Potassium

Potassium		Women	Men	Children		
	g/d			2-5 y	6-9 y	10-13 y boys/girls
Recommended intake	RI	3.1	3.5	1.8	2.0	2.9/3.3
Lower intake level	LI	1.6	1.6			
Upper intake level	UL	-	-			

Introduction

The major proportion of the potassium in the body (98 %) is found in the cells and potassium is the quantitatively most important intracellular cation. Extracellular potassium, which constitutes the remaining 2 %, is important for regulating the membrane potential of the cells, and thereby for nerve and muscle function, blood pressure regulation etc. Potassium also participates in the acid-base balance. 1 mmol potassium is equivalent to 39 mg.

Dietary sources and intake

Important potassium sources in the Nordic diets are potatoes, fruit and berries, vegetables, and milk products. The average dietary intake ranges from 3.4 to 4.8 g/10 MJ (see Chapter XX Intake of Vitamins and minerals in Nordic countries).

Physiology and metabolism

The absorption of potassium is effective and about 90 % of the dietary potassium is normally absorbed from the gut. The potassium balance is primarily regulated by renal excretion in urine. A small proportion can be lost in sweat.

Requirement and recommended intake

Potassium deficiency can develop as a consequence of increasing losses from the gastrointestinal tract and kidneys, e.g. during prolonged diarrhoea or vomiting, and in connection with the use of laxatives or diuretics. Potassium deficiency due to low dietary intake alone is very uncommon, due to the widespread occurrence of potassium in foods. Treatment with diuretics without potassium compensation or potassium sparing diuretics can, however, lead to deficiency. Hyperaldosteronism, hereditary defects of renal salt transporters, such as

Bartter's syndrome and Gitelman's syndrome, and excessive consumption of licorice increase sodium retention and potassium excretion and may lead to hypokalemia. Symptoms of potassium deficiency are associated with disturbed cell membrane function and include muscle weakness and disturbances in heart function, which can lead to arrhythmia and heart seizure. Mental disturbances, e.g. depression and confusion, can also develop.

The losses of potassium via the gastrointestinal tract, urinary excretion and sweat comprise about 800 mg/d (20 mmol), but 1.6 g/d (40 mmol) is needed to avoid low plasma levels and loss of total body potassium in adults (1). The potassium intake may affect sodium balance and potassium intakes of 10-30 mmol/d may induce sodium retention and an increase in blood pressure, both in normotensive and hypertensive subjects (2-4). In the Intersalt study a 30-45 mmol increase in urinary potassium excretion was associated with a 2-3 mm Hg lower systolic blood pressure (5). An inverse relationship between blood pressure and potassium excretion and K/Na ratio in urine was also observed (6). A number of studies of both normotensive and hypertensive subjects indicate that an increased potassium intake as supplements can lower blood pressure and increase urinary sodium excretion (7-11). However, a clear dose-response effect was not observed, and not all showed a beneficial effect. The lack of clear dose-response observed in the studies could be due to factors such as differences in duration of studies, initial blood pressure, sodium intake, habitual diet, race and age.

Two meta-analyses of randomised trials with potassium supplementation showed a significant reduction of blood pressure (7, 8). In the study by Whelton et al. (7) a mean increased potassium excretion of about 60 mmol/d was associated with a mean 4.4 mm Hg decrease in systolic blood pressure and a 2.5 mm Hg decrease in diastolic blood pressure among hypertensives. Corresponding figures for normotensives were 1.8 and 1.0 mm Hg, respectively, although the effects were not significantly different between hypertensives and normotensives. The median duration of the trials was 5 weeks. The blood pressure lowering effect of potassium supplementation was greater in trials with a higher urinary sodium excretion, indicating the close interrelationship between sodium and potassium in this aspect. Using urinary excretion data for potassium, the average intake of potassium in the supplemented groups is estimated at 4.5-5 g/d. In a subsequent meta-analysis including randomised controlled trials with potassium supplementation with a duration of more than two weeks (8), an increased median potassium excretion of 44 mmol/d was associated with a 2.4 mm Hg decrease in systolic and 1.6 mm Hg in diastolic blood pressure. A third meta-analysis included only RCT studies with a duration of at least 8 weeks, participants over 18 years with raised blood pressure and with no changes in the medication during the follow-up. Meta-analysis of the five studies and 483 participants showed non-significant reductions in systolic and diastolic blood pressures. The authors concluded that the small number of participants in the two high quality trials, the short duration of follow-up and the unexplained heterogeneity between the trials made the evidence not conclusive. In a randomised controlled 6-week trial, a moderate potassium supplement of 24 mmol/d (900 mg/d) resulted in a decrease in systolic blood pressure of 7.6 mm Hg and in diastolic blood pressure of 6.5 mm Hg in healthy volunteers (12). This study indicates that a moderate increase in potassium intake may be sufficient to influence blood pressure. Most of the studies have used KCl supplements. A few studies with a limited number of subjects have investigated the effect of other potassium salts, e.g. citrate, but the results are conflicting with respect to any differential effects on blood pressure (13,14).

An inverse association between potassium intake and the risk of stroke has been shown in most cohort studies (15, 16, 17, 18). One outcome-trial of potassium-enriched salt on cardiovascular mortality has been published (19).-Five kitchens of a veteran's retired home in Taiwan were randomized into two groups, and 1981 veterans assigned to those kitchens were given either potassium-enriched salt (n=768) or regular salt (n=1213) for approximately 31 months. 103 CVD-related deaths were observed during the follow –up. A 17 % lower urinary sodium-to-creatinine ratio and a 76 % higher urinary potassium-to-creatinine ratio in the experimental group was associated with a significant reduction in CVD mortality (HR 0.59, 95 % CI: 0.37, 0.95).

In controlled intervention studies using diets designed to meet recommended levels of e.g. fat, fat quality and dietary fibre similar to those in NNR, the dietary potassium intakes (estimated from urinary potassium excretion) have been of the same magnitude, i.e. 3-4 g/d (20-22). In these studies blood pressure reductions were observed both with and without changes in sodium intake. However, sodium restriction still decreases blood pressure at the level of 3-4 g/d of potassium intake.

Reasoning behind the recommendation

The recommended intake of potassium in NNR 2004 was based on data on the effect of potassium on blood pressure. Several clinical trials and population surveys published thereafter support the finding that a diet rich in potassium alone, or in combination with calcium and magnesium, may have a favourable effect on blood pressure (5-12;20-23). The reference values are kept unchanged compared to NNR 2004, since there are no new scientific data to justify any major changes. The recommended intakes are set at 3.5 g/d (90 mmol) for men and 3.1 g/d (80 mmol) for women. The figure for women also includes pregnant and lactating women. It should be pointed out that potassium intakes somewhat over and above these values might have further beneficial effects. The reference values for children and adolescents are extrapolated from adult values based on needs for growth and adjusted for body weight.

The lower limit is estimated to 1.6 g/d (40 mmol) for adults.

Upper intake levels and toxicity

Potassium chloride has been associated with acute poisoning in humans. Case reports have described heart failure, cyanosis and cardiac arrest after ingestion of high doses of potassium chloride tablets. Gastrointestinal effects have also been described after chronic ingestion of potassium chloride in case studies and supplementation studies. This is characterised by abdominal pain, nausea and vomiting, diarrhoea, and ulceration of the oesophagus, stomach and duodenum and ileum. The occurrence and severity of the effects depend on a number of factors of which formulation of the preparation, dose and gut transit time seem to be the most important. Slow release, wax-coated KCl tablets appear to induce more lesions than microencapsulated tablets (24).

Dietary potassium has not been associated with any negative effects in healthy subjects. Prolonged high potassium intakes from diet and potassium-containing salt substitutes may, however, cause hyperkalaemia and affect heart function in subjects with renal insufficiency or impaired kidney function (24,25).

The available data are insufficient to set an upper level for dietary potassium. A British expert group proposed an intake of 3.7 g/d from supplements as an upper guidance level for adults. Supplemental intakes up to this level are generally not associated with overt adverse effects, but certain preparations may induce mild lesions of the gastrointestinal mucosa (23). It seems prudent to include potassium from potassium-containing mineral salt in this figure.

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