Anatomy and Physiology II Exam 1 Review

1. Mix and Match the endocrine gland location with their name from the mix and match list

2. What glands produce steroid hormones and what glands produce protein type hormones? Which gland produces amino acid hormones that are fat soluble and cross cell membranes. What glands produce fat soluble hormones and what is the significance of hormones being fat soluble?

Gland	Hormone Type	Fat Soluble
Hypothalamus	Peptide	No
Pituitary	Protein	No
	Peptide	No
Pineal	Amino acid	No
Thyroid	Amino acid	(T3 & T4)Yes
Parathyroid	Peptide	No
Thymus	Peptide	No
Adrenal	Steroids	Yes
	Amino acid	No
Pancreas	Protein	No
Digestive System	Protein & Peptide	No
Testes / Ovaries	Steroid (One Protein in ovaries)	Steroid Yes Protein No

3. Both nerves and hormones produce signals that control body function. Contrast the signals from nerves and hormones with respect to their speed and how long the affects of the signal last.

Hormones: Responses take longer then to nerve signals, duration is also longer. Used for slow responses like onset of puberty.

Nerve signals: Responses are rapid, duration is very short like a pain signal. Used for rapid response with a short duration.

4. Explain in detail how protein or steroid type hormones work at the cellular level.

Steroid:

- **Fat soluble and readily crosses cell membranes.**
- **Produced by:**
  - **Adrenal cortex**
  - **Testes**
  - **Ovaries**
  - **The thyroid hormones (T3 & T4) also work like steroid hormones**

- **How work:**
  - **Move across cell membrane and into nucleus of cell**
  - **Bond with protein receptors in nucleus**
  - **The steroid-protein complex then bonds with regulatory segments on the DNA and acts by turning genes on/off. This causes them to control the synthesis of proteins.**

Protein (also amino acid & peptide):

- **Long chains of amino acids, not fat soluble**
- **Produced by:**
  - **Anterior pituitary**
  - **Pancreas**
  - **Digestive System**
  - **Ovaries(one only)**
- **How work:**
  - **Hormone bonds to G-protein receptor site on cell membrane**
  - **Adenylate cyclase is activated, convertes ATP to C-AMP. NOTE: cyclic guanosine monophosphate (C-GMP) is another secondary messenger used by some hormones.**
  - **C-AMP activates Kinase "A"**
  - **Kinase "A" catalyzes the addition of phosphates to enzymes, activating the enzyme in the cell**
  - **The activated enzyme changes the cell functions**
  - **The enzyme calmodulin is also activated and it converts the C-AMP back to ATP, aiding in stopping the effect of the hormone.**

5. For the following hormones, know where they are produced, the target tissues they affect, what effects they have, and how they are regulated. Know the hormone related diseases and their symptoms.

Pituitary gland and Hypothalamus gland interaction

Anterior Pituitary Gland	Hypothalamus Release Trigger	Hypothalamus Inhibition Trigger	Tissue And Effect	Diseases
hGH	GHRH	GHIH	Blood sugar levels, protein synthesis, etc.	Dwarfism, Giantism, Acromegaly
FSH + LH	GnRH ↑	GnRH ↓	Body maturation, follicle development in ovaries, sperm production in testes	

PRL	PRH	PIH	Initiates milk production in women, unclear in men	

Other pituitary gland hormones

Pituitary	Effect	Notes
TSH (anterior)	Thyroid increases production of T3& T4 hormones	
ACTH (anterior)	Adrenal cortex gland to release glucocorticoids (cortisol, cortisone, and corticosterone)	
MSH (anterior)	Melanocytes increase productivity of melanin	Triggered by UV exposure.
Oxytocin/OT (Posterior)	Contracts uterus during birth, contracts mammary glands during breast feeding	
ADH (Posterior)	Causes body to retain water (blood plasma).	Disease: Diabetes Insipidus

[gland] Hormone	Effect	Releaes	Inhibition	Disease/Notes
[pineal] Melatonin	High levels help you go to sleep	Night: Darkness reduces the levels of norepinephrine, reducing the inhabitation of melatonin	Day: Photoreceptors on eyes cause the sympathetic nervous system to produce norepinehrine, inhibiting the release of melatonin	
[Thyroid] T3& T4	↑ basal metabolic rate & O_2 usage ↑ cellular metabolism ↑ growth & development ↑ use of glucose for ATP creation ↑ protein synthysis ↑ actions of epinephrine & norepinephrine through up regulation of beta receptors	Trigger: Low blood levels of T3 & T4 1. Hypothalamus releases TRH 2. Pituitary releases TSH 3. Thyroid releases T3 & T4 Effect: ↑ blood lvl of T3 & T4	Trigger: High blood lvl of T3 & T4 1. Hypothalamus inhibits release of TRH 2. Pituitary inhibits release of TSH 3. Thyroid reduces releasing T3 & T4 Effect: ↓ blood lvl of T3 & T4	Cretinism, Myxedema, Graves disease, Goiter T3 = triiodothyronine (3 iodine) T4 = Tetraiodothyronine (4 iodine)
[Thyroid] Calcitonin (CT)	↓ blood Ca^{++} and HPO_4^{2-} lvls ↑ Activity of osteocytes (bone production) ↓ Activity of osteoclasts (bone	↑ blood lvls of Ca^{++}	↓Blood lvls of Ca^{++}	

	breakdown)			
[Parathyroid] Parathyroid (PTH)	↑ blood Ca^{++} and HPO_4^{2-} lvls ↓ Activity of osteocytes (bone production) ↑ Activity of osteoclasts (bone breakdown) ↑ reabsorption of Ca^{++} and Mg^{++} in the kidney Promotes the formation of hormone calcitriol from Vitamin D in the kidneys. Calcitriol increases the absorption of Ca^{++}, HPO_4^{2-}, and Mg^{++} in the gut.	↓Blood lvls of Ca^{++}	↑ blood lvls of Ca^{++}	Hypoparathyroidism
[Thymus] Thymosin, Thymic hormonal factor (THF), Thymic factor (TF), and Thymopoietin	All of these hormones promote the maturation and development of immune system cells (i.e. T-cells and B-cells).			
[Adrenal Cortex - Mineralocorticoid group]	Triggers the reabsorption of Na^{++} from the	Trigger: Angiotensin II	Trigger: Low Angiotensin II	

Aldosterone	urine to the blood in the kidneys. Cl^- and HCO_3^- are reabsorbed due to the osmotic gradient created by moving the ions above. This increases blood pressure. H^+ excretion to the urine is promoted, preventing blood acidosis.	Steps: 1. Low blood pressure reduces the filtration rate at the kidneys, reducing urine levels of Cl^- 2. Low lvls of Cl^- in the urine trigger the juxtaglomerular apparatus (located in the distal convoluted tubule of the kidney) to release rennin. 3. Renin causes the plasma protein angiotensinogen to form angiotensin I 4. Angiotensin I moves through the blood to the lungs where the enzyme angiotensin converting enzyme (ACE) converts it to angiotensin II 5. Angiotensin II	Other aldosterone release mechanisms: - High blood K^+ or low blood Na^+ can also trigger aldosterone release - Low blood K^+ or high blood Na^+ can inhibit aldosterone release.	

		triggers aldosterone release from the adrenal cortex and causes vasoconstriction increasing the blood pressure. 6. Increased blood pressure increases the filtration rate at the kidneys increasing the amount of Cl^- in the urine and hence reducing the release of rennin.		
[Adrenal cortex – Glucocorticoid group] Cortisol (hydrocortisone), Corticosterone, Cortisone	↑ protein catabolism in muscle to increase blood amino acid lvls for production of enzymes ↑ use of amino acids or lactic acids in liver to make glucose (i.e. gluconeogenesis making of glucose from other then	Trigger: Low blood lvls of glucocorticoids 1. Hypothalamus: releases CRH 2. Pituitary: releases ACTH 3. Adrenal cortex: releases glucocorticoids Effect: Blood lvls of glucocorticoids increase	Trigger: High blood lvls of glucocorticoids 1. Hypothalamus: inhibits release of CRH 2. Pituitary: inhibits release of ACTH Effect: Blood lvls of glucocorticoids decrease	NOTE: CRH is also released in response to emotional or physical stressors that take the body away from homeostasis (general adaptation syndrome). Reduces the bodies immune response to deal with some immediate

	glycogens) ↑ lipolysis – break down of triglycerides and release of fatty acids ↑ blood glucose lvls ↑ sensitivity of blood vessels to compounds that cause vasoconstriction ↓ inflammatory response ↓ number of mast cells ↓ reduce histamine release ↓ capillary permeability ↓ phagocytosis of dead and foreign cells ↓ connective tissue formation ↓ immune response			stressor. Explains how stressors increase disease. Treatment uses: 1. Hydrocortisone (cortisol) is used to treat chronic inflammation. 2. Glucocorticoids are used to reduce the immune response in patients that have had an organ transplant, reduces the rejections. Disease: Addison's disease, Cushing's syndrome, Hyperglycemia, Osteoporosis, Poor wound healing, Mood swings
[Adrenal cortex – Androgen group] Dehydroepiandosterone (DHEA) Androgens are male sex hormones, two main are testosterone	In Men causes the development of secondary sex characteristics. In Women DHEA is converted into estrogen by	It is unclear how DHEA is regulated but its release appears to be triggered by ACTH		Congenital adrenal hyperplasia

from the testes and dehydroepiandosterone produced by the adrenal cortex	various body tissues and can be an important source of estrogen in post menopausal women. May also contribute to sex drive in women. In prepuberty boys and girls will cause growth spurts and growth of pubic hair			
[Adrenal medulla] epinephrine (adrenaline) norepinephrine (NE)	Same effect as the sympathetic nervous system	Stimulated by the sympathetic preganglionic nerves when the sympathetic nervous system is activated. Most (80%) of the hormone released is epinephrine		
[Pancreas – Islets of Langerhans – Alpha cells] Glucagon	Increases blood sugar lvl by causing the liver to convert glycogen to glucose Promotes the formation of glucose in the liver from amino	Low blood sugar lvl stimulates the release of glucagons	High blood sugar lvl inhibits the release of glucagons	

	acids and lactic acid (gluconeogenesis)			
[Pancreas – Islets of Langerhans – Beta cells] Insulin	Decreases blood sugar lvls by causing the liver to convert glucose to glycogen Accelerates facilitated diffusion of glucose into cells, particularly skeletal muscle Increases movement of amino acids into cells and promotes protein synthesis Increases synthesis of fatty acids	High blood sugar lvls stimulates insulin release	Low blood sugar lvls inhibits insulin release	Other factors which may cause insulin release: 1. Acetylcholine from parasympathetic vegas nerve stimulates the release of insulin 2. Food high in carbohydrates raise blood sugar triggering insulin release 3. Food high in proteins increase blood levels of the amino acids, arginine and leucine, triggering insulin release. 4. hGH and ACTH stimulate insulin release because they raise blood sugar lvls. Diseases: Diabetes mellitus (Type I & Type II)

[Pancreas – Islets of Langerhans – Delta cells] Somatostatin	Inhibits the secretion of insulin and glucagons Slows the absorption of nutrients from the gut Overall reduces digestion	Low lvls of pancreatic polypeptide increase the release of somatostatin	High lvls of pancreatic polypeptide inhibit the release of somatostatin	
[Pancreas – Islets of Langerhans – F-cells] Pancreatic polypeptide	Inhibits release of somatostatin Causes contraction of the gallbladder and secretion of pancreatic digestive enzymes Overall increases digestion	Acute hypoglycemia Fasting Exercise Diets high in protein	High blood glucose High somatostatin	Note: Usually the release triggers override the inhibitory effect of somatostatin
[Eicosanoids] Prostaglandins	Many effects, act somewhat similar to neurotransmitters	Some are released when tissues are damaged or irritated, triggering the nerve signals that cause pain	Asprin and Ibuprofen inhibit prostaglandins	

Hormone Related Diseases

Hormone	Disease	Details
Dwarfism	hGH	Hyposecretion of hGH before puberty. Causes person to be short.
Giantism	hGH	Hypersecretion of hGH before puberty. Causes person to be tall.
Acromegaly	hGH	Hypersecretion of hGH after puberty. Causes bones to enlarge in diameter because they can no longer grow in length.
Diabetes insipidus	ADH	Brain damage causes low ADH lvls or the kidney receptors for ADH do not respond. SxS: This causes the person to produce large amounts of dilute urine, dehydrating the body.
Cretinism	T3 & T4	Low T3/T4 lvls during development causes mental retardation and dwarfism
Myxedema	T3 & T4	Low T3/T4 in adults causes the following: SxS: Edema of the face Slow heart beat, low body temp, low metabolic rate Dry skin and hair Muscular weakness Mental dullness
Graves Disease	T3 & T4	An autoimmune disease where immunoglobulins mimic the actions of TSH. SxS: Exophtyalmos (eyes protrude) Thyroid gland 2x-3x normal size Increased metabolic rate Increased heart rate Weight loss despite a good appetite Insomnia No heat tolerance (i.e. body overheats easily)
Goiter	T3 & T4	Excessive growth of the thyroid gland usually caused by a diet low in iodine

Hypoparathyroidism	PTH	Low blood PTH lvl due to parathyroid dmg, causes blood calcium lvls to be abnormally low. This reduces extracellular Ca++ to the point that skeletal muscle can depolarize and contract. It causes tetany (i.e. sustained contraction) in skeletal muscle because most of the calcium for contractions is stored in the sarcoplasmic reticulum. In smooth muscle and cardiac muscle it can cause depolarization and contraction, but it reduces the force of contractions because most of the calcium that causes muscle contractions come from outside the cell. These effects can cause serious heart arrhythmia to occur.
Addison's Disease	Glucocorticoid group of the Adrenal cortex hormones	Hyposecretion of glucocoticoids which causes the following SxS: Mental dullness Anorexia Nausea Weight loss Hypoglycemia Muscle weakness Low aldosterone ACTH may be high due to a lack of negative feedback and it may mimic the effects of melanin stimulating hormone, increasing skin pigmentation
Cushing's Syndrome	Glucocorticoid group of the Adrenal cortex hormones	Hypersecretion of glucocorticoids which causes the following Sxs: Fat redistribution: fat puffy face and thin spindly arms and legs due to muscle catabolism Buffalo hump on back Large pendulous hanging abdomen Flushed facial skin Hyperglycemia Osteoporosis Poor wound healing Mood swings
Congenital adrenal	Androgen group	Hypersecretion of DHEA which causes the following SxS in

hyperplasia	of the adrenal cortex hormones	women: Deep voice Baldness Atrophy of the breast Infrequent menstruation This disease has little effect on adult men. In boys it causes early development of secondary sex characteristics.
Diabetes mellitus	Insulin	A disease caused by improper regulation of insulin with the following SxS: High blood sugar High sugar in the urine Excessive thirst (polydipsia) Excessive eating (polyphagia)
Diabetes mellitus Type I AKA Juvenile onset	Insulin	Autoimmune disease that leads to the destruction of beta cells. Usually occurs in young people. Because glucose doesn't move into the cells, cells burn fatty acids raising the levels of blood ketones causing ketoacidosis (low blood pH). Treatment – inject insulin Insulin shock – too much insulin injected, blood sugar lvl drops too low.
Diabetes mellitus Type II AKA Adult onset	Insulin	Person has the proper amount of insulin in the blood stream, but the target cells in the body are less sensitive to insulin due to down regulation. This type of diabetes represents 90% of cases and usually can be controlled by exercise and loss of weight