

Potassium chloride

CAS: 7447-40-7

MF: KCl

MW: 74.5

logKow= -0.46 at 20°

Solubility in water: 34.4 g/100 ml; in ethanol: 40 g/100 ml; insoluble in acetone.

K⁺

MW 39.1

Major uses

Potassium chloride (KCl) is inorganic salt used for making fertilizers, since the growth of many plants is limited by their potassium intake. Potassium in plants is important for the osmotic and ionic regulation, plays a key role in the water homeostasis and is closely connected with processes involved in the protein synthesis.

KCl is also used for the manufacture of potassium hydroxide and potassium metal; for food processing; and for experimentation in laboratory.

KCl is an important therapeutic agent, which is used mainly in the treatment of hypokalemia and associated conditions. Hypokalemia (potassium deficiency) is a potentially fatal condition in which the body fails to retain sufficient potassium to maintain health.

Human toxicity

Usual oral therapeutic doses of KCl are 1.5-3.0 g/day to prevent depletion of potassium. Patients with normal renal function ingesting KCl usually have minimal toxicity. However, ingestion of larger amount of salt can result in hyperkalemia (potassium accumulates in plasma at high concentrations). Toxic effects may include: vomiting, diarrhea, abdominal cramps, and weakness. Cardiac toxicity may occur in patients with normal renal function, but with underlying cardiac disease, even with therapeutic doses. Patients with renal insufficiency may develop toxicity from even small doses of KCl.

Hyperkalemia may result in depolarization of cardiac muscle and subsequent changes in cardiac muscle excitability. This may result in hypotension, cardiac dysrhythmia, heart block, and cardiac arrest.

At overdosage of KCl, the major life-threatening effect is on the cardiovascular system, where bradycardia, vascular collapse, and cardiac arrest may develop rapidly.

Neurologic symptoms may range from mild muscle crumps, listlessness, and confusion to paralysis [1].

The upper limit of normal potassium concentration in serum or plasma is variously given as 4.5 up to 5.5 mEq/l (4500-5500 μ M). Potassium levels under 6.5 mEq/l (corresponds to 254.2 mg/l or to 6500 μ M) rarely manifest cardiac toxicity.

Moderate toxicity was reported at serum potassium levels between 250 and 310 mg/l, whereas a severe toxicity, sometimes with a fatal end, was observed at serum potassium levels over 300 mg/l [1]. However, in some cases patients survived at serum potassium concentrations over 360 mg/l [2].

The mean clinically measured acute lethal serum concentration of potassium, based on data from several handbooks, was 370 mg/l [3].

Carcinogenicity: no evidence of carcinogenicity [1].

Kinetic data

Absorption: KCl is completely absorbed from the gastrointestinal tract [3].

Kinetics: multiphasic [3].

Volume of distribution: not reported.

Distribution: Potassium is accumulated intracellular by energy-dependent mechanism that extrudes sodium. There is a high concentration gradient for potassium from cell to extracellular fluid, and a high gradient for sodium in the opposite direction.

Potassium leaves the plasma space very rapidly (half-life approximately 16 sec) by transcapillary transfer. It distributes preferentially to kidney, gut, liver, skin, and muscle (in that order) [1].

Plasma half-life: not reported.

Time to peak blood concentration: 0.5 h [3].

Plasma protein binding: none [3].

Passage of blood-brain barrier: free? [3]. Knowledge is insufficient.

Elimination: the main route of elimination is renal. Most filtered potassium is absorbed in the proximal tubule by passive diffusion, and excreted in the urine through the distal part of the renal tubule [1].

Metabolism and excretion

Potassium (K^+) is an essential constituent of the body for intracellular osmotic pressure and buffering, cell permeability, acid-base balance, muscle contraction and nerve function.

The metabolism of the potassium ion may be considered pathological when its concentration in either the extracellular or intracellular fluid is above (hyperkalemia) or below (hypokalemia) normal concentration (see Human toxicity, above).

Excretion: mainly in the urine (80-90%); only a small amount is excreted in the feces, sweat, saliva and tears. An excess hydrogen ion concentration inhibits excretion, while alkalosis favors excretion [1].

Toxicological mechanisms

K^+ is the predominant cation in intracellular fluid (together with other cations, Na^+ , Ca^{++} and Mg^{++} , which are presented in much lower concentrations compared to K^+). The high intracellular – low extracellular concentration of potassium and the reverse for sodium favor movement of potassium out of and sodium into cells. Such ionic gradients are maintained by the presence of sodium-potassium pump in the cell membrane and are critical in protecting cell volume [3, 4].

At poisoning situation, when concentration of potassium in plasma is elevated, the normal transmembrane potential is not maintained. It leads to adverse effects, such as a disturbance of the rhythm of the heart (a slow, weak pulse, heightened T waves of

ECG, arrhythmias, heart block) and eventually weaken cardiac contractility (fall in blood pressure) [1, 4].

Target organs: heart (muscle), CNS [3].

References

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4. Haddad, L.M. & Winchester, J.F. (1990) *Clinical Management of Poisoning and Drug Overdose*. 2nd ed., pp.102-110. W.B. Saunders Company.

Written by Ada Kolman, January 2006; revised March 2007
ada.kolman@telia.com