POTASSIUM

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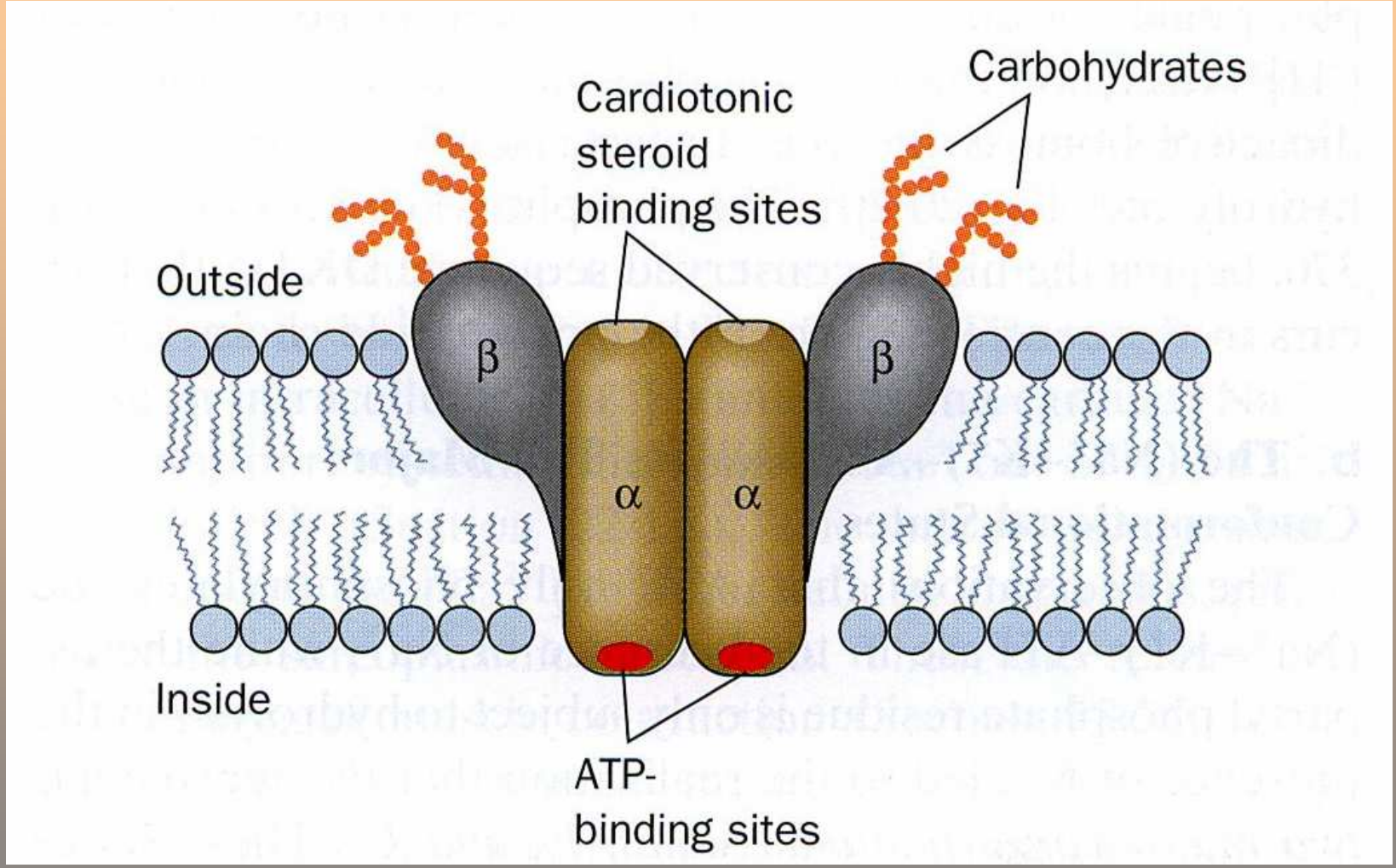
K^+ - the most abundant cation in the human body

- **Reference values /S, P = 3.8 – 5.1 mmol/l** (adults)

- ↑ *concentration = hyperkalemia*
- ↓ *concentration = hypokalemia*

- **total body K^+ ~ 3.5 - 4 mol**
- **located predominantly IC: 3.5 mol IC (98%); 0.05 mol EC (2%)**
- **The difference is maintained by the action of Na/K pump situated in the cell membrane.**

Na^+/K^+- ATPase



$3\ Na^+$ (in) + $2\ K^+$ (out) + ATP + $H_2O \leftrightarrow 3\ Na^+$ (out) + $2\ K^+$ (in) + ADP + P_i

K^+

- RDI = 100 mmol = 3 - 4 g
- dietary sources: apricots, animal products, whole grains, legumes, pumpkins, watermelons, raisins, bananas, and spinach
- urinary excretion: 40 – 90 mmol / day;
  increased by aldosterone, ↑ $[K^+]$ in renal tubular cells, ↑ urinary flow rate
- stools excretion: 10 mmol / day

Regulation of kalemia

- **renal handling** (aldosterone, K^+ content, flow rate)
- **Na/K-ATPase** of the cell membranes
- **acid-base status:**
- **acidosis:** $[H^+]/P$ ↑, H^+ move into cells, where they bind phosphate buffer and K^+ are released and move out to maintain electroneutrality → **$[K^+]/P$ ↑**
- **alkalosis:** a reverse process occurs: EC K^+ move into cells to bind the phosphate buffer → **$[K^+]/P$ ↓**
- **↑ plasma tonicity** ↑ $[K^+]/P$

K^+ significance

- **maintenance of resting cell membrane potencial → critical to cardiac and neuromuscular electrical activity**
- **maintenance of IC osmolality → IC volume**
- **Its optimal concentrations are nessesary for enzymatic reactions of proteosynthesis, growth, and IC metabolic processes.**
- **regulation of hormone secretion** (insulin, glucagon, aldosterone, catecholamines)

CAUSES OF HYPOKALEMIA

- **K^+ redistribution**
- **alkalosis**
- **insulin therapy, β-agonist therapy**
- **treatment of megaloblastic anemia (K^+ is needed for new ercs)**

- **K^+ depletion**
- **impaired intake**
- **↑ urinary loss (polyuria, diuretics, hyperaldosteronism, renal tubular acidosis)**
- **↑ gastrointestinal loss (diarrhea, laxative abuse, vomiting)**

CLINICAL SYMPTOMS OF HYPOKALEMIA

- **Renal:** ↓ of concentrating ability, ↑ H^+ and phosphate excretion
- **Neuromuscular:** hyperpolarization of the cell membrane → Na^+ influx → muscle weakness and adynamic ileus in GIT
- **Cardiovascular:** delayed repolarization and rhythm disturbances (tachycardias, ectopy), ↓ myocardial contractility
- **Metabolic:** ↓ release of insulin, STH, renin, and aldosterone

CAUSES OF HYPERKALEMIA

- K^+ redistribution
- acidosis
- insulin deficiency
- tissue/cell breakdown
- hypertonicity
- ↑ K^+ load (oral or i.v.)
- ↓ K^+ excretion
- renal failure
- hypoaldosteronism
- K^+-sparing diuretics
- distal tubule disorders

CLINICAL SYMPTOMS OF HYPERKALEMIA

include life-threatening complications!

- **Neuromuscular:** depolarization of the cell membranes + hyperpolarization due to inactivation of Na^+ channels → **muscle weakness (respiratory muscles!;** $[K^+]/P > 8$ mmol/l)
- **Cardiovascular:** **bradyarrhythmias (**$[K^+]/P > 6.5$ mmol/l), **asystoly (**$[K^+]/P > 8$ mmol/l)
- **Metabolic:** ↑ **release of insulin, glucagon, aldosterone, and prostaglandins**

Pseudohyperkalemia

= ↑ [K^+] in a test-tube, normal [K^+] in examined patient

- **Caused by a release of K^+ from the blood cells (IC space) to plasma/serum as a result of haemolysis.**