

5 Nutritional status and health

There is a direct causal link between diet and nutritional deficiency conditions; undernutrition is rare, except in some Aboriginal and Torres Strait Islander communities in Australia, and the most common deficiency is iron deficiency. A range of clinical conditions, such as 'inborn errors of metabolism', or a genetic predisposition to disease may influence the ability to maintain an adequate dietary intake or, conversely, may require nutritional intervention to prevent or alleviate the disease syndrome. An apposite example is non-insulin-dependent diabetes mellitus (NIDDM), to which a substantial proportion of the population is predisposed genetically; overweight may lead to expression of the condition and attention to diet can prevent or alleviate the symptoms. There is also some concern about the possible increase in prevalence of anorexia nervosa and bulimia nervosa, although these constitute at present a clinical, rather than public health, problem.

Primarily, however, the diet-related diseases of greatest public health significance in Australia are the chronic, preventable, non-communicable, 'lifestyle-related' conditions associated with inactivity and over-consumption of food.

Causes of death discussed in this chapter are identified specifically by an internationally recognised coding system; these 'ICD-9' codes are shown for relevant conditions in Tables 5.6 and 5.8.

5.1 Growth and nutritional status

Infant nutrition

Exclusive breastfeeding by healthy, well-nourished women is promoted as being the optimal method of infant feeding for at least the first four to six months of life.¹ 'Increase breastfeeding' was one of the Dietary Goals for Australia announced in 1979,² and the inclusion of a guideline encouraging breastfeeding in the Dietary Guidelines for Australians—a set of advice aimed at the general population of health adults—recognises the nutritional, health, social and economic benefits of breastfeeding for the community as a whole.¹

Hitchcock and Coy describe the decline in breastfeeding rates that began at the end of the nineteenth century in Australia and other developed countries.³ As the number of women breastfeeding decreased, artificial formulae and cows' milk were more widely given to infants. The quality of modern 'breast-milk substitutes' is such that they are recognised as adequate where breastfeeding is not possible (see Box 5.1). The decline stopped in the late 1960s, but Victorian hospital records, for example, shows that by the early 1970s, whereas 50–60 per cent of mothers were breastfeeding on discharge from hospital, only 21 per cent were fully breastfeeding at three months and 10 per cent fully breastfeeding at six months.⁴ For 124 Queensland mothers who gave birth after 1974, the mean duration of unsupplemented breastfeeding was 13 weeks, compared with about 7 weeks for 208 mothers with children born between 1970 and 1974; the duration for partial breastfeeding was approximately 30 weeks (feeding after 1974) compared with 12 weeks (1970–74).⁵ Records from 739 primiparous Victorian women

who gave birth between 1 May 1984 and 30 April 1985 showed that smoking, greater maternal age, and excess weight were independent risk factors for early cessation of breastfeeding in mothers who breastfed for at least 14 days; an incidental finding from this study was that 8 per cent of mothers breastfeeding at discharge from hospital ceased within 14 days.⁶

Box 5.1: Quality of infant formulae

Although breastfeeding rates began to increase in the 1970s, the intensity of research into infant nutritional requirements and increasingly sophisticated technology were making available infant formulae of improved and consistent quality. Rickets was reported in Victorian children in the early years of the twentieth century and Dr J Cumpston, Commonwealth Director-General of Health from 1921 to 1945, observed that 'a child artificially fed before six months of age is at least twice as likely to suffer from rickets as the child breast-fed for that period.'⁷ Recent studies confirm the quality of modern infant formulae. Results over six months for 263 Canadian infants suggested little difference in growth between infants who were breastfed or bottle fed with infant formulae based on soy or cows' milk.⁸ Heinig et al.⁹ compared the intakes and growth of matched cohorts of infants breastfed or bottle fed for at least 12 months, finding higher energy intake and faster growth in formula-fed infants at three, six and nine months (noting, however, that there was no evidence for functional advantage with rapid growth). Normal growth is reported for cows'-milk-intolerant infants and children under 8 years of age fed a soy-based formula for at least one month.¹⁰ A trial of a milk-based formula in 781 healthy infants under 1 year also found normal increases in weight and length.¹¹

Prevalence of breastfeeding

Varying definitions of terms make it difficult to compare studies of breastfeeding rates. 'Breastfed' may mean exclusively breastfed or it may mean that an infant took breast-milk at some time in the survey period. The age at which breastfeeding occurs is also subject to interpretation: 'at three months' may mean over the three-month period or until some time in the third month.¹²

An example of difficulties within a survey is the questions on breastfeeding asked in the 1989–90 National Health Survey. These were asked of women aged 18–50 years who had children aged 5 years or less at the time of interview. Questions were contained in a separate self-completion questionnaire on selected women's health issues. Both age of infant and duration of feeding were recorded in months, enabling identification of children who were currently or not currently breastfed. The Australian Bureau of Statistics reports some limitations on the analyses possible with these data. Design restrictions on the computer records limited the extent to which breastfeeding characteristics could be related to other characteristics of the child. The questions elicited information about whether or not the infant was breastfed and the duration of breastfeeding but could not identify whether children were fully or partially breastfed. The use of a self-completion questionnaire may have affected the reliability of the estimates relative to other parts of the survey and, since the completed questionnaires were not sighted by the interviewer, responses could not be verified at interview. Some

reporting errors, such as respondents misunderstanding the questions, missing questions or incorrectly following sequence guides, survived into the final data set.

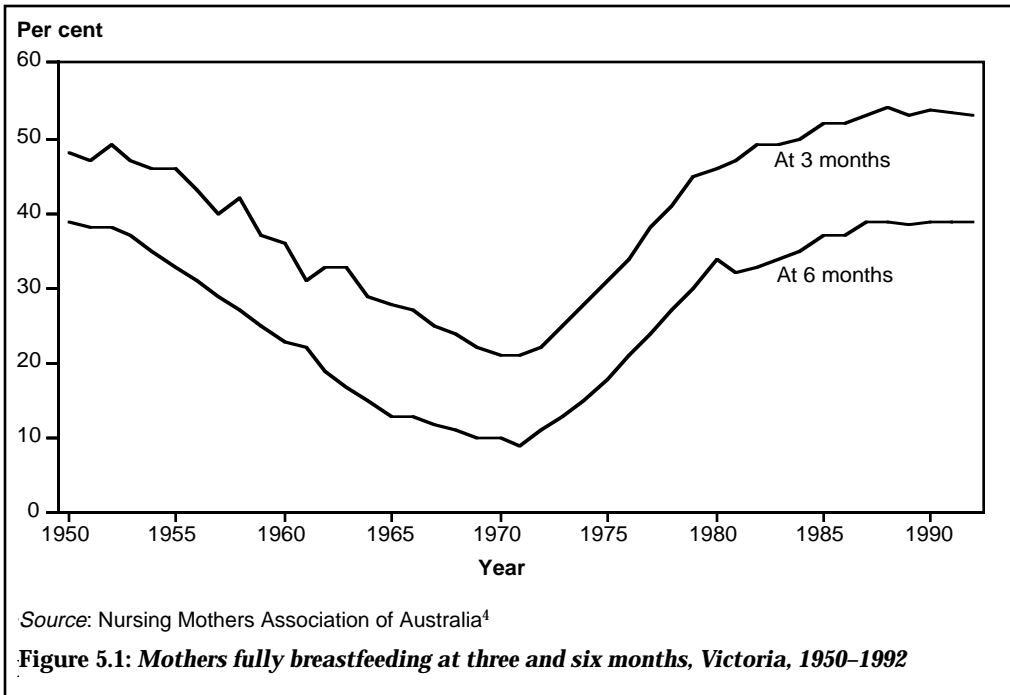
The 1989–90 National Health Survey information¹³ and the results of a small survey by the Brotherhood of St Laurence in 1992¹⁴ were consistent, with 77 per cent and 78 per cent respectively reporting that they initiated breastfeeding; this suggests a decline in initiation of breastfeeding since 1983. In a recent study of a group of primiparous women from the Newcastle (New South Wales) area interviewed four months after delivery, 49 per cent were fully breastfeeding, 12 per cent partially breastfeeding and 39 per cent bottle feeding.¹⁵

The National Health Survey data also showed that mothers aged 25–34 years had the highest proportion of breastfeeding (80 per cent) followed by 18–24-year-old women (77 per cent) and 35–44-year-old women (71 per cent). Women in the age group 45–50 years were more likely to have fed their infants with infant formulae. Of mothers who breastfed, most were likely to have breastfed for between six and 12 months (37 per cent). Only 7.4 per cent of breastfeeding mothers did so for longer than 18 months. The Brotherhood of St Laurence survey also found that mothers on low incomes were less likely to breastfeed and that between four and seven months the rate of breastfeeding fell to 58 per cent.¹⁴ The Newcastle study found that those aged 26 years and over were twice as likely to be fully or partially breastfeeding after four months; and breastfeeding at four months was strongly associated with education level (the odds ratio for feeding with infant formulae between those with tertiary education and those who had not completed high school was 0.06).

A similar pattern held for the introduction of complementary foods before 16 weeks. The study group noted that the sample was not representative of the local population: of 294 mothers contacted, the 165 available for analysis were significantly more likely to be older and married, to have private health insurance, and to have achieved a higher level of education. In addition, these were self-reported data. These factors all would tend to overestimate desirable behaviours.¹⁵

The National Health Survey indicated that younger mothers were likely to have breastfed for shorter periods than older mothers. Nearly half (48 per cent) of the mothers aged 18–24 years breastfed for less than three months, and only 32 per cent breastfed their baby for more than six months. In comparison, 74 per cent of mothers aged 35–44 years and 63 per cent of mothers aged 25–34 years breastfed for more than six months. Of the mothers aged 18–24 years, 10 per cent breastfed for more than 12 months, compared with 40 per cent of mothers aged 35–44 years.

Apart from the 1989–90 National Health Survey, national data on the prevalence and duration of breastfeeding were collected in Australia only in 1983, when 85 per cent of mothers were breastfeeding at discharge and 54–55 per cent three months later.¹⁶ There is no ongoing data collection at the national level. Victoria has records from 1950 of mothers fully breastfeeding at three and six months (see Figure 5.1) and for those fully and partially breastfeeding since 1985–86 on a sample in excess of 60 000.⁴ Rates of those breastfeeding fully at three and six months have remained at about 53 per cent and 39 per cent respectively between 1985–86 and 1991–92; rates for those partially breastfeeding at three and six months rose from 3 per cent to 4.7 and 4.4 per cent respectively.⁴



With the exception of Victoria, data collection at the State level has been sporadic. Appendix C lists data from a compilation by the Nursing Mothers Association of Australia for those years when data from other States were available to compare with the Victorian data. In 1984–85 a joint survey in Western Australia and Tasmania indicated a continued trend to increasing breastfeeding rates and duration in those States. Over the preceding five years, prevalence rates on hospital discharge rose from 82 per cent to 86 per cent in Western Australia and 72 to 81 per cent in Tasmania. At six months from discharge 45 per cent of mothers in both States were still breastfeeding.¹³

Siskind et al. collected infant feeding histories from 864 Queensland mothers with a mean age of 54.8 years, between 1982 and 1985.⁵ These data show the same pattern as the Victorian breastfeeding data: the proportion of those whose infants were breastfed and never bottle fed fell from nearly 50 per cent before 1940 to a low of 20 per cent in the early 1970s, increasing after 1975. The proportion never breastfed also peaked in the late 1960s and early 1970s. The Queensland survey also found that mothers with the highest level of education led the general trend: the proportion of babies never breastfed fell more steeply, reached the nadir sooner—in 1965–69—and was increasing by 1970–74 when the mean for all groups was reaching its low point.

Implementation of the WHO International Code of Marketing of Breast Milk Substitutes

In 1981 Australia became a signatory to the WHO International Code of Marketing of Breast Milk Substitutes.¹⁷ The stated aim of the Code was to contribute to the provision of safe and adequate nutrition for infants by protecting and promoting breastfeeding and by ensuring the proper use of breast-milk substitutes when their use is appropriate. The Code does not have treaty or convention status; its intended use was

as a model set of recommendations that could be adapted to national circumstances by member states. In Australia an NHMRC working party was established to develop guidelines for the promotion of breastfeeding and implementation of the Code, and guidelines were adopted by the NHMRC in 1984.¹⁸

Implementation in Australia of the WHO Code was reviewed in 1991–92. The review aimed to assess action taken to give effect to the Code, what had facilitated or impeded such action, and what impact there had been.¹²

The review found that implementation had been initiated in several ways: legislation had been developed to regulate compositional, hygiene and labelling requirements for infant formulas, and the States had developed and begun implementing policies on breastfeeding.¹² A self-regulatory Agreement on the Marketing in Australia of Infant Formula for Manufacturers and Importers (Australian Agreement) was signed by all manufacturers and importers in May 1992. It covers the marketing of infant formulae for infants aged to one year, and in this sense is stricter than the WHO Code.⁴ An important feature of the Australian Agreement is the establishment of a panel to monitor its implementation. The responsibilities of health workers arising from the WHO Code are to be addressed through guidelines being developed by the NHMRC. These guidelines will revise and expand the ongoing (1984) guidelines. Similar agreements are expected to be developed for retailers and bottle and teat manufacturers.¹²

The WHO Code review steering committee surveyed the available literature on the prevalence of breastfeeding, advertising in professional journals and magazines for women and parents, information made available for mothers, and labelling on infant formula packaging. It also sought information on knowledge about breastfeeding by mail surveys of mothers, community support groups, professional associations and government bodies. The committee reported that, although individuals surveyed had little or no knowledge of either the WHO Code or the Australian Agreement, they were acting in a manner consistent with recommended practice. It was felt that the Nursing Mothers Association of Australia and other interest groups and health professionals were responsible for the high level of public awareness of inappropriate medical advice and commercial influences on breastfeeding.¹²

Monitoring breastfeeding rates

Hospital records may be the only long-term sources of data on breastfeeding but, with the exception of Victoria, collation to State level has been sporadic. The WHO Code review steering committee recommended that the Australian Government establish a 'coordinated national system whereby the rates of breastfeeding at discharge, 2, 4 and 6 weeks, and 3, 6 and 12 months in both the general community and specific groups are collected on an ongoing basis'.¹² The 1992 Workshop to revise nutrition goals and targets recommended 10 years targets for the prevalence of full and partial breastfeeding at 3 and 6 months, and of breastfeeding at the time of discharge from hospital.¹⁹ At a minimum, a monitoring program should include this level of collection at the national level.

Growth rates of infants and children

There are currently no national growth data for Australian infants and young children. The Perth Growth Study, begun in 1979, followed the growth of 200 second-generation, full-term infants to 5 years of age. In the first year of life, growth rates were similar to those reported by Clements for New South Wales infants in 1933 but lower than those reported in the 1960s.^{20,21} The Perth Growth Study workers suggested that 'the increased rates of growth in the 1960s [may have been] due to the early introduction of solids and the use of cows' milk formulas with added sugar, sometimes made excessively concentrated'.²¹

Growth in weight and length of the Perth Growth Study children in the second and third years of life was similar to the 1986 WHO reference values but lower, after six months of age, than the 1972 NHMRC median reference weights. The difference was interpreted as an indication that the 1972 sample was relatively overweight from six months onwards, perhaps arising from sampling procedures or as a result of infant feeding practices in Sydney at that time.

The most comprehensive survey of physical measurements of infants and children was that of Jones and Hemphill²² conducted in 1971–72 in New South Wales; 16 000 infants and pre-school children from 'virtually all baby health centres in Sydney' were used in the development of the national reference *Percentile charts—charts and tables of heights, masses and head circumferences of infants, children and adolescents*, endorsed by the NHMRC in 1972.²³ Table 5.1 shows mean body weights for infants from published studies since 1933 collated by Gracey and Hitchcock.

For children aged from 7 to 15 years, the 1985 Australian Health and Fitness Survey provides the most recent data; 8000 students from all States were surveyed.²⁴ This cross-sectional study cannot be used to derive rates of growth but may be compared with the WHO reference values for growth. The median values for the sample closely followed the fiftieth percentile of the WHO reference values for weight and height. There was also correspondence with the reference values at the tenth and ninetieth percentiles, except that for girls the ninetieth percentile for weight tended to be about 2 kg below the WHO reference value. This may be a reflection of the high level of concern about overweight in adolescent girls and is consistent with the virtual lack of increase in total energy intake in girls aged 10–15 years from this sample found in the associated National Dietary Survey of Schoolchildren.²⁵ There was no corresponding tendency for the tenth percentile weight to be lower than the reference value.

Table 5.1: Mean body weights of Australian infants from studies published since 1933^(a)

Age	Mean body weight (kg)				
	1933	1964	PGS	NMAA	Adelaide
Boys					
Birth	3.74	3.51	3.56	na	3.39
6 weeks	4.67	5.18	4.81	na	na
3 months	5.91	6.73	5.92	6.31	6.07
6 months	7.63	8.84	7.85	7.96	7.89
9 months	8.97	10.71	9.11	9.25	na
12 months	10.02	11.20	10.01	10.29	10.15
Girls					
Birth	3.62	3.45	3.47	na	3.25
6 weeks	4.52	4.95	4.60	na	na
3 months	5.73	6.22	5.61	5.71	5.62
6 months	7.49	8.18	7.44	7.26	7.31
9 months	8.71	9.40	8.56	8.45	na
12 months	9.73	10.48	9.50	9.49	9.56

na Not available

(a) 1933—a retrospective study by F W Clements of records from more than 1000 infants attending child health centres in Sydney

1964—a comparative study from Sydney child health centres, reported by Bell and Lay

PGS—Perth Growth Study, by Gracey and Hitchcock, 1985

NMAA—a report from the Nursing Mothers Association of Australia, information obtained by questionnaire

Adelaide—a prospective study of the growth of infants in South Australia, reported by Boulton

Source: Gracey and Hitchcock²¹

5.2 Dietary inadequacies

Food supply and dietary intake data suggest that nutrient deficiencies should be uncommon. This view is supported by the Australian mortality surveillance profiles for nutritional deficiencies (ICD-9:260–269) and anaemias (ICD-9:280–285). There was a slight decline in age-standardised mortality for both these groups of conditions between 1979 and 1990. Nevertheless, under-nutrition has been reported in some population subgroups, particularly in young children and in pregnant and lactating women.

Energy

Protein–energy malnutrition (ICD-9:260–263) was recorded as cause of death for 627 people for the period from 1979 to 1991 (328 females and 299 males). Deaths due to anorexia (ICD-9:307.1) and other ‘unspecified eating disorders’ (ICD-9:347.5) were recorded for four males and seven females. Starvation (ICD-9:E904.1) was the cause recorded for the deaths of three males and eight females. One other nutrition-related classification is recorded: ‘slow fetal growth, fetal malnutrition and immaturity’ (ICD-9:764, 765): age-specific death rates in 1990 were 1064 and 938 per million for males and females respectively.²⁶ Some of these deaths may be attributable to nutrition as the primary cause.

The prevalence of underweight in women aged 20–24 years was nearly 30 per cent, compared with 11 per cent for men. The rate for women declined sharply with age to age 45–49 years, thereafter remaining at about 5 per cent. The rate for men also declined to age 45–49 years, but the change was less dramatic than for women.²⁷ Classification of weight status is based on Body Mass Index (BMI); see Box 5.2 for definitions.

The 1989 National Heart Foundation Risk Factor Prevalence Study identified 4.5 per cent of men and 15 per cent of women aged 20–69 years as underweight (BMI < 20).²⁷ The prevalence of underweight was higher for women than for men in each five-year age group (see Figure 5.2).

Box 5.2: Body Mass Index (BMI) and weight classification for nutritional assessment

Definition of BMI

$BMI = (\text{body weight in kilograms}) \div (\text{height in metres})^2$

where *weight is in light clothing and without shoes*
 height is without shoes

N.B. The value obtained should be rounded to the nearest whole number.

Weight categories by BMI

Measured in units of kg/m², the acceptable range for BMI (that consistent with lowest mortality and morbidity risk) is $20 \leq BMI \leq 25$. The complete list is:

<i>Underweight</i>	<i>BMI < 20</i>
<i>'Normal'</i>	<i>$20 \leq BMI \leq 25$</i>
<i>Overweight</i>	<i>$25 < BMI \leq 30$</i>
<i>Obese</i>	<i>BMI > 30</i>

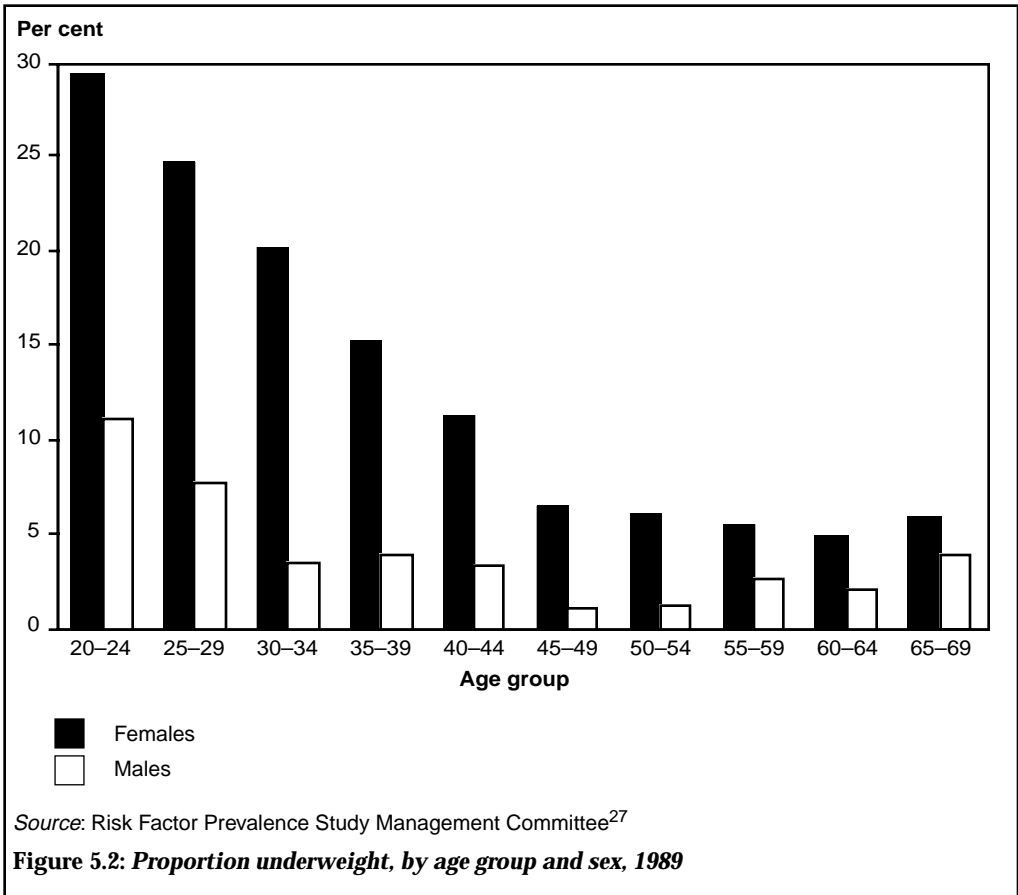
Waters has shown that BMI based on self-reported weight and height provides a reliable estimate of actual BMI (using data from the 1989 National Heart Foundation Risk Factor Prevalence Survey). There was a tendency, however, for a greater proportion of BMIs based on self-reported measurements to fall within the normal range.²⁸

Micronutrients

The symptoms of selenium deficiency have not been reported in Australia, although calculated selenium intakes based on the 1987 Market Basket Survey suggested that men and women aged 25–34 years had intakes below the RDIs.²⁹ At mean consumption levels intakes would be 0.6 of RDI for men (~50µg/day) and 0.5 of RDI for women (~35µg/day) in that age range.* A recent survey of 20 mother–infant pairs in Brisbane

* The 'market basket' simulated diet is calculated by using the mean intakes to derive amounts of representative foods to give an overall equivalent intake at the mean energy intake. Because the principal use of the survey is to estimate contaminant levels, the amounts of foods are multiplied by the ratio of the ninety-fifth percentile energy intake to mean energy intake. This calculation was reversed to estimate mean selenium intakes.

found that the breast milk selenium concentration was $11.9 \pm 3.5 \mu\text{g}/\text{kg}$ and the maternal serum concentration was $81 \pm 15 \mu\text{g}/\text{kg}$; there was no correlation between serum selenium and milk selenium levels.³⁰



Vitamin C deficiency (scurvy) is rarely found in Australia and reported occurrences have been in institutionalised elderly people and infants fed on breast-milk substitutes with low vitamin C levels.³¹ It was reported in ‘isolated persons such as shepherds and boundary riders’ as late as 1901, and there was ‘an outbreak of scurvy’ in the ‘Idiot Cottages of the Victorian Lunacy Department’ in 1919.³²

Thiamin deficiency has been reported in Australia but clinical beri-beri is uncommon.³³ Symptoms of thiamin deficiency are usually found associated with chronic alcoholism, where it contributes to Wernicke–Korsakoff syndrome.³⁴ There has been long-term controversy in Australia about the thiamin adequacy of the food supply (see also Box 2.4) but in general, the population thiamin status appears to be adequate.^{33,35} Historically, beri-beri was reported among Asian pearl divers in Broome (Western Australia) and on Thursday Island in Torres Strait, and occasionally among Chinese

and Aboriginal people elsewhere in northern Australia.³² In southern Australia it was common among Chinese on the Victorian goldfields and in Melbourne in the nineteenth century.³⁶

Like selenium deficiency, iodine deficiency (which causes goitre) results initially from low levels in the soil and so is usually confined to specific locations. In developed countries, it became less common due to the wide use of iodised table salt, the addition of potassium iodate in bread baking, and the wider spread of food sources. Tasmania is an endemic goitre area: 644 cases of goitre were notified in 1921, of which 90 per cent were female and 21 per cent under 15 years of age.³² Cases were also reported in the 1920s in the Bairnsdale district in Victoria and in Cairns in north Queensland.³² Concern that reduced salt intakes in response to public education about sodium and hypertension might lead to a resurgence of goitre is unwarranted because iodised salt is not the only iodine source.³⁷

Zinc intakes appear to be low for some of the population, although deficiency conditions have not been reported. Zinc deficiency has been reported in adolescents and women in developed countries—for example, Canada, the United States and the United Kingdom³⁸—and cannot be discounted in Australia. Zinc status requires further investigation.

The paucity of relevant biochemical information about the Australian population limits the efficacy of monitoring of micronutrient status except for iron status.

Calcium and vitamin D

In otherwise well people, calcium deficiency is asymptomatic because of the large reservoir (in bone) to maintain necessary metabolic functions, but is manifested in increased bone fragility in later in life. Vitamin D deficiency can occur where there is little exposure to sunlight and the diet is low in the vitamin. Those at risk are the very young and the elderly. In Australia, deficiency is virtually restricted to housebound or institutionalised people, particularly the elderly.^{39–41} Low vitamin D status will contribute to poor calcium uptake and thus to decreased bone density. The importance of calcium in the cytology of osteoporosis was recognised by the NHMRC Panel to review the dietary guidelines with the inclusion of a supplementary guideline—'eat foods containing calcium'—directed primarily to girls and women.¹

Nutritional anaemias

Nutritional anaemias arise from chronic, inadequate uptake of the micronutrients essential for the synthesis of the oxygen-transport protein haem; these micronutrients are the essential haem co-factor, iron, and the vitamins folate and cyanocobalamin (vitamin B-12). The most common nutritional anaemia is iron deficiency anaemia, accounting for more than half of all cases of anaemia globally.⁴² Vitamin A deficiency is also associated with iron deficiency anaemia⁴³ and supplementation with iron and vitamin A was shown recently to be of value in the control of iron deficiency anaemia in pregnant Indonesian women.⁴⁴ Vitamin B-12 occurs in foods of animal origin, and dietary vitamin B-12 deficiency in otherwise healthy people is due to the total exclusion of such foods from the diet—even so, it can take years for the body's supplies to be depleted.⁴⁵ Folate deficiency is usually associated with the increased physiological demands for erythropoiesis during pregnancy.⁴⁶ These anaemias are

uncommon, although folate and vitamin B-12 deficiency (ICD-9:281) were the recorded cause of death for 11 males and 23 females in 1991.

Nutritional anaemias are more commonly found in Aboriginal children than in non-Aboriginal children^{47,48} and are more common in pregnant women than in other groups.⁴⁷ Older studies suggested a higher rate of anaemias in pregnant women of Italian and Greek origin, although the probable cause was the higher prevalence of the genetically determined condition β -thalassaemia.^{49,50,51}

Iron intakes and iron status: a special concern

Iron intakes

Over 70 per cent of women aged 25–55 years from the 1983 National Dietary Survey had iron intakes below the RDI; 14 per cent of women aged 25–34 years and over 20 per cent of women aged 35–55 years had intakes below 0.5 of RDI.⁵² Approximately 10 per cent of girls aged 12–15 years surveyed in 1985 also had iron intakes less than 0.5 of RDI.²⁵ These groups represent the population of women of reproductive age; they have relatively high physiological requirements, particularly for iron, and therefore higher RDIs.

Iron status of Australian adults, 1989

In 1989 the Department of Community Services and Health conducted an iron status study of a national sample of urban Australian men and women in conjunction with the 1989 Risk Factor Prevalence Study.⁵³ The study was funded by the Australian Meat and Livestock Corporation and the Meat Research Corporation. Plasma levels of iron, transferrin and ferritin were analysed in 1704 men and 4267 women aged 20 to 69 years. The criteria used to define iron deficiency were based on the ferritin and transferrin saturation (TS) model, using two iron status indicators (ferritin < 12 μ g/L and TS < 16 per cent) to increase the sensitivity in categorising a deficiency status.⁵⁴

Ferritin and TS percentage were determined in five-year age groups between 20 and 69 years for both men and women. Iron deficiency was identified in 0.4 per cent of men and 7.5 per cent of women. In women, iron deficiency was greatest during the reproductive years (20–49 years) and markedly less following menopause (50–69 years). The high prevalence of iron deficiency in women of reproductive age is consistent with the prevalence of 9.2 per cent reported in Australian schoolgirls aged 15 years.⁵⁵ Prevalence was greatest in women aged between 40 and 49 years.

Other iron intake and status information

Iron intakes and iron status studies in Australia have been reviewed comprehensively by Cobiac and Baghurst;⁴⁷ Table 5.2 summarises data from the studies they reviewed. The studies reviewed were undertaken in the period 1966–1991. Of these, 12 studies measured iron intakes and 11 measured iron intakes. There is a notable variation in criteria for risk in both intake and status studies; five different methods for collecting intake data were recorded. The variation of age groups studied adds to the difficulty of extracting trends or patterns. It is notable that over nearly 30 years, there have been no data on 3–8-year-olds or 16–19-year-olds. It was possible to conclude that very young children and children aged 12–15 years were most likely to have low iron intakes (of ages for which there were data).

Table 5.2: Summary table, iron intakes and status of Australians

Group	Number	Criteria for risk	% of group at risk	Ref. no.
Children and adolescents				
10–15 years schoolchildren	5224	<70% RDI, 24-hour record	Boys 2–21 Girls 4–30	25
16–17 years girls	154	<67% RDI, 4-day record	18	56
0–3 years healthy	1000	Anaemia, Hb<10g/100mL	3	57,58
0–2 years hospitalised	7408	Anaemia, Hb<9.5g/100mL	12	59
Schoolchildren aged 9, 12, 15 years	1204	ferritin <12µg/L, transferrin saturation <16%	9.2 of 15-year-old girls	55
Adults				
25–64 years urban	6255	<70% RDI, 24-hour recall	M 1–3, F 3–47	52
18+ to 60+ urban and rural	1541	<70% RDI, food frequency	M 1, F 0–13	60
18+ to 60+ urban and rural	3000 ea.	<70% RDI, food frequency	M 0–2, F 0–17	61,62
16–61 years hospital staff	1024	Hb<12g/100mL	Women 2–8	63
20–70 years Busselton	3331	Hb Men <13.5g/100mL Hb Women<11.5g/100mL	Men <1–9 Women 2–5	64
Adults bank/insurance	2000	Ferritin <10µg/L	M 1, F 6	65
20–69 years urban	1704 M 4267 F	Iron<8µmol/L Ferritin men <20µg/L Ferritin women <10µg/L Transferrin sat<10%	M 8*, F 16* Men 2* Women 8* M 4*, F 9*	53
Elderly				
60+ to 80+ years	178	<70% RDI, food frequency	8	66
54+ to 70+ years	1313	<70% RDI, food frequency	M 1, F <1	67
60+ to 80+ years	178	Anaemia	8.3	66
Low income				
Redfern adults	51	<70% RDI, food frequency	16	68
Homeless men	107	<70% RDI, 24-hour recall	17*	69
Migrants				
Vietnamese adult women	201	<70% RDI, 24-hour recall	40	70
Aboriginal people				
Children 0–6 years	121	Hb<10g/100ml, ferritin<10µg/L	12–17	71
Adults	322	Hb<11g/100mL	11	72
Vegetarians				
Adults	117	Hb<12g/100mL Ferritin <15µg/L	M 0, F 7 M 5, F 27	73
Athletes				
Adults	706	Hb M<13, F<12g/100mL	<1–2	74
Mothers				
Adults, some pregnant	419	<67% RDI, 24-hour recall	24–35	75
Pregnancy				
Adult women	84	Iron deficient	10–15	53

* Approximate figures only

Source: Cobiac & Baghurst⁴⁷

Higher prevalences of iron deficiency were found in adolescent girls and other women of reproductive age (particularly vegetarians), the elderly, blood donors, and Aboriginal people living in remote areas.⁴⁷ For example, a 1992 survey of 120 children attending primary school in a central Queensland Aboriginal community showed that 28 per cent of children tested were iron deficient.⁷⁶

There is insufficient information to support the reasonable hypothesis that homelessness is associated with anaemia or to confirm low iron status in some migrant groups with low intakes.⁴⁷

In a study of biochemical indices of iron status (plasma iron, ferritin, transferrin, and transferrin saturation percentage) carried out on 9-, 12- and 15-year-olds from the 1985 National Dietary Survey of Schoolchildren sample, English and Bennett found a prevalence of iron deficiency of 9.2 per cent in 15-year-old girls, 1.6 per cent in 12-year-old girls and 1.7 per cent in 15-year-old boys; the prevalence was 0.5 per cent or less in boys aged 9 and 12 years, and no deficiencies were found in girls aged 9 years.⁵⁵

Telford et al. found no association between plasma ferritin levels and dietary intakes of iron, meat, fat or fibre in 69 non-anaemic athletes.⁷⁷ There was a positive association with protein intake and a negative association with carbohydrate or energy intake.

Chronic blood loss, for whatever reason, is the principal cause of iron deficiency anaemia. One form of chronic blood loss is blood donation. The 1991–92 Australian Red Cross annual report lists the collection of 940 000 donations.⁷⁸ The traditional pint of blood contains about 300 mg of iron; donated six-monthly, this means an additional requirement for a regular donor of about 1 mg per day, which is of the same order as the requirement to replace menstrual blood loss.⁷⁹ This is of most significance where iron intakes are marginally adequate and where requirements are high. Women of reproductive age who donate blood are most at risk of iron deficiency.

A controlled clinical trial of normal, healthy infants, either breastfed or formula fed with substitutes containing either 3 mg per L or 6 mg per L of iron, found that none of the formula-fed infants were iron depleted at nine months, although iron intakes were different. Of the breastfed infants, 13 per cent had depleted iron stores at six months, but only three per cent at nine months.⁸⁰

5.3 Which are the diet-related diseases?

The evidence linking diet with the chronic, preventable, non-communicable diseases is recognised in Australia and internationally to provide sufficient reason for including improved nutrition as a major component of public health initiatives. Table 5.3 lists important review publications dealing with the evidence.

Particularly worthy of attention are several recent Australian publications: the NHMRC's *Dietary Guidelines for Australians*¹ and *The role of polyunsaturated fats in the Australian diet*⁸¹ and the National Heart Foundation's *Diet and coronary heart disease*.⁸²

Rather than establishing a causal link, the evidence to date more appropriately defines a strong association between diet and many of the chronic diseases found in modern, technologically advanced cultures. The major causes of death, illness and disability in Australia that are thought to have a nutrition component in their aetiology and for

which some form of prevention is likely to be applicable are coronary heart disease, stroke, hypertension, atherosclerosis, some forms of cancer, non-insulin-dependent diabetes mellitus (NIDDM), osteoporosis, dental caries, gall bladder disease, non-cancer disorders of the large bowel (such as diverticular disease and constipation) and nutritional anaemias (anaemias are considered in previous sections). Of these conditions, ischaemic heart disease (ICD-9:410–414) was the cause of 31 174 deaths in Australia in 1990, or 26 per cent of all deaths.^{83,84}

Table 5.3: Selected reviews of the diet–disease relationship

Source	Title	Year	Origin	Ref. no.
HHLGCS	Goals and targets for Australia's health in the year 2000 and beyond	1993	Aust	85
NHMRC	Dietary Guidelines for Australians	1992	Aust	1
NHMRC	The role of polyunsaturated fats in the Australian diet. Report of the NHMRC working party	1992	Aust	81
National Heart Foundation	Diet and coronary heart disease	1992	Aust	82
HHCS	Diet-related diseases and health. Review paper	1991	Aust	86
Department of Health	The health of the nation. A consultative document for health in England	1991	UK	87
Department of Health	Food for health. Report of the Nutrition Taskforce	1991	NZ	88
WHO Study Group	Diet, nutrition and the prevention of chronic diseases	1990	WHO	89
Health and Welfare Canada	Action towards healthy eating. Canada's guidelines for healthy eating and recommended strategies for implementation	1990	Canada	90
Department of Health and Human Services	Healthy people 2000	1990	USA	91
NHMRC	Implementing the Dietary Guidelines for Australians. Report of the Subcommittee on Nutrition Education	1989	Aust	92
NHMRC	Appendix XIII. Role of exercise in nutrition and health	1989	Aust	93
National Research Council	Diet and health: implications for reducing diet-related disease	1989	USA	94
DCSH	Health for all Australians report	1988	Aust	95
Department of Health and Human Services	Surgeon General's report on nutrition and health	1988	USA	96
WHO Regional Office for Europe	Healthy nutrition. Preventing nutrition-related disease in Europe	1988	WHO	97
Better Health Commission	Better nutrition for Australians	1986	Aust	26
Better Health Commission	Cardiovascular disease: preventing an unnecessary way of death	1986	Aust	98

The concept of risk

The diseases associated with diet are also associated with multiple environmental, behavioural, biological, social and genetic factors. Assessing the role of diet in ill-health or in promoting better health must include assessing the extent to which changes in health are attributable to diet among other factors such as smoking, stress or inactivity, all of which themselves may be inter-related.

Groups, and individuals within groups, can be at varying risk of nutritional disorders because of different ethnic origin, religion, sex, age, physiological traits, education, occupation, economic circumstances, and so on. Lesser or greater risk may be defined for most groups of individuals; for example, women compared with men, older people compared with younger, women who are pregnant compared with those not pregnant. Whether the risk is sufficient to merit the description 'at risk', 'vulnerable', or 'disadvantaged' is a matter of judgment. Moreover, even when, as in older people, vulnerability would usually be acknowledged, it is the synergy of risk factors other than chronological age that defines an excess of risk for a subgroup relative to the corresponding population.

Although risk factors can be shown to have an independent association with disease, they may also be contributory causes of other risk factors (examples are alcohol intake and hypertension, hypertension and obesity, obesity and NIDDM). The complex aetiology of these conditions makes it difficult to assess the contribution of diet to diet-related disease and health outcomes.

The main causes of diet-related mortality and selected morbid conditions are considered in the remainder of this section.

Cardiovascular disease risk

Coronary heart disease is the major cause of mortality in Australia and in other developed countries. Among the suggested or established risk factors for this disease are saturated fatty acid intakes, *trans*-fatty acid intakes, total fat intakes, high alcohol intakes, total abstinence from alcoholic drinks, raised blood lipids (cholesterol and triglycerides), hypertension, cigarette smoking, overweight or obesity, dietary cholesterol intakes, diabetes mellitus, physical inactivity, psychological stress, and genetic predisposition. Unsaturated fatty acids when substituted for saturated fatty acids in the diet and a high intake of plant foods appear to protect against coronary heart disease.^{83,99} Diet is also linked to most of the non-dietary factors. For example, obesity is a direct risk factor for coronary heart disease, and obesity is itself directly related to diet—and to physical activity, stress, genetic factors, and socioeconomic status and cultural factors.

Hypertension

Hypertension is an independent risk factor for total mortality, coronary heart disease mortality and cerebrovascular disease mortality in both men and women.^{100,101} For total mortality, ischaemic heart disease mortality, and cerebrovascular mortality, systolic blood pressure is a more powerful predictor than diastolic blood pressure.¹⁰¹ Data from the 20-year follow-up of the Framingham Study showed for congestive heart failure a relative risk of 4.9 when hypertensive men aged 45–74 years were compared with normotensive men of the same age group.¹⁰² For hypertensive men compared with

borderline hypertensive men the relative risk was 2.2. For women the corresponding relative risks were 3.6 and 1.9.¹⁰² The Framingham data also indicate that those with borderline systolic hypertension (140–159 mmHg) and normal diastolic blood pressure (<90 mmHg) are at greater risk of progression to cardiovascular disease than those with blood pressure below 140/90 mmHg.¹⁰³ Hypertension is also associated with NIDDM,¹⁰⁴ and with raised serum cholesterol.¹⁰⁵

Studies of children have shown consistent increases in blood pressure with age. In 1977 Gracey et al. reported systolic blood pressures in Busselton (Western Australia): for boys, 108 mmHg at age 13 years, 121 mmHg at age 15 years and 129mmHg at age ≥17 years; for girls, 109mmHg at 13 years, 113 mmHg at 15 years, and 116 mmHg at ≥17 years.¹⁰⁶ Children in Geelong (Victoria) aged 15 years in 1987 had systolic blood pressures of 123 mmHg for boys and 114 mmHg for girls.¹⁰⁷

Systolic and diastolic blood pressures in children participating in the 1985 Australian Council for Health, Physical Education and Recreation survey were highest in 15-year-olds and lowest in 9-year-olds.¹⁰⁸ A Perth study showed a weak but statistically significant inverse relationship between diastolic blood pressure and energy intake in a sample of 526 11–12-year-old boys, but not for 504 girls, and not with systolic blood pressure for either sex. Waist-to-hip ratio and level of fitness were not associated with blood pressure measures; weight and body mass index were the better predictors of blood pressure in children at this age.¹⁰⁹ A South Australian survey found that systolic blood pressure in girls aged over 13 years changed little, but that boys over 13 years exhibited a steady increase with age.¹¹⁰

Hypercholesterolaemia

Good scientific evidence exists to support a link between blood cholesterol level and the development and progression of atherosclerosis and subsequent coronary heart disease.^{111,112} For example, data from the Multiple Risk Factor Intervention Trial (MRFIT) 12-year follow-up show a strong, continuous and graded relationship between serum cholesterol and coronary heart disease mortality,¹¹³ and an earlier MRFIT analysis reported that a 1 per cent higher serum cholesterol was independently associated with an almost 2 per cent higher coronary heart disease risk.¹¹⁴ Other studies have also shown a strong, graded positive relationship between serum cholesterol and cardiovascular disease mortality independent of other risk factors.^{115–117} In addition, lipid fractions within total cholesterol have been identified as separate indicators of risk: lower HDL (high-density lipoprotein) cholesterol and raised LDL (low-density lipoprotein) cholesterol concentrations are significant predictors of cardiovascular disease mortality in both men and women, including those without pre-existing cardiovascular disease.^{118–120}

The only recent source of data for children is the Australian Health and Fitness Survey conducted in 1985 (see Table 5.4). Fifty per cent of boys aged 9 and 12 years had cholesterol concentrations above the recommended limit of 4.5 mmol/L, while over 50 per cent of girls aged 9, 12 and 15 years had total cholesterol levels of 4.47 mmol/L or more.²⁴

The mean plasma cholesterol levels reported from the 1977 Busselton Children's Survey were higher for girls than for boys: for 15-year-olds they were 4.71 mmol/L for boys and 4.97 mmol/L for girls.¹⁰⁶ In the 1987 Geelong study the levels for 15-year-olds were 4.01 mmol/L for boys and 4.34 mmol/L for girls.¹⁰⁷

Non-insulin-dependent diabetes mellitus

Non-insulin-dependent diabetes mellitus is a chronic metabolic disorder to which some of the population is genetically predisposed and the expression of which may be initiated by obesity, physical inactivity or stress.^{121,122} Many diet-related factors commonly present in individuals with diabetes may lead to increased risk of coronary heart disease; for example, hypercholesterolaemia, hypertriglyceridaemia and hyperinsulinaemia.^{105,123} In people of European origin, onset usually occurs in middle adulthood and its prevalence is reported to be from 1.1 per cent¹³ to 2.9 per cent.¹²⁴ Prevalence of the prodromal condition, impaired glucose tolerance, was reported to be 6.3 per cent in a 1981 study in Busselton.¹²⁴ Haffner et al. found that in a subsample of non-diabetic individuals from the San Antonio Heart Study higher fasting levels of insulin and pro-insulin were positively associated with serum triglyceride, systolic blood pressure, body mass index and waist-to-hip ratio and negatively associated with HDL cholesterol.⁴²

Table 5.4: Fasting total cholesterol, percentiles for children ages 9, 12 and 15 years, by sex, (mmol/L), 1985

	Boys			Girls		
	Age 9	Age 12	Age 15	Age 9	Age 12	Age 15
Sample size (number)	371	349	291	348	307	253
Mean total cholesterol	4.35	4.31	3.92	4.42	4.33	4.19
Standard deviation	1.22	1.21	1.03	1.27	1.38	1.35
Percentile						
5th	3.53	3.44	3.13	3.39	3.53	3.52
10th	3.69	3.65	3.22	3.84	3.76	3.72
25th	4.11	4.05	3.62	4.28	4.18	3.99
50th	4.55	4.51	4.02	4.65	4.65	4.47
75th	4.99	4.97	4.57	5.08	5.02	4.91
90th	5.53	5.56	4.96	5.59	5.57	5.40
95th	5.78	5.82	5.19	5.89	5.90	5.59

Source: Australian Council for Health, Physical Education and Recreation²⁴

The national organisation Diabetes Australia estimates a prevalence of 4 per cent (including insulin-dependent diabetes) in the general population, rising to 11 per cent in those aged over 65 years.¹²⁵ In this latter group it is to be expected that almost all will have NIDDM. Diabetes Australia also believes that of an estimated 500 000 Australians with diabetes, as many as half remain undiagnosed.¹²⁵ People of Aboriginal origin may be genetically more susceptible to NIDDM than people of European origin although lifestyle and environmental factors are also important. The average age of onset is lower and its prevalence higher in Aboriginal people.¹²⁶⁻¹²⁸ Prevalence has been reported to be from 4.5 per cent to 19 per cent in Aboriginal populations,¹²⁶ and a high prevalence of impaired glucose tolerance has also been reported.^{129,130}

Obesity is the major risk factor for NIDDM. Colditz et al.¹³¹ found from the Nurses Health Study cohort aged 30-55 years that even for women of average weight (with a BMI of 23-23.9 kg/m²) risk of NIDDM increased with increasing BMI. A direct

association has also been observed in men. Lapidus et al. found an independent association between a family history of diabetes and BMI in a 12-year follow-up of 1400 Swedish women; the association was with overall rather than abdominal obesity.¹³²

Diet is also the principal mode for management of NIDDM and, because the onset of the disorder is closely associated with overweight, a diet consistent with the Dietary Guidelines for Australians and increased physical activity are recognised components of therapy. A diet high in fibre lowers plasma glucose levels in both diabetic and non-diabetic individuals and significantly reduces the insulin requirements of diabetic individuals.^{87,105} Physical activity has a beneficial effect on insulin metabolism and can improve glucose tolerance by reducing overweight.⁸⁷

Preventable cancers

Food factors may be associated with carcinogenesis as protective factors or risk factors, and the mechanism of interaction may be direct or through promotion of the action of other carcinogens. The main components of diet associated with risk are high fat or alcohol intakes, low fibre intakes and high salt intakes. The actions of food components such as fat, fibre, vitamin A or calcium are dose-dependent and reversible.¹³³ The active components may be a result of cooking or otherwise processing food. For example, heterocyclic arylamines, generated in the browning of meat or fish, are possible causative carcinogens. Another example is the association of risk with foods high in nitrates and other nitrogen radicals either from processing or from soils with high nitrate levels.¹³³

Meta-analyses by Steinmetz and Potter¹³⁴ and Trock et al.¹³⁵ indicate an inverse association between cancer (particularly cancers of the alimentary tract) and vegetable and fruit consumption. Giovannucci et al.,¹³⁶ in a prospective study of 7284 males of whom 170 developed colon or rectal cancer, found a positive association with saturated fat intake and with the ratio of red meat to chicken and fish and an inverse association with dietary fibre intake. Cancers of the colon, rectum and anus have been shown to have a positive relationship with the consumption of meat and animal fats and high energy intakes, while vegetable fibre, cereal bran and calcium are believed to reduce risk.¹³ Stomach cancer is also thought to be associated with salt intake. Its incidence is declining in Australia as a result of refrigeration and it is believed that the decline in consumption of salted, pickled and smoked food and the increase in fruit and vegetable consumption are responsible.

Breast cancer risk has shown positive associations with excess alcohol and animal fat consumption.¹³⁷ A case-control study conducted between 1983 and 1985 found a strong correlation between increased risk of fibroadenoma of the breast and increasing BMI, and between decreased risk and increasing vitamin C intake. There was no statistically significant association with energy, total fat, fibre, β -carotene or alcohol intake (fibroadenoma may be a precursor of breast cancer).¹³⁸

In case-control studies of diet and pancreatic cancer using a common method and undertaken in Australia (Adelaide), Canada (Montreal and Toronto), Holland (Utrecht) and Poland (Opole), positive associations were found with carbohydrate and cholesterol intakes and inverse associations with dietary fibre and vitamin C.¹³⁹

Diets high in carotenoid-containing plant foods may be associated with lower rates of colon, oesophageal, stomach and lung cancers.⁹⁴ Mono-unsaturated fatty acids,

n-3 (ω -3) polyunsaturated oils, cereal fibre, pectins and medium-chain triglycerides may be protective against endocrine-related, prostate, breast, endometrial or ovarian cancers, while saturated and n-6 (ω -6) polyunsaturated lipids are risk factors.¹³³ The mode of action of lipids is in their effect on the secretion of bile acids, key promoters of colon cancer.¹³³ In a case-control study of diet and colon cancer in Adelaide there was a positive association with egg consumption in females and weaker positive associations with red meat, liver, seafood and dairy products.¹⁴⁰ The associations in men were with red meat and poultry. An inverse association was found with onions, cabbage and legumes and a positive association with potatoes for men and women.¹⁴¹ Intakes of green leafy vegetables and carrots were associated with decreased cancer risk in men.¹⁴¹

Dental caries

Based on several major reviews undertaken between 1986 and 1990, the NHMRC Panel to revise the dietary guidelines concluded that dental caries are associated with the amount and frequency of consumption of foods containing non-milk added sugars. Foods such as confectionary bars, biscuits and some extruded snack food products were cariogenic.¹

Osteoporosis

This condition of reduced bone mass predisposes to fractures, particularly of the upper thigh and spine, even under little stress. Dietary factors that can affect the risk of osteoporosis are calcium, vitamin D, salt, alcohol, animal protein and caffeine.¹⁴² It is postulated that adequate calcium intake during growth and until the mid-twenties, when peak bone mass is achieved, reduces risk of osteoporosis, and an adequate intake in later life allows for the increased rate of bone loss and less effective assimilation from the diet; the role of dietary calcium in the treatment of osteoporosis is still uncertain.¹⁴²

Bone density decreases later in life in both sexes, but post-menopausal women are at the greatest risk of developing osteoporosis, because oestrogens protect against bone loss. A 1990–91 study of Sydney women aged 65 years and over suggested that breastfeeding protected against hip fracture in old age, with a dose-response relationship between average duration of breastfeeding per child and risk of hip fracture.¹⁴³ It is estimated that 20–25 per cent of women, by the age of 70 years, will have undergone hospitalisation for bone fractures resulting from osteoporosis, and that 16 per cent of those with hip fractures will die within six months.¹⁴²

Box 5.3: Nutrition and infectious diseases

Two very different kinds of infectious diseases of worldwide importance are linked to nutrition and of current interest in Australia: human immunodeficiency virus (HIV) infection and acute gastrointestinal infection.

Nutrition and HIV

The links between HIV infection and nutrition include optimum nutritional status to maintain normal immune function as long as possible, the importance of good nutrition in maintaining health and extending the asymptomatic period, and appropriate nutritional support for those who develop acquired immuno-deficiency syndrome (AIDS). The role of nutrition in HIV disease has been reviewed in detail by Oliver and Hyder.¹⁴⁴

HIV-positive people are particularly vulnerable to exploitation. There is a need for accurate information and targeted education to avoid reliance on, and the unnecessary costs of, inappropriate diets and unproven prophylactic nutrient supplementation regimes that may replace adherence to a suitable diet. The NHMRC noted that 'many unscientifically based nutrition regimens had been claimed to be effective in the management of patients with [HIV] infections [and] it was agreed that sound nutritional advice was needed to counter such claims'; it recommended that people infected with HIV have access to scientifically sound advice from qualified dietitians.¹⁴⁵

Nutrition and diarrhoeal disease

The relationship between diarrhoeal disease and nutrition has been summarised by Dr Michael Gracey of the Aboriginal Health Unit, Health Department of Western Australia:

Diarrhoeal illnesses have significant deleterious effects on nutrition, growth and development in infants and young children through the combined effects of loss of nutrients, suppression of appetite, vomiting, interruption of normal feeding practices and the catabolic effects of infection. Compromised nutritional status, in turn, makes affected children more prone to gastro-intestinal infections.¹⁴⁶

In Australia, diarrhoeal disease is found mainly in children under five years old and is still much more prevalent in Aboriginal children than in non-Aboriginal children. In Aboriginal children it is often complicated by other concurrent infections and by undernutrition.¹⁴⁶

Mortality due to intestinal infectious disease (ICD-9:001-009) is declining: age-specific rates in the first year of life in 1979 were 115 per million for male infants and 46 per million for female infants; in 1990 the rates were 8 per million for males and females.⁸³ The age-standardised death rate stood at 2 per million in 1990. The elderly are also vulnerable.

Acute gastrointestinal infection—diarrhoeal disease—is the larger public health