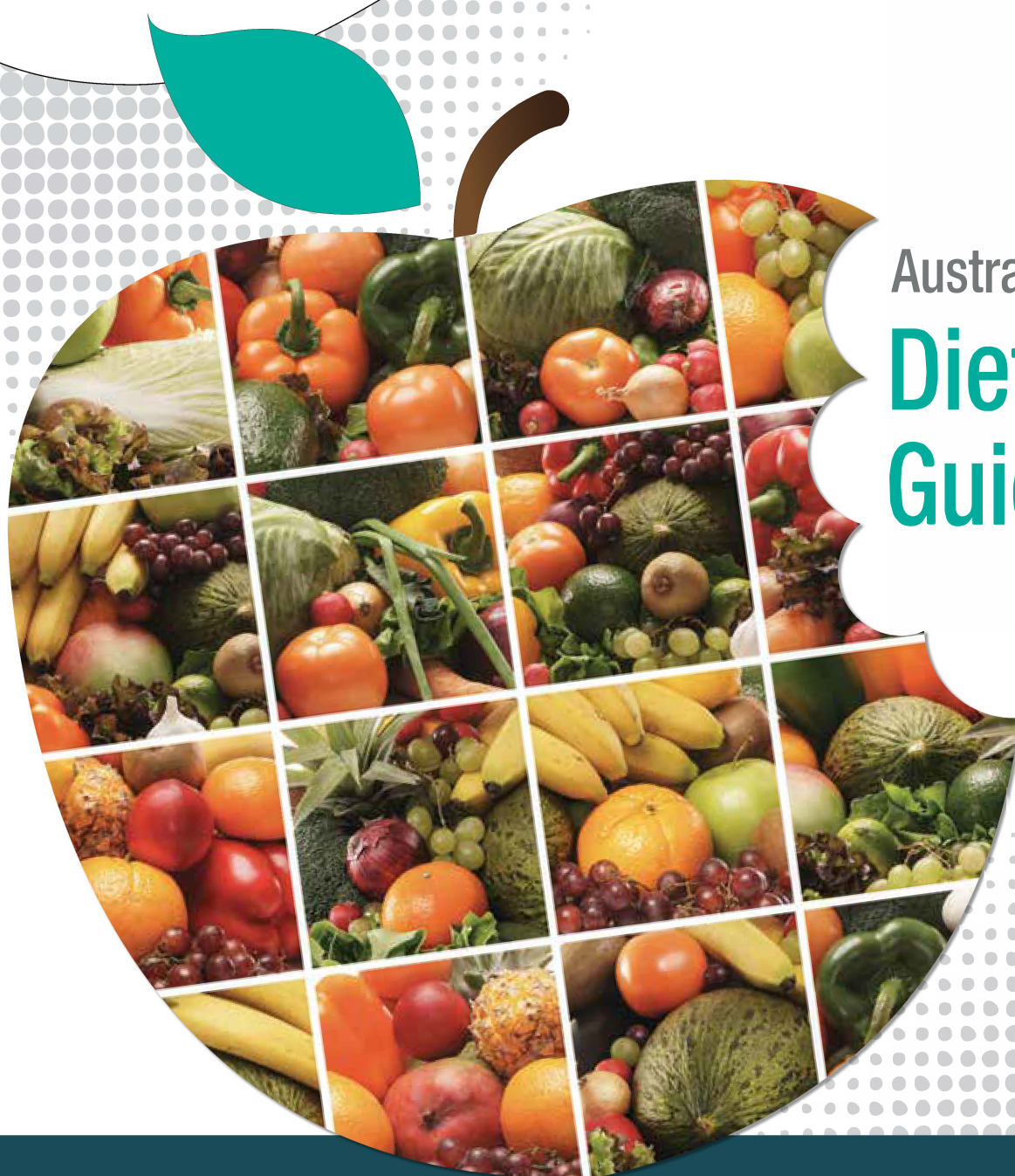




Australian Government
National Health and Medical Research Council
Department of Health and Ageing

EAT FOR HEALTH



Australian
**Dietary
Guidelines**

www.eatforhealth.gov.au



Australian Government

National Health and Medical Research Council

Department of Health and Ageing

EAT FOR HEALTH

Australian Dietary Guidelines

*Providing the scientific evidence for
healthier Australian diets*

2013

Publication Details

Publication title:	Australian Dietary Guidelines
Published:	February 2013
Publisher:	National Health and Medical Research Council
NHMRC Publication reference:	N55
Online version:	www.nhmrc.gov.au/guidelines-publications/n55
ISBN Online:	1864965754
ISBN Print:	1864965746
Suggested citation:	National Health and Medical Research Council (2013) <i>Australian Dietary Guidelines</i> . Canberra: National Health and Medical Research Council.
Disclaimer:	<p>This document is a general guide to appropriate practice, to be followed subject to the clinician's judgement and patient's preference in each individual case.</p> <p>The Guideline is designed to provide information to assist decision-making and is based on the best available evidence at the time of development of this publication.</p>

Copyright

© Commonwealth of Australia 2016



All material presented in this publication is provided under a Creative Commons Attribution 4.0 International licence (www.creativecommons.org.au), with the exception of the Commonwealth Coat of Arms, NHMRC logo and any content identified as being owned by third parties. The details of the relevant licence conditions are available on the Creative Commons website (www.creativecommons.org.au), as is the full legal code for the CC BY 4.0 International licence.

Attribution

Creative Commons Attribution 4.0 International Licence is a standard form licence agreement that allows you to copy, distribute, transmit and adapt this publication provided that you attribute the work. The NHMRC's preference is that you attribute this publication (and any material sourced from it) using the following wording: Source: National Health and Medical Research Council.

Use of images

Unless otherwise stated, all images (including background images, icons and illustrations) are copyrighted by their original owners.

The Australian Guide to Healthy Eating artwork must be reproduced in full, without alteration.

Contact us

To obtain information regarding NHMRC publications or submit a copyright request, contact:

E: nhmrc.publications@nhmrc.gov.au

P: (02) 6217 9000

Preface

Never in our nation's history have Australians had such a wide variety of dietary options. Yet the rising incidence of obesity and type 2 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 edition of the 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.

Choice of food in Australia has become more complex as we have become a more diverse society, and many Australians take into account issues such as environmental concerns or cultural priorities in making their choices. We hope that these Guidelines act as a firm basis upon which these individual decisions may be made.

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 AM

Chief Executive Officer
National Health and Medical Research Council

February 2013

Australian Dietary Guidelines

GUIDELINE 1

To achieve and maintain a healthy weight, 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 2

Enjoy 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 and/or high cereal fibre varieties, such as breads, cereals, rice, pasta, noodles, polenta, couscous, oats, quinoa and barley
- Lean meats and poultry, fish, eggs, tofu, 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 plenty of water.

GUIDELINE 3

Limit intake of foods containing saturated fat, added salt, added sugars and alcohol.

- a. Limit intake of foods high in saturated fat such as many biscuits, cakes, pastries, pies, processed meats, commercial burgers, pizza, fried foods, potato chips, crisps and other savoury snacks.
 - Replace high fat foods which contain predominantly saturated fats such as butter, cream, cooking margarine, coconut and palm oil with foods which contain predominantly polyunsaturated and monounsaturated fats such as oils, spreads, nut butters/pastes and avocado.
 - Low fat diets are not suitable for children under the age of 2 years.
- 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 in cooking or at the table.
- c. Limit intake of foods and drinks containing added sugars such as confectionary, sugar-sweetened soft drinks and cordials, fruit drinks, vitamin waters, energy and sports drinks.
- d. If you choose to drink alcohol, limit intake. For women who are pregnant, planning a pregnancy or breastfeeding, not drinking alcohol is the safest option.

GUIDELINE 4

Encourage, support and promote breastfeeding.

GUIDELINE 5

Care for your food; prepare and store it safely.

Contents

Preface	iii
Australian Dietary Guidelines	v
Introduction	1
Why the Guidelines matter	1
Scope and target audience	2
Companion documents	3
How the Guidelines were developed	5
Five key evidence sources for the Guidelines	5
How the evidence was used	7
Challenges for adoption of the Guidelines	8
How to use the Guidelines	9
 GUIDELINE 1	
■ Achieve and maintain a healthy weight	11
1.1 Setting the scene	12
1.1.1 Weight status and trends in Australia	12
1.1.2 Health effects associated with weight status	13
1.1.3 Causes of overweight and obesity in the population	14
1.1.4 Physical activity	17
1.2 The evidence for ‘achieve and maintain a healthy weight’	18
1.2.1 Primary and secondary prevention	19
1.2.2 Dietary patterns and specific foods and drinks	19
1.3 How dietary patterns can affect energy balance and weight outcomes	22
1.4 Practical considerations: achieve and maintain a healthy weight	22
1.4.1 Physical activity and dietary patterns in achieving and maintaining a healthy weight	23
1.4.2 Weight loss in adults who are overweight	24
1.4.3 Pregnant and breastfeeding women	24
1.4.4 Infants, children and adolescents	26
1.4.5 Older people	27
1.4.6 Aboriginal and Torres Strait Islander peoples	27
1.4.7 Australians of Asian origin	28
1.4.8 People with eating disorders	28
1.5 Practice guide for Guideline 1	29

GUIDELINE 2

■ Enjoy a wide variety of nutritious foods	31
2.1 Enjoy a wide variety of nutritious foods	32
2.1.1 Setting the scene	32
2.1.2 The evidence for consuming 'a wide variety of nutritious foods'	32
2.1.3 How consuming a wide variety of nutritious foods may improve health outcomes	33
2.1.4 Practical considerations: enjoy a wide variety of nutritious foods	33
2.2 Enjoy plenty of vegetables, including different types and colours, and legumes/beans, and enjoy fruit	36
2.2.1 Setting the scene	36
2.2.2 The evidence for consuming 'plenty of vegetables'	36
2.2.3 The evidence for consuming 'plenty of legumes/beans'	38
2.2.4 The evidence for consuming 'fruit'	39
2.2.5 How consuming plenty of vegetables, including different types and colours, and legumes/beans, and fruit may improve health outcomes	40
2.2.6 Practical considerations: enjoy plenty of vegetables, including different types and colours, legumes/beans, and enjoy fruit	42
2.3 Enjoy grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties	44
2.3.1 Setting the scene	44
2.3.2 The evidence for consuming 'grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties'	45
2.3.3 How consuming grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties may improve health outcomes	46
2.3.4 Practical considerations: enjoy grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties	46
2.4 Enjoy lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans	48
2.4.1 Setting the scene	48
2.4.2 The evidence for consuming 'lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans'	49
2.4.3 How consuming lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans may improve health outcomes	52
2.4.4 Practical considerations: enjoy lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans	52
2.5 Enjoy milk, yoghurt, cheese and/or alternatives, mostly reduced fat	56
2.5.1 Setting the scene	56
2.5.2 The evidence for consuming 'milk, yoghurt, cheese and/or alternatives, mostly reduced fat'	56
2.5.3 How consuming milk, yoghurt, cheese and/or alternatives, mostly reduced fat, may improve health outcomes	58
2.5.4 Practical considerations: enjoy milk, yoghurt, cheese and/or alternatives, mostly reduced fat	58
2.6 Drink plenty of water	61
2.6.1 Setting the scene	61
2.6.2 The evidence for 'drink plenty of water'	61
2.6.3 How drinking plenty of water may improve health outcomes	63
2.6.4 Practical considerations: drink plenty of water	63
2.7 Practice guide for Guideline 2	64

GUIDELINE 3

■ Limit intake of foods containing saturated fat, added salt, added sugars and alcohol	67
3.1 Limit intake of foods high in saturated fat	68
3.1.1 Setting the scene	68
3.1.2 The evidence for 'limit intake of foods high in saturated fat'	69
3.1.3 How limiting intake of foods high in saturated fat may improve health outcomes	71
3.1.4 Practical considerations: limit intake of foods high in saturated fat	71
3.2 Limit intake of foods and drinks containing added salt	73
3.2.1 Setting the scene	73
3.2.2 The evidence for 'limit intake of foods and drinks containing added salt'	74
3.2.3 How limiting intake of foods and drinks containing added salt may improve health outcomes	75
3.2.4 Practical considerations: Limit intake of foods and drinks containing added salt	75
3.3 Limit intake of foods and drinks containing added sugars	76
3.3.1 Setting the scene	76
3.3.2 The evidence for 'limit intake of foods and drinks containing added sugars'	77
3.3.3 How limiting intake of foods and drinks containing added sugars may improve health outcomes	78
3.3.4 Practical considerations: limit intake of foods and drinks containing added sugars	79
3.4 Limit intake of alcohol	80
3.4.1 Setting the scene	80
3.4.2 The evidence for 'limit intake of alcohol'	81
3.4.3 How limiting intake of alcohol may improve health outcomes	83
3.4.4 Practical considerations: limit intake of alcohol	83
3.5 Practice guide for Guideline 3	85

GUIDELINE 4

■ Encourage, support and promote breastfeeding	87
4.1 Setting the scene	88
4.2 The evidence for 'encourage, support and promote breastfeeding'	89
4.2.1 Breastfeeding incidence and duration	90
4.2.2 Infant growth	91
4.2.3 Cardiovascular disease and excess weight	92
4.2.4 Other benefits	92
4.3 Practical considerations: encourage, support and promote breastfeeding	93
4.3.1 Breastfeeding initiation and duration	93
4.3.2 Supporting and promoting breastfeeding	94
4.3.3 Safe storage of breast milk	95
4.3.4 Alcohol and breastfeeding	95
4.3.5 When an infant is not receiving breast milk	95
4.4 Practice guide for Guideline 4	96

GUIDELINE 5

■ Food safety	97
5.1 Setting the scene	98
5.2 The evidence for 'care for your food; prepare and store it safely'	98
5.2.1 Foods that may cause problems if not handled correctly	98
5.3 Why it is important to prepare and store food safely	99
5.4 Practical considerations: care for your food; prepare and store it safely	99
5.4.1 Pregnant and breastfeeding women	99
5.4.2 Infants	100
5.4.3 Adults with illness	100
5.4.4 Older people	100
5.5 Practice guide for Guideline 5	100

Appendices

A Equity and the social determinants of health and nutrition status	101
B Process report	109
C History and timeline of Australian nutrition documents	117
D Questions for the literature review to underpin the revision of the dietary guidelines	120
E Summary of evidence statements of negative associations and those of Grade D	124
F Evidence gradings used in the World Cancer Research Fund report	127
G Food, nutrition and environmental sustainability	130
H Assessing growth and healthy weight in infants, children and adolescents, and healthy weight in adults	135
I Physical activity guidelines	138
J Studies examining the health effects of intake of fruit and vegetables together	140
K Alcohol and energy intake	142

Glossary	143
----------	-----

Acronyms and abbreviations	151
----------------------------	-----

References	153
------------	-----

List of tables

Table 1.1:	Evidence statements on 'achieve and maintain a healthy weight'	18
Table 1.2:	2010 Institute of Medicine recommendations for total and rate of weight gain during pregnancy, by pre-pregnancy BMI	25
Table 1.3:	Weight gain during pregnancy: recommendations for Asian women, by pre-pregnancy BMI	25
Table 1.4:	Summary of practical considerations for Guideline 1	29
Table 2.1:	Evidence statements for consuming 'a wide variety of nutritious foods'	33
Table 2.2:	Evidence statements for consuming 'plenty of vegetables'	37
Table 2.3:	Evidence statements for consuming 'plenty of legumes/beans'	38
Table 2.4:	Evidence statements for consuming 'fruit'	39
Table 2.5:	Minimum recommended number of serves of vegetables, legumes/beans and fruit per day	42
Table 2.6:	Standard serve size equivalents for vegetables, legumes/beans and fruit	43
Table 2.7:	Evidence statements for consuming 'grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties'	45
Table 2.8:	Minimum recommended number of serves of grain (cereal) foods per day, mostly wholegrain and/or high cereal fibre varieties	47
Table 2.9:	Standard serve size equivalents for grain (cereal) mostly wholegrain and/or high cereal fibre varieties	47
Table 2.10:	Evidence statements for consuming 'lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans'	49
Table 2.11:	Minimum recommended number of serves of lean meats, poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans per day	53
Table 2.12:	Standard serve size equivalents for lean meats, poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans	53
Table 2.13:	Evidence statements for consuming 'milk, yoghurt, cheese and/or alternatives, mostly reduced fat'	56
Table 2.14:	Minimum recommended number of serves of milk, yoghurt, cheese and/or alternatives per day, mostly reduced fat	59
Table 2.15:	Standard serve size equivalents for milk, yoghurt, cheese and/or alternatives, mostly reduced fat	59
Table 2.16:	Evidence statements for 'drink plenty of water'	61
Table 2.17:	Considerations in advising people from specific groups to consume a wide variety of foods	64
Table 3.1:	Evidence statements for 'limit intake of foods high in saturated fat'	69
Table 3.2:	Recommended allowance for number of serves of unsaturated spreads and oils per day	72
Table 3.3:	Standard serve size equivalents of unsaturated spreads and oils	72
Table 3.4:	Evidence statements for 'limit intake of foods and drinks containing added salt'	74
Table 3.5:	Evidence statements for 'limit intake of foods and drinks containing added sugars'	77
Table 3.6:	Evidence statements for 'limit intake of alcohol'	81
Table 3.7:	Considerations in advising people from specific groups to limit intake of foods containing saturated fat, added salt, added sugars and alcohol	85
Table 4.1:	Evidence statements for 'encouraging, supporting and promoting breastfeeding'	89
Table 4.2:	Factors associated with duration of exclusive breastfeeding	91
Table 4.3:	Considerations in encouraging, supporting and promoting breastfeeding	96
Table 5.1:	Considerations in providing advice on food safety to people from specific groups	100
Table A.1:	Mean daily intakes (g/day) of various food groups, people aged 19 years and over, by SEIFA quintile	103

Table A2:	Mean daily intakes of energy and nutrient densities, adults aged 19 years and over, by SEIFA quintile	104
Table B1:	Terms of reference of the Dietary Guidelines Working Committee	109
Table B2:	Members of the Working Committee	110
Table B3:	Levels of evidence in the literature review	112
Table E1:	Grade C 'no association' relationships that informed the <i>Australian Dietary Guidelines</i>	124
Table E2:	Evidence statements (Grade D) that did not inform the <i>Australian Dietary Guidelines</i>	125
Table H1:	Practical points in the use of growth reference charts in infants aged 0–2 years	136
Table H2:	WHO body mass index classification	137
Table K1:	Median percentage of contribution of alcohol to energy intake per consumer	142
Table K2:	Energy and alcohol content of common alcoholic drink serves	142

List of figures

Figure 1.1:	Mean energy intakes of adults: 1983 and 1995	15
Figure 1.2:	Mean energy intakes of children aged 10–15 years: 1985 and 1995	16
Figure 1.3:	Mean consumption of selected food groups by boys aged 10–15 years: 1985 and 1995	17
Figure G1:	Examples of environmental consequences within the food system	132

Introduction

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 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.

Suboptimal nutrition is associated with ill health. Many diet-related chronic diseases such as cardiovascular disease, type 2 diabetes and some forms of cancer are major causes 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. The prevalence of type 2 diabetes is increasing and is expected to become Australia's leading cause of disease burden by 2023. Cancer is Australia's leading broad cause of disease burden (19%), followed by CVD (16%).³ The most recent available estimate for the total cost of poor nutrition was more than \$5 billion per year, based on 1990 costings.⁴ Given that the cost of obesity alone was estimated to be \$8.283 billion per year in 2008,⁵ 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 excess intake of energy-dense and relatively nutrient-poor foods high in energy, saturated fat, added or refined sugars or salt, and/or inadequate intake of nutrient-dense foods, including vegetables, fruit and wholegrain cereals.^{2,6} Deficiency in some nutrients such as iodine, folate,⁷ iron and vitamin D is also a concern for some Australians.^{8,9}

Overconsumption of some foods and drinks, leading to excess energy intake and consequent increases in adiposity, is now a key public health problem for Australia.^{6,10} The prevalence of overweight and obesity has increased dramatically over the past 30 years and is now around 60% in adults¹¹ and 25% in children and adolescents.^{11,12}

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 a Mediterranean dietary pattern and reduced mortality (Grade B; Evidence Report, Section 20.1).^{13–15} Previous studies have also indicated inverse associations between plant-based or vegetarian diets and all-cause and cardiovascular mortality, particularly among older adults.^{16–18} The effects of dietary quality tended to be greater for men than women, with common determinants being age, education and socioeconomic status.^{19,20}

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; Evidence Report, Section 20.3).^{19,20}

More recent evidence from Western societies confirms that dietary patterns consistent with 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.^{21,22} 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.²³

In relation to obesity, dietary advice 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 1).

Social determinants of food choices and health are important considerations

Life expectancy and health status are relatively high overall in Australia.^{11,24} Nonetheless, there are differences in health and wellbeing between Australians, including rates of death and disease, life expectancy, self-perceived health, health behaviours, health risk factors, and use of health services.²⁵⁻²⁷

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.^{25,27} 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.^{25,26} Conversely, a person's poor health status can contribute to social isolation and limit his or her ability to gain employment or education and earn an income, which can in turn have a negative impact on health determinants such as quality and stability of housing.

Social determinants of health and nutrition status are important considerations for users of these Guidelines, particularly when giving advice to individuals or groups in the community. Australians who are at greater risk of diet-mediated poor health include the very young, the very old, those living in remote areas, Aboriginal and Torres Strait Islander peoples, people from culturally and linguistically diverse groups and those in lower socioeconomic groups.²⁵⁻³⁰ The Guidelines address some of the issues faced by these population groups, in the 'Practical considerations' section for each Guideline. Further discussion of social determinants of food choices and health is included in Appendix A.

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 (see Appendix B for further information on how the Guidelines were developed). They are important tools that 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 1992³¹ in considering health, wellbeing, equity and the environment. A brief history of the development of Australian nutrition documents and resources is included in Appendix C.

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.

The Guidelines do not apply to people with medical conditions requiring specialised dietary advice, or to frail elderly people who are at risk of malnutrition.

The Guidelines are based on whole foods

Foods exert certain health effects because of the nutrients they contain. As such, dietary recommendations are often couched in terms of individual nutrients (such as requirements of vitamins and minerals). However, people eat whole foods rather than single nutrients, so such advice 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 may have some increased nutrient requirements that are more difficult to meet through diet alone and supplementation may be required. This is noted in the 'Practical considerations' sections.

Regulation of the food supply, such as fortification, use of food additives or special dietary products, is dealt with by Food Standards Australia New Zealand (FSANZ) (see www.foodstandards.gov.au).

Target audience for the Guidelines

The target audience for the Guidelines comprises health professionals (including dietitians, nutritionists, general practitioners (GPs), nurses and lactation consultants), educators, government policy makers, the food industry and other interested parties.

Companion documents

The Guidelines form part of a suite of resources on nutrition and dietary guidance. Other documents in the suite include the following:

- *Nutrient Reference Values for Australia and New Zealand* (NRV Document) – This details quantitative nutrient reference values (NRVs) for different ages and genders. The NRVs detail the recommended amounts of macronutrients and micronutrients required to avoid deficiency, toxicity and chronic disease. As an example, the NRV Document would be the reference for finding out how much iron is needed by women aged between 19 and 30 years.
- *A modelling system to inform the revision of the Australian Guide to Healthy Eating* (Food Modelling System) – This describes a range of computer-generated diets that translate the NRVs into dietary patterns to describe some types, combinations and amounts of foods that can deliver nutrient requirements for each age and gender group of different height and activity levels in the Australian population. A range of models including omnivore, lacto-ovo vegetarian, pasta and rice-based dietary patterns were developed, and primarily omnivorous dietary patterns were used to inform the *Australian Guide to Healthy Eating* and companion resources.
- *A review of the evidence to address targeted questions to inform the revision of the Australian Dietary Guidelines* (Evidence Report) – This is a systematic approach to literature review relevant to targeted questions published in the peer-reviewed nutrition literature from 2002–2009. As an example, the Evidence Report would be the reference for looking at the evidence for a particular evidence statement included in these Guidelines.
- *Infant Feeding Guidelines* – This document aims to support optimum infant nutrition by providing health workers with a review of the evidence, and clear evidence-based recommendations on infant feeding. It is relevant to healthy, term infants of normal birth weight (>2,500g).
- *Australian Guide to Healthy Eating* – This is the food selection tool (see page 10) which reflects dietary patterns divided into portions from the five food groups – fruit, vegetables, grains, milk, yoghurt and cheese products and lean meats (or alternatives), representing the proportion of these food groups required each day.
- Companion resources – These include an interactive website, summary booklets, brochures and posters for health professionals and consumers.

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

Eat for Health Program
www.eatforhealth.gov.au

Evidence products

- *A Review of the Evidence to Address Targeted Questions to Inform the Revision of the Australian Dietary Guidelines* (2011)
- *A Modelling System to Inform the Revision of the Australian Guide to Healthy Eating* (2011)
- *Review: Nutritional Requirements and Dietary Advice Targeted for Pregnant and Breastfeeding Women* (2013)
- *Infant Feeding Guidelines Literature Review* (2012)
- 2003 edition of the Dietary Guidelines (rescinded)

Guidelines

- *Australian Dietary Guidelines* (2013)
- *Infant Feeding Guidelines* (2012)

Health professional resources

- *Australian Dietary Guidelines*
- *Infant Feeding Guidelines*
- *Australian Guide to Healthy Eating* (Food Modelling Tool)
- *Educator Guide*
- Summary booklet for the *Australian Dietary Guidelines*
- Summary booklet for the *Infant Feeding Guidelines*
- Brochures for infants, children, pregnant women and adults
- Posters
- Interactive web tools
- Healthy eating information such as fact pages and tips

Consumer resources

- *Australian Guide to Healthy Eating* (Food Modelling Tool)
- Summary booklet for the *Australian Dietary Guidelines*
- Brochures for infants, children, pregnant women and adults
- Posters
- Interactive web tools
- Healthy eating information such as fact pages and tips

How the Guidelines were developed

These Guidelines are an evolution of the 2003 edition of the dietary guidelines and build 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 (e.g. 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.^{32,33} The main methods are summarised below and given in more detail in Appendix B.

The Guidelines were further informed by substantial advances in the methodology for guideline development and useability since publication of the previous edition of the 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 to the analysis of all studies combined.

Five key evidence sources for the Guidelines

In developing the Guidelines, NHMRC drew upon the following key sources of evidence:

- the previous series of dietary guidelines and their supporting documentation^{34,35}
- the Evidence Report³³
- the NRV Document⁸
- the Food Modelling System⁹
- key authoritative government reports and additional literature (including a commissioned review on pregnant and breastfeeding women).³⁷

Evidence Report – answers to key questions in the research literature

The NHMRC commissioned a literature review to answer targeted questions on food, diet and disease/health relationships covering the period 2002–2009. This addressed specific questions developed by the expert Dietary Guidelines Working Committee (the Working Committee), where evidence might have changed since the 2003 edition of the dietary guidelines was developed (see Appendix D).³⁸

The NHMRC followed critical appraisal processes to ensure rigorous application of the review methodology.^{32,39} Data were extracted from included studies and assessed for strength of evidence, size of effect and relevance of evidence according to standardised NHMRC processes.^{32,40,42} 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.^{33,42}

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 evidence statement could be made. The individual studies in meta-analyses were considered as separate studies. The evidence statements were graded A to D according to standard NHMRC protocols.⁴²

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

In this way, the Evidence Report was used to develop the graded evidence statements included in these 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 the diets of individuals 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 effects 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, lifelong, randomised controlled trials with dietary manipulation to examine disease prevention.

As a result, evidence in the nutrition literature tends to be based on longer term observational studies, leading to a majority of Grade C evidence statements, with some reaching 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 applying this evidence for specific diet-disease relationships, particularly at the individual level.^{32,35}

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 these 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, but can be useful to inform health professionals about the strength of evidence from recent research. The full set of Grade D evidence statements can be found in the Evidence Report³³ and is summarised in Appendix E.

Food Modelling System – translating nutrient requirements into dietary patterns

The Food Modelling System was commissioned by the NHMRC between 2008 and 2010. It determines a range of combinations of amounts and types of foods that can 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. The modelling applies the NRV Document⁶ to meet all NRVs within energy requirements for all groups, including acceptable macronutrient distribution ranges and suggested dietary targets for all nutrients, with the exception of meeting the high iron requirements of pregnant girls and women and meeting vitamin D requirements (this is a special case as vitamin D is also produced in the body by the action of sunlight on the skin).

The resultant dietary models – Foundation Diets and Total Diets – demonstrate that while nutritional needs are met through the whole diet and not by single foods, the combination of foods is critical. The models are realistic and flexible in providing information on the serve sizes and minimum recommended number of daily serves required for each population group to achieve the required intake of vitamins, minerals and macronutrients.

Several processes contributed to 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 System informed the revision of the *Australian Guide to Healthy Eating* and companion resources and was considered together with other sources of evidence to determine the recommendations in these Guidelines.

Capturing new evidence

Nutrition research is continuously evolving and studies are published regularly. Relevant results from high quality studies (primarily systematic reviews) assessing food, diet and health relationships that were published after the literature review for the Evidence Report (i.e. 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 that provide other information ('Setting the scene', 'How eating a particular food may improve health outcomes', and 'Practical considerations' 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 grades, these sources were used to assist in refining translation of the evidence.

How the evidence was used

Getting the Guideline wording right

The final wording of each Guideline was developed by the Working Committee through a consensus approach, based on the information gained from the five key sources discussed above. Each Guideline is considered to be equally important in terms of public health outcomes.

For example, to translate all available evidence regarding consumption of vegetables and health outcomes into a guideline recommendation 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 outcomes³³
- the importance of vegetables as a source of key nutrients in the Australian diet from the Food Modelling System⁸ and the NRV Document²
- the relatively low energy content of vegetables^{2,9}
- findings of international authoritative reports including the World Cancer Research Fund report (WCRF Report) (see Appendix F)⁴³
- information provided in the 2003 edition of the dietary guidelines.³⁶

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

Using evidence statements

The graded evidence statements from the Evidence Report are included in the section titled 'Evidence' under each guideline recommendation. These sections also include relevant ungraded referenced evidence from the other four key evidence sources to ensure comprehensiveness and currency. The Evidence sections provide the basis of the scientific information that was translated to form the 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 recommendation was considered, along with potential mechanisms through which particular dietary patterns may influence health, to help assess the plausibility of the associations described in the Evidence sections. This information originated predominantly from the previous dietary guidelines series, updated by narrative reviews of additional literature sourced from authoritative reports, from the Food Modelling System,⁸ the NRV Document² and high quality studies published since the 2003 edition of the dietary guidelines.

Challenges for adoption of the Guidelines

Dietary patterns consistent with the Guidelines

Adherence to dietary recommendations in Australia is poor.⁴⁴ 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, sugar and salt exceeds recommendations.¹² Analysis of Australia's 1995 National Nutrition Survey⁴⁵ found that energy-dense, nutrient-poor 'extra foods'⁴⁶ contributed 41 % of the total daily energy intake of 2–18 year olds.⁴⁷

The most recent dietary data available for Australian adults (collected in the 1995 National Nutrition Survey) also showed a poor dietary pattern, with inadequate intake of vegetables, fruit, wholegrain cereals and milk, yoghurt and cheese products and alternatives, and higher than recommended proportions of fat intake derived from saturated fat.^{45,48} More than 35% of daily energy intake was derived from energy-dense nutrient-poor 'extra foods'.⁴⁷

Barriers to dietary patterns consistent with the Guidelines

Influences on dietary choices throughout life are complex, ranging from individual, physical and social factors through to societal and environmental factors.⁴⁹⁻⁵⁷

Possible barriers to compliance with recommendations may include the food environment (including availability and affordability), 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 (such as advertising and promotion of energy-dense nutrient-poor foods and drinks), and particular dietary preferences.^{58,59}

There appear to be complex relationships between dietary patterns established in childhood and dietary quality over time. Studies suggest that frequency of consuming takeaway food increases with age and is associated with higher intakes of energy, total fat, saturated fat and sodium,^{75-77,80} while frequency of breakfast consumption decreases with age and skipping breakfast is associated with reduced intake of calcium and dietary fibre.⁸¹ There is some evidence that family meal patterns during adolescence predict diet quality and meal patterns during early young adulthood.⁸² Childhood smoking is also associated with poor dietary habits,^{78,83} as is a stressful family life.^{86,87}

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 1995 National Nutrition Survey; however the 2011–13 Australian Health Survey⁸³ will provide a better understanding of the current diet and nutrition of Australians. This survey is collecting data on food and nutrient intake, dietary behaviours and supplement usage. Detailed data from the survey will be released from May 2013 for the general population and from June 2014 for the Aboriginal and Torres Strait Islander population.

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

Dietary choices and the environment

The food we eat has an impact on our personal health. The food system (including the production, processing, distribution and consumption of food and drinks) also has broader implications for the health of the environment and hence the ability of individuals to continue to enjoy a healthy diet. Numerous Australian Government agencies have responsibilities that cross all of these areas, and a range of activities is underway to monitor, better understand and improve the issues.

The concept of sustainable dietary patterns is not straightforward, as there are many complex interactions as food is grown, transported, sold, prepared, consumed and the remnants disposed. Considering a specifically Australian context is also important, as some food production and subsequent handling differs considerably to processes in North America and Europe, where much of the literature originates.

Nevertheless, many Australians consider it important to think about the environmental impact of their food choices, in the same way they make other lifestyle choices in the context of environmental impact.⁹⁴⁻⁹⁷ Increasingly, people seek advice from health professionals to help guide these decisions (see Appendix G).

Available Australian and international evidence cannot support specific advice on the environmental impact of particular food items or brands, but there are some practical steps that people can take, including:

- buy and consume foods and drinks that are consistent with these Guidelines
- avoid overconsumption
- minimise food wastage
- consider your food buying, storage, preparation and disposal practices
- minimise and recycle the packaging of food.

Choosing quantities of foods and drinks that are beyond an individual's energy needs is unhealthy; it also encourages an unnecessary use of natural resources, which increases the person's environmental footprint. Food wastage further increases the environmental burden. The general principles of these Guidelines are compatible with reducing environmental impact as well as promoting good health (see Appendix G).

How to use the Guidelines

This edition of the *Australian Dietary Guidelines* has been developed as a single comprehensive report covering all healthy Australians. Further consideration of dietary patterns for specific subpopulations are provided where there is a significant difference in dietary needs.

A consistent approach has been used throughout the Guidelines, with four main subheadings for each component of a guideline.

- *Setting the scene* – provides a brief background to the topic.
- *Evidence* – outlines the scientific evidence base from 2002–2009, 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.
- *Practical considerations* – identifies practical issues and health outcomes for subgroups within the population, including different life stages.

Practice guides are included to provide a summary of how each guideline can be put into practice.

Australian Guide to Healthy Eating



Australian Government
National Health and Medical Research Council
Department of Health and Ageing

www.eatforhealth.gov.au

Australian Guide to Healthy Eating

Enjoy a wide variety of nutritious foods from these five food groups every day.
Drink plenty of water.

Grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties



Vegetables and legumes/beans



Lean meats and poultry, fish, eggs, tofu, nuts and seeds and legumes/beans



Fruit



Milk, yoghurt, cheese and/or alternatives, mostly reduced fat



Use small amounts



Only sometimes and in small amounts



GUIDELINE 1

Achieve and maintain a healthy weight

Guideline 1

To achieve and maintain a healthy weight, 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.

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 a 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 suitable energy intake, within suitable macronutrient distribution ranges for protein, fat and carbohydrates. In general, the quantities of foods outlined in the *Australian Guide to Healthy Eating* and companion resources 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.

1.1 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.⁹⁸⁻¹⁰² While it is unhealthy to be underweight, overweight or obese,^{88,98} ideal weight varies from one person to another and at different stages of life. Persistent excess energy intake, whether small or large, can cause unwanted weight gain in people of any age.

There is widespread recognition that overweight and obesity constitute a global problem requiring urgent attention.^{89,98,103,104} Overweight and obesity contributed 7.5% of the burden of premature death and disability in Australia in 2003.ⁱ¹⁰ More recent data from Western Australia and Queensland indicate that overweight and obesity have now overtaken cigarette smoking as the single greatest contributing risk factor, being responsible for 8.3–8.6% of the total burden of disease and injury.^{i105,106}

As is the case internationally,¹⁰⁷ 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–\$56.6 billion^{5,108} with direct costs estimated at \$8–\$21 billion.^{5,109} It is predicted that by 2023, the projected health expenditure for type 2 diabetes will have risen \$1.4 billion to \$7 billion per year, due mostly to increasing weight gain.ⁱ¹⁰

1.1.1 Weight status and trends in Australia

The prevalence of overweight and obesity has increased significantly in Australia and most developed countries since the 1970s.^{98,104,110}

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.¹¹ Overweight and obesity were more common in men than women (68% versus 55%) and in people aged 65–74 years (75%) than in other age groups.

Children and adolescents

A number of national and state-based surveys of children and adolescents using measured height and weight data have found that 21–25% of children and adolescents are overweight or obese (with 5–8% classified as obese) and 2–5% are underweight.^{i11,12,111-113} The prevalence of obesity is higher for boys than girls (9% compared with 6%),¹¹ although the prevalence of combined overweight and obesity is similar (26% for boys and 24% for girls).ⁱ¹¹ The prevalence of overweight and obesity in children has increased significantly over the past two decades. Although the rate of increase appears to be slowing,^{111,112} the high prevalence remains of concern.ⁱ¹¹³

Childhood obesity has been identified as one of the most serious public health challenges of the 21st century.ⁱ¹¹⁴ In the United States (US) 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 that of their parents.ⁱ¹¹⁵ 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^{5,110} and cause a crisis in health and economic systems across the world.^{88,98,101}

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.ⁱ¹¹⁶

The predicted increases would significantly affect disease burden and health care costs, mostly due to an increased incidence of type 2 diabetes. 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.ⁱ¹⁰

How is healthy weight assessed?

The most common approach is the use of the *body mass index* (BMI) calculated as weight (in kilograms) divided by height (in metres) squared (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, with risk lowest in the healthy weight range.^{i100,104}

BMI categories for adults are as follows:^{104,117}

- $< 18.5 \text{ kg}/\text{m}^2$ – underweight
- 18.5 to $24.9 \text{ kg}/\text{m}^2$ – healthy weight
- 25.0 to $29.9 \text{ kg}/\text{m}^2$ – overweight
- $\geq 30.0 \text{ kg}/\text{m}^2$ – obese.

However, this classification may not be suitable for all population groups:^{i118,119}

- some groups may have equivalent levels of risk at a lower BMI (e.g. people of Asian origin) or higher BMI (e.g. people of Polynesian origin including Torres Strait Islanders and Māori)¹²⁰
- while specific BMI ranges have not been developed, Aboriginal people have a relatively high limb to trunk ratio and may have equivalent levels of risk at a lower BMI
- a higher BMI range may be desirable for people aged over 70 years.

Waist circumference is also used as a proxy for chronic disease risk in adults:^{i12,121}

- risk is increased at $\geq 80 \text{ cm}$ and high at $\geq 88 \text{ cm}$ for women and increased at $\geq 94 \text{ cm}$ and high at $\geq 102 \text{ cm}$ for men
- as with BMI, thresholds for other ethnic groups may differ from those for people of European descent.

For children and adolescents, continuing growth means that it is not possible to have a single set of numerical values for BMI cut-offs that apply to all ages and both sexes. Weight appropriateness is commonly assessed using age-related cut-off values^{122,123} or Z-scores (or standard deviation scores).ⁱ¹²⁴

For more information, see Appendix H.

1.1.2 Health effects associated with weight status

Adults

Overweight and obesity

Overweight and obesity are associated with increased risk of type 2 diabetes, cardiovascular disease, hypertension, metabolic syndrome, some cancers, musculoskeletal conditions, respiratory conditions, sleep apnoea, gall bladder disease, hernia, reproductive disorders, urinary incontinence, fatty liver disease, and depression and other mental health disorders.^{88,89,98,125} About 70% of people who are obese have at least one established comorbidity, resulting in medical costs that are about 30% greater than those of their healthy weight peers.¹⁰⁷

Compared to having a BMI of 18.5 – $25 \text{ kg}/\text{m}^2$, having a BMI of 30 – $35 \text{ kg}/\text{m}^2$ reduces life expectancy by 2–4 years, and having a BMI of 40 – $45 \text{ kg}/\text{m}^2$ reduces it by 8–10 years.ⁱ¹²⁶ The relative increase in mortality rate attributable to obesity tends to decline with age.¹²⁷ Mortality and morbidity are also associated with the amount of weight gained in adult life.^{89,98,101}

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.^{89,98,101}

Underweight

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.³⁶ Among older people, being underweight may be more deleterious to health than being overweight.¹²⁸

Inappropriate dietary restriction and eating disorders occur in some adults.¹²⁹

Children and adolescents

Overweight and obesity

The most immediate consequences of overweight and obesity in childhood are social discrimination (associated with poor self-esteem and depression), increased risk of developing negative body image issues, and eating disorders.^{101,130} Overweight children and adolescents are more likely to develop sleep apnoea, breathlessness on exertion and reduced exercise tolerance, some orthopaedic and gastrointestinal problems, non-alcoholic fatty liver disease, and early signs of metabolic and clinical consequences, such as hypertension, hyperinsulinaemia, hypertriglyceridaemia and type 2 diabetes.^{101,130}

A major long-term consequence is that overweight children are more likely to become overweight or obese adults, with an increased risk of chronic diseases and early mortality.^{101,130-132} The risk of chronic disease is increased with rapid weight gain in infancy and early childhood.^{88,89}

Underweight

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 conditions¹³³ but can also occur among affluent sections of the community due to inappropriate dietary restrictions (e.g. based on fears about 'unhealthy' dietary habits).¹³⁴ Specialist advice should be sought on underweight and failure to thrive in infants and children (for further information on growth see Appendix H).

Inappropriate dietary restriction and eating disorders occur in some adolescents.¹²⁹

1.1.3 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).^{89,101,135} At the population level, there is increasing evidence that excess energy intake is a major contributor to energy imbalance.^{88,135-140}

The obesogenic (obesity promoting) environment

Excess weight gain is directly and indirectly influenced by a wide range of factors, including inherited biological factors and early life experiences, as well as behavioural, environmental and social factors that influence individual behaviours. The relationships between these factors are complex and not yet fully understood.

Although genetic factors may increase an individual's susceptibility to being overweight or obese, the dramatic increase in prevalence of overweight and obesity over the past few decades suggests that socioenvironmental factors are a major contributor to the current epidemic.^{88,89,98,101,138,139,141} Significant 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-poor¹⁴²
- 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^{138,143}
- reduced perceptions of safety leading to fewer opportunities for physical activity^{144,145}
- economic and consumer changes, such as a greater number of women in the paid workforce, decreased food literacy and cooking skills, and greater reliance on convenience and takeaway foods.^{140,141,146}

These changes contribute to an 'obesogenic' environment, which promotes excess weight gain by fostering consumption of energy-dense and relatively nutrient-poor foods and/or reduced physical activity.^{133,147-150}

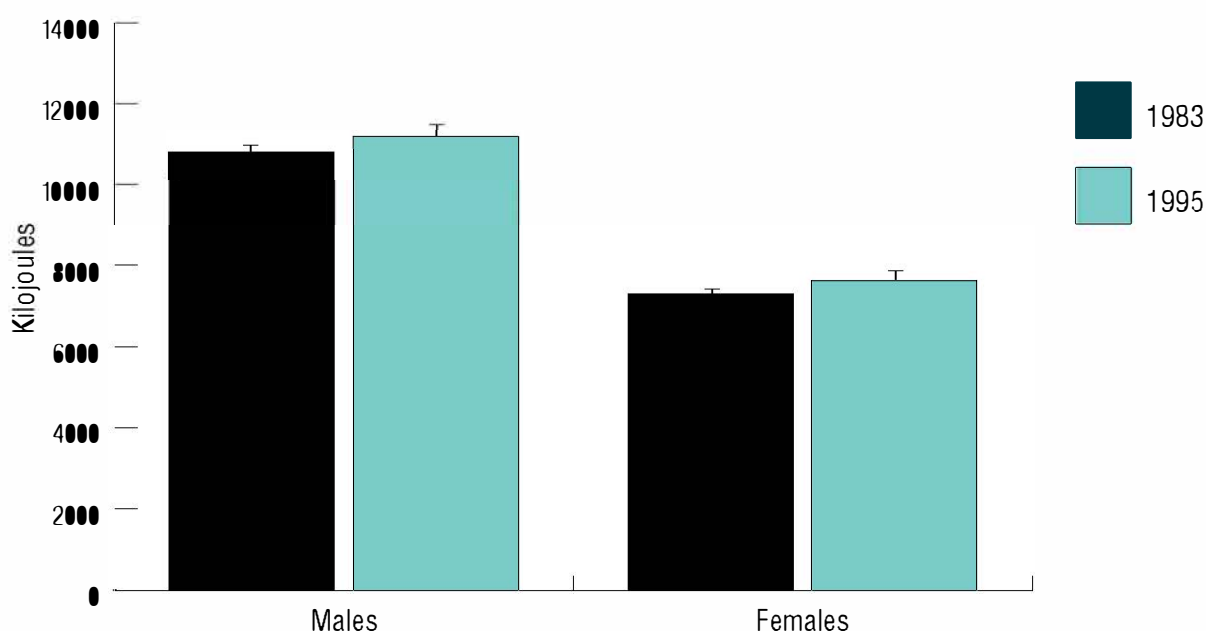
In the context of this social and physical environment, overweight and obese individuals need support, rather than criticism and discrimination. Education has an important role in establishing health behaviours and the readiness of individuals to effect behavioural change. Healthy weight initiatives must achieve a balance between individual and societal responsibility and be culturally appropriate, widely available and accessible, particularly to disadvantaged and vulnerable groups.^{39,151}

Energy intake and trend data

In adults, energy intake increased by 3–4% in the decade to 1995, equivalent to an additional 350 kilojoules per day (see Figure 1.1).¹⁵² Greater increases were seen in children and adolescents, with energy intake increasing by 11% for 10–15 year-old girls (equivalent to an additional 900 kilojoules per day) and by 15% for 10–15 year-old boys (equivalent to an additional 1,400 kilojoules per day) (see Figure 1.2).¹⁵² Without compensatory increases in physical activity, these changes are enough to have resulted in the significant observed increase in mean body weight.¹⁵²

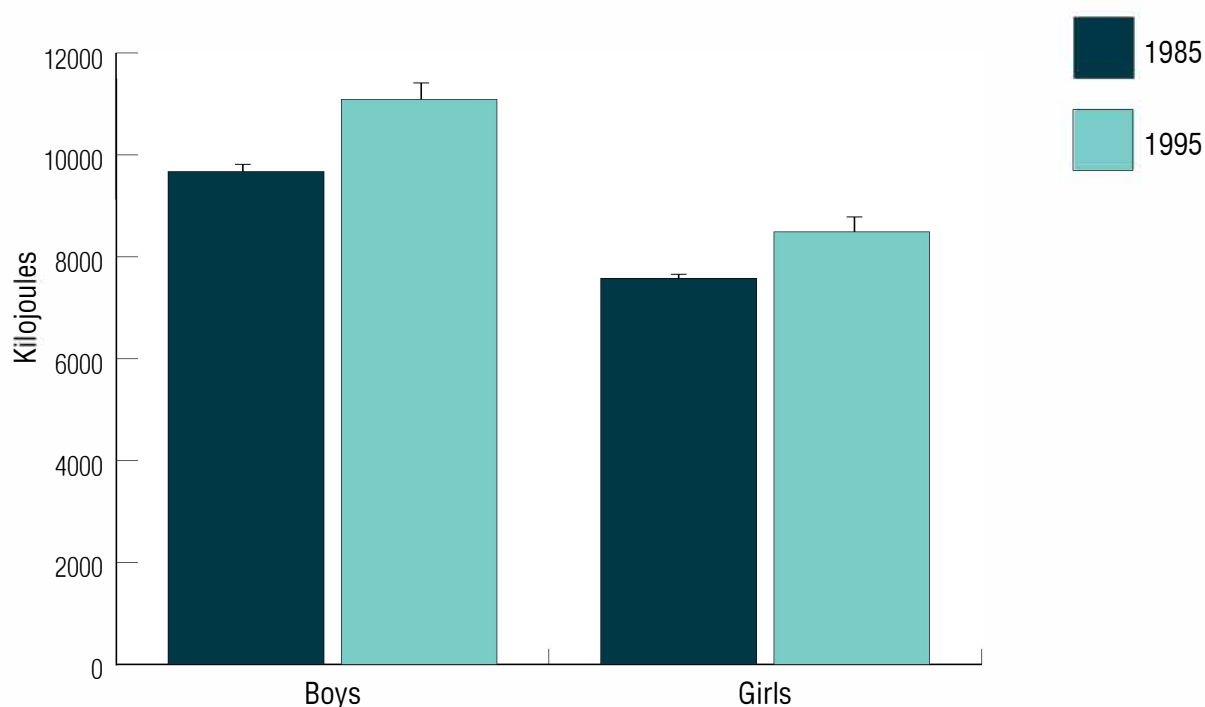
It should be noted that results of the 2007 Australian National Children's Nutrition and Physical Activity Survey cannot be compared with previous national surveys due to differences in methodology. As well, in some groups (such as older girls) there is likely to have been significant under-reporting of energy intake.¹² Up-to-date data will be provided by the 2011–13 Australian Health Survey, which is collecting information about the dietary intake of Australians aged 2 years and over.⁸³

Figure 1.1: Mean energy intakes of adults: 1983 and 1995



Source: Adapted from Cook et al (2001).¹⁵²

Figure 1.2: Mean energy intakes of children aged 10–15 years: 1985 and 1995



Source: Adapted from Cook et al (2001).¹⁵²

Energy from macronutrients

Macronutrients (proteins, fats and carbohydrates) all contribute to dietary energy intake.⁸ There is a growing body of evidence that the relative proportions of macronutrients consumed affect the risk of chronic disease and may also affect micronutrient intake.⁸ Optimal proportions of the type of fat (e.g. saturated, polyunsaturated or monounsaturated, or specific fatty acids within these categories) and carbohydrate (e.g. complex [starches] or simple [sugars]) may also be important in reducing chronic disease risk.⁸

The estimated Acceptable Macronutrient Distribution Ranges (AMDR) related to reduced risk of chronic disease are:⁸

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

Outside these ranges, the risk of chronic disease, overweight and obesity, and inadequate micronutrient intake may increase, but there are insufficient data available at extremes of population intake.⁸

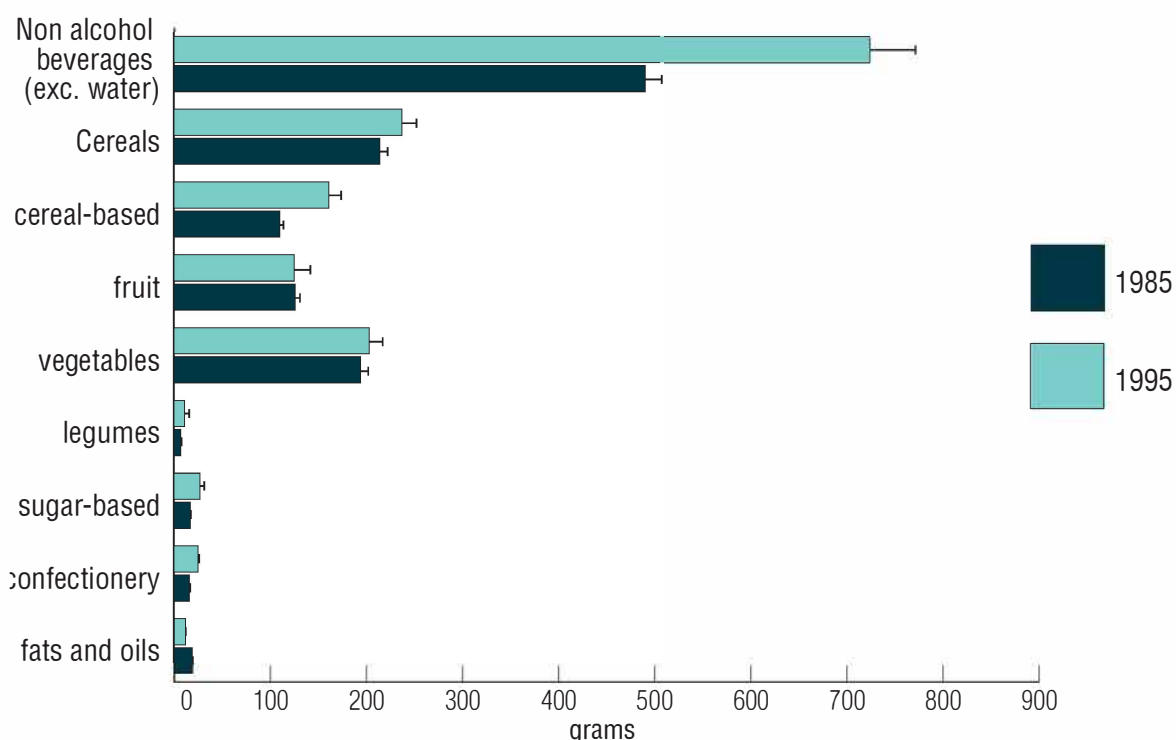
Alcohol also contributes to dietary energy. It is recommended that alcohol intake contribute less than 5% of dietary energy because of the negative association between intake of alcohol and health outcomes (see Section 3.4).

Energy intake from specific food groups

Increased energy consumption in the decade to 1995 was largely driven by rising consumption of cereal-based foods (including cakes, biscuits, pies, pizza and some desserts), confectionery and sugar-sweetened drinks.¹⁵² In 1995, consumption of energy-dense and nutrient-poor foods contributed almost 36% of adults' total energy intake and 41% of their total fat intake. For children, such foods contributed 41% of total energy intake and 47% of total fat intake.^{47,153}

As an example, changes in food group consumption for boys are illustrated in Figure 1.3. Among these was a decrease in intake of fats from oils and spreads, but an increase in intake of fats from other sources, such as cereal-based foods and confectionery,¹⁵² most of which were classified as 'extra foods' in the previous edition of the *Australian Guide to Healthy Eating*.

Figure 1.3: Mean consumption of selected food groups by boys aged 10–15 years: 1985 and 1995



Source: Adapted from Cook et al (2001).¹⁵²

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.⁸ Resting energy expenditure makes up 60–80% of total energy expenditure, and is mainly related to lean body mass.⁸ Active energy expenditure, which accounts for up to 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 that is under conscious control through physical activity.^{8,154}

Resting energy expenditure, active energy expenditure and total energy expenditure are all substantially increased in obesity,¹⁵⁵ contradicting the view that obesity is due to 'low metabolism' and is maintained despite a low level of food intake.¹³⁵ However, energy expenditure per kilogram of body mass does decline with increasing BMI, even at the same levels of physical activity.¹⁵⁴

1.1.4 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 (e.g. gardening or walking/cycling for leisure or transport).¹⁵⁶

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

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 1999.¹⁵⁷ While the different methodologies do not allow comparison, the 2007–08 National Health Survey showed that only 37% of adults exercised sufficiently to obtain benefits to their health.²⁴ State-based surveys suggest that since around 2004, there have been small increases in physical activity participation at levels that provide health benefits.^{106,158,159}

New data to enable monitoring and reporting of adults' physical activity against national physical activity guidelines and recommendations will be collected in the 2011–13 Australian Health Survey, with detailed results available from May 2013.

The 2007 National Children's Nutrition and Physical Activity Survey found that 69% of Australian children were likely to meet the physical activity guidelines on any given day.¹² 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.¹² Available state data were generally consistent with these findings.¹⁶⁰⁻¹⁶²

In 2007, Australian children aged 9–16 years spent more than 3.5 hours/day on average in sedentary behaviours such as watching television, playing video or computer games and/or using computers more generally.¹² On any given day, 67% of children spent more than the recommended maximum of 120 minutes of recreational screen time.

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 when the community adopts more regular moderate physical activity.¹⁰ Being physically active:

- reduces the risk of all-cause mortality^{163, 164}
- is an important factor in preventing and managing a range of chronic diseases, including heart disease, stroke, hypertension, type 2 diabetes and some cancers^{165, 166}
- is associated with reduced risk of injury^{165, 166}
- offers other health benefits, including building and maintaining healthy bones, muscles and joints^{165, 166}
- improves self-esteem, self-image and quality of life.¹⁶⁵⁻¹⁶⁸

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 being more active.^{163, 164} Benefits have been described for all age groups and physically active children are more likely to remain physically active throughout adolescence and into adulthood.^{169, 170}

1.2 The evidence for 'achieve and maintain a healthy weight'

There is convincing and increasing evidence in a range of areas to justify the recommendation to achieve and maintain a healthy weight, including:

- the effectiveness of combined interventions in primary and secondary prevention of overweight and obesity
- the association between particular foods and risk of excess weight gain
- the protective effect of other foods in reducing risk of excess weight gain
- the role of a range of socioenvironmental factors in development of overweight and obesity.

Table 1.1: Evidence statements on 'achieve and maintain a healthy weight'

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
Excess weight gain relative to height during childhood is associated with an increased risk of overweight later in life.	A
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 three to five serves per day of cereal foods (mainly wholegrain) is associated with a reduced risk of weight gain.	B

Evidence statement	Grade
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 are associated with reduced risk of overweight and obesity in children.	C
Combined diet and physical activity interventions are 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 socioeconomic 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

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

1.2.1 Primary and secondary prevention

Favourable outcomes have been consistently observed in interventions focusing on both reduced energy intake and increased physical activity, supporting the evidence statements that combined interventions assist weight management in children, adolescents and adults.

- **Adults:** There is evidence that diet and physical activity interventions in adults can prevent overweight and obesity (Grade C; Evidence Report, Section 22.2).¹⁷¹ Lifestyle interventions combining diet and physical activity interventions are probably associated with reduced risk of developing type 2 diabetes in adults (Grade B; Evidence Report, Section 22.3).¹⁷¹ For further information, see the NHMRC *Management of overweight and obesity in adults, adolescents and children* (NHMRC Overweight and Obesity Guidelines).¹²¹
- **Children and adolescents:** Recent reviews of combined diet and physical activity interventions for children and adolescents suggest that these can prevent overweight and obesity (Grade C; Evidence Report, Section 22.1).^{172–175} 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; Evidence Report, Section 17.12).^{176–179} Recent evidence suggests that interventions delivered in the school environment that focus on healthy eating and physical activity improve weight outcomes in children (Grade C; Evidence Report, Section 17.1).^{180,181} There is a limited amount of quality data on specific components of the combined programs in treating overweight and obesity; however, the results of combined programs indicate significant and clinically meaningful reductions in the level of overweight in obese children and adolescents.^{182,183}

1.2.2 Dietary patterns and specific foods and drinks

There is increasing evidence that consumption of specific foods and food groups is associated with risk of excess weight gain, while other food types are associated with a reduced risk of weight gain.

- **Fat:** The 2003 edition of the dietary guidelines and many international public health organisations, including the World Health Organization (WHO),⁹⁸ emphasised the major role of fat consumption in the development of overweight and obesity and of reducing fat intake as part of management. More recently, WHO has shifted its emphasis, stating that there is convincing evidence that energy balance is critical to maintaining healthy weight and ensuring optimal nutrient intakes, regardless of macronutrient distribution.¹⁸⁴ Although no specific macronutrient may be responsible for the development of obesity, the proportion of macronutrients in the diet does influence energy and nutrient intake, which may impinge on weight management and health outcomes.

- **Sugar:** No large long-term studies have measured the long-term development of overweight or obesity specifically related to consumption of added sugars (Evidence Report, Section 14.3). However, recent evidence indicates that 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; Evidence Report, Section 15.1),¹⁸⁵⁻¹⁹⁶ a finding confirmed by a later longitudinal study.¹⁹⁷
- **Sugar-sweetened drinks:** 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.¹⁹⁸ Most of the relevant research (see Section 3.3.2) was conducted in the US where, unlike Australia, corn syrup containing fructose 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 grading the relevant evidence statement.
- **Glycaemic index:** The US review found strong and consistent evidence that glycaemic index and/or glycaemic load are not associated with body weight and that modifying either of these does not lead to greater weight loss or better weight management.¹⁹⁸ There is 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.¹⁹⁹ These factors were not included in the literature review to inform the revision of these Guidelines.
- **Vegetables and fruit:** There is evidence suggesting that consumption of vegetables is associated with a reduced risk of weight gain (Grade C; Evidence Report, Section 2.2).²⁰⁰⁻²⁰³ The evidence also suggests that consuming fruit is associated with a reduced risk of obesity and weight gain (Grade C; Evidence Report, Section 1.3).²⁰⁰⁻²⁰⁸ The US review 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.¹⁹⁸
- **Portion size:** The US review found strong evidence of a positive relationship between portion size and body weight.¹⁹⁸
- **Dairy foods:** Recent evidence suggests that consumption of dairy foods is not associated with weight change or risk of obesity (Grade C; Evidence Report, Section 5.8)²⁰⁹⁻²¹⁴ and that consuming milk is not associated with BMI or BMI change in childhood (Grade C; Evidence Report, Section 5.9).^{209,215-219} These findings are consistent with those of the US review, which found strong evidence that intake of milk and milk products do not have a distinct role in weight control.¹⁹⁸
- **Grains:** There is evidence of a probable association between consumption of three to five serves per day of grain (cereal) foods (mainly wholegrain) and a reduced risk of weight gain (Grade B; Evidence Report, Section 6.5).^{215,220-229}
- **Nuts:** There is evidence to suggest that consuming nuts (65–110g per day) is not related to risk of weight gain in the short term (Grade C; Evidence Report, Section 8.1).²³⁰⁻²³⁵
- **Fruit juice:** The US review found that, for most children, there was limited evidence that intake of 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 fruit juice was found to be associated with increased adiposity in children who were already overweight or obese.¹⁹⁸

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.²³⁶ The US review found strong and consistent evidence that dietary patterns that are relatively low in energy density improve weight loss and weight maintenance in young adults.¹⁹⁸

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; Evidence Report, Section 17.2).²³⁷
- **Birth weight:** Increased birth weight, especially above 4,000g, is associated with increased risk of overweight or obesity in childhood, adolescence and later life (Grade A; Evidence Report, Section 17.1).^{54,58,61,62,79,238,239} There is a J- or U-shaped relationship between birth weight and increased risk of child or adult obesity, with both low birth weight and high birth weight babies at increased risk.²⁴⁰

- **Childhood weight gain:** There is convincing evidence that excess weight gain relative to height during childhood is associated with increased risk of being overweight later in life (Grade A; Evidence Report, Section 17.4).^{53,241,242}
- **Parental weight:** 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 parents (rather than one) are overweight or obese (Grade A; Evidence Report, Section 17.7).^{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; Evidence Report, Section 17.5).^{52,54,59,61,243}
- **Television:** Recent evidence suggests that hours spent watching television by children is associated with increased risk of overweight or obesity (Grade C; Evidence Report, Section 17.3).^{49,60,64-71,244-246} Media use, including television viewing, may displace time children spend in physical activities^{246,247} and eating meals and snacks in front of the television may also be associated with increased energy intake.²⁴⁸
- **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; Evidence Report, Section 17.9).^{49,52,55,64,65,68-74} Similarly, the evidence suggests that low socioeconomic status is associated with an increased risk of overweight or obesity (Grade C; Evidence Report, Section 17.10).^{59,62,238,249,252}
- **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 adolescence⁷³
 - consuming takeaway food and low quality snacks⁷⁵⁻⁷⁷
 - childhood smoking^{73,83}
 - increased price of fruit and vegetables^{82,79}
 - low self-esteem and/or depression^{80,82}
 - low locus of control score^{84,85}
 - stressful family life^{86,87}
 - food insecurity^{65,81,253}
 - self-reported dieting,^{76,82,254} particularly in girls²⁵⁵
 - inadequate sleep^{60,244,256,257}
 - low rates of breakfast consumption.²⁵⁸

The literature review to inform the revision of the US dietary guidelines found strong and consistent evidence 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 to evaluate any association between eating at other types of restaurants and risk of weight gain, overweight and obesity.¹⁹³ The US 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.¹⁹³

The US review 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.¹⁹³

Finally, an emerging body of evidence documents the impact of the obesogenic food environment on body weight in children and adults. Current evidence indicates that the food environment is associated with dietary intake, especially lower consumption of vegetables and fruits, and resulting in higher body weight.¹⁹³ This is discussed further in Appendix A.

1.3 How dietary patterns can affect energy balance and weight outcomes

Environmental and lifestyle factors resulting in overconsumption of energy in the diet and a decrease in physical activity are major contributors to the obesity epidemic. A small, persistent energy imbalance is enough to cause excess weight gain in both children and adults, which over time progressively increases the BMI.^{135,139} 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 1980.¹³⁹

There has been much debate about the role of energy, carbohydrate and fat intake in the obesity epidemic.^{236,259,260} While reducing excess fat intake has been recognised as an important strategy to reduce energy intake in successful weight loss interventions,²⁵⁹ there is little evidence that population fat intake is associated with the obesity epidemic independently of total energy intake.²⁶⁰

Foods with a higher energy density encourage energy intake above requirements.^{198,261} Foods that are high in energy density also tend to be more palatable, and high palatability is associated with increased food intake in single-meal studies.^{142,262} 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 concentrations 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 that tend to be relatively low in total fat and moderate (not high) in carbohydrate are consistent with reduced risk of excess 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 sugar-sweetened drinks may contribute to excess energy intake through lack of impact on satiety.²¹⁶ The satiety value of foods may also be important in managing appetite and hunger.²⁶³

Energy-dense dietary patterns are associated with higher consumption of grain-based foods, fats and sweets and lower consumption of vegetables and fruit.²⁶⁴ International data suggest that the major foods contributing to increased energy intake include sweetened drinks, snack foods and fast food^{261,265,266} and that increasing portion size is also an important contributor.^{198,267,268} The low cost of energy-dense nutrient-poor food relative to nutrient-dense food also has a major role.^{261,264}

The three key lifestyle areas related to overweight and obesity are dietary pattern, physical activity and behavioural change. Multicomponent interventions that address all three areas are more effective than those that address only one or two. Positive outcomes have been described, at least in the short term, in clinical weight loss regimes that include a range of interventions.^{269,270} For further information, see the NHMRC Overweight and Obesity Guidelines.¹²¹

1.4 Practical considerations: achieve and maintain a healthy weight

Intentional weight loss in overweight and obese individuals reduces the risk factors for mortality and morbidity, and alleviates the symptoms of many chronic conditions.^{269,270} It is not necessary to lose large amounts of weight to achieve substantial health gains. For example, it is estimated that a weight loss of 5 kg in all people who are overweight or obese would reduce the national prevalence of type 2 diabetes by 15%.²⁷¹ Improving nutrition and/or increasing physical activity also benefits health in areas beyond weight control, such as bone strength, mental health and immune function.^{2,272}

There is a need to provide population guidance 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 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 to 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.

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 people who are overweight without serious comorbidities. Clinical guidance for managing overweight and obesity and its complications for individuals is included in NHMRC Overweight and Obesity Guidelines.¹²¹

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 the person has been classified as overweight or obese
- it is more efficient and cost effective to prevent weight gain rather than treat overweight and obesity.^{22,23,101}

The community as a whole has a social responsibility to address the current epidemic of obesity. It is especially important that supportive social, economic and physical environments are created, so that it is easier for individuals, families, groups and communities to choose healthy dietary and physical activity patterns and achieve and maintain a healthy weight.

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

1.4.1 Physical activity and dietary patterns in achieving and maintaining a healthy weight

Physical activity

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.²⁷³⁻²⁷⁵ However, in the current environment of abundant availability, promotion and consumption of energy-dense food, it is now internationally recommended 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.^{22,276-279} 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.²⁷⁶

The current physical activity guidelines for Australian infants, children and adolescents may be adequate to support optimum growth and development and weight control (together with consuming a nutritious diet and appropriate energy intake).^{35,121} This is supported by recent evidence from Europe.²⁸⁰

The current physical activity guidelines for Australians are summarised in Appendix H, and these are under review in 2012.

Dietary patterns

Foundation Diets developed by the Food Modelling System⁹ represent the basis of optimum diets to achieve and maintain a healthy weight as they provide nutrient requirements with minimum energy intake.

Compared to the most recent available data on 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 grain (cereal) foods, higher and medium fat milk, yoghurt and cheese products and lean red meats (latter for men only).⁹ 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 grain (cereal) foods to wholegrain (cereal) foods would also be necessary.⁹

To avoid excess 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.⁹

Taller and/or more active adults in each age and sex group can choose additional serves of foods from the five food groups and/or unsaturated spreads and oils and/or discretionary foods to increase energy intake to meet energy requirements (that is, to comprise Total Diets),⁹ but they need to monitor weight or waist circumference 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 (e.g. every 2–3 months) and the amount and/or quality of dietary intake and physical activity levels adjusted accordingly.⁹

As a basic principle when adjusting dietary patterns, the first steps are to choose nutritious foods from the five food groups and the unsaturated fat allowance 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.⁹

Recommended quantities of specific food groups to suit those preferring different dietary patterns are included in the Foundation Diets for adults set out in the Food Modelling System.⁹

1.4.2 Weight loss in adults who are 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, outlined throughout this document and in the companion resources should not be exceeded. Adhering to Foundation Diets only, without discretionary foods or additional serves of the five food groups, could represent a daily reduction in energy intake of 1,850 kilojoules for women and 2,700 kilojoules for men who are average height for their age and sex group. This could result in a satisfactory amount 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 lower 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.ⁱ²¹ 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 4.0 kg 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.^{j21,281}

Individuals who are overweight or obese and have associated cardiovascular disease or type 2 diabetes risk factors should seek clinical advice about the range of available treatment options.ⁱ²¹

1.4.3 Pregnant and breastfeeding women

Obesity in pregnancy is one of the most common, and potentially modifiable, risk factors for adverse pregnancy outcomes²⁸² and longer term adverse outcomes for mothers²⁸³ and children.²⁸⁴ Since about one-third of pregnant women in Australia are overweight or obese,²⁸⁵ preventing excess gestational weight gain is an urgent health priority.

Appropriate maternal weight gain during pregnancy (see Tables 1.2 and 1.3) is important for the health of the infant:

- too little weight gain during pregnancy increases the risk of a low birth weight infant
- excess weight gain during pregnancy increases the risk of macrosomia and gestational diabetes and is also associated with increased risk of obesity and metabolic syndrome in infants later in life.^{286,287}

Evidence supports recommendations to manage pregnancy to reduce the risk of excess weight gain and gestational diabetes.^{288,289}

Appropriate steady weight gain during pregnancy is important to optimise the health outcomes (short term and long term) for the infant and the mother.^{288,289} Steady weight gain helps to avoid adverse effects on specific foetal organ systems during critical periods (e.g. neural tube 6–8 weeks, kidney development 28–30 weeks).

Weight loss diets are not recommended at any time during pregnancy.²⁸⁹

Mothers who gain excess weight during pregnancy are unlikely to lose it later,²⁹⁰ although dietary patterns that comprise regular meals, plenty of fruit and vegetables, high-fibre bread and restricted high sugar snacks²⁹¹ may help women reach a healthy weight after giving birth.

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.²⁹² There is also increasing evidence of the importance of growth and optimum nutrition in relation to cognitive development²⁹³ and future bone mass.²⁹⁴

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; Evidence Report, Section 17.5).^{52,54,59-61,243}

For further information on pregnant and breastfeeding women, see the Infant Feeding Guidelines.²⁹⁵

Table 1.2: 2010 Institute of Medicine recommendations for total and rate of weight gain during pregnancy, by pre-pregnancy BMI

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)
Multiple pregnancy		
Twin pregnancy	15.9–20.4	0.7
Triplet pregnancy	22.7	—

Note: *Calculations assume a 0.5–2kg weight gain in the first trimester

Source: Institute of Medicine 2009.²⁸⁹

Table 1.3: Weight gain during pregnancy: recommendations for Asian women, by pre-pregnancy BMI

Pre-pregnancy BMI (kg/m ²)	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	—

Source: Adapted from Institute of Medicine²⁸⁹ and matched with Asian BMI cut-offs.

1.4.4 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 H). Maintaining 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 – for example, through dieting – is usually inappropriate.

Growth

Relative to their body weight, children's nutrient and energy requirements are greater than those of adults.⁸ Children are nutritionally vulnerable up to around 5 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, 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 20-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 1–3 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 healthy nutrition practices for life. Food intake may drop off during the second year of life, when parents' encouragement and example may 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.

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. 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 and/or unsaturated spreads and oils and/or discretionary food choices may be made to increase energy intake until energy requirements are met.⁹ Growth and weight of children should be checked regularly (as outlined in Appendix G) and the amount and/or quality of diet and physical activity adjusted accordingly.⁹

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 grain (cereal) foods, 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* and companion resources, 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 a range of dietary patterns are included in the Foundation Diets for infants, children and adolescents set out in the Food Modelling System.⁹

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

Management of overweight and obesity in childhood is recommended to reduce risk of overweight in later life. Individual assessment and clinical supervision is recommended to ensure appropriate growth and development while managing weight in overweight or obese children and adolescents. As parental overweight or obesity is associated with increased risk of overweight or obesity in children, a family-focused approach to weight management is likely to be beneficial.¹⁷⁶⁻¹⁷⁹ For further information on weight management, see NHMRC Overweight and Obesity Guidelines.¹²¹

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 suboptimal growth and development. Adherence to Foundation Diets should assist maintenance of body weight during growth for children who are overweight. The aim is to maintain weight while the child grows in height, thus 'normalising' BMI for age.

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

1.4.5 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.⁴⁵ 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.²⁹⁶ The decline in energy expenditure must be balanced by adjusting energy intake to maintain body weight within the healthy range and to prevent an increase in body fat.⁸ Dietary patterns and quantities consistent with the Foundation and Total Diets are for older people who are generally fit and well.⁹

Most older people will benefit from increased physical activity^{137,297} including reduced sedentary behaviour, increased moderate-intensity aerobic activity such as brisk walking or running²⁹⁸ and particularly activities promoting bone and muscle strength, flexibility and balance such as yoga or tai chi.²⁹⁹ In addition to assisting with weight management, these can help reduce the risk of falls^{299,300} and may increase opportunities for social engagement.³⁰¹

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.³⁰² 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 overweight and obesity are still prevalent in older adults,³⁰³ consideration of overall morbidity and mortality suggests that for obese older people a substantial reduction in BMI may not provide the healthiest long-term option.³⁰⁴ 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.³⁰³ Lowering blood pressure and normalising blood lipids rather than reducing weight may be more appropriate for older people who are overweight.³⁰²

While most of the older population live independently, it has been estimated that 25–40% of those over 80 years of age could be considered frail.³⁰⁵ Malnutrition in older people 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.^{305,306} In Australia, 5–11% of people eligible for Home and Community Care services are malnourished.³⁰⁷ In acute care, 20–30% of people are admitted with malnutrition, the prevalence increasing with age and the number of health problems.^{307,308} 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 excess body fat.³⁰³ As such the guidelines in this document are not appropriate for frail elderly people or those with complex health conditions and an appropriate health professional should be consulted.^{34,302}

The decrease in energy expenditure with ageing is generally accompanied by decreased appetite and diminished food intake, so may account for the undernutrition seen in some older people.³⁰⁹ Recent studies suggest that, for an older person, being underweight may be more deleterious for health than being overweight.¹²⁸

1.4.6 Aboriginal and Torres Strait Islander peoples

The last national survey measuring height and weight in Aboriginal and Torres Strait Islander peoples was conducted in 1994.³¹⁰ 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 peoples than from self-reported data from non-Indigenous Australians.^{311–313}

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.^{99,140,264,314–317} The 2011–13 Australian Health Survey will provide data on objective and self-reported height and weight measures in Aboriginal and Torres Strait Islander peoples. For more information see Appendix A.

1.4.7 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 origin¹¹⁸ 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, so applying the current WHO BMI cut-off points may underestimate body fatness and comorbidity risk in this population.¹¹⁸

1.4.8 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.ⁱ¹²⁹ 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.ⁱ¹²⁹ Estimated lifetime prevalence of anorexia nervosa, bulimia nervosa, and binge eating among women is in the range of 0.3–1.5%, 0.9–2.1% and 2.5–4.5% respectively, with estimated rates among men considerably lower.ⁱ¹²⁹ 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:ⁱ¹²⁹

- 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
- promotion of good mental health.

1.5 Practice guide for Guideline 1

Table 1.4: Summary of practical considerations for Guideline 1

Population group	Considerations
Adults—to achieve and maintain a healthy weight	<ul style="list-style-type: none"> Nutritious foods should be chosen from the five food groups and the unsaturated fat allowance, in amounts consistent with Foundation Diets Discretionary (energy-dense, nutrient-poor) choices should be limited Taller and/or more active adults in each age and sex group can choose additional serves of foods from the five food groups and/or unsaturated spreads and oils and/or discretionary foods to increase energy intake to meet energy requirements Weight and waist circumference should be measured regularly (e.g. every 2 or 3 months) The recommendations of the Australian physical activity guidelines should be followed
Adults—to promote weight loss	<ul style="list-style-type: none"> The Foundation Diets should be adhered to, without discretionary foods or any additional serves of the five food groups Weight loss will not be achieved unless energy intake is lower than total energy expenditure Behavioural change to increase physical activity and improve dietary intake is fundamental to weight management
Infants, children and adolescents—to achieve and maintain a healthy weight	<ul style="list-style-type: none"> Foundation Diets represent the basis of optimum diets to support optimum growth and development Growth and weight should be checked regularly and the amount and/or quality of diet and physical activity adjusted Childhood is a period of education about good nutrition—appropriate use of food helps to establish healthy nutrition practices for life
Infants, children and adolescents—to manage overweight	<ul style="list-style-type: none"> Restricting diet — beyond adherence to the Foundation Diets and limiting intake of discretionary foods and drinks—is not recommended The recommendations of the Australian physical activity guidelines should be followed
Pregnant and breastfeeding women	<ul style="list-style-type: none"> Appropriate steady weight gain during pregnancy is important to optimise short- and long-term health outcomes for both infant and mother Weight loss diets are not recommended at any time during pregnancy
Older people	<ul style="list-style-type: none"> Declining energy expenditure with age must be balanced by adjusting energy intake to maintain body weight within the healthy range Physical activity is needed to help maintain muscle strength Lowering blood pressure and normalising blood lipids may be more appropriate for overweight older people than reducing weight The upper range of BMI for healthy adults may be more appropriate for older persons The <i>Australian Dietary Guidelines</i> are not appropriate for frail elderly people or those with complex health conditions—an appropriate health professional should be consulted
Australians of Asian origin	<ul style="list-style-type: none"> Applying the current WHO BMI cut-off points may underestimate body fatness and comorbidity risk

Where to next?

The next chapter provides information on the importance of consuming a wide variety of nutritious foods to promote healthy weight, to promote health and wellbeing and to prevent chronic disease.

GUIDELINE 2

Enjoy a wide variety of nutritious foods

Guideline 2

Enjoy 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 and/or high cereal fibre varieties, such as bread, cereals, rice, pasta, noodles, polenta, couscous, oats, quinoa and barley
- Lean meats and poultry, fish, eggs, tofu, 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 plenty of water.

Summary

- Dietary patterns that 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 and/or high cereal fibre varieties; lean meats 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 2 years.
- There are many different ways that these nutrient-dense foods can be chosen to contribute to nutritious dietary patterns that suit personal preferences. However economic, social and cultural factors can affect the ability of individuals and groups to access nutritious foods.
- Although there have been improvements in diet-related health over the past decades, current consumption patterns still contribute to obesity and an increased risk of chronic disease including cardiovascular disease, type 2 diabetes and several cancers.
- Together with adherence to Guideline 3 (on limiting intake of specific foods high in saturated fat, added sugars and/or salt) and Guideline 1 (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 Enjoy a wide variety of nutritious foods

2.1.1 Setting the scene

No single food – with the exception of breast milk for about the first 6 months of life – can provide all the nutrients in the amounts needed for good health. Dietary patterns that include a wide variety of nutritious foods and water are more likely than restricted diets to meet nutrient requirements⁸ 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 meats and poultry, fish, eggs, nuts and seeds 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.⁹ Australia is also fortunate in having a safe food supply with low levels of contaminants and pollutants.³¹⁸

The most recent dietary survey data available for Australian adults – the National Nutrition Survey 1995 – showed an increase in the number of foods being consumed by adults in that year compared with 1983.⁴⁵ New data on food consumption patterns are being collected in the 2011–13 Australian Healthy Survey, with detailed results available from May 2013. 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.^{319,320} 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.³²¹ 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.³²²

Despite the variety of foods available in Australia, comparison of actual intakes with NRVs⁸ 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 age⁹ and iodine intake is inadequate in some pregnant and breastfeeding women.³²³

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 1 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,^{8,9,33} and promote health and wellbeing.

Food choices to reduce the impact on the environment are consistent with those to improve health. Each food group provides a variety of foods from which people can choose based on dietary preferences or plant-based or belief-based dietary patterns.

2.1.2 The evidence for consuming ‘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; Evidence Report, Section 20.3).^{20,324-327} Reviews of studies of a range of eating patterns suggest that:

- higher quality diet is associated with reduced morbidity
- the negative 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; Evidence Report, Section 20.3).^{19,20}

Recent evidence also confirms that a dietary pattern consistent with current guidelines to consume a wide variety of nutritious foods may be associated with superior nutritional status, quality of life and survival in older adults.^{21,22}

Table 2.1: Evidence statements for consuming 'a wide variety of nutritious foods'

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

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

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

Nutritional science has traditionally characterised foods according to their macro- and micronutrient 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.^{328,329} 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.⁹

Dietary variety has the benefit of diluting potential toxicants found naturally in foods.³³⁰ 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 5).

Another potential benefit of food variety comes from maximising the bioavailability of nutrients.³³¹ 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 excess intake of nutrients are nearly always associated with intakes of supplements.⁸ 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.^{8,332} Examples include excessive consumption of carrot juice or regularly eating very large quantities of liver, which may cause vitamin A toxicity.³³³

2.1.4 Practical considerations: enjoy a wide variety of nutritious foods

It is important to consider this Guideline together with other Guidelines, particularly Guideline 3 on limiting intakes of specific foods high in saturated fats, added sugars and/or added salt and Guideline 1 on healthy weight. The word 'plenty' is used judiciously to encourage increased consumption of vegetables (except those that are fried). 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 *overconsumption*, as there is some evidence that there may be a link between eating a variety of energy-dense food and drinks and excess food intakes (see Chapter 1). *Variety* 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*⁹ and companion resources. Discussion of dietary patterns and the environment is included in Appendix G.

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,³³⁴⁻³³⁶ or being large for their gestational age,³³⁷ and may also help reduce the risk of pregnant women developing pre-eclampsia.^{338,339} Quality nutritious dietary patterns before and during pregnancy may help reduce the risk of women developing gestational diabetes mellitus.³³⁹⁻³⁴²

Maternal diet during pregnancy and while breastfeeding does not appear to affect the risk of asthma, eczema or other allergy symptoms in infants.³⁴³⁻³⁴⁷ 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.³⁴⁸

Foods that should be avoided during pregnancy include those associated with increased risk of *Listeria* bacteria, such as soft cheeses, cold seafood, sandwich meats, pâté, bean sprouts and packaged or pre-prepared salads (see Chapter 5 for further information on food safety).³⁴⁹

Consumption of fish can be valuable in pregnancy however care may be required with intakes of certain species of fish due to the potential risk of excessive mercury intake (see Section 2.4.4).

Constipation is a common symptom during pregnancy.³⁵⁰ Clinical treatment of constipation generally includes advice to consume a high fibre diet, including wholegrain cereals, fruit, vegetables and legumes, and to avoid dehydration by drinking sufficient water, which is consistent with these Guidelines.

Infants

Exclusive breastfeeding is recommended for around the first 6 months of age after which solid foods of suitable texture can be introduced. At around the age of 6 months, infants are physiologically and developmentally ready for new foods, textures and modes of feeding and need more nutrients than can be provided by breast milk or formula alone. There is no particular order or rate for the introduction of new foods, other than the first foods should be rich in iron. Food choices should be varied to ensure adequate energy (kilojoule) and nutrient supply. Breastfeeding should continue until 12 months and beyond for as long as the mother and child desire. For more information on breastfeeding and the transition to solids, see the Infant Feeding Guidelines.³⁵¹

When introducing solid 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.

Nuts are a problem for small children as their size and consistency increases the risk of inhalation and choking. For this reason, they should not be given to children aged less than 3 years. However nut pastes and nut spreads can be offered to infants from around 6 months of age. For further information on appropriate foods for infants, see the Infant Feeding Guidelines.³⁵¹

Children and adolescents

Children and adolescents should be encouraged to consume enough of a wide variety of nutritious foods to support normal growth and development.¹²⁹

Eating disorders and disordered eating are believed to affect a significant number of the Australian population, although the exact number is unknown due to the absence of accurate data. Adolescents, mainly girls, may be vulnerable to disordered eating and may choose a restricted diet.

Adults

Males living alone are at particular risk of not eating a wide variety of nutritious foods. Analysis of the 1995 National Nutrition Survey³⁶ showed that, in nearly all age groups, adult men consumed significantly fewer types of foods than other groups.

Older people

The diet of older people is generally more varied than that of younger groups.⁴⁵ However some older people 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.

Aboriginal and Torres Strait Islander peoples

Limited data on dietary intake among Aboriginal and Torres Strait Islander peoples 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,³⁵² with relatively little change observed recently.³⁵³ 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 many people in lower socioeconomic groups. For further information please see Appendix A.

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.^{354,355} 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 family's disposable income. Health professionals should be aware of the budget challenges healthy food habits may pose for people who are welfare dependent and should note that checking and comparing the price of food products can reduce the weekly food cost by about 13%.³¹⁶ Further details regarding the association of equity issues and consumption of varied and nutritious diets are included in Appendix A.

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.^{28,317,356,357}

People with vegetarian or vegan dietary patterns

About 4% of all respondents in the 1995 National Nutrition Survey described themselves as vegetarian or vegan.⁴⁵ 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.⁴⁵ Many more people eat vegetarian meals regularly or occasionally.

Appropriately planned vegetarian diets, including total vegetarian or vegan diets, are healthy and nutritionally adequate. Well-planned vegetarian diets are appropriate for individuals during all stages of the lifecycle.³⁵⁸ Those following a strict vegetarian or vegan diet can meet nutrient requirements as long as energy needs are met and an appropriate variety of plant foods are eaten throughout the day. Those following a vegan diet should choose foods to ensure adequate intake of iron and zinc and to optimise the absorption and bioavailability of iron, zinc and calcium.³⁵⁹ Supplementation of vitamin B₁₂ may be required for people with strict vegan dietary patterns.⁸

2.2 Enjoy plenty of vegetables, including different types and colours, and legumes/beans, and enjoy 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. 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.⁹ Some vegetables are suitable to eat raw, while it is best to cook others to make them more palatable and digestible. Dried legumes should be cooked. Some processed fruits and vegetables, such as those that are canned or frozen in natural juices, are nutritious alternatives as long as they are produced without added salt, sugar (including concentrated fruit juice) or fat (in particular saturated 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 or vegan meals.⁹

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.^{35,36,198,360} However fruit and vegetable access, affordability and availability may be difficult for some groups. Further discussion can be found in Appendix A.

2.2.2 The evidence for consuming 'plenty of vegetables'

The scientific evidence for 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.³⁶¹ 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 some 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 J).

Although serve sizes of vegetables differed between studies considered in the Evidence Report,³³ the evidence statements presented in Table 2.2 are based on the Australian standard serve size of 75g.

Table 2.2: Evidence statements for consuming 'plenty of vegetables'

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 one to two serves per day of tomato is associated with a reduced risk of prostate cancer.	C
Consumption of more than one serve 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

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

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; Evidence Report, Section 2.1).³⁶²⁻³⁶⁷ It is probable that consumption of vegetables is associated with a reduced risk of stroke (Grade B; Evidence Report, Section 2.4).^{368,369} 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; Evidence Report, Section 2.3).³⁷⁰⁻³⁷² This supports the inconsistent findings described in other studies for fruit and vegetables considered together and for fruit alone.^{371,372} However, as there is a strong relationship between type 2 diabetes and body weight (see Chapter 1), the association between consumption of vegetables and reduced risk of excess 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; Evidence Report, Section 2.2).^{200-203,207}

Cancer

- **Oral, nasopharyngeal and oesophageal:** Evidence suggests that consuming vegetables is associated with a reduced risk of oral and nasopharyngeal cancers (Grade C; Evidence Report, Section 2.10).³⁷³⁻³⁷⁷ This is consistent with the findings described by the WCRF report (see Appendix F).⁴³ 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; Evidence Report, Section 2.10).³⁷³⁻³⁷⁷
- **Other alimentary cancers:** 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^{35,36} still tend to be present when a longer time frame and different types of vegetables are considered (Evidence Report, Section 2). Although recent evidence suggests that total consumption of vegetables is not associated with reduced risk of oesophageal cancer (Grade C; Evidence Report, Section 2.9).³⁷⁸⁻³⁸¹ evidence from the WCRF report suggests that consumption of non-starchy vegetables probably reduces risk of cancer of the oesophagus (see Appendix F).⁴³

- **Prostate cancer:** The evidence suggests that consumption of one to two serves of tomatoes a day is associated with reduced risk of prostate cancer (Grade C; Evidence Report, Section 2.13).^{382,383} This is consistent with the probable relationship between intake of lycopene-containing foods and probable reduced risk of prostate cancer described by the WCRF report (see Appendix F).⁴³
- **Endometrial, ovarian and pancreatic cancer:** The 2003 edition of the dietary guidelines reported a possible reduction in risk of endometrial and pancreatic cancer with vegetable consumption.³⁶ However, in more recent studies there is no evidence to suggest an association between total vegetable consumption and ovarian (Grade C; Evidence Report, Section 2.11)^{384,385} or endometrial cancer (Grade C; Evidence Report, Section 2.12).³⁸⁶⁻³⁸⁸ However, findings from the WCRF report suggest decreased risk of both ovarian and endometrial cancer with consumption of non-starchy vegetables specifically (see Appendix F).⁴³
- **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; Evidence Report, Section 2.15).³⁸⁹ More specific studies suggest that consumption of more than one serve per week of spinach is associated with reduced risk of colorectal cancer (Grade C; Evidence Report, Section 2.15)³⁸⁹ and evidence of a suggestive protective effect of intake of non-starchy vegetables on colorectal cancer has been described in the WCRF report (see Appendix F).⁴³
- **Lung cancer:** Recent evidence³⁹⁰ suggests consuming cruciferous vegetables is associated with reduced risk of lung cancer (Grade C; Evidence Report, Section 2.14). The WCRF report found evidence of a probable association with the reduced risk of lung cancer and consumption of vegetables containing carotenoids and also found evidence suggesting that non-starchy vegetables were protective of lung cancer (see Appendix F).⁴³ 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 other types of vegetable consumption and gastric, breast, lung and colorectal cancers (Evidence Report, Sections 2.5, 2.6, 2.7 and 2.8).

Other conditions

The 2003 edition of the dietary guidelines included evidence of associations between the consumption of vegetables and some aspects of eye health, including cataracts and macular degeneration.³⁶ Further evidence was not available from more recent studies.³⁹¹

2.2.3 The evidence for consuming ‘plenty of legumes/beans’

While evidence of the health benefits of consumption of legumes/beans appears to have strengthened since the 2003 edition of the 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*.¹

Table 2.3: Evidence statements for consuming ‘plenty of legumes/beans’

Evidence statement	Grade
Consumption of soy foods is associated with reduced total cholesterol and low-density lipoprotein (LDL) cholesterol.	C
Consumption of legume foods is associated with reduced risk of colorectal cancer.	C

Notes: Grades — A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

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

¹ The evidence presented here was reviewed with a focus on whole foods, and for this reason studies of soy isolates are not reported.

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.

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; Evidence Report, Section 7.4).³⁹²
- **Type 2 diabetes:** No recent studies of the relationship between legumes/beans and type 2 diabetes were identified.
- **Excess weight:** No recent studies of the relationship between legumes/beans and weight loss were identified.³⁹³⁻³⁹⁵

Cancer

- **Colorectal cancer:** Evidence suggests that consuming legumes is associated with reduced risk of colorectal cancer (Grade C; Evidence Report, Section 7.3).³⁹⁶⁻⁴⁰⁰ However, in one study the effect was only significant for women,³⁹⁹ as also seen in the recent analysis of the European Prospective Investigation into Cancer and Nutrition (EPIC) database.⁴⁰¹ However no evidence of an association between consumption of legumes and colorectal cancer was described in the WCRF report (see Appendix F).⁴³
- **Other cancers:** Recent evidence is limited and/or inconclusive for an association regarding legume/bean consumption and breast or prostate cancer (Evidence Report, Sections 7.1 and 7.2). Similar limited associations have been described in the WCRF report (see Appendix F).⁴³ 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 (see Appendix F).⁴³

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 infarction, and mortality due to cardiovascular disease and hypertension.

2.2.4 The evidence for consuming ‘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 J). 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.

Table 2.4: Evidence statements for consuming ‘fruit’

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 one and a half serves of fruit a day, ideally two and a half 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

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

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. Although serve sizes differed between studies, the evidence statements presented below are based on standard serve sizes of 150g.

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; Evidence Report, Section 1.1).³⁶²⁻³⁶⁴ 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; Evidence Report, Section 1.2).^{368,369}
- **Type 2 diabetes:** The recent evidence suggests that consumption of fruit is not associated with risk of type 2 diabetes (Grade C; Evidence Report, Section 1.4). However, as there is a strong relationship between type 2 diabetes and body weight (see Chapter 1), the association between consumption of fruit and reduced risk of excess 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; Evidence Report, Section 1.3).²⁰⁰⁻²⁰⁸

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; Evidence Report, Section 1.10).^{373,374,376,377,402} consistent with findings of a convincing effect on reduced risk of cancers of the mouth, pharynx and larynx and a limited effect on nasopharyngeal cancers described in the WCRF report (see Appendix F).⁴³
- **Breast cancer, ovarian cancer and endometrial cancer:** Expanding on previous reports,³⁶ recent evidence now suggests that consumption of fruit is not associated with risk of breast cancer (Grade C; Evidence Report, Section 1.6).^{378,403-407} ovarian cancer (Grade C; Evidence Report, Section 1.11)^{384,385} or endometrial cancer (Grade C; Evidence Report, Section 1.12).^{386-388,408}
- **Colorectal cancer:** Recent evidence suggests that consumption of fruit is not associated with risk of colorectal cancer (Grade C; Evidence Report, Section 1.8).^{378,389,402,407,409-411} Further, there is limited evidence to suggest an association between the consumption of most fruits by specific type and colorectal cancer (Evidence Report, Section 1.14),^{389,411} which expands on earlier studies by the WCRF (see Appendix F).⁴³
- **Other cancers:** Recent evidence is limited and/or inconclusive for an association regarding fruit consumption and gastric, lung, oesophageal and pancreatic cancers (Evidence Report, Sections 1.5, 1.7, 1.9 and 1.13).

2.2.5 How consuming plenty of vegetables, including different types and colours, and legumes/beans, and 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.

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 atherogenic plaques that underlie many cardiovascular conditions.^{392,412-414} 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.^{8,415} Plant foods including vegetables and fruit provide potassium and magnesium, both of which have been linked to lower blood pressure.⁸ 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.⁴¹⁶

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.^{1,2} 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.³ 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).⁴ However, starchy vegetables are less nutrient-dense and are higher in kilojoules, but provide a rich source of complex carbohydrates.⁵

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.⁶

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.^{417,418} These reviews suggest the isoflavone in soy foods may have a role in cholesterol reduction, improved vascular health, preservation of bone-mineral density⁴¹⁷ and anti-oestrogenic, anti-proliferative, pro-apoptotic, anti-oxidative and anti-inflammatory processes.⁴¹⁸ However more research is needed to determine the relative effect of legumes/beans and soy foods themselves as opposed to isoflavones specifically.

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.⁴¹⁹ Phytochemicals and several vitamins and minerals found in vegetables and fruit are thought to protect against some cancers by a range of mechanisms. Vegetables in the green leafy and *Brassica* subgroup are particularly high in folate⁵ 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.⁸ 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.⁸ 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.^{420,421}

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.⁸ Lycopenes found in tomatoes are thought to be protective of prostate cancer, particularly when tomatoes are cooked in olive oil.⁴³ Carotenoids found predominantly in orange, red and yellow fruit and vegetables are also thought to be associated with maintenance of immune function.⁸ Dithiolthiones and isothiocyanates (found in *Brassica* or cruciferous vegetables) and allyl sulphides (found in *Allium* vegetables) have been shown to stimulate detoxification processes.³⁶ The anti-inflammatory action of other phytochemicals, such as flavonoids, is also thought to be important in reducing cancer risk.⁴²² 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.⁴³ 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.^{423,424}

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:^{8,425}

- 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.

Dietary fibre from vegetables and fruit is thought to reduce the risk of some cancers.⁸ However, although a probable decreased risk of colorectal cancer with intake of foods containing dietary fibre has been described in the WCRF report (see Appendix F),⁴³ only one of the human trials⁴²⁶ 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.⁸

2.2.6 Practical considerations: enjoy plenty of vegetables, including different types and colours, legumes/beans, and enjoy 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 (see Tables 2.5 and 2.6). The most recent dietary surveys^{12,45} show that consumption of vegetables 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. Based on the most recent consumption data, to meet the recommended food group intakes, most adults should increase their total consumption of vegetables by more than 30%.⁹ 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 to increase the variety of vegetables consumed.⁹

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 Tables 2.5 and 2.6). The most recent dietary surveys^{12,45} show that fruit consumption should approximately double to meet recommended intakes.⁹

The companion resources provide more detailed information on the recommended number of serves and serve sizes of vegetables, legumes/beans and fruit required for different omnivore population groups. Fruit and vegetables do not need to be perfectly shaped or unmarked to provide nutritional value. Discussion of dietary patterns and the environment is included in Appendix G.

Table 2.5: Minimum recommended number of serves of vegetables, legumes/beans and fruit per day

	Recommended number of serves per day*		
	Age (years)	Vegetables and legume/beans	Fruit
Boys	2–3	2 ½	1
	4–8	4 ½	1 ½
	9–11	5	2
	12–13	5 ½	2
	14–18	5 ½	2
Men	19–50	6	2
	51–70	5 ½	2
	70+	5	2
Girls	2–3	2 ½	1
	4–8	4 ½	1 ½
	9–11	5	2
	12–13	5	2
	14–18	5	2
	Pregnant (up to 18 years)	5	2
	Breastfeeding (up to 18 years)	5 ½	2
Women	19–50	5	2
	51–70	5	2
	70+	5	2
	Pregnant (18–50 years)	5	2
	Breastfeeding (18–50 years)	7 ½	2

Note: *Additional amounts of the Five Food Groups or unsaturated spreads and oils or discretionary choices are needed only by people who are taller or more active to meet additional energy requirements.

Source: *Food Modelling System*.⁹

Table 2.6: Standard serve size equivalents for vegetables, legumes/beans and fruit

Food group	Serve sizes (100 –350kJ)
Vegetables and legumes/beans	75g (½ cup) cooked green or <i>Brassica</i> or cruciferous vegetables
	75g (½ cup) cooked orange vegetables
	75g (½ cup) cooked dried or canned beans, chickpeas or lentils, no added salt
	75g (1 cup) raw green leafy vegetables
	75g starchy vegetables (e.g. ½ medium potato, or equivalent of sweet potato, taro, sweet corn or cassava)
	75g other vegetables e.g. 1 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 (½ cup) 100% fruit juice**
	30g dried fruit** e.g. 4 dried apricot halves, 1½ tablespoons of sultanas

Notes: *Preferably with no added sugar

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

Source: *Food Modelling System*.⁹

Vegetables and fruit should be stored and prepared properly to maintain nutrient status. Vegetables should be washed to remove microbes and surface debris, and overcooking should be avoided. Root vegetables should be peeled or scrubbed (see Chapter 5). Tinned and/or frozen varieties are nutritious alternatives to raw produce.

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.⁴²⁷ While fruit and vegetables are valuable foods for pregnant women, pre-prepared or pre-packaged cut fruit and vegetables should be avoided due to risk of listeriosis.^{349,428}

Infants

Pureed and mashed vegetables, including legumes/beans, and fruit are important in the diets of infants from around 6 months of age. Choices should be varied to ensure adequate energy (kilojoule) and nutrient intake. Fruit juice is not recommended for infants. When additional liquids are required for infants, water is preferable. All acidic drinks, including juices, increase the risk of dental erosion.^{351,429}

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 ten times before being accepted.⁴³⁰ Whole fruit is preferable to fruit juice due to its higher fibre content.

Children and adolescents

The recommended quantities of vegetables and fruit intakes for children and adolescents vary depending on their age and sex.⁹ To meet the dietary recommendations, children and adolescents need to approximately double their overall vegetable consumption⁹ and increase the overall variety of vegetables consumed. 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 preschoolers. 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 and territories 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.⁴³¹⁻⁴³⁸ Fruit can be 'fast food' to help satisfy increasing appetites. Unless prescribed by a dietitian, special diets that restrict intake of any fruit or vegetables should be avoided for children and adolescents. The intake of energy-dense hot fried potato chips as a snack or with meals should be limited.

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 have higher intakes of fruit and vegetables than younger adults.⁴⁵ Due to poor dentition, softer textured or cooked vegetables and fruit may be preferable for some older people. Tinned and/or frozen varieties are nutritious alternatives to raw produce.

Aboriginal and Torres Strait Islander peoples

Very low intakes of vegetables and fruit have been described among Aboriginal and Torres Strait Islander groups in urban and rural communities.^{30,353,439,440} Availability of quality fresh produce can be a particular problem in many remote areas.^{28317,356} Frozen and canned vegetables and fruit, plus available traditional plant foods, are nutritious alternatives.

2.3 Enjoy grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties

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 (in particular saturated fat) and/or salt/sodium, such as cakes.

Key nutrients in wholegrain foods include starch (complex carbohydrate), 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.^{35,36} 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.⁴⁴¹

2.3.2 The evidence for consuming 'grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties'

The evidence for the association of grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties, with reduced risk of cardiovascular disease, type 2 diabetes and excess weight gain has strengthened since the 2003 edition of the dietary guidelines.

The literature is difficult to interpret because studies use varied definitions of 'wholegrain'. FSANZ applies the term to products that use every part of the grain (cereal) including the outer layers, bran and germ even if these parts are separated during processing and regardless of whether the grain (cereal) is in one piece or milled into smaller pieces.⁴⁴¹ In the literature review for these Guidelines, the most commonly used definition was found to be that of Jacobs et al 1998,⁴⁴² who defined wholegrain foods as those containing 25% or more of wholegrains. Other studies have included bran cereals as part of the definition of wholegrain and others only examined certain types of grain (cereal) food such as oats.

Table 2.7: Evidence statements for consuming 'grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties'

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 one to three serves per day of wholegrain cereals is associated with a reduced risk of cardiovascular disease.	B
Consumption of cereal foods (especially three serves a day of wholegrains) is associated with reduced risk of type 2 diabetes	B
Consumption of three to five serves per day of cereal foods (mainly wholegrain) is associated with a reduced risk of weight gain.	B
Consumption of one to three serves per day of cereals high in fibre is associated with reduced risk of colorectal cancer in adults.	C

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

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; Evidence Report, Section 6.3).^{226,443-458} 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 one to three 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; Evidence Report, Section 6.7).^{445,457,459-466} The evidence supports three serves per day of wholegrain foods conferring between 21 % and 42% reduction in risk of type 2 diabetes.
- **Excess weight:** There is evidence of a probable association between consumption of three to five serves per day of grain (cereal) foods (mainly wholegrain) and reduced risk of weight gain (Grade B; Evidence Report, Section 6.6).^{215,220-229}

Cancer

- **Colorectal cancer:** There is recent evidence suggesting that consumption of one to three serves of cereals high in dietary fibre per day is associated with reduced risk of colorectal cancer in adults (Grade C; Evidence Report, Section 6.2).^{397,467-471} Although previously the WCRF report noted a probable relationship, it recently reviewed the evidence and found it convincing that fibre-rich foods offer protection against colorectal cancer (see Appendix F).^{43,472} This is also supported by a recent systematic review and dose response meta-analysis of prospective studies showing that three serves of wholegrain and high fibre cereals per day reduced the risk of colorectal cancer.⁴⁷³
- **Other cancers:** Recent evidence is inconclusive for an association regarding the consumption of grain (cereal) foods and risk of other cancers in adults (Evidence Report, Section 6.1).

2.3.3 How consuming grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties may improve health outcomes

Much depends on which wholegrain is being considered.⁴⁷⁴ 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 from oats reduces the requirement for insulin.⁴⁷⁵ 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.⁴⁷⁶ Other sources of dietary fibre, such as psyllium may act in a similar fashion. Grain (cereal) foods also contain starch (complex carbohydrate) 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.⁴⁷⁷ 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.⁴⁷⁸

Because wholegrains contain more nutrients and phytochemicals, concentrated in the bran and germ,⁴⁷⁹ they are likely to have greater effects than refined grains (cereals). Wholegrains contain phenolic compounds with strong anti-oxidant capacity that may be protective against processes involved in the pathology of type 2 diabetes, cardiovascular disease and cancer.⁴⁸⁰ Choosing wholegrain options may also assist with satiety and help in not over consuming food beyond energy (kilojoule) requirements.

The 2003 edition of the dietary guidelines discussed the glycaemic index (GI) of individual foods as a physiologically based classification of carbohydrate-containing foods according to their potential to raise blood glucose.³⁶ Various factors such as the particle size of milled grains (cereals), 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 may affect the GI value of a food and may limit practical application.¹⁹⁹ Additionally, the accuracy with which published values can be used to predict GI when foods are eaten together as a meal is limited.⁴⁸¹ Lower GI diets may assist in the management of type 1 and type 2 diabetes but are not associated with weight loss (see Chapter 1).^{36,198} Further research on GI and health outcomes may be required to investigate other potential associations.

The 2003 edition of the dietary guidelines also noted that consumption of dietary fibre assists in maintaining the health and function of the digestive system, for example in preventing constipation.^{35,36}

2.3.4 Practical considerations: enjoy grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties

Consuming at least four to six serves of grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties, per day is recommended for Australian adults, while the amount recommended for children and adolescents depends on their age and sex (see Tables 2.8 and 2.9), ranging from four serves a day for 2–8 year olds to seven a day for older adolescents.

The companion resources provide more detailed information on the recommended number of serves and serve sizes of grain (cereal) foods required for different population groups. Discussion of dietary patterns and the environment is included in Appendix G.

Table 2.8: Minimum recommended number of serves of grain (cereal) foods per day, mostly wholegrain and/or high cereal fibre varieties

	Age (years)	Number of serves*
Boys	2–3	4
	4–8	4
	9–11	5
	12–13	6
	14–18	7
Men	19–50	6
	51–70	6
	70+	4 ½
Girls	2–3	4
	4–8	4
	9–11	4
	12–13	5
	14–18	7
	Pregnant (up to 18 years)	8
	Breastfeeding (up to 18 years)	9
Women	19–50	6
	51–70	4
	70+	3
	Pregnant (19–50 years)	8 ½
	Breastfeeding (19–50 years)	9

Note: *Additional amounts of the Five Food Groups or unsaturated spreads and oils or discretionary choices are needed only by people who are taller or more active to meet additional energy requirements.

Source: Food Modelling System.⁹

Table 2.9: Standard serve size equivalents for grain (cereal) mostly wholegrain and/or high cereal fibre varieties

Food group	Serve sizes (500kJ)
Grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties, such as breads, cereals, rice, pasta, noodles, polenta, couscous, oats, quinoa and barley	1 slice of bread or ½ a medium roll or flat bread (40g)
	½ cup cooked rice, pasta, noodles, barley, buckweheat, semolina, polenta, bulgur or quinoa (75–120g)
	½ cup cooked porridge (120g), 2/3 cup wheat cereal flakes (30g) or ¼ cup muesli (30g)
	3 crispbreads (35g)
	1 crumpet (60g) or 1 small English muffin or scone (35g)
	¼ cup flour (30g)

Source: Food Modelling System⁹

Based on current consumption data, to meet recommended food group intakes⁹ 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.⁹ Refined grain (cereal) food products with high levels of added sugar, fat (in particular saturated 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*⁹ and companion resources 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 starches (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 saturated fat are kept to a minimum.

Pregnant and breastfeeding women

As periconceptional folic acid intake helps protect against neural tube defects in the developing foetus,⁴⁸² the mandatory fortification of flour used for bread making in Australia with folic acid⁴⁸³ provides an additional reason for women of reproductive age to consume bread. However, for women planning a pregnancy and during the first 3 months of pregnancy, a daily folic acid supplement that contains at least 400 µg of folic acid is recommended in addition to eating foods that are naturally rich in folate or are fortified with folic acid.^{483,484}

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 µg/day) for women planning a pregnancy, throughout pregnancy and while breastfeeding.³²³

Infants

Iron-fortified cereals can be one of the first foods offered to infants at around 6 months of age.

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.

People with vegetarian or vegan dietary patterns

Wholegrain foods are valuable sources of nutrients for the whole population but particularly important in vegetarian and vegan diets as a source of iron and zinc.

2.4 Enjoy lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans

2.4.1 Setting the scene

Lean meats and poultry, fish, eggs and plant-based alternatives such as tofu, legumes/beans, nuts and seeds are an important component of Australian cuisine, culture and lifestyle. The 'lean meats 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 that may be more important in the typical high-protein Australian diet. Important nutrients include iodine, iron, zinc and other minerals, vitamins, especially B₁₂, and essential fatty acids including omega-3 long chain polyunsaturated fatty acid (omega-3 LCPUFAs). All indigenous Australian fish contain omega-3 LCPUFAs.^{485,486} 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.^{35,36,198,360}

Processed and cured meats can be high in added salt and saturated fat and are not recommended as substitutes for unprocessed meat. These foods fit in the 'discretionary foods' category. Lean poultry and eggs can be included in the diet within the overall recommended quantities for this whole food group.⁹ 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 purposes of these Guidelines.⁹

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 and vitamins A and D (some species only) as well as omega-3 LCPUFAs. Evidence of the health benefits of fish consumption is consistently recognised in international dietary guidelines.^{35,36,198,360}

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 fatty acids and are rich in polyphenols, phytosterols and micronutrients including folate, several valuable forms of vitamin E, selenium, magnesium and other minerals. They are nutritious alternatives to meat, fish and eggs, and play an important role in plant-based, vegetarian and vegan meals and diets.⁹

Legumes/beans, including lentils, tofu and tempeh, 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 any diet, and are especially useful for people who consume plant-based meals.⁹

2.4.2 The evidence for consuming 'lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans'

In the following studies, serve sizes of the different foods are as included in the companion resources (see also Table 2.12, Section 2.4.4).

Table 2.10: Evidence statements for consuming 'lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans'

Evidence statement	Grade
Consumption of greater than 100–120 g/day red meat per day 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 increased risk of renal cancer.	C
Consumption of at least two 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 two or more times per week is associated with reduced risk of age-related macular degeneration.	C
Consumption of nuts (65–110g per day) is associated with a reduction in serum cholesterol.	C

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

Lean meats

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 isolation³⁶ or the ability of the body to absorb nutrients rather than looking at the whole food. Since the 2003 edition of the dietary guidelines, 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.

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. A large cohort trial that found modest increases in total mortality, cardiovascular mortality and cancer mortality with red and processed meat intakes⁴⁸⁷ was not included due to lack of clarity over the inclusion of processed meats, liver and sausages with unprocessed red meat.

Cancer

- **Colorectal cancer:** There is evidence of a probable association between consumption of red meat and increased risk of colorectal cancer (Grade B; Evidence Report, Section 4.7).^{43,488-496} The WCRF reported a convincing relationship between red and processed meat and increased risk of colorectal cancer (see Appendix F).⁴³ Several studies from Asian countries showed no increased risk of colorectal cancer associated with low intakes of red meat such as 27g per day⁴⁹⁴ and 42g per day.⁴⁹²
- **Renal cancer:** The evidence suggests that consumption of red meat is associated with an increased risk of renal cancer (Grade C; Evidence Report, Section 4.6).
- **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; Evidence Report, Section 4.1).^{43,497,498} The evidence suggests that consumption of red meat is not associated with risk of prostate cancer (Grade C; Evidence Report, Section 4.3).^{43,499,500}
- **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; Evidence Report, Section 4.2).^{43,501} The WCRF report also found limited evidence to suggest red meat increases the risk of pancreatic cancer (see Appendix F).⁴³
- **Other cancers:** Recent evidence is inconclusive for an association regarding the consumption of red meat and breast and lung cancer (Evidence Report, Sections 4.4 and 4.5).

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

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.

Cancer

Recent evidence examining an association between poultry consumption and breast or colorectal cancer is inconclusive (Evidence Report, Sections 10.1 and 10.2). The WCRF report also concluded that the evidence is too limited in amount, consistency or quality to draw any conclusions about the relationship between poultry and cancer risk (see Appendix F).⁴³

Fish

The evidence regarding the health benefits of fish has strengthened since the 2003 edition of the dietary guidelines. 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 (omega-3 LCPUFAs) 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.³⁶ 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 LCPUFAs, 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.

Cardiovascular disease

- **Incidence and mortality:** 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; Evidence Report, Section 9.1).⁵⁰²⁻⁵⁰⁵ and with reduced risk of mortality from cardiovascular disease (Grade C; Evidence Report, Section 9.1).^{502,504,506-513}
- **Stroke:** The evidence suggests that consumption of fish at least twice a week is associated with a reduced risk of stroke (Grade C; Evidence Report, Section 9.5).^{505,514-516}
- **Other cardiovascular conditions:** 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 heart failure, atrial fibrillation and high blood lipids.

Cancer

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

Other conditions

- **Dementia:** The recent body of evidence demonstrates that it is probable that the consumption of fish more than once a week is associated with a reduced risk of developing dementia in older adults (Grade B; Evidence Report, Section 9.2).^{503,517-524}
- **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; Evidence Report, Section 9.3).⁵²⁵⁻⁵²⁹
- **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; Evidence Report, Section 9.4).⁵³⁰

Eggs

Since the 2003 edition of the dietary guidelines, the evidence associating egg consumption with health outcomes has not changed greatly (Evidence Report, Section 11). There do not appear to be any increased health risks associated with consumption of eggs.⁵³¹⁻⁵³³ There is recent evidence to suggest that consumption of eggs every day is not associated with increased risk of coronary heart disease (Grade C; Evidence Report, Section 11.1).^{455,534-551}

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.²³⁰⁻²³⁵ Since the 2003 edition of the 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 (Grade C; Evidence Report, Section 8.1).²³⁰⁻²³⁵

Cardiovascular disease 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; Evidence Report, Section 8.2).^{231,232,234,235,552} Results of two large cohort studies report that substituting one serve of nuts for red meat per day results in a significant reduction (19–30%) in cardiovascular disease risk.^{553,554}
- **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; Evidence Report, Section 8.1).²³⁰⁻²³⁵

Cancer

No recent studies investigating the association of consumption of nuts and seeds with cancer were identified.

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 Evidence Report, Section 7.

2.4.3 How consuming lean meats and poultry, fish, eggs, tofu, 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 N-nitroso compounds has been suggested as a possible link between red meat and colorectal cancer^{555,556} or that inflammatory effects of haem iron may be relevant⁵⁵⁷ and further research is occurring. 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.^{558,559}

It has been suggested that components of meat such as saturated fats, or other dietary and lifestyle factors associated with meat intake, may be relevant factors for further study. Previous evidence related to the amount of 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 omega-3 LCPUFAs. 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 Section 3.1.1).

Nut consumption provides benefits by enhancing anti-inflammatory processes⁵⁶⁰ and lowering serum cholesterol possibly due to the presence of phytosterols, which reduce cholesterol re-absorption,⁵⁶¹ 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.⁵⁶² 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.⁵⁶⁰ Other nuts are also significant sources of arginine. Proposed mechanisms for effects on weight control include increased satiety, increased faecal fat excretion, increased thermogenesis⁵⁶³ and increased fat oxidation.⁵⁶⁴

2.4.4 Practical considerations: enjoy lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans

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.⁴⁵ Adult men and women daily consumed 99g and 54g of lean red meat respectively.⁴⁵

The recommended consumption of lean meats and poultry, fish, eggs, tofu, nuts and seeds and legume/beans for children and adults is one to three serves a day depending on age and sex (see Tables 2.11 and 2.12). During pregnancy, three to four 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 (one serve [65 g] per day) of lean meats is recommended for Australian adults.⁹

The companion resources provide more detailed information on the recommended number of serves and serve sizes of lean meats, poultry, fish, eggs, nuts and seeds and legumes/beans required for different population groups. Discussion of dietary patterns and the environment is included in Appendix G.

Table 2.11: Minimum recommended number of serves of lean meats, poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans per day

	Age (years)	Number of serves*
Boys	2–3	1
	4–8	1 ½
	9–11	2 ½
	12–13	2 ½
	14–18	2 ½
Men	19–50	3
	51–70	2 ½
	70+	2 ½
Girls	2–3	1
	4–8	1 ½
	9–11	2 ½
	12–13	2 ½
	14–18	2 ½
	Pregnant (up to 18 years)	3 ½
	Breastfeeding (up to 18 years)	2 ½
Women	19–50	2 ½
	51–70	2
	70+	2
	Pregnant (19–50 years)	3 ½
	Breastfeeding (19–50 years)	2 ½

Note: *Additional amounts of the Five Food Groups or unsaturated spreads and oils or discretionary choices are needed only by people who are taller or more active to meet additional energy requirements.

Source: Food Modelling System.⁹

Table 2.12: Standard serve size equivalents for lean meats, poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans

Food group	Serve sizes (500–600kJ)
Lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans	65g cooked lean red meats (e.g. beef, lamb, pork, venison or kangaroo) or ½ 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 (150g) cooked dried beans, lentils, chickpeas, split peas, or canned beans
	170g tofu
	30g nuts or seeds or nut/seed paste, no added salt*

Note: *Only to be used occasionally as a substitute for other foods in the group.

Source: Food Modelling System.⁹

Based on most recent consumption data, to meet recommended intakes, omnivorous adults in Australia would need to consume 40% more poultry, fish, seafood, eggs, tofu, nuts and seeds, and legumes/beans but men consuming an omnivorous diet would need to consume around 20% less lean red meat than currently.⁹ For children 2–16 years of age, around 30–85% more 'poultry, fish, seafood, eggs, tofu, nuts and seeds, and legumes/beans' and around 25–70% more lean red meat (by replacing fatty cuts of meat) would be required above current intakes to meet recommendations. Exact quantities depend on age and sex.⁹

Depending on age and sex, health benefits may be seen with consumption of 1.4 to 2.8 serves (140–280g) of fish 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.⁴⁵ To meet recommended food group intakes, fish consumption will need to increase by more than 40%, particularly for men.⁹ The extent to which Australian fish populations are sufficient to meet the guideline advice needs consideration. 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.^{565,566} Some imported fish may have lower levels of omega-3 LCPUFAs compared with Australian seafood.^{485,486}

Legumes are not only important in vegetarian and vegan diets but their significant nutrient profile could benefit all Australians. There is variability in the amount of legumes recommended in the diet; the dietary models developed for these guidelines include a minimum of two serves per week.⁹

Previous reported intakes of nuts and seeds in Australia have been very low (only around 4g per day for adults).⁴⁵ Consumption may have risen since. The dietary models 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, including in omnivore Foundation Diet models.⁹

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

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 pâté and meat spreads should be avoided due to risk of listeriosis (see Chapter 5).^{349,428} 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 discuss checking their iron status 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.⁵⁶⁷ Pregnant women are advised:

- if consuming shark/flake, marlin or broadbill/swordfish, have no more than one serve (100g cooked) per fortnight and no other fish that fortnight
- if consuming orange roughy (deep sea perch) or catfish, have one serve (100 g cooked) per week and no other fish that week.^{349,567}

Pregnant women should avoid eating foods containing raw eggs, because of risk of salmonella.⁴²⁸

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.⁵⁶⁸⁻⁵⁷²

Infants

Foods from the meats and alternatives group are important in the diets of infants over the age of around 6 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.³⁵¹ The texture should be suitable (e.g. peanut butter/paste). Only children over 3 years should be offered whole nuts due to the risk of choking.³⁵¹

Children and adolescents

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

Women of child-bearing age

Menstrual loss doubles the iron requirements of young women compared to young men,³⁶ although that increased requirement is lessened for women taking the oral contraceptive pill. Good food sources of iron are particularly important for this group. Lean red meats and some types of seafood are excellent sources of iron.⁹ Poultry, legumes, tofu, nuts and eggs also provide some iron. For pregnant women, additional sources of iron may be required and an appropriate health professional should be consulted.

Older people

Lean meats and poultry, fish, eggs, tofu, 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.⁹ 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.

People with vegetarian or vegan dietary patterns

Nuts and seeds and legumes/beans are important foods for people who choose vegetarian or vegan dietary patterns and meals without meat as they can provide an alternative source of nutrients. For several nutrients, including iron, calcium and vitamin B₁₂, animal foods are highly bioavailable sources and care needs to be taken to ensure a variety of alternatives if these foods are excluded.

For lacto-ovo vegetarian diets, eggs provide vitamin B₁₂ and milk, yoghurt and cheese products can supply calcium and vitamin B₁₂. The Food Modelling System used a ratio of a 5:1:1 'legume: egg: nuts and 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.⁹ As no Australian national data were available about choices within food categories for lacto-ovo vegetarians, the same proportions within food groups (e.g. for vegetables, fruit, cereals) were used as for the equivalent omnivore group.

For pregnant women and children consuming vegetarian diets, it should be noted that iron was limiting in the diets as modelled and additional sources of iron would be essential to meet their requirements.⁹ Vitamin B₁₂ is only available from animal food sources and supplementation may be needed by people with vegetarian or vegan dietary patterns.

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 Bridge⁵⁷³ – exposure in Australia is generally low.⁵⁷⁴ The Food and Agriculture Organisation of the United Nations (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.⁵⁷⁵

2.5 Enjoy 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. Fermented milk products that contain viable micro-organisms in the product are also available. Other sources for milk and related products include goats, sheep, soy, almonds and various cereals including oats and rice. Milk substitutes that are not fortified with calcium and other nutrients are not included in this food group.

Milk, cheese and yoghurt have various health benefits and 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. However, in reflecting on Australian consumption patterns and ensuring all nutrient requirements are met within energy requirements, it is recommended that reduced fat varieties should be chosen on most occasions.⁹ The inclusion of mostly or all full fat milk, cheese and yoghurts proportionally increases total fat, saturated fat and overall energy components of the diet. Unmodified milk from animal sources such as cow's, goats or sheep, should not be given as a main drink before 12 months of age.

Alternatives to milk, cheese and yoghurt include calcium-enriched legume/bean/cereal milk products such as calcium-enriched soy, rice and oat drinks. Soy (except soy follow-on formula) and other nutritionally incomplete plant-based drinks such as rice, oat, coconut or almond drinks are inappropriate alternatives to breast milk or formula in the first 12 months. Fortified soy drink or calcium-enriched plant-based drinks can be used after 12 months of age under health professional supervision, as long as other sources of protein and vitamin B₁₂ are included in the diet. Further information about legumes/beans is included with vegetables and meat and alternative groups above.

It should be noted that reduced fat varieties of milks and/or plant-based drinks are not suitable as a drink for children under the age of 2 years due to energy requirements for growth and they may also not be appropriate for adults over the age of 70 years due to reduced body weight and energy intake.

2.5.2 The evidence for consuming 'milk, yoghurt, cheese and/or alternatives, mostly reduced fat'

The evidence for the health benefits of consumption of these dairy foods (mainly reduced fat varieties) has strengthened since the 2003 edition of the dietary guidelines,³⁶ 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.³⁶

Table 2.13: Evidence statements for consuming 'milk, yoghurt, cheese and/or alternatives, mostly reduced fat'

Evidence statement	Grade
Consumption of at least two serves per day of dairy foods (milk, yoghurt, and cheese) is associated with reduced risk of ischemic heart disease and myocardial infarction.	B
Consumption of two or more serves of dairy foods per day is associated with reduced risk of stroke.	B
Consumption of three serves of low fat dairy foods is associated with reduced risk of hypertension.	B
Consumption of more than one serve of dairy foods per day, especially milk, is associated with a reduced risk of colorectal cancer.	B
Consumption of three or more serves of milk per day is not associated with risk of renal cell cancer.	B
Consumption of three serves of any milk, cheese or yoghurt products a day is associated with reduced risk of hypertension.	C
Consumption of two to four serves of dairy foods per day is associated with reduced risk of metabolic syndrome.	C

Evidence statement	Grade
Consumption of at least one and a half serves of dairy foods (milk, yoghurt, cheese) per day is associated with reduced risk of type 2 diabetes.	C
Consumption of more than one serve 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

Notes: The term 'dairy' refers to cow's milk and the yoghurt and cheese produced from it.

Grades – A: convincing association, **B:** probable association, **C:** suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

Cardiovascular disease, type 2 diabetes and excess weight

- **Coronary heart disease:** It is probable that the consumption of at least two serves per day of dairy foods (milk, cheese and yoghurt) is associated with reduced risk of ischaemic heart disease and myocardial infarction (Grade B; Evidence Report, Section 5.3).⁵⁷⁶
- **Stroke:** It is probable that the consumption of two or more serves of dairy foods per day (milk, cheese and yoghurt) is associated with reduced risk of stroke (Grade B; Evidence Report, Section 5.4)^{576,577} particularly reduced fat varieties.
- **Hypertension:** It is probable that consumption of three serves of low fat dairy foods (milk, cheese and yoghurt) is associated with reduced risk of hypertension (Grade B; Evidence Report, Section 5.5). The evidence also suggests that consumption of three serves of any milk, cheese or yoghurt products per day is associated with reduced risk of hypertension (Grade C; Evidence Report, Section 5.5).^{213,578-581}
- **Type 2 diabetes:** The evidence suggests that consumption of two to four serves of dairy foods (milk, cheese, yoghurt) per day is associated with reduced risk of metabolic syndrome (Grade C; Evidence Report, Section 5.7)^{576,582} and that consumption of at least one and a half serves of milk, cheese and yoghurt per day is associated with reduced risk of type 2 diabetes (Grade C; Evidence Report, Section 5.6).^{213,576,583}
- **Excess weight:** The evidence suggests that consumption of dairy foods is not associated with weight change or risk of obesity in adults (Grade C; Evidence Report, Section 5.8)²⁰⁹⁻²¹⁴ and consumption of milk is not associated with BMI or change in BMI in childhood (Grade C; Evidence Report, Section 5.9).^{215,217-219,584,585} Total energy intake is always important in weight management.⁹

Cancer

- **Rectal and colorectal cancer:** Recent evidence suggests it is probable that consumption of more than one serve of dairy foods per day (especially milk) is associated with reduced risk of colorectal cancer (Grade B; Evidence Report, Section 5.11)^{492,586,587} The evidence suggests that consumption of more than one serve of milk per day is associated with reduced risk of rectal cancer (Grade C; Evidence Report, Section 5.12).⁵⁸⁶⁻⁵⁸⁸ The WCRF report found probable evidence that consuming milk is associated with reduced the risk of colorectal cancer (see Appendix F).⁴³
- **Renal cell, breast and endometrial cancer:** It is probable that consumption of three or more serves of milk per day is not associated with risk of renal cell cancer (Grade B; Evidence Report, Section 5.13).⁵⁸⁹ There is evidence to suggest that mean consumption of one serve of dairy food (milk cheese, yoghurt) per day is not associated with the risk of breast cancer (Grade C; Evidence Report, Section 5.15)^{590,591} and that consumption of dairy food (milk, cheese, yoghurt) is not associated with risk of endometrial cancer (Grade C; Evidence Report, Section 5.16).⁵⁹²
- **Prostate cancer:** Recent evidence suggesting an association between milk consumption and prostate cancer is inconclusive (Evidence Report, Section 5.14). The WCRF also found limited evidence that consumption of milk and dairy products is associated with risk of prostate cancer (see Appendix F).⁴³

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.⁸

- **Bone mineral density:** Recent evidence suggests that consumption of dairy foods (particularly milk) is associated with improved bone mineral density (Grade C; Evidence Report, Section 5.1)^{590,593-598} but this is contradicted by evidence suggesting that less than one serve of milk per day during adult life is not associated with risk of osteoporotic or hip fracture (Grade C; Evidence Report, Section 5.2).^{599,600}

2.5.3 How consuming milk, yoghurt, cheese and/or alternatives, mostly reduced fat, may improve health outcomes

There is a range of bioactive substances in milk products that may have a contributing role in health outcomes. The mechanism by which milk, yoghurt and cheese consumption (especially milk) affect blood pressure⁶⁰¹ might be due to calcium modulating endothelial function.⁶⁰² 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 events⁶⁰³ suggested that calcium supplements, without co-administered vitamin D, were associated with an increased risk of myocardial infarction. The same group has more recently reported a similar effect from calcium supplements with vitamin D.⁶⁰⁴ 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.⁶⁰⁵

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.⁶⁰⁶

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.⁶⁰⁷

A randomised controlled trial of children who were overweight found that 200ml of milk per day combined with nutrition education was associated with a reduced consumption of sugar-sweetened drinks and increased lean body mass.⁶⁰⁸

2.5.4 Practical considerations: enjoy milk, yoghurt, cheese and/or alternatives, mostly reduced fat

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 (see Tables 2.14 and 2.15).⁹ Varieties of cheese that are lower in salt are also preferable (see Section 3.2).

The companion resources provide more detailed information on the recommended number of serves and serve sizes of milk, yoghurt, cheese and/or alternatives required for different omnivore population groups. Discussion of dietary patterns and the environment is included in Appendix G.

Table 2.14: Minimum recommended number of serves of milk, yoghurt, cheese and/or alternatives per day, mostly reduced fat

Age (years)		Number of serves*
Boys	2–3	1 ½
	4–8	2
	9–11	2 ½
	12–13	3 ½
	14–18	3 ½
Men	19–50	2 ½
	51–70	2 ½
	70+	3 ½
Girls	2–3	1 ½
	4–8	1 ½
	9–11	3
	12–13	3 ½
	14–18	3 ½
	Pregnant (up to 18 years)	3 ½
	Breastfeeding (up to 18 years)	4
Women	19–50	2 ½
	51–70	4
	70+	4
	Pregnant (19–50 years)	2 ½
	Breastfeeding (19–50 years)	2 ½

Note: *Additional amounts of the Five Food Groups or unsaturated spreads and oils or discretionary choices are needed only by people who are taller or more active to meet additional energy requirements.

Source: Food Modelling System.⁹

Table 2.15: Standard serve size equivalents for milk, yoghurt, cheese and/or alternatives, mostly reduced fat

Food group	Serve sizes (500–600kJ)
Milk, yoghurt, cheese and/or alternatives, mostly reduced fat	1 cup (250ml) milk – fresh, UHT long life or reconstituted powdered
	1/2 cup (120ml) evaporated unsweetened milk
	3/4 cup (200g) yoghurt
	40g (2 slices or 4 x 3 x 2 cm piece) hard cheese e.g. cheddar
	1/2 cup (120g) ricotta cheese
	1 cup (250ml) soy, rice or other cereal drink with at least 100mg of added calcium per 100ml

Source: Food Modelling System.⁹

The 1995 National Nutrition Survey⁴⁵ 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 men 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 adults⁹ 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 or other drinks could be substituted for these dairy foods.

Pregnant and breastfeeding women

Milk, yoghurt and cheese product intake during pregnancy and lactation have benefits for both women and their babies. Reduced fat milk, yoghurt and cheese products are recommended during pregnancy and while breastfeeding.^{428,572}

Pregnant women are advised to avoid consuming unpasteurised dairy products and soft, semi-soft and surface-ripened cheeses (e.g. brie, camembert, ricotta, fetta and blue cheeses) due to risk of listeriosis.^{349,609}

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, with cereals or as yoghurt. Any milk given to infants (as a drink) should be breast milk or infant formula.

Milk given to infants and toddlers should be pasteurised. As children under 2 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 are suitable after 2 years of age.

For further information, see the Infant Feeding Guidelines.³⁵¹

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.⁴⁵ Diets restricting intake of milk and milk products (or calcium-fortified alternatives) are not generally suitable for growing children and adolescents. Suspected lactose intolerance and allergy in children and adolescents should be confirmed by a medical practitioner.

Older people

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

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.^{610,611} Lactose-intolerant adults and children often avoid milk and milk products, however up to 250ml of milk may be well tolerated if broken up throughout the day and consumed with other foods.⁶¹² Cheese contains little lactose and the lactose in yoghurt is partially broken down by bacteria that thicken the yoghurt, so should be well tolerated. Lactose-free dairy products and calcium-fortified beverages are also available.

People with vegetarian or vegan dietary patterns

The 2003 edition of the dietary guidelines addressed vegetarian and vegan diets and their influence on calcium needs because of the relatively high oxalate and phytate content in some plants. However, lacto-ovo vegetarians appear to have calcium intakes similar to those of omnivores. Those who follow a vegan or vegetarian diet and avoid milk products, should consume alternative calcium-fortified products and seek advice from a health professional about whether they need to take supplements.³⁶

2.6 Drink plenty of water

2.6.1 Setting the scene

Water is essential for life. All biochemical reactions occur in water. It fills the spaces between the cells and helps form structures of large molecules such as protein and glycogen. Water is also required for digestion, absorption and transportation, as a solvent for nutrients, for elimination of waste products and to regulate body temperature.⁶¹³

Water is lost from the body in sweating (from 100ml to several 1,000ml/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.⁶¹⁴ Excluding obvious sweating, the normal turnover of water has been estimated at approximately 4% of total body weight per day in adults. In a 70 kg adult, this is equivalent to 2,500–3,000ml/day.

Some of this turnover will come from the water contained in foods but the majority of dietary intake comes from free water and/or other water-containing fluids.^{8,36} 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.^{615,616}

Details of water requirements for all ages are found in the NRV Document, but for adult males and females the estimated daily average intake is 2.6 L and 2.1 L (8–10 cups) respectively.⁸ This includes all fluids, but it is preferable that the majority of this intake is from plain water. Water requirements can increase considerably in high temperatures and in increased physical activity.

There are no reports of any specific hazards from the usual consumption of water by healthy individuals. 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 plenty of water’

Water is essential for human life because it is required in amounts that exceed the body’s ability to produce it. Given this, no recent studies investigating the relationship between water consumption and health outcomes were identified in the literature review. Many commonly consumed fluids provide water, however they may also be acidic such as low-joule soft drink or contain added sugar, alcohol or caffeine. Recent studies that investigated intake of these fluids and health outcomes are reviewed below.

Table 2.16: Evidence statements for ‘drink plenty of water’

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 four 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 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

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

Reviews have also shown an association between inadequate water consumption and poor health. Dehydration of as little as 2% loss of body weight results in impaired physiological responses and performance.⁸ The reported health effects of chronic mild dehydration and poor fluid intake include increased risk of kidney stones, urinary tract cancers, colon cancer and mitral valve prolapse as well as diminished physical and mental performance.⁸

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; Evidence Report, Section 15.21).⁶¹⁷⁻⁶²¹
 - The evidence suggests that consumption of green and black tea is associated with reduced risk of stroke (Grade C; Evidence Report, Section 15.20).^{619,622-625}
 - The evidence suggests that consumption of coffee is not associated with risk of coronary heart disease (Grade C; Evidence Report, Section 15.13),⁶²⁶⁻⁶²⁹ although this was not the case for cigarette smokers.
- **Blood pressure:** Recent evidence is inconclusive in regard to an association between coffee consumption and systolic blood pressure (Evidence Report, Section 15.14).
- **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; Evidence Report, Section 15.15).⁶³⁰⁻⁶³⁷
- **Excess weight:** It is probable that consumption of sugar-sweetened beverages is associated with weight gain in children and adults (Grade B; Evidence Report, Section 15.1).¹⁸⁵⁻¹⁹⁶

Cancer

- **Breast, colorectal and ovarian cancer:** The evidence suggests that consumption of coffee is not associated with risk of breast cancer,⁶³⁸ colorectal cancer^{494,639-641} or ovarian cancer⁶⁴²⁻⁶⁴⁴ (Grade C; Evidence Report, Sections 15.7, 15.9 and 15.11).
- **Endometrial and hepatocellular cancer:** The evidence suggests that consumption of coffee is associated with decreased risk of hepatocellular cancer⁶⁴⁵ and endometrial cancer^{646,647} (Grade C; Evidence Report, Sections 15.6 and 15.8).
- **Gastric cancers:** The evidence is inconclusive regarding an association between coffee consumption and risk of gastric cancer (Evidence Report, Section 15.5).
- **Other cancers:** The WCRF report states it is unlikely that coffee has any substantial effect on the risk of renal cancers (see Appendix F).⁴³
- **Ovarian and colorectal cancer:** The evidence suggests that consumption of green or black tea is not associated with ovarian cancer (Grade C; Evidence Report, Section 15.18)⁶⁴²⁻⁶⁴⁴ or colorectal cancer (Grade C; Evidence Report, Section 15.19).^{639-641,648,649}
- **Other cancers:** Recent evidence is inconclusive regarding an association between consumption of green and black tea and breast, gastric or lung cancer (Evidence Report, Sections 15.16, 15.17 and 15.22).

Dental health

- The evidence suggests that consumption of soft drinks is associated with increased risk of dental caries in children (Grade C; Evidence Report, Section 15.14) and increased risk of reduced bone strength (Grade C; Evidence Report, Section 15.3).^{195,650-653}
- Fluoride added to tap water provides protection against dental caries.⁶⁵⁴ The mildly alkaline nature of saliva is the key protective element against erosion 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.⁶⁵⁵ 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.⁶⁵⁶ 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 3.3.2). The relationship between the acidity of sweetened drinks and dental erosion is also supported by the American Academy of Pediatrics Committee on School Health.⁶⁵⁷

2.6.3 How drinking plenty of 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 and energy (see Section 3.3), the carbonation process creates an acidic environment that contributes to the erosion of tooth enamel.⁶⁵ Tap water also has the beneficial effects of added fluoride in many communities in Australia and fluid intake can affect saliva production and saliva, which is primarily water and essential for the maintenance of oral health.⁶⁶

The mechanism by which coffee consumption may protect against type 2 diabetes is not clear. It has been reported that insulin resistance is decreased in peripheral tissues after exposure to caffeine⁶⁷ and conversely, the effect of caffeine on thermoregulation has been suggested as a mechanism by which glucose homeostasis is improved.⁶⁸ Other components of coffee, possibly magnesium, may also assist by benefiting insulin sensitivity and reducing risk of type 2 diabetes.⁶⁹ Tea contains polyphenols known as catechins which have been reported to reduce hypertension, atherosclerosis and thrombogenesis.⁶⁹ 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.⁶⁹

2.6.4 Practical considerations: drink plenty of water

Adequate fluid consumption is an integral component of a healthy diet. Water is a good source of fluids and has the advantage of not adding energy (kilojoules) to the diet. It is preferable to meet most fluid needs by drinking plain water. Many commonly consumed fluids such as tea and coffee provide water, although they can have unwanted stimulant effects in susceptible people.

The NHMRC has developed comprehensive guidelines on tap water standards for drinking.⁶⁶ Australian tap water is an ideal option because it is inexpensive and meets high palatability and hygiene standards. Most tap water in Australia is fluoridated, which has been shown to be a safe and effective public health measure. Fluoridation of tap water 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 tap water is limited. Not all bottled waters contain fluoride.

However, access to and availability of clean and safe water may be limited for some population groups, particularly in remote regions of Australia. Discussion of dietary patterns and the environment is included in Appendix G.

Pregnant and breastfeeding women

As stated in the 2003 edition of the dietary guidelines, pregnant and breastfeeding women have an increased water requirement because of expanding extracellular fluid space and the needs of the foetus and the amniotic fluid. The fluid need is therefore 750–1,000ml a day above basic needs.³⁶

Many authoritative bodies advise pregnant and breastfeeding women to limit their caffeine intake. For example the Australian Department of Health and Ageing and New Zealand Ministry of Health advise that intake be restricted to a maximum of 300 mg of caffeine per day (about three cups of coffee or six cups of tea).^{42,66}

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 (e.g. due to hot weather or fever). Breast milk supplies adequate water up to around 6 months of age, but cooled boiled water may need to be provided for formula-fed infants.³⁵

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.³⁴ These changes may be normal adaptations of the ageing process but the outcomes of dehydration in older people are serious and include cognitive impairment, functional decline, falls or stroke. Particular care must be taken to ensure adequate water intake in this group.

2.7 Practice guide for Guideline 2

Table 2.17: Considerations in advising people from specific groups to consume a wide variety of foods

Population group	Considerations
Pregnant and breastfeeding women	<ul style="list-style-type: none"> Quality nutritious dietary patterns are essential in meeting the nutrient requirements of mother and foetus within the mother's energy requirements Pregnant women are at higher risk of foodborne illness. Foods associated with a risk of <i>Listeria</i> bacteria should be avoided While bread in Australia contains iodine and folate, supplementary folate is recommended preconception and in the first trimester and iodine should be supplemented preconception and throughout pregnancy and breastfeeding During pregnancy, care needs to be taken with consumption of some fish species due to the high mercury content Nuts need only be avoided if the woman herself has an allergy to them Fluid need is 750–1,000ml a day above basic needs A high-fibre diet and drinking sufficient water is effective in treating constipation
Infants (less than 12 months)	<ul style="list-style-type: none"> Exclusive breastfeeding is recommended for around the first 6 months, and should continue for 12 months and beyond for as long as the mother and child desire Breast milk supplies adequate water up to around 6 months of age, but cooled boiled water may need to be provided for formula-fed infants from birth A wide variety of solid foods should be introduced from around 6 months, with first foods being iron rich (e.g. iron-fortified cereal, meat and alternatives) Texture of solid foods should be appropriate to the infant's development Some foods may need to be introduced many times before they are accepted Hard pieces of food(e.g. some raw vegetables/fruit, whole nuts) should be avoided. Nut butters or pastes do not increase the risk of allergies and can be introduced from 6 months Breast milk or infant formula should be the main drinks in the first 12 months, however cow's milk may be served in small quantities as custards, with cereals, or as yoghurt between 6 and 12 months
Children and adolescents	<ul style="list-style-type: none"> A wide variety of nutritious foods is needed to support normal growth and development Parents and carers can support quality optimal dietary patterns by modelling behaviours and purchasing and preparing nutritious foods Reduced fat milk, yoghurt and cheese products are recommended for children 2 years and older Dietary restrictions are not generally suitable for growing children and adolescents and suspected food intolerance and allergy should be confirmed by a medical practitioner Adolescents may be vulnerable to disordered eating

Older people	<ul style="list-style-type: none"> • Reduced mobility, isolation, poor dentition and poverty may reduce access to nutritious foods • Particular care is required to ensure adequate water intake as the outcomes of dehydration are serious • People with chronic health issues and the frail elderly often have dietary requirements that are different to those of healthy, free-living older people
People living in remote areas and people in lower socioeconomic groups	<ul style="list-style-type: none"> • Healthy food habits may be difficult to afford • In urban areas there may be less access to supermarket foods and greater access to fast foods • In rural and remote areas a wide variety of fresh foods may not be locally available or may be expensive. Available traditional foods can be a nutritious alternative • Seasonal, frozen and canned fruit and vegetables, dried milk powder and grains and tinned fish can be nutritious, accessible options
People with vegetarian or vegan dietary patterns	<ul style="list-style-type: none"> • A variety of plant foods should be chosen to ensure adequate intake of iron and zinc • Vitamin B₁₂ is only available from animal food sources and supplementation may be needed

Where to next?

Both the quality and quantity of foods and drinks consumed can have a significant impact on health and wellbeing. The following chapter discusses how the overconsumption of some foods, such as those containing saturated fat, added salt, added sugars and alcohol, is associated with increased risk of some health conditions.

GUIDELINE 3

Limit intake of foods containing saturated fat, added salt, added sugars and alcohol

Guideline 3

Limit intake of foods containing saturated fat, added salt, added sugars and alcohol.

- a. Limit intake of foods high in saturated fat such as many biscuits, cakes, pastries, pies, processed meats, commercial burgers, pizza, fried foods, potato chips, crisps and other savoury snacks.
 - Replace high fat foods which contain predominantly saturated fats such as butter, cream, cooking margarine, coconut and palm oil with foods which contain predominantly polyunsaturated and monounsaturated fats such as oils, spreads, nut butters/pastes and avocado.
 - Low fat diets are not suitable for children under the age of 2 years.
- 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 in cooking or at the table.
- c. Limit intake of foods and drinks containing added sugars such as confectionary, sugar-sweetened soft drinks and cordials, fruit drinks, vitamin waters, energy and sports drinks.
- d. If you choose to drink alcohol, limit intake. For women who are pregnant, planning a pregnancy or breastfeeding, not drinking alcohol is the safest option.

Summary

- This Guideline emphasises the importance of limiting intake of foods and drinks high in saturated fat, added salt, added sugars and alcohol, based on evidence that these foods are associated with increased risk of obesity and/or chronic diseases, including cardiovascular disease, type 2 diabetes and/or some cancers. There is limited capacity for including energy-dense discretionary foods in nutritious dietary patterns within the energy requirements of many Australians.
- The link between dietary saturated fat, serum cholesterol and cardiovascular disease is well established. Replacing dietary saturated 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, which is prudent to consider in a total dietary pattern that seeks to control overall energy intake. Low fat diets are not suitable for children under the age of 2 years. Reduced fat milk may be used from the age of 2 years.
- Reducing sodium intake decreases blood pressure in both normotensive and hypertensive adults, and the evidence has strengthened that reducing sodium intake may decrease risk of mortality, stroke and heart disease in people with hypertension. Salt in processed foods is 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 an 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.
- Discretionary foods should only be consumed sometimes and in small amounts. While discretionary foods can contribute to the overall enjoyment of eating, often as part of social activities and family or cultural celebrations, if their intake is not reduced, most Australians need to greatly increase physical activity to 'burn up' the additional energy (kilojoules) from discretionary foods to help achieve and maintain a healthy weight (see Guideline 1).

This chapter provides information on why consumption of these food types should be limited. It includes practical considerations for meeting the guideline recommendation, for example, by choosing a variety of nutritious foods (Guideline 2), using only small amounts of unsaturated fats, spreads and oils, and avoiding or limiting discretionary foods and drinks.

3.1 Limit intake of foods high in saturated fat

3.1.1 Setting the scene

Continuing research into diet and cardiovascular disease emphasises reducing saturated fat in the diet, which means limiting intake of foods with high saturated fat content. Fat is a nutrient in food but the term 'fats' has also been applied to whole foods (e.g. butter, margarine and oils). Foods known as fats can also be ingredients in other foods (e.g. cakes and biscuits) or added as a culinary adjunct (e.g. oil in cooking or dressings). As a nutrient, fat has high energy value (fat delivers about 37 kJ/g, compared to around 17 kJ/g for carbohydrate and protein).

The evidence indicates that replacing dietary saturated fat with monounsaturated and polyunsaturated fats is associated with improved blood lipid profiles and reduced risk of cardiovascular disease. Replacing the type of fatty acids in fats requires a total diet approach and is not always possible with all foods. Both the amount and type of fat need to be carefully considered as all types of fat provide kilojoules and the proportion of total fat in a diet influences energy intake, which may have an impact on weight management (see Chapter 1).

As people choose to eat foods, rather than food components or nutrients, the focus of this guideline recommendation is on foods containing fats, not fatty acids *per se*. Information on particular types of fatty acids is included in the NRV Document.⁸

Most fats in foods are in the form of triglycerides, which are made up of a unit of glycerol combined with three fatty acids that 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.⁸

Over time, the understanding of physiological effects and pathways have been gradually refined – for example, with the discovery of low-density and high-density cholesterol and more recently, the discovery of sub-fractions of these.⁶⁶⁴ Additional physiological characteristics are being studied as possible markers of cardiovascular risk (e.g. vascular reactivity and carotid intima medial thickness). It is also apparent that not all fatty acids within each group have the same effects – for example, stearic acid might not have the same effects as some other saturated fatty acids (SFAs).⁶⁶⁴ However, as a general message to the public, limiting total dietary saturated fat remains the best guide.

Fatty acids include SFAs, monounsaturated fatty acids (MUFAs) or polyunsaturated fatty acids (PUFAs). 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 fatty acids differing chemical properties that cause different biological effects. MUFAs and PUFAs with one or more double bonds in the trans configuration are known as trans-fatty acids (TFAs).⁶⁶⁵ Omega fatty acids (omega-3 and omega-6 PUFAs) are sub-classes of PUFAs. The essential fatty acids (which humans do not make) are considered to be linoleic acid (omega-6 PUFA) and alpha-linolenic acid (omega-3 PUFA). The most common omega-9 fatty acid is oleic acid, a MUFA.

Staple foods with a relatively higher fat content, such as nuts, seeds, some grains (e.g. oats), dairy foods and meats, have various combinations of fatty acids. Fish is the predominant source of two omega-3 LCPUFAs, eicosapentaenoic acid (EPA) (20:5) and docosahexaenoic acid (DHA) (22:6). Grass-fed meat, kangaroo and offal contain small quantities of these omega-3 LCPUFAs as well as another, docosapentaenoic acid (DPA) (22:5).

The NRV Document recommends that:⁸

- total fat account for 20–35% of energy (kilojoule) intake
- total SFAs and trans fats comprise no more than 10% of energy intake
- 4–10% of energy comes from linoleic acid (omega-6 PUFA) and 0.4–1% from alpha-linolenic acid (omega-3 PUFA).

Given that total energy intake reflects the sum of the energy value of all foods consumed, food choices and culinary practices can have a substantial impact on whether these fatty acid targets are met.⁹

3.1.2 The evidence for 'limit intake of foods high in saturated fat'

The early work on dietary fats and heart disease focused on the type of fat in the overall diet, with the fat being contributed by a wide range of foods. The evidence that replacing saturated fats with polyunsaturated fats affects serum cholesterol levels has been accumulating for the last 60 years,^{36,666} and the relationship has been confirmed in a recent review of human intervention trials and other studies.^{94,667-670}

The intake of trans fats is low in Australia and consequently there is no specific recommendation to limit their intake compared to current intake.⁶⁷¹ However, it is important to ensure that intake remains at its current low level.

The evidence for associations between foods high in fat and the development of type 2 diabetes, hypertension, cancer and poor mental health has been reviewed in detail.

The Guidelines recommend some caution in choosing foods high in fat (in particular saturated fat) because of the implications for weight management and cardiovascular disease risk. Fat-rich foods are energy (kilojoule) dense, heightening the risk of excess energy intake⁶⁷² as shown by dietary modelling.⁹ Additionally, there is ample evidence of the relationship between dietary patterns and disease risk at the population level.⁹⁴ Fat content is an important component of dietary quality and it may be that the evidence for limiting saturated fat in the diet is best considered from a whole-of-diet perspective, with additional reference to overall nutritional quality.

Scientific evidence on the effect of dietary fat on health comes 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
- a diet enriched with specific fatty acids (e.g. omega-3 fatty acids)
- oils and fats (e.g. olive oil, spreads) in a defined dietary pattern.

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 effect of dietary fat on disease risk is reflected in several recent reviews on the topic.^{94,667-670} It is important to note that inconsistency in results affects the strength of the evidence statements below.

Table 3.1: Evidence statements for 'limit intake of foods high in saturated fat'

Established evidence

Saturated fat is the strongest dietary determinant of plasma LDL concentration.

Replacing saturated fat with polyunsaturated and monounsaturated fats is associated with improved blood lipids related to cardiovascular disease.

Evidence statement	Grade
Higher consumption of omega-3 LCPUFA fat (intake amount not specified) is associated with reduced risk of dementia.	C

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

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,^{664,673-681} only one review⁶⁶⁴ 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 SFAs 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 SFAs with carbohydrate (as is the case in some low fat diets) depends on the effects on body weight.⁶⁶⁴

The Joint FAO/WHO Expert consultation on fats and fatty acids in human nutrition 2010⁶⁸² reported convincing evidence that replacing SFAs with PUFAs decreases the risk of coronary heart disease. The *Dietary Guidelines for Americans, 2010* drew similar conclusions, finding:¹⁹⁸

- strong evidence that dietary SFAs are positively associated with increased serum cholesterol and LDL cholesterol, with increased risk of cardiovascular disease
 - an association between replacing dietary SFAs or trans fats with PUFAs with improved blood lipid profiles and reductions in levels of numerous markers of inflammation
 - strong evidence that replacing dietary SFAs with MUFAs and/or PUFAs is associated with improved blood lipids related to cardiovascular disease
 - moderate evidence that 250 mg of omega-3 LCPUFAs delivered from two servings of seafood per week is associated with reduced cardiac mortality from coronary heart disease and reduced risk of sudden death from cardiovascular disease.
- **Hypertension:** The evidence suggests that consumption of fat, irrespective of amount or type, is not associated with hypertension in the short term (Grade C; Evidence Report, Section 12.4).⁶⁸³⁻⁶⁹¹
 - **Type 2 diabetes:** While overweight and obesity increase the risk of type 2 diabetes,⁸⁸ recent studies of 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 consuming omega-3 LCPUFAs (0.4 –6 g/day) and diets of varying fat content are not associated with fasting plasma glucose or insulin concentrations (Grade C; Evidence Report, Section 12.3).^{674,681,691-701}
- In the longer term, the review conducted for the *Dietary Guidelines for Americans 2010* found strong evidence that dietary SFAs were positively associated with increased markers of insulin resistance and increased risk of type 2 diabetes. In addition, it found that decreasing dietary SFAs and replacing them with PUFAs or MUFAs 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.¹⁹⁸
- **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 into the effect of dietary fat alone on weight gain⁶⁷² (see Chapter 1).

Cancer

- **All-cause:** The evidence suggests there is no association between consumption of omega-3 LCPUFAs with total all-cause cancer incidence or mortality (Grade C; Evidence Report, Section 12.5).^{677,702}
- **Other cancers:** Evidence of an association between total fat consumption across a range of intakes and breast or endometrial cancer is inconclusive (Evidence Report, Sections 12.6 and 12.7). However, the WCRF report states there is suggestive evidence that total fat consumption increases the risk of post-menopausal breast cancer (see Appendix F).⁴³

Other conditions

- **Dementia:** The evidence suggests that higher consumption of omega-3 LCPUFAs is associated with a reduced risk of dementia (Grade C; Evidence Report, Section 12.8).^{517,529,703-706}

3.1.3 How limiting intake of foods high in saturated fat may improve health outcomes

Fat is an energy-dense macronutrient, so consuming too much fat may lead to excess energy (kilojoule) intake and weight gain;⁶⁷² however an individual's genetic makeup, level of physical activity and other dietary factors also play a part.⁷⁰⁷ Fat cells secrete compounds that influence appetite, inflammation and possibly also cancer development.⁷⁰⁸⁻⁷¹⁰ 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 coexist with insulin resistance, a phenomenon often referred to as the metabolic syndrome.⁷¹¹

Fatty acids do not only contribute to body fat, different fatty acids influence disease risk factors. Dietary SFAs and TFAs have been associated with raised plasma LDL cholesterol, and dietary TFAs have been associated with reduced plasma high-density lipoprotein (HDL) cholesterol.²³ 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.⁷¹² Several large cohort studies have also demonstrated that reduced HDL cholesterol is a significant risk factor for coronary heart disease.^{713,714}

PUFA intake appears to reduce coronary heart disease risk,⁹⁴ but the balance between omega-6 and omega-3 PUFA may be important.⁷¹⁵ In addition to their effects on lipid metabolism, PUFA may exert a positive influence on insulin action, appetite regulation, inflammatory responses, and muscle function.⁷¹⁶ Among the omega-3 LCPUFAs, EPA is the precursor of the 3 series of prostaglandins and the 5 series of leukotrienes.⁷¹⁷ This suggests anti-inflammatory and anticoagulant 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.⁷¹⁸ Further information on fish sources of omega-3 LCPUFAs can be found in publications from the Commonwealth Scientific and Industrial Research Organisation (CSIRO).^{435,436}

3.1.4 Practical considerations: limit intake of foods high in saturated fat

Fats are found in many foods in the five food groups especially nuts and seeds, legumes/beans, avocado, oats, fish, meat (lean meats), poultry, eggs, milk and cheese. Omega-3 LCPUFA intake can be increased by eating according to the Foundation Diets and Total Diets. When eating meals outside the home, lower-fat menu choices are preferable.

The most recent dietary data available in Australia show adults consume 73–101 g/day of fat, with higher intakes among men and younger age groups.⁴⁵ Children consume 51–87 g/day of fat, with intake being higher in older age groups.¹² 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.¹⁵² Intake of total fat, in particular saturated fat, remains higher than recommended.¹⁵² However, the mean total population trans fat intake for Australia is estimated to be 0.5% of total dietary energy, which is below the WHO population goal of less than 1% of total dietary energy from TFAs.⁶⁷¹

The Guidelines are realistic and practical, allowing a small amount of unsaturated oils and spreads to reflect culinary behaviour, while ensuring that the energy these foods provide are within the total energy constraints of the diet. Dietary fat included in the Foundation Diets comes mainly from fish, lean meats, poultry and milk, yoghurt and cheese products, with a small allowance of unsaturated oils/fats/spreads. 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, such as additional nuts and seeds, unsaturated spreads and oils, and/or discretionary foods. However, where possible, the best choices are foods where unsaturated fats exist in greater quantities than saturated fats. As well, people who are shorter, smaller or sedentary may have little or no scope within their usual dietary patterns for any discretionary foods and drinks. The extra energy (kilojoules) provided by these foods and drinks is an additional reason to limit them.⁹

Foods containing predominantly saturated fat such as butter, cream, cooking margarine, coconut and palm oil, many biscuits, cakes, pastries, pies, desserts, confectionery, processed meats and some commercial burgers, pizza, fried foods, potato chips, crisps and other savoury snacks should be limited. Increasing the proportion of unsaturated fats in the diet can be achieved by choosing vegetables, fruit, lean meats and low fat milk, yoghurt and cheese products, nuts and seeds and using small amounts of unsaturated spreads and oils. Sunflower and safflower seed, soybean, cottonseed, sesame, corn and grape seed and the spreads and oils made from them as well as walnuts contain predominantly PUFAs. Canola seeds, nuts, peanuts, rice bran, avocados and olives and the oils and spreads made from them, contain mainly MUFAs.

The foods included in the *Australian Guide to Healthy Eating* generally contain low levels of saturated fat and include PUFAs and MUFAs in proportions that appear to be protective against heart disease risk and support the maintenance of cognitive function.⁸

The companion resources provide more detailed information on the recommended number of serves and serve sizes of unsaturated spreads and oils required for different population groups (see Tables 3.2 and 3.3). Discussion of dietary patterns and the environment is included in Appendix G.

Table 3.2: Recommended allowance for number of serves of unsaturated spreads and oils per day

	Age (years)	Number of serves*
Boys	2–3	½
	4–8	1
	9–11	1
	12–13	1 ½
	14–18	2
Men	19–50	4
	51–70	4
	70+	2
Girls	2–3	½
	4–8	1
	9–11	1
	12–13	1 ½
	14–18	2
	Pregnant (up to 18 years)	2
	Breastfeeding (up to 18 years)	2
Women	19–50	2
	51–70	2
	70+	2
	Pregnant (19–50 years)	2
	Breastfeeding (19–50 years)	2

Note: *Additional amounts of the five food groups or unsaturated spreads and oils or discretionary choices are needed only by people who are taller or more active to meet additional energy requirements.

Source: *Food Modelling System*.⁹

Table 3.3: Standard serve size equivalents of unsaturated spreads and oils

Food group	Serve sizes (250kJ)
Unsaturated spreads and oils	10g polyunsaturated spread
	10g monounsaturated spread
	7g monounsaturated or polyunsaturated oil, for example olive, canola or sunflower oil
	10g tree nuts or peanuts or nut pastes/butters

Source: *Food Modelling System*.⁹

Infants

For infants under the age of around 6 months, breast milk provides an ideal amount and type of fat. The guideline recommendation on fat intake for the adult population does not apply to young children, particularly those aged less than 2 years. The amounts and types of fat required for infants are related to physiological and health outcomes.⁸ Even a small energy deficit during this period of rapid development may affect growth.⁸ Neurological development is particularly rapid in the first 2 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 omega-3 LCPUFAs needed by developing nervous tissue, adversely affecting growth and development.

Children and adolescents

Reduced fat milk is recommended from the age of 2 years, when milk plays a less dominant role in the diet. A high-fat diet is likely to be energy-dense, contributing to excess energy intake and the development of obesity. 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 2 years of age.^{35,718} Introducing healthy eating patterns in early childhood influences dietary patterns in later childhood.^{720,721}

Older people

Low fat diets are not suitable for convalescent older people and frail elderly 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 lipid levels can reduce risk of ischaemic heart disease regardless of age.⁷²²

Aboriginal and Torres Strait Islander peoples

Limiting intake of excess energy from any source, including foods high in fat, is particularly important given the higher prevalence of obesity in Aboriginal and Torres Strait Islander groups compared to non-Indigenous Australians.^{28,38} Limiting saturated fat and increasing unsaturated fats are important given the high prevalence of coronary heart disease, and decreased saturated fat intake may also improve insulin sensitivity.

People from 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 US Surgeon General's report released in 1979.⁷²³ Initial advice to the public was framed to reduce consumption of discretionary salt, such as salt added at the table or during cooking. However, sodium occurs naturally in food, and sodium-containing additives are also added to manufactured and processed foods. It is now recognised that processed foods are the major source of sodium in Western diets. In these Guidelines, 'salt' refers to sodium chloride and is never used as a synonym for the total amount of sodium in foods.

Since the 2003 edition of the dietary guidelines, the evidence for a relationship between reducing sodium intake and reducing blood pressure has strengthened, particularly in people classified as having normal blood pressure. In addition, there is now some evidence indicating that reducing sodium may result in a reduction in 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 'limit intake of foods and drinks containing added salt'

The evidence for the guideline recommendation 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).

Table 3.4: Evidence statements for 'limit intake of foods and drinks containing added salt'

Evidence statement	Grade
Decreasing consumption of sodium decreases blood pressure in normotensive adults; a reduction of 1,800 mg reduces systolic blood pressure by about 2 mmHg and diastolic blood pressure by about 1 mmHg.	A
Decreasing consumption of sodium decreases blood pressure in hypertensive adults; a reduction of 1,800 mg reduces systolic blood pressure by about 5 mmHg and diastolic blood pressure by about 3 mmHg.	A
Consuming a diet low in sodium reduces blood pressure in children up to 18 years of age.	B
Reducing sodium intake by about 1,000 mg/day is associated with reduced risk of cardiovascular events.	C

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

Cardiovascular disease

- **Blood pressure:** 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.⁷²⁴⁻⁷²⁷
 - in adults with normal blood pressure, a reduction of 1,800 mg per day reduces systolic blood pressure by about 2 mmHg and diastolic blood pressure by about 1 mmHg (Grade A; Evidence Report, Section 13.1)
 - in adults with hypertension, a reduction of 1,800 mg per day reduces systolic blood pressure by about 5 mmHg and diastolic blood pressure by about 3 mmHg (Grade A; Evidence Report, Section 13.1)⁷²⁴⁻⁷³⁹
 - 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; Evidence Report, Section 13.2).^{726,739,740}

The *Dietary Guidelines for Americans, 2010* drew 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'.¹⁹⁸ 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 related to changes in cardiovascular morbidity and mortality. Past studies have mainly included subjects with hypertension. The evidence suggests that reducing sodium intake by about 1,000 mg/day is associated with reduced risk of cardiovascular events (Grade C; Evidence Report, Section 13.4).^{725,741-746} The results are consistent with drug trials showing that reductions in hypertension also lead to decreases in adverse cardiovascular outcomes.^{747,748}

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.⁷⁴⁹ This association was described as convincing by the WCRF report (see Appendix F).⁴³ The WCRF report 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 (see Appendix F)⁴³ but it is unclear whether the factor responsible is the salt, other components such as nitrites, or a combination of these factors.

Bone health

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

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.⁷

Future data may lead to a refined understanding of the underlying mechanisms. For example, the ratio of sodium to potassium may influence blood pressure more strongly than the amount of sodium alone⁷⁴²⁻⁷⁴⁴ and reducing sodium intake may be particularly important for overweight people with certain conditions.⁷⁴⁵ 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 intake in the US is lower than desirable.¹⁹³

3.2.4 Practical considerations: Limit 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 recommendation to 'Eat less salt'.⁷⁵⁰ The second edition revised this to 'Choose low salt foods and use salt sparingly'.⁷⁵¹ The third edition advised Australians to 'Choose foods low in salt'.³⁶ In these Guidelines, it is recognised that any reduction in dietary sodium will reduce blood pressure in those with normal levels of blood pressure, reduce risk of developing hypertension and also reduce blood pressure in those with elevated blood pressure.

The NRV Document recommends 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.⁸ Among those with high sodium intake, reduction in sodium intake would reduce blood pressure even if specific targets were 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 cheeses have higher levels of 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 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' if the food contains no more than 120mg of sodium per 100g.⁷⁵² Not all foods that meet this criterion carry a 'low sodium' claim.

Because salt is 40% sodium, avoiding discretionary salt in cooking or at the dinner table will also reduce sodium intake (the set UL of 2,300mg/day is approximately equal to 6g of table salt). In the 1995 National Nutrition Survey, 62% of the population aged 2 years and older reported always or usually adding salt at the table or during cooking.⁴⁵ Since 1995, Asian-style cooking has become increasingly popular in Australia and many Asian-style sauces such as soy, oyster and fish sauces are particularly 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 that replace some sodium chloride with potassium chloride can provide a salt taste with lower levels of sodium. Herbs, spices, garlic, lemon juice and vinegars 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).⁷⁵³ Milk and dairy foods are an important source of iodine in Australia.

Pregnant and breastfeeding women

Advice to limit the intake of sodium for the general population is also applicable to pregnant and breastfeeding women.⁷⁵⁴

Infants

FSANZ 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.⁷⁵⁵ 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 the infant's sodium intake. This means preparing homemade 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.

Children

For children with average energy needs, the dietary patterns in the Food Modelling System⁹ contain up to 50% less sodium than the average sodium intakes reported in the 2007 Australian National Children's Nutrition and Physical Activity Survey.¹²

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 hypertension, clinicians need to weigh up the benefit of adding 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 hypertension, salt intake can be determined by personal preference 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 – examples include fructose, glucose, lactose and 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 that is added to foods and is termed 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. Dietary modelling illustrates that nutrient density may also be compromised by a high intake of added sugars and the Total Diet does not allow for excess amounts of foods or drinks with added sugars.⁹ At any given level of energy intake, as the proportion of added sugars in the diet increases, the nutrient density will fall.⁷⁵⁶ This was quantified in a recent analysis of National Health and Nutrition Examination Survey (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.⁷⁵⁷

Sugars are a factor in dental caries and diets high in added sugars are also associated with some adverse health outcomes. Sugar-sweetened drinks (sugar-sweetened soft drinks and cordials, fruit drinks, vitamin waters, energy and sports drinks) are the largest source of sugars in the Australian diet, with consumption highest in adolescents and children.⁴⁵ New evidence emphasises the relevance of sugar-sweetened drinks to the development of excess weight (see Chapter 1).

Dental caries are a significant public health problem in Australia. In 2007–08, \$6.1 billion was spent on total dental services in Australia, representing 6.2% of all health expenditure.⁷⁵⁸ In severe cases, dental caries can cause loss of teeth and pain that may reduce dietary intake and compromise nutritional status.

Fructose and/or high fructose corn syrups are commonly used as a sweetener in the US. It should be noted that these are not commonly used in the Australian food supply.

3.3.2 The evidence for 'limit 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.

Table 3.5: Evidence statements for 'limit intake of foods and drinks containing added sugars'

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

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

Cardiovascular disease, type 2 diabetes and excess weight

- **Cardiovascular disease:** There is no new evidence that sugars play a causal role in the development or moderation of cardiovascular risk factors. Early studies suggested that reducing dietary sucrose could lower elevated triglyceride levels, but it is likely that the effects were the result of a reduction in energy intake and body weight.⁷⁵⁹
- **Type 2 diabetes:** Insufficient studies were identified to develop an evidence statement for intake of sugars and type 2 diabetes.^{760,761} However, more recent studies indicate that sugar-sweetened drinks may increase the risk of developing type 2 diabetes.⁷⁶² A recent meta-analysis also supports an increased risk of type 2 diabetes and the metabolic syndrome from consumption of sugar-sweetened drinks.⁷⁶³
- **Excess weight:** Recent studies show evidence of a probable association between sugar-sweetened drinks and weight gain in adults and children (Grade B; Evidence Report, Section 15.1).¹⁸⁵⁻¹⁹⁶ A more recent longitudinal study also strengthens the evidence associating sugar-sweetened drinks with weight gain.⁷⁶⁴
- There is insufficient consistent evidence available to form an evidence statement about fruit juices and weight gain. Some studies found no association in children,^{193,217,218,585} while two studies in children^{186,765} and one in adults⁷⁶⁶ did find an association. Children drinking less fruit juice²⁰⁶ and those consuming more fruit¹⁹⁴ had lower BMI Z-scores, and children at risk of becoming overweight had a higher risk of gaining fat if they consumed fruit juice.¹⁸⁶
- 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 fewer sugar-sweetened foods had less increase in skinfold fat and waist circumference over a 5-year period (Evidence Report, Section 14.3).²⁰⁶

Cancer

There is evidence suggesting that consumption of sucrose is not associated with risk of cancer (Grade C; Evidence Report, Section 14.1).⁷⁶⁷⁻⁷⁷¹ The WCRF report 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 (see Appendix F).⁴³ 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 (see Appendix F).⁴³

Other conditions

- **Dental caries:** The relationship between sucrose and dental caries was first documented more than a century ago⁷⁷² and has been confirmed in numerous studies since then.⁷⁷³ 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; Evidence Report, Section 14.2).^{774,778} The evidence also suggests that dental caries are related to sugar-sweetened drinks (Grade C; Evidence Report, Section 15.4).^{774,776}
- Caries are associated with national per capita yearly sucrose consumption, with very few caries in children consuming 10kg/year (about 30g/day) or less. A steep increase may occur from 15kg/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 added sugars are most damaging to teeth.⁷⁷⁹
- **Bone strength:** Evidence suggests an association between consumption of soft drinks and increased risk of reduced bone strength (Grade C; Evidence Report, Section 15.3).⁶⁵⁰⁻⁶⁵³ 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.⁶⁵³ 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.⁶⁵⁰ A systematic review reported an inverse relationship between soft drink consumption and milk intake.¹⁹⁵
- 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 an inverse relationship with milk consumption.
- **Attention deficit hyperactivity disorder (ADHD):** There is no evidence that added sugars are involved in the aetiology of ADHD.⁷⁸⁰

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.⁷⁸¹ Dietary sugars other than sucrose (e.g. 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.⁷⁸² 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 preventing 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.⁶⁵⁷ This applies equally to sugar-sweetened or diet soft drinks, since their acidity is comparable.

Excess weight: Many foods containing added sugars (e.g. 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 of 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.⁷⁸³ Newer evidence supports this failure to compensate by reducing energy intake from other foods or drinks.⁷⁸² Sugar-sweetened drinks therefore add to total energy intake.⁷⁸⁴

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

In light of the current prevalence of overweight and obesity (see Chapter 1), the dietary guidelines of many countries recommend significant reductions in foods that contribute to energy while providing few, if any, nutrients.¹⁹⁸ Many foods and drinks containing some fats, added sugars (simple carbohydrates) and some starches (complex carbohydrates), 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.¹²

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.²² Recent data from the US suggests a level of 5–10% of energy from added sugars may be appropriate.⁷⁵⁷ This is much less than current Australian consumption, reinforcing the continued need for this Guideline.

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.⁷⁸⁵ Infants do not need added sugars and FSANZ stipulates that ready-prepared infant foods with more than 4g of added sugars per 100g must be labelled as 'sweetened'.⁷⁵⁵ For further information, see the Infant Feeding Guidelines.³⁵¹

Children and adolescents

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, fruit drinks and energy drinks. Energy drinks may also be high in caffeine and are not suitable for children. Sweetened flavoured milk provides nutrients but can be energy dense; plain milk is preferable.

Older people

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

Aboriginal and Torres Strait Islander peoples

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.³⁵² In remote communities where apparent consumption was measured, added 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 Aboriginal and Torres Strait Islander 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.⁷⁸⁶ Aboriginal children and children from lower socioeconomic groups have not had the improvement in dental health seen in other children.

3.4 Limit intake of alcohol

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. There is some evidence that people who drink small quantities of alcohol may have better health outcomes than those who do not drink,⁷⁸⁷ but such findings have been challenged.^{788,789} Heavy drinking has no health benefits and studies consistently report that abstainers have better health outcomes than heavy drinkers.

In the Australian population, alcohol is responsible for 3.3% of the total disease burden and prevents 1% of the total disease burden. This equates to a net effect of 2.3%, equivalent to 61,091 disability-adjusted life years (DALYs) and 0.8% (1,084) of all deaths.¹⁰ Among Aboriginal and Torres Strait Islander peoples, alcohol is responsible for a net 5.4% of the total disease burden and 6.7% of all deaths.⁷⁹⁰ Alcohol is second only to tobacco as a preventable cause of drug-related death and hospitalisation.⁷⁹¹

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.^{792,793}

The NHMRC 2009 *Australian guidelines to reduce health risks from drinking alcohol* (NHMRC Alcohol Guidelines)⁷⁹⁴ provide guidance for Australians on reducing their risk of harm from drinking alcohol.

The NHMRC Alcohol Guidelines are as follows.

- Guideline 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.
- Guideline 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.
- Guideline 3** For children and young people under 18 years of age, not drinking alcohol is the safest option.
 - 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.
- Guideline 4** Maternal alcohol consumption can harm the developing foetus or breastfeeding baby.
 - 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).⁷⁹⁴ The alcohol concentration of drinks is printed on the label in terms of percentage by volume. However in social situations serve sizes are greater than standard drinks – for example a typical glass of wine in Australia is 170ml⁷⁹⁵ which, depending on the alcohol content of the wine, is more than one 'standard' drink and likely to be closer 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 in Appendix K. 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 'limit intake of alcohol'

The evidence underpinning the guideline recommendation utilises the NHMRC Alcohol Guidelines and additional evidence sourced from the Evidence Report.

Table 3.6: Evidence statements for 'limit intake of alcohol'

Evidence statement	Grade
Consuming alcohol regularly at an intake of one standard drink per day for women and one and a half to two per day for men is associated with a reduced risk of cardiovascular disease morbidity and mortality.	B
Consuming alcohol regularly at an intake of one standard drink per day for women and one and a half to two per day for men increases HDL cholesterol.	B
Consumption of alcohol, even at low levels (10–15g/day), 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), is associated with an increased risk of colon cancer and rectal cancer.	C
Consumption of alcohol, even at low levels (10g/day), 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 one standard drink per day for women and one and a half to two per day for men, with a maximum intake of four standard drinks per day, is associated with reduced risk of dementia in older adults.	C

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

Cardiovascular disease and type 2 diabetes

- **Cardiovascular disease:** There is evidence of a probable association between consumption of one standard drink per day for women and one and a half to two per day for men with a reduced risk of cardiovascular disease morbidity and mortality (Grade B; Evidence Report, Section 16.1).⁷⁹⁶⁻⁷⁹⁸ 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 26 g/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.⁷⁹⁹ However the evidence is not yet conclusive, particularly for Asian populations.^{787,788,800,801}
- There is evidence of a probable association between consumption of one standard drink per day for women and one and a half to two standard drinks per day for men with an increase in HDL cholesterol (Grade B; Evidence Report, Section 16.1).⁸⁰²⁻⁸⁰⁹ Recent reviews of population-based studies^{799,810,811} support these findings.
- Although an insufficient number of studies were identified to formulate an evidence statement, excess alcohol consumption has been found to increase the risk of hypertension.^{812,813} A more recent review supports these findings, suggesting a synergistic effect between alcohol, hypertension and cerebrovascular events.⁸¹⁴
- Canadian and Australian guidelines on the management of hypertension include moderation of alcohol intake as an important intervention.⁸¹⁵ Hypertension is a major risk factor for both ischaemic and haemorrhagic stroke.⁸¹⁶
- **Type 2 diabetes:** Evidence regarding an association between alcohol intake and risk of type 2 diabetes is inconclusive (Evidence Report, Section 16.2).

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.⁴³ The WCRF report concludes that the number of cancers for which alcohol is a known risk factor is increasing (see Appendix F).⁴³ 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; Evidence Report, Section 16.4).⁸¹⁷⁻⁸²³ The WCRF report found a convincing association between consumption of alcohol and risk of breast cancer (see Appendix F).⁴³
- **Oesophageal cancer:** There is evidence of a probable association between consumption of alcohol and an increased risk of cancer of the oesophagus (Grade B; Evidence Report, Section 16.6).^{43,817} The WCRF report found a convincing association between consumption of alcohol and risk of cancer of the oesophagus (see Appendix F).⁴³
- **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; Evidence Report, Section 16.5).^{43,817,824,825}
- **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; Evidence Report, Section 16.9).^{43,817} The WCRF report found a probable association between the consumption of alcohol and risk of liver cancer (see Appendix F).⁴³
- **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; Evidence Report, Section 16.7).^{43,817,826}
- **Non-Hodgkin lymphoma:** Evidence that the consumption of alcohol is associated with non-Hodgkin lymphoma is inconclusive (Evidence Report, Section 16.11).
- **Other cancers:** The evidence that consumption of alcohol is associated with renal, pancreatic and ovarian cancer is inconclusive (Evidence Report, Sections 16.8, 16.10 and 16.12).

Other conditions

- **Alcohol-related liver disease:** As discussed in the 2003 edition of the 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, alcoholic hepatitis).⁸²⁷ The same level of average consumption is related to a higher risk of liver cirrhosis in women than in men.⁸²⁸
- **Dementia:** The evidence suggests an association between the consumption of one standard drink per day for women and one and a half to two standard drinks 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; Evidence Report, Section 16.3).⁸²⁹⁻⁸³⁵
- **Mental health:** Alcohol use is associated with an increased risk of a number of mental health and social problems in young adults.⁸³⁶ 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.⁸³⁶⁻⁸⁴⁰
- **Nutrition-related conditions:** Alcohol consumption is linked to malnutrition, Wernicke-Korsakoff syndrome, folate deficiency, vitamin A depletion and pellagra.⁸⁴¹ 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 thiamine has contributed to a 40% reduction in the incidence of Wernicke-Korsakoff syndrome.^{842,843}

Other conditions associated with harmful levels of alcohol consumption include:⁷⁹⁴

- dependence and addiction
- endocrine conditions (e.g. hypercortisonism and sexual dysfunction)
- alcohol-related brain damage including alcoholic dementia
- gastritis and gastric ulcers
- aspiration pneumonia
- cardiomyopathy
- interactions with pharmaceuticals and illegal recreational drugs.

3.4.3 How limiting intake of alcohol may improve health outcomes

Alcohol begins to affect the brain within 5 minutes of consumption, with blood alcohol concentration peaking after 30–45 minutes. It takes approximately 1 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.^{§44-§46} Because the rate of metabolism is fixed, rapid consumption of multiple drinks results in a higher blood alcohol concentration.^{7§4}

Young adults who drink heavily tend to have smaller prefrontal cortices and white matter, structural abnormalities of white matter and reduced hippocampal volumes.^{§47-§48} These structural changes lead to a diminished ability to retrieve verbal and non-verbal material and poorer performance in attention-based tests.^{§39}

The loss of brain tissue that occurs in people with chronic alcoholism seems to occur independently of ~~Vernicke'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 HDL cholesterol and reduce accumulation of plaque in arteries.^{§10-§49} Alcohol can also have a mild anticoagulant effect.
- **Diabetes:** Alcohol affects the management of type 1 and type 2 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.^{§50, §51}
- **Dementia:** The suggested protective relationship between alcohol and dementia may relate to the effect of alcohol on blood lipids as one of the causal factors of dementia is microvascular changes within the brain.^{§52, §53}

3.4.4 Practical considerations: limit intake of 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.^{§54} 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.^{§54}

Nearly all alcohol is consumed as drinks, principally beers, wines, spirits and ciders. Alcoholic drinks contain few other nutrients except for the bioactive flavonoids found in wine (mainly 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. Some alcoholic drinks are mixed with additives including stimulants, sugars and other flavours.

The apparent average consumption of alcohol is estimated at 10.08 L per person over 15 years old per year,^{§55} and declines with age.^{§56} 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 even more energy. If alcohol is consumed in addition to the normal diet, leading to excess energy intake compared to requirements, weight will increase.

A full stomach reduces 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 exercising do not reduce blood alcohol content.^{7§4}

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 is the safest option.^{7§4}

There is limited evidence from human research on the effects of maternal alcohol consumption during lactation and infant development. In Australia, it is suggested that mothers who do consume alcohol are more likely to stop breastfeeding before six months compared to mothers who do not drink.^{§57, §58} Animal and observational

studies show that consuming two standard drinks or more per day during lactation is associated with deficits in infant psychomotor development and disrupted infant sleep-wake behavioural patterns.⁸⁵⁷ Although the extent is unknown, a baby's intake of alcohol from breast milk is not harmless.⁸⁵⁷ Alcohol levels in breast milk 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.⁷⁹⁴ For those who drink, expressing milk before consuming alcohol is the next best option.⁷⁹⁴

Children and adolescents

Alcohol use by younger people is associated with harmful effects on brain development. 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.⁷⁹⁴

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.⁸⁵⁹

Many older people take medications that may interact with alcohol. A combination of alcohol and medication increases the risk of falls and injury.^{860,862}

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'.⁷⁹⁴

Aboriginal and Torres Strait Islander peoples

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.⁸⁵⁴ In response to severe problems related to excess alcohol consumption in many Indigenous communities, including foetal alcohol syndrome^{863,864} 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 peoples should follow the alcohol guidelines described above.⁷⁹⁴

People from culturally and linguistically diverse groups

People from culturally and linguistically diverse groups are more likely than the general adult population in Australia to abstain from alcohol (43% compared to 15%).⁸⁵⁴ 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.

People with diabetes

As alcohol and hypoglycaemia have independent but additive effects on cognitive function and behaviour, it is recommended that people with type 1 or type 2 diabetes abstain from alcohol if they plan to drive.⁸⁶⁵ Alcohol worsens medical conditions associated with diabetes, such as liver disease, hypertension and advanced neuropathy.^{850,866} People with type 1 or type 2 diabetes may need to take special precautions when drinking and should discuss alcohol use with a health professional.

Interaction of alcohol with caffeine and other stimulants

A new category of alcoholic drinks is now available in Australia that have added caffeine and/or other stimulants. There is concern that consuming stimulants such as 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.⁸⁶⁷⁻⁸⁷⁰

In the absence of any long-term research to quantify safe levels of concurrent consumption of alcohol and added stimulants, this combination should be used with caution.

3.5 Practice guide for Guideline 3

Table 3.7: Considerations in advising people from specific groups to limit intake of foods containing saturated fat, added salt, added sugars and alcohol

Population group	Considerations
Pregnant and breastfeeding women	<ul style="list-style-type: none"> • Additional energy requirements should be met through extra foods from the five food groups rather than energy-dense discretionary foods • Not drinking alcohol while pregnant is the safest option – there is no lower limit that can be guaranteed to be completely safe for the foetus • Abstaining from alcohol is the safest option for women who are breastfeeding – for those who drink, expressing milk before consuming alcohol is the next best option
Infants	<ul style="list-style-type: none"> • 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 • When solid foods are introduced, salt and sugar should not be added to prepared food and the salt and sugar content of ready made foods should be checked
Children and adolescents	<ul style="list-style-type: none"> • Introducing healthy eating patterns in early childhood influences dietary patterns in later years, however reduced fat milk, yoghurt and cheese products are not recommended for children under 2 years • Water and plain milk are recommended drinks – consumption of soft drink, 'sports drinks', 'vitamin waters', cordials, fruit drinks and energy drinks should be limited • Not drinking alcohol is especially important in children under 15 years of age, who are at the greatest risk of harm from drinking • For adolescents aged 15–17 years, the safest option is to delay drinking for as long as possible
Older people	<ul style="list-style-type: none"> • Older people with complex health issues or frail elderly people often have dietary requirements that are different to those of healthy, free-living older people where maintaining energy intake is a priority and discretionary foods may assist in preventing malnutrition • Older people are more susceptible than others to the toxic effects of alcohol and for some older adults, drinking alcohol increases the risk of falls and injuries • Older people are advised to consult their health professionals about the most appropriate level of drinking for their health

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.

GUIDELINE 4

Encourage, support and promote breastfeeding

Guideline 4

Encourage, support and promote breastfeeding.

Summary

- The World Health Organization states that 'breastfeeding is an unequalled way of providing ideal food for the healthy growth and development of infants'.⁸⁷¹
- Breast milk contains many unique compounds, including live cells, which provide all the nutritional requirements to support growth and development of infants to around 6 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 and protects 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 towards a healthy body weight. Breastfeeding is also associated with reduced risk of some cancers.
- Infants should be exclusively breastfed until around 6 months of age when solid foods are introduced. Breastfeeding should be continued while solid foods are introduced until 12 months of age and beyond, for as long as the mother and child desire.
- Breastfeeding outcomes (including initiation rates and duration) are improved where the mother has support and encouragement from the infant's father, other family members, health workers, the hospital 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, supporting and promoting breastfeeding.

4.1 Setting the scene

The World Health Organization (WHO) states that 'breastfeeding is an unequalled way of providing ideal food for the healthy growth and development of infants'.⁸⁷¹ 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 the WHO *International code of marketing of breast-milk substitutes* (WHO Code),⁸⁷² the main aim of which was to protect and promote breastfeeding. The importance of breastfeeding led to its inclusion in the first edition of the dietary guidelines endorsed by the NHMRC in 1982.⁷⁵⁰

In Australia, the WHO code is implemented through the Marketing in Australia of Infant Formulas (MAIF) Agreement, a voluntary agreement by infant formula manufacturers and importers. The Infant Feeding Guidelines³⁵¹ provide more information on the WHO Code, the MAIF Agreement and the obligations of all health workers. They also provide detailed information on the benefits of breastfeeding for the infant and mother, and recommendations on appropriate foods for infants from birth to about 2 years of age. Other Government initiatives and policies are outlined in the *Australian national breastfeeding strategy 2010–2015* which aims to promote, protect, support and monitor breastfeeding.⁸⁷³

Breast milk is a living tissue that 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 her newborn infant. Colostrum contains higher levels of protein, vitamin A and vitamin B₁₂ and less fat than breast milk. 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.^{657,874,875} Colostrum feeding is important for the infant and also stimulates the mother's breast milk production. Skin-to-skin contact and colostrum feeding should begin as soon as possible after birth, preferably within 30 to 60 minutes. 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 kilojoules per 100ml, largely due to variation in the fat content.^{876,877} Fat content typically increases through each breastfeed. It provides energy and omega-3 and omega-6 LCPUFAs, plus the fat-soluble vitamins A, D, E and K, as well as prostaglandins. The fat in breast milk is typically better absorbed by an infant's gastrointestinal tract than the fat in cow's milk. Mature milk continues to provide the infant with immune factors and enzymes.^{35,878}

Breast milk provides all the vitamins, major minerals and trace elements known to be essential for healthy full-term infants for around the first 6 months of life.^{143,879} These nutrients are more bioavailable than those found in infant formula. Because the composition of breast milk constantly changes throughout lactation and during a single breastfeed, no infant formula can exactly mimic the composition of breast milk. Breast milk is a convenient, hygienic and inexpensive food source with no environmental costs.⁸⁸⁰

Breast milk continues to be an important source of vitamins, minerals and trace elements when other foods are introduced at around 6 months (complementary feeding).

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

4.2 The evidence for ‘encourage, support and promote breastfeeding’

There is considerable evidence to justify the recommendation to encourage, support and promote breastfeeding, including:

- the many benefits of breastfeeding for both infants – in babyhood and later life – and mothers
- the effectiveness of antenatal and postnatal support in increasing the duration of breastfeeding.

Table 4.1: Evidence statements for ‘encouraging, supporting and promoting breastfeeding’

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
Prenatal and perinatal support for breastfeeding can increase the proportion of women breastfeeding (both exclusive and non-exclusive) up to age 6 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 6 months experience less morbidity from gastrointestinal infection than those who are mixed breastfed as of 3 or 4 months.	B
Infants, from either developing or developed countries, who are exclusively breastfed for 6 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 6 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
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 postnatal period and at 6 months of age.	B
Being breastfed initially, particularly exclusively breastfed, is associated with lower total and LDL cholesterol concentrations in adult life.	C
Breastfeeding is associated with a reduced risk of asthma and atopic disease.	C
Not breastfeeding is associated with an increased risk of sudden infant death syndrome.	C
Maternal perceived insufficient milk supply is associated with increased risk of early cessation of lactation.	C

Notes: Grades – A: convincing association, B: probable association, C: suggestive association

Includes evidence statements and gradings from the Evidence Report (literature from years 2002–2009). Does not include evidence from other sources, such as the 2003 edition of the dietary guidelines (in which individual studies were classified according to their design as level I, II or III but overall grades for relationships were not derived), although these sources have been used to inform these Guidelines. Grade C evidence statements showing no association and all Grade D statements can be found in Appendix E.

4.2.1 Breastfeeding incidence and duration

Exclusive breastfeeding

Systematic reviews from developed and developing countries provide evidence of a probable association that exclusive breastfeeding for around 6 months of life² is the best method of feeding for full-term infants (Grade B; Evidence Report, Section 23.2). Breastfeeding can then continue while appropriate solid foods are introduced. The WHO and almost all national and international paediatric and public health organisations make similar recommendations. The Infant Feeding Guidelines³⁵¹ recommend that exclusive breastfeeding be encouraged, supported and promoted to around 6 months of age. For further information about the evidence, refer to the literature review for the Infant Feeding Guidelines.⁸⁸¹⁻⁸⁸⁴

No apparent risks have been reported in recommending exclusive breastfeeding for around the first 6 months of life, in both developing and developed countries (Grade B; Evidence Report, Section 23.2). It is important, however, that health professionals manage all infants on an individual basis, no matter how they are fed, so that any faltering growth or other adverse outcomes do not go unnoticed.³⁵¹

Adding other foods

The introduction of complementary feeding (adding solid foods and liquids other than breast milk or infant formula) at around 6 months is consistent with introduction of solid foods during the probable 'window of tolerance' between 4 and 7 months.⁸⁸⁵ The majority of Australian infants have solids introduced during this period.^{886, 887}

Early cessation of lactation

Recent evidence suggests an association between maternal perceived insufficient milk supply and an increased risk of early cessation of lactation (Grade C; Evidence Report, Section 25.3). Despite this perception, there is little evidence that there is an inability to produce adequate milk, except in rare circumstances related to maternal illness. The advice and support of family and health professionals can be very helpful.

Supportive environments

There is convincing evidence that antenatal and perinatal support for breastfeeding can increase the proportion of women breastfeeding (both exclusive and non-exclusive) up to age 6 months (Grade A; Evidence Report, Section 25.3).

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 postnatal period and when the infant is 6 months of age (Grade B; Evidence Report, Section 25.3).

² In Australia, 'around 6 months' for exclusive breastfeeding is used to acknowledge that different infants develop at different rates.

Table 4.2: Factors associated with duration of exclusive breastfeeding

Association with longer duration of exclusive breastfeeding	Factor
Consistently positive	<ul style="list-style-type: none"> • Higher level of maternal education • Support of midwives and community health professionals • Father's preference for breastfeeding • Doctors' support of breastfeeding • Pleasant birth experience (minimal complications) • Greater breastfeeding knowledge • Rural environment • Time decision to breastfeed is made (preferably before pregnancy or early in pregnancy)
Consistently negative	<ul style="list-style-type: none"> • Lower socioeconomic background • Formula supplementation in maternity ward • Mother's intention to use supplementation • Previous breastfeeding experience short-term (<5 weeks) or absent • Newborn infant not rooming in with mother • Early use of pacifier • High number of intended hours of work per week after maternity leave • Perceived insufficient milk supply and other breastfeeding problems • Smoking, alcohol use • Intention to return to work before 6 months
Effect varies in different cultures	<ul style="list-style-type: none"> • Maternal age • Vaginal delivery • Multiparity

Source: Summarised from Simopoulos *et al* 1995; Landers *et al* 1998; Scott & Binns 1999; Scott 1999; Scott 2001; Kramer *et al* 2002a; 2002b; Hector & King 2005; Britton *et al* 2007; Chung *et al* 2008.^{879,888,896}

4.2.2 Infant growth

For ethical reasons, randomised control trials (RCTs) of breastfeeding are not possible. The best alternative is to use 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 study.⁸⁹⁷

Breastfed infants grow more slowly than formula-fed infants.⁸⁷⁴ 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.⁸⁹⁸ Differences in feeding behaviour 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 over meal sizes and feeding intervals than infants who were formula-fed.⁸⁹⁹

4.2.3 Cardiovascular disease and excess weight

- **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; Evidence Report, Section 23.1).
- **Total and LDL cholesterol:** Recent evidence suggests that being breastfed initially, and particularly exclusively breastfed, is associated with lower total and LDL concentrations in adult life when compared with being formula-fed (Grade C; Evidence Report, Section 23.1).
- **Excess weight:** There is convincing evidence that compared with being formula-fed, being breastfed is associated with reduced risk of infants becoming obese in childhood, adolescence and early adulthood (Grade A; Evidence Report, Section 17.2). The protection offered by breastfeeding appears to increase with duration of breastfeeding and plateaus at 9 months.^{900,901}

Evidence of an association between the age of introduction of solid foods and risk of overweight in children younger than age 7 years is inconclusive (Evidence Report, Section 19.1).

4.2.4 Other benefits

- **Sudden infant death syndrome (SIDS):** There is evidence to suggest that breastfeeding reduces the risk of SIDS (Grade C; Evidence Report, Section 23.4).
- **Gastrointestinal infection:** There is probable evidence that infants who are exclusively breastfed for 6 months experience less morbidity from gastrointestinal infection than those who are mixed breastfed as of age 3–4 months (Grade B; Evidence Report, Section 23.2). Factors in breast milk such as secretory IgA, oligosaccharides and lactoferrin may protect the infant from various infections through passive immunity.⁶⁵⁷
- **Asthma:** Recent evidence suggests an association between breastfeeding and lower incidence of asthma (Grade C; Evidence Report, Section 23.3). 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.
- **Atopic disease:** Recent evidence also suggests an association between breastfeeding and protection against atopic disease (Grade C; Evidence Report, Section 23.3).
- **Lactational amenorrhea:** There is probable evidence that women who exclusively breastfeed for 6 months experience more prolonged lactational amenorrhea, with a delay in the return of fertility (Grade B; Evidence Report, Section 23.2), which may be a benefit for some women. Lactational amenorrhoea is a commonly used form of contraception.⁹⁰²⁻⁹⁰⁴
- **Allergy:** The evidence for an association between a delay in introduction of solid foods until after the age of 6 months and risk of developing allergic syndromes is inconclusive (Evidence Report, Section 19.2).
- **Other benefits for infants:** Additional benefits for infants who are breastfed compared with those who are formula-fed, in both developed and developing countries,^{874,905} may include:
 - protection against respiratory infection and reduced prevalence of asthma⁹⁰⁶
 - reduced occurrence and recurrence of otitis media⁹⁰⁵
 - protection against neonatal necrotising enterocolitis, bacteraemia-meningitis and urinary tract infection^{874,907}
 - reduced risk of autoimmune diseases, such as type 1 diabetes⁹⁰⁵
 - lower rates of coeliac disease, Crohn's disease and ulcerative colitis⁹⁰⁸
 - improved visual acuity, psychomotor development and cognitive development^{880,909}
 - reduced malocclusion as a result of better jaw shape and development⁹¹⁰
 - improved mother and infant bonding and attachment.⁹¹¹

- **Other benefits for mothers:** Additional maternal benefits may include:
 - promotion of maternal recovery from child birth through accelerated uterine involution and reduced risk of haemorrhage (thus reducing maternal mortality)⁹¹²
 - accelerated weight loss and return to pre-pregnancy body weight^{979,986}
 - reduced risk of pre-menopausal breast cancer⁹⁸⁵
 - reduced risk of ovarian cancer⁹⁸⁵
 - improved bone mineralisation and thereby decreased risk of post-menopausal hip fracture.^{913,914}

4.3 Practical considerations: encourage, support and promote breastfeeding

Information relevant to infants and pregnant and breastfeeding women can be found in other chapters of these Guidelines under the 'Practical considerations' sub-headings. Further information can also be found in the *Infant Feeding Guidelines*.³⁵¹

4.3.1 Breastfeeding initiation and duration

It is estimated that in Australia, 96% of mothers initiate breastfeeding and approximately 50% continue to offer some breast milk at 6 months.^{987,915,916} Few infants are exclusively breastfed until 6 months.³³ However, reliable national data on breastfeeding duration rates are difficult to obtain for several reasons, including inconsistent use of definitions of breastfeeding and different methods in studies.^{977,917}

Early interaction

Mothers should have skin-to-skin contact with their babies soon after birth and for as long as they wish, where possible.⁹⁸⁸ Most mothers remain close to their infants and 'rooming-in' 24 hours a day is the usual practice in most Australian hospitals to facilitate frequent mother and child contact.⁹¹⁸ Additional infant feeds are rarely needed and may interfere with establishment of breastfeeding.

Young mothers

Breastfeeding initiation and duration rates are below recommended levels among adolescent mothers.³³ Specific breastfeeding education programs in the US, United Kingdom (UK) and Australia have been somewhat effective in increasing breastfeeding initiation in adolescent mothers.^{919,920} Adolescent mothers identify emotional and network support as well as self-esteem as being crucial to breastfeeding success.⁹¹⁹

Women and paid work

The workplace and parental leave environment has an important impact on breastfeeding rates. The relationship between returning to work and breastfeeding for mothers in Australia is complex, with other interplaying factors, such as maternal and family characteristics, having an impact on the decision to breastfeed.⁹⁷³ There is probable evidence that intention to work or return to paid employment is negatively associated with both the initiation and duration of breastfeeding.^{921,922} Women who are not employed full-time,⁹²² are self-employed or have flexible working hours are more likely to breastfeed for 6 months. Using only parental childcare has a positive association with continuation of breastfeeding.⁹²¹ Where mothers are separated from their infants, they may continue breastfeeding whenever they are together. Continuation of any breastfeeding is of benefit to mother and infant.

Lower socioeconomic status mothers

Women from the lowest socioeconomic quintile in Australia have lower breastfeeding rates than those from the most affluent quintile.^{873,923-925}

Mothers from culturally and linguistically diverse groups

Limited available data suggest that, in general, the rates of breastfeeding among women from culturally and linguistically diverse groups in Australia reflect trends in their countries of origin.⁹²⁶⁻⁹³⁰

Aboriginal and Torres Strait Islander mothers

Breastfeeding status varies by remoteness. In non-remote areas, non-Indigenous infants are likely to be breastfed for longer than Aboriginal and Torres Strait Islander infants. However, in remote areas Aboriginal and Torres Strait Islander infants breastfeed for longer than non-Indigenous infants.^{28,873} In the Perth Aboriginal Breastfeeding Study, Aboriginal mothers had higher breastfeeding rates than non-Aboriginal mothers.⁹³¹

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,⁹³² but each case requires detailed medical assessment.⁹²²

4.3.2 Supporting and promoting breastfeeding

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 primary care breastfeeding interventions significantly increased rates of exclusive breastfeeding in the short and long term.⁸⁸⁹

Effective education programs include information about the benefits of breastfeeding, principles of lactation, myths, common problems and solutions, and skills training.⁹³³ 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 policies, media campaigns, and paid maternity leave.⁸⁹⁰

A more recent Cochrane review shows a protective effect of providing support on increasing duration of breastfeeding.⁸⁸⁸

Promoting breastfeeding in hospitals

The UNICEF and WHO Baby Friendly Hospital Initiative (BFHI) has been shown to increase breastfeeding rates in accredited hospitals.⁹³⁴ The steps in BFHI include the following:

1. Have a written breastfeeding policy that is routinely communicated to all health care staff.
2. Train all health care staff in skills necessary to implement this policy.
3. Inform all pregnant women about the benefits and management of breastfeeding.
4. Help mothers initiate breastfeeding within one hour of birth.
5. Show mothers how to breastfeed and how to maintain lactation even if they should be separated from their infants.
6. Give newborn infants no food or drink other than breast milk, unless medically indicated.
7. Practice rooming-in (allow mothers and infants to remain together) 24 hours a day.
8. Encourage breastfeeding on demand.
9. Give no artificial teats or pacifiers (also called dummies or soothers) to breastfeeding infants.
10. Foster the establishment of breastfeeding support and refer mothers to them on discharge from the hospital or clinic.

Note: In the Australian version of the BFHI step 4 is phrased: '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'. These Guidelines refer to the original BFHI, as this version has been extensively evaluated.^{297,336,337}

Early contact improves breastfeeding outcomes.³³ 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.³¹⁸ The use of prelacteal feeds or other liquids while in hospital interferes with the establishment of lactation and is contrary to BFHI principles.

Community support

Successfully managing breastfeeding problems in the first weeks after birth has a major impact on breastfeeding duration.³³⁸ Sources of support for mothers in the first few weeks include family members, community health nurses, voluntary organisations and GPs. Preparation and education before birth are very important in achieving successful breastfeeding. Efforts to improve community acceptance of breastfeeding are also critical.

The legislative environment plays an important role in reducing discrimination against breastfeeding mothers. It is legal to breastfeed in public in every state and territory of Australia. Most jurisdictions also have specific legislation making it unlawful to discriminate against breastfeeding mothers.⁷³

4.3.3 Expressing breast milk

Many mothers find it convenient to express breast milk so that others can feed their baby if required. Appropriate hygiene is essential for the expression and storage of breast milk. For details of methods of expression and the safe storage of breast milk see the *Infant Feeding Guidelines*.³⁵¹

The use of a breast pump to express and measure breast milk 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 infant growth are the best way to assess nutritional adequacy.

4.3.4 Alcohol and breastfeeding

Alcohol is best avoided during breastfeeding. For more details, see Section 3.4 and the *Infant Feeding Guidelines*.³⁵¹

4.3.5 When an infant is not receiving breast milk

When an infant is not breastfed or is partially breastfed, commercial infant formulas should be used as an alternative to breast milk until 12 months of age. All formulas available in Australia are regulated by the Australia New Zealand Food Standards Code and contain adequate nutrients for infants. A mother's decision not to breastfeed should be respected and support should be provided where needed. Cow's milk should not be given to infants under 12 months of age as a main drink. Complete details on the use of infant formula and the introduction of solid foods are provided in the *Infant Feeding Guidelines*.³⁵¹

4.4 Practice guide for Guideline 4

Table 4.3: Considerations in encouraging, supporting and promoting breastfeeding

Strategy	Considerations
Increased duration of breastfeeding	<ul style="list-style-type: none">• Encourage exclusive breastfeeding until around 6 months of age when solid foods are introduced• Encourage the continuation of breastfeeding while solid foods are introduced until 12 months of age and beyond, for as long as the mother and child desire
Antenatal education	<ul style="list-style-type: none">• Information should include the benefits of breastfeeding and the risks of not breastfeeding to mothers, fathers and primary carers, as well as principles of lactation, myths, common problems and their solutions
Hospital support of breastfeeding	<ul style="list-style-type: none">• Promote the principles of the Baby Friendly Hospital Initiative including:<ul style="list-style-type: none">– Measures to encourage initiation of breastfeeding include early skin-to-skin contact, rooming-in and breastfeeding on demand– Staff should be trained in skills to support initiation of breastfeeding and resolution of any early problems– Discourage interventions that interfere with establishing lactation e.g. the use of artificial teats or pacifiers
Community support of breastfeeding	<ul style="list-style-type: none">• Seek to address breastfeeding problems in the first few weeks. Encourage mothers to seek support from community health nurses, voluntary organisations (such as Australian Breastfeeding Association helpline) and GPs• Community acceptance and support of breastfeeding should be encouraged

Where to next?

Everyone should prepare and store food safely; however particular care should be taken when handling food intended for consumption by those at 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 or storage of food can contribute to food poisoning and poor health outcomes.

GUIDELINE 5

Food safety

Guideline 5

- Care for your food; prepare and store it safely.

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 preventing foodborne illness and why it is important to store and prepare food safely.

5.1 Setting the scene

Foodborne illness is caused by contaminated foods. Contaminants include pathogens, environmental contaminants and adulterants.⁹³⁹ These Guidelines focus on bacterial and viral contaminants.

Food poisoning generally occurs when pathogenic micro-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 (e.g. 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 (e.g. *Clostridium botulinum*, *Staphylococcus aureus*, *Bacillus cereus* and *Shigella spp.*)⁹⁴⁰

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.⁹⁴¹ Heat can kill many bacteria and viruses and is the basis for many food safety strategies. However, even reheating food to high temperatures will not destroy all toxins.

The increasing age of many populations, migration, mass production of food due to population growth and changed food habits are threats to food safety.⁹⁴² Lack of access to quality food, as well as 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.

Dramatic scientific and technological improvements in detecting pathogens have contributed to the identification of increasing numbers of cases of foodborne disease. Foodborne illness appears to be increasing in incidence in Australia and worldwide, and is a significant public health problem.^{943,944} In 2000, the Australian Government Department of Health and Ageing established OzFoodNet, an enhanced foodborne disease surveillance system designed to improve national surveillance of gastrointestinal and foodborne illness.⁹⁴⁵ The most recent estimates indicate there are 5.4 million cases of foodborne illness in Australia every year, leading to 1.2 million visits to medical practitioners and 2.1 million days of work lost.⁹⁴⁶ Multiple episodes increase the risk of long-term consequences such as reactive arthritis, irritable bowel syndrome and, rarely, Guillain-Barre Syndrome.⁹⁴⁵

5.2 The evidence for ‘care for your food; prepare and store it safely’

The true incidence of foodborne illness is consistently underestimated because cases are under-reported, most cases are sporadic and full diagnostic testing is usually only done in more severe cases or when there are extensive common-source outbreaks.⁹⁴⁷⁻⁹⁴⁹ 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 illness in Australia.⁹⁴⁸ 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^{947,950}
- improper holding temperatures^{947,950}
- contaminated equipment (such as knives, cutting boards and dishcloths)⁹⁵⁰
- contaminated food storage and preparation areas
- unsafe raw food⁹⁵⁰
- allowing raw foods to make direct contact with ready-to-eat foods⁹⁴⁷
- poor personal hygiene of food handlers (such as not washing hands adequately, particularly after handling raw food or immediately after using the toilet).⁹⁵⁰

5.2.1 Foods that may cause problems if not handled correctly

The following are examples of foods that are normally considered higher risk because pathogenic bacteria can be present and grow if not stored and prepared safely:⁹⁵¹

- 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 (e.g. sandwiches).

The foods most commonly implicated in outbreaks of foodborne illness in Australia are meat, seafood and eggs.^{545,547} Contamination of any ready-to-eat food with a pathogen such as a virus or enterohaemorrhagic strain of *E. coli* can result in foodborne illness.

5.3 Why it is important to prepare and store food safely

Foodborne illness results from consuming contaminated food or drink. Correct handling of food during all stages of its preparation and storage is essential in reducing the risk of contamination and foodborne illness.

The ability of bacteria to grow in a food depends on external factors (such as temperature) as well as characteristics of the food itself, such as protein content,^{552,553} 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.⁵⁵¹ 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.⁵⁵¹

Some bacteria multiply within 2 hours and can reach an infective dose in 4–6 hours.^{552,554} Refrigeration at or below 5°C slows the growth of bacteria and the rate of chemical change in food.⁵⁵¹

The method chosen for thawing food should minimise the time the food is at room temperature, where micro-organisms can multiply. Ready-to-eat frozen foods should be thawed in the refrigerator or under cold water in an airtight plastic wrapper or bag, with the water changed every 30 minutes.⁵⁵⁵

Many foods should be cooked to at least 75°C. A thermometer should be used to check food is properly cooked to a minimum safe temperature (roasts and meats 62°C; mince, eggs, soups 71°C; whole poultry 82°C).^{556,557}

Not all meats need to be cooked thoroughly: steaks, whole fillet, chops and whole pieces of roast meat can be eaten rare. In contrast, rolled and/or stuffed meats, poultry, pork, sausages and mince should always be cooked all the way through, until the juices run clear when the meat is pierced.⁵⁵²

Cooking does not guarantee safety because some bacterial spores can survive several hours of cooking and still grow later in the food if there is poor temperature control. Foods such as stews and other meat and poultry dishes that will be eaten later should be cooled as quickly as possible to prevent spores from germinating and bacteria from multiplying. Foods that have just been cooked and are still very hot can be cooled at room temperature until the temperature of the food drops to 60°C. The food should then be cooled to 5°C as quickly as possible.^{552,558}

5.4 Practical considerations: care for your food; prepare and store it safely

The use of date marking provides a guide on the shelf life of a food item in terms of quality and safety. The term 'best before' indicates the length of time a food should keep before it begins to deteriorate while 'use by' indicates how long a food can be expected to remain safe provided it has been stored according to any stated storage conditions and the package is unopened.⁵⁴¹

5.4.1 Pregnant and breastfeeding women

As the immune system in pregnancy is suppressed, pregnant women are more susceptible to foodborne illness. 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.⁵⁵⁹ As a precaution, pregnant women are advised to avoid specific foods that are more likely to contain *Listeria* bacteria (for further information see Sections 2.4.4 and 2.5.4 and the FSANZ website³⁴⁹). Pregnant women are also advised to be careful with foods more likely to contain mercury (such as fish).⁵⁶⁷

5.4.2 Infants

The immune system of infants is not fully developed, making them more susceptible to foodborne illness. Particular care should therefore be taken when preparing infant formula (if this is being used), including the sterilisation of bottles and other equipment used to prepare formula.⁹⁵⁰ Further information is included in the Infant Feeding Guidelines.³⁵¹ Pacifiers (dummies) should also be sterilised. Prepared foods should not be reheated more than once, and any foods served but not eaten should be discarded.

5.4.3 Adults with illness

Adults with conditions that reduce immune function such as human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), cancer, type 1 or type 2 diabetes, kidney or liver disease, haemochromatosis, stomach problems (including previous stomach surgery) and low stomach acid (e.g. 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.

5.4.4 Older people

Older adults, especially those who are frail, are at higher risk of foodborne illness than other adults due to their weakened immune systems, decreased intestinal motility, decreased acid in the stomach, loss of sense of smell and taste, and increased risk of dementia and malnutrition.^{956,957} High-risk foods should be cooked, dairy products should be pasteurised, and specific foods at risk of *Listeria* contamination avoided. Information on *Listeria* bacteria is available from FSANZ.^{3,49}

People of any age, but especially older people receiving pre-prepared meals, need to ensure that they are safely stored if not consumed immediately.

5.5 Practice guide for Guideline 5

Table 5.1: Considerations in providing advice on food safety to people from specific groups

Population group	Considerations
Pregnant and breastfeeding women	<ul style="list-style-type: none">• Foods associated with a risk of <i>Listeria</i> bacteria should be avoided• Care should be taken with foods more likely to contain mercury (e.g. certain types of fish)
Infants	<ul style="list-style-type: none">• If formula-fed, care should be taken when preparing formula, including sterilising bottles and other equipment• Pacifiers (dummies) should be sterilised• Foods should not be reheated more than once, and any foods served but not consumed should be discarded
Adults with illness	<ul style="list-style-type: none">• Impaired immune function increases the risk of foodborne illness – prepared foods should be handled with particular care
Older people	<ul style="list-style-type: none">• High-risk foods should be cooked, dairy products pasteurised, and specific foods associated with a risk of <i>Listeria</i> bacteria avoided• Pre-prepared meals should be safely stored if not consumed immediately

Appendices

Appendix A Equity and the social determinants of health and nutrition status

The World Declaration on Nutrition (1992) states that ‘access to nutritionally adequate and safe food is a basic individual right’.⁹⁶⁰ Australia is fortunate to have an abundant and safe food supply. Life expectancy and health status are relatively high.^{11,24,45} Australians are generally literate and have good access to health and nutrition information and sufficient education to make informed food choices.⁹⁶¹

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 preventive health services.⁹⁶² As in other countries, there is a socioeconomic gradient whereby health status generally improves the higher a person is up the socioeconomic ladder.²⁴

The determinants of health inequities are largely outside the health system and reflect the distribution of social, economic and cultural resources and opportunities.^{25,26,960,962} 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.^{25,26}

The relationship between these factors and health status is complex and it is often difficult to determine the nature and direction of causal relationships.^{25,26} 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 (e.g. 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.^{25,26}

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.²⁴

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.³³ 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.^{44,314,316,963-979}

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.^{44,314,316,963-979}

In a Melbourne study it was found that areas of greater socioeconomic advantage had closer access to supermarkets, whereas areas of less socioeconomic advantage had closer access to fast food outlets.³⁵⁵

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 to reduce the risk of adding to health inequities, for example by promoting consumption of expensive or hard to access foods.

A1 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 socioeconomic group than the highest socioeconomic group.²⁴ These rates reflect the higher prevalence of type 2 diabetes and cardiovascular disease among people living in the lowest socioeconomic group.^{11,24}

Gradients in risk factor prevalence are also apparent across quintiles of social disadvantage as defined by socioeconomic 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.²⁴ Overweight and obesity rates are also highest among the lower socioeconomic areas, although there was not a clear gradation across SEIFA quintiles.²⁴ 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 1). There is also a social gradient in physical activity levels; self-reported 'sedentary' behaviour rises from 25% of people in the highest socioeconomic areas to 44% in the lowest socioeconomic areas.²⁴

A2 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,³¹⁴ 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 disadvantage³¹⁵ and determinants of healthy eating for those with low income.³¹¹ They found that economic and cultural influences affect consumption of specific foods or food groups. Nutrition interventions can have greater impact in higher socioeconomic areas and non-ethnic groups.^{310,311}

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.³¹² 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.³¹³

Economics of food choice

There is a growing body of research indicating 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.^{142,264} For example, 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.^{142,264,315,314} Although high-quality nutrition is known to protect against chronic diseases, energy-dense, nutrient-poor foods cost considerably less than nutrient-rich foods.¹⁴⁸

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. Between 2000 and 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.³¹⁷

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.³¹⁶

Food security

Food security refers to the ability to access safe 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.^{315–317} It is more common among:

- people who are unemployed³¹⁷
- Aboriginal and Torres Strait Islander peoples³¹⁵
- people living in single parent households³¹⁷
- people in the second lowest income quintile³¹⁷

- people living in rental households⁹⁸⁷
- younger people than older people⁹⁸⁷
- women than men (in some surveys but not others).^{985,986}

Issues of food security in the face of rising food costs are of concern in Australia.³¹⁷ 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 effects 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%.⁹⁸⁹

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.³¹⁷

Social distribution of intake of foods and nutrients

The 1995 National Nutrition Survey³⁶ showed few systematic differences in food and nutrient intake across quintiles of social disadvantage, as defined by SEIFA based on the 1991 census. Table A1 shows the intake of various food groups across SEIFA quintiles from the Survey.

Table A1: Mean daily intakes (g/day) of various food groups, people aged 19 years and over, by SEIFA quintile

Food group	Grams per day				
	First quintile (most dis- advantaged)	Second quintile	Third quintile	Fourth quintile	Fifth quintile (least dis- advantaged)
Cereal and cereal products	196	222	203	217	232
Cereal-based products and dishes	113	115	130	135	136
Fruit products and dishes	126	147	141	143	156
Vegetable products and dishes	264	258	260	262	251
Legumes and pulses	9.8	7.9	10.8	9.5	10.7
Milk products and dishes	281	284	285	292	301
Meat, poultry, game	149	163	164	155	158
Fish and seafood	22	24.5	26.3	25.8	28.8
Egg products and dishes	16	15	16	13	19
Snack foods	3.2	3.0	4.2	3.3	3.9
Sugar products and dishes	20	21	20	18	17
Confectionery	7.8	9.2	8.3	9.3	9.1
Seeds and nuts	3.6	4.1	5.2	4.0	4.8
Fats and oils	12	13	13	12	11
Soup	53	62	55	48	57
Savoury sauces and 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:⁹⁹⁰

- 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 foods³⁶).
- Consumption of sugar products and dishes tended to decrease with social advantage.
- Consumption of cereals and cereal-based foods (e.g. 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 (e.g. cakes and biscuits) was about 20% lower in the two most disadvantaged groups compared with the other three.

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.⁹⁹¹

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.⁹⁹⁰ 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.³⁶

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 (e.g. 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 A2: Mean daily intakes of energy and nutrient densities, adults aged 19 years and over, by SEIFA quintile

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
<i>Nutrient density (per 10 MJ energy)</i>					
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
Monounsaturated (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

Food group	First quintile (most disadvantaged)	Second quintile	Third quintile	Fourth quintile	Fifth quintile (least disadvantaged)
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 undernutrition in lower socioeconomic groups further contributes to the incidence, severity and case fatality of childhood illnesses⁸⁸⁴ and incidence of chronic disease in later life.

A3 Aboriginal and Torres Strait Islander peoples

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.⁹⁸⁸ 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.⁹⁹²

Socioeconomic disadvantage underlies many of these health statistics.⁹⁹³ Compared with non-Indigenous Australians, Aboriginal and Torres Strait Islander peoples report lower incomes, higher rates of unemployment, lower educational attainment, more overcrowded and inadequate housing,⁹⁹⁴ higher rates of incarceration and limited access to transport.²⁴ Disrupted family and community cohesion, social marginalisation, stress, lack of control over circumstances, and discrimination and racism are also apparent.^{28,993}

Overweight and obesity are common.^{28,311} Measured anthropometric data for Aboriginal and Torres Strait Islander peoples 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.⁹⁹⁵

Undernutrition among young children and relatively poor growth from around 6 months of age persists in some parts of Australia.⁹⁹⁶⁻⁹⁹⁹

Foetal alcohol syndrome is also still present in some Aboriginal and Torres Strait Islander communities.^{863,864}

Vitamin and mineral status has been measured infrequently in Aboriginal and Torres Strait Islander populations.²⁸ Multiple deficiencies have frequently been described in the same subject, suggesting the generally poor nutritional status of such individuals, rather than a specific micronutrient problem. In particular, vitamin status (in relation to folate, ascorbic acid and beta-carotene) consistent with low intakes of fruit and vegetables has often been described.^{7,352} More recently iodine deficiency in an Aboriginal birth cohort in the Northern Territory¹⁰⁰⁰ and low vitamin D status in a South Australian Aboriginal population¹⁰⁰¹ have been found.

Food intake, diet and nutritional status

The available evidence suggests that, traditionally, Aboriginal and Torres Strait Islander peoples 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 micronutrients. Energy expenditure appears to have been high.^{28,100,102,106}

With the transition from a traditional hunter-gatherer lifestyle to a settled Westernised existence, the diet of Aboriginal and Torres Strait Islander people has generally changed to an energy-dense diet that is high in fat and added sugars.^{311,1005,1006} The diet is also poor in fibre and certain nutrients including folate, retinol, vitamin E and other vitamins.³⁵²

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.⁹³³ 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 New South Wales to 45% in the Northern Territory.⁹³³ Women are at extra risk, partly because there is a cultural predisposition for women to feed men and children before themselves.⁹⁶¹ 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.¹⁰⁰⁷

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.^{7,352,353,440}

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.^{352,353,440} 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.^{353,1005} Food supply is an ongoing issue¹⁰⁰² with people in rural and remote areas paying at least 30% more for basic nutritious foods than people living in urban and metropolitan areas.^{317,1005,1014} Basic food items are less available in the more remote stores, as are fresh vegetables and fruits and better nutritional choices.³¹⁷ The quality of dietary intake has been shown to vary in close association with the income cycle in remote Aboriginal communities.^{1015,1016}

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 1 hour a week hunting or foraging for traditional foods and, of these, more than half reported spending more than 5 hours a week doing so.⁹⁸³ 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.^{7,1005,1017-1019}

Aboriginal and Torres Strait Islander 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.^{1007,1020-1022}

Recommendations specific to Aboriginal and Torres Strait Islander peoples

The general Australian dietary guidelines are relevant to Aboriginal and Torres Strait Islander peoples.^{353,440,1023-1026} 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 3–5 years may, however, be problematic in some areas or for some individuals.^{610,611} Alternative calcium sources such as chewing meat and fish bones, and consumption of small, soft fish bones (e.g. 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.¹⁰²⁷ In addition, there may be a problem with high levels of heavy metals in the organ meat of turtle and dugong.¹⁰²⁸

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:

- enjoying 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 meats and poultry.

A4 Women

Women are particularly vulnerable to poverty, illiteracy, food insecurity and poor health.^{25,26,334} Single parent families, of which 87% are headed by women in Australia, face higher risk of poverty and food security.^{1029,1030}

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.

A5 Infants and children

Children, particularly those under 5 years of age, 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.³³⁴ According to the 2005-06 New South Wales Population Health Survey, exclusive breastfeeding of children at 6 months of age was significantly lower for infants with:³⁷⁷

- mothers without tertiary qualifications (13%) compared with those with tertiary qualifications (25%)
- mothers living in the lowest socioeconomic status areas (11%) compared with those in the highest socioeconomic status areas (26%)
- mothers aged younger than 25 years (9%) compared with mothers aged 25 years and over (17%).

Further information is available in the *Infant Feeding Guidelines*.³⁵¹

A6 Older people

Living alone, as many older adults do, has been associated with a poorer, less varied diet. 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 older people has increased.

The Dietary Guidelines are not applicable to frail elderly people as reducing food components such as fat, salt and sugar – which may make food more palatable — is not always appropriate in this group.

A7 People born overseas

Many migrants enjoy health that is as good as, if not better than, that of the Australian-born population.²⁴ 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.²⁴

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.²⁴

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, the United Kingdom and southern and eastern Europe are more likely to be overweight or obese than those born in Australia.²⁴

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.²⁴

A8 Rural and remote Australians

Mortality rates increase as remoteness increases.²⁴ People living outside major cities are more likely to have hypertension, 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.²⁴

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 risky alcohol use, less access to health services and staff, and the hazards of driving long 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.²⁴

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.^{317,333,1005,1011-1014} Among the most remote communities, costs are highest in those areas greater than 2,000 km from capital cities, suggesting prices are influenced markedly by transport costs.³¹⁷ The availability of nutritious foods is limited in regional and remote areas in northern Australia.^{317,333,1005,1011-1014} But even in south-west Victoria, people living in more remote towns without a major supermarket faced limited availability of healthy food basket items.³²⁷ In New South Wales, the variety of fruit and vegetables available also decreases with remoteness and also with decreasing socioeconomic status of the community.¹⁰³¹

Appendix B Process report

In 2003, the 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 5 years, the 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 a single document.

The *Australian Dietary Guidelines* (the 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 frail elderly people who are at risk of malnutrition. The target audience of the Guidelines includes health professionals (including dietitians, nutritionists, GPs and nurses/lactation consultants), educators, government policy makers, the food industry and other interested parties. Other resources are being developed from the Guidelines for other target audiences.

B1 Contributors

The Guidelines were developed utilising a collaborative approach, combining the content expertise of the Dietary Guidelines Working Committee with the assistance of expert methodologists. The NHMRC managed the process and representatives from DoHA participated as observers throughout the process. DoHA provided a significant proportion of the funding for developing these Guidelines.

Dietary Guidelines Working Committee

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 and advise the redevelopment of the *Australian Dietary Guidelines*. Representatives from DoHA attended working committee meetings as observers.

The Working Committee developed the guidelines in accordance with NHMRC procedures and in keeping with the following terms of reference established by the NHMRC.

Table B1i: Terms of reference of the Dietary Guidelines Working Committee

The Dietary Guidelines Working Committee (the Working Committee) will oversee and provide expertise on the revision of *The Core Food Groups* (1994), the *Dietary Guidelines for Older Australians* (1999), the *Dietary Guidelines for Children and Adolescents incorporating the Infant Feeding Guidelines for Health Workers* (2003), the *Dietary Guidelines for Australian Adults* (2003). This will include:

1. Input to the development and conduct of necessary literature reviews
2. Advice on development of a consultation strategy
3. Input to the development of appropriate documents including guidelines which will take into account:
 - the best available scientific evidence
 - comments provided by the broader community through public consultation
 - the needs of health service providers
 - any other relevant matter.

Table B2: Members of the Working Committee

Professor Amanda Lee (Chair)

School of Public Health and Social Work and School of Exercise and Nutrition Sciences,
Queensland University of Technology

Professor Colin Binns (Deputy Chair)

School of Public Health, Curtin University

Dr Geoffrey Annison (from August 2010)

Australian Food and Grocery Council

Professor Sandra Capra, AM

School of Human Movement Studies, University of Queensland

Professor Peter Davies

School of Medicine, University of Queensland
Children's Nutrition Research Centre

Professor Sharon Friel

National Centre for Epidemiology and Population Health, Australian National University

Ms Clare Hughes

Cancer Council NSW

Associate Professor Mark Lawrence

School of Exercise and Nutrition Sciences, Deakin University

Professor Dorothy Mackerras

Food Standards Australia New Zealand

Dr Rosemary Stanton, OAM

Visiting Fellow, School of Medical Sciences, University of New South Wales

Professor Linda Tapsell

School of Health Sciences, University of Wollongong

Dr David Roberts (until May 2010)

Food and Nutrition Consultant

Dr Karen Webb (until June 2009)

School of Public Health, University of Sydney

NHMRC project team

- Ms Cathy Connor
- Ms Emma Breen
- Ms Tess Winslade
- Ms Bronwyn Battison

Department of Health and Ageing Project Team

- Ms Jacinta McDonald
- Ms Erica Nixon
- Ms Marina Dron
- Ms Rosalind Knox
- Ms Fiona Styles

Contractors

- Ms Skye Newton, Adelaide Health Technology
- Ms Philippa Middleton, Methodologist, University of Adelaide
- Dr Katrina Baghurst, Australian Guide to Healthy Eating
- Ragga Ahmed, Technical writers (until public consultation)
- Mr Simon Grose, Editor (public consultation draft)
- Ampersand Health Science Writing, Editor (final version)
- Quantum Market Research, Focus Group Testing
- Folk Pty Ltd, Graphic Design for Australian Guide to Healthy Eating

Declarations of interest process

Declarations of interest were made by all Working Committee members during the review process in accordance with the requirements of the *National Health and Medical Research Council Act 1992*. A record of interests was managed by NHMRC and relevant information was made publicly available on the NHMRC website to ensure transparency.

During meetings where committee members were identified as having a significant real or perceived conflict of interest, the Working Party Chair could request they leave the room or not participate in discussions on matters where they were conflicted. Working Committee members were required to update their information as soon as they became aware of any changes to their interests and there was a standing agenda item at each meeting where declarations of interest were called for and these were recorded in the meeting minutes.

B2 Literature review

In the past, Australian 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 (DAA) 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 edition of the dietary guidelines where the evidence base may have changed. A number of established food, diet and health relationships covered in the 2003 edition, 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 serum 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 (systematic review of the primary literature)
- umbrella review (systematic review of systematic reviews)
- narrative reviews (comprehensive review of the literature to answer more qualitative questions e.g. 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.³²

Standardised processes were used to review the literature.^{32,40-42} 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 (e.g. letters and editorials) were also excluded.

The evidence was assessed according to *NHMRC levels and grades for recommendations for developers of guidelines*,⁴² 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.

Table B3: Levels of evidence in the literature review

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: <ul style="list-style-type: none"> • 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: <ul style="list-style-type: none"> • 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.^{32,40,42} 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.^{38,42} 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:⁴²

- 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 included in meta-analysis studies were considered as separate studies.

Once the evidence statements had been drafted and graded, the NHMRC commissioned a methodologist through the NHMRC methodologist panel, Ms Philippa 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* (Evidence Report).³³

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 Ms Philippa Middleton and comprising Professor Maria Makrides, Dr Carmel Collins, Dr Alice Rumbold, Dr Jo Zhou, Professor Caroline Crowther and Associate Professor 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 Professor Friel and a team at the Australian National University. On the basis of this work, NHMRC recognised that the inter-relationship between diet and the environment is a cross-sectoral matter and should be considered as such.

B3 Food Modelling System

In 2008, the DAA was commissioned through an open Request for Tender process to undertake an extensive review of *The Core Food Groups* (1994). This revised document, *A modelling system to inform the revision of the Australian Guide to Healthy Eating* (Food Modelling System), was released for public consultation from 27 March 2010 to 10 May 2010. The finalised Food Modelling System was released in 2011.^{9,10,32}

The Food 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 Food Modelling System were considered together with other sources of evidence to determine the recommendations in these Guidelines (see Sections B2, B4 and B5).

For further information or to download the Food Modelling System visit www.eatforhealth.gov.au.

B4 Development of the Australian Guide to Healthy Eating

The development of the *Australian Guide to Healthy Eating* was undertaken in conjunction with an update of the *Dietary Guidelines for Australian Adults*, the *Dietary Guidelines for Children and Adolescents in Australia* and the *Dietary Guidelines for Older Australians*.

It was informed by the Food Modelling System and under the guidance of the Working Committee, Professor Katrina Baghurst was directly sourced to develop the revised *Australian Guide to Healthy Eating* given her extensive knowledge of the Food Modelling System.

While the modelling was undertaken to inform the revision of the *Australian Guide to Healthy Eating*, some of the complexities of the modelling were simplified for the final presentation of the guide. For example, several different subgroups of vegetables were modelled but the final recommendation in terms of serves per day is for the Vegetable group as a whole, with additional advice to choose across the various subgroups.

As part of the consultation process, Quantum Market Research was commissioned through an open Request for Tender process to conduct focused discussion in October 2010 and November 2011 to determine the preferred graphics and the messages for the *Australian Guide to Healthy Eating*. Discussion was undertaken with:

- eight groups consisting of members of the community
- four groups consisting of professionals including health, teaching, child care and community health
- two groups consisting of Aboriginal and Torres Strait Islander populations and professionals.

The groups from the general public (all adults) were divided into two age groups with a good representation across the population.

Results were also available from a survey of 40 dietitians attending a workshop at the DAA meeting in 2007. This survey was concerned with attitudes to the current *Australian Guide to Healthy Eating* including:

- use of the guide and what changes would make it more usable
- which other food guides were used and the advantages/disadvantages of these guides
- suitability of food groupings
- changes that might be needed and preferred presentation formats.

A web-based survey was also undertaken by NHMRC in 2007 before commencing the reviews of the *Australian Guide to Healthy Eating* and the *Guidelines*.

Under the direction of the NHMRC Project Team and NHMRC Publications Team, Folk Pty Ltd were engaged from the Australian Government Department of Human Services Creative and Design Panel to complete the graphic design work for the Australian Guide to Healthy Eating.

B5 Development of the *Australian Dietary Guidelines*

In translating the evidence to formulate the dietary guideline recommendations, the Working Committee considered the following sources of evidence:

- the commissioned literature review (Evidence Report)³³
- the Food Modelling System⁹
- the NRV Document⁸
- 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, and
- the previous dietary guidelines for Australians series and their supporting documentation.³⁴⁻³⁶

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 guideline recommendations was developed by a Working Committee consensus approach and was focus tested by Quantum Market Research. Guidelines recommendations were further refined following the public consultation process.

Ragga Ahmed was commissioned in October 2010 by NHMRC through a Request for Quote process through the NHMRC Technical Writers and Editors Panel to edit the draft chapters and collate them into one succinct report including all referencing. This report underwent further editing by Mr Simon Grose from the NHMRC Technical Writers and Editors Panel before release for public consultation.

Following public consultation and expert review, with the advice of the Working Committee, the NHMRC project team finalised the technical writing of the Guidelines. Ampersand Health and Science Writing was selected through a Request for Quote process from the NHMRC Technical Writers and Editors Panel to complete the final editing.

The NHMRC and DoHA project teams also developed the companion resources including an interactive website, guideline summaries, posters and brochures relating to the general population, infants, children and pregnant women.

Public consultation

On 2 November 2011 the Council of NHMRC 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 13 December 2011 to 29 February 2012. This process was conducted in accordance with Section 13 of the *National Health and Medical Research Council Act 1992*. The public consultation was advertised in 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. Over 200 submissions were received from a variety of stakeholders including individuals, professionals and industry.

The Working Committee met on 17 and 18 April 2012 and again on 22 May 2012 to consider the submissions. More information and/or consideration were requested for:

- the impact of food choices and the environment (practical recommendations required for environmentally sustainable food choices)
- changed recommendation wording from 'enjoy' to 'eat'
- the approach to vegetarian and vegan diets
- content around 'red meat'
- the placement of fats and oils 'outside' of the five major food groups
- lack of information on glycaemic index
- the recommendation on added sugars
- the recommendation on foods to limit with consideration for 'food-based' approach
- the recommendation for mostly 'reduced fat' dairy
- increased information on the concept of 'energy density'
- the beneficial properties of fibre
- practical issues relating to reducing fat and types of fats
- legumes appearing in two food groups, and
- difficulties interpreting the *Australian Guide to Healthy Eating* and companion information (including comments on cultural diversity and relative sizes of foods).

A second public consultation on Appendix G to the Guidelines was undertaken from 3 October 2012 to 2 November 2012. This process was conducted in accordance with Section 13 of the National Health and Medical Research Council Act (1992). The public consultation was advertised in 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 and sustainability. Over 70 submissions were received from a variety of stakeholders including individuals, professionals, government and industry.

These submissions were considered by the Office of NHMRC. More information and/or consideration were requested for:

- focus on improving health but allowing for considerations of the environmental impact of dietary patterns, specifically overconsumption and waste management.
- greater acknowledgment of the complexities of the food system, including the concept of 'triple bottom line' sustainability, and the difficulties in assessing commodities individually and/or in isolation from the whole food system.
- clarification of Figure G1 to better represent a system (cyclic) rather than linear process, including recognition of potential 'drivers' such as consumer and industry interest, and government policy.
- acknowledgement of initiatives to improve sustainability at both government and non-government levels.
- further information for health professionals (with references) with more comprehensive and practical suggestions.

Expert review

The Guidelines underwent independent expert review to gain advice, primarily on the evidence base used. The expert reviewers were required to declare any conflicts of interest as per the process outlined in Section B1.

Table B4: Expert review participants

Professor Louise Baur

University of Sydney, Australia

Professor Johanna Dwyer

Tufts Medical Centre, Tufts University, United States of America

Professor Caryl Nowson

School of Exercise and Nutrition Sciences, Deakin University, Australia

Professor Murray Skeaff

Department of Human Nutrition, University of Otago, New Zealand

Professor Boyd Swinburn

School of Health and Social Development, Deakin University, Australia

School of Population Health, University of Auckland

Council of NHMRC Endorsement

The Guidelines were considered by the Council on 30 November 2012 for recommendation to the CEO for issuing. The CEO was pleased to accept the Council's advice and agreed to issue the guidelines under Section 7(1a) of the *National Health and Medical Research Council Act 1992*.

Review

In line with NHMRC policy, Council will look at the current Guidelines after 5 years and recommend to NHMRC CEO if they need to be reviewed.

Appendix C 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 1,100 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 1982.⁷⁵ 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.¹⁰³³

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.¹⁰³⁴

1989–95 – 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 1991.¹⁰³⁵ 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 1992,³¹ aimed 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*¹⁰³² was the modelling document that underpinned the development of *The Australian Guide to Healthy Eating* (see below). The purpose of *The Core Food Groups* was to discuss the basis for a core food group system that reflected advances in nutrition knowledge and to complement existing nutrition references at the time (e.g. *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 could be updated 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*⁴⁸ was a food guide for Australia that reflected the multicultural nature of the population. Based on the modelling of *The Core Food Groups*, it was 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*²⁴ were based on the *Dietary Guidelines for Australians* (1992)⁷⁵¹ and were designed to take account of the changes in nutritional needs that occur with ageing. The Guidelines were aimed at healthy, independent Australians aged 65 and over, but additional advice was provided on how the Guidelines applied 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 AUSNUT (Australian Food and Nutrient Database) was 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.¹⁰³⁶ A second edition, used to code the 2007 Australian Children's Nutrition and Physical Activity Survey,¹² was released in 2008.¹⁰³⁷ The third edition, developed to code the 2011–13 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 health professionals 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)¹⁰³ 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
- 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

The *Dietary Guidelines for Australian Adults*³⁶ and *Dietary Guidelines for Children and Adolescents in Australia*³⁵ (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 eating and lifestyle 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 (NRV Document)

In 2002, the NHMRC was commissioned by 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 NRV Document,⁸ which 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 2011.¹⁰³ Most of the NUTTAB data is now Australian analytical data and covers more than 2,500 foods available in Australia and up to 45 nutrients per food.

2011 – A review of the evidence to address targeted questions to inform the revision of the Australian Dietary Guidelines (Evidence Report)

The primary aim of the Evidence Report³³ 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. New and emerging evidence was prioritised in this review.

2011 – A modelling system to inform the revision of the Australian Guide to Healthy Eating (Food Modelling System)

In 2009 and 2010, the nutrient recommendations from the 2006 Nutrient Reference Values 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 that constitute healthy dietary patterns that prevent nutrient deficiency, reduce risk of chronic disease, and are culturally acceptable, socially equitable and environmentally sustainable. This document updates *The Core Food Groups (1995)*.

The Food Modelling System⁹ also informed 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 D Questions for the literature review to underpin the revision of the dietary guidelines

Systematic literature review questions

1. Does the evidence suggest a particular maximum and/or minimum level of consumption of specific foods including:
 - fruit
 - vegetables
 - meat
 - dairy (cheese, milks and yoghurt)
 - cereals/grains
 - legumes
 - nuts and seeds
 - fish
 - poultry
 - eggs
 - fat/oil
 - salt/sodium
 - sugars
 - beverages (including water)
 - alcohol

is beneficial/detrimental in respect to:

- chronic diseases including:
 - obesity
 - cardiovascular disease
 - stroke
 - diabetes
 - cancer
 - hypertension
 - eye-health
 - bone health
 - dental health
 - mental health
- environmental impacts
- social equity
- health and well being (DALY/QALYs)?

Note: Need to consider serve sizes for different age/sex groups in current guidelines (infants, children, adolescents, adults, older Australians) and pregnant and breastfeeding women.

-
2. Which dietary patterns, foods, drinks and food components are associated with minimal green house gas emissions, minimal water use and maximum biodiversity?
-
3. What factors lead to children adopting appropriate life course food consumption and dietary patterns? For example body image and family meals.
-
4. What are the economic, physical and psychosocial barriers and the enablers to different population groups achieving diets consistent with the dietary guidelines?
-

5.	<p>What is the most appropriate age to introduce solid foods to infants (e.g. 4 months vs 6 months)?</p> <p>Does the introduction of solid foods at different ages change the risk of developing allergic syndromes (e.g. eczema, wheezing, GIT symptoms)?</p> <ul style="list-style-type: none"> • Which solids? • How many different foods? • Method of introduction – one at a time, length of time for one food, before another introduced, mixed products? • Method of preparation of solids (e.g. cooked, mixed with breastmilk, formula or cow's milk) <p>Does the earlier introduction of solids result in:</p> <ul style="list-style-type: none"> • decreased breastmilk production? • increased risk of overweight and/or obesity? • increased morbidity and mortality, short term and long term?
6.	<p>Is there a dose response between consuming red meat (not including processed meat as red meat) and an increased risk of cancer?</p>
7.	<p>Is there a dose response of sucrose or other refined sugars in foods or beverages on body weight indices over the long term (1 year+)?</p>
8.	<p>What is the effect of sugar-containing beverages on total energy intake and dental health in the diet?</p> <p>Is there a difference between fruit juice, flavoured milk and other beverages?</p>
9.	<p>What are the health benefits of grain-based foods (such as bread, breakfast cereals, oats, pasta and rice) in both refined and wholegrain forms? (comparison of refined vs wholegrain forms)</p>
10.	<p>Is there an association between specific fruits and vegetables or subgroups of these, for example <i>Brassica</i> vegetables, tomatoes, cruciferous and citrus etc, and:</p> <ul style="list-style-type: none"> • obesity • cardiovascular disease • stroke • diabetes • cancer • hypertension • eye-health • bone health • dental health • mental health?
11.	<p>How does greenhouse gas emissions, water scarcity and loss of biodiversity change capacity to access a balanced, varied and sufficient diet?</p>
12.	<p>Is there a dose response between the glycaemic index and glycaemic load of the diet and development of obesity and insulin resistance?</p>
13.	<p>Do children with iron deficiency have higher incidence of cognitive deficits?</p>
14.	<p>Is there a safe sun exposure which will improve vitamin D status in the general population, the elderly and children?</p>
15.	<p>What are the most appropriate indices and references to be used for the assessment of body weight, growth rates and obesity in children in Australia?</p>
16.	<p>What is the dose response between different types of milk intake and weight change in adults?</p>

Umbrella review questions

1. What dietary patterns, food groups including:

- fruit
- vegetables
- meat
- dairy (cheese, milks and yoghurt)
- cereals/grains
- legumes
- nuts and seeds
- fish
- poultry
- eggs
- fat/oil
- salt/sodium
- sugars
- beverages (including water)
 - alcohol

whole foods and food components are associated with:

- health
- longevity
- reduced risk of chronic diseases including:
 - obesity
 - cardiovascular disease
 - stroke
 - diabetes
 - cancer
 - hypertension
 - eye health
 - bone health
 - dental health
 - mental health

in the general population and vulnerable groups including low socioeconomic status, Aboriginal and Torres Strait Islander peoples and culturally and linguistically diverse groups, and those living in rural and remote areas, without serious disease?

2. What is the inter-relationship between dietary patterns, foods, drinks and food components and environmental sustainability?

3. What are the most recent data on dietary patterns and intakes of foods and food components (including nutrients) in Australia, and how does the data vary across age/sex groups in the general population and vulnerable groups including low socio economic status, Aboriginal and Torres Strait Islander peoples and culturally and linguistically diverse groups, and those living in rural and remote areas?

4. Is there a significant health advantage in balancing food intake and physical activity to maintain a healthy weight, to maintain muscle strength and a healthy body weight in older Australians, and in children to support normal growth and healthy development?

5. What are the benefits of breastfeeding and the risks of not breastfeeding (any and exclusive), to infants and mothers, both in the short term and long term?

6. What nutritional factors are important in optimising pregnancy outcomes?

7. What nutritional factors are important in optimising breastfeeding outcomes?

8. How does the processing and preparation of food, including:
- frozen/ canned/ dried/ juice; and
 - cooking methods, for example boiling, stir frying, roasting, microwaving, steaming etc;
- change bioavailability/nutritional value, food safety and environmental impact?
-

Narrative review questions

1. What current and past national food selection guides are used/have been used in Australia?
-
2. What are the major national food selection guides currently used internationally?
-
3. What methods have been used to develop national food selection guides?
-

Appendix E Summary of evidence statements of negative associations and those of Grade D

A number of relationships between food consumption and disease outcomes have not been considered or presented in these 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, is provided below.

E1 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 are included in Table E1.

Please note that these evidence statements are discussed in further detail within the body of the document.

Table E1: 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
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
Lean meats and poultry, fish, eggs, tofu, nuts and seeds and legumes/beans	
Consumption of red meat one to six times per week (or an intake range of 14–70 g/ 1,000 calories/d) is not associated with risk of bladder cancer.	C
Consumption of red meat, irrespective of frequency or serve size, is not associated with risk of prostate cancer	C
Consumption of 30–200g of red meat per day is not associated with risk of pancreatic cancer.	C
Consumption of at least one 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 (65–110g / day) does not lead to weight gain in the short-term.	C

Evidence statement	Grade
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 one serve of dairy food per day is not associated with the risk of breast cancer.	C
Consumption of less than one serve of milk per day during adult life is not associated with risk of osteoporotic or hip fracture.	C
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

E2 Grade D evidence statements that did not inform these Guidelines

In the systematic literature review (Evidence Report), a number of food, diet, and health relationships were examined for which the evidence of an association was Grade D. This was because the evidence was limited, inconclusive or contradictory. These Grade D relationships were not used to inform the development of the *Australian Dietary Guidelines*. Table E2 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. Grade D 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 E2: 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 (Evidence Report, Section 2.5).	D
The effect of total vegetable consumption on the risk of breast cancer is inconclusive (Evidence Report, Section 2.6).	D
The effect of total vegetable consumption on the risk of colorectal cancer is inconclusive (Evidence Report, Section 2.8).	D
The effect of total vegetable consumption on the risk of lung cancer is inconclusive (Evidence Report, Section 2.7).	D

Evidence statement	Grade
Fruit	
The effect of fruit consumption on the risk of oesophageal cancer is inconclusive (Evidence Report, Section 1.9).	D
The effect of fruit consumption on the risk of gastric cancer is inconclusive (Evidence Report, Section 1.5).	D
There is limited evidence that fruit consumption reduces the risk of lung cancer (Evidence Report, Section 1.7).	D
The effect of citrus fruit consumption on the risk of pancreatic cancer is inconclusive (Evidence Report, Section 1.13).	D
Grain (cereal) foods	
The effect of grain (cereal) consumption on the risk of cancer is inconclusive (Evidence Report, Section 6.1).	D
Lean meats and poultry, fish, eggs, tofu, nuts and seeds and legumes/beans	
The consumption of unprocessed red meat is associated with an increased risk of lung cancer (Evidence Report, Section 4.5).	D
There is limited evidence showing no association between the consumption of red meat and the risk of breast cancer (Evidence Report, Section 4.4).	D
The effect of consuming poultry at least once a week on the risk of breast cancer is inconclusive (Evidence Report, Section 10.1).	D
The effect of consuming poultry at least once a week on the risk of colorectal cancer is inconclusive (Evidence Report, Section 10.2).	D
The effect of fish consumption on the risk of breast cancer is inconclusive (Evidence Report, Section 9.6).	D
The effect of fish consumption on the risk of colorectal cancer is inconclusive (Evidence Report, Section 9.7).	D
The effect of fish consumption on the risk of prostate cancer is inconclusive (Evidence Report, Section 9.8).	D
The effect of fish consumption on the risk of renal cell cancer is inconclusive (Evidence Report, Section 9.9).	D
The effect of egg consumption on the risk of cancer is inconclusive (Evidence Report, Section 11.2).	D
Consumption of legumes (especially soy foods) is associated with a reduced risk of breast cancer (Evidence Report, Section 7.1).	D
There is unlikely to be a significant protective effect against prostate cancer from consuming soy foods (Evidence Report, Section 7.2).	D
Milk, yoghurt, cheese and/or alternatives	
The effect of milk consumption on the risk of prostate cancer is inconclusive (Evidence Report, Section 5.14).	D
Water	
The effect of coffee consumption on the risk of increased systolic blood pressure is inconclusive (Evidence Report, Section 15.14).	D
The effect of consuming four cups of coffee a day on the risk of gastric cancer is inconclusive (Evidence Report, Section 15.5).	D
Green and black tea consumption is not associated with an increased risk of breast cancer (Evidence Report, Sections 15.16, 15.17 and 15.22).	D
Consumption of coffee is associated with increased risk of bladder cancer (Evidence Report, Section 15.10).	D
Consumption of coffee is associated with increased risk of lung cancer (Evidence Report, Section 15.12).	D

Appendix F Evidence gradings used in the World Cancer Research Fund report

Below are excerpts on the evidence presented in the WCRF report; *Food, nutrition, physical activity and the prevention of cancer: A global perspective*.⁴³ Further information is available at www.dietandcancerreport.org/.

THE WCRF criteria for grading evidence are as follows.

Convincing

These criteria are for evidence strong enough to support a judgement of a convincing causal relationship, which justifies goals and recommendations designed to reduce the incidence of cancer.

A convincing relationship should be robust enough to be highly unlikely to be modified in the foreseeable future as new evidence accumulates. All of the following were generally required.

- Evidence from more than one study type.
- Evidence from at least two independent cohort studies.
- No substantial unexplained heterogeneity within or between study types or in different populations relating to the presence or absence of an association, or direction of effect.
- Good quality studies to exclude with confidence the possibility that the observed association results from random or systematic error, including confounding, measurement error, and selection bias.
- Presence of a plausible biological gradient ('dose response') in the association. Such a gradient need not be linear or even in the same direction across the different levels of exposure, so long as this can be explained plausibly.
- Strong and plausible experimental evidence, either from human studies or relevant animal models, that typical human exposures can lead to relevant cancer outcomes.

Probable

These criteria are for evidence strong enough to support a judgement of a probable causal relationship, which would generally justify goals and recommendations designed to reduce the incidence of cancer.

All the following were generally required.

- Evidence from at least two independent cohort studies, or at least five case control studies.
- No substantial unexplained heterogeneity between or within study types in the presence or absence of an association, or direction of effect.
- Good quality studies to exclude with confidence the possibility that the observed association results from random or systematic error, including confounding, measurement error, and selection bias.
- Evidence for biological plausibility.

Limited – suggestive

These criteria are for evidence that is too limited to permit a probable or convincing causal judgement, but where there is evidence suggestive of a direction of effect. The evidence may have methodological flaws, or be limited in amount, but shows a generally consistent direction of effect. This almost always does not justify recommendations designed to reduce the incidence of cancer. Any exceptions to this require special explicit justification.

All the following were generally required.

- Evidence from at least two independent cohort studies or at least five case control studies.
- The direction of effect is generally consistent though some unexplained heterogeneity may be present.
- Evidence for biological plausibility.

Limited – no conclusion

Evidence is so limited that no firm conclusion can be made. This category represents an entry level, and is intended to allow any exposure for which there are sufficient data to warrant Panel consideration, but where insufficient evidence exists to permit a more definitive grading. This does not necessarily mean a limited quantity of evidence. A body of evidence for a particular exposure might be graded 'limited – no conclusion' for a number of reasons. The evidence might be limited by the amount of evidence in terms of the number of studies available, by inconsistency of direction of effect, by poor quality of studies (e.g. lack of adjustment for known confounders), or by any combination of these factors.

When an exposure is graded 'limited – no conclusion', this does not necessarily indicate that the Panel has judged that there is evidence of no relationship. With further good quality research, any exposure graded in this way might in the future be shown to increase or decrease the risk of cancer. Where there is sufficient evidence to give confidence that an exposure is unlikely to have an effect on cancer risk, this exposure will be judged 'substantial effect on risk unlikely'.

There are also many exposures for which there is such limited evidence that no judgement is possible. In these cases, evidence is recorded in the full systematic literature review reports contained on the CD included with this Report. However, such evidence is usually not included in the summaries and is not included in the matrices in this printed Report.

Substantial effect on risk unlikely

Evidence is strong enough to support a judgement that a particular food, nutrition, or physical activity exposure is unlikely to have a substantial causal relation to a cancer outcome. The evidence should be robust enough to be unlikely to be modified in the foreseeable future as new evidence accumulates.

All of the following were generally required.

- Evidence from more than one study type.
- Evidence from at least two independent cohort studies.
- Summary estimate of effect close to 1.0 for comparison of high versus low exposure categories.
- No substantial unexplained heterogeneity within or between study types or in different populations.
- Good quality studies to exclude, with confidence, the possibility that the absence of an observed association results from random or systematic error, including inadequate power, imprecision or error in exposure measurement, inadequate range of exposure, confounding, and selection bias.
- Absence of a demonstrable biological gradient ('dose response').
- Absence of strong and plausible experimental evidence, either from human studies or relevant animal models, that typical human exposures lead to relevant cancer outcomes.
- Factors that might misleadingly imply an absence of effect include imprecision of the exposure assessment, an insufficient range of exposure in the study population, and inadequate statistical power. Defects in these and other study design attributes might lead to a false conclusion of no effect.
- The presence of a plausible, relevant biological mechanism does not necessarily rule out a judgement of 'substantial effect on risk unlikely'. But the presence of robust evidence from appropriate animal models or in humans that a specific mechanism exists, or that typical exposures can lead to cancer outcomes, argues against such a judgement.
- Because of the uncertainty inherent in concluding that an exposure has no effect on risk, the criteria used to judge an exposure 'substantial effect on risk unlikely' are roughly equivalent to the criteria used with at least a 'probable' level of confidence. Conclusions of 'substantial effect on risk unlikely' with a lower confidence than this would not be helpful, and could overlap with judgements of 'limited – suggestive' or 'limited – no conclusion'.

Special upgrading factors

These are factors that form part of the assessment of the evidence that, when present, can upgrade the judgement reached. So an exposure that might be deemed a 'limited – suggestive' causal factor in the absence, say, of a biological gradient, might be upgraded to 'probable' in its presence. The application of these factors (listed below) requires judgement, and the way in which these judgements affect the final conclusion in the matrix are stated.

- Presence of a plausible biological gradient ('dose response') in the association. Such a gradient need not be linear or even in the same direction across the different levels of exposure, so long as this can be explained plausibly.
- A particularly large summary effect size (an odds ratio or relative risk of 2.0 or more, depending on the unit of exposure) after appropriate control for confounders.
- Evidence from randomised trials in humans.
- Evidence from appropriately controlled experiments demonstrating one or more plausible and specific mechanisms actually operating in humans.
- Robust and reproducible evidence from experimental studies in appropriate animal models showing that typical human exposures can lead to relevant cancer outcomes.

Appendix G Food, nutrition and environmental sustainability

G1 Key messages

Overconsumption is unsustainable

- *Avoid overconsumption*—Overconsumption of foods and drinks involves greater use of natural resources and puts more pressure on the environment, including increased disposal of waste food and packaging.^{1040,1043}
- *Maintain a healthy weight*—It is estimated that an overweight population has a greater environmental impact than a normal weight population because they have a higher food (energy) intake – 19% more food energy is required for a population with an obesity prevalence of 40% than for a population with an obesity prevalence of 3%.¹⁰⁴⁴
- *Choose foods for health and sustainability*—Dietary patterns in line with the recommendations in these Guidelines – eating nutrient-dense foods and limiting consumption of discretionary foods high in saturated fat, added sugars and added salt – provide health benefits and reduce the environmental impact associated with foods.^{1053,1045,1046}
- *Plan meals and shopping*—Planning meals and food purchases and moderating the size of food portions can assist with avoiding overconsumption. Pre-shop planning, including checking cupboards, refrigerators and freezers before shopping¹⁰⁴⁷ is economical and reduces food waste from spoilage.^{1040,1046}
- *Conserve water and energy*—If individuals seek further advice on sustainable food preparation, health professionals may encourage conserving water and the appropriate use and maintenance of energy-efficient appliances, and provide practical tips on reducing the use of natural resources when preparing foods (e.g. only using the oven when more than one item requires this cooking method).^{1047,1049}

Food wastage and food safety

Store foods appropriately—Decreasing food waste can substantially reduce the environmental impact of food and has financial benefits for households.^{1050,1051} Food wastage (not including packaging) accounts for about 10% of food purchased.¹⁰⁵² The appropriate storage of foods avoids the unnecessary use and degradation of natural resources.¹⁰⁴⁷

Dispose of food waste appropriately—Most household food waste that enters landfills can be composted or mulched. The use of composting bins, worm farms or chicken runs promotes the recycling of nutrients back into the home garden.¹⁰⁴⁰

Keep food safely—Correct handling of food during all stages of its preparation and storage, including temperature control, is critical in ensuring food safety and preventing wastage.¹⁰⁴⁷ 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.⁹⁵³ Date marking provides a guide on the shelf life of food in terms of quality and safety.⁹⁴³

- use-by date indicates how long a food can be expected to remain safe, provided it has been stored according to any stated storage conditions and the package is unopened
- best-before date indicates the length of time a food should keep before perceptible changes in quality occur.

Select foods with appropriate packaging and recycle—Appropriate packaging protects food products through transit and storage, thereby reducing food waste. Packaging that is more than is required to preserve food and ensure food safety places higher demands on natural resources.^{1042,1047} Drinking tap water rather than bottled water decreases production and disposal of plastic bottles. Choosing biodegradable or recyclable packaging reduces the amount of waste entering landfill.

Eating seasonally

Eat fruit and vegetables that are currently growing in the given climate—This can assist with lessening pressure on the food supply by potentially reducing processing, distribution and storage.¹⁰⁴⁰ However, consumers may require advice from health professionals as to which fruits or vegetables are in season and also the challenges associated with avoiding 'year-round' produce.¹⁰⁵³

Focus on nutritional value—Some fruit and vegetables may look imperfect but are still nutritionally valuable.¹⁰⁵⁴

G2 Background

These Guidelines have a firm evidence base and a primary focus on meeting population nutritional requirements, this appendix may assist health professionals to discuss the complex issue of food, nutrition and environmental sustainability with interested individuals. The aim would be to encourage people to review their dietary patterns with a primary focus on improving their health, while allowing them to consider ways to reduce environmental consequences.

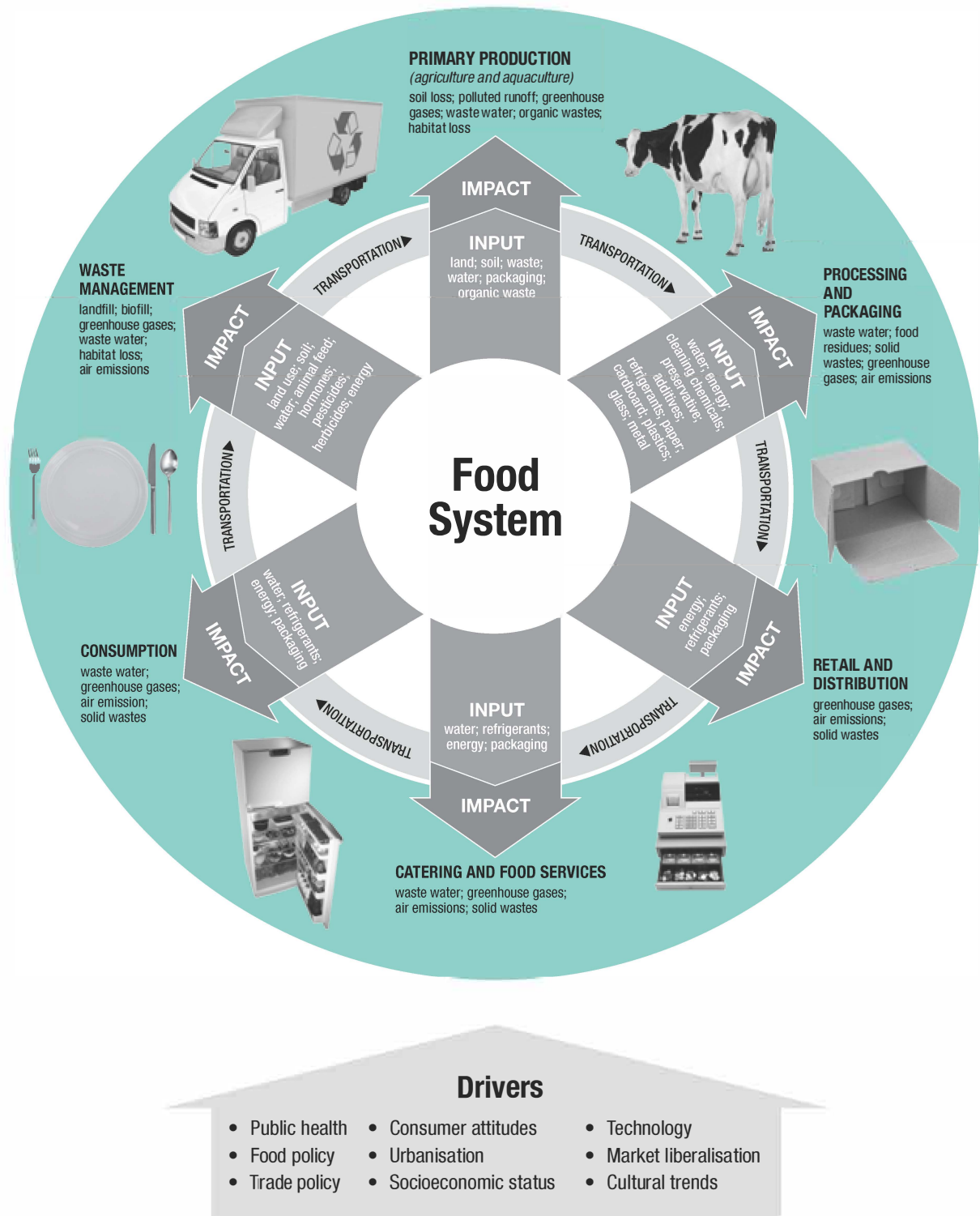
The production and consumption of food has a range of environmental consequences. The food system includes interdependent components that provide food for local consumption or export.¹⁰⁴⁰ It is a subset of the natural environment, and depends on and impacts on biodiversity – in Australia and globally.^{1051,1055} The food system encompasses numerous environmental inputs such as land, water and energy during the many stages from 'paddock to plate'. The resultant outputs may include greenhouse gases, waste water, land deterioration, packaging and food waste. Figure G1 gives examples of environmental consequences within the food system.

The concept of food consumption habits with lower environmental impact is not new but it is complex.¹⁰⁵⁶ In 2003, the joint Expert Panel of the World Health Organisation and the Food and Agricultural Organisation outlined basic recommendations for dietary patterns that are 'not only healthier but more favourable to the environment and sustainable development'.¹⁰⁴⁶ This integration of environment and health was also noted in the 2003 edition of the Dietary Guidelines for Australian Adults.³⁶ Since then, the body of evidence relating to the multifaceted relationship between food systems, sustainability and health has increased but there are still many gaps in our understanding of what this means within the Australian context.

In Australia, the Prime Minister's Science, Engineering and Innovation Council's 2010 report into food security emphasised the need to balance the imperative of feeding a growing population with maintaining environmental integrity.¹⁰⁵⁷ However, in easing the pressure on the food system, it is also important to balance nutritional requirements for health and the prevention of chronic disease.⁹⁷ This highlights the complex challenge faced by health professionals, where recommended dietary patterns that meet nutritional needs must also be sustainable — that is, equitable, affordable and considerate of environmental factors.^{1042,1056,1058}

Fostering a sustainable, globally competitive, resilient food supply that supports access to nutritious and affordable food is the primary aim of the National Food Plan.¹⁰⁵⁹ Appropriately, health should be considered in sustainable food systems, where the nutritional requirements of the population can be met without placing pressure on natural resources.^{97,1040,1042,1059} A range of concurrent approaches are required to achieve this, relating primarily to food production and food consumption.

Figure G1: Examples of environmental consequences within the food system



G3 Complexities of measuring the sustainability of the food supply

Historically, dietary guidance has been based on experimental evidence from nutritional science and epidemiology, with sustainability often referred to in the context of 'triple bottom line' considerations of economic, social and environmental factors.¹⁰⁵ Understanding the environmental implications of food choices involves considering new, often non-empirical evidence drawn from assessment of the relationship between the food system, food choices and environmental outcomes. This requires evidence from research in environmental science, agricultural science and economics. Government reports are also useful, especially in areas with policy implications, such as carbon accounting.

The food system comprises many stages from inputs on-farm to waste management following consumer use. As shown in Figure G1, these include agriculture and aquaculture, processing, manufacturing, transport, retail, preparation, consumption and waste management. The complexity of the system challenges the development of standardised methodologies for measuring the environmental consequences of producing particular foods.¹⁰⁶ As well, some current analyses do not consider the process through to the consumer and the subsequent disposal of waste.

The environmental consequences of food production depend on the agricultural system used and the particular environmental aspect examined, as these differ in impact, with implications for yield, quality and affordability.¹⁰⁷ Given this, the consequences cannot be assessed solely on a commodity basis but are dependent on where and how the product is grown. For example, it is estimated that in NSW, the amount of water used in beef cattle production ranges broadly from 3.3 L to 221 L H₂Oe kg⁻¹ live weight at farm gate.¹⁰⁸ As well, it is important to ensure that focussing on one outcome (e.g. emissions) does not have an unfavourable effect on another (e.g. soil degradation) and consequently add further pressure to the food system.

New technologies will play an important role in developing sustainable food systems, but the extent depends on consumer acceptance and economic constraints.¹⁰⁹ There is a need for more Australian data on many components of the food system and further research and development is required.

Two methodologies – process life cycle analysis (Process-LCA) and input-output analysis – are increasingly used to critically analyse the environmental impact of various components of the food system:

- Process-LCA considers the environmental impact of all processes involved in bringing a product to the consumer, including the way it is used and the impact of disposing packaging and waste. LCA methodologies are highly standardised, however the complexity of analysing sustainability at the food supply level means that many different questions are asked and models used which can limit meaningful comparisons.¹¹⁰ Additionally, to obtain an overall perspective, the same process must be completed across the entire life cycle. LCA methodologies are complex, time consuming and expensive, which inhibits their use for data collection.
- Input-output modelling is a useful tool for assessing the environmental impact of consumption. It was originally developed for economic analysis, but has been applied to environmental analysis since the late 1960s. It can be used to assess environmental indicators such as land disturbance, water and energy use, and total indicator intensities (the total amount of an indicator required to produce and deliver a value unit of a particular commodity).¹¹¹ This can be used in decision making to predict and forecast the impact of future performance of an economy and the effect of changes to relevant systems.

In developing an evidence-base for sustainable food choices, different methodological approaches limit the comparability of findings. For example, scope and definitions vary according to the stage of the food system being considered. Some studies incorporate the environmental impact of manufacturing on-farm machinery and the production of all other on-farm inputs such as fertiliser, while others include only fertiliser production. Much of the environmental impact evidence for specific foods is based on primary production and on-farm effects, with less information on aspects of the food supply chain such as processing, distribution, retail, consumption and waste management. However, the environmental impact of the life cycle of a particular food cannot be considered only as a characteristic of the food, but of all the specific and local aspects of its production, movement, storage, preparation and ultimate fate.

While overseas studies are not always generalisable to the Australian context, the body of Australian evidence is steadily growing, and the interest in this field may serve to stimulate more research. Despite the above challenges, enough evidence exists to suggest pragmatic guiding principles on how dietary patterns and home practices can contribute to reducing the environmental consequences of the food system.

G4 Australia's progress toward a sustainable food system

A systems approach to food consumption enables consideration of the many factors involved in getting food from farm to consumer, as well as the implications for health. In recognition of the complex relationship between sustainable food production and consumption, improved environmental practices are being applied to existing components of the food system. These initiatives mainly focus on resource efficiencies (e.g. reducing energy, water and land usage). However, as previously mentioned, this involves interaction and consideration for the entire food supply to ensure any changes are not counterproductive for other areas.¹⁰⁴⁰

Consumers and producers are the primary drivers for the direction of the food system. With increasing consumer demand for sustainably produced foods, many producers are changing what they produce and how they produce it.¹⁰⁴⁰ These changes are generally well supported by industry, with many implementing initiatives that encourage sustainable production practices.¹⁰⁶³⁻¹⁰⁶⁵ Industry is also responsible for funding much of the current research in this area, with millions of dollars invested annually into research and development activities leading to more sustainable practices while also ensuring economic and social viability.

The Australian and other governments are also active participants in planning for and managing a sustainable food system. Australia is committed to reducing threats to biodiversity, through nationally agreed strategies such as Australia's Biodiversity Conservation Strategy 2010-2030 and through internationally agreed conventions such as the United Nations' Convention on Biological Diversity.¹⁰⁶⁶ These impacts on biodiversity are key to any approach to sustainable food consumption because conserving biodiversity is an essential part of safeguarding the Earth's biological support systems.^{1056,1066}

Relevant state, territory and national departments are also promoting policies, programs and regulations that foster and support ecologically sustainable development both broadly and at the food system level.^{1059,1067,1068} However, there is room for improvement. Strengthening these arrangements through the development of the National Food Plan will help Australia's food system respond to new opportunities and challenges.¹⁰⁵⁹

As the planning and management of a sustainable food system progresses at government and industry levels, it will be important to link the strong evidence on health and nutrition with the accruing evidence on the environmentally sustainable attributes of food consumption habits.

G5 Sustainability and food consumption

Health, food choices and environmental consequences can all be influenced by behaviour change. Changing food consumption habits can influence demand on the food system and consequently the pressure on natural resources and biodiversity.^{1042,1056,1059} Discussing sustainability in the context of consumption habits not only has the potential to improve population health but also supports the objective of achieving a sustainable food supply with improved food security.^{1042,1056,1059} While the current evidence is not yet strong enough to make recommendations about specific foods in terms of 'more sustainable choices', it is suggested that food intake that is aligned with better health outcomes, has a lower environmental impact overall compared to most current western diets.^{1042,1046,1056,1070-1073} The nutrient composition of foods is important and people should be encouraged to consume a wide variety of foods from the Five Food Groups to not only meet the nutrients required for health and wellbeing but also contribute to better environmental outcomes.^{8,1056}

Consumers can contribute to sustainability efforts through the dietary patterns they choose to follow.¹⁰⁵⁶ As the wealth of our nation has increased and the availability of food has shifted, the foods produced have also changed to meet consumer preferences.^{1056,1058} This has resulted in a move toward greater consumption of processed foods that are high in saturated fat, added sugars and added salt.^{1041,1046} It is suggested that changing consumer food choices can make a bigger difference to overall household sustainability than reducing water and energy use.¹⁰⁴⁰ Therefore, avoiding overconsumption of food and food wastage should form the primary areas of focus by health professionals in communicating sustainable food consumption habits.¹⁰⁵⁶

Concerns about, and interest in, both health and the environment will continue to shape our dietary patterns. The practical considerations in G1 aimed at improving sustainability are easily aligned with the recommendations in these Guidelines as they aim for people to eat only to their nutritional requirements.

Appendix H Assessing growth and healthy weight in infants, children and adolescents, and healthy weight in adults

H1 Assessing growth and healthy weight in infants, children and adolescents

Measuring and recording 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.

Measuring height and weight regularly is important. The most practical measures of growth status in childhood are comparisons with standard growth charts that show the normal ranges of height for age, weight for age and BMI, by sex as well as length for age and head circumference for age for infants aged 0–2 years. As growth is a dynamic process, several measurements are preferable when assessing infants and children. When only a single measurement of weight and/or height is available, care is needed in interpretation.

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)¹²²
- 2000 US Centers for Disease Control and Prevention (CDC) BMI curves (also recommended by WHO before 2006)¹⁷⁴
- WHO growth charts.¹⁷⁵⁻¹⁷⁷

The differences between these options have been reviewed.¹⁷⁸

Growth charts are not intended to be diagnostic but to contribute to the overall clinical impression of the child being measured. Generally, irrespective of the reference or standard used, if a child is growing normally, growth approximately follows one of the lines on the chart. The growth trajectory may increase or decrease in adolescence dependent upon timing of the adolescent growth spurt.

If the line of growth crosses a number of percentile lines or tends towards or crosses the 10th or 90th percentiles, the advice of health professionals should be sought.³⁵¹ In the first months of life it is normal for 5% of the population to fall below the 5th percentile, and this does not always indicate a problem.³⁵¹

Care should always be taken when both measuring and plotting growth patterns to minimise error associated with poor measurement technique or error in plotting.

It is important to note that the use of different methods of assessing overweight or obesity will give different results, so results cannot be directly compared. For that reason it is essential to state the criteria used when assessing the weight of an individual or population. This also applies for underweight, stunting or wasting level in a population.

In Australia, the 2006 WHO BMI charts and the 2000 US CDC charts are commonly used.

WHO growth charts

The WHO has developed and uses growth reference charts for:¹⁴¹⁻¹⁴³

- infants aged 0–2 years
- children from birth to age 5 years
- children from age 5–10 years
- children and adolescents aged 5–19 years.

For children younger than 5 years, tables and charts showing percentiles and Z-scores for BMI-for-age, weight-for-age, weight-for-length and head circumference 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.¹⁷⁶ For children and adolescents aged 5–19 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 more than two standard deviations below normal, and severe thinness as more than three standard deviations below normal.¹⁷⁵

It is suggested that by 2011, 125 countries representing 75% of the world's under-5 population had adopted the WHO growth standards and were at varying stages of implementation.¹⁰⁷⁹ This includes the US, Canada, UK and New Zealand. The WHO growth standards for 0–19 years were implemented in the Northern Territory in 2009 and Victoria implemented WHO charts for 0–2 years in 2011. In 2012, all Australian jurisdictions agreed to adopt the WHO 2006 growth charts as the standard for Australian children aged 0–2 years. They will be phased in by other states and territories for use at the primary health care level in child health records. Education and training materials to assist Australian health professionals in using the WHO Charts are being developed and will become available in 2013. Other specialised growth charts may still be used by clinicians for groups with particular needs or characteristics.

Care should be taken to consider the differences between charts when assessing the growth of infants. The new WHO growth standards (2006) are heavier than the CDC 2000 growth reference in the first 6 months of life but lighter after 6 months.

Table H1: Practical points in the use of growth reference charts in infants aged 0–2 years

-
- Health professionals should use a growth reference chart appropriate to the age and sex of the infant or child and be aware which chart they are using
-
- In using the WHO growth reference charts, it is important to note that the 2nd percentile corresponds approximately to the 5th percentile on the CDC growth chart for infants under 6 months and that lower positions on the growth reference chart are acceptable
-
- In determining whether breast milk production or infant formula consumption is adequate (or for infants fed formula the amount is excessive), growth trajectory is more important than position on the growth reference chart
-

CDC BMI curves

In Australia, the NHMRC Overweight and Obesity Guidelines¹²¹ suggest the use of the US CDC growth charts¹⁰⁴⁶ for monitoring growth in children and adolescents aged 2–18 years.^{35,1081} Overweight is categorised as between the 85th and 95th percentiles in the BMI charts and obesity as above the 95th percentile.

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 6 years of age.

BMI using international reference standards

BMI measurement is a low-cost, simple method that has been validated against more direct measures of adiposity.¹⁰⁸² In 1999 an expert committee, on behalf of the IOTF, recommended that BMI be used to assess adiposity in children and adolescents and that the adult cut-off points be used as a reference.^{1083,1084} However, normal BMI among children and adolescents changes with age, so choosing a single cut-off number is not possible.

Following on from this recommendation, an international reference for defining overweight and obesity in children and adolescents was 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/m² for overweight and 30 kg/m² for obesity.¹²² Designed for epidemiological use, the tables will allow international comparison of the prevalence of overweight and obesity, as well as assessment of trends in children.^{1074,1085-1087}

Other measurements

Waist circumference and waist:height ratio are becoming more common measures of child, adolescent and adult central adiposity, but there is little consensus regarding standard cut-off values to define overweight and obesity.^{130,1088-1093} A waist:height ratio equal to or less than half (ratio ≤ 0.5) may be useful in predicting cardiovascular risk in children.¹⁰⁹⁴⁻¹⁰⁹⁷

Skinfold measurement – an index of subcutaneous adipose tissue – is another method of defining of under- or over-nutrition.¹⁰⁹⁸ 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 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.¹⁰⁹⁹ The most accurate measure of body composition is provided by using doubly labelled water, but cost limits its use to research.

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 charts are available in both formats and a calculator is available on the US CDC website.¹⁰⁸⁰ BMI can be converted into a BMI Z-score using a BMI-for-age growth chart and the formula: Z-score = ((BMI/M)-1)/(LS), where M, L, and S are values selected from reference tables corresponding to the age of the child in months.³⁵

Differences between IOTF criteria and the new WHO growth standard

The IOTF uses cut-off points based on WHO adult BMI but extrapolated for use in children based on six international data sets. In comparison, the WHO growth standards are a standard rather than a reference. The standard is how children should grow as opposed to a reference which describes how they grew at a particular time and place. The WHO growth standards reflect the growth of children who were predominantly breastfed for the first 4–6 months of life and were breastfed until 12 months of age. In addition, the data for babies was taken from those who were single, term births and those whose mothers did not smoke. There are major differences in the percentage of children classified as obese or overweight according to the reference used, ranging from 5% to 25% in a study of 3-year old children.¹⁰⁷⁸ The study 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'.¹⁰⁷⁸

H2 How is healthy weight measured in adults?

For adults, the standard WHO BMI cut-off points are most commonly used for assessment of obesity. BMI is used to define weight status in adults (except older adults) as follows.

Table H2: WHO body mass index classification

Classification (WHO)	BMI (kg/m ²)
Underweight	< 18.5
Healthy weight	18.5 – 24.9
Overweight	25.0– 29.9
Obese class I	30.0– 34.9
Obese class II	35.0 – 39.9
Obese class III	> 40.0

However, this classification may not be suitable for all population groups.^{118,119}

- some groups may have equivalent levels of risk at a lower BMI (e.g. people of Asian origin) or higher BMI (e.g. people of Polynesian origin including Torres Strait Islanders and Māori)¹²⁰
- while specific BMI ranges have not been developed, Aboriginal people have a relatively high limb to trunk ratio and may have equivalent levels of risk at a lower BMI
- a higher BMI range may be desirable for people aged over 70 years.

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 assessing risk of chronic disease in adults, and provides a better estimate of risk than BMI.^{1100,1101} Risk is increased at ≥80 cm and high at ≥88 cm for women and increased at ≥94 cm and high at ≥102 cm for men.^{121,169} As with BMI, thresholds for other ethnic groups may differ from those for people of European descent.

Appendix I 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 in 2010 by the Australian Department of Health and Ageing.²⁹³

A review of Australia's physical activity guidelines will be completed in 2013. Refer to www.health.gov.au for more information on current physical activity guidelines.

Physical activity recommendations for children 0–5 years

The recommendations for children aged 0–5 years state that being physically active every day is important for the healthy growth and development of infants, toddlers and preschoolers.²⁹³

For infants (0–1 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 (1–3 years) and preschoolers (3–5 years) should be physically active every day for at least 3 hours, spread throughout the day.

- Young children don't need to do their 3 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 2–5 years of age, these activities should be limited to less than 1 hour per day.

- Television, DVDs and playing computer games usually involve sitting for long periods – time that 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 1 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 is available on the Department of Health and Ageing website.¹¹⁰²

Physical activity recommendations for 5–12 year olds

The recommendations for children aged 5–12 years²⁹³ 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 2 hours a day using electronic media for entertainment (e.g. computer games, TV, internet), particularly during daylight hours.

Physical activity recommendations for 12–18 year olds

The recommendations for children aged 12–18 years²⁹ 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 3–4 days a week
- children shouldn’t spend more than 2 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.

Physical activity guidelines for Australian adults

The guidelines for adults²⁹ 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 the recommended 30 minutes (or more) of moderate intensity physical activity throughout the day, may be accumulated by combining short bouts of around 10 to 15 minutes each. These accumulated short bouts of physical activity are as effective as continuous activity at improving indicators of health such as hypertension and blood cholesterol.¹¹⁰³ However, this level of activity appears to be insufficient for preventing weight gain or weight loss or weight regain in most people.^{276,1104}

Physical activity recommendations for older Australians

The recommendations for older Australians²⁹ 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 J Studies examining the health effects of intake of fruit and vegetables together

The literature includes studies that 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.

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; Evidence Report, Section 3.1).^{362-364,367,1105}

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; Evidence Report, Section 3.2).^{362,363} 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 77 g),³⁶³ and consuming each additional serve of fruit and vegetables (of average serve size 106 g) reduced the risk of stroke by 5%.³⁶² 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 serves of vegetables and fruits per day'.¹⁵²

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.¹⁵² 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.¹⁵²

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; Evidence Report, Section 3.5).¹¹⁰⁶⁻¹¹⁰⁸

Colorectal cancer: Recent evidence suggests that there is no association between consumption of fruit and vegetables together and risk of colorectal cancer (Grade C; Evidence Report, Section 3.4).^{323,410} 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.¹¹⁰⁹ 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.¹¹¹⁰

Ovarian cancer: It is probable that there is no association between consumption of fruit and vegetables and risk of ovarian cancer (Grade B; Evidence Report, Section 3.6).^{324,325} A more recent meta-analysis has described evidence of a probable inverse relationship between consumption of vegetables and fruit, and bladder cancer.¹¹¹¹ 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.¹¹¹²⁻¹¹¹⁴

Epithelial cancer: In general, comparison of the results of systematic reviews of the evidence on diet and cancer sponsored by the WCRF in 1997¹¹¹⁵ and 2007⁴³ 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 IARC report.¹¹¹⁶

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.¹¹⁰⁹ 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.¹¹¹⁷

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 tobacco¹¹¹⁸ and alcohol.⁴¹⁹

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 hypertension¹¹¹⁹
- decreased risk of dementia⁵¹⁷
- decreased risk of head and neck cancer¹¹²⁰
- decreased risk of various upper digestive tract cancers^{45, 877, 1121-1123}
- decreased risk of prostate cancer^{437, 1124}
- decreased risk of obesity.¹¹²⁵⁻¹¹²⁶

Although not sufficient to develop evidence statements, individual studies found no evidence of an association between consumption of fruit and vegetables and pancreatic cancer.^{771, 1127, 1128}

Appendix K Alcohol and energy intake

Table K1: Median percentage of contribution of alcohol to energy intake per consumer

	Age groups (years)					
	16–18	19–24	25–44	45–64	≥65	≥19 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 1995.⁴⁸

Table K2: Energy and alcohol content of common alcoholic drink serves

Drink type	Serve size	Energy (kJ/serve)	Alcohol (g/serve)*	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#)	340 – 395	9.5	1
Spirits	1 nip (30ml)	255	8.8	1
Pre-mixed spirits	1 can (375ml)	1182	13.9	1½

Notes: * Using 1995 National Nutrition Survey data.⁴⁵

A glass of wine is usually more than 100ml, i.e. more than one standard drink (10g alcohol).

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 a recommended daily intake (RDI).

Adolescents: For the purposes of these Guidelines, an adolescent is someone aged 12–18 years. A marked increase in the rate of growth and development during adolescence increases the need for most nutrients including energy, protein, vitamins and minerals.

Allium vegetables: Vegetables derived from a bulbous plant having an onion odour, including garlic, leeks, shallots, chives and onions.

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/m² and obesity as a BMI of at least 30kg/m².

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.

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 17 kilojoules per gram.

Cereals: *See grain foods.*

Cereal fibre: Cereal fibre refers to dietary fibre obtained from core grain-based foods, including bread, breakfast cereals, rice and pasta.

Children: For the purposes of these Guidelines, children are defined as toddlers aged 1–3 years, preschoolers aged 3–5 years and primary school age 6–11 years. It is important for children to receive a nutritious diet that 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 cholesterol, 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 breast milk or infant formula when either becomes insufficient to satisfy an infant's nutritional requirements.

Complex carbohydrate: *See starch.*

Core food groups: This was a concept of the previous modelling system and included foods that formed the basis of a healthy diet, based on or developed with reference to recommended daily intakes (RDIs).

Cruciferous vegetables: *See Brassica vegetables.*

Dairy food: *See milks, yoghurts and cheeses.*

Dietary fibre: *See fibre.*

Discretionary foods: This includes foods and drinks not necessary to provide the nutrients the body needs, but that 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.

Eggs: Eggs are defined as containing a protective shell, albumen (egg white) and vitellus (egg yolk). Eggs are protein-rich foods and in the Guidelines they are classified as a meat alternative.

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 breast milk, which includes expressed breast milk 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.

Fermented milk: FSANZ defines fermented milk as a milk product obtained by fermentation of milk or products derived from milk, where the fermentation involves the action of micro-organisms and results in coagulation and a reduction in pH for example, yoghurt. Micro-organisms used in the fermentation of fermented milk must remain viable in the product.

Fibre: FSANZ defines fibre as the fraction of the edible parts of plants or their extracts, or synthetic analogues, that are resistant to digestion and absorption in the small bowel, usually with complete or partial fermentation in the large bowel. This includes polysaccharides, oligosaccharides and lignins, and promotes one or more of these beneficial physiological effects – laxation, reduction in blood cholesterol and modulation of blood glucose.

Fish: *See seafood.*

Flavoured milk: Sweetened flavoured milk provides nutrients but can be high in energy density (due to added flavours and added sugars). Plain milk is preferable.

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 that, in modern sedentary society, requires a reduction in serve 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 2006 Nutrient Reference Values. The diets were modelled to provide as close to 100% of the RDIs of ten 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 ten key nutrients.

Fruit: Fruit means the edible portion of a plant or constituents of the edible portion that are present in the typical proportion of the whole fruit (with or without the peel or water). 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: 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 people: For the purposes of these Guidelines, frail elderly people 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: Refers to the entire class of cereal/grain foods, including whole or partially processed cereal grains (e.g. 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.

HDL cholesterol: High-density lipoprotein (HDL) cholesterol assists in removing excess cholesterol out of cells, including cells in the arteries.

High quality diet: High quality diet refers to food patterns that closely align with national dietary guidelines with a diverse variety of healthy choices within the five food groups. This is usually aligned with protective dietary patterns.

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.

LDL cholesterol: Low-density lipoprotein (LDL) cholesterol is the main carrier of the cholesterol that is delivered into cells.

Legumes/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. The better known legumes include butter beans, haricot (navy) beans, red kidney beans, soybeans, mung beans, lentils, chick peas, snow peas, peanuts 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 (e.g. 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: Foods that 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 100ml of liquid.

Low salt food: For labelling purposes a low salt food is one with a sodium concentration of up to and including 120 mg per 100 g. The following are the conversion factors for the units used to express the sodium content of food:

1 mmol = 23mg

1 gram = 43 mmol

One gram of sodium chloride (NaCl) contains 17 mmol, or 391mg, 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 meat 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, tofu, legumes, nuts and nut pastes, and certain seeds, such as sunflower and sesame seeds.

Mediterranean dietary pattern: It is suggested that the Mediterranean diet is one of the healthiest dietary patterns in the world due to its relation with a low morbidity and mortality for some chronic diseases. The Mediterranean diet traditionally includes fruits, vegetables, nuts, pasta, rice and small amounts of meat. Grains in the Mediterranean region are typically wholegrain and bread is eaten plain or dipped in olive oil.

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 (MUFAs): In chemical terms, MUFAs contain one unsaturated bond. MUFAs occur in considerable amounts in olive oil, canola oil and many kinds of nuts.

Mostly: The term 'mostly' is derived from the Food Modelling System, where more than 50% of the food group was made up of a specific characteristic for example reduced fat varieties. This descriptor ensures that the variety of foods chosen not only meet nutrient needs but are also within individual energy requirements.

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]). Where possible, 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 these Guidelines.

Nuts and seeds: A nut is a simple dry fruit with one or two seeds in which the ovary wall becomes very hard (stony or woody) at maturity, and where the seed remains attached or fused with the ovary wall. Most nuts are indehiscent (not opening at maturity). Any large, oily kernel found within a shell and used in food may be regarded as a nut. Examples include almonds, pecans, walnuts, brazil nuts, cashew nuts, chestnuts, hazelnuts, macadamia nuts, pine nuts and pistachio nuts.

The term 'nut' is applied to many seeds that are not botanically true nuts. These may include cape seed, caraway, chia, flaxseed, linseed, passionfruit, poppy seed, pepita or pumpkin seed, sesame seed and sunflower seed.

Older adults: For the purposes of these Guidelines, older adults are defined as healthy people aged 65 years and over, not including frail elderly people.

Omega-3 long chain polyunsaturated fatty acids (LCPUFAs): Omega-3 LCPUFAs have the first double bond in the n-3 position. The best known are those in fatty fish, their names abbreviated to EPA (20:5 eicosapentaenoic acid) and DHA (22:6 docosahexaenoic acid). Another omega-3 LCPUFA, ALA (18:3 alpha-linolenic acid), occurs in considerable amounts in canola and flaxseed oils and in walnuts. Omega-3 LCPUFAs are suggested to be protective in cardiovascular health.

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 standard deviations 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) that causes the muscles to work and uses more energy than the person would use if resting.

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: This term is used judiciously to encourage increased consumption of a variety of vegetables (particularly non-starchy varieties).

Polyunsaturated fatty acids (PUFAs): PUFAs 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 PUFA is linoleic acid (18:2) whose first double bond is in the n-6 position. It occurs in seed oils including sunflower oil, safflower oil and corn oil. Smaller amounts of PUFAs with the first double bond 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 omega-3 PUFA, 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 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 these 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 same quantity of the reference food.

Refined grain (cereal) foods: Refers to highly processed grain (cereal) foods where the outer layer of the grain is lost during processing. These also include cereal or grain-based products with a significant amount of added fat and sugar, such as cakes, pastries, and biscuits.

Regularly: The term 'regularly' is used in discussions on weight management and growth. In this content, taking measurements such as weight or height every month provide a fair indication of change. Measurements taken more frequently than this are often not a true picture of your weight and/or growth.

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 2,300 mg per day. This is equivalent to about 6g of salt, or one and a half teaspoons.

Saturated fatty acids (SFAs): In chemical terms, SFAs contain no double bond – that is, they are fully saturated with hydrogen. Foods that predominantly comprise SFAs are usually solid at room temperature (e.g. butter, fat on meat). SFAs 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 SFAs palmitic (16:0), myristic (14:0) and lauric (12:0) predominate in the diet they tend to raise plasma cholesterol.

Seafood: Refers to seafood (including fish) that is safe for human consumption and is produced or traded commercially in Australia, including seafood exports and imports.

Simple carbohydrate: *See sugars.*

Sodium: *See salt.*

Solid foods: All foods other than liquids – includes semi-solid/pureed foods, finger foods and family foods.

Standard drink: A standard drink contains 10 grams of alcohol – equivalent to 12.5ml of alcohol.

Starch: FSANZ defines starch as a complex carbohydrate (polysaccharide) containing a mixture of two molecules: amylose and amylopectin. Starch is determined chromatographically after enzymatic treatment of a de-sugared extract of the food.

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 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.

Tofu: Tofu is made from soybeans, water and a coagulant, or curdling agent. Tofu is a protein-rich food and in these Guidelines it is classified as a meat alternative.

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 ULs and 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 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 (TFAs): TFAs 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 cooking margarines. Small amounts of trans-fatty acids occur naturally in meats and dairy foods.

Unsaturated fatty acid: *See monounsaturated fatty acids and polyunsaturated fatty acids.*

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 UL of intake, the potential risk of adverse effects increases.

Vegetables: Vegetable means the edible portion of a plant or constituents of the edible portion that are present in the typical proportion of the whole vegetable (with or without the peel or water). Examples include: leafy green vegetables (spinach, lettuce, silverbeet and bok choy), members of the crucifer or *Brassica* family (broccoli, cabbage, and brussel sprouts), starchy root and tuber vegetables (yams and potatoes), and edible plant stems (celery and asparagus), gourd vegetables (pumpkin, squash and cucumber), *Allium* vegetables (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.

Whole foods: This refers to foods themselves for example fruit, vegetables, bread, pasta, lean meat, milk, yoghurt and not the food component for example calcium, iron, protein.

Acronyms and abbreviations

ADHD	attention deficit hyperactivity disorder
AIDS	acquired immunodeficiency syndrome
ALA	alpha-linolenic acid
AMDR	Acceptable Macronutrient Distribution Ranges
AUSNUT	Australian Food and Nutrient Database
BFHI	Baby Friendly Hospital Initiative
BMI	body mass index
CDC	Centers for Disease Control and Prevention (US)
CEO	Chief Executive Officer
CSIRO	Commonwealth Scientific and Industrial Research Organisation
DAA	Dietitians Association of Australia
DALY	disability-adjusted life years
DHA	docosahexaenoic acid
DoHA	Department of Health and Ageing
DPA	docosapentaenoic acid
EAR	Estimated Average Requirement
EPA	eicosapentaenoic acid
EPIC	European Prospective Investigation into Cancer and Nutrition
FAO	Food and Agriculture Organisation of the United Nations
FSANZ	Food Standards Australia New Zealand
GI	glycaemic index
GP	general practitioner
HDL	high-density lipoprotein
HIV	human immunodeficiency virus
IARC	International Agency for Research on Cancer
IOTF	International Obesity Task Force
LCA	life cycle analysis
LCPUFA	long chain polyunsaturated fatty acid
LDL	low-density lipoprotein
MAIF	Marketing in Australia of Infant Formula

MUFA	monounsaturated fatty acid
NATSINSAP	National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan
NHANES	National Health and Nutrition Examination Survey
NHMRC	National Health and Medical Research Council
NRV	nutrient reference value
NUTTAB	Nutrient Tables for Use in Australia
PMSEIC	Prime Minister's Science, Engineering and Innovation Council
PUFA	polyunsaturated fatty acid
RCT	randomised controlled trial
RDI	recommended dietary intake
SDT	Suggested Dietary Target
SEIFA	socioeconomic indexes for areas
SFA	saturated fatty acid
SIDS	sudden infant death syndrome
TFA	trans-fatty acid
UK	United Kingdom
UL	Upper Level
US	United States
USDA	United States Department of Agriculture
WCRF	World Cancer Research Fund
WHO	World Health Organization

References

1. Rayner M, Scarborough P. The burden of food related ill health in the UK. *J Epidemiol Community Health* 2005;59(12):1054–7.
2. National Public Health Partnership. *Eat well Australia: an agenda for action for public health nutrition, 2000-2010*. Canberra: National Public Health Partnership; 2001.
3. Australian Institute of Health and Welfare. *Premature mortality from chronic disease*. AIHW Bulletin no. 84. Cat. no. AUS 133. Canberra: Australian Institute of Health and Welfare, 2010. <http://www.aihw.gov.au/publication-detail/?id=6442472466>
4. Crowley S, Antioch K, Carter R, Waters AM, Conway L, Mathers C. *The cost of diet-related disease in Australia: a discussion paper*. Canberra: Australian Institute of Health and Welfare, 1992.
5. Access Economics. *The growing cost of obesity in 2008: three years on*. Canberra: Diabetes Australia, Access Economics, 2008. Available from: <http://www.accesseconomics.com.au/publicationsreports/showreport.php?id=172>
6. National Preventative Health Taskforce. *Australia: the healthiest country by 2020 – National Preventative Health Strategy – the roadmap for action*. Canberra: Commonwealth of Australia; 2009a.
7. Lee AJ, Bailey AP, Yarmirr D, O’Dea K, Mathews JD. Survival tucker: Improved diet and health indicators in an Aboriginal community. *Aust J Public Health* 1994;18(3):277–85.
8. National Health and Medical Research Council, Australian Government Department of Health and Ageing, New Zealand Ministry of Health. *Nutrient reference values for Australia and New Zealand including recommended dietary intakes*. Canberra: Commonwealth of Australia; 2006.
9. National Health and Medical Research Council. *A modelling system to inform the revision of the Australian Guide to Healthy Eating*. Canberra: Commonwealth of Australia; 2011b.
10. Begg S, Vos T, Barker B, Stevenson C, Stanley L, Lopez AD. *The burden of disease and injury in Australia 2003*. Canberra: Australian Institute of Health and Welfare, 2007.
11. Australian Bureau of Statistics. *National Health Survey: Summary of Results, 2007-2008*. Cat. no. 4364.0. Canberra: Australian Bureau of Statistics, 2009. <http://www.abs.gov.au/ausstats/abs@.nsf/mf/4364.0/>
12. Commonwealth Scientific and Industrial Research Organisation, Preventative Health National Research Flagship, University of South Australia. *2007 Australian National Children’s Nutrition and Physical Activity Survey: main findings*. Canberra: Commonwealth of Australia, 2008. [http://www.health.gov.au/internet/main/publishing.nsf/content/66596E8FC68FD1A3CA2574D50027DB86/\\$File/childrens-nut-phys-survey.pdf](http://www.health.gov.au/internet/main/publishing.nsf/content/66596E8FC68FD1A3CA2574D50027DB86/$File/childrens-nut-phys-survey.pdf)
13. Buckland G, Bach A, Serra-Majem L. Obesity and the Mediterranean diet: a systematic review of observational and intervention studies. *Obes Rev* 2008;9(6):582–93.
14. Roman B, Carta L, Martinez-Gonzalez MA, Serra-Majem L. Effectiveness of the Mediterranean diet in the elderly. *Clin Interv Aging* 2008;3(1):97–109.
15. Serra-Majem L, Roman B, Estruch R. Scientific evidence of interventions using the Mediterranean diet: a systematic review. *Nutr Rev* 2006;64(2):S27.
16. Bamia C, Trichopoulos D, Ferrari P, Overvad K, Bjerregaard L, Tjønneland A 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):590–8.

17. Diehr P, Derleth A, Cai L, Newman AB. The effect of different public health interventions on longevity, morbidity, and years of healthy life. *BMC Public Health* 2007;7:52.
18. Kant AK, Graubard BI, Schatzkin A. Dietary patterns predict mortality in a national cohort: the National Health Interview Surveys, 1987 and 1992. *J Nutr* 2004;134(7):1793–9.
19. Togo P, Osler M, Sorensen TI, Heitmann BL. Food intake patterns and body mass index in observational studies. *Int J Obes Relat Metab Disord* 2001;25(12):1741–51.
20. Wirt A, Collins CE. Diet quality--what is it and does it matter? *Public Health Nutr* 2009;12(12):2473–92.
21. Anderson AL, Harris TB, Tyllavsky FA, Perry SE, Houston DK, Hue TF et al. Dietary patterns and survival of older adults. *J Am Diet Assoc* 2011;111(1):84–91.
22. de Groot CP, van Staveren WA. Nutritional concerns, health and survival in old age. *Biogerontol* 2010;11(5):597–602.
23. Scarborough P, Nnoaham KE, Clarke D, Capewell S, Rayner M. Modelling the impact of a healthy diet on cardiovascular disease and cancer mortality. *J Epidemiol Community Health* 2010.
24. Australian Institute of Health and Welfare. Australia's health 2010. Australia's health series no. 12. Cat. no. AUS 122. Canberra: Australian Institute of Health and Welfare, 2010. <http://www.aihw.gov.au/publication-detail/?id=6442468376>
25. Wilkinson R, Marmot M. Social determinants of health: The solid facts. Copenhagen: World Health Organization, 2003. Available from: http://www.euro.who.int/__data/assets/pdf_file/0005/98438/e81384.pdf
26. Marmot MG. The status syndrome: how social standing affects our health and longevity. New York: Henry Holt, 2004.
27. Friel S, Baker PL. Equity, food security and health equity in the Asia Pacific region. *Asia Pac J Clin Nutr* 2009;18(4):620–32.
28. National Health and Medical Research Council. Nutrition in Aboriginal and Torres Strait Islander peoples: an information paper. Canberra: Commonwealth of Australia, 2000.
29. Australian Bureau of Statistics, 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. Canberra: Commonwealth of Australia, 2005. <http://www.aihw.gov.au/publication-detail/?id=6442467754>
30. Australian Bureau of Statistics. National Aboriginal and Torres Strait Islander Health Survey 2004–2005. Canberra: Australian Bureau of Statistics, 2006. [http://www.ausstats.abs.gov.au/Ausstats/subscriber.nsf/0/B1BCF4E6DD320A0BCA25714C001822BC/\\$File/47150_2004-05.pdf](http://www.ausstats.abs.gov.au/Ausstats/subscriber.nsf/0/B1BCF4E6DD320A0BCA25714C001822BC/$File/47150_2004-05.pdf)
31. Commonwealth Department of Health and Family Services. Food and nutrition policy. 1992 (cited 29 March 2011). <http://www.health.gov.au/internet/main/publishing.nsf/content/phd-nutrition-fnp-1992>. <http://www.patonsyarns.com/pattern.ph>
32. National Health and Medical Research Council. How to review the evidence: systematic identification and review of the scientific literature. Canberra: Commonwealth of Australia, 2000a. <http://www.nhmrc.gov.au/publications/synopses/cp65syn.htm>
33. National Health and Medical Research Council. A review of the evidence to address targeted questions to inform the revision of the Australian dietary guidelines. Canberra: Commonwealth of Australia; 2011a.
34. National Health and Medical Research Council. Dietary Guidelines for Older Australians. Canberra: Commonwealth of Australia, 1999. <http://www.nhmrc.gov.au/publications/synopses/n23syn.htm>
35. National Health and Medical Research Council. Dietary Guidelines for Children and Adolescents in Australia. Canberra: Commonwealth of Australia, 2003a. <http://www.nhmrc.gov.au/publications/synopses/dietsyn.htm>
36. National Health and Medical Research Council. Dietary Guidelines for Australian Adults. Canberra: Commonwealth of Australia; 2003b.

37. National Health and Medical Research Council. Review: Nutritional requirements and dietary advice targeted for pregnant and breastfeeding women. Canberra: Commonwealth of Australia; 2013.
38. Williams P, Allman-Farrinelli M, Collins C, Gifford J, Byron A. A review of the evidence to address targeted questions to inform the revision of the Australian dietary guidelines 2009: Process Manual. Dietitians Association of Australia; 2011.
39. National Health and Medical Research Council. NHMRC additional levels of evidence and grades for recommendations for developers of guidelines. Canberra: Commonwealth of Australia; 2007.
40. National Health and Medical Research Council. How to use the evidence: assessment and application of scientific evidence. Canberra: Commonwealth of Australia, 2000b. <http://www.nhmrc.gov.au/publications/synopses/cp69syn.htm>
41. National Health and Medical Research Council. NHMRC standards and procedures for externally developed guidelines. Canberra: Commonwealth of Australia; 2007.
42. National Health and Medical Research Council. NHMRC levels of evidence and grades for recommendations for developers of guidelines. Canberra: Commonwealth of Australia; 2009.
43. World Cancer Research Fund, American Institute for Cancer Research. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Washington DC.: 2007. Available from: <http://www.dietandcancerreport.org/?p=er>
44. Ball K, Mishra GD, Thane CW, Hodge A. How well do Australian women comply with dietary guidelines? Public Health Nutr 2004;7(3):443–52.
45. Australian Bureau of Statistics. National Nutrition Survey: Foods eaten, Australia 1995. Canberra: Australian Bureau of Statistics, 1999. <http://www.abs.gov.au/AUSSTATS/abs@.nsf/0/9A125034802F94CECA2568A9001393CE?OpenDocument>
46. Commonwealth Department of Health and Family Services. The Australian guide to healthy eating. Canberra: Commonwealth of Australia, 1998. <http://www.health.gov.au/internet/main/publishing.nsf/content/health-pubhlth-publicat-document-fabrox-cnt.htm>
47. Rangan AM, Randall D, Hector DJ, Gill TP, Webb KL. Consumption of 'extra' foods by Australian children: types, quantities and contribution to energy and nutrient intakes. Eur J Clin Nutr 2008;62(3):356–64.
48. Australian Bureau of Statistics. National Nutrition Survey: Nutrient intakes and physical measurements, Australia 1995. Canberra: Australian Bureau of Statistics, 1998. <http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/95E87FE64B144FA3CA2568A9001393C0>
49. Burke V, Beilin LJ, Simmer K, Oddy WH, Blake KV, Doherty D 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):15–23.
50. Classen T, Hokayem C. Childhood influences on youth obesity. Econ Hum Biol 2005;3(2):165–87.
51. Dubois L, Farmer A, Girard M, Porcherie M. Family food insufficiency is related to overweight among preschoolers. Soc Sci Med 2006;63(6):1503–16.
52. Dubois L, Girard M. Early determinants of overweight at 4.5 years in a population-based longitudinal study. Int J Obesity 2006;30(4):610–7.
53. Field AE, Austin SB, Gillman MW, Rosner B, Rockett HR, Colditz GA. Snack food intake does not predict weight change among children and adolescents. Int J Obes Relat Metab Disord 2004;28(10):1210–6.
54. Hawkins SS, Cole TJ, Law C. 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):147–55.
55. Hesketh K, Carlin J, Wake M, Crawford D. Predictors of body mass index change in Australian primary school children. Int J Pediatr Obes 2009;4(1):45–53.

56. Hesketh K, Wake M, Waters E, Carlin J, Crawford D. Stability of body mass index in Australian children: a prospective cohort study across the middle childhood years. *Public Health Nutr* 2004;7(2):303–9.
57. Li L, Law C, Lo Conte R, Power C. Intergenerational influences on childhood body mass index: the effect of parental body mass index trajectories. *Am J Clin Nutr* 2009;89(2):551–7.
58. Mamun AA, Lawlor DA, O’Callaghan MJ, Williams GM, Najman JM. 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):475–82.
59. Power C, Li L, Manor O, Davey Smith G. 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):969–73.
60. Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Rogers I et al. Early life risk factors for obesity in childhood: cohort study. *Br Med J* 2005;330(7504):1357.
61. Salsberry PJ, Reagan PB. Taking the long view: the prenatal environment and early adolescent overweight. *Res Nurs Health* 2007;30(3):297–307.
62. Yang S, Lynch J, Schulenberg J, Roux AV, Raghunathan T. 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):468–77.
63. Erik Landhuis C, Poulton R, Welch D, Hancox RJ. Programming obesity and poor fitness: the long-term impact of childhood television. *Obesity (Silver Spring)* 2008;16(6):1457–9.
64. Danner FW. 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):1100–7.
65. Bhargava A, Jolliffe D, Howard LL. 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):438–44.
66. Henderson VR. Longitudinal associations between television viewing and body mass index among white and black girls. *J Adolesc Health* 2007;41(6):544–50.
67. Viner RM, Cole TJ. Television viewing in early childhood predicts adult body mass index. *J Pediatr* 2005;147(4):429–35.
68. Sturm R, Datar A. Body mass index in elementary school children, metropolitan area food prices and food outlet density. *Public Health* 2005;119(12):6059–68.
69. Baker E, Balistreri KS, Van Hook J. Maternal employment and overweight among Hispanic children of immigrants and children of natives. *J Immigr Minor Health* 2009;11(3):158–67.
70. Gorely T, Marshall SJ, Biddle SJ. Couch kids: correlates of television viewing among youth. *Int J Behav Med* 2004;11(3):152–63.
71. Epstein LH, Roemmich JN, Robinson JL, Paluch RA, Winiewicz DD, Fuerch JH 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):239–45.
72. Matijasevich A, Victora CG, Golding J, Barros FC, Menezes AM, Araujo CL et al. Socioeconomic position and overweight among adolescents: data from birth cohort studies in Brazil and the UK. *BMC Public Health* 2009;9:105.
73. Oliver LN, Hayes MV. Effects of neighbourhood income on reported body mass index: an eight year longitudinal study of Canadian children. *BMC Public Health* 2008;8:e16.
74. Poulton R, Caspi A, Milne BJ, Thomson WM, Taylor A, Sears MR et al. Association between children’s experience of socioeconomic disadvantage and adult health: a life-course study. *Lancet* 2002;360(9346):1640–5.

75. Taveras EM, Berkey CS, Rifas-Shiman SL, Ludwig DS, Rockett HR, Field AE 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):e512–24.
76. Viner RM, Cole TJ. 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):1362–74.
77. Bisset S, Gauvin L, Potvin L, Paradis G. 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):780–9.
78. Laitinen J, Pietilainen K, Wadsworth M, Sovio U, Jarvelin MR. Predictors of abdominal obesity among 31-year-old men and women born in Northern Finland in 1966. *Eur J Clin Nutr* 2004;58(1):180–90.
79. Sturm R, Datar A. Food prices and weight gain during elementary school: 5-year update. *Public Health* 2008;122(11):1140–3.
80. Goodman E, Whitaker RC. A prospective study of the role of depression in the development and persistence of adolescent obesity. *Pediatrics* 2002;110(3):497–504.
81. Hesketh K, Wake M, Waters E. 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):1233–7.
82. Klein EG, Lytle LA, Chen V. 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):1163–9.
83. Fidler JA, West R, Van Jaarsveld CH, Jarvis MJ, Wardle J. Does smoking in adolescence affect body mass index, waist or height? Findings from a longitudinal study. *Addiction* 2007;102(9):1493–501.
84. Gale CR, Batty GD, Deary IJ. 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):397–403.
85. Francis LA, Susman EJ. Self-regulation and rapid weight gain in children from age 3 to 12 years. *Arch Pediatr Adolesc Med* 2009;163(4):297–302.
86. Koch FS, Sepa A, Luvigsson J. Psychological stress and obesity. *J Pediatr* 2008;153(6):839–44.
87. Thomas C, Hypponen E, Power C. Obesity and type 2 diabetes risk in midadult life: the role of childhood adversity. *Pediatrics* 2008;121(5):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. Geneva: 2003. Available from: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=12768890
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 Canberra: Commonwealth of Australia; 2009b.
90. Schmidt M, Affenito SG, Striegel-Moore R, Khoury PR, Barton B, Crawford P 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):626–31.
91. Affenito SG, Thompson DR, Barton BA, Franko DL, Daniels SR, Obarzanek E 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):932–45.
92. Larson NI, Neumark-Sztainer D, Hannan PJ, Story M. Family meals during adolescence are associated with higher diet quality and healthful meal patterns during young adulthood. *J Am Diet Assoc* 2007;107(9):1502–10.

93. Australian Bureau of Statistics. Australian Health Survey. 2011 (cited 2011). <http://www.abs.gov.au/websitedbs/D3310114.nsf/Home/Australian+Health+Survey?OpenDocument> <http://www.patonsyarns.com/pattern.ph>
94. Mozaffarian D, Micha R, Wallace S. 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):e1000252.
95. Yngve A, Tseng M. Dietary guidelines and goal-setting. *Public health Nutrition* 2010;13(8):1149-50.
96. Holdsworth M. Sustainability should be integral to nutrition and dietetics. *Human Nutrition and Dietetics* 2010;23:467-68.
97. Kickbusch I. The Food System: a prism of present and future challenges for health promotion and sustainable development. Geneva 2010.
98. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation (WHO Technical Report Series No. 894). Geneva: World Health Organization, 2000. Available from: http://whqlibdoc.who.int/trs/WHO_TRS_894.pdf
99. Caballero B. The global epidemic of obesity: an overview. *Epidemiol Rev* 2007;29:1-5.
100. World Health Organization Expert Committee on Physical Status. Physical status: the use and interpretation of anthropometry. Geneva: World Health Organization, 1995. Available from: http://whqlibdoc.who.int/trs/WHO_TRS_854.pdf
101. World Health Organization. Global strategy on diet, physical activity and health. Geneva: World Health Organization, 2004. Available from: http://www.goforyourlife.vic.gov.au/hav/articles.nsf/pracpages/WHO_Global_Strategy_on_Diet_Physical_Activity_and_Health?OpenDocument
102. Willett WC, Dietz WH, Colditz GA. Guidelines for healthy weight. *N Engl J Med* 1999;341(6):427-34.
103. National Health and Medical Research Council. Acting on Australia's weight: A strategic plan for the prevention of overweight and obesity. Canberra: Commonwealth of Australia, 1997. http://www.nhmrc.gov.au/_files_nhmrc/file/publications/synopses/withdrawn/n21.pdf
104. World Health Organization. Global database on body mass index: An interactive surveillance tool for monitoring nutrition transition 2011 (cited 18 May 2011). <http://apps.who.int/bmi/index.jsp> <http://www.patonsyarns.com/pattern.ph>
105. Hocking S, Draper G, Somerford P, Xiao J, Weeramanthri T. The Western Australian Chief Health Officer's Report 2010. Perth: Department of Health, WA, 2010. http://www.publichealth.wa.gov.au/3/1045/1/chief_health_officers_report_.pm
106. Queensland Health. Risk factor impact on the burden of disease and injury in Queensland, 2007. Queensland Health, 2010.
107. Withrow D, Alter DA. The economic burden of obesity worldwide: a systematic review of the direct costs of obesity. *Obes Rev* 2011;12(2):131-41.
108. Medibank Private Ltd. Obesity in Australia: Financial impacts and cost benefits of intervention. Medibank Private Ltd., 2010. Available from: http://www.medibank.com.au/Client/Documents/Pdfs/Obesity_Report_2010.pdf
109. Colagiuri S, Lee CM, Colagiuri R, Magliano D, Shaw JE, Zimmet PZ et al. The cost of overweight and obesity in Australia. *Med J Aust* 2010;192(5):260-4.
110. Institute of Medicine. Bridging the evidence gap in obesity prevention: a framework to inform decision making. Washington DC: Institute of Medicine, 2010. Available from: <http://www.iom.edu/Reports/2010/Bridging-the-Evidence-Gap-in-Obesity-Prevention-A-Framework-to-Inform-Decision-Making.aspx>
111. Rokholm B, Baker JL, Sorensen TI. The levelling off of the obesity epidemic since the year 1999—a review of evidence and perspectives. *Obes Rev* 2010;11(12):835-46.

112. Olds TS, Tomkinson GR, Ferrar KE, Maher CA. Trends in the prevalence of childhood overweight and obesity in Australia between 1985 and 2008. *Int J Obesity* 2010;34(1):57–66.
113. Gill TP, Baur LA, Bauman AE, Steinbeck KS, Storlien LH, Fiatarone Singh MA et al. Childhood obesity in Australia remains a widespread health concern that warrants population-wide prevention programs. *Med J Aust* 2009;190(3):146–8.
114. Lobstein T, Baur L, Uauy R. Obesity in children and young people: a crisis in public health. *Obes Rev* 2004;5(Suppl 1):4–104.
115. Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, Brody J et al. A potential decline in life expectancy in the United States in the 21st century. *N Engl J Med* 2005;352(11):1138–45.
116. Haby M, Markwick A. Future prevalence of overweight and obesity in Australian children and adolescents, 2005–2025. Melbourne: Department of Human Services, 2008. http://www.health.vic.gov.au/healthstatus/downloads/future_overweight_prevalence_report.pdf
117. Australian Institute of Health and Welfare. Australia's health 2008. Cat. no. AUS 99. Canberra: Australian Institute of Health and Welfare, 2008. <http://www.aihw.gov.au/publication-detail/?id=6442468102>
118. Barba C, Cavalli-Sforza T, Cutter J, Darnton-Hill I, Deurenberg P, Deurenberg-Yap M et al. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 2004;363(9403).
119. Flicker L, McCaul KA, Hankey GJ, Jamrozik K, Brown WJ, Byles JE et al. Body mass index and survival in men and women aged 70 to 75. *J Am Geriatr Soc* 2010;58(2):234–41.
120. Rush E, Plank L, Chandu V, Lulu M, Simmons D, Swinburn B et al. Body size, body composition, and fat distribution: a comparison on young New Zealand men of European, Pacific Island, and Asian ethnicities. *NZ Med J* 2004;117:U1203.
121. National Health and Medical Research Council. Clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children. Melbourne: National Health and Medical Research Council; 2013.
122. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *Br Med J* 2000;320(7244):1–6.
123. Cole TJ, Flegal KM, Nicholls D, Jackson AA. Body mass index cut offs to define thinness in children and adolescents: international survey. *Br Med J* 2007;335(7612):194.
124. de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. *Bull World Health Organ* 2007;85(9):660–7.
125. Sinatra FR. Nonalcoholic fatty liver disease in paediatric patients. *Journal of Parenteral and Enteral Nutrition* 2012;36(1):43S–48S.
126. Prospective Studies Collaboration, Whitlock G, Lewington S, Sherliker P, Clarke R, Emberson J et al. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. *Lancet* 2009;373(9669):1083–96.
127. Stevens J, Cai J, Pamuk ER, Williamson DF, Thun MJ, Wood JL. The effect of age on the association between body-mass index and mortality. *N Engl J Med* 1998;338(1):1–7.
128. Diehr P, O'Meara ES, Fitzpatrick A, Newman AB, Kuller L, Burke G. Weight, mortality, years of healthy life, and active life expectancy in older adults. *J Am Geriatr Soc* 2008;56(1):76–83.
129. National Eating Disorders Collaboration. Eating disorders- prevention, treatment and management: an evidence review. 2010.
130. Denney-Wilson E, Hardy LL, Dobbins T, Okely AD, Baur LA. Body mass index, waist circumference, and chronic disease risk factors in Australian adolescents. *Arch Pediatr Adolesc Med* 2008;162(6):566–73.

131. Clarke PJ, O'Malley PM, Schulenberg JE, Johnston LD. 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):540-8.
132. Biro FM, Wien M. Childhood obesity and adult morbidities. *Am J Clin Nutr* 2010;91(5):1499S-505S.
133. Oates RK. Non-organic failure to thrive. *Aust Paediatr J* 1984;20(2):95-100.
134. Pugliese MT, Weyman-Daum M, Moses N, Lifshitz F. Parental health beliefs as a cause of nonorganic failure to thrive. *Pediatrics* 1987;80(2):175-82.
135. Stubbs CO, Lee AJ. The obesity epidemic: both energy intake and physical activity contribute. *Med J Aust* 2004;181(9):489-91.
136. Bensimhon DR, Kraus WE, Donahue MP. Obesity and physical activity: a review. *Am Heart J* 2006;151(3):598-603.
137. Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA 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):1081-93.
138. Swinburn B, Egger G. The runaway weight gain train: too many accelerators, not enough brakes. *Br Med J* 2004;329(7468):736-9.
139. Swinburn B, Sacks G, Ravussin E. Increased food energy supply is more than sufficient to explain the US epidemic of obesity. *Am J Clin Nutr* 2009;90(6):1453-6.
140. Swinburn BA, Caterson I, Seidell JC, James WP. Diet, nutrition and the prevention of excess weight gain and obesity. *Public Health Nutr* 2004;7(1A):123-46.
141. Sacks G, Swinburn BA, Lawrence MA. A systematic policy approach to changing the food system and physical activity environments to prevent obesity. *Aust NZ Health Policy* 2008;5:13.
142. Drewnowski A, Specter SE. Poverty and obesity: the role of energy density and energy costs. *Am J Clin Nutr* 2004;79(1):6-16.
143. Ballal H, Schofield G. Transport, urban design, and physical activity: an evidence-based update. *Transport Res* 2005;10(3):177-96.
144. Jackson RJ, Kochitzky C. Creating a healthy environment: the impact of the built environment on public health. Washington DC.: Sprawl Watch, 2001. Available from: <http://www.sprawlwatch.org/health.pdf>
145. Oliver LN, Hayes MV. Neighbourhood socio-economic status and the prevalence of overweight Canadian children and youth. *Can J Public Health* 2005;96(6):415-20.
146. Gill T, King L, Webb K. Best options for promoting healthy weight and preventing weight gain in NSW. Sydney: NSW Centre for Public Health Nutrition, 2005. Available from: http://sydney.edu.au/science/molecular_bioscience/cphn/pdfs/healthy_weight_report.pdf
147. Kumanyika SK. The obesity epidemic: looking in the mirror. *Am J Epidemiol* 2007;166(3):243-5.
148. Ebbeling CB, Pawlak DB, Ludwig DS. Childhood obesity: public-health crisis, common sense cure. *Lancet* 2002;360(9331):473-82.
149. McLaren L. Socioeconomic status and obesity. *Epidemiol Rev* 2007;29:29-48.
150. Papas MA, Alberg AJ, Ewing R, Helzlsouer KJ, Gary TL, Klassen AC. The built environment and obesity. *Epidemiol Rev* 2007;29:129-43.
151. National Obesity Taskforce. Healthy weight 2008 - Australia's future - The national action agenda for children and young people and their families. Canberra: Commonwealth of Australia; 2003.
152. Cook T, Rutishauser I, Seelig M. Comparable data on food and nutrient intake and physical measurements from the 1983, 1985 and 1995 national nutrition surveys. Canberra: Commonwealth of Australia, 2001. [http://www.health.gov.au/internet/main/publishing.nsf/Content/6A40E29D690738DECA25725F00810008/\\$File/nutrient.pdf](http://www.health.gov.au/internet/main/publishing.nsf/Content/6A40E29D690738DECA25725F00810008/$File/nutrient.pdf)

153. Rangan AM, Schinleler 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):665-71.
154. Stubbs CO. Determinants of the global obesity epidemic: Contributions of early life, nutrition, physical activity, television, smoking and economic and social change. In: Melrose LA, ed. *New developments in obesity research*. New York: Nova Publishers, 2006: 121–52.
155. Prentice AM, Black AE, Coward WA, Cole TJ. 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):93–7.
156. Gebel K, King L, Bauman A, Vita P, Gill T, Rigby A et al. *Creating healthy environments: A review of links between the physical environment, physical activity and obesity*. Sydney: NSW Health Department and NSW Centre for Overweight and Obesity, 2005.
157. Armstrong T, Bauman A, Davies J. *Physical activity patterns of Australian adults. Results of the 1999 National Physical Activity Survey*. Canberra: Australian Institute of Health and Welfare, 2000. Available from: <http://www.aihw.gov.au/publication-detail/?id=6442467175>
158. Queensland Health. *The health of Queenslanders 2006. Report of the Chief Health Officer, Queensland*. Brisbane: Queensland Health; 2006.
159. Centre for Epidemiology and Research. *The health of the people of New South Wales - Report of the Chief Health Officer. Summary report, 2010*. Sydney: NSW Department of Health, 2010. <http://www.health.nsw.gov.au/publichealth/chorep/>
160. Booth ML, Cheyde, Wake M, Norton K, Hesketh K, Dollman J et al. Change in the prevalence of overweight and obesity among young Australians, 1969-1997. *Am J Clin Nutr* 2003;77(1):29–36.
161. Booth ML, Denney-Wilson E, Okely AD, Hardy LL. Methods of the NSW Schools Physical Activity and Nutrition Survey (SPANS). *J Sci Med Sport* 2005;8(3):284–93.
162. Abbott RA, Macdonald D, Mackinnon L, Stubbs CO, Lee AJ, Harper C et al. *Healthy Kids Queensland Survey 2006 — Summary Report*. Brisbane: Queensland Health, 2007.
163. Bauman A, Bellew B, Vita P, Brown W, Owen N. *Getting Australia active: Towards better practice for the promotion of physical activity*. Melbourne: National Public Health Partnership, 2002. Available from: <http://fulltext.ausport.gov.au/fulltext/2002/nphp/gaa.asp>
164. Woodcock J, Franco OH, Orsini N, Roberts I. Non-vigorous physical activity and all-cause mortality: systematic review and meta-analysis of cohort studies. *Int J Epidemiol* 2011;40(1):121–38.
165. Janssen I, Leblanc AG. Systematic review of the health benefits of physical activity and fitness in school-aged children and youth. *Int J Behav Nutr Phys Act* 2010;7:40.
166. Warburton DE, Nicol CW, Bredin SS. Health benefits of physical activity: the evidence. *Can Med Assoc J* 2006;174(6):801–9.
167. Brown WJ, Mishra G, Lee C, Bauman A. Leisure time physical activity in Australian women: relationship with well being and symptoms. *Res Q Exerc Sport* 2000;71(3):206–16.
168. Nelson MC, Gordon-Larsen P. Physical activity and sedentary behavior patterns are associated with selected adolescent health risk behaviors. *Pediatrics* 2006;117(4):1281–90.
169. Commonwealth Department of Health and Ageing. *Measure up - weight and waist measurements. 2010* (cited 27 March 2011). <http://www.health.gov.au/internet/abhi/publishing.nsf/Content/Weight,+waist+circumference+and+BMI-lp>. <http://www.patonsyarns.com/pattern.ph>
170. Dunstan D, Zimmet P, Welborn T, Sicree R, Armstrong T, Atkins R et al. *Diabetes and associated disorders in Australia - 2000*. Melbourne: International Diabetes Institute, 2001. Available from: http://www.diabetes.com.au/pdf/AusDiab_Report.pdf
171. Orozco Leonardo J, Buchleitner Ana M, Gimenez-Perez G, Roqué i Figuls M, Richter B, Mauricio D. Exercise or exercise and diet for preventing type 2 diabetes mellitus. *Cochrane Database Syst Rev* 2008(3).

172. Kamath CC, Vickers KS, Ehrlich A, McGovern L, Johnson J, Singhal V 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):4606–15.
173. Campbell KJ, Hesketh KD. 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):327–38.
174. Summerbell CD, Waters E, Edmunds L, Kelly SAM, Brown T, Campbell KJ. Interventions for preventing obesity in children. *Cochrane Database Syst Rev* 2005(3).
175. Stice E, Shaw H, Marti CN. A meta-analytic review of obesity prevention programs for children and adolescents: the skinny on interventions that work. *Psychol Bull* 2006(5):667–91.
176. Gilles A, Cassano M, Shepherd EJ, Higgins D, Hecker JE, Nangle DW. Comparing active pediatric obesity treatments using meta-analysis. *J Clin Child Adolesc Psychol* 2008;37(4):886–92.
177. Young KM, Northern JJ, Lister KM, Drummond JA, O'Brien WH. A meta-analysis of family-behavioral weight-loss treatments for children. *Clin Psychol Rev* 2007;27(2):240–9.
178. Berry D, Sheehan R, Heschel R, Knafel K, Melkus G, Grey M. Family-based interventions for childhood obesity: a review. *J Family Nursing* 2004;10(4):429.
179. Kelly SA, Melnyk BM. Systematic review of multicomponent interventions with overweight middle adolescents: implications for clinical practice and research. *Worldviews Evid Based Nurs* 2008;5(3):113–35.
180. Doak CM, Visscher TL, Renders CM, Seidell JC. The prevention of overweight and obesity in children and adolescents: a review of interventions and programmes. *Obes Rev* 2006;7(1):411–36.
181. Cole K, Waldrop J, D'Auria J, Garner H. An integrative research review: effective school-based childhood overweight interventions. *J Spec Pediatr Nurs* 2006;11(3):166–77.
182. Oude Luttikhuis H, Baur L, Jansen H, Shrewsbury VA, O'Malley C, Stolk RP et al. Interventions for treating obesity in children. *Cochrane Database Syst Rev* 2009(1):CD001872.
183. Waters E, de Silva-Sanigorski A, Hall BJ, Brown T, Campbell KJ, Gao Y et al. Interventions for preventing obesity in children. *Cochrane Database Syst Rev* 2011(12):CD001871.
184. Nations FAO/WHO. Fats and fatty acids in human nutrition. Report of an expert consultation. Rome: Food and Agriculture Organization of the United Nations, 2010.
185. Dubois L, Farmer A, Girard M, Peterson K. Regular sugar-sweetened beverage consumption between meals increases risk of overweight among preschool-aged children. *J Am Diet Assoc* 2007;107(6):924–34.
186. Faith MS, Dennison BA, Edmunds LS, Stratton HH. Fruit juice intake predicts increased adiposity gain in children from low-income families: weight status-by-environment interaction. *Pediatrics* 2006;118(5):2066–75.
187. Forshee RA, Anderson PA, Storey ML. Sugar-sweetened beverages and body mass index in children and adolescents: a meta-analysis. *Am J Clin Nutr* 2008;87(6):1662–71.
188. 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):134–47.
189. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr* 2006;84(2):274–88.
190. Phillips SM, Bandini LG, Naumova EN, Cyr H, Colclough S, Dietz WH et al. Energy-dense snack food intake in adolescence: longitudinal relationship to weight and fatness. *Obes Res* 2004;12(3):461–72.
191. Sichieri R, PaulaTrotte A, de Souza RA, Veiga GV, Sichieri R, PaulaTrotte A et al. School randomised trial on prevention of excessive weight gain by discouraging students from drinking sodas. *Public Health Nutr* 2009;12(2):197–202.

192. Stookey JD, Constant F, Gardner CD, Popkin BM. Replacing sweetened caloric beverages with drinking water is associated with lower energy intake. *Obesity (Silver Spring)* 2007;15(12):3013–22.
193. Tam CS, Garnett SP, Cowell CT, Campbell K, Cabrera G, Baur LA. Soft drink consumption and excess weight gain in Australian school students: results from the Nepean study. *Int J Obesity* 2006;30(7):1091–3.
194. Taylor RW, McAuley KA, Barbezat W, Strong A, Williams SM, Mann JI. APPLE Project: 2-y findings of a community-based obesity prevention program in primary school age children. *Am J Clin Nutr* 2007;86(3):735–42.
195. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health* 2007;97(4):667–75.
196. Wolff E, Dansinger ML. Soft drinks and weight gain: how strong is the link? *Medscape J Med* 2008;10(8):189.
197. Fiorito LM, Marini M, Francis LA, Smicklas-Wright H, Birch LL. Beverage intake of girls at age 5 y predicts adiposity and weight status in childhood and adolescence. *Am J Clin Nutr* 2009;90(4):935–42.
198. U.S. Department of Agriculture and U.S. Department of Health and Human Services. *Dietary guidelines for Americans*, 2010. 7th Edition ed. Washington, DC: U.S. Government Printing Office; 2010.
199. Vrolix R MR. Variability of the glycemic response to single food products in healthy subjects. *Contemporary Clinical Trials* 2010;31(1):5–11.
200. Field AE, Gillman MW, Rosner B, Rockett HR, Colditz GA. 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):821–6.
201. Sartorelli DS, Franco LJ, Cardoso MA, Sartorelli DS, Franco LJ, Cardoso MA. High intake of fruits and vegetables predicts weight loss in Brazilian overweight adults. *Nutr Res* 2008;28(4):233–8.
202. te Velde SJ, Twisk JW, Brug J, te Velde SJ, Twisk JWR, Brug J. Tracking of fruit and vegetable consumption from adolescence into adulthood and its longitudinal association with overweight. *Br J Nutr* 2007;98(2):431–8.
203. Vioque J, Weinbrenner T, Castello A, Asensio L, de la Hera MG. Intake of fruits and vegetables in relation to 10-year weight gain among Spanish adults. *Obesity (Silver Spring)* 2008;16(3):664–70.
204. Booth A, Nowson C, Worsley A, Margeterison C, Jorna M. Dietary approaches for weight loss with increased intakes of fruit, vegetables and dairy products. *Nutr Diet* 2008;65(2):115–20.
205. Conceição de Oliveira M, Sichieri R, Sanchez Moura A. Weight loss associated with a daily intake of three apples or three pears among overweight women. *Nutrition* 2003;19(3):253–56.
206. Drapeau V, Despres JP, Bouchard C, Allard L, Fournier G, Leblanc C et al. Modifications in food-group consumption are related to long-term body-weight changes. *Am J Clin Nutr* 2004;80(1):29–37.
207. He K, Hu FB, Colditz GA, Manson JE, Willett WC, Liu S. 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):1569–74.
208. Rodriguez MC, Parra MD, Marques-Lopes I, De Morentin BE, Gonzalez A, Martinez JA. Effects of two energy-restricted diets containing different fruit amounts on body weight loss and macronutrient oxidation. *Plant Foods Hum Nutr* 2005;60(4):219–24.
209. Barr SI. Increased dairy product or calcium intake: is body weight or composition affected in humans? *J Nutr* 2003;133(1):245S–48S.
210. Lanou AJ, Barnard ND. Dairy and weight loss hypothesis: an evaluation of the clinical trials. *Nutr Rev* 2008;66(5):272–79.
211. Rajpathak SN, Rimm EB, Rosner B, Willett WC, Hu FB. Calcium and dairy intakes in relation to long-term weight gain in US men. *Am J Clin Nutr* 2006;83(3):559–66.

212. Wagner G, Kindrick S, Hertzler S, DiSilvestro RA, Wagner G, Kindrick S 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):456–61.
213. Pereira MA, Jacobs DR, Jr., Van Horn L, Slattery ML, Kartashov AI, Ludwig DS. Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study. *J Am Med Assoc* 2002;287(16):2081–9.
214. Rosell M, Hakansson NN, Wolk A. Association between dairy food consumption and weight change over 9 y in 19,352 perimenopausal women. *Am J Clin Nutr* 2006;84(6):1481–8.
215. Huus K, Brekke HK, Ludvigsson JF, Ludvigsson J, Huus K, Brekke HK et al. Relationship of food frequencies as reported by parents to overweight and obesity at 5 years. *Acta Paediatr* 2009;98(1):139–43.
216. Johnson L, Wilks DC, Lindroos AK, Jebb SA. 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):681–92.
217. Newby PK, Peterson KE, Berkey CS, Leppert J, Willett WC, Colditz GA. 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):1086–94.
218. Striegel-Moore RH, Thompson D, Affenito SG, Franko DL, Obarzanek E, Barton BA 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):183–7.
219. Berkey CS, Rockett HR, Willett WC, Colditz GA. Milk, dairy fat, dietary calcium, and weight gain: a longitudinal study of adolescents. *Arch Pediatr Adolesc Med* 2005;159(6):543–50.
220. De La Hunty A, Ashwell M. Are people who regularly eat breakfast cereals slimmer than those who don't? A systematic review of the evidence. *Nutrition Bulletin* 2007;32(2):818–28.
221. Gilhooly CH, Das SK, Golden JK, McCrory MA, Rochon J, DeLany JP et al. Use of cereal fiber to facilitate adherence to a human caloric restriction program. *Aging Clin Exp Res* 2008;20(6):513–20.
222. Hallfrisch J, Scholfield DJ, Behall KM. Blood pressure reduced by whole grain diet containing barley or whole wheat and brown rice in moderately hypercholesterolemic men. *Nutr Res* 2003;23(12):1631–42.
223. Hamedani A, Akhavan T, Samra RA, Anderson GH, Hamedani A, Akhavan T 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):1343–49.
224. Lightowler HJ, Henry CJK. 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):48–53.
225. Ortega RM, Andres P, Lopez-Sobaler AM, Rodriguez-Rodriguez E, Aparicio A, Bermejo LM 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):77–82.
226. Ortega RM, Rodriguez-Rodriguez E, Aparicio A, Marin-Arias LI, Lopez-Sobaler AM, Ortega RM et al. Responses to two weight-loss programs based on approximating the diet to the ideal: differences associated with increased cereal or vegetable consumption. *Int J Vitamin Nutrition Res* 2006;76(6):367–76.
227. Rodriguez-Rodriguez E, Aparicio A, Bermejo LM, Lopez-Sobaler AM, Ortega RM, 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):44–50.
228. Waller SM, VanderWal JS, Klurfeld DM, McBurney MI, Cho S, Bijlani S et al. Evening ready-to-eat cereal consumption contributes to weight management. *J Am Coll Nutr* 2004;23(4):316–21.
229. Williams PG, Grafenauer SJ, O'Shea JE, Williams PG, Grafenauer SJ, O'Shea JE. Cereal grains, legumes, and weight management: a comprehensive review of the scientific evidence. *Nutr Rev* 2008;66(4):171–82.
230. Fraser GE, Bennett HW, Jaceldo KB, Sabate J. 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):275.

231. Hyson DA, Schneeman BO, Davis PA. Almonds and almond oil have similar effects on plasma lipids and LDL oxidation in healthy men and women. *J Nutr* 2002;132(4):703.
232. Kocyigit A, Koylu AA, Keles H. Effects of pistachio nuts consumption on plasma lipid profile and oxidative status in healthy volunteers. *Nutr Metab Cardiovasc Dis* 2006;16(3):202–09.
233. Sabate J. Nut consumption and change in weight: the weight of the evidence. *Br J Nutr* 2007;98(3):456–7.
234. Sabate J, Haadland E, Tanzman JS, Jambazian P, Rajaram S. 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):1379–84.
235. Spaccarotella KJ, Kris-Etherton PM, Stone WL, Bagshaw DM, Fishell VK, West SG et al. The effect of walnut intake on factors related to prostate and vascular health in older men. *Nutr J* 2008;7:13.
236. Hite AH, Feinman RD, Guzman GE, Satin M, Schoenfeld PA, Wood RJ. In the face of contradictory evidence: report of the Dietary Guidelines for Americans Committee. *Nutrition* 2010;26(10):915–24.
237. Owen CG, Martin RM, Whincup PH, Smith GD, Cook DG. Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence. *Pediatrics* 2005;115(5):1367–77.
238. Araujo CL, Hallal PC, Nader GA, Neutzling MB, deFatima Vieira M, Menezes AM 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):634–9.
239. Kuh D, Hardy R, Chaturvedi N, Wadsworth ME. Birth weight, childhood growth and abdominal obesity in adult life. *Int J Obes Relat Metab Disord* 2002;26(1):40–7.
240. Monasta L, Batty GD, Cattaneo A, Lutje V, Ronfani L, Van Lenthe FJ et al. Early-life determinants of overweight and obesity: a review of systematic reviews. *Obes Rev* 2010;11(10):695–708.
241. Monteiro PO, Victora CG. Rapid growth in infancy and childhood and obesity in later life--a systematic review. *Obes Rev* 2005;6(2):143–54.
242. Nader PR, O'Brien M, Houts R, Bradley R, Belsky J, Crosnoe R et al. Identifying risk for obesity in early childhood. *Pediatrics* 2006;118(3):e594–601.
243. Mamun AA, Lawlor DA, Alati R, O'Callaghan MJ, Williams GM, Najman JM. 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):317–25.
244. Landhuis CE, Poulton R, Welch D, Hancox RJ. Childhood sleep time and long-term risk for obesity: a 32-year prospective birth cohort study. *Pediatrics* 2008;122(5):955–60.
245. Marshall SJ, Biddle SJ, Gorely T, Cameron N, Murley I. Relationships between media use, body fatness and physical activity in children and youth: a meta-analysis. [Review] [76 refs]. *Int J Obes Relat Metab Dis* 2004;28(10):1238–46.
246. Rey-Lopez JP, Vicente-Rodriguez G, Biosca M, Moreno LA. Sedentary behaviour and obesity development in children and adolescents. *Nutr Metab Cardiovasc Dis* 2008;18(3):242–51.
247. Biddle SJ, Gorely T, Marshall SJ, Murley I, Cameron N. Physical activity and sedentary behaviours in youth: issues and controversies. *Her Soc Promot Health* 2004;124(1):29–33.
248. Coon KA, Tucker KL. Television and children's consumption patterns: a review of the literature. *Minerva Pediatr* 2002;54(5):423–36.
249. Goldani MZ, Haeflner LS, Agranonik M, Barbieri MA, Bettiol H, Silva AA. Do early life factors influence body mass index in adolescents? *Braz J Med Biol Res* 2007;40(9):1231–6.
250. Langenberg C, Hardy R, Kuh D, Brunner E, Wadsworth M. 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):816–22.

251. Power C, Atherton K, Strachan DP, Shepherd P, Fuller E, Davis A 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):532–9.
252. Wright CM, Parker L. Forty years on: the effect of deprivation on growth in two Newcastle birth cohorts. *Int J Epidemiol* 2004;33(1):147–52.
253. Jyoti DF, Frongillo EA, Jones SJ. Food insecurity affects school children's academic performance, weight gain, and social skills. *J Nutr* 2005;135(12):2831–9.
254. Senf JH, Shisslak CM, Crago MA. Does dieting lead to weight gain? A four-year longitudinal study of middle school girls. *Obesity* 2006;14(12):2236–41.
255. Field AE, Austin SB, Taylor CB, Malspeis S, Rosner B, Rockett HR et al. Relation between dieting and weight change among preadolescents and adolescents. *Pediatrics* 2003;112(4):900–6.
256. Mamun AA, Lawlor DA, Cramb S, O'Callaghan M, Williams G, Najman J. Do childhood sleeping problems predict obesity in young adulthood? Evidence from a prospective birth cohort study. *Am J Epidemiol* 2007;166(12):1368–73.
257. Taveras EM, Rifas-Shiman SL, Oken E, Gunderson EP, Gillman MW. Short sleep duration in infancy and risk of childhood overweight. *Arch Pediatr Adolesc Med* 2008;162(4):305–11.
258. Timlin MT, Pereira MA, Story M, Neumark-Sztainer D. Breakfast eating and weight change in a 5-year prospective analysis of adolescents: Project EAT (Eating Among Teens). *Pediatrics* 2008;121(3):e638–45.
259. Astrup A, Grunwald GK, Melanson EL, Saris WH, Hill JO. 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):1545–52.
260. Willett WC, Leibel RL. Dietary fat is not a major determinant of body fat. *Am J Med* 2002;113(Suppl 9B):47S–59S.
261. Prentice AM, Jebb SA. Fast foods, energy density and obesity: a possible mechanistic link. *Obes Rev* 2003;4(4):187–94.
262. McCrory MA, Saltzman E, Rolls BJ, Roberts SB. A twin study of the effects of energy density and palatability on energy intake of individual foods. *Physiol Behav* 2006;87(3):451–9.
263. Paddon-Jones D, Westman E, Mattes RD, Wolfe RR, Astrup A, Westerterp-Plantenga M. Protein, weight management, and satiety. *Am J Clin Nutr* 2008;87(5):1558S–61S.
264. Drewnowski A, Darmon N. The economics of obesity: dietary energy density and energy cost. *Am J Clin Nutr* 2005;82(1 Suppl):265S–73S.
265. Bowman SA, Gortmaker SL, Ebbeling CB, Pereira MA, Ludwig DS. Effects of fast-food consumption on energy intake and diet quality among children in a national household survey. *Pediatrics* 2004;113(1 Pt 1):112–8.
266. Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR, Jr. et al. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. *Lancet* 2005;365(9453):36–42.
267. Ledikwe JH, Ello-Martin JA, Rolls BJ. Portion sizes and the obesity epidemic. *J Nutr* 2005;135(4):905–9.
268. Cameron-Smith D, Bilsborough SA, Crowe TC. Upsizing Australia's waistline: the dangers of "meal deals". *Med J Aust* 2002;177(11-12):2–16.
269. Franz MJ, VanWormer JJ, Crain AL, Boucher JL, Histon T, Caplan W 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):1755–67.
270. Goodpaster BH, Delany JP, Otto AD, Kuller L, Vockley J, South-Paul JE 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):1795–802.

271. Marks GC, Coyne C, Pang G. Type 2 diabetes costs in Australia - the potential impact of changes in diet, physical activity and levels of obesity. Brisbane: Australian Food and Nutrition Modelling Unit, 2001. Available from: <http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-food-pd-f-diabetes-cnt.htm>
272. National Public Health Partnership. Be active Australia: A framework for health sector action for physical activity 2005–2010. In: Department of Human Services V, editor. Melbourne: National Public Health Partnership; 2005.
273. Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. 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):1785–91.
274. Martinez-Gonzalez MA, Martinez JA, Hu FB, Gibney MJ, Kearney J. Physical inactivity, sedentary lifestyle and obesity in the European Union. *Int J Obes Relat Metab Disord* 1999;23(11):1192–201.
275. Brown WJ, Miller YD, Miller R. Sitting time and work patterns as indicators of overweight and obesity in Australian adults. *Int J Obesity Relat Metabol Dis* 2003;27(11):1340–6.
276. Saris WH, Blair SN, van Baak MA, Eaton SB, Davies PS, Di Pietro L 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):101–14.
277. Blair SN, LaMonte MJ, Nichaman MZ. The evolution of physical activity recommendations: how much is enough? *Am J Clin Nutr* 2004;79(5).
278. Petersen L, Schnohr P, Sørensen T. Longitudinal study of the long-term relation between physical activity and obesity in adults. *Int J Obes* 2003;28(1):105–12.
279. Schoeller DA. But how much physical activity? *Am J Clin Nutr* 2003;78(4):669–70.
280. Martinez-Gomez D, Ruiz JR, Ortega FB, Veiga OL, Moliner-Urdiales D, Mauro B 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):203–11.
281. Caterson ID, Finer N. Emerging pharmacotherapy for treating obesity and associated cardiometabolic risk. *Asia Pacific* 2006;15:55–62.
282. Callaway LK, O'Callaghan MJ, McIntyre HD. Barriers to addressing overweight and obesity before conception. *Med J Aust* 2009;191(8):425–28.
283. Mamun AA, Kinarivala M, O'Callaghan MJ, Williams GM, Najman JM, Callaway LK. 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):1336–41.
284. Dodd JM, Grivell RM, Crowther CA, Robinson JS. Antenatal interventions for overweight or obese pregnant women: a systematic review of randomised trials: a systematic review of randomised trials. *BJOG* 2010;117(11):1316–26.
285. Callaway LK, Prins JB, Chang AM, McIntyre HD. The prevalence and impact of overweight and obesity in an Australian obstetric population. *Med J Aust* 2006;184(2):56–9.
286. Budge H, Gnanalingham MG, Gardner DS, Mostyn A, Stephenson T, Symonds ME. Maternal nutritional programming of fetal adipose tissue development: long-term consequences for later obesity. *Birth Defects Res* 2005;75(3):193–9.
287. Boney CM, Verma A, Tucker R, Vohr BR. Metabolic syndrome in childhood: association with birth weight, maternal obesity, and gestational diabetes mellitus. *Pediatrics* 2005;115(3):e290–6.
288. Institute of Medicine. Preventing childhood obesity: Health in the balance. Washington DC: Institute of Medicine of the National Academies, 2005.
289. Institute of Medicine NRC. Weight Gain During Pregnancy: Reexamining the Guidelines. Washington, DC: The National Academies Press, 2009.

290. Fraser A, Tilling K, Macdonald-Wallis C, Hughes R, Sattar N, Nelson SM 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.
291. Kinnunen TI, Pasanen M, Aittasalo M, Fogelholm M, Weiderpass E, Luoto R. Reducing postpartum weight retention--a pilot trial in primary health care. *Nutr J* 2007;6:21.
292. Harding JE. The nutritional basis of the fetal origins of adult disease. *Int J Epidemiol* 2001;30(1):15.
293. Makrides M. Early childhood nutrition and cognitive outcome. *Proc Nutr Soc Aust* 1998;22:216-22.
294. Cooper C, Fall C, Egger P, Hobbs R, Eastell R, Barker D. Growth in infancy and bone mass in later life. *Ann Rheum Dis* 1997;56(1):17-21.
295. National Health and Medical Research Council. *Infant Feeding Guidelines*. Canberra: Commonwealth of Australia; 2012.
296. Evans WJ. Effects of aging and exercise on nutrition needs of the elderly. *Nutr Rev* 1996;54(1 Pt 2):S35-9.
297. Nelson ME, Rejeski WJ, Blair SN, Duncan PW, Judge JO, King AC 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):1094-105.
298. Department of Health and Ageing. *Physical activity guidelines*. 2010 (cited 30 March 2011). <http://www.health.gov.au/internet/main/publishing.nsf/content/health-pubhlth-strateg-phys-act-guidelines>. <http://www.patonsyarns.com/pattern.ph>
299. Gregg EW, Pereira MA, Caspersen CJ. Physical activity, falls, and fractures among older adults: a review of the epidemiologic evidence. *J Am Geriatr Soc* 2000;48(8):883-93.
300. Kujala UM, Kaprio J, Kannus P, Sarna S, Koskenvuo M. Physical activity and osteoporotic hip fracture risk in men. *Arch Intern Med* 2000;160(5):705-8.
301. Glass TA, De Leon CM, Marottoli RA, Berkman LF. Population based study of social and productive activities as predictors of survival among elderly Americans. *Br Med J* 1999;319(7208):478.
302. Truswell AS. Dietary guidance for older Australians. *Nutr Diet* 2009;66(4):243-48.
303. Nowson C. Nutritional challenges in the elderly. *Australian Doctor* 2009(27 March):25-30.
304. Corrada MM, Kawas CH, Mozaffar F, Paganini-Hill A. Association of body mass index and weight change with all-cause mortality in the elderly. *Am J Epidemiol* 2006;163(10):938-49.
305. Strandberg TE, Pitkala KH. Frailty in elderly people. *Lancet* 2007;369(9570):1328-9.
306. Wahleqvist ML, Kouris-Blazos A, Ross K, Setter T, Tienboon T. Growth and Ageing. In: *Nutrition and Metabolism*. M Gibney, editor. Oxford: British Nutrition Society, 2002.
307. Leggo M, Banks M, Isenring E, Stewart L, Tweeddale M. A quality improvement nutrition screening and intervention program available to Home and Community Care eligible clients. *Nutr Diet* 2008;65(2):162-67.
308. Banks M, Ash S, Bauer J, Gaskill D. Prevalence of malnutrition in adults in Queensland public hospitals and residential aged care facilities. *Nutr Diet* 2007;64(3):672-78.
309. Morley JE, van Staveren WA. Undernutrition: diagnosis, causes, consequences and treatment. In: Raats M, de Groot L, van Staveren WA, eds. *Food for the ageing population*. Cambridge: Woodhead Publishing Limited, 2009.
310. Cunningham J, Mackerras D. Occasional paper - Overweight and obesity Indigenous Australians 1994. Canberra: Australian Bureau of Statistics, 1998. <http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/4702.01994?OpenDocument>
311. O'Dea K. Body fat distribution and health outcome in Australian Aborigines. *Proc Nutr Soc Aust* 1987;12:56-65.

312. Cunningham J, O'Dea K, Dunbar T, Maple Brown L. Perceived weight versus Body Mass Index among urban Aboriginal Australians: do perceptions and measurements match? *Aust N Z J Public Health* 2008;32(2):135–38.
313. Australian Institute of Health and Welfare. Aboriginal and Torres Strait Islander Health Performance Framework, 2008 report: Detailed analyses. Canberra: Australian Institute of Health and Welfare, 2008.
314. Turrell G, Hewitt B, Patterson C, Oldenburg B, Gould T. Socioeconomic differences in food purchasing behaviour and suggested implications for diet-related health promotion. *J Hum Nutr Diet* 2002;15(5):355–64.
315. Maillot M, Darmon N, Darmon M, Lafay L, Drewnowski A. Nutrient-dense food groups have high energy costs: an econometric approach to nutrient profiling. *J Nutr* 2007;137(7):8815.
316. Kettings C, Sinclair AJ, Voevodin M. A healthy diet consistent with Australian health recommendations is too expensive for welfare dependent families. *Aust N Z J Public Health* 2009;33(6):566–72.
317. Harrison M, Lee A, Findlay M, Nicholls R, Leonard D, Martin C. The increasing cost of healthy food. *Aust N Z J Public Health* 2010;34(2):179–86.
318. Food Standards Australia New Zealand. The 22nd Australian Total Diet Study. Canberra, Australia and Wellington, NZ: Food Standards Australia New Zealand, 2008. Available from: <http://www.foodstandards.gov.au/scienceandeducation/publications/22ndaustraliantotaldietstudy/>
319. Commonwealth Department of Foreign Affairs and Trade. Australia in brief: a diverse people. 2010 (cited 29 March 2011). <http://www.dfat.gov.au/aib/society.html>. <http://www.patonsyarns.com/pattern.ph>
320. Symons M. One continuous picnic: a gastronomic history of Australia. 2nd ed. Melbourne: Melbourne University Press, 2007. <http://www.loc.gov/catdir/toc/fy0709/2007386861.html>
321. Stanton RA. Nutrition problems in an obesogenic environment. *Med J Aust* 2006;184(2):76–9.
322. Walker KZ, Woods JL, Rickard CA, Wong CK. Product variety in Australian snacks and drinks: how can the consumer make a healthy choice? *Public Health Nutr* 2008;11(10):1046–53.
323. National Health and Medical Research Council. Iodine supplementation for pregnant and breastfeeding women. Canberra: Commonwealth of Australia; 2010.
324. Harriss LR, English DR, Powles J, Giles GG, Tonkin AM, Hodge AM et al. Dietary patterns and cardiovascular mortality in the Melbourne Collaborative Cohort Study. *Am J Clin Nutr* 2007;86(1):221–9.
325. Jacques PF, Tucker KL. Are dietary patterns useful for understanding the role of diet in chronic disease? *Am J Clin Nutr* 2001;73(1):1–2.
326. Kant AK, Schatzkin A, Graubard BI, Schairer C. A prospective study of diet quality and mortality in women. *J Am Med Assoc* 2000;283(16):2109–15.
327. McCullough ML, Feskanich D, Stampfer MJ, Giovannucci EL, Rimm EB, Hu FB 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):1261–71.
328. Wahlqvist ML, Wattanapenpaiboon N, Kannar D, Dalais F, Kouris-Blazos A. Phytochemical deficiency disorders: inadequate intake of protective foods. *Curr Therapeut* 1998;39(7):53–60.
329. Van Dokkum W, Frölisch W, Saltmarsh M, Gee J. The health effects of bioactive plant components in food: results and opinions of the EU COST 926 action. *Nutrition Bulletin* 2008;33(2):133–39.
330. Dolan LC, Matulka RA, Burdock GA. Naturally occurring food toxins. *Toxins* 2010;2(9):2289–332.
331. Gibson RS. 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):S77–100.
332. Lopez MA, Martos FC. Iron availability: an updated review. *Int J Food Sci Nutr* 2004;55(8):597–606.
333. Russell RM. The vitamin A spectrum: from deficiency to toxicity. *Am J Clin Nutr* 2000;71(4):878–84.

334. Knudsen VK, Orozova-Bekkevold IM, Mikkelsen TB, Wolff S, Olsen SF. Major dietary patterns in pregnancy and fetal growth. *Eur J Clin Nutr* 2008;62(4):463–70.
335. Rodríguez-Bernal CL, Rebagliato M, Iniguez C, Vioque J, Navarrete-Munoz EM, Murcia M 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):1659–66.
336. Thompson JM, Wall C, Becroft DM, Robinson E, Wild CJ, Mitchell EA. Maternal dietary patterns in pregnancy and the association with small-for-gestational-age infants. *Br J Nutr* 2010;103(11):1665–73.
337. Kinnunen TI, Pasanen M, Aittasalo M, Fogelholm M, Hilakivi-Clarke L, Weiderpass E et al. Preventing excessive weight gain during pregnancy - a controlled trial in primary health care. *Eur J Clin Nutr* 2007;61(7):884–91.
338. Brantsæter AL, Haugen M, Samuelsen SO, Torjusen H, Trogstad L, Alexander J 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):1162.
339. Rifas-Shiman SL, Rich-Edwards JW, Kleinman KP, Oken E, Gillman MW. Dietary quality during pregnancy varies by maternal characteristics in Project Viva: a US cohort. *J Am Diet Assoc* 2009;109(6):1004–11.
340. Radesky JS, Oken E, Rifas-Shiman SL, Kleinman KP, Rich-Edwards JW, Gillman MW. Diet during early pregnancy and development of gestational diabetes. *Pediatr Perinat Epidemiol* 2008;22(1):47–59.
341. Tieu J, Crowther CA, Middleton P. Dietary advice in pregnancy for preventing gestational diabetes mellitus. *Cochrane Database Syst Rev* 2008(2):CD006674.
342. Zhang C, Liu S, Solomon CG, Hu FB. Dietary fiber intake, dietary glycemic load, and the risk for gestational diabetes mellitus. *Diabetes Care* 2006;29(10):2223–30.
343. Chatzi L, Torrent M, Romieu I, Garcia-Esteban R, Ferrer C, Vioque J et al. Mediterranean diet in pregnancy is protective for wheeze and atopy in childhood. *Thorax* 2008;63(6):507.
344. De Batlle J, Garcia Aymerich J, Barraza Villarreal A, Antó J, Romieu I. Mediterranean diet is associated with reduced asthma and rhinitis in Mexican children. *Allergy* 2008;63(10):1310–16.
345. Hattevig G, Kjellman B, Sigurs N, Björkstén B, Kjellman N. Effect of maternal avoidance of eggs, cow's milk and fish during lactation upon allergic manifestations in infants. *Clin Exp Allergy* 1989;19(1):27–32.
346. Lange NE, Rifas-Shiman SL, Camargo CA, Gold DR, Gillman MW, Litonjua AA. Maternal dietary pattern during pregnancy is not associated with recurrent wheeze in children. *J Allergy Clin Immunol* 2010;126(2):250–55.
347. Shaheen SO, Northstone K, Newson RB, Emmett PM, Sherriff A, Henderson AJ. Dietary patterns in pregnancy and respiratory and atopic outcomes in childhood. *Thorax* 2009;64(5):411–7.
348. Robinson S, Marriott L, Poole J, Crozier S, Borland S, Lawrence W et al. Dietary patterns in infancy: the importance of maternal and family influences on feeding practice. *Br J Nutr* 2007;98(5):1029–37.
349. Food Standards Australia New Zealand. Listeria. 2011 (cited 29 March 2011). <http://www.foodstandards.gov.au/consumerinformation/listeria/>. <http://www.patonsyarns.com/pattern.ph>
350. Cullen G, O'Donoghue D. Constipation and pregnancy. *Best Pract Res Clin Gastroenterol* 2007;21(5):807–18.
351. National Health and Medical Research Council. Infant feeding guidelines for health workers Canberra: Commonwealth of Australia; 2011.
352. Lee AJ, O'Dea K, Mathews JD. Apparent dietary intake in remote Aboriginal communities. *Aust J Public Health* 1994;18(2):190–7.
353. Brimblecombe JK, O'Dea K. The role of energy cost in food choices for an Aboriginal population in northern Australia. *Med J Aust* 2009;190(10):549–51.
354. Ball K, Timperio A, Crawford D. Neighbourhood socioeconomic inequalities in food access and affordability. *Health & place* 2009;15(2):578–85.

355. Burns C, Inglis A. 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):377-85.
356. Landrigan T, Pollard C. Food Access and Cost Survey (FACS), Western Australia, 2010. Perth: Department of Health, WA, 2011.
357. NT Department of Health and Community Services. NT Market basket survey, 2006. NT Department of Health and Community Services; 2007.
358. American Dietetic Association. Position of the American Dietetic Association: Vegetarian Diets. *J Am Diet Assoc* 2009;109(4):266-82.
359. Marsh K, Zeuschner C, Saunders A, Reid M. Meeting nutritional needs on a vegetarian diet. *Aust Fam Physician* 2009;38(8):600-2.
360. U.S. Department of Health and Human Services, U.S. Department of Agriculture. Dietary guidelines for Americans 2005. 6th edition ed. Washington DC: U.S. Government Printing Office; 2005.
361. Martinez-Gonzalez MA et al. Low consumption of fruit and vegetables and risk of chronic disease: a review of the epidemiological evidence and temporal trends among Spanish graduates. *Public Health Nutr* 2011;14(12A):2309-15.
362. Dauchet L, Amouyel P, Hercberg S, Dallongeville J, Dauchet L, Amouyel P et al. Fruit and vegetable consumption and risk of coronary heart disease: a meta-analysis of cohort studies. *J Nutr* 2006;136(10):2588-93.
363. He FJ, Nowson CA, Lucas M, MacGregor GA. 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):717-28.
364. Joshipura KJ, Hung HC, Li TY, Hu FB, Rimm EB, Stampfer MJ et al. Intakes of fruits, vegetables and carbohydrate and the risk of CVD. *Public Health Nutr* 2009;12(1):415-21.
365. Rastogi T, Reedy KS, Vaz M, Spiegelman D, Prabhakaran D, Willett WC et al. Diet and risk of ischemic heart disease in India. *Am J Clin Nutr* 2004;79(4):582-92.
366. Rodríguez-Rodríguez E, Ortega RM, López-Sobaler AM, Andrés P, Aparicio A, Bermejo LM et al. Restricted-energy diets rich in vegetables or cereals improve cardiovascular risk factors in overweight/obese women. *Nutr Res* 2007;27(6):313-20.
367. Takachi R, Inoue M, Ishihara J, Kurahashi N, Iwasaki M, Sasazuki S 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):59-70.
368. Dauchet L, Amouyel P, Dallongeville J. Fruit and vegetable consumption and risk of stroke: a meta-analysis of cohort studies. *Neurology* 2005;65(8):1193-7.
369. He FJ, Nowson CA, MacGregor GA. Fruit and vegetable consumption and stroke: meta-analysis of cohort studies. *Lancet* 2006;367(9507):320-6.
370. Villegas R, Shu XO, Gao YT, Yang G, Elasy T, Li H et al. Vegetable but not fruit consumption reduces the risk of type 2 diabetes in Chinese women. *J Nutr* 2008;138(3):574-80.
371. Harding AH, Wareham NJ, Bingham SA, Khaw K, Luben R, Welch A 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):1493-9.
372. Hamer M, Chida Y. Intake of fruit, vegetables, and antioxidants and risk of type 2 diabetes: systematic review and meta-analysis. *J Hypertens* 2007;25(12):2361-9.
373. Heck JE, Sapkota A, Vendenhan G, Roychowdhury S, Dikshit RP, Jetly DH et al. Dietary risk factors for hypopharyngeal cancer in India. *Cancer Causes Control* 2008;19(10):1329-37.

374. Kreimer AR, Randi G, Herrero R, Castellsague X, La Vecchia C, Franceschi S. 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):2293–7.
375. Gallicchio L, Matanoski G, Tao XG, Chen L, Lam TK, Boyd K et al. Adulthood consumption of preserved and nonpreserved vegetables and the risk of nasopharyngeal carcinoma: a systematic review. *Int J Cancer* 2006;119(5):1125–35.
376. Guneri P, Cankaya H, Yavuzer A, Guneri EA, Erisen L, Ozkul D et al. Primary oral cancer in a Turkish population sample: association with sociodemographic features, smoking, alcohol, diet and dentition. *Oral Oncol* 2005;41(10):1005–12.
377. Escribano Uzcudun A, Rabanal Retolaza I, Garcia Grande A, Miralles Olivar L, Garcia Garcia A, Gonzalez Baron M et al. Pharyngeal cancer prevention: evidence from a case-control study involving 232 consecutive patients. *J Laryngol Otol* 2002;116(7):523–31.
378. George SM, Park Y, Leitzmann MF, Freedman ND, Dowling EC, Reedy J et al. Fruit and vegetable intake and risk of cancer: a prospective cohort study. *Am J Clin Nutr* 2009;89(1):347.
379. Freedman ND, Park Y, Subar AF, Hollenbeck AR, Leitzmann MF, Schatzkin A et al. Fruit and vegetable intake and esophageal cancer in a large prospective cohort study. *Int J Cancer* 2007;121(12):2753–60.
380. Anderson LA, Watson RG, Murphy SJ, Johnston BT, Comber H, McGuigan J et al. Risk factors for Barrett's oesophagus and oesophageal adenocarcinoma: results from the FINBAR study. *World J Gastroenterol* 2007;13(10):1585–94.
381. González CA, Pera G, Agudo A, Bueno de Mesquita HB, Ceroti M, Boeing H 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):2559–66.
382. Etminan M, Takkouche B, Caamano-Isorna F. 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):340–5.
383. Stram DO, Hankin JH, Wilkens LR, Park S, Henderson BE, Nomura AM 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):1193–207.
384. Koushik A, Hunter DJ, Spiegelman D, Anderson KE, Arslan AA, Beeson WL et al. Fruits and vegetables and ovarian cancer risk in a pooled analysis of 12 cohort studies. *Cancer Epidemiol Biomarkers Prev* 2005;14(9):2160–7.
385. Schulz M, Lahmann PH, Boeing H, Hoffmann K, Allen N, Key TJA 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):2531.
386. Bandera EV, Kushi LH, Moore DF, Gifkins DM, McCullough ML. Fruits and vegetables and endometrial cancer risk: a systematic literature review and meta-analysis. *Nutr Cancer* 2007;58(1):6–21.
387. Bravi F, Scotti L, Bosetti C, Zucchetto A, Talamini R, Montella M et al. Food groups and endometrial cancer risk: a case-control study from Italy. *Am J Obstet Gynecol* 2009;200(3):293 e1–7.
388. Yeh M, Moysich KB, Jayaprakash V, Rodabaugh KJ, Graham S, Brasure JR et al. Higher intakes of vegetables and vegetable-related nutrients are associated with lower endometrial cancer risks. *J Nutr* 2009;139(2):317–22.
389. Koushik A, Hunter DJ, Spiegelman D, Beeson WL, van den Brandt PA, Buring JE et al. Fruits, vegetables, and colon cancer risk in a pooled analysis of 14 cohort studies. *J Natl Cancer Inst* 2007;99(19):1471–83.
390. Lam TK, Gallicchio L, Linolsley K, Shiels M, Hammond E, Tao XG et al. Cruciferous vegetable consumption and lung cancer risk: a systematic review. *Cancer Epidemiol Biomarkers Prev* 2009;18(1):184–95.
391. Cho E, Seddon JM, Rosner B, Willett WC, Hankinson SE, 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):883–92.

392. Harland JL, Haffner TA. 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):13–27.
393. Deibert P, König D, Schmidt-Trucksass A, Zaenker K, Frey I, Landmann U et al. Weight loss without losing muscle mass in pre-obese and obese subjects induced by a high-soy-protein diet. *Int J Obes (Lond)* 2004;28(10):1349–52.
394. Liao FH, Shieh MJ, Yang SC, Lin SH, Chien YW. 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):551–6.
395. St-Onge MP, Claps N, Wolper C, Heymsfield SB. Supplementation with soy-protein-rich foods does not enhance weight loss. *J Am Diet Assoc* 2007;107(3):500–05.
396. Akhter M, Inoue M, Kurahashi N, Iwasaki M, Sasazuki S, Tsugane S. 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):2128–35.
397. Deneo-Pellegrini H, Boffetta P, De Stefani E, Ronco A, Brennan P, Mendilaharsu M. Plant foods and differences between colon and rectal cancers. *Eur J Cancer Prev* 2002;11(4):369.
398. Michels KB, Giovannucci E, Chan AT, Singhania R, Fuchs CS, Willett WC. Fruit and vegetable consumption and colorectal adenomas in the Nurses' Health Study. *Cancer Res* 2006;66(7):3942–53.
399. Oba S, Nagata C, Shimizu N, Shimizu H, Kametani M, Takeyama N et al. Soy product consumption and the risk of colon cancer: a prospective study in Takayama, Japan. *Nutr Cancer* 2007;57(2):151–7.
400. Yang G, Shu XO, Li H, Chow WH, Cai H, Zhang X et al. Prospective cohort study of soy food intake and colorectal cancer risk in women. *Am J Clin Nutr* 2009;89(2):577–83.
401. Ward HA, Kuhnle GGC, Mulligan AA, Lentjes MAH, Luben RN, Khaw KT. 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):440.
402. Riboli E, Norat T. Epidemiologic evidence of the protective effect of fruit and vegetables on cancer risk. *Am J Clin Nutr* 2003;78(3 Suppl):559S–69S.
403. Gaudet MM, Britton JA, Kabat GC, Steck-Scott S, Eng SM, Teitelbaum SL 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):1485–94.
404. Hermann S, Linseisen J, Chang-Claude J. Nutrition and breast cancer risk by age 50: a population-based case-control study in Germany. *Nutr Cancer* 2002;44(1):23–34.
405. 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):513–24.
406. Malin AS, Qi D, Shu XO, Gao YT, Friedman JM, Jin F et al. Intake of fruits, vegetables and selected micronutrients in relation to the risk of breast cancer. *Int J Cancer* 2003;105(3):413–8.
407. van Gils CH, Peeters PH, Bueno-de-Mesquita HB, Boshuizen HC, Lahmann PH, Clavel-Chapelon F et al. Consumption of vegetables and fruits and risk of breast cancer. *J Am Med Assoc* 2005;293(2):183–93.
408. McCullough ML, Bandera EV, Patel R, Patel AV, Gansler T, Kushi LH et al. A prospective study of fruits, vegetables, and risk of endometrial cancer. *Am J Epidemiol* 2007;166(8):902–11.
409. Oh S-Y, Lee JH, Jang DK, Heo SC, Kim HJ. Relationship of nutrients and food to colorectal cancer risk in Koreans. *Nutr Res* 2005;25(9):805–13.
410. Sato Y, Tsubono Y, Nakaya N, Ogawa K, Kurashima K, Kuriyama S et al. Fruit and vegetable consumption and risk of colorectal cancer in Japan: The Miyagi Cohort Study. *Public Health Nutr* 2005;8(3):309–14.
411. Wu H, Dai Q, Shrubsole MJ, Ness RM, Schlundt D, Smalley WE et al. Fruit and vegetable intakes are associated with lower risk of colorectal adenomas. *J Nutr* 2009;139(2):340–4.

412. Hamilton CA. Low-density lipoprotein and oxidised low-density lipoprotein: their role in the development of atherosclerosis. *Pharmacol Ther* 1997;74(1):55–72.
413. Thompson HJ, Heimendinger J, Diker A, O'Neill C, Haeghele A, Meinecke B 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):2207.
414. Wannamethee SG, Lowe GD, Rumley A, Bruckdorfer KR, Whincup PH. Associations of vitamin C status, fruit and vegetable intakes, and markers of inflammation and hemostasis. *Am J Clin Nutr* 2006;83(3):567–74; quiz 726–7.
415. Jialal I, Singh U. Is vitamin C an antiinflammatory agent? *Am J Clin Nutr* 2006;83(3):525.
416. Tavani A, La Vecchia C. [beta]-Carotene and risk of coronary heart disease. A review of observational and intervention studies. *Biomed Pharmacother* 1999;53(9):409–16.
417. Messina MJ. Legumes and soybeans: overview of their nutritional profiles and health effects. *Am J Clin Nutr* 1999;70(3):439S.
418. Nagata C. Factors to consider in the association between soy isoflavone intake and breast cancer risk. *J Epidemiol* 2010(0):1002160133.
419. Willett WC. Fruits, vegetables, and cancer prevention: turmoil in the produce section. *J Natl Cancer Inst* 2010;102(8):510–1.
420. Giovannucci E. Alcohol, one-carbon metabolism, and colorectal cancer: recent insights from molecular studies. *J Nutr* 2004;134(9):2475S–81S.
421. Zhang SM, Willett WC, Selhub J, Hunter DJ, Giovannucci EL, Holmes MD et al. Plasma folate, vitamin B₆, vitamin B₁₂, homocysteine, and risk of breast cancer. *J Natl Cancer Inst* 2003;95(5):373–80.
422. García-Lafuente A, Guillaumon E, Villares A, Rostagno MA, Martínez JA. Flavonoids as anti-inflammatory agents: implications in cancer and cardiovascular disease. *Inflammation Res* 2009;58(9):537–52.
423. Lippman SM, Klein EA, Goodman PJ, Lucia MS, Thompson IM, Ford LG 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):39–51.
424. Paolini M, Abdel-Rahman SZ, Sapone A, Pedullini GF, Perocco P, Cantelli-Forti G et al. Beta-carotene: a cancer chemopreventive agent or a co-carcinogen? *Mutat Res* 2003;543(3):195–200.
425. Khan N, Afaq F, Mukhtar H. Cancer chemoprevention through dietary antioxidants: progress and promise. *Antioxidants Redox Signaling* 2008;10(3):475–510.
426. Bingham SA, Day NE, Luben R, Ferrari P, Slimani N, Norat T 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):1496–501.
427. Inskip HM, Crozier SR, Godfrey KM, Borland SE, Cooper C, Robinson SM. Women's compliance with nutrition and lifestyle recommendations before pregnancy: general population cohort study. *Br Med J* 2009;338.
428. New Zealand Ministry of Health. Food and nutrition guidelines for healthy pregnant and breastfeeding women: a background paper. Wellington: New Zealand Ministry of Health; 2006 (revised 2008).
429. Lussi A MB, Peter Shellis R, Wang X. Analysis of the erosive effect of different dietary substances and medications. *Br J Nutr* 2011;30:1-11.
430. Birch L. Development of food preferences. *Annual Review of Nutrition* 1999;19:41-62.
431. Tasmanian School Canteen Association Inc. The Cool Canteen Accreditation Program. 2011 (cited 30 June 2011). <http://www.tascanteenassn.org.au/coolcap>. <http://www.patonsyarns.com/pattern.ph>
432. Office of Learning and Teaching. Go for your life school canteens and other school food services policy. Melbourne: Department of Education; 2006.

433. Northern Territory Government. Canteen, nutrition and health eating policy. In: Training DoEa, editor. Darwin 2009.
434. New South Wales Department of Health, New South Wales Department of Education and Training. Fresh tastes@ school NSW healthy school canteen strategy. Sydney: NSW Department of Health and NSW Department of Education and Training,; 2006.
435. Government of Western Australia. Healthy food and drink. Perth: Department of Education, 2008.
436. Government of South Australia. Right bite, healthy food and drink supply strategy for south Australian schools and preschools. Adelaide: Government of South Australia, 2008.
437. Dick M, Farquharson R, Bright M, Turner K, Lee AJ. Smart choices – healthy food and drink supply strategy for Queensland schools: evaluation report. Brisbane: Queensland health and department of education and training, 2009.
438. Commonwealth Department of Health and Ageing. Guidelines for healthy foods and drinks supplied in school canteens. Canberra: Commonwealth of Australia, 2010. <http://www.health.gov.au/internet/main/publishing.nsf/Content/phd-nutrition-canteens>
439. AIHW. Australia's Health 2010. In: AIHW, editor. Canberra: AIHW; 2010.
440. McDermott R, Campbell S, Li M, McCulloch B. 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):2143-49.
441. Food Standards Australia New Zealand. Australia New Zealand Food Standards Code. Canberra: FSANZ, 2011.
442. Jacobs DR Jr, Meyer KA, Kushi LH, AR. F. Whole-grain intake may reduce the risk of ischemic heart disease death in postmenopausal women: the Iowa Women's Health Study. Am J Clin Nutr 1998;68(2):248-57.
443. Anderson JW. Whole grains protect against atherosclerotic cardiovascular disease. Proc Nutr Soc 2003;62(1):135-42.
444. Berg A, König D, Deibert P, Grathwohl D, Baumstark MW, Franz IW. 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):306-11.
445. De Moura F. Whole grain intake and cardiovascular disease and whole grain intake and diabetes: A review. Bethesda, MD: Life Sciences Research Organization, 2008.
446. Djousse L, Gaziano JM. Breakfast cereals and risk of heart failure in the physicians' health study I. Arch Intern Med 2007;167(19):2080-5.
447. Erkkila AT, Herrington DM, Mozaffarian D, Lichtenstein AH. 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):94-101.
448. Flight I, Clifton P. 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): 1145-59.
449. Jacobs DR, Jr., Gallaher DD. Whole grain intake and cardiovascular disease: a review. Curr Atheroscler Rep 2004;6(6):415-23.
450. Katcher HI, Legro RS, Kunselman AR, Gillies PJ, Demers LM, Bagshaw DM 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):79-90.
451. Kelly SA, Summerbell CD, Brynes A, Whittaker V, Frost G. Wholegrain cereals for coronary heart disease. Cochrane Database Syst Rev 2007(2):CD005051.
452. Liu S, Buring JE, Sesso HD, Rimm EB, Willett WC, Manson JAE. A prospective study of dietary fiber intake and risk of cardiovascular disease among women. J Am Coll Cardiol 2002;39(1):49-56.

453. Liu S, Sesso HD, Manson JAE, Willett WC, Buring JE. Is intake of breakfast cereals related to total and cause-specific mortality in men? *Am J Clin Nutr* 2003;77(3):594.
454. Negri E, La Vecchia C, Pelucchi C, Bertuzzi M, Tavani A. Fiber intake and risk of nonfatal acute myocardial infarction. *Eur J Clin Nutr* 2003;57(3):464–70.
455. Nettleton JA, Steffen LM, Loefer LR, Rosamond WD, Folsom AR. 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):1881–87.
456. Pereira MA, O'Reilly E, Augustsson K, Fraser GE, Goldbourt U, Heitmann BL et al. Dietary fiber and risk of coronary heart disease: a pooled analysis of cohort studies. *Arch Intern Med* 2004;164(4):370–6.
457. Rave K, Roggen K, Dellweg S, Heise T, Tom Dieck H. 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):929–36.
458. Truswell AS. Cereal grains and coronary heart disease. *Eur J Clin Nutr* 2002;56(1):1–14.
459. de Munter JS, Hu FB, Spiegelman D, Franz M, van Dam RM. Whole grain, bran, and germ intake and risk of type 2 diabetes: a prospective cohort study and systematic review. *PLoS Med* 2007;4(8):e261.
460. Henry CJK, Lightowler H, Tyde E, Skeath R. Use of low-glycaemic index bread to reduce 24-h blood glucose: implications for dietary advice to non-diabetic and diabetic subjects. *Int J Food Sci Nutrition* 2006;57(3-4):273–78.
461. Hodge AM, English DR, O'Dea K, Giles GG. Glycemic index and dietary fiber and the risk of type 2 diabetes. *Diabetes Care* 2004;27(11):2701–6.
462. Krishnan S, Rosenberg L, Singer M, Hu FB, Djousse L, Cupples LA 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):2304–9.
463. Pereira MA, Jacobs DR, Jr., Pins JJ, Raatz SK, Gross MD, Slavin JL et al. Effect of whole grains on insulin sensitivity in overweight hyperinsulinemic adults. *Am J Clin Nutr* 2002;75(5):848–55.
464. Priebe MG, van Binsbergen JJ, de Vos R, Vonk RJ. Whole grain foods for the prevention of type 2 diabetes mellitus. *Cochrane Database Syst Rev* 2008(1):CD006061.
465. Venn BJ, Mann JI. Cereal grains, legumes and diabetes. *Eur J Clin Nutr* 2004;58(11):1443–61.
466. Andersson A, Tengblad S, Karlstrom B, Kamal-Eldin A, Landberg R, Basu S 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):1401–7.
467. Asano T, McLeod RS. Dietary fibre for the prevention of colorectal adenomas and carcinomas. *Cochrane Database Syst Rev* 2002(2):CD003430.
468. Jacobs ET, Lanza E, Alberts DS, Hsu CH, Jiang R, Schatzkin A et al. Fiber, sex, and colorectal adenoma: results of a pooled analysis. *Am J Clin Nutr* 2006;83(2):343–9.
469. La Vecchia C, Chatenoud L, Negri E, Franceschi S. Session: whole cereal grains, fibre and human cancer wholegrain cereals and cancer in Italy. *Proc Nutr Soc* 2003;62(1):45–9.
470. Peters U, Sinha R, Chatterjee N, Subar AF, Ziegler RG, Kulleroff M et al. Dietary fibre and colorectal adenoma in a colorectal cancer early detection programme. *Lancet* 2003;361(9368):1491–95.
471. Schatzkin A, Mouw T, Park Y, Subar AF, Kipnis V, Hollenbeck 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):1353–60.
472. Chan D, Lau R AD, R V, D G, Kampman E, T N. P1-109The WCRF/AICR continuous update project: dietary fibre intake and colorectal cancer incidence. *J Epidemiol Community Health* 2011;65(A97).

473. Aune D, Chan DSM, Lau R, Vieira R, Greenwood DC, Kampman E et al. Dietary fibre, whole grains, and risk of colorectal cancer: systematic review and dose-response meta-analysis of prospective studies. *BMJ* 2011;343:d6617.
474. Theuvsen E, Mensink RP. Water-soluble dietary fibers and cardiovascular disease. *Physiol Behav* 2008;94(2):285–92.
475. Kerckhoffs DA, Brouns F, Hornstra G, Mensink RP. 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):2494–505.
476. Beck EJ, Tosh SM, Batterham MJ, Tapsell LC, Huang XF. Oat beta-glucan increases postprandial cholecystokinin levels, decreases insulin response and extends subjective satiety in overweight subjects. *Mol Nutr Food Res* 2009;53(10):1343–51.
477. Toden S, Belobrajdic DP, Bird AR, Topping DL, Conlon MA. 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):454.
478. Lazaridou A, Biliaderis C. Molecular aspects of cereal [beta]-glucan functionality: physical properties, technological applications and physiological effects. *Journal of Cereal Science* 2007;46(2):101–18.
479. Liu RH. Whole grain phytochemicals and health. *J Cereal Sci* 2007;46(3):207–19.
480. Slavin J. Why whole grains are protective: biological mechanisms. *Proc Nutr Soc* 2003;62(1):129–34.
481. Dodd H, Williams S, Brown R, B V. Calculating meal glycaemic index by using measured and published food values compared with directly measured meal glycaemic index. *The American Journal of Clinical Nutrition* 2011;94(4):992–96.
482. De-Regil LM, Fernández-Gaxiola AC, Dowswell T, Peña-Rosas JP. Effects and safety of periconceptional folate supplementation for preventing birth defects. *Cochrane Database of Systematic Reviews* 2010;DOI: 10.1002/14651858.CD007950.pub2.
483. Food Standards Australia and New Zealand. Folic acid/Folate. 2011 (cited 12 May 2011). <http://www.foodstandards.gov.au/consumerinformation/adviceforpregnantwomen/folicacidfolateandpr4598.cfm>. <http://www.patonsyarns.com/pattern.ph>
484. National Institute for Clinical Studies. Encouraging periconceptional use of folic acid supplements. Melbourne: National Institute for Clinical Studies, 2005. http://www.nhmrc.gov.au/_files_nhmrc/file/nics/material_resources/evidence_practice_gaps_report_volume_2.pdf
485. Mooney BD et al. Seafood the good food II: oil profiles for further Australian seafoods and influencing factors. CSIRO Marine Research and Fisheries Research & Development Corporation Australia; 2002.
486. Nichols PD et al. Seafood the good food: the oil (fat) content and composition of Australian commercial fishes, shellfishes and crustaceans. CSIRO Marine Research Australia; 1998.
487. Sinha R, Cross AJ, Graubard BI, Leitzmann MF, Schatzkin A. Meat intake and mortality: a prospective study of over half a million people. *Arch Intern Med* 2009;169(6):562–71.
488. Brink M, Weijenberg MP, de Goeij AF, Roemen GM, Lentjes MH, de Bruine AP 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):1310–20.
489. Chan AT, Tranah GJ, Giovannucci EL, Willett WC, Hunter DJ, Fuchs CS. Prospective study of N-acetyltransferase-2 genotypes, meat intake, smoking and risk of colorectal cancer. *Int J Cancer* 2005;115(4):648–52.
490. Kabat GC, Miller AB, Jain M, Rohan TE. A cohort study of dietary iron and heme iron intake and risk of colorectal cancer in women. *Br J Cancer* 2007;97(1):118–22.
491. Larsson SC, Wolk A. Meat consumption and risk of colorectal cancer: a meta-analysis of prospective studies. *Int J Cancer* 2006;119(11):2657–64.

492. Lee SA, Shu XO, Yang G, Li H, Gao YT, Zheng W et al. Animal origin foods and colorectal cancer risk: a report from the Shanghai Women's Health Study. *Nutr Cancer* 2009;61(2):194–205.
493. Norat T, Lukanova A, Ferrari P, Riboli E. Meat consumption and colorectal cancer risk: dose-response meta-analysis of epidemiological studies. *Int J Cancer* 2002;98(2):241–56.
494. Oba S, Shimizu N, Nagata C, Shimizu H, Kametani M, Takeyama N 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):260–67.
495. Sato Y, Nakaya N, Kuriyama S, Nishino Y, Tsubono Y, Tsuji I. Meat consumption and risk of colorectal cancer in Japan: the Miyagi Cohort Study. *Eur J Cancer Prev* 2006;15(3):211.
496. Seow A, Quah SR, Nyam D, Straughan PT, Chua T, Aw TC. Food groups and the risk of colorectal carcinoma in an Asian population. *Cancer* 2002;95(11):2390–96.
497. Garcia-Closas R, Garcia-Closas M, Kogevinas M, Malats N, Silverman D, Serra C et al. Food, nutrient and heterocyclic amine intake and the risk of bladder cancer. *Eur J Cancer* 2007;43(11):1731–40.
498. Larsson SC, Johansson JE, Andersson SO, Wolk A. Meat intake and bladder cancer risk in a Swedish prospective cohort. *Cancer Causes Control* 2009;20(1):35–40.
499. Koutros S, Cross AJ, Sandler DP, Hoppin JA, Ma X, Zheng T et al. Meat and meat mutagens and risk of prostate cancer in the Agricultural Health Study. *Cancer Epidemiol Biomarkers Prev* 2008;17(1):80–7.
500. Mori M, Masumori N, Fukuta F, Nagata Y, Sonoda T, Sakauchi F et al. Traditional Japanese diet and prostate cancer. *Mol Nutr Food Res* 2009;53(2):191–200.
501. Chan JM, Wang F, Holly EA. Pancreatic cancer, animal protein and dietary fat in a population-based study, San Francisco Bay Area, California. *Cancer Causes Control* 2007;18(10):153–67.
502. Iso H, Kobayashi M, Ishihara J, Sasaki S, Okada K, Kita Y 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):195.
503. Virtanen JK, Mozaffarian D, Chiuve SE, Rimm EB. Fish consumption and risk of major chronic disease in men. *Am J Clin Nutr* 2008;88(6):1612–25.
504. Whelton SP, He J, Whelton PK, Muntner P. Meta-analysis of observational studies on fish intake and coronary heart disease. *Am J Cardiol* 2004;93(9):1119–23.
505. Yamagishi K, Iso H, Date C, Fukui M, Wakai K, Kikuchi S 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):988–96.
506. Folsom AR, Demissie Z. Fish intake, marine omega-3 fatty acids, and mortality in a cohort of postmenopausal women. *Am J Epidemiol* 2004;160(10):1005–10.
507. He K, Song Y, Davi GL, Liu K, Van Horn L, Dyer AR et al. Accumulated evidence on fish consumption and coronary heart disease mortality: a meta-analysis of cohort studies. *Circulation* 2004;109(22):2705–11.
508. Jarvinen R, Knekt P, Rissanen H, Reunanen A. 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):824–9.
509. Kaushik S, Wang JJ, Flood V, Liew G, Smith W, Mitchell P. Frequency of fish consumption, retinal microvascular signs and vascular mortality. *Microcirculation* 2008;15(1):27–36.
510. Konig A, Bouzan C, Cohen JT, Connor WE, Kris-Etherton PM, Gray GM et al. A quantitative analysis of fish consumption and coronary heart disease mortality. *Am J Prev Med* 2005;29(4):335–46.
511. Nakamura Y, Ueshima H, Okamura T, Kadowaki T, Hayakawa T, Kita 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):239–45.

512. Ness A, Maynard M, Frankel S, Davey Smith G, Frobisher C, Leary S et al. Diet in childhood and adult cardiovascular and all cause mortality: the Boyd Orr cohort. *Heart* 2005;91(7):994.
513. Streppel MT, Ocke MC, Boshuizen HC, Kok FJ, Kromhout D. 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):2024–30.
514. Bouzan C, Cohen JT, Connor WE, Kris-Etherton PM, Gray GM, Konig A et al. A quantitative analysis of fish consumption and stroke risk. *Am J Prev Med* 2005;29(4):347–52.
515. Ding EL, Mozaffarian D. Optimal dietary habits for the prevention of stroke. *Semin Neurol* 2006;26(1):11–23.
516. Gochfeld M, Burger J. Good fish/bad fish: a composite benefit-risk by dose curve. *Neurotoxicology* 2005;26(4):511–20.
517. Barberger-Gateau P, Raffaitin C, Letenneur L, Berr C, Tzourio C, Dartigues JF et al. Dietary patterns and risk of dementia: the three-city cohort study. *Neurology* 2007;69(20):1921–30.
518. Huang T, Zandi P, Tucker K, Fitzpatrick A, Kuller L, Fried L et al. Benefits of fatty fish on dementia risk are stronger for those without APOE 4. *Neurology* 2005;65(9):1409.
519. Kalmijn S, Van Boxtel M, Ocke M, Verschuren W, Kromhout D, Launer L. Dietary intake of fatty acids and fish in relation to cognitive performance at middle age. *Neurology* 2004;62(2):275.
520. Morris MC, Evans DA, Tangney CC, Bienias JL, Wilson RS. Fish consumption and cognitive decline with age in a large community study. *Arch Neurol* 2005;62(12):1849–53.
521. van Gelder BM, Tijhuis M, Kalmijn S, Kromhout D. 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):1142–7.
522. Weih M, Wiltfang J, Kornhuber J. Non-pharmacologic prevention of Alzheimer's disease: nutritional and life-style risk factors. *J Neural Transm* 2007;114(9):187–97.
523. Larrieu S, Letenneur L, Helmer C, Dartigues J, Barberger-Gateau P. Nutritional factors and risk of incident dementia in the PAQUID longitudinal cohort. *J Nutrition Health Aging* 2004;8(3):150–54.
524. Maclean CH, Issa AM, Newberry SJ, Mojica WA, Morton SC, Garland RH et al. Effects of omega-3 fatty acids on cognitive function with aging, dementia, and neurological diseases. *Evid Rep Technol Assess (Summ)* 2005(114):1–3.
525. Schiepers OJ, de Groot RH, Jolles J, van Boxtel MP. Plasma phospholipid fatty acid status and depressive symptoms: association only present in the clinical range. *J Affect Disord* 2009;118(1-3):209–14.
526. Sontrop J, Campbell MK. Omega-3 polyunsaturated fatty acids and depression: a review of the evidence and a methodological critique. *Prev Med* 2006;42(1):4–13.
527. Sanchez-Villegas A, Henriquez P, Figueiras A, Ortuno F, Lahortiga F, Martinez-Gonzalez MA et al. Long chain omega-3 fatty acids intake, fish consumption and mental disorders in the SUN cohort study. *Eur J Nutrition* 2007;46(6):337–46.
528. Colangelo LA, He K, Whooley MA, Davila ML, Liu K. Higher dietary intake of long-chain omega-3 polyunsaturated fatty acids is inversely associated with depressive symptoms in women. *Nutrition* 2009;25(10):1011–9.
529. Kyrozis A, Psaltopoulou T, Stathopoulos P, Trichopoulos D, Vassilopoulos D, Trichopoulou A. Dietary lipids and geriatric depression scale score among elders: The EPIC-Greece cohort. *J Psychiatr Res* 2009;43(8):763–69.
530. Chong EW, Kreis AJ, Wong TY, Simpson JA, Guymer RH. 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):826–33.
531. Djousse L, Gaziano JM, Buring JE, Lee IM. Egg consumption and risk of type 2 diabetes in men and women. *Diabetes Care* 2009;32(2):295–300.

532. Sauvaget C, Nagano J, Allen N, Grant EJ, Beral V. Intake of animal products and stroke mortality in the Hiroshima/Nagasaki Life Span Study. *Int J Epidemiol* 2003;32(4):536-43.
533. Kramer Michael S, Kakuma R. 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).
534. Chakrabarty G, Bijlani RL, Mahapatra SC, Mehta N, Lakshmy R, Vashisht S 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):492-8.
535. Chakrabarty G, Manjunatha S, Bijlani RL, Ray RB, Mahapatra SC, Mehta N et al. The effect of ingestion of egg on the serum lipid profile of healthy young Indians. *Indian J Physiol Pharmacol* 2004;48(3):286-92.
536. Djousse L, Gaziano JM. Egg consumption in relation to cardiovascular disease and mortality: the Physicians' Health Study. *Am J Clin Nutr* 2008;87(4):964-9.
537. Gillingham LG, Caston L, Leeson S, Hourtovenko K, Holub BJ. The effects of consuming docosahexaenoic acid (DHA)-enriched eggs on serum lipids and fatty acid compositions in statin-treated hypercholesterolemic male patients. *Food Research Int* 2005;38(10):1117-23.
538. Goodrow EF, Wilson TA, Houde SC, Vishwanathan R, Scollin PA, Handelsman G 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):2519.
539. Harman NL, Leeds AR, Griffin BA. Increased dietary cholesterol does not increase plasma low density lipoprotein when accompanied by an energy-restricted diet and weight loss. *Eur J Nutrition* 2008;47(6):287-93.
540. Katz DL, Evans MA, Nawaz H, Njike VY, Chan W, Comerford BP et al. Egg consumption and endothelial function: a randomized controlled crossover trial. *Int J Cardiol* 2005;99(1):65-70.
541. Lee JY, Lewis NM, Scheideler SE, Carr TP. Consumption of omega-3 fatty acid-enriched eggs and serum lipids in humans. *J Nutraceut Functional Med Foods* 2003;4(1):3-13.
542. Maki KC, Van Elswyk ME, McCarthy D, Seeley MA, Veith PE, Hess SP 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):357-68.
543. Makrides M, Hawkes JS, Neumann MA, Gibson RA. 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):1084-92.
544. Nakamura Y, Iso H, Kita Y, Ueshima H, Okada K, Konishi M 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):921-8.
545. Nakamura Y, Okamura T, Tamaki S, Kadowaki T, Hayakawa T, Kita 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, 1990 (NIPPON DATA90). *Am J Clin Nutr* 2004;80(1):58.
546. Ohman M, Akerfeldt T, Nilsson I, Rosen C, Hansson LO, Carlsson M et al. Biochemical effects of consumption of eggs containing omega-3 polyunsaturated fatty acids. *Ups J Med Sci* 2008;113(3):315-23.
547. Rose EL, Holub BJ. Effects of a liquid egg product containing fish oil on selected cardiovascular disease risk factors: A randomized crossover trial. *Food Res Int* 2006;39(8):910-16.
548. Sindelar CA, Scheerger SB, Plugge SL, Eskridge KM, Wander RC, Lewis NM. Serum lipids of physically active adults consuming omega-3 fatty acid-enriched eggs or conventional eggs. *Nutr Res* 2004;24(9):731-39.
549. VanderWal JS, Gupta A, Khosla P, Dhurandhar NV. Egg breakfast enhances weight loss. *Int J Obes (Lond)* 2008;32(10):1545-51.

550. Vislocky LM, Pikosky MA, Rubin KH, Vega-López S, Gaine PC, Martin WF 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. *J Nutritional Biochem* 2009;20(1):26–34.
551. Wenzel AJ, Gerweck C, Barbato D, Nicolosi RJ, Handelman GJ, Curran-Celentano J. A 12-wk egg intervention increases serum zeaxanthin and macular pigment optical density in women. *J Nutr* 2006;136(10):2562–73.
552. Alper CM, Mattes RD. Peanut consumption improves indices of cardiovascular disease risk in healthy adults. *J Am Coll Nutr* 2003;22(2):133–41.
553. Pan A et al. Red meat consumption and mortality: results from two prospective cohort studies. *Arch Intern Med* 2012;172(7):555–63.
554. Bernstein AM et al. Major dietary protein sources and risk of coronary heart disease in women. *Circulation* 2010;122:276–83.
555. Hebel DG et al. Red meat intake-induced increases in fecal water genotoxicity correlate with pro-carcinogenic gene expression changes in the human colon. *Food Chem Toxicol* 2012;50(2):95–103.
556. Kuhnle GGC, Story GW, Reda T, Mani AR, Moore KP, Lunn JC et al. Diet-induced endogenous formation of nitroso compounds in the GI tract. *Free Radical Biol Med* 2007;43(7):1040–47.
557. Bastie NM et al. Heme iron from meat and risk of colorectal cancer: a meta-analysis and a review of the mechanisms involved. *Cancer Prev Res* 2011;4(2):177–84.
558. Ma RW, Chapman K. A systematic review of the effect of diet in prostate cancer prevention and treatment. *J Hum Nutr Diet* 2009;22(3):187–99; quiz 200–2.
559. Sofi F, Cesari F, Abbate R, Gensini GF, Casini A. Adherence to Mediterranean diet and health status: meta-analysis. *Br Med J* 2008;337:a1344.
560. Ros E, Tapsell LC, Sabaté J. Nuts and berries for heart health. *Curr Atheroscler Rep* 2010;1–10.
561. Segura R, Javierre C, Lizarraga MA, Ros E. Other relevant components of nuts: phytosterols, folate and minerals. *Br J Nutr* 2006;96(Suppl 2):S36–44.
562. Ziemke F, Mantzoros CS. Adiponectin in insulin resistance: lessons from translational research. *Am J Clin Nutr* 2010;91(1):25S–61S.
563. Mattes RD, Dreher ML. Nuts and healthy body weight maintenance mechanisms. *Asia Pac J Clin Nutr* 2010;19(1):137–41.
564. Tapsell L, Batterham M, Tan SY, Warensjo E. 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):611.
565. Commonwealth Department of Agriculture Fisheries and Forestry. Australia's agriculture, fisheries and forestry at a glance 2010. Canberra: Commonwealth Department of Agriculture, Fisheries and Forestry, 2010. http://www.daff.gov.au/about/publications/australias_agriculture_fisheries_and_forestry_at_a_glance_2010
566. Australian Bureau of Agricultural and Resource Economics and Sciences. Fishery status reports 2010: Status of Fish Stocks and Fisheries managed by the Australian Government. In: Australian Government Department of Agriculture Fisheries and Forestry, editor. 2011.
567. Food Standards Australia New Zealand. Mercury in fish. 2011 (cited 29 March 2011). <http://www.foodstandards.gov.au/consumerinformation/adviceforpregnantwomen/mercuryinfish.cfm>. <http://www.patonsyarns.com/pattern.ph>
568. Greer FR, Sicherer SH, Burks A. 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):183.
569. Host A, Halken S, Muraro A, Dreborg S, Niggemann B, Aalberse R et al. Dietary prevention of allergic diseases in infants and small children. *Pediatr Allergy Immunol* 2008;19(1):1–4.

570. Sicherer SH, Burks AW. Maternal and infant diets for prevention of allergic diseases: understanding menu changes in 2008. *J Allergy Clin Immunol* 2008;122(1):29–33.
571. Prescott SL, Tang ML. The Australasian Society of Clinical Immunology and Allergy position statement: Summary of allergy prevention in children. *Med J Aust* 2005;182(9):464–7.
572. UK Royal College of Obstetricians and Gynaecologists. Nutrition in pregnancy (SAC opinion paper 18). 2010. Available from: <http://www.rcog.org.uk/nutrition-pregnancy-sac-opinion-paper-18>
573. Food Authority NSW. Sydney Harbour seafood and dioxins. 2010 (cited 29 March 2011). <http://www.foodauthority.nsw.gov.au/consumers/keeping-food-safe/special-care-foods/sydney-harbour-seafood/>. <http://www.patonsyarns.com/pattern.ph>
574. Food Standards Australia New Zealand. Dioxins in food: dietary exposure assessment and risk characterisation. Canberra: 2004. Available from: http://www.foodstandards.gov.au/_srcfiles/FINAL%20DEA-RC%20Report%20Dioxin%2024May04final.pdf
575. 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. Rome: Food and Agriculture Organization of the United Nations and the World Health Organization, 2010 25-29 January 2010. <http://www.who.int/foodsafety/chem/meetings/jan2010/en/index.html>
576. Elwood PC, Givens DI, Beswick AD, Fehily AM, Pickering JE, Gallacher J. 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):723S–34S.
577. He K, Merchant A, Rimm EB, Rosner BA, Stampfer MJ, Willett WC 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):777–82.
578. Alonso A, Beunza JJ, Delgado-Rodriguez M, Martinez JA, Martinez-Gonzalez MA. Low-fat dairy consumption and reduced risk of hypertension: the Seguimiento Universitario de Navarra (SUN) cohort. *Am J Clin Nutr* 2005;82(5):972–9.
579. Engberink MF, Geleijnse JM, de Jong N, Smit HA, Kok FJ, Verschuren WM. Dairy intake, blood pressure, and incident hypertension in a general Dutch population. *J Nutr* 2009;139(3):582–7.
580. Toledo E, Delgado-Rodriguez M, Estruch R, Salas-Salvadó J, Corella D, Gomez-Gracia 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):59–67.
581. Wang L, Manson JAE, Buring JE, Lee I. Dietary intake of dairy products, calcium, and vitamin D and the risk of hypertension in middle-aged and older women. *Hypertension* 2008;51(4):1073.
582. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities study. *Circulation* 2008;117(6):754–61.
583. Pittas AG, Lau J, Hu FB, Dawson-Hughes B. The role of vitamin D and calcium in type 2 diabetes. A systematic review and meta-analysis. *J Clin Endocrinol Metab* 2007;92(6):2017–29.
584. Berkey CS, Rockett HR, Field AE, Gillman MW, Colditz GA. Sugar-added beverages and adolescent weight change. *Obes Res* 2004;12(5):778–88.
585. Johnson L, Mandler AP, Jones LR, Emmett PM, Jebb SA. Is sugar-sweetened beverage consumption associated with increased fatness in children? *Nutrition* 2007;23(7-8):557–63.
586. Cho E, Smith-Warner SA, Spiegelman D, Beeson WL, van den Brandt PA, Colditz GA et al. Dairy foods, calcium, and colorectal cancer: a pooled analysis of 10 cohort studies. *J Natl Cancer Inst* 2004;96(13):1015–22.
587. Huncharek M, Muscat J, Kupelnick B. 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):47–69.

588. Sellers TA, Vierkant RA, Djeu J, Celis E, Wang AH, Kumar N et al. Unpasteurized milk consumption and subsequent risk of cancer. *Cancer Causes Control* 2008;19(8):805–11.
589. Lee JE, Hunter DJ, Spiegelman D, Adami HO, Bernstein L, van den Brandt PA 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):2246–53.
590. Alvarez-Leon EE, Roman-Vinas B, Serra-Majem L. Dairy products and health: a review of the epidemiological evidence. *Br J Nutr* 2006;96(Suppl 1):S94–9.
591. Missmer SA, Smith-Warner SA, Spiegelman D, Yaun SS, Adami HO, Beeson WL et al. Meat and dairy food consumption and breast cancer: a pooled analysis of cohort studies. *Int J Epidemiol* 2002;31(1):78–85.
592. Bandera EV, Kushi LH, Moore DF, Gifkins DM, McCullough ML. Consumption of animal foods and endometrial cancer risk: a systematic literature review and meta-analysis. *Cancer Causes Control* 2007;18(9):967–88.
593. Bonjour JP, Brandolini-Bunlon M, Boirie Y, Morel-Laporte F, Braesco V, Bertiere MC et al. Inhibition of bone turnover by milk intake in postmenopausal women. *Br J Nutr* 2008;100(4):66–74.
594. Hartman JW, Tang JE, Wilkinson SB, Tarnopolsky MA, Lawrence RL, Fullerton AV 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):373.
595. Javaher MK, Crozier SR, Harvey NC, Taylor P, Inskip HM, Godfrey KM et al. Maternal and seasonal predictors of change in calcaneal quantitative ultrasound during pregnancy. *J Clin Endocrinol Metab* 2005;90(9):5182–7.
596. Lanou AJ, Berkow SE, Barnard ND. Calcium, dairy products, and bone health in children and young adults: a reevaluation of the evidence. *Pediatrics* 2005;115(3):736–43.
597. Teegarden D, Legowski P, Gunther CW, McCabe GP, Peacock M, Lyle RM. Dietary calcium intake protects women consuming oral contraceptives from spine and hip bone loss. *J Clin Endocrinol Metab* 2005;90(9):5127–33.
598. Volek JS, Gomez AL, Scheett P, Sharman MJ, French DN, Rubin MR et al. Increasing fluid milk favorably affects bone mineral density responses to resistance training in adolescent boys. *J Am Diet Assoc* 2003;103(10):1353–6.
599. Feskanich D, Willett WC, Colditz GA. Calcium, vitamin D, milk consumption, and hip fractures: a prospective study among postmenopausal women. *Am J Clin Nutr* 2003;77(2):504.
600. Kanis JA, Johansson H, Odén A, De Laet C, Johnell O, Eisman JA et al. A meta-analysis of milk intake and fracture risk: low utility for case finding. *Osteoporos Int* 2005;16(7):799–804.
601. Griffith LE, Guyatt GH, Cook RJ, Bucher HC, Cook DJ. 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):84–92.
602. Clare DA, Swaisgood HE. Bioactive milk peptides: a prospectus. *J Dairy Sci* 2000;83(6):8187–95.
603. Bolland MJ, Avenell A, Baron JA, Grey A, MacLennan GS, Gamble GD et al. Effect of calcium supplements on risk of myocardial infarction and cardiovascular events: meta-analysis. *Br Med J* 2010;341:c3691.
604. Bolland MJ, Grey A, Avenell A, Gamble GD, Reid IR. 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:d2040.
605. Green JH, Booth C, Bunning R. 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):109–19.
606. Resnick L, DiFabio B, Marion R, James G, Laragh J. Dietary calcium modifies the pressor effects of dietary salt intake in essential hypertension. *J Hypertens* 1986;4(suppl 6):S679–S81.

607. Kalkwarf HJ, Khoury JC, Lanphear BP. Milk intake during childhood and adolescence, adult bone density, and osteoporotic fractures in US women. *Am J Clin Nutr* 2003;77(1):257–65.
608. Albala C, Ebbeling CB, Cifuentes M, Lera L, Bustos N, Ludwig DS. Effects of replacing the habitual consumption of sugar-sweetened beverages with milk in Chilean children. *Am J Clin Nutr* 2008;88(3):605–11.
609. Einarson A, Tam C, Erebara A, Koren G. Food-borne illnesses during pregnancy. *Can Fam Physician* 2010;56(9):869–70.
610. Brand JC, Darnton-Hill I, Gracey MS, Spargo RM. Lactose malabsorption in Australian Aboriginal children. *Am J Clin Nutr* 1985;41(3):620–2.
611. Buttenshaw R, Sheridan J, Tye V, Miller O, Carseldine JBD. Lactose malabsorption and its temporal stability in Aboriginal children. *Proc Nutr Soc Aust* 1990;15:228.
612. Shaikat A, Levitt MD, Taylor BC, MacDonald R, Shamliyan TA, Kane RL et al. Systematic review: effective management strategies for lactose intolerance. *Ann Intern Med* 2010;152(12):797–803.
613. Kleiner SM. Water: an essential but overlooked nutrient. *J Amer Diet Assoc* 1999;99(2):200–6.
614. Pfeil LA, Katz PR, Davis PJ. Water metabolism. In: Morley JE, Glick Z, Rubenstein LZ, eds. *Geriatric nutrition: a comprehensive view*. 2nd edition ed. New York: Raven Press, 1995.
615. Cheung SS, McLellan TM. Influence of hydration status and fluid replacement on heat tolerance while wearing NBC protective clothing. *Eur J Appl Physiol Occupat Physiol* 1998;77(1):139–48.
616. Hubbard RW, Armstrong LE. The heat illnesses: biochemical, ultrastructural, and fluid-electrolyte considerations. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human performance physiology and environmental medicine at terrestrial extremes*. Indianapolis: Benchmark Press, Inc, 1988: 305–60.
617. Gardner E, Ruxton C, Leeds A. Black tea—helpful or harmful? A review of the evidence. *Eur J Clin Nutr* 2006;61(1):3–18.
618. Greenberg JA, Chow G, Ziegelstein RC. Caffeinated coffee consumption, cardiovascular disease, and heart valve disease in the elderly (from the Framingham Study). *Am J Cardiol* 2008;102(11):1502–8.
619. Kuriyama S. The relation between green tea consumption and cardiovascular disease as evidenced by epidemiological studies. *J Nutr* 2008;138(8):1548S.
620. Lagiou P, Samoli E, Lagiou A, Tzonou A, Kalandidi A, Peterson J et al. Intake of specific flavonoid classes and coronary heart disease—a case-control study in Greece. *Eur J Clin Nutr* 2004;58(12):1643–8.
621. Sesso HD, Paffenbarger RS, Oguma Y, Lee I. Lack of association between tea and cardiovascular disease in college alumni. *Int J Epidemiol* 2003;32(4):527.
622. Fraser ML, Mok GS, Lee AH. Green tea and stroke prevention: emerging evidence. *Complement Ther Med* 2007;15(1):46–53.
623. 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):367–71.
624. Tanabe N, Suzuki H, Aizawa Y, Seki N. 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):1030–40.
625. Arab L, Liu W, Elashoff D. Green and black tea consumption and risk of stroke: a meta-analysis. *Stroke* 2009;40(5):1786–92.
626. Hammar N, Andersson T, Alfredsson L, Reuterwall C, Nilsson T, Hallqvist J 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):653–9.
627. Klatsky AL, Koplik S, Kipp H, Friedman GD. The confounded relation of coffee drinking to coronary artery disease. *Am J Cardiol* 2008;101(6):825–7.

628. Rosner SA, Åkesson A, Stampfer MJ, Wolk A. Coffee consumption and risk of myocardial infarction among older Swedish women. *Am J Epidemiol* 2007;165(3):288.
629. Sofi F, Conti AA, Gori AM, Elia L, Casini A, Abbate R et al. Coffee consumption and risk of coronary heart disease: a meta-analysis. *Nutr Metab Cardiovasc Dis* 2007;17(3):209–23.
630. Greenberg JA, Axen KV, Schnoll R, Boozer CN. Coffee, tea and diabetes: the role of weight loss and caffeine. *Int J Obes (Lond)* 2005;29(9):1121–9.
631. Hamer M, Witte DR, Mosler A, Marmot MG, Brunner EJ. 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):1046–53.
632. Hu G, Jousilahti P, Peltonen M, Bidel S, Tuomilehto J. 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):1742–9.
633. Odegaard AO, Pereira MA, Koh WP, Arakawa K, Lee HP, Yu MC. Coffee, tea, and incident type 2 diabetes: the Singapore Chinese Health Study. *Am J Clin Nutr* 2008;88(4):979–85.
634. Paynter NP, Yeh HC, Voutilainen S, Schmidt MI, Heiss G, Folsom AR 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):1075–84.
635. Smith B, Wingard DL, Smith TC, Kritz-Silverstein D, Barrett-Connor E. Does coffee consumption reduce the risk of type 2 diabetes in individuals with impaired glucose? *Diabetes Care* 2006;29(11):2385.
636. van Dam RM, Hu FB. Coffee consumption and risk of type 2 diabetes: a systematic review. *J Am Med Assoc* 2005;294(1):97–104.
637. van Dam RM, Willett WC, Manson JE, Hu FB. 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):398–403.
638. Tang N, Zhou B, Wang B, Yu R. Coffee consumption and risk of breast cancer: a metaanalysis. *Am J Obstet Gynecol* 2009;200(3):290 e1–9.
639. Michels KB, Willett WC, Fuchs CS, Giovannucci E. Coffee, tea, and caffeine consumption and incidence of colon and rectal cancer. *J Natl Cancer Inst* 2005;97(4):282–92.
640. Lee KJ, Inoue M, Otani T, Iwasaki M, Sasazuki S, Tsugane S. Coffee consumption and risk of colorectal cancer in a population-based prospective cohort of Japanese men and women. *Int J Cancer* 2007;121(6):1312–8.
641. Tavani A, La Vecchia C. Coffee, decaffeinated coffee, tea and cancer of the colon and rectum: a review of epidemiological studies, 1990–2003. *Cancer Causes Control* 2004;15(8):743–57.
642. Steevens J, Schouten LJ, Verhage BA, Goldbohm RA, van den Brandt PA. Tea and coffee drinking and ovarian cancer risk: results from the Netherlands Cohort Study and a meta-analysis. *Br J Cancer* 2007;97(9):1291–4.
643. Song YJ, Kristal AR, Wicklund KG, Cushing-Haugen KL, Rossing MA. Coffee, tea, colas, and risk of epithelial ovarian cancer. *Cancer Epidemiol Biomarkers Prev* 2008;17(3):712–6.
644. Silvera SA, Jain M, Howe GR, Miller AB, Rohan TE. Intake of coffee and tea and risk of ovarian cancer: a prospective cohort study. *Nutr Cancer* 2007;58(1):22–7.
645. Bravi F, Scotti L, Bosetti C, Gallus S, Negri E, La Vecchia C et al. Coffee drinking and endometrial cancer risk: a metaanalysis of observational studies. *Am J Obstet Gynecol* 2009;200(2):130–35.
646. Larsson SC, Giovannucci E, Wolk A. Coffee consumption and stomach cancer risk in a cohort of Swedish women. *Int J Cancer* 2006;119(9):2186–9.
647. Bravi F, Bosetti C, Tavani A, Bagnardi V, Gallus S, Negri E et al. Coffee drinking and hepatocellular carcinoma risk: a meta-analysis. *Hepatology* 2007;46(2):430–5.

648. Su LJ, Arab L. Tea consumption and the reduced risk of colon cancer -- results from a national prospective cohort study. *Public Health Nutr* 2002;5(3):419-25.
649. Dora I, Arab L, Martinchik A, Solvzhkov A, Urbanovich L, Weisgerber U. Black tea consumption and risk of rectal cancer in Moscow population. *Ann Epidemiol* 2003;13(6):405-11.
650. Kristensen M, Jensen M, Kudsk J, Henriksen M, Molgaard C. 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):1803-8.
651. Libuda L, Alexy U, Remert T, Stehle P, Schoenau E, Kersting M. 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):1670.
652. Manias K, McCabe D, Bishop N. Fractures and recurrent fractures in children; varying effects of environmental factors as well as bone size and mass. *Bone* 2006;39(3):652-7.
653. Tucker KL, Morita K, Qiao N, Hannan MT, Cupples LA, Kiel DP. 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):936.
654. Ahokas JT, Demos L, Donohue DC, Killalea S, McNeil J, Rix CJ. Review of water fluoridation and fluoride intake from discretionary fluoride supplements. Melbourne: National Health and Medical Research Council, 1999.
655. Young WG. Tooth wear: diet analysis and advice. *Int Dent J* 2005;55(2):62-72.
656. Kazoullis S, Seow WK, Holcombe T, Newman B, Ford D. Common dental conditions associated with dental erosion in schoolchildren in Australia. *Pediatr Dent* 2007;29(1):33-9.
657. American Academy of Pediatrics. Soft drinks in schools. *Pediatrics* 2004;113(1 Pt 1):152-4.
658. Keijzers GB, De Galan BE, Tack CJ, Smits P. Caffeine can decrease insulin sensitivity in humans. *Diabetes Care* 2002;25(2):364.
659. Yoshioka K, Kogure A, Yoshida T, Yoshikawa T. Coffee consumption and risk of type 2 diabetes mellitus. *Lancet* 2003;361(9358):703.
660. Lopez-Ridaura R, Willett WC, Rimm EB, Liu S, Stampfer MJ, Manson JAE et al. Magnesium intake and risk of type 2 diabetes in men and women. *Diabetes Care* 2004;27(1):634.
661. Curin Y, Andriantsitohaina R. Polyphenols as potential therapeutical agents against cardiovascular diseases. *Pharmacological reports* 2005;57:97.
662. National Health and Medical Research Council. Australian Drinking Water Guidelines 6. Canberra 2011.
663. Commonwealth Department of Health and Ageing. Healthy eating at various life stages: Pregnant Women. 2009 (cited). www.health.gov.au/internet/healthyactive/publishing.nsf/content/pregnant-women.
<http://www.patonsyarns.com/pattern.ph>
664. Mensink RP, Zock PL, Kester AD, Katan MB. 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):1146-55.
665. Ratnayake WM, Galli C. Fat and fatty acid terminology, methods of analysis and fat digestion and metabolism: a background review paper. *Ann Nutr Metab* 2009;55(1-3):8-43.
666. Truswell AS. Review of dietary intervention studies: effect on coronary events and on total mortality. *Aust NZ J Med* 1994;24:98-106.
667. Hooper L, Summerbell CD, Thompson R, Sills D, Roberts FG, Moore H et al. Reduced or modified dietary fats for preventing cardiovascular disease. *The Cochrane Library* 2011; July 2011.
668. Skeaff M, Miller J. Dietary fat and coronary heart disease: Summary of evidence from prospective cohort and randomised controlled trials. *Annals of nutrition and metabolism* 2009;55(1-3):173-U287.

669. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. *The American Journal of Clinical Nutrition* 2010;91(3):535-46.
670. Danaei G et al. The preventable causes of death in the United States: Comparative risk assessment of dietary lifestyle and metabolic risk factors. *PLoS Medicine* 2009;6(4):e1000058.
671. Food Standards Australia New Zealand. Review Report: Trans fatty acids in the New Zealand and Australian food supply. In: Food Standards Australia New Zealand, editor. Canberra 2009.
672. Melanson EL, Astrup A, Donahoo WT. The relationship between dietary fat and fatty acid intake and body weight, diabetes, and the metabolic syndrome. *Ann Nutr Metab* 2009;55(1-3):229-43.
673. Balk EM, Lichtenstein AH, Chung M, Kupelnick B, Chew P, Lau J. Effects of omega-3 fatty acids on coronary restenosis, intima-media thickness, and exercise tolerance: A systematic review. *Atherosclerosis* 2006;184(2):237-46.
674. Balk EM, Lichtenstein AH, Chung M, Kupelnick B, Chew P, Lau J. Effects of omega-3 fatty acids on serum markers of cardiovascular disease risk: a systematic review. *Atherosclerosis* 2006;189(1):19-30.
675. Castro IA, Barroso LP, Sinnecker P. Functional foods for coronary heart disease risk reduction: a meta-analysis using a multivariate approach. *Am J Clin Nutr* 2005;82(1):32-40.
676. Eslick GD, Howe PR, Smith C, Priest R, Bensoussan A. Benefits of fish oil supplementation in hyperlipidemia: a systematic review and meta-analysis. *Int J Cardiol* 2009;136(1):4-16.
677. Hooper L, Harrison RA, Summerbell DC, Moore H, Worthington HV, Ness A et al. Omega 3 fatty acids for prevention and treatment of cardiovascular disease. *Cochrane Database Syst Rev* 2004(4):CD003177.
678. Lee KW, Lip GYH. Effects of lifestyle on hemostasis, fibrinolysis, and platelet reactivity: a systematic review. *Arch Intern Med* 2003;163(19):2368-92.
679. Studer M, Briel M, Leimenstoll B, Glass TR, Bucher HC. Effect of different antilipidemic agents and diets on mortality: a systematic review. *Arch Intern Med* 2005;165(7):725.
680. Wang C, Harris WS, Chung M, Lichtenstein AH, Balk EM, Kupelnick B 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):5-17.
681. Wendland E, Farmer A, Glasziou P, Neil A. Effect of alpha linolenic acid on cardiovascular risk markers: a systematic review. *Heart* 2006;92(2):166-69.
682. Nations FAO/WHO. Fats and fatty acids in human nutrition. Report of an expert consultation. Rome: Food and Agriculture Organization of the United Nations 2010. Available from: http://www.fao.org/ag/agn/nutrition/Call_for_experts.pdf
683. Ulbak J, Lauritzen L, Hansen HS, Michaelsen KF. Diet and blood pressure in 2.5-year-old Danish children. *Am J Clin Nutr* 2004;79(6):1095-102.
684. Schack-Nielsen L, MÃ¸gaard C, Larsen D, Martyn C, Michaelsen KF. Arterial stiffness in 10-year-old children: current and early determinants. *Br J Nutr* 2005;94(6):1004-11.
685. Sanders TAB, Gleason K, Griffin B, Miller GJ. Influence of an algal triacylglycerol containing docosahexaenoic acid (22:6n-3) and docosahexaenoic acid (22:5n-6) on cardiovascular risk factors in healthy men and women. *Br J Nutr* 2006;95(3):525-31.
686. Raitakari OT, RÃnnmaa T, JÃrvisalo MJ, Kaitosaari T, Volanen I, Kallio K 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):3786-94.
687. Psaltopoulou T, Naska A, Orfanos P, Trichopoulos D, Mountokalakis T, Trichopoulou A. 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):1012-18.

688. Perona JS, Canizares J, Montero E, Sanchez-Dominguez JM, Ruiz-Gutierrez V, Perona JS 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):219–26.
689. Bonadia-Pons I, Schroder H, Covas MI, Castellote AI, Kaikkonen J, Poulsen HE et al. Moderate consumption of olive oil by healthy European men reduces systolic blood pressure in non-Mediterranean participants. *J Nutr* 2007;137(1):24–7.
690. Alonso A, Martinez-Gonzalez MA, Alonso A, Martinez-Gonzalez MA. Olive oil consumption and reduced incidence of hypertension: the SUN study. *Lipids* 2004;39(12):1233–8.
691. Damsgaard CT, Frøkiær H, Andersen AD, Lauritzen L. 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):1061–66.
692. Berry SE, Miller GJ, Sanders TA, Berry SEE, Miller GJ, Sanders TAB. The solid fat content of stearic acid-rich fats determines their postprandial effects. *Am J Clin Nutr* 2007;85(6):1426–94.
693. Eckel RH, Hernandez TL, Bell ML, Weil KM, Shepard TY, Grunwald GK et al. Carbohydrate balance predicts weight and fat gain in adults. *Am J Clin Nutr* 2006;83(4):803–08.
694. Kratz M, von Eckardstein A, Fobker M, Buyken A, Posny N, Schulte H et al. The impact of dietary fat composition on serum leptin concentrations in healthy nonobese men and women. *J Clin Endocrinol Metabol* 2002;87(11):5002–14.
695. Lovegrove JA, Lovegrove SS, Lesauvage SVM, Brady LM, Saini N, Minihane AM 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):974–82.
696. Nicholls SJ, Lundman P, Harmer JA, Cutri B, Griffiths KA, Rye KA 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):715–20.
697. Nosaka N, Kasai M, Nakamura M, Takahashi I, Itakura M, Takeuchi H et al. Effects of dietary medium-chain triacylglycerols on serum lipoproteins and biochemical parameters in healthy men. *Biosci Biotech Biochem* 2002;66(8):1713–8.
698. Owen B, Wolever TMS. Effect of fat on glycaemic responses in normal subjects: a dose-response study. *Nutr Res* 2003;23(10):1341–47.
699. Rueda-Clausen CF, Silva FA, Lindarte MA, Villa-Roel C, Gomez E, Gutierrez R 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):50–57.
700. Tahvonen RL, Schwab US, Linderborg KM, Mykkänen HM, Kallio HP. 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. *J Nutr Biochem* 2005;16(6):353–59.
701. Tholstrup T, Raff M, Basu S, Nonboe P, Sejrsen K, Straarup EM. 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):237–43.
702. MacLean CH, Newberry SJ, Mojica WA, Khanna P, Issa AM, Suttorp MJ et al. Effects of omega-3 fatty acids on cancer risk: a systematic review. *J Am Med Assoc* 2006;295(4):403–15.
703. van de Rest O, Geleijnse JM, Kok FJ, van Staveren WA, Hoefnagels WH, Beekman ATF 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):706–13.
704. Laitinen M, Ngandu T, Rovio S, Helkala E, Uusitalo U, Viitonen M et al. Fat intake at midlife and risk of dementia and Alzheimer's disease: a population-based study. *Dementia Geriatric Cognitive Dis* 2006;22(1):99–107.

705. Mamalakis G, Kiriakakis M, Tsibinos G, Hatzis C, Flouri S, Mantzoros C et al. Depression and serum adiponectin and adipose omega-3 and omega-6 fatty acids in adolescents. *Pharmacol Biochem Behav* 2006;85(2):474–79.
706. Morris MC, Evans DA, Bienias JL, Tangney CC, Bennett DA, Wilson RS et al. Consumption of fish and n-3 fatty acids and risk of incident Alzheimer disease. *Arch Neurol* 2003;60(7):940–46.
707. Hiroi M, Nagahara Y, Miyauchi R, Misaki Y, Goda T, Kasezawa N et al. The combination of genetic variations in the PRDX3 gene and dietary fat intake contribute to obesity risk. *Obesity* (Silver Spring) 2010.
708. Fantuzzi G. Adipose tissue, adipokines, and inflammation. *J Allergy Clin Immunol* 2005;115(5):911–19.
709. Jain SS, Bird RP. Elevated expression of tumor necrosis factor- α signaling molecules in colonic tumors of Zucker obese (fa/fa) rats. *Int J Cancer* 2010;127(9):2042–50.
710. Renehan AG, Soerjomataram I, Tyson M, Egger M, Zwahlen M, Coebergh JW et al. Incident cancer burden attributable to excess body mass index in 30 European countries. *Int J Cancer* 2010;126(3):692–702.
711. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA 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):1640–5.
712. Baghurst K. Dietary fats, marbling and human health. *Australian Journal of Experimental Agriculture* 2004;44:635–44.
713. National Heart Foundation of Australia (National Blood Pressure and Vascular Disease Advisory Committee). Guide to management of hypertension. National Heart Foundation of Australia; 2008.
714. Sacks FM, Pfeffer MA, Moye LA, Rouleau JL, Rutherford JD, Cole TG 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):1001–9.
715. Ramsden CE, Hibbeln JR, Majchrzak SF, Davis JM. 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):1586–600.
716. Brown MA, Storlien LH, Huang X-F, Tapsell LC, Else PL, Higgins JA et al. Dietary fat and carbohydrate composition: Metabolic disease. In: Montmayeur J-P, leCoutre J, eds. *Fat detection: taste, texture and post ingestive effects*. Florida: CRC Press 2010.
717. Galli C, Calder PC. Effects of fat and fatty acid intake on inflammatory and immune responses: a critical review. *Ann Nutr Metab* 2009;55(1-3):123–39.
718. Crawford MA, Bazinet RP, Sinclair AJ. Fat intake and CNS functioning: ageing and disease. *Ann Nutr Metab* 2009;55(1-3):202–28.
719. Daniels SR, Greer FR. Lipid screening and cardiovascular health in childhood. *Pediatrics* 2008;122(1):198–208.
720. McGill HC, McMahan CA, Gidding SS. Are pediatricians responsible for prevention of adult cardiovascular disease? *Nat Clin Pract Cardiovasc Med* 2009;6(1):10–1.
721. Niinikoski H, Laegstrom H, Jokinen E, Siltala M, Ronnema T, Viikari J 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):1032.
722. Clarke R, Emberson JR, Parish S, Palmer A, Shipley M, Linksted P et al. Cholesterol fractions and apolipoproteins as risk factors for heart disease mortality in older men. *Arch Intern Med* 2007;167(13):1373–8.
723. U.S. Department of Health, Education, Welfare. Healthy People - The Surgeon General's report on health promotion and disease prevention Washington D.C.: U.S. Department of Health, Education and Welfare, 1979. <http://profiles.nlm.nih.gov/ps/access/NNBBGK.pdf>

724. Jürgens G, Graudal Niels A. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterols, and triglyceride. *Cochrane Database Syst Rev* 2004(1).
725. Hooper L, Bartlett C, Davey SG, Ebrahim S, Davey SG. 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):CD003656.
726. He FJ, MacGregor GA. Effect of longer-term modest salt reduction on blood pressure. *Cochrane Database Syst Rev* 2004(3):CD004937.
727. Geleijnse JM, Kok FJ, Grobbee DE. Impact of dietary and lifestyle factors on the prevalence of hypertension in Western populations. *Eur J Public Health* 2004;14(3):235-39.
728. Chen J, Gu D, Huang J, Rao DC, Jaquish CE, Hixson JE 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):829-35.
729. Charlton KE, Steyn K, Levitt NS, Peer N, Jonathan D, Gogela T 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):1397-406.
730. 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):2011-8.
731. Takahashi Y, Sasaki S, Okubo S, Hayashi M, Tsugane S. Blood pressure change in a free-living population-based dietary modification study in Japan. *J Hypertens* 2006;24(3):451-58.
732. Robertson JL, Robertson JLS. Dietary salt and hypertension: a scientific issue or a matter of faith? *J Eval Clin Pract* 2003;9(1):1-22.
733. Obarzanek E, Proschan MA, Vollmer WM, Moore TJ, Sacks FM, Appel LJ et al. Individual blood pressure responses to changes in salt intake: results from the DASH-Sodium trial.[see comment]. *Hypertension* 2003;42(4):459-67.
734. Nowson CA, Wattanapenpaiboon N, Pachett A, Nowson CA, Wattanapenpaiboon N, Pachett A. Low-sodium dietary approaches to stop hypertension-type diet including lean red meat lowers blood pressure in postmenopausal women. *Nutr Res* 2009;29(1):8-18.
735. Melander O, von Wöhrn F, Frandsen E, Burri P, Willsteén G, Aurell M 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):619-27.
736. Kawano Y, Ando K, Matsuura H, Tsuchihashi T, Fujita T, Ueshima H 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. *Hypertens Res Clin Exp* 2007;30(10):879-86.
737. Geleijnse JM, Kok FJ, Grobbee DE. Blood pressure response to changes in sodium and potassium intake: a meta-regression analysis of randomised trials. *J Hum Hypertens* 2003;17(7):471-80.
738. Dickinson KM, Keogh JB, Clifton PM. Effects of a low-salt diet on flow-mediated dilatation in humans. *Am J Clin Nutr* 2009;89(2):485-90.
739. Dickinson BD, Havas S. 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):1460-68.
740. Geleijnse JM, Grobbee DE. High salt intake early in life: does it increase the risk of hypertension? *J Hypertens* 2002;20(11):2121-24.
741. Beevers DG. The epidemiology of salt and hypertension. *Clinical Autonomic Research* 2002;12(5):353-7.
742. Chang HY, Hu YW, Yue CS, Wen YW, Yeh WT, Hsu LS et al. Effect of potassium-enriched salt on cardiovascular mortality and medical expenses of elderly men. *Am J Clin Nutr* 2006;83(6):1289-96.

743. Cook NR, Cutler JA, Obarzanek E, Buring JE, Rexrode KM, Kumanyika SK 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):885-88.
744. Cook NR, Obarzanek E, Cutler JA, Buring JE, Rexrode KM, Kumanyika SK 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):32-40.
745. He J, Ogden LG, Bazzano LA, Vupputuri S, Loria C, Whelton PK. 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):1619-24.
746. Reedy KS, Katan MB. 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):167-86.
747. Lawes CM, Vander Hoorn S, Rodgers A. Global burden of blood-pressure-related disease, 2001. *Lancet* 2008;371(9623):1513-8.
748. Law MR, Morris JK, Wald NJ. 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:b1665.
749. Key TJ, Schatzkin A, Willett WC, Allen NE, Spencer EA, Travis RC. Diet, nutrition and the prevention of cancer. *Public Health Nutr* 2004;7(1A):187-200.
750. Commonwealth Department of Health. Dietary guidelines for Australians. Canberra: Commonwealth Department of Health, 1992.
751. National Health and Medical Research Council. Dietary guidelines for Australians. Canberra: Commonwealth of Australia, 1992. http://www.nhmrc.gov.au/_files_nhmrc/file/publications/synopses/withdrawn/n4.pdf
752. Food Standards Australia New Zealand. Food Standards Code- Standard 1.2.8- Clause 17(1). 2012.
753. Food Standards Australia and New Zealand. Final assessment report - Proposal P230 – Consideration of mandatory fortification with iodine for New Zealand. Wellington: Food Standards Australia New Zealand, 2008 12 February 2008. <http://www.foodstandards.gov.au/foodstandards/proposals/proposalp230iodinefo2002.cfm>
754. Duley L, Henderson-Smith D. Reduced salt intake compared to normal dietary salt, or high intake, in pregnancy. *Cochrane Database Syst Rev* 1999(2):CD001687.
755. Food Standards Australia New Zealand. Food Standards Code – Standard 2.9.2 Foods for Infants. 2009.
756. Krebs-Smith SM. Choose beverages and foods to moderate your intake of sugars: measurement requires quantification. *J Nutr* 2001;131(2S-1):527S-35S.
757. Marriott BP, Olsho L, Hadden L, Connor P. 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):228-58.
758. Australian Institute of Health and Welfare. Health expenditure Australia 2007-08, cat. no. HWE 46. Canberra: Australian Institute of Health and Welfare, 2009.
759. Mann JI, Truswell AS, Manning EB. Effects on serum lipids of reducing dietary sucrose or starch for 22 weeks in normal men. *S Afr Med J* 1972;46(25):827-34.
760. Palmer JR, Boggs DA, Krishnan S, Hu FB, Singer M, Rosenberg L. Sugar-sweetened beverages and incidence of type 2 diabetes mellitus in African American women. *Arch Intern Med* 2008;168(14):1487-92.
761. Laville M, Nazare JA. Diabetes, insulin resistance and sugars. *Obes Rev* 2009;10(Suppl 1):24-33.
762. Esposito K, Kastorini CM, Panagiotakos DB, Giugliano D. Prevention of type 2 diabetes by dietary patterns: a systematic review of prospective studies and meta-analysis. *Metab Syndr Relat Disord* 2010;8(6):471-6.

763. Malik VS, Popkin BM, Bray GA, Despres JP, Willett WC, Hu FB. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. *Diabetes Care* 2010;33(11):2477–83.
764. Fiorito LM, Marini M, Mitchell DC, Smiciklas-Wright H, Birch LL. 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):543–50.
765. Libuella L, Alexy U, Sichert-Hellert W, Stehle P, Karaolis-Danckert N, Buyken AE 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):1370–9.
766. Bes-Rastrollo M, Sanchez-Villegas A, Gomez-Gracia E, Martinez JA, Pajares RM, Martinez-Gonzalez MA. Predictors of weight gain in a Mediterranean cohort: the Seguimiento Universidad de Navarra Study 1. *Am J Clin Nutr* 2006;83(2):362–70; quiz 94–5.
767. Silvera SA, Jain M, Howe GR, Miller AB, Rohan TE, Silvera SAN 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):653–8.
768. Michaud DS, Liu S, Giovannucci E, Willett WC, Colditz GA, Fuchs CS et al. Dietary sugar, glycemic load, and pancreatic cancer risk in a prospective study. *J Natl Cancer Inst* 2002;94(17):1293–300.
769. Michaud DS, Fuchs CS, Liu S, Willett WC, Colditz GA, Giovannucci E et al. Dietary glycemic load, carbohydrate, sugar, and colorectal cancer risk in men and women. *Cancer Epidemiol Biomarkers Prev* 2005;14(1):138–47.
770. De Stefani E, Boffetta P, Ronco AL, Deneo-Pellegrini H, Acosta G, Mendilaharsu M. Dietary patterns and risk of bladder cancer: a factor analysis in Uruguay. *Cancer Causes Control* 2008;19(10):1243–9.
771. Nothlings U, Murphy SP, Wilkens LR, Henderson BE, Kolonel LN. Dietary glycemic load, added sugars, and carbohydrates as risk factors for pancreatic cancer: the Multiethnic Cohort Study. *Am J Clin Nutr* 2007;86(5):1495–501.
772. Miller AVD. Dental caries. *Am J Dent Sci* 1883;17:77–130.
773. Burt BA, Eklund SA, Morgan KJ, Larkin FE, Guire KE, Brown LO 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):1422–9.
774. Warren JJ, Weber Gasparoni K, Marshall TA, Drake DR, Dehkordi Vakili F, Dawson DV et al. A longitudinal study of dental caries risk among very young low SES children. *Community Dent Oral Epidemiol* 2009;37(2):116–22.
775. Ruottinen S, Karjalainen S, Pienihäkkinen K, Lagström H, Niinikoski H, Salminen M et al. Sucrose intake since infancy and dental health in 10-year-old children. *Caries research* 2000;33(2):142–48.
776. Marshall TA, Eichenberger-Gilmore JM, Larson MA, Warren JJ, Levy SM. Comparison of the intakes of sugars by young children with and without dental caries experience. *J Am Dent Assoc* 2007;138(1):39.
777. Levy SM, Warren JJ, Broffitt B, Hillis SL, Kanellis MJ. Fluoride, beverages and dental caries in the primary dentition. *Caries Res* 2003;37(3):157–65.
778. Anderson CA, Curzon ME, Van Loveren C, Tatsi C, Duggal MS. Sucrose and dental caries: a review of the evidence. *Obes Rev* 2009;10(Suppl 1):41–54.
779. Gibson S, Williams S. 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):101.
780. American Academy of Pediatrics. Clinical practice guideline: Diagnosis and evaluation of the child with attention-deficit/hyperactivity disorder. *Pediatrics* 2000;105(5).
781. Walsh LJ. Preventive dentistry for the general dental practitioner. *Aust Dent J* 2000;45(2):76–82.

782. Balakrishnan M, Simmonds RS, Tagg JR. Dental caries is a preventable infectious disease. *Aust Dent J* 2000;45(4):235-45.
783. Mattes RD. Dietary compensation by humans for supplemental energy provided as ethanol or carbohydrate in fluids. *Physiol Behav* 1996;59(1):179-87.
784. Raben A, Vasilaras TH, Moller AC, Astrup A. 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):721-9.
785. Lam M, Riedy CA, Coldwell SE, Milgrom P, Craig R. Children's acceptance of xylitol-based foods. *Community Dent Oral Epidemiol* 2000;28(2):97-101.
786. Davies MJ, Spencer AJ, Westwater A, Simmons B. Dental caries among Australian Aboriginal, non-Aboriginal Australian-born, and overseas-born children. *Bull World Health Organ* 1997;75(3):497.
787. Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA. Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis. *BMJ* 2011;342:d671.
788. Chikritzhs T, Fillmore K, Stockwell T. A healthy dose of scepticism: Four good reasons to think again about protective effects of alcohol on coronary heart disease. *Drug Alcohol Rev* 2009;28(4):441-44.
789. Stockwell T, Greer A, Fillmore K, Chikritzhs T, C. Z. Health benefits of moderate alcohol consumption: How good is the science? *BMJ* 2012;344(e2276).
790. Calabria B, Doran CM, Vos T, Shakeshaft AP, Hall W. Epidemiology of alcohol related burden of disease among Indigenous Australians. *Aust N Z J Public Health* 2010;34:S47-S51.
791. Chikritzhs TN, Jonas HA, Stockwell TR, Heale PF, Dietze PM. Mortality and life-years lost due to alcohol: a comparison of acute and chronic causes. *Med J Aust* 2001;174(6):281-84.
792. Collins DJ, Lapsley HM. The costs of tobacco, alcohol and illicit drug abuse to Australian society in 2004/05. Canberra: Commonwealth Department of Health and Ageing, 2008. <http://www.health.gov.au/internet/drugstrategy/publishing.nsf/Content/mono64>
793. Xiao J, Rowe T, Somerford P, Draper G, Martin J. Impact of alcohol on the population of Western Australia. In: *Epidemiology Branch DoHW, editor*. 2008.
794. National Health and Medical Research Council. Australian guidelines to reduce health risks from drinking alcohol. Canberra: Commonwealth of Australia; 2009.
795. Banwell C. How many standard drinks are there in a glass of wine? *Drug Alcohol Rev* 1999;18(1):99-101.
796. Arriola L, Martinez-Camblor P, Larranaga N, Basterretxea M, Amiano P, Moreno-Iribas C et al. Alcohol intake and the risk of coronary heart disease in the Spanish EPIC cohort study. *Heart* 2010;96(2):124-30.
797. Simons LA, McCallum J, Friedlander Y, Simons J. Alcohol intake and survival in the elderly: a 77 month follow-up in the Dubbo study. *Aust N Z J Med* 1996;26(5):662-70.
798. Cullen KJ, Knuiman MW, Ward NJ. Alcohol and mortality in Busselton, Western Australia. *Am J Epidemiol* 1993;137(2):242-8.
799. Costanzo S, Di Castelnuovo A, Donati MB, Iacoviello L, de Gaetano G. Alcohol consumption and mortality in patients with cardiovascular disease: a meta-analysis. *J Am Coll Cardiol* 2010;55(13):1339-47.
800. Roerecke M, Rehm J. Ischemic heart disease mortality and morbidity rates in former drinkers: a meta-analysis. *Am J Epidemiol* 2011;173(3):245-58.
801. Schooling CM, Wenjie S, Ho SY, Chan WM, Tham MK, Ho KS et al. Moderate alcohol use and mortality from ischaemic heart disease: a prospective study in older Chinese people. *PLoS One* 2008;3(6).
802. Sierksma A, van der Gaag MS, Kluft C, Hendriks HF, Hendriks HFJ. Moderate alcohol consumption reduces plasma C-reactive protein and fibrinogen levels; a randomized, diet-controlled intervention study. *Eur J Clin Nutr* 2002;56(11):1130-6.

803. Badia E, Sacanella E, Fernandez-Sola J, Nicolas JM, Antunez E, Rotilio D 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):225–30.
804. Hansen AS, Marckmann P, Dragsted LO, Finne Nielsen IL, Nielsen SE, Gronbaek M. 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):449–55.
805. Tsang C, Higgins S, Duthie GG, Duthie SJ, Howie M, Mullen W 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):233–40.
806. Avellone G, Di Garbo V, Campisi D, De Simone R, Raneli G, Scaglione R et al. Effects of moderate Sicilian red wine consumption on inflammatory biomarkers of atherosclerosis. *Eur J Clin Nutr* 2006;60(1):41–7.
807. Adolorato G, Leggio L, Ojetti V, Capristo E, Gasbarrini G, Gasbarrini A. Effects of short-term moderate alcohol administration on oxidative stress and nutritional status in healthy males. *Appetite* 2008;50(1):50–56.
808. Beulens JWW, van den Berg R, Kok FJ, Helander A, Vermunt SHF, Hendriks HFJ. Moderate alcohol consumption and lipoprotein-associated phospholipase A2 activity. *Nutr Metab Cardiovasc Dis* 2008;18(8):539–44.
809. Tousoulis D, Ntarladimas I, Antoniadou C, Vasiliadou C, Tentolouris C, Papageorgiou N et al. Acute effects of different alcoholic beverages on vascular endothelium, inflammatory markers and thrombosis fibrinolysis system. *Clinical Nutrition* 2008;27(4):594–600.
810. Costanzo S, Di Castelnuovo A, Donati MB, Iacoviello L, de Gaetano G. Cardiovascular and overall mortality risk in relation to alcohol consumption in patients with cardiovascular disease. *Circulation* 2010;121(17):1951.
811. Bagnardi V, Zatonski W, Scotti L, La Vecchia C, Corrao G. Does drinking pattern modify the effect of alcohol on the risk of coronary heart disease? Evidence from a meta-analysis. *J Epidemiol Community Health* 2008;62(7):615.
812. Ma XJ, Jia WP, Hu C, Zhou J, Lu HJ, Zhang R 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):2541–3.
813. Taylor B, Irving HM, Baliunas D, Roerecke M, Patra J, Mohapatra S et al. Alcohol and hypertension: gender differences in dose-response relationships determined through systematic review and meta analysis. *Addiction* 2009;104(12):1981–90.
814. Savica V, Bellinghieri G, Kopple JD. The effect of nutrition on blood pressure. *Annu Rev Nutr* 2010;30:365–401.
815. Hackam DG, Khan NA, Hemmelgarn BR, Rabkin SW, Touyz RM, Campbell NR et al. The 2010 Canadian Hypertension Education Program recommendations for the management of hypertension: part 2 – therapy. *Can J Cardiol* 2010;26(5):249–58.
816. National Stroke Foundation. Clinical guidelines for acute stroke management. Melbourne: National Stroke Foundation, 2007. http://www.nhmrc.gov.au/_files_nhmrc/file/publications/synopses/cp109.pdf<http://www.strokefoundation.com.au/news/welcome/clinical-guidelines-for-acute-stroke-management>
817. Allen NE, Beral V, Casabonne D, Kan SW, Reeves GK, Brown A et al. Moderate alcohol intake and cancer incidence in women. *J Natl Cancer Inst* 2009;101(5):296–305.
818. Terry MB, Zhang FF, Kabat G, Britton JA, Teitelbaum SL, Neugut AI et al. Lifetime alcohol intake and breast cancer risk. *Ann Epidemiol* 2006;16(3):230–40.
819. Petri AL, Tjonneland A, Gamborg M, Johansen D, Hoidrup S, Sorensen TI et al. Alcohol intake, type of beverage, and risk of breast cancer in pre- and postmenopausal women. *Alcoholism Clin Exp Res* 2004;28(7):1084–90.

820. Newcomb PA, Nichols HB, Beasley JM, Egan K, Titus-Ernstoff L, Hampton JM et al. No difference between red wine or white wine consumption and breast cancer risk. *Cancer Epidemiol Biomarkers Prev* 2009;18(3):1007–10.
821. Li Y, Baer D, Friedman GD, Udal'tsova N, Shim V, Klatsky AL et al. Wine, liquor, beer and risk of breast cancer in a large population. *Eur J Cancer* 2009;45(5):843–50.
822. Deandrea S, Talamini R, Foschi R, Montella M, Dal Maso L, Falcini F 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):2025–8.
823. Bessaoud F, Daurès JP. 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):467–75.
824. Fuchs CS, Willett WC, Colditz GA, Hunter DJ, Stampfer MJ, Speizer FE 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):227.
825. Bongaerts BW, de Goeij AF, van den Brandt PA, Weijenberg MP. Alcohol and the risk of colon and rectal cancer with mutations in the K-ras gene. *Alcohol* 2006;38(3):147–54.
826. Purdue MP, Hashibe M, Berthiller J, La Vecchia C, Dal Maso L, Herrero R 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):132–42.
827. Liang W, Chikritzhs T, Pascal R, Binns CW. 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. *Int Med J* 2011;41(1a):34–41.
828. Rehm J, Taylor B, Mohapatra S, Irving H, Baliunas D, Patra J et al. Alcohol as a risk factor for liver cirrhosis: a systematic review and meta analysis. *Drug and Alcohol Review* 2010;29(4):437–45.
829. Stampfer MJ, Kang JH, Chen J, Cherry R, Grodstein F. Effects of moderate alcohol consumption on cognitive function in women. *N Engl J Med* 2005;352(3):245–53.
830. Solfrizzi V, D'Introno A, Colacicco AM, Capurso C, Del Parigi A, Balassarre G et al. Alcohol consumption, mild cognitive impairment, and progression to dementia. *Neurology* 2007;68(21):1790–9.
831. Ruitenberg A, van Swieten JC, Witteman JC, Mehta KM, van Duijn CM, Hofman A et al. Alcohol consumption and risk of dementia: the Rotterdam Study. *Lancet* 2002;359(9303):281–6.
832. Luchsinger JA, Tang MX, Siddiqui M, Shea S, Mayeux R. Alcohol intake and risk of dementia. *J Am Geriatr Soc* 2004;52(4):540–6.
833. Letenneur L, Larrieu S, Barberger-Gateau P. Alcohol and tobacco consumption as risk factors of dementia: a review of epidemiological studies. *Biomed Pharmacother* 2004;58(2):95–99.
834. Harper C. The neuropathology of alcohol-specific brain damage, or does alcohol damage the brain? *J Neuropathol Exp Neurol* 1998;57(2):101.
835. Deng J, Zhou DH, Li J, Wang YJ, Gao C, Chen M. A 2-year follow-up study of alcohol consumption and risk of dementia. *Clin Neurol Neurosurg* 2006;108(4):378–83.
836. Brown SA, Tapert SF. Adolescence and the trajectory of alcohol use: basic to clinical studies. *Annals NY Acad Sci* 2004;1021(1):234–44.
837. Carpiulo T. Understanding the health impact of alcohol dependence. *Am J Health Syst Pharm* 2007;64(5 Suppl 3):S5–11.
838. Chen CY, Storr CL. Alcohol use and health-related quality of life among youth in Taiwan. *J Adol Health* 2006;39(5):752. e9–52. e16.
839. Deas D, Brown ES. Adolescent substance abuse and psychiatric comorbidities. *J Clin Psychiatry* 2006;67(7):e02.

840. Turner RJ, Gil AG. Psychiatric and substance use disorders in South Florida: racial/ethnic and gender contrasts in a young adult cohort. *Arch Gen Psychiatry* 2002;59(1):43.
841. National Health and Medical Research Council. Australian alcohol guidelines: Health risks and benefits. Canberra: Commonwealth of Australia, 2001.
842. Truswell AS. Australian experience with the Wernicke-Korsakoff syndrome. *Addiction* 2000;95(6):829–32.
843. Rolland S, Truswell AS. Wernicke-Korsakoff syndrome in Sydney hospitals after 6 years of thiamin enrichment of bread. *Public Health Nutr* 1998;1(2):117–22.
844. Edenberg HJ. The genetics of alcohol metabolism: role of alcohol dehydrogenase and aldehyde dehydrogenase variants. *Alcohol Res Health* 2007;30(1):5–13.
845. Whitfield JB, Martin NG. Alcohol consumption and alcohol pharmacokinetics: interactions within the normal population. *Alcoholism: Clin Exp Res* 1994;18(2):238–43.
846. Li TK, Beard JD, Orr WE, Kwo PY, Ramchandani VA. Gender and ethnic differences in alcohol metabolism. *Alcoholism Clin Exp Res* 1998;22(3):771–72.
847. Bellis MD, Narasimhan A, Thatcher DL, Keshavan MS, Soloff P, Clark DB. 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):1590–600.
848. White AM, Swartzwelder HS. Hippocampal function during adolescence: a unique target of ethanol effects. *Annals NY Acad Sci* 2004;1021(1):206–20.
849. Brinton EA. Effects of ethanol intake on lipoproteins and atherosclerosis. *Curr Opin Lipidol* 2010;21(4):346–51.
850. American Diabetes Association. Nutrition recommendations and interventions for diabetes: a position statement of the American Diabetes Association. *Diabetes Care* 2007;30(Suppl 1):S48–65.
851. CMP Medica. MIMS Annual 2007. St Leonards, NSW: CMP Medica, 2007.
852. Lee Y, Back JH, Kim J, Kim SH, Na DL, Cheong HK et al. Systematic review of health behavioral risks and cognitive health in older adults. *Int Psychogeriatr* 2010;22(2):874–87.
853. Arntzen K, Schirmer H, Wilsaard T, Mathiesen E. 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;122:23–29.
854. Australian Institute of Health and Welfare. 2007 National Drug Strategy Household Survey: Detailed findings. Cat. no. PHE 107. Canberra: Australian Institute of Health and Welfare, 2008.
855. Australian Bureau of Statistics. Apparent consumption of alcohol, Australia, 2008–09. Cat. no. 43070.55.001. Canberra: Australian Bureau of Statistics, 2010.
856. National Preventative Health Taskforce. Australia: the healthiest country by 2020. A discussion paper. Canberra: Commonwealth of Australia, 2008.
857. Giglia R, Binns C. Alcohol and lactation: a systematic review. *Nutr Diet* 2006;63(2):103–16.
858. Giglia RC, Binns CW, Alfonso HS, Scott JA, Oddy WH. The effect of alcohol intake on breastfeeding duration in Australian women. *Acta Paediatr* 2008;97(5):624–9.
859. Aira M, Hartikainen S, Sulkava R. 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):680–85.
860. Fletcher PC, Hirdes JP. Risk factors for accidental injuries within senior citizens' homes: analysis of the Canadian survey on ageing and independence. *J Gerontol Nurs* 2005;31(2):49.
861. Mukamal K, Robbins J, Cauley J, Kern L, Siscovick D. Alcohol consumption, bone density, and hip fracture among older adults: the cardiovascular health study. *Osteoporosis int* 2007;18(5):593–602.

862. Australian Institute of Health and Welfare. The problem of osteoporotic hip fracture in Australia. Canberra: Australian Institute of Health and Welfare, 2010. <http://www.aihw.gov.au/publication-detail/?id=6442463333&tab=2>
863. Mutch R, Peardon E, Elliott E, C. B. Need to establish a national diagnostic capacity for foetal alcohol spectrum disorders. *Journal of Paediatrics and Child Health* 2009;45(79-81).
864. Kirby A. Blunting the legacy of alcohol abuse in Western Australia. *The Lancet* 2012;379:207-8.
865. Cheyne EH, Sherwin RS, Lunt MJ, Cavan DA, Thomas PW, Kerr D. Influence of alcohol on cognitive performance during mild hypoglycaemia; implications for Type 1 diabetes. *Diabet Med* 2004;21(3):230-7.
866. Tolman KG, Dalpiaz AS. Treatment of non-alcoholic fatty liver disease. *Ther Clin Risk Manag* 2007;3(6):1153-63.
867. Ferreira SE, de Mello MT, Pompeia S, de Souza-Formigoni ML. Effects of energy drink ingestion on alcohol intoxication. *Alcohol Clin Exp Res* 2006;30(4):598-605.
868. Price SR, Hilchey CA, Darredeau C, Fulton HG, Barrett SP. Energy drink co administration is associated with increased reported alcohol ingestion. *Drug and Alcohol Review* 2010;29(3):331-33.
869. Verster JC, Aufricht C, Alford C. Energy drinks mixed with alcohol: misconceptions, myths and facts. *International Journal of General Medicine* 2012;5:487-98.
870. de Haan L, de Haan HA, van der Palen J, Olivier B, Verster JC. Effects of consuming alcohol mixed with energy drinks versus consuming alcohol only on overall alcohol consumption and negative alcohol-related consequences. *International Journal of General Medicine* 2012;5:953-60.
871. World Health Organization. Exclusive breastfeeding statement. 2011. Available from: http://www.who.int/nutrition/topics/exclusive_breastfeeding/en/
872. World Health Organization. International code of marketing of breast-milk substitutes. Geneva: World Health Organization, 1981. http://www.who.int/nutrition/publications/code_english.pdf
873. Commonwealth Department of Health and Ageing. Australian National Breastfeeding Strategy 2010-2015. In: Commonwealth Department of Health and Ageing, editor. Canberra: Australian Health Ministers' Conference; 2009.
874. Ip S, Chung M, Raman G, Chew P, Magula N, DeVine D et al. Breastfeeding and maternal and infant health outcomes in developed countries. *Evid Rep Technol Assess (Full Rep)* 2007(153):1-186.
875. American Academy of Pediatrics. Pediatric Nutrition Handbook. Fifth ed. Kleinman RE, editor. Washington DC: American Academy of Pediatrics, 2004. 1178 p. http://books.google.com.au/books?id=LNt5AAAAMAAJ&q=Pediatric+Nutrition+Handbook.+Fifth.+2004.&dq=Pediatric+Nutrition+Handbook.+Fifth.+2004.&hl=en&ei=XcySTsSsDs_lcYHZwIkH&sa=X&oi=book_result&ct=result&resnum=2&ved=0CDMQ6AEwAQ
876. Prentice A. Constituents of human milk. *Food Nutr Bull* 1996;17:305-12.
877. Centre for Epidemiology and Research. 2005-2006 Report on child health from the New South Wales Population Healthy Survey. Sydney: NSW Department of Health, 2008. Available from: <http://www.health.nsw.gov.au/pubs/2008/pdf/childreport0506.pdf>
878. Akre J. Infant feeding. The physiological basis. *Bull World Health Organ* 1989(67 Suppl): 1-108.
879. Kramer M, Kakuma R. Optimal duration of exclusive breastfeeding. *Cochrane Database Syst Rev* 2002(1):CD003517.
880. Horta BL, Bahl R, Martines JC, Victora CG. Evidence on the long-term effects of breastfeeding. Geneva: World Health Organization, 2007.
881. Anderson J, Malley K, Snell R. 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 Rev* 2009;17(2):23.

882. Kramer MS, Matush L, Vanilovich I, Platt RW, Bogdanovich N, Sevkovskaya Z 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):1717.
883. Agostoni C, Decsi T, Fewtrell M, Goulet O, Kolacek S, Koletzko B et al. Complementary feeding: a commentary by the ESPGHAN Committee on Nutrition. *J Pediatr Gastroenterol Nutr* 2008;46(1):99.
884. World Health Organization. Equity, social determinant and public health programmes. Blas E, Kurup AS, editors. Geneva: World Health Organization, 2010.
885. Prescott SL, Smith PT, Tang M, Palmer DJ, Sinn J, Huntley SJ et al. The importance of early complementary feeding in the development of oral tolerance: concerns and controversies. *Pediatr Allergy Immunol* 2008;19(5):375–80.
886. Scott JA, Binns CW, Graham KI, Oddy WH. Predictors of the early introduction of solid foods in infants: results of a cohort study. *BMC Pediatr* 2009;9:60.
887. Australian Institute of Health and Welfare. Australian National Infant Feeding Survey: Indicator results. In: AIHW, editor. Canberra 2011.
888. Britton C, McCormick FM, Renfrew MJ, Wade A, King SE. Support for breastfeeding mothers. *Cochrane Database Syst Rev* 2007(1):CD001141.
889. Chung M, Raman G, Trikalinos T, Lau J, Ip S. Interventions in primary care to promote breastfeeding: an evidence review for the U.S. Preventive Services Task Force. *Ann Intern Med* 2008;149(8):565–82.
890. Hector D, King L. Interventions to encourage and support breastfeeding. *NSW Public Health Bull* 2005;16(3-4):56–61.
891. Landers M, Hughes R, Graham K. The Darling Downs Breastfeeding Study. Toowoomba: Darling Downs Public Health Unit, 1998.
892. Scott J, Binns C. Breastfeeding: are boys missing out? *Birth (Berkeley, Calif)* 1999;26(4):276.
893. Scott JA, Aitkin I, Binns CW, Aroni RA. Factors associated with the duration of breastfeeding amongst women in Perth, Australia. *Acta Paediatr* 1999;88(4):416–21.
894. Scott JA, Landers MCG, Hughes RM, Binns CW. Psychosocial factors associated with the abandonment of breastfeeding prior to hospital discharge. *J Human Lact* 2001;17(1):24.
895. Simopoulos AP, Oliveira J, Desai ID. Behavioral and metabolic aspects of breastfeeding: international trends. Basel: S Karger AG, 1995.
896. Kramer MS, Guo T, Platt RW, Shapiro S, Collet JP, Chalmers B et al. Breastfeeding and infant growth: biology or bias? *Pediatrics* 2002;110(2 Pt 1):343–7.
897. Kramer MS, Chalmers B, Hoganett ED, Sevkovskaya Z, Dzikovich I, Shapiro S et al. Promotion of Breastfeeding Intervention Trial (PROBIT): a randomized trial in the Republic of Belarus. *J Am Med Assoc* 2001;285(4):413–20.
898. Dewey KG. Growth characteristics of breast-fed compared to formula-fed infants. *Biol Neonate* 1998;74(2):94–105.
899. Demmelmair H, von Rosen J, Koletzko B. Long-term consequences of early nutrition. *Early Hum Dev* 2006;82(3):567–74.
900. Harder T, Bergmann R, Kallischnigg G, Plagemann A. Duration of breastfeeding and risk of overweight: a meta-analysis. *Am J Epidemiol* 2005;162(5):397–403.
901. White House Task Force on Childhood Obesity. Solving the problem of childhood obesity within a generation: report to the President. Washington, DC.: 2010. Available from: http://www.letsmove.gov/pdf/TaskForce_on_Childhood_Obesity_May2010_FullReport.pdf
902. Kuti O, Aleyemi AB, Owolabi AT. Breast-feeding pattern and onset of menstruation among Yoruba mothers of South-west Nigeria. *Eur J Contracept Reprod Healthcare* 2007;12(4):335–39.

903. Romero-Gutiérrez G, Vaca-Ortiz N, Ponce-Ponce de Leon A, López-Martínez MG. Actual use of the lactational amenorrhoea method. *Eur J Contracept Reprod Healthcare* 2007;12(4):340-44.
904. Türk R, Terzio lu F, Ero lu K. The use of lactational amenorrhea as a method of family planning in eastern Turkey and influential factors. *J Midwifery Womens Health* 2010;55(1):e1-e7.
905. Ip S, Chung M, Raman G, Trikalinos TA, Lau J. A summary of the Agency for Healthcare Research and Quality's evidence report on breastfeeding in developed countries. *Breastfeed Med* 2009;4 Suppl 1:S17-30.
906. Oddy WH. The long-term effects of breastfeeding on asthma and atopic disease. *Adv Exp Med Biol* 2009;639:237-51.
907. McGuire W, Anthony M. Donor human milk versus formula for preventing necrotising enterocolitis in preterm infants: systematic review. *Arch Dis Childhood Fetal Neonatal Edition* 2003;88(1):F11.
908. Barclay AR, Russell RK, Wilson ML, Gilmour VH, Satsangi J, Wilson DC. Systematic review: the role of breastfeeding in the development of pediatric inflammatory bowel disease. *J Pediatr* 2009;155(3):421-6.
909. Quigley M, Hockley C, Carson C, Kelly Y, Renfrew M, Sacker A. Breastfeeding is associated with improved child cognitive development: evidence from the UK Millennium Cohort Study. *J Epidemiol Community Health* 2009;63(Suppl 2):2.
910. Medeiros APM, Ferreira JTL, Felício CM. Correlation between feeding methods, non-nutritive sucking and orofacial behaviors. *Pró-Fono Revista de Atualização Científica* 2009;21(4):315-19.
911. Allen J, Hector D. Benefits of breastfeeding. *N SW Public Health Bull* 2005;16(3-4):42-6.
912. Sobhy SI, Mohame NA. 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):1-12.
913. Chantry CJ, Auinger P, Byrd RS. Lactation among adolescent mothers and subsequent bone mineral density. *Arch Pediatr Adolesc Med* 2004;158(7):650.
914. Dursun N, Ak n S, Dursun E, Sade I, Korkusuz F. 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):651-55.
915. Centre for Epidemiology and Research. 2007-2008 Report on child health from the New South Wales population health survey. In: NSW Department of Health, editor. Sydney 2010.
916. Graham KI SJ, Binns CW, Oddy WH. National targets for breastfeeding at hospital discharge have been achieved in Perth. *Acta Paediatr* 2005;94(3):352-56.
917. House of Representatives Standing Committee on Health and Ageing. The best start – Report on the inquiry into the health benefits of breastfeeding. Canberra: Commonwealth of Australia, 2007. <http://www.aph.gov.au/house/committee/haa/breastfeeding/report/fullreport.pdf>
918. Kuan LW, Britto M, Decolongon J, Schoettker PJ, Atherton HD, Kotagal UR. Health system factors contributing to breastfeeding success. *Pediatrics* 1999;104(3):e28.
919. Hall Moran V, Edwards J, Dykes F, Downe S. A systematic review of the nature of support for breast-feeding adolescent mothers. *Midwifery* 2007;23(2):157-71.
920. Volpe EM, Bear M. Enhancing breastfeeding initiation in adolescent mothers through the Breastfeeding Educated and Supported Teen (BEST) Club. *J Human Lact* 2000;16(3):196.
921. Baxter J, Cooklin AR, Smith J. Which mothers wean their babies prematurely from full breastfeeding? An Australian cohort study. *Acta Paediatr* 2009;98(8):1274-77.
922. Cooklin AR, Donath SM, Amir LH. Maternal employment and breastfeeding: results from the longitudinal study of Australian children. *Acta Paediatr* 2008;97(5):620-3.
923. Amir LH, Donath SM. Socioeconomic status and rates of breastfeeding in Australia: evidence from three recent national health surveys. *Med J Aust* 2008;189(5):254-6.

924. Lobbok MH, Clark D, Goldman AS. Breastfeeding: maintaining an irreplaceable immunological resource. *Nat Rev Immunol* 2004;4(7):565–72.
925. Scott J, Landers M, Hughes RM, Binns C. Factors associated with breastfeeding at discharge and duration of breastfeeding. *J Paediatrics Child Health* 2001;37(3):254–61.
926. Duong DV, Binns CW, Lee AH. Breast-feeding initiation and exclusive breast-feeding in rural Vietnam. *Public Health Nutr* 2004;7(6):795–9.
927. Kaewsarn P, Moyle W. Breastfeeding duration of Thai women. *Aust Coll Midwives Inc J* 2000;13(1):21–6.
928. Li L, Zhang M, Scott JA, Binns CW. Factors associated with the initiation and duration of breastfeeding by Chinese mothers in Perth, Western Australia. *J Hum Lact* 2004;20(2):188–95.
929. Utaka H, Li L, Kagawa M, Okada M, Hiramatsu N, Binns C. Breastfeeding experiences of Japanese women living in Perth, Australia. *Breastfeed Rev* 2005;13(2):5–11.
930. Xu F, Qiu L, Binns CW, Liu X. Breastfeeding in China: a review. *Int Breastfeed J* 2009;4:6.
931. Binns CW, Gilchrist D, Woods B, Gracey M, Scott J, Smith H et al. Breastfeeding by Aboriginal mothers in Perth. *Nutr Diet* 2006;63(1):8–14.
932. World Health Organization. Acceptable medical reasons for use of breastmilk substitutes. Geneva: World Health Organization, 2009. http://whqlibdoc.who.int/hq/2009/WHO_FCH_CAH_09.01_eng.pdf
933. Guise JM, Palda V, Westhoff C, Chan BK, Helfand M, Lieu TA. 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):70–8.
934. Baby Friendly Health Initiative. The ten steps to successful breastfeeding. 2011 (cited 29 March 2011). http://www.bfhi.org.au/text/bfhi_ten_steps.html. <http://www.patonsyarns.com/pattern.ph>
935. Abrahams SW, Lobbok MH. Exploring the impact of the Baby-Friendly Hospital Initiative on trends in exclusive breastfeeding. *Int Breastfeed J* 2009;4(11).
936. Broadfoot M et al. The Baby Friendly Hospital Initiative and breast feeding rates in Scotland. *Arch Dis Child Fetal Neonatal Ed* 2005;F114–16.
937. Merten S, Dratva J, Ackermann-Liebrich U. Do baby-friendly hospitals influence breastfeeding duration on a national level? *Pediatrics* 2005;116(5):e702–08.
938. Binns C, Scott J. Breastfeeding: reasons for starting, reasons for stopping and problems along the way. *Breastfeed Rev* 2002;10(2):13–19.
939. Australia New Zealand Food Authority. Food safety standards: costs and benefits. An analysis of the regulatory impact of the proposed national food safety reforms Canberra: Australia New Zealand Food Authority, 1999. <http://www.foodstandards.gov.au/scienceandeducation/publications/foodsafetystandardscostsandbenefits/>
940. Bacterial toxins, National Food Safety database [database on the Internet]. 2001. Available from: www.foodsafety.ufl.edu/consumer/nc/nc816a.htm.
941. Food Standards Australia New Zealand. Date marking: user guide to Standard 1.2.5, Date Marking of Packaged Food. In: FSANZ, editor. Canberra 2010.

942. Käferstein F, Abulussalam M. Food safety in the 21st century. *Bull World Health Organ* 1999;77(4):347.
943. Desmarchelier PM. Foodborne disease: emerging problems and solutions. *Med J Aust* 1996;165 (11-12):668–71.
944. Nyachuba DG. Foodborne illness: is it on the rise? *Nutr Rev* 2010;68(5):257–69.
945. The OzFoodNet Working Group. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet Network 2009. *Communicable Diseases Intelligence* 2010;32(4).
946. Abelson P, Forbes MP, Hall G, Economics A, Health ADo, Ageing. The annual cost of foodborne illness in Australia. Canberra: Department of Health and Ageing, 2006.
947. Crerar SK, Dalton CB, Longbottom HM, Kraa E. Foodborne disease: current trends and future surveillance needs in Australia. *Med J Aust* 1996;165:672–5.
948. Redmond EC, Griffith CJ. Consumer food handling in the home: a review of food safety studies. *J Food Prot* 2003;66(1):130–61.
949. Australia New Zealand Communicable Diseases Network. Fortnightly notifiable diseases tables: *Communicable Diseases Intelligence*, 1999.
950. Redmond EC, Griffith CJ. 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):69–76.
951. State Government of Victoria. Food safety at home. In: Department of Health Victoria, editor. Melbourne, Victoria. 2011.
952. Merry G. Food poisoning prevention. Sydney: Palgrave Macmillan Australia, 1997.
953. Sprenger RA. Hygiene for management: a text for food hygiene courses. South Yorkshire UK Highfield, 1998.
954. US Department of Agriculture. Kitchen companion: your safe food guide. Washington, DC.: US Department of Agriculture; 2008.
955. Brewer MS. Food storage, food spoilage and foodborne illness. 1991 (cited). www.aces.uiuc.edu/~fshn/extension/food_storage.html, <http://www.patonsyarns.com/pattern.ph>
956. Almanza BA, Namkung Y, Ismail JA, Nelson DC. Clients' safe food-handling knowledge and risk behavior in a home-delivered meal program. *J Am Diet Assoc* 2007;107(5):816–21.
957. Kendall PA, Hillers VV, Medeiros LC. Food safety guidance for older adults. *Clin Infect Dis* 2006;42(9):1298–304.
958. US Food Safety and Inspection Service. Safe food to go. In: Department of Agriculture, editor. Washington, DC 1997.
959. Cates SC, Carter-Young HL, Conley S, O'Brien B. Pregnant women and listeriosis: preferred educational messages and delivery mechanisms. *J Nutr Educ Behav* 2004;36(3):121–7.
960. 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. Geneva: Food And Agriculture Organization of the United Nations and the World Health Organization, 1992.
961. Lester IH. Australia's food and nutrition. Canberra: Australian Government Publishing Service, 1994.
962. Australia PHAo. Health inequities policy. Canberra: Public Health Association of Australia, 2009.
963. Atlantis E, Barnes EH, Ball K. Weight status and perception barriers to healthy physical activity and diet behavior. *Int J Obes (Lond)* 2008;32(2):343–52.
964. Baker EA, Schootman M, Barnidge E, Kelly C. The role of race and poverty in access to foods that enable individuals to adhere to dietary guidelines. *Prev Chronic Dis* 2006;3(3):A76.

965. Bodnar LM, Siega-Riz AM. A diet quality index for pregnancy detects variation in diet and differences by socio-demographic factors. *Public Health Nutr* 2002;5(6):801–9.
966. Cassady D, Jetter KM, Culp J. Is price a barrier to eating more fruits and vegetables for low-income families? *J Am Diet Assoc* 2007;107(11):1909–15.
967. Giskes K, Turrell G, van Lenthe FJ, Brug J, Mackenbach JP. 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):75–83.
968. Giskes K, Van Lenthe FJ, Brug J, Mackenbach JP, Turrell G. Socioeconomic inequalities in food purchasing: the contribution of respondent-perceived and actual (objectively measured) price and availability of foods. *Prev Med* 2007;45(1):41–8.
969. Kolodinsky J, Harvey-Berino JR, Berlin L, Johnson RK, Reynolds TW. Knowledge of current dietary guidelines and food choice by college students: better eaters have higher knowledge of dietary guidance. *J Am Dietetic Assoc* 2007;107(8):1409–13.
970. Inglis V, Ball K, Crawford D. Why do women of low socioeconomic status have poorer dietary behaviours than women of higher socioeconomic status? A qualitative exploration. *Appetite* 2005;45(3):334–43.
971. McNaughton SA, Ball K, Crawford D, Mishra GD. An index of diet and eating patterns is a valid measure of diet quality in an Australian population. *J Nutr* 2008;138(1):86–93.
972. Nelson M, Dick K, Holmes B. Food budget standards and dietary adequacy in low-income families. *Proc Nutr Soc* 2002;61(4):569–77.
973. Sanchez A, Norman GJ, Sallis JF, Calfas KJ, Cella J, Patrick K. Patterns and correlates of physical activity and nutrition behaviors in adolescents. *Am J Prev Med* 2007;32(2):124–30.
974. Story M, Neumark-Sztainer D, French S. Individual and environmental influences on adolescent eating behaviors. *J Am Diet Assoc* 2002;102(3 Suppl):S40–51.
975. Turrell G, Kavanagh AM. Socio-economic pathways to diet: modelling the association between socio-economic position and food purchasing behaviour. *Public Health Nutr* 2006;9(3):375–83.
976. Turrell G, Hewitt B, Patterson C, Oldenburg B. Measuring socio-economic position in dietary research: is choice of socio-economic indicator important? *Public Health Nutr* 2003;6(2):191–200.
977. Collins CE, Young AF, Hodge A. Diet quality is associated with higher nutrient intake and self-rated health in mid-aged women. *J Am Coll Nutr* 2008;27(1):146–57.
978. Dynesen AW, Haraldsdottir J, Holm L, Astrup A. Socio-demographic differences in dietary habits described by food frequency questions--results from Denmark. *Eur J Clin Nutr* 2003;57(12):1586–97.
979. Hearty AP, McCarthy SN, Kearney JM, Gibney MJ. Relationship between attitudes towards healthy eating and dietary behaviour, lifestyle and demographic factors in a representative sample of Irish adults. *Appetite* 2007;48(1):1–11.
980. Oldroyd J, Burns C, Lucas P, Haikerwal A, Waters E. The effectiveness of nutrition interventions on dietary outcomes by relative social disadvantage: a systematic review. *J Epidemiol Community Health* 2008;62(7):573–9.
981. Power EM. Determinants of healthy eating among low-income Canadians. *Can J Public Health* 2005;96 Suppl 3:S37–42, S42–8.
982. Wong KC, Coveney J, Ward P, Muller R, Carter P, Verity F et al. Availability, affordability and quality of a healthy food basket in Adelaide, South Australia. *Nutr Diet* 2011;68(1):8–14.
983. Statistics ABo. National Aboriginal and Torres Strait Islander Survey 1994. In: ABS, editor. Canberra: Commonwealth of Australia; 1994.
984. Andrieu E, Darmon N, Drewnowski A. Low-cost diets: more energy, fewer nutrients. *Eur J Clin Nutr* 2006;60(3):434–6.

985. Centre for Epidemiology and Research. 2007 report on adult health from the New South Wales Population Health Survey. Sydney: NSW Department of Health, 2008b.
986. Department of Human Services. Victorian Population Health Survey 2007. Melbourne: Department of Human Services, 2008.
987. Burns C. A review of the literature describing the link between poverty, food insecurity and obesity with specific reference to Australia. Melbourne: Victorian Health Promotion Foundation, 2004.
988. Australian Institute of Health and Welfare. Aboriginal and Torres Strait Islander Health Performance Framework, 2006 report: detailed analyses. . In: AIHW, editor. Canberra: AIHW; 2007.
989. Australian Bureau of Statistics. Consumer price index, Australia, March 2011 - Cat. no. 6401.0. 2011 (cited 2011). <http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/6401.0Mar%202011?OpenDocument>. <http://www.patonsyarns.com/pattern.ph>
990. Baghurst KI. Social status, nutrition and the cost of healthy eating. In: Baghurst KI, Binns C, eds. Dietary guidelines for Australian adults. Canberra: National Health and Medical Research Council, 2003.
991. Sanchez-Villegas A, Martinez JA, Prattala R, Toledo E, Roos G, Martinez-Gonzalez MA. A systematic review of socioeconomic differences in food habits in Europe: consumption of cheese and milk. *Eur J Clin Nutr* 2003;57(2):917–29.
992. Vos T. The burden of disease and injury in Aboriginal and Torres Strait Islander peoples 2003. Brisbane: University of Queensland Centre for Burden of Disease and Cost-Effectiveness, 2007.
993. Carson B, Dunbar T, Chenhall RD. Social determinants of Indigenous health. Sydney: Allen & Unwin Academic, 2007.
994. Torzillo PJ, Pholeros P, Rainow S, Barker G, Sowerbutts T, Short T et al. The state of health hardware in Aboriginal communities in rural and remote Australia. *Aust N Z J Public Health* 2008;32(1):7–11.
995. Australian Bureau of Statistics, Australian Institute of Health and Welfare. The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples Canberra: Australian Institute of Health and Welfare, 2008. Available from: <http://www.aihw.gov.au/publication-detail/?id=6442468085>
996. Ruben AR, Walker AC. Malnutrition among rural aboriginal children in the Top End of the Northern Territory. *Med J Aust* 1995;162(2):400.
997. Sayers SM, Mackerras D, Singh G, Bucens I, Flynn K, Reid A. 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):1.
998. Northern Territory Government. Department of Health and Community Services: Annual Report 2006-2007. Casuarina: Department of Health and Community Services, 2007. Available from: http://www.health.nt.gov.au/library/scripts/objectifyMedia.aspx?file=polf/10/05.polf&siteID=1&str_title=DHCS+Annual+Report+2006-2007.polf
999. Northern Territory Government. Northern Territory Market Basket Survey 2008. Darwin: Northern Territory Department of Health and Families; 2008.
1000. Mackerras DE, Reid A, Sayers SM, Singh GR, Bucens IK, Flynn KA. Growth and morbidity in children in the Aboriginal birth cohort study: the urban-remote differential. *Med J Aust* 2003;178(2):56–60.
1001. Vanlint SJ, Morris HA, Newbury JW, Crockett AJ. Vitamin D insufficiency in Aboriginal Australians. *Women* 2011;100:120.
1002. Coyne T, Darnton-Hill I. Australian Aborigines' nutrition and changing disease patterns. *New Doctor* 1979;12:32–7.
1003. Naughton JM, O'Dea K, Sinclair AJ. Animal foods in traditional Australian aboriginal diets: polyunsaturated and low in fat. *Lipids* 1986;21(11):684–90.
1004. O'Dea K. Traditional diet and food preferences of Australian Aboriginal hunter-gatherers. *Philos Trans R Soc Lond B Biol Sci* 1991;334(1270):233–40; discussion 40–1.

1005. Shannon C. Acculturation: Aboriginal and Torres Strait Islander nutrition. *Asia Pac J Clin Nutr* 2002; 11 Suppl 3:S576–8.
1006. Lee A. Transition of Australian Aboriginal diet and nutritional health. *World Rev Nutr Diet* 1996;79:1–52.
1007. Booth S, Smith A. Food security and poverty in Australia-challenges for dietitians. *Aust J Nutrition Dietetics* 2001;58(3):150–56.
1008. House of Representatives Standing Committee on Aboriginal and Torres Strait Islander affairs. Everybody's business: remote Aboriginal and Torres Strait community stores. Canberra: Commonwealth of Australia, 2009. <http://www.aph.gov.au/house/committee/ATSIA/communitystores/http://www.aph.gov.au/house/committee/ATSIA/communitystores/report.htm>
1009. Leonard D, Beilin R, Moran M. Which way kaikai blo umi? Food and nutrition in the Torres Strait. *Aust J Public Health* 1995;19(6):589–95.
1010. Meedeniya J, Smith A, Carter P. Food supply in rural South Australia: a survey on food cost, quality and variety. 2000.
1011. Beaumont S. Tasmanian food price, availability, and quality survey. Hobart: Tasmanian Department of Community and Health Services, 1998.
1012. Bowcock R. Kimberley Market Basket Survey. Derby: Kimberley Public Health, 1999.
1013. Price R, McComb J, Grieve H, Graham E. 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.
1014. Sullivan H, Gracey M, Hevron V. Food costs and nutrition of Aborigines in remote areas of northern Australia. *Med J Aust* 1987;147(7):334–7.
1015. 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: 63–70.
1016. House of Representatives. Inquiry into community stores in remote Aboriginal and Torres Strait Islander communities 2009. Canberra: Commonwealth of Australia; 2009.
1017. Altman J. Hunter-gatherers today: An Aboriginal economy in north Australia. Canberra: Australian Institute of Aboriginal Studies, 1987.
1018. Devitt J. Traditional Aboriginal preferences in a changed context. *Central Australian Rural Practitioners' Association Newsletter* 1991;14:38–41.
1019. Rae C, Lamprell V, Lion R, Rae A. The role of bushfoods in contemporary Aboriginal diets. *Proc Nut Soc Aus* 1982;7:45–9.
1020. Browne J, Laurence S, Thorpe S. 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. Melbourne: Victorian Aboriginal Community Controlled Health Organisation, 2009. Available from: <http://www.healthinonet.ecu.edu.au/health-risks/nutrition/reviews/other-reviews>
1021. Guest CS, O'Dea K. Diabetes in aborigines and other Australian populations. *Aust J Public Health* 1992;16(4):340–9.
1022. Doyle J, Reilly R, Cincotta M, Firebrace B, Rowley K. Developing nutrition and physical activity guidelines and interventions for Aboriginal and Torres Strait Islander communities. Shepparton: Onemda VicHealth Koori Health Unit, 2007.
1023. Clough AR, Rowley K, O'Dea K. 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):1090–93.
1024. Chan LCK, Ware RS, Kesting J, Marczak M, Good D, Shaw JTE. 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):515.

1025. Lee A, Bonson A, Yarmirr D, O'Dea K, JD M. Sustainability of a successful health and nutrition program in a remote Aboriginal community. *MJA* 1995;162(12):632–35.
1026. Rowley KG, Daniel M, Skinner K, Skinner M, White GA, O'Dea K. Effectiveness of a community directed 'healthy lifestyle' program in a remote Australian Aboriginal community. *Aust N Z J Public Health* 2000;24(2):136–44.
1027. Johannes RE, MacFarlane JW. *Traditional fishing in the Torres Strait islands*. Cleveland, Queensland: CSIRO Division of Fisheries, Marine Laboratories, 1991.
1028. Haynes D M-WK, Kwan D. Trace metal concentrations in Torres Strait green turtle (*Chelonia mydas*) tissues, 1997–1998. . Report prepared for the Torres Strait Regional Authority, 2001.
1029. Australian Bureau of Statistics. Australian social trends 2010 (cited). www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/4102.0Main+Features30Mar+2010. <http://www.patonsyarns.com/pattern.ph>
1030. Australian Bureau of Statistics. Census provides an updated picture of the average Australian [Media fact sheet]. 2007 (cited). www.abs.gov.au/ausstats/abs@.nsf/7d12b0f6763c78caca257061001cc588/96e08af8f4178a45ca257306000d4fd4!OpenDocument. <http://www.patonsyarns.com/pattern.ph>
1031. Cancer Council NSW. NSW healthy food basket cost, availability and quality survey. Sydney: Cancer Council NSW, 2007. <http://www.cancercouncil.com.au/editorial.asp?pageid=2389>
1032. National Health and Medical Research Council. The core food groups: the scientific basis for developing nutrition education tools. Canberra: Commonwealth of Australia, 1995. <http://www.nhmrc.gov.au/publications/synopses/n16covr.htm>
1033. National Health and Medical Research Council. Implementing the dietary guidelines for Australians. Report on the subcommittee on nutrition education. Canberra 1989.
1034. Cashel K LJ. NUTTAB89-Nutrient Data Table for Use in Australia [Floppy Disc]. In: Department of Community Services and Health, editor. Canberra 1989.
1035. National Health and Medical Research Council. Recommended dietary intakes for use in Australia. Canberra: Commonwealth of Australia, 1991. <http://www.nhmrc.gov.au/publications/synopses/n6syn.htm>
1036. Sobolewski R CJ, Mackerras D. Which Australian food composition database should I use? *Nutr Dietetics* 2010;67:37-40.
1037. Food Standards Australia and New Zealand. AUSNUT 2007. 2007 (updated September 2011; cited September 2011). <http://www.foodstandards.gov.au/consumerinformation/ausnut2007/>. <http://www.patonsyarns.com/pattern.ph>
1038. National Public Health Partnership. National Aboriginal and Torres Strait Islander nutrition strategy and action plan 2000-2010. 2001.
1039. Food Standards Australia and New Zealand. NUTTAB 2010. 2010 (updated September 2011; cited September 2011). <http://www.foodstandards.gov.au/consumerinformation/nuttab2010/>. <http://www.patonsyarns.com/pattern.ph>
1040. Larsen K, Ryan C, Abraham AB. Sustainable and secure food systems for Victoria: What do we know? What do we need to know? Melbourne: University of Melbourne, 2008.
1041. Lang T. Reshaping the food system for ecological public health. *Journal of Hunger & Environmental Nutrition* 2009;4(3-4):315-35.
1042. Riley H & Buttriss JL. A UK public health perspective: What is a healthy sustainable diet? *British Nutrition Foundation Nutrition Bulletin* 2011; 36:426-31.
1043. Vieux F, Darmon N, Touazi D, Soler LG. Greenhouse gas emissions of self-selected individual diets in France: Changing the diet structure or consuming less? *Ecological Economics* 2012;75:91-101.
1044. Edwards P, Roberts I. Population adiposity and climate change. *International Journal of Epidemiology* 2009; 38 (4):1137-40.

1045. Golley RK, Henneke GA, McNaughton SA. Scores on the dietary guideline index for children and adolescents are associated with nutrient intake and socio-economic position but not adiposity. *The Journal of Nutrition* 2011;141:1340-47.
1046. World Health Organisation/Food and Agriculture Organisation. Diet, nutrition and the prevention of chronic diseases: Report of the joint WHO/FAO Expert Consultation. Geneva: 2003. Available from: http://whqlibdoc.who.int/trs/who_trs_916.pdf
1047. Parfitt J, Barthel M, Macnaughton S. Food waste within food supply chains: quantification and potential for change to 2050. *Phil Trans R Soc B* 2010;365:3065-81.
1048. Wallgren C, Höjer M. Eating energy-Identifying possibilities for reduced energy use in the future food supply system. *Energy Policy* 2009;37(12):5803-13.
1049. Carlsson-Kanyama A, Boström-Carlsson K. Energy use for cooking and other stages in the life cycle of food. Stockholm, Sweden: Stockhoms Universitet, 2001.
1050. Baker D, Fear J, Denniss R. What a waste-an analysis of household expenditure on food: Policy Brief No. 6., The Australian Institute.; 2009.
1051. Foresight. The future of food and farming. London: The Government Office for Science; 2011.
1052. Foster C, Green K, Blea M, Dewick P, Evans B, Flynn A et al. Environmental impacts of food production and consumption: A report to the department of environment, food and rural affairs. London: DEFRA, 2006.
1053. Food Ethics Council. Sustainable behaviour - if we really cared, wouldn't we pay more? Business Forum Meeting; 13th September 2007.
1054. Bunn D, Feenstra GW, Lynch L, Sommer R. Consumer Acceptance of Cosmetically Imperfect Produce. *Journal of Consumer Affairs* 1990;2:268-79.
1055. Australia State of the Environment Report. Independent report to the Australian Government Minister for Sustainability, Environment, Water, Population and Communities. In: Department of Sustainability, Environment, Water, Population, and Communities, editors. Canberra 2011.
1056. Burlingame B, Dernini S. Sustainable diets and biodiversity: Directions and solutions for policy, research and action: United Nations Food and Agriculture Organization.; 2012.
1057. PMSEIC. Australia and food security in a changing world. In: The Prime Minister's Science Engineering and Innovation Council, editor. Canberra 2010.
1058. Huang MH, Rust RT. Sustainability and consumption. *J of the Acad Mark Science* 2011;39:40-54.
1059. Department of Agriculture, Fisheries, and Forestry. National Food Plan. 2012 (cited). www.daff.gov.au/nationalfoodplan/national-food-plan.<http://www.patonsyarns.com/pattern.ph>
1060. IOM (Institute of Medicine) and NRC (National Research Council). Exploring health and environmental costs of food: Workshop summary. Washington DC.; The National Academies Press, 2012.
1061. Ridoutt BG, Sangsri P, Freer M, Harper GS. Water footprint of livestock: comparison of six geographically defined beef production systems. *International Journal of Lifecycle Assessment* 2012;17:165-75.
1062. Lenzen M et al. Environmental impact assessment including indirect effects - a case study using input-output analysis. *Environmental Impact Assessment Review* 2003;23(3):263-82.
1063. Closs DJ, Speier C, Meacham N. Sustainability to support end-to-end value chains: the role of supply chain management. *Journal of the Academy of Marketing Science* 2011;39:101-16.
1064. Hult GTM. Market-focused sustainability: market orientation plus! *Journal of the Academy of Marketing Science* 2011;39:1-6.
1065. Jackson T. Prosperity without growth? A transition to a sustainable economy. London, UK: Sustainable Development Commission; 2009.

1066. Natural Resource Management Ministerial Council 2010. Australia's Biodiversity Conservation Strategy 2010-2030. In: Department of Sustainability, Environment, Water, Population, and Communities, editors. Canberra 2010.
1067. Department of Sustainability, Environment, Water, Population and Communities. Fact Sheet: Measuring sustainability program. In: SEWPaC, editor. Canberra 2012.
1068. CSIRO. Sustainability. 2012 (cited). www.csiro.au/Outcomes/Environment/Population-Sustainability.aspx. <http://www.patonsyarns.com/pattern.ph>
1069. Sheth JN, Sethia NK, Srinivas S. Mindful consumption: a consumer-centric approach to sustainability. *Journal of the Acad Mark Sci* 2011;39:21-39.
1070. Friel S, Dangour AD, Garnett T, Lock K, Chalabi Z, Roberts I et al. Public health benefits of strategies to reduce greenhouse gas emissions: food and agriculture. *Lancet* 2009;374(2016-25).
1071. McMichael AJ, Powles JW, Butler CD, Uauy R. Food, livestock production, energy, climate change and health. *Lancet* 2007;370:1253-63.
1072. Powles J. Commentary: Why diets need to change to avert harm from global warming. *International journal of epidemiology* 2009;38(4):1141-42.
1073. Macdiarmid J, Kyle J, Horgan GW, Loe J, Fyfe C, Johnstone A et al. Sustainable diets for the future: can we contribute to reducing greenhouse gas emissions by eating a healthy diet? *Am J Clin Nutr* 2012;96:632-39.
1074. Denney-Wilson E, Booth M, Baur L. Development of the Australian standard definition of child/adolescent overweight and obesity. *Nutr Diet* 2003;60(2):74-77.
1075. World Health Organization. Growth reference data for 5-19 years. 2007 (cited 30 March 2011). <http://www.who.int/growthref/en/>. <http://www.patonsyarns.com/pattern.ph>
1076. WHO Statistical Information System. Children aged <5 years. 2008 (cited 30 March 2011). <http://www.who.int/whosis/indicators/compendium/2008/2nu5/en/>. <http://www.patonsyarns.com/pattern.ph>
1077. World Health Organization. The WHO child growth standards. 2006 (cited 30 June 2011). <http://www.who.int/childgrowth/en/>. <http://www.patonsyarns.com/pattern.ph>
1078. Monasta L, Lobstein T, Cole TJ, Vigneronova J, Cattaneo A. Defining overweight and obesity in pre-school children: IOTF reference or WHO standard? *Obes Rev* 2011;12(4):295-300.
1079. de Onis M, Onyango A, Borghi E, Siyam A, Blossner M, C L. Worldwide implementation of the WHO Child Growth Standards. *Public Health Nutrition* 2010:1-8.
1080. Centres for Disease Control and Prevention. Growth charts. 2010 (cited 30 March 2011). <http://www.cdc.gov/growthcharts/>. <http://www.patonsyarns.com/pattern.ph>
1081. McLennan J. Obesity in children. Tackling a growing problem. *Aust Fam Physician* 2004;33(1-2):33-6.
1082. Pietrobelli A, Faith MS, Allison DB, Gallagher D, Chiumello G, SB H. Body mass index as a measure of adiposity among children and adolescents: a validation study. *J Pediatr* 1998;132:204-10.
1083. Himes JH, WH D. Guidelines for overweight in adolescent preventative services: recommendations from an expert committee. *Am J Clin Nutr* 1994;59:307-16.
1084. Bellizzi MC, WH D. Workshop on childhood obesity: summary of the discussion. *Am J Clin Nutr* 1999;70(173S-175S).
1085. Dietz WH, Bellizzi MC. Introduction: the use of body mass index to assess obesity in children. *Am J Clin Nutr* 1999;70(1):123S-5S.
1086. Beyerlein A, Toschke AM, von Kries R. 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):642-8.

1087. Lee JM, Pilli S, Gebremariam A, Keirns CC, Davis MM, Vijan S et al. Getting heavier, younger: Trajectories of obesity over the life course. *Int J Obes (Lond)* 2010;34(4):614–23.
1088. Nambiar S, Truby H, Abbott RA, Davies PSW. Validating the waist height ratio and developing centiles for use amongst children and adolescents. *Acta Paediatr* 2009;98(1):148–52.
1089. Sanigorski A, Bell A, Kremer P, Cuttler R, Swinburn B. 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):1060–67.
1090. Rasmussen-Torvik LJ, Pankow JS, Jacobs DR, Jr., Steinberger J, Moran AM, Sinaiko AR. Influence of waist on adiponectin and insulin sensitivity in adolescence. *Obesity (Silver Spring)* 2009;17(1):156–61.
1091. Valery PC, Moloney A, Cotterill A, Harris M, Sinha AK, Green AC. Prevalence of obesity and metabolic syndrome in Indigenous Australian youths. *Obes Rev* 2009;10(3):255–61.
1092. McCarthy HD. Body fat measurements in children as predictors for the metabolic syndrome: focus on waist circumference. *Proc Nutr Soc* 2006;65(04):385–92.
1093. Watts K, Bell LM, Byrne SM, Jones TW, Davis EA. Waist circumference predicts cardiovascular risk in young Australian children. *J Paediatr Child Health* 2008;44(12):709–15.
1094. Garnett SP, Baur LA, CT C. Waist-to-height ratio: a simple option for determining excess central adiposity in young people. *Int J Obes (Lond)* 2008;32(6):1028–30.
1095. McCarthy HD, M A. A study of central fatness using waist-to-height ratios in UK children and adolescents over two decades supports the simple message-- 'keep your waist circumference to less than half your height'. *Int J Obes (Lond)* 2006;30(6):988–92.
1096. Taylor RW, Williams SM, Grant AM, Taylor BJ, A G. Predictive ability of waist-to-height in relation to adiposity in children is not improved with age and sex-specific values. *Obesity (Silver Spring)* 2011;19(5):1062–8.
1097. Ashwell M, Gunn P, S G. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. *Obesity Reviews* 2012;13(3):275–86.
1098. Eisenmann JC, Welk GJ, Ihmels M, Dollman J. Fatness, fitness, and cardiovascular disease risk factors in children and adolescents. *Med Sci Sports Exerc* 2007;39(8):1251.
1099. Telford RD, Cunningham RB, Daly RM, Reynolds GJ, Lafferty AR, Gravenmaker KJ et al. Discordance of international adiposity classifications in Australian boys and girls - the LOOK study. *Ann Hum Biol* 2008;35(3):334–41.
1100. Dhaliwal SS, Welborn TA. Central obesity and multivariable cardiovascular risk as assessed by the Framingham prediction scores. *Am J Cardiol* 2009;103(10):1403–7.
1101. Welborn TA, Dhaliwal SS. Preferred clinical measures of central obesity for predicting mortality. *Eur J Clin Nutr* 2007;61(12):1373–9.
1102. Department of Health and Ageing. Physical activity guidelines. 2010 (cited 29 March 2011). <http://www.health.gov.au/internet/main/publishing.nsf/content/health-pubhlth-strateg-phys-act-guidelines>. <http://www.patonsyarns.com/pattern.ph>
1103. Australian Government Department of Health and Ageing. National Physical Activity Guidelines for Adults. 2005 (cited). <http://www.health.gov.au/internet/main/publishing.nsf/content/phd-physical-activity-adults-pd-f-cnt.htm> <http://www.patonsyarns.com/pattern.ph>
1104. Miller YD, Dunstan DW. 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):52–9.
1105. Genkinger JM, Platz EA, Hoffman SC, Comstock GW, Helzlsouer KJ. 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):1223.

1106. Liu Y, Sobue T, Otani T, Tsugane S. Vegetables, fruit consumption and risk of lung cancer among middle-aged Japanese men and women: JPHC study. *Cancer Causes Control* 2004;15(4):349–57.
1107. Smith-Warner SA, Spiegelman D, Yaun SS, Albanes D, Beeson WL, van den Brandt PA et al. Fruits, vegetables and lung cancer: a pooled analysis of cohort studies. *Int J Cancer* 2003;107(6):1001–11.
1108. Wright ME, Park Y, Subar AF, Freedman ND, Albanes D, Hollenbeck A 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):1024–34.
1109. Boyle P, Levin B. *World Cancer Report 2008*. World Cancer Report 2008.
1110. van Duijnhoven FJB, Bueno-De-Mesquita HB, Ferrari P, Jenab M, Boshuizen HC, Ros MM et al. Fruit, vegetables, and colorectal cancer risk: the European Prospective Investigation into Cancer and Nutrition. *Am J Clin Nutr* 2009;89(5):1441.
1111. Lee JE, Mannisto S, Spiegelman D, Hunter DJ, Bernstein L, van den Brandt PA 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):1730–9.
1112. Dolwick Grieb SM, Theis RP, Burr D, Benardot D, Siddiqui T, Asal NR. Food groups and renal cell carcinoma: results from a case-control study. *J Am Diet Assoc* 2009;109(4):656–67.
1113. Weikert S, Boeing H, Pischon T, Olsen A, Tjønneland A, Overvad K 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):3133–39.
1114. Rashidkhani B, Lindblad P, Wolk A, Rashidkhani B, Lindblad P, Wolk A. Fruits, vegetables and risk of renal cell carcinoma: a prospective study of Swedish women. *Int J Cancer* 2005;113(3):451–5.
1115. World Cancer Research Fund, American Institute for Cancer Research. *Food, nutrition, and the prevention of cancer: a global perspective*. Washington DC: American Institute for Cancer Research, 1997.
1116. Stewart BW, Kleihues P. *World Cancer Research Report*. Lyon: International Agency for Research on Cancer, 2003. <http://www.iarc.fr/en/publications/pdfs-online/wcr/2003/index.php>
1117. Boffetta P, Couto E, Wichmann J, Ferrari P, Trichopoulos D, Bueno-de-Mesquita HB 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):529.
1118. Liang W, Binns CW. Fruit, vegetables, and colorectal cancer risk: the European Prospective Investigation into Cancer and Nutrition. *Am J Clin Nutr* 2009;90(4):1112.
1119. Miura K, Greenland P, Stamler J, Liu K, Davi G, Nakagawa H 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):572–80.
1120. Freedman ND, Park Y, Subar AF, Hollenbeck AR, Leitzmann MF, Schatzkin A 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):2330–6.
1121. Ahn YS, Jy W, Harrington WJ, Shanbaky N, Fernandez LF, Haynes DH. Increased platelet calcium in thrombosis and related disorders and its correction by nifedipine. *Thromb Res* 1987;45(2):135–43.
1122. Alberola A, Clarke CS, Haynes DA, Pascu SI, Rawson JM. Crystal structures and magnetic properties of a sterically encumbered dithiadiazolyl radical, 2,4,6-(F3C)3C6H2CNSSN. *Chem Commun (Camb)* 2005(37):4726–8.
1123. Atkins GJ, Bouralexis S, Evdokiou A, Hay S, Labrinidis A, Zannettino AC et al. Human osteoblasts are resistant to Apo2L/TRAIL-mediated apoptosis. *Bone* 2002;31(4):448–56.
1124. Sunny L, Sunny L. 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):490–6.

1125. Ello-Martin JA, Roe LS, Ledikwe JH, Beach AM, Rolls BJ. Dietary energy density in the treatment of obesity: a year-long trial comparing 2 weight-loss diets. *Am J Clin Nutr* 2007;85(6):1465–77.
1126. Whybrow S, Harrison CLS, Mayer C, Stubbs RJ. Effects of added fruits and vegetables on dietary intakes and body weight in Scottish adults. *Br J Nutr* 2006;95(3):496–503.
1127. Vrieling A, Verhage BAJ, van Duijnhoven FJB, Jenab M, Overvad K, Tjønneland 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):1926–34.
1128. Larsson SC, Bergkvist L, Wolk A. Fruit and vegetable consumption and incidence of gastric cancer: a prospective study. *Cancer Epidemiol Biomarkers Prev* 2006; 15(10): 1998–2001.

Notes

Notes

www.nhmrc.gov.au
www.eatforhealth.gov.au

GPO Box 1421, Canberra ACT 2601

16 Marcus Clarke Street, Canberra City ACT

T. 13 000 NHMRC (13 000 64672) or +61 2 6217 9000 F. 61 2 6217 9100 E. nhmrc@nhmrc.gov.au