The following is an extract from:

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

ENDORSED BY THE NHMRC ON 9 SEPTEMBER 2005

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DIETARY FIBRE

BACKGROUND

Adequate dietary fibre is essential for proper functioning of the gut and has also been related to risk reduction for a number of chronic diseases including heart disease, certain cancers and diabetes (see 'Chronic disease' section for further discussion).

There is no single definition of dietary fibre, which is a component of all plant materials. What can be said with certainty is that most of the components of dietary fibre are carbohydrate in nature, lignin being an exception. Hipsley first used the term 'dietary fibre' in 1953 to describe plant cell walls in the diet, which were thought to protect against toxaemia of pregnancy. This term, later taken up by Trowell (1972), encompassed only components of the plant cell wall that resisted digestion by secretions of the human alimentary tract, namely cellulose, hemicelluloses, pectin and lignin.

Trowell described dietary fibre as either 'the skeletal remains of cell walls' or as 'remnants of the plant cell wall' (Trowell 1972, 1975). As it is difficult to determine whether indigestible materials from plants came from the cell wall or other parts, the definition was expanded to include all indigestible components of plant origin (Trowell et al 1976). In 1987, the Life Sciences Research Office of the Federation of American Societies for Experimental Biology (1987) adopted a definition of dietary fibre as 'the endogenous components of plant materials in the diet which are resistant to digestion by enzymes produced by humans'. This definition can be considered to include some components of what is now known as resistant starch (RS). As pointed out by Southgate (1991), this definition is virtually identical to that for 'unavailable carbohydrates' as originally defined in McCance & Lawrence (1929).

One difficulty with the word endogenous in this definition is that it excludes, for example, those forms of RS that arise as a consequence of cooking and processing techniques. It also excludes substances which are intimately associated with the major components of dietary fibre and which are capable of having important nutritional and/or physiologic effects such as phytates, lectins, saponins, non-polymeric polyphenols, and inorganic constituents. Recent data have indicated that while non-starch polysaccharides (NSP) are important for human health, RS may be as significant if not more so for many health conditions (Topping & Clifton, 2001).

Food Standards Australia New Zealand (FSANZ) defines Dietary Fibre as follows:

> 'Dietary fibre means that fraction of the edible parts of plants or their extracts, or synthetic analogues, that are resistant to the digestion and absorption in the small intestine, usually with complete or partial fermentation in the large intestine. Dietary fibre includes polysaccharides, oligosaccharides (degree of polymerisation >2) and lignins, and promotes one or more of the following beneficial physiological effects:
>
> (i)   laxation
>
> (ii) reduction in blood cholesterol
>
> (iii) modulation of blood glucose'.

This definition was gazetted in Standard 1.2.8 of the ANZ Food Standards Code in August 2001. The code also prescribes a number of acceptable Association of Official Analytical Chemists (AOAC) methods of analysis for total dietary fibre or its components that led to the inclusion of inulin, fructo-oligosaccharides and polydextrose in the category of dietary fibre. At the time of publication of the current document, FSANZ has not assessed a method for assaying RS.

In Australia, the National Nutrition Survey of 1995 indicated that 45% of dietary fibre comes from breads and other cereal foods, 10% from fruit and 30% from vegetables (NNS 1998). The distribution is similar in New Zealand, with 44% from breads and cereals, 13% from fruit and 28% from vegetables (MOH 1999). However, it is worth noting that the food data bases for dietary fibre used for these surveys do not equate precisely to the FSANZ definition as the analytical methods used (AOAC in Australia and

Englyst in New Zealand) measure a different set of components. Nevertheless, the differences have been assumed to be relatively small.

Resistant starch comes within the FSANZ definition but is only partially assessed using currently approved methods that account for only about 40% of RS. Baghurst et al (1996) estimated intakes of RS in Australia and New Zealand based on national nutrition surveys in the mid 1980s for Australia and early 1990s for New Zealand. This analysis showed an average figure of 4.0 g RS/100 g starch for men, 4.7 g RS/100 g starch for women and 4.5 g RS/100 g starch for children.

It has been postulated that diets high in fibre have a lower energy density and may therefore help in moderating obesity. The exact mechanisms by which these apparent health benefits may arise have not been determined. In almost every instance, there exists the possibility that the observed associations are indirect as a consequence of chemoprotective effects of non-nutrients closely associated with the fibre components of fruits, vegetables and cereal foods. Further discussion of the potential role of fibre in relation to chronic disease is given in the 'Chronic disease' section.

Only in the case of laxation is there evidence of both protective (Sanjoaquin et al 2004) and therapeutic actions (Topping & Clifton 2001). This laxative effect accounts for the role of dietary fibre in conditions such as hiatus hernia, diverticular disease and haemorrhoids. These latter conditions may also be affected by adequacy of fluid ingestion. Regional differences in the occurrence of these diseases generated the original hypothesis of Burkitt & Trowell (1975). However, there are few studies that have looked at the role of dietary fibre in the aetiology, rather than treatment, of these diseases. Dietary fibre is the most effective treatment for all forms of constipation due to its influence on faecal bulk and consistency.

Assessment of dietary fibre needs is complex as the endpoints are ill defined. There is no biochemical marker that can be used to determine dietary fibre needs, so appearance or disappearance of clinical endpoints needs to be considered. In keeping with the concept of setting EARs and RDIs or AIs for prevention of deficiency states, the endpoints chosen in the estimation of requirements were adequate gastrointestinal function and adequate laxation rather than reduction of risk for chronic disease.

From a meta analysis of about 100 studies of changes in stool weight with various forms of fibre, the increase in faecal weight due to ingestion of fibre has been estimated (Cummings 1993). An increase of 1 g in faecal bulk can be achieved with an additional 3 g of isolated cellulose, 5.4 g of wheat bran, 1.3 g of isolated pectin and 4.9 g of fruit and vegetables (Hillman 1983). Resistant starch has very limited effect (Behall & Howe 1996, Cummings et al 1996, Heijnen et al 1998, Jenkins et al 1998). However, increased faecal weight does not necessarily equate to enhanced laxation as other factors such as water can affect laxation directly or be a necessary adjunct to increased fibre intakes (Anti et al 1998).

Assessing the stool weight that will promote laxation and prevent constipation is very difficult. For these reasons, it is not possible to establish an EAR. Instead, an AI has been derived based on median intakes in populations like Australia and New Zealand where laxation problems are not common.

The potential benefits of higher than AI intakes on chronic disease aetiology are discussed in the 'Chronic disease' section.

RECOMMENDATIONS BY LIFE STAGE AND GENDER

Infants	**AI**	**Dietary Fibre**
0–6 months	No AI has been set	
7–12 months	No AI has been set	

Rationale: There are no functional criteria for dietary fibre in infants. Human milk contains no dietary fibre and as such no AI is set.

Children & adolescents	AI	Dietary Fibre
All		
1–3 yr	**14 g/day**	
4–8 yr	**18 g/day**	
Boys		
9–13 yr	**24 g/day**	
14–18 yr	**28 g/day**	
Girls		
9–13 yr	**20 g/day**	
14–18 yr	**22 g/day**	

Rationale: The AI is set at the median for dietary fibre intake in Australia and New Zealand for children of these ages based on the National Dietary Surveys of Australia undertaken in 1995 and New Zealand undertaken in 2002 (ABS 1998, MOH 2003) plus an allowance ranging from 2–4 g/day for the different age/gender groups for a component of RS not included in the food data base used for these surveys, and rounding.

Adults	AI	Dietary Fibre
Men		
19–30 yr	**30 g/day**	
31–50 yr	**30 g/day**	
51–70 yr	**30 g/day**	
>70 yr	**30 g/day**	
Women		
19–30 yr	**25 g/day**	
31–50 yr	**25 g/day**	
51–70 yr	**25 g/day**	
>70 yr	**25 g/day**	

Rationale: The AI is set at the median for dietary fibre intake in Australia and New Zealand based on the 1995 National Nutrition Survey of Australia (ABS 1998) and the 1997 National Nutrition Survey of New Zealand (MOH 1999). The value within each gender was set for all ages at the highest median of any of the age groups plus an allowance of slightly more than 4 g/day for men and slightly less than 3 g/day for women for the component of RS not included in the food data base for dietary fibre used for these surveys, and rounding.

Pregnancy	AI	Dietary Fibre
14–18 yr	**25 g/day**	
19–30 yr	**28 g/day**	
31–50 yr	**28 g/day**	

Rationale: There is no evidence for increased metabolic needs in pregnancy. To allow for additional body weight, the AI is increased in relation to increased energy needs of about 12%, with rounding.

Lactation	**AI**	**Dietary Fibre**
14–18 yr	27 g/day	
19–30 yr	30 g/day	
31–50 yr	30 g/day	

Rationale: There is no evidence for increased metabolic needs in lactation. The AI is increased in relation to additional energy needs of about 20%, with rounding.

UPPER LEVEL OF INTAKE - DIETARY FIBRE

There is no UL set for dietary fibre.

Rationale: A number of potential adverse effects have been identified for high intakes of dietary fibre. Potential adverse effects on mineral and vitamin bioavailability were first identified in McCance & Widdowson (1942). However, Gordon et al (1995) stated in a review of the literature: 'We are of the strong conviction and can find no convincing scientific evidence that any dietary fibre, even when consumed in large amounts (ie 50 g total dietary fibre per day), has or should have any adverse effect on mineral absorption or nutrition in humans.'

There are three other potential adverse effects of diets high in dietary fibre. The first relates to the potential increase in the incidental intake of pesticides and other agricultural chemicals, heavy metals, nitrates and antinutrients such as lectins, haemagglutinins and solanine (National Research Council 1989) associated particularly with consumption of the bran layer or skins of plants. The second is the possibility of the development of food intolerances due to alteration of gut microflora (British Nutrition Foundation, 1990). Thirdly, diets with a high content of leafy vegetables may cause problems with benzoar formation in people with upper gastrointestinal dysfunction (Vinik & Jenkins 1988). However, in practice, these potential adverse effects are not likely to cause problems at the levels of recommended intake if dietary fibre is derived from a variety of sources.

Dietary fibre is variable in composition, so it is difficult to link a specific fibre with a particular adverse outcome, especially if phytate is present. A high intake of dietary fibre will not produce substantial deleterious effects when part of a healthy diet, so no upper level of intake is set.

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