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PREFACE

This document is the draft version of the Australian Dietary Guidelines. After Public Consultation further revisions will be made. This document should only be used for discussion up until August 2001 when it should be discarded.

For more than 75 years the Australian Government has been providing nutrition advice. For the past two decades the National Health and Medical Research Council (NHMRC) has undertaken the role of developing and disseminating public health guidelines providing dietary advice for Australians. This document is the third revision of the dietary guidelines for adults. (A separate draft document has been produced containing the second revision of the Children and Adolescents Dietary Guidelines and Infant Feeding Guidelines). The dietary guidelines seek to promote the potential benefits of healthy eating, not only to reduce the risk of development of diet-related diseases but also to improve the community’s health and wellbeing.

The Australian Food and Nutrition Policy published in 1992 aimed to improve the health and reduce the preventable burden of diet-related early death, illness and disability among Australians, through strategies which support the Australian Dietary Guideline. Current estimates of the economic cost to the nation of the principal diet-related condition (ie. cardiovascular disease, stroke and cancer) amounts to about $6 billion per annum, which clearly reflects the magnitude of the potential economic benefit which could accrue to Australia from an effective nutrition-based preventive strategy.

However, as noted in the Australian Food and Nutrition policy, the food system must be both economically viable and maintain the quality and integrity of the environment. Major issues in this context are identified to include conservation of scarce resources such as top soil, water and fossil energy, and amelioration of land degradation associated with salinity, deforestation and chemical contamination. Further important considerations in this regard have been noted in the report on Food for Health by the Nutrition Taskforce to the New Zealand Department of Health. They include the shift in consumer demand for foods which are lower in fat and fresher and the recent restructuring of the food industry from a protected industry to an open, competitive market. This in turn has led to a concentration of ownership in this sector, thereby facilitating access to the industry by policy makers but greatly enhancing the industries’ control over the food supply and pricing.
The Dietary Guidelines for Australians are aimed at healthy, independent Australians. This book provides the scientific rationale for the guideline and is designed to be read by health professionals. Other documents will be developed in a format more suitable for consumers. The guidelines may also be of assistance to health professionals who wish to develop suitable diets for adults with a range of different health circumstances. It must always be remembered that these guidelines are for healthy people and may not meet the specific nutritional requirements of various disease states.

With this edition, the guidelines have been focused more on food groups and lifestyle patterns, moving away from specific nutrients. In particular the references back to the Australian Guide to Healthy Eating (AGHE) will make it easier for consumers and nutrition educators to implement the guidelines. The AGHE is not the only food guide in use in Australia and the working party recognises the potential to use other suitable guides to promote diets consistent with these guidelines.

The guidelines apply to the total diet: they should not be used to assess the ‘healthiness’ of individual food items, nor should individual guidelines be considered in isolation. The first guideline creates a positive setting for nutrition and reflects the fact that good nutritious food is one of the great pleasures of life. The sections of this guideline detail the relationships between different food groups as part of the total diet. Other guidelines, deal with aspects of nutrition where more care is needed and other further aspects of nutrition and a healthy lifestyle.

The guidelines are not ranked in order of importance; they form a consistent package when taken together. Detailed information about requirements for specific nutrients in the Australian diet is contained in the NHMRC publication entitled Recommended Dietary Intakes for Use in Australia. The recommended dietary intakes and the dietary guidelines complement each other in providing comprehensive nutrition advice for the Australian community.

The development of Dietary Guidelines involves extensive consultation with the Australian community, the food industry and with relevant experts. The guidelines are based on the best evidence available, although the Working Party recognises that in some cases the evidence is not complete. (See Appendix A and B for a discussion of Evidence in Nutrition). Where this is the case, the guidance provided is the best available and is made with the safety and health of our community as our foremost concern. The guidelines are a distillation of current knowledge about the relationship between diet and disease, the nutrients available in the Australian food supply, and the contribution that diet can make to optimising the quality of life and reducing morbidity and mortality of Australians.

Each guideline is supported by background information prepared by members of the Working Party established to develop the guidelines, with some additional assistance as detailed on page 6.

Dr Katrine Baghurst, CSIRO Health Sciences and Nutrition and Professor
Colin Binns, School of Public Health at Curtin University of Technology, chaired the Working Party.

Following the period of public consultation all submissions received will be considered by the Working Party and the final document will be edited by Prof Binns and Dr Baghurst. The document will then be presented to the full NHMRC Council for their endorsement. Until the formal endorsement process is complete, this document must be regarded as a draft for discussion purposes only.

Katrine Baghurst and Colin Binns, July 2001
WORKING PARTY TO REVIEW THE DIETARY GUIDELINES

The guidelines were developed in accordance with NHMRC procedures for developing guidelines and in keeping with the following Terms of Reference established by the NHMRC.

Terms of Reference

• Undertake a review of the Dietary Guidelines for Australians (NHMRC 1992) and the Dietary Guidelines for Children and Adolescents (NHMRC 1995) and other related NHMRC dietary guidelines as identified

• Undertake broad consultation to develop a suite of resources for both sets of guidelines including:
  - comprehensive scientific background papers explaining the rationale for each guideline; and
  - appropriate consumer resources

• Produce a Dissemination and Evaluation Plan for both sets of Guidelines

• Report to the Health Advisory Committee

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In addition to the documents published here:

Ms P Crotty provided a working paper on social aspects of Dietary Guidelines to assist the working party; and

Ms Leanne Lester helped with statistical analysis of the results of the 1995 National Nutrition Survey.
THE CONSULTATION PROCESS

The development of the draft dietary guidelines has involved consultation with the Australian community and with experts working in the fields of public health and nutrition.

Preliminary consultation took place from December 2000 – May 2001 and involved the following:

- The analysis of 104 questionnaires completed and returned about the content and use of the current version of the NHMRC’s Adult Dietary Guidelines;
- Establishment of an interactive website containing information about the Review of the Dietary Guidelines; and
- Several meetings with stakeholders.

The current public consultation process will commence in early July until mid August, allowing approximately 6 weeks for consideration of the draft Adult Dietary Guideline and lodgement of submissions.

Notification of this public consultation process will be published in the Commonwealth Government Gazette and on the NHMRC website. Copies of draft documents and supporting information will be available free of charge from the Office of NHMRC and on the NHMRC website. In addition, notices will be included in other publications and media such as newspapers and radio and will be circulated to bodies that are expected to be interested.

All submissions will be acknowledged on receipt and considered by the Working Party.

Following consideration of all submissions, the Dietary Guidelines will be re-drafted. An independent expert Panel will then be established to consider the revised draft.
DIETARY GUIDELINES FOR AUSTRALIANS

1. Enjoy a wide variety of nutritious foods
   
   • Eat plenty of vegetables (including legumes) and fruits
   
   • Eat plenty of cereals (including breads, rice, pasta, noodles), preferably wholegrain
   
   • Include lean meat, fish, poultry and/or alternatives such as legumes and nuts
   
   • Include reduced fat dairy foods and/or alternatives
   
   • Drink plenty of water

2. Take Care to:
   
   • Limit saturated fat and moderate total fat intake
   
   • Choose foods low in salt
   
   • Limit your alcohol intake if you choose to drink
   
   • Consume only moderate amounts of sugars and foods containing added sugars

3. Prevent weight gain by being physically active and eating according to your needs

4. Care for your food and keep it safe to eat

5. Encourage and support breastfeeding
ENJOY A WIDE VARIETY OF NUTRITIOUS FOODS

The nutrients that are essential for human life are found in varying amounts in many different foods. A varied diet is essential to obtain sufficient quantities of all required nutrients (known and not yet known), for increasing the consumption of protective factors (phytochemicals) and for minimising exposure to toxicants.

Australians today enjoy a wide variety of foods, relatively independent of season and location, and can choose from a number of cuisines. The available food supply is adequate to meet the nutritional needs of Australians, but appropriate choices must be made so that all nutrient requirements are met (1). There are also disadvantaged groups within Australia, where for reasons, including poverty, incorrect food beliefs, distance or disability, special efforts are needed to ensure an adequate diet. Australia is also fortunate in having a food supply that is relatively free of contaminants and pollutants, as shown by the Australian Total Diet Survey (2).

DEFINITIONS

Variety
Food variety can be defined as the consumption of foods that are biologically diverse or nutritionally distinct from each other. Variety means a mixture of foods across the range of food types as illustrated in the Australian Guide to Healthy Eating (AGHE) (3). ‘Variety’ further refers to choosing a range of foods from within each food group.

Nutritious
The term ‘nutritious foods’ is used to describe foods which make a substantial contribution towards providing a range of nutrients, have an appropriate nutrient density, and are compatible with the overall aims of the Dietary Guidelines for Australians (4).

THE IMPORTANCE OF FOOD VARIETY

Consuming a wide variety of foods in the diet offers a degree of protection against non-communicable chronic diseases such as vascular disease, obesity, diabetes, and possibly even cancer, although recent work suggests that the situation with obesity is not straightforward (5). The benefits are gained by reducing the intake of foods that supply excessive amounts of fat, salt and alcohol and by maximising the intake of protective factors such as vegetables, fruits, cereals (see Chapters on Vegetables and Fruit and Cereals). Eating a varied diet also increases the possibility of receiving essential nutrients in adequate amounts (6).

Plants contain various toxic substances that, although often useful for discouraging insects and other predators, have the potential to harm humans. Minimising the risk posed by naturally occurring toxicants is a useful goal of public health policy (7). Consuming a wide variety of nutritious foods minimises the intake of toxic components and not only protects against the accumulation of toxic substances and the development of nutrition-related diseases, but also works to retard the processes responsible for the physiological decline associated with ageing (7).
Historically, until fire was first used only raw foods could have been eaten. The ability to cook must have immensely increased the safety and availability of these foods by destroying thermolabile poisons in otherwise edible plants as well as the parasites and toxins common in flesh and carrion (8). Now a large number of processing and storage methods are used to reduce any toxicity problems of foods.

**SCIENTIFIC BASIS**

**The 1995 National Nutrition Survey**

The National Nutrition Survey (NNS) was conducted between February 1995 and March 1996, as an adjunct to the 1995 National Health Survey (9). The dietary intakes of approximately 13,800 people aged 2 years or older, from urban and rural areas throughout all States and Territories were recorded. Additional information on physical measurements and eating habits and patterns was also collected. Data from the NNS showed that during the 12 years since the previous survey food variety in Australia increased significantly, with a much greater number of foods being recorded than in 1983. The increase in the variety of foods available reflects the wide range of fresh, processed, mixed or prepared food forms that are now conveniently obtainable in Australia on a daily basis.

The expansion in the number of foods available in Australia is largely due to the ethnic diversity that now characterises our population. The influx of European immigrants after World War II and the migration of Asian people in more recent decades have led to the development of an Australian population consuming a wide variety of cuisines, in place of the ‘traditional’ Anglo-Celtic foods. Few countries have such ready access to a variety of cuisines from different cultures. On 30 June 1999 there were approximately 4.5 million overseas-born living in Australia, around 24 per cent of the population. Twenty-seven per cent of overseas-born people were originally from the United Kingdom and Ireland; 5 per cent were from Italy, 3 per cent were from Greece, and 3 per cent were from Germany. The number of immigrants from Asia is increasing and the Vietnamese community is now second in numbers after the English (10). The ready availability of different cuisines does, however, enable most Australians, at least those in urban areas and larger regional centres, to experiment with foods not common in the everyday diet, thus increasing the opportunity for expanding their food variety.

The NNS data shows that generally, older Australians consume a diet that is more varied. Males who live alone eat significantly fewer food groups.

In the 1995 NNS the foods eaten were classified into 14 different groups. As the following graph illustrates, in virtually all age groups, males who live alone eat a significantly fewer number of food groups each day. Figure 1 shows the cumulative frequency of the ABS food groups. (See Appendix C for further graphs relating to specific groups).
Dietary Variety and Chronic Disease

Research that illustrates the health benefits of increasing food variety in a diet comes from the United States (US) National Health and Nutrition Examination Survey Epidemiologic Follow-up Study. Using a dietary diversity score of 0 to 5, with 5 being the maximum possible score and indicating high food variety, increased risk of mortality was associated with a low dietary diversity score at nearly every level of age, income, education, race, smoking status and fibre intake. There was also an increased risk of mortality from all causes for both men and women in dietary patterns where a food group was omitted from the diet. In this study of 4 160 men and 6 264 women, less than 5 per cent reported omitting foods from the meat or grain groups on the survey day, whereas 46 per cent reported no fruit, 25 per cent reported no dairy products, and 17 per cent reported no vegetables. Not reporting consumption of fruits and vegetables was associated with low serum vitamin C, whereas reporting fruit and vegetable consumption was associated with a high vitamin C concentration (11).
In a prospective US study, Kant et al (12), evaluated the association between dietary quality (based on dietary guidelines) and mortality in women. A total of 42,254 women (mean age 61.1 years) completed the food frequency questionnaire and were followed up for an average of 5.6 years. The results showed that women who reported dietary patterns that included fruits, vegetables, whole grains, low-fat dairy and lean meats as recommended by the dietary guidelines had a lower risk of mortality. The data from this study suggested that a dietary pattern characterised by consumption of foods recommended in current dietary guidelines is associated with decreased risk of mortality in women.

Australians are in a position to include in their diet, cuisines from various cultures, that add variety to the traditional Australian diet and that have been associated with health gains. Mennell et al (13) refer to the varying cuisines of the world as ‘culinary culture’ and define this as ‘the ensemble of attitudes and tastes people bring to cooking and eating’. Ecological comparisons of cuisines and health outcomes have suggested that the Japanese diet (in particular the Okinawan) and the “Mediterranean” diets may show benefits in decreasing mortality from chronic diseases and an increased life expectancy in countries where these diets predominate (14). A study of Greek populations in Melbourne and in Greece and has concluded that food variety was an important determinant of morbidity and mortality (15). Traditional Mediterranean diets are based on plant foods, contain small amounts of animal foods, use olive oil as the principal fat, contain moderate amounts of alcohol, and balance energy intake with energy expenditure. They are low in saturated fat and high in the protective compounds found throughout a variety of plant foods (16) (17). The diet of Okinawa is varied, with a substantial amount of fish and is associated with the longest reported life spans of any population (18).

**Dietary Variety and Nutrient Intake**

A study of Chinese migrants living in Melbourne showed that when the diet failed to provide more than 40 per cent of the maximum achievable variety (over 12 months), study participants were far more likely to have at least one nutrient level fall below two-thirds of the Australian recommended dietary intakes (See Appendix D in the Dietary Guidelines for Older Australians for a method of assessing food variety). Interestingly, food variety in the diet of these migrants increased with length of stay in Australia, independent of age, which suggests that food variety can be increased if there is continued exposure to new foods (5).

Recommending to Australians that they ‘enjoy a wide variety of nutritious foods’ will not only help ensure appropriate intakes of major dietary components such as protein, carbohydrates and fats but also ensure adequate and appropriate intakes of vitamins and minerals, individual fatty acids and amino acids.

The term ‘vitamin’ was first introduced in 1912 by Funk when he suggested that conditions such as scurvy, beri beri, rickets and pellagra were due to deficiencies in the diet (19). The isolation and identification of the first vitamin essential to health and for which biological activity had been established was thiamin in 1926 by Jansen and Donath (20). For many of the subsequently identified vitamins, more than one chemically identifiable constituent has been shown to provide the important physiological activity. Since then the essential nature of many food constituents has been identified including
vitamins, minerals and trace elements, specific fatty acids and amino acids as well as energy and overall protein requirements. These have been specified and quantified to the extent that ‘Recommended Dietary Intakes’ for Australians of various ages, have been established (21). While nutritional science has made considerable advances to date, research into further food constituents essential or beneficial to human health is still required as is ongoing revision of recommendations for intake.

While eating a wide variety of foods will maximise the potential benefits of the biological diversity of foods, particularly of plant foods, nutrition is complex and over consumption can be potentially as big a problem as deficiency. Excessive intakes of essential nutrients usually cause only minor problems, but in some rare cases can be potentially fatal. Excessive nutrient intakes that cause significant problems are nearly always related to intakes in the form of supplements, although it is certainly possible to develop symptoms of toxicity with very unbalanced diets. This even applies to different chemical forms of the ‘same’ nutrient. Some examples include:

- Excessive intakes of the pro-vitamin form of vitamin A, carotene, will result in a yellow or orange skin colour, while excessive intakes of the preformed vitamin A, retinol, will lead to headache, vomiting, extensive skin peeling, bone abnormalities and liver damage (22). While Vitamin A is essential for child survival the consumption excessive amounts as vitamin A supplements during pregnancy may result in serious birth defects.

- Excessive intakes of niacin in the form of nicotinic acid may result in flushing, hyperglycaemia and abnormalities of liver function (23).

- Excessive intake of vitamin C has been associated with nausea, vomiting and diarrhoea [Jacob 1999 - cited in (24)]. High intakes during pregnancy have been associated with rebound scurvy, due to vitamin C deficiency, in the newborn infant [Johnston 1999 and Jacob 1999 - cited in (24)].

- Excessive intakes of pyridoxine (one of the chemical forms of vitamin B-6) have been reported to result in peripheral neuropathy (25) (26).

- Excessive intake of vitamin D can lead to hypercalcaemia, dehydration and calcification of soft tissue including kidney failure (27); and

- Acute excessive intake of iron can result in vomiting and gastrointestinal bleeding (28). Chronic excessive consumption can lead to haemosiderosis with liver damage.

There are only limited examples of the consumption of foods causing problems such as those listed above, without the concurrent consumption of dietary supplements. But a diet limited in the range of foods consumed and with excessive, long term consumption of a particular food can potentially cause problems. Examples include the high and prolonged consumption of carrot juice (which will result in excess beta-carotene intake) or eating very large serves of liver which may cause vitamin A poisoning. It is impossible to consume nutrients to the excessive levels necessary for these effects through the consumption of a varied, nutritious and healthy diet consistent with the Australian Guide to Healthy Eating (3).
The average intakes (as recorded in the National Nutrition Survey of 1995/96) of selected nutrients are shown in Tables 1 A & B. Despite our varied diet in Australia certain nutrients are still at risk, including iron in premenopausal women and calcium.

**Table One A  Mean nutrient intakes of males**  
**National Nutrition Survey, 1995**

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>16-18</th>
<th>19-24</th>
<th>25-44</th>
<th>45-64</th>
<th>over</th>
<th>19 &amp; over</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kj)</td>
<td>13,525</td>
<td>13,275</td>
<td>11,725</td>
<td>10,296</td>
<td>8510</td>
<td>11,049</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>120.0</td>
<td>127.7</td>
<td>115.2</td>
<td>104.5</td>
<td>83.7</td>
<td>109.2</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>119.6</td>
<td>119.1</td>
<td>105.6</td>
<td>90.6</td>
<td>74.0</td>
<td>98.5</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>409.4</td>
<td>375.9</td>
<td>316.8</td>
<td>274.3</td>
<td>235.1</td>
<td>300.5</td>
</tr>
<tr>
<td>Fibre (g)</td>
<td>26.5</td>
<td>26.2</td>
<td>26.1</td>
<td>26.3</td>
<td>24.0</td>
<td>25.9</td>
</tr>
<tr>
<td>Alcohol (g)*</td>
<td>9.1</td>
<td>15.2</td>
<td>19.7</td>
<td>20.2</td>
<td>14.7</td>
<td>18.5</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>1,186</td>
<td>1,233</td>
<td>1,306</td>
<td>1,360</td>
<td>1,301</td>
<td>1,311</td>
</tr>
<tr>
<td>Thiamin (mg)</td>
<td>2.3</td>
<td>2.3</td>
<td>2.1</td>
<td>1.8</td>
<td>1.6</td>
<td>1.9</td>
</tr>
<tr>
<td>Niacin Equivalent (mg)</td>
<td>53.5</td>
<td>57.6</td>
<td>53.9</td>
<td>48.8</td>
<td>38.8</td>
<td>50.7</td>
</tr>
<tr>
<td>Folate (mcg)</td>
<td>312.7</td>
<td>321.8</td>
<td>310.6</td>
<td>309.3</td>
<td>276.6</td>
<td>306.8</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>153.8</td>
<td>149.6</td>
<td>132.6</td>
<td>137.7</td>
<td>127.1</td>
<td>135.6</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>1,280.01101.19</td>
<td>88.6</td>
<td>885.3</td>
<td>795.6</td>
<td>945.5</td>
<td></td>
</tr>
<tr>
<td>Phosphorus (mg)</td>
<td>2,065.9</td>
<td>2,051.5</td>
<td>1,866.7</td>
<td>1,691.9</td>
<td>1,419.1</td>
<td>775.6</td>
</tr>
<tr>
<td>Magnesium (mg)</td>
<td>379.6</td>
<td>390.1</td>
<td>392.5</td>
<td>383.3</td>
<td>334.2</td>
<td>381.1</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>17.9</td>
<td>17.9</td>
<td>16.7</td>
<td>16.2</td>
<td>14.4</td>
<td>16.4</td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>14.8</td>
<td>17.3</td>
<td>14.9</td>
<td>14.0</td>
<td>11.4</td>
<td>14.4</td>
</tr>
<tr>
<td>Potassium (mg)</td>
<td>4,065.2</td>
<td>3,943.0</td>
<td>3,818.3</td>
<td>3,732.8</td>
<td>3,232.0</td>
<td>3,725.2</td>
</tr>
</tbody>
</table>

* represents pure alcohol
Table One B  Mean nutrient intakes of Females  
National Nutrition Survey, 1995

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>16-18</th>
<th>19-24</th>
<th>25-44</th>
<th>45-64</th>
<th>65 &amp; over</th>
<th>19 and over</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kj)</td>
<td>8,690.4</td>
<td>8,369.6</td>
<td>7,875.2</td>
<td>7,220.6</td>
<td>6,367.0</td>
<td>7,480.9</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>80.3</td>
<td>78.4</td>
<td>76.2</td>
<td>74.6</td>
<td>64.3</td>
<td>73.9</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>76.4</td>
<td>75.4</td>
<td>72.0</td>
<td>64.4</td>
<td>56.9</td>
<td>67.6</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>263.6</td>
<td>243.4</td>
<td>220.3</td>
<td>199.8</td>
<td>182.1</td>
<td>210.6</td>
</tr>
<tr>
<td>Fibre (g)</td>
<td>19.4</td>
<td>19.2</td>
<td>20.0</td>
<td>21.5</td>
<td>20.2</td>
<td>20.3</td>
</tr>
<tr>
<td>Alcohol (g)*</td>
<td>3.9</td>
<td>6.6</td>
<td>8.2</td>
<td>8.0</td>
<td>4.6</td>
<td>7.3</td>
</tr>
<tr>
<td>Vitamin A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retinol Equivalent (mcg)</td>
<td>877.3</td>
<td>889.1</td>
<td>1,024.4</td>
<td>1,145.1</td>
<td>1,058.6</td>
<td>1,047.2</td>
</tr>
<tr>
<td>Thiamin (mg)</td>
<td>1.5</td>
<td>1.5</td>
<td>1.4</td>
<td>1.3</td>
<td>1.2</td>
<td>1.4</td>
</tr>
<tr>
<td>Niacin Equivalent (mg)</td>
<td>35.3</td>
<td>36.1</td>
<td>35.3</td>
<td>34.5</td>
<td>29.4</td>
<td>34.1</td>
</tr>
<tr>
<td>Folate (mcg)</td>
<td>216.7</td>
<td>232.9</td>
<td>227.0</td>
<td>246.9</td>
<td>224.8</td>
<td>232.8</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>125.6</td>
<td>119.8</td>
<td>108.5</td>
<td>118.1</td>
<td>111.5</td>
<td>113.1</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>801.3</td>
<td>750.0</td>
<td>762.1</td>
<td>769.2</td>
<td>685.6</td>
<td>746.6</td>
</tr>
<tr>
<td>Phosphorus (mg)</td>
<td>1,336.6</td>
<td>1,331.8</td>
<td>1,299.8</td>
<td>1,294.7</td>
<td>1,131.7</td>
<td>1,271.7</td>
</tr>
<tr>
<td>Magnesium (mg)</td>
<td>257.3</td>
<td>272.5</td>
<td>283.6</td>
<td>297.1</td>
<td>267.9</td>
<td>283.1</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>11.1</td>
<td>11.9</td>
<td>12.0</td>
<td>12.3</td>
<td>11.3</td>
<td>11.9</td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>10.0</td>
<td>10.2</td>
<td>9.9</td>
<td>9.8</td>
<td>9.0</td>
<td>9.7</td>
</tr>
<tr>
<td>Potassium (mg)</td>
<td>2,673.8</td>
<td>2,752.4</td>
<td>2,816.3</td>
<td>2,929.7</td>
<td>2,626.0</td>
<td>2,805.0</td>
</tr>
</tbody>
</table>

* represents pure alcohol

Source (29) (21)

**Nutrient interactions**

There are many complex relationships between foods and nutrients, which can mutually influence the absorption, metabolism and retention of other nutrients. When the diet is well balanced and nutrients are in adequate supply such interactions pose few problems, but when the intake of some nutrients is habitually low, excesses of others can have detrimental consequences (30).

The following are examples of interactions:

A common interaction is that between sodium, protein and calcium. Sodium and calcium compete for the same transport mechanism in the kidney, and an excess of one will cause excretion of the other. Protein has a similar effect on urine calcium levels. This interaction is important in the older population because factors that affect urinary calcium loss are likely to affect bone health: recognition of this interaction allows for the prevention of calcium losses related to high protein intakes. When diets are high in protein, a reduction in sodium intake can reduce the physiological need for calcium (31) and so improve calcium nutrition (see Salt Chapter). Other inhibitors of calcium absorption are phosphates in cow’s milk, phytic acid from the husks of cereals, and oxalic acid in spinach and rhubarb, which form insoluble complexes with calcium (32).
The positive impact of vitamin C containing foods such as citrus fruit and of meats on the absorption of iron from plant foods when eaten at the same meal (28) (33).

Iron is also susceptible to nutrient interactions. Non-haem iron absorption is inhibited by phytates, polyphenols (for example, tannins) and calcium (34). Consuming animal sources of iron, such as meat, which is also high in protein, will promote iron absorption (35).

Zinc bioavailability is also affected by phytate found in cereal products. Zinc found in animal products, crustaceans and molluscs is more readily absorbed than zinc found in plant foods. Legumes and unrefined cereals contain phytates that reduce zinc absorption. The zinc content of refined cereals is lower than that of unrefined cereals but, because a large part of the phytic acid present in cereals is removed during the refining process, zinc bioavailability is increased. Phytate in the presence of calcium may also reduce zinc bioavailability (36). By including adequate amounts of wholegrain products and legumes, in a varied diet, lacto-ovo vegetarians can meet their zinc requirements and maintain zinc balance (37). In most cases eating a varied diet protects against these interaction effects.

It must also be remembered that in storing, preparing, cooking and processing foods, losses of some nutrients occur, while in other cases absorption or availability is improved by processing. Riboflavin, for example, will be destroyed by exposure to ultra violet light; ascorbic acid by exposure to oxygen (air); B-carotene, thiamin, and ascorbic acid by heating; and minerals will be leached out of foods soaked in water. In some cases the chemical form of a nutrient used for fortification may be less available than the natural product (e.g. iron added to breakfast cereal compared to haem iron) or in other cases may be more active (e.g. folic acid compared to naturally occurring folates).

**PRACTICAL ASPECTS OF THIS GUIDELINE**

**Too much of a good thing**

As variety in a diet increases, it is important to reduce the amounts of each food eaten to avoid over-consumption of energy (to avoid obesity). There have been a number of short-term studies and McCrory et al (38) have reported a long-term study to determine whether dietary variety within food groups influences energy intake and body fatness. Their data suggests that a high variety of sweets, snacks, condiments, entrees and carbohydrates coupled with a low variety of vegetables promotes long-term increases in energy intake and body fatness. Dietary variety was positively associated with energy intake within each of the 10 food groups. They suggest that these findings may help explain the rising prevalence of obesity. In another study the same group found that an increased number of meals eaten in restaurants was also associated with obesity.

**Food Contaminants**

Eating a variety of foods dilutes the naturally occurring toxicants and any added contaminants. As noted, the Australian food supply is one of the safest and cleanest in the world, ensuring that a minimum of toxicants are ingested.
The 19th Australian Total Diet Survey has found that levels of pesticide residues and heavy metal contaminants in the Australian food supply remain very low and well within safety standards set by Australian and international health authorities. (44)

**Phytochemicals**

Variety in the diet is becoming increasingly important as the emphasis on non-nutrients increases. Foods have traditionally been classified according to their macronutrient and micronutrient value, but now their non-nutrient value is gaining recognition in terms of food’s role in chronic non-communicable diseases. Most non-nutrient factors are phytochemicals that are not directly associated with deficiency syndromes but do have some relationship to optimal health. Phytochemicals may be multi-functional; alternatively, a particular function may be provided by more than one class of phytochemicals. Interactions between compounds are likely to be complex and deep, causing a masking or synergy of effects (39).

Phytochemicals can fall into one of a number of chemical categories: carotenoids, flavonoids and isoflavonoids, polyphenols, isothiocyanates, indoles, sulphoraphane, monoterpenes, xanthin, and non-digestible oligosaccharides. Variety in the diet is recommended so that the protective benefits of nutrients and non-nutrients can be obtained: it is not known exactly which food constituents are responsible for the protective effect against chronic diseases. With the exception of breast milk in the first six months of life, no single nutritious food can provide a complete and healthy diet. A diet containing a wide range of foods from the different food groups is most likely to offer protection against non-communicable chronic diseases (39). (See also Fruit and Vegetables chapter)

**RELATIONSHIP TO OTHER GUIDELINES**

Variety means that we choose to eat a mixture of foods across the range of food types as illustrated in the Australian Guide to Healthy Eating (AGHE, page 20). ‘Variety’ further refers to choosing a range of foods from within each food type. Across food types - cereal, fruit, vegetable, meat, dairy - there is a varying amount, availability and chemical form of the essential and beneficial constituents of foods. For example, meats and cereal foods are the major sources of iron in the Australian diet, while fruits and dairy products are very minor sources (29).

The nutritious and healthy diet will have as its central and essential core foods from the main “plate” of the AGHE (3).

**Prevent weight gain by being physically active and eating according to your needs**

Activity equals higher energy intake and scope for eating a wider variety of foods. The work of McCrory and Coulston (41) highlights the risks of a varied diet. While variety is an important nutritional principle, with the evolution of modern sedentary society, if variety is to be maintained, food serve sizes must be reduced. Consumer education that focuses on reduced portion sizes may help reduce opportunities for overeating. The food and restaurant industry need to take care with serving sizes, especially of high-energy-density foods (42).
Eat plenty of vegetables (including legumes) and fruit
Adopting a varied diet means that a wide choice of vegetables and fruits can be enjoyed, in addition to those regularly eaten; for example, having stewed fruit on porridge and other breakfast cereals and adding fruit to salads. Variety also means variety within the vegetable and fruit groups. Vegetables should include green leafy varieties, and red and yellow and starchy vegetables. Fruits should include those high in vitamin C and those high in vitamin A (and its analogues).

Preferable forms are those without added fats, refined sugars and (added) salt, and including, where possible, the edible skins and seeds. These foods form a major part of a healthy diet in terms of quantity, weight, volume and number of serves of food. They provide the desirable bulk and satiety of the diet together with essential vitamins and minerals. In themselves, these foods are not high in fat or energy.

Eat plenty of cereals (including breads, rice, pasta, noodles) preferably wholegrain:
Choose from a wide range of cereal-based products from different cuisines, including wholemeal, wholegrain and fibre-enriched products such as bagels, pita bread and pumpernickel.

The wholegrain forms of these foods without added fats, refined sugars and salt are preferable. For foods such as breads where the commonly available forms contain added salt, reduced salt forms are available. Similarly, with foods such as bread that are commonly eaten with a fat spread, the type (ie of fat and amount of added salt) of spread should be considered and the quantity of the spread used should be limited. While fat spreads (butter and margarine) are useful sources of vitamin A and vitamin D, they are virtually all fat and hence of high energy density.

Include lean meat, fish, poultry and/or alternatives such as legumes and nuts
Choose from a variety of these foods. Red meat should be included three to four times a week or other good sources of bioavailable iron will need to be included. Fish should be included regularly to provide omega-3 fats.

The lean muscle of animal meats, trimmed of visible fat or skin are relatively low in fat. Forms of these foods cooked with no or little fat and eaten without sauces that are high in added fat or salt are preferable. These foods are valuable sources of protein, key vitamins and nutrients and, in fish, omega-3 fatty acids.

Include reduced fat dairy foods and/or alternatives
Reduced fat forms of these foods are generally preferable, but not for infants and young children. The diet may be augmented from time to time with foods from the optional group, for taste and variety.

Increase dietary variety by using high-calcium foods from other countries; for example, Parmesan cheese and some varieties of tofu (Note that calcium content of tofu, cheeses and low fat dairy products can vary and the labels will need to be checked).
**Drink Plenty of Water**
An important part of a varied diet is the water content, as drinks or contained within food. The average requirement for an adult is around eight glasses per day. Water is the preferred drink, as it contains no additional energy. For infants and older persons, the risks of actual dehydration are greater (see Dietary Guidelines for Older Australians). For those who undertake strenuous work in hot climates, water requirements increase dramatically (40).

**Limit saturated fat and moderate total fat intake**
A diet that is rich in variety will contain foods from many different sources and be less concentrated in sources of fat.

**Choose foods low in salt**
Inclusion in the diet of foods from a variety of sources will displace the number of foods that are high in salt. Experimenting with different types of foods will introduce new flavourings to the diet.

**Consume only moderate amounts of sugars and foods containing added sugars**
Foods that are naturally sweet are more likely to be consumed in a varied diet because many different foods types are chosen, not just those that are high in added sugars.

**CONCLUSION**
Enjoying a variety of nutritious foods remains an important message for all age groups. Making mealtimes a social occasion, experimenting with other cuisines, and incorporating new and traditional foods will encourage variety in the diet, meet nutrient requirements and provide some protection against chronic diseases.

**EVIDENCE**
Evidence about the importance of variety to gain sufficient nutrients is available at Level III (refs 11,12,16,38) and at Level IV (refs 5 and 6). Evidence was also provided from cross-cultural observational studies of diet, health and longevity.
AUSTRALIAN GUIDE TO HEALTHY EATING

Enjoy a variety of foods every day

Vegetables, legumes

Fruit

Bread, cereals, rice, pasta, noodles

Milk, yogurt, cheese

Lean meat, fish, poultry, eggs, nuts, legumes

Drink plenty of water

Choose these sometimes or in small amounts
AUSTRALIAN GUIDE TO HEALTHY EATING

Table 1: Sample serves suggested for children and adolescents

<table>
<thead>
<tr>
<th>Children &amp; Adolescents</th>
<th>Cereals (including breads, rice, pasta, noodles)</th>
<th>Vegetables (including legumes) and Fruit</th>
<th>Reduced Fat Dairy and/or alternatives</th>
<th>Lean meat, fish, poultry and/or alternatives such as legumes &amp; nuts</th>
<th>Extra Foods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children 4-7 years</td>
<td>5-7</td>
<td>3</td>
<td>2</td>
<td>½ - 1</td>
<td>1-2</td>
</tr>
<tr>
<td>8-11 years</td>
<td>6-9</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>1-2</td>
</tr>
<tr>
<td>12-18 years</td>
<td>5-11</td>
<td>7</td>
<td>3</td>
<td>1</td>
<td>1-3</td>
</tr>
</tbody>
</table>

Table 2: Sample serves suggested for women

<table>
<thead>
<tr>
<th>Women</th>
<th>Cereals (including breads, rice, pasta, noodles)</th>
<th>Vegetables (including legumes) and Fruit</th>
<th>Reduced Fat Dairy and/or alternatives</th>
<th>Lean meat, fish, poultry and/or alternatives such as legumes &amp; nuts</th>
<th>Extra Foods</th>
</tr>
</thead>
<tbody>
<tr>
<td>19-60 years</td>
<td>4-9</td>
<td>4-6</td>
<td>7</td>
<td>1</td>
<td>0-2 ½</td>
</tr>
<tr>
<td>Pregnant</td>
<td>4-6</td>
<td>6-10</td>
<td>2</td>
<td>1 ½</td>
<td>0-2 ½</td>
</tr>
<tr>
<td>Breastfeeding</td>
<td>5-7</td>
<td>12</td>
<td>2</td>
<td>2</td>
<td>0-2 ½</td>
</tr>
<tr>
<td>60+ years</td>
<td>4-7</td>
<td>6-9</td>
<td>2</td>
<td>1-1 ½</td>
<td>0-2</td>
</tr>
</tbody>
</table>

Table 3: Sample serves suggested for men

<table>
<thead>
<tr>
<th>Men</th>
<th>Cereals (including breads, rice, pasta, noodles)</th>
<th>Vegetables (including legumes) and Fruit</th>
<th>Reduced Fat Dairy and/or alternatives</th>
<th>Lean meat, fish, poultry and/or alternatives such as legumes &amp; nuts</th>
<th>Extra Foods</th>
</tr>
</thead>
<tbody>
<tr>
<td>19-60 years</td>
<td>6-12</td>
<td>7</td>
<td>2</td>
<td>1</td>
<td>0-3</td>
</tr>
<tr>
<td>60+ years</td>
<td>4-9</td>
<td>7</td>
<td>2</td>
<td>1-1 ½</td>
<td>0-2 ½</td>
</tr>
</tbody>
</table>

The sample serves allow for two different eating patterns:
- the top rows include a lot of cereals, bread, rice, pasta and noodles; or
- the bottom rows include less cereals etc… and more of the other groups
References

27. Fraser DR. Vitamin D. J Food Nutr 1987;44:3-8.
44. 19th Australian Total Diet Survey, ANZFA April 2001.
EAT PLENTY OF VEGETABLES (INCLUDING LEGUMES) AND FRUIT

DEFINITIONS

Vegetables
This category of plant foods includes all leafy green vegetables (eg. spinach, lettuce, silver-beet, bok choi), members of the Cruciferous family (eg. broccoli, cabbage, brussel sprouts), all root and tuber vegetables (eg. carrots, yams, potatoes), edible plant stems (eg. celery, asparagus), gourd vegetables (eg. pumpkin, cucumber), allium vegetables (eg. onion, garlic, shallot), and corn. Some vegetables are eaten raw as salads, others are best cooked before eating as this makes them more palatable and digestible.

Fruits
The term “fruit” generally applies to the sweet, fleshy, edible portion of a plant which arises from the base of the flower and surrounds the seeds (eg. apples, oranges, plums, berries), tomatoes and avocados. Most fruit is eaten raw, although cooking in some cases may offer a tasty alternative.

Legumes
This term includes all forms of prepared beans and peas (eg. dried legumes, legume flour products, eg. papadams, bean curd, tofu, canned legumes, cooked legumes). Well known edible legumes include butter beans, haricot (navy) beans, red kidney beans, soybeans, mung beans, lentils, chick peas, snow peas and various fresh green peas and beans. Legumes are generally cooked as this improves their nutritional value and reduces the risk of toxicity which occurs with some legumes due to the presence of heat-labile toxins. However, occasionally they may be eaten raw (eg. snow peas). Strictly speaking legumes are specialised forms of fruit as the pod surrounds the seeds and arises from the base of the flower as occurs with fruit. However, because the main food material in legumes is the seeds, they are generally classified in a separate category.

Nuts
Similar to legumes many nuts are actually fruits in which the seed forms the main edible component and the whole structure becomes dry on maturing. As most nuts provide a wide range of nutrients and are generally pleasantly flavoured they can usefully be included with fruits and vegetables in plant based dishes.

NEED FOR GUIDELINE

In Australia, each year approximately 40% of all deaths can be attributed to diseases of the circulatory system and 27% to cancer, with an annual health care cost to the nation of around $4 billion and $2 billion respectively\(^1\). However, scientific surveys carried out on populations around the world have consistently provided good epidemiological evidence that people who regularly eat diets high in fruits and vegetables including legumes, have substantially lower risks of coronary heart disease\(^2\text{-}^4\), stroke\(^2\text{-}^5\), several major cancers\(^6\text{-}^7\) and possibly hypertension\(^8\text{-}^9\), non-insulin dependent diabetes mellitus\(^10\text{-}^11\), cataract\(^12\text{-}^13\) and macular degeneration of the eye\(^14\text{-}^15\). Experimental studies with model systems have
afforded further evidence of a protective effect of fruits and vegetables against these non-communicable degenerative diseases and have provided some clues concerning the actual substances in these foods which may provide this protection as well as the mechanisms by which they may act. Accordingly, a new term “phytochemicals” has been added to the vocabulary of nutritionists which refers to the many different substances occurring in plant foods in small amounts, in addition to the well established nutrients, and which appear to contribute significantly to reducing the risk of non-infectious degenerative diseases.

SCIENTIFIC BASIS

Original and Recent Studies

Cardiovascular disease

In 1997 twenty-eight studies in humans were reviewed in relation to fruit and vegetable consumption and risk of cardiovascular disease and good evidence was found of a protective effect associated with higher intakes of plant foods\(^2\). Some years earlier (1993) the United States of America Food and Drug Administration allowed a “Health Claim” to the effect that diets low in saturated fat and cholesterol and rich in fruits, vegetables and grain products that contain fibre, particularly soluble fibre, may reduce the risk of coronary heart disease\(^16\). A later large study with women has also reported a significant inverse association between fruit and vegetable intake and cardiovascular disease \(^3\). Recent experimental studies suggest that protection against heart disease may arise in several ways, including the presence of antioxidant phytochemicals (e.g. bioflavonoids, carotenoids) and antioxidant vitamins (e.g. vitamins E and C) at significant levels in fruits and vegetables, which may reduce the risk of cholesterol becoming oxidised in coronary blood vessels and deposited to form atheromatous plaques\(^17\). Importantly a review of the effect of β-carotene on coronary heart disease in several observational and intervention studies indicates protection only in the observational studies, which highlights the possibility that the benefit reported in some studies may be related to foods rich in β-carotene and other antioxidants and micronutrients rather than to the β-carotene alone \(^18\). Also important is the apparent capacity of vegetable protein to reduce blood cholesterol levels in people habitually consuming an omnivorous diet\(^19\). Currently, particular emphasis is being focussed on the importance of the vitamin folate, to reduce blood levels of the compound homocysteine which is an established risk factor for coronary heart disease\(^4,20\). Particularly noteworthy is the fact that a major source of dietary folate is from green, leafy vegetables and studies suggest that many adults have folate intakes well below the level needed to minimise the risk associated with raised levels of homocysteine\(^21\).

Stroke

Evaluation of fourteen studies that related to stroke and fruit and vegetables found strong evidence of a protective effect associated with higher intakes of plant foods\(^2\). A mechanism for this apparent protection is not clear but it appears to exist for strokes of both haemorrhagic and ischaemic origin\(^5\). In one large study which extended over 8 years, protection was associated with vegetable intake rather than fruit\(^22\), although generally both types of plant foods are considered to be likely protective agents\(^23\).
Hypertension
Because plant foods contribute significantly to the intake of potassium and magnesium, both of which have been proposed to be associated with a lower blood pressure, diets high in fruits and vegetables will increase the daily intake of both minerals and may help prevent or control hypertension\(^6,24\). In a study with women in the USA, lowered blood pressure was found to be associated with higher intakes of fruits and vegetables, fibre and magnesium\(^8\), and more recently data from the Dietary Approaches to Stop Hypertension (DASH) randomised clinical trial has indicated that diets rich in fruits and vegetables, with or without low-fat dairy products, significantly reduced ambulatory blood pressure after an 8-week intervention period\(^25\), especially in African Americans and those with hypertension\(^26\). Similar results were found with adolescents in the USA with elevated blood pressures where it was found that blood pressure was lower in those subjects with higher intakes of a combination of nutrients including potassium, calcium, magnesium and vitamins as provided by diets rich in fruits and vegetables and low-fat dairy products\(^27\).

Cancer
Health authorities have estimated that at least 30% of many major cancers have a strong dietary link, and this link may be far greater in some cases\(^6\). Dietary factors underlying this association include those substances which may aggravate the development of cancer and, very importantly, those which reduce cancer risk. Dietary components in this latter group include fibre, fruits and vegetables. In fact, the association between fruits and vegetables is sufficiently widely recognised that the US Food and Drug Administration has allowed a health claim to the effect that diets low in fat and rich in fruit and vegetables may reduce the risk of some cancers\(^16\).

Not surprisingly protection by fruit and vegetables has been noted especially in relation to the oral cavity, larynx, oesophagus, stomach and large bowel where the protective effect may involve local contact, although significant risk reduction has also been observed with respect to cancers of the lung and possibly the breast, endometrium and pancreas\(^6,7\). Many putative factors in fruit and vegetables have been proposed to account for their protective effect and many potential mechanisms suggested. Currently, much emphasis is placed on the many novel phytochemicals found in plant foods (eg. carotenoids, bioflavonoids, isothiocyanates, indole carbinols, etc.) and on several established vitamins and minerals (eg. vitamins C and E, folate, selenium, calcium). Proposed mechanisms range from reduced formation of cancer promoting substances in the gastrointestinal tract, through antioxidant activity to the part played by phytochemicals and micronutrients in detoxification of carcinogenic substances, and to functions relating to the containment and destruction of existing cancer cells through a variety of physiological processes and through improved immunosurveillance\(^6,7,28\).

In the 1997 World Cancer Research Fund and the American Institute of Cancer Research (WCRF/AICR) global review of nutrition and cancer prevention\(^7\). Prevention by fruit and vegetables was rated to be convincing for cancers of the mouth, pharynx, esophagus, stomach, colon, rectum and lunge; probable for the larynx, pancreas, breast, bladder and possibly for the ovaries, cervix, endometrium, thyroid, liver, prostate and kidney. Since that report, data has become available from a number of further case-control and cohort studies which generally confirm the earlier findings. In particular lower risks of cancer were again found in relation to higher intakes of vegetables and fruit and cancers and the
oral cavity 29,30, stomach 31 and colon and rectum 32, although a recent study found no evidence that one extra serving of fruit and vegetables provides any measurable additional protection 33. Two recent studies on lung cancer also consistently indicate that a high intake of fruit and vegetables is protective, particularly with respect to Brassicae vegetables, tomatoes, lettuce and cabbage 34,35. Further suggestive evidence of protection by fruits and vegetables has been noted for cancer of the bladder 36,37, breast 38,39 and to a lesser extent cancer of the prostate 40,41.

However, it should be noted that although considerable emphasis has been placed on the WCRF/AICR review, attention should also be paid to the study by the United Kingdom Department of Health Committee of the Medical Aspects of the Food Supply (COMA) which also reviewed the evidence concerning the potential protection afforded by fruit and vegetables against the development of cancer 42. The COMA study ranked the evidence into 4 categories, the top two being “strong” and “moderate”. According to COMA no strong association was found between fruit and vegetable consumption and cancer at any site, while a moderate association was noted for cancers of the stomach, colon and rectum. In contrast WCRF/AICR rate the evidence for an association as convincing for the mouth/pharynx, stomach, colon, rectum and lung (7). Clearly interpretation of the data by COMA is more cautious than WCRF/AICR but both committees recognise the importance of these foods in reducing cancer risk.

**Non-insulin dependent diabetes mellitus**

At the population level an association has been noted between increased consumption of plant foods and lower incidences of obesity (a risk factor for diabetes) and non-insulin dependent diabetes mellitus, although it is not clear at this stage whether this apparent protection by plant foods arises principally from a lower body weight. In the dietary control of the disease, vegetables in particular are likely to be of value due to their content of fibre and complex carbohydrates and their possible hypoglycaemic activity 6,10. Recently, a cross-sectional study undertaken in the United Kingdom revealed an inverse association between the risk of non-insulin-dependent diabetes mellitus and frequent consumption of vegetables throughout the year, although the effect did not appear to be significant during the summer months 43.

**Cataract and macular degeneration of the eye**

Several studies with humans have reported that the risk of developing ocular cataracts is significantly higher in people with low dietary intakes of fruit and vegetables and vitamins C, E and -carotene 12,44. A similar increased risk was observed in people with low levels of vitamins C and E in their blood. Experimental studies with model systems have added further support to the notion that above average intakes of antioxidant nutrients may delay the onset of senile cataract 12. More recently a modest protective effect against the development of cataracts has been observed for higher intakes of the carotenoids lutein and zeaxanthin 45.

Age-related degeneration of the macular (the colour-sensitive yellow spot on the retina of the eye) is another serious cause of acute blindness of the elderly, and one that is not reversible. Findings from a number of human studies suggest that people with low levels of carotenoids and the antioxidant vitamins C and E in their blood, and who smoke, are at increased risk of developing macular degeneration. Experimental studies indicate that two
carotenoids in particular (ie. lutein and zeaxanthin) appear to be accumulated by the macula, and in a human study when the dietary intake of carotenoids was analysed, the sum of the intake of lutein and zeaxanthin had the strongest protective effect against macular degeneration. Taken together these findings suggest that in many cases macular degeneration may be preventable by eliminating smoking and ensuring an adequate intake of fruit and vegetables\textsuperscript{14}. Of particular interest are several recent reports which highlight the presence of lutein and zeaxanthin in precise but different orientations in the membranes of the macula, which suggests that these two carotenoids may serve a special role in reducing the risk of age-related macular degeneration\textsuperscript{46,47}.

PRACTICAL ASPECTS

Relationship to the Australian Guide to Healthy Eating

The Australian Guide to Healthy Eating (AGHE) recognises the importance of fruits and vegetables in a healthy diet and recommends consumption of between 2-4 serves of fruit and 4-8 serves of vegetables each day for adults\textsuperscript{49}, which is generally in line with the minimum 5 serves of vegetables and 2 of fruit established by the Core Food Group Analysis endorsed by the NHMRC\textsuperscript{50}. It should however be noted that average current fruit consumption in Australia falls significantly short of this recommendation, as indicated in Table 2.

Table 2: Intakes of fruits and vegetables in Australia in relation to from the NHMRC Core Food Group analysis\textsuperscript{51}

<table>
<thead>
<tr>
<th>Ages</th>
<th>Fruit</th>
<th>Vegetables</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Inclusions % recommended</td>
<td>Recommended</td>
</tr>
<tr>
<td></td>
<td>Including juice</td>
<td>Excluding juice</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>F</td>
</tr>
<tr>
<td>4-7</td>
<td>165</td>
<td>154</td>
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<tr>
<td>8-11</td>
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<td>12-15</td>
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</tr>
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<td>45-64</td>
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<td>65+</td>
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</tbody>
</table>

* serve of fruit equals 150 g; serve of vegetables equals 75g; where recommendations are a range, the mid point has been used for calculations
* note: WCRF recommends 400-800g/day excluding potatoes and legumes for adults

Recommended fruits include apples and pears, citrus, melons, tomatoes, berries, grapes, bananas and stone fruits (eg. apricots and peaches). A variety of vegetables are recommended including dark green vegetables (eg. spinach, broccoli), orange/yellow vegetables (eg pumpkin, carrots), cruciferous plants (eg. broccoli, cauliflower, cabbage), starchy vegetables like potatoes, corn and yams, and salad vegetables for example lettuce, tomato, cucumber and capsicum. Many of these foods can be eaten raw or slightly cooked and some should be served that way to maximise nutrient content.
AGHE recommendations for fruit include raw, stewed or canned varieties, with rather less emphasis on fruit juices or dried fruit as they tend to be lower in fibre and more energy dense respectively, although a modest intake of both (eg. one serving per day) is acceptable.

**Preparation of Fruit and Vegetables**

Certain nutrients and phytochemicals in plant food are damaged by cooking, others are not. In fact, in some cases the availability of a nutrient may be increased by the cooking process, for example carotenoids are absorbed better from cooked tomatoes than when eaten raw. As a general rule fruit and vegetables may be eaten in a manner most palatable to the consumer, although there should always be a good proportion that are eaten raw as salads and fruits.

When vegetables are cooked, they should not be overcooked as this will cause loss of nutrients. An effective method of cooking vegetables is stir frying as this technique tends to minimise nutrient loss and provides a tasty product with good texture. Light microwaving and steaming are also better than deep frying or prolonged boiling. Generally, when cooking vegetables it is of value to use a small amount of oil as this will enhance the absorption of the fat soluble vitamins (eg. vitamins A and E) as well as other fat soluble dietary components like the carotenoids.

It should be noted that the variety of fruits and vegetables recommended by AGHE will ensure an adequate intake of some of the less widely distributed dietary components, for example green leafy vegetables for folate, yellow and orange fruits and vegetables for carotenoids, cruciferous vegetables for dithiolthiones and isothiocyanates which improve the body’s detoxification capacity, the allium vegetable family for allyl sulfides which also improve detoxification processes, fruit for bioflavonoids which appear to serve many beneficial functions in the body including acting as antioxidants, and citrus fruit and capsicum for vitamin C. Where required, frozen and canned varieties of fruits and vegetables are acceptable as good levels of nutrients are retained by both processes, especially in frozen foods.

**Changes Needed to the Diet**

The objective of the fruit and vegetable guideline is not to encourage people to eat only vegetarian meals but rather to highlight the important health benefits to be derived from the regular consumption of plant-based dishes together with individual fresh and cooked fruit and vegetables.

**Special Groups Needs**

**Pregnancy**

The AGHE advises an increased daily intake of around one serve of fruit and one serve of vegetables during pregnancy. This increase is especially important to provide the extra folate, vitamin C and other micronutrients recommended in the Recommended Dietary Intakes for Use in Australia.
**Lactation**
The AGHE advises an increased daily intake of around three serves of fruit and two of vegetables during lactation\(^49\), which is needed to meet the substantially increased requirement for vitamin A, folate, vitamin C, vitamin E and other micronutrients at that time\(^52\).

**Vegetarians**
The Fruit and Vegetable Guideline applies equally to vegetarians and to people eating other diets. However, particular emphasis should be given to eating legumes and nuts in order to increase the iron and complementary protein intake from plant sources. Also, fruit juices or fruit should be consumed in the same meal in order to provide vitamin C which will increase iron absorption.

**CHANGES FROM PREVIOUS GUIDELINES**
The present guideline differs from the previous guideline in that separate guidelines have been established for Breads and Cereals, and for Vegetables (including Legumes) and Fruits. This change reflects the approach taken in the recent Dietary Guidelines for Older Australians\(^53\), as it is felt that the health benefits conferred by these two categories of plant foods tend to often occur by largely different mechanisms and the dietary components involved are distributed differently between cereal grains and fruits and vegetables.

**RELATIONSHIP TO OTHER CURRENT GUIDELINES**

*Enjoy a wide variety of nutritious foods*
In order to obtain optimal health benefits from fruit and vegetables a wide variety of these foods need to be consumed, which supports and contributes to this guideline.

*Eat plenty of cereals (including breads, rice, pasta, noodles), preferably wholegrain*
Apart from providing a good source of energy these cereal based foods contribute a number of protective factors to the diet, which complement and extend many of the benefits derived from fruit and vegetables.

*Limit saturated fat and moderate total fat intake*
Fruit and vegetables are low in saturated fat.

*Choose foods low in salt*
Fruits and vegetables are low in salt (sodium) but are good suppliers of potassium.

*Care for your food and keep it safe to eat*
Although less likely to cause sickness if not stored carefully, compared with animal products, the nutritional value of fruit and vegetables will decline along with their palatability in food that is spoilt due to inadequate storage. Also, there is a risk that moist fruit and vegetables that are not peeled may develop bacteria on their surface and may cause sickness if not well washed in clean water. Sprouts, salads etc… are prone to contamination with bacteria and viruses and need appropriate washing and sanitisation.
CONCLUSIONS

Strong evidence now exists that many compounds in fruit and vegetables (phytochemicals) may help to protect against the development of a number of non-infectious degenerative diseases including cancer, cardiovascular disease, diabetes, hypertension and cataract and macular degeneration of the eye. Adults are encouraged to consume on average at least two helpings of fruit and 5 of vegetables each day, selected from a wide variety of types and colours of these foods and served both cooked or raw where appropriate.

EVIDENCE

There is Level II (ref 9) and Level III (refs 3,4,8,27) evidence in relation to the benefits of fruits and vegetables and cardiovascular disease, hypertension and stroke and Level III evidence in relation to fruit and vegetable consumption and cancer of various kinds (refs 29-41).
REFERENCES


EAT PLENTY OF CEREALS (INCLUDING BREADS, RICE, PASTA AND NOODLES), PREFERABLY WHOLEGRAIN

BACKGROUND

From an evolutionary perspective, cereal grain consumption is relatively recent, dating from only 5,000 to 10,000 years ago, yet today eight cereals (wheat, maize, rice, barley, sorghum, oats, rye and millet) provide more than 56% of the energy and 50% of the protein consumed on earth (1). Many traditional hunter-gatherer societies had diets with a relatively low proportion of energy from carbohydrate (22-40%) and only small amounts of grain (2), although indigenous Australians may have consumed large quantities of grain in some areas. However it is difficult to base conclusions about desirable dietary patterns for modern societies on this evidence, and recent reports recommend that carbohydrate should provide greater than 55% of energy for optimal health (3).

Cereal grains form the basis of diets in many different cultures and cuisines. They generally are excellent sources of carbohydrate and dietary fibre and are also a significant source of protein (ranging from 8-16g/100g). They are mostly low in fat and are good sources of B-group vitamins, vitamin E and many minerals, notably iron, zinc, magnesium and phosphorus. Eating enough cereal foods helps ensure an adequate nutritional intake. They can be stored safely for long periods and are also relatively inexpensive: in 1998-99, purchases of cereal products accounted for only seven percent of household food expenditure (4). Ecologically, a high carbohydrate diet based on cereals also makes good use of the world’s resources, since grain crops require relatively few input resources per unit of food energy produced (5). For these reasons all current dietary guides have cereal foods as the largest component of the recommended daily food intake.

DEFINITIONS

Cereal foods
‘Cereal foods’ refers to the whole class of foods, including cereal grains, bread, breakfast cereals, low-fat crackers, flours, pasta, noodles and other plain cereal items but excludes cereal-based products with significant amount of added fat and sugar such as cakes, pastries and biscuits.

Cereals
‘Cereals’ refers to whole or partially processed cereal grains (for example, rice, oats, corn, barley), breakfast cereals and other cereal products (for example, flour, polenta, semolina, burgul, brans and wheatgerm).

Breads
‘Breads’ refers to leavened and unleavened wholemeal, white, mixed-grain and rye breads, as well as rolls, bagels, crispbreads and low-fat crackers.

Pasta and Noodles
These foods include a wide range of European and Asian products based on sheets of dough made from flours – usually wheat or rice – and water, sometimes with added egg.
Examples include spaghetti, lasagne, fettucine, udon and hokkein noodles, rice paper and wonton wrappers.

**Wholegrain**

‘Wholegrain’ refers to cereal foods that incorporate all the components of the natural grain, including the bran and germ. The US Food and Drug Administration has recently proposed that foods that contain at least 51% by weight of any combination of whole grains can be termed ‘wholegrain’ (6). This definition would include such foods as wholemeal breads and crispbreads, many high fibre breakfast cereals, oatmeal, wholemeal pasta, brown rice and popcorn.

**NEED FOR THIS GUIDELINE**

Apparent consumption of cereal foods in Australia (based on national food disappearance data) has remained relatively constant since the 1930s; in 1998-9 it was 138.1 kilograms per person (7). There have, however, been changes in the mix of products since the 1930s: consumption of rice and breakfast cereals has increased significantly and consumption of flour has fallen. Bread consumption has remained relatively stable.

The 1995 National Nutrition Survey (NNS), using 24-hour diet recall, found that 94.5 per cent of Australians aged more than 19 years had eaten cereal foods on the day of the survey, with the most commonly consumed foods being bread (80.5 per cent) and breakfast cereals (50.9 per cent) (8). The mean adult daily intakes were 250g for men and 181g for women. Intakes were somewhat lower among people in rural and remote areas compared with people in metropolitan areas and significantly higher among people born in Southeast Asia (because of their much higher consumption of rice).

The NNS found that for adult Australians cereal foods are important sources of energy, carbohydrate, dietary fibre, thiamin, iron and magnesium, providing more than 20 per cent of the total daily intake of these nutrients (Table 1). Cereal foods also provide more than 10 per cent of the daily intakes of protein, polyunsaturated fat, riboflavin, niacin, folate, calcium, phosphorus and zinc.

**Table 1. Percentage of adult nutrient intake provided by cereal foods, 1995**

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Men aged 19+</th>
<th>Women aged 19+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy</td>
<td>20.0</td>
<td>20.8</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>33.2</td>
<td>33.1</td>
</tr>
<tr>
<td>Dietary fibre</td>
<td>34.9</td>
<td>33.6</td>
</tr>
<tr>
<td>Thiamin</td>
<td>41.3</td>
<td>40.5</td>
</tr>
<tr>
<td>Iron</td>
<td>30.1</td>
<td>29.3</td>
</tr>
<tr>
<td>Magnesium</td>
<td>24.3</td>
<td>24.8</td>
</tr>
</tbody>
</table>

*Note: Biscuits, cakes, and other cereal-based items are excluded.*

One study of the cereal intake of various socio-economic groups in Australia found that cereal foods contribute more to nutrient intakes among upper occupational groups for both males and females (10). A study of 18-year-olds in Western Australia also found higher levels of cereal consumption in higher SES groups (11).
Most Australians seem to be satisfied with the amount of cereal foods they eat. In the NNS only eight per cent of respondents aged more than 19 years reported they would like to change the amount they consumed; this compared with up to 30 per cent reporting that they wanted to eat more fruit and vegetables (12). Despite this, data from the 1995 NNS show that even amongst adults with the highest intakes (those aged 19-24 years), on the day of the survey only 34% of men and 21% of women met the recommended core food group cereal targets of seven serves per day (13).

SCIENTIFIC BASIS

While there have been many experimental studies with individual nutritional components provided by cereal foods - such as dietary fibre, starch or vitamin E - there are relatively few prospective studies or controlled experimental trials using whole foods to support this dietary guideline. It is difficult to achieve acceptance of long term changes to the largest staple components of diets, and generally impossible to do so in a double blind manner. Most of the available evidence therefore comes from ecological, cross-sectional, case-control and cohort studies. Even in these there is often inadequate dietary methodology to analyse the consumption of different types of cereals or to quantify dose-responses.

All recent reviews have supported the beneficial effects of cereal fibre and whole grains in relation to decreased risk of coronary heart disease (CHD) and some cancers (6, 14-17) and data from several countries suggest that higher intakes of breads and cereals help people to achieve dietary targets for lower fat consumption (18, 19). In 1999 the US Food and Drug Administration approved the health claim: ‘diets rich in whole-grain foods and other plant foods and low in total fat, saturated fat and cholesterol may reduce the risk of heart disease and certain cancers’ (17).

Coronary Heart Disease

The published results of over 200 human trials have led to the general conclusion that foods rich in soluble fibre can lower plasma cholesterol (20-22). Australia’s National Heart Foundation has stated, ‘the consumption of dietary fibre, especially cereal fibre, is associated with a lower risk of CHD’ (23). Meta-analyses of intervention trials with two cereal foods - oats and psyllium - have shown that these are particularly effective in reducing serum cholesterol (24, 25). By contrast, controlled human trials with supplements of isolated wheat fibre have consistently shown no effect on plasma cholesterol (20).

A large prospective study of US male health professionals found dietary fibre intake was strongly associated with reduced rates of myocardial infarction and that cereal fibre was apparently more protective than fibre from fruits or vegetables (26). That study reported a 29 per cent reduction in coronary heart disease for every 10 gram increase in daily intake of cereal fibre. Preliminary analysis of a prospective study of 31284 postmenopausal women in Iowa has also found the relative risk of CHD was 0.76 (95%CI: 0.55–1.05) among women in the highest quintile of dietary fibre intakes compared to the lowest (14).

The principal mechanism is probably the action of viscous polysaccharides acting in the gastrointestinal tract to decrease reabsorption of biliary cholesterol (27) but other components may be involved in the protective effect of wholegrain cereals. Vitamin E, folate, selenium, phytoestrogens and phytic acid may all be important (28). In the Nurses Health Study, wholegrain consumption was associated with significant reductions in risk
of both CHD (29) and ischaemic stroke (30). In older women there is also evidence from the Iowa Women’s Health Study of a clear inverse association between wholegrain intake and the risk of ischaemic heart disease (26) as well as all-cause mortality (31). The authors calculated that if all women consumed one serving per day of whole grain foods, total mortality rates might be reduced by 8% or more.

In that study there was a small positive association (adjusted hazard rate ratio 1.16) between refined grain intake and total mortality, but this was attenuated and lost significance when wholegrains were added to the model. There was no association between refined grain intake and risk of CHD (32). In a study of dietary associates in patients with established coronary disease, a high intake of not only wholegrain but also total cereal products was associated with lower total cholesterol (33).

Dietary carbohydrates may also exert an influence on cardiovascular disease risk via their effect on insulin response. High glycaemic index (GI) carbohydrates are characterised by rapid absorption and high postprandial glucose and insulin responses and may result in decreased insulin sensitivity (34), a risk factor for CHD (35). At least three cross-sectional studies have also found an inverse relationship between HDL cholesterol and GI (36-38). In the prospective Nurses Health Study, over ten years the glycaemic load and the total diet GI were both predictive of CHD risk (39). One randomised crossover study with Type 2 diabetics found lowering the GI of a diet (mainly by altering the physical form of the cereals) resulted in significantly lower LDL and higher HDL cholesterol levels (40). A study in free living Australian diabetic subjects also found HDL cholesterol levels were higher on a low versus high GI diet (41). These recent findings support the earlier FAO/WHO consultation recommendation that ‘the glycaemic index of foods be used in conjunction with information about food composition to guide food choices’ (3).

Obesity

A high-carbohydrate diet/low fat diet is recommended for maintenance of body weight and prevention of obesity (42, 43), and obesity is associated with low fibre intake (44). When high starch, high sucrose and high fat ad libitum diets were compared, energy intake was lowest on the high-starch diet (45) and higher intakes of carbohydrates have been linked to lower waist-hip ratios and lower BMI (46).

There are several ways high-fibre cereals can reduce energy intake and help maintain weight: they take longer to eat; they decrease the energy density of a meal; and some fibres may slow gastric emptying and affect gastrointestinal hormones that influence food intake (47). Consumption of high GI carbohydrates promotes a more rapid return of hunger and increases subsequent energy intake, compared to low GI choices, and slower digestion of carbohydrate is associated with higher satiety (48). Thus consumption of wholegrain and lower GI cereals instead of highly refined cereals may help prevent excess weight gain (49).

Diabetes

The recent joint WHO/FAO consultation on carbohydrate concluded that foods rich in slowly digested or resistant starch, or high in soluble fibre might be protective against diabetes (3). Recent large prospective studies of men and women have found cereal fibre
intake was inversely associated with risk of developing NIDDM and that the protective effect was even greater when combined with a low total glycaemic load (50, 51).

A large prospective study of adult US women found a lower risk of type 2 diabetes associated with higher intakes of all cereal grains (RR 0.75; 95%CI: 0.63-0.89) and wholegrains in particular (RR 0.73; 95%CI: 0.63-0.85), whereas a higher intake of refined grain was related to increased risk (RR 1.26; 95%CI: 1.08-1.46)(52). The individual foods associated with the strongest protective effects were wholegrain breakfast cereal, brown rice and bran. However, in this study ‘refined grain’ included a wide range of higher fat cereal-based foods such as cakes, desserts and pizzas, and ‘wholegrain’ foods included some that are relatively refined (eg ‘couscous’).

For people with established NIDDM, use of low GI foods is associated with improvement in glycaemic control (40, 53). In southern European patients with type 1 diabetes, HbA1c was 11% lower in patients with the lowest quartile of dietary GI compared to the highest, and was related to eating more pasta (37). During pregnancy, women on a low GI diet (eating bran breakfast cereal, wholegrain bread and pasta) experienced no change in their glycaemic response to a 500 calorie test meal with 55% energy from carbohydrate, whereas those who switched to a high GI diet experienced a 190% increase in their response (54).

Cancer

Major reviews of the relationship between cereal consumption and cancer prevention have been published (55, 56). It is difficult to evaluate many studies because of the paucity of biological markers, the inadequacy of many food intake measurements which often do not distinguish the degree of refinement of cereal foods, and the low overall intakes of cereal fibre in many of the studies from the United States. There is, however, emerging agreement on the probable protective role of cereals in relation to some important cancer types. In particular it appears that wholegrain intake confers benefits. In a review of 40 case-control studies of 20 cancers the pooled odds ratio for high versus low wholegrain intake was 0.66 (95%CI: 0.60-0.72) (57). The protective components in wholegrains may include fermentable carbohydrates, oligosaccharides, flavonoids, phenolics, phytoestrogens, lignans, protease inhibitors, saponins and selenium (58-60).

Some case-control studies have suggested not only that wholegrains are protective, but that conversely consumption of refined cereals (including bread, pasta and rice) increases the risk of cancers of the oral cavity, oesophagus, larynx, stomach and colon (60-62). However the authors have been cautious about inferring causality from these associations, noting that diets high in refined cereals are often poor in fruit and vegetables as well.

Colorectal Cancer

Prospective data from the large Health Professionals Follow-up Study suggest that dietary fibre intake is inversely associated with the risk of colorectal adenoma in men, the relative risk in the highest versus the lowest quintile being 0.36. All sources of dietary fibre were protective, but the effect was stronger for grain sources than for fruit or vegetables (63).

A World Cancer Research Fund (WCRF) review concluded that diets high in both starch and dietary fibre could possibly decrease the risk of colorectal cancer (55). It was
concluded that cereals may well have a protective effect but that there was still insufficient
evidence. Two subsequent reviews have reported more definite conclusions. A recent
consensus statement from the European Cancer Prevention Organisation (ECPO), based on
a review of 58 epidemiological studies, concluded ‘a diet rich in high-fibre cereal is
associated with a reduced risk of colorectal cancer’ (56). This conclusion is supported by a
meta-analysis of case-control studies of wholegrain intake and colorectal cancer, which
calculated a pooled odds ratio of 0.79 (95%CI: 0.69–0.89) when high and low intakes of
wholegrains were compared (57).

In experimental studies, wholemeal rye bread reduced the concentration of some
compounds that are putative colon cancer risk markers, compared with white bread (64).
Resistant starch may also favourably affect some of the faecal markers of colon cancer
risk, in a way similar to dietary fibre. Cereal foods are estimated to provide 42 per cent of
the resistant starch in the Australian diet (65).

In some prevention trials, supplements of wheat bran have reduced the incidence of rectal
polyps in predisposed individuals (66) and, when combined with a low-fat diet, reduced
the incidence of large adenomas (67). However the effectiveness of fibre has been disputed
in three recent studies. Analysis of 16 years of follow-up in the Nurses Health Study did
not support the hypothesis that dietary fibre intake can reduce the risk of colon cancer,
although the power of this study was limited by the low range of median cereal fibre
intakes (ranging from 1.0g a day to only 4.8g a day in the highest quintile)(68). The
multicentre Phoenix colon prevention trial involving 1429 subjects with preexisting polyps
examined the effect of a wheat bran breakfast cereal supplement taken over three years.
This study found no effect, but the authors noted that the actual supplementary fibre intake
(9.4g/d) may have been too small to effect a significant change (69). Lastly, a polyp
prevention trial in the US found a low fat, high fibre diet (with increased intakes of fruit,
vegetables, cereals and legumes) did not alter the recurrence of adenomatous polyps (70).

**Breast Cancer**

Fibre may reduce the intestinal reabsorption of oestrogen, and bioactive cereal components
such as lignans may be protective through their action as weak phytoestrogens. National
consumption data suggest energy from cereals is inversely related to breast cancer risk
(71) and a meta-analysis of 12 case-control studies found a significant reduction in risk
with increasing dietary fibre (72). The WCRF concluded that dietary fibre possibly
decreases the risk of breast cancer (55) and the ECPO consensus meeting agreed that there
is evidence to suggest cereal fibre provides protection against breast cancer (56), but this is
still uncertain (73). More recently a study of breast cancer recurrence over five years found
energy-adjusted bread and cereal consumption was protective (hazard ratio 0.55),
especially in postmenopausal women (74).

**Stomach Cancer**

In relation to stomach cancer, the WCRF report concluded from the evidence of six case-
control studies that there was a possible protective association for consumption of
wholegrain cereals and cereal products but that the evidence for cereals as a whole was
inconsistent and inconclusive (55).
Other Cancers

One large cross-national study has found prostate cancer mortality inversely associated with estimated consumption of cereals (75) and case-control studies suggest wholegrain foods are protective (73). A few case-control studies report a protective effect of wholegrain consumption on oral and pharyngeal cancers (76, 77), but for none of these cancers are data available from human intervention studies.

Constipation and Diverticular Disease

There is a strong correlation between dietary fibre intake and mean daily stool weight (78), and cereal fibre has been found to improve bowel function by increasing faecal bulk and reducing transit time, resulting in softer, larger stools and more frequent bowel action (79-81). Diets rich in insoluble fibre, such as those present in wholegrain cereals and breads, are associated with a lower prevalence of constipation and diverticular disease. The US prospective study of 43881 male health professionals found evidence that a diet high in fibre, particularly the cellulose component which is obtained largely from cereal foods, was significantly associated with decreased risk of diverticular disease (82).

Hypertension

Hypertension remains an important risk factor for cardiovascular morbidity and mortality, and a reduction in sodium intake is one of the primary preventive measures. Cereals in their natural state are very low in salt and have a favourable potassium:sodium ratio, but processed cereal foods, especially bread, are significant sources of salt in the Australian diet. The Victorian Nutrition Survey found that processed cereal products gave men 28 percent of their daily sodium intake and women 26 percent - more than twice as much as any other food group (83). Both dietary fibre and magnesium may be protective against hypertension and cereal foods are important sources of both these nutrients, but fruit sources of fibre appear more protective than cereal sources (84, 85).

Nutrient Density

Two of the main cereal foods - breakfast cereals and breads - are often fortified with vitamins and minerals that may be marginal in the diet. For example, data from the NNS show that those who regularly include breakfast cereal in their diet are much more likely to meet the recommended dietary intakes for iron, calcium, magnesium, folate, riboflavin and thiamin (86). Bread has long been a useful staple for fortification (87) and some are now sources of additional fibre, iron, folate, and omega-3 fats. In 1991 it became mandatory for all bread to be fortified with thiamin and since then there has been an apparent decline in the prevalence of Wernicke-Korsakoff syndrome in Australia (88, 89).

PRACTICAL ASPECTS OF THIS GUIDELINE

Relationship to the Australian Guide to Healthy Eating (AGHE)

The AGHE recommends that consumption of breads, cereals, rice, pasta and noodles form the bases of selection of a healthy diet, with the greatest proportion of food coming from this group (90). The recommended number of serves for adults aged 19-60 are 4-9 for women and 5-12 for men. Cereal based foods such as cakes, biscuits, and pastries – that
can have high levels of added fats and sugars – are not included in this recommendation and should be regarded as occasional treats only.

Some easy ways to achieve the recommended targets include:
- Consuming breads with each meal
- Regularly using rice, couscous, pasta or noodles to accompany hot dishes
- Eating breakfast cereals daily
- Including whole grains cereals as extenders to soups and casseroles
- Using oats in crumble toppings on desserts

**Sodium Intake**

Bread is the most commonly consumed cereal food in Australia; it has a typical sodium content of around 450mg per 100g. The mean daily consumption of regular and fancy breads by people over 19 years reported in NNS was 101.7g, which would contribute 458 milligrams of sodium - around 20 per cent of the recommended maximum sodium intake (91) compared to their contribution of only 10% to the daily energy intake (9). Greater consumption of cereal foods with high salt levels could make it more difficult for people to limit their sodium intake, but this is not a reason to recommend against plentiful consumption of cereals. People seeking to increase their intake should prefer those choices that are lower in salt - such as rice, oats, couscous, pasta and many lower salt varieties of breakfast cereals and breads.

**Glycaemic Index**

Many processed starchy cereal foods - such as most breads, rices and breakfast cereals - tend to have high GI values. Pasta, grainy breads and some breakfast cereals have low GI values. This does not mean that high-GI grain foods need to be avoided altogether: the glycaemic load of a diet can be balanced by combining high and low GI carbohydrate sources in the same meal. Exchanging half the carbohydrate from high to low GI will lower the GI of the overall diet by about 15 units, sufficient to bring about clinical improvements in glucose metabolism (92).

**RELATION TO OTHER GUIDELINES**

In the 1992 edition the guideline relating to cereal foods was: ‘Eat plenty of breads and cereals (preferably wholegrain), vegetables (including legumes) and fruits’ (93). In these new guidelines, the recommendation relating to vegetables and fruit has been separated, to give it greater prominence. The emphasis on ‘wholegrain’ has been retained because of the growing evidence of the health benefits of wholegrain compared to refined cereal products.

**Enjoy a wide variety of nutritious foods**

Different cereals provide varying amounts and types of dietary fibre as well as different levels of potentially active phytochemicals and nutritive antioxidants (94). The levels of some nutrients, such as selenium, in cereals vary considerably according to the growing region. It is important to eat a wide variety of cereal foods to maximise their nutritional benefits.
Choose low salt foods
Standard commercial breads and some breakfast cereals are major sources of salt in the diet. To cut down on salt intake, lower salt cereal products and unprocessed whole grains should be preferred.

Limit saturated fat and moderate total fat intake
Cereals are naturally very low in saturated fat, so increased cereal consumption assists with this guideline, as long as the amount of fat added to such foods is limited (eg, fat spreads on bread, oil added to pasta, fried rice). Care needs to be taken, however, with many other cereal-based foods such as biscuits, cakes and pastries: they can have high levels of added saturated fat.

CONCLUSION
All breads and cereals are economical foods that are a major source of essential macro- and micro-nutrients. Wholegrain cereal choices, which generally are higher in dietary fibre and have a lower glycaemic index, offer protection against CHD, some cancers and obesity, and should be preferred. The words ‘eat plenty’ are used to encourage people to choose these foods liberally as the basis of their daily diet.

EVIDENCE
There is Level I evidence of the cholesterol-lowering properties of oats and psyllium (refs 24, 25); of the cholesterol-lowering properties of cereal fibres generally (ref 22) and of the preventive effect of dietary fibre on constipation (ref 80). There is Level II evidence for a protective effect of wheat fibre on rectal polyps (ref 66) and in combination with low fat diets, on large adenomas (ref 67) but also Level II evidence (refs 69, 70) of no effect on recurrence of adenomas.

There is Level III evidence for low GI diets and improved lipid profile and improved glycaemic control in diabetics (refs 36, 37, 38, 40, 41, 54); cereal fibre and reduced risk of colorectal cancer (ref 64); wholegrain cereal and reduced risk of diabetes (refs 50, 51, 52); cereal fibre and reduced risk of CHD (refs 14, 26, 29, 30, 31, 32); cereal fibre and reduced risk of breast cancer (refs 71, 72, 74); wholegrain cereal and reduced risk of cancers (refs 57, 61, 62, 63, 68, 73, 75, 76, 77); wholegrain cereal and weight control (refs 45, 46) and dietary fibre and reduced risk of diverticular disease (ref 82).
REFERENCES


INCLUDE LEAN MEAT, FISH, POULTRY AND/OR ALTERNATIVES SUCH AS LEGUMES AND NUTS

DEFINITIONS

Meat
Includes the whole or part of the carcass of any cattle, sheep, goat, buffalo, kangaroo, camel, deer, goat, pig or rabbit.

Red Meat
In Australia, ‘red meat’ refers to the muscle meat from cattle, sheep and goat. It does not include pork, ham, bacon or kangaroo. In other parts of the world (eg US, UK and Europe) ‘red meat’ includes pig meat.

Poultry
Includes chicken, duck, turkey and all other avian foods, except eggs

Alternatives
Alternatives refers to other protein-rich foods such as legumes, nuts and nut pastes and certain seeds such as sunflower and sesame as well as eggs, shellfish.

Anaemia
A deficiency of red blood cells or their haemoglobin often, but not always, related to iron deficiency.

Iron Deficiency
A condition of low body iron which may manifest itself as low serum iron, high serum iron-binding capacity, reduced transferring saturation index and or high free erythrocyte protoporphyrin. It can produce symptoms of fatigue, listlessness and pallor and may progress to anaemia. However, it can have widespread, non-haematological effects on behaviour, cognition, and motor development, physical work performance and body temperature regulation.

BACKGROUND

Meats, poultry and fish and their alternatives contribute a number of key nutrients some of which are marginal in the Australian diet. The foods in this food group are very valuable sources of protein as well as being a major source of a number of vitamins and minerals such as iron, zinc and vitamin B12 (see Table 1). Meats, and to a lesser degree, poultry and fish, are the best sources of bioavailable iron in the Australian diet. Alternatives such as legumes, nuts, certain seeds and eggs are also valuable sources of protein and, to a lesser degree, iron and zinc. However, the iron and zinc from plant sources are less bioavailable than from animal sources. The plant-based alternatives do not provide any vitamin B12. Fish, and to a lesser degree, red meats, are also valuable sources of omega-3 fatty acids in the Australian diet. However, it is as a rich source of bioavailable iron that this food group plays its major public health role in Australia.
Table 1  Nutrient content per 100g of sample lean meats, poultry, fish and equivalents*

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Lean beef</th>
<th>Lean lamb</th>
<th>Skinless chicken</th>
<th>Lean pork</th>
<th>Fresh flathead fish</th>
<th>Canned red salmon</th>
<th>Liver-lamb</th>
<th>Eggs</th>
<th>Soybean s dry-cooked</th>
<th>Canned baked beans</th>
<th>Almonds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kJ)</td>
<td>450</td>
<td>501</td>
<td>466</td>
<td>438</td>
<td>395</td>
<td>815</td>
<td>680</td>
<td>632</td>
<td>537</td>
<td>285</td>
<td>2455</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>21.6</td>
<td>20.4</td>
<td>20.4</td>
<td>21.6</td>
<td>21.1</td>
<td>21.9</td>
<td>21.4</td>
<td>13.2</td>
<td>13.5</td>
<td>4.6</td>
<td>20.0</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>2.4</td>
<td>2.3</td>
<td>0.95</td>
<td>1.0</td>
<td>0.2</td>
<td>1.2</td>
<td>9.4</td>
<td>1.8</td>
<td>2.2</td>
<td>1.6</td>
<td>3.5</td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>3.6</td>
<td>3.4</td>
<td>1.4</td>
<td>2.2</td>
<td>0.6</td>
<td>0.9</td>
<td>4.3</td>
<td>0.9</td>
<td>1.6</td>
<td>0.5</td>
<td>3.6</td>
</tr>
<tr>
<td>Vitamin B-12 (µg)</td>
<td>2.5</td>
<td>0.96</td>
<td>0.41</td>
<td>0.7</td>
<td>1.5</td>
<td>4</td>
<td>84.0</td>
<td>1.1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total Fat (g)</td>
<td>1.8</td>
<td>4.2</td>
<td>3.3</td>
<td>1.7</td>
<td>1.0</td>
<td>12.0</td>
<td>7.5</td>
<td>10.9</td>
<td>7.7</td>
<td>0.5</td>
<td>55.3</td>
</tr>
<tr>
<td>Saturated fa (g)</td>
<td>0.87</td>
<td>1.35</td>
<td>0.92</td>
<td>0.50</td>
<td>0.36</td>
<td>2.21</td>
<td>2.20</td>
<td>3.10</td>
<td>1.10</td>
<td>0.10</td>
<td>3.55</td>
</tr>
<tr>
<td>Mono unsaturated fa (g)</td>
<td>0.82</td>
<td>1.41</td>
<td>1.37</td>
<td>0.51</td>
<td>0.29</td>
<td>2.46</td>
<td>2.00</td>
<td>4.30</td>
<td>1.20</td>
<td>0.10</td>
<td>36.05</td>
</tr>
<tr>
<td>Polyunsaturated fa (g)</td>
<td>0.21</td>
<td>0.34</td>
<td>0.39</td>
<td>0.36</td>
<td>0.52</td>
<td>2.69</td>
<td>1.30</td>
<td>1.00</td>
<td>4.80</td>
<td>0.30</td>
<td>13.10</td>
</tr>
<tr>
<td>Total ω3 (g)</td>
<td>0.07</td>
<td>0.13</td>
<td>0.04</td>
<td>0.04</td>
<td>0.43</td>
<td>2.50</td>
<td>1.13</td>
<td>0.06</td>
<td>0.17</td>
<td>0.03</td>
<td>0</td>
</tr>
</tbody>
</table>

* All raw meats. Nutrient data from: NNS nutrient data file; Nuttab 95;NZ food data base and Spadek T, Chemistry Centre WA (fatty acids in soybean) and reference 44

Low iron intakes are common in Australia. Low iron intakes coupled with increased requirements among population sub-groups such as adolescent girls, vegetarians and pregnant women, makes iron deficiency a significant public health issue. Low iron stores or iron deficiency without anaemia, appears to be relatively common in Australian women (1,2). In adults, iron deficiency may be associated with a reduced work capacity, less efficient response to exercise and impaired immune function (3,4). It may also affect mood and cognitive performance (5,6). In the early 1990s, it was estimated that iron deficiency anaemias alone were associated with health care costs of some $14 million annually in Australia (7). The additional costs related to the sequelae of the more common condition of iron deficiency, have not been estimated.

SCIENTIFIC RATIONALE

Iron

Iron, as part of the haemoglobin in red blood cells, helps to transport oxygen around the body. As part of the enzymes of the electron transport chain, it is necessary for the production of energy from glucose, the main fuel for both the brain and the rest of the body. It is also a critical component of enzymes responsible for brain development and essential for the synthesis of key neurotransmitters required for normal brain function (8).

There has been no recent national assessment of iron status in Australian adults. However, if the results obtained from two recent surveys of young women in West Australia (1,2) can be generalised to other women of this age, low iron stores or iron deficiency without anaemia, could be relatively common in this population group (Table 2).
Table 2 Percentages of females with low iron status from two Australian studies

<table>
<thead>
<tr>
<th>Subjects</th>
<th>% subjects in various categories</th>
</tr>
</thead>
<tbody>
<tr>
<td>265 female university students aged 15-30 years (1)</td>
<td>Iron deficiency:</td>
</tr>
<tr>
<td></td>
<td>Serum ferritin &lt;16µg /L</td>
</tr>
<tr>
<td></td>
<td>Serum ferritin &lt; or = 12µg /L</td>
</tr>
<tr>
<td></td>
<td>Serum ferritin &lt; or = 12 µg /L and transferrin saturation &lt;16%)</td>
</tr>
<tr>
<td></td>
<td>Anaemia:</td>
</tr>
<tr>
<td></td>
<td>Hb&lt;12g/dL</td>
</tr>
<tr>
<td></td>
<td>Hb&lt;12g/dL, serum ferritin &lt; or = 12 µg /L and transferrin saturation &lt;16%)</td>
</tr>
<tr>
<td>Perth study: 211 women aged 15-30y (2)</td>
<td>Low iron stores:</td>
</tr>
<tr>
<td></td>
<td>serum ferritin &lt;30µg /L</td>
</tr>
<tr>
<td></td>
<td>Iron deficiency:</td>
</tr>
<tr>
<td></td>
<td>serum ferritin &lt;16µg /L</td>
</tr>
</tbody>
</table>

The effects of anaemia and iron deficiency on brain development in infancy and very early childhood are well documented (9-12) but evidence is also emerging, from randomised trials, of effects of inadequate iron status on cognitive processes in both adolescence, in relation to verbal learning and memory (5), and in adulthood (6) in relation to reasoning and perceptual organisation.

Western mixed diets contain 1–1.4 mg of iron per 1000 kJ (13). Hence most adults will ingest in excess of 10 mg of iron daily which, theoretically, should be sufficient to meet the needs of most of the population (14). However, only a small and variable proportion (5–20 per cent) of dietary iron is absorbed. Haem iron, found in muscle meat is approximately 25% absorbed and non-haem, as in plants is about 15% absorbed (15). Consumption of meat, fish or poultry , even in relatively small amounts (50-75g) in a mixed meal, can increase absorption from the plant foods in the meal, up to two-fold. (16). The addition of foods rich in vitamin C or fruit juices, to the meal may also greatly increase the absorption of iron from that meal in a dose-dependent manner up to a factor of five or more. (17)

Conversely, certain plant foods can contain inhibitors to absorption. The bioavailability iron from various natural plant sources has been extensively reviewed by Bothwell et al (18). Polyphenols (such as tannins) which form a characteristic component of all plant tissues, have an inhibitory action (18,19). The degree of inhibition correlates well with the polyphenol content of individual vegetables. Tea and to a lesser extent, coffee also profoundly inhibit iron absorption, by binding the iron to form insoluble compounds with tannins (20). Whilst most alcoholic drinks appear to assist iron absorption, this does not apply to some red wines which contain tannins (16,20). Phytates are also inhibitory and are
present in substantial quantities in many cereals and legumes. However, the quantitative relation between these compounds and iron absorption is less clear cut (18). Calcium supplements have been shown to inhibit iron absorption (21) but, in contrast, addition of milk to a cereal-based meal was shown to have no effect on iron absorption in a group of young women (22). In addition, long term calcium supplementation has been shown not to lower plasma ferritin concentration in human subjects (23). Where the plant-based foods from this category are preferred, care therefore needs to be taken to include adequate iron intake to overcome the lower bioavailability and the inhibitory components in the plant foods. There are now also available a wide range of iron-fortified food products with varying, but often unknown, iron bioavailability.

**Recommended intakes and current consumption of iron**

The current Australian Recommended Dietary Intakes for iron (14) are 7mg/day for adult men, 12-16 mg/day for women aged 19-54 years and 5-7mg/day for women over 54 years. An additional 10-20 mg is recommended during pregnancy, but no additional iron is recommended in lactation. The recent US review of Recommended Dietary Allowances (RDA) set an “Estimated Average Requirement” (EAR) of 6.0mg a day for men, 8.1mg/day for women 19-50 years and 5mg/day for women 51 years and over (24) . This resulted in an RDA (RDA=EAR+2SD_{EAR}) of 8mg/day for men of all ages and women 51 years and over, and 18mg/day for women aged 19-50 years. The difference between the EAR and RDA was much greater for women aged 19-50 years (over a doubling) because of the substantially greater variance in the EAR for this group compared to other age/gender groups. The US RDA for women aged 19-50 years is thus some 50% higher than the lower end of the Australian RDI range for this age/gender group (12-16mg) and some 2mg /day higher than the upper end of the current Australian RDI range.

In the 1995/6 National Nutrition Survey (NNS), two-thirds (66%) of women aged 19-44 years consumed less than the lower end of range (12mg/day) of the Australian RDI for iron (25), a similar proportion to the findings of the 1983 National Dietary Survey (26).

A CSIRO analysis of the NNS showed that red meat is a significant source of iron in the Australian diet contributing 14% of the total dietary iron in adults, and 52% of the haem iron intake (27). Other meat, poultry and fish contribute a further 7% of dietary iron. Legumes and other vegetables provided 14%.

**Zinc**

Zinc is important in a number of major metabolic processes including protein and nucleic acid synthesis and the synthesis and action of insulin. It is involved in immune function and cell growth and repair. The long term effects of mild zinc deficiency are unclear, but may include delayed wound healing, impaired immune function and problems with taste and smell acuity (28).

There are strong homeostatic mechanisms regulating zinc and this, together with a lack of sensitive indicators of zinc status, means it is difficult to determine the prevalence of zinc deficiency in a community or to set recommended intakes. Furthermore, the bioavailability of zinc varies markedly between foods and, like iron, it is affected by the composition of the diet. The zinc from animal sources, including eggs, is generally better absorbed than from plant foods. For instance, 21-26% of the zinc in beef is absorbed.
compared to 11-14% from wholemeal bread (29). Absorption of zinc is reduced by phytate in plant foods such as peanuts and soybeans (30). While calcium and iron can potentially reduce zinc absorption, the effect from food intake of these is likely to be relatively small (31).

**Recommended and current intakes of zinc**

The Australian Recommended Daily Intake (RDI) for zinc (14), is 12mg for both male and female adults with an additional 4 mg/day recommended during pregnancy and an extra 6 mg/day in lactation. The recent revision of the US Recommended Dietary Allowances (RDA) concluded that the “Estimated Average Requirement” (EAR) of zinc for men was 9.4 mg/day and for women, 6.8mg/day (24) with a resulting RDA of 11mg/day for men and 8mg/day for women (RDA = EAR + 2SD$_{EAR}$). For pregnancy an additional 5mg a day was added to the RDA for women 14-18 yrs of age (total 13mg/day), and an extra 3 mg a day for women over 18 years of age (total 11mg/day). For lactation, the RDA was set at 14 mg/day for women 18 years old and under and 12 mg/day for those over 18 years old.

The 1995/6 National Nutrition Survey (NNS) (25) showed that nearly three quarters of adolescent girls and women had intakes less than the RDI of 12mg/day on the day of the survey with 45% of adult men and 63% of elderly men having zinc intakes less than the RDI.

It is of interest to note that the new US RDA for zinc for women (8mg/day) is one third lower than the current Australian RDI for women (12mg) which was set in 1991. The figure for men is approximately the same (11mg for the USA vs 12mg in Australia). It is therefore possible that the large proportion of women apparently at risk in Australia of low zinc intake might, in part, be due to an overestimate of requirements based on the limited data available at the time of setting the Australian RDIs in 1991.

In the NNS survey (25,27), the meat, poultry and game products and dishes category provided 32% of the zinc in the diet of adult women and 39% in men. Muscle meats provided 15% of the zinc and fish and seafood, 4%. The proportion of the population who recorded low zinc intakes in the NNS was inversely related to red meat consumption on the day of the survey (27).

**Vitamin B-12**

Vitamin B-12 plays an important biochemical role in the maintenance of myelin in the nervous system and, in conjunction with folate, in the synthesis of DNA. Research at CSIRO has demonstrated the importance of vitamin B-12 in the maintenance of genetic stability. Chromosome damage was shown to be lower with higher plasma vitamin B-12 levels and lower plasma homocysteine levels. Supplementation with folate and vitamin B-12 (at 3.5-10 times the recommended dietary intake) can also reduce such chromosome damage (32).

The main forms of vitamin B-12 available to humans come from animal products in which the vitamin has accumulated from bacterial synthesis. Occasional contamination of soil or water with microbes that produce vitamin B-12 occurs, but plant foods are usually devoid of the active form of the vitamin. Because of the importance of animal foods as a source of this vitamin, dietary vitamin B-12 deficiency can be a problem in vegetarians (33).
The most prevalent deficiency of vitamin B12 is subclinical deficiency, recognised by changes in biochemical levels in the blood. The normal serum vitamin B-12 is usually taken as 200 picograms per ml (or 150 picomoles per litre). Low vitamin B12 levels (as well as low folate and low B6) have been shown to correlate with raised plasma homocysteine (34,35) which in turn is a risk factor for cardiovascular diseases. However, the importance of dietary intake of vitamin B12 in prevention (or correction) of raised plasma homocysteinuria is not clear.

In one Melbourne study, Mann et al measured serum vitamin B-12, homocysteine and folate in healthy men aged 20-55 years (36) eating a wide range of diets from high meat to vegan and found a strong negative correlation \( r = -0.37 \) between serum vitamin B-12 and plasma homocysteine in the combined subjects of the four groups. All meat eaters in Mann’s study had serum vitamin B-12 in the normal range (200-1100 pg/ml) but 23% of ovolactovegetarians and 65% of the vegans had serum vitamin B-12 below 200 pg/ml (37).

Low vitamin B12 status has also been associated with impaired cognitive function (38) in relation to fluid intelligence in adolescents who had previously been long-term vegans and in relation to memory performance in adult women from the Australian population (39).

**Current recommended intakes and consumption of vitamin B12**

The Australian RDI for vitamin B12 is 2.0 mcg a day for men and women of all ages with an additional 1.0 mcg/day in pregnancy and 0.5mcg/day in lactation (15 ). The recent US revision of their Recommended Dietary Allowances (40) established an “Estimated Average Requirement” (EAR) of 2mcg/day for adult men and women of all ages with an RDA (RDA = EAR+2SD EAR) of 2.4mcg/day for all adults. However, for people over 51 years of age, they recommend that most of the 2.4mcg should be obtained by consumption of foods fortified with B12 or a B12 –containing supplement because of concerns with decreasing ability to absorb B12 from foods as people age.

An analysis of the National Nutrition Survey (27) using vitamin B12 food data base from the USA, gave an estimated intake of 6 mcg for adult males and 3.9 mcg for adult females.

**Protein**

Proteins are the fundamental structural compounds of the cell, antibodies, enzymes and many hormones. They are essential constituents of the nucleus and protoplasm of every cell and they are almost the sole form in which man can replace nitrogen. Twenty-three amino acids are used to construct proteins of which, eight are classified as essential since they must be supplied in the food.

Proteins vary in their digestibility. The protein from meats, poultry and fish is highly digestible, (90% or more), compared with a digestibility of 78% in beans and 86% in whole wheat (41).

Net protein utilisation which is a combined measure of digestibility and the efficiency of absorption of the amino acids (42) is generally higher for animal protein sources (NPU 0.75 to 0.8) compared with the NPU of many (but not all) plant foods (NPU 0.5-0.6).
Recommended and current intakes of protein

The current recommended intake of protein in Australia for adults is based on a value of 0.75g/kg/day (14). For men of all ages, 55g/day is recommended and for women 45g/day. In pregnancy, an additional 6g/day is recommended and in lactation, an additional 16g/day. The Australian RDI was developed in the late 1980s. The ongoing revision of RDAs in the USA has not yet re-assessed recommended protein intakes.

According to the 1995/6 National Nutrition Survey (25) men were consuming, on average, 109 g/day and women, 74 g/day. According to a CSIRO analysis of the NNS (27), red meat was the most significant source of protein in the Australian diet, providing 20% of protein in the diets of adult Australians.

Dietary Fats

Meats are often perceived as a major source of dietary fat and saturated fat. However, whilst some individual cuts or products can be relatively high in fat, an analysis of the National Nutrition Survey of Australia showed that meats do not contribute as much fat as commonly perceived. For instance, an analysis of the National Nutrition Survey of 1995/6 showed that red meat per se contributes an average of only 6% of the total fat in adults’ diets, 9% of the saturated fat and 12% of the unsaturated fats, mainly monounsaturated, as well as 17% of the cholesterol (27).

Excess dietary fat (and saturated fat) intake has been linked to a number of adverse health outcomes (see dietary fat and obesity guidelines). However, the confounding of fat intake with intake of certain components of this food group, notably meats, has led to some confusion in interpretation of epidemiologic data linking dietary components to chronic disease outcome, particularly in relation to cancer causation. In the USA, where much of the epidemiologic research data is sourced, the fat content of meat is considerably higher than that in Australia (43-45) and meats contribute more markedly to overall fat and saturated fat intake.

Three recent Australian reviews of the cancer epidemiology literature have largely exonerated red meats per se from a role in cancer causation and, in particular, colon cancer. A review by an expert panel of the role of red meat in colon cancer (46), concluded that the balance of epidemiologic evidence indicates that the prevailing levels of Australian lean red meat consumption are not linked with the development of cancer. However, caution is recommended in relation to charring or grilling of muscle protein foods. This conclusion is consistent with that of the National Health and Medical Research Council (1999) concerning diet and colorectal cancer, which recommends a reduction in total fat intake but makes no recommendation about meat intake (47) and with an earlier review on red meat and various cancers by Baghurst (48).

An expert review of red meat and health (49), endorsed by the National Heart Foundation and Dietitians Association of Australia also concluded that diets rich in lean red meat could still be low in fat and saturated fat and not adversely affect plasma cholesterol levels and that lean red meat could be included in management strategies for the prevention and treatment of obesity.
Omega-3 fats, found predominantly in fish, appear to have a number of beneficial actions notably in relation to brain development and function and cardiovascular health. A more detailed discussion about the role of the role omega fats in the diet is included in the dietary fat guideline. The intake of long chain omega-3 fatty acids has been estimated to be less than 200 mg/day for Australians (50) with most authorities recommending that the desired intake be between 214 mg/day and 650 mg/day (51). Fish and seafood are by far the richest sources of omega fats. Because of the relative amounts consumed, pasture-fed red meats are also a good contributor to overall intake in Australia (52). In vegetarian populations, the principal omega-3 fatty acid from vegetables is alpha-linolenic acid. Vegetarians have significantly lower plasma and platelet omega-3 levels (53,54), since alpha-linolenic acid is not as effective a source of long chain omega-3 fatty acids as is the direct consumption of EPA, DPA and DHA (54) from fish and meat.

Recommended intakes and current consumption of meat, poultry, fish and alternatives

The Australian Guide to Healthy Eating (AGHE) (55), based on the NHMRC’s Core Food Group model (56), recommends one to one and a half serves of this food group a day for men aged 19-60 years depending on the pattern of intake of other foods and one to one and a half serves for women 19-60 years, One and a half serves a day are recommended in pregnancy and two serves during lactation. A sample serve equates to 65-100g cooked meat or chicken or half a cup (cooked) of (dried) beans, lentils, chick peas or split peas or canned beans; 80-120g of cooked fish fillet; 2 small eggs; one third of a cup of almonds or peanuts or a quarter of a cup of sunflower or sesame seeds.

The AGHE recommends that red meat be eaten 3-4 times a week or high iron replacement foods will be required adding that this is especially true for girls, women, vegetarians and athletes.

SPECIAL GROUPS

Adolescent girls and women

Menstrual losses virtually double the iron requirement of women, compared to men. Adolescents have the added needs of growth. Iron balance under these circumstances is problematical and requires a substantial food intake, as well as an appropriate composition of meals. The National Nutrition survey data suggests that some women may be limiting overall food intake, possibly because of concerns with body weight. Increased activity would allow greater food intake, hence increasing the likelihood of adequate iron intake, whilst maintaining desirable body weight.

Pregnancy

Pregnant women are at higher risk of inadequate iron and zinc intakes because of their increased needs and the potential for morning sickness to reduce nutrient availability. Pregnant women who are vegetarians are at additional risk (57).

For adult women, each pregnancy represents a major drain on iron reserves. Women who have had several children over a relatively short time span are at high risk of iron depletion, especially if iron supplementation during their pregnancies was inadequate.
Infants born to women with low iron stores will themselves have low stores and, if breast-fed for prolonged periods, will be more likely to develop anaemia. Also, low iron status in early pregnancy is more likely to result in premature birth and low birth weight (58,59).

Low maternal serum zinc levels have been associated with congenital malformations, prematurity, foetal growth retardation (leading to low birth-weight babies) and maternal morbidity (28). However, the results have not always been consistent.

**Vegetarians**

A properly constructed vegetarian diet can be adequate in all nutrients and, indeed, some vegetarian communities have been shown to have health advantages over the general population, notably in the cardiovascular area, with reductions in risk factors such as plasma cholesterol, low antioxidant status, clotting factors and blood pressure (60-70). It is thus possible to construct a healthy diet without the use of foods derived from animal sources but there are several micro-nutrients for which meat, poultry and fish are the dominant and most bioavailable source and, as discussed above, great care needs to be taken with these nutrients if these foods are excluded. Care is also needed to include protein from diverse plant sources (legumes, nuts, cereals or dairy, if eaten) to attain the appropriate mix of amino acids.

Legumes have not been commonly eaten in Australia but provide a valuable source of protein, fibre and micronutrients not only for vegetarians but for the wider community. Included in this category are beans such as soybeans, kidney beans, broad beans and haricot beans as well as mature dried peas, lentils and chick peas.

**Athletes**

Recent heightened interest in the relation between iron status and performance of athletes has revealed an increased requirement of iron in that group mainly because of increased intestinal losses (71,72). Because of their overall requirements, female athletes are particularly vulnerable.

**PRACTICAL GUIDE**

This guideline specifically refers to inclusion of lean cuts of meat and poultry. Some meat and poultry products or dishes which are popular in Australia such as pies, sausages, crumbed and fried meats, poultry or fish or mettwurst/salami can have significant amounts of saturated fat either from the meat itself and/or other components such as the pastry. Whilst these foods can be included occasionally in a balanced diet, care needs be taken with the rest of the diet so as not to overconsume saturated fats. In addition, these types of meat and poultry products also do not provide the same level of iron, zinc and Vitamin B12 as the lean cuts. Removal of visible fat from meat and poultry cuts before cooking and selection of lean mince varieties can also help to limit fat intake.

Whilst consumption of a variety of foods from this food group are encouraged, the Australian Guide to Healthy Eating (55) recommends inclusion of red meat three to four times a week or high iron replacement foods will be needed. This is especially true for girls women and athletes. Inclusion of two to three meals of fatty fish per week is also recommended to obtain the necessary requirements of omega-3 fatty acids.
From this food group, vegetarians should choose from a variety of legumes, nuts and seeds to obtain protein, iron and zinc. Wholegrain or wholemeal cereals are also good sources of zinc and iron and supplemented varieties are available. Drinking fruit juice or eating fruit at the same meal will increase absorption of iron and zinc.

**CHANGES FROM EARLIER GUIDELINES**

In earlier editions, a guideline which encouraged the consumption of “iron-rich foods”, was included. In this revision, to more clearly define the concept of variety, to provide advice cohesive with the Australian Guide to Healthy Eating and to take a more consistent food-based approach, this guideline has been replaced by one encouraging inclusion of lean meats and poultry, fish or their alternatives, with an emphasis on their value as a source of dietary iron.

**RELATIONSHIP TO OTHER GUIDELINES**

*Limit saturated fat and moderate total fat intake*

Lean meats and poultry and low fat cooking methods are recommended. Australian red meat cuts are generally much leaner than meat from countries such as the United States.

*Prevent weight gain by being physically active and eating according to your needs*

Obesity is increasing in many countries throughout the world. Whilst many genetic, environmental and lifestyle factors contribute to this, dietary fat intake can be a major factor in the development of obesity (73). Choice of low fat varieties and cooking techniques are therefore encouraged.

*Care for your food and keep it safe to eat*

Illness due to food borne, pathogenic bacteria is a public health issue. All foods are potential vectors of pathogens. In Australia the risk of food borne illness in primary food industries is managed across the food chain, with industry, government and consumers sharing responsibility for the delivery of microbiologically safe products. Nevertheless, some foods from this food group have been implicated in outbreaks of food borne disease (74,75) and constant vigilance is required.

**EVIDENCE**

The scientific rationale for this guideline was based on a variety of evidence sources including a well-designed randomised control trial (Level II evidence) relating to iron supplementation and cognition (ref 5), a meta-analyses of case-control and cohort studies (Level III evidence) assessing the effects of red meat on cancer (ref 48); to iron deficiency and cognition (refs 6,10,11), vitamin B12 and cognition (ref 39), red meat consumption and cardiovascular disease risk factors (ref 63) and the effect of various foods, drinks or nutrients on iron or zinc bioavailability and absorption (refs 17, 20,21,22,23,29,31) or diet on homocysteine status (ref 34,35,36).

In addition, evidence was obtained from a number of cross-sectional population studies as well as human experimental evidence relating to bioavailability and nutrient requirements and intakes and expert reviews of selected issues.
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14. NHMRC (1991) Recommended Dietary Intakes for use in Australia AGPS, Canberra


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INCLUDE REDUCED FAT DAIRY FOODS AND/OR ALTERNATIVES

DEFINITIONS

Dairy foods or alternatives
This term includes milks (fresh, long-life or reconstituted dried), evaporated milk, cheese, yoghurt and custards under the term "dairy foods" as well as calcium-fortified soy drinks. Sardines and other fish eaten with bones, and certain nuts such as almonds also contain moderate to good amounts of calcium, and in this respect can be considered as "alternatives".

Reduced Fat
Reduced fat products generally contain 75% (or less) of the fat contained in the equivalent full-fat product.

Osteoporosis
Osteoporosis is a condition of low bone mass. It can lead to greater bone fragility and increased risk of fractures(1). Most fractures in older adults are related to osteoporosis; in young adults trauma is the primary cause of fractures (2). Clinically, osteoporosis is measured in terms of bone mineral density that is below the age-adjusted reference range. Individuals are considered osteoporotic if their bone mineral density is 2.5 SD or more below the young adult mean (3). This definition identifies about 30 per cent of all post-menopausal women as having osteoporosis and, of these, more than 50 per cent will have suffered a previous fracture (3). Osteoporosis is also of increasing importance in men (76).

BACKGROUND

Dairy foods are a major source of protein and a number of vitamins and minerals in the Australian diet and milk is the most complete of all foods, containing nearly all the constituents of nutritional importance to humans. In particular, however, the dairy foods are the richest source of calcium in the Australian diet (4). Few other foods provide such a readily absorbable and convenient source of this nutrient.

Calcium is required for the normal development and maintenance of the skeleton (5). It is stored in the teeth and bones, where it provides structure and strength.

In western cultures, low intakes of calcium have been associated with a condition of low bone mass called osteoporosis which often results in bone fracture and is one of the major causes of morbidity amongst older Australians, particularly women.

In light of the ageing of Australia’s population, it is estimated that hospital admissions for osteoporotic fractures will increase by 84 per cent by 2011 (6). The Dubbo Osteoporosis Epidemiology Study found that after the age of 60 years approximately 60 per cent of women and 30 per cent of men suffer from osteoporotic fractures (7). The most common fracture sites related to osteoporosis are the hip, vertebrae and wrist, but hip fractures have the greatest overall public health impact. Considerable morbidity and mortality are associated with hip fractures and they lead to a substantial decline in physical function (8).
In 1992, it was estimated that the resulting costs for rehabilitation and outpatient treatment of such fractures were some $779 million (1)

<table>
<thead>
<tr>
<th>Food</th>
<th>Energy (kJ)</th>
<th>Protein (g)</th>
<th>Fat (g)</th>
<th>Sat fat (g)</th>
<th>Calcium (mg)</th>
<th>Sodium (mg)</th>
<th>Vit B2 (mcg)</th>
<th>Vit B12 (mcg)</th>
<th>Vit A equiv (mcg)</th>
<th>Zinc (mg)</th>
<th>Iron (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk, fluid, whole</td>
<td>272</td>
<td>3.3</td>
<td>3.8</td>
<td>2.5</td>
<td>114</td>
<td>51</td>
<td>0.2</td>
<td>0.3</td>
<td>48</td>
<td>0.4</td>
<td>0.1</td>
</tr>
<tr>
<td>Milk, fluid, reduced fat (fat 1 - 2%)</td>
<td>203</td>
<td>3.9</td>
<td>1.4</td>
<td>0.9</td>
<td>137</td>
<td>58</td>
<td>0.2</td>
<td>0.3</td>
<td>14</td>
<td>0.4</td>
<td>0.1</td>
</tr>
<tr>
<td>Milk, fluid, low fat (fat &lt; 1.1%)</td>
<td>190</td>
<td>4.6</td>
<td>0.2</td>
<td>0.1</td>
<td>160</td>
<td>66</td>
<td>0.3</td>
<td>0.3</td>
<td>5</td>
<td>0.5</td>
<td>0.1</td>
</tr>
<tr>
<td>Milk, fluid, skim or nonfat (fat &lt; 0.16%)</td>
<td>145</td>
<td>3.6</td>
<td>0.1</td>
<td>0.1</td>
<td>123</td>
<td>54</td>
<td>0.2</td>
<td>0.3</td>
<td>0</td>
<td>0.4</td>
<td>0.1</td>
</tr>
<tr>
<td>Yoghurt, regular fat, plain</td>
<td>304</td>
<td>4.7</td>
<td>3.4</td>
<td>2.2</td>
<td>171</td>
<td>77</td>
<td>0.3</td>
<td>0.3</td>
<td>39</td>
<td>0.5</td>
<td>0.1</td>
</tr>
<tr>
<td>Cream, pure (fat &gt; 35%)</td>
<td>1660</td>
<td>1.9</td>
<td>42.8</td>
<td>28.3</td>
<td>69</td>
<td>27</td>
<td>0.2</td>
<td>0.1</td>
<td>580</td>
<td>0.3</td>
<td>0.1</td>
</tr>
<tr>
<td>Cheese, cheddar</td>
<td>1690</td>
<td>25.4</td>
<td>33.8</td>
<td>21.5</td>
<td>775</td>
<td>656</td>
<td>0.0</td>
<td>0.2</td>
<td>390</td>
<td>3.6</td>
<td>0.0</td>
</tr>
<tr>
<td>Cheese, cottage</td>
<td>512</td>
<td>15.3</td>
<td>5.8</td>
<td>3.8</td>
<td>73</td>
<td>200</td>
<td>0.0</td>
<td>0.2</td>
<td>65</td>
<td>0.5</td>
<td>0.0</td>
</tr>
<tr>
<td>Soy beverage, unfortified, unflavoured</td>
<td>164</td>
<td>2.5</td>
<td>2.1</td>
<td>0.3</td>
<td>13</td>
<td>59</td>
<td>0.0</td>
<td>1.5</td>
<td>0</td>
<td>0.3</td>
<td>0.4</td>
</tr>
<tr>
<td>Soy beverage, fortified, unflavoured</td>
<td>260</td>
<td>3.5</td>
<td>3.5</td>
<td>0.4</td>
<td>116</td>
<td>59</td>
<td>0.2</td>
<td>0.5</td>
<td>39</td>
<td>0.2</td>
<td>0.5</td>
</tr>
<tr>
<td>Soy beverage, low fat, unfortified</td>
<td>110</td>
<td>2.5</td>
<td>0.3</td>
<td>0.0</td>
<td>12</td>
<td>40</td>
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<td>0.0</td>
<td>0</td>
<td>0.3</td>
<td>0.4</td>
</tr>
<tr>
<td>Soy beverage, low fat, fortified</td>
<td>175</td>
<td>3.5</td>
<td>0.5</td>
<td>0.1</td>
<td>110</td>
<td>40</td>
<td>0.2</td>
<td>0.3</td>
<td>39</td>
<td>0.2</td>
<td>0.5</td>
</tr>
<tr>
<td>Tofu, cooked, fat not added in cooking</td>
<td>304</td>
<td>8.1</td>
<td>4.2</td>
<td>0.6</td>
<td>330</td>
<td>7</td>
<td>0.0</td>
<td>0.0</td>
<td>0</td>
<td>0.7</td>
<td>1.2</td>
</tr>
<tr>
<td>Muesli, average comp.</td>
<td>1459</td>
<td>9.2</td>
<td>8.9</td>
<td>2.6</td>
<td>56</td>
<td>115</td>
<td>0.7</td>
<td>0.0</td>
<td>26</td>
<td>1.9</td>
<td>6.1</td>
</tr>
<tr>
<td>Sardine, canned in oil, drained</td>
<td>952</td>
<td>21.8</td>
<td>15.7</td>
<td>5.1</td>
<td>380</td>
<td>608</td>
<td>0.3</td>
<td>28.0</td>
<td>65</td>
<td>1.8</td>
<td>2.7</td>
</tr>
<tr>
<td>Sardine, canned in water, drained</td>
<td>767</td>
<td>21.8</td>
<td>10.7</td>
<td>2.8</td>
<td>380</td>
<td>608</td>
<td>0.3</td>
<td>28.0</td>
<td>65</td>
<td>1.8</td>
<td>2.7</td>
</tr>
<tr>
<td>Seed, sesame</td>
<td>2450</td>
<td>22.2</td>
<td>55.6</td>
<td>6.8</td>
<td>62</td>
<td>60</td>
<td>0.2</td>
<td>0.0</td>
<td>1</td>
<td>5.5</td>
<td>5.2</td>
</tr>
<tr>
<td>Almond, raw</td>
<td>2455</td>
<td>20.0</td>
<td>55.3</td>
<td>3.6</td>
<td>235</td>
<td>6</td>
<td>1.2</td>
<td>0.0</td>
<td>2</td>
<td>3.6</td>
<td>3.5</td>
</tr>
<tr>
<td>Almond, roasted</td>
<td>2618</td>
<td>18.6</td>
<td>60.5</td>
<td>4.2</td>
<td>218</td>
<td>6</td>
<td>1.0</td>
<td>0.0</td>
<td>1</td>
<td>3.3</td>
<td>3.3</td>
</tr>
<tr>
<td>Spinach, English, cooked,</td>
<td>78</td>
<td>3.0</td>
<td>0.4</td>
<td>0.0</td>
<td>50</td>
<td>20</td>
<td>0.2</td>
<td>0.0</td>
<td>370</td>
<td>0.6</td>
<td>3.0</td>
</tr>
<tr>
<td>Bean, haricot, dry, cooked</td>
<td>385</td>
<td>8.2</td>
<td>0.7</td>
<td>0.1</td>
<td>57</td>
<td>15</td>
<td>0.1</td>
<td>0.0</td>
<td>0</td>
<td>0.9</td>
<td>2.1</td>
</tr>
</tbody>
</table>

**SCIENTIFIC BASIS**

Although dairy foods are a valuable source of a number of nutrients including protein, retinol, riboflavin and vitamin B12 (9), the major rationale for inclusion of this food group as part of the dietary guidelines, is because of its role as a rich source of calcium.

The scientific basis of this guideline is thus centred on the role of dairy foods as a key source of readily absorbable dietary calcium and the role of calcium, in conjunction with a number of other factors, in attaining peak bone mass and in the prevention of osteoporosis.

**The importance of peak bone mass**

The skeleton is in a rapid phase of growth throughout childhood and adolescence. From birth to puberty the skeleton increases in mass about 7-fold and a further 3-fold in adolescence (10). The best protection against age-related bone loss and subsequent
fracture risk is considered to be the attainment of a high peak bone mass at skeletal maturity. Peak bone mass is attained between 19 and 30 years of age. It is a product of the interaction between endogenous (hereditary, endocrine) and exogenous (nutritional, weight-bearing exercise) factors. Calcium seems to be the most important nutritive factor that determines peak bone mass in young adults (5,10).

It is important to achieve peak bone mass as the greater the mass before age-related loss begins, the less likely it will decrease to levels where fractures may occur (2,5). During the adolescent growth spurt, the required calcium retention is two to three times higher than the average figures required for the development of peak bone mass (11). Restricted food intakes at this age are therefore of particular concern and young girls with very heavy exercise regimes and who restrict total food intake, are at particular risk of developing an inadequate peak bone mass. During the period that peak bone mass is being achieved, it is necessary to ingest sufficient calcium to maintain positive balance. This quantity will vary from person to person depending on the individual efficiency of intestinal calcium absorption. Once peak bone mass is achieved, it is maintained without much change for 10-20 years. Daily requirements at this stage are about two thirds of that required to attain peak mass as bone building has been completed and intestinal absorption of calcium is normal. However, men and women lose bone at a constant rate of 0.2 to 0.5% per year starting at ages 40-45. For approximately 10 years immediately before, during and after menopause (12), women lose bone more rapidly than men (2-5% per year). The rate of loss in menopausal women returns to the slower rate shared by the sexes after this 10 year period.

**Calcium balance and bone mass**

The calcium content of the skeleton at any period during life represents previous absorption from the diet and retention in the bone, after obligatory losses have been met. Almost all of the body’s calcium reserve is stored in the skeleton and is indirectly affected by the dietary calcium intake and the amount of calcium lost from the body, either in urine, sweat or as calcium not absorbed from the intestine. Even though a low calcium intake will affect the size of the skeletal reserve of calcium, bone mass is also affected by other non-nutritional factors such as genetics, hormonal status and weight-bearing exercise (5). A low dietary intake of calcium will not necessarily result in a low bone mass, but it will increase the risk of this occurring, especially if the low intake is maintained long term or if it is accompanied by other dietary habits which lead to increased obligatory calcium excretion such as high protein or salt intakes.

**Dietary calcium, bone loss and fractures**

If, as postulated, dietary calcium deficiency is related to bone loss, individuals with low levels of calcium consumption should have lower bone mineral density than do those having higher levels of calcium intake. A meta analysis of 27 cross-sectional, two longitudinal and four intervention studies assessing the effect of calcium intake on bone mass in young and middle aged females and males (13) concluded that overall there was evidence that calcium intakes were positively associated with bone mass in premenopausal women although calcium intake alone did not account for a large amount of the variance in bone mass.
Low bone density is associated with an increased risk of fracture; the lower the bone density the greater the risk (1). If an absolute or relative dietary calcium deficiency per se contributes to the development of osteoporosis, therapy with calcium should improve not only bone density and bone loss but also fracture rates.

At older ages, there is evidence that a high calcium intake will slow the rate of bone loss and may reduce the risk of fracture. A number of randomised trials have shown that calcium supplements are effective in slowing bone loss in older women (14-17) but a meta-analysis of nine random controlled trials of the first and second year effects in trials of calcium supplementation on bone density in post menopausal women (18) concluded that whilst the rate of bone loss was less in supplemented women in the first year of treatment it was not in the second year. Only a limited number of randomised controlled trials of calcium supplementation have used fracture end-points (19-22). These studies did, however, consistently show a reduction in risk of 26 to 70 per cent. A systematic review of fourteen studies including randomised and non-randomised control trials, case-control studies and cohort studies also concluded that calcium supplements and dietary calcium probably reduce risk of osteoporotic fractures in women (23).

The effects of menopause, ageing and vitamin D on calcium balance

Intestinal calcium absorption and the ability to adapt to low calcium diets decline with ageing (24,25). This results in an increased calcium requirement needed to maintain calcium balance and skeletal integrity. For women, superimposed on the effects of ageing is the effect of menopause. Calcium balance deteriorates at menopause and this deterioration may be due to a decline in intestinal calcium absorption and/or an increase in urinary calcium excretion. Heaney et al (26) demonstrated that the dietary calcium requirement needed to prevent negative calcium balance was increased some 50% by the menopause and the inter-relationship of oestrogen deficiency and intestinal calcium absorption was studied by Gallagher et al (27) who demonstrated that oestrogen therapy increases both calcium absorption and 1,25 (OH)2D3 concentrations. The relative importance of hormone replacement, increased dietary intake and/or calcium supplementation in prevention of osteoporosis is not yet clear.

Impaired intestinal calcium absorption with ageing and menopause may in part be related to changes in vitamin D metabolism. This maybe a consequence of inadequate dietary intake of vitamin D, less efficient intestinal absorption, less efficient skin synthesis of vitamin D, inadequate exposure to ultraviolet light or a lessening ability of kidneys around menopause or in ageing, to produce the major biologically active metabolite of vitamin D (1,25- dihydroxy vitamin D).

Whatever the cause, 1,25-dihydroxy vitamin D the major regulator of intestinal calcium absorption is present in lower concentrations in the elderly and in menopause and may be lower in patients with osteoporosis than age matched control subjects. The main food sources of vitamin D in Australia are margarines fortified with vitamin D, fatty fish and eggs (28).

Some studies suggest that Vitamin D supplementation is effective in preventing fractures only in people who have marginal vitamin D serum levels (26, 29,30). Details of these studies are given in the "Dietary Guidelines for Older Australians" (31). As a result of these considerations, vitamin D supplementation of housebound, older people and others
not exposed to sunlight was first recommended by the NHMRC’s Nutrition and Public Health Committees in 1989(32).

Some factors affecting calcium needs

**Bioavailability**
For food sources of calcium, content is of greater importance than bioavailability. Calcium absorption efficiency is similar from most foods but calcium may be poorly absorbed from foods rich in oxalic acid (e.g., spinach, rhubarb, beans) or phytic acid (seeds, nuts, grains, raw beans and soy isolates). Soybeans have large amounts of phytate but absorption of calcium is still quite high (33). Compared to milk, calcium absorption from dried beans is about half and from spinach, one tenth. Bioavailability from non-food sources e.g., supplements depends on the dosage and whether taken with a meal. In standardised studies of 250mg calcium supplements given with a breakfast meal, calcium citrate malate gave a fractional absorption rate of 35%, calcium carbonate 27%; and tricalcium phosphate 25%, compared to a rate for calcium from milk of 29% (34-37). Efficiency of absorption of calcium from supplements is greatest at doses of 500mg (38, 39).

**Physical activity**
It is generally accepted that weight-bearing exercise determines the strength, shape and mass of bone (40) but the mechanisms are still not clear. It is also unclear whether calcium intake influences the benefit gained from exercise. With complete immobilisation, rapid bone loss occurs despite high calcium intake (41). In a calcium intervention in children, calcium and exercise both affected bone mineralisation but the effects appeared to be independent (42). A review (43) of sixteen studies in adults, mostly women, concluded that high calcium intakes (over 1000mg) enhanced bone mineral density benefits of exercise to different degrees in various parts of the skeleton. The recent review of calcium requirements undertaken for development of the US Dietary Reference Values (44) concluded there was insufficient evidence to justify different calcium requirements for people with varying activity levels.

**Sodium**
Sodium and calcium excretion are linked in the kidney tubules. High salt intake increases salt absorption and urinary sodium resulting in increased obligatory loss of urinary calcium. 500mg of sodium draws 10mg of calcium into the urine in postmenopausal women (45). In children and adolescents, urinary sodium is an important determinant of urinary calcium excretion (46,47) but no association has been shown between salt intake or excretion and skeletal development in children. However, one longitudinal study in postmenopausal women showed a link between high urinary sodium and increased hip bone loss (48). No study has yet shown a direct link between sodium intake and bone loss or fracture rates. The US DRI committee for calcium requirement (44) concluded that despite the relatively high salt intake in the US, the available evidence did not warrant different calcium requirements being set for people with varying sodium intakes.

**Protein**
Protein increases urinary calcium excretion but its effect on calcium retention is unclear. Walker and Linkswiler (49) found that urinary calcium increased by about 0.5mg for each gram of dietary protein over about 47g/day.
However, low dietary protein intakes (below 34g/day) have been shown to be associated with poor health and poor recovery from osteoporotic hip fracture (50). The US review of calcium requirements (44) concluded that evidence of the effect of protein intake on calcium requirement was not sufficient to recommend different calcium intakes for varying intakes of protein.

**Calcium and other chronic diseases**

**Hypertension**

Many studies have investigated links between calcium and hypertension. In a review of 22 randomised intervention trials (51), calcium supplementation was found to reduce systolic blood pressure slightly in hypertensives but not affect normotensives, diastolic blood pressure was not affected in either group. In another study using a diet with increased low fat dairy products, fruit and vegetables but with reduced total and saturated fat (52), blood pressure fell in both normo and hypertensives. The increased dairy consumption provided an almost three fold increase in calcium from 443mg to 1265mg/day. However, the effect of calcium on blood pressure has generally been found to be modest and variable across populations.

**Colon cancer**

Although suggestions have been made about a link between calcium and colon cancer, the evidence is weak. Observational and case-control studies have had mixed results (53-55). Greater mucosal proliferation is seen in patients at high risk of colon cancer (56-58) and one study has shown increased mucosal proliferation with calcium supplements (59). However, another showed a decrease in proliferation (60).

**Requirements and Recommended intakes for calcium**

Calcium requirements are largely determined by skeletal needs, which increase during periods of rapid growth (such as childhood and adolescence), during pregnancy and lactation, and in later life. Needs can be assessed in a number of ways including balance studies, a factorial estimate approach and changes in bone mineral density or content (24).

In Australia, the calcium requirement has been used to estimate the Recommended Dietary Intake (RDI), which is traditionally set to meet the needs of 95 per cent of the population (61).

The NHMRC’s current recommended intake of dietary calcium, which was set over a decade ago in 1991, increases from 800 milligrams a day in pre-menopausal women to 1000 milligrams a day in post-menopausal women (61). This is to account for the accelerated loss of calcium from the skeleton after menopause. In pregnancy, an additional 300mg/day (total 1100mg/day) is currently recommended and, in lactation, an additional 400 mg/day (total 1200mg/day).

For men, the Australian RDI for calcium is 800 milligrams a day for all ages. There is some evidence, however, that this may not be sufficient in older men since it does not take into account the age-related changes in calcium and vitamin D metabolism. This has gained some support from the recent review of dietary allowances in the USA.
The ongoing revision of the USA Recommended Dietary Allowances (RDA) is being undertaken by a group of committees under the auspices of the Institute of Medicine (24). It uses a new multi-stage form of expression for recommendations, similar to that first used in the UK in the early 1990s (62). This includes an evidence-based determination of an "Estimated Average Requirement (EAR)" for individuals, the mean and variance of which is used to derive the more familiar "Recommended Dietary Allowance" for individuals (RDA = EAR + 2SD_{EAR}).

The committee assessing calcium (24) concluded that there was insufficient evidence to establish an evidence-based "Estimated Average Requirement" for calcium, for any age or gender group. As such, they did not produce Recommended Dietary Allowances for calcium but did estimate what they term an "Adequate Intake (AI)" figure for each age and gender group. An "AI" for a nutrient is set as an alternative to the RDA where data is considered to be insufficient (or not certain enough) to develop a reliable variance estimate for the population. The “AI” is believed to cover the needs of most people in the population but the percentage of the population covered by this recommended intake level cannot be specified with confidence.

The "AI" for calcium for adults from 19-50 years, was set at 1,000 mg/day and that for adults from 51 years on was set at 1,200 mg per day. The figure set was the same for men and women within each age category. For pregnancy and lactation, the figure was set at 1,000 mg a day (ie no additional requirement from normal because of increased absorptive capacity) unless the mother was 18 years or under, in which case it was set at 1,300 mg/day to account for ongoing maternal needs for growth.

It is of interest to note that the British recommendations (62) of 1991 (termed "Reference Nutrient Intakes" - equivalent to the American RDAs) also set the same figure for males and females but at a much lower level than the US recommendations (700mg/day for all adults irrespective of age and gender). The British also have no increased recommendation for pregnancy but do recommend an additional 550mg/day for lactation.

One group in Australia who may need to pay particular attention to calcium requirements are recent migrants from countries where the background diet is traditionally lower in protein and salt, than in Australia, and where everyday physical activity may be greater (such as certain Asian countries). If current Australian dietary and lifestyle patterns are adopted by such migrants, this will also increase their calcium requirements.

**Current intakes of dairy foods, calcium and other key nutrients in dairy foods**

The most recent National Nutrition Survey (NNS) was undertaken in 1995/6 using a 24 hr recall technique supplemented by a qualitative food frequency questionnaire (4). On the day of the 1995/6 National Nutrition Survey, 93% of subjects consumed foods from the dairy category with the average intake for what was termed the "milk and milk products" group being 322g for men over 19 years of age and 258g for women over 19 years. Intakes fell with age in adult men but remained relatively stable in women. On the day of the survey, 38% of men and 45% of women consumed less than one serve of dairy foods with only 16% of men and 10% of women consuming three serves or more.

The NNS survey, found that the mean daily intake of calcium in men over 19 years, was 946 mg/day and, in women, 749 mg/day. In men, intakes fell with age ranging from 1,101
in males aged 19-24 years, through to 989mg/day in those 25-44 years, 885 mg/day in those 45-64 years and 796 mg/day in those over 60 years. In women, there was no clear age trend with younger adults (19-24 years) consuming 750 mg/day on average, compared to 762mg/day in those who were 25-44 years of age and 769 mg/day in the 45-64 years group but falling off somewhat to 686mg/day in those over 60 years. Dairy products provided just over half the calcium in the diets of adults with milk contributing 30% of total calcium and cheese, 12%.

The National Nutrition Survey (4) also included information on the intake of people who were taking calcium supplements on the day of the survey. Supplement use was much greater among women especially in the older age groups. 1.6% of women aged 19-24 years took a calcium supplement rising to 4.3% of those aged 25-44 years, 9.8% aged 45–64 years and 10.9 % aged 65 and over; for men, only 0.4% of 19-24 year olds took calcium supplements, rising to 1.1% at age 25-44 years, 1.6% aged 45–64 years and 2.4% aged 65 and over. This is probably a reflection of women’s greater awareness of calcium’s role in osteoporosis prevention.

As well as their role as a source of calcium, the NNS survey showed that dairy foods also provided 12% of dietary energy, 15% of protein, 13-14% of vitamin A (25% of retinol), 30% of the riboflavin (vitamin B2), 27% of the vitamin B12 (64) and 13% of the zinc. The importance of protein, zinc and B12 in the diet is discussed in more detail in the chapter on lean meats, poultry, fish and alternatives. Retinol is the preformed form of vitamin A that is found only in foods of animal origin. However, beta-carotene from plant sources can be converted in the body to retinol. It is essential for maintaining epithelial integrity. Deficiency can lead to a range of eye conditions ranging from inability to see in dim light through to conditions causing blindness. Riboflavin is a B vitamin that is important in cell respiration. Deficiency can lead to oedema of the pharynx and oral mucosa, cheilosis, glossitis, angular stomatitis, conjunctivitis, corneal vascularisation and certain forms of anaemia. Deficiency has been documented in both industrialised and developing nations and across varying demographic groups (64,65).

The dairy category also provide 17% of total fat and 27% of saturated fat, emphasising the need to promote the low or reduced fat varieties. More information on dietary fats and their health effects, can be found in the dietary fats guideline chapter.

SPECIAL GROUPS

Because of the importance of obtaining a good peak bone mass, it is essential that intakes of calcium in adolescence are maintained. However, data from the Australian NNS indicate that some 36% of 12-15 year old girls and 44% of 16-18 year old girls had less than one serve of dairy foods on the day of the survey. For boys the figures were 23% and 21% respectively.

Amenorrhea resulting from anorexia is associated with lowered calcium absorption, higher urinary calcium excretion and a lower rate of bone formation (66). Exercise-induced amenorrhea results in reduced calcium retention and lower bone mass (67,68).

Lactose intolerance is high in Asian communities (85%) but relatively low in Caucasians (10%). Small amounts can be tolerated but lactose-free dairy products are now available.
Lactose intolerant people will often avoid milk products although avoidance may not be necessary.

Consumption of vegetarian diets may influence calcium needs because of their relatively high oxalate and phytate content. However, vegetarian diets produce metabolisable anions that lower urinary calcium excretion and on balance, lacto-ovo-vegetarians appear to have similar calcium intakes to omnivores (69, 70, 71) and similar urinary excretion (74, 75) and one 5 year study in postmenopausal lacto-ovo-vegetarians and omnivores with similar calcium intakes lost radius bone mineral density at similar rates (71). Bone data on strict vegetarians are not available.

**PRACTICAL ASPECTS OF THIS GUIDELINE**

The Australian Guide to Healthy Eating (74), which was based on the NHMRCs Core Food Group Analysis (75), recommends 2-3 serves of dairy foods or alternatives a day for women and 2-4 serves for men where a serve is equivalent to a cup of milk, half a cup of evaporated milk, 40g of cheese or 200g of yoghurt or equivalent (see below). The 1995/6 National Nutrition Survey indicates that some 42% of adults consumed less than one serve of dairy foods on the day of the survey.

Although rich in calcium, dairy foods are also high in saturated fat, so it is recommended that reduced-fat dairy foods or alternatives be chosen. A variety of calcium-enriched milks that are also low in fat are readily available. Low and reduced fat yoghurts and cheeses are also available but low-fat soft cheeses like cottage and ricotta have very little calcium and cannot be counted as a "serve" of dairy food although they may add variety to a low fat diet.

Low lactose milks and dairy products are now available for those with lactose intolerance. If people cannot or do not want to eat dairy foods, the following are examples of what can be substituted in terms of calcium equivalents:

- A cup of calcium-fortified soy beverages containing 100 mg calcium/100 ml
  (non-fortified soy beverages do not provide sufficient calcium)
- Calcium fortified tofu
- A cup of almonds
- Five sardines or half a cup of pink salmon (with bones).

**CHANGES FROM EARLIER GUIDELINES**

In the earlier dietary guidelines for adults, children or older Australians, one guideline on variety in food choice and others on encouraging consumption of calcium-rich and iron-rich foods were included. In 1998, the Australian Guide to Healthy Eating (AGHE) was developed by the Department of Health, based on the NHMRC Core Food Groups analysis. The Core Food Group analysis modelled the general eating patterns required in the community to achieve the Recommended Dietary Intakes for certain energy intake levels.

For this revision of the Dietary Guidelines, it was felt that the former “variety” guideline should be more closely linked to the AGHE which recommends daily consumption of a variety of foods from five basic food groups, fruits; vegetables and legumes; breads,
cereals and grains; dairy and alternatives and meats, fish poultry and alternatives. Guidelines about consumption of fruits, vegetables and the breads, grains, cereal group were already included in earlier guidelines. In this revision, guidelines and background papers have been included for the dairy and alternatives and meats, poultry and fish and alternatives groups. It was also felt that a food-based, rather than nutrient-based, approach to guidelines would be more consistent with international trends in setting of dietary guidelines. The issue of the importance of including calcium-rich foods in the diet are therefore included within the dairy foods and alternative guideline and that for iron-rich foods in the meat, poultry, fish and alternatives guideline.

RELATIONSHIP TO OTHER GUIDELINES

Enjoy a wide variety of nutritious foods
Dairy foods and alternatives are recommended as part of a varied diet to achieve the balance of nutrients required for optimal health. The NHMRC Core Food Group Analysis (75) confirms a central role for dairy foods in the Australian diet in this context. The Australian Guide to Healthy Eating includes the dairy group as one of its five core food groups (73).

Choose foods low in salt
As discussed above, there is evidence that high intakes of sodium chloride increase urinary calcium loss. Conservation of calcium is thus an additional reason for following the salt guideline. This is more important for older people, whose ability to absorb dietary calcium may be impaired.

Limit saturated fat and moderate total fat intake
The 1995/6 National Nutrition Survey (4) showed that dairy foods contributed some 17% of total fat and 27% of saturated fat to the diet. As dairy foods are valuable sources of other nutrients, it is more appropriate to choose low or reduced fat varieties of dairy foods rather than reducing overall dairy intake in order to limit fat intake. If full fat cheeses are used these serves should be limited to 3-4 times a week.

Prevent weight gain by being physically active and eating according to your needs
As noted earlier, regular, weight-bearing exercise is also an important component in bone mineralisation. The encouragement to participate in regular physical activity from early childhood will not only contribute to a healthy body weight but also to peak bone mass.

CONCLUSION

The health costs associated with hospital admissions for osteoporotic fractures are high in Australia. An adequate intake of calcium will help delay bone loss and the onset of osteoporosis and so reduce the number of related fractures in older people. Dairy products are the most reliable source of calcium; they are readily available and convenient to use. If foods that are high in calcium are part of daily diet the physiological and social costs associated with a low-calcium diet will be reduced.
EVIDENCE

The scientific rationale for this guideline was based on a variety of evidence sources including some meta-analyses of randomised controlled trials (Level I evidence) concerning calcium supplementation and bone density (ref 18) and calcium and blood pressure (ref 51) and well-designed individual randomised control trials (Level II evidence) relating to calcium supplementation and bone loss and bone density (refs 14,15,16,17, 20,21,22), to vitamin D and fracture (ref 29), calcium and colorectal cancer (ref 60).

Level III evidence included a cohort study assessing the effects of calcium on colorectal cancer (ref 53), a meta-analysis of case-control and cohort studies relating to calcium intake and bone mass (ref 13); evidence of vitamin D and bone density (30), sodium and calcium and bone density (48), dietary supplementation and fractures (50) and dietary patterns and blood pressure (52).

In addition, evidence was obtained from a number of cross-sectional and population studies as well as human experimental evidence relating to bioavailability and nutrient requirements and intakes.
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DRINK PLENTY OF WATER

BACKGROUND

Water is an essential nutrient for life. All biochemical reactions occur in water. It fills the spaces in and between cells and helps form structures of large molecules such as protein and glycogen. Water is also required for digestion, absorption, transportation and as a solvent for nutrients, elimination of waste products and thermoregulation (1). To be properly hydrated, the average adult man needs about 3,000 mL of fluid a day and the average woman at least 2,200 mL (2). Solid foods contribute approximately 1,000 mL water with an additional 250 mL coming from the water of oxidation (3). The remainder needs to come from water and/or other fluids.

The normal daily turnover of water is approximately 4% of total body weight in adults (3). Several factors increase the possibility of chronic, mild dehydration, including a poor thirst mechanism (4,5), dissatisfaction with the taste of water (6,7), consumption of common diuretics such as caffeine (8) and alcohol, participation in exercise (9) and environmental conditions (5). Dehydration of as little as 2% loss of body weight results in impaired physiological responses and performance (10-16). Fluid consumption can also have an effect on urinary stone disease (17-20), cancer of the breast (21), colon (22), urinary tract (23,24) and bladder (25) obesity (26,27) mitral valve prolapse (28), salivary gland function (29), mental performance (30) and overall health in the elderly (4).

No estimate is available in Australia for the health cost of inadequate fluid intake.

SCIENTIFIC BASIS

Body water

Water accounts for one half to four-fifths of body weight depending on lean body mass. On average, men have a higher lean body mass than women and as a percentage of body mass, body water is higher in men than in women and falls in both with age (2).

Water is an essential nutrient because it is required in amounts that exceed the body’s ability to produce it. Human requirements for water are related to metabolic needs and is highly variable depending to some extent on individual metabolism. However, the body must retain a minimal amount to maintain a tolerable solute load by the kidneys. Even without perspiration, the normal turnover is approximately 4% of total body weight in adults. In a 70kg adult this is equivalent to 2500-3000 mL/day (3). Water loss from lungs and skin (insensible losses) are responsible for half the total water turnover (2), are sensitive to environmental conditions, and can be increased at high temperatures, high altitude and low humidity. During summer, when heat stress may be high, water depletion can lead to heat exhaustion, loss of consciousness and heat stroke (31,16). Unfit, overweight older people may be especially at risk, particularly if they are subjected to strenuous exercise. Losses from urine and stool account for the rest of the total losses.
Influence of hydration on health and disease

The reported health effects of chronic mild dehydration and poor fluid intake include:

- Increased risk of kidney stones (17-20)
- Increased risk of urinary tract cancers (23,24,25)
- Increased risk of breast cancers (21)
- Increased risk of colon cancer (22)
- Increased risk of childhood obesity (26,27)
- Diminished physical performance (10-16)
- Diminished mental performance (30)
- Diminished salivary gland function (29)
- Increased risk of mitral valve prolapse (28)

The scientific basis of some of these reported health risks is discussed below.

Renal calculi or stones

Data from the US show that approximately 12-15% of the general population will form a kidney stone at some time (32,33). There are many risk factors for the formation of kidney stones including age, gender, heredity, occupation, social status, geographic location, climate and diet. Of these, diet is the only one that is readily modifiable. Stone prevalence is higher in populations with low urinary volume (17-20, 32-36). Lower fluid intake leads to low urine volume and increased concentration of stone-forming salts. Studies have shown that high fluid intakes can provide an effective and economical preventive strategy and inhibit the recurrence of renal calculi or stones (17-20). The Nurses Health Study (37), a prospective cohort study of women aged 40-65 years and the Health Professionals Follow-up study (38) undertaken in men aged 45-75 yrs in the US, also found that an increased total fluid intake reduces risk of stones but also showed that the reduction varied according to the choice of fluids as well as the amount.

Urinary tract, colon and breast cancer

Several studies have shown a direct correlation between the quantity of fluid consumed and the incidence of certain cancers. A study in Israel (23) found that patients with urinary tract cancer (bladder, prostate, kidney, testicle) consumed significantly less fluid than healthy controls but no association was found with a specific beverage. Another study in Hawaii (24) showed that total fluid intake and intake of tap water in particular had a strong inverse dose-response relationship to lower urinary tract cancer (bladder, renal pelvis, ureter) in women. The association was stronger in smokers. Similar findings have been observed for breast and colon cancer. One US study of colon cancer (22) showed an inverse dose-related relationship between water intake, measured as glasses per day, and risk of colon cancer in women. Women drinking more than 5 glasses a day had a 45% reduced risk versus those with 2 or fewer glasses a day. Among men there was an apparent 32% decrease in risk with increasing water consumption although it was not statistically significant.

Results from the prospective Health Professionals Follow-up study of men aged 45-75yrs (25) also showed that total daily fluid intake was inversely associated with the risk of bladder cancer, the study showed that the consumption of water contributed to the lower risk of bladder cancer when compared to other fluids.
One pilot, hospital based case-control study of breast cancer (21) in the US claimed to show a strong inverse relationship with water consumption. This finding was reported in a letter to the editor when commenting on a similar finding in a colon cancer study. Amounts consumed were not reported. Overall risk reduction was 79% when data was adjusted for age, height, exercise, family history, use of hormone replacement, endogenous oestrogen exposure, use of oral contraceptives or birth control pills, and tea, coffee and alcohol consumption. The authors concluded that subclinical or chronic dehydration may compromise intracellular water, alter cellular concentrations affect enzyme activity and inhibit cellular carcinogen removal.

Other public health issues

It is often reported that drinking fluids has a satiating effect that makes people feel more full and eat less, and may help in controlling overweight and obesity. Two studies from the US, one in children (26) and one in adults (39), give support to this assertion. Levine (27) reviewing the results of liquid intake in childhood obesity and disease concluded, amongst other things, that replacing drinks in the diet with milk and water would help control and greatly improve the overall health of the children and adolescents in the US.

Oral health may also be affected by fluid consumption. Apart from the beneficial affects of fluoride added to tap water in many communities in Australia, fluid intake can affect saliva production. Saliva which is primarily water, is essential for maintenance of oral health, decreased body water has been associated with salivary dysfunction, especially in older adults. However, one investigation (29) found that decreased salivary gland function was associated with dehydration independent of age.

Numerous studies have shown diminished thirst sensations in the elderly. Despite the fact that these changes may be normal adaptations of the ageing process, the outcomes of dehydration in the elderly are serious and range from constipation to cognitive impairment and functional decline. This issue has therefore been specifically addressed in the Dietary Guidelines for Older Australians.

Mental and Physical performance

The effect of dehydration on cognition has not been well studied but it is likely that as physical impairment is caused by hypohydration, mental performance will be impaired (40,41). One study of mental performance under different levels of stress-induced dehydration in acclimatised subjects (30) showed that after recovery from exercise in the heat subjects demonstrated significant and progressive reductions in arithmetic performance, short-term memory and visual-motor tracking at a deficit of 2% body fluid compared to the hydrated state.

The effects of hydration on physical performance are well studied and well known. (10-14) When exercise is performed in excessive heat or cold, low humidity, or high altitude, fluid losses increase. Dehydration as little as 1% decrease in body weight impairs physiologic and performance measures during continuous exercise and even minor body mass loss from dehydration negatively affects heart rate, tolerance times and stroke volume in light and heavy exercise in the heat.
Current Intakes in Australia

Table 1 below shows the percentile distribution and mean daily intake of non-alcoholic beverages from the 1995/6 National Nutrition Survey (42). Intakes of water (including tap water, bottled water and plain mineral waters) decreased both in absolute terms (g/day) and as a percentage of non-alcoholic beverages with age.

It is recommended that adults consume some 6-8 glasses of fluid per day (approximately 1500 mLs – 2000 mLs). This data would indicate that the population mean intake is within this range (excluding alcoholic drinks) but 30-40% of the population did not reach this target on the day of the survey. Water constituted 42-44% of the non-alcoholic fluids consumed on the day of the survey.

Table 1  Percentile distribution and mean intakes of non-alcoholic fluids in adults 19 yrs and over from the 1995/6 National Nutrition Survey

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* tap, bottled or plain mineral water
** includes soft drinks, flavoured mineral water and electrolyte drinks

SPECIAL GROUPS

Older adults

Kidney function can decline as part of the normal ageing process with decrease in kidney mass; declines in renal blood flow and glomerular filtration rate, distal renal tubular diluting capacity, renal concentrating capacity, sodium conservation and renal response to vasopressin. This together with hormonal changes and factors such as decreased thirst perception, medication, cognitive changes, limited mobility and increased use of diuretics and laxatives, make older adults a group of particular concern (43).
Pregnant and lactating women

A pregnant woman has slightly increased water requirements because of expanding extracellular fluid space, the needs of the fetus and the amniotic fluid. Compared with the nongravid state, the National Research Council in the US calculated that pregnant women require an extra 30 mL fluid per day (2). This does not take into account any increase in fluid loss from increased heat production and perspiration especially in the summer months. It is thus likely that this is a minimal additional requirement. A lactating woman must replace fluid lost in breast milk. 87% of milk is water and the average milk production in the first six months of lactation is 750 mL/day (2,44). The increased fluid need is therefore 750-10000 mL/day above basic needs.

PRACTICAL ASPECTS - What to Drink?

Water

Water is the preferred drink to ensure adequate hydration, for a number of reasons. It provides water without additional dietary energy. With the high levels of overweight and obesity in the Australian population this is an important consideration. Fluoridated tap water provides an additional benefit for development of strong teeth and bones. As can be seen from above, for a number of the health outcomes, water appears to have specific advantages over many other fluids especially those which have a diuretic action such as alcoholic drinks or caffeinated drinks such as coffee, tea, cola and certain “energy” drinks (with guarana). Alcoholic drinks in excess can also have other health consequences (see Alcohol guideline).

Tea and coffee

Tea, especially green tea, but also black tea and to a lesser extent coffee, contain substantial amounts of polyphenols, antioxidants with purported benefits in relation to cancer and cardiovascular protection. Green tea is the most potent source and has been used for many years in Asian cultures. The literature on the benefits of consumption in relation to specific health outcome in human subjects is still sparse and it has been speculated that the effect of tea consumption may depend on the causative factors of the particular condition of concern (45). Nevertheless because of its widespread use, in Australia, black tea is a major contributor to polyphenols in the Australian diet (46) rivaling the contribution of what is traditionally considered as the major source - fruits and vegetables. In contrast, both standard coffee and, to a lesser extent tea, contain substantial amounts of caffeine which can have unwanted stimulant effects in susceptible people and do act as diuretics. Tea and coffee are often consumed with added milk and sugar and the additional energy intake from these sources can be substantial in some people.

Milk or alternatives

The value of milk as a component of the diet is outlined in the “Reduced Dairy foods and/or alternatives” guideline. Milk is a key supplier of calcium, protein and a number of other valuable nutrients in the diet and should be included as part of the fluid intake. Use of reduced fat or skim milks will reduce additional fat and energy intake whist retaining
the calcium contribution. Soy milks are available for those who do not want to or cannot use cow’s milk, but calcium fortified varieties should be selected. Again, it is necessary to be aware that use of soy milks, as for dairy milks, will add energy to the diet.

**Fruit and vegetable juices**

Fruit and vegetable juices can be a useful source of vitamin C, potassium and folate. They may add variety to the diet and fluid intake. There is, however, no reason for an obligatory consumption of fruit and fruit juice if fruits and vegetables are consumed in line with the “Vegetables (including legumes) and Fruits” dietary guideline.

**Alcohol**

The issue of alcoholic drinks is covered in more detail in the Alcohol guideline. In relation to the issue of hydration, it is important to note that alcohol is a strong diuretic that can increase fluid loss from the body. It is also very high in dietary energy.

**Other high energy density drinks**

Other fluids with high energy density such as soft drinks and cordials of various sorts with added sugars should be limited. This area is discussed in more detail in the “Consume only moderate amounts of sugars” dietary guideline. Drinks of this kind add substantial dietary energy to the diet without additional nutrient value. Occasional use of low-joule soft drinks can add variety and palatability.

**RELATIONSHIP TO OTHER GUIDELINES**

*Enjoy a wide variety of nutritious foods*

Water is contained within foods and a varied diet will contribute to water requirements. Some variety is also recommended in fluid consumption but water is the preferred drink to maintain adequate hydration.

*Prevent weight gain by being physically active and eating according to your needs*

Consumption of fluids other than water should be governed by a consideration of their nutritional value in relation to the additional energy they supply in a population with high levels of overweight and obesity.

*Consume only moderate amounts of sugars and foods containing added sugars*

Soft drinks and cordials are a major source of added sugars in the Australian diet and consumption of drinks with added sugars should be moderated.

**EVIDENCE**

Level II (refs 17,19) and Level III (refs 18,20,37,38) evidence exists to link water consumption and urinary volume to occurrence of kidney stones. Level III evidence exists for a link between fluid consumption and cancer of the urinary tract (ref 35); between total fluid intake, and tap water in particular, for lower urinary tract cancer in women (ref 24); for water intake and colon cancer (ref 22); for fluid intake, and water in particular, for bladder cancer (ref 25); for water consumption and breast cancer (ref 21) and for dehydration and salivary gland function (ref 29) and for dehydration and mental performance (ref 30).
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LIMIT SATURATED FAT AND MODERATE TOTAL FAT INTAKE

DEFINITIONS

Fats
Chemically, most of the fats in foods are triglycerides, made up of a unit of glycerol (glycerine) combined with three fatty acids (which may be the same or different). The differences between one fat and another are largely due to the fatty acids they contain (which together make up 90% of the weight of the molecule).

Fats in the diet can be classified as ‘visible’ or ‘invisible’. Visible fats include butter, margarine, cooking oils and the fat on meat. Invisible fats occur in foods such as cheese, biscuits, cakes, pastries and nuts. In most diets about half the fats are visible and half invisible.

Fats are the most concentrated form of energy (38 kilojoules or 9 calories per gram). They are the chemical form in which most of the energy reserve of animals and some seeds is stored.

Cholesterol, a lipid has important functions in the body. It is part of the cell membrane of all cells, part of the myelin in the brain and nervous system and the starting material for synthesis in the body of bile acids, adrenocortical and sex hormones.

Saturated Fats
In these the majority of the fatty acids, in chemical terms, contain no double bond, ie are fully saturated with hydrogen. Saturated fats are usually solid at room temperature. They are the main type of fat in milk, cream, butter and cheese; in some meats (most of the land animal fats) and in palm oil and coconut oil. Most predominantly saturated fats contain one or more of the fatty acids palmitic (16:0), myristic (14:0) and lauric (12:0). When they predominate in the dietary fat they tend to raise plasma cholesterol.

Monounsaturated Fats
In these the main fatty acid is oleic acid (18:1), which has one (mono) double (unsaturated) bond. Olive oil, canola and peanut oils are rich in oleic, and it is the most abundant fatty acid in most meats.

Polyunsaturated Fats
In these the main fatty acid contains two or more double bonds – ‘poly’ (= many) unsaturated. They are liquid at room temperature, ie oils. The most common polyunsaturated fatty acid is linoleic (18:2); its double bonds are in the omega (ω)–6 position. It occurs in seed oils, eg sunflower, safflower and corn oils.

Smaller amounts of polyunsaturated fatty acids with double bonds in the omega-3 position also occur in the diet. Best known are those in fatty fish, their names abbreviated to EPA (20:5) and DHA (22:6). Another omega-3 polyunsaturated fatty acid, ALNA (18:3) occurs in small amounts in leafy vegetables. There is more of this in canola oil and most in flaxseed oil.
Dietary Cholesterol
Cholesterol, chemically a sterol, occurs in all the cell membranes of land animals. Brains and egg yolks are very rich in cholesterol. Oils and fats from plants never contain cholesterol.

Eating cholesterol doesn’t necessarily increase cholesterol in human blood plasma because when it is absorbed the liver tends to reduce its own endogenous cholesterol synthesis (about half the body’s cholesterol is made in the body from acetate).

Plant Sterols
Also called phytosterols, these are chemically very similar to cholesterol but with a small difference in their chemical structure (in the side chain). They occur in oils from plants (though they may be taken out by refining) and when eaten interfere with absorption of cholesterol from the intestine. To make use of this effect some margarines have been introduced recently which contain extra plant sterols.

Trans Fatty Acid
A form of unsaturated fatty acid that is straight at a double bond (rather than bent as in the usual cis form); not common in nature but formed during some manufacturing process, eg hydrogenation of edible oils to make hard margarines.

Fat Replacers (1)
There are two groups. Fat substitutes resemble fats and can replace fats in food. They contain fatty acids but they have been joined to the centre of the molecule with chemical links that can’t be digested by human enzymes. They provide no dietary energy when eaten, eg sucrose polyester.

Fat mimetics
Are food ingredients based on starch, cellulose or protein that have been physically modified to have the mouth feel of fats but do not have their other functions. They have some energy value but it is small.

BACKGROUND
Historically the first Dietary Guidelines for Australians (1982) (2) recommended “Avoid eating too much fat”, ie total fat. It did not consider type of fat, as in the 1977 Dietary Goals for the United States (3) which recommended 10% total energy from saturated fats, 10% from monounsaturated fats and 10% from polyunsaturated fats.

In the second edition of Dietary Guidelines for Australians (1992) (4) the guideline had evolved to “Eat a diet low in fat and, in particular, low in saturated fat”. The most recent Dietary Guidelines for Older Australians (1999) (5) moved further and recommends “Eat a diet low in saturated fat”. The text behind this last guideline explains that low fat diets do not provide health benefits at the two ends of life.
An estimate of the present fat intake of Australia is provided by the 1995 National Nutrition Survey (6). Mean total fat was about one-third of total energy, with saturated fat (SATFA) around 12.5%, polyunsaturated (PUFA) fat around 4.5% and monounsaturated fat (MUFA) around 11.5%. (Table 1).

Table 1   Mean Fat intakes from National Nutrition Survey Australia 1995 (6)

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>Males</th>
<th>2-3</th>
<th>4-7</th>
<th>8-11</th>
<th>12-15</th>
<th>16-18</th>
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<th>45-64</th>
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<tbody>
<tr>
<td>SATFA</td>
<td></td>
<td>15.3</td>
<td>14.5</td>
<td>13.9</td>
<td>14.2</td>
<td>13.7</td>
<td>13.2</td>
<td>12.9</td>
<td>12.9</td>
<td>12.0</td>
<td>12.6</td>
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<tr>
<td>MUFA</td>
<td></td>
<td>10.7</td>
<td>11.3</td>
<td>11.4</td>
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<td>12.1</td>
<td>11.9</td>
<td>11.7</td>
<td>11.7</td>
<td>11.3</td>
<td>11.7</td>
</tr>
<tr>
<td>PUFA</td>
<td></td>
<td>3.8</td>
<td>4.1</td>
<td>4.3</td>
<td>4.2</td>
<td>4.4</td>
<td>4.3</td>
<td>4.5</td>
<td>4.6</td>
<td>4.6</td>
<td>4.5</td>
</tr>
<tr>
<td>TOTAL</td>
<td></td>
<td>33.2</td>
<td>32.9</td>
<td>33.2</td>
<td>33.6</td>
<td>33.6</td>
<td>33.2</td>
<td>33.0</td>
<td>32.3</td>
<td>31.3</td>
<td>32.6</td>
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</table>

<table>
<thead>
<tr>
<th>Females</th>
<th></th>
<th>16.1</th>
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<th>14.7</th>
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<th>11.9</th>
<th>12.3</th>
<th>12.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>SATFA</td>
<td></td>
<td>10.9</td>
<td>11.1</td>
<td>11.8</td>
<td>11.5</td>
<td>10.9</td>
<td>11.4</td>
<td>11.6</td>
<td>11.3</td>
<td>11.2</td>
<td>11.4</td>
</tr>
<tr>
<td>MUFA</td>
<td></td>
<td>3.7</td>
<td>4.0</td>
<td>4.3</td>
<td>4.3</td>
<td>4.0</td>
<td>4.6</td>
<td>4.6</td>
<td>4.7</td>
<td>4.7</td>
<td>4.6</td>
</tr>
<tr>
<td>TOTAL</td>
<td></td>
<td>34.1</td>
<td>32.4</td>
<td>34.2</td>
<td>33.2</td>
<td>31.9</td>
<td>32.8</td>
<td>33.0</td>
<td>32.1</td>
<td>32.1</td>
<td>32.6</td>
</tr>
</tbody>
</table>

SATFA, MUFA and PUFA are groups of fatty acids. Combined they are approximately 90% of total fats (the rest of total fat is glycerol).

These numbers are very similar to the fat intakes recorded earlier in the 1983 Australian National Dietary Survey (7). Total fat (mean) then was 36.6% in men and 37% in women.

Median grams per day (all ages over 19) were: in men 105g in 1983 and 98.5 in 1995; in women 70 g in 1983 and 67.6 in 1995. There appears to have been a small reduction in total fat between 1983 and 1995. The median ratios between PUFA, MUFA and SATFA were almost identical in the two surveys. In men 1983: 0.36/0.93/1.0 and 1995: 0.36/0.93/1.0; in women 1983 0.36/0.92/1.0 and 1995: 0.37/0.91/1.0. In the 1995 survey the $10^{th}$ to $90^{th}$ percentile distribution of saturated fat ranged from 23g to 58g in all men over 18 years and from 16g to 40g/day in all women over 18 years. The intake of polyunsaturated fat ranged from 19 to 21g/day in men and from 7 to 15g/day in women.

The main food groups providing saturated fat in the 1995 survey were (in descending order, all ages over 18 y): Dairy 27.2% (cheese 8.3%); cereal-based products and dishes 20.2%; meat group 18.7%; fats and oils 8.9%; potatoes (esp. chips) 5.7%; chocolate 3.2%; fish 1.8%. The main food groups providing polyunsaturated fats in the 1995 survey were: fats and oils 19.2% (margarine 16.4); cereal-based products and dishes 15.4%; meat group 14.2%; breads + breakfast cereals 13.8%; vegetable products and dishes 11.9%; nuts and seeds 4.9%; fish 4.6%.
SCIENTIFIC BASIS

Dietary Fat and Overweight/Obesity

Overweight and obesity have been increasing rapidly in Australia, with acceleration in the last 2 decades (8, 9, 10, 6). (The chapter titled Prevent Weight Gain by Being Physically Active and Eating According to Your Needs provides further discussion).

Many Australians have become obese in the last 20 years while fat intakes do not appear to have increased (either as g/d or as % energy) and may have declined slightly. This dramatic increase of obesity and overweight has occurred in most other countries (11) and is the outstanding challenge for nutrition research today.

Fat is the macronutrient with the highest energy value per unit weight, 2.25 times that of carbohydrates and proteins. Fats and oils are largely invisible in the diet, in baked goods, sauces, confectionery, cheese, snack foods, nuts and as coating on fried foods. People like the taste and mouth feel and enjoy eating foods containing it, so there is passive overconsumption of fat.

<table>
<thead>
<tr>
<th>Table 2 Overweight and Obesity in Australian Surveys</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MEN</strong></td>
</tr>
<tr>
<td>NHF 1980 (8)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>NHF 1983 (9)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>NHF 1989 (10)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>NNS 1995 (6)</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

(Lower numbers in brackets are overweight + obesity)
NHF = National Heart Foundation (surveys in capital cities)
NNS = National Nutrition Survey (randomly sampled the whole nation from voters rolls).

There is now evidence that Rubner’s isodynamic law (kilojoules from carbohydrate ≡ kilojoules from fat ≡ kilojoules from protein) does not quite hold in practice. Kilojoules (kJ) from different macronutrients are not really equal. Fats eaten above the day’s energy requirement are nearly all stored in the body (12). This contrasts with the fate of extra carbohydrate or protein, which stimulate their own oxidation and are only partly stored short-term, and alcohol which is completely metabolised. In addition, Blundell has shown in the appetite laboratory that foods high in fat have a high palatability, together with weak satiation, compared (kJ for kJ) with carbohydrate and protein (13). As well as this, fatty meals induce lower thermic response than meals high in carbohydrate and/or protein (14).

These effects of fat suggest that either obese people will be found to eat more fat than lean people or that obese people are in some way more susceptible to the effects of fat.
There is some epidemiological evidence that fat consumption is associated with overweight (15). In the MONICA surveys median body mass index (BMI) of communities did not increase with FAO figures for percent energy available from fat, but in individuals 9 out of 12 cross sectional studies have shown higher fat intakes in those with high BMI (15). Prospective studies have given inconsistent results (15). This could mean that fat is leading to obesity only in some communities. One problem with food intake measurements is whether they were made at the time of gaining weight. Another is that obese people under-report their food intake, especially the fats (16) and no biomarker is available to indicate total fat intake objectively. Like Jack Spratt and his wife, some people do not like fatty food and others do. They have different satiation responses (17).

In a careful controlled trial in Denmark (18), obese subjects first lost 12.6 kg (on low energy diets), then for the weight maintenance phase they were randomised to an ad lib, low-fat, high-carbohydrate diet or a prescribed fixed energy diet (≥7.8 MJ/d) for 1 year. Regain of weight was 0.3 kg on the low fat diet and 4.1 kg on fixed energy. A year after the end of the trial, at follow up the low-fat group had now regained 5.4 kg and the fixed energy diet group had regained 11.3 kg (almost all the weight originally lost). In a 6-month trial in the Netherlands (19) 200 people around 40 years of age, a little overweight but not obese, were asked to eat standard or low fat unrestricted eating, or restrained usual fat or low fat. Body weights rose with usual fat unrestrained; they stayed the same with low fat unrestrained and fell with low fat and restrained eating. This nicely demonstrates the separate effects of low fat and restrained eating. Of a registry of 784 previously obese people (20) who had successfully lost 30 lb (13.6 kg) and maintained their weight for at least a year, 33% had limited the percentage of daily energy from fat and 25% had counted fat grams.

The WHO (11), the British Obesity Task Force (21), the Scottish Intercollegiate Guidelines Network (22) and review by George Bray, who carried out a thorough review (23), are all emphasising the major role of fat consumption in the development of obesity and of cutting fat intake in its dietary management.

None of this should be taken to mean that reducing fat intake is all that is needed to deal with the contemporary epidemic of overweight and obesity. Physical activity is as important as diet (24) as discussed in the chapter titled Prevent Weight Gain by Being Physically Active and Eating According to Your Needs.

**Saturated and Trans-Fats and Coronary Heart Disease (CHD)**

Of the risk factors for CHD that can be influenced by diet, plasma LDL cholesterol, which is reflected in plasma total cholesterol concentration, remains the best established. It has been found to be a significant risk factor in at least 50 prospective cohort studies involving more than 600,000 subjects in 18 countries. The importance of plasma cholesterol has been confirmed by trials with ‘statin’ cholesterol-lowering drugs, which are showing reductions in coronary events – and in all-causes mortality – even among people starting with average US plasma cholesterol levels (25).

Saturated fat is the strongest dietary determinant of plasma LDL-concentration. This has been demonstrated repeatedly in controlled human experiments (26-32).
Mensink and Katan (30) included 27 trials in their meta-analysis, Hegsted et al (31) reviewed 248 metabolic experiments and 96 field observations. Clark et al (32) analysed only metabolic ward experiments, 395 with solid foods and 32 with liquid formula diets. 5,076 subjects participated in the 71 publications that they analysed.

As far as it has been possible to separate the effects of individual fatty acids, it is only lauric (12:0), myristic (14:0) and palmitic (16:0) that have the LDL and total cholesterol-raising effect. Stearic (18:0) does not appear to have this effect (28, 32, 33) and lauric appears less active than palmitic and myristic (33).

The cholesterol-raising effect of trans unsaturated fatty acids (34) was rediscovered in the early 1990s (35) and confirmed in different laboratories (36, 37). Unlike C12 to 16 saturated fatty acids, trans fatty acids are reported to lower HDL-cholesterol and to increase Lp(a).

In relations between diet and CHD prospective cohort studies are considered the most reliable observational epidemiology. The first to report a positive association of saturated fat intake with subsequent CHD mortality was the classic Seven Countries Study (38). Association of saturated fats with CHD events was supported by results from the Nurses Health Study (39), the Health Professionals follow up study (40), both in the USA, and in a cohort in Finland where saturated fat intake was ascertained from plasma phospholipid palmitate (41).

Trans-fatty acids were reported to be associated with CHD in the Nurses Health Study (42), although the reliability of the food composition data was queried (43). Re-analysis of dietary data and longer follow up of the Nurses Cohort by Willett’s group supported their earlier finding on trans fatty acids (44). However, adipose tissue percentages of trans fatty acids have not been associated with CHD cases in Europe (45, 46).

The UK Committee on the Medical Aspects of Food Policy recommended as early as 1984 that trans-fatty acids should be regarded as equivalent to saturated fatty acids for the purposes of preventing CHD (47). The NHMRC Working Party on the Role of Polyunsaturated Fats in the Australian Diet (48) came to the same conclusion; so did the Heart Foundation’s 1994 review of dietary fats and blood cholesterol (49).

The Heart Foundation’s 1999 position statement on dietary fats (50), based on a thorough review of the literature with ranking of the strength of evidence recommended that saturated fatty acids and trans fatty acids together contribute no more than 8% of total energy intake. Trans fatty intakes in Australia have not been accurately quantified but now that they have been virtually eliminated from our soft margarines average intake is believed to be probably 1%-2% of total energy (51). The 2000 Dietary Guidelines for Americans recommends a saturated fat intake of 10 percent of kilojoules (52). This was recommended by the NHMRC in 1992 (48) and is a feasible target for the Australian average to reach from the present level of 12.5 percent of energy. Major remaining sources of trans fatty acids should be identified and reduced.
Unsaturated Fats and Coronary Heart Disease (CHD)

The last edition of Dietary Guidelines for Australians (2) quoting the NHMRC Working Party on Polyunsaturated Fats (48) recommended that reduced total fat and saturated fat be replaced with combinations of:

- “complex carbohydrates”
- omega-6 polyunsaturated fats (PUF)
- monounsaturated fats
- oily fish.

The case for omega-6 (\(\omega-6\)) polyunsaturated fatty acids (n-6 PUFA)

In human nutrition, linoleic acid (18:2) is almost synonymous with “\(\omega-6\) PUFA”.

(a) Oils rich in \(\omega-6\) PUFA (ie linoleic acid) consistently lower plasma total and LDL-cholesterol (26 - 32), even when the oil increases the fat intake (53, 54).

(b) In experimental animals, rats and marmosets, long-term feeding of diets rich in \(\omega-6\) PUFA reduces the frequency of dangerous arrhythmias when a coronary artery is ligated (55, 56, 57).

(c) Three prospective cohort studies estimated 18:2 intake from plasma concentrations (on CE or PL). In all three there was a negative association with subsequent coronary heart disease (41, 59, 60).

Nine prospective cohort study publications estimated intake of fatty acid types and followed subjects to see who developed CHD. In 3 of these, significant inverse association of PUFA or 18:2 with CHD was found in the US Nurses Study (39, 44) and the Western Electric Study (Chicago) (58). In the other 6 studies no significant association was found. In some of these the dietary method may not have had enough detail of fat composition (eg 24 hour recall) or the range of PUFA intakes was too small.

(d) Eight intervention trials (61-68) prescribed substitution of polyunsaturated fats (ie omega-6) for saturated fat in the experimental group (Table 3). Diet was the only lifestyle factor changed. All the subjects were randomised except in the two Helsinki mental hospital trials (one in men, the other in women) which exposed all the residents to experimental and control diets in turn.
### Table 3  Dietary intervention trials for prevention of CHD with increased polyunsaturated and decreased saturated fat

<table>
<thead>
<tr>
<th>Type of Trial</th>
<th>n</th>
<th>Reduction of TCX years</th>
<th>CHD Intervention</th>
<th>Total death INTERVENTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rose et al (61)</td>
<td>SD</td>
<td>52</td>
<td>-9%x2y</td>
<td>12/6</td>
</tr>
<tr>
<td>MRC (62) Soybean oil</td>
<td>SD</td>
<td>393</td>
<td>-15%x3.4y</td>
<td>45/51</td>
</tr>
<tr>
<td>Dayton (63), VA</td>
<td>PD</td>
<td>846</td>
<td>-13%x7y</td>
<td>60/78</td>
</tr>
<tr>
<td>Leren (64), Oslo</td>
<td>SD</td>
<td>412</td>
<td>-14%x5y</td>
<td>79/94</td>
</tr>
<tr>
<td>Helsinki men (65) *</td>
<td>PD</td>
<td>1900</td>
<td>-15%x12y</td>
<td>34/76</td>
</tr>
<tr>
<td>Helsinki women (66)*</td>
<td>PD</td>
<td>2836</td>
<td>-13%x12y</td>
<td>73/129</td>
</tr>
<tr>
<td>DART (67) fat advice</td>
<td>SD</td>
<td>2033</td>
<td>-3.5x2y</td>
<td>132/144</td>
</tr>
<tr>
<td>Frantz (68) men</td>
<td>PD</td>
<td>4393</td>
<td></td>
<td>69/74</td>
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<td>4664</td>
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<td>62/47</td>
</tr>
<tr>
<td></td>
<td></td>
<td>17,529</td>
<td></td>
<td>566/699 (81%)</td>
</tr>
</tbody>
</table>

PD = Primary prevention trial, diet the only intervention  
SD = Secondary prevention trial, diet the only intervention.  
*Institutions were alternated. In all the other trials, subjects were randomised.

(In other trials not cited here there were other dietary changes: reduced total fat, increased cereals, vegetables and fruits or increased omega-3 PUFA, or the dietary prescription was not clearly described). These eight trials involved a total of 17,529 subjects in four countries, half in the increased P/S group and half in the usual diet group. There were 566 new coronary events in the combined increased P/S experimental diet groups which was only 81% of the 699 CHD events in the control group (P<0.05 by Wilcoxon test). All causes mortality was lower in the experimental group, 1,291 against 1,360 (95%), but not significantly so (69).

(e) The decline of CHD deaths in Australia and the USA between the mid 1960s and 1980 (compared with static rates in the UK and Sweden in that period) was more from reduction of sudden deaths outside hospital than from better survival of admitted cases of myocardial infarction (70). The most likely dietary change was increased omega-6 PUFA (71-76). Around this time in history people at risk, ie middle aged men, some who already had diagnosed CHD, increased PUFA by a larger percentage than they reduced saturated fat (because PUFA intake had been much smaller). In 1961 corn oil and sunflower seed oil were being recommended by Professor H.M. Whyte (77), at that time Australia’s leading researcher on dietary fats and CHD, and by 1967 soft margarines were available and recommended by the National Heart Foundation to help increase the dietary P/S ratio to 1.25 (78).

For Geoffrey Rose (75)

*In the US and Australia the changing balance of unsaturated to saturated fatty acids correlates better with the fall in CHD than do changes in total or saturated fat intake. In the UK the average dietary P/S ratio rose from 0.24 in 1980 to 0.35 in 1985 and the timing of this large change was reasonably close to the changes in CHD mortality. In the USSR, the severe and alarming increase in CHD mortality seems to have coincided with a big fall in the dietary P/S ratio ..... In the light of all the evidence that has*
accrued .... I now think that the P/S ratio is probably one of the most potent determinants of CHD rates and trends and that its practical importance should be emphasised accordingly” (75).

It was at first difficult to see a mechanism for prompt falls in sudden cardiac death with increased national uptakes of polyunsaturated oils and margarines. Changes in plasma cholesterol were either not well documented (as in Australia) or relatively small reductions (in the USA (79)). There should be a time lag between lower plasma cholesterol, slower advance of atherosclerosis and less clinical CHD. Hetzel et al (1989) (74) introduced the then new discovery of preventing cardiac arrhythmias as a plausible explanation for a prompt reduction of sudden death with increased PUF intakes. Note that there was no increase of fish, canola or olive oil intake in Australia while CHD mortality came down from its peak in 1966.

The FAO/WHO Consultation of Fats and Oils in Human Nutrition recommends that “desirable intakes of linoleic acid (ie ω-6 PUF) should provide between 4 and 10 percent of energy. Intakes in the upper end of this range are recommended when intakes of saturated fatty acids and cholesterol are relatively high”(80). The National Heart Foundation of Australia (48) concludes that there is good evidence that replacing saturated fatty acids with ω-6 polyunsaturated fatty acids reduces the risk of coronary events and deaths. It is recommended that ω-6 polyunsaturated fatty acids contribute 8 to 10% of total energy intake”(50).

The case for omega-3 (ω-3) polyunsaturated fatty acids (n-3 PUFA)

Unlike omega-6 PUF with only one fatty acid usually considered, there are three important fatty acids in the omega-3 series:

- α-linolenic (ALNA) (18:3) occurs in leafy plants, canola, flaxseed oils
- eicosapentaenoic (EPA) (20:5) richest source is oily fish
- docosahexaenoic (DHA) (22:6) richest source is oily fish

All three occur in low concentrations in human and other animal tissues and in human milk. EPA is the precursor of the 3 series of prostaglandins and the 5 series of leukotrienes. DHA is found in very high concentration in the photoreceptors of the retina and the membranes of the brain. Its role in infant nutrition is considered in the Dietary Guidelines for Children and Adolescents.

Some of the ALNA is chain-elongated and desaturated to EPA and DHA in the liver and brain and this is the only dietary source of these very long chain ω-3 PUFAs in vegans.

(a) α-linolenic (ALNA), as the predominant fatty acid in flaxseed oil lowers plasma total and LDL-cholesterol and large intakes of fish oil with EPA and DHA its major polyunsaturated fatty acids usually lower plasma total and LDL-cholesterol (29). However, in the diet the three ω-6 PUFAs are present in much smaller amount than linoleic acid and their cholesterol-lowering effect is of purely academic interest (29). Fish oil has a greater lowering effect on plasma triglycerides than ω-6 rich oil (81).
(b) In experimental animals fish oil has a more potent effect in preventing ischaemic arrhythmias than plant oils rich in ω-6 PUF (82, 83), and canola oil also has an antiarrhythmic action (84), presumably because of its α-linolenic acid content because monounsaturated olive oil does not have this effect.

(c) Fish consumption has been related to outcome in 11 prospective cohort studies systematically reviewed by Marckmann and Grønbaek (85). Fish were found to be protective in 5 of the 11 studies. Some of the studies with negative results were of high scientific quality. It appears that a protective effect of fish – and presumably of fish oil, ie EPA and DHA, is only seen in populations at high risk of CHD. In two prospective studies intake of ALNA appeared to be protective (40, 86).

(d) Two randomised controlled trials have been reported with fish or fish oil. In the Diet and Reinfarction Trial (DART) (67) one of three treatments given for 2 years to people who had survived a myocardial infarction was to eat 200 to 400 g of fatty fish a week or take fish oil capsules. Compared with controls, coronary mortality was significantly reduced by 24% in the fish group. Plasma cholesterols were not different in this group and there were more non fatal infarcts. The GISSI-Prevenzione Investigators (87) gave 1g EPA+DHA or vitamin E or both or neither to 11,324 survivors of myocardial infarction for 3.5 years in a multi-centre trial in Italy. The fish oil groups had 10-15% fewer deaths, non-fatal re-infarcts and strokes. The greatest benefit of the fish oil was a 45% reduction of sudden deaths. The authors saw no evidence of reduction of thrombotic events.

The third trial relevant to omega-3 PUF is the Lyon Diet Heart Study (88) in which, among other dietary differences, α-linolenic acid (ALNA) was increased in the experimental group. Though α-linolenic appears in the title of the paper and its plasma level increased, it is difficult (89) to be sure whether this or other dietary (or other) differences can explain the much lower mortality in the experimental group which showed no reduction of plasma cholesterol.

The FAO/WHO Consultation on Fats and Oils (80) recommended that the ratio of linoleic to α-linolenic acid in the diet should be between 5:1 and 10:1. Individuals with a ratio in excess of 10:1 should be encouraged to consume more ω-3 rich foods such as green leafy vegetables, legumes, fish and other seafood.

An expert workshop in the Netherlands (90) reviewed the health effects of ω-3 PUFA and concluded that consumption of fish may reduce the risk of CHD. People at risk for CHD were therefore advised to eat (preferably fatty) fish once a week. The workshop concluded that there should be separate recommendations for plant (18:3) and marine (20:5, 22:6) ω-3 PUFAs and that the ω-3/ω-6 ratio will not be helpful.

The National Heart Foundation of Australia (50) recommends:-
• At least two fish meals per week (preferably oily fish).
• Consume both plant and marine ω-3 PUFAs since it is possible that they protect against CHD by different mechanisms.
• Plant ω-3 PUFA intakes at least 2 g/day.
The case for Monounsaturated fats (MUFA)

Oleic acid (18:1,cis) predominates among the mono-unsaturated fatty acids. There are however small amounts in the diet of 16:1, 17:1, 20:1, 22:1.

a) When dietary fats are changed from mostly saturated to mostly monounsaturated, total and LDL-cholesterol fall. But polyunsaturated fatty acids are more cholesterol-lowering (26, 28-33). Some oils rich in oleic acid have more effect on plasma cholesterol than others. This seems to depend on their content of phytosterols (cholesterol-lowering) and squalene (cholesterol-raising) (91).

b) Olive oil feeding did not prevent dangerous cardiac arrhythmias in experimental animals subjected to coronary artery ligation (56, 84) and with neonatal rat cardiac myocytes in culture oleic acid (unlike PUFAs) has no antiarrhythmic effect (92).

c) In prospective cohort studies monounsaturated fatty acid intake has not usually been associated with significant increase or decrease in coronary events. In the Seven Country Study “monoenes” were negatively correlated but not significantly so (93). In the younger cohort in Framingham MUFA intake was significantly positively associated with CHD incidence (94). In the US Nurses Study a negative association of MUFA with CHD was marginally significant after several adjustments (44).

d) There has been no preventive trial with monounsaturated fats.

The FAO/WHO Consultation made no specific recommendations about monounsaturated fat intake (80).

The National Heart Foundation of Australia (50) notes there is little evidence that mono-unsaturated fatty acids have an independent effect on coronary end points. The position statement recommends that “a proportion of dietary saturated fatty acids should be replaced by monounsaturated fatty acids as a strategy for reducing the intake of saturated fatty acids”.

Dietary Cholesterol

The cholesterol-elevating effect of dietary cholesterol is less consistent than that of saturated fats (29). Dietary cholesterol only occurs in animal fats, which are also the major source of saturated fatty acids in the diet (6). Of the two most concentrated sources of cholesterol in the diet, animal brains are hazardous because of the risk of bovine spongiform encephalopathy but eggs are rich in several nutrients. The position of the NHMRC on dietary cholesterol up to now has been that at the public health level, advice to reduce saturated fat will bring with it smaller cholesterol intakes. This policy is supported by a report from the Harvard prospective cohorts which find that consumption of one egg per day was not associated with any increase in CHD rate (95).

The National Heart Foundation (50) recommends that individuals with plasma cholesterol greater than 5 mmol/L or with other risk factors should restrict their intake of cholesterol-rich foods. This is clinical, rather than public health advice.
Dietary Fats and Cancer

The 2nd edition of Dietary Guidelines for Australians (2) stated that “epidemiological evidence suggests that total fat intake is associated with cancer of the breast and of the large intestine”.

More evidence has accumulated on this question. From nine prospective cohort studies on diet and breast cancer reviewed by the British Committee on Medical Aspects of Food Policy (96), the evidence is moderately consistent that no association exists between higher total and saturated fat intakes and risk of breast cancer (96). The World Cancer Research Fund agrees on these prospective studies (97).

In experiments with rats given chemical carcinogens, there has been more mammary tumour development with diets containing moderate amounts of ω-6 PUF than with the fat saturated, eg lard. The FAO/WHO expert consultation (80) found that tumour yields increase with linoleic acid added up to a threshold of 4 to 5 percent of total calories. When this threshold is reached, increasing total fat causes further increase in tumours but this is independent of type of fat. In human epidemiology, in the US Nurses cohort there was no correlation of breast cancer with total fat, or with PUF (98). Colorectal cancer in this cohort was associated with total and saturated fat, not with PUF intake (99).

Zock and Katan made a systematic review of the case-control and prospective cohort studies reporting linoleic acid or PUFA and cancer incidence (100). For case-control studies the combined relative risks were 0.84 for breast, 0.92 colorectal cancer. For prospective studies combined relative risks were 1.05 for breast (NS), 0.92 for colon and 0.83 for prostate cancer. In Europe, adipose tissue fatty acids were analysed in breast cancer cases and controls in five cities (in 5 countries). Except in Spain the linoleic acid percentages were practically identical (101).

Except in the cohort reported by Willett et al (100) no association between total fat and colorectal cancer has been found in a total of 8 reported prospective studies (96).

Thus, with much more epidemiological data than was available for the 1992 Dietary Guidelines, particularly prospective studies, it would seem now that there is little or no increased risk from cancer in adults from total or type of fat intake. Cancer is not mentioned in the fats section of the 2000 edition of Dietary Guidelines for Americans (52).

Dietary Fats and Micronutrients

Margarines and dairy foods (not fat reduced) contain retinol and β-carotene and are important sources of vitamin A activity. In the 1995 National Nutrition Survey (6) milk and products provided one-quarter to nearly half the preformed vitamin A in different age groups and margarine provided over 10%. They provide some β-carotene as well. Carotenoids are better absorbed from meals that contain fat or oil (102).

Most foods do not contain vitamin D. The best sources are margarines (to which it has to be added by regulation at 8µg/100g) and some oily fish, eg herring, pilchards, sardines. In the British food tables (103) margarines contain 7.9 µg vitamin D/100g and sardines 7.5 µg. The vitamin D of butter is only 0.76 µg and of cheese 0.26. Margarine was supplying
50 to 60% of vitamin D in Australia in the 1980s. Margarine consumption has, however, declined somewhat in the 1990s. As more cases of vitamin D deficiency are being reported, this extra component is important in margarine and some fish.

Vitamin E comes nearly all from vegetable oils and products made from them. Sunflower oil (49 mg/100g), cottonseed (43 mg), safflower (41 mg), palm (33 mg), canola (22 mg), corn (17 mg), soya (16 mg) and peanut oil (15 mg) are good sources and olive oil contains 5 mg/100 g (103). Margarines made from these oils contain the corresponding amounts of vitamin E and here food regulations permit extra vitamin E in margarines as an antioxidant additive.

The only inorganic nutrient of any consequence in fats and oils is sodium chloride (salt), added traditionally to butter and margarines. This salt is not inherent or needed for processing. Standard varieties of these yellow fat spreads can be classified high salt foods (104) so, with the dietary guidelines to “Choose low salt foods”, reduced salt and low salt margarines are recommended.

CONCLUSIONS

Total fat is providing about one-third of dietary energy in Australia. It appears to have declined a little but it is still relatively high from a world perspective. For anyone overweight a reduction of total fat to 20 to 25% energy should be a major part of dietary management, together with more physical exercise. This is a public health matter because about half the Australian adult population were overweight in the 1995 National Nutrition Survey.

The biological effects and health risks of dietary fats and oils are determined in large part by their predominant fatty acids.

Saturated fatty acids raise plasma LDL-cholesterol, a major risk factor for coronary heart disease (CHD). In three large prospective epidemiological studies, saturated fatty acid intake was directly associated with subsequent CHD. Trans unsaturated fatty acids appear to behave similarly, though their consumption is now small in Australia. Saturated plus trans fatty acid intakes averaged over 12.5% energy in 1995. A population average of 10% energy is recommended.

Intake of omega-6 polyunsaturated fatty acids (essentially linoleic) should be in the range 6 to 8% of energy because there is strong evidence that they protect against CHD by lowering plasma LDL-cholesterol and probably by reducing the risk of dangerous cardiac arrhythmias.

Omega-3 polyunsaturated fatty acids occur in fish and a few vegetable oils. Present intake of these fatty acids is low (approx 200 mg). It would appear desirable to double this intake as a measure aimed to reduce the risk of CHD. This recommendation brings challenges for the environment and for the fats and oils industry. Possibilities of producing from single cell culture the biologically active omega-3 polyunsaturated fatty acids EPA and DHA found in fish deserve further research.
Monounsaturated fatty acids do not raise plasma cholesterol and do not have the action of polyunsaturated fatty acids on arrhythmia. Present intake levels appear to be satisfactory except in individuals who need to reduce total fat as part of body weight management.

Dietary cholesterol intake will decline if people eat smaller amounts of saturated fat, since these two lipid classes usually occur in the same foods.

**PRACTICAL ASPECTS OF THIS GUIDELINE**

**Fats and Oils**
- Choose vegetable oils – sunflower, canola, corn, soya, olive, flaxseed – rather than palm or coconut oil.
- Use yellow spread for bread and for baking choose unsaturated tub margarines rich in omega 6 and omega 3-polyunsaturated with monounsaturated fat, made from canola or sunflower or safflower or olive oils rather than butter or hard (stick) margarine.
- Try to eat 2-3 fish meals per week, preferably oily fish (sardines, tuna, salmon, herring) and grilled.
- Use low fat milks (1% or 2% fat) in place of full cream milk (4% fat).
- Choose low fat yoghurts in place of full cream yoghurt.
- Restrict your consumption of hard (full fat) cheeses. (They contain about 75% energy as fat, mostly saturated). Look for reduced fat hard cheeses and especially cottage cheeses.
- Use cream only as an occasional luxury. Choose reduced fat forms.
- Buy lean cuts of meat and trim the obvious fat before eating. This includes discarding the skin of cooked chicken.
- Limit consumption of sausages, fatty mince, processed meats and luncheon meats (ie higher fat meat products).
- Discard fat drippings from cooked meat.
- Limit fried savoury snack foods such as potato crisps. Prefer those fried in cottonseed or sunola oil.
- Limit consumption of biscuits (which are high in saturated fat).
- Limit consumption of bought pastry products. If making pastry at home, use (poly)unsaturated margarine.
- Limit consumption of cakes. If making cakes at home, use (poly)unsaturated margarine.
- Eat only sparingly chocolate and chocolate-containing confectionery.
- Limit your intake of foods with creamy sauces and gravies. If preparing sauces at home, use (poly)unsaturated margarine.
- Choose (poly)unsaturated salad creams and dressings.
- Use a little sunflower oil or olive oil for frying, not butter, dripping, lard or palm oil. Use non-stick pans and minimise frying fats.
- Be moderate in your use of eggs, eg an average (whole or in dishes) of one a day.
EVIDENCE

Level II evidence exists for the effect of diet fat on overweight (refs 18, 19) and Level III evidence from metabolic trials of the effect of various dietary fats on plasma cholesterol - (refs 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 53, 54, 91).

Level II and III evidence exists for the effect of diet fat type on coronary heart disease from controlled intervention trials (8/10 randomised) (refs 61, 62, 63, 64, 65, 66, 67, 68, 87, 88) as well as Level III evidence from prospective studies (refs 39, 40, 41, 42, 43, 44, 45, 46, 58, 59, 60, 85, 86, 93, 94, 95); biomarker case-control studies (refs 45, 46).

Level III evidence for the effect of dietary fat on cancer is available from prospective studies (refs 96, 97, 98, 99, 100) and biomarker case-control studies (refs 100, 101).
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51. Estimate of present trans fatty acid consumption in Australia.


78. Standing Sub-Committee appointed by the National Heart Foundation of Australia to maintain a continuing review of research developments in the field of diet as related to heart disease. Dietary fat and coronary heart disease : a review. Med J Australia 1967; j : 309-322.
CHOOSE FOODS LOW IN SALT

BACKGROUND

Salt and sodium
Salt is a simple compound consisting of molecules of sodium chloride, and is found naturally in many foods. Salt is also added to many foods because of its preservative and flavouring characteristics. It has been shown that both the sodium and the chloride can be detrimental to health when consumed in excess (1). About 90% of all the sodium added to food is sodium chloride and therefore dietary intake of sodium represents intake of sodium chloride for practical purposes.

The current NHMRC recommendation for the Australian population in general is that dietary sodium intake be under 2,300 mg (100 mmol) per day (2).

A low salt food is defined by the Australia and New Zealand Food Authority as one with a sodium concentration of up to 120 mg/100g (Joint Australia and New Zealand Food Code, clause 17 of Standard 1.2.8, also p15 of ANZFA Code of Practice on Nutrient Claims).

The conversion factors for the units used to express sodium content of food are:

1 mmol = 23 mg
1 g = 43 mmol
1 g sodium chloride (NaCl) contains 17 mmol sodium, or 391 mg sodium.

NEED FOR THIS GUIDELINE

It is now well established that a reduction in dietary sodium intake will decrease the mean population blood pressure and will reduce the prevalence of hypertension. It has not yet been conclusively established that a mean dietary sodium intake within the range recommended for Australian adults will result in lower morbidity and mortality rates than at present (3), but the balance of evidence suggests it will. Similarly, it has not been demonstrated that a mean dietary sodium intake within the range recommended for Australian adults for many years will result in a lower incidence of hypertension (i.e. prevent the occurrence of hypertension), but again the balance of evidence suggests it will. It has been said that ‘few measures in preventive medicine are as simple and economical and yet can achieve so much’ (4).

The risk of stroke and ischemic heart disease increases continuously with blood pressure as indicated by 9 major prospective cohort studies (5). Within the range of diastolic blood pressure studied (about 70 - 110 mm Hg) there is no evidence of a threshold below which the relationship alters. If dietary salt were decreased by an average of 3g (50 mmol sodium) per day, the average systolic blood pressure of those over 50 years of age would be expected to fall by about 5 mmHg; the diastolic blood pressure would be lowered by about half as much. The minority who are hypertensive would experience a greater average blood pressure fall, but the reduction in the number of new cases of cardiovascular diseases would be greatest for the large proportion who are close to average blood pressure. It has been estimated that a reduction in dietary salt by an average of 3g (50
mmol sodium) per day in a whole Western population would reduce age-specific stroke mortality by about 22% and ischemic heart disease mortality by about 16% (4, 6).

Cardiovascular disease is the largest cause of premature death and death overall in Australia, accounting for 50,797 deaths or 40% of all deaths in 1998 (7). Most of these deaths are due to coronary heart disease (55%) followed by stroke (24%). Each year, around 40,000 Australians have a stroke, with 70% of these being first ever strokes. Stroke is the cause of nearly 25% of all chronic disability in Australia (8). Hypertension is the most frequent problem seen by general practitioners, accounting for 5.7% of all problems (9). In 1995, an estimated 2.8 million Australians aged 18 years and over reported a recent and/or long term cardiovascular condition. High blood pressure was the most common condition for both males and females (10). The prevalence of these conditions increased with age and was 61% among those aged 75 years and over. Although the death rates from cardiovascular disease between 1985 and 1996 in Australia declined by about 3.6% per year in both males and females, the number of people with disease is expected to increase over the next few decades as the number of older people increases and life sustaining treatment improves. The total direct costs of cardiovascular disease was estimated to be $3,719 million in 1993-94, 12% of the total health care costs for all diseases (11).

A recent review (12) has indicated a number of health conditions other than raised blood pressure that are associated with excess sodium intake. They include any condition exacerbated by water retention (including heart failure, cirrhosis, nephrotic syndrome, idiopathic and cyclic oedema), stroke (independently of blood pressure), gastric cancer, and left ventricular hypertrophy. Excess sodium intake also increases the rate of kidney function deterioration in patients with renal disease, is associated with urinary stones and may aggravate asthma (13) and osteoporosis. Excretion of sodium is associated with an obligatory loss of calcium due to interference with the tubular reabsorption of calcium. Calcium is conserved on low salt intakes and wasted on high salt intakes.

SCIENTIFIC BASIS

The scientific literature relating dietary sodium intake to blood pressure is extensive and dates back more than 100 years (14). In this section it is only possible to summarise the state of our knowledge, highlight important studies and draw attention to important issues. While the literature provides evidence that it is possible for different commentators to interpret similar scientific results quite differently, there is general consensus on most issues.

The extent to which dietary sodium reduction reduces blood pressure is generally agreed to depend on age and initial blood pressure – it is greater with age and at higher blood pressures (15, 16). Law et al (16) have found that the full effect of dietary sodium reduction on blood pressure is not seen for at least 5 weeks.

Major original studies have included animal studies and human studies. Controlled experiments in chimpanzees have shown an important effect of dietary salt reduction on blood pressure (17), consistent with earlier experiments on rats (18). For chimpanzees that were allocated a high sodium diet for only 2 years, up to 6 months of a low sodium diet was required before blood pressure lowering had reached its greatest extent.
In relation to human populations, each of three recent reviews (3,6,19) is in agreement that dietary sodium reduction is associated with reduction in blood pressure. Kuller (19) and Law (6) call for a public health approach to lowering salt in the diet – i.e. for the average salt intake of the population to be lowered through reducing the amount of salt entering the food supply. Alderman (3), however, calls for randomized controlled studies of the long-term health benefits and safety of dietary sodium reduction and concludes ‘without knowledge of the sum of the multiple effects of a reduced sodium diet, no single universal prescription for sodium intake can be scientifically justified’.

Meta-analyses of Sodium and Hypertension Trials

Law et al have taken a different approach to a standard meta-analysis. They analysed cross-sectional data from 24 different communities worldwide involving more than 47,000 people (16) and derived relationships between dietary sodium intake and blood pressure that depended on age, centile in the blood pressure distribution, and development of the community. They tested the associations on 14 studies examining the association of blood pressure with sodium intake within populations, importantly after adjusting for the large effect of regression dilution bias (20). They found the within population associations consistent with the relationships estimated using between population data. Finally they examined how closely the results from 68 crossover trials and 10 randomised controlled trials conformed to the relationships they originally estimated from between population studies (4). They found that for the 33 trials lasting 5 weeks or more the observed reductions in blood pressure were similar to the predicted values (within 95% confidence intervals for 30 out of 33 trials). For trials last less than 5 weeks they found that the predicted fall in blood pressure was less than the observed fall, leading them to conclude that dietary sodium reduction does not have its full effect on blood pressure until at least 5 weeks of intervention. The consistency of results from different study types, and in different populations, have increased the investigators confidence in their estimates for the relationship between dietary sodium intake and blood pressure (4).

The meta-analysis of Cutler et al published in 1997 (21), updates an earlier meta-analysis conducted by Cutler and others (22). They include 32 trials in their analysis and conclude ‘the blood pressure reduction that would result from a substantial lowering of dietary sodium in the US population could reduce cardiovascular morbidity and mortality.

Midgley et al (23) include a total of 56 trials in their analysis and conclude ‘dietary sodium restriction might be considered, but the evidence in the normotensive population does not support current recommendations for universal dietary sodium restriction’. Graudal et al (15) include 58 trials of hypertensive people and 56 trials of normotensive people in their analysis and conclude ‘These results do not support a general recommendation to reduce sodium intake…but ideally trials with hard end points such as morbidity and survival should end the controversy’.
Table 1 summarises the results from the meta-analyses.

**How to interpret the different conclusions between meta-analyses?**

The first point to be made is that the estimated effects are not greatly different from one another. The effects are lower than what might be expected because the inclusion of all randomized subjects (intention to treat analysis) dilutes the number of subjects actually complying with the intervention treatment. There are four effect-modifying factors in determining the extent to which change in dietary sodium will change blood pressure. They are the magnitude of the change in sodium intake, the age of the subjects, the initial blood pressure of the subjects and the duration of the intervention. Ideally, one would like to conduct meta-analyses at different levels of these factors (as has been done for normotension vs hypertension). Many of the studies included in the meta-analyses were of short duration and conducted on young people. This would also tend to decrease the effect measure observed. Those studies with larger dietary sodium reduction also tended to be the shorter studies because of the difficulty in maintaining a free-living population at a low sodium intake for a long period of time. Technical issues such as how to weight individual trials and how to construct a summary regression line differed between meta-analyses. The choice of methodology alters the summary estimates considerably.

In effect, the meta-analyses indicate what might be expected from a dietary sodium reduction intervention undertaken in the current food environment where avoiding dietary sodium is relatively difficult. Even under these circumstances there is a fall in blood pressure for both hypertensive and relatively young normotensive subjects.

**Table 1**

The decrease in systolic and diastolic blood pressure on reduction of dietary sodium – meta-analyses of randomized controlled trials (15, 21, 23).

<table>
<thead>
<tr>
<th></th>
<th>Normotensive Subjects</th>
<th>Hypertensive Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Diastolic blood</td>
<td>Systolic blood</td>
</tr>
<tr>
<td></td>
<td>pressure</td>
<td>pressure</td>
</tr>
<tr>
<td>Midgley 1996</td>
<td>0.5 mmol</td>
<td>1.6 mmol</td>
</tr>
<tr>
<td></td>
<td>(Weighted average 125mmol difference in dietary sodium)</td>
<td>(Weighted average 95mmol difference in dietary sodium)</td>
</tr>
<tr>
<td>Cutler 1997</td>
<td>0.8 mmol</td>
<td>1.5 mmol</td>
</tr>
<tr>
<td></td>
<td>(Weighted average 71mmol difference in dietary sodium)</td>
<td>(Weighted average 76mmol difference in dietary sodium)</td>
</tr>
<tr>
<td>Graudal 1998</td>
<td>0.3 mmol</td>
<td>1.2 mmol</td>
</tr>
<tr>
<td></td>
<td>(Weighted average 160mmol difference in dietary sodium)</td>
<td>(Weighted average 118mmol difference in dietary sodium)</td>
</tr>
</tbody>
</table>

No randomized controlled trials have been conducted to test the effectiveness of dietary sodium reduction in primary prevention of hypertension, and there is little information from randomized controlled trials on the effect of dietary sodium reduction on mortality or morbidity from cardiovascular disease (3).
It is more enlightening to examine the results of two large randomised controlled trials that were published after the meta-analyses. Both the DASH-Sodium trial (24) and the TONE study (25) provide compelling evidence for the benefits of dietary sodium reduction.

DASH-Sodium was a sequel to the first DASH (Dietary Approaches to Stop Hypertension) study (26). Both were multicentre randomized controlled trials. The first held sodium constant at 130 mmol/day and compared the standard American diet with an 'ideal' diet that emphasized fruits, vegetables, low-fat dairy foods, fish, legumes, nuts and lean meat and poultry while DASH-Sodium (24) repeated the experiment at three sodium levels -- the US guideline of 104 mmol/day plus or minus 39 mmol/day (143, 104 and 65 mmol/day). The first DASH study obtained a highly significant fall in blood pressure with the 'ideal' diet, and the DASH-Sodium study demonstrated incremental further falls at 104 mmol/day and 65 mmol/day, confirming that an otherwise ideal diet is more effective when it includes a sodium guideline. All the food was provided to the participants, thereby controlling the important confounding variable of dietary compliance – an important difference between DASH-Sodium and previous sodium studies.

The mean decrease in systolic blood pressure when changing from 143 mmol/day on the control diet to 65 mmol/day on the DASH diet was 7.1 mmHg in normotensives and 11.5 mmHg in hypertensives, the latter deriving as much benefit as they might expect from antihypertensive medication (27). While on the control diet only and changing from 143 mmol/day to 65 mmol/day, the change in systolic blood pressure was 9.8 mmHg for negroes with hypertension and 6.8 mmHg for other racial groups with hypertension.

Reducing the sodium intake by 39 mmol/day caused a greater decrease in blood pressure when the starting sodium level was 104 mmol/day (which is the upper level of intake currently recommended in the US). The combined effects on blood pressure of low sodium intake and the DASH diet were greater than the effects of either intervention alone and were substantial.

The first DASH trial was widely misinterpreted as having negated the importance of other factors in hypertension such as overweight, alcohol and sodium intake (24), but the design of the first study purposely omitted the well established factors in order to test the other general dietary guidelines (27). Long term health benefits of the DASH-Sodium diet remain to be demonstrated, but this large randomized controlled trial with high subject retention rates and excellent compliance to dietary protocols has provided compelling evidence that true reduction of dietary sodium has a substantial effect on blood pressure. The effect on normotensives was enough to make it a guideline for the whole population and to predict a substantial effect at the population level (27).

The TONE study was a randomized controlled trial of reduced sodium intake or weight loss in older hypertensives aged 60 to 80 years (25). Of the 975 subjects, 585 were obese and 390 were not obese. Withdrawal of hypertensive medication was a goal for all subjects. Follow-up visits at 9, 12 and 30 months had attendance rates of 91%, 86% and 86% respectively.

The sodium reduction group reduced their intake by a mean of only 46.6 mmol/d at 9 months, 49.3 mmol/d at 18 months and 39.5 mmol/d at 30 months. The goal for sodium reduction was a total intake of 80 mmol/d or less, and only about 38% of the subjects met this target at each visit compared to about 11% in the control groups. This modest
compliance resulted in about a 30% decrease in the need for antihypertensive medication in the sodium reduction group, and a better result in subjects who combined weight loss with sodium reduction. The modest dietary sodium reduction of about 40 mmol/day was well tolerated and sustained, and the subjects reported no adverse effects (25).

PRACTICAL ASPECTS OF THIS GUIDELINE

Relationship to Australian Guide to Healthy Eating (AGHE)

The Australian Guide to Healthy Eating (29) focuses on food and food selection. Individuals should avoid selection of higher salt foods and replace them with other foods within the same group that are lower in salt. Much of the salt intake of Australians comes from recommended foods such as bread, cereals and cheese, and other frequently consumed foods such as butter, margarine and snack foods. It has become apparent that the source of most dietary sodium is not discretionary salt (30, 31, 32, 33). In particular, cooking salt is a much less important source than was once thought. James et al. (30) used lithium as a marker and found that only a quarter of cooking salt actually enters the consumed food (the rest is discarded with the cooking water).

The estimated percentages of sodium from different sources were:

<table>
<thead>
<tr>
<th>Food Sources of Sodium</th>
<th>James et al.(30)</th>
<th>Edwards et al.(31)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Natural</td>
<td>10.0</td>
<td>18.5</td>
</tr>
<tr>
<td>Added by Food Manufacturers</td>
<td>75.0</td>
<td>65.0</td>
</tr>
<tr>
<td>Discretionary</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- cooking</td>
<td>9.0</td>
<td>9.0</td>
</tr>
<tr>
<td>- at table</td>
<td>6.0</td>
<td>6.0</td>
</tr>
</tbody>
</table>

While advising people to consume less salt and to seek to purchase low salt foods is important, the widespread use of salt in processed food and food prepared away from home is a significant barrier to achieving significant reduction in dietary sodium intake. The important public health objective of reducing the mean intake of sodium to at least the top end of the RDI range (34) requires more universal action. Realistic medium term policy objectives would include a gradual reduction in the amount of salt added to processed food, and consumer friendly labelling of the sodium content of food.

The recommendation that food manufacturers reduce the amount of salt added to their product is not without precedent. In 1982, the NHMRC Working Party on Sodium (35) recommended that food manufacturers be requested to reduce the sodium added to foods during manufacture and processing. Foods such as bread, cheese, butter, margarine, processed meats and snack foods were named as items of critical importance. Food manufacturers responded to this call with the range of reduced and low salt alternatives now available. The US National High Blood Pressure Education Program Working Group called for food processors to lower the sodium content of their products (36) and there is at least some evidence that a modest sodium content reduction may have occurred (37). While information on sodium content is now included on food labels, it may be difficult to
comprehend, partly because the content is given as density (per 100g) and per standard serve, it is easy to confuse salt and sodium, and also because the sodium content may need to be considered in terms of the actual amount of the food consumed.

Compositional information on prepared food such as takeaway foods and restaurant meals is particularly difficult for the consumer to access.

When consumers reduce the amount of salt in their diet, the rated intensity of salt in a solid food increases, and the concentration of salt in soup and crackers that produced maximum pleasantness decreases (38, 39). The effects are observed within 2 months although they may take many months to reach their full extent.

In clinical trials, moderate sodium reduction was not associated with physical complaints or with impairment of the quality of life (40, 41).

**Sodium Sensitivity**

People vary in the sensitivity of their blood pressure response to dietary sodium (42, 43, 44), and salt sensitivity may be related to mortality independently of blood pressure (45). Salt sensitivity appears to be a continuous phenomenon (46), and the definition of salt sensitivity is arbitrary, however it appears to be reproducible in individuals (44). A clinically practical means to identify it is yet to be found (43). It has been shown that the prevalence of ‘sensitivity’ increases with age (46), raising doubts about the persistence of a determination of being dietary salt ‘insensitive’. Salt sensitivity is also associated with defective endothelial-dependent vasodilation in people with hypertension (47) however the reason for this is not understood. Salt sensitivity is an active area of research and a better understanding of the mechanisms are likely to improve understanding of the health effects of dietary salt intake.

**Changes needed to current diet**

The Recommended Dietary Intake for Australian adults for sodium is 40-100mmol/day, with no extra recommendation for pregnancy or lactation (34).

Sodium intake is poorly measured by many dietary survey methods that are used to measure other food components because foods of similar type vary widely in their sodium content (eg breakfast cereal) and dietary addition can be discretionary but not easily quantifiable (eg adding salt in cooking and at meals). Measurement of sodium output in urine over a 24 hour period is an accurate way of estimating sodium intake, with about 93% of dietary sodium being recovered in the urine (30). Day-to-day variation in sodium intake is high (20, 48), so that intake for a single day usually does not accurately reflect a person’s usual long term intake.

Two small Australian surveys based on systematic samples from the Commonwealth electoral roll have produced similar findings (49, 50). The larger and more recent survey (49) found that men (n=87) had a mean urinary sodium excretion rate (±SD) of 170 (±52) mmol a day and women (n=107) had a rate of 118 (±42) mmol a day. The range for men for a single day was 39–337 mmol and for women 26–241 mmol. In a Sydney survey of adults mostly from a university community, the figures were very similar, except for
subjects with an Asian diet, where the men (n=29) averaged 195 mmol a day of urinary sodium and the women (n=21) averaged 140 mmol a day (51).

The results from ordinary Australian diets are similar to those of respondents selected from the UK electoral roll a decade earlier (52), where the mean urinary sodium excretion rate for men (n=681) was 171 mmol a day and for women (n=712) 132 mmol a day, with a range for men of 20–498 mmol a day and for women 21–354 mmol a day.

Population surveys show that women excrete about 20–25 per cent less sodium than men, but the gender difference was non-significant after adjusting for creatinine excretion in the 1995 Hobart data, suggesting that it reflects the lower muscle mass and thus food energy intake of women (53). A lower RDI of perhaps 30–80 mmol a day may be more appropriate for women, and downward adjustment for older people of both sexes could be proposed on the same grounds.

There is a need for more systematic monitoring of the sodium intake of Australians. People wanting to achieve a low sodium intake should closely adhere to the guideline ‘choose low salt food’ (i.e. sodium content up to 120 mg/100g). Fresh foods such as fruit, vegetables, meat, milk and yoghurt are well under the sodium limit but most manufactured foods are well over it (54). Breads have a sodium content typically as high as 400-725 mg/100g. ‘Salt-free’ bread can be difficult to find, but it (and other low salt products) should be sought out. People used to a higher salt intake will at first miss the taste of salt when they begin a lower salt intake. The palate adapts to lower sodium levels and people will find that the intensity of salt in food increases and their ‘preferred saltiness’ of food reduces (38, 39). Changes will be noticed within a week and taste will continue to change for many months.

Salt substitutes are available as a flavouring, however this maintains the preference for a ‘salt taste’. Most people following a low salt intake are more than happy with the hundreds of other ways of adding flavour to their food and do not miss salt after their palate has adapted. Lite Salt (NaCl plus an equimolar amount of potassium chloride) is a practical alternative, but carries a warning on the packet not to use it without medical advice (because of the interaction between potassium chloride and ACE inhibitors and also potassium-sparing diuretics). The use of salt substitutes is likely to be only marginal because they lower the discretionary salt intake which is only a minor source of salt in the diet.

Asian-style cooking has increased in popularity in Australia in recent years. As mentioned above, people from a university community in Sydney who consumed an Asian-style diet had a sodium intake that was about 15% higher (51). Many Asian dishes use ingredients such as soy, oyster and fish sauce which are high in sodium. In the INTERSALT study (involving urinary measurement of sodium excretion at 52 sites from 32 countries), the study sites of northern Japan and the Peoples Republic of China were among those with the highest sodium intakes (55).

Special groups/needs

Cardiovascular disease mortality is higher among indigenous Australians, in rural areas and among socioeconomically disadvantaged groups (11). There is a paucity of information on the salt intake of indigenous Australians. A survey of the food habits of
adults living in Victoria (56) indicated that indigenous Australians, whether living in the city or rural towns, were much more likely to add salt to cooked food than European Australians.

The entire population experiences an age-related increase in blood pressure which can probably be largely prevented by the population consuming dietary sodium within the recommended dietary intake. In the current food environment, it is very difficult to maintain a low dietary sodium intake – what is needed are population measures to gradually decrease the amount of salt in our food supply and increase the ability of people to make healthy choices in relation to their food intake.

Hypertensive patients especially need a better choice of groceries than at present. During the 1980s a major supermarket chain brought out a range of no-added-salt processed foods consisting at one point of 23 different items (57). Producers will meet consumer demand, and there is no question that this range would have increased much further if the turnover had reflected the real needs of approximately 3 million Australians who have hypertension. But the Australian medical profession at present ignores the international consensus which is in favour of improving the lifestyle before starting drug treatment (58, 59). Except for the emergency treatment of severe cases, medication for hypertension should be considered only after a six-month trial of non-pharmacological measures including a low salt diet, and when drug treatment is necessary these measures should be continued as an adjunct, to permit better control at a lower dose (58,59). Doctors behave very differently in Finland, where the government withholds the subsidy for antihypertensive medication unless the prescriber implements this policy (60).

The financial implications of the salt guideline are considerable in Australia, where the Heart Foundation has been advising doctors for a number of years that effective non-pharmacological measures may abolish the need for medication altogether in up to 40% of patients with mild hypertension (61). Most antihypertensive drugs are more effective at a low salt intake, especially some of the more expensive drugs such as ACE inhibitors and angiotensin antagonists. Prescriptions for a single ACE inhibitor (enalapril) cost the Australian government about $60 million in 1998 (62), an amount that could be substantially reduced if patients followed the salt guideline.

Diuretics (at the low dosages currently recommended) have no effect on BP at sodium excretion rates above 190 mmol/day (63) and are of no practical value at or below 70 mmol/day (64). They are absolutely contra-indicated below 50 mmol/day (65) because of the danger of iatrogenic hyponatraemia and lack of benefit.

Iodine deficiency disorders were once common in several Australian states and a traditional control measure has been the sale of iodised salt. Only table salt has been iodised, and its use has declined to the point where over 50% of both sexes stated in a Hobart survey in 1995 that they neither cooked with salt nor used it at the table (49). Mild iodine deficiency is now regarded as an important cause of preventable mental retardation, and it is alarming that urinary excretion has revealed moderate to severe iodine deficiency even in a survey of out-patients (including pregnant women) at a metropolitan hospital in Sydney (66). Observation of the salt guideline reduces its availability as a vehicle for iodine, and iodine fortification of one or more staple foods such as bread may need to be considered.
RELATION TO OTHER GUIDELINES

This dietary guideline is not inconsistent with any of the other dietary guidelines. Many manufactured foods have a large amount of salt added during their manufacture, and these should be avoided while maintaining (or attaining) a healthy intake of breads, cereals, fruit and vegetables. This would be much more easily achieved if manufacturers were to decrease the amount of salt added to their products and consumers were easily able to assess the sodium content.

CHANGES FROM PREVIOUS GUIDELINES

The first edition of the Dietary Guidelines for Australians (2) included the guideline ‘Eat less salt’. The second edition of the guidelines revised this guideline to become ‘Choose low salt foods and use salt sparingly’ in recognition of the fact that the major source of sodium in the Australian diet is salt added to manufactured food. The present review finds no reason to alter this guideline.

CONCLUSION

The last decade has produced an international consensus that a modest dietary sodium reduction for people with normal and raised blood pressure has a large enough effect on blood pressure (and therefore health benefit) to justify a guideline advising restraint for the entire population (67). This has now been strongly supported by a large and well-conducted randomized controlled trial (24) where subject retention was high and dietary compliance optimized by providing all of the food throughout the trial period.

The proportion of the population who would benefit at older ages from lower dietary sodium is becoming increasingly large, yet salt in foods is difficult to avoid, mainly because of the large amount added by the manufactured food industry. Lifelong intake of dietary sodium within the recommended dietary intake range would remove the often stated difficulty of reducing dietary sodium intake in later life. The Australian diet contains an unnecessarily large amount of salt and a gradual reduction will certainly benefit the large numbers of people presently destined to develop hypertension, and probably benefit a substantial proportion of people who would otherwise develop disease, in particular cardiovascular disease.

The primary prevention of hypertension provides one of the greatest challenges to public health in the 21st century, and the reduction of dietary salt is a leading contender for its achievement.
EVIDENCE

There is evidence that dietary sodium reduction reduces the average blood pressure in groups of people where blood pressure is raised is strong. The evidence is derived from well-conducted randomised controlled trials and is supported by meta-analyses of these (Level I evidence; refs 15,21,22,23). The size of the effect is clinically important, and is even larger for older individuals and at higher blood pressure. The effect appears to be evident for ‘high-normal’ blood pressure – i.e. when blood pressure is not high enough to be categorized as ‘hypertensive’. The evidence is relevant although randomised controlled trials have not been conducted to assess mortality or morbidity endpoints (other than change in blood pressure). There is substantial and strong evidence relating raised blood pressure to both morbidity and mortality (see ref 28).

There is also Level II (refs 4, 18, 24, 25, 26, 40, 41), Level III (refs 5,14, 45) and Level IV (refs 42, 49, 50, 52) evidence of the relationship, between blood pressure and salt reduction but the evidence that dietary sodium reduction reduces the average blood pressure in groups with normal blood pressure is not strong. Most of the subjects with normal blood pressure in the randomised controlled trials are also young, and it is suggested that the size of the effect depends on both age and the initial blood pressure. The relevance of this finding is positive because there is no scientific or theoretical evidence of benefit for lowering blood pressure below normal.

The evidence for dietary sodium reduction to the recommended dietary intake of between 40-100mmol/day causing adverse health effects is weak. Adverse health effects have not been observed in the randomised controlled trials conducted to date. Adverse health effects that become apparent over long time periods seem unlikely based on the low dietary sodium intake observed in many populations without apparent specific ill-effect, and the presumed low dietary sodium intake by humans over much of their history.
REFERENCES:

42. Luft FC, Weinberger MH. Heterogeneous responses to changes in dietary salt intake: the salt-sensitivity paradigm. Am J Clin Nutr 1997;65:612S-7S.
LIMIT YOUR ALCOHOL INTAKE IF YOU CHOOSE TO DRINK

DEFINITIONS

Standard Drink
Contains 10 grams of alcohol (equivalent to 12.5mls alcohol).

Pattern of drinking
Refers to aspects of drinking behaviour other than level of drinking, including the number and characteristic of drinking occasions, and the types of drinks consumed.

BACKGROUND

Alcohol has been consumed in various forms in Australia since the earliest days of European settlement. Alcohol, however, was not at that stage, part of the diet of the original Australians, the Aboriginal people. It was introduced by the British with the arrival of the First Fleet on Australian shores in 1788.

In terms of nutrition, alcohol is in a somewhat unique position in that it is the only substance that is both a nutrient and a drug affecting brain function (classified as a general anaesthetic). Advice about alcohol was omitted from the first two international sets of dietary goals or guidelines produced in the Nordic countries in 1968 and in the United States in 1997. In 1997 a Lancet editorial (1) in welcoming the development of dietary guidelines for the community pointed out “their major blind spot is to ignore alcohol consumption, which is increasing fast, along with its pernicious effects”. Since then, caution about alcohol intake has been included in nearly all national sets of dietary guidelines, from North America, Europe, Asia and Oceania. Alcohol is the fourth macronutrient (together with carbohydrates, fats and proteins) which provides dietary energy (kilojoules) and inclusion in the dietary guidelines is justified on this point alone.

The main reason why people take alcoholic drinks is for the relaxing and socialising effect on the brain of small to moderate doses. The reasons why health authorities caution about alcohol use is that high doses severely impair brain function (can produce coma and even death from direct intoxication) and alcohol is a habit forming drug, the most commonly used recreational drug in Australia (after tobacco). Alcohol also can have toxic effects on other systems in the body, directly or indirectly and can affect, amongst other things, liver function, the cardiovascular system, several metabolic processes, fetal development and the cancer process. Alcohol is an outstanding example of the toxicological principle “the dose determines the effect”.

Society’s attitudes to alcohol, and its use, are markedly ambivalent. Many social occasions, from celebrations of births and birthdays, engagements, graduations, weddings and job promotions, and nearly all entertainment and hospitality, are accompanied by the dispensing of alcoholic drinks. Vineyards and wine making are seen as romantic; wine tasting is considered a sophisticated occupation or pastime; some wines fetch enormous prices. Beer and the pub are important for mateship. Production and retailing of alcoholic drinks provides large numbers of direct and indirect jobs. Wine is our most rapidly
growing agricultural export and the government receives considerable revenue from the
taxes on wine, beer and spirits.

At the same time, there are restrictions and avoidances. Many people abstain or drink little
and seldom. There are restrictions on who may sell and who may serve alcohol and to
whom and when they may do it. There are strict limits on the amount of alcohol people
may drink before or while driving or piloting vehicles or when operating certain
machinery and alcohol is generally not consumed in offices and workshops.

Drinking alcohol has health, social and economic costs and benefits for both individuals
and populations. People who drink small quantities of alcohol appear to have better health
outcomes than those who do not drink, although abstainers achieve better health outcomes
than heavy drinkers. It does not follow, however, that abstainers would achieve better
health outcomes by drinking as there are many social and health reasons why people
choose not to drink.

Excessive drinking of the type to cause long term harm, has been estimated to have caused
3,290 deaths in Australia in 1997, accounting for 4% of male deaths and 2% of female
deaths and 50,000 hospitalisations. Acute and chronic effects of excessive drinking made
equal contributions (2).

It has been estimated that the costs of excessive alcohol drinking to the Australian health
care system and to industry through absenteeism, premature retirement and impaired or
lost productivity was some $4.5 billion in 1992 (3). The NHMRC Australian Alcohol
Consumption Guidelines, due for release in 2001, provide a more in depth review of the
area (4).

**SCIENTIFIC RATIONALE**

The medical and social complications of alcohol consumption differ according to a
person’s pattern of drinking alcohol. Five broad drinking categories have been identified:

1. The inexperienced drinker who misjudges the dose and has an accident –
common in adolescents, indeed the major cause of death in adolescents and
young men;
2. The person who doesn’t drink during the week but may drink to excess and get
drunk on a weekend evening or at a party;
3. The person who enjoys a controlled drink or two most days with the evening
meal;
4. The person who has too many drinks each day but more or less maintains a
normal (but probably increasingly inefficient) lifestyle; and
5. The person who engages in weeks of heavy drinking (and eats very little during
this time).

Some of these patterns of consumption may result in adverse acute health and/or social
outcomes, some in long term health and/or social consequences and some in both types of
adverse outcome.
Acute health effects of alcohol (4-6)

The first potentially adverse acute effects of consumption are disinhibition and loss of skilled movements. These start to happen at blood levels of about 0.05% (0.05 g/dl or nearly 11 mmol/L). This is the legal limit of blood alcohol for driving in Australia, which was based on experiments testing such skills as reversing double-decker buses in narrow spaces after different doses of alcohol (5).

Above this intake and blood level, performance, behaviour and health deteriorate progressively. The subject is obviously drunk, then stuporose and passes into a coma. Alcohol intoxication is a major factor in violence and accidents, especially motor vehicle accidents. It also leads to losses of productivity in the workplace and to social discord, including homicide and domestic violence.

In Australia, 418 deaths on the road were caused by hazardous and harmful alcohol consumption in 1997 with consequent loss of 17,174 person-years of life as most of the deaths affected young people. There were also a total of 7,789 people hospitalised for 44,997 bed days (6) for acute conditions caused by alcohol consumption.

The Australian Alcohol Consumption Guidelines (4) which were based on a systematic review of evidence, concluded that the amounts of alcohol consumed per occasion, and more specifically, the blood alcohol concentration, is the critical feature in determining risk of injury. Blood alcohol concentrations as low as 0.04 to 0.05 may affect psychomotor skills and increase the risk of injury in circumstances such as driving. However, significant individual differences make it difficult to calculate blood alcohol concentration accurately from the number of drinks and time taken to consume them. The degree of risk also depends on the setting, the need for high order physical skills and the person’s experience with the tasks in hand.

Table 1 The proportions of types of traumatic injury and death attributed to alcohol in Australia (4)

<table>
<thead>
<tr>
<th>Harm</th>
<th>Proportion attributed to alcohol</th>
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<tbody>
<tr>
<td></td>
<td>Males (% all male cases)</td>
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<td>Road injuries</td>
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<td>Suicide</td>
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<td>47%</td>
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<td>Child abuse</td>
<td>16%</td>
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</tbody>
</table>

Table 1 The proportions of types of traumatic injury and death attributed to alcohol in Australia (4)
Chronic health effects of alcohol (4, 7-14)

There are many chronic effects of excess alcohol consumption. Perhaps the most important of these in terms of public health burden are:

- high blood pressure and stroke;
- cancer of the pharynx, oesophagus, colo-rectum, liver and female breast;
- fatty liver, alcoholic hepatitis and cirrhosis of the liver; and
- dependence and addiction

Additional long term effects related to excess alcohol consumption include:

- gastritis and gastric ulcers
- cognition
- cardiomyopathy
- aspiration pneumonia
- fetal alcohol syndrome
- convulsions
- peripheral neuropathy
- hypoglycaemic, lactic acidosis
- hyperuricaemia and gout
- hypertriglyceridaemia
- hypercortisonism
- sexual dysfunction
- acetaldehyde reaction
- Wernicke’s encephalopathy, leading to Korsakoff’s psychosis
- alcohol-related brain degeneration
- folate deficiency
- vitamin A depletion
- pellagra
- withdrawal syndrome – delirium tremens; and
- undesirable defects of interaction with drugs both pharmaceuticals and illegal recreational drugs.

An evidence-based review of the scientific literature developed as a background document for the Australian Alcohol Consumption Guidelines (4) looked in depth at the major sequelae of alcohol consumption. Some of the conclusions in relation to three major areas - hypertension, cancers and cirrhosis are summarised below.

Hypertension/stroke

The risk of hypertension increases with heavier drinking and reducing alcohol consumption will reduce blood pressure. At lower levels of drinking the picture is not as consistent and, at these levels, the possible effects of reduced alcohol consumption on blood pressure need to be considered against evidence that there are potential cardiovascular benefits associated with regular low level drinking (one or two standard drinks for men and less than one a day for women).
High blood pressure is a major risk factor for ischaemic and haemorrhagic stroke. However, alcohol reduces the risk of atherosclerosis which might overcome some of this effect in relation to ischaemic stroke. Alcohol also has a complex effect on blood clotting which may increase risk of haemorrhagic stroke and may explain in part the decreased risk of ischaemic stroke in light drinkers and increased risk in heavy drinkers.

Overall, it seems clear that heavy drinking (at risky or high risk levels) is a risk factor for both hypertension and stroke. Evidence concerning the effect of more moderate consumption is less consistent. The weight of evidence suggests that low level alcohol consumption may offer some protection against stroke. However, some studies have either shown no effect or suggested that alcohol increases risk. The pattern of drinking may also be important in determining stroke risk.

Cancer
There is clear evidence to show that alcohol is associated with an increased risk of cancer overall and that it is a cause of cancer of the mouth, throat and oesophagus. In addition, the evidence suggests that it may also play a role in other specific cancers. In particular further research is needed to clarify the possible role of alcohol in relation to breast and bowel cancer.

Unlike cardiovascular disease, there is no evidence that alcohol at any level has any protective effect against cancer. While there is a clear relationship between cancer and level of drinking, little evidence is available on the relationship between cancer risk and patterns of alcohol intake. The sole exception is that prolonged direct contact between the tissues of the mouth throat and oesophagus and drinks of high alcohol content seems to pose a higher risk of these cancers and should be avoided – and this may relate to a lower risk associated with alcohol taken with meals.

Cirrhosis of the liver
There is good evidence to show that drinking alcohol over many years can cause cirrhosis in the absence of other causes. In Australia, alcohol consumption is the most common cause of cirrhosis of the liver, and alcoholic cirrhosis is the most common cause of illness and death related to chronic alcohol consumption.

Overall, the evidence suggests that, regarding the liver cirrhosis that occurs within a given community, the degree to which alcohol is responsible for it varies with the per capita alcohol consumption.

Potential cardiovascular health benefits of alcohol - the paradox of the J-shaped curve

From prospective cohort studies reporting during the 1980s and 1990s it has emerged that people who average one or two alcoholic drinks a day have better life expectancy than teetotallers (15). This result has been found in over 20 large studies in at least 9 countries, one of them Australia (with two studies) (16,17).

Moderate intake of alcohol reduces the risk of Coronary Heart Disease (CHD) (18). It increases concentration of high density lipoprotein cholesterol (19), probably by reducing platelets ability to aggregate (20). Alcohol also increases insulin sensitivity (21) and
possibly the polyphenols in red wine (which are antioxidants in vitro) reduce atheroma formation (22). Doll concluded that for middle-aged and older men in Britain alcohol has beneficial effects in the intake range 1 to 4 units a day, in women somewhat less (23).

However, it is only in people who are carefully moderate drinkers and at an age and in a section of the population at fairly high risk of CHD that alcohol reduces mortality. For the majority of people in every country alcohol consumption increases accidents, social disruption, disease and deaths from all the acute and chronic effects of excess alcohol intake. Prospective studies are nearly always made with middle-aged people. Their results cannot be applied to the rest of the population.

Scragg in New Zealand (24) has estimated the number of deaths caused or prevented by alcohol. The association between alcohol and total mortality was related to age. Alcohol was estimated to have caused 20% of deaths among 15-34 year olds, mostly from road accidents. In contrast it prevented 0.5% of all deaths among 35-64 year olds and 3.4% of deaths among those 65 years and older due to its protective effect against CHD. For all ages combined alcohol prevented 1.5% of deaths. However, the number of person-years of life lost among ages less than 35 years was greater than those saved in the older age groups, and more in males than females.

In Australia, Mathers et al (25) estimated that the harm associated with alcohol consumption accounted for 4.9% of the total burden of disease and injury in 1996 (6.6% of males; 3.1% females). However, the protective effect of low to moderate consumption was estimated to have averted 2.8% of the total burden (2.4% in males and 3.2% in females). In comparison, tobacco smoking accounted for 9.7% of total burden, physical inactivity 6.7%, hypertension, 5.4%, obesity 4.3%, inadequate fruit and vegetable consumption, 2.7% and illicit drugs 1.8%.

**INTAKES IN AUSTRALIA**

Table 2 shows the percentiles of intake of alcohol in grams consumed on the day of the 1995/6 National Nutrition Survey. It also shows the percentiles for energy derived from alcohol. It should be emphasized that these data were gathered using a 24hr recall technique which assesses intake on the day of the survey for any given individual. It does not therefore give an estimate of the “usual” intake of individuals but can shed some light on the population consumption patterns.

In the 1995 Australian National Nutrition Survey (26), alcohol provided on average 4.8% of energy intake in men (an average of 18.5g per day) and 2.6% in women (an average of 7.3g per day). In those who reported drinking alcohol on the day of the survey, the average dietary energy provided by alcohol was 11.4% in men and 10.6% in women. Beyond these averages are a minority of people who obtain more dietary energy from alcohol than from protein.

Alcohol is nearly all consumed in beverages, principally beers and wines which contain other nutrients: sugars, some inorganics (eg potassium), a few vitamins (eg folate in beer) and bioactive substances (flavonoids). Alcoholic beverages are usually consumed with foods, either as part of meals or accompanied by snack foods.
The most recent apparent consumption figures for various alcoholic drinks in Australia show a per capita consumption of:

- 94.7 litres for beer;
- 17.3 litres for wines;
- 1.7 litres for fortified wines
- 1.4 litres for spirits (27)

The total pure alcohol (ethanol) per head of total population per year, was 7.6 litres.

### Table 2  Means and percentiles of intake of alcohol (% Energy as alcohol, g alcohol and Numbers of standard drinks) in adults 19+ years from the National Nutrition Survey 1995/6 for all subjects or only those consuming alcohol on the day of the survey

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<tr>
<td>N standard drinks</td>
<td>0.4</td>
<td>0.7</td>
<td>1.1</td>
<td>1.4</td>
<td>1.8</td>
<td>2.2</td>
<td>2.7</td>
<td>3.5</td>
<td>4.4</td>
<td>5.9</td>
<td>8.2</td>
<td>3.0</td>
</tr>
</tbody>
</table>

In 1998 (27):

- 59% of males and 39% of females (older than 14) drank alcoholic beverages at least once a week;
- of current drinkers 14% of men, 6% of women drank every day; and
- 13% of men and women drank four to six days a week;
- 36% of men and 62% of women reported drinking 1-2 glasses of alcohol at a time;
- 31% of men and 22% of women reported drinking 3-4 glasses at a time; and
- 33% of men and 15% of women stated they drank 5 or more glasses at a time.

**RECOMMENDED INTAKES**

Most recommendations about alcohol consumption are made on the basis of “standard” drinks. A standard drink in Australia is considered to contain 10g alcohol (equivalent to 12.5ml of alcohol). The alcohol concentration of drinks is printed on the label as percentage by volume.
Table 3 below shows how various drinks equate to the “standard” 10gm drink and Table 4 shows the energy and alcohol contents of a number of common drinks. It is of interest to note that many of these common “serves” of alcoholic drinks contain more than the “standard” 10gm of alcohol.

Until recently, most recommendations concerning alcohol consumption in Australia and overseas (ref 4, Appendix 5) have given a single average daily maximum figure for standard drinks for men and women with the male figure generally being higher than that recommended for women. Women not only generally have a smaller body size but also some differences in how they metabolise alcohol that make them more susceptible to adverse effects at a given intake (28, 29).

Table 3  Standard drink equivalents

<table>
<thead>
<tr>
<th>BEER</th>
<th>STANDARD DRINKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular beer</td>
<td>One and a half “standard” drinks</td>
</tr>
<tr>
<td>(4.9% alc)</td>
<td>4 “standard” drinks</td>
</tr>
<tr>
<td>1 can or stubbie</td>
<td>About 36 “standard” drinks</td>
</tr>
<tr>
<td>1 jug</td>
<td></td>
</tr>
<tr>
<td>1 slab (cans or stubbies)</td>
<td></td>
</tr>
<tr>
<td>Medium light beer (3.5% alc)</td>
<td>1 “standard” drink</td>
</tr>
<tr>
<td>1 can or stubbie</td>
<td></td>
</tr>
<tr>
<td>Light beer (92.7% alc)</td>
<td>Half a “standard” drink</td>
</tr>
<tr>
<td>1 can or stubbie</td>
<td></td>
</tr>
<tr>
<td>WINE (9.5% - 13% alc)</td>
<td>One “standard” drink</td>
</tr>
<tr>
<td>100 ml glass</td>
<td>About 7-8 “standard” drinks</td>
</tr>
<tr>
<td>750 ml bottle</td>
<td>About 30-40 “standard” drinks</td>
</tr>
<tr>
<td>4-litre cask</td>
<td></td>
</tr>
<tr>
<td>SPIRITS</td>
<td></td>
</tr>
<tr>
<td>1 nip (30ml)</td>
<td>1 “standard” drink</td>
</tr>
<tr>
<td>PRE-MIXED SPIRITS (about 5% alc)</td>
<td>One and a half “standard” drinks</td>
</tr>
<tr>
<td>1 can (375ml)</td>
<td></td>
</tr>
</tbody>
</table>
## Table 4  Energy and alcohol content of common alcoholic drink “serves”

<table>
<thead>
<tr>
<th>Description</th>
<th>Serve size</th>
<th>Energy (kJ)/serve</th>
<th>Alcohol (g/serve) (using NNS95/6 data)</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beer, regular (4.9% alcohol)</td>
<td>1 can/stubby (375 ml)</td>
<td>568</td>
<td>15.4</td>
<td>Highest alcohol beer in NNS95 is 4.1%. However, most regular beers appear to be around 4.5%</td>
</tr>
<tr>
<td>Beer, medium-light (3.5% alcohol)</td>
<td>1 can/stubby (375 ml)</td>
<td>546</td>
<td>14.0</td>
<td>Used NNS95 “beer, bitter or draught” (3.7% alcohol)</td>
</tr>
<tr>
<td>Beer, light (2.7% alcohol)</td>
<td>1 can/stubby (375 ml)</td>
<td>395</td>
<td>8.0</td>
<td>NNS95 gives 2.1% for light beer. However Coopers light=2.9%; Lion Nathan defines “light” as 0.9-3.3% alcohol</td>
</tr>
<tr>
<td>Wine (9.5%-13.0% alcohol)</td>
<td>1 glass (100 ml)</td>
<td>227</td>
<td>9.5</td>
<td>NNS: Ave of red wine &amp; dry white wine</td>
</tr>
<tr>
<td>Spirits</td>
<td>1 nip (30 ml)</td>
<td>255</td>
<td>8.8</td>
<td>NNS95: used “spirits, type not stated”</td>
</tr>
<tr>
<td>Pre-mixed spirits</td>
<td>1 can (375 ml)</td>
<td>1182</td>
<td>13.9</td>
<td>NNS95: avg of 7 pre-mixed canned spirit drinks</td>
</tr>
</tbody>
</table>

During the process of absorption, some alcohol is metabolised by gastric alcohol dehydrogenase. This “first pass metabolism” can be demonstrated by the lower blood alcohol after oral intake than after intravenous infusion. First pass metabolism is less active in women (28). Once absorbed, alcohol is distributed throughout the total body water. This volume (in litres) of distribution in women is 65 to 70% of that in men because on average women’s weight is 80% of men’s and women contain a higher percentage of body weight as fat so a smaller % of water. Thus after a drink, women have a somewhat higher concentration of alcohol in a smaller water volume to be metabolised mostly by the liver, and in women the liver is smaller, hence alcohol dehydrogenase capacity is less than in men.

The same level of alcohol intake for women is therefore approximately half what it is for men, though men and women vary considerable in size and in individual’s ability to metabolise alcohol. Women develop cirrhosis of the liver after long-term consumption of smaller amounts of alcohol than men.

In the latest Australian Alcohol Consumption Guidelines (4) a single recommendation for each gender has been replaced with recommendations for both maximum average daily intake as well as a maximum for an “occasion” in recognition of the potential acute harm effects of what is commonly termed, “binge” drinking.

The Australian Alcohol Consumption Guidelines categorised patterns of drinking into a number of risk categories based on average daily or weekly consumption limits and on “occasional day” limits.

Using this system, men were considered to be at “low risk” of longterm harm if their daily average intake did not exceed 4 drinks a day (up to 28 a week) and women, if it did not exceed 2 drinks a day on average (up to 14 a week). An average of 5-6 drinks a day for men (or 29-42 per week) or 3-4 a day for women (or 15-28 per week) was considered
“risky” for long term health and a daily average of 7 or more for men (or 43 a week or more) and 5 or more for women (or 29 a week or more) was considered high risk.

In terms of short term harm, an intake on any one day of up to 6 standard drinks for men or 4 for women was considered “low risk”, 7-10 for men and 5-6 for women, “risky” and 11 or more for men and 7 or more for women to be “high risk”.

For someone to be considered overall at “low risk” for both short term and long term harm they had to be within both the occasional day limits and usual weekly limits.

Most dietary guidelines have recommendations of no more than 2-3 standard drinks a day for men and one to two for women, considerably less than the “low risk” category of the Australian Alcohol Consumption Guidelines (ref 4 Appendix 5). This, in part, reflects a concern with the issue of nutrient displacement, the energy contributed by alcoholic drinks (see Table 4) and the current concern with overweight and obesity rather than links to selected chronic disease states.

The Dietary guidelines for Americans 2000 (30) recommend “If you drink alcohol beverages, do so in moderation”. Moderation is defined as no more than two drinks per day in men and one drink per day in women. As noted above, this limit is based on differences between the sexes in both weight and metabolism.

In Canada, the nutrition recommendation (1990) is that adults consuming alcohol should limit their intake to less than 5% of total energy or two drinks per day, whichever is less. In the UK (31) a 1990 set of dietary guidelines states “Men and women have different metabolisms. It is therefore recommended that men drink less than 21 units a week and women less than 14. During pregnancy it is best to avoid alcohol completely”.

In Australia, no more than four standard serves a day for men and two for women has been the standard recommendation. This is based on a consideration of the social and medical correlates of alcohol consumption but the issue of dietary energy is also relevant especially in the context of dietary guidelines.

Table 5 below shows the contribution that 4 standard alcohol drinks, in various forms, would make to the overall average energy intake of men and how much 2 standard drinks would contribute to overall energy intake in females. It should be noted that a can or stubby of the regular and medium-light beers and a can of pre-mixed spirits contain 14-15g alcohol not the “standard” 10gm but they are common serve units and as such are included in the Table.

If men chose to consume 4 standard drinks of beer (250mls - enough to provide 10g alcohol), this would account for some 13-14% of average male energy intake as assessed by the 1995/6 National Nutrition Survey. If they consumed 4 cans or stubbies (common “serves”) this would contribute as high as 20% of overall energy intake. Because of the high sugar and alcohol content, four standard serves of premixed spirits would provide some 29% of dietary energy in men. Four standard serves of wine or sprits would account for 8-9% of average energy intake.

For women, two standard drinks a day would provide about 6-10% of dietary energy unless taken as premixed spirits (just over 20% for two “standard” serves).
With increasing obesity and overweight in the Australian community, and with marginal intakes of micronutrients in some groups such as young women. It would be prudent, in the context of dietary guidelines for optimal health, to recommend a limit of 2 standard drinks a day for men and one for women. It is of interest to note that this is also the level at which cardiovascular risk protection is seen in population studies.

Alcohol, together with refined or added sugars contributes what are often termed “empty kilojoules” to the diet. They provide energy without substantial amounts of other essential nutrients. An analysis of the 1995/6 National Nutrition Survey data (Table 6) showed that alcohol and added sugar contributed, on average, some 15.5% of the energy in men’s diets and 12.5% in women’s diets but for 20% of the population, alcohol and refined sugars together contributed about one fifth of dietary energy on the day of the survey. If these “empty” kilojoules are consumed on top of normal energy requirements, overweight and obesity is a likely outcome over time. If consumed instead of foods or drinks which also supply essential nutrients, it could, over time, lead to deficiency of key nutrients.

Table 5  The contribution that four or two common alcoholic drinks could make to the energy intake of average male or female diet

<table>
<thead>
<tr>
<th>Description of drink</th>
<th>Serve size</th>
<th>Energy (kj) per 4 drinks</th>
<th>% energy provided by 4 drinks to average male diet*</th>
<th>Energy (kj) per 2 drinks</th>
<th>% energy provided by 2 drinks to average female diet*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beer, regular (4.9% alcohol)</td>
<td>1 can/stubby (375 ml)</td>
<td>2272</td>
<td>20.6 %</td>
<td>1136</td>
<td>15.2 %</td>
</tr>
<tr>
<td></td>
<td>1 glass* (250ml)</td>
<td>1514</td>
<td>13.7 %</td>
<td>757</td>
<td>10.1 %</td>
</tr>
<tr>
<td>Beer, medium-light (3.5% alcohol)</td>
<td>1 can/stubby (375 ml)</td>
<td>2184</td>
<td>19.8 %</td>
<td>1092</td>
<td>14.6 %</td>
</tr>
<tr>
<td></td>
<td>1 glass * (250ml)</td>
<td>1454</td>
<td>13.2 %</td>
<td>727</td>
<td>9.7 %</td>
</tr>
<tr>
<td>Beer, light * (2.7% alcohol)</td>
<td>1 can/stubby (375 ml)</td>
<td>1580</td>
<td>14.3 %</td>
<td>790</td>
<td>10.6 %</td>
</tr>
<tr>
<td>Wine *(9.5%-13.0% alcohol)</td>
<td>1 glass (100 ml)</td>
<td>908</td>
<td>8.2 %</td>
<td>454</td>
<td>6.1 %</td>
</tr>
<tr>
<td>Spirits *</td>
<td>1 nip (30 ml)</td>
<td>1020</td>
<td>9.2 %</td>
<td>510</td>
<td>6.8 %</td>
</tr>
<tr>
<td>Pre-mixed spirits</td>
<td>1 can (375 ml)</td>
<td>4728</td>
<td>43 %</td>
<td>2364</td>
<td>32 %</td>
</tr>
<tr>
<td></td>
<td>1 glass * (250ml)</td>
<td>3151</td>
<td>29 %</td>
<td>1576</td>
<td>21 %</td>
</tr>
</tbody>
</table>

standard alcohol serve (about 10g alcohol)
* using average energy intakes from 1995/6 National Nutrition survey of 11050 kj for males and 7480 kj for females
Table 6  Percentiles of energy from alcohol plus added sugar (“empty kilojoules”) in adults aged 19+ yrs from the National Nutrition Survey 1995/6

<table>
<thead>
<tr>
<th></th>
<th>5th</th>
<th>10th</th>
<th>20th</th>
<th>30th</th>
<th>40th</th>
<th>50th</th>
<th>60th</th>
<th>70th</th>
<th>80th</th>
<th>90th</th>
<th>95th</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>All males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Energy</td>
<td>2.3</td>
<td>4.1</td>
<td>7.0</td>
<td>9.6</td>
<td>11.8</td>
<td>13.9</td>
<td>16.5</td>
<td>19.2</td>
<td>23.0</td>
<td>28.1</td>
<td>33.6</td>
<td>15.5</td>
</tr>
<tr>
<td>Energy (kJ)</td>
<td>178</td>
<td>343</td>
<td>663</td>
<td>952</td>
<td>1205</td>
<td>1453</td>
<td>1742</td>
<td>2093</td>
<td>2586</td>
<td>3477</td>
<td>4411</td>
<td>1748</td>
</tr>
<tr>
<td>All females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Energy</td>
<td>0.9</td>
<td>2.2</td>
<td>4.4</td>
<td>6.3</td>
<td>8.2</td>
<td>10.3</td>
<td>12.8</td>
<td>15.5</td>
<td>18.8</td>
<td>24.3</td>
<td>30.0</td>
<td>12.3</td>
</tr>
<tr>
<td>Energy (kJ)</td>
<td>44</td>
<td>119</td>
<td>264</td>
<td>406</td>
<td>558</td>
<td>726</td>
<td>935</td>
<td>1176</td>
<td>1499</td>
<td>2045</td>
<td>2660</td>
<td>963</td>
</tr>
</tbody>
</table>

SPECIAL GROUPS (4)

The recommendations above relate to healthy members of the general population. Within the general population there are however, those for whom recommendations would be much lower or for whom alcohol consumption would not be recommended at all.

Children and adolescents

The accompanying publication of the Dietary Guidelines for Children and Adolescents (DGCA) explains the scientific basis of the social convention that children should drink no alcohol (or occasionally very little). DGCA also outlines the dangers of alcohol consumption by adolescents, who are more susceptible than adults, have a lower tolerance and in whom relatively small quantities can impair judgement and control.

Previous alcoholics

People who have been addicted to excess alcohol consumption are least likely to start the cycle again if they avoid any alcoholic drink entirely.

Pregnancy or planned pregnancy

The malformations and mental deficiency of the fetal alcohol syndrome (FAS) occur in women who drink heavily during pregnancy. The damage is thought to occur during the 4 to 10 weeks after conception. FAS is not seen in women who have one drink a day (29) but some authorities recommend total abstinence. Most women do restrict alcohol intake once they receive a positive pregnancy test but women who might be pregnant or are trying to become pregnant should avoid drinking until more research has proved it to be completely safe.

Drivers and machine operators

All drivers and operators of dangerous machinery are liable to random breath testing and in the event of an accident their blood alcohol concentration will be measured, either directly or via breath testing. They must drink nothing or little in the hour before they drive or operate machinery and not at all while driving. A standard drink contains about 10g of alcohol. After it is absorbed alcohol is distributed throughout the total body water.
If the total body water is 40 L (or kg) the concentration after absorption of 10 g alcohol will be 10/40,000 or 0.025g/100 ml (half the legal driving limit) or somewhat less because of first pass metabolism, and 0.050 g/100ml after 2 standard drinks. However, in a woman with total body water 34 L the concentration will be 10/34,000 = 0.029 g/100 ml after 1 drink and 0.058 g/100 ml after 2 drinks. On average, people can metabolise 100 mg of ethanol per kg body weight hour (5-8 g/hr). The rate of metabolism varies about two-fold between individuals. Alcohol absorption can be slowed by having a meal in the stomach, but there is no agent that increases the rate of alcohol metabolism.

**People taking certain prescribed or non-prescribed drugs**

People taking some analgesics, antidepressants, some antihistaminics, some antipsychotics can have enhanced sedative effects if they drink. There are other side effects of drug interactions with alcohol. All these are listed in the information about proprietary drugs issued to doctors and pharmacists and in prescribers manuals. Responsibility for providing information to the individual is primarily the prescribing doctor.

**Certain cultural groups**

In response to severe problems related to alcohol in many of their communities, Aboriginal and Torres Straight Islanders have established initiatives to encourage non-harmful use, limit access to alcohol and establish “dry” areas and communities. These are only a small part of the major efforts by Aboriginal and Torres Straight Islanders to improve their health status and their social and economic circumstances.

There is limited evidence about the patterns of drinking in migrant communities in Australia but there are some known physiological constraints on alcohol use in some migrant groups. The flushing response (13) is an inherited characteristic seen most often in people of Asian descent. The flushing is caused by a slower metabolism of alcohol which also results in other side effects as well as reduced tolerance. In the flushing response there is a build up of acetaldehyde which can cause nausea and vomiting and many people with this response choose not to drink at all.

**CONCLUSION**

Because of its effect on both short term and long term health and social outcomes and the additional kilojoules it provides in the diet, it is recommended that adults, if they drink at all, take care to limit their average daily intake of alcohol to no more than two standard drinks a day for men and one for women.

**EVIDENCE**

There has been a recent and extensive review undertaken by NHMRC assessing alcohol consumption in the Australian context which involved a detailed review if the literature along the NHMRC guidelines (ref 4). This detailed evidence is available and is not repeated here.

The background paper includes summary evidence from this review in relation to alcohol consumption and chronic disease outcome but also references selected Level III evidence...
in relation to alcohol consumption and blood pressure (ref 7), alcohol limitation and blood lipids (ref 8), alcohol and cancer (ref 9), alcohol and breast cancer (ref 10), alcohol and liver disease (ref 11,15,29), moderate alcohol intake and protection from heart disease and lowering of heart disease risk factors (ref 18, 19, 20).
REFERENCES

4. NHMRC Alcohol: Australian Guidelines for Consumption Canberra NHMRC, 2001
6. Developing National Priorities for Alcohol Research. (report of a meeting in Adelaide May 2001) Canberra; Commonwealth Dept Health and Aged Care, 2001
CONSUME ONLY MODERATE AMOUNTS OF SUGARS AND FOODS CONTAINING ADDED SUGARS

INTRODUCTION

Many of the foods found in the Australian diet contain naturally occurring sugars. In other foods sugars, particularly sucrose, may be added to food products during processing, to increase their palatability and acceptability and sometimes to add bulk. Sugars provide a readily absorbed source of energy and have an important role in the palatability of food as sweeteners and flavour enhancers. However the presence of significant amounts of sugar dilutes the nutrient density of the diet and diets high in added sugar are associated with obesity and dental caries, particularly in children.

Because sugars are a significant source of energy in the Australian diet, all previous sets of dietary guidelines, for Adults, Children and Adolescents, and Older Australians have included a guideline on sugar or sugars. Over the years the emphasis has changed, from a guideline aimed at reducing the amount eaten, to one which emphasises care and moderation in the amount consumed. This reflects the changing scientific knowledge and the relative stability of sugar consumption in Australia. The USA year 2000 guidelines include in the section “eat sensibly” the guideline “choose beverages and foods that limit your intake of sugars” (1). The pros and cons of a sugar dietary guideline have been debated in the literature by Williams (2) and Stanton (3).

DEFINITIONS

Carbohydrates are polyhydroxy aldehydes, ketones, alcohols, acids, their simple derivatives and their polymers having linkages of the acetal type. They may be classified according to their degree of polymerization and may be divided initially into three principal groups, namely sugars, oligosaccharides and polysaccharides (Table 1).

Table 1  The major dietary carbohydrates

<table>
<thead>
<tr>
<th>Class (DP*)</th>
<th>Sub-Group</th>
<th>Components</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sugars (1-2)</td>
<td>Monosaccharides</td>
<td>Glucose, galactose, fructose</td>
</tr>
<tr>
<td></td>
<td>Disaccharides</td>
<td>Sucrose, lactose, trehalose</td>
</tr>
<tr>
<td></td>
<td>Polysols</td>
<td>Sorbitol, mannitol</td>
</tr>
<tr>
<td>Oligosaccharides (3-9)</td>
<td>Malto-oligosaccharides</td>
<td>Maltodextrins</td>
</tr>
<tr>
<td></td>
<td>Other oligosaccharides</td>
<td>Raffinose, stachyose, fructo-oligosaccharides</td>
</tr>
<tr>
<td>Polysaccharides (&gt;9)</td>
<td>Starch</td>
<td>Amylose, amylpectin,modified starches</td>
</tr>
<tr>
<td></td>
<td>Non-starch polysaccharides</td>
<td>Cellulose, hemicellulose, pectins, hydrocolloids</td>
</tr>
</tbody>
</table>

DP* = Degree of polymerization
Source: (4)
Sugars
The term “sugars” is conventionally used to describe the mono and disaccharides including sucrose, glucose and fructose.

“Sugar”, by contrast, is used to describe purified sucrose as are the terms “refined sugar” and “added sugar”.

Extrinsic and Intrinsic Sugars
Intrinsic sugars were defined as sugars occurring within the cell walls of plants, ie. naturally occurring, while extrinsic sugars were those which were usually added to foods. Because lactose in milk is also an extrinsic sugar, an additional phrase “non-milk extrinsic sugars” was developed. These terms have not gained wide acceptance and there are no current plans to measure these sugars separately in the diet nor to incorporate their use into food tables (4).

The terms ‘refined’, ‘added’ and ‘extrinsic’ sugars are sometimes used to denote sucrose and glucose used in the food industry and at home. Physiologically, there is no difference between the sugars that occur naturally in food and the refined sugars that are added to the diet. Among foods rich in added sugars are confectionery, cakes, pastries, biscuits, fruit drinks, cordials and carbonated soft drinks. Foods with high-added sugar content often have a lower nutrient content but are energy dense.

The term “no added sugar” means no sugars have been added during the foods manufacture. It does not mean that no sugar is present, since most foods have sugars present in some form.

BACKGROUND

Sugars in the Australian Diet

Australian adults derive about 45% of their energy from carbohydrates and about one half of this is from sugars. The results of the 1995 National Nutrition Survey (NNS) show that the percentage of energy from total sugars intake declines from ages 2–3 years to 45–64 years, followed by a slight increase in intake in the 65 years and over age group (see Table 2) (5).

| Carbohydrates: mean contribution to energy intake, 1995 Percentage |
|-----------------|--------|--------|--------|--------|--------|--------|--------|--------|--------|
| Carbohydrate    | 52.1   | 52.7   | 50.9   | 52.1   | 49.6   | 46.9   | 45.0   | 44.1   | 45.8   |
| Total sugars    | 29     | 28     | 25     | 26     | 25     | 22     | 19     | 19     | 21     |
| Added sugars    | 14     | 15     | 15     | 15     | 13     | 10     | 9      | 9      | 9      |
| Natural sugars  | 16     | 13     | 10     | 10     | 10     | 9      | 9      | 11     | 12     |

Source: (5)

- Added sugars are at a similar level from ages 2-3 years to 19-24 years but are lower from 25 years and over.
- Natural sugars were highest in very young children and adults over 65 years and lowest in 19-44 year age group.
The most recent source of information on sugars (only sucrose) consumption is from the 1998-99 Apparent Consumption of Foodstuffs (6). Apparent consumption has fallen by about 15% from pre-war levels or by 23% from the post-WWII peak reached in 1948 (see Table 3). The apparent consumption data do not represent actual consumption by individuals or population groups, since some sugar is wasted, and some is used for brewing and other purposes, but they do give an indication of the trends in consumption. Baghurst et al. (7) discuss the differences between apparent consumption of sugar and actual dietary consumption.

Using a different methodology (food frequency questionnaires), the CSIRO has also shown that between 1988 and 1993 the intake of refined sugars decreased slightly for all age groups. However by 1999 intakes of refined sugars had returned to 1988 levels in all adult age groups.(8) Honey, which is a solution of sugars, has also declined in consumption in recent years and is now 0.5 kg per capita (6).

**Table 3  Apparent Per Capita Consumption of sugar (per year)**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cane sugar as</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>refined sugar (kg)</td>
<td>32.0</td>
<td>31.2</td>
<td>27.0</td>
<td>21.0</td>
<td>14.9</td>
<td>8.8</td>
<td>n.a</td>
</tr>
<tr>
<td>Cane sugar in</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>manufactured foods (kg)</td>
<td>16.3</td>
<td>23.1</td>
<td>23.6</td>
<td>27.7</td>
<td>34.6</td>
<td>33.9</td>
<td>n.a</td>
</tr>
<tr>
<td>Cane sugar total (kg)</td>
<td>48.3</td>
<td>54.3</td>
<td>50.6</td>
<td>48.7</td>
<td>49.5</td>
<td>42.7</td>
<td>37.6</td>
</tr>
<tr>
<td>Total sugars (kg)</td>
<td>50.8</td>
<td>56.8</td>
<td>53.0</td>
<td>51.9</td>
<td>54.5</td>
<td>48.3</td>
<td>43.4</td>
</tr>
</tbody>
</table>

Source: (6)

In the 1930’s, 60% of sugar used in Australia was in the form of added sugar. Now the proportions are reversed: 73% of sugar is used in food processing.

In remote Aboriginal Communities the apparent consumption of sugar is much higher than the Australian average, as shown in the following Table Four. This study shows that sugar consumption is high, whereas fruit and vegetable consumption is well below the Australian average. In the communities where apparent consumption was measured, refined sugars contributed approximately 30% of total energy intake. 60% of the apparent high intake of sugars was derived from white sugar per se which is in marked contrast to recent figures for the wider Australian community. Unfortunately no data is available for urban indigenous communities.
Table 4  Apparent mean consumption of selected foods in Aboriginal Communities compared with national data (kg per capita per year)

<table>
<thead>
<tr>
<th>Food</th>
<th>Central Desert (n=3)</th>
<th>Northern Coastal (n=3)</th>
<th>Australian data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flour (white)</td>
<td>37.6</td>
<td>44.4</td>
<td>NA</td>
</tr>
<tr>
<td>Bread (all)</td>
<td>34.1</td>
<td>30.5</td>
<td>45.5</td>
</tr>
<tr>
<td>Beef and veal</td>
<td>51.6</td>
<td>25.8</td>
<td>41.4</td>
</tr>
<tr>
<td>Poultry</td>
<td>22.3</td>
<td>19.7</td>
<td>23</td>
</tr>
<tr>
<td>Lamb</td>
<td>22.8</td>
<td>3.3</td>
<td>16.8</td>
</tr>
<tr>
<td>Fish</td>
<td>0</td>
<td>4.8</td>
<td>4</td>
</tr>
<tr>
<td>Fruits</td>
<td>33.2</td>
<td>17.6</td>
<td>106.9</td>
</tr>
<tr>
<td>Vegetables</td>
<td>24.3</td>
<td>19.6</td>
<td>136.2</td>
</tr>
<tr>
<td>Sugar (refined)</td>
<td>54.1</td>
<td>50.3</td>
<td>8.2</td>
</tr>
<tr>
<td>Carbonated beverages</td>
<td>67.9</td>
<td>224.6</td>
<td>73</td>
</tr>
<tr>
<td>Fruit juice</td>
<td>48.3</td>
<td>12.8</td>
<td>NA</td>
</tr>
</tbody>
</table>

Bread includes flour used in bread making.

NA = not available

Source: (9) (10)

Intake of Sugars in relation to the total diet

There are two important nutrition issues relating to the consumption of sugar in the diet. The first relates to other nutrients that might be associated with sugars, and much has been written about a fat-sugar relationship. The second relates to nutrient density. If sugar (sucrose) provides around 10%-15% of energy in the diet, is the remainder of the diet sufficiently nutrient dense to provide all necessary nutrients?

A number of authors have suggested that high extrinsic sugar consumption is associated with high dietary fat intakes (11). However studies from Europe and Australia suggest that while it is possible to identify some foods rich in both fat and sugars, in the context of the whole diet, foods that are the primary sources of sugars are only minor sources of fat and vice versa (12). Studies of the relationship between a low-fat diet and refined sugar intake often show an inverse relationship (12) (13) (14) (15). One study, of 3,290 people living in Victoria and South Australia, found that respondents who had the lowest relative intake of fats had high intakes of simple sugars, both natural and refined (16). In the NNS only data on “total sugars” is available and this does not show a consistent relationship between sugars and fat intake (5). These results have led some authors to suggest that it may not be practical to give advice to reduce fat and sugar simultaneously (17).

Some studies, particularly the larger studies from the USA, suggest that high intakes of sugar are linked to diet quality. Using food intake data from a representative sample of 15,011 persons, Britten et al (18) divided the sample into quartiles based on added sugar consumption. Many high consumers of sugar also overconsumed total energy. The intake of fruit in this group was lower than others with similar energy intakes. The 41% of sugar overconsumers who did not consume excessive energy, compensated for the additional...
energy by reducing intakes of other foods, including the fruit, vegetable, milk and grains groups. Similarly an analysis of the Third National Health and Nutrition Examination Survey (n=15,611, aged 20 or over) showed that energy-dense, nutrient-poor foods tended to be consumed at the expense of those that are nutrient-dense. These foods included foods high in fat and/or sugar such as soft drinks, confectionery, biscuits, cakes, desserts, pastries and processed savoury snacks. A recent review by Williams summarising a number of studies from the United States and United Kingdom noted that in most cases energy and nutrient intakes were positively related to total sugar intake (48, 49,50,51,52).

The United Kingdom Committee of Medical Aspects of Food report concluded:
- on average people with high total energy intakes eat more of all nutrients including sugar; and
- sugar intake is a weaker predictor of absolute micronutrient intake than total energy consumption.(53)

The association between high-refined sugar intake and low micronutrient intake was investigated by re-examining data from three large-scale Australian population surveys of dietary intake (20) and from the CSIRO sugars analysis of the 1995-96 NNS. The results of this review did not show a consistent relationship between refined sugar consumption and micronutrient intake. A study of older South Africans (21) showed that as sugar intake increased there was a significant decrease in the proportion of energy derived from fat. But a negative aspect of the increasing sugar intake was evidence of nutrient dilution: with the exception of folate and Vitamin B12, more than one-fifth of the subjects failed to consume 67% of the recommended daily allowance for several vitamins and minerals. This sub-optimal nutrient intake can be explained by the intake of cakes, puddings, tarts, meat pies, snacks, soups, sauces and cool drinks contributing to overall energy intake. (It should be noted however that this study was in older people. Its relevance to younger adults and children and adolescents is not clear.) Results from several other studies vary depending on the classification of different sugars used but in general moderate sugar consumers appear to have the most adequate diet.(50, 54,55)

The National Nutrition Survey shows that an increasing proportion of energy is obtained from meals and snacks eaten outside the home (5). It is likely that these have a higher sugar and fat content than the rest of the diet. The study by Summerbell et al (22) found that 25% of adolescents and 20% of adults’ daily energy intake was in the form of snacks and that the proportion of energy derived from total sugars in snacks was greater than that in meals. Most often this sugar was provided by plain biscuits, milk and sugar added to cups of tea and coffee.

If energy balance is to be maintained (or approximately maintained), foods of high-energy content and lower nutrient density, must be replacing other food groups. When more foods of low nutritional worth (lower nutrient density) are consumed, either less of the foods that supply essential nutrients will be consumed or total kilojoule intake will rise.

To summarise this section, it is likely that the results of surveys reported as averages of group consumption, obscure the effects of those who are in the upper percentiles of sugar consumers. It is important that care is taken with snack foods that are high in added sugars, since foods that are high in refined sugars (such as soft drinks and confectionery) are energy dense but do not provide vital nutrients. Foods such as cakes, biscuits and
sweets are high in both sugar and fat and are also energy dense: they provide few nutrients and are often eaten instead of more nutritious, necessary foods.

**SCIENTIFIC BASIS**

Carbohydrates provide the largest source of energy in the diets of most people - on average around 45% of the energy in the Australian diet. Dietary carbohydrates are usually associated in foods with important micronutrients and phytochemicals. Diets high in carbohydrate are not associated with the development of obesity (23). People whose diets are high in carbohydrates usually have a lower prevalence of obesity, heart disease, non-insulin dependent diabetes mellitus, and some forms of cancer (24).

Among carbohydrate’s physiological functions are the following (23):

- provision of energy;
- effects on satiety and gastric emptying;
- effects on blood glucose and insulin metabolism;
- protein glycosylation;
- bile acid dehydroxylation;
- fermentation - production of hydrogen and methane;
- production of short-chain fatty acids;
- control of colonic epithelial cell function;
- bowel habit, laxation and motor activity; and
- effects on large bowel microflora.

Epidemiological and clinical studies help to give us an understanding of the role of carbohydrates in the aetiology of disease. Few of these studies suggest a direct causal link between carbohydrate consumption and disease.

**Obesity**

The 1995 National Nutrition Survey showed that obesity is an increasing problem for all age groups in Australia (5). This epidemic is described by the WHO as part of an “escalating epidemic of overweight and obesity that is affecting many countries in the world” and “The principal causes of the accelerating obesity problem worldwide are sedentary lifestyles and high-fat, energy-dense diets” (http://www.who.int/nut/obs.htm). It has been suggested that an excess consumption of sugar contributes to an energy dense diet that may lead to energy imbalance and obesity. In a randomised controlled trial of diets and weight reduction (25), subjects were randomised into groups with diets which included reduced fat and high simple carbohydrate and reduced fat and high complex carbohydrate. The study showed that a reduction in fat intake resulted in a modest, but significant weight loss. Whether the carbohydrate was in simple or complex form, made no difference to weight outcomes or to the lipid profiles of the subjects.

It is, however, important to stress that excess energy in any form will promote the accumulation of excess body fat and that high-carbohydrate diets should be promoted only in accordance with an individual’s energy needs (23).

In a longitudinal study over two years of 548 ethnically diverse schoolchildren (age 11·7 years), Ludwig et al (26) found that an increase in sweetened soft drink consumption increased the risk of increasing BMI and of becoming obese. On the other hand, a number of studies have concluded that intake of carbohydrate or even sucrose has no relationship
with obesity, or that the relationship may even be negative (27). Children and adults, who ingest large amounts of carbohydrate, sucrose, or both, have been reported to be leaner than their peers. However the high intake of carbohydrate in the respondents in these studies might reflect higher levels of physical activity. Another reason for the lack of relation between CHO intake and adiposity noted above, might be inaccuracy in assessing intake and expenditure using traditional dietary methods. The "doubly-labelled water method" used for measuring energy expenditure in free-living individuals, has recently cast doubt on the validity of self-reported food intake for adults (refer to Bellisle 2001- in press), although dietary data do appear to be more valid for children. Yet another reason could be that very active children need and ingest more sugar (28).

The study by Ludwig et al (26) shows that the consumption of sugar-sweetened beverages is an independent risk factor for obesity in children aged 11-12 years. This may be related to the reduced effect of sugar in a liquid medium on satiety. They (Ludwig and colleagues) suggest, that compensation at subsequent meals for energy consumed in the form of a liquid is less complete than for energy consumed in solid form (29). The Ludwig study also showed that the consumption of diet soft drinks was inversely associated with becoming obese.

In Australia the consumption of beverages, most of which are sweetened, is increasing. Carbonated and aerated beverages have become the most popular beverage. The consumption of carbonated and aerated beverages has continued to increase from the late 1980s figure of 87.4 litres consumed per capita to 113.0 litres per capita in 1998-99. This is an increase of 30% in a decade and an increase in 3.7% over the previous year (6). The per capita trend in soft drink consumption is shown in Figure One. By the end of 2000, 19.3% of soft drinks consumed in Australia were sweetened with non-nutritive sweeteners.

**Figure 1**

![Per Capita Softdrink Consumption](image)

*Source: Australasian Softdrink Association, 2001 (official statistics 2001)*

Sugar sweetened drinks make up the major portion of the soft drink market. In the National Nutrition Survey, the consumption of soft drinks in the 16-18 year and 19-24 year age groups was just over 400 grams per day (See Figure One). About 7% of the reported intake was non-sugar containing drinks.
Historically the prevalence of dental caries has increased in centuries where the diet has changed to include more sugars and other refined foods. The relationship between sugar (sucrose) and dental caries was first documented by Miller in the scientific literature in 1883 and confirmed in numerous studies since that time (30). Dental caries remains a significant public health problem in Australia, and is estimated to be the most expensive diet related health problem in Australia (31). However there have been dramatic declines in average levels of dental decay, as defined by the number of decayed, missing and filled teeth (DMFT). In 12-year-old children DMFT scores fell from approximately 8 in 1965 to 1.01 in 1995. These improvements are obviously the starting point for future improvements in oral health in later life, but even in adults the average number of missing teeth has fallen from 8.3 in 1973 to only 3.6 in 1995 (32). The role of fluoridation in dental caries prevention has been documented (33) (34).

The improvement in the dental health of children does not extend to aboriginal children or to those from the lowest socio-economic groups. Historically, Aboriginal groups have had substantially fewer dental caries than non-Aboriginal peoples. More recently, however, this trend appears to have been reversed, with improvements in the oral health of non-Aboriginal children and a deterioration in that of Aboriginal children. (35)

Dental caries can be defined as a dietary carbohydrate and salivary-modified infectious disease. The key microbiological feature of dental caries is a dietary-carbohydrate enrichment of the dental plaque microflora with bacteria such as Streptococcus sobrinus and Streptococcus mutans which increases the acid producing potential of dental plaque (36).
The development of dental caries is a dynamic process involving the metabolism of a carbohydrate substrate by oral bacteria to produce acid, with saliva and host resistance offering protective elements (37). Streptococcus mutans can ferment sugars to lactic acid. Dietary sugars other than sucrose, for example glucose and lactose, can also induce caries formation. However these sugars are less cariogenic than sucrose, because in addition to being converted to acid metabolites, sucrose is uniquely utilised for extracellular polysaccharide synthesis. Starch is less cariogenic than other dietary sugars because it does not readily diffuse into plaque and is less readily hydrolysed. Streptococcus mutans, dietary sugars and a susceptible tooth surface are the important factors in dental caries. If there is frequent exposure to sugars, the rate of demineralisation of the tooth will exceed the rate of remineralisation and dental caries will occur (38). The duration of exposure depends on the extent of retention of sugary foods in the mouth and the number of eating occasions and can be difficult to describe and quantify (37).

Comparisons of international data indicate that low sugar consumption does not necessarily relate to less dental caries, nor that a higher consumption inevitably leads to more (37). The relationship between the quantity and frequency of sugar consumption approximates a sigmoid curve. Thus dental caries incidence rises more steeply as the consumption of sucrose increases until the curve flattens and the increase in dental caries is small with further increases in sucrose intake (39). The WHO study group (39) noted that very little caries occurs in children when the national per capita sugar (sucrose) consumption is below 10 kg per annum (approximately 30 g/day) but a steep increase may occur from 15 kg upwards. Studies have also shown that the frequency of eating sugar is related to dental caries rather than the amount of sugar per se (40). The sugars contained in the cellular structure of foods (such as the intrinsic sugars of fresh fruits and vegetables) has been found to have little cariogenic potential; it is foods high in extrinsic sugars that are most damaging to the teeth (41). Petti et al [cited in (37)] found that good oral hygiene was three times more likely to predict a low caries prevalence than a ‘low cariogenic’ diet. The principal diet and health association given for the retention of a sugar guideline in the US Dietary Guidelines continues to be dental caries (1). In severe cases dental caries can cause loss of teeth and pain that may reduce dietary intake and compromise nutritional status.

Based on the scientific evidence, advice on sugar intake for the prevention of dental caries should include the frequency of sugar intakes and not just on the amount. The FAO report (4) summarises the evidence: “The incidence of dental caries is influenced by a number of factors. Foods containing sugars or starch may be easily broken down by I-amylase and bacteria in the mouth and can produce acid which increases the risk of caries. Starches with a high glycaemic index produce more pronounced changes in plaque pH than low glycaemic index starch, especially when combined with sugars. However, the impact of these carbohydrates on caries is dependent on the type of food, frequency of consumption, degree of oral hygiene performed, availability of fluoride, salivary function, and genetic factors. Prevention programs to control and eliminate dental caries should focus on fluoridation and adequate oral hygiene, and not on sucrose intake alone.”

**Dental Caries and Infants**

Baby Bottle Tooth Decay (BBTD) is a recognised problem in infants who are pacified by giving them a bottle to suck on for long periods. Whether the bottle contains infant
formula, fruit juice or some other carbohydrate containing food does not seem to matter (42).

**Carbohydrates and the Prevention of Dental Caries**

Both xylitol and sorbitol have been shown to have a preventive effect on dental caries. The daily consumption of xylitol (5-10 g/day) added to chewing gum and confectionery foods has been shown to prevent dental caries in children (43). Highly acidogenic snack foods should be consumed at mealtimes to reduce the risk, and between-meal snacks should be either nonacidogenic (such as xylitol products) or hypoacidogenic (such as sorbitol and HSH products). Cheeses present a naturally occurring situation that may provide anticariogenic effects from the diet (44). A systematic review of published double-blind comparative trials showed that xylitol-containing gums may provide superior efficacy in reducing caries rates in high-risk populations (45). One mechanism of action of the xylitol-containing gums is the stimulation of salivary flow (38).

**Non–insulin dependent diabetes mellitus**

The rapid cultural change experienced by many populations previously consuming a traditional diet and the high incidence of centrally distributed abdominal obesity have been concurrent with high rates of non–insulin dependent diabetes mellitus in these populations. Some populations appear to have a stronger predisposition to the development of NIDDM than others, suggesting the involvement of genetic factors. Family history, diet, and lifestyle conditions that lead to obesity will influence the risk of developing NIDDM. The development of NIDDM does not appear to be related to the ingestion of sugar or other carbohydrates: it is predominantly influenced by genetics, body weight and lifestyle factors. Avoiding obesity and increasing intakes of a wide range of foods rich in non-starch polysaccharide and carbohydrate-containing foods with a low glycaemic index offers the best means of reducing the rapidly increasing rates of NIDDM in many countries (23).

**Cardiovascular disease**

Body mass index, abdominal obesity and genetic and lifestyle factors appear to be primary in the aetiology of coronary heart disease. There is some evidence that antioxidants offer protection against the development of heart disease: fruits and vegetables, which are sources of sugars and carbohydrates, are rich in antioxidants. Increasing the amount of these foods in the diet can assist in the reduction of saturated fat, which will provide further protection against heart disease. There is no evidence of a causal role for sugar in the development of cardiovascular disease. Ensuring that the diet contains adequate amounts of fruit, vegetables and carbohydrate-rich foods—at the expense of fat—and maintaining a healthy body weight are the basis of dietary advice aimed at reducing the risk of coronary heart disease (23).

**Cancer**

Although it is widely recognised that diet influences the development of cancer, a role for sugar has not been identified. Fruit, vegetables and cereal foods are considered to be protective against some forms of cancer.
Gastrointestinal diseases other than cancer

Consumption of non-starch polysaccharides and resistant starch contributes to stool weight: increasing the intake of these foods can effectively prevent constipation, haemorrhoids and anal fissures (23).

Attention-Deficit/Hyperactivity Disorder

Attention-Deficit/Hyperactivity Disorder is the most common neurobehavioural disorder in children. It is among the most prevalent chronic conditions in school-aged children. There is no evidence that sugars or sugar-containing foods are involved in the aetiology of Attention-Deficit/Hyperactivity Disorder (46).

Summary

The evidence for sugar’s role in the aetiology of dental caries is strong. Excess of energy intake over expenditure, from whatever sources, is the cause of obesity. There are special factors, which may mean that consumption of sugar-sweetened drinks is more likely to cause obesity. Sugar may also displace more nutrient dense foods from the diet. No other links to the causation of specific disease has been identified. On the other hand the moderate use of sugars as sweeteners, or to add flavour, may actually improve the quality food consumption and improve overall nutrient consumption. For most Australians, consumption of up to 15–20% of energy as sugars is compatible with a healthy diet. Consumption of greater amounts than this would lead to a decrease in nutrient density. A diet without any sugar would be impractical and is not considered further. Hence this guideline: ‘Use added sugars in moderation’.

PRACTICAL ASPECTS OF THIS GUIDELINE

Adding a small amount of refined sugar can increase the palatability of some highly nutritious foods and increase the overall nutrient intake. For example, a small amount of sugar or honey added to porridge and spreading jam on bread or toast can greatly improve the taste and acceptability of the high-carbohydrate, nutrient-dense foods. On the other hand, stewed fruit added to porridge would offer equal palatability, with less sugar. There are an increasing number of non-nutritive sweeteners available which have been promoted as substitutes for sugars. While there are still problems with heat stability, the use of non-nutritive sweeteners in carbonated beverages, could reduce the energy load in the highest consuming age groups. While some studies on obesity control have shown mixed results, at least one study has shown a long-term benefit on weight control (47).

RELATION TO OTHER GUIDELINES

Enjoy a wide variety of nutritious foods

It is important that a wide variety of foods is included in the diet and that the consumption of foods high in added sugars is kept to moderate levels.

Eat plenty of vegetables (including legumes) and fruit

Adding a small amount of sugar to stewed fruits and some cooked vegetables can increase their palatability.
Eat plenty of cereals (including breads, rice, pastas, noodles), preferably wholegrain
Cereals, breads and pastas are an excellent source of energy and nutrients. Adding small amounts of sugar to cereals and breads can greatly increase their palatability.

Drink plenty of water
Adding sugar to hot beverages is a common practice; it should be regulated if a sizeable number of drinks are consumed each day. Artificial sweeteners can be useful in providing the sweetened flavour but reducing the amount of added sugar consumed.

CONCLUSION

The inclusion of added sugar in the diet of Australians should be moderate, to ensure that valuable nutrients are not diluted by foods high in added sugar and limited in nutrient density. On the other hand, adding small amounts of sugar to foods that are energy and nutrient dense—for example, porridge with suitably sweetened stewed fruit or toast and jam—can increase the palatability of these foods and promote their intake.

EVIDENCE

Much of the evidence presented in this paper relates to dispelling commonly held beliefs about sugar and disease. Therefore much of the evidence is negative in that it disproves a hypothesised relationship. There is Level I evidence of dental caries prevention with xylitol (ref 34); and Level III evidence for the role of carbohydrates in dental caries (ref, 30, 40) and for a link between consumption of sugar-sweetened drinks and childhood obesity (ref 26).

On the other hand, a number of studies have concluded that intake of carbohydrate or even sucrose has no relationship with obesity or that the relationship may be negative (see ref 27). Other evidence concerning sugar and dental cares comes from cross-population studies and observational studies within population
REFERENCES

PREVENT WEIGHT GAIN BY KEEPING PHYSICALLY ACTIVE AND EATING ACCORDING TO YOUR NEEDS

BACKGROUND

Energy needs are influenced by genetic and environmental factors. The major direct environmental factors influencing energy balance that are under the discretionary control of an individual are physical activity and dietary energy intake. Eating more food energy on a regular basis than is needed to meet your energy requirements can lead to energy storage in the form of excess body fat.

Due to the increased use of labour-saving devices and technology, the need for physical activity has decreased. Meanwhile, for most Australians food is plentiful, palatable, energy-dense, easily accessible and heavily promoted.

Excess body fat is associated with adverse health consequences including increased mortality and is now a major public health problem in Australia. Similarly (and independently) being inactive is also associated with poorer health and increased mortality. For Australian adults, the mean level of excess body fat is increasing and the proportion of the adult population that is overweight or obese is substantial and increasing. The proportion of the adult population that is inactive also appears to be increasing.

The reduction of excess body fat is an important issue for many people and this difficult task is best dealt with at an individual treatment level. The dietary guideline, developed for the adult population, focuses on the prevention of weight gain. Concentrating efforts to manage obesity on people with existing weight problems will do little to prevent the occurrence of new cases of overweight or obesity. Weight gain is associated with additional health risk regardless of the starting body mass index, and the increase in morbidity and mortality associated with excess body fat begin at low levels of BMI.

It is not the intent of this dietary guideline to encourage inappropriate food restriction at any age, rather to encourage at least moderate intensity physical activity at all ages, and to discourage the development, or increase, of excess body fat in adult life.

In general, Australians are advised to enjoy an appropriate amount of food to meet their energy needs. For best results, they should combine healthy eating with an active lifestyle.

DEFINITIONS

Physical inactivity (sedentary behaviour) is a state when body movement is minimal and includes participation in physically passive activities such as watching television, reading, working at a computer, talking on the telephone, driving a car or meditating.
‘Sufficient’ physical activity to confer a health benefit requires regular participation in activity of ‘sufficient’ duration and intensity. There is no clear absolute threshold for health benefit, however the 1996 US Surgeon General’s report (1) provided a scientific basis for health benefits to be achieved from participation in regular moderate intensity physical activity. This has been interpreted into the current Australian guideline (2) of the accumulation of 30 minutes of moderate physical activity on most, preferably all, days of the week. This guideline is not formulated with objectives such as weight loss or prevention of weight gain in mind – it is a modest target and considered to be sufficient to result in a health benefit. Additional health benefits are expected to accrue from additional physical activity.

Body Mass Index (BMI) is the weight of an individual (measured in kilograms) divided by the square of their height (measured in metres). The BMI is used as a simple estimate of the body fatness of a human being who does not have abnormal physical characteristics. The World Health Organisation (WHO) (3), and the National Institutes of Health of the United States (4) have recommended that an operational definition of overweight be a BMI of at least 25 kg/m\(^2\), and obesity as a BMI of at least 30 kg/m\(^2\). A recent definition from the National Health and Medical Research Council of Australia (5) differs slightly in that overweight is defined as a BMI of above 25 kg/m\(^2\) and obesity as a BMI of above 30 kg/m\(^2\).

The more comprehensive classification of the WHO (3) is:

<table>
<thead>
<tr>
<th>Classification</th>
<th>Body Mass Index (BMI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>&lt;18.5</td>
</tr>
<tr>
<td>Normal range</td>
<td>18.5 – 24.9</td>
</tr>
<tr>
<td>Overweight:</td>
<td>≥ 25.0</td>
</tr>
<tr>
<td>Pre-obese</td>
<td>25.0 - 29.9</td>
</tr>
<tr>
<td>Obese class I</td>
<td>30 – 34.9</td>
</tr>
<tr>
<td>Obese class II</td>
<td>35.0 – 39.9</td>
</tr>
<tr>
<td>Obese class III</td>
<td>≥ 40.0</td>
</tr>
</tbody>
</table>

NEED FOR THIS GUIDELINE

The 1995 National Nutrition Survey (NNS) (6) indicated that 55.2% of Australians 19 years or older are overweight or obese. The percentage of males was 63.7%, and the percentage of females was 47.0%. The proportion of the population who were overweight or obese peaked at 50-54 years of age for men and 60-64 years for women. The percentage of the adult population with a BMI less than 18.5 kg/m\(^2\) was 1.4% - in the 19-24 years old group it was 5.4% in women and 2.4% in men.

To determine dietary and anthropometric change between the 1995 NNS and the 1983 NHF Risk Factor Prevalence Study, a subset of the 1995 survey was used to control for a number of important study design characteristics (included residence, age, season of measurement) that differed between the two studies. The mean BMI for men had increased from 25.5 (95% CI 25.3 to 25.6) kg/m\(^2\) in 1983 to 27.2 (95% CI 27.0 to 27.5) kg/m\(^2\) in 1995. For women, the increase in mean BMI was from 24.3 (95% CI 24.2 to 24.5) kg/m\(^2\)
in 1983 to 26.8 (95% CI 26.5 to 27.1) kg/m² in 1995. The proportion of adult women who were overweight or obese in 1995 had increased by 41% since 1983, and the proportion of adult men had increased by 29% (AFNMU personal communication).

Cost of obesity (BMI > 30 kg/m²) in Australia has been estimated at 2% of the total health budget (7) and conservatively at $840 million in 1992-3 (5). A recent estimate for the United States has suggested that the direct and indirect costs of obesity amount to 10% of the national health care budget (8).

Energy intake for males had increased from 10,824 (95% CI 10,685 to 10,963) kJ in 1983 to 11,195 (95% CI 10,956 to 11,434) kJ in 1995, and for females from 7,299 (95% CI 7,204 to 7,395) kJ in 1983 to 7,624 (95% CI 7,464 to 7,785) kJ in 1995.

The recent National Physical Activity Surveys (telephone-administered, 4,824 (1997) and 3841 (1999) adult Australians aged 18 to 75 years), indicates the proportion of Australian adults participating in sufficient physical activity to provide a health benefit declined from 62% to 57% (9). The proportion of Australian adults who reported not doing any physical activity increased from 13% to 15%.

A recent preliminary analysis of the cost of illness attributable to physical inactivity in Australia (10) estimated the direct health care cost for preventing and treating 6 major conditions in 1993-94. The costs attributable to physical inactivity, and the percentage of the total estimated costs were $161 million (18.0%) for coronary heart disease, $16 million (8.7%) for breast cancer, $15.7 million (19.2%) for colon cancer, $101 million (16.0%) for stroke, $27.5 million (12.7%) for Non-Insulin Dependent Diabetes Mellitus, and $56.2 million (10.0%) for depressive disorders. Although the estimate is considered to be unreliable at this stage, the direct health care costs in 1993-94 for all causes of mortality attributable to physical inactivity was $5651 million – 18% of the estimated total direct health care costs.

**SCIENTIFIC BASIS**

**Overweight and Obesity**

A high level of heritability for obesity has been established by twin studies with recent estimates of this being 30-40%. However, the recent dramatic increases in the prevalence of obesity in Australia (5, 6) cannot be explained by genetic factors – lifestyle factors such as over consumption of energy and decrease in physical activity provide the most reasonable explanation.

Most observational studies indicate a U- or J-shaped relationship between BMI and mortality, with individuals at very low and very high weights at increased risk. Recent studies have suggested that one reason for the relationship between low BMI and mortality might be the detrimental effects of low lean body mass rather than low body fat (11, 12). Others (13) have found that when analyses are controlled for smoking and weight loss associated with illness, an almost linear continuous relationship between BMI and mortality is found.
Individuals with a BMI of at least 30 kg/m² have a 50% to 100% increased risk of death due to all causes compared to those at a BMI of 20 to 25 kg/m², with most of the increase being due to cardiovascular causes (4,14). The relative increase in mortality rate attributable to obesity declines with age, however an increased risk for death with higher BMI is seen even among individuals aged 65 to 74 years (15).

The health risk of being either overweight or obese has recently been reviewed (16) (see box 1).

**Box 1: Diseases and Conditions associated with Obesity**

<table>
<thead>
<tr>
<th>Disease/Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary Heart Disease</td>
</tr>
<tr>
<td>Type 2 Diabetes Mellitus</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Dyslipidemia</td>
</tr>
<tr>
<td>Stroke</td>
</tr>
<tr>
<td>Sleep Apnoea</td>
</tr>
<tr>
<td>Pulmonary Dysfunction</td>
</tr>
<tr>
<td>Gallbladder Disease</td>
</tr>
<tr>
<td>Liver Disease</td>
</tr>
<tr>
<td>Osteoarthritis</td>
</tr>
<tr>
<td>Gout</td>
</tr>
<tr>
<td>Some cancers – colon, endometrial, postmenopausal breast cancer</td>
</tr>
<tr>
<td>Menstrual irregularities</td>
</tr>
<tr>
<td>Polycystic Ovary Syndrome</td>
</tr>
<tr>
<td>Infertility</td>
</tr>
<tr>
<td>Gestational Diabetes</td>
</tr>
<tr>
<td>Neural Tube Defects</td>
</tr>
<tr>
<td>Low back pain</td>
</tr>
<tr>
<td>Increased risk of anaesthesia complications</td>
</tr>
<tr>
<td>Carpal Tunnel Syndrome</td>
</tr>
<tr>
<td>Venous Insufficiency</td>
</tr>
<tr>
<td>Deep Vein Thrombosis</td>
</tr>
<tr>
<td>Poor Wound Healing</td>
</tr>
<tr>
<td>Psychosocial problems</td>
</tr>
<tr>
<td>Protective against Osteoporosis</td>
</tr>
</tbody>
</table>

For common causes of morbidity and mortality such as coronary heart disease, type 2 diabetes mellitus, hypertension, and dyslipidemia, the associations with obesity follow a monotonic dose-response relationship where risk increases with degree of obesity. It has been estimated that more than 70% of persons who are obese have at least 1 established co-morbid condition.
In cross-sectional analysis of a large and representative survey of US adults (17) in 1988 to 1994 (NHANES III) 63% of the men and 55% of the women were overweight or obese. The risk of self-reporting type 2 diabetes mellitus, gallbladder disease, high blood pressure was greater in these individuals and the risk increased among those who were heavier.

In both sexes, weight gain during adult life is associated with increased risk of heart disease and death (14, 18). Weight gain is a health risk that is independent of the actual level of BMI (19).

Mean BMI in a population is closely associated with the proportion of people classified as obese or overweight. As population mean BMI increases above 23 kg/m$^2$, the prevalence of obesity in that population increases at a faster rate because the BMI curves flatten and skew to the right.

Obesity refers to an excess of body fat, however most data on the effects of obesity on health rely on measurement of body weight. A limitation of BMI is that it does not incorporate body fat distribution, which is an independent predictor of health risk (4). Body fat may be preferentially deposited in the abdomen (android distribution) or surrounding the hips and thighs (gynoid distribution). The android distribution pattern has been demonstrated to reflect an accumulation of fat around the abdominal visceral organs (20). Even at the same level of overweight, an individual with a greater amount of visceral fat is more likely to have many of the serious health conditions associated with obesity. Methods to accurately assess visceral fat (such as computer assisted tomography or magnetic resonance imaging) are not routinely used, however an easily measured surrogate is waist circumference. Waist circumference of at least 88cm in women or 102 cm in men has been associated with increased health risk (4).

It is now believed that the measurement of waist circumference alone is more useful to identify health risk in adults than the ratio of waist circumference to hips circumference (20). Physical activity may favourably affect the distribution of body fat independently of its effect on body weight (21).

Many studies (eg 22, 23) have indicated that intentional weight loss in obese individuals reduces risk factors for, and improves symptoms of, obesity-related conditions including heart disease, type 2 diabetes mellitus and osteoarthritis over the short term (weeks or months). It is not necessary to lose large amounts of weight to achieve substantial health gains. For example, in a population of non-smoking US white women aged 40 to 64 years, weight loss of 5 to 10 kg over 1 year was associated with a 25% reduction in mortality (24). Most studies measuring the impact of weight loss for a year or more tend to show continuing risk factor reduction (25,26,27). For most people who are classified as obese, and many who are overweight, a return to a normal range BMI is not an appropriate target (3).

In the context of populations such as Australia where BMI tends to increase with age, the avoidance of weight gain in adult life is a successful outcome of weight management. Two trials to evaluate low intensity programs targeted at weight gain prevention in adults have been conducted in the United States (28, 29). Forster et al (29) randomised 219 normal-weight adults to a control or treatment group for a period of 12 months. The treatment consisted of monthly newsletters related to weight management, a financial incentive system and an optional four-session education course in the sixth month of the program. The treatment group had an average weight loss of one kg compared to no change in the
untreated control group. In the treatment group, 82% maintained or lost weight compared to 56% of the control group. Older participants benefited more from the treatment than younger, and men more than women. Those with prior experience in weight loss programs were significantly less likely to maintain pre-treatment weight than inexperienced participants.

Jeffrey and French (28) recruited 228 men and 998 women into a randomised controlled trial for 3 years. Participants were randomised to a control group (no treatment), a group who received education mainly through monthly newsletters and a group who received education plus a financial incentive (monthly $100 lottery draw). The overall mean weight change over 3 years was +1.7kg and there was no difference in weight change, or rate of gain, between the groups. The study demonstrated that it was possible to run a mail-based education program and maintain the interest of the heterogeneous participants for 3 years. They concluded that either stronger educational strategies were needed or that education alone is insufficient to address increasing weight throughout adulthood.

Physical Activity

The benefit of adopting an active lifestyle on cardiovascular and all-cause mortality is experienced more rapidly than changes to other risk factors – within 2 years in a US male cohort (30). In general, with mortality from cardiovascular disease as the outcome, large cohort studies have found a relative risk of 1.5 to 2.0 in sedentary adults compared to adults who were at least moderately active (31). Consistently across studies, the greatest cardiovascular disease benefit occurs in moving from sedentary or low fitness groups in the population to groups with moderate activity or moderate fitness levels.

The effects of physical activity on cardiovascular disease and non-insulin dependent diabetes are independent of the effects of other risk factors – physical activity is beneficial to health at any level of BMI regardless of whether BMI changes (21). The best evidence supporting a causal association between physical activity and health is from population based cohort studies. Meta-analyses indicate a stronger association between physical activity and health where research methodology is more highly rated. The strength and consistency of associations between physical activity and health have recently been reviewed (1, 9).

Recreational physical activity is predictive of future weight gain in prospective cohort studies (32,33,34), however reducing inactivity (or sedentary activities) is also an important strategy to increase incidental physical activity.

The following conclusions have been reached regarding physical activity and weight loss (21). The combination of physical activity and energy restriction is more effective for weight loss (and possibly maintenance of weight loss) than energy restriction alone. Physical activity effects body composition favourably during weight loss by preserving or increasing lean mass while promoting fat loss. Physical activity affects the rate of weight loss in a dose-response manner that is based on both frequency and duration of physical activity.

Six large-scale multiple risk factor community intervention trials that evaluated diet and exercise interventions have recently been reviewed (35). None of the studies focussed exclusively on weight, however each included significant nutrition and exercise education efforts. The trials were the Stanford Three-Community Project, the Stanford Five City Study, the Minnesota Heart Health Program, the North Karelia Project, the Pawtucket
Heart Health Program and a national program for Mauritius. The trials ranged in length from 2 to 7 years, and showed considerable success in intervention delivery. Only two of the studies showed any statistically significant weight related effects and these were small. The Stanford Three-Community project and the Pawtucket heart health program reported small attenuation of increases in relative weight (Stanford) or BMI (Pawtucket) in treatment versus control communities. For the Stanford Three-Community study a difference in relative weight of no change versus +0.3% in the control community was observed 1 year after the 2 year intervention was completed. This small difference was not maintained when a comparison was made a year later. In each of the community intervention trials, there were significant effects on other cardiovascular disease risk factors suggesting that weight gain in a more difficult risk factor to address than other risk factors.

### Dietary Intake

Experiments in animals and clinical studies in humans have indicated that both fat and energy intake are strongly and positively associated with excess body weight (36). Results from population-based studies are less consistent (37, 38), however a consensus is developing that fat consumption, at least, has an association with excess weight gain (3). Dietary fat has a higher energy density than other macronutrients, and fat-induced appetite control signals are thought to be too weak or too delayed to prevent overconsumption of energy from a fatty meal. The capacity for fat storage in the body is virtually unlimited. Excess dietary fat does not markedly increase fat oxidation and is stored with about 96% efficiency. Because carbohydrate and protein intake are auto-regulated much better than fat, it is becoming clear that weight changes are primarily due to an imbalance in total energy accounted for primarily by disruption to fat balance (4, 39, 40).

### PRACTICAL ASPECTS OF THIS GUIDELINE

#### Relationship to Australian Guide to Healthy Eating

The Australian Guide to Healthy Eating promotes a healthy lifestyle to include physical activity; and gives some direction to people who are overweight on how to follow a healthy eating plan that is consistent with weight loss.

#### CHANGES NEEDED TO LIFESTYLE

There is growing consistency in recommendations made to achieve prevention of weight gain in populations. They are:

1) **Increase the level of physical activity by what ever means appropriate to a much higher level than at present. This includes incidental activity, low-intensity (but long duration) leisure pursuits as well as moderate and vigorous exercise.**

2) **Reduce the time spent being physically inactive**

3) **Choose a less energy dense diet.**

Recent attention has been given to the need to facilitate the adoption and maintenance of lifestyle physical activities (5, 42). These are self-selected activities, could be planned or unplanned, and can include leisure, occupational and household activities to be accumulated throughout the day. It has been realised that a large proportion of the population are completely inactive and many people feel they do not have time to exercise,
dislike vigorous exercise and/or dislike the imposed conformity of organised exercise programs such as gymnasiums. Evidence has now accumulated that moderate level physical exercise taken intermittently also results in health benefit (42). The change in emphasis from ‘exercise training – physical fitness’ to ‘lifestyle physical activity’ allows people to adopt healthy behaviours which take into account their individual, cultural and environmental differences. The broader public health approach dispels the misconception that vigorous exercise is the only way to become physically fit. Furthermore, it lays a scientific basis for the design of public infrastructure to encourage incidental physical activity (for example pleasant walkways, and wide stairways in buildings).

Three evidence-based recommendations have been made for the delivery of brief physical activity interventions during routine health care delivery (43). These are: An initial focus on physical activity only is recommended, although maintenance may be enhanced when supported by other risk factor interventions; tailored interventions and written materials enhance success rates; and physical activity counselling can be successfully implemented by a variety of health care team members. The person who delivers the intervention should be whoever is most likely to do so consistently, given time, training and interest.

It has been suggested (44) that successful behaviour change in relation to weight can result from patient education and counselling and should include behavioural techniques, especially self-monitoring, and both personal communication and written or other audio-visual materials.

There is evidence to suggest that foods with a higher energy density encourages excess energy intake above requirements (45, 46). Energy from drinks in particular may add to total energy intake without displacing energy consumed in the form of solid food (47, 48, 49).

**Special groups/needs**

*Indigenous Australians*

The magnitude of the problem of overweight and obesity in Indigenous Australians can hardly be overstated. In the 1994 National Aboriginal and Torres Strait Islander survey, of those adults who were measured, about 25% of males had a BMI of at least 30 kg/m² and 28% of females (50, 51). The percentage for Australian adults was 19% in 1995. There is a suggestion that the prevalence of obesity is higher in rural communities, at least among women. It appears that the prevalence of obesity has rapidly increased in rural communities in recent years (52). In general, obesity and overweight in Indigenous Australians tends to have a central distribution (51). In surveys conducted in 3 regions of North Queensland between 1998 and 2000 (53), the percentage of males aged 35 years or more who had a BMI of at least 30kg/m² varied from 19% to 50%; for females aged 35 years or more, the percentage varied from 24% to 69%. Waist to hip ratio above 0.8 for women, or 0.9 for men varied from 87% to 94% in men aged more than 35 years, and from 90% to 97% for women aged more than 35 years.

*Blue Collar Workers*

Analysis of the 1995 Australian Health Survey (54) indicates that blue-collar employees (tradespersons, plant and machine operators and drivers, and labourers and related workers) were 50% more likely to be classified as insufficiently active with respect to
leisure-time physical activity. This group was also identified as being more likely to be overweight or obese (5), and to experience higher cardiovascular disease mortality and morbidity rates (55).

RELATION TO PREVIOUS DIETARY GUIDELINES

The last edition of the Dietary Guidelines for Australians (57) included ‘MAINTAIN A HEALTHY BODY WEIGHT BY BALANCING FOOD INTAKE AND REGULAR PHYSICAL ACTIVITY’. Since the publication of that guideline, the proportion of the Australian population who are classified as being overweight or obese has increased substantially. The increase has occurred in both children and adults. The dietary guideline to the general public must now be directed towards reversing the secular trend towards increasing mean BMI. The diminishing physical activity of Australians has been recognized as an important determinant of the epidemic of overweight and obesity. Physical Activity Guidelines for Australians were published in 1999 (2) and should be combined with the Dietary Guidelines to achieve the best health benefit.

Dietary intake is an important aspect of achieving and maintaining a healthy body weight, however the phrase ‘balancing food intake’ has been removed from the guideline because its meaning is not clear, and aspects of food intake are addressed by considering all the dietary guidelines together.

RELATION TO OTHER GUIDELINES

This guideline advocates that Australians incorporate substantial regular physical activity into their lifestyle, and consume an appropriate amount of food for their energy needs. Increased physical activity increases dietary energy requirements and will therefore require food intake to increase according to need. In addition to extra food energy, a greater absolute intake of all nutrients should follow from increased physical activity. To take best advantage of this, the dietary guidelines should be followed to provide a diet that is rich in fibre, vitamins, minerals and other beneficial food components, without unnecessarily increasing the total amount of less beneficial components such as saturated fat, salt and sugar.

CONCLUSION

The increasing prevalence of overweight and obesity in the Australian population is a major public health issue. Strategies to address the problem at a population level must include an increase in the level of regular physical activity. Physical activity has other health benefits that are unrelated to body fatness. Dietary intake must also be a part of the achievement and maintenance of a healthy level of body fatness.

EVIDENCE
The strong and consistent evidence linking body fatness with mortality and morbidity is from large prospective cohort studies. Randomised controlled trials of interventions to gain body fat are not feasible, although randomised controlled trials (Level II evidence) to examine intervention to lose body fat have been conducted (ref 27). The results have been consistent with the results of the cohort and cross-sectional studies ie. that excess body fat is associated with poorer health.

There is Level II evidence for the effect of weight loss on hypertension (ref 27) and Level III evidence for a relationship between body weight or body fat and mortality (refs 12, 13,15); between weight status and heart disease (ref 14); between increased obesity and diabetes and decreased plasma cholesterol in Aborigines (ref 52); between weight gain or body weight on blood pressure (ref 19); between weight loss and reduced mortality (ref 24); between increased physical fitness and mortality (ref 30) and between physical activity and future weight gain (refs 32 33 34). There is additional evidence from population-based cross-sectional studies.

The strength of the association between BMI and specific diseases ranges from strong (a relative risk much greater than 3) for Type 2 diabetes mellitus and dyslipidaemia, moderate (relative risk between 2 and 3) for coronary heart disease and hypertension, and slight (relative risk less than 2) for postmenopausal breast cancer and fetal defects (see ref 3). The mortality and morbidity associations are of substantial clinical and public health significance particularly in view of the high and increasing prevalence of overweight and obesity in Australia.

The evidence for health benefit of increased physical activity is considered good to excellent (see ref 31). Most of the evidence comes from good quality prospective cohort studies (eg 30, 41). Despite many different methods of measuring physical activity, the health benefits found are very consistent between studies, and reviews have found stronger associations for studies that used better research designs and methods.

Many of the reports referred to in this background paper are review articles across a range of subjects. The studies highlighted above in relation to levels of evidence, also cover a range of subjects, from studies to assess the effect of particular interventions relating to physical activity to studies describing change in health parameters according to body weight change with time. There are many more published studies relevant to this dietary guideline – this background paper does not attempt to comprehensively review them. Most of the stronger published evidence relating adult weight gain, body mass and physical activity to health comes from prospective cohort studies.
REFERENCES

47. Mattes RD. Dietary compensation by humans for supplemental energy provided as ethanol or carbohydrate in fluids. Physiol Behav 1996;59:179-87.
CARE FOR YOUR FOOD AND KEEP IT SAFE TO EAT

INTRODUCTION

Food safety is important because foodborne illnesses can have very serious health consequences, especially for pregnant women or people with weakened immune systems. Despite having one of the world’s safest food-supply systems, there has nevertheless been an increase in the number of foodborne diseases in Australia. Correct handling of food, during all stages of preparation and storage, reduces the incidence of foodborne illnesses.

BACKGROUND

While healthy adults are often unaffected, or only mildly affected, by infection with foodborne pathogens, within the adult population there are people who are more susceptible to infection. People considered to be at risk of infection are pregnant women and those with weakened immune systems. Within the adult population, this includes people with AIDS/HIV, cancer, diabetes, kidney or liver disease, hemochromatosis (an iron disorder), stomach problems (including previous stomach surgery) and low stomach acid (from antacid use), people treated with immunosuppressing medications, people undergoing marrow or stem cell transplantation, and people who have a history of long term steroid use (as for asthma and arthritis).

Attention to food safety is therefore important for all adults. The main causes of foodborne illness in Australia are:

- inadequate cooking of raw meat;
- inadequate washing of raw fruit and vegetables that are to be eaten raw;
- improper holding temperature;
- contaminated equipment;
- unsafe food source;
- poor personal hygiene.

The foods most likely to harbour micro-organisms that cause foodborne illnesses are undercooked and raw foods (particularly meat, eggs and unwashed fruit and vegetables), pre-cooked cooled foods¹ and ready-to-eat (RTE) foods. Ready-to-eat food is defined as “food that is normally consumed in the same state as that in which it is sold, and does not include nuts in the shell and whole raw fruits and vegetables that are intended for hulling, peeling, or washing by the consumer.”²

SCIENTIFIC BASIS

Food safety and adults

Healthy adults have well developed immune systems whereas people with weakened immune systems are more susceptible to infection by foodborne pathogens and can become ill even from eating foods that are prepared in a safe way.
Immune System Function

The body’s ability to defend itself against specific invading agents such as foodborne micro-organisms declines in those with weakened immune systems. They are more susceptible to all types of infection and are likely to suffer more severe consequences. In the case of foodborne illnesses, these consequences range from mild dehydration to neuromuscular dysfunction or death.

Gastrointestinal Tract Function

In healthy adults, no specific gastrointestinal functions contribute to infection by foodborne pathogens.

In the latter stages of pregnancy constipation may occur. At this stage a decrease in the motility of the digestive process may allow for the rapid growth of pathogens.

In people with weakened immune systems, malnutrition predisposes to gastrointestinal infection. Nutritional deficiency or infection cause gastritis and a resultant decline in gastric secretion of hydrochloric acid. A decrease in stomach acidity increases the chance of infection if foodborne pathogens are ingested.

The Incidence Of Foodborne Illnesses

Reported data on foodborne illnesses consistently underestimate the true incidence of these illnesses, and complete diagnostic testing is usually undertaken only in more severe cases or when there are extensive common-source outbreaks. Because of the apparently increasing incidence in Australia, and worldwide, foodborne diseases remain a significant public health problem. A number of factors are responsible for this apparently increasing incidence:

- the global economy—international trade in agricultural products and commodities is extensive and growing. It poses some risk of introducing new foodborne pathogens into countries or spreading pathogens across boundaries;
- changing eating patterns—foodborne hazards have emerged as a result of the considerable changes in eating patterns. Consumers are demanding healthier and minimally processed foods such as fresh cut fruits, vegetables, and salads; as well as convenient, pre-prepared heat-and-eat foods.
- demographic changes—as noted, foodborne illnesses pose the greatest risk for very young children, pregnant women, older people and people with weakened immune systems. People now live longer and as they age they become more susceptible to foodborne diseases;
- changes in food production—the potential hazards associated with advances in food production (for example, animal husbandry, extensive food distribution and increased scales of production) need to be identified and controlled if we are to minimise the risk of foodborne illness. Recent outbreaks in the US have been caused by foods previously considered to be “safe”, for example, ice-cream, pasteurized milk, apple juice, hot dogs, lettuce, and alfalfa sprouts.
• better reporting and identification of pathogens\textsuperscript{7}—dramatic scientific and technological improvements in the detection of pathogens have contributed to the reporting of increasing numbers of cases of foodborne disease that may have previously gone unreported;

• increased awareness among consumers and health professionals;

• new pathogens—some pathogens (for example, cyclospora, \textit{Listeria monocytogenes}, and \textit{E. coli} O157:H7) have only recently been shown to be predominantly foodborne.\textsuperscript{10,9} Many pathogens are also becoming resistant to antimicrobial agents;

• new foods being eaten or foods being eaten in new ways—for example, raw bean sprouts;

• changing bacterial behaviour—for example, acid-resistant \textit{Salmonella}.

In a 1999 report, it was estimated that foodborne illnesses cost Australia $2.6 billion each year.\textsuperscript{7} A reduction in the incidence of such illnesses would benefit the Australian community through lower health care costs, less absenteeism, improved business productivity, increased competitiveness in world markets, and a reduction in business failure and associated costs, including litigation.\textsuperscript{7} It is estimated that an average case of foodborne illness costs Australians $630; this is lower than the estimated cost of A$1679 in the United Kingdom and A$1531 in the United States.\textsuperscript{7}

In 1999 and 2000 the three most common notified foodborne diseases in Australia were infections with \textit{Campylobacter}, \textit{Salmonella} and hepatitis A (see also Table 4.1).

\textbf{Table 4.1 Notifications of foodborne illnesses received by Australian health authorities: selected pathogens, 1991 to 2000\textsuperscript{11}}

<table>
<thead>
<tr>
<th>Year</th>
<th>\textit{Campylobacter}</th>
<th>Hepatitis A</th>
<th>\textit{Listeria}</th>
<th>\textit{Salmonella}</th>
<th>\textit{Yersinia}</th>
</tr>
</thead>
<tbody>
<tr>
<td>1991</td>
<td>8 672</td>
<td>2 195</td>
<td>44</td>
<td>5 440</td>
<td>515</td>
</tr>
<tr>
<td>1992</td>
<td>9 136</td>
<td>2 109</td>
<td>38</td>
<td>4 614</td>
<td>567</td>
</tr>
<tr>
<td>1993</td>
<td>8 111</td>
<td>2 006</td>
<td>53</td>
<td>4 731</td>
<td>459</td>
</tr>
<tr>
<td>1994</td>
<td>10 117</td>
<td>1 901</td>
<td>34</td>
<td>5 327</td>
<td>414</td>
</tr>
<tr>
<td>1995</td>
<td>10 933</td>
<td>1 600</td>
<td>58</td>
<td>5 895</td>
<td>306</td>
</tr>
<tr>
<td>1996</td>
<td>12 158</td>
<td>2 150</td>
<td>70</td>
<td>5 819</td>
<td>268</td>
</tr>
<tr>
<td>1997</td>
<td>11 851</td>
<td>3 076</td>
<td>71</td>
<td>7 005</td>
<td>245</td>
</tr>
<tr>
<td>1998</td>
<td>13 449</td>
<td>2 503</td>
<td>58</td>
<td>7 700</td>
<td>207</td>
</tr>
<tr>
<td>1999</td>
<td>12643</td>
<td>1563</td>
<td>62</td>
<td>7 330</td>
<td>142</td>
</tr>
<tr>
<td>2000</td>
<td>13455</td>
<td>824</td>
<td>66</td>
<td>6 017</td>
<td>71</td>
</tr>
</tbody>
</table>

Incidences of infection with Hepatitis A virus, \textit{E. coli} O157:H7, \textit{Clostridium botulinum} and \textit{Salmonella} in the United States over the same period are presented in Table 4.2.
Table 4.2 Reported Cases of notifiable disease, USA 1991 – 1999

<table>
<thead>
<tr>
<th>Year</th>
<th>Hepatitis A (not infant)</th>
<th>E. coli O157:H7</th>
<th>Botulism</th>
</tr>
</thead>
<tbody>
<tr>
<td>1991</td>
<td>24378</td>
<td>27</td>
<td>48154</td>
</tr>
<tr>
<td>1992</td>
<td>23112</td>
<td>21</td>
<td>40912</td>
</tr>
<tr>
<td>1993</td>
<td>24238</td>
<td>27</td>
<td>41641</td>
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<tr>
<td>1994</td>
<td>26796</td>
<td>50</td>
<td>43323</td>
</tr>
<tr>
<td>1995</td>
<td>31582</td>
<td>24</td>
<td>45970</td>
</tr>
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<td>1996</td>
<td>31032</td>
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<td>30021</td>
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<tr>
<td>1998</td>
<td>23229</td>
<td>22</td>
<td>43694</td>
</tr>
<tr>
<td>1999</td>
<td>17047</td>
<td>39</td>
<td>40596</td>
</tr>
</tbody>
</table>

The general trend is that over the last 10 years in Australia, there have been gradual increases in the incidence of foodborne illnesses associated with Campylobacter, Salmonella and Listeria. In the United States, an increase in the incidence E. coli O157:H7 has been reported.

The Centers for Disease Control in the United States have targeted Campylobacter, Escherichia coli, Salmonella, Shigella, Vibrio and Yersinia because they are significant contributors to outbreaks of foodborne illnesses; they are easily transmitted and multiply rapidly in food.

Effect of Illness/Symptoms

The symptoms experienced by infected persons, are dependent upon the pathogen responsible, and the immune status of the affected person. Symptoms can range from so mild as to be hardly noticeable (in healthy adults) to so severe that hospitalisation is needed. Common symptoms of foodborne illness are abdominal pain, nausea, vomiting, diarrhoea, bloody stools, fever and dehydration. People may experience fatigue, fever and muscle pains. In serious cases, food poisoning causes double vision, trouble with swallowing or breathing, paralysis or death. Some new agents of foodborne illness (cyclospora, Listeria monocytogenes, and E. coli O157:H7) cause more than the typical gastroenteritis symptoms of vomiting and diarrhoea. They have also triggered reactive arthritis, autoimmune disorders, encephalopathy, meningitis, and septicemia.

The time taken between infection by the pathogen and development of symptoms also varies depending on the pathogen involved. Symptoms of commonly acquired foodborne infections may present themselves within 2-4 hours, or up to 10 days after infection.
Methods of food contamination

Food contamination can occur when:

- the area used for preparing food is not kept clean. For example, when insects or rodents get into the kitchen area.

- utensils used for cooking are not cleaned properly, or raw foods contaminate cooked foods. For example, a board used to cut raw meat is then used to cut foods that will be eaten raw, or juices from raw poultry come into contact with other foods during storage. This is called cross-contamination and is an important pathway for food poisoning\(^1\).

- the person preparing or serving contaminates the food as it is being cooked, prepared or served. This can happen when hands are not adequately washed, there are sores on the hands, or food handlers sneeze or cough into the food.

Causes of Foodborne Illnesses

Foodborne illnesses can be caused by bacteria, viruses or bacterial toxins.\(^7\) Bacterial food poisoning is the most common cause of foodborne illness in Australia\(^16\) and often occurs as a result of the growth of pathogenic bacteria to harmful levels if food is stored incorrectly. The principal factors affecting bacterial growth are temperature, time, nutrients and water\(^1,16,17\).

**Temperature**

Cooking to high temperatures will destroy the vegetative cells of bacteria; while cooling at low temperatures will slow bacterial growth. Some bacteria do, however, have heat-resistant spores and toxins that survive the cooking process—*Bacillus cereus* is an example.

Foods which support the growth of foodborne bacteria, should be stored at or below 5°C or at or above 60°C. Temperatures inside this range are considered to be the “danger zone” for food safety. Within this temperature range, bacterial replication can occur.

**Time**

The longer food is left in the temperature danger zone, the more quickly bacteria will multiply. The shorter the time food—cooked or uncooked—is left in the temperature danger zone, the lower the risk of food poisoning.\(^1,16\) Bacteria in the temperature danger zone can reach an infective dose for healthy adults in four to six hours.\(^1,16\)

**Nutrients**

Bacteria need adequate nutrients for replication. Among the most suitable media are high-protein, perishable foods such as dairy products, egg products, seafood, meat and
The acidity or alkalinity of a food will affect bacterial growth: bacteria are least active in very acidic foods (those with a pH less than 4.5). Often foods are preserved using vinegar to reduce the bacterial growth, although moulds may still grow in these conditions.

**Water**

Bacterial growth is limited in the absence of moisture, so bacteria are less likely to survive in dried products such as breakfast cereals and powdered milk. Some bacteria, or some forms of bacteria, can, however, survive the dry conditions and when fluid is added to the food they will grow again.

Not all foodborne pathogens need to multiply in food to cause illness. Viruses such as Hepatitis A and Norwalk virus, and even some strains of bacteria such as *E. coli* (e.g. *E. coli* O157:H7 and *E. coli* O111), *Campylobacter jejuni* and *Shigella* spp., can cause illness even when present in low numbers. Food must be protected from contamination to prevent the presence of these pathogens. If a ready-to-eat food is contaminated with these pathogens, illness may occur. Once the pathogens are present, keeping the food at a safe temperature will not make the food any less or more safe.

A number of microorganisms produce toxins when allowed to grow to high levels in food. Eating food which contains toxins can cause foodborne illness. For example, the illness botulism is caused by ingestion of a toxin produced by *Clostridium botulinum* present in contaminated food. Other microorganisms which are linked with toxin production in food are *Staphylococcus aureus* (staphylococcal food poisoning) and *Shigella*. Toxin formation can be prevented by ensuring that foods are kept at safe temperatures. Even cooking food at high temperatures will not destroy toxins.

**PRACTICAL ASPECTS OF THIS GUIDELINE**

**Preventing spoilage**

Spoilage bacteria cause foods to deteriorate and develop unpleasant odours, tastes, and textures. Spoilage decreases the quality of food and care should be taken to prevent spoilage during storage.

Uncooked meats, poultry, offal and seafood need particular attention because they always carry large numbers of spoilage bacteria; food poisoning bacteria may also be present. These foods should be stored in the coldest part of the refrigerator, as close to 0°C as possible. If these foods will not be used within 2-3 days, they should be frozen to prevent spoilage.

Frozen foods should be put into the freezer as soon as possible after they have been bought. Thawing, or even a rise in temperature without completely defrosting, stimulates chemical and microbiological activity and may result in spoilage. Freezing food almost completely stops microbial deterioration. The freezer temperature should be −18°C or lower. Place only fresh or freshly cooked food in the freezer. Foods lose their flavour and quality if they are frozen for too long.
Dehydrated and Dried Foods

Dehydration inhibits the growth of micro-organisms by removing water but it does not make the food sterile\(^\text{21}\): a high level of micro-organisms may remain, only to become active again when the food is rehydrated. Rehydrated foods should be treated as perishables and be stored in the refrigerator.\(^\text{21}\)

Dried food should be stored in a sealed container and in a cool, dry place, away from direct heat or sunlight. It should be regularly inspected for insect infestation.\(^\text{21}\) Opened packages can also be stored in the refrigerator to maintain quality for longer.

Optimising Food Safety

To optimise food safety, care should be taken at all stages of the consumer’s ‘food chain’— purchasing, transport, storage, preparation, cooking, serving and cleaning.

Purchasing

Although the standard of foods in Australia is generally very high, it is important that buyers examine each package carefully, looking for defects in packaging (such as improper sealing of boxes), foreign objects, and signs of spoilage. Cans that are damaged, swollen, rusted or deeply dented should be rejected\(^\text{24}\), as should any vacuum-packed foods that are not properly sealed.

Always check the use-by or best-before date, and be mindful of how the food is being stored and displayed. It is best to leave the buying of chilled and frozen foods to the end of the shopping trip, to prevent them from warming.\(^\text{1,16,25}\) Foods such as this can be put in a cooler with ice, and groceries can be put in the air-conditioned part of the car during warm weather.\(^\text{7,21,26}\) To avoid the need to transport goods in coolers, purchase food from retailers close to home and deliver it immediately after purchase.

Care should be taken to select unblemished fruits and vegetables.

Storage

The various food types need to be stored correctly to retain their nutrient value, freshness, aroma and texture and to keep them safe.\(^\text{1,16,21,20}\) Always read the label for storage conditions.

Ensure that storage areas such as cupboards and pantries are clean\(^\text{24}\), and that foods are stored in food-grade containers. Store raw foods separately from ready-to-eat foods to prevent cross-contamination of the RTE foods\(^\text{18}\).

Refrigeration

Refrigeration retards the growth of bacteria and the rate of chemical change in food. The refrigerator temperature should be around 5°C or less. Care should be taken to ensure that this temperature is maintained.

All cooked foods should be covered and stored on a shelf above uncooked foods\(^\text{1,16,21,20}\).
Leftovers and ready-to-eat meals should be used the next day or stored in the freezer. Raw meats should be wrapped or placed in a container and stored near the bottom of the refrigerator, to ensure that the juices do not drip onto other foods. Any spills should be cleaned up immediately and fridge and freezer shelves and doors sanitised regularly.

Ready-to-eat (RTE) chilled foods are becoming widely available. These should be stored in the coldest part of the fridge i.e. at or below 5°C and used within the use-by or best-before date or as soon as possible after purchase.

Frozen Foods
Care should be taken to ensure that the correct temperature (<18°C) of the freezer is maintained. Freezer burn is dehydration or drying that occurs on the surface of a product if it is improperly wrapped. The food is safe to eat but poorer quality. Foods should be stored in packages that are free of air and sealed airtight to prevent freezer burn.

Canned Food
Canned foods are sterilised during processing and should be stored in a cool place. Read labels carefully for storage instructions and take note of the use-by date. Once opened, canned foods should be stored in the refrigerator, preferably not in the can. Swollen or leaking cans indicate faulty processing and their contents should not be eaten. Throw out the contents of any can if there is an unusual odour.

Vacuum Packed and Modified Atmosphere Packed (Map) Foods
Vacuum packing extends the shelf life of food by removing air from packages. Modified atmosphere packaging extends shelf life by replacing oxygen in a packaged food with other gases to slow bacterial growth. MAP is often used with meat and poultry products. An increasing number of blister packs of foods such as fresh pasta, lunch meat, bacon and olives, are now available.

Vacuum packed and MAP foods should be stored according to the instructions on the individual package.

Date Marked Packaged Foods
The best before date on packaged food signifies the end of the period during which the intact package of food, if stored in accordance with any stated storage conditions, will remain fully marketable.

The use-by date on packaged food signifies the end of the estimated period, if stored in accordance with any stated storage conditions, after which the intact package of food should not be consumed because of health and safety reasons.

Although the sale of packaged food past the expiration of the use-by date is prohibited, care should still be taken during purchasing to ensure that the use-by date on packaged foods has not yet been reached.
**Preparation**

Before beginning food preparation and after handling raw foods, touching animals, using the bathroom, assisting others with bathroom use, and changing children’s nappies, hands should be washed with soap and warm (43-48ºC) water. Hands should be lathered thoroughly and held under running water for 20 seconds to ensure any micro-organisms are washed away. Particular attention should be paid to washing between fingers and under fingernails. After washing, dry hands thoroughly using either a clean towel or a paper towel. Food should not be prepared by anyone who may be suffering from a foodborne illness or who is carrying a food-borne disease. Special care should be taken with cleaning after cutting up raw meat and before dicing vegetables. Marinate raw foods in the refrigerator. The marinade can be used during cooking but do not add it to the cooked dish or use it as a dressing if raw meat has been in it. Chopping boards with a non-absorptive surface (for example, glass or plastic) are easier to clean than wooden chopping boards. Use a different chopping board and utensils when preparing foods to be eaten raw and those for cooking. During food preparation, do not taste the food with the same utensil used for stirring.

Never place cooked food on plates that have held raw meat, poultry or seafood. And never use the tea towel as a hand towel or for cleaning surfaces. Never thaw foods on the counter. Thaw them in the refrigerator or under cold water in an airtight plastic wrapper or bag, changing the water every 30 minutes. Only thaw in the microwave if the foods are to be cooked immediately afterwards. Thawed foods should not be refrozen and the thawing fluids should not contaminate other foods, containers and serving utensils.

Thaw ready to eat foods (such as TV dinners) in the fridge or re-heat from frozen. Check information on pre-packaged foods to determine whether they need to be cooked or re-heated before being eaten.

**Fruits and vegetables should be washed thoroughly under running water before peeling and cutting, and cutting away bruised areas.** Special care should be taken washing produce such as parsley or lettuce as they are harder to clean than fruits with smooth skins. It is also recommended that, where possible, fruits and vegetables that are grown in or on the ground be cooked before being eaten.

**Cooking**

Not all meat needs to be cooked thoroughly—steaks, whole fillet, chops and whole pieces of roast meat can be eaten rare. In contrast, rolled and/or stuffed meats and other pieces of meat, poultry, pork, sausages and mince should always be cooked to a centre temperature of at least 74°C. To check the temperature, use a thermometer: the colour of the meat and the juices is not an accurate indicator of centre temperature.

When using a microwave to cook, rotate and stir the food so that it cooks evenly. Cover it with a lid or plastic wrap so that the steam can aid thorough cooking. Food finishes cooking during standing time, so it is important to wait until the standing time has elapsed before checking that cooking is complete. Never refrigerate partially cooked products and finish cooking them later. Meat, fish and poultry must be cooked thoroughly; they can then be refrigerated and reheated later.
Cooling

Cooking alone does not guarantee safety because some bacterial spores can survive several hours of cooking. Foods such as stews and other meat and poultry dishes that are not intended for immediate consumption, should be cooled as quickly as possible to prevent spores from germinating and bacteria from multiplying. Place leftovers in the refrigerator to cool—for quick cooling, divide them into smaller portions and store them, covered, in shallow containers. Do not leave them to cool completely on the bench, instead cool hot foods uncovered in the fridge and cover after cooling – make sure covers seal tightly. Foods should be refrigerated within two hours of cooking and be used within 2-3 days.

Re-heating

When reheating food, cook it until it is ‘steaming hot’ throughout: this will destroy any bacteria that have grown on the food during refrigeration. Bring soups, sauces and gravies to a rolling boil. Do not reheat food more than once.

When heating pre-prepared frozen or refrigerated dinners, follow the instructions on the packet to ensure the food is safely heated before being eaten.

Serving

Serving food safely is essential. Food poisoning can result if food is not handled and served safely as soon as possible after it is cooked. Hands should be washed with soap and water and dried thoroughly using a clean towel or a paper towel, and food should be served on clean plates. Never put cooked food on a plate that has held raw food or re-use utensils used during food preparation. Unless foods are to be served immediately, they should be covered until ready to be eaten.

If hot foods are not to be consumed immediately, keep them above 60°C. The time between cooking and eating is when a food is most vulnerable to contamination from the growth of micro-organisms with heat-resistant spores. For buffets, food can be kept hot by using chafing dishes or warming trays. Cold food should be kept cold (<5°C) by keeping it in the refrigerator until served, or for buffets by keeping it on ice.

Cleaning

Use hot, soapy water to wash all work surfaces, crockery, cutlery, cooking utensils and other equipment. Clean cutting boards with an antibacterial cleaner or bleach and water mixture, or put them in the dishwasher. Dishwashers wash dishes and utensils at 60°C, a much higher temperature than used when washing dishes by hand.

Foodborne bacteria readily persist in kitchen towels, sponges and cloths: wash and dry them often and replace sponges regularly. Paper towels reduce the risk of cross-contamination because they are disposable, and therefore cannot harbour and spread bacteria.

For added protection, keep appliances such as microwave ovens, toasters, can openers, and blender/mixer blades free of food particles - use a bleach solution to sanitise chopping blades and hard to clean areas. Keep counters and surfaces free of food particles.
Lifestyle factors

The above guidelines are simple to follow when preparing food in the home, however additional food safety precautions should be taken when travelling, at BBQs, and picnics, preparing bulk foods, and when eating out at restaurants or selecting take-away foods.

TRAVELLING

When travelling through areas of poor sanitation any raw foods can be contaminated. Foods of particular concern include salads, uncooked vegetables and fruit, un-pasteurised milk and milk products, raw meat, and shellfish. The basic rules of eating and drinking in an unfamiliar place are “boil it, cook it, peel it, or throw it away”. Where possible, avoid street vendors because of the high possibility that foods have been prepared and stored using inadequate safety procedures.

Tap water and ice-cubes used in beverages may also contain pathogens. Buy bottled water for drinking and for cleaning teeth with. Buy canned or bottled soft drinks or beer and wine and check that the seals on these are intact before consuming them. In eating establishments request that drinks are served without ice.

Picnics

For transporting picnic foods pack them in a cooler with ice or ice packs. Only pack foods that have been chilled to a temperature below 4 degrees C – do not use the cooler to chill room temperature foods. Do not store things that do not need to be cooled in the cooler – eg fruits, vegetables, chips, bread.

Do not put the cooler in the boot instead carry it within the air-conditioned car. At the picnic put the cooler in shade and keep the lid closed. Avoid repeated openings of the food by keeping drinks in a separate cooler. If possible, replenish ice when it melts.

When finished serving cold foods, promptly return them to the cooler. Potentially hazardous foods should be discarded upon your return home.

If no soap and water is available, liquid sanitiser or disposable wet wipes should be used to clean hands before and after eating or preparing food. This is especially important if handling raw foods.

BBQs

Use coolers and icepacks to transport food from the supermarket or from home to the BBQ. If taking raw meat to barbeque, ensure it is packed carefully to prevent leakage and cross-contamination of raw products.

Before cooking, thoroughly clean all BBQ tools and surfaces where food is placed. Keep all food (including marinating food) in the fridge until just prior to cooking, or transporting to the site of the BBQ.

Ensure meat is fully defrosted so that it cooks evenly on the BBQ. Use one set of utensils for raw meats/poultry and another set for cooked. Do not put cooked meat on plates that have previously held raw meat.
Do not partially grill meat patties to use later. Once cooking has begun, it should continue until patties are completely cooked to assure that bacteria are destroyed (FSIS, 1997). Burgers, sausages, pork and poultry should be cooked until the juices run clear and all foods should be eaten as soon as possible after cooking. Keep food hot on the side of the grill rack or in the oven until eaten.  

**PREPARATION OF BULK FOODS/ENTERTAINING**

If cooking large amounts of food for an occasion, only prepare in advance what your refrigerator can cool and hold. Divide large amounts into small containers for fast chilling and easier use.  

**Foods purchased from delicatessens are intended for immediate consumption. If purchasing bulk delicatessen foods to serve to guests, ensure that they are transported and handled in a safe manner (using appropriate temperature control), and are consumed as soon as possible after purchasing.**

A variety of boutique foods are becoming increasingly popular and may be served to groups of people when entertaining. Examples include tapenades, homemade herb and vegetable flavoured dipping oils, and hommus. Certain specific food safety precautions should be taken in the preparation of these. For example, incorrect preparation or storage of garlic flavoured oils have resulted in serious outbreaks of botulism in Canada and the United States.  

**Restaurant dining and takeaway meals**

At restaurants or cafes the consumer has very little control over the way in which food is prepared. Added food safety can be achieved by selecting restaurants which are generally clean and where staff are well presented. Ensure staff use tongs and gloves to serve food, and clean cloths to wipe surfaces. If possible, check that raw and cooked foods are well separated. Check the toilets are clean.  

At buffet or self-service meals, check that hot food is stored in hot food display cabinets or over burners, and that cold food is displayed on ice or in special refrigerated cabinets. Each dish should have its own serving utensils and fresh food should be replenished regularly. Foods should be covered by some type of guard or cover, and plates and cutlery should be clean and dry.  

Some evidence suggests that eating food from a common bowl or sharing eating utensils can result in the transfer of the bacterium *Helicobacter pylori* from person to person. This however, is not proven. To avoid the transfer of any micro-organisms, people should, when possible, avoid sharing common bowls or utensils.  

Some styles of food (for example, Indian or Turkish foods) encourage the consumer to eat with hands instead of utensils. Where this is the case, standard personal hygiene procedures should be followed, and hands washed thoroughly before and after eating.  

Takeaway meals should be eaten within 2 hours of their purchase or immediately put in the fridge and eaten within 2 days. For high risk groups extra precautions can be taken.
fast food establishments ask that food be prepared fresh while you wait. Request single serving condiment packages and avoid self-serve bulk condiment containers to reduce the risk of infection by *Salmonella*. Check sandwiches and salads purchased at restaurants and delicatessens and request that sprouts are not added to food.\[^{40}\]

**Foods recommended during pregnancy**

*Safe food suggestions for pregnant women include pasteurised reduced fat milk products, freshly washed vegetables and fruit, dried foods and cereals.*\[^{41}\] Freshly prepared, thoroughly cooked meats may also form a significant part of the diet. In addition, it is recommended that pregnant women avoid chilled pre-cooked seafood products (unless freshly cooked and eaten hot), uncooked seafoods (clams, mussels, oysters and scallops), pre-cooked meat products such as chicken, ham and luncheon meats (unless thoroughly re-heated), undercooked or rare meat, pate and meat spreads, hot dogs, stored salads and coleslaw, raw (un-pasteurised) milk and products made from un-pasteurised milk, and soft cheeses (*Camembert, Brie, blue-veined cheese, Mexican style cheeses*).

What should you do if you believe you have suffered from effects of food poisoning?

- **Seek treatment as necessary.** Seek medical care immediately if you are pregnant, have a weakened immune system, or if symptoms persist or are severe (for example, bloody diarrhoea, excessive nausea and vomiting, high temperature).

- **If the suspect food was served at a large gathering, from a restaurant or other foodservice facility, or if it is a commercially produced product, contact your local health department.**

- **If possible, preserve the evidence.** Wrap the remaining portion of the food securely, label it with “DANGER” and freeze it. Save all packaging materials, and any identical unopened products. Record details of when the food was consumed, and when the onset of symptoms occurred.\[^{35}\]

**RELATION TO OTHER GUIDELINES**

*Prevent weight gain by being physically active and eating according to your needs*

Research has shown that immune cells are responsive to the effects of acute exercise, in terms of both number and function. Regular physical activity can be beneficial for older people’s immune system function and increase the body’s ability to defend itself against foodborne illnesses.\[^{42}\]

*Eat plenty of vegetables (including legumes) and fruits*
This guideline closely relates to food hygiene and the purchase, transport, storage, preparation and cooking of vegetables and fruits. When these foods are stored correctly their nutritional quality and storage life are maximised. Buy fresh vegetables and fruits that are ‘firm’ and make sure canned and frozen varieties have complete packaging and are undamaged.

*Eat plenty of cereals (including breads, rice, pastas, noodles), preferably wholegrain*

Correct storage will maximise the storage life and prevent spoilage of breads, cereals, pastas, fats (including cooking oils) and dairy products. It is important to make sure that packaging is whole and undamaged.

*Include lean meat, fish, poultry and/or alternatives such as legumes and nuts*

Illness due to food borne, pathogenic bacteria is a public health issue. All foods are potential vectors of pathogens. In Australia the risk of food borne illness in primary food industries is managed across the food chain, with industry, government and consumers sharing responsibility for the delivery of microbiologically safe products. Nevertheless, some foods from this food group have been implicated in outbreaks of food borne disease (74,75) and constant vigilance is required.

**CONCLUSION**

The education of all food handlers, health care providers and the general public is essential if we are to reduce the incidence of foodborne illness. Although most foodborne illnesses can be avoided by safe food-handling procedures, risk reduction is very important at every step of the way, from food purchase to meal serving.

**EVIDENCE**

Because of the nature of this guideline, there is no evidence of the kind required for the NHMRC Levels of evidence. It is not possible to conduct trials, case-control, cohort or experimental studies around safe food. Evidence relating to safe practice, comes from a basic understanding of food microbiology and human physiology and is based on a wide variety of evidence from laboratory studies opportunistic evidence from outbreaks of food poisoning.
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ENCOURAGE AND SUPPORT BREASTFEEDING

INTRODUCTION

Breastfeeding is the appropriate method for feeding infants and is closely related to the immediate and long-term health of the infant. Exclusive breastfeeding to the age of about six months gives the best nutritional start to infants and is recommended by a number of authorities (1) (2) (3) (4). A review of breastfeeding duration was undertaken by the WHO and more than 3 000 references were identified (www.who.int/inf-pr-2001/en/note2001-07.html). The WHO Expert Consultation recommended exclusive breastfeeding for about 6 months, with the introduction of complementary foods and continued breastfeeding thereafter. It is recommended that breastfeeding continue until 12 months of age and thereafter as long as mutually desired (3).

If for any reason, breastmilk is discontinued before 12 months of age, then an infant formula should be used, rather than cow’s milk as the main source of milk. Breastmilk from a healthy well-nourished mother is adequate as the sole source of nutrients for full-term infants from birth until about six months of life. Aside from nutritional suitability, colostrum and mature human milk are hygienic and provide immunoglobulins and other anti-infective agents, which play a major role in protecting the infant against infection and disease. Breastmilk also includes a number of unique growth factors (5). Breastmilk is also a convenient and inexpensive food source, with no environmental cost. (See the NHMRC Infant Feeding Guidelines for a more detailed discussion of Breastfeeding).

In Australia, although the majority of women commence breastfeeding (82%), just under a third of these women have introduced supplements or have ceased breastfeeding by three months (6). There is also evidence for considerable variation between socioeconomic groups in both the acceptance and maintenance of breastfeeding in the Australian community; those in higher socio-economic groups are more likely to breastfeed (6) (7). The support and encouragement of family, friends and the whole community are required to maximize breastfeeding rates and duration.

From a public health viewpoint there is considerable room for improvement in both the rates and duration of breastfeeding in Australia that would bring benefits to maternal, infant and child health. Exclusive breastfeeding of infants for about the six months of life for as many infants as possible should be the goal. Of the developed countries, Norway consistently reports the highest rates, rates that Australia should strive to achieve:

- 92% of mothers are breastfeeding their child at 3 months of age
- 80% are breastfeeding at 6 months
- 40% are still breastfeeding at 12 months (8).

The advantages of breastfeeding continue beyond the six-month period. Australians other than mothers, can play a major role in making breastfeeding an easy and viable option. Encouragement and support by hospitals and health centres, families, friends, social groups and places of work combine to ensure that women can breastfeed successfully. The inclusion of breastfeeding as a dietary guideline is thus a recognition of the nutritional, health, social and economic benefits of breastfeeding to the Australian community and of
the need for family and community support for breastfeeding. Breastfeeding should be combined with other health promotion programs (9).

DEFINITIONS

Exclusive breastfeeding means that the infant is receiving only breastmilk, including expressed breastmilk, or from a wet nurse. The infant may also receive medications, including vitamins or minerals as required.

Complementary food means any food, whether manufactured or locally prepared, suitable as a complement to breastmilk or to infant formula, when either becomes insufficient to satisfy the nutritional requirements of the infant. Such food is also commonly called ‘weaning food’ or ‘breastmilk supplement’ (10).

Research methodology and data collection
A variety of methods are used to study and record breastfeeding rates. Reported studies use different sampling methods and may rely on the mothers’ memory of past events. Studies that use frequent interviews of a representative cohort and use standard definitions are more accurate (11).

BACKGROUND

In reviews of infant feeding in Australia, Hitchcock (12) and Lund-Adams and Heywood (13) describe the decline in breastfeeding rates in Australia and other developed countries that occurred during the 20th century. Rickets, scurvy and hypernatraemia were associated with early artificial feeding as the knowledge of infant requirements was limited.

Breastfeeding reached a low point in the in the 1960s and records from Victoria showed that only 50-60% of mothers were breastfeeding on discharge from hospital, and only 21% at three months. In the early 1970s breastfeeding rates began to increase again in Australia and comparable overseas countries, beginning in the higher socio-economic groups. By 1983 both the prevalence and duration of breastfeeding in Australia were among the highest in the western world with 85% at discharge and 54-55% three months later (14). Breastfeeding have remained around this level for the past two decades. In 1984–85 a joint survey in Western Australia and Tasmania indicated a continued trend to increasing breastfeeding rates and duration in those States. Over the preceding five years, prevalence rates at hospital discharge rose from 82% to 86% in Western Australia and 72% to 81% in Tasmania. At six months after discharge 45% of mothers in both States were still breastfeeding (15).

In 1992-93 in Western Australia and in 1995-96 in Queensland a survey was carried out by Scott et al (7) that indicated a continued trend to increasing breastfeeding rates and duration in those States. Over the preceding years the hospitals had a breastfeeding discharge rate of 82.3% and by six months 46.4 % of mothers were still breastfeeding. Women who were born in Australia or New Zealand were almost twice as likely to be breastfeeding at discharge than women born in other countries.
Donath and Amir (6) have analysed the data from the National Nutrition Survey (1995) and found that breastfeeding rates were 81.8% on discharge from hospital, and 57.1% fully breastfed at 3 months. At 6 months, it is estimated that 18.6% of babies are fully breastfed and 46.2% fully or partially breastfed. At one year of age, 21.2% of infants are receiving some breastmilk. Thus in Australia at present fewer than 20% are achieving the goal of being exclusively breast fed to six months of age.

Australia has a long history of promoting and supporting breastfeeding in its public health policy. The importance of breastfeeding led to its inclusion in the Dietary Guidelines for Australians endorsed by National Health and Medical Research Council (NHMRC) at its ninety-third session in June 1982. In 1981 Australia became a signatory to the World Health Organization (WHO) International Code for the Marketing of Breastmilk Substitutes. The stated aim of the code was “to contribute to the provision of safe and adequate nutrition for infants, by the protection and promotion of breastfeeding and by ensuring the proper use of breastmilk substitutes, when these are necessary, on the basis of adequate information and through appropriate marketing and distribution” (10).

More information on the WHO Code and its implications for health workers is included in the NHMRC Infant Feeding Guidelines for Health Workers. The Nutrition Taskforce of the Better Health Commission (BHC) set targets for the year 2000 of increasing prevalence rates for breastfeeding at discharge from hospital to 95% and increasing the proportion still breastfeeding at three months to 80% (16). The rationale behind the targets was to continue the promotion of breastfeeding so that rates in at-risk groups would increase, the average period of breastfeeding would be lengthened and current overall levels of breastfeeding would be maintained. With current knowledge of benefits of breastfeeding, extending the goal to 80% breastfeeding at six months would be appropriate.

**SCIENTIFIC BASIS**

**Breastfeeding Physiology**
Milk production and secretion are under endocrine and autocrine control (17). When the infant suckles at the breast, mechanoreceptors are stimulated, resulting in the release of oxytocin and prolactin into the blood from the posterior and anterior pituitary respectively. Oxytocin stimulates the contraction of cells and secretion of milk from the alveolus. Prolactin is responsible for milk production in the alveolus. The commonly termed let-down reflex can also be stimulated by sighting the infant or hearing its cries. The reflex can be inhibited by stress such as pain or anxiety (18). Close mother-child contact immediately after birth assists the establishment of lactation, and frequent suckling or feeding on demand helps to maintain it (19) (20). Milk synthesis is related to the rate at which the breast is emptied (17)

A review of early contact practices stated “Mothers should have contact with their babies as soon after birth and for as long as they wish. Interventions aimed at either delaying or speeding up the time of the first feed should be avoided” (21). Hospital practices at the time of birth can be the first line of support to the new mother. Difficulties encountered here can be quickly resolved by staff with appropriate experience, and hospitals can encourage rooming-in to facilitate frequent mother-child contact. The composition of breastmilk is uniquely suited to the needs of a healthy infant at term and in the early
months of life. The composition of breastmilk is only compromised in mothers with severe malnutrition. Breastmilk is a living tissue that cannot be duplicated by any other means. Breastmilk is also most important for preterm infants.

Colostrum, the secretion produced in the first few days after giving birth, provides all the nutrients, including water, required by the neonate. Its composition is distinct from both transitional and mature milk, with higher levels of protein, vitamins A and B-12, and less fat. Colostrum contains lactoferrin, immuno-globulin A, enzymes, maternal antibodies, living cells—leukocytes, neutrophils and macrophages—and non-pathogenic bacteria, which act in the gut of the newborn to limit growth of pathogenic bacteria and viruses and protect against illness (22) (19)

The composition of this first secretion after birth gradually changes as lactation is established, and production of milk begins in the breast tissue. By 7–14 days after birth, lactation should be established and the transition from colostrum to mature milk under way. The nutrient composition of mature, expressed human milk shows variation in and between individuals, with maternal diet and with the stage of lactation, however, mean ranges are remarkably consistent for the species. The energy content is based upon the fat, protein and carbohydrate levels, and varies between 270–315 kJ (65–75 kcal)/100ml, largely as a result of variation in the fat content. Fat typically increases three to four-fold during a single feed, and also shows diurnal variation. Fat provides much of the energy and omega-3, omega-6 long chain polyunsaturated fatty acids, and also carries the fat soluble vitamins A, D, E and K, and prostaglandins (22) (19). This fat is typically better absorbed by the infant’s gastrointestinal tract than the fat in cow’s milk. The lipase present also works to increase the efficiency of absorption. Mature milk continues to provide immune factors and enzymes to the infant.

Human breastmilk will also provide all the major minerals and trace elements known to be essential for healthy term infants. Although the levels of some micronutrients appear to be low in comparison to other milks, the high bioavailability of these components in human milk ensure that no deficiencies occur. Actual requirements of young infants for all nutrients are not precisely known, however, nutrients in human milk have clearly been adequate for infants for thousands of years. Consequently, the composition of infant formulae (23) and Recommended Dietary Intakes for groups of infants in Australia are based upon the nutrient composition of human milk (24). However no infant formula can resemble breastmilk. Breastmilk is constantly changing throughout lactation and throughout the feed. As well constituents of breastmilk are still being discovered and many of the constituents in breastmilk cannot be replicated (25).

**Health Benefits of Breastfeeding**

There are many benefits to be gained from breastfeeding for the infant, the mother and the community. The benefits of Breastfeeding are summarised in Table One (See the Dietary Guidelines for Children for an expanded version). Increasingly there is interest in the long-term effects of perinatal nutrition, commonly referred to as the “Barker Hypothesis”. Inadequate or inappropriate foetal and early infant nutrition has been linked with subsequent chronic disease in adulthood (26) (27).
Table 1: Health advantages of breastfeeding for infants and mothers

<table>
<thead>
<tr>
<th>NT</th>
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<tbody>
<tr>
<td>• Reduced incidence and duration of diarrhoeal illnesses.</td>
</tr>
<tr>
<td>• Protection against respiratory infection and reduced prevalence of asthma</td>
</tr>
<tr>
<td>• Reduced occurrence of otitis media and recurrent otitis media.</td>
</tr>
<tr>
<td>• Possible protection against neonatal necrotizing enterocolitits, bacteraemia, meningitis, botulism and urinary tract infection.</td>
</tr>
<tr>
<td>• Possible reduced risk of autoimmune disease, such as diabetes mellitus type 1 and inflammatory bowel disease.</td>
</tr>
<tr>
<td>• Reduced risk of developing cow’s milk allergy.</td>
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<tr>
<td>• Possible reduced risk of adiposity later in childhood.</td>
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<tr>
<td>• Improved visual acuity and psychomotor development, which may be caused by polyunsaturated fatty acids in the milk, particularly decosahexaenoic acid.</td>
</tr>
<tr>
<td>• Higher IQ scores, which may be the result of factors present in the milk or to greater stimulation.</td>
</tr>
<tr>
<td>• Reduced malocclusion due to better jaw shape and development</td>
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<tr>
<td>• Early initiation of breastfeeding after birth promotes maternal recovery from childbirth; accelerates uterine involution and reduces the risk of haemorrhaging, thereby reducing maternal mortality; and preserves maternal haemoglobin stores through reduced blood loss, leading to improved iron status.</td>
</tr>
<tr>
<td>• Prolonged period of postpartum infertility, leading to increased spacing between successive pregnancies if no contraceptives are used.</td>
</tr>
<tr>
<td>• Possible accelerated weight loss and return to pre-pregnancy body weight.</td>
</tr>
<tr>
<td>• Reduced risk of premenopausal breast cancer.</td>
</tr>
<tr>
<td>• Possible reduced risk of ovarian cancer.</td>
</tr>
<tr>
<td>• Possible improved bone mineralization and thereby decreased risk of postmenopausal hip fracture.</td>
</tr>
</tbody>
</table>

Sources: modified from (28) (29) (30) (31)

The protective benefits of breastfeeding against mortality are obviously of greater magnitude in countries with higher infant mortality rates. A pooled study by a WHO working group has illustrated just how valuable the protection of breastfeeding can be, especially in developing countries where these studies were undertaken (See Figure One). The odds ratios for mortality from all causes and from infectious diseases show substantial benefit until the age of six months.
The Promotion of Breastfeeding Intervention Trial (PROBIT), undertaken in Belarus is the largest cluster-randomised controlled trial of breastfeeding promotion and outcomes to be published (33). A total of 17,046 mother-infant pairs consisting of full-term singleton infants weighing at least 2,500 g and their healthy mothers who intended to breastfeed, were studied. The 31 hospitals involved were randomised to receive a health promotion program based on the WHO/UNICEF Baby Friendly Hospital initiative. The infants form the intervention group were much more likely to be breastfed at 12 months and exclusively breastfed at 3 months and 6 months than the control group. The intervention group showed a significant reduction in the risk of one or more gastrointestinal tract infections (9.1% vs 13.2%; adjusted OR, 0.60; 95% CI, 0.40-0.91) and of atopic eczema (3.3% vs 6.3%; adjusted OR, 0.54; 95% CI, 0.31-0.95), but no significant reduction in episodes of respiratory tract infection.

The psychological and behavioural aspects of breastfeeding are also important. Breastfeeding is largely a pleasurable and positive interaction between mother and baby. The maternal hormones of prolactin and oxytocin stimulate the development of maternal behaviour and bonding, and as well, reduce the response to stress (34).

Factors in the Initiation and Duration of Breastfeeding

An extensive review of the literature extensively has documented the demographic, social and economic factors associated with breastfeeding (35). There is a higher prevalence and
a longer duration of breastfeeding among mothers from higher socioeconomic groups who are better educated, older and who have previously breastfed (6) (36) (37) (38) (39) (40).

In a longitudinal study Scott et al (7) confirmed the known demographic factors that influence breastfeeding rates and duration. Other factors that were found to have influenced a mother’s decision to breastfeed and the duration of breastfeeding, included the perceptions of partners and other family members, the mother’s decision to breastfeed prior to pregnancy, age and country of birth of the mother. They also found that a mother who had more than one child, intended to return to work or study within six months or had an infant in a special care nursery (SCN) was less likely to breastfeed (41). In Australia boys are breastfed for a shorter period of time than their sisters (42). Further research is needed to understand the reasons for this. The factors involved in the initiation and duration of breastfeeding in the Perth, Melbourne and Queensland studies, which all used the same methodology (cohort studies with frequent interviews) are summarised in Table Three.

These studies have demonstrated the importance of family support, particularly from the father, and the early education of the parents about the benefits of breastfeeding. McIntyre et al (43) analysed social support and found that social support for breastfeeding as provided by fathers, grandmothers and the general community in a low socioeconomic area is not high, particularly in relation to breastfeeding in public, in combining breastfeeding and work, and the appropriate management of breastfeeding.

Most women experience a number of “minor” issues while breastfeeding. If appropriate advice and support are not given, a mother may prematurely terminate breastfeeding. In studies in Australia and other developed countries the main reason for termination cited by women is a perceived insufficient milk supply (18). However the actual number of mothers who may be physiologically incapable of providing sufficient milk is extremely low (44). For the remainder of women who prematurely terminate breastfeeding, there are numerous causes, both biological and psychological, the majority of which are temporary and can be resolved with experienced advice, or avoided by better preparation, hospital management or appropriate support. For example the practice of encouraging rooming-in of infants while in hospital facilitates frequent feeding and hence, establishment of lactation (45). Breastfeeding is disrupted when the infant is housed away from the mother in the hospital therefore the rooming in option is offered by most hospitals today (46). Modern hospital practice means that mothers are discharged too early (often within 24-48 hours of delivery) for the hospital staff to have had much involvement in the establishment of breastfeeding. A review of negative hospital practices, such as the distribution of commercial publicity packs, has shown that they can have a detrimental effect on breastfeeding (47).

Frequent stimulation is required to maintain breastmilk production, one very good reason for discouraging any complementary feeds or the use of pacifiers (dummies). Feeding according to need (on demand) is the best method to maintain lactation and if this is prevented by lack of facilities or social acceptance, lactation can be adversely affected.

The Nursing Mothers’ Association of Australia (NMAA) and other similar organisations, have an important role to play within the health care system, providing the one-to-one support required to overcome transient problems with lactation, particularly after hospital discharge (48). NMAA also provides mothers with breastfeeding education classes, access to a local network of mothers and group discussion meetings, and a range of publications relating to all aspects of breastfeeding. NMAA also undertakes a range of other strategies
to promote breastfeeding in the community including: community education sessions run by local groups all around Australia; promotion of breastfeeding in local areas during Breastfeeding Awareness Month; encouragement to the community to support breastfeeding through ‘Baby Care Room’ awards, ‘Mother Friendly Workplace Accreditation’, and ‘Breastfeeding Welcome Here’ Stickers; participation in consultations relating to policies impacting on breastfeeding; access for the community and health professionals to comprehensive and readily usable information and resources on all aspects of human lactation through the Lactation Resource Centre (http://www.nmaa.asn.au).

Breastfeeding and employment need not be mutually exclusive although in some cases work may be a reason why women do not commence breastfeeding (18) (49) (50). For successful lactation to continue after returning to work, supportive worksite health promotion policies are required that provide education and facilitate either frequent feeding or frequent expression and storage of breastmilk as required. A recent publication provides helpful information for mothers and employers (Balancing Breastfeeding and Work, http://www.health.gov.au/hfs/pubhlth/strateg/brfeed/index). While not the ideal situation, many mothers who return to work are unable to breastfeed exclusively, and rather than using expressed breastmilk, carers may use infant formulae for some feeds. While mothers should be supported in this decision, as any breastfeeding is better than none, every effort should be made to change the conditions of our society to make exclusive breastfeeding possible for working mothers.

**Breastfeeding and adverse outcomes** (See Infant Feeding Guidelines for details)

There are few contraindications to breastfeeding. Most medications required by mothers are compatible with breastfeeding, but each drug needs to be specifically checked in a reliable reference (51).

Some disease situations, such as HIV/AIDS may be absolute contraindications (52).

Exclusive breastfeeding for periods much beyond six months of age will result in undernutrition and micronutrient deficiency (4). Supplementation with solid foods will then be required. Beyond the age of six months, additional sources of iron are required, usually from iron-fortified cereals or other foods (53) (See Children’s Dietary Guidelines, Variety Chapter for details).

**Breastfeeding and community support**

Community efforts should focus on strategies and policies which will:

1. Influence the proportion of mothers who intend to breastfeed; the earlier the decision is made before or during the pregnancy, the greater the likelihood of successful breastfeeding;

2. Influence the intended duration of breastfeeding through education, example and support;

3. Influence the attitudes and beliefs of the mother’s support network, and particularly their partners (fathers);
4. Provide antenatal and postnatal education about the day to day realities and practicalities of breastfeeding;

5. Promote breastfeeding as the social norm, with support and the provision of adequate facilities in social situations and in the workplace;

6. Include fathers and/or other support persons in as much of the antenatal preparation as possible;

7. Post discharge support needed for minor problems, from the community services, medical profession and support organizations; and

8. Enhance support for lactation in the workplace to allow working mothers to continue to breastfeed.

In a meta-analysis of support for mothers postnatally in the community, Sikorski and Renfrew (54) showed that “one more mother will breastfeed for two months if support is provided for nine women and one more woman will breastfeed exclusively if support is given to nine women.”

The role of the media is important in portraying the importance of breastfeeding and in supporting breastfeeding in the community as the norm. Breastfeeding is often portrayed in a negative way by the Australian media (55). In the United Kingdom it was found that television and press coverage routinely implies that breastfeeding is problematic, funny or embarrassing or associates it with “particular types of women”. On the other hand bottle-feeding is seen as “largely normalised, socially integrated, associated with 'ordinary' and 'normal' families and represented as being problem-free” (56).

The provision of physical facilities adequate for breastfeeding is important and are often lacking in places where mothers and their infants have to go, such as in shopping centres and other public places (57).

Recent research has shown how important the support of fathers is to encourage the initiation and duration of breastfeeding. The fathers of infants who were breastfed had the following characteristics:
- Had other breast-fed children
- Attended antenatal classes
- Discussed breast-feeding antenatally

However in general, fathers have poor knowledge about the practical issues of breastfeeding (7). It is important to include fathers in discussions about breast-feeding. Fathers need to provide practical help (such as occupying other children and household chores) and emotional support for breastfeeding mothers. They need to attend antenatal classes and learn of the nutritive and protective advantages of breast-feeding and some of the likely practical issues.

All health professionals need to continuously promote the benefits of breastfeeding and to ensure that their activities do not discourage mothers from breastfeeding. In a program to promote “Baby friendly doctors offices”, workshops were conducted for office staff. The
workshops resulted in positive changes in breastfeeding promotion. The change was maintained at 6 and 12 months after the intervention (58). In a controlled trial in an obstetrician’s office the negative effect of exposure to formula promotion materials was also demonstrated. Educational materials about infant feeding should support unequivocally breast-feeding as optimal nutrition for infants (59).

A meta-analysis of distribution of commercial information packs (with or without formula) to mothers while in hospital reduces the number of women exclusively breastfeeding at all times but does not effect the earlier termination of non-exclusive breastfeeding (47).

**Economic Benefits**

There have been many studies of the economic benefits to be gained from breastfeeding. Based on longitudinal studies in Arizona and Scotland, it was estimated that for each 1,000 infants never breastfed, there is an excess of 2,030 doctors visits and more than 200 inpatient days, 600 prescriptions compared with infants exclusively BF for more than 3 months (60). They estimated the total health care costs of non-breastfed infants was $331,000 greater than breastfed infants in the first year of life.

In a recent study in the US, Weimer (61) estimated that a minimum of $3.6 billion would be saved, if breastfeeding were increased from current US levels (64% BF in-hospital, 29% BF at 6 months) to the targets recommended by the US Surgeon General (75 and 5%). This figure is likely an underestimation of the total savings because it represents cost savings from the treatment of only three childhood illnesses: otitis media, gastroenteritis, and necrotising enterocolitis. In Australia the total value of breastfeeding to the community makes it one the most cost effective primary prevention measures available and well worth the support of the whole community.

**OTHER GUIDELINES**

**Children’s Dietary Guidelines**
- Pregnancy and breastfeeding in the adolescent
- Further information on indications for the introduction of solids (Variety and Growth Guidelines)

**Infant Feeding Guidelines**
- Advice on breastfeeding initiation and management
- Advice on issues and problems encountered in breastfeeding
- Health professionals’ responsibilities under the WHO Code
- Use of formulae

**CONCLUSION**

Breastfeeding is most important for infant nutrition. Exclusive breastfeeding until around six months should be the aim for very infant. However where that is not possible mothers should be encouraged to breastfeed as much, and for as long, as they can. Breastfeeding beyond six months is of continuing value to baby and mother, although the maximum benefits of breastfeeding are in the earliest months of life.
The promotion of breastfeeding is an important public health strategy. Support and encouragement at all levels of the community is essential to maintain and improve the rates and duration of breastfeeding by Australian women, particularly those who are disadvantaged in any way. The inclusion of breastfeeding in the dietary guidelines is aimed at contributing to the health of all Australians from birth.
Table 2  Factors associated with breastfeeding in a rural population compared with an urban population

<table>
<thead>
<tr>
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<tbody>
<tr>
<td><strong>Factors associated with the decision to breastfeed:</strong></td>
<td><strong>Factors associated with the decision to breastfeed:</strong></td>
</tr>
<tr>
<td>1. Mothers aged 25-30 years were 0.37 times as likely to initiate breastfeeding as mothers aged less than 25 years (CI&lt;sub&gt;95%&lt;/sub&gt; 0.16-0.89)</td>
<td>1. Where fathers preferred breastfeeding mothers were 5.6 times more likely to initiate breastfeeding than where fathers were ambivalent or preferring formula feeding (CI&lt;sub&gt;95%&lt;/sub&gt; 2.83-11.04)</td>
</tr>
<tr>
<td>2. Where fathers preferred breastfeeding mothers were 11.16 times to initiate breastfeeding then where fathers were ambivalent or preferring formula feeding (CI&lt;sub&gt;95%&lt;/sub&gt; 4.03-30.96)</td>
<td>2. Where maternal grandmothers preferred breastfeeding mothers were 4.00 times as likely to initiate breastfeeding as where grandmothers were ambivalent or preferred formula feeding (CI&lt;sub&gt;95%&lt;/sub&gt; 1.76-9.08)</td>
</tr>
<tr>
<td>3. Mothers who decided to breastfeed pre-pregnancy were 4.11 times as likely to initiate breastfeeding as mothers who made the decision during or after pregnancy (CI&lt;sub&gt;95%&lt;/sub&gt; 2.10-8.06)</td>
<td>3. Mothers who decided to breastfeed pre-pregnancy were 3.68 times as likely to initiate breastfeeding as mothers who made the decision during or after pregnancy (CI&lt;sub&gt;95%&lt;/sub&gt; 2.19-6.27)</td>
</tr>
<tr>
<td>4. Multiparous mothers were 0.33 times as likely to initiate breastfeeding as primiparous mothers (CI&lt;sub&gt;95%&lt;/sub&gt; 0.14-0.78)</td>
<td>4. Primiparous mothers were 2.24 times as likely to initiate breastfeeding as multiparous mothers (CI&lt;sub&gt;95%&lt;/sub&gt; 1.23-4.17)</td>
</tr>
<tr>
<td>5. Mothers not born in Australia, UK, Asia, Middle East or North Africa were 0.35 times as likely to breastfeed as were mothers from other countries (CI&lt;sub&gt;95%&lt;/sub&gt; 0.13-0.93)</td>
<td>5. Mothers not born in Australia, UK, Asia, Middle East or North Africa were 0.35 times as likely to breastfeed as were mothers from other countries (CI&lt;sub&gt;95%&lt;/sub&gt; 0.13-0.93)</td>
</tr>
<tr>
<td>6. Women whose husbands were tradesmen or labourers were 0.37 times as likely to initiate breastfeeding as women whose partners were professionals or administrators (CI&lt;sub&gt;95%&lt;/sub&gt; 0.18-0.78)</td>
<td>6. Women whose husbands were tradesmen or labourers were 0.37 times as likely to initiate breastfeeding as women whose partners were professionals or administrators (CI&lt;sub&gt;95%&lt;/sub&gt; 0.18-0.78)</td>
</tr>
</tbody>
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## DURATION OF BREASTFEEDING

### AL

**Factors associated with the risk of ceasing breastfeeding:**

1. Mothers aged over 30 years were 0.41 times as likely to cease breastfeeding as mothers aged less than 25 years (CI\textsubscript{95%} 0.22-0.77)

2. Mothers who planned to breastfeed for 2 to 6 months were 0.35 times as likely to cease breastfeeding as mothers who planned to breastfeed for less than 2 months (CI\textsubscript{95%} 0.07-0.44)

3. Where fathers preferred breastfeeding mothers were 0.62 times as likely to cease breastfeeding as where fathers were ambivalent or preferring formula feeding (CI\textsubscript{95%} 0.39-0.96)

4. Mother who decided to breastfeed pre-pregnancy were 0.45 times as likely to cease breastfeeding as were mothers who made the decision during or after pregnancy (CI\textsubscript{95%} 0.28-0.74)

5. Mothers whose infants received complementary formula feeds in hospital were 1.75 more likely to cease breastfeeding as mothers whose infants did not receive complementary feeds (CI\textsubscript{95%} 1.06-2.90)

### URBAN

**Factors associated with the risk of ceasing breastfeeding:**

1. For every extra year of age, a mother was 0.94 times as likely to cease breastfeeding (CI\textsubscript{95%} 0.91-0.97)

2. For every extra year of education, a mother was 0.91 times as likely to cease breastfeeding (CI\textsubscript{95%} 0.85-0.97)

3. Mothers born in the Middle East or Africa were 0.29 times as likely to cease breastfeeding as mothers born in Australia, New Zealand or the United Kingdom (CI\textsubscript{95%} 0.14-0.58)

4. Mothers from Perth were 0.64 as likely to cease breastfeeding as mothers from Melbourne (CI\textsubscript{95%} 0.45-0.91)

5. Mothers who planned to breastfeed for less than 4 months were 3.10 times as likely to cease breastfeeding as mothers who planned to breastfeed for more than 4 months (CI\textsubscript{95%} 2.09-4.61). Mothers who were undecided about how long they would feed where 1.51 times as likely to cease breastfeeding as mothers who planned to breastfeed for more than 4 months (CI\textsubscript{95%} 1.10-2.70)

6. Where maternal grandmothers were ambivalent or preferred formula feeding mothers were 1.40 times as likely to cease breastfeeding as where maternal grandmothers preferred breastfeeding (CI\textsubscript{95%} 1.03-1.90)

7. Mothers who didn’t receive conflicting advice in hospital were 0.68 as likely to cease breastfeeding as mothers who receive conflicting advice (CI\textsubscript{95%} 0.50-0.92)

Source: (62)
EVIDENCE

There are three relevant Cochrane reviews available. There is Level I evidence for the effect of early versus delayed initiation of breastfeeding (ref 21); for the biological suitability of breastmilk (ref 3); for exclusive breastfeeding to about six months (ref 4); for the role of breastfeeding in prevention of infant mortality in less developed countries (ref 32); for the effect of commercial hospital discharge packs for breastfeeding women (ref 47) and for community support of breastfeeding. (ref 54).

There is Level III evidence for the effect of baby-friendly initiatives (ref 58); for the effect of office prenatal formula advertising (ref 59) and for factors associated with breastfeeding in women in Australia (refs 7, 41, 42).
REFERENCES


38. Scott JA, Gowans MC, Hughes RM, Binns CW. Psychosocial factors associated with breastfeeding at discharge and duration of breastfeeding amongst two populations of

42. Scott JA, Binns CW. Breastfeeding: are boys missing out? Birth 1999;26:276-277.


Levels and kinds of evidence for public-health nutrition

Evidence-based medicine is now being used for two features of public-health nutrition: the development and revision of dietary guidelines, and the validation of health claims on foods. Guidelines advise people—for example, to eat less of saturated fat. Health claims declare a benefit—for example, that a food contains less saturated fat than does similar food and may thus help reduce the risk of coronary heart disease (CHD). Many countries have sets of dietary guidelines. Most countries do not permit health claims on foods, although in the USA a dozen claims are allowed by the Food and Drug Administration. The evidence must be more rigorous for health claims than for guidelines because they could give some food companies a commercial advantage.

The Australia New Zealand Food Authority (ANZFA) permits only one health claim at present—that foods providing folate, naturally occurring or appropriately fortified, may reduce the risk of a woman having a baby with neural-tube defect. Last year ANZFA reviewed how they might process possible submissions for further health claims on foods. Its expert advisory committee on scientific substantiation decided to modify, for health claims, the levels of evidence used by Australia's National Health and Medical Research Council (NHMRC) for developing clinical guidelines, which put multiple, then different types of controlled trials, in the top three levels, and cohort and case-control and time-series studies into the fourth level. ANZFA's levels for interpreting nutrition research (panel) has fewer levels for controlled trials, which are less available than are observational epidemiology for relations between nutrition and disease. Cohort (prospective) studies are considered more reliable than case-control studies because food intakes are collected by the same method for those who become cases and those who do not.

At an invited workshop held by the Australian Academy of Science's national nutrition committee, participants generally agreed that ANZFA's levels of evidence provide a guide for assessing the relation between diet and disease. The second point, made repeatedly, was that a rank order of levels of evidence is only one basis for assessing the value of evidence about diet and disease. Randomised controlled trials (RCTs) can be poor—for instance, when the study is too small, when there is no independent confirmation of the experimental dietary change, or when the outcome data may be insecure. Cohort studies can be good if dietary assessments are carefully validated, if correction is made for likely confounding factors, and if there is independent support from cohort studies that obtained similar findings in different populations.

RCTs of dietary change through to disease outcome are uncommon and most likely to involve addition or
National Health and Medical Research Council

COMMENTS

ANZFA proposed levels of evidence for food or health claims

<table>
<thead>
<tr>
<th>Grade</th>
<th>Evidence</th>
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<tbody>
<tr>
<td>A</td>
<td>Systematic review of all RCTs</td>
</tr>
<tr>
<td>B</td>
<td>Properly designed RCTs or well-designed pseudo-randomised RCTs</td>
</tr>
<tr>
<td>C</td>
<td>Cohort studies</td>
</tr>
<tr>
<td>D</td>
<td>Case-control studies or interrupted time-series with a control group</td>
</tr>
<tr>
<td>E</td>
<td>Comparative studies with a historical control</td>
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<tr>
<td>F</td>
<td>Case-series</td>
</tr>
<tr>
<td>G</td>
<td>Other relevant information, such as reports of expert committees</td>
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</table>

Subtraction of a single nutrient. Trials with whole diets are rare. They are complex and more labour-intensive than drug trials but less funding is available. There are, however, hundreds of controlled trials of dietary change with a surrogate outcome—a risk factor such as plasma lipids, plasma glucose, or blood pressure. Meta-analyses of collections of such trials have established that saturated fat increases plasma cholesterol, and many cohort studies have found that plasma cholesterol is a risk factor for CHD. Hence, by two-step reasoning, saturated fat increases the risk of CHD.

Besides, randomisation is not important for controlled dietary experiments in which all participants are exposed to the same experimental diet, with control periods before and after and with multiple measurements of the variable of interest, all analysed in the same batch. And for some of the most firmly accepted precepts of healthy eating, RCT findings are unlikely to be available. It is hard to imagine a trial in which half of a large group of people (randomised) agree to avoid vegetables for 5 years to assess their chances of developing cancer. Most of the health claims allowed by the US FDA have not been supported by RCTs.

Cohort (prospective) studies stand out among types of observational epidemiology for their driving role in building present concepts of diet and disease. Earlier cohort studies recorded as baseline data simple potential risk factors such as "do you smoke cigarettes?" or "how many alcoholic drinks do you usually have in a week?" These are much easier questions to answer than how many grams of the different fatty acids people eat in an average day. Development of food-frequency questionnaires (FFQs) has made it possible to include estimates of detailed dietary intake in large numbers of people, and in recent years, intakes of different fatty-acid classes, and of fish, fibre, folate, flavonoids, fruit, vitamin E, and wholegrain cereals, have been reported in different cohort studies followed to CHD events. But there are still far fewer cohort studies of dietary components and disease than of risk factors and disease.

Cohort studies have three weaknesses: the estimate of food intake, confounding, and practical duration of follow-up. For such studies, FFQs are commonly collected by postal survey with no interviewer's help; they do not correlate well with more direct measurements of actual food intakes or with dietary records. A single snapshot of diet does not reflect the gradual changes that take place over years in most people's eating habits. Cohort studies with periodic estimates of food intake, backed by biochemical tests of key nutrients, must clearly rank higher than once-only questionnaires on quality. Dietary components associated (or not) with a disease (eg, dietary fibre) may accompany a feature of lifestyle that was not tested but is more directly related. This confounding can never be completely eliminated. Lastly, cohort studies are short-term compared with the average human life-span. Most cohorts are of middle-aged people in whom there is a fair risk of the disease developing, so such studies cannot reveal effects of the diet in childhood and adolescence.

Case-control studies have the additional drawbacks of uncertainty about how long the likely incubation period is between the possibly causative diet and onset of the chronic disease and of inaccuracy in summoning up past dietary habits.

When the results of RCTs and observational epidemiology appear to be in conflict, it would be unsafe to assume that one level of evidence has automatic priority over another. Two sets of discrepancies between RCTs and observational epidemiology have shaken nutritional knowledge. Foods rich in β-carotene are inversely associated with lung and other cancers but β-carotene has shown no protective effect in RCTs. Possible explanations are that forms and/or dosage of β-carotene were unphysiological, and that carotene might still be protective against an earlier stage of carcinogenesis than that which occurs in the 5 years of a trial. It is even more difficult to explain why two large cohort studies found that high (supplementary) vitamin E intakes seemed protective against CHD but several secondary and primary prevention trials did not.

Agreement to apply appropriate principles of evidence-based medicine to public-health nutrition will bring welcome objectivity and the opportunity to have rules of evidence for claims and disputes. The evidence will never consist mostly of RCTs. Emphasis instead will have to be on all the evidence, including animal studies and molecular biology and critical interpretation of the observational epidemiology.

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THE LANCET • Vol 357 • April 7, 2001
NHMRC LEVELS OF EVIDENCE

The National Health and Medical Research Council (NHMRC) has released a guidebook explaining “How to use the evidence assessment and application of scientific evidence.” This guidebook, however, relates to evidence assessment in relation to clinical practice. In many cases the development of evidence-based guidelines for clinical practice deals with evidence in relation to a specific disease and a specific therapeutic agent. Similar criteria are not easily used for assessment of evidence related to food in relation to maintenance of general health and well being in the community. The latter is the primary focus of Dietary Guidelines.

There are a number of initiatives underway around the world to try to develop an evidence-based approach to nutrition and health issues, but this has generally been in response to the need for "proof" in relation to health claims for food components. In Australia, a set of proposed levels of evidence for food or health claims has been developed by ANZFA, which is similar to, but somewhat broader in scope than the NHMRC approach for clinical guidelines. Nevertheless it is still primarily intended to assess evidence related to the efficacy of individual nutrients or food components in relation to a specific health outcome.

However, it may still be useful to consider the NHMRC designation of levels of evidence for clinical practice in relation to the scientific data discussed in this document.

NHMRC’s Level of Evidence are outlined below:

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Evidence obtained from a systematic review of all relevant randomised controlled trials.</td>
</tr>
<tr>
<td>II</td>
<td>Evidence obtained from at least one properly-designed randomised controlled trial.</td>
</tr>
<tr>
<td>III-1</td>
<td>Evidence obtained from well-designed pseudorandomised controlled trials (alternate allocation or some other method).</td>
</tr>
<tr>
<td>III-2</td>
<td>Evidence obtained from comparative studies (including systematic reviews of such studies) with concurrent controls and allocation not randomised, cohort studies, case-control studies, or interrupted time series with a control group.</td>
</tr>
<tr>
<td>III-3</td>
<td>Evidence obtained from comparative studies with historical control, two or more single arm studies, or interrupted time series without a parallel control group.</td>
</tr>
<tr>
<td>IV</td>
<td>Evidence obtained from case series, either post-test or pretest/post-test.</td>
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</table>


Six levels of evidence are designated by NHMRC, (Level I) being based on a systematic review of all relevant randomised control trials with Level II being on evidence obtained from at least one properly designed randomised control trial. There are very few Level I or Level II food-based nutrition trials although some
nutrient-supplement trials fall into these categories. Most food-health studies fall within Level III, the level of evidence which include study designs such as cohort studies, case-control studies and comparative ecological studies with historical controls.

It is of interest to note that the NHMRC states that “a decision should be made about what is feasible and appropriate in a given situation and the extent to which reasonable standards have been met by the available body of evidence”.

APPENDIX C

SELECTED RESULTS OF NATIONAL NUTRITION SURVEY

The first background paper titled ‘Enjoy a Wide Variety of Nutritious Foods’ refers.

In the 1995 National Nutrition Survey the foods eaten were classified into 14 different groups. As the following two graphs illustrate, in virtually all age groups, males who live alone eat a significantly fewer number of food groups each day.
Food Groups Males and Females 25-29 years: NNS

- 25-29 FE With
- 25-29 FE Alone
- 25-29 M With
- 25-29 M Alone

Number of Food Groups vs. Percentage
ASSESSMENT OF OBESITY

Australia, and most of the western world, is in the grip of an obesity epidemic. In children the growth chart will remain the primary tool in the assessment of growth and the risk of obesity. In children the Body Mass Index (BMI) [Weight (kg)/Height$^2$(M)] has now become accepted as a useful measurement and monitoring tool (Dietz and Bellizzi 1999). These authors suggested using the commonly used adult cut off points of 25 (overweight) and 30 (obese). Particularly for older children the BMI has gained increasing acceptance.

Adults

The BMI has gained wide acceptance for the assessment of obesity in adults. In Australian adults the cutoff points used and recommended by the NHMRC are:

- Normal range BMI 20-25
- Overweight BMI 25-30
- Obese BMI >30

A more comprehensive classification from the WHO is:

- Underweight BMI <18.5
- Normal range BMI 18.5-25
- Overweight BMI >25
- Pre Obese BMI 25.0-29.9
- Obese Class I BMI 30.0-34.9
- Obese Class II BMI 35.0-39.9
- Obese Class III BMI >40

Recently Cole and his colleagues (2000) used pooled data from 192,727 subjects from a number of countries, aged from birth to 25 years, to develop cut off points for each age which correspond to the adult levels of overweight and obesity listed above. These cut off points, are a big improvement on current alternatives, and should help to provide internationally comparable prevalence rates of overweight and obesity in children. These cut-off points are shown on the following page.

International cut-off points for body mass index for overweight and obesity by sex between 6 and 18 years, defined to pass through body mass index of 25 and 30 kg/m$^2$ at age 18, obtained by averaging data from Brazil, Great Britain, Hong Kong, Netherlands, Singapore, and United States.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Body Mass Index 25 kg/m$^2$</th>
<th>Body Mass Index 30 kg/m$^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
</tr>
<tr>
<td>6</td>
<td>17.55</td>
<td>17.34</td>
</tr>
<tr>
<td>6.5</td>
<td>17.71</td>
<td>17.53</td>
</tr>
<tr>
<td>7</td>
<td>17.92</td>
<td>17.75</td>
</tr>
<tr>
<td>7.5</td>
<td>18.16</td>
<td>18.03</td>
</tr>
<tr>
<td>8</td>
<td>18.44</td>
<td>18.35</td>
</tr>
<tr>
<td>8.5</td>
<td>18.76</td>
<td>18.69</td>
</tr>
<tr>
<td>9</td>
<td>19.10</td>
<td>19.07</td>
</tr>
<tr>
<td>9.5</td>
<td>19.46</td>
<td>19.45</td>
</tr>
<tr>
<td>10</td>
<td>19.84</td>
<td>19.86</td>
</tr>
<tr>
<td>10.5</td>
<td>20.20</td>
<td>20.29</td>
</tr>
<tr>
<td>11</td>
<td>20.55</td>
<td>20.74</td>
</tr>
<tr>
<td>11.5</td>
<td>20.89</td>
<td>21.20</td>
</tr>
<tr>
<td>12</td>
<td>21.22</td>
<td>21.68</td>
</tr>
<tr>
<td>12.5</td>
<td>21.56</td>
<td>22.14</td>
</tr>
<tr>
<td>13</td>
<td>21.91</td>
<td>22.58</td>
</tr>
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<td>13.5</td>
<td>22.27</td>
<td>22.98</td>
</tr>
<tr>
<td>14</td>
<td>22.62</td>
<td>23.34</td>
</tr>
<tr>
<td>14.5</td>
<td>22.96</td>
<td>23.66</td>
</tr>
<tr>
<td>15</td>
<td>23.29</td>
<td>23.94</td>
</tr>
<tr>
<td>15.5</td>
<td>23.60</td>
<td>24.17</td>
</tr>
<tr>
<td>16</td>
<td>23.90</td>
<td>24.37</td>
</tr>
<tr>
<td>16.5</td>
<td>24.19</td>
<td>24.54</td>
</tr>
<tr>
<td>17</td>
<td>24.46</td>
<td>24.70</td>
</tr>
<tr>
<td>17.5</td>
<td>24.73</td>
<td>24.85</td>
</tr>
<tr>
<td>18</td>
<td>25</td>
<td>25</td>
</tr>
</tbody>
</table>
APPENDIX E

ABORIGINAL AND TORRES STRAIT ISLANDERS

Current Health and Nutritional Status

Aboriginal and Torres Strait Islander peoples continue to suffer a much greater burden of ill-health and particularly nutrition-related chronic disease than other Australians (58, 3, 54). In 1998, indigenous death rates were at least three times greater than the total Australian population, with indigenous life expectancy around 20 years less than for the total Australian population (3). Much of this poor health can be attributed to poor nutrition (58).

Indigenous Australians continue to suffer disproportionally high rates of many nutrition-related conditions, such as type-2 diabetes mellitus, cardiovascular disease, renal disease, poor dental health, iron deficiency anaemia and some forms of cancers. Overweight and obesity tend to underpin the development of many of these chronic diseases (38, 61).

The potential intergenerational effects of poor health and nutritional status have been well described (9, 10, 11, 77). Low birth rate, failure to thrive and inappropriate child growth are major concerns in indigenous Australian communities (58,71, 33). Diabetes in pregnancy also has potential intergenerational effects (65, 73) and is an additional concern.

Vitamin and mineral status has been measured infrequently but some studies have been conducted in a variety of groups and environments (58). Samples have generally been small and have often been selected from ‘stress’ groups of the community (infants and pregnant and breast-feeding women). Quantitative comparison of prevalence of vitamin deficiencies may be misleading as varying methods and ‘normal’ ranges have been used in these studies. Multiple deficiencies have frequently been described in the same subject and suggest the generally poor nutritional status of such individuals, rather than a specific micronutrient problem. In particular, levels of vitamin status (ascorbic acid, folate, beta-carotene) consistent with the very low contemporary dietary intakes of fruit and vegetables have been described frequently (41).

Social Determinants of Indigenous Health

A range of social determinants underpin the poor nutritional health status of indigenous Australians. These include poverty, disrupted family and community cohesion, social marginalisation, stress, lower levels of education, unemployment, inadequate and overcrowded housing, inadequate sanitation, water supplies and hygiene, limited access to transport, and discrimination (68, 50, 3, 57, 7, 30). Cultural factors may have both positive and negative influence on health and nutritional status. The relationship between social environment and poor health
operates in both directions; poor health can increase the risks of deprivation through stigma and reduced earning capacity (48).
Indigenous Australians now comprise 2.1% of the population (3). Compared with the Australian population as a whole, indigenous Australians are younger (median age 20.1 years compared to 34.0 years), live mainly in south-eastern Australia, are less likely to have post-school education (11% compared with 31%), have higher unemployment rates (23% compared with 9%), have a lower financial income (average weekly income for men $189 compared with $415 and for women $190 compared with $224), are less likely to own homes (31% compared with 71%) (3, 4). Aboriginal and Torres Strait Islander Australians are more likely to live outside metropolitan areas than other Australians, with twice the proportion of indigenous people (32%) living in rural areas of less than 1000 people than non-indigenous people (15%) (36). All Australians living in non-metropolitan areas experience higher mortality rates than those in metropolitan areas: 15% higher for men and 9% higher for women in 1996 (8). The higher morbidity and mortality rates described in rural areas are likely to be related to limited occupational and educational opportunities and the effect on income, than any special attributes of the physical environment. Additional factors include poor access to medical services and limited lifestyle choices (68). In particular, people in rural and remote areas pay up to 50% more for basic healthy food than those living in urban and metropolitan regions (45, 12, 14, 66, 82).

A recent Queensland study has assessed this disparity by remoteness/accessibility as measured by Accessibility/Remoteness Index of Australia (ARIA) category (26). In contrast to expectations, fruit and vegetables were less affected by remoteness/accessibility than other food groups, with the price of meat and meat alternatives and dairy food groups being the most affected (69). The cost of tobacco and take-away food items was less affected by remoteness/accessibility than other items. Basic food items were less available in the more remote stores, as were fresh vegetables and fruits and better nutritional choices (69).

**Health Aspects of Traditional Diets and Lifestyles**

In contrast to the current health crisis, all available evidence suggests that traditionally, Australian indigenous peoples were fit and healthy (24, 55, 62, 41, 56, 82).

Traditional dietary intake and associated lifestyle have been reviewed in detail recently and are summarized below (58). Additional information regarding food collection, preparation, storage, and distribution is also available (58).

On the basis of available information, the traditional diet was generally low in energy density but high in nutrient density, being high in protein, low in sugars, high in complex carbohydrate of low glycaemic index and high in micronutrients. Even though the traditional Aboriginal diet contained a high proportion of animal foods, it would have been low in total fat, extremely low in saturated fat and relatively high in polyunsaturated fatty acids including the long-chain highly polyunsaturated fatty acids of both the omega-3 and omega-6 families, and hence protective against cardiovascular disease and related conditions.
The composition of most traditional vegetable foods is typical of uncultivated plants worldwide, being high in fibre and relatively high in protein with a generally low energy density (17). The carbohydrate in most traditional plant foods is of low glycaemic index, producing lower glucose and insulin levels than similar Western foods, and may be protective against diabetes (16).

Although some animal foods, such as witchetty grubs (Cossidae sp.) and green ants (Oecophylla smaragdina), have a relatively high fat content, most native land animals are very lean (17). Traditional meat foods have a much lower carcass fat content and intramuscular lipid content than meat from domesticated animals, such as cattle and sheep (55). Most carcass fat is stored in discrete depots within the abdomen. These fat depots tend to be small and were traditionally shared by many people. However marine animals, such as turtle and dugong, tend to be high in fat (17, 37). Chewing bones of animal and marine foods (58) would have provided calcium.

Energy expenditure was high. Several accounts highlight the labour-intensive aspects of collecting and preparing traditional foods (75, 76, 27). Food procurement and preparation by Aboriginal hunter-gatherers were energy-intensive processes that could involve sustained physical activity for many hours. Activities included walking long distances, digging for tubers, digging for reptiles, eggs, honey ants, and witchetty grubs, chopping with a stone axe, winnowing and grinding of seeds, digging pits for cooking large animals, and gathering wood for fires (62, 41).

Children were traditionally breast-fed until approximately three years of age, the age of weaning depending on the arrival of another sibling. Solids were not introduced until eruption of teeth (49, 33).

Traditional Aboriginal diet and food preferences

Aboriginal people successfully adopted a hunter-gatherer lifestyle across widely different geographical and climatic conditions until European occupation. Survival depended on intimate knowledge of the land, sources of water, and the detailed effects of the seasonal cycles on plant foods and game (58).

The traditional diet was characterised by diversity and most early observers describe a varied and ample range of both animal and plant foods, even in dry inland regions. On a day-to-day basis, both quantity and quality of the food intake varied greatly; the usual pattern of subsistence was supplemented by ‘feasts’ when large game were successfully hunted.

The most highly prized components of the Aboriginal hunter-gatherer diet were the relatively few energy-dense foods. These included depot fat and organ meats. Other favored foods included those with a high fat content such as witchetty grubs (Cossidae sp) and marine mammals. Traditional diets were generally low in sugars, although sweetness was highly valued and provided by honey ants (Melophorus inflatus), the honey of the native bee, blossoms (eg Grevillea sp), lerp (secretion from the insect Psylla living on the leaves of Eucalyptus sp) and gums (62, 41, 28).
Traditional Torres Strait Islander diet and food preferences

Torres Strait Islanders were marine hunting, but the cultivation of garden foods and gathering of wild foods was also practiced to varying degrees depending on the local habitat (13). Some garden foods were stored and preserved. Turtle and dugong occupied a particular place in the cultural life of the Torres Strait and their fat content was considered a principal indicator of meat quality and was particularly prized (59).

Contemporary diet

The process of acculturation from traditional to contemporary diet and lifestyle, including the effect of social, political and environmental factors, has been described in detail (58, 41). With the transition from a traditional hunter-gather lifestyle to a settled westernised existence, Aboriginal and Torres Strait Islander diet has changed generally from a varied, nutrient-dense diet to an energy-dense diet, high in fat and refined sugars (Table 1).

The limited data available support the notion that contemporary indigenous diets tend to reflect dietary intake of wider Australia both during the depression years of the 1930s (5) and during colonial times (78) when food supply, transport, storage and costs were issues of general concern.

Available dietary studies were recently reviewed in Chapter 3 of Nutrition in Aboriginal and Torres Strait Islander Peoples: an Information paper (58).

Table 1 Characteristics of hunter-gatherer and western lifestyles

<table>
<thead>
<tr>
<th></th>
<th>Hunter-gatherer lifestyle</th>
<th>Western lifestyle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical activity level</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>Principal characteristics of diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy density</td>
<td>low</td>
<td>high</td>
</tr>
<tr>
<td>Energy intake</td>
<td>usually adequate</td>
<td>excessive</td>
</tr>
<tr>
<td>Nutrient density</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>Nutrient composition of diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>high</td>
<td>low-moderate</td>
</tr>
<tr>
<td>Animal</td>
<td>high</td>
<td>moderate</td>
</tr>
<tr>
<td>Vegetable</td>
<td>low-moderate</td>
<td>low</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>moderate (slowly digested)</td>
<td>high (rapidly digested)</td>
</tr>
<tr>
<td>Complex carbohydrate</td>
<td>moderate</td>
<td>moderate</td>
</tr>
<tr>
<td>Simple carbohydrate</td>
<td>usually low (honey)</td>
<td>high (sucrose)</td>
</tr>
<tr>
<td>Dietary fibre</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>Fat</td>
<td>low</td>
<td>high</td>
</tr>
<tr>
<td>Alcohol</td>
<td>not available</td>
<td>available</td>
</tr>
<tr>
<td>Sodium:potassium ratio</td>
<td>low</td>
<td>high</td>
</tr>
</tbody>
</table>

Source: based on (41, 62).

\[1\] much of the information pertaining to the Torres Strait has been provided by Dympna Leonard
Urban Aboriginal communities

Little quantitative dietary data are available pertaining to the urban setting (19). Due to methodological difficulties, individual dietary studies have tended to focus on qualitative and semi-quantitative assessment of the diet and reflect dietary patterns and preferences, rather than actual, habitual intake (32).

A comparison of the food habits of Aboriginal and non-Indigenous Australians in a city and a country town showed that in both localities indigenous groups consumed take-away meals more frequently and used added salt more frequently than their non-Indigenous counterparts (32). 24-hr dietary recall data from a small number of individuals suggested little difference between dietary intake of indigenous and non-indigenous Australians in a country town, but these data have not been validated (72) and highlight some methodological considerations (44).

Remote Aboriginal communities

In remote Aboriginal communities in the Northern Territory, a study using the ‘store-turnover’ method showed that sugar, flour, bread and meat provided more than half the apparent total energy intake. Fatty meats contributed nearly 40 per cent of the total fat intake in northern coastal communities and over 60 per cent in central desert communities. In both regions, white sugar per se contributed approximately 60 per cent of all sugars consumed (42). Compared with national Australian apparent consumption data, intakes of sugar, white flour and sweetened carbonated beverages were much higher in Aboriginal communities in the Northern Territory and intakes of wholemeal bread, fruit and vegetables were much lower (42) (Table 2).

Table 2  Apparent mean consumption of selected foods in Aboriginal communities compared with national data (kg per capita per year)

<table>
<thead>
<tr>
<th>Food</th>
<th>Aboriginal Communities</th>
<th>Australian data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Central Desert (n=3)</td>
<td>Northern Coastal (n=3)</td>
</tr>
<tr>
<td>Flour (white)</td>
<td>37.6</td>
<td>44.4</td>
</tr>
<tr>
<td>Bread (all)</td>
<td>34.1</td>
<td>30.5</td>
</tr>
<tr>
<td>Beef and veal</td>
<td>51.6</td>
<td>25.8</td>
</tr>
<tr>
<td>Poultry</td>
<td>22.3</td>
<td>19.7</td>
</tr>
<tr>
<td>Lamb</td>
<td>22.8</td>
<td>3.3</td>
</tr>
<tr>
<td>Fish</td>
<td>0</td>
<td>4.8</td>
</tr>
<tr>
<td>Fruits</td>
<td>33.2</td>
<td>17.6</td>
</tr>
<tr>
<td>Vegetables</td>
<td>24.3</td>
<td>19.6</td>
</tr>
<tr>
<td>Sugar</td>
<td>54.1</td>
<td>50.3</td>
</tr>
<tr>
<td>Carbonated beverages</td>
<td>67.9</td>
<td>224.</td>
</tr>
<tr>
<td>Fruit juice</td>
<td>48.3</td>
<td>12.8</td>
</tr>
<tr>
<td>Tinned meat</td>
<td>9.4</td>
<td>10.1</td>
</tr>
<tr>
<td>Pie/Pastie</td>
<td>9.6</td>
<td>15.1</td>
</tr>
<tr>
<td>Snack Foods (eg potato crisps)</td>
<td>1.8</td>
<td>2.7</td>
</tr>
</tbody>
</table>
Nutritional analysis revealed the average diet was high in energy and sugar (more than 3 times recommended intake), moderately high in fat, particularly saturated fat, and relatively low in complex carbohydrate, dietary fibre and nutrient density, supporting qualitative and semi-quantitative dietary assessment (35, 25, 23). The intake of some minerals (calcium and zinc) and some vitamins (vitamin B2, vitamin E, beta-carotene and folic acid) appear low (43). These results have been confirmed by subsequent studies in different communities (51, 81, 29).

In contrast, very low energy intakes were described in one anthropological study in very remote Aboriginal outstation communities in north-east Arnhem Land (47). The energy intake of all subjects was approximately 50 per cent of the Australian recommended dietary intake, and the vitamin C, retinol activity, magnesium and calcium intakes were low. Where traditional bushfoods (predominantly of animal origin) were consumed, zinc and iron intakes were higher and iron intake was equal to or above the recommended dietary intake. Low intakes of fruit and vegetables were also described in this study (47).

Dietary intake has been shown to vary in close association with the income cycle in remote Aboriginal communities (25). Meat and vegetables (mainly as stew) and fruits were included in the diet after pay day but were usually absent for at least several days before the next pay day. A staple diet of bread or damper has been described frequently (25, 39).

Even in remote traditionally orientated outstations, foods purchased at the store accounted for most of the energy intake while traditional bushfoods provided the greatest proportion of protein intake (52, 6, 27). All available studies showed that flour, sugar, sweets and fats provided much of the energy intake from store-purchased foods. Animal foods, particularly those high in fat such as lizards, provided most of the energy from the bush. In general, dietary patterns in these small traditionally-orientated communities are meat-orientated.

Torres Strait Island communities

In the Torres Strait (and also in a few other island communities, such as Tiwi) marine foods continue to make substantial contributions to the diet. Men, women and children are involved in different aspects of gleaning, fishing and hunting. Torres Strait Islanders living on three outer islands, were estimated to consume 191 g to 450 g per person per day of seafood (including turtle and dugong), considerably more than Japanese seafood intake (102 g per day) (37). However concerns have been raised about the issue of heavy metal content in the organ meat of dugong and particularly of turtle and the potentially negative impact of these on health (34). Production of traditional garden staple foods continues to be important for some ceremonial purposes (45).

A study using the ‘store-turnover’ method was undertaken in a small island community in the Torres Strait. More than half the energy in the diet came from white flour, white rice,
tinned meat and vegetable oil. The amount of fruit and vegetables available through the
store was low. Fruit available per person was about one-sixth of the amount recommended
and vegetables about one-third of the amount recommended. People who depended on
store foods would not meet their needs for vitamins A, C, and E and folic acid (45).

Contemporary Use of Traditional Foods

It had been suggested that in the short-term, after establishment of ‘new’ settlements or
outstations, there may be an increase in yields of traditional foods due to high initial
availability and the use of Western technology (74, 25, 6). In some areas, introduced feral
animals such as rabbits and buffalo were also popular (21, 6). However, the longer-term
effect appears to be a reduction in the availability of traditional foods due to several
factors. These include the effect of environmental degradation caused by stock and feral
animals, the introduction of exotic plant species, the increasing incidence of hot,
destructive bush fires due to poor rural land management practices, the restricted access to
some areas of land, depletion of resources and population pressure around permanent
settlements, high costs associated with the acquisition and maintenance of equipment,
fires, vehicles, and fuel, changing demographic patterns and cultural loss from
generation to generation (64, 22).

Contrary to some expectations, actual dietary intake of traditional foods is relatively low
where measured on mainland Australia (39, 40) except for during some seasons in very
remote areas (27, 52, 6, 47). Bushfoods contribute only a small proportion of nutrients in
many areas (43). In a study of a northern coastal Aboriginal community an average of less
than 15 per cent of the population sought traditional foods at least three days per fortnight
throughout the year. It was estimated that the proportion of total energy intake derived
from bushfoods averaged over the population would be less than 8 per cent during the dry
season and less than half this proportion during the peak of the wet season (40).

In the NATSIS, 10 per cent of people aged over 14 years reported spending more than one
hour per week hunting or foraging for traditional foods, and of these, more than half
reported spending more than five hours per week doing this (2). However, the rate of
return for this effort is unknown. Even though the actual intake of traditional foods is low,
traditional foods are still popular and culturally important for both Aboriginal and Torres
Strait Islander peoples (70, 45).

Dietary Guidelines for Indigenous peoples

Proposed guidelines
Choose store-foods which are most like traditional bushfoods
Enjoy traditional bushfoods whenever possible

Rationale
Indigenous people may be assisted in the selection of nutritious food by encouraging the
consumption of store-foods which are most like traditional bush foods, ie fresh plant
foods, wholegrain cereal foods, seafoods, lean meat and poultry. This approach is
generally consistent with the Australian Dietary Guidelines, for all but the dairy food
group (see below).
All available evidence suggests that the health and well-being of indigenous Australians would benefit from closer adherence to the general Australian Dietary Guidelines (53, 67, 60, 43, 79, 80, 63, 81). In particular, the increased consumption of both vegetables and fruits would be expected to enhance the health and nutritional status of this population group. However, lactose intolerance after the age of three to five years may be problematic in some areas/individuals (15, 20) and may affect tolerance of consumption of lactose-containing dairy foods. Alternative calcium sources such as low lactose dairy foods (matured cheese, yoghurt), chewing meat and fish bones and consumption of small soft fish bones (eg tinned salmon) may be recommended in these cases.

The consumption of traditional bushfoods should also be supported wherever possible. However, this may be a nutritional issue where high-fat marine animal foods are hunted in large numbers with modern procurement and distribution (37). In addition there may be an issue of high levels of heavy metals in the organ meat of turtle and dugong (34). Several environmental issues may also need to be considered within the framework of sustainability.

Where non-indigenous Australian have sufficient understanding of the traditional indigenous food supply, these guidelines may also be useful in a wider context.
REFERENCES


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SOCIAL STATUS, NUTRITION AND THE COST OF HEALTHY EATING

Dietary differences across social status groups

There is no doubt that a social gradient exists for many diet-related chronic disease conditions in Australia but, with the exception of extreme poverty conditions, it is unclear what role dietary differences across social groups plays in the Australian context in terms of chronic disease outcome or nutrition status.

The 1995/6 National Nutrition Survey (1,2) showed few systematic differences in food and nutrient intake across quintiles of social disadvantage as defined by the SEIFA index based on the 1991 census. This index assesses relative social disadvantage of respondents based on their area of residence as assessed by economic resources, education and occupation patterns in that area.

Table 1 shows the intake of various food groups across SEIFA quintiles, with the first quintile living in the most disadvantaged areas.

In summary, for the major food groups:

- Consumption of cereals and cereal based foods (eg rice, pasta, breads) were somewhat lower in the most disadvantaged group and the middle group compared to all other groups with cereal-based products and dishes (eg cakes, biscuits) also being about 20% lower in the two most disadvantaged groups compared to the other three.

- Consumption of fruit and fruit products was also lower in the most disadvantaged group compared to the other four groups (10-20%) but vegetable and legume consumption showed no consistent trend across the groups.

- Milk and milk product consumption increased slightly with social advantage (about 10% increase across the groups)

- Meat, poultry and game consumption was slightly higher in the middle quintiles of disadvantage but fish and seafood consumption gradually increased with social advantage.
Table 1. Mean daily intakes (g/day) for persons aged 19yrs and over by SEIFA.

<table>
<thead>
<tr>
<th>Food group</th>
<th>1st SEIFA quintile (greatest disadvantage)</th>
<th>2nd SEIFA quintile</th>
<th>3rd SEIFA quintile</th>
<th>4th SEIFA quintile</th>
<th>5th SEIFA quintile (least disadvantaged)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cereal &amp; cereal products</td>
<td>196</td>
<td>222</td>
<td>203</td>
<td>217</td>
<td>232</td>
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<tr>
<td>Cereal based products &amp; dishes</td>
<td>113</td>
<td>115</td>
<td>130</td>
<td>135</td>
<td>136</td>
</tr>
<tr>
<td>Fruit products and dishes</td>
<td>126.2</td>
<td>147.2</td>
<td>141</td>
<td>143</td>
<td>156</td>
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<tr>
<td>Vegetable products &amp; dishes</td>
<td>264</td>
<td>258</td>
<td>260</td>
<td>262</td>
<td>251</td>
</tr>
<tr>
<td>Legumes &amp; pulses</td>
<td>9.8</td>
<td>7.9</td>
<td>10.8</td>
<td>9.5</td>
<td>10.7</td>
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<tr>
<td>Milk products &amp; dishes</td>
<td>281</td>
<td>284</td>
<td>285</td>
<td>292</td>
<td>301</td>
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<tr>
<td>Meat, poultry, game</td>
<td>149</td>
<td>163</td>
<td>164</td>
<td>155</td>
<td>158</td>
</tr>
<tr>
<td>Fish &amp; seafood</td>
<td>22</td>
<td>24.5</td>
<td>26.3</td>
<td>25.8</td>
<td>28.8</td>
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<tr>
<td>Egg products &amp; dishes</td>
<td>16</td>
<td>15</td>
<td>16</td>
<td>13</td>
<td>19</td>
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<tr>
<td>Snack foods</td>
<td>3.2</td>
<td>3.0</td>
<td>4.2</td>
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<td>3.9</td>
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<tr>
<td>Sugar products &amp; dishes</td>
<td>20</td>
<td>21</td>
<td>20</td>
<td>18</td>
<td>17</td>
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<tr>
<td>Confectionery</td>
<td>7.8</td>
<td>9.2</td>
<td>8.3</td>
<td>9.3</td>
<td>9.1</td>
</tr>
<tr>
<td>Seeds &amp; nuts</td>
<td>3.6</td>
<td>4.1</td>
<td>5.2</td>
<td>4.0</td>
<td>4.8</td>
</tr>
<tr>
<td>Fats &amp; oils</td>
<td>12</td>
<td>13</td>
<td>13</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>Soup</td>
<td>53</td>
<td>62</td>
<td>55</td>
<td>48</td>
<td>57</td>
</tr>
<tr>
<td>Savoury sauces &amp; condiments</td>
<td>30</td>
<td>30</td>
<td>28</td>
<td>30</td>
<td>29</td>
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<tr>
<td>Alcoholic beverages</td>
<td>239</td>
<td>254</td>
<td>273</td>
<td>270</td>
<td>234</td>
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Table 2. Mean daily intakes of energy and nutrient densities for persons aged 19yrs and over by SEIFA

<table>
<thead>
<tr>
<th>Nutrient density (nutrient /10MJ energy)</th>
<th>1st quintile SEIFA</th>
<th>2nd quintile SEIFA</th>
<th>3rd quintile SEIFA</th>
<th>4th quintile SEIFA</th>
<th>5th quintile SEIFA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy MJ/day</td>
<td>8.82</td>
<td>9.18</td>
<td>9.37</td>
<td>9.31</td>
<td>9.45</td>
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<tr>
<td>Protein g/10MJ</td>
<td>98.2</td>
<td>98.4</td>
<td>98.5</td>
<td>98.6</td>
<td>99.4</td>
</tr>
<tr>
<td>Fat g/10MJ</td>
<td>89.8</td>
<td>90.7</td>
<td>91.1</td>
<td>88.9</td>
<td>88.8</td>
</tr>
<tr>
<td>Saturated fat g/10MJ</td>
<td>35.7</td>
<td>35.5</td>
<td>36.0</td>
<td>35.2</td>
<td>35.0</td>
</tr>
<tr>
<td>Monounsaturated fat g/10MJ</td>
<td>32.5</td>
<td>32.8</td>
<td>33.4</td>
<td>32.4</td>
<td>32.3</td>
</tr>
<tr>
<td>Polyunsaturated fat g/10MJ</td>
<td>13.5</td>
<td>13.5</td>
<td>13.9</td>
<td>13.4</td>
<td>13.4</td>
</tr>
<tr>
<td>Cholesterol mg/10MJ</td>
<td>332</td>
<td>331</td>
<td>332</td>
<td>319</td>
<td>305</td>
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<tr>
<td>Total carbohydrate g/10MJ</td>
<td>276</td>
<td>277</td>
<td>272</td>
<td>276</td>
<td>277</td>
</tr>
<tr>
<td>Sugars g/10MJ</td>
<td>128</td>
<td>125</td>
<td>123</td>
<td>124</td>
<td>123</td>
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<tr>
<td>Starch g/10MJ</td>
<td>147</td>
<td>150</td>
<td>148</td>
<td>151</td>
<td>152</td>
</tr>
<tr>
<td>Fibre g/10MJ</td>
<td>24.4</td>
<td>24.4</td>
<td>24.9</td>
<td>25.2</td>
<td>25.6</td>
</tr>
<tr>
<td>Alcohol g/10MJ</td>
<td>13.4</td>
<td>13.3</td>
<td>14.3</td>
<td>14.6</td>
<td>13.8</td>
</tr>
<tr>
<td>Vit A ug/10MJ</td>
<td>1280</td>
<td>1299</td>
<td>1236</td>
<td>1218</td>
<td>1329</td>
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<tr>
<td>Thiamin mg/10MJ</td>
<td>1.81</td>
<td>1.74</td>
<td>1.81</td>
<td>1.83</td>
<td>1.80</td>
</tr>
<tr>
<td>Riboflavin mg/10MJ</td>
<td>2.27</td>
<td>2.18</td>
<td>2.24</td>
<td>2.25</td>
<td>2.22</td>
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<tr>
<td>Niacin mg/10MJ</td>
<td>45.8</td>
<td>45.9</td>
<td>45.6</td>
<td>45.5</td>
<td>45.9</td>
</tr>
<tr>
<td>Folate ug/10MJ</td>
<td>289</td>
<td>286</td>
<td>299</td>
<td>272</td>
<td>292</td>
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<tr>
<td>Vit C mg/10MJ</td>
<td>132</td>
<td>131</td>
<td>130</td>
<td>135</td>
<td>142</td>
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<tr>
<td>Calcium mg/10MJ</td>
<td>907</td>
<td>888</td>
<td>900</td>
<td>926</td>
<td>945</td>
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<tr>
<td>Phosphorus mg/10MJ</td>
<td>1626</td>
<td>1631</td>
<td>1630</td>
<td>1654</td>
<td>1673</td>
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<tr>
<td>Magnesium mg/10MJ</td>
<td>353</td>
<td>356</td>
<td>354</td>
<td>361</td>
<td>366</td>
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<tr>
<td>Iron mg/10MJ</td>
<td>15.1</td>
<td>15.0</td>
<td>15.3</td>
<td>15.4</td>
<td>15.6</td>
</tr>
<tr>
<td>Zinc mg/10MJ</td>
<td>12.9</td>
<td>13.07</td>
<td>12.8</td>
<td>13.0</td>
<td>13.3</td>
</tr>
<tr>
<td>Potassium mg/10MJ</td>
<td>3541</td>
<td>3495</td>
<td>3507</td>
<td>3528</td>
<td>3551</td>
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</tbody>
</table>
An assessment of nutrient intake across quintiles of social disadvantage (Table 2) showed that energy intake increased with social advantage as did intake of most nutrients. However, when correcting for energy differences across groups few differences were apparent in dietary quality defined as nutrient intake per unit energy. It is unclear from the published data whether there are other factors such as the age profile which differed across these quintiles of disadvantage and how much variation in factors such as age, which are known to influence total food intake, might account for such differences as were apparent (e.g., total energy intake).

Neither is it clear whether these relatively small differences in nutrient profiles could explain a significant proportion of the variation in health profile across the groups. In interpreting this data set however, it should be borne in mind that a relatively crude, area-based measure of social disadvantage was used and it is also possible that many of the most disadvantaged individuals in the community may not have taken part in the survey.

**The cost of healthy eating**

In recent years, most countries in the western world, including Australia, have developed dietary guidelines and recommendations such as those outlined in this document, aimed at improving health and nutritional status and preventing chronic diseases such as coronary heart disease, certain cancers, hypertension and diabetes. However, if the changes encouraged by these guidelines are financially costly there will be groups, such as low income families, in the community that are unable, or unwilling, to make these adjustments.

Although speculation about the expense of healthful eating is widespread, few cost analyses of healthful diets have been reported. One study in the United Kingdom compared the cost of one-day diets that did and did not conform with the British dietary guidelines (3). They found that following the guidelines was potentially more expensive. In Australia, Santich looked at the eating patterns recommended in various federal government nutrition education publications and concluded that the changes recommended may not be financially realistic for low income families to follow (4). However, this conclusion was based on an analysis of specific recipes given in the pamphlets and on the substitution models for healthful eating used in the education materials (e.g., the substitution of lean, minced beef for fattier versions, or the use of wholegrain breads instead of white, processed bread). Crowley (1992), using a qualitative sociological analysis of the issues has also suggested that the "cost" of a diet that follows the dietary guidelines is potentially higher compared with the cost of the average Australian diet (5). His analysis was qualitative, and included issues outside the direct financial cost of food as purchased, such as access and transportation.

In the early 1990s, McAllister et al (6) undertook an analysis based on Australian data, designed to determine if it was necessarily more directly, financially costly to follow a healthful diet in Australia. Three different approaches were used: a substitution approach, a relative costing of individual diets that are in line with the dietary guidelines and an analysis of the cost of eating according to a healthy eating plan, the 12345+ Nutrition Plan, developed by the CSIRO (7). This healthy eating plan was highly flexible. It was designed to produce diets conforming to both the existing Australian dietary guidelines and the Recommended Dietary Intakes for people with varying energy needs and/or special

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nutrient needs (e.g. pregnancy, lactation, adolescence) and from a variety of cultural backgrounds.

The cost of 229 foods and drinks used in the analyses was determined by assessing prices in four major supermarkets and other food outlets, such as take-away stores, when necessary. The food outlets used for pricing were located within a lower socioeconomic suburban area of Adelaide, where there is a large concentration of low income families (8). In each supermarket, for each food or drink, the cheapest branded item (items with the manufacturer’s brand name) was recorded, together with the price of the equivalent generic item (items without the manufacturer's brand name - commonly associated with a particular retailer).

An analysis of the potential cost of direct substitution of healthful alternatives for less healthful food choices (eg product-by-product substitution through the use of fat, fibre or salt-modified alternatives, etc.) indicated that this approach would result in a more costly diet, while providing limited nutritional improvement at the population level. Pricing of self-selected diets of people whose diets currently comply with the dietary guidelines and targets for healthful eating showed that these people are currently paying more per megajoule for their diet. However, costing of diets that conformed to a new healthful eating plan, designed to produce eating patterns that meet both the dietary goals set for components of the food supply such as dietary fat, refined sugars or fibre and the recommended daily intakes for energy, protein, vitamins and minerals, showed that healthful eating need not be more expensive and indeed, for most people, would bring cost savings.

In summary, this study showed that "healthful" eating is not necessarily more expensive, but that restructuring the diet rather than using a direct substitution approach is the more cost effective strategy. Education programs that stress this approach and its cost advantages are therefore more likely to be successful in promoting an affordable and effective healthful alternative for people with limited financial means.

Unfortunately, it is generally easier for people to understand and adopt a substitution approach rather than it is to basically restructure their diets. They also receive encouragement to adopt this substitution approach to healthful eating not only from food manufacturers who, understandably, wish to promote specific "healthful" products, but also from much of the educational literature designed by health professionals.

The study described above was designed to look only at the potential financial costs of healthful eating. The study showed that the theoretical cost savings relating to purchasing healthier diets would be similar across all social groups in Australia. However, there are obviously other factors that may make a healthful diet more difficult to achieve, such as the ready availability of healthful food choices, skills, facilities, time, taste factors and motivation. These factors may indeed vary across social, educational and income groups. These aspects have been previously discussed by Santich (4) and Crowley (5), but there is little data available and this would be a valuable area for further investigation, especially with respect to low income groups in the community. These issues, however, will need to be addressed in implementing the Dietary Guidelines in socially disadvantaged groups and will be addressed in the Implementation Plan for the Dietary Guidelines.
### Financial costs of healthy eating

#### 1989 ABS Household Expenditure Survey

$4.92

#### 1990 Victorian/SA Surveys

- **current cost,**
  - **Generic** $4.58
  - **Branded** $4.99
- **conformers to dietary guidelines**
  - **Generic** $4.60
  - **Branded** $4.96
- **non-conformers with equivalent energy intakes**
  - **Generic** $4.01
  - **Branded** $4.33
- **substitution of "healthy choices" (adjusted for energy content)**
  - **Generic** $5.22
  - **Branded** $5.54

#### Healthy Eating Plan (CSIRO 12345+ Plan)

<table>
<thead>
<tr>
<th></th>
<th>Average woman</th>
<th>Average man</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Generic</strong></td>
<td>$3.48</td>
<td>$3.81</td>
</tr>
<tr>
<td><strong>Branded</strong></td>
<td>$3.76</td>
<td>$4.09</td>
</tr>
</tbody>
</table>
References


5. Crowley S. Will the poor be able to afford a healthy diet? *Nutridate* June 1992; 3: 5-7


