25.3B: Sodium Balance Regulation

Sodium is an important cation that is distributed primarily outside the cell.

Learning Objectives

- Describe the mechanisms by which sodium balance is regulated

Key Points

- The body has a potent sodium-retaining mechanism: the rennin–angiotensin system.
- In states of sodium depletion, aldosterone levels increase; in states of sodium excess, aldosterone levels decrease.
- The major physiological controller of aldosterone secretion is the plasma angiotensin II level that increases aldosterone secretion.

Key Terms

- **sodium**: A chemical element with symbol Na (from Latin: natrium) and atomic number 11. It is a soft, silvery white, highly reactive metal and is a member of the alkali metals.
- **aldosterone**: A mineralocorticoid hormone that is secreted by the adrenal cortex and regulates the balance of sodium and potassium in the body.
- **angiotensin**: Any of several polypeptides that narrow the blood vessels and regulate arterial pressure.
Sodium Regulation

Sodium is an important cation that is distributed primarily outside the cell. The cell sodium concentration is about 15 mmol/l, but it varies in different organs; it has an intracellular volume of 30 liters and about 400 mmol are inside the cell.

The plasma and interstitial sodium is about 140 mmol/l with an extracellular volume of about 13 liters, 1,800 mmol are in the extracellular space. The total body sodium, however, is about 3,700 mmol as there is about 1,500 mmol stored in bones.

The body has potent sodium-retaining mechanisms and even if a person is on five mmol Na+/day they can maintain sodium balance. Extra sodium is lost from the body by reducing the activity of the renin–angiotensin system that leads to increased sodium loss from the body. Sodium is lost through the kidneys, sweat, and feces.

In states of sodium depletion, the aldosterone levels increase. In states of sodium excess, aldosterone levels decrease. The major physiological controller of aldosterone secretion is the plasma angiotensin II level that increases aldosterone secretion.

A high plasma potassium level also increases aldosterone secretion because, besides retaining Na+, high plasma aldosterone causes K+ loss by the kidney. Plasma Na+ levels have little effect on aldosterone secretion.

Renin–angiotensin–aldosterone system

**Renin–angiotensin system**: The regulation of sodium via the hormones renin, angiotensin, and aldosterone. In states of sodium depletion, the aldosterone levels increase, and in states of sodium excess, the aldosterone levels decrease.

A low renal perfusion pressure stimulates the release of renin, which forms angiotensin I that is converted to angiotensin II. Angiotensin II will correct the low perfusion pressure by causing the blood vessels to constrict, and increase sodium retention by its direct effect on the proximal renal tubule and by an effect operated through aldosterone. The perfusion pressure to the adrenal gland has little direct effect on aldosterone secretion and the low blood pressure operates to control aldosterone via the renin–angiotensin system.

Aldosterone also acts on the sweat ducts and colonic epithelium to conserve sodium. When aldosterone is activated to retain sodium the plasma sodium tends to rise. This immediately causes the release of ADH, which causes water to be retained, thus balancing Na+ and H2O in the right proportion to restore plasma volume.

In addition to aldosterone and angiotensin II, other factors influence sodium excretion.
• Atrial peptide causes the loss of sodium by the kidneys: it is secreted from the heart in high sodium states due to excess intake or cardiac disease.

• Elevated blood pressure will also cause Na+ loss, and a low blood pressure usually leads to sodium retention.