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

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PANTOTHENIC ACID

BACKGROUND

Pantothenic acid is a component of coenzyme A (CoA) and phosphopantetheine, both of which are involved in fatty acid metabolism (Tahikliani & Beinlich 1991). It is essential to almost all forms of life and is widely distributed in foods. Chicken, beef, potatoes, oat-based cereals, tomato products, liver, kidney, egg yolks and whole grains are major sources in western diets (Plesofsky-Vig 1996, Walsh et al 1981). Little information is available about bioavailability, with estimates ranging from 40 to 61% (Tarr et al 1981). Neither is there much information about interactions with other nutrients, although there is some information that implies that thiamin, and to a lesser extent riboflavin, can affect pantothenate metabolism and excretion (Koyanagi et al 1969).

Absorption is by active transport at low concentrations and by passive transport at high concentrations. The active system can be saturated, so absorption is less efficient at higher intakes. Pantothenic acid can be synthesised by microbes but the extent to which this happens in man is unknown.

CoA is synthesised from pantothenate in a reaction catalysed by pantothenate kinase. In the form of acetyl CoA and succinyl CoA, CoA plays an important role in the synthesis of fatty acids and membrane phospholipids and also of amino acids, steroid hormones, vitamins A and D, porphyrin and corrin rings, and neurotransmitters. CoA is also needed for acetylation and acylation of proteins. CoA is hydrolysed to pantothenate and the pantothenic acid is excreted intact in urine. Pantothenic acid deficiency is only seen in individuals fed synthetic diets (Fry et al 1976) or in those fed an antagonist (Hodges et al 1958, 1959), although it was implicated in 'burning feet' syndrome in Asia during World War II (Glusman 1947). The symptoms include irritability, restlessness, fatigue, apathy, malaise, sleep disturbance, nausea, vomiting and cramping, numbness and staggering gait, as well as hypoglycaemia and increased insulin sensitivity.

A number of markers have been used to assess adequacy of intake including urinary excretion (Eissenstat et al 1986, Fry et al 1976, Tarr et al 1981) and blood levels (Annous & Song 1995, Baker et al 1969, Cohenour & Calloway 1972, Eissenstat et al 1986, Wittner et al 1989).

RECOMMENDATIONS BY LIFE STAGE AND GENDER

| <i>Infants</i> | AI | Pantothenic acid |
|--------------------|-------------------|-------------------------|
| 0–6 months | 1.7 mg/day | |
| 7–12 months | 2.2 mg/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 pantothenate in breast milk of 2.2 mg/L (Picciano 1995), and rounding (FNB:IOM 1998). For 7–12 months, the AI was derived by extrapolating up from younger infants using metabolic body weight ratios.

| Children & adolescents | AI | Pantothenic acid |
|-----------------------------------|------------|-------------------------|
| All | | |
| 1–3 yr | 3.5 mg/day | |
| 4–8 yr | 4.0 mg/day | |
| Boys | | |
| 9–13 yr | 5.0 mg/day | |
| 14–18 yr | 6.0 mg/day | |
| Girls | | |
| 9–13 yr | 4.0 mg/day | |
| 14–18 yr | 4.0 mg/day | |

Rationale: As there are no data to set EARs and thus RDIs, AIs were set for children and adolescents. AIs were set on the median intake level from National Dietary Surveys in Australia, 1995 and New Zealand, 1991 (Baghurst & Record 2004, LINZ 1992), with cross-referencing to some dietary intake/urinary excretion data for children (Eissenstat et al 1986, Kathman & Kies 1984, Kerrey et al 1968, Pace et al 1961, Wittner et al 1989).

| Adults | AI | Pantothenic acid |
|---------------|-----------|-------------------------|
| Men | | |
| 19–30 yr | 6 mg/day | |
| 31–50 yr | 6 mg/day | |
| 51–70 yr | 6 mg/day | |
| >70 yr | 6 mg/day | |
| Women | | |
| 19–30 yr | 4 mg/day | |
| 31–50 yr | 4 mg/day | |
| 51–70 yr | 4 mg/day | |
| >70 yr | 4 mg/day | |

Rationale: As there are limited data to set EARs, AIs were set for adults using the median population intake data from Australia and New Zealand men and women (Baghurst & Record 2004, LINZ 1992). As dietary intake data often underestimate intakes somewhat, the highest intake for any age category for the men or women was applied to the other age groups within that gender. The data for women are supported by the only study of the relationship between dietary intake and excretion in adults (Fox & Linkswiler 1961) that showed that a pantothenic acid intake of 4 mg/day was adequate.

| Pregnancy | AI | Pantothenic acid |
|------------------|-----------|-------------------------|
| 14–18 yr | 5 mg/day | |
| 19–30 yr | 5 mg/day | |
| 31–50 yr | 5 mg/day | |

Rationale: There are limited data about the needs for pantothenic acid in pregnancy. The AI was therefore set by reference to the non-pregnant intake data with an allowance for the average additional body weight in pregnancy.

| Lactation | AI | Pantothenic acid |
|------------------|-----------|-------------------------|
| 14–18 yr | 6 mg/day | |
| 19–30 yr | 6 mg/day | |
| 31–50 yr | 6 mg/day | |

Rationale: Needs in lactation increase as a substantial amount of pantothenate is secreted in human milk (1.7 mg/day). An additional 2 mg/day is therefore added to the non-pregnant, non-lactating AI.

UPPER LEVEL OF INTAKE - PANTOTHENIC ACID

A UL cannot be determined at this stage.

There are no reports of adverse effects of oral pantothenic acid in either humans or animals on which to base a quantitative estimate. Thus a UL cannot be determined at this stage, but current intakes are unlikely to be associated with adverse health effects.

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