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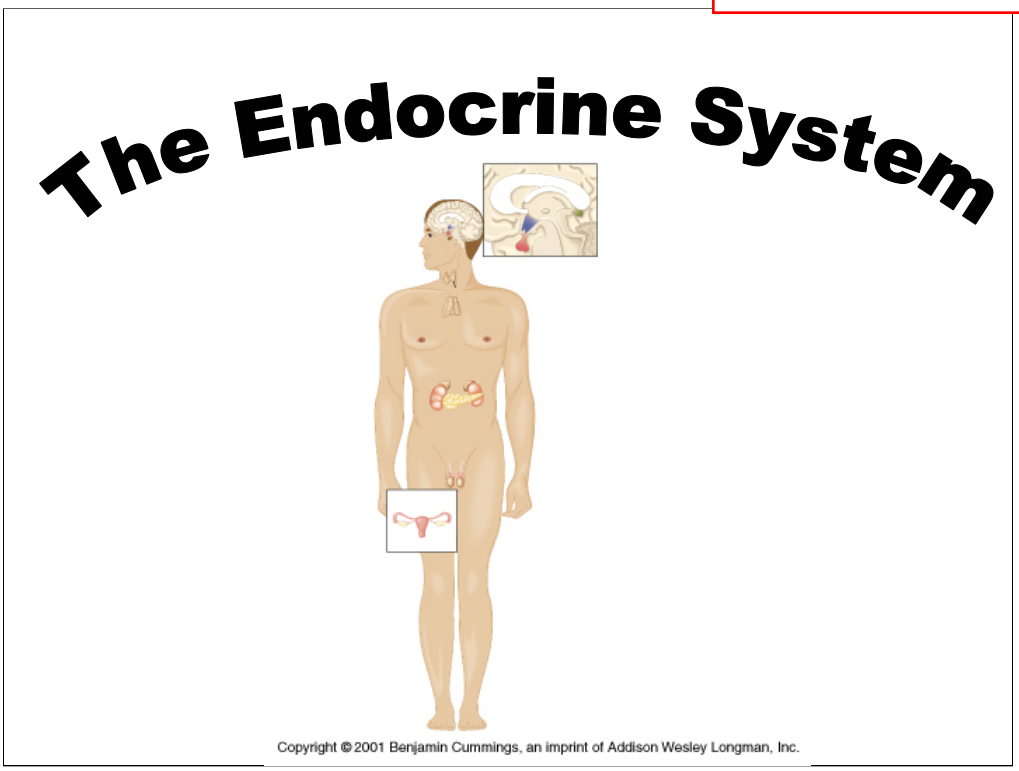

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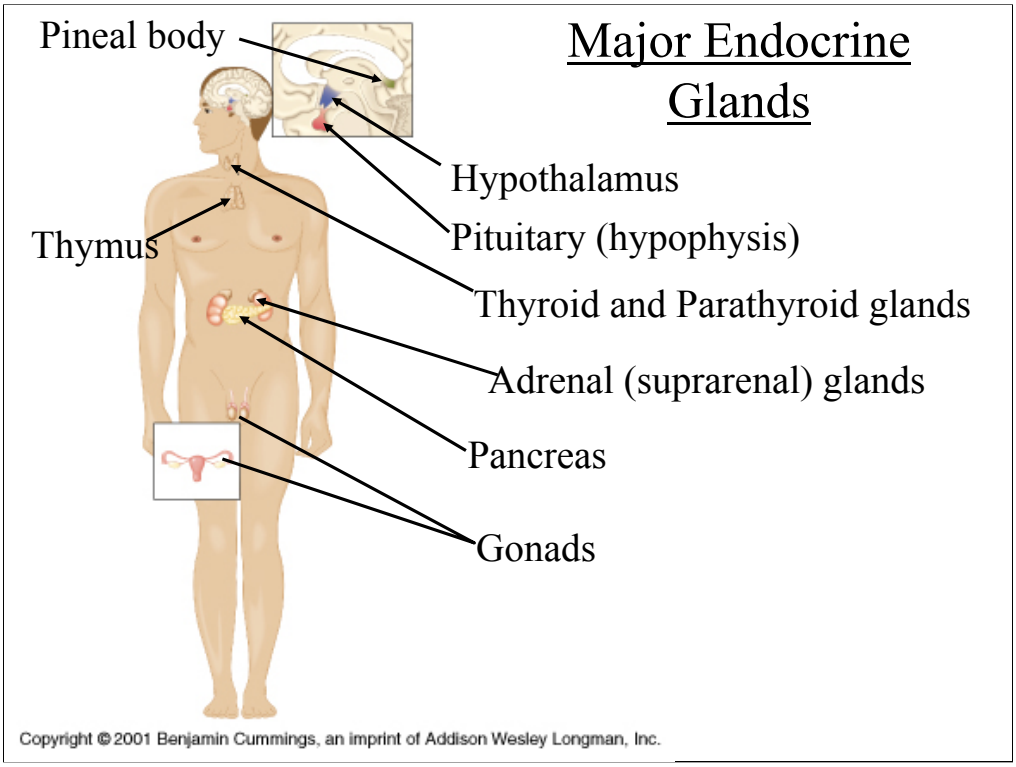
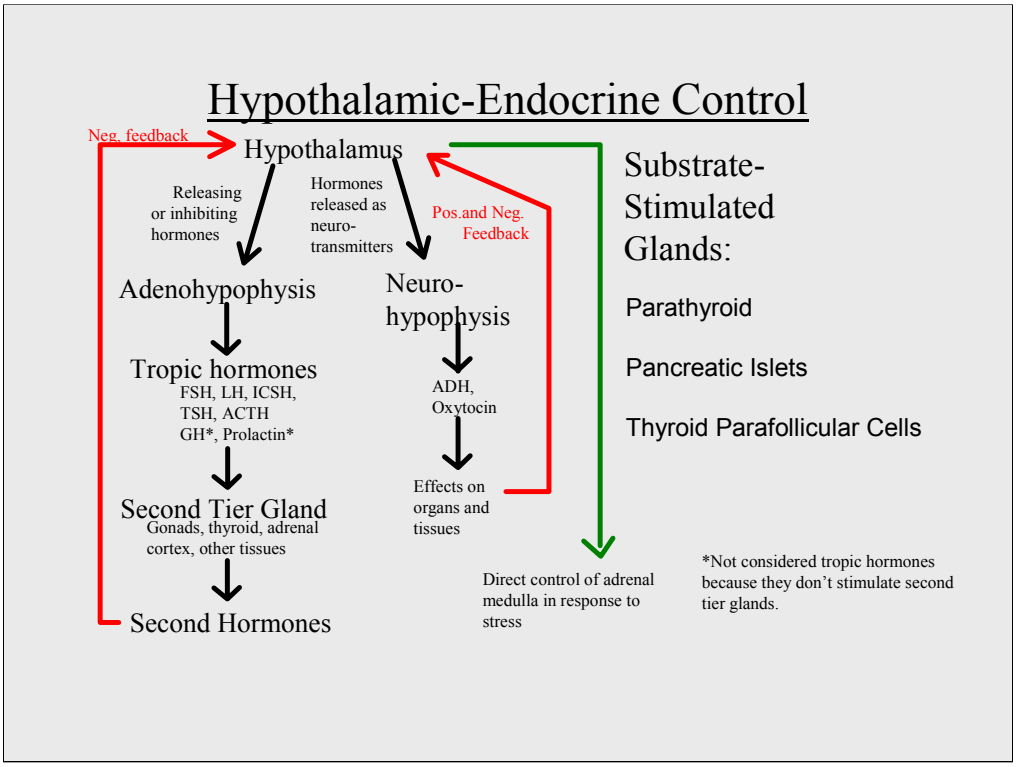
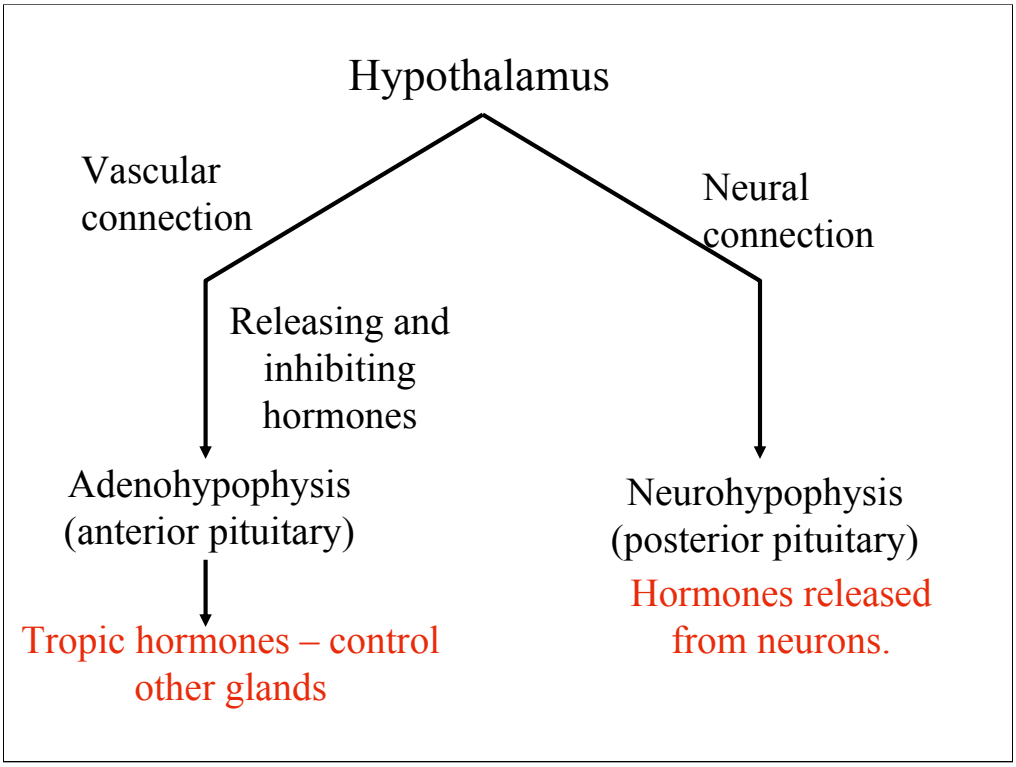


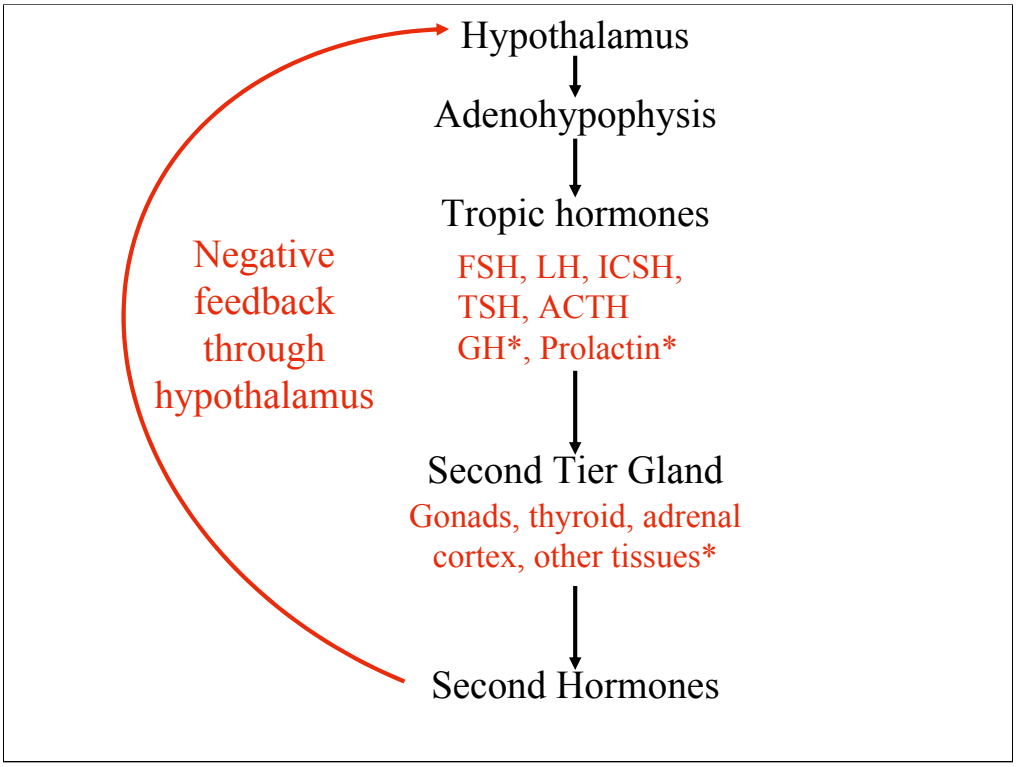
We will discuss all the glands shown above except the pineal body and thymus. These glands have been discussed already in other contexts (the brain, and the immune system). The gonads will be discussed in the reproductive laboratories.



Endocrine glands secrete chemical messengers into the bloodstream. Through the bloodstream they can have effects on a few or many target organs or other glands. Some of the endocrine glands have already been considered in other contexts or will be shortly. In addition to the endocrine glands per se, the **hypothalamus** is an important component in regulating endocrine function, and in fact is an endocrine secretor. The hypothalamus controls the **anterior pituitary gland** (**adenohypophysis**) and through it the thyroid, gonads, adrenal cortex, and the melanocytes. The hypothalamus actually produces the hormones secreted by the **posterior pituitary** (**neurohypophysis**). In addition the hypothalamus acts through the autonomic nervous system to control the adrenal medulla. Other glands such as the parathyroid and pancreas respond directly to humoral stimuli.

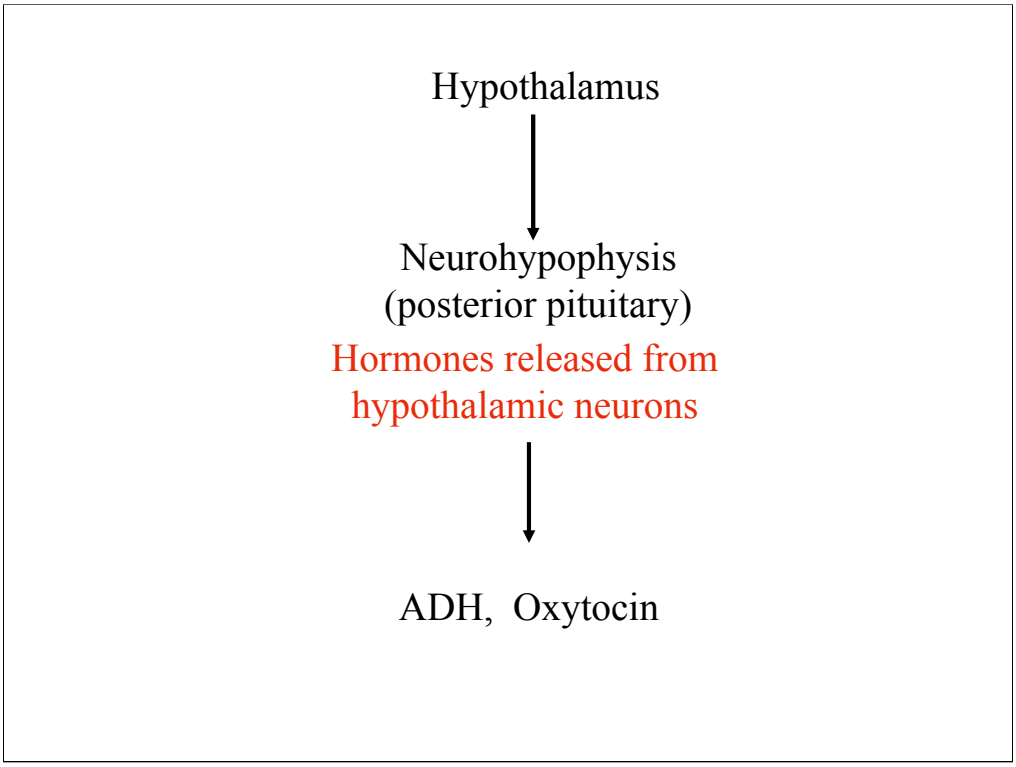


The connection of the hypothalamus and pituitary involves neurons and blood vessels which travel lo the pituitary through the infundibulum.



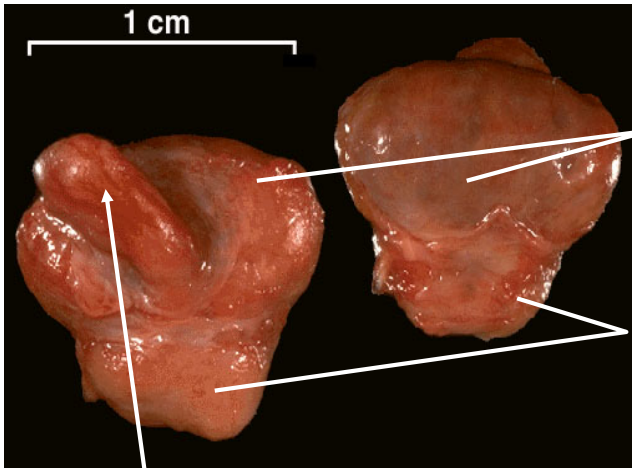
The anterior pituitary is also called the **adenohypophysis**. ***Adeno*** means **gland** and is given to this organ because it actually secretes a group of hormones known as the **tropic** hormones. These hormones control other glands or act on other tissues. The glands controlled by the tropic hormones are also endocrine glands and represent a **second tier gland** in the control mechanism. They secrete a second hormone which has actions on specific body tissues or organs **and** has a feedback effect on the hypothalamus to control its secretion. The hypothalamus controls the adenohypophysis through releasing and/or inhibiting hormones. These hormones either stimulate release of the tropic hormone or inhibit it as part of feedback control.

Growth Hormone (GH) and Prolactin (PTH) are not considered tropic hormones proper because they do not stimulate second tier endocrine glands, but rather stimulate other types of body tissues.



The posterior pituitary is called the **neurohypophysis** because the hormones it releases are actually released by neurons arising in the hypothalamus. The posterior pituitary stores these hormones for release on command, again controlled by the hypothalamus.

Pituitary Gland



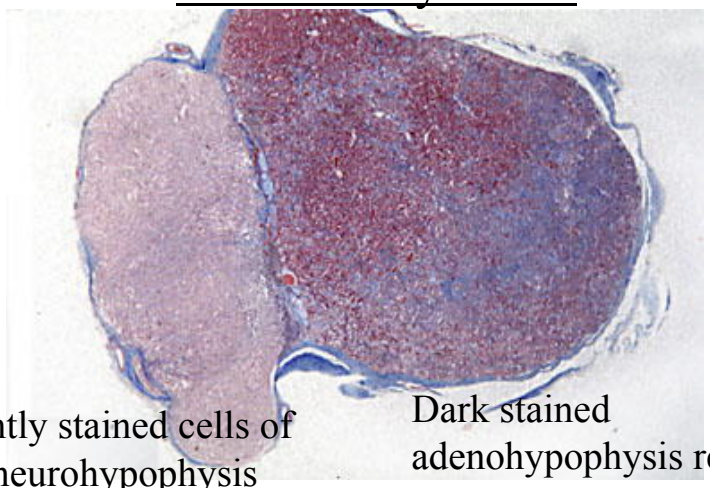
Anterior pituitary
(adenohypophysis)

Posterior pituitary
(neurohypophysis)

Infundibulum (stalk connecting to hypothalamus)

The pituitary (hypophysis) is composed of two separate glands which are attached together. Blood vessels and neurons connect these glands with the hypothalamus through the infundibulum.

The Pituitary Gland



Lightly stained cells of the neurohypophysis reflects the neural nature of this gland.

Dark stained adenohypophysis reflects the epithelial secretory cells of this gland.

Note the distinct difference between the anterior and posterior pituitary glands, reflecting their different structure and functions.

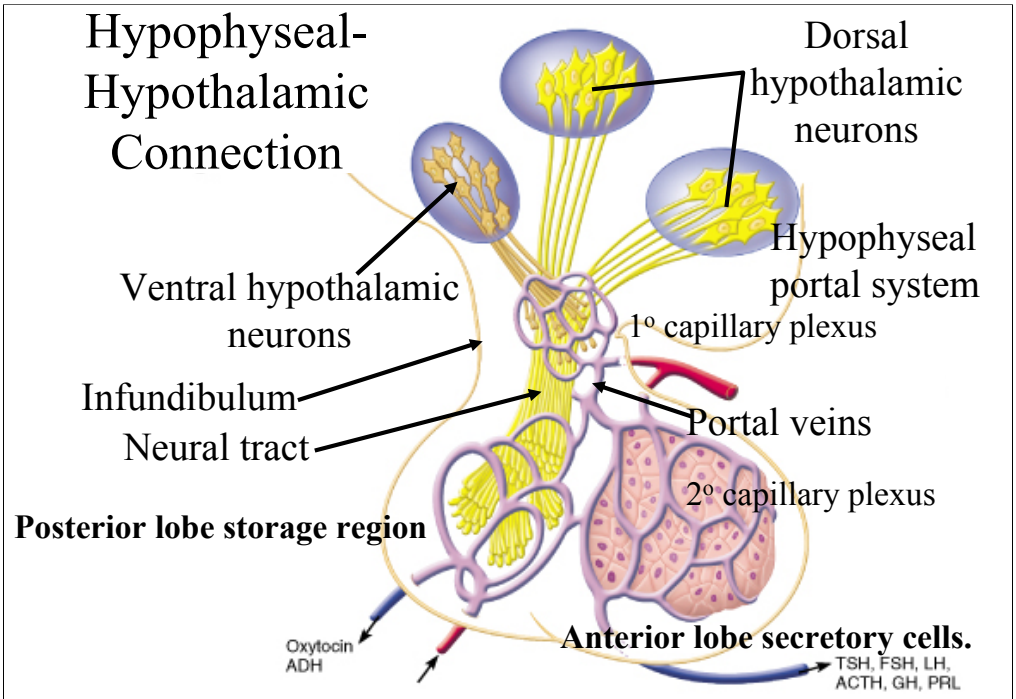


Figure 17.05

Neurons from the dorsal nucleus of the hypothalamus lead to the posterior pituitary. These neurons release **ADH** and **oxytocin** which are stored in the posterior pituitary.

Neurons from the ventral hypothalamic nucleus lead to the **primary capillary plexus** of the **hypophyseal portal system**. These neurons secrete **releasing and inhibiting hormones** which are carried by the portal veins to the **secondary capillary plexus** in the **anterior pituitary**. The releasing or inhibiting hormones then regulate the secretion of hormones by the **adenohypophysis**.

Other Control Mechanisms

Direct hypothalamic control – stimulation of the adrenal medulla to secrete epinephrine.

Substrate control – direct response by glands.

- parathyroid glands – secrete PTH (parathyroid hormone) in response to ↓ blood calcium
- thyroid parafollicular cells (C cells) – secrete calcitonin in children, respond to↑ blood calcium
- Pancreas – α and β Islet cells secrete glucagon and insulin, in response to plasma glucose.

While the hypothalamus controls most endocrine glands through the pituitary, there are several which are controlled in other ways.

Hormone Chart

GLAND	HORMONE	TARGET	ACTION
Adeno-hypophysis	Tropic hormones	Second tier glands	Activation of glands
	Follicle stimulating hormone FSH	ovaries	follicle dev. estrogen secretion
		testes	spermatogenesis

Controlled by GnRH, gonado-tropin releasing hormone, other controls are postulated.

The **gonadotropins FSH** and **LH (ICSH)** are released in response to releasing hormone **GnRH**. **FSH** (**Follicle Stimulating Hormone**) stimulates gametogenesis in both males and females. In females this involves follicle development and the first stage of oogenesis. FSH also stimulates estrogen secretion. In males FSH stimulates spermatogenesis in complex mechanism to be discussed later.

GLAND	HORMONE	TARGET	ACTION
Also controlled by GnRH, but other controls are postulated.	Leuteinizing Hormone LH,	ovaries	completion of meiosis I, ovulation, Corpus luteum, progesterone & estrogen secretion
	a.k.a. ICSH, Interstitial Cell Stimulating Hormone	testes interstitial cells	testosterone secretion

LH (**Luteinizing Hormone**) causes ovulation and progesterone secretion. In males the same hormone is called **ICSH** (**Interstitial Cell Stimulating Hormone**) stimulates interstitial cells in the testes to secrete testosterone.

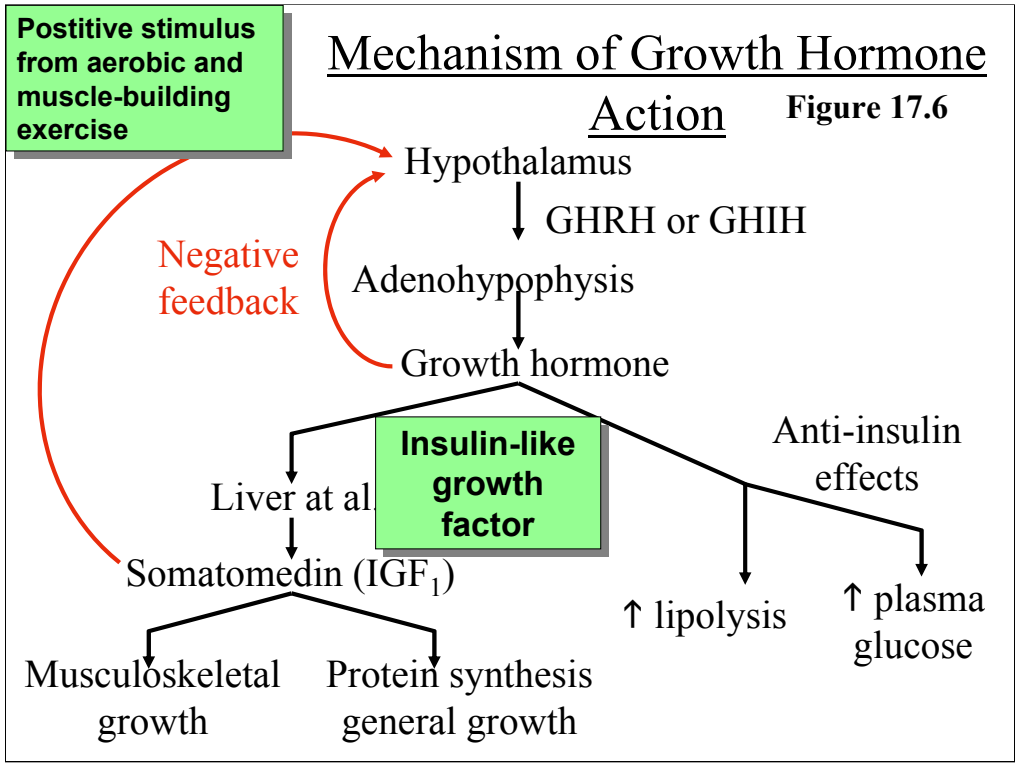
GLAND	HORMONE	TARGET	ACTION
Controlled by TRH	Thyroid stimulating hormone TSH	Thyroid gland	Gland development secretion of T4 and T3
Controlled by CRH	Adrenal cortico-tropic hormone ACTH	Adrenal cortex	Secretion of most corticosteroids (except gonado-corticoids)

Thyroid Stimulating Hormone (**TSH**) is secreted in response to TRH from the hypothalamus. TSH causes the thyroid to secrete its hormones, T4 (thyroxine) and T3.

Adrenal Corticotropic Hormone (**ACTH**) - stimulates the release of **corticosteroids** from the adrenal cortex. **ACTH** is released in response to **CRH** from the hypothalamus.

GLAND	HORMONE	TARGET	ACTION
Stimulated by GHRH **Inhibited by GHIH** **(somato-statin)**	Growth hormone GH (Somato-tropin)	Musculo-skeletal tissues	Normal growth and maintenance Anabolic for proteins Catabolic for fats
	Prolactin (luteotropin)	Mammary glands	Milk production
The above are from the adeno-hypophysis, but are not considered tropic hormones			

The two hormones above are secreted by the adenohypophysis but are not considered tropic hormones (despite their synonyms) because they don't simulate a second tier gland.



Growth Hormone (**GH**, somatotropin) - controlled by both releasing and inhibiting hormones, GHRH and GHIH (somatostatin), from the hypothalamus. GH (See Figure 17.6)causes growth and development of the musculoskeletal system and other tissues. It stimulates amino acids to be used for protein synthesis and causes lipolysis to provide fatty acids for catabolism. For these reasons it is sometimes abused to stimulate muscle growth and catabolize fat. Negative feedback results from GH itself and also from mediators called somatomedins (Somatomedin is also known as Insulin-like Growth Factor 1 [**IGF-1**]) produced by the liver, muscles, and other tissue. Positive feedback is produced by strenuous exercise and energy demanding activities.

Disorders Associated with Growth Hormone

Dwarfism – hyposecretion in children

Gigantism – hypersecretion in children

Acromegaly – hypersecretion or abuse in adults. Results in exaggerated features, especially facial bones.

Pituitary diabetes – produced by anti-insulin effects of excessive growth hormone.

Childhood hypersecretion of GH causes the excessive growth seen in **gigantism**, adulthood hypersecretion causes **acromegaly**, a condition in which the bones are exaggerated in shape. Hyposecretion in childhood causes **dwarfism**. Abuse of GH can lead to acromegaly and pituitary diabetes caused by the overstimulation of pancreatic beta cells.

GLAND	HORMONE	TARGET	ACTION
Stimulated by PRH in response to ↑ estrogen and progesterone in pregnancy, suckling after birth. **Inhibited by PIH**	Prolactin (luteotropin)	Mammary glands	Milk production

Prolactin (**PRL**) - promotes breast development and milk production. PRL is secreted in response to high estrogen and progesterone levels which occur in pregnancy, and in response to infant suckling. Control is through PRH and PIH from the hypothalamus.

GLAND	HORMONE	TARGET	ACTION
Neuro-hypophysis	oxytocin	Uterine smooth muscle	Labor
		Mammary tissue	Ejection of milk
	ADH (vasopressin)	Kidney collecting tubules	↑ water reabsorption

Stimulated by uterine pressure in a positive feedback cycle.

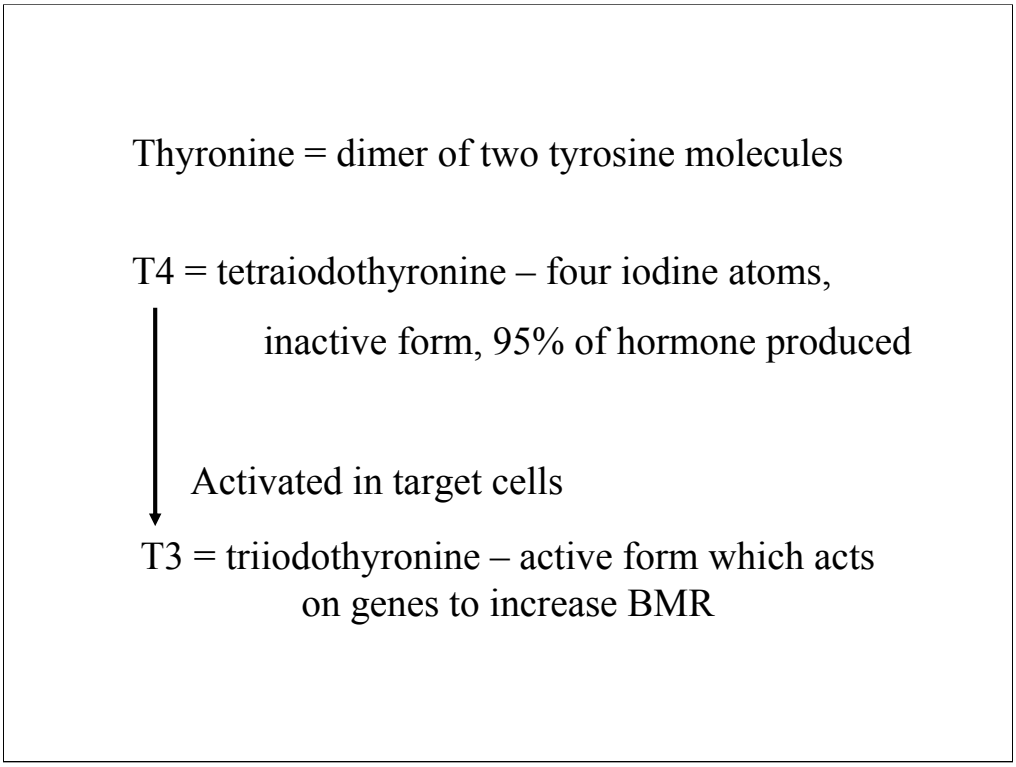
The hormones from the **neurohypophysis** are secreted by neurons from the hypothalamus. They are: **ADH - Anti Diuretic Hormone**

- as discussed earlier ADH increases reabsorption of water from the kidney's collecting tubules in response to increasing blood osmolarity. Insufficiency of ADH usually results from destruction of cells in the hypothalamus and results in **diabetes insipidus**, the production of a large volume of dilute urine. It renders the individual unable to concentrate the urine with frequent dehydration.

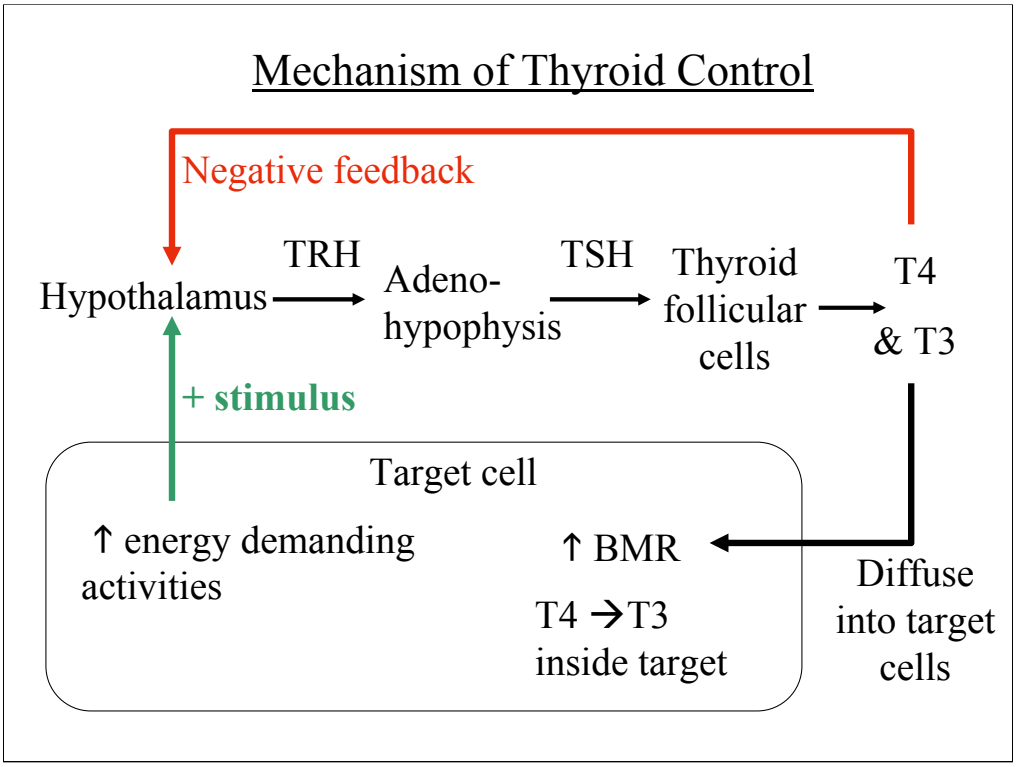
Oxytocin (OT) - Stimulates uterine smooth muscle contractions in labor, and also triggers milk ejection by the mammary glands. Released by hypothalamic neurons in response to physical and chemical stimuli at the end of pregnancy and by infant suckling. Also used clinically to induce labor.

GLAND	HORMONE	TARGET	ACTION
Thyroid follicular cells	T4 (thyroxine) and T3	Most tissue (except brain, spleen, gonads)	↑ basal metabolic rate BMR
Thyroid para-follicular cells	calcitonin	Bone tissue (children)	↑ mineral uptake and growth

The thyroid gland (Figure 17.8) consists of **follicles** whose cells secrete the two thyroid hormones, **T4** and **T3**. T4, also called **thyroxine** or **tetraiodothyronine**, is the inactive form, while T3, **triiodothyronine**, is the active hormone. T4 has four iodine atoms while T3 has three. Thyronine is the name given to a dimer of the amino acid tyrosine. T4 is produced in about 20 times as much volume as T3 and both are stored as thyroglobulin colloid in the lumen of the follicles. The follicular cells release the thryroglobulin into the follicle by exocytosis, and also resorb it and release T4 and T3 into the interstitial space to be taken into fenestrated capillaries. T4 and T3 are taken into target cells (muscle and other cells, but NOT the brain, spleen, or gonads) and into the nucleus where T3 activates genes which control cellular metabolism. Target cells convert T4 to T3. These hormones are controlled by TSH from the adenohypophysis in response to TRH from the hypothalamus.



95% of the hormone produced by the thyroid follicles is the inactive T4. But T4 is converted to the active T3 by the target cells. T3 enters the nucleus to activate genes which increase the **basal metabolic rate** (**BMR**).



The stimulus for increased T4 and T3 production is an increase in energy demanding activities. This is mediated through the hypothalamus which sends out more TRH. TRH then increases the TSH produced by the adenohypophysis, which in turn increases the production of hormones by the thyroid. By activating genes which produce enzymes important in cell metabolism they facilitate the demand for energy by the cells and also raise the BMR slightly. The increase in thyroid hormones T4 and T3 feeds back to the hypothalamus to control the process.

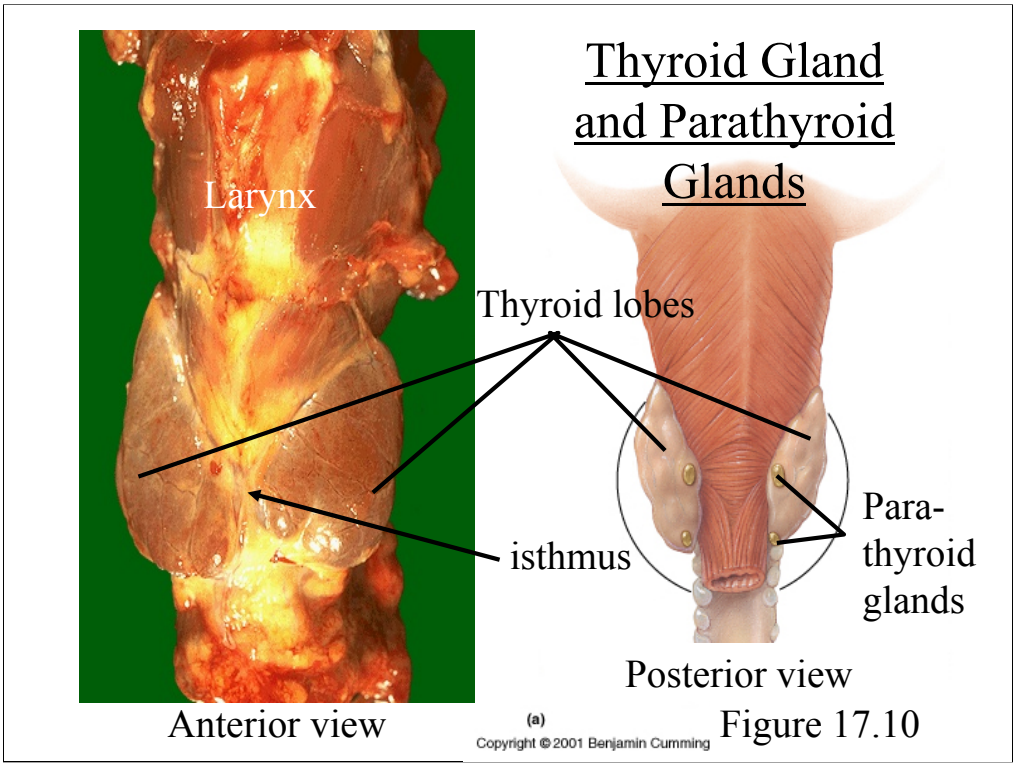
Disorders of the Thyroid

Hypothyroidism – can be due to deficiency in TSH or iodine; autoimmunity. Produces cretinism in children, myxedema in adults.

Hyperthyroidism – caused by tumors of the thyroid or pituitary and autoimmunity (Grave's Disease).

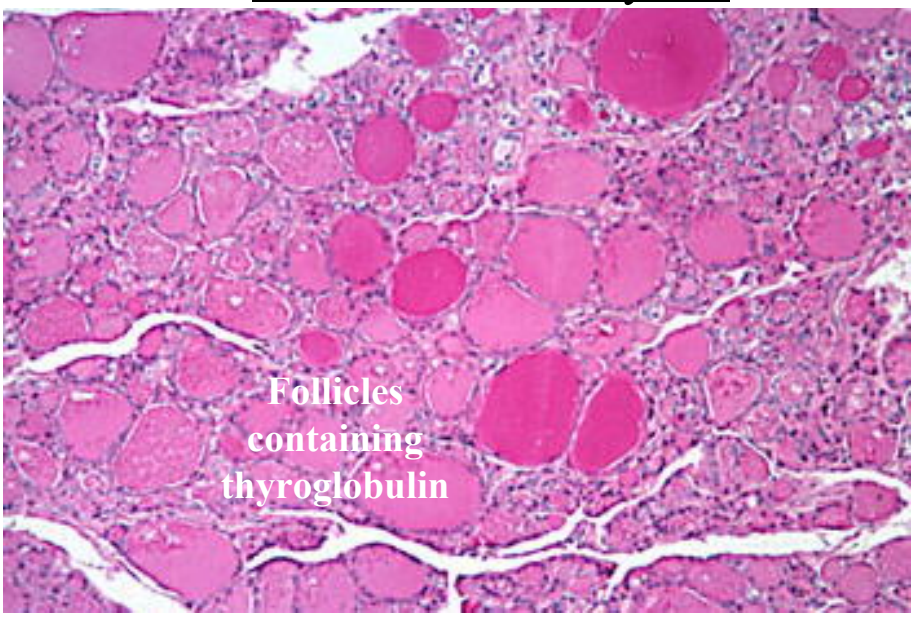
[Hypothyroidism] A detailed look at the several causes of low thyroid function, their diagnoses, and treatments.

[Hyperthyroidism] Called Graves Disease when produced by autoimmunity.

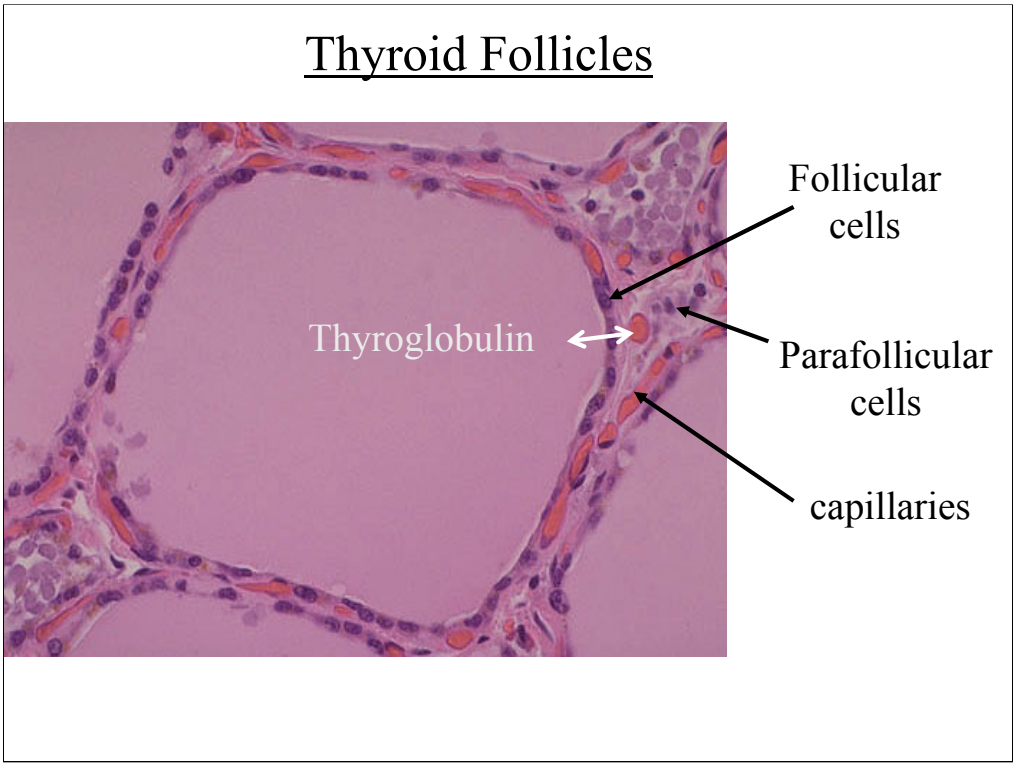


Location of the thyroid and parathyroid glands. The major lobes of the thyroid lie at the lateral lower margin of the larynx, connected by an isthmus. The parathyroid glands are tiny, bean-shaped glands embedded in the posterior portion of the thyroid. They are often difficult to find on gross examination.

Thyroglobulin is Stored in the Follicles of the Thyroid



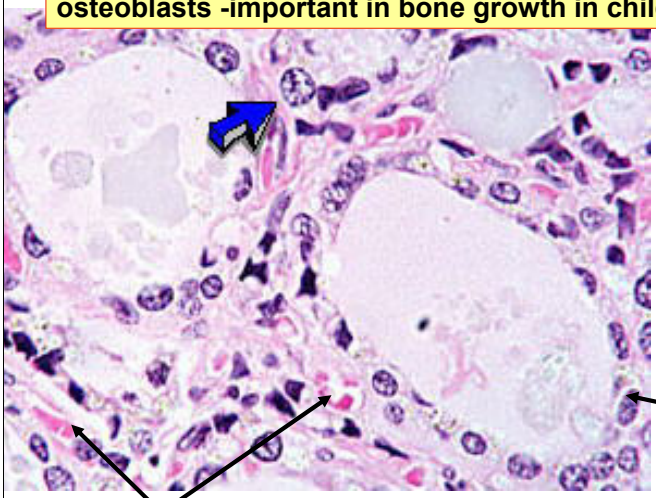
Each of the round structures seen in the thyroid gland is a follicle. The thyroid hormones T4 and T3 are stored as **thyroglobulin** in the follicles of the thyroid.



The follicular cells produce and regulate the storage of thyroglobulin, as well as its breakdown and the release of T4 and T3 into the bloodstream.

Parafollicular Cells of the Thyroid

a.k.a. C- cells: secrete calcitonin which i blood Ca^{+2} by stimulating osteoblasts -important in bone growth in children



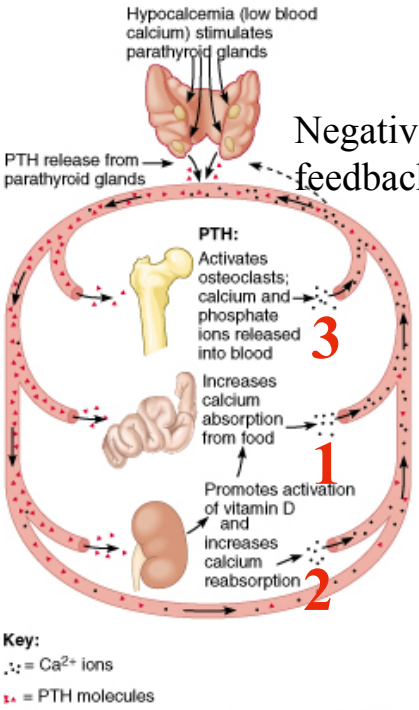
The **parafollicular cells** secrete **calcitonin** (a.k.a. thyrocalcitonin). This hormone stimulates deposition of the inorganic calcium into bone in children, and lowers blood calcium. The specific function of calcitonin is to increase osteoblastic activity. It is normally unimportant in bone maintenance in adults, however it is being used clinically to aid patients in reversing osteoporosis. The parafollicular cells are stimulated directly by rising blood calcium levels. But in adults rising calcium levels do not result in increased bone deposition.

GLAND	HORMONE	TARGET	ACTION
Parathyroid glands	Parathormone (PTH)	GI, kidneys, skeleton	↑ blood calcium

The most important hormone for Ca^{+2} homeostasis.

1) **Increases absorption of Ca^{+2} by activating more Vit D_3**
2) **Increases reabsorption of Ca^{+2} in the kidneys.**
3) **Increases osteoclastic activity in bones**

The **parathyroid gland** (Figure 17.10) consists of small bean-like glands embedded in the posterior portion of the thyroid. They secrete **parathyroid hormone** (**parathormone, PTH**) which is the hormone responsible for calcium homeostasis. (See Figure 17.11)The parathyroid glands respond to lowering blood calcium levels by secreting PTH. PTH uses several sources to raise blood calcium levels:



Mechanism of PTH Action

1) **Increases absorption of Ca^{+2} by activating more Vit D_3**
2) **Increases reabsorption of Ca^{+2} in the kidneys.**
3) **Increases osteoclastic activity in bones**

Figure 17.11

1) first, PTH triggers increased Vit D_3 (the active form) formation in the kidney. Vitamin D_3 is necessary for calcium absorption from the gut, and increased levels of the vitamin will reduce the amount of calcium which is unabsorbed. This can be significant when calcium is taken in with insufficient Vit D.

2) PTH increases Ca^{+2} reabsorption from the kidney tubules. This reduces the calcium lost to the urine.

3) The last resort for increasing blood calcium is your bones. PTH increases osteoclastic activity which causes resorption of the bone matrix.

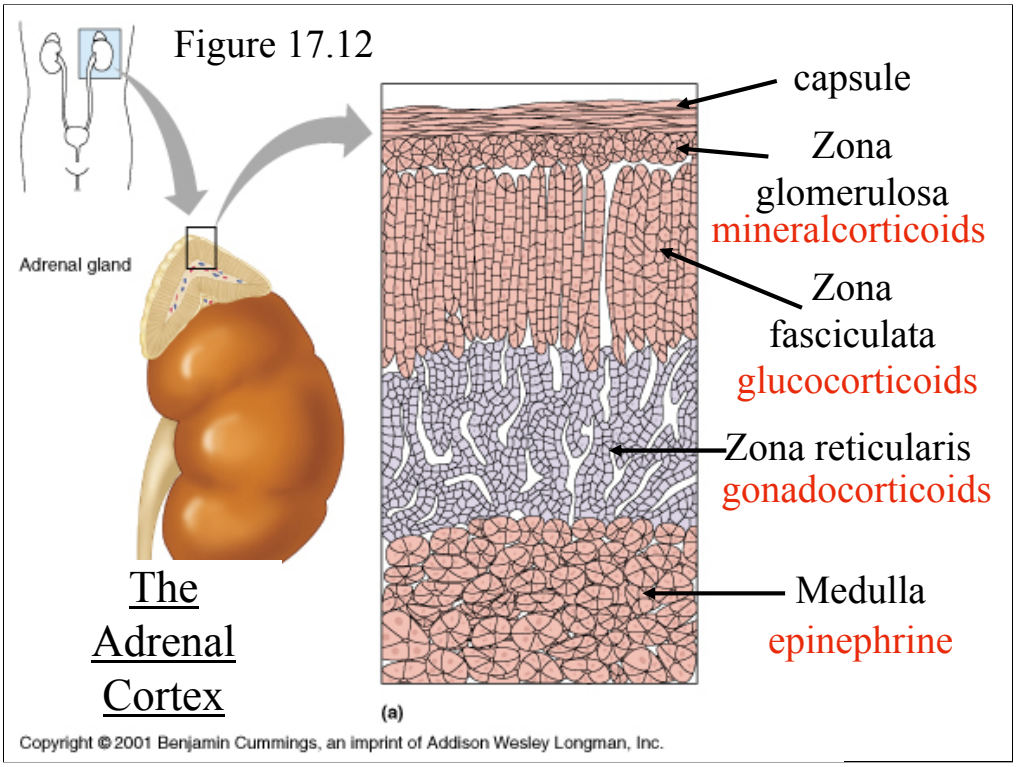
GLAND	HORMONE	TARGET	ACTION
Adrenal cortex	Corticosteroids: Mineralcorticoids e.g. aldosterone	kidneys	Electrolyte balance, ↑ Na^+ reabsorption
Controlled by ACTH	Glucocorticoids e.g. cortisol	Liver, muscle, connective,	Gluco-neogenesis
	Gonadocorticoids e.g. sex hormones testosterone, estrogen	Gonads and other tissues	Complement gonadal secretion

The **Adrenal Cortex**: (See Figure 17.12) This is the outer layer of the adrenal gland. It secretes a group of hormones known as the **corticosteroids**. This name reflects their origin and their chemical structure, based on the cholesterol molecule. They comprise three groups: 1) **mineralcorticoids** - best known is **aldosterone** which has already been studied. These are produced mostly by the outer **zona glomerulosa** layer of the cortex. The mineralcorticoids regulate electrolyte balance by increasing Na^+ reabsorption and K^+secretion.

2) **glucocorticoids** - best known is **cortisol**, also already studied. Cortisol makes glucose available from breakdown of proteins and fats during serve stress and is anti-inflammatory.

The two groups above are released in response to **ACTH** from the adenohypophysis. This is a stress response mediated by the hypothalamus (See Figures 17.13 and 17.15) which mobilizes the body's resources and raises blood pressure.

3) The third group is the **gonadocorticoids**, the sex hormones. These are **not controlled by ACTH**. The adrenal cortex complements the gonads by releasing androgens and estrogens which help regulate bone and muscle development and provide the source of estrogens for women after menopause.



Location of the **adrenal** (a.k.a. **suprarenal gland**) and its **cortex** and **medulla**, as well as the hormones each produces.

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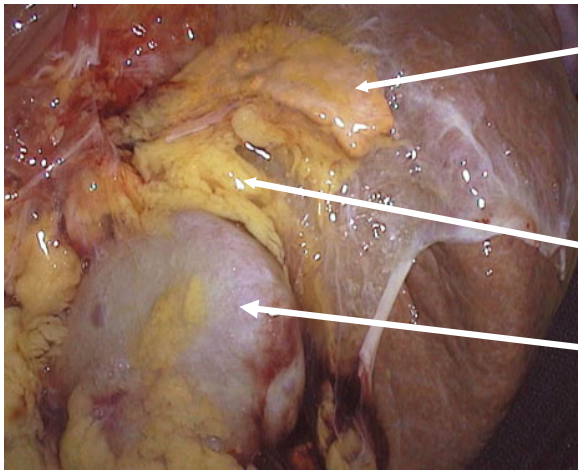
Adrenal Gland, in situ



The normal adrenal gland is a “cap shaped” structure lying atop the kidney (*adrenal* means *next to the kidney*).

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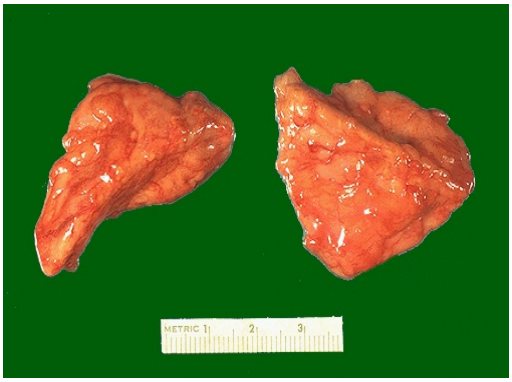
Adrenal Gland, gross specimen



Here are normal adrenal glands. Each adult adrenal gland weighs from 4 to 6 grams

Here you can see the cap shape which allows the gland to articulate with the top of the kidney.

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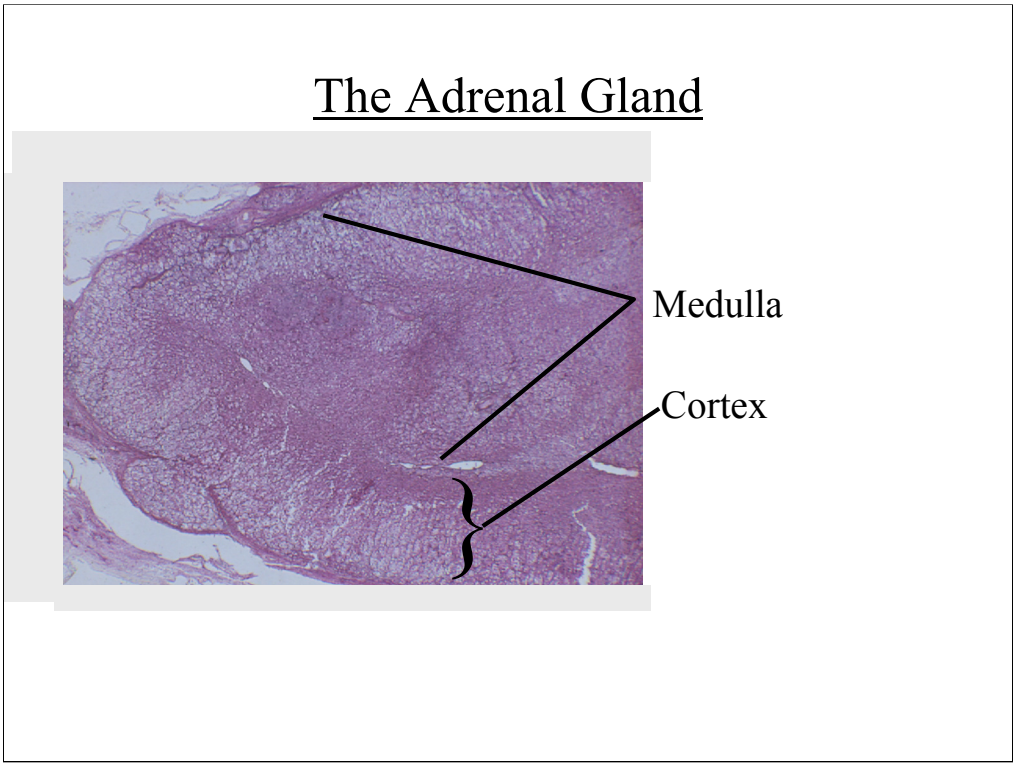

The medulla is the large inner portion of the gland (*medulla* means *middle*). The cortex is the outer layer, which is in turn composed of several sub-layers.

GLAND	HORMONE	TARGET	ACTION
Adrenal Medulla	The catecholamines e.g. epinephrine	Sympathetic receptors	Sympatho-mimetic

The **Adrenal Medulla** - the medulla (center) of the adrenal gland is sympathomimetic, i.e. it complements and enhances the effect of the sympathetic nervous system by secreting epinephrine into the bloodstream. This causes more diverse and prolonged responses than result from sympathetic stimulation by itself. The hypothalamus stimulates the adrenal medulla during "Fight or Flight", exercise, and other short term stress situations.

GLAND	HORMONE	TARGET	ACTION
Pancreas β cells	Insulin	Muscle, fat, liver, et al.	↓plasma glucose etc. uptake of glucose and amino acids into cells for metabolism and storage: glycogenesis and lipogenesis
α cells	Glucagon	Liver, fat cells	↑ plasma glucose, etc, glycogenolysis and lipolysis

Absorptive phase hormone: storage of foodstuffs

Post-absorptive phase hormone to replace glucose used in metabolism

The Pancreas (See Figure 17.16) - the pancreas has both exocrine acini (groups of secretory cells) and endocrine cells in isolated groups known as Islets of Langerhans. In these islets are two types of cells which concern us, the alpha and the beta cells. The alpha cells produce glucagon and the beta cells produce insulin. (See Figure 17.17) You learned the basic functions of those hormones as well as the disorders resulting from hyper or hyposecretion of these hormones in the last unit.

Slides 36-40 are a duplicate of those in the nutrition PDF. There are no new audio files for these slides.

Diabetes Mellitus

Type I - a.k.a. insulin dependent diabetes mellitus IDDM

usually childhood onset - < 30 yrs. old.

β cells unable to secrete insulin due to damage:

congenital, damage due to toxins or radiation,

autoimmunity, secondary to other disorders.

Managed with insulin injections, oral insulin.

Damage to tissues caused by hyperglycemia.

Life threatening acidosis when unmanaged.

Type I, a.k.a. Insulin Dependent Diabetes Mellitus, **IDDM**. (Formerly called childhood onset diabetes because typically it surfaces early in life, before the age of 30). In these individuals the **beta cells of the Islets of Langerhans** have suffered damage, usually due to autoimmunity, childhood disease, exposure to toxins, or congenital damage. As a result the pancreas produces inadequate amounts of insulin, sometimes none. Because of this the individual is unable to maintain normal plasma glucose concentration, and is unable to uptake and use glucose in metabolism. Treatment is by means of insulin administration, either by injection or orally depending on the individual. The amount of insulin must correspond to the amount of carbohydrate intake. Formerly this was difficult and would result in periods of hyperglycemia. With modern insulin pumps the process is much more exact and effective. Complications include: 1) hypoglycemia from overdose of insulin - this results in weakness, sometimes fainting, and, in the extreme, coma, all reversible with administration of glucose. 2) **ketoacidosis** from fat metabolism when insulin is under-administered. This complication can be life threatening. 3) **Hyperglycemia** when insulin administration is imprecise. Hyperglycemia damages cells and tissues

Slides 36-40 are a duplicate of those in the nutrition PDF. There are no new audio files for these slides.

Diabetes Mellitus

Type II – non insulin dependent diabetes mellitus - NIDDM

"adult onset" – usually over 30 yrs old

Due to insulin resistance of receptors on target cells.

Insulin secretion may be normal or overabundant. Many early Type II diabetics hypersecrete insulin.

May have abnormal insulin, presence of insulin antagonists, receptor defects.

Often associated with poor diet, obesity and lack of exercise.

Hyperglycemia is most damaging effect.

Type II - Non Insulin Dependent Diabetes Mellitus, NIDDM. (Formerly *adult onset diabetes* because it tends to show up after the age of 30, usually around middle age). This type is caused by **insulin-resistant receptors** on the target cells. Insulin resistance can be the result of:

•**1)** abnormal insulin - this might result from mutation to the beta cells.

•**2)** insulin antagonists - this can be the result of adaptation to hypersecretion of insulin.

•**3)** receptor defect - this can be:

a) the result of an inherited **mutation**.

b) due to **abnormal or deficient receptor proteins**. This is the result of adaptation to hypersecretion of insulin. Receptors to hormones and other chemical messengers are not stable in number or position. They move in the membrane matrix and they increase or decrease in number (called **down regulation**) in order to modulate the response, i.e. with the object of maintaining the response within a normal range. When the insulin stimulus increases tremendously, as it does in Type II diabetics, the receptors decrease in number and the receptor proteins may be deficient or abnormal.

Slides 36-40 are a duplicate of those in the nutrition PDF. There are no new audio files for these slides.

Treatment of Type II - NIDDM

Insulin analogs – stimulate receptors better than insulin

Drugs which increase insulin binding and glucose utilization in muscles.

Coordinate with diet (reduced carbohydrates) and exercise (significantly improves effectiveness).

Individuals with NIDDM often start out as **hypersecretors of insulin**. They may take in large amounts of carbohydrate and calories and this causes the secretion of insulin to be excessive and nearly constant. Over the long term this results in the receptor defects mentioned above. The **obesity** which usually accompanies Type II diabetes is both the result of the consumption of large quantities of carbohydrate and fat, and the effect of insulin in directing these excess calories to fat storage. Another contributing factor is often the lack of stimulus to cells which use glucose, i.e lack of exercise. Exercise increases the demand for glucose by muscle cells and increases the number of receptors and therefore the efficiency of glucose uptake. This is important in reducing the **hyperglycemia** which is a perennial part of NIDDM. Exercise is often used, together with a diet low in carbohydrates. Insulin analogs or mimics are also used to stimulate the receptors and make the receptors more efficient. In the later stages of the disease insulin production by the pancreas declines and insulin itself may also be used in treatments.

Keto acidosis is not usually a risk in NIDDM because these individuals do not rely solely on fats for metabolism, even when untreated. However it may occur in NIDDM when insulin secretion has been eliminated by burnout of the beta Islet cells. More likely it is a complication of kidney or liver failure in these patients.

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Effects of Hyperglycemia

Increases blood osmolarity – interferes with electrolyte and water transport.

Causes hypoxia to cells and ischemia in tissues by damaging blood vessels.

The **effects of hyperglycemia**. Hyperglycemia is the damaging result of NIDDM. It increases blood osmolarity which causes dehydration of tissues. This interferes with electrolyte and water transport and ultimately transport of nutrients and wastes. Cells and tissues break down. Among the first to show damage are the small vessels in the retina. These can be easily visualized with an ophthalmoscope, and this technique, together with urinalysis, remains one of the most important diagnostic tools for diabetes. Vessels in other tissues break down as well, and this destruction of vasculature leads to hypoxia and ischemia of body tissues including the retina, kidneys, limbs etc.

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Causes of Hypoglycemia

Hyposecretion by the α cells.

Reactive hypoglycemia – exaggerated response by the β cells.

Hypoglycemia - low blood glucose level can result from: **1) hyposecretion of glucagon**. The **alpha cells** may also be damaged and insufficient glucagon secretion will result. This leads to hypoglycemia during the early **post-absorptive** phase. But this condition is transitory because reversal will occur as the declining blood glucose stimulates the hypothalamus to cause adrenal medullary release of epinephrine. Epinephrine will bring glucose levels back up through glycogenolysis.

2) reactive hypoglycemia. This is a condition often preceding and presaging NIDDM. In individuals said to be "carbohydrate sensitive" the pancreas exhibits an exaggerated response to rising blood glucose after a carbohydrate-rich meal. This will produce hypersecretion of insulin causing the plasma glucose level to plummet. These individuals feel weak and may faint due to the hypoglycemia which results, about an hour after the meal. The usual response is to quickly eat some sugar-rich food, which does reverse the hypoglycemia. But this only compounds the problem over the long term. The solution is to reduce the carbohydrate in the meal, replacing it with protein. And to exercise before or after the meal. This releases epinephrine which helps to keep the plasma glucose level up. Many of us experience hunger about an hour after a sugar-rich meal. But individuals with reactive hypoglycemia this is in the extreme with weakness, shaking, and even blackouts occurring.

GLAND	HORMONE	TARGET	ACTION
Testes	Testosterone	Testes Other body tissues	Development and sperm production 2^{o} sex characteristics
Ovaries	Estrogen and progesterone	Uterus Other body tissues	Preparation for pregnancy 2^{o} sex characteristics

The **gonads** - secretion of the male and female sex hormones occurs in response to gonadotropin control. We will cover this in the reproductive system. The only item to mention here is that these hormones are important, in addition to their sexual functions, in producing the secondary sex characteristics of bone and muscle growth and maintenance, distribution of fat and body hair, breast development, etc.

GLAND	HORMONE	TARGET	ACTION
Pineal gland "Third Eye"	Melatonin (secreted during darkness)	Hypothalamus	Diurnal cycle regulation

SAD – seasonal affective disorder due to insufficient light stimulation.

The **pineal gland** secretes melatonin in response to the absence of light. This sets the diurnal clock mechanism for determining body rhythms and other activities coordinated with the day-night cycle. Although we can reset the clock, it is sometimes difficult and disturbs other body processes. In the far north lack of light in the winter has been known to cause psychological and physical problems. This can be alleviated with a sunlight-like artificial light stimulation. Jet lag is notorious for upsetting physical and psychological well being. Some people have found that taking melatonin at the time they want to sleep can help to reset their body clock.