

Physiology Exam 9 Study Guide

Thyroid gland continued...

- Thyroid hormones (TH): T_3/T_4 – nonpolar (bind to nuclear receptors) → impact DNA
- Metabolic effects → regulate energy
 - Stimulate metabolism (Increases equation 2 → produces ATP and heat)
 - Ex: can stay comfy walking to class in frigid weather → maintains body temp
 - Generates heat
 - Enhance use of metabolic fuels (glucose, protein, fats) – Use whatever necessary to ensure engine is running properly (body)
 - Enhance results of sympathetic nervous system
 - Stimulates ATP/energy production needed for fight or flight response
 - Less likely to escape in situation if thyroid gland issues
 - Neural development
 - Necessary for neurons to have environment in which they can survive
 - Promote effects of growth hormone → to make a bigger you, need more energy
- Thyroid Disorders (Thyroid gland problem = most common endocrine disease)
- Hypothyroidism** – too little T_3/T_4 production
 - *Commonly known as iodine deficiency worldwide → Not in US b/c it's added to table salt
 - *In US it is primarily due to an autoimmune disease, but on the rise due to “au-natural”
 - No T_3/T_4 to do negative feedback from thyroid hormones (E3 has 3 jobs)
 - * T_{0-2} cannot bind to receptors in nucleus (wrong shape/charge – not specific)
 - When body has need for metabolic stimulation, releases TRH → TSH released → bind to receptors on follicular cells to cause production of T_3/T_4 (NOT there to do job!!!)
 - Body continues to send signal b/c seeing no response of metabolic stimulation
 - Leads to excess TSH secreted, low levels of T_3/T_4
 - Overstimulated follicular cells – trying to become bigger gland to increase production capacity of T_3/T_4 (TSH keeps telling them to do so)
 - Goiter** – enlarged gland (in mammals too)
 - Symptoms:
 - Cold intolerance (don't generate heat)
 - Increase weight (not running metabolism)
 - Fatigue more easily (no ATP generation)
 - Hyperthyroidism** – Too much T_3/T_4 production
 - *Commonly known as Grave's disease—autoimmune disease (soldiers we create cause problems)
 - Antibodies created activate TSH receptor on follicular cells of thyroid gland
 - *Competitors for T_3/T_4 – same response → agonists (same shape and charge)
 - T_3/T_4 doing negative feedback → decreases TSH/TRH levels → DOES NOT decrease TH production
 - Ineffective on controlling production of antibody → increased TH production
 - T_3/T_4 don't affect immune system (can't stop antibodies)
 - Enlarged gland → **goiter**
 - Symptoms:
 - Heat intolerant (already producing lot of heat)
 - Losing weight (revving that furnace)
 - Increases sympathetic activity (tons of energy, quick flight/fight response)

Adrenal Gland – named as a result of anatomy, not physiology

- Sits above kidney (renal) → but NOT associated with kidney function

-Production: CRH (E1) → APG releases ACTH (E2) → adrenal gland releases corticosteroids (**Cortisol = E3**)

-Actions: Mediates stress (historically → about food)

-Cortisol turns on system → assumes you are starving

-Focuses on short-term energy levels (NOW) → enough to survive

*How does **cortisol** help maintain energy?

-Metabolic endocrine: mainly impacts liver (helps you get energy)

-Increases glucose utilization (more easily used to increase energy)

-Breaks down fat if not enough glucose (can lose weight when stressed)

-Breakdown protein in muscles (can lose muscle mass)

*If stress continues → can lose bone

-Increases vascular smooth muscle responsive to Epi/Norepi (sympathetic)

-Able to move nutrients around

-BP increases when stressed!

*Cortisol must shut down some systems to conserve energy for survival → Negative feedback to lower need for energy

-Anti-inflammatory: higher cortisol levels inhibit immune response

*Inflammation from injury → athlete gets cortisol shot to suppress it

-Inflammation = immune response trying to fix area

-Ex: During/after finals → sick b/c high cortisol levels suppress immune system

*Must survive until tomorrow, then you can treat sickness

-Lower immune system temporarily to save energy now

-Growth suppressed – high cortisol levels stunt growth

-If I can't feed myself now, don't need to feed a larger me

-Reproduction suppressed – high cortisol levels inhibit sexual drive

-Take a girl to a chick-flick!

-Disorders

-No cortisol → dead within days

-Needed to maintain specific levels to survive

-Adrenal insufficiency – too little cortisol

-Symptoms: Weak (not enough energy), decreased glucose levels, decreased BP

-AKA Addison's syndrome

-Cushing's syndrome – Too much cortisol (not from stress)

-Symptoms: decrease bone strength, increased glucose, high BP, decreased immune response, obesity (storing energy near vital areas (belly fat) → not used in metabolism)

*Can be diagnosed early wrong as diabetes

Somatic Growth → current growth (here and now), not future reproduction

-Somatic growth requires protein synthesis for all processes to work (cell division)

-Height – due to growth of bones (specific location)

-Adding new cells at epiphyseal plates (growth plates – where growth occurs)

-Steroids (sex endocrines) fuse plates during puberty → stop getting taller and fill out

*Careful when kids break bones – want in middle away from growth plates

-Adults don't have these issues – plates sealed

-Growth of other tissues based on cell proliferation via mitosis (same DNA – identical cells)

-Regulated by endocrines:

- Growth Hormone (GH)**: released by GHRH, inhibited by Somatostatin
- *Unique: E2 is main talking point as opposed to E3s for thyroid, adrenal
 - See highest level during development (growing the most)
 - Actions: Primarily to stimulate IGF-1 (E3) released from liver and bones
 - Causes cell proliferation – sends message to liver to release IGF into blood, and to bone to stimulate plates directly
 - Other option: GH impacts cell directly by stimulating muscle development
 - To make a bigger you, need bigger muscles b/c bigger loads
 - *Different message sent – not making muscle cells but adding to inside of muscle cells
 - Mobilizes nutrients to have energy for process (growth spurts)
 - Periods of getting taller, then fill out (repeat)
- TH**: needed for GH synthesis and effect
 - Need right levels of T_3/T_4 to stimulate proper amount GH (amount of metabolism to provide energy for growth to occur)
- Cortisol**: Can inhibit growth by inhibiting GH secretion in APG (similar to SS)
 - If can't sustain self now, don't want to grow bigger
 - Opposes the effects of GH in tissues
 - *Athletes don't realize that as cortisol levels increase (stress from poor stats) → decreases impacts of GH
- Insulin**: Promotes growth
 - Helps pack away nutrients you ingest to energize growth
- Sex Steroids**: stimulate secretion of GH and IGF-1
 - Historically: bigger male = reproductive advantage in finding a mate
heavier female = more success in carrying a baby
 - Closure of epiphyseal plates → grow wide not tall
 - Testosterone- anabolic → redundant (all sex endocrines build tissue)

**Unique: GH has many roles – impacts muscle development too

- Needs another way to shut down pathway besides negative feedback
- Somatostatin (E2) – acts directly to shut down pathway

Regulation of Calcium – need specific range → important for neurons, muscles, etc.

-3 systems involved in Calcium homeostasis

1. Bone – contains 99% of total
 - Constant remodeling → bones are dynamic (NOT fixed)
 - *Energy taken away from area not used (trade-offs)
 - If not moving, lose calcium – important to get old people moving (they have less energy/strength involved in bones)
2. Kidney – filters plasma to determine amount of calcium in urine
 - Regulates calcium excretion (excrete more if high [calcium])
3. Gastrointestinal tract – site of calcium absorption from diet (regulates it)

*How do we impact all of these factors?

2 Major Endocrines

1. **Parathyroid hormone (PTH)** – Produced by parathyroid glands

- Glands have a receptor for calcium (chemosensory component) – checks levels
 - decreased $[Ca^{2+}]$ in plasma → increased PTH (negative feedback)
- PTH acts to increase $[Ca^{2+}]$ in plasma by impacting 3 areas: ^^