Australian Dietary Guidelines

Incorporating the

Australian Guide to Healthy Eating

Providing the scientific evidence for healthier Australian diets

DRAFT FOR PUBLIC CONSULTATION

National Health and Medical Research Council

December 2011

Preface

Never in our nation's history have Australians had such a wide variety of dietary options. Yet the rising incidence of obesity and diabetes in our population is evidence of the need for Australians to improve their health by making better dietary decisions.

There are many ways for Australians to choose foods that promote their health and wellbeing while reducing their risk of chronic disease. NHMRC's *Australian Dietary Guidelines* provide recommendations for healthy eating that are realistic, practical, and - most importantly - based on the best available scientific evidence.

These *Guidelines* are an evolution of the *2003 Dietary Guidelines*, integrating updates of the *Dietary Guidelines for Older Australians* (1999), the *Dietary Guidelines for Adults (2003)* and the *Dietary Guidelines for Children and Adolescents in Australia* (2003). They also include an update of the *Australian Guide to Healthy Eating* (1998).

Providing the recommendations and the evidence that underpins them in a single volume, the *Guidelines* will help health professionals, policy makers and the Australian public cut through the background noise of ubiquitous dietary advice that is often based on scant scientific evidence. They form a bridge between research and evidence based advice to address the major health challenge of improving Australians' eating patterns.

The evidence for public health advice should be the best available. NHMRC is confident that the available evidence underpinning these guidelines meets that criterion and is stronger than for any previous NHMRC dietary guideline.

NHMRC acknowledges that population growth, economic issues and environmental pressures affect food availability and affordability on global, national and regional scales. The interaction between dietary advice, the environment and food production raise cross-sectoral issues including the impact of food choices and future food security. The NHMRC and other Commonwealth agencies are jointly considering these.

For more than 75 years the Australian Government, primarily through NHMRC and Australian Government health departments, has provided nutrition advice to the public through food and nutrition policies, dietary guidelines and national food selection guides.

NHMRC and all involved in developing these *Guidelines* are proud and privileged to have the responsibility to continue this important public service.

Professor Warwick Anderson
Chief Executive Officer
National Health & Medical Research Council

Australian Dietary Guidelines

Australian Dietary Guidelines	
Guideline 1	Eat a wide variety of nutritious foods from these five groups every day: ▪ plenty of vegetables, including different types and colours, and legumes/beans ▪ fruit ▪ grain (cereal) foods, mostly wholegrain, such as breads, cereals, rice, pasta, noodles, polenta, couscous, oats, quinoa and barley ▪ lean meat and poultry, fish, eggs, nuts and seeds, and legumes/beans ▪ milk, yoghurt, cheese and/or their alternatives, mostly reduced fat (reduced fat milks are not suitable for children under the age of 2 years). And drink water.
Guideline 2	Limit intake of foods and drinks containing saturated and trans fats, added salt, added sugars and alcohol. a. Limit intake of foods and drinks containing saturated and trans fats ▪ Include small amounts of foods that contain unsaturated fats ▪ Low-fat diets are not suitable for infants. b. Limit intake of foods and drinks containing added salt ▪ Read labels to choose lower sodium options among similar foods. ▪ Do not add salt to foods. c. Limit intake of foods and drinks containing added sugars. In particular, limit sugar-sweetened drinks. d. If you choose to drink alcohol, limit intake.
Guideline 3	To achieve and maintain a healthy weight you should be physically active and choose amounts of nutritious food and drinks to meet your energy needs. ▪ Children and adolescents should eat sufficient nutritious foods to grow and develop normally. They should be physically active every day and their growth should be checked regularly. ▪ Older people should eat nutritious foods and keep physically active to help maintain muscle strength and a healthy weight.
Guideline 4	Encourage and support breastfeeding.
Guideline 5	Care for your food; prepare and store it safely.

Contents

1. Introduction

1.1 Why the *Guidelines* matter

There are many ways for Australians to achieve dietary patterns that promote health and wellbeing and reduce the risk of chronic disease. Diet is arguably the single most important behavioural risk factor that can be improved to have a significant impact on health [1, 2]. As the quality and quantity of foods and drinks consumed has a significant impact on the health and wellbeing of individuals, society and the environment, better nutrition has a huge potential to improve individual and public health and decrease healthcare costs. Optimum nutrition is essential for the normal growth and physical and cognitive development of infants and children. In all Australians, nutrition contributes significantly to healthy weight, quality of life and wellbeing, resistance to infection, and protection against chronic disease and premature death.

Sub-optimal nutrition can be associated with ill-health. Many diet-related chronic diseases such as cardiovascular disease, type 2 diabetes and some forms of cancer are the major cause of death and disability among Australians [3]. More than one-third of all premature deaths in Australia are the result of chronic diseases that could have been prevented [3]. Many of these are mediated by overweight and obesity.

Poor nutrition is responsible for around 16% of the total burden of disease [1, 4] and is implicated in more than 56% of all deaths in Australia [5]. The most recent available estimates for the total cost of poor nutrition were more than $5 billion per year, based on 1990 costings [5]. Given that the cost of obesity alone was estimated to be $8.283 billion per year in 2008 [6], the current cost of poor nutrition in Australia is now likely to greatly exceed the 1990 estimates.

Most of the burden of disease due to poor nutrition in Australia is associated with excessive intake of energy-dense and relatively nutrient-poor foods high in energy (kilojoules), saturated fat, added or refined sugars or salt, and/or inadequate intake of nutrient-dense foods, including vegetables, fruit and wholegrain cereals [2, 7]. Deficiency in some nutrients such as iodine, folate [8], iron and vitamin D is also of concern for some Australians [9, 10].

Overconsumption of some foods and drinks, leading to excess energy intake and consequent overweight and obesity, is now a key public health problem for Australia [7, 11]. The prevalence of overweight and obesity has increased dramatically in Australia over the past 30 years and is now 62% in adults [12] and around 25% in children and adolescents [12, 13].

These *Guidelines* summarise the evidence underlying food, diet and health relationships that improve public health outcomes.

Dietary patterns consistent with the *Guidelines* improve health

Recent reviews of the evidence on food and health confirm that dietary patterns consistent with the *Guidelines* are positively associated with indicators of health and wellbeing.

Two systematic reviews found that higher dietary quality was consistently associated with a 10–20% reduction in morbidity. For example, there is evidence of a probable association between consumption of a Mediterranean dietary pattern and reduced mortality (Grade B, Section 20.1 in Evidence Report [14]) [15-17]. Previous studies have also indicated inverse associations between plant-based diets and all-cause and cardiovascular mortality, particularly among older adults [18-20]. The effects of dietary quality tended to be greater for men than women, with common determinants being age, education and socioeconomic status [21, 22].

There is likely to be great variation in the interpretation and implementation of dietary guidelines. Nevertheless, when a wide range of eating patterns was assessed for compliance with different guidelines using a variety of qualitative tools, the assessment suggested an association between adherence to national dietary guidelines and recommendations, and reduced morbidity and mortality (Grade C, Section 20.3 in Evidence Report [14]) [21, 22].

More recent evidence from Western societies confirms that dietary patterns consistent with current guidelines recommending relatively high amounts of vegetables, fruit, whole grains, poultry, fish, and reduced fat milk, yoghurt and cheese products may be associated with superior nutritional status, quality of life and survival in older adults [23, 24]. Robust modelling of dietary patterns in accordance with dietary guidelines has demonstrated achievable reductions in predicted cardiovascular and cancer disease mortality in the population, particularly with increased consumption of fruit and vegetables [25].

In relation to obesity, actual dietary recommendations and measures of compliance and weight outcomes vary greatly in published studies. Overall energy intake is the key dietary factor affecting weight status (see Chapter 4).

1.2 Social determinants of food choices and health

Life expectancy and health status are relatively high overall in Australia [12, 26]. Nonetheless, there are differences in the health and wellbeing between Australians, including in rates of death and disease, life expectancy, self-perceived health, health behaviours, health risk factors, and use of health services [27-29].

The causes of health inequities are largely outside the health system and relate to the inequitable distribution of social, economic and cultural resources and opportunities [27-29]. Employment,

income, education, cultural influences and lifestyle, language, sex and other genetic differences, isolation (geographic, social or cultural), age and disability, the security and standard of accommodation, and the availability of facilities and services all interact with diet, health and nutritional status[27, 28]. Conversely, a person's poor health status can contribute to social isolation and limit their ability to gain employment or education and earn an income, which can in turn impact negatively on health determinants such as quality and stability of housing.

Australians who are at greater risk of diet-mediated poor health include the very young, the very old, Aboriginal and Torres Strait Islander peoples and those in lower socioeconomic groups [27-32]. The *Guidelines* address some of the issues these population groups face under 'Practical considerations for health professionals' in each guideline. Further discussion of the social determinants of health and food choices is provided in Appendix 7.

1.3 Scope and target audience

The *Guidelines*, together with the underlying evidence base, provide guidance on foods, food groups and dietary patterns that protect against chronic disease and provide the nutrients required for optimal health and wellbeing. They are important tools which support broader strategies to improve nutrition outcomes in Australia, as highlighted in *Eat Well Australia: an agenda for action in public health nutrition, 2000-2010* [2]. They are consistent with the most recent Australian *Food and Nutrition Policy* 1992 [33] in considering health and wellbeing, equity and the environment.

The *Guidelines* apply to all healthy Australians

The *Guidelines* aim to promote the benefits of healthy eating, not only to reduce the risk of diet-related disease but also to improve community health and wellbeing. The *Guidelines* are intended for people of all ages and backgrounds in the general healthy population, including people with common diet-related risk factors such as being overweight.

They do not apply to people with medical conditions requiring specialised dietary advice, nor to the frail elderly who are at risk of malnutrition.

The *Guidelines* are based on whole foods

Dietary recommendations are often couched in terms of individual nutrients (such as vitamins and minerals). People chose to eat whole foods not single nutrients, so such recommendations can be difficult to put into practice. For this reason, these *Guidelines* make recommendations based only on whole foods, such as vegetables and meats, rather than recommendations related to specific food components and individual nutrients.

This practical approach makes the recommendations easier to apply. Dietary patterns consistent with the *Guidelines* will allow the general population to meet nutrient requirements, although some subpopulations (for example, pregnant and breastfeeding women) may have some increased nutrient requirements that are more difficult to meet through diet alone. This is noted for each *Guideline* under 'Practical considerations for health professionals'.

For information on specific micro- and macro-nutrients, refer to the *Nutrient Reference Values for Australia and New Zealand* [9].

Issues related to food composition and food supply, such as fortification, use of food additives or special dietary products are dealt with by Food Standards Australia New Zealand (see http://www.foodstandards.gov.au).

Target audience for the Guidelines

The target audience for the *Guidelines* comprises health professionals (including dietitians, nutritionists, general practitioners, nurses and lactation consultants), educators, government policy makers, the food industry and other interested parties. A suite of resources for the general public, including the revised *Australian Guide to Healthy Eating* has also been produced (see www.eatforhealth.gov.au).

Companion documents

The *Guidelines* form part of a suite of documents on nutrition and dietary guidance (see Figure 1.1). Other documents in this suite include:

Nutrient Reference Values for Australia and New Zealand

This details quantitative nutrient reference values (NRVs) for Australians of difference ages and gender. These reference values detail the recommended amounts of nutrients (vitamins, minerals, protein, carbohydrate etc.) required to avoid deficiency, toxicity and chronic disease. As an example, you would refer to the NRVs document to know how much iron is needed by women aged between 19 and 30.

The Food Modelling Document
(A modelling system to inform the revision of the Australian Guide to Healthy Eating)

This describes a range of computer-generated diets that translate the NRVs into dietary patterns to describe the types, combinations and amounts of foods that deliver nutrient requirements for each age and gender group of different physical activity level in the Australian population.

A range of models including omnivore, lacto-ovo vegetarian, pasta and rice-based dietary patterns were developed, and dietary patterns were used to inform the Australian Guide to Healthy Eating.

The Evidence Report
(A review of the evidence to address targeted questions to inform the revision of the Australian dietary guidelines)

This is a systematic literature review relevant to targeted questions published in the peer-reviewed nutrition literature from 2003-2009. This document is described further in Section 1.4. As an example, if you would like to look at the evidence for a particular Evidence Statement, you would refer to the Evidence Report.

The Australian Guide to Healthy Eating

This package of resources includes:

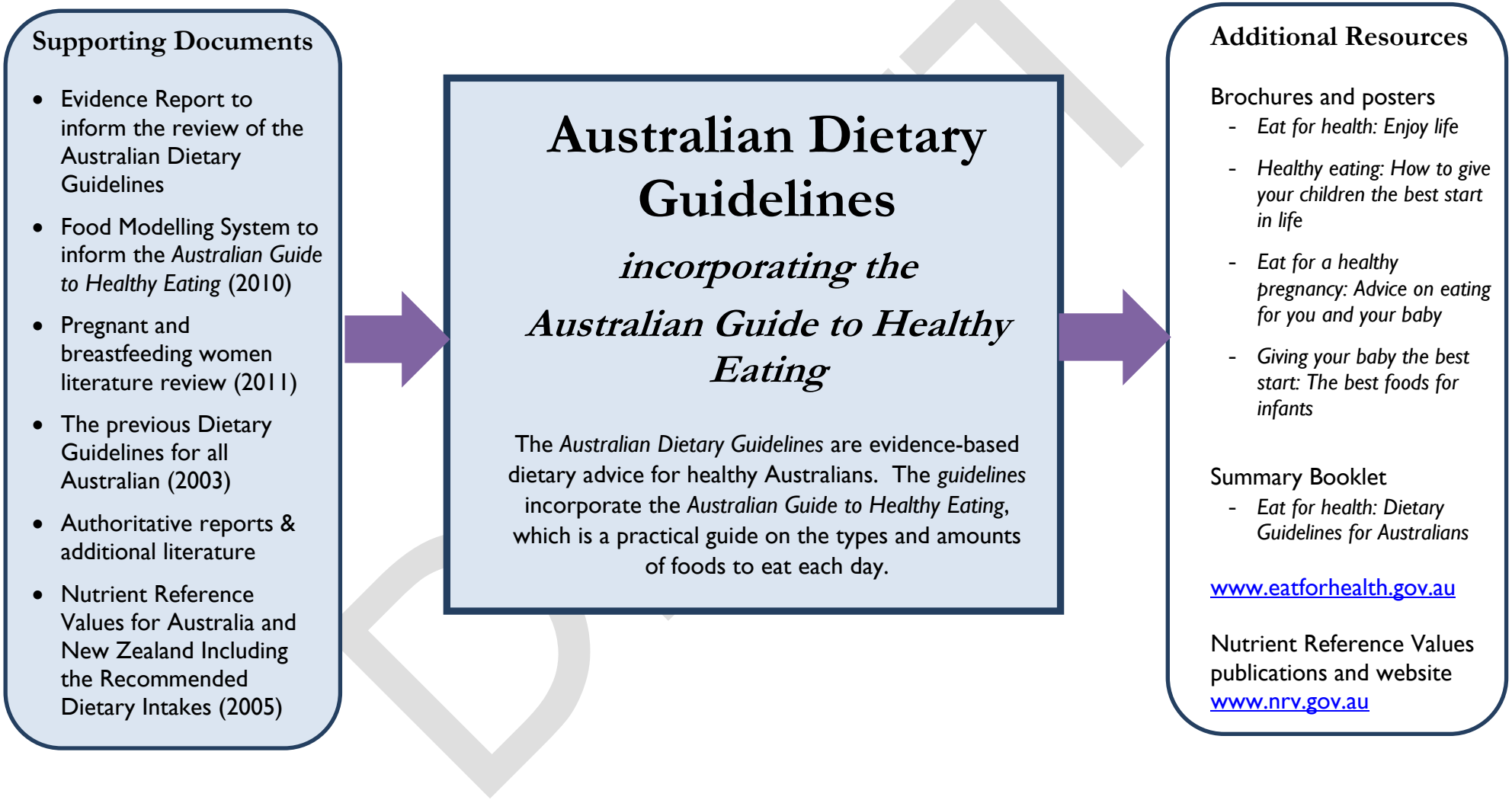
- the 'plate' graphic divided into portions of fruit, vegetables, grains, milk, yoghurt and cheese products and lean meat and alternatives, representing the number of serves of each type of food required per day
- the recommended number of serves of each of the food groups, and discretionary foods, for different sub-population groups
- examples of what a serve size is for each food group

As an example, if you are would like to know how many serves of vegetables men aged between 19 and 50 should eat each day you would refer to the *Australian Guide to Healthy Eating*. This information is also included in the *Guidelines* under 'Practical considerations for health professionals' for each food group.

Related brochures and posters for health professionals and consumers

All these documents are available on the web at www.eatforhealth.gov.au.

Figure 1.1: Relationship between the documents related to the Australian Dietary Guidelines



1.4 How the *Guidelines* were developed

These *Guidelines* are an evolution of the *2003 Dietary Guidelines,* building upon their evidence and science base. New evidence was assessed to determine whether associations between food, dietary patterns and health outcomes had strengthened, weakened, or remained unchanged. Where the evidence base was unlikely to have changed substantially (for example, the relationship between intake of foods high in saturated fat and increased risk of high serum cholesterol), additional review was not conducted.

The methods used to analyse the evidence were in accordance with international best practice [14, 34]. They are summarised below, and provided in more detail in Appendix 2.

The *Guidelines* are further informed by substantial advances in the methodology for guideline development and usability in the eight years since publication of the previous dietary guidelines.

Human feeding studies and clinical trials provide direct evidence of the impact of food consumption on physiological responses and disease biomarkers. Although the breadth and depth of knowledge generated from these kinds of studies is uneven, a consistent alignment of results with plausible mechanisms adds confidence in the analysis of all studies combined.

1.4.1 Sources of information

Five key evidence streams

In developing the *Guidelines*, NHMRC drew upon the following key sources of evidence (see figure 1.1):

- the previous Dietary Guidelines for Australians series and their supporting documentation [35-37]
- a commissioned literature review: A review of the evidence to address targeted questions to inform the revision of the Australian dietary guidelines (referred to as 'the Evidence Report') [14]
- NHMRC and the New Zealand Ministry of Health 2006: Nutrient reference values for Australia and New Zealand including recommended dietary intakes (referred to as 'the NRV document') [9]
- a commissioned report: A modelling system to inform the revision of the Australian Guide to Healthy Eating (referred to as 'the Food Modelling' document) [10]
- key authoritative government reports and additional literature

The Evidence Report – answers to key questions in the research literature

NHMRC commissioned a literature review (*A review of the evidence to address targeted questions to inform the revision of the Australian dietary guidelines*—the Evidence Report) on food, diet and disease/health relationships, covering the period 2003–2009. This addressed specific questions developed by the expert Dietary Guidelines Working Committee (the Working Committee) on food, diet and disease/health relationships where evidence might have changed since the previous dietary guidelines were developed.

NHMRC followed critical appraisal processes to ensure rigorous application of the review methodology [34, 38]. Data were extracted from included studies and assessed for strength of evidence, size of effect and relevance of evidence according to standardised NHMRC processes [34, 39-41]. The components of the body of evidence—*evidence base* (quantity, level and quality of evidence); *consistency* of the study results; *clinical impact*; *generalisability*; and *applicability* to the Australian context—were rated as *excellent*, *good*, *satisfactory* or *poor* according to standard NHMRC protocols [41].

The reviewers then summarised the evidence into draft body of evidence statements. The Working Committee advised that a *minimum of five high quality studies* was required before a graded draft evidence statement could be made. The individual studies in meta-analyses were considered as separate studies. The draft Evidence Statements were graded A to D according to standard NHMRC protocols [41].

- Grade A (convincing association) indicates that the body of evidence can be trusted to guide practice
- Grade B (probable association) indicates that the body of evidence can be trusted to guide practice in most situations
- Grade C (suggestive association) indicates that the body of evidence provides some support for the recommendations but care should be taken in its application
- Grade D indicates that the body of evidence is weak and any recommendation must be applied with caution.

Once the evidence statements and grades had been drafted, NHMRC commissioned an external methodologist to ensure that the review activities had been undertaken in a transparent, accurate, consistent and unbiased manner. This ensures that the work can be easily double-checked by other experts in nutrition research.

In this way, the Evidence Report was used to develop the graded Evidence Statements included in the *Guidelines*. It is important to note that these grades relate to individual diet-disease relationships only—the *Guidelines* summarise evidence from a number of sources and across a number of health/disease outcomes.

Levels of evidence in public health nutrition

Randomised controlled trials provide the highest level of evidence regarding the effects of dietary intake on health. However, as with many public health interventions, changing individuals' diets raises ethical, logistical and economic challenges. This is particularly the case in conducting randomised controlled trials to test the effects of exposure to various types of foods and dietary patterns on the development of lifestyle-related disease.

Lifestyle-related diseases generally do not develop in response to short-term dietary changes; however short-term studies enable biomarkers of disease to be used to evaluate the effects of particular dietary patterns. The question of how long dietary exposure should occur to demonstrate effect on disease prevention is subject to much debate. While it may be possible to conduct a dietary intervention study for 12 months or more to examine intermediate effects, there would be many ethical and practical barriers to conducting much longer, or indeed, life-long, randomised controlled trials with dietary manipulation to examine disease prevention.

As a result, the nature of the evidence in the nutrition literature tends to be based on longer term observational studies, leading to a majority of grade C evidence statements and some which reach grade B where several quality studies with minimal risk of bias have been conducted. For shorter term and intermediary effects, particularly when studying exposure to nutrients and food components rather than dietary patterns, grade A is possible.

The relatively high proportion of evidence statements assessed as grade C should not be interpreted as suggesting lack of evidence to help guide practice. However, care should still be applied in the application of this evidence for specific diet-disease relationships, particularly at the level of the individual [34, 38].

Health professionals and the public can be assured that the process of assessing the scientific evidence provides for the best possible advice. Only evidence statements graded A, B, or C influenced the development of the *Guidelines*.

Grade D evidence statements

Grade D evidence statements occur when the evidence for a food-diet-health relationship is limited, inconclusive or contradictory. These D-grade relationships were not used to inform the development of *Guidelines* statements, however can be useful to inform health professionals about the strength of evidence from recent research. The full set of D-grade evidence statements can be found in the Evidence Report [14].

The Food Modelling Report – translating nutrient requirements into dietary patterns

The report *A modelling system to inform the revision of the Australian Guide to Healthy Eating* (the Food Modelling Report) was commissioned by the NHMRC between 2008 and 2010. It determined a range of combinations of amounts and types of foods that could be consumed to meet nutritional needs with the least amount of energy for the smallest and least active people within an age and sex group. This report applies the *Nutrient reference values for Australia and New Zealand including recommended dietary intakes* [9] and provides information on the serve sizes and minimum number of daily serves required for each population group to achieve the required intake of vitamins, minerals and macronutrients.

There were several inputs in the development of this report including consultation processes arranged by NHMRC and a public consultation of the draft report in April/May 2010 after which the models were finalised.

The Food Modelling Report informed the revision of the *Australian Guide to Healthy Eating* (see Section 1.7) and was considered together with other sources of evidence to determine the recommendations in the *Guidelines*.

Capturing new evidence

Nutrition is a continuously evolving area and research studies are published on a regular basis. Relevant results from high quality studies (primarily systematic reviews) assessing food, diet and health relationships published after the literature review for the Evidence Report (after 2009) were also considered in the development of the *Guidelines*. While results from these studies were not graded, and did not influence the Evidence Statements, they were included in the *Guidelines* and were deemed warranted to ensure currency.

As the Evidence Report only included studies investigating food, diet and health relationships, the results of other high quality studies published since 2002 were used to update the sections in the *Guidelines* which provided other information ('Setting the scene', 'How eating a particular food may improve health outcomes', and 'Practical considerations for health professionals' sections) if they met the following criteria:

- the study was a high quality randomised controlled trial, intervention, cohort, or observational study, but not an editorial or opinion piece (meta-analyses were considered)
- the outcome of the study related to some aspect of health or chronic disease

- the study results were generalisable to the Australian population
- the study was related to foods or the total diet rather than nutrients.

While they did not influence the Evidence Statements or grading's, these sources were used to assist in refining translation of the evidence.

1.4.2 How the evidence was used

Getting the guideline wording right

The final wording of each recommendation was developed by a Working Committee consensus approach, based on the information gained from the five key sources listed Section 1.4.1.

For example, to translate all available evidence regarding consumption of vegetables and health outcomes to develop dietary guideline recommendations the following evidence was considered:

- the graded Evidence Statements (from Grade A through to C) about the relationship between consumption of vegetables and various health outcomes [14]
- the importance of vegetables as a source of key nutrients in the Australian diet from the Food Modelling document [10] and the NRV document [9]
- the relatively low energy content of vegetables [9, 10]
- findings of international authoritative reports including the World Cancer Research Fund report [42]
- information provided in the 2003 *Dietary Guidelines* [37].

Assessment of all available sources of evidence confirmed the importance of consumption of vegetables for promoting health and wellbeing. The Working Committee translated this evidence into the recommendation to 'eat plenty of vegetables'.

Using Evidence Statements

The manner in which Evidence Statements were developed is described in Section 1.4.1. In the sections titled 'Evidence', all the graded evidence statements are underpinned by evidence from the Evidence Report, and referred to clearly in the text in these sections. This section also includes relevant ungraded referenced evidence from the other four key sources (see Section 1.4.1) to ensure comprehensiveness and currency. These Evidence sections provide the basis of the scientific information that was translated to form each guideline recommendation at the beginning of each chapter.

To ensure the *Guidelines* are realistic, practical and achievable, the scientific and social context for each Guideline was considered. This information is included for each Guideline under the heading 'Setting the Scene'.

Potential mechanisms through which particular dietary patterns may influence health were considered to help assess the plausibility of the associations described in the Evidence sections. This information is included for each Guideline under the heading 'How a particular food/dietary pattern may improve health outcomes'.

This information has originated predominantly from previous dietary guidelines series, updated by narrative reviews of additional literature sourced from authoritative reports, from the Food Modelling Report, from the NRV document [9] and from high quality studies published since the last Dietary Guidelines in 2003.

1.5 Adherence to dietary advice in Australia

Adherence to dietary recommendations in Australia is poor [43]. Most children's intake of vegetables, fruit, grain (cereal) foods and milk, yoghurt and cheese products and alternatives is below recommended levels, while their intake of saturated fat and sugar exceed recommendations [13]. Analysis of Australia's *1995 National Nutrition Survey* [44] found that energy-dense, nutrient-poor 'extra foods' [45] contributed 41% of the total daily energy intake of 2–18 year olds [46].

The most recent dietary data available for Australian adults (collected in the *1995 National Nutrition Survey*) also showed a poor dietary pattern with inadequate intakes of vegetables, fruit, wholegrain cereals and milk, yoghurt and cheese products and alternatives, with higher than recommended proportions of fat intake derived from saturated fat [44, 47]. More than 35% of daily energy intake was derived from energy-dense nutrient-poor 'extra foods' [46].

There have been changes in the intakes of macro-nutrients over the past three decades, generally in the direction encouraged by previous dietary guidelines (see Table 1.1) [48].

Table 1.1: Changes in macronutrient intake in Australia for adults and children/adolescents between 1983 and 1995

	Adults (25–64 years) 1983 to 1995		Adolescents (10–15 years) 1985 to 1995	
Nutrient / indicator	Direction (a)	Extent of change	Direction (a)	Extent of change
Energy	Increased	Men 3% Women 4%	Increased	Boys 15% Girls 11%
Protein	Unchanged	-	Increased	Boys 14% Girls 13%
Carbohydrate	Increased	Men 17% Women 16%	Increased	Boys 22% Girls 18%
Fat	Decreased	Men 6% Women 4%	Unchanged	
Dietary fibre	Increased	Men 13% Women 10%	Increased	Boys 13% Girls 8%

Note: (a) Where there is a trend in mean intake it is significant at 1% level.
Source: Cook et al. 2001 [48]

Barriers to compliance

Influences on dietary choices throughout life are complex, ranging from individual, physical and social factors through to societal and environmental factors [49-87].

Possible barriers to compliance with recommendations may include poor communication of advice, low levels of understanding of the information, low levels of food literacy and high levels of food insecurity (this may include the inability to access adequate amounts of nutritious, culturally acceptable foods), conflicting messages (including advertising and promotion of energy-dense nutrient-poor foods and drinks), and particular dietary preferences [88, 89].

There appear to be complex relationships between dietary patterns established in childhood and dietary quality over time. Studies suggest that frequency of takeaway food consumption increases during childhood, adolescence and young adulthood [90] and, together with consumption of low-quality snacks, is associated with higher intakes of energy, total fat, saturated fat and sodium [75-77, 90]. The frequency of eating breakfast decreases with age and is associated with reduced intake of calcium and dietary fibre [91]. There is some evidence that family meal patterns during

adolescence predict diet quality and meal patterns during early young adulthood [92]. Childhood smoking is also associated with poor dietary habits [78, 83] as is stressful family life [86, 87].

Challenges for adoption of the *Guidelines*

An improved understanding of what Australians *are* eating will assist the implementation and uptake of the *Guidelines* which provide the evidence for what Australians *should be* eating. Much of our current knowledge of adult Australian dietary patterns comes from the *National Nutrition Survey 1995*, however the *Australian Health Survey 2011 – 2013* [93] will provide a better understanding of the current diet and nutrition of Australians, or what Australians *are* eating.

Although the key messages of the *Guidelines* may not have changed significantly since 2003, the evidence base supporting them has strengthened considerably. The challenge now is to ensure that these *Guidelines* – particularly with renewed emphasis on achieving and maintaining a healthy weight – are strongly promoted in a context that encourages and supports more nutritious food choices, dietary patterns and healthy lifestyles within the community.

1.6 Dietary choices and the environment

Increasingly, Australians are seeking advice from health and medical practitioners about food choices and their possible impact on the environment. These concerns, coupled with a growing population, means rethinking matters of food security, what is available to eat, and more importantly what people are recommended to eat [94-97].

Dietary guidelines evolve as knowledge grows. Preliminary work indicates that dietary patterns consistent with the Dietary Guidelines are likely to have a lower environmental impact than other dietary patterns. Available Australian and international evidence is insufficient to be able to provide advice on the environmental impact of specific food items or brands, however there may be some practical steps that people can take. For example:

- Buy and consume foods and drinks that are consistent with the Dietary Guidelines
- Avoid overconsumption
- Minimise food wastage
- Consider your food buying, storage, preparation and disposal practices, and
- Minimise and recycle the packaging of food

NHMRC aims to work with other agencies to provide guidance for health professionals as they work with clients and patients. Many complex interactions exist as food is grown, transported, sold and consumed. As a health agency, NHMRC will need partners to achieve this aim of providing useful, practical and well informed advice to both health professionals and the general

population. NHMRC intends to work with other Commonwealth government agencies to develop this guidance.[1]

As public health professionals throughout the world increasingly provide advice on the interaction between food choices and the environment, they will need expert advice on how the food system, including the production, processing, retail and distribution, preparation, consumption and disposal of waste all have implications for the environment. The *Australian Dietary Guidelines* must consider the Australian context, as some food production and subsequent handling differs considerably to that which occurs in North America and Europe, where much of the literature originates. NHMRC acknowledges and understands the need to develop Australian guidance, taking into account these factors, and the Australian food regulatory framework.

Environmental issues in the production, processing and sale of food are hotly debated areas, but increasingly, numbers of international bodies charged with dietary advice are beginning to consider this issue, as NHMRC intends to as discussed above. However, despite the complexities surrounding food choices, it is clear that the general principles of these *Dietary Guidelines* are compatible with reducing environmental impacts as well as promoting good health.

1.7 How to use the *Guidelines*

This edition of the *Australian Dietary Guidelines* has been developed as a single comprehensive report covering all healthy Australians. The *Guidelines* will be supported by a number of brochures and booklets for parents of infants, children and adolescents, the general population, pregnant and breastfeeding women and Aboriginal and Torres Strait Islanders, which can be found at www.eatforhealth.gov.au. The *Infant Feeding Guidelines for Health Workers* has also been updated as part of a separate process (see www.eatforhealth.gov.au).

The chapters in the *Guidelines* use a consistent approach with three main subheadings for each guideline.

- *Setting the Scene*, which provides a brief background to the topic.
- *Evidence*, which outlines the scientific evidence base since 2002 from studies of associations between human consumption patterns and health outcomes, and the effects of dietary interventions on health outcomes relating to foods, food groups and whole dietary patterns.
- *How eating a particular food (or particular dietary pattern) may improve health outcomes*, describes the mechanisms of action that may underlie the evidence presented.

[1] These agencies have a distinct role in this field, and more information on environmental sustainability and food security is available at their websites www.daff.gov.au and www.environment.gov.au.

- *Practical considerations for health professionals*, which identifies practical issues and health impacts for subgroups within the population including at different life stages.

Dietary guidelines can be effective in directing attention to the types of food people should consume, but there remains a need to focus on the *amount* of food consumed. Overconsumption, even of nutritious foods, can lead to excessive energy intake compared to need and thereby an increase in body weight.

1.8 The *Australian Guide to Healthy Eating*

While the *Australian Dietary Guidelines* provide broad dietary advice, with the underpinning evidence, the *Australian Guide to Healthy Eating* is a practical, pictorial guide to recommended types and serves of foods to consume every day[10, 14]. It also includes information on standard serve sizes for different food types.

The recommended foods and number of daily serves for different population groups have been included in each of the *Guideline* chapters under 'Practical Considerations', and are also available at www.eatforhealth.gov.au.

Figure 1.2: Australian Guide to Healthy Eating

2. Eat a wide variety of nutritious foods

Guideline 1

Eat a wide variety of nutritious foods from these five groups every day:

- plenty of vegetables, including different types and colours, and legumes/beans
- fruit
- grain (cereal) foods, mostly wholegrain, such as bread, cereals, rice, pasta, noodles, polenta, couscous, oats, quinoa and barley
- lean meat and poultry, fish, eggs, nuts and seeds, and legumes/beans
- milk, yoghurt, cheese and/or their alternatives, mostly reduced fat (reduced fat milks are not suitable for children under 2 years).

And drink water.

Executive Summary

Dietary patterns which include a wide variety of nutritious foods are more likely to meet nutrient requirements, promote health and wellbeing and confer health benefits than restricted diets.

A variety of foods should be consumed from each of the five food groups: vegetables and legumes/beans; fruit; grain (cereal) foods mostly wholegrain; lean meat and poultry, fish, eggs, nuts and seeds, and/or legumes/beans; and milk, yoghurt, cheese and/or alternatives. Mostly reduced-fat milk, yoghurt and cheese products are recommended for adults, but reduced fat milks are not suitable as the main milk drink for children under the age of two years.

There are many different ways to combine these nutrient-dense foods to produce nutritious dietary patterns that suit cultural, economic, social and culinary preferences, as well as delivering health benefits.

There is increasing evidence that current consumption patterns are associated with reduced risk of chronic disease including cardiovascular disease, type 2 diabetes, and several cancers.

Together with adherence to Guideline 2 (on limiting intake of specific foods high in saturated fat, sugar and/or salt) and Guideline 3 (on achieving and maintaining a healthy weight), consumption of a wide variety of nutritious foods and choosing water as a drink will substantially reduce the risk of diet-related chronic disease and promote health and wellbeing in Australia.

This chapter provides information on why the consumption of a wide variety of nutritious foods is beneficial to health, the evidence for the recommended approach, and includes practical advice for the general population and specific subpopulation groups.

2.1 Eat a wide variety of nutritious foods

2.1.1 Setting the scene

No single food - with the exception of breastmilk for about the first six months of life - can provide all the nutrients in the amounts needed for good health. Dietary patterns which include a wide variety of nutritious foods and water are more likely than restricted diets to meet nutrient requirements [9] and confer health benefits. A dietary pattern needs to include a variety of choices from each of the five food groups – vegetables, fruit, grain (cereal) foods, lean meat and poultry, fish, eggs, nuts and seed and legumes/beans, and milk, yoghurt, cheese and/or alternatives.

Most Australians today eat a wide variety of foods from different cuisines. The available food supply generally meets the nutritional needs of the population, but appropriate choices must be made to ensure that all nutrient requirements are met, so that diet-related chronic disease can be prevented or delayed, and so that optimum health and wellbeing can be achieved [10]. Australia is also fortunate in having a safe food supply with low levels of contaminants and pollutants[98].

The most recent dietary survey data available for Australian adults – the *National Nutrition Survey 1995* – showed an increasing number of foods being consumed by adults in that year compared with 1983 [44]. It is expected that the variety of foods consumed has continued to increase since 1995. This is largely as a result of cultural diversity in the population arising from waves of immigration from European countries after World War II and Asian and African countries since the 1970s [99, 100]. Initially, new varieties of fresh fruit and vegetables, grain (cereal) foods and different types of meat and legume/beans became available. Increasing demand for convenience and/or fast foods - also as a result of changes in social and economic conditions - has led to the availability of approximately 30,000 different types of foods and drinks [101]. However, many of these - particularly snack and fast foods and drinks - are energy-dense and nutrient-poor, so care is required to choose diets consistent with the *Guidelines* [102].

Despite the variety of foods available in Australia, comparison of actual intakes with recommended nutrient intakes [9] shows that some people are still at risk for deficiencies of particular nutrients. For example, intakes of iron and calcium continue to be low in relation to recommendations for some girls and women of reproductive age [10] and iodine intake is inadequate in some pregnant and breastfeeding women [103].

Consuming a dietary pattern consistent with the evidence presented in this chapter will help to promote health, protect against disease and reduce the prevalence of nutrient deficiencies. Together with adherence to Guideline 2 and Guideline 3, adherence to this guideline will help to reduce the risk of diet-related chronic disease such as cardiovascular disease, type 2 diabetes and some cancers [9, 10, 14], and promote health and wellbeing.

Consuming a wide variety of foods may also help to ensure that an adequate variety of foods remains available into the future. Food choices to reduce the impact on the environment are consistent with those to improve health.

2.1.2 The evidence for *'Eat a wide variety of nutritious foods'*

Evidence of the health benefits of a dietary pattern consisting of a variety of nutritious foods in appropriate amounts has strengthened over the past decade. The evidence suggests that high diet quality is associated with a reduced risk of chronic disease and improved health outcomes (Grade C, Section 20.3 in Evidence Report [14]) [22, 104-107]. Reviews of studies of a range of eating patterns suggest that:

- higher quality diet is associated with reduced morbidity
- the health effects tend to be greater in those with lower quality diets, such as men, young people and people with lower educational and socioeconomic status
- consuming a dietary intake pattern consistent with national dietary guidelines or other scientifically informed recommendations is associated with reduced morbidity and mortality (Grade C, Section 20.3 in Evidence Report [14]) [21, 22].

Recent evidence also confirms that a dietary pattern consistent with current guidelines to consume a wide variety of foods including vegetables, fruit, wholegrains, lean meat, poultry, fish and reduced-fat milk, yoghurt and cheese products may be associated with superior nutritional status, quality of life and survival in older adults [23, 24].

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) related to 'eat a wide variety' from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines*.

Evidence Statement	Grade
The evidence suggests that high quality diet is associated with a reduced risk of chronic disease and improved health outcomes.	C
The evidence suggests the consumption of a dietary intake pattern aligned with national dietary guidelines or recommendations is associated with reduced morbidity and mortality.	C

2.1.3 How *eating a wide variety of nutritious foods* may improve health outcomes

Nutritional science has traditionally characterised foods according to their macro- and micro-nutrient values, but there is increasing evidence of the role that whole foods play in promoting health and wellbeing, and of the role of food components other than nutrients in protecting against some communicable and non-communicable chronic diseases when consumed as part of a varied nutritious diet [108, 109]. These non-nutrient components include phytochemicals, which are obtained from plants and are biologically active but not directly associated with deficiency syndromes. They include carotenoids, flavonoids, isoflavonoids, polyphenols, isothiocyanates, indoles, sulphoraphanes, monoterpenes, xanthins and non-digestible oligosaccharides. It is not always known which food constituents are responsible for the protective effects of specific foods against specific chronic diseases, and it is likely that many other active constituents will be discovered in the future. Interactions between these compounds are likely to be complex, either causing or masking effects, or acting synergistically with other compounds [10].

Dietary variety has the benefit of diluting potential toxicants found naturally in foods [110]. Enjoying diversity in food intake can reduce an individual's exposure to any one group of toxicants. Other ways of minimising this risk include appropriate and careful processing, cooking and storage of food (see Chapter 7).

Another potential benefit of food variety comes from maximising the bioavailability of nutrients [111]. The many complex relationships between foods, nutrients and food components (such as phytates) can influence the absorption, metabolism and retention of nutrients. When dietary patterns are varied and nutrients are in adequate supply, these interactions pose fewer problems than in restricted, monotonous dietary patterns.

Clinical problems associated with excessive intake of nutrients are nearly always associated with intakes of supplements [9]. It is also possible to develop symptoms of toxicity when dietary patterns concentrate on particular foods, or if the same nutrient is consumed in different chemical forms [9, 112]. Examples include excessive consumption of carrot juice or regularly eating very large quantities of liver, which may cause vitamin A toxicity [113].

2.1.4 Practical considerations: *Eat a wide variety of nutritious foods*

It is important to consider this guideline together with other guidelines, particularly Guideline 2 on limiting intakes of specific foods high in saturated fats, added sugars and/or added salt and Guideline 3 on healthy weight. The word 'plenty' is used judiciously to encourage increased consumption of vegetables (particularly non-starchy varieties). For the other food groups, the *guidelines* do not advocate *plenty*, but rather focus on an adequate amount of the preferred

varieties within each food group, for example *wholegrain*, or *lean*, or *lower fat* and plain water, preferably from the tap. This serves to distinguish between eating a *variety* and *over-consumption* as there is some evidence that there may be a link between eating a variety of energy-dense food and drinks and excessive food intakes (see Chapter 4). V*ariety* refers to *nutritious* food, not discretionary foods.

Different quantities of different types of foods from the five food groups are recommended for different ages, sexes, and those with different energy (kilojoule) intake requirements. These are based on dietary modelling to inform the revised *Australian Guide to Healthy Eating* [10]. For example, men aged 19-50 need to eat around 19.5 – 22.5 serves of food from the five food groups each day while 19-50 year old women need to eat 18 – 20.5 serves from these five food groups. Detailed information on the number and types of serves of food for different population groups is in the *Australian Guide to Healthy Eating* [114].

2.1.4.1 Pregnant and breastfeeding women

Consuming a variety of nutritious foods is particularly important during pregnancy and while breastfeeding. Quality nutritious dietary patterns during pregnancy may reduce the risk of babies being small for their gestational age or exhibiting restricted intrauterine foetal growth [115-117], being large for their gestational age [118], and may also help reduce the risk of pregnant women developing pre-eclampsia [119, 120]. Quality nutritious dietary patterns before and during pregnancy may help reduce the risk of women developing gestational diabetes mellitus [120-123].

Maternal diet during pregnancy and while breastfeeding does not appear to affect the risk of asthma, eczema or other allergy symptoms in infants [124-128]. Some health outcomes (such as allergies) in children will be affected more by their diet through infancy and childhood than their mother's diet during pregnancy, with in-utero influences likely to be minimal. However, a cohort study found that mothers who adopt high quality dietary patterns are more likely to have children who also consume nutritious diets [129].

Foods that should be avoided during pregnancy include those associated with increased risk of *Listeria* bacteria, such as soft cheeses, cold seafood, sandwich meats, bean sprouts and packaged or pre-prepared salads [130]. Intake of certain species of fish should be limited, due to the potential risk of excessive mercury intake. Pregnant women are advised to consume no more than one serve per fortnight of shark, marlin or broadbill/swordfish, and no other fish that fortnight, or one serve per week of orange roughy (deep sea perch) and no other fish that week [130, 131].

Constipation is a common symptom during pregnancy [132]. Clinical treatment of constipation generally includes advice to consume a high fibre diet, including wholegrain cereals, fruit, and vegetables, and to drink water, which is consistent with these *Guidelines*.

2.1.4.2 Infants

Exclusive breastfeeding is recommended for around the first six months of age, after which solid (pureed or spoon) foods of suitable texture can be introduced. There is no particular order or rate for the introduction of new foods, other than the first foods should be rich in iron. Breastfeeding should continue until 12 months and beyond for as long as the mother and child desire (for more information, see the *Infant Feeding Guidelines for Health Workers* [133]), Food choices should be varied to ensure adequate energy (kilojoule) and nutrient supply.

When introducing solids (spoon foods) to infants, parents and carers should ensure that a wide variety of nutritious foods of different colours, suitable textures and types is offered. This will provide the additional nutrients required as infants grow and the variety will help to increase acceptance of different nutritious foods. It is also more likely to improve the acceptance of a varied diet during childhood. Hard foods, such as nuts, should not be offered to children until they are over three years of age and they are sufficiently mature to reduce the risk of choking.

2.1.4.3 Adults

Males living alone are at particular risk of not eating a wide variety of nutritious foods. Analysis of the *1995 National Nutrition Survey* [37] showed that in nearly all age groups adult males consumed significantly fewer types of foods than other groups.

2.1.4.4 Older people

The diet of older people is generally more varied than that of younger groups [44]. However the frail elderly are at increased risk of consuming monotonous, limited diets due to factors such as reduced mobility, poor dentition and poverty which may reduce access to a range of fresh foods.

2.1.4.5 Aboriginal and Torres Strait Islanders

Limited data on Aboriginal and Torres Strait Islander dietary intake are available. Very restricted dietary patterns - in which over 50% of energy (kilojoule) intake was derived by meat, flour and sugar - have been described in remote Aboriginal communities [134] with relatively little change observed recently [135]. However the majority of Aboriginal and Torres Strait Islander people live in urban areas and their most significant dietary issues tend to be also experienced by all people in lower socioeconomic groups.

2.1.4.6 People in lower socioeconomic groups

In some urban centres, people in lower socioeconomic groups have less access to supermarkets and greater access to fast food outlets than more advantaged groups [136, 137]. Supermarkets generally offer a wider variety of food products, as well as fresh raw food.

In Australia, the cost of a nutritious diet has been estimated to account for about 40% of the disposable income of welfare-dependent families, compared to only 20% of an average families' disposable income. Health professionals should be aware of the budget challenges which healthy food habits may pose for people who are welfare dependent and should note that substituting generic brands for market brands can reduce the weekly food cost by about 13% [138]. Further details regarding the association of equity issues and consumption of varied and nutritious diets are included in Appendix 7.

2.1.4.7 People living in remote areas

The decreased availability of nutritious foods (such as fresh fruit and vegetables, wholegrain bread and low-fat milk products) in remote and regional areas in Australia has been described frequently. The cost of nutritious foods in these areas is also over 30% higher than in major cities and may impact on food security [30, 139-141].

2.1.4.8 Vegetarians

About 4% of respondents in the *National Nutrition Survey 1995* described themselves as vegetarian or vegan [44]. The food frequency questionnaire data recorded only 2% as consuming no animal products, and a further 2% as restricting consumption of animal foods to fish or white meat [44]. Many more people eat vegetarian meals regularly or occasionally. Those following a strict vegetarian or vegan diet need to choose a variety of protein sources throughout the day to get an adequate mix of amino acids. Health professionals should encourage vegetarians to choose foods carefully to ensure adequate intake of iron, zinc and vitamin B12 and to optimise the absorption and bioavailability of iron, zinc and calcium [142].

2.2 Plenty of vegetables, including different types and colours, and legumes/beans, and eat fruit

2.2.1 Setting the scene

There are many nutritional, societal, culinary and environmental reasons to ensure that vegetables, including legumes/beans, and fruit are a major component of Australian dietary patterns. As a group, these foods are nutrient dense, relatively low in energy (kilojoules) and are good sources of minerals and vitamins (such as magnesium, vitamin C and folate), dietary fibre and a range of phytochemicals including carotenoids. Many of the sub-components of foods and their relationships have not been studied in detail, and it is expected that other sub-components, and their biological effects, are still to be discovered.

The inclusion of a variety of vegetables, including legumes/beans, and fruit provides a diversity of colours, textures and flavours, adding to the enjoyment of eating. Vegetables, including legumes/beans, and fruit should be eaten in their whole food forms to maximise the impact on a range of health benefits. Fruit should mostly be eaten fresh and raw because of the low fibre content of fruit juice and the high energy density and 'stickiness' (which may have implications for dental caries) of dried fruit [10]. Some vegetables are suitable to eat raw, while it is best to cook others to make them more palatable and digestible. Legumes should always be cooked. Some processed fruits and vegetables are nutritious alternatives as long as they are produced without added salt, sugar (including concentrated fruit juice) or fat.

Different fruits and vegetables are rich in different nutrients. For example, green leafy and *Brassica* (or cruciferous) vegetables are generally high in folate, and starchy vegetables are a good source of complex carbohydrates. Legumes/beans provide a valuable and cost efficient source of protein, iron, some essential fatty acids, soluble and insoluble dietary fibre and micronutrients for all Australians, but particularly for those consuming vegetarian meals [10].

The health benefits of consuming diets high in vegetables, including legumes/beans, and fruit have been reported for decades and are consistently recognised in international dietary guidelines [36, 37, 143, 144].

2.2.2 The evidence for *'plenty of vegetables'*

The scientific evidence of the health benefits of consuming vegetables (including legumes/beans) has been strong for several decades and has generally continued to strengthen over recent years, particularly the evidence for a protective effect against cardiovascular disease. Recent research on

vegetable intake and cancer has focused more on investigating the health effects of consuming different subgroups of vegetables on site-specific cancers than the effect of total vegetable intake. There is strengthened evidence of the beneficial effects of intake of various non-starchy vegetables in reducing risk of some site-specific cancers. High dietary intakes of starchy vegetables may help explain the weaker association between total vegetable intake and many site-specific cancers. There is also greater clarity on the quantity of vegetables to produce beneficial health effects, plus increasing evidence of a protective effect against a number of chronic diseases for consumption of vegetables and fruit when considered together (see Appendix 5).

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines.*

Evidence Statement	Grade
Consumption of each additional daily serve of vegetables is associated with a reduced risk of coronary heart disease.	B
Consumption of vegetables is associated with reduced risk of stroke.	B
Consumption of vegetables is associated with reduced risk of weight gain	C
Consumption of vegetables is associated with a reduced risk of oral and nasopharyngeal cancers.	C
Consumption of preserved vegetables is associated with increased risk of oral and nasopharyngeal cancer.	C
Consumption of 1-2 serves per day of tomato is associated with a reduced risk of prostate cancer.	C
Consumption of more than 1 serving per week of spinach is associated with reduced risk of colorectal cancer.	C
Consumption of cruciferous vegetables is associated with reduced risk of lung cancer.	C

Although serving sizes of vegetables differed between studies considered in the Evidence Report [14], Evidence Statements presented below are based on the Australian standard serve size of 75g.

2.2.2.1 Cardiovascular disease, type 2 diabetes and excess weight

Cardiovascular disease: It is probable that each additional daily serve of vegetables is associated with a reduced risk of coronary heart disease (Grade B, Section 2.1 in Evidence Report [14]) [145-150].

It is probable that consumption of vegetables is associated with a reduced risk of stroke (Grade B, Section 2.4 in Evidence Report [14]) [151, 152]. The protective effect is stronger at higher intakes.

Type 2 diabetes: Recent evidence suggests that consumption of vegetables does not appear to be directly associated with the risk of type 2 diabetes (Grade C, Section 2.3 in Evidence Report [14]) [153-155]. This supports the inconsistent findings described in other studies for fruit and vegetables considered together and for fruit alone [154, 155]. However, as there is a strong relationship between type 2 diabetes and body weight (see Chapter 4), the association between consumption of vegetables and reduced risk of excessive weight gain (see below) suggests longer-term studies may be required to further investigate potential effects.

Excess weight: Recent evidence suggests that consuming vegetables is associated with a reduced risk of weight gain (Grade C, Section 2.2 in Evidence Report [14]) [156-160].

2.2.2.2 Cancer

Oral, nasopharyngeal and oesophageal: Evidence suggests that consuming vegetables is associated with a reduced risk of oral and nasopharyngeal cancers (Grade C, Section 2.10 in Evidence Report [14]) [161-165]. This is consistent with the findings described by the WCRF report [42]. However the evidence also suggests that consumption of preserved vegetables (salted, dried, fermented or pickled) is associated with increased risk of these cancers (Grade C, Section 2.10 in Evidence Report [14]) [161-165].

It is unclear from recent studies whether there is an association between total vegetable consumption and risk of other alimentary cancers, however relationships found previously [36, 37] still tend to be present when a longer time frame and different types of vegetables are considered (Section 2 in Evidence Report). Although recent evidence suggests that total consumption of vegetables is not associated with reduced risk of oesophageal cancer (Grade C, Section 2.9 in Evidence Report [14]) [166-169], evidence from the WCRF report suggests that consumption of non-starchy vegetables probably reduces risk of cancer of the oesophagus [42].

Prostate cancer: The evidence suggests that consumption of 1–2 serves of tomatoes a day is associated with reduced risk of prostate cancer (Grade C, Section 2.13 in Evidence Report [14]) [170, 171]. This is consistent with the probable relationship between intake of lycopene-containing foods and reduced risk of prostate cancer described by the WCRF report [42].

Endometrial, Ovarian and pancreatic cancer: Previous dietary guidelines reported a possible reduction in risk of endometrial and pancreatic cancer with vegetable consumption [37]. However, in more recent studies there is no evidence to suggest an association between total vegetable consumption and ovarian (Grade C, Section 2.11 in Evidence Report [14]) [172, 173] or endometrial cancer (Grade C, Section 2.12 in Evidence Report [14]) [174-176]. However findings from the WCRF report suggest decreased risk of both ovarian and endometrial cancer with consumption of non-starchy vegetables specifically [42].

Colorectal cancer: The evidence suggests no association between consumption of green leafy, cruciferous vegetables, or carrots, potatoes, beans or lentils and risk of colorectal cancer (Grade C, Section 2.15 in Evidence Report [14]) [177]. More specific studies suggest that consumption of more than one serving per week of spinach is associated with reduced risk of colorectal cancer (Grade C, Section 2.15 in Evidence Report [14]) [177] and evidence of a suggestive protective effect of intake of non-starchy vegetables on colorectal cancer has been described by the WCRF report [42].

Lung cancer: Recent evidence [178] suggests consuming cruciferous vegetables is associated with reduced risk of lung cancer (Grade C, Section 2.14 in Evidence Report [14]). The World Cancer Research Fund report found evidence of a probable association with the reduced risk of lung cancer with consumption of vegetables containing carotenoids and also found evidence suggesting that non-starchy vegetables were protective of lung cancer [42]. This supports the notion that different types of vegetables may have different effects which may help to explain conflicting results seen when all vegetables are grouped together in varying proportions in different studies.

Other cancers: Recent evidence is limited and/or inconclusive for the association regarding vegetable consumption and gastric, breast, lung and colorectal cancers (Section 2.5, 2.6, 2.7 & 2.8 in Evidence Report [14]).

2.2.2.3 Other conditions

The previous dietary guidelines included evidence of associations between the consumption of vegetables and some aspects of eye health, including cataracts and macular degeneration of the eye [37]. Further evidence was not available from more recent studies [179].

2.2.3 The evidence for *'plenty of legumes/beans'*

While evidence of the health benefits of consumption of legumes/beans appears to have strengthened since the 2003 Dietary Guidelines, the recent research is dominated by studies into the health benefits of soy foods and products rather than investigations into legumes *per se*[2].

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines*.

Evidence Statement	Grade
Consumption of soy foods is associated with reduced total cholesterol and LDL-cholesterol.	C
Consumption of legume foods is associated with reduced risk of colorectal cancer.	C

Recent evidence confirms a protective effect for consumption of legumes, and particularly soy foods, against several risk factors and diseases. However more research is needed to determine the quantities of legume/beans required to produce health benefits, long-term efficacy, and the relative effect of legume foods, including of soy-based foods themselves, as opposed to food components such as isoflavones.

2.2.3.1 Cardiovascular disease, type 2 diabetes and excess weight

Cardiovascular disease: Recent evidence suggests that consumption of soy foods is associated with reduced total cholesterol and LDL cholesterol levels, as markers for coronary heart disease risk (Grade C, Section 7.4 in Evidence Report [14]) [180].

Type 2 diabetes: No recent studies of the relationship between legumes/beans and type 2 diabetes were identified [14].

Excess weight: No recent studies of the relationship between legumes/beans and weight loss were identified [181-183].

[2] The evidence presented here was reviewed with a focus on whole foods, and for this reason studies of soy isolates are not reported.

2.2.3.2 Cancer

Colorectal cancer: Evidence suggests that consuming legumes is associated with reduced risk of colorectal cancer (Grade C, Section 7.3 in Evidence Report [14]) [184-188]. However, in one study the effect was only significant for women [187], as also seen in the recent analysis of the European Prospective Investigation into Cancer and Nutrition (EPIC) database [189]. However no evidence of an association between consumption of legumes and colorectal cancer was described in the WCRF report [42].

Other cancers: Recent evidence is limited and/or inconclusive for an association regarding legume/bean consumption and breast or prostate cancer (Section 7.1 & 7.2 in Evidence Report [14]). An insufficient number of studies were available to form an evidence statement on legume/bean consumption and gastric cancer. However the WCRF report found limited evidence of a relationship between the consumption of legumes and a decreased risk of gastric cancer [42].

2.2.3.3 Other conditions

Recent evidence is limited and/or inconclusive regarding an association between consumption of soy foods and bone fracture in post-menopausal women, cerebral and myocardial infarct, and mortality due to cardiovascular disease and high blood pressure.

2.2.4 The evidence for *'eat fruit'*

Evidence for the health advantages of including fruit in the diet has been strong for decades, but has strengthened considerably recently, particularly for cardiovascular disease. There is also increasing evidence of a protective effect against a number of chronic diseases for consumption of vegetables and fruit when considered together (see Appendix 5). Protective effects are increasingly described in quantitative terms, although different serve sizes have been used in different studies, which make comparison difficult, while findings about dose response are not always consistent across studies.

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines*.

Evidence Statement	Grade
Consumption of each additional daily serve of fruit is associated with a reduced risk of coronary heart disease.	B
Consumption of at least 1.5 serves of fruit a day, ideally 2.5 or more is associated with reduced risk of stroke.	B
Consumption of fruit is associated with a reduced risk of obesity and weight gain.	C
Consumption of fruit is associated with a reduced risk of oral and nasopharyngeal cancer	C

The following studies relate primarily to whole fruit, although some included dried fruit and/or fruit juice in their definitions of fruit intake. The evidence regarding fruit juice and excess weight is included under drinks in Section 3.3.2.1. Although serving sizes differed between studies, the evidence statements presented below are based on standard serve sizes of 150g.

2.2.4.1 Cardiovascular disease, type 2 diabetes and excess weight

Cardiovascular disease: It is probable that consumption of each additional daily serve of fruit is associated with a reduced risk of coronary heart disease (Grade B, Section 1.1 in Evidence Report [14]) [145-147]. Increased protection of at least 7% was gained from each additional serve of fruit consumed per day.

It is probable that consuming at least one and a half serves of fruit a day, ideally two and a half or more, is associated with a reduced risk of stroke (Grade B, Section 1.2 in Evidence Report [14]) [151, 152].

Type 2 diabetes: The recent evidence suggests that consumption of fruit is not associated with risk of type 2 diabetes (Grade C, Section 1.4 in Evidence Report [14]). However, as there is a strong relationship between type 2 diabetes and body weight (see Chapter 4), the association between consumption of fruit and reduced risk of excessive weight gain (see below) suggests longer-term studies may be required to investigate potential effects.

Excess weight: The recent body of evidence suggests that consumption of fruit is associated with a reduced risk of obesity and weight gain (Grade C, Section 1.3 in Evidence Report [14]) [156-160, 190-193].

2.2.4.2 Cancer

Alimentary tract cancer: There is emerging evidence that fruit consumption is associated with reduced risk of several types of cancer along the alimentary tract. The recent body of evidence suggests that consumption of fruit is associated with a reduced risk of oral and nasopharyngeal cancer (Grade C, Section 1.10 in Evidence Report [14]) [161, 162, 164, 165, 194], consistent with findings of a convincing effect on reduced risk of cancers of the mouth, pharynx and larynx and a suggestive effect on nasopharyngeal cancers described in the WCRF report [42].

Breast cancer, ovarian cancer and endometrial cancer: Expanding on previous reports [37], recent evidence now suggests that consumption of fruit is not associated with risk of breast cancer (Grade C, Section 1.6 in Evidence Report [14]) [166, 195-199], ovarian cancer (Grade C, Section 1.11 in Evidence Report [14]) [172, 173] or endometrial cancer (Grade C, Section 1.12 in Evidence Report [14]) [174-176, 200].

Colorectal cancer: Recent evidence suggests that consumption of fruit is not associated with risk of colorectal cancer (Grade C, Section 1.8 in Evidence Report [14]) [166, 177, 194, 199, 201-203]. Further, there is limited evidence to suggest an association between the consumption of most fruits by specific type and colorectal cancer (Section 1.14 in Evidence Report [14]) [177, 203], which expands on earlier studies by the WCRF [42].

Other cancers: Recent evidence is limited and/or inconclusive for an association regarding fruit consumption and gastric, lung, oespohageal and pancreatic cancers (Section 1.5, 1.7, 1.9 & 1.13 in Evidence Report [14]).

2.2.5 How *plenty of vegetables, including different types and colours, and legumes/beans, and eating fruit* may improve health outcomes

Various mechanisms may explain the different health benefits of diets high in vegetables, legumes/beans and fruit. These include potential synergies between the foods as well as the action of specific components found at high levels in these foods, including vitamins and minerals, various phytochemicals including carotenoids and bioflavonoids (such as anthocyanins and flavonols), as well as dietary fibre and other specific characteristics of these foods such as low energy (kilojoules) density.

2.2.5.1 Cardiovascular disease, type 2 diabetes and excess weight mechanisms

Food components with anti-oxidant activity including vitamins (vitamin C and E) and phytochemicals in these foods may reduce the risk of inflammation and haemostasis, and of cholesterol becoming oxidised and deposited in blood vessels to form the atherogenous plaques that underlie many cardiovascular conditions [180, 204-206]. Several studies have shown that consumption of vitamin C is associated with reduced risk of cardiovascular disease and stroke, however, other studies have shown no protective effect [207] [9]. Vegetables and fruit are low in sodium and saturated fat [10]. Plant foods including vegetables and fruit provide potassium and magnesium, both of which have been linked to lower blood pressure [9]. Importantly, reviews of the effect of beta-carotene on coronary heart disease suggest that benefits may be related to the components of the foods, various antioxidants and micronutrients in these foods or other confounding factors, rather than to the beta-carotene alone [208].

Most fruit, vegetables and legumes have a low energy (kilojoule) density and high dietary fibre and water content, providing a plausible mechanism for the association of consumption of these foods with reduced risk of weight gain. Any effect is likely to be mediated through potentially increasing satiety, and also through taking longer to chew, which leads to reducing total energy (kilojoule) intake [9, 10]. High dietary fibre intakes have been linked to lower rates of obesity, type 2 diabetes and cardiovascular disease, mainly through an effect on plasma cholesterol [9]. The majority of vegetables are nutrient-dense and low in kilojoules - in particular, green, *Brassica* and other 'salad' vegetables are relatively low in energy (kilojoules) [10]. However, starchy vegetables are less nutrient-dense and are higher in kilojoules, but provide a rich source of complex carbohydrates [10].

Diets high in fibre and specific complex carbohydrates such as non-starch polysaccharides have been used with modest success by people with type 2 diabetes attempting to lose weight. The small effects seen in these experimental situations might relate to a satiating effect due to the prolongation of food absorption and a smoothing of the blood glucose response after meals [9].

In the prevention and dietary control of type 2 diabetes, some vegetables are likely to be of particular value because of their low energy density and relatively high content of dietary fibre, although longer term studies may be required to demonstrate effects.

Legumes are also believed to confer cardiovascular health benefits because they provide a valuable low saturated fat source of protein as an alternative to meats, and because of their glycaemic properties and phytoestrogen and isoflavone content [209, 210]. These reviews suggest the isoflavone in soy foods may have a role in cholesterol reduction, improved vascular health, preservation of bone-mineral density [209] and anti-oestrogenic, anti-proliferative, pro-apoptotic, anti-oxidative and anti-inflammatory processes [210]. However more research is needed to determine the relative effect of legumes/beans and soy foods themselves as opposed to isoflavones specifically.

2.2.5.2 Cancer mechanisms

There is no dominant mechanism to explain the protective effect of vegetables, legumes/beans and fruit for some cancers, and this is complicated by the range of site-specific cancer mechanisms. Some risk factors for cancer, such as oxidising radiation, can operate primarily from childhood or early adult life and antioxidants or other protective constituents of vegetables, including legumes/beans, and fruit may need to be consumed regularly from early life to be effective [211]. Phytochemicals and several vitamins and minerals found in vegetables and fruit are thought to protect against some cancers by a range of mechanisms. Vegtables in the green leafy and *Brassica* subgroup are particularly high in folate [10] and inadequate amounts of folate are thought to increase the risk of cancer by leading to a rise in homocysteine and megaloblastic changes in bone marrow and other rapidly dividing tissues [9]. Poor folate status is thought to affect the induction of DNA hypomethylation, increasing chromosomal fragility or diminishing DNA repair, as well as increasing secondary choline deficiency, reducing killer cell surveillance, and increasing risk of faulty DNA synthesis and metabolism of cancer-causing viruses [9]. Some studies have suggested that folate, primarily from fruit and vegetables, may be more effective in reducing cancer risk among those with habitual high alcohol intake [212, 213].

Several studies have indicated that vitamin C (found in most fruit and vegetables but particularly in citrus fruit, capsicum and tomatoes) is protective against cancer, whereas others have not found convincing evidence of this [9]. Lycopenes found in tomatoes are thought to be protective of prostate cancer, particularly when tomatoes are cooked in olive oil [42]. Carotenes found predominantly in orange, red and yellow fruit and vegetables are also thought to be associated with maintenance of immune function [9]. Dithiolthiones and isothiocyanates (found in *Brassica* or cruciferous vegetables) and allyl sulphides (found in *Allium* vegetables) have been shown to stimulate detoxification processes [37]. The anti-inflammatory action of other phytochemicals, such as flavonoids, is also thought to be important in reducing cancer risk [214]. Potatoes are not as rich in phytochemicals as other types of vegetables, and this may help explain the weaker association with decreased cancer risk and consumption of starchy vegetables compared with other vegetables [42]. There is increasing evidence that whole foods are more effective in reducing risk of cancer than specific vitamin and mineral supplements, and that some supplements may actually increase risk of cancer [215, 216].

Localised contact with phytochemicals may be an important factor in the aetiology of epithelial cancers of the alimentary system (oral, oesophagus, stomach and bowel). Proposed protective mechanisms include:

- the reduced formation of cancer-promoting substances in the gastrointestinal tract due to antioxidant activity
- the part played by phytochemicals and micronutrients in the detoxification of carcinogenic substances
- functions relating to the containment and destruction of existing cancer cells by means of a variety of physiological processes and improved immunological activity against cancer cells [9, 217].

Vegetables and fruit also provide dietary fibre, the intake of which is thought to reduce the risk of some cancers [9]. However, although a probable decreased risk of colorectal cancer with intake of foods containing dietary fibre has been described in the WCRF report [42], only one [218] of the human trials has shown any benefits of high fibre intakes *per se* on colon cancer or on markers of the risk of colon cancer. It may be that the dietary fibre component of these foods is not solely responsible for any apparent protective effect [9].

2.2.6 Practical considerations: *Eat plenty of vegetables, including different types and colours, legumes/beans, and fruit*

Consuming at least five serves of vegetables per day (75g per serve) is recommended for Australian adults, while amounts recommended for children and adolescents depend on their age and sex. The most recent dietary surveys [13, 44] show that consumption of vegetable and legumes/beans in Australia is generally less than half that recommended for adults and children, and the mix of vegetables consumed also needs to be addressed. To meet the recommended food group intakes, most adults should increase their total consumption of vegetables by more than 30% [10]. A 30% increase in intake of green and *Brassica* vegetables, 140% in red- and orange-coloured vegetables and 90% in other vegetables would be optimal, while consumption of starchy vegetables needs to decrease by 40% [10]. Replacing potatoes with other vegetables would increase the variety of vegetables consumed and provide additional health benefits.

Consuming at least two serves of fruit per day (150g per serve) is recommended for adults, while amounts recommended for children and adolescents depend on age and sex (see Table 2.1 and 2.2). The most recent dietary surveys [13, 44] show that fruit consumption should approximately double to meet recommended intakes [10].

The *Australian Guide to Healthy Eating* provides detailed information on the number of serves and serve sizes of vegetables, legumes/beans and fruit required for different population groups (see tables 2.1 & 2.2).

Australian Government
National Health and Medical Research Council
N H M R C

Table 2.1: Recommended number of serves of vegetables, legumes/beans and fruit per day *

		Number of serves/day of vegetables and legumes/beans	Number of serves/day of fruit
	Age		
Men	19-50	6	2
Men	51-70	5½	2
Women	19-50	5	2
Women	51-70	5	2
Pregnancy	(19–50)	5	2
Lactating	(19–50)	7½	2

* Additional portions of the five food groups or discretionary choices are needed only by people who are taller or more active to meet additional energy requirements.

Source; Australian Guide to Healthy Eating [114]

Table 2.2: Standard serve size equivalents for vegetables, legumes/beans and fruit

Food group	Serve sizes
Vegetables and legumes/ beans	75g (1/2 cup) cooked green or Brassica or cruciferous vegetables
	75g (1/2 cup) cooked orange vegetables
	75g (1/2 cup) cooked dried or canned beans, chickpeas or lentils, no added salt
	75g (1 cup) raw green leafy vegetables
	75g starchy vegetables (e.g. 1 small or 1/2 medium potato, or equivalent of sweet potato, taro, sweet corn or cassava)
	75g other vegetables e.g. 1 small–medium tomato
Fruit	150g (1 piece) of medium-sized fruit e.g. apple, banana, orange, pear
	150g (2 pieces) of small fruit e.g. apricots, kiwi fruit, plums
	150g (1 cup) diced, cooked or canned fruit*
	125mL (1/2 cup) 100% fruit juice ^
	30g dried fruit*,^ e.g. 4 dried apricot halves, 1½ tablespoons of sultanas

* No added sugar
^ Only to be used occasionally as a substitute for other foods in the group

Source; Australian Guide to Healthy Eating [114]

Vegetables and fruit should be stored and prepared properly to maintain nutrient status. Avoid overcooking and wash vegetables to remove microbes and surface debris. Peel or scrub root vegetables (see Chapter 7). Tinned and/or frozen varieties, preferably without added sugar, salt or fat, are nutritious alternatives to raw produce.

2.2.6.1 Pregnant and breastfeeding women

Fruit and vegetable consumption before and during pregnancy makes an important contribution to health outcomes for women and their children. Many women may need to increase their current consumption of these valuable foods as a prospective cohort study found that only about half of pregnant women may be consuming the recommended amounts of fruits and vegetables [219]. Pre-prepared or pre-packaged cut fruit and vegetables should be avoided due to risk of listeriosis [130, 220].

2.2.6.2 Infants

Pureed and mashed vegetables, including legumes/beans, and fruit are important in the diets of infants from around six months of age. Choices should be varied to ensure adequate energy (kilojoule) and nutrient intake.

By 12 months of age, infants should be consuming a wide variety of foods consumed by the rest of the family, having progressed from pureed or mashed foods to foods that are chopped into small pieces. Care should be taken early on to choose foods of a suitable texture. Hard pieces of foods, such as some raw vegetables and nuts, should be avoided as they can cause choking. A variety of tastes and textures may be more appealing. As with all foods, some vegetables may need to be introduced more than 10 times before being accepted [221]. Whole fruit is preferable to fruit juice due to its higher fibre content. Fruit juice is not suitable for infants under six months and like any acidic drink, consumption requires care to avoid dental erosion [133, 222].

2.2.6.3 Children and adolescents

The recommended quantities of vegetables and fruit intakes for children and adolescents vary depending on their age and sex [10]. To meet the dietary recommendations, children and adolescents need to approximately double their overall vegetable consumption [10] and decrease by one third their intake of potatoes. Current fruit intakes by 2-3 year olds are close to the recommended levels but need to increase proportionately with age.

A wide variety of different coloured, textured and tasting vegetables and fruit, both fresh and cooked, should be offered frequently to toddlers and pre-schoolers. Parents and carers can provide model behaviour by consuming a wide range of vegetables and fruit.

Children and adolescents should be encouraged to select a wide variety of vegetables and fruit, at meal times and between meals. In recent years, most Australian states have improved the nutritional quality of food supplied at schools through strategies incorporating a colour-coded system in which vegetables, including legumes/beans, and fruit are classified ‘green’ with the recommendation that children eat plenty [223-230]. Fruit can be ‘fast food’ to help satisfy

increasing appetites. Unless prescribed by a dietitian, special diets which restrict intake of any fruit or vegetables should be avoided for children and adolescents. The intake of energy (kilojoule)-dense hot fried potato chips as a snack or with meals should be limited.

2.2.6.4 Older people

Although most of the emphasis is on the value of dietary patterns rich in vegetables and fruit throughout life, there is still benefit in adopting such habits later in life. In general, older adults tend to consumer higher intakes of fruit and vegetables than younger adults. Due to poor dentition, softer textured or cooked vegetables and fruit may be preferable for some older people. Tinned and/or frozen varieties, preferably without added sugar, salt or fat, are nutritious alternatives to raw produce.

2.2.6.5 Aboriginal and Torres Strait Islanders

Very low intakes of vegetables and fruit have been described among Aboriginal and Torres Strait Islander groups in urban and rural communities [32, 135, 231, 232]. Availability of quality fresh produce can be a particular problem in many remote areas [10, 30]. Frozen and canned vegetables and fruit, plus available traditional plant foods, can be useful in these areas.

2.2.6.6 Vegetarians

Advice regarding vegetables and legumes/beans applies to everyone, including vegetarians. Legumes (and also nuts and seeds) provide valuable sources of iron and other nutrients, including protein. In all vegetarian and plant-based diets and meals, legumes/beans play an important role. Some more restrictive vegetarian diets can be adequate for adults, but may not be suitable for children.

2.3 Grain (cereal) foods (mostly wholegrain)

2.3.1 Setting the scene

Foods originating from grains (cereals) include those from wheat, oats, rice, barley, millet and corn. They range from highly nutrient-dense wholegrain breads and grain (cereal) foods such as oats, to lower-nutrient dense white rice, white bread, pasta and noodles. Excluded are refined grain (cereal) food products with high levels of added sugar, fat and/or salt/sodium, such as cakes.

Key nutrients in wholegrain foods include carbohydrate (starch), protein, dietary fibre, B group vitamins, vitamin E, iron, zinc, magnesium and phosphorus. Other protective components are

fermentable carbohydrates, oligosaccharides, flavonoids, phenolics, phytoestrogens, lignans, protease inhibitors, saponins and selenium [36, 37]. In Australia it is mandatory for wheat flour used in bread making to be fortified with folic acid and thiamin, and for the salt used to be iodised [233].

2.3.2 The evidence for *'grain (cereal) foods'*

The evidence for the association of grain (cereal) foods (mostly wholegrain) with reduced risk of cardiovascular disease, type 2 diabetes and excess weight gain has strengthened since the previous dietary guidelines.

The literature is difficult to interpret because studies use varied definitions of 'wholegrain'. FSANZ applies the term to products which uses every part of the grain including the outer layers, bran and germ even if these parts are separated during processing and regardless of whether the grain is in one piece or milled into smaller pieces [234]. In this review, the most commonly used definition was found to be that of Jacobs et al. (1998) [235] who defined wholegrain foods as those containing 25% or more of wholegrains, whereas some studies included bran cereals as part of the definition of wholegrain and others only examined certain types of grain (cereal) food such as oats.

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines.*

Evidence Statement	Grade
Consumption of cereal foods (especially wholegrains and those with fibre from oats or barley) is associated with a reduced risk of cardiovascular disease in adults.	B
Consumption of 1-3 serves per day of wholegrain cereals is associated with a reduced risk of cardiovascular disease	B
Consumption of cereal foods (especially 3 serves a day of wholegrains) is associated with reduced risk of type 2 diabetes	B
Consumption of 3-5 serves per day of cereal foods (mainly wholegrain) is associated with a reduced risk of weight gain	B
Consumption of 1-3 serves per day of cereals high in fibre is associated with reduced risk of colorectal cancer in adults.	C

2.3.2.1 Cardiovascular disease, type 2 diabetes and excess weight

Cardiovascular disease: There is evidence of a probable association between the consumption of grain (cereal) foods (especially wholegrains and those with fibre from oats or barley) and a reduced risk of cardiovascular disease in adults (Grade B, Section 6.3 in Evidence Report [14]) [236-252]. Almost all the high level trials were conducted with oats, with the evidence of beneficial lowering of levels of LDL and total cholesterol levels. The protective effect was noted with between 1–3 serves per day of wholegrain foods (predominantly oats).

Type 2 diabetes: There is evidence of a probable association between the consumption of grain (cereal) foods (especially wholegrains) and reduced risk of type 2 diabetes (Grade B, Section 6.7 in Evidence Report [14]) [238, 251, 253-260]. The evidence supports three serves per day of wholegrain foods conferring between 21-42% reduction in risk of type 2 diabetes.

Excess weight: There is evidence of a probable association between consumption of 3–5 serves per day of grain (cereal) foods (mainly wholegrain) and reduced risk of weight gain (Grade B, Section 6.6 in Evidence Report [14]) [249, 261-270].

2.3.2.2 Cancer

Colorectal cancer: There is recent evidence suggesting that consumption of 1-3 serves of cereals high in dietary fibre per day is associated with reduced risk of colorectal cancer in adults (Grade C, Section 6.2 in Evidence Report [14]) [185, 271-275]. Although previously the WCRF report noted that the evidence was too limited to draw conclusions, it recently reviewed the evidence and found it convincing that fibre-rich foods offer protection against colorectal cancer [42].

Other cancers: Recent evidence is inconclusive for an association regarding the consumption of grain (cereal) foods and risk of other cancers in adults (Section 6.1 in Evidence Report [14]).

2.3.3 How *eating cereal (mostly wholegrain) foods* may improve health outcomes

Much depends on which wholegrain is being considered [276]. For example, oat β-glucan binds with bile acids, so the liver breaks down more cholesterol to maintain a supply of bile acids. Wheat does not contain these soluble fibres. Also, the slow rate of glucose delivery of oats reduces the requirement for insulin [277]. The slower rate of absorption created by the presence of dietary fibre from some grain (cereal) foods can also influence appetite. For example, oat β-glucan has been shown to have effects on postprandial cholecystokinin levels, decreased insulin response and extended subjective satiety in overweight adults [278]. Other sources of dietary fibre, such as *psyllium* may act in a similar fashion. Grain (cereal) foods also contain starch that may be resistant to digestion in the small intestine (resistant starch) and may help to provide a more

protective environment in the colon, particularly in the context of meals [279]. Note that as some degree of processing is applied to most grains (cereals) to aid digestion, the effects can be significantly influenced by the technology applied in processing and cooking [280].

Because wholegrains contain more nutrients and phytochemicals, concentrated in the bran and germ [281], they are likely to have greater effects than refined grains (cereals). Wholegrains contain phenolic compounds with strong anti-oxidant capacity which may be protective against processes involved in the pathology of diabetes, cardiovascular disease and cancer [282]. Choosing wholegrain options may also assist with satiety and help in not over consuming food beyond energy (kilojoule) requirements.

The previous dietary guidelines discussed the glycaemic index (GI) of a food as a physiologically based classification of carbohydrate-containing foods according to their potential to raise blood glucose. Various factors may affect the GI value of a food, among them the particle size of milled grains, the ratio of amylose to amylopectin, the degree of starch gelatinisation and the presence of other food components such as viscous soluble fibres, fat, protein and organic acids which may limit practical application. Lower GI diets may assist in the management of diabetes [37]. Further research on GI and health outcomes may be required to investigate other potential associations.

The previous guidelines also noted that consumption of dietary fibre assists in maintaining the health and function of the digestive system, for example in preventing constipation [36, 37].

2.3.4 Practical considerations: *Eat grain (cereal) foods, mostly wholegrain*

Consuming at least 4–6 serves of grain (cereal) foods per day is recommended for Australian adults, while the amount recommended for children and adolescents depends on their age and sex (see table 2.3 below), ranging from four serves a day for 2–8 year olds to seven a day for older adolescents. The amount of different grain (cereal) foods which comprise a serve is outlined in the *Australian Guide to Healthy Eating*. One slice of bread, or half a medium roll or flat bread (about 40g), is equivalent to 1/2 cup of cooked rice, pasta, noodles, 2/3 cup of breakfast cereal flakes (30g) or one quarter of a cup of muesli (30g).

The *Australian Guide to Healthy Eating* provides detailed information on the number of serves and serve sizes of grain (cereal) mostly wholegrain foods required for different population groups (see tables 2.3 and 2.4).

Table 2.3: Recommended number of serves of grain (cereal) mostly wholegrain foods per day *

		Number of serves/day of grain (cereal) foods, mostly wholegrain
	Age	
Men	19-50	6
Men	51-70	6
Women	19-50	6
Women	51-70	4
Pregnancy	(19–50)	8½
Lactating	(19–50)	9

* Additional portions of the five food groups or discretionary choices are needed only by people who are taller or more active to meet additional energy requirements.

Source; Australian Guide to Healthy Eating [114]

Table 2.4: Standard serve size equivalents for grain (cereal) mostly wholegrain foods

Food group	Serve sizes
Grain (cereal) foods, mostly wholegrain, such as breads, cereals, rice, pasta, noodles, polenta, couscous, oats, quinoa and barley	1 slice of bread or 1/2 a medium roll or flat bread (about 40g)
	1/2 cup cooked rice, pasta, noodles
	1/2 cup cooked porridge or polenta 2/3 cup breakfast cereal flakes (30g) or 1/4 cup muesli (30g)
	3 crispbreads
	1 crumpet (60g) or 1 small English muffin or scone (35g)
	1/2 cup cooked barley, buckwheat, semolina, cornmeal, quinoa
	1/4 cup flour
	1 slice of bread or 1/2 a medium roll or flat bread (about 40g)

Source; Australian Guide to Healthy Eating [114]

To meet recommended food group intakes [10] adults would require a 30% increase in grain (cereal) foods, comprising a 160% increase in current wholegrain consumption and a 30% decrease in refined grain (cereal) food consumption. For children over the age of 4 years, 20–60% more wholegrain foods and 10–30% less refined cereal foods would be required to meet recommended intakes [10]. Refined grain (cereal) food products with high levels of added sugar, fat and/or salt, such as cakes and biscuits, are classified as discretionary foods and are not included in the grain recommendation. While these can be included in small amounts in *Total Diets* with higher energy (kilojoules) allowances, for the Australian population as a whole their intake would need to be reduced substantially.

The suite of models which informed the update of the *Australian Guide to Healthy Eating* [10] allows for variation in choice and amount of grain (cereal) foods to accommodate a range of cuisines. A

variety of grain (cereal) food choices allow for different forms of dietary fibre and complex carbohydrates. Reading the labels on processed grain (cereal) foods such as rice and pasta is important to check that added sodium, sugar and/or fat (in particular saturated fat) are kept to a minimum.

2.3.4.1 Pregnant and breastfeeding women

As periconceptional folic acid intake helps protect against neural tube defects in the developing foetus [283], the mandatory fortification of flour used for breadmaking in Australia with folic acid [284] provides an additional reason for women of reproductive age to consume bread. However, for women planning a pregnancy and during the first three months of pregnancy, a daily folic acid supplement (0.4 mg) is recommended in addition to eating foods which are naturally rich in folate or are fortified with folic acid [285, 286].

Pregnant and breastfeeding women have increased iodine requirements. As most leavened bread in Australia is made with iodised salt, this contributes to total iodine intake. However, this does not replace the need for iodine supplements (150 mcg/day) for women planning a pregnancy, throughout pregnancy and while breastfeeding [103].

2.3.4.2 Infants

Cereal should be one of the first foods offered to infants at around six months of age. Iron-enriched infant cereals are recommended at this stage.

2.3.4.3 Older people

A high intake of wholegrain forms of grain (cereal) foods may not be well tolerated if there is a problem with dentition. Softer varieties such as finely milled wholemeal bread and/or other fibre-enriched foods may be suitable.

2.3.4.4 Vegetarians

Wholegrain foods are particularly important in vegetarian diets as a source of iron and zinc.

2.4 Lean meat and poultry, fish, eggs, nuts and seeds, and legumes/beans

2.4.1 Setting the scene

Lean meats and poultry, fish, eggs and plant-based alternatives such as legumes/beans, nuts and seeds are an important component of Australian cuisine, culture and lifestyle. The 'lean meat and alternatives' food group is diverse, both nutritionally and biologically. The foods in this group have traditionally been seen as 'protein-rich', but they also provide a wide variety of other nutrients which may be more important in the typical high-protein Australian diet. Important nutrients include iodine, iron, zinc, vitamins, especially B12, and essential fatty acids including long chain omega-3 fatty acids. All seafood contains long chain omega-3s. Grass-fed meats, poultry and some eggs are also sources of these essential fatty acids. Evidence of the health benefits of lean meats and alternatives is consistently recognised in international dietary guidelines [36, 37, 143, 144].

Processed and cured meats can be high in added salt and saturated fat and are not recommended as substitutes for unprocessed meat. Moderate amounts of lean poultry and eggs can be included in the diet within the overall recommended quantities for this whole food group. Eggs are an alternative to meat, a relatively inexpensive source of protein and are versatile foods. Note that although pork is not considered red meat for marketing purposes in Australia, it is classified as red meat in the international literature, and so has been considered as red meat for the purpose of the *Guidelines* [10].

Fish and other seafood are central foods in the cuisines of many traditional cultural and religious groups, and are popular foods in Australian society. Fish is nutritious, providing energy (kilojoules), protein, selenium, zinc, iodine, vitamins A and D (some species only), and long-chain omega-3 fatty acids. Evidence of the health benefits of fish consumption is consistently recognised in international dietary guidelines [36, 37, 143, 144].

Nuts and seeds are rich in energy (kilojoules) and nutrients, reflective of their biological role in nourishing plant embryos to develop into plants. In addition to protein and dietary fibre, they contain significant levels of unsaturated fat, although this varies within the category. Nuts are also rich in polyphenols and micronutrients, including folate, several valuable forms of vitamin E, selenium, magnesium and other minerals. They are useful alternatives to meat, fish and eggs, and are particularly important in plant-based, vegetarian and vegan meals and diets.

Legumes/beans, including lentils, tofu and tempe, provide a valuable and cost efficient source of protein, iron, some essential fatty acids, soluble and insoluble dietary fibre and micronutrients. They are valuable inclusions in the diet, and are especially useful for people who consume plant-based or vegetarian diets or meals [10].

2.4.2 The evidence for *'lean meat and poultry, fish, eggs, nuts and seeds, and legumes/beans'*

In the following studies, serve sizes of the different foods are as included in the *Australian Guide to Healthy Eating* (see also Section 2.4.4, table 2.5).

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines.*

Evidence Statement	Grade
Consumption of greater than100-120g/d red meat is associated with an increased risk of colorectal cancer.	B
Consumption of fish more than once per week is associated with a reduced risk of developing dementia in older adults.	B
Consumption of red meat is associated with risk of renal cancer.	C
Consumption of at least 2 serves a week of fish is associated with reduced risk of mortality from cardiovascular disease, and with reduced incidence of cardiovascular disease.	C
Consumption of fish at least twice a week is associated with a reduced risk of stroke.	C
Consumption of fish 2 or more times per week is associated with reduced risk of age-related macular degeneration.	C
Consumption of nuts (65-110 g per day) reduces cholesterol levels.	C

2.4.2.1 Lean meat

Past literature reporting on the health benefits and/or risks of consuming meat has been predominantly based on studies investigating the nutrient effects related to (for example) iron, protein or zinc in isolation [37] or the ability of the body to absorb nutrients rather than looking at the whole food. Since 2003, the evidence linking meat consumption and increased risk of disease has strengthened in some areas and remains unclear in others. The evidence is difficult to

interpret because of widely varying definitions of 'meat'. Some studies include only unprocessed red meat. Others may include some or all of a variety of processed meats, including smoked, salted and chemically preserved foods, with meat within dishes such as pizza, lasagna or casseroles variously included or excluded. The poor definitions partly explain the often inconsistent findings in relation to health effects, with several large cohort studies and some Asian studies failing to adequately disaggregate possibly different effects of unprocessed red meat and processed meats. In particular, the definition of red meat varies greatly between studies.

2.4.2.1.1 Lean meat: Cardiovascular disease, type 2 diabetes and excess weight

An insufficient number of recent studies investigating the relationships between consumption of meat and cardiovascular disease, type 2 diabetes and excess weight were identified in the literature review to develop evidence statements [14]. A large cohort trial that found modest increases in total mortality, cardiovascular mortality and cancer mortality with red and processed meat intakes [287] was not included due to lack of clarity over the inclusion of processed meats, liver and sausages with unprocessed red meat.

2.4.2.1.2 Lean meat: Cancer

Colorectal cancer: There is evidence of a probable association between consumption of red meat and increased risk of colorectal cancer (Grade B, Section 4.7 in Evidence Report [14]) [42, 288-296]. The WCRF reported a convincing relationship between red and processed meat and increased risk of colorectal cancer [42]. Several studies from Asian countries showed no increased risk of colorectal cancer associated with low intakes of red meat such as 27g per day [294] and 42g per day [292].

Renal cancer: The evidence suggests that consumption of red meat is associated with an increased risk of renal cancer (Grade C, Section 4.6 in Evidence Report [14]).

Bladder and prostate cancer: The evidence suggests that consumption of red meat one to six times per week is not associated with risk of bladder cancer (Grade C, Section 4.1 in Evidence Report [14]) [42, 297, 298]. The evidence suggests that consumption of red meat is not associated with risk of prostate cancer (Grade C, Section 4.3 in Evidence Report [14]) [42, 299, 300].

Pancreatic cancer: A review of the current evidence suggests that consumption of 30-200 grams of red meat per day is not associated with risk of pancreatic cancer (Grade C, Section 4.2 in Evidence Report [14]) [42, 301].

Other cancers: Recent evidence is inconclusive for an association regarding the consumption of red meat and breast and lung cancer (Section 4.4, & 4.5 in Evidence Report [14]).

Given these risks, advice is provided on how much meat can be eaten to maximize the health benefits of consuming meat, while minimizing the health risks- see Section 2.4.4.

2.4.2.2 Lean poultry

2.4.2.2.1 Lean poultry: Cardiovascular disease, type 2 diabetes and excess weight

An insufficient number of recent studies investigating the relationships between consumption of poultry and cardiovascular disease, type 2 diabetes and excess weight were identified in the literature review to develop evidence statements [14].

2.4.2.2.2 Lean poultry: Cancer

Recent evidence examining an association between poultry consumption and breast or colorectal cancer is inconclusive (Section 10.1 & 10.2 in Evidence Report [14]).

2.4.2.3 Fish

The evidence regarding the health benefits of fish has strengthened since 2002. People who regularly consume diets high in fish tend to have lower risks of a range of conditions, including cardiovascular disease, stroke, and macular degeneration, and dementia in older adults.

Early literature focused on evidence indicating that fish oils (and in particular long chain omega-3 fatty acids) provided specific health benefits for brain development and function and cardiovascular health, and extrapolated this to fish as the predominant food containing these fatty acids [37]. Recent research continues to be dominated by pharmacological studies of the effects of nutrients derived from fish, particularly delivered in fish oils. The evidence obtained from the literature considers relationships with the consumption of fish *per se*, so studies of fish oil or omega-3 supplements are not reported in the evidence statements below. However, the overall chapter examines evidence relating to omega-3 fats, bearing in mind that these fats can also be delivered in foods other than fish.

The evidence provided below for health benefits relates consistently to fish in the form of finfish from marine or freshwater sources, either farmed or wild. Few studies have investigated the effects of other types of seafood. As for red meat, the distinction between different preparation methods and different fish types is not always well addressed in the literature and may contribute to a lack of consistency in some instances.

2.4.2.3.1 Fish: Cardiovascular disease, type 2 diabetes and excess weight

Cardiovascular disease: Evidence suggests that consuming at least two serves of fish per week is associated with reduced incidence of cardiovascular disease (particularly myocardial infarction) (Grade C, Section 9.1 in Evidence Report [14]) [302-305] and with reduced risk of mortality from cardiovascular disease (Grade C, Section 9.1 in Evidence Report [14]) [302, 304, 306-313].

The evidence suggests that consumption of fish at least twice a week is associated with a reduced risk of stroke (Grade C, Section 9.5 in Evidence Report [14]) [305, 314-316].

Only a small number of case-control and cohort studies are available, with inconsistent results, to examine the relationship between fish consumption and the incidence of cardiac arrest, heart failure, atrial fibrillation and high blood lipids.

2.4.2.3.2 Fish: Cancer

Evidence of an association between fish consumption and breast, colorectal, prostate and renal cancer was examined however no conclusive associations could be drawn (Section 9.6, 9.7, 9.8 & 9.9 in Evidence Report).

2.4.2.3.3 Fish: Other conditions

Dementia: The recent body of evidence demonstrates that it is probable that the consumption of fish more than once per week is associated with a reduced risk of developing dementia in older adults (Grade B, Section 9.2 in Evidence Report [14]) [303, 317-324].

Depression: The recent evidence suggests that consumption of at least one serve of fish per week is not associated with reduced risk of depression (Grade C, Section 9.3 in Evidence Report [14]) [325-329].

Macular degeneration: The recent evidence suggests that eating fish two or more times a week is associated with reduced risk of age-related macular degeneration (Grade C, Section 9.4 in Evidence Report [14]) [330].

2.4.2.4 Eggs

Since 2003 the evidence associating egg consumption with health outcomes has not changed greatly (Section 11 in Evidence Report [14]). There do not appear to be any increased health risks associated with consumption of eggs [331-333]. There is recent evidence which suggests that consumption of eggs every day is not associated with increased risk of coronary heart disease (Grade C, Section 11.1 in Evidence Report [14]) [248, 334-351].

2.4.2.5 Nuts and seeds

Consumption of nuts and seeds may help reduce the risk of heart disease and is not associated with weight gain if total energy (kilojoule) intake is controlled. Since the previous dietary guidelines, the evidence for the benefits of consumption of nuts and seeds on heart disease has been shown to be related to favourable effects on serum cholesterol. The evidence on lack of association with weight gain is a new development.

2.4.2.5.1 Nuts and Seeds: Cardiovascular disease, type 2 diabetes and excess weight

Cardiovascular disease: Evidence suggests that consumption of nuts (65–110g per day) is associated with a reduction in serum cholesterol, a surrogate marker for cardiovascular disease (Grade C, Section 8.2 in Evidence Report [14]) [352-356].

Excess weight: Evidence suggests that the consumption of nuts (65–110g per day) does not lead to weight gain, at least in the short term (Grade C, Section 8.1 in Evidence Report [14]) [353-358].

2.4.2.5.2 Nuts and Seeds: Cancer

No recent studies investigating the association of consumption of nuts and seeds were identified [14].

2.4.2.6 Legumes/beans

Few studies on legumes/bean foods, other than soy foods, were identified in the literature review to inform the review of these dietary guidelines. For the evidence regarding legumes/beans, see Section 7 in the Evidence Report [14].

2.4.3 How eating *lean meat and poultry, fish, eggs, nuts and seeds, and legumes/beans* may improve health outcomes

This is such a broad and diverse group of foods that a variety of possible mechanisms may influence the effects of their consumption.

Smoked, salted and chemically preserved foods have properties that may be responsible for increased health risks. Endogenous formation of nitrous compounds has been suggested as a possible link between red meat and colorectal cancer [359], but this remains a hypothesis. As with other areas of diet and disease risk, an individual's dietary pattern may be more relevant than a direct effect from a single component [360, 361].

It has been suggested that haem iron or other components of meat such as saturated fats, or other dietary and lifestyle factors associated with meat intake, may be relevant factors for further study [362]. Previous evidence related to saturated fat in red meat and increased risk of cardiovascular disease may not be relevant for lean Australian meats.

The protective effect of fish consumption on cardiovascular disease is thought to be mediated through the influence of specific nutrients such as long-chain n-3 fatty acids. These essential fatty acids exert their physiological effect by altering cell membrane composition, fluidity, receptors and membrane-bound enzymes, gene expression and eicosanoid production (see Chapter 3).

Nut consumption provides benefits by enhancing anti-inflammatory processes [363] and lowering serum cholesterol possibly due to the presence of phytosterols, which reduce cholesterol re-absorption [364], and/or the effects of shifting dietary fat quality, notably replacing saturated with unsaturated fat. Nut consumption is also associated with increased levels of adiponectin which has anti-inflammatory and anti-atherogenic properties [365]. Early work suggests that the delivery of components such as tocopherols and phenolic acids may help to reduce lipid peroxidation and oxidative DNA damage, and there is some indication that walnuts with a relatively high content of the amino acid L-arginine may have an effect on vasodilation through nitrous oxide pathways [363]. Proposed mechanisms for effects on weight control include increased satiety, increased faecal fat excretion, increased thermogenesis [366] and increased fat oxidation [367].

2.4.4 Practical considerations: *Lean meat and poultry, fish, eggs, legumes/beans and nuts/seeds*

The most recent dietary survey data for adults in Australia showed that daily mean consumption of meat, poultry and game was 200g for men and 120g for women [44]. Adult men and women daily consume 99g and 54g of red meat respectively [44].

The recommended consumption of lean meat and poultry, fish, eggs and/or alternatives for both children and adults is 1–3 serves a day depending on age. During pregnancy, 3–4 serves a day are recommended to provide additional iron and zinc.

To enhance dietary variety and reduce some of the health risks associated with consuming meat, up to a maximum of 455g per week (1 serve or 65g per day) of lean meat is recommended for Australian adults.

The *Australian Guide to Healthy Eating* provides detailed information on the number of serves and serve sizes of lean meat, poultry, fish, eggs, legumes/beans and nuts/seeds required for different population groups (see tables 4.5 & 4.6).

Table 2.5: Recommended number of serves of lean meat, poultry, fish, eggs, legumes/beans and nuts/seeds per day *

		Number of serves/day of lean meat, poultry, fish, eggs, legumes/beans and nuts/seeds per day
	Age	
Men	19-50	3
Men	51-70	2½
Women	19-50	2½
Women	51-70	2
Pregnancy	(19–50)	3½
Lactating	(19–50)	2½

* Additional portions of the five food groups or discretionary choices are needed only by people who are taller or more active to meet additional energy requirements.

Source; Australian Guide to Healthy Eating [114]

Table 2.6: Standard serve size equivalents for lean meat, poultry, fish, eggs, legumes/beans and nuts/seeds

Food group	Serve sizes
Lean meat and poultry, fish, eggs, nuts and seeds, and legumes/ beans	65g cooked lean red meats (e.g. beef, lamb, pork, venison or kangaroo) or 1/2 cup of lean mince, 2 small chops, 2 slices of roast meat (about 90 – 100g raw weight)
	80g cooked poultry (about 100g raw weight) e.g. chicken, turkey
	100g cooked fish fillet (about 115g raw weight) or 1 small can of fish, no added salt, not in brine
	2 large eggs (120g)
	1 cup (170g) cooked dried beans, lentils, chickpeas, split peas, or canned beans
	170g tofu
	30g nuts or seeds or nut/seed paste, no added salt^

^ Only to be used occasionally as a substitute for other foods in the group

Source; Australian Guide to Healthy Eating [114]

To meet recommended intakes, omnivorous adults in Australia would need to consume 40% more 'poultry, fish, seafood and eggs or legumes/beans, nuts or seeds' but omnivorous men would need to consume around 20% less red meat than currently [10]. For children 2–16 years of age, around 30–85% more 'poultry, fish, seafood and eggs or legumes/beans, nuts or seeds' and around 25–70% more red meat would be required above current intakes to meet recommendations. Exact quantities depend on age and sex [10].

Depending on age and sex, health benefits may be seen with consumption of 1.4–2.8 serves of fish per week (140–280g per week) for adults, with proportionately less for adolescents and children. The most recent dietary survey data available for adults showed that mean weekly consumption of fish and seafood was 168g for men and 119g per week for women (ABS 1999 [44] cited in NHMRC 2011 [10]). To meet recommended food group intakes, fish consumption will need to increase by more than 40%, particularly for men [10]. The extent to which Australian fish populations are sufficient to meet the guideline advice needs consideration. The previous dietary guidelines note that inclusion of around 2-3 serves of fish per week may not be attainable or sustainable depending on population levels [37]. Information from the Department of Agriculture, Fisheries and Forestry shows that Australia has become a net importer of fish products over the past decade, and several industry initiatives have now been developed to address the sustainability of fish stocks in Australia [368].

Previous reported intakes of nuts and seeds in Australia have been very low (only around 4g per day for adults) [44]. Consumption may have risen since. The dietary models developed for these guidelines include modest amounts of nuts and seeds. However, because intakes have been low, this would require tripling the intake for children over 8 years of age and increasing adult intakes substantially, even in omnivore *Foundation Diet* models [10].

Nuts and seeds can be included in the diet in a variety of ways, including as snacks, in dishes (for example, added to salads, vegetables, various main course dishes and breakfast cereals) and in food products such as breads and spreads.

2.4.4.1 Pregnant and breastfeeding women

Lean red meat is a good source of protein, iron and zinc for pregnant and breastfeeding women, although raw or undercooked meat, chilled pre-cooked meats, and pate and meat spreads are best avoided due to risk of listeriosis (see Chapter 7) [130, 220]. While meat is a good source of iron, iron deficiency in pregnancy is common in Australia and iron supplements may be needed. Pregnant women should check with their doctor.

Maternal consumption of fish during pregnancy is likely to have a number of health benefits for women and their children. However the fish should be low in mercury. FSANZ provides guidance for pregnant women on suggested amounts and frequency of consumption of particular fish species [131, 284]. Pregnant women are advised to consume no more than one serve (150g) per fortnight of shark, marlin or broadbill/swordfish, and no other fish that fortnight, or one serve (150g) per week of orange roughy (deep sea perch) and no other fish that week [130, 131].

Pregnant women should avoid eating foods containing raw eggs, because of risk of salmonella [220].

Pregnant and breastfeeding women do not need to avoid consuming nuts for fear of causing an allergic reaction in their babies. Only women who are allergic to these foods themselves need to avoid them [362, 369-372].

2.4.4.2 Infants

Foods from the meats and alternatives group are important in the diets of infants over the age of around six months. In particular, this group of foods helps meet infants' increased needs for iron, zinc and protein. The food should be initially pureed before serving. For all infants, special care is required to adequately de-bone fish.

Previously nuts were often restricted for infants and children because of the risk of inducing allergy. However this has not been proven in nutrition studies. The texture should be suitable (for example, peanut butter/paste). Only children over three years should be offered whole nuts due to the risk of choking.

2.4.4.3 Children and adolescents

The demands of growth increase the need for iron, protein and many nutrients found in this food group.

2.4.4.4 Women of child-bearing age

Menstrual loss doubles the iron requirements of young women compared to young men [37], although that increased requirement is lessened for women taking the oral contraceptive pill. Lean meat and some types of seafood are excellent sources of iron [10].

2.4.4.5 Older people

Lean meat and poultry, fish, eggs, legumes/beans and nuts and seeds are nourishing foods for older adults and should be included in the diet as a ready source of protein to help maintain muscle mass [10]. People with poor dentition should be advised to choose forms of food that are easier to eat, such as softer foods, soups and casseroles.

Whole nuts may not be well tolerated where there is a problem with dentition. In these cases, the inclusion of ground nuts, nut pastes or nut meal in dishes may be more appropriate.

2.4.4.6 Vegetarians

For several nutrients, including iron, calcium and vitamin B12, animal foods are highly bioavailable sources and care needs to be taken if these foods are excluded. Eggs and milk, yoghurt and cheese products can supply calcium and vitamin B_{12} in lacto-ovo vegetarian diets.

For lacto-ovo vegetarian diets, the Modelling System used a ratio of a 5:1:1 'legume: egg: nuts/seeds' mixed food group. As comparable data was not available for Australia, this was based on the United States Department of Agriculture (USDA) recommendations for proportions of these foods that would provide an adequate amino acid balance [10]. As no Australian national data were available about choices within food categories for lacto-ovo vegetarians, the same proportions within food groups (for example, for vegetables, fruit, cereals) were used as for the equivalent omnivore group. For children consuming vegetarian diets, it should be noted that iron was limiting in the diets as modelled [10].

Nuts and seeds (and legumes/beans) are important foods in vegetarian dietary patterns and meals without meat as they can help provide an alternative source of nutrients. For lacto-ovo vegetarian diets, refer to the Food Modelling document [10].

2.4.4.7 Food safety

Fish that have been exposed to heavy metals, dioxins and dioxin-like polychlorinated biphenyls (PCBs) from industrial pollution of marine and freshwater environments can present a toxicological food safety risk to human biological systems if consumed in sufficiently large amounts. Although fish caught in specific locations could lead to high human exposure - for example, west of the Sydney Harbour Bridge [373] - exposure in Australia generally is low [374]. The FAO and WHO held an expert consultation on this issue and concluded in general that the disadvantages of foregone health benefits from fish consumption outweigh the risks of increased exposure to heavy metals, dioxins and PCBs, although it was acknowledged that close monitoring and evaluation of the fish supply and dietary exposure is needed [375].

2.5 Milk, yoghurt, cheese and/or alternatives (mostly reduced fat)

2.5.1 Setting the scene

In past dietary guidelines these foods have been referred to as dairy foods, but greater specificity is required to reflect definitions in the relevant literature (for example butter, cream and ice-cream are not included). Milks, cheeses and yoghurts are most frequently produced from cow's milk in

Australia, but other sources include milk and products from goats, sheep and soy and various cereals. Milk substitutes that are not fortified with calcium and other nutrients are not included in this food group.

Milk, cheese and yoghurt are a good source of many nutrients, including calcium, protein, iodine, vitamin A, vitamin D, riboflavin, vitamin B12 and zinc. These foods provide calcium in a readily absorbable and convenient form. The proportion of total fat and saturated fat content in some milk, cheese and yoghurts has led to the recommendation that reduced fat varieties should be chosen on most occasions [10]. However reduced fat varieties of milks are not suitable as a milk drink for children under the age of two due to energy requirements for growth.

Alternatives to milk, cheese and yoghurt include calcium-enriched legume/bean milk products such as calcium enriched soy drinks. Information about legumes/beans is included with vegetables and meat and alternative groups above.

2.5.2 The evidence for *'milk, yoghurt, cheese and/or alternatives'*

The evidence for the health benefits of consumption of these dairy foods (mainly reduced fat varieties) has strengthened since the previous Dietary Guidelines [37], however the evidence base primarily comprises small, short term studies with varied definitions of dairy foods. The evidence for the relationship between foods containing calcium and increased bone density in post-menopausal women was not re-examined because it was regarded as an accepted relationship [37].

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines*.

Evidence Statement	Grade
Consumption of at least 2 servings per day dairy foods (milk, yoghurt, and cheese) is associated with reduced risk of ischemic heart disease and myocardial infarction.	B
Consumption of 2 or more servings of dairy foods per day is associated with reduced risk of stroke.	B
Consumption of 3 servings of low fat dairy foods is associated with reduced risk of hypertension	B

Evidence Statement	Grade
Consumption of more than 1 serving of dairy per day, especially milk, is associated with a reduced risk of colorectal cancer.	B
Consumption of 3 or more servings of milk per day is not associated with risk of renal cell cancer.	B
Consumption of 3 servings of any milk, cheese or yoghurt products a day is associated with reduced risk of hypertension	C
Consumption of 2-4 serves of dairy foods per day is associated with reduced risk of metabolic syndrome	C
Consumption of at least 1.5 servings of dairy foods (milk, yoghurt, cheese) per day is associated with reduced risk of type 2 diabetes.	C
Consumption of more than 1 serving of milk per day is associated with reduced risk of rectal cancer.	C
Consumption of dairy products (particularly milk) is associated with improved bone mineral density	C

2.5.2.1 Cardiovascular disease, type 2 diabetes and excess weight

Cardiovascular disease: It is probable that the consumption of at least two servings per day of dairy foods (milk, cheese and yoghurt) is associated with reduced risk of ischemic heart disease and myocardial infarction (Grade B, Section 5.3 in Evidence Report [14]) [376].

It is probable that the consumption of two or more servings of dairy foods per day (milk, cheese and yoghurt) is associated with reduced risk of stroke (Grade B, Section 5.4 in Evidence Report [14]) [376, 377] particularly reduced fat varieties.

It is also probable that consumption of three servings of low-fat dairy foods (milk, cheese and yoghurt) is associated with reduced risk of hypertension (Grade B, Section 5.5 in Evidence Report [14]). The evidence also suggests that consumption of three servings of any milk, cheese or yoghurt products per day is associated with reduced risk of hypertension (Grade C, Section 5.5 in Evidence Report [14]) [378-382].

Type 2 diabetes: The evidence suggests that consumption of 2-4 serves of dairy foods (milk, cheese, yoghurt) per day is associated with reduced risk of metabolic syndrome (Grade C, Section 5.7 in Evidence Report [14]) [376, 383] and that consumption of at least 1.5 serves of milk, cheese and yoghurt per day is associated with reduced risk of type 2 diabetes (Grade C, Section 5.6 in Evidence Report [14]) [376, 382, 384].

Excess weight: The evidence suggests that consumption of milk is not associated with weight change or risk of obesity in adults (Grade C, Section 5.8 in Evidence Report [14]) [382, 385-389] or with BMI or change in BMI in childhood (Grade C, Section 5.9 in Evidence Report [14]) [265, 390-394].

2.5.2.2 Cancer

Rectal and colorectal cancer: Recent evidence suggests it is probable that consumption of more than one serving of dairy foods per day (especially milk) is associated with reduced risk of colorectal cancer (Grade B, Section 5.11 in Evidence Report [14]) [292, 395, 396]. The evidence suggests that consumption of more than one serving of milk per day is associated with reduced risk of rectal cancer (Grade C, Section 5.12 in Evidence Report [14]) [395-397].

Renal cell, breast and endometrial cancer: It is probable that consumption of three or more servings of milk per day is not associated with risk of renal cell cancer (Grade B, Section 5.13 in Evidence Report [14]) [398]. There is evidence to suggest that mean consumption of one serving of dairy food (milk cheese, yoghurt) per day is not associated with the risk of breast cancer (Grade C, Section 5.15 in Evidence Report [14]) (from assessment of studies in Alvarez-Leon et al. 2006 [399]; Missmer et al. 2002 [400]) and that consumption of dairy food (milk, cheese, yoghurt) is not associated with risk of endometrial cancer (Grade C, Section 5.16 in Evidence Report [14]) [401].

Prostate cancer: Recent evidence suggesting an association regarding milk and prostate cancer is inconclusive (Section 5.14 in Evidence Report [14]).

2.5.2.3 Other conditions

The traditional nutritional rationale for the inclusion of dairy foods such as milk, cheese and yoghurt is their high calcium content and the positive relationship between calcium and bone mass [9].

Bone mineral density: Recent evidence suggests that consumption of dairy foods (particularly milk) is associated with improved bone mineral density (Grade C, Section 5.1

in Evidence Report [14]) [399, 402-407] but this is contradicted by evidence suggesting that less than one serving of milk per day during adult life is not associated with risk of osteoporotic or hip fracture (Grade C, Section 5.2 in Evidence Report [14]) [408, 409].

2.5.3 How *drinking milk and eating yoghurt, cheese and/or alternatives* may improve health outcomes

Two proposed mechanisms link the consumption of milk, yoghurt and cheese products with a reduction in cardiovascular risk. Firstly, the consumption of milk, yoghurt and cheese products has been linked to an increase in the levels of high density lipoprotein (HDL) cholesterol [410, 411]. Secondly, there is evidence of an inverse relationship between milk, yoghurt and cheese consumption (especially milk) and blood pressure [412] which might be mediated by calcium modulation of endothelial function [413]. Calcium from foods may be preferable to calcium from some supplements. A recent meta-analysis of the effect of calcium supplementation on myocardial infarction and cardiovascular events [414] suggested that calcium supplements, without co-administered vitamin D, were associated with an increased risk of myocardial infarction. The same group have more recently reported a similar effect from calcium supplements with vitamin D [415]. However, the effect of an equivalent dose of calcium from milk, yoghurt and cheese products has a much smaller effect than calcium supplements on the proposed risk factor, namely raised serum calcium levels [416].

There may be a negative association between calcium and blood pressure although this is variable, depending on other dietary factors, and it has been suggested that calcium supplementation may lower blood pressure only in people with a relatively high salt intake [417].

Several reasons have been suggested for the lack of an association between a low consumption of milk and increased risk of osteoporotic fracture of the hip. For example it has been suggested that women who consumed higher quantities of milk chose to do so because they were known to be at an elevated risk of osteoporosis or that milk intake during childhood was more important [418].

A randomized control trial of overweight children found that 200mL of milk per day combined with nutrition education reduced consumption of sugar-sweetened drinks and increased lean body mass [419].

2.5.4 Practical considerations: *Milk, yoghurt, cheese and/or alternatives*

Consuming at least two and a half serves of milk, cheese, yoghurt and/or alternatives (mostly reduced fat) is recommended for Australian adults, while the recommended food group intakes for children and adolescents depend on age and sex [10]. Varieties of cheese which are lower in salt are also preferable (see Section 4.4).

The *Australian Guide to Healthy Eating* provides detailed information on number of serves and serve sizes of milk, yoghurt, cheese and/or alternatives required for different population groups (see tables 2.7 and 2.8 below).

Table 2.7: Recommended number of serves of number of serves of milk, yoghurt, cheese and/or alternatives (mostly reduced fat) per day *

		Number of serves/day of milk, yoghurt, cheese and/or alternatives (mostly reduced fat) per day
	Age	
Men	19-50	2½
Men	51-70	2½
Women	19-50	2½
Women	51-70	4
Pregnancy	(19–50)	2½
Lactating	(19–50)	2½

* Additional portions of the five food groups or discretionary choices are needed only by people who are taller or more active to meet additional energy requirements.

Source; Australian Guide to Healthy Eating [114]

Table 2.8: Standard serve size equivalents for milk, yoghurt, cheese and/or alternatives (mostly reduced fat)

Food group	Serve sizes
Milk, yoghurt, cheese and/or alternatives (mostly reduced fat)	250mL (1 cup) milk – fresh, UHT long life or reconstituted dried
	125mL (1/2 cup) evaporated unsweetened milk
	200g (3/4 cup or 1 small carton) yoghurt
	40g (2 slices, or 4x3x2cm piece) hard cheese (e.g. cheddar)
	120g ricotta cheese

Source; Australian Guide to Healthy Eating [114]

Analysis of the *1995 National Nutrition Survey* [44] included a category called 'milk products and dishes' which included items such as milks, yoghurts, cheese, cream, ice-cream and milk-based custards, as well as milk substitutes such as soy drinks and soy cheeses. On the day of the survey, 93% of subjects consumed foods from this category, the average intake being 322g for adult males and 258g for adult women. Older men consumed less of these foods than younger men, but consumption remained relatively stable in women across age groups.

To meet recommended food group intakes for adults [10] consumption of milk, yoghurt and cheese products would need to approximately double overall, with a halving of higher fat varieties and a four-fold increase in reduced fat varieties of milk, cheese and yoghurt. Alternatives, such as calcium-enriched soy drinks could be substituted for these dairy foods.

2.5.4.1 Pregnant and breastfeeding women

Milk, yoghurt and cheese product intake during pregnancy has clear benefits for both women and their babies. Reduced-fat milk, yoghurt and cheese products are recommended during pregnancy and while breastfeeding [220, 362].

Pregnant women are advised to avoid consuming unpasteurised dairy products and soft, semi-soft and surface-ripened cheeses (for example, brie, camembert, ricotta, fetta and blue cheeses) due to risk of listeriosis [130, 420].

2.5.4.2 Infants

Cow's milk should not be given as a drink to infants under 12 months of age. However cow's milk may be served in small quantities as custards and desserts. All milk given to infants over 12 months should be pasteurised. Any milk given to infants under 12 months should be specially prepared infant formula.

Children under two years are growing rapidly and have relatively high energy (kilojoule) needs. Reduced fat milks are not recommended as a main milk food for this age, but can be used after two years.

2.5.4.3 Children and adolescents

Milk is an important source of calcium and protein for growing children and adolescents. The decreased intake of milk and milk products among adolescent girls is of concern, and can lead to low calcium intake in this group. Diets restricting intake of milk and milk products are not generally suitable for growing children and adolescents. Suspected lactose intolerance in children and adolescents should be confirmed by a medical practitioner.

2.5.4.4 Older people

For older Australians, milk is a good source of protein, calcium and energy (kilojoules). All milks, including full cream milk, are good choices for older people whose appetite is reduced or who have lost weight, unless medically advised otherwise.

2.5.4.5 People with lactose intolerance

The rate of lactose intolerance is relatively high in many Asian communities compared with Caucasians. High rates of lactose intolerance have been described among Aboriginal and Torres Strait Islander groups [421, 422]. Lactose-intolerant adults and children often avoid milk and milk products, however up to 250mL of milk may be well tolerated [423]. Cheese contains little lactose

and the lactose in yoghurt is partially broken down by bacteria that thicken the yoghurt. Lactose-free dairy products, such as specialised milks, are also available.

2.5.4.6 Vegetarians

The previous dietary guidelines addressed vegetarian diets and their influence on calcium needs because of the relatively high oxalate and phytate content. On balance, lacto-ovo-vegetarians appear to have calcium intakes similar to those of omnivores. Vegetarians who avoid milk and calcium-fortified soy products should seek advice about whether they need to take calcium supplements [37].

2.6 Water

2.6.1 Setting the scene

Water is essential for life. It is required for digestion, absorption and transportation, as a solvent for nutrients, for elimination of waste products and to regulate body temperature. All biochemical reactions occur in water.

Water is lost from the body in sweating (from 100mL to several 1000mL/day), insensible losses from the lungs and skin (approximately 800mL/day), faecal losses (200mL/day) and urine. A minimal urine volume to excrete solute load is estimated at 500mL/day but may need to be much greater in older people due to declining kidney function [424]. Excluding obvious sweating, the normal turnover of water has been estimated at approximately 4% of total body weight in adults. In a 70kg adult, this is equivalent to 2,500-3,000mL/day. Some of this turnover will come from the water contained in foods. Ambient temperature, physical activity and body size all influence requirements, creating wide variations in daily needs for water. Losses from the lungs and skin increase 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 [425, 426].

The body can compensate in the short term for over- or under-hydration [9], there are no data on specified hazards from usual consumption. Excess water intake can cause hyponatraemia, but this is rare in the general population. In general, self-regulation of excess water consumption occurs in healthy people in temperate climates.

2.6.2 The evidence for *'drink water'*

Consumption of adequate water is essential for human life. Given this, no recent studies investigating the relationship between water consumption and health outcomes were identified in

the recent review [14]. Many commonly consumed fluids provide water, however they may also be acidic e.g. tea and coffee or contain added sugar, alcohol or caffeine. Recent studies which investigated intake of these fluids and health outcomes are reviewed below.

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines.*

Evidence Statement	Grade
Consumption of sugar sweetened beverages is associated with increased risk of weight gain in adults and children.	B
Consumption of coffee of 4 or more cups per day is associated with reduced risk of type 2 diabetes.	B
Consumption of green and black tea is associated with reduced risk of stroke.	C
Consumption of coffee is associated with increased risk of bladder cancer.	C
Consumption of coffee is associated with increased risk of lung cancer.	C
Consumption of coffee is associated with reduced risk of endometrial cancer.	C
Consumption of coffee is associated with reduced risk of hepatocellular cancer	C
Consumption of soft drink is associated with increased risk of dental caries in children.	C
Consumption of soft drinks is associated with increased risk of reduced bone strength.	C

2.6.2.1 Cardiovascular disease, type 2 diabetes and excess weight

Cardiovascular disease: The evidence suggests that consumption of black tea is not associated with risk of cardiovascular disease (Grade C, Section 15.21 in Evidence Report [14]) [427-431]. The evidence suggests that consumption of green and black tea is associated with reduced risk of stroke (Grade C, Section 15.20 in Evidence Report [14]) [429, 432-435].

The evidence suggests that consumption of coffee is not associated with risk of coronary heart disease (Grade C, Section 15.13 in Evidence Report [14]) [436-439], although this was not the case for cigarette smokers.

Type 2 diabetes: There is evidence of a probable association between consumption of more than four cups of coffee a day and a reduced risk of type 2 diabetes (Grade B, Section 15.15 in Evidence Report [14]) [440-447]

Excess weight: It is probable that consumption of sugar-sweetened beverages is associated with weight gain in children and adults (Grade B, Section 15.1 in Evidence Report [14]) [448-459] (see Chapters 2 and 4).

2.6.2.2 Cancer

2.6.2.2.1 Cancer: Coffee

Bladder and lung cancer: The evidence suggests that consumption of coffee is associated with increased risk of bladder cancer (Grade C, Section 15.10 in Evidence Report [14]) [460-462] and lung cancer (Grade C, Section 15.12 in Evidence Report [14]) [463].

Breast, colorectal and ovarian cancer: The evidence suggests that consumption of coffee is not associated with risk of breast cancer [464], colorectal cancer [294, 465-467] or ovarian cancer [468-470] (Grade C, Section 15.7, 15.9, 15.11 in Evidence Report [14]).

Endometrial and hepatocellular cancer: The evidence suggests that consumption of coffee is associated with decreased risk of hepatocellular cancer [471] and endometrial cancer [472, 473] (Grade C, Section 15.6, 15.8 in Evidence Report [14]).

Gastric cancers: The evidence is inconclusive regarding an association between coffee consumption and risk of gastric cancer (Section 15.5 in Evidence Report [14]).

2.6.2.2.2 Cancer: Tea

Ovarian and colorectal cancer: The evidence suggests that consumption of green or black tea is not associated with ovarian cancer (Grade C, Section 15.18 in Evidence Report [14]) [468-470] or colorectal cancer (Grade C, Section 15.19 in Evidence Report [14]) [465-467, 474, 475].

Other cancers: Recent evidence is inconclusive regarding an association between consumption of green and black tea and breast, gastric or lung cancer (Section 15.16, 15.17 and 15.22 in Evidence Report [14]).

2.6.2.3 Other conditions

Dental caries and bone strength: The evidence suggests that consumption of soft drinks is associated with increased risk of dental caries in children (Grade C, Section 15.14 in Evidence Report [14]) and increased risk of reduced bone strength (Grade C, Section 15.3 in Evidence Report [14]) [458, 476-479] (see Chapter 4).

Oral health is also affected by fluids. Fluoride added to tap water provides protection against dental caries [480]. The mildly alkaline nature of saliva is the key protective element against corrosion of teeth by acids. If dehydration occurs after exercise or from particular medications, an adequate intake of water is essential for maximising the protective effect of saliva on oral health [481]. Dehydration also causes loss of salivary protection against attrition, erosion and abrasion.

The Australian Dental Association reports that 68% of Australian school students have at least one tooth eroded [482]. Erosion is related to the acidity of drinks, whether sweetened with sugar or artificial sweeteners, as well as the bacterial fermentation that can occur with consumption of sugar (see Section 4.3). The relationship between the acidity of sweetened drinks and dental erosion is also supported by the American Academy of Pediatrics Committee on School Health [483].

Blood pressure: Recent evidence is inconclusive in regards to an association between coffee consumption and systolic blood pressure (Section 15.14 in Evidence Report [14]).

2.6.3 How *drinking water* may improve health outcomes

For oral health, water has an advantage over many commercially available drinks, including sugar-sweetened or low-kilojoule soft drinks, 'sports' and 'energy' drinks. As well as the potentially adverse direct effects of sugar (see Section 3.3) the carbonation process creates an acidic environment that contributes to the erosion of tooth enamel [481].

The mechanism by which coffee consumption may protect against type 2 diabetes is not clear. It has been reported that insulin resistance is increased in peripheral tissues after exposure to caffeine [484] and conversely, the effect of caffeine on thermoregulation has been suggested as a mechanism by which glucose homeostasis is improved [485]. Other components of coffee, possibly magnesium, may also assist by benefiting insulin sensitivity and reducing risk of type 2 diabetes [486]. Tea contains polyphenols known as catechins which have been reported to reduce hypertension, atherosclerosis and thrombogenesis [487]. Polyphenols also play a part in nitric oxide production from the vascular endothelium, enhancing endothelial health and reducing the risk of cardiovascular diseases and stroke [487].

2.6.4 Practical considerations: *Drink water*

The National Health and Medical Research Council has developed comprehensive guidelines on tap water standards for drinking. Australian tap water is an ideal option because it is inexpensive and meets high palatability and hygiene standards. Most tapwater in Australia is fluoridated, which has been shown to be a safe and effective public health measure. Fluoridation of tapwater provides an additional benefit for development of strong teeth and bones, making it a very good choice to ensure adequate hydration. Tank water and bottled waters are a useful alternative when access to tapwater is limited. Not all bottled waters contain fluoride.

However, access to and availability of clean and safe water may not be available among some population groups, particularly in remote regions of Australia.

2.6.4.1 Pregnant and breastfeeding women

As stated in the previous dietary guidelines, pregnant and breastfeeding women have a slightly increased water requirement because of expanding extracellular fluid space and the needs of the foetus and the amniotic fluid. The fluid need is therefore 750–1000mL a day above basic needs [37].

Many authoritative bodies advise pregnant and breastfeeding women to limit their caffeine intake – for example the New Zealand Ministry of Health advises that intake be restricted to a maximum of 300mg of caffeine per day (about three cups of coffee or six cups of tea) [220].

2.6.4.2 Infants

Water turnover is higher in infants and young children than in adults. Adequate levels of hydration are important, especially during times of potential heat stress (for example, due to hot weather or fever). Breastmilk supplies adequate water up to around 6 months of age, but cooled boiled water may need to be provided for formula fed infant [133].

2.6.4.3 Older people

Older people can experience dehydration due to inadequate intake of water or other drinks. The normal decline in kidney function with age, plus hormonal changes, decreased thirst perception, medication, cognitive changes, limited mobility and increased use of diuretics and laxatives may create concern for older people [35]. These changes may be normal adaptations of the ageing process but the outcomes of dehydration in the elderly are serious and include cognitive impairment, functional decline, falls or stroke.

Where to next

Both the quality and quantity of foods and drinks consumed can significantly impact on health and wellbeing. Overconsumption of some foods and drinks containing some fats, added sugar and salt, and alcohol and relatively poor in nutrients, is associated with increased risk of some health conditions, and are discussed in the following chapter.

3. Limit intake of foods and drinks containing saturated and trans fats, added salt, added sugars and alcohol

Guideline 2

Limit intake of foods and drinks containing saturated and trans fats, added salt, added sugars and alcohol.

a. Limit intake of foods and drinks containing saturated and trans fats

- Include small amounts of foods that contain unsaturated fats.
- Low-fat diets are not suitable for infants.

b. Limit intake of foods and drinks containing added salt

- Read labels to choose lower sodium options among similar foods.
- Do not add salt to foods.

c. Limit intake of foods and drinks containing added sugars

- In particular, limit sugar-sweetened drinks.

d. If you choose to drink alcohol, limit intake

Executive summary

This Guideline emphasises the importance of limiting intake of foods and drinks high in saturated and trans fats, added salt, added sugars and alcohol, due to evidence that these foods are associated with increased risk of obesity and/or chronic disease, including cardiovascular disease, type 2 diabetes and/or some cancers.

The link between dietary saturated fat, cholesterol and cardiovascular disease is well established. Replacing dietary saturated fat or trans fat with monounsaturated and polyunsaturated fats is associated with improved blood lipid profiles and reduced risk of cardiovascular disease.

Fat-rich foods are energy-dense, and it is prudent to choose low-fat and low energy-density foods in a total dietary pattern that seeks to control overall energy intake. Low-fat diets are not suitable for children under two years of age. Reduced-fat milk may be used from the age of two.

Reducing intake of sodium decreases blood pressure in both normotensive and hypertensive adults, while the evidence has strengthened that reducing sodium intake might decrease risk of mortality, stroke and heart disease in people with hypertension. Processed foods are the major source of sodium in western diets.

Frequent consumption of foods and drinks high in added sugars is a major risk factor in dental caries. There is strengthened evidence of association between intake of sugar-sweetened drinks and risk of excess weight gain.

The health, social and economic costs associated with excessive alcohol consumption are well-documented. Limiting alcohol intake is also an important strategy for achieving appropriate energy intake.

This chapter provides information on why consumption of these food types should be limited. It includes practical considerations for meeting the guideline, for example, by choosing a variety of nutritious foods (Guideline 1), using only small amounts of unsaturated spreads and oils, and avoiding or limiting discretionary foods and drinks.

3.1 Limiting intake of foods and drinks containing saturated and trans fat

3.1.1 Setting the scene

Like protein and carbohydrate, fat is a macronutrient which contributes to dietary energy intake. 'Fat' is a broad term which can apply to foods such as butter, margarine and oils, and to fatty acids which are food components. Fats add palatability and texture to the diet, can be a vehicle for essential nutrients, and have high energy value (fat delivers about 37 kJ/g, compared to around 17 kJ/g for protein and carbohydrate). The proportion of fat in a diet influences its energy density, which is important for weight management (see Chapter 4). Different fats have different health effects, so both the amounts and types of fat need to be carefully considered in a diet.

Most fats in foods are in the form of triglycerides, which are made up of a unit of glycerol combined with three fatty acids which may be the same or different. The differences between one triglyceride and another are largely due to the fatty acids attached to the glycerol unit. Other dietary fats include phospholipids, phytosterols and cholesterol [9].

Fatty acids can be saturated (SFA), monounsaturated (MUFA) or polyunsaturated (PUFA). The type of fatty acid depends on the chemical bonding within the fatty acid molecule, specifically the number of double bonds between the carbon atoms. This gives them different chemical properties that cause different biological effects. MUFA and PUFA with one or more double bonds in the *trans* configuration are known as trans-fatty acids (TFA) [488]. Omega fatty acids (omega-3 and omega-6 PUFAs) are sub-classes of PUFA. The most common omega-9 is the MUFA called oleic acid. Staple foods with a relatively higher fat content, such as nuts, seeds, some grains (for example, oats), dairy foods and meats have various combinations of fatty acids. Fish is the predominant source of omega-3 fatty acids, eicosapentaenoic (EPA) (20:5) and docosahexaenoic (DHA) (22:6). Grass-fed meat, kangaroo and offal also contain these fatty acids as well as the omega-3 fat docosapentaenoic acid (DPA) (22:5). EPA, DHA and DPA (and some other minor fatty acids) are referred to as long chain polyunsaturated fatty acids, omega-3 LCPUFA.

The *Nutrient Reference Values for Australia and New Zealand* recommend total fat should account for no more than 20–35% of energy (kilojoule) intake. Total SFA and trans-fats should comprise no more than 10% of energy intake [9]. As humans do not make essential fatty acids, 4–10% of energy should come from linoleic acid(omega-6 PUFA) and 0.4–1% from α-linolenic acid (omega-3 PUFA) [9]. Given that total energy intake reflects the sum of the energy value of all foods consumed, these fatty acid targets can only be met through careful dietary selection, so food choices and culinary practices can have a substantial impact on the ability to meet these targets [10].

3.1.2 The evidence for *'limiting intake of foods and drinks containing saturated and trans fat'*

The evidence for associations between dietary fat and the development of type 2 diabetes, hypertension, cancer and poor mental health was reviewed. The link between dietary saturated fat, cholesterol levels, atherosclerosis and other components of cardiovascular disease has been well established in previous guidelines, and was not reviewed here.

The *Guidelines* recommend caution in choosing foods high in fat because of the implications for weight gain and cardiovascular disease risk. Fat-rich foods are energy (kilojoule) dense, heightening the risk of excess energy intake [489]. The evidence base for the effects of dietary fat has grown substantially and increased in specificity with respect to different fatty acids.

Scientific evidence on the effect of dietary fat on health can come from studies that address dietary variables in a number of ways. These include whole-of-diet studies examining the proportion of fat in the diet (relative to protein and carbohydrate), the type of fat in the diet (relative to other types of fat), the effects of specific fatty acids in the diet, and the effects of individual foods in which fat is a significant component. For example, studies could examine the effects of:

- a low-fat diet
- a diet with a modified dietary fat ratio, for example, a high polyunsaturated:saturated fat ratio (P:S or PUFA:SFA)
- a diet enriched with specific fatty acids (for example, omega-3 fatty acids)
- oils and fats (for example, olive oil, spreads) in a defined dietary pattern.

Examining the evidence for the effects of fats and oils on health found a full range of these types of studies.

Methodological issues arise when considering the effects of fats and of dietary fat in the total diet. The difficulty in designing studies that address the question of the effect of dietary fat on disease risk is reflected in several recent reviews on the topic. It is important to note that inconsistency in results affects the strength of the evidence statements below.

There is ample evidence of the relationships between dietary patterns and disease risk at the population level [490]. Fat content is an important component of diet quality and it may be that the evidence for limiting fat in the diet is best considered from the food and whole-of-diet perspective with additional reference to overall nutritional quality.

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) related to 'limit fat' from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual

studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines.*

Evidence Statement	Grade
Consumption of higher LCPUFA fat (intakes amount not specified) is associated with a reduced risk of dementia	C

3.1.2.1. Cardiovascular disease, type 2 diabetes and excess weight

Cardiovascular disease: Of the systematic reviews addressing the risk of cardiovascular disease and consumption of fats and oils [410, 491-499], only one review [410] focused on the proportions of dietary fatty acids in the overall diet rather than the effects of omega-3 fatty acids on markers of cardiovascular health. This review confirmed that replacing SFA with unsaturated fatty acids may reduce the risk of coronary heart disease, and that replacing trans fats with unsaturated fats improves blood cholesterol levels. From a whole-of-diet perspective, this review found that reducing the risk of cardiovascular disease by replacing SFA with carbohydrate (as is the case in some low-fat diets) depends on effects on body weight [410].

The FAO Expert consultation on fats and fatty acids in human nutrition 2010 [500] reports convincing evidence that replacing SFA with PUFA decreases the risk of coronary heart disease. The *Dietary Guidelines for Americans, 2010* [143] have come to similar conclusions. They acknowledge strong evidence that dietary SFA is positively associated with increased serum cholesterol and LDL cholesterol, with increased risk of cardiovascular disease, while replacing dietary SFA or trans fats with PUFA is associated with improved blood lipid profile and reductions in levels of numerous markers of inflammation. The review of the *Dietary Guidelines for Americans, 2010* also noted strong evidence that replacing dietary SFA with MUFA and/or PUFA is associated with improved blood lipids related to cardiovascular disease, and moderate evidence that 250mg of LCPUFA delivered with 2 servings of seafood per week is associated with reduced cardiac mortality from coronary heart disease and reduced risk of sudden death from cardiovascular disease [143].

Hypertension: The evidence suggests that consumption of fat, irrespective of amount or type, is not associated with hypertension in the short term (Grade C, Section 12.4 in Evidence Report [14]) [501-509].

Type 2 diabetes: While overweight and obesity increase the risk of type 2 diabetes [88], recent short-term interventions are inconclusive and may not adequately reflect the nature of the effect of dietary fat on type 2 diabetes. The evidence suggests that consumption of LCPUFA (from 0.4 to 6g/day) and diets of varying fat content are not associated with fasting plasma glucose or insulin concentrations (Grade C, Section 12.3 in Evidence Report [14]) [492, 499, 509-519].

In the longer term, the review conducted for the *Dietary Guidelines for Americans 2010* found strong evidence that dietary SFA was positively associated with increased markers of insulin resistance and increased risk of type 2 diabetes. In addition, it found decreasing dietary SFA and replacing it with PUFA or MUFA decreases the risk of type 2 diabetes in healthy adults and improves insulin responsiveness in insulin resistant and type 2 diabetes subjects. PUFA intake was associated with a significant decrease in the risk of type 2 diabetes [143].

Excess weight: Dietary fat provides a substantial amount of energy (kilojoules) per gram but total dietary energy is the variable that affects weight. Reducing the amount of dietary fat will not necessarily reduce dietary energy, but it is prudent to choose low-fat and low energy-density foods in a total dietary pattern that seeks to control overall energy intake. Because of this total energy effect, there are difficulties in appraising research on the effect of dietary fat alone on weight gain [489] (see Chapter 4).

3.1.2.2. Cancer

The evidence suggests there is no association between consumption of LCPUFA with total all-cause cancer incidence or mortality (Grade C, Section 12.5 in Evidence Report [14]) [495, 520].

Other cancers: Evidence of an association between total fat consumption across a range of intakes and breast or endometrial cancer is inconclusive (Section 12.6 & 12.7, Evidence Report [14])

3.1.2.3. Other conditions

Dementia: The evidence suggests that consumption of higher LCPUFA is associated with a reduced risk of dementia (Grade C, Section 12.8 in Evidence Report [14]) [317, 329, 521-524].

3.1.3 How *limiting intake of foods and drinks containing saturated and trans fat* may improve health outcomes

Fat is an energy-dense macronutrient, so consumption of excess fat may lead to excess energy (kilojoule) intake and weight gain [489], but an individual's genetic makeup, physical activity and other dietary factors also play a part [525]. Fat cells secrete compounds that influence appetite, inflammation and possibly also cancer development [526-528]. Insulin resistance, reflected in high insulin and glucose levels, is linked to obesity, and leads to type 2 diabetes. Other cardiovascular disease risk factors such as high cholesterol levels and hypertension tend to co-exist with insulin resistance, a phenomenon often referred to as the metabolic syndrome [529].

Fatty acids do not only contribute to body fat. Different fatty acids influence disease risk factors. Dietary SFA and dietary TFA have been associated with raised plasma LDL-cholesterol, and

dietary TFA has been associated with a reduced plasma HDL-cholesterol [88]. These changes in plasma cholesterol fractions are established risk factors for coronary heart disease that can be influenced by diet. Raised LDL-cholesterol 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 [530]. Several large cohort studies have also demonstrated that reduced HDL-cholesterol is a significant risk factor for coronary heart disease [531, 532].

PUFA intake appears to reduce coronary heart disease risk [490], but the balance between omega-6 and omega-3 PUFA may be important [533]. In addition to effects on lipid metabolism, PUFA may exert a positive influence on insulin action, appetite regulation, inflammatory responses, and muscle function [534]. Among the LCPUFA fatty acids, EPA is the precursor of the 3 series of prostaglandins and the 5 series of leukotrienes [535]. This suggests anti-inflammatory and anti-coagulant effects which may explain the protective influences on cardiovascular disease. DHA is found in high concentrations in the photoreceptors of the retina and the membranes of the brain, with implications for cognitive development and mental health [536].

3.1.4 Practical considerations: *Limiting intake of foods and drinks containing saturated and trans fat*

The most recent dietary data available in Australia show adults consume 73–101g/day of fat, with higher intakes among men and younger age groups [44]. Children consume 51–87g/day of fat, with intake being higher in older age groups [13]. The total fat intake of the population has not decreased in recent years, but now constitutes a lower proportion of overall energy intake due to a relative increase in the consumption of carbohydrates, especially refined carbohydrates [48]. Intakes of total fat, in particular saturated fat, are higher than recommended [114]. However, the mean total population trans-fat intake for Australia is estimated to be 0.5-0.6% of total dietary energy, which is well below the WHO population goal of less than 1% of total dietary energy from TFA [234].

Dietary fat included in the *Australian Guide to Healthy Eating's Foundation Diets* mainly comes from fish, lean meat, poultry and milk, yoghurt and cheese products. Where more energy is required in moving from *Foundation* to *Total Diets*, additional serves of these and/or other foods containing fats can be included as additional nuts and seeds, unsaturated spreads and oils, and/or discretionary foods.

Limiting the amount and type of fat in the diet can be achieved by choosing vegetables, fruit, lean meats and low-fat milk, yoghurt and cheese products, and using only small amounts of unsaturated spreads and oils. Limiting intake of fried foods (including chips) and baked cereal products (including snack foods, biscuits and cakes) helps to reduce 'invisible fat' in the diet. LCPUFA intake can be increased by eating according to the *Foundation Diets* and *Total Diets*, which includes fish around twice a week. When eating meals outside the home, lower-fat menu choices are preferable.

The P:M:S ratio is a useful tool in assessing the type and amount of fat consumed and its relative impact on chronic disease risk. The foods included in the *Australian Guide to Healthy Eating* generally contain low levels of saturated fat and include PUFA and MUFA in proportions that appear to be protective against heart disease risk and support the maintenance of cognitive function [9].

For quantitative population recommendations see the *Nutrient Reference Values for Australia and New Zealand* [9].

3.1.4.1 Infants

For infants under the age of around six months, breastmilk provides an ideal amount and type of fat. The *Guidelines* on fat intake for the adult population are not applicable to young children, particularly those under two years. The amount and type of fats required for infants are related to physiological and health outcomes [9]. Even a small energy deficit during this period of rapid development may affect growth [9]. Neurological development is particularly rapid in the first two years of life and restriction of the fat intake during that time may interfere with optimal energy intake and reduce the supply of essential fatty acids, particularly long chain omega-3 PUFA needed by developing nervous tissue, adversely affecting growth and development.

3.1.4.2 Children and adolescents

Reduced fat milk may be used from the age of two years, when milk plays a less dominant role in the diet (see Chapter 2). A high-fat diet is likely to be energy-dense, contributing to excess energy intake and the development of obesity (see Chapter 4). Even at a young age, a diet high in saturated fats may predispose children and adolescents to the development of cardiovascular disease later in life and the evidence supports this advice on fat intake for children from two years of age [537]. Introducing healthy eating patterns in early childhood influences dietary patterns in later childhood [538, 539].

3.1.4.3 Older people

Low-fat diets are not suitable for convalescent older people and frail aged people (to whom these guidelines do not apply) because of the possible adverse effects of energy restriction in these groups. However, for those aged 65–75 who are well, the type and amount of fat in the diet deserves consideration. Although the increased relative risk of raised plasma cholesterol for coronary heart disease tends to be lower in older people than in younger adults, lowering of lipid levels can reduce risk of ischemic heart disease regardless of age [540].

3.1.4.4 Aboriginal and Torres Strait Islanders

Limiting intake of excess energy from any source, including total fat, is particularly important given the higher prevalence of obesity in Aboriginal and Torres Strait Islander groups compared to non-Indigenous Australians [30, 32]. Limiting saturated and trans fat are important given the high prevalence of coronary heart disease, and decreased saturated fat intake may also improve insulin sensitivity.

3.1.4.5 Culturally and linguistically diverse groups

The profile of dietary fat will vary depending on traditional culinary use. Food product labels may assist people in learning about the amounts and types of fats in unfamiliar or newly introduced manufactured foods.

3.2 Limit intake of foods and drinks containing added salt

3.2.1 Setting the scene

Dietary guidelines have recognised the role of sodium in elevating blood pressure since the draft United States Surgeon General's report released in 1979 [541]. Initial advice to the public was framed to reduce consumption of discretionary salt, such as salt added at the table or during cooking. More recently it has been recognised that processed foods are the major source of sodium in Western diets, so advice needs to include processed food. Sodium occurs naturally in food, and a range of sodium-containing additives are also added to manufactured and processed foods. In these *Guidelines*, salt always refers to sodium chloride and is never used as a synonym for the total amount of sodium in foods.

Since the previous edition of the Dietary Guidelines in 2003, the evidence for a relationship between reducing sodium intake and reducing blood pressure has strengthened, particularly in those classified as normotensive. In addition there is now some evidence indicating that reducing sodium may result in a reduction in outcomes such as mortality, stroke and heart disease for those with hypertension, but not, as yet, for those with normal blood pressure.

3.2.2 The evidence for *'limiting intake of foods and drinks containing added salt'*

The evidence for the *Guidelines* focuses on dietary sodium intake, rather than salt intake, because most studies forming the body of evidence statements measured total sodium intake or urinary sodium excretion (which is a marker of sodium intake from all sources including salt, other additives containing sodium, and naturally occurring sodium).

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) related to 'limit added salt' from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines*.

Evidence Statement	Grade
Decreasing consumption of sodium decreases blood pressure (BP) in normotensive adults; a reduction of 1,800mg reduces systolic BP by about 2mmHg and diastolic BP by about 1mmHg	A
Decreasing consumption of sodium decrease blood pressure (BP) in hypertensive adults; a reduction of 1800mg reduces systolic BP by about 5mmHg and diastolic by about 3mmHg	A
Consuming a diet low in sodium reduces blood pressure in children up to 18 years of age	B
Reducing sodium intake by about 1000mg/day is associated with reduced risk of cardiovascular events	C

3.2.2.1 Cardiovascular disease, type 2 diabetes and excess weight

Blood pressure: The evidence is convincing that decreasing consumption of sodium decreases blood pressure in normotensive adults; a reduction of 1,800mg per day reduces systolic BP by about 2 mmHg and diastolic blood pressure by about 1mmHg (Grade A, Section 13.1 in Evidence Report [14]). The evidence is also convincing that decreasing consumption of sodium decreases blood pressure in hypertensive adults - a reduction of 1,800mg per day reduces systolic BP by about 5mmHg and diastolic blood pressure by about 3mmHg (Grade A, Section 13.1 in Evidence Report [14]) [542-557]. Reducing sodium intake reduces both systolic and diastolic blood

pressure, and the effect is greater in those with hypertension than in those with normal blood pressure [551-554].

There is evidence of a probable association between a diet low in sodium and a reduction in blood pressure in children up to 18 years of age (Grade B, Section 13.2 in Evidence Report [14]) [553, 557, 558].

The *Dietary Guidelines for Americans, 2010* draw similar conclusions regarding the relationship between sodium and blood pressure. They state 'a strong body of evidence has documented that in adults, as sodium intake decreases, so does blood pressure. A moderate body of evidence has documented that as sodium intake decreases, so does blood pressure in children, from birth to 18 years of age' [143]. They also note that people with hypertension, diabetes and chronic kidney disease are more sensitive to sodium than healthier younger people, and that sensitivity to sodium increases with age.

Cardiovascular disease: Despite extensive research on the relationship between sodium and blood pressure, few long term studies have examined changes in sodium intake and changes in cardiovascular morbidity and mortality. Past studies have mainly included subjects with hypertension. The evidence suggests that reducing sodium intake by about 1,000mg/day is associated with reduced risk of cardiovascular events (Grade C, Section 13.4 in Evidence Report [14]) [552, 559-564]. The results are consistent with results from drug trials that have shown that reductions in high blood pressure also lead to reductions in adverse cardiovascular outcomes [565, 566].

3.2.2.2 Cancer

Evidence of a probable association between consumption of salt and salt-preserved foods with gastric cancer was found in the systematic review prepared as the background paper for the Joint WHO/FAO Expert Consultation on diet, nutrition and prevention of chronic diseases [567]. This association was described as convincing by the WCRF [42]. The WCRF also reported convincing evidence of an association between processed meats (meat preserved by smoking, curing, salting or addition of chemical preservatives such as nitrites) and increased risk of colorectal cancer [42] but it is unclear whether the responsible factor in the food is the salt, other components such as nitrites, or a combination of these factors.

3.2.2.3 Other conditions

Bone health: Evidence suggesting an association between a low sodium diet and markers of bone health in postmenopausal women is inconclusive (Section 13.3, Evidence Report [14]).

3.2.3 How *limiting intake of foods and drinks containing added salt* may improve health outcomes

Sodium and other electrolytes are needed to maintain extracellular volume and serum osmolality. There are various systems and hormones that influence sodium balance including the renin-angiotensin-aldosterone hormone system, the sympathetic nervous system, atrial natriuretic peptide, the kallikrein-kinin system, various intrarenal mechanisms, and other factors that regulate renal and medullary blood flow [9].

Future data may lead to some refinement in understanding the underlying mechanisms. For example, the ratio of sodium to potassium may influence blood pressure more strongly than the amount of sodium alone [560-562] and reducing sodium intakes may be particularly important for overweight people with certain conditions [563]. The *Dietary Guidelines for Americans, 2010* comment that the effects of higher sodium intake can be countered if potassium intake is also higher and note that potassium intakes in the US are lower than desirable [143].

3.2.4 Practical considerations: *Limiting intake of foods and drinks containing added salt*

There has been a shift in the language on this issue. The first edition of the Dietary Guidelines included the guideline 'Eat less salt' [568]. The second edition revised this to 'Choose low salt foods and use salt sparingly' [569]. The third edition advised Australians to 'Choose foods low in salt' [37]. In these *Guidelines*, it is recognised that any reduction in dietary sodium would reduce blood pressure in both hypertensive and normotensive people.

The *Nutrient Reference Values for Australia and New Zealand* recommend an intake of sodium in the range 460–920mg/day for adults to cover the essential requirement. Current advice in Australia is for dietary sodium intake to be preferably less than 1,600mg/day for adults or no higher than the set Upper Level (UL) of 2,300mg/day (approximately equal to 6g of table salt) ([9]). Among those with high sodium intakes, reduction in sodium intake would reduce blood pressure even if specific targets are not achieved.

Diets that are consistent with the *Guidelines* will help to limit sodium intake. Fresh, unprocessed or minimally processed foods such as fresh vegetables, including legumes/beans, and fruit, frozen or tinned vegetables, including legumes/beans, and fruit with no added salt, meats, fish and milk are generally lower in sodium. Some breads and cereal products and cheese are high in salt and make a significant contribution to sodium intake, although there is considerable variation between products and brands. However, these foods provide other important health benefits outlined in the relevant sections of the *Guidelines*. The public should not avoid these foods, but should be encouraged to check food labels and select lower-sodium products in these categories. Some foods are labelled 'low sodium/salt' because they contain less than 120mg of sodium per 100g. Not

all foods which meet this criterion carry a 'low sodium' claim, and some higher sodium foods such as bread and cheese also contain important nutrients.

Because salt is 40% sodium, avoiding discretionary salt in cooking or at the dinner table will also reduce sodium intake. In the *1995 National Nutrition Survey*, 62% of the population aged two years and older reported always or usually adding salt at the table or during cooking [570]. Since 1995, Asian-style cooking is increasingly popular in Australia and many Asian-style sauces such as soy, oyster and fish sauces are high in sodium. Lower sodium options of these foods should be chosen, recognising that they may still have high sodium content. Salt substitutes or 'lite' salts which replace some sodium chloride with potassium chloride can provide a salt taste with lower levels of sodium. Herbs, spices, garlic, lemon juice and vinegar can also be used to season foods without adding salt.

Iodine deficiency previously existed in Australia and has re-emerged in recent years. Iodised salt is one way to increase iodine intake. Since 2009, it has been mandatory for any salt used in commercial bread baking to be iodised (organic bread is exempt from this requirement) [570]. Consequently it would unnecessary for most individuals to use discretionary iodised salt for the purpose of obtaining iodine. Milk and dairy foods are an important source of iodine in Australia.

3.2.4.1 Pregnant and breastfeeding women

Advice to limit the intake of sodium for the general population is also applicable to pregnant and breastfeeding women [571].

3.2.4.2 Infants

The Australia New Zealand Food Standard Code sets maximum limits for the sodium content of commercially prepared infant foods such as rusks, biscuits and other ready-to-eat foods, and prohibits the addition of salt to fruit-based foods, fruit drinks and vegetable juices [234]. These restrictions are needed because infants have a lower renal capacity than older children and adults.

Parents and carers who are introducing infants to solid foods should be advised to minimise sodium intake. This means preparing home-made infant foods without salt or ingredients that are high in salt or sodium, and minimising infants' intake of other processed foods that are high in sodium.

3.2.4.3 Children

For children with average energy needs, the dietary patterns in the Modelling System [10] contained up to 50% less sodium than the average sodium intakes reported in the *2007 Australian National Children's Nutrition and Physical Activity Survey* [13].

3.2.4.4 Older people

Taste perception decreases with age and can be a factor in decreased food intake and malnutrition. For a chronically ill older person who has high blood pressure, clinicians need to weigh up the benefit of the addition of salt to food to improve flavour (with improved intake and quality of life, and reduced risk of malnutrition) against the risks of hypertension and its management. For chronically ill older people who do not have high blood pressure, there is no reason to reduce salt as there are no additional health benefits to be gained and maintaining food intake is a priority.

3.3 Limit intake of foods and drinks containing added sugars

3.3.1 Setting the scene

Sugars are carbohydrates, for example fructose, glucose, lactose or sucrose. When sugars occur naturally in foods such as fruit, vegetables and dairy products they are referred to as intrinsic sugars. However, the major source of sugar in the Australian diet is sucrose from sugar cane which is added to foods and is termed an extrinsic sugar. Sucrose is widely used in processed foods and drinks as a sweetener and also plays a role as a flavour enhancer and preservative.

Sugars provide a readily absorbed source of energy, but added sugars can increase the energy content of the diet while diluting its nutrient density. Sugars are a major factor in dental caries and diets high in added sugars are also associated with some adverse health outcomes. Sugar-sweetened drinks (fruit juice drinks, soft drinks, flavoured mineral waters and sports drinks) are the largest source of sugars in the Australian diet, with consumption highest in adolescents and children [44]. New evidence emphasises the relevance of sugar-sweetened drinks to the development of excess weight (see below).

Dental caries remains a significant public health problem in Australia. In 2007–8, $6.1 billion was spent on dental services in Australia, representing 6.2% of all health expenditure [572]. In severe cases, dental caries can cause loss of teeth and pain that may reduce dietary intake and compromise nutritional status.

There are particular concerns about the recent increase in consumption of added sugars from sweetened drinks (see Chapter 4).

3.3.2 The evidence for *'limiting intake of foods and drinks containing added sugars'*

All previous Australian dietary guidelines have recommended restricting added sugars. There has been little change in the evidence linking added sugars with dental caries, but the targeted literature review found strengthened evidence for a relationship between sugar-sweetened drinks and excess weight gain.

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines*.

Evidence Statement	Grade
Consumption of sugar-sweetened beverages is associated with increased risk of weight gain in adults and children	B
High or frequent consumption of added sugars, particularly for infants and young children, is associated with increased risk of dental caries	C
Consumption of soft drink is associated with increased risk of dental caries in children	C
Consumption of soft drinks is associated with increased risk of reduced bone strength.	C

3.3.2.1 Cardiovascular disease, type 2 diabetes and excess weight

Excess weight: Recent studies show evidence of a probable association between sugar-sweetened drinks and weight gain in adults and children (Grade B, Section 15.1 in Evidence Report [14]) [448-459]. A more recent longitudinal study also strengthens the evidence associating sugar-sweetened drinks with weight gain [573].

There is insufficient consistent evidence available to form an evidence statement about fruit juices and weight gain. Some studies found no association in children [392-394, 456] while two studies in children [449, 574] and one in adults [575] did find an association. Children drinking less fruit juice [192] and those who consume more fruit [457] had lower BMI Z-scores, and children at risk of becoming overweight had a higher risk of gaining fat if they consumed fruit juice [449].

No large studies have measured the long-term development of overweight and obesity specifically related to consumption of sugars, although one retrospective cohort study reported that adults who consumed less sugar-sweetened foods had less increase in skinfold fat and waist circumference over a five-year period (Section 14.3, Evidence Report [14]; [192].

Type 2 diabetes: Insufficient studies were identified to develop an evidence statement for intake of sugars and type 2 diabetes [576, 577]. However, more recent studies indicate that sugar-sweetened drinks may increase the risk of developing type 2 diabetes [578]. A recent meta-analysis also supports an increased risk for type 2 diabetes and the metabolic syndrome from consumption of sugar-sweetened drinks [579].

Cardiovascular disease: There is no new evidence that sugars play a causal role in the development or moderation of the risk factors for cardiovascular disease. Early studies suggested that a reduction in dietary sucrose could lower elevated triglyceride levels, but it is likely that the effects seen were the result of a reduction in energy intake and body weight [580].

3.3.2.2 Cancer

There is evidence suggesting that consumption of sucrose is not associated with the risk of cancer (Grade C, Section 14.1 in Evidence Report [14]) [581-585]. The World Cancer Research Fund found no convincing or probable evidence of increased risk of all cancers with the intake of sugars, but some limited evidence of an association between a high intake of sugars and increased risk of colorectal cancer [586]. The most recent WCRF statements urge caution with energy-dense foods and sugar-sweetened drinks because of their association with obesity and its link with some cancers [42].

3.3.2.3 Other conditions

Dental caries: The relationship between sucrose and dental caries was first documented more than a century ago [587] and has been confirmed in numerous studies since [588]. Historically, the prevalence of dental caries has increased when dietary patterns have changed to include more added sugars and foods containing refined starches. New evidence supports past findings and suggests that high or frequent consumption of added sugars, particularly for infants and young children, is associated with increased risk of dental caries (Grade C, Section 14.2 in Evidence Report [14]) [589-593]. The evidence also suggests that dental caries are related to sugar-sweetened drinks (Grade C, Section 15.4 in Evidence Report [14]) [589, 591].

Caries are associated with national per capita yearly sucrose consumption, with very little caries in children at 10 kg/year (about 30 g/day) or less. A steep increase may occur from 15 kg/year upwards [88].

The sugars contained in the cellular structure of foods, such as the intrinsic sugars of fresh fruit and vegetables, have been found to have little cariogenic potential, whereas foods high in extrinsic sugars are most damaging to teeth [594].

Bone strength: Evidence suggests an association between consumption of soft drinks and increased risk of reduced bone strength (Grade C, Section 15.3 in Evidence Report [14]) [476-479]. Cola drinks (sugar-sweetened and diet varieties), but not other carbonated drinks, are associated with significantly lower bone mineral density in women, but not in men [479]. In young men, significant adverse changes in indices of bone remodelling and bone resorption markers occurred when cola drinks were added to a low-calcium diet compared with adding milk [476]. A systematic review reported an inverse relationship between soft drink consumption and milk intake [458].

It appears that soft drink consumption is associated with some problems related to bone health, but, with the exception of some limited evidence related to cola drinks, it remains unclear whether soft drinks exert a direct effect or reflect their inverse relationship with milk consumption.

ADHD: There is no evidence that added sugars are involved in the aetiology of attention deficit/hyperactivity disorder (ADHD) [595].

3.3.3 How *limiting intake of foods and drinks containing added sugars* may improve health outcomes

Dental caries: Important factors for development of caries include the bacterium *Streptococcus mutans*, dietary sugars and a susceptible tooth surface. Fermentable carbohydrates (both sugars and starches) are a substrate for bacteria such as *S. mutans* and *S. sobrinus*, which increase the acid-producing potential of dental plaque [596]. Dietary sugars other than sucrose - for example, glucose and lactose - can also induce caries, although these sugars are less cariogenic than sucrose because, in addition to being converted to acid metabolites, sucrose is uniquely used for extracellular polysaccharide synthesis.

Oral hygiene, dental care, fluoridated water supplies, the type of food and salivary function are also important. The more frequently foods containing added sugars are consumed, the greater the risk of caries since frequent consumption does not allow remineralisation of the teeth [597]. The duration of exposure depends on how long sugary foods stay in the mouth and the number of eating occasions. On the basis of the scientific evidence, advice on sugar intake for the prevention of dental caries should include frequency of intake as well as the amount. The acidity of sweetened drinks is also relevant to dental erosion, a major factor in dental decay [483]. This applies equally to sugar-sweetened or diet soft drinks, since their acidity is comparable.

Excess weight: Many foods containing added sugars, including soft drinks, confectionery, cakes and biscuits, are energy-dense but nutrient-poor. The association between sugar-sweetened drinks and weight gain appears to be related to the reduced effect on satiety with sugars in a liquid medium. Past studies found that compensation for energy from sugar-sweetened drinks is less complete than that for energy in solid form [598]. Newer evidence backs this failure to compensate by reducing energy intake from other foods or drinks [578]. Sugar-sweetened drinks therefore add to total energy intake [599].

Nutrient density may also be compromised by a high intake of added sugars. At any given level of energy intake, as the proportion of added sugars in the diet increases, the nutrient density will fall [600]. This was quantified in a recent analysis of NHANES data (2003–2006) which noted that intake of essential nutrients was less with each 5% increase in added sugars above 5–10% of energy [601].

3.3.4 Practical considerations: *Limiting intake of foods and drinks containing added sugars*

In light of the current prevalence of overweight and obesity (see Chapter 4), the dietary guidelines of many countries recommend significant reductions in foods that contribute to energy while providing few, if any, nutrients [143]. Many foods and drinks containing some fats, added sugars and some starches, and alcohol fit into this category. Sugars provide approximately a quarter of children's energy intake, with 4.6–7.6% of energy coming from sugar-sweetened drinks other than milk [13].

There is insufficient evidence to recommend an exact intake of added sugars suitable for the whole population. From a nutritional perspective, good health can be achieved without the addition of sugars in any form to the diet. For those who are not overweight and are already consuming an adequate diet (a minority of the population), added sugars relate mainly to the problem of dental caries. For the majority of the population, however, overweight and obesity are major problems and require a reduction in energy intake. Limiting added sugars, particularly from sugar-sweetened drinks, is one strategy for adults and children. The World Health Organization recommends that no more than 10% of energy should come from added sugars [88]. Recent data from the US suggests a level of 5–10% of energy from added sugars may be appropriate [601]. This is much less than current Australian consumption, reinforcing the continued need for this Guideline.

3.3.4.1 Infants

Baby-bottle caries is a recognised problem in infants who are pacified by sucking on a bottle for long periods. Babies who fall asleep while continuing to feed from a bottle containing infant formula, fruit juice or other sugar-containing liquid can develop a severe form of tooth decay [602]. Infants do not need added sugars and the Australia New Zealand Food Standards Code

stipulates that ready-prepared infant foods with more than 4g of added sugars per 100g must be labelled as 'sweetened' [234].

3.3.4.2 Children

Milk and water are the recommended drinks for children. Children and adolescents should limit intake of sugar-sweetened drinks. Common sugar-sweetened drinks include soft drink, 'sports drinks', 'vitamin waters', cordials and energy drinks. Energy drinks may also be high in caffeine (see Chapter 2) and are not suitable for children. Sweetened flavoured milk provides nutrients but can be high in energy-density; plain milk is preferable.

3.3.4.3 Older people

Including a moderate amount of added sugars as a flavour enhancer can increase variety and palatability for older people and will not compromise nutrient intake if added to nutritious foods. Sugars are also a readily-absorbed source of energy for the frail aged.

3.3.4.4 Aboriginal and Torres Strait Islanders

In remote Aboriginal communities, apparent consumption of sugars is much higher than the Australian average while consumption of fruit and vegetables is well below the Australian average [134]. In remote communities where apparent consumption was measured, refined sugars contributed approximately 30% of total energy intake, with 60% of the sugars in the form of white sugar added to foods and drinks. No data are available for urban Indigenous communities. Historically, Aboriginal Australians had substantially fewer dental caries than non-Indigenous people, but this trend has been reversed with the oral health of non-Indigenous children improving and that of Aboriginal children deteriorating [603]. Aboriginal children and other Australian children from the lowest socioeconomic groups have not had the improvement in dental health seen in other children.

3.4 Alcoholic drinks

3.4.1 Setting the scene

For many people, an alcoholic drink is a regular and enjoyable part of meals. In terms of nutrition, alcohol is uniquely the only substance that is both a food providing energy and a drug affecting brain function. For these reasons advice on alcohol is included in these *Guidelines*.

Drinking alcohol has health, social and economic costs and benefits for both individuals and populations. It was previously believed that people who drink small quantities of alcohol may have

better health outcomes than those who do not drink, but this finding is being increasingly challenged [604]. Most studies have found that abstainers have better health outcomes than heavy drinkers.

In the Australian population, alcohol is responsible for 3.3% of the total disease burden while it prevents 1% of the total disease burden. This equates to a net effect of 2.3%, equivalent to 61,091 DALYS (Disability Adjusted Life Years) and 0.8% (1,084) of all deaths [11]. In Indigenous Australians, alcohol is responsible for a net 5.4% of the total disease burden and 6.7% of all deaths [605]. Alcohol is second only to tobacco as a preventable cause of drug-related death and hospitalization [606].

The total social costs of alcohol were $15.3 billion in 2004/05, the majority (71%) being for tangible costs such as reduction of the workforce, absenteeism, health care, law enforcement, alcohol education campaigns and research [607, 608].

This chapter utilises the recent NHRMC publication *Australian Guidelines to Reduce Health Risks from Drinking Alcohol* [609] with additional evidence sourced from the Evidence Report to inform the revision of the *Guidelines* [14].

The alcohol guidelines are as follows.

1. For healthy men and women, drinking no more than two standard drinks on any one day reduces the lifetime risk of harm from alcohol-related disease or injury.

2. For healthy men and women, drinking no more than four standard drinks on a single occasion reduces the risk of alcohol-related injury arising from that occasion.

3. a. Parents and carers should be advised that children under 15 years of age are at the greatest risk of harm from drinking and that for this age group, not drinking alcohol is especially important.

   b. For young people aged 15-17 years, the safest option is to delay the initiation of drinking for as long as possible.

4. a. For women who are pregnant or planning a pregnancy, not drinking is the safest option.

   b. For women who are breastfeeding, not drinking is the safest option.

Most recommendations on alcohol consumption are made on the basis of 'standard' drinks consumed. A standard drink in Australia contains 10g of alcohol (equivalent to 12.5mL of alcohol) [609]. The alcohol concentration of drinks is printed on the label in terms of percentage by

volume. However in social situations serving sizes are greater than standard drinks – for example a typical glass of wine in Australia is 170mL [610] which contains close to two 'standard' drinks.

For some groups, the contribution of alcohol to energy intake is significant. Median percentages of contribution of alcohol to energy intake for age and sex groups and the energy content of common alcoholic drinks is included at Appendix 6. For example, if a man with average energy intake consumed four standard drinks of beer, this would account for 13–15% of his energy intake. The proportion of energy obtained from alcohol for those who consume it peaks at age 19–24 for women and 25–44 for men, and declines thereafter.

If the consumption of other foods or drinks is reduced to adjust for the extra energy intake from alcohol, over time this could lead to a deficiency of key nutrients. In view of the increasing prevalence of overweight and obesity, limiting alcohol intake is an important strategy for achieving energy balance.

3.4.2 The evidence for *'limiting alcohol'*

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines*.

Evidence Statement	Grade
Consumption of alcohol regularly at an intake of 1 standard drink per day for women and 1.5-2 per day for men is associated with a reduced risk of CVD morbidity and mortality.	B
Consumption of alcohol regularly at an intake of 1 standard drink per day for women and 1.5-2 per day for men increases HDL cholesterol.	B
Consumption of alcohol, even at low levels (10-15 g/d), is associated with increased risk of breast cancer.	B
Consumption of alcohol is associated with increased risk of cancer of the oesophagus.	B
Consumption of alcohol, even at low levels (10g/day) of consumption, is associated with an increased risk of colon cancer and rectal cancer.	C

Evidence Statement	Grade
Consumption of alcohol, even at low levels (10 g/d) is associated with increased risk of liver cancer in some populations.	C
Consumption of alcohol is associated with an increased risk of cancer of the oral cavity, pharynx and larynx.	C
Consumption of alcohol at the level of 1 standard drink per day for women and 1.5-2 per day for men, with a maximum intake of 4 standard drinks per day, is associated with reduced risk of dementia in older adults.	C

3.4.2.1 Cardiovascular disease, type 2 diabetes and excess weight

Cardiovascular disease: There is evidence of a probable association between consumption of one standard drink per day for women and 1.5–2 per day for men with a reduced risk of cardiovascular disease morbidity and mortality (Grade B, Section 16.1 in Evidence Report [14]) [611-613]. A meta-analysis evaluating the relationship between alcohol intake and cardiovascular mortality in patients with a history of cardiovascular disease showed a J-shaped dose effect curve, with an alcohol intake of approximately 26g/day having maximal protection. The authors concluded that light to moderate alcohol consumption, defined as 5–25g/day, is significantly associated with a lower incidence of cardiovascular and total mortality [614]. However the evidence is not yet conclusive, particularly for Asian populations [615];[604];[616];[617].

There is evidence of a probable association between consumption of one standard drink per day for women and 1.5–2 per day for men with an increase in HDL cholesterol (Grade B, Section 16.1 in Evidence Report [14]) [618-625]. Recent reviews of population-based studies [614, 626, 627] support these statements.

Although an insufficient number of studies were identified to formulate an evidence statement, excess alcohol consumption has been found to increase the risk of high blood pressure [628, 629]. A more recent review supports these findings, suggesting a synergistic effect between alcohol, hypertension and cerebrovascular events [630].

The Canadian and Australian guidelines on the management of hypertension include the moderation of alcohol intake as an important intervention [631]. High blood pressure is a major risk factor for both ischaemic and haemorrhagic stroke [632].

Type 2 diabetes: Evidence regarding an association between alcohol intake and risk of type 2 diabetes is inconclusive (Section 16.2, Evidence Report [14]).

3.4.2.2 Cancer

There is increasing evidence of an association between alcohol consumption and heightened risk of specific cancers. In general, the evidence suggests that different types of alcoholic drink have similar effects [42]. The WCRF concludes that the number of cancers for which alcohol is a known risk factor is increasing. It appears unlikely that there is a threshold of alcohol intake below which there is no effect on cancer risk.

Breast cancer: There is evidence of a probable association between consumption of alcohol, even at low levels (10 g/day), and an increased risk of breast cancer (Grade B, Section 16.4 in Evidence Report [14]) [633-639].

Oesophageal cancer: There is evidence of a probable association between consumption of alcohol and an increased risk of cancer of the oesophagus (Grade B, Section 16.6 in Evidence Report [14]) [42, 633].

Colon and rectal cancer: The evidence suggests that consumption of alcohol, even at low levels (10 g/day), is associated with an increased risk of colon cancer and rectal cancer (Grade C, Section 16.5 in Evidence Report [14]) [42, 633, 640, 641].

Liver cancer: The evidence suggests that consumption of alcohol, even at low levels (10 g/day), is associated with increased risk of liver cancer in some populations (Grade C, Section 16.9 in Evidence Report [14]) [42, 633].

Oral cavity, pharynx and larynx: The evidence suggests that consumption of alcohol is associated with an increased risk of cancer of the oral cavity, pharynx and larynx (Grade C, Section 16.7 in Evidence Report [14]) [42, 633, 642].

Non-Hodgkins lymphoma: Evidence that the consumption of alcohol is associated with Non-Hodgkins lymphoma is inconclusive (Section 16.11, Evidence Report [14]).

Other cancers: The evidence that consumption of alcohol is associated with renal, pancreatic and ovarian cancer is inconclusive (Section 16.8, 16.10, 16.12 Evidence Report [14]).

3.4.2.3 Other conditions

Alcohol-related liver disease: As discussed in the previous dietary guidelines, there is continuing evidence that excess alcohol consumption is associated with an increased risk of alcohol-related liver disease (fatty liver, cirrhosis of the liver, alcohol hepatitis) [643]. The same level of average consumption is related to a higher risk of liver cirrhosis in women than in men [644].

Dementia: The evidence suggests an association between the consumption of one standard drink per day for women and 1.5-2 per day for men, with a maximum intake of four standard drinks per day, and a reduced risk of dementia in older adults (Grade C, Section 16.3 in Evidence Report [14]) [645-651].

Alcohol use is associated with an increased risk of a number of mental health and social problems in young adults [652]. The existence of psychiatric comorbidities in young people who drink heavily is common, especially for conditions such as depression, anxiety, bipolar disorder, conduct disorder and attention-deficit/hyperactivity disorder [652-656].

Nutrition-related conditions: Alcohol consumption is linked to malnutrition, Wernicke-Korsakoff syndrome, folate deficiency, Vitamin A depletion and pellagra [657]. Excessive consumption of alcohol (severe alcoholism) leads to malnutrition if normal diet is neglected. The financial resources of the patient can be diverted away from purchase of food to acquiring and consuming alcohol. In Australia the fortification of bread with thiamin has contributed to a 40% reduction in the incidence of Wernicke-Korsakoff syndrome [658, 659].

Other conditions associated with harmful levels of alcohol consumption include:

- dependence and addiction
- endocrine conditions, for example, hypercortisonism and sexual dysfunction
- alcoholic related brain damage including alcoholic dementia
- gastritis and gastric ulcers
- aspiration pneumonia
- cardiomyopathy
- interactions with pharmaceuticals and illegal recreational drugs [609].

3.4.3 How *limiting alcohol* may improve health outcomes

Alcohol begins to affect the brain within five minutes of consumption, with blood alcohol concentration peaking after 30–45 minutes. It takes approximately one hour for the liver to clear the alcohol from one standard drink from the body, although this time varies depending upon liver size, lean body mass, individual alcohol tolerance and genes controlling the expression of alcohol-metabolising enzymes in the liver [660-662]. Because the rate of metabolism is fixed, rapid consumption of multiple drinks results in a higher blood alcohol concentration.

Young adults who drink heavily tend to have smaller prefrontal cortices and white matter, structural abnormalities of white matter and reduced hippocampal volumes [663, 664]. These structural changes lead to a diminished ability to retrieve verbal and non-verbal material and poorer performance in attention-based tests [652].

The loss of brain tissue that occurs in people with chronic alcoholism seems to occur independently of Wernicke's encephalopathy and may be related to ethanol toxicity and poor nutrition.

Cardiovascular effects: The effect of alcohol on the cardiovascular system is complex. Alcohol can raise blood pressure and increase the risk of arrhythmias, shortness of breath, some types of cardiac failure, haemorrhagic stroke and other circulatory problems. However, low levels of alcohol raise high-density lipoprotein cholesterol and reduce plaque accumulations in arteries [626, 665]. Alcohol can also have a mild anticoagulant effect.

Diabetes: Alcohol affects the management of diabetes through its effects on diet and control of blood glucose levels. Alcohol interferes with the action of insulin, insulin secretagogues and glucagon, thereby increasing the risk of hypoglycaemia in people with Type 1 or 2 diabetes who take these medications [666, 667].

Dementia: If alcohol has a protective effect against dementia, this may relate to the effect of alcohol on blood lipids as one of the causal factors of dementia is microvascular changes within the brain [668, 669].

3.4.4 Practical considerations: *Limiting alcohol*

Of Australians aged over 14 years, 83% reported having consumed alcohol at least once in the 12 months preceding the 2007 National Drug Strategy Household Survey, with 8% drinking alcohol on a daily basis and about 47% consuming alcohol at least weekly [670]. The majority of Australians who reported consuming alcohol also reported moderating their intake, primarily to reduce the risk to their health. Methods included counting and limiting the number of drinks, eating food while consuming alcohol, alternating between alcoholic and non-alcoholic drinks and drinking low-alcohol drinks [670].

Nearly all alcohol is consumed as drinks, principally beers and wines. Alcoholic drinks contain few other nutrients except for the bioactive flavonoids found in wine, mainly in red wine. Alcoholic drinks are usually consumed with foods, either as part of a meal or accompanied by snack foods, increasing the associated energy intake.

The apparent average consumption of alcohol is estimated at 10.08L per person over 15 years old per year [671], and declines with age [672]. Consumption is 45% higher in the Northern Territory than in the rest of Australia. The average consumption equates to an additional 650 kJ/day for every person over 15 years of age from alcohol. Alcoholic drinks that contain added sugar have additional energy. Clearly if alcohol is consumed in addition to the normal diet, leading to excess energy intake compared to requirements, weight will increase.

A full stomach will reduce the rate of absorption of alcohol into the bloodstream. Drinking alcohol in combination with eating therefore reduces the rate at which blood alcohol content increases.

Drinking coffee, having a cold shower, vomiting or exercise do not reduce blood alcohol content [609].

3.4.4.1 Pregnant and breastfeeding women

Alcohol consumption by pregnant women may harm the unborn baby. Heavy daily drinking or heavy episodes of drinking have the most risk, and the risk from low-level drinking (one or two drinks per week) is likely to be small. However there is no lower limit that can be guaranteed to be completely safe, so avoiding alcohol while pregnant or breastfeeding is the safest option [609].

There is limited research on the effects of maternal alcohol consumption during lactation and infant development. Mothers who consume alcohol are more likely to stop breastfeeding before six months than mothers who do not drink [673, 674]. Animal and observational studies suggest that the consumption of two standard drinks or more per day during lactation is associated with deficits in infant psychomotor development and disrupted infant sleep-wake behavioural patterns [673]. A baby's intake of alcohol from breastmilk is not harmless. Alcohol levels in breastmilk parallel blood alcohol levels and therefore the longer the time between drinking alcohol and breastfeeding, the safer for the baby. The safest option for women who are breastfeeding is to abstain from alcohol (see Section 3.4) [609]. For those who drink, expressing milk before consuming alcohol is the next best option [609].

3.4.4.2 Children and adolescents

Alcohol use by younger people is associated with harmful effects on brain development. The NHMRC recommends that parents and carers should be advised that children under 15 years of age are at the greatest risk of harm from drinking. For this age group, not drinking alcohol is especially important. For people aged 15–17 years, the safest option is to delay drinking for as long as possible [609].

3.4.4.3 Older people

Older people are more susceptible than others to the toxic effects of alcohol due to changes in their body composition, decreased metabolic capacity, the presence of co-morbid conditions and medications that regulate these conditions [675].

Many older people take medications that may interact with alcohol. A combination of alcohol and medication increases the risk of falls and injury [676-678].

The NHMRC Alcohol Guidelines state that 'cumulative alcohol-related harm is more evident among older people. For some older adults, drinking alcohol increases the risk of falls and injuries,

as well as some chronic conditions. Older people are advised to consult their health professionals about the most appropriate level of drinking for their health' [609].

3.4.4.4 Aboriginal and Torres Strait Islanders

Aboriginal or Torres Strait Islander peoples are more likely than other Australians to abstain from drinking alcohol (23% compared to about 15%). However, those who do consume alcohol are more likely to do so at risky or high-risk levels for short-term harm [670]. In response to severe problems related to excess alcohol consumption in many Indigenous communities, initiatives have been introduced to encourage non-harmful alcohol use, limit access to alcohol, and establish 'dry' areas and communities. As with the general population, Aboriginal and Torres Strait Islander people should follow the alcohol guidelines described above [609].

3.4.4.5 Culturally and linguistically diverse groups

People from culturally diverse groups are more likely than the general adult population in Australia to abstain from alcohol (43% compared to 15%) [670]. The possible protective health effect of moderate drinking has not been demonstrated in Asian groups. Alcohol drinking customs vary in different cultures and typically immigrant groups bring their drinking patterns from their country of origin.

3.4.4.6 People with diabetes

As alcohol and hypoglycaemia have independent but additive effects on cognitive function and behaviour, it is recommended that people with diabetes abstain from alcohol if they plan to drive [679]. Alcohol worsens medical conditions associated with diabetes, such as liver disease, hypertension and advanced neuropathy [666, 680]. People with diabetes may need to take special precautions when drinking and should discuss alcohol use with a health professional.

3.4.4.7 Interaction of alcohol and energy drinks

A new category of alcoholic drinks is now being marketed in Australia with added caffeine and sometimes other stimulants. The effects of alcohol and other components in these energy drinks appear to be synergistic, resulting in increased intoxication. In comparison to alcohol alone, the combination of alcohol and energy drink significantly reduces the intensity of subjective perceptions of headache, dry mouth, weakness and impairment of motor coordination [681]. The concern is that consuming caffeine, a central nervous system stimulant, and alcohol, a depressant, at the same time will reduce subjective perceptions of alcohol-induced impairment in comparison to alcohol alone [682, 683]. Reduced ability in recognising alcohol impairment may enhance risk-taking behaviour and possibly lead to greater alcohol intake [684].

In the absence of any research to quantify safe levels of concurrent consumption of energy drinks and alcohol, this combination should be avoided.

Where to next

The next chapter provides information on the importance of quantity, as well as quality and overall energy balance, when choosing foods to promote healthy weight, to promote health and wellbeing and to prevent chronic disease.

4. Achieve and maintain a healthy weight

Guideline 3

To achieve and maintain a healthy weight you should be physically active and choose amounts of nutritious foods and drinks to meet your energy needs.

- Children and adolescents should eat sufficient nutritious foods to grow and develop normally. They should be physically active every day and their growth should be checked regularly.
- Older people should eat nutritious foods and keep physically active to help maintain muscle strength and a healthy weight.

Executive summary

Healthy weight is associated with reduced risk of chronic disease, including cardiovascular disease, type 2 diabetes and some cancers. Unhealthy weight refers to being underweight, overweight or obese.

An optimum dietary pattern for adults to achieve and maintain healthy weight is one in which nutrient requirements are met and total energy intake does not often exceed total energy expenditure.

Physical activity is an important part of a healthy, active life.

The high and increasing prevalence of overweight and obesity in Australia highlights the need to provide guidance for achieving and maintaining a healthy weight. Small, persistent excess energy intake will cause excess weight gain in people of any age.

Recent evidence highlights the importance of achieving an appropriate energy intake, rather than targeting specific macronutrients (protein, fat or carbohydrates). In general the quantities of foods outlined in the *Australian Guide to Healthy Eating* should not be exceeded and consumption of energy-dense, nutrient-poor discretionary foods and drinks should be limited.

Weight should be measured regularly in adults and the amount and/or quality of food, drinks and physical activity adjusted accordingly. Children and adolescents need sufficient nutritious foods to grow and develop normally and their growth should be checked regularly to ensure appropriate development is occurring.

As most Australian adults are now of unhealthy weight, this chapter offers population level advice on managing weight gain and reduction while discouraging inappropriate food restriction, particularly among high risk groups.

This chapter provides information on the importance of quantity, as well as quality, when choosing foods to achieve healthy weight, to promote health and wellbeing and prevent chronic disease.

4.1 Setting the scene

A healthy weight is a body weight associated with normal growth and development in children, and a reduced risk of short- and long-term morbidity and mortality among people of all ages [685-689]. While it is unhealthy to be underweight, overweight or obese [88, 685], ideal weight varies from one person to another and at different stages of life. A small or large persistent excess energy intake can cause unwanted weight gain in people of any age.

There is widespread recognition of a global epidemic of overweight and obesity which requires urgent attention [89, 685, 690, 691]. Overweight and obesity contributed 7.5% of the burden of premature death and disability in Australia in 2003[11]. In Western Australia and Queensland, where more recent data are available, at 8.3–8.6% it has now overtaken cigarette smoking as the single greatest contributor of the biomedical risk factors assessed [692, 693].

A focus on healthy weight is a more positive way to address weight issues than focusing on obesity and overweight. It encourages those who are a healthy weight to maintain that weight. It also helps reduce the risk of any unintended negative consequences, such as disordered eating. Promotion of healthy weight incorporates prevention and management of underweight, overweight and obesity in children and adults and promotes healthy growth in children.

4.1.1 Weight status and trends in Australia

4.1.1.1 Adults

Measured height and weight data in 2007-08 showed that 2% of Australian adults were underweight, 37% were of healthy weight, 37% were overweight and 25% were obese [694]. Overweight and obesity was more common in men than women (68% v 55%) and in people aged 65–74 years (75%) than in other age groups.

The prevalence of overweight and obesity has increased significantly in Australia and most developed countries since the 1970s [685, 691, 695], although there is some evidence of a reduction in the rate of increase in children and adolescents in Australia over the past decade [696].

4.1.1.2 Children and adolescents

A number of national and state-based surveys of children and adolescents using measured height and weight data have found that between 21–25% of children and adolescents are overweight and/or obese. Between 5–8% are obese and 2–5% are underweight [12, 13, 697-700]. The

prevalence of obesity alone is higher for boys than girls (10% compared with 6%) [12], although the prevalence of combined overweight and obesity is similar (26% for boys and 24% for girls) [12]. The prevalence of overweight and obesity has increased significantly over the past two decades. Although the rate of increase appears to be slowing in children [701], the high prevalence remains of concern [702].

Childhood obesity has been identified as one of the most serious public health challenges of the 21st century [703]. In the United States it has been predicted that - due to premature mortality associated with obesity developing at a younger age - the current generation of children will be the first in that country's history to have a life expectancy lower than their parents[704]. As obesity is an important determinant of a range of health disorders, unless the increasing prevalence of overweight and obesity is arrested the burden of chronic disease in future generations will be pandemic [6, 695], causing a crisis in health and economic systems across the world [88, 685, 688].

4.1.1.3 Predicted trends

If current trends continue in Australia, it is estimated that by 2025, 83% of men and 75% of women aged 20 years or more will be overweight or obese [705].

The predicted increases would significantly affect disease burden and health care costs, mostly due to an increased incidence of type 2 diabetes. For example, without intervention, type 2 diabetes will account for around 9% of the total disease burden in Australia in 2023, up from around 5% in 2003 [11].

How is healthy weight assessed?

The most common approach is the use of the body mass index (BMI) calculated as weight (in kilograms)/height (in metres)^2 (kg/m^2).

BMI is a measure of body size that is widely used as an index of relative risk of mortality and morbidity at the population level [687, 691]. The association of mortality with BMI is a U-shaped curve with the lowest risk within the healthy weight range [687, 706].

An adult with a BMI < 18.5 kg/m^2 is categorized as underweight. An adult with a BMI from 18.5 to 25.0 is a healthy weight, while an adult with a BMI ≥ 25.0 kg/m^2 is overweight and an adult with a BMI ≥ 30.0 kg/m^2 is obese [691, 707].

However, this classification may not be suitable for all ethnic groups. Some groups may have equivalent levels of risk at lower BMI (e.g. people of Asian origin) or higher BMI (e.g. people of Polynesian origin) [708]. A BMI range of 23–28kg/m^2 may be desirable for people aged over 70 [709]. BMI ranges have not been developed for Aboriginal and Torres Strait Islander people.

Waist circumference is also used as a proxy for health risk in adults. A waist circumference above 80 centimetres in women or above 94 centimetres in men signifies being overweight. A waist circumference above 88 centimetres in women or above 102 centimetres in men signifies being obese [13, 710].

Growth during childhood means it is not possible to have a single set of numerical values for BMI cut-offs that apply to all ages and both sexes. Therefore, among children and adolescents, weight appropriateness is commonly categorised using Z-scores (or SD Scores) [711] or age-related cut-off values [712, 713]. For more information, see Appendix 3.

4.1.2 Health effects associated with weight status

4.1.2.1 Adults

Overweight and obesity are associated with increased risk of type 2 diabetes, cardiovascular disease, some cancers, hypertension, musculoskeletal conditions, respiratory conditions, social isolation, depression and other psychological disorders, sleep apnoea, cholecystitis, insulin resistance and metabolic syndrome, hernia, reproductive disorders, urinary incontinence and skin conditions [88, 89, 685]. About 70% of people who are obese have at least one established co-morbidity, resulting in medical costs about 30% greater than their healthy-weight peers [714].

Compared to having a BMI between 18.5 and 25, at a BMI of 30–35kg/m^2, median survival is reduced by 2–4 years, at BMI 40–45kg/m^2 it is reduced by 8–10 years [706]. The relative increase in mortality rate attributable to obesity tends to decline with age [715]. Mortality and morbidity are also associated with the amount of weight gained in adult life [89, 685, 688].

Many obesity-related conditions are preventable, and several are at least partially reversible through weight loss achieved by adopting a nutritious dietary pattern and active lifestyle [89, 685, 688].

While the greatest risk to health on a population basis is associated with being overweight, being underweight can also have adverse health consequences including decreased immunity (leading to increased susceptibility to some infectious diseases), osteoporosis, decreased muscle strength, and hypothermia[37]. Among older people, being underweight may be more deleterious to health than being overweight [716].

4.1.2.2 Children and adolescents

The most immediate consequences of overweight/obesity in childhood is social discrimination associated with poor self-esteem and depression, increased risk of development of negative body image issues, and eating disorders [688]. Overweight children and adolescents are more likely to develop sleep apnoea, heat intolerance, breathlessness on exertion and reduced exercise tolerance, tiredness, some orthopaedic and gastrointestinal problems, steatohepatitis and early signs of metabolic and clinical consequences, such as hypertension, hyperinsulinaemia, hypertriglyceridaemia and type 2 diabetes [688, 717].

A major long-term consequence is that overweight children are more likely to become overweight adults with an increased risk of chronic diseases and early mortality [688, 717-719]. The risk of chronic disease is increased with rapid weight gain in infancy and early childhood [88, 89].

In infancy and early childhood, underweight and failure to thrive can be more prevalent than overweight and obesity in some communities. Failure to thrive is most commonly a result of socioeconomic factors, including poor living conditions [720] but can also occur among affluent sections of the community due to inappropriate dietary restrictions, for example based on fears about 'unhealthy' dietary habits leading to the development of atherosclerosis [721]. Specialist advice should be sought on underweight and failure to thrive in infants and children.

Inappropriate dietary restriction and eating disorders occur in some adolescents [722].

4.1.3 Causes of overweight and obesity in the population

Healthy body weight results from an appropriate balance between energy intake and expenditure (of which physical activity is a component) [89, 688, 723]. At the population level, there is increasing evidence that excess energy intake is a major contributor to current energy imbalances [88, 723-728].

4.1.3.1 The obesogenic environment

Obesity is a complex condition mediated by an individual's metabolism and behaviour as well as societal and environmental factors.

Although some genetic factors may increase an individual's susceptibility to being overweight or obese, the dramatic increases in prevalence of overweight and obesity over the past three to four decades without any major change in the gene pool suggests that socio-environmental factors are primarily responsible for the current epidemic [88, 89, 685, 688, 726, 727, 729]. Significant socio-environmental changes during this period include, but are not limited to:

- changes in the food supply, particularly increased availability and decreased relative cost of foods which tend to be higher in energy density and relatively nutrient-poor [730]
- increased availability and marketing of energy-dense, nutrient-poor foods and drinks
- urban design which reduces energy expenditure during daily activities and increased reliance on car and labour-saving devices [726, 731]
- reduced perceptions of safety leading to fewer opportunities for physical activity [732, 733]
- economic and consumer changes, such as a greater number of females in the paid workforce, decreased food literacy and cooking skills, and greater reliance on convenience and takeaway foods [728, 729, 734].

These changes all promote excess weight gain by fostering consumption of energy-dense and relatively nutrient-poor foods and/or reduced physical activity [726, 735-738].

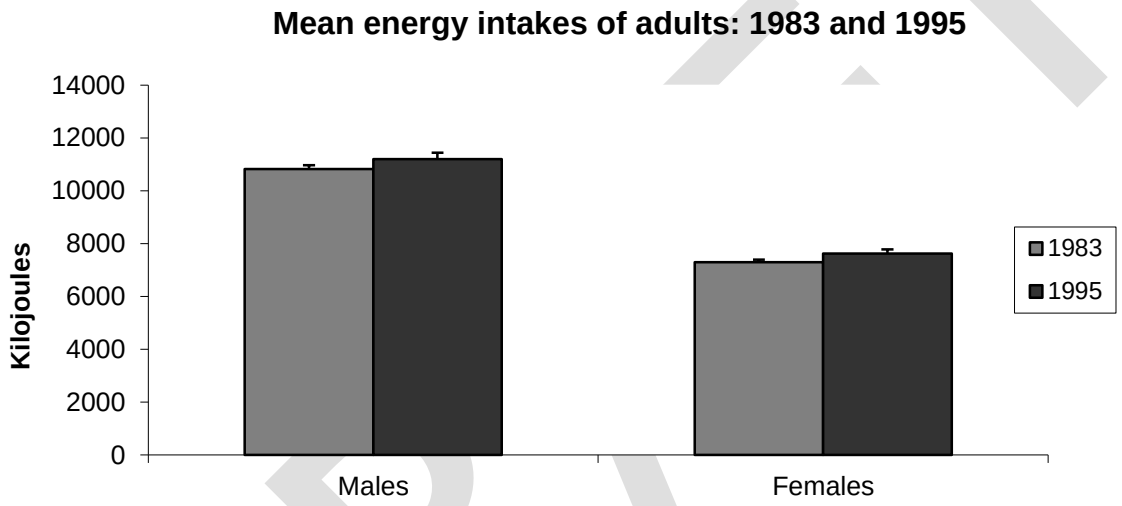
Against such social and physical environments, overweight and obese individuals need support, rather than criticism and discrimination. Healthy weight initiatives must achieve a balance between individual and societal responsibility and be culturally appropriate, widely available and accessible, particularly to disadvantaged and vulnerable groups [89, 739].

4.1.3.2 Energy intake and trend data

Energy intake increased in the decade or so to 1995 by 3–4% in adults (see Figure 4.1) [740-743], This increase equates to an additional 900kJ to1400kJ per day across all groups. Without compensatory increases in energy expenditure, these changes are enough to result in the significant observed increase in mean body weight [48].

More recent data will be available soon as dietary intakes of people aged two years and over are being collected in the Australian Health Survey in 2011-2012 [93].

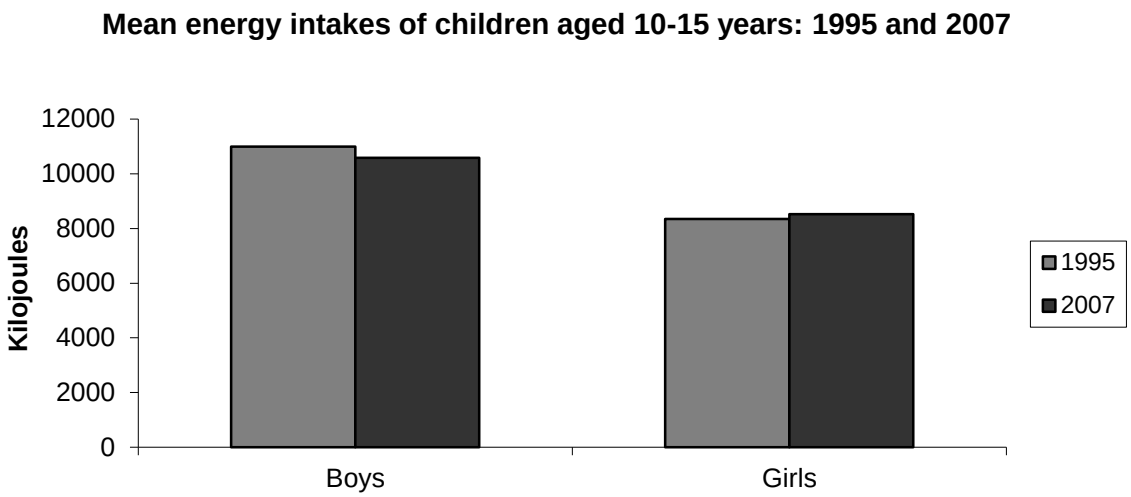
Figure 4.1 Mean energy intakes of adults: 1983 and 1995*



* Figure adapted from Cook et al (2001)[48]

Energy intake in the decade or so from 1983 to 1995 increased by 11% for 10–15 year-olds girls and by 15% for 10–15 year-olds boys [48]. Accounting for seasonal differences in the data collections, the results of the *2007 Australian National Children's Nutrition and Physical Activity Survey* show that mean energy intakes have since decreased in boys aged 10-15 years and slightly increased in girls aged 10-15 years from 1995 to 2007 (see Figure 4.2).

Figure 4.2 Mean energy intakes of children aged 10-15 years: 1995 and 2007**



** Unpublished FSANZ analysis; 1995 data restricted to February to August to match the time frame of the 2007 survey; 2007 survey analysis used the first day of food data only.

4.1.3.3 Energy from macronutrients

Macronutrients (proteins, fats and carbohydrates) all contribute to dietary energy intake [9]. Alcohol also contributes to dietary energy, and it is recommended that alcohol intakes should contribute less than 5% of dietary energy because of the negative association between intake of alcohol and health outcomes (see Chapter 3).

There is a growing body of evidence that the relative proportions of macronutrients consumed can affect the risk of chronic disease and may also affect micronutrient intake [9]. Optimal proportions of the type of fat (for example, saturated, polyunsaturated or monounsaturated or specific fatty acids within one or more of these categories) and carbohydrate (for example, complex or simple, starches or sugars) may also be important to reduce chronic disease risk [9]. The estimated Acceptable Macronutrient Distribution Ranges related to reduced risk of chronic disease, are:

- 20-35% of total energy intake from fat
- 45-65% from carbohydrate
- 15–25% from protein [9].

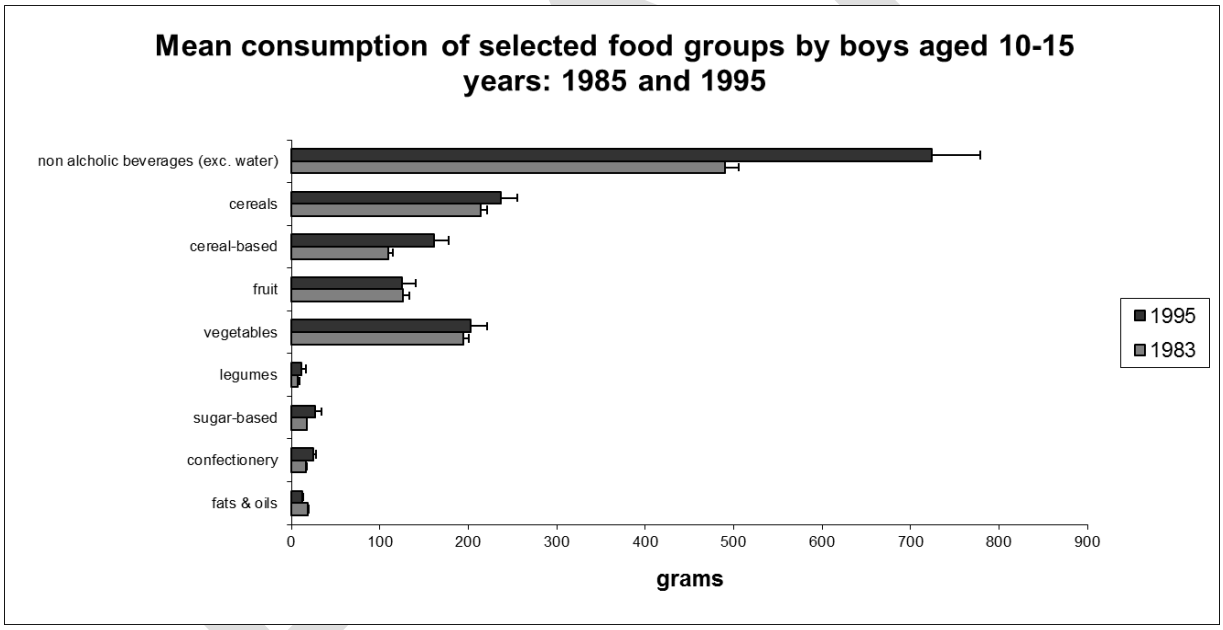
The risk of chronic disease, overweight and obesity, and inadequate micronutrient intake may increase outside these ranges, but there are insufficient data available at extremes of population intake [9].

4.1.3.4 Energy intake from specific food groups

The increased energy consumption in the decade or so to 1995 was largely driven by an increase in the consumption of cereal-based foods (including cakes, biscuits, pies, pizza and some desserts), confectionery and sugar-sweetened drinks [48]. In 1995, Australians consumed two to four times the recommended limits of energy-dense and nutrient-poor foods, classified as extra foods, contributing almost 36% and 41% of total energy intake and 41% and 47% of total fat intake in adults and children, respectively [46, 744].

As an example, changes in food group consumption for boys are illustrated in Figure 4.3. Among these was a significant decrease in intake of fats and oils from oils and spreads, but an increase in intake of fats from other sources, such as cereal-based foods and confectionery [48], most of which were extra foods in the *Australian Guide to Healthy Eating* [45]. No subsequent reliable, comparable national data are available.

Figure 4.3 Mean consumption of selected food groups by boys aged 10–15 years: 1985 and 1995*



* Figure adapted from Cook et al (2001)[48]

4.1.3.5 Energy expenditure

Total daily energy expenditure includes resting energy expenditure (basal metabolic rate plus necessary tissue repair and the thermic effect of food), and energy expended in physical activity [9]. Resting energy expenditure makes up 60-80% of total energy expenditure, and is mainly related to lean body mass [9]. Active energy expenditure, which accounts for up 20-40% of total

energy expenditure, depends on both the amount of physical activity and the body mass to be moved during the process. Active energy expenditure is the only aspect of energy expenditure which is under conscious control through physical activity [9, 745].

Resting energy expenditure, active energy expenditure and total energy expenditure are all substantially increased in obesity, [746] which contradicts the view that obesity is due to 'low metabolism' and is maintained despite a low level of food intake [723]. However energy expenditure per kilogram of body mass does decline with increasing BMI, even at the same physical activity level [745].

4.1.4 Physical activity

Physical activity includes both structured activities such as sport or organised recreation and unstructured activities such as incidental daily activities at work or home such as gardening or walking/cycling for leisure or transport [747].

For the current Australian recommendations for physical activity, see Appendix 4.

4.1.4.1 Physical activity levels of specific groups

The proportion of Australian adults reporting recommended levels of physical activity declined from 62% in 1997 to 57% in 2000 [748], with no subsequent reliable national data available for comparison. The 2007–08 National Health Survey results cannot directly assess compliance with physical activity recommendations due to different methodology. However, state-based survey results suggest small increases in physical activity participation at levels providing health benefits since around 2004 [4, 749, 750]

The National Children's and Adolescent's Nutrition and Physical Activity Survey found that 69% of Australian children were likely to meet the physical activity guidelines on any given day [13]. Adolescent girls were less active than boys, particularly in the older age groups. Underweight and obese children tended to have lower physical activity levels than children of a healthy weight [13]. Available state data were generally consistent with these findings [697, 751, 752].

In 2007, Australian children aged 9–16 years spent more than 3.5 hours/day on average in sedentary behaviour such as watching television, playing video/computer games and/or using computers more generally [13]. On any given day, 67% of children spend more than the recommended maximum of 120 minutes of recreational screen time.

4.1.4.2 Benefits of physical activity

Physical inactivity accounted for 6.6% of the burden of disease in Australia in 2003. Substantial population health gains are possible if the community adopts more regular moderate physical activity [11]. Being physically active:

- reduces the risk of all-cause mortality [753, 754]
- is an important factor in the prevention and management of a range of chronic diseases, including heart disease, stroke, high blood pressure, type 2 diabetes and some cancers [755, 756]
- is associated with reduced risk of injury [755, 756]
- offers other health benefits, including building and maintaining healthy bones, muscles and joints [755, 756]
- improves self-esteem, self-image and quality of life [755-758].

The greatest health benefit is found in moving from no activity to low levels of activity, but even at higher levels of activity, benefits accrue from additional activity [753, 754]. Benefits have been described for all age groups and physically active children are more likely to remain physically active throughout adolescence and into adulthood [759, 760].

4.2 The evidence for *'achieving and maintaining a healthy weight'*

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) related to 'healthy weight' from the Evidence Report (literature from years 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III in which individual studies were classified according to their design but overall grades for relationships were not derived), although these sources have been used to inform the *Guidelines*.

Evidence Statement	Grade
Compared to infants who are formula fed, being breastfed is associated with reduced risk of becoming obese in childhood, adolescence, and early adulthood.	A
Increased birth weight, especially above 4,000g, is associated with increased risk of overweight or obesity in childhood, adolescence, and later in life.	A
Excessive weight gain relative to height during childhood is associated with	A

an increased risk of overweight later in life.	
Parental overweight or obesity is associated with increased risk of child overweight or obesity. The risk is greater when both rather than one parent is overweight or obese.	A
Behavioural interventions including diet and exercise reduce the risk of overweight or obesity in overweight children. These interventions are more effective when they are family-based.	A
Lifestyle interventions combining diet and physical activity interventions are associated with reduced risk of developing type 2 diabetes in adults.	B
Consumption of sugar-sweetened beverages is associated with increased risk of weight gain in adults and children	B
Consumption of 3-5 serves per day of cereal foods (mainly wholegrain) is associated with a reduced risk of weight gain	B
Babies born to mothers who smoke during pregnancy are at an increased risk of development of overweight or obesity in adolescence and adulthood.	B
Combined diet and physical activity interventions is associated with reduced risk of overweight and obesity in children	C
Combined diet and physical activity interventions is associated with reduced risk of overweight and obesity in adults.	C
Consumption of vegetables is associated with reduced risk of weight gain	C
Consumption of fruit is associated with a reduced risk of obesity and weight gain.	C
Hours spent watching television by children is associated with increased risk of development of overweight or obesity.	C
In developed countries, a low family income or socioeconomic status is associated with increased risk of overweight or obesity during childhood, adolescence, and young adulthood.	C
Low socio-economic status is associated with an increased risk of overweight or obesity.	C
Interventions delivered in the school environment that are focused on eating and physical activity improve weight outcomes in children.	C

4.2.1 Primary prevention

Diet and physical activity: The evidence from recent reviews of combined diet and physical activity interventions for children suggests that these can prevent overweight and obesity (Grade C, Section 22.1 in Evidence Report [14]) [761-765].

The evidence from primary prevention diet and physical activity interventions in adults also suggests that these can prevent overweight and obesity (Grade C, Section 22.2 in Evidence Report [14]) [766]. Lifestyle interventions combining diet and physical activity interventions are probably associated with reduced risk of developing type 2 diabetes in adults (Grade B, Section 22.3 in Evidence Report [14]) [761-765].

Favourable outcomes were consistently observed in interventions focusing on both reduced energy intake and increased physical activity, supporting the previous evidence statements that combined interventions assist weight loss and weight control in both children and adults [767].

4.2.1.1 Dietary patterns and specific foods and drinks

Fat: The previous dietary guidelines and many international public health organisations, including the World Health Organization [685], emphasised the major role of fat consumption in the development of obesity and of reducing fat intake in the dietary management of obesity or overweight. More recently, WHO has shifted its emphasis, saying there is convincing evidence that energy balance is critical to maintaining healthy weight and ensuring optimal nutrient intakes, regardless of macronutrient distribution and percentage of total fat [768].

Sugar: No large long-term studies have measured the long-term development of overweight or obesity specifically related to sugar consumption (Section 14.3, Evidence Report [14]). However, recent evidence shows it is probable that consumption of sugar-sweetened drinks (soft-drinks) is associated with increased risk of weight gain in adults and children (Grade B, Section 15.1 in Evidence Report [14]) [448-459]. A later longitudinal study adds confirmation [769].

The literature review to inform the revision of the *Dietary Guidelines for Americans, 2010* found strong evidence that greater intake of sugar-sweetened drinks is associated with increased adiposity in children and moderate evidence that consumption of sugar-sweetened drinks is associated with increased body weight in adults [143]. Most of the relevant research (see Chapter 3) was conducted in the United States where, unlike Australia, fructose/high fructose corn syrup is commonly used to sweeten soft drinks. Although these sweeteners differ only slightly from those used commonly in Australia, this was taken into consideration in the grading of the Evidence Statement for this guideline.

The review for the US guidelines also found strong and consistent evidence that glycaemic index and/or glycaemic load are not associated with body weight and do not lead to greater weight loss

or better weight management [143]. These factors were not included in the literature review to inform the revision of these *Guidelines.* There is also considerable variability in these indices depending on inter-and intra-individual factors and the form of food (including the degree of processing, stage of ripeness, cooking and cooling times), which may limit practical application [770].

Specific foods and drinks: There is increasing evidence that consumption of specific foods and food groups is associated with risk of excess weight gain. There is evidence suggesting that consumption of vegetables is associated with a reduced risk of weight gain (Grade C, Section 2.2 in Evidence Report [14]) [156, 158-160]. The evidence also suggests that consumption of fruit is associated with a reduced risk of obesity and weight gain (Grade C, Section 1.3 in Evidence Report [14]) [156-160, 190-193]. The literature review to inform the revision of the *Dietary Guidelines for Americans, 2010* found that the evidence for an association between increased fruit and vegetable intake and lower body weight is modest, but may be important in the long term [143].

The US literature review also found strong evidence of a positive relationship between portion size and body weight [143].

Recent evidence suggests that consumption of dairy foods is not associated with weight change or risk of obesity (Grade C, Section 5.8 in Evidence Report [14]) [382, 385-389] and that consumption of milk is not associated with BMI or BMI change in childhood (Grade C, Section 5.9 in Evidence Report [14]) [265, 385, 391, 393, 394, 771]. These findings are consistent with those of the literature review to inform the revision of the *Dietary Guidelines for Americans, 2010* which found strong evidence that intake of milk and milk products provide no unique role in weight control [143].

There is evidence of a probable association between consumption of 3–5 serves per day of grain (cereal) foods (mainly wholegrain) and a reduced risk of weight gain (Grade B, Section 6.5 in Evidence Report [14]) [249, 261-270].

There is evidence to suggest that consumption of nuts (between 65–110gm per day) is not related to risk of weight gain in the short term (Grade C, Section 8.1 in Evidence Report [14]) [353-358].

The literature review to inform the revision of the *Dietary Guidelines for Americans, 2010* also found that, for most children, there was limited evidence that intake of 100% fruit juice is associated with increased adiposity when consumed in amounts that are appropriate for the age and energy needs of the child. However, increased intake of 100% fruit juice was found to be associated with increased adiposity in children who were already overweight or obese [143].

In seeking to achieve and maintain a healthy weight it is prudent to choose nutrient-dense foods of lower energy density (that is, those low in total fat, particularly saturated fat, and added sugars (see Chapter 3) in a total dietary pattern that seeks to control overall energy intake [772]. The literature review to inform the revision of the *Dietary Guidelines for Americans, 2010* found strong

and consistent evidence that dietary patterns that are relatively low in energy density improve weight loss and weight maintenance in young adults [143].

4.2.1.2 Factors associated with risk of overweight and obesity

Breastfeeding: There is convincing evidence that breastfeeding infants, compared with formula feeding, is associated with a reduced risk of becoming obese in childhood, adolescence and early adulthood (Grade A, Section 17.2 in Evidence Report ([14])[773].

Birthweight: Increased birthweight, especially above 4,000g, is associated with increased risk of overweight or obesity in childhood, adolescence, and later in life (Grade A, Section 17.1 in Evidence Report [14]) [54, 58, 61, 62, 79, 774, 775]. There is a J- or U-shaped relationship between birthweight and increased risk of child or adult obesity, with both low birthweight and high birthweight babies being at increased risk [776].

Childhood weight gain: There is convincing evidence that excessive weight gain relative to height during childhood is associated with increased risk of being overweight later in life (Grade A, Section 17.4 in Evidence Report [14]) [58, 777, 778].

There is convincing evidence that parental overweight or obesity is associated with increased risk of child overweight or obesity. The risk is greater when both rather than one parent is overweight or obese (Grade A, Section 17.7 in Evidence Report [14]) [49-62].

Maternal smoking: There is evidence that babies born to mothers who smoke during pregnancy, as an independent risk factor, probably have a higher risk of becoming overweight or obese in adolescence and adulthood (Grade B, Section 17.5 in Evidence Report [14]) [52, 54, 59-61, 779].

Television: Recent evidence suggests that hours spent watching television by children is associated with increased risk of overweight or obesity (Grade C, Section 17.3 in Evidence Report [14]) [49, 60, 64-71, 780-782]. Media use, including television viewing, may displace time children spend in physical activities [782, 783] and eating meals and snacks in front of television may also be associated with increased energy intake [784].

Socioeconomic status: There is evidence from developed countries to suggest that a low family income or socioeconomic status is associated with increased risk of overweight or obesity during childhood, adolescence and young adulthood (Grade C, Section 17.9 in Evidence Report [14]) [49, 52, 55, 64, 65, 68, 72-74]. Similarly the evidence suggests that low socio-economic status is associated with an increased risk of overweight or obesity (Grade C, Section 17.10 in Evidence Report [14]) [59, 62, 775, 785-788].

Other factors: Although there were insufficient studies to make an evidence statement, other factors associated with increased risk of overweight and obesity throughout life included:

- being overweight in adolescence [78]
- consumption of any take away food and low quality snacks [75-77]
- childhood smoking [78, 83]
- increased price of fruit and vegetables [68, 79]
- low self esteem and/or depression [80-82]
- low locus of control score [84, 85]
- stressful family life [86, 87]
- food insecurity [65, 81, 789]
- self-reported dieting [76, 82, 790], particularly in girls [791]
- inadequate sleep [60, 780, 792, 793]
- low rates of breakfast consumption [794].

The literature review to inform the revision of the *Dietary Guidelines for Americans, 2010* found strong and consistent evidence indicating that children and adults who eat fast food, particularly those eating at least one fast food meal per week, are at increased risk of weight gain, overweight and obesity. There was not enough evidence at this time to similarly evaluate eating at other types of restaurants and risk of weight gain, overweight and obesity [143]. The US literature review also found moderate evidence suggesting that children who do not eat breakfast are at increased risk of overweight and obesity, with the evidence being stronger for adolescents [143].

There appear to be complex relationships between dietary patterns as a child and dietary quality over time. Studies in the US suggest that frequency of consuming take away food increases with age and is associated with higher intakes of energy, total fat, saturated fat and sodium [90], while frequency of breakfast consumption decreases with age and skipping breakfast is associated with reduced intake of calcium and dietary fibre [91]. There is some evidence that family meal patterns during adolescence predict diet quality and meal patterns during early young adulthood [92].

The literature review to inform the revision of the *Dietary Guidelines for Americans, 2010* also found a limited body of evidence showing conflicting results about whether liquid and solid foods differ in their effects on energy intake and body weight, except that soup at a meal may lead to decreased energy intake and body weight [143].

Finally, an emerging body of evidence documents the impact of the food environment on body weight in children and adults. Moderately strong evidence now indicates that the food environment is associated with dietary intake, especially lower consumption of vegetables and fruits and intakes resulting in higher body weight [143]. This is discussed further in Appendix 7.

4.2.2 Secondary prevention

Behavioural interventions: There is convincing evidence that behavioural interventions including diet and physical activity reduce the risk of obesity in overweight children. These interventions are more effective when they are family-based (Grade A, Section 17.12 in Evidence Report [14]) [795-798].

Recent evidence suggests that interventions delivered in the school environment that are focused on healthy eating and physical activity improve weight outcomes in children (Grade C, Section 17.11 in Evidence Report [14]) [799, 800].

4.3 How *dietary patterns* can affect energy intake and balance and weight outcomes

From a population perspective, the dramatic increase in the prevalence of obesity in Australia and other developed countries over the past 30 years cannot be explained by genetic factors. Environmental and lifestyle factors resulting in a decrease in physical activity and overconsumption of energy in the diet provide the most reasonable explanation. Only a small, persistent energy imbalance is required to cause excessive weight gain in both children and adults, which over time progressively increases BMI [723, 727]. Available data from developed countries, including Australia, confirm an increase in energy intake concurrent with the dramatic increase in the prevalence of overweight and obesity in children and adults since 1980 [727].

There has been much debate about the role of energy, carbohydrate and fat intake in the obesity epidemic [772, 801, 802]. While reducing fat intake has been recognised as an important strategy to reduce energy intake in successful weight loss interventions [801], there is little evidence that population fat intake is associated with the obesity epidemic independently of total energy intake [802].

Foods with a higher energy density encourage energy intake above requirements [143, 803]. However, foods that are high in energy density tend to be more palatable, and high palatability is associated with increased food intake in single-meal studies [730, 804]. Fat and sugar are positively associated with energy density while water and dietary fibre are negatively associated. Fat plays a role because of its high energy density compared to protein and carbohydrates. Water and dietary fibre play a role through a dilution effect, although this is less for dietary fibre because of the much smaller range of fibre concentration in food compared with both water and fat.

It is plausible that the proportions of macronutrients (fat, carbohydrate, protein and alcohol) and types of macronutrients comprising total energy intake may affect an individual's propensity to habitually overconsume. In this regard, dietary patterns which tend to be relatively low in total fat

and moderate (not high) in carbohydrate are consistent with reduced risk of excessive weight gain. Energy from drinks, in particular, may add to total energy intake without displacing energy consumed in the form of solid food, and it is plausible that energy-dense drinks may contribute to excessive energy intake through lack of impact on satiety [771]. The satiety value of foods may also be important in managing appetite and hunger. Evidence suggests that protein-rich foods and dietary patterns have a greater effect on satiety than foods and dietary patterns high in fat and carbohydrate [805].

Energy-dense dietary patterns are associated with higher consumption of grain-based foods, fats and sweets and lower consumption of vegetables and fruit [806]. International data suggest that the major foods contributing to increased energy intake include sweetened drinks, snack foods and fast food [803, 807, 808] and that increasing portion size is also an important contributor [143, 809, 810]. The low cost of energy-dense nutrient-poor food relative to nutrient-dense food is also a major contributor to the obesity epidemic [803, 806].

Positive outcomes have been described, at least in the short term, in clinical weight loss regimes that include both dietary and physical activity interventions [811, 812].

4.4 Practical considerations: *Achieving and maintaining a healthy weight*

As is the case internationally [714], overweight, obesity and associated health problems place a significant economic burden on the Australian health care system. The total direct, indirect and social cost has been estimated at $37.7–$58.6 billion [6, 813] with direct costs estimated at $8–$21 billion [6, 814]. It is predicted that by 2023, the projected health expenditure for diabetes will have risen $1.4 billion to $7 billion per year, due mostly to increasing weight gain [11].

Intentional weight loss in overweight and obese individuals reduces the risk factors for mortality and morbidity, and alleviates the symptoms of many chronic conditions [811, 812]. It is not necessary to lose large amounts of weight to achieve substantial health gains. For example, a weight loss of 5kg in all people who are overweight or obese is estimated to reduce the national prevalence of type 2 diabetes by 15% [815]. Improving nutrition and/or increasing physical activity also benefits health in a number of areas beyond weight control, such as bone strength, mental health and immune function [2, 816].

There is a need to provide guidance for the population on promotion of healthy weight, primary prevention of overweight and obesity, weight maintenance, weight loss, and management of weight-related conditions, disorders and diseases. A stepped approach to obesity prevention and management is recommended (see Table 4.4) [710].

Table 4.4 A stepped model for the management of overweight and obesity

Target population	Intervention
General population	Population education, awareness raising and environmental interventions
Overweight/obese	Above + healthy lifestyle education and skills training
Overweight/obese with disordered eating or cognition	Above + behaviour modification
Overweight/obese with risk factors (BMI > 30 or BMI > 27 + risk factors)	Above + medical/surgical intervention (drugs, very low energy diets, surgery)

Given the scope of the *Guidelines*, the guidance here focuses on promotion of healthy weight through primary prevention of overweight and obesity, weight maintenance and achieving healthy weight (through weight loss) in overweight groups without serious co-morbidities. Clinical guidance for treatment and management of obesity and its complications for individuals is being reviewed under a separate process and will lead to the production of NHMRC's *Clinical Practice Guidelines for the Management of Overweight and Obesity* [710].

Prevention of overweight and obesity is important because:

- weight loss is difficult to achieve and even more difficult to maintain
- for most people who are classified as obese, and for many who are overweight, a return to a healthy-range BMI may not be a realistic target
- the health consequences of obesity tend to be cumulative and may not be fully reversible once classified as overweight
- it is more efficient and cost effective to prevent weight gain rather than treat overweight and obesity [88, 685, 688].

All available evidence suggests that the current epidemic of obesity is a normal response to an abnormal 'obesogenic' environment. The community as a whole has a social responsibility to address the problem. It is especially important that supportive social, economic and physical environments are created to make it easier for individuals, families, groups and communities to choose healthy dietary and physical activity patterns to achieve and maintain a healthy weight.

All initiatives and approaches which promote physical activity, healthy eating, access to nutritious food, and the healthy growth of children contribute to promoting healthy weight at the population level.

4.4.1 Achieving and maintaining a healthy weight

4.4.1.1 Physical activity (see Appendix 4)

Previously it was thought that if energy intake was controlled, 30 minutes of moderate-intensity daily physical activity would be sufficient to prevent weight gain in adults, providing sitting time was less than 4.5 hours/day [817-819]. However, in the current environment of abundant availability, promotion and consumption of energy-dense food, there is now international consensus that 45–60 minutes of moderate-intensity daily physical activity is the minimum required for the general population to prevent the transition to overweight and obesity without reduction in current energy intake [88, 820-823]. At least 60–90 minutes of moderate-intensity activity or lesser amounts of vigorous activity may be required to prevent weight regain in formerly obese people [820].

The current physical activity guidelines for Australian infants, children and adolescents may be adequate to support optimum growth and development and – together with consuming a nutritious diet and appropriate energy intake - weight control [36, 710]. This is supported by recent evidence from Europe [824].

4.4.1.2 Dietary patterns

Foundation Diets developed by the Modelling System [10] represent the basis of optimum diets to achieve and maintain a healthy weight as they provide nutrient requirements within minimum energy intake.

Compared to the most recent available adult intakes, *Foundation Diets* include higher quantities of vegetables, fruit, wholegrain cereals, poultry, fish, eggs and low-fat milk, yoghurt and cheese products, and lower quantities of starchy vegetables, refined cereals, higher and medium fat milk, yoghurt and cheese products and red meats (latter for men only) [10]. Decreased consumption of discretionary foods would be needed to achieve the dietary patterns within energy constraints. Changes from higher fat to lower fat milk, yoghurt and cheese products and from refined to wholegrain cereals would also be necessary [10].

To prevent excessive or inappropriate weight gain and therefore prevent development of overweight and obesity, the smallest, least active adults in each age and sex group should adopt dietary patterns consistent with the *Foundation Diets*. Additional foods and drinks can only be included without leading to weight gain if physical activity levels are increased [10].

Taller or larger and more active adults in each age and sex group can choose additional serves of foods from the food groups and/or occasionally some discretionary foods to increase energy intake to meet energy requirements (that is, to comprise *Total Diets*) [10], but they need to ensure energy intake does not exceed expenditure. If energy requirements are exceeded by energy intake on a regular basis, weight gain will occur.

There is wide variation in individual energy needs, so to prevent weight gain (or inappropriate weight loss) at an individual level, weight (and waist circumference) should be measured regularly (for example, every two or three months) and the amount and/or quality of dietary intake and physical activity levels adjusted accordingly [10].

As a basic principle when adjusting dietary patterns, the first steps are to choose nutritious foods from the five food groups in amounts consistent with *Foundation Diets* and to limit discretionary (energy-dense, nutrient-poor) choices. If further restrictions are required, rather than eliminating one category of food from the five food groups, smaller serves should be chosen [114].

Recommended quantities of specific food groups to suit those preferring an omnivorous, lacto-ovo vegetarian diet or a dietary pattern that favours pasta or rice are included in the *Foundation Diets* for adults set out in the Food Modelling System [10].

4.4.2 Weight loss in adults who are already overweight

These *Guidelines* do not encourage inappropriate food restriction. However, they do discourage consumption of energy-dense, nutrient-poor discretionary foods and drinks.

In dietary patterns to achieve a healthy weight and assist weight loss, the recommendations of types and quantities of foods outlined in the *Foundation Diets* in the *Australian Guide to Healthy Eating* should not be exceeded. Adhering to *Foundation Diets* only, without discretionary foods, could represent a daily reduction in energy intake of up to 2,000 kJ for the average size person in each age and sex group. This could result in a satisfactory rate of weight loss, even if physical activity levels are not increased.

A combination of increased physical activity and energy restriction is more effective than energy restriction alone for weight loss and maintenance of weight loss. Physical activity can affect 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 based on its frequency, intensity and duration.

Weight loss will not be achieved unless energy intake is less than total energy expenditure. Lifestyle improvements, through cognitive and behaviour change, to increase physical activity and improve dietary intake is fundamental to weight management. Such education, training and support may be provided to individuals or groups [710]. Weight loss is most likely to be maintained where

dietary and physical activity habits are acceptable and sustainable. In this regard, regular weight loss of initially around 1.0 to 4.0kg per month, reaching 10% loss of initial weight in the medium term and 10–20% loss of initial weight over 1–5 years is likely to be most effective and sustained [710, 825].

Individuals who are overweight or obese and have associated cardiovascular disease or diabetes risk factors should seek clinical advice about the range of available treatment options [826].

4.4.3 Pregnant and breastfeeding women

Obesity in pregnancy is one of the most common, and potentially modifiable, risk factors for adverse pregnancy outcomes [827] and longer term adverse outcomes for mothers [828] and children [829]. Since about one-third of pregnant women in Australia are overweight or obese [830], preventing excessive gestational weight gain is an urgent health priority.

Appropriate maternal weight gain during pregnancy (see table 4.5) is important for the health of the infant. Evidence supports recommendations to manage pregnancy to reduce the risk of excessive weight gain and gestational diabetes [831, 832]. Too little weight gain during pregnancy increases the risk of a low birthweight infant. Excessive weight gain during pregnancy increases the risk of macrosomia and gestational diabetes. Excessive gestational weight gain is also associated with increased risk of obesity and metabolic syndrome in infants later in life [833, 834].

Appropriate steady weight gain during pregnancy is important to optimise the health outcomes (short term and long term) for the infant and the mother [831, 832]. Weight gain should be steady throughout pregnancy to avoid adverse effects on specific foetal organ systems during critical periods (for example, neural tube 6-8 weeks, kidney development 28–30 weeks). Weight loss diets are not recommended at any time during pregnancy [832].

Mothers who gain excessive amounts of weight during pregnancy are unlikely to lose it later [835], although dietary patterns which comprise regular meals, plenty of fruit and vegetables, high-fibre bread and restricted sugar [836] may help mothers reach a healthy weight after giving birth.

Evidence of a probable association between babies born to mothers who smoke during pregnancy and a higher risk of overweight or obesity in adolescence and adulthood supports recommendations that pregnant women should not smoke cigarettes (Grade B, Section 17.5 in Evidence Report [14]) [52, 54, 59-61, 779].

Table 4.5 2010 Institute of Medicine recommendations for total and rate of weight gain during pregnancy, by pre-pregnancy BMI [832]

Pre-pregnancy BMI	Total weight gain in kg	Rates of weight gain* 2nd and 3rd trimester in kg/week
Underweight (< 18.5 kg/m^2)	12.5 – 18.0	0.51 (0.44 – 0.58)
Normal weight (18.5 – 24.9 kg/m^2)	11.5 – 16.0	0.42 (0.35 – 0.50)
Overweight (25.0 – 29.9 kg/m^2)	7.0 – 11.5	0.28 (0.23 – 0.33)
Obese (≥ 30.0 kg/m^2)	5.0 – 9.0	0.22 (0.17 – 0.27)

* Calculations assume a 0.5-2kg weight gain in the first trimester [832]

Table 4.6 Weight gain during pregnancy: recommendations for Asian women, by pre-pregnancy BMI *

Pre-pregnancy BMI	Total weight gain in kg (during pregnancy)	Weight gain per week in kg (after 12 weeks)
<18.5	12.5 – 18.0	0.5
18.5-22.9	11.5 – 16.0	0.4
23-27.5	7.0 – 11.5	0.3
>27.5	≤ 7.0	
Others		
Twin pregnancy	15.9 – 20.4	0.7
Triplet pregnancy	22.7	

* Adapted from IOM guidelines and matched with Asian BMI cut-offs

4.4.4 Infants, children and adolescents

Infants, children and adolescents need sufficient nutritious food to grow and develop normally. The focus should be on maintaining a rate of growth consistent with the norms for age, sex and stage of physiological maturity. Physical growth is best assessed by the conventional measures of weight, length or height, and head circumference (see Appendix 3). Maintenance of a positive energy balance and adequate nutrient intake is critical in achieving and sustaining normal growth and

development. During periods of rapid growth, intentional restriction of weight gain - through dieting, for example - is usually inappropriate.

In recent years there has been increasing awareness of the importance of perinatal nutrition in terms of the development of disease in adulthood, known as foetal origins of disease or Barker hypothesis [837]. There is also increasing evidence of the importance of growth and optimum nutrition in relation to cognitive development [838] and future bone mass [839].

4.4.4.1 Growth

Relative to their body weight, children's nutrient and energy requirements are greater than those of adults [9]. Children are nutritionally vulnerable up to around five years of age, after which their growth rate slows and their nutritional needs reduce relative to their body size. As a child's rate of growth is a fundamental indicator of nutritional status and health and wellbeing, parents and carers and health professionals must be responsive to the developmental and nutritional needs of children.

Between birth and 18 years of age, body weight increases about twenty-fold. During infancy and adolescence the rate of growth can change rapidly, while from 12 months of age the rate of increase in weight and length is essentially linear. Growth decelerates rapidly during the first year of life. During adolescence it accelerates over one to three years and then decelerates rapidly until growth in height ceases at about 16 years in girls and 18 years in boys.

Childhood is a period of education about eating and good nutrition, so appropriate use of food is important in establishing lifetime nutrition practices. Food intake may drop off during the second year of life, when parents' skills of encouragement and example will be needed. After starting school, children are subject to an increasing array of influences from outside the home, particularly peer pressure which peaks in adolescence.

4.4.4.2 Dietary patterns for achieving and maintaining a healthy weight in infants, children and adolescents

Foundation Diets represent the basis of optimum diets for infants, children and adolescents. However sufficient nutritious foods must be provided to support optimum growth and development in all children.

For the youngest, shortest or least active in each age and sex group, dietary modelling suggests that there is no opportunity for additional energy intake beyond *Foundation Diets* unless increased physical activity increases energy expenditure and requirement. For these children *Foundation Diets* are equivalent to *Total Diets*.

For older, taller or more active children in each age and sex group, additional serves of foods from the five food groups (preferably) and/or discretionary food choices may be made to increase energy intake until energy requirements are met [10]. Growth and weight of children should be checked regularly (as outlined in Appendix 3) and the amount and/or quality of diet and physical activity levels adjusted accordingly [10].

Compared to current intakes, *Foundation Diets* for infants, children and adolescents include higher quantities of vegetables, fruit, wholegrain cereals, poultry, fish, eggs and low-fat milk, yoghurt and cheese products, and include lower quantities of starchy vegetables, refined cereals, higher and medium fat milk, yoghurt and cheese products. For children, as for adults, decreased consumption of discretionary food choices is needed to achieve the dietary patterns in the *Australian Guide to Healthy Eating*, together with a change from refined grain (cereal) foods to wholegrain cereals and from higher fat to lower fat milk, yoghurt and cheese products for children over 2 years of age.

Recommended quantities of specific food groups to suit those preferring an omnivorous, lacto-ovo vegetarian diet or a dietary pattern that favours rice or pasta are included in the *Foundation Diets* for infants, children and adolescents set out in the Food Modelling document [10].

4.4.4.3 Dietary patterns for weight loss in children and adolescents who are already overweight and obese

Management of overweight and obesity in childhood is recommended to reduce risk of being overweight in later life (see evidence above). Individual assessment and clinical supervision is recommended to ensure appropriate growth and development, for weight management of all overweight and obese infants, children and adolescents.

Parental overweight or obesity is associated with increased risk of overweight or obesity in children, with even greater risk when both parents are overweight or obese. A family-focused approach to weight management has been found to be beneficial [795-798].

Dietary restriction beyond prudent adherence to the *Foundation Diets* and limited intake of discretionary foods and drinks is not recommended for infants, children or adolescents, as this may result in nutrient deficiencies and sub-optimal growth and development. Adherence to *Foundation Diets* should assist maintenance of body weight during growth for overweight children - with the aim to maintain weight while the child grows in height thus 'normalising' BMI for age.

To help achieve a healthy weight, most Australian infants, children and adolescents should also follow the recommendations of the Australian physical activity guidelines (see Appendix 4).

4.4.5 Older people

Older people should eat nutritious foods and keep physically active to help maintain muscle strength and a healthy weight**.**

Daily energy expenditure declines throughout adult life, as does physical activity [44]. Energy expenditure is dependent on fat-free mass, which decreases by about 15% between the third and eighth decades of life, contributing to lowered metabolic rate in older people [840]. The decrease in energy expenditure is generally accompanied by decreased appetite and diminished food intake, so may account for the undernutrition seen in some older people [841]. While overweight and obesity are still prevalent in older adults [842], consideration of overall morbidity and mortality suggests that for obese older people a substantial reduction of the BMI may not provide the healthiest long-term option [843]. Although weight loss achieved by following a nutrient-dense diet and increasing physical activity may confer benefits, this is still to be tested in good quality trials [842]. Lowering blood pressure and normalising blood lipids rather than reducing weight may be more appropriate for elderly overweight people [844].

Older people commonly have a decrease in skeletal muscle mass and strength, which is the result of a decline in the production of muscle tissue [844]. Height may also decrease with age as a result of changing spinal shape and intervertebral thickness, making it difficult to determine height and therefore BMI.

While most of the elderly population live independently, it has been estimated that 25–40% of those over 80 years of age could be considered frail [845]. Malnutrition in the elderly is often associated with one or more illnesses such as chronic obstructive lung disease and heart failure, dementia, dysphagia, poor dentition, depression, social isolation, use of drugs, alcohol and other substance abuse, poverty, and despair [845, 846]. In Australia 5–11% of people eligible for Home and Community Care services are malnourished [847]. In acute care, 20–30% of people are admitted with malnutrition, the prevalence increasing with age and the number of health problems [847, 848]. Older people can develop sarcopenia, a form of muscle wasting, and some older people also experience sarcopenic obesity, where there is a combination of reduced muscle mass and/or strength and excessive body fat [842].

Recent studies suggest that, for an older person, being underweight may be more deleterious for health than being overweight [716]. A BMI range of 23–28 is considered desirable for people over 70 years of age [709].

The decline in energy expenditure with ageing must be balanced by adjusting energy intake to maintain body weight within the healthy range and to prevent an increase in body fat [9]. Dietary patterns and quantities consistent with the *Foundation* and *Total Diets* are for older people who are generally fit and well [10]. However low-fat diets are not appropriate for the frail elderly or those with complex health conditions [35, 844].

Most older people will benefit from increased physical activity [725, 849] including reduced sedentary behaviour, increased moderate-intensity aerobic activity [850]and particularly activities promoting bone and muscle strength, flexibility and balance [851]. In addition to assisting with weight management, these can help reduce the risk of falls [851, 852] and may increase opportunities for social engagement [853].

4.4.6 Aboriginal and Torres Strait Islanders

The last national survey measuring height and weight in Aboriginal and Torres Strait Islanders was conducted in 1994 [854]. Although recent measured data are lacking, prevalence of overweight and obesity calculated from self-reported height and weight data (at around 60%) is higher among Aboriginal and Torres Strait Islander people than self-reported data from non-Indigenous Australians [855-857]. Among Aboriginal and Torres Strait Islander groups living in rural and remote areas, disparities in the cost of nutritious foods are potential barriers to the adoption of nutrient-dense, low energy-dense diets [138, 140, 686, 728, 806, 858, 859].

4.4.7 Australians of Asian origin

There is little evidence that Australians of Asian origin are at increased risk of overweight, although special consideration might need to be given to this group in assessing body fatness. The WHO levels of BMI that correspond to increasing degrees of risk of chronic morbidity and of mortality were primarily derived for populations of European origin [708] so may not apply to Australians of Asian origin.

Australians of Asian origin have a higher proportion of body fat for the same BMI than Caucasians. This suggests that the application of the current WHO BMI cut-off points may underestimate body fatness and comorbidity risk in this population [708].

4.4.8 People with eating disorders

When promoting healthy weight, optimum nutrition and physical activity, it is essential to avoid inadvertently encouraging disturbed body image and disordered eating or exercise behaviour [722]. Characteristics of disordered eating, such as restrained eating, binge eating, fear of fatness, purging and distorted body image, are commonly reported in adolescents, particularly in early adolescence and late teens, but eating disorders may occur at any age [722]. Estimates of lifetime prevalence of anorexia nervosa, bulimia nervosa, and binge eating among women range from between 0.3-1.5%, 0.9-2.1% and 2.5-4.5% respectively, with estimated rates among men considerably lower[722]. People with suspected eating disorders need to be referred for specialist assistance from a health professional.

Effective interventions to reduce the risk of eating disorders include:

- promotion of nutritious dietary patterns rather than negative focus on specific foods
- avoiding stigmatisation of those of various body shapes and weight
- promotion of media literacy, such as critical evaluation of presented body ideals and promotion of good mental health [722].

Where to next

The next chapter provides information on why breastfeeding is beneficial to the health of the infant and the mother, and practical considerations for encouraging and supporting breastfeeding.

5. Encourage and support breastfeeding

Guideline 4
Encourage and support breastfeeding.

Executive summary

The World Health Organization states that "breastfeeding is an unequalled way of providing ideal food for the healthy growth and development of infants".

Breastmilk contains many unique compounds, including live cells, which provide all the nutritional requirements to support growth and development of infants to around six months of age.

Breastfeeding provides health benefits to infants including reduced risk of infection, asthma and atopic disease and Sudden Infant Death Syndrome. It contributes to improved cognitive development while protecting against obesity, hypertension and some chronic diseases in later life.

Benefits to mothers from breastfeeding include improved bonding with their infant, accelerated recovery from childbirth and progress to a healthy body weight. Breastfeeding is also associated with reduced risk of some cancers and of post-menopausal hip fracture in mothers.

As many infants as possible should be exclusively breastfed until around six months of age when solid foods are introduced. Breastfeeding should be continued until 12 months of age and beyond, for as long as the mother and child desire.

Breastfeeding prevalence, including initiation rates and duration, are improved where the mother has support and encouragement from the infant's father, other family members, the health system and the community.

This chapter provides information on why breastfeeding is beneficial to the health of the infant and the mother, plus practical considerations for encouraging and supporting breastfeeding.

5.1 Setting the scene

The World Health Organization states that "breastfeeding is an unequalled way of providing ideal food for the healthy growth and development of infants". Breastmilk is a living tissue that includes many unique compounds. Breastfeeding has short-term and long-term health and other benefits for infants and mothers. Maximising the benefits of breastfeeding to the infant and mother requires the support of the other family members and a supportive community environment.

Australia has a long history of promoting and supporting breastfeeding in its public health policy. In 1981 Australia became a signatory to WHO's *International Code of Marketing of Breast-milk Substitutes* [860], the stated aim of which was to protect and promote breastfeeding. The importance of breastfeeding led to its inclusion in the *Australian Dietary Guidelines* endorsed by the NHMRC in 1983 [568].

In Australia, part of the WHO code is implemented by the *Marketing in Australia of Infant Formulas (MAIF) Agreement,* a voluntary agreement by infant formula manufacturers and importers. The *Infant Feeding Guidelines for Health Workers* [133] provide more information on the WHO code, the MAIF Agreement and the obligations of all health workers. They also provide recommendations on the appropriate foods for infants from birth to about two years of age, including detailed information on the benefits of breastfeeding for the infant and mother.

Breastmilk contains many unique compounds, including antibodies and immune cells. In the first few days after giving birth a mother's breasts produce colostrum which provides all the nutrients and water required by their newborn infant. Colostrum contains higher levels of protein, vitamin A and vitamin B_{12} and less fat than breastmilk. It also contains lactoferrin, immunoglobulin A, enzymes, maternal antibodies, living cells (leukocytes, neutrophils and macrophages) and prebiotics, which limit the growth of pathogenic bacteria and viruses, stimulate the growth of an appropriate human microbiome and protect against illness [483, 861, 862]. Colostrum feeding is important for the infant and also stimulates the mother's breastmilk production. The composition of colostrum gradually changes as lactation is established and milk production begins 48–72 hours after birth.

The nutrient composition of mature human milk varies between individuals and across the stages of lactation. The energy content varies between 270 and 315 kJ per 100 mL, largely due to variation in the fat content [863, 864]. Fat content typically increases through each breastfeed. It provides much of the energy and omega-3 and omega-6 long-chain polyunsaturated fatty acids, plus the fat-soluble vitamins A, D, E and K, as well as prostaglandins. The fat in breastmilk is typically better absorbed by an infant's gastrointestinal tract than the fat in cow's milk. Mature milk continues to provide immune factors and enzymes to the infant [36, 865].

Breastmilk provides all the vitamins, major minerals and trace elements known to be essential for healthy full-term infants for around the first six months [731, 866]. These are more bioavailable

than nutrients found in infant formula. Because the composition of breastmilk constantly changes throughout lactation and during a single breastfeed, no infant formula can exactly mimic the composition of breastmilk. Breastmilk is a convenient, hygienic and inexpensive food source posing no environmental costs [867].

Breastmilk continues to be an important source of vitamins, minerals and trace elements when other foods are introduced at around six months (complementary feeding).

Consuming a variety of nutritious foods is particularly important for breastfeeding women.

5.2 The evidence for *'Encouraging and supporting breastfeeding'*

The evidence statements and gradings (A- convincing association, B- probable association, C- suggestive association) from the Evidence Report (literature from 2002 – 2009) are presented in the table below. This does not include evidence from other sources, such as the 2003 Dietary Guidelines (where evidence was classified as level I, II or III), although these sources have been used to inform the *Guidelines*.

Evidence Statement	Grade
Compared to infants who are formula fed, being breastfed is associated with reduced risk of becoming obese in childhood, adolescence, and early adulthood.	A
Pre-natal and perinatal support for breastfeeding can increase the proportion of women breastfeeding (both exclusive and non-exclusive) up to age six months.	A
Being breastfed in infancy is associated with lower systolic and diastolic blood pressure up to adolescence.	B
Infants who are exclusively breastfed for six months experience less morbidity from gastrointestinal infection than those who are mixed breastfed as of three or four months.	B
Infants, from either developing or developed countries, who are exclusively breastfed for six months or longer do not have deficits in growth compared to those who are not exclusively breastfed.	B
There are no apparent risks in a general recommendation for exclusive breastfeeding for the first six months of life, in both developing and developed-countries. However, infants should still be managed individually in order to achieve sufficient growth and minimise adverse outcomes.	B

Evidence Statement	Grade
Exclusive breastfeeding for 6 months or more prolongs lactational amenorrhea for mothers.	B
Breastfeeding support (any type) increases duration of both exclusive and non-exclusive breastfeeding both in the immediate post-natal period and at six months of age.	B
Being breastfed initially, particularly exclusively breastfed is associated with lower total and LDL concentrations in adult life.	C
Breastfeeding is associated with a reduced risk of asthma and atopic disease.	C
Breastfeeding is associated with a reduced risk of atopic disease.	C
Not breastfeeding is associated with an increased risk of Sudden Infant Death Syndrome.	C
Maternal perceived insufficient milk (PIM) supply is associated with increased risk of early cessation of lactation.	C

5.2.1 Breastfeeding incidence and duration

Exclusive breastfeeding: Systematic reviews from developed and developing countries provide evidence of a probable association that exclusive breastfeeding for around six months of life[3] is the best method of feeding for full-term infants (Grade B, Section 23.2 in Evidence Report [14]). Breastfeeding can then continue while appropriate solid (spoon) foods are introduced.WHO and almost all national and international paediatric and public health organisations make similar recommendations. NHMRC's Infant Feeding Guidelines for Health Workers (public consultation draft 2011) recommend that exclusive breastfeeding be encouraged, supported and promoted to around six months of age. —see *Infant Feeding Guidelines for Health Workers – Literature Review* for a full list [868-871].

Adding other foods: The introduction of complementary feeding (adding solid foods and liquids other than breastmilk or infant formula) at around six months is consistent with introduction of solid foods during the probable 'window of tolerance' between 4–7 months [872]. The majority of Australian infants have solids introduced during this period [873].

[3] In Australia, 'around six months' for exclusive breastfeeding is used to acknowledge that different infants develop at different rates. 22 – 26 weeks is considered to be "around six months".

No apparent risks have been reported in recommending, as a general policy, exclusive breastfeeding for around the first six months of life, in both developing and developed countries (Grade B, Section 23.2 in Evidence Report [14]). It is important, however, that health professionals manage infants individually so that faltering growth or other adverse outcomes do not go unnoticed.

Early cessation of lactation: Recent evidence suggests an association between maternal perceived insufficient milk (PIM) supply and an increased risk of early cessation of lactation (Grade C, Section 25.3 in Evidence Report [14]).

Supportive environments: There is convincing evidence that pre-natal and perinatal support for breastfeeding can increase the proportion of women breastfeeding (both exclusive and non-exclusive) up to age 6 months (Grade A, Section 25.3 in Evidence Report [14]).

Recent evidence also suggests a probable association between breastfeeding support (any type) and an increased duration of both exclusive and non-exclusive breastfeeding, both in the Immediate post-natal period and at 6 months of age (Grade B, Section 25.3 in Evidence Report [14]).

Table 5.1 Factors associated with duration of exclusive breastfeeding

Association with longer duration of exclusive breastfeeding		
Consistently positive	**Consistently negative**	**Effect varies in different cultures**
Higher level of maternal education	Lower socioeconomic backgrounds	Maternal age
Support of midwives and community health professionals	Formula supplementation in the maternity ward	Vaginal delivery
Father's preference for breastfeeding	Mother's intention to use supplementation	Multiparity
Doctors' support of breastfeeding	Previous breastfeeding experience short-term (<5 weeks) or absent	
Pleasant birth experience	Newborn infant not rooming in with mother	
Greater breastfeeding knowledge	Early use of pacifier	
Rural environment	High number of intended hours of work per week after maternity leave	
Time decision to breastfeed is made (preferably before pregnancy or early in pregnancy)	Perceived insufficient milk supply and other breastfeeding problems	
	Smoking, alcohol use	
	Intention to return to work early (before six months)	

Source: Summarised from Kramer et al. 2002 (1); Britton et al. 2007; Chung et al. 2008; Hector & King 2005; Landers, Hughes & Graham 1998; Scott & Binns 1999; Scott 1999; Scott 2001; Simopoulos, Oliveria & Desai 1995; Kramer et al. 2002 (2) [866, 874-882].

5.2.2 Infant growth

For ethical reasons, randomised control trials (RCTs) of breastfeeding are not possible. The best research alternative is the use of RCTs of health promotion interventions to increase breastfeeding rates. For this reason, most evidence is based on prospective cohort studies and one large health promotion RCT, the Promotion of Breastfeeding Intervention Trial (PROBIT) study [883].

Breastfed infants grow more slowly than formula-fed infants [861]. A systematic review of 19 observational studies in developed countries concluded that the cumulative difference in body weight at 12 months of age was 600–650g less in infants breastfed for 12 months than formula-fed infants [884]. Differences in feeding behavior and mother–child interaction between breastfed and formula-fed infants may account for some of the differences reported. For instance, breastfed infants showed a different suckling pattern, and appeared to have greater degree of control on meal sizes and feeding intervals than infants who were formula-fed [885].

5.2.3 Cardiovascular disease, type 2 diabetes and excess weight

Excess weight: There is convincing evidence that breastfeeding , compared with formula feeding, is associated with reduced risk of infants becoming obese in childhood, adolescence and early adulthood (Grade A, Section 17.2 in Evidence Report [14]). The protection offered by breastfeeding appears to increase with duration of breastfeeding until it plateaus at nine months [886, 887].

Evidence of an association between the age of introduction of solid foods and excess weight in children is inconclusive (Section 19.1 in Evidence Report [14]).

Blood pressure: There is probable evidence that infants who are breastfed exclusively in the first few months of life have a lower adult systolic and diastolic blood pressure (approximately 1.5/0.5 mmHg) compared with those who are formula-fed (Grade B, Section 23.1 in Evidence Report [14]).

Total and LDL cholesterol: Recent evidence suggests being breastfed initially, and particularly exclusively breastfed, is associated with lower total and LDL concentrations in adult life when compared with those who were formula-fed (Grade C, Section 23.1 in Evidence Report [14]).

5.2.4 Other benefits

Sudden Infant Death Syndrome (SIDS): There is evidence to suggest that breastfeeding reduces the risk of SIDS (Grade C, Section 23.4 in Evidence Report [14]).

Gastrointestinal infection: There is probable evidence that children who are exclusively breastfed for six months have lower morbidity from gastrointestinal infection than those who are given both breastmilk and formula at the age of 3-4 months (Grade B, Section 23.2 in Evidence Report [14]). Factors in breastmilk such as secretory IgA, oligosaccharides and lactoferrin may protect the infant from various infections through passive immunity [483].

Asthma: Recent evidence suggests an association between breastfeeding and lower incidences of asthma (Grade C, Section 23.3 in Evidence Report [14]). Data from the *Longitudinal Study of Australian Children* suggest a strong and significant protective effect of breastfeeding on wheezing and asthma in infancy, which increases with increasing breastfeeding duration (see http://www.asthmamonitoring.org).

Atopic dermatitis: Recent evidence also suggests an association between breastfeeding and protection against atopic dermatitis in children with a family history of this condition, but not in those without a family history (Grade C, Section 23.3 in Evidence Report [14]).

Lactational amenorrhea: Exclusive breastfeeding for six months or more prolongs lactational amenorrhea for mothers and delays the return of fertility (Grade B, Section 23.2 in Evidence Report [14]), which may be a benefit for some women as the lactational amenorrhoea method is a commonly used form of contraception [888-890].

Allergy: The evidence for an association between a delay in the introduction of solid foods until after the age of 6 months and risk of developing allergic syndromes is inconclusive (Section 19.2 in Evidence Report [14]).

Other factors: Additional benefits for infants who are exclusively breastfed compared with those who are formula fed in both developed and developing countries [861, 891] may include:

- protection against respiratory infection and reduced prevalence of asthma [892]
- reduced occurrence and recurrence of otitis media [891]
- protection against neonatal necrotising enterocolitits, bacteraemia, meningitis, botulism and urinary tract infection [861, 893]
- reduced risk of autoimmune disease, such as type 1 diabetes [891]
- lower rates of coeliac disease, Crohn's disease and ulcerative colitis [894]
- improved visual acuity and psychomotor development [867]
- higher IQ scores, which may be the result of factors present in the milk or of greater stimulation [867, 895]
- reduced malocclusion as a result of better jaw shape and development [896]

- improved mother and infant bonding and attachment [897].

Additional health benefits to women who breastfeed may include:

- promotion of maternal recovery from childbirth through accelerated uterine involution and reduced risk of haemorrhage [898]
- accelerated weight loss and return to pre-pregnancy body weight [866, 882]
- reduced risk of pre-menopausal breast cancer [891]
- reduced risk of ovarian cancer [891]
- improved bone mineralisation and thereby decreased risk of post-menopausal hip fracture [899, 900].

5.3 Practical considerations: *Encourage and support breastfeeding*

Further information is included in the *Infant Feeding Guidelines for Health Workers [133].*

5.3.1 Breastfeeding initiation and duration

It is estimated that in Australia 90–92% of mothers initiate breastfeeding and approximately 50% are still breastfeeding at six months. Few infants are exclusively breastfed until six months in Australia [901]. However reliable national data on breastfeeding rates is difficult to obtain for several reasons, including inconsistent use of definitions of breastfeeding [902] [864].

5.3.1.1 Early interaction

Mothers should have contact with their babies as soon after birth and for as long as they wish [874]. Interventions aimed at either delaying or speeding up the length of the first feed should be avoided. Hospitals can encourage 'rooming-in' to facilitate frequent mother and child contact [903].

5.3.1.2 Adolescent mothers

Breastfeeding initiation and duration rates are below recommended levels among adolescent mothers [14]. Specific breastfeeding education programs in USA, UK and Australia targeted at pregnant adolescents have been somewhat effective in increasing breastfeeding initiation in adolescent mothers [904, 905]. Adolescent mothers identify emotional and network support as well as self-esteem as being crucial to breastfeeding success [904].

5.3.1.3 Mothers in the workplace

Evidence is emerging that a mother's employment status and number of hours worked outside the home influences the initiation and duration of breastfeeding. Women who are not employed full-time [906], are self-employed or have flexible working hours are more likely to breastfeed for six months. Using only parental childcare has a positive association with continuation of breastfeeding [907].

5.3.1.4 Low socioeconomic status mothers

Women from the lowest socioeconomic quintile in Australia have lower breastfeeding rates than those from the most affluent quintile [908-910].

5.3.1.5 Culturally and linguistically diverse mothers

Limited available data suggest that, in general, the rates of breastfeeding among CALD women in Australia reflect trends in their countries of origin [911-915].

5.3.1.6 Aboriginal and Torres Strait Islander mothers

Indigenous mothers tend to breastfeed for longer than non-Indigenous mothers, especially in rural areas [30]. In the Perth Aboriginal Breastfeeding Study, Aboriginal Australians had higher breastfeeding rates than non-Aboriginal women [916].

5.3.1.7 Mothers who use illicit drugs

WHO recommends that mothers who use illicit drugs while breastfeeding should be evaluated on an individual basis. Breastfeeding may need to be discontinued [917], but each case needs detailed medical assessment [906].

5.3.2 Support for breastfeeding

5.3.2.1 Promoting breastfeeding in prospective parents

Overall, reviews of interventions to support breastfeeding have found that education before birth and continuing support after birth for breastfeeding mothers were effective in breastfeeding continuation.

The US Preventive Services Task Force found that any intervention to promote breastfeeding significantly increased rates of exclusive breastfeeding in the short term [875].

Effective education programs include information about the benefits of breastfeeding, principles of lactation, myths, common problems and solutions, and skills training [918]. Peer support was particularly useful for socioeconomically disadvantaged women, and peer counsellors were most effective if they were of similar cultural and social status to the women they were counselling. The optimal mix of interventions to improve breastfeeding practices includes education of mothers, peer support, hospital practices such as 'rooming-in' and 'early skin-to-skin contact', staff training, development and implementation of hospital policy, media campaigns, and paid maternity leave [876].

A more recent Cochrane review agrees that there is a protective effect of provision of support on increasing duration of breastfeeding [874].

5.3.2.2 Promoting breastfeeding in hospitals

The Baby Friendly Hospital Initiative (UNICEF and WHO) has been shown to increase breastfeeding rates in accredited hospitals [919]. The steps include:

- Have a written breastfeeding policy that is routinely communicated to all health care staff
- Train all health care staff in skills necessary to implement this policy
- Inform all pregnant women about the benefits and management of breastfeeding
- Place babies in skin-to-skin contact with their mothers immediately following birth for at least an hour and encourage mothers to recognise when their babies are ready to breastfeed, offering help if needed.
- Show mothers how to breastfeed and how to maintain lactation even if they should be separated from their infants
- Give newborn infants no food or drink other than breastmilk, unless medically indicated
- Practice rooming-in, allow mothers and infants to remain together-24 hours a day
- Encourage breastfeeding on demand
- Give no artificial teats or dummies to breastfeeding infants
- Foster the establishment of breastfeeding support and refer mothers on discharge from the facility.

Early contact improves breastfeeding outcomes [14]. Interventions aimed at either delaying or speeding up the length of the first feed should be avoided. Hospital practices at the time of birth can be the first line of support for a new mother. Difficulties encountered can be quickly resolved by staff with appropriate experience, and hospitals can encourage 'rooming-in' to facilitate frequent mother and child contact [903]. The use of prelacteal feeds or other liquids while in hospital interferes with the establishment of lactation and is contrary to the BFHI principles.

Mothers should have contact with their babies as soon after birth and for as long as they wish as this improves breastfeeding outcomes [874].

5.3.1.3 Community support

The successful management of breastfeeding problems in the first weeks after birth has a major impact on breastfeeding duration [920]. Sources of support for mothers in the first few weeks include family members, community health nurses, voluntary organisations and general practitioners. Preparation and education before birth are very important in achieving successful breastfeeding. Efforts to improve community acceptance of breastfeeding are also critical.

5.3.3 Safe storage of breastmilk

Many mothers find it convenient to express breastmilk so that others can feed their baby if required. Appropriate hygiene is essential for the expression and storage of breastmilk.

When expressing breastmilk, mothers should:

- wash hands thoroughly with soap and water
- if using a breastpump, sterilise it according to the manufacturer's instructions and ensure the storage bottle is sterile
- refrigerate or freeze milk immediately after expressing
- discard any breastmilk that has been thawed but not used
- freeze milk that will not be used within two days. NHMRC draft *Infant Feeding Guidelines for Health Workers(2011)* refer to a maximum refrigerated storage time of 96 hours.
- date containers at the time of collection and use the oldest milk first.

The use of a breast pump to express and measure breastmilk production is not recommended as a way to assess the adequacy of breastfeeding because expression from the breast may not be as effective as an infant suckling. Serial measurements of growth are the best way of assessing nutritional adequacy.

5.3.4 Alcohol and breastfeeding

See Section 3.4.

Where to next

Everyone should prepare and store food safely; however particular care should be taken when handling food intended for consumption by those who have increased risk of foodborne illness, such as pregnant women, infants, older people and people with certain medical conditions. The next chapter discusses how incorrect handling of food or storing of food can contribute to food poisoning and poor health outcomes.

6. Food safety

Guideline 5
Care for your food; prepare and store it safely.

Executive summary

More than five million cases of foodborne illness are estimated to occur every year in Australia.

Bacterial and viral food poisoning is a result of pathogenic organisms reaching harmful levels or the production of pathogenic toxins.

Incorrect handling of food and storing food at inappropriate temperatures are major causes of food poisoning. Particular care should be taken when handling food to be consumed by people who have an increased risk of foodborne illness, such as pregnant women, infants, older people and people with certain medical conditions.

This chapter provides information on the prevention of foodborne illness and why it is important to store and prepare food safely.

6.1 Setting the scene

Foodborne illnesses are caused by contaminated foods. Contaminants include pathogens, environmental contaminants and adulterants [921]. These guidelines focus on bacterial and viral contaminants.

Bacterial and viral food poisoning generally occurs when pathogenic organisms multiply to harmful levels as a result of incorrect handling of food, particularly when temperature control is inadequate. Some foodborne pathogens can cause illness even when present in low numbers (for example, hepatitis A virus, Norovirus, some strains of *E. coli*, *Campylobacter jejuni* and *shigella spp.*). Other pathogens produce toxins when allowed to multiply to high levels in food (for example, *Clostridium botulinum*, *Staphylococcus aureus*, *Bacillus cereus* and *shigella* spp) [922].

Prevention of contamination is the key to avoiding foodborne illness. Once pathogens contaminate food they can multiply and/or produce toxins if not handled correctly [923]. As heat can kill many bacteria and viruses it is the basis for many strategies for making food safe. However, even reheating food to high temperatures will not destroy all toxins.

The increasing age of human populations, migration, mass production of food due to population growth, and changed food habits pose threats to food safety [924]. Lack of access to quality food, lack of refrigeration and suitable storage pose threats to vulnerable groups. Isolated and poorer communities can be at higher risk as a result of inadequate storage facilities or limited access to regular food supplies.

Foodborne illnesses appear to be increasing in incidence in Australia and worldwide, posing a significant public health problem [925, 926]. The most recent estimates are that there are 5.4 million cases per year of foodborne illness in Australia, leading to 1.2 million visits to medical practitioners and 2.1 million days of work lost each year [927]. Multiple episodes increase the risk of long-term consequences such as reactive arthritis, irritable bowel syndrome and, rarely, Guillain-Barre Syndrome.

6.2 The evidence for *'caring for your food; prepare and store it safely'*

The true incidence of foodborne illnesses is consistently underestimated because cases are underreported, most cases are sporadic, and full diagnostic testing is usually done only in more severe cases or when there are extensive common-source outbreaks [928-930]. The majority of reported episodes are caused by foods prepared outside the home, regardless of where they are consumed. Foods prepared in the home account for 20–40% of foodborne illnesses in Australia

[929]. Fresh fruit and vegetables can also be contaminated, depending on soils and farming practices. The main causes of foodborne illness in Australia are:

- inadequate cooking [928, 931]
- improper holding temperatures [928, 931]
- contaminated equipment (such as knives, cutting boards and dishcloths) [931]
- contaminated food storage and preparation areas
- unsafe raw food [931]
- allowing raw foods to make direct contact with ready-to-eat foods [928]
- poor personal hygiene of food handlers (such as not washing hands adequately, particularly after handling raw food and immediately after using the toilet) [931]

6.2.1 Foods which may cause problems if not handled correctly

The following are examples of foods that are normally considered potentially hazardous if not stored and prepared safely:

- raw and cooked meat or foods containing raw or cooked meat
- dairy products and foods containing dairy products
- seafood and foods containing seafood
- cooked rice and pasta
- processed fruit and vegetables such as salads
- processed foods containing eggs or other protein-rich food
- foods that contain any of the foods above, for example, sandwiches [932].

The foods most commonly implicated in foodborne illness in Australia are meat and seafood [928]. Some foodborne pathogens, such as viruses and enterohaemorrhagic strains of *E. coli*, do not need to grow in foods to produce illness. Contamination of any ready-to-eat food with such a pathogen can result in foodborne illness.

6.3 Why it is important *to prepare and store food safely*

The ability of micro-organisms to grow in a food depends on external factors (such as temperature) as well as characteristics of the food itself, such as protein content [933, 934], water content and pH. For example, bacteria are least active in very acidic foods (pH less than 4.5).

Most bacteria can multiply at temperatures between 5°C and 60°C but a few pathogenic bacteria multiply at temperatures at or below 5°C [932]. Exposure to high temperatures destroys the

vegetative cells of most bacteria, although some have heat-resistant spores or produce histamines and toxins that survive the cooking process [923].

Some bacteria multiply within 2 hours and can reach an infective dose in 4–6 hours [933-935]. Refrigeration at or below 5°C slows the growth of bacteria [923].

6.4 Practical considerations: *Food safety*

6.4.1 Pregnant and breastfeeding women

As the immune system in pregnancy is suppressed, pregnant women are more susceptible to foodborne illnesses. Pregnant women have a higher risk of listeriosis, caused by the bacterium *Listeria monocytogenes*. Listeriosis can be transmitted to the unborn child and possibly cause a miscarriage, premature birth or stillbirth [936]. As a precaution, pregnant women are advised to avoid specific foods which are more likely to contain *Listeria*. Pregnant women are also advised to be careful with foods more likely to contain mercury [131].

6.4.2 Infants

The immune system of infants is not fully developed and so they are more susceptible to foodborne illnesses. Particular care should therefore be taken when preparing infant formula (if this is being used), including the sterilisation of bottles and dummies and other equipment used to prepare formula [931]. Prepared foods should be handled with care and not reused or reheated, as outlined in the *Infant Feeding Guidelines for Health Workers* [133].

6.4.3 Adults with illness

Adults with illnesses that reduce immune function such as HIV/AIDS, cancer, diabetes, kidney or liver disease, haemochromatosis, stomach problems (including previous stomach surgery) and low stomach acid (for example, from antacid use) are more susceptible to foodborne illness. Also at risk are people taking immunosuppressants, undergoing bone marrow or stem cell transplantation, or who have a history of long-term steroid use. Prepared foods should be handled with care.

6.4.4 Older people

Older adults are at higher risk of foodborne illness than other adult groups due to their weakened immune systems, decreased intestinal motility, decreased acid in the stomach, loss of sense of smell and taste, and dementia and malnutrition especially in frail older people [937, 938]. High-risk foods should be cooked, dairy products should be pasteurised, and specific foods at risk of listeria

contamination (see above) avoided. See http://www.foodstandards.gov.au/consumerinformation/listeria/.

People of any age, but especially older people receiving pre-prepared meals, need to ensure that they are safely stored if not consumed immediately.

Appendix 1. History and timeline of Australian nutrition documents

The Commonwealth Advisory Council on Nutrition was formed in 1936 and became the Nutrition Committee of the NHMRC in 1939, at the same time as a Nutrition Unit was established in the then Commonwealth Department of Health. Soon after, state and territory health departments set up similar nutrition committees and have since had a role in providing nutrition information and education programs in Australia.

From the 1940s through to the 1970s the Commonwealth and the states and territories published pamphlets and booklets to guide food selection and provide education tools, including the Five Food Group plan, which listed the food groups 'bread and other cereals', 'vegetables and fruit', 'meat and meat alternatives', 'milk and milk products', and 'butter/table margarine'.

1938 *Tables of composition of Australian foods*

First set of tables of composition of Australian foods, including data from both local and overseas sources for over 1100 foods. It was revised and reprinted many times over the next decades.

1979-83 *Dietary Guidelines for Australians*

In response to an increase in nutritional problems related to overconsumption of food, in 1979 the Department of Health developed eight Dietary Goals for Australians. These were modified to provide direct advice to members of the community as *Dietary Guidelines for Australians*, which were endorsed by the NHMRC in 1983. It was noted that these guidelines should be reviewed as further data became available on the nutritional status of Australian and the relationships between diet and disease [939].

1989 *Nutrient Data Table (NUTTAB)*

The first electronic food composition data release (on diskette), as the first edition of the Nutrient Data Table for use in Australia (NUTTAB), containing a combination of Australian and British data [940].

1989 - 1995 *Composition of Foods, Australia (COFA)*

The Composition of Foods, Australia (COFA) series was released in seven volumes, containing the first compilation of new Australian-sourced data for Australian foods from the analytical work of Greenfield and colleagues.

1991 *Recommended Dietary Intakes for use in Australia*

The development of the *Recommended Dietary Intakes* (RDIs) began in 1980 and the report was published in 1991 [941]. The RDIs are derived from estimates of requirements for each age/sex category and incorporate generous factors to accommodate variations in absorption and metabolism. They therefore apply to group needs. RDIs exceed the actual nutrient requirements of practically all healthy persons and are not synonymous with requirements.

1992 *Food and Nutrition Policy*

The Australian *Food and Nutrition Policy*, endorsed in 1992 [33], aims to improve the health of Australians and reduce the burden of preventable diet-related death, illness and disability. The policy strategies were developed in alignment with dietary guidelines and based on principles of good nutrition, ecological sustainability and equity.

1992 *Dietary Guidelines for Australians*

An expert panel was set up in 1989 by the Public Health Committee to review the existing dietary guidelines. In 1992, the *Dietary Guidelines for Australians* were published by the NHMRC for use by healthy adults. They represented the best consensus of scientific knowledge and public health advice available. A guideline to encourage and support breastfeeding was included, as were specific guidelines on calcium and iron.

1995 *The Core Food Groups*

The Core Food Groups [942] was the modeling document that underpinned the development of the food guide- *the Australian Guide to Healthy Eating* (see below). The purpose of *The Core Food Groups* [942] was to discuss the basis for a core food group system that reflects advances in nutrition knowledge and to complement existing nutrition references at the time (for example, *Dietary Guidelines for Australians, 1992).* The need for a core food group system arose because existing food selection guides in Australia at the time differed in the advice offered. *The Core Food Groups* document was developed to provide an approach that was objective, scientifically rigorous and updateable as new evidence on nutrition became available. It provided advice on core food

quantities consistent with national nutrition recommendations and targets, creating a platform for the interpretation of food and nutrition research into recommendations regarding food choices.

1998 *The Australian Guide to Healthy Eating*

The Australian Guide to Healthy Eating [45] is a food guide for Australia that reflects the multicultural nature of the population. Based on the modeling of the *Core Food Groups*, it is designed for all sectors of the food system to use as a nutrition education and information tool.

1999 *Dietary Guidelines for Older Australians*

The *Dietary Guidelines for Older Australians* [35] were based on the *Dietary Guidelines for Australians, 1992* [569] and were designed to take account of the changes in nutritional needs that occur with ageing. The *Guidelines* are aimed at healthy, independent Australians aged 65 and over, but additional advice was provided on how the *Guidelines* apply to older Australians who receive assistance with meals or live in residential aged care facilities. The *Guidelines* were also useful for health professionals who wished to develop suitable diets for older people in a range of health circumstances.

1999 *Australian Food and Nutrient Database (AUSNUT)*

The first in the AUSNUT (Australian Food and Nutrient Database) series was first released, an electronic file of nutrient data based on the technical support file for the 1995 National Nutrition Survey. The AUSNUT series of databases are derived from the NUTTAB series for the purpose of coding the food intake data collected in national nutrition surveys [943]. A second edition, used to code the 2007 Australian Children's Nutrition and Physical Activity Survey (insert ref), was released in 2008 (available at http://www.foodstandards.gov.au/consumerinformation/ausnut2007/) [944]. The third edition, developed to code the 2011 Australian Health Survey data, is planned for release in 2013.

2000 *Nutrition in Aboriginal and Torres Strait Islander Peoples: An Information Paper*

This information paper, endorsed by NHMRC, presented information about nutrition and nutrition-related disease in Aboriginal and Torres Strait Islander peoples. This information was specifically targeted to practitioners working to improve the nutritional health of Aboriginal and Torres Strait Islander peoples, and provided a reference material for practice and teaching.

2001 *Eat Well Australia: An Agenda for Action in Public Health Nutrition* and the *National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan*

Eat Well Australia: An agenda for action in public health [2] was designed to provide government and other sectors with a strategic framework and an agenda for action on public health nutrition for the first decade of the 21st century. This document provides the detailed *Eat Well Australia* Agenda for Action, as outlined in the summary *Eat Well Australia* Strategic Framework document. *Eat Well Australia* is a coherent national approach to the underlying causes of the preventable burden of diet-related disease and early death, providing a set of interlinked initiatives for the prevention and management of these diseases.

The *National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan (NATSINSAP*) [945] set out a framework for action to improve Aboriginal and Torres Strait Islander nutritional health under seven priority areas:

- food supply in rural and remote communities
- food security and socioeconomic status
- family-focused nutrition promotion
- nutrition issues in urban areas
- the environment and household infrastructure
- the Aboriginal and Torres Strait Islander nutrition workforce, and
- national food and nutrition information systems.

An evaluation of the *NATSINSAP* was completed in 2010, and is expected to contribute to informing future national policy development aimed at improving nutrition in Aboriginal and Torres Strait Islander peoples.

2003 *Dietary Guidelines for Adults*

Dietary Guidelines for Children and Adolescents incorporating the Infant Feeding Guidelines for Health Workers

The *Dietary Guidelines for Australian Adults* [37] and *Dietary Guidelines for Children and Adolescents in Australia [36]*, incorporating the *Infant Feeding Guidelines for Health Workers*, were based on the best available scientific evidence and provided information for health professionals and the general population about healthy food choices. The use of the *Guidelines* was intended to encourage healthy lifestyles to minimise the risk of developing diet-related diseases in the Australian population.

The *Guidelines* highlighted the groups of foods and lifestyle patterns that promote good nutrition and health. As with all previous dietary guidelines, recommendations were presented as an integral whole and no specific guideline was considered more important than any another.

2006 *Nutrient Reference Values for Australia and New Zealand Including Recommended Dietary Intakes*

In 2002, the NHMRC was commissioned by the Australian Department of Health and Ageing (DoHA) in collaboration with the New Zealand Ministry of Health to manage the revision process for the *Recommended Dietary Intakes* document. This resulted in the publication of the *Nutrient Reference Values for Australia and New Zealand Including Recommended Dietary Intakes* (NRVs) in 2006 [9]. The NRVs identified a number of reference values that address nutrient needs and excess intake for various age/sex groups.

2006 *NUTTAB Online*

The first online release of nutrient data, with the 6th edition of NUTTAB released by FSANZ. The 7th edition of NUTTAB was released in 2011 and is available at http://www.foodstandards.gov.au/consumerinformation/nuttab2010/ [946]. Most of NUTTAB's data is now Australian analytical data and covers more than 2500 foods available in Australia and up to 245 nutrients per food.

2011 *A Review of the Evidence to Address Targeted Questions to Inform the Revision of the Australian Dietary Guidelines*

The primary aim of the 'Evidence Report' [14] was to undertake a series of targeted systematic reviews of the literature published since 2002 on the interrelationship between food, diet, health and disease for different population subgroups.

2011 *A Modelling System to Inform the Revision of the Australian Guide to Healthy Eating*

In 2009 and 2010, the nutrient recommendations from the NRV 2006 document were translated (informed by *A Review of the Evidence to Address Targeted Questions to Inform the Revision of the Australian Dietary Guidelines*) into recommended amounts and types of foods which constitute a healthy diet that prevents nutrient deficiency, reduces risk of chronic disease, and is culturally acceptable, socially equitable and environmentally sustainable. This document updates the *Core Food Groups* 1995.

The *Modelling* document [10] also informs the development of these guidelines, which is an update of the previously separate reports: the *Dietary Guidelines for Adults* (2003), *Dietary Guidelines for Older Australians* (1999) and *Dietary Guidelines for Children and Adolescents* (2003).

Appendix 2. Process report

In 2003, NHMRC issued the *Dietary Guidelines for Australian Adults* and *Dietary Guidelines for Children and Adolescents in Australia,* having already issued *Dietary Guidelines for Older Australians* in 1999. With a policy of reviewing guidelines every five years, NHMRC in collaboration with the Healthy Living Branch of the Department of Health and Ageing (DoHA) commenced a review of the suite of Dietary Guidelines in 2008. The above three reports have now been combined into one set of *Australian Dietary Guidelines*.

The *Australian Dietary Guidelines* provide a resource that promotes the benefits of healthy eating to improve community health and wellbeing and to reduce the risk of diet-related disease. The *Guidelines* are intended for healthy members of the general population of any age, including people with common diet-related risk factors such as being overweight. They are not intended for people with medical conditions who require specialised dietary advice, or for the frail elderly who are at risk of malnutrition. The target audience of the *Guidelines* includes health professionals (including dietitians, nutritionists, general practitioners and nurses/lactation consultants), educators, government policy makers (for example, the government departments of health), the food industry and other interested parties. Other resources are being developed from the *Guidelines* for other target audiences.

RaggAhmed was commissioned in October 2010 by NHMRC through a Request for Quote process through the NHMRC technical writing panel to edit the draft chapters and collate them into one succinct report.

A2.1 Contributors

An expert working committee (the Working Committee), chaired by
Dr Amanda Lee and deputy-chaired by Professor Colin Binns, was appointed in April 2008 to guide, advise and author the redevelopment of the *Australian Dietary Guidelines*. Representatives from DoHA attended working committee meetings as observers.

A2.1.1 Members of the Working Committee

- Dr Amanda Lee (Chair)
- Professor Colin Binns (Deputy Chair)
- Dr Geoffrey Annison (from August 2010)
- Professor Sandra Capra, AM
- Professor Peter Davies
- A/Professor Sharon Friel
- Ms Clare Hughes
- A/Professor Mark Lawrence

- Professor Dorothy Mackerras
- Dr David Roberts (until May 2010)
- Dr Rosemary Stanton, OAM
- Professor Linda Tapsell

A2.1.2 NHMRC project team

- Ms Cathy Connor
- Ms Bronwyn Battisson
- Ms Marisa Bialowas
- Mrs Marion Carey
- Ms Cheryl Cooke
- Ms Vesna Cvjeticanin
- Ms Emma Milde
- Ms Tess Winslade

A2.1.3 Department of Health and Ageing project team

- Ms Erica Nixon
- Ms Caroline Arthur
- Ms Marina Dron
- Ms Erica Kneipp
- Ms Jacinta McDonald
- Ms Bronia Rowe
- Ms Leticia White

A2.1.4 Contractors

- Sky Newton, Adelaide Health Technology
- Philippa Middleton, methodologist, University of Adelaide
- Dr Katrine Baghurst (*Australian Guide to Healthy Eating*)
- RaggAhmed, technical writers
- Simon Grose, editor
- Dr Rachel Nowak, consultant in science policy, analysis and communication
- Quantum Market Research, health professional and consumer focus group testing

A2.2 Literature review

In the past, the *Dietary Guidelines* have provided recommendations based on evidence including that of nutrients and associations with health outcomes. However, as people eat foods rather than isolated nutrients, the Working Committee determined that the literature review should primarily

seek evidence on the relationship between foods, dietary patterns and health outcomes. The Working Committee determined that the revised guidelines would be an evolution from the previous versions and build upon their evidence and science base.

In 2009, the Dietitians Association of Australia was commissioned through an open Request for Tender process to systematically review the literature.

The Working Committee, with the assistance of a NHMRC Guideline Assessment Register Panel Consultant, Ms Skye Newton of Adelaide Health Technology Assessment, developed 27 complex search questions for the literature review in areas emerging in the literature and areas included in the 2003 Dietary Guidelines where the evidence base may have changed. A number of established food, diet and health relationships covered in the 2003 Dietary Guidelines, where the evidence base was unlikely to have changed substantially, were identified as not needing specific search questions to be asked. For example, the relationship between diets high in saturated fat and increased risk of high cholesterol.

The 27 search questions for the literature review were prioritised to 12 complex questions in consideration of time and financial constraints. These formed the basis of questions for three types of review: systematic literature review (the systematic review of the primary literature); umbrella review (the systematic review of systematic reviews); and narrative reviews (the comprehensive review of the literature to answer more qualitative questions, for example, such as that concerning nature and scope of international food guides and practices promoting food safety).

The final questions for systematic and umbrella reviews were transcribed into PICO format. Detailed definitions and search terms were developed for each component of the final complex search questions and a number of specific search questions for each variable arising from various permutations were formulated for each complex question.

Standardised processes were used to review the literature [34, 39-41]. Databases searched included CINAHL, MEDLINE, DARE, Cochrane, ScienceDirect, PsychLit and ERIC. For each specific search question, the identified articles were retrieved and reviewed for relevance by a team of reviewers. Papers published before 2002 were excluded. Duplicates, papers not within the scope of the search questions and papers that were already included in meta-analyses, described cross-sectional studies or were not research studies (for example, letters and editorials) were also excluded.

The evidence was assessed according to *NHMRC levels and grades for recommendations for developers of guidelines* [41] which allowed each article to be critically appraised and assigned a level of evidence based on a hierarchy according to the type of research question. As this review looked at causality and intervention research questions the following hierarchy was used:

- Level I - A systematic review of level II studies
- Level II - A randomised controlled trial
- Level III-1 - A pseudorandomised controlled trial

- Level III-2 - A comparative study with concurrent controls:
  Non-randomised experimental trial
  Cohort study
  Case-control study
  Interrupted time series with a control group
- Level III-3 - A comparative study without concurrent controls:
  Historical control study
  Two or more single arm study
  Interrupted time series without a parallel control group
- Level IV - Case series with either post-test or pre-test/post-test outcomes

- Data was extracted from included studies and assessed for strength of evidence, size of effect and relevance of evidence according to standardised NHMRC processes [34, 39-41]. The components of the body of evidence - *evidence base* (quantity, level and quality of evidence); *consistency* of the study results; *clinical impact*; *generalisability*; and *applicability* to the Australian context) - were rated as *excellent*, *good*, *satisfactory* or *poor* according to standard NHMRC protocols [41]. The reviewers then translated the evidence into a draft body of evidence statement. The draft Evidence Statements were graded A to D according to standard NHMRC protocols [41] where:

- Grade A indicates that the body of evidence can be trusted to guide practice
- Grade B indicates that the body of evidence can be trusted to guide practice in most situations
- Grade C indicates that the body of evidence provides some support for the recommendations but care should be taken in its application
- Grade D indicates that the body of evidence is weak and any recommendation must be applied with caution.

In order to reduce potential risks associated with advice being based on findings of single or only a few studies, the Working Committee and DAA Contractors advised that a minimum of five quality studies were required before a graded draft evidence statement could be made. The individual studies in included meta-analysis studies were considered as separate studies.

Once the Evidence Statements and Grades had been drafted the NHMRC commissioned a methodologist through the NHMRC methodologist panel, Ms Phillippa Middleton from the University of Adelaide, to assist the Working Committee to ensure that the review activities had been undertaken in a transparent, accurate and unbiased manner. The methodologist and the Working Committee scrutinised each step of the review process by accessing the original papers and reviewing the rating of evidence components, and the wording and grading of each draft Evidence Statement. As a result, some Evidence Statements and Grades were amended using a Working Committee consensus approach, and the final Evidence Statements and Grades were agreed.

As nutrition is a continuously evolving area and research studies are published on a regular basis, the Working Committee also considered results from high quality studies (primarily systematic reviews) published after the literature review, and where deemed warranted, included the findings and references in the relevant evidence sections in each Chapter. However, only the Evidence Statements from systematic review of the literature until 2009 were graded.

The results of this literature review, including the questions that were asked, are included in the report of the commissioned literature review: *A Review of the Evidence to Address Targeted Questions to Inform the Revision of the Australian Dietary Guidelines* (referred to as 'the Evidence Report') [14].

The original literature search did not locate a sufficient number of food-based studies - rather than nutrient-based studies - to be able to draft evidence statements regarding food intake and dietary patterns for pregnant and breastfeeding women and health outcomes of infants and/or mothers. Therefore, a team from the University of Adelaide and the Women's and Children's Health Research Institute was commissioned through a Request for Quote process from the NHMRC multi-use panel to conduct a more extensive search of the literature to locate and summarise studies meeting specified eligibility criteria. This team, led by Philippa Middleton and comprising Professor Maria Makrides, Dr Carmel Collins, Dr Alice Rumbold, Dr Jo Zhou, Professor Caroline Crowther and Associate Professory Vicki Flenady reviewed the literature for health outcomes for infants and mothers from food-based studies on pregnant and breastfeeding women.

Parts of this review were used to inform the 'practical considerations' for pregnant and breastfeeding women that have been included in these guidelines.

The Working Committee also determined that there was an evolving and increasing body of literature on the environmental impacts of the production and consumption of food and vice-versa. Therefore, in addition to the original narrative review, a review of the relevant literature, particularly that pertaining to Australia, was conducted by A/Professor Friel and a team at the Australian National University. Following this work, it became clear that the inter-relationship between diet and the environment is a cross-sectoral matter and should be considered as such. NHMRC will work further with other relevant Australian government agencies to develop this work.

A2.3 Modelling system

In 2008 to 2010, NHMRC undertook an extensive review of the *Core Food Groups* (1994) and released *A modelling system to inform the revision of the Australian Guide to Healthy Eating* (the Modelling System) in 2011 [10, 942]. The modelling system determined a range of combinations of the amounts and types of foods that could be consumed to meet the nutritional needs within the least amount of energy for the smallest and least active people within an age and sex group. These are called *Foundation Diets*. For those that are more physically active or taller (and older, in the case of infants, children and adolescents) within each age/sex group, the models provide additional

food options to meet additional energy needs. These are called *Total Diets*. (Note that the *Total Diet* for the smallest, least active people in each age/sex groups is equivalent to the *Foundation Diet*.)

The number of serves and serve sizes modelled in the Modelling System were considered together with other sources of evidence to determine the recommendations in these guidelines (see Section A2.3 below).

The Modelling System is located at www.eatforhealth.gov.au.

A2.3 Development of the *Australian Dietary Guideline* recommendations

In translating the evidence to formulate the Dietary Guideline recommendations the Working Committee considered the following sources of evidence.

- The commissioned literature review: *A Review of the Evidence to Address Targeted Questions to Inform the Revision of the Australian Dietary Guidelines* (referred to as 'the Evidence Report') [14].
- The commissioned report: *A Modelling System for Australia to Inform the Revision of the Australian Guide to Healthy Eating* (referred to as 'the Food Modelling' document) [10].
- NHMRC and NZ Ministry of Health 2006 *Nutrient Reference Values for Australia and New Zealand Including Recommended Dietary Intakes* (referred to as 'the NRV document') [9].
- Key authoritative government reports and documents provided by the Working Committee and the NHMRC, including evidence in material provided by stakeholders during consultation processes and findings of relevant large, quality, peer-reviewed studies published after 2009.
- The previous Dietary Guidelines for Australians series and their supporting documentation [35-37].

The information in the previous guidelines was used as the basis. New evidence was assessed to determine whether associations between food, dietary patterns and health outcomes had strengthened, weakened, or stayed the same since the last review of the evidence.

The final wording of the Dietary Guideline recommendations was developed by a Working Committee consensus approach and focus tested.

A2.3.1 Public consultation

The Council of NMHRC considered the draft guidelines on 1 April 2011, on 16 June 2001, and again on 2 November 2011. On 2 November 2011the Council agreed to recommend the Chief Executive Officer (CEO) of NHMRC release the *Guidelines* for public consultation.

Public consultation on the draft guidelines was undertaken from xxxxx to xxxx. The public consultation was advertised in the major Australian newspapers and on the NHMRC website. Invitations were also sent to a large number of key stakeholders and those with a known interest in nutrition. XXX submissions were received from a variety of stakeholders including …..

The Working Committee met on xxx to consider the submissions. Specific concerns included:

A2.3.2 Peer review

Text to come …

A2.3.3 Independent review

Text to come …

A2.3.4 Endorsement

The *Guidelines* were considered by the Council on xxx for recommendation to the CEO for issuing. The CEO was pleased to accept the Council's advice and agreed to issue to guidelines under Section 7(1a) of the *National Health and Medical Research Council Act 1992.*

A2.3.5 Dissemination and implementation

Text to come …

A2.3.6 Review

In line with NHMRC policy, the *Guidelines* will be reviewed within five years of publication.

Appendix 3. Assessing growth and healthy weight in infants, children and adolescents, and healthy weight in adults

A3.1 Assessing growth and healthy weight in infants, children and adolescents

Measuring height and weight regularly is important. The most practical measures of growth status in childhood are comparisons with reference growth charts that show the normal ranges of height for age, weight for age and BMI, by sex. As growth is a dynamic process, several measurements are preferable when assessing children. When only a single measurement of weight and/or height is available, care is need in interpretation. For adults an assessment of obesity is usually made on the basis of one measurement.

There are several widely accepted and practical tools available for assessment. The options are:

- BMI using international reference standards (The International Obesity Task Force [IOTF] criteria) [712]
- CDC BMI curves (also recommended by WHO before 2006) [947]
- The new WHO growth reference (2006) BMI charts [948-950]

The IOTF table uses cutoff points based on WHO adult BMI but extrapolated for use in children based on six international data sets. The differences between these options have been reviewed by Monasata [951]. It is important to note that the use of different methods of assessing overweight or obesity will give different results. The results cannot be directly compared. For that reason it is essential to state the criteria used when stating the overweight or obesity level of an individual or population.

Measurement and recording of weight and height at regular intervals remains the best method to monitor growth. Generally, irrespective of the reference standard used, if a child is growing normally, the lines connecting the plotted values will proceed along - or parallel to - one of the percentile lines on the charts. However at adolescence the growth trajectory may increase or decrease dependent upon timing of the adolescent growth spurt. Care should always be taken when both measuring and plotting growth patterns to minimize error associated with poor measurement technique or error in plotting.

A3.1.1 Growth monitoring in the first few years of life

Measurement and recording of the growth of infants and young children has been standard practice in Australia for decades. Growth monitoring remains the best method of assessing nutritional status and overall health at the community and primary care level. Commonly 'Weight for Age' and 'Length/Height for Age' growth reference charts are used. The most common growth reference in use in Australia was derived from US data (CDC 2000 [952]). The rate of growth is the most important factor, although if growth is above the 95th percentile or below the 5th, or crosses these percentiles, further assessment is required. In the first months of life monitoring an infant's weight and using growth reference charts is one of the ways in which the adequacy of breastfeeding is assessed.

The new WHO growth reference (2006) [950] is heavier than the older growth reference currently in use in Australia in the first six months of life. Care should be taken to adjust for this difference when assessing the growth of infants. The 2nd percentile of the new WHO chart equivalent to the 5th percentile on the old chart. It is important when using growth charts to remember that they are a tool, not a diagnostic instrument. The pattern of the infant's growth, and in particular whether babies are crossing the centile channels as well as the position on the growth chart, are used in conjunction with clinical assessment in determining if there is a problem.

A3.1.2 BMI using international reference standards

An international reference (International Obesity Task Force) for defining overweight and obesity in children and adolescents were developed with data from six countries, and provide age and sex-specific BMI cut-off points for children and adolescents aged 2–18 years that correspond to adult BMI values of 25 kg/m^2 for overweight and 30 kg/m2 for obesity [712]. This method was recommended for use in the 2003 NHMRC *Dietary Guidelines for Children and Adolescents*. BMI has become increasingly accepted as a useful tool for identifying and monitoring obesity in children [947, 953-955].

Table A3.1 International BMI cut-off points for overweight and obesity: males and females aged 2-18 years

	Body mass index equivalent to 25kg/m² in adults		Body mass index equivalent to 30kg/m² in adults	
Age (years)	Males	Females	Males	Females
2	18.41	18.02	20.09	19.81
2.5	18.13	17.76	19.80	19.55
3	17.89	17.56	19.57	19.36
3.5	17.69	17.40	19.39	19.23
4	17.55	17.28	19.29	19.15
4.5	17.47	17.19	19.26	19.12
5	17.42	17.15	19.30	19.17
5.5	17.45	17.20	19.47	19.34
6	17.55	17.34	19.78	19.65
6.5	17.71	17.53	20.23	20.08
7	17.92	17.75	20.63	20.51
7.5	18.16	18.03	21.09	21.01
8	18.44	18.35	21.60	21.57
8.5	18.76	18.69	22.17	22.18
9	19.10	19.07	22.77	22.81
9.5	19.46	19.45	23.39	23.46
10	19.84	19.86	24.00	24.11
10.5	20.20	20.29	24.57	24.77
11	20.55	20.74	25.10	25.42
11.5	20.89	21.20	25.58	26.05
12	21.22	21.68	26.02	26.67
12.5	21.56	22.14	26.43	27.24
13	21.91	22.58	26.84	27.76
13.5	22.27	22.98	27.25	28.20
14	22.62	23.34	27.63	28.57
14.5	22.96	23.66	27.98	28.87
15	23.29	23.94	28.30	29.11
15.5	23.60	24.17	28.60	29.29
16	23.90	24.37	28.88	29.43
16.5	24.19	24.54	29.14	29.56
17	24.46	24.70	29.41	26.69
17.5	24.73	24.85	29.70	29.84
18	25.00	25.00	30.00	30.00

A3.1.3 CDC BMI curves

The Centers for Disease Control (CDC) age and sex-specific BMI curves [947] are commonly used. In Australia, NHMRC and DoHA had recommended use of the US CDC growth charts [952] for children and adolescents aged 2–18 years [36, 956]. This reference was also used by WHO before 2006. NHMRC defined *overweight* as above the 85th percentile for BMI-for-age and *obese* as above the 95th percentile for BMI-for-age.

However, use of the CDC Charts has been criticised as they are based on US data only, from five national surveys undertaken between 1963 and 1994, the most recent being NHANES 111. The age and sex-specific CDC growth charts were last updated in 2000, and do not include weight data (and thus BMI data) from NHANES 111 from children over six years of age.

A3.1.4 WHO growth charts

The World Health Organization (WHO) has developed and uses two sets of growth reference charts, one for children from birth to age five years [949], and one for children and adolescents from age 5 to 19 years [948]. For children younger than five years, tables and charts showing percentiles and Z-scores for BMI-for-age, weight-for-age, and weight-for-length were published in 2006. Overweight is defined as two standard deviations above normal on the weight-for-height chart and underweight is defined as weight-for-age two standard deviations below normal [949]. For children and adolescents aged five to 19 years, tables and charts showing percentiles and Z-scores for BMI-for-age, height-for-age, and weight-for-age were published in 2007. The BMI Z-score-for-age chart defines overweight as greater than one standard deviation above normal, obesity as greater than two standard deviations above normal, thinness as less than two standard deviations below normal, and severe thinness as less than three standard deviations below normal [948]. Recently the US has adopted the WHO growth charts [952] for children below 2 years of age. However, these WHO growth charts are not commonly used in Australia at present.

A3.1.5 Others

Waist circumference and waist/height ratio are becoming more common measures of child and adolescent central adiposity, but there is little consensus regarding standard cut-off values to define overweight and obesity [717, 957-962]. Waist circumference is widely used in adults because of its relationship to morbidity and mortality.

Skinfold measurement – an index of subcutaneous adipose tissue – is another method of defining of under- or over-nutrition [963]. Reliable and accurate measurements depend to a large extent on the use of trained operators and properly calibrated instruments.

Other methods of body composition assessment, for example DEXA (dual energy X-ray absorptiometry), can provide accurate measurements, but cost limits their application to experimental use and to clinical settings where more accurate diagnosis is required for management [964]. The most accurate measure of body composition is provided by using doubly labelled water, but cost limits its use to research.

A3.1.6 Z-scores and percentiles: Converting between measures

While percentiles are most commonly used in Australia, in some situations, particularly for research, it is more appropriate to use the Z-scores (standard deviations above or below the mean). The conversion of percentiles to Z-scores requires a table of normal distributions. The 50th percentile is a Z score of 0, the 90th percentile is +1.28, and 10th percentile is –1.28. Growth reference charts are available in both formats and a calculator is available on the internet (http://www.cdc.gov/growthcharts/ [952]). BMI can be converted into a BMI Z-score using a BMI-for-age growth chart and the formula: Z-score = ((BMI/M)L-1)/(LS), where M, L, and S are values selected from reference tables corresponding to the age of the child in months [36].

A3.1.7 Differences between IOTF criteria and the new WHO growth reference

There are major differences in the percentage of children classified as obese or overwight according to the reference used, ranging from 5% to 25% in a study of 3 year old children [951]. These authors concluded that 'the IOTF reference and cut-offs could be preferable for the identification of overweight and obesity both at individual and population levels because they are at least based on a crude association with ill health later in life, namely the definition of overweight and obesity at age 18 years' [951].

A3.2 How is healthy weight measured in adults?

For adults the standard WHO BMI cutoff points are most commonly used for assessment of obesity, and separate cutoffs are available for Asian populations.

Body mass index (BMI) is a measure of body size that is widely used as an index of relative risk of mortality and morbidity at the population level [687, 691]. The association of mortality with BMI is a J-shaped curve with the lowest risk – normal healthy weight – within the range 18.5 to 25 [687, 691]. The cutoff point is 28 for older Australians.

BMI = weight (in kilograms)/height (in metres)^2 can be used to define weight status in adults (except older adults) as follows:

Underweight <18.5
Healthy weight 18.5–24.9
Overweight ≥25.0

Obesity ≥30.0 [112, 126].

People of Asian origin have the equivalent risk at a lower BMI and people of Polynesian origin have the equivalent risk at a higher BMI [708].

It is important to measure weight and height accurately to assess overweight and obesity, as self-reported data is usually inaccurate.

Waist circumference is increasingly being used as an alternative measure of body size for risk assessment in adults, and provides a better estimate of risk in adults than BMI [965, 966]. Above 80 centimetres in women or 94 centimetres in men is regarded as being overweight and over 88 centimetres in women or above 102 centimetres in men is classified as obese [710, 759].

Appendix 4. Physical activity guidelines

Australia's physical activity guidelines outline the minimum levels of physical activity required to gain a health benefit and identify ways to incorporate physical activity into everyday life. Below are excerpts from physical activity guidelines developed and published by Australia's Commonwealth Department of Health and Ageing: http://www.health.gov.au/internet/main/publishing.nsf/content/health-pubhlth-strateg-phys-act-guidelines.

A4.1 Physical activity recommendations for children 0–5 years

The *Physical Activity Recommendations for Children 0–5 years* state that being physically active every day is important for the healthy growth and development of infants, toddlers and preschoolers [850].

For infants (birth to 1 year) physical activity—particularly supervised floor-based play in safe environments should be encouraged from birth.

> Before infants begin to crawl, encourage them to be physically active by reaching and grasping, pulling and pushing, moving their head, body and limbs during daily routines and supervised floor play, including tummy time. Once infants are mobile, encourage them to be as active as possible in a safe, supervised and nurturing play environment.

Toddlers (1 to 3 years) and preschoolers (3 to 5 years) should be physically active every day for at least three hours, spread throughout the day.

> Young children don't need to do their three hours of physical activity all at once. It can be accumulated throughout the day and can include light activity like standing up, moving around and playing as well as more vigorous activity like running and jumping. Active play is the best way for young children to be physically active.

Children younger than 2 years of age should not spend any time watching television or using other electronic media (DVDs, computer and other electronic games). For children 2 to 5 years of age, these activities should be limited to less than one hour per day.

> Television, DVDs and playing computer games usually involve sitting for long periods—time which could be spent playing active games or interacting with others.

Infants, toddlers and preschoolers should not be sedentary, restrained, or kept inactive, for more than one hour at a time, with the exception of sleeping.

> All children need some 'down time' but they are not naturally inactive for long periods of time. Sitting in strollers, highchairs and car seats (restrained) for long periods isn't good for children's health and development. Try to take regular breaks on long car trips and walk or pedal for short trips when you can.

While meeting these recommendations may seem like a challenge at times, a brochure that includes tips and ideas to help you include more activity in your child's day and further information on the recommendations can be found at National Physical Activity Recommendations for Children 0–5 years.

A4.2 Physical activity recommendations for 5–12 year olds

The *Physical Activity Recommendations for Children 5–12 years* [850] recommend that children need at least 60 minutes (and up to several hours) of moderate to vigorous physical activity every day.

Examples of moderate activities are a brisk walk, a bike ride or any sort of active play. More vigorous activities to make kids 'huff and puff' include organised sports such as football and netball, as well as ballet, running and swimming laps. Children typically accumulate activity in intermittent bursts ranging from a few seconds to several minutes, so any sort of active play will usually include some vigorous activity. Most importantly, kids need the opportunity to participate in a variety of fun activities that suit their interests, skills and abilities. Variety will also offer your child a range of health benefits, experiences and challenges.

Children shouldn't spend more than two hours a day using electronic media for entertainment (for example, computer games, TV, internet), particularly during daylight hours.

A4.3 Physical activity recommendations for 12–18 year olds

The *Physical Activity Recommendations for Children 12–18 years* [850] state that:

- at least 60 minutes of physical activity every day is recommended. This can build up throughout the day with a variety of activities
- physical activity should be done at moderate to vigorous intensity
- for additional health benefits, try to include 20 minutes or more of vigorous activity that causes young people to 'huff and puff at least three to four days a week
- children shouldn't spend more than two hours a day using electronic media for entertainment (computer games, TV, internet), particularly during daylight hours.

Suggested activities include:

- moderate activities like brisk walking, bike riding with friends skateboarding and dancing, walking the dog, replacing short car trips with a walk or bike ride
- vigorous activities such as football, netball, soccer, running, swimming laps or training for sport
- trying to be active in as many ways as possible – variety is important in providing a range of fun experiences and challenges and provides an opportunity to learn new skills.

A4.4 Physical activity guidelines for Australian adults

The *Physical Activity Guidelines for Australian Adults* [850] make the following recommendations.

- Think of movement as an opportunity, not an inconvenience.
- Be active every day in as many ways as possible.
- Put together at least 30 minutes of moderate-intensity physical activity on most, preferably all, days.
- If possible, also enjoy some regular vigorous exercise for extra health and fitness.

Moderate-intensity activity will cause a slight, but noticeable, increase in your breathing and heart rate. A good example of moderate-intensity activity is brisk walking, that is at a pace where you are able to comfortably talk but not sing. Other examples include mowing the lawn, digging in the garden, or medium paced swimming or cycling. The *guidelines* note that accumulated short bouts of moderate-intensity activity of around 10–15 minutes without stopping is just as effective as continuous activity of at least 30 minutes per day on most days of the week, which is the minimum level of activity required for preventing or improving specific health indicators such as hypertension, heart disease and diabetes in adults [967]. However, this level of activity appears to be insufficient for preventing weight gain or weight loss or weight regain in most people [820, 968].

A4.5 Physical activity recommendations for older Australians

The *Physical Activity Recommendations for Older Australians* [850] indicate that it is never too late to become physically active and to feel the associated benefits. Most physical activities can be adjusted to accommodate older people with a range of abilities and health problems, including those living in residential care facilities.

There are five physical activity recommendations for older Australians.

- Older people should do some form of physical activity, no matter what their age, weight, health problems or abilities.
- Older people should be active every day in as many ways as possible, doing a range of physical activities that incorporate fitness, strength, balance and flexibility.

- Older people should accumulate at least 30 minutes of moderate-intensity physical activity on most - preferably all - days.
- Older people who have stopped physical activity, or who are starting a new physical activity, should start at a level that is easily manageable and gradually build up the recommended amount, type and frequency of activity.
- Older people who have enjoyed a lifetime of vigorous physical activity should carry on doing so in a manner suited to their capability into later life, provided recommended safety procedures and guidelines are adhered to.

Appendix 5. Studies examining the health effects of intake of fruit and vegetables together

The literature includes studies which have investigated the effect of consumption of fruit and vegetables together. This evidence clearly confirms the positive health effects of consuming vegetables and fruits, particularly in reducing the risk of cardiovascular disease, but also in reducing risk of obesity and some cancers. It is compiled as an Appendix to aid the conciseness of Chapter 2.

A5.1 Cardiovascular disease, type 2 diabetes and excess weight

There is evidence regarding a probable association between the consumption of each additional daily serve of fruit and vegetables and reduced risk of coronary heart disease (Grade B, Section 3.1 in the Evidence Report [14]) [145-147, 150, 969].

Similarly, recent evidence supports a probable association between consumption of each additional daily serve of fruit and vegetables and a reduced risk of stroke (Grade B, Section 3.2 in the Evidence Report [14]) [145, 146]. Consuming more than five serves of fruit and vegetables a day was found to reduce the risk of stroke by 26% (fruit serve was 80g and vegetables 77g) [146], and consuming each additional serve of fruit and vegetables (of average serve size 106g) reduced the risk of stroke by 5% [145]. These results are consistent with those presented in the literature reviews to inform the recent review of the *Dietary Guidelines for Americans, 2010* which found that 'consistent evidence suggests at least a moderate inverse relationship between vegetable and fruit consumption with myocardial infarction and stroke, with significantly larger, positive effects noted above five servings of vegetables and fruits per day' [143].

In the literature reviews to inform the revision of the *Dietary Guidelines for Americans, 2010*, the evidence for an association between increased fruit and vegetable intake and reduced risk of excess body weight was found to be modest, with a trend towards decreased weight gain over five or more years in middle adulthood associated with increased fruit and vegetable intake. However no conclusions could be drawn from the evidence of efficacy of increased fruit and vegetable consumption in weight loss diets [143]. However, in children and adolescents, the limited body of evidence from longitudinal studies suggested that greater intakes of fruits and/or vegetables may protect against increased adiposity [143].

A5.2 Cancer

Lung cancer: The recent body of evidence suggests that consumption of fruit and vegetables is associated with reduced risk of lung cancer (Grade C, Section 3.5 in the Evidence Report [14]) [970-972].

Colorectal cancer: Recent evidence suggests that there is no association between consumption of fruit and vegetables together and risk of colorectal cancer (Grade C, Section 3.4 in the Evidence Report [14]) [177, 202]. In its 2008 report, the International Agency for Research on Cancer (IARC) also concluded that intakes of fruit and vegetables were either not associated or only slightly associated with risk of colorectal, breast and prostate cancer [973]. However, recently published findings from the European Prospective Investigation into Cancer and Nutrition (EPIC) study suggest that a high consumption of fruit and vegetables is associated with reduced risk of colorectal cancer, especially of colon cancer, but that the effect may also depend on smoking status [974].

Ovarian cancer: It is probable that there is no association between consumption of fruit and vegetables and risk of ovarian cancer (Grade B, Section 3.6 in Evidence Report [14]) [172, 173]. A more recent meta-analysis has described evidence of a probable inverse relationship between consumption of vegetables and fruit, and bladder cancer [975]. An insufficient number of studies were found to produce an evidence statement for an association between the consumption of fruit and vegetables and renal cancer [976-978].

Epithelial cancer: In general, comparison of the results of systematic reviews of the evidence on diet and cancer sponsored by the World Cancer Research Fund in 1997 [586] and 2007 [42] suggests weaker evidence of a protective effect of high intakes of fruits and vegetables against several common epithelial cancers, with a downgrading of the association from 'convincing' to 'probable.' This is also consistent with the evidence presented in the International Agency for Research on Cancer report [979].

Overall cancer: Analyses of prospective studies have generally failed to demonstrate consistent evidence of a convincing association between the intake of fruits and vegetables and overall risk of cancers [973]. However the more recent EPIC cohort study found a weak but statistically significant inverse association between consumption of fruit and vegetables and risk of overall cancers —a 4% lower incidence of all cancers combined for an increment of 200g total fruit and vegetable intake per day [980].

It should also be noted that a very weak association between the consumption of fruit and vegetables as a whole and overall cancer rates is not inconsistent with the evidence of the effects of fruit and vegetable consumption on site-specific cancers, or the evidence that specific types of fruit or vegetables may have an effect, particularly on the risk of site-specific cancers, and suggests

that further specific studies are required, particularly those which attempt to account for potential interactions between tobacco [981] and alcohol [211].

A5.3 Other conditions

Although not sufficient to develop evidence statements, individual studies found an association between other health conditions and consumption of fruit and vegetables. These include:

- decreased risk of high blood pressure [982]
- decreased risk of dementia [317]
- decreased risk of head and neck cancer [983]
- decreased risk of various upper digestive tract cancers [44, 864, 984-986]
- decreased risk of prostate cancer [229, 987]
- obesity [988, 989].

Although not sufficient to develop evidence statements, individual studies found no evidence an association between consumption of fruit and vegetables and pancreatic cancer [585, 990, 991].

Appendix 6. Alcohol and energy intake

Table A6.1 Median percentage of contribution of alcohol to energy intake per consumer

	Age groups					
	16-18 y	19-24 y	25-44 y	45-64 y	≥65 y	≥19y and over
Male (%)	4.2	7.7	9.2	9.5	9.0	9.1
Female (%)	4.9	9.0	8.4	7.5	7.4	8.1

Source: *National Nutrition Survey 1995* [47]

Table A6.2 Energy and alcohol content of common alcoholic drink serves

Drink type	Serve size	Energy (kJ/serve)	Alcohol (g/serve)a	Standard drink equivalent
Beer, regular (4.5–4.9% alcohol)	1 can/stubby (375mL)	568	15.4	1½
Beer, medium–light (3.7% alcohol)	1 can/stubby (375mL)	546	14.0	1
Beer, light (2.1% alcohol)	1 can/stubby (375mL)	395	8.0	½
Wine (average of dry white and red)	1 glass (100mL[b])	340 – 395	9.5	1
Spirits	1 nip (30mL)	255	8.8	1
Pre-mixed spirits	1 can (375mL)	1182	13.9	1½

a. Using *1995 National Nutrition Survey* data [44]
b. Note a glass of wine is usually more than 100mL, i.e. more than one standard drink.

Appendix 7. Equity and the social determinants of health and nutrition status

A7.1 Introduction

The World Declaration on Nutrition (1992) states that 'access to nutritionally adequate and safe food is a basic individual right' [992]. Australia is fortunate to have an abundant and safe food supply. Life expectancy and health status are relatively high [12, 26, 44]. Australians are generally literate and have good access to health and nutrition information and sufficient education to make informed food choices [993].

However, there are differences in health and wellbeing between groups of Australians. People in lower socioeconomic groups have shorter life spans and poorer health. They have higher rates of death and disease, are more likely to be hospitalised and are less likely to use specialist and preventative health services [994]. As in other countries, there is a socioeconomic gradient whereby health status generally improves the higher a person is up the socioeconomic ladder [26].

The determinants of health inequities are largely outside the health system and reflect the distribution of social, economic and cultural resources and opportunities [27, 28, 992, 994]. Employment, income, education, cultural influences, lifestyle, language, sex and other genetic differences, geographic, social or cultural isolation, age and disability, the security and standard of accommodation, and the availability of facilities and services, all influence diet, health and nutritional status [27, 28].

The relationship between these factors and health status is complex and it is often difficult to determine the nature and direction of causal relationships [27, 28]. For example, those on higher incomes tend to have greater opportunity to attain higher levels of education and afford housing in higher socioeconomic areas with better access to goods and services (for example, health services, transport, shops including food outlets) that support healthy lifestyles. Lower levels of education and/or an individual's poor health status can limit opportunities for employment and therefore income and access to other goods and services, including nutritious food [27, 28].

While higher education can improve health literacy, just because a person can understand healthy lifestyle and nutrition information does not mean they can or will act on it. For example, one Australian study of people 16 years and older found that, although 80% and 35% of people knew the recommended daily intake of fruit and vegetables respectively, only 56% and under 10% met these respective recommendations [26].

The economic, social and cultural factors that influence health inequities also influence the ability of an individual to choose nutritious foods consistent with dietary guidelines [14]. The ability of parents and carers to make nutritious food choices is likely to affect their family's nutrition status too.

Factors associated with complying with dietary guidelines include being female, older age, higher socioeconomic status, with higher education and having nutrition knowledge [43, 138, 858, 995-1011].

Conversely, lower socioeconomic status and lower educational attainment are barriers to complying with dietary guidelines, and lower socioeconomic groups perceive cost as a barrier to healthy food purchase [43, 138, 858, 995-1011].

In a Melbourne study it was found that areas of greater socio-economic advantage had closer access to supermarkets, whereas areas of less socio-economic advantage had closer access to fast food outlets [137].

A greater understanding of the barriers to consuming a nutritious diet will help ensure that appropriate messages, education and public health strategies are developed for groups who experience a greater burden of diet-related disease. It was essential that the social determinants of health and nutrition status were considered in the *Guidelines* so as to reduce the risk of adding to health inequities, for example, by promoting consumption of expensive or hard to access foods.

A7.2 Social distribution of diet-related health outcomes

In 2002–06 the death rate for people between 15 and 64 years was 70% higher in the lowest SES group than the highest SES group [26]. These rates reflect the higher prevalence of type 2 diabetes and cardiovascular disease among people living in the lowest SES group [12, 26].

Gradients in risk factor prevalence are also apparent across quintiles of social disadvantage as defined by socio-economic indexes for areas (SEIFA). The SEIFA assesses the relative social disadvantage of respondents by the economic resources, education and occupation patterns of their area of residence [26]. Overweight and obesity rates are also highest among the lower SES areas, although there was not a clear gradation across SEIFA quintiles [26]. Some of the factors that contribute to the development of overweight are also related to equity, particularly the social, cultural and economic barriers and enablers to healthy food choices (see Chapter 2). There is also a social gradient in physical activity levels; self-reported 'sedentary' behaviour rises from 25% of people in the highest SES areas to 44% in the lowest SES areas [26].

A7.3 Social distribution of food intake and nutrition status

Socioeconomic factors have a large impact on food and nutrient intakes and food purchasing decisions and patterns [858], and there is clear evidence of a social gradient for the distribution of diet-related chronic disease. Yet evidence for a social gradient related to specific foods - rather than overall dietary patterns - is less clear.

Two recent systematic reviews assessed the impact of dietary interventions relative to social disadvantage [1012] and determinants of healthy eating for those with low income [1013]. They found that economic and cultural influences impact on consumption of specific foods or food groups. While nutrition interventions can have greater impact in higher socioeconomic areas and non-ethnic groups, they do not have a detrimental impact in low socioeconomic groups [1012, 1013].

Those with the least disposable income are at the greatest risk of poor nutrition as households vulnerable to poverty spend less per person on food but a greater proportion of their total expenditure on food [1014]. Analysis of Australian household food expenditure data suggests that a substantial proportion of the population is severely restricted in its capacity to make nutritious food choices and to achieve a healthy diet [1015].

A7.3.1 Economics of food choice

There is a growing body of research which indicates that food groups with more favourable nutrient profiles are more expensive. The 'economics of food choice' theory states that people's dietary decisions, when made within the context of sustained budgetary constraints, are driven by maximising energy value for money (dollars per megajoule [$/MJ]), resulting in energy-dense, nutrient-poor diets [730, 806]. For instance, meat, fruit and vegetables food groups have the highest nutritional quality however are usually associated with highest costs, while sweets and salted snacks have the lowest nutritional quality but are an inexpensive source of dietary energy [730, 806, 859, 1016]. Although high-quality nutrition is known to protect against chronic diseases, energy-dense, nutrient-poor foods costs considerably less than nutrient-rich foods [728].

The costs of healthy (low energy-density, high nutrient density) foods are reported to be increasing in Australia in comparison with higher energy density, lower nutrient density food. Over six years from 2000 to 2006 the Consumer Price Index for food in Brisbane increased by 32.5% while the cost of a standard basket of healthy food increased approximately 50% across Queensland [140].

A diet consistent with the *Guidelines* is expensive for welfare-dependent families. An Australian study found it cost around 40% of their disposable income compared with 20% for families on the average income [138].

A7.3.2 Food security

Food security refers to the ability to access nutritious, affordable foods and the capacity to obtain them. At an individual or family level, food insecurity can be characterised by running out of food and being unable to afford to buy more. About 5% of the Australian population suffer food insecurity in a year [1017-1019]. It is more common among:

- the unemployed [1019]
- Indigenous Australians [1020]
- those living in single parent households [1019]
- those in the second lowest income quintile [1019]
- those living in rental households [1019]
- younger than older people [1019]
- women than men (in some surveys but not others) [1017, 1018].

Issues of food security in the face of rising food costs are of concern in Australia [140] (see Section A7.3.1). For example, national Consumer Price Index data for the March 2011 quarter indicates that the cost of food had increased by 2.9% and that, primarily due to impacts of flooding in Queensland and Victoria and Cyclone Yasi in Queensland, the cost of fruit increased by 14.5% and the cost of vegetables increased by 16.2% [1021].

There is an urgent need to nationally monitor and sustainably address the factors affecting the price of nutritious foods, particularly for vulnerable groups who suffer a disproportionate burden of poor health [140].

A7.3.3 Social distribution of intake of foods and nutrients

A7.3.3.1 Foods

The *1995 National Nutrition Survey* [37] showed few systematic differences in food and nutrient intake across quintiles of social disadvantage, as defined by SEIFA based on the 1991 census. Table A7.1 shows the intake of various food groups across SEIFA quintiles from the NNS 1995.

Table A7.1 Mean daily intakes (g/day)from various food groups: people aged 19 years and over, by SEIFA quintile[2]

(grams per day)

Food group	First quintile (most disadvantaged)	Second quintile	Third quintile	Fourth quintile	Fifth quintile (least disadvantaged)
Cereal & cereal products	196	222	203	217	232
Cereal-based products & dishes	113	115	130	135	136
Fruit products & dishes	126	147	141	143	156
Vegetable products & dishes	264	258	260	262	251
Legumes & pulses	9.8	7.9	10.8	9.5	10.7
Milk products & dishes	281	284	285	292	301
Meat, poultry, game	149	163	164	155	158
Fish & seafood	22	24.5	26.3	25.8	28.8
Egg products & dishes	16	15	16	13	19
Snack foods	3.2	3.0	4.2	3.3	3.9
Sugar products & dishes	20	21	20	18	17
Confectionery	7.8	9.2	8.3	9.3	9.1
Seeds & nuts	3.6	4.1	5.2	4.0	4.8
Fats & oils	12	13	13	12	11
Soup	53	62	55	48	57
Savoury sauces & condiments	30	30	28	30	29
Non-alcoholic beverages	2001	2005	1978	2007	1938
Alcoholic beverages	239	254	273	270	234

The key findings were as follows.

- Consumption of fruit and fruit products was lower (10–20%) in the most disadvantaged group compared with the other four groups, but vegetable and legume consumption showed no consistent trend across the groups. This may be difficult to interpret as potato chips were included within this category.
- Consumption of milk and milk products increased slightly with social advantage—about a 10% increase across the groups.
- Consumption of meat, poultry and game was slightly higher in the middle quintiles.
- Fish and seafood consumption increased with social advantage – this was thought to be due to better access to seafood in coastal areas where cities are located, and the higher price of seafood products compared to other foods [37]).
- Consumption of sugar products and dishes tended to decrease with social advantage.
- Consumption of cereals and cereal-based foods (for example, rice, pasta and breads) was lower in the most disadvantaged group and the middle group compared with all other groups. Consumption of cereal-based products and dishes (for example, cakes and biscuits) was about 20% lower in the two most disadvantaged groups compared with the other three [1022].

In other more recent studies, higher occupation level is associated with consumption of cheese and skim milk and higher education level is associated with consumption of cheese. There is also a significant positive relationship between skim milk consumption and occupation level based on four individual studies [1023].

A7.3.3.2 Nutrients

An assessment of energy and nutrient intakes across the SEIFA quintiles showed that energy intake increased with social advantage, as did intakes of most nutrients [1022]. However, when correcting for energy differences across groups, few differences were apparent in dietary quality, defined as nutrient intake per unit energy. Social advantage as indicated by SEIFA was positively associated with higher nutrient densities for iron, zinc, magnesium and potassium and with intake of intrinsic sugars but inversely associated with energy from fat [37].

It is unclear from the published data whether other factors, such as the age profile, differed across the quintiles of disadvantage and how much variation in factors such as age (which are known to influence total food intake) might account for the differences that were apparent (for example, in total energy intake). Physical activity may also vary across quintiles.

Neither is it clear whether these relatively small differences in nutrient profiles could explain a significant proportion of the variation in the health profiles across the groups. In interpreting the data set, however, it should be borne in mind that a relatively crude, area-based measure of social disadvantage was used. It is also possible that many of the most disadvantaged individuals in the community did not take part in the survey.

Table A7.2 Mean daily intakes of energy and nutrient densities: people aged 19 years and over, by SEIFA quintile[1]

Food group	First quintile (most disadvantaged)	Second quintile	Third quintile	Fourth quintile	Fifth quintile (least disadvantaged)
Energy (MJ)	8.82	9.18	9.37	9.31	9.45
Nutrient density (per 10MJ energy)					
Protein (g)	98.2	98.4	98.5	98.6	99.4
Fat (g)	89.8	90.7	91.1	88.9	88.8
Saturated (g)	35.7	35.5	36.0	35.2	35.0
Mono-unsaturated (g)	32.5	32.8	33.4	32.4	32.3
Polyunsaturated (g)	13.5	13.5	13.9	13.4	13.4
Cholesterol (mg)	332	331	332	319	305
Total carbohydrate (g)	276	277	272	276	277
Sugars (g)	128	125	123	124.	123
Starch (g)	147	150	148	151	152
Fibre (g)	24.4	24.4	24.9	25.2	25.6
Alcohol (g)	13.4	13.3	14.3	14.6	13.8
Vitamin A (μg)	1280	1299	1236	1218	1329
Thiamin (mg)	1.81	1.74	1.81	1.83	1.80
Riboflavin (mg)	2.27	2.18	2.24	2.25	2.22
Niacin (mg)	45.8	45.9	45.6	45.5	45.9
Folate (μg)	289	286	299	272	292
Vitamin C (mg)	132	131	130	135	142
Calcium (mg)	907	888	900	926	945
Phosphorus (mg)	1626	1631	1630	1654	1673
Magnesium (mg)	353	356	354	361	366
Iron (mg)	15.1	15.0	15.3	15.4	15.6
Zinc (mg)	12.9	13.07	12.8	13.0	13.3
Potassium (mg)	3541	3495	3507	3528	3551

Because inadequate nutritional status is part of the vicious cycle of malnutrition and infection, higher prevalence of under-nutrition in lower socio-economic groups further contributes to the incidence, severity and case fatality of childhood illnesses [871] and incidence of chronic disease in later life.

A7.4 Aboriginal and Torres Strait Islander peoples

A7.4.1. Diet-related health outcomes

Aboriginal and Torres Strait Islander people in Australia suffer significant health inequities compared with the broader community. Aboriginal and Torres Strait Islander people typically die at much younger ages and are more likely to experience ill health, disability and reduced quality of life [1020]. Poor nutrition is a major risk factor for many of the diseases with higher prevalence among Aboriginal and Torres Strait Islander groups and it has been estimated that 19% of the national Indigenous burden of disease is attributable to poor diet [1024].

Socioeconomic disadvantage underlies many of these health statistics [1025]. Compared with non-Indigenous Australians, Aboriginal and Torres Strait Islanders report lower incomes, higher rates of unemployment, lower educational attainment, more overcrowded and inadequate housing [1026], higher rates of incarceration and limited access to transport [26]. Disrupted family and community cohesion, social marginalisation, stress, lack of control over circumstances, and discrimination and racism are also apparent [30, 1025].

Overweight and obesity are common [30, 855]. Measured anthropometric data for Aboriginal and Torres Strait Islanders are unavailable, however less reliable self-reported data indicated that more than half of Aboriginal and Torres Strait Islander people aged 15 years and over were overweight or obese [1027].

Under-nutrition among young children and relatively poor growth from around six months of age persists in some parts of Australia [1028-1031].

Vitamin and mineral status has been measured infrequently in Aboriginal and Torres Strait Islander populations [30]. Multiple deficiencies have frequently been described in the same subject, suggesting the generally poor nutritional status of such individuals, rather than a specific micro-nutrient problem. In particular, vitamin status (in relation to folate, ascorbic acid and beta-carotene) consistent with low intakes of fruit and vegetables have often been described [8, 134]. More recently iodine deficiency in an Aboriginal birth cohort in the Northern Territory [1032] and low vitamin D status in a South Australian Aboriginal population [1033] have been found.

A7.4.2 Food intake, diet and nutritional status

All the available evidence suggests that, traditionally, Aboriginal and Torres Strait Islanders were fit and healthy. The traditional diet appears to have been low in energy density but high in nutrient density—high in protein, low in sugars, high in complex carbohydrate, and high in micro-nutrients. Energy expenditure appears to have been high [30, 1034-1038].

With the transition from a traditional hunter-gather lifestyle to a settled Westernised existence, Aboriginal and Torres Strait Islander people's diet has generally changed to an energy-dense diet that is high in fat and added sugars [855, 1037, 1038]. The diet is also poor in fibre and certain nutrients including folate, retinol, vitamin E and other vitamins [134].

In 2004-2005, 24% of Aboriginal and Torres Strait Islander people aged 15 years and over reported they ran out of food in the last 12 months, compared to 5% of non-Indigenous Australians [1020]. While Aboriginal and Torres Strait Islander people living in remote areas were more likely to report having run out of food in the last 12 months (36%), this figure was also high for those in non-remote areas (20%) and ranged from 18% in NSW to 45% in the Northern Territory[1020]. Women are at extra risk, partly because there is a cultural predisposition for women to feed men and children before themselves [993]. Psychological suffering due to food insecurity can exacerbate feelings of exclusion, social disruption to family life and in some cases, anxiety about possible loss of custody of children [1039].

There is very little recent dietary and nutrition data available for Aboriginal and Torres Strait Islander communities. Self-reported intakes of fruit and vegetables described in the 2004–05 NATSIHS are much higher than would be expected from more reliable, objective data [8, 134, 135, 232].

A7.4.3.1 Aboriginal people living in remote communities

Dietary intakes in remote Aboriginal communities have been consistently measured to be high in refined carbohydrates and low in fresh fruit and vegetables [134, 135, 232]. Foods with high energy density were associated with lower costs, contributing disproportionately to energy availability and limiting the capacity of people living in these communities to attain a healthy diet [135, 1040]. Food supply is an ongoing issue [1040] with people in rural and remote areas paying at least 30% more for basic nutritious foods than people living in urban and metropolitan areas [140, 1041-1046]. Basic food items are less available in the more remote stores, as are fresh vegetables and fruits and better nutritional choices [140]. The quality of dietary intake has been shown to vary in close association with the income cycle in remote Aboriginal communities [1047, 1048].

In some communities purchased food intake is supplemented by procurement of traditional foods. In the 1994 National Aboriginal and Torres Strait Islander Survey - the last time these questions were asked - 10% of respondents aged over 14 years reported spending more than one hour a week hunting or foraging for traditional foods and, of these, more than half reported spending more than five hours a week doing so [1015]. Even though the actual intake of traditional foods may be low in some areas, traditional foods are still popular and culturally important for Aboriginal and Torres Strait Islander peoples [8, 1041, 1049-1051].

A7.4.3.2. Aboriginal people living in urban areas

Only limited dietary data are available for Aboriginal and Torres Strait Islander groups in urban areas. High food costs, poor access to nutritious foods, convenience of take-away foods, budgeting issues, overcrowding, and poor knowledge and skills have been identified as barriers to healthy eating in these areas and can lead to food insecurity and overconsumption of energy-dense nutrient-poor foods and drinks [1039, 1052-1054].

A7.4.3.3 Recommendations specific to Aboriginal and Torres Strait Islander Australians

The general Australian dietary guidelines are relevant to Aboriginal and Torres Strait Islander peoples [135, 232, 1055-1058]. In particular, increased consumption of vegetables and fruits could be expected to improve the health and nutritional status of Aboriginal and Torres Strait Islander people.

Lactose intolerance after the age of three to five years may, however, be problematic in some areas or for some individuals [421, 422]. Alternative calcium sources such as chewing meat and fish bones, and consumption of small, soft fish bones (for example, in tinned salmon), and low-lactose dairy foods (such as matured cheese and yoghurt) are recommended in these cases.

Consumption of traditional bush foods should be supported wherever possible, although intake of some high saturated fat marine animal foods, such as dugong, should be limited, as was the case traditionally[1059]. In addition, there may be a problem with high levels of heavy metals in the organ meat of turtle and dugong [1060].

As with other population groups, it is important to encourage and support breastfeeding, to ensure that children and adolescents receive sufficient nutritious food to grow and develop normally, and to ensure that the growth of young children is checked regularly.

Aboriginal and Torres Strait Islander people would benefit from:

- eating traditional foods whenever possible
- when choosing store foods, choosing those most like traditional bush foods, such as fresh plant foods, wholegrain cereal foods, seafoods, and lean meat and poultry.

A7.5 Women

Where women are household heads in less advantaged urban and rural areas, there is increased risk of poverty, illiteracy and ill health [27, 28, 871].

Women are particularly subject to anaemia between puberty and menopause because of folate or iron deficiency, and after menopause to osteoporosis and breast cancer. Pregnancy and lactation

have an associated nutrition risk due to increased nutrient requirements. Maternal nutritional status is a major determinant of foetal and infant nutritional status.

A7.6 Infants and children

Children, particularly those under five, are particularly susceptible to socioeconomic inequalities that lead to marked differentials in health and nutrition. There is a clear association between the wealth of the environment the child grows up in, including socioeconomic indicators such as maternal education, and family income [871]. According to the 2005–06 NSW Population Health Survey, exclusive breastfeeding of children at six months of age was significantly lower for infants with mothers without tertiary qualifications (13%) compared with for those with tertiary qualifications (25%); mothers living in the lowest socioeconomic status (SES) areas (11%) compared with those in the highest SES areas (26%); and mothers aged younger than 25 years (9%) compared with mothers aged 25 years and over (17%) [864]. Further information is also available in the revised *Infant Feeding Guidelines for Health Workers* [133].

A7.7 Older people

The *Dietary Guidelines* are not for the frail elderly and reducing food components such as fat, salt and sugar which may make food more palatable are not always applicable to this population. Living alone, as many older adults do, has been associated with a poorer, less varied diet.[4] Older people often rely on pensions and have increasing difficulty with transport and communication, access to facilities, and preparation of food. Ill health and poor dentition can also compromise nutritional status. As the population continues to age, the demand for residential, respite and day-care services for the elderly has increased.

A7.8 People born overseas

Many migrants enjoy health that is as good as, if not better than, that of the Australian-born population [26]. This could be partly because migrants are selected for their health status, or because, in some cases, they are less likely to be exposed to risk factors for non-communicable disease before they arrive in Australia. However there is a small proportion of the migrant population, such as refugees, who experience poorer health than other Australians due to socioeconomic and political factors [26].

[4] Note that the *Guidelines* do not apply for the frail elderly or for residents of aged care institutions.

Mortality rates for people born overseas are generally lower than for people born in Australia, but the causes of mortality differ depending on country of birth, with some migrants experiencing higher mortality rates for particular conditions than Australian-born people. The prevalence of diet-related diseases also varies in different migrant groups. For example, diabetes is more prevalent among those born in Germany, Greece, India, Italy, Lebanon and Poland, and coronary heart disease is more prevalent among those born in Poland [26].

The prevalence of health risk factors also varies depending on country of birth. People from South-East Asia are less likely than Australian-born people to smoke, drink alcohol at risky or high-risk levels and be overweight or obese. In contrast, people from Oceania, United Kingdom and southern and eastern Europe are more likely to be overweight or obese than those born in Australia [26].

The health of migrants approaches the health of the local population over time, as migrants' habits and lifestyles move towards those of the wider Australian community. Food habits may change out of choice, because of the limited availability of traditional and familiar foods, or because of change in economic circumstances in Australia. Similarly, financial and language difficulties may affect access to education and employment opportunities which then affects income, health and nutrition literacy, and access to nutritious foods. Some migrants experience disadvantages such as social isolation and poor housing, which can affect access to safe food and safe preparation of food, and are generally in a relatively vulnerable position in their new environments, regardless of the type of migration [871].

A7.9 Rural and remote Australians

Mortality rates increase as remoteness increases [26]. People living outside major cities are more likely to have high blood pressure, high cholesterol and report drinking at risky or high-risk levels. They were also more likely to be classified as overweight or obese. However rates of some cancers decrease slightly with remoteness [26].

Underlying factors contributing to increased health risk include the lower levels of education, income and employment of many rural communities, occupation risks from farm or mining work, greater levels of smoking and alcohol abuse, less access to health services and staff, and the hazards of driving over long road distances. Among specific dietary factors, people living outside major cities were less likely to eat too few vegetables but more likely to have insufficient fruit intake [26].

In Australia, the cost of a basket of nutritious foods has been consistently found to be at least 30% higher in remote areas compared to capital cities [140, 1031, 1041, 1043-1046]. Among the most remote communities, costs are highest in those areas greater than 2000km from capital cities, suggesting prices are influenced markedly by transport costs [140].

The availability of nutritious foods is limited in regional and remote areas in northern Australia [140, 1031, 1041, 1043-1046]. But even in south west Victoria, people living in more remote towns without a major supermarket faced limited availability of healthy food basket items [1019]. In NSW, the variety of fruit and vegetables available also decreases with remoteness and also with decreasing socioeconomic status of the community [1061].

Appendix 8: Glossary

Adequate Intake (AI)

The average daily nutrient intake level based on observed or experimentally-determined approximations or estimates of nutrient intake by a group (or groups) of apparently healthy people that are assumed to be adequate. An AI is set when there is inadequate evidence to support setting an RDI.

Adolescents

For the purposes of these *Guidelines*, an adolescent is someone aged 12 to 18 years. A marked increase in the rate of growth and development during adolescence increases the need for most nutritents including energy, protein, vitamins and minerals.

Allium vegetables

Vegetables derived from a bulbous plant having an onion odour, including garlic, leeks, shallots, chives and onion.

Anaemia

There are several forms of anaemia. Microcytic anaemia (referring to small red blood cells) is a deficiency of red blood cells or their haemoglobin, often, but not always, related to iron deficiency. Macrocytic anaemia (referring to large red blood cells) is prevalent in some groups (such as Aboriginal and Torres Strait Islander people) and may in some cases be associated with deficiencies of other nutrients, especially folate and vitamin B12.

Body mass

Body mass and *body weight* are often used interchangeably to describe the weight of a person's body.

Body mass index (BMI)

An index calculated by dividing the weight of an individual (in kilograms) by the square of their height (in metres), BMI is a simple estimate of the body fatness of a human being who does not have abnormal physical characteristics. The World Health Organization and the US National

Institutes of Health have recommended that an operational definition of overweight be a BMI of at least 25kg/m^2 and obesity as a BMI of at least 30kg/m^2.

A large number of anthropometric measurements and indices have been proposed for assessing and monitoring levels of obesity. Methods used in research studies to measure the percentage of body fat are not practical for regular clinical and community use.

Table A8.1 Body mass index classification (WHO)

Classification (WHO)	BMI (kg/m^2)
Underweight	< 18.5
Normal range	18.5 – 24.9
Pre-obese (overweight)	25.0 – 29.9
Obese class I	30.0 – 34.9
Obese class II	35.0– 39.9
Obese class III	> 40.0

Body weight

See body mass.

Brassica vegetables

Vegetables from the brassica or crucifer family, collectively known as cabbages or mustards and including broccoli, cabbage and brussel sprouts.

Breads

Refers to leavened and unleavened wholemeal, white, mixed-grain, rye and fruit breads, as well as rolls, bagels, English muffins, crispbreads, crumpets and low-fat crackers.

Carbohydrates

Carbohydrates are polyhydroxy aldehydes, ketones, alcohols, acids, their simple derivatives, and their polymers with linkages of the acetal type. They can be classified according to their degree of polymerisation and can be divided initially into three principal groups—sugars, oligosaccharides and polysaccharides. Carbohydrates are the least concentrated form of energy providing 16 kilojoules per gram.

Cereals

Refers to the entire class of cereal/grain foods, including whole or partially processed cereal grains (for example, rice, oats, corn and barley), breads, cereals, rice, pasta, noodles, polenta, couscous, oats, quinoa and barley. It excludes cereal or grain-based products with a significant amount of added fat and sugar, such as cakes, pastries, and biscuits.

Children

For the purposes of these *Guidelines*, children are defined as toddlers aged 1 to 2 years, preschoolers aged 3 to 5 years and primary school age 6 to 11 years.

It is important for children to receive a nutritious diet which includes all the nutrients they need to grow and develop normally.

Cholesterol

Cholesterol, chemically a sterol, occurs in all the cell membranes of land animals. Brains and egg yolks are very rich in it, oils and fats from plants never contain it. Eating cholesterol does not 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.

Complementary foods

Any food—manufactured or locally prepared—that is suitable as a complement to breastmilk or infant formula when either becomes insufficient to satisfy an infant's nutritional requirements.

Core food groups

This was a concept of the previous modeling system and included foods that formed the basis of a healthy diet, based on or developed with reference to Recommended Daily Intakes (RDIs) and the *Guidelines*.

Cruciferous vegetables

See brassica vegetables.

Dairy Food

See milks, yoghurts and cheeses.

Discretionary foods

This includes foods and drinks not necessary to provide the nutrients the body needs, but may add variety. However, many of these are high in saturated fats, sugars, salt and/or alcohol, and are therefore described as energy dense. They can be included sometimes in small amounts by those who are physically active, but are not a necessary part of the diet.

Foods in this category include cakes, biscuits; confectionary, chocolate; pastries, pies; ice confections, butter, cream, and spreads which contain predominantly saturated fats; potato chips, crisps and other fatty or salty snack foods; sugar-sweetened soft drinks and cordials, sports and energy drinks and alcoholic drinks.

Estimated Average Requirement (EAR)

A daily nutrient level estimated to meet the requirements of half the healthy individuals in a particular life stage and gender group.

Energy Expenditure

Total daily energy expenditure includes energy expended in physical activity and resting energy expenditure (basal metabolic rate plus necessary tissue repair and the thermic effect of food) over a 24-hour period.

Exclusive breastfeeding

Means an infant is receiving only breastmilk, which includes expressed breastmilk and milk from a wet nurse. The infant might also receive medications and vitamins or minerals as required.

Fats

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. Differences between fats are largely a consequence of the fatty acids they contain, which together make up 90% of the weight of the molecule. Fats in the diet can be 'visible' or 'invisible'. Among visible fats are butter, margarine, cooking oils, and the fat on meat. Invisible fats occur in foods such as cheese, sauces, mayonnaise, 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, providing 37 kilojoules 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 as part of all cell membranes, part of the myelin in the brain and nervous system, and the starting material for synthesis in the body of bile acids and adrenocortical and sex hormones. Cholesterol can, however, accumulate in blood and in the inner walls of arteries, leading to disease.

Food variety

Refers to foods that are biologically diverse or nutritionally distinct from each other. Eating a variety of nutritious foods means consuming different food types in appropriate amounts to attain all the required nutrients without excess energy intake. Variety further refers to choosing a range of items from within each food group, particularly within the plant-based groups (vegetables, fruits and cereals). Variety is an important nutritional principle which, in modern sedentary society, requires a reduction in serving sizes, particularly of more energy-dense foods with limited nutrient content.

Foundation Diet

The Foundation Diet was informed by current scientific evidence derived from the literature, the most current national intake data and the NHMRC's 2006 Nutrient Reference Values. The diets were modelled to provide as close to 100% of the RDIs of 10 key nutrients as was feasible and to provide the estimated energy requirements of the smallest and very sedentary category (PAL 1.4) for each age and gender group. These Foundation Diets based on low energy requirements were then tested using 100 7-day simulations with the aim that all of the simulations would meet the EARs of the 10 key nutrients.

Fruit

Generally applies to the sweet, fleshy, edible portion of a plant that arises from the base of the flower and surrounds the seeds. Examples include pome fruit such as apples and pears, citrus fruit such as oranges and lemons, stone fruit such as apricots and plums, and berries.

Fruit juice

100% fruit juice, including pulp, is a good source of vitamins such as vitamin C and folate and also provides fibre and carbohydrates, particularly natural sugars. Whole fruit is preferable to fruit juice however the occasional use of fruit juice may assist with nutrient intake when fresh, frozen or tinned fruit supply is sub-optimal. Fruit juice is energy-dense and if consumed in excess, it can displace other nutritious foods from the diet and may lead to problems such as obesity.

Frail elderly

For the purposes of these Guidelines, the frail elderly are defined as older persons (usually over the age of 75 years) with a physical or mental disability that may interfere with their ability to perform activities of daily living independently.

Grain foods

See cereals.

Infant

For the purposes of these *Guidelines*, infants are defined as children under the age of 12 months.

Iron deficiency

Refers to a condition of low body iron, which may manifest itself as low serum iron, low serum ferritin, high serum iron–binding capacity, a reduced transferrin saturation index and/or high–free erythrocyte protoporphyrin. It can cause fatigue, listlessness and pallor and may progress to anaemia. It can also have widespread non-haematological effects on behaviour, cognition and motor development, physical work performance, and body temperature regulation. In Australia, iron deficiency appears to be a condition predominantly seen in young women.

Legume/Beans

Refers to all forms of edible beans and peas and preparations made from them—dried legumes, legume flour, bean curd, canned legumes, cooked legumes. Amongst the better known legumes include butter beans, haricot (navy) beans, red kidney beans, soybeans, mung beans, lentils, chick peas, snow peas and various other types of fresh green peas and beans. Legumes are usually cooked because this increases their nutritional value and improves their taste, but are occasionally eaten raw (for example, snow peas). Legumes are technically a specialised form of fruit (the pod surrounds the seeds and arises from the base of the flower) but because the main food material in legumes is generally the seeds rather than the flesh surrounding the seeds they are categorised separately.

Limit

Limit is used to emphasise the importance of limiting intake of foods and drinks high in saturated and trans fats, added salt, added sugars and alcohol, due to evidence that these foods are associated with increased risk of obesity and/or chronic disease, including cardiovascular disease, type 2 diabetes and/or some cancers.

Low fat food

For labelling purposes, foods which claim to be 'low fat' must meet criteria before a manufacturer is allowed to print this on the food label. A 'low fat' or 'low in fat' product must contain no more than 3g of fat per 100g of food. A liquid must contain no more than 1.5g of fat per 100g of liquid.

Low salt food

For labelling purposes, a low salt food as one with a sodium concentration of up to 120 milligrams per 100 grams (Australia New Zealand Food Standards Code, clause 17 of Standard 1.2.8). The following are the conversion factors for the units used to express the sodium content of food:

- 1 mmol = 23 milligrams
- 1 gram = 43 mmol

One gram of sodium chloride (NaCl) contains 17 mmol, or 391 milligrams, of sodium.

Meat

Refers to all or part of the carcass of any cattle, sheep, goat, buffalo, kangaroo, camel, deer, goat, pig or rabbit. For the purpose of the *Guidelines* refers to the muscle component only, excluding

offal such as liver and kidney.

Meat alternatives

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

Milks, yoghurts and cheeses

Generally refers to cow's milk and the yoghurt and cheese produced from it but can also include milks, yoghurts and cheeses from goat and sheep milks.

Milk, yoghurt and cheese alternatives

Inclusion in this 'alternative' category is based primarily on calcium content, although most of the alternatives also provide substantial amounts of protein. Calcium fortified grain-based beverages, fish whose bones are eaten (such as sardines), and some nuts (such as almonds), contain moderate to good amounts of calcium and protein and in this respect can be considered as alternatives.

Monounsaturated fatty acids

In chemical terms, monounsaturated fatty acids (MUFA) contain one unsaturated bond. MUFA occurs in considerable amounts in olive oil, canola oil and many kinds of nuts.

Nutrient Reference Values (NRVs)

Amounts of nutrients required on an average daily basis for adequate physiological function and prevention of deficiency disease (EAR, AI or RDI) or chronic disease prevention (Acceptable Macronutrient Distribution Range (AMDR) or Suggested Dietary Target (SDT)). For each nutrient, an Upper Level of Intake (UL) was also set to specify the highest average daily nutrient intake likely to pose no adverse health effects to almost all individuals in the general population.

Nutritious foods

Refers to foods that make a substantial contribution towards providing a range of nutrients, have an appropriate nutrient density, and are compatible with the overall aims of the *Guidelines*.

Older adults

For the purposes of these Guidelines, older adults are defined as healthy people aged 65 years and over, not including the frail elderly.

Osteoporosis

Osteoporosis, a condition of low bone mass, can lead to bone fragility and increased risk of fractures. Most fractures in older adults are related to osteoporosis whereas trauma is the primary cause of fractures in young adults. Clinically, individuals are considered osteoporotic if their bone mineral density is 2.5 SD or more below the young adult mean. This criterion identifies about 30% of all post-menopausal women as having osteoporosis. Of these, more than 50% will have suffered a previous fracture. Osteoporosis is also of growing importance in men.

Pasta and noodles

Includes a wide range of Italian and Asian products based on sheets of dough made from flours—usually wheat or rice flour—and water, sometimes with egg added. Examples are plain spaghetti, lasagne, fettuccine, udon and Hokkien noodles, rice paper and wonton wrappers. The term excludes some instant noodles and flavoured pasta mixes with significant amounts of added fat and salt.

Physical activity

Any structured or incidental body movement (light, moderate or vigorous) which causes your muscles to work and uses more energy than you would use if you where resting (see Chapter 4).

Physical inactivity

Physical inactivity (or sedentary behaviour) is defined as a state in which body movement is minimal, such as sitting time while watching television, reading, working at a computer, talking on the telephone, driving a car, or meditating.

Phytochemicals

Substances found in plant materials which may confer some health benefits and which include a number of chemical categories such as carotenoids, flavonoids and isoflavonoids, polyphenols, isothiocyanates, indoles, sulphoraphane, monoterpenes, xanthin, and non-digestible oligosaccharides.

Plenty

Plenty is used judiciously to encourage increased consumption of vegetables (particularly non-starchy varieties).

Polyunsaturated fatty acids

Polyunsaturated fatty acids (PUFA) contain two or more (poly) double (unsaturated) bonds. Foods with a high PUFA content are liquid at room temperature that is, they tend to be oils'. The most common polyunsaturated fatty acid is linoleic acid (18:2) whose double bonds are in the n-6 position. It occurs in seed oils including sunflower oil, safflower oil and corn oil. Smaller amounts of polyunsaturated fatty acids with double bonds in the n-3 position also occur in the diet. The best known are those in fatty fish, their names abbreviated to EPA (20:5 eicosapentaenoic) and DHA (22:6 docosahexaenoic). Another n-3 polyunsaturated fatty acid, ALA (18:3 alpha linolenic), occurs in considerable amounts in canola and flaxseed oils and in walnuts.

Poultry

Refers to chicken, duck, turkey and all other avian foods except eggs.

Quinoa

See cereals or grain foods.

Recommended Dietary Intake (RDI)

The average daily dietary intake level that is sufficient to meet the nutrient requirements of nearly all (97–98 per cent) healthy individuals in a particular life stage and gender group.

Red meat

The muscle meat from cattle, sheep, pig, goat and kangaroo . Note that although pork is not considered red meat for marketing purposes in Australia, it is classified as red meat in the international literature, and has been treated as red meat for the purpose of the *Guidelines.*

Reduced fat products

For a food to be labelled 'reduced fat', it must contain at least 25% less fat than is present in the equivalent full-fat product, and at least 3g less fat per 100g of food.

Salt

Dietary salt is an inorganic compound consisting of sodium and chloride ions. It is found naturally in many foods, but it is also added to many foods because of its preservative and flavouring characteristics. Research has shown that both the sodium and the chloride can be detrimental to health when consumed in excess. About 90% of all the sodium added to food is sodium chloride, so dietary intake of sodium approximates intake of sodium chloride for practical purposes. Sodium in the diet of Australian adults comes mostly from processed foods, although sodium added in cooking, at the table, in medications and naturally present in foods can contribute to the total dietary intake.

Australian adults are recommended to limit their intake of sodium to less than 2300 mg per day. This is equivalent to about six grams of salt, or one and a half teaspoons.

Saturated fatty acids

In chemical terms, saturated fatty acids (SFA) contain no double bond - that is, they are fully saturated with hydrogen. Foods which predominantly comprise SFA are usually solid at room (e.g. butter, fat on meat). SFA are the main type of fatty acid in milk, cream, butter and cheese, in some meats (most of the land animal fats), and can also be found in considerable amounts in some oils such as in palm and coconut oil. When the saturated fatty acids palmitic (16:0), myristic (14:0) and lauric (12:0) predominate in the diet they tend to raise plasma cholesterol.

Sodium

See salt.

Standard drink

A standard drink contains 10 grams of alcohol—equivalent to 12.5 millilitres of alcohol.

Sugars

Conventionally used to describe monosaccharides and disaccharides such as sucrose, glucose and fructose which can be found naturally in foods or can be added to in processing. *Sugars* is the term used in the analysis of the *1995 National Nutrition Survey*. *Sugar*, by contrast, is commonly used to describe purified sucrose, as are the terms *refined sugar* and *added sugar*. *Added sugars* may also include other sugars such as glucose, fructose and corn syrup.

Total Diet

Progression from *Foundation Diets* to *Total Diets* can occur when total energy needs are greater than the energy provided by a *Foundation Diet* for a particular age and sex group. General principles were determined to ensure that diets remained within acceptable limits for percentage of energy from fat and the various fat components, protein and carbohydrate (AMDRs), the Upper Levels (ULs) and Suggested Dietary Targets (SDTs) for chronic disease prevention. The principles allow free addition of vegetables (including legumes), fruits, nuts and seeds, and cereal foods. The principles also encourage a variety of choice of additional foods while defining the choices allowed in the modelling for the meat, milk, yoghurt and cheese products and unsaturated margarines and oils categories. 'Discretionary choices' can be included but it is important to note that they do not need to be included in the diet, and *Total Diets* without inclusion of any 'Discretionary choices' were also modelled for all age and sex groups.

Trans-fatty acids

Trans-fatty acids are a form of unsaturated fatty acid that is straight at a double bond rather than bent, as in the usual cis form. They are not common in nature but are formed during some manufacturing processes, such as when edible oils are hydrogenated to make hard margarines. Small amounts of trans-fatty acids occur naturally in meats and dairy foods.

Upper level of intake (UL)

The highest average daily nutrient intake level likely to pose no adverse health effects to almost all individuals in the general population. As intake increases above the ULUpper level of intake, the potential risk of adverse effects increases.

Vegetables

Applies to leafy green vegetables (for example, spinach, lettuce, silverbeet and bok choi), members of the crucifer or brassica family (for example, broccoli, cabbage, and brussels sprouts), starchy root and tuber vegetables (for example, yams and potatoes), edible plant stems (for example,

celery and asparagus), gourd vegetables (for example, pumpkin and cucumber), allium vegetables (for example, onions, garlic and shallots), and sweet corn.

Wholegrain

This term applies to products which uses every part of the grain including the outer layers, bran and germ even if these parts are separated during processing and regardless of whether the grain is in one piece or milled into smaller pieces.

The term wholegrain may apply to whole and intact grains as found in some bread and crisp breads, puffed or flaked grains in some breakfast cereals, coarsely milled or kibbled wheat found in breads such as pumpernickel and ground grains such whole wheat flour used to make wholemeal bread.

Appendix 9: Summary of omitted evidence statements

A number of relationships between food consumption and disease outcomes have not been considered or presented in the Australian Dietary Guidelines, even though these relationships were investigated as part of the systematic literature review process. However, these relationships and the reviewed evidence can still be found in the Evidence Report. A summary of the relationships that have been omitted, with reasoning, are provided below.

A9.1 Grade C Evidence Statements: Food consumption with no association with a health outcome

To avoid unnecessary confusion for the reader, Grade C evidence statements suggesting no association between a food and specific health outcome were not included in the evidence summary tables at the beginning of each section within the document. These evidence statements have been provided in Table A9.1.

Please note that these evidence statements are discussed in further detail within the body of the document.

Table A9.1: Grade C 'no association' relationships that informed the Australian Dietary Guidelines

Evidence Statement	Grade
Vegetables	
Consumption of vegetables is not associated with reduced risk of type 2 diabetes.	C
Consumption of vegetables is not associated with reduced risk of oesophageal cancer.	C
Consumption of vegetables is not associated with reduced risk of ovarian cancer.	C
Consumption of vegetables is not associated with reduced risk of endometrial cancer	C
Consumption of cruciferous vegetables, carrots, potatoes and beans and lentils is not associated with risk of colorectal cancer.	C

Evidence Statement	Grade
Fruit	
Consumption of fruit is not associated with risk of type 2 diabetes.	C
Consumption of fruit is not associated with risk of breast cancer.	C
Consumption of fruit is not associated with risk of ovarian cancer.	C
Consumption of fruit is not associated with risk of endometrial cancer.	C
Consumption of fruit is not associated with risk of colorectal cancer.	C
Consumption of fresh red meat 1 to 6 times per week (or an intake range of 14-70 g/1000 Calories/d) is not associated with risk of bladder cancer.	C
Lean meat and poultry, fish, eggs, legumes/beans and nuts/seeds	
Consumption of fresh red meat, irrespective of frequency or serving size, is not associated with risk of prostate cancer	C
Consumption of 30-200grams of fresh red meat per day is not associated with risk of pancreatic cancer.	C
Consumption of at least 1 serve of fish a week is not associated with reduced risk of depression.	C
Consumption of eggs daily is not associated with increased risk of coronary heart disease.	C
Consumption of nuts (65-110 g / day) does not lead to weight gain in the short-term.	C
Milk, yoghurt, cheese and/or alternatives	
Consuming dairy food is not associated with risk of endometrial cancer	C
Consumption of dairy foods is not associated with weight change or risk of obesity.	C
Consumption of milk is not associated with BMI or BMI change in childhood.	C
Mean consumption of 1 serving of dairy food per day is not associated with the risk of breast cancer.	C
Consumption of less than 1 serving of milk per day during adult life is not associated with risk of osteoporotic or hip fracture	C

Evidence Statement	Grade
Water	
Consumption of black tea is not associated with risk of cardiovascular disease.	C
Consumption of coffee is not associated with risk of coronary heart disease.	C
Consumption of coffee is not associated with risk of breast cancer.	C
Consumption of coffee is not associated with risk of colorectal cancer.	C
Consumption of coffee is not associated with risk of ovarian cancer.	C
Consumption of green or black tea is not associated with risk of ovarian cancer.	C
Consumption of green or black tea is not associated with risk of colorectal cancer.	C

A9.2 Grade D Evidence Statements that did not inform the Australian Dietary Guidelines

In the systematic literature review (the Evidence Report), a number of food, diet, and health relationships were examined for which the evidence of an association was D-grade level. This was because the evidence was limited, inconclusive or contradictory. These D-grade relationships were not used to inform the development of the *Australian Dietary Guidelines*. Table A9.2 provides a list of the evidence statements for these relationships.

Grade D evidence can inform health professionals about the strength of evidence from recent research, particularly in emerging areas, and help identify areas where further research on dietary patterns and health outcomes may be required. D-grade evidence can also assist health professionals correct diet related misconceptions among the general population. *For example*, members of the general population may be inclined to alter their dietary patterns when the results of a new study are widely publicised in the media.

This evidence, while useful as mentioned above, was not used in the development of *Guideline* statements.

Table A9.2: Evidence statements (Grade D) that did not inform the Australian Dietary Guidelines

Evidence Statement	Grade
Vegetables	
The effect of total vegetable consumption on gastric (stomach) cancer is inconclusive (Section 2.5 in Evidence Report)	D
The effect of total vegetable consumption on the risk of breast cancer is inconclusive (Section 2.6 in Evidence Report)	D
The effect of total vegetable consumption on the risk of colorectal cancer is inconclusive (Section 2.8 in Evidence Report)	D
The effect of total vegetable consumption on the risk of lung cancer is inconclusive (Grade D, Section 2.7 in Evidence Report	D
Fruit	
The effect of fruit consumption on the risk of oesophageal cancer is inconclusive (Section 1.9 in Evidence Report)	D
The effect of fruit consumption on the risk of gastric cancer is inconclusive (Section 1.5 in Evidence Report)	D
There is limited evidence that fruit consumption reduces the risk of lung cancer (Section 1.7 in Evidence Report)	D
The effect of citrus fruit consumption on the risk of pancreatic cancer is inconclusive (Section 1.13 in Evidence Report)	D
Grain (cereal) foods	
The effect of grain (cereal) consumption on the risk of cancer is inconclusive (Section 6.1 in Evidence Report)	D
Lean meat and poultry, fish, eggs, legumes/beans and nuts/seeds	
The consumption of unprocessed red meat is associated with an increased risk of lung cancer (Section 4.5 in Evidence Report)	D
There is limited evidence showing no association between the consumption of red meat and the risk of breast cancer (Section 4.4 in Evidence Report)	D
The effect of consuming poultry at least once a week on the risk of breast	D

Evidence Statement	Grade
cancer is inconclusive (Section 10.1 in Evidence Report)	
The effect of consuming poultry at least once a week on the risk of colorectal cancer is inconclusive (Section 10.2 in Evidence Report)	D
The effect of fish consumption on the risk of breast cancer is inconclusive (Section 9.6 in Evidence Report)	D
The effect of fish consumption on the risk of colorectal cancer is inconclusive (Section 9.7 in Evidence Report)	D
The effect of fish consumption on the risk of prostate cancer is inconclusive (Section 9.8 in Evidence Report)	D
The effect of fish consumption on the risk of renal cell cancer is inconclusive (Section 9.9 in Evidence Report)	D
The effect of egg consumption on the risk of cancer is inconclusive (Section 11.2 in Evidence Report)	D
Consumption of legumes (especially soy foods) is associated with a reduced risk of breast cancer (Section 7.1 in Evidence Report)	D
There unlikely to be a significant protective effect against prostate cancer from consuming soy foods (Section 7.2 in Evidence Report)	D
Milk, yoghurt, cheese and/or alternatives	
The effect of milk consumption on the risk of prostate cancer is inconclusive (Section 5.14 in Evidence Report)	D
Water	
The effect of coffee consumption on the risk of increased systolic blood pressure is inconclusive (Section 15.14 in Evidence Report)	D
The effect of consuming four cups of coffee a day on the risk of gastric cancer is inconclusive (Section 15.5 in Evidence Report)	D
Green and black tea consumption is not associated with an increased risk of breast cancer (Sections 15.16, 15.17, 15.22 in Evidence Report)	D

References

1. Rayner, M. and P. Scarborough, *The burden of food related ill health in the UK.* J Epidemiol Community Health, 2005. **59**(12): p. 1054-7.

2. National Public Health Partnership, *Eat Well Australia: an agenda for action for public health nutrition, 2000-2010*, 2001, National Public Health Partnership: Canberra.

3. Australian Institute of Health and Welfare, *Premature mortality from chronic disease. AIHW Bulletin no. 84. Cat. no. AUS 133*, 2010, Australian Institute of Health and Welfare: Canberra.

4. Queensland Health, *The health of Queenslanders 2006. Report of the Chief Health Officer, Queensland*, 2006, Queensland Health: Brisbane

5. Crowley, S., et al., eds. *The cost of diet-related disease in Australia: a discussion paper*. 1992, Australian Institute of Health and Welfare: Canberra.

6. Access Economics, *The growing cost of obesity in 2008: three years on*, 2008, Diabetes Australia, Access Economics: Canberra.

7. National Preventative Health Taskforce, *Australia: the healthiest country by 2020 – National Preventative Health Strategy – the roadmap for action*, 2009a, Commonwealth of Australia: Canberra.

8. Lee, A.J., et al., *Survival tucker: improved diet and health indicators in an aboriginal community.* Aust J Public Health, 1994. **18**(3): p. 277-85.

9. National Health and Medical Research Council, Australian Government Department of Health and Ageing, and New Zealand Ministry of Health, *Nutrient reference values for Australia and New Zealand including recommended dietary intakes*, 2006, Commonwealth of Australia: Canberra.

10. National Health and Medical Research Council, *A modelling system to inform the revision of the Australian Guide to Healthy Eating*, 2011a, Commonwealth of Australia: Canberra.

11. Begg, S., et al., *The burden of disease and injury in Australia 2003*, 2007, Australian Institute of Health and Welfare: Canberra.

12. Australian Bureau of Statistics, *National Health Survey: Summary of Results, 2007-2008. Cat. no. 4364.0*, 2009, Australian Bureau of Statistics: Canberra.

13. Commonwealth Scientific and Industrial Research Organisation, Preventative Health National Research Flagship, and University of South Australia, *2007 Australian National*

Children's Nutrition and Physical Activity Survey - Main Findings, 2008, Commonwealth of Australia: Canberra.

14. National Health and Medical Research Council, *A review of the evidence to address targeted questions to inform the revision of the Australian dietary guidelines*, 2011b, Commonwealth of Australia: Canberra.

15. Buckland, G., A. Bach, and L. Serra-Majem, *Obesity and the Mediterranean diet: a systematic review of observational and intervention studies.* Obes Rev, 2008. **9**(6): p. 582-93.

16. Roman, B., et al., *Effectiveness of the Mediterranean diet in the elderly.* Clin Interv Aging, 2008. **3**(1): p. 97-109.

17. Serra-Majem, L., B. Roman, and R. Estruch, *Scientific evidence of interventions using the Mediterranean diet: a systematic review.* Nutr Rev, 2006. **64**(2): p. S27.

18. Bamia, C., et al., *Dietary patterns and survival of older Europeans: the EPIC-Elderly Study (European Prospective Investigation into Cancer and Nutrition).* Public Health Nutr, 2007. **10**(6): p. 590-8.

19. Diehr, P., et al., *The effect of different public health interventions on longevity, morbidity, and years of healthy life.* BMC Public Health, 2007. **7**: p. 52.

20. Kant, A.K., B.I. Graubard, and A. Schatzkin, *Dietary patterns predict mortality in a national cohort: the National Health Interview Surveys, 1987 and 1992.* J Nutr, 2004. **134**(7): p. 1793-9.

21. Togo, P., et al., *Food intake patterns and body mass index in observational studies.* Int J Obes Relat Metab Disord, 2001. **25**(12): p. 1741-51.

22. Wirt, A. and C.E. Collins, *Diet quality--what is it and does it matter?* Public Health Nutr, 2009. **12**(12): p. 2473-92.

23. Anderson, A.L., et al., *Dietary patterns and survival of older adults.* J Am Diet Assoc, 2011. **111**(1): p. 84-91.

24. de Groot, C.P. and W.A. van Staveren, *Nutritional concerns, health and survival in old age.* Biogerontology, 2010. **11**(5): p. 597-602.

25. Scarborough, P., et al., *Modelling the impact of a healthy diet on cardiovascular disease and cancer mortality.* J Epidemiol Community Health, 2010.

26. Australian Institute of Health and Welfare, *Australia's health 2010. Australia's health series no. 12. Cat. no. AUS 122*, 2010, Australian Institute of Health and Welfare: Canberra.

27. Wilkinson, R. and M. Marmot, *Social determinants of health: the solid facts*, 2003, World Health Organization: Copenhagen.

28. Marmot, M.G., *The status syndrome: how social standing affects our health and longevity* 2004, New York: Henry Holt.

29. Friel, S. and P.I. Baker, *Equity, food security and health equity in the Asia Pacific region.* Asia Pac J Clin Nutr, 2009. **18**(4): p. 620-32.

30. National Health and Medical Research Council, *Nutrition in Aboriginal and Torres Strait Islander peoples: an information paper*, 2000, Commonwealth of Australia: Canberra.

31. Australian Bureau of Statistics and Australian Institute of Health and Welfare, *The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples. ABS Cat. no. 4704.0. AIHW Cat. no. IHW14*, 2005, Commonwealth of Australia: Canberra.

32. Australian Bureau of Statistics, *National Aboriginal and Torres Strait Islander Health Survey 2004-2005*, 2006, Australian Bureau of Statistics: Canberra.

33. Commonwealth Department of Health and Family Services. *Food and nutrition policy*. 1992; Available from: http://www.health.gov.au/internet/main/publishing.nsf/content/phd-nutrition-fnp-1992.

34. National Health and Medical Research Council, *How to review the evidence: systematic identification and review of the scientific literature*, 2000a, Commonwealth of Australia: Canberra.

35. National Health and Medical Research Council, *Dietary Guidelines for Older Australians*, 1999, Commonwealth of Australia: Canberra.

36. National Health and Medical Research Council, *Dietary Guidelines for Children and Adolescents in Australia*, 2003, Commonwealth of Australia: Canberra.

37. National Health and Medical Research Council, *Dietary Guidelines for Australian Adults*, 2003, Commonwealth of Australia: Canberra.

38. National Health and Medical Research Council, *NHMRC additional levels of evidence and grades for recommendations for developers of guidelines*, 2007, Commonwealth of Australia: Canberra.

39. National Health and Medical Research Council, *How to use the evidence: assessment and application of scientific evidence*, 2000b, Commonwealth of Australia: Canberra.

40. National Health and Medical Research Council, *NHMRC standards and procedures for externally developed guidelines*, 2007, Commonwealth of Australia: Canberra.

41. National Health and Medical Research Council, *NHMRC levels of evidence and grades for recommendations for developers of guidelines*, 2009, Commonwealth of Australia: Canberra.

42. World Cancer Research Fund and American Institute for Cancer Research, *Food, nutrition, physical activity, and the prevention of cancer: a global perspective.*, 2007: Washington DC.

43. Ball, K., et al., *How well do Australian women comply with dietary guidelines?* Public Health Nutr, 2004. **7**(3): p. 443-52.

44. Australian Bureau of Statistics, *National Nutrition Survey: Foods Eaten, Australia 1995.*, 1999, Australian Bureau of Statistics: Canberra.

45. Commonwealth Department of Health and Family Services, *The Australian Guide to Healthy Eating*, 1998, Commonwealth of Australia: Canberra.

46. Rangan, A.M., et al., *Consumption of 'extra' foods by Australian children: types, quantities and contribution to energy and nutrient intakes.* Eur J Clin Nutr, 2008. **62**(3): p. 356-64.

47. Australian Bureau of Statistics, *National Nutrition Survey: Nutrient intakes and physical measurements, Australia 1995*, 1998, Australian Bureau of Statistics: Canberra.

48. Cook, T., I. Rutishauser, and M. Seelig, *Comparable data on food and nutrient intake and physical measurements from the 1983, 1985 and 1995 national nutrition surveys*, 2001, Commonwealth of Australia: Canberra.

49. Burke, V., et al., *Predictors of body mass index and associations with cardiovascular risk factors in Australian children: a prospective cohort study.* Int J Obes (Lond), 2005. **29**(1): p. 15-23.

50. Classen, T. and C. Hokayem, *Childhood influences on youth obesity.* Econ Hum Biol, 2005. **3**(2): p. 165-87.

51. Dubois, L., et al., *Family food insufficiency is related to overweight among preschoolers.* Soc Sci Med, 2006. **63**(6): p. 1503-16.

52. Dubois, L. and M. Girard, *Early determinants of overweight at 4.5 years in a population-based longitudinal study.* Int J Obesity, 2006. **30**(4): p. 610-7.

53. Field, A.E., et al., *Snack food intake does not predict weight change among children and adolescents.* Int J Obes Relat Metab Disord, 2004. **28**(10): p. 1210-6.

54. Hawkins, S.S., T.J. Cole, and C. Law, *An ecological systems approach to examining risk factors for early childhood overweight: findings from the UK Millennium Cohort Study.* J Epidemiol Community Health, 2009. **63**(2): p. 147-55.

55. Hesketh, K., et al., *Predictors of body mass index change in Australian primary school children.* Int J Pediatr Obes, 2009. **4**(1): p. 45-53.

56. Hesketh, K., et al., *Stability of body mass index in Australian children: a prospective cohort study across the middle childhood years.* Public Health Nutr, 2004. **7**(2): p. 303-9.

57. Li, L., et al., *Intergenerational influences on childhood body mass index: the effect of parental body mass index trajectories.* Am J Clin Nutr, 2009. **89**(2): p. 551-7.

58. Mamun, A.A., et al., *Family and early life factors associated with changes in overweight status between ages 5 and 14 years: findings from the Mater University Study of Pregnancy and its outcomes.* Int J Obes (Lond), 2005. **29**(5): p. 475-82.

59. Power, C., et al., *Combination of low birth weight and high adult body mass index: at what age is it established and what are its determinants?* J Epidemiol Community Health, 2003. **57**(12): p. 969-73.

60. Reilly, J.J., et al., *Early life risk factors for obesity in childhood: cohort study.* Br Med J, 2005. **330**(7504): p. 1357.

61. Salsberry, P.J. and P.B. Reagan, *Taking the long view: the prenatal environment and early adolescent overweight.* Res Nurs Health, 2007. **30**(3): p. 297-307.

62. Yang, S., et al., *Emergence of socioeconomic inequalities in smoking and overweight and obesity in early adulthood: the national longitudinal study of adolescent health.* Am J Public Health, 2008. **98**(3): p. 468-77.

63. Erik Landhuis, C., et al., *Programming obesity and poor fitness: the long-term impact of childhood television.* Obesity (Silver Spring), 2008. **16**(6): p. 1457-9.

64. Danner, F.W., *A national longitudinal study of the association between hours of TV viewing and the trajectory of BMI growth among US children.* J Pediatr Psychol, 2008. **33**(10): p. 1100-7.

65. Bhargava, A., D. Jolliffe, and L.L. Howard, *Socio-economic, behavioural and environmental factors predicted body weights and household food insecurity scores in the Early Childhood Longitudinal Study-Kindergarten.* Br J Nutr, 2008. **100**(2): p. 438-44.

66. Henderson, V.R., *Longitudinal associations between television viewing and body mass index among white and black girls.* J Adolesc Health, 2007. **41**(6): p. 544-50.

67. Viner, R.M. and T.J. Cole, *Television viewing in early childhood predicts adult body mass index.* J Pediatr, 2005. **147**(4): p. 429-35.

68. Sturm, R. and A. Datar, *Body mass index in elementary school children, metropolitan area food prices and food outlet density.* Public Health, 2005. **119**(12): p. 1059-68.

69. Baker, E., K.S. Balistreri, and J. Van Hook, *Maternal employment and overweight among Hispanic children of immigrants and children of natives.* J Immigr Minor Health, 2009. **11**(3): p. 158-67.

70. Gorely, T., S.J. Marshall, and S.J. Biddle, *Couch kids: correlates of television viewing among youth.* Int J Behav Med, 2004. **11**(3): p. 152-63.

71. Epstein, L.H., et al., *A randomized trial of the effects of reducing television viewing and computer use on body mass index in young children.* Arch Pediatr Adolesc Med, 2008. **162**(3): p. 239-45.

72. Matijasevich, A., et al., *Socioeconomic position and overweight among adolescents: data from birth cohort studies in Brazil and the UK.* BMC Public Health, 2009. **9**: p. 105.

73. Oliver, L.N. and M.V. Hayes, *Effects of neighbourhood income on reported body mass index: an eight year longitudinal study of Canadian children.* BMC Public Health, 2008. **8**: p. 16.

74. Poulton, R., et al., *Association between children's experience of socioeconomic disadvantage and adult health: a life-course study.* Lancet, 2002. **360**(9346): p. 1640-5.

75. Taveras, E.M., et al., *Association of consumption of fried food away from home with body mass index and diet quality in older children and adolescents.* Pediatrics, 2005. **116**(4): p. e518-24.

76. Viner, R.M. and T.J. Cole, *Who changes body mass between adolescence and adulthood? Factors predicting change in BMI between 16 year and 30 years in the 1970 British Birth Cohort.* Int J Obesity, 2006. **30**(9): p. 1368-74.

77. Bisset, S., et al., *Association of body mass index and dietary restraint with changes in eating behaviour throughout late childhood and early adolescence: a 5-year study.* Public Health Nutr, 2007. **10**(8): p. 780-9.

78. Laitinen, J., et al., *Predictors of abdominal obesity among 31-y-old men and women born in Northern Finland in 1966.* Eur J Clin Nutr, 2004. **58**(1): p. 180-90.

79. Sturm, R. and A. Datar, *Food prices and weight gain during elementary school: 5-year update.* Public Health, 2008. **122**(11): p. 1140-3.

80. Goodman, E. and R.C. Whitaker, *A prospective study of the role of depression in the development and persistence of adolescent obesity.* Pediatrics, 2002. **110**(3): p. 497-504.

81. Hesketh, K., M. Wake, and E. Waters, *Body mass index and parent-reported self-esteem in elementary school children: evidence for a causal relationship.* Int J Obes Relat Metab Disord, 2004. **28**(10): p. 1233-7.

82. Klein, E.G., L.A. Lytle, and V. Chen, *Social ecological predictors of the transition to overweight in youth: results from the Teens Eating for Energy and Nutrition at Schools (TEENS) study.* J Am Diet Assoc, 2008. **108**(7): p. 1163-9.

83. Fidler, J.A., et al., *Does smoking in adolescence affect body mass index, waist or height? Findings from a longitudinal study.* Addiction, 2007. **102**(9): p. 1493-501.

84. Gale, C.R., G.D. Batty, and I.J. Deary, *Locus of control at age 10 years and health outcomes and behaviors at age 30 years: the 1970 British Cohort Study.* Psychosom Med, 2008. **70**(4): p. 397-403.

85. Francis, L.A. and E.J. Susman, *Self-regulation and rapid weight gain in children from age 3 to 12 years.* Arch Pediatr Adolesc Med, 2009. **163**(4): p. 297-302.

86. Koch, F.S., A. Sepa, and J. Ludvigsson, *Psychological stress and obesity.* J Pediatr, 2008. **153**(6): p. 839-44.

87. Thomas, C., E. Hypponen, and C. Power, *Obesity and type 2 diabetes risk in midadult life: the role of childhood adversity.* Pediatrics, 2008. **121**(5): p. e1240-9.

88. Joint WHO/FAO Expert Consultation on Diet Nutrition and the Prevention of Chronic Diseases, *Diet, nutrition and the prevention of chronic diseases: report of a joint WHO/FAO expert consultation*, in *World Health Organ Tech Rep Ser*2003: Geneva. p. i-viii, 1-149,.

89. National Preventative Health Taskforce, *Australia: the healthiest country by 2020. Technical report no. 1 Obesity in Australia: a need for urgent action Including addendum for October 2008 to June 2009* 2009b, Commonwealth of Australia: Canberra.

90. Schmidt, M., et al., *Fast-food intake and diet quality in black and white girls: the National Heart, Lung, and Blood Institute Growth and Health Study.* Arch Pediatr Adolesc Med, 2005. **159**(7): p. 626-31.

91. Affenito, S.G., et al., *Breakfast consumption by African-American and white adolescent girls correlates positively with calcium and fiber intake and negatively with body mass index.* J Am Diet Assoc, 2005. **105**(6): p. 938-45.

92. Larson, N.I., et al., *Family meals during adolescence are associated with higher diet quality and healthful meal patterns during young adulthood.* J Am Diet Assoc, 2007. **107**(9): p. 1502-10.

93. Australian Bureau of Statistics. *Australian Health Survey*. 2011 [cited 31 May 2011]; Available from: http://www.abs.gov.au/websitedbs/D3310114.nsf/Home/Australian+Health+Survey?OpenDocument

94. Mozaffarian, D. and D.S. Ludwig, *Dietary Guidelines in the 21st Century: a Time for Food.* Journal of the American Medical Association, 2010. **304**(6): p. 681-682.

95. Yngve, A. and M. Tseng, *Dietary guidelines and goal-setting.* Public Health Nutrition, 2010. **13**(8): p. 1149-1150.

96. Holdsworth, M., *Sustainability should be integral to nutrition and dietetics.* Human Nutrition and Dietetics, 2010. **23**: p. 467-468.

97. Kickbusch, I., *The Food System: a prism of present and future challenges for health promotion and sustainable development*, 2010, Health Promotion Switzerland: Geneva.

98. Food Standards Australia New Zealand, *The 22nd Australian Total Diet Study*, 2008, Food Standards Australia New Zealand: Canberra, Australia and Wellington, NZ.

99. Commonwealth Department of Foreign Affairs and Trade. *Australia in brief: a diverse people*. 2010 [cited 2011 29 March]; Available from: http://www.dfat.gov.au/aib/society.html.

100. Symons, M., *One continuous picnic : a gastronomic history of Australia*. 2nd ed2007, Carlton, Vic.: Melbourne University Press. xviii, 366 p.

101. Stanton, R.A., *Nutrition problems in an obesogenic environment.* Med J Aust, 2006. **184**(2): p. 76-9.

102. Walker, K.Z., et al., *Product variety in Australian snacks and drinks: how can the consumer make a healthy choice?* Public Health Nutr, 2008. **11**(10): p. 1046-53.

103. National Health and Medical Research Council, *Iodine supplementation for pregnant and breastfeeding women*, 2010, Commonwealth of Australia: Canberra.

104. Harriss, L.R., et al., *Dietary patterns and cardiovascular mortality in the Melbourne Collaborative Cohort Study.* Am J Clin Nutr, 2007. **86**(1): p. 221-9.

105. Jacques, P.F. and K.L. Tucker, *Are dietary patterns useful for understanding the role of diet in chronic disease?* Am J Clin Nutr, 2001. **73**(1): p. 1-2.

106. Kant, A.K., et al., *A prospective study of diet quality and mortality in women.* J Am Med Assoc, 2000. **283**(16): p. 2109-15.

107. McCullough, M.L., et al., *Diet quality and major chronic disease risk in men and women: moving toward improved dietary guidance.* Am J Clin Nutr, 2002. **76**(6): p. 1261-71.

108. Wahlqvist, M.L., et al., *Phytochemical deficiency disorders: inadequate intake of protective foods.* Current therapeutics, 1998. **39**(7): p. 53-60.

109. Van Dokkum, W., et al., *The health effects of bioactive plant components in food: results and opinions of the EU COST 926 action.* Nutrition Bulletin, 2008. **33**(2): p. 133-139.

110. Dolan, L.C., R.A. Matulka, and G.A. Burdock, *Naturally occurring food toxins.* Toxins, 2010. **2**(9): p. 2289-2332.

111. Gibson, R.S., *The role of diet- and host-related factors in nutrient bioavailability and thus in nutrient-based dietary requirement estimates.* Food Nutr Bull, 2007. **28**(1 Suppl International): p. S77-100.

112. Lopez, M.A. and F.C. Martos, *Iron availability: an updated review.* Int J Food Sci Nutr, 2004. **55**(8): p. 597-606.

113. Russell, R.M., *The vitamin A spectrum: from deficiency to toxicity.* Am J Clin Nutr, 2000. **71**(4): p. 878-84.

114. National Health and Medical Research Council, *The Australian Guide to Healthy Eating*, 2011, National Health and Medical Research Council: Canberra.

115. Knudsen, V.K., et al., *Major dietary patterns in pregnancy and fetal growth.* Eur J Clin Nutr, 2008. **62**(4): p. 463-70.

116. Rodriguez-Bernal, C.L., et al., *Diet quality in early pregnancy and its effects on fetal growth outcomes: the Infancia y Medio Ambiente (Childhood and Environment) Mother and Child Cohort Study in Spain.* Am J Clin Nutr, 2010. **91**(6): p. 1659-66.

117. Thompson, J.M., et al., *Maternal dietary patterns in pregnancy and the association with small-for-gestational-age infants.* Br J Nutr, 2010. **103**(11): p. 1665-73.

118. Kinnunen, T.I., et al., *Preventing excessive weight gain during pregnancy - a controlled trial in primary health care.* Eur J Clin Nutr, 2007. **61**(7): p. 884-91.

119. Brantsæter, A.L., et al., *A dietary pattern characterized by high intake of vegetables, fruits, and vegetable oils is associated with reduced risk of preeclampsia in nulliparous pregnant Norwegian women.* J Nutr, 2009. **139**(6): p. 1162.

120. Rifas-Shiman, S.L., et al., *Dietary quality during pregnancy varies by maternal characteristics in Project Viva: a US cohort.* J Am Diet Assoc, 2009. **109**(6): p. 1004-1011.

121. Radesky, J.S., et al., *Diet during early pregnancy and development of gestational diabetes.* Paediatr Perinat Epidemiol, 2008. **22**(1): p. 47-59.

122. Tieu, J., C.A. Crowther, and P. Middleton, *Dietary advice in pregnancy for preventing gestational diabetes mellitus.* Cochrane Database Syst Rev, 2008(2): p. CD006674.

123. Zhang, C., et al., *Dietary fiber intake, dietary glycemic load, and the risk for gestational diabetes mellitus.* Diabetes Care, 2006. **29**(10): p. 2223-30.

124. Chatzi, L., et al., *Mediterranean diet in pregnancy is protective for wheeze and atopy in childhood.* Thorax, 2008. **63**(6): p. 507.

125. De Batlle, J., et al., *Mediterranean diet is associated with reduced asthma and rhinitis in Mexican children.* Allergy, 2008. **63**(10): p. 1310-1316.

126. Hattevig, G., et al., *Effect of maternal avoidance of eggs, cow's milk and fish during lactation upon allergic manifestations in infants.* Clinical & Experimental Allergy, 1989. **19**(1): p. 27-32.

127. Lange, N.E., et al., *Maternal dietary pattern during pregnancy is not associated with recurrent wheeze in children.* J Allergy Clin Immunol, 2010. **126**(2): p. 250-255.

128. Shaheen, S.O., et al., *Dietary patterns in pregnancy and respiratory and atopic outcomes in childhood.* Thorax, 2009. **64**(5): p. 411-7.

129. Robinson, S., et al., *Dietary patterns in infancy: the importance of maternal and family influences on feeding practice.* Br J Nutr, 2007. **98**(5): p. 1029-37.

130. Food Standards Australia New Zealand. *Listeria*. 2011 [cited 2011 29 March]; Available from: http://www.foodstandards.gov.au/consumerinformation/listeria/.

131. Food Standards Australia New Zealand. *Mercury in fish*. 2011 [cited 2011 29 March]; Available from: http://www.foodstandards.gov.au/consumerinformation/adviceforpregnantwomen/mercuryinfish.cfm.

132. Cullen, G. and D. O'Donoghue, *Constipation and pregnancy.* Best practice & research. Clinical gastroenterology, 2007. **21**(5): p. 807-18.

133. National Health and Medical Research Council, * *Title to be confirmed - Infant Feeding Guidelines for Health Workers* 2011, Commonwealth of Australia: Canberra.

134. Lee, A.J., K. O'Dea, and J.D. Mathews, *Apparent dietary intake in remote Aboriginal communities.* Aust J Public Health, 1994. **18**(2): p. 190-7.

135. Brimblecombe, J.K. and K. O'Dea, *The role of energy cost in food choices for an Aboriginal population in northern Australia.* Med J Aust, 2009. **190**(10): p. 549-51.

136. Ball, K., A. Timperio, and D. Crawford, *Neighbourhood socioeconomic inequalities in food access and affordability.* Health & place, 2009. **15**(2): p. 578-585.

137. Burns, C. and A. Inglis, *Measuring food access in Melbourne: access to healthy and fast foods by car, bus and foot in an urban municipality in Melbourne.* Health & place, 2007. **13**(4): p. 877-885.

138. Kettings, C., A.J. Sinclair, and M. Voevodin, *A healthy diet consistent with Australian health recommendations is too expensive for welfare dependent families.* Aust N Z J Public Health, 2009. **33**(6): p. 566-572.

139. Landrigan, T. and C. Pollard, *Food Access and Cost Survey (FACS), Western Australia, 2010*, W. Department of Health, Editor 2011, Department of Health, WA: Perth, WA.

140. Harrison, M., et al., *The increasing cost of healthy food.* Aust N Z J Public Health, 2010. **34**(2): p. 179-186.

141. NT Department of Health and Community Services, *NT Market basket survey, 2006*, 2007, NT Department of Health and Community Services.

142. Marsh, K., et al., *Meeting nutritional needs on a vegetarian diet.* Aust Fam Physician, 2009. **38**(8): p. 600-2.

143. U.S. Department of Agriculture and U.S. Department of Health and Human Services, *Dietary Guidelines for Americans, 2010*, 2010, U.S. Government Printing Office: Washington, DC.

144. U.S. Department of Health and Human Services and U.S. Department of Agriculture, *Dietary Guidelines for Americans 2005*, 2005, U.S. Government Printing Office: Washington DC.

145. Dauchet, L., et al., *Fruit and vegetable consumption and risk of coronary heart disease: a meta-analysis of cohort studies.* J Nutr, 2006. **136**(10): p. 2588-93.

146. He, F.J., et al., *Increased consumption of fruit and vegetables is related to a reduced risk of coronary heart disease: meta-analysis of cohort studies.* J Hum Hypertens, 2007. **21**(9): p. 717-28.

147. Joshipura, K.J., et al., *Intakes of fruits, vegetables and carbohydrate and the risk of CVD.* Public Health Nutr, 2009. **12**(1): p. 115-21.

148. Rastogi, T., et al., *Diet and risk of ischemic heart disease in India.* Am J Clin Nutr, 2004. **79**(4): p. 582-92.

149. Rodríguez-Rodríguez, E., et al., *Restricted-energy diets rich in vegetables or cereals improve cardiovascular risk factors in overweight/obese women.* Nutr Res, 2007. **27**(6): p. 313-320.

150. Takachi, R., et al., *Fruit and vegetable intake and risk of total cancer and cardiovascular disease: Japan Public Health Center-Based Prospective Study.* Am J Epidemiol, 2008. **167**(1): p. 59-70.

151. Dauchet, L., P. Amouyel, and J. Dallongeville, *Fruit and vegetable consumption and risk of stroke: a meta-analysis of cohort studies.* Neurology, 2005. **65**(8): p. 1193-7.

152. He, F.J., C.A. Nowson, and G.A. MacGregor, *Fruit and vegetable consumption and stroke: meta-analysis of cohort studies.* Lancet, 2006. **367**(9507): p. 320-6.

153. Villegas, R., et al., *Vegetable but not fruit consumption reduces the risk of type 2 diabetes in Chinese women.* J Nutr, 2008. **138**(3): p. 574-80.

154. Harding, A.H., et al., *Plasma vitamin C level, fruit and vegetable consumption, and the risk of new-onset type 2 diabetes mellitus: the European prospective investigation of cancer–Norfolk prospective study.* Arch Intern Med, 2008. **168**(14): p. 1493-9.

155. Hamer, M. and Y. Chida, *Intake of fruit, vegetables, and antioxidants and risk of type 2 diabetes: systematic review and meta-analysis.* J Hypertens, 2007. **25**(12): p. 2361-9.

156. Field, A.E., et al., *Association between fruit and vegetable intake and change in body mass index among a large sample of children and adolescents in the United States.* Int J Obes Relat Metab Disord, 2003. **27**(7): p. 821-6.

157. He, K., et al., *Changes in intake of fruits and vegetables in relation to risk of obesity and weight gain among middle-aged women.* Int J Obes Relat Metab Disord, 2004. **28**(12): p. 1569-74.

158. Sartorelli, D.S., et al., *High intake of fruits and vegetables predicts weight loss in Brazilian overweight adults.* Nutr Res, 2008. **28**(4): p. 233-8.

159. te Velde, S.J., et al., *Tracking of fruit and vegetable consumption from adolescence into adulthood and its longitudinal association with overweight* Br J Nutr, 2007. **98**(2): p. 431-8.

160. Vioque, J., et al., *Intake of fruits and vegetables in relation to 10-year weight gain among Spanish adults.* Obesity (Silver Spring), 2008. **16**(3): p. 664-670.

161. Heck, J.E., et al., *Dietary risk factors for hypopharyngeal cancer in India.* Cancer Causes Control, 2008. **19**(10): p. 1329-37.

162. Kreimer, A.R., et al., *Diet and body mass, and oral and oropharyngeal squamous cell carcinomas: analysis from the IARC multinational case-control study.* Int J Cancer, 2006. **118**(9): p. 2293-7.

163. Gallicchio, L., et al., *Adulthood consumption of preserved and nonpreserved vegetables and the risk of nasopharyngeal carcinoma: a systematic review.* Int J Cancer, 2006. **119**(5): p. 1125-35.

164. Guneri, P., et al., *Primary oral cancer in a Turkish population sample: association with sociodemographic features, smoking, alcohol, diet and dentition.* Oral Oncol, 2005. **41**(10): p. 1005-12.

165. Escribano Uzcudun, A., et al., *Pharyngeal cancer prevention: evidence from a case--control study involving 232 consecutive patients.* J Laryngol Otol, 2002. **116**(7): p. 523-31.

166. George, S.M., et al., *Fruit and vegetable intake and risk of cancer: a prospective cohort study.* Am J Clin Nutr, 2009. **89**(1): p. 347.

167. Freedman, N.D., et al., *Fruit and vegetable intake and esophageal cancer in a large prospective cohort study.* Int J Cancer, 2007. **121**(12): p. 2753-60.

168. Anderson, L.A., et al., *Risk factors for Barrett's oesophagus and oesophageal adenocarcinoma: results from the FINBAR study.* World J Gastroenterol, 2007. **13**(10): p. 1585-94.

169. González, C.A., et al., *Fruit and vegetable intake and the risk of stomach and oesophagus adenocarcinoma in the European Prospective Investigation into Cancer and Nutrition (EPIC–EURGAST).* Int J Cancer, 2006. **118**(10): p. 2559-2566.

170. Etminan, M., B. Takkouche, and F. Caamano-Isorna, *The role of tomato products and lycopene in the prevention of prostate cancer: a meta-analysis of observational studies.* Cancer Epidemiol Biomarkers Prev, 2004. **13**(3): p. 340-5.

171. Stram, D.O., et al., *Prostate cancer incidence and intake of fruits, vegetables and related micronutrients: the multiethnic cohort study (United States).* Cancer Causes Control, 2006. **17**(9): p. 1193-207.

172. Koushik, A., et al., *Fruits and vegetables and ovarian cancer risk in a pooled analysis of 12 cohort studies.* Cancer Epidemiol Biomarkers Prev, 2005. **14**(9): p. 2160-7.

173. Schulz, M., et al., *Fruit and vegetable consumption and risk of epithelial ovarian cancer: the European Prospective Investigation into Cancer and Nutrition.* Cancer Epidemiol Biomarkers Prev, 2005. **14**(11): p. 2531.

174. Bandera, E.V., et al., *Fruits and vegetables and endometrial cancer risk: a systematic literature review and meta-analysis.* Nutr Cancer, 2007. **58**(1): p. 6-21.

175. Bravi, F., et al., *Food groups and endometrial cancer risk: a case-control study from Italy.* Am J Obstet Gynecol, 2009. **200**(3): p. 293 e1-7.

176. Yeh, M., et al., *Higher intakes of vegetables and vegetable-related nutrients are associated with lower endometrial cancer risks.* J Nutr, 2009. **139**(2): p. 317-22.

177. Koushik, A., et al., *Fruits, vegetables, and colon cancer risk in a pooled analysis of 14 cohort studies.* J Natl Cancer Inst, 2007. **99**(19): p. 1471-83.

178. Lam, T.K., et al., *Cruciferous vegetable consumption and lung cancer risk: a systematic review.* Cancer Epidemiol Biomarkers Prev, 2009. **18**(1): p. 184-95.

179. Cho, E., et al., *Prospective study of intake of fruits, vegetables, vitamins, and carotenoids and risk of age-related maculopathy.* Arch Ophthalmol, 2004. **122**(6): p. 883-92.

180. Harland, J.I. and T.A. Haffner, *Systematic review, meta-analysis and regression of randomised controlled trials reporting an association between an intake of circa 25g soya protein per day and blood cholesterol.* Atherosclerosis, 2008. **200**(1): p. 13-27.

181. Deibert, P., et al., *Weight loss without losing muscle mass in pre-obese and obese subjects induced by a high-soy-protein diet.* International Journal of Obesity, 2004. **28**(10): p. 1349-1352.

182. Liao, F.H., et al., *Effectiveness of a soy-based compared with a traditional low-calorie diet on weight loss and lipid levels in overweight adults.* Nutrition, 2007. **23**(7-8): p. 551-6.

183. St-Onge, M.P., et al., *Supplementation with soy-protein-rich foods does not enhance weight loss.* J Am Diet Assoc, 2007. **107**(3): p. 500-505.

184. Akhter, M., et al., *Dietary soy and isoflavone intake and risk of colorectal cancer in the Japan public health center-based prospective study.* Cancer Epidemiol Biomarkers Prev, 2008. **17**(8): p. 2128-35.

185. Deneo-Pellegrini, H., et al., *Plant foods and differences between colon and rectal cancers.* Eur J Cancer Prev, 2002. **11**(4): p. 369.

186. Michels, K.B., et al., *Fruit and vegetable consumption and colorectal adenomas in the Nurses' Health Study.* Cancer Res, 2006. **66**(7): p. 3942-53.

187. Oba, S., et al., *Soy product consumption and the risk of colon cancer: a prospective study in Takayama, Japan.* Nutr Cancer, 2007. **57**(2): p. 151-7.

188. Yang, G., et al., *Prospective cohort study of soy food intake and colorectal cancer risk in women.* Am J Clin Nutr, 2009. **89**(2): p. 577-83.

189. Ward, H.A., et al., *Breast, colorectal, and prostate cancer risk in the European Prospective Investigation into Cancer and Nutrition–Norfolk in relation to phytoestrogen intake derived from an improved database.* Am J Clin Nutr, 2010. **91**(2): p. 440.

190. Booth, A., et al., *Dietary approaches for weight loss with increased intakes of fruit, vegetables and dairy products.* Nutr Diet, 2008. **65**(2): p. 115-120.

191. Conceição de Oliveira, M., R. Sichieri, and A. Sanchez Moura, *Weight loss associated with a daily intake of three apples or three pears among overweight women.* Nutrition, 2003. **19**(3): p. 253-256.

192. Drapeau, V., et al., *Modifications in food-group consumption are related to long-term body-weight changes.* Am J Clin Nutr, 2004. **80**(1): p. 29-37.

193. Rodriguez, M.C., et al., *Effects of two energy-restricted diets containing different fruit amounts on body weight loss and macronutrient oxidation.* Plant Foods Hum Nutr, 2005. **60**(4): p. 219-24.

194. Riboli, E. and T. Norat, *Epidemiologic evidence of the protective effect of fruit and vegetables on cancer risk.* Am J Clin Nutr, 2003. **78**(3 Suppl): p. 559S-569S.

195. Gaudet, M.M., et al., *Fruits, vegetables, and micronutrients in relation to breast cancer modified by menopause and hormone receptor status.* Cancer Epidemiol Biomarkers Prev, 2004. **13**(9): p. 1485-94.

196. Hermann, S., J. Linseisen, and J. Chang-Claude, *Nutrition and breast cancer risk by age 50: a population-based case-control study in Germany.* Nutr Cancer, 2002. **44**(1): p. 23-34.

197. Kruk, J., *Association of lifestyle and other risk factors with breast cancer according to menopausal status: a case-control study in the Region of Western Pomerania (Poland).* Asian Pac J Cancer Prev, 2007. **8**(4): p. 513-24.

198. Malin, A.S., et al., *Intake of fruits, vegetables and selected micronutrients in relation to the risk of breast cancer.* International journal of cancer. Journal international du cancer, 2003. **105**(3): p. 413-8.

199. van Gils, C.H., et al., *Consumption of vegetables and fruits and risk of breast cancer.* J Am Med Assoc, 2005. **293**(2): p. 183-93.

200. McCullough, M.L., et al., *A prospective study of fruits, vegetables, and risk of endometrial cancer.* Am J Epidemiol, 2007. **166**(8): p. 902-11.

201. Oh, S.-Y., et al., *Relationship of nutrients and food to colorectal cancer risk in Koreans.* Nutr Res, 2005. **25**(9): p. 805-813.

202. Sato, Y., et al., *Fruit and vegetable consumption and risk of colorectal cancer in Japan: The Miyagi Cohort Study.* Public Health Nutr, 2005. **8**(3): p. 309-14.

203. Wu, H., et al., *Fruit and vegetable intakes are associated with lower risk of colorectal adenomas.* J Nutr, 2009. **139**(2): p. 340-4.

204. Hamilton, C.A., *Low-density lipoprotein and oxidised low-density lipoprotein: their role in the development of atherosclerosis.* Pharmacol Ther, 1997. **74**(1): p. 55-72.

205. Thompson, H.J., et al., *Dietary botanical diversity affects the reduction of oxidative biomarkers in women due to high vegetable and fruit intake.* J Nutr, 2006. **136**(8): p. 2207.

206. Wannamethee, S.G., et al., *Associations of vitamin C status, fruit and vegetable intakes, and markers of inflammation and hemostasis.* Am J Clin Nutr, 2006. **83**(3): p. 567-74; quiz 726-7.

207. Jialal, I. and U. Singh, *Is vitamin C an antiinflammatory agent?* Am J Clin Nutr, 2006. **83**(3): p. 525.

208. Tavani, A. and C. La Vecchia, *[beta]-Carotene and risk of coronary heart disease. A review of observational and intervention studies.* Biomedecine & Pharmacotherapy, 1999. **53**(9): p. 409-416.

209. Messina, M.J., *Legumes and soybeans: overview of their nutritional profiles and health effects.* Am J Clin Nutr, 1999. **70**(3): p. 439S.

210. Nagata, C., *Factors to consider in the association between soy isoflavone intake and breast cancer risk.* Journal of Epidemiology, 2010(0): p. 1002160133.

211. Willett, W.C., *Fruits, vegetables, and cancer prevention: turmoil in the produce section.* J Natl Cancer Inst, 2010. **102**(8): p. 510-1.

212. Giovannucci, E., *Alcohol, one-carbon metabolism, and colorectal cancer: recent insights from molecular studies.* J Nutr, 2004. **134**(9): p. 2475S-2481S.

213. Zhang, S.M., et al., *Plasma folate, vitamin B6, vitamin B12, homocysteine, and risk of breast cancer.* J Natl Cancer Inst, 2003. **95**(5): p. 373-80.

214. García-Lafuente, A., et al., *Flavonoids as anti-inflammatory agents: implications in cancer and cardiovascular disease.* Inflammation Research, 2009. **58**(9): p. 537-552.

215. Lippman, S.M., et al., *Effect of selenium and vitamin E on risk of prostate cancer and other cancers: the Selenium and Vitamin E Cancer Prevention Trial (SELECT).* J Am Med Assoc, 2009. **301**(1): p. 39-51.

216. Paolini, M., et al., *Beta-carotene: a cancer chemopreventive agent or a co-carcinogen?* Mutat Res, 2003. **543**(3): p. 195-200.

217. Khan, N., F. Afaq, and H. Mukhtar, *Cancer chemoprevention through dietary antioxidants: progress and promise.* Antioxidants & Redox Signaling, 2008. **10**(3): p. 475-510.

218. Bingham, S.A., et al., *Dietary fibre in food and protection against colorectal cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC): an observational study.* Lancet, 2003. **361**(9368): p. 1496-1501.

219. Inskip, H.M., et al., *Women's compliance with nutrition and lifestyle recommendations before pregnancy: general population cohort study.* Br Med J, 2009. **338**.

220. New Zealand Ministry of Health, *Food and nutrition guidelines for healthy pregnant and breastfeeding women: a background paper*, 2006 (revised 2008), New Zealand Ministry of Health: Wellington.

221. Birch L, *Development of food preferences.* Annual Review of Nutrition, 1999. **19**: p. 41-62.

222. Lussi A, M.B., Peter Shellis R, Wang X., *Analysis of the erosive effect of different dietary substances and medications.* Br J Nutr, 2011. **30**: p. 1-11.

223. Tasmanian School Canteen Association Inc. *The Cool Canteen Accreditation Program*. 2011 [cited 2011 30 June]; Available from: http://www.tascanteenassn.org.au/coolcap.

224. Office of Learning and Teaching, *Go for your life school canteens and other school food services policy*, 2006, Department of Education: Melbourne.

225. Northern Territory Government, *Canteen, nutrition and health eating policy*, D.o.E.a. Training, Editor 2009: Darwin.

226. New South Wales Department of Health and New South Wales Department of Education and Training, *Fresh tastes @ school NSW healthy school canteen strategy*, 2006, NSW Department of Health & NSW Department of Education and Training,: Sydney.

227. Government of Western Australia, *Healthy food and drink*, Department of Education, Editor 2008.

228. Government of South Australia, *Right bite, healthy food and drink supply strategy for south Australian schools and preschools*, Government of South Australia, Editor 2008: Adelaide

229. Dick, M., et al., *Smart choices – healthy food and drink supply strategy for queensland schools: evaluation report*, Queensland health and department of education and training, Editor 2009: Brisbane

230. Commonwealth Department of Health and Ageing, *Guidelines for healthy foods and drinks supplied in school canteens*, 2010, Commonwealth of Australia: Canberra.

231. AIHW, *Australia's Health 2010*, AIHW, Editor 2010, AIHW: Canberra.

232. McDermott, R., et al., *The health and nutrition of young indigenous women in north Queensland–intergenerational implications of poor food quality, obesity, diabetes, tobacco smoking and alcohol use.* Public Health Nutr, 2009. **12**(11): p. 2143-2149.

233. Food Standards Australia New Zealand, *Australia New Zealand Food Standards Code*, 2011.

234. Food Standards Australia and New Zealand, *Standard 2.9.2 Foods for Infants*, 2009.

235. Jacobs et al, *Whole-grain intake may reduce the risk of ischemic heart disease death in postmenopausal women: the Iowa Women's Health Study.* American Journal of Clinical Nutrition, 1998. **68**(2): p. 248-57.

236. Anderson, J.W., *Whole grains protect against atherosclerotic cardiovascular disease.* Proc Nutr Soc, 2003. **62**(1): p. 135-42.

237. Berg, A., et al., *Effect of an oat bran enriched diet on the atherogenic lipid profile in patients with an increased coronary heart disease risk. A controlled randomized lifestyle intervention study.* Ann Nutr Metab, 2003. **47**(6): p. 306-11.

238. De Moura, F., *Whole grain intake and cardiovascular disease and whole grain intake and diabetes: A review.* Life Sciences Research Organization: Bethesda, MD, 2008.

239. Djousse, L. and J.M. Gaziano, *Breakfast cereals and risk of heart failure in the physicians' health study I.* Arch Intern Med, 2007. **167**(19): p. 2080-5.

240. Erkkila, A.T., et al., *Cereal fiber and whole-grain intake are associated with reduced progression of coronary-artery atherosclerosis in postmenopausal women with coronary artery disease.* Am Heart J, 2005. **150**(1): p. 94-101.

241. Flight, I. and P. Clifton, *Cereal grains and legumes in the prevention of coronary heart disease and stroke: a review of the literature.* Eur J Clin Nutr, 2006. **60**(10): p. 1145-59.

242. Jacobs, D.R., Jr. and D.D. Gallaher, *Whole grain intake and cardiovascular disease: a review.* Curr Atheroscler Rep, 2004. **6**(6): p. 415-23.

243. Katcher, H.I., et al., *The effects of a whole grain-enriched hypocaloric diet on cardiovascular disease risk factors in men and women with metabolic syndrome.* Am J Clin Nutr, 2008. **87**(1): p. 79-90.

244. Kelly, S.A., et al., *Wholegrain cereals for coronary heart disease.* Cochrane Database Syst Rev, 2007(2): p. CD005051.

245. Liu, S., et al., *A prospective study of dietary fiber intake and risk of cardiovascular disease among women.* J Am Coll Cardiol, 2002. **39**(1): p. 49-56.

246. Liu, S., et al., *Is intake of breakfast cereals related to total and cause-specific mortality in men?* Am J Clin Nutr, 2003. **77**(3): p. 594.

247. Negri, E., et al., *Fiber intake and risk of nonfatal acute myocardial infarction.* Eur J Clin Nutr, 2003. **57**(3): p. 464-70.

248. Nettleton, J.A., et al., *Incident heart failure is associated with lower whole-grain intake and greater high-fat dairy and egg intake in the Atherosclerosis Risk in Communities (ARIC) study.* J Am Diet Assoc, 2008. **108**(11): p. 1881-1887.

249. Ortega, R.M., et al., *Responses to two weight-loss programs based on approximating the diet to the ideal: differences associated with increased cereal or vegetable consumption.* International Journal for Vitamin & Nutrition Research, 2006. **76**(6): p. 367-76.

250. Pereira, M.A., et al., *Dietary fiber and risk of coronary heart disease: a pooled analysis of cohort studies.* Arch Intern Med, 2004. **164**(4): p. 370-6.

251. Rave, K., et al., *Improvement of insulin resistance after diet with a whole-grain based dietary product: results of a randomized, controlled cross-over study in obese subjects with elevated fasting blood glucose.* Br J Nutr, 2007. **98**(5): p. 929-36.

252. Truswell, A.S., *Cereal grains and coronary heart disease.* Eur J Clin Nutr, 2002. **56**(1): p. 1-14.

253. de Munter, J.S., et al., *Whole grain, bran, and germ intake and risk of type 2 diabetes: a prospective cohort study and systematic review.* PLoS Med, 2007. **4**(8): p. e261.

254. Henry, C.J.K., et al., *Use of low-glycaemic index bread to reduce 24-h blood glucose: implications for dietary advice to non-diabetic and diabetic subjects.* International journal of food sciences and nutrition, 2006. **57**(3-4): p. 273-278.

255. Hodge, A.M., et al., *Glycemic index and dietary fiber and the risk of type 2 diabetes.* Diabetes Care, 2004. **27**(11): p. 2701-6.

256. Krishnan, S., et al., *Glycemic index, glycemic load, and cereal fiber intake and risk of type 2 diabetes in US black women.* Arch Intern Med, 2007. **167**(21): p. 2304-9.

257. Pereira, M.A., et al., *Effect of whole grains on insulin sensitivity in overweight hyperinsulinemic adults.* Am J Clin Nutr, 2002. **75**(5): p. 848-55.

258. Priebe, M.G., et al., *Whole grain foods for the prevention of type 2 diabetes mellitus.* Cochrane Database Syst Rev, 2008(1): p. CD006061.

259. Venn, B.J. and J.I. Mann, *Cereal grains, legumes and diabetes.* Eur J Clin Nutr, 2004. **58**(11): p. 1443-61.

260. Andersson, A., et al., *Whole-grain foods do not affect insulin sensitivity or markers of lipid peroxidation and inflammation in healthy, moderately overweight subjects.* J Nutr, 2007. **137**(6): p. 1401-7.

261. De La Hunty, A. and M. Ashwell, *Are people who regularly eat breakfast cereals slimmer than those who don't? A systematic review of the evidence.* Nutrition Bulletin, 2007. **32**(2): p. 118-128.

262. Gilhooly, C.H., et al., *Use of cereal fiber to facilitate adherence to a human caloric restriction program.* Aging Clin Exp Res, 2008. **20**(6): p. 513-20.

263. Hallfrisch, J., D.J. Scholfield, and K.M. Behall, *Blood pressure reduced by whole grain diet containing barley or whole wheat and brown rice in moderately hypercholesterolemic men.* Nutr Res, 2003. **23**(12): p. 1631-1642.

264. Hamedani, A., et al., *Reduced energy intake at breakfast is not compensated for at lunch if a high-insoluble-fiber cereal replaces a low-fiber cereal.* Am J Clin Nutr, 2009. **89**(5): p. 1343-1349.

265. Huus, K., et al., *Relationship of food frequencies as reported by parents to overweight and obesity at 5 years.* Acta Paediatr, 2009. **98**(1): p. 139-43.

266. Lightowler, H.J. and C.J.K. Henry, *An investigation of the effectiveness of ready-to-eat breakfast cereals in weight loss: comparison between single and mixed varieties.* Nutrition Bulletin, 2009. **34**(1): p. 48-53.

267. Ortega, R.M., et al., *Changes in thiamin intake and blood levels in young, overweight/obese women following hypocaloric diets based on the increased relative consumption of cereals or vegetables.* Eur J Clin Nutr, 2007. **61**(1): p. 77-82.

268. Rodriguez-Rodriguez, E., et al., *Changes in the sensation of hunger and well-being before and after meals in overweight/obese women following two types of hypoenergetic diet.* Public Health Nutr, 2009. **12**(1): p. 44-50.

269. Waller, S.M., et al., *Evening ready-to-eat cereal consumption contributes to weight management.* J Am Coll Nutr, 2004. **23**(4): p. 316-21.

270. Williams, P.G., et al., *Cereal grains, legumes, and weight management: a comprehensive review of the scientific evidence.* Nutr Rev, 2008. **66**(4): p. 171-82.

271. Asano, T. and R.S. McLeod, *Dietary fibre for the prevention of colorectal adenomas and carcinomas.* Cochrane Database Syst Rev, 2002(2): p. CD003430.

272. Jacobs, E.T., et al., *Fiber, sex, and colorectal adenoma: results of a pooled analysis.* Am J Clin Nutr, 2006. **83**(2): p. 343-9.

273. La Vecchia, C., et al., *Session: whole cereal grains, fibre and human cancer wholegrain cereals and cancer in Italy.* Proc Nutr Soc, 2003. **62**(1): p. 45-9.

274. Peters, U., et al., *Dietary fibre and colorectal adenoma in a colorectal cancer early detection programme.* Lancet, 2003. **361**(9368): p. 1491-1495.

275. Schatzkin, A., et al., *Dietary fiber and whole-grain consumption in relation to colorectal cancer in the NIH-AARP Diet and Health Study.* Am J Clin Nutr, 2007. **85**(5): p. 1353-60.

276. Theuwissen, E. and R.P. Mensink, *Water-soluble dietary fibers and cardiovascular disease.* Physiol Behav, 2008. **94**(2): p. 285-92.

277. Kerckhoffs, D.A., et al., *Effects on the human serum lipoprotein profile of beta-glucan, soy protein and isoflavones, plant sterols and stanols, garlic and tocotrienols.* J Nutr, 2002. **132**(9): p. 2494-505.

278. Beck, E.J., et al., *Oat beta-glucan increases postprandial cholecystokinin levels, decreases insulin response and extends subjective satiety in overweight subjects.* Mol Nutr Food Res, 2009. **53**(10): p. 1343-51.

279. Toden, S., et al., *Effects of dietary beef and chicken with and without high amylose maize starch on blood malondialdehyde, interleukins, IGF-I, insulin, leptin, MMP-2, and TIMP-2 concentrations in rats.* Nutr Cancer, 2010. **62**(4): p. 454.

280. Lazaridou, A. and C. Biliaderis, *Molecular aspects of cereal [beta]-glucan functionality: physical properties, technological applications and physiological effects.* Journal of Cereal Science, 2007. **46**(2): p. 101-118.

281. Liu, R.H., *Whole grain phytochemicals and health.* Journal of Cereal Science, 2007. **46**(3): p. 207-219.

282. Slavin, J., *Why whole grains are protective: biological mechanisms.* Proc Nutr Soc, 2003. **62**(1): p. 129-34.

283. De-Regil, L.M., et al. *Effects and safety of periconceptional folate supplementation for preventing birth defects*. 2010. DOI: 10.1002/14651858.CD007950.pub2.

284. Food Standards Australia New Zealand, *Thinking about having a baby?*, 2010, Food Standards Australia New Zealand.

285. Food Standards Australia and New Zealand. *Folic acid / Folate*. 2011 [cited 2011 12 May]; Available from: http://www.foodstandards.gov.au/consumerinformation/adviceforpregnantwomen/folicacidfolateandpr4598.cfm.

286. National Institute for Clinical Studies, *Encouraging periconceptional use of folic acid supplements*, in *Evidence–practice gaps report, volume 2*.2005, National Institute for Clinical Studies: Melbourne.

287. Sinha, R., et al., *Meat intake and mortality: a prospective study of over half a million people.* Arch Intern Med, 2009. **169**(6): p. 562-71.

288. Brink, M., et al., *Meat consumption and K-ras mutations in sporadic colon and rectal cancer in The Netherlands Cohort Study.* Br J Cancer, 2005. **92**(7): p. 1310-20.

289. Chan, A.T., et al., *Prospective study of N-acetyltransferase-2 genotypes, meat intake, smoking and risk of colorectal cancer.* Int J Cancer, 2005. **115**(4): p. 648-52.

290. Kabat, G.C., et al., *A cohort study of dietary iron and heme iron intake and risk of colorectal cancer in women.* Br J Cancer, 2007. **97**(1): p. 118-22.

291. Larsson, S.C. and A. Wolk, *Meat consumption and risk of colorectal cancer: a meta-analysis of prospective studies.* Int J Cancer, 2006. **119**(11): p. 2657-64.

292. Lee, S.A., et al., *Animal origin foods and colorectal cancer risk: a report from the Shanghai Women's Health Study.* Nutr Cancer, 2009. **61**(2): p. 194-205.

293. Norat, T., et al., *Meat consumption and colorectal cancer risk: dose-response meta-analysis of epidemiological studies.* Int J Cancer, 2002. **98**(2): p. 241-56.

294. Oba, S., et al., *The relationship between the consumption of meat, fat, and coffee and the risk of colon cancer: a prospective study in Japan.* Cancer Lett, 2006. **244**(2): p. 260-267.

295. Sato, Y., et al., *Meat consumption and risk of colorectal cancer in Japan: the Miyagi Cohort Study.* Eur J Cancer Prev, 2006. **15**(3): p. 211.

296. Seow, A., et al., *Food groups and the risk of colorectal carcinoma in an Asian population.* Cancer, 2002. **95**(11): p. 2390-2396.

297. Garcia-Closas, R., et al., *Food, nutrient and heterocyclic amine intake and the risk of bladder cancer.* Eur J Cancer, 2007. **43**(11): p. 1731-40.

298. Larsson, S.C., et al., *Meat intake and bladder cancer risk in a Swedish prospective cohort.* Cancer Causes Control, 2009. **20**(1): p. 35-40.

299. Koutros, S., et al., *Meat and meat mutagens and risk of prostate cancer in the Agricultural Health Study.* Cancer Epidemiol Biomarkers Prev, 2008. **17**(1): p. 80-7.

300. Mori, M., et al., *Traditional Japanese diet and prostate cancer.* Mol Nutr Food Res, 2009. **53**(2): p. 191-200.

301. Chan, J.M., F. Wang, and E.A. Holly, *Pancreatic cancer, animal protein and dietary fat in a population-based study, San Francisco Bay Area, California.* Cancer Causes Control, 2007. **18**(10): p. 1153-67.

302. Iso, H., et al., *Intake of fish and n3 fatty acids and risk of coronary heart disease among Japanese: the Japan Public Health Center-Based (JPHC) Study Cohort I.* Circulation, 2006. **113**(2): p. 195.

303. Virtanen, J.K., et al., *Fish consumption and risk of major chronic disease in men.* Am J Clin Nutr, 2008. **88**(6): p. 1618-25.

304. Whelton, S.P., et al., *Meta-analysis of observational studies on fish intake and coronary heart disease.* Am J Cardiol, 2004. **93**(9): p. 1119-23.

305. Yamagishi, K., et al., *Fish, omega-3 polyunsaturated fatty acids, and mortality from cardiovascular diseases in a nationwide community-based cohort of Japanese men and women the JACC (Japan Collaborative Cohort Study for Evaluation of Cancer Risk) Study.* J Am Coll Cardiol, 2008. **52**(12): p. 988-96.

306. Folsom, A.R. and Z. Demissie, *Fish intake, marine omega-3 fatty acids, and mortality in a cohort of postmenopausal women.* Am J Epidemiol, 2004. **160**(10): p. 1005-10.

307. He, K., et al., *Accumulated evidence on fish consumption and coronary heart disease mortality: a meta-analysis of cohort studies.* Circulation, 2004. **109**(22): p. 2705-11.

308. Jarvinen, R., et al., *Intake of fish and long-chain n-3 fatty acids and the risk of coronary heart mortality in men and women.* Br J Nutr, 2006. **95**(4): p. 824-9.

309. Kaushik, S., et al., *Frequency of fish consumption, retinal microvascular signs and vascular mortality.* Microcirculation, 2008. **15**(1): p. 27-36.

310. Konig, A., et al., *A quantitative analysis of fish consumption and coronary heart disease mortality.* Am J Prev Med, 2005. **29**(4): p. 335-46.

311. Nakamura, Y., et al., *Association between fish consumption and all-cause and cause-specific mortality in Japan: NIPPON DATA80, 1980-99.* Am J Med, 2005. **118**(3): p. 239-245.

312. Ness, A., et al., *Diet in childhood and adult cardiovascular and all cause mortality: the Boyd Orr cohort.* Heart, 2005. **91**(7): p. 894.

313. Streppel, M.T., et al., *Long-term fish consumption and n-3 fatty acid intake in relation to (sudden) coronary heart disease death: the Zutphen study.* Eur Heart J, 2008. **29**(16): p. 2024-30.

314. Bouzan, C., et al., *A quantitative analysis of fish consumption and stroke risk.* Am J Prev Med, 2005. **29**(4): p. 347-52.

315. Ding, E.L. and D. Mozaffarian, *Optimal dietary habits for the prevention of stroke.* Semin Neurol, 2006. **26**(1): p. 11-23.

316. Gochfeld, M. and J. Burger, *Good fish/bad fish: a composite benefit-risk by dose curve.* Neurotoxicology, 2005. **26**(4): p. 511-20.

317. Barberger-Gateau, P., et al., *Dietary patterns and risk of dementia: the three-city cohort study.* Neurology, 2007. **69**(20): p. 1921-30.

318. Huang, T., et al., *Benefits of fatty fish on dementia risk are stronger for those without APOE 4.* Neurology, 2005. **65**(9): p. 1409.

319. Kalmijn, S., et al., *Dietary intake of fatty acids and fish in relation to cognitive performance at middle age.* Neurology, 2004. **62**(2): p. 275.

320. Morris, M.C., et al., *Fish consumption and cognitive decline with age in a large community study.* Arch Neurol, 2005. **62**(12): p. 1849-53.

321. van Gelder, B.M., et al., *Fish consumption, n-3 fatty acids, and subsequent 5-y cognitive decline in elderly men: the Zutphen Elderly Study.* Am J Clin Nutr, 2007. **85**(4): p. 1142-7.

322. Weih, M., J. Wiltfang, and J. Kornhuber, *Non-pharmacologic prevention of Alzheimer's disease: nutritional and life-style risk factors.* J Neural Transm, 2007. **114**(9): p. 1187-97.

323. Larrieu, S., et al., *Nutritional factors and risk of incident dementia in the PAQUID longitudinal cohort.* Journal of Nutrition, Health & Aging, 2004. **8**(3): p. 150-154.

324. Maclean, C.H., et al., *Effects of omega-3 fatty acids on cognitive function with aging, dementia, and neurological diseases.* Evidence Report: Technology Assessment (Summary), 2005(114): p. 1-3.

325. Schiepers, O.J., et al., *Plasma phospholipid fatty acid status and depressive symptoms: association only present in the clinical range.* J Affect Disord, 2009. **118**(1-3): p. 209-14.

326. Sontrop, J. and M.K. Campbell, *Omega-3 polyunsaturated fatty acids and depression: a review of the evidence and a methodological critique.* Prev Med, 2006. **42**(1): p. 4-13.

327. Sanchez-Villegas, A., et al., *Long chain omega-3 fatty acids intake, fish consumption and mental disorders in the SUN cohort study.* European journal of nutrition, 2007. **46**(6): p. 337-46.

328. Colangelo, L.A., et al., *Higher dietary intake of long-chain omega-3 polyunsaturated fatty acids is inversely associated with depressive symptoms in women.* Nutrition, 2009. **25**(10): p. 1011-9.

329. Kyrozis, A., et al., *Dietary lipids and geriatric depression scale score among elders: The EPIC-Greece cohort.* J Psychiatr Res, 2009. **43**(8): p. 763-769.

330. Chong, E.W., et al., *Dietary omega-3 fatty acid and fish intake in the primary prevention of age-related macular degeneration: a systematic review and meta-analysis.* Arch Ophthalmol, 2008. **126**(6): p. 826-33.

331. Djousse, L., et al., *Egg consumption and risk of type 2 diabetes in men and women.* Diabetes Care, 2009. **32**(2): p. 295-300.

332. Sauvaget, C., et al., *Intake of animal products and stroke mortality in the Hiroshima/Nagasaki Life Span Study.* Int J Epidemiol, 2003. **32**(4): p. 536-43.

333. Kramer Michael, S. and R. Kakuma, *Maternal dietary antigen avoidance during pregnancy or lactation, or both, for preventing or treating atopic disease in the child.* Cochrane Database Syst Rev, 2006(3).

334. Chakrabarty, G., et al., *The effect of ingestion of egg on serum lipid profile in healthy young free-living subjects.* Indian J Physiol Pharmacol, 2002. **46**(4): p. 492-8.

335. Chakrabarty, G., et al., *The effect of ingestion of egg on the serum lipid profile of healthy young Indians.* Indian J Physiol Pharmacol, 2004. **48**(3): p. 286-92.

336. Djousse, L. and J.M. Gaziano, *Egg consumption in relation to cardiovascular disease and mortality: the Physicians' Health Study.* Am J Clin Nutr, 2008. **87**(4): p. 964-9.

337. Gillingham, L.G., et al., *The effects of consuming docosahexaenoic acid (DHA)-enriched eggs on serum lipids and fatty acid compositions in statin-treated hypercholesterolemic male patients.* Food research international, 2005. **38**(10): p. 1117-1123.

338. Goodrow, E.F., et al., *Consumption of one egg per day increases serum lutein and zeaxanthin concentrations in older adults without altering serum lipid and lipoprotein cholesterol concentrations.* J Nutr, 2006. **136**(10): p. 2519.

339. Harman, N.L., A.R. Leeds, and B.A. Griffin, *Increased dietary cholesterol does not increase plasma low density lipoprotein when accompanied by an energy-restricted diet and weight loss.* European journal of nutrition, 2008. **47**(6): p. 287-293.

340. Katz, D.L., et al., *Egg consumption and endothelial function: a randomized controlled crossover trial.* Int J Cardiol, 2005. **99**(1): p. 65-70.

341. Lee, J.Y., et al., *Consumption of omega-3 fatty acid-enriched eggs and serum lipids in humans.* Journal of nutraceuticals, functional & medical foods, 2003. **4**(1): p. 3-13.

342. Maki, K.C., et al., *Lipid responses in mildly hypertriglyceridemic men and women to consumption of docosahexaenoic acid-enriched eggs.* Int J Vitam Nutr Res, 2003. **73**(5): p. 357-68.

343. Makrides, M., et al., *Nutritional effect of including egg yolk in the weaning diet of breast-fed and formula-fed infants: a randomized controlled trial.* Am J Clin Nutr, 2002. **75**(6): p. 1084-92.

344. Nakamura, Y., et al., *Egg consumption, serum total cholesterol concentrations and coronary heart disease incidence: Japan Public Health Center-based prospective study.* Br J Nutr, 2006. **96**(5): p. 921-8.

345. Nakamura, Y., et al., *Egg consumption, serum cholesterol, and cause-specific and all-cause mortality: the National Integrated Project for Prospective Observation of Non-communicable Disease and Its Trends in the Aged, 1980 (NIPPON DATA80).* Am J Clin Nutr, 2004. **80**(1): p. 58.

346. Ohman, M., et al., *Biochemical effects of consumption of eggs containing omega-3 polyunsaturated fatty acids.* Ups J Med Sci, 2008. **113**(3): p. 315-23.

347. Rose, E.L. and B.J. Holub, *Effects of a liquid egg product containing fish oil on selected cardiovascular disease risk factors: A randomized crossover trial.* Food research international, 2006. **39**(8): p. 910-916.

348. Sindelar, C.A., et al., *Serum lipids of physically active adults consuming omega-3 fatty acid-enriched eggs or conventional eggs.* Nutr Res, 2004. **24**(9): p. 731-739.

349. Vander Wal, J.S., et al., *Egg breakfast enhances weight loss.* Int J Obes (Lond), 2008. **32**(10): p. 1545-51.

350. Vislocky, L.M., et al., *Habitual consumption of eggs does not alter the beneficial effects of endurance training on plasma lipids and lipoprotein metabolism in untrained men and women.* The Journal of Nutritional Biochemistry, 2009. **20**(1): p. 26-34.

351. Wenzel, A.J., et al., *A 12-wk egg intervention increases serum zeaxanthin and macular pigment optical density in women.* J Nutr, 2006. **136**(10): p. 2568-73.

352. Alper, C.M. and R.D. Mattes, *Peanut consumption improves indices of cardiovascular disease risk in healthy adults.* J Am Coll Nutr, 2003. **22**(2): p. 133-41.

353. Hyson, D.A., B.O. Schneeman, and P.A. Davis, *Almonds and almond oil have similar effects on plasma lipids and LDL oxidation in healthy men and women.* J Nutr, 2002. **132**(4): p. 703.

354. Kocyigit, A., A.A. Koylu, and H. Keles, *Effects of pistachio nuts consumption on plasma lipid profile and oxidative status in healthy volunteers.* Nutr Metab Cardiovasc Dis, 2006. **16**(3): p. 202-209.

355. Sabate, J., et al., *Serum lipid response to the graduated enrichment of a Step I diet with almonds: a randomized feeding trial.* Am J Clin Nutr, 2003. **77**(6): p. 1379-84.

356. Spaccarotella, K.J., et al., *The effect of walnut intake on factors related to prostate and vascular health in older men.* Nutr J, 2008. **7**: p. 13.

357. Fraser, G.E., et al., *Effect on body weight of a free 76 kilojoule (320 calorie) daily supplement of almonds for six months.* J Am Coll Nutr, 2002. **21**(3): p. 275.

358. Sabate, J., *Nut consumption and change in weight: the weight of the evidence.* Br J Nutr, 2007. **98**(3): p. 456-7.

359. Kuhnle, G.G.C., et al., *Diet-induced endogenous formation of nitroso compounds in the GI tract.* Free Radical Biology and Medicine, 2007. **43**(7): p. 1040-1047.

360. Ma, R.W. and K. Chapman, *A systematic review of the effect of diet in prostate cancer prevention and treatment.* J Hum Nutr Diet, 2009. **22**(3): p. 187-99; quiz 200-2.

361. Sofi, F., et al., *Adherence to Mediterranean diet and health status: meta-analysis.* Br Med J, 2008. **337**: p. a1344.

362. UK Royal College of Obstericians and Gynaecologists, *Nutrition in pregnancy (SAC opinion paper 18)*, 2010.

363. Ros, E., L.C. Tapsell, and J. Sabaté, *Nuts and berries for heart health.* Curr Atheroscler Rep, 2010: p. 1-10.

364. Segura, R., et al., *Other relevant components of nuts: phytosterols, folate and minerals.* Br J Nutr, 2006. **96**(Suppl 2): p. S36-44.

365. Ziemke, F. and C.S. Mantzoros, *Adiponectin in insulin resistance: lessons from translational research.* Am J Clin Nutr, 2010. **91**(1): p. 258S-261S.

366. Mattes, R.D. and M.L. Dreher, *Nuts and healthy body weight maintenance mechanisms.* Asia Pac J Clin Nutr, 2010. **19**(1): p. 137-41.

367. Tapsell, L., et al., *The effect of a calorie controlled diet containing walnuts on substrate oxidation during 8-hours in a room calorimeter.* J Am Coll Nutr, 2009. **28**(5): p. 611.

368. Commonwealth Department of Agriculture Fisheries and Forestry, *Australia's agriculture, fisheries and forestry at a glance 2010*, 2010, Commonwealth Department of Agriculture, Fisheries and Forestry: Canberra.

369. Greer, F.R., S.H. Sicherer, and A. Burks, *Effects of early nutritional interventions on the development of atopic disease in infants and children: the role of maternal dietary restriction, breastfeeding, timing of introduction of complementary foods, and hydrolyzed formulas.* Pediatrics, 2008. **121**(1): p. 183.

370. Host, A., et al., *Dietary prevention of allergic diseases in infants and small children.* Pediatr Allergy Immunol, 2008. **19**(1): p. 1-4.

371. Sicherer, S.H. and A.W. Burks, *Maternal and infant diets for prevention of allergic diseases: understanding menu changes in 2008.* J Allergy Clin Immunol, 2008. **122**(1): p. 29-33.

372. Prescott, S.L. and M.L. Tang, *The Australasian Society of Clinical Immunology and Allergy position statement: Summary of allergy prevention in children.* Med J Aust, 2005. **182**(9): p. 464-7.

373. Food Authority NSW. *Sydney Harbour seafood and dioxins*. 2010 [cited 2011 29 March]; Available from: http://www.foodauthority.nsw.gov.au/consumers/keeping-food-safe/special-care-foods/sydney-harbour-seafood/.

374. Food Standards Australia New Zealand, *Dioxins in food: dietary exposure assessment and risk characterisation*, in *Technical Report Series No. 27*2004: Canberra.

375. Food and Agriculture Organization of the United Nations and the World Health Organization, *Joint FAO/WHO Expert Consultation on the risks and benefits of fish consumption*, 2010, Food and Agriculture Organization of the United Nations and the World Health Organization: Rome.

376. Elwood, P.C., et al., *The survival advantage of milk and dairy consumption: an overview of evidence from cohort studies of vascular diseases, diabetes and cancer.* J Am Coll Nutr, 2008. **27**(6): p. 723S-34S.

377. He, K., et al., *Dietary fat intake and risk of stroke in male US healthcare professionals: 14 year prospective cohort study.* Br Med J, 2003. **327**(7418): p. 777-82.

378. Alonso, A., et al., *Low-fat dairy consumption and reduced risk of hypertension: the Seguimiento Universidad de Navarra (SUN) cohort.* Am J Clin Nutr, 2005. **82**(5): p. 972-9.

379. Engberink, M.F., et al., *Dairy intake, blood pressure, and incident hypertension in a general Dutch population.* J Nutr, 2009. **139**(3): p. 582-7.

380. Toledo, E., et al., *Low-fat dairy products and blood pressure: follow-up of 2290 older persons at high cardiovascular risk participating in the PREDIMED study.* Br J Nutr, 2009. **101**(01): p. 59-67.

381. Wang, L., et al., *Dietary intake of dairy products, calcium, and vitamin D and the risk of hypertension in middle-aged and older women.* Hypertension, 2008. **51**(4): p. 1073.

382. Pereira, M.A., et al., *Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study.* J Am Med Assoc, 2002. **287**(16): p. 2081-9.

383. Lutsey, P.L., L.M. Steffen, and J. Stevens, *Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities study.* Circulation, 2008. **117**(6): p. 754-61.

384. Pittas, A.G., et al., *The role of vitamin D and calcium in type 2 diabetes. A systematic review and meta-analysis.* J Clin Endocrinol Metab, 2007. **92**(6): p. 2017-29.

385. Barr, S.I., *Increased dairy product or calcium intake: is body weight or composition affected in humans?* J Nutr, 2003. **133**(1): p. 245S-248S.

386. Lanou, A.J. and N.D. Barnard, *Dairy and weight loss hypothesis: an evaluation of the clinical trials.* Nutr Rev, 2008. **66**(5): p. 272-279.

387. Rajpathak, S.N., et al., *Calcium and dairy intakes in relation to long-term weight gain in US men.* Am J Clin Nutr, 2006. **83**(3): p. 559-66.

388. Rosell, M., N.N. Hakansson, and A. Wolk, *Association between dairy food consumption and weight change over 9 y in 19,352 perimenopausal women.* Am J Clin Nutr, 2006. **84**(6): p. 1481-8.

389. Wagner, G., et al., *Effects of various forms of calcium on body weight and bone turnover markers in women participating in a weight loss program.* J Am Coll Nutr, 2007. **26**(5): p. 456-61.

390. Berkey, C.S., et al., *Sugar-added beverages and adolescent weight change.* Obes Res, 2004. **12**(5): p. 778-88.

391. Berkey, C.S., et al., *Milk, dairy fat, dietary calcium, and weight gain: a longitudinal study of adolescents.* Arch Pediatr Adolesc Med, 2005. **159**(6): p. 543-50.

392. Johnson, L., et al., *Is sugar-sweetened beverage consumption associated with increased fatness in children?* Nutrition, 2007. **23**(7-8): p. 557-563.

393. Newby, P.K., et al., *Beverage consumption is not associated with changes in weight and body mass index among low-income preschool children in North Dakota.* J Am Diet Assoc, 2004. **104**(7): p. 1086-1094.

394. Striegel-Moore, R.H., et al., *Correlates of beverage intake in adolescent girls: the National Heart, Lung, and Blood Institute Growth and Health Study.* J Pediatr, 2006. **148**(2): p. 183-7.

395. Cho, E., et al., *Dairy foods, calcium, and colorectal cancer: a pooled analysis of 10 cohort studies.* J Natl Cancer Inst, 2004. **96**(13): p. 1015-22.

396. Huncharek, M., J. Muscat, and B. Kupelnick, *Colorectal cancer risk and dietary intake of calcium, vitamin D, and dairy products: a meta-analysis of 26,335 cases from 60 observational studies.* Nutr Cancer, 2009. **61**(1): p. 47-69.

397. Sellers, T.A., et al., *Unpasteurized milk consumption and subsequent risk of cancer.* Cancer Causes Control, 2008. **19**(8): p. 805-11.

398. Lee, J.E., et al., *Intakes of coffee, tea, milk, soda and juice and renal cell cancer in a pooled analysis of 13 prospective studies.* Int J Cancer, 2007. **121**(10): p. 2246-53.

399. Alvarez-Leon, E.E., B. Roman-Vinas, and L. Serra-Majem, *Dairy products and health: a review of the epidemiological evidence.* Br J Nutr, 2006. **96**(Suppl 1): p. S94-9.

400. Missmer, S.A., et al., *Meat and dairy food consumption and breast cancer: a pooled analysis of cohort studies.* Int J Epidemiol, 2002. **31**(1): p. 78-85.

401. Bandera, E.V., et al., *Consumption of animal foods and endometrial cancer risk: a systematic literature review and meta-analysis.* Cancer Causes Control, 2007. **18**(9): p. 967-88.

402. Bonjour, J.P., et al., *Inhibition of bone turnover by milk intake in postmenopausal women.* Br J Nutr, 2008. **100**(4): p. 866-74.

403. Hartman, J.W., et al., *Consumption of fat-free fluid milk after resistance exercise promotes greater lean mass accretion than does consumption of soy or carbohydrate in young, novice, male weightlifters.* Am J Clin Nutr, 2007. **86**(2): p. 373.

404. Javaid, M.K., et al., *Maternal and seasonal predictors of change in calcaneal quantitative ultrasound during pregnancy.* J Clin Endocrinol Metab, 2005. **90**(9): p. 5182-7.

405. Lanou, A.J., S.E. Berkow, and N.D. Barnard, *Calcium, dairy products, and bone health in children and young adults: a reevaluation of the evidence.* Pediatrics, 2005. **115**(3): p. 736-43.

406. Teegarden, D., et al., *Dietary calcium intake protects women consuming oral contraceptives from spine and hip bone loss.* J Clin Endocrinol Metab, 2005. **90**(9): p. 5127-33.

407. Volek, J.S., et al., *Increasing fluid milk favorably affects bone mineral density responses to resistance training in adolescent boys.* J Am Diet Assoc, 2003. **103**(10): p. 1353-6.

408. Feskanich, D., W.C. Willett, and G.A. Colditz, *Calcium, vitamin D, milk consumption, and hip fractures: a prospective study among postmenopausal women.* Am J Clin Nutr, 2003. **77**(2): p. 504.

409. Kanis, J.A., et al., *A meta-analysis of milk intake and fracture risk: low utility for case finding.* Osteoporos Int, 2005. **16**(7): p. 799-804.

410. Mensink, R.P., et al., *Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials.* Am J Clin Nutr, 2003. **77**(5): p. 1146-55.

411. Temme, E., R.P. Mensink, and G. Hornstra, *Comparison of the effects of diets enriched in lauric, palmitic, or oleic acids on serum lipids and lipoproteins in healthy women and men.* Am J Clin Nutr, 1996. **63**(6): p. 897.

412. Griffith, L.E., et al., *The influence of dietary and nondietary calcium supplementation on blood pressure: an updated metaanalysis of randomized controlled trials.* Am J Hypertens, 1999. **12**(1 Pt 1): p. 84-92.

413. Clare, D.A. and H.E. Swaisgood, *Bioactive milk peptides: a prospectus.* J Dairy Sci, 2000. **83**(6): p. 1187-95.

414. Bolland, M.J., et al., *Effect of calcium supplements on risk of myocardial infarction and cardiovascular events: meta-analysis.* Br Med J, 2010. **341**: p. c3691.

415. Bolland, M.J., et al., *Calcium supplements with or without vitamin D and risk of cardiovascular events: reanalysis of the Women's Health Initiative limited access dataset and meta-analysis.* BMJ, 2011. **342**: p. d2040.

416. Green, J.H., C. Booth, and R. Bunning, *Postprandial metabolic responses to milk enriched with milk calcium are different from responses to milk enriched with calcium carbonate.* Asia Pac J Clin Nutr, 2003. **12**(1): p. 109-19.

417. Resnick, L., et al., *Dietary calcium modifies the pressor effects of dietary salt intake in essential hypertension.* J Hypertens, 1986. **4**(suppl 6): p. S679-S681.

418. Kalkwarf, H.J., J.C. Khoury, and B.P. Lanphear, *Milk intake during childhood and adolescence, adult bone density, and osteoporotic fractures in US women.* Am J Clin Nutr, 2003. **77**(1): p. 257-65.

419. Albala, C., et al., *Effects of replacing the habitual consumption of sugar-sweetened beverages with milk in Chilean children.* Am J Clin Nutr, 2008. **88**(3): p. 605-611.

420. Einarson, A., et al., *Food-borne illnesses during pregnancy.* Can Fam Physician, 2010. **56**(9): p. 869-70.

421. Brand, J.C., et al., *Lactose malabsorption in Australian Aboriginal children.* Am J Clin Nutr, 1985. **41**(3): p. 620-2.

422. Buttenshaw, R., et al. *Lactose malabsorption and its temporal stability in Aboriginal children*. in *Proc Nutr Soc Aust*. 1990.

423. Shaukat, A., et al., *Systematic review: effective management strategies for lactose intolerance.* Ann Intern Med, 2010. **152**(12): p. 797-803.

424. Pfeil, L.A., P.R. Katz, and P.J. Davis, *Water metabolism*, in *Geriatric nutrition: a comprehensive view*, J.E. Morley, Z. Glick, and L.Z. Rubenstein, Editors. 1995, Raven Press: New York.

425. Cheung, S.S. and T.M. McLellan, *Influence of hydration status and fluid replacement on heat tolerance while wearing NBC protective clothing.* European journal of applied physiology and occupational physiology, 1998. **77**(1): p. 139-148.

426. Hubbard, R.W. and L.E. Armstrong, *The heat illnesses: biochemical, ultrastructural, and fluid-electrolyte considerations*, in *Human performance physiology and environmental medicine at terrestrial extremes*, K.B. Pandolf, M.N. Sawka, and R.R. Gonzalez, Editors. 1988, Indianapolis: Benchmark Press, Inc. p. 305-360.

427. Gardner, E., C. Ruxton, and A. Leeds, *Black tea–helpful or harmful? A review of the evidence.* Eur J Clin Nutr, 2006. **61**(1): p. 3-18.

428. Greenberg, J.A., G. Chow, and R.C. Ziegelstein, *Caffeinated coffee consumption, cardiovascular disease, and heart valve disease in the elderly (from the Framingham Study).* Am J Cardiol, 2008. **102**(11): p. 1502-8.

429. Kuriyama, S., *The relation between green tea consumption and cardiovascular disease as evidenced by epidemiological studies.* J Nutr, 2008. **138**(8): p. 1548S.

430. Lagiou, P., et al., *Intake of specific flavonoid classes and coronary heart disease--a case-control study in Greece.* Eur J Clin Nutr, 2004. **58**(12): p. 1643-8.

431. Sesso, H.D., et al., *Lack of association between tea and cardiovascular disease in college alumni.* Int J Epidemiol, 2003. **32**(4): p. 527.

432. Fraser, M.L., G.S. Mok, and A.H. Lee, *Green tea and stroke prevention: emerging evidence.* Complement Ther Med, 2007. **15**(1): p. 46-53.

433. Okamoto, K., *Habitual green tea consumption and risk of an aneurysmal rupture subarachnoid hemorrhage: a case-control study in Nagoya, Japan.* Eur J Epidemiol, 2006. **21**(5): p. 367-71.

434. Tanabe, N., et al., *Consumption of green and roasted teas and the risk of stroke incidence: results from the Tokamachi-Nakasato cohort study in Japan.* Int J Epidemiol, 2008. **37**(5): p. 1030-40.

435. Arab, L., W. Liu, and D. Elashoff, *Green and black tea consumption and risk of stroke: a meta-analysis.* Stroke, 2009. **40**(5): p. 1786-92.

436. Hammar, N., et al., *Association of boiled and filtered coffee with incidence of first nonfatal myocardial infarction: the SHEEP and the VHEEP study.* J Intern Med, 2003. **253**(6): p. 653-9.

437. Klatsky, A.L., et al., *The confounded relation of coffee drinking to coronary artery disease.* Am J Cardiol, 2008. **101**(6): p. 825-7.

438. Rosner, S.A., et al., *Coffee consumption and risk of myocardial infarction among older Swedish women.* Am J Epidemiol, 2007. **165**(3): p. 288.

439. Sofi, F., et al., *Coffee consumption and risk of coronary heart disease: a meta-analysis.* Nutr Metab Cardiovasc Dis, 2007. **17**(3): p. 209-23.

440. Greenberg, J.A., et al., *Coffee, tea and diabetes: the role of weight loss and caffeine.* Int J Obes (Lond), 2005. **29**(9): p. 1121-9.

441. Hamer, M., et al., *Prospective study of coffee and tea consumption in relation to risk of type 2 diabetes mellitus among men and women: The Whitehall II study.* Br J Nutr, 2008. **100**(05): p. 1046-1053.

442. Hu, G., et al., *Joint association of coffee consumption and other factors to the risk of type 2 diabetes: a prospective study in Finland.* Int J Obes (Lond), 2006. **30**(12): p. 1742-9.

443. Odegaard, A.O., et al., *Coffee, tea, and incident type 2 diabetes: the Singapore Chinese Health Study.* Am J Clin Nutr, 2008. **88**(4): p. 979-85.

444. Paynter, N.P., et al., *Coffee and sweetened beverage consumption and the risk of type 2 diabetes mellitus: the atherosclerosis risk in communities study.* Am J Epidemiol, 2006. **164**(11): p. 1075-84.

445. Smith, B., et al., *Does coffee consumption reduce the risk of type 2 diabetes in individuals with impaired glucose?* Diabetes Care, 2006. **29**(11): p. 2385.

446. van Dam, R.M. and F.B. Hu, *Coffee consumption and risk of type 2 diabetes: a systematic review.* J Am Med Assoc, 2005. **294**(1): p. 97-104.

447. van Dam, R.M., et al., *Coffee, caffeine, and risk of type 2 diabetes: a prospective cohort study in younger and middle-aged U.S. women.* Diabetes Care, 2006. **29**(2): p. 398-403.

448. Dubois, L., et al., *Regular sugar-sweetened beverage consumption between meals increases risk of overweight among preschool-aged children.* J Am Diet Assoc, 2007. **107**(6): p. 924-934.

449. Faith, M.S., et al., *Fruit juice intake predicts increased adiposity gain in children from low-income families: weight status-by-environment interaction.* Pediatrics, 2006. **118**(5): p. 2066-75.

450. Forshee, R.A., P.A. Anderson, and M.L. Storey, *Sugar-sweetened beverages and body mass index in children and adolescents: a meta-analysis.* Am J Clin Nutr, 2008. **87**(6): p. 1662-1671.

451. Gibson, S., *Sugar-sweetened soft drinks and obesity: a systematic review of the evidence from observational studies and interventions.* Nutr Res Rev, 2008. **21**(2): p. 134-147.

452. Malik, V.S., M.B. Schulze, and F.B. Hu, *Intake of sugar-sweetened beverages and weight gain: a systematic review.* Am J Clin Nutr, 2006. **84**(2): p. 274-288.

453. Phillips, S.M., et al., *Energy-dense snack food intake in adolescence: longitudinal relationship to weight and fatness.* Obes Res, 2004. **12**(3): p. 461-72.

454. Sichieri, R., et al., *School randomised trial on prevention of excessive weight gain by discouraging students from drinking sodas.* Public Health Nutr, 2009. **12**(2): p. 197-202.

455. Stookey, J.D., et al., *Replacing sweetened caloric beverages with drinking water is associated with lower energy intake.* Obesity (Silver Spring), 2007. **15**(12): p. 3013-22.

456. Tam, C.S., et al., *Soft drink consumption and excess weight gain in Australian school students: results from the Nepean study.* Int J Obesity, 2006. **30**(7): p. 1091-3.

457. Taylor, R.W., et al., *APPLE Project: 2-y findings of a community-based obesity prevention program in primary school age children.* Am J Clin Nutr, 2007. **86**(3): p. 735-42.

458. Vartanian, L.R., M.B. Schwartz, and K.D. Brownell, *Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis.* Am J Public Health, 2007. **97**(4): p. 667-75.

459. Wolff, E. and M.L. Dansinger, *Soft drinks and weight gain: how strong is the link?* Medscape J Med, 2008. **10**(8): p. 189.

460. Pelucchi, C. and C. La Vecchia, *Alcohol, coffee, and bladder cancer risk: a review of epidemiological studies.* Eur J Cancer Prev, 2009. **18**(1): p. 62-8.

461. Villanueva, C.M., et al., *Total and specific fluid consumption as determinants of bladder cancer risk.* Int J Cancer, 2006. **118**(8): p. 2040-2047.

462. Zeegers, M.P.A., et al., *The association between smoking, beverage consumption, diet and bladder cancer: a systematic literature review.* World J Urol, 2004. **21**(6): p. 392-401.

463. Tang N, W.Y., Ma J, Mang B & Yu R., *Coffee consumption and risk of lung cancer: A meta-analysis.* Lung Cancer, 2009. **(In Press at time of review)**.

464. Tang, N., et al., *Coffee consumption and risk of breast cancer: a metaanalysis.* Am J Obstet Gynecol, 2009. **200**(3): p. 290 e1-9.

465. Michels, K.B., et al., *Coffee, tea, and caffeine consumption and incidence of colon and rectal cancer.* J Natl Cancer Inst, 2005. **97**(4): p. 282-92.

466. Lee, K.J., et al., *Coffee consumption and risk of colorectal cancer in a population-based prospective cohort of Japanese men and women.* Int J Cancer, 2007. **121**(6): p. 1312-8.

467. Tavani, A. and C. La Vecchia, *Coffee, decaffeinated coffee, tea and cancer of the colon and rectum: a review of epidemiological studies, 1990-2003.* Cancer Causes Control, 2004. **15**(8): p. 743-57.

468. Steevens, J., et al., *Tea and coffee drinking and ovarian cancer risk: results from the Netherlands Cohort Study and a meta-analysis.* Br J Cancer, 2007. **97**(9): p. 1291-4.

469. Song, Y.J., et al., *Coffee, tea, colas, and risk of epithelial ovarian cancer.* Cancer Epidemiol Biomarkers Prev, 2008. **17**(3): p. 712-6.

470. Silvera, S.A., et al., *Intake of coffee and tea and risk of ovarian cancer: a prospective cohort study.* Nutr Cancer, 2007. **58**(1): p. 22-7.

471. Bravi, F., et al., *Coffee drinking and endometrial cancer risk: a metaanalysis of observational studies.* Am J Obstet Gynecol, 2009. **200**(2): p. 130-135.

472. Larsson, S.C., E. Giovannucci, and A. Wolk, *Coffee consumption and stomach cancer risk in a cohort of Swedish women.* Int J Cancer, 2006. **119**(9): p. 2186-9.

473. Bravi, F., et al., *Coffee drinking and hepatocellular carcinoma risk: a meta-analysis.* Hepatology, 2007. **46**(2): p. 430-5.

474. Su, L.J. and L. Arab, *Tea consumption and the reduced risk of colon cancer – results from a national prospective cohort study.* Public Health Nutr, 2002. **5**(3): p. 419-25.

475. Dora, I., et al., *Black tea consumption and risk of rectal cancer in Moscow population.* Ann Epidemiol, 2003. **13**(6): p. 405-11.

476. Kristensen, M., et al., *Short-term effects on bone turnover of replacing milk with cola beverages: a 10-day interventional study in young men.* Osteoporos Int, 2005. **16**(12): p. 1803-8.

477. Libuda, L., et al., *Association between long-term consumption of soft drinks and variables of bone modeling and remodeling in a sample of healthy German children and adolescents.* Am J Clin Nutr, 2008. **88**(6): p. 1670.

478. Manias, K., D. McCabe, and N. Bishop, *Fractures and recurrent fractures in children; varying effects of environmental factors as well as bone size and mass.* Bone, 2006. **39**(3): p. 652-7.

479. Tucker, K.L., et al., *Colas, but not other carbonated beverages, are associated with low bone mineral density in older women: The Framingham Osteoporosis Study.* Am J Clin Nutr, 2006. **84**(4): p. 936.

480. Ahokas, J.T., et al., *Review of water fluoridation and fluoride intake from discretionary fluoride supplements*, 1999, National Health and Medical Research Council: Melbourne.

481. Young, W.G., *Tooth wear: diet analysis and advice.* Int Dent J, 2005. **55**(2): p. 68-72.

482. Kazoullis, S., et al., *Common dental conditions associated with dental erosion in schoolchildren in Australia.* Pediatr Dent, 2007. **29**(1): p. 33-9.

483. American Academy of Pediatrics, *Soft drinks in schools.* Pediatrics, 2004. **113**(1 Pt 1): p. 152-4.

484. Keijzers, G.B., et al., *Caffeine can decrease insulin sensitivity in humans.* Diabetes Care, 2002. **25**(2): p. 364.

485. Yoshioka, K., et al., *Coffee consumption and risk of type 2 diabetes mellitus.* Lancet, 2003. **361**(9358): p. 703.

486. Lopez-Ridaura, R., et al., *Magnesium intake and risk of type 2 diabetes in men and women.* Diabetes Care, 2004. **27**(1): p. 134.

487. Curin, Y. and R. Andriantsitohaina, *Polyphenols as potential therapeutical agents against cardiovascular diseases.* Pharmacological reports, 2005. **57**: p. 97.

488. Ratnayake, W.M. and C. Galli, *Fat and fatty acid terminology, methods of analysis and fat digestion and metabolism: a background review paper.* Ann Nutr Metab, 2009. **55**(1-3): p. 8-43.

489. Melanson, E.L., A. Astrup, and W.T. Donahoo, *The relationship between dietary fat and fatty acid intake and body weight, diabetes, and the metabolic syndrome.* Ann Nutr Metab, 2009. **55**(1-3): p. 229-243.

490. Mozaffarian, D., R. Micha, and S. Wallace, *Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials.* PLoS Med, 2010. **7**(3): p. e1000252.

491. Balk, E.M., et al., *Effects of omega-3 fatty acids on coronary restenosis, intima-media thickness, and exercise tolerance: A systematic review.* Atherosclerosis, 2006. **184**(2): p. 237-246.

492. Balk, E.M., et al., *Effects of omega-3 fatty acids on serum markers of cardiovascular disease risk: a systematic review.* Atherosclerosis, 2006. **189**(1): p. 19-30.

493. Castro, I.A., L.P. Barroso, and P. Sinnecker, *Functional foods for coronary heart disease risk reduction: a meta-analysis using a multivariate approach.* Am J Clin Nutr, 2005. **82**(1): p. 32-40.

494. Eslick, G.D., et al., *Benefits of fish oil supplementation in hyperlipidemia: a systematic review and meta-analysis.* Int J Cardiol, 2009. **136**(1): p. 4-16.

495. Hooper, L., et al., *Omega 3 fatty acids for prevention and treatment of cardiovascular disease.* Cochrane Database Syst Rev, 2004(4): p. CD003177.

496. Lee, K.W. and G.Y.H. Lip, *Effects of lifestyle on hemostasis, fibrinolysis, and platelet reactivity: a systematic review.* Arch Intern Med, 2003. **163**(19): p. 2368-2392.

497. Studer, M., et al., *Effect of different antilipidemic agents and diets on mortality: a systematic review.* Arch Intern Med, 2005. **165**(7): p. 725.

498. Wang, C., et al., *n-3 fatty acids from fish or fish-oil supplements, but not alpha-linolenic acid, benefit cardiovascular disease outcomes in primary- and secondary-prevention studies: a systematic review.* Am J Clin Nutr, 2006. **84**(1): p. 5-17.

499. Wendland, E., et al., *Effect of alpha linolenic acid on cardiovascular risk markers: a systematic review.* Heart, 2006. **92**(2): p. 166-169.

500. Food and Agriculture Organization of the United Nations and the World Health Organization, *Fats and fatty acids in human nutrition. Report of an expert consultation*, 2010, Food and Agriculture Organization of the United Nations and the World Health Organization: Rome.

501. Ulbak, J., et al., *Diet and blood pressure in 2.5-y-old Danish children.* Am J Clin Nutr, 2004. **79**(6): p. 1095-1102.

502. Schack-Nielsen, L., et al., *Arterial stiffness in 10-year-old children: current and early determinants.* Br J Nutr, 2005. **94**(6): p. 1004-1011.

503. Sanders, T.A.B., et al., *Influence of an algal triacylglycerol containing docosahexaenoic acid (22:6n-3) and docosahentaenoic acid (22:5n-6) on cardiovascular risk factors in healthy men and women.* Br J Nutr, 2006. **95**(3): p. 525-531.

504. Raitakari, O.T., et al., *Endothelial function in healthy 11-year-old children after dietary intervention with onset in infancy: the Special Turku Coronary Risk Factor Intervention Project for children (STRIP).* Circulation, 2005. **112**(24): p. 3786-3794.

505. Psaltopoulou, T., et al., *Olive oil, the Mediterranean diet, and arterial blood pressure: the Greek European Prospective Investigation into Cancer and Nutrition (EPIC) study [corrected] [published erratum appears in AM J CLIN NUTR 2005 May;81(5):1181].* Am J Clin Nutr, 2004. **80**(4): p. 1012-1018.

506. Perona, J.S., et al., *Plasma lipid modifications in elderly people after administration of two virgin olive oils of the same variety (Olea europaea var. hojiblanca) with different triacylglycerol composition.* Br J Nutr, 2003. **89**(6): p. 819-26.

507. Bondia-Pons, I., et al., *Moderate consumption of olive oil by healthy European men reduces systolic blood pressure in non-Mediterranean participants.* J Nutr, 2007. **137**(1): p. 84-7.

508. Alonso, A., et al., *Olive oil consumption and reduced incidence of hypertension: the SUN study.* Lipids, 2004. **39**(12): p. 1233-8.

509. Damsgaard, C.T., et al., *Fish oil in combination with high or low intakes of linoleic acid lowers plasma triacylglycerols but does not affect other cardiovascular risk markers in healthy men.* J Nutr, 2008. **138**(6): p. 1061-1066.

510. Berry, S.E., et al., *The solid fat content of stearic acid-rich fats determines their postprandial effects.* Am J Clin Nutr, 2007. **85**(6): p. 1486-94.

511. Eckel, R.H., et al., *Carbohydrate balance predicts weight and fat gain in adults.* Am J Clin Nutr, 2006. **83**(4): p. 803-808.

512. Kratz, M., et al., *The impact of dietary fat composition on serum leptin concentrations in healthy nonobese men and women.* Journal of Clinical Endocrinology & Metabolism, 2002. **87**(11): p. 5008-14.

513. Lovegrove, J.A., et al., *Moderate fish-oil supplementation reverses low-platelet, long-chain n-3 polyunsaturated fatty acid status and reduces plasma triacylglycerol concentrations in British Indo-Asians.* Am J Clin Nutr, 2004. **79**(6): p. 974-982.

514. Nicholls, S.J., et al., *Consumption of saturated fat impairs the anti-inflammatory properties of high-density lipoproteins and endothelial function.* J Am Coll Cardiol, 2006. **48**(4): p. 715-720.

515. Nosaka, N., et al., *Effects of dietary medium-chain triacylglycerols on serum lipoproteins and biochemical parameters in healthy men.* Bioscience, Biotechnology & Biochemistry, 2002. **66**(8): p. 1713-8.

516. Owen, B. and T.M.S. Wolever, *Effect of fat on glycaemic responses in normal subjects: a dose-response study.* Nutr Res, 2003. **23**(10): p. 1341-1347.

517. Rueda-Clausen, C.F., et al., *Olive, soybean and palm oils intake have a similar acute detrimental effect over the endothelial function in healthy young subjects.* Nutr Metab Cardiovasc Dis, 2007. **17**(1): p. 50-57.

518. Tahvonen, R.L., et al., *Black currant seed oil and fish oil supplements differ in their effects on fatty acid profiles of plasma lipids, and concentrations of serum total and lipoprotein lipids, plasma glucose and insulin.* The Journal of Nutritional Biochemistry, 2005. **16**(6): p. 353-359.

519. Tholstrup, T., et al., *Effects of butter high in ruminant trans and monounsaturated fatty acids on lipoproteins, incorporation of fatty acids into lipid classes, plasma C-reactive protein, oxidative stress, hemostatic variables, and insulin in healthy young men.* Am J Clin Nutr, 2006. **83**(2): p. 237-243.

520. MacLean, C.H., et al., *Effects of omega-3 fatty acids on cancer risk: a systematic review.* J Am Med Assoc, 2006. **295**(4): p. 403-15.

521. van de Rest, O., et al., *Effect of fish-oil supplementation on mental well-being in older subjects: a randomized, double-blind, placebo-controlled trial.* Am J Clin Nutr, 2008. **88**(3): p. 706-13.

522. Laitinen, M., et al., *Fat intake at midlife and risk of dementia and Alzheimer's disease: a population-based study.* Dementia and Geriatric Cognitive Disorders, 2006. **22**(1): p. 99-107.

523. Mamalakis, G., et al., *Depression and serum adiponectin and adipose omega-3 and omega-6 fatty acids in adolescents.* Pharmacology Biochemistry and Behavior, 2006. **85**(2): p. 474-479.

524. Morris, M.C., et al., *Consumption of fish and n-3 fatty acids and risk of incident Alzheimer disease.* Arch Neurol, 2003. **60**(7): p. 940-946.

525. Hiroi, M., et al., *The combination of genetic variations in the PRDX3 gene and dietary fat intake contribute to obesity risk.* Obesity (Silver Spring), 2010.

526. Fantuzzi, G., *Adipose tissue, adipokines, and inflammation.* J Allergy Clin Immunol, 2005. **115**(5): p. 911-919.

527. Jain, S.S. and R.P. Bird, *Elevated expression of tumor necrosis factor-alpha signaling molecules in colonic tumors of Zucker obese (fa/fa) rats.* Int J Cancer, 2010. **127**(9): p. 2042-50.

528. Renehan, A.G., et al., *Incident cancer burden attributable to excess body mass index in 30 European countries.* Int J Cancer, 2010. **126**(3): p. 692-702.

529. Alberti, K.G., et al., *Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity.* Circulation, 2009. **120**(16): p. 1640-5.

530. Baghurst K, *Dietary fats, marbling and human health.* Australian Journal of Experimental Agriculture, 2004. **44**: p. 635-644.

531. National Heart Foundation of Australia (National Blood Pressure and Vascular Disease Advisory Committee), *Guide to management of hypertension*, 2008, National Heart Foundation of Australia.

532. Sacks, F.M., et al., *The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels.* N Engl J Med, 1996. **335**(14): p. 1001-9.

533. Ramsden, C.E., et al., *n-6 fatty acid-specific and mixed polyunsaturate dietary interventions have different effects on CHD risk: a meta-analysis of randomised controlled trials.* Br J Nutr, 2010. **104**(11): p. 1586-600.

534. Brown, M.A., et al., *Dietary fat and carbohydrate composition: Metabolic Disease. In, Fat Detection: taste, texture and post ingestive effects*, F.i.N. Montmayeur J-P and leCoutre, Editor 2010, CRC Press Florida.

535. Galli, C. and P.C. Calder, *Effects of fat and fatty acid intake on inflammatory and immune responses: a critical review.* Ann Nutr Metab, 2009. **55**(1-3): p. 123-39.

536. Crawford, M.A., R.P. Bazinet, and A.J. Sinclair, *Fat intake and CNS functioning: ageing and disease.* Ann Nutr Metab, 2009. **55**(1-3): p. 202-28.

537. Daniels, S.R. and F.R. Greer, *Lipid screening and cardiovascular health in childhood.* Pediatrics, 2008. **122**(1): p. 198-208.

538. McGill, H.C., C.A. McMahan, and S.S. Gidding, *Are pediatricians responsible for prevention of adult cardiovascular disease?* Nat Clin Pract Cardiovasc Med, 2009. **6**(1): p. 10-1.

539. Niinikoski, H., et al., *Impact of repeated dietary counseling between infancy and 14 years of age on dietary intakes and serum lipids and lipoproteins: the STRIP study.* Circulation, 2007. **116**(9): p. 1032.

540. Clarke, R., et al., *Cholesterol fractions and apolipoproteins as risk factors for heart disease mortality in older men.* Arch Intern Med, 2007. **167**(13): p. 1373-8.

541. U.S. Department of Health, Education, and Welfare, *Healthy People - The Surgeon General's report on health promotion and disease prevention* 1979, Washington D.C.: U.S. Department of Health, Education and Welfare.

542. Chen, J., et al., *Metabolic syndrome and salt sensitivity of blood pressure in non-diabetic people in China: a dietary intervention study.[see comment].* Lancet, 2009. **373**(9666): p. 829-35.

543. Charlton, K.E., et al., *A food-based dietary strategy lowers blood pressure in a low socio-economic setting: a randomised study in South Africa.* Public Health Nutr, 2008. **11**(12): p. 1397-406.

544. China Salt Substitute Study Collaborative, G., *Salt substitution: a low-cost strategy for blood pressure control among rural Chinese. A randomized, controlled trial.[see comment].* J Hypertens, 2007. **25**(10): p. 2011-8.

545. Takahashi, Y., et al., *Blood pressure change in a free-living population-based dietary modification study in Japan.* J Hypertens, 2006. **24**(3): p. 451-458.

546. Robertson, J.I. and J.I.S. Robertson, *Dietary salt and hypertension: a scientific issue or a matter of faith?* Journal of Evaluation in Clinical Practice, 2003. **9**(1): p. 1-22.

547. Obarzanek, E., et al., *Individual blood pressure responses to changes in salt intake: results from the DASH-Sodium trial.[see comment].* Hypertension, 2003. **42**(4): p. 459-67.

548. Nowson, C.A., et al., *Low-sodium dietary approaches to stop hypertension-type diet including lean red meat lowers blood pressure in postmenopausal women.* Nutr Res, 2009. **29**(1): p. 8-18.

549. Melander, O., et al., *Moderate salt restriction effectively lowers blood pressure and degree of salt sensitivity is related to baseline concentration of renin and N-terminal atrial natriuretic peptide in plasma.* J Hypertens, 2007. **25**(3): p. 619-627.

550. Kawano, Y., et al., *Report of the working group for dietary salt reduction of the Japanese Society of Hypertension: (1) Rationale for salt restriction and salt-restriction target level for the management of hypertension.* Hypertension Research - Clinical & Experimental, 2007. **30**(10): p. 879-86.

551. Jürgens, G. and A. Graudal Niels, *Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterols, and triglyceride.* Cochrane Database Syst Rev, 2004(1).

552. Hooper, L., et al., *Advice to reduce dietary salt for prevention of cardiovascular disease.[update of Cochrane Database Syst Rev. 2003;(3):CD003656; PMID: 12917977].* Cochrane Database Syst Rev, 2004(1): p. CD003656.

553. He, F.J. and G.A. MacGregor, *Effect of longer-term modest salt reduction on blood pressure.* Cochrane Database Syst Rev, 2004(3): p. CD004937.

554. Geleijnse, J.M., F.J. Kok, and D.E. Grobbee, *Impact of dietary and lifestyle factors on the prevalence of hypertension in Western populations.* European Journal of Public Health, 2004. **14**(3): p. 235-239.

555. Geleijnse, J.M., F.J. Kok, and D.E. Grobbee, *Blood pressure response to changes in sodium and potassium intake: a metaregression analysis of randomised trials.* J Hum Hypertens, 2003. **17**(7): p. 471-80.

556. Dickinson, K.M., J.B. Keogh, and P.M. Clifton, *Effects of a low-salt diet on flow-mediated dilatation in humans.* Am J Clin Nutr, 2009. **89**(2): p. 485-490.

557. Dickinson, B.D. and S. Havas, *Reducing the population burden of cardiovascular disease by reducing sodium intake: a report of the council on science and public health.* Arch Intern Med, 2007. **167**(14): p. 1460-1468.

558. Geleijnse, J.M. and D.E. Grobbee, *High salt intake early in life: does it increase the risk of hypertension?* J Hypertens, 2002. **20**(11): p. 2121-2124.

559. Beevers, D.G., *The epidemiology of salt and hypertension.* Clinical Autonomic Research, 2002. **12**(5): p. 353-7.

560. Chang, H.Y., et al., *Effect of potassium-enriched salt on cardiovascular mortality and medical expenses of elderly men.* Am J Clin Nutr, 2006. **83**(6): p. 1289-96.

561. Cook, N.R., et al., *Long term effects of dietary sodium reduction on cardiovascular disease outcomes: observational follow-up of the trials of hypertension prevention (TOHP).* Br Med J, 2007. **334**(7599): p. 885-888.

562. Cook, N.R., et al., *Joint effects of sodium and potassium intake on subsequent cardiovascular disease: the Trials of Hypertension Prevention follow-up study.* Arch Intern Med, 2009. **169**(1): p. 32-40.

563. He, J., et al., *Dietary sodium intake and incidence of congestive heart failure in overweight US men and women: first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study.* Arch Intern Med, 2002. **162**(14): p. 1619-1624.

564. Reddy, K.S. and M.B. Katan, *Diet, nutrition and the prevention of hypertension and cardiovascular diseases... Diet, Nutrition and the Prevention of Chronic Diseases: scientific background papers of the Joint WHO/FAO Expert Consultation (Geneva, 28 January-1 February 2002).* Public Health Nutr, 2004. **7**(1A): p. 167-186.

565. Lawes, C.M., S. Vander Hoorn, and A. Rodgers, *Global burden of blood-pressure-related disease, 2001.* Lancet, 2008. **371**(9623): p. 1513-8.

566. Law, M.R., J.K. Morris, and N.J. Wald, *Use of blood pressure lowering drugs in the prevention of cardiovascular disease: meta-analysis of 147 randomised trials in the context of expectations from prospective epidemiological studies.* Br Med J, 2009. **338**: p. b1665.

567. Key, T.J., et al., *Diet, nutrition and the prevention of cancer.* Public Health Nutr, 2004. **7**(1A): p. 187-200.

568. Commonwealth Department of Health, *Dietary guidelines for Australians*, 1982: Canberra, ACT.

569. National Health and Medical Research Council, *Dietary Guidelines for Australians*, 1992, Commonwealth of Australia: Canberra.

570. Food Standards Australia and New Zealand, *Final assessment report - Proposal P230 - Consideration of mandatory fortification with iodine for New Zeland*, 2008, Food Standards Australia New Zealand.

571. Duley, L. and D. Henderson-Smart, *Reduced salt intake compared to normal dietary salt, or high intake, in pregnancy.* Cochrane Database Syst Rev, 1999(2): p. CD001687.

572. Australian Institute of Health and Welfare, *Health expenditure Australia 2007-08, cat. no. HWE 46*, 2009: Canberra, ACT.

573. Fiorito, L.M., et al., *Girls' early sweetened carbonated beverage intake predicts different patterns of beverage and nutrient intake across childhood and adolescence.* J Am Diet Assoc, 2010. **110**(4): p. 543-550.

574. Libuda, L., et al., *Pattern of beverage consumption and long-term association with body-weight status in German adolescents--results from the DONALD study.* Br J Nutr, 2008. **99**(6): p. 1370-9.

575. Bes-Rastrollo, M., et al., *Predictors of weight gain in a Mediterranean cohort: the Seguimiento Universidad de Navarra Study 1.* Am J Clin Nutr, 2006. **83**(2): p. 362-70; quiz 394-5.

576. Palmer, J.R., et al., *Sugar-sweetened beverages and incidence of type 2 diabetes mellitus in African American women.* Arch Intern Med, 2008. **168**(14): p. 1487-92.

577. Laville, M. and J.A. Nazare, *Diabetes, insulin resistance and sugars.* Obes Rev, 2009. **10**(Suppl 1): p. 24-33.

578. Esposito, K., et al., *Prevention of type 2 diabetes by dietary patterns: a systematic review of prospective studies and meta-analysis.* Metab Syndr Relat Disord, 2010. **8**(6): p. 471-6.

579. Malik, V.S., et al., *Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis.* Diabetes Care, 2010. **33**(11): p. 2477-83.

580. Mann, J.I., A.S. Truswell, and E.B. Manning, *Effects on serum lipids of reducing dietary sucrose or starch for 22 weeks in normal men.* S Afr Med J, 1972. **46**(25): p. 827-34.

581. Silvera, S.A., et al., *Dietary carbohydrates and breast cancer risk: a prospective study of the roles of overall glycemic index and glycemic load.[erratum appears in Int J Cancer. 2006 May 1;118(9):2372].* Int J Cancer 2005. **114**(4): p. 653-8.

582. Michaud, D.S., et al., *Dietary sugar, glycemic load, and pancreatic cancer risk in a prospective study.* Journal of the National Cancer Institute, 2002. **94**(17): p. 1293-300.

583. Michaud, D.S., et al., *Dietary glycemic load, carbohydrate, sugar, and colorectal cancer risk in men and women.* Cancer Epidemiol Biomarkers Prev, 2005. **14**(1): p. 138-47.

584. De Stefani, E., et al., *Dietary patterns and risk of bladder cancer: a factor analysis in Uruguay.* Cancer Causes Control, 2008. **19**(10): p. 1243-9.

585. Nothlings, U., et al., *Dietary glycemic load, added sugars, and carbohydrates as risk factors for pancreatic cancer: the Multiethnic Cohort Study.* Am J Clin Nutr, 2007. **86**(5): p. 1495-501.

586. World Cancer Research Fund and American Institute for Cancer Research, *Food, nutrition, and the prevention of cancer: a global perspective*, 1997, American Institute for Cancer Research: Washington DC.

587. Miller, W.D., *Dental caries.* Am J Dent Sci, 1883. **17**: p. 77-130.

588. Burt, B.A., et al., *The effects of sugars intake and frequency of ingestion on dental caries increment in a three-year longitudinal study.* J Dent Res, 1988. **67**(11): p. 1422-9.

589. Warren, J.J., et al., *A longitudinal study of dental caries risk among very young low SES children.* Community Dent Oral Epidemiol, 2009. **37**(2): p. 116-122.

590. Ruottinen, S., et al., *Sucrose intake since infancy and dental health in 10-year-old children.* Caries research, 2000. **38**(2): p. 142-148.

591. Marshall, T.A., et al., *Comparison of the intakes of sugars by young children with and without dental caries experience.* J Am Dent Assoc, 2007. **138**(1): p. 39.

592. Levy, S.M., et al., *Fluoride, beverages and dental caries in the primary dentition.* Caries Res, 2003. **37**(3): p. 157-65.

593. Anderson, C.A., et al., *Sucrose and dental caries: a review of the evidence.* Obes Rev, 2009. **10**(Suppl 1): p. 41-54.

594. Gibson, S. and S. Williams, *Dental caries in pre-school children: associations with social class, toothbrushing habit and consumption of sugars and sugar-containing foods. Further analysis of data from the National Diet and Nutrition Survey of children aged 1.5-4.5 years.* Caries research, 1999. **33**(2): p. 101.

595. American Academy of Pediatrics, *Clinical Practice Guideline: Diagnosis and Evaluation of the Child With Attention-Deficit/Hyperactivity Disorder.* Pediatrics, 2000. **105**(5).

596. Walsh, L.J., *Preventive dentistry for the general dental practitioner.* Aust Dent J, 2000. **45**(2): p. 76-82.

597. Balakrishnan, M., R.S. Simmonds, and J.R. Tagg, *Dental caries is a preventable infectious disease.* Aust Dent J, 2000. **45**(4): p. 235-245.

598. Mattes, R.D., *Dietary compensation by humans for supplemental energy provided as ethanol or carbohydrate in fluids.* Physiol Behav, 1996. **59**(1): p. 179-187.

599. Raben, A., et al., *Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects.* Am J Clin Nutr, 2002. **76**(4): p. 721-9.

600. Krebs-Smith, S.M., *Choose beverages and foods to moderate your intake of sugars: measurement requires quantification.* J Nutr, 2001. **131**(2S-1): p. 527S-535S.

601. Marriott, B.P., et al., *Intake of added sugars and selected nutrients in the United States, National Health and Nutrition Examination Survey (NHANES) 2003-2006.* Crit Rev Food Sci Nutr, 2010. **50**(3): p. 228-58.

602. Lam, M., et al., *Children's acceptance of xylitol-based foods.* Community Dent Oral Epidemiol, 2000. **28**(2): p. 97-101.

603. Davies, M.J., et al., *Dental caries among Australian Aboriginal, non-Aboriginal Australian-born, and overseas-born children.* Bull World Health Organ, 1997. **75**(3): p. 197.

604. Chikritzhs, T., K. Fillmore, and T. Stockwell, *A healthy dose of scepticism: Four good reasons to think again about protective effects of alcohol on coronary heart disease.* Drug and Alcohol Review, 2009. **28**(4): p. 441-444.

605. Calabria, B., et al., *Epidemiology of alcohol related burden of disease among Indigenous Australians.* Aust N Z J Public Health, 2010. **34**: p. S47-S51.

606. Chikritzhs, T.N., et al., *Mortality and life-years lost due to alcohol: a comparison of acute and chronic causes.* Med J Aust, 2001. **174**(6): p. 281-284.

607. Collins, D.J. and H.M. Lapsley, *The costs of tobacco, alcohol and illicit drug abuse to Australian society in 2004/05*, 2008, Commonwealth Department of Health and Ageing: Canberra.

608. Xiao, J., et al., *Impact of Alcohol on the Population of Western Australia*, D.o.H.W. Epidemiology Branch, Editor 2008.

609. National Health and Medical Research Council, *Australian Guidelines to Reduce Health Risks from Drinking Alcohol*, 2009, Commonwealth of Australia: Canberra.

610. Banwell, C., *How many standard drinks are there in a glass of wine?* Drug Alcohol Rev, 1999. **18**(1): p. 99-101.

611. Arriola, L., et al., *Alcohol intake and the risk of coronary heart disease in the Spanish EPIC cohort study.* Heart, 2010. **96**(2): p. 124-30.

612. Simons, L.A., et al., *Alcohol intake and survival in the elderly: a 77 month follow-up in the Dubbo study.* Aust N Z J Med, 1996. **26**(5): p. 662-70.

613. Cullen, K.J., M.W. Knuiman, and N.J. Ward, *Alcohol and mortality in Busselton, Western Australia.* Am J Epidemiol, 1993. **137**(2): p. 242-8.

614. Costanzo, S., et al., *Alcohol consumption and mortality in patients with cardiovascular disease: a meta-analysis.* J Am Coll Cardiol, 2010. **55**(13): p. 1339-47.

615. Ronksley, P.E., et al., *Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis.* BMJ, 2011. **342**: p. d671.

616. Roerecke, M. and J. Rehm, *Ischemic heart disease mortality and morbidity rates in former drinkers: a meta-analysis.* Am J Epidemiol, 2011. **173**(3): p. 245-258.

617. Schooling, C.M., et al., *Moderate alcohol use and mortality from ischaemic heart disease: a prospective study in older Chinese people.* PLoS One, 2008. **3**(6).

618. Sierksma, A., et al., *Moderate alcohol consumption reduces plasma C-reactive protein and fibrinogen levels; a randomized, diet-controlled intervention study.* Eur J Clin Nutr, 2002. **56**(11): p. 1130-6.

619. Badia, E., et al., *Decreased tumor necrosis factor-induced adhesion of human monocytes to endothelial cells after moderate alcohol consumption.* Am J Clin Nutr, 2004. **80**(1): p. 225-30.

620. Hansen, A.S., et al., *Effect of red wine and red grape extract on blood lipids, haemostatic factors, and other risk factors for cardiovascular disease.* Eur J Clin Nutr, 2005. **59**(3): p. 449-55.

621. Tsang, C., et al., *The influence of moderate red wine consumption on antioxidant status and indices of oxidative stress associated with CHD in healthy volunteers.* Br J Nutr, 2005. **93**(2): p. 233-240.

622. Avellone, G., et al., *Effects of moderate Sicilian red wine consumption on inflammatory biomarkers of atherosclerosis.* Eur J Clin Nutr, 2006. **60**(1): p. 41-7.

623. Addolorato, G., et al., *Effects of short-term moderate alcohol administration on oxidative stress and nutritional status in healthy males.* Appetite, 2008. **50**(1): p. 50-56.

624. Beulens, J.W.J., et al., *Moderate alcohol consumption and lipoprotein-associated phospholipase A2 activity.* Nutr Metab Cardiovasc Dis, 2008. **18**(8): p. 539-544.

625. Tousoulis, D., et al., *Acute effects of different alcoholic beverages on vascular endothelium, inflammatory markers and thrombosis fibrinolysis system.* Clinical Nutrition, 2008. **27**(4): p. 594-600.

626. Costanzo, S., et al., *Cardiovascular and overall mortality risk in relation to alcohol consumption in patients with cardiovascular disease.* Circulation, 2010. **121**(17): p. 1951.

627. Bagnardi, V., et al., *Does drinking pattern modify the effect of alcohol on the risk of coronary heart disease? Evidence from a meta-analysis.* Journal of epidemiology and community health, 2008. **62**(7): p. 615.

628. Ma, X.J., et al., *[Genetic characteristics of familial type 2 diabetes pedigrees: a preliminary analysis of 4468 persons from 715 pedigrees].* Zhonghua Yi Xue Za Zhi, 2008. **88**(36): p. 2541-3.

629. Taylor, B., et al., *Alcohol and hypertension: gender differences in dose–response relationships determined through systematic review and meta analysis.* Addiction, 2009. **104**(12): p. 1981-1990.

630. Savica, V., G. Bellinghieri, and J.D. Kopple, *The effect of nutrition on blood pressure.* Annu Rev Nutr, 2010. **30**: p. 365-401.

631. Hackam, D.G., et al., *The 2010 Canadian Hypertension Education Program recommendations for the management of hypertension: part 2 - therapy.* Can J Cardiol, 2010. **26**(5): p. 249-58.

632. National Stroke Foundation, *Clinical Guidelines for Acute Stroke Management*2007, Melbourne: National Stroke Foundation.

633. Allen, N.E., et al., *Moderate alcohol intake and cancer incidence in women.* J Natl Cancer Inst, 2009. **101**(5): p. 296-305.

634. Terry, M.B., et al., *Lifetime alcohol intake and breast cancer risk.* Ann Epidemiol, 2006. **16**(3): p. 230-40.

635. Petri, A.L., et al., *Alcohol intake, type of beverage, and risk of breast cancer in pre- and postmenopausal women.* Alcoholism: Clinical and Experimental Research, 2004. **28**(7): p. 1084-1090.

636. Newcomb, P.A., et al., *No difference between red wine or white wine consumption and breast cancer risk.* Cancer Epidemiol Biomarkers Prev, 2009. **18**(3): p. 1007-10.

637. Li, Y., et al., *Wine, liquor, beer and risk of breast cancer in a large population.* European Journal of Cancer, 2009. **45**(5): p. 843-50.

638. Deandrea, S., et al., *Alcohol and breast cancer risk defined by estrogen and progesterone receptor status: a case-control study.* Cancer Epidemiol Biomarkers Prev, 2008. **17**(8): p. 2025-8.

639. Bessaoud, F. and J.P. Daurès, *Patterns of alcohol (especially wine) consumption and breast cancer risk: a case-control study among a population in Southern France.* Annals of Epidemiology, 2008. **18**(6): p. 467-475.

640. Fuchs, C.S., et al., *The influence of folate and multivitamin use on the familial risk of colon cancer in women.* Cancer Epidemiol Biomarkers Prev, 2002. **11**(3): p. 227.

641. Bongaerts, B.W., et al., *Alcohol and the risk of colon and rectal cancer with mutations in the K-ras gene.* Alcohol, 2006. **38**(3): p. 147-54.

642. Purdue, M.P., et al., *Type of alcoholic beverage and risk of head and neck cancer–a pooled analysis within the INHANCE Consortium.* Am J Epidemiol, 2009. **169**(2): p. 132-42.

643. Liang, W., et al., *Mortality rate of alcoholic liver disease and risk of hospitalization for alcoholic liver cirrhosis, alcoholic hepatitis and alcoholic liver failure in Australia between 1993 and 2005.* Internal Medicine Journal, 2011. **41**(1a): p. 34-41.

644. Rehm, J., et al., *Alcohol as a risk factor for liver cirrhosis: a systematic review and meta analysis.* Drug and Alcohol Review, 2010. **29**(4): p. 437-445.

645. Stampfer, M.J., et al., *Effects of moderate alcohol consumption on cognitive function in women.* N Engl J Med, 2005. **352**(3): p. 245-53.

646. Solfrizzi, V., et al., *Alcohol consumption, mild cognitive impairment, and progression to dementia.* Neurology, 2007. **68**(21): p. 1790-9.

647. Ruitenberg, A., et al., *Alcohol consumption and risk of dementia: the Rotterdam Study.* Lancet, 2002. **359**(9303): p. 281-6.

648. Luchsinger, J.A., et al., *Alcohol intake and risk of dementia.* J Am Geriatr Soc, 2004. **52**(4): p. 540-6.

649. Letenneur, L., S. Larrieu, and P. Barberger-Gateau, *Alcohol and tobacco consumption as risk factors of dementia: a review of epidemiological studies.* Biomedecine & Pharmacotherapy, 2004. **58**(2): p. 95-99.

650. Harper, C., *The neuropathology of alcohol-specific brain damage, or does alcohol damage the brain?* Journal of Neuropathology & Experimental Neurology, 1998. **57**(2): p. 101.

651. Deng, J., et al., *A 2-year follow-up study of alcohol consumption and risk of dementia.* Clin Neurol Neurosurg, 2006. **108**(4): p. 378-83.

652. Brown, S.A. and S.F. Tapert, *Adolescence and the trajectory of alcohol use: basic to clinical studies.* Annals of the New York Academy of Sciences, 2004. **1021**(1): p. 234-244.

653. Cargiulo, T., *Understanding the health impact of alcohol dependence.* Am J Health Syst Pharm, 2007. **64**(5 Suppl 3): p. S5-11.

654. Chen, C.Y. and C.L. Storr, *Alcohol use and health-related quality of life among youth in Taiwan.* Journal of Adolescent Health, 2006. **39**(5): p. 752. e9-752. e16.

655. Deas, D. and E.S. Brown, *Adolescent substance abuse and psychiatric comorbidities.* J Clin Psychiatry, 2006. **67**(7): p. e02.

656. Turner, R.J. and A.G. Gil, *Psychiatric and substance use disorders in South Florida: racial/ethnic and gender contrasts in a young adult cohort.* Archives of General Psychiatry, 2002. **59**(1): p. 43.

657. National Health and Medical Research Council, *Australian Alcohol Guidelines: health risks and benefits*, 2001, Commonwealth of Australia: Canberra.

658. Truswell, A.S., *Australian experience with the Wernicke-Korsakoff syndrome.* Addiction, 2000. **95**(6): p. 829-32.

659. Rolland, S. and A.S. Truswell, *Wernicke-Korsakoff syndrome in Sydney hospitals after 6 years of thiamin enrichment of bread.* Public Health Nutr, 1998. **1**(2): p. 117-22.

660. Edenberg, H.J., *The genetics of alcohol metabolism: role of alcohol dehydrogenase and aldehyde dehydrogenase variants.* Alcohol Res Health, 2007. **30**(1): p. 5-13.

661. Whitfield, J.B. and N.G. Martin, *Alcohol consumption and alcohol pharmacokinetics: interactions within the normal population.* Alcoholism: Clinical and Experimental Research, 1994. **18**(2): p. 238-243.

662. Li, T.-K., et al., *Gender and ethnic differences in alcohol metabolism.* Alcoholism: Clinical and Experimental Research, 1998. **22**(3): p. 771-772.

663. Bellis, M.D., et al., *Prefrontal cortex, thalamus, and cerebellar volumes in adolescents and young adults with adolescent onset alcohol use disorders and comorbid mental disorders.* Alcohol Clin Exp Res, 2005. **29**(9): p. 1590-1600.

664. White, A.M. and H.S. Swartzwelder, *Hippocampal function during adolescence: a unique target of ethanol effects.* Annals of the New York Academy of Sciences, 2004. **1021**(1): p. 206-220.

665. Brinton, E.A., *Effects of ethanol intake on lipoproteins and atherosclerosis.* Curr Opin Lipidol, 2010. **21**(4): p. 346-51.

666. American Diabetes Association, *Nutrition recommendations and interventions for diabetes: a position statement of the American Diabetes Association.* Diabetes Care, 2007. **30**(Suppl 1): p. S48-65.

667. CMP Medica, *MIMS Annual 2007*, 2007, CMP Medica: St Leonards, NSW.

668. Lee, Y., et al., *Systematic review of health behavioral risks and cognitive health in older adults.* Int Psychogeriatr, 2010. **22**(2): p. 174-87.

669. Arntzen, K., et al., *Moderate wine consumption is associated with better cognitive test results: a 7 year follow up of 5033 subjects in the Tromsø Study.* Acta Neurologica Scandinavica, 2010. **122**: p. 23-29.

670. Australian Institute of Health and Welfare, *2007 National Drug Strategy Household Survey: detailed findings. Cat. no. PHE 107*, 2008, Australian Institute of Health and Welfare: Canberra.

671. Australian Bureau of Statistics, *Apparent Consumption of Alcohol, Australia, 2008-09. Cat. no. 4307.0.55.001*, 2010, Australian Bureau of Statistics: Canberra.

672. National Preventative Health Taskforce, *Australia: the healthiest country by 2020. A discussion paper*, 2008, Commonwealth of Australia: Canberra.

673. Giglia, R. and C. Binns, *Alcohol and lactation: a systematic review.* Nutr Diet, 2006. **63**(2): p. 103-116.

674. Giglia, R.C., et al., *The effect of alcohol intake on breastfeeding duration in Australian women.* Acta Paediatr, 2008. **97**(5): p. 624-9.

675. Aira, M., S. Hartikainen, and R. Sulkava, *Community prevalence of alcohol use and concomitant use of medication—a source of possible risk in the elderly aged 75 and older?* Int J Geriatr Psychiatry, 2005. **20**(7): p. 680-685.

676. Fletcher, P.C. and J.P. Hirdes, *Risk factors for accidental injuries within senior citizens' homes: analysis of the Canadian survey on ageing and independence.* Journal of Gerontological Nursing, 2005. **31**(2): p. 49.

677. Mukamal, K., et al., *Alcohol consumption, bone density, and hip fracture among older adults: the cardiovascular health study.* Osteoporosis international, 2007. **18**(5): p. 593-602.

678. Australian Institute of Health and Welfare, *The problem of osteoporotic hip fracture in Australia.*, 2010.

679. Cheyne, E.H., et al., *Influence of alcohol on cognitive performance during mild hypoglycaemia; implications for Type 1 diabetes.* Diabet Med, 2004. **21**(3): p. 230-7.

680. Tolman, K.G. and A.S. Dalpiaz, *Treatment of non-alcoholic fatty liver disease.* Ther Clin Risk Manag, 2007. **3**(6): p. 1153-63.

681. Ferreira, S.E., et al., *Effects of energy drink ingestion on alcohol intoxication.* Alcohol Clin Exp Res, 2006. **30**(4): p. 598-605.

682. Price, S.R., et al., *Energy drink co administration is associated with increased reported alcohol ingestion.* Drug and Alcohol Review, 2010. **29**(3): p. 331-333.

683. Thombs, D.L., et al., *Event-level analyses of energy drink consumption and alcohol intoxication in bar patrons.* Addict Behav, 2010. **35**(4): p. 325-30.

684. Weldy, D.L., *Risks of alcoholic energy drinks for youth.* J Am Board Fam Med, 2010. **23**(4): p. 555-8.

685. World Health Organization, *Obesity: preventing and managing the global epidemic. Report of a WHO consultation (WHO Technical Report Series No. 894)*, in *World Health Organ Tech Rep Ser*2000, World Health Organization: Geneva. p. i-xii, 1-253.

686. Caballero, B., *The global epidemic of obesity: an overview.* Epidemiol Rev, 2007. **29**: p. 1-5.

687. World Health Organization Expert Committee on Physical Status, *Physical status: the use and interpretation of anthropometry*, 1995, World Health Organization: Geneva.

688. World Health Organization, *Global strategy on diet, physical activity and health*, 2004, World Health Organization: Geneva.

689. Willett, W.C., W.H. Dietz, and G.A. Colditz, *Guidelines for healthy weight.* N Engl J Med, 1999. **341**(6): p. 427-34.

690. National Health and Medical Research Council, *Acting on Australia's weight: A strategic plan for the prevention of overweight and obesity*, 1997, Commonwealth of Australia: Canberra.

691. World Health Organization. *Global database on Body Mass Index: an interactive surveillance tool for monitoring nutrition transition* 2011 [cited 2011 18 May]; Available from: http://apps.who.int/bmi/index.jsp.

692. Hocking, S., et al., *The Western Australian Chief Health Officer's Report 2010*, 2010, Department of Health, WA: Perth.

693. Queensland Health, *Risk factor impact on the burden of disease and injury in Queensland, 2007*, 2010, Queensland Health.

694. Australian Bureau of Statistics, *National Health Survey: Summary of Results, 2007-2008*, 2010, Australian Bureau of Statistics: Canberra.

695. Institute of Medicine, *Bridging the evidence gap in obesity prevention:a framework to inform decision making*, 2010, Institute of Medicine: Washington DC.

696. Rokholm, B., J.L. Baker, and T.I. Sorensen, *The levelling off of the obesity epidemic since the year 1999–a review of evidence and perspectives.* Obes Rev, 2010. **11**(12): p. 835-46.

697. Abbott, R.A., et al., *Healthy Kids Queensland Survey 2006 — Summary Report*, Queensland Health, Editor 2007: Brisbane.

698. Hands, B., et al., *Physical activity and nutrition levels in Western Australian children and adolescents: report*, 2004, Western Australian Government: Perth.

699. Hardy, L., *SPANS 2010 - NSW Schools Physical Activity and Nutrition Survey - Executive Summary*, 2011, NSW Department of Health: Sydney.

700. Martin, K., et al., *Child and Adolescent Physical Activity and Nutrition Survey 2008: key findings*, 2008, Physical Activity Taskforce (Western Australia): Perth.

701. Olds, T.S., et al., *Trends in the prevalence of childhood overweight and obesity in Australia between 1985 and 2008.* Int J Obesity, 2010. **34**(1): p. 57-66.

702. Gill, T.P., et al., *Childhood obesity in Australia remains a widespread health concern that warrants population-wide prevention programs.* Med J Aust, 2009. **190**(3): p. 146-8.

703. Lobstein, T., L. Baur, and R. Uauy, *Obesity in children and young people: a crisis in public health.* Obes Rev, 2004. **5**(Suppl 1): p. 4-104.

704. Olshansky, S.J., et al., *A potential decline in life expectancy in the United States in the 21st century.* N Engl J Med, 2005. **352**(11): p. 1138-45.

705. Haby, M. and A. Markwick, *Future prevalence of overweight and obesity in Australian children and adolescents, 2005-2025*, 2008, Department of Human Services: Melbourne.

706. Prospective Studies Collaboration, et al., *Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies.* Lancet, 2009. **373**(9669): p. 1083-96.

707. Australian Institute of Health and Welfare, *Australia's health 2008. Cat. no. AUS 99*, 2008, Australian Institute of Health and Welfare: Canberra.

708. Barba, C., et al., *Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies.* Lancet, 2004. **363**(9403).

709. Flicker, L., et al., *Body mass index and survival in men and women aged 70 to 75.* J Am Geriatr Soc, 2010. **58**(2): p. 234-41.

710. National Health and Medical Research Council, *Clinical Practice Guidelines for the Management of Overweight and Obesity in Adults*, 2003, Commonwealth of Australia: Canberra.

711. de Onis, M., et al., *Development of a WHO growth reference for school-aged children and adolescents.* Bull World Health Organ, 2007. **85**(9): p. 660-7.

712. Cole, T.J., et al., *Establishing a standard definition for child overweight and obesity worldwide: international survey.* Br Med J, 2000. **320**(7244): p. 1240-3.

713. Cole, T.J., et al., *Body mass index cut offs to define thinness in children and adolescents: international survey.* Br Med J, 2007. **335**(7612): p. 194.

714. Withrow, D. and D.A. Alter, *The economic burden of obesity worldwide: a systematic review of the direct costs of obesity.* Obes Rev, 2011. **12**(2): p. 131-41.

715. Stevens, J., et al., *The effect of age on the association between body-mass index and mortality.* N Engl J Med, 1998. **338**(1): p. 1-7.

716. Diehr, P., et al., *Weight, mortality, years of healthy life, and active life expectancy in older adults.* J Am Geriatr Soc, 2008. **56**(1): p. 76-83.

717. Denney-Wilson, E., et al., *Body mass index, waist circumference, and chronic disease risk factors in Australian adolescents.* Arch Pediatr Adolesc Med, 2008. **162**(6): p. 566-73.

718. Clarke, P.J., et al., *Midlife health and socioeconomic consequences of persistent overweight across early adulthood: findings from a national survey of American adults (1986-2008).* Am J Epidemiol, 2010. **172**(5): p. 540-8.

719. Biro, F.M. and M. Wien, *Childhood obesity and adult morbidities.* Am J Clin Nutr, 2010. **91**(5): p. 1499S-1505S.

720. Oates, R.K., *Non-organic failure to thrive.* Aust Paediatr J, 1984. **20**(2): p. 95-100.

721. Pugliese, M.T., et al., *Parental health beliefs as a cause of nonorganic failure to thrive.* Pediatrics, 1987. **80**(2): p. 175-82.

722. National Eating Disorders Collaborative, *Eating disorders- prevention, treatment and management: an evidence review*, 2010, National Eating Disorders Collaborative 2010.

723. Stubbs, C.O. and A.J. Lee, *The obesity epidemic: both energy intake and physical activity contribute.* Med J Aust, 2004. **181**(9): p. 489-91.

724. Bensimhon, D.R., W.E. Kraus, and M.P. Donahue, *Obesity and physical activity: a review.* Am Heart J, 2006. **151**(3): p. 598-603.

725. Haskell, W.L., et al., *Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association.* Circulation, 2007. **116**(9): p. 1081-93.

726. Swinburn, B. and G. Egger, *The runaway weight gain train: too many accelerators, not enough brakes.* Br Med J, 2004. **329**(7468): p. 736-9.

727. Swinburn, B., G. Sacks, and E. Ravussin, *Increased food energy supply is more than sufficient to explain the US epidemic of obesity.* Am J Clin Nutr, 2009. **90**(6): p. 1453-6.

728. Swinburn, B.A., et al., *Diet, nutrition and the prevention of excess weight gain and obesity.* Public Health Nutr, 2004. **7**(1A): p. 123-46.

729. Sacks, G., B.A. Swinburn, and M.A. Lawrence, *A systematic policy approach to changing the food system and physical activity environments to prevent obesity.* Aust New Zealand Health Policy, 2008. **5**: p. 13.

730. Drewnowski, A. and S.E. Specter, *Poverty and obesity: the role of energy density and energy costs.* Am J Clin Nutr, 2004. **79**(1): p. 6-16.

731. Badland, H. and G. Schofield, *Transport, urban design, and physical activity: an evidence-based update.* Transportation Research Part D: Transport and Environment, 2005. **10**(3): p. 177-196.

732. Jackson, R.J. and C. Kochtitzky, *Creating a healthy environment: the impact of the built environment on public health*, 2001, Sprawl Watch: Washington DC.

733. Oliver, L.N. and M.V. Hayes, *Neighbourhood socio-economic status and the prevalence of overweight Canadian children and youth.* Can J Public Health, 2005. **96**(6): p. 415-20.

734. Gill, T., L. King, and K. Webb, *Best options for promoting healthy weight and preventing weight gain in NSW*, 2005, NSW Centre for Public Health Nutrition: Sydney.

735. Kumanyika, S.K., *The obesity epidemic: looking in the mirror.* Am J Epidemiol, 2007. **166**(3): p. 243-5.

736. Ebbeling, C.B., D.B. Pawlak, and D.S. Ludwig, *Childhood obesity: public-health crisis, common sense cure.* Lancet, 2002. **360**(9331): p. 473-82.

737. McLaren, L., *Socioeconomic status and obesity.* Epidemiol Rev, 2007. **29**: p. 29-48.

738. Papas, M.A., et al., *The built environment and obesity.* Epidemiol Rev, 2007. **29**: p. 129-43.

739. National Obesity Taskforce, *Healthy Weight 2008 - Australia's future - The national action agenda for children and young people and their families*, 2003, Commonwealth of Australia: Canberra.

740. Commonwealth Department of Community Services and Health, *National dietary survey of schoolchildren (10-15 years), 1985. Report no. 1. Foods consumed*, 1988, Australian Government Publishing Service: Canberra.

741. Commonwealth Department of Community Services and Health, *National dietary survey of schoolchildren (10-15 years), 1985. Report no. 2. Nutrient intakes* 1989, Australian Government Publishing Service: Canberra.

742. Commonwealth Department of Community Services and Health in collaboration with the National Heart Foundation, *National dietary survey of adults: 1983. No. 2. Nutrient intakes*, 1987, Australian Government Printing Service: Canberra.

743. Commonwealth Department of Health in collaboration with the National Heart Foundation, *National dietary survey of adults: 1983 : No. 1. Foods consumed* 1986, Australian Government Publishing Service: Canberra.

744. Rangan AM, S.S., Hector DJ, Gill TP, Webb KL., *Consumption of 'extra' foods by Australian adults: types, quantities and contribution to energy and nutrient intake.* Eur J Clin Nutr, 2009. **63**(Oct 29): p. 865-871.

745. Stubbs, C.O., *Determinants of the Global Obesity Epidemic: Contributions of Early Life, Nutrition, Physical Activity, Television, Smoking and Economic and Social Change*, in *New Developments in Obesity Research*, L.A. Melrose, Editor 2006, Nova Publishers: New York. p. 121-152.

746. Prentice, A.M., et al., *Energy expenditure in overweight and obese adults in affluent societies: an analysis of 319 doubly-labelled water measurements.* Eur J Clin Nutr, 1996. **50**(2): p. 93-7.

747. Gebel, K., et al., *Creating healthy environments: A review of links between the physical environment, physical activity and obesity*, NSW Health Department and NSW Centre for Overweight and Obesity, Editor 2005: Sydney

748. Armstrong, T., A. Bauman, and J. Davies, *Physical activity patterns of Australian adults. Results of the 1999 National Physical Activity Survey*, 2000, Australian Institute of Health and Welfare: Canberra.

749. Queensland Health, *The Health of Queenslanders 2010; third report of the Chief Health Officer Queensland*, Queensland Health, Editor 2010: Brisband, Queensland. p. 115.

750. Centre for Epidemiology and Research, *The health of the people of New South Wales - Report of the Chief Health Officer. Summary Report, 2010*, NSW Department of Health, Editor 2010, NSW Department of Health: Sydney, NSW.

751. Booth, M.L., et al., *Change in the prevalence of overweight and obesity among young Australians, 1969-1997.* Am J Clin Nutr, 2003. **77**(1): p. 29-36.

752. Booth, M.L., et al., *Methods of the NSW Schools Physical Activity and Nutrition Survey (SPANS).* J Sci Med Sport, 2005. **8**(3): p. 284-93.

753. Bauman, A., et al., *Getting Australia active: towards better practice for the promotion of physical activity*, 2002, National Public Health Partnership: Melbourne.

754. Woodcock, J., et al., *Non-vigorous physical activity and all-cause mortality: systematic review and meta-analysis of cohort studies.* Int J Epidemiol, 2011. **40**(1): p. 121-38.

755. Janssen, I. and A.G. Leblanc, *Systematic review of the health benefits of physical activity and fitness in school-aged children and youth.* Int J Behav Nutr Phys Act, 2010. **7**: p. 40.

756. Warburton, D.E., C.W. Nicol, and S.S. Bredin, *Health benefits of physical activity: the evidence.* Can Med Assoc J, 2006. **174**(6): p. 801-9.

757. Brown, W.J., et al., *Leisure time physical activity in Australian women: relationship with well being and symptoms.* Res Q Exerc Sport, 2000. **71**(3): p. 206-16.

758. Nelson, M.C. and P. Gordon-Larsen, *Physical activity and sedentary behavior patterns are associated with selected adolescent health risk behaviors.* Pediatrics, 2006. **117**(4): p. 1281-90.

759. Commonwealth Department of Health and Ageing. *Measure up - weight and waist measurements*. 2010 [cited 2011 27 March]; Available from: http://www.health.gov.au/internet/abhi/publishing.nsf/Content/Weight,+waist+circumference+and+BMI-lp.

760. Dunstan, D., et al., *Diabesity & Associated Disorders in Australia - 2000*, 2001, International Diabetes Institute: Melbourne.

761. Bautista-Castana, I., J. Doreste, and L. Serra-Majem, *Effectiveness of interventions in the prevention of childhood obesity (Structured abstract).* Eur J Epidemiol, 2004(7): p. 617-622.

762. Campbell, K.J. and K.D. Hesketh, *Strategies which aim to positively impact on weight, physical activity, diet and sedentary behaviours in children from zero to five years. A systematic review of the literature.* Obes Rev, 2007. **8**(4): p. 327-38.

763. Kamath, C.C., et al., *Clinical review: behavioral interventions to prevent childhood obesity: a systematic review and metaanalyses of randomized trials.* J Clin Endocrinol Metab, 2008. **93**(12): p. 4606-15.

764. Stice, E., H. Shaw, and C.N. Marti, *A meta-analytic review of obesity prevention programs for children and adolescents: the skinny on interventions that work.* Psychol Bull, 2006(5): p. 667-691.

765. Summerbell, C.D., et al., *Interventions for preventing obesity in children.* Cochrane Database Syst Rev, 2005(3).

766. Lemmens, V.E., et al., *A systematic review of the evidence regarding efficacy of obesity prevention interventions among adults.* Obes Rev, 2008. **9**(5): p. 446-55.

767. Orozco Leonardo, J., et al., *Exercise or exercise and diet for preventing type 2 diabetes mellitus.* Cochrane Database Syst Rev, 2008(3).

768. Food and Agriculture Organization of the United Nations and the World Health Organization, *FAO/WHO Expert Consultation on Fats and Fatty Acids in Human Nutrition*, 2008, Food and Agriculture Organization of the United Nations and the World Health Organization,.

769. Fiorito, L.M., et al., *Beverage intake of girls at age 5 y predicts adiposity and weight status in childhood and adolescence.* Am J Clin Nutr, 2009. **90**(4): p. 935-42.

770. Vrolix R, M.R., *Variability of the glycemic response to single food products in healthy subjects.* Contemporary Clinical Trials, 2010. **31**(1): p. 5-11.

771. Johnson, L., et al., *Reflections from a systematic review of dietary energy density and weight gain: is the inclusion of drinks valid?. [Review] [62 refs].* Obesity Reviews, 2009. **10**(6): p. 681-92.

772. Hite, A.H., et al., *In the face of contradictory evidence: report of the Dietary Guidelines for Americans Committee.* Nutrition, 2010. **26**(10): p. 915-24.

773. Owen, C.G., et al., *Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence.* Pediatrics, 2005. **115**(5): p. 1367-77.

774. Araujo, C.L., et al., *Effect of birth size and proportionality on BMI and skinfold thickness in early adolescence: prospective birth cohort study.* Eur J Clin Nutr, 2009. **63**(5): p. 634-9.

775. Kuh, D., et al., *Birth weight, childhood growth and abdominal obesity in adult life.* Int J Obes Relat Metab Disord, 2002. **26**(1): p. 40-7.

776. Monasta, L., et al., *Early-life determinants of overweight and obesity: a review of systematic reviews.* Obes Rev, 2010. **11**(10): p. 695-708.

777. Monteiro, P.O. and C.G. Victora, *Rapid growth in infancy and childhood and obesity in later life--a systematic review.* Obes Rev, 2005. **6**(2): p. 143-54.

778. Nader, P.R., et al., *Identifying risk for obesity in early childhood.* Pediatrics, 2006. **118**(3): p. e594-601.

779. Mamun, A.A., et al., *Does maternal smoking during pregnancy have a direct effect on future offspring obesity? Evidence from a prospective birth cohort study.* Am J Epidemiol, 2006. **164**(4): p. 317-25.

780. Landhuis, C.E., et al., *Childhood sleep time and long-term risk for obesity: a 32-year prospective birth cohort study.* Pediatrics, 2008. **122**(5): p. 955-60.

781. Marshall, S.J., et al., *Relationships between media use, body fatness and physical activity in children and youth: a meta-analysis. [Review] [76 refs].* International Journal of Obesity & Related Metabolic Disorders: Journal of the International Association for the Study of Obesity, 2004. **28**(10): p. 1238-46.

782. Rey-Lopez, J.P., et al., *Sedentary behaviour and obesity development in children and adolescents.* Nutr Metab Cardiovasc Dis, 2008. **18**(3): p. 242-51.

783. Biddle, S.J., et al., *Physical activity and sedentary behaviours in youth: issues and controversies.* J R Soc Promot Health, 2004. **124**(1): p. 29-33.

784. Coon, K.A. and K.L. Tucker, *Television and children's consumption patterns: a review of the literature.* Minerva Pediatr, 2002. **54**(5): p. 423-36.

785. Goldani, M.Z., et al., *Do early life factors influence body mass index in adolescents?* Braz J Med Biol Res, 2007. **40**(9): p. 1231-6.

786. Langenberg, C., et al., *Central and total obesity in middle aged men and women in relation to lifetime socioeconomic status: evidence from a national birth cohort.* J Epidemiol Community Health, 2003. **57**(10): p. 816-22.

787. Power, C., et al., *Life-course influences on health in British adults: effects of socio-economic position in childhood and adulthood.* Int J Epidemiol, 2007. **36**(3): p. 532-9.

788. Wright, C.M. and L. Parker, *Forty years on: the effect of deprivation on growth in two Newcastle birth cohorts.* Int J Epidemiol, 2004. **33**(1): p. 147-52.

789. Jyoti, D.F., E.A. Frongillo, and S.J. Jones, *Food insecurity affects school children's academic performance, weight gain, and social skills.* J Nutr, 2005. **135**(12): p. 2831-9.

790. Senf, J.H., C.M. Shisslak, and M.A. Crago, *Does dieting lead to weight gain? A four-year longitudinal study of middle school girls.* Obesity, 2006. **14**(12): p. 2236-41.

791. Field, A.E., et al., *Relation between dieting and weight change among preadolescents and adolescents.* Pediatrics, 2003. **112**(4): p. 900-6.

792. Mamun, A.A., et al., *Do childhood sleeping problems predict obesity in young adulthood? Evidence from a prospective birth cohort study.* Am J Epidemiol, 2007. **166**(12): p. 1368-73.

793. Taveras, E.M., et al., *Short sleep duration in infancy and risk of childhood overweight.* Arch Pediatr Adolesc Med, 2008. **162**(4): p. 305-11.

794. Timlin, M.T., et al., *Breakfast eating and weight change in a 5-year prospective analysis of adolescents: Project EAT (Eating Among Teens).* Pediatrics, 2008. **121**(3): p. e638-45.

795. Gilles, A., et al., *Comparing active pediatric obesity treatments using meta-analysis.* J Clin Child Adolesc Psychol, 2008. **37**(4): p. 886-92.

796. Young, K.M., et al., *A meta-analysis of family-behavioral weight-loss treatments for children.* Clin Psychol Rev, 2007. **27**(2): p. 240-9.

797. Berry, D., et al., *Family-based interventions for childhood obesity: a review.* Journal of family nursing, 2004. **10**(4): p. 429.

798. Kelly, S.A. and B.M. Melnyk, *Systematic review of multicomponent interventions with overweight middle adolescents: implications for clinical practice and research.* Worldviews Evid Based Nurs, 2008. **5**(3): p. 113-35.

799. Doak, C.M., et al., *The prevention of overweight and obesity in children and adolescents: a review of interventions and programmes.* Obes Rev, 2006. **7**(1): p. 111-36.

800. Cole, K., et al., *An integrative research review: effective school-based childhood overweight interventions.* J Spec Pediatr Nurs, 2006. **11**(3): p. 166-77.

801. Astrup, A., et al., *The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies.* Int J Obes Relat Metab Disord, 2000. **24**(12): p. 1545-52.

802. Willett, W.C. and R.L. Leibel, *Dietary fat is not a major determinant of body fat.* Am J Med, 2002. **113**(Suppl 9B): p. 47S-59S.

803. Prentice, A.M. and S.A. Jebb, *Fast foods, energy density and obesity: a possible mechanistic link.* Obes Rev, 2003. **4**(4): p. 187-94.

804. McCrory, M.A., et al., *A twin study of the effects of energy density and palatability on energy intake of individual foods.* Physiol Behav, 2006. **87**(3): p. 451-9.

805. Paddon-Jones, D., et al., *Protein, weight management, and satiety.* Am J Clin Nutr, 2008. **87**(5): p. 1558S-1561S.

806. Drewnowski, A. and N. Darmon, *The economics of obesity: dietary energy density and energy cost.* Am J Clin Nutr, 2005. **82**(1 Suppl): p. 265S-273S.

807. Bowman, S.A., et al., *Effects of fast-food consumption on energy intake and diet quality among children in a national household survey.* Pediatrics, 2004. **113**(1 Pt 1): p. 112-8.

808. Pereira, M.A., et al., *Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis.* Lancet, 2005. **365**(9453): p. 36-42.

809. Ledikwe, J.H., J.A. Ello-Martin, and B.J. Rolls, *Portion sizes and the obesity epidemic.* J Nutr, 2005. **135**(4): p. 905-9.

810. Cameron-Smith, D., S.A. Bilsborough, and T.C. Crowe, *Upsizing Australia's waistline: the dangers of "meal deals".* Med J Aust, 2002. **177**(11-12): p. 2-16.

811. Franz, M.J., et al., *Weight-loss outcomes: a systematic review and meta-analysis of weight-loss clinical trials with a minimum 1-year follow-up.* J Am Diet Assoc, 2007. **107**(10): p. 1755-67.

812. Goodpaster, B.H., et al., *Effects of diet and physical activity interventions on weight loss and cardiometabolic risk factors in severely obese adults: a randomized trial.* J Am Med Assoc, 2010. **304**(16): p. 1795-802.

813. Medibank Private Ltd, *Obesity in Australia: financial impacts and cost benefits of intervention*, 2010, Medibank Private Ltd.

814. Colagiuri, S., et al., *The cost of overweight and obesity in Australia.* Med J Aust, 2010. **192**(5): p. 260-4.

815. Marks, G.C., C. Coyne, and G. Pang, *Type 2 diabetes costs in Australia - the potential impact of changes in diet, physical activity and levels of obesity*, 2001, Australian Food and Nutrition Modelling Unit: Brisbane.

816. National Public Health Partnership, *Be Active Australia: A Framework for Health Sector Action for Physical Activity 2005–2010*, V. Department of Human Services, Editor 2005, National Public Health Partnership: Melbourne.

817. Hu, F.B., et al., *Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women.* J Am Med Assoc, 2003. **289**(14): p. 1785-91.

818. Martinez-Gonzalez, M.A., et al., *Physical inactivity, sedentary lifestyle and obesity in the European Union.* Int J Obes Relat Metab Disord, 1999. **23**(11): p. 1192-201.

819. Brown, W.J., Y.D. Miller, and R. Miller, *Sitting time and work patterns as indicators of overweight and obesity in Australian adults.* International Journal of Obesity & Related Metabolic Disorders: Journal of the International Association for the Study of Obesity, 2003. **27**(11): p. 1340-6.

820. Saris, W.H., et al., *How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement.* Obes Rev, 2003. **4**(2): p. 101-14.

821. Blair, S.N., M.J. LaMonte, and M.Z. Nichaman, *The evolution of physical activity recommendations: how much is enough?* Am J Clin Nutr, 2004. **79**(5).

822. Petersen, L., P. Schnohr, and T. Sørensen, *Longitudinal study of the long-term relation between physical activity and obesity in adults.* International Journal of Obesity, 2003. **28**(1): p. 105-112.

823. Schoeller, D.A., *But how much physical activity?* Am J Clin Nutr, 2003. **78**(4): p. 669-70.

824. Martinez-Gomez, D., et al., *Recommended levels of physical activity to avoid an excess of body fat in European adolescents: the HELENA Study.* Am J Prev Med, 2010. **39**(3): p. 203-11.

825. Caterson, I.D. and N. Finer, *Emerging pharmacotherapy for treating obesity and associated cardiometabolic risk.* Asia Pacific, 2006. **15**: p. 55-62.

826. National Health and Medical Research Council, * *Title to be confirmed once published - Obesity treatment guidelines*, 2011, National Health and Medical Research Council: Canberra.

827. Callaway, L.K., M.J. O'Callaghan, and H.D. McIntyre, *Barriers to addressing overweight and obesity before conception.* Med J Aust, 2009. **191**(8): p. 425-428.

828. Mamun, A.A., et al., *Associations of excess weight gain during pregnancy with long-term maternal overweight and obesity: evidence from 21 y postpartum follow-up.* Am J Clin Nutr, 2010. **91**(5): p. 1336-41.

829. Dodd, J.M., et al., *Antenatal interventions for overweight or obese pregnant women: a systematic review of randomised trials: a systematic review of randomised trials.* BJOG: An International Journal of Obstetrics & Gynaecology, 2010. **117**(11): p. 1316-1326.

830. Callaway, L.K., et al., *The prevalence and impact of overweight and obesity in an Australian obstetric population.* Med J Aust, 2006. **184**(2): p. 56-9.

831. Institute of Medicine, *Preventing Childhood Obesity: Health in the Balance*, 2005, Institute of Medicine of the National Academies: Washington DC.

832. Institute of Medicine, N.R.C., *Weight Gain During Pregnancy: Reexamining the Guidelines.* , 2009, The National Academies Press.: Washington, DC.

833. Budge, H., et al., *Maternal nutritional programming of fetal adipose tissue development: long-term consequences for later obesity.* Birth defects research. Part C, Embryo today: reviews, 2005. **75**(3): p. 193-9.

834. Boney, C.M., et al., *Metabolic syndrome in childhood: association with birth weight, maternal obesity, and gestational diabetes mellitus.* Pediatrics, 2005. **115**(3): p. e290-6.

835. Fraser, A., et al., *Associations of gestational weight gain with maternal body mass index, waist circumference, and blood pressure measured 16 y after pregnancy: the Avon Longitudinal Study of Parents and Children.* Am J Clin Nutr, 2011.

836. Kinnunen, T.I., et al., *Reducing postpartum weight retention--a pilot trial in primary health care.* Nutr J, 2007. **6**: p. 21.

837. Harding, J.E., *The nutritional basis of the fetal origins of adult disease.* Int J Epidemiol, 2001. **30**(1): p. 15.

838. Makrides, M., *Early childhood nutrition and cognitive outcome.* Proc Nutr Soc Aust., 1998. **22**: p. 216-222.

839. Cooper, C., et al., *Growth in infancy and bone mass in later life.* Ann Rheum Dis, 1997. **56**(1): p. 17-21.

840. Evans, W.J., *Effects of aging and exercise on nutrition needs of the elderly.* Nutr Rev, 1996. **54**(1 Pt 2): p. S35-9.

841. Morley, J.E. and W.A. van Staveren, *Undernutrition: diagnosis, causes, consequences and treatment*, in *Food for the ageing population*, M. Raats, L. de Groot, and W.A. van Staveren, Editors. 2009, Woodhead Publishing Limited: Cambridge.

842. Nowson, C., *Nutritional challenges in the elderly.* Australian Doctor, 2009(27 March): p. 25-30.

843. Corrada, M.M., et al., *Association of body mass index and weight change with all-cause mortality in the elderly.* Am J Epidemiol, 2006. **163**(10): p. 938-49.

844. Truswell, A.S., *Dietary guidance for older Australians.* Nutr Diet, 2009. **66**(4): p. 243-248.

845. Strandberg, T.E. and K.H. Pitkala, *Frailty in elderly people.* Lancet, 2007. **369**(9570): p. 1328-9.

846. Wahlqvist ML, K.-B.A., Ross K, Setter T, Tienboon T., *Growth and Ageing. In: Nutritrion and metabolism*, ed. M.I. Gibney M, Roche HM (eds)2001, London: The Nutrition Society.

847. Leggo, M., et al., *A quality improvement nutrition screening and intervention program available to Home and Community Care eligible clients.* Nutr Diet, 2008. **65**(2): p. 162-167.

848. Banks, M., et al., *Prevalence of malnutrition in adults in Queensland public hospitals and residential aged care facilities.* Nutr Diet, 2007. **64**(3): p. 172-178.

849. Nelson, M.E., et al., *Physical activity and public health in older adults: recommendation from the American College of Sports Medicine and the American Heart Association.* Circulation, 2007. **116**(9): p. 1094-105.

850. Commonwealth Department of Health and Ageing. *Physical Activity Guidelines*. 2010 [cited 2011 30 March]; Available from: http://www.health.gov.au/internet/main/publishing.nsf/content/health-pubhlth-strateg-phys-act-guidelines.

851. Gregg, E.W., M.A. Pereira, and C.J. Caspersen, *Physical activity, falls, and fractures among older adults: a review of the epidemiologic evidence.* J Am Geriatr Soc, 2000. **48**(8): p. 883-893.

852. Kujala, U.M., et al., *Physical activity and osteoporotic hip fracture risk in men.* Arch Intern Med, 2000. **160**(5): p. 705-8.

853. Glass, T.A., et al., *Population based study of social and productive activities as predictors of survival among elderly Americans.* Br Med J, 1999. **319**(7208): p. 478.

854. Cunningham, J. and D. Mackerras, *Occasional Paper - Overweight and Obesity Indigenous Australians 1994*, 1998, Australian Bureau of Statistics: Canberra.

855. O'Dea, K., *Body fat distribution and health outcome in Australian Aborigines.* Proc Nutr Soc Aust, 1987. **12**: p. 56-65.

856. Cunningham, J., et al., *Perceived weight versus Body Mass Index among urban Aboriginal Australians: do perceptions and measurements match?* Aust N Z J Public Health, 2008. **32**(2): p. 135-138.

857. Australian Institute of Health and Welfare, *Aboriginal and Torres Strait Islander Health Performance Framework, 2008 report: Detailed analyses.*, 2008, Australian Institute of Health and Welfare: Canberra.

858. Turrell, G., et al., *Socioeconomic differences in food purchasing behaviour and suggested implications for diet-related health promotion.* J Hum Nutr Diet, 2002. **15**(5): p. 355-64.

859. Maillot, M., et al., *Nutrient-dense food groups have high energy costs: an econometric approach to nutrient profiling.* J Nutr, 2007. **137**(7): p. 1815.

860. World Health Organization, *International Code of Marketing of Breast-milk Substitutes*, 1981, World Health Organization: Geneva.

861. Ip, S., et al., *Breastfeeding and maternal and infant health outcomes in developed countries.* Evid Rep Technol Assess (Full Rep), 2007(153): p. 1-186.

862. American Academy of Pediatrics, *Pediatric Nutrition Handbook*. Fifth ed, ed. R.E. Kleinman2004, Washington DC: American Academy of Pediatrics. 1178.

863. Prentice, A., *Constituents of human milk.* Food Nutr Bull, 1996. **17**: p. 305-312.

864. Centre for Epidemiology and Research, *2005-2006 Report on child health from the New South Wales Population Healthy Survey*, N.D.o. Health, Editor 2008a, NSW Department of Health: Sydney.

865. Akre, J., *Infant feeding. The physiological basis.* Bull World Health Organ, 1989(67 Suppl): p. 1-108.

866. Kramer, M. and R. Kakuma, *Optimal duration of exclusive breastfeeding.* Cochrane Database Syst Rev, 2002(1): p. CD003517.

867. Horta, B.L., et al., *Evidence on the long-term effects of breastfeeding.* Geneva: World Health Organization, 2007.

868. Anderson, J., K. Malley, and R. Snell, *Is 6 months still the best for exclusive breastfeeding and introduction of solids? A literature review with consideration to the risk of the development of allergies.* Breastfeeding review: professional publication of the Nursing Mothers' Association of Australia, 2009. **17**(2): p. 23.

869. Kramer, M.S., et al., *Effects of prolonged and exclusive breastfeeding on child height, weight, adiposity, and blood pressure at age 6.5 y: evidence from a large randomized trial.* Am J Clin Nutr, 2007. **86**(6): p. 1717.

870. Agostoni, C., et al., *Complementary feeding: a commentary by the ESPGHAN Committee on Nutrition.* J Pediatr Gastroenterol Nutr, 2008. **46**(1): p. 99.

871. World Health Organization, *Equity, social determinant and public health programmes*, E. Blas and A.S. Kurup, Editors. 2010, World Health Organization: Geneva.

872. Prescott, S.L., et al., *The importance of early complementary feeding in the development of oral tolerance: concerns and controversies.* Pediatr Allergy Immunol, 2008. **19**(5): p. 375-80.

873. Scott, J.A., et al., *Predictors of the early introduction of solid foods in infants: results of a cohort study.* BMC Pediatr, 2009. **9**: p. 60.

874. Britton, C., et al., *Support for breastfeeding mothers.* Cochrane Database Syst Rev, 2007(1): p. CD001141.

875. Chung, M., et al., *Interventions in primary care to promote breastfeeding: an evidence review for the U.S. Preventive Services Task Force.* Ann Intern Med, 2008. **149**(8): p. 565-82.

876. Hector, D. and L. King, *Interventions to encourage and support breastfeeding.* NSW Public Health Bull, 2005. **16**(3-4): p. 56-61.

877. Landers, M., R. Hughes, and K. Graham, *The Darling Downs Breastfeeding Study*1998, Toowoomba: Darling Downs Public Health Unit.

878. Scott, J. and C. Binns, *Breastfeeding: are boys missing out?* Birth (Berkeley, Calif.), 1999. **26**(4): p. 276.

879. Scott, J.A., et al., *Factors associated with the duration of breastfeeding amongst women in Perth, Australia.* Acta Paediatr, 1999. **88**(4): p. 416-21.

880. Scott, J.A., et al., *Psychosocial factors associated with the abandonment of breastfeeding prior to hospital discharge.* Journal of Human Lactation, 2001. **17**(1): p. 24.

881. Simopoulos, A.P., J. Oliveira, and I.D. Desai, *Behavioral and metabolic aspects of breastfeeding: international trends*1995: S Karger AG.

882. Kramer, M.S., et al., *Breastfeeding and infant growth: biology or bias?* Pediatrics, 2002. **110**(2 Pt 1): p. 343-7.

883. Kramer, M.S., et al., *Promotion of Breastfeeding Intervention Trial (PROBIT): a randomized trial in the Republic of Belarus.* J Am Med Assoc, 2001. **285**(4): p. 413-20.

884. Dewey, K.G., *Growth characteristics of breast-fed compared to formula-fed infants.* Biol Neonate, 1998. **74**(2): p. 94-105.

885. Demmelmair, H., J. von Rosen, and B. Koletzko, *Long-term consequences of early nutrition.* Early Hum Dev, 2006. **82**(8): p. 567-74.

886. Harder, T., et al., *Duration of breastfeeding and risk of overweight: a meta-analysis.* Am J Epidemiol, 2005. **162**(5): p. 397-403.

887. White House Task Force on Childhood Obesity, *Solving the problem of childhood obesity within a generation: report to the President*, 2010: Washington, DC.

888. Kuti, O., A.B. Adeyemi, and A.T. Owolabi, *Breast-feeding pattern and onset of menstruation among Yoruba mothers of South-west Nigeria.* European J. of Contraception and Reproductive Healthcare, 2007. **12**(4): p. 335-339.

889. Romero-Gutiérrez, G., et al., *Actual use of the lactational amenorrhoea method.* European J. of Contraception and Reproductive Healthcare, 2007. **12**(4): p. 340-344.

890. Türk, R., F. Terzio lu, and K. Ero lu, *The Use of Lactational Amenorrhea as A Method of Family Planning in Eastern Turkey and Influential Factors.* The Journal of Midwifery & Women's Health, 2010. **55**(1): p. e1-e7.

891. Ip, S., et al., *A summary of the Agency for Healthcare Research and Quality's evidence report on breastfeeding in developed countries.* Breastfeed Med, 2009. **4 Suppl 1**: p. S17-30.

892. Oddy, W.H., *The long-term effects of breastfeeding on asthma and atopic disease.* Adv Exp Med Biol, 2009. **639**: p. 237-51.

893. McGuire, W. and M. Anthony, *Donor human milk versus formula for preventing necrotising enterocolitis in preterm infants: systematic review.* Archives of Disease in Childhood Fetal and Neonatal Edition, 2003. **88**(1): p. F11.

894. Barclay, A.R., et al., *Systematic review: the role of breastfeeding in the development of pediatric inflammatory bowel disease.* J Pediatr, 2009. **155**(3): p. 421-6.

895. Quigley, M., et al., *Breastfeeding is associated with improved child cognitive development: evidence from the UK Millennium Cohort Study.* Journal of epidemiology and community health, 2009. **63**(Suppl 2): p. 8.

896. Medeiros, A.P.M., J.T.L. Ferreira, and C.M. Felício, *Correlation between feeding methods, non-nutritive sucking and orofacial behaviors.* Pró-Fono Revista de Atualização Científica, 2009. **21**(4): p. 315-319.

897. Allen, J. and D. Hector, *Benefits of breastfeeding.* N S W Public Health Bull, 2005. **16**(3-4): p. 42-6.

898. Sobhy, S.I. and N.A. Mohame, *The effect of early initiation of breast feeding on the amount of vaginal blood loss during the fourth stage of labor.* J Egypt Public Health Assoc, 2004. **79**(1-2): p. 1-12.

899. Chantry, C.J., P. Auinger, and R.S. Byrd, *Lactation among adolescent mothers and subsequent bone mineral density.* Arch Pediatr Adolesc Med, 2004. **158**(7): p. 650.

900. Dursun, N., et al., *Influence of duration of total breast-feeding on bone mineral density in a Turkish population: does the priority of risk factors differ from society to society?* Osteoporosis international, 2006. **17**(5): p. 651-655.

901. National Health and Medical Research Council, * *Title to be confirmed - Infant Feeding Literature Review*, 2011, Commonwealth of Australia: Canberra.

902. House of Representatives Standing Committee on Health and Ageing, *The Best Start - Report on the inquiry into the health benefits of breastfeeding*, 2007, Commonwealth of Australia: Canberra.

903. Kuan, L.W., et al., *Health system factors contributing to breastfeeding success.* Pediatrics, 1999. **104**(3): p. e28.

904. Hall Moran, V., et al., *A systematic review of the nature of support for breast-feeding adolescent mothers.* Midwifery, 2007. **23**(2): p. 157-71.

905. Volpe, E.M. and M. Bear, *Enhancing breastfeeding initiation in adolescent mothers through the Breastfeeding Educated and Supported Teen (BEST) Club.* Journal of Human Lactation, 2000. **16**(3): p. 196.

906. Cooklin, A.R., S.M. Donath, and L.H. Amir, *Maternal employment and breastfeeding: results from the longitudinal study of Australian children.* Acta Paediatr, 2008. **97**(5): p. 620-3.

907. Baxter, J., A.R. Cooklin, and J. Smith, *Which mothers wean their babies prematurely from full breastfeeding? An Australian cohort study.* Acta Paediatr, 2009. **98**(8): p. 1274-1277.

908. Amir, L.H. and S.M. Donath, *Socioeconomic status and rates of breastfeeding in Australia: evidence from three recent national health surveys.* Med J Aust, 2008. **189**(5): p. 254-6.

909. Labbok, M.H., D. Clark, and A.S. Goldman, *Breastfeeding: maintaining an irreplaceable immunological resource.* Nat Rev Immunol, 2004. **4**(7): p. 565-572.

910. Scott, J., et al., *Factors associated with breastfeeding at discharge and duration of breastfeeding.* Journal of paediatrics and child health, 2001. **37**(3): p. 254-261.

911. Duong, D.V., C.W. Binns, and A.H. Lee, *Breast-feeding initiation and exclusive breast-feeding in rural Vietnam.* Public Health Nutr, 2004. **7**(6): p. 795-9.

912. Kaewsarn, P. and W. Moyle, *Breastfeeding duration of Thai women.* Aust Coll Midwives Inc J, 2000. **13**(1): p. 21-6.

913. Li, L., et al., *Factors associated with the initiation and duration of breastfeeding by Chinese mothers in Perth, Western Australia.* J Hum Lact, 2004. **20**(2): p. 188-95.

914. Utaka, H., et al., *Breastfeeding experiences of Japanese women living in Perth, Australia.* Breastfeed Rev, 2005. **13**(2): p. 5-11.

915. Xu, F., et al., *Breastfeeding in China: a review.* Int Breastfeed J, 2009. **4**: p. 6.

916. Binns, C.W., et al., *Breastfeeding by Aboriginal mothers in Perth.* Nutr Diet, 2006. **63**(1): p. 8-14.

917. World Health Organization, *Acceptable medical reasons for use of breastmilk substitutes*, 2009, World Health Organization,: Geneva.

918. Guise, J.M., et al., *The effectiveness of primary care-based interventions to promote breastfeeding: systematic evidence review and meta-analysis for the US Preventive Services Task Force.* Ann Fam Med, 2003. **1**(2): p. 70-8.

919. Baby Friendly Health Initiative. *The Ten Steps to Successful Breastfeeding*. 2011 [cited 2011 29 March]; Available from: http://www.bfhi.org.au/text/bfhi_ten_steps.html.

920. Binns, C. and J. Scott, *Breastfeeding: reasons for starting, reasons for stopping and problems along the way.* Breastfeed Rev, 2002. **10**(2): p. 13–19.

921. Australia New Zealand Food Authority, *Food safety standards: costs and benefits. An analysis of the regulatory impact of the proposed national food safety reforms* 1999, Canberra: Australia New Zealand Food Authority.

922. Fraser, A., *Bacterial toxins*, 2001, National Food Safety database <www.foodsafety.ufl.edu/consumer/nc/nc816a.htm>.

923. Australia New Zealand Food Authority, *Date marking: user guide to Standard 1.2. 5, Date Marking of Packaged Food*, 2001, Australia New Zealand Food Authority: Canberra.

924. Käferstein, F. and M. Abdussalam, *Food safety in the 21st century.* Bull World Health Organ, 1999. **77**(4): p. 347.

925. Desmarchelier, P.M., *Foodborne disease: emerging problems and solutions.* Med J Aust, 1996. **165**(11-12): p. 668-71.

926. Nyachuba, D.G., *Foodborne illness: is it on the rise?* Nutr Rev, 2010. **68**(5): p. 257-269.

927. Abelson, P., et al., *The annual cost of foodborne illness in Australia*2006: Department of Health and Ageing.

928. Crerar, S.K., et al., *Foodborne disease: current trends and future surveillance needs in Australia.* Med J Aust, 1996. **165**: p. 672-5.

929. Redmond, E.C. and C.J. Griffith, *Consumer food handling in the home: a review of food safety studies.* J Food Prot, 2003. **66**(1): p. 130-61.

930. Australia New Zealand Communicable Diseases Network, *Fortnightly notifiable diseases tables*, 1999, Communicable Diseases Intelligence,.

931. Redmond, E.C. and C.J. Griffith, *The importance of hygiene in the domestic kitchen: implications for preparation and storage of food and infant formula.* Perspectives in Public Health, 2009. **129**(2): p. 69-76.

932. State Government of Victoria, *Food safety at home.*, Department of Health Victoria, Editor 2011: Melbourne, Victoria.

933. Merry, G., *Food poisoning prevention*1997: Palgrave Macmillan Australia.

934. Sprenger, R.A., *Hygiene for management: a text for food hygiene courses*1998, South Yorkshire UK Highfield.

935. US Department of Agriculture, *Kitchen companion: your safe food guide*, 2008, US Department of Agriculture: Washington, DC. .

936. Cates, S.C., et al., *Pregnant women and listeriosis: preferred educational messages and delivery mechanisms.* J Nutr Educ Behav, 2004. **36**(3): p. 121-7.

937. Almanza, B.A., et al., *Clients' safe food-handling knowledge and risk behavior in a home-delivered meal program.* J Am Diet Assoc, 2007. **107**(5): p. 816-21.

938. Kendall, P.A., V.V. Hillers, and L.C. Medeiros, *Food safety guidance for older adults.* Clin Infect Dis, 2006. **42**(9): p. 1298-304.

939. National Health and Medical Research Council, *Implementing the dietary guidelines for Australians. Report on the subcommittee on nutrition education.*, 1989: Canberra.

940. Cashel K, L.J., *NUTTAB89-Nutrient Data Table for Use in Australia [Floppy Disc]*, Department of Community Services and Health, Editor 1989: Canberra.

941. National Health and Medical Research Council, *Recommended Dietary Intakes for use in Australia*, 1991, Commonwealth of Australia: Canberra.

942. National Health and Medical Research Council, *The Core Food Groups: the scientific basis for developing nutrition education tools*, 1995, Commonwealth of Australia: Canberra.

943. Sobolewski R, C.J., Mackerras D., *Which Australian food composition database should I use?* Nutr Dietetics, 2010. **67**: p. 37-40.

944. Food Standards Australia and New Zealand. *AUSNUT 2007*. 2007 September 2011 September 2011]; Available from: http://www.foodstandards.gov.au/consumerinformation/ausnut2007/.

945. National Public Health Partnership, *National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan 2000-2010*, 2001.

946. Food Standards Australia and New Zealand. *NUTTAB 2010*. 2010 September 2011 September 2011]; Available from: http://www.foodstandards.gov.au/consumerinformation/nuttab2010/.

947. Denney-Wilson, E., M. Booth, and L. Baur, *Development of the Australian standard definition of child/adolescent overweight and obesity.* Nutr Diet, 2003. **60**(2): p. 74-77.

948. World Health Organization. *Growth reference data for 5-19 years*. 2007 [cited 2011 30 March]; Available from: http://www.who.int/growthref/en/.

949. WHO Statistical Information System. *Children aged <5years*. 2008 [cited 2011 30 March]; Available from: http://www.who.int/whosis/indicators/compendium/2008/2nu5/en/.

950. World Health Organization. *The WHO Child Growth Standards*. 2006 [cited 2011 30 June]; Available from: http://www.who.int/childgrowth/en/.

951. Monasta, L., et al., *Defining overweight and obesity in pre-school children: IOTF reference or WHO standard?* Obes Rev, 2011. **12**(4): p. 295-300.

952. Centres for Disease Control and Prevention. *Growth charts*. 2010 [cited 2011 30 March]; Available from: http://www.cdc.gov/growthcharts/.

953. Dietz, W.H. and M.C. Bellizzi, *Introduction: the use of body mass index to assess obesity in children.* Am J Clin Nutr, 1999. **70**(1): p. 123S-5S.

954. Beyerlein, A., A.M. Toschke, and R. von Kries, *Risk factors for childhood overweight: shift of the mean body mass index and shift of the upper percentiles: results from a cross-sectional study.* Int J Obes (Lond), 2010. **34**(4): p. 642-8.

955. Lee, J.M., et al., *Getting heavier, younger: trajectories of obesity over the life course.* Int J Obes (Lond), 2010. **34**(4): p. 614-23.

956. McLennan, J., *Obesity in children. Tackling a growing problem.* Aust Fam Physician, 2004. **33**(1-2): p. 33-6.

957. Nambiar, S., et al., *Validating the waist height ratio and developing centiles for use amongst children and adolescents.* Acta Paediatr, 2009. **98**(1): p. 148-152.

958. Sanigorski, A., et al., *Reducing unhealthy weight gain in children through community capacity-building: results of a quasi-experimental intervention program, Be Active Eat Well.* Int J Obesity, 2008. **32**(7): p. 1060-1067.

959. Rasmussen-Torvik, L.J., et al., *Influence of waist on adiponectin and insulin sensitivity in adolescence.* Obesity (Silver Spring), 2009. **17**(1): p. 156-61.

960. Valery, P.C., et al., *Prevalence of obesity and metabolic syndrome in Indigenous Australian youths.* Obes Rev, 2009. **10**(3): p. 255-61.

961. McCarthy, H.D., *Body fat measurements in children as predictors for the metabolic syndrome: focus on waist circumference.* Proc Nutr Soc, 2006. **65**(04): p. 385-392.

962. Watts, K., et al., *Waist circumference predicts cardiovascular risk in young Australian children.* J Paediatr Child Health, 2008. **44**(12): p. 709-15.

963. Eisenmann, J.C., et al., *Fatness, fitness, and cardiovascular disease risk factors in children and adolescents.* Med Sci Sports Exerc, 2007. **39**(8): p. 1251.

964. Telford, R.D., et al., *Discordance of international adiposity classifications in Australian boys and girls - the LOOK study.* Ann Hum Biol, 2008. **35**(3): p. 334-41.

965. Dhaliwal, S.S. and T.A. Welborn, *Central obesity and multivariable cardiovascular risk as assessed by the Framingham prediction scores.* Am J Cardiol, 2009. **103**(10): p. 1403-7.

966. Welborn, T.A. and S.S. Dhaliwal, *Preferred clinical measures of central obesity for predicting mortality.* Eur J Clin Nutr, 2007. **61**(12): p. 1373-9.

967. Australian Government Department of Health and Ageing. *National Physical Activity Guidelines for Adults.* . 2005; Available from:

http://www.health.gov.au/internet/main/publishing.nsf/content/phd-physical-activity-adults-pdf-cnt.htm

968. Miller, Y.D. and D.W. Dunstan, *The effectiveness of physical activity interventions for the treatment of overweight and obesity and type 2 diabetes.* J Sci Med Sport, 2004. **7**(1 Suppl): p. 52-9.

969. Genkinger, J.M., et al., *Fruit, vegetable, and antioxidant intake and all-cause, cancer, and cardiovascular disease mortality in a community-dwelling population in Washington County, Maryland.* Am J Epidemiol, 2004. **160**(12): p. 1223.

970. Liu, Y., et al., *Vegetables, fruit consumption and risk of lung cancer among middle-aged Japanese men and women: JPHC study.* Cancer Causes Control, 2004. **15**(4): p. 349-357.

971. Smith-Warner, S.A., et al., *Fruits, vegetables and lung cancer: a pooled analysis of cohort studies.* Int J Cancer, 2003. **107**(6): p. 1001-11.

972. Wright, M.E., et al., *Intakes of fruit, vegetables, and specific botanical groups in relation to lung cancer risk in the NIH-AARP Diet and Health Study.* Am J Epidemiol, 2008. **168**(9): p. 1024-34.

973. Boyle, P. and B. Levin, *World Cancer Report 2008.* World Cancer Report 2008, 2008.

974. van Duijnhoven, F.J.B., et al., *Fruit, vegetables, and colorectal cancer risk: the European Prospective Investigation into Cancer and Nutrition.* Am J Clin Nutr, 2009. **89**(5): p. 1441.

975. Lee, J.E., et al., *Intakes of fruit, vegetables, and carotenoids and renal cell cancer risk: a pooled analysis of 13 prospective studies.* Cancer Epidemiol Biomarkers Prev, 2009. **18**(6): p. 1730-9.

976. Dolwick Grieb, S.M., et al., *Food groups and renal cell carcinoma: results from a case-control study.* J Am Diet Assoc, 2009. **109**(4): p. 656-67.

977. Weikert, S., et al., *Fruits and vegetables and renal cell carcinoma: findings from the European prospective investigation into cancer and nutrition (EPIC).* Int J Cancer 2006. **118**(12): p. 3133-3139.

978. Rashidkhani, B., et al., *Fruits, vegetables and risk of renal cell carcinoma: a prospective study of Swedish women.* Int J Cancer 2005. **113**(3): p. 451-5.

979. Stewart, B.W. and P. Kleihues, eds. *World Cancer Research Report*. 2003, International Agency for Research on Cancer: Lyon.

980. Boffetta, P., et al., *Fruit and vegetable intake and overall cancer risk in the European Prospective Investigation into Cancer and Nutrition (EPIC).* J Natl Cancer Inst, 2010. **102**(8): p. 529.

981. Liang, W. and C.W. Binns, *Fruit, vegetables, and colorectal cancer risk: the European Prospective Investigation into Cancer and Nutrition.* Am J Clin Nutr, 2009. **90**(4): p. 1112.

982. Miura, K., et al., *Relation of vegetable, fruit, and meat intake to 7-year blood pressure change in middle-aged men: the Chicago Western Electric Study.* Am J Epidemiol, 2004. **159**(6): p. 572-80.

983. Freedman, N.D., et al., *Fruit and vegetable intake and head and neck cancer risk in a large United States prospective cohort study.* Int J Cancer, 2008. **122**(10): p. 2330-6.

984. Ahn, Y.S., et al., *Increased platelet calcium in thrombosis and related disorders and its correction by nifedipine.* Thromb Res, 1987. **45**(2): p. 135-43.

985. Alberola, A., et al., *Crystal structures and magnetic properties of a sterically encumbered dithiadiazolyl radical, 2,4,6-(F3C)3C6H2CNSSN.* Chem Commun (Camb), 2005(37): p. 4726-8.

986. Atkins, G.J., et al., *Human osteoblasts are resistant to Apo2L/TRAIL-mediated apoptosis.* Bone, 2002. **31**(4): p. 448-56.

987. Sunny, L. and L. Sunny, *A low fat diet rich in fruits and vegetables may reduce the risk of developing prostate cancer.* Asian Pac J Cancer Prev, 2005. **6**(4): p. 490-6.

988. Ello-Martin, J.A., et al., *Dietary energy density in the treatment of obesity: a year-long trial comparing 2 weight-loss diets.* Am J Clin Nutr, 2007. **85**(6): p. 1465-1477.

989. Whybrow, S., et al., *Effects of added fruits and vegetables on dietary intakes and body weight in Scottish adults.* Br J Nutr, 2006. **95**(3): p. 496-503.

990. Vrieling, A., et al., *Fruit and vegetable consumption and pancreatic cancer risk in the European Prospective Investigation into Cancer and Nutrition.* Int J Cancer, 2009. **124**(8): p. 1926-1934.

991. Larsson, S.C., L. Bergkvist, and A. Wolk, *Fruit and vegetable consumption and incidence of gastric cancer: a prospective study.* Cancer Epidemiol Biomarkers Prev, 2006. **15**(10): p. 1998-2001.

992. Food And Agriculture Organization of the United Nations and the World Health Organization, *International Conference on Nutrition: World Declaration and Plan of Action for Nutrition.*, 1992, Food And Agriculture Organization of the United Nations and the World Health Organization: Geneva.

993. Lester, I.H., *Australia's food and nutrition*, 1994, Australian Government Publishing Service: Canberra.

994. PHAA, *Health inequities policy*, 2009, Public Health Association of Australia: Canberra.

995. Atlantis, E., E.H. Barnes, and K. Ball, *Weight status and perception barriers to healthy physical activity and diet behavior.* Int J Obes (Lond), 2008. **32**(2): p. 343-52.

996. Baker, E.A., et al., *The role of race and poverty in access to foods that enable individuals to adhere to dietary guidelines.* Prev Chronic Dis, 2006. **3**(3): p. A76.

997. Bodnar, L.M. and A.M. Siega-Riz, *A diet quality index for pregnancy detects variation in diet and differences by sociodemographic factors.* Public Health Nutr, 2002. **5**(6): p. 801-9.

998. Cassady, D., K.M. Jetter, and J. Culp, *Is price a barrier to eating more fruits and vegetables for low-income families?* J Am Diet Assoc, 2007. **107**(11): p. 1909-15.

999. Giskes, K., et al., *A multilevel study of socio-economic inequalities in food choice behaviour and dietary intake among the Dutch population: the GLOBE study.* Public Health Nutr, 2006. **9**(1): p. 75-83.

1000. Giskes, K., et al., *Socioeconomic inequalities in food purchasing: the contribution of respondent-perceived and actual (objectively measured) price and availability of foods.* Prev Med, 2007. **45**(1): p. 41-8.

1001. Kolodinsky, J., et al., *Knowledge of current dietary guidelines and food choice by college students: better eaters have higher knowledge of dietary guidance.* Journal of the American Dietetic Association, 2007. **107**(8): p. 1409-13.

1002. Inglis, V., K. Ball, and D. Crawford, *Why do women of low socioeconomic status have poorer dietary behaviours than women of higher socioeconomic status? A qualitative exploration.* Appetite, 2005. **45**(3): p. 334-43.

1003. McNaughton, S.A., et al., *An index of diet and eating patterns is a valid measure of diet quality in an Australian population.* J Nutr, 2008. **138**(1): p. 86-93.

1004. Nelson, M., K. Dick, and B. Holmes, *Food budget standards and dietary adequacy in low-income families.* Proc Nutr Soc, 2002. **61**(4): p. 569-77.

1005. Sanchez, A., et al., *Patterns and correlates of physical activity and nutrition behaviors in adolescents.* Am J Prev Med, 2007. **32**(2): p. 124-30.

1006. Story, M., D. Neumark-Sztainer, and S. French, *Individual and environmental influences on adolescent eating behaviors.* J Am Diet Assoc, 2002. **102**(3 Suppl): p. S40-51.

1007. Turrell, G. and A.M. Kavanagh, *Socio-economic pathways to diet: modelling the association between socio-economic position and food purchasing behaviour.* Public Health Nutr, 2006. **9**(3): p. 375-83.

1008. Turrell, G., et al., *Measuring socio-economic position in dietary research: is choice of socio-economic indicator important?* Public Health Nutr, 2003. **6**(2): p. 191-200.

1009. Collins, C.E., A.F. Young, and A. Hodge, *Diet quality is associated with higher nutrient intake and self-rated health in mid-aged women.* J Am Coll Nutr, 2008. **27**(1): p. 146-57.

1010. Dynesen, A.W., et al., *Sociodemographic differences in dietary habits described by food frequency questions--results from Denmark.* Eur J Clin Nutr, 2003. **57**(12): p. 1586-97.

1011. Hearty, A.P., et al., *Relationship between attitudes towards healthy eating and dietary behaviour, lifestyle and demographic factors in a representative sample of Irish adults.* Appetite, 2007. **48**(1): p. 1-11.

1012. Oldroyd, J., et al., *The effectiveness of nutrition interventions on dietary outcomes by relative social disadvantage: a systematic review.* J Epidemiol Community Health, 2008. **62**(7): p. 573-9.

1013. Power, E.M., *Determinants of healthy eating among low-income Canadians.* Can J Public Health, 2005. **96 Suppl 3**: p. S37-42, S42-8.

1014. Wong, K.C., et al., *Availability, affordability and quality of a healthy food basket in Adelaide, South Australia.* Nutr Diet, 2011. **68**(1): p. 8-14.

1015. Statistics, A.B.o., *National Aboriginal and Torres Strait Islander Survey 1994*, ABS, Editor 1994, Commonwealth of Australia: Canberra.

1016. Andrieu, E., N. Darmon, and A. Drewnowski, *Low-cost diets: more energy, fewer nutrients.* Eur J Clin Nutr, 2006. **60**(3): p. 434-6.

1017. Centre for Epidemiology and Research, *2007 report on adult health from the New South Wales Population Health Survey*, 2008b, NSW Department of Health: Sydney, NSW.

1018. Department of Human Services, *Victorian Population Health Survey 2007*, D.o.H. Services, Editor 2008: Melbourne.

1019. Burns, C., *A review of the literature describing the link between poverty, food insecurity and obesity with specific reference to Australia*, P.A. Unit, Editor 2004, Victorian Health Promotion Foundation: Melbourne.

1020. Australian Institute of Health and Welfare, *Aboriginal and Torres Strait Islander Health Performance Framework, 2006 report: detailed analyses.* , AIHW, Editor 2007, AIHW: Canberra.

1021. Australian Bureau of Statistics. *Consumer Price Index, Australia, March 2011 - Cat. no. 6401.0*. 2011 [cited 1 June 2011]; Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/6401.0Mar%202011?OpenDocument.

1022. Baghurst, K.I., *Social status, nutrition and the cost of healthy eating*, in *Dietary Guidelines for Australian Adults*, K.I. Baghurst and C. Binns, Editors. 2003, National Health and Medical Research Council: Canberra.

1023. Sanchez-Villegas, A., et al., *A systematic review of socioeconomic differences in food habits in Europe: consumption of cheese and milk.* Eur J Clin Nutr, 2003. **57**(8): p. 917-29.

1024. Vos, T., *The burden of disease and injury in Aboriginal and Torres Strait Islander peoples 2003.* 2007: University of Queensland Centre for Burden of Disease and Cost-Effectiveness.

1025. Carson, B., T. Dunbar, and R.D. Chenhall, *Social determinants of Indigenous health*2007: Allen & Unwin Academic.

1026. Torzillo, P.J., et al., *The state of health hardware in Aboriginal communities in rural and remote Australia.* Aust N Z J Public Health, 2008. **32**(1): p. 7-11.

1027. Australian Bureau of Statistics and Australian Institute of Health and Welfare, *The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples* 2008, Australian Institute of Health and Welfare: Canberra.

1028. Ruben, A.R. and A.C. Walker, *Malnutrition among rural aboriginal children in the Top End of the Northern Territory.* Med J Aust, 1995. **162**(8): p. 400.

1029. Sayers, S.M., et al., *An Australian Aboriginal birth cohort: a unique resource for a life course study of an Indigenous population. A study protocol.* BMC Int Health Hum Rights, 2003. **3**(1): p. 1.

1030. Northern Territory Government, *Department of Health and Community Services: Annual Report 2006-2007*, 2007, Department of Health and Community Services: Casuarina.

1031. Northern Territory Government, *Northern Territory Market Basket Survey 2008*, 2008, Northern Territory Department of Health and Families: Darwin.

1032. Mackerras, D.E., et al., *Growth and morbidity in children in the Aboriginal birth cohort study: the urban-remote differential.* Med J Aust, 2003. **178**(2): p. 56-60.

1033. Vanlint, S.J., et al., *Vitamin D insufficiency in Aboriginal Australians.* Women, 2011. **100**: p. 120.

1034. Coyne, T. and I. Darnton-Hill, *Australian Aborigines' nutrition and changing disease patterns.* New Doctor, 1979. **12**: p. 32-7.

1035. Naughton, J.M., K. O'Dea, and A.J. Sinclair, *Animal foods in traditional Australian aboriginal diets: polyunsaturated and low in fat.* Lipids, 1986. **21**(11): p. 684-90.

1036. O'Dea, K., *Traditional diet and food preferences of Australian Aboriginal hunter-gatherers.* Philos Trans R Soc Lond B Biol Sci, 1991. **334**(1270): p. 233-40; discussion 240-1.

1037. Shannon, C., *Acculturation: Aboriginal and Torres Strait Islander nutrition.* Asia Pac J Clin Nutr, 2002. **11 Suppl 3**: p. S576-8.

1038. Lee, A., *Transition of Australian Aboriginal diet and nutritional health. .* Wld Rev Nutr Diet, 1996. **79**: p. 1-52.

1039. Booth, S. and A. Smith, *Food security and poverty in Australia-challenges for dietitians.* Australian Journal of Nutrition and Dietetics, 2001. **58**(3): p. 150-156.

1040. House of Representatives Standing Committee on Aboriginal and Torres Strait Islander affairs, *Everybody's business: remote Aboriginal and Torres Strait community stores*, 2009, Commonwealth of Australia: Canberra.

1041. Leonard, D., R. Beilin, and M. Moran, *Which way kaikai blo umi? Food and nutrition in the Torres Strait.* Aust J Public Health, 1995. **19**(6): p. 589-595.

1042. Meedeniya, J., A. Smith, and P. Carter, *Food supply in rural South Australia: a survey on food cost, quality and variety.* 2000.

1043. Beaumont, S., *Tasmanian food price, availability, and quality survey*1998, Hobart: Tasmania Department of Community and Health Services.

1044. Bowcock, R., *Kimberley Market Basket Survey.* Derby: Kimberley Public Health, 1999.

1045. Price, R., et al., *Surveys of food availability, quality and price in rural and remote communities of the Alice Springs and Barkly district: April and May 1998.* Darwin: Territory Health Service, 1998.

1046. Sullivan, H., M. Gracey, and V. Hevron, *Food costs and nutrition of Aborigines in remote areas of northern Australia.* Med J Aust, 1987. **147**(7): p. 334-7.

1047. Cutter, T., *Nutrition and food habits of the central Australian Aboriginal.* The nutrition of Aborigines in relation to the ecosystem of central Australia. Melbourne: CSIRO, 1978: p. 63-70.

1048. House of Representatives, *Inquiry into community stores in remote Aboriginal and Torres Strait Islander communities 2009.*, 2009, Commonwealth of Australia: Canberra.

1049. Altman, J., *Hunter-gatherers today: An Aboriginal economy in north Australia*1987, Canberra: Australian Institute of Aboriginal Studies.

1050. Devitt, J., *Traditional Aboriginal preferences in a changed context.* Central Australian Rural Practitioners' Association Newsletter, 1991. **14**: p. 38-41.

1051. Rae, C., et al., *The role of bushfoods in contemporary Aboriginal diets.* Proc Nut Soc Aus, 1982. **7**: p. 45-9.

1052. Browne, J., S. Laurence, and S. Thorpe, *Acting on food insecurity in urban Aboriginal and Torres Strait Islander communities: Policy and practice interventions to improve local access and supply of nutritious food*, 2009, Victorian Aboriginal Community Controlled Health Organisation: Melbourne.

1053. Guest, C.S. and K. O'Dea, *Diabetes in aborigines and other Australian populations.* Aust J Public Health, 1992. **16**(4): p. 340-9.

1054. Doyle, J., et al., *Developing nutrition and physical activity guidelines and interventions for Aboriginal and Torres Strait Islander communities*, 2007, Onemda Vic Health Koori Health Research Unit and Department of Human Services.: Shepparton.

1055. Clough, A.R., K. Rowley, and K. O'Dea, *Kava use, dyslipidaemia and biomarkers of dietary quality in Aboriginal people in Arnhem Land in the Northern Territory (NT), Australia.* Eur J Clin Nutr, 2004. **58**(7): p. 1090-1093.

1056. Chan, L.C.K., et al., *Association between anthropometric measures of obesity and cardiovascular risk markers in a self-selected group of indigenous Australians.* Journal of Cardiovascular Risk, 2007. **14**(4): p. 515.

1057. Lee, A., Bonson (Bailey), APV., Yarmirr, D., Mathews, J., O'Dea, K. , *Sustainability of a successful Aboriginal community-based nutrition project.* Med J Aust 1995. **162**: p. 632-635.

1058. Rowley, K.G., et al., *Effectiveness of a community directed 'healthy lifestyle'program in a remote Australian Aboriginal community.* Aust N Z J Public Health, 2000. **24**(2): p. 136-144.

1059. Johannes, R.E. and J.W. MacFarlane, *Traditional fishing in the Torres Strait islands*1991: CSIRO Division of Fisheries, Marine Laboratories.

1060. Haynes D, M.-W.K., Kwan D., *Trace metal concentrations in Torres Strait green turtle (Chelonia mydas) tissues, 1997–1998.* , 2001, Report prepared for the Torres Strait Regional Authority.

1061. Cancer Council NSW, *NSW healthy food basket cost, availability and quality survey*, 2007, Cancer Council NSW: Sydney.