# Potassium[1]

Potassium is the main intracellular cation in the body and is principally involved in membrane potential and electrical excitation of both nerve and muscle cells and acid-base regulation. The compartmentalization of potassium is predominantly maintained by the (energy-dependent) cellular uptake of the element and the concomitant extrusion of sodium by the specific cell membrane–bound enzyme: sodium:potassium adenosine triphosphatase.

On average, the potassium content of an adult human is estimated to be ~40–50 mmol (1.6–2.0 g)/kg body weight, so a 70-kg adult would contain ~2800–3500 mmol of potassium (110–137 g). Intracellular potassium concentration is ~150 mmol/L (5.9 g/L), with the remainder present in the extracellular fluid. The extracellular fluid concentration of potassium is rather strictly maintained at a level of 3.5–5.5 mmol/L (137–215 mg/L). The total body potassium reflects lean tissue mass, ~90%–95% of this potassium is intracellular in the muscle and bone; thus, there is variation according to the degree of the body's muscularity (1).

Ingested potassium is mainly excreted in the urine (~80%–90%). The remaining 10%–20% is excreted in the feces and sweat. Most of the potassium that is filtered by the kidney glomerulus is reabsorbed throughout the kidney tubules. High extracellular potassium levels stimulate the release of aldosterone, which then promotes increased distal tubular secretion of potassium into the urine.

***Deficiencies:*** A potassium deficiency of nutritional origin is uncommon because potassium is widely available in a number of foods. However, due to the extensive consumption of processed foods, significant percentages of populations in Westernized regions may have discrete but relevant potassium deficiency. This could result in moderate chronic total body potassium depletion (2).

***Diet recommendations:*** Requirements for dietary potassium intake are difficult to determine precisely. They can be estimated from the amount of potassium that is accumulated with growth as well as from urinary and fecal excretion. Mandatory potassium loss from the kidneys is ~10–15 mmol/L of urine (that is, the kidneys can not make the urine potassium content more concentrated). Fecal excretion of potassium may represent homeostatic excretion of excessive intakes or losses incurred in maintaining sodium homeostasis. The amount of potassium required for both growth and the synthesis of lean tissue has been determined to be 50 mmol/L (2.0 g/kg/wt) in the United Kingdom. The US adequate intakes (AI), the UK lower reference nutrient intake (LRNI) and reference nutrient intake (RNI) are shown in **Table 1**. For adult populations (>18 y), the US AI is 4.7 g and the UK RNI is 3.5 g. The EU population reference intake (PRI) for potassium is 3.1 g/d and the lowest threshold intake (LTI) is 1.6 g/d.

**Table 1 Dietary recommendations for potassium[1]**

| Age groups | US AI | UK LRNI | RNI |
|---|---|---|---|
| | | *g/d* | |
| 0–6 mo | 0.4 | 0.4 | 0.8 |
| 7–12 mo | 0.7 | 0.45 | 0.7 |
| 1–3 y | 3.0 | 0.45 | 0.8 |
| 4–8 y | 3.8 | 0.61 | 1.1[2] |
| 9–13 y | 4.5 | 0.95[2] | 2.0[3] |
| 14–18 y | 4.7 | 1.6[3] | 3.1[4] |
| >18 y | 4.7 | 2.0[4] | 3.5[5] |

[1]AI, adequate intake; LRNI, lower reference nutrient intake; RNI, reference nutrient intake.
[2]4–6 y.
[3]7–10 y.
[4]11–14 y.
[5]15–18 y, >18 y.

***Food sources:*** Not surprisingly, because potassium is a main intracellular ion, potassium in the diet is derived from a wide variety of sources. Potatoes, soft drinks, meat and meat products, cereal and cereal products, and milk and milk products each provide >10% of the potassium intake in US and UK population groups (**Table 2**). Vegetables, fruits, and nuts provide >5% of potassium intake in the diet. For milk and milk products, about half (6%) comes from semiskimmed milk. Bananas are a rich source of potassium as are leafy green vegetables and root vegetables (**Table 3**). Dietary supplements make a negligible contribution to mean potassium intake in the US, UK, and European Union. Current dietary trends in Western societies show decreased consumption of fruit and vegetables and increased intake of sodium-rich foods, which results in reduced potassium intake and an increased sodium intake. When potassium is added as a preservative during processing, it is usually as potassium chloride, whereas it is usually present in fruit and vegetables as potassium citrate. Approximately 85% of potassium is absorbed, and it is generally considered that the best way to increase potassium intake is to consume more fruit and vegetables.

***Clinical use of potassium:*** A high potassium intake has been shown to have protective effects against a number of pathological states that affect the cardiovascular system, kidneys, and bones. Increased potassium intake lowers blood pressure, and this effect has been consistent in both hypertensive and normotensive populations. Evidence suggests that potassium may be effective in reducing stroke and could help prevent chronic kidney damage. Increasing daily potassium alkali intake (found in fruits and vegetables) helps to reduce calcium excretion in the urine and thus may have a positive effect on bone health. There is also growing evidence of a strong link between increasing potassium intake and favorable effects on muscle function, overall muscle health, and potentially prevention of falls.

***Toxicity:*** Healthy individuals ingesting the usual dietary intakes of potassium are unlikely to have problems with potassium


 Adv. Nutr. 3: 820–821, 2012; doi:10.3945/an.112.003012.

**Table 2 Percentage of contribution of food types to potassium intake**

| Food type | % Contribution |
|---|---|
| Potatoes and savory snacks | 18 |
| Drinks[1] | 17 |
| Meat and meat products | 16 |
| Cereal and cereal products | 14 |
| Milk and milk products | 13 |
| Vegetables | 9 |
| Fruit and nuts | 6 |
| Fish and fish dishes | 3 |
| Eggs and egg dishes | 1 |
| Sugars, preserves, and confectionery | 1 |
| Miscellaneous | 2 |

[1]Fruit juice, beer, coffee, and tea.

**Table 3 Potassium content of common foods**

| Food | Potassium (mg/100 g) |
|---|---|
| Dried apricots | 1900 |
| Dark chocolate | 830 |
| Dates | 696 |
| Salmon | 628 |
| White beans | 561 |
| Spinach | 558 |
| Baked potato (with skin) | 535 |
| Cod | 516 |
| Avocado | 485 |
| Sweet potato | 475 |
| Pork chop | 449 |
| Brussel sprouts | 389 |
| Bananas | 358 |
| Low-fat yogurt | 234 |
| Orange juice | 200 |
| Milk (semiskimmed) | 154 |

toxicity. Potassium intake in Western societies is normally in the range of 1.6 to 5.9 g/d. Extremely high intakes of potassium >17.6 g/d (usually only obtained with potassium supplements) have been associated with symptomatic hyperkalemia. High blood concentrations of potassium can cause muscle weakness and cardiac arrhythmias. Chronic damage to the kidneys and some kinds of medications (e.g., those used for blood pressure control that block the renin-angiotensin system) can cause high potassium levels if dietary potassium intake is not controlled.

***Recent research:*** Dietary potassium intake has been shown to significantly lower blood pressure. Studies have demonstrated that this occurs in a dose-responsive manner in hypertensive and nonhypertensive patients, with evidence coming from a number of important observational studies and clinical trials. Several key meta-analyses also support this finding. Data also show that increasing potassium intake and reducing sodium intake are additive in lowering blood pressure. Potassium-induced reductions in blood pressure significantly lower the incidence of a cerebrovascular accident (stroke), coronary heart disease, myocardial infarction, and other cardiovascular events. There are also data to show that potassium reduces the risk of cerebrovascular accidents independent of blood pressure.

Increasing potassium intake has also been shown to conclusively reduce urinary calcium excretion, thus creating a positive calcium balance. In the longer term, this is likely to have very beneficial effects on bone mass and concomitant risk of osteoporosis. Clinical trials support these findings in the short term (3–6 mo), with a number of important randomized, controlled trials showing a reduction in bone resorption markers in those individuals on potassium citrate/potassium bicarbonate supplementation/high dietary potassium intake. Further long-term bone studies (>12 mo) are urgently required. The DASH (Dietary Approaches to Stopping Hypertension) study, with a high dietary potassium intake (and high calcium intake) reduced blood pressure, reduced urinary calcium excretion, and lowered bone resorption in the short term and a longer term DASH/osteoporosis reduction study is now urgently required.

Increasing potassium intake has also been shown to reduce the risk of kidney stones. Interestingly, studies in hypertensive rats show that high potassium intake prevents renal vascular, glomerular, and tubular damage independent of blood pressure. Currently, in humans there is no direct evidence that potassium protects against renal arteriolar/tubular lesions that specifically occurs in kidney disease or hypertension. Further research is needed, given that particular ethnic population groups often have low potassium intake and a high prevalence of hypertensive renal failure.

Finally, the transition toward the Westernized diet has led to populations consuming substantially less potassium compared with dietary intakes in preagricultural times, when humans consumed a diet high in potassium (>200 mmol/d). With the increase in consumption of processed foods and the concomitant reductions (often dramatic) in fruits and vegetables, potassium intakes are <50 nmol/L in large numbers of populations. These reductions are, in many populations, also mirrored with high sodium intakes. Further research is urgently required to determine how these dietary patterns affect key health outcomes and how such food intake trends can be reversed.

Susan A. Lanham-New* and Helen Lambert
*Department of Nutritional Sciences, Faculty of Health and Medical Sciences, University of Surrey, Guildford, Surrey, England

Lynda Frassetto
Department of Medicine and General Clinical Research Center, University of California, San Francisco, CA

[1]Author disclosures: S. Lanham-New, L. Frassetto, and H. Lambert, no conflicts of interest.

*To whom correspondence should be addressed. E-mail: s.lanham-new@surrey.ac.uk.

## Literature Cited

1. Institute of Medicine. Dietary reference intakes: the essential guide to nutrient requirements 2006. New York, USA.
2. Rose BD. Potassium homeostasis. In: Clinical physiology of acid-base and electrolyte disorders. 4th ed. New York: McGraw-Hill; 1994. p. 346–76.