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Nutrient Reference Values for Australia and New Zealand
Including Recommended Dietary Intakes

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SODIUM

BACKGROUND

Sodium is a cation needed to maintain extracellular volume and serum osmolality. Approximately 95% of the total sodium content of the body is found in extracellular fluid, being maintained outside the cell via the sodium/potassium-ATPase pump. Sodium is also important for maintaining the membrane potential of cells and for active transport of molecules across cell membranes. Absorption of sodium and chloride occurs primarily in the small intestine and is approximately 98% efficient across a wide range of intakes.

Sodium is found in most foods as sodium chloride, generally known as 'salt'. It is also present in the diet as sodium bicarbonate and as monosodium glutamate in processed foods. Sodium is also present in other food additives such as sodium phosphate, sodium carbonate and sodium benzoate. Sodium chloride, however, accounts for approximately 90% of the total sodium excreted in countries like Australia and New Zealand (Fregly 1984, Mattes & Donnelly 1991).

In industrialised countries, the majority of ingested sodium chloride is excreted in the urine, provided that sweating is not excessive (Holbrook et al 1984, Pitts 1974). Absorbed sodium and chloride remain in the extracellular compartments that include plasma and interstitial fluid. There are various systems and hormones that influence sodium balance, including the renin-angiotensin-aldosterone hormone system, the sympathetic nervous system, atrial natriuretic peptide, the kallikrein-kinin system, various intrarenal mechanisms, and other factors that regulate renal and medullary blood flow. In sodium and fluid balance, with minimal sweat losses, the amount of sodium excreted in urine roughly equals intake.

The Intersalt Cooperative Research Group (1988) found that the rate of sodium excretion ranges from less than 0.2 mmol of sodium/day in the Yanomamo Indians of Brazil to 242 mmol/day in Tianjin in China (Intersalt Cooperative Research Group 1988). Estimated intakes in Australia are about 150 mmol/day (Beard et al 1997, Notowidjojo & Truswell 1993). An almost identical figure has been found in New Zealand (Thomson & Colls 1998).

There many healthy populations with estimated intakes of less than 40 mmol/day (Intersalt Cooperative Research Group 1988). Survival at extremely low levels such as that of the Yanomamo reflects the ability to conserve sodium by reducing urine and sweat losses. With maximal adaptation, the smallest amount of sodium needed to replace losses is estimated to be no more than 0.18 g/day (8 mmol/day). However, a diet providing this level of sodium intake is unlikely to meet other dietary requirements in countries such as Australia and New Zealand.

Physical activity can potentially affect sodium chloride balance, mostly from increased losses in sweat. People who regularly undertake strenuous activity in the heat can lose substantial amounts of sodium. Loss of sodium in sweat is dependent on overall diet, sodium intake, sweating rate, hydration status and degree of acclimatisation to the heat (Allan & Wilson 1971, Allsopp et al 1998, Brouns 1991). Acclimatisation to the heat decreases the amount of sodium lost in sweat (Sawka & Montain 2002). Exposure to heat without exercise also alters the sodium concentration of sweat.

Other factors that can affect sodium needs include the intake of potassium (Liddle et al 1953) and calcium (Breslau et al 1982, Castenmiller et al 1985, McCarron et al 1981). Administration of potassium salts has been shown to increase urinary sodium excretion and a substantial body of evidence has documented that higher intakes of sodium result in increased urinary excretion of calcium.

The major adverse effect of increased sodium chloride intake is elevated blood pressure, a risk factor for cardiovascular and renal diseases. Blood pressure increases progressively in a dose-dependent relationship with sodium chloride excretion across the range seen in populations around the world. There has been a number of meta-analyses of the effect of reduction of sodium on diastolic and systolic blood pressure (Cutler et al 1997, Graudal et al 1998, Midgley et al 1996).

The strongest dose-response evidence comes from clinical trials that specifically examined the effects of at least three levels of sodium intake on blood pressure (Bruun et al 1990, Ferri et al 1996, Fuchs et al 1987, Johnson et al 2001, Kirkendall et al 1976, Luft et al 1979, MacGregor et al 1989, Sacks et al 2001, Sullivan et al 1980). The range of sodium intakes in these studies was 10 mmol/day to 1,500 mmol/day. Several trials included sodium intake levels close to 65 mmol/day and 100 mmol/day.

There is a well-recognised heterogeneity in the blood pressure response to changes in sodium chloride intake. People with hypertension, diabetes and chronic kidney disease and greater age tend to be more sensitive to the blood pressure raising effects of sodium chloride intake. Overweight also appears to increase susceptibility as demonstrated by He et al (1999) in a study of 14,407 participants with a 19-year follow up. In this study, relative risks for stroke and cardiovascular mortality were 1.89 and 1.61, respectively, for an increase in sodium intake of 100 mmol. However, the excess mortality was seen in overweight but not normal weight adults. Given the increasing level of overweight in the community, this is of particular importance. Genetic factors also influence the blood pressure response to sodium chloride. Sodium sensitivity is modifiable with the rise in blood pressure, being less on a diet high in potassium. In non-hypertensive individuals, a reduced sodium intake can decrease the risk of developing hypertension.

Indicators that have been used for assessing the need for sodium include sodium balance, serum or plasma sodium concentration, plasma renin activity, elevation in blood pressure, blood lipid concentrations and insulin resistance.

1 mmol sodium = 23 mg sodium 1 gram of sodium chloride (salt) contains 390 mg (17 mmol) of sodium

RECOMMENDATIONS BY LIFE STAGE AND GENDER

Infants	**AI**		**Sodium**
0–6 months	**120 mg/day**	**(5.2 mmol)**	
7–12 months	**170 mg/day**	**(7.4 mmol)**	

***Rationale*:** The AI for 0–6 months was calculated by multiplying together the average intake of breast milk (0.78 L/day) and the average concentration of sodium of 160 mg/L from the studies of Dewey & Lonnerdal (1983), Gross et al (1980), Keenan et al (1982), Lemons et al (1982), Morriss et al (1986) and Picciano et al (1981). The AI for 7–12 months was extrapolated from that for 0–6 months from a consideration of metabolic body weights and relative energy requirements.

Children & adolescents	**AI**		**Sodium**
All			
1–3 yr	**200–400 mg/day**	**(9–17 mmol)**	
4–8 yr	**300–600 mg/day**	**(13–26 mmol)**	
9–13 yr	**400–800 mg/day**	**(17–34 mmol)**	
14–18 yr	**460–920 mg/day**	**(20–40 mmol)**	

***Rationale*:** There are not enough dose-response data to set an EAR for children and adolescents, so AIs have been set. There is no reason to expect that the sodium requirement of children ages 1 to 18 years would be fundamentally different from that of adults, given that maturation of kidneys is similar in normal children by 12 months of age (Seikaly & Arant 1992). The AIs for children and adolescents were derived from adult AIs based on relative energy intake.

Adults	**AI**		**Sodium**
Men	**460-920 mg/day**	**(20-40 mmol)**	
Women	**460-920 mg/day**	**(20-40 mmol)**	

***Rationale*:** As there are insufficient data from dose-response trials, an EAR could not be established, and thus a RDI could not be derived. An AI for adults for sodium was set at 460–920 mg/day (20–40 mmol/day) to ensure that basic requirements are met and to allow for adequate intakes of other nutrients. This AI may not apply to highly active individuals, such as endurance athletes or those undertaking highly physical work in hot conditions, who lose large amounts of sweat on a daily basis.

Pregnancy	**AI**		**Sodium**
14–18 yr	**460-920 mg/day**	**(20-40 mmol)**	
19–30 yr	**460-920 mg/day**	**(20-40 mmol)**	
31–50 yr	**460-920 mg/day**	**(20-40 mmol)**	

***Rationale*:** During pregnancy there is a small increase in extracellular fluid, but as the AI for women was set generously, there should be no additional requirement in pregnancy.

Lactation	**AI**		**Sodium**
14–18 yr	**460-920 mg/day**	**(20-40 mmol)**	
19–30 yr	**460-920 mg/day**	**(20-40 mmol)**	
31–50 yr	**460-920 mg/day**	**(20-40 mmol)**	

***Rationale*:** In lactation, there is a small increase in maternal extracellular fluids and some sodium is excreted in breast milk. However, these additional requirements are well within the additional margin added to the adult AI so there are no additional requirements.

UPPER LEVEL OF INTAKE - SODIUM

Infants		
0–12 months	**Not possible to establish. Source of intake should be through breast milk, formula and food only.**	
Children		
1–3 yr	**1,000 mg/day**	**(43 mmol)**
4–8 yr	**1,400 mg/day**	**(60 mmol)**
9–13 yr	**2,000 mg/day**	**(86 mmol)**
14–18 yr	**2,300 mg/day**	**(100 mmol)**
Adults 19+ yr		
Men	**2,300 mg/day**	**(100 mmol)**
Women	**2,300 mg/day**	**(100 mmol)**
Lactation		
All ages	**2,300 mg/day**	**(100 mmol)**

***Rationale*:** The adverse effects of higher levels of sodium intake on blood pressure provide the scientific rationale for setting the UL. Because the relationship between sodium intake and blood pressure is progressive and continuous, it is difficult to set a UL precisely. To complicate the analysis, other environmental factors (weight, exercise, potassium intake, overall dietary pattern and alcohol intake) and genetic factors also affect blood pressure. However, there is evidence of a population intake threshold for increased prevalence of hypertension.

For adults, a UL of 2,300 mg/day (100 mmol/day) is set on the basis of population studies showing low levels of hypertension (less than 2%) and no other observed adverse effects in communities with intakes below this level. The UL was also based on experimental studies such as the DASH-sodium trial that showed an additional systolic blood pressure reduction of 4.6 mmHg ($p < 0.001$) at intakes of 1,500 mg/day (65 mmol/day) compared to 2,500 mg/day (107 mmol/day) in people on the control diet. In this study, decreasing sodium intake by approximately 920 mg/day (40 mmol/day) caused a greater lowering of blood pressure when the starting sodium intake was at the intermediate level than when it was at a higher intake similar to the Australian/New Zealand average of about 6g/day. A NOAEL of 2,300 mg/day (100 mmol) was therefore adopted. However, this NOAEL was adopted in recognition of the fact that the considerable number of older and overweight people in the Australian and New Zealand population who have pre-existing hypertension will derive additional benefit from lowering intakes to 1,600mg/day (70 mmol). This level of 1,600 mg has thus been set as a Suggested Dietary Target for chronic disease prevention in line with WHO recommendations (WHO 2000) (see also 'Chronic disease' section).

A UF of 1 was applied as, by definition, there is no convincing evidence of harm in the general population at levels of intake of 100 mmol or less. There are no data to suggest increased susceptibility in pregnancy or lactation, so the UL is set at the same level as for adult women.

For infants, a UL could not be established because of insufficient data documenting the adverse effects of chronic over-consumption of sodium in this age group. The UL for children was extrapolated from the adult UL on an energy intake basis as numerous observational studies have documented that blood pressure tracks with age from childhood into the adult years (Bao et al 1995, Dekkers et al 2002, Gillman et al 1993, Van Lenthe et al 1994).

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