

Lecture 22

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Hormone Classifications

- Amine hormones - amino acids with modified groups (e.g. norepinephrine's carboxyl group is replaced with a benzene ring)
- Peptide hormone - short chains of linked amino acids
 - o Epinephrine
 - o Made in cells from amino acids (just like all peptides/proteins)
 - o Water soluble (so they can't diffuse across sarcolemma)
 - o Usually act through second messenger on cell surface
 - o Fast initiation, temporary action
- Protein hormone - long chains of linked amino acids
- Steroid hormones - derived from the lipid cholesterol; fat soluble
 - o Testosterone
 - o Made from cholesterol
 - o Fat soluble (so they can diffuse across sarcolemma)
 - o Adrenal cortex, testes, ovaries
 - o Nuclear or cytosolic receptors
 - o Slow initiation, long action
 - A. Steroid hormone enters a cell
 - B. Hormone binds to a specific receptor in the cytoplasm or in the nucleus
 - C. Hormone-receptor complex activates the cell's DNA, which forms mRNA
 - D. mRNA leaves the nucleus and enters cytoplasm
 - E. mRNA directs protein synthesis in the cytoplasm
- Most hormones come from glands, get shuttled around in circulation, and exert some physiological effect on a distant bunch of cells (a tissue or an organ)
- The signal from a hormone (and the consequence of that signal) only affects cells that express a specific receptor, one that is specific to that *exact* hormone
 - o Otherwise, they would affect every (or at least any) cell in the body

- Autocrine secretion: cell releases hormone by itself for itself; hormone never exits the tissue that produced it
- Paracrine secretion: hormone gets released, acts with adjacent cells, doesn't need to enter circulation to get there
- Binding proteins: carry hormones through circulation, prolonging the (otherwise brief) half-life of the hormone; major role in endocrine function
- Signaling cascades
- PKA's initiation of lipolysis
 - o Has multiple functions:
 - o Phosphorylates (activating) HSL; this initiates its translocation to the lipid droplet
 - o Phosphorylates perilipin, causing a conformational change; this is the more important event in the stimulation of lipolysis
 - Lipolysis is regulated by growth hormone, catecholamines, *insulin*, glucagon, cortisol, TNF- α
- cAMP system
 - o There are receptors which, when activated, stimulate adenylate cyclase: R_s receptors
 - o "inhibit": R_i receptors
 - o Stimulation: B1 and B2 adrenergic receptors, glucagon receptors, ACTH receptors
 - o Inhibition: α 2 adrenergic receptors, opioid receptors, cannabinoid receptors, adenosine receptors
 - o Once you have adenylate cyclase activated, and cAMP made, what stops it then?
 - PDE (phosphodiesterase) disrupts and destructs that signaling
- How does insulin resistance cause fat gain?
 - o Steps:
 - 1) Insulin binds to its receptor
 - 2) PI3K gets activated
 - 3) Downstream of PI3K
 - 4) PKB gets activated
 - 5) PI3K and PKB activate PDE
 - 6) PDE breaks down cAMP
 - 7) Thus PKA does not get activated, so it doesn't phosphorylate anything
 - 8) So lipolysis can't happen

- o) Lipolysis can happen
 - GLUT4 translocation signaling pathway can become resistant to the effects of insulin (poor insulin sensitivity)
- Lipolysis is regulated by: growth hormone, catecholamines, insulin, glucagon, cortisol, TNF- α