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

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## FATS: TOTAL FAT AND FATTY ACIDS

### BACKGROUND

Fats are the most concentrated form of energy for the body (37 kJ/g). They also aid in the absorption of the fat-soluble vitamins, A, D, E and K and other fat-soluble biologically-active components. Chemically, most of the fats in foods are triglycerides, made up of a unit of glycerol combined with three fatty acids which may be the same or different. The differences between one triglyceride and another are largely due to the fatty acids content. Other dietary fats include phospholipids, phytosterols and cholesterol.

There are three major types of naturally-occurring fatty acids – saturated, *cis*-monounsaturated and *cis*-polyunsaturated. A fourth form, the *trans* fatty acids, are produced by partial hydrogenation of polyunsaturated oils in food processing and they also occur naturally in ruminant animal foods. Saturated fats are found mainly in animal-based foods and polyunsaturates and monounsaturates predominate in plant-based foods.

Saturated fatty acids contain no double bond; they are fully saturated with hydrogen. They are the main type of fatty acids found in milk, cream, butter and cheese, meats from most of the land animals, palm oil and coconut oil as well as in products such as pies, biscuits, cakes and pastries. Saturated fatty acids have both physiological and structural functions. They can be synthesised by the body so are not required in the diet.

The main monounsaturated fatty acid is oleic acid with one double bond. Olive, canola and peanut oils are rich in oleic acid. The monounsaturates are also synthesised by the body and are thus not required in the diet.

Polyunsaturated fatty acids contain two or more double bonds. The most common is linoleic acid (LA, 18:2). It is described as 'n-6' due to the position of the double bonds and occurs in seed oils, eg sunflower, safflower and corn. Other n-6 fatty acids include  $\gamma$ -linolenic (18:3), dihomo- $\gamma$ -linolenic (20:3), arachidonic acid (20:4) and adrenic acid (22:4). LA is the precursor of arachidonic acid, a substrate for eicosanoid production which is also involved in the regulation of gene expression (Ou et al 2001). LA is also found as a structural component of cell membranes and is important in cell signalling. High intakes of n-6 polyunsaturated fats have been associated with blood lipid profiles associated with a lower risk of coronary heart disease (eg lower total and LDL cholesterol, increased HDL cholesterol and reduced triacylglycerol) (Arntzenius et al 1985, Becker et al 1983, Sonnenberg et al 1996).

Smaller amounts of polyunsaturated fatty acids with double bonds in the n-3 position also occur in the diet. These are sometimes referred to as omega fatty acids. Humans are unable to insert a double bond at the n-3 position of a fatty acid and thus require a dietary source. The parent fatty acid of the n-3 series is  $\alpha$ -linolenic (ALA, 18:3). ALA is found in legumes, canola oils and margarines, linseed oils and products, certain nuts such as walnuts, and in small amounts in leafy vegetables. Canola oils and margarines and linseed oils are rich sources and legumes contribute some. A second group of n-3 fatty acids are the long chain (LC) acids eicosapentaenoic acid (EPA, 20:5), docosahexaenoic acid (DHA, 22:6) and docosapentaenoic acid (DPA, 22:5) that are found predominantly in oily fish such as mackerel, herrings, sardines, salmon and tuna and other seafood. Whilst  $\alpha$ -linolenic acid predominates in western diets, the fish oils, DHA, EPA and DPA predominate in other communities consuming their traditional diet, such as the Inuit (Holman et al 1982).

ALA primarily functions as a precursor for the synthesis of EPA which in turn forms DHA but may also have an independent role in protection against coronary heart disease via different mechanisms (Crawford et al 2000). Conversion of ALA to EPA and DHA is limited and varies according to the intakes of other fatty acids (Burdge et al 2003, Emken 2003, Pawlosky et al 2001). Thus, a typical intake of ALA may be less able to satisfy the physiological requirements for LC n-3 fatty acids than the smaller and often more variable intakes of pre-formed LC n-3 fatty acids.

DHA plays an important role as a structural membrane lipids, particularly in nerve tissue and the retina, and can also act as a precursor to certain eicosanoids. EPA is the precursor of the 3 series of prostaglandins and the 5 series of leukotrienes. In recent years, research has shown both cardiovascular and anti-inflammatory benefits of LC n-3 fatty acids (Albert et al 1998, 2002, Burr et al 1989, Dallongeville et al 2003, Djousse et al 2001, Dolecek 1992, GISSI-Prevenzione Investigators 1999, Hu et al 1999, Pischon et al 2003, WHO 2003). Early on, because of the nature of the fish oils used in studies, these benefits were attributed to EPA and its impact on eicosanoid production (Simopoulos 1991) but recent studies suggest that DHA is the primary mediator of cardiovascular benefits, influencing gene expression of key metabolic regulators, particularly in endothelial cells (Mori et al 1999). The potential role of DPA, as a very minor component of fish oil, has been largely ignored, despite the fact that recent research shows DPA contributes almost 30% of total LC n-3 in our diet (Howe et al 2003, 2005).

Until dose-response relationships have been established, the relative efficacy of EPA, DPA and DHA remains uncertain. Moreover, the extent of their interconversion is also uncertain. Hence it is not possible to differentiate between intake requirements for EPA, DPA and DHA at this stage.

A lack of dietary n-6 or n-3 polyunsaturated fatty acids is characterised by rough, scaly skin, dermatitis, increased transepidermal water loss, reduced growth and a high triene: tetraene ratio (Goodgame et al 1978, Holman et al 1982, Jeppesen et al 2000, Mascioli et al 1996, O'Neill et al 1977). They cannot be formed in the body and is therefore essential in the diet. Studies on patients given fat-free parenteral feeding have provided insight into the levels at which essential fatty acid deficiency occurs but are not sufficient to establish an average requirement (Fleming et al 1976, Goodgame et al 1978, Jeppesen et al 1998, Riella et al 1975).

There is some evidence that the ratio of n-6 to n-3 fatty acids may be important. Jensen et al (1997) reported that infants fed formulas containing an LA:ALA ratio of 4.8:1 had lower arachidonic acid concentrations and impaired growth compared to infants fed ratios of 9.7:1 or above. However, more recent large trials of ratios of 5:12 and 10:1 found no evidence of reduced growth or other problems (Simmer 2002). Various authorities have recommended ratios of LA:ALA or n-6:n-3 ratios ranging from 5:1 to 10:1 or 5:1 to 15:1 or 6:1 to 16:1 for infant formula (ESPGAN, Committee on Nutrition 1991, ISSFAL 1994, LSRO 1998).

A number of studies have looked at the n-6:n-3 ratio in relation to heart disease with inconsistent results (Dolecek & Graditis 1991, Ezaki et al 1999, Hu et al 1999, Kromhout et al 1985, Lands et al 1990, 1992, Nelson et al 1991, Shekelle et al 1985). However, on the basis of these results, the FAO:WHO Consultation on Fats and Oils (1994) recommended that the ratio of LA to ALA in the diet should be between 5:1 and 10:1 and suggested that individuals with a ratio greater than 10:1 should be encouraged to consume more n-3-rich foods. In contrast, an expert workshop in the Netherlands (de Deckere 1998) concluded that setting an n-6:n-3 ratio would not be helpful. They also proposed that there should be separate recommendations for plant (18:3) and marine (20:5, 22:5, 22:6) n-3 fatty acids.

Based on the concept of essentiality and given the lack of dose-response data to derive EARs for those components considered essential, AIs have been set for LA (n-6 in infants), ALA and the combined LC n-3 fatty acids, DHA:EPA:DPA. The AIs are based on median population intakes in Australia.

For children, adolescents and adults an EAR, RDI or AI for total fat was not set as it is the type of fats consumed that relate to essentiality and to many of the physiological and health outcomes. A suggested range of per cent energy as fat in relation to chronic disease prevention is addressed in the 'Chronic disease' section. In infancy, as fat is the major single source of energy in breast milk, an AI recommendation for total fat has been made based on breast milk composition. Recommendations for fatty acids in infancy are also based on total n-6 or n-3 derived from the composition of breast milk.

## RECOMMENDATIONS BY LIFE STAGE AND GENDER

<i>Infants</i>	AI		Fats
0–6 months	Total fat	31 g/day	
	n-6 polyunsaturated fats	4.4 g/day	
	n-3 polyunsaturated fats	0.5 g/day	
7–12 months	Total fat	30 g/day	
	n-6 polyunsaturated fats	4.6 g/day	
	n-3 polyunsaturated fats	0.5 g/day	

**Rationale:** The AI for 0–6 months was set by multiplying together the average intake of breast milk (0.78 L/day) and the average concentration of fat, n-6 or n-3 in breast milk (40; 5.6 and 0.63 g/L, respectively) from nine studies reviewed by FNB:IOM (2002) and rounding. The AI for 7–12 months was set by multiplying together the average intake of breast milk (0.6 L/day) and the average concentration of fat, n-6 or n-3 in breast milk (40; 5.6 and 0.63 g/L respectively) from nine studies reviewed by FNB:IOM (2002) and adding the median intake from complementary foods (5.7, 1.2 and 0.11 g/day, respectively) from the US CSFII data for 1994–96 (FNB:IOM 2002).

<i>Children, adolescents &amp; adults</i>	AI			Fats
	Linoleic acid	$\alpha$ -linolenic acid	Total LC n-3 (DHA+EPA+DPA)	
<b>Boys and girls</b>				
1–3 yr	5 g/day	0.5 g/day	40 mg/day	
4–8 yr	8 g/day	0.8 g/day	55 mg/day	
<b>Boys</b>				
9–13 yr	10 g/day	1.0 g/day	70 mg/day	
14–18 yr	12 g/day	1.2 g/day	125 mg/day	
<b>Girls</b>				
9–13 yr	8 g/day	0.8 g/day	70 mg/day	
14–18 yr	8 g/day	0.8 g/day	85 mg/day	
<b>Adults 19+ yr</b>				
Men	13 g/day	1.3 g/day	160 mg/day	
Women	8 g/day	0.8 g/day	90 mg/day	

**Rationale:** The AIs for LA and ALA were based on the highest median intakes of any of the gender-related age groups taken from an analysis of the National Nutrition Survey of Australia of 1995 (Howe et al 2003, 2005). For LC n-3, to overcome a marked gender disparity caused by particularly higher relative intakes in younger adult males (19–30 years), the AI was based on the median intake for all adults of the relevant gender. As national data were not available for New Zealand, similar values were assumed. The AIs do not necessarily reflect optimal intakes but are the values found in a population with no apparent essential fatty acid deficiency. (The ‘Chronic disease prevention’ section includes a suggested dietary target.)

<b>Pregnancy</b>	<b>AI</b>			<b>Fats</b>
	<b>Linoleic acid</b>	<b><math>\alpha</math>-linolenic acid</b>	<b>Total LC n-3 (DHA+EPA+DPA)</b>	
<b>14–18 yr</b>	<b>10 g/day</b>	<b>1.0 g/day</b>	<b>110 mg/day</b>	
<b>19–50 yr</b>	<b>10 g/day</b>	<b>1.0 g/day</b>	<b>115 mg/day</b>	

**Rationale:** Demand for n-6 and n-3 fatty acids for placental and fetal tissue must be met from maternal stores or by increased dietary intake, but there is a lack of data for assessing additional needs. The AIs for pregnancy were therefore based on that of the non-pregnant women, with an additional amount based on the increased average body weight in pregnancy (x 1.25).

<b>Lactation</b>	<b>AI</b>			<b>Fats</b>
	<b>Linoleic acid</b>	<b><math>\alpha</math>-linolenic acid</b>	<b>Total LC n-3 (DHA+EPA+DPA)</b>	
<b>14–18 yr</b>	<b>12 g/day</b>	<b>1.2 g/day</b>	<b>140 mg/day</b>	
<b>19–50 yr</b>	<b>12 g/day</b>	<b>1.2 g/day</b>	<b>145 mg/day</b>	

**Rationale:** There is a lack of data about the requirements in pregnancy, so the AIs were based on that for non-pregnant, non-lactating women plus that of the infant. As the infant recommendation includes only an AI for total n-3 based on milk concentration, this amount was apportioned between ALA and LC omega-3 in the same ratio as in the maternal AI when assessing the additional requirement.

## UPPER LEVEL OF INTAKE - TOTAL FAT AND FATTY ACIDS

**Linoleic acid:** No UL was set because there is no known level at which adverse effects may occur.

**$\alpha$ -linolenic acid:** No UL was set because there is no known level at which adverse effects may occur. The longer chain DHA, EPA and DPA fatty acids derived from ALA are more biologically-potent than ALA itself.

**LC n-3 fatty acids (DHA, EPA, DPA):**

<b>Infants 0–12 months</b>	<b>Not possible to establish</b>
<b>Children, adolescents and adults</b>	<b>3,000 mg/day</b>

**Rationale:** There is some evidence to suggest that high levels of these fatty acids may impair immune response and prolong bleeding time. However the immune function tests were performed in vitro and it is unclear how the results would translate to the in vivo situation. Prolonged bleeding times have been seen in the Inuit, but it is not known if they were caused by high LC n-3 consumption. The US Food and Drug Administration (DHHS 1997) has set a 'Generally Regarded as Safe' level of 3000 mg/day for LC n-3 which has been adopted here as the upper level of intake for children, adolescents and adults. (Note that is unlikely that this level of intake would be reached by consumption of seafood alone. If it were, then consideration would need to be given to the possible effects of concomitant intakes of other potential toxins such as mercury.) It is not possible to estimate an upper level of intake for infants.

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