

Lecture 5

HORMONAL CONTROL OF OSMOREGULATION

- **As the primary link between environmental change and physiological response, the neuroendocrine system is a critical part of osmoregulatory adaptations.**
- **While the kidneys operate to maintain osmotic balance and blood pressure in the body, they also act in concert with hormones.**
- **Hormones are small molecules that act as messengers within the body. Hormones are typically secreted from one cell and travel in the bloodstream to affect a target cell in another portion of the body.**
- **Different regions of the nephron bear specialized cells that have receptors to respond to chemical messengers and hormones.**

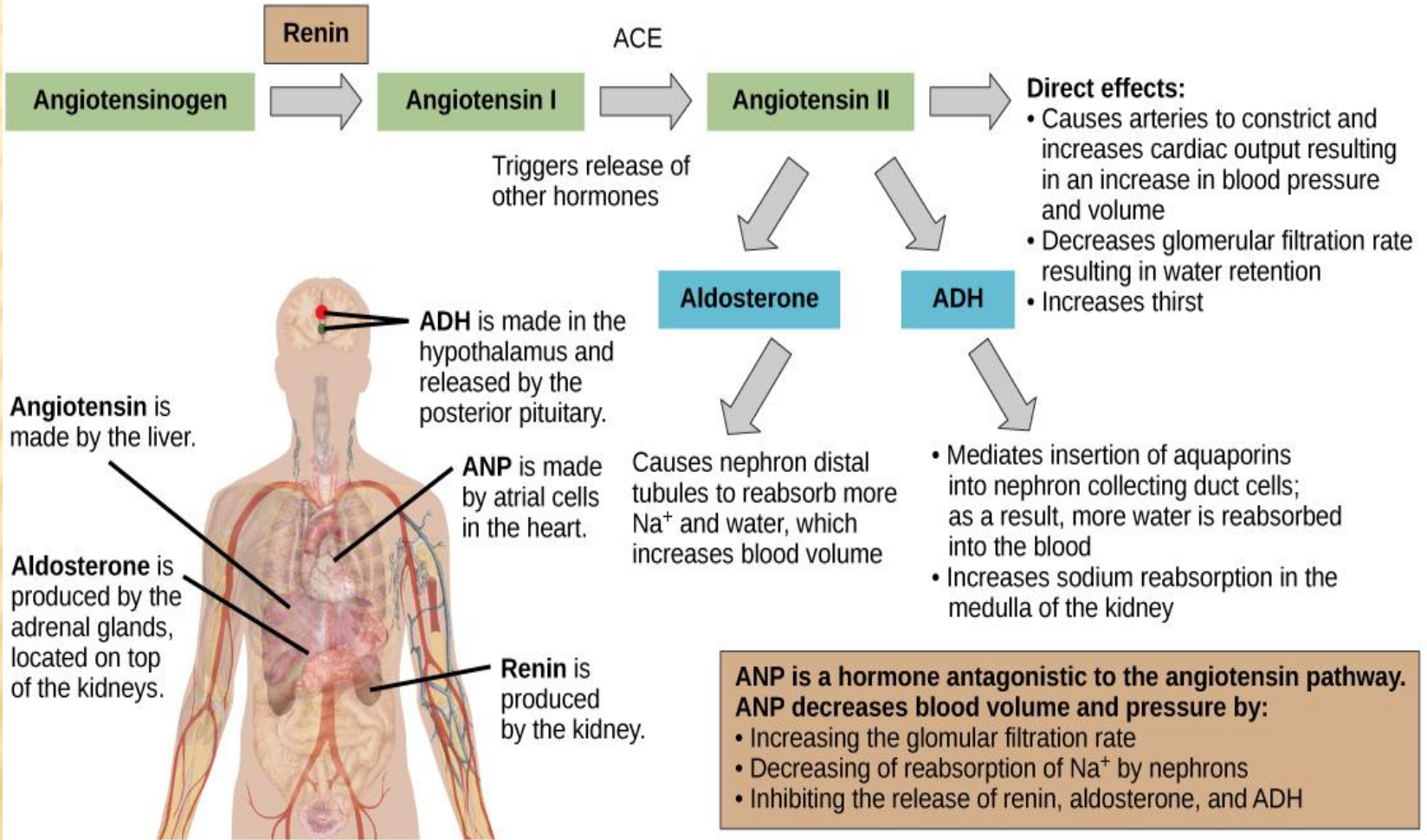
Hormones That Affect Osmoregulation

Hormone	Where produced	Function
Epinephrine and Norepinephrine	Adrenal medulla	Can decrease kidney function temporarily by vasoconstriction
Renin	Kidney nephrons	Increases blood pressure by acting on angiotensinogen
Angiotensin	Liver	Angiotensin II affects multiple processes and increases blood pressure
Aldosterone	Adrenal cortex	Prevents loss of sodium and water
Anti-diuretic hormone (vasopressin)	Hypothalamus (stored in the posterior pituitary)	Prevents water loss
Atrial natriuretic peptide	Heart atrium	Decreases blood pressure by acting as a vasodilator and increasing glomerular filtration rate; decreases sodium reabsorption in kidneys

How does the renin-angiotensin-aldosterone mechanism function? Why is it controlled by the kidneys?

- ❖ The **renin-angiotensin-aldosterone system** acts through several steps to produce **angiotensin II**, which acts to stabilize blood pressure and volume. Thus, the kidneys control blood pressure and volume directly.
- ❖ **Renin** acts on angiotensinogen, which is made in the liver and converts it to angiotensin I. ACE (angiotensin converting enzyme) converts angiotensin I to angiotensin II. Angiotensin II raises blood pressure by constricting blood vessels.
- ❖ It triggers the release of aldosterone from the adrenal cortex, which in turn stimulates the renal tubules to reabsorb more sodium.
- ❖ **Angiotensin II** also triggers the release of anti-diuretic hormone from the hypothalamus, which leads to water retention. It acts directly on the nephrons and decreases GFR.

The renin-angiotensin-aldosterone system increases blood volume and pressure



Endocrine Control of Osmoregulation in Fish

Hormones are critical to the physiological alterations necessary for ion homeostasis when fish move between freshwater and seawater.

Cortisol promotes seawater acclimation through differentiation of salt-secreting mitochondrion-rich cells and ion transport proteins in the gill. The growth hormone/insulin-like growth factor I axis is also important in seawater acclimation and acts in synergy with cortisol.

Prolactin (PRL) is important in freshwater acclimation through regulation of ion and water permeability in the gill, gut, and kidney.

Cortisol also promotes ion uptake and may interact with PRL during freshwater acclimation. For many species of fish, growth hormone promotes acclimation to seawater, PRL promotes acclimation to freshwater, and cortisol interacts with both hormones, thus having a dual osmoregulatory function.

Most of the recent work on the endocrine control of ion transport in fish has focused on the gill. It has been known for some time that the mitochondrion-rich chloride cell is the site of salt secretion .

There is substantial evidence indicating that the major transporters involved in salt secretion in the gill includes basolaterally located Na^+ , K^+ -ATPase (the sodium pump) and Na^+ , K^+ , 2Cl^- cotransporter (NKCC), and an apical Cl^- channel that appears to be homologous with the cystic fibrosis transmembrane conductance regulator (see [Fig. 2](#))

Chloride cells are **secretory cells** in the base of the gill filaments characterized by **numerous mitochondria** and an **extensive tubular system**.

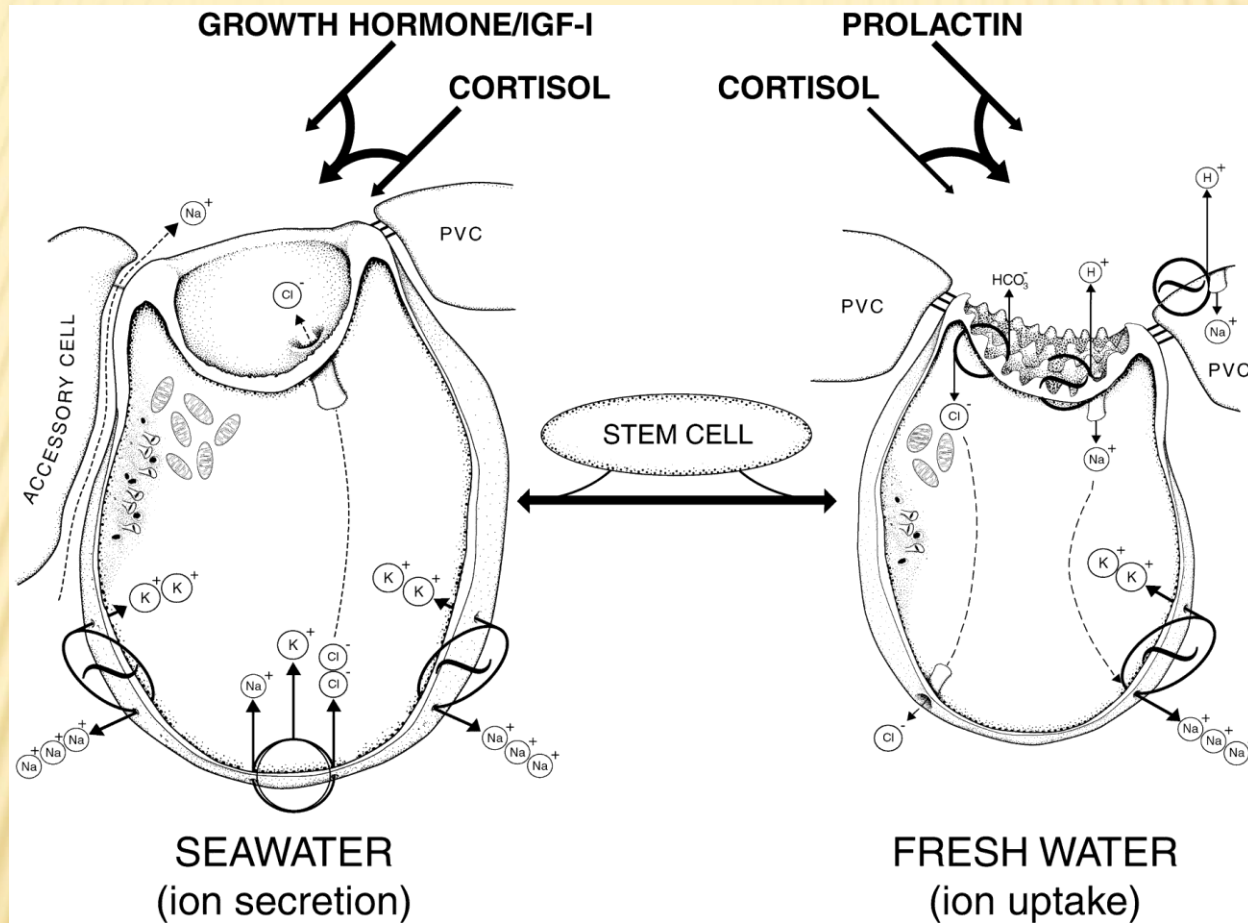
These cells are involved in the **secretion of excess ions** from the body fluid in sea water fishes, and possibly in **ion uptake** in fresh water fishes.

In euryhaline fish:

Transition from Fresh water to seawater will increase the number and size of the chloride cells. *The process is influenced by **Cortisol***

✦ While the migration from seawater to fresh water will reduce, or completely disappear those cells in some species. *The process is influenced by **Prolactin**.*

FIG. 2. MORPHOLOGY AND TRANSPORT MECHANISMS OF GILL CHLORIDE CELLS IN SEAWATER AND FRESH WATER.



Prolactin

- ✦ Prolactin has a role in ion uptake mechanisms of teleost fish in freshwater
- ✦ **PRL is an important freshwater-adapting hormone.**
- ✦ Gene expression, synthesis, secretion, and plasma levels of PRL all **increase** following exposure to **freshwater**.

PRL has been shown to **affect chloride cells**, both by **inhibiting** the development of seawater chloride cells and **promoting** the morphology of ion uptake cells "in fresh water fishes".

- ✦ In gastrointestinal tract of euryhaline fish, PRL generally **decreases NaCl and water absorption by reducing the permeability of the epithelium.**

Cortisol and Freshwater Acclimation

- ✦ Cortisol has a dual osmoregulatory function since it can be identified as a seawater-adapting hormone and also involved in **ion uptake in fresh water fish**.
- ✦ Cortisol treatment in freshwater fish **increases the surface area of gill chloride cells and the flow of sodium and chloride**, affecting both renal and branchial functions "gills function".

Cortisol and Seawater Acclimation

- ✦ Cortisol has largely been identified as a **seawater-adapting hormone** in a large number of teleost species.
- ✦ It has been shown for many species of euryhaline fish that treatment with cortisol in freshwater improves their subsequent survival and **capacity to maintain low levels of plasma ions after exposure to seawater.**
- ✦ This effect is due to increases in the **size and abundance of gill chloride cells.**
- ✦ Cortisol has also been shown to **increase the transcription and abundance of the major transport proteins involved in salt secretion by the gill.**

Growth hormone

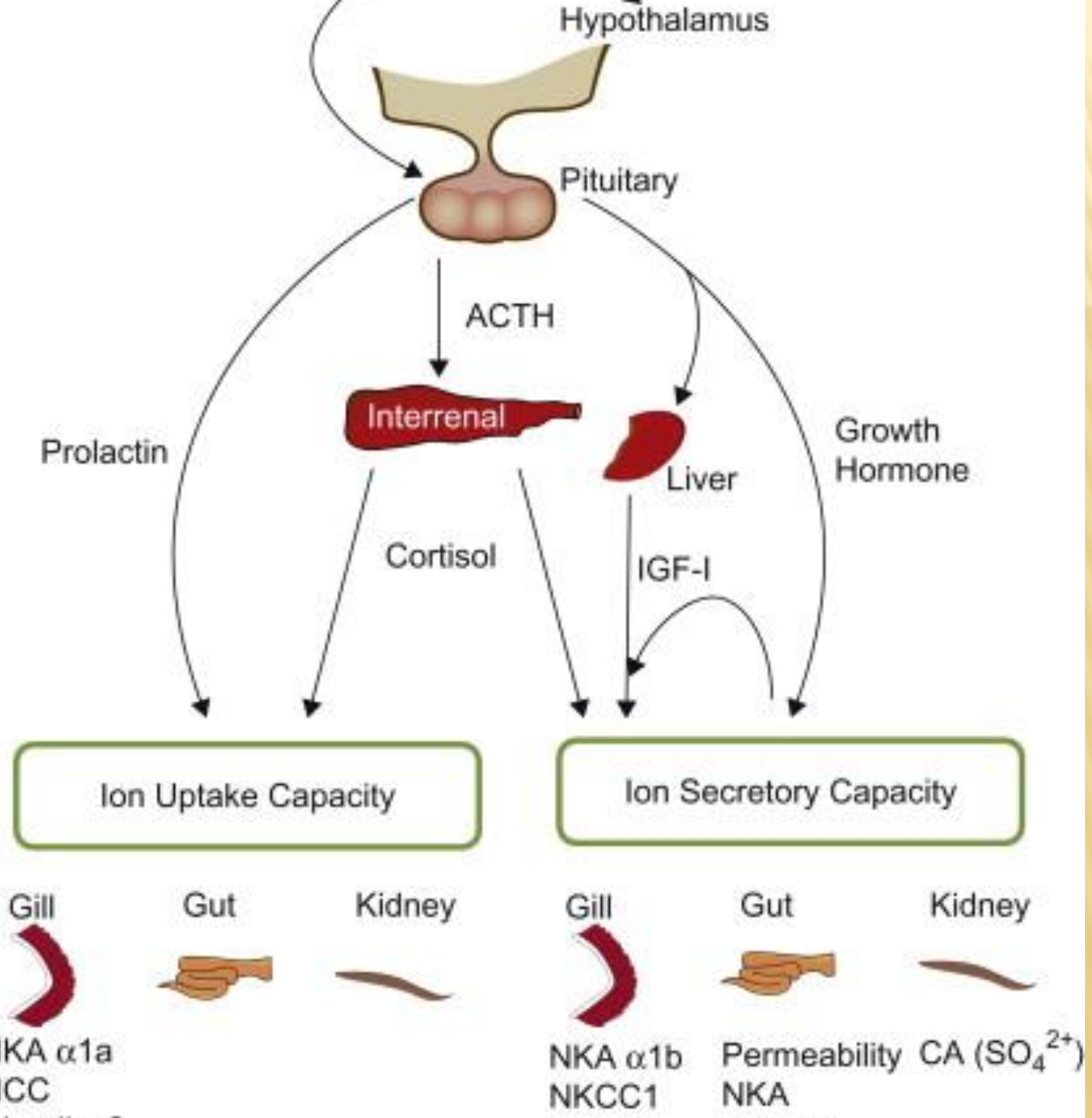
✦ Growth hormone is part of the anterior pituitary polypeptide hormones family.

✦ It stimulates growth, cell reproduction, and cell regeneration in humans and other animals.

✦ Growth hormone has the capacity to **increase the number and size of gill chloride cells ion transporters involved in salt secretion.**

Some of the interaction of **GH and cortisol may be through GH's capacity to upregulate the number of gill cortisol receptors**

✦ Growth hormone acts in synergy with cortisol to **increase seawater tolerance**, at least partly through the **upregulation of gill cortisol receptors**



Hormones controlling osmoregulation

fresh water fish	salt water fish
<p>Prolactin Cortisol</p> <p>the control of these endocrine systems for gill(chloride cell) to uptake ions like: Na⁺/Cl⁻</p>	<p>Growth hormone (GH) insulin-like growth factor-1 (IGF-I)</p> <p>the control of these endocrine systems for gill(chloride cell) , gut, and kidney, to secret ions like: Na⁺ ,K⁺ -ATPase</p>

SALMON MIGRATION AND HORMONAL CHANGES

A number of hormones secreted by the pituitary gland such as prolactin, corticotrophin and growth hormone etc. are responsible for osmo and ion regulation in salmon during migration.

Some of the hormones involved in salmon migration

-Hypothalmo pituitary gonadal (HPG) axis

Gonadal maturation which is controlled by different hormones causes the fishes to migrate long distance from sea to rivers or vice versa

-Insulin like growth factor-1 (IGF-1), prolactin, growth hormone and somatolactin-

In wild chum salmon, before starting migration the level of IGF-1 was high suggesting that IGF-1 stimulate the HPG-axis as a somatotropic signal.

Similarly prolactin and growth hormone, in addition to osmoregulation has an important role in final maturation.

Therefore growth hormone-1receptor mRNA, prolactin mRNA and somatolactin level increases near the spawning ground.