

Produced: Anterior Pituitary Gland – Human Growth Hormone (hGH) Related: Growth Hormone Release Hormone (GHRH) – hypothalamus Related: Growth Hormone Inhibiting Hormone (GHIH) – hypothalamus Target Tissue & Effect: Increased Cell division; Increased protein synthesis by triggering IGFs; Stimulates the breakdown of fats for ATP; Reduces use of glucose for ATP by reducing uptake of glucose by cells, causes increase in blood glucose levels and increase in insulin levels. <u>Regulation – Release:</u> Trigger: Low blood sugar 1. Hypothalamus: Releases GHRH (GHIH inhibited) 2. Pituitary: Releases hGH Effect: Blood sugar rises Regulation – Inhibition: Trigger: High blood sugar 1. Hypothalamus: Releases GHIH ((GHRH inhibited) 2. Pituitary: Inhibits release of hGH Effect: Blood sugar drops Diseases: Dwarfism, Giantism, Acromegaly Produced: Anterior Pituitary Gland - Follicle stimulating hormone (FSH) / Luteinizing hormone (LH) Related: Gonadotropin releasing hormone (GnRH) - hypothalamus Target Tissue & Effect: In women it promotes the development of follicles in the ovaries (FSH) and ovulation (LH); In men it initiates sperm production in the testes (FSH) and release (LH). Regulation (Women) – Release: Trigger: High levels of estrogen in the blood from developing follicles. 1. Hypothalamus: Releases GnRH 2. Pituitary: Releases FSH & LH Effect: Follicles n the ovaries develop, upon maturation they produce a hormone inhibin which inhibits the pituitary from releasing FSH and to a minor extend LH. Higher LH levels then FSH levels eventually cause ovulation and what is left of the follicle in the ovary is called a corpus luteum and produces estrogen and progesterone. Regulation (Women(– Inhibition: Trigger: High levels of estrogen and progesterone in blood from the corpus luteum. 1. Hypothalamus: Inhibits GnRH 2. Pituitary: Inhibits release of FSH & LH Effect: Follicle development and ovulation is inhibited. Diseases:



<u>Produced:</u> Thyroid Gland – Triiodothyronine (T3) & Tetraiodothyronine (T4) [AA]
<u>Related:</u> Anterior Pituitary Gland – Thyroid Stimulating Hormone (TSH)
<u>Related:</u> Thyrotropin releasing hormone (TRH)
<u>Target Tissue & Effect:</u> ↑ Following: Basal metabolic rate & O₂ use, cellular metabolism, growth and development, use of glucose for ATP, protein synthesis, lipolysis and cholesterol excretion into bile, actions of norepinephrine and epinephrine by up regulation of beta receptors.
<u>Regulation – Release:</u> Trigger: Low T3/T4 blood levels
1. Hypothalamus: Releases TRH
2. Pituitary: Releases TSH
3. Thalamus: Releases T4 & T3

Effect: Blood levels of T3/T4 increase

<u>Regulation – Inhibition:</u>

Trigger: High T3/T4 blood levels

1. Hypothalamus: Inhibits release of TRH

2. Pituitary: Inhibits release of TSH

3. Thalamus: Inhibits release of T3/T4

Effect: Blood levels of T3/T4 decline

Diseases: Cretinism, Myxedema, Graves Disease, Goiter

<u>Produced:</u> Anterior Pituitary Gland – Prolactin (PRL)

<u>Related</u>: Hypothalamus – Prolactin Releasing Hormone (PRH)

Related: Hypothalamus - Prolactin Inhibiting Hormone (PIH)

Target Tissue & Effect: (Women) initiates milk production (in association with other hormones released during pregnancy: estrogen, progesterone, glucocorticoids, hGH, thyroxine, and insulin); (Men) function is unclear however it is known that high levels lead to impotency and infertility.

Regulation - Release:

Trigger: Pregnancy or the mechanical action of a baby nursing

1. Hypothalamus: Releases PRH (Inhibits PIH)

2. Pituitary: Releases PRL

Effect: Milk production increases

<u>Regulation – Inhibition:</u>

Trigger: Not breast feeding baby

1. Hypothalamus: Releases PIH (Inhibits PRH)

2. Pituitary: Inhibits PRL

Effect: Milk production decreases/stops.

Diseases:



Produced: Anterior Pituitary Gland – Melanocyte Stimulating Hormone (MSH) Related: Hypothalamus – Corticotropin Releasing Hormone (CRH) Target Tissue & Effect: [Melanocytes] Increase production of melanin, increasing skin pigmentation. Regulation – Release: Trigger: Exposure to sunlight (UV radiation) 1. Hypothalamus: Releases CRH 2. Pituitary: Releases MSH Effect: Melanocytes produce more melanin, skin becomes darker Regulation – Inhibition: Trigger: Lack/less exposure to sunlight 1. Hypothalamus: Inhibits release of CRH 2. Pituitary: Inhibits release of MSH Effect: Melanocytes produce less melanin, skin becomes lighter Disease: Produced: Anterior Pituitary Gland – Oxytocin (OT) Related: None Target Tissue & Effect: [Uterus & Mammary Glands] During birth causes stronger contractions of uterus, after birth causes ejection of milk from mammary glands Regulation – Release: Trigger: [Birth] stretching of the cervix 1. Pituitary: Releases OT Effect: Stronger contractions, more release of OT, POSITIVE FEEDBACK LOOP <u>Regulation – Release:</u> Trigger: [After Birth] Mechanical act of breast feeding 1. Pituitary: Releases OT Effect: Ejection of milk from breast Disease:



<u>Produced</u>: Anterior Pituitary Gland – AntiDiuretic Hormone (ADH) [Vasopressin] Related: None

<u>Target Tissue & Effect</u>: [Kidney Tubules] Causes the body to retain water (i.e. increase blood plasma) by increasing the permeability of the kidney tubules to water, water is reabsorbed from urine into blood plasma

<u>Regulation – Release:</u>

Trigger: Osmoreceptors in the hypothalamus sense low blood water content

1. Pituitary: Releases ADH

Effect: Increases levels of ADH, kidney tubules become more permeable, blood plasma levels rise.

<u>Regulation – Inhibition:</u>

Trigger: High blood plasma levels detected by hypothalamus

1. Pituitary: Inhibit release of ADH

Effect: Decreased lvls of ADH, kidney tubules become less permeable, blood plasma lvls decrease.

Disease: Diabetes insipidus

Produced: Pineal Gland – Melatonin [AA]

Related: Norepinephrine

Target Tissue & Effect: High levels of melatonin causes you to sleep

<u>Regulation – Release:</u>

Trigger: Dark/night – Photoreceptors in eyes detect less light, decrease production of norepinephrine, less norepinehrine allows the pineal gland to produce more melatonin Effect: Increased melatonin levels, more sleepy

<u>Regulation – Inhibition:</u>

Trigger: Day/light – Photoreceptors in eyes detect light, cause norepinephrine to be produced which inhibits the production of melatonin by the pineal gland.

Effect: Lower melatonin levels, more awake and alert.

Disease:



Produced: Thyroid – Calcitonin (CT) [AA] Produced: Parathyroid – ParaThyroid Hormone (PTH) [Pep] Target Tissue & Effect: Regulate blood Ca⁺² (and HPO₄⁻², Mg⁺²) levels and bone development Regulation – Release CT / Inhibition PTH: Trigger: High blood Ca⁺² levels 1a. Thyroid: Release CT 1b. Parathyroid: Inhibit PTH Effect:

Osteocytes ossification of bone (using calcium and phosphate);

Osteoclast breaking down bone. Regulation – Inhibition CT / Release PTH: Trigger: Low blood Ca⁺² levels 1a. Thyroid: Inhibit CT 1b. Parathyroid: Release PTH Effect: Use Osteocytes ossification of bone (using calcium and phosphate); tosteoclast breaking down bone, reabsorption of calcium and magnesium in the kidneys and promotes the formation of the hormone calcitriol from Vitamin D in the kidney. Calcitriol increases the absorption of Ca⁺², HPO₄⁻², and Mg⁺² in the gut. Disease: Hypoparathyroidism (PTH)

<u>Produced</u>: Thymus Gland – Thymosin, Thymic Hormone Factor (THF), Thymic Factor (TF), Thymopoietin

Related:

<u>Target Tissue & Effect:</u> [Immune System – T-Cells & B-Cells] All of these hormones promote the maturation and development of immune system cells (T-Cells & B-Cells).

<u>Regulation – Release:</u>

Regulation – Inhibition:

Disease:



<u>Produced</u>: Adrenal cortex / Mineralocorticoid group – Aldosterone [S/FS] <u>Related</u>: None

<u>Tissue Type & Effect:</u> [Kidneys] Triggers the reabsorption of Na⁺ from the urine in kidneys; Cl⁻ and HCO₃⁻ are reabsorbed due to the potential gradient created by reabsorbing Na⁺; Water is 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.

<u>Regulation – Release:</u>

Trigger: Angiotensin II

1. Low blood pressure reduces filtration rate at kidneys, reducing urine levels of Cl⁻

2. Low Cl⁻ in urine triggers the **juxtaglomerular apparatus** [distal convoluted tubule of kidney] to release **renin**

3. Renin causes the plasma protein angiotensinogen to form angiotensin I

4. Angiotensin I moves through the blood -> lungs where angiotensin converting enzyme (ACE) converts it to Angiotensin II

5. Angiotensin II triggers Adrenal cortex to release aldosterone.

Effect: Vasoconstriction, increased blood pressure, increased Cl⁻ in urine, reduced release of resin.

Other release mechanisms:

High blood K^{+} or low blood Na^{+} can also trigger aldosterone release

Low blood K^+ or high blood Na⁺ can also inhibit aldosterone release

Diseases:

<u>Produced</u>: Adrenal cortex / Glucocorticoid group – Cortisol (hydrocortisone), Corticosterone, Cortisone [S/FS]

Related: Hypothalamus - CRH

<u>Related</u>: Pituitary - ACTH

<u>Tissue Type & Effect</u>: (following) protein catabolism in muscle (increase blood amino acid lvls to make enzymes), use of amino acids or lactic acid in liver to make glucose (**gluconeogenesis**), lipolysis, blood glucose, sensitivity of blood vessels to compounds that cause vasoconstriction; \downarrow inflamatory response (reduce # mast cells, reducing histamine

release), stabilizing lysosomal membranes, reducing the break down of damaged cells,

↓capillary permeability, ↓phagocytosis of dead/foreign cells, ↓connective tissue formation, ↓immune system responses

Regulation – [Release/Inhibit]:

Trigger: blood levels of glucocorticoids

1. Hypothalamus: Releases/Inhibit CRH

2. Pituitary: Releases/Inhibits ACTH

3. Adrenal Cortex: Releases/Inhibits glucocorticoids

Effect: Blood lvls of glucocorticoids rise/decrease

Diseases: Addison's Disease, Cushings Syndrome,

<u>Treatment Uses</u>: Hydrocortisone (cortisol) for chronic inflammation, Glucocorticoids to reduce immune response in patients with organ transplants (reduce rejection)

Note: CRH is also released in response to emotional or physical stress (General adaptation

syndrome) which reduces the body's immune system, thus stressors \uparrow disease.



<u>Produced</u>: Adrenal cortex / androgen group (male sex hormones) – Dehydroepiandosterone (DHEA) [S/FS]

<u>Related</u>: Pituitary – Adrenocorticotropic Hormone (ACTH)

Tissue Type & Effect: [Men] causes the secondary sex characteristics to develop; [Women] converted to estrogen, important source of estrogen in post menopausal women. May also contribute to sex drive in women. [Prepuberty boys and girls] causes growth spurts & pubic hair growth

Regulation:

It is unclear how DHEA is regulated, but it's release appears to be triggered by ACTH <u>Diseases</u>: Congenital adrenal hyperplasia,

<u>Produced</u>: Adrenal medulla – Epinephrine (adrenalin), Norepinephrine (NE) [AA] Related:

<u>Tissue Type & Effect</u>: Both of these hormones have the same effect as the sympathetic nervous system.

Regulation:

Release of these hormones is stimulated by the sympathetic preganglionic nerves when the sympathetic nervous system is activated. Most of the hormone released is epinephrine (80%) <u>Diseases</u>:



Produced: Pancreas / Islets of Langerhans / Alpha cells – Glucagon [protein]
Related:
<u>Tissue Type & Effect</u> : [liver] Increase blood sugar by causing the liver to convert glycogen to
glucose & promotes the formation of glucose in the liver from amino acids and lactic acid
(gluconeogenesis).
Regulation – Release:
Trigger: Low blood sugar
<u>Regulation – Inhibition:</u>
Trigger: High blood sugar
Diseases:
Produced: Pancreas / Islets of Langernans / Beta cells – Insulin [protein]
<u>Issue Type & Effect:</u> [liver & skeletal muscle / all]Decreases blood sugar by causing the liver
to convert glucose into 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.
<u>Regulation – Release:</u>
Irigger: High blood sugar
Regulation – Inhibition.
Trigger: Low blood sugar
Trigger: Low blood sugar <u>Diseases</u> : Diabetes mellitus (Type I & II)
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Produced: Pancreas / Islets of Langerhans / Delta cells – Somatostatin [protein]
Related:
<u>Tissue Type & Effect</u> : Inhibits the secretion of insulin and glucagons, slows the absorption of
nutrients from the gut (reduces digestion)
Regulation – Release:
Trigger: Low lvls of pancreatic polypeptide
Regulation – Inhibition:
Trigger: High lyls of pancreatic polypeptide
Diseases:
Produced: Pancreas / Islets of Langerhans / F-cells – Pancreatic polypeptide [protein]
Related:
<u>Tissue Type & Effect</u> : Inhibits release of somatostatin, causes contraction of the gallbladder
and secretion of pancreatic digestive enzymes (insulin & glucagons). Overall increases
digestion.
<u>Regulation – Release:</u>
Triggers: 1. Acute hypoglycemia, 2. Fasting, 3. Exercise, 4. Diets high in protein
Regulation – Inhibition:
Triggers: 1. High blood glucose, 2. High somatostatin
Note: usually the release triggers for pancreatic polypeptide hormone override the inhibitory
effects of somatostatin.
Diseases:



<u>Produced</u>: Eicosanoids (like hormones, cause effect on same tissue) Prostaglandins <u>Related</u>: Pain neurotransmitters

<u>Tissue Type & Effect:</u> Many effects and act similar to neurotransmitters <u>Regulation – Known Release:</u>

When tissues are damaged or irritated, trigger nerve signals that cause pain sensation Asprin and Ibuprofen (motrin) inhibit prostaglandins, reduce nerve signals associated with pain.

<u>Produced</u>: Eicosanoids (like hormones, cause effect on same tissue) Leukotrienes <u>Related</u>: inflammation, havfever and asthma <u>Tissue Type & Effect</u>: <u>Regulation</u>: Diseases:



Disease: Dwarfism
Hormone: hGH
Details:
Hyposecretion of hGH before puberty. Causes person to be short.

<u>Disease</u>: Giantism <u>Hormone</u>: hGH <u>Details</u>: Hypersecretion of hGH before puberty. Causes person to be tall.



Disease: Acromegaly Hormone: hGH

Details:

Hypersecretion of hGH after puberty. Causes bones to enlarge in diameter because they can no longer grow in length.

<u>Disease</u>: Diabetes insipidus <u>Hormone</u>: ADH <u>Details</u>: Brain damage causes low AD

Brain damage causes low ADH levels or the kidney receptors for ADH do not respond. <u>SxS:</u> produces large amounts of dilute urine and is dehydrated.



Disease: Cretinism
Hormone: T3/T4
Details:
Low T3/T4 levels during development cause mental retardation and dwarfism

Disease: Myxedema Hormone: T3/T4 Details: $\overline{\text{Low T3/T4}}$ levels in adults cause the following: <u>SxS:</u>

- Edema of the face -
- Slow heart beat, low body temp, low metabolic rate Dry skin & hair -
- -
- Muscular weakness -
- Mental dullness -



<u>Disease</u>: Graves Disease <u>Hormone</u>: T3/T4

Details:

An autoimmune disease where immunoglobulins mimic the actions of TSH. <u>SxS:</u>

- 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)

<u>Disease</u>: Goiter <u>Hormone</u>: T3/T4 <u>Details</u>: Excessive growth of the thyroid gland usually caused by a diet low in iodine.



Disease: Hypoparathyroidism Hormone: PTH

Details:

Low blood PTH levels due to parathyroid damage, causes blood calcium levels to be abnormally low. This reduces extracellular Ca^{+2} to the point that skeletal muscle can depolarize and contract. It causes tetany (i.e. sustained contraction) in skeletal muscle because most of the Ca^{+2} 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 these contractions because most of the Ca^{+2} that causes muscle contractions comes from outside the cell.

These effects can cause serious heart arrhythmia to occur.

<u>Disease</u>: Addison's Disease <u>Hormone</u>: Glucocorticoid group of Adrenal cortex hormones <u>Details</u>:

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



<u>Disease</u>: Cushing's Syndrome <u>Hormone</u>: Glucocorticoid group of Adrenal cortex hormones <u>Details</u>: Hypersecretion of glucocorticoids which causes the following:

<u>SxS</u>:

- Fat redistribution: fat puffy face and thin spindly arms/legs due to muscle catabolism
- Buffalo hump on back
- Large pendulous hanging abdomen
- Flushed facial skin
- Hyperglycemia
- Osteoporosis
- Poor wound healing
- Mood swings

<u>Disease</u>: Congenital Adrenal Hyperplasia <u>Hormone</u>: Androgen group of Adrenal cortex hormones <u>Details</u>: Hypersecretion of DHEA which causes the following (w

Hypersecretion of DHEA which causes the following (women): $\tilde{a} = \tilde{a}$

- <u>SxS</u>:
 - 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.



Disease: Diabetes Mellitus (Type I) Juvenile onset
Hormone: Insulin
Details:
A disease caused by improper regulation of 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 levels drop too low.
Disease: Diabetes Mellitus (Type II) Adult onset
Hormone: Insulin
Details:
A disease caused by improper regulation of insulin.
Person has the proper amount of insulin in the blood stream, but the target cens in the body are
This type of diabetes represents 90% of cases and usually can be controlled by exercise and
loss of weight
loss of weight.