Attachment A – Draft recommendations

Iodine

Background

Iodine is a mineral that is found in soil and ocean waters and is an essential nutrient required for synthesis of thyroid hormones such as thyroxine (T4) and 3,5,3′-tri-iodothyronine (T3). These hormones are involved in regulating cellular processes including metabolism, cellular oxidation and thermoregulation. They also play an essential role in early development, growth and maturation of organs including the brain, muscles, heart, pituitary gland, kidneys reproductive system and bones.

Iodine-rich foods include seafoods such as fish, shellfish, or seaweed (Barkley and Thompson, 1960, Blikra et al 2022) eggs, milk and their products, and iodised salt (EFSA 2014). The levels of iodine in animal products such as meat, dairy and eggs varies based on the iodine content of feed. The level of iodine in cereal and grain foods varies depending on the iodine content of soil in which the food is grown. Low soil iodine levels are typical for New Zealand and in some parts of Australia such as Tasmania (AIHW, 2016). In 2009 Australia and New Zealand introduced mandatory fortification requirements for the addition of iodine (via iodised salt) to commercial bread, to address iodine deficiency in the population. Consequently, consumption of commercial bread and bread-products is also a main contributor to dietary iodine intake.

Vegans and vegetarians – and consumers of plant-based milk alternatives – have lower intakes of iodine-containing foods such as meat, seafood, dairy and eggs, and may be at greater risk of deficiency (Eveleigh, 2023, Dineva et al 2021, Lundquist et al 2024). Given the role of mandatory iodine fortification of bread in preventing iodine deficiency in Australia and New Zealand, the impact of vegan/vegetarian or dairy-free diets on status may be minimised for individuals who consume bread. Individuals with low commercial bread intake may be at greater risk of deficiency in Australia and New Zealand.

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). Absorption of iodine from food is estimated at 90–92% under normal conditions (Thomson et al 1996, Jahreis et al 2001, Aquaron et al 2002).

Iodine ingested from food is reduced to iodide in the gut, with absorption occurring primarily in the small intestine. Once in the thyroid gland, iodine is incorporated into thyroglobulin (Tg) and this can provide up to 3 months of thyroid hormone to sustain hormone synthesis during periods of low iodine intake (Vanderpas, 2003).

Thyroid hormone synthesis is a complex process involving the thyroid, pituitary, brain and peripheral tissues. Iodinated tyrosines are removed from Tg via proteolytic enzymes, releasing T4 into circulation (Kidd et al 1974). Deiodination of T4 then produces T3 or reverse T3 – an inactive

form. Excess iodine is excreted through urine, with small amounts also excreted via faeces and sweat (Lamberg 1993).

Some compounds – known as goitrogens – impair iodine uptake and increase an individual's risk of deficiency. Identified goitrogens include tobacco and electronic cigarettes (Colzani et al 1998, Knudsen et al 2002, Shields et al 2008, Flieger et al. 2019), along with foods such as cassava, millet, maise and cruciferous vegetables (Gibson, 1991). However, the risk of deficiency from consumption of these foods may be negligible where iodine intake is adequate and a varied diet is consumed (Zimmermann et al. 2008). Absorption can also be affected by deficiencies in other micronutrients including selenium, iron and zinc (Yang et al 1997, Kohrle 1999, Thomson 2004, Zimmermann et al 2000, O'Kane et al 2018).

Iodine deficiency is associated with a range of adverse health and developmental outcomes, collectively termed 'iodine deficiency disorders' (Hetzel 1983) including thyroid dysfunction, thyroid disease, and adverse child neurocognitive development. Iodine deficiency disorders affect individuals of all ages, with the range effects varying depending on the developmental stage at which exposure occurs and severity of deficiency. Severe iodine deficiency during pregnancy is associated with adverse birth outcomes, such as miscarriage and stillbirth. Severe deficiency during pregnancy and lactation is also associated with adverse child developmental outcomes including intellectual impairment, hearing loss, and psychomotor disorders (EFSA 2014). Iodine deficiency is the primary cause of preventable child cognitive impairment world-wide (Ristić-Medić 2013). Although the relationship between severe iodine deficiency during pregnancy and global impairments in child neurocognitive development is well-established, the evidence for mild iodine deficiency is less certain. Robust supportive evidence of consistent adverse neurocognitive effects associated with mild-to-moderate iodine deficiency are lacking, with the evidence base limited by inconsistent findings and significant heterogeneity (Monaghan 2021).

Due to the critical role of iodine in early neurocognitive development, the fetus and infant are particularly sensitive to the effects of maternal deficiency during pregnancy or lactation. Individuals who are planning pregnancy (and their treating health care providers) should seek to understand current recommendations about iodine supplementation during pre-conception – and throughout pregnancy and lactation – to ensure optimum child development.

Chronic iodine deficiency can also result in progressive thyroid gland enlargement (goitre) as a compensatory measure to increase thyroidal iodine capture and support thyroid hormone production (UK SACN 2014). This may progress to hyperthyroidism and can also increase the risk of thyroid cancer (EFSA 2014). Hypothyroidism can also occur due to thyroid hormone dysfunction, affecting metabolic rate, body weight and cognitive function.

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. Estimated requirements vary substantially across studies, which may reflect individual variability in requirements, and homeostatic mechanisms that vary thyroidal iodine capture – and potentially iodine bioavailability – depending on individual intake and status. This poses difficulties with interpreting findings for estimating requirements.

Other international bodies have concluded that evidence from thyroid accumulation and balance studies are insufficient to derive an estimated average requirement (EFSA 2014, Blomhoff et al 2023). However, several evidence sources support establishment of an EAR (Estimated Average

Requirement) in the range of 90–110 µg/day for adults. Establishment of an EAR facilitates important population health monitoring, including for evaluating the adequacy of iodine in the food supply following mandatory fortification. Consequently, the EAR and RDI (Recommended Dietary Intake) were maintained, whilst acknowledging that there is some uncertainty in the underlying evidence base.

Excess iodine can result in thyroid abnormalities including hypothyroidism and hyperthyroidism. Chronic exposure to excess iodine has also been associated with autoimmune thyroid disease (Wang et al 2019) and thyroid cancer (Lee et al 2017). Excessive habitual iodine intake may also have adverse effects on child intellectual development (Li et al 2022).

In healthy adults, high intakes of iodine are generally well tolerated owing to intrinsic regulatory mechanisms within the thyroid. However, some individuals are particularly sensitive to the effects of iodine excess and they may respond adversely at levels of intake below the UL (Upper Level of Intake). This includes older adults, people with a long history of iodine deficiency, and those with pre-existing thyroid dysfunction or disorders. Frequent consumption of seaweed – or seaweed containing products – may increase a person's risk of exceeding the UL, due to the high iodine content in some seaweed varieties (EFSA 2023; Blikra et al 2022; Blikra et al 2024).

The first effect seen in iodine excess is challenged thyroid function. Elevated thyroid-stimulating hormone (TSH) concentration is an early biomarker of thyroid dysfunction and serves as a critical endpoint, in the absence of more robust biomarkers. Although these measures have limitations as endpoints of toxicity, they remain the most reliable biomarkers upon which to base an UL.

1 mmol iodine = 127 mg iodine

Recommendations by life stage and sex

Infants

Table 1 provides recommendations for iodine intake in infants at different stages. The AIs (Adequate Intake) for infants were not reviewed in the 2025/26 update.

Table 1. Recommendations for iodine intake in infants

Age	AI
0 - 6 months	90 µg/day
7 - 12 months	110 µg/day

Rationale: The AI for 0 to 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 to 12 months was extrapolated from that of younger infants using a metabolic weight ratio.

Children and adolescents

Table 2 outlines iodine intake recommendations for children and adolescents at different age groups and sexes based on extrapolation from adult values.

Table 2. **Iodine intake recommendations for children and adolescents**

Age	EAR	RDI
All		
1 to under 4 years	65 µg/day	90 µg/day
4 to under 9 years	65 µg/day	90 µg/day
Males		
9 to under 14 years	75 µg/day	120 µg/day
14 to under 18 years	95 µg/day	150 µg/day
Females		
9 to under 14 years	75 µg/day	120 µg/day
14 to under 18 years	95 µg/day	150 µg/day

Rationale: The EARs for children was derived by extrapolation from adults using the metabolic body weight ratio approach, based on the reference weights in the NHMRC Methodological framework (NHMRC, 2025). The RDI was set assuming a CV (coefficient of variation) of 20% (FNB:IOM 2001) and rounded.

Alternative age groupings – preschool, primary school and adolescents

For reporting usual intake against the national nutrition survey results, ages have also been grouped to provide levels for pre-school (2 to under 5 years), primary school age (5 to under 12 years) and adolescents (12 to under 18 years) in table 3. The derivation of the values uses the same methodology as table 2 and is based on the same reference adult value. If you are not comparing to survey intake data or school age groups, you should use table 2.

Table 3. **Alternative age groups for preschool, primary school and adolescents – iodine intake recommendations**

Age	EAR	RDI
All		
12 to under 24 months	65 µg/day	90 µg/day
2 to under 5 years (preschool)	65 µg/day	90 µg/day
Males		
5 to under 12 years (primary school)	70 µg/day	110 µg/day
12 to under 18 years (adolescents)	90 µg/day	140 µg/day
Females		

Age	EAR	RDI
5 to under 12 years (primary school)	70 µg/day	110 µg/day
12 to under 18 years (adolescents)	90 µg/day	140 µg/day

Adults

Table 4. **Iodine intake recommendations for adults at various life stages and sexes.**

Age	EAR	RDI
Males		
18 to under 30 years	100 µg/day	150 µg/day
30 to under 50 years	100 µg/day	150 µg/day
50 to under 65 years	100 µg/day	150 µg/day
65 to under 75 years	100 µg/day	150 µg/day
75 years and over	100 µg/day	150 µg/day
Females		
18 to under 30 years	100 µg/day	150 µg/day
30 to under 50 years	100 µg/day	150 µg/day
50 to under 65 years	100 µg/day	150 µg/day
65 to under 75 years	100 µg/day	150 µg/day
75 years and over	100 µg/day	150 µg/day

Rationale: The EARs for adults were based on evidence suggesting average daily thyroid iodine accumulation and turnover between 91 and 97 µg/day (Fisher and Oddie 1969a; Fisher and Oddie 1969b), and balance studies suggesting that neutral balance is achieved at intakes between 90 around 110 µg/day (Tan 2019). From these data, 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

Table 5. Iodine intake recommendations for during pregnancy

Age	EAR	RDI
Pregnancy (all ages)	160 µg/day	220 µg/day

Rationale: The EAR during pregnancy was calculated based on the EAR for adults, and factoring in additional requirements during pregnancy.

Daily fetal thyroid iodine uptake was estimated at 75 µg/day based on 100% daily turnover of iodine in the newborn thyroid, and an estimated thyroid content of 50–100 µg in newborns (Delange and Burgi, 1989; Delange and Ermans, 1991). However, full compensation for fetal losses is not required in iodine sufficient populations, with an additional 50 µg/day likely to be sufficient where iodine stores are adequate (EFSA, 2014).

Despite implementation of mandatory fortification, evidence suggests that mild deficiency may persist among Australian and New Zealand women of childbearing age. Consequently, adequate iodine stores pre-conception cannot be assumed. Therefore, the EAR during pregnancy was set at 160 µg/day to account for almost full replacement. This also corresponds with balance data suggesting neutral balance among women with iodine intakes around 160 µg/day (Dworkin et al, 1966).

Lactation

Table 6. Iodine intake recommendations for during lactation

Age	EAR	RDI
Lactation (all ages)	190 µg/day	270 µg/day

Rationale: The EAR during lactation was calculated based on the EAR for adults, factoring in additional requirements during lactation.

As data suggests that mild iodine deficiency persists among Australian and New Zealand women of reproductive age, recommendations aimed to achieve almost full replacement of losses through breast milk, to ensure that dietary intakes are sufficient to meet both maternal and child requirements.

Studies suggest an additional requirement of 90–150 µg/day of iodine to account for iodine losses through breast milk (Dror & Allen 2018, Andersen et al 2014, EFSA 2014). On this basis, an EAR of 190 µg/day was estimated.

Upper level of intake

Table 7 outlines the upper levels of iodine intake recommended for various age groups and life stages to avoid the risk of excessive iodine consumption. This UL for infants was not reviewed in the 2025/26 update.

Table 7. Upper levels of iodine intake recommended to avoid the risk of excessive consumption

Age	UL
All	
0 - 12 months	Not possible to establish. Source of intake should be milk, formula and food only
1 to under 4 years	200 µg/day
4 to under 9 years	300 µg/day
Males	
9 to under 14 years	450 µg/day
14 to under 18 years	550 µg/day
Females	
9 to under 14 years	450 µg/day
14 to under 18 years	550 µg/day
Adults (18 years and over)	
Men (18 years and over)	600 µg/day
Women (18 years and over)	600 µg/day
Pregnancy	
All persons (18 years and over)	600 µg/day
Lactation	
All persons (18 years and over)	600 µg/day

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

Rationale: Recommendations were based on findings from a study that examined the effect of varying intakes on thyroid function in healthy euthyroid adults (Sang et al 2012). Elevated TSH concentrations were observed with intakes of 869 µg/day and higher, indicating a LOAEL (lowest observed adverse effect level) of 869 µg/day as a reference point. A UF (uncertainty factor) of 1.5 was applied, to reflect individual variability in sensitivity and account for uncertainty in the generalisability of the evidence to the Australian and New Zealand context. The estimated UL of 579.3 µg/day was then rounded up to 600 µg/day, owing to the soft and reversible nature of the end-point.

As there is insufficient evidence for other age groups, ULs for children and adolescents were extrapolated from the adult recommendation on a metabolic body weight basis using reference weights from the Methodological Framework (NHMRC, 2025).

The adult UL was also applied to pregnant and lactating populations, in the absence of robust evidence to suggest increased maternal or child sensitivity at levels below the adult UL.

Alternative age groupings – preschool, primary school and adolescents

For reporting usual intake against the national nutrition survey results, ages have also been grouped to provide levels for pre-school (2 to under 5 years), primary school age (5 to under 12 years) and adolescents (12 to under 18 years) in table 8. The derivation of the values uses the same methodology as table 7 and is based on the same reference adult value. If you are not comparing to survey intake data or school age groups, you should use the values in table 7.

Table 8. Alternative age groups for preschool, primary school and adolescents – upper levels of iodine intake recommended to avoid the risk of excessive consumption

Age	UL
Infants	
0 - 12 months	Not possible to establish. Source of intake should be milk, formula and food only
Children and adolescents	
12 to under 24 months	200 µg/day
2 to under 5 years (preschool)	250 µg/day
5 to under 12 years (primary school)	350 µg/day
12 to under 18 years (adolescence)	500 µg/day

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