23. Potassium

Physiology

Potassium is predominantly an intracellular cation. This compartmentalisation is maintained by the energy-dependent cellular uptake of the element and simultaneous excretion of sodium by the cell membrane bound enzyme Na-K ATPase. This process is fundamental to the cellular uptake of molecules against electrochemical and concentration gradients, to the electro-physiology of nerves and muscle, and to acid-base regulation [1,2,3].

An adult male contains approximately 40-50 mmol (1.6-2 g)/kg body weight, on which basis a 75 kg adult would contain 3000-3750 mmol (117-147 g) potassium. At least 95 % of this is intracellular at an activity concentration of 150 mmol (5.9 g)/L, the remainder is in the ECF at a concentration of 3.5-5.5 mmol (137-215 mg)/L. The total body potassium reflects lean tissue mass and consequently varies with muscularity.

The systemic homoeostasis of potassium is understood imperfectly. Over 90 % of dietary potassium is absorbed in the proximal small intestine, possibly by a combination of diffusional mechanisms and solvent drag. The body content is regulated by the renal glomerular filtration and tubular secretion but up to 10 % of the daily loss of potassium can occur via the distal ileum and colon; additionally a small amount is lost in sweat. The glomerular filtration of potassium is approximately 3 % of that for sodium, and amounts to only about 680 mmol (26.5 g)/d; however, renal tubular secretion of the element, which is regulated predominantly by aldosterone and other mineralocorticoids, is highly efficient and the kidney is able to excrete potassium considerably in excess of its filtered load. As long as renal function is normal, on habitual dietary intakes it is almost impossible to induce potassium excess. An additional but usually less important regulation of ECF and plasma potassium excess is achieved by the capacity of cells induced by glucose and insulin to take up the element.

Deficiency and excess

Potassium deficiency arising from inadequate dietary intake is unlikely because of the ubiquity of potassium in all foodstuffs. Potassium deficiency alters the

electrophysiological phenomena of cell membranes. This causes weakness of skeletal muscles and the effect on cardiac muscle is reflected by electrocardiographic changes characteristic of impaired polarisation, which may lead to arrhythmia and cardiac arrest. Similar functional changes in intestinal muscle cause intestinal ileus. Mental depression and confusion can also develop.

The reported intakes of potassium by Western populations are 40-150 mmol (1.65-9 g)/d [4]. An inverse correlation exists between increased blood pressure and urinary potassium excretion or urinary Na:K excretion ratios [4]. An adequate potassium intake is needed to achieve effective homoeostasis of sodium. Young normotensive men on a potassium intake of 10 mmol/d (390 mg/d) were less able to excrete an imposed sodium excess than when they had a potassium intake of 90 mmol (3.5 g)/d [5]; simultaneously their blood pressure increased. In the Intersalt study urinary potassium excretion, an assumed indicator of potassium intake, was negatively related to blood pressure as was the urinary Na:K concentration ratio [4]. Increasing potassium intakes to levels achievable with customary diets [i.e. 65 and 100 mmol/d (2.5 and 3.9 g/d)] reduced blood pressure in normotensive and hypertensive individuals and increased urinary sodium loss [6,7]. This effect of potassium on blood pressure is supported by a recent meta-analysis of published reports [8]. It has been calculated that an increase in potassium intakes from 60 to 80 mmol/d (2.3 to 3.1 g/d) could induce a fall of 4 mmHg systolic blood pressure and that this could possibly achieve a 25 % reduction in deaths related to hypertension [6].

Requirements

Adults

Renal and faecal losses each amount to about 10 mmol (390 mg)/d and there are also integumental losses. However, an intake of 40 mmol/d (1.6 g/d) is needed to avoid low plasma potassium concentrations and loss of total body potassium [9] and on this basis a lower intake of 40 mmol/d (1.6 g/d) is suggested.

An Average Requirement is not set. On the basis of the evidence cited earlier, a PRI of 80 mmol/d (3.1 g/d), which would also cover pregnancy and lactation, is proposed.

This intake could be reliably achieved by an appropriate intake of vegetables, fruit and derived juices, rather than by the use of potassium salts as substitutes for sodium chloride [10], because such measures if pursued indiscriminately could result in intakes at which toxicity might develop in individuals with undetected renal insufficiency and abnormal retention of potassium. Intakes above 450 mmol (17.5g)/d induce symptomatic hyperkalaemia in some otherwise normal individuals and can

thus be used as a threshold for acute toxicity, but such intakes are highly unlikely to arise from usual diets. However, for chronic intakes, intakes above 150 mmol (5.9 g)/d could be dangerous for individuals with undetected renal dysfunction [10]. Additionally, since there is no apparent benefit of exceeding an intake of 150 mmol (5.9 g)/d, this is proposed as an upper safe level of intake.

Children

The basal losses of children are not known reliably. Urinary excretion has been reported as 0.7-2.3 mmol (27-90 mg)/d. The amount needed for growth and lean tissue synthesis has been taken as 50 mmol (2 g)/kg. With these and other factors to allow for faecal losses (which are higher in children) and for integumental losses, PRIs for ages up to 17 were estimated factorially.

Summary

Adults	mg/d	mmol/d
Population Reference Intake	3100	80
Lowest Threshold Intake	1600	40

Population Reference Intakes of other groups

Age Group		*mg/d*	*mmol/d*
	6-11 m	800	20
	1-3 y	800	20
	4-6 y	1100	28
	7-10 y	2000	50
Males	11-14 y	3100	80
	15-17 y	3100	80
Females	11-14 y	3100	80
	15-17 y	3100	80
	Pregnancy	3100	80
	Lactation	3100	80

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