The adrenal glands are bean-shaped glands that sit on top of each kidney like a hat.
Adrenal Glands

- Actually two glands:
  - Adrenal medulla
  - Adrenal cortex.

While each adrenal is one structure, it is really two separate glands that secrete different hormones and have different effects on the body. The adrenal medulla is the “core” or center of the adrenal gland. It is composed of neural tissue – sympathetic nervous system neurons secrete their hormones (called catecholamines) directly into the blood stream. The outer part of the adrenal gland is the adrenal cortex. The adrenal cortex consists of three layers, each of which secretes a different type of hormone. Adrenal cortex hormones are steroids while the adrenal medulla secretes amino acid-based catecholamines.
The outermost layer of the adrenal cortex, called the glomerulosa, secretes a class of hormones called **mineralcorticoids**, so-called because their main function is to regulate mineral concentrations in the blood. The most important of the mineralcorticoids is **aldosterone**.

The main function of aldosterone is to work with **anti-diuretic hormone** to retain water. It does this, though, by retaining **sodium** (it prevents the excretion of sodium by the kidneys). When aldosterone is released, the kidneys sequester sodium and excrete potassium, which makes the blood hypertonic and, through simple osmosis, water is retained in the blood rather than excreted by the kidneys. The net effect of aldosterone, then, is an increase in water retention and reabsorption, an increase in blood sodium, and a decrease in blood potassium.

Control of aldosterone release is somewhat complicated because so many factors can influence it. An increase in potassium concentration or a decrease in sodium concentration can each stimulate aldosterone secretion through humoral effects. In addition, though, blood pressure changes can influence aldosterone. A drop in blood pressure or blood volume stimulates the kidneys to release a chemical called **renin** which, through a chemical cascade, results in aldosterone secretion as well. In addition, high blood pressure can cause the heart to secrete a hormone called **ANP** which inhibits aldosterone release, resulting in a lower blood volume. There are also neural effects: stress can result in secretion of **ACTH** from the anterior pituitary, which increases the production of all andrenocortical steroids (including aldosterone).
Hormones of the Adrenal Cortex

Complexity of aldosterone control.
Hormones of the Adrenal Cortex

- **Middle layer: glucocorticoids**
  - **Cortisol, cortisone**
    - ↑ glucose (long-term stress), reduce inflammation
    - Target tissues: Many, esp. liver

The middle layer of the adrenal cortex secretes a class of hormones called **glucocorticoids**, which generally increase blood glucose levels by stimulating the breakdown of glycogen, fats, and proteins. The main glucocorticoid is the stress hormone **cortisol**.

Cortisol is released from the adrenal cortex when the body is undergoing long-term (resistive) stress. Cortisol stimulates metabolism to make more fuel available for muscles and for tissue repair. Proteins and fats are broken down in particular. In addition, cortisol suppresses the inflammatory response and reduces the experience of chronic pain and swelling. It does this by inhibiting another type of hormone called prostaglandins. Some anti-inflammatory medications are derived from cortisol, such as prednisone and cortisone.

Cortisol is controlled through a hormonally-mediated negative feedback loop, but there's a twist. When the body is experiencing long-term stress, the hypothalamus secretes the releasing hormone CRH (corticotropin releasing hormone) into the hypothalamic-pituitary portal, which causes the pituitary to secrete the tropic hormone ACTH. Circulating ACTH stimulates cortisol production, which feeds back on the hypothalamus, inhibiting it from making additional CRH. Once the stressors are gone, the cycle stops, but if the stressors are still present, the hypothalamus will resume producing CRH once cortisol levels begin to fall.
The innermost layer of the adrenal cortex produces low levels of weak sex steroids, most **andro gens**. Androgens are, literally, “male hormones” such as precursors to testosterone. Most androgens are secreted by the gonads, but the adrenal cortex does secrete some. These low levels of hormones occur in both males and females and may be involved in the timing of puberty.

Interestingly, an oversecretion of adrenal androgens after puberty (such as from a tumor) has little or no effect in males because there are such high levels of circulating androgens (from the testes) anyway. In females, though, oversecretion can cause the development of male secondary sex characteristics, such as facial hair.

Control of the adrenal sex steroids is not very well-understood. Like all adrenal steroids, ACTH stimulates secretion, but high levels of circulating androgens does not feed back on the hypothalamus.
Hormones secreted by the adrenal medulla are called **catecholamines**. The best-described of these hormones is adrenalin, which is a stress hormone secreted along with its partner, noradrenaline. Adrenalin secretion is stimulated neurally (remember that the adrenal medulla is nervous tissue) when the brain interprets a situation as dangerous or requiring a “fight or flight” response. Glycogen is broken down into glucose more quickly, heart rate increases, respiration increases, blood vessels constrict, etc. The major benefit of these changes in the target tissues is that more nutrients and oxygen become available to the muscles and brain, and these substances are circulated through the body more quickly.
This figure summarizes the major adrenal hormones.
The gonads (testes in males and ovaries in females) have wide-ranging and complex effects that will be discussed in detail when we cover the reproductive system, but here’s an overview. Males have high circulating levels of androgens which females have high circulating levels of estrogens. Both sexes have both types of hormones (in fact, testosterone is a modified form of an estrogen), but the relative levels are very different.

You’re probably aware of the developmental and reproductive role of the sex steroids: they determine a person’s gender during development, they are responsible for the developmental changes during puberty, and they’re responsible for reproductive behavior and gamete (sperm and egg) formation. In females, they also regulate the menstrual cycle, pregnancy, and childbirth.

The sex steroids have additional, non-reproductive, effects as well. Estrogen plays an important role in maintaining bone density. It acts antagonistically to parathyroid hormone and prevents PTH from stimulating excess bone resorption. This is why postmenopausal women are at risk for osteoporosis. Even men who have low or absent estrogen levels develop osteoporosis. Estrogen has also been implicated in regulating muscle and bone development, blood clotting, mood, and some types of learning.

Control of the sex-steroids is complex, but at its core, a simple negative feedback loop is involved. The hypothalamus secretes GnRH (gonadotropin releasing hormone), which stimulates the anterior pituitary to secrete lutenizing hormone. Lutenizing hormone stimulates the gonads to secrete sex steroids, which inhibit further GnRH production by the hypothalamus.