Renal Physiology 6:

# Potassium Balance

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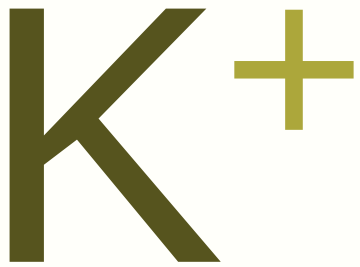
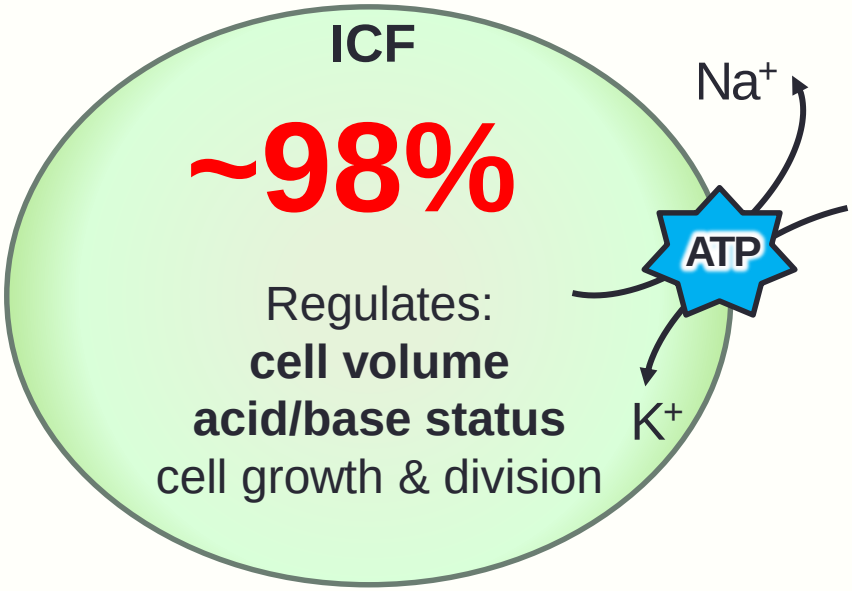


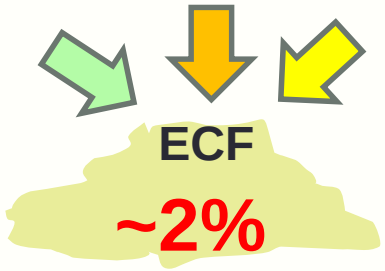
**Case 1:** A 60 year old man is followed for a 20 year history of essential hypertension. He has a moderate renal insufficiency with a baseline creatinine level of 2.5 mg/dl (estimated GFR of 20 ml/min) and a potassium level of 4.0 mEq/L (normal 3.5 - 4.8). He has heard that his blood pressure can be better controlled if he decreases his sodium intake. He replaces his table salt with salt substitute and his potassium rises to 5.4 mEq/L (creatinine is unchanged).

- Why his potassium level rose after starting the table salt substitute?

# $K^+$ is the most abundant cation in the body



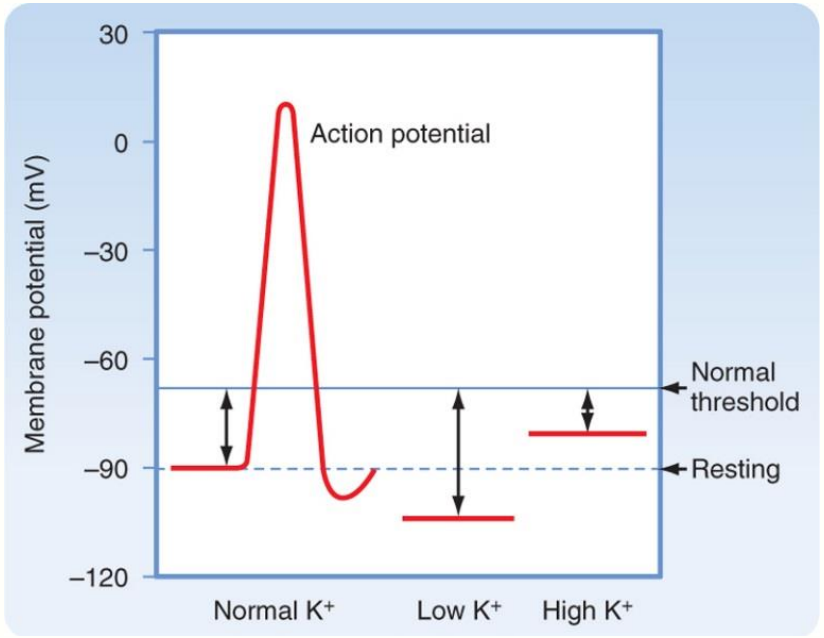

**precise control mechanisms**




Plasma [$K^+$] **3.5-4.8 mmol/L**

Regulates:
**membrane potentials** in excitable cells

- $K^+$ concentrations in equilibrium → Equal diffusion into and out of cell
- ⬇ EC $K^+$ → ⬆ diffusion of $K^+$ out of cell → cells hyperpolarized
- ⬆ EC $K^+$ → ⬇ diffusion of $K^+$ out of cell → cells partially depolarized



# $K^+$ Balance


GI intake
100 mmole/day

Gut

90
mmole/day

Feces
10 mmole/day

ECF
65 mmole
[K] = 4.5 mM

~2%

Muscle
2600 mmoles

Liver
250 mmoles

Bone
300 mmoles

RBC
250 mmoles

~98%

Reabsorbed 770
mmole/day

Secreted 50
mmoles/day

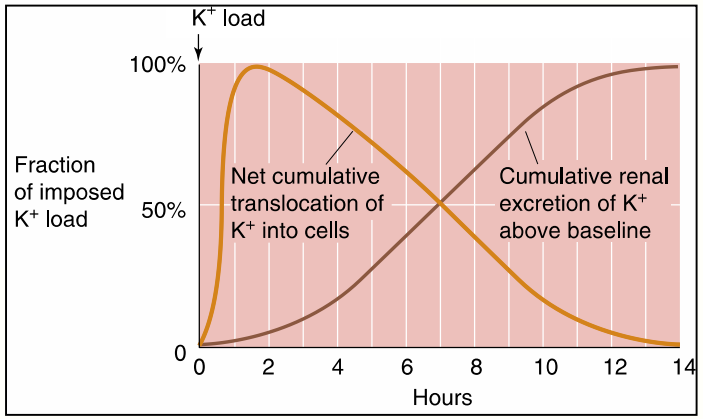
Filtered 810
mmoles/day

Kidneys

90
mmole/day

Control mechanism must be **sensitive** and **rapid**

- **Internal distribution** (regulates extracellular [$K^+$])
- **Renal $K^+$ excretion** (regulates total body $K^+$)



# Internal potassium distribution

# ↑ $K^+$ uptake into the cells is due to:

- **Insulin** ↑ after high $K^+$ meal.

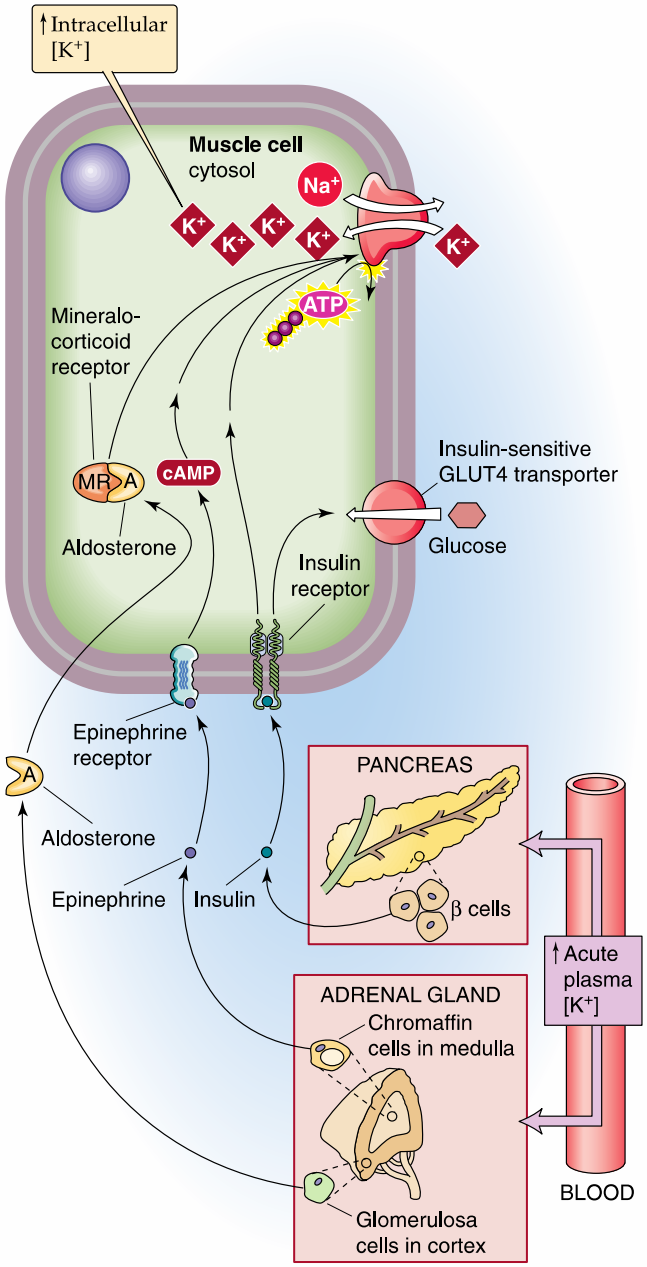
Insulin + glucose to treat hyperkalaemia.

- **Adrenaline** via $\beta_2$ receptors

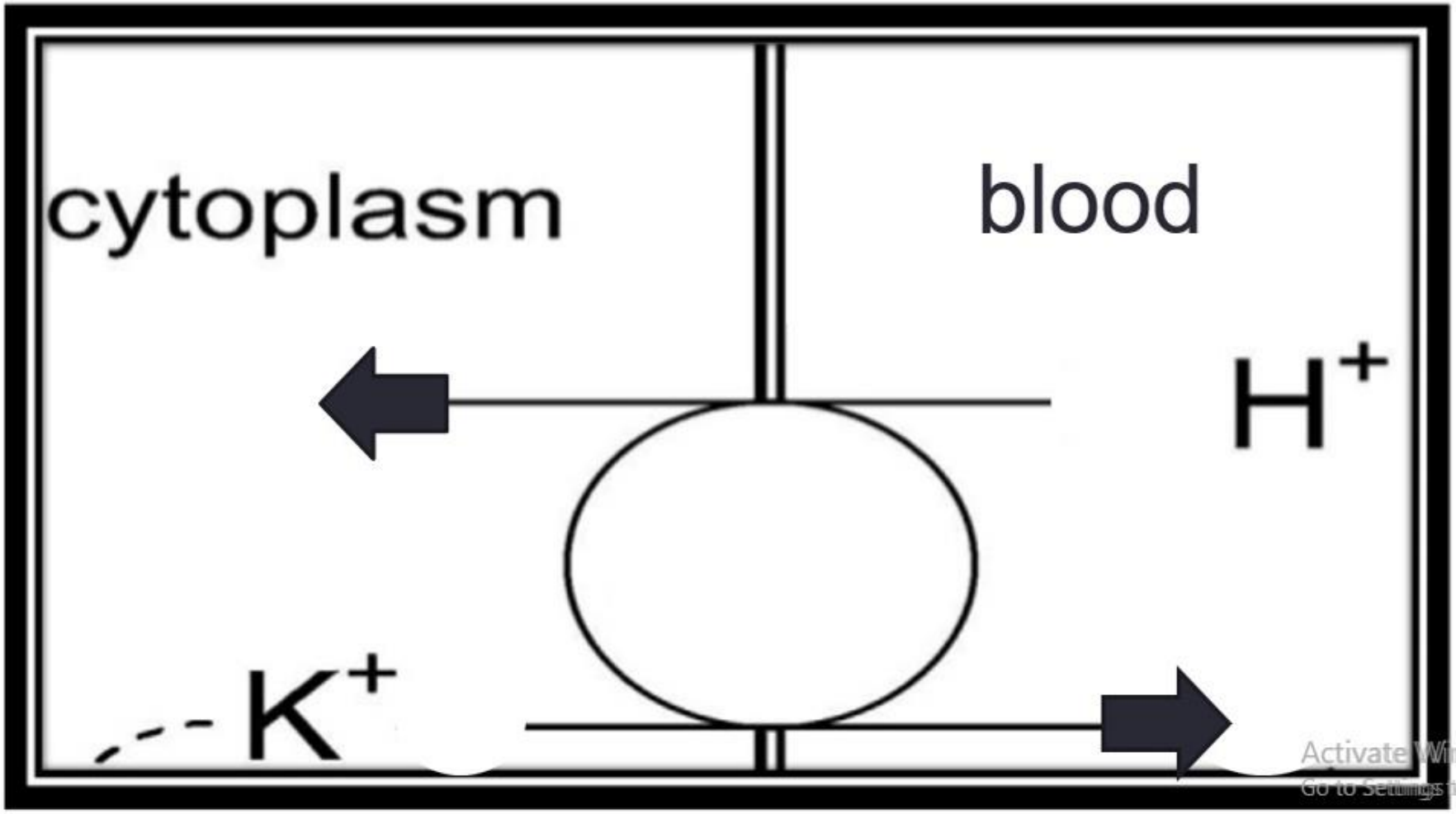
β blockers ↑ plasma $K^+$ after a meal or an exercise ☠.

- **Aldosterone**

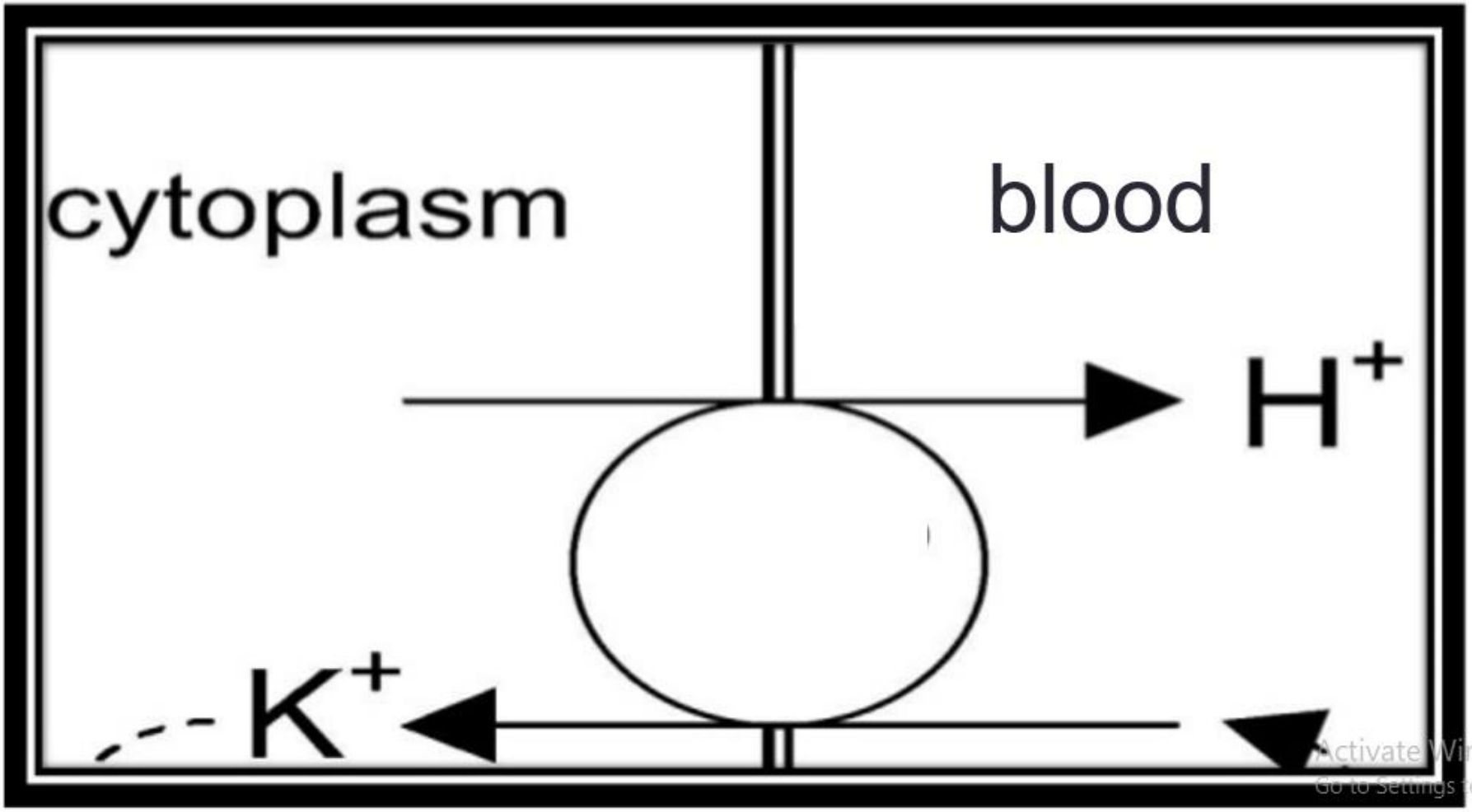
- **Alkalosis** $H^+$ is "*exchanged*" for extracellular $K^+$.



# During Acidosis

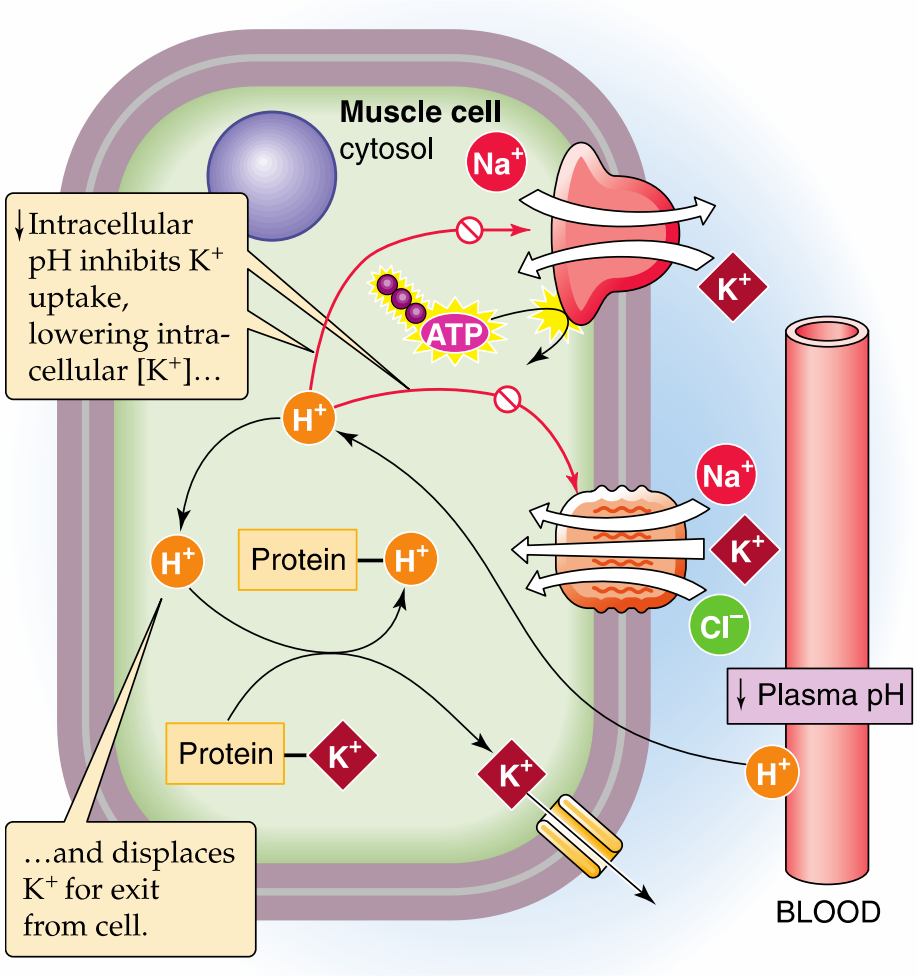


# During Alkalosis



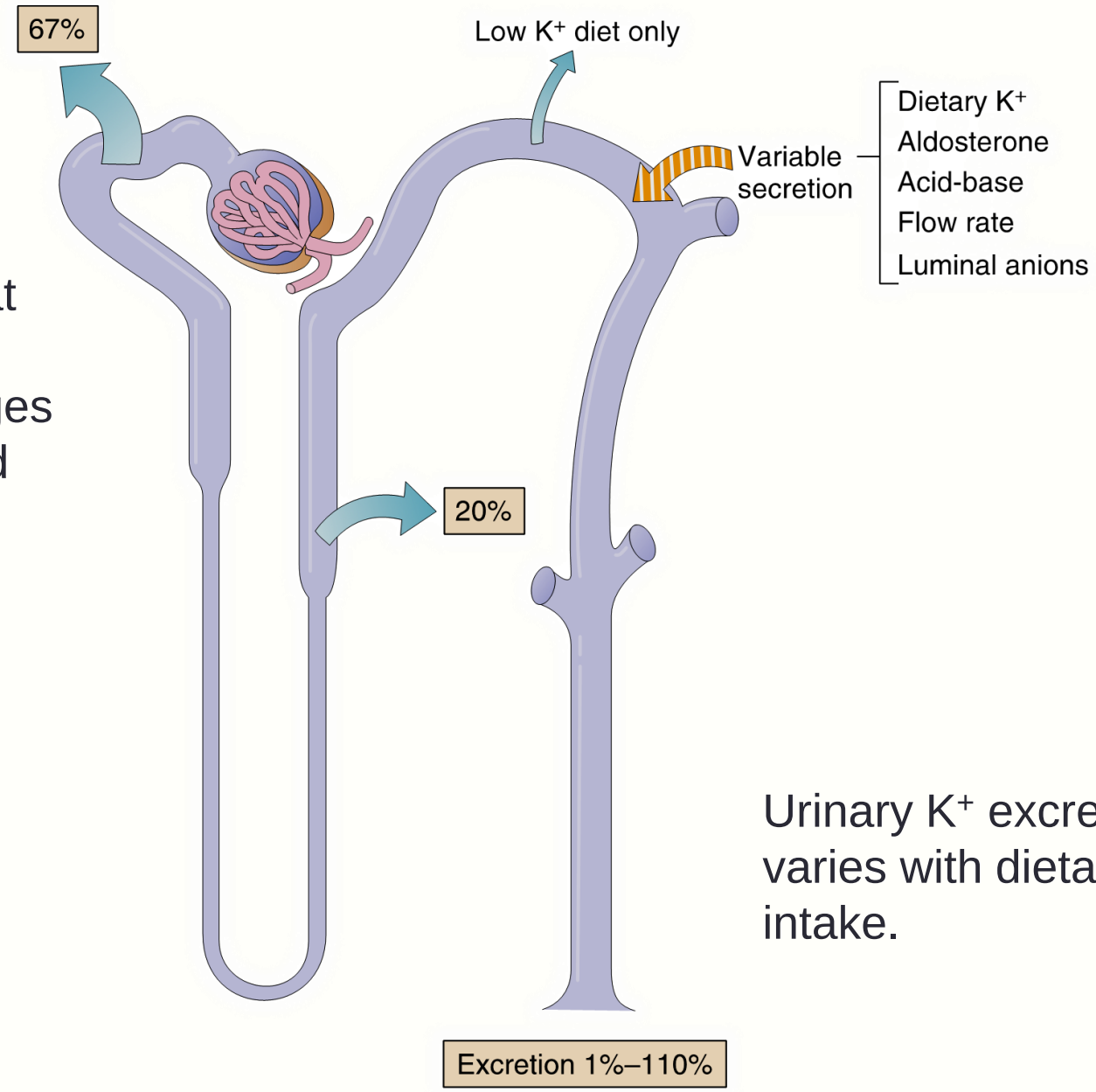
# ⬆ Plasma $K^+$ levels can be due to:

- **Acidosis:** ICF $K^+$ is "**<u>exchanged</u>**" for extracellular $H^+$.
- **⬆ Osmolality** → $K^+$ moves out secondary to $H_2O$ movement out of cells
- **Exercise** → loss of $K^+$ from muscles
- **Cell lysis** → release of cellular contents



# Renal excretion of potassium

$K^+$ reabsorption at PCT does not respond to changes in $K^+$ balance and are not physiologically regulated.




Urinary $K^+$ excretion varies with dietary $K^+$ intake.

# Major Factors and Hormones Influencing $K^+$ Excretion

**Homeostatic:** Keep $K^+$ Balance Constant

- Plasma [$K^+$] (⬆ $K^+$ excretion)
- Aldosterone (⬆ $K^+$ excretion)

**Pathophysiological:** Displace $K^+$ Balance

- Flow rate of tubule fluid (⬆ $K^+$ excretion)
- Acid-base balance

# Plasma [$K^+$]

**Hyperkalaemia** stimulates secretion of $K^+$ within minutes

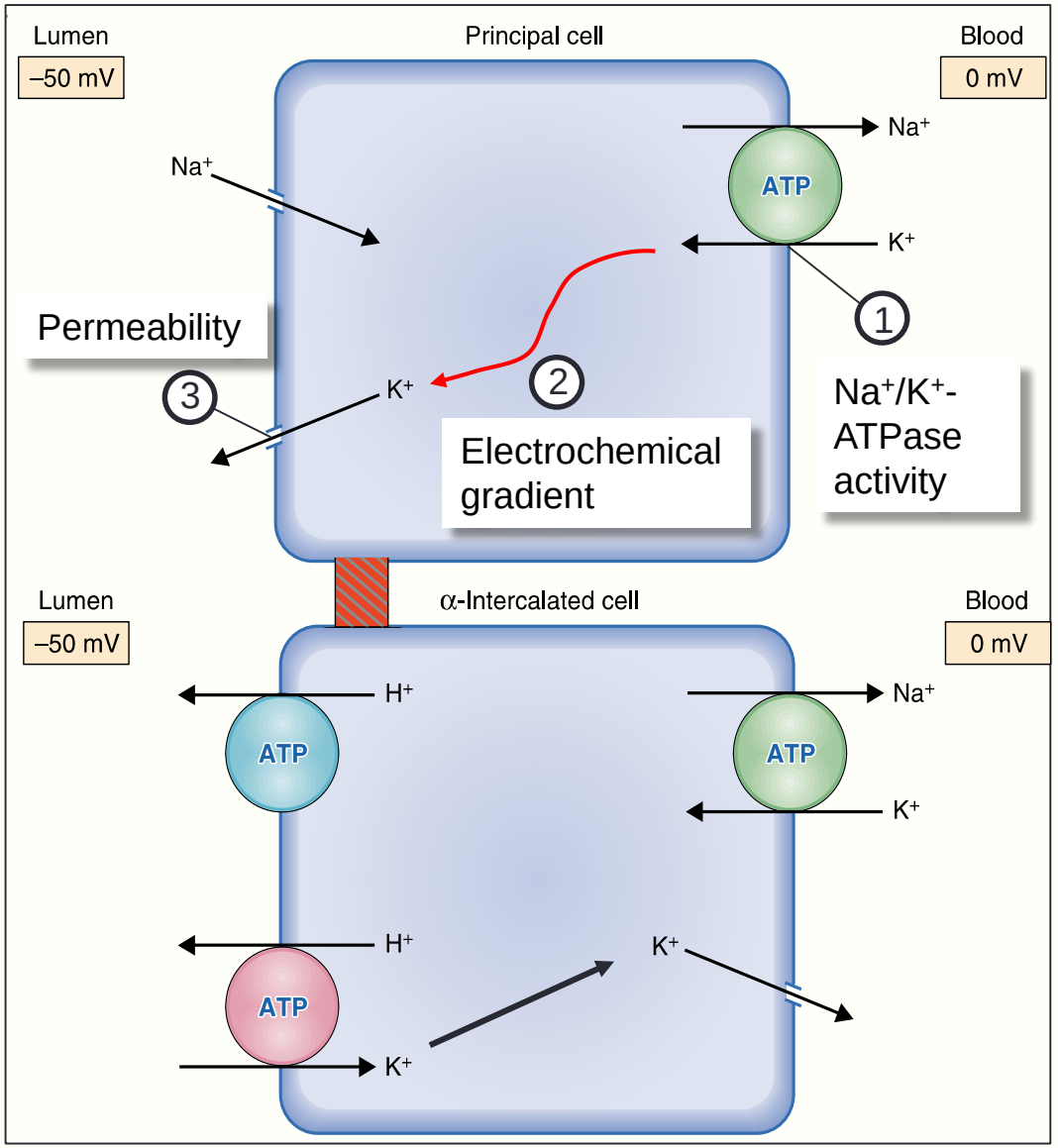
## How?

**1. Stimulates Na/K-ATPase** → ↑ $K^+$ uptake (*basolateral*) → ↑ electrochemical gradient.

**2.** ↑ **permeability** to $K^+$ (*apical*).

**3.** ↑ **aldosterone** → ↑ secretion of $K^+$.

**Hypokalaemia** produces an opposite effect



# Aldosterone

## ↑ $K^+$ secretion by:

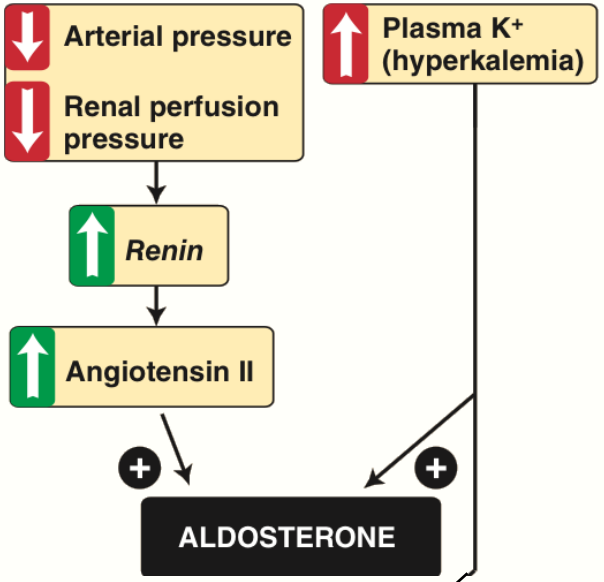
**1.** ↑ Na/K ATPase → ↑ $Na^+$ reabsorption → ↑ $K^+$ secretion.

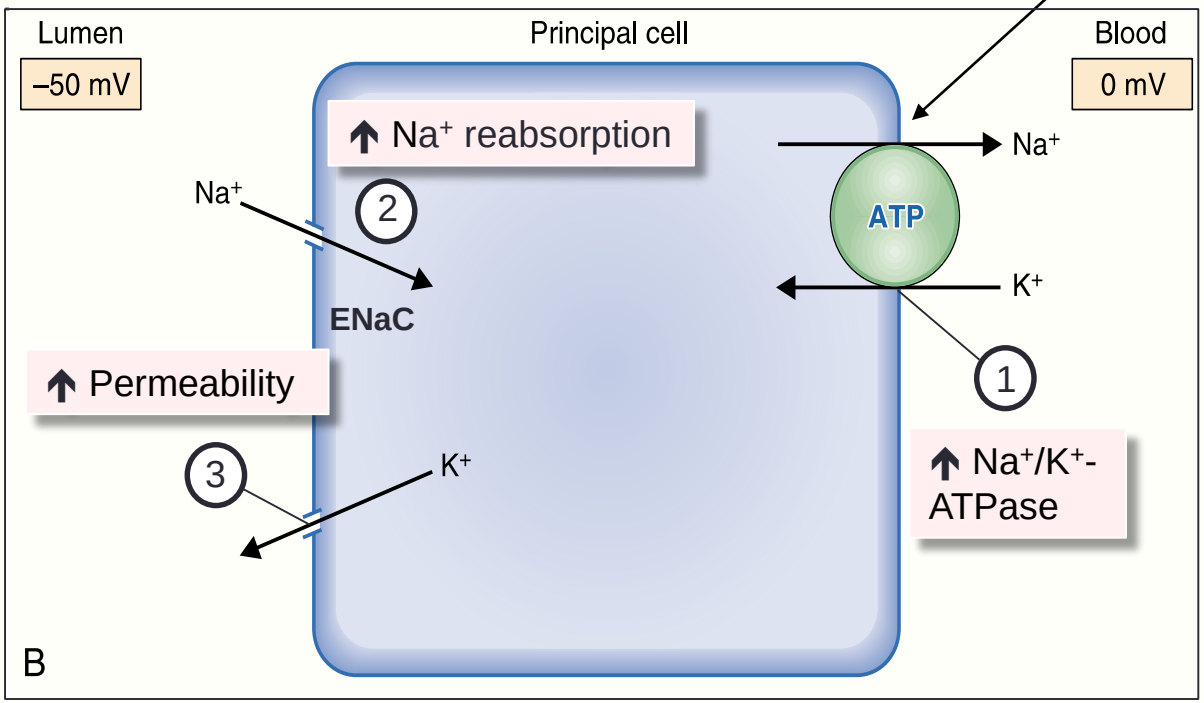
**2.** ↑ Na+ reabsorption (↑ ENaC) → -ve lumen potential → ↑ $K^+$ secretion

**3.** ↑ permeability of apical membrane → ↑ $K^+$ secretion

**Conn's syndrome** (↑ aldo) → hypokalaemia

**Addison's disease** (↓ aldo) → hyperkalaemia



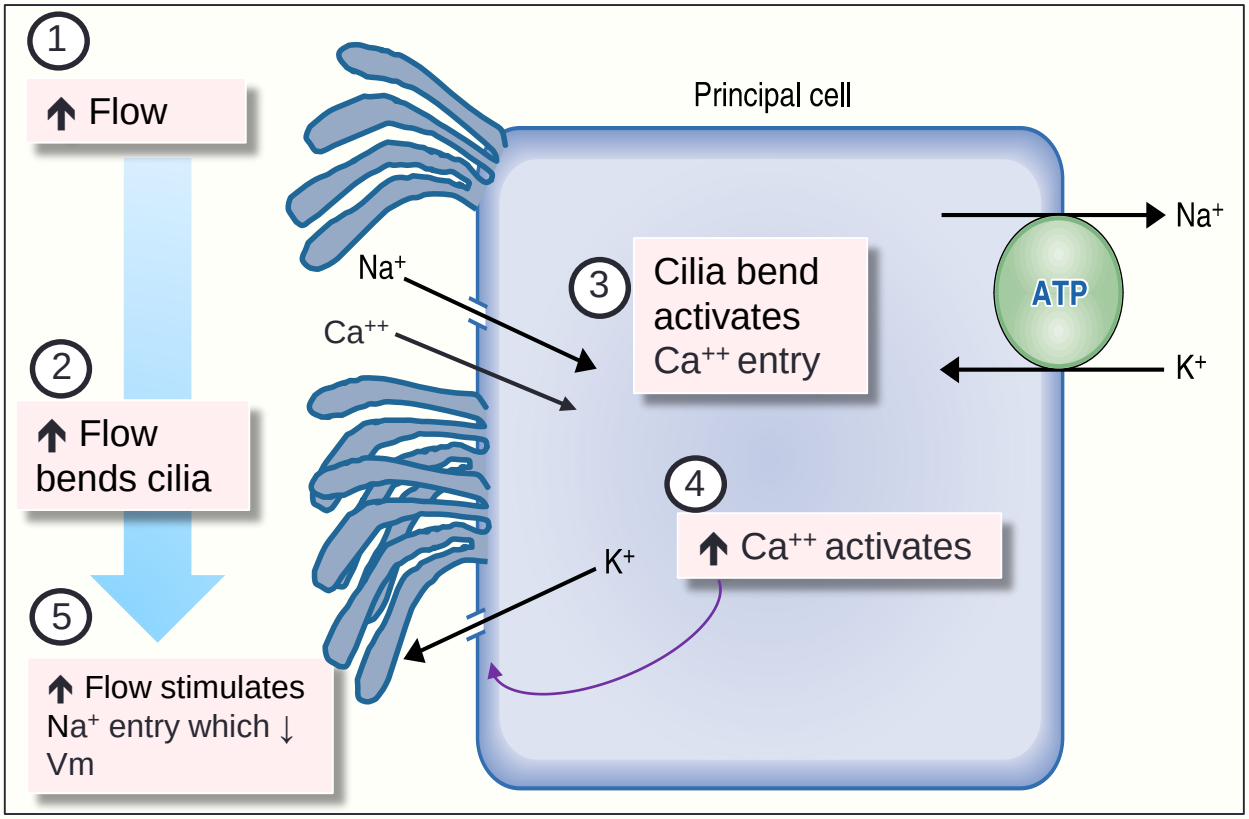



# Flow rate of tubule fluid

↑ **Flow rate of tubule fluid** → ↑ $K^+$ secretion because it causes:

Cilium bending → ↑ $Ca^{++}$ entry → activates $K^+$ channels (apical)

Secreted $K^+$ flushed down the tubule → maintain the gradient for $K^+$ diffusion across the luminal membrane

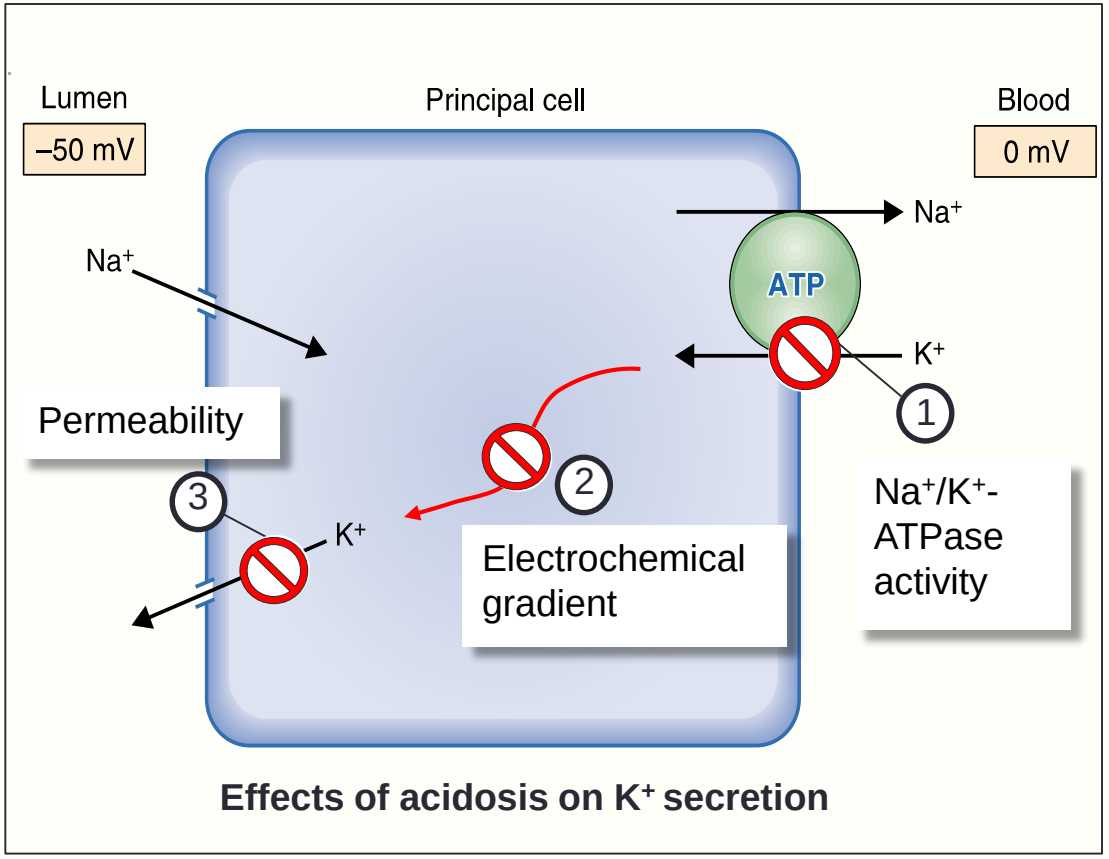



↑ $Na^+$ reabsorption (principle cell) → ↑ $K^+$ uptake by $Na^+/K^+$ ATPase

# Acid-base balance

## Acidosis inhibits $K^+$ secretion in principal cells by INHIBITING ⊘:

- **Na/K ATPase** → ↓ $K^+$ uptake from blood → ↓ conc. gradient for $K^+$ efflux into the lumen.
- **$K^+$ channels** (*apical*) → ↓ $K^+$ secretion directly → hyperkalemia.




**Alkalosis** has the opposite effect, promoting $K^+$ secretion and hypokalemia.