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

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VITAMIN D

BACKGROUND

The major function of Vitamin D in humans is to maintain appropriate serum calcium concentrations by enhancing the ability of the small intestine to absorb calcium from the diet. Vitamin D also plays a role in enhancing absorption of phosphorus from the diet, but the blood concentration of phosphorus is not well regulated and varies according to supply and the renal excretory threshold.

Vitamin D maintains the blood calcium at supersaturating levels such that it is deposited in the bone as calcium hydroxyapatite. When dietary calcium is inadequate for the body's needs, 1,25-dihydroxyvitamin D [$1,25(OH)_2D$ or calcitriol] – the active form of vitamin D – together with parathyroid hormone, can mobilise stem cells in bone marrow to become mature osteoclasts which in turn increase the mobilisation of calcium stores from bone. However, there is a limited capacity to mobilise sufficient calcium from bone to have a significant effect on blood calcium levels.

Vitamin D occurs in two forms. One is produced by the action of sunlight on skin (D_3 or cholecalciferol) and the other is found in a limited range of foods (D_2 or ergocalciferol). With current food supplies and patterns of eating, it is almost impossible to obtain sufficient vitamin D from the diet alone (Fuller & Casparian 2001). Vitamin D in foods is fat soluble and is biologically less active. Its metabolite, 1.25-dihydroxyvitamin D ($1,25(OH_2)D$, or calcitriol) is the biologically active hormone responsible for its physiological actions. In the circulation, vitamin D appears as 25-hydroxyvitamin D (25(OH)D) which is five times more potent than cholecalciferol.

Vitamin D status is generally maintained in the population by exposure to sunlight (Glerup et al 2000, Holick 1996, Rasmussen et al 2000). If sunlight exposure is adequate, dietary vitamin D can be considered unnecessary (Holick 2001). In skin, 7-dehydrocholesterol is converted to pre-vitamin D_3 by a narrow band of solar ultraviolet radiation (290–320 nm) which undergoes isomerisation in a temperature-dependent manner to vitamin D_3.

Thus, vitamin D is not a nutrient in the usual sense, since under normal conditions it is supplied mainly by the skin. In addition, its physiological actions are attributable to the active metabolite, 1,25-dihydroxyvitamin D which, because it is synthesised in the kidneys and acts elsewhere, is often called a hormone.

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1 μg cholecalciferol is equal to 0.2 μg 25(OH)D.

Vitamin D is also sometimes expressed in International Units where 1 IU equals 0.025 μg cholecalciferol or 0.005 μg 25(OH)D.

Seasonal changes have been shown to have a significant effect on the cutaneous production of cholecalciferol (Pettifor et al 1996, Webb et al 1990). In the winter months in temperate latitudes, solar UV light in the wavelength range of 290–320 nm is absorbed by the atmosphere. People also spend less time outdoors and wear more clothing. For this reason, vitamin D deficiency is more common in the winter months (Holick 1995).

Despite the sunny climate, a seasonal variation in vitamin D levels also occurs in Australia. In the Geelong Osteoporosis Study, the mean vitamin D levels for winter were 58 nmol/L compared with 70 nmol/L in summer (Pasco et al 2001). However, after regular sun exposure, people under the age of 50 can produce and store approximately 6 months' worth of vitamin D, so vitamin D stored in the body is available during the winter when production is minimal (Holick 1996). However, in older people, the efficiency of cutaneous synthesis of vitamin D is significantly less than that in younger people (Holick et al 1989, Need et al 1993).

Other environmental factors such as the angle of the sun, distance from the equator, the amount of cloud cover and the amount of particulate matter in the atmosphere (Holick 1995, Kimlin et al 2003, Madronich et al 1998) can affect the amount of vitamin D produced. Comparative data indicate that Northern and Southern latitudes are not equivalent. It has been estimated that ultraviolet levels in summer are up to 40% higher in New Zealand than in the equivalent Northern latitudes (Madronich et al 1998).

Deficiency of Vitamin D results in inadequate mineralisation or demineralisation of the skeleton. This can lead to rickets in young children, causing bowed legs and knocked knees. A study in China showed that vitamin D given as a supplement over 2 years increased both total body bone mineral content and bone mineral density in older children (Du et al 2004). In adults, deficiency can lead to increased bone turnover and osteoporosis and less commonly to osteomalacia for which the associated secondary hyperparathyroidism enhances mobilisation of calcium from the skeleton, resulting in porotic bone. Vitamin D may also affect fracture rates via mechanisms other than its influence on bone mass. Bischoff-Ferrari et al (2004) showed that on the basis of five RCTs involving 1,237 participants, vitamin D reduced the number of falls by 22% compared with patients receiving calcium or placebo.

Vitamin D is also thought to play a role in maintaining the immune system (Brown et al1999, DeLuca 1998) and helping maintain healthy skin (DeLuca 1998, Jones et al 1998) and muscle strength (Brown et al 1999).

There is increasing recognition that a significant number of Australians and New Zealanders may have less than optimal 25(OH)D status, however limited published information of the prevalence of vitamin D deficiency in Australia is available, other than from relatively small subpopulations (Nowson & Margerison 2002, Pasco et al 2004). Some information is available currently in unpublished form, from the national surveys of 1997 and 2002 in New Zealand (Green et al 2004a,b). Recent analyses of blood samples from these surveys showed that 31% of New Zealand children aged 5–14 years whose bloods were sampled in 2002 had a serum 25(OH)D concentration indicative of vitamin D insufficiency. Between 0% (for 5–6 year olds of European background) and 14% (for girls aged 11–14 years of Pacific Island backgrounds) had vitamin D deficiency. For adolescents at or above 15 years and adults whose bloods were sampled in 1997, the prevalence of deficiency, defined as <17.5 nmol/L, was 2.8%, but the prevalence of insufficiency, defined as <37.5 nmol/L, was 27.6%. Vitamin D concentrations were lower in winter than summer and lower in Pacific peoples and Mäori than those of European and other origins.

The groups thought to be at particular risk in Australia and New Zealand include older persons living in the community, those in residential care with limited mobility for whom frank deficiency may be 22–67% and mild deficiency may be 45–84%, dark-skinned peoples and veiled women who have limited exposure to sunlight (as many as 80% having mild deficiency) and breast-fed infants of these groups of women. Some of these groups (eg the institutionalised elderly) are often not represented in National Surveys.

Adolescents and young children growing rapidly who are on marginal calcium intakes may also have increased requirements for vitamin D that may not be met in winter, when reduced exposure to sunlight depletes the body's stores of vitamin D. There is also some evidence that up to 8% of younger women (20–39 years) may have a frank vitamin D deficiency at the end of winter and 33% may have a marginal deficiency. People who wear protective clothing, always use sunscreen and those who have intestinal, hepatic, renal or cardiopulmonary disease or are taking anticonvulsants may also be at increased risk (Compston 1998, Fitzpatrick et al 2000, Fuller & Casparian 2001, Thomas et al 1998).

Very few foods contain significant amounts of vitamin D (Holick 2001, Vieth 1999). In Australia, fortified margarine appears to be the major dietary source of vitamin D, together with fatty fish such as salmon, herring and mackerel, and eggs (Baghurst & Record 2002).

Accurate estimates of dietary intakes of vitamin D in Australia and New Zealand are not yet available as local food databases are limited. Some estimates have been made using a mix of local and overseas information on food composition with figures between 2-3mg/day for adults (Baghurst & Record 2002,

LINZ 1992). Currently in Australia, vitamin D fortification is mandated for edible oil spreads (table margarine) and voluntary for modified and skim milks, powdered milk, yoghurts and table confections and cheese. In New Zealand, fortification of margarine or milk products with vitamin D is not mandated, however since 1996, voluntary fortification of margarine, fat spreads and their reduced fat counterparts has been permitted. It is also permitted to add vitamin D to dried milk, dried skim milk and non-fat milk solids, skim milk and reduced fat cows' milk, legume beverages and 'food' drinks.

Serum 25(OH)D is the indicator of choice for assessing requirements since it accounts for both dietary and cutaneous sources of the vitamin. However, there is some disagreement in the literature and clinical practice over quantification of the optimal range. A 25(OH)D below 27.5 nmol/L is consistent with vitamin D deficiency in infants, neonates and young children (Specker et al 1992) and is thus used as the key indicator for determining a vitamin D reference value. Little information is available on the levels required to maintain normal calcium metabolism and peak bone mass in children, or young and middle-aged adults but in a recent position statement a Working Group of the Australian and New Zealand Bone and Mineral Society, the Endocrine Society of Australia and Osteoporosis Australia (2005) defined mild deficiency for adults as serum 25-OHD levels between 25 and 50nmol/L; moderate deficiency as between 12.5 and 25nmol/L and severe, below 12.5nmol/L based on various indicators such as increases in parathyroid hormone secretion and various bone indicators. There is mounting evidence for the elderly to support increased dietary requirements for the maintenance of normal metabolism and maximum bone health (Dawson-Hughes et al 1991, Krall et al 1989, Lips et al 1988) and some researchers recommend levels of 75–100 nmol/L, especially for the elderly, on the basis of optimising bone (Dawson-Hughes 2004, Dawson-Hughes et al 1997, Heaney 1999, 2004, Kinyamu et al 1998, Sahota 2000, Vieth et al 1999, Vieth 2004).

When 25(OH)D concentrations are in the deficient range, serum PTH levels are inversely proportional to 25(OH)D levels, and can therefore also be a valuable indication of inadequate vitamin D status, as can skeletal health including bone development and prevention of rickets in infants and children and bone mineral content, bone mineral density and fracture risk in adults.

The recommendations herein assume no, or minimal, exposure to sunlight as sunlight exposure factors and environmental factors can vary widely between individuals across Australia and New Zealand. An assessment of the effect of environmental and personal factors in reducing this requirement is also given, although data are limited.

RECOMMENDATIONS BY LIFE STAGE AND GENDER

Infants	AI	Vitamin D
0–6 months	5.0µg/day	
7–12 months	5.0µg/day	

Rationale: Maternal vitamin D status in pregnancy affects the status of the infant for the first few months of life. If maternal vitamin D status is good during the last stages of pregnancy the newborn child should have adequate vitamin D status for some time after birth in the absence of significant input from the diet. Human milk has very little vitamin D, so infants not exposed to sunlight are unlikely to obtain adequate vitamin D from mother's milk beyond early infancy (Nakao 1988, Specker et al 1985). The AI for infants 0–12 months is based on the lowest dietary intake of vitamin D associated with a mean serum 25 (OH)D concentration of greater than 27.5 nmol/L (lower limit of normal) assuming little or no exposure to sunlight (FNB:IOM 1997). In these circumstances, a minimal intake of 2.5 µg/day will likely prevent rickets in babies 0–6 months (Glaser et al 1949, Specker et al 1992). At this intake, in the absence of sunlight, many will have 25(OH)D levels within the range sometimes seen in rickets (Specker et al 1992). Thus the AI is set at 5 µg/day. Several studies have shown that this level would also be adequate for older babies (Greer et al 1982a, Leung et al 1989, Markestad & Elzouki 1991) and for formula-fed infants (Koo & Tsang 1995, Markestad & Elzouki 1991).

Role of sunlight exposure: Estimates from the Midwest in the US suggest that to get sufficient vitamin D from sunlight alone, infants need to be exposed for 2 hours a week if just their face is exposed or 30 minutes a week with just a nappy on (Specker et al 1985). With habitual small doses of sunshine, breast or formula-fed infants do not require supplemental vitamin D. However, the infants of dark-skinned and/or veiled women may be at higher risk of developing rickets (Grover & Morley 2001). Their mothers often have marginal or frank vitamin D deficiency resulting in low status at birth. The vitamin D status of the infants is further compromised by restricted exposure to sunlight, and reduced ability to synthesise 25(OH)D due to skin pigmentation.

Children & adolescents	AI	Vitamin D
All		
1–3 yr	**5.0 µg/day**	
4–8 yr	**5.0 µg /day**	
Boys		
9–13 yr	**5.0 µg/day**	
14–18 yr	**5.0 µg/day**	
Girls		
9–13 yr	**5.0 µg/day**	
14–18 yr	**5.0 µg/day**	

Rationale: In the absence of data on how much vitamin D is required to prevent deficiency in 1–8-year olds, recommendations were derived from data on slightly older children with limited sunlight exposure (Aksnes & Aarskog 1982, Gultekin et al 1987). Most children with a dietary intake of 1.9–2.5 µg/day had no evidence of deficiency as defined by blood levels of 25(OH)D below 27.5 nmol/L. Adolescents and young children growing rapidly who are on marginal calcium intakes may have increased requirements for vitamin D which may not be met in winter, when reduced exposure to sunlight depletes the body stores of vitamin D. A requirement of 2.5 µg/day regardless of sunlight was seen as prudent and was doubled to cover the needs of all children of this age to give the AI of 5 µg/day (FNB:IOM 1997).

Role of sunlight exposure: With regular sun exposure, there would not be a dietary need for vitamin D in children and adolescents (Ala-Houhala et al 1984, Gultekin et al 1987, Pettifor et al 1978, Riancho et al 1989, Taylor & Norman 1984). However, children living in far southern latitudes and those with dark skins such as indigenous Australians and New Zealanders, and certain migrant groups, or those who are covered for cultural reasons, may be unable to synthesise enough vitamin D in their skin in store for winter. Jones et al (1999) showed that 10% of children in southern Tasmania assessed in mid-winter had plasma 25(OH)D lower than 25 nmol/L, a level considered insufficient. There has been a reported increase in the presentation of rickets in Victorian children, mainly due to restricted sun exposure in mothers who are often dark skinned and veiled. In New Zealand, from national survey data, 4% of children aged 5–14 years had levels below 17.5 nmol/L and 1–2% of adolescents aged 15–18 years (Green et al 2004a,b).

Adults	**AI**	**Vitamin D**
Men		
19–30 yr	5.0 µg /day	
31–50 yr	5.0 µg /day	
51–70 yr	10.0 µg /day	
>70 yr	15.0 µg /day	
Women		
19–30 yr	5.0 µg /day	
31–50 yr	5.0 µg /day	
51–70 yr	10.0 µg /day	
>70 yr	15.0 µg /day	

Rationale: The AI for younger adults (19–50 years) is based on the amount of vitamin D required to maintain serum 25(OH)D at a level of at least 27.5 nmol/L with minimal exposure to sunlight. One study of US women of this age (Kinyamu et al 1997) showed that an average intake of 3.3–3.4 µg/day resulted in serum 25(OH)D of greater than 30 nmol/L. A study of females in Australia undertaken across both the summer and winter months at latitude 38º (Pasco et al 2001), assessed median intakes to be only 1.3 µg/day (much lower than other estimates for Australia and New Zealand), but had only 7% of subjects with serum 25(OH)D below 28 nmol/L in summer and 11% in winter. A vitamin D intake of 2.5 µg/day was seen as prudent for this age group. There are no data on men on which to set a figure except from one study of submariners not exposed to sunlight, whose status was assessed with or without a 15 µg/day supplement (Holick, 1994). However, the effects of lower doses were not assessed in this study. It is therefore assumed that requirements for men will be the same as those for women.

To cover the needs of all adults in the age range of 19–50 years, regardless of exposure to sunlight and in recognition of the fact that the available data were collected in women, a figure of 5 µg/day was set as the AI for younger adults. The AI was raised to 10 µg/day for adults aged 51–70 years to account for the reduced capacity for the skin to produce vitamin D with ageing (Holick et al 1989, Need et al 1993). Data on bone loss and vitamin D supplementation in women were also taken into consideration (Dawson-Hughes et al 1991, 1995). For adults over 70 years, the AI was raised to 15 µg/day. Studies of elderly people with intakes of 9.6 µg, 7.1 µg or 5.2 µg vitamin D/day showed that 8, 14 and 45%, respectively had low levels of serum 25(OH)D (Gloth et al 1995, Kinyamu et al 1997, O'Dowd et al 1993). A value of 7.5 µg/day was considered prudent for those with limited sun exposure and was doubled to 15 µg/day to cover the needs of all adults of this age, regardless of sun exposure or body stores.

It should be noted that the effect of increasing the dietary intake of vitamin D on 25(OH)D concentration in blood varies according to the existing vitamin D status of the individual. The status of those with low 25(OH)D levels in plasma will be improved to a more significant degree than of those with pre-existing high status (eg plasma levels above about 50 nmol/L) who may benefit little from the additional dietary intake.

Role of sunlight exposure: There is evidence from selected subpopulations that about 4–8% of adults in Australia have serum 25(OH)D levels below 28 nmol/L and about 30% have levels below 50 nmol/L (Pasco et al 2001, MacGrath et al 2001, Vasikaran et al 2000). National surveys in New Zealand have indicated that some 2.8% of adults have levels of less than 17.5 nmol/L and 27.6% have levels below 37.5 nmol/L. Both sunlight and diet play an essential role in vitamin D status in younger adults. Kimlin et al (2003) estimated that for an older woman with fair skin, exposure of 6% of the body surface (face, hands, forearm) to sunlight for 15–30 minutes, 2–3 times per week would provide the equivalent of 15 µg vitamin D/day. Because of reduced cutaneous production, young adults (19–50 years) who live in southern latitudes such as Tasmania and the southern island of New Zealand are particularly at risk of becoming vitamin D deficient during the winter months.

For dark-skinned peoples such as indigenous Australians and New Zealanders and certain migrant groups and veiled women, there is evidence in Australia of high rates of vitamin D deficiency. Grover et al (2001) found that 80% of pregnant dark-skinned, veiled women attending one antenatal clinic in a large teaching hospital had vitamin D levels of less than 22 nmol/L. For people with little access to sunlight a supplement of 10 µg/day would not be excessive.

Institutionalised elderly: Several studies in Australia and New Zealand have shown high rates of deficiency in very elderly people with restricted access to sunlight, many of whom live in institutions. Estimates of deficiency range from 15–52% in Australia (Bruce et al 1999, Flicker et al 2003, Inderjeeth et al 2000, Stein 1996). Ley et al (1999) found that 49% of older New Zealand subjects in winter and 33% in summer had low serum 25(OH)D while McAuley et al (1997) reported 69% of subjects in Dunedin having low levels in winter, but only 26% in summer. Data from the National Nutrition Survey of New Zealand (Green et al 2004b) showed that 1.6% of males over 65 years and 5.8% of females had blood levels below 17.5 nmol/L for serum 25(OH)D and that 20.5% of men and 39.6% of women had levels below 37.5 nmol/L. This survey did not include institutionalised people. The recommendation of 15 µg/day for those over 70 years relates to the general population over 70 years. A number of recent studies demonstrate protection from falls and fractures with supplemental intakes of vitamin D in the elderly.

For institutionalised or bed-bound elderly who have very restricted exposure to sunlight often accompanied by reduced food intake, supplementation with vitamin D in the order of 10–25 µg/day may be necessary (Brazier et al 1995, Byrne et al 1995, Chapuy et al 1992, Egsmose et al 1987, Fardellone et al 1995, Kamel et al 1996, McKenna 1992, Sebert et al 1995, Sorva et al 1991).

Pregnancy	AI	Vitamin D
14–18 yr	5.0 µg/day	
19–30 yr	5.0 µg/day	
31–50 yr	5.0 µg/day	

Rationale: Although there is placental transfer of vitamin D and its metabolites from mother to fetus, the amounts are too small to affect the mother's vitamin D requirement, particularly as there is a rise in serum calcitriol (probably of placental origin) and a rise in calcium absorption in late pregnancy (Paunier et al 1978, Specker 2004). However, maternal deficiency of vitamin D can affect the fetus and needs to be prevented. Pregnant women who receive regular exposure to sunlight do not require supplementation. However, at intakes of less than 3.8 µg/day, pregnant women in winter months at high latitudes have been shown to have low serum 25(OH)D (Paunier et al 1978). For women who have little access to sunlight, a supplement of 10 µg/day prenatally would not be excessive. In the last trimester of pregnancy there is quite a large transfer of 25(OH)D across the placenta.

Lactation	AI	Vitamin D
14–18 yr	5.0 µg/day	
19–30 yr	5.0 µg/day	
31–50 yr	5.0 µg/day	

Rationale: There is no evidence that lactation increases the AI of the mother for vitamin D. Thus, if sunlight is inadequate, an AI of 5 µg/day is needed. As noted above, the infants of dark-skinned and/or veiled women may be at higher risk of developing rickets partly because of marginal or frank vitamin D deficiency in the mother. For mothers and their babies with limited exposure to sunlight, a supplemental intake during lactation of 10 µg/day would not be excessive.

UPPER LEVEL OF INTAKE - VITAMIN D

Infants

0–12 months	25 µg /day

Children and adolescents

1–3 yr	80 µg/day
4–8 yr	80 µg/day
9–13 yr	80 µg/day
14–18 yr	80 µg/day

Adults 19+ yr

Men	80 µg/day
Women	80 µg/day

Pregnancy

14–18 yr	80 µg/day
19–50 yr	80 µg/day

Lactation

14–18 yr	80 µg/day
19–50 yr	80 µg/day

Rationale: The UL for infants was set on the basis of a NOAEL of 45 µg/day (Fomon et al 1966, Jeans & Stearns 1938) together with a UF of 1.8 (FNB:IOM 1997) because of the small sample sizes and insensitivity of the endpoint used (linear growth). For children and adolescents, there are little available data, so the recommendation for adults was adopted.

The UL for adults was based on studies assessing the effect of vitamin D on serum calcium in humans (Honkanen et al 1990, Johnson et al 1980, Narang et al 1984, Vieth et al 2001). Johnson et al (1980) and Honkanen et al (1990) conducted studies with supplementation at 50 µg/day or 45 µg/day for several months and saw no adverse effects. Narang et al (1984), using dosages of 60 µg and 95 µg/day over several months in a non-randomised trial that included 30 normal controls, saw increases above 2.75 mmol/L in serum calcium levels a level considered as defining hypercalcaemia, at 95 µg/day but not at 60 µg/day. However, a recent, well-designed, RCT by Vieth et al (2001) saw no adverse effect of dosages of 25 µg/day or 100 µg/day over six months in 30 subjects. This finding was confirmed in a later randomised study (Vieth et al 2004) of inpatients with subclinical or marginal deficiency. Vieth et al (2001) felt that the earlier data of Narang et al (1984) may have been erroneous in dosage, citing concerns about lack of independent confirmation of the actual amount of vitamin D administered (there were no measures of serum 25(OH)D). There is also some animal evidence of oral vitamin D causing non-calcified atherosclerosis of large arteries (Taura et al 1979, Toda et al 1985), suggesting that a cautious approach should be taken to high dose vitamin D in people other than the elderly.

Taking all of this into account, the figure of 100 µg/day from Vieth's studies was adopted as the NOAEL and a UF of 1.2 was applied because of the inconsistencies in the studies and they were performed on relatively small number of subjects with pre-existing marginal vitamin D status. Vieth et al (2001) have themselves cautioned about the relatively small numbers in their studies.

The available data for pregnancy and lactation are inadequate to derive a figure different from that of other adults. There appears to be no increased sensitivity during these physiological states.

It should be noted that the intake of vitamin D via food would add to the vitamin D formed by exposure to sunlight.

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