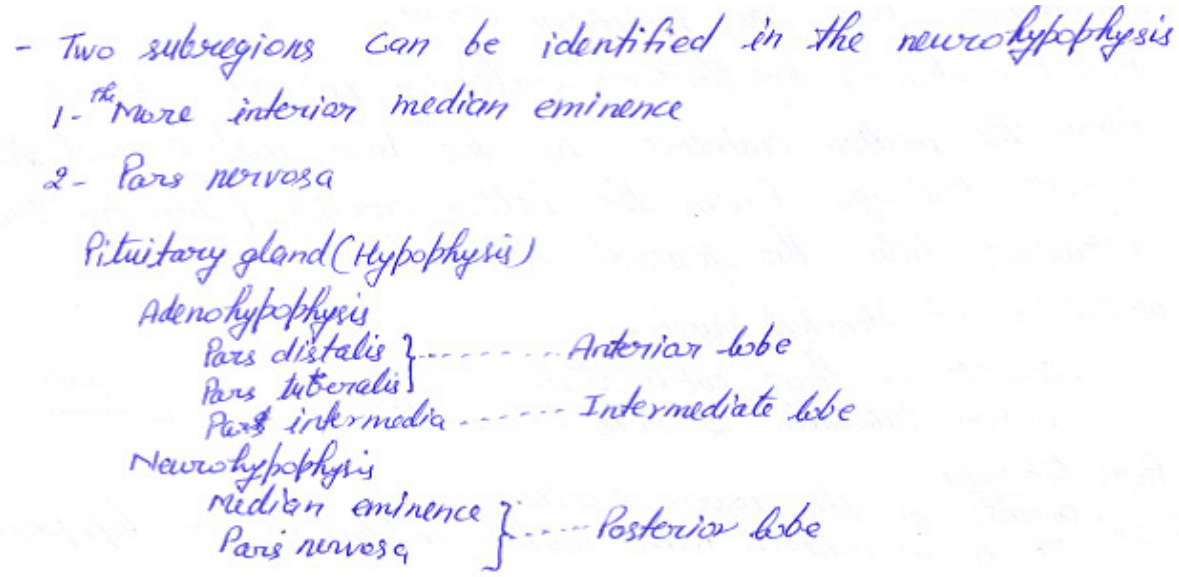
The conversion of tyrosine to epinephrine involves 4 steps-

- Hydroxylation at the 3-position of the phenolic ring
- Side chain decarboxylation
- Side chain hydroxylation
- N-methylation

Norepinephrine is primary amine whereas epinephrine is an N-methylated secondary amine. These differences provide an important structural basis for differing potency of these catecholamines on adrenoceptors.

Q 7

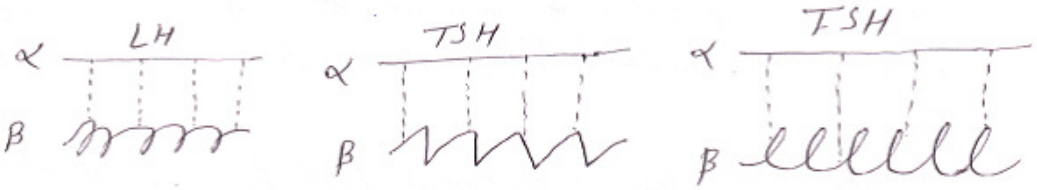
Answer



Pituitary gland secretes tropic hormones that regulate the functions of other endocrine glands such as thyroid, adrenal, testis and ovary. In other words pituitary gland was considered to exert control over other endocrine glands. So, it was considered as master gland. Later attention was shifted to hypothalamus through which pituitary gland is regulated by means of releasing and inhibiting hormones.

Category I Tropic Hormones -

- All mammalian glycoprotein tropic hormones are composed of two peptide subunits. (Mol. wt. ≃ 32 kDa)
- Biological half-~~life~~lives for TSH and LH in mammals are about 60 minutes whereas that of FSH is about 3X longer.
- Each glycoprotein tropic hormones consists of two subunits, an α-subunits and a β-subunit.



- The α-subunit is identical and β-subunit is specific.
- β-subunit is important for unique biological activity.

1) LH : Actions -

- Synthesis of androgens in both males and females is caused by LH action on testes and ovaries.
- LH acts through a G-protein-based, cAMP second messenger system.
- Gamete release (sperm release in males and ovulation in females) also is under control of of LH.
- LH causes formation of corpus luteum from ruptured ovarian follicles remaining after ovulation, and also stimulate corpus luteum to secrete progesterone.

2) FSH : Actions.

- Like LH, FSH binds to a membrane receptor and stimulates cAMP production as a second messenger
- FSH is primarily involved with gamete preparation: that is, ovarian follicle development in females and spermatogenesis in males.

- In females, TSH also stimulates the conversion of androgens into estrogens.

3) TSH : Actions -

- TSH also operates via a cAMP - dependent mechanism to increase synthesis of thyroid hormones, cause release of stored thyroid hormones, and secondarily increase iodide uptake by cells of the thyroid.
- Human may produce a variant of TSHs, one of which is associated with pathological condition known as Graves' disease.
- Normally, TSH has a biological half-life of about 0.25 hours. The so called long-acting thyroid stimulator (LATS) in Grave's' disease has a biological half-life of 7.5 hours.

Category II Tropic Hormones - (PRL & GH)

- PRL and GH are large, single-polypeptide hormones of similar structure
- Both GH and PRL have common effects on osmoregulation (renal function, intestinal fluid absorption), selective tissue growth (prostate gland, sebaceous gland), lactation, and other processes.
- Membrane receptors for GH and PRL are monomeric proteins that span the cell membrane only once.
- They do not activate adenylyl cyclase.

GH -

- GH ~~enhances~~ affects protein metabolism and electrolyte balance. These actions are mediated indirectly through somatomedins (insulin-like growth factors IGFs)

child, whereas overproduction of GH during early postnatal development leads to gigantism.

- In adult, excess GH secretion leads to acromegaly.
- Short stature may result from a pituitary failure of GH production or from a failure of the liver to respond to GH and synthesize somatomedins (Laron syndrome).
- The pathogenesis of acromegali has been explained by either a pituitary or a hypothalamic mechanism.
- In pituitary mechanism view overproduction of growth hormone may result from GH-secreting tumors of adenohypophysis.
- The hypothalamic hypothesis indicates the defect possibly from an overproduction of GHRH or an underproduction of somatostatin (GH release-inhibiting hormone).

PRL -

- Consists of single chain of 199 amino acids.
- It produces a variety of distinctive actions in animals, including effects associated with reproduction, growth, osmoregulation, and integument.
- Furthermore, PRL may produce synergistic actions with ovarian, testicular, thyroid, and adrenal hormones.
- The best-known action for PRL is the lactogenic effect on the mammary gland.
- PRL stimulates DNA synthesis, cellular proliferation, and the synthesis of milk proteins (casein and lactalbumin).
- In some sp. PRL may influence the synthesis of progesterone by corpus luteum. This is why earlier PRL was named as

Category III tropic Hormones -

- This category comprises several hormones derived from the same precursor, a prohormone known as proopiomelanocortin or POMC, and includes ACTH, α-MSH, LPH and β-endorphins.

- In corticotropes of pars distalis ⇒ POMC (cleaved) → N-terminal 16K fragment = No biological role
  → ACTH (39 a.a.)
  → 91 a.a. LPH known as β-LPH (C-terminal)
  (may again be cleaved)
  → β-endorphin (61-91 a.a. of β-LPH)
  → γ-LPH (1-58 a.a. of β-LPH)

- In melanotropes of pars intermedia ⇒ ACTH → α-MSH (residue 1-13 of ACTH)
  → Corticotropin-like peptide (CLIP) (residue 18-39 of ACTH)

1- Corticotropin (ACTH)

- Corticotropin stimulates the adrenal cortex to secrete glucocorticoids (cortisol and/or corticosterone), hormones that alter protein and carbohydrate metabolism.
- ACTH consists of 39 amino acids in a single peptide chain.
- Amino acids 1-23 of ACTH have full biological activity, 1-19 have 80% but fragment ~~1-13~~ 1-16 has very little ACTH activity.

2- Melanotropin (α-MSH)

- In mammals, the epidermal melanin-producing cell is the melanocyte that synthesizes melanin under the influence of α-MSH but extrudes it into the extracellular compartment of

Q 8

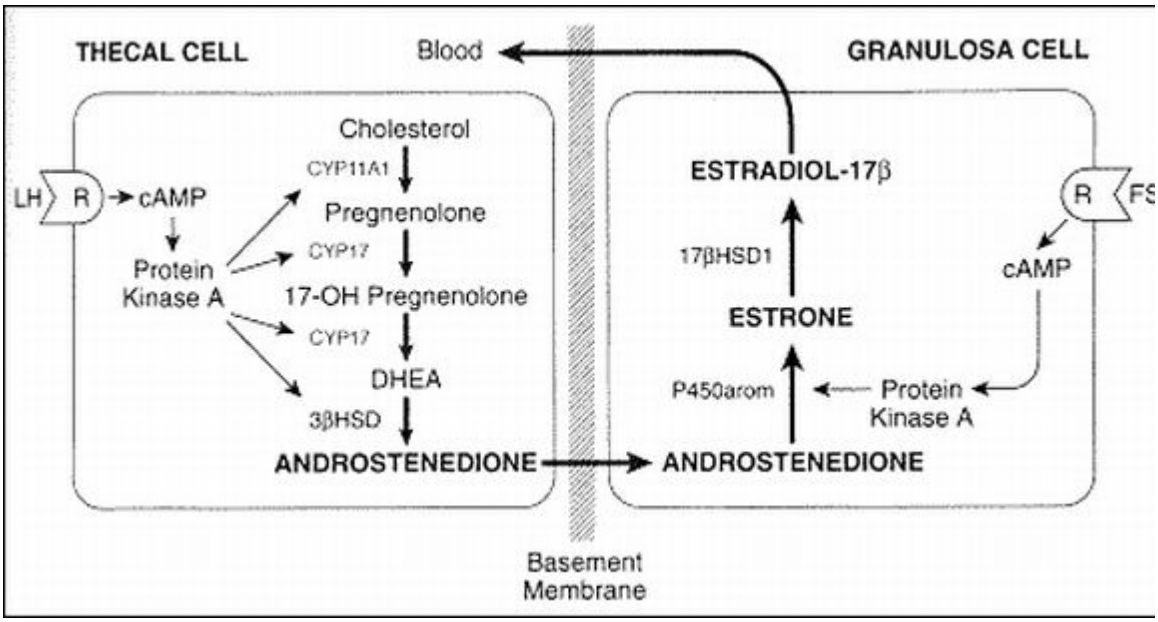
Answer

Ovary performs two functional roles - hormonogenesis & Gametogenesis. Embryonic ovary is populated by about one thousand pr. Germ cells which by rapid division give rise to approx. 3 million oocytes. At birth, however, number reduces to about one million and by puberty only approx. 2,50,000 oocytes are there. Single ovum is ovulated in each cycle, a total of only 400-500 oocytes are released (99.9% are destined for atresia). In embryonic ovary pr. follicle begin reduction division but delayed in prophase. During each ovarian cycle pr. Oocyte completes first meiotic division which gives rise to secondary oocyte and first polar body. Sec. oocyte immediately enters second meiotic division, a process arrested in metaphase unless fertilization occurs. Interstitial tissue adjacent to follicle becomes arranged concentrically around mature follicle to form theca. The follicle cell that ovulates is the one whose granulosa cells acquire high levels of aromatase and LH receptors in response to FSH rise that is the one with lowest FSH "threshold". This follicle increases estradiol (E_2) secretion and E_2 feed back to regulate FSH

secretion negatively. Thus low level of FSH now limits FSH dependent development of other follicles with high FSH threshold. This mature follicle is known as Graafian follicles.

Steroid synthesis by follicle

Follicular phase	Estradiol
Luteal phase and pregnency	Progesterone



Physiological roles of GnRH

Both FSH and LH are required to stimulate ovarian changes leading to ovulation. Pituitary gonadotropin secretion is initiated during pubertal development. Gonadotropin secretion is dependent on GnRH release. Pulsatile secretion of gonadotropin occurs with

a frequency of approximately one pulse per hour (circhorol). Both estrogen and progesterone exert a negative feedback suppression of GnRH pulse generator activity and gonadotropin secretion. Feedback suppression of progesterone inhibits gonadotropin surge. Low levels of estrogen exert negative feedback action. After estrogen levels rise and remain above a critical level for at least 36 hours, negative feedback effect is reversed and a positive feedback ensues, which results in gonadotropin surge. Luteal progesterone secretion then reestablishes negative feedback until demise of the corpus luteum.

Luteinizing hormone

- Synthesis of androgens in both male and female is caused by LH actions on testis and ovary.
- LH acts through GPCR pathway activating production of cAMP.
- Gamete release (sperm release in male and ovulation in female) is also under control of LH.
- LH causes formation of corpus luteum from ruptured follicles remaining after ovulation and also production of progesterone from corpus luteum.

Follicle stimulating hormone

- Like LH, FSH also binds with a membrane receptor and stimulates formation of cAMP as sec. messenger.
- FSH is primarily involved in gamete preparation that is ovarian follicle development in females and spermatogenesis in males.
- In females, FSH also stimulates conversion of androgen to estrogen.