3 Determinants of health

An overview of health determinants in the wider context of health was shown in Figure 1.1 in Chapter 1. This chapter begins by discussing health determinants in general, their major types, and their relationships to health and to each other. It then provides information on the levels, trends and other patterns of those determinants for which there are adequate data.

What are health determinants?

The health of individuals and populations is influenced and determined by many factors acting in various combinations. The dominant view is that health is 'multicausal': healthiness, disease, disability and, ultimately, death are seen as the result of the interaction of human biology, lifestyle and environmental (including social) factors, modified by health interventions.

Health determinants can be described as those factors that raise or lower the level of health in a population or individual. Determinants help explain and predict trends in health and explain why some groups have better or worse health than others. They are the key to the prevention of disease, illness and injury.

Determinants may have positive or negative impacts. Factors such as cigarette smoking or low socioeconomic status increase the risk of ill health and are commonly termed 'risk factors'. Positive influences such as a high intake of fruit and vegetables are known as 'protective factors'. Unlike behaviours, some determinants such as age and sex clearly cannot be altered. Advancing age is associated with a greater risk for many diseases. Numerous diseases are sex-specific and for many others sex can be either a risk factor or a protective factor. Health interventions are covered in Chapter 6 and are not considered here.

For almost all risk and protective factors the associated effect is not 'all or nothing'. For risk factors, rather than there being one point at which risk begins, there is an increasing effect as the exposure increases. For example, each increment in a person's body weight above their 'optimal' level is associated with an increase in the risk of ill health. Although the increasing risk often starts at relatively low levels, the usual practice is to monitor a risk factor by reporting the proportion at the riskier end of the spectrum.

A framework for determinants

Determinants are in complex interplay and range from the very broad level, with many health and non-health effects, to the highly specific. They are often described as a web of causes, but they can also be thought of as part of broad causal 'pathways' or 'chains' that affect health. Figure 3.1 is a simple framework of determinants and their pathways, with the general direction of effects going from left to right.

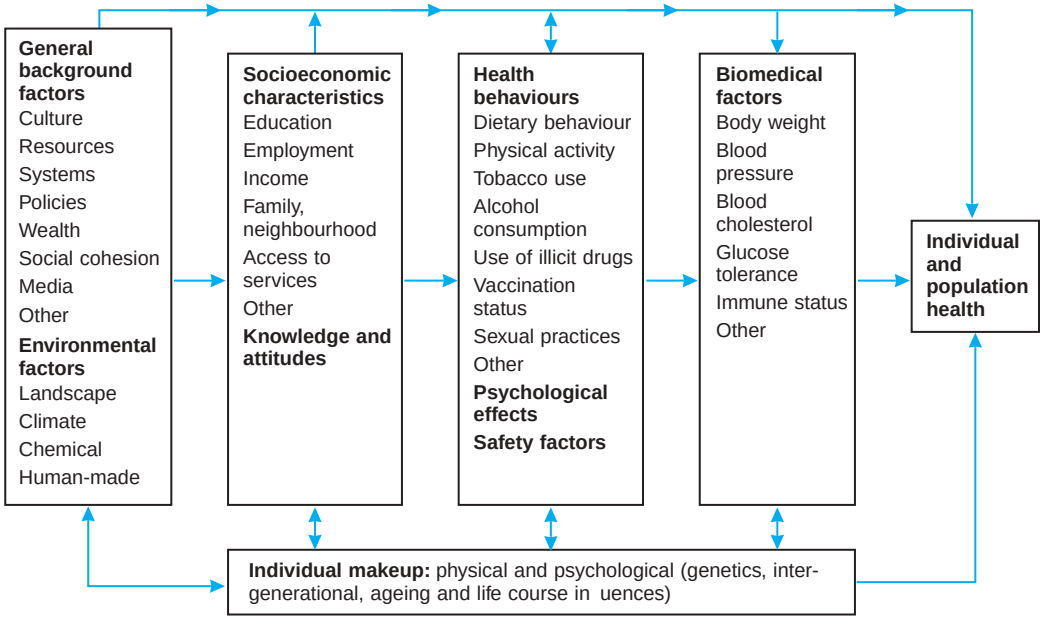


Figure 3.1: A conceptual framework for determinants of health

General background and environmental factors can determine the nature and degree of socioeconomic characteristics (left-hand boxes) and both can influence people's health behaviours, their psychological state and factors relating to safety (middle). These in turn can influence biomedical factors (shown at right), such as blood pressure and body weight, which may have health effects through various further pathways. At all stages along the path these various factors interact with an individual's makeup (bottom box). In addition, the factors within a box often interact and are highly related to each other. (Despite the general direction of these influences they can occur in reverse. For example, an individual's health can also influence their physical activity levels, employment status and wealth.) These groups of determinants are now discussed briefly.

General background factors (left-hand box) are those factors that affect virtually all people in society to some extent, although to varying degrees. These factors combine to influence the basic levels of security, safety, hygiene, nourishment, technology, information, freedom and morale of societies. It is difficult to put values and quantities on most of these broad factors, let alone measure them and assess their impact precisely. However, it is widely agreed that, at least up to a fair degree of societal development, they are a vital determinant of a population's health. They set the background level around which variations then occur between groups and individuals.

Environmental factors are confined here to the physical environment, such as the climate, the land, plant and animal life, and human-made factors such as chemical pollution and waste products. Among many things, these can affect a society's supply of primary goods,

and therefore its wealth, and they can influence where and how people live and spend their time. Large-scale environmental disruptions, such as human-induced climate change, can have major health implications in the longer term as well as in the short term.

Variations in **socioeconomic characteristics** are influenced by society's policies, structures and history, and can be affected by environmental factors as well. These variations can in turn lead to marked variations in health. Differences in people's levels of education and income, for example, along with attitudes and values gained from their families and social settings, can lead to strong differences in the opportunities and choices that affect their health.

Both general background factors and varying socioeconomic factors influence people's **health behaviours** and can have significant **psychological effects** and influence on **safety factors** (middle box). A particular health behaviour such as an individual's diet, for example, can result partly from the general availability and range of foods due to the system as a whole. It can also reflect a person's 'inherent' preferences modified by both cultural and family influences. Finally, it may further reflect the person's financial and political freedom to exercise those preferences.

A person's psychological state and behaviour clearly affect each other and both can in turn lead to biomedical changes or disease. Diseases such as asthma, for example, are believed to be often influenced by psychological factors (AIHW: Australian Centre for Asthma Monitoring 2003). It is also argued that various stressful aspects, such as depression, being socially isolated or less free to make decisions, can lead to problems such as heart disease, independent of any intermediary behavioural effects (Bunker et al. 2003).

As with all the factors mentioned, health behaviours tend to interact with each other and to influence a variety of **biomedical factors** (right-hand box). Both physical activity and diet, for example, can affect body weight, blood pressure and blood cholesterol. They can do this alone or, with greater effect, together. The biomedical factors listed in the box are only a selection and they are often highly interrelated. Excess body weight, high blood pressure and high blood cholesterol can all contribute to the risk of heart disease and amplify each other's effects if they occur together. In addition, obesity can in itself lead to high blood pressure and high blood cholesterol. Biomedical disturbances do not lead to ill health automatically or in a single step. They can pass through various further biological pathways before producing outward disease, providing further points for intervention. Both behavioural and biomedical risk factors tend to increase each other's effects when they occur together in an individual.

Finally, it is important to note that determinants act upon and are influenced by an **individual's makeup**, both physical and mental (bottom box). This makeup can greatly modify a person's response to other new or continuing determinants. It can be seen as the complex product of a person's genetic endowment, inter-generational effects, their ageing, and physical or social influences at various stages over their life course. These influences can become built into a person's makeup for various periods or for life. Some diseases, such as muscular dystrophy, result entirely from a person's genetic features, whereas most others reflect the interaction between those features and the many other influences mentioned here.

Information in this chapter is presented in four sections which correspond to those in Figure 3.1. These are biomedical and genetic factors, health behaviours, socioeconomic

factors and environmental factors. In each of these sections, available information on specific determinants is presented. However, the population health impact of individual risk factors varies, depending on their frequency in the population and relative effect on individual people. Therefore, Table 3.1 aims to give an overall perspective by providing an estimate of the relative importance in 1996 of most of the determinants included in this chapter. From this, tobacco smoking was estimated to cause the most premature death and illness, followed by physical inactivity and high blood pressure.

Table 3.1: Proportion of total disease burden attributed to selected risk factors, 1996

Risk factor	Males	Females
	(per cent)	
Tobacco smoking	12.1	6.8
Physical inactivity	6.0	7.5
High blood pressure	5.1	5.8
Alcohol harm	6.6	3.1
Alcohol benefit	–2.4	–3.2
Overweight	4.4	4.3
Lack of fruit/veg.	3.0	2.4
High blood cholesterol	3.2	1.9
Illicit drugs	2.2	1.3
Unsafe sex	1.1	0.7

Note: Attributable disability-adjusted life years (DALYs) as a proportion of total DALYs. One DALY equals one year of healthy life lost through premature death or living with disability due to illness or injury.

Source: AIHW: Mathers et al. 1999.

3.1 Biomedical factors

Body weight

The prevalence of obesity has risen dramatically worldwide and the World Health Organization (WHO) has called the increase a global epidemic. Australia is no exception to this increase.

Overweight, and in particular obesity, is associated with higher mortality and morbidity. Excess body fat increases the risk of developing a range of health problems including Type 2 diabetes, cardiovascular disease, high blood pressure, certain cancers, sleep apnoea, osteoarthritis, psychological disorders and social problems (WHO 2000). Children and adolescents who are overweight also have an increased risk of health problems. They have a greater likelihood of becoming overweight adults and of developing conditions such as Type 2 diabetes (Ludwig & Ebbeling 2001).

At the other end of the weight spectrum is underweight. While underweight and associated malnutrition is mainly a problem in developing countries, being underweight in developed countries is also associated with poor health.

Overweight was estimated to account for 4.3% of the total burden of disease in Australia in 1996 (AIHW: Mathers et al. 1999). The study that made this estimate did

not consider underweight, as it is not a common problem in Australia. Globally, childhood and maternal underweight is the risk factor responsible for the largest burden of disease, with overweight ranking tenth (Ezzati et al. 2002).

Overweight arises through an energy imbalance over a sustained period of time. While many factors may influence a person's weight, weight gain is essentially due to the energy intake from the diet being greater than the energy expended through physical activity. The energy imbalance need only be minor for weight gain to occur, and some people—due to genetic and biological factors—may be more likely to gain weight than others (WHO 2000). For more information, see the sections on dietary behaviour and physical activity in this chapter.

Prevalence and trends

Trends in body weight—whether based upon body mass index (BMI: see Box 3.1) or waist circumference—show that the prevalence of overweight and obesity has been increasing at an alarming rate over the last two decades. Comparisons of population groups show that all Australians are affected: children and adults, men and women, Indigenous and non-Indigenous Australians, and people from all socioeconomic backgrounds. Some groups are worse off than others, in particular Aboriginal and Torres Strait Islander people and people from the most disadvantaged socioeconomic groups.

Box 3.1 outlines issues in measuring and reporting on statistics on body weight. In the following section, results are from a number of surveys, using BMI derived from self-reported and measured height and weight, and waist circumference. Data sources are detailed in Table 3.2.

Table 3.2: Body weight data sources

Survey	Scope (as presented)	Measure of weight
1989–90 and 2001 National Health surveys	National, 18 years and over	Self-reported BMI
1980, 1983 and 1989 Risk Factor Prevalence surveys	Capital cities (states only for 1980, 1983), 25–64 years	Measured BMI Waist circumference
1995 National Nutrition Survey	Capital cities and other urban areas, 25–64 years	Measured BMI
1999–2000 Australian Diabetes, Obesity and Lifestyle Study	National, 25 years and over (for prevalence) Capital cities (excluding ACT), 25–64 years (for trends)	Measured BMI Waist circumference

Results based on self-reported data

The most recent national data based on self-reported height and weight come from the 2001 National Health Survey (NHS). From this survey, 2.4 million Australian adults were estimated to be obese (16% of men and 17% of women aged 18 years and over, with a BMI of 30 or more). A further 4.9 million Australian adults were estimated to be overweight but not obese (42% of men and 25% of women aged 18 years and over, with a BMI of 25 or more but less than 30). Only 1% of men and nearly 5% of women were considered to be underweight (a BMI less than 18.5).

Box 3.1: Classifying body weight

There are two main methods used for monitoring body weight: body mass index and waist circumference.

Body mass index

The most common population-level measure of body weight is the body mass index (BMI). BMI is calculated by dividing weight in kilograms by the square of height in metres (kg/m^2). Classifications of body weight are based primarily on the association between BMI and illness and mortality, and are the standard recommended by the WHO and are included in the National Health Data Dictionary.

Weight categories for adults aged 18 years and over based on BMI are:

- *underweight (BMI < 18.5)*
- *healthy weight (BMI ≥ 18.5 and BMI < 25)*
- *overweight (BMI ≥ 25)*
  - *overweight but not obese (BMI ≥ 25 and BMI < 30)*
  - *obese (BMI ≥ 30).*

This classification may not be suitable for all ethnic groups, who may have equivalent levels of risk at lower BMI (for example Asians) or higher BMI (for example Polynesians).

For children and adolescents aged 2–17 years, Cole et al. (2000) have developed a separate classification of overweight and obesity based on age and sex.

Waist circumference

For monitoring overweight, waist circumference is a useful addition to BMI because abdominal fat mass can vary greatly within a narrow range of total body fat or BMI. The National Health Data Dictionary *defines waist circumference cut-offs for increased and substantially increased risk of ill health. Waist circumferences of 94 cm or more in men and 80 cm or more in women indicate increased risk (referred to here as abdominal overweight). Waist circumferences of 102 cm or more in men and 88 cm or more in women indicate substantially increased risk (referred to here as abdominal obesity). This classification is not suitable for use in people aged less than 18 years and the cut-off points may not be suitable for all ethnic groups.*

Self-reported versus measured data

BMI is more commonly used than waist circumference as a measure of overweight and obesity in the population (particularly in self-report surveys), as people are more likely to know their height and weight than their waist circumference.

Height and weight data may be collected in surveys as measured or self-reported data. Previous comparisons have shown that people tend to overestimate their height and underestimate their weight, leading to an underestimate of BMI. Thus, rates of overweight and obesity based on self-reported data are likely to be underestimates of the true prevalence, and should not be directly compared with rates based on measured data.

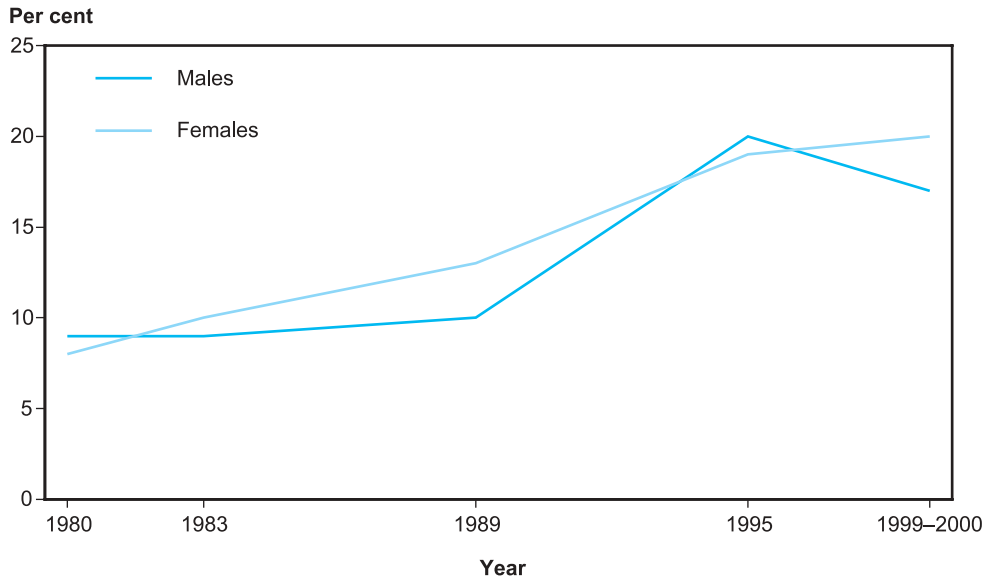
Sources: ABS 1998; AIHW: Waters 1993; Cole et al. 2000; Flood et al. 2000; NHDC 2003; Niedhammer et al. 2000; WHO 2000.

The prevalence of overweight increased significantly over time. At the more severe end of the spectrum—obesity—the prevalence increased from 9% to 16% in men and from 10% to 17% in women between 1989–90 and 2001.

Results based on measured data

Measured height and weight were collected in the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Analysis of this survey found that 19% of men and 22% of women aged 25 years and over were obese (a BMI of 30 or more) and an additional 48% of men and 30% of women were overweight but not obese (a BMI of 25 or more but less than 30). The prevalence of underweight (a BMI less than 18.5) was less than 1% for men and nearly 2% for women.

Trends in the urban population of Australia show that between 1980 and 1999–2000, the proportion of men aged 25–64 who were obese rose from 9% to 17%. Over this same period, the rate of obesity among women aged 25–64 increased from 8% to 20% (Figure 3.2).



Notes

1. Age-standardised to the 2001 Australian population.
2. Urban areas only.
3. People aged 25–64 years.
4. Based on measured data.

Source: AIHW analysis of the 1980, 1983 and 1989 Risk Factor Prevalence surveys, the 1995 National Nutrition Survey and the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study.

Figure 3.2: Proportion of adults who are obese (BMI of 30 or more), 1980 to 1999–2000

Comparison of results based on self-reported and measured data

The results given above based on self-reported and measured data cannot be directly compared: as well as systematic differences in the two types of data (mentioned in Box 3.1), the two data sets here apply to different age ranges and the prevalence of overweight relates to age. However, additional analysis of the 2001 NHS shows that 61% of men and 45% of women aged 25 years and over were classified as overweight (a BMI of 25 or more) based on self-reported information. Results from the 1999–2000 AusDiab showed that 67% of men and 52% of women aged 25 years and over were overweight based upon measured BMI. These differing results support previous studies which showed that rates of overweight derived from self-reported data are likely to be underestimates (see Box 3.1).

Results based on waist circumference

Abdominal obesity is an independent risk factor for Type 2 diabetes, coronary heart disease and other health disorders (WHO 2000). Waist circumference is a useful indicator of abdominal fat. More than a quarter of men (27%) (waist circumference of 102 cm or more) and over a third of women (34%) (waist circumference of 88 cm or more) aged 25 years and over were classified as abdominally obese in 1999–2000. A further 28% of men and 22% of women were classified as abdominally overweight but not obese (a waist circumference of 94 cm or more but less than 102 cm for men, and 80 cm or more but less than 88 cm for women).

Trends in the urban population of Australians aged 25–69 years show that from 1989 to 1999–2000 the prevalence of abdominal obesity increased from 14% to 21% in men and from 16% to 28% in women (AIHW: Dixon & Waters 2003).

Comparisons by population groups

Sex and age

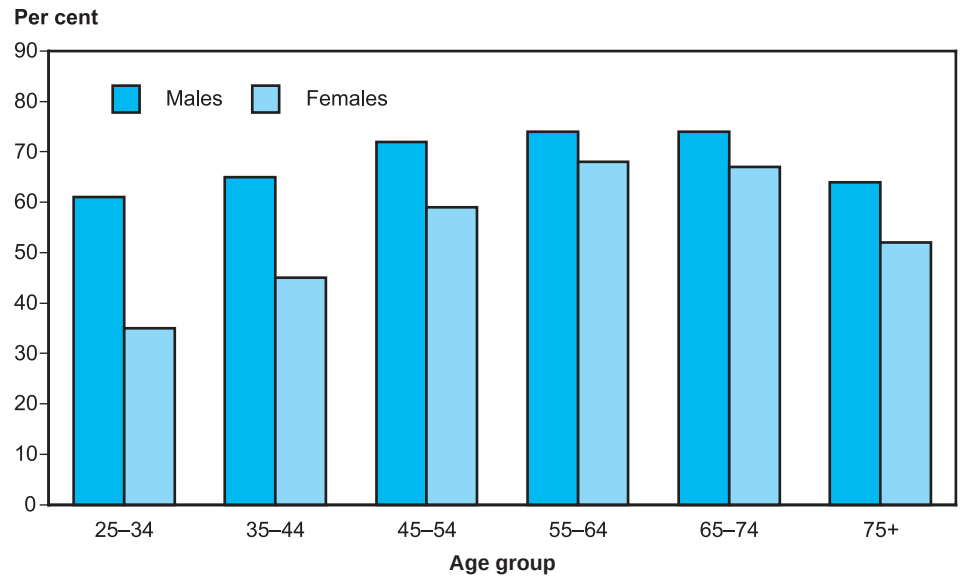
Estimates based on BMI from the 1999–2000 AusDiab showed that men were more likely than women to be overweight (67% versus 52%), although rates of obesity were similar (19% for men and 22% for women).

Among adults, the prevalence of obesity was highest among 55–64-year-olds (29%) with the lowest rates in 25–34-year-olds (15%) and people aged 75 years and over (14%). Results for all overweight people show a similar pattern, with the prevalence of overweight increasing with age up to 65–74 years, and declining thereafter (Figure 3.3).

Older Australians who are obese are a group of particular concern as excess weight may impair mobility, participation in social activities and mental health. Unlike age patterns in BMI, the prevalence of abdominal obesity as measured by waist circumference continues to rise with increasing age (AIHW: Bennett et al. 2004).

Children and adolescents

In 1995, the prevalence of overweight among children and adolescents aged 2–18 years was 19.5% for boys and 21.1% for girls. The proportion of overweight was greatest among boys aged 12–15 years (26.1%) and girls aged 7–11 years (23.5%) (Magarey et al. 2001).



Note: Based on measured data.

Source: AIHW analysis of the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study.

Figure 3.3: Proportion of people who are overweight (BMI of 25 or more), 1999–2000

For children and adolescents aged 7–15 years, obesity increased from 1.4% of boys and 1.2% of girls in 1985 to 4.7% of boys and 5.5% of girls in 1995. The proportions of boys and girls that were overweight but not obese also increased from 1985 to 1995: from 9.3% to 15.3% for boys and from 10.6% to 16.0% for girls (Magarey et al. 2001).

Socioeconomic status

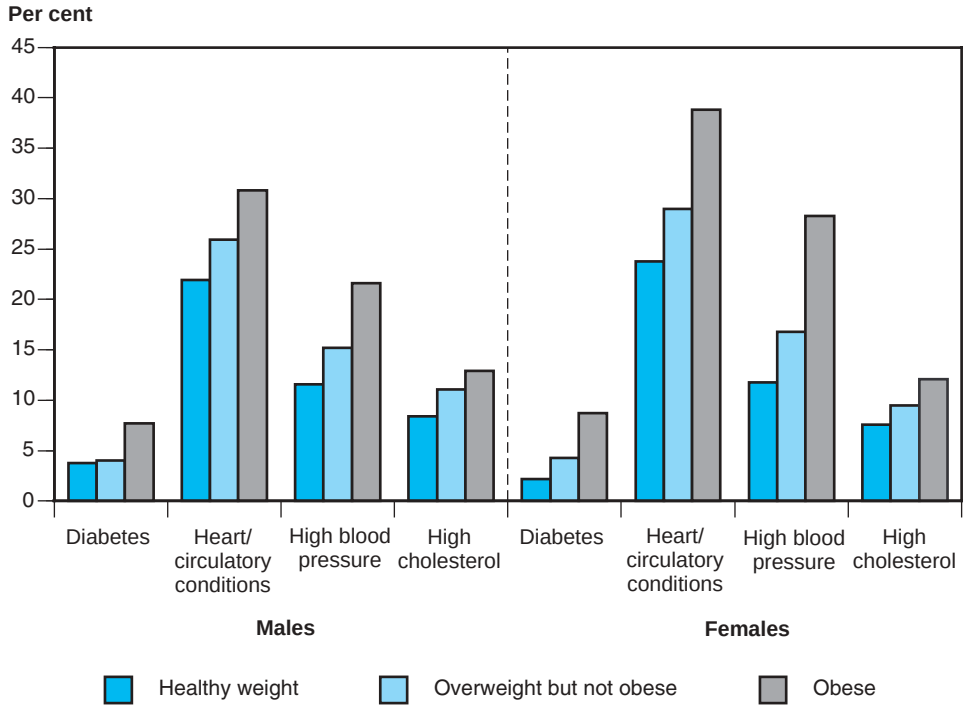
Estimates from the 2001 NHS for adults aged 20 years and over showed that women in the most disadvantaged socioeconomic group had nearly double the rate of obesity (23%) of those in the most advantaged group (12%). Men in the most disadvantaged group were also more likely to be obese than those in the most advantaged group (19% compared with 13%) (AIHW: O'Brien & Webbie 2003).

Aboriginal and Torres Strait Islander people

The proportions of people that were overweight but not obese was similar for Indigenous people and other Australians in 2001 (32% and 34% respectively), unchanged from 1995 for both of these groups. However, Aboriginal and Torres Strait Islander people were almost twice as likely to be obese—31% of Indigenous Australians compared with 16% of non-Indigenous Australians. A marked increase in the prevalence of obesity was seen over this period for both Aboriginal and Torres Strait Islander people (from 24% to 31%) and other Australians (from 12% to 16%). These results are based upon BMI derived from self-reported height and weight and apply to Australians living in non-remote areas only (AIHW: O'Brien & Webbie 2003).

Long-term health conditions

Diabetes was reported four times as often among obese women than among healthy weight women (8.7% and 2.2% respectively) in the 2001 NHS. Obese men were twice as likely to report diabetes as healthy weight men (7.7% compared with 3.8%) (AIHW: O'Brien & Webbie 2004). Similarly, in 2001 heart and circulatory conditions were reported more often among obese men and women than their healthy weight counterparts. In particular, obese people were more likely to report having high blood pressure than people of healthy weight (28.3% compared with 11.8% for women, 21.6% versus 11.6% for men) and high blood cholesterol was reported more commonly among obese people than those of healthy weight (Figure 3.4). These survey data do not allow conclusions to be made about cause and effect but they do highlight the excess burden of poor health experienced by overweight and particularly obese Australians.



Notes

1. Age-standardised to the 2001 Australian population.
2. Diabetes, high blood pressure and high blood cholesterol results for persons aged 25 years and over. Heart/ circulatory conditions for persons aged 20 years and over.
3. Based on self-reported data.

Source: AIHW analysis of the 2001 National Health Survey.

Figure 3.4: Proportion of adults with selected conditions by BMI category, 2001

International comparisons

While there is a large amount of international data on body weight, it is difficult to find directly comparable information. However, comparable self-reported data show that the prevalence of obesity among Australian adults aged 25–64 years (18%) is lower than for adults of the same age range in the United Kingdom or United States (both 22%). Data based on measured height and weight indicate that the prevalence of obesity is slightly higher among Australian adults aged 25–64 years (20%) than among adults of the same age in New Zealand (18%) but is twice as high as in Italy (9%) (AIHW: Dixon & Waters 2003).

Blood pressure

High blood pressure (also referred to as hypertension; see Box 3.2) is a major risk factor for coronary heart disease, stroke, heart failure and kidney failure. The risk of disease increases as the level of blood pressure increases. When high blood pressure is controlled, the risk of cardiovascular disease is reduced, but not necessarily to the levels of unaffected people.

Box 3.2: High blood pressure

Blood pressure represents the forces exerted by blood on the wall of the arteries and is written as systolic/diastolic (for example 120/80 mmHg, stated as '120 over 80'). Systolic blood pressure reflects the maximum pressure in the arteries when the heart muscle contracts to pump blood. Diastolic blood pressure reflects the minimum pressure in the arteries, when the heart muscle relaxes.

There is a continuous relationship between blood pressure levels and cardiovascular disease risk. This makes the definition of high blood pressure somewhat arbitrary. The WHO defines high blood pressure as:

- *systolic blood pressure of 140 mmHg or more; or*
- *diastolic blood pressure of 90 mmHg or more; or*
- *receiving medication for high blood pressure.*

In this report high blood pressure is defined using these guidelines.

Source: WHO-ISH 1999.

Major causes of high blood pressure include diet (particularly a high salt intake), obesity, excessive alcohol consumption and insufficient physical activity. Whether sustained psychological stress has a direct effect on blood pressure levels is subject to further research, but stress is likely to have indirect effects by influencing harmful health behaviours associated with high blood pressure (WHO 2002).

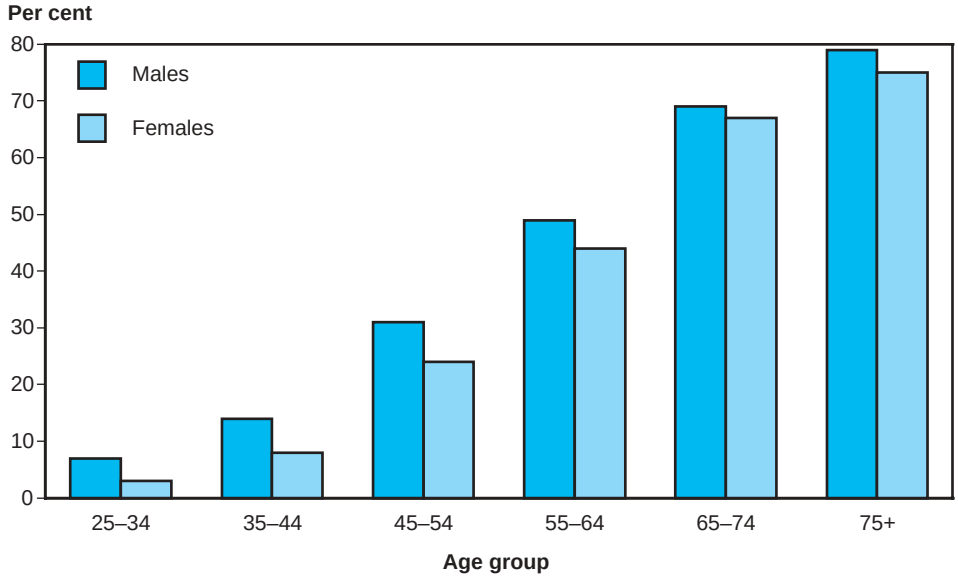
Despite the definition of high blood pressure in Box 3.2, there is in fact no threshold level of risk. Starting from quite low levels, as blood pressure increases so does the risk of stroke, heart attack and heart failure. Both systolic blood pressure and diastolic blood pressure are predictors of cardiovascular disease.

The burden of disease in Australia that can be attributed to high blood pressure was estimated to be more than 5% of the total among Australians in 1996 (AIHW: Mathers et al. 1999). As this figure was based on a threshold of 160/95 mmHg for high blood

pressure, it underestimates the burden corresponding to the cut-offs outlined in Box 3.2. Hypertension was the problem most commonly managed by general practitioners (GPs) in 2002–03, accounting for 6.1% of all problems managed (AIHW: Britt et al. 2003).

Prevalence and trends

Data from the 1999–2000 AusDiab indicate that 30% or 3.7 million Australians over the age of 25 years had high systolic or diastolic blood pressure or were on medication for that condition—32% of men and 27% of women. The proportion of men and women with high blood pressure increased with age (Figure 3.5).



Note: Based on WHO definition of high blood pressure (Box 3.2).

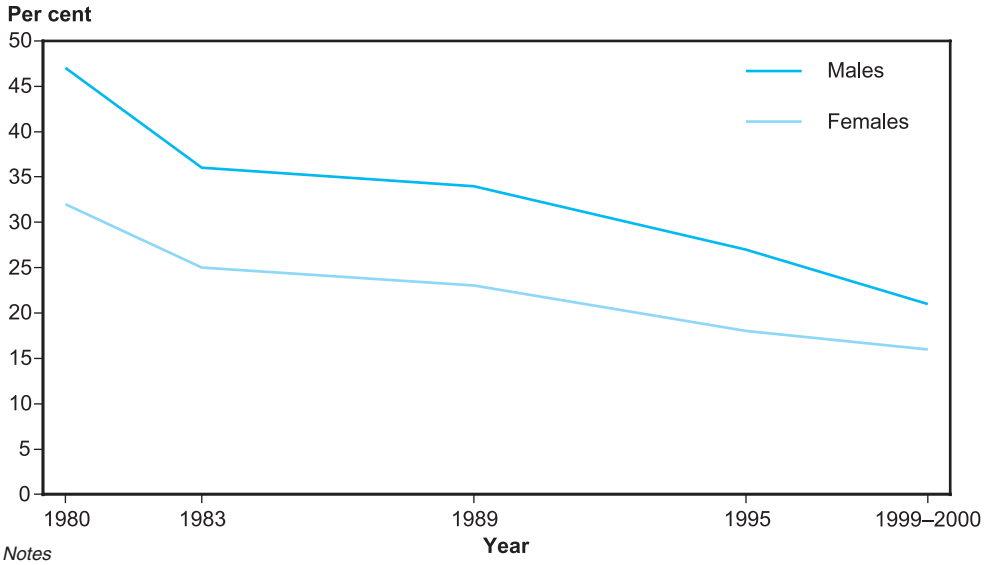
Source: AIHW analysis of the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study.

Figure 3.5: Proportion of people with high blood pressure, 1999–2000

Since 1980 the prevalence of high blood pressure has decreased markedly for both males and females (Figure 3.6) (trends are available only for the urban population). The proportion of men aged 25–64 years with high blood pressure has more than halved from 47% in 1980 to 21% in 1999–2000 and has halved for women from 32% in 1980 to 16% in 1999–2000. Average blood pressure has also decreased over this period (Table S.65).

Aboriginal and Torres Strait Islander people

There are no national data on measured blood pressure to assess the prevalence of hypertension among Aboriginal and Torres Strait Islander people. However, the 2001 NHS collected data on self-reported high blood pressure. These showed that Indigenous Australians reported high blood pressure from a younger age than non-Indigenous Australians. Among Australians of all ages, 14% of Aboriginal and Torres Strait Islander people reported high blood pressure, compared with 10% of other Australians (ABS 2002c).



Notes

1. Age-standardised to the 2001 Australian population.
2. Based on WHO definition of high blood pressure (Box 3.2).
3. Urban areas only.
4. People aged 25–64 years.

Source: AIHW analysis of the 1980, 1983, 1989 Risk Factor Prevalence surveys, the 1995 National Nutrition Survey and the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study.

Figure 3.6: Proportion of adults with high blood pressure, 1980 to 1999–2000

Blood cholesterol

High blood cholesterol is a major risk factor for coronary heart disease and ischaemic stroke. It is one of the main causes of atherosclerosis, the process by which the blood vessels that supply the heart and other parts of the body become clogged (see Box 3.3).

For most people, saturated fat in the diet is regarded as the main factor that raises blood cholesterol levels. Cholesterol in foods can also raise blood cholesterol levels, but usually less than saturated fat does (NHFA 1999). Genetic factors can also affect blood cholesterol significantly.

High blood cholesterol was estimated to have caused nearly 3% of the total burden of disease of Australians in 1996 (AIHW: Mathers et al. 1999). Lipid disorders, which include high blood cholesterol and high triglyceride levels, represented 2% of all problems managed by GPs in 2002–03 (AIHW: Britt et al. 2003). From 1998–99 to 2002–03 there was a significant increase in the management of lipid disorders by GPs, equivalent to 110,000 additional GP contacts on average per year nationally. This reflects a rise in workload (through ongoing management) rather than an increase in new cases presenting to general practice.

The 1999–2000 AusDiab estimated that around 50% of men and women in Australia had blood cholesterol levels of 5.5 mmol/L or more; that is, nearly six and a half million Australian adults aged 25 years and over. The prevalence increased with age to 65–74 years in women and 55–64 in men (Figure 3.7).

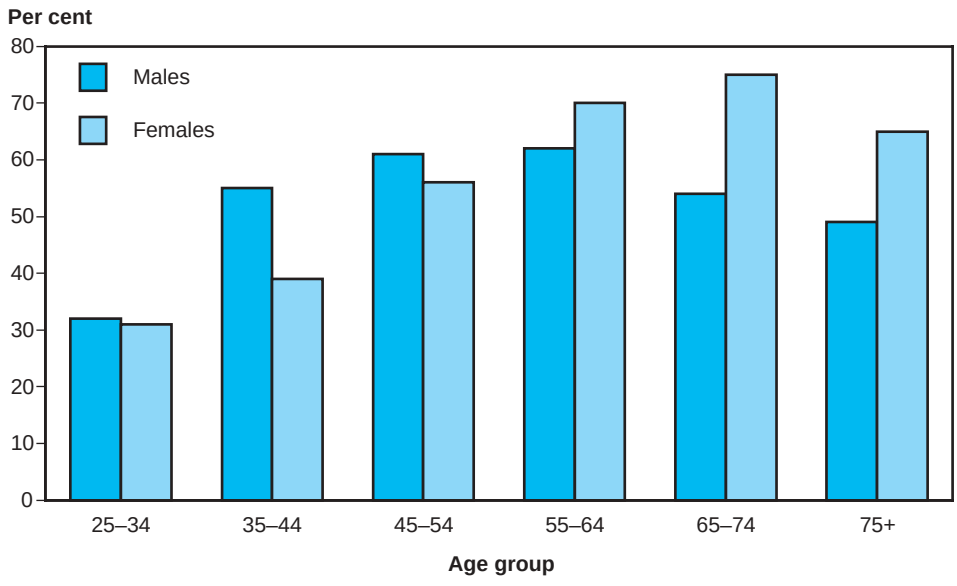
Box 3.3: High blood cholesterol

Cholesterol is a fatty substance produced by the liver and carried by the blood to the rest of the body. Its natural function is to provide material for cell walls and for steroid hormones. If levels in the blood are too high, this can lead to the artery-clogging process known as atherosclerosis that can bring on heart attacks, angina or stroke. The risk of heart disease increases steadily from a low base with increasing blood cholesterol levels. A total cholesterol level of 5.5 mmol/L or more is considered 'high' but this is an arbitrary cut-off.

Total cholesterol has several parts:

- *Low-density lipoprotein (LDL) cholesterol, often known as 'bad' cholesterol. Excess levels of LDL cholesterol are the main way that cholesterol contributes to atherosclerosis.*
- *High-density lipoprotein (HDL) cholesterol, often known as 'good' cholesterol. High levels have a protective effect against heart disease by helping reduce atherosclerosis.*
- *Triglyceride (TG) is another form of fat that is made by the body and its levels can fluctuate according to dietary fat intake. Under some conditions excess levels may contribute to atherosclerosis.*

In this report, high blood cholesterol is defined as a total cholesterol of 5.5 mmol/L or more.



Note: High blood cholesterol is 5.5 mmol/L or more.

Source: AIHW analysis of the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study.

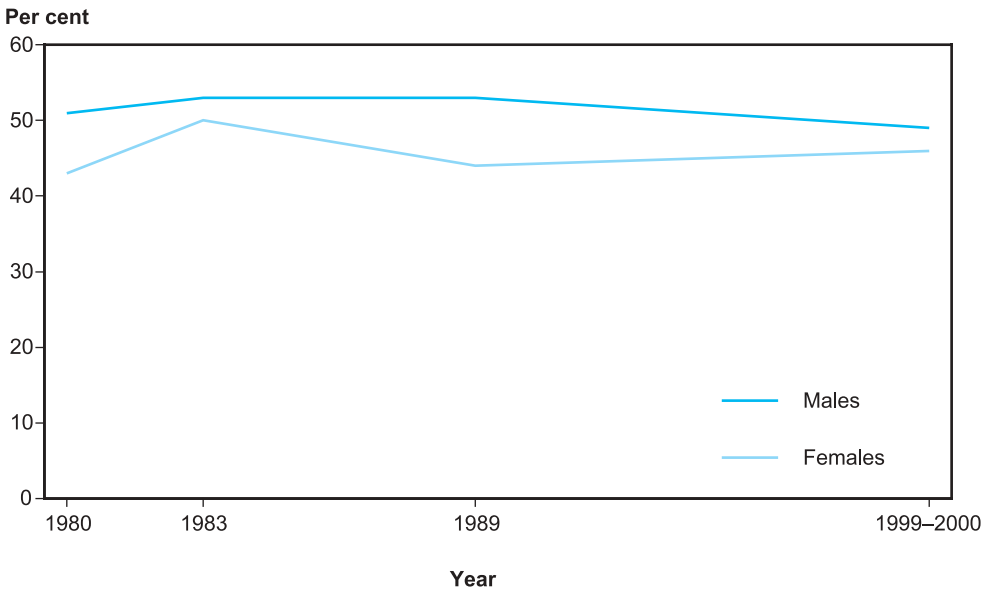
Figure 3.7: Proportion of adults with high blood cholesterol, 1999–2000

Trends in average blood cholesterol and high blood cholesterol prevalence are only available for people aged 25–64 years living in capital cities.

Average blood cholesterol levels in 1999–2000 were very similar to those 20 years earlier, for men and for women. Average cholesterol levels in Australia in 1999–2000 were 5.5 mmol/L for men and 5.4 mmol/L for women. Cholesterol levels in some societies are much lower than in Australia, as are their rates of cardiovascular disease, and this has been linked to their diet (Forge 1999).

Consistent with the trends in average levels, there has been no apparent reduction in the prevalence of people with high blood cholesterol since 1980 (Figure 3.8).

There are no national data on blood cholesterol levels among Aboriginal and Torres Strait Islander people.



Notes

1. Age-standardised to the 2001 Australian population.
2. Capital cities only.
3. People aged 25–64 years.
4. High blood cholesterol is 5.5 mmol/L or more.

Source: AIHW analysis of the 1980, 1983 and 1989 Risk Factor Prevalence surveys and the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study.

Figure 3.8: Proportion of adults with high blood cholesterol, 1980 to 1999–2000

Glucose tolerance

Impaired glucose tolerance (IGT) is a metabolic stage between normal glucose tolerance and diabetes. As well as being a risk factor for Type 2 diabetes, IGT is linked to a greater risk of heart disease.

In people with IGT, blood glucose levels are higher than normal but less than the level required for a diagnosis of diabetes. Blood glucose levels normally rise after eating a meal then gradually fall as the meal is digested, but in people with IGT these levels remain elevated for longer after a meal. This is a result of reduced sensitivity of the body's cells to insulin with or without a reduction of insulin production by the pancreas (insulin is the hormone that enables the body to convert glucose to energy). Although many people with IGT may revert to normal glucose tolerance, one in three people with IGT is likely to develop Type 2 diabetes within 10 years (Harris & Zimmet 1992).

IGT is detected through the same test used to detect diabetes—the oral glucose tolerance test. People with IGT have a plasma glucose concentration of less than 7.0 millimoles/litre (mmol/L) after fasting, and 7.8 or more but less than 11.1 mmol/L two hours after the oral glucose load.

A new category, 'impaired fasting glucose' (IFG) is also considered to be predictive of diabetes and is based on an abnormal blood glucose measurement after fasting. The IFG category covers fasting plasma glucose levels of 6.1 mmol/L or more but less than 7.0 mmol/L (Colagiuri et al. 2002).

IGT is common in people who are physically inactive or obese, particularly with high fat deposits in the abdominal region, and is more common in older people where such risk factors are more widespread. With increasing age, the cells in the pancreas that make insulin—beta cells—become less efficient. This, combined with decreased physical activity and increased body weight, contributes to higher prevalence among older people (Table 3.3). For similar reasons, Type 2 diabetes is also more common among older people. Genetic factors are also important; people who have a family history of diabetes are more likely to suffer from IGT and to develop diabetes.

Table 3.3: Age-specific prevalence of impaired glucose tolerance (IGT) and impaired fasting glucose (IFG), people aged 25 years and over, 1999–2000

	Males		Females	
Age (years)	IGT	IFG	IGT	IFG
			(per cent)	
25–34	2.1	3.4	4.9	0.5
35–44	4.7	8.4	8.9	2.1
45–54	9.0	9.3	11.0	5.1
55–64	14.8	12.8	15.7	4.5
65–74	20.4	11.5	21.9	4.3
75+	24.8	4.6	22.1	8.4

Source: Dunstan et al. 2002.

Results from the 1999–2000 AusDiab indicated that 10.6% of Australians aged 25 years and over had IGT and an additional 5.8% had IFG (Dunstan et al. 2002). IGT was slightly more common in women (11.9%) than men (9.2%). In contrast, IFG was more common in men (8.1%) than in women (3.4%).

The prevalence of IGT has more than trebled since 1981, with age-adjusted rates increasing from 3.2% to 9.8% in men and 3.0% to 12.3% in women (Dunstan et al. 2001). Dunstan et al. (2002) found that the increasing prevalence of obesity in Australia since 1981 was a significant contributing factor to the increasing prevalence of diabetes since that time. Given the links between obesity and IGT, this trend may also contribute to the escalating prevalence of IGT.

3.2 Genetic factors

Genetic factors play an important role in disease susceptibility and resistance. An individual's genetic makeup (the genome) provides the background against which the environment interacts with the human body.

Just as each individual is unique in genetic constitution, and hence in their susceptibility or resistance to disease, the genetic basis of a population's health can also be quite distinctive. Some of these genetic differences between populations are reflected in varying disease patterns and outcomes. The low incidence in Aboriginal and Torres Strait Islander Australians of rheumatoid arthritis and Type 1 diabetes—two diseases with well-known genetic susceptibilities and common in non-Indigenous populations—are good examples of this variation.

The contribution of genetic factors to health varies with age and environmental exposure. A study in Canada suggested that, before age 25, more than 5% of all live-born individuals would be affected with a disease that is mainly genetic in origin (Baird et al. 1988). However, it is often difficult to clearly identify genetic and environmental factors involved in diseases.

In addition to genetic variation that is inherited via reproductive cells (eggs and sperm), significant changes occur in the genetic material of non-reproductive cells over the life of an individual. Damage to these non-reproductive cells may also be subject to genetic control, but is limited only to the individual in which it occurs. Various cancers are examples of these genetic changes which are not passed on to children.

Genetic diseases and disorders

The genetic contribution to disease can vary considerably between individuals. In some cases the disease will occur regardless of the environment. In others it may be expressed partially, depending on how the genetic defect and the environment interact. Broadly, there are three major types of genetic diseases or disorders: monogenic and polygenic diseases, and chromosomal anomalies.

Monogenic diseases

Monogenic diseases result from an alteration or a change in the structure of a single (mono) gene. About 2% of the population will have a monogenic problem or condition,

with some of the problems showing up at birth and others later in life. Common examples are cystic fibrosis, muscular dystrophy and haemophilia.

Cystic fibrosis is one of the most serious monogenic diseases in Australia today. One in twenty-five people carry a copy of its gene, first identified in 1989, but only those people with two copies have any symptoms. In 2000, 45 Australian deaths were attributed to cystic fibrosis.

Limited information is available on the prevalence of monogenic diseases. It is now possible to detect several of these disorders early in life or even before birth.

Polygenic diseases

Quite often, more than one gene may contribute to the development of a disease (polygenic) together with environmental factors. The genetic contribution to these multifactorial diseases varies considerably. Common examples are cancers and several chronic diseases. Genetic factors may also affect susceptibility to various infections.

Cancers are typically due to damage to genes from exposure before or after birth to certain environmental agents (for example, tobacco smoke). A minority of cancers result from inheritance of a damaged gene.

Chronic diseases such as asthma, diabetes and Alzheimer's disease are the largest source of genetic burden of disease, mainly because of their high prevalence in the population. A variety of genes make a subtle contribution to a person's susceptibility to these chronic diseases. For example, genes located on chromosomes 5, 6, 11, 12 and 14 have been implicated in asthma (Khoury 1996). Similarly, about ten genes that increase the risk of Type 1 diabetes have now been described in the human genome.

Chromosomal anomalies

Chromosomal anomalies are a large source of chronic disease, disability and premature mortality. These anomalies arise through changes in the physical structure of the chromosomes or changes in the number of chromosomes. Common examples are Down syndrome, Klinefelter's syndrome and Turner's syndrome.

Chromosomal anomalies are among the best-defined causes of foetal loss or congenital disease. The incidence of chromosomal anomalies among live-born infants is estimated at about 0.5%. These anomalies also account for almost 50% of all spontaneous abortions. Tests are available to detect many chromosomal anomalies before birth (Khoury 1996).

Down syndrome, with three copies of chromosome 21 (trisomy 21) instead of the normal two, leads to much morbidity and premature mortality. Approximately 75% of cases with trisomy 21 die in the embryonic or foetal stage. Approximately 85% of affected infants survive to 1 year and 50% can be expected to live longer than 50 years. The presence of congenital heart disease is the most significant factor that determines survival. Individuals with Down syndrome have a greatly increased morbidity, primarily due to infections involving impaired immune response.

3.3 Health behaviours

Dietary behaviour

Diet plays a major role in health and disease. In recent decades much evidence has shown that dietary patterns can either reduce or increase the risk of various diseases and their risk factors. There are many areas of interest in dietary behaviour, but the greatest issue in Australia today is overconsumption.

Dietary guidelines from the National Health and Medical Research Council (NHMRC) (2003a, 2003b) recommend consuming a wide variety of nutritious foods including a high intake of plant foods (such as cereals, fruit, vegetables, legumes and nuts). They also recommend moderating total fat intake and limiting saturated fat intake to reduce the risk of coronary heart disease, Type 2 diabetes, several of the common cancers, and overweight and obesity. Other common diseases and risk factors where good nutrition may reduce risk include stroke, osteoporosis, tooth decay and high blood pressure (Table 3.4).

Table 3.4: Components of food which may help protect against diseases and conditions of public health importance

Dietary factor	Diseases (or conditions) against which protection may be provided or for which risk may be reduced
High intake of plant foods, low fat and saturated fat intake, high dietary fibre intake	Coronary heart disease, angina, colon, bowel, breast and prostate cancers, overweight and obesity
High intake of plant foods, low salt intake	High blood pressure, stroke
High intake of plant foods	Type 2 diabetes, constipation, gastrointestinal cancers (including cancers of the colon, rectum, stomach, pancreas and oesophagus), lung cancer and cancers of the breast, prostate, cervix and bladder
Low fat and saturated fat intake	Colorectal cancer
Low alcohol intake	Most cancers, liver cirrhosis, brain damage and foetal alcohol syndrome
Adequate to high calcium intake	Osteoporosis
Infrequent and low sugar intake	Tooth decay

Source: Adapted from Smith et al. 1998.

There have been so few data collected in recent years on the food and nutrient intake of Australians that much of the following discussion relates to data that are five to ten years old.

Folate intake

The impact of good nutrition on health begins early in life. It has been known for many years that insufficient folate or folic acid (a B vitamin) in the diet of women of child-bearing age increases the risk of having a foetus affected with spina bifida or other neural tube defects. The NHMRC (1994) recommends that women capable of becoming pregnant consume 400 µg per day of folate. Based on analysis of data from the 1995 National Nutrition Survey, only 1% of women aged 15–49 years consumed the recommended amount in their diet (excluding supplements) (Abraham & Webb