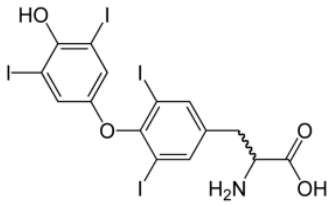
THYROID, PARATHYROID



Revised 9 Feb 2016
Marieb, p 554-559, MM 1075-, Martini 6th, pp 620-627, 7th: 606-620, 8th: 620-627, 9th: , 10th: 626-631

Thyroid: largest pure endocrine gland, lateral lobes with isthmus, high blood flow (p 627)
Two hormones: **thyroid hormone**
calcitonin

THYROID HORMONE ACTIONS: thyroxine (T_4, tissues convert to T_3, more active)
Affects every cell in body except for brain, spleen, testes, uterus and thyroid (p 628)
Stimulates enzymes involved in glucose oxidation = **increase Basal Metabolic Rate (BMR)**
increases O_2 consumption, heat production (**calorigenic** effect)
increases adrenergic receptors in blood vessels
Regulates tissue growth & development, **esp skeletal, nervous and reproductive**

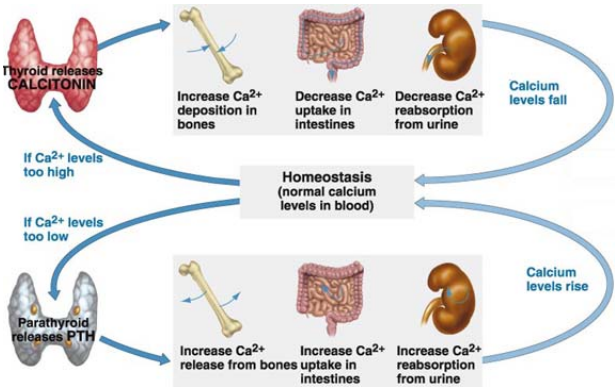
SYNTHESIS: Follicles lined by cuboidal epithelium, make **glycoprotein thyroglobulin, I-rich** (p 627)
1. thyroglobulin is synthesized on rough ER, Golgi packs, dump in to lumen, stored as **colloid**
2. Iodide concentrated fr blood, oxidized to iodine, directed into lumen, attaches to tyrosine in thyroglobulin, making DIT or MIT (diiodotyrosine etc)
3. follicle cells reclaim thyroglobulin by **endocytosis**, cleave it with lysozymes
4. Thyroxine which results diffuses into blood stream

TSH triggers secretion of thyroxine. Colloid is then restocked
90% of thyroid hormone secreted is T_4. **T_3 is 10x more active than T_4,**
Tissues convert T_4 to T_3.
cAMP is second messenger, but T_3 can enter, bind to mitochondria and nucleus (increase transcription)
Stimulus for TSH release: increased energy requirements, pregnancy, cold weather.
(steroid hormones inhibit TSH release.)

HYPOTHYROID:
myxedema: low BMR, slow speech, cold intolerance, constipation, thick dry skin, puffy eyes, edema, lethargy, mental sluggish. (**goiter** = hypertrophy, unusable colloid due to inadequate iodine)
cretinism: short disproportionate body, thick neck and tongue, mentally retarded

HYPERTHYROID:
Grave's Disease: MM: 1075- **exophthalmos**, **tachycardia**, sweating, wt. loss, nervousness, insomnia

REGULATION OF CALCIUM BLOOD LEVELS: (p 630)
Ca^{++} functions extremely important:
1) bone formation
2) coagulation of blood
3) maintain cell permeability
4) neuromuscular irritability (lack causes tetany)



Two hormones regulate blood calcium
(homeostasis of calcium on p 631):

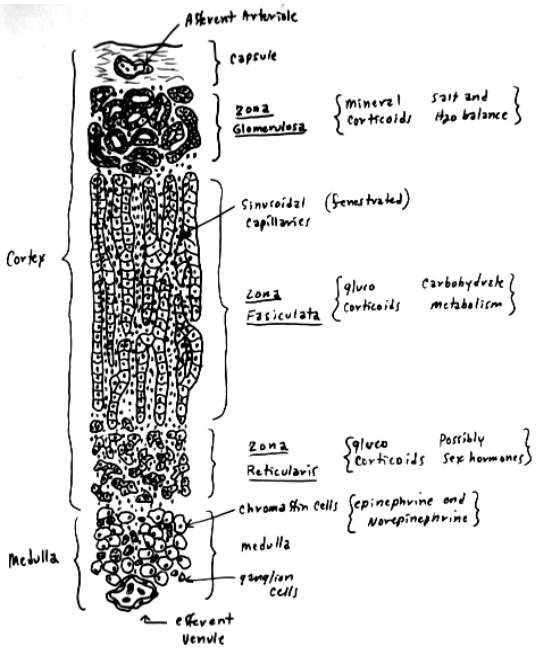
CALCITONIN:
(32 AA) synth in **parafollicular cells**, released due to high Ca^{++} in blood.
1) Inhibits bone resorption (inhibits osteoclasts)
2) Stimulates cerllular uptake of Ca^{++}, incorporation into bone.

PARATHYROID: (discovery: thyroidectomy led to tetany)
parathormone: most important blood Ca^{++} regulator.
Low blood Ca^{++} triggers its release. Inhibited by hypercalcemia. (xs parathormone = kidney stones)
Effects: 1) activates **osteoclasts**
2) enhance resorption of Ca^{++} in kidneys
3) increase absorption of Ca^{++} in intestines (activates calcitriol)

ADRENAL CORTEX

rvsd 8Feb2016
Marieb p 559-, Martinis 4th: 614-618, Martini's 6th: 627-630, 7th: 613-616, 8th: 10th: 631-634

Also known as suprarenal glands, about level of 12th rib, upon kidneys, retroperitoneal.
Medulla derived from neural crest cells, **cortex** from mesodermal cells

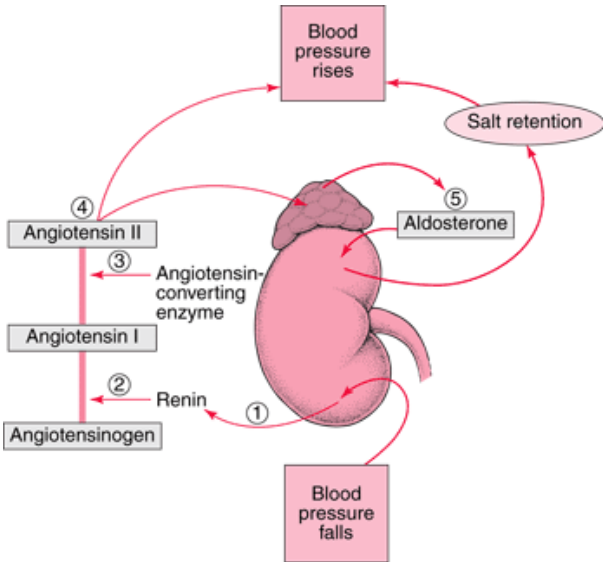


CORTEX: (p 633) Produces more than 2 dozen corticosteroids, stress response hormones made from cholesterol (or acetate if low on cholesterol) lipid rich organ = yellow
alter transcription in nucleus which affects enzyme levels, which alters metabolism
Three layers of cortex: **zona glomerulosa** **mineral corticoids**
zona fasciculata **glucocorticoids**
zona reticulata **androgens**

MINERALCORTICOIDS: regulates electrolyte balance: (p 633, 991)
Aldosterone most impt (95% of total), reg Na^+ bal, conserve Na^+ , excrete K^+
Mech: **stim Na^+ resorption in kidney distal convoluted tubules**,
therefore water is retained, ADH potentiates the effect
stimulates salt taste buds, increasing consumption of salt
release is stimulated by high K^+, low Na^+, low blood vol, hypotension

REGULATION, EFFECTS OF ALDOSTERONE: (p. 991)
Renin-angiotensin system:

renin	**juxtaglomerular complex** (in kidney) released due to hypotension, low Na^+
angiotensinogen	synthesized by liver**: renin** cleaves, becomes **angiotensin**
angiotensin I	converted to angiotensin II in lung capillaries (angiotensin converting enzyme. Note **ACE inhibitors**)
angiotensin II	1) stimulates adrenal cortex to release aldosterone 2) increases secretion of **ADH** by Posterior Pituitary 3) Increases thirst 4) stim. cardiac output, constricts arterioles: **elevates BP.**
aldosterone	activates Na^+/K^+ pump, stim. Na^+ retention in the kidney)
excess aldosterone	causes hypokalemia, cardiac arrhythmia, weak contractions



GLUCOCORTICOIDS **response to stress, raises blood glucose** by:
1. decrease cellular uptake of blood glucose
2. decrease amino acid incorporation into muscle
3. gluconeogenesis (break down protein to AA, deaminate, fabricate glucose)
4. mobilization of fatty acids from hips and thighs to abdomen and jowls (camel hump)
5. Inhibit inflammation, immune response

ANDROGENS Alias "anabolic steroids" and comprise only 7 % of corticosteroids secreted.
Some are converted to estrogens in the blood stream (normal amounts do not affect sex characteristics).
Stimulates libido in females. Excess ACTH, high BP, xs blood salt, edema, loss of sexual function

Cortisone (prednisone) side-effects:
(NOTE THAT THESE ARE A SUMMARY OF ALL THE ABOVE)
↑ blood glucose (steroid diabetes)
↓ wound healing, peptic ulcers
↑ susceptibility to infections
muscle wasting, osteoporosis
negative nitrogen balance
redistribution of fat
↓ ACTH, atrophy of adrenal cortex
hirsutism
retention of water, Na^+
hypertension, congestive heart failure
↑ intraocular pressure glaucoma
convulsions (fr hypokalemia)
suppress growth in children

Cortisone: (artificial glucocorticoid)