Potassium

Potassium (K^+) is the chief cation found within the intracellular fluid of all cells. The total body content of K^+ is around 3,500 mmol, of which 98% is contained in the **intracellular fluid (ICF)** and only 2% is found in the **extracellular fluid (ECF)**. Potassium cations are involved in the energetic metabolism of cells as they are needed for the synthesis and breakdown of **adenosine triphosphate** (ATP). Increasing intracellular concentration of K^+ is typical for anabolic states, while decreasing intracellular concentration of K^+ is consistent with catabolic states. K^+ is bound to proteins and glycogen within cells and therefore is released during their breakdown.

Serum concentration of K^+ ($S\text{-}K^+$), also called **kalemia** is closely associated with pH levels of both ICF and ECF. Acidemia is consistent with hyperkalemia (increasing $S\text{-}K^+$), while alkalemia results in hypokalemia (decreasing $S\text{-}K^+$). For every change of pH by 0.1 points, kalemia decreases by approximately 0.6 mmol/L. [1]

Reference values:

- plasma: 3.8–5.4 mmol/l
- urine: 45–90 mmol/day
- daily intake and expenditure 50–100 mmol (2–4 g).[2]

Regulation

- Normal serum potassium values must be strictly maintained between 3.8–5.4 mmol/L, pathological hypokalemia and hyperkalemia can lead to cardiac arrhythmias (changes in $S\text{-}K^+$ influence the resting membrane potential and excitability of cardiomyocytes)
- $S\text{-}K^+$ is maintained by excretion and reabsorption of K^+ in distal tubules and collecting ducts of the nephron in the kidney
- $S\text{-}K^+$ is influenced by:
  - intake of K^+ in food;
  - sodium (Na^+) levels and renal tubular flow;
  - acid-base balance (ABB);
  - mineralocorticoid (aldosterone) activity;
  - sensitivity of cells of the distal tubules to mineralocorticoids;
  - type and interchangeability of anions.
- It is hypothesised that a rapid increase in concentration of K^+ in the ECF may lead to increased glucagon secretion → hyperglycemia → increased insulin secretion → increased uptake of glucose into muscle and adipose cells → glucose-K^+ cotransport into cells → normalisation of kalemia (in the ECF)[1]
- The concentration gradient between ECF and ICT (cca 110–140 mmol/l) is maintained by a Na^+/K^+-ATPase pump in the cellular membrane

Hypokalemia

- Causes:

1. **Losses in the GIT**:
   - acute and chronic diarrhoea;
   - laxative overuse;
   - vomiting;
   - fistulas.
2. **Renal losses**:
   - diuretic therapy;
   - polyuric phase of **acute kidney injury (AKI)** – also *acute renal failure (ARF)*;
   - renal tubular acidosis;
   - hyperaldosteronism;
   - Cushing syndrome and/or exogenous glucocorticoid use.
3. **Alkalosis**
4. **Infusion of IV fluid** (without K^+)
5. **Diet low in potassium**

- Signs & symptoms – weakness, ileus, renal disease, arrhythmias, EKG – flattening and inversion of T waves, visible U wave, QT interval prolongation
- *Therapeutic substitution (calculation)* – **K [mmol] = ECF x (4.4 – measured K) x 3 + K substitute for losses**
  - Therapeutic substitution of potassium is contraindicated in oliguria or anuria. [2]

Hyperkalemia

- Causes:

1. **Decreased excretion (renal cause)**:
   - oliguria and anuria in **acute kidney injury** (AKI);
   - terminal phase of chronic kidney disease (CKD)
   - renal tubular acidosis (type 4, hyperkalemic RTA);
   - adrenal insufficiency;
   - K^+-sparing diuretics.
2. **Movement of K^+ from ICF to ECF**:
   - acidosis (acute);
   - increased catabolic processes in cells or necrosis.
3. **Increased intake**
4. **Low levels of mineralocorticoids**[1]

- Signs & symptoms: bradycardia, malignant arrhythmias (ventricular tachycardia and fibrillation), changes in EKG – **peaked T waves**, PQ interval prolongation, wide QRS, **ST wave depression**.
- The **role of insulin** in regulation of kalemia:
  - insulin activates H^+/Na^+ antiport → increased intracellular concentration of Na^+ → increased activity of **Na^+/K^+ATPase** → Na^+ is pumped out of the cell and K^+ is pumped into the cell → extracellular concentration of K^+ **decreases**

Pseudohyperkalemia

- Increased S-K^+ in significant thrombocytosis (K^+ cations are released from the thrombocytes that break down during the blood clot formation in the test tube)[1];
- Hemolysis (*in vitro*); disintegration of erythrocytes during a blood test – occurs due to improper specimen sample handling.

References

Related articles

- Hypokalemia
- Hyperkalemia
- Acidosis
- Alkalosis

External links

- Potassium & CKD Diet [National Kidney Foundation] (https://www.kidney.org/atoz/content/potassium)
- Potassium [StatPearls NCBI] (https://www.ncbi.nlm.nih.gov/books/NBK539791/)

References

1. MASOPUST, Jaroslav – PRŮŠA, Richard. *Patobiochemie metabolických drah.* 2. edition. Univerzita Karlova, 2004. 208 pp. pp. 174–175.
2. SCHNEIDERKA, Petr. *Kapitoly z klinické biochemie.* 2. edition. Karolinum, 2004. ISBN 80-246-0678-X.

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- MASOPUST, Jaroslav – PRŮŠA, Richard. *Patobiochemie metabolických drah.* 2. edition. Univerzita Karlova, 2004. 208 pp. pp. 174–175.

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- wikipedia: Hyperkalemia