

Dietary Guidelines for Australian Adults

Endorsed 10 April 2003



NHMRC

*National Health &
Medical Research Council*

© Commonwealth of Australia 2003

ISBN Print: 1864961414 Online:186496135X

This work is copyright. Apart from any use as permitted under the *Copyright Act 1968*, no part may be reproduced by any process without prior written permission from AusInfo. Requests and enquiries concerning reproduction and rights should be addressed to the Manager, Legislative Services, AusInfo, GPO Box 1920, Canberra ACT 2601. Email address: cwealthcopyright@dcita.gov.au

The strategic intent of the NHMRC is to provide leadership and work with other relevant organisations to improve the health of all Australians by:

- fostering and supporting a high quality and internationally recognised research base;
- providing evidence based advice;
- applying research evidence to health issues thus translating research into better health practice and outcomes; and
- promoting informed debate on health and medical research, health ethics and related issues.

Materials including a poster, booklet and brochure for the general public and nutrition educators are available by contacting the Population Health Publications Officer, Commonwealth Department of Health and Ageing on toll free 1800 020 103 Ext 8654 or at email: phd.publications@health.gov.au

The Australian dietary guidelines and Food for Health information can also be found on the internet at <http://www.nhmrc.gov.au/publications/nhome.htm>

Reliable information about food, nutrition and health is also available from:

- Nutrition Australia — www.nutritionaustralia.org
- Dietitians Association of Australia (DAA) — www.daa.asn.au
- Food Standards Australia New Zealand (FSANZ) — www.foodstandards.gov.au who also produce *The official shopper's guide to food additives and labels: know what you are eating at a glance* (published by Murdoch)
- Local community health centres
- State Departments of health
- Baby, child and youth health centres
- Accredited practising dietitians in private practice (look in the yellow pages) or in hospitals and community centres
- National Heart Foundation of Australia — www.heartfoundation.com.au
- Diabetes Australia — www.diabetesaustralia.com.au

Disclaimer

This document is a general guide to appropriate practice, to be followed only subject to the clinician's judgement in each individual case.

The guidelines are designed to provide information to assist decision-making and are based on the best information available at the date of compilation.

It is planned to review this Guideline in 2008. For further information regarding the status of this document, please refer to the NHMRC web address: <http://www.nhmrc.gov.au>

This document is sold through AusInfo Government Info Bookshops at a price which covers the cost of printing and distribution only. For publication purchases please contact AusInfo on their toll-free number 132 447.

CONTENTS

Preface	vii
The Working Party	xi
Assessing the evidence	xiii
The consultation process	xv
The Dietary Guidelines for Australian Adults	xvii
BACKGROUND INFORMATION	
1 Enjoy a wide variety of nutritious foods	1
Terminology	1
Background	1
Scientific basis	8
Practical aspects of this guideline	10
Relationship to other guidelines	12
Conclusion	12
Evidence	13
References	13
1.1 Eat plenty of vegetables, legumes and fruits	17
Terminology	17
Background	18
Scientific basis	19
Practical aspects of this guideline	23
Relationship to other guidelines	24
Conclusion	25
Evidence	26
References	26
1.2 Eat plenty of cereals (including breads, rice, pasta and noodles), preferably wholegrain	31
Terminology	31
Background	32
Scientific basis	33
Practical aspects of this guideline	39
Relationship to other guidelines	41
Conclusion	41
Evidence	42
References	42

1.3	Include lean meat, fish, poultry and/or alternatives	51
	Terminology	51
	Background	52
	Scientific basis	56
	Practical aspects of this guideline	65
	Relationship to other guidelines	66
	Conclusion	67
	Evidence	67
	References	67
1.4	Include milks, yoghurts, cheeses and/or alternatives	75
	Terminology	75
	Background	76
	Scientific basis	79
	Practical aspects of this guideline	86
	Relationship to other guidelines	87
	Conclusion	88
	Evidence	88
	References	89
1.5	Drink plenty of water	95
	Background	95
	Scientific basis	96
	Practical aspects of this guideline	99
	Relationship to other guidelines	101
	Conclusion	102
	Evidence	102
	References	102
1.6	Limit saturated fat and moderate total fat intake	107
	Terminology	107
	Background	109
	Scientific basis	110
	Practical aspects of this guideline	122
	Relationship to other guidelines	123
	Conclusions	123
	Evidence	124
	References	124

1.7	Choose foods low in salt	133
	Terminology	133
	Background	134
	Scientific basis	136
	Practical aspects of this guideline	140
	Relationship to other guidelines	144
	Conclusion	144
	Evidence	145
	References	146
1.8	Limit your alcohol intake if you choose to drink	151
	Terminology	151
	Background	151
	Scientific basis	156
	Practical aspects of this guideline	165
	Relationship to other guidelines	166
	Conclusion	166
	Evidence	166
	References	167
1.9	Consume only moderate amounts of sugars and foods containing added sugars	171
	Terminology	171
	Background	172
	Scientific basis	178
	Practical aspects of this guideline	186
	Relationship to other guidelines	186
	Conclusion	187
	Evidence	187
	References	187
2	Prevent weight gain: be physically active and eat according to your energy needs	193
	Terminology	193
	Background	194
	Scientific basis	197
	Practical aspects of this guideline	202
	Relationship to other guidelines	204
	Conclusion	204
	Evidence	205
	References	206

3	Care for your food: prepare and store it safely	211
	Background	211
	Scientific basis	212
	Practical aspects of this guideline	216
	Relationship to other guidelines	223
	Conclusion	224
	Evidence	224
	References	225
4	Encourage and support breastfeeding	227
	Terminology	227
	Background	227
	Scientific basis	230
	Relationship to other guidelines	240
	Conclusion	240
	Evidence	240
	References	241

SPECIAL CONSIDERATIONS

A	The nutrition of Aboriginal and Torres Strait Islander peoples	249
	Current health and nutritional status	249
	Social determinants of Indigenous Australians' health	250
	Health aspects of traditional diets and lifestyles	252
	Traditional Aboriginal diet and food preferences	253
	Contemporary diet	253
	Contemporary use of traditional foods	256
	The National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan	257
	Dietary guidelines for Australia's Indigenous peoples	258
	References	259
B	Social status, nutrition and the cost of healthy eating	265
	Social status and nutrition	265
	The cost of healthy eating	268
	References	270
C	Dietary guidelines and the sustainability of food systems	271
	References	273

PREFACE

The Australian government has been providing nutrition advice for more than 75 years. In the past two decades the National Health and Medical Research Council has developed and disseminated guidelines providing dietary advice for Australians. This document is the third edition of the *Dietary Guidelines for Australian Adults*. The second edition was published in 1992. A new edition of the *Dietary Guidelines for Children and Adolescents in Australia, incorporating the Infant Feeding Guidelines for Health Workers* has also been produced, and the *Dietary Guidelines for Older Australians* was published in 1999. All these guidelines seek to promote the potential benefits of healthy eating, not only to reduce the risk of diet-related disease but also to improve the community's health and wellbeing.

The Australian Food and Nutrition Policy, endorsed in 1992, aims to improve the health of Australians and reduce the burden of preventable diet-related death, illness and disability through strategies that support the dietary guidelines. It is estimated that the current economic cost to the nation of the principal diet-related conditions—coronary heart disease, stroke and cancer—is about \$6 billion a year, so the potential economic benefit of an effective nutrition-based preventive strategy is enormous.

The Australian Food and Nutrition Policy is based on the principles of good nutrition, ecological sustainability and equity. This third edition of the *Dietary Guidelines for Australian Adults* is consistent with these principles. The food system must be economically viable and the quality and integrity of the environment must be maintained. In this context, among the important considerations are conservation of scarce resources such as topsoil, water and fossil fuel energy and problems such as salinity. Other important considerations have been noted in *Food for Health*, the Nutrition Taskforce's report to the New Zealand Ministry of Health. They include the change in consumer demand towards foods that are fresher and lower in fat and the recent restructuring of the food industry from a protected market to an open, competitive one. Although this has led to greater concentration of ownership, pricing strategy and policy development in the food sector, it has also given health policy makers greater access to the industry. In addition, globalisation is playing an increasing role in framing the management of the food supply.

The *Dietary Guidelines for Australian Adults* are aimed at healthy, independent adults. This document describes the scientific rationale for the guidelines and is intended for health professionals. Other documents will be produced in a format that is more suitable for consumers. The guidelines may also be useful for health professionals wanting to develop suitable diets for adults in other health

circumstances: it must always be remembered, however, that these guidelines are for healthy people and may not satisfy the specific nutritional requirements of people with particular diseases or conditions.

The Dietary Guidelines are an essential tool to support broader strategies to improve nutrition outcomes in Australia as outlined in *Eat Well Australia: An Agenda for Action in Public Health Nutrition* which was endorsed in 2001 by the Australian Health Ministers.

Compared with the two previous editions, this edition of the guidelines focuses more on food groups and lifestyle patterns, moving away from specific nutrients. In particular, the references to the *Australian Guide to Healthy Eating* will make it easier for consumers and nutrition educators to implement the guidelines. The *Australian Guide to Healthy Eating* is not the only food guide in use in Australia, and the Working Party recognises the potential for using other suitable guides to promote diets consistent with these guidelines.

The guidelines apply to the total diet: they should not be used to assess the 'healthiness' of individual food items, nor should individual guidelines be considered in isolation. The guidelines are not ranked in order of importance; they form a consistent and complete package when taken together. Each one deals with an issue that is key to optimal health.

Two of the guidelines relate to the quantity and quality of the food we eat—getting the right types of foods in the right amounts to meet the body's nutrient needs and to reduce the risk of chronic disease risk. The 'variety' guideline creates a positive setting for nutrition and reflects the fact that nutritious food can be one of the great pleasures of life. Sections 1.1 to 1.9 within this guideline detail the relationships between different food groups as part of the total diet. Given the epidemic of obesity we are currently experiencing in Australia, the other of these two guidelines specifically relates to the need to be active and to avoid overeating. Another guideline stresses the need to be vigilant about food safety and, in view of the increasing awareness of the importance of early nutrition, there is a further guideline that encourages everyone to support and promote breastfeeding.

Detailed information about requirements for specific nutrients in the Australian diet is provided in the NHMRC's *Recommended Dietary Intakes for Use in Australia*. The recommended dietary intakes and the dietary guidelines complement each other in providing comprehensive nutrition advice for the Australian community. Implementation of the dietary guidelines will result in significant health gains.

The revision process for this edition involved extensive consultation with the Australian community, the food industry and experts. The guidelines are based on the best evidence available, although the Working Party notes that in some cases the evidence for each guideline statement is not complete. In these instances the guidance is provided with the community's safety and health as the primary concern. The guidelines are a distillation of current knowledge about the relationship between diet and disease, the nutrients available in the Australian

food supply, and the contribution diet can make to optimising quality of life and reducing the levels of morbidity and mortality among Australians.

Each guideline is supported by background information prepared by members of the Working Party, with some additional assistance, as detailed in the next section.

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

Katrine Baghurst
Colin Binns

September 2002

THE WORKING PARTY

The Working Party developed the guidelines in accordance with National Health and Medical Research Council procedures and in keeping with the following terms of reference established by the NHMRC.

TERMS OF REFERENCE

- Undertake a review of the *Dietary Guidelines for Australians ...* and the *Dietary Guidelines for Children and Adolescents ...* and other related NHMRC dietary guidelines as identified.
- Undertake broad consultation to develop a suite of resources for both sets of guidelines, including:
 - comprehensive scientific background papers explaining the rationale for each guideline
 - appropriate consumer resources.
- Produce a dissemination and evaluation plan for both sets of guidelines.
- Report to the Health Advisory Committee.

MEMBERS OF THE WORKING PARTY

Dr Katrine Baghurst (Co-chair)
CSIRO Health Sciences and Nutrition

Prof. Colin Binns (Co-chair)
School of Public Health, Curtin University of Technology

Prof. A Stewart Truswell
Human Nutrition Unit, University of Sydney

Dr Amanda Lee
Public Health Services, Queensland Department of Health

Dr Peter Williams
School of Nutrition and Dietetics, University of Wollongong

Dr Ivor Dreosti
CSIRO Health Sciences and Nutrition

Assoc. Prof. Malcolm Riley
Nutrition & Dietetics Unit, Monash University

WORKING PARTY

Ms Isobel Brown
Government Relations Australia Ltd

Dr Merelie Hall
Royal Australian College of General Practitioners

Dr Geoff Davidson
Gastroenterology Unit, Women's and Children's Hospital, Adelaide

Ms Pat Crotty
Consumer representative

Ms Sue Jeffreson
Food Standards Australia New Zealand

Secretariat

Ms Karina Desarmia, Ms Lorraine O'Connor, Ms Tess Hill and
Ms Linda Robertson
National Health and Medical Research Council

Ms Jacinta Dugbaza, Ms Leticia White and Ms Michelle Coad
Commonwealth Department of Health and Ageing

OTHER CONTRIBUTORS

A number of the background papers were co-authored by experts, and the Working Party thanks them for their contribution:

Dr Trevor Beard
Menzies Centre for Population Health Research, University of Tasmania

Dr Tim Gill
International Taskforce on Obesity, Human Nutrition Unit, Sydney University

Ms Kirsti McVay, Ms Rochelle Finlay and Ms Patricia Blenman
Food Standards Australia New Zealand

Dr Mi Kyung Lee
School of Public Health, Curtin University of Technology

The Working Party expresses particular thanks to Ms Dympna Leonard (Tropical Public Health Unit, Queensland Health, Cairns) for assistance in preparing the paper on Aboriginal and Torres Strait Islander peoples.

Ms Leanne Lester (School of Public Health, Curtin University of Technology) and Ms Sally Record (CSIRO Health Sciences and Nutrition) helped with statistical analysis of the results of the 1995 National Nutrition Survey.

ASSESSING THE EVIDENCE

The National Health and Medical Research Council has released a guide called *How to Use the Evidence: assessment and application of scientific evidence* (2000). This guide relates, however, to evidence assessment in connection with clinical practice. In many cases evidence-based guidelines for clinical practice deal with evidence associated with a specific disease and a specific therapeutic agent. Similar criteria are not easily used for evidence assessment related to food and the maintenance of general community health and wellbeing, which is the primary focus of dietary guidelines.

A number of initiatives are under way around the world to try to develop an evidence-based approach to nutrition and public health, but this has generally been in response to the need for ‘proof’ in relation to health claims for food components. Food Standards Australia New Zealand (formerly the Australia New Zealand Food Authority) developed a set of proposed levels of evidence for food or health claims that is similar to, but somewhat broader in scope than, the NHMRC approach for clinical guidelines. Nevertheless, the FSANZ set is still primarily intended for assessing evidence of the efficacy of individual nutrients or food components in relation to a specific health outcome.

The Working Party considered, however, that it would still be useful to consider the NHMRC designation of levels of evidence for clinical practice in relation to the scientific data discussed in this document. These levels of evidence are outlined in the box.

NHMRC levels of evidence

- I Evidence obtained from a systematic review of all relevant randomised controlled trials.
- II Evidence obtained from at least one properly designed randomised controlled trial.
- III-1 Evidence obtained from well-designed pseudo-randomised controlled trials (alternate allocation or some other method).
- III-2 Evidence obtained from comparative studies (including systematic reviews of such studies) with concurrent controls and allocation not randomised, cohort studies, case-control studies, or interrupted time series with a control group.
- III-3 Evidence obtained from comparative studies with historical control, two or more single-arm studies, or interrupted time series without a parallel control group.
- IV Evidence obtained from case series, either post-test or pre-test/post-test.

Source: National Health and Medical Research Council. *A guide to the development, implementation and evaluation of clinical practice guidelines*. Canberra: NHMRC, 1999.

Six levels of evidence are designated by the NHMRC. Level I is based on a systematic review of all relevant randomised controlled trials and Level II is based on evidence obtained from at least one properly designed randomised controlled trial. There are very few Level I or Level II food-based nutrition trials, although some nutrient-supplement trials fall into these categories. Most food–health studies fall into Level III, the level of evidence that includes study designs such as cohort studies, case-control studies, and comparative ecological studies with historical controls.

Because of the nature of the dietary guidelines, the background papers were developed as a result of a process of comprehensive, rather than systematic, review of the literature. At the conclusion of each guideline, there is a summary of the NHMRC levels of evidence for the literature cited.

The NHMRC notes, ‘A decision should be made about what is feasible and appropriate in a given situation and the extent to which reasonable standards have been met by the available body of evidence’.

The evidence base for the background papers was developed using a variety of data bases and search terms. The literature was assessed using data bases and abstracting systems including the Cochrane Data Base for Randomised Control Trials; Medline, HealthStar, CINAHL using ‘systematic review’ filter, PubMed, Embase, Food & Technology Abstracts, Emerald, BioSis, Australasian Medical Index, Science Direct, Current Contents and searches of citations found in identified papers. Terms used in searches included food groupings such as fruits, vegetables, nuts and seeds, legumes, cereals, meat, poultry, fish, dairy, milk, yoghurt, cheeses, soy, water, alcohol, breastmilk (and breastfeeding) and dietary/food intake patterns as well as nutrients such as fats (total and types), carbohydrates sugars, starches, protein, iron, zinc, B12, calcium and salt as well as physical activity. These were investigated where relevant in relation to health outcomes such as overweight, obesity, growth, heart disease, cancers of various sorts, diabetes, bone density and osteoporosis, cognition and ageing. Whilst searches concentrated on human studies and those available in the English language, findings from some animal studies were included to provide evidence on possible mechanisms. The reviews were completed in January 2002 but some key papers published since then have been included.

THE CONSULTATION PROCESS

Development of the Dietary Guidelines for Australian Adults has involved consultation with the community and with experts working in the fields of public health and nutrition.

Preliminary work took place from December 2000 until May 2001 and involved the following:

- analysis of 104 completed and returned questionnaires dealing with the content and use of the second edition of the dietary guidelines
- establishment of an interactive website providing information about the review of the guidelines
- several meetings with stakeholders.

The public consultation process took place between July and August 2001, allowing about six weeks for consideration of the draft Dietary Guidelines for Australian Adults and preparation and lodgment of submissions. Notification was published in the *Commonwealth of Australia Gazette* and on the NHMRC website. Copies of draft documents and supporting information were available free of charge from the Office of the NHMRC and on the website. In addition, notices were placed in other publications and with media such as newspapers and radio and circulated to bodies expected to be interested.

The Dietary Guidelines for Australian Adults were submitted for consultation along with the Dietary Guidelines for Children and Adolescents in Australia, incorporating the Infant Feeding Guidelines for Health Workers. Ninety-three submissions were received. The Working Party met in September 2001 to consider the submissions; initial revisions were made by the end of December 2001 and were then reconsidered by the Working Party and revisions finalised by February 2002. Some additional key references were added during the technical editing and review period.

Additional specialist comment was obtained from Dr Peter Hartman (University of Western Australia), Dr Jane Scott (University of Glasgow), Dr Karen Cashell (University of Canberra), Ms Anne Croker (Australian Breastfeeding Association, formerly the Nursing Mothers Association of Australia), Ms Judy Seal (Strategic Inter-governmental Nutrition Alliance and Tasmania Health), Dr Wendy Oddy (NHMRC fellow, Curtin University of Technology) and Dr Gulnara Semonova (Director, Australian Breastfeeding Association Lactation Resource Centre).

The document was technically edited by Chris Pirie.

Food for health

Dietary Guidelines for
Children and Adolescents

Electronic and support browsing

Children and adolescents need sufficient nutritional foods to grow and develop normally.

- * Figures should be checked against the original data.

Enjoy a wide variety of nutritious foods

- [illegible]

Care for your child's blood pressure and stress it safely



Dietary Guidelines for Australian Adults

Enjoy a wide variety of nutritious foods

- Lack of regulation, efficiency and time
- Lack of security (e.g. possible hacks, data loss and corruption, possibly misleading)
- Possible bias: the actors' own environment
- Not well supported, often not
- Anonymous, therefore cannot be traced, abuse possible
- Only joining of some and lack of any
- Lack of security for and standard for the system
- Come from the right
- Some users might feel free to abuse it
- Consensus still, sometimes prevented by lack of
- Needs various, various aspects

Prevent weight gain: be physically active and eat according to your energy needs

Care for your food: prepare and store it safely

Encourage and support breastfeeding

THE DIETARY GUIDELINES FOR AUSTRALIAN ADULTS

Enjoy a wide variety of nutritious foods

- Eat plenty of vegetables, legumes and fruits
- Eat plenty of cereals (including breads, rice, pasta and noodles), preferably wholegrain
- Include lean meat, fish, poultry and/or alternatives
- Include milks, yoghurts, cheeses and/or alternatives. Reduced-fat varieties should be chosen, where possible
- Drink plenty of water.

and take care to

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

Prevent weight gain: be physically active and eat according to your energy needs

Care for your food: prepare and store it safely

Encourage and support breastfeeding

These guidelines are not in order of importance.

Each one deals with an issue that is key to optimal health.

Two relate to the quantity and quality of the food we eat—getting the right types of foods in the right amounts to meet the body's nutrient needs and to reduce the risk of chronic disease. Given the epidemic of obesity we are currently experiencing in Australia, one of these guidelines specifically relates to the need to be active and to avoid overeating.

Another guideline stresses the need to be vigilant about food safety, and, in view of the increasing awareness of the importance of early nutrition, there is a further guideline that encourages everyone to support and promote breastfeeding.

Background information

I ENJOY A WIDE VARIETY OF NUTRITIOUS FOODS

Colin Binns and Mi Kyung Lee

TERMINOLOGY

Food variety

Food variety can be defined on the basis of 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, as illustrated by the *Australian Guide to Healthy Eating* (shown in Figure 1.1), to attain all the required nutrients without excess energy intake.¹ *Variety* further refers to choosing a range of items from within each food group, particularly within the plant-based groups (vegetables, fruits and cereals). While variety is an important nutritional principle—and given the evolution of modern sedentary society—if it is to be maintained, a reduction in serving size needs to be considered, particularly for more energy dense foods with limited nutrient content (see the ‘Practical aspects of this guideline’ section).

Nutritious foods

The term *nutritious foods* is used to describe foods that make a substantial contribution towards providing a range of nutrients, have an appropriate nutrient density, and are compatible with the overall aims of the Dietary Guidelines for Australians.² The nutrients that are essential for human life are found in varying amounts in many different foods, and a varied diet is essential for obtaining sufficient quantities of all required nutrients (known and not yet known), for increasing the consumption of protective factors (phytochemicals), and for minimising exposure to toxicants.

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.

BACKGROUND

Australians today enjoy a wide variety of foods, relatively independent of season and location, and can choose from a number of cuisines. The available food supply is adequate to meet the nutritional needs of Australians, but appropriate

I. ENJOY A WIDE VARIETY OF NUTRITIOUS FOODS

choices must be made so that all nutrient requirements are met.³ There are also disadvantaged groups in Australia, for whom, because of factors such as poverty, particular food beliefs, distance or disability, special efforts are needed to ensure an adequate diet. Australia is also fortunate in having a food supply that is relatively free of contaminants and pollutants, as shown by the Australian Total Diet Survey.⁴

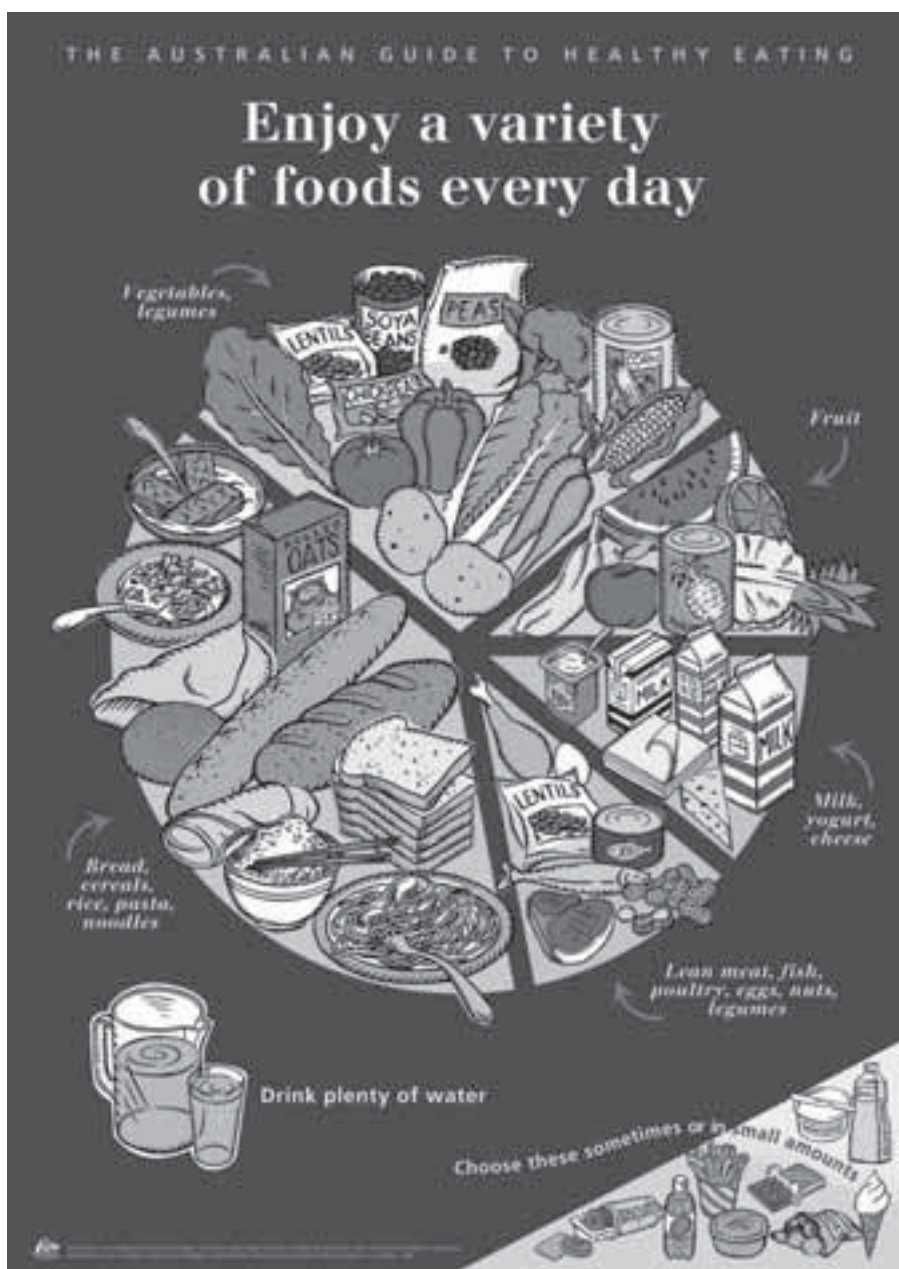


Figure 1.1 The Australian Guide to Healthy Eating¹

Sample serves suggested for adults

Food group	Cereals (including breads, rice pasta and noodles)	Vegetables and legumes	Fruit	Milk yoghurt, cheese	Lean meat, fish, poultry, nuts and legumes	Extra foods
Women						
19–60 yrs	4–9	5	2	2	1	0–2 °
	4–6	4–7	2–3	2–3	1–1 °	0–2 °
60+ yrs	4–7	5	2	2	1	0–2
	3–5	4–6	2–3	2–3	1–1 °	0–2
Pregnant	4–6	5–6	4	2	1 °	0–2 °
Breastfeeding	5–7	7	5	2	2	0–2 °
Men						
19–60 yrs	6–12	5	2	2	1	0–3
	5–7	6–8	3–4	2–4	1 °–2	0–3
60+ yrs	4–9	5	2	2	1	0–2 °
	4–6	4–7	2–3	2–3	1–1 °	0–2 °

Notes: The sample serves allow for two different eating patterns: the top row in each category includes a lot of cereals, bread, rice, pasta and noodles; the bottom row includes less of these products and more of the other groups.

Examples of sample sizes are:

- 2 slices (60g) bread, 1 medium bread roll, 1 cup cooked rice, pasta or noodles
- ° cup (75g) cooked vegetables or legumes, 1 cup salad vegetables, 1 small potato
- 1 medium piece (150g) of fruit, 1 cup diced pieces or canned fruit, ° cup fruit juice
- 1 cup (250ml) fresh milk, 2 slices (40g) cheese, 1 small carton (200g) yoghurt
- 65–100g cooked meat or chicken, 80–120g cooked fish fillet, 2 small eggs, ° cup cooked legumes, 1/3 cup nuts, ~ cup sesame seeds.

With the exception of breastmilk in the first six months of life, no single food can provide a complete and healthy diet. A diet containing a wide range of foods from the different food groups is most likely to offer protection against non-communicable chronic diseases such as vascular disease, obesity, diabetes, and possibly even cancer (see Sections 1.1 and 1.2). The benefits are gained by reducing the intake of foods that supply excessive amounts of fat, salt and alcohol and by maximising the intake of protective factors such as vegetables, fruits and cereals although recent work suggests that the situation with obesity is not straightforward.⁵ A varied diet also increases the possibility of receiving essential nutrients in adequate amounts.⁶

Variety in the diet is becoming increasingly important as the emphasis on non-nutrients increases. Foods have traditionally been classified according to their macro-nutrient and micro-nutrient value, but now their non-nutrient value is gaining recognition in terms of food's role in non-communicable chronic diseases and in ageing (see Section 1.1 and 1.2). Most non-nutrient factors are phytochemicals that are not directly associated with deficiency syndromes but do

have some relationship to optimal health. Phytochemicals can be multi-functional; alternatively, more than one class of phytochemicals can provide a particular function. Interactions between compounds are likely to be complex and deep, causing a masking or synergy of effects.⁷

Phytochemicals can fall into one of a number of chemical categories including carotenoids, flavonoids and isoflavonoids, polyphenols, isothiocyanates, indoles, sulphoraphane, monoterpenes, xanthin, and non-digestible oligosaccharides. Variety in the diet is recommended so that the protective benefits of nutrients and non-nutrients can be obtained: it is not known exactly which food constituents are responsible for the protective effect against chronic diseases.

Another benefit of variety may come from dilution of potentially toxic components in foods. Plants contain various toxic substances that, although often useful for discouraging insects and other predators, have the potential to harm humans. Minimising the risk posed by naturally occurring toxicants is a useful goal of public health policy.⁸ Historically, until fire was first used, only raw foods could have been eaten. Cooking must have immensely increased the safety and availability of these foods by destroying the thermolabile poisons in otherwise edible plants as well as the parasites and toxins common in flesh and carrion.⁹ Now a large number of processing and storage methods are used to reduce any toxicity problems foods might pose.

Consuming a wide variety of nutritious foods in appropriate amounts will thus increase dietary quality, improve chronic disease status and minimise the intake of toxic components.

Current Australian dietary practices

The National Nutrition Survey was conducted between February 1995 and March 1996, as an adjunct to the 1995 National Health Survey.¹⁰ The dietary intakes of approximately 13 800 people aged 2 years or more from urban and rural areas in all states and territories were recorded for one day by 24-hour recall. Additional information on physical measurements and eating habits and patterns was also collected. The resultant data showed that, during the 12 years since the previous survey, food variety in Australia had increased significantly, with a much greater number of foods being recorded in 1995 than in 1983. The increase in the variety of foods available reflects the wide range of fresh, processed, mixed and prepared food forms that are now conveniently obtainable in Australia on a daily basis.

This expansion in the number of foods available is largely a result of the cultural diversity that now characterises our population. The influx of European immigrants after World War 2 and the migration of Asian people in more recent decades have led to the development of an Australian population consuming a wide variety of cuisines, in place of the 'traditional' Anglo-Celtic foods.

Few countries have such ready access to such a variety of cuisines. On 30 June 1999 there were approximately 4.5 million overseas-born people living in Australia—about 24 per cent of the population. Twenty-seven per cent of overseas-born people were originally from the United Kingdom and Ireland; 5 per cent were from Italy; 3 per cent were from Greece; and 3 per cent were from Germany. The number of immigrants from Asia is increasing: the Vietnamese community is now second in number after the English.¹¹ The ready availability of different cuisines allows most Australians—at least those in urban areas and larger regional centres—to experiment with foods not common in the everyday diet, thus increasing the opportunity for expanding their food variety.

Some Australians choose diets that are mainly or exclusively vegetarian but the numbers are relatively small. In the National Nutrition Survey 4 per cent of subjects described themselves as vegetarian; the food-frequency questionnaire data recorded only 2 per cent as consuming no animal products, and a further 2 per cent restricting themselves to fish or white meat only. Ninety-six per cent of respondents reported consuming some red meat, with 79 per cent having red meat at least three times a week. However, in some selected groups, such as female university students in Perth, larger numbers reported eating only minimal amounts of meat. In the Perth study, 13 per cent classified themselves as vegetarian and 17 per cent as semi-vegetarian.¹²

The 1995 National Nutrition Survey data show that, generally, the diet of older Australians is more varied than that of younger groups. In the survey, the foods eaten were classified into 14 different groups. Figure 1.2 shows the cumulative percentage of people consuming various numbers of food groups on the day of the survey. This analysis showed that, in virtually all age groups, males who live alone eat significantly fewer food groups each day. For example, just over 40 per cent of males living alone ate five or fewer food groups on the day of the survey; this compares with only 10–12 per cent of the other three groups eating five or fewer food groups. No other demographic group showed such a divergence from the mean.

A comparison of the National Nutrition Surveys of 1983 and 1995 shows that there have been changes in the intakes of many nutrients, generally in the direction encouraged by the Dietary Guidelines for Australians (see Table 1.1). The most noteworthy change contrary to the dietary guidelines is the increasing prevalence of obesity; this is discussed in relation to Chapter 2.

Table 1.2 shows the average intakes recorded in the 1995 National Nutrition Survey for selected nutrients. Despite the variety of foods and cuisines in Australia, comparison with recommended dietary intakes shows that some nutrients are still at risk, among them iron in pre-menopausal women and calcium in women.

I. ENJOY A WIDE VARIETY OF NUTRITIOUS FOODS

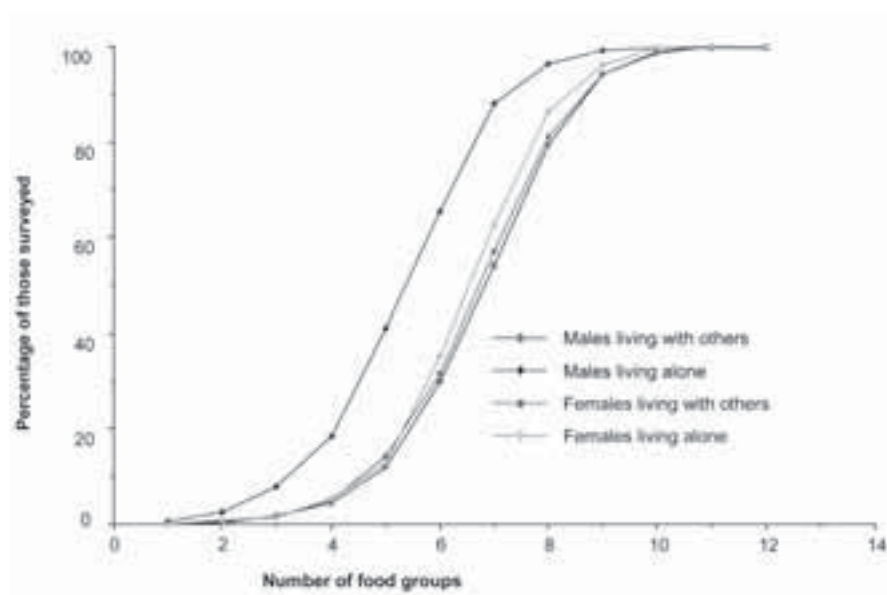


Figure 1.2 Food groups eaten on the day of the 1995 National Nutrition Survey

Table 1.1 Nutrient intakes in Australia, 1980s and 1995

Nutrient	Adults (25–64 yrs)		Adolescents (10–15 yrs)	
	Direction	Extent of change	Direction	Extent of change
Energy	Increased	Males 3%; females 4%	Increased	Boys 15%; girls 11%
Protein	Unchanged		Increased	Boys 14%; girls 13%
Carbohydrate	Increased	Males 17%; females 16%	Increased	Boys 22%; girls 18%
Fat	Decreased	Males 6%; females 4%	Unchanged	
Cholesterol	Decreased	Males 14%; females 22%	Unchanged	
Fibre	Increased	Males 13%; females 10%	Increased	Boys 13%; girls 8%
Calcium	Increased	Males 18%; females 14%	Unchanged	
Iron	Increased	Males 11%; females 15%	Increased	Boys 16%; girls 11%
Vitamin C	Decreased	Males 8%	Decreased	Girls 10%

Notes: Estimates based on 24-hour intake; capital cities only. Where there is a trend in mean intake direction, it is significant to the 1 per cent level.

Source: Unpublished results of the Australian Food and Nutrition Monitoring Unit (2001), based on analysis of comparable samples from the 1983 National Dietary Survey of Adults, the 1985 National Dietary Survey of School Children and the 1995 National Nutrition Survey.

Table 1.2 Mean nutrient intakes on the day of the 1995 National Nutrition Survey¹³

Nutrient	(RDI ^a)	Age group				
		19–24	25–44	45–64	65+	19+
Males						
Energy (Mj)		13.3	11.7	10.3	8.5	11.1
Protein (g)	(55)	128	115	105	84	109
Fat (g)		119	106	91	74	99
Carbohydrate (g)		376	317	274	235	301
Fibre (g)		26.2	26.1	26.3	24.0	25.9
Alcohol (g) ^b		15.2	19.7	20.2	14.7	18.5
Vitamin A (µg) ^c	(750)	1233	1306	1360	1301	1311
Thiamin (mg)	(1.1)	2.3	2.1	1.8	1.6	1.9
Niacin (equiv. mg)	(19)	57.6	53.9	48.8	38.8	50.7
Folate (µg)	(200)	322	311	310	277	307
Vitamin C (mg)	(40)	150	133	138	127	136
Calcium (mg)	(800)	1101	989	885	796	946
Phosphorus (mg)	(1000)	2052	1867	1692	1419	1776
Magnesium (mg)	(320)	390	393	383	334	381
Iron (mg)	(7)	17.9	16.7	16.2	14.4	16.4
Zinc (mg)	(12)	17.3	14.9	14.0	11.4	14.4
Potassium (mg)	(1950–5460)	3943	3818	3733	3232	3725
Females						
Energy (Mj)		8.4	7.9	7.2	64.	7.5
Protein (g)	(45)	78	76	75	64.3	74
Fat (g)		75	72	64	57	68
Carbohydrate (g)		243	220	200	182	211
Fibre (g)		19.2	20.0	21.5	20.2	20.3
Alcohol (g) ^b		6.6	8.2	8.0	4.6	7.3
Vitamin A (µg) ^c	(750)	889	1024	1145	1059	1047
Thiamin (mg)	(0.8)	1.5	1.4	1.3	1.2	1.4
Niacin (equiv. mg)	(13)	36.1	35.3	34.5	29.4	34.1
Folate (µg)	(200)	233	227	247	225	233
Vitamin C (mg)	(30)	120	109	118	112	113
Calcium (mg)	(800)	750	762	769	686	749
Phosphorus (mg)	(1000)	1332	1300	1295	1132	1272
Magnesium (mg)	(270)	273	284	297	268	283
Iron (mg)	(12–16)	11.9	12.0	12.3	11.3	11.9
Zinc (mg)	(12)	10.2	9.9	9.8	9.0	9.7
Potassium (mg)	(1950–5460)	2752	2816	2930	2626	2805

- a. The recommended dietary intakes are for males aged 19–64 years and females aged 19–54 years. The RDIs are soon to be reviewed.
- b. Represents pure alcohol.
- c. Retinol equivalents.

SCIENTIFIC BASIS

Dietary variety and chronic disease

There is evidence about the relationship between dietary variety and chronic disease from two prospective cohort studies and two ecological studies. Research that illustrates the health benefits of increasing food variety in the diet comes from the US National Health and Nutrition Examination Survey Epidemiologic Follow-up Study, which studied 4 160 men and 6 264 women. A dietary diversity score of 0 to 5 was used, with 5 being the maximum possible score and indicating high food variety. An increased risk of mortality was associated with a low dietary diversity score at nearly every level of age, income, education, race, smoking status and fibre intake. There was also an increased risk of mortality from all causes for both men and women where a food group was omitted from the diet. Fewer than 5 per cent of study participants reported omitting foods from the meat or grain groups on the survey day, whereas 46 per cent reported no fruit, 25 per cent reported no dairy products, and 17 per cent reported no vegetables. Reporting no consumption of fruits and vegetables was associated with low serum vitamin C, whereas reporting fruit and vegetable consumption was associated with a high vitamin C concentration.¹⁴

In a prospective US study, Kant et al.¹⁵ evaluated the association between dietary quality (based on dietary guidelines) and mortality in women. A food-frequency questionnaire was completed by 42 254 women (mean age 61.1 years) who were followed up for an average of 5.6 years. The results showed that women who reported dietary patterns that included fruits, vegetables, whole grains, low-fat dairy products and lean meats, as recommended by the dietary guidelines, had a lower risk of mortality. The data suggested that a dietary pattern characterised by consumption of foods recommended in current dietary guidelines is associated with decreased risk of mortality in women.

Ecological comparisons of cuisines and health outcomes have suggested that 'Mediterranean' diets and the Japanese diet (in particular the Okinawan diet) and may show benefits in terms of decreased mortality from chronic diseases and increased life expectancy in countries where these diets predominate.¹⁶ A study of Greek populations in Melbourne and Greece concluded that food variety was an important determinant of morbidity and mortality.¹⁷ Traditional Mediterranean diets are based on plant foods, contain small amounts of animal products, use olive oil as the principal fat, contain moderate amounts of alcohol, and balance energy intake with energy expenditure. They are low in saturated fat and high in the protective compounds found throughout a variety of plant foods.^{18,19} The Okinawan diet is varied, with a substantial amount of fish, and is associated with the longest reported life spans of any population.²⁰

Dietary variety and nutrient intake

Apart from consideration of specific chronic disease health outcomes, one Melbourne study of Chinese migrants⁵ has shown that variety in food choice is more likely to result in diets with an acceptable nutrient profile. In this study,

when the diet failed to provide more than 40 per cent of the maximum achievable variety (over 12 months), participants were far more likely to have at least one nutrient level fall below two-thirds of the Australian recommended dietary intakes. It is unclear how this would relate to the general Australian population. Interestingly, food variety in the diet of these migrants increased with length of stay in Australia, independent of age, which suggests that food variety can be increased if there is continued exposure to new foods.⁵

Nutrient interactions

Maximising nutrient bioavailability is another potential benefit of variety. There are many complex relationships between foods and nutrients, and they can mutually influence the absorption, metabolism and retention of other nutrients. When a diet is well balanced and nutrients are in adequate supply, such interactions pose few problems; when the intake of some nutrients is habitually low, excesses of others can have detrimental effects.²¹ The following are examples.

- *Interactions between sodium, protein and calcium.* Sodium and calcium compete for the same transport mechanism in the kidney, and an excess of one will cause excretion of the other. Protein has a similar effect on urine calcium levels. This interaction is important in the older population because factors that affect urinary calcium loss are likely to affect bone health: recognition of the interaction allows for the prevention of calcium losses related to high protein intakes. When diets are high in protein, a reduction in sodium intake can reduce the physiological need for calcium²² and so improve calcium nutrition (see Section 1.7). Other inhibitors of calcium absorption are phosphates, phytic acid from the husks of cereals, and oxalic acid in spinach and rhubarb, which form insoluble complexes with calcium.²³
- *The effects of various nutrients on iron absorption.* Vitamin C-containing foods (such as citrus fruit) and meats have a positive effect on the absorption of iron from plant foods when eaten at the same meal.²⁴ Consuming iron from animal sources (such as meat, which is also high in protein) will also promote iron absorption.²⁵ In contrast, non-haem iron absorption is inhibited by phytates, polyphenols (for example, tannins) and calcium.²⁶ This is discussed further in Section 1.3.
- *Zinc bioavailability.* Zinc found in animal products, crustaceans and molluscs is more readily absorbed than zinc found in plant foods. In contrast, legumes and unrefined cereals contain phytates that reduce zinc absorption. The zinc content of refined cereals is lower than that of unrefined cereals but, because a large part of the phytic acid present in cereals is removed during the refining process, zinc bioavailability is increased. Phytate in the presence of calcium may also reduce zinc bioavailability.²⁷ By including adequate amounts of wholegrain products and legumes in a varied diet, lacto-ovo-vegetarians can meet their zinc requirements and maintain zinc balance.²⁸ Eating a varied diet usually protects against these interaction effects.

I. ENJOY A WIDE VARIETY OF NUTRITIOUS FOODS

It must also be remembered that, in storing, preparing, cooking and processing foods, losses of some nutrients occur, while in other cases absorption or availability is improved by processing. For example, riboflavin is destroyed by exposure to ultraviolet light; ascorbic acid is destroyed by exposure to oxygen (air); beta-carotene, thiamin and ascorbic acid are destroyed by heating; and minerals are leached out of foods soaked in water.

In some cases the chemical form of a nutrient used for fortification may be less available than the natural product (for example, iron added to breakfast cereal compared with haem iron); in other cases it may be more bioavailable (for example, folic acid compared with naturally occurring folates).

The balance between different nutrients also needs to be considered. There is emerging evidence that the balance between n-3 and n-6 fatty acids might be important (see Section 1.6).^{29,30}

Special groups

People following a strict vegetarian diet need to be careful to include a variety of protein sources to get the right mix of amino acids. Iron, zinc, and vitamin B₁₂^{31–33} may also pose some problems. Plant foods can provide some iron and zinc but these sources have lower bioavailability. Vitamin B₁₂ is found only in animal products and, in strict vegetarians, may need to be sourced from enriched foods or supplements. Suitable diets would normally include a higher proportion of legumes and nuts to provide additional nutrients, including iron and protein.

PRACTICAL ASPECTS OF THIS GUIDELINE

The balance between variety and over-consumption

Energy

As variety in a diet increases, it is important to reduce serving sizes and the amounts of each food eaten to avoid over-consumption of energy (and thus avoid obesity). There have been a number of short term studies in humans showing links between overconsumption and variety^{34–39} and McCrory et al.⁴⁰ have reported on a long-term human study to determine whether dietary variety within food groups is associated with energy intake and body fatness. Dietary energy from the individual food groups examined was positively related to variety within that group. However, whilst high variety of more energy-dense food groups such as sweets, snacks, condiments, entrees and carbohydrate-based foods was associated with body fatness, variety in vegetable consumption was inversely associated. The authors suggested that the wide range of energy dense foods available might be partly responsible for increasing body fatness in the community. Thus high variety in vegetable consumption does not appear to have detrimental effects on body weight but portion size must be taken into account when the variety principle is applied to more energy dense food groups.

Other nutrients

While eating a wide variety of foods will maximise the potential benefits of the biological diversity of foods (particularly plant foods), nutrition is complex and over-consumption has the potential to be as big a problem as deficiency. Excessive intakes of individual essential nutrients usually cause only minor problems, but in some rare cases can be fatal. Major problems are nearly always associated with excessive intakes in the form of supplements, although it is certainly possible to develop symptoms of toxicity with very unbalanced diets. This applies even to different chemical forms of the ‘same’ nutrient. Table 1.3 provides some examples.

A diet limited in the range of foods consumed and with excessive, long-term consumption of a particular food can cause problems however this is rare unless accompanied by supplementation. Examples are high and prolonged consumption of carrot juice, which will result in excess beta-carotene intake, or eating very large portions of liver, which may cause vitamin A poisoning. It is impossible to consume nutrients to the excessive levels necessary for these effects if a person’s diet is varied, nutritious and healthy, consistent with the *Australian Guide to Healthy Eating*.

Table 1.3 Potential outcomes of excessive vitamin intake^{41–47}

Nutrient excess	Potential outcome
Provitamin A	Yellow or orange skin colour
Preformed vitamin A	Headache, vomiting, extensive skin peeling, bone abnormalities and liver damage ⁴¹
Vitamin A supplements in pregnancy	Serious birth defects
Niacin as nicotinic acid	Flushing, hyperglycaemia and abnormalities of liver function ⁴²
Vitamin C	Nausea, vomiting and diarrhoea (cited in reference ⁴³)
Vitamin C in pregnancy	Rebound scurvy, due to vitamin C deficiency, in the newborn infant (cited in reference ⁴³)
Pyridoxine (vitamin B ₆)	Peripheral neuropathy ^{44,45}
Vitamin D	Hypercalcaemia, dehydration and calcification of soft tissue, including kidney failure ⁴⁶
Iron	Acute excessive intake can result in vomiting and gastrointestinal bleeding ⁴⁷ ; chronic excessive consumption can lead to haemosiderosis with liver damage

Food contaminants

Eating a variety of foods dilutes the naturally occurring toxicants and any added contaminants. The Australian food supply is one of the safest and cleanest in the world, ensuring that a minimum of toxicants are ingested. The 19th Australian

I. ENJOY A WIDE VARIETY OF NUTRITIOUS FOODS

Total Diet Survey found that levels of pesticide residues and heavy metal contaminants in the Australian food supply remain very low and well within safety standards set by Australian and international health authorities.⁴

RELATIONSHIP TO OTHER GUIDELINES

Eat plenty of vegetables, legumes and fruits

Including a variety of vegetables, legumes and fruits in the diet will ensure the intake of a wide range of vitamins, minerals, dietary fibres and beneficial, non-nutrient phytochemicals found in plant foods. These occur in the various vegetables, legumes and fruits to varying degrees. The only way to ensure that all the beneficial components are eaten is to include a wide variety of vegetables, legumes and fruits. Vegetables should include green leafy varieties, red and yellow and starchy vegetables. Fruits should include those high in vitamin C and those high in vitamin A (and its analogues).

Eat plenty of cereals (including breads, rice, pasta and noodles), preferably wholegrain

As with vegetables, legumes and fruits, different cereal grains can contribute a variety of nutrient and non-nutrient benefits. A wide range of cereal-based products is advisable and could include those from different cuisines; such as wholegrain or wholemeal bagels, pita bread and pumpernickel. Low-fat, low-salt and low-sugar products are preferable where possible.

Prevent weight gain: be physically active and eat according to your energy needs

The work of McCrory and Coulston⁴⁸ indicates that a varied diet can result in higher energy intake if care is not taken with portion size. While variety is an important nutritional principle—particularly given the evolution of modern sedentary society—if variety is to be maintained, activity must be encouraged and food serving sizes, especially of the energy-dense foods, may need to be reduced. Consumer and food service education⁴⁹ that focuses on reduced portion sizes may help reduce opportunities for overeating, especially of high-energy density foods.

CONCLUSION

Australians are in a position to include in their diet a range of cuisines that add variety to the ‘traditional’ Australian diet and have been associated with health gains. Recommending to Australians that they ‘enjoy a wide variety of nutritious foods’ will not only help ensure appropriate intakes of major dietary components such as protein, carbohydrates and fats but also help ensure adequate and

appropriate intakes of vitamins and minerals, individual fatty acids and amino acids. Mennell et al.⁵⁰ refer to the varying cuisines of the world as ‘culinary culture’ and define this as ‘the ensemble of attitudes and tastes people bring to cooking and eating’. Enjoying a variety of nutritious foods remains an important message for all age groups. Experimenting with other cuisines, and incorporating new and traditional foods will encourage variety in the diet, help meet nutrient requirements, and provide some protection against non-communicable chronic diseases. Serving sizes of more energy dense foods may need to be reduced to accommodate variety.

EVIDENCE

Evidence of the importance of variety to gain sufficient nutrients is available at Level III (references 14, 15, 18 and 40) and at Level IV (references 5 and 6). Evidence also comes from cross-cultural observational studies of diet, health and longevity.

REFERENCES

1. Department of Health and Family Services. The Australian guide to healthy eating: background information for nutrition educators. Canberra: DHFS, 1998.
2. National Health and Medical Research Council. Dietary guidelines for Australians. Canberra: Australian Government Publishing Service, 1992.
3. Australian Bureau of Statistics. *Apparent consumption of foodstuffs and nutrients, Australia, 1998*. Canberra: ABS, 2000.
4. Australia New Zealand Food Authority. *19th Australian Total Diet Survey*. Canberra: ANZFA, 2001.
5. Hsu-Hage B, Wahlqvist M. Food variety of adult Melbourne Chinese: a case study of population in transition. *World Rev Nutr Diet* 1996;79:53–69.
6. Hodgson J, Hsu-Hage B, Wahlqvist M. Food variety as a quantitative descriptor of food intake. *Ecol Food Nutr* 1994;32:137–48.
7. Wahlqvist M, Wattanapenpaiboon N, Kannar D, Dalais F, Kouris-Blazos A. Phytochemical deficiency disorders. *Curr Ther* 1998;July:53–60.
8. Park D. Surveillance programmes for managing risks from naturally occurring toxicants. *Food Addit Contam* 1998;12(3):361–71.
9. Cuthbertson WFJ. Evolution of infant nutrition. *Brit J Nutr* 1999;81:359–71.
10. Australian Bureau of Statistics. *National Nutrition Survey: users’ guide*. Cat. no. 4801.0. Canberra: ABS, 1998.
11. Australian Bureau of Statistics. *Migration*. Canberra: ABS, 2000.

I. ENJOY A WIDE VARIETY OF NUTRITIOUS FOODS

12. Rangan A, Aitkin I, Blight G, Binns C. Factors affecting iron status in 15–30 year old female students. *Asia Pacific J Clin Nutr* 1997;6:291–5.
13. Australian Bureau of Statistics. *National Nutrition Survey: nutrient intakes and physical measurements*. Cat. no. 4805.0. Canberra: ABS, 1995.
14. Kant A, Schatzkin A, Harris T, Ziegler R, Block G. Dietary diversity and subsequent mortality in the first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study. *Am J Clin Nutr* 1993;57:434–40.
15. Kant AK, Schatzkin A, Graubard BI, Schairer C. A prospective study of diet quality and mortality in women. *JAMA* 2000;283(6):2109–15.
16. Yamori Y, Miura A, Taira K. Implications from and for food cultures for cardiovascular diseases: Japanese food, particularly Okinawan diets. *Asia Pacific J Clin Nutr* 2001;10(2):144–5.
17. Wahlqvist ML, Kouris-Blazos A, Hsa-Hage BH. Aging, food, culture and health. *Southeast Asian J Trop Med Pub Hlth* 1997;28(suppl. 2):100–12.
18. de Groot LC, van Staveren WA, Burema J. Survival beyond age 70 in relation to diet. *Nutr Rev* 1996;54:211–12.
19. Trichopoulou A, Kouris-Blazos A, Vassilakou T, Gnardellis C, Polychronopoulos E, Venizelos M et al. Diet and overall survival of elderly Greeks: a link to the past. *Am J Clin Nutr* 1995;61:1346S–1350S.
20. Wahlqvist ML. Nutrition and diabetes in the Asia–Pacific region with reference to cardiovascular disease. *Asia Pacific J Clin Nutr* 2001;10:90–6.
21. Heaney R. Nutrient interactions and the calcium requirement. *J Lab Clin Med* 1994;124:15–16.
22. Heaney R. Protein intake and the calcium economy. *J Am Diet Assoc* 1993;93:1259–60.
23. Wahlqvist M ed. *Food and nutrition: Australasia, Asia and the Pacific*. Sydney: Allen & Unwin, 1997.
24. Gerster H. High-dose vitamin C: a risk for persons with high iron stores? *Internat J Vit Nutr Res* 1999;69(2):67–82.
25. Cobiac L, Baghurst K. Iron status and dietary iron intakes of Australians. *Food Aust* 1993;April:S1–S24.
26. Mendoza C, Viteri FE, Lonnerdal B, Raboy V, Young KA, Brown KH. Absorption of iron from unmodified maize and genetically altered low-phytate fortified with ferrous sulfate or sodium iron EDTA. *Am J Clin Nutr* 2001;73:80–5.
27. Horwath C. Dietary intake studies in elderly people. *World Rev Nutr Diet* 1989;59:1–70.

28. Hunt JR, Matthys LA, Johnson LK. Zinc absorption, mineral balance, and blood lipids in women consuming controlled lactoovo-vegetarian and omnivorous diets for 8 weeks. *Am J Clin Nutr* 1998;67:421–30.
29. National Health and Medical Research Council. *The role of polyunsaturated fats in the Australian diet*. Canberra: National Health and Medical Research Council, 1992.
30. James M, Gibson R, Cleland L. Dietary polyunsaturated acids and inflammatory mediator production. *Am J Clin Nutr* 2000;71:343S–348S.
31. Haddad E, Sabate J, Whitten C. Vegetarian food guide pyramid: a conceptual framework. *Am J Clin Nutr* 1999;70:615S–619S.
32. Ball M, Bartlett M. Dietary intake and iron status of Australian vegetarian women. *Am J Clin Nutr* 1999;70:353–8.
33. Messina V, Burke K. Vegetarian diets—position of the American Dietetics Association. *J Am Diet Assoc* 1997;97:1317–21.
34. Pliner P, Polivy J, Herman CP, Zakaluzn I. Short term intake of overweight individuals and normal weight dieters and non-dieters with and without choice among a variety of foods. *Appetite* 1980; 1: 203–13.
35. Bellisle F, Le Magnen J. The structure of meals in humans: eating and drinking patterns in lean and obese subjects *Physiol Behav* 1981; 27:649–58.
36. Rolls BJ, Rowe EA, Rolls ET, Kingston B, Megson , Gunary R. Variety in a meal enhances food intake in man *Physiol Behav* 1981; 26: 215–21.
37. Rolls, BJ, Rolls ET, Rowe EA, Sweeney K. Sensory specific satiety in man. *Physiol Behav* 1981; 27 :137–42.
38. Rolls BJ, Rowe EA, Rolls ET. How sensory properties of foods affect human feeding behaviour *Physiol Behav* 1982; 29: 409–17.
39. Spiegel TA, Stellar E. Effects of variety on food intake of underweight, normal weight and overweight women. *Appetite* 1990; 15: 47–61.
40. McCrory MA, Fuss PJ, McCallum JE, Yao M, Vinken AG, Hays NP et al. Dietary variety within food groups: association with energy intake and body fatness in men and women. *Am J Clin Nutr* 1999;69:440–7.
41. Russell RM. The vitamin A spectrum: from deficiency to toxicity. *Am J Clin Nutr* 2000;71:878–84.
42. Dreosti IE. Niacin. *J Food Nutr* 1984;41:126–34.
43. Ausman LM, Mayer J. Criteria and recommendations for vitamin C intake. *Nutr Rev* 1999;57(7):222–9.
44. Rutishauser IHE. Vitamin B-6. *J Food Nutr* 1982;39:158–67.

I. ENJOY A WIDE VARIETY OF NUTRITIOUS FOODS

45. Rutishauser IHE. Vitamin B-6—update. In: Truswell A, ed. *Recommended nutrient intakes—Australian papers*. Sydney: Australian Professional Publications, 1990.
46. Fraser DR. Vitamin D. *J Food Nutr* 1987;44:3–8.
47. Roeser HP. Iron. *J Food Nutr* 1985;42:82–92.
48. McCrory MA, Coulston AM. Limitations on the adage ‘eat a variety of foods’? *Am J Clin Nutr* 1999;69(3):350–1.
49. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. *Science* 1998;280:1371–4.
50. Mennell S, Murcott A, van Otterloo A. *The sociology of food: eating, diet and culture*. London: Sage, 1992.

I.1 EAT PLENTY OF VEGETABLES, LEGUMES AND FRUITS

Ivor Dreosti

TERMINOLOGY

Vegetables

Vegetables includes all leafy green vegetables (for example, spinach, lettuce, silver beet and bok choy), members of the crucifer family (for example, broccoli, cabbages and brussels sprouts), all root and tuber vegetables (for example, carrots, yams and potatoes), edible plant stems (for example, celery and asparagus), gourd vegetables (for example, pumpkin and cucumber), allium vegetables (for example onion, garlic and shallot) and corn, although this last food is usually regarded as a cereal. Some vegetables are eaten raw; others are best cooked because this makes them more palatable and digestible.

Fruits

The term *fruit* generally applies to the sweet, fleshy edible portion of a plant that arises from the base of the flower and surrounds the seeds; apples, oranges, plums, berries, tomatoes and avocados are examples. Most fruit is eaten raw, although in some cases cooking can offer a tasty alternative.

Legumes

Legumes refers also to pulses and includes all forms of prepared beans and peas—dried, canned and cooked legumes, bean curd, tofu, and legume-flour products such as pappadams. Among the well-known edible legumes are butter beans, haricot (navy) beans, red kidney beans, soybeans, mung beans, lentils, chick peas, snow peas and various other fresh green peas and beans. Legumes are generally cooked: this improves their nutritional value and reduces the risk of toxicity that occurs with some legumes because of the presence of heat-labile toxins. Occasionally, however, they can be eaten raw; snow peas are an example. Strictly speaking, legumes are specialised forms of fruit since the pod surrounds the seeds and arises from the base of the flower, as occurs with fruit. But, because the main food material in legumes is the seeds, they are generally placed in a separate category.

BACKGROUND

Each year in Australia about 40 per cent of all deaths can be attributed to diseases of the circulatory system and 27 per cent to cancer, accounting for annual health care costs of around \$4 billion and \$2 billion respectively.¹

Scientific surveys of populations around the world have consistently provided good epidemiological evidence that people who regularly eat diets high in fruits and vegetables and legumes have substantially lower risks of coronary heart disease^{2–4}, stroke^{2,5}, several major cancers^{6,7} and possibly hypertension^{8,9}, type 2 diabetes mellitus^{10,11}, cataracts^{12,13}, and macular degeneration of the eye.^{14,15} A large number of experimental studies with model systems have afforded further evidence of a protective effect of fruits and vegetables against these non-communicable chronic diseases and offer some clues about the actual substances in these foods that may be protective as well as the mechanisms by which they may act. Accordingly, a new term, *phytochemicals*, has been added to the vocabulary of nutritionists; it refers to the many different substances occurring in plant foods in small amounts (in addition to the well-established nutrients) and which appear to contribute significantly to reducing the risk of non-communicable chronic diseases.

The *Australian Guide to Healthy Eating* recognises the importance of fruits and vegetables in a healthy diet and recommends consumption of two to four servings of fruit and four to eight servings of vegetables each day for adults¹⁶, which is generally in line with the minimum five servings of vegetables and two of fruit established by the core food group analysis endorsed by the National Health and Medical Research Council.¹⁷ It should, however, be noted that average current vegetable and fruit consumption in Australia falls significantly short of this recommendation, as Table 1.1.1 shows.

Table 1.1.1 Mean intakes of fruits and vegetables in Australian adults in relation to the NHMRC core food group recommendations¹⁸

Age group	Fruit					Vegetables				
	Mean intake					Mean intake				
	(% recommended)					(% recommended)				
	Including juice		Excluding juice		Recommended (g/day)	Including potato/ legumes		Without potato/ legumes		Recommended (g/day)
	M	F	M	F		M	F	M	F	
16–18	49	65	26	29	300–450	98	66	42	29	300
19–24	65	62	31	31	300	86	68	46	42	300–375
25–44	67	67	42	44	300	87	69	52	45	300–375
45–64	76	74	53	57	300	95	79	60	52	300–375
65+	74	75	60	59	300	88	75	54	49	300–375

Notes: Mean intake data are from the 1995 National Nutrition Survey. One serve of fruit equals 150g; one serve of vegetables equals 75g; where recommendations are a range, the mid-point has been used for calculations. The World Cancer Research Fund recommends 400–800g per day of fruits and vegetables combined, but excluding potatoes and legumes, for adults.

SCIENTIFIC BASIS

Original and recent studies

Cardiovascular disease

In 1997, 28 studies in humans of fruit and vegetable consumption and the risk of cardiovascular disease were reviewed, and good evidence was found of a protective effect associated with higher intakes of plant foods.² Some years earlier, in 1993, the US Food and Drug Administration allowed a health claim to the effect that diets low in saturated fat and cholesterol and rich in fruits, vegetables and grain products containing fibre, particularly soluble fibre, may reduce the risk of coronary heart disease¹⁹, although a similar claim was not allowed by the Canadian food authority. A subsequent large study in females also reported a significant inverse association between fruit and vegetable intake and cardiovascular disease.³

Recent experimental studies suggest that protection against heart disease may arise in several ways, including through the presence of antioxidant phytochemicals (for example, bioflavonoids and carotenoids) and antioxidant vitamins (for example, vitamins E and C) at significant levels in fruits and vegetables, which may reduce the risk of cholesterol becoming oxidised in coronary blood vessels and deposited to form atheromatous plaques.²⁰ Importantly, a review of the effect of beta-carotene on coronary heart disease in several observational and intervention studies suggests protection only in the observational studies, highlighting the possibility that the benefit reported in some studies may be related to foods rich in beta-carotene and other antioxidants and micro-nutrients—or indeed other confounding factors—rather than to the beta-carotene alone.²¹ Also important is the apparent capacity of vegetable protein to reduce blood cholesterol levels in people habitually consuming an omnivorous diet.²²

Particular emphasis is being focused at present on the importance of the vitamin folate in reducing blood levels of the compound homocysteine, which is a possible risk factor for coronary heart disease.^{4,23} Especially noteworthy is the fact that a major source of dietary folate is green, leafy vegetables, and studies suggest that many adults have folate intakes well below the level needed to minimise the risk associated with raised levels of homocysteine.²⁴

Stroke

A systematic review of 14 studies including ecological, case-control and cohort studies dealing with stroke and fruit and vegetables found strong evidence of a protective effect associated with higher intakes of plant foods.² The mechanism for this apparent protection is not clear, but it appears to exist for strokes of both haemorrhagic and ischaemic origin.⁵ In one large study extending over eight years, protection was associated with vegetable intake rather than fruit²⁵, although generally both types of plant food are considered to be likely protective agents.²⁶

Hypertension

Because plant foods contribute significantly to the intake of potassium and magnesium—both of which have been proposed to be associated with a lower blood pressure—diets high in fruits and vegetables will increase the daily intake of both minerals and may help prevent or control hypertension.^{6,27} In a study with women in the United States, lowered blood pressure was found to be associated with higher intakes of fruits and vegetables, fibre and magnesium⁸; more recently, data from the Dietary Approaches to Stop Hypertension (DASH) randomised clinical trial have indicated that diets rich in fruits and vegetables, with or without low-fat dairy products, significantly reduced ambulatory blood pressure after an eight-week intervention period²⁸, especially in African Americans and people with hypertension.²⁹ Similar results were found with US adolescents who had elevated blood pressures: blood pressure was lower in those subjects with higher intakes of a combination of nutrients including potassium, calcium, magnesium and vitamins, as provided by diets rich in fruits and vegetables and low-fat dairy products.³⁰

Cancer

Health researchers have estimated that at least 30 per cent of many major cancers have a strong dietary link and that the link may be even stronger for some cancers.⁶ Among the dietary factors underlying this association are substances that may aggravate the development of cancer and, very importantly, substances that reduce cancer risk. Dietary components in the latter group include fibre, fruits and especially vegetables. In fact, the association between fruits and vegetables is sufficiently widely recognised that the US Food and Drug Administration has allowed a health claim to the effect that diets low in fat and rich in fruit and vegetables may reduce the risk of some cancers.¹⁹

Not surprisingly, the protective effect of fruit and vegetables has been noted especially in relation to the oral cavity, oesophagus, stomach and large bowel, where local contact may be a factor. Significant risk reduction has also been observed for cancers of the lung and possibly the breast, endometrium and pancreas.^{6,7} Many factors in fruit and vegetables have been proposed to account for the foods' protective effect and many potential mechanisms suggested. Much emphasis is currently placed on the many novel phytochemicals found in plant foods (for example, carotenoids, bioflavonoids, isothiocyanates and indole carbinols) and on several established vitamins and minerals (for example, vitamins C and E, folate, selenium and calcium). Proposed mechanisms range from reduced formation of cancer-promoting substances in the gastrointestinal tract (through antioxidant activity), to the part played by phytochemicals and micro-nutrients in detoxification of carcinogenic substances, and to 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.^{6,7,31}

In the 1997 World Cancer Research Fund and American Institute for Cancer Research (WCRF–AICR) global review of nutrition and cancer prevention⁷,

prevention by fruit and vegetables was rated to be ‘convincing’ for cancers of the mouth, pharynx, oesophagus, stomach, colon, rectum and lung; ‘probable’ for the larynx, pancreas, breast and bladder; and ‘possible’ for the ovaries, cervix, endometrium, thyroid, liver, prostate and kidney. Since that report, data generally confirming these findings have become available from a number of further case-control and cohort studies. In particular, lower risks of cancer have again been found for the oral cavity^{32,33}, stomach³⁴ and colon and rectum³⁵ in relation to higher vegetable and fruit intake, although a recent study found no evidence that one extra serving of fruit and vegetables provides any measurable additional protection.³⁶

Two recent studies on lung cancer also consistently indicate that a high intake of fruit and vegetables is protective, particularly with respect to brassicae vegetables, tomatoes, lettuce and cabbage.^{37,38} Further suggestive evidence of protection by fruits and vegetables has been noted for cancer of the bladder^{39,40}, breast^{41,42} and, to a lesser extent, prostate, notably in relation to the carotenoid lycopene.^{43,44}

It should be noted, however, that although considerable emphasis has been placed on the WCRF–AICR review, attention should be paid to the study by the UK Department of Health Committee on the Medical Aspects of the Food Supply (COMA), which also reviewed the evidence concerning the potential protection against cancer afforded by fruit and vegetables.⁴⁵ The COMA study ranked the evidence into four categories, the top two being ‘strong’ and ‘moderate’. No ‘strong’ association was found between fruit and vegetable consumption and cancer at any site, while a ‘moderate’ association was noted for cancers of the stomach, colon and rectum. In contrast, the WCRF–AICR rated as ‘convincing’ the evidence for an association for the mouth/pharynx, stomach, colon, rectum and lung.⁷ *Convincing* was defined to mean that the evidence of causal relationships was conclusive and sufficient for making dietary recommendations. Clearly, COMA’s interpretation of the data is more cautious than the WCRF–AICR interpretation, but both committees recognise the importance of these foods in reducing cancer risk. The WCRF–AICR is currently updating its analysis to incorporate studies published since 1997.

Type 2 diabetes mellitus

Several recent reports have noted an association between increased consumption of plant foods and lower incidence of obesity (which is a risk factor for diabetes) and type 2 diabetes itself, although it is not clear at this stage whether this apparent protection arises principally from a lower body weight. In the dietary control of type 2 diabetes, vegetables are likely to be of particular value because of their content of fibre and low-energy density carbohydrates and their possible hypoglycaemic activity.^{6,10} Recently, a cross-sectional study in the United Kingdom revealed an inverse association between the risk of type 2 diabetes and frequent consumption of vegetables throughout the year, although the effect did not appear to be significant during the summer months.⁴⁶

Cataract and macular degeneration of the eye

Several studies in humans have reported that the risk of developing ocular cataracts is significantly higher in people with low dietary intakes of fruit and vegetables, vitamins C and E, and beta-carotene.^{12,47} A similar increased risk was observed in people with low levels of vitamins C and E in their blood. Experimental studies with model systems have added further support to the notion that above-average intakes of antioxidant nutrients may delay the onset of senile cataract.¹² More recently, a modest protective effect against the development of cataracts has been observed for higher intakes of the carotenoids lutein and zeaxanthin.⁴⁸

Age-related degeneration of the macula—the colour-sensitive yellow spot on the retina of the eye—is another serious cause of acute blindness in the elderly and is not reversible. Findings from a number of human studies suggest that people with low levels of carotenoids and the antioxidant vitamins C and E in their blood, and who smoke, are at increased risk of developing macular degeneration. Experimental studies indicate that two carotenoids in particular—lutein and zeaxanthin—appear to be accumulated by the macula, and in a human study when the dietary intake of carotenoids was analysed the sum of the intake of lutein and zeaxanthin had the strongest protective effect against macular degeneration. Taken together, these findings suggest that in many cases macular degeneration may be prevented by eliminating smoking and ensuring an adequate intake of fruit and vegetables.¹⁴ Of particular interest are several recent reports that highlight the presence of lutein and zeaxanthin in precise but different orientations in the membranes of the macula, which suggests that these two carotenoids may serve a special role in reducing the risk of age-related macular degeneration.^{49,50}

Special groups

Pregnancy

The *Australian Guide to Health Eating* advises an additional daily intake of around one serving of fruit and one of vegetables during pregnancy.¹⁶ This increase is especially important to provide the extra folate, vitamin C and other micro-nutrients recommended in the *Recommended Dietary Intakes for Use in Australia*.⁵¹

Lactation

The *Australian Guide to Health Eating* advises an additional daily intake of around three servings of fruit and two of vegetables during lactation.¹⁶ This is needed to meet the substantially increased requirement for vitamin A, folate, vitamins C and E, and other micro-nutrients at that time.⁵¹

Vegetarians

This guideline applies equally to vegetarians and to people eating other diets. Vegetarians should, however, give particular emphasis to eating legumes and nuts in order to increase their iron and complementary protein intake from plant sources. In addition, fruit juices or fruit should be consumed in the same meal in order to provide vitamin C, which will increase iron absorption.

PRACTICAL ASPECTS OF THIS GUIDELINE

Relationship to the *Australian Guide to Healthy Eating*

A wide variety of fruits is recommended, including apples and pears, citrus fruits, melons, tomatoes, berries, grapes, bananas, and stone fruits such as apricots and peaches. The *Australian Guide to Healthy Eating* contains recommendations for fruit include raw, stewed or canned varieties, with rather less emphasis on fruit juices and dried fruit since they tend to be lower in fibre and more energy dense respectively, although a modest intake of both (say, one serving a day) is acceptable.

A variety of vegetables is also recommended, including dark green vegetables such as spinach and broccoli; orange or yellow vegetables such as pumpkin and carrots; crucifers such as broccoli, cauliflower and cabbage; starchy vegetables such as potatoes, yams and the cereal food corn; and salad vegetables and fruits such as lettuce, tomato, cucumber and capsicum. Many of these foods can be eaten raw or slightly cooked, and some, for example salad items, should be served that way to maximise their nutrient content.

The dominance of potatoes as a source of vegetables in Australia is of some concern: they are not as rich in phytochemicals as many other vegetables and some of the more popular forms—French fries, for example—can also be relatively high in fat.

How do nuts and seeds fit in?

Many nuts and seeds are similar to fruits except that the seed is the main edible component and the whole structure becomes dry on maturing. Most nuts and seeds provide a wide range of nutrients and are generally pleasantly flavoured, so they can usefully be included with fruits and vegetables in plant-based dishes or other dishes such as stir-fries and in desserts. These foods are of particular value in providing significant levels of protein and essential fatty acids, both the n-6 fatty acids and, in some cases (such as walnuts, canola and flaxseed), the n-3 fatty acids.

Preparation of fruit and vegetables

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

When vegetables are cooked they should not be overcooked since this will cause loss of nutrients. Stir-frying is an effective method of cooking vegetables: it tends to minimise nutrient loss and provides a tasty product with good texture. Light microwaving and steaming are also better than deep-frying or prolonged boiling. Generally, when cooking vegetables it is useful to use a small amount of oil because this enhances absorption of the fat-soluble vitamins (for example, vitamins A and E) as well as other fat-soluble dietary components such as the carotenoids.

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

Dietary change

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

RELATIONSHIP TO OTHER GUIDELINES

The present guideline differs from the previous guideline in that two separate guidelines have been established, one for breads and cereals and one for vegetables, legumes and fruits. This change reflects the approach taken in the recent *Dietary Guidelines for Older Australians*⁵²: it is felt that the health benefits conferred by these two categories of plant foods often occur through largely different mechanisms and the dietary components involved are distributed differently between cereal grains and vegetables, legumes and fruits.

Enjoy a wide variety of nutritious foods

In order to obtain optimal health benefits from vegetables, legumes and fruit, a wide variety should be consumed.

Eat plenty of cereals (including breads, rice, pasta and noodles), preferably wholegrain

Apart from providing a good source of energy, cereal-based foods contribute a number of protective factors to the diet, complementing and extending many of the benefits derived from vegetables, legumes and fruits.

Limit saturated fat and moderate total fat intake

Vegetables, legumes and fruits are low in saturated fat.

Choose foods low in salt

Vegetables, legumes and fruits are low in salt (sodium) but are good suppliers of potassium.

Care for your food: prepare and store it safely

The nutritional value and palatability of fruit and vegetables will decline if these foods are not adequately stored. In addition, there is a risk that moist vegetables, legumes and fruits that are not peeled may develop bacteria on their surface and cause sickness if they are not well washed in clean water. Sprouts and salads are prone to contamination with bacteria and viruses and need thorough washing and sanitation.

CONCLUSION

There is strong evidence of a protective effect of certain vegetables, legumes and fruit against the development of a number of non-communicable chronic diseases, among them cancer, cardiovascular disease, type 2 diabetes, hypertension, and cataract and macular degeneration of the eye. This may, in part, be mediated through phytochemicals. Adults are encouraged to consume on average at least two helpings of fruit and five of vegetables each day, selected from a wide variety of types and colours and served cooked or raw, as appropriate.

EVIDENCE

There is Level II evidence (reference 9) and Level III evidence (references 3, 4, 8 and 30) in relation to the benefits of fruit and vegetable consumption and coronary heart disease, hypertension and stroke. There is Level III evidence (references 32 to 44) in relation to fruit and vegetable consumption and cancer of various kinds.

Although current evidence concerning the benefit of vegetables and fruit in protecting against several degenerative diseases is strongly persuasive, it is largely based on retrospective observational studies. Further prospective intervention studies are needed, although it is recognised that these are difficult, and costly, to carry out. Nevertheless, a considerable number are already under way and they will provide invaluable information in the coming decade. In addition, more needs to be established concerning the roles of the various phytochemicals in disease prevention, as well as their interaction with the range of genotypes found in the human population.

REFERENCES

1. Australian Parliament. *National Health Priority Areas initiative*. Current Issues Brief 18, 1999–2000. <www.aph.gov.au/library/pubs/cib/1999-2000cib18.htm>, viewed February 2002.
2. Ness AR, Powles JW. Fruit and vegetables, and cardiovascular disease: a review. *Int J Epidemiol* 1997;26:1–13.
3. Liu S, Manson JE, Lee IM, Cole SR, Hennekens CH, Willett WC et al. Fruit and vegetable intake and risk of cardiovascular disease: the Women's Health Study. *Am J Clin Nutr* 2000;72:922–8.
4. Law MR, Morris JK. By how much does fruit and vegetable consumption reduce the risk of ischaemic heart disease? *Eur J Clin Nutr* 1998;52:549–56.
5. Gillman MW, Cupples LA, Gagnon D, Posner BM, Ellison RC, Castelli WP, Wolf PA. Protective effects of fruits and vegetables on development of stroke in men. *JAMA* 1995;273:113–17.
6. Steinmetz KA, Potter JD. Vegetables, fruit and cancer prevention: a review. *J Am Diet Assoc* 1996;96:1027–39.
7. World Cancer Research Fund & American Institute for Cancer Research. Vegetables and fruits. In: *Food, nutrition and the prevention of cancer: a global perspective*. Washington, US: American Institute for Cancer Research 1997;436–46.
8. Ascherio A, Hennekens C, Willett WC, Sacks F, Rosner B, Manson J et al. Prospective study of nutritional factors, blood pressure and hypertension among US women. *Hypertension* 1996;27:1065–72.
9. Moore TJ, Vollmer WM, Appel LJ, Sacks FM, Svetkey LP, Vogt TM et al. Effects of dietary patterns on ambulatory blood pressure: results from the

- Dietary Approaches to Stop Hypertension (DASH) trial. *Hypertension* 1999;34:472–7.
10. Platel K, Srinivasan K. Plant foods in the management of diabetes mellitus: vegetables as potential hypoglycaemic agents. *Nahrung* 1997;41:68–74.
 11. Williams DE, Wareham DJ, Cox BD, Byrne CD, Hales CN, Day NE. Frequent salad vegetable consumption is associated with a reduction in the risk of diabetes mellitus. *J Clin Epidemiol* 1999;52:329–37.
 12. Taylor A. Cataract: relationship between nutrition and oxidation. *J Am Coll Nutr* 1993;12:138–46.
 13. Brown L, Rimm EB, Seddon JM, Giovannucci EL, Chasan-Taber L, Spiegelman D et al. A prospective study of carotenoid intake and risk of cataract extraction in US men. *Am J Clin Nutr* 1999;70:517–24.
 14. Snodderly DM. Evidence for protection against age-related macular degeneration by carotenoids and antioxidant vitamins. *Am J Clin Nutr* 1995;62:1448S–1461S.
 15. Goldberg J, Flowerdew G, Smith E, Brody JA, Tso MO. Factors associated with age-related macular degeneration: an analysis of data from the first National Health and Nutrition Examination Survey. *Am J Epidemiol* 1998;128:700–10.
 16. Department of Health and Family Services. *The Australian guide to healthy eating*. Canberra: DHFS, 1998.
 17. Cashel K, Jeffreson S. *The core food groups*. Endorsed by the National Health and Medical Research Council. Canberra: Australian Government Publishing Service, 1995;83.
 18. Baghurst P, Beaumont-Smith N, Baghurst KI, Cox D. *The relationship between the consumption of fruits and vegetables and health status*. Report to Department of Health and Aged Care. Adelaide: CSIRO, 1999:75.
 19. Kurtzweil P. Staking a claim to good health. *FDA Consumer Magazine* 1998;Nov/Dec:1–7.
 20. Hamilton CA. Low-density lipoprotein and oxidised low-density lipoprotein: their role in the development of atherosclerosis. *Pharmacol Therapeut* 1997;74:55–72.
 21. Tavani A, LaVerchia C. Beta-carotene and risk of coronary heart disease: a review of observational and intervention studies. *Biomed Pharmacother* 1999;53:409–16.
 22. Jenkins DJ, Popovich DG, Kendall CW, Vidgen E, Tariq N, Ranson TP et al. Effect of a diet high in vegetables, fruit and nuts on serum lipids. *Metabolism* 1997;46:530–7.
 23. Verhoef P, Stampfer MJ, Rimm EB. Folate and coronary heart disease. *Current Opinion in Lipidology* 1998;9:17–22.

24. Brussaard JH, Lowik MR, van der Berg H, Brants HA, Goldbohm RA. Folate intake and status among adults in the Netherlands. *Europ J Clin Nutr* 1997;51(suppl. 3):S46–S50.
25. Manson JE, Willett WC, Stampfer MJ, Colditz GA, Speizer FE, Hennekens CH. Vegetable and fruit consumption and incidence of stroke in women. *Circulation* 1994;89:932.
26. Van Duyn MA, Pivonka E. Overview of the health benefits of fruit and vegetable consumption for the dietetics professional: selected literature. *J Am Diet Assoc* 2000;100:1511–21.
27. Kawano Y, Matsuoka H, Takashita S, Omae T. Effects of magnesium supplementation in hypertensive patients: assessment by office, home and ambulatory blood pressures. *Hypertension* 1998;32:260–5.
28. Moore TJ, Vollmer WM, Appel LJ, Sacks FM, Svetkey LP, Vogt TM et al. Effect of dietary patterns on ambulatory blood pressure: results from the Dietary Approaches to Stop Hypertension (DASH) Trial Collaborative Research Group. *Hypertension* 1999;34:472–7.
29. Svetkey LP, Simons-Morton D, Vollmer WM, Appel LJ, Conlin PR, Ryan DH et al. Effects of dietary patterns on blood pressure: subgroup analysis of the Dietary Approaches to Stop Hypertension (DASH) randomised clinical trials. *Arch Intern Med* 1999;159:285–93.
30. Falkner B, Sherif K, Michel S, Kushner H. Dietary nutrients and blood pressure in urban minority adolescents at risk for hypertension. *Arch Pediat Adolesc Med* 2000;154:918–22.
31. Dreosti IE. What do our guidelines do for cancer prevention? Cereals, vegetables including legumes and fruit guideline. *Aust J Nutr Diet* 2000;57:221–2.
32. Franceschi S, Favero A, Conti E, Talamini R, Volpe R, Negri E et al. Food groups and oils and butter, and cancer of the oral cavity and pharynx. *Brit J Cancer* 1999;80:614–20.
33. Bosetti C, Negri E, Franceschi S, Conti E, Levi F, Tomei F et al. Risk factors for oral and pharyngeal cancer in women: a study from Italy and Switzerland. *Brit J Cancer* 2000;82:204–7.
34. Ekstrom AM, Serafini M, Nyren O, Hansson LE, Ye W, Wolk A. Dietary antioxidant intake and the risk of cardia cancer and non-cardia cancer in Sweden. *Int J Cancer* 2000;87:133–40.
35. Franceschi S. Nutrients and food groups and large bowel cancer in Europe. *Europ J Cancer Prev* 1999;9:549–52.
36. Michels KB, Giovannucci E, Joshipura KJ, Rosner BA, Stampfer MJ, Fuchs CS et al. Prospective study of fruit and vegetable consumption and incidence of colon and rectal cancer. *J Nat Cancer Inst* 2000;92:1740–52.
37. Voorrips LE, Goldbohm RA, Verhoeven DT, van Popper GA, Sturmans F, Herman RJ et al. Vegetable and fruit consumption and lung cancer risk in

- the Netherlands Cohort Study on diet and cancer. *Cancer Causes and Control* 2000;11:101–15.
38. Michaud DS, Feskanich D, Rimm EB, Colditz GA, Speizer FG, Willett WC et al. Intake of specific carotenoids and risk of lung cancer in two prospective US cohorts. *Am J Clin Nutr* 2000;72:990–7.
 39. Steinmaus CM, Nunez S, Smith AH. Diet and bladder cancer: a meta-analysis of six dietary variables. *Am J Epidemiol* 2000;151:693–703.
 40. Nagano J, Kano S, Preston DL, Moriwaki H, Sharp GB, Koyama K et al. Bladder cancer incidence in relation to vegetable and fruit consumption: a prospective study of atomic bomb survivors. *Int J Cancer* 2000;86:132–8.
 41. Ronco A, De Stefani E, Boffetta P, Deneo-Pellegrini H, Mendilaharsu M, Leborgne F. Vegetables fruits and related nutrients and risk of breast cancer: a case control study in Uruguay. *Nutr & Cancer* 1999;35:111–19.
 42. Gandini S, Merzenich H, Robertson L, Boyle P. Meta-analysis of breast cancer risk and diet: the role of fruit and vegetable consumption and the intake of associated micronutrients. *Europ J Cancer* 2000;36:636–46.
 43. Jain MG, Hislop GT, Howe GR, Ghadirian P. Plant foods, antioxidants and prostate cancer risk. *Nutr and Cancer* 1999;34:173–84.
 44. Cohen JH, Kristal AR, Stanford JL. Fruit and vegetable intake and prostate cancer risk. *J Nat Cancer Inst* 2000;92:61–8.
 45. UK Department of Health: *Nutritional aspects of the development of cancer: report of the working group on diet and cancer of the Committee on Medical Aspects of Food and Nutrition Policy*. Norwich, UK: The Stationery Office, 1998.
 46. Williams DE, Wareham NJ, Cox BD, Byrne CD, Hales CN, Day NE. Frequent salad vegetable consumption is associated with a reduction in the risk of diabetes mellitus. *J Clin Epidemiol* 1999;52:329–35.
 47. Tavani A, Negri E, LaVecchia C. Food and nutrient intake and cataract. *Ann Epidemiol* 1996;6:41–6.
 48. Brown L, Rimm EB, Seddon JM, Giovannucci EL, Chasan-Taber L, Spiegelman D et al. A prospective study of carotenoid intake and risk of cataract extraction in US men. *Am J Clin Nutr* 1999;70:517–24.
 49. Bone RA, Landrum JT, Friedes LM. Distribution of lutein and zeaxanthin stereoisomers in the human retina. *Expl Eye Res* 1997;64:211–18.
 50. Sommerburg EG, Siems WG, Hurst JS, Lewis JW, Klinger DS, van Kuijku. Lutein and zeaxanthin are associated with photoreceptors in the human retina. *Curr Eye Res* 1999;19:491–5.
 51. National Health and Medical Research Council. *Recommended dietary intakes for use in Australia*. Canberra: Australian Government Publishing Service, 1991.
 52. National Health and Medical Research Council. *Dietary guidelines for older Australians*. Canberra: NHMRC, 1999.

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

Peter Williams

TERMINOLOGY

Cereals

Cereals refers to the entire class of cereal foods, including whole or partially processed cereal grains (for example, rice, oats, corn and barley), breads, breakfast cereals, pasta, noodles, and other plain cereal products such as flour, polenta, semolina, burghul, bran and wheatgerm. It excludes cereal-based products with a significant amount of added fat and sugar—cakes, pastries, biscuits, and so on.

Breads

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.

Pasta and noodles

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.

Wholegrain

Wholegrain refers to cereal foods that incorporate all the components of the natural grain, including the bran and germ. Foods that contain at least 51 per cent by weight of any combination of whole grains can be termed *wholegrain*.¹ This definition includes such foods as wholemeal breads and crispbreads, many high-fibre breakfast cereals, oatmeal, wholemeal pasta, brown rice and popcorn.

BACKGROUND

From an evolutionary perspective, consumption of cereal grain is relatively recent, dating from only 5000 to 10 000 years ago, yet, today, eight cereals—wheat, maize, rice, barley, sorghum, oats, rye and millet—provide more than 56 per cent of the energy and 50 per cent of the protein consumed on earth.² Many traditional hunter-gatherer societies had diets with a relatively low proportion of energy from carbohydrate (22–40 per cent) and only small amounts of grain³, although Indigenous Australians may have consumed large quantities of grain in some areas. However, it is difficult to base conclusions about desirable dietary patterns for modern societies simply from an assessment of traditional eating patterns of hunter-gatherers. Many things such as activity patterns, availability of various foods and genetic background can influence food consumption patterns or dietary needs. Recommendations made in a recent UN report state that carbohydrate should provide more than 55 per cent of energy for optimal health.⁴

Cereal grains form the basis of diets in many different cultures and cuisines. They are generally an excellent source of carbohydrate and dietary fibre and are also an important source of protein (ranging from 8 to 16 grams per 100 grams). They are mostly low in fat and are good sources of B-group vitamins, vitamin E and many minerals, notably iron, zinc, magnesium and phosphorus. Eating enough cereal foods helps ensure an adequate nutritional intake. They can also be stored safely for long periods and are relatively inexpensive: in 1998–99 purchases of cereal products accounted for only 7 per cent of household food expenditure.⁵ Ecologically, a high-carbohydrate diet based on cereals makes good use of the world's resources, since grain crops require relatively few input resources per unit of food energy produced.⁶ For these reasons all current dietary guides have cereal foods as the largest component of the recommended daily food intake.

Current intakes in Australia

Apparent consumption of cereal foods in Australia (an estimate of intake based on national food-disappearance data), has remained relatively constant since the 1930s. In 1998–99 apparent consumption was 138.1 kilograms per person.⁷ There have, however, been changes in the mix of products since the 1930s. Consumption of rice and breakfast cereals has increased significantly and consumption of flour has fallen. Bread consumption has varied over time: from 69.1 kilograms per head a year in 1958–59 it fell to a low of 44.4 kilograms in 1988–89 then rose to 53.4 kilograms in 1998–99.

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

areas and significantly higher among people born in Southeast Asia (because of their much higher consumption of rice).

The National Nutrition Survey also found that for adult Australians, cereals are important sources of energy, carbohydrate, dietary fibre, thiamin, iron and magnesium, providing more than 20 per cent of the total daily intake of these nutrients (see Table 1.2.1). They also provided more than 10 per cent of the daily intakes of protein, polyunsaturated fat, riboflavin, niacin, folate, calcium, phosphorus and zinc.

Table 1.2.1 Percentage of mean adult nutrient intake provided by cereal foods, 1995⁹

Nutrient	Men aged 19 years and over	Women aged 19 years and over
	%	%
Energy	20.0	20.8
Carbohydrate	33.2	33.1
Dietary fibre	34.9	33.6
Thiamin	41.3	40.5
Iron	30.1	29.3
Magnesium	24.3	24.8

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

Two Australian studies have shown that socioeconomic status may affect cereal intake. One study of cereal intakes in various socio-economic groups in Australia found that cereal foods contribute more to nutrient intakes among upper occupational groups for both males and females.¹⁰ A study of 18-year-olds in Western Australia also found higher levels of cereal consumption in groups of higher socio-economic status.¹¹

Most Australians seem to be satisfied with the amount of cereal foods they eat. In the National Nutrition Survey only 8 per cent of respondents aged 19 years or more reported they would like to change the amount they ate; this compares with up to 30 per cent reporting that they wanted to eat more fruit and vegetables.¹² Despite this, the survey data show that even among adults with the highest intakes (those aged 19–24 years), on the day of the survey only 34 per cent of men and 21 per cent of women met the recommended core food group cereal targets of seven servings a day.¹³

SCIENTIFIC BASIS

There have been many experimental studies dealing with individual nutritional components provided by cereal foods (such as dietary fibre, starch and vitamin E), but relatively few prospective studies or controlled experimental trials have

used whole foods to find support for this dietary guideline. It is difficult to gain people's acceptance of long-term changes to the largest staple components of their diets—and generally impossible to do so in a double-blind manner. As a result, most of the available evidence comes from ecological, cross-sectional, case control and cohort studies. Even in these the dietary methodology is often inadequate for analysing the consumption of different types of cereals or quantifying dose-responses.

All recent reviews have supported the beneficial effects of cereal fibre and whole grains in relation to decreased risk of coronary heart disease and some cancers^{1,14–17}, and data from several countries suggest that higher intakes of breads and cereals help people achieve dietary targets for lower fat consumption.^{18,19} Cereals are also a major source of resistant starch in the diet, which is important for colon health.²⁰ In 1999 the US Food and Drug Administration approved the health claim that 'diets rich in whole-grain foods and other plant foods and low in total fat, saturated fat and cholesterol may reduce the risk of heart disease and certain cancers'.¹⁴

Coronary heart disease

The published results of over 200 human trials have led to the general conclusion that foods rich in soluble fibre can lower plasma cholesterol.^{21–23} The National Heart Foundation of Australia has stated, 'The consumption of dietary fibre, especially cereal fibre, is associated with a lower risk of CHD'.²⁴ Meta-analyses of intervention trials with two cereal foods, oats and psyllium, have shown that these are particularly effective in reducing serum cholesterol.^{25,26} By contrast, controlled human trials with supplements of isolated wheat fibre have consistently shown no effect on plasma cholesterol.²¹

A large prospective study of male health professionals in the United States found that dietary fibre intake was strongly associated with reduced rates of myocardial infarction and that cereal fibre was apparently more protective than fibre from fruits or vegetables.²⁷ The study reported a 29 per cent reduction in coronary heart disease for every 10-gram increase in daily intake of cereal fibre. Other studies have also found a stronger association between cereal fibre and reduced risk of coronary heart disease than with fibre from fruit or vegetables.^{28–30} Analysis of a prospective study of 31 284 post-menopausal women in Iowa found the relative risk of CHD was 0.76 (95%CI: 0.55–1.05) among women in the highest quintile of dietary fibre intakes compared with the lowest.³⁵

The principal mechanism is probably viscous polysaccharides acting in the gastrointestinal tract to decrease reabsorption of biliary cholesterol³¹, but other components may be involved in the protective effect of wholegrain cereals: vitamin E, folate, selenium, phytoestrogens and phytic acid may all be important.³²

In the Nurses Health Study, wholegrain consumption was associated with significant reductions in risk of both CHD³³ and ischaemic stroke.³⁴ In older women there is also evidence from the Iowa Women's Health Study of a clear

inverse association between wholegrain intake and the risk of ischaemic heart disease³⁵ as well as all-cause mortality.³⁶ The authors calculated that if all women consumed one serving of wholegrain foods each day total mortality rates might be reduced by 8 per cent or more.

In that study there was a small positive association (adjusted hazard rate ratio 1.16) between refined grain intake and total mortality, but this was attenuated and lost significance when wholegrains were added to the model. There was no association between refined grain intake and risk of CHD.³⁵ In a study of dietary associates in patients with established coronary disease, a high intake of not only wholegrain but also total cereal products was associated with lower total cholesterol.³⁷

Dietary carbohydrates may also exert an influence on cardiovascular disease risk via their effect on insulin response. High-glycaemic index (or high GI) carbohydrates are characterised by rapid absorption and high post-prandial glucose and insulin responses and may result in decreased insulin sensitivity³⁸, a risk factor for CHD.³⁹ (Appendix I to the *Dietary Guidelines for Older Australians* provides a detailed discussion of the glycaemic index.) At least three cross-sectional studies have also found an inverse relationship between HDL cholesterol and the dietary glycaemic load.^{40–42} In the prospective Nurses Health Study, over 10 years both the glycaemic load and the total diet GI were predictive of CHD risk.⁴³ One randomised crossover study with type 2 diabetics found that lowering the GI of a diet (mainly by altering the physical form of the cereals) resulted in significantly lower LDL and higher HDL cholesterol levels.⁴⁴ A study in free-living Australian diabetic subjects also found HDL cholesterol levels were higher on a low- versus high-GI diet.⁴⁵

Obesity

Although total energy intake and overall nutrient density appear to be the most important factors affecting weight regulation, a high-fibre, low-fat diet is recommended for maintenance of body weight and prevention of obesity.^{46–48} Obesity is associated with low fibre intake.⁴⁹ When high-starch, high-sucrose and high-fat ad libitum diets were compared, energy intake was lowest on the high-starch, high-fibre diet⁵⁰, and higher intakes of carbohydrates have been linked to lower waist-hip ratios and lower body mass index.⁵¹ The recent CARMEN study found there were no significant differences in weight loss when fat was replaced with either simple or complex carbohydrate, but energy density and energy intake were lower with diets high in complex carbohydrates.⁵²

There are several ways high-fibre cereals can reduce energy intake and help maintain weight: they take longer to eat; they decrease the energy density of a meal; and some fibres may slow gastric emptying and affect gastrointestinal hormones that influence food intake.⁵³ Compared with low-GI choices, consumption of high-GI carbohydrates promotes a more rapid return of hunger and increases subsequent energy intake, and slower digestion of carbohydrate is associated with higher satiety.⁵⁴ Thus, consumption of wholegrain and lower GI cereals, instead of highly refined cereals, may help prevent excess weight gain.^{55,56}

Diabetes

The joint WHO–FAO consultation on carbohydrates concluded that foods rich in slowly digested, or resistant, starch or high in soluble fibre might be protective against diabetes.⁴ Recent large prospective studies of men and women have found cereal fibre intake was inversely associated with the risk of developing type 2 diabetes and that the protective effect was even greater when combined with a low total glycaemic load.^{30,57}

A large prospective study of adult women in the United States found that a lower risk of type 2 diabetes was associated with higher intakes of all cereal grains (RR 0.75; 95%CI: 0.63–0.89) and wholegrains in particular (RR 0.73; 95%CI: 0.63–0.85), whereas a higher intake of refined grain was related to increased risk (RR 1.26; 95%CI: 1.08–1.46).⁵⁸ The individual foods associated with the strongest protective effects were wholegrain breakfast cereal, brown rice and bran. However, in that study *refined grain* included a wide range of higher fat cereal-based foods such as cakes, desserts and pizzas, and *wholegrain* foods included some that are relatively refined (such as couscous).

For people with established type 2 diabetes, use of low-GI foods is associated with improvement in glycaemic control.^{44,59} In Southern European patients with type 2 diabetes, HbA_{1c} was 11 per cent lower in patients whose diets were in the lowest quartile for GI compared with those in the highest and was related to eating more pasta.⁴¹ During pregnancy, women on a low-GI diet (eating bran breakfast cereal, wholegrain bread and pasta) experienced no change in their glycaemic response to a 500-calorie test meal with 55 per cent of energy from carbohydrate, whereas those who switched to a high-GI diet experienced a 190 per cent increase in their response.⁶⁰

Cancer

Two major reviews of the relationship between cereal consumption and cancer prevention have been published.^{61,62} It is difficult to evaluate many studies because of the paucity of biological markers; the inadequacy of many food-intake measurements, which often do not distinguish the degree of refinement of cereal foods; and the low overall intakes of cereal fibre in many of the studies from the United States. There is, however, emerging agreement on the probable protective role of cereals in relation to some important cancer types. In particular, it appears that wholegrain intake confers benefits. In a review of 40 case-control studies of 20 cancers, the pooled odds ratio for high versus low wholegrain intake was 0.66 (95%CI: 0.60–0.72).⁶³ Among the protective components in wholegrains may be fermentable carbohydrates, oligosaccharides, flavonoids, phenolics, phytoestrogens, lignans, protease inhibitors, saponins and selenium.^{64–66}

Some case-control studies have suggested not only that wholegrains are protective but also that, conversely, consumption of refined cereals (including bread, pasta and rice) increases the risk of cancers of the oral cavity,

oesophagus, larynx, stomach and colon.^{66–68} However, the authors have been cautious about inferring causality from these associations, noting that diets high in refined cereals are often also poor in fruit and vegetables.

Colorectal cancer

Prospective data from the large Health Professionals Follow-up Study suggest that dietary fibre intake is inversely associated with the risk of colorectal adenoma in men, the relative risk in the highest quintile versus the lowest being 0.36. All sources of dietary fibre were protective, but the effect was stronger for grain sources than for fruit or vegetables.⁶⁹

The World Cancer Research Fund review also found that diets high in both starch and dietary fibre could possibly decrease the risk of colorectal cancer.⁶¹ It was concluded that cereals may well have a protective effect but that there was still insufficient evidence. Two subsequent reviews have reported more definite conclusions. A recent consensus statement from the European Cancer Prevention Organisation, based on a review of 58 epidemiological studies, concluded, 'A diet rich in high-fibre cereal is associated with a reduced risk of colorectal cancer'.⁶² This conclusion is supported by a meta-analysis of case-control studies of wholegrain intake and colorectal cancer, which calculated a pooled odds ratio of 0.79 (95%CI: 0.69–0.89) when high and low intakes of wholegrains were compared.⁶³

Resistant starch may also favourably affect some of the faecal markers of colon cancer risk, in a way similar to dietary fibre.²⁰ Epidemiological studies show a strong inverse correlation between resistant starch intake and the risk of cancer of the large bowel.⁷⁰ Cereal foods are estimated to provide 42 per cent of the resistant starch in the Australian diet.⁷¹

In experimental studies, wholemeal rye bread, as compared with white bread, reduced the concentration of some compounds that are putative colon cancer risk markers.⁷² In some prevention trials, supplements of wheat bran have reduced the incidence of rectal polyps in predisposed individuals⁷³ and, when combined with a low-fat diet, reduced the incidence of large adenomas.⁷⁴

The effectiveness of fibre, however, has been disputed in recent years. Analysis of 16 years of follow-up in the Nurses Health Study did not support the hypothesis that dietary fibre intake can reduce the risk of colon cancer, although the power of this study was limited by the low range of median cereal fibre intakes (ranging from 1 gram a day to only 4.8 grams a day in the highest quintile).⁷⁵ The multi-centre Phoenix colon cancer prevention trial, involving 1429 subjects with pre-existing polyps, examined the effect of a wheat bran breakfast cereal supplement taken over three years. This study found no effect, but the authors noted that the actual supplementary fibre intake (9.4 grams a day) may have been too small to produce a significant change.⁷⁶ Third, a polyp-prevention trial in the United States found that a low-fat, high-fibre diet (with increased intakes of fruit, vegetables, cereals and legumes) did not alter the recurrence of adenomatous polyps.⁷⁷

The most recent Cochrane Database systematic review of five intervention studies concluded that there is currently no evidence from RCTs to suggest that increased dietary fibre intake will reduce the incidence of adenomatous polyps within a two to four year period.⁷⁸ However, most of the studies that were considered used isolated dietary supplements rather than whole foods and the consistent findings of a protective association from high fibre diets in the case-control and cohort studies suggest that the mechanisms may not be fully understood at this stage.

Breast cancer

Fibre may reduce the intestinal reabsorption of oestrogen, and bioactive cereal components such as lignans may be protective through their action as weak phytoestrogens. A comparison of national consumption data from various countries suggests that energy from cereals is inversely related to breast cancer risk⁷⁹, and a meta-analysis of 12 case-control studies found a significant reduction in risk with increasing dietary fibre.⁸⁰ The World Cancer Research Fund report concluded that dietary fibre possibly decreases the risk of breast cancer⁶¹ and the European Cancer Prevention Organisation consensus meeting agreed that there is evidence to suggest cereal fibre provides protection against breast cancer⁶², although this is still uncertain.⁸¹ More recently, a study of breast cancer recurrence over five years found energy-adjusted bread and cereal consumption was protective (hazard ratio 0.55), especially in post-menopausal women.⁸²

Stomach cancer

In relation to stomach cancer, the World Cancer Research Fund report concluded from the evidence of six case-control studies that there was a possible protective association for consumption of wholegrain cereals and cereal products but that the evidence for cereals as a whole was inconsistent and inconclusive.⁶¹

Other cancers

One large cross-national study has found that prostate cancer mortality is inversely associated with estimated consumption of cereals⁸³, and case-control studies suggest that wholegrain foods are protective.⁸¹ A few case-control studies report a protective effect of wholegrain consumption on oral and pharyngeal cancers^{84,85}, but data from human intervention studies are not available for any of these cancers.

Constipation and diverticular disease

There is a strong correlation between dietary fibre intake and mean daily stool weight⁸⁶, and cereal fibre has been found to improve bowel function by increasing faecal bulk and reducing transit time, resulting in softer, larger stools and more frequent bowel action.^{87–89} Diets rich in insoluble fibre, such as that present in wholegrain cereals and breads, are associated with a low prevalence of constipation and diverticular disease. The US prospective study of 43 881 male health professionals found evidence that a diet high in fibre—particularly the

cellulose component, which is obtained largely from cereal foods—was significantly associated with a decreased risk of diverticular disease.⁹⁰

Hypertension

Hypertension remains an important risk factor for cardiovascular and cerebrovascular morbidity and mortality, and a reduction in sodium intake is one of the primary preventive measures. Cereals in their natural state are very low in salt and have a favourable potassium–sodium ratio, but processed cereal foods, especially bread, are major sources of salt in the Australian diet. The Victorian Nutrition Survey found that processed cereal products gave men 28 per cent of their daily sodium intake and women 26 per cent—more than twice as much as any other food group.⁹¹ Both dietary fibre and magnesium may be protective against hypertension, and cereal foods are important sources of both these nutrients; but fruit sources of fibre appear more protective than cereal sources.^{92,93}

Nutrient density

Two of the main cereal foods, breakfast cereals and breads, are often fortified with vitamins and minerals that can be marginal in the diet. For example, data from the National Nutrition Survey show that people who regularly include breakfast cereal in their diet are much more likely to meet the recommended dietary intakes for iron, calcium, magnesium, folate, riboflavin and thiamin.⁹⁴ Bread has long been a useful staple for fortification⁹⁵, and some breads are now sources of additional fibre, iron, folate and n-3 fats. In 1991 it became mandatory for all bread to be fortified with thiamin, and since then there has been an apparent decline in the prevalence of Wernicke-Korsakoff syndrome in Australia.^{96,97}

PRACTICAL ASPECTS OF THIS GUIDELINE

Relationship to the *Australian Guide to Healthy Eating*

The *Australian Guide to Healthy Eating* recommends that breads, cereals, rice, pasta and noodles form the basis of a healthy diet, with the greatest proportion of food coming from this group.⁹⁸ The recommended number of daily cereal servings for adults aged 19 to 60 years is four to nine for women and five to 12 for men. A serving equates to two slices of bread; one cup of cooked rice, pasta or noodles; one cup of porridge; one-and-a-third cups of breakfast cereal; or half a cup of muesli. Cereal-based foods such as cakes, biscuits and pastries—which can have high levels of added fats and sugars—are not included in this recommendation and should be regarded as occasional treats only.

There are some easy ways of achieving these recommended targets:

- consuming breads with each meal

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

- regularly using rice, couscous, pasta or noodles to accompany hot dishes
- eating breakfast cereals daily
- including wholegrain cereals as extenders to soups and casseroles
- using oats in crumble toppings on desserts
- choosing grain-based snacks such as low-fat cereal bars, muffins and popcorn.

Sodium intake

Bread is the most commonly consumed cereal food in Australia; it has a typical sodium content of around 450 milligrams per 100 grams. The National Nutrition Survey reported the mean daily consumption of regular and fancy breads by people aged 19 years and over was 101.7 grams, which would contribute 458 milligrams of sodium (around 20 per cent of the recommended maximum sodium intake⁹⁹) compared with these products' contribution of only 10 per cent to the daily energy intake.⁹

Greater consumption of cereal foods with high salt levels could make it more difficult for people to limit their sodium intake, but this is not a reason to recommend against plentiful consumption of cereals. People seeking to increase their cereal intake should opt for cereals that are lower in salt—such as rice, oats, couscous, pasta, and many lower salt varieties of breakfast cereals and breads.

Glycaemic index

The glycaemic index of a food is a physiologically based classification of carbohydrate-containing foods according to their potential to raise blood glucose. The FAO–WHO consultation on carbohydrates recommended that ‘the glycaemic index of foods be used in conjunction with information about food composition to guide food choices’.⁴ Various factors can affect the GI value of a food, among them the particle size of milled grains; the ratio of amylose to amylopectin; the degree of starch gelatinisation; and the presence of other food components such as viscous soluble fibres, fat, protein and organic acids. Lower GI diets may possibly be protective against both diabetes and heart disease and low-GI diets may help with weight control.

Many processed starchy cereal foods—such as most breads, rices and breakfast cereals—tend to have high GI values. This does not mean that high-GI cereal foods need to be avoided altogether: the glycaemic load of a diet can be balanced by combining high- and low-GI carbohydrate sources in the same meal. Exchanging half the carbohydrate from high to low GI will lower the GI of the overall diet by about 15 units, sufficient to bring about clinical improvements in glucose metabolism in people with diabetes.¹⁰⁰

To achieve a lower GI diet, consumption of slowly digested cereal foods, such as grainy breads, pasta, low-GI breakfast cereals and high-amylose rice, is preferred. Choosing lower GI foods from wholegrain foods with low levels of

saturated fat will also increase the protective cereal fibre and phytochemical content of the diet.

RELATIONSHIP TO OTHER GUIDELINES

In the 1992 edition of the *Dietary Guidelines for Australians*, the guideline relating to cereal foods was, ‘Eat plenty of breads and cereals (preferably wholegrain), vegetables, legumes and fruits’.¹⁰¹ The 2002 guidelines make two separate recommendations, one relating to vegetables and fruit and one relating to cereals—to give both groupings greater prominence. The emphasis on ‘wholegrain’ has been retained because of the growing evidence of the health benefits of wholegrain cereal products as compared with refined ones.

Enjoy a wide variety of nutritious foods

Different cereals provide differing amounts and types of dietary fibre, as well as differing levels of potentially active phytochemicals and nutritive antioxidants.¹⁰² The levels of some nutrients (such as selenium) in cereals vary considerably according to the growing region: it is important to eat a wide variety of cereal foods to maximise their nutritional benefits.

Limit saturated fat and moderate total fat intake

Cereals are naturally very low in saturated fat, so increased cereal consumption is consistent with this guideline as long as the amount of fat added—in the form of fat spreads on bread, oil added to pasta, fried rice, and so on—is limited. Care also needs to be taken to limit the intake of other cereal-based foods such as biscuits, cakes and pastries and some instant noodles: they can contain high levels of added saturated fat and are treated as ‘extra foods’ in the *Australian Guide to Healthy Eating*.

Choose foods low in salt

Standard commercial breads and some breakfast cereals are major sources of salt in the diet. To cut down on salt intake, lower salt cereal products and unprocessed whole grains should be preferred.

CONCLUSION

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

EVIDENCE

There is Level I evidence of the cholesterol-lowering properties of oats and psyllium (references 25 and 26), of the cholesterol-lowering properties of cereal fibres generally (reference 23) and of the preventive effect of dietary fibre on constipation (reference 88). There is Level II evidence for a protective effect of wheat fibre on rectal polyps (reference 73) and, in combination with a low-fat diet, on large adenomas (reference 74), but there is also Level II evidence of no effect on the recurrence of adenomas (references 76 and 77).

There is Level III evidence for the following:

- low-GI diets and improved lipid profile and glycaemic control in diabetics (references 40, 42, 44, 45 and 60)
- cereal fibre and improvement in risk markers for colorectal cancer (reference 72)
- wholegrain cereal and reduced risk of coronary heart disease (references 33 to 35)
- wholegrain cereal and reduced risk of diabetes (reference 58)
- cereal fibre and reduced risk of coronary heart disease (references 15, 27 to 29 and 57)
- cereal fibre and reduced risk of breast cancer (references 78 to 82)
- wholegrain cereal and reduced risk of cancers (references 63, 67 to 69, 75, 81 and 83 to 85)
- cereal and weight control (references 50 and 51)
- dietary fibre and reduced risk of diverticular disease (reference 90).

REFERENCES

1. Jacobs D, Pereira M, Slavin J, Marquart L. Defining the impact of whole-grain intake on chronic disease. *Cereal Foods World* 2000;45:51–3.
2. Cordain L. Cereal grains: humanity's double-edged sword. *World Rev Nutr Diet* 1999;84:19–73.
3. Cordain L, Brand Miller J, Eaton S, Mann N, Holt S, Speth J. Plant–animal subsistence ratios and macronutrient energy estimations in worldwide hunter–gatherer diets. *Am J Clin Nutr* 2000;71:682–92.
4. UN Food and Agriculture Organization. *Carbohydrates in human nutrition: report of a joint FAO–WHO expert consultation*. Rome: FAO, 1998.
5. Australian Bureau of Statistics. *Household Expenditure Survey: detailed expenditure items, Australia, 1998–99*. Canberra: ABS, 2000.
6. Connor W. The benefits of a high-carbohydrate diet. In: Wardlaw G, ed. *Perspectives in nutrition*. Boston: McGraw-Hill, 1999:86–7.

I.2 EAT PLENTY OF CEREALS (INCLUDING BREADS, RICE, PASTA AND NOODLES), PREFERABLY WHOLEGRAIN

7. Australian Bureau of Statistics. *Apparent consumption of foodstuffs, Australia, 1997–98, 1998–99*. Canberra: ABS, 2000.
8. McLennan W, Podger A. *National Nutrition Survey: foods eaten, Australia, 1995*. Canberra: Australian Bureau of Statistics, 1999.
9. McLennan W, Podger A. *National Nutrition Survey: nutrient intakes and physical measurements, Australia, 1995*. Canberra: Australian Bureau of Statistics, 1998.
10. Syrette J, Baghurst K, Record S. Socioeconomic determinants of the contribution of cereal foods to nutrient intake in the Australian population. *Food Aust* 1990;42:330–7.
11. Milligan R, Burke V, Beilin L, Dunbar D, Spencer M, Balde E et al. Influence of gender and socio-economic status of dietary patterns and nutrient intakes in 18-year-old Australians. *Aust NZ J Pub Hlth* 1998;22:485–93.
12. McLennan W, Podger A. *National Nutrition Survey: selected highlights, Australia, 1995*. Canberra: Australian Bureau of Statistics, 1997.
13. Cashel K, Jeffreson S. *The core food groups: the scientific basis for developing nutrition education tools*. Canberra: NHMRC, 1995.
14. Anderson J, Hanna T, Peng X, Kryscio R. Whole grain foods and heart disease. *J Am Coll Nutr* 2000;19:291S–299S.
15. Kushi L, Meyer K, Jacobs D. Cereals, legumes, and chronic disease risk reduction: evidence from epidemiological studies. *Am J Clin Nutr* 1999;70(suppl.):451S–458S.
16. Munoz de Chavez M, Chavez A. Diet that prevents cancer: recommendations from the American Institute for Cancer Research. *Int J Cancer* 1998;11:85–9.
17. Truswell A. Cereal grains and coronary heart disease. *Eur J Clin Nutr* 2002;56:1–14.
18. Pryer J, Brunner E, Elliott P, Nichols R, Dimond H, Marmot M. Who complied with COMA 1984 dietary fat recommendations among a nationally representative sample of British adults in 1986–87 and what did they eat? *Eur J Clin Nutr* 1995;49:718–28.
19. Haraldsdottir J. Dietary guidelines and patterns of intake in Denmark. *Br J Nutr* 1999;81(suppl. 2):S43–S48.
20. Topping D, Clifton P. Short chain fatty acids and human colonic function roles of resistant starch and nonstarch polysaccharide. *Physiol Rev* 2001;81:1031–64.
21. Truswell A. Dietary fibre and blood lipids. *Curr Opin Lipidol* 1995;6:14–19.
22. Glore S, van Treek D, Kneehams A, Guild M. Soluble fibre and serum lipids: a literature review. *J Am Diet Assoc* 1994;94:425–36.

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

23. Brown L, Rosner B, Willett W, Sacks F. Cholesterol-lowering effects of dietary fibre: a meta-analysis. *Am J Clin Nutr* 1999;69:30–42.
24. National Heart Foundation. *Dietary fibre: a policy statement prepared by the Diet and Heart Disease Advisory Committee for the National Heart Foundation*. Canberra: NHF, 1997.
25. Ripsin C, Keenan J, Jacobs D, Elmer P, Welch R, Van Horn L et al. Oat products and lipid lowering: a meta-analysis. *JAMA* 1992;267:3317–25.
26. Olson B, Anderson S, Becker M, Anderson J, Hunninghake D, Jenkins D et al. Psyllium-enriched cereals lower blood total cholesterol and LDL cholesterol, but not HDL cholesterol in hypercholesterolemic adults: results of a meta-analysis. *J Nutr* 1997;127:1973–80.
27. Rimm E, Ascherio A, Giovannucci E, Spiegelman D, Stampfer M, Willett W. Vegetable, fruit, and cereal fibre intake and risk of coronary heart disease among men. *JAMA* 1996;275:447–51.
28. Pietinen P, Rimm E, Korkkohen P, Hartmen A, Willett W, Albanes D et al. Intake of dietary fibre and risk of coronary heart disease in a cohort of Finnish men. The a-Tocopherol, b-Carotene Cancer Prevention Study. *Circulation* 1996;94:2720–7.
29. Wolk A, Manson J, Stampfer M, Colditz G, Hu F, Speizer F et al. Long-term intake of dietary fiber and decreased risk of coronary heart disease. *JAMA* 1999;281:1998–2004.
30. Salmeron J, Ascherio A, Rimm E, Colditz G, Spiegelman D, Jenkins D et al. Dietary fiber, glycaemic load and risk of NIDDM in men. *Diab Care* 1997;20:545–50.
31. Van Horn L. Fiber, lipids, and coronary heart disease: a statement for healthcare professionals from the Nutrition Committee, American Heart Foundation. *Circulation* 1997;95:2701–4.
32. Anderson J, Hanna T. Whole grains and protection against coronary heart disease: what are the active components and mechanisms? *Am J Clin Nutr* 1999;70:307–8.
33. Liu S, Stampfer M, Hu F, Giovannucci E, Rimm E, Manson J et al. Whole-grain consumption and risk of coronary heart disease: results from the Nurses' Health Study. *Am J Clin Nutr* 1999;70:412–19.
34. Liu S, Manson J, Stampfer M, Rexrode K, Hu F, Rimm E et al. Whole grain consumption and risk of ischemic stroke in women: a prospective study. *JAMA* 2000;284:1534–40.
35. Jacobs D, Meyer K, Kushi L, Folsom A. Whole-grain intake may reduce the risk of ischemic heart disease death in post-menopausal women: the Iowa Women's Health Study. *Am J Clin Nutr* 1998;68:248–57.
36. Jacobs D, Meyer K, Kushi L, Folsom A. Is whole grain intake associated with reduced total and cause-specific death rates in older women? The Iowa Women's Health Study. *Am J Pub Hlth* 1999;89:322–9.

37. Erkkila A, Sarkkinen E, Lehto S, Pyorala K, Uusitupa M. Dietary associates of serum total, LDL, and HDL cholesterol and triglycerides in patients with coronary heart disease. *Prev Med* 1999;28:558–65.
38. Wolever T. The glycemic index. *World Rev Nutr Diet* 1990;62:120–85.
39. Despres J, Lamarache B, Mauriege P, Cantin B, Dagenais G, Mooranji S et al. Hyperinsulinemia as an independent risk factor for ischemic heart disease. *New Engl J Med* 1996;334:952–7.
40. Frost G, Leeds A, Dore C, Madeiros S, Brading S, Dornhorst A. Glycaemic index as a determinant of serum HDL-cholesterol concentration. *Lancet* 1999;353:1045–8.
41. Buyken A, Toeller M, Heitkamp G, Karamanos B, Rottiers R, Muggeo M et al. Glycemic index in the diet of European outpatients with type 1 diabetes: relations to glycated hemoglobin and serum lipids. *Am J Clin Nutr* 2001;73:574–81.
42. Liu S, Manson J, Stampfer M, Holmes M, Hu F, Hankinson S et al. Dietary glycemic load assessed by food frequency questionnaire in relation to plasma high-density-lipoprotein cholesterol and fasting plasma triacylglycerols in postmenopausal women. *Am J Clin Nutr* 2001;73:560–6.
43. Liu S, Willett W, Stampfer M, Hu F, Franz M, Sampson L et al. A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *Am J Clin Nutr* 2000;71:1455–61.
44. Jarvi A, Karlstrom B, Granfeldt Y, Bjorck I, Asp N, Vessby B. Improved glycemic control and lipid profile and normalized fibrinolytic activity on a low-glycemic index diet in type 2 diabetic patients. *Diab Care* 1999;22:10–18.
45. Luscombe N, Noakes M, Clifton P. Diets high and low in glycemic index versus high monounsaturated fat diets: effects on glucose and lipid metabolism in NIDDM. *Eur J Clin Nutr* 1999;53:473–8.
46. Rolls B, Hill J. *Carbohydrates and weight management*. Washington, DC: ILSI Press, 1998.
47. Franklin J, Caterson I. *Setting the record straight—the role of carbohydrates in weight control*. Sydney: GRDC & BRI Australia, 1999.
48. Yao M, Roberts S. Dietary energy density and weight regulation. *Nutr Rev* 2001;59:247–58.
49. Alfieri M, Pomerleau J, Grace D, Anderson L. Fiber intakes of normal weight, moderately obese and severely obese subjects. *Obes Res* 1995;3:541–7.
50. Raben A, Macdonald I, Astrup A. Replacement of dietary fat by sucrose or starch: effects of a 14d ad libitum energy intake, energy expenditure and body weight in formerly obese and never-obese subjects. *Int J Obes* 1997;21:846–59.

51. Brunner E, Wunsch H, Marmot M. What is an optimal diet? Relationship of macronutrient intake to obesity, glucose tolerance, lipoprotein cholesterol levels and the metabolic syndrome in the Whitehall II study. *Int J Obes* 2001;25:45–53.
52. Saris W, Astrup A, Prentice A, Zunft H, Formiguerra X, Verboetket-van de Venne W et al. Randomized controlled trial of changes in dietary carbohydrate/fat ratio and simple vs complex carbohydrates on body weight and blood lipids: the CARMEN study. *Int J Obes* 2000;24:1310–18.
53. Levine A, Billington C. Dietary fiber: does it affect food intake and body weight? In: Fernstrom J, Miller G, eds. *Appetite and body weight regulation: sugar, fat and macronutrient substitutes*. Boca Raton, FL: CRC Press, 1994.
54. Holt S, Brand Miller J. Particle size, satiety and the glycaemic response. *Eur J Clin Nutr* 1994;48:496–502.
55. Roberts D. High glycemic index foods, hunger and obesity: is there a connection? *Nutr Rev* 2000;58:163–9.
56. Astrup A, Ryan L, Grunwals G, Storgaard M, Saris W, Melanson E et al. The role of dietary fat in body fatness: evidence from a preliminary meta-analysis of ad libitum low-fat dietary intervention studies. *Br J Nutr* 2000;83:S25–S32.
57. Salmeron J, Manson J, Stampfer M, Colditz G, Wing A, Willett W. Dietary fiber, glycaemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* 1997;277:472–7.
58. Liu S, Manson J, Stampfer M, Hu F, Giovannucci E, Colditz G et al. A prospective study of whole-grain intake and risk of type 2 diabetes mellitus in US women. *Am J Pub Hlth* 2000;90:1409–15.
59. Brand Miller J. The importance of glycaemic index in diabetes. *Am J Clin Nutr* 1994;50(suppl.):747S–752S.
60. Clapp J. Effect of dietary carbohydrate on the glucose and insulin response to mixed caloric intake and exercise in both nonpregnant and pregnant women. *Diab Care* 1998;21(suppl. 2):B107–B112.
61. 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.
62. European Cancer Prevention Organisation Consensus Panel on Cereals and Cancer. Consensus statement on cereals, fibre and colorectal and breast cancers. *Eur J Cancer Prev* 1998;7(suppl. 2):S1–S2.
63. Jacobs D, Marquart L, Slavin J, Kushi L. Whole-grain intake and cancer: an expanded meta-analysis. *Nutr Cancer* 1998;20:85–96.
64. Slavin J, Martini M, Jacobs D, Marquart L. Plausible mechanisms for the protectiveness of whole grains. *Am J Clin Nutr* 1999;70(suppl.):459S–463S.

65. Slavin J, Marquart L, Jacobs D. Consumption of whole-grain foods and decreased risk of cancer: proposed mechanisms. *Cereal Foods World* 2000;45:54–8.
66. Slavin J. Mechanisms for the impact of whole grain foods on cancer risk. *J Am Coll Nutr* 2000;19:300S–307S.
67. Levi F, Pasche C, Lucchini F, Chatenoud L, Jacobs D, La Vecchia C. Refined and whole grain cereals and the risk of oral, oesophageal and laryngeal cancer. *Eur J Clin Nutr* 2000;54:487–9.
68. Chatenoud L, La Vecchia C, Franceschi A, Tavani A, Jacobs D, Parpinel M et al. Refined-cereal intake and risk of selected cancers in Italy. *Am J Clin Nutr* 1999;70:1107–10.
69. Giovannucci E, Stampfer M, Colditz G, Rimm E, Willett W. Relationship of diet to risk of colorectal adenoma in men. *J Nat Cancer Inst* 1992;84:91–8.
70. Cassidy A, Bingham S, Cummings J. Starch intake and colorectal cancer risk: an international comparison. *Br J Cancer* 1994;69:937–42.
71. Baghurst P, Baghurst K, Record S. Dietary fibre, non-starch polysaccharides and resistant starch: a review. *Food Aust* 1996;48:S1–S36.
72. Grasten S, Juntunen K, Poutanen K, Gylling H, Miettinen T, Mykkanen H. Rye bread improves bowel function and decreases concentrations of some compounds that are putative colon cancer risk markers in middle-aged women and men. *J Nutr* 2000;130:2215–21.
73. DeCosse J, Miller H, Lesser M. Effects of wheat fiber and vitamins C and E on rectal polyps in patients with familial adenomatous polyposis. *J Nat Cancer Inst* 1989;81:1290–7.
74. MacLennan R, Macrae F, Bain C, Battistutta D, Chapuis P, Gratten H et al. Randomized trial of intake of fat, fiber, and beta-carotene to prevent colorectal cancer. *J Nat Cancer Inst* 1995;87:1760–6.
75. Fuchs C, Giovannucci E, Colditz G, Hunter D, Stampfer M, Rosner B et al. Dietary fibre and the risk of colorectal cancer and adenoma in women. *New Engl J Med* 1999;340:169–76.
76. Alberts D, Martinez M, Roe D, Guillen-Rodriguez J, Marshall J, van Leeuwen B et al. Lack of effect of a high-fibre cereal supplement on the recurrence of colorectal adenomas. *New Engl J Med* 2000;342:1156–62.
77. Shatzkin A, Lanza E, Corle D, Lance P, Iber F, Caan B et al. Lack of effect of a low-fat, high-fiber diet on the recurrence of colorectal adenomas. *New Engl J Med* 2000;342:1149–55.
78. Asano T, McLeod RS. Dietary fibre for the prevention of colorectal adenomas and carcinomas. Cochrane Database of Systematic Reviews 2002. Issue 4.

79. Caygill C, Charlett A, Hill M. Relationship between intake of high-fibre foods and energy and the risk of cancer of the large bowel and breast. *Eur J Cancer Prev* 1998;7(suppl. 2):S11–S17.
80. Howe G, Hirohata T, Hislop T, Iscovich J, Yuan J, Katsouyanni E. Dietary factors and risk of breast cancer: combined analysis of 12 case-control studies. *J Nat Cancer Inst* 1990;82:561–9.
81. La Vecchia C, Chatenoud L. Fibres, whole-grain foods and breast and other cancers. *Eur J Cancer Prev* 1998;7(suppl. 2):S25–S28.
82. Saxe G, Rock C, Wicha M, Schottenfeld D. Diet and risk of breast cancer recurrence and survival. *Breast Cancer Research and Treatment* 1999;53:241–53.
83. Herbert J, Hurley T, Olendzki B, Teas J, Ma Y, Hampl J. Nutritional and socioeconomic factors in relation to prostate cancer mortality: a cross-national study. *J Nat Cancer Inst* 1998;90:1637–47.
84. McGlaughlin J, Gridley G, Block G, Winn D, Preston-Martin S, Schoenberg J et al. Dietary factors in oral and pharyngeal cancer. *J Nat Cancer Inst* 1988;80:1237–43.
85. Bosetti C, Negri E, Franceschi A, Conti E, Levi F, Tomel F et al. Risk factors for oral and pharyngeal cancer in women: a study from Italy and Switzerland. *Br J Cancer* 2000;82:204–7.
86. Cummings J, Bingham S, Heaton K, Eastwood M. Fecal weight, colon cancer risk, and dietary intake of nonstarch polysaccharides (dietary fiber). *Gastroenterology* 1992;103:1783–9.
87. Trowell H. Dietary fiber and hypothesis that it is a protective factor in certain disease. *Am J Clin Nutr* 1978;29:417–27.
88. Muller-Lissner S. Effect of wheat bran on weight of stool and gastrointestinal transit time: a meta-analysis. *BMJ* 1988;296:615–17.
89. Cummings J. Non-starch polysaccharides (dietary fibre) including bulk laxatives in constipation. In: Kamm M, Lennard-Jones J, eds. *Constipation*. Petersfield, UK: Wrightson Biomedical Publishing, 1994:307–14.
90. Aldoori W, Giovannucci E, Rockett H, Sampson L, Rimm E, Willett W. A prospective study of dietary fiber types and symptomatic diverticular disease. *J Nutr* 1998;128:714–19.
91. Baghurst K, Crawford D, Worsley A, Syrette J, Record S, Baghurst P. The Victorian Nutrition Survey: a profile of the energy, macronutrient and sodium intakes of the population. *Comm Hlth Stud* 1988;12:42–54.
92. Ascherio A, Rimm E, Giovannucci E, Colditz G, Rosner B, Willett W. A prospective study of nutritional factors and hypertension among US men. *Circulation* 1992;86:1475–84.

I.2 EAT PLENTY OF CEREALS (INCLUDING BREADS, RICE, PASTA AND NOODLES), PREFERABLY WHOLEGRAIN

93. Ascherio A, Hennekens C, Willett W, Sacks F, Rosner B, Manson J et al. Prospective study of nutritional factors, blood pressure and hypertension among US women. *Hypertens* 1996;27:1065–72.
94. Williams P. *The contribution of breakfast to the nutrition of Australians*. Kellogg Nutrition Symposium, 'Food, Mood and Performance'. Sydney: Kellogg (Aust) Pty Ltd, 1998:24–29.
95. Pearn J. Panis populi—bread and public health in Australia. *Aust NZ J Pub Hlth* 1998;22:282–5.
96. Ma J, Truswell A. Wernicke-Korsakoff syndrome in Sydney hospitals: before and after thiamine enrichment of flour. *Med J Aust* 1995;163:531–4.
97. Harper C, Sheedy D, Lara A, Garrick T, Hilton J, Raisanen J. Prevalence of Wernicke-Korsakoff syndrome in Australia: has thiamine fortification made a difference? *Med J Aust* 1998;168:542–5.
98. Department of Health and Family Services. *The Australian guide to healthy eating: background information for nutrition educators*. Canberra: DHFS, 1998.
99. National Health and Medical Research Council. *Recommended dietary intakes for use in Australia*. Canberra: Australian Government Publishing Service, 1991.
100. Brand J, Colagiuri S, Crossman S, Allen A, Roberts D, Truswell A. Low glycaemic index foods improve long term glycaemic control in NIDDM. *Diab Care* 1991;14:95–101.
101. National Health and Medical Research Council, *Dietary guidelines for Australians*. Canberra: Australian Government Publishing Service, 1992.
102. Andlauer W, Furst P. Antioxidant power of phytochemicals with special reference to cereals. *Cereal Foods World* 1998;43:356–60.

1.3 INCLUDE LEAN MEAT, FISH, POULTRY AND/OR ALTERNATIVES

Katrine Baghurst

TERMINOLOGY

Meat

Meat includes all or part of the carcass of any cattle, sheep, goat, buffalo, kangaroo, camel, deer, goat, pig or rabbit. For the purpose of this discussion, the term refers to the muscle component only; it excludes offal such as liver and kidney.

Red meat

For the purposes of this guideline, *red meat* refers to the muscle meat from cattle, sheep, goat and kangaroo. It does not include pork, ham or bacon; in other parts of the world—such as the United States, the United Kingdom and Europe—*red meat* includes pig meat.

Poultry

Poultry refers to chicken, duck, turkey and all other avian foods except eggs.

Alternatives

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

Anaemia

There are a number of forms of anaemia. Microcytic anaemia (referring to small red blood cells) is a deficiency of red blood cells or their haemoglobin; it is often, but not always, related to iron deficiency. Macrocytic anaemia (referring to large red blood cells) is prevalent in some groups (such as Indigenous Australians) and may in some cases be associated with deficiencies of other nutrients, especially folate and vitamin B₁₂.

Iron deficiency

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.

BACKGROUND

Food categories

Meats, fish, poultry and their alternatives contribute a number of important nutrients, some of which are marginal in the Australian diet. The foods in this food group are very valuable sources of protein as well as being a major source of a number of minerals and vitamins, such as iron, zinc, vitamin B₁₂ (see Table 1.3.1) and, in the case of fish, n-3 fats.

Red meats

Red meats are a valuable source of dietary protein and the best sources of bioavailable iron in the Australian diet. They provide substantial amounts of zinc and vitamin B₁₂ and the lean varieties provide from 2 to 5 grams per 100 grams of dietary fat, with almost equal contributions from saturated and mono-unsaturated fats and a small amount of polyunsaturated fat.

Pork and poultry

Pork and poultry contain amounts of protein equivalent to those in red meats—about 20 grams per 100 grams. They are also valuable sources of bioavailable iron and zinc, but their content of these minerals is less than half that of the red meats per unit weight. Their vitamin B₁₂ content is also substantially less (see Table 1.3.1). The fat content of lean pork and skinless chicken is in the same range as that for lean red meats; lean pork has equal amounts of saturated and mono-unsaturated fats but a higher proportion of polyunsaturated fats compared with lean red meats. Skinless chicken has a higher proportion of both mono-unsaturates and polyunsaturates compared with the other meats.

Fish

Fish contain amounts of protein equivalent to those in red meats, pork and poultry. They also provide bioavailable iron and zinc, but at markedly lower levels than red meats. In contrast, the vitamin B₁₂ level of fish is similar to that of red meat or even higher, depending on the species. Fish are also a valuable source of iodine, which is in marginal supply in some areas of Australia. The fat content of fish is variable (see Table 1.3.1), ranging from 1 per cent to 10 per

cent or more by weight for oily fish. Fish, particularly the oily fish, are a very rich source of n-3 polyunsaturated fats, which are also found in some other muscle meats but at very much lower levels. These n-3 fats have been shown to provide specific health benefits, notably in relation to brain development and function and cardiovascular health (see ‘Scientific basis’ in this guideline and Section 1.6).

Alternatives

A number of foods can provide some of the key nutrients found in meats, fish and poultry, among them eggs, liver and kidney, shellfish, and plant foods such as legumes, nuts and some seeds. These foods are generally good sources of protein but have highly variable amounts of bioavailable iron, zinc and vitamin B₁₂. Consumption of legumes, nuts and seeds is encouraged for everyone; for vegetarians, additional serves of these foods, together with cereals, can also contribute many of the nutrients provided by meats, poultry and fish in an omnivore diet.

Eggs

Eggs have slightly lower protein content than the muscle meats. They are a good source of vitamin B₁₂ and provide substantial amounts of iron and zinc, although the iron is not as bioavailable. They also contain substantial amounts of cholesterol, which might be important for some individuals, but they represent a valuable occasional protein alternative to muscle foods.

Shellfish

Shellfish have a nutrient profile similar to that of eggs, although the cholesterol content is variable, with prawns and squid having relatively high levels, mussels, crab and lobster being intermediate, and scallops having low levels.

Liver and kidneys

Liver and kidneys are also good protein sources, very high in bioavailable iron and zinc, and particularly high in vitamin B₁₂; they are, however, somewhat high in cholesterol.

Legumes, nuts and certain seeds

Legumes, nuts and certain seeds are also valuable sources of protein and, to a lesser degree, iron and zinc. However, the iron and zinc from plant sources are less bioavailable than they are from animal sources. Legumes, nuts and certain seeds, along with other plant foods, have been shown to offer specific health benefits (see Section 1.1) and their inclusion in the diet is recommended for everyone. They are particularly valuable in a vegetarian diet as an alternative source of protein and other important nutrients. Other health benefits are thought to relate to their glycaemic properties (legumes), their phytoestrogen content (soybeans) or their fatty acid profile (nuts and certain seeds). The plant-based alternatives to meat, fish and poultry do not naturally provide any vitamin B₁₂, but fortified products are available. For vegetarians, these foods, together

with cereal foods, can provide most (but not all) of the nutrients provided by meats, fish and poultry. Other key nutrients, such as vitamin B₁₂ and n-3 fatty acids, may need to be obtained through fortified foods or supplements.

Current intakes in Australia

The *Australian Guide to Healthy Eating*¹—which is based on the NHMRC's core food group model²—recommends one to one-and-a-half serves of meat, fish, poultry or alternatives each day for males aged 19–60 years, depending on the pattern of intake of other foods, and one to one-and-a-half serves for females aged 19–60 years. One-and-a-half serves a day are recommended in pregnancy and two during lactation. A sample serve equates to 65–100 grams of cooked meat or chicken; half a cup (cooked) of dried beans, lentils, chick peas, split peas or canned beans; 80–120 grams of cooked fish fillet; two small eggs; one-third of a cup of almonds or peanuts; or a quarter of a cup of sunflower or sesame seeds.

The *Australian Guide to Healthy Eating* recommends that red meat be eaten three to four times a week; less than this and high-iron replacement foods will be required. The guide adds that this is especially important for girls, women, vegetarians and athletes.

The 1995 National Nutrition Survey showed mean intakes of about 200g/day for men for all meat, poultry and game products and dishes and 116g for women but this included other food groups where they occurred in mixed dishes with meats as the major component. Mean muscle meat consumption was about 63g/day for men and 32g for women; poultry was 26g/day for men and 18g for women; fish was 8g/day for men and 5g for women; eggs were 10g/day for men and 6g for women; processed meats and sausages together averaged 22g/day for men and 10g for women and legumes and pulses averaged 12.2g/day for men and 7.5g for women.

Table I.3.1 Nutrient content per 100 grams of sample lean meats, fish, poultry and alternatives

	Energy (kJ)	Protein (g)	Iron (mg)	Zinc (mg)	Vitamin B₁₂ (µg)
Lean beef	450	21.6	2.40	3.6	2.50
Lean lamb	501	20.4	2.30	3.4	0.96
Lean pork	438	21.6	1.00	2.2	0.70
Fresh flathead	395	21.1	0.20	0.6	1.50
Canned red salmon	815	21.9	1.20	0.9	4.00
Skinless chicken	466	20.4	0.95	1.4	0.41
Liver—lamb	680	21.4	9.40	4.3	84.00
Eggs	632	13.2	1.80	0.9	1.10
Soybeans (dry-cooked)	537	13.5	2.20	1.6	—
Canned baked beans	285	4.6	1.60	0.5	—
Almonds	2455	20.0	3.50	3.6	—

	Total fat (g)	Saturated fat (g)	Mono- unsaturated (g)	Poly- unsaturated (g)	Total n-3 (g)
Lean beef	1.8	0.87	0.82	0.21	0.07
Lean lamb	4.2	1.35	1.41	0.34	0.13
Lean pork	1.7	0.50	0.51	0.36	0.04
Fresh flathead	1.0	0.36	0.29	0.52	0.43
Canned red salmon	12.0	2.21	2.46	2.69	2.50
Skinless chicken	3.3	0.92	1.37	0.39	0.04
Liver—lamb	7.5	2.20	2.00	1.30	1.13
Eggs	10.9	3.10	4.30	1.00	0.06
Soybeans (dry-cooked)	7.7	1.10	1.20	4.80	0.17
Canned baked beans	0.5	0.10	0.10	0.30	0.03
Almonds	55.3	3.55	36.05	13.10	—

— Zero.

Note: Figures are for raw meats.

Sources: National Nutrition Survey nutrient data file; Nuttab '95; NZ food database; T Spadek, Chemistry Centre, WA (fatty acids in soybean); reference 46.

SCIENTIFIC BASIS

Although this food group is a major provider of a number of important nutrients—protein, zinc, vitamin B₁₂ and, for fish, n-3 fatty acids—it is as a source of bioavailable iron that it plays its most unique public health role.

Iron

Low iron intakes are common in Australia. Low iron intakes—coupled with increased requirements among population subgroups such as women of childbearing age and in pregnancy—make iron deficiency a significant public health concern. Vegetarians and semi-vegetarians may also be at increased risk because of the higher intake needs resulting from the low bioavailability of plant-based iron sources. Low iron stores or iron deficiency without anaemia appear to be relatively common in Australian women.^{3,4} In adults, iron deficiency may be associated with reduced work capacity, less efficient response to exercise, and impaired immune function^{5,6}; it may also affect mood and cognitive performance.^{7,8}

In the early 1990s it was estimated that iron deficiency anaemias alone were associated with health care costs of some \$14 million annually in Australia.⁹ The additional costs related to the sequelae of the more common condition of iron deficiency have not been estimated.

As part of the haemoglobin in red blood cells, iron helps to transport oxygen around the body. As part of the enzymes of the electron transport chain, it is necessary for the production of energy from glucose, the main fuel for both the brain and the rest of the body. Iron is also a vital component of enzymes responsible for brain development and essential for the synthesis of key neurotransmitters required for normal brain function.¹⁰

There has been no recent national assessment of iron status in Australian adults. However, if the results obtained from two recent surveys of young women in Western Australia^{3,4} can be generalised to other women of this age, low iron stores or iron deficiency without anaemia could be relatively common in this population group (see Table 1.3.2).

Table I.3.2 Percentages of females with low iron status from two Western Australian studies^{3,4}

Subjects	Status	% of subjects
265 female university students aged 15–30 years ³	Iron deficiency	
	Serum ferritin <16mg/L	19.8
	Serum ferritin ≤12mg/L	12.5
	Serum ferritin ≤12mg/L and transferrin saturation <16%	7.2
	Anaemia	
	Hb<12g/dL	10.2
211 women aged 15–30 years ⁴	Hb<12g/dL serum ferritin ≤12mg/L and transferrin saturation <16%	4.5
	Low iron stores	
	Serum ferritin <30mg/L	42.7
	Iron deficiency	
	Serum ferritin <16mg/L	14.2

Note: In the second study, 1.4% of women in the total sample were anaemic.

The effects of anaemia and iron deficiency on brain development in infancy and very early childhood are well documented^{11–14}, but randomised trials are also producing evidence of effects of inadequate iron status on cognitive processes in both adolescence (in relation to verbal learning and memory)⁷ and adulthood (in relation to reasoning and perceptual organisation).⁸

Western mixed diets contain 1.0 to 1.4 milligrams of iron per 1000 kilojoules.¹⁵ Hence most adults will ingest over 10 milligrams of iron daily, which, theoretically, should be sufficient to meet the needs of most of the population¹⁶ but only a small, and variable, proportion (5–20 per cent) of dietary iron is absorbed. Haem iron, found in muscle meat, is approximately 25 per cent absorbed, and non-haem, found in plants, is about 15 per cent absorbed.¹⁷ Consumption of meat, fish or poultry increases absorption of haem iron and, even in relatively small amounts (50–75 grams) in a mixed meal, can increase absorption from the plant foods in the meal up to twofold.¹⁸ Non-tissue animal foods such as eggs and milk do not appear to enhance iron absorption and can decrease non-haem iron absorption.¹⁹ Addition of foods rich in vitamin C or fruit juices to a meal can also greatly increase the absorption of iron from that meal in a dose-dependent manner—a factor of five or greater.²⁰

Conversely, certain plant foods can contain inhibitors to absorption. Bothwell et al.²¹ have extensively reviewed the bioavailability of iron from various natural plant sources. Polyphenols (such as tannins), which are a component of all plant tissue, have an inhibitory action^{21,22}, the degree of inhibition correlating well with the polyphenol content of individual vegetables. Tea and, to a lesser extent, coffee also profoundly inhibit iron absorption by binding the iron to form insoluble compounds with tannins.²³ Whilst most alcoholic drinks appear to assist iron absorption, this does not apply to some red wines, because of the tannin

content.^{18,23} Phytates are also inhibitory and are present in substantial quantities in many cereals and legumes; the quantitative relation between these compounds and iron absorption is, however, less clear cut.²¹

Calcium supplements have been shown to inhibit iron absorption²⁴, and some practitioners recommend consumption of primary iron and calcium sources on different meal occasions to optimise absorption. However, addition of milk to a cereal-based meal has been shown to have no effect on iron absorption in a group of young women²⁵, and long-term calcium supplementation has been shown not to lower plasma ferritin concentration in human subjects.²⁶

Where the plant-based foods from this category are preferred, care is therefore necessary, so that iron intake is sufficient to overcome the lower bioavailability and the inhibitory components in plant foods. A wide range of iron-fortified food products are now available—with varying, but often unknown, iron bioavailability. It is equally important to combine foods in such a way as to maximise bioavailability.

Recommended intakes and current consumption of iron

The current Australian recommended dietary intakes for iron¹⁶ are 7 milligrams a day for adult males, 12–16mg/day for females aged 19–54 years, and 5–7mg/day for females over 54 years. An additional 10–20mg is recommended during pregnancy, but no additional iron is recommended in lactation. The recent US review of recommended dietary allowances (RDAs) set an ‘estimated average requirement’ (EAR) for iron of 6.0mg/day for men, 8.1mg/day for females aged 19–50 years, and 5.0mg/day for females aged 51 years and over.²⁷ This resulted in an RDA ($RDA = EAR + 2SD_{EAR}$) of 8mg/day for males of all ages and females aged 51 years and over and 18mg/day for females aged 19–50 years. The difference between the EAR and the RDA was much greater for females aged 19–50 years (more than a doubling) because of the substantially greater variance in the EAR for this group compared with other age and gender groups. The US RDA for women aged 19–50 years is thus some 50 per cent higher than the lower end of the Australian recommended dietary intake range for this age and gender group (12–16mg) and some 2mg/day higher than the upper end of the current Australian RDI range.

In the revised US dietary reference intakes, a higher iron intake figure (just under double that for omnivores) has been set for vegetarians in recognition of the lower bioavailability of plant iron sources. For adult vegetarian males, the RDA is 14 mg/day and for pre-menopausal vegetarian females it is 33mg/day.

A CSIRO analysis²⁸ has shown that in the 1995 National Nutrition Survey²⁹ two-thirds (66 per cent) of females aged 19–44 years consumed less than the lower end of the range (12mg/day) of the Australian RDI for iron on the day of the survey, a proportion similar to the findings of the 1983 National Dietary Survey.³⁰

The CSIRO analysis of the National Nutrition Survey also showed that red meat is a significant source of iron in the Australian diet, contributing 14 per cent of total dietary iron in adults and 52 per cent of the haem iron intake.²⁸ Other meats and

fish and poultry contributed a further 7 per cent of dietary iron. Legumes and other vegetables provided 14 per cent.

Zinc

Zinc is important in a number of major metabolic processes, among them the synthesis of protein and nucleic acid and the synthesis and action of insulin. It is involved in immune function and cell growth and repair. The long-term effects of mild zinc deficiency are unclear but may include delayed wound healing, impaired immune function, and problems with taste and smell acuity.³¹

Strong homeostatic mechanisms regulate zinc, and this, together with a lack of sensitive indicators of zinc status, means it is difficult to determine the prevalence of zinc deficiency in a community and to set recommended intakes. Furthermore, the bioavailability of zinc varies markedly between foods and, as with iron, is affected by the composition of the diet. Zinc from animal sources, including eggs, is generally better absorbed than zinc from plant foods. For example, 21–26 per cent of the zinc in beef is absorbed, compared with 11–14 per cent of the zinc in wholemeal bread.³² Absorption of zinc is reduced by phytate in plant foods such as peanuts and soybeans.³³ While calcium and iron can potentially reduce zinc absorption, the effect caused by food intake of these minerals is likely to be relatively small.³⁴

Recommended and current intakes of zinc

The Australian recommended dietary intake for zinc¹⁶ is 12 milligrams a day for both male and female adults, with an additional 4mg/day recommended during pregnancy and an extra 6mg/day in lactation. The recent revision of the US recommended dietary allowances concluded that the estimated average requirement of zinc for men was 9.4mg/day and for females 6.8mg/day²⁷, with a resulting RDA of 11mg/day for men and 8mg/day for women. For pregnancy, an additional 5mg/day was added to the RDA for females aged 14–18 years (a total of 13mg/day) and an extra 3mg/day for females aged over 18 years (a total of 11mg/day). For lactation, the RDA was set at 14mg/day for females aged 18 years and less and 12mg/day for those over 18 years old.

The CSIRO analysis of the 1995 National Nutrition Survey²⁸ showed that nearly three-quarters of adolescent girls and women had zinc intakes below the RDI of 12mg/day on the day of the survey; 45 per cent of adult males and 63 per cent of elderly males had zinc intakes below the RDI.

It is of interest that the new US recommended dietary allowance for zinc for women (8mg/day) is one-third lower than the current Australian recommended dietary intake for women (12mg/day), which was set in 1991. The figure for men is approximately the same (11mg in the United States and 12mg in Australia). It is therefore possible that the large proportion of women in Australia estimated to be at risk of low zinc intake might, in part, be the consequence of an overestimate of requirements based on the limited data available at the time of setting the Australian RDIs.

The National Nutrition Survey²⁹ results showed that the 'meat, poultry and game products and dishes' category provided 32 per cent of the zinc in the diet of adult females and 39 per cent in adult males. Muscle meats provided 15 per cent; fish and seafood provided 4 per cent. The proportion of the population recording low zinc intakes in the survey was inversely related to red meat consumption on the day of the survey.²⁸

Vitamin B₁₂

Vitamin B₁₂ plays an important biochemical role in the maintenance of myelin in the nervous system and, in conjunction with folate, in the synthesis of DNA. CSIRO research has demonstrated the importance of vitamin B₁₂ in maintaining genetic stability: chromosome damage was shown to be lower with higher plasma vitamin B₁₂ levels and lower plasma homocysteine levels. Supplementation with folate and vitamin B₁₂ (at 3.5 to 10 times the recommended dietary intake) can also reduce such chromosome damage.³⁵ It is not clear, however, what the overall health implications are in relation to the associations seen between vitamin B₁₂ and genetic damage.

Another CSIRO study demonstrated that non-vegetarian males aged 20–40 years had a significantly lower rate of genetic damage than vegetarian males of the same age.³⁶ This is thought to be a result of the protective effects of vitamin B₁₂ in the diet of non-vegetarians.

The main forms of vitamin B₁₂ available to humans come from animal products in which the vitamin has accumulated from bacterial synthesis. Although occasional contamination of soil or water with microbes that produce vitamin B₁₂ occurs, plant foods are usually devoid of the active form of the vitamin. Because of the importance of animal foods as a source of this vitamin, dietary vitamin B₁₂ deficiency can be a problem in strict vegetarians who exclude all sources of animal products such as eggs and dairy products and/or do not take a B₁₂ supplement or consume sufficient amounts of B₁₂-fortified plant products.³⁷

The most prevalent deficiency of vitamin B₁₂ is sub-clinical deficiency, recognised by changes in biochemical levels in the blood. The normal serum vitamin B₁₂ is usually taken as 200 picograms per millilitre (or 150 picomoles per litre). Low vitamin B₁₂ levels (as well as low folate and low B₆) have been shown to correlate with raised plasma homocysteine^{38,39}, which is a risk factor for cardiovascular disease. However, the importance of dietary intake of vitamin B₁₂ in prevention (or correction) of raised plasma homocysteinuria is not clear.

In one Melbourne study, Mann et al. measured serum vitamin B₁₂, homocysteine and folate in healthy men aged 20–55 years⁴⁰ eating a wide range of diets, from high-meat to vegan, and found a strong negative correlation ($r = -0.37$) between serum vitamin B₁₂ and plasma homocysteine in the combined subjects of the four groups. All meat-eaters in the study had serum vitamin B₁₂ in the normal range (200–1100pg/ml), but 23 per cent of the lacto-ovo-vegetarians and 65 per cent of the vegans had serum vitamin B₁₂ below 200pg/ml.⁴¹ It is not certain how

representative these figures might be of the sub-populations involved since they were based on 18 vegans, 43 lacto-ovo-vegetarians, 60 meat-eaters and 18 high meat-eaters, but they do indicate marked differences across eating styles. If homocysteine levels are higher in vegetarians or vegans as a result of lower B₁₂ levels, it is unclear whether normalising this would bring them cardiovascular benefits additional to those sometimes seen with vegetarian diets.

Low vitamin B₁₂ status has also been associated with impaired cognitive function in terms of fluid intelligence in adolescents who had previously been long-term vegans⁴² and in terms of memory performance in adult females from the Australian population.⁴³

Recommended and current intakes of vitamin B₁₂

The Australian recommended dietary intake for vitamin B₁₂ is 2.0 micrograms a day for males and females of all ages, with an additional 1.0mg/day in pregnancy and 0.5mg/day in lactation.¹⁷ The recent revision of the US recommended dietary allowances⁴⁴ established an estimated average requirement of 2mg /day for adult males and females of all ages, with an RDA of 2.4mg /day for all adults. However, for people over 51 years of age, it is recommended that most of the 2.4mg be obtained by consumption of foods fortified with B₁₂ or a B₁₂-containing supplement because of concerns about decreasing ability to absorb B₁₂ from foods as people age.

An analysis of the National Nutrition Survey²⁸ using the vitamin B₁₂ food database from the United States, gave an average estimated intake of 6mg/day for adult males and 3.9mg/day for adult females.

Protein

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

Proteins vary in their digestibility. The protein from meats, fish and poultry is highly digestible (90 per cent or more); this compares with a digestibility of 78 per cent in beans and 86 per cent in whole wheat.⁴⁵

Net protein utilisation⁴⁶ is generally higher for animal protein sources (NPU 0.75–0.8) compared with many, but not all, plant foods (NPU 0.5–0.6).

Recommended and current intakes of protein

In Australia the recommended dietary intake of protein for adults is based on a value of 0.75 grams per kilogram per day.¹⁶ For men of all ages 55 grams a day is recommended and for females 45 grams a day. In pregnancy an additional 6 grams a day is recommended and in lactation an additional 16 grams a day. The Australian RDI was developed in the late 1980s. The new US recommended

dietary allowances⁴⁷ are however, very similar at 0.08g/kg/day for men and women which equates on average to 56g/day for men and 46g/day for women.

According to the 1995 National Nutrition Survey²⁹, males were consuming, on average, 109 grams of protein a day and females 74 grams a day. A CSIRO analysis²⁸ of the survey found that red meat was the primary source of protein in the Australian diet, providing a mean of 20 per cent of the protein in the diets of adult Australians.

Total and saturated fats

Meats are often perceived as a major source of dietary fat and saturated fat. However, although some individual cuts or products can be relatively high in fat, an analysis of the 1995 National Nutrition Survey showed that meats do not contribute as much fat as is commonly believed. For instance, the analysis showed that red meat per se contributes an average of only 6 per cent of the total fat in adults' diets, 9 per cent of the saturated fat, 12 per cent of the unsaturated fats (mainly mono-unsaturated), and 17 per cent of the cholesterol.²⁸ Changes in both the meat supply and consumer preferences in Australia are affecting the contribution of meats to fat and saturated fat intakes.

Excess dietary fat (and saturated fat) intake has been linked to a number of adverse health outcomes, including cardiovascular and diabetic complications (see Section 1.6). However, the confounding of fat intake with the intake of certain components of this food group, notably meats, has led to some confusion in interpretation of epidemiological data linking dietary components to chronic disease outcomes, particularly in relation to cancer. In the United States, where much of the epidemiological research data comes from, the fat content of meat is considerably higher than in Australia^{48–50}, and meats contribute more markedly to overall fat and saturated fat intake.

An important 1997 publication claimed a role for fat and meat in colon cancer⁵¹, but the recent European Conference on Nutrition and Cancer⁵² concluded there was no association between fresh red meat and colorectal cancer. In addition, three recent Australian reviews of the cancer epidemiology literature have largely exonerated fresh meats per se from a role in cancer causation and, in particular, colon cancer. An expert panel review of the role of red meat in colon cancer concluded that the balance of epidemiological evidence indicates that prevailing levels of lean red meat consumption in Australia are not linked with the development of cancer.⁵³ This conclusion is consistent with that of the National Health and Medical Research Council concerning diet and colorectal cancer—which recommends a reduction in total fat intake but makes no recommendation about meat intake⁵⁴—and with that of an earlier review of red meat and various cancers.⁵⁵ The Cancer Council of Australia's National Cancer Prevention Policy 2001–2003 also supports this view.⁵⁶ Recent research in Europe suggests, however, that caution may still be necessary in connection with certain food-preparation techniques and the consumption of cured and smoked meats⁵²,

although the Cancer Council of Australia concluded that there was insufficient evidence at present to support a causal relationship.⁵⁶

An expert review of red meat and health also concluded that diets rich in lean red meat could still be low in fat and saturated fat and not adversely affect plasma cholesterol levels and that lean red meat could be included in management strategies for the prevention and treatment of obesity.⁵⁷ The conclusions of the review concur with nutrition statements from the National Heart Foundation of Australia and have the support of the Dietitians Association of Australia as a useful summary of the contribution of red meat to healthy eating.

n-3 fats

Found predominantly in fish, n-3 fats appear to have a number of beneficial actions, notably in relation to brain development and function and cardiovascular health. (The role of n-3 fats in the diet is discussed in more detail in Section 1.6.) Australians' intake of long-chain n-3 fatty acids has been estimated to be less than 200 milligrams a day (an average of about 100mg)⁵⁸, and most authorities recommend an intake of 214–650mg/day.⁵⁹ Fish and seafood are by far the richest sources of n-3 fats. However, Ollis et al.⁶⁰ found in a study of 83 healthy Australian adults that meat was also a major contributor to the dietary intake of very long chain n-3 fats—contributing 29 per cent of the total. They attributed this to what they called the 'relatively high' meat intake of the group, which averaged 164 grams a day. The principle n-3 fatty acid from vegetables is alpha-linolenic acid. Vegetarians have significantly lower plasma and platelet n-3 levels^{61,62} since alpha-linolenic acid is not as effective a source of long-chain n-3 fatty acids as is the direct consumption of eicosapentaenoic acid (EPA), docosapentaenoic acid (DPA) and decosahexaenoic acid (DHA)⁶² from fish and meat. There is, however, no evidence that this results in higher cardiovascular risk in vegetarians: other beneficial dietary factors might offset the low n-3 levels.

Special groups

Adolescent girls and women

Menstrual loss virtually doubles the iron requirement of females compared with males.¹⁶ Adolescents have the added demands of growth. Iron balance in these circumstances is problematic and requires a substantial food intake, as well as an appropriate composition of meals. The National Nutrition Survey data²⁹ suggest that some females may be limiting their overall food intake, possibly because of concerns about body weight. Increased activity would allow greater food intake, thus increasing the likelihood of adequate iron intake whilst maintaining a desirable body weight.

Pregnancy

Pregnancy places females at higher risk of inadequate iron and zinc intake because of their increased needs and the potential for morning sickness to reduce nutrient availability. There is, however, some evidence that iron absorption increases during pregnancy. Pregnant vegetarians are at additional risk because of the low iron bioavailability in plant foods.⁶³

For adults, each pregnancy represents a major drain on iron reserves. Women who have had several children over a relatively short period are at high risk of iron depletion, especially if iron supplementation during their pregnancies was inadequate. Infants born to women with low iron stores will themselves have low stores and, if breastfed for prolonged periods, will be more likely to develop anaemia. Further, low iron status in early pregnancy is more likely to result in premature birth and low birthweight.^{64,65}

Low maternal serum zinc levels have been associated with congenital malformations, prematurity, foetal growth retardation (leading to low-birthweight babies) and maternal morbidity.³¹ However, the results have not always been consistent.

Vegetarians

With the exception of vitamin B₁₂, a balanced vegetarian diet can be adequate in all nutrients; indeed, some vegetarian communities have been shown to have health advantages over the general population—notably in the cardiovascular area, with reductions in risk factors such as plasma cholesterol, antioxidant status, clotting factors and blood pressure.^{66–76} It is thus possible to have a healthy diet without using foods derived from animal sources. There are, however, several micro-nutrients for which meat, fish and poultry are the dominant and most bioavailable source and, as discussed, care needs to be taken if these foods are excluded. The American Dietetic Association recommends that vegetarians regularly consume B₁₂-fortified food products. Protein from diverse plant sources—legumes, nuts, cereals or dairy (if eaten)—should be included to attain the appropriate mix of amino acids and give variety.

Until recently legumes have not been commonly eaten in Australia, but they are a valuable source of protein, fibre and micro-nutrients, not only for vegetarians but also for the wider community. Included in the legume category are beans such as soybeans, kidney beans, broad beans and haricot beans, as well as mature dried peas, lentils and chick peas, and foods made from this produce; for example, hummus, falafel, bean burrito, soy and lentil burgers, soy schnitzels, vegetarian sausages, soy slices, nutmeat, textured vegetable protein, baked beans and tofu. The evidence for the potential health benefits of legumes—and in particular soybeans, which contain compounds called isoflavones—has recently been reviewed by Messina.⁷⁷ Isoflavones, and in some instances soy foods themselves, have been investigated in relation to a number of potential health benefits, including cholesterol reduction, improved vascular health, preservation of bone-mineral density, lower incidence of certain cancers (notably prostate and

breast) and reduced menopausal symptoms. They show potential for benefit, but more research is needed to determine the levels needed to produce health benefits, their long-term efficacy, and the relative effect of isoflavones themselves, as opposed to soy-based foods.

Nuts and certain seeds also provide some protein and other nutrients and can help improve the unsaturated–saturated fat ratio in the diet. Because of their fatty acid profile, nut consumption has been investigated in relation to the potential to promote heart health. Several large prospective cohort studies have examined the relationship between nut consumption and the risk of coronary heart disease, and all found an inverse relationship.^{78–80} These findings and related epidemiological and clinical studies have been reviewed by Sabate⁸¹ and Hu and Stampfer.⁸² Nuts and seeds are, however, energy dense, so should be consumed in moderate amounts.

Athletes

Recent heightened interest in the relationship between iron status and athletes' performance has revealed an increased requirement for iron in that group, mainly because of increased intestinal losses.^{83,84} Because of their overall requirements, female athletes are particularly vulnerable.

PRACTICAL ASPECTS OF THIS GUIDELINE

This guideline specifically refers to the inclusion of lean cuts of meat and poultry. Some meats and some meat and poultry products or dishes that are popular in Australia—such as pies; sausages; crumbed and fried meats, fish or poultry; and mettwurst and salami—can contain significant amounts of saturated fat, either from the ingredients themselves (including non-meat components such as pastry or fillers) or from added cooking fats. These foods can be included occasionally in a balanced diet, but care does need to be taken with the rest of the diet, so as to avoid overconsumption of saturated fats. In addition, these types of products do not provide the same level of iron, zinc and Vitamin B₁₂ as the lean cuts. Selection of lean mince and removal of visible fat from meat and poultry cuts before cooking can also help to limit fat intake.

Whilst consumption of a variety of foods from this food group is encouraged, the *Australian Guide to Healthy Eating*¹ recommends inclusion of red meat three to four times a week; otherwise, high-iron replacement foods will be needed. This is especially the case for females and athletes. One Australian study³ showed that in young female students an intake of 1.6 milligrams of haem iron a day (the amount in about 100 grams of lean beef) reduced the odds of having low iron stores in the body by 60 per cent. There was no relationship between iron status and total iron intake.

Inclusion of two to three meals a week of fish high in n-3 fats—pilchards, sardines, salmon, tuna, herring mackerel, and so on—has also been recommended by some health authorities. However, with limited fish supplies,

this recommendation may not be attainable, or sustainable, at a population level, and n-3 fatty acid intake may have to be achieved through foods fortified with n-3 fats, in addition to natural fish sources.

Vegetarians should choose from a variety of legumes, nuts and seeds to obtain protein, iron and zinc. Wholegrain or wholemeal cereals are also good sources of zinc and iron, and supplemented varieties are available. Drinking fruit juice or eating fruit at the same meal increases absorption of iron and zinc.

RELATIONSHIP TO OTHER GUIDELINES

Earlier editions of the *Dietary Guidelines for Australians* included a guideline encouraging the consumption of ‘iron-rich foods’. In this revision—to more clearly define the concept of variety, to provide advice consistent with the *Australian Guide to Healthy Eating*, and to take a more consistent food-based approach—this guideline has been replaced by one encouraging inclusion of lean meats and fish, poultry or their alternatives, with an emphasis on these foods’ value as a source of dietary iron, zinc and B₁₂ as well as protein.

Limit saturated fat and moderate total fat intake

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

Prevent weight gain: be physically active and eat according to your energy needs

Obesity is increasing in many countries. Although many genetic, environmental and lifestyle factors contribute to this, dietary fat intake can also be a major factor in the development of obesity through its effect on the energy density of the diet.⁸⁵ A CSIRO analysis of the National Nutrition Survey showed a high correlation between energy density and fat content of the diet on the day of survey, with dietary fat (both grams per day and percentage of fat) being a major determinant of energy density. Choosing low-fat varieties and low-fat cooking techniques is therefore encouraged.

Care for your food: prepare and store it safely

Illness caused by food-borne bacteria is a public health concern. All foods are potential vectors of pathogens. In Australia the risk of food-borne illness in primary food industries is managed across the food chain, with industry, government and consumers sharing responsibility for the delivery of microbiologically safe products. Nevertheless, some foods from the meat, fish and poultry group have been implicated in outbreaks of food-borne disease^{86,87}, and constant vigilance is required.

CONCLUSION

Inclusion of lean meats, fish, poultry and alternatives in the daily diet will help to ensure adequate iron, zinc and vitamin B₁₂ intake as well as providing a valuable source of protein. Lean red meats are a particularly valuable source of iron, zinc and B₁₂, and fish is a particularly good source of omega-3 fats. Whilst well planned vegetarian diets can provide both iron and zinc in adequate amounts, care needs to be taken to ensure intake of adequate iron and zinc from the less bioavailable plant sources and particular attention has to be paid to alternative vitamin B₁₂ sources.

EVIDENCE

The scientific rationale for this guideline is based on a variety of evidence sources among them the following:

- two well-designed randomised controlled trials (Level II evidence) relating to iron supplementation and cognition (reference 7, 12)
- a meta-analysis of case-control and cohort studies (Level III evidence) assessing the effects of red meat on cancer (reference 55), Level III studies of iron deficiency on cognition (references 8, 13), of vitamin B₁₂ on cognition (reference 43), of red meat consumption on cardiovascular disease risk factors (reference 69), of nuts on cardiovascular disease (references 78, 80-82), and of various foods, drinks or nutrients on iron or zinc bioavailability and absorption (references 20, 23 to 26, 32 and 34) or diet on homocysteine status (references 38 to 40).

Evidence was also obtained from a number of cross-sectional population studies, as well as human experimentation relating to bioavailability and nutrient requirements and intakes, and expert reviews of selected issues.

REFERENCES

1. Children's Health Development Foundation & Deakin University. *The Australian guide to healthy eating*. Canberra: AGPS, 1998.
2. Cashel K, Jefferson S. *The core food groups. the scientific basis for developing nutrition education tools*. Canberra: AGPS, 1995.
3. Rangan AM, Blight GD, Binns CW. Factors affecting iron status in 15–30 year old female students. *Asia Pacific J Clin Nutr* 1997;6(4):291–5.
4. Sadler S, Blight G. Iron status and dietary iron intake of young women. *Proc Nutr Soc Aust* 1996;20:216.
5. Dallmann PR. Iron deficiency: does it matter? *J Int Med* 1982;226:367–72.
6. Sherman AR. Zinc, copper and iron nutriture and immunity. *J Nutr* 1992;122:604–9.

7. Bruner AB, Joffe A, Duggan AK, Casella JF, Brandt J. Randomised study of cognitive effects of iron supplementation in non-anaemic, iron-deficient adolescents. *Lancet* 1996;348:992–6.
8. Patterson AJ. Iron deficiency in Australian women: development, implications and treatment. PhD thesis. University of Newcastle, NSW, 1999.
9. Australian Institute of Health and Welfare. *Australia's food and nutrition*. Canberra: AGPS, 1994.
10. Shoham S, Glinka Y, Tanne Z, Youdim MBH. Brain iron: function and dysfunction in relation to cognitive processes. In: Hallberg L, Asp NG, eds. *Iron nutrition in health and disease*. London: John Libbey & Co., 1996:205–17.
11. Walter T, Kovalsky SJ, Stekel A. Effect of a mild iron deficiency on infant mental development scores. *J Paediatr* 1983;102:519–22.
12. Lozoff B, Brittenham GM, Wolf AW, McClish DK, Kuhnert PM, Jimenez E et al. Iron deficiency anaemia and iron therapy effects on infant development test performance. *Paediatrics* 1987;79:981–95.
13. De Andraca I, Walter T, Castillo M, Pino P, Rivera P, Cobo C. Iron deficiency anaemia and its effects upon psychological development at preschool age: a longitudinal study. *Nestle Foundation annual report*, 1990;53–62.
14. Lozoff B, Jimenez E, Wolf AW. Long term developmental outcome of infants with iron deficiency. *New Engl J Med* 1991;325(10):687–94.
15. UN Food and Agriculture Organization. *Apparent consumption data: iron in western diets*. Rome: FAO.
16. National Health and Medical Research Council. *Recommended dietary intakes for use in Australia*. Canberra: AGPS, 1991.
17. Hulten L, Gramatkowski E, Gleerup A, Halberg L. Iron absorption from the whole diet: relation to meal composition, iron requirements and iron stores. *Eur J Clin Nutr* 1995;49:794–808.
18. Hurrel LRF. Bioavailability of different iron compounds used to fortify formulas and cereals: technological problems. In: Stekel A, ed. *Iron nutrition in infancy and childhood*. New York: Raven Press, 1984:158–65.
19. Rossander L, Hallberg L, Bjorn-Rasmussen E. Absorption of iron from breakfast meals. *Am J Clin Nutr* 1979;32:2484–9.
20. Ballot D, Baynes RD, Bothwell TH, Gillooly M, MacFarlane BJ, MacPhail AP et al. The effects of fruit juices and fruits on the absorption of iron from a rice meal. *Brit J Nutr* 1987;57:331–43.
21. Bothwell TH, Baynes RD, MacFarlane BJ, Macphail AP. Nutritional iron requirements and food iron absorption. *J Int Med* 1989;226:357–65.

22. Brune M, Rossander L, Hallberg L. Iron absorption and phenolic compounds: importance of different phenolic structures. *Eur J Clin Nutr* 1989;43:547–58.
23. Hallberg L, Rossander L. Effect of different drinks on the absorption of non-heme iron from composite meals. *Hum Nutr: Appl Nutr* 1982;36a:116–23.
24. Cook JD, Dassenko SA, Whitaker P. Calcium supplementation: effect on iron absorption *Am J Clin Nutr* 1991;53:106–11.
25. Kretsch MJ, Keys WR, Shah AG. Milk's effect on the bioavailability of iron from cereal-based diets in young women by use of in vitro and in vivo methods. *Am J Clin Nutr* 1990;52:373–8.
26. Minihaane AM, Fairweather-Tait SJ. Effect of calcium supplementation on daily nonheme iron absorption and long term iron status. *Am J Clin Nutr* 1998;68(1):96–102.
27. Institute of Medicine. *Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium and zinc*. Washington, DC: National Academy Press, 2000:9-1–9-78.
28. Baghurst KI, Record SJ, Leppard P. Red meat consumption in Australia: intakes, nutrient contribution and changes over time. *Aust J Nutr Diet* 2000;57(4):S1–S36.
29. Australian Bureau of Statistics. *National Nutrition Survey: nutrient intakes and physical measurements, Australia, 1995*. Cat. no. 4805.0. Canberra: ABS, 1998.
30. Department of Community Services and Health. *National Dietary Survey of Adults: 1983. No 2. Nutrient intakes*. Canberra: Australian Government Publishing Service, 1987.
31. King JC, Keen CL. Zinc. In: Shils ME, Olson JA, Shike M, eds. *Modern nutrition in health and disease*. Philadelphia PA: Lea and Febiger, 1994:214–30.
32. Sandstrom B, Kivisto B, Cederblad A. Absorption of zinc from soy protein meals in humans. *Nutr* 1987;117:321–7.
33. Gibson RS. Content and bioavailability of trace elements in vegetarian diets. *Am J Clin Nutr* 1994;59:1223S–1232S.
34. Wood R, Zheng J. Calcium supplementation reduces intestinal zinc absorption and balance in humans. *FASEB Journal* 1995;9:A1640.
35. Fenech M, Aitken C, Rinaldi J. Folate, vitamin B₁₂, homocysteine status and DNA damage in young Australian adults. *Carcinogenesis* 1998;19(7):1163–71.
36. Fenech M, Rinaldi J. A comparison of lymphocyte micronuclei and plasma micronutrients in vegetarians and non-vegetarians. *Carcinogenesis* 1995;16(2):223–30.

37. Immerman AM. Vitamin B-12 status on a vegetarian diet: a critical review. *World Rev Nutr and Diet* 1981;37:38–54.
38. Verhoef P, Stampfer MJ, Buring JE, Gaziano JM, Allen RH, Stabler SP et al. Homocysteine metabolism and risk of myocardial infarction: relation with vitamins B₆ and B₁₂ and folate. *Am J Epid* 1996;143:845–59.
39. Ubbink JB, van der Merwe A, Vermaak WJH, Delport R. Hyperhomocysteinuria and the response to vitamin supplements. *Clin Investig* 1993;71:993–8.
40. Mann NJ, Li D, Sinclair AF, Dudman NPB, Guo XW, Wilson AK et al. The effect of diet on plasma homocysteine concentrations in healthy male subjects. *Eur J Clin Nutr* 1999;53:895–9.
41. Li D, Sinclair AJ, Mann NJ, Turner A, Ball MJ. Selected micro-nutrient intake and status in men with differing meat intakes, vegetarians and vegans. *Asia Pac J Clin Nutr* 2000;9:18–23.
42. Louwman MWJ, van Dusseldorp M, van de Vijver FJ, Thomas CM, Schneede J, Ueland PM et.al. Signs of impaired cognitive function in adolescents with marginal cobalamin status. *Am J Clin Nutr* 2000;72:762–9.
43. Bryan J, Calvaresi E, Hughes D. The effect of short term folate, B₁₂ and B₆ supplementation and dietary intake on cognition and mood in women. *J Geront: Psych Sci* 2001 (in press).
44. Institute of Medicine. *Dietary reference intakes for thiamine, riboflavin, niacin, vitamin B₆, folate, vitamin B₁₂, pantothenic acid, biotin and choline*. Washington DC: National Academy Press, 1998;306–56.
45. Bhutta ZA. Protein: digestibility and availability. In: Sadler MJ, Strain JJ, Caballero B, eds. *Encyclopedia of human nutrition*. San Diego: Academic Press, 1999:1646–54.
46. Bender A. *Meat and meat products in human nutrition in developing countries*. Food and Nutrition paper 53. Rome: Food Policy and Nutrition Division, 1992.
47. Institute of Medicine. *Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids*. Washington, DC: The National Academies Press, 2002; 465–608.
48. USDA Nutrient Database for Standard Reference. <www.nal.usda.gov/fnic/cgi-bin/nut_search.pl> February 2002.
49. Sadler MF, Lewis JL, Buick DR. Composition of trim lamb. *Food Australia* 1993;45:S3–S12.
50. Watson MA, Mann NJ, Sinclair AJ, O'Dea K. Fat content of untrimmed beef and lamb cuts. *Food Australia* 1992;44:511–14.
51. 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.

52. Riboli E. Meat, processed meat and colorectal cancer. Presentation at European Conference on Nutrition and Cancer. Lyon, France, 2001.
53. Truswell AS. Report of an expert workshop on meat intake and colorectal cancer risk convened in December 1998 in Adelaide, South Australia. *Eur J Cancer Prev* 1999;8:175–81.
54. National Health and Medical Research Council. *Guidelines for the prevention, early detection and management of colorectal cancer: a guide for patients, their families and friends*. Canberra: NHMRC, 1999.
55. Baghurst PA. Does red meat cause cancer? *Aust J Nutr Diet* 1997;54(4):S1–S44.
56. Cancer Council of Australia. *National Cancer Prevention Policy 2001–2003*. Sydney: Cancer Council of Australia, 2001.
57. Expert Panel on Red Meat and Health. *The role of red meat in a healthy diet*. Sydney: Meat and Livestock Australia, 2001.
58. Sinclair AJ, Vingrys AJ. Estimation of the long chain n-3 fatty acid status of Australians. *Proc Nutr Soc Aust* 1998;22:196.
59. Simpopoulos AP, Leaf A, Salem Jnr N. Workshop statement on the essentiality of and recommended dietary intakes for omega 6 and omega 3 fatty acids. *Prostaglandins, leucotrienes and essential fatty acids* 2000;63:119–21.
60. Ollis TE, Meyer B, Howe PRC. Food sources and intakes of omega-6 and omega-3 polyunsaturated fatty acids consumed by adults in the Illawarra region of NSW. *Proc 2nd South West Pacific Nutrition and Dietetic Conference*. Auckland, New Zealand, 1999.
61. Conquer JA, Holub BJ. Dietary docosaehaenoic acid as a source of eicosapentaenoic acid in vegetarians and omnivores. *Lipids* 1997;32:341–5.
62. Li D, Sinclair AJ, Wilson A, Nakkote S, Kelly F, Abedin L et al. Effect of dietary alpha-linolenic acid intake on thrombotic risk factors in vegetarian men. *Am J Clin Nutr* 1999;69:872–82.
63. Australian Iron Status Advisory Panel. *Iron and pregnancy: recommended guidelines*. Sydney: AISAP, 1997.
64. Kim I, Hungerford DW, Yip R, Kuester SA, Zyrkowski C, Trowbridge FL. Pregnancy nutrition surveillance system: United States, 1979–1990. *MMWR CDC Surveillance Summaries* 1992;41:25–41.
65. Scholl TO, Hediger ML, Fischer RL, Shearer JW. Anaemia vs iron deficiency: increased risk of preterm delivery in a prospective survey. *Am J Clin Nutr* 1992;55:985–8.
66. Burr ML, Butland BK. Heart disease in British vegetarians. *Am J Clin Nutr* 1988;48:830–2.
67. Fonnebo V. The Tromso heart study: diet, religion and risk factors for heart disease. *Am J Clin Nutr* 1988;48:826–9.

68. Fraser GE. Determinants of ischaemic heart disease in Seventh Day Adventists. *Am J Clin Nutr* 1988;48:833–6.
69. Kestin M, Rouse IL, Correll R, Nestel PJ. Cardiovascular disease risk factors in free-living men: comparison of two prudent diets, one based on ovolacto-vegetarianism and the other allowing red meat. *Am J Clin Nutr* 1989;50:280–7.
70. Reddy S, Sanders TAB. Lipoprotein risk factors in vegetarian women of Indian descent are unrelated to dietary intake. *Atherosclerosis* 1992;95:223–9.
71. Pronczuk A, Kipervarg Y, Hayes KC. Vegetarians have higher plasma alpha-tocopherol relative to cholesterol than do non-vegetarians. *J Am Coll Nutr* 1992;11:50–5.
72. Prasad K, Reddy S, Sanders TAB. Plasma ubiquinone (Q10) concentrations in female vegetarians and omnivores. *Proc Nutr Soc* 1993;52:332A.
73. Haines AP, Chakraharti R, Fisher D, Meade TW, North WR, Stirling Y. Haemostatic variables in vegetarians and non vegetarians. *Thromb Res* 1980;19:139–48.
74. Sanders TAB, Key TJA. Blood pressure, plasma rennin activity and aldosterone concentrations in vegans and omnivore controls. *Hum Nutr: Appl Nutr* 1987;41:101–8.
75. Armstrong BK, Clarke H, Martin C, Ward W, Norman N, Masarei J. Urinary sodium and blood pressure in vegetarians. *Am J Clin Nutr* 1979;32:2472–6.
76. Margetts BM, Beilin LJ, Vandongen R, Armstrong BK. Vegetarian diet in mild hypertension: a randomised controlled trial. *BMJ* 1986;293:129–33.
77. Messina MJ. Legumes and soybeans: an overview of their nutritional profiles and health effects. *Am J Clin Nutr* 1999;70(suppl.):439S–450S.
78. Fraser GE, Sabate J, Beeson WL, Strahan TM. A possible protective effect of nut consumption on risk of coronary heart disease: the Adventist Health Study. *Arch Intern Med* 1992;152:1416–24.
79. Kushi LH, Folsom AR, Prineas RJ, Mink PJ, Wu Y, Bostick RM. Dietary antioxidant vitamins and death from coronary heart disease in post menopausal women. *N Engl J Med* 1996;334:1156–62.
80. Hu FB, Stampfer MJ, Manson JE, Rimm EB, Colditz GA, Rosner BA et al. Frequent nut consumption and risk of coronary heart disease: prospective cohort study. *BMJ* 1998;317:1341–5.
81. Sabate J. Nut consumption, vegetarian diets, ischaemic heart disease risk and all-cause mortality: evidence from epidemiologic studies. *Am J Clin Nutr* 1999;70(suppl.):500S–503S.
82. Hu FB, Stampfer MJ. Nut consumption and risk of coronary heart disease: a review of epidemiologic evidence. *Current Athero Rep* 1999;1:205–10.
83. Snyder AC, Dvorak LL, Roepke JB. Influence of dietary source on measures of iron status among female runners. *Med Sci Sports Exerc* 1989;21:7–10.

84. Haymes EM, Lamanca JJ. Iron loss in runners during exercise: implications and recommendations. *J Sports Med* 1989;7:277–85.
85. Lissner L, Heitman BL. Dietary fat and obesity: evidence from epidemiology. *Eur J Clin Nutr* 1995;49:79–90.
86. Riley LW, Remis RS, Helgerson SD, McGee HB, Wells JG, Davis BR et al. Hemorrhagic colitis associated with a rare *Escherichia coli* serotype. *New Engl J Med* 1983;308:681–5.
87. Centers for Disease Control and Prevention. Community outbreak of hemolytic uremic syndrome attributable to *Escherichia coli* 0111:NM—South Australia, 1995. *Morbidity and Mortality Weekly Report* 1995;44:157–60.

I.4 INCLUDE MILKS, YOGHURTS, CHEESES AND/OR ALTERNATIVES

- **Reduced-fat varieties should be chosen where possible**

Katrine Baghurst

TERMINOLOGY

Milks, yoghurts and cheeses

The term *milks, yoghurts and cheeses*, as used in this guideline, 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. Goat and sheep milks have overall nutrient profiles that are similar to that of cow's milk, although goat's milk is slightly more 'watery' (less nutrient dense), whereas sheep's milk is almost twice as concentrated as cow's milk, containing more calcium per unit weight but also more fat and energy. In addition, goat and sheep milks are generally not pasteurised and can constitute a health risk. Milks, and products made from them, are good sources of a number of nutrients—for example, protein, riboflavin and vitamin B₁₂—but notably calcium.

Alternatives

Inclusion in the 'alternative' category is based primarily on calcium content, although most of the alternatives also provide substantial amounts of protein. *Alternatives* includes milk-based custards, ice-creams and evaporated milks, as well as fortified soy milk and derivatives. Sardines and other fish whose bones are eaten, and certain nuts (such as almonds), also contain moderate to good amounts of calcium and protein and in this respect can be considered as alternatives for groups such as vegetarians.

Milk foods

For the purposes of this guideline, where *milk foods* is used it generally refers to cow's milk (fresh, long-life, reconstituted dried and evaporated milks) and products made from it, such as yoghurts, ice-creams, cheeses and milk-based custards, which are good sources of calcium as well as a range of other nutrients. The term can also include goat and sheep milk products, although their nutrient profile may differ. Reduced-fat varieties are recommended because of their relatively high saturated fat content (see also Section 1.6). It is important, however, to note that low-fat soft cheeses such as cottage cheese and ricotta contain very little calcium. In the 1995 National Nutrition Survey a category

called 'milk products and dishes' was used; this category included the foods listed under 'milk foods' here but also included cream and soy alternatives such as soy milks and cheeses.

Reduced-fat products

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

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; in young adults, trauma is the primary cause of fractures.¹ Clinically, osteoporosis is measured in terms of bone mineral density that is below the age-adjusted reference range. Individuals are considered osteoporotic if their bone mineral density is 2.5 SD or more below the young adult mean.² This criterion identifies about 30 per cent of all post-menopausal women as having osteoporosis and, of these, more than 50 per cent will have suffered a previous fracture.² Osteoporosis is also of growing importance in men.³

BACKGROUND

Milk foods are a major source of nutrients in the Australian diet. Milk itself is one of the most complete of all foods, containing nearly all the constituents of nutritional importance to humans. Milk foods are the richest source of calcium in the Australian diet⁴ but are also important contributors to protein, vitamin A, riboflavin, vitamin B₁₂ and zinc. Few other foods provide such a readily absorbable and convenient source of calcium. Calcium is required for the normal development and maintenance of the skeleton.⁵ It is stored in the teeth and bones, where it provides structure and strength. In western cultures low intakes of calcium have been associated with osteoporosis, which often results in bone fracture and is one of the main causes of morbidity among older in Australians, particularly women.

Given the ageing of the Australian population, in 1986 it was estimated that in the next 25 years hospital admissions for osteoporotic fractures would increase 84 per cent.⁶ The Dubbo Osteoporosis Epidemiology Study found that, after the age of 60 years, about 60 per cent of women and 30 per cent of men suffer osteoporotic fractures.⁷ The most common fracture sites related to the condition are the hip, vertebrae and wrist; hip fractures have the greatest overall public health impact. Considerable morbidity and mortality are associated with hip fractures, and they lead to a substantial decline in physical function.⁸ In 2001 it was estimated that the cost of osteoporosis in Australia was \$1.9 billion in direct health care costs and a further \$5.6 billion in indirect costs associated with loss of earnings, volunteer carers, modifications and equipment.⁹

Milk foods do, however, also supply over a quarter of the saturated fat in the diet of Australian adults⁴, so reduced- or low-fat varieties are generally recommended. Cow's milk is the most common form of milk consumed in Australia. The composition of milk and some of its alternatives are shown in Table 1.4.1.

Current intakes of milk, milk-based foods, calcium and other nutrients

The 1995 National Nutrition Survey used a 24-hour recall technique supplemented by a qualitative food-frequency questionnaire.⁴ The survey had a category of consumption called 'milk products and dishes', which includes items such as milks, yoghurts, cheese, cream, ice-cream and milk-based custards, as well as milk substitutes such as soy milks and soy cheeses. On the day of the survey, 93 per cent of subjects consumed foods from this category, the average intake being 322 grams for males aged over 19 years and 258 grams for women over 19 years. Intakes fell with age in adult males but remained relatively stable in adult females. Thirty-eight per cent of men and 45 per cent of women consumed less than one serving of these foods on the day of the survey, and only 16 per cent of men and 10 per cent of women consumed three or more servings.

The National Nutrition Survey found that the mean daily intake of calcium in males aged over 19 years was 946 mg a day; in females aged more than 19 years it was 749 mg a day. In men, intakes fell with age, ranging from 1101mg/day in males aged 19–24 years to 989mg/day in those aged 25–44 years, 885 mg/day in those aged 45–64 years, and 796mg/day in those over 60 years. In women, there was no clear age trend, with younger adults (19–24 years) consuming 750mg/day on average, compared with 762mg/day in those who were 25–44 years of age and 769mg/day in the 45–64 year group, but then falling off somewhat to 686mg/day in those aged over 60 years. Milk products and dishes provided just over half the calcium in the diets of adults: milk itself contributed 30 per cent of total calcium and cheese contributed 12 per cent.

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

In addition to their role as a source of calcium, the National Nutrition Survey found that milk products and dishes provided 12 per cent of dietary energy, 15 per cent of protein, 13–14 per cent of vitamin A (25 per cent of retinol), 30 per cent of riboflavin (vitamin B₂), 27 per cent of vitamin B₁₂¹⁰, and 13 per

Table 3.4.1 Nutrient content per 100 grams: milk, yoghurts, cheeses and calcium-rich alternatives

Food	Energy (kJ)	Protein (g)	Fat (g)	Sat. fat (g)	Calcium (mg)	Sodium (mg)	Vitamin B ₂ (mg)	Vitamin B ₁₂ (µg)	Vitamin A equiv (µg)	Zinc (mg)	Iron (mg)
Milk: fluid, whole	272	3.3	3.8	2.5	114	51	0.2	0.3	48	0.4	0.1
Milk: fluid, reduced-fat (fat 1–2%)	203	3.9	1.4	0.9	137	58	0.2	0.3	14	0.4	0.1
Milk: fluid, low-fat (fat <1.1%)	190	4.6	0.2	0.1	160	66	0.3	0.3	5	0.5	0.1
Milk: fluid, skim or non-fat (fat <0.16%)	145	3.6	0.1	0.1	123	54	0.2	0.3	0	0.4	0.1
Yoghurt: regular fat, plain	304	4.7	3.4	2.2	171	77	0.3	0.3	39	0.5	0.1
Ice-cream: regular	766	3.5	10.6	6.9	119	86	0.3	0.4	140	0.3	0.1
Ice-cream: reduced-fat	607	4.5	6.0	3.9	130	95	0.3	0.4	80	0.4	0.1
Cheese: cheddar	1690	25.4	33.8	21.5	775	656	0.0	0.2	390	3.6	0.0
Soy beverage: fortified, unflavoured	260	3.5	3.5	0.4	116	59	0.2	0.5	39	0.2	0.5
Soy beverage: low-fat, fortified with calcium	175	3.5	0.5	0.1	110	40	0.2	0.3	39	0.2	0.5
Tofu: cooked, fat not added in cooking	304	8.1	4.2	0.6	330	7	–	–	–	0.7	1.2
Sardine: canned in oil, drained	952	21.8	15.7	5.1	380	608	0.3	28.0	65	1.8	2.7
Sardine: canned in water, drained	767	21.8	10.7	2.8	380	608	0.3	28.0	65	1.8	2.7
Almond: raw	2455	20.0	55.3	3.6	235	6	1.2	0.0	2	3.6	3.5
Almond: roasted	2618	18.6	60.5	4.2	218	6	1.0	0.0	1	3.3	3.3
Examples of milk-based and soy foods with limited calcium content											
Cheese: cottage	512	15.3	5.8	3.8	73	200	–	0.2	65	0.5	0.0
Cream: pure (fat >35%)	1660	1.9	42.8	28.3	60	27	0.2	0.1	580	0.3	0.1
Soy beverage: unfortified, unflavoured	164	2.5	2.1	0.3	13	59	–	1.5	–	0.3	0.4
Soy beverage: low-fat, unfortified	110	2.5	0.3	0.0	12	40	–	–	–	0.3	0.4

– Zero.

Note: The milk analysis is for cow's milk.

Source :AUSTNUT food composition tables (FSANZ, 1999) for all nutrients except salt (Nurttab '95) and vitamin B₁₂ (British food tables—McCance & Widdowson).

cent of zinc. The importance of protein, zinc and vitamin B₁₂ in the diet is discussed in more detail in Section 1.3. Retinol is the preformed form of vitamin A; it is found only in foods of animal origin, although beta-carotene from plant sources can be converted in the body to retinol. It is essential for maintaining epithelial integrity: deficiency can lead to a variety of eye conditions, ranging from inability to see in dim light to conditions causing blindness. Riboflavin is a B vitamin that is important in cell respiration. Deficiency can lead to oedema of the pharynx and oral mucosa, cheilosis, glossitis, angular stomatitis, conjunctivitis, corneal vascularisation and some forms of anaemia. Deficiency has been documented in both industrialised and developing nations and across varying demographic groups.^{11,12}

The National Nutrition Survey also found that milk products and dishes provided 17 per cent of total fat and 27 per cent of saturated fat, emphasising the need to promote low- or reduced-fat varieties. More information on dietary fats and their health effects is provided in Section 1.6.

SCIENTIFIC BASIS

Although milk foods are valuable sources of a number of nutrients, including protein, retinol, riboflavin, vitamin B₁₂ and zinc¹³, the primary rationale for including this food group in the dietary guidelines lies in its role as a rich source of calcium.

The scientific basis for this guideline thus centres on the role of the milk foods as a key source of readily absorbable dietary calcium and the role of calcium (in conjunction with a number of other factors) in attaining peak bone mass and in preventing osteoporosis.

The importance of peak bone mass

The skeleton is in a phase of rapid growth throughout childhood and adolescence. Between birth and puberty it increases in mass about seven-fold and a further three-fold during adolescence.¹⁴ The best protection against age-related bone loss and consequent fracture risk is considered to be the attainment of a high peak bone mass at skeletal maturity. Peak bone mass is attained between 19 and 30 years of age, after which bone mass gradually declines at a rate that varies from person to person. Bone mass is a product of the interaction between endogenous (hereditary and endocrine) and exogenous (nutrition and weight-bearing exercise) factors. Calcium seems to be the pre-eminent nutritive factor determining peak bone mass in young adults.^{5,14}

It is important to achieve a high peak bone mass: the greater the mass before age-related loss begins, the less likely it is that the mass will decrease to levels where fractures may occur.¹⁵ During the adolescent growth spurt, the required calcium retention is two to three times greater than the average level required for the development of peak bone mass.¹⁵ Restricted food intakes at this age are

therefore of particular concern, and young girls with very heavy exercise regimes and who restrict their total food intake are at particular risk of developing an inadequate peak bone mass. During the period that peak bone mass is developing, it is necessary to ingest sufficient calcium to maintain positive balance. This quantity will vary from person to person, depending on individual efficiency of intestinal calcium absorption. Once peak bone mass is achieved, it is maintained without much change for 10 to 20 years. Daily requirements at this stage are about two-thirds of what is required to attain peak mass, since bone building has been completed. But males and females lose bone at a constant rate of 0.2–0.5 per cent a year starting at age 40–45 years. For about 10 years immediately before, during and after menopause¹⁶, women lose bone more rapidly than men (2–5 per cent a year); after this 10-year period, the rate of loss returns to the slower rate shared by the two genders.

Calcium balance and bone mass

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

Dietary calcium, bone loss and fractures

If, as postulated, bone loss is related to dietary calcium deficiency, individuals with low levels of calcium consumption should have lower bone mineral density than those with higher levels of calcium intake. A meta-analysis of 27 cross-sectional, two longitudinal and four intervention studies assessing the effect of calcium intake on bone mass in young and middle-aged females and males¹⁷ concluded that, overall, there was evidence that calcium intake was positively associated with bone mass in pre-menopausal women, although it alone did not account for a large amount of the variation in bone mass.

Low bone density is associated with an increased risk of fracture: the lower the bone density the greater the risk. If an absolute or relative dietary calcium deficiency of itself contributes to the development of osteoporosis, therapy with calcium should improve not only bone density and bone loss but also fracture rates.

There is evidence that a high calcium intake at older ages slows the rate of bone loss and may reduce the risk of fracture. A number of randomised trials have

shown that calcium supplements are effective in slowing bone loss in older women^{18–21} but a meta-analysis of nine randomised controlled trials of the effect of calcium supplementation on bone density in post-menopausal women²² concluded that, whilst the rate of bone loss was less in supplemented women in the first year of treatment, in the second year it was not. Only a limited number of randomised controlled trials of calcium supplementation have used fracture end-points^{23–26}; these studies have, however, consistently shown a reduction in risk, albeit ranging from 26 to 70 per cent. A systematic review of 14 studies—randomised and non-randomised controlled trials, case-control studies and cohort studies—also concluded that calcium supplements and dietary calcium probably reduce the risk of osteoporotic fractures in women.²⁷

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

Intestinal calcium absorption and the ability to adapt to low-calcium diets change with age. Fractional calcium absorption is highest (about 60 per cent) in infancy^{28,29} and rises again in early puberty. Fractional absorption in Caucasian girls consuming about 900 milligrams of calcium a day has been shown to average 28 per cent pre-puberty, 34 per cent in early puberty, and 25 per cent two years later.³⁰ Fractional absorption remains at this level in young adults, then gradually declines with ageing. In post-menopausal women, Heaney et al.³¹ showed a decline of 0.21 per cent a year, and Bullmore et al.³² reported that men lose absorption efficiency at the same rate as women. This results in an increased calcium requirement if calcium balance and skeletal integrity are to be maintained.

For women, superimposed on the effects of ageing is the effect of menopause. Calcium balance deteriorates at menopause, perhaps as a result of a decline in intestinal calcium absorption or an increase in urinary calcium excretion, or both. Heaney et al.³³ demonstrated that the dietary calcium requirement needed to prevent negative calcium balance was increased some 50 per cent by menopause. The interrelationship between oestrogen deficiency and intestinal calcium absorption was also studied by Gallagher et al.³⁴, who demonstrated that oestrogen therapy increases both calcium absorption and the dihydroxy form of vitamin D. The relative importance of hormone replacement, increased dietary calcium intake, and/or calcium supplementation in prevention of osteoporosis is not yet clear.

Impaired intestinal calcium absorption with ageing and menopause may, in part, be related to changes in vitamin D metabolism. This may be a consequence of inadequate dietary intake of vitamin D, less efficient intestinal absorption, less efficient skin synthesis of vitamin D, inadequate exposure to ultraviolet light, or a decreasing ability of the kidneys—around menopause or in ageing—to produce 1,25-dihydroxy vitamin D, the major biologically active metabolite of vitamin D. Whatever the cause, 1,25-dihydroxy vitamin D, the primary regulator of intestinal calcium absorption, is present in lower concentrations in the elderly and in menopause and may be lower in patients with osteoporosis compared with age-matched control subjects.

There is recent evidence that vitamin D deficiency might be becoming a public health concern among certain cultural and linguistic groups in Australia, with deficiencies being seen in dark-skinned or veiled women and their children.^{35,36} The extent of the problem is not yet clear. The main food sources of vitamin D in Australia are margarines fortified with vitamin D, fatty fish and eggs.³⁷

Some studies suggest that vitamin D supplementation is effective in preventing fractures only in people who have marginal vitamin D serum levels.^{33,38,39} Details of these studies are given in the *Dietary Guidelines for Older Australians*.⁴⁰ As a result of these considerations, in 1989 the NHMRC's Nutrition and Public Health Committee recommended vitamin D supplementation of housebound older people and of other people not exposed to sunlight.⁴¹

Some factors affecting calcium needs

Bioavailability

For food sources of calcium, content is of greater importance than bioavailability. Calcium absorption efficiency is similar from most foods, but it may be poor from foods rich in oxalic acid (for example, spinach, rhubarb and beans) and phytic acid (seeds, nuts, grains, raw beans and soy isolates). Soybeans have large amounts of phytate but absorption of calcium is still quite high.⁴² Compared with milk, calcium absorption from dried beans is about half; from spinach it is about one-tenth. Bioavailability from non-food sources such as supplements depends on the dose and whether taken with a meal. In standardised studies of 250-milligram calcium supplements given with a breakfast meal, calcium citrate malate gave a fractional absorption rate of 35 per cent, calcium carbonate 27 per cent, and tricalcium phosphate 25 per cent; this compares with a rate of 29 per cent for calcium from milk.^{43–46} Efficiency of absorption of calcium from supplements is greatest at doses of 500 milligrams.^{47,48}

Physical activity

It is generally accepted that weight-bearing exercise determines the strength, shape and mass of bone⁴⁹, although the mechanisms are still not clear. It is also unclear whether calcium intake influences the benefit gained from exercise. With complete immobilisation, rapid bone loss occurs despite a high calcium intake.⁵⁰ In an intervention study in children, calcium and exercise both affected bone mineralisation but the effects appeared to be independent.⁵¹ A review of 16 studies in adults, mostly women, concluded that high calcium intakes (over 1000 milligrams) enhanced the bone mineral density benefits of exercise to different degrees in various parts of the skeleton.⁵² The review of calcium requirements undertaken for development of the US dietary reference values⁵³ concluded there was insufficient evidence to justify different calcium requirements for people with different activity levels.

Sodium

Sodium and calcium excretion are linked in the kidney tubules. A high salt intake increases urinary sodium, resulting in increased obligatory loss of urinary calcium. In post-menopausal women, 500 milligrams of excreted sodium draws 10 milligrams of calcium into the urine.⁵⁴ In children and adolescents, urinary sodium is an important determinant of urinary calcium excretion^{55,56}, but no association has been shown between salt intake or excretion and skeletal development in children. One longitudinal study in post-menopausal women did, however, show a link between high urinary sodium and increased hip bone loss.⁵⁷ No study has yet shown a direct link between sodium intake and bone loss or fracture rates. The US dietary reference intakes committee examining calcium requirements⁵³ concluded that, despite the relatively high salt intake in the United States, the available evidence did not warrant the setting of different calcium requirements for people with different sodium intakes.

Protein

Protein increases urinary calcium excretion but its effect on calcium retention is unclear. Walker and Linkswiler⁵⁸ found that urinary calcium increased by about 0.5 milligrams for each gram of dietary protein over about 47 grams a day. However, low dietary protein intakes (below 34 grams a day) have been shown to be associated with poor health and poor recovery from osteoporotic hip fracture.⁵⁹ The US review of calcium requirements⁵³ concluded that evidence of the effect of protein intake on calcium requirement was not sufficient to recommend different calcium intakes for different intakes of protein.

Calcium and other chronic diseases

Hypertension

Many studies have investigated links between calcium and hypertension. Some cross-sectional studies have shown that lower dairy intakes are associated with higher blood pressure, possibly through a calcium effect⁶⁰, and some (but not all) intervention studies have shown a similar effect. In a review of 22 randomised intervention trials⁶¹, calcium supplementation was found to reduce systolic blood pressure slightly in hypertensives but not affect normotensives; diastolic blood pressure was not affected in either group. In another study using a diet with increased low-fat dairy products, fruit and vegetables but reduced total and saturated fat⁶², blood pressure fell in both normotensives and hypertensives. The increased dairy consumption provided an almost three-fold increase in calcium—from 443 to 1265 milligrams a day—but other dietary changes, such as an increase in potassium and a decrease in sodium, may have played a role. The effect of calcium supplementation on blood pressure has generally been found to be modest and variable across populations, and it has been suggested that calcium supplementation may lower blood pressure only in those on a relatively high salt intake.⁶³

Colon cancer

Although suggestions have been made about a link between calcium and colon cancer, the evidence is weak. Observational and case-control studies have had mixed results.^{64–66} Greater mucosal proliferation is seen in patients at high risk of colon cancer^{67–69}; one study showed increased mucosal proliferation with calcium supplements⁷⁰, while another showed a suggestion of decreased proliferation although this did not reach statistical significance possibly due to the small numbers involved.⁷¹

Requirements and recommended intakes for calcium

Calcium requirements are largely determined by skeletal needs, which increase during periods of rapid growth (such as childhood and adolescence), during pregnancy and lactation, and in later life. Needs can be assessed in a number of ways, among them balance studies, a factorial estimate approach, and changes in bone mineral density or content.³²

In Australia the calcium requirement has been used to estimate the recommended dietary intake, which is traditionally set to meet the requirements of the majority of the population.⁷² The NHMRC's current recommended intake of dietary calcium, which was set over a decade ago, in 1991, increases from 800 milligrams a day in pre-menopausal women to 1000 milligrams a day in post-menopausal women.⁷² This is to account for the accelerated loss of calcium from the skeleton after menopause. In pregnancy, an additional 300mg/day (total 1100mg/day) is currently recommended; in lactation, an additional 400mg/day (total 1200mg/day) is recommended.

For men of all ages, the Australian recommended dietary intake for calcium is 800 milligrams a day. There is some evidence, however, that this may not be sufficient in older men since it does not take into account the age-related changes in calcium and vitamin D metabolism. This notion has gained some support from the review of recommended dietary allowances in the United States.

The continuing revision of the US recommended dietary allowances, or RDAs, is being undertaken by a group of committees under the auspices of the Institute of Medicine.⁵³ A new multi-stage form of expression for the recommendations is being used, similar to that first used in the United Kingdom in the early 1990s.⁷³ This includes an evidence-based determination of an 'estimated average requirement', or EAR, for individuals, the mean and variance of which is used to derive the more familiar recommended dietary allowance for individuals ($RDA = EAR + 2SD_{EAR}$).

The committee assessing calcium concluded that there was insufficient evidence to establish an evidence-based estimated average requirement for calcium, for any age or gender group. As a result, it did not produce recommended dietary allowances for calcium but instead estimated what it called an 'adequate intake' (AI) figure for each age and gender group. An AI for a nutrient is set as an

alternative to the RDA where the data are considered insufficient (or not certain enough) to develop a reliable variance estimate for the population. The AI is believed to cover the needs of most people in the population, although the percentage of the population covered by this recommended intake level cannot be specified with confidence.

The AI for calcium for adult males and females aged 19–50 years was set at 1000 milligrams a day and that for adult males and females aged over 51 years was set at 1200 milligrams a day. For pregnancy and lactation, the figure was set at 1000mg/day (that is, no additional requirement because of increased absorptive capacity) unless the mother was aged 18 years or less, in which case it was set at 1300mg/day to account for ongoing needs for growth.

It is of interest to note that the 1991 UK recommendations⁷³—termed ‘reference nutrient intakes’ and equivalent to the US RDAs—also set the same figure for males and females but at a much lower level than the US recommendations (700mg/day for all adults, irrespective of age and gender). The British also have no increased recommendation for pregnancy but do recommend an additional 550mg/day for lactation.

Special groups

Vegetarians

A vegetarian diet can influence calcium needs because of its relatively high oxalate and phytate content. Recent short-term studies have, however, indicated that isoflavone-rich soy protein may have a beneficial effect on bone loss.⁷⁴ On balance, lacto-ovo-vegetarians appear to have calcium intakes similar to those of omnivores^{75–77} and similar urinary excretion.^{78,79} One five-year study in post-menopausal lacto-ovo-vegetarians and omnivores with similar calcium intakes showed that the two groups lost radius bone mineral density at similar rates.⁷⁷ Bone data on strict vegetarians are not available. Vegetarians who avoid milk and calcium-fortified soy products should seek advice about whether they need to take calcium supplements.

Migrants and people with lactose intolerance

One group in Australia that may need to pay particular attention to calcium requirements is recent migrants from countries where the background diet is traditionally lower in protein and salt than in Australia and where everyday physical activity may be greater—for example, some Asian countries. If these migrants adopt Australian dietary and lifestyle patterns, their calcium requirement will increase. This group may be at particular risk of developing osteoporosis in the future.

Lactose intolerance is also high in Asian communities (80–90 per cent) but relatively low among Caucasians (10–20 per cent). A 1994 review found that only limited data were available on the extent of lactose intolerance in Australians.⁸⁰ At that time, most studies of adult Caucasians showed a rate of 17–

20 per cent for lactose maldigestion; one study showed a rate of only 4 per cent. Data for Aboriginal adults showed rates of 80 per cent or above, and data for Asian-Australian adults showed rates of 80–90 per cent. Figures of 3–9 per cent were obtained for Caucasian children, 50–90 per cent for various studies in Aboriginal children, and about 50 per cent for children of Mediterranean background. Study numbers for both adults and children are, however, generally very limited. Small amounts of milk or dairy foods can often be tolerated by people with lactose intolerance, but lactose-free dairy products are also available now. Lactose-intolerant people often avoid milk products, although this may not be necessary. Fortified soy milks can also be used for people with lactose intolerance.

Women with amenorrhea

Amenorrhea resulting from anorexia is associated with lowered calcium absorption, higher urinary calcium excretion, and a lower rate of bone formation.⁸¹ Exercise-induced amenorrhea results in reduced calcium retention and lower bone mass.^{82,83}

PRACTICAL ASPECTS OF THIS GUIDELINE

The *Australian Guide to Healthy Eating*⁸⁴ which was based on the NHMRC's core food groups analysis⁸⁵, recommends two to three serves of milk, yoghurt or cheese or alternatives each day for women and two to four serves for men, where a serve is equivalent to a cup of milk, half a cup of evaporated milk, 40 grams of cheese or 200 grams of yoghurt, or alternative (see below). The 1995 National Nutrition Survey found that some 42 per cent of adults consumed less than one serve of these kinds of foods on the day of the survey.

Although rich in calcium, these foods (particularly cheeses) can be relatively high in saturated fat, so it is recommended that reduced-fat varieties or reduced-fat alternatives be chosen where possible. Even so, the energy and/or fat to calcium ratio of some items (for example, milks and yoghurts) will generally be lower than that for others (for example, cheese and ice-cream, even reduced-fat varieties) and the former selections should predominate.

A variety of calcium-enriched milks that are also low in fat are readily available. Low- and reduced-fat yoghurts and cheeses are also available, but low-fat soft cheeses such as cottage cheese and ricotta have very little calcium and cannot be counted as a 'serve', even though they may add variety to a low-fat diet. Low-lactose milks, yoghurt and cheese products are now available for people with lactose intolerance. Most adults who are lactose intolerant can, however, consume sufficient milk, yoghurt and cheese products without developing significant symptoms.

If people cannot, or do not want to, eat dairy foods, the following are examples of what can be substituted in terms of calcium equivalents for one serve:

- a cup of calcium (and vitamin B₁₂)–fortified soy beverage containing 100 milligrams of calcium per 100 millilitres—non-fortified soy beverages do not provide sufficient calcium
- a cup of almonds
- five sardines or half a cup of pink salmon—with bones
- a cup of calcium-fortified breakfast cereal.

RELATIONSHIP TO OTHER GUIDELINES

In the earlier dietary guidelines for adults, children and older Australians, there was one guideline dealing with variety in food choice and others encouraging consumption of calcium-rich and iron-rich foods. In 1998 the *Australian Guide to Healthy Eating* was developed; as noted, it is based on the NHMRC's core food groups analysis, which modelled the general eating patterns required in the community to achieve the recommended dietary intakes for specific energy intake levels.

For this revision of the dietary guidelines, it was felt that the previous 'variety' guideline should be more closely linked to the *Australian Guide to Healthy Eating*, which recommends daily consumption of a variety of foods from five basic food groups:

- vegetables & legumes
- fruit
- breads, cereals and grain
- milk, yoghurt, cheese and alternatives
- meats, fish, poultry and alternatives.

Guidelines on consumption of vegetables, legumes and fruit and of breads, cereals and grains were included in earlier guidelines. In this revision, guidelines are included for milks, yoghurts, cheeses and alternatives and for meats, fish, poultry and alternatives. It was also felt that a food-based, rather than a nutrient-based, approach to the guidelines would be more consistent with international trends in the setting of dietary guidelines. As a consequence, calcium-rich foods are included in the milks, yoghurts, cheeses and alternatives guideline and iron-rich foods are included in the meat, fish, poultry and alternatives guideline.

Enjoy a wide variety of nutritious foods

Milks, yoghurts, cheeses and alternatives are recommended as part of a varied diet to achieve the balance of nutrients required for optimal health. The NHMRC's core food group analysis⁸⁵ confirms a central role for these foods in the Australian diet in this context. As noted, the *Australian Guide to Healthy Eating* includes the milk group as one of its five core food groups.⁸⁴

Limit saturated fat and moderate total fat intake

The 1995 National Nutrition Survey⁴ found that milk products and dishes contributed some 17 per cent of total fat and 27 per cent of saturated fat to the diet. But these foods are valuable sources of other nutrients, so it is appropriate to choose low- or reduced-fat varieties rather than reducing overall intake in order to limit fat intake. Serves of full-fat cheeses should be limited to three or four times a week.

Choose foods low in salt

There is evidence that high intakes of sodium increase urinary calcium loss. Conservation of calcium is thus an additional reason for following the salt guideline. This is more important for older people, whose ability to absorb dietary calcium may be impaired.

Prevent weight gain: be physically active and eat according to your energy needs

Regular weight-bearing exercise is an important contributor to bone mineralisation. Participation in regular physical activity from early childhood will not only contribute to a healthy body weight but also help to achieve optimal peak bone mass.

CONCLUSION

The health costs associated with hospital admissions for osteoporotic fractures are high in Australia. An adequate intake of calcium will help delay bone loss and the onset of osteoporosis and so reduce the number of related fractures in older people. Dairy products are the most reliable source of calcium; they are readily available and convenient to use. They are also valuable sources of protein, riboflavin, vitamin A, vitamin B₁₂ and zinc. If foods high in calcium are part of people's daily diet, the physiological and social costs associated with a low-calcium diet will be reduced.

EVIDENCE

The scientific rationale for this guideline is based on a variety of evidence sources, among them the following:

- meta-analyses of randomised controlled trials (Level I evidence) relating calcium supplementation to bone density (reference 22) and calcium to blood pressure (reference 61)
- well-designed, individual randomised controlled trials (Level II evidence) relating calcium supplementation to bone loss and bone density (references 18 to 21 and 24 to 26), vitamin D to fracture (reference 38)

- Level III evidence—a cohort study assessing the effects of calcium on colorectal cancer (reference 64), a meta-analysis of case-control and cohort studies relating calcium intake to bone mass (reference 17), and evidence concerning vitamin D and bone density (reference 39), sodium and calcium and bone density (reference 57), dietary supplementation and fractures (reference 59), and dietary patterns and blood pressure (reference 62).

Evidence was also obtained from a number of cross-sectional and population studies, as well as human experimentation relating to bioavailability and nutrient requirements and intakes.

REFERENCES

1. Geelhoed EA, Criddle A, Prince RL. The epidemiology of osteoporotic fracture and its causative factors. *Clin Biochem Rev* 1994;15:173–8.
2. World Health Organization. *Assessment of fracture risk and its application to screening for postmenopausal osteoporosis*. Technical Report Series no. 843. Geneva: WHO, 1994.
3. Orwoll ES, Klein RE. Osteoporosis in men. *Endocr Rev* 1995;116:87–116.
4. Australian Bureau of Statistics. *National Nutrition Survey: nutrient intakes and physical measurements, Australia, 1995*. Cat. no. 4805.0. Canberra: ABS, 1998.
5. Heaney RP. Nutrition and risk for osteoporosis. In: Marcus R, Feldman D, Kelsey J, eds. *Osteoporosis*. San Diego: Academic Press, 1996:483–509.
6. Lord SR, Sinnett PF. Femoral neck fractures: admissions, bed use, outcome and projections. *Med J Aust* 1986;145:493–6.
7. Eisman JA. Symptomatic fracture incidence in elderly men and women: the Dubbo Osteoporosis Epidemiology Study (DOES). *Osteoporosis Int* 1994;4:277–82.
8. Marottoli RA, Berkman LF, Cooney LM. Decline in physical function following hip fracture. *J Am Geriatr Soc* 1992;40:861–6.
9. Access Economics. *The burden of brittle bones: costing osteoporosis in Australia*. Report prepared for Osteoporosis Australia. Canberra: Access Economics, 2001.
10. Cobiac L, Record S, Syrette J. *Dairy foods in the Australian diet: results from the 1995/6 National Nutrition Survey*. Adelaide: CSIRO, 1999.
11. Komindr S, Nichoalds GE. Clinical significance of riboflavin deficiency. In: Brewster MA, Naito HK, eds. *Nutritional elements and clinical biochemistry*. New York: Plenum Press, 1980:15–68.
12. Nichoalds GE. Riboflavin. In: Labbae RF, ed. *Symposium on laboratory assessment of nutritional status clinics in laboratory medicine series*. Vol. 1. Philadelphia: WB Saunders, 1981:685–98.

13. ANZFA. Australian Food and Nutrient Database 1999. Canberra 1999
14. Peacock M. Calcium absorption efficiency and calcium requirements in children and adolescents. *Am J Clin Nutr* 1991;54(suppl.):261S–265S.
15. Nordin BEC, Horseman A, Marshall DH, Simpson M, Waterhouse GM. Calcium requirement and calcium therapy. *Clin Orthop* 1979;140:216–46.
16. Heaney RP. Calcium, bone health and osteoporosis. In: Peck WA, ed. *Bone and mineral research, annual 4: a yearly survey of developments in the field of bone and mineral metabolism*. New York: Elsevier, 1986.
17. Welken DC, Kemper HC, Post GB, van Staveren W. A meta-analysis of the effect of calcium intake on bone mass in young and middle-aged females and males *J Nutr* 1995;125(11):2802–13.
18. Prince R, Devine A, Dick I, Criddle A, Kerr D, Kent N et al. The effects of calcium supplementation (milk powder or tablets) and exercise on bone density in postmenopausal women. *J Bone Miner Res* 1995;10:1068–75.
19. Elders PJM, Lips P, Netelenbos JC, van Ginkel FC, Khoe E, van der Vijgh WJF et al. Long-term effect of calcium supplementation on bone loss in perimenopausal women. *J Bone Miner Res* 1994;9:963–70.
20. Dawson-Hughes B, Dallal GE, Krall EA, Sadowski L, Sahyoun N, Tannenbaum S. A controlled trial of the effect of calcium supplementation on bone density in postmenopausal women. *N Engl J Med* 1990;323:878–83.
21. Reid IR, Ames RW, Evans MC, Gamble GD, Sharpe SJ. Effect of calcium supplementation on bone loss in postmenopausal women. *N Engl J Med* 1993;328:460–4.
22. Mackerras D, Lumley T. First and second year effects in trials of calcium supplementation on loss of bone density in postmenopausal women *Bone* 1997;21(6):527–33.
23. Chapuy MC, Arlot ME, Duboeuf F, Brun J, Crouzet B, Arnaud S et al. Vitamin D and calcium to prevent hip fractures in elderly women. *N Engl J Med* 1992;327:1637–42.
24. Reid IR, Ames RW, Evans MC, Gamble GD, Sharpe SJ. Long-term effects of calcium supplementation on bone loss and fractures in postmenopausal women: a randomized controlled trial. *Am J Med* 1995;98:331–5.
25. Chevalley T, Rozzoli R, Nydegger V, Slossman D, Rapin CH, Michel JP et al. Effects of calcium supplements on femoral bone mineral density and vertebral fracture rate in vitamin-D-replete elderly patients. *Osteopor Int* 1994;4:245–52.
26. Recker R, Hinders S, Davies KM, Heaney RP, Stegman MR, Lappe JM et al. Correcting calcium nutritional deficiency prevents spine fractures in elderly women. *J Bone Min Res* 1996;11:1961–66.
27. Cumming RG, Nevitt MC. Calcium for prevention of osteoporotic fracture in postmenopausal women. *J Bone Min Res* 1987;12(9):1321–9.

28. Abrams SA, Wen J, Stuff JE. Absorption of calcium, zinc and iron from breast milk by 5- to 7-month old infants. *Pediatr Res* 1997;41:1–7.
29. Fomon SJ, Nelson SE. Calcium, phosphorus, magnesium and sulfur. In: Fomon SJ, ed. *Nutrition of normal infants*. St Louis: Mosby-Year Book Inc., 1993:192–216.
30. Abrams SA, Stuff JE. Calcium metabolism in girls: current dietary intakes lead to low rates of calcium absorption and retention during pregnancy. *Am J Clin Nutr* 1994;60:739–43.
31. Heaney RP, Recker RR, Stegman RR, Moy AJ. Calcium absorption in women: relationships to calcium intake, estrogen status and age. *J Bone Min Res* 1989;4:469–75.
32. Bullmore JR, Gallagher JC, Wilkinson R, Nordin BEC. Effect of age on calcium absorption. *Lancet* 1970;2:535–7.
33. Heaney RP, Recker RR, Saville PD. Menopausal changes in calcium balance performance. *J Lab Clin Med* 1978;92:953–63.
34. Gallagher JC, Riggs BL, DeLuca HF. Effects of estrogen on calcium absorption and serum vitamin D metabolites in postmenopausal osteoporosis. *J Clin Endocrinol Metab* 1991;51:1359–64.
35. Grover SR, Morley R. Vitamin D deficiency in veiled or dark-skinned pregnant women. *Med J Aust* 2001;175:251–2.
36. Nozza JM, Rodda CP. Vitamin D deficiency in mothers of infants with rickets. *Med J Aust* 2001;175:253–5.
37. Baghurst KI, Record SJ, Leppard P. *Trends in food and nutrient intakes in Australia 1988–2000: results from the 1988, 1993 and 2000 CSIRO national dietary surveys*. Adelaide: CSIRO, 2001.
38. Lips P, Graafmans WC, Ooms ME, Bezemer D. Vitamin D supplementation and fracture incidence in elderly persons: a randomized, placebo-controlled clinical trial. *Ann Intern Med* 1996;124:400–6.
39. Dawson-Hughes B, Harris SS, Krall EA, Dallal GE. Effect of calcium and vitamin D supplementation on bone density in men and women 65 years of age or older. *N Engl J Med* 1997;337:670–6.
40. National Health and Medical Research Council. *Dietary guidelines for older Australians*. Canberra: NHMRC, 1999.
41. National Health and Medical Research Council Nutrition Committee & Public Health Committee. *Vitamin D status of the elderly. Food and nutrition: regulatory and policy matters*. Canberra: Australian Government Publishing Service, 1989.
42. Heaney RP, Weaver CM, Fitzsimmons ML. Soybean phytate: content effect on calcium absorption. *Am J Clin Nutr* 1991;53:745–7.

43. Heaney RP, Recker RR, Stegman MR, Moy AJ. Calcium absorption in women: relationship to calcium intake, oestrogen status and age. *J Bone Min Res* 1989;4:469–75.
44. Heaney RP, Recker RR, Weaver CM. Absorbability of calcium sources: the limited role of solubility. *Calcif Tissue Int* 1990;46:300–4.
45. Miller JZ, Smith DL, Flora L, Slenda C, Jiang X, Johnston CC. Calcium absorption from calcium carbonate and a new form of calcium in healthy male and female adolescents. *Am J Clin Nutr* 1988;138:225–36.
46. Smith KT, Heaney RP, Flora L, Hinders SM. Calcium absorption from a new calcium delivery system. *Calcif Tissue Int* 1987;41:351–2.
47. Heaney RP, Saville PD, Recker RR. Calcium absorption as a function of calcium intake. *J Lab Clin Med* 1975;85:881–90.
48. Heaney RP, Recker RR, Hinders SM. Variability of calcium absorption. *Am J Clin Nutr* 1988;47:262–4.
49. Frost HM. The mechanostat: a proposed pathogenic mechanism of osteoporosis and the bone mass effects of mechanical and nonmechanical agents. *Bone Min* 1987;2:73–85.
50. LeBlanc A, Schneider V, Spector E, Evans H, Rowe R, Lane H et al. Calcium absorption, endogenous excretion, and endocrine changes during and after long bed rest. *Bone* 1995;16:301S–304S.
51. Slemenda CW, Reister TK, Hui SL, Miller JZ, Christian JC, Johnston CC. Influences on skeletal mineralisation in children and adolescents: evidence for varying effect of sexual maturation and physical activity. *J Paediat* 1994;125:201–7.
52. Specker BL. Evidence for an interaction between calcium intake and physical activity on changes in bone mineral density. *J Bone Min Res* 1996;11:1539–44.
53. Institute of Medicine. *Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D and fluoride*. Washington: National Academy Press, 1997.
54. Nordin BEC, Polley KJ. Metabolic consequences of the menopause: a cross-sectional, longitudinal and intervention study on 557 normal postmenopausal women. *Calcif Tissue Int* 1987;41:S1–S59.
55. Matkovic V, Illich JZ, Andon MB, Hsieh LC, Tzagournis MA, Lagger BJ et al. Urinary calcium, sodium, and bone mass of young females. *Am J Clin Nutr* 1995;62:417–25.
56. O'Brien KO, Abrams SA, Stuff JE, Liang LK, Welch TR. Variables related to urinary calcium excretion in young girls. *J Paediat Gastroenterol Nutr* 1996;23:8–12.
57. Devine A, Criddle RA, Dick IM, Kerr DA, Prince RL. A longitudinal study of the effect of sodium and calcium intakes on regional bone density in postmenopausal women. *Am J Clin Nutr* 1995;62:740–5.

58. Walker RM, Linkswiler HM. Calcium retention in the adult human male as affected by protein intake. *J Nutr* 1972;102:1297–1302.
59. Delmi M, Rapin CH, Bengoa JM, Delmas PD, Vasey H, Bonjour JP. Dietary supplementation in elderly patients with fractured neck of femur. *Lancet* 1990;335:1013–16.
60. Ackly A, Barrett-Connor E, Suarez L. Dairy products, calcium and blood pressure. *Am J Clin Nutr* 1983;38:457–61.
61. Allender PS, Cutler JA, Follmann D, Cappuccio FP, Pryer J, Elliot P. Dietary calcium and blood pressure: a meta-analysis of randomised clinical trials. *Ann Intern Med* 1996;124:825–31.
62. Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM et al. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med* 1998;336:1117–24.
63. Resnick LM, DiFabio B, Marion R, James DG, Laragh J. Dietary calcium modifies the pressor effects of dietary salt intake in essential hypertension. *J Hypertension* 1986;4(suppl. 6):S679–S681.
64. Garland C, Shekelle RB, Barrett-Connor E, Criqui MH, Rossof AH, Paul O. Dietary vitamin D and calcium and risk of colorectal cancer: a 19 year prospective study in men. *Lancet* 1985;1:307–9.
65. Meyer F, White E. Alcohol and nutrients in relation to colon cancer in middle-aged adults. *Am J Epidemiol* 1993;138:225–36.
66. Slattery ML, Sorenson AW, Ford MH. Dietary calcium intake as a mitigating factor in colon cancer. *Am J Epidemiol* 1988;128:504–14.
67. Kanemitsu T, Koike A, Yamamoto S. Study of cell proliferation kinetics in ulcerative colitis, adenomatous polyps and cancer. *Cancer* 1985;56:1094–8.
68. Ponz de Leon M, Roncucci L, Di Donnato P, Tassi L, Smerieri O, Amorico MG et al. Pattern of epithelial cell proliferation in colorectal mucosa of normal subjects and of patients with adenomatous polyps or cancer of the large bowel. *Cancer Res* 1988;48:4121–6.
69. Roncucci L, Scalmati A, Ponz de Leon M. Patterns of cell kinetics in colorectal mucosa of patients with different types of adenomatous polyps of the large bowel. *Cancer* 1991;68:873–8.
70. Kleibeuker JH, Welberg JW, Mulder NH, van der Meer R, Cats A, Limburg AJ et al. Epithelial cell proliferation in the sigmoid colon of patients with adenomatous polyps increases during oral calcium supplementation. *Br J Cancer* 1993;67:500–3.
71. Bostick RMN, Potter JD, Fosdick L, Grambsch P, Lampe JW, Wood JR et al. Calcium and colorectal epithelial cell proliferation: a preliminary randomised, double-blinded placebo-controlled clinical trial. *J Natl Cancer Inst* 1993;85:132–41.

72. National Health and Medical Research Council. *Recommended dietary intakes for use in Australia*. Canberra: Australian Government Publishing Service, 1991.
73. Committee on Medical Aspects of Food and Nutrition Policy. Dietary Reference values for food energy and nutrients for the United Kingdom. In: *Report of the Panel on Dietary Reference Values*. London: HMSO, 1991.
74. Alekel DL, Germain AS, Peterson CT, Hanson KB, Stewart JW, Toda T. Isoflavone-rich soy protein isolate attenuates bone loss in the lumbar spine of perimenopausal women. *Am J Clin Nutr* 2000;72(3):844–52.
75. Marsh AG, Sanchez TV, Midkelsen O, Keiser J, Mayor G. Cortical bone density in adult lacto-ovo-vegetarian and omnivorous women. *J Am Diet Assoc* 1980;76:148–51.
76. Pedersen AB, Bartholomew MJ, Dolence IA, Aljadir LP, Netteburg KL, Lloyd T. Menstrual differences due to vegetarian and nonvegetarian diets *Am J Clin Nutr* 1991;53:879–85.
77. Reed JA, Anderson JJ, Tylavsky FA, Gallagher PN. Comparative changes in radial bone density of elderly female lacto-ovo-vegetarians and omnivores. *Am J Clin Nutr* 1994;59:1197S–1202S.
78. Lloyd T, Schaeffer JM, Walker MA, Demers LM. Urinary hormonal concentrations and spinal bone densities of premenopausal vegetarian and non vegetarian women. *Am J Clin Nutr* 1991;54:1005–10.
79. Tesar R, Notelowitz M, Shim E, Kauwell G, Brown J. Axial and peripheral bone density and nutrient intakes of postmenopausal vegetarian and omnivorous women. *Am J Clin Nutr* 1992;56:699–704.
80. Cobiac L. Lactose: a review of intakes and of importance to health of Australians and New Zealanders. *Food Australia* 1994;46(7)(suppl.):S1–S28.
81. Abrams SA, Silber TJ, Esteban NV, Vierira NE, Stuff JE, Meyers R et al. Mineral balance and bone turnover in adolescents with anorexia nervosa. *J Paediat* 1993;123:326–31.
82. Drinkwater B, Buemner B, Chestnut C. Menstrual history as a determinant of current bone density in young athletes. *JAMA* 1990;263:545–8.
83. Marcus RT, Cann C, Madvig P, Minkoff J, Goddard M, Bayer M et al. Menstrual function and bone mass in elite women distance runners: endocrine and metabolic features. *Ann Intern Med* 1985;102:158–63.
84. Children's Health Development Foundation & Deakin University. *The Australian guide to healthy eating*. Canberra: Australian Government Publishing Service, 1998.
85. Cashel K, Jefferson S. *The core food groups: scientific basis for developing nutrition education tools*. Canberra: Australian Government Publishing Service, 1995.

1.5 DRINK PLENTY OF WATER

Katrine Baghurst

BACKGROUND

Water is an essential nutrient for life. All biochemical reactions occur in it. It fills the spaces in and between cells and helps form structures of large molecules such as protein and glycogen. Water is also required for digestion, absorption and transportation and as a solvent for nutrients, and for elimination of waste products and thermoregulation.¹ To be properly hydrated in a temperate climate, adults require some 2500–3000 millilitres of fluid a day, depending on body size.² In the Australian climate older adults (and very young children) are at particular risk of dehydration. Solid foods contribute approximately 1000 millilitres of water; an additional 250 millilitres comes from the water produced by the body's metabolism (water of oxidation).³ The remainder needs to come from free water or other fluids, or both.

The normal daily turnover of water is approximately 4 per cent of total body weight in adults.³ Several factors increase the possibility of chronic mild dehydration, among them a poor thirst mechanism^{4,5}, dissatisfaction with the taste of water^{6,7}, consumption of common diuretics such as caffeine⁸ and alcohol, participation in exercise⁹ and environmental conditions.⁵ Dehydration amounting to as little as a 2 per cent loss of body weight results in impaired physiological responses and performance.^{10–16} Fluid consumption may also be related to urinary stone disease^{17–20}, cancers of the colon²¹, urinary tract^{22,23} and bladder²⁴, obesity^{25,26}, mitral valve prolapse²⁷, salivary gland function²⁸, mental performance²⁹; and overall health in the elderly.⁴

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

Current intakes in Australia

Table 1.5.1 shows the percentile distribution and mean daily intake of non-alcoholic beverages from the 1995 National Nutrition Survey.³⁰ Intakes of 'water' which included tapwater, bottled water and plain mineral waters, decreased with age, both in absolute terms (grams per day) and as a percentage of non-alcoholic beverages.

Many health authorities recommend that adults consume some six to eight glasses of fluid a day (that is, approximately 1.5 to 2 litres). The National Nutrition Survey data would suggest that the population mean intake (excluding alcoholic drinks) is within this range, but 30–40 per cent of the population did

not reach this target on the day of the survey. Water constituted 42–44 per cent of the non-alcoholic fluids consumed on the day of the survey.

Table 1.5.1 Percentile distribution and mean daily intake of non-alcoholic fluids: adults aged 19 years and over, 1995 National Nutrition Survey

	Percentile											
Beverage	5th	10th	20th	30th	40th	50th	60th	70th	80th	90th	95th	Mean
Total non-alcoholic beverages												
Males	609	837	1148	1394	1599	1825	2075	2378	2787	3462	4254	2052
Females	760	953	1218	1394	1542	1769	1996	2227	2538	3053	3541	1917
Waters ^a												
Males	—	—	—	250	439	500	750	1000	1500	2000	2800	855
Females	—	—	180	300	500	750	900	1000	1500	2000	2250	849
Tea												
Males	—	—	—	—	—	—	254	508	711	1115	1472	345
Females	—	—	—	—	63	254	508	634	761	1218	1523	453
Coffee and substitutes												
Males	—	—	—	—	154	254	472	609	761	1218	1776	475
Females	—	—	—	—	—	254	317	508	761	1015	1269	379
Fruit and vegetable juices and drinks												
Males	—	—	—	—	—	—	—	105	263	518	735	139
Females	—	—	—	—	—	—	—	73	257	368	528	109
Soft drinks ^b												
Males	—	—	—	—	—	—	—	261	392	782	1157	236
Females	—	—	—	—	—	—	—	—	260	465	750	126

– Zero.

a. Tapwater, bottled water or plain mineral water.

b. Includes soft drinks, flavoured mineral water and electrolyte drinks.

SCIENTIFIC BASIS

Body water

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

Water is an essential nutrient because it is required in amounts that exceed the body's ability to produce it. Human requirements for water are related to metabolic needs and are highly variable, depending to some extent on individual metabolism. The body must retain a minimal amount for the kidneys to maintain

a tolerable solute load. Even without perspiration, the normal daily turnover is approximately 4 per cent of total body weight in adults. In a 70-kilogram adult this is equivalent to 2.5–3.0 litres a day.³ Water losses from lungs and skin (insensible losses) are responsible for half the total water turnover², are sensitive to environmental conditions, and can be increased at high temperatures, high altitude and low humidity. During summer, when heat stress may be high, water depletion can lead to heat exhaustion, loss of consciousness and heat stroke.^{31,16} Unfit, overweight older people may be especially at risk, particularly if they are subjected to strenuous exercise. Losses from urine and stool account for the rest of the total losses.

Influence of hydration on health and disease

Among the reported health effects of chronic mild dehydration and poor fluid intake are:

- increased risk of kidney stones^{17–20}
- increased risk of urinary tract cancers^{22–24}
- increased risk of colon cancer²¹
- increased risk of childhood obesity^{25,26}
- diminished physical performance^{10–16}
- diminished mental performance²⁹
- diminished salivary gland function²⁸
- increased risk of mitral valve prolapse.²⁷

Kidney stones

Data from the United States show that approximately 12–15 per cent of the general population will form a kidney stone, or renal calculus, at some time.^{32,33} There are many risk factors for the formation of kidney stones, including age, gender, heredity, occupation, social status, geographic location, climate and diet. Of these, diet is the only one that is readily modifiable. Stone prevalence is higher in populations with low urine volume.^{17–20,32–36} Lower fluid intake leads to low urine volume and increased concentration of stone-forming salts. Studies have shown that high fluid intakes can be an effective and economical preventive strategy and can inhibit the recurrence of kidney stones.^{17–20} The Nurses Health Study³⁷, a prospective cohort study of women aged 40–65 years, and the prospective Health Professionals Follow-up Study³⁸, undertaken in men aged 45–75 years in the United States, found that an increased total fluid intake reduced the risk of stones but that the reduction varied according to the choice of fluids as well as the amount.

Cancers

Several studies have shown a direct correlation between the quantity of fluid consumed and the incidence of certain cancers. A study in Israel²² found that patients with urinary tract cancer (bladder, prostate and kidney, as well as testicle) consumed significantly less fluid than healthy controls, but no

association with a specific beverage was found. Another study, in Hawaii²³, showed that total fluid intake, and intake of tapwater in particular, had a strong inverse dose–response relationship to lower urinary tract cancer (bladder, renal-pelvis and ureter) in women. The association was stronger in smokers. Similar findings have been observed for breast and colon cancer. One US study of colon cancer²¹ showed an inverse dose-related relationship between water intake, measured as glasses per day, and the risk of colon cancer in women. Women drinking more than five glasses a day had a 45 per cent reduced risk compared with those drinking two or fewer glasses a day. Among men there was an apparent 32 per cent decrease in risk with increasing water consumption, although it was not statistically significant.

Results from the Health Professionals Follow-up Study²⁴ also showed that total daily fluid intake was inversely associated with the risk of bladder cancer. Consumption of water was shown to contribute to the lower risk of bladder cancer when compared with other fluids.

Other public health factors

It is often reported that drinking fluids has a satiating effect that makes people feel ‘fuller’ and eat less and may help in controlling overweight and obesity. Two studies from the United States, one in children²⁵ and one in adults³⁹, lend support to this assertion. Reviewing the results of liquid intake in relation to childhood obesity and disease, Levine²⁶ concluded, among other things, that replacing drinks in the diet with milk and water would help control weight and greatly improve the overall health of children and adolescents in the United States. The relevance of this to adults is not known.

Oral health may also be affected by fluid consumption. Apart from the beneficial affects of fluoride added to tapwater in many communities in Australia, fluid intake can affect saliva production, and saliva, which is primarily water, is essential for maintenance of oral health. Decreased body water has been associated with salivary dysfunction, especially in older adults. One investigation²⁸ found, however, that decreased salivary gland function was associated with dehydration, independent of age.

Numerous studies have shown diminished thirst sensations in the elderly. Despite the fact that these changes may be a normal part of the ageing process, the consequences of dehydration in the elderly are serious, ranging from constipation to cognitive impairment and functional decline. This problem is specifically addressed in the Dietary Guidelines for Older Australians.⁴⁰ The relevance to people in young to mid-adulthood is unclear.

Mental and physical performance

The effect of dehydration on cognition has not been well studied, but it is likely that, since physical impairment is caused by hypohydration, mental performance will also be impaired.^{41,42} One study of mental performance under different levels

of stress-induced dehydration in 11 acclimatised subjects in India showed that, after recovery from exercise in the heat, subjects demonstrated significant and progressive reductions in arithmetic performance, short-term memory and visual-motor tracking at a deficit of 2 per cent body fluid compared with the hydrated state.²⁹ The relevance of this to the general population in Australia is unclear.

The effects of hydration on physical performance are well studied and well known.^{10–14} When exercise is performed in conditions of excessive heat or cold, low humidity or high altitude, fluid losses increase. Dehydration producing as little as a 1 per cent decrease in body weight impairs physiologic and performance measures during continuous exercise, and even minor body mass loss from dehydration negatively affects heart rate, tolerance times and stroke volume in light and heavy exercise in the heat.

Special groups

Older adults

Kidney function can decline as part of the normal ageing process: kidney mass decreases, and there are declines in renal blood flow and glomerular filtration rate, distal renal tubular diluting capacity, renal concentrating capacity, sodium conservation, and renal response to vasopressin. This, together with hormonal changes and factors such as decreased thirst perception, medication, cognitive changes, limited mobility and increased use of diuretics and laxatives, makes older adults a group of particular concern.⁴⁰

Pregnant and lactating women

A pregnant woman has a slightly increased water requirement because of expanding extracellular fluid space and the needs of the foetus and the amniotic fluid. The UK National Research Council has calculated that, compared with the non-gravid state, pregnant women require an extra 30 millilitres of fluid a day.² This does not take into account any increase in fluid loss from increased heat production and perspiration, especially in the summer months. It is thus likely that this is a minimal additional requirement.

A lactating woman must replace fluid lost in breastmilk. Eighty-seven per cent of breastmilk is water, and the average milk production in the first six months of lactation is 750 millilitres a day.^{2,43} The fluid need is therefore 750–1000 millilitres a day above basic needs.

PRACTICAL ASPECTS OF THIS GUIDELINE

Water

Plain water is a safe and low-cost way to ensure adequate fluid ingestion without additional dietary energy. The National Health and Medical Research Council has developed comprehensive guidelines on water standards for drinking.⁴⁴ Given

the high levels of overweight and obesity in the Australian population, this is an important consideration. Most tapwater in Australia is fluoridated, which has been shown to be a safe and effective public health measure. Fluoridation of tapwater provides an additional benefit for development of strong teeth and bones. It is therefore a very good choice to ensure adequate hydration. Bottled waters are a useful alternative when access to tapwater is limited, but not all bottled waters contain fluoride. In terms of health outcomes, water appears to have advantages over many other fluids, especially those that have a diuretic action—for example, alcoholic drinks; caffeinated drinks such as coffee, tea and cola; and certain ‘energy’ drinks (with guarana). Alcoholic drinks in excess can also have other health consequences (see Section 1.8).

Milk or alternatives

The value of milk as a component of the diet is discussed in Section 1.4. Milk is an important supplier of calcium, protein, and a number of other valuable nutrients and should be included as part of the fluid intake. Use of reduced-fat or skim milks will reduce additional fat and energy intake whilst retaining the calcium contribution. Soy milks are available for people who do not want to or cannot use cow’s milk, but calcium- and vitamin B₁₂-fortified varieties should be chosen. Again, it is necessary to be aware that, as with cow’s milk, consumption of soy milks will add energy to the diet.

Fruit and vegetable juices

Fruit and vegetable juices can be a useful source of vitamin C, potassium and folate. They add variety to the diet and fluid intake. There is, however, no reason for obligatory consumption of fruit and vegetable juices if fruits and vegetables are consumed in line with Section 1.1.

Caffeine and caffeine-containing drinks

Caffeine is a naturally occurring substance found in many plant species. It is known to stimulate the central nervous system, and at high doses it can cause nervousness, irritability, anxiety and disturbances of heart rate and rhythm. Its influence on blood pressure, coronary circulation and secretion of gastric acids, as well as its diuretic effect, have also been documented.⁴⁵ Caffeine is found in foods and beverages such as tea, coffee, chocolate, cola-type soft drinks and ‘energy drinks’. Coffee contains about three or four times more caffeine than an equal volume of cola soft drink.

Tea and coffee

Coffee and, to a lesser extent, tea contain substantial amounts of caffeine, which can have unwanted stimulant effects in susceptible people and acts as a diuretic. Tea (especially green tea but also black tea) and coffee also contain substantial amounts of polyphenols, antioxidants which are claimed to offer benefits in

terms of protection against cancer and cardiovascular disease. Green tea is the most potent source of polyphenols and has been used for many years in Asian cultures. The literature on its benefits in relation to specific health outcomes in humans is sparse, but it has been speculated that the effect of its consumption may depend on the causative factors of the condition of concern.⁴⁶ Because of its widespread use in Australia, black tea is a major contributor to polyphenols in the Australian diet⁴⁷, rivaling the contribution of fruits and vegetables, which are traditionally considered the primary source. Tea and coffee are often consumed with added milk and sugar, and the additional energy intake from these sources can be substantial in some people.

Energy drinks

The term *energy drinks* refers to a group of non-alcoholic water-based beverages characterised by the addition of so-called energy-enhancing ingredients, among them a number of water-soluble B vitamins, amino acids and other substances, and caffeine. Most energy drinks contain caffeine levels of up to 80 milligrams per 250 millilitres, the equivalent of a cup of strong coffee.⁴⁸ Energy drinks are a relatively new product in the marketplace in Australia, although they are well established in Europe and the United States. Current data on their consumption by adults in Australia are limited.

Soft drinks and cordials

Moderation is recommended in relation to consumption of soft drinks and cordials containing added sugars. (This is discussed in more detail in Section 1.9.) Although they provide fluid, drinks of this kind add energy to the diet without additional nutrient value. Occasional use of low-joule drinks can, however, bring variety to the diet.

Alcohol

Alcoholic drinks are discussed in detail in Section 1.8. In relation to hydration, it is important to note that alcohol is a strong diuretic and can increase fluid loss from the body. It is also very high in dietary energy.

RELATIONSHIP TO OTHER GUIDELINES

Enjoy a wide variety of nutritious foods

Foods contain water, and a varied diet will contribute to water requirements. Some variety is also recommended in fluid consumption, but water is the preferred drink for maintaining adequate hydration.

Consume only moderate amounts of sugars and foods containing added sugars

Intake of fluids containing substantial amounts of added sugars should be moderated.

Prevent weight gain: be physically active and eat according to your energy needs

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

CONCLUSION

Adequate fluid consumption is an integral component of a healthy diet. Water is a good source of fluids as it can hydrate without adding additional energy to the diet. Nevertheless other drinks such as milks, fruit juices, low energy soft drinks beverages can add variety and in some cases (eg milks and juices) can add valuable nutrients to the diet. Intakes of fluids containing substantial amounts of added sugars should be moderated.

EVIDENCE

There is Level II evidence (references 17 and 19) and Level III evidence (references 18, 20, 37 and 38) linking water consumption and urinary volume with the occurrence of kidney stones. There is Level III evidence for a link between fluid consumption and cancer of the urinary tract (reference 22), between total fluid intake (tapwater in particular) and lower urinary tract cancer in women (reference 23), between water intake and colon cancer (reference 21), between fluid intake (water in particular) and bladder cancer (reference 24), and between dehydration and salivary gland function (reference 28) and Level IV evidence for an association between dehydration and mental performance (reference 29).

REFERENCES

1. Kleiner SM. Water: an essential but overlooked nutrient. *J Amer Diet Assoc* 1999;99(2):200–6.
2. Food and Nutrition Board. *Recommended dietary allowances*. 10th edn. Washington, DC: National Academy Press, 1989.
3. Food and Nutrition Board. *Recommended dietary allowances*. 9th edn. Washington, DC: National Academy Press, 1980.

4. Sansevero AC. Dehydration in the elderly: strategies for prevention and management. *Nurse Pract* 1997;22:41–2,51–7,63–72.
5. Sagawa S, Miki K, Tajima F, Tanaka H, Choi JK, Keil LC et al. Effect of dehydration on thirst and drinking during immersion in men. *J Appl Physiol* 1992;72:128–34.
6. Weissman AM. Bottled water use in an immigrant community: a public health issue? *Am J Publ Hlth* 1997;87:1379–80.
7. Meyer F, Bar-Or O, Passe D, Salsberg A. Hypohydration in children during exercise in the heat: effect on thirst, drink preferences and rehydration. *Int J Sport Nutr* 1994;4:22–35.
8. Neuhauser-Berthold M, Beine S, Verwied SC, Luhrmann PM. Coffee consumption and total body water homeostasis as measured by fluid balance and bioelectrical impedance analysis. *Ann Nutr Metab* 1997;41:29–36.
9. Convertino VA, Armstrong LE, Coyle EF, Mack GW, Sawka MN, Senay LC et al. American College of Sports Medicine position stand: exercise and fluid replacement. *Med Sci Sports Exerc* 1996;28:i–vii.
10. Torranin C, Smith DP, Byrd RJ. The effect of acute thermal dehydration and rapid rehydration on isomeric and isotonic endurance. *J Sports Med Phys Fitness* 1979;19:1–9.
11. Armstrong LE, Costill DL, Fink WJ. Influence of diuretic-induced dehydration on competitive running performance. *Med Sci Sports Exerc* 1985;17:456–61.
12. Sawka MN, Pandolf KR. Effects of body water loss on physiological function and exercise performance. In: Gisolfi CV, Lamb DR, eds. *Fluid homeostasis during exercise*. Carmel, IN: Benchmark Press, 1990:1–38.
13. Kristel-Boneh E, Blusman JG, Chaemovitz C, Cassuto Y. Improved thermoregulation caused by forced water intake in human desert dwellers. *Eur J Appl Physiol* 1988;57:220–4.
14. Brooks GA, Fahey TD. *Exercise physiology: human bioenergetics and its applications*. New York: John Wiley & Sons, 1984.
15. Brouns F. Nutritional aspects of health and performance at lowland and altitude. *Int J Sports Med* 1992;13(suppl. 1):S100–S106.
16. Cheung SS, McLennan TM. Influence of hydration status and fluid replacement on heat tolerance while wearing NBC protective clothing. *Eur J Appl Physiol Occupat Physiol* 1998;77:139–48.
17. Borghi L, Meschi T, Amato F, Briganti A, Novarini A, Gianninin A. Urinary volume, water and recurrences in idiopathic calcium nephrolithiasis: a five year randomised prospective trial. *Urology* 1996;13:33–8.
18. Hughes J, Norman RW. Diet and calcium stones. *Can Med Assoc J* 1992;146:137–43.

19. Iguchi M, Umekewa T, Ishikawa Y, Katayama Y, Kodama M, Takada M et al. Clinical effects of prophylactic dietary treatment on renal stones. *J Urology* 1990;144:229–32.
20. Embon OM, Rose GA, Rosenbaum T. Chronic dehydration stone disease. *Br J Urology* 1990;66:357–62.
21. Shannon J, White E, Shattuck AL, Potter JD. Relationship of food groups and water intake to colon cancer risk. *Cancer Epidemiol Biomarkers Prev* 1996;5:495–502.
22. Bitterman WA, Farhadian H, Abu S-C, Lerner D, Amoun H, Krapf D et al. Environmental and nutritional factors significantly associated with cancer of the urinary tract among different ethnic groups. *Urologic Clin North Am* 1991;18:501–8.
23. Wilkens LR, Kadir MM, Kolonel LN, Nomura AM, Hankin JH. Risk factors for lower urinary tract cancer: the role of total fluid consumption, nitrites and nitrosamines, and selected foods. *Cancer Epidemiol Biomarkers Prev* 1996;5:161–6.
24. Michaud DS, Spiegelman D, Clinton SK, Rimm EB, Cuhán GC, Willett WC et al. Fluid intake and risk of bladder cancer in men. *N Engl J Med* 1999;340(18):1390–7.
25. Vido L, Facchin P, Antonello L, Gobber D, Rigon F. Childhood obesity treatment: double blinded trial on dietary fibres (glucomannan) versus placebo. *Padiatrie und Padologie* 1993;28:133–6.
26. Levine B. Role of liquid intake in childhood obesity and related diseases. *Curr Concepts Perspect Nutr* 1996;8(2).
27. Lax D, Eicher M, Goldberg SJ. Mild dehydration induces echocardiographic signs of mitral valve prolapse in healthy females with prior normal cardiac findings. *Am Heart J* 1992;124:1533–40.
28. Ship JA, Fischer DJ. The relationship between dehydration and parotid salivary gland function in young and older healthy adults. *J Gerontol* 1997;52A:M310–M319.
29. Gopinathan PM, Pichan G, Sharma VM. Role of dehydration in heat stress–induced variations in mental performance. *Arch Environ Hlth* 1988;4:15–17.
30. Australian Bureau of Statistics. *National Nutrition Survey: foods eaten, Australia, 1995*. Canberra: ABS, 1999.
31. 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, IN: Benchmark Press, 1988:305–60.
32. Curhan GC, Curhan SG. Dietary factors and kidney stone formation. *Comp Ther* 1994;20:485–9.
33. Goldfarb S. The role of diet in the pathogenesis and therapy of nephrolithiasis. *Endocrinol Metab Clin North Am* 1990;19:805–20.

34. Pin NT, Ling NY, Siang LH. Dehydration from outdoor work and urinary stones in a tropical environment. *Occ Med* 1992;42:30–2.
35. Hiatt RA, Ettinger B, Caan B, Queensberry CP, Duncan D, Ciron JTY. Randomised control trial of a low animal protein, high fibre diet in the prevention of recurrent calcium oxalate kidney stones. *Am J Epid* 1996;144:25–33.
36. Ackermann D. Prophylaxis in idiopathic calcium urolithiasis. *Urologic Res* 1990;18(suppl. 1):S37–S40.
37. Curhan GC, Willet WC, Speizer FE, Stampfer MJ Beverage use and risk for kidney stones in women. *Ann Intern Med* 1998;128(7):534–40.
38. Curhan GC, Willett WC, Rimm EB, Spiegelmann D, Stampfer MJ. Prospective study of beverage use and risk of kidney stones. *Am J Epidemiol* 1996;143(3):240–7.
39. Wadhwa NK, Friend R, Gaus V, Taylor KL, Schneider MS. Weight reduction and fluid intake in an obese and fluid noncompliant ESRD patient. *Clin Nephrol* 1996;45:320–4.
40. National Health and Medical Research Council. *Dietary guidelines for older Australians*. Canberra: NHMRC, 1999.
41. Burke LM. Fluid balance during team sports. *J Sports Sci* 1997;15:287–95.
42. Salmon P. Nutrition, cognitive performance and mental fatigue. *Nutrition* 1994;10:427–8.
43. Dusdieker LB, Stumbop PJ, Booth BM, Wilmoth RM. Prolonged maternal fluid supplementation in breast feeding. *Pediatrics* 1990;86:737–40.
44. National Health and Medical Research Council & Agriculture and Resource Management Council of Australia and New Zealand. *Australian drinking water guidelines*. Canberra: Australian Government Publishing Service, 1996.
45. Australia New Zealand Food Authority. *Application A344—caffeine in non-alcoholic beverages: full assessment report and regulation impact statement*. Canberra: ANZFA, 1999.
46. Bushman JL. Green tea and cancer in humans: a review of the literature. *Nutrition and Cancer* 1998;31(3):151–9.
47. Beaumont-Smith N, Record S, Baghurst K. *Beverage consumption: results from the 1995/6 National Nutrition Survey*. Adelaide: CSIRO, 1999.
48. Australia New Zealand Food Authority. *Application A394—formulated caffeinated beverages: full assessment report and regulation impact statement*, Canberra: ANZFA, 2000.

1.6 LIMIT SATURATED FAT AND MODERATE TOTAL FAT INTAKE

A Stewart Truswell

TERMINOLOGY

Fats

Chemically, most of the fats in foods are triglycerides, made up of a unit of glycerol (glycerine) combined with three fatty acids, which may be the same or different. The differences between one fat and another are largely a consequence of the fatty acids they contain, which together make up 90 per cent of the weight of the molecule. Fats in the diet can be 'visible' or 'invisible'. Among the 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: it is part of the cell membrane of all cells, part of the myelin in the brain and nervous system, and the starting material for synthesis in the body of bile acids and adrenocortical and sex hormones. Cholesterol can, however, accumulate in blood and in the inner walls of arteries, leading to disease.

Saturated fats

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

Mono-unsaturated fats

In mono-unsaturated fats the main fatty acid is oleic acid (18:1), which has one (*mono*) double (unsaturated) bond. Olive, canola and peanut oils are rich in oleic acid.

Polyunsaturated fats

In polyunsaturated fats the main fatty acid contains two or more (*poly*) double (unsaturated) bonds. These fats are liquid at room temperature; that is, they are oils. The most common polyunsaturated fatty acid is linoleic acid (18:2); its double bonds are in the n-6 position, and it occurs in seed oils- for example, sunflower oil, safflower oil and corn oil. Smaller amounts of polyunsaturated fatty acids with double bonds in the n-3 position also occur in the diet; best known are those in fatty fish, their names abbreviated to EPA (20:5 eicosapentaenoic) and DHA (22:6 docosahexaenoic). Another n-3 polyunsaturated fatty acid, ALA (18:3 alpha linolenic), occurs in small amounts in leafy vegetables; there is more of it in canola oil and most in flaxseed oil.

Dietary cholesterol

Cholesterol, chemically a sterol, occurs in all the cell membranes of land animals. Brains and egg yolks are very rich in it; oils and fats from plants never contain it. Eating cholesterol does not necessarily increase cholesterol in human blood plasma because when it is absorbed the liver tends to reduce its own endogenous cholesterol synthesis: about half the body's cholesterol is made in the body from acetate.

Plant sterols

Also called *phytosterols*, plant sterols are chemically very similar to cholesterol but with a small difference in their chemical structure (in the side chain). They occur in oils from plants - for example, in nuts and seeds, although they may be taken out by refining and, when eaten, interfere competitively with absorption of cholesterol from the intestine. To make use of this effect, some margarines containing extra plant sterols have recently been introduced.

Trans-fatty acids

Trans-fatty acids are a form of unsaturated fatty acid that is straight at a double bond rather than bent, as in the usual *cis* form. They are not common in nature but are formed during some manufacturing processes, such as when edible oils are hydrogenated to make hard margarines. Small amounts of trans-fatty acids occur naturally in meats and dairy foods.

Fat replacers

There are two groups of fat replacers:¹

- Fat substitutes resemble fats and can replace fats in food; an example is sucrose polyester. They contain fatty acids but they have been joined to the centre of the molecule with chemical links that cannot be digested by human enzymes. They provide no dietary energy when eaten.

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

BACKGROUND

The first *Dietary Guidelines for Australians*², published in 1982, recommended, ‘Avoid eating too much fat’—that is, total fat. The type of fat was not considered, unlike the 1977 *Dietary Goals for the United States*³, which recommended 10 per cent of total energy from saturated fats, 10 per cent from mono-unsaturated fats, and 10 per cent from polyunsaturated fats.

In the second edition of *Dietary Guidelines for Australians*⁴, published in 1992, the guideline had evolved to ‘Eat a diet low in fat and, in particular, low in saturated fat’. The more recent *Dietary Guidelines for Older Australians*⁵, published in 1999, moved further and recommended that older adults ‘Eat a diet low in saturated fat’. The text supporting this last guideline explains that low-fat diets do not provide health benefits at the two ends of life.

The 1995 National Nutrition Survey⁶ provided an estimate of the present fat intake in Australia. Mean total fat was about one-third of total energy, with saturated fatty acids (SFAs) around 12.5 per cent of energy, polyunsaturated fatty acids (PUFAs) around 4.5 per cent, and mono-unsaturated fatty acids (MUFAs) around 11.5 per cent (see Table 1.6.1).

Table 1.6.1 Percentage of total energy (including alcohol) coming from various fats: 1995 National Nutrition Survey⁶, by age and gender

Gender	Age (yrs)									
	2–3	4–7	8–11	12–15	16–18	19–24	25–44	45–64	65+	19+
Males										
SFAs	15.3	14.5	13.9	14.2	13.7	13.2	12.9	12.9	12.0	12.6
MUFAs	10.7	11.3	11.4	11.6	12.1	11.9	11.7	11.7	11.3	11.7
PUFAs	3.8	4.1	4.3	4.2	4.4	4.3	4.5	4.6	4.6	4.5
Total fatty acids	33.2	32.9	33.2	33.6	33.6	33.2	33.0	32.3	31.3	32.6
Females										
SFAs	16.1	14.4	14.7	13.8	13.2	12.7	12.8	11.9	12.3	12.5
MUFAs	10.9	11.1	11.8	11.5	10.9	11.4	11.6	11.3	11.2	11.4
PUFAs	3.7	4.0	4.3	4.3	4.0	4.6	4.6	4.7	4.7	4.6
Total fatty acids	34.1	32.4	34.2	33.2	31.9	32.8	33.0	32.1	32.1	32.6

Note: SFAs, MUFAs and PUFAs are groups of fatty acids. Combined, they represent approximately 90 per cent of total fats; the remainder is glycerol.

The estimates in the table are very similar to those recorded in the 1983 Australian National Dietary Survey.⁷ Total fat (mean) then contributed 36.6 per cent of the energy in men and 37 per cent in women. The median fat consumption per day for men aged 19 years and over was 105 grams in 1983 and 98.5 grams in 1995; for women aged 19 years and over, it was 70 grams in 1983 and 67.6 grams in 1995. There thus appears to have been a small reduction in total fat consumption between 1983 and 1995. The median ratios between PUFAs, MUFAs and SFAs were almost identical in the two surveys (for men, in 1983 the ratio was 0.36:0.93:1.0 and in 1995 it was 0.36:0.93:1.0; for women, in 1983 the ratio was 0.36:0.92:1.0 and in 1995 it was 0.37:0.91:1.0). In the 1995 survey, the 10th to 90th percentile distribution of saturated fat ranged from 23 to 58 grams a day in men aged 19 years and over; for women in the same age group it range from 16 to 40 grams a day. The intake of polyunsaturated fat ranged from 19 to 21 grams a day in men and from 7 to 15 grams a day in women.

The main food groups providing saturated fat in the 1995 survey were—in descending order, for males and females aged 19 years and over—milk products and dishes, 26.9 per cent (cheese, 8.0 per cent); cereal-based products and dishes, 20.3 per cent; meat, poultry and game products and dishes, 20.6 per cent; fats and oils, 8.9 per cent; potatoes (especially chips), 5.9 per cent; chocolate, 2.7 per cent; and fish, 1.8 per cent. The main food groups providing polyunsaturated fats in the 1995 survey were fats and oils, 20.5 per cent (margarine, 17.8 per cent); cereal-based products and dishes, 15.2 per cent; meat, poultry and game products and dishes, 15.3 per cent; breads and breakfast cereals, 13.6 per cent; vegetable products and dishes, 11.7 per cent; nuts and seeds, 4.9 per cent; and fish, 4.4 per cent.

The main food groups providing mono-unsaturated fat in the 1995 survey were meat, poultry and game products and dishes, 25.7 per cent; cereal-based products and dishes, 18.0 per cent; milk products and dishes, 12.7 per cent; fats and oils, 12.2 per cent (margarine, 8.2 per cent); and potatoes, 7.0 per cent.

SCIENTIFIC BASIS

Dietary fat and overweight and obesity

Overweight and obesity have been increasing rapidly in Australia, with particular acceleration in the last two decades.^{8–10,6} Chapter 2 discusses this in more detail.

Many Australians have become obese in the last 20 years (see Table 1.6.2), even though fat intakes do not appear to have increased (in terms of either grams per day or percentage of energy) and may have declined slightly. This dramatic increase in obesity and overweight has also occurred in most other countries¹¹; it is the outstanding challenge for nutrition research and practice today.

Table I.6.2 Overweight and obesity in Australian adults, by gender

Survey	Males %	Females %
NHF 1980 ⁸		
Overweight	34.1	24.5
Obese	7.2	7.0
Total	41.3	31.5
NHF 1983 ⁹		
Overweight	36.2	26.4
Obese	6.4	8.7
Total	42.6	35.1
NHF 1989 ¹⁰		
Overweight	38.6	22.4
Obese	9.3	11.1
Total	47.9	33.5
NNS 1995 ⁶		
Overweight	45.0	29.0
Obese	18.5	18.0
Total	63.7	47.0

Notes: NHF = National Heart Foundation (surveys in capital cities); NNS = National Nutrition Survey (random sample of entire nation from a probability sample of households). Obesity is defined as BMI greater than 30 kg/m². Overweight is defined as BMI greater than 25kg/m²

Fat is the macro-nutrient with the highest energy value per unit weight, at 2.25 times that of carbohydrates and proteins. About half the fats and oils in the Australian diet are 'invisible', being present in baked foods, sauces, confectionery, cheese, snack foods and nuts, and as coating on fried foods. People like the taste and mouth feel of fat and enjoy eating foods containing it, so there is passive over-consumption of fat.

There is now evidence that Rubner's isodynamic law—energy from carbohydrate ... energy from fat ... energy from protein—does not quite hold in practice. Kilojoules from different macro-nutrients are not really equal. Carbohydrates or proteins which are eaten above daily requirement stimulate their own oxidation and are only partly stored short term, and alcohol is completely metabolised. Additional fats, however, are nearly all stored in the body.¹² In addition, Blundell and Macdiarmid have shown in the appetite laboratory that foods high in fat have high palatability, together with weak satiation, compared (kilojoule for kilojoule) with carbohydrate and protein.¹³ Fatty meals also induce lower thermic response than meals high in carbohydrate or protein, or both.¹⁴

These effects of fat suggest either that obese people eat more fat than their lean friends or that obese people are in some way more susceptible to the effects of fat. There is some epidemiological evidence that fat consumption is associated

with overweight.¹⁵ In the MONICA surveys, the median body mass index of *communities* did not increase in line with FAO figures for percentage of energy available from fat, but nine out of 12 cross-sectional studies have shown higher fat intakes in *individuals* with high BMI.¹⁵ Prospective studies have given inconsistent results.¹⁵ This could mean that fat is leading to obesity only in some communities. One problem with food intake measurements is whether they are taken at a time of gaining weight. Another is that obese people under-report their food intake, especially fat intake¹⁶, and no biomarker is available to indicate total fat intake objectively. Some people do not like, or consume, fatty food and others do. There is evidence that people who regularly consume high-fat diets have satiation responses that differ from those of people who regularly consume lower fat diets.¹⁷

In a careful controlled trial in Denmark¹⁸, obese subjects first lost 12.6 kilograms on low-energy diets, then for the weight-maintenance phase they were randomised to an ad lib low-fat, high-carbohydrate diet or a prescribed fixed-energy diet (≈7.8 megajoules a day) for a year. Regain of weight was 0.3 kilograms on the low-fat diet and 4.1 kilograms on fixed-energy diet. A year after the end of the trial, at follow-up the low-fat group had regained 5.4 kilograms and the fixed-energy diet group had regained 11.3 kilograms (almost all the weight originally lost).

In a six-month trial in the Netherlands¹⁹, 200 people aged about 40 years and a little overweight but not obese were asked to eat usual-fat or low-fat unrestrained diets, or restrained usual-fat diets or low-fat diets. Body weights rose with usual-fat unrestrained; they stayed the same with low-fat unrestrained; and they fell with usual-fat restrained and low-fat eating. This nicely demonstrates the separate effects of low-fat and restrained eating. Of a registry of 784 previously obese people who had successfully lost 30 pounds (13.6 kilograms) and maintained their weight for at least a year²⁰, 33 per cent had limited the percentage of daily energy from fat and 25 per cent had counted fat grams.

The World Health Organization¹¹, the British Obesity Task Force²¹, the Scottish Intercollegiate Guidelines Network²² and a specialist review²³ all emphasise the major role of fat consumption in the development of obesity and of cutting fat intake in the dietary management of obesity.

None of this should be taken to mean that reducing fat intake is all that is needed to deal with the contemporary epidemic of overweight and obesity. Physical activity is important²⁴, and energy density (the concentration of energy in a food) is probably the primary factor from the energy-intake side of the equation. Fat content is, however, closely linked to energy density in the Australian diet. Water content and, to a lesser extent, fibre content also play a role in determining the energy density of foods. This is discussed further in Chapter 2, which also emphasises the role of physical activity in weight control.

Dietary fat and type 2 diabetes mellitus

Diabetes affects an increasing proportion of Australians²⁵; Aboriginal and Torres Strait Islander peoples are especially susceptible. The common form of diabetes is type 2, or non-insulin dependent, diabetes mellitus. Overweight and obesity are the strongest diet-related risk factors for type 2 diabetes. Since dietary (total) fat is energy dense and high fat intakes can be associated with overweight and obesity (as discussed), the policy of moderation in fat intake aims to reduce the incidence of diabetes as well as obesity.

There is evidence that high-fat diets increase insulin resistance. In a study of 123 glucose-intolerant individuals, intake of total fat, saturated fats and mono-unsaturated fat—but not polyunsaturated fat—was associated with deterioration of glucose tolerance.²⁶ In a 10-year follow-up in Sweden, people were more likely to develop type 2 diabetes if they started with high saturated fatty acids or lower linoleate in serum cholesterol esters.²⁷ In the US Nurses Health Study, 2500 subjects developed type 2 diabetes over 14 years. The risk of diabetes was positively (significantly) related to saturated trans-fat and mono-unsaturated fat intakes and negatively related to polyunsaturated fat intake.²⁸

Coronary heart disease is the major cause of death in type 2 diabetics, and diabetic women lose their relative protection from CHD. In the treatment of people with established diabetes, both the European Association for the Study of Diabetes and the major US textbook of medicine²⁹ recommend that saturated fat should provide less than 7 to less than 10 per cent of energy and polyunsaturated fat not more than 10 per cent of energy. With protein at 10–20 per cent of energy intake, the remainder of the energy should come from a combination of carbohydrates (preferably high in fibre, low in glycaemic index) and mono-unsaturated fat and oil.

Saturated and trans-fats and coronary heart disease

Of the risk factors for CHD that can be influenced by diet, plasma LDL cholesterol—which is reflected in plasma total cholesterol concentration—remains the best established. It has been found to be a significant risk factor in at least 50 prospective cohort studies involving more than 600 000 subjects in 18 countries. The importance of plasma cholesterol has been confirmed by trials with ‘statin’ cholesterol-lowering drugs, which are showing reductions in coronary events, and in all-causes mortality, even among people starting with average US plasma cholesterol levels. Some of these trials, however, are in high risk groups such as those who have already experienced myocardial infarction.³⁰

Saturated fat is the strongest dietary determinant of plasma LDL concentration. This has been demonstrated repeatedly in controlled human experiments.^{31–37} Mensink and Katan³⁵ included 27 trials in their meta-analysis. Hegsted et al.³⁶ reviewed 248 metabolic experiments and 96 field observations. Clark et al.³⁷ analysed only metabolic ward experiments, 395 with solid foods and 32 with

liquid formula diets; 5076 subjects participated in the studies described in the 71 publications they analysed.

To the extent that it has been possible to separate the effects of individual fatty acids, it is only lauric (12:0), myristic (14:0) and palmitic (16:0) that have the effect of raising LDL and total cholesterol. Stearic (18:0) does not appear to have this effect^{33,37,38}, and lauric appears less active than palmitic and myristic.³⁸

The cholesterol-raising effect of trans-fatty acids³⁹ was rediscovered in the early 1990s⁴⁰ and confirmed in different laboratories.^{41,42} Unlike C12 to C16 saturated fatty acids, trans-fatty acids are reported to lower HDL cholesterol and to increase Lp(a).

In determining connections between diet and CHD, prospective cohort studies are considered the most reliable observational epidemiology. The first report of a positive association between saturated fat intake and subsequent CHD mortality was the classic Seven Countries Study.⁴³ The association between saturated fats and coronary events was supported by results from the Nurses Health Study⁴⁴, the Health Professionals Follow-up Study⁴⁵—both in the United States—and in a cohort study in Finland, where saturated fat intake was ascertained from plasma phospholipid palmitate.⁴⁶

Trans-fatty acids were reported to be associated with CHD in the Nurses Health Study⁴⁷, although the reliability of the food composition data was queried.⁴⁸ Re-analysis of dietary data and longer follow-up of the nurses cohort supported Willet et al.'s earlier finding on trans-fatty acids.⁴⁹ However, adipose tissue percentages of trans-fatty acids have not been associated with CHD cases in Europe.^{50,51}

The UK Committee on the Medical Aspects of Food Policy as early as 1984 claimed that trans-fatty acids had an effect equivalent to that of saturated fats in relation to CHD outcomes.⁵² The NHMRC's working party on the role of polyunsaturated fats in the Australian diet⁵³ came to the same conclusion; so did the National Heart Foundation of Australia's 1994 review of dietary fats and blood cholesterol.⁵⁴

The National Heart Foundation's 1999 position statement on dietary fats⁵⁵, which was based on a thorough review of the literature and ranked the strength of the evidence, recommended that saturated fatty acids and trans-fatty acids together contribute no more than 8 per cent of total energy intake. Trans-fatty acid intakes in Australia have not been accurately quantified. In the early to mid-90s they were estimated at 2 per cent⁵⁶ but, now that they have been virtually eliminated from our soft margarines, average intake is believed to be between 1 and 2 per cent. The *Dietary Guidelines for Americans 2000* recommends a saturated fat intake of 10 per cent of energy⁵⁷, the figure recommended by the NHMRC in 1992.⁵³ This is a feasible target for the Australian average to reach from the present level of 12.5 per cent. Major remaining sources of trans-fatty acids should be identified and reduced.

The second edition of the *Dietary Guidelines for Australians*⁴, quoting the NHMRC working party on polyunsaturated fats⁵³, recommended that total fat and saturated fat be reduced by substituting combinations of:

- 'complex carbohydrates'
- n-6 polyunsaturated fats
- mono-unsaturated fats
- oily fish.

The case for n-6 polyunsaturated fatty acids

In human nutrition, linoleic acid (18:2) is almost synonymous with n-6 polyunsaturated acids. Oils rich in n-6 PUFA—that is, linoleic acid—consistently lower plasma total and LDL cholesterol^{31–37}, even when the oil increases the fat intake.^{58,59} In experimental work on animals, rats and marmosets, long-term feeding of diets rich in n-6 PUFA has been found to reduce the frequency of dangerous arrhythmias when a coronary artery is ligated.^{60,61,62}

Three prospective cohort studies estimated linoleic acid intake from plasma concentrations. In all three there was a negative association with subsequent coronary heart disease.^{46,63,64} Nine prospective cohort studies estimated the intake of fatty acid types from food-frequency questionnaires and followed subjects to see who developed coronary heart disease. In two of these studies (the US Nurses Study^{44,49} and the Western Electric Study in Chicago⁶⁵) significant inverse associations of PUFA, or 18:2, with CHD were found. In the other six studies no significant association was found; in some of these, however, the dietary method may not have had enough detail of fat composition (for example, 24-hour recall) or the range of PUFA intakes may have been too small.

Eight intervention trials centred on coronary heart disease^{66–73} have assessed the effects of substitution of polyunsaturated fats (that is, n-6 fats) for saturated fat in the experimental group (see Table 1.6.3). Diet was the only lifestyle factor changed. Except for the two Helsinki mental hospital trials (one in men, the other in women), which exposed all the residents to experimental and control diets in turn, all the subjects were randomised. (In other trials not detailed here there were other dietary changes: reduced total fat; increased cereals, vegetables and fruits or increased n-3 PUFA; or the dietary prescription was not clearly described.)

These eight intervention trials involved 17 529 subjects in four countries, half in an 'increased polyunsaturated–saturated fat ratio' (or P–S) group and half in a 'usual diet' control group. There were 566 new coronary events in the combined increased P–S experimental diet group, which represented only 81 per cent of the 699 CHD events in the control group. (A meta-analysis show this to be significant, at $p < 0.05$ by Wilcoxon test.) All-causes mortality was slightly lower in the experimental group—1291 against 1360, or 95 per cent—but this was not statistically significant.⁷⁴

Table 1.6.3 Dietary intervention trials for prevention of CHD with increased polyunsaturated and decreased saturated fat (P–S) ratio

	Type of trial	n	% reduction of total plasma cholesterol x years of study	CHD events: intervention/control	Total death: intervention/control
Rose et al. ⁶⁶	SD	52	–9 x 2	12/6	5/1
MRC ⁶⁷ : soybean oil	SD	393	–15 x 3.4	45/51	28/31
Dayton et al. ⁶⁸ :VA	PD	846	–13 x 7	60/78	174/177
Leren ⁶⁹ : Oslo	SD	412	–14 x 5	79/94	101/108
Helsinki ^a					
Men ⁷⁰	PD	1 900	–15 x 12	34/76	188/217
Women ⁷¹	PD	2 836	–13 x 12	73/129	415/456
DART ⁷² : fat advice	SD	2 033	–3.5 x 2	132/144	111/113
Frantz et al. ⁷³					
Men	PD	4 393	–14 x 1	69/74	158/153
Women		4 664	–14 x 1	62/47	111/95
Total—all studies		17 529		566/699 (81%) p<0.05	1291/1360 (95%) n.s.

n.s. Not significant.

a. Institutions were alternated. In all the other trials subjects were randomised.

Note: PD = primary prevention trial, diet the only intervention; SD = secondary prevention trial, diet the only intervention.

The decline in deaths from coronary heart disease in Australia and the United States between the mid-1960s and 1980 (compared with static rates in the United Kingdom and Sweden in that period) was more the result of a reduction in sudden deaths outside hospital than of better survival of admitted cases of myocardial infarction.⁷⁵ The dietary change most likely to account for the decline is increased intakes of n-6 PUFA.^{76–81} Around this time, people at risk—that is, middle-aged men, some of whom had already been diagnosed with coronary heart disease—increased their PUFA intake by a larger percentage than they reduced their saturated fat intake (because PUFA intake had been much smaller). In 1961 a leading researcher⁸² in Australia was recommending corn oil and sunflower seed oil, and by 1967 soft margarines were available and recommended by the National Heart Foundation to help increase the dietary P–S ratio to 1.25.⁸³

In 1989 Rose wrote,

In the US and Australia the changing balance of unsaturated to saturated fatty acids correlates better with the fall in CHD than do changes in total or saturated fat intake. In the UK, the average dietary P/S ratio rose from 0.24

in 1980 to 0.35 in 1985, and the timing of this large change was reasonably close to the changes in CHD mortality. In the USSR, the severe and alarming increase in CHD mortality seems to have coincided with a big fall in the dietary P/S ratio ... In the light of all the evidence that has accrued ... I now think that the P/S ratio is probably one of the most potent determinants of CHD rates and trends and that its practical importance should be emphasised accordingly.⁸⁰

It was at first difficult to see a mechanism for prompt falls in sudden cardiac death in response to increased national uptakes of polyunsaturated oils and margarines. Changes in plasma cholesterol either were not well documented (as in Australia) or showed relatively small reductions (as in the United States).⁸⁴ There should be a time lag between lower plasma cholesterol, slower advance of atherosclerosis and less clinical CHD. Hetzel et al.⁷⁹ first proposed, in 1989, that prevention of cardiac arrhythmias was a plausible mechanistic explanation for a prompt reduction in sudden death with increased PUFA intakes. Note that there was no increase in intakes of fish, canola oil or olive oil in Australia as CHD mortality declined from its peak in 1966.

The FAO–WHO Consultation on Fats and Oils recommends, ‘Desirable intakes of linoleic acid (ie w-6 PUFA) should provide between 4 and 10 percent of energy. Intakes in the upper end of this range are recommended when intakes of saturated fatty acids and cholesterol are relatively high’.⁸⁵ The National Heart Foundation of Australia⁵⁵ concludes that there is good evidence that replacing saturated fatty acids with n-6 polyunsaturated fatty acids reduces the risk of coronary events and deaths. It recommends that n-6 polyunsaturated fatty acids contribute 8 to 10 per cent of total energy intake.⁵⁵

The case for n-3 polyunsaturated fatty acids

Unlike n-6 PUFA, where only one fatty acid (linoleic) is usually considered, there are three important fatty acids in the n-3 series:

- α -linolenic (ALA) (18:3)—occurring in leafy plants, canola oil and flaxseed oil
- eicosapentaenoic (EPA) (20:5)—the richest source being oily fish
- docosahexaenoic (DHA) (22:6)—the richest source being oily fish.

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

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

Alpha-linolenic acid, as the predominant fatty acid in flaxseed oil, lowers plasma total and LDL cholesterol, and large intakes of fish oil with EPA and DHA usually lower plasma total and LDL cholesterol.³⁴ In the diet, however, the three n-3 PUFAs are present in much smaller amounts than linoleic acid, and their cholesterol-lowering effect is of academic interest only.³⁴ In contrast, fish oil has a greater lowering effect on plasma triglycerides than does oil rich in n-6 PUFA.⁸⁶

In animal experiments fish oil has been found to have a more potent effect in preventing ischaemic arrhythmias than plant oils rich in n-6 PUFA.^{87,88} Canola oil also has an antiarrhythmic action⁸⁹, presumably because of its α -linolenic acid content: mono-unsaturated olive oil does not have this effect.

Fish consumption has been related to CHD outcome in 11 prospective cohort studies systematically reviewed by Marckmann and Grønbaek.⁹⁰ Fish were found to be protective in five of the studies. Some of the studies with negative results were of high scientific quality. It appears that a protective effect of fish, and presumably of fish oil (that is, EPA and DHA), is seen only in populations at high risk of CHD. In two prospective studies, intake of α -linolenic acid appeared to be protective.^{45,91}

Two randomised controlled secondary prevention trials have been reported with fish or fish oil. In the Diet and Reinfarction Trial (DART)⁷², one of three treatments given for two years to people who had survived a myocardial infarction was to eat 200–400 grams of oily fish a week or take fish oil capsules. Compared with the controls, coronary mortality was significantly reduced (by 24 per cent) in the fish group. Plasma cholesterol levels were not different, and in the fish group there were more non-fatal infarcts. In a multi-centre trial in Italy, the GISSI-Prevenzione Investigators⁹² gave 1 gram of EPA+DHA or vitamin E, or both or neither, to 11 324 survivors of myocardial infarction for three-and-a-half years. The fish oil groups had 10–15 per cent fewer deaths, non-fatal re-infarcts and strokes. The greatest benefit of the fish oil was a 45 per cent reduction in sudden deaths. The authors saw no evidence of a reduction in thrombotic events.

The third trial relevant to n-3 PUFAs is the Lyon Diet Heart Study⁹³, in which, among other dietary differences, α -linolenic acid was increased in the experimental group. Although *α -linolenic* appears in the title of the paper and plasma levels of it increased, it is difficult⁹⁴ to be sure whether this or other dietary (or even other) differences can explain the much lower mortality in the experimental group, which showed no reduction in plasma cholesterol.

The FAO-WHO Consultation on Fats and Oils⁸⁵ recommended that the ratio of linoleic to α -linolenic acid in the diet be between 5:1 and 10:1. Individuals with a ratio in excess of 10:1 should be encouraged to consume more n-3 rich foods, such as green leafy vegetables, legumes, fish and other seafood.

An expert workshop in the Netherlands⁹⁵ reviewed the health effects of n-3 PUFAs and concluded that consumption of fish may reduce the risk of CHD. People at risk were therefore advised to eat (preferably fatty) fish once a week. The workshop concluded that there should be separate recommendations for

plant (18:3) and marine (20:5, 22:6) n-3 PUFAs and that the n-3 to n-6 ratio would not be helpful.

The National Heart Foundation of Australia⁵⁵ recommends as follows:

- at least two fish meals per week, preferably oily fish. With the low stocks of fish currently available, this may not be feasible at a population level unless more farmed fish become available. It is possible that in the future EPA and DHA may be produced from microbiological culture
- consumption of both plant and marine n-3 PUFAs, since it is possible that they protect against CHD by different mechanisms
- plant n-3 PUFA intakes of at least 2 grams a day.

The case for mono-unsaturated fats

Oleic acid (18:1 cis) predominates among the mono-unsaturated fatty acids. There are, however, also small amounts in the diet of the fatty acids 16:1, 17:1, 20:1 and 22:1. When dietary fats are changed from mostly saturated to mostly mono-unsaturated, total and LDL cholesterol fall. But polyunsaturated fatty acids lower cholesterol more.^{31,33–38} Some oils rich in oleic acid have more effect on plasma cholesterol than others; this seems to depend on their content of phytosterols (which are cholesterol lowering), saturated fatty acids and squalene (cholesterol raising).⁹⁶

In the case of arrhythmias, intake of olive oil does not seem to prevent dangerous cardiac arrhythmias in experimental animals subjected to coronary artery ligation^{61,89}; with neonatal rat cardiac myocytes in culture, oleic acid (unlike PUFAs) shows no antiarrhythmic effect.⁹⁷

In prospective cohort studies mono-unsaturated fatty acid intake has not usually been associated with significant increases or decreases in coronary events. In the Seven Countries Study, ‘monoenes’ were negatively correlated but not significantly so.⁹⁸ In the younger cohort in Framingham, MUFA intake was significantly positively associated with CHD incidence.⁹⁹ In the US Nurses Health Study, a negative association of MUFA with CHD was marginally significant after several adjustments.⁴⁹

There has been no preventive trial with mono-unsaturated fats, and the FAO–WHO Consultation made no specific recommendations about mono-unsaturated fat intake.⁸⁵

The National Heart Foundation of Australia⁵⁵ also notes there is little evidence that mono-unsaturated fatty acids have an independent effect on coronary outcomes. Its position statement recommends ‘A proportion of dietary saturated fatty acids should be replaced by monounsaturated fatty acids as a strategy for reducing the intake of saturated fatty acids’.

Dietary cholesterol

The cholesterol-elevating effect of dietary cholesterol is less consistent than that of saturated fats.³⁴ Dietary cholesterol occurs only in animal fats, which are also the major source of saturated fatty acids in the diet.⁶ The two most concentrated sources of cholesterol in the diet, are eggs and animal brains. Eggs are rich in several other nutrients. Animal brains have more limited nutritional value and may carry a risk of bovine spongiform encephalopathy. To date, the NHMRC's position on dietary cholesterol has been that, at the public health level, advice to reduce saturated fat will bring with it lower cholesterol intakes. This position is supported by the Harvard prospective cohort study, which found that consumption of up to one egg a day was not associated with any increase in the rate of CHD.¹⁰⁰

The National Heart Foundation of Australia⁵⁵ recommends that individuals with plasma cholesterol greater than 5mmol/L or with other risk factors restrict their intake of cholesterol-rich foods. Over half the adult Australian population has plasma cholesterol levels above this.

Dietary fats and cancer

The second edition of the *Dietary Guidelines for Australians*⁴ stated, 'Epidemiological evidence suggests that total fat intake is associated with cancer of the breast and of the large intestine'. More evidence has now accumulated on this question. From nine prospective cohort studies on diet and breast cancer reviewed by the British Committee on Medical Aspects of Food and Nutrition Policy¹⁰¹, there is moderately consistent evidence that no association exists between higher total and saturated fat intakes and the risk of breast cancer.¹⁰¹ The World Cancer Research Fund agrees.¹⁰²

In experiments with rats given chemical carcinogens, there has been more mammary tumour development with diets containing moderate amounts of n-6 PUFA than with saturated fat such as lard. The FAO-WHO Consultation⁸⁵ found that tumour yields increase with linoleic acid added up to a threshold of 4 to 5 per cent of energy. When this threshold is reached, increasing total fat causes a further increase in tumours, but this is independent of the type of fat. In the US Nurses Health Study there was no correlation of breast cancer with total fat or with PUFA.¹⁰³ Colorectal cancer in this cohort was associated with total and saturated fat, not with PUFA intake.¹⁰⁴

Zock and Katan systematically reviewed the case-control and prospective cohort studies reporting linoleic acid, or n-6 PUFA intake, and cancer incidence.¹⁰⁵ For the case-control studies, the combined relative risks for high versus low intakes of linoleic acid were 0.84 for breast cancer and 0.92 for colorectal cancer. For the prospective studies, the combined relative risks were 1.05 for breast (not significant), 0.92 for colon and 0.83 for prostate cancer. In Europe, adipose tissue fatty acids were analysed in breast cancer cases and controls in five cities (in five countries). With the exception of Spain, the linoleic acid percentages were

practically identical.¹⁰⁶ Apart from the cohort reported by Willett et al.¹⁰⁴, no association between total fat and colorectal cancer has been found in eight reported prospective studies.¹⁰¹

Thus, with much more epidemiological data, particularly from prospective studies, than was available for the 1992 dietary guidelines, it would seem now that there is little or no association between cancer in adults and total fat or type of fat. Cancer is not mentioned in the fats section of the *Dietary Guidelines for Americans 2000*.⁵⁷

Dietary fats and micro-nutrients

Margarines and dairy foods contain retinol and beta-carotene and are important sources of vitamin A activity, although reduced-fat dairy foods contain much less retinol, depending on the degree of fat reduction. The 1995 National Nutrition Survey⁶ found that milk and milk products provided one-quarter to nearly half the preformed vitamin A in different age groups and margarine provided over 10 per cent; they provide some beta-carotene as well. Carotenoids are better absorbed from meals that contain fat or oil.¹⁰⁷

Most foods do not contain vitamin D. The best sources are margarine, to which it has to be added by regulation, at a minimum of 5.5 micrograms per 100 grams, and oily fish such as herring, pilchards and sardines. In the UK food tables¹⁰⁸ margarines contain 7.9µg of vitamin D per 100 grams and sardines 7.5µg. The vitamin D of butter is only 0.76µg and of cheese 0.26µg. Margarine was supplying 50 to 60 per cent of vitamin D in Australia in the 1980s, but its consumption declined somewhat in the 1990s. In recent years, increased cases of vitamin D deficiency have been reported in Australia, heightening the need to further understand, and respond to, changes in vitamin D supply (from sun and/or diet).

Nearly all vitamin E comes from vegetable oils and products made from them. Sunflower oil (49 milligrams per 100 grams), cottonseed (43mg), safflower (41mg), palm (33mg), canola (22mg), corn (17mg), soya (16mg) and peanut oil (15mg) are good sources, and olive oil contains 5 milligrams per 100 grams.¹⁰⁸ Margarines made from these oils should contain the corresponding amounts of vitamin E, and food regulations in Australia permit extra vitamin E in margarines as an antioxidant additive.

The only inorganic nutrient of any consequence in fats and oils is sodium chloride (salt), which is traditionally added to butters and margarines. This salt is not inherent, nor is it needed for processing. Standard varieties of these yellow fat spreads can be classified as high-salt foods¹⁰⁹ so, in keeping with Section 1.7, reduced-salt and low-salt margarines are recommended.

PRACTICAL ASPECTS OF THIS GUIDELINE

A number of practices can be incorporated in everyday life to optimise the fat profile of the diet.

- Choose predominantly unsaturated vegetable oils—such as sunflower, canola, corn, soya, olive and flaxseed—rather than animal fats, palm or coconut oil, or hydrogenated vegetable oils (often of unspecified origin).
- As a spread for bread and for baking, choose (reduced-salt) unsaturated margarines rich in n-6 and n-3, made from canola, sunflower, safflower or olive oil, rather than butter or hard margarine.
- Try to include in your diet fish high n-3 polyunsaturated fats—for example, sardines, tuna, salmon and herring. They can be eaten as such (grilled, say), in mixed dishes such as pasta, or in sandwiches.
- Use low- or reduced-fat milks (1 or 2 per cent fat) instead of full-cream milk (4 per cent fat).
- Choose low-fat yoghurts instead of full-cream yoghurt. Fortified soy milks and yoghurts contain little saturated fat.
- Limit your consumption of hard (full-fat) cheeses: they contain about 75 per cent energy as fat, mostly saturated fat. Look for reduced-fat hard cheeses and especially cottage cheeses.
- Use cream only as an occasional luxury. Choose reduced-fat forms.
- Buy lean cuts of meat and trim away the obvious fat before eating. Discard the skin of cooked chicken.
- Limit consumption of sausages, fatty mince, processed meats and ‘luncheon meats’ (that is, higher fat meat products).
- Discard fat drippings from cooked meat. Another way of reducing saturated fat is to replace some of the meat with plant-based protein-rich foods such as legumes and nuts.
- Limit consumption of fried savoury snack foods such as potato crisps. Choose those fried in sunola or cottonseed oil.
- Limit consumption of biscuits, which are high in saturated fat.
- Limit consumption of bought pastry products. When making pastry at home, use (poly)unsaturated margarine.
- Limit consumption of cakes. When making cakes at home, use (poly)unsaturated margarine.
- Eat only sparing amounts of chocolate and chocolate-containing confectioneries.
- Limit consumption of foods with creamy sauces and gravies. When preparing sauces at home, use (poly)unsaturated margarine.
- Choose (poly)unsaturated salad creams and dressings.
- Use a little canola, sunflower oil or olive oil for frying, not butter, dripping, lard or palm oil. Use non-stick pans and minimise frying fats.

- Moderate consumption of eggs; eat, at most, an average (whole or in dishes) of one a day.
- Read the nutrition information labels on packaged foods and avoid products high in saturated fat.

RELATIONSHIP TO OTHER GUIDELINES

Include lean meat, fish, poultry and/or alternatives

Lean meat is recommended.

Include milks, yoghurts, cheeses and/or alternatives. Reduced-fat varieties should be chosen where possible

Reduced-fat varieties are recommended.

Choose foods low in salt

Use low-salt varieties of fat spreads where possible.

Prevent weight gain: be physically active and eat according to your energy needs

Fat is energy dense and as such a high-fat diet can result in a high-energy diet, which may lead to obesity if physical activity is not maintained.

CONCLUSIONS

Total fat is providing about one-third of dietary energy in Australia. Consumption appears to have declined a little but is still relatively high from a world perspective. For anyone who is overweight, a reduction in total fat intake to 20–25 per cent of energy should be part of dietary management, as a contribution to reducing the energy density of food and drink, together with more physical exercise. This is a public health concern: the 1995 National Nutrition Survey found that about half the Australian adult population was overweight. For adult Australians who are not overweight, a moderate total fat intake is around 30 per cent of energy and, while watching the fat intake, it is important for heart health not to reduce the major sources of n-6 and n-3 polyunsaturated fatty acids.

The biological effects and health risks of dietary fats and oils are determined in large part by their predominant fatty acids. Saturated fatty acids raise plasma LDL cholesterol, a major risk factor for coronary heart disease. In three large prospective epidemiological studies, saturated fatty acid intake was directly associated with subsequent CHD. Trans-fatty acids appear to behave similarly,

although they are now little consumed in Australia. Saturated plus trans-fatty acid intakes averaged over 12.5 per cent of energy in Australia in 1995. A population average of 10 per cent of energy is recommended as a realistic target.

Intake of n-6 polyunsaturated fatty acids (essentially linoleic) should be in the range of 6 to 8 per cent of energy because there is strong evidence that n-6 PUFAs protect against CHD by lowering plasma LDL cholesterol and probably by reducing the risk of dangerous cardiac arrhythmias. The 1995 National Nutrition Survey found that the intake of PUFAs (mainly n-6) was less than 5 per cent of energy. This should be substantially increased. The current intake of n-3 polyunsaturated fatty acids, which occur in fish and a few vegetable oils, is low (about 200 milligrams a day). It would appear desirable to double this intake as a measure designed to reduce the risk of CHD, but such a recommendation would pose challenges for both the environment and the fats and oils industry. Work on producing from single-cell culture the biologically active n-3 polyunsaturated fatty acids EPA and DHA found in fish should be a key area of future research and development.

Mono-unsaturated fatty acids do not raise plasma cholesterol and do not have the action of polyunsaturated fatty acids on arrhythmias. With the exception of individuals who need to reduce total fat as part of body-weight management, current intake levels appear to be satisfactory.

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

EVIDENCE

Level II evidence exists for the effect of dietary fat on overweight (references 18 and 19), and there is Level III evidence from metabolic trials of the effect of various dietary fats on plasma cholesterol (references 31 to 42, 58, 59 and 96).

There is Level II and Level III evidence for the effect of dietary fat type on coronary heart disease from controlled intervention trials (eight out of the 10 being randomised) (references 66 to 73, 92 and 93) and Level III evidence from prospective studies (references 44 to 49, 63 to 65, 90, 91 and 98 to 100) and biomarker case-control studies (references 50 and 51).

Level III evidence for the effect of dietary fat on cancer is available from prospective studies (references 101 to 105) and biomarker case-control studies (references 105 and 106).

REFERENCES

1. Truswell AS. *Dietary fat: some aspects of nutrition and health and product development*. Brussels: ILSI Europe, 1995.
2. Department of Health. *Dietary guidelines for Australians*. Canberra: Australian Government Publishing Service, 1982.

3. US Senate Select Committee on Nutrition and Human Needs. *Dietary goals for the United States*. Washington, DC: US Government Publishing Service, 1977.
4. National Health and Medical Research Council. *Dietary guidelines for Australians*. Canberra: Australian Government Publishing Service, 1992 (reprinted 1998).
5. National Health and Medical Research Council. *Dietary guidelines for older Australians*. Canberra: NHMRC, 1999.
6. McLennan W, Podger A. *National Nutrition Survey: nutrient intakes and physical measurements, Australia, 1995*. Canberra: Australian Bureau of Statistics, 1998.
7. Department of Community Services and Health. *National Dietary Survey of Adults, 1983. No. 2, nutrient intakes*. Canberra: Australian Government Publishing Service, 1987.
8. National Heart Foundation of Australia. *Risk Factor Prevalence Study: report no. 1*. Canberra: NHF, 1980.
9. National Heart Foundation of Australia. *Risk Factor Prevalence Study: report no. 2*. Canberra: NHF, 1983.
10. National Heart Foundation of Australia. *Risk Factor Prevalence Study: survey no. 3*. Canberra: NHF, 1989.
11. World Health Organization. *Obesity: preventing and managing the global epidemic. Technical Report Series 894*. Geneva: WHO, 2000.
12. Schutz Y, Flatt JP, Jecquier E. Failure of dietary fat intake to promote fat oxidation: a factor favouring the development of obesity. *Am Clin Nutr* 1989;50:307–14.
13. Blundell JE, Macdiarmid JI. Fat as a risk factor for overnutrition: satiation, satiety and patterns of eating. *J Am Diet Assoc* 1997;97(suppl.):S63–S69.
14. Westerterp KR, Wilson SAJ, Rolland V. Diet induced thermogenesis measured over 24 h in a respiration chamber: effect of diet composition. *Int J Obesity* 1999;23:287–92.
15. Lissner L, Heitmann BL. Dietary fat and obesity: evidence from epidemiology. *Eur J Clin Nutr* 1995;49:79–90.
16. Goris AHC, Westerterp-Plantenga MS, Westerterp KR. Undereating and underrecording of habitual food intake in obese men: selective underreporting of fat intake. *Am J Clin Nutr* 2000;71:130–4.
17. Cooling J, Blundell J. Are high fat and low fat consumers distinct phenotypes? Differences in the subjective and behavioural response to energy and nutrient challenges. *Eur J Clin Nutr* 1998;52:193–201.
18. Toubro S, Astrup A. Randomised comparison of diets for maintaining obese subjects' weight after major weight loss: ad lib, low fat, high carbohydrate diet v fixed energy intake. *BMJ* 1997;314:29–34.

19. Westerterp-Plantenga MS, Wijckmans-Duijsens NEG, Verboekt-van de Venne WPG, de Graff K, van het Hof KH, Weststrate JA. Energy intake and body weight effects of six months reduced or full fat diets, as a function of dietary restraint. *Int J Obesity* 1998;22:14–22.
20. Klem ML, Wing RR, McGuire MT, Seagle HM, Hill JO. A descriptive study of individuals successful at long-term maintenance of substantial weight loss. *Am J Clin Nutr* 1997;66:239–46.
21. British Obesity Task Force. *The health of the nation: obesity. Reversing the increasing problem of obesity in England. Request from the Nutrition and Physical Activity Task Forces*. London: Department of Health, 1995.
22. Scottish Intercollegiate Guidelines Network. *Obesity in Scotland: integrating prevention with weight management. National clinical guideline*. Edinburgh: SIGN Secretariat, 1991.
23. Bray GA, Popkin BM. Dietary fat does effect obesity! *Am J Clin Nutr* 1998;68:1157–73.
24. Truswell AS. Energy balance, food and exercise. *World Rev Nutr Diet* 2001;90:13–25.
25. Dunstan D, Zimmet P, Welborn T, Sicree R, Armstrong T, Atkins R et al. *Diabetes and associated disorders in Australia—2000: the accelerating epidemic. Final report of the Australian Diabetes, Obesity and Lifestyle Study (AusDiab)*. International Diabetes Institute: Melbourne, 2001.
26. Marshall JA, Hoag S, Shetterly S, Hamman RF. Dietary fat predicts conversion from impaired glucose tolerance to NIDDM. *Diab Care* 1994;17:50–6.
27. Vessby B, Aro A, Skarfors E, Berglund L, Salminen I & Lithell H. The risk to develop NIDDM is related to the fatty acid composition of serum cholesterol esters. *Diabetes* 1994;43:1353–7.
28. Salmeron J, Hu FB, Manson JAE, Stampfer MJ, Colditz GA, Rimm EB et al. Dietary fat intake and risk of type 2 diabetes in women. *Am J Clin Nutr* 2001;73:1019–26.
29. Powers AC. Diabetes mellitus. In: Braunwald E, Fauci AS, Kasper DL, Hansen SL, Longo DL, Jameson JL, eds. *Harrison's principles of internal medicine*, 15th edn. New York: McGraw Hill, 2001:2128.
30. 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;335:1001–9.
31. Keys A, Anderson JT, Grande F. Prediction of serum-cholesterol responses of man to changes of fats in the diet. *Lancet* 1957;ii:959–66.
32. Bronte-Stewart B. The effect of dietary fats on the blood lipids and their relation to ischaemic heart disease. *Brit Med Bull* 1958;14:243–52.

33. Hegsted DM, McGandy RB, Myers ML, Stare FH. Quantitative effects of dietary fat on serum cholesterol in man. *Am J Clin Nutr* 1965;17:281–95.
34. Beynen AC, Katan MB. Impact of dietary cholesterol and fatty acids on serum lipids and lipoproteins in man. In: Vergroesen AJ, Crawford M, eds. *The role of fats in human nutrition*. 2nd edn. London: Academic Press, 1989:237–86.
35. Mensink RP, Katan MB. Effect of dietary fatty acids on serum lipids and lipoproteins: a meta-analysis of 27 trials. *Arteriosclerosis & Thrombosis* 1992;12:911–19.
36. Hegsted DM, Ausman LM, Johnson JA, Dallal GE. Dietary fat and serum lipids: an evaluation of the experimental data. *Am J Clin Nutr* 1993;57:875–83.
37. Clarke R, Frost C, Collins R, Appleby P, Peto R. Dietary lipids and blood cholesterol: qualitative meta-analysis of metabolic ward studies. *BMJ* 1997;314:112–17.
38. McGandy RB, Hegsted DM, Myers ML. Use of semisynthetic fats in determining effects of specific dietary fatty acids on serum lipids in man. *Am J Clin Nutr* 1970;23:1288–98.
39. Anderson JT, Grande F, Keys A. Hydrogenated fats in the diet and lipids in the serum of man. *J Nutr* 1961;75:388–94.
40. Mensink RPM, Katan MB. Effect of dietary trans fatty acids on high-density and low-density lipoprotein cholesterol levels in healthy subjects. *N Engl J Med* 1990;323:439–45.
41. Nestel P, Noakes M, Belling B, McArthur R, Clifton P, Janus E et al. Plasma lipoprotein lipid and Lp(a) changes with substitution of elaidic acid for oleic acid in the diet. *J Lipid Res* 1992;33:1029–36.
42. Judd JT, Baer DJ, Muesing R, Wittes J, Sunkin ME, Podczasy JJ. Dietary trans fatty acids: effects on plasma lipids and lipoproteins of healthy men and women. *Am J Clin Nutr* 1994;59:861–8.
43. Keys A, Menotti A, Karvonen MJ, Aravanis C, Blackburn H, Buzina R et al. The diet and 15-year death rate in the Seven Countries Study. *Am J Epidemiol* 1986;124:903–15.
44. Hu FB, Stampfer MJ, Manson JAE, Rimm E, Colditz GA, Rosner BA et al. Dietary saturated fats and their food sources in relation to the risk of coronary heart disease in women. *Am J Clin Nutr* 1999;70:1001–8.
45. Ascherio A, Rimm EB, Giovannucci EL, Spiegelman D, Stampfer M, Willett WC. Dietary fat and risk of coronary heart disease in men: cohort follow up study in the United States. *BMJ* 1996;313:84–90.
46. Miettinen TA, Naukkarinen V, Huttunen JK, Mattila S, Kumlin T. Fatty-acid composition of serum lipids predicts myocardial infarction. *BMJ* 1982;285:993–6.

47. Willett WC, Stampfer MJ, Manson JAE, Colditz GA, Speizer FE, Rosner BA et al. Intake of trans fatty acids and risk of coronary heart disease among women. *Lancet* 1993;341:581–5.
48. Applewhite TH. Trans-isomers, serum lipids and cardiovascular disease: another point of view. *Nutr Rev* 1993;51:344–5.
49. Hu FB, Stampfer MJ, Manson JAE, Rimm E, Colditz GA, Rosner BA et al. Dietary fat intake and the risk of coronary heart disease in women. *N Engl J Med* 1997;337:1491–9.
50. Roberts TL, Wood DA, Riemersma RA, Gallagher PJ, Lampe FC. Trans isomers of oleic and linoleic acids in adipose tissue and sudden cardiac death. *Lancet* 1995;345:278–82.
51. Aro A, Kardinaal AFM, Salminen I, Kark, JD, Riemersma RA, Delgado-Rodriguez M et al. Adipose tissue isomeric trans fatty acids and risk of myocardial infarction in nine countries: the EURAMIC study. *Lancet* 1995;345:273–8.
52. Department of Health and Social Security Committee on Medical Aspects of Food Policy. *Diet and cardiovascular disease: report of the panel on diet in relation to cardiovascular disease*. DHSS report on health and social subjects no. 28. London: HMSO, 1984.
53. National Health and Medical Research Council. *The role of polyunsaturated fats in the Australian diet*. Working party report. Canberra: Australian Government Publishing Service, 1992.
54. Shrapnel WS, Truswell AS, Nestel PJ, Simons LA. Dietary fatty acids and blood cholesterol. Canberra: National Heart Foundation of Australia, 1994.
55. National Heart Foundation of Australia. Position statement on dietary fats. *Aust J Nutr Diet* 1999;56(4)(suppl.):S3–S4.
56. Noakes M, Nestel P. Trans fatty acids in the Australian diet. *Food Aust* 1994;46:124–9.
57. Department of Agriculture & US Department of Health and Human Services. *Dietary guidelines for Americans 2000*. 5th edn. Washington, DC: Department of Agriculture, 2001.
58. Bronte-Stewart B, Antonis A, Eales L, Brock JF. Effects of feeding different fats on serum cholesterol levels. *Lancet* 1956;i:521–7.
59. Rassias G, Kestin M, Nestel PJ. Linoleic acid lowers LDL-cholesterol without proportionate displacement of saturated fatty acid. *Eur J Clin Nutr* 1991;45:315–20.
60. McLennan P, Abeywardena MY, Charnock JS. Influence of dietary lipids on arrhythmias and infarction after coronary artery ligation in rats. *Can J Physiol Pharmacol* 1985;63:1411–17.

61. McLennan PL. Relative effects of dietary saturated, monounsaturated and polyunsaturated fatty acids on cardiac arrhythmias in rats. *Am J Clin Nutr* 1993;57:207–13.
62. McLennan PL, Bridle TM, Abeywardena MY, Charnock JS. Dietary lipid modulation of ventricular fibrillation threshold in the marmoset monkey. *Am Heart J* 1992;123:1555–61.
63. Kingsbury KJ, Morgan DM, Stocold R, Brett CG, Andrews J. Polyunsaturated fatty acids and myocardial infarction: follow up of patients with aortoiliac and femoropopliteal atherosclerosis. *Lancet* 1969;ii:1325–9.
64. Simon JA, Hodgkins ML, Browner WS, Neuhaus JM, Bernert JT Jr, Hulley SB. Serum fatty acids and the risk of coronary heart disease. *Am J Epidemiol* 1995;142:469–76.
65. Shekelle RB, Shryock AM, Paul O, Lepper M, Stamler J, Liu S et al. Diet, serum cholesterol and death from coronary heart disease: the Western Electric Study. *N Engl J Med* 1981;304:65–70.
66. Rose FA, Thomson WE, Williams RT. Corn oil in treatment of ischaemic heart disease. *BMJ* 1965;i:1531–3.
67. Research Committee to the Medical Research Council. Controlled trial of soya-bean oil in myocardial infarction: report. *Lancet* 1968;ii:693–700.
68. Dayton S, Pearce ML, Hashimoto S, Dixon WJ, Taniyasu U. A controlled clinical trial of a diet high in unsaturated fat in preventing complications of atherosclerosis. *Circulation* 1969; suppl. II to vols 39, 40.
69. Leren P. The effect of plasma cholesterol lowering diet in male survivors of myocardial infarction. *Acta Med Scand* 1996;suppl.:466. Leren P. The Oslo diet heart study: eleven year report. *Circulation* 1970;42:935–42.
70. Turpeinen O, Karvonen MJ, Pekkarinen M, Miettinen M, Eluosi R, Paavilainen E. Dietary prevention of coronary heart disease: the Finnish Mental Hospitals Study. *Int J Epidemiol* 1979;8:99–117.
71. Miettinen M, Turpeinen O, Karvonen MJ, Pekkarinen M, Paavilainen E, Eluosi R. Dietary prevention of coronary heart disease in women: the Finnish Mental Hospitals Study. *Int J Epidemiol* 1983;12:17–25.
72. Burr ML, Gilbert JF, Holliday RM, Elwood DC, Fehily AM, Rogers S et al. Effects of changes in fat, fish and fibre intakes on death and myocardial reinfarction: Diet & Reinfarction Trial (DART). *Lancet* 1989;ii:757–61.
73. Frantz ID Jr, Dawson EA, Ashman PL, Gatewood LC, Bartsch GE, Kuba K et al. Test of effect of lipid lowering by diet on cardiovascular risk: the Minnesota Coronary Survey. *Atherosclerosis* 1989;9:129–35.
74. Truswell AS. Dietary fat type and coronary heart disease. In: *Nutrition in the nineties: are we in agreement?* Proceedings of Sydney University Nutrition Research Foundation Symposium, 5 Nov 1997. Nutrition Research Foundation publication no. 5. Sydney University, 1997.

75. Thompson PL, Hobbs MST, Martin CA. The rise and fall of ischaemic heart disease in Australia. *Aust NZ J Med* 1988;18:327–37.
76. Pyörälä K, Epstein FH, Kornitzer M. Changing trends in coronary heart disease mortality: possible explanations. *Cardiology* 1985;72:5–10.
77. Katan MB, Beynen AC. Linoleic acid consumption and coronary heart disease in USA and UK. *Lancet* 1981;ii:371.
78. Truswell AS. Recent trends in mortality from coronary heart disease (CHD) in different countries: possible relation to diet. In: Yasumoto K, Itokawa Y, Koishi H, Samo Y, eds. *Proceedings of the Fifth Asian Congress of Nutrition (Osaka, Japan, October 26–29, 1987)*. Tokyo: Center for Academic Publications, 1987.
79. Hetzel BS, Charnock JS, Dwyer T, McLennan PL. Fall in coronary heart disease mortality in USA and Australia due to sudden death: evidence for the role of polyunsaturated fat. *J Clin Epidemiol* 1989;42:885–93.
80. Rose G. Causes of the trends and variations in CHD mortality in different countries. *Int J Epidemiol* 1989;18:S174–S179.
81. Lloyd BL. Declining cardiovascular disease incidence and environmental components. *Aust NZ Med J* 1994;24:124–32.
82. Whyte HM. *The fats of life: the theory and practice of eating and cooking to avoid coronary heart disease*. Sydney: Ure Smith, 1961.
83. Standing Sub-committee Appointed by the National Heart Foundation of Australia to Maintain a Continuing Review of Research Developments in the Field of Diet as Related to Heart Disease. Dietary fat and coronary heart disease: a review. *Med J Aust* 1967;i:309–22.
84. National Center for Health Statistics – National Heart, Lung and Blood Institute Collaborative Lipid Group. Trends in serum cholesterol levels among US adults aged 20–74 years: data from the National Health & Nutrition Examination Surveys 1960 to 1980. *JAMA* 1987;257:937–42.
85. FAO–WHO Consultation on Fats and Oils. *Fats and oils in human nutrition*, FAO nutrition paper no. 57. Rome: Food and Agriculture Organization, 1994.
86. Harris WS, Connor WE, McMurray MP. The comparative reductions of the plasma lipids and lipoproteins by dietary polyunsaturated fats: salmon oil versus vegetable oils. *Metabolism* 1983;32:179–84.
87. McLennan PL, Bridle TM, Abeywardena MY, Charnock JS. Comparative efficacy of n-3 and n-6 polyunsaturated fatty acids in modulating ventricular fibrillation threshold in marmoset monkeys. *Am J Clin Nutr* 1993;58:666–9.
88. McLennan PL, Abeywardena MY, Charnock JS. Reversal of the arrhythmogenic effects of long-term saturated fatty acid intake by dietary n-3 and n-6 polyunsaturated fatty acids. *Am J Clin Nutr* 1990;51:53–8.

89. McLennan PL, Dallimore JA. Dietary canola oil modifies myocardial fatty acids and inhibits cardiac arrhythmias in rats. *J Nutr* 1995;125:1003–9.
90. Marckmann P, Grønbaek M. Fish consumption and coronary heart disease mortality: a systematic review of prospective cohort studies. *Eur J Clin Nutr* 1999;53:585–90.
91. Dolecek TA. Epidemiological evidence of relationships between dietary polyunsaturated fatty acids and mortality in the multiple risk factor intervention trial. *Proc Soc Exp Biol Med* 1992;200:177–82.
92. GISSI-Prevenzione Investigators. Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction: results of the GISSI-Prevenzione trial. *Lancet* 1999;354:447–55.
93. de Lorgeril M, Renaud S, Mamelle N, Salen P, Martin J-L, Monjaud I et al. Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease. *Lancet* 1994;343:1454–9.
94. Hartman IS. Alpha-linolenic acid: a preventive in secondary coronary events? *Nutr Rev* 1995;53:194–201.
95. de Deckere EAM, Korver O, Verschuren PM, Katan MB. Health aspects of fish and n-3 polyunsaturated fatty acids from plant and marine origin. *Eur J Clin Nutr* 1998;52:749–53.
96. Truswell AS, Choudhury N. Monounsaturated oils do not all have the same effect on plasma cholesterol. *Eur J Clin Nutr* 1998;52:1–4.
97. Kang JX, Leaf A. Antiarrhythmic effects of polyunsaturated fatty acids: recent studies. *Circulation* 1996;94:1774–80.
98. Keys A ed. Coronary heart disease in seven countries. *Circulation* 1970;41,42;suppl. 1:1-171–1-179.
99. Posner BM, Cobb JL, Belanger AJ, Cupples A, D’Agostino RB, Stokes J. Dietary lipid predictors of coronary heart disease in man: the Framingham Study. *Arch Int Med* 1991;151:1181–7.
100. Hu FB, Stampfer MJ, Rimm EB, Manson JE, Ascherio A, Colditz GA et al. A prospective study of egg consumption and risk of cardiovascular disease in men and women. *JAMA* 1999;281:1387–94.
101. Committee on Medical Aspects of Food and Nutrition Policy. *Nutritional aspects of the development of cancer*. Report of the Working Group on Diet and Cancer. London: HMSO, 1998.
102. World Cancer Research Fund. *Food, nutrition and the prevention of cancer: a global perspective*. Washington, DC: American Institute for Cancer Research, 1997.
103. Holmes MD, Hunter DJ, Colditz GA, Stampfer MJ, Hankison SE, Speizer FE et al. Association of dietary intake of fat and fatty acids with risk of breast cancer. *JAMA* 1999;281:914–20.

104. Willett WC, Stampfer MJ, Colditz GA, Rosner BA, Speizer FE. Relation of meat, fat and fiber to the risk of colon cancer in a prospective study among women. *N Engl J Med* 1990;323:1664–72.
105. Zock PL, Katan MB. Linoleic acid intake and cancer risk: a review and meta-analysis. *Am J Clin Nutr* 1998;68:142–53.
106. Simonsen N, van't Veer P, Strain JJ, Martin-Moreno JM, Huttunen JK, Navajas JF-C et al. Adipose tissue omega-3 and omega-6 fatty acid content and breast cancer in the EURAMIC study. *Am J Epidemiol* 1998;147:342–52.
107. Institute of Medicine. *Dietary reference intakes for vitamin C, vitamin E, selenium and carotenoids*. Washington, DC: National Academy Press, 2000.
108. Holland B, Welch AA, Unwin ID, Buss DH, Paul AA, Southgate DAT. *McCance & Widdowson's the composition of foods*, 5th edn. Cambridge: Royal Society of Chemistry, 1991.
109. National Health and Medical Research Council. *Report of the Working Party on Sodium in the Australian Diet*. Canberra: Australian Government Publishing Service, 1984.

I.7 CHOOSE FOODS LOW IN SALT

Malcolm Riley and Trevor Beard

TERMINOLOGY

Salt and sodium

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.¹ About 90 per cent of all the sodium added to food is sodium chloride, so dietary intake of sodium represents 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.

Recommended intake for sodium

The current National Health and Medical Research Council recommendation for the Australian adult population in general is that dietary sodium intake be under 2300 milligrams of sodium (100mmol) per day.² This is the equivalent of about 6 grams of common salt—about one-and-a-half teaspoons.

Low-salt food

Food Standards Australia New Zealand defines a low-salt food as a food with a sodium concentration of up to 120 milligrams per 100 grams (Joint Australia and New Zealand Food Code, clause 17 of Standard 1.2.8).

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

1mmol = 23 milligrams

1 gram = 43mmol

One gram of sodium chloride (NaCl) contains 17mmol, or 391 milligrams, of sodium.

BACKGROUND

It is now well accepted that a reduction in dietary sodium intake will decrease the mean population blood pressure and reduce the prevalence of hypertension. It has not yet been conclusively established that a mean dietary sodium intake within the range recommended for Australian adults will result in lower morbidity and mortality rates than at present³, but the balance of evidence suggests it will. It has been said that 'few measures in preventive medicine are as simple and economical and yet can achieve so much'.⁴ There is strong evidence that other components of the diet also influence blood pressure; this guideline focuses, however, on the independent influence of sodium.

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

Cardiovascular disease is the largest cause of premature death and death overall in Australia; it accounted for 50 797 deaths, or 40 per cent of all deaths, in 1998.⁷ Most of these deaths are caused by coronary heart disease (55 per cent), followed by stroke (24 per cent). Each year about 40 000 Australians have a stroke; 70 per cent of these are first-ever strokes. Stroke is the cause of nearly 25 per cent of all chronic disability in Australia.⁸ Hypertension is the problem most frequently seen by general practitioners, accounting for 5.7 per cent of all problems they see.⁹ In 1995 an estimated 2.8 million Australians aged 18 years and over reported a recent and/or long-term cardiovascular condition. High blood pressure was the most common condition for both males and females.¹⁰ The prevalence of cardiovascular conditions increased with age, reaching 61 per cent among people aged 75 years and over.

Although male and female death rates from cardiovascular disease in Australia declined by about 3.6 per cent a year between 1985 and 1996, the number of people with the disease is expected to increase in the next few decades as the number of older people increases and life-sustaining treatments improve. The total direct costs to Australia of cardiovascular disease were estimated to be \$3719 million in 1993–94, which represents 12 per cent of total health care costs for all diseases.¹¹

The financial implications of the salt guideline are thus considerable in Australia, where the National Heart Foundation has been advising doctors for a number of years that non-pharmacological measures should be the first line of treatment for patients with hypertension and may abolish or reduce the need for medication.¹² Most antihypertensive drugs are more effective at a low salt intake, especially some of the more expensive drugs such as ACE inhibitors and angiotensin antagonists. Prescriptions for a single ACE inhibitor (enalapril) cost the Australian government about \$60 million in 1998¹³, an amount that could be substantially reduced if patients followed the salt guideline.

Diuretics used at the low dosages currently recommended have no effect on blood pressure if sodium excretion rates are above 190mmol a day¹⁴ and are of no practical value at or below 70mmol a day.¹⁵ They are contra-indicated at rates below 50mmol a day¹⁶ because of the danger of iatrogenic hyponatraemia and lack of benefit.

A recent review¹⁷ has pointed to a number of health conditions other than raised blood pressure that are associated with excess sodium intake. Among them are any condition exacerbated by water retention (including heart failure, cirrhosis, nephrotic syndrome, and idiopathic and cyclical oedema), stroke (independently of blood pressure), gastric cancer and left ventricular hypertrophy. Excess sodium intake also increases the rate of deterioration in kidney function in patients with renal disease, is associated with urinary stones, and may aggravate asthma¹⁸ and osteoporosis. Excretion of sodium is associated with an obligatory loss of calcium as a result of interference with the tubular reabsorption of calcium. Calcium is conserved on low salt intakes and wasted on high salt intakes. This has relevance to calcium stones of the urinary tract (the commonest variety of stone) and osteoporosis (see also Section 1.4).

Current intakes

Sodium intake is poorly measured by many dietary survey methods that are used to measure other food components because foods of similar type vary widely in their sodium content (for example, breakfast cereals) and dietary addition can be discretionary but not easily quantifiable (for example, adding salt when cooking and at meals). Measuring sodium output in urine over a 24-hour period is an accurate way of estimating sodium intake: about 93 per cent of dietary sodium is recovered in the urine.⁵¹ Day-to-day variation in sodium intake is high^{30,19}, so intake for a single day generally does not accurately reflect a person's usual long-term intake.

Two small Australian surveys based on systematic samples from the Commonwealth electoral roll have produced similar findings.^{20,21} The larger and more recent of the two²⁰ found that men (n=87) had a mean urinary sodium excretion rate of 170mmol/day (SD±52) and women (n=107) had a rate of 118mmol/day (SD±42). The range for men for a single day was 39–337mmol and for women 26–241mmol. In a Sydney survey of adults mostly from a university community, the figures were very similar, except for subjects with an

Asian diet, where the men (n=29) averaged 195mmol/day of urinary sodium and the women (n=21) averaged 140mmol/day.²²

The results from ordinary Australian diets are similar to those of respondents selected from the UK electoral roll a decade earlier²³, where the mean urinary sodium excretion rate for men (n=681) was 171mmol/day and for women (n=712) 132mmol/day, with a range for men of 20–498mmol/day and for women 21–354mmol/day.

SCIENTIFIC BASIS

The scientific literature relating dietary sodium intake to blood pressure is extensive and dates back more than 100 years.²⁴ In this section it is possible only to summarise the state of current knowledge, highlight important studies, and draw attention to important considerations. The literature provides evidence that different commentators can interpret similar scientific results quite differently, but there is general consensus on most issues.

It is generally agreed that the extent to which a reduction in dietary sodium intake reduces blood pressure depends on age and initial blood pressure; it is greater with age and at higher blood pressures.^{25,26} Law et al.²⁶ found that the full effect of dietary sodium reduction on blood pressure is not seen for at least five weeks after the dietary change.

Among the major original studies are animal studies and human studies. Controlled experiments in chimpanzees have shown an important effect of dietary salt reduction on blood pressure²⁷, consistent with earlier experiments on rats.²⁸ For chimpanzees that were allocated a high-sodium diet for only two years, up to six months of a return to their normal low-sodium diet was required before blood pressure lowering had reached its greatest extent.

In relation to human populations, three recent reviews^{3,6,29} are in agreement that dietary sodium reduction is associated with reduction in blood pressure. Kuller²⁹ and Law⁶ have called for a public health approach to lowering salt in the diet—that is, for the average salt intake of the population to be lowered through reducing the amount of salt entering the food supply. Alderman³, however, calls for randomised controlled studies of the long-term health benefits and safety of dietary sodium reduction and concludes, ‘Without knowledge of the sum of the multiple effects of a reduced sodium diet, no single universal prescription for sodium intake can be scientifically justified.’

The mass addition of salt to the food supply is relatively recent in human history and relies on some technological sophistication. Salt’s initial use was as a preservative, but it gradually became a common addition, both in the commercial processing of foods and in the home, for its preserving and taste-enhancing properties. In more recent years, awareness of salt’s role in the diet has led to a reduction in salt in many processed foods and in its use in the home, but its

cheap price and its preservative and sensory properties have limited that reduction. Societies without the technology for mass addition of salt to the food supply still exist—and their members are all normotensive throughout life, with little, if any, increase in blood pressure with age.³⁰

Meta-analyses of sodium and hypertension trials

Law et al. took an approach that differs from a standard meta-analysis. They analysed cross-sectional data from 24 communities worldwide, involving more than 47 000 people²⁶, and derived relationships between dietary sodium intake and blood pressure that depended on age, centile in the blood pressure distribution, and the level of development of the community. They tested the relationships on 14 studies examining the association of blood pressure with sodium intake within populations—importantly, after adjusting for the large effect of regression dilution bias.³¹ They found that the within-population associations were consistent with the relationships estimated using between-population data. Finally, they examined how closely the results from 68 crossover trials and 10 randomised controlled trials conformed to the relationships they originally estimated from the between-population studies.⁴ They found that, for the 33 trials lasting five weeks or more, the observed reductions in blood pressure were similar to the predicted values (within a 95 per cent confidence interval for 30 of the trials). For trials lasting less than five weeks, they found that the predicted fall in blood pressure was less than the observed fall, which led them to conclude that dietary sodium reduction does not have its full effect on blood pressure until at least five weeks after intervention begins. The consistency of results from different study types and in different populations has increased the investigators' confidence in their estimates for the relationship between dietary sodium intake and blood pressure.⁴

Cutler et al.'s meta-analysis, published in 1997³², updates an earlier meta-analysis conducted by Cutler and others.³³ They included 32 trials in their analyses and concluded, 'The blood pressure reduction that would result from a substantial lowering of dietary sodium in the US population could reduce cardiovascular morbidity and mortality'. Midgley et al.³⁴ included a total of 56 trials in their analysis and concluded, 'Dietary sodium restriction might be considered, but the evidence in the normotensive population does not support current recommendations for universal dietary sodium restriction'. Graudal et al.²⁵ included 58 trials of hypertensive people and 56 trials of normotensive people in their analysis and concluded, 'These results do not support a general recommendation to reduce sodium intake ... but ideally trials with hard end points such as morbidity and survival should end the controversy'. It is important to note that the mean duration of the studies in the Midgley et al. meta-analysis was only two weeks, and in the Graudal et al. analysis it was seven days. Table 1.7.1 summarises the results of the meta-analyses.

Table 1.7.1 Decrease in systolic and diastolic blood pressure on reduction of dietary sodium: meta-analyses of randomised controlled trials^{25,32,34}

Meta-analysis	Diastolic blood pressure (mmHg)	Systolic blood pressure (mmHg)
Midgley et al. 1996		
Normotensive subjects (weighted average 125mmol difference in dietary sodium)	0.5	1.6
Hypertensive subjects (weighted average 95mmol difference in dietary sodium)	3.8	5.9
Cutler et al. 1997		
Normotensive subjects (weighted average 71mmol difference in dietary sodium)	0.8	1.5
Hypertensive subjects (weighted average 76mmol difference in dietary sodium)	2.1	3.8
Graudal et al. 1998		
Normotensive subjects (weighted average 160mmol difference in dietary sodium)	0.3	1.2
Hypertensive subjects (weighted average 118mmol difference in dietary sodium)	1.9	3.9

The question is how to interpret the different conclusions between meta-analyses. The first point to be made is that the estimated effects are not greatly different from one another. The overall effects are lower than what might be expected with full dietary compliance because the inclusion of all randomised subjects ('intention to treat' analysis) dilutes the number of subjects actually complying with the intervention treatment. This is the correct form of analysis for randomised controlled trials and incorporates both the effect of treatment and the extent of compliance. In determining the extent to which change in dietary sodium will change blood pressure, there are four effect-modifying factors: the magnitude of the change in sodium intake; the age of the subjects; the initial blood pressure of the subjects; and the duration of the intervention. Ideally, one would like to conduct meta-analyses at different levels of these factors, as has been done for normotension versus hypertension.

Many of the studies included in the meta-analyses were of short duration and conducted on young people. This would also tend to decrease the effect observed. The studies involving larger reductions in dietary sodium also tended to be the shorter ones because of the current difficulty in maintaining a free-living population at a low sodium intake for a long period. In addition, technical factors—such as how to weight individual trials and how to construct a summary regression line—differed between meta-analyses. The choice of methodology alters the summary estimates considerably.

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

No randomised controlled trials have been conducted to test the effectiveness of dietary sodium reduction for primary prevention of hypertension. Furthermore, there is little information from randomised controlled trials about the effect of dietary sodium reduction on mortality or morbidity from cardiovascular disease.³

Two large randomised controlled trials whose results were published after the meta-analyses—the DASH-Sodium trial³⁵ and the TONE study³⁶—warrant discussion.

DASH-Sodium was a sequel to the first DASH (Dietary Approaches to Stop Hypertension) study.³⁷ Both were multi-centre randomised controlled trials for a dietary period of 30 days. The first study held sodium constant at 130mmol/day and compared the standard American diet with an ‘ideal’ diet that emphasised fruits, vegetables, low-fat dairy foods, fish, legumes, nuts, and lean meat and poultry; DASH-Sodium repeated the experiment at three sodium levels—the US guideline of 104mmol/day plus or minus 39mmol/day (that is, 143, 104 and 65mmol/day). The first DASH study observed a highly significant fall in blood pressure with the ‘ideal’ diet, indicating the benefits of a diet that decreased total and saturated fat and cholesterol and increased dietary potassium, calcium, magnesium, fibre and protein in relation to the standard American diet. The DASH-Sodium study demonstrated incremental further falls in blood pressure at 104mmol/day and 65mmol/day of sodium, confirming that an otherwise ideal diet is more effective when it includes a sodium-reduction guideline. All the food was provided to the participants, thus enabling better control of the important confounding variable of dietary compliance; this is an important difference between DASH-Sodium and previous sodium studies.

The mean decrease in systolic blood pressure when changing from 143mmol/day on the control diet to 65mmol/day on the DASH diet was 7.1mmHg in normotensives and 11.5mmHg in hypertensives, the latter deriving as much benefit as they might expect from antihypertensive medication.³⁸ While on the control diet only and changing from 143mmol/day to 65 mmol/day, the change in systolic blood pressure was 9.8mmHg for African-Americans with hypertension and 6.8mmHg for other racial groups with hypertension. The mean decrease in diastolic blood pressure between the high and low sodium intakes was –3.5mmHg (95%CI: –2.6 to –4.3) on the control diet and –1.6mmHg (95%CI: –0.8 to –2.5) on the DASH diet. The combined effects on blood pressure of low sodium intake and the DASH diet were greater than the effects of either intervention alone and were substantial.

The first DASH trial was widely misinterpreted as having negated the importance of other factors in hypertension such as overweight, alcohol and sodium intake³⁵, but the design of the first study purposely omitted the well-established factors in

order to test the other general dietary guidelines.³⁸ Long-term health benefits of the DASH-Sodium diet remain to be demonstrated, but this large randomised controlled trial with high subject retention rates and excellent compliance with dietary protocols has provided compelling evidence that true reduction of dietary sodium has a substantial effect on blood pressure. The effect on normotensives was enough to justify it as a guideline for the whole population and to predict a substantial effect at the population level.^{38,39}

The TONE study was a randomised controlled trial of reduced sodium intake or weight loss in hypertensives aged 60 to 80 years.³⁶ Of the 975 subjects, 585 were obese and 390 were not. Withdrawal of hypertensive medication was a goal for all subjects. Follow-up visits at nine, 12 and 30 months had attendance rates of 91, 86 and 86 per cent respectively.

The sodium-reduction group reduced their intake by a mean of only 46.6mmol/day at nine months, 49.3mmol/day at 18 months, and 39.5mmol/day at 30 months. The goal for sodium reduction was a total intake of 80mmol/day or less, and only about 38 per cent of the subjects met this target at each visit, compared with about 11 per cent in the control groups. This modest compliance rate resulted in about a 30 per cent decrease in the need for antihypertensive medication in the sodium-reduction group and a better result in subjects who combined weight loss with sodium reduction. The modest dietary sodium reduction of about 40mmol/day was well tolerated and sustained, and the subjects reported no adverse effects.³⁶

Sodium sensitivity

The sensitivity of people's blood pressure response to dietary sodium varies^{41–63}, and salt sensitivity may be related to mortality independently of blood pressure.⁴² Salt sensitivity appears to be a continuous phenomenon⁴³, and its definition is arbitrary; however, it appears to be reproducible in individuals.⁴⁴ A clinically practical means of identifying salt sensitivity is yet to be found.⁴¹ It has been shown that the prevalence of sensitivity increases with age⁴³, raising doubts about the persistence of a determination of being 'insensitive' to dietary salt. Salt sensitivity is also associated with defective endothelial-dependent vasodilation in people with hypertension⁴⁵, although the reason for this is not understood. Salt sensitivity is an area of active research, and a better understanding of the mechanisms involved will probably improve our understanding of the health effects of dietary salt intake.

PRACTICAL ASPECTS OF THIS GUIDELINE

The recommended dietary intake for Australian adults for sodium is 40–100mmol/day, with no extra recommendation for pregnancy or lactation.⁵⁵ To achieve this intake, adults should consume fresh food, foods normally processed without salt, and low-salt or no-added-salt groceries, and they should avoid adding salt to food.⁴⁶ Among the substitutes for salt are acidic ingredients such as

as vinegar, lemon, lime, plum and other fruit juices; curry spices; garlic and onion; and herbs.⁴⁶

Population surveys show that women excrete about 20–25 per cent less sodium than men, but the gender difference was non-significant after adjusting for creatinine excretion in the 1995 Hobart data, suggesting that it reflects the lower muscle mass and thus food energy intake of women.⁴⁷ A lower recommended dietary intake of perhaps 30–80mmol/day may be more appropriate for women, and downward adjustment for older people of both sexes could be proposed on the same grounds.

People wanting to achieve a low sodium intake should choose low-salt foods—that is, with a sodium content up to 120 milligrams per 100 grams. Fresh foods such as fruit, vegetables, meat, milk and yoghurt are well under the sodium limit but most manufactured foods are well over it.⁴⁸ Breads have a sodium content typically as high as 400–725 milligrams per 100 grams. ‘Salt-free’ bread can be difficult to find, but it (and other low-salt products) should be sought out. People used to a higher salt intake will at first miss the taste of salt when they begin a lower salt intake. But the palate adapts to lower sodium levels, and people will find that the intensity of salt in food increases and their ‘preferred saltiness’ of food reduces.^{60,61} Changes will be noticed within a week and taste change will continue for many months.

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

Asian-style cooking has increased in popularity in Australia in recent years. As noted, however, people from a university community in Sydney who consumed an Asian-style diet had a sodium intake that was about 15 per cent higher²², than that of people on a standard Australian diet. Many Asian dishes use ingredients such as soy, oyster and fish sauces, which are high in sodium. In the INTERSALT study—involving urinary measurement of sodium excretion at 52 sites from 32 countries—the study locations in northern Japan and the People’s Republic of China were among those with the highest sodium intakes.⁴⁹

Relationship to the Australian Guide to Healthy Eating

The *Australian Guide to Healthy Eating*⁵⁰ focuses on food and food selection. Individuals should avoid choosing higher salt foods and replace them with foods in the same group that are lower in salt. Much of the salt intake of Australians comes from recommended foods—such as bread, cereals and cheese—and other

frequently consumed foods such as butter, margarine and snack foods. Although equivalent studies have not been conducted in Australia, it has become apparent that the source of most dietary sodium in western countries is not discretionary salt.^{51–55} In particular, cooking salt is a much less important source than was once thought. James et al.⁵¹ used lithium as a marker and found that only a quarter of cooking salt actually enters the consumed food: the rest is discarded with the cooking water. Table 1.7.2 shows the estimated percentages of sodium from different sources, as derived from two studies.

Although it is important to advise people to consume less salt and to buy low-salt foods, the widespread use of salt in processed food and food prepared away from home is a major barrier to achieving any meaningful reduction in dietary sodium intake. If the important public health objective of reducing the mean intake of sodium for all people to at least the top end of the recommended dietary intake range⁵⁵ is to be achieved, far-reaching action is necessary. Realistic medium-term policy objectives would include a gradual reduction in the amount of salt added to processed food and consumer-friendly labelling showing the sodium content of food.

Table 1.7.2 Estimated percentages of sodium intake from different sources in two studies

Sodium source	James et al. ⁵¹	Edwards et al. ⁵²
Natural	10.0	18.5
Added in food manufacturing	75.0	65.0
Discretionary		
Cooking	9.0	9.0
At table	6.0	6.0

A recommendation that food manufacturers reduce the amount of salt added to their products is not without precedent. In 1982 the NHMRC Working Party on Sodium⁵⁶ recommended that food manufacturers be asked to do this; foods such as bread, cheese, butter, margarine, processed meats and snack foods were named as items of critical importance. Food manufacturers responded to this call with the range of reduced- and low-salt alternatives now available. A 1995 survey of 63 brands of processed food sold in Australian supermarkets found an overall decrease in sodium concentration of 10 per cent compared with 15 years previously, the greatest change being evident in convenience foods, cheeses, potato crisps and breakfast cereals.⁵⁷ In 1993 the US National High Blood Pressure Education Program Working Group called for food processors to lower the sodium content of their products⁵⁸, and there is at least some evidence that a modest reduction may have occurred.⁵⁹ Information on sodium content is now included on food labels (in terms of density or standard serve), but it is easy to confuse salt and sodium and consumers may not be aware of the definition of a

low-salt food. As with other nutrients, the sodium content usually needs to be considered in terms of the actual amount of the food consumed. It is particularly difficult for consumers to obtain compositional information on prepared food such as takeaway foods and restaurant meals.

When consumers reduce the amount of salt in their diet, the rated intensity of salt in a solid food increases and the concentration of salt in soup and crackers that previously produced maximum pleasantness decreases.^{60,61} The effects are observed within two months, although they may take many months to reach their full extent. In clinical trials, moderate sodium reduction was not associated with physical complaints or with impairment of quality of life.^{62,63}

Practical considerations for select populations

High-risk groups

Cardiovascular disease mortality is higher among Indigenous Australians, in rural areas, and among socio-economically disadvantaged groups.¹¹ There is a dearth of information on the salt intake of Indigenous Australians. A survey of the food habits of adults living in Victoria⁶⁴ found that Indigenous Australians, whether living in the city or in rural towns, were much more likely to add salt to cooked food than Australians of European descent.

The entire population experiences an age-related increase in blood pressure, which could probably be largely prevented if the population were to consume dietary sodium within the recommended dietary intake. In the current food environment, it is very difficult to maintain a low intake of dietary sodium: population measures are needed that will gradually decrease the amount of salt in our food supply and improve people's ability to make healthy choices in relation to their food.

Hypertensives

Hypertensive patients need a better choice of groceries than is available at present. During the 1980s a major supermarket chain brought out a range of no-added-salt processed foods, consisting at one stage of 23 different items.⁶⁵ Producers will meet consumer demand, and there is no doubt that this range would have expanded had the turnover reflected the real needs of the 3 million or so Australians who have hypertension. But the Australian medical profession at present ignores the international consensus that is in favour of changing a person's lifestyle before starting drug treatment.^{66,67} With the exception of emergency treatment of severe cases, medication for hypertension should be considered only after a six-month trial of non-pharmacological measures, including a low-salt diet; and when drug treatment is necessary these measures should be continued as an adjunct, to permit better control at a lower dose.^{66,67} Doctors behave very differently in Finland, where the government withholds the subsidy for antihypertensive medication until the prescriber implements this policy.⁶⁸

People living in iodine deficient areas

Iodine deficiency disorders were once common in several Australian states, and a traditional control measure has been the sale of iodised salt. Only table salt is iodised, however, and its use has declined to the point where in a Hobart survey in 1995 over 50 per cent of both sexes stated that they neither cooked with salt nor used it at the table.²⁰ Mild iodine deficiency is now regarded as an important cause of preventable mental retardation, and it is alarming that urinary excretion has revealed moderate to severe iodine deficiency, even in a survey of outpatients (including pregnant women) at a metropolitan hospital in Sydney.⁶⁹ Adherence to the salt guideline reduces salt's availability as a vehicle for iodine, and iodine fortification of one or more staple foods, such as bread, may need to be considered.

RELATIONSHIP TO OTHER GUIDELINES

The first edition of the *Dietary Guidelines for Australians*² included the guideline 'Eat less salt'. The second edition revised this to 'Choose low salt foods and use salt sparingly', in recognition of the fact that the primary source of sodium in the Australian diet is salt added to manufactured food. For this third edition, a review of the literature leads to the conclusion that the most rational way to achieve lower sodium intakes is through selection of low-salt foods. It is acknowledged that discretionary salt use is an important source of dietary sodium for many people; however, a single-focus, simple message is considered an important priority.

This guideline is consistent with each of the other guidelines. Many manufactured foods have a large amount of salt added: these should be avoided while maintaining (or attaining) a healthy intake of breads and cereals and fruit and vegetables. A lower dietary sodium intake would be much more easily achieved if manufacturers in general were to decrease the amount of salt added to their products—following the lead that some manufacturers have set—and consumers were easily able to assess the sodium content of manufactured food.

Many diet-related factors are likely to be helpful in treating hypertension and limiting the risk of developing hypertension. Among these are maintaining a healthy body weight, a moderate alcohol intake, and a relatively high potassium intake, and opting for a diet high in fruits and vegetables and low-fat dairy products (the DASH diet).

CONCLUSION

The past decade has seen the emergence of international consensus that a modest reduction in dietary sodium intake for people with normal and raised blood pressure has a sufficiently large effect on blood pressure (and therefore

health) to justify a guideline advising restraint for the entire population.⁷⁰ This consensus is strongly supported by a large and well-conducted randomised controlled trial³¹ in which subject retention was high and dietary compliance was optimised by providing all of the food throughout the trial period.

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

Primary prevention of hypertension poses one of the greatest challenges for public health in the 21st century, and reducing dietary salt is a leading population health strategy for achieving this goal.

EVIDENCE

There is strong evidence that reducing dietary sodium lowers average blood pressure in groups of people whose blood pressure is raised. The evidence comes from well-conducted randomised controlled trials and is supported by meta-analyses of these—Level I evidence (references 25 and 32 to 34). The size of the effect is clinically important and is larger for older individuals and at higher blood pressures. The effect appears to be evident for ‘high-normal’ blood pressure; that is, when blood pressure is not high enough to be categorised ‘hypertensive’. The evidence is relevant even though randomised controlled trials have not been conducted to assess mortality or morbidity outcomes other than change in blood pressure.

There is also Level II evidence (references 4, 28, 35 to 37, 62 and 63), Level III evidence (references 5, 24 and 43) and Level IV evidence (references 20, 21, 23 and 40) of the relationship between blood pressure and salt reduction.

The evidence that reducing dietary sodium intake to the recommended intake of 40–100mmol/day causes adverse health effects is weak. Adverse health effects have not been observed in the randomised controlled trials conducted to date. Given the low dietary sodium intake observed in many populations without apparent specific ill-effects and the presumed low intake by humans (by analogy with other terrestrial mammals) over much of their history, it is unlikely that any adverse health effects of lowering sodium intake would be seen over time.

REFERENCES

1. Kurtz TW, Al-Bander H, Morris RC. 'Salt-sensitive' essential hypertension in man. Is the sodium ion alone important? *New Engl J Med* 1987;317:1043–8.
2. National Health and Medical Research Council. *Recommended dietary intakes for use in Australia*. Canberra: AGPS, 1991.
3. Alderman MH. Salt, blood pressure and human health. *Hypertension* 2000;36:890–3.
4. Law MR, Frost CD, Wald NJ. By how much does dietary salt reduction lower blood pressure? III. Analysis of data from trials of salt reduction. *BMJ* 1991;302:819–24.
5. MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J et al. Blood pressure, stroke and coronary heart disease. Part 1. Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. *Lancet* 1990;335:765–74.
6. Law MR. Epidemiologic evidence on salt and blood pressure. *Am J Hypertension* 1997;10:42S–45S.
7. Australian Institute of Health and Welfare. *Australia's health 2000: the seventh biennial health report of the Australian Institute of Health and Welfare*. Canberra: AIHW, 2000.
8. National Health and Medical Research Council. *Clinical practice guidelines: prevention of stroke—the role of anticoagulants, antiplatelet agents and carotid endoarterectomy*. Canberra: Australian Government Publishing Service, 1997.
9. Britt H, Sayer GP, Miller GC, Charles J, Scahill S, Horn F et al. *General practice activity in Australia, 1998–99*. General Practice Series no. 2. Cat. no. GEP2. Canberra: Australian Institute of Health and Welfare, 1999.
10. Australian Institute of Health and Welfare. *Heart, stroke and vascular diseases: Australian facts*. Cardiovascular Disease Series no. 10. Cat. no. CVD 7. Canberra: AIHW, National Heart Foundation of Australia & National Stroke Foundation of Australia, 1999.
11. Department of Health and Aged Care & Australian Institute of Health and Welfare. *National health priority areas report: cardiovascular health, 1998—summary*. AIHW cat. no. PHE 12. Canberra: DHAC & AIHW, 1999.
12. National Heart Foundation of Australia. *1999 guide to the management of hypertension for doctors*. NHF <www.heartfoundation.com.au>.
13. Top 10 drugs. *Australian Prescriber* 1999;22(5):119.
14. Freis ED, Wanko A, Wilson IM, Parrish AE. Treatment of essential hypertension with chlorothiazide (Diuril). *JAMA* 1958;166:137–40.
15. Morgan T, Myers J. Diuretics. *Curr Therapeutics* 1981;22:93–7.

16. Van Brummelen P, Schalekamp M, de Graff J. Influence of sodium intake on hydrochlorothiazide-induced changes in blood pressure, serum electrolytes, renin and aldosterone in essential hypertension. *Acta Med Scand* 1978;204:151–7.
17. MacGregor GA. Salt—more adverse effects. *Am J Hypertension* 1997;10:37S–41S.
18. Carey OJ, Locke C, Cookson JB. Effect of alterations of dietary sodium on the severity of asthma in men. *Thorax* 1993;48:714–18.
19. Liu K, Cooper R, McKeever J, McKeever P, Byington R, Soltero I et al. Assessment of the association between habitual salt intake and high blood pressure: methodological problems. *Am J Epidemiol* 1979;110:219–26.
20. Beard TC, Woodward DR, Ball P, Hornsby H, von Witt RJ, Dwyer T. The Hobart salt study 1995: few meet national sodium intake target. *Med J Aust* 1997;166:404–7.
21. Beard TC, Eickhoff R, Mejglo ZA, Jones M, Bennett SA, Dwyer T. Population-based survey of human sodium and potassium excretion. *Clin Exp Pharmacol Physiol* 1992;19:327–30.
22. Notowidjojo L, Truswell AS. Urinary sodium and potassium in a sample of healthy adults in Sydney, Australia. *Asia Pacific J Clin Nutr* 1993;2:25–33.
23. Beard TC, Blizzard L, O'Brien DJ, Dwyer T. Association between blood pressure and dietary factors in the Dietary & Nutritional Survey of British Adults. *Arch Intern Med* 1997;157:234–8.
24. Ambard L, Beaujard E. Causes de l'hypertension artérielle. *Archives générales de médecine* 1904;1:520–33.
25. Graudal NA, Galloe AM, Garred P. Effects of sodium restriction on blood pressure, rennin, aldosterone, catecholamines, cholesterol and triglyceride: a meta-analysis. *JAMA* 1998;279:1383–91.
26. Law MR, Frost CD, Wald NJ. By how much does dietary salt reduction lower blood pressure? I. Analysis of observational data among populations. *BMJ* 1991;302:811–15.
27. Denton D, Weisinger R, Mundy NI, Wickings EJ, Dixon A, Moisson P et al. The effect of increased salt intake on blood pressure of chimpanzees. *Nature Med* 1995;1:1009–16.
28. Dahl LK. Salt and hypertension. *Am J Clin Nutr* 1972;25:231–44.
29. Kuller LH. Salt and blood pressure: population and individual perspectives. *Am J Hypertens* 1997;10:29S–36S.
30. Intersalt Cooperative Research Group. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. *BMJ* 1988;297:319–28.

31. Frost CD, Law MR, Wald NJ. By how much does dietary salt reduction lower blood pressure? II. Analysis of observational data within populations. *BMJ* 1991;302:815–18.
32. Cutler JA, Follmann D, Allender PS. Randomized trials of sodium reduction: an overview. *Am J Clin Nutr* 1997;65(suppl.):643S–651S.
33. Cutler JA, Follmann D, Elliott P, Suh IL. An overview of randomized trials of sodium reduction and blood pressure. *Hypertension* 1991;17(suppl. 1):27–33.
34. Midgley JP, Matthew AG, Greenwood CMT, Logan AG. Effect of reduced dietary sodium on blood pressure: a meta-analysis of randomized controlled trials. *JAMA* 1996;275:1590–7.
35. Sacks FM, Svetley LP, Vollmer WM, Appel LJ, Bray GA, Harsha D et al. Effects on blood pressure of reduced dietary sodium and the dietary approaches to stop hypertension (DASH) diet. *New Engl J Med* 2001;344:3–10.
36. Whelton PK, Appel LJ, Espeland MA, Applegate WB, Ettinger WH, Kostis JB et al. for the TONE Collaborative Research Group. Sodium reduction and weight loss in the treatment of hypertension in older persons: a randomized controlled trial of nonpharmacologic interventions in the elderly (TONE). *JAMA* 1998;279:839–46.
37. Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM et al. for the DASH Collaborative Research Group. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med* 1997;336:1117–24.
38. Greenland P. Beating high blood pressure with low-sodium DASH. Editorial. *New Engl J Med* 2001;344:53–5.
39. Cook NR, Cohen J, Hebert PR, Taylor JO, Hennekens CH. Implications of small reductions in diastolic blood pressure for primary prevention. *Arch Intern Med* 1995;155:701–9.
40. Luft FC, Weinberger MH. Heterogeneous responses to changes in dietary salt intake: the salt-sensitivity paradigm. *Am J Clin Nutr* 1997;65:612S–617S.
41. Mattes RD, Falkner B. Salt-sensitivity classification in normotensive adults. *Clin Sci* 1999;96:449–59.
42. Weinberger MH, Fineberg NS, Fineberg SE, Weinberger M. Salt sensitivity, pulse pressure, and death in normal and hypertensive humans. *Hypertension* 2001;37(part 2):429–32.
43. Weinberger M. Sodium and volume sensitivity of blood pressure: age and pressure change over time. *Hypertension* 1991;18:67–71.
44. Weinberger MH. Salt sensitivity of blood pressure in humans. *Hypertension*. 1996;27:481–90.
45. Bragulat E, Sierra A, Antonio MT, Coca A. Endothelial dysfunction in salt-sensitive essential hypertension. *Hypertension* 2001;37(part 2):444–8.

46. McCarron DA. The dietary guideline for sodium: should we shake it up? Yes! *Am J Clin Nutr* 2000;71:1013–19.
47. Woodward DR, Beard TC, Ball PJ, Hornsby H, von Witt RJ, Dwyer T. Should the male and female RDI for sodium be the same? Abstract. In: *Proceedings of 16th Dietitians Association of Australia National Conference, 14–17 May. Hobart*. Canberra: Dietitians Association of Australia, 1997.
48. Beard TC. *Salt in medical practice*. 2nd edn. Holland Park: Queensland Hypertension Association, 2000.
49. Intersalt Cooperative Research Group. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. *BMJ* 1988;297:319–28.
50. Department of Health and Family Services. *The Australian guide to healthy eating: background information for nutrition educators*. Canberra: DHFS, 1998.
51. James WPT, Ralph A, Sanchez-Castillo CP. The dominance of salt in manufactured food in the sodium intake of affluent societies. *Lancet* 1987;1:426–9.
52. Edwards DG, Kaye AE, Druce E. Sources and intakes of sodium in the United Kingdom diet. *Eur J Clin Nutr* 1989;43:855–61.
53. Stamler J. Dietary salt and blood pressure. *Ann NY Acad Sci* 1993;676:122–56.
54. Mattes RD, Donnelly D. Relative contributions of dietary sodium sources. *J Am Coll Nutr* 1991;10:383–93.
55. National Health and Medical Research Council. *Recommended dietary intakes for use in Australia*. Canberra: Australian Government Publishing Service, 1991.
56. National Health and Medical Research Council. *Report of the working party of sodium in the Australian diet*. Canberra: AGPS, 1982.
57. National Blood Pressure Advisory Committee. *Salt and hypertension: a paper for health professionals*. National Heart Foundation of Australia <www.heartfoundation.com.au>.
58. National High Blood Pressure Education Program Working Group. Report on primary prevention of hypertension. *Arch Intern Med* 1993;153:186–208.
59. Jacobson MF, Liebman BF. Letter. Sodium in processed foods. *Am J Clin Nutr* 1996;63:138–40.
60. Blais CA, Pangborn RM, Borhani NO, Ferrell MF, Prineas RJ, Laing B. Effect of dietary sodium restriction on taste responses to sodium chloride: a longitudinal study. *Am J Clin Nutr* 1986;44:232–43.
61. Bertino M, Beauchamp GK, Engelman K. Long-term reduction in dietary sodium alters the taste of salt. *Am J Clin Nutr* 1982;36:1134–44.

62. Grimm RH Jr, Grandits GA, Cutler JA, Stewart AL, McDonald RH, Svendsen K et al. Relationships of quality-of-life measures to long-term lifestyle and drug treatment in the Treatment of Mild Hypertension Study. *Arch Intern Med* 1997;157:638–48.
63. Kumanyika S. Behavioral aspects of intervention strategies to reduce dietary sodium. *Hypertension* 1991;17(suppl. 1):190–5.
64. Guest C, O'Dea K. Food habits in Aborigines and persons of European descent of southeastern Australia. *Aust J Publ Hlth* 1993;17:321–4.
65. Beard TC, Farrance I. Danger of over-medication with no-added-salt diets. Letter. *Med J Aust* 1987;147:46–7.
66. WHO–ISH Guidelines Committee. 1993 guidelines for the management of mild hypertension. *Hypertension* 1993;22:392–403.
67. The management of hypertension: a consensus statement. *Med J Aust* 1994;160(suppl.):S11.
68. Godlee F. The food industry fights for salt. *BMJ* 1996;312:1239–40.
69. Gunton JE, Hams G, Fiegert M, McElduff A. Iodine deficiency in ambulatory participants at a Sydney teaching hospital: is Australia truly replete? *Med J Aust* 1999;171:467–70.
70. WHO Expert Committee. *Hypertension control*, WHO Technical Report Series no. 862. Geneva: World Health Organization, 1986.

I.8 LIMIT YOUR ALCOHOL INTAKE IF YOU CHOOSE TO DRINK

A Stewart Truswell

TERMINOLOGY

Standard drink

A standard drink contains 10 grams of alcohol—equivalent to 12.5 millilitres of alcohol.

Pattern of drinking

Pattern of drinking refers to aspects of drinking behaviour other than total or mean amounts consumed in a specific period. It includes measures of the number and characteristics of drinking occasions and the types of drinks consumed.

BACKGROUND

Alcohol has been consumed in various forms in Australia since the earliest days of European settlement. At that time it was not part of the diet of the original Australians, the Aboriginal and Torres Strait Islander peoples. It was introduced by the British when the First Fleet arrived in 1788.

In terms of nutrition, alcohol is in a unique position: it is the only substance that is both a nutrient and a drug affecting brain function. Advice about alcohol was omitted from the first two international sets of dietary goals or guidelines, produced in the Nordic countries in 1968 and in the United States in 1977. Welcoming the development of dietary guidelines for the community, a 1997 *Lancet* editorial¹ pointed out, ‘Their major blind spot is to ignore alcohol consumption, which is increasing fast, along with its pernicious effects’. Since then, caution about alcohol intake has been included in nearly all national sets of dietary guidelines from North America, Europe, Asia and Oceania. Alcohol is the fourth macro-nutrient (with carbohydrates, fats and proteins) that provides dietary energy (kilojoules), and its inclusion in these Dietary Guidelines for Australian Adults is justified on this point alone.

The main reason people take alcoholic drinks is for the relaxing and socialising effect on the brain of small to moderate doses. The reason health authorities caution about alcohol use is that high doses severely impair brain function—they can cause coma and even death from direct intoxication—and alcohol is a habit-forming drug, the most commonly used recreational drug in Australia after

tobacco. In addition, alcohol can have toxic effects, directly or indirectly, on other systems in the body and can affect, among other things, liver function, the cardiovascular system, several metabolic processes, foetal development and the risk of cancer. Alcohol is an outstanding example of the toxicological principle: 'The dose determines the effect'.

Society's attitudes to alcohol and its use are markedly ambivalent. Many social occasions—from celebrations such as birthdays, engagements, graduations, weddings, sports victories and job promotions to nearly all entertainment and hospitality events—are accompanied by the dispensing of alcoholic drinks. Vineyards and wine making are seen as romantic; wine tasting is considered a sophisticated occupation or pastime; some wines fetch very high prices. Beer and the pub are important for mateship. The production and retailing of alcoholic drinks provide large numbers of direct and indirect jobs, and wine is Australia's most rapidly growing agricultural export. On the other hand, society controls the sale and use of alcohol where it may create a hazard.

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

Excessive drinking of the type that causes long-term harm was estimated to have caused 3290 deaths in Australia in 1997—accounting for 4 per cent of male deaths and 2 per cent of female deaths—and 50 000 hospitalisations. Acute and chronic effects of excessive drinking made equal contributions.²

It has been estimated that the cost of excessive alcohol drinking—to the Australian health care system and to industry through absenteeism, premature retirement and impaired or lost productivity—was some \$4.5 billion in 1992.³ The NHMRC's *Australian Alcohol Guidelines: health risks and benefits* provide a detailed review of the subject.⁴

Current intakes

Table 1.8.1 shows the percentiles of intake of alcohol (in grams) consumed on the day of the 1995 National Nutrition Survey.²⁵ It also shows the percentiles for energy derived from alcohol. It should be remembered that the data were gathered using a 24-hour recall technique, which assessed intake on the day before the survey for any given individual. The data do not therefore give an estimate of the 'usual' intake of individuals, but they can shed some light on the population's consumption patterns.

The survey results showed that alcohol provided on average 4.8 per cent of energy intake in men (an average of 18.5 grams a day) and 2.6 per cent in women (an average of 7.3 grams a day). Since intakes were 1.3 to 2.1 times higher on Saturday and Sunday and 76 per cent of survey participants were interviewed on a week day these are slight underestimates. In those who

reported drinking alcohol on the day of the survey, the average dietary energy provided by alcohol was 11.4 per cent for men and 10.6 per cent for women. Beyond these averages is a minority of people who obtain more dietary energy from alcohol than from protein.

Nearly all alcohol is consumed in beverages, principally beers and wines, which contain other nutrients—sugars, some inorganics (such as potassium), a few vitamins (for example, folate in beer) and bioactive substances (flavonoids). Alcoholic beverages are usually consumed with foods, either as part of a meal or accompanied by snack foods.

Table 1.8.1 Means and percentiles of alcohol intake in adults aged 19 years and over: 1995 National Nutrition Survey²⁵

and over 1975 National Nutrition Survey													
	Percentile												
Adults (19+ years)	5th	10th	20th	30th	40th	50th	60th	70th	80th	90th	95th	Mean	
Males													
% energy from alcohol	—	—	—	—	—	—	1.8	5.3	9.7	15.9	21.9	4.8	
Grams of alcohol	—	—	—	—	—	—	8.0	19.1	35.1	57.2	85.8	18.5	
No. of standard drinks	—	—	—	—	—	—	0.8	1.9	3.5	5.7	8.6	1.9	
Females													
% energy from alcohol	—	—	—	—	—	—	—	—	3.5	9.8	15.8	2.6	
Grams of alcohol	—	—	—	—	—	—	—	—	9.9	25.9	43.1	7.3	
No. of standard drinks	—	—	—	—	—	—	—	—	1.0	2.6	4.3	0.7	
Male consumers ^a													
% energy from alcohol	1.9	2.8	4.2	5.6	7.3	9.2	11.3	13.7	17.4	23.5	28.4	11.4	
Grams of alcohol	8.0	10.7	14.3	21.2	28.1	32.4	42.5	50.1	65.4	88.1	114.4	44.2	
No. of standard drinks	0.8	1.1	1.4	2.1	2.8	3.2	4.3	5.0	6.5	8.8	11.4	4.4	
Female consumers ^a													
% energy from alcohol	1.6	2.6	3.8	5.3	6.5	8.2	10.1	12.8	15.9	22.1	27.9	10.6	
Grams of alcohol	4.3	7.2	10.7	14.0	17.5	21.7	27.1	34.6	43.5	59.3	81.7	30.0	
No. of standard drinks	0.4	0.7	1.1	1.4	1.8	2.2	2.7	3.5	4.4	5.9	8.2	3.0	

— Zero.

a. 'Consumers' refers to survey participants consuming alcohol on the survey day.

Note: Care should be taken when interpreting the percentile distributions: they relate to intakes reported on the survey day only.

1.8 LIMIT YOUR ALCOHOL INTAKE IF YOU CHOOSE TO DRINK

The most recent apparent consumption figures for various alcoholic drinks in Australia show an annual per capita consumption of:

- 94.7 litres for beer
- 17.3 litres for wines
- 1.7 litres for fortified wines
- 1.4 litres for spirits.⁵

Total pure alcohol (ethanol) consumption per head of total population per year was 7.6 litres. This is slightly higher than the 1995 National Nutrition Survey data suggest, but the two measures use different methodologies. The National Nutrition Survey reported individual consumption on a given day, whereas the apparent consumption methodology estimates alcohol available per head of population by assessing production, imports, exports, and so on, in relation to population size.

The Australian Institute of Health and Welfare⁵ produced the following estimates for 1998:

- Fifty-nine per cent of males and 39 per cent of females aged over 14 years drank alcoholic beverages at least once a week.
- Of current drinkers, 14 per cent of males and 6 per cent of females drank every day and 13 per cent of men and women drank four to six days a week.
- Thirty-six per cent of males and 62 per cent of females reported drinking one to two glasses of alcohol at a time.
- Thirty-one per cent of males and 22 per cent of females reported drinking three to four glasses at a time.
- Thirty-three per cent of males and 15 per cent of females stated they drank five or more glasses at a time.

Most recommendations concerning alcohol consumption are made on the basis of 'standard' drinks consumed. A standard drink in Australia is considered to contain 10 grams of alcohol (equivalent to 12.5 millilitres of alcohol—the density of ethyl alcohol is 0.79). The alcohol concentration of drinks is printed on the label in terms of percentage by volume.

Table 1.8.2 shows how various drinks equate to the standard 10-gram drink and Table 1.8.3 shows the energy and alcohol content of a number of common drinks. It is of interest to note that many of these common 'serves' of alcoholic drinks contain more than the 'standard' 10 grams of alcohol.

Table 1.8.2 Standard drink equivalents

Drink type	Standard drink equivalent
Beer	
Regular beer	
1 can/stubby	1° standard drinks
1 jug	4 standard drinks
1 slab (cans or stubbies)	About 36 standard drinks
Medium–light beer	
1 can or stubbie	1 standard drink
Light beer	
1 can or stubbie	° standard drink
Wine	
100-ml glass	1 standard drink
750-ml bottle	About 7–8 standard drinks
4-litre cask	About 30–40 standard drinks
Spirits	
1 nip (30ml)	1 standard drink
Pre-mixed spirits	
1 can (375ml)	1° standard drinks

Table 1.8.3 Energy and alcohol content of common alcoholic drink serves

Drink type	Serve size	Energy (kJ/serve)	Alcohol (g/serve) ^a	Notes ^b
Beer, regular	1 can/stubby (375ml)	568	15.4	Highest-alcohol beer in NNS database is 4.9%. Most regular beers appear to be around 4.5%.
Beer, medium–light	1 can/stubby (375ml)	546	14.0	NNS gives a figure for medium–light ‘beer, bitter or draught’ of 3.7% alcohol.
Beer, light	1 can/stubby (375ml)	395	8.0	NNS data tables give 2.1% alcohol as an average for light beer. However, the description of ‘light’ covers beers from 0.9 to 3.3% alcohol.
Wine	1 glass (100ml)	227	9.5	NNS used average of red wine and dry white wine (9.5–13% alcohol).
Spirits	1 nip (30ml)	255	8.8	NNS used ‘spirits, type not stated’.
Pre-mixed spirits	1 can (375ml)	1182	13.9	NNS used average of 7 pre-mixed canned spirit drinks.

a. Using 1995 National Nutrition Survey data.²⁵

b. NNS = National Nutrition Survey.

Until recently, most recommendations concerning alcohol consumption in Australia and overseas (see appendix 5 in reference 4) have given a single average daily maximum figure for standard drinks for men and women, with the male figure generally being higher than the female one. Not only do women generally have a smaller body size; they also metabolise alcohol differently. This makes them more susceptible to adverse effects at a particular intake.^{6,7}

SCIENTIFIC BASIS

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

- the inexperienced drinker who misjudges the dose and has an accident—the primary cause of death in adolescents and young men
- the person who doesn't drink during the week but may drink to excess and get drunk on a weekend evening or at a party
- the person who enjoys a controlled drink or two most days with their evening meal
- the person who has too many drinks each day but more or less maintains a normal—but probably increasingly inefficient—lifestyle
- the person who engages in weeks of heavy drinking—and eats very little during this time.

Some of these patterns of consumption may result in adverse acute health and/or social outcomes, some in long-term health and/or social consequences, and some in both types of consequence.

Acute health effects of alcohol⁴⁻⁹

The first potentially adverse acute effects of consumption are disinhibition and loss of skilled movements, which start to happen at blood alcohol levels of about 0.05 per cent (0.05 grams per decilitre, or nearly 11mmol/L). This is the legal limit of blood alcohol for driving in Australia; it was based on experiments testing such skills as reversing double-decker buses in narrow spaces after different doses of alcohol.⁸

Above this intake and blood alcohol concentration, performance, behaviour and health deteriorate progressively: the subject becomes obviously drunk, then stuporose, then comatose. Alcohol intoxication is a major factor in violence and accidents, especially motor vehicle accidents. It also leads to loss of productivity in the workplace and to social discord, including domestic violence and homicide. Figure 1.8.1 shows the risk of accidents associated with particular blood alcohol levels.

In 1997 in Australia 418 deaths on the road were caused by hazardous and harmful alcohol consumption, with a consequent loss of 17 174 person-years of life since most of the deaths occurred in young people. Additionally, 7789 people were hospitalised—for 44 997 bed-days⁹—for acute conditions caused by alcohol consumption.

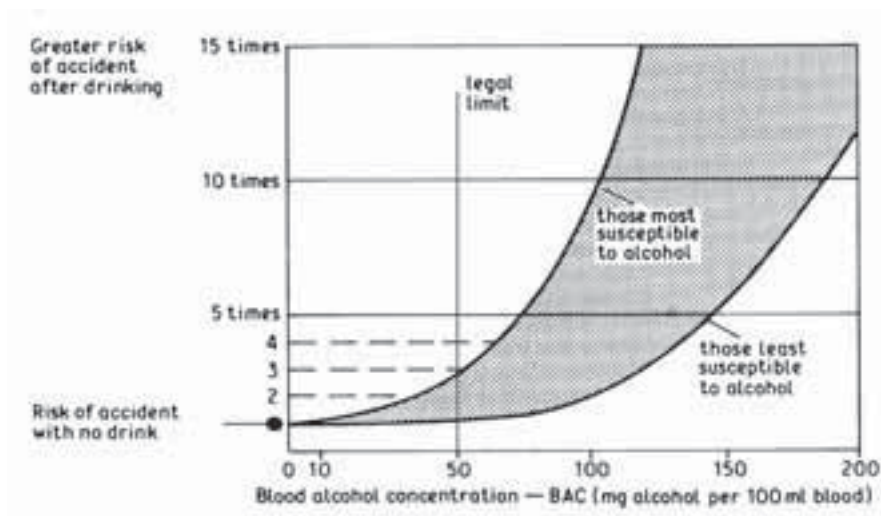


Figure 1.8.1 Blood alcohol levels and the risk of accidents

Source: Adapted from Royal College of Physicians.

A great and growing evil: the medical consequences of alcohol abuse. London: Tavistock, 1987.

Table 1.8.4 Types of traumatic injury and death attributed to alcohol in Australia, 1997⁴

Trauma	Proportion attributed to alcohol	
	Males (% of all cases)	Females (% of all cases)
Road injuries	37	18
Suicide	12	8
Males and females (% of all cases)		
Fall injuries	34	
Fire injuries	44	
Drowning	34	
Assault	47	
Child abuse	16	

The NHMRC's Australian Alcohol Guidelines⁴, which were based on a systematic review of the evidence, concluded that the amount of alcohol consumed per occasion—and, more specifically, the blood alcohol concentration—is the critical feature in determining risk of injury. Blood alcohol concentrations as low as 0.05 grams per decilitre can affect psychomotor skills and increase the risk of injury in circumstances such as driving. Significant individual differences do, however, make it difficult to calculate blood alcohol concentration accurately from the number of drinks and the time taken to consume them. The degree of risk also depends on the setting, the need for high-order physical skills, and the

person's experience with the task at hand. Table 1.8.4 provides information on the types of traumatic injury and death attributed to alcohol in Australia in 1997.

Chronic health effects of excess alcohol consumption^{4,10–17}

Excess alcohol consumption can result in many chronic health effects. Perhaps the most important of these in terms of public health are:

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

Overweight and obesity, which are of increasing concern, may also be related to excess alcohol intake.

Among other long-term effects related to excess alcohol consumption are the following:

- metabolic conditions—for example, hypoglycaemia, lactic acidosis, hyperuricaemia and gout, hypertriglyceridaemia, and acetaldehyde reaction
- nutritional conditions—for example, Wernicke-Korsakoff syndrome, folate deficiency, vitamin A depletion, pellagra, and foetal alcohol syndrome
- endocrine conditions—for example, hypercortisonism and sexual dysfunction
- nervous system disorders—for example, impaired cognition, convulsions, withdrawal syndrome (delirium tremens), peripheral neuropathy, and alcohol-related brain damage
- gastritis and gastric ulcers
- aspiration pneumonia
- cardiomyopathy
- interactions with both pharmaceuticals and illegal recreational drugs.

An evidence-based review of the scientific literature developed as a background document for the NHMRC's Australian Alcohol Guidelines⁴ looked in depth at the major sequelae of alcohol consumption. Some of the conclusions in relation to three important areas—hypertension, cancers and cirrhosis—are summarised in the following paragraphs.

Hypertension and stroke

The risk of hypertension increases with heavier drinking, and reducing alcohol consumption will reduce blood pressure. At lower levels of drinking, the picture is not as consistent, the possible effects of reduced alcohol consumption on blood pressure need to be considered against evidence that there are potential cardiovascular benefits associated with regular low-level drinking—one or two standard drinks for men and less than one a day for women.

High blood pressure is a major risk factor for both ischaemic and haemorrhagic stroke. Alcohol does, however, reduce the risk of atherosclerosis, which might overcome some of this effect in relation to ischaemic stroke. Alcohol also has a complex effect on blood clotting, which may increase the risk of haemorrhagic stroke and partly explain the decreased risk of ischaemic stroke in light drinkers and the increased risk in heavy drinkers.

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

Cancer

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

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

Cirrhosis of the liver

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

Overall, the evidence suggests that, in terms of the incidence of liver cirrhosis in a given community, the degree to which alcohol is responsible varies according to the per capita alcohol consumption.

Alcohol, energy intake and body weight

The Australian Alcohol Guidelines did not specifically deal with the question of alcohol and overweight. Although epidemiological studies show little relationship between alcohol intake and body mass index in men, and even an inverse

relationship in women^{18,19}, experimental studies suggest that alcohol energy is additive to the normal diet^{18,20,21} and that it contributes to excess energy intake and fat storage by increasing appetite²² and by displacing fat and carbohydrate oxidation.²³ Prentice has suggested that the disparity between the epidemiological and the experimental data might be a result of confounding by other lifestyle factors.²⁴

Table 1.8.5 shows the contribution that four standard alcohol drinks, in various forms, would make to the overall energy intake of men and how much two standard drinks would contribute to the overall energy intake in females. It should be noted that a can or stubby of the regular and medium-light beers and a can of pre-mixed spirits contain 14–15 grams of alcohol, not the ‘standard’ 10 grams, but they are common serving units and so are included in the table.

If men chose to consume four standard drinks of beer—each of 250 millilitres, enough to provide 10 grams of alcohol—this would account for some 13–14 per cent of average male energy intake, as assessed by the 1995 National Nutrition Survey.²⁵ If they consumed four cans or stubbies, this would contribute as much as 20 per cent of overall energy intake. Because of their high sugar and alcohol content, four standard serves of pre-mixed spirits would provide some 29 per cent of dietary energy in men; four standard serves of wine or spirits would account for 8–9 per cent of average energy intake. For women, two standard drinks a day would provide about 6–10 per cent of dietary energy, unless taken as pre-mixed spirits, which would account for just over 20 per cent for two standard serves.

Table 1.8.5 The potential contribution of four or two common alcoholic drinks to energy intake in the average male or female diet

Drink type	Serve size	Four drinks		Two drinks	
		Energy (kJ)	% energy contributed to average male diet	Energy (kJ)	% energy contributed to average female diet
Beer, regular	1 can/stubby (375ml)	2272	20.6	1136	15.2
	1 glass (250ml)	1514	13.7	757	10.1
Beer, medium-light	1 can/stubby (375ml)	2184	19.8	1092	14.6
	1 glass (250ml)	1454	13.2	727	9.7
Beer, light	1 can/stubby (375ml)	1580	14.3	790	10.6
Wine	1 glass (100ml)	908	8.2	454	6.1
Spirits	1 nip (30ml)	1020	9.2	510	6.8
Pre-mixed spirits	1 can (375ml)	4728	43.0	2364	32.0
	1 glass (250ml)	3151	29.0	1576	21.0

Note: One standard alcohol serve contains about 10 grams of alcohol. Table uses average energy intakes from the 1995 National Nutrition Survey²⁵ of 11 050 kJ for males and 7480 kJ for females.

Alcohol, together with refined or added sugars contributes what are often termed *empty kilojoules* to the diet: they provide energy without substantial amounts of other essential nutrients. An analysis of the 1995 National Nutrition Survey data (see Table 1.8.6) showed that alcohol and added sugar contributed, on average, some 15.5 per cent of the energy in men's diets and 12.5 per cent in women's diets, but for 20 per cent of the population alcohol and refined sugars together contributed about one-fifth of dietary energy on the day of the survey. If these empty kilojoules are consumed on top of normal energy requirements, as is suggested by some researchers^{18,20,21}, overweight and obesity are likely outcomes over time. If consumed instead of foods or drinks that also supply essential nutrients, this could over time lead to deficiency of key nutrients.

Table 1.8.6 Percentiles of energy from alcohol plus added sugar in adults aged 19 and over: 1995 National Nutrition Survey²⁵

Adults (19+ years)	Percentile											
	5th	10th	20th	30th	40th	50th	60th	70th	80th	90th	95th	Mean
Males												
% energy	2.3	4.1	7.0	9.6	11.8	13.9	16.5	19.2	23.0	28.1	33.6	15.5
Energy (kJ)	178	343	663	952	1205	1453	1742	2093	2586	3477	4411	1748
Females												
% energy	0.9	2.2	4.4	6.3	8.2	10.3	12.8	15.5	18.8	24.3	30.0	12.3
Energy (kJ)	44	119	264	406	558	726	935	1176	1499	2045	2660	963

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

From prospective cohort studies reporting during the 1980s and 1990s it has emerged that people who average one or two alcoholic drinks a day have better life expectancy than teetotallers.²⁶ This result has been reported for more than 20 large studies in at least nine countries, one of them Australia (with two studies).^{27,28}

Moderate intake of alcohol reduces the risk of coronary heart disease.²⁹ It increases the concentration of HDL cholesterol³⁰ and probably reduces platelets' ability to aggregate.³¹ It may also increase insulin sensitivity^{32,33}, and it is possible that the polyphenols in red wine (which are antioxidants *in vitro*) reduce atheroma formation.³⁴ Doll³⁵ concluded that for middle-aged and older men in Britain alcohol has beneficial effects in the intake range of one to four units a day and that for women the beneficial intake range is somewhat less.

But it is only among people who are carefully moderate drinkers and who are at an age and in a section of the population at fairly high risk of CHD that alcohol reduces mortality. For the majority of people in every country, alcohol

consumption increases accidents, social disruption, and disease and death from the acute and chronic effects of excess alcohol intake. Prospective studies are nearly always made with middle-aged people: their results cannot be applied to the rest of the population.

Scragg³⁶ estimated the number of deaths caused or prevented by alcohol in New Zealand. The association between alcohol and total mortality was related to age. Alcohol was estimated to have caused 20 per cent of deaths among 15–34 year olds, mostly from road accidents. In contrast, it prevented 0.5 per cent of deaths among 35–64 year olds and 3.4 per cent of deaths among people aged 65 years or more because of its protective effect against CHD. For all ages combined, alcohol prevented 1.5 per cent of deaths. The number of person-years of life lost among people aged less than 35 years was, however, greater than the number of person-years of life saved in the older age groups and greater in males than in females.

In Australia, Mathers et al.³⁷ estimated that the harm associated with alcohol consumption accounted for 4.9 per cent of the total burden of disease and injury in 1996 (6.6 per cent of males and 3.1 per cent of females). However, the protective effect of low to moderate consumption was estimated to have averted 2.8 per cent of the total burden (2.4 per cent in males and 3.2 per cent in females). In comparison, tobacco smoking accounted for 9.7 per cent of the total burden; physical inactivity, 6.7 per cent; hypertension, 5.4 per cent; obesity, 4.3 per cent; inadequate fruit and vegetable consumption, 2.7 per cent; and illicit drug use, 1.8 per cent.

Alcohol metabolism: differences between men and women

During the process of absorption, some alcohol is metabolised by gastric alcohol dehydrogenase. This ‘first-pass metabolism’ can be demonstrated by the lower blood alcohol after oral intake than after intravenous infusion. First-pass metabolism is less active in women.⁶ Once absorbed, alcohol is distributed throughout the total body water. This volume (in litres) of distribution in women is 65–70 per cent of that in men because on average women’s weight is 80 per cent of men’s weight and women contain a higher percentage of body weight as fat and so a smaller percentage of water. Thus, after a drink women have a somewhat higher concentration of alcohol in a smaller water volume to be metabolised mostly by the liver, and in women the liver is smaller, so alcohol dehydrogenase capacity is less than in men.

In a substantial minority of adults, two standard drinks within a short time will result in a blood alcohol concentration near or above the legal driving limit of 0.05 grams per 100 millilitres. After it is absorbed, alcohol is distributed throughout the total body water. If the total body water is 40 litres (or kilograms) the concentration after absorption of 10 grams of alcohol will be 10/40 000, or 0.025g/100ml (half the legal driving limit), or somewhat less because of first-pass metabolism; after two standard drinks the concentration will be 0.05g/100ml. However, in a woman with total body water of 34 litres the concentration will be

10/34 000, or 0.029g/100ml, after one drink and 0.058g/100ml after two drinks. On average, people can metabolise 100 milligrams of ethanol per kilogram of body weight per hour (5–8 grams an hour). The rate of metabolism varies about twofold between individuals. Alcohol absorption can be slowed by having a meal; there is no agent that increases the rate of alcohol metabolism.

The equivalent alcohol intake for women is therefore approximately half what it is for men, although men and women vary considerably in size and each individual's ability to metabolise alcohol varies. Women develop cirrhosis of the liver after long-term consumption of smaller amounts of alcohol than men.³⁸

The latest Australian Alcohol Guidelines⁴ have replaced the previous single recommendation for each gender with recommendations for each gender in terms of both maximum average daily intake and maximum for an 'occasion' in recognition of the potential acute harm effects of what is commonly termed *binge drinking*.

Special groups

The foregoing recommendation relates to healthy members of the general population. Within the general population there are, however, people for whom recommendations should be much lower or for whom alcohol consumption should not be recommended at all.

Children and adolescents

The accompanying publication, the *Dietary Guidelines for Children and Adolescents*, explains the scientific basis for the social convention that children should drink no alcohol (or, on occasion, only a very small supervised amount). It also outlines the dangers of alcohol consumption by adolescents, who are more susceptible than adults; that is, they have a lower tolerance than adults to alcohol and relatively small quantities can impair their judgment and control.

Previous alcoholics

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

Women who are pregnant or planning pregnancy

The malformations and mental deficiency associated with foetal alcohol syndrome occur in the offspring of women who drink heavily during pregnancy. The damage is thought to occur during the four to 10 weeks following conception. Some studies indicate that having up to one drink a day³⁹ is not related to the occurrence of foetal alcohol syndrome, but the data are limited and some authorities recommend total abstinence when pregnant or planning a pregnancy, as a precautionary principal.

Lactating mothers

The concentration of alcohol in breastmilk is about the same as that in the plasma of a breastfeeding mother who drinks some alcohol, so a single drink (10 grams of alcohol) would provide only a harmless 40–50 milligrams to the baby. But the babies of heavy drinkers can be affected, and women should not breastfeed if intoxicated. If alcohol is consumed immediately after breastfeeding, the blood alcohol level will be relatively lower by the time the next feed is due, minimising the amount of alcohol in the breastmilk.

Drivers and machine operators

All drivers and operators of dangerous machinery are liable to random breath-testing, and in the event of an accident their blood alcohol concentration will be measured, either directly or via breath-testing. They should drink nothing or very little in the hour before they drive or operate machinery and nothing at all while driving.

People taking certain prescribed or non-prescribed drugs

People taking some analgesics, antidepressants, some antihistamines and some antipsychotics can experience enhanced sedative effects if they drink alcohol. In addition, there are other side effects of drug interactions with alcohol: these are listed in the information about proprietary drugs issued to doctors and pharmacists and in prescribers' manuals. Responsibility for providing information to the individual rests primarily with the prescribing doctor, or with the pharmacist in the case of over-the-counter medicines.

Specific cultural groups

In response to severe problems related to alcohol in many of their communities, Aboriginal and Torres Strait Islander people have introduced initiatives to encourage non-harmful alcohol use, limit access to alcohol, and establish 'dry' areas and communities. These are only a small part of Indigenous Australians' efforts to improve their health status and their social and economic circumstances.

There is limited evidence about the patterns of drinking in migrant communities in Australia, but there are some known physiological constraints on alcohol use in some groups. The 'flushing response'¹⁶ is an inherited characteristic seen most often in people of Asian descent. It involves reduced activity of aldehyde dehydrogenase, so that alcohol is metabolised into acetaldehyde at the usual rate but the acetaldehyde tends to accumulate and cause flushing, headache and nausea. Many people who experience this response choose not to drink alcohol.

PRACTICAL ASPECTS OF THIS GUIDELINE

The Australian Alcohol Guidelines placed patterns of drinking in a number of risk categories based on average daily or weekly consumption limits and on 'occasional day' limits. In the case of usual daily and weekly consumption, the following risk categories were defined:

- Men were considered to be at 'low risk' of long-term harm if their average intake did not exceed four drinks a day (up to 28 a week) and women if their intake did not exceed two drinks a day on average (up to 14 a week).
- An average of five to six drinks a day for men (or 29–42 a week) or three to four a day for women (or 15–28 a week) was considered 'risky' for long-term health.
- An average of seven or more drinks a day for men (or 43 or more a week) and five or more a day for women (or 29 or more a week) was considered 'high risk'.

In terms of short-term harm, an intake on any one day of up to six standard drinks for men or four for women was considered 'low risk'; seven to 10 drinks for men and five to six for women was considered 'risky'; and 11 or more for men and seven or more for women were considered 'high risk'.

For someone to be considered overall at 'low risk' of both short-term and long-term harm, they had to be within both the occasional day limits and the usual weekly limits.

Most dietary guidelines round the world, however, recommend no more than two to three standard drinks a day for men and one to two for women, which is considerably less than the 'low risk' category of the Australian Alcohol Guidelines (see appendix 5 in reference 4). This, in part, reflects concern about nutrient displacement and the energy contributed by alcoholic drinks (see Table 1.8.4) and the current particular concern about overweight and obesity, as well as links to selected chronic disease states.

The *Dietary Guidelines for Americans 2000*⁴⁰ recommends, 'If you drink alcohol beverages, do so in moderation'. *Moderation* is defined as no more than two drinks a day in men and one drink a day in women. As noted, this limit is based on differences between the sexes in both weight and metabolism. In Canada, the 1990 nutrition recommendation is that adults consuming alcohol limit their intake to less than 5 per cent of total energy or two drinks a day, whichever is least. In the United Kingdom⁴¹, a 1990 set of dietary guidelines states, 'Men and women have different metabolisms. It is therefore recommended that men drink less than 21 units a week and women less than 14 (a unit = 8g alcohol, hence 21 units/week = 24g max/day on average). During pregnancy it is best to avoid alcohol completely'. Similarly, the Swedish Medical Research Council (1997), the Singapore Dietary Guidelines (1993), the American Cancer Society (1996) and the American Heart Association (1996) all recommend no more than two standard drinks or 20 grams of alcohol a day.⁴

In Australia, no more than four standard drinks a day for men and two for women has been the general recommendation. This was based on a consideration of the social and medical correlates of alcohol consumption, but the question of dietary energy is also relevant, especially in the context of dietary guidelines. Considering the increasing obesity and overweight in the Australian community and the marginal intakes of micro-nutrients in some groups (such as young women), it would be prudent, in the context of dietary guidelines for optimal health, to recommend a limit of two standard drinks a day for men and one for women. This is also the level at which cardiovascular protection is seen in population studies.

RELATIONSHIP TO OTHER GUIDELINES

Prevent weight gain: be physically active and eat according to your energy needs

Alcohol intake should be moderated as alcohol is energy dense and can contribute to weight gain

CONCLUSION

Because of alcohol's effect on both short-term and long-term health and social outcomes, and because of the additional kilojoules it provides in the diet of a society with increasing rates of obesity, adults—if they drink at all—should limit their average daily intake of alcohol to no more than two standard drinks a day for men and one for women.

EVIDENCE

The National Health and Medical Research Council has recently conducted an extensive review of alcohol consumption in the Australian context. This involved a detailed review of the literature and resulted in publication of the *Australian Alcohol Guidelines*.⁴ This detailed evidence is publicly available and is not repeated here.

This chapter incorporates a summary of the evidence from the NHMRC review in relation to alcohol consumption and chronic disease outcomes. It also discusses Level III evidence in relation to alcohol consumption and blood pressure (reference 10), alcohol limitation and blood lipids (reference 11), alcohol and cancer (reference 12), alcohol and breast cancer (reference 13), alcohol and liver disease (references 14, 26 and 38), and moderate alcohol intake and protection from heart disease and lowering of heart disease risk factors (references 29 to 31).

REFERENCES

1. Editorial. Dietary goals. *Lancet* 1977;1:887–8.
2. Chikritzhs T, Jonas H, Stockwell T, Heale P, Dietze P. Mortality and life years lost due to alcohol: a comparison of acute and chronic cases. *Med J Aust* 174:in press.
3. Collins DJ, Lapsley HM. *The social cost of drug abuse in Australia in 1988 and 1992*. Canberra: AGPS, 1996.
4. National Health and Medical Research Council *Australian Alcohol Guidelines: health risks and benefits*. Canberra: NHMRC, 2001.
5. Higgins K, Cooper-Stanbury M, Williams P. *Statistics on drug use in Australia, 1998*. Canberra: Australian Institute of Health and Welfare, 2000.
6. Frezza M, diPadova C, Pozzato G, Terpin M, Baraona E, Lieber CS. High blood alcohol levels in women: the role of decreased gastric alcohol dehydrogenase activity and first-pass metabolism. *N Engl J Med* 1990;322:95–9.
7. Norton R, Batey R, Dwyer T, MacMahon S. Alcohol consumption and the risk of alcohol related cirrhosis in women. *BMJ* 1987;295:80–2.
8. Transport and Road Research Laboratory. *The facts about drinking and driving*. Crowthorne, Berks: Transport and Road Research Laboratory. Quoted in Royal College of Physicians. *A great and growing evil: the medical consequences of alcohol abuse*, London: Tavistock, 1987:84.
9. Department of Health and Aged Care. *Developing national priorities for alcohol research*. Report of a meeting in Adelaide, May 2001. Canberra: DHAC, 2001.
10. MacMahon SW, Blacket RB, Macdonald GJ, Hall W. Obesity, alcohol consumption and blood pressure in Australian men and women: the National Heart Foundation of Australia Risk Factor Prevalence Study. *J Hypertens* 1984;2:85–91.
11. De Man FH, van der Laarse A, Hopman EG, Gevers Leuven JA, Onkenhout W, Dallinga-Thie GM et al. Dietary counselling effectively improves lipid levels in patients with endogenous hypertriglyceridemia: emphasis on weight reduction and alcohol limitation. *Eur J Clin Nutr* 1999;53:413–18.
12. World Cancer Research Fund. *Food, nutrition and the prevention of cancer: a global perspective*. Washington, DC: American Institute for Cancer Research, 1997:231–4.
13. Smith-Warner SA, Spiegelman D, Yaun SS, van den Brandt PA, Folsom AR, Goldbohm RA et al. Alcohol and breast cancer in women. A pooled analysis of cohort studies. *JAMA* 1998;279:535–40.
14. Sørensen TIA, Orholm M, Bentsen KD, Høybye G, Eghøj K, Christoffersen P. Prospective evaluation of alcohol abuse and alcoholic liver injury in men as predictors of development of cirrhosis. *Lancet* 1984;ii:241–4.

15. Truswell AS. Australian experience with the Wernicke-Korsakoff syndrome. *Addiction* 2000;95:829–32.
16. Harada S, Agarwal DP, Goedde HW. Aldehyde dehydrogenase deficiency as a cause of facial flushing reaction to alcohol in Japanese. *Lancet* 1981;ii:982.
17. Drug interactions: Alcohol. In: *2000 MIMS annual*. St Leonards, NSW: Havas MediMedia, 2000: G43–G44.
18. Colditz GA, Giovannucci E, Rimm EB, Stampfer MJ, Rosner B, Speizer FE et al. Alcohol intake in relation to diet and obesity in women and men. *Am J Clin Nutr* 1991;54:49–55.
19. Bennett N, Dodd T, Flatley J, Freeth S, Bolling K. *Health Survey for England 1993*. London: HMSO, 1995.
20. de Castro JM, Orozco S. Moderate alcohol intake and spontaneous eating patterns of humans: evidence of unregulated supplementation. *Am J Clin Nutr* 1990;52:246–53.
21. Gruchow HW, Sobocinski KA, Barboriak JJ, Scheller BS. Alcohol consumption, nutrient intake and relative body weight among US adults. *Am J Clin Nutr* 1985;42:289–95.
22. Tremblay A, St-Pierre, S. The hyperphagic effect of a high-fat diet and alcohol intake persists after control for energy density. *Am J Clin Nutr* 1996;63:479–82.
23. Suter PM, Jequier E, Shutz Y. Effect of ethanol on energy expenditure. *Am J Physiol* 1994;266:1204–12.
24. Prentice AM. Alcohol and obesity. *Int J Obesity* 1995;19(suppl. 5):S44–S50.
25. Australian Bureau of Statistics. *National Nutrition Survey: nutrient intake and physical measurements, 1995*. Canberra: ABS, 1998.
26. Holman JD'AJ, English DR, Milne E, Winter MG. Meta-analysis of alcohol and all-cause mortality: a validation of NHMRC recommendations. *Med J Aust* 1991;164:141–5.
27. Cullen KJ, Kniuman MW, Ward NJ. Alcohol and mortality in Busselton, Western Australia. *Am J Epidemiol* 1993;137:242–8.
28. Simons LA, Friedlander Y, McCallum J, Simons J. Alcohol intake and survival in the elderly: a 77 month follow up in the Dubbo study. *Aust NZ J Med* 1996;26:662–70.
29. Rimm EB, Giovannucci EL, Willett WC, Colditz GA, Ascherio A, Rosner B et al. Prospective study of alcohol consumption and risk of coronary disease in men. *Lancet* 1991;338:464–8.
30. Rimm EB, Williams P, Fosker K, Criqui M, Stampfer MJ. Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. *BMJ* 1999;319:1523–8.

31. Mikhailidis DP, Jenkins WJ, Barradas MA, Jeremy JY, Daddona P. Platelet function in chronic alcoholism. *BMJ* 1986;293:715–18.
32. Razay G, Heaton KW. Moderate alcohol consumption has been shown previously to improve insulin sensitivity in men. *BMJ* 1997;314:443–4.
33. Conigreve KM, Hu BF, Camargo CA, Stampfer MJ, Willett WC, Rimm EB. A prospective study of drinking patterns in relation to risk of Type II diabetes among men. *Diabetes* 2001;50:2390–5
34. Frankel EN, Waterhouse AL, Teissedere PL. Principal phenolic phytochemicals in selected Californian wines and their antioxidant activity in inhibiting oxidation of human low-density lipoprotein. *J Ag Fd Chem* 1995;43:890–4.
35. Doll R. One for the heart. *BMJ* 1997;315:20–7.
36. Scragg R. A quantification of alcohol-related mortality in New Zealand. *Aust NZ J Med* 1995;25:5–11.
37. Mathers C , Vos T, Stevenson C. *The burden of disease and injury in Australia*. Cat. no. PHE 17. Canberra: Australian Institute of Health and Welfare, 1999.
38. Norton R, Batey R, Dwyer T, MacMahon S. Alcohol consumption and the risk of alcohol related cirrhosis in women. *BMJ* 1987;295:80–2.
39. Sulaiman ND, Florey C du V, Taylor DJ, Ogston SA. Alcohol consumption in Dundee primagravidas and its effect on outcome of pregnancy. *BMJ* 1998;296:1500–3.
40. Department of Agriculture. *Dietary Guidelines for Americans 2000*. 5th edn. Washington, DC: Department of Agriculture, 2000.
41. Ministry of Agriculture, Fisheries and Food. *Advice for healthy eating from HM government: eight guidelines for a healthy diet*. London: MAFF, 1990.

I.9 CONSUME ONLY MODERATE AMOUNTS OF SUGARS AND FOODS CONTAINING ADDED SUGARS

Colin Binns

TERMINOLOGY

Carbohydrates

Carbohydrates are polyhydroxy aldehydes, ketones, alcohols, acids, their simple derivatives, and their polymers having 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 (see Table 1.9.1).

Table 1.9.1 The major dietary carbohydrates¹

Class (DP)	Subgroup	Components
Sugars (1–2)	Monosaccharides	Glucose, galactose, fructose
	Disaccharides	Sucrose, lactose, trehalose
	Polyols	Sorbitol, mannitol
Oligosaccharides (3–9)	Malto-oligosaccharides	Maltodextrins
	Other oligosaccharides	Raffinose, stachyose, fructo-oligosaccharides
Polysaccharides (>9)	Starch	Amylose, amylopectin, modified starches
	Non-starch polysaccharides	Cellulose, hemicellulose, pectins, hydrocolloids

Note: DP = degree of polymerisation.

Sugars

The term *sugars* is conventionally used to describe monosaccharides and disaccharides such as sucrose, glucose and fructose. These can be found naturally in foods or can be added to foods in processing. *Sugars* is the term used in the analysis of the 1995 National Nutrition Survey; *sugar*, by contrast, is used to describe purified sucrose, as are the terms *refined sugar* and *added sugar*, although in some instances partly refined products such as corn syrup may also be regarded as added sugars.

Intrinsic and extrinsic sugars

Intrinsic sugars describes sugars occurring within the cell walls of plants—that is, naturally occurring sugars—while *extrinsic sugars* is used to describe sugars that are usually added to foods. However, the naturally occurring sugar in milk, lactose, is also an extrinsic sugar, so an additional phrase, *non-milk extrinsic sugars* is used in the literature. These terms have not gained wide acceptance, and there are no current plans to measure these sugars separately in the diet or to incorporate such data in food tables.¹ The terms *refined*, *added* and *extrinsic* sugars are sometimes used to denote sucrose and glucose used in the food industry and in the home.

Physiologically, there is no difference between the sugars that occur naturally in food and the refined sugars that are added to the diet. Among foods rich in added sugars are confectionery, cakes, pastries, biscuits, fruit drinks, cordials and carbonated soft drinks. Foods with high added-sugar content often have a lower nutrient content but are energy dense. The term *no added sugar* means no sugars have been added during the manufacturing process; it does not mean that no sugar is present, since most foods contain sugars in some form.

BACKGROUND

Many of the foods found in the Australian diet contain naturally occurring sugars. In other foods, sugars (particularly sucrose) may be added during processing, to increase the food's palatability and acceptability and sometimes to add bulk. Sugars provide a readily absorbed source of energy and have an important role as sweeteners and flavour enhancers. The presence of high amounts of sugar can, however, dilute the nutrient density of the diet, and diets high in added sugar have been associated with development of obesity and dental caries.

Because sugars are a significant source of energy in the Australian diet, all previous sets of dietary guidelines—for adults, children and adolescents, and older Australians—have included a guideline on sugar or sugars. Over the years the emphasis has changed, from a guideline aimed at reducing the amount eaten to one that emphasises care and moderation in the amount consumed. This reflects changing scientific knowledge and the relative stability of sugar consumption in Australia. The US year 2000 dietary guidelines include in the section on eating sensibly the guideline 'Choose beverages and foods that limit your intake of sugars'.² A review by Sheiham lists 23 countries that currently make dietary recommendations on sugars.³ The pros and cons of retaining an Australian dietary guideline on sugar have recently been debated in the literature by Stanton⁴, O'Dea and Mann⁵, and Williams.⁶

Sugars in the Australian diet

Australian adults derive about 45 per cent of their energy from carbohydrates and about half of this comes from sugars. The results of the 1995 National Nutrition Survey show that the percentage of energy from total sugars intake declines from age 2–3 years to age 45–64 years; this is followed by a slight increase in intake in the 65 years and over age group (see Table 1.9.2).^{7,8} The percentage of energy from added sugars remains at a similar level from age 2–3 years to age 19–24 years but falls from age 25 years. Natural sugars as a percentage of energy were highest in very young children and adults over 65 years and lowest in the 19–44 year age group.

Table 1.9.2 Carbohydrates: mean % contribution to energy intake, by age, 1995⁸

	Age (years)								
	2–3	4–7	8–11	12–15	16–18	19–24	25–44	45–64	65+
	%	%	%	%	%	%	%	%	%
Carbohydrate	52	52	50	52	49	46	45	44	45
Total sugars	29	28	25	26	25	22	19	19	21
Added sugars	14	15	15	15	15	13	10	9	9
Natural sugars	16	13	10	10	10	9	9	11	12

Figure 1.9.1 shows the amounts (in grams per day) of sugars consumed, as recorded in the 1995 National Nutrition Survey.

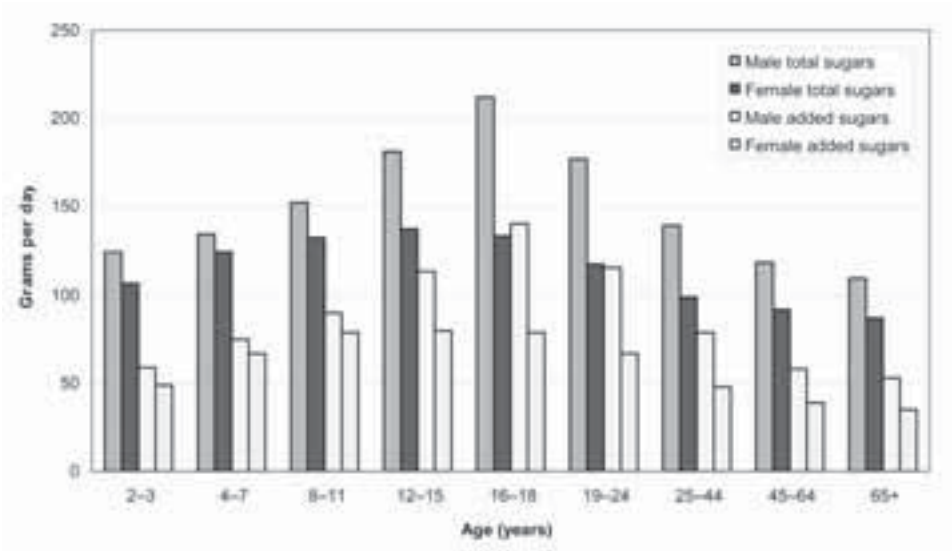


Figure 1.9.1 Consumption of sugars, by age: 1995 National Nutrition Survey⁸

Figure 1.9.2 shows the amount of sugars in the diet as a percentage of energy. Because of the higher energy consumption of adolescents and young adults, when consumption of sugars is expressed as a percentage of energy the peaks are much lower than for Figure 1.9.1. Male adolescents are the highest consumers of added sugars, both in absolute amounts and as a percentage of energy.

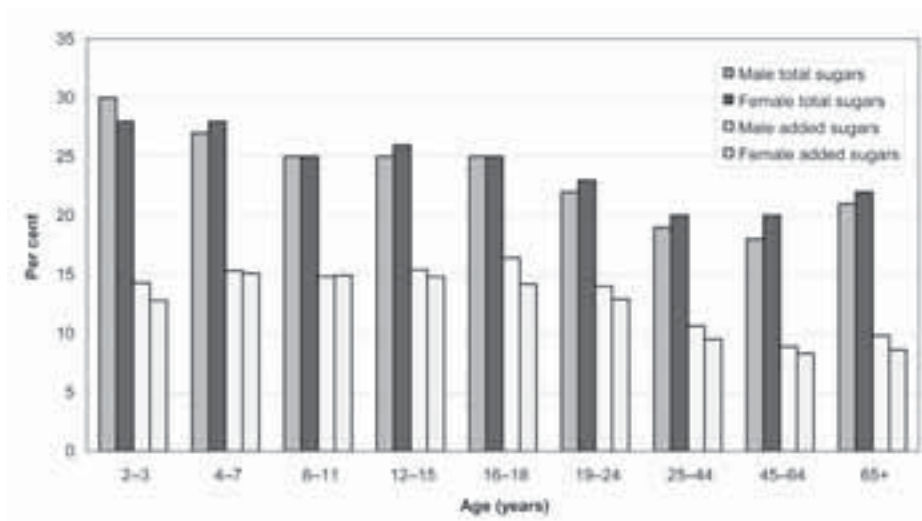


Figure 1.9.2 Consumption of total and added sugars as a percentage of energy, by age: 1995 National Nutrition Survey⁸

Figure 1.9.3 shows the sources of added sugars in the Australian diet, as recorded in the 1995 National Nutrition Surveys, which used a 24-hour recall method.

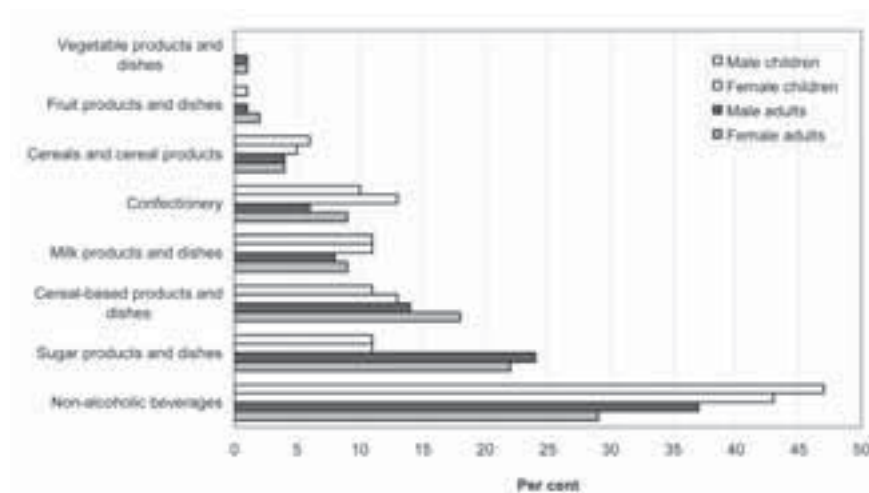


Figure 1.9.3 Sources of added sugars, by age: 1995 National Nutrition Survey⁸

Note: Adults = people aged 19 years and over; children = people aged 2 to 18 years.

The most recent information on sugars (sucrose only) consumption comes from the Australian Bureau of Statistics *Apparent Consumption of Foodstuffs, 1997–98 and 1998–99*.⁹ Apparent consumption has fallen by about 15 per cent from pre-war levels, or by 23 per cent from the post–World War 2 peak reached in 1948 (see Table 1.9.3). The apparent consumption data do not represent actual consumption by individuals or population groups—some sugar is wasted and some is used for brewing and other purposes—but they do give an indication of trends. Baghurst et al.¹⁰ discuss the differences between apparent consumption of sugar and actual dietary consumption.

Table 1.9.3 Apparent annual consumption of sugar (kg/person/yr) 1938–39 to 1998–99⁹

Sugar	1938–39 kg	1948–49 kg	1958–59 kg	1968–69 kg	1978–79 kg	1988–89 kg	1998–99 kg
Cane sugar as refined sugar	32.0	31.2	27.0	21.0	14.9	8.8	n.a.
Cane sugar in manufactured foods	16.3	23.1	23.6	27.7	34.6	33.9	n.a.
Total cane sugar	48.3	54.3	50.6	48.7	49.5	42.7	37.6
Total sugars	50.8	56.8	53.0	51.9	54.5	48.3	43.4

n.a. Not available.

Apparent consumption data show that consumption of honey, which is a solution of sugars, has also declined in recent years and is now 0.5 kg per capita.⁹

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

In remote Aboriginal communities, apparent consumption of sugar is much higher than the Australian average, as shown in the Table 1.9.4. Lee et al.'s study¹¹ shows that sugar consumption is high, whereas fruit and vegetable consumption is well below the Australian average. In the communities where apparent consumption was measured, refined sugars contributed approximately 30 per cent of total energy intake. Sixty per cent of the apparent high intake of sugars was derived from white sugar per se, which is in marked contrast to recent figures for the wider Australian community. No data are available for urban Indigenous communities.

A review of the nutrition of Indigenous Australians has suggested that a reduction in sugar consumption in this group would be an important strategy to improve their health and nutritional status.¹³

Table 1.9.4 Apparent mean consumption of selected foods in Aboriginal communities compared with national data (kg/head/yr)^{11,12}

Food	Aboriginal communities		Australia
	Central Desert (n=3)	Northern coastal (n=3)	
Flour (white)	37.6	44.4	n.a.
Bread (all)	34.1	30.5	45.5
Beef and veal	51.6	25.8	41.4
Poultry	22.3	19.7	23.0
Lamb	22.8	3.3	16.8
Fish	–	4.8	4.0
Fruits	33.2	17.6	106.9
Vegetables	24.3	19.6	136.2
Sugar (refined)	54.1	50.3	8.2
Carbonated beverages	67.9	224.6	73.0
Fruit juice	48.3	12.8	n.a.

– Zero.

n.a. Not available.

Note: 'Bread' includes flour used in bread-making.

Intake of sugars in relation to the total diet

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

A number of authors have suggested that high consumption of extrinsic sugar is associated with high intakes of dietary fat.¹⁴ Studies from Europe and Australia suggest, however, that—although it is possible to identify some foods rich in both fat and sugars—in the context of the whole diet, foods that are the primary sources of sugars are only minor sources of fat and vice versa.¹⁵ Studies of the relationship between a low-fat diet and refined sugar intake often show an inverse relationship.^{15–19} One study, of 3290 people living in Victoria and South Australia, found that respondents who had the lowest relative intake of fat had high intakes of simple sugars, both natural and refined.²⁰ In the 1995 National Nutrition Survey publications, only information on total sugars is available and this does not show a consistent relationship between sugars and fat intake.⁷ A further analysis of the survey data⁸ showed that those adults in the highest tertile of percentage of energy from total sugars had a significantly lower percentage of fat in their diet, but there was no difference in the percentage of fat intake between the lowest, middle and highest tertiles of percentage of energy from added sugars.

Some studies, particularly the larger studies from the United States, suggest that high intakes of sugar are linked to diet quality. Using food intake data from a representative sample of 15 011 people, Naismith et al.¹⁸ divided the sample into quartiles based on added sugar consumption. Many high consumers of sugar also overconsumed total energy. The intake of fruit in this group was lower than in other groups with similar energy intakes. The 41 per cent of sugar overconsumers who did not consume excessive energy compensated for the additional energy by reducing their intakes of other foods, including the fruit, vegetable, milk and grains groups. Similarly, an analysis of the third National Health and Nutrition Examination Survey (n=15, 611, aged 20 years or over) showed that energy-dense, nutrient-poor foods tended to be consumed at the expense of foods that are nutrient dense. The former group included foods high in fat and/or sugar, such as soft drinks, confectionery, biscuits, cakes, desserts, pastries and processed savoury snacks. A recent review by Williams⁶, summarising a number of studies from the United States and the United Kingdom, noted that in most cases energy and nutrient intakes were positively related to total sugar intake. At any given level of energy intake, however, as the proportion of sugars in the diet is increased the nutrient density will fall.²¹

The report of the UK Committee on Medical Aspects of Food concluded:

- on average people with high total energy intakes eat more of all nutrients including sugar
- sugar intake is a weaker predictor of absolute micronutrient intake than total energy consumption.

The association between high refined-sugar intake and low micro-nutrient intake was investigated by re-examining data from three large-scale Australian population surveys of dietary intake²² and from the CSIRO sugars analysis of the 1995 National Nutrition Survey. The results of this review did not show a consistent relationship between refined sugar consumption and micro-nutrient intake. A study of older South Africans²³ showed that as sugar intake increased there was a significant decrease in the proportion of energy derived from fat but there was also nutrient dilution. (It should be noted, however, that this study was in older people: its relevance to younger adults and children and adolescents is not clear.) Results from several other studies vary depending on the classification of different sugars used, but in general moderate sugar consumers appear to have the most adequate diet.^{24,25,26}

The 1995 National Nutrition Survey shows that an increasing proportion of energy is obtained from meals and snacks eaten outside the home.⁷ Summerbell et al.²⁷ found that 25 per cent of adolescents' and 20 per cent of adults' daily energy intake was in the form of snacks and that the proportion of energy derived from total sugars in snacks was greater than that in meals. Most often this sugar was provided by plain biscuits and milk and sugar added to cups of tea and coffee.

If obesity is to be avoided in a predominantly sedentary society such as Australia, not only should greater everyday activity be encouraged: foods that are less energy-nutrient dense should also predominate in the diet.

In summary, then, it is likely that the results of surveys reported as averages of group consumption obscure the effects of consumers who are in the upper percentiles of sugar intake. It is important that care is taken with snack foods that are high in added sugars, since foods that are high in refined sugars (for example, soft drinks and confectionery) are energy dense but do not provide vital nutrients. Foods such as cakes, biscuits and confectionery are high in both sugar and fat and also energy dense; they provide few nutrients and are often eaten instead of more nutritious, necessary foods by both children and adults.

SCIENTIFIC BASIS

Carbohydrates constitute the largest source of energy in the diets of most people—on average around 45 per cent of the energy in the Australian diet. Dietary carbohydrates are usually associated in foods with important micro-nutrients and phytochemicals. Diets high in carbohydrate are not associated with the development of obesity independently of energy intake.²⁸ People whose diets are high in carbohydrates usually have a lower prevalence of obesity, heart disease, type 2 diabetes, and some forms of cancer.²⁹

Among carbohydrates' physiological functions are the following:

- provision of energy
- effects on satiety and gastric emptying
- effects on blood glucose and insulin metabolism
- protein glycosylation
- bile acid dehydroxylation
- fermentation—production of hydrogen and methane
- production of short-chain fatty acids
- control of colonic epithelial cell function
- bowel habit, laxation and motor activity
- effects on large bowel microflora.²⁸

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

Obesity

The 1995 National Nutrition Survey results demonstrated that obesity is an increasing problem for all age groups in Australia.⁷ The World Health Organization describes this epidemic as part of an 'escalating epidemic of overweight and obesity that is affecting many countries in the world' and notes, 'The principal causes of the accelerating obesity problem worldwide are sedentary lifestyles and high-fat, energy-dense diets' <<http://www.who.int/nut/obs.htm>>.

The links between sugar intake and obesity are not clear: many studies show no links but others suggest there may be cause for concern. In Australia, obesity has been increasing during the past two decades. Lowered physical activity may, of course, be involved in the overall picture, but changes in diet may also be playing a role (see Chapter 2). The national dietary surveys of 1983 to 1985 and 1995 showed a small increase in energy intake in adults (3–4 per cent) and a larger increase in children aged 10–15 years (11–15 per cent). Carbohydrates were the macro-nutrient whose consumption increased most over this period (see Table 1.2).

It has been suggested that excess consumption of sugar contributes to an energy-dense diet that may lead to energy imbalance and obesity. In the CARMEN study³⁰, a randomised controlled trial of diets and weight reduction, subjects were randomly placed in groups with diets that included reduced fat and high simple carbohydrate and reduced fat and high complex carbohydrate. The study found that a reduction in fat intake resulted in a modest, but significant, weight loss. Whether the carbohydrate was in simple or complex form made no difference to weight outcomes or to the subjects' lipid profiles. The lack of a significant difference between complex and simple carbohydrates may, however, have been a consequence of insufficient sample size.⁴

It is nevertheless important to stress that excess energy in any form will promote the accumulation of excess body fat and that high-carbohydrate diets should be promoted only in accordance with an individual's energy needs.²⁸

In a two-year longitudinal study of 548 ethnically diverse school children in the United States (mean age 11.7 years), Ludwig et al.³¹ found that an increase in consumption of sweetened soft drink was linked to increasing body mass index and risk of obesity. On the other hand, a number of studies have concluded that intake of carbohydrate or even sucrose has no association with obesity or that the association may even be negative.³² The Ludwig study used a standardised food frequency questionnaire to obtain dietary data. It was undertaken in adolescents and similar studies have not yet been reported in adults. It does, however, suggest that there may be cause for concern, and in such an important and growing area of concern it is essential that additional studies be done to assess the results' relevance to the Australian situation—for both children and adults.

In some studies, children and adults who ingest large amounts of carbohydrate or sucrose, or both, have been reported to be leaner than their peers. But the study participants' high carbohydrate intake might reflect higher levels of physical activity. Another reason for the lack of relation between carbohydrate intake and adiposity, as just noted, might be inaccuracy in assessing intake and energy expenditure using traditional dietary methods. The 'doubly-labelled water method' used for measuring energy expenditure in free-living individuals has recently cast doubt on the validity of self-reported food intake for adults³³, although dietary data do appear to be more valid for children. Yet another reason could be that very active children need and ingest more sugar.³³

If the study by Ludwig et al.³¹ is generalisable to the Australian situation, it suggests that the consumption of sugar-sweetened beverages could be an independent risk factor for development of obesity in children. This may be related to the reduced effect on satiety of sugar in a liquid medium. A meta-analysis has shown that compensation for energy in liquid form is less complete than that for energy in solid form.³⁴ In this context it is of interest to note that the contribution of fluids (sweetened soft drinks and fruit juices and drinks) to sugar intake increased in Australian adults between 1983 and 1995 relative to non-fluid sources, particularly sugar products and fruit products (see Table 1.9.5). The increase mirrors the situation in the United States and elsewhere.^{21,35,36} This is against a background of little change in intake of total sugars over the period (see Table 1.9.6).

The increasing consumption of sweetened drinks (fruit juices and soft drinks) as a component of increasing energy consumption (see Chapter 1) suggests that moderation in the consumption of these products is advisable.

Table 1.9.5 Relative intake of sugars from various food groups: adults, 1983 and 1995^{8,37}

Food group	Males %		Females %	
	1983	1995	1983	1995
Confectionery	4	3	5	5
Sugar products and dishes	21	14	18	11
Vegetable products	5	4	5	5
Milk products	15	16	16	17
Fruit products	17	12	22	17
Cereals and cereal products	16	16	16	17
Non-alcoholic beverages (total)	13	26	12	20
Fruit and vegetable juices and drinks	6	10	6	10
Sweetened soft drinks	6	16	5	9

Table 1.9.6 Percentage of energy from total carbohydrates and total sugars: adults, 1983 and 1995^{8,37}

Energy	Age 25–44 years %		Age 45–64 years %	
	1983	1995	1983	1995
Total carbohydrates	41	45	41	44
Total sugars	20	19	19	19

In the Ludwig et al.³¹ study, the consumption of diet soft drinks was inversely associated with becoming obese. In Australia, consumption of beverages, most of which are sweetened with sugars, is increasing. Carbonated and aerated beverages have become the most popular beverages, and consumption of these beverages has continued to increase—from the late 1980s figure of 87.4 litres per person per year to 113.0 litres in 1998–99. This is an increase of 30 per cent in a decade and an increase of 3.7 per cent in the year prior to 1998–99.⁹

Figure 1.9.4 shows the trend in soft drink consumption. By the end of 2000, 19.3 per cent of soft drinks consumed in Australia were sweetened with non-nutritive sweeteners.

Sugar-sweetened drinks make up the major portion of the soft drink market. The 1995 National Nutrition Survey results showed that consumption of soft drinks in the 16–18 year and 19–24 year age groups was just over 400 grams a day (see Figure 1.9.5). About 7 per cent of the reported intake was of non-sugar containing drinks.

Dental caries

Historically, the prevalence of dental caries has increased when the diet has changed to include more sugars and other refined foods. Dental caries is a disease of both children and adults, but much of the research has centred on childhood. The relationship between sugar (sucrose) and dental caries was first documented in the scientific literature by Miller in 1883, and it has been confirmed in numerous studies since then.³⁹ Dental caries remains a significant public health problem in Australia: it is estimated to be the most expensive diet-related health problem.^{40,41} There have, however, been dramatic declines in average levels of dental decay, as defined by the number of decayed, missing and filled teeth. In 12-year-old children, scores for this criterion fell from

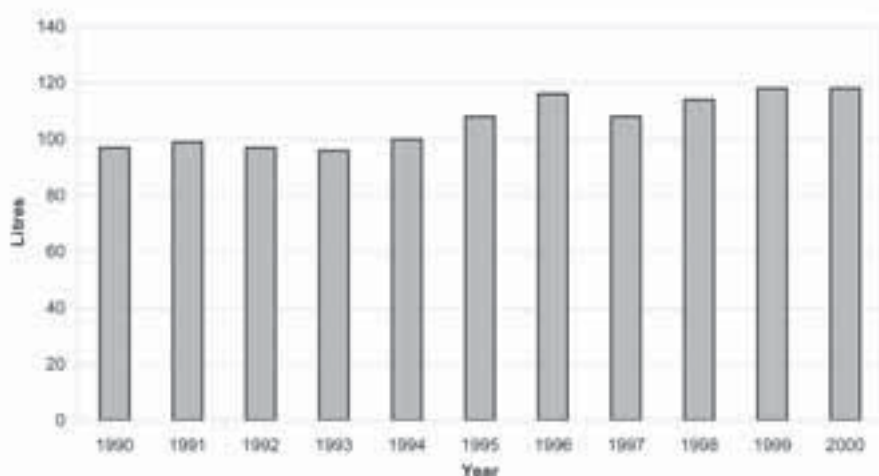


Figure 1.9.4 Per capita consumption of soft drinks, 1990 to 2000³⁸

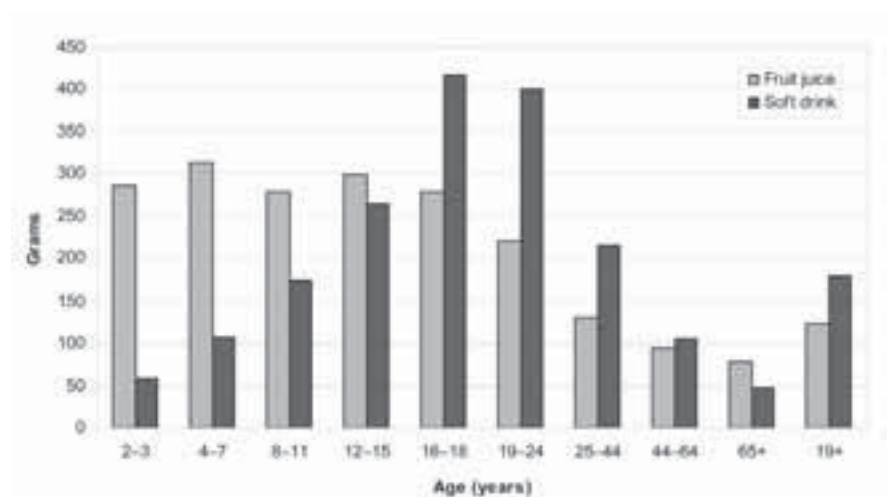


Figure 1.9.5 Average daily consumption of fruit juice and soft drink: 1995 National Nutrition Survey⁷

approximately 8 in 1965 to 1.01 in 1995. These improvements are obviously the starting point for improvements in oral health in later life, but even in adults the average number of missing teeth has fallen from 8.3 in 1973 to only 3.6 in 1995.⁴² The role of fluoridation in prevention of dental caries has been documented.^{43,44}

This improvement in the dental health of children does not, however, extend to Australian Aboriginal children or to Australian children from the lowest socio-economic groups. Historically, Aboriginal Australians have had substantially less dental caries than non-Indigenous people, but more recently this trend appears to have been reversed: the oral health of non-Indigenous children has improved and that of Aboriginal children has deteriorated.⁴⁵

Dental caries can be defined as a dietary carbohydrate and saliva-modified infectious disease. Its key microbiological feature is a dietary carbohydrate enrichment of the dental plaque microbia with bacteria such as *Streptococcus sobrinus* and *S. mutans*, which increase the acid-producing potential of dental plaque.⁴⁶ Development of the disease is a dynamic process involving the metabolism of a carbohydrate substrate by oral bacteria to produce acid, with saliva and host resistance offering protective elements.⁴⁷ *Streptococcus mutans* can ferment sugars to lactic acid. Dietary sugars other than sucrose—for example, glucose and lactose—can also induce caries formation, 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. Starch is less cariogenic than other dietary sugars because it does not readily diffuse into plaque and is less readily hydrolysed. *Streptococcus mutans*, dietary sugars and a susceptible tooth surface are the important factors in dental caries. If there is frequent exposure to sugars, the rate of demineralisation of the tooth will exceed the rate of remineralisation and dental caries will occur.⁴⁸ The

duration of exposure depends on the extent of retention of sugary foods in the mouth and the number of eating occasions; it can be difficult to describe and quantify.⁴⁷

Comparisons of international data indicate that low sugar consumption does not necessarily translate into less dental caries or that higher consumption inevitably leads to more.⁴⁷ The relationship between the quantity and frequency of sugar consumption approximates a sigmoid curve. Thus, as the consumption of sucrose increases, dental caries incidence rises ever more steeply until the curve flattens out and the increase in dental caries is small with further increases in sucrose intake.⁴⁹ A WHO study group⁴⁹ noted that very little caries occurs in children when the national per capita sugar (sucrose) consumption is below 10 kilograms a year (about 30 grams a day) but that a steep increase may occur from 15 kilograms upwards. Studies have also shown that it is the frequency of eating sugar, rather than the amount of sugar per se, that is related to dental caries.⁵⁰ The sugars contained in the cellular structure of foods (such as the intrinsic sugars of fresh fruits and vegetables) have been found to have little cariogenic potential; it is foods high in extrinsic sugars that are most damaging to the teeth.⁵¹ Petti et al. (cited in reference 47) found that good oral hygiene was three times more likely to predict low caries prevalence than a 'low cariogenic' diet. The principal diet and health association given for the retention of a sugar guideline in the US dietary guidelines continues to be dental caries.² In severe cases, dental caries can cause loss of teeth and pain that may reduce dietary intake and compromise nutritional status.

On the basis of the scientific evidence, advice on sugar intake for the prevention of dental caries should include advice on the frequency of intakes, not just the amount. The FAO report¹ summarises the evidence:

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

The British Nutrition Foundation's report concludes, 'The evidence establishing sugars as an aetiological factor in dental caries is overwhelming. The foundation of this lies in the multiplicity of studies rather than the power of any one'.⁵¹ While infants and young children are at risk of dental caries, as people age the risk increases again.²

Carbohydrates and the prevention of dental caries

Both xylitol and sorbitol have been shown to have a preventive effect on dental caries. Daily consumption of xylitol (5–10 grams a day) added to chewing gum and confectionery foods has been shown to prevent dental caries in children.⁵² Highly acidogenic snack foods should be consumed only at meal times to reduce the risk, and between-meal snacks should be either non-acidogenic (such as xylitol products) or hypo-acidogenic (such as sorbitol and HSH products). Cheeses are a natural product that may provide anti-cariogenic effects²⁴ and can provide an alternative to high-sugar snacks. A systematic review of published double-blind comparative trials showed that xylitol-containing chewing gums may provide superior efficacy in reducing caries rates in high-risk populations.⁵³ One mechanism of the action of xylitol-containing gums is the stimulation of salivary flow.⁴⁸

Type 2 diabetes

The rapid cultural change experienced by many populations that previously consumed a traditional diet and the high incidence of centrally distributed abdominal obesity in these populations have coincided with high rates of type 2 diabetes. Some populations appear to have a stronger predisposition to the development of type 2 diabetes than others, suggesting the involvement of genetic factors. Family history, diet, and lifestyle conditions that are conducive to obesity will influence the risk of developing diabetes. Development of type 2 diabetes does not appear to be related to ingestion of sugar or other carbohydrates: it is predominantly influenced by genetics, body weight and lifestyle factors. Avoiding obesity and increasing intakes of a wide range of foods that are rich in non-starch polysaccharides and carbohydrates with a low glycaemic index offers the best means of reducing the rapidly increasing rates of type 2 diabetes in many countries.²⁸ Dietary fibre may also have a beneficial effect on insulin metabolism.⁵⁴

Cardiovascular disease

Body mass index, abdominal obesity, hyperlipidaemia, homocysteinaemia, and genetic and lifestyle factors are all important in the aetiology of coronary heart disease. Early studies suggested that a reduction in dietary sucrose could lower elevated triglyceride levels, but it is likely that the effects seen were the result of a reduction in energy intake and body weight.^{25,55} Metabolic studies of lean and obese volunteers have shown that solid-food diets that are very low in fat and high in simple sugars markedly stimulate fatty acid synthesis from carbohydrate and that plasma triglycerides increase in proportion to the amount of fatty acid synthesis.²⁶

There is some evidence that antioxidants confer protection against the development of cardiovascular disease. Fruits and vegetables, which are sources of sugars and carbohydrates, are rich in antioxidants, and increasing the amount

of these foods in the diet can assist in the reduction of saturated fat, which will provide further protection against cardiovascular disease. There is no evidence of a causal role for sugar in the development of cardiovascular disease. Ensuring that the diet contains adequate amounts of fruit, vegetables and carbohydrate-rich foods—at the expense of fat—and maintaining a healthy body weight are the basis of dietary advice aimed at reducing the risk of cardiovascular disease.²⁸ When the content of dietary carbohydrate is elevated above the usual level in our diets (more than 55 per cent of energy), blood concentrations of triglycerides rise.⁵⁶ There is, however, a concurrent reduction in LDL cholesterol concentration, which makes it difficult to predict whether negative health consequences will result.⁵⁶

Cancer

In a case-control study of gastric cancer (382 cases and 561 controls), higher intakes of sugar were found to decrease the likelihood of developing this cancer.⁵⁷ Other studies have suggested a link between sugar consumption and colorectal cancer.^{58–60} The World Cancer Research Fund reviewed the eight case-control studies available to it and concluded that there was a correlation between sugar intake and colorectal cancer.⁶¹ In contrast, when the National Health and Medical Research Council reviewed the risk factors for colorectal cancer, sugar was not included as a significant factor.⁶² The FAO–WHO Expert Consultation concluded, ‘There is little evidence of any significant correlation between intake of mono-, di- and oligosaccharides and cancer at any site that could not be explained by total energy intake’.¹ Although it is widely recognised that diet influences the development of cancer, a consistent role for sugar has not been identified. Fruit, vegetables and cereal foods are considered to be protective against some forms of cancer, including colorectal cancer.⁶²

Attention deficit/hyperactivity disorder

Attention deficit/hyperactivity disorder is the most common neuro-behavioural disorder in children and among the most prevalent chronic conditions in school-aged children. There is no evidence that sugars or sugar-containing foods are involved in the aetiology of attention deficit/hyperactivity disorder.⁶³

Summary

The evidence for sugar’s role in the aetiology of dental caries is strong. When energy intake exceeds energy expenditure over a sustained period, overweight or obesity will result. Excess dietary energy intake—from whatever source, including sugars—can thus contribute to weight gain, overweight and obesity. Inappropriately high levels of intake of sugars may also displace other nutrients from the diet. No other links to the causation of specific disease have been identified.

On the other hand, moderate use of sugars as sweeteners or to add flavour may actually improve the palatability of food and increase overall nutrient consumption. There is no evidence that, for most Australians, consumption of up to 15–20 per cent of energy as sugars is incompatible with a healthy diet. Consumption of greater amounts than this could lead to a decrease in nutrient density. A diet without any sugar would be impractical, hence this guideline: ‘Consume only moderate amounts of sugars and foods containing added sugars’.

PRACTICAL ASPECTS OF THIS GUIDELINE

Adding a small amount of refined sugar can increase the palatability of some highly nutritious foods and increase the overall nutrient intake. For example, adding a small amount of sugar or honey to porridge and spreading jam on bread or toast can greatly improve the taste and acceptability of these high-carbohydrate, nutrient-dense foods. But adding stewed fruit to porridge would offer equal palatability, with less sugar. A growing number of non-nutritive sweeteners are available and promoted as substitutes for sugars. Although there are still problems with heat stability, use of non-nutritive sweeteners in carbonated beverages could reduce the energy load in the highest-consuming age groups. Some studies on obesity control have shown mixed results, but at least one study of the use of an artificial sweetener has shown a long-term benefit for weight control.⁶⁴

RELATIONSHIP TO OTHER GUIDELINES

Enjoy a wide variety of nutritious foods

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

Eat plenty of vegetables, legumes and fruits

Sugars are an important constituent of vegetables and fruit, contributing to their palatability. Adding a small amount of sugar to stewed fruits and some cooked vegetables can increase their palatability.

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

Cereals, breads and pasta are an excellent source of energy and nutrients. Adding small amounts of sugar to cereals and breads can greatly increase their palatability.

Drink plenty of water

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

Prevent weight gain: be physically active and eat according to your energy needs

Consumption of excessive energy from any source, including sugar, will lead to obesity. For some, consumption of excessive amounts of sugar in a liquid medium may be a problem that leads to obesity.

CONCLUSION

The amount of sugar added to the diet of Australians should be moderate, to ensure that valuable nutrients are not diluted by foods high in added sugar and limited in nutrient density. On the other hand, adding small amounts of sugar to foods that are energy and nutrient dense can increase the palatability of these foods and promote their intake.

EVIDENCE

Much of the evidence presented in this guideline relates to dispelling commonly held beliefs about sugar and disease. As a result, much of it is negative in that it provides evidence against a hypothesised relationship. There is Level I evidence of dental caries prevention with xylitol (reference 53) and Level III evidence for the role of carbohydrates in dental caries (references 39 and 50) and for a link between consumption of sugar-sweetened drinks and childhood obesity (reference 31).

In contrast, a number of studies have concluded that intake of carbohydrate, or even sucrose, has no relationship with obesity or that the relationship may be negative (see reference 32). Other evidence concerning sugar and dental caries comes from cross-population studies and observational studies within populations.

REFERENCES

1. UN Food and Agriculture Organization. *Carbohydrates in human nutrition*. FAO Food and Nutrition Paper no. 66. Rome: FAO, 1997.
2. Johnson C, Kennedy E. The 2000 Dietary Guidelines for Americans: What are the changes and why were they made? *J Am Diet Assoc* 2000;100:769–74.

3. Sheiham A. Dietary effects on dental diseases. *Publ Hlth Nutr* 2001;4(2B):569–91.
4. Stanton R. Sugar: why Australia should retain a dietary guideline. *Aust J Nutr Diet* 2001;58:31–6.
5. O'Dea K, Mann J. Importance of retaining a national dietary guideline for sugar. *Med J Aust* 2001;175:165–6.
6. Williams P. Sugar: is there a need for a dietary guideline in Australia? *Aust J Nutr Diet* 2001;58:26–31.
7. Australian Bureau of Statistics. *National Nutrition Survey: selected highlights, Australia, 1995*. Canberra: ABS, 1997.
8. Cobiac L, Record S, Leppard P, Syrette J, Flight I. *Sugars in the Australian diet: results from the 1995–96 National Nutrition Survey*. Adelaide: CSIRO, 2001.
9. Australian Bureau of Statistics. *Apparent consumption of foodstuffs, 1997–98 and 1998–99, Australia*. Canberra: ABS, 2000.
10. Baghurst K, Record S, Syrette J, Crawford D, Baghurst P. Intakes and sources of a range of dietary sugars in various Australian populations. *Med J Aust* 1989;151:515–18.
11. Lee A, O'Dea K, Mathews J. Apparent dietary intake in remote Aboriginal communities. *Aust J Publ Hlth* 1994;18:190–7.
12. Australian Bureau of Statistics. *Apparent consumption of foodstuffs, Australia*. Canberra: ABS, 1987.
13. National Health and Medical Research Council. *Nutrition in Aboriginal and Torres Strait Islander peoples: an information paper*. Canberra: NHMRC, 2000.
14. Emmett P, Heaton K. Is extrinsic sugar a vehicle for dietary fat? *Lancet* 1995;345:1537–40.
15. Gibney M, Sigman-Grant M, Stanton J, Keast D. Consumption of sugars. *Am J Clin Nutr* 1995;62(suppl.):178S–194S.
16. Bolton-Smith C, Woodward M. Dietary composition and fat to sugar ratios in relation to obesity. *Int J Obes* 1994;18:820–8.
17. Lewis C, Youngmee K, Dexter P, Yetley E. Nutrient intakes and body weights of persons consuming high and moderate levels of added sugars. *J Am Diet Assoc* 1992;92:708–13.
18. Naismith D, Nelson M, Burley, Gatenby S. Does a high-sugar diet promote overweight in children and lead to nutrient deficiencies? *J Hum Nutr Diet* 1995;8:249–54.
19. Flynn M, Sugrue D, Codd M, Gibney M. Women's dietary fat and sugar intakes: implications for food based guidelines. *Eur J Clin Nutr* 1996;50:713–19.

20. Baghurst K, Baghurst P, Record S. Demographic and dietary profiles of high and low fat consumers in Australia. *J Epidemiol Comm Health* 1994;48:26–32.
21. Krebs-Smith S. Choose beverages and foods to moderate your intake of sugars: measurement requires quantification. *J Nutr* 2001;131(suppl.):527S–535S.
22. Baghurst KI, Baghurst PA, Record S. Nutritional status of high consumers of refined sugars *Nutr Res* 1992; 12: 1455–1465
23. Charlton K, Wolmarans P, Lombard C. Evidence of nutrient dilution with high sugar intakes of older South Africans. *J Hum Nutr Diet* 1998;11:331–43.
24. Jensen ME. Diet and dental caries. *Dent Clin North Am* 1999;43(4):615–33.
25. Mann J, Truswell A. Effects of isocaloric exchange of dietary sucrose and starch on fasting serum lipids, postprandial insulin secretion and alimentary lipidaemia in human subjects. *Br J Nutr* 1972;27:395–405.
26. Hudgins L, Hellerstein M, Seidman C, Neese R, Tremaroli J, Hirsch J. Relationship between carbohydrate-induced hypertriglyceridemia and fatty acid synthesis in lean and obese subjects. *J Lipid Res* 2000;41:595–604.
27. Summerbell C, Moody R, Shanks J, Stock M, Geissler C. Sources of energy from meals versus snacks in 220 people in four age groups. *Eur J Clin Nutr* 1995;49:33–41.
28. UN Food and Agriculture Organization. *Carbohydrates in human nutrition: report of a joint FAO–WHO expert consultation*. Rome: FAO, 1997.
29. Mann J, Truswell A, eds. *Essentials of human nutrition*. Oxford: Oxford University Press, 1988.
30. Saris WH, Astrup A, Prentice AM, Zunft HJ, Formiguera X, Verboeket-van de Venne WP et al. Randomized controlled trial of changes in dietary carbohydrate/fat ratio and simple vs complex carbohydrates on body weight and blood lipids: the CARMEN study. The carbohydrate ratio management in European national diets. *Int J Obes Rel Metab Dis* 2000;24(10):1310–18.
31. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 2001;357:505–8.
32. Hill J, Prentice A. Sugar and body weight regulation. *Am J Clin Nutr* 1995;62(suppl. 1):264S–273S.
33. Bellisle F, Rolland-Cachera M. How sugar-containing drinks might increase adiposity in children. *Lancet* 2001;357:490–1.
34. Mattes R. Dietary compensation by humans for supplemental energy provided as ethanol or carbohydrate in fluids. *Physiol Behav* 1996;59:179–87.

35. Johnson R, Frary C. Choose beverages and foods to moderate your intake of sugars: the 2000 Dietary Guidelines for Americans—what's all the fuss about? *J Nutr* 2001;131(suppl.):2766S–2771S.
36. Kantor LS. *A dietary assessment of the US food supply. comparing per capita food consumption with food guide pyramid service recommendations*. Report no. 772/1998. Washington, DC: Department of Agriculture, 1998.
37. Department of Community Services and Health. *National Dietary Survey of Adults: 1983*. Canberra: AGPS, 1987.
38. Australian Softdrink Association. Soft drink consumption statistics. Sydney: ASA, 2001.
39. Burt B, Eklund S, Morgan K, Larkin F, Guire K, Brown L 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;67:1422–9.
40. Crowley S, Antioch K, Carter R, Waters A-M, Conway L, Mathers C. *The cost of diet-related disease in Australia*. Canberra: Australian Institute of Health and Welfare, 1992.
41. Australian Institute of Health and Welfare. *Australia's health 2000: the seventh biennial report of the Australian Institute of Health and Welfare*, Canberra: AIHW, 2000.
42. Australian Institute of Health and Welfare. *Australia's health 1998: the sixth biennial health report of the Australian Institute of Health and Welfare*. Canberra: AIHW, 1998.
43. Slade GD, Spencer AJ, Davies MJ, Stewart JF. Caries experience among children in fluoridated Townsville and unfluoridated Brisbane. *Aust NZ J Publ Hlth* 1996;20(6):623–9.
44. McDonagh M. *A systematic review of public water flouridation*. Report no. 18. York: University of York, 2000.
45. Davies MJ, Spencer AJ, Westwater A, Simmons B. Dental caries among Australian Aboriginal, non-Aboriginal Australian-born, and overseas-born children. *Bull World Hlth Org* 1997;75(3):197–203.
46. Walsh LJ. Preventive dentistry. *Aust Dent J* 2000;45:76–82.
47. Ruxton C, Garceau F, Cotrell R. Guidelines for sugar consumption in Europe: is a quantitative approach justified? *Eur J Clin Nutr* 1999;53:503–13.
48. Balakrishnan M, Simmonds R, Tagg J. Dental caries is a preventable infectious disease. *Aust Dent J* 2000;45:235–45.
49. World Health Organization. *Diet, nutrition and the prevention of chronic diseases*. Geneva: WHO, 1990.
50. Gibson S, Williams S. Dental caries in pre-school children: association with social class, toothbrushing habit and consumption of sugars and sugar containing foods. *Caries Res* 1999;33:101–13.

51. British Nutrition Foundation. *Oral health diet and other factors*. Amsterdam: Elsevier, 1999.
52. Lam M, Riedy C, Coldwell S, Milgrom P, Craig R. Children's acceptance of xylitol-based foods. *Comm Dent Oral Epidemiol* 2000;28(2):97–101.
53. Gales MA, Nguyen TM. Sorbitol compared with xylitol in prevention of dental caries. *Ann Pharmacother* 2000;34(1):98–100.
54. Bessesen D. The role of carbohydrates in insulin resistance. *J Nutr* 2001;131(suppl.): 2782S–2786S.
55. Mann J, Truswell A, Manning E. Effects on serum lipids of reducing dietary sucrose or starch for 22 weeks in normal men. *S Afr Med J* 1972;4:827–34.
56. Parks E. Effect of dietary carbohydrate on triglyceride metabolism in humans. *J Nutr* 2001;131(suppl.):2772S–2774S.
57. Palli D, Russo A, Decarli A. Dietary patterns, nutrient intake and gastric cancer in a high-risk area of Italy. *Cancer Causes Control* 2001;12:163–72.
58. Franceschi S, Favero A, La Vecchia C, Negri E, Conti E, Montella M et al. Food groups and the risk of colorectal cancer in Italy. *Int J Cancer* 1997;72(1):56–61.
59. Boutron-Ruault M, Senesse P, Faivre J, Chatelain N, Belghiti C, Meance S. Foods as risk factors for colorectal cancer: a case-control study in Burgundy (France). *Eur J Cancer Prev* 1999;8:229–35.
60. Slattery M, Benson J, Berry T, Duncan D, Edwards S, Caan B et al. Dietary sugar and colon cancer. *Cancer Epid Bio Prev* 1997;6:677–85.
61. World Cancer Research Fund. *Food, nutrition and the prevention of cancer: a global perspective*. Washington, DC: American Institute for Cancer Research, 1997.
62. National Health and Medical Research Council. *The prevention, early detection and management of colorectal cancer*. Canberra: NHMRC, 1999.
63. American Academy of Pediatrics. Diagnosis and evaluation of the child with attention-deficit/hyperactivity disorder. *Pediatrics* 2000;105:1158–70.
64. Blackburn G, Kandars B, Lavin P, Keller S, Whatley J. The effect of aspartame as part of a multidisciplinary weight-control program on short and long term control of body weight. *Am J Clin Nutr* 1997;65:409–18.

2 PREVENT WEIGHT GAIN

- **Be physically active and eat according to your energy needs**

Malcolm Riley and Tim Gill

TERMINOLOGY

Physical inactivity

Physical inactivity (or *sedentary behaviour*) is defined as a state in which body movement is minimal—for example, when watching television, reading, working at a computer, talking on the telephone, driving a car, or meditating.

Sufficient physical activity

Sufficient physical activity to confer a health benefit requires regular participation in activity of *sufficient* duration and intensity. There is no clear, absolute threshold for health benefit, although the 1996 US Surgeon General's report¹ provided a scientific basis for health benefits to be achieved from participation in regular, moderate-intensity physical activity. This has been interpreted in the current Australian Physical Activity Guidelines² as the accumulation of 30 minutes' moderate physical activity on most, preferably all, days of the week. This guideline is not formulated with objectives such as weight loss or prevention of weight gain in mind: it is a modest target, one considered to be sufficient to result in a health benefit. Additional health benefits are expected to accrue from additional or more vigorous physical activity.

Body mass index

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. The body mass index, or BMI, has gained wide acceptance for the assessment of obesity in adults.

An index calculated by dividing the weight of an individual (measured in kilograms) by the square of their height (measured in metres), it is used as a simple estimate of the body fatness of a human being who does not have abnormal physical characteristics. The World Health Organization³ and the US National Institutes of Health⁴ 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 recent definition from Australia's National Health and Medical Research Council⁵ differs slightly in that overweight is defined as a BMI of *above* 25kg/m²

and obesity as a BMI of *above* 30kg/m². Table 2.1 shows the WHO and NHMRC classifications for BMI.

Table 2.1 Body mass index classification^{3,5}

Category	Body mass index (kg/m ²)	
	WHO	NHMRC
Underweight	<18.5	<20.0
Normal range	18.5–24.9	20.0–25.0
Overweight	≥25.0	>25.0–30.0
Pre-obese	25.0–29.9	
Obese class I	30.0–34.9	>30.0
Obese class II	35.0–39.9	
Obese class III	>40.0	

BACKGROUND

The second edition of the *Dietary Guidelines for Australians*⁶ included the guideline ‘Maintain a healthy body weight by balancing food intake and regular physical activity’. Since publication of that guideline in 1992, the proportion of the Australian population—both children and adults—classified as overweight or obese has increased substantially. As a result, the dietary guideline to the general public must now be directed towards reversing the trend towards increasing mean BMI. The diminishing physical activity of Australians has been recognised as an important determinant of the increasing prevalence of overweight and obesity. The *National Physical Activity Guidelines for Australians* was published in 1999², and those guidelines should be combined with the recommendations put forward in these dietary guidelines to achieve the greatest long-term health benefit.

Energy needs are influenced by genetic and environmental factors. The primary direct environmental factors that influence energy balance and are under individuals’ discretionary control are physical activity and dietary energy intake. Eating more food energy on a regular basis than is needed to meet your energy requirements can lead to energy storage in the form of excess body fat. Similarly, insufficient physical activity in relation to energy intake can lead to storage of energy as excess body fat.

Because of the increased use of labour-saving devices and technology, opportunities for physical activity have decreased. Meanwhile, for most Australians food is plentiful, palatable, energy dense, easily accessible and heavily promoted. Excess body fat is associated with adverse health consequences, including increased mortality, and is now a major public health

problem in Australia. Similarly—and independently—being inactive is associated with poorer health and increased mortality. For Australian adults, the mean level of excess body fat is increasing and the proportion of the adult population that is overweight or obese is substantial and increasing.

The cost of obesity (BMI greater than 30) in Australia has been estimated at 2 per cent of the total health budget⁷ and conservatively at \$840 million in 1992–93.⁵ A recent estimate for the United States suggests that the direct and indirect costs of obesity amount to 10 per cent of the national health care budget.⁸ A recent preliminary analysis of the cost of illness attributable to physical inactivity in Australia⁹ estimated the direct health care cost of preventing and treating six major conditions in 1993–94. The costs attributable to physical inactivity (and the percentage of the total estimated costs) were \$161 million (18.0 per cent) for coronary heart disease, \$16 million (8.7 per cent) for breast cancer, \$15.7 million (19.2 per cent) for colon cancer, \$101 million (16.0 per cent) for stroke, \$27.5 million (12.7 per cent) for type 2 diabetes, and \$56.2 million (10.0 per cent) for depressive disorders. Although the estimate is considered to be unreliable at this stage, the direct health care costs in 1993–94 for all causes of mortality attributable to physical inactivity was estimated at \$5651 million—18 per cent of the estimated total direct health care costs.

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

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

In general, Australians are advised to enjoy an amount of food that is sufficient to meet their energy needs. If their energy needs are low because of a sedentary occupation, this must be balanced by increased leisure-time physical activity or by a moderated appropriate energy intake. For best results, they should combine healthy eating with an active lifestyle. The health benefits that accrue from regular physical activity are substantial and are not restricted to control of excess body fat.

Current levels of obesity, overweight and physical activity

The 1995 National Nutrition Survey¹⁰ found that, at that time, 55.2 per cent of Australians aged 19 years or more were overweight or obese. In this study, heights and weights were measured by trained staff. For males the figure was

2. PREVENT WEIGHT GAIN

63.7 per cent; for females it 47.0 per cent. Overweight and obesity peaked at 50–54 years of age for men and 60–64 years for women. The proportion of the adult population with a BMI less than 18.5 (that is, underweight) was 1.4 per cent; in the 19–24 year age group it was 5.4 per cent in women and 2.4 per cent in men. Recent data from the National Health Survey¹¹ have indicated that overweight and obesity may have further increased. Their data showed a rise from 52% overweight and obesity based on self reported heights and weights in 1995 to 58% in 2001.

To determine dietary and anthropometric change between the 1983 National Heart Foundation Risk Factor Prevalence Survey and the 1995 National Nutrition Survey, a subset of the 1995 survey was used to adjust for a number of important study-design characteristics (including residence, age, and season of measurement) that differed between the two surveys. After the adjustment, the mean BMI for men had increased from 25.5 (95%CI: 25.3–25.6) in 1983 to 27.2 (95%CI: 27.0–27.5) in 1995. For women, the increase in mean BMI was from 24.3 (95%CI: 24.2–24.5) in 1983 to 26.8 (95%CI: 26.5–27.1) in 1995. The mean energy intake for males increased from 10 824 kilojoules (95%CI: 10 685–10 963) in 1983 to 11 195 kilojoules (95%CI: 10 956–11 434) in 1995; for females it increased from 7299 kilojoules (95%CI: 7204–7395) in 1983 to 7624 kilojoules (95%CI: 7464–7785) in 1995. These estimates are for adults aged 25 to 64 years living in capital cities.

Between 1983 and 1995 the proportion of adult women who were overweight or obese increased by 41 per cent and the proportion of adult men increased by 29 per cent (Australian Food and Nutrition Monitoring Unit, pers. comm. 2001). Mean BMI in a population is closely associated with the proportion of people classified as obese or overweight. As the population mean BMI increases above 23, the prevalence of obesity in that population increases at a faster rate because the BMI curves flatten out and skew to the right.³

Recent data from the National Health Survey ¹¹ showed that the numbers of people undertaking exercise in 2001 had increased from the 1989–90 and 1995 surveys but differences were small and most increase was in the light exercise category (from 33% in 1989–90 to 38% in 2001) with little change in those exercising at moderate to high levels. In contrast, the 1997 and 1999 National Physical Activity Surveys—telephone-administered surveys of 4824 (in 1997) and 3841 (in 1999) adult Australians aged 18 to 75 years—show that the proportion of Australian adults participating in sufficient physical activity to provide a health benefit declined from 62 per cent to 57 per cent.¹² The proportion of Australian adults who reported not doing any physical activity at all increased from 13 per cent to 15 per cent.

SCIENTIFIC BASIS

Overweight and obesity

A high level of heritability for obesity has been established by twin studies; recent estimates put the figure at 30–40 per cent. But the dramatic increase in the prevalence of obesity in Australia in the past 20 years^{5,7} cannot be explained by genetic factors: lifestyle factors such as a decrease in physical activity and overconsumption of energy provide the most reasonable explanation.

Most observational studies indicate a U- or J-shaped relationship between BMI and mortality, with individuals at very low and very high weights being at increased risk. Studies have suggested that one reason for the relationship between low BMI and mortality might be the detrimental effects of low lean body mass rather than low body fat.^{13,14} Others¹⁵ have found that, when analyses are controlled for smoking and weight loss associated with illness, an almost linear continuous relationship between BMI and mortality is found.

Individuals with a BMI of at least 30 have a 50–100 per cent increased risk of premature death due to all causes compared with individuals with a BMI of 20–25; most of the increase is due to cardiovascular causes.^{4,16} The relative increase in mortality rate attributable to obesity declines with age; however, an increased risk of death with higher BMI is seen even among individuals aged 65 to 74 years.¹⁷

The health risks of being either overweight or obese have recently been reviewed.¹⁸ The following conditions and symptoms are associated with obesity:

- coronary heart disease
- type 2 diabetes
- hypertension
- dyslipidaemia
- stroke
- sleep apnoea
- pulmonary dysfunction
- gall bladder disease
- liver disease
- osteoarthritis
- gout
- some cancers—colon, endometrial, post-menopausal breast cancer
- menstrual irregularities
- polycystic ovary syndrome
- infertility
- gestational diabetes
- neural tube defects in offspring

2. PREVENT WEIGHT GAIN

- low-back pain
- increased risk of anaesthetic complications
- carpal tunnel syndrome
- venous insufficiency
- deep vein thrombosis
- poor wound healing
- psychosocial problems
- osteoporosis—obesity is protective
- stress incontinence and leaking urine¹⁹
- prolapse
- oesophageal reflux
- constipation²⁰
- tiredness.²¹

For common causes of morbidity and mortality such as coronary heart disease, type 2 diabetes, hypertension and dyslipidaemia, the association with obesity follows a monotonic dose–response relationship, where the risk increases with the degree of obesity. It has been estimated that more than 70 per cent of people who are obese have at least one established co-morbidity.¹⁸

In a cross-sectional analysis of a large and representative survey of US adults²² from 1988 to 1994 (the NHANES III Survey), 63 per cent of the men and 55 per cent of the women were found to be overweight or obese. The risk of self-reported type 2 diabetes, gall bladder disease and high blood pressure was greater in the people who were overweight or obese, and the risk increased among those who were heavier.

In both sexes, weight gain during adult life is associated with increased risk of heart disease and death.^{16,23} Weight gain is a health risk that is independent of actual BMI.²⁴

Obesity refers to an excess of body fat, yet most data on the effects of obesity on health rely on measurement of body weight. One limitation of BMI as a measurement is that it does not incorporate body fat distribution, which is an independent predictor of health risk.⁴ Body fat may be preferentially deposited in the abdomen (android distribution) or surrounding the hips and thighs (gynoid distribution). The android distribution pattern reflects an accumulation of fat around the abdominal visceral organs.²⁵ Even at the same level of overweight, an individual with a greater amount of visceral fat is more likely to suffer health conditions associated with obesity than an individual with gynoid fat distribution.

Methods for accurately assessing visceral fat (such as computer assisted tomography or magnetic resonance imaging) are not routinely used, but an easily measured surrogate is waist circumference. A waist circumference of at least 88 centimetres in women and 102 centimetres in men has been associated with

increased health risk.⁴ It is now believed that measurement of waist circumference alone is more useful in identifying health risk in adults than the ratio of waist circumference to hip circumference.²⁵ Physical activity may favourably affect the distribution of body fat, independently of its effect on body weight.²⁶

Many studies (for example, references 27, 28) have indicated that intentional weight loss in obese individuals reduces the risk factors for, and alleviates the symptoms of, obesity-related conditions such as heart disease, type 2 diabetes and osteoarthritis in the short term (weeks or months). It is not necessary to lose large amounts of weight to achieve substantial health gains. For example, in a population of non-smoking US white women aged 40 to 64 years, a weight loss of 5 to 10 kilograms over one year was associated with a 25 per cent reduction in mortality.²⁹ Most studies measuring the impact of weight loss for a year or more tend to show continuing risk factor reduction.^{30–32} However, for most people who are classified as obese, and for many who are overweight, a return to a normal-range BMI is not a realistic target.³

In Australia—where BMI tends to increase with age—avoidance of weight gain in adult life is a successful outcome of weight management. Two trials to evaluate low-intensity programs designed to prevent weight gain in adults have been conducted in the United States.^{33,34} Forster et al.³³ randomised 219 normal-weight adults to either a control or a treatment group for 12 months. The ‘treatment’ consisted of monthly newsletters related to weight management, a financial incentive system, and an optional four-session education course in the sixth month of the program. The treatment group had an average weight loss of 1 kilogram, compared with no change in the untreated control group. Eighty-two per cent of the treatment group maintained or lost weight; 56 per cent of the control group did so. Older participants benefited more from the treatment than younger participants, and men more than women. Participants with prior experience of weight-loss programs were significantly less likely to maintain their pre-treatment weight than inexperienced participants.

Jeffrey and French³⁴ recruited 228 men and 998 women into a randomised controlled trial for three years. Participants were randomised into a control group (no treatment), a group that received education (mainly through monthly newsletters), and a group that received education plus a financial incentive (a \$100 ticket in a monthly lottery draw). The overall mean weight change over the three years was a gain of 1.7 kilograms, and there was no difference in weight change, or rate of gain, between the three groups. The study demonstrated that it was possible to run a mail-based education program and sustain the interest of the heterogeneous population of participants for three years. Jeffrey and French concluded that either stronger educational strategies were needed or education alone is insufficient to redress the problem of increasing weight throughout adulthood.

Physical activity and weight gain

Assistance with control of excess body fat is but one benefit of adopting an active lifestyle. In terms of cardiovascular and all-cause mortality, the benefit of an increase in physical activity is experienced more rapidly than changes to other risk factors—within two years in a US male cohort.³⁵ Studies consistently show that the greatest cardiovascular benefit occurs when sedentary or low-fitness groups in the population change to become groups with moderate activity or moderate fitness levels.

The effects of physical activity on cardiovascular disease and type 2 diabetes are independent of the effects of other risk factors: physical activity is beneficial to health at any level of BMI, regardless of whether BMI changes.²⁶

Recreational physical activity is predictive of future weight gain in prospective cohort studies^{36–38}; however, reducing inactivity (or sedentary activity) is also an important strategy for increasing incidental physical activity.

The following conclusions have been reached regarding physical activity and weight loss²⁶:

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

Six large-scale multiple risk factor community intervention trials that evaluated diet and exercise interventions have recently been reviewed.³⁹ None of the studies focused exclusively on weight, but each included significant nutrition and exercise education efforts. The trials were the Stanford Three Community Project, the Stanford Five City Study, the Minnesota Heart Health Program, the North Karelia Project, the Pawtucket Heart Health Program, and a national program for Mauritius. They ranged in duration from two to seven years and showed considerable success in intervention delivery. Only two of the studies showed any statistically significant weight-related effects and these were small. The Stanford Three Community Project and the Pawtucket Heart Health Program reported small attenuation of increases in relative weight (Stanford) or BMI (Pawtucket) in treatment versus control communities. For the Stanford Study, a difference in relative weight of no change versus a gain of 0.3 per cent in the control community was observed one year after the two-year intervention was completed. This small difference was not maintained when a comparison was made a year later. In each of the six trials, there were significant effects on other cardiovascular disease risk factors, suggesting that weight gain is a more difficult risk factor to deal with than other risk factors.

Dietary intake and weight gain

Experiments in animals and clinical studies in humans have shown that a long-term imbalance between total energy intake and expenditure is strongly and positively associated with excess body weight.⁴⁰ Consensus is emerging that the dietary factors that are key contributors to overeating and adult weight regulation are energy density and palatability.^{41–47} *Energy density* is the amount of food energy able to be metabolised per unit of weight or volume.⁴³ The two most important determinants of energy density in common foods are fat and water content. Fat plays a role because of its high energy density compared to protein and carbohydrates and water through a dilution effect. Dietary fibre also has an effect through ‘dilution’, but it is small because of the much smaller range of fibre concentration in food compared with water and fat. 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. The dietary fat content of meals does not influence energy intake provided energy density and palatability are held constant.^{41–45}

The satiety value of foods may also be important in the management of appetite and hunger. There is evidence to suggest that protein-rich foods and diets have a greater effect on satiety than high-fat foods.^{48,49}

Special groups

Indigenous Australians

The magnitude of the problem of overweight and obesity among Indigenous Australians can hardly be overstated. In the 1994 National Aboriginal and Torres Strait Islander Survey, of those adults who were measured, about 25 per cent of males and 28 per cent of females had a BMI of at least 30.^{50,51} The figure for Australian adults was 19 per cent in 1995. There is a suggestion that the prevalence of obesity is higher in rural communities, at least among women, and it appears that the prevalence has rapidly increased in rural communities in recent years.⁵² In general, obesity and overweight among Indigenous Australians tend to have a central distribution.⁵¹ It should be noted that Torres Strait Islanders are substantially heavier than Aboriginal people.

In surveys conducted in three regions of north Queensland between 1998 and 2000⁵³, the proportion of males aged 35 years or more who had a BMI of at least 30 varied from 19 to 50 per cent; for females, the equivalent figures were 24 to 69 per cent. The proportion of adults aged 35 years or more with a waist-to-hip ratio above 0.9 for males and 0.8 for females varied from 87 to 94 per cent in males and from 90 to 97 per cent in females.

Blue-collar workers

An analysis of the 1995 Australian National Health Survey results⁵⁴ shows that blue-collar workers—tradespeople, plant and machine operators and drivers, and labourers and related workers—were 50 per cent more likely to be classified as

insufficiently active with respect to leisure-time physical activity. This group was also identified as being more likely to be overweight or obese⁵ and to experience higher rates of mortality and morbidity from cardiovascular disease.⁵⁵

Australians of Asian origin

Whilst there is as yet little evidence that Australians of Asian origin may be at increased risk of overweight, special consideration might need to be given to this group in assessing body fatness. The WHO-proposed levels of BMI that correspond to increasing degrees of risk of chronic morbidity and of mortality were primarily derived for populations of European ethnicity. Assessment of obesity in people of non-European ethnicity has been the subject of many studies and much debate. The levels of BMI at which morbidity develops in non-Europeans are lower than the levels shown in Table 2.1. Most studies of obesity in non-Europeans have been done on subjects of Asian origin. At present there is no international agreement on the cut-off levels to be used. Researchers in Singapore have proposed that the BMIs for their population that would equate to the body fat proportion of a Caucasian with a BMI of 30.0 would be 27.5 for people of Chinese origin, 27.0 for Malays and 26.0 for Indians.⁵⁶ An alternative classification has also been proposed by a WHO working party—see Table 2.2.

Since there is no agreement on reference standards for non-Europeans, as an interim arrangement it is recommended that the WHO standards shown in Table 2.2 be used for community reporting. The numbers or proportions of different ethnic groups at each level should be reported.

Table 2.2 WHO-proposed classification of weight and co-morbidity risk by BMI in adult Asians⁵⁷

Category	BMI (kg/m ²)	Risk of co-morbidities
Underweight	<18.5	Low (but increased risk of other clinical problems)
Normal range	18.5–22.9	Average
Overweight	>23.0	
At risk	23.0–24.9	Increased
Obese I	25.0–29.9	Moderate
Obese II	>30.0	Severe

PRACTICAL ASPECTS OF THIS GUIDELINE

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

There is growing consistency in the recommendations made to prevent weight gain in populations:

- Increase the level of physical activity, by whatever means are appropriate, to a higher level than at present. This includes incidental activity and low-intensity (but long-duration) leisure pursuits as well as moderate and vigorous exercise.
- Reduce the time spent being physically inactive.
- Choose a less energy dense diet.

Attention has recently been given to the need to facilitate the adoption and maintenance of lifestyle physical activities.^{5,58} These are self-selected activities, could be planned or unplanned, and can include leisure, occupational and household activities that are accumulated throughout the day. It has been realised that a substantial proportion of the population is completely inactive; many people feel they do not have time to exercise, dislike vigorous exercise, and/or dislike the imposed conformity of organised exercise programs such as those offered by gyms. Evidence has now accumulated that moderate-level physical exercise taken intermittently also produces a health benefit.⁵⁸ The change in emphasis from ‘exercise training/physical fitness’ to ‘lifestyle physical activity’ allows people to adopt healthy behaviours that take into account their individual, cultural and environmental differences. This broader public health approach helps to dispel the misconception that vigorous exercise is the only way to become physically fit. Furthermore, it presents a scientific basis for the design of public infrastructure to encourage incidental physical activity—for example, pleasant walkways and wide stairways in buildings.

Three evidence-based recommendations have been made for the delivery of brief physical activity interventions during routine health care delivery⁵⁹:

- An initial focus on physical activity only is recommended, although maintenance may be enhanced when supported by other risk factor interventions.
- Tailored interventions and written materials enhance success rates.
- Physical activity counselling can be successfully implemented by a variety of members of the health care team: the person who delivers the intervention should be whoever is most likely to do so consistently, given time, training and interest.

It has been suggested⁶⁰ that successful behaviour change in relation to weight can result from education and counselling of patients and should include behavioural techniques (especially self-monitoring) and both personal communication and written or other audiovisual materials.

As noted, there is strong evidence to suggest that foods with a higher energy density encourage energy intake above requirements.^{41–47,61,62} Energy from drinks in particular may add to total energy intake without displacing energy consumed in the form of solid food.^{63–65}

2. PREVENT WEIGHT GAIN

Getting Started, a 20-page publication produced by Active Australia, provides practical advice for people considering changing their behaviour in relation to both physical activity and dietary intake.⁶⁶

RELATIONSHIP TO OTHER GUIDELINES

Enjoy a wide variety of nutritious foods

Variety in food choices is necessary if we are to obtain a balance of all the nutrients required for optimal health. In the context of prevention of weight gain, eating a wide variety of low-energy density foods, together with appropriate amounts of the other key food groups, will help prevent weight gain.

Eat plenty of vegetables, legumes and fruits

Vegetables and fruits are low-fat foods with high water and fibre content and are therefore low in energy density.

Drink plenty of water

Water is a low-energy drink that can quench thirst without adding energy to the diet.

Limit saturated fat and moderate total fat intake

Fat in foods is one of the main determinants of energy density, so intakes of fats and fatty foods should be moderated.

Limit your alcohol intake if you choose to drink

Compared with water and some other drinks, alcoholic drinks can be high in energy, and this should be taken into account when trying to moderate overall energy intake.

CONCLUSION

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

EVIDENCE

Strong, consistent evidence linking body fatness with mortality and morbidity comes from large prospective cohort studies. Randomised controlled trials of interventions to gain body fat are not feasible, although randomised controlled trials (Level II evidence) to examine interventions to lose body fat have been conducted (reference 32). The results have been consistent with those of the cohort and cross-sectional studies; that is, that excess body fat is associated with poorer health.

There is Level II evidence for the effect of weight loss on hypertension (reference 32). There is Level III evidence for a relationship between the following:

- body weight or body fat and mortality (references 14, 15 and 17)
- weight status and heart disease (reference 16)
- increased obesity and diabetes and decreased plasma cholesterol in Aboriginal Australians (reference 52)
- weight gain or body weight and elevated blood pressure (reference 24)
- weight loss and reduced mortality (reference 29)
- increased physical fitness and reduced mortality (reference 35)
- physical activity and limiting future weight gain (references 36 to 38).

Additional evidence comes from population-based cross-sectional studies.

The association between BMI and specific diseases ranges from strong (relative risk much greater than 3) for type 2 diabetes and dyslipidaemia, to moderate (relative risk between 2 and 3) for coronary heart disease and hypertension, and slight (relative risk less than 2) for post-menopausal breast cancer and foetal defects (see reference 3). The mortality and morbidity associations are of substantial clinical and public health significance, particularly in view of the high and increasing prevalence of overweight and obesity in Australia.

The evidence for the health benefit of increased physical activity is considered good to excellent. Most of it comes from good-quality prospective cohort studies (for example, reference 35). Despite the many different methods of measuring physical activity, the health benefits observed are very consistent between studies, and reviews have found stronger associations for studies that used better research designs and methods.

Many of the publications referred to in this chapter are review articles covering a range of subjects. The studies just noted in relation to the levels of evidence also cover a range of subjects—from studies of the effect of particular interventions relating to physical activity to studies describing changes in health parameters according to changes in body weight over time. Many more published studies are relevant to this dietary guideline: no attempt is made here to comprehensively review them. Most of the stronger published evidence relating adult weight gain, body mass and physical activity to health comes from prospective cohort studies.

REFERENCES

1. US Department of Health and Human Services. *Physical activity and health: a report of the Surgeon General*. Atlanta, GA: Department of Health and Human Services, Centers for Disease Control and Prevention & National Center for Chronic Disease Prevention and Health Promotion, 1996.
2. Department of Health and Aged Care. *National physical activity guidelines for Australians*. DHAC: Canberra, 1999.
3. World Health Organization. *Obesity: preventing and managing the global epidemic*. Report of a WHO consultation. WHO Technical Report Series no. 894. WHO: Geneva, 2000.
4. US National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. *Obes Res* 1998;6(suppl. 2):51S–209S.
5. National Health and Medical Research Council. *Acting on Australia's weight: a strategic plan for the prevention of overweight and obesity*. Canberra: Australian Government Publishing Service, 1997.
6. Department of Health. *Dietary guidelines for Australians*. Canberra: AGPS, 1982.
7. Segal L, Carter R, Zimmet P. The costs of obesity: the Australian perspective. *Pharmacoeconomics* 1994;5(suppl.):S45–S52.
8. Wolf AM, Colditz GA. Current estimates of the economic costs of obesity in the United States. *Obes Res* 1998;6:97–106.
9. Stephenson J, Bauman A, Armstrong T, Smith B, Bellew B. *The costs of illness attributable to physical inactivity in Australia: a preliminary study*. Canberra: Department of Health and Aged Care, 2000.
10. Australian Bureau of Statistics & Department of Health and Family Services. *National Nutrition Survey: selected highlights, Australia*. ABS cat. no. 4802.0. Canberra: ABS, 1997.
11. ABS National Health Survey 2001. <http://www.abs.gov.au/Ausstats/abs%40.nsf>
12. 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.
13. Allison DB, Faith MS, Heo M, Kotler DP. Hypothesis concerning the U-shaped relation between body mass index and mortality. *Am J Epidemiol* 1997;146:339–49.
14. Allison DB, Zanolli R, Faith MS, Heo M, Pietrobelli A, VanItallie TB et al. Weight loss increases and fat loss decreases all-cause mortality rate: results from two independent cohort studies. *Int J Obes Relat Metab Disord* 1999;23(6):603–611.

15. Manson JE, Willett WC, Stampfer MJ. Body weight and mortality among women. *New Engl J Med* 1995;333:677–85.
16. Rimm EB, Stampfer MJ, Giovannucci E, Ascherio A, Spiegelman D, Colditz GA et al. Body size and fat distribution as predictors of coronary heart disease among middle-aged and older US men. *Am J Epidemiol* 1995;141:1117–27.
17. 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.
18. National Taskforce on the Prevention and Treatment of Obesity. Overweight, obesity and health risk. *Arch Int Med* 2000;160:898–904.
19. Chiarelli P, Brown WJ, McElduff P. Leaking urine: prevalence and associated risk factors in Australian women. *Urology and Urodynamics* 1999;18:567–77.
20. Chiarelli P, Brown WJ, McElduff P. Constipation in Australian women: prevalence and associated factors. *Int Urogynaecology J* 2000;11:71–8.
21. Brown WJ, Mishra G, Kenardy J, Dobson AJ. Relationships between BMI and well-being in young Australian women. *Int J Obes* 2000;24:1360–8.
22. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA* 1999;282:1523–38.
23. Bray GA. In defence of a body mass index of 25 as the cut-off point for defining overweight. *Obes Res* 1998;6:461–2.
24. Sonne-Holm S, Sorensen TI, Jensen G, Schnohr P. Independent effects of weight change and attained body weight on prevalence of arterial hypertension in obese and non-obese men. *BMJ* 1989;299:767–70.
25. Despres JP, Lemieux I, Prud'homme D. Treatment of obesity: need to focus on high risk abdominally obese patients. *BMJ* 2001;322:716–20.
26. Rippe JM, Hess S. The role of physical activity in the prevention and management of obesity. *J Am Diet Ass* 1998;98(suppl.):S31–S38.
27. Goldstein DJ. Beneficial effects of modest weight loss. *Int J Obes* 1992;16:397–415.
28. Pi-Sunyer FX. Short-term medical benefits and adverse effects of weight loss. *Ann Intern Med* 1993;119:722–6.
29. Williamson DF, Pamuk E, Thun M, Flanders D, Byers T, Heath C. Prospective study of intentional weight loss and mortality in never-smoking overweight US white women aged 40–64 years. *Am J Epidemiol* 1995;141(12):1128–41.
30. Pi-Sunyer FX. A review of long-term studies evaluating the efficacy of weight loss in ameliorating disorders associated with obesity. *Clin Ther* 1996;18:1006–35.

2. PREVENT WEIGHT GAIN

31. Sjoström CD, Lissner L, Sjoström L. Relationships between changes in body composition and changes in cardiovascular risk factors: the SOS Intervention Study. Swedish Obese Subjects. *Obes Res* 1997;5:519–30.
32. Whelton PK, Appel LJ, Espeland MA, Applegate WB, Ettinger WH, Kostis JB et al. for the TONE Collaborative Research Group. Sodium reduction and weight loss in the treatment of hypertension in older persons—a randomized controlled trial of nonpharmacologic interventions in the elderly (TONE). *JAMA* 1998;279:839–46.
33. Forster JL, Jeffrey RW, Schmid TL, Kramer FM. Preventing weight gain in adults: a pound of prevention. *Hlth Psych* 1988;7:515–25.
34. Jeffrey RW, French SA. Preventing weight gain in adults: the Pound of Prevention Study. *Am J Publ Hlth* 1999;89:747–51.
35. Blair SN, Kohl HW III, Barlow CE, Paffenberger RS Jr, Gibbons LW, Macera CA. Changes in physical fitness and all causes mortality: a prospective study of healthy and unhealthy men. *JAMA* 1995;273:1093–8.
36. Coakley EH, Rimm EB, Colditz G, Kawachi I, Willett W. Predictors of weight change in men: results from the Health Professionals Follow-up Study. *Int J Obes Rel Metab Disord* 1998;22:89–96.
37. Williamson DF, Madans J, Anda RF, Kleinman JC, Kahn HS, Byers T. Recreational physical activity and ten-year weight change in a US national cohort. *Int J Obes Rel Metab Disord* 1993;17:279–86.
38. Haapanen N, Miilunpalo S, Pasanen M, Oja P, Vuori I. Association between leisure time physical activity and 10-year body mass change among working-aged men and women. *Int J Obes Rel Metab Disord* 1997;21:288–96.
39. Schmitz MKH, Jeffrey RW. Public health interventions for the prevention and treatment of obesity. *Med Clin N Am* 2000;84:491–512.
40. Bray GA, Popkin BM. Dietary fat intake does effect obesity? *Am J Clin Nutr* 1998;68:1157–73.
41. McCorty MA, Fuss PJ, Saltzman E, Roberts S. Dietary determinants of energy intake and weight regulation in healthy adults. *J Nutr* 2000;130(suppl.):276S–279S.
42. Rolls BJ. The role of energy density in the overconsumption of fat. *J Nutr* 2000;130(suppl.):268S–271S.
43. Yao M, Roberts SB. Dietary energy density and weight regulation. *Nutr Rev* 2001;59:247–58.
44. Bell EA, Rolls BJ. Energy density of foods affects energy intake across multiple levels of fat content in lean and obese women. *Am J Clin Nutr* 2001;73:1010–18.
45. Saltzman E, Dallal GE, Roberts SB. Effect of high-fat and low-fat diets on voluntary energy intake and substrate oxidation: studies in identical twins consuming diets matched for energy density, fiber and palatability. *Am J Clin Nutr* 1997;66:1332–9.

46. Stubbs RJ, Johnstone AM, O'Reilly LM, Barton K, Reid C. The effect of covertly manipulating the energy density of mixed diets on ad libitum food intake in 'pseudo free-living' humans. *Int J Obes* 1998;22:980–7.
47. Stubbs RJ, Johnstone AM, Harbron CG, Reid C. Covert manipulation of energy density of high carbohydrate diets in 'pseudo free-living' humans. *Int J Obes* 1998;22:885–92.
48. Holt SHA, Brand Miller JC, Petocz P, Farmakalidis E. A satiety index of common foods. *Eur J Clin Nutr* 1995;49:675–90.
49. Stubbs R. Macronutrient effects on diet. *Int J Obes* 1995;19(suppl. 5):S11–S19.
50. Mackerras D, Cunningham J. Body mass index distribution in the 1994 National Aboriginal and Torres Strait Islander Survey. *Proc Nutr Soc Aust* 1996;20:169.
51. National Health and Medical Research Council. *Nutrition in Aboriginal and Torres Strait Islander peoples: an information paper*. Canberra: NHMRC, 1999.
52. McDermott R, Rowley K, Lee AJ, Knight S, O'Dea K. Increase in the prevalence of obesity and diabetes and decrease in plasma cholesterol in a Central Australian Aboriginal community. *Med J Aust* 2000;172:480–4.
53. Queensland Health. *Community report: well person's health check*. Brisbane: Queensland Health, 2001.
54. Burton NW, Turrell G. Occupation, hours worked, and leisure-time physical activity. *Prev Med* 2000;31:673–81.
55. Bennett S. Socioeconomic inequalities in coronary heart disease and stroke mortality among Australian men, 1979–1993. *Int J Epidemiol* 1996;25:266–75.
56. Deurenberg-Yap M, Schmidt G, van Staveren WA, Deurenberg P. The paradox of low body mass index and high body fat percentage among Chinese, Malays and Indians in Singapore. *Int J Obes Rel Metab Disord* 2000;24(8):1011–17.
57. Inoue S, Zimmet P. *The Asian–Pacific Perspective: redefining obesity and its treatment*. Sydney: Health Communications Australia Pty Limited, 2000.
58. Dunn AL, Andersen RE, Jakicic JM. Lifestyle physical activity interventions—history, short- and long-term effects, and recommendations. *Am J Prev Med* 1998;15:398–412.
59. Eakin EG, Glasgow RE, Riley KM. Review of primary care based physical activity intervention studies—effectiveness and implications for practice and future research. *J Fam Pract* 2000;49:158–68.
60. Mullen PD, Simons-Morton DG, Ramirez G, Frankowski RF, Green LW, Mains DA. A meta-analysis of trials evaluating patient education and counseling for three groups of preventive health behaviours. *Patient Educ Couns* 1997;32:157–73.

2. PREVENT WEIGHT GAIN

61. Bell EA, Castellanos VH, Pelkman CL, Thorwart ML, Rolls BJ. Energy density affects energy intake in normal-weight women. *Am J Clin Nutr* 1998;67:412–20.
62. Stubbs RJ, Ritz P, Coward WA, Prentice AM. Covert manipulation of the ratio of dietary fat to carbohydrate and energy density: effect on food intake and energy balance in free-living men eating ad libitum. *Am J Clin Nutr* 1995;62:330–7.
63. Mattes RD. Dietary compensation by humans for supplemental energy provided as ethanol or carbohydrate in fluids. *Physiol Behav* 1996;59:179–87.
64. De Castro JM. The effects of the spontaneous ingestion of particular foods or beverages on the meal pattern and overall nutrient intake of humans. *Physiol Behav* 1993;53:1133–44.
65. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 2001;357:505–8.
66. Australian Sports Commission. *Getting started*. Canberra: ASC, 1988.

3 CARE FOR YOUR FOOD

- **Prepare and store it safely**

Rochelle Finlay and Patricia Blenman

BACKGROUND

Despite having one of the world's safest food-supply systems, Australia has seen an increase in the number of reported foodborne illnesses in the last 10 years. Foodborne illness can have very serious health consequences, particularly for vulnerable groups in the population such as the elderly and people who are immuno-compromised because of disease or medical treatments.

Correct handling of food during all stages of its preparation and storage is essential in reducing the incidence of foodborne illness. Because of their relative importance, this background guideline concentrates on microbiological aspects of food safety and practical matters.

The incidence of foodborne illnesses

Reported data on foodborne illnesses consistently underestimate the true incidence of these illnesses, and full diagnostic testing is usually done only in more severe cases or when there are extensive common-source outbreaks.^{1,2} As a result of their apparently increasing incidence, in Australia and worldwide, foodborne diseases pose a significant public health problem.³ A number of factors are thought responsible for the increase in foodborne illness^{4,5}:

- changes in human demographics, resulting in a greater proportion of the population with increased susceptibility to severe foodborne illness
- changes in food-production methods, including intensive farming practices and more extensive food-distribution systems. Food grown in one country can now be transported and consumed halfway across the world
- new and emerging pathogens. Three of the four most significant foodborne pathogens in the United States (campylobacter, listeria, and enterohaemorrhagic *Escherichia coli*) were unrecognised as causes of foodborne illness 20 years ago
- consumer preferences, particularly with the increasing trend towards takeaway food and dining out.

An increase in notifications of foodborne illness has also occurred, for two main reasons:

- better reporting and identification of pathogens.⁴ Dramatic scientific and technological improvements in the detection of pathogens have contributed to the reporting of increasing numbers of cases of foodborne disease that may have previously gone unreported
- increased awareness among consumers and health professionals.

3. CARE FOR YOUR FOOD

In 1999 it was estimated that the annual cost to Australia of foodborne illness was \$2.6 billion.⁴ A reduction in the incidence of such illness would benefit the Australian community through lower health care costs, less absenteeism, improved business productivity, increased competitiveness in world markets, and reduced levels of business failure and associated costs, including the cost of litigation.⁴

In 1999 and 2000 the three most common notified foodborne diseases in Australia were infections with campylobacter, salmonella and hepatitis A (see also Table 3.1).

Table 3.1 Notifications of foodborne illness received by Australian health authorities: selected pathogens, 1991 to 2000⁶

Year	Pathogen				
	Campylobacter	Hepatitis A	Listeria	Salmonella	Yersinia
1991	8 672	2 195	44	5 440	515
1992	9 136	2 109	38	4 614	567
1993	8 111	2 006	53	4 731	459
1994	10 117	1 901	34	5 327	414
1995	10 933	1 600	58	5 895	306
1996	12 158	2 150	70	5 819	268
1997	11 851	3 076	71	7 005	245
1998	13 449	2 503	58	7 700	207
1999	12 643	1 563	62	7 330	142
2000	13 455	824	66	6 017	71

Note: It is generally recognised that only a small proportion of cases are reported.

The general trend in the last 10 years in Australia is for gradual increases in notifications of foodborne illnesses associated with campylobacter, salmonella and listeria.

SCIENTIFIC BASIS

Characteristics of foodborne illness

The symptoms of foodborne illness are dependent on the pathogen responsible and the immune status of the affected person. Symptoms can range from being so mild as to be hardly noticeable (in healthy adults) to being so severe that hospitalisation is needed.⁷ Common symptoms are abdominal pain, nausea, vomiting, diarrhoea, bloody stools, fever, and dehydration. People may

experience fatigue, fever and muscle pain.⁷ In serious cases, and depending on the pathogen, foodborne illness may result in double vision, trouble with swallowing or breathing, paralysis, encephalopathy, kidney failure, or death.⁴ Some foodborne pathogens can also trigger longer term effects such as reactive arthritis and auto-immune disorders.⁸

The time taken between infection by the pathogen and development of symptoms also varies according to the pathogen involved. Symptoms of commonly acquired foodborne infections can present themselves within two to four hours or up to 10 days after infection. For some agents, such as *Listeria monocytogenes*, the average incubation period is three weeks.

Health status and susceptibility

Immune system function

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

Gastrointestinal tract function

In healthy adults, no specific gastrointestinal functions contribute to infection by foodborne pathogens. In people with weakened immune systems, malnutrition predisposes to gastrointestinal infection. Nutritional deficiency or infection causes gastritis and a resultant decline in gastric secretion of hydrochloric acid. This decrease in stomach acidity increases the chance of infection if foodborne pathogens are ingested.⁹

Causes of foodborne illnesses

Foodborne illnesses can be caused by bacteria, viruses or bacterial toxins.⁴ Bacterial food poisoning occurs when pathogenic bacteria multiply to harmful levels as a result of incorrect handling of food, particularly if temperature control is inadequate. But not all foodborne pathogens need to multiply in food to cause illness. Viruses such as hepatitis A and Norwalk virus, and some strains of bacteria—such as *Escherichia coli* (for example, *E. coli* O157:H7 and *E. coli* O111), *Campylobacter jejuni* and *Shigella* spp.—can cause illness, even when present in low numbers. Food must be protected from contamination if these pathogens are to be excluded. If a ready-to-eat food is contaminated with these pathogens, illness may occur and, once the pathogens are present, keeping the food at a safe temperature will not have any effect.¹⁰

A number of micro-organisms produce toxins when allowed to multiply to high levels in food, and eating food that contains such toxins can cause foodborne illness. For example, botulism is caused by ingestion of a toxin produced by

3. CARE FOR YOUR FOOD

Clostridium botulinum present in contaminated food; other micro-organisms linked with toxin production in food are *Staphylococcus aureus* (which causes staphylococcal food poisoning), *Bacillus cereus* and shigella. Toxin formation can be prevented if foods are kept at safe temperatures. Even re-heating food to high temperatures will not destroy toxins.¹¹

The following are the main causes of foodborne illness in Australia:

- inadequate cooking
- improper holding temperatures
- contaminated equipment
- unsafe food sources
- poor personal hygiene.¹

Temperature

Exposure to high temperatures, such as those used in cooking, should destroy the vegetative cells of bacteria. Some bacteria do, however, have heat-resistant spores and toxins that survive the cooking process; an example is *Bacillus cereus*. Cooling to low temperatures, such as refrigeration, will slow bacterial growth. With the exception of *Listeria monocytogenes* and *Yersinia enterocolitica*, pathogenic bacteria do not multiply at temperatures at or below 5°C. Foods that support the growth of foodborne bacteria should be stored at or below 5°C or at or above 60°C. Between 5°C and 60°C is considered to be the ‘danger zone’ for food safety: within this temperature range, bacterial replication can occur.

Time

The longer food is left in the temperature danger zone, the more time bacteria will have to multiply. Some bacteria can reach an infective dose in four to six hours at temperatures within the danger zone.^{12,13}

Food contamination

Microbiological food contamination can occur in a number of ways:

- The utensils used to prepare raw food—such as a chopping board used to cut raw meat and poultry—are then used on ready-to-eat food without having been cleaned, sanitised and dried.
- Raw foods are allowed to make direct contact with ready-to-eat foods.
- People preparing or serving food contaminate it by not washing their hands adequately, particularly after handling raw food and immediately after using the toilet.
- Food storage and preparation areas are themselves contaminated through inadequate cleaning and sanitising or are open to contamination by pests such as insects and rodents.

Potentially hazardous foods

The ability of micro-organisms to grow in a food depends on external factors (such as temperature) as well as the characteristics of the food itself (such as nutrient content, water content and pH). Bacteria need adequate nutrients for replication; among their most suitable media are high-protein, perishable foods such as dairy products, egg products, seafood, meat and poultry.^{12,13} These foods also have a relatively high moisture content: bacterial growth is limited in the absence of moisture. The acidity or alkalinity of a food also affects bacterial growth: bacteria are least active in very acidic foods (those with a pH less than 4.5). Often foods are preserved using vinegar to reduce bacterial growth, although moulds may still grow in these conditions.¹³

The following are examples of foods that are normally considered potentially hazardous¹⁰:

- raw and cooked meat or foods containing raw or cooked meat—for example, casseroles, curries and meat pies
- dairy products and foods containing dairy products—for example, milk, custard and dairy-based deserts
- seafood and foods containing seafood
- cooked rice and pasta
- processed fruits and vegetables such as salads
- processed foods containing eggs or other protein-rich food
- foods that contain any of the foods just listed—for example, sandwiches.

The foods most commonly implicated in foodborne illness in Australia are meat and seafood.¹

Some foodborne pathogens, such as viruses and enterohaemorrhagic strains of *Escherichia coli*, do not need to grow in foods to produce illness. Contamination of any ready-to-eat food with such a pathogen can result in foodborne illness.

Special groups

People at particular risk because of a medical condition

In the adult population there are people who are more susceptible to, or ‘at risk’ of, foodborne illness. This includes people with HIV or AIDS, cancer, diabetes, kidney or liver disease, haemochromatosis (an iron disorder), stomach problems (including previous stomach surgery) and low stomach acid (from antacid use). Also at risk are people treated with immuno-suppressant medications, people undergoing bone marrow or stem cell transplantation, and people who have a history of long-term steroid use (for asthma or arthritis, for example). The elderly are also at greater risk because of their weakened immune systems.

Pregnancy

Pregnant women are considered to be at risk from listeriosis, a foodborne infection which, if transmitted to the unborn child, can lead to miscarriage, stillbirth or premature birth. Foods that are more likely to contain listeria should therefore be avoided during pregnancy; they are mostly chilled, ready-to-eat foods such as the following:

- soft cheeses such as Brie, Camembert and ricotta—although these are safe if cooked and served hot
- takeaway chilled cooked, diced chicken, as used in chicken sandwiches. Freshly cooked chicken is safe
- cold meats bought from supermarkets, sandwich bars, and so on
- pate
- pre-prepared or stored salads
- raw seafood—such as oysters and sashimi
- smoked seafood—such as smoked salmon and smoked oysters, although canned varieties are safe
- unpasteurised milk and foods made from it.

Food Standards Australia New Zealand's brochure *Listeria and Pregnancy* provides further information on listeria and reducing the risk of infection during pregnancy <www.foodstandards.gov.au>.

PRACTICAL ASPECTS OF THIS GUIDELINE

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

Purchasing

Although the standard of foods available in Australia is generally very high, buyers should look for defects in packaging, such as improper sealing, foreign objects and signs of spoilage. It is best to leave the buying of chilled and frozen foods until the end of a shopping trip to prevent them from warming.^{12,14} Foods such as this can be put in a cooler or Esky for transport home.

Storage

The various food types need to be stored properly to retain their nutrient value, freshness, aroma and texture, and to keep them safe.^{12–16} Always read the label for storage instructions. Ensure that storage areas such as cupboards and pantries are clean¹⁷ and that foods are stored in food-grade containers away from chemicals. Store raw foods separately from ready-to-eat foods to prevent cross-contamination.¹⁰

Refrigeration

Refrigeration retards the growth of bacteria and the rate of chemical change in food. The refrigerator temperature should be 5°C or less—this can be checked using a suitable thermometer—and care should be taken to ensure that the temperature is maintained. All cooked foods should be covered and stored on a shelf above uncooked foods.^{12,13,15} Leftovers and ready-to-eat meals should be used the next day or stored in the freezer. Raw meats should be wrapped or placed in a container and stored near the bottom of the refrigerator, so that the juices do not drip onto other foods. Any spills should be cleaned up immediately, and fridge and freezer shelves and doors should be cleaned regularly. Ready-to-eat chilled foods are becoming widely available; they should be stored in the coldest part of the fridge and used before the ‘use by’ or ‘best before’ date or as soon as possible after purchase.

Frozen foods

Care should be taken to ensure that frozen food is kept hard frozen. It should be stored in packages that are free of air and fully sealed, to prevent ‘freezer burn’. Freezer burn is dehydration or drying that occurs on the surface of a product if it is improperly wrapped; the product is safe to eat but of poorer quality.

Canned and other hermetically sealed foods

Canned and other hermetically sealed foods—such as foods sealed in glass jars—should be stored in a cool place. Read the labels carefully for any storage instructions. Once opened, canned foods should be stored in the refrigerator, preferably not in the can. Swollen or leaking cans indicate faulty processing: their contents should not be eaten. In addition, throw out the contents of any can if there is an unusual odour. When opening vacuum-sealed jars, listen for a popping sound, which shows that the jar’s seal was intact.

Vacuum-packed and modified-atmosphere packed foods

Vacuum packing extends the shelf life of food by removing air from the packages. Modified-atmosphere packaging extends shelf life by replacing the oxygen in a packaged food with other gases that slow bacterial growth; the method is often used with meat and poultry products. An increasing number of blister packs of foods such as fresh pasta, lunch meat, bacon and olives are now available. Vacuum-packed and modified-atmosphere packed foods should be stored according to the instructions on the package.

Dehydrated and dried foods

Dehydration inhibits the growth of micro-organisms by removing water, but it does not make the food sterile¹⁵: a high level of micro-organisms can remain, only to become active again when the food is rehydrated. Rehydrated foods should be treated as perishables and be stored in the refrigerator. Dried food should be stored in a sealed container and in a cool, dry place away from direct heat or sunlight. It should be regularly inspected for insect infestation. Opened packages of dried food can be stored in the refrigerator to maintain quality for longer.

Date-marked packaged foods

The ‘best before’ date on packaged food signifies the end of the period during which the intact package of food—if stored in accordance with any stated storage conditions—will retain all of its quality attributes, such as colour, taste, texture and flavour.¹⁸ Foods that are date-marked in this way can continue to be sold after that date provided the food is not damaged or has not deteriorated or perished. Check foods that have passed their ‘best before’ date for signs of spoilage.

The ‘use by’ date on packaged food signifies the end of the estimated period—if the food is stored in accordance with any stated storage conditions—after which the intact package of food should not be consumed for health and safety reasons.¹⁸ Foods marked with a ‘use by’ date are prohibited from being sold after this date because the food might then pose a health risk.

Food spoilage

Food spoilage occurs when food-spoiling bacteria multiply and cause the food to deteriorate and develop unpleasant odours, tastes and textures. The bacteria spoil the food so that it becomes inedible, but they do not themselves cause foodborne illness. On the other hand, food in which pathogenic micro-organisms have grown to high levels may appear and taste normal. Food handling measures used to optimise food safety, such as proper storage and temperature controls, also help prevent premature spoilage.

Food preparation

Handwashing and hygiene

Before starting to prepare food, people should thoroughly wash and dry their hands. This is particularly important after:

- handling raw foods—such as raw meat
- touching animals
- using the toilet
- assisting others with toilet use
- blowing noses
- changing children’s nappies.^{16,19–21}

Hands should be lathered and held under running water to ensure that any micro-organisms are washed away. Particular attention should be paid to washing between fingers and under fingernails. After washing, hands should be dried using either a clean towel or a paper towel. Food should not be prepared by anyone who might be suffering from a foodborne illness or who has a foodborne disease.¹⁰

Preventing cross-contamination

A number of measures should be taken to help prevent cross-contamination of food with potentially harmful micro-organisms:

- Special care should be taken with cleaning after cutting up raw meat and before dicing vegetables, particularly if the vegetables are to be eaten raw or with minimal cooking.
- Use a different chopping board and utensils when preparing foods to be eaten raw and foods for cooking.
- Never place cooked food on plates that have held raw meat, poultry or seafood.
- Never use a tea towel as a hand towel or for cleaning surfaces.
- During food preparation, do not taste the food with the utensil used for stirring.^{13,20,21}

Thawing foods

The method chosen for thawing food should be the one that minimises the time the food is at a temperature that supports the growth of micro-organisms—for example, room temperature. Ready-to-eat frozen foods should be thawed in the refrigerator or under cold water in an airtight plastic wrapper or bag, the water being changed every 30 minutes.^{14,22} Foods can also be thawed in a microwave oven, using the defrost setting. When thawing raw meat, it is important that fluids produced during the thawing process do not contaminate other foods or containers and other utensils that might be used for other foods. Make sure that larger portions of raw meat, such as chickens and turkeys, are thawed completely before cooking. This might call for some forward planning, to allow sufficient time for thawing these meats before they are required; for example, if a turkey is thawed in the fridge it might take several days, depending on the bird's size. Follow the manufacturer's instructions for re-heating packaged ready-to-eat frozen foods (such as TV dinners). And be sure to check the information on pre-packaged foods to determine whether they need to be cooked or simply re-heated before being eaten.

Preparing fruits and vegetables

Fruits and vegetables should be washed thoroughly under running water before peeling and cutting. Special care should be taken with produce such as parsley and lettuce: they are harder to clean than smooth-skinned produce.

Marinating

Marinate raw foods in the refrigerator. The marinade can be used *during* cooking, but do not add it to the cooked dish or use it as a dressing if raw meat has been in it.

Cooking

Not all meat needs to be cooked thoroughly: steaks, whole fillet, chops and whole pieces of roast meat can be eaten rare. In contrast, rolled and/or stuffed meats, poultry, pork, sausages and mince should always be cooked all the way through, until the juices run clear when the meat is pierced.¹⁰

When using a microwave to cook, rotate and stir the food so that it cooks evenly. Cover it with a lid or plastic wrap so that the steam can aid thorough cooking. Food finishes cooking during standing time, and it is important to wait until the standing time has elapsed before checking that cooking is complete.²³

Never partially cook products then finish cooking them later.²³ Meat, fish and poultry must be cooked thoroughly; they can then be refrigerated and reheated later.

Cooling

Cooking of itself does not guarantee safety: some bacterial spores can survive several hours of cooking and later grow 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. To cool a large portion of food more quickly, divide it into smaller quantities or place it in shallow containers (5 centimetres deep) in the refrigerator.¹⁰ All leftovers should be placed in the refrigerator to cool as soon as possible and should be used within two or three days.²³

Reheating

When reheating food, heat it until it is 'steaming hot' throughout: this should destroy any vegetative cells of foodborne pathogens that may be present, but it will not destroy toxins. Bring soups, sauces and gravies to a rolling boil. Do not reheat food more than once.²³ When heating pre-prepared frozen or refrigerated dinners, follow the instructions on the packet.

Serving

It is essential to serve food safely: foodborne illness can result if food is not safely handled and served as soon as possible after it is cooked. Hands should be washed with soap and water and dried thoroughly using a clean towel or a paper towel, and the food should be served on clean plates. Never put cooked food on a plate that has held raw food^{14,15} or re-use utensils used during food

preparation.¹⁶ Unless foods are to be served immediately, they should be covered until ready to be eaten.

Freshly cooked food that is eaten straight away is safest; if foods cannot be consumed immediately, keep them cold (at 5°C or below) or hot (at 60°C or above). For buffets, food can be kept hot by using chafing dishes and warming trays. Cold food should be kept cold by keeping it in the refrigerator or in a cooler with ice until served; for buffets, it can be kept on ice.

Cleaning

All work surfaces, crockery, cutlery, cooking utensils and other equipment should be thoroughly cleaned to remove any food or other residue. This can usually be done by using warm water with detergent. After cleaning, utensils and work surfaces can be sanitised using hot water or chemical sanitisers (such as a mixture of bleach and water) if necessary. Utensils and other equipment should be thoroughly dry before they are re-used.

Foodborne bacteria readily persist in kitchen towels, sponges and cloths^{20,21}; wash and dry them often, and replace sponges regularly. Using paper towels can reduce the risk of cross-contamination because they are disposable and so cannot harbour and spread bacteria.¹⁶

For added protection, keep appliances such as microwave ovens, toasters, can openers, and blender and mixer blades free of food particles. After thorough cleaning, use a bleach solution to sanitise chopping blades and hard-to-clean areas. Keep benches, shelves and work surfaces free of food particles.

Cooking large quantities

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

Eating and food handling away from home

The foregoing guidelines are simple to follow when preparing food in the home, but additional precautions should be taken when travelling, at barbecues and picnics, and when eating in restaurants or having takeaway meals.

Travelling

Any raw foods can be contaminated if you are travelling through areas of poor sanitation. Of particular concern are salads, uncooked vegetables and fruit, unpasteurised milk and milk products, raw meat, shellfish, lightly cooked (runny) eggs, and foods containing raw egg. The basic rule for eating and drinking in an unfamiliar place is, 'Boil it, cook it, peel it, or throw it away'.

3. CARE FOR YOUR FOOD

Where possible, avoid street vendors: there is a high possibility that the foods they are selling have been prepared and stored with insufficient attention to safety procedures.⁸ Tapwater and ice cubes used in beverages can also contain pathogens: buy bottled water for drinking and for cleaning your teeth, and in eating establishments ask that drinks be served without ice. Buy canned or bottled soft drinks, or beer and wine, and check that the seals are intact.

Picnics

Picnic foods should be transported in a cooler with ice or ice packs. Only pack foods that have already been chilled: never pack warm foods in a cooler to cool them. Foods that do not need to be kept cool—for example, fruits, vegetables, chips and bread—should not be packed in the cooler.²⁴ Keep the temperature in the cooler as cold as possible by keeping it out of the sun and leaving the lid on. Store drinks in a separate cooler, to avoid repeated openings of the food cooler. If possible, replenish ice when it melts. When cold foods have been served, promptly return the remainder to the cooler. Leftover perishable foods should be discarded. If there is no soap and water available, use liquid sanitiser or disposable wet wipes to clean hands before preparing and eating food. This is especially important when handling raw foods, such as raw meat.

Barbecues

Barbecue foods should be transported in an Esky or cooler with ice or ice packs. Ensure that raw meats are packed properly—to prevent leakage and contamination of other foods in the Esky or cooler—either by wrapping well or packing them in a separate Esky or cooler.¹⁷ Before cooking, thoroughly clean all barbecue tools and surfaces where food will be placed. Keep all food (including marinating food) in the fridge or Esky until just before cooking it or taking it to the barbecue site. Ensure that the meat is fully defrosted, so that it cooks evenly on the barbecue. Use one set of utensils for raw meats and poultry and another set for the cooked food.²⁵ Never put cooked meat on plates that have previously held raw meat. Burgers, sausages, pork and poultry should be cooked until the juices run clear, and all foods should be eaten as soon as possible after cooking. Keep food hot on the side of the grill rack until people are ready to eat.²⁵

Restaurant and takeaway meals

Consumers have little control over the way food is prepared at restaurants and cafes, but some observations can be made to help make safer food choices.

If possible, check that raw and cooked foods are well separated. Make sure hot foods are served to you hot, not lukewarm. At buffet or self-service meals, check that hot food is stored in hot-food display cabinets or over burners and that cold food is displayed on ice or in special refrigerated cabinets. Each dish should have its own serving utensils and be replenished regularly. Foods should be covered by some type of guard or cover. Plates and cutlery should be clean and dry.²⁶ Takeaway meals should be eaten within two hours of purchase²⁴ or immediately put in the fridge and eaten within two days.²⁶

For high-risk groups, extra precautions can be taken:

- Ask that food be freshly prepared while you wait.
- Ask that sprouts not be added to food. Check sandwiches and salads bought in restaurants and from delicatessens.²⁷
- Avoid pre-prepared salads, such as those in salad bars.

What to do if you think you have suffered food poisoning?

If you think you have suffered food poisoning, seek treatment as necessary. Seek medical care immediately if you are pregnant, if you have a weakened immune system, or if symptoms persist or are severe—for example, bloody diarrhoea, excessive nausea and vomiting, or a high temperature.

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

If possible, preserve the evidence. Wrap the remaining portion of the food securely, label it 'DANGER' and freeze it. Save all the packaging materials and any identical unopened products. Record details of when the food was consumed and when the onset of symptoms occurred.²⁴

The last meal eaten is often blamed for causing illness, whereas it is often a food that has been eaten the previous day. It is useful if you can recall and write down the foods you ate in the last 48 hours.

RELATIONSHIP TO OTHER GUIDELINES

Eat plenty of vegetables, legumes and fruits

Section 1.1 closely relates to food hygiene and the purchase, transport, storage, preparation and cooking of vegetables and fruits. When these foods are stored correctly, their nutritional quality and storage life are maximised. Buy fresh vegetables and fruits that are 'firm', and make sure that canned and frozen varieties have complete, undamaged packaging.

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

Include milks, yoghurts, cheeses and/or alternatives (reduced-fat varieties should be chosen where possible)

Limit saturated fat and moderate total fat intake

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

Include lean meat, fish, poultry and/or alternatives

Illness caused by foodborne pathogenic bacteria is a serious public health problem, and all foods are potential vectors of pathogens. In Australia the risk of foodborne illness from primary food industries is managed across the food chain, with industry, government and consumers sharing responsibility for the delivery of microbiologically safe products. Nevertheless, some foods from the meat, fish, poultry and alternatives food group have been implicated in outbreaks of foodborne disease, and constant vigilance is required.

Prevent weight gain: be physically active and eat according to your energy needs

Research has shown that immune cells are responsive to the effects of acute exercise, in terms of both number and function. Regular physical activity can be beneficial for older people's immune system function and can enhance the body's ability to defend itself against foodborne illnesses.

CONCLUSION

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

EVIDENCE

Because of the nature of this guideline, there is no evidence of the kind required for rating according to the National Health and Medical Research Council's levels of evidence. It is not possible to conduct trials or case-control, cohort or experimental studies of the microbiological safety of foods. Information about safe practice comes from a basic understanding of food microbiology and human physiology and is based on a wide variety of evidence from laboratory studies and 'opportunistic' evidence from outbreaks of food poisoning.

REFERENCES

1. 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.
2. Australia New Zealand Communicable Diseases Network. Fortnightly notifiable diseases tables. *Communicable Diseases Intelligence*. <www.health.gov.au/pubhlth/cdi/cdifort.htm>, 11 January 1999.
3. Desmarchelier PM. Foodborne disease: emerging problems and solutions. *Med J Aust* 1996;165:668–71.
4. 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: ANZFA, 1999.
5. Kaferstin FK. Food safety: a commonly underestimated public health issue. *Wld Hlth Stats Quart* 1997;50:3–4.
6. Australia New Zealand Communicable Diseases Network. National Notifiable Diseases Surveillance System. *Communicable Diseases Intelligence*. <www.health.gov.au:80/pubhlth/cdi/nndss2.htm>, April 2001.
7. Hench CP, Simpkins SM. *Bugs at the banquet table: foodborne illness*. <www.nurseweek.com/ce/ce3240a.html>.
8. Bender JB, Smith KE, Hedberg C, Osterholm MT. Foodborne disease in the 21st century. What challenges await us? *Postgrad Med* 1999;106(2):109–12, 115–16, 119. <www.postgradmed.com/issues/1999/08_99/bender.htm>.
9. Cano RJ, Colome JS. Determinants of health and disease. In: *Essentials of Microbiology*. St Paul, MN: West Publishing Company, 1988.
10. Australia New Zealand Food Authority. *Safe food Australia: a guide to the Food Safety Standards*. 2nd edn. Canberra: ANZFA, 2001.
11. Fraser A. *Bacterial toxins*. National Food Safety database. <www.foodsafety.ufl.edu/consumer/nc/nc816a.htm>, 2001.
12. Sprenger RA. *Hygiene for management: a text for food hygiene courses*. South Yorkshire, UK: Highfield Publications, 1989.
13. Merry G. *Food poisoning prevention*. 2nd edn. Melbourne: Macmillan Education Australia, 1997.
14. Food Safety Campaign Group. *Food safety tips*. <www.safefood.net.au/food_august/tips/index.html>, 15 December 1998.
15. CSIRO. *Handling food in the home*. North Ryde: Food Science Australia, 1993.
16. US Federal Consumer Information Center. *How to help avoid foodborne illness in the home*. Pueblo, CO: FCIC, 1998. <www.pueblo.gsa.gov/press/nfcpubs/foodborn.txt>.

3. CARE FOR YOUR FOOD

17. NZ Ministry of Health. *Barbeque food safety stressed*. Media release. <www.moh.govt.nz/moh.nsf>, 1998.
18. Australia New Zealand Food Authority. *Date marking: user guide to Standard 1.2.5, Date Marking of Packaged Food*. Canberra: ANZFA, 2001.
19. Altekruze SF, Street DA, Fein SB, Levy AS. Consumer knowledge of foodborne microbial hazards and food-handling practices. *J Food Protect* 1995;59(3):287–94.
20. Abdussalan M, Kaferstein FK. Food safety in primary health care. *World Health Forum* 1994;15:393–9.
21. Department of Health and Community Services. *Food Premises Code*. 2nd edn. Melbourne: DHCS, 1996.
22. Brewer MS. *Food storage, food spoilage and foodborne illness*. Circular 1313. Chicago, IL: Department of Food Science and Human Nutrition, University of Illinois, 1991. <www.aces.uiuc.edu/~fshn/extension/food_storage.html>.
23. US Food Safety and Inspection Service. *Safe food to go*. Washington, DC: Department of Agriculture, 1997. <www.fsis.usda.gov/OA/pubs/foodto go.htm>.
24. Nix J. Questions and answers about foodborne illness and safe food handling procedures. North Carolina Cooperative Extension Service, 1998. <lenoir.ces.state.nc.us/staff/jnix/pubs/food.ill.html>.
25. *Food science: preparing food safely*. <foodsci.rutgers.edu/schaffner/factsheets/fs587.htm>, 10 December 1998.
26. Food Safety Victoria. *Food safety when eating out*. <[www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/\(Pages\)/Food_safety_when_eating_out?OpenDocument](http://www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/(Pages)/Food_safety_when_eating_out?OpenDocument)>.
27. US Food and Drug Administration. Consumers advised of risks associated with raw sprouts. *HHS News*. Washington, DC: Centre for Food Safety and Applied Nutrition, 1999. <vm.cfsan.fda.gov/~1rd/hhssprts.html>.

4 ENCOURAGE AND SUPPORT BREASTFEEDING

Colin Binns

TERMINOLOGY

Exclusive breastfeeding

Exclusive breastfeeding means that an infant is receiving only breastmilk, which includes expressed breastmilk and milk from a wet nurse. The infant might also receive medications and vitamins or minerals, as required.

Complementary food

Complementary food means any food—be it manufactured or locally prepared—that is suitable as a complement to breastmilk or infant formula when either becomes insufficient to satisfy an infant's nutritional requirements. Such food is also commonly called *weaning food* or *breastmilk supplement*.¹

Research methodology and data collection

A variety of methods are used to study and record breastfeeding rates. Reported studies use different sampling methods and may rely on mothers' memory of past events. Studies that use frequent interviews of a representative cohort and use standard definitions are more accurate.²

BACKGROUND

Breastfeeding is the normal and most appropriate method for feeding infants and is closely related to immediate and long-term health outcomes. Exclusive breastfeeding to the age of six months gives the best nutritional start to infants and is now recommended by a number of authorities.^{3–6} The World Health Organization reviewed breastfeeding duration and identified more than 3000 references <www.who.int/inf-pr-2001/en/note2001-07.html>. The WHO Expert Consultation then recommended exclusive breastfeeding for six months, then introduction of complementary foods and continued breastfeeding thereafter. It is recommended that breastfeeding continue until 12 months of age and thereafter as long as mutually desired.⁵ In many societies breastfeeding continues well beyond the age of 12 months, with benefit to both infant and mother.^{7,8}

If for any reason breastmilk is discontinued before 12 months of age, a commercial infant formula should be used—instead of cow's milk—as the main source of milk. Breastmilk from a healthy, well-nourished mother is adequate as

the sole source of nutrients for full-term infants from birth until about six months of life. Low-birthweight infants should have their nutritional needs assessed by a paediatrician.

Apart from their nutritional suitability, colostrum and mature human milk are hygienic and provide immunoglobulins and other anti-infective agents, which play a major role in protecting infants against infection and disease. Breastmilk also contains a number of unique growth factors⁹ and is a convenient, inexpensive food source posing no environmental cost. (See the Infant Feeding Guidelines for Health Workers, which are incorporated in the Dietary Guidelines for Children and Adolescents in Australia, for a more detailed discussion of breastfeeding.)

Current practices

Although the majority (80–90 per cent) of women in Australia commence breastfeeding, just under a third of them have introduced other foods or have stopped breastfeeding by three months.⁸ There is also evidence of considerable variation between socio-economic groups in terms of both the acceptance and the maintenance of breastfeeding in the Australian community: women in higher socio-economic groups are more likely to breastfeed.^{10,11} Support and encouragement from family members, friends and the whole community are required if breastfeeding rates and the duration of breastfeeding are to be maximised.

From a public health viewpoint, there is considerable room for improvement in both the rates and the duration of breastfeeding in Australia; such improvements offer benefits for maternal, infant and child health. An initiation rate in excess of 90 per cent, and 80 per cent of mothers breastfeeding at six months, are achievable goals in Australia. Of the developed countries, Norway consistently reports the highest breastfeeding rates, ones that Australia should strive to achieve:

- Ninety-two per cent of mothers are breastfeeding their child when it is three months of age.
- Eighty per cent are breastfeeding their child at six months.
- Forty per cent are still breastfeeding their child at 12 months.¹²

The advantages of breastfeeding continue beyond the six-month period, and Australians other than mothers can play an important part in making breastfeeding an easy and viable option. Encouragement and support—from a combination of hospitals and health centres, families, friends, social groups and places of work—will ensure that women can breastfeed successfully.

Breastfeeding is included in these Dietary Guidelines for Australian Adults in acknowledgment of the nutritional, health, social and economic benefits it provides for the Australian community and of the need for family and community support. Breastfeeding promotion should be combined with other health-promotion programs.¹³

Historically, breastfeeding or the use of a wet nurse was the only way to feed an infant. Rickets, scurvy and hypernatraemia were associated with early artificial feeding; knowledge of infant requirements was limited before the development of modern infant formula. In reviews of infant feeding in Australia, Hitchcock¹⁴ and Lund-Adams and Heywood¹⁵ describe the decline in breastfeeding rates in Australia and other developed countries that occurred during the 20th century.

Breastfeeding reached a low point in Australia in the 1960s; records from Victoria show that only 50–60 per cent of mothers were breastfeeding on discharge from hospital and only 21 per cent after three months.¹⁶ In the early 1970s breastfeeding rates started to rise again in Australia and comparable overseas countries, beginning in the higher socio-economic groups. By 1983 both the prevalence and the duration of breastfeeding in Australia were among the highest in the western world, with 85 per cent at discharge and 54–55 per cent three months later.¹⁷ Breastfeeding has remained around this level for the past two decades. In 1984–85 a joint survey in Western Australia and Tasmania indicated a continued trend to increasing breastfeeding rates and duration. During the preceding five years, prevalence rates at hospital discharge rose from 82 to 86 per cent in Western Australia and from 72 to 81 per cent in Tasmania. At six months after discharge 45 per cent of mothers in both states were still breastfeeding.¹⁸

In 1992–93 in Western Australia and in 1995–96 in Queensland, Scott et al.¹¹ conducted a survey that found a continuing trend to increased breastfeeding rates and duration in those states. In the preceding years the hospitals had a breastfeeding discharge rate of 82 per cent, and at six months 46 per cent of mothers were still breastfeeding. Women born in Australia or New Zealand were almost twice as likely to be breastfeeding at discharge compared with women born in other countries.

Donath and Amir¹⁰ analysed the data from the 1995 National Nutrition Survey and found that breastfeeding rates were 81.8 per cent on discharge from hospital and 57.1 per cent fully breastfed at three months. At six months, it is estimated that 18.6 per cent of babies are fully breastfed and 46.2 per cent fully or partially breastfed. At one year of age, 21.2 per cent of infants are receiving some breastmilk. Thus, in Australia at present fewer than 20 per cent are achieving the goal of being exclusively breastfed to age six months.

Australia has a long history of promoting and supporting breastfeeding in its public health policy. The importance of breastfeeding led to its inclusion in the Dietary Guidelines for Australians endorsed by National Health and Medical Research Council in June 1982. In 1981 Australia became a signatory to the WHO International Code of Marketing of Breast-milk Substitutes, the stated aim of which was:

... to contribute to the provision of safe and adequate nutrition for infants, by the protection and promotion of breastfeeding and by ensuring the proper use of breastmilk substitutes, when these are necessary, on the basis of adequate information and through appropriate marketing and distribution.¹⁴

The Infant Feeding Guidelines for Health Workers provide more information on the WHO Code and its implications for health workers. In 1987 the Nutrition Taskforce of the Better Health Commission set targets for the year 2000 of increasing the prevalence of breastfeeding at discharge from hospital to 95 per cent and increasing the proportion still breastfeeding at three months to 80 per cent.¹⁹ The rationale behind the targets was to continue the promotion of breastfeeding, so that rates in at-risk groups would increase, the average period of breastfeeding would be lengthened, and overall levels of breastfeeding would be maintained. With current knowledge of the benefits of breastfeeding and the health risks for infants not receiving breastmilk, extending the goal to 80 per cent breastfeeding at six months would be appropriate.

SCIENTIFIC BASIS

Breastfeeding physiology

Milk production and secretion are under endocrine and autocrine control.²⁰ When the infant suckles at the breast, mechano-receptors are stimulated, resulting in the release of oxytocin and prolactin into the blood, from the posterior and anterior pituitary respectively. Oxytocin stimulates the contraction of cells and secretion of milk from the alveolus; prolactin is responsible for milk production in the alveolus. The commonly termed *let-down reflex* can also be stimulated by seeing the infant or hearing its cries; it can be inhibited by stress such as pain or anxiety.²¹ Close mother–child contact immediately after birth helps to establish lactation, and frequent suckling or feeding on demand helps to maintain it.^{22,23} Milk synthesis is related to the rate at which the breast is emptied.²⁰

A review of early contact practices found, ‘Mothers should have contact with their babies as soon after birth and for as long as they wish. Interventions aimed at either delaying or speeding up the time of the first feed should be avoided’.²⁴ 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–child contact.

Breastmilk is uniquely suited to the needs of infants throughout the duration of lactation and provides all the nutrients required for at least the first six months of life. The composition of breastmilk is compromised only in mothers with severe malnutrition. Breastmilk is a living tissue that cannot be duplicated by any other means. It is very important for pre-term infants.

Colostrum, the secretion produced in the first few days after giving birth, provides all the nutrients, including water, required by the neonate. In composition, it differs from both transitional milk and mature milk, containing higher levels of protein, vitamin A and vitamin B₁₂ and less fat. It also contains lactoferrin, immunoglobulin A, enzymes, maternal antibodies, living cells—leukocytes, neutrophils and macrophages—and non-pathogenic bacteria, which

act in the gut of the newborn to limit the growth of pathogenic bacteria and viruses and to protect against illness.^{22,25}

The composition of this first secretion after birth gradually changes as lactation is established and production of milk begins in the breast tissue. By seven to fourteen days after birth, lactation should be established and the transition from colostrum to mature milk should be under way. The nutrient composition of mature expressed human milk shows variation in and between individuals—depending on maternal diet and the stage of lactation—although mean ranges are remarkably consistent for the species. The energy content is based on the fat, protein and carbohydrate levels and varies between 270 and 315 kilojoules (65–75 kcal) per 100 millilitres, largely as a result of variation in the fat content. Fat typically increases three- to four-fold during a single feed and also shows diurnal variation. It provides much of the energy and omega-3 and omega-6 long-chain polyunsaturated fatty acids; it also carries the fat-soluble vitamins A, D, E and K, as well as prostaglandins.^{22,25} This fat is typically better absorbed by the infant's gastrointestinal tract than the fat in cow's milk, and the lipase present increases the efficiency of absorption. Mature milk continues to provide immune factors and enzymes to the infant. (For details of milk composition see the Infant Feeding Guidelines for Health Workers.)

Breastmilk also provides all the major minerals and trace elements known to be essential for healthy full-term infants. Although the levels of some micronutrients appear to be low in comparison with other milks, the high bioavailability of these components in human milk ensures that no deficiencies occur. Infants' actual nutrient requirements are not precisely known, but the nutrients in human milk have obviously been adequate for infants for thousands of years. As a result, the composition of infant formula²⁶ and the recommended dietary intakes for groups of infants in Australia are based on the nutrient composition of human milk.²⁷ No infant formula can exactly mimic breastmilk, though. Breastmilk is constantly changing—throughout lactation and throughout the feed. In addition, constituents of breastmilk are still being discovered, and many of them cannot be replicated.²⁸

The health benefits of breastfeeding

There are many benefits to be gained from breastfeeding—for the infant, the mother and the community. These benefits are summarised in Box 4.1 (see also the Infant Feeding Guidelines for Health Workers for an expanded version). Costs to the community of not breastfeeding are also discussed later in this section. Increasingly, there is interest in the long-term effects of perinatal nutrition, commonly referred to as the *foetal origins of disease*, or *Barker hypothesis*. Inadequate or inappropriate foetal and early infant nutrition has been linked with subsequent chronic disease in adulthood.^{29,30}

Box 4.1 Health advantages of breastfeeding for infants and mothers^{31–34}**Infant**

- reduced incidence and duration of diarrhoeal illnesses
- protection against respiratory infection and reduced prevalence of asthma
- reduced occurrence of otitis media and recurrent otitis media
- possible protection against neonatal necrotising enterocolitis, bacteraemia, meningitis, botulism and urinary tract infection
- possible reduced risk of auto-immune disease, such as type 1 diabetes and inflammatory bowel disease
- reduced risk of developing cow's milk allergy
- possible reduced risk of adiposity later in childhood
- improved visual acuity and psychomotor development, which may be caused by polyunsaturated fatty acids in the milk, particularly decosahexaenoic acid
- higher IQ scores, which may be the result of factors present in the milk or of greater stimulation
- reduced malocclusion as a result of better jaw shape and development.

Mother

- promotion of maternal recovery from childbirth—accelerated uterine involution and reduced risk of haemorrhaging (thus reducing maternal mortality) and preservation of maternal haemoglobin stores through reduced blood loss, leading to improved iron status
- prolonged period of post-partum infertility, leading to increased spacing between pregnancies
- possible accelerated weight loss and return to pre-pregnancy body weight
- reduced risk of pre-menopausal breast cancer
- possible reduced risk of ovarian cancer
- possible improved bone mineralisation and thereby decreased risk of post-menopausal hip fracture.

Breastfeeding's protective effects against mortality are obviously of greater magnitude in countries with higher infant mortality rates. A pooled study by a WHO working group has illustrated just how valuable the protection conferred by breastfeeding can be, especially in developing countries, where these studies were undertaken (see Figure 4.1). The odds ratios for mortality from all causes and from infectious diseases show substantial benefit until the age of six months.

The Promotion of Breastfeeding Intervention Trial (PROBIT), undertaken in Belarus, is the largest cluster-randomised controlled trial of breastfeeding promotion and outcomes to have been published.³⁶ A total of 17 046 mother–infant pairs—consisting of full-term singleton infants weighing at least 2500 grams and their healthy mothers, who intended to breastfeed—were studied. The 31 hospitals involved were randomised to receive a health-promotion program based on the WHO–UNICEF Baby Friendly Hospital initiative. Compared with the control group, the infants from the intervention group were much more likely to be breastfed at 12 months and exclusively

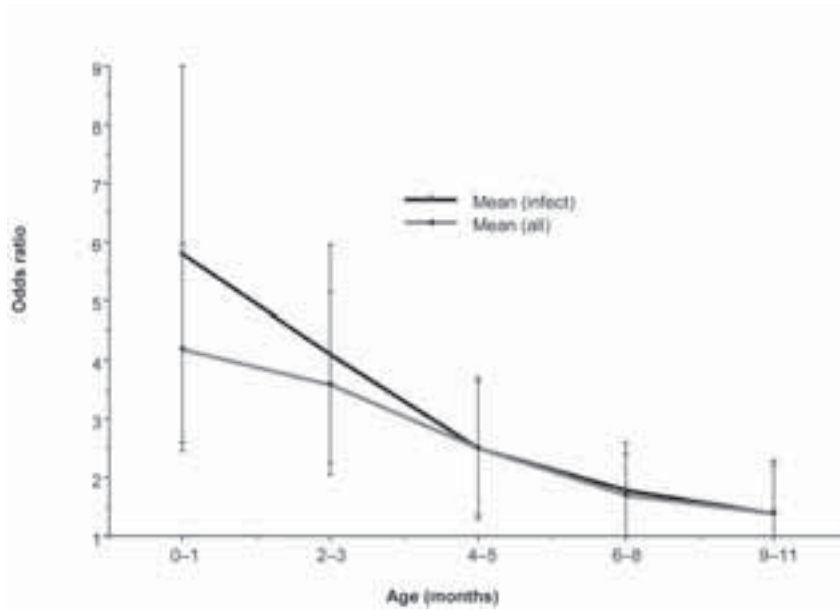


Figure 4.1 Odds ratio for not breastfeeding: all mortality and mortality from infection, by age³⁵

breastfed at three and six months. The intervention group also showed a significant reduction in the risk of one or more gastrointestinal tract infections (9.1% vs 13.2%; adjusted OR: 0.60; 95%CI: 0.40–0.91) and of atopic eczema (3.3% vs 6.3%; adjusted OR: 0.54; 95%CI: 0.31–0.95) but no significant reduction in episodes of respiratory tract infection.

The psychological and behavioural aspects of breastfeeding are also important. Breastfeeding is largely a pleasurable and positive interaction between mother and baby. The maternal hormones prolactin and oxytocin stimulate the development of maternal behaviour and bonding and also reduce the response to stress.³⁷

Breastmilk has also been shown to be the most suitable way of feeding low-birthweight infants in a trial of 108 infants. The unique properties of human milk promote an improved host defense and gastrointestinal function compared with infant formula. The benefits of improved health (less sepsis and necrotising enterocolitis) associated with feeding of fortified breastmilk outweighed the slower rate of growth observed, suggesting that feeding of fortified breastmilk should be actively promoted in premature infants.³⁸ Other types of human milk fortifier can enhance growth rates.³⁹

Pre-lacteal feeds are commonly given in some cultures. Best practice in Australia is to place the infant at the breast as soon as practicable after delivery and to offer it colostrum. Although respect is always due to other cultures, an infant has no need for any other solid or liquid for around six months.

Factors affecting the initiation and duration of breastfeeding

An extensive review of the literature has documented the demographic, social and economic factors associated with breastfeeding.⁴⁰ There is a higher prevalence and a longer duration of breastfeeding among mothers from higher socio-economic groups who are better educated, are older, and have previously breastfed.^{10,41–45} In the Australian studies, age was not found to increase breastfeeding in the rural sample.⁴⁶

In a longitudinal study, Scott, Aitken et al.¹¹ confirmed the known demographic factors (as just mentioned) that influence breastfeeding rates and duration. Among other factors that were found to have influenced a mother's decision to breastfeed and the duration of breastfeeding were the perceptions of partners and other family members, the mother's decision to breastfeed prior to pregnancy, and the mother's age and country of birth. Scott, Gowans et al.⁴⁷ also found that a mother who had more than one child, intended to return to work or study within six months, or had an infant in a special care nursery was less likely to breastfeed. In Australia, boys are breastfed for a shorter time than their sisters.⁴⁸ Further research is needed to understand the reasons for this.

Box 4.2 summarises the factors involved in the initiation and duration of breastfeeding in the two Australian studies and that by Landers et al.⁴⁶, which all used the same methodology (cohort studies with frequent interviews). The studies demonstrate the importance of family support, particularly from the father, and of early parental education about the benefits of breastfeeding. McIntyre et al.⁴⁹ analysed social support and found that social support for breastfeeding—as provided by fathers, grandmothers and the general community—in a low socio-economic area is not strong, particularly in relation to breastfeeding in public, combining breastfeeding and work, and appropriately managing breastfeeding.

Most women experience a number of other difficulties while breastfeeding (see the Infant Feeding Guidelines for Health Workers). If appropriate advice and support are not given, a mother may prematurely terminate breastfeeding. In studies in Australia and other developed countries, the main reason for termination cited by women is a perceived insufficient milk supply.²¹ The actual number of mothers who may be physiologically incapable of providing sufficient milk is, however, extremely low.⁵⁰ For the remainder of women who prematurely terminate breastfeeding, there are numerous causes—both biological and psychological—the majority of which are temporary and can be resolved with experienced advice or avoided by better preparation, hospital management or appropriate support. For example, rooming-in of infants while in hospital facilitates frequent feeding and thus the establishment of lactation.⁵¹ Breastfeeding is disrupted when the infant is housed away from the mother in the hospital, so the rooming-in option is offered by most hospitals today.⁵²

Box 4.2 Factors associated with the initiation and duration of breastfeeding in a rural population compared with an urban population^{11,46,47}

Factors associated with the decision to breastfeed

In a Rural area, breast feeding was more likely if:

- fathers preferred breastfeeding
- mothers were younger
- mothers decided pre-pregnancy to breastfeed
- mothers were primiparous.

Factors associated with risk of ceasing breastfeeding

In a Rural area risk of early cessation of breastfeeding was higher:

- in younger mothers
- in mothers who planned to breastfeed for less than two months
- where fathers did not prefer breastfeeding
- in mothers who did not decide to breastfeed before becoming pregnant
- in mothers whose infants received complementary formula feeds in hospital.

In an Urban area, breast feeding was more likely if:

- fathers preferred breastfeeding
- maternal grandmothers preferred breastfeeding
- mothers decided pre-pregnancy to breastfeed
- mothers were primiparous
- mothers were born in Australia, the United Kingdom, Asia, the Middle East or North Africa
- husbands were professional or administrators.

In an Urban area, risk of early cessation of breastfeeding was higher:

- in younger mothers
- in less educated mothers
- in mothers born in Australia, New Zealand or the United Kingdom compared with mothers born in the Middle East or Africa
- in mothers who planned to breastfeed for less than four months
- when maternal grandmothers were ambivalent or preferred formula feeding
- when mothers received conflicting advice on infant feeding while in hospital.

Note: See reference 46 for details of odds ratios and their confidence intervals.

Another difficulty is that modern hospital practice involves discharging mothers too early (often within 24–48 hours of delivery); this means hospital staff will have had insufficient time to help establish breastfeeding. A cohort study has demonstrated the importance of a supportive health system in successful breastfeeding.⁵³ A review of negative hospital practices—such as distribution of commercial publicity packs—has shown that these can have a detrimental effect on breastfeeding.⁵⁴ (See the Infant Feeding Guidelines for more details).

4. ENCOURAGE AND SUPPORT BREASTFEEDING

Frequent stimulation and frequent emptying are required to maintain breastmilk production, which is a very good reason for discouraging the use of complementary food or pacifiers (dummies). Feeding according to need—that is, feeding on demand—offers the best way of maintaining lactation, and if this is prevented by lack of facilities or social acceptance, lactation can be adversely affected.

The Australian Breastfeeding Association (formerly the Nursing Mothers Association of Australia) and other similar organisations have an important role to play within the health care system, providing the one-to-one support that is needed to overcome transient problems with lactation, particularly after hospital discharge.⁵⁵ The association also provides breastfeeding education classes, access to a local network of mothers and group discussion meetings, and a variety of publications dealing with all aspects of breastfeeding. In addition, it has a range of strategies designed to promote breastfeeding in the community, among them the following:

- community education sessions run by local groups all around Australia
- promotion of breastfeeding in local areas during Breastfeeding Awareness Month
- encouragement to the community to support breastfeeding through ‘Baby Care Room’ awards, ‘Mother Friendly Workplace’ accreditation, and ‘Breastfeeding Welcome Here’ stickers
- participation in consultations relating to policies affecting breastfeeding
- access for the community and health professionals to comprehensive and readily usable information and resources dealing with all aspects of human lactation through the Lactation Resource Centre. <www.abas.asn.au>

Breastfeeding and paid employment need not be mutually exclusive, although in some cases work may be a reason for women not to commence breastfeeding.^{21,56,57} A longitudinal study of 10 500 mothers found that planning to return to employment earlier than six weeks post-partum reduces the likelihood of initiating breastfeeding.⁵⁸ For successful lactation to continue after returning to work, supportive worksite health-promotion policies are required that provide education and facilitate either frequent feeding or frequent expression and storage of breastmilk, as required. A recent publication provides helpful information for mothers and employers (see *Balancing Breastfeeding and Work* <www.health.gov.au/hfs/pubhlth/strateg/brfeed/index>). Although it is not an ideal situation, many mothers who return to work are unable to breastfeed exclusively and, rather than using expressed breastmilk, carers use infant formulae for some feeds. Although mothers should be supported in this decision—any breastfeeding is better than none—every effort should be made to change the conditions of our society and so make exclusive breastfeeding possible for working mothers. The message should be that many mothers successfully combine breastfeeding and paid employment.

Breastfeeding and special circumstances

[This subject is discussed in detail in the Infant Feeding Guidelines for Health Workers.]

There are few contra-indications to breastfeeding. Most medications a mother might need are compatible with breastfeeding, but each drug should be specifically checked in a reliable reference⁵⁹ or with your GP.

Some disease situations—for example, if a mother has HIV or AIDS—can be absolute contra-indications.⁶⁰ Research is progressing rapidly, however, and this may change. Studies have now shown that exclusive breastfeeding to six months, combined with the use of antiretroviral drugs, substantially reduces the risk of HIV transmission.⁶¹

Exclusive breastfeeding for periods much beyond six months of age may result in under-nutrition and micro-nutrient deficiency.⁶ Supplementation with solid foods is necessary after about this time. In particular, beyond the age of six months additional sources of iron are required, usually from iron-fortified cereals and pureed foods containing meat.⁶² Mothers who are vegans or strict vegetarians can be at risk of vitamin B₁₂ and other nutrient deficiencies. (See Sections 3.2 and 3.3 in the Dietary Guidelines for Children and Adolescents for more information about this.)

Breastfeeding and community support

Community efforts associated with breastfeeding should focus on strategies and policies that will:

- influence the proportion of mothers who intend to breastfeed—the earlier the decision is made, before or during the pregnancy, the greater the likelihood of successful breastfeeding
- influence the intended duration of breastfeeding through education, example and support
- influence the attitudes and beliefs of the mother's support network, particularly the father
- provide antenatal and postnatal education about the day-to-day practicalities of breastfeeding
- promote breastfeeding as the social norm, with support and the provision of adequate facilities in social situations and the workplace
- include the father and/or other support people in as much of the antenatal preparation as possible
- provide post-discharge support for minor problems—from community services, the medical profession and support organisations
- enhance support for lactation in the workplace, to allow working mothers to continue to breastfeed.

In a meta-analysis of postnatal support for mothers in the community, Sikorski and Renfrew showed that ‘one more mother will breastfeed for two months if support is provided for nine women and one more woman will breastfeed exclusively if support is given to nine women’.⁶³

The media have an important role in portraying the importance of breastfeeding and in supporting it in the community as the norm. The Australian media often portray breastfeeding in a negative way.⁶⁴ In the United Kingdom it was found that television and press coverage routinely implies that breastfeeding is problematic, funny or embarrassing or associates it with ‘particular types of women’. On the other hand, bottle-feeding is seen as ‘largely normalised, socially integrated, associated with “ordinary” and “normal” families and represented as being problem-free’.⁶⁵

Provision of physical facilities that are adequate for breastfeeding is important; such facilities are often lacking in places mothers and their infants need to visit—for example, shopping centres and other public places.⁶⁶

Recent research has shown how important it is for fathers to encourage the initiation and duration of breastfeeding. The fathers of infants who were breastfed were found to have three particular characteristics:

- They had other children who had been breastfed.
- They attended antenatal classes.
- They discussed breastfeeding antenatally with their partner.

In general, however, fathers have poor knowledge about the practical aspects of breastfeeding¹¹, and it is important to include them in discussion about breastfeeding. Fathers also need to provide practical help—such as occupying other children and doing household chores—and emotional support for breastfeeding mothers. They should attend antenatal classes and learn about the nutritive and protective advantages of breastfeeding and some of the potential practical difficulties.

All health professionals need to constantly promote the benefits of breastfeeding. The benefits should be discussed with mothers (or potential mothers) at the earliest opportunity, such as the first antenatal visit. Health professionals should ensure that their patients know about the protective properties of breastmilk and the risks involved when infants do not receive it. They should also ensure that their activities do not discourage mothers from breastfeeding. In a program to promote ‘baby-friendly doctors’ offices’, workshops were conducted for office staff and resulted in positive changes in breastfeeding promotion. The changes were maintained at six and 12 months after the intervention.⁶⁷ In a controlled trial in an obstetrician’s office, the negative effect of exposure to formula-promotion materials was also demonstrated. Educational materials about infant feeding should unequivocally support breastfeeding as optimal nutrition for infants.⁶⁸

The Health Technology Assessment Program⁶⁹ has undertaken a systematic review of health promotion for breastfeeding (see Table 4.1). The reviewers

were very strict about inclusion criteria. For example, the introduction of paid maternity leave in Norway (one year on 80 per cent of pay or 46 weeks on full pay) has been associated with an increase in breastfeeding by working mothers. Similarly, introduction of the WHO International Code of Marketing of Breast-milk Substitutes has been associated with improved breastfeeding outcomes, yet no studies meeting the reviewers' criteria were found. Despite this, the reviewers found three types of intervention to be effective: small-group health education (antenatal); peer support programs (antenatal and postnatal); and one-to-one health education (low-income groups).⁶⁹

Table 4.1 Classification of breastfeeding health-promotion studies⁶⁹

Areas of health-promotion action^a	Types of intervention to promote the uptake of breastfeeding
Public policy	No studies identified
Supportive environments	No studies identified
Community action	Peer support activities
Development of personal skills	Health education and media programs
Reorientation of health services	Health sector initiatives
	Multi-faceted studies

a. Using the classification of the Ottawa Charter framework for health promotion studies, plus an additional category of multi-faceted studies.

A meta-analysis found that distribution of commercial information packs (with or without samples of formula) to mothers while in hospital reduces the number of women exclusively breastfeeding but does not affect the earlier termination of non-exclusive breastfeeding.⁵⁴

Costs to the community of not breastfeeding

There have been many studies of the economic benefits to be gained from breastfeeding. On the basis of longitudinal studies in Arizona and Scotland, it was estimated that for each 1000 infants who were never breastfed there is an excess of 2030 doctor visits and more than 200 inpatient days and 600 prescriptions compared with infants exclusively breastfed for more than three months.⁷¹ In addition, in the first year of life the total health care costs for infants who were not breastfed were estimated to be \$331 000 greater than those for breastfed infants. Ball and Bennet have proposed a comprehensive model for documenting the economic impact of breastfeeding; they showed that a breastfeeding education program reduced medical claims by \$1435 per infant and saved three days a year of maternal sick leave.⁷² (Note: costs are in 2001 US dollars and are not adjusted for inflation.)

4. ENCOURAGE AND SUPPORT BREASTFEEDING

In another study in the United States, Weimer⁷³ estimated that a minimum of \$3.6 billion would be saved if breastfeeding were increased from current US levels (64 per cent breastfed in hospital, 29 per cent breastfed at six months) to the targets recommended by the US Surgeon General (75 per cent and 50 per cent respectively). This figure of \$3.6 billion is probably an underestimation of the total savings because it represents cost savings from the treatment of only three childhood illnesses—otitis media, gastroenteritis and necrotising enterocolitis.

The health costs of weaning 30 per cent of infants onto infant formula by three months of age could be around \$290 million a year in Australia; this is based on an analysis of just five illnesses for which breastfeeding is proven to have protective effects.⁷⁴

The total value of breastfeeding to the community makes it one of the most cost-effective primary prevention measures available and well worth the support of the entire community.

RELATIONSHIP TO OTHER GUIDELINES

The Dietary Guidelines for Children and Adolescents in Australia, incorporating the Infant Feeding Guidelines for Health Workers, provide detailed advice on adolescent pregnancy and breastfeeding; indications for the introduction of solids; breastfeeding initiation and management; problems encountered in breastfeeding; health professionals' responsibilities under the WHO Code; and the use of infant formula.

CONCLUSION

Breastfeeding is very important for infant nutrition. Exclusive breastfeeding until around six months should be the aim for every infant. If that is not possible, mothers should be encouraged to breastfeed as much, and for as long, as they can. Breastfeeding beyond six months is of continuing value to baby and mother, although the maximum benefits of breastfeeding are in the earliest months of life.

Promotion of breastfeeding is an important public health strategy. Support and encouragement at all levels of the community are essential to maintaining and improving initiation rates and the duration of breastfeeding by Australian women, particularly those who are disadvantaged. Breastfeeding is included in the Dietary Guidelines for Australian Adults because it will contribute to the health of all Australians from birth.

EVIDENCE

There are three relevant Cochrane reviews^{24,54,63} which support this guideline and further National Technology Centre reviews of health education for breastfeeding.

For ethical reasons, it is not possible to do randomised control trials of breastfeeding.

There is Level III and Level IV for the following:

- the effect of early, as opposed to delayed, initiation of breastfeeding (reference 24)
- the effect of commercial hospital discharge packs for breastfeeding women (reference 54)
- community support for breastfeeding. (reference 63)
- the biological suitability of breastmilk (reference 5)
- exclusive breastfeeding to about six months (reference 6)
- the role of breastfeeding in prevention of infant mortality in less developed countries (reference 35)

There is Level III evidence for the effect of baby-friendly initiatives (reference 67); for the effect of prenatal advertising of formula (reference 68); and for factors associated with breastfeeding in women in Australia (references 11, 47 and 48).

REFERENCES

1. World Health Organization. *International Code of Marketing of Breast-milk Substitutes*. Geneva: WHO, 1981.
2. Scott JA, Binns CW. Breastfeeding in Perth—recent trends. *Aust J Publ Hlth* 1996;20(2):210–11.
3. Royal Australian College of General Practitioners Council. *RACGP breastfeeding position statement*. Melbourne: RACGP Council, 2000.
4. American Academy of Pediatrics. Breastfeeding and the use of human milk. *Pediatrics* 1997;100:1035–9.
5. Michaelsen KF, Weaver L, Branca F, Robertson A. *Feeding and nutrition of infants and young children: guidelines for the WHO European region, with emphasis on the former Soviet countries*. Copenhagen: WHO Regional Office for Europe, 2000.
6. World Health Organization. *The optimal duration of exclusive breastfeeding*. Geneva: WHO, 2001.
7. Dettwyler K. A time to wean. In: Stuart-Macadam P, Dettwyler K, eds. *Breastfeeding: biocultural perspectives*. New York: Aldine de Gruyter, 1995.
8. Binns CW. Food, sickness and death in children of the highlands of Papua New Guinea. *J Trop Pediatr Environ Child Hlth* 1976;22(1):9–11.
9. Binns CW. Infant feeding and growth. In: Ulijaszek SJ, Johnston FE, Preece MA, eds. *Cambridge encyclopedia of human growth and development*. Cambridge, UK: University of Cambridge Press, 1998.

4. ENCOURAGE AND SUPPORT BREASTFEEDING

10. Donath S, Amir LH. Rates of breast feeding in Australia, by state and socioeconomic status: evidence from the 1995 National Health Survey. *J Paediatr Child Hlth* 2000;36(2):164–8.
11. Scott JA, Aitkin I, Binns CW, Aroni RA. Factors associated with the duration of breastfeeding amongst women in Perth, Australia. *Acta Paediatr Scand* 1999;88(4):416–21.
12. Brundtland GH. *WHO Director-General's speech on infant feeding*. Geneva: WHO, 2000.
13. Redman S, Watkins J, Evans L, Lloyd D. Evaluation of an Australian intervention to encourage breast feeding in primiparous women. *Hlth Prom Int* 1995;10(2):101–13.
14. Hitchcock NE. Infant feeding in Australia: an historical perspective. Part 2: 1900–1988. *Aust J Nutr Diet* 1989;46(4):102–8.
15. Lund-Adams M, Heywood P. Breastfeeding in Australia. *Wld Rev Nutr Diet* 1995;78:74–113.
16. Lester I. *Australia's food and nutrition*. Canberra: Australian Government Publishing Service, 1994.
17. Palmer N. Breastfeeding: the Australian situation. *J Food Nutr* 1985;42:13–18.
18. Hitchcock NE, Coy JF. Infant feeding practices in Western Australia and Tasmania: a joint survey, 1984–1985. *Med J Aust* 1988;148:114–17.
19. English R (comp.). *Towards better nutrition for Australians: report of the Nutrition Taskforce of the Better Health Commission*. Canberra: Australian Government Publishing Service, 1987.
20. Hartmann P, Sherriff J, Kent J. Maternal nutrition and the regulation of milk synthesis. *Proc Nutr Soc* 1995;54(2):379–89.
21. Simopoulos AP, Dutra de Oliveira JE, Desai ID, eds. *Behavioral and metabolic aspects of breastfeeding*. Basel: Karger, 1995.
22. Department of Health and Social Security. *Present day practice in infant feeding: third report*. Report of a working party of the Panel on Child Nutrition, Committee on Medical Aspects of Food Policy. London: HMSO, 1988.
23. Ogle KS, Alfano MA. Common problems of initiating breast-feeding. The physician's role in encouraging success for the 'nursing' couple. *Postgrad Med* 1987;82(6):159–62, 165–7.
24. Renfrew MJ, Lang S, Woolridge MW. *Early versus delayed initiation of breastfeeding (Cochrane Review)*. Oxford: Cochrane Library, 2000.
25. Akre J, ed. Infant feeding: the physiological basis. *Bull Wld Hlth Org* 1989;67(suppl.):S1–S108.
26. National Health and Medical Research Council. *Food Standards Code*. Canberra: Australian Government Publishing Service, 1990.

27. National Health and Medical Research Council. *Recommended dietary intakes for use in Australia*. Canberra: Australian Government Publishing Service, 1991.
28. Newburg D, Street J. Bioactive materials in human milk: milk sugars sweeten the argument for breast-feeding. *Nutr Today* 1997;32(5):September–October.
29. Cox SE. The fetal origins hypothesis: an overview and implications. *Nutr Abs Revs* 1999;69(10):929–37.
30. Robinson R. The protective effect of childhood infections. *BMJ* 2001;322:376–7.
31. Michaelsen KF, Weaver L, Branca F, Robertson A. *Feeding and nutrition of infants and young children: guidelines for the WHO European region*. Copenhagen: World Health Organization, 2000.
32. Heinig MJ, Dewey KG. Health advantages of breastfeeding for infants: a critical review. *Nutr Res Rev* 1996;9:89–110.
33. Heinig MJ, Dewey KG. Health effects of breastfeeding for mothers: a critical review. *Nutr Res Rev* 1997;10:35–56.
34. Oddy W, Holt P, Sly P, Read A, Landau L, Stanley F et al. Association between breastfeeding and asthma in 6 year old children: findings of a prospective birth cohort study. *BMJ* 1999;319:815–19.
35. WHO Collaborative Study Team on the Role of Breastfeeding in the Prevention of Infant Mortality. Effect of breastfeeding on infant and child mortality due to infectious diseases in less developed countries: a pooled analysis. *Lancet* 2000;355(February):451–5.
36. Kramer M, Chalmers B, Hodnett E, Sevkovskaya E, Dzihovich I, Shapiro S et al. The Promotion of Breastfeeding Intervention Trial (PROBIT): a randomized trial in the Republic of Belarus. *JAMA* 2001;285(4):413–20.
37. Unvas-Moberg K, Eriksson M. Breastfeeding: physiological, endocrine and behavioural adaptations caused by oxytocin and local neurogenic activity in the nipple and mammary gland. *Acta Paediatr Scand* 1996;85:525–30.
38. Schanler R, Shulman R, Lau C. Feeding strategies for premature infants: beneficial outcomes of feeding fortified human milk versus preterm formula. *Pediatrics* 1999;103(6):1150–7.
39. Reis B, Hall R, Schanler R, Berseth C, Chan G, Ernst J et al. Enhanced growth of preterm infants fed a new powdered human milk fortifier: a randomized, controlled trial. *Pediatrics* 2000;106(3):581–8.
40. Scott JA, Binns CW. Factors associated with the initiation and duration of breast feeding. *Aust J Nut Diet* 1998;55(2):51–61.
41. Hartmann PE. Lactation and reproduction in Western Australian women. *J Reprod Med* 1987;32(7):543–7.

42. Hitchcock NE, Coy JF. The growth of healthy Australian infants in relation to infant feeding and social group. *Med J Aust* 1989;150:306–11.
43. Kocturk T, Zetterstrom R. Breastfeeding and its promotion. *Acta Paediatr Scand* 1988;183–90.
44. Milligan RA, Pugh LC, Bronner YL, Spatz DL, Brown LP. Breastfeeding duration among low income women. *J Midwif Women's Hlth* 2000;45(3):246–52.
45. Simopoulos AP, Grave GD. Review of research on the factors associated with choice and duration of infant feeding practice. *Pediatrics* 1984;74:S603–S614.
46. Landers M, Hughes RM, Graham K. *The Darling Downs Breastfeeding Study*. Toowoomba, Qld: Darling Downs Public Health Unit, 1998.
47. Scott JA, Gowans MC, Hughes RM, Binns CW. Psychosocial factors associated with breastfeeding at discharge and duration of breastfeeding amongst two populations of Australian women. *Proc Nut Soc Aust* 2000;25:240.
48. Scott JA, Binns CW. Breastfeeding: are boys missing out? *Birth* 1999;26:276–7.
49. McIntyre E, Hiller JE, Turnbull D. Attitudes towards infant feeding among adults in a low socioeconomic community: what social support is there for breastfeeding? *Breastfeeding Rev* 2001;9(1):13–24.
50. Baghurst KI. Infant feeding: public health perspectives. *Med J Aust* 1988;148(2):112–13.
51. Yamauchi Y, Yamanouchi I. The relationship between rooming-in/not rooming-in and breastfeeding variables. *Acta Paediatr Scand* 1990;79:1017–22.
52. Auerbach KG. Evidence-based care and the breastfeeding couple: key concerns. *J Midwif Women's Hlth* 2000;45(3):205–11.
53. Kuan L, Britto M, Decolongon J, Schoettker P, Atherton H, Kotagal U. Health system factors contributing to breastfeeding success. *Pediatrics* 1999;104(3):e28.
54. Donnelly A, Snowden HM, Renfrew MJ, Woolridge MW. *Commercial hospital discharge packs for breastfeeding women (Cochrane Review)*. Oxford: Cochrane Library, 2000.
55. Kyenkya-Isabirye M, Magalheas R. The mothers' support group role in the health care system. *Int J Gynecol Obstet* 1990;31(suppl. 1):S85–S90.
56. Barber-Madden R, Petschek MA, Pakter J. Breastfeeding and the working mother: barriers and intervention strategies. *J Publ Hlth Policy* 1987;8(4):531–41.
57. James J. Working and breastfeeding: a contemporary workplace dilemma. *Aust Coll Midwives* 1999;12(4):8–11.

58. Noble S, Team TAS. Maternal employment and the initiation of breastfeeding. *Acta Paediatr* 2001;90:423–8.
59. Ito S. Drug therapy for breast-feeding women. *N Engl J Med* 2000;343:118–28.
60. American Academy of Pediatrics. Human milk, breastfeeding, and transmission of human immunodeficiency virus in the United States. *Pediatrics* 1995;96:977–9.
61. Coutsooudis A. Promotion of exclusive breastfeeding in the face of the HIV pandemic. *Lancet* 2000;356:1620–1.
62. Calvo EB, Galindo AC, Aspres NB. Iron status in exclusively breastfed infants. *Pediatrics* 1992;90:375–9.
63. Sikorski J, Renfrew MJ. Support for breastfeeding mothers. In: *Cochrane database of systematic reviews*, 2000. <www.cochrane.org/cochrane/revabftr/mainindex.htm>.
64. Henderson AM. Mixed messages about the meanings of breast-feeding representations in the Australian press and popular magazines. *Midwifery* 1999;15(1):24–31.
65. Henderson L, Kitzinger J, Green J. Representing infant feeding: content analysis of British media portrayals of bottle feeding and breastfeeding. *BMJ* 2000;321(7270):1196–8.
66. McIntyre E, Turnbull D, Hiller JE. Breastfeeding in public places. *J Hum Lact* 1999;15(2):131–5.
67. Shariff F, Levitt C, Kaczorowski J, Wakefield J, Dawson H, Sheehan D et al. Workshop to implement the baby-friendly office initiative: effect on community physicians' offices. *Canad Fam Phys* 2000;46(May):1090–7.
68. Howard C, Howard F, Lawrence R, Andresen E, De Blicke E, Weitzman M. Office prenatal formula advertising and its effect on breast-feeding patterns. *Obstet Gynecol* 2000;95:296–303.
69. Fairbank L, O'Meara S, Renfrew MJ, Woolridge M, Sowden AJ, Lister-Sharp D. A systematic review to evaluate the effectiveness of interventions to promote the initiation of breastfeeding. *Hlth Technol Assess* 2000;4(25):1–171.
70. World Health Organization. Ottawa Charter for Health Promotion. *Health Promotion* 1987;1:3–4.
71. Ball T, Wright A. Health care costs of formula feeding in the first year of life. *Pediatrics* 1999;103:870–6.
72. Ball T, Bennett D. The economic impact of breastfeeding. *Pediatr Clin Nth Amer* 2001;48(1):253–69.
73. Weimer J. *The economic benefits of breastfeeding: a review and analysis*. Food Assistance and Nutrition Research Report no.13. Washington, DC: Department of Agriculture, 2001.
74. Smith J. Mother's milk, money and markets. *Ann Congress Perinatal Soc Aust NZ* 2001.

Special considerations

A THE NUTRITION OF ABORIGINAL AND TORRES STRAIT ISLANDER PEOPLES

Amanda Lee

CURRENT HEALTH AND NUTRITIONAL STATUS

Aboriginal and Torres Strait Islander peoples continue to suffer a much greater burden of ill-health—particularly nutrition-related chronic disease—compared with other Australians.^{1–3} In 1998 death rates among Indigenous Australians were at least three times greater than those for the total Australian population, and Indigenous people's life expectancy was around 20 years less.² Much of this poor health can be attributed to poor nutrition.¹

The prevalence of many nutrition-related conditions—such as type 2 diabetes, cardiovascular disease, renal disease, poor dental health, iron deficiency anaemia, and some forms of cancer—is disproportionately high among Indigenous Australians. Overweight and obesity tend to underpin the development of many of these conditions^{4,5}, which are discussed in detail in *Nutrition in Aboriginal and Torres Strait Islander Peoples: an information paper* (pp. 25–34 and 143–79) and *The Health and Welfare of Australia's Aboriginal and Torres Strait Islander Peoples*.²

The potential intergenerational effects of poor health and nutritional status have been well described.^{6–9} Low birthweight, failure to thrive and inappropriate child growth are serious concerns in Indigenous Australian communities.^{1,10–12} Diabetes in pregnancy also has potential intergenerational effects^{13,14} and is an additional concern.

Good maternal nutrition and healthy infant and childhood growth are fundamental to the achievement and maintenance of health throughout the life cycle.¹² Factors associated with maternal and infant health and childhood growth and nutrition are well documented in *Nutrition in Aboriginal and Torres Strait Islander Peoples*¹ (pp. 83–140).

High Indigenous infant mortality rates have been reported throughout Australia.^{1,2} Several studies have indicated that infection—especially of the gastrointestinal and respiratory tracts—associated with malnutrition and growth retardation was, and continues to be, the most common cause of death in children, particularly before the age of two.¹

Low Aboriginal birthweights have been documented, ranging from a prevalence of less than 7 per cent in Victoria to over 20 per cent in parts of northern Australia.¹ Several causal factors are implicated: maternal ill-health and malnutrition are significant determinants.¹⁵ Although the birthweight distribution for Aboriginal and Torres Strait Islander infants is similar to that for non-Indigenous infants, the infant mortality rate is 2.5 times higher.¹⁶

Growth retardation among Aboriginal infants after the age of four to six months has consistently been noted.¹ Relatively poor growth has also been shown to persist in older children, although overweight and obesity are becoming increasing concerns, particularly among Torres Strait Islanders.¹

National breastfeeding data for Indigenous Australians are limited.^{1,17} The 1995 National Health Survey found that Indigenous mothers breastfed for longer than non-Indigenous mothers.² The 1994 National Aboriginal and Torres Strait Islander Survey confirmed that Indigenous mothers of higher socio-economic status were more likely to breastfeed and to do so for longer than Indigenous mothers from lower socio-economic groups, but that Indigenous babies in rural areas were more likely to be breastfed for longer than six months compared with those in urban areas.^{12,18} One small study in Melbourne found that, although 98 per cent of Indigenous mothers initiated breastfeeding, only 50 per cent and 32 per cent were still breastfeeding at three and six months respectively.¹⁹

The nutritional and immunological effects of prolonged breastfeeding are particularly important in communities with a high prevalence of infectious diseases. However, introduction of appropriate solids at around six months is essential to ensure appropriate growth and development.¹

Vitamin and mineral status has been measured infrequently in Indigenous populations, but there have been some studies in a variety of groups and environments.¹ Samples have generally been small and have often been selected from vulnerable groups in the community—infants and pregnant and breastfeeding women. Quantitative comparison of the prevalence of vitamin deficiencies may be misleading since varying methods and ‘normal’ ranges have been used in these studies. Multiple deficiencies have frequently been described in the same subject, suggesting the generally poor nutritional status of such individuals, rather than a specific micro-nutrient problem. In particular, vitamin status (in relation to ascorbic acid, folate and beta-carotene) consistent with the very low contemporary dietary intakes of fruit and vegetables have often been described.²⁰

SOCIAL DETERMINANTS OF INDIGENOUS AUSTRALIANS’ HEALTH

A range of social determinants underpin the poor nutritional health status of Indigenous Australians. Among them are poverty; disrupted family and community cohesion; social marginalisation; stress; lower levels of education; unemployment; lack of control over circumstances; inadequate and overcrowded housing; inadequate sanitation, water supplies and hygiene; limited access to transport; and discrimination.^{2,21–26} The broader social environment affecting Indigenous Australians and their health status is well documented in the draft of the National Aboriginal and Torres Strait Islander Health Strategy.²¹

Cultural factors can have both positive and negative influences on health and nutritional status. The relationship between social environment and poor health

operates in both directions: poor health can increase the risks of deprivation through stigma and reduced earning capacity.²⁷

Indigenous Australians now make up 2.1 per cent of Australia's population.² Compared with the Australian population as a whole, Indigenous Australians are younger (a median age 20.1 years compared with 34.0 years), live mainly in south-eastern Australia, are less likely to have post-school education (11 per cent compared with 31 per cent), have higher unemployment rates (23 per cent compared with 9 per cent), have a lower financial income (for men, an average weekly income of \$189 compared with \$415; for women, \$190 compared with \$224), and are less likely to own homes (31 per cent compared with 71 per cent).^{2,28} In addition, Aboriginal and Torres Strait Islander Australians are more likely to live outside metropolitan areas than other Australians: 32 per cent of Indigenous Australians live in rural areas containing less than 1000 people, whereas only 15 per cent of non-Indigenous Australians do.²⁹ All Australians living in non-metropolitan areas experience higher mortality rates than those in metropolitan areas—15 per cent higher for men and 9 per cent higher for women in 1996.³⁰ The higher morbidity and mortality rates observed for rural areas are probably related more to limited occupational and educational opportunities, and the effect of this on income, than any special attributes of the physical environment. Poor access to medical services and limited lifestyle options are additional factors.²²

Many other social, economic, geographical, environmental and infrastructure issues and factors influence food choices and nutrition in Indigenous groups; these are discussed in detail in *Nutrition in Aboriginal and Torres Strait Islander Peoples*¹ (pp. 51–66). Examples are inadequate housing and food storage and preparation facilities.

In particular, people in rural and remote areas pay up to 50 per cent more for basic healthy foods than people living in urban and metropolitan areas.^{31–35} A recent Queensland study has assessed this disparity in terms of remoteness and accessibility, as measured by the Accessibility/Remoteness Index of Australia.³⁶ In contrast to expectations, the price of fruit and vegetables was less affected by remoteness/accessibility than other food groups; the prices of meat and meat alternatives and dairy foods were the most affected.³⁷ The cost of tobacco and takeaway food items was less affected by remoteness/accessibility than other items. Basic food items were less available in the more remote stores, as were fresh vegetables and fruits and better nutritional choices.³⁷

Among the factors contributing to the higher costs of foods in rural and remote areas are increased transport costs, high store overheads (including capital costs of building and maintaining long-term storage facilities and high accountancy costs) and greater wastage of stock.³⁸ Commitment and partnership across a range of sectors are necessary if the factors contributing to the high costs and limited supply of nutritious foods in rural and remote regions are to be tackled.^{1,37}

HEALTH ASPECTS OF TRADITIONAL DIETS AND LIFESTYLES

All the available evidence suggests that, traditionally, Indigenous Australian were fit and healthy.^{20,35,39–42}

Traditional dietary intakes and associated lifestyles have been reviewed in detail recently and are summarised in the following paragraphs. Additional information about food collection, preparation, storage and distribution is also available in *Nutrition in Aboriginal and Torres Strait Islander Peoples*.¹

The available information suggests that the traditional diet was generally low in energy density but high in nutrient density—high in protein, low in sugars, high in complex carbohydrate of low glycaemic index, and high in micro-nutrients. Even though the traditional Aboriginal diet contained a high proportion of animal foods, it would have been low in total fat, extremely low in saturated fat, and relatively high in polyunsaturated fatty acids (including the long-chain highly polyunsaturated fatty acids of both the omega-3 and omega-6 families) and hence protective against cardiovascular disease and related conditions.

The composition of most traditional vegetable foods is typical of that of uncultivated plants worldwide—high in fibre and relatively high in protein, with a generally low energy density.⁴³ The carbohydrate in most traditional plant foods is of low glycaemic index, producing lower glucose and insulin levels than similar western foods, and may be protective against diabetes.⁴⁴

Although some animal foods—such as witchetty grubs (*Cossidae* spp.) and green ants (*Oecophylla smaragdina*)—have a relatively high fat content, most native land animals are very lean.⁴³ Traditional meat foods have a much lower carcass fat content and intramuscular lipid content than meat from domesticated animals such as cattle and sheep.⁴⁰ Most carcass fat is stored in discrete ‘depots’ within the abdomen; these fat depots tend to be small and were traditionally shared by many people. However, marine animals such as turtle and dugong tend to be high in fat.^{43,45} Chewing the bones of land and marine animals¹ would have provided calcium.

Energy expenditure was high. Several accounts highlight the labour-intensive nature of collecting and preparing traditional foods.^{46–48} Food procurement and preparation by Aboriginal hunter-gatherers were energy-intensive processes that could involve sustained physical activity for many hours—for example, walking long distances; digging for tubers, reptiles, eggs, honey ants and witchetty grubs; chopping with a stone axe; winnowing and grinding seeds; digging pits for cooking large animals; and gathering wood for fires.^{20,41}

Children were traditionally breastfed until they were about 3 years old, the age of weaning depending on the arrival of another sibling. Solids were not introduced until teeth erupted.^{11,49} Responsibility for feeding tended to rest with the child, who was expected to express a desire for food and was fed on demand; older children had priority over the feeding of infants.¹¹

TRADITIONAL ABORIGINAL DIET AND FOOD PREFERENCES

Until European occupation, Aboriginal people successfully pursued a hunter-gatherer lifestyle across widely different geographical and climatic conditions. Survival depended on an intimate knowledge of the land, sources of water, and the seasonal cycle's effects on plant foods and game.¹

The traditional diet was characterised by diversity, and most early observers describe a varied and ample range of both animal and plant foods, even in dry regions. On a day-to-day basis, both the quantity and the quality of the food intake varied greatly; the usual pattern of subsistence was supplemented by 'feasts' when large game animals had been successfully hunted.

The most prized components of the Aboriginal hunter-gatherer diet were the relatively few energy-dense foods, such as depot fat and organ meats. Among other favoured foods were those with a high fat content—for example, witchetty grubs and marine mammals. Traditional diets were generally low in sugars, although sweetness was highly valued and provided by honey ants (*Melophorus inflatus*), the honey of the native bee, blossoms (for example, *Grevillea* spp.), lerp (a secretion from the insect *Psylla*, which lives on the leaves of eucalypts) and gums.^{20,41,50}

Traditional Torres Strait Islander diet and food preferences

Torres Strait Islanders were marine hunters, but they cultivated garden foods and gathered wild foods to varying degrees, depending on the local habitat.⁵¹ Some garden foods were stored and preserved. Turtle and dugong occupied a particular place in Torres Strait Islanders' cultural life; the fat content of these animals was considered a principal indicator of meat quality, and the fat itself was particularly prized.⁵²

CONTEMPORARY DIET

The process of acculturation from a traditional to a contemporary diet and lifestyle—including the effects of social, political and environmental factors—has been described in detail.^{1,20} With the transition from a traditional hunter-gatherer lifestyle to a settled westernised existence, Aboriginal and Torres Strait Islander people's diet has generally changed from a varied, nutrient-dense diet to an energy-dense diet that is high in fat and refined sugars (see Table A.1).

The limited data available support the notion that contemporary Indigenous diets tend to reflect the dietary intake of wider Australia, both during the depression years of the 1930s⁵³ and during colonial times⁵⁴, when food supply, transport, storage and costs were matters of general concern. The available dietary studies were recently reviewed in Chapter 3 of *Nutrition in Aboriginal and Torres Strait Islander Peoples*.¹

Table A.1 **Characteristics of hunter–gatherer and western lifestyles**^{20,41}

Criterion	Hunter–gatherer lifestyle	Western lifestyle
Physical activity level	High	Low
Principle characteristics of diet		
Energy density	Low	High
Energy intake	Usually adequate	Excessive
Nutrient density	High	Low
Nutrient composition of diet		
Protein	High	Low–moderate
Animal	High	Moderate
Vegetable	Low–moderate	Low
Carbohydrate	Moderate (slowly digested)	High (rapidly digested)
Complex carbohydrate	Moderate	Moderate
Simple carbohydrate	Usually low (honey)	High (sucrose)
Dietary fibre	High	Low
Fat	Low	High
Alcohol	Not available	Available
Sodium:potassium ratio	Low	High

Urban Aboriginal communities

Only limited quantitative dietary data are available pertaining to Aboriginal Australians' diet in the urban setting.⁵⁵ Because of methodological difficulties, individual dietary studies have tended to focus on qualitative and semi-quantitative assessment of the diet and to reflect dietary patterns and preferences, rather than actual, habitual intake.⁵⁶ A comparison of the food habits of Aboriginal and non-Indigenous Australians in a city and a country town showed that in both localities Aboriginal groups consumed takeaway meals and added salt more often than their non-Indigenous counterparts.⁵⁶ Twenty-four-hour dietary recall data from a small number of individuals suggested little difference between the dietary intakes of Aboriginal and non-Indigenous Australians in a country town, but the data have not been validated⁵⁷ and highlight some methodological shortcomings.⁵⁸

Remote Aboriginal communities

A study using the 'store-turnover' method in remote Aboriginal communities in the Northern Territory showed that sugar, flour, bread and meat provided more than half the apparent total energy intake. Fatty meats contributed nearly 40 per cent of the total fat intake in northern coastal communities and over 60 per cent in central desert communities. In both regions, white sugar per se contributed approximately 60 per cent of all sugars consumed.⁵⁹ Compared with national data

on apparent consumption in Australia, intakes of sugar, white flour and sweetened carbonated beverages were much higher in Aboriginal communities in the Northern Territory and intakes of wholemeal bread, fruit and vegetables were much lower (see Table A.2).⁵⁹

Table A.2 Apparent mean consumption (kg/capita/yr) of selected foods in Aboriginal communities compared with national data^{59,60}

(kg per person per year)			
Aboriginal communities			
Food	Central desert (n = 3)	Northern coastal (n = 3)	Australian data
Flour (white)	37.6	44.4	n.a.
Bread (all)	34.1	30.5	45.5
Beef and veal	51.6	25.8	41.4
Poultry	22.3	19.7	23.0
Lamb	22.8	3.3	16.8
Fish	—	4.8	4.0
Fruits	33.2	17.6	106.9
Vegetables	24.3	19.6	136.2
Sugar	54.1	50.3	8.2
Carbonated beverages	67.9	224	673
Fruit juice	48.3	12.8	n.a.
Tinned meat	9.4	10.1	n.a.
Pie/pasty	9.6	15.1	n.a.
Snack foods (e.g. potato crisps)	1.8	2.7	n.a.

— Zero.

n.a. Not available.

Note: 'Bread' includes flour used in bread-making.

Nutritional analysis revealed the average diet of Aboriginal Australians in remote areas was high in energy and sugar (more than three times the Australian recommended dietary intake), moderately high in fat (particularly saturated fat), and relatively low in complex carbohydrate, dietary fibre and nutrient density; these results support qualitative and semi-quantitative dietary assessment.^{61–63} Intakes of calcium and zinc and of some vitamins (vitamin B₂, vitamin E, beta-carotene and folic acid) appear low⁶⁴; these results have been confirmed by subsequent studies in different communities.^{65–67}

In contrast, very low energy intakes were described in one anthropological study in remote Aboriginal outstation communities in north-east Arnhem Land.⁶⁸ The energy intake of all subjects was approximately 50 per cent of the Australian recommended dietary intake, and the vitamin C, magnesium and calcium intakes

were low, as was retinol activity. Where traditional bushfoods (predominantly of animal origin) were consumed, zinc and iron intakes were higher and iron intake was equal to or above the recommended intake. Low intakes of fruit and vegetables were also described in this study.⁶⁸

Dietary intake has been shown to vary in close association with the income cycle in remote Aboriginal communities.⁶² Meat and vegetables (mainly as stew) and fruits were included in the diet after pay day but were usually absent for at least several days before the next pay day. A staple diet of bread or damper has often been described.^{62,69}

Even in remote traditionally oriented outstations, foods bought at the store accounted for most of the energy intake, while traditional bush foods provided the greatest proportion of protein intake.^{48,70,71} All the available studies show that flour, sugar, sweets and fats provided much of the energy intake from store-bought foods. Animal foods—particularly those high in fat, such as lizard—provided most of the energy from the bush. In general, dietary patterns in these small outstation communities are meat-oriented.

Torres Strait communities

In Torres Strait communities—and also in a few other island communities, such as the Tiwi community—marine foods continue to make substantial contributions to the diet. Men, women and children are involved in different aspects of gleaning, fishing and hunting. Torres Strait Islanders living on three outer islands were estimated to consume between 191 and 450 grams per person per day of seafood (including turtle and dugong), which is considerably more than the Japanese seafood intake (102 grams per person per day).⁴⁵ Concerns have, however, been raised about the heavy metal content in the organ meat of dugong, and particularly turtle, and the potentially negative effects of this on health.⁷² Production of traditional garden staple continues to be important for some ceremonial purposes.³¹

A study using the store-turnover method⁵⁹ was undertaken in a small island community in Torres Strait. More than half the energy in the diet was found to come from white flour, white rice, tinned meat and vegetable oil. The amount of fruit and vegetables available through the store was low: the fruit available per person was about one-sixth of the recommended amount and vegetables about one-third. People who depended on store foods would thus not be able to meet their needs for vitamins A, C and E and folic acid.³¹

CONTEMPORARY USE OF TRADITIONAL FOODS

It had been suggested that in the short term, after establishment of 'new' settlements or outstations, there may be an increase in yields of traditional foods due to high initial availability and the use of western technology.^{62,71,73} In some areas introduced feral animals such as rabbits and buffalo have also been

popular.^{71,74} But the longer term effect appears to be a reduction in the availability of traditional foods for several reasons, among them the following:

- environmental degradation caused by stock and feral animals
- introduction of exotic plant species
- the increasing incidence of hot, destructive bush fires as result of poor land management practices
- restricted access to some areas of land
- depletion of resources and population pressure around permanent settlements
- high costs associated with the acquisition and maintenance of equipment, firearms, vehicles and fuel
- changing demographic patterns
- cultural loss from generation to generation.^{75,76}

Contrary to some expectations, except for during some seasons in very remote areas^{48,68,70,71} the actual dietary intake of traditional foods has been found to be relatively low where it has been measured on mainland Australia.^{69,77} Bush foods contribute only a small proportion of nutrients in many areas.⁶⁴ A study of a northern coastal Aboriginal community found that an average of less than 15 per cent of the population sought traditional foods on at least three days a fortnight throughout the year. It was estimated that the proportion of total energy intake derived from bush foods averaged over the population would be less than 8 per cent during the dry season and less than half this during the peak of the wet season.⁷⁷

In the 1994 National Aboriginal and Torres Strait Islander Survey 10 per cent of respondents aged over 14 years reported spending more than one hour a week hunting or foraging for traditional foods and, of these, more than half reported spending more than five hours a week doing so.¹⁸ The rate of return for this effort is, however, unknown. Even though the actual intake of traditional foods is low, traditional foods are still popular and culturally important for Aboriginal and Torres Strait Islander peoples.^{31,78}

THE NATIONAL ABORIGINAL AND TORRES STRAIT ISLANDER NUTRITION STRATEGY AND ACTION PLAN

The National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan, NATSINSAP, provides a framework for national action to bring about an improvement in the nutritional status of Aboriginal and Torres Strait Islander peoples.¹² It was developed following wide-ranging consultations by a working group (with broad Indigenous representation) as a key component of *Eat Well Australia*.⁷⁹ The seven primary action areas in the plan are:

- food supply in remote and rural communities
- food security and socio-economic status

- family-focused nutrition promotion, involving resourcing programs, communicating and disseminating ‘good practice’
- nutrition in urban areas
- the environment and household infrastructure
- the Aboriginal and Torres Strait Islander nutrition workforce
- national food and nutrition information systems.¹²

DIETARY GUIDELINES FOR AUSTRALIA’S INDIGENOUS PEOPLES

The Dietary Guidelines for Australian Adults; the Dietary Guidelines for Children and Adolescents in Australia, incorporating the Infant Feeding Guidelines for Health Workers; and the Dietary Guidelines for Older Australians are pertinent to Indigenous Australians. Two other recommendations are, however, also important:

- Choose store foods that are most like traditional bush foods.
- Enjoy traditional bush foods whenever possible.

Rationale

Indigenous people can select nutritious food by choosing those store foods that are most like traditional bush foods—for example, fresh plant foods, wholegrain cereal foods, seafoods, and lean meat and poultry. With the exception of the dairy food group, this approach is consistent with the general Australian dietary guidelines.

All the available evidence suggests that, in terms of health and wellbeing, Indigenous Australians would benefit from closer adherence to the general Australian dietary guidelines.^{12,64,66,80–84} In particular, increased consumption of vegetables and fruits could be expected to improve the health and nutritional status of this population group. Lactose intolerance after the age of 3 to 5 years may, however, be problematic in some areas or individuals^{85,86} and may affect consumption of lactose-containing dairy foods. Alternative calcium sources such as low-lactose dairy foods (matured cheese, yoghurt), chewing meat and fish bones, and consumption of small, soft fish bones (for example, in tinned salmon) are recommended in these cases.

Consumption of traditional bush foods should also be supported wherever possible, although this may be a nutritional issue where high-fat marine animal foods are hunted in large numbers using modern procurement and distribution methods.⁴⁵ In addition, there may be a problem with high levels of heavy metals in the organ meat of turtle and dugong.⁷² Several other environmental factors might also need to be considered within the framework of sustainability.

It is particularly 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.

Where non-Indigenous Australians have sufficient understanding of the traditional Indigenous food supply, the additional Indigenous guidelines may also be useful in a wider context.

REFERENCES

1. National Health and Medical Research Council. *Nutrition in Aboriginal and Torres Strait Islander peoples: an information paper*. Canberra: NHMRC, 2000.
2. 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. Canberra: ABS, 1999.
3. McClelland A, Pirkis J, Willcox S. *Enough to make you sick: how income and environment affect health*. Melbourne: National Health Strategy Unit, 1992.
4. Jung RT. Obesity as a disease. *Br Med Bull* 1997;53:307–21.
5. O'Dea K. Body fat distribution and health outcome in Australian Aborigines. *Proc Nut Soc Aust* 1987;12:56–65.
6. Barker DJ. Fetal origins of cardiovascular disease. *Ann Med* 1999;31(suppl. 1):3–6.
7. Barker D. The fetal origins of type 2 diabetes mellitus. *Ann Int Med* 1999;130:322–3.
8. Barker D. *Mothers and babies and health in later life*. 2nd edn. Edinburgh: Churchill Livingstone, 1998.
9. Wadsworth M. Early life. In: Marmot M, Wilkinson R, eds. *Social determinants of health*. New York: Oxford University Press, 1999.
10. Ruben AR, Walker AC. Malnutrition among rural Aboriginal children in the Top End of the Northern Territory. *Med J Aust* 1995;162:400–3.
11. Hamilton A. *Nature and nurture*. Canberra: Australian Institute of Aboriginal Studies, 1981.
12. Strategic Inter-Governmental Nutrition Alliance. *National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan 2000–2010*. Canberra: National Public Health Partnership, 2001.
13. Pettitt DJ, Aleck KA, Baird HR, Carraher MJ, Bennett PH, Knowler WC. Congenital susceptibility to NIDDM: role of the intrauterine environment. *Diabetes* 1988;37(5):622–8.
14. Silverman BL, Metzger BE, Cho NH, Loeb CA. Impaired glucose tolerance in adolescent offspring of diabetic mothers: relationship to fetal hyperinsulinism. *Diab Care* 1995;18(5):611–17.
15. Sayers S, Powers J. Risk factors for Aboriginal low birthweight, intrauterine growth retardation and preterm birth in the Darwin Health Region. *Aust NZ J Publ Hlth* 1997;21:524–30.

16. Coory M. Is birthweight an appropriate outcome measure for Torres Strait Islander babies? *Aust NZ J Publ Hlth* 2000;24:60–3.
17. Engeler T, McDonald M, Miller M, Groos A, Black M, Leonard D. *Review of current interventions and identification of best practice currently used by community based Aboriginal and Torres Strait Islander health service providers in promoting and supporting breast feeding and appropriate infant nutrition*, Canberra: Office for Aboriginal and Torres Strait Islander Health Services, 1998.
18. Australian Bureau of Statistics. *National Aboriginal and Torres Strait Islander Survey, 1994: health of Indigenous Australians*. Cat. no. 4395.0. Canberra: ABS, 1996.
19. Holmes W, Thorpe L, Phillips J. Influences on infant-feeding beliefs and practices in an urban Aboriginal community. *Aust NZ J Publ Hlth* 1990;21:504–10.
20. Lee A. Transition of Australian Aboriginal diet and nutritional health. *Wld Rev Nutr Diet* 1996;79:1–52.
21. National Aboriginal and Torres Strait Islander Health Council. *The National Aboriginal and Torres Strait Islander Health Strategy*, Draft for discussion. Canberra: NATSIHC, 2001.
22. Queensland Health. *Social determinants of health: the role of public health services*. Brisbane: Queensland Health, 2001.
23. McClelland A, Scotton R. Poverty and health. In: Fincher R, Nieuwenhuysen J, eds. *Australian poverty—then and now*. Melbourne: Melbourne University Press, 1998.
24. National Health and Medical Research Council. *Promoting the health of Indigenous Australians: a review of infrastructure support for Aboriginal and Torres Strait Islander health advancement*. Canberra: NHMRC, 1997.
25. Altman J, Hunter B. Indigenous poverty. In: Fincher R, Nieuwenhuysen J, eds. *Australian poverty—then and now*. Melbourne: Melbourne University Press, 1998.
26. Glover J, Woollacott T. *A social health atlas of Australia*. Vol. 1. *South Australia*. 2nd edn. Adelaide: Public Health Information Development Unit, University of Adelaide, 1999.
27. Marmot M. Introduction. In: Marmot M, Wilkinson R, eds. *Social determinants of health*. New York: Oxford University Press, 1999.
28. Australian Bureau of Statistics. *Australia now. Aboriginal and Torres Strait Islander Australians: a statistical profile from the 1996 Census*. Cat. no. 1301.0. Canberra: ABS, 2000.
29. Humphreys J, Mathews-Cowey S, Rolley, F. *Health service frameworks for small rural and remote communities: issues and options*. Armidale, NSW: Department of Geography and Planning, University of New England, 1996.

30. Australian Institute of Health and Welfare. *Australia's health*. Canberra: AIHW, 1996.
31. Leonard D, Beilin R, Moran M. Which way kaikai blo umi? Food and nutrition in the Torres Strait. *Aust J Publ Hlth* 1995;19:589–95.
32. Beaumont S. *Tasmanian Food Price, Availability and Quality Survey*. Hobart: Community Nutrition Unit, Tasmanian Department of Community and Health Services, 1998.
33. Bowcock R. *1998 Kimberley Market Basket Survey*. Derby: Kimberley Public Health, 1999.
34. 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 Barkley district: April and May 1998*. Darwin: Northern Territory Health Services, 1998.
35. Sullivan H, Gracey M, Hevron V. Food costs and nutrition of Aborigines in remote areas of northern Australia. *Med J Aust* 1987;147:334–7.
36. Department of Health and Aged Care & University of Adelaide. *Measuring remoteness: Accessibility/Remoteness Index of Australia*. Adelaide: University of Adelaide, 1999.
37. Queensland Health. *The Healthy Food Access Basket Survey 2000*. Brisbane: Public Health Services, Queensland Health, 2001.
38. McMillan S. Food and nutrition policy issues in remote Aboriginal communities: lessons from Arnhem Land. *Aust J Publ Hlth* 1991;15:281–5.
39. Coyne T, Darnton-Hill I. Australian Aborigines' nutrition and changing disease patterns. *New Doctor* 1979;12:32–7.
40. Naughton J, O'Dea K, Sinclair A. Animal foods in traditional Aboriginal diets: polyunsaturated and low in fat. *Lipids* 1986;21:684–90.
41. O'Dea K. Traditional diet and food preferences of Australian Aboriginal hunter–gatherers. *Phil Trans Roy Soc Lond. Series B. Biol Sci* 1991;334:233–41.
42. Needeniya J, Smith A, Carter P. *Food supply in rural South Australia: a survey on food cost, quality and variety*. Adelaide: Department of Human Services, 2000.
43. Brand Miller JC, James KW, Maggiore PMA. *Tables of composition of Australian Aboriginal foods*. Canberra: Aboriginal Studies Press, 1993.
44. Brand Miller JC, Holt SHA. Australian Aboriginal plant foods: a consideration of their nutritional composition and health implications. *Nut Res Rev* 1998;11:5–23.
45. Johannes R, Macfarlane J. *Traditional fishing in the Torres Strait Islands*. Hobart: CSIRO Division of Fisheries, 1991.

46. Spencer B, Gillen F. *The native tribes of central Australia*. London: Macmillan, 1899.
47. Tindale NB. *Aboriginal tribes of Australia*. Canberra: Australian National University Press, 1974.
48. Devitt J. Contemporary Aboriginal women and subsistence in remote, arid Australia. PhD thesis. Brisbane: Department of Anthropology and Sociology, University of Queensland, 1988.
49. McArthur M. *Food consumption and dietary levels of the Aborigines at the settlements*. In: Mountford C, ed. *Records of the American–Australian scientific expedition to Arnhem Land*. Vol. 2. *Anthropology and nutrition*. Melbourne: Melbourne University Press, 1960.
50. Devitt J. Traditional Aboriginal preferences in a changed context. *Central Australian Rural Practitioners' Association Newsletter* 1991;14:38–41.
51. Beckett, J. *Torres Strait Islanders' custom and colonialism*, Cambridge, UK: Cambridge University Press, 1987:26–9.
52. Nietschmann B, Nietschmann J. Good dugong, bad dugong; bad turtle, good turtle. *Natural History* 1981;90(5):54.
53. Australian Bureau of Statistics. *Apparent consumption of foodstuffs and nutrients, Australia, 1998–99*. Cat. no. 4306.0. Canberra: ABS, 2000.
54. Walker RB, Roberts DCK. Colonial food habits. In: Truswell AS, Wahlqvist ML, eds. *Food habits in Australia*. Proceedings of first Deakin/Sydney Universities symposium on Australian nutrition, Melbourne: Deakin University, 1988:40–59.
55. Butlin A, Cashel K, Lee A, Taylor V. *Food and nutrition programs for Aboriginal and Torres Strait Islander peoples*. Canberra: Office for Aboriginal and Torres Strait Islander Health Services, 1997.
56. Guest CS, O'Dea K. Diabetes in Aborigines and other Australian populations. *Aust J Publ Hlth* 1992;16:340–9.
57. Sibthorpe B. All our people are dying: diet and stress in an urban Aboriginal Community. PhD thesis. Canberra: Australian National University, 1988.
58. Lee A, Smith A, Bryce S. Measuring dietary intake in remote Australian Aboriginal communities. *Ecol Food Nutr* 1995;34:19–31.
59. Lee A, O'Dea K, Mathews J. Apparent dietary intake in remote Aboriginal communities. *Aust J Publ Hlth* 1994;18:190–7.
60. Australian Bureau of Statistics. *Apparent consumption of foodstuffs and nutrients, Australia, 1985–86*. Canberra: ABS, 1987.
61. Hitchcock NE, Gracey M. Dietary patterns in a rural Aboriginal community in south-west Australia. *Med J Aust* 1975;2(suppl.):12–16.

62. Cutter T. Nutrition and food habits of the central Australian Aboriginal. In: Hetzel BS, Frith HJ, eds. *The nutrition of Aborigines in relation to the ecosystem of central Australia*. Melbourne: CSIRO, 1978:63–72.
63. Coles-Rutishauser IHE. Growing up in Western Australia: if you are Aboriginal. *Proc Nut Soc Aust* 1979;4:27–32.
64. Lee A, Bailey A, Yarmirr D, O'Dea K, Mathews JD. Survival tucker: improved diet and health indicators in an Aboriginal community. *Aust J Publ Hlth* 1994;18(3):277–85.
65. McDermott R, Rowley K, Lee A, Knight S, O'Dea K. Increase in prevalence of obesity and diabetes and decrease in plasma cholesterol in a central Australian Aboriginal community. *Med J Aust* 2000;175:480–4.
66. Zakrevsky E, Binns C, Gracey M. *Aboriginal Community Foodstores Project: assessment of nutritional status*. Perth: Health Department of Western Australia, 1996.
67. Gault A. *Health survey, Urapuntja Health Service*. Alice Springs: Institute of Aboriginal Development, 1986.
68. Maggiore PM. Analysis and interpretation of food record data from remote Aboriginal communities. *Proc Nut Soc Aust* 1990;15:220–3.
69. King R, Smith R, Spargo R. Dietary patterns of Aboriginal children in the Kimberley. *Proc Nut Soc Aust* 1985;10:173.
70. Meehan B. *Shell bed to shell midden*. Canberra: Australian Institute of Aboriginal Studies, 1982.
71. Altman J. *Hunter–gatherers today: an Aboriginal economy in north Australia*. Canberra: Australian Institute of Aboriginal Studies, 1987.
72. Haynes D, Michalek-Wagner K, Kwan D. *Trace metal concentrations in Torres Strait green turtle (Chelonia mydas) tissues, 1997–1998*. Report prepared for the Torres Strait Regional Authority, 2001.
73. Sinclair AJ, Mann NJ, Kelly J. Kangaroo meat for human consumption. *Proc Nut Soc Aust* 1997;21:52–7.
74. Calaby J. Man, fauna and climate in Aboriginal Australia. In: Mulvaney DJ, Golson J, eds. *Aboriginal man and environment*. Canberra: Australian National University Press, 1971:80–93.
75. Peterson N. The traditional pattern of subsistence to 1975. In: Hetzel BS, Frith HJ, eds. *The nutrition of Aborigines in relation to the ecosystem of central Australia*. Melbourne: CSIRO, 1978:25–35.
76. Cane S, Stanley O. *Land use and resources in desert homelands*. Canberra: Northern Australia Research Unit, Australian National University, 1985.
77. Lee A. Survival tucker: Aboriginal dietary intake and a successful community-based nutrition intervention project. PhD Thesis, Sydney: University of Sydney, 1992.

78. Rae C, Lamprell V, Lion R, Rae AM. The role of bushfoods in contemporary Aboriginal diets. *Proc Nut Soc Aust* 1982;7:45–9.
79. Strategic Inter-Governmental Nutrition Alliance. *Eat Well Australia: an agenda for action for public health nutrition, 2000–2010*, Canberra: National Public Health Partnership, 2001.
80. Queensland Health. *Queensland Aboriginal and Torres Strait Islander Food and Nutrition Strategy: Indigenous Health Program and Nutrition Program*. Brisbane: Australian Centre for International and Tropical Health and Nutrition, University of Queensland, 1995.
81. O'Dea K. Marked improvement in carbohydrate and lipid metabolism in diabetic Australian Aborigines after temporary reversion to traditional lifestyle. *Diabetes* 1984;33:596–603.
82. Weeramanthri T, Edmond K. *Northern Territory Preventable Chronic Disease Strategy—the evidence base*. Darwin: Northern Territory Health Services, 1999.
83. White GA, Rowley KG, Daniel M, Skinner K, O'Dea K. Effectiveness of a community directed healthy lifestyle program in a remote Australian Aboriginal community. *Aust NZ J Publ Hlth* 2000;24:136–44.
84. O'Dea K, White NG, Sinclair AJ. An investigation of nutrition-related risk factors in an isolated Aboriginal community in northern Australia: advantages of a traditionally-orientated lifestyle. *Med J Aust* 1988;148:177–80.
85. Brand JC, Darnton-Hill I, Gracey MS, Spargo RM. Lactose malabsorption in Australian Aboriginal children. *Am J Clin Nutr* 1985;41(3):620–2.
86. Buttenshaw R, Sheridan J, Tye V, Miller O, Carseldine J, Battistuta D et al. Lactose malabsorption and its temporal stability in Aboriginal children. *Proc Nut Soc Aust* 1990;15:228.

B SOCIAL STATUS, NUTRITION AND THE COST OF HEALTHY EATING

Katrine Baghurst

SOCIAL STATUS AND NUTRITION

There is no doubt that a social gradient exists for many diet-related chronic disease conditions in Australia, but—with the exception of extreme poverty conditions—it is unclear to what extent dietary differences across social groups effect chronic disease outcome or nutrition status. Diet may have a direct affect but may also play a role in the chronic disease risk experienced by the next generation. This is discussed in detail in the ‘Special considerations’ section of the Dietary Guidelines for Children and Adolescents in Australia.

The 1995 National Nutrition Survey^{1,2} showed few systematic differences in food and nutrient intake across quintiles of social disadvantage, as defined by SEIFA (socio-economic indexes for areas), based on the 1991 census. This index assesses the relative social disadvantage of respondents on the basis of their area of residence; relative social disadvantage is determined by economic resources, education and occupation patterns in that area. Table B.1 shows the intake of various food groups across SEIFA quintiles.

Table B.1 Mean daily intakes (g/day) from various food groups: people aged 19 years and over, by SEIFA quintile²

Food group	(grams per day)				
	First quintile (most disadvantaged)	Second quintile	Third quintile	Fourth quintile	Fifth quintile (least disadvantaged)
Cereal & cereal products	196	222	203	217	232
Cereal-based products & dishes	113	115	130	135	136
Fruit products & dishes	126	147	141	143	156
Vegetable products & dishes	264	258	260	262	251
Legumes & pulses	9.8	7.9	10.8	9.5	10.7
Milk products & dishes	281	284	285	292	301
Meat, poultry, game	149	163	164	155	158
Fish & seafood	22	24.5	26.3	25.8	28.8
Egg products & dishes	16	15	16	13	19
Snack foods	3.2	3.0	4.2	3.3	3.9
Sugar products & dishes	20	21	20	18	17
Confectionery	7.8	9.2	8.3	9.3	9.1
Seeds & nuts	3.6	4.1	5.2	4.0	4.8
Fats & oils	12	13	13	12	11
Soup	53	62	55	48	57
Savoury sauces & condiments	30	30	28	30	29
Non-alcoholic beverages	2001	2005	1978	2007	1938
Alcoholic beverages	239	254	273	270	234

Note: SEIFA = socio-economic indexes for areas.

The findings can be summarised thus for the major food groups:

- Consumption of cereals and cereal-based foods (for example, rice, pasta and breads) was somewhat lower in the most disadvantaged group and the middle group compared with all other groups. Consumption of cereal-based products and dishes (for example, cakes and biscuits) was about 20 per cent lower in the two most disadvantaged groups compared with the other three.
- Consumption of fruit and fruit products was lower (10–20 per cent) in the most disadvantaged group compared with the other four groups, but vegetable and legume consumption showed no consistent trend across the groups.
- Consumption of milk and milk products increased slightly with social advantage—about a 10 per cent increase across the groups.
- Consumption of meat, poultry and game was slightly higher in the middle quintiles; fish and seafood consumption gradually increased with social advantage.

An assessment of energy and nutrient intakes across the SEIFA quintiles (see Table B.2) showed that energy intake increased with social advantage, as did intakes of most nutrients. However, when correcting for energy differences across groups, few differences were apparent in dietary quality, defined as nutrient intake per unit energy. It is unclear from the published data whether other factors, such as the age profile, differed across the quintiles of disadvantage and how much variation in factors such as age (which are known to influence total food intake) might account for the differences that were apparent (for example, in total energy intake). Physical activity may also vary across quintiles.

Table B.2 Mean daily intakes of energy and nutrient densities: people aged 19 years and over, by SEIFA quintile¹

Food group	First quintile (most disadvantaged)	Second quintile	Third quintile	Fourth quintile	Fifth quintile (least disadvantaged)
Energy (MJ)	8.82	9.18	9.37	9.31	9.45
Nutrient density (per 10MJ energy)					
Protein (g)	98.2	98.4	98.5	98.6	99.4
Fat (g)	89.8	90.7	91.1	88.9	88.8
Saturated (g)	35.7	35.5	36.0	35.2	35.0
Mono-unsaturated (g)	32.5	32.8	33.4	32.4	32.3
Polyunsaturated (g)	13.5	13.5	13.9	13.4	13.4
Cholesterol (mg)	332	331	332	319	305
Total carbohydrate (g)	276	277	272	276	277
Sugars (g)	128	125	123	124	123
Starch (g)	147	150	148	151	152
Fibre (g)	24.4	24.4	24.9	25.2	25.6
Alcohol (g)	13.4	13.3	14.3	14.6	13.8
Vitamin A (µg)	1280	1299	1236	1218	1329
Thiamin (mg)	1.81	1.74	1.81	1.83	1.80
Riboflavin (mg)	2.27	2.18	2.24	2.25	2.22
Niacin (mg)	45.8	45.9	45.6	45.5	45.9
Folate (µg)	289	286	299	272	292
Vitamin C (mg)	132	131	130	135	142
Calcium (mg)	907	888	900	926	945
Phosphorus (mg)	1626	1631	1630	1654	1673
Magnesium (mg)	353	356	354	361	366
Iron (mg)	15.1	15.0	15.3	15.4	15.6
Zinc (mg)	12.9	13.07	12.8	13.0	13.3
Potassium (mg)	3541	3495	3507	3528	3551

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.

THE COST OF HEALTHY EATING

In recent years most countries in the western world have developed dietary guidelines and recommendations similar to those outlined in this Australian document. The aim is to improve health and nutritional status and help prevent chronic diseases such as coronary heart disease, certain cancers, hypertension and diabetes. If, however, the changes encouraged by these guidelines are financially costly, there will be groups, including some low-income families, in the community that are unable or unwilling to make these adjustments.

Although there is widespread speculation about the expense of healthy, or 'healthful', eating, few cost analyses of healthful diets have been reported. One study, in the United Kingdom, compared the cost of one-day diets that did and did not conform to the British dietary guidelines and found that following the guidelines was in fact potentially more expensive.³ In Australia, Santich looked at the eating patterns recommended in various federal government nutrition education publications and concluded that the changes recommended may not be financially realistic for low-income families.⁴ However, this conclusion was based on an analysis of specific recipes given in the pamphlets and on the substitution models for healthful eating used in the education materials—for example, using lean minced beef instead of fattier versions and using wholegrain breads instead of white bread. Using a qualitative sociological analysis, Crowley has also suggested that the 'cost' of a diet that follows the dietary guidelines is potentially higher compared with the cost of the average Australian diet.⁵ His analysis included factors outside the direct financial cost of food as purchased, such as access and transportation.

In the early 1990s McAllister et al.⁶ undertook an analysis, based on Australian data, to determine whether it was necessarily more directly financially costly to follow a healthful diet in Australia (see Table B.3). Three different approaches were used: a substitution approach; a relative costing of individual diets that are in line with the dietary guidelines; and an analysis of the cost of eating according to a healthy eating plan, the 12345+ Nutrition Plan developed by the CSIRO.⁷ This healthy eating plan was highly flexible; it was designed to produce diets conforming to both the existing Australian dietary guidelines and the recommended dietary intakes for people with varying energy needs and/or special nutrient needs (for example, during pregnancy, lactation and adolescence) and from a variety of cultural backgrounds.

The cost of 229 foods and drinks used in the analysis was determined by assessing prices in four major supermarkets and other food outlets such as takeaway stores when necessary. The food outlets used for pricing were located in a suburban area of Adelaide where there is a large concentration of low-income families.⁸ In each supermarket, for each food or drink, the cheapest branded item (that is, bearing the manufacturer's brand name) was recorded, together with the price of the equivalent generic item (that is, without the manufacturer's brand name but commonly associated with a particular retailer).

An analysis of the potential cost of direct substitution of healthful food choices for less healthful ones (for example, product-by-product substitution through the use of fat, fibre or salt-modified alternatives) showed that this approach would result in a more costly diet while providing limited nutritional improvement at the population level. Pricing of self-selected diets of people who currently comply with the dietary guidelines and targets for healthful eating showed that these people are paying more per megajoule. In contrast, costing of diets that conformed to a new healthful eating plan—designed to produce eating patterns that meet both the dietary goals set for components of the food supply (such as dietary fat, refined sugars and fibre) and the recommended daily intakes for energy, protein, vitamins and minerals—showed that healthful eating need not be more expensive and, indeed, for most people would bring cost savings.

In summary, this study showed that healthful eating is not necessarily more expensive but that restructuring the diet, rather than using a direct-substitution approach, is the more cost effective strategy. Education programs that stress this restructuring approach and its cost advantages are therefore more likely to be successful in promoting an affordable and effective healthful alternative for people with limited financial means.

Unfortunately, it is generally easier for people to understand and adopt a substitution approach rather than to basically restructure their diets. They also receive encouragement to adopt the substitution approach not only from food manufacturers, who, understandably, wish to promote specific healthful products, but also from much of the educational literature produced by health professionals.

The study just described was designed to look only at the potential financial costs of healthful eating. It showed that the theoretical cost savings associated with healthier diets would be similar across all social groups in Australia. But there are obviously other factors that could make a healthful diet more difficult to achieve—such as the ready availability of healthful foods, skills, facilities, time, taste factors and motivation. And these factors might vary across social, educational and income groups. This has been discussed by Santich⁴ and Crowley⁵, but the data available are limited and further investigation would be valuable, especially with respect to low-income groups in the community. These issues do, however, need to be dealt with in implementing the dietary guidelines in socially disadvantaged groups and will be addressed in the implementation plan for the dietary guidelines.

Table B.3 summarises the financial costs of healthy eating, as determined in the 1989 Australian Bureau of Statistics Household Expenditure Survey, the 1990 Victorian and South Australian surveys, and the CSIRO's 12345+ Nutrition Plan.

Table B.3 The financial costs of healthy eating⁶

	Product type	Cost (\$)
1989 Household Expenditure Survey	Branded	4.92
1990 Victorian and South Australian surveys		
Current cost	Generic	4.58
	Branded	4.99
Conformers to dietary guidelines	Generic	4.60
	Branded	4.96
Non-conformers with equivalent energy intakes	Generic	4.01
	Branded	4.33
Substitution of health choices (adjusted for energy content)	Generic	5.22
	Branded	5.54
Healthy eating plan (CSIRO 12345+ Nutrition Plan)		
Average woman	Generic	3.48
	Branded	3.76
Average man	Generic	3.81
	Branded	4.09

REFERENCES

1. Australian Bureau of Statistics, Department Health and Aged Care. *National Nutrition Survey: nutrient intakes and physical measurements, Australia, 1995*. Canberra: ABS, 1998.
2. Australian Bureau of Statistics, Department Health and Aged Care. *National Nutrition Survey: foods eaten, Australia, 1995*. Canberra: ABS, 1999.
3. Cade J, Booth S. What can people eat to meet the dietary goals—and how much does it cost? *J Hum Nutr Diet* 1990;3(1):99–207.
4. Santich B. The compatibility of nutritional ideals with low incomes. *Food Aust* 1992;44:230–4.
5. Crowley S. Will the poor be able to afford a healthy diet? *Nutridate* 1992;3:5–7.
6. McAllister M, Baghurst KI, Record SJ. Financial costs of healthy eating: a comparison of three different approaches. *J Nutr Ed* 1994;26:131–9.
7. Baghurst, KI, Hertzler, AA, Record, SJ, Spurr C. The development of a simple dietary assessment and education tool for use by individuals and health professionals. *J Nutr Educ* 1992;24:165–72.
8. South Australian Health Commission. *A social health atlas of South Australia*. Adelaide: SAHC, 1990.

C **DIETARY GUIDELINES AND THE SUSTAINABILITY OF FOOD SYSTEMS**

Malcolm Riley

Sustainable development is relevant to dietary guidelines because the production and consumption of food is a fundamental human activity guided by what we choose to eat. Human activity has affected all the major planetary processes and cycles, and the earth's human population continues to grow, as does its appetite for resources. The immediate problems facing the world concern not limits to those resources but the increasing disturbances to global and natural systems. Systemic changes have been recognised—climate change, ozone depletion, biodiversity loss, freshwater depletion and degraded food-producing systems.¹ All of these have important implications for activities such as food production. Although sustainable development is a global concern, solutions also need to be sought at national and local levels. If we are to deal comprehensively with the problems that have been identified, we need integrated policies across many sectors; these policies must be adequately resourced and have an effective legislative and administrative base.²

The World Commission on Environment and Development defined *sustainable development* as 'the ability to meet the needs of the current generation without compromising future generations' ability to meet their economic needs'. Common elements in more comprehensive definitions of *sustainable agriculture and animal production* are resource efficiency, profitability, productivity, environmental soundness, biodiversity, social viability, and other ethical aspects.³ Important prerequisites for *sustainable production* are appropriate governmental policies, awareness of our way of thinking, and a more communal world view.

The consensus on human impact is that every major planetary process—whether in the biosphere, the lithosphere, the hydrosphere or the atmosphere—is already dominated by our activity.⁴ The dominant species on earth (domesticated animals and plants) are heavily selected for specific traits, and this has reduced genetic heterogeneity and adaptability. Maintaining the desirable traits in adverse environments, and in the face of mounting disease and pathogen attacks (predicted results of global climate change), requires ever-increasing energy inputs and environmental modification.⁴

It can be argued that the limits of sustainability have already been reached in the human population—with 6 billion humans alive today—since at least 20 per cent of the population suffers from hunger, our natural resources are overexploited, and biodiversity is threatened.⁵ Demographers now believe that the world population will reach a peak of 8 to 10 billion during the 21st century, before beginning to slowly decline as fertility rates drop below the level necessary for replacement.⁶ Problems relating to sustainable development will then be focused on managing for the peak world population, rather than for a continually rising population. This task will be difficult enough in its own right, and ecological

sustainability will necessarily move from being a side issue to being a central force in managing development, including the development of our food production and consumption systems.

Humans are highly successful in an evolutionary sense, as demonstrated by steady population increase, and this may be proof of our ability to modify ecosystems to our advantage. It has been argued that humankind can take care of environmental concerns when it can afford to and that environmental clean-up follows wealth creation. But it is now clear that this might be misleading: for example, the rate of increase in agricultural productivity is slowing and major food production systems such as fisheries are approaching maximum capacity; it has been estimated that nearly half the world's marine fish populations are fully exploited and another 22 per cent are overexploited.

Agricultural sustainability can be enhanced by a switch from linear solutions to circular approaches to food production—for example, closing water and nutrient loops to reduce reliance on external inputs and reducing outputs of waste from the system. These general strategies would help restore soil fertility and ultimately improve food security.⁷

In Australia the most pressing environmental problems are loss of biodiversity, land degradation, and disturbances to inland waterways.² Effective solutions to these problems will require halting large-scale land clearing, measures to deal with dryland salinity, restoration of adequate environmental flows to our rivers, and major land use changes, including the retirement of large areas of land from grazing and similar uses.²

In the last 30 years there have been substantial changes to eating habits in Australia, with the result that both apparent consumption and the level of waste (packaging and food wastes) have increased. Between 1970 and 1990 annual apparent food consumption in Sydney increased from 0.52 tonnes per person to 1.00 tonne per person—a 92 per cent increase. The increase in consumption is attributable to changes in food processing, retailing and lifestyle, rather than an increase in food intake by each individual.⁸

Australians are the highest per capita users of water in the world, using 2.3 times the global average. Domestic water use accounts for only 12 per cent of this; agricultural use accounts for 79 per cent and industrial use for 9 per cent.

Published guidelines to help people consume food in a manner more consistent with sustainable development focus on matters such as avoiding over-consumption, eating less processed food, and eating food produced locally and in season.^{9,10} Suggestions for ways that dietitians can contribute to protection of the environment have also been the subject of recent discussion.¹¹

It is apparent that a move towards more sustainable food-production methods will require policy development and change in many different sectors. If successful, we can expect that this will result in substantial changes to the way we eat. While these Dietary Guidelines for Australian Adults are consistent with sustainable food production and consumption, dietary guidelines of the future

will probably become more and more focused on sustainability as the problems caused by non-sustainable systems become more starkly obvious.

REFERENCES

1. McMichael AJ. Global environmental change as 'risk factor': can epidemiology cope? *Am J Pub Hlth* 2001;91:1172–4.
2. Yencken D, Wilkinson D. *Resetting the compass—Australia's journey towards sustainability*. Collingwood: CSIRO Publishing, 2000.
3. Olesen I, Groen AF, Gjerde B. Definition of animal breeding goals for sustainable production systems. *J Animal Sci* 2000;78:570–82.
4. Western D. Human-modified ecosystems and future evolution. *Proc Nat Acad Sc* 2001;98:5458–65.
5. Nentwig W. The importance of human ecology at the threshold of the next millenium: how can population growth be stopped? *Naturwissenschaften* 1999;86:411–21.
6. Caldwell JC. The demographic dimension: past and future. *Asia Pac J Clin Nutr* 2001;10(suppl.):S93.
7. Esrey SA. Towards a recycling society: ecological sanitation—closing the loop to food security. *Water and Science Technology* 2001;43:177–87.
8. State of the Environment Advisory Council. *Australia: state of the environment, 1996*. Collingwood, Victoria: CSIRO Publishing, 1996:3–34.
9. Gussow JD, Clancy KL. Dietary guidelines for sustainability. *J Nutr Ed* 1986;18:1–5.
10. Gussow JD. Dietary guidelines for sustainability: twelve years later. *J Nutr Ed* 1999;31:194–200.
11. American Dietetic Association. Dietetic professionals can implement practices to conserve natural resources and protect the environment. Position statement. *J Amer Diet Assoc* 2001;101:1221–7.

The National Health and Medical Research Council

The National Health and Medical Research Council (NHMRC) is a statutory body within the portfolio of the Commonwealth Minister for Health and Ageing, established by the *National Health and Medical Research Council Act 1992*. The NHMRC advises the Australian community and Commonwealth; State and Territory Governments on standards of individual and public health, and supports research to improve those standards.

The NHMRC advises the Commonwealth Government on the funding of medical and public health research and training in Australia and supports many of the medical advances made by Australians.

The NHMRC also develops guidelines and standards for the ethical conduct of health and medical research.

The Council comprises nominees of Commonwealth, State and Territory health authorities, professional and scientific colleges and associations, unions, universities, business, consumer groups, welfare organisations, conservation groups and the Aboriginal and Torres Strait Islander Commission.

The Council meets up to four times a year to consider and make decisions on reports prepared by committees and working parties following wide consultation on the issue under consideration.

A regular publishing program ensures that Council's recommendations are widely available to governments, the community, scientific, industrial and educational groups.

The Council publishes extensively in the following areas:

- Aged care
- Child health
- Dentistry
- Drugs and poisons
- Environmental health
- Ethics – Human
- Health promotion
- Mental health
- NHMRC – National Health and Medical Research Council
- Public health
- Sport/Injury
- Workforce
- Communicable diseases
- Clinical practice guidelines
- Diabetes
- Drug and substance abuse
- Ethics – Animal
- Health procedures
- Infection control
- Men's health
- Nutrition
- Research
- Women's health

A list of current publications is available from:

The Publications Officer
NHMRC
MDP 100
GPO Box 9848
Canberra ACT 2601

Phone: (02) 6289 9520 (24-hour answering machine)
Toll free: 1800 020 103
Fax: (02) 6289 9197
E-mail: nhmrc.publications@nhmrc.gov.au
Internet: <http://www.nhmrc.gov.au>