

## 5.2.2

# Aldosterone (Mineralocorticoid)

Watch the video [Aldosterone - Mechanism of Action](#)

**Mineralocorticoids** are steroids synthesized in the zona glomerulosa. They bind with specific intracellular receptors in the cells of the kidney. Aldosterone is the most well-known mineralocorticoid. **Aldosterone** helps to increase sodium reabsorption in the kidneys. Aldosterone release is stimulated by increased levels of angiotensin II or extracellular  $K^+$ . If extracellular  $Na^+$  is increased, then aldosterone release will decrease. Aldosterone increases the expression of  $Na^+/K^+$  pumps on the basolateral membrane and ENaC (passive sodium channels) on apical membranes of renal tubular cells of the distal convoluted tubule and collecting duct. Increasing these transporters allows for more sodium to be reabsorbed from the filtrate and enter back into the blood. This sodium attracts water and acts to increase intravascular fluid volume and therefore increase blood pressure. Aldosterone will also increase excretion of potassium and hydrogen ions, which means that excess aldosterone can cause hypokalemia and possible alkalosis.

## Primary Aldosteronism (Conn's Syndrome)

People with primary aldosteronism, otherwise known as **Conn's Syndrome**, have a benign tumor in the zona glomerulosa that secretes excess amounts of aldosterone. As suggested above, this hypersecretion of aldosterone leads to hypernatremia, high blood pressure, hypokalemia, and decreased serum renin levels. Hypokalemia leads to muscle weakness because the resting membrane potential of neurons becomes hyperpolarized, leading to a decreased ability to experience an action potential.

A useful treatment for primary aldosteronism is **spironolactone**, a drug that blocks mineralocorticoid receptors and thus acts as an aldosterone antagonist. The most common treatment is surgical removal of the tumor.

## Secondary Aldosteronism

Secondary aldosteronism is characterized by high levels of aldosterone caused by excessive adrenal stimulation (specifically of the zona glomerulosa). For example: increased renin production leads to increased angiotensin II which then stimulates aldosterone release. Renin can be increased because of a decrease in blood volume or pressure, decreased blood supply to the kidney because of renal artery stenosis, low cardiac output, or possibly even a renin secreting tumor in the kidney.



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