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

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IODINE

BACKGROUND

Iodine was one of the first trace elements to be identified as essential. In the 1920s it was shown to be an integral component of the thyroid hormone, thyroxine (T_4), required for normal growth and metabolism. Soon after, it was recognised as a component of 3,5,3'-tri-iodothyronine (T_3), a key regulator of important cell processes. The thyroid hormones are required for normal growth and development of tissues such as the central nervous system and have a broader role in maturation of the body as a whole. They are important for energy production and oxygen consumption in cells thereby helping to maintain the body's metabolic rate. Iodine occurs in tissues in both organic and organically bound forms. The iodine content of the adult body is approximately 15–20 mg, of which 70–80% is in the thyroid gland – which concentrates iodine (Freake 2000) – and the rest is in blood.

Once iodine is absorbed in the form of iodide and reaches the circulation, it is concentrated in the thyroid gland where it is converted to iodine and combined with tyrosine residues of thyroglobulin. The iodinated tyrosines are removed from the thyroglobulin by proteolytic enzymes and T_4 is released into the circulation (Kidd et al 1974). T_4 is inert until deiodinated either to T_3 (or reverse T_3, an inactive form of T_4). Deiodination requires selenocysteine as the active form of selenium in the iodothyronine deiodinases (Arthur & Beckett 1999). Regulation of thyroid hormone synthesis, release and action is complex. It involves the thyroid, pituitary, brain and peripheral tissues. Excess inorganic iodine is readily excreted in urine, with smaller amounts in faeces and sweat (Lamberg 1993).

Iodine in foods is in the inorganic iodide form and is easily absorbed in the stomach and upper small intestine (Sumar & Ismail 1997) as is supplemental iodine. Thus the amount of bioavailable iodine depends on the amount consumed rather than the chemical form or composition of the diet (Fairweather-Tait & Hurrell 1996). However, the utilisation of absorbed iodine is influenced by goitrogens. Goitrogens such as sulphur-containing thionamides found in brassica vegetables such as cabbage, broccoli and brussel sprouts can interfere with the synthesis of the thyroid hormones. They impair the binding of iodine to thyroglobulin and prevent oxidation of iodide by thyroid iodide peroxidase (Gaitan 1980). Foods containing goitrogenic cyanoglucosides such as sweet potato and maize release thiocyanate that competes with iodide, blocking its uptake by the thyroid (Gaitan 1980, Lamberg 1993).

The iodine content of most foods is low and can be affected by soil, irrigation and fertilisers. Losses can occur in cooking. Most soils in New Zealand are low in iodine resulting in low concentrations in locally grown foods. The major food sources are of marine origin. Processing aids such as calcium iodate, potassium iodate, potassium iodide and cuprous iodide act to increase the content of iodine in certain foods. Iodophores used by the dairy industry, which opportunistically enter the food supply, were the major, if not the prime, contributors to intake of iodine in Australia and New Zealand in the 1960s. However, controls introduced in the early 1970s saw changes in practices leading to reduced iodine in milk. As the use of iodised salt has also declined since that time, intakes of iodine have fallen in both Australia and New Zealand (Eastman 1999, Gunton et al 1999, Hynes et al 2004, Skeaff et al 2002, 2005, Thomson 2002, 2004).

Iodine deficiency results in a range of conditions collectively termed 'iodine deficiency disorders' (Hetzel et al 1990, Thomson 2002). In severe deficiency, these include major effects on the fetus, such as abortion or stillbirth, congenital anomalies, increased perinatal and infant mortality, neurological cretinism or mental deficiency with deaf mutism, spastic diplegia and squint, myxoedematous cretinism and dwarfism and psychomotor effects. In neonatal life, childhood or adulthood, iodine deficiency can lead to goitre or hypothyroidism as well as impaired mental and physical development.

Several indicators are used to assess iodine requirements, including urinary iodide excretion, thyroid hormones in plasma or serum, assessment of thyroid size and goitre rate, radioactive iodine uptake, balance studies and epidemiologic, population studies. Thyroid iodine accumulation and turnover is generally considered to be the best measure.

1 mmol iodine = 127 mg iodine

RECOMMENDATIONS BY LIFE STAGE AND GENDER

Infants	**AI**	**Iodine**
0–6 months	**90 µg/day**	
7–12 months	**110 µg/day**	

***Rationale*:** The AI for 0–6 months was calculated by multiplying the average intake of breast milk (0.78 L/day) by the average concentration of iodine in breast milk (115 µg/L), and rounding. The figure used for breast milk was that recommended by FAO:WHO (2001) which is also consistent with the study of Johnson et al (1990) in New Zealand. The AI for 7–12 months was extrapolated from that of younger infants using a metabolic weight ratio.

Children & adolescents	**EAR**	**RDI**	**Iodine**
All			
1–3 yr	**65 µg/day**	**90 µg/day**	
4–8 yr	**65 µg/day**	**90 µg/day**	
Boys			
9–13 yr	**75 µg/day**	**120 µg/day**	
14–18 yr	**95 µg/day**	**150 µg/day**	
Girls			
9–13 yr	**75 µg/day**	**120 µg/day**	
14–18 yr	**95 µg/day**	**150 µg/day**	

***Rationale*:** The EAR for children was based on balance studies for the age groups 1–3 years, 4–8 years and 14–18 years (Ingenbleek & Malvaux 1974, Malvaux et a 1969) and by extrapolation from adults using metabolic body weight ratios for 9–13 year olds. The RDI was set assuming a CV of 20% for the EAR from studies in adults (FNB:IOM 2001), and rounded.

Adults	**EAR**	**RDI**	**Iodine**
Men			
19–30 yr	**100 µg/day**	**150 µg/day**	
31–50 yr	**100 µg/day**	**150 µg/day**	
51–70 yr	**100 µg/day**	**150 µg/day**	
>70 yr	**100 µg/day**	**150 µg/day**	
Women			
19–30 yr	**100 µg/day**	**150 µg/day**	
31–50 yr	**100 µg/day**	**150 µg/day**	
51–70 yr	**100 µg/day**	**150 µg/day**	
>70 yr	**100 µg/day**	**150 µg/day**	

***Rationale*:** The EARs for adults were based on iodine balance studies indicating that iodine balance is achieved at intakes over 100 µg/day but not below 40 µg/day. From these data, particularly the iodine accumulation and turnover studies, and a New Zealand study in adults relating urinary iodide to thyroid volume (Thomson et al 2001) that indicated physiological requirements of 85–100 µg/day, a value of 100 µg/day was adopted for the EAR. The RDI was set assuming a CV of 20% for the EAR (FNB:IOM 2001), and rounded up to reflect the possible influence of natural goitrogens.

Pregnancy	**EAR**	**RDI**	**Iodine**
14–18 yr	**160 µg/day**	**220 µg/day**	
19–30 yr	**160 µg/day**	**220 µg/day**	
31–50 yr	**160 µg/day**	**220 µg/day**	

***Rationale*:** The EAR for pregnancy was based on data relating to the thyroid content of newborns, iodine balance studies and iodine supplementation studies in pregnancy (FNB:IOM 2001). The RDI was set assuming a CV of 20% for the EAR.

Lactation	**EAR**	**RDI**	**Iodine**
14–18 yr	**190 µg/day**	**270 µg/day**	
19–30 yr	**190 µg/day**	**270 µg/day**	
31–50 yr	**190 µg/day**	**270 µg/day**	

***Rationale*:** The EAR for lactation was based on the adult female needs (100 µg/day) and the replacement needs for iodine secreted in breast milk (90 µg/day). The RDI was set assuming a CV of 20% for the EAR.

UPPER LEVEL OF INTAKE - IODINE

Infants	
0–12 months	**Not possible to establish. Source of intake should be milk, formula and food only**
Children and adolescents	
1–3 yr	**200 µg/day**
4–8 yr	**300 µg/day**
9–13 yr	**600 µg/day**
14–18 yr	**900 µg/day**
Adults 19+ yr	
Men	**1,100 µg/day**
Women	**1,100 µg/day**
Pregnancy	
14–18 yr	**900 µg/day**
19–50 yr	**1,100 µg/day**
Lactation	
14–18 yr	**900 µg/day**
19–50 yr	**1,100 µg/day**

***Rationale*:** The first effect seen in iodine excess is challenged thyroid function by elevated TSH concentrations. This is the critical adverse effect (FNB:IOM 2001). Two studies of TSH concentrations after supplemental iodine showed increased TSH at 1,800 µg/day and 1,700 µg/day (Gardner et al 1988, Paul et al 1988) indicating a LOAEL of 1,700 µg/day. A UF of 1.5 is applied to derive a NOAEL that is the basis of the UL for adults. As there is little evidence for other age groups, ULs for children and adolescents were extrapolated from the adult recommendation on a metabolic body weight basis. The adult UL was also used for pregnancy and lactation as there was no evidence of increased sensitivity associated with these.

***Note*:** Individuals with thyroid disorders or a long history of iodine deficiency may still respond adversely at levels of intake below the UL.

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