2 RISK FACTORS

Background

There are a number of determinants of CVD, Type 2 diabetes and CKD in terms of prevention, causation, disease progression and maintenance of the disease. Determinants that have a negative effect or increase the chance of a person developing a disease are often referred to as risk factors.

Some risk factors are not modifiable, such as age, sex and genetics, whereas others are associated with health-related behaviours or biomedical factors. The determinants of health, however, go beyond these to the underlying social, economic, psychological and cultural factors that can contribute to disease.

For almost all risk factors there is no known threshold at which risk begins. Rather, there is an increasing effect as the exposure increases. Although the increase in risk often starts at relatively low levels, the usual practice when monitoring is to focus on the riskier end of the spectrum. However, there is also value in monitoring moderate risk to assess trends in the wider population and to identify people who may benefit from preventive interventions that will help to reduce or maintain their risk profile.

CVD, Type 2 diabetes and CKD share many common risk factors. This chapter focuses on the modifiable risk factors for these diseases that can in some way be prevented. Table 2.1 lists the risk factors that will be discussed in this chapter and indicates the diseases with which they are associated. While each of the risk factors listed are directly associated with their corresponding diseases, there is a lot of overlap between the risk factors and, in some circumstances, one risk factor may lead to another risk factor, which will then lead to the disease. For example, overweight and obesity is a direct risk factor for CVD, Type 2 diabetes and CKD but it can also lead directly to high blood pressure, which in turn can lead to CVD, Type 2 diabetes and CKD. Where possible, information has been presented by different population groups including socioeconomic status, Indigenous status and geographical location. This is to identify particular groups that may have better or worse health than others.

Table 2.1: Relationships among cardiovascular disease, Type 2 diabetes and chronic kidney disease and risk factors

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
Overweight and obesity	✖	✖	✖
Physical inactivity	✖	✖	✖
Poor diet	✖	✖	✖
Tobacco smoking	✖	✖	✖
Excessive alcohol	✖		
High blood pressure	✖		✖
High blood cholesterol	✖	✖	
Impaired glucose regulation		✖	
Depression	✖		
Low birthweight	✖	✖	✖

Absolute risk

The concept of 'absolute risk' is explored at the end of this chapter. 'Absolute risk' is the term used to define the chance of an individual developing a particular disease within a specified period. It takes into account an individual's comprehensive risk factor profile instead of only focusing on single risk factors.

Multiple risk factors

Each of the risk factors covered in this chapter are individually important. However, having more than one risk factor can magnify the risk of disease (Kannel 2000; Poulter 1999). For example, a person with mildly raised blood pressure and no other risk factors will be at a lower risk of a cardiovascular event than someone with mildly raised blood pressure and one or two other risk factors (WHO-ISH 1999).

In 2001, it was estimated that more than half of all adults had two or three of nine risk factors associated with CVD (53%), with nearly one person in six (16%) having four or more risk factors. Men and women were equally likely to have five or more risk factors (5%), while women were slightly more likely than men not to report any risk factors (10% compared with 6%). The risk factors analysed in this study included low vegetable consumption, low fruit consumption, physical inactivity, smoking, obesity, high blood pressure, risky alcohol consumption, high blood cholesterol and diabetes (AIHW: O'Brien 2005).

Burden of disease

Individual risk factors can contribute to the overall burden of a disease. *The burden of disease and injury in Australia 2003* (Begg et al. 2007*)* showed that twelve different risk factors were associated with CVD and together explained 70% of the burden from this group of causes. High blood pressure and high blood cholesterol were the largest contributors, followed by physical inactivity, high body mass, tobacco, and low fruit and vegetable consumption. Two risk factors were associated with Type 2 diabetes (including the proportion of CVD caused by diabetes) and together explained 60% of the burden from this cause. High body mass was by far the largest contributor to this disease (Begg et al. 2007). There is no information available on the burden of CKD in Australia.

Overweight and obesity

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
Overweight and obesity	✘	✘	✘

Excess weight, particularly obesity, has been well established as a risk factor for CVD, diabetes and CKD (NHMRC 2003a; Wang et al. 2008). Overweight normally arises from a sustained energy imbalance due to the energy intake from the diet being greater than the energy expended through physical activity. Obesity is a severe form of overweight.

How do we measure overweight and obesity?

There are a number of methods used for measuring overweight and obesity including BMI, waist circumference and waist-to-hip ratio. BMI and waist circumference are the main methods used. BMI is most commonly used in population surveys and is calculated by dividing a person's weight in kilograms by the square of their height in metres (kg/m^2). Waist circumference is also useful because abdominal fat mass over certain thresholds indicates increased risk of chronic disease (Box 2.1; NHMRC 2003a). It may also be more strongly linked to disease than BMI (Seidell & Bouchard 1999).

Height and weight data may be collected in surveys as measured or self-reported data. When people self-report these measures, they 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 rates, and should not be directly compared with rates based on measured data (Flood et al. 2000; Niedhammer et al. 2000).

Box 2.1: Measuring body weight

Body mass index

The standard recommended by the World Health Organization (WHO 2000) and included in the National Health Data Dictionary *(HDSC 2006) to measure BMI for adults aged 18 years and over is:*

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

This classification may not be suitable for all ethnic groups. For children and adolescents aged 2–17 years, Cole and colleagues (2000) have developed a separate classification of overweight and obesity based on age and sex.

Waist circumference

The National Health Data Dictionary *defines waist circumference cut-offs for increased and substantially increased risk of ill health (NHDC 2003). 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.*

BMI can be categorised into groups of not overweight (including those who are in the healthy weight and underweight categories), overweight (but not obese) and obese to quantify prevalence. However, with such categorisation, valuable information about the overall picture can be lost. Therefore, the information is presented here as continuous, over the whole BMI range (Figure 2.1).

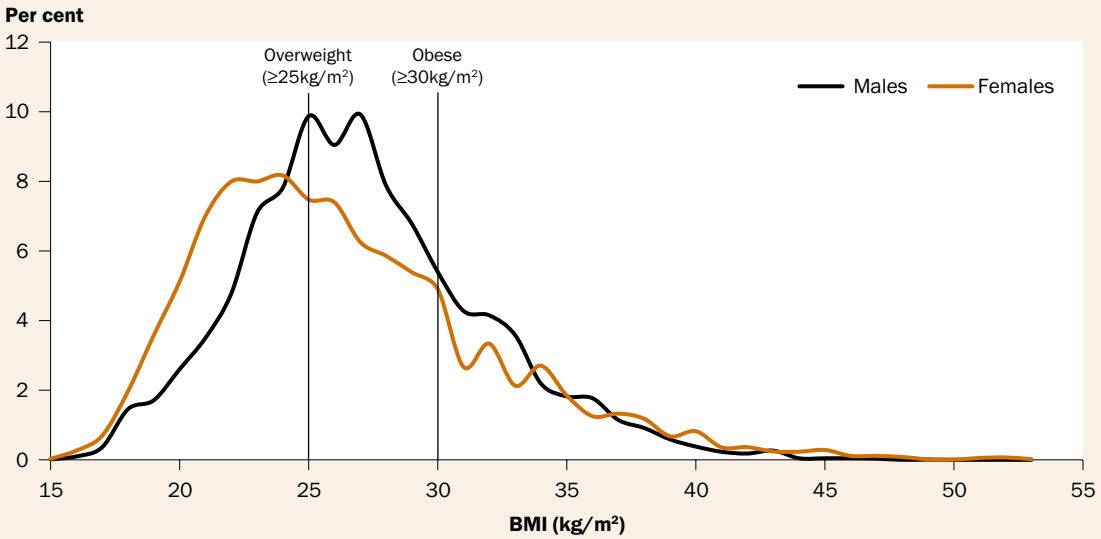
How many Australians are overweight or obese?

The most recent source of information that includes measured height, weight and waist circumference information is the 2007–08 NHS. According to this survey, 24% of people aged 15 years and over were classified as obese, 36% overweight (but not obese) and 40% not overweight.

Data from the 2007–08 NHS displayed in Figure 2.1 represent the distribution of BMI for both males and females in the Australian population. BMI measurements ranged from 15 kg/m^2, which is very underweight, to 53 kg/m^2, which is severely obese; however, most of the population had a BMI well within this range. For both males and females a greater proportion of the Australian population are obese rather than underweight.

On average, males had a higher BMI than females. This can be seen in Figure 2.1, as the distribution of BMI measurements for males is shifted slightly to the right of the distribution for females. Interestingly, when BMI was rounded to the nearest whole number, the most frequently measured BMI for males was 27 kg/m^2, which is categorised as overweight. In females, it was substantially lower at 24 kg/m^2, which is categorised in the healthy weight range.

As the data from the 2007–08 NHS have been analysed in a continuous fashion, it is possible to see that a large proportion of people who were not overweight had a BMI of 24 and therefore could be at risk of moving to the overweight category. Also, 16% of the overweight (but not obese) people had a BMI of 29, therefore being very close to the obese category. However, a similar proportion (19% of obese) had a BMI of 30 and therefore were only just classified as obese.



Notes

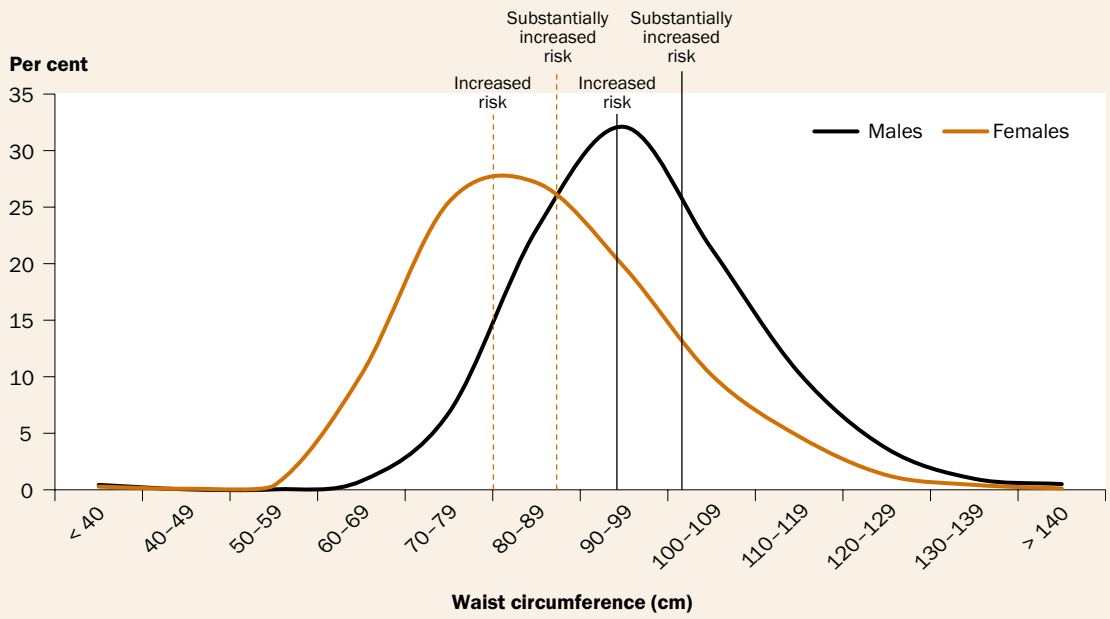
1. Based on measured data.
2. Missing values were excluded from the numerator and the denominator.

Source: AIHW analysis of the ABS 2007–08 National Health Survey confidentialised unit record file (CURF).

Figure 2.1: Body mass index (measured), people aged 15 years and over, 2007–08

The World Health Organization further categorises obesity into three categories by risk of comorbidity (NHMRC 2003a). All people who are obese are at risk; however, people with a BMI of 30–34.9 kg/m^2 are considered to be at moderate risk of comorbidity, 35–39.9 kg/m^2 at severe risk and ≥ 40 kg/m^2 at very severe risk. When analysed, the 2007–08 NHS shows that 68% of all people who were obese were considered to be of moderate risk, 24% at severe risk and 8% at very severe risk.

Figure 2.2 shows the distribution of waist circumference measurements for the Australian population as measured in the 2007–08 NHS. Over half of those surveyed were at increased or substantially increased risk of ill health (≥ 94 cm in males and ≥ 80 cm in females). The figure also indicates that a greater proportion of females had a higher level of risk (substantial risk) than males.



Notes

1. Based on measured data.
2. Missing values were excluded from the numerator and the denominator.
3. The increased and substantially increased risk indications are to be used as a guide only. Waist circumference measurements have been grouped into 10 cm groups and the proportion of people at each risk level can not therefore be accurately measured.

Source: AIHW analysis of the ABS 2007–08 National Health Survey CURF.

Figure 2.2: Waist circumference (measured), people aged 18 years and over, 2007–08

Population groups

Some population groups are at greater risk of overweight and obesity than others. Table 2.2 summarises the prevalence of overweight and obesity for different age groups, sex, socioeconomic status, Indigenous status and geographic location based on data from the 2007–08 NHS and the 2004–05 National Aboriginal and Torres Strait Islander Health Survey.

Middle-aged to older people (aged 55–74 years) were more likely to be obese than people in other age groups, and people aged 65–74 years had the highest rate of overweight (but not obesity). Males were more likely to be overweight or obese than females, with 66% of males aged 15 years and over overweight or obese compared with 53% of females.

Obesity increased with decreasing socioeconomic position and was highest among people living in outer regional, rural or remote areas. The prevalence of overweight (but not obesity), however, remained fairly constant among the different socioeconomic and geographic positions, with no obvious trend or pattern.

The crude prevalence of overweight and obesity in Indigenous Australians was 29% overweight and 31% obese. When Indigenous Australians were compared with non-Indigenous Australians, Indigenous males were slightly more likely (67%) to be overweight or obese than non-Indigenous males (64%). Also, Indigenous females were more likely (63%) to be overweight or obese than non-Indigenous females (46%).

Trends

The prevalence of overweight and obesity has increased steadily in recent years. Measured height and weight information from the 1995 and 2007–08 National Health Surveys indicates that the age-standardised rate of overweight and obesity in people aged 15 years and over has increased between 1995 and 2007–08, from 43% to 60%. The age-standardised rate of overweight (but not obesity) among Australian adults aged 15 years and over has increased from 32% in 1995 to 36% in 2007–08, and the proportion of Australian adults who were obese increased from 11% in 1995 to 24% in 2007–08.

An AIHW report titled *Obesity trends in older Australians* (AIHW: Bennett et al. 2004) analysed trend data using both self-reported and measured weight that had been obtained from various population surveys. This report showed that the average self-reported weight for every age group was higher in 2001 than for the same age group in 1995, which was in turn higher than its counterpart in 1989. Using measured weight data, the average measured weight increased during the 1980s and 1990s by around 6.5 kg for men and 7.1 kg for women. This increase in average measured weight occurred in each 5-year age group. The report also showed that men aged 30–34 years in 1980 gained over 8 kg as they aged to 50–54 years in 2000. Similarly, women aged 30–34 years in 1980 gained over 12 kg as they aged to 50–54 years. These findings have implications for future health services because not only are people heavier than previous generations at the same age, they are also likely to gain weight rather than lose weight as they become older, placing them at greater risk of being overweight or obese and therefore at increased risk of CVD, Type 2 diabetes and CKD.

Table 2.2: Prevalence of overweight (measured) based on body mass index[a] for people aged 15 years and over, 2007–08

	Per cent of population			
	Males		Females	
Population subgroup	Overweight (but not obese)	Obese	Overweight (but not obese)	Obese
Age group (years)				
15–24[b]	24.6	13.0	21.5	12.7
25–34	42.4	19.5	26.5	18.0
35–44	44.2	26.6	32.5	22.8
45–54	47.0	29.8	32.5	26.4
55–64	40.0	34.9	34.7	33.2
65–74	45.1	33.8	41.9	29.3
75 and over	52.8	21.5	32.5	24.2
15 and over ASR[c]	*41.2*	*24.7*	*30.5*	*22.7*
Socioeconomic status				
Group 1 (lowest socioeconomic position)	33.2	33.9	29.3	31.1
Group 2	38.6	26.0	30.5	25.5
Group 3	43.3	24.1	34.4	23.5
Group 4	45.0	22.4	30.9	20.7
Group 5 (highest socioeconomic position)	43.9	19.3	27.5	15.3
Aboriginal and Torres Strait Islander status[d]				
Indigenous	35.9	30.9	26.7	36.7
Non-Indigenous	44.8	19.6	29.0	17.3
Geographic location				
Major cities	41.5	22.7	29.3	20.9
Inner regional	42.7	26.2	34.5	25.7
Outer regional, rural and remote areas	37.5	32.3	30.7	27.0

(a) See Box 2.1 for classification of body mass index.

(b) For children aged 15–17 years, a separate classification of overweight and obesity developed by Cole et al. (2000) has been used.

(c) Age standardised to the 2001 Australian population.

(d) Data extracted from the ABS 2004–05 National Aboriginal and Torres Strait Islander Health Survey (data are from non-remote areas only). Please note, in this survey height and weight were self-reported.

Notes

1. Based on measured data.
2. All rates other than age-specific rates are standardised to the 2001 Australian population.
3. Rows may not add to 100.0 due to rounding.
4. Pregnant women were excluded from participating in this section of the NHS.
5. Cases with missing values were excluded from the numerator and the denominator.

Source: AIHW analysis of the ABS 2007–08 National Health Survey CURF and the ABS 2004–05 National Aboriginal and Torres Strait Islander Health Survey.

Physical inactivity

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
Physical inactivity	✘	✘	✘

Physical activity is important for maintaining good health. Regular participation in moderate to vigorous physical activity is protective against a range of diseases and conditions, including diabetes, heart disease and some forms of cancer (AIHW 2006). Participation in sufficient physical activity can modify or reduce the effects of some of the risk factors for CVD, Type 2 diabetes and CKD such as obesity, high blood pressure and high blood cholesterol. Participation in regular physical activity is one of the major recommendations of the evidence-based guidelines for the primary prevention of Type 2 diabetes (NHMRC 2001b). Physical inactivity has also been shown to increase the risk of CKD (Stengel et al. 2003; White et al. in press). Physical inactivity has been shown to be almost as important as tobacco smoking, and similar to high blood pressure and high blood cholesterol, in contributing to the prevalence of CVD in Australia (AIHW 2004).

What is physical activity?

Physical activity is any bodily movement produced by the muscles that results in energy expenditure. Exercise is a subset of physical activity; 'exercise' is defined as planned, structured and repetitive bodily movement done to improve or maintain one or more components of physical fitness. Recommended physical activity levels for both adults and children have been outlined in the National Physical Activity Guidelines for Australians (DHAC 1999).

These guidelines recommend 'at least 30 minutes of moderate-intensity physical activity on most, preferably all, days of the week' to achieve health benefits. This is generally interpreted as 30 minutes on at least 5 days of the week, a total of at least 150 minutes of moderate-intensity activity per week. Examples of moderate-intensity activity include brisk walking, medium-paced swimming or cycling, mowing the lawn and digging in the garden. The guidelines also suggest including some regular, vigorous activity for extra health and fitness for those who are able. Examples of vigorous activity include aerobics, speed walking, jogging and fast cycling.

How many Australians are physically inactive?

The most recent source of national data for physical activity levels is the 2007–08 NHS, in which exercise was categorised into four levels—high, medium, low and sedentary. Box 2.2 shows the methodology used in the 2007–08 NHS to define each exercise level. The results presented here from the 2007–08 NHS cannot be directly assessed against the Australian National Physical Activity Guidelines but all people in the sedentary category and some people in the low category would be classified as having insufficient activity levels according to the guidelines.

Overall, according to this survey, the majority (about 70%) of Australians were sedentary or undertook low levels of physical activity (Table 2.3).

Box 2.2: Defining exercise levels in the 2007–08 National Health Survey

Information was recorded from each respondent aged 15 years and over about the frequency, duration and intensity of exercise undertaken for sport, recreation or fitness during the last 2 weeks and an exercise level was derived for each respondent.

The level is based on a score derived from:

No. of times activity undertaken (in last 2 weeks) × Average time per session (in minutes) × Intensity

Exercise level was derived using intensity values of:

3.5 for walking

5.0 for moderate exercise

7.5 for vigorous exercise.

Scores for each activity were summed and ranges were grouped and labelled as follows:

Sedentary	*scores less than 100 (includes no exercise)*
Low exercise level	*scores of 100 to less than 1,600*
Moderate exercise level	*scores of 1,600 to 3,200 but less than 2 hours vigorous exercise*
High exercise level	*scores greater than 3,200 and 2 hours or more of vigorous exercise (ABS 2009).*

Population groups

Physical inactivity varied with age. The proportion of people with sedentary physical activity levels increased with age. As can be seen in Table 2.3, the proportion of people who had a sedentary physical activity level was highest in those aged 75 years and over (57%) and lowest in those aged 15–24 years (27%).

People who were categorised into the lowest socioeconomic position (that is, Group 1) were more likely than those from other groups to have sedentary physical activity levels (45%). When sedentary activity is combined with the low level of activity, 79% of people in the lowest socioeconomic group were categorised into these physical activity levels. These rates then decrease with increasing socioeconomic position.

Overall, a large proportion of Indigenous Australians reported sedentary physical activity levels (47%). Compared to non-Indigenous Australians using age-standardised rates, Indigenous Australians were more likely to be sedentary (53%) than other Australians (33%).

When physical activity levels were analysed by geographical location, no major differences were observed although some slight variations were evident. People living in areas other than *Major cities* and *Inner regional* had a higher proportion of people with sedentary exercise levels (39%) compared with those living in *Major cities* and in *Inner regional* areas (both 35%).

Trends

Published trend information indicates that the proportion of people in the low and sedentary group remained fairly constant, at about 70%, during the period 1995 to 2004–05 (ABS 2006). Results presented here suggest that this trend has continued to 2007–08, with about 70% of Australians aged 15 years and over still having sedentary or low physical activity levels.

Table 2.3: Prevalence of physical activity among people aged 15 years and over, 2007–08

	Per cent of population			
Population subgroup	**Sedentary**	**Low activity levels**	**Moderate activity levels**	**High activity levels**
Age group (years)				
15–24	27.2	37.5	24.2	11.1
25–34	30.4	41.1	20.0	8.5
35–44	34.7	39.8	19.6	6.0
45–54	36.5	37.1	21.0	5.4
55–64	36.6	35.6	24.1	3.7
65–74	39.5	32.3	25.9	2.2
75 and over	57.2	26.3	16.0	0.4
15 and over ASR[(a)]	35.2	37.1	21.5	6.2
Socioeconomic status				
Group 1 (lowest socioeconomic position)	45.5	33.4	16.9	4.2
Group 2	38.8	37.3	19.1	4.8
Group 3	38.2	34.7	20.8	6.4
Group 4	30.9	39.7	22.6	6.9
Group 5 (highest socioeconomic position)	24.9	39.6	27.2	8.3
Aboriginal and Torres Strait Islander status[(b)]				
Indigenous	53.0	26.7	15.6	4.7
Non-Indigenous	33.3	36.1	23.7	6.9
Geographic location				
Major cities	34.5	37.3	21.8	6.4
Inner regional	34.8	37.8	20.8	6.5
Outer regional, rural and remote areas	39.3	34.9	21.3	4.5

(a) Age standardised to the 2001 Australian population.

(b) Data extracted from the ABS 2004–05 National Aboriginal and Torres Strait Islander Health Survey. Due to the methodology used in this report, these results may differ to results published elsewhere.

Notes

1. See Box 2.2 for a definition of exercise level.
2. Based on self-reported data.
3. All rates other than age-specific rates are standardised to the 2001 Australian population.
4. Rows may not add to 100.0 due to rounding.
5. Cases with missing values were excluded from the numerator and the denominator.

Source: AIHW analysis of the ABS 2007–08 National Health Survey CURF and the ABS 2004–05 National Aboriginal and Torres Strait Islander Health Survey.

Poor diet

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
Poor diet	✖	✖	✖

Diet plays an important role in health and wellbeing. Poor diet is a risk factor for CVD, Type 2 diabetes and CKD largely through its adverse influence on body weight, particularly obesity (NHMRC 2001b; WHO 2003).

The promotion of good health and prevention of chronic diseases through dietary behaviour may be achieved, in the first instance, by following dietary guidelines such as those developed by the National Health and Medical Research Council (NHMRC). According to these guidelines, Australian adults and children should consume a wide variety of nutritious foods including a high intake of plant foods, and limit the intake of salt, saturated fat and alcohol (NHMRC 2003b).

Dietary fat intake

Recent evidence suggests that total fat intake does not appear to be an independent risk factor for coronary heart disease, but a diet high in fat may contribute to an increased risk of being overweight (AIHW 2004). There is also good evidence to support an association between a high consumption of saturated fatty acids and an increased risk of coronary heart disease by increasing total and low-density lipoprotein (LDL) cholesterol (the 'bad' cholesterol). For Type 2 diabetes, reducing saturated fat intake can decrease the risk of developing diabetes by increasing the body's ability to use insulin properly, promoting weight loss (in people who are overweight or obese) and reducing LDL cholesterol levels (NHMRC 2001b).

Dairy products are a main contributor to saturated fat intake, therefore the proportion of people consuming whole milk can be used as an indicator for higher total and saturated fat intake (Marks et al. 2001). Data from the 2007–08 NHS showed that 46% of the Australian population drank whole milk—51% of males and 40% of females. Other types of milk (which include low or reduced fat and skim milk) were consumed by 49% of the Australian population, 5% did not drink milk at all and approximately 1% did not know what type of milk they consumed.

Dietary fibre intake

A high-fibre diet is recommended to reduce the risk of developing Type 2 diabetes and CVD (ADA 2002; Mann et al. 2004; NHMRC 2001b; WHO 2003). Fruit and vegetable consumption can be used as an indicator of dietary fibre intake. The most recent source of information on fruit and vegetable consumption in the Australian population is the 2007–08 NHS. This survey found that 56% of males and 46% of females did not meet the recommended daily intake of fruit, and 92% of males and 90% of females did not meet the recommended daily intake of vegetables.

Population groups

Inadequate fruit intake appeared to decrease as age increased and was therefore less prevalent in older people than younger people. A less distinct difference in vegetable intake by age was observed (Table 2.4).

Levels of fruit intake were related to socioeconomic position, with 54% of people in the lowest socioeconomic group having inadequate fruit intake compared with 47% of people in the highest group. There was not a lot of difference, however, in vegetable intake across different socioeconomic groups, with 90–92% of people having inadequate vegetable intake.

A large proportion of Indigenous Australians consumed inadequate amounts of fruit (71%) and vegetables (81%). When compared with non-Indigenous Australians, the age-standardised prevalence of inadequate fruit intake in Indigenous Australians was 58% compared with 48% in non-Indigenous Australians. The age-standardised prevalence of inadequate vegetable intake in Indigenous Australians was 89% compared with 85% of non-Indigenous Australians.

People living in *Major cities* were less likely to have inadequate fruit intake (50%) than those living in *Inner regional* (53%) or other areas (57%). For vegetable consumption the opposite was observed, with a higher proportion of people living in *Major cities* having inadequate vegetable intake.

Trends

In order to compare the proportions of people consuming sufficient fruit and vegetables over time, sufficient intake has been based on two or more serves of fruit and four or more serves of vegetables. The proportion of people usually eating sufficient serves of fruit remained stable between 2001 and 2004–05 at around 50% (AIHW 2008f). The proportion of people usually consuming enough vegetables rose slightly for both males and females, from 27% of males and 33% of females in 2001 to 30% and 35% respectively in 2004–05 (AIHW 2008f). Information from the most recent NHS suggests that in 2007–08 the proportion of people consuming both sufficient fruit and vegetables has declined since 2004–05 (ABS 2009).

Table 2.4: Inadequate fruit and vegetable intake among people aged 15 years and over, 2007–08

	Per cent of population	
Population subgroup	**Inadequate fruit intake**	**Inadequate vegetable intake**
Age group (years)		
15–24	65.7	90.5
25–34	55.9	94.4
35–44	54.5	92.7
45–54	48.8	90.7
55–64	40.9	87.9
65–74	34.9	86.3
75 and over	34.6	87.3
15 and over ASR[(a)]	*51.0*	*90.8*
Socioeconomic status		
Group 1 (lowest socioeconomic position)	54.2	92.1
Group 2	54.3	90.2
Group 3	50.3	91.5
Group 4	49.9	89.7
Group 5 (highest socioeconomic position)	47.2	90.7
Aboriginal and Torres Strait Islander status[(b)]		
Indigenous	58.4	88.5
Non-Indigenous	47.9	84.9
Geographic location		
Major cities	49.6	92.6
Inner regional	52.5	86.1
Other areas	56.7	89.2

(a) Age-standardised to the 2001 Australian population.

(b) Data extracted from the ABS 2004–05 National Aboriginal and Torres Strait Islander Health Survey (data are from non-remote areas only).

Notes

1. Inadequate fruit intake is defined as less than two serves of fruit per day for adults and less than three serves a day for children and adolescents aged 15–18 years.
2. Inadequate vegetable intake is defined as less than five serves of vegetables per day for adults and less than four serves a day for children and adolescents aged 15–18 years.
3. Based on self-reported data.
4. All rates other than age-specific rates are standardised to the 2001 Australian population.
5. Rows may not add to 100.0 due to rounding.
6. Cases with missing values were excluded from the numerator and the denominator.

Source: AIHW analysis of the ABS 2007–08 National Health Survey CURF and the ABS 2004–05 National Aboriginal and Torres Strait Islander Health Survey.

Tobacco smoking

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
Tobacco smoking	✘	✘	✘

Tobacco smoking is known to be a major contributor to sickness and deaths in Australia and has been confirmed as an independent risk factor for CVD (AIHW 2008c), Type 2 diabetes (AIHW 2008d) and CKD (AIHW 2009b).

What is tobacco smoking?

Tobacco smoking includes the smoking of tobacco products such as packet cigarettes, roll-your-own cigarettes, pipes and cigars. People who smoke inhale a range of chemicals. The addictive substance in cigarettes is nicotine, but a range of other noxious substances, such as carbon monoxide and cadmium, are also inhaled (Foy et al. 2005). There can also be harmful effects associated with inhaling environmental tobacco smoke, or passive smoking. Passive smoking has been associated with a number of health problems including coronary heart disease. Passive smoking has not been covered further in this report.

How many Australians smoke?

According to the 2007 National Drug Strategy Household Survey, 16.6% of people aged 14 years and over smoked tobacco daily in 2007, 1.3% smoked tobacco weekly and 1.5% smoked less than weekly. In total, just over 19% of Australians aged 14 years and over were current smokers in 2007 (AIHW 2008h).

Figure 2.3 shows the number of times tobacco was consumed per month by people who identified as smokers. Overall, for smokers, tobacco consumption ranged from 1 time per month to about 3,700 times per month. Smokers most frequently consumed tobacco 300 to 499 times per month, with 27% of male smokers and 34% of female smokers consuming this amount. This is equal to 10–17 times per day.

Population groups

Table 2.5 shows how smoking behaviour varied among different population groups. In 2007, of those aged 14 years or over, the proportion who smoked tobacco was inversely related to socioeconomic status—26% for the lowest socioeconomic group against 14% for the highest group. Smoking was also related to remoteness, with 25% of people living in *Remote and very remote* areas being smokers compared with 18% of people in *Major cities*.

In order to assess the prevalence of smoking in Indigenous Australians, two data sources can be used. According to the 2007 National Drug Strategy Household Survey, which surveyed people aged 14 years and over, Indigenous Australians were more likely than non-Indigenous Australians to be current smokers (34% versus 19%) (Table 2.5). In addition to this, the 2004–05 National Aboriginal and Torres Strait Islander Health Survey, which surveyed only people aged 18 years and over, found that 48% of Indigenous Australians and 23% of non-Indigenous Australians were current smokers (AIHW 2008a).

Trends

The proportion of Australians aged 14 years and over who reported smoking tobacco in the previous year fell from 29.1% in 1993 to 19.4% in 2007 (AIHW 2008i). The proportion of daily smokers also declined to 16.6% in 2007, compared with 25.9% in 1993 and 17.4% in 2004.

Table 2.5: Prevalence of smoking among people aged 14 years and over, 2007

	Number per 100 population		
Population subgroup	**Never smoked[a]**	**Ex-smoker[b]**	**Smokers[c]**
Age group (years)			
14–19	87.9	2.3	9.8
20–29	60.5	12.8	26.7
30–39	51.0	24.1	24.8
40–49	47.1	28.8	24.2
50–59	48.3	32.8	18.9
60 and over	52.2	37.2	10.6
14 and over	*55.4*	*25.1*	*19.4*
Socioeconomic status			
Group 1 (lowest socioeconomic position)	49.6	24.5	25.9
Group 2	53.4	25.1	21.5
Group 3	55.8	23.7	20.5
Group 4	56.9	25.2	17.8
Group 5 (highest socioeconomic position)	59.6	26.6	13.9
Aboriginal and Torres Strait Islander status			
Indigenous	47.6	18.3	34.1
Non-Indigenous	55.6	25.5	19.0
Geographic location			
Major cities	57.6	24.4	18.0
Inner regional	51.7	26.4	21.9
Outer regional	50.2	26.9	23.0
Remote and very remote	47.8	27.1	25.0

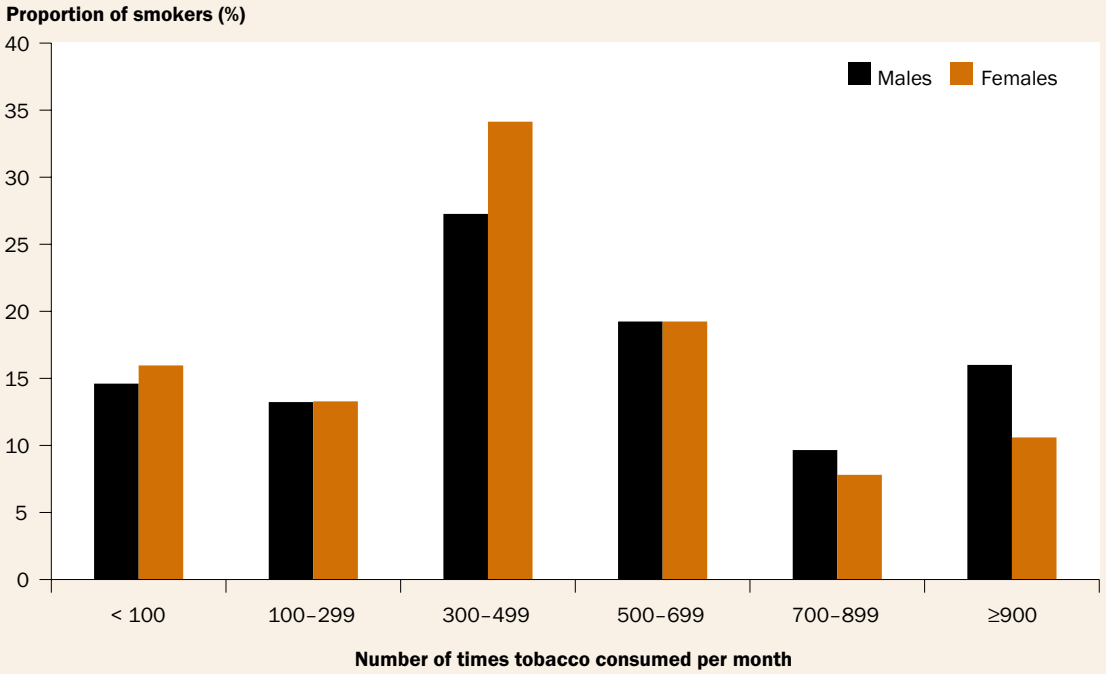
(a) Never smoked more than 100 cigarettes or the equivalent tobacco in their life.

(b) Smoked at least 100 cigarettes or the equivalent tobacco in their life, and no longer smoke.

(c) At the time of the survey smoked daily, weekly or less than weekly.

Note: Estimates of tobacco use by younger people should be interpreted with caution due to the low smoking prevalence and smaller sample size of this population group.

Source: 2007 National Drug Strategy Household Survey.



Source: 2007 National Drug Strategy Household Survey.

Figure 2.3: Number of times tobacco consumed per month, 2007

Excessive alcohol consumption

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
Excessive alcohol	✘		

Excessive alcohol consumption is a major risk factor for disease and death in Australia. Long-term excessive consumption of alcohol contributes to CVD.

The effect of alcohol consumption on CVD varies with levels of consumption—low to moderate consumption of alcohol is potentially protective whereas high consumption is associated with a higher risk of CVD. High intake of alcohol (and particularly binge drinking, also known as 'short-term risk of harm') is associated with higher blood pressure and increased risk of death from stroke. Alcohol can also affect blood triglyceride levels, complicating the effects of high blood cholesterol where present (AIHW 2004).

What is excessive alcohol consumption?

In this report, alcohol consumption is presented according to the 2001 NHMRC alcohol guidelines (NHMRC 2001a), which were the current guidelines at the time the data were collected. These alcohol risk guidelines have recently been revised and updated (NHMRC 2009).

Risk of alcohol-related harm can be measured as either short-term or long-term risk of harm. Short-term risk of harm is associated with given levels of drinking on any drinking occasion whereas long-term risk of harm is associated with regular daily patterns of drinking. This report will focus on the long-term risk of alcohol-related harm because it is the regular daily pattern of drinking that contributes to CVD.

The 2001 alcohol guidelines describe three risk categories for alcohol-related harm in the long term. For adult males, the consumption of up to 28 standard drinks per week is considered 'low risk', 29 to 42 per week 'risky' and 43 or more per week 'high risk'. For females, the consumption of up to 14 standard drinks per week is considered 'low risk', 15 to 28 per week 'risky' and 29 or more per week 'high risk'.

How many Australians drink alcohol?

In terms of long-term alcohol-related harm and based on self-reported information from the 2007 National Drug Strategy Household Survey, it was estimated that the majority (73%) of Australians aged 14 years and over consumed alcohol in moderation ('low risk'). In contrast, 10% of those aged 14 years and over drank at levels considered to be harmful ('risky' and 'high risk') to their overall health—this corresponds to 2.1 million Australians. Around 17% of people aged 14 years and over abstained from alcohol in the previous 12 months.

The 2007 National Drug Strategy Household Survey asked all people who reported having had an alcoholic drink in the previous 12 months how many standard alcoholic drinks they consumed on the previous day. This ranged from 0 to 47 standard alcoholic drinks that day (Figure 2.4). The majority of the Australian population aged 14 years or over (53% of males and 66% of females) had consumed less than one alcoholic drink on the previous day. The proportion of people then decreased exponentially as the number of drinks increased.

Population groups

Alcohol consumption patterns varied among different population groups in 2007 (Table 2.6). When comparing different age groups, younger people (16% of those aged 20–29 years) were more likely than other age groups to consume high levels of alcohol. There were no significant differences by socioeconomic group in the consumption of alcohol at harmful levels ('risky' and 'high risk'). Indigenous Australians were slightly more likely to consume alcohol at 'high risk' levels than non-Indigenous Australians (12.5% compared with 10.2%). However, Indigenous Australians were also more likely to abstain from alcohol compared with non-Indigenous Australians (23% compared with 17%). Among people aged 14 years and over, a higher percentage of people living in *Remote and very remote* areas reported consuming alcohol at 'risky' and 'high risk' (15%) levels, compared with those living in *Major cities* (10%).

Trends

One in 10 people aged 14 years or over consumed alcohol once a month or more at levels considered harmful in the long term. This level of harmful consumption has been relatively stable since 2001 (AIHW 2008h).

Table 2.6: Prevalence of alcohol consumption behaviour based on long-term risk[a] of harm among people aged 14 years and over, 2007

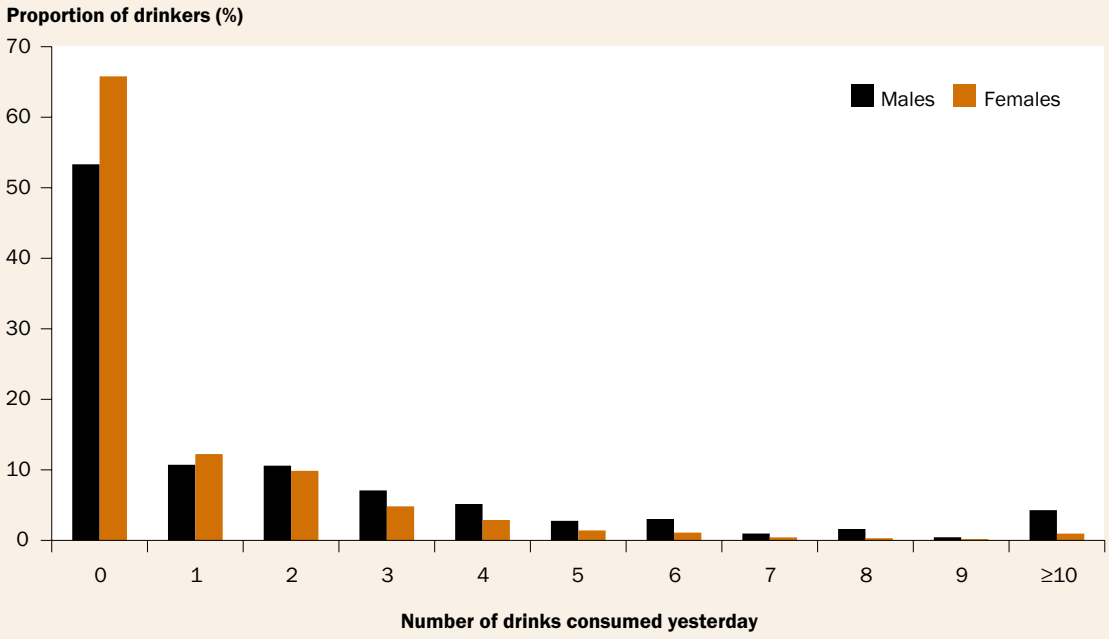
	Number per 100 population		
Population subgroup	**Abstainer/ex-drinker**	**Low risk[b]**	**Risky or high risk[c]**
Age group (years)			
14–19	29.0	62.2	8.8
20–29	12.9	71.1	16.0
30–39	12.2	77.5	10.3
40–49	12.4	76.8	10.8
50–59	14.0	75.6	10.4
60 and over	24.7	68.9	6.4
14 and over	*17.1*	*72.6*	*10.3*
Socioeconomic status			
Group 1 (lowest socioeconomic position)	22.5	66.7	10.8
Group 2	19.5	70.5	9.9
Group 3	17.9	71.9	10.2
Group 4	16.5	74.4	9.1
Group 5 (highest socioeconomic position)	11.5	77.1	11.5
Aboriginal and Torres Strait Islander status			
Indigenous	23.4	64.2	12.5
Non-Indigenous	16.8	73.0	10.2
Geographic location			
Major cities	17.3	72.9	9.8
Inner regional	17.2	72.0	10.9
Outer regional	16.5	71.5	12.0
Remote and very remote	12.6	72.1	15.3

(a) Long-term risk of harm is associated with regular daily patterns of drinking. The model used is that outlined in the 2001 Australian Alcohol Guidelines (NHMRC 2001b).

(b) For males the consumption of up to 28 standard drinks per week and for females up to 14 standard drinks per week is considered 'Low risk'.

(c) For males 29 or more standard drinks per week and for females 15 or more standard drinks per week is considered 'Risky or high risk'.

Source: 2007 National Drug Strategy Household Survey.



Note: Number of drinks reported has been rounded to the nearest whole standard drink.
Source: 2007 National Drug Strategy Household Survey.

Figure 2.4: Number of standard drinks consumed on the previous day, 2007

High blood pressure

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
High blood pressure	✖		✖

Elevated blood pressure is a major risk factor for CVD and CKD (Barri 2008; NHFA 2008). It is more likely to occur if a number of other risk factors are present including obesity, physical inactivity and poor diet (NHMRC 2004). Controlling or reducing the chance of the onset of high blood pressure reduces the risk of CVD and overall deaths (NHFA 2008).

How do we measure blood pressure?

Blood pressure measures the forces exerted on the artery walls as the heart pumps blood around the body. It is written as systolic/diastolic (for example 120/80 mm Hg, 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 before its next contraction.

There is a continuous relationship between blood pressure levels and CVD risk. This makes the definition of high blood pressure somewhat arbitrary. Clinically, individuals may have different blood pressure targets based on a number of different factors. However, for population-level monitoring, the World Health Organization (Whitworth 2003) defines high blood pressure as:

- systolic blood pressure of 140 mm Hg or more, or
- diastolic blood pressure of 90 mm Hg or more, or
- receiving medication for high blood pressure.

How many Australians have high blood pressure?

The Australian Diabetes, Obesity and Lifestyle (AusDiab) study conducted in 1999–2000 is the most recent source of national data available. The study measured the blood pressure of each participant. The data show that, overall, 30% of adults aged 25 years and over had high blood pressure, and males were more likely to have high blood pressure (33%) than females (27%).

Although the point at which an individual is considered to have high blood pressure is well defined, there is actually no clear threshold at which the risk of disease begins. For example, each increment in a person's blood pressure above their optimal level is associated with an increase in the risk of stroke. It is therefore useful to examine the total range of blood pressures in the Australian population, and to assess both the proportion of people that is on the border of high blood pressure and the severity of blood pressure levels within the group of people that is classified as having high blood pressure.

Figure 2.5 represents the distribution of systolic blood pressure for males and females in the Australian population. The most common systolic blood pressure measurement for both males and females was 120 mm Hg. Figure 2.6 represents the distribution of diastolic blood pressure for males and females in the Australian population. The most common diastolic blood pressure measurement for males was 75 mm Hg and for females 65 mm Hg. Figures 2.5 and 2.6 show that, on average, males had higher systolic and diastolic blood pressure than females. A greater proportion of males were also more likely to be categorised as having high systolic or diastolic blood pressure than females.

Figures 2.5 and 2.6 also indicate the proportions of males and females that were receiving medication to lower blood pressure. They show that a larger proportion of females were receiving medication to lower blood pressure (15%) than males (12%). Interestingly, both figures also show that a larger proportion of males than females with high blood pressure levels were not receiving medication. This could suggest that males are more likely to have undetected or untreated high blood pressure.

Theoretically, all people receiving medication to lower blood pressure should not have a systolic blood pressure that is greater than or equal to 140 mm Hg or a diastolic blood pressure that is greater than or equal to 90 mm Hg. This is because blood-pressure–lowering medications should successfully lower an individual's blood pressure to the target levels. There may be circumstances in which an individual's blood pressure does not respond to pharmacological treatment (NHFA 2008); however, with further review and adjustments to the treatment strategy, most high blood pressure should eventually be controlled. Figures 2.5 and 2.6 show that substantial proportions of males and females were receiving medication to lower blood pressure, but continued to have blood pressure levels that are considered high.

This could suggest that a substantial proportion of the population has detected and treated high blood pressure that remains uncontrolled. These people remain at an unacceptable risk of CVD and CKD unless their treatment is reviewed and their blood pressure reduced to the target levels.

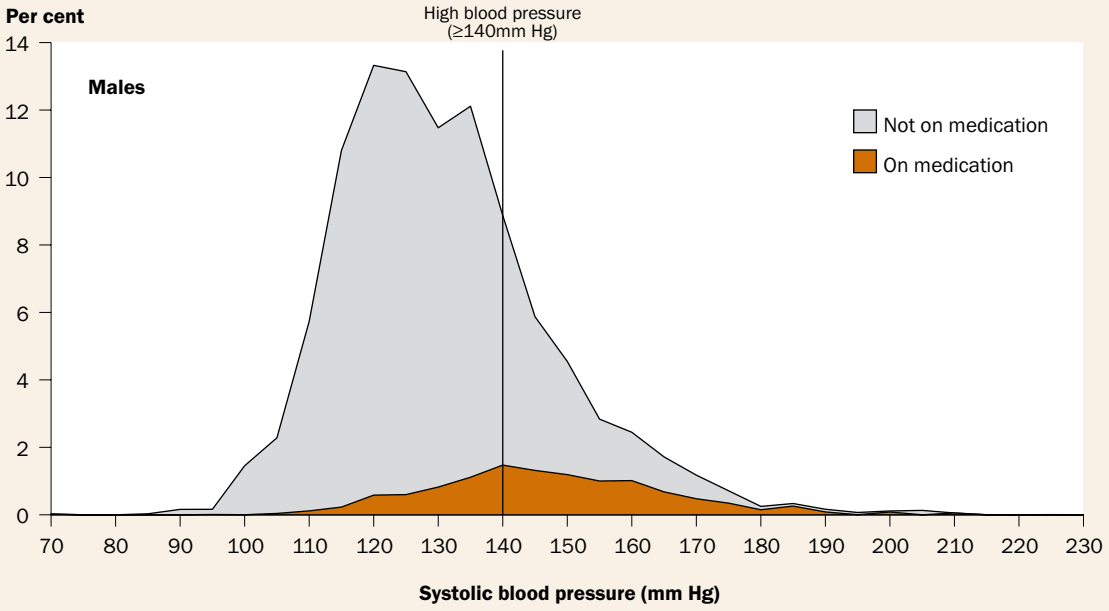
Population groups

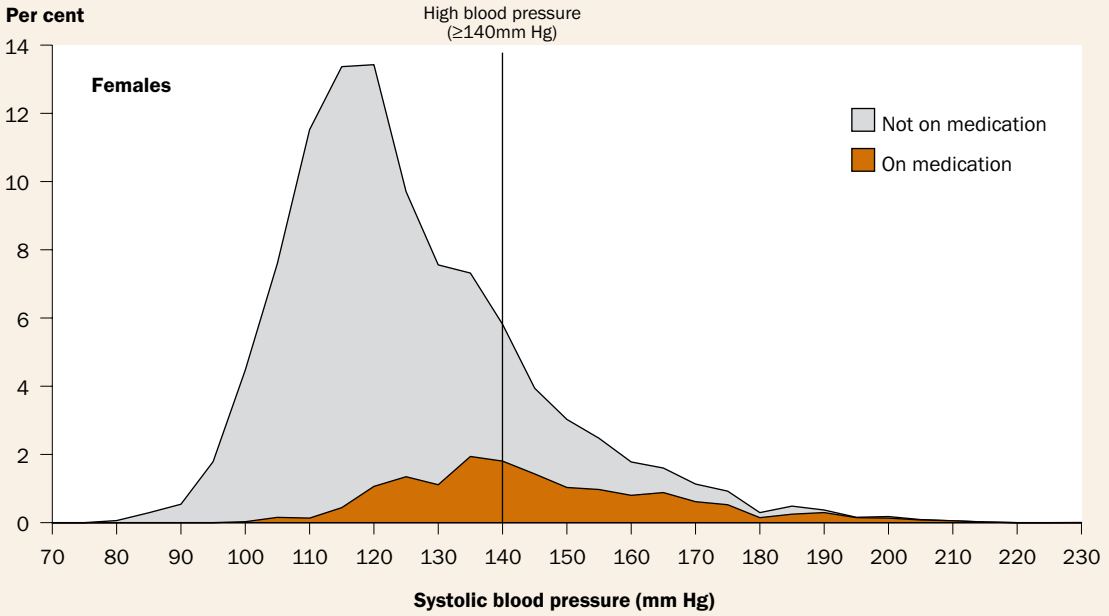
The prevalence of high blood pressure varies among different population groups in Australia (Table 2.7). In 1999–2000, the prevalence of high blood pressure increased with age and was higher in males than females. People who did not complete secondary school were more likely than people who completed secondary school or a higher level of education to have high blood pressure. The proportions of people with high blood pressure were similar in urban and rural areas.

There are no data sources available that contain measured blood pressure information for Indigenous Australians. However, based on self-reported blood pressure data in the 2004–05 National Aboriginal and Torres Strait Islander Health Survey, about 14% of Indigenous males and 16% of Indigenous females reported high blood pressure, compared with 10% of non-Indigenous males and females (AIHW 2008a).

Trends

The prevalence of high blood pressure among people aged 25 years and over in 1995 (31%) was similar to that in 1999–2000 (30%) (AIHW 2008c). Longer term trends are available only for the urban population aged 25 to 64 years. They indicate that between 1980 and 1999–2000 the prevalence of high blood pressure more than halved for males aged 25 to 64 years (from 47% in 1980 to 21% in 1999–2000) and halved for females of the same age (from 32% in 1980 to 16% in 1999–2000) (AIHW 2008c).



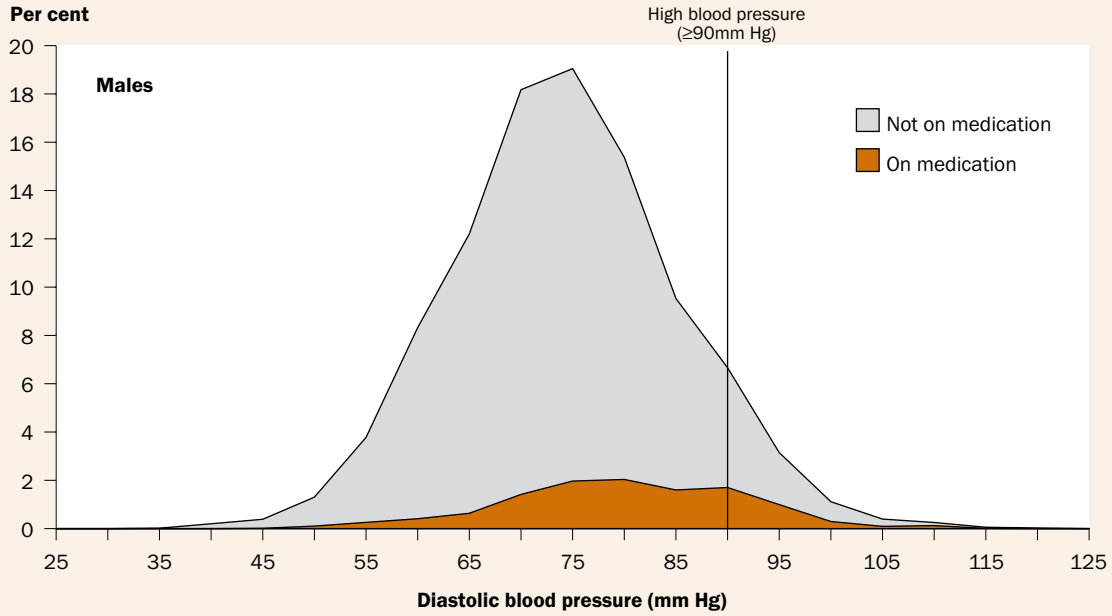


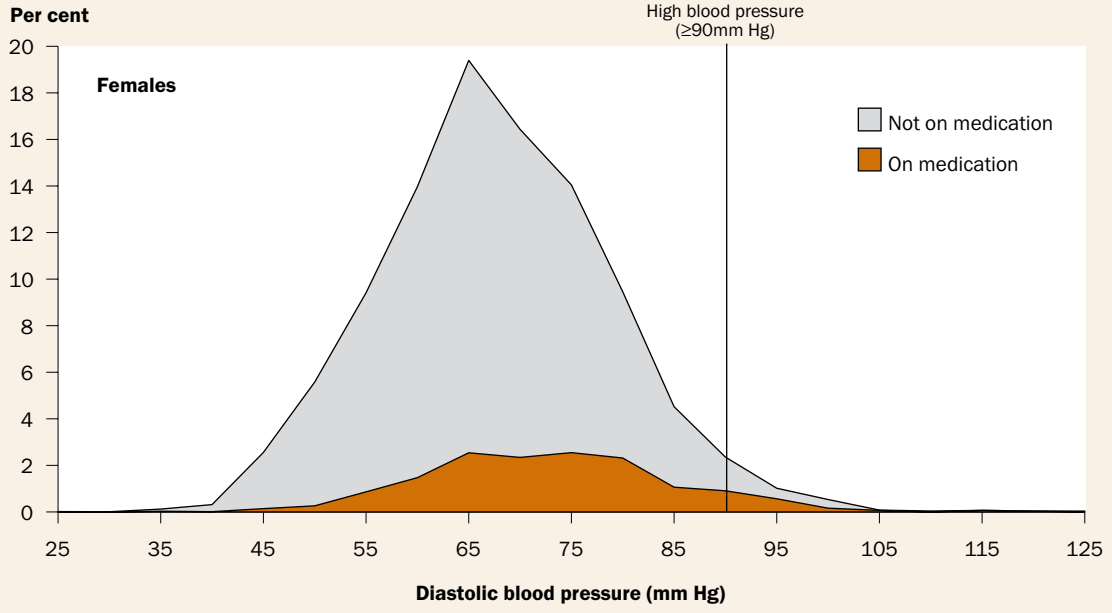
Notes

1. Based on measured data.
2. Blood pressure measurements have been rounded to the nearest multiple of 5.
3. Missing values were excluded from the numerator and the denominator.
4. Excludes people who self-reported taking medications for high blood pressure

Source: AIHW analysis of the 1999–2000 AusDiab study.

Figure 2.5: Systolic blood pressure (measured), people aged 25 years and over, 1999–2000





Notes

1. Based on measured data.
2. Blood pressure measurements have been rounded to the nearest multiple of 5.
3. Missing values were excluded from the numerator and the denominator.
4. Excludes people who self-reported taking medications for high blood pressure

Source: AIHW analysis of the 1999–2000 AusDiab study.

Figure 2.6: Diastolic blood pressure (measured), people aged 25 years and over, 1999–2000

Table 2.7: Prevalence of high blood pressure[a] (measured) among people aged 25 years and over, 1999–2000

	Per cent of population		
Population subgroup	**Males**	**Females**	**Persons**
Age group (years)			
25–34	7.1	3.3	5.2
35–44	14.1	7.6	10.8
45–54	30.6	23.6	27.1
55–64	49.4	44.6	47.0
65–74	69.7	67.4	68.5
75 and over	80.0	75.9	77.6
25 and over ASR[b]	*32.5*	*27.4*	*29.9*
Socioeconomic status (highest level of education)			
Did not complete secondary school	35.3	30.4	32.6
Completed secondary school	31.6	23.4	27.2
Tertiary/technical and further education	30.6	25.8	28.5
Aboriginal and Torres Strait Islander status			
Indigenous	n.a.	n.a.	n.a.
Non-Indigenous	n.a.	n.a.	n.a.
Geographic location			
Urban	32.2	26.8	29.5
Rural	33.0	28.1	30.5

n.a. Not available.

(a) High blood pressure defined as systolic blood pressure of 140 mm Hg or more or diastolic blood pressure of 90 mm Hg or more or receiving medication for high blood pressure.

(b) Age-standardised to the 2001 Australian population.

Notes

1. Based on measured data.
2. Column totals may not sum to 100.0 due to rounding.
3. Missing values were excluded from the numerator and the denominator.
4. Excludes people who self-reported taking medications for high blood pressure

Source: AIHW analysis of the 1999–2000 AusDiab study.

High blood cholesterol

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
High blood cholesterol	✖	✖	

High blood cholesterol is a major risk factor for coronary heart disease and ischaemic stroke, two highly prevalent forms of CVD (AIHW 2008c). It is a basic cause of plaque, the process by which the blood vessels that supply the heart and certain other parts of the body become clogged.

For most people, saturated fat in the diet is the main factor that raises blood cholesterol levels. Genetic factors can also affect blood cholesterol levels, severely in some individuals. Attention to factors such as physical activity and diet plays an important role in maintaining a healthy blood cholesterol level.

What is cholesterol?

Cholesterol (see Box 2.3) 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 an artery-clogging process known as atherosclerosis that can trigger heart attacks, angina or stroke.

As with most risk factors, there is not a single point at which the risk of disease from cholesterol begins. Rather, as total blood cholesterol increases, so does the risk of CVD and Type 2 diabetes. In general, a total cholesterol level of 5.5 mmol/L or more is considered high, but it is important to remember that this is an arbitrary definition.

Box 2.3: Blood cholesterol

Two important parts of blood cholesterol are:

- *low-density lipoprotein (LDL) cholesterol, often known as 'bad' cholesterol. Excess levels of LDL cholesterol are the main way that cholesterol contributes to plaque*
- *high-density lipoprotein (HDL) cholesterol, often known as 'good' cholesterol. High levels have a protective effect against heart disease by helping to reduce plaque.*

Triglyceride is another form of fat that is made by the body. Its levels can fluctuate widely according to dietary fat intake and under some conditions levels that are too high may contribute to plaque.

In this report, 'high blood cholesterol' is based on a total cholesterol level of 5.5 mmol/L or more.

How many Australians have high total blood cholesterol?

The AusDiab study conducted in 1999–2000 is the most recent source of national data available in which total cholesterol was measured in study participants. Figure 2.7 displays the distribution of total cholesterol levels for the Australian population. The average total cholesterol levels for both males and females were 5.5–5.6 mmol/L. The data show that roughly half of all Australian adults aged 25 years and over had a total cholesterol level that was considered high (Figure 2.7).

Population groups

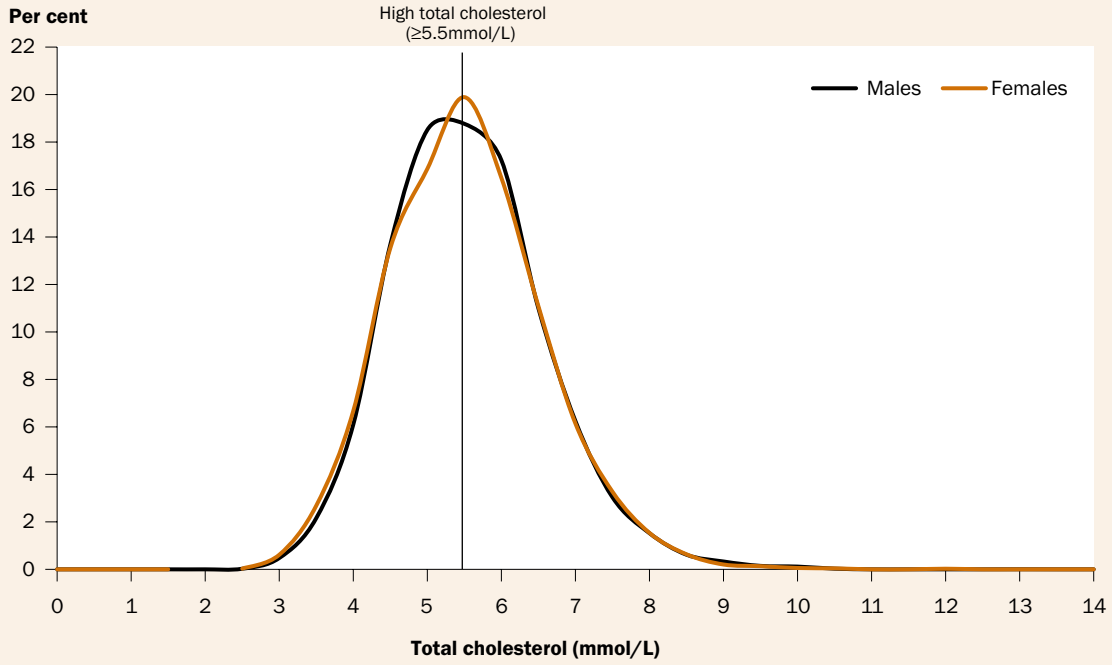
High total cholesterol was more common in older people; however, the rate was highest in people aged 55–64 years. Males had a higher rate of high total cholesterol than females up until age 55 years, but above that age females were more likely than males to have high total cholesterol (Table 2.8).

People living in rural communities had a slightly higher rate of high total cholesterol (50%) than people living in urban areas (47%). And, interestingly, people who did not complete secondary school were also slightly more likely to have high total cholesterol than those who did complete secondary school (Table 2.8).

The 1999–2000 AusDiab study cannot be used to report the prevalence of high total cholesterol in Aboriginal and Torres Strait Islander people. However, the 2004–05 National Aboriginal and Torres Strait Islander Health Survey estimates that 60% of Indigenous Australians had high cholesterol, compared with 45% of non-Indigenous Australians, based on self-reported information.

Trends

Trends in the prevalence of high blood cholesterol are only available to the year 2000 for people aged 25–64 years living in capital cities, and show no apparent change in the prevalence of high blood cholesterol since 1980 (AIHW 2008c).



Notes

1. Based on measured data.
2. Missing values were excluded from the numerator and the denominator.

Source: AIHW analysis of the 1999–2000 AusDiab study.

Figure 2.7: Total cholesterol (measured), people aged 25 years and over, 1999–2000

Table 2.8: Prevalence of high total cholesterol (measured) among people aged 25 years and over, 1999–2000

	Per cent of population		
Population subgroup	**Males**	**Females**	**Persons**
Age group (years)			
25–34	28.8	28.4	28.6
35–44	51.4	34.3	42.8
45–54	56.8	53.4	55.1
55–64	56.9	67.5	62.2
65–74	48.9	70.3	60.6
75 and over	47.7	61.4	55.8
25 and over ASR[(a)]	*47.6*	*47.8*	*47.9*
Socioeconomic status (highest level of education)			
Did not complete secondary school	50.1	48.6	49.5
Completed secondary school	46.9	48.2	47.9
Tertiary/technical and further education	46.8	45.9	46.1
Aboriginal and Torres Strait Islander status			
Indigenous	n.a.	n.a.	n.a.
Non-Indigenous	n.a.	n.a.	n.a.
Geographic location			
Urban	45.4	47.5	46.6
Rural	50.5	48.2	49.6

n.a. Not available.

Notes

1. Based on measured data.
2. Column totals may not sum to 100.0 due to rounding.
3. Missing values were excluded from the numerator and the denominator.

Source: AIHW analysis of the 1999–2000 AusDiab study.

Impaired glucose regulation

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
Impaired glucose regulation		✖	

Impaired glucose regulation is the metabolic state between normal glucose regulation and diabetes (WHO 1999). There are two categories of impaired glucose regulation: impaired fasting glucose (IFG) and impaired glucose tolerance (IGT). IFG and IGT are not considered to be clinical entities in their own right but rather risk factors for the future development of diabetes (NHMRC 2001b; Twigg et al. 2007).

Early treatment and improved management of impaired glucose regulation may reduce the incidence of Type 2 diabetes (Bennett 1999; Shaw & Chisholm 2003). In a review of six studies exploring IFG and IGT as predictors of future diabetes, it was found that, in the majority of the populations studied, 60% of

people who developed diabetes had either IGT or IFG 5 years before they were diagnosed with diabetes (Unwin et al. 2002).

There is some evidence that lifestyle changes incorporating increased physical activity and healthy eating could reduce or stop the progression of IFG and IGT to diabetes. For example, results from a large longitudinal study by Tuomilehto and colleagues (2001) showed that lifestyle interventions, such as counselling aimed at reducing weight and total fat intake, and increasing fibre intake and physical activity among obese adults with IGT reduced the rate of progression to diabetes by 40–60% over a 3–6-year period.

How is impaired glucose regulation defined?

IFG and IGT are measured using the oral glucose tolerance test—the same test used to diagnose diabetes. In this test a blood glucose measurement is taken after about 8 hours of fasting. Another measurement is taken 2 hours after consuming 75 g of glucose, often in the form of a high-sugar drink. IFG represents abnormalities of glucose regulation immediately after an overnight fast while IGT represents abnormalities of glucose regulation 2 hours after consuming glucose. Table 2.9 summarises how the categories of impaired glucose regulation are diagnosed and defined.

Table 2.9: Defining impaired glucose regulation

Impaired fasting glucose (IFG)	
Fasting blood glucose	6.1–6.9 mmol/L and, if measured,
2-hour blood glucose level[(a)]	< 7.8 mmol/L
Impaired glucose tolerance (IGT)	
Fasting blood glucose	< 7.0 mmol/L and
2-hour blood glucose level[(a)]	≥ 7.8 mmol/L and < 11.1 mmol/L
Type 2 diabetes	
Fasting blood glucose	≥ 7.0 mmol/L or
2-hour blood glucose level[(a)]	≥ 11.1 mmol/L

(a) Blood glucose 2 hours after ingestion of 75 g oral glucose load.

Source: National Evidence Based Guideline for Case Detection and Diagnosis of Type 2 Diabetes (Colagiuri et al. 2009).

How many Australians have impaired glucose regulation?

Based on measured data from the 1999–2000 AusDiab study, it was estimated that about one in six Australians aged 25 years and over had impaired glucose regulation, with IGT more prevalent than IFG (11% and 6% respectively) (Table 2.10).

The prevalence of impaired glucose regulation (which is made up of the prevalence of IFG and IGT) varies with sex and age. For example, in the 1999–2000 AusDiab study the overall prevalence of impaired glucose regulation was higher in males (17%) than females (15%) (Table 2.10). The age distribution indicates that the prevalence peaked in females aged 75 years and over, and in males aged 65–74 years (Figure 2.8). In almost all age groups, the prevalence of impaired glucose regulation was higher in males than females.

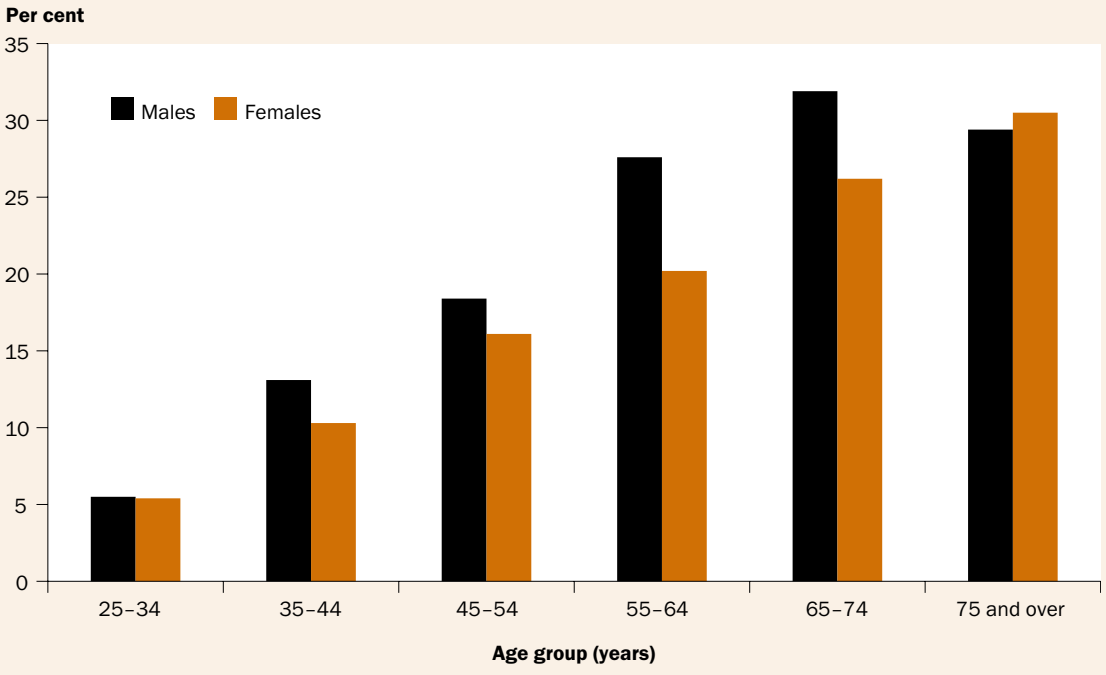
Trends

A comparison of results from the 1981 Busselton Study and 1999–2000 AusDiab study suggests a substantial increase in the age-standardised prevalence of IGT for both males (3–10%) and females (3–12%) between the two study periods (Dunstan et al. 2001).

Table 2.10: Prevalence of impaired glucose regulation among adults aged 25 years and over, 1999–2000

	Per cent of population		
	Males	**Females**	**Persons**
Impaired glucose tolerance	9.2	11.9	10.6
Impaired fasting glucose	8.1	3.4	5.8
Impaired glucose regulation	**17.4**	**15.4**	**16.4**

Source: AIHW analysis of the 1999–2000 AusDiab study.



Source: AIHW analysis of the 1999–2000 AusDiab study.

Figure 2.8: Age-specific prevalence of impaired glucose regulation in adults, 1999–2000

Depression

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
Depression	✘		

There is evidence suggesting that depression can directly lead to problems such as heart disease, independent of any intermediary behavioural effects such as increased rates of smoking or poor diet (Bunker et al. 2003). The strength of the association is similar to that of other standard risk factors such as high cholesterol (Bunker et al. 2003).

The relationship between depression and CVD is complex. CVD is a strong risk factor for depression, while depression is a risk factor for CVD (Clarke & Currie 2009). Depression can also affect other risk factors for CVD, and often co-exists with other risk factors. For example, people with depression are more likely to smoke and be physically inactive (Bunker et al. 2003), and depression is a major barrier to the adoption of healthy lifestyle behaviours (Hayes 2006).

What is depression?

Depression is a mental illness that affects many Australians. It is a mood disorder with prolonged feelings of being sad, hopeless, low and inadequate. It may include a loss of interest or pleasure in activities, often with suicidal thoughts or self-blame (AIHW 2008c). In 2006–07, depression was the fifth most common problem managed by GPs in Australia (AIHW 2008c). Other types of mental illness include anxiety, bipolar disorder and schizophrenia (AIHW 2008c). In total, mental illness was estimated to be responsible for 13% of the total burden of disease in Australia in 2003, placing it third as a broad disease group after cancers and CVD (Begg et al. 2007).

Box 2.4: Measuring mental health in the 2007 National Survey of Mental Health and Wellbeing

To estimate the prevalence of specific mental disorders, the 2007 National Survey of Mental Health and Wellbeing used the World Mental Health Survey Initiative's version of the World Health Organization's Composite International Diagnostic Interview, version 3.0 (WMH–CIDI 3.0). The WMH–CIDI 3.0 was chosen because it:

- *provides a fully structured diagnostic interview*
- *can be administered by lay interviewers*
- *is widely used in epidemiological surveys*
- *is supported by the World Health Organization*
- *provides comparability with similar surveys conducted worldwide.*

Source: *National Survey of Mental Health and Wellbeing: summary of results 2007 (ABS 2008).*

How many Australians have depression?

The 2007 National Survey of Mental Health and Wellbeing found that, of all people aged 16–85 years in Australia (16 million), almost 1.9 million people (11.6%) had experienced a depressive episode at some stage in their lives and 650,000 people (4.1%) had experienced a depressive episode in the previous 12 months (Table 2.11). These results equate to just over 1 in 25 Australians having experienced a depressive episode in the preceding 12 months.

Females had a higher prevalence of depressive episodes than males. Overall, 15% of women (equating to 1,150,700 females) and 9% of men (equating to 694,000 males) aged 18–85 years had experienced a depressive episode at some point in their lives. Females were also more likely than males to have experienced a depressive episode in the previous 12 months (5% of females compared with 3% males) (ABS 2008).

The prevalence of lifetime depressive episodes appeared to be higher among younger males and females. Figure 2.9 shows that females aged 18–64 years (16%) had a higher prevalence of lifetime depressive episodes than females aged 65–85 years (8%). Similarly, males aged 18–64 years (10%) had a higher prevalence of lifetime depressive episodes than males aged 65–85 years (6%).

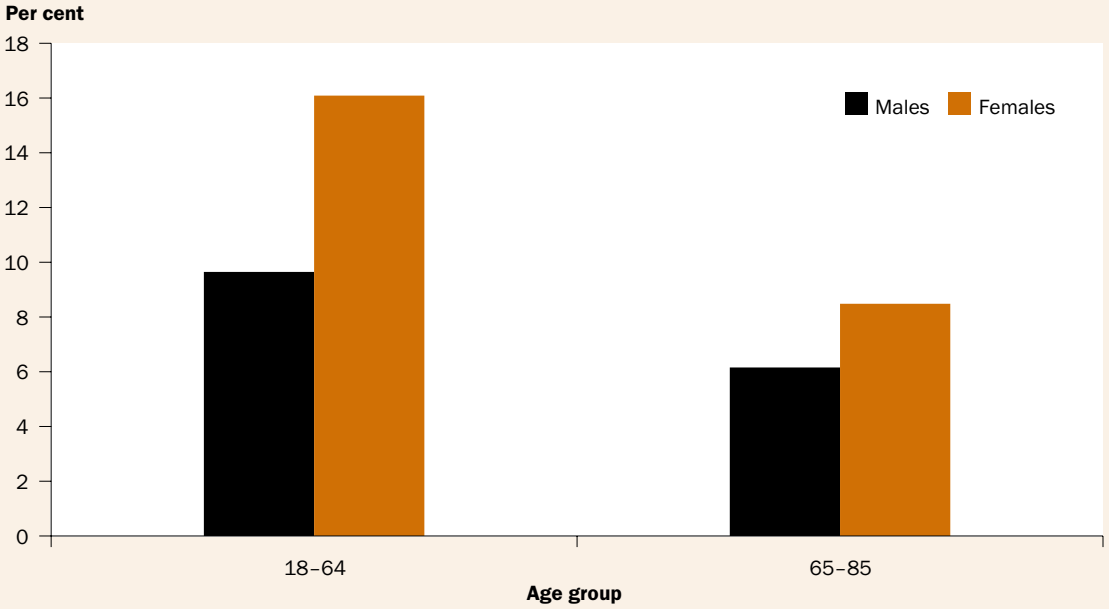
Table 2.11: 12-month and lifetime prevalence of depressive episodes in the Australian population, 2007

	12-month depressive episode[a]		Lifetime depressive episode[b]	
	Number ('000)	**Per cent**	**Number ('000)**	**Per cent**
Males	245.0	3.1	697.0	8.8
Females	407.4	5.1	1,168.1	14.5
Persons	**652.4**	**4.1**	**1,865.1**	**11.6**

(a) 12-month depressive episode is based on lifetime diagnosis and the presence of symptoms of a mild, moderate or severe depressive episode in the 12 months before the survey interview.

(b) Persons who met the criteria for a mild, moderate or severe depressive episode at some stage in their lifetime.

Source: National Survey of Mental Health and Wellbeing: summary of results 2007 (ABS 2008).



(a) Persons who met the criteria for mild, moderate or severe depressive episode at some stage in their lifetime.

Source: National Survey of Mental Health and Wellbeing: summary of results 2007 (ABS 2008).

Figure 2.9: Proportion of males and females with lifetime[(a)] depressive episode, 2007

Low birthweight

Risk factor	Cardiovascular disease	Type 2 diabetes	Chronic kidney disease
Low birthweight	✖	✖	✖

It is hypothesised that low birthweight babies have cardiovascular, metabolic and endocrine adaptations that permanently change the structure and function of the body (Barker 1999). It is theorised that these changes may to lead to CVD and Type 2 diabetes in adult life (Barker 1999). Low birthweight may also result in a number of factors associated with CKD (White et al. 2009). In particular, low birthweight is often associated with intrauterine malnutrition, which can impair nephron development in the kidneys. Nephrons are the functional and structural unit of the kidney and are responsible for the purification and filtration of the blood (Hughson et al. 2003).

A baby may be small due to being born early, or may be small for its gestational age. Some factors contributing to low birthweight include the mother's socioeconomic position, age of the mother, number of previously born siblings, mother's nutritional status, mother's smoking and alcohol intake, and illness during pregnancy (Ashdown-Lambert 2005; Mohsin et al. 2003).

What is low birthweight?

Babies are defined as having low birthweight if their weight at birth is less than 2,500 g. Within this category, babies weighing less than 1,500 g are defined as very low birthweight and babies less than 1,000 g as extremely low birthweight (WHO 1992).

How many babies are born with a low birthweight in Australia?

Data from the National Perinatal Data Collection show that the average birthweight of live born babies in Australia in 2006 was 3,370 g. The average birthweight ranged from 3,262 g in the Northern Territory to 3,407 g in Tasmania (Laws & Hilder 2008).

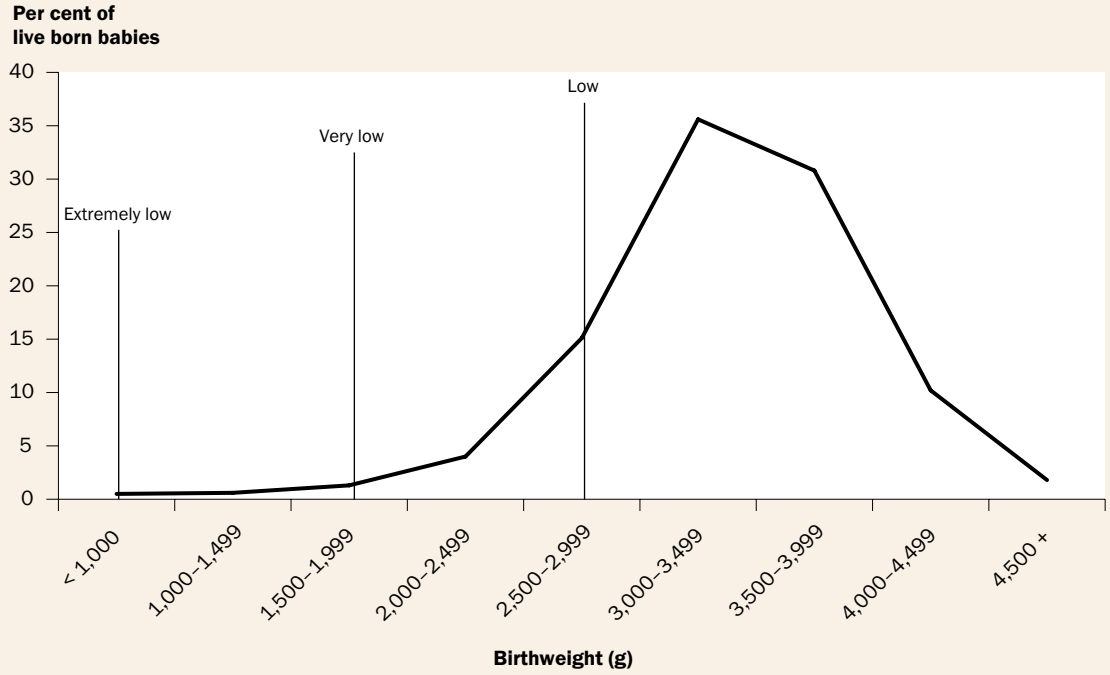
In 2006, there were almost 18,000 live born babies (6.4% of all births) with low birthweight in Australia (Laws & Hilder 2008). A further 3,000 babies were of very low birthweight, constituting a further 1.1% of live births, and 1,300 of these were extremely low birthweight (Figure 2.10 and Table 2.12). The data suggest that male live born babies were less likely to be of low birthweight (5.9%) than female babies (6.9%) in 2006. Since 2000, the proportion of live born babies with low birthweight has remained stable at around 6–7%.

Population groups

The proportion of low birthweight babies varied between different population groups (Table 2.12). In 2006, there was a higher proportion of low birthweight in live born babies of mothers living in the lower socioeconomic position (the most disadvantaged group) (7.3%) compared with babies born to mothers living in the highest socioeconomic position (5.5%).

The average birthweight of live born babies of Aboriginal and Torres Strait Islander mothers was about 3,169 g. This was just over 200 g lighter than the average of 3,378 g for live born babies of non-Indigenous mothers (Laws & Hilder 2008). The proportion of low birthweight live born babies of Aboriginal and Torres Strait Islander mothers was 12.4%, twice that of babies of non-Indigenous mothers (6.2%) (Laws & Hilder 2008).

The proportion of low birthweight babies also varied according to mother's geographical location of usual residence. In 2006, live born babies of mothers living in very remote locations were more likely to be of low birthweight (10.2%) than those born in other areas (Table 2.12).



Source: Australia's mothers and babies 2006 (Laws & Hilder 2008).

Figure 2.10: Birthweight of live born babies in grams, 2006

Table 2.12: Birthweight proportions for live born babies of mothers of different population groups, 2006

	Per cent of live born babies		
Population subgroup	**Extremely low[a]**	**Very low[b]**	**Low[c]**
Overall	0.5	1.1	6.4
Socioeconomic status			
Group 1 (lowest socioeconomic position)	0.6	1.3	7.3
Group 2	0.5	1.1	6.8
Group 3	0.4	1.0	6.3
Group 4	0.4	1.1	6.0
Group 5 (highest socioeconomic position)	0.4	0.9	5.5
Aboriginal and Torres Strait Islander status			
Indigenous	1.1	2.3	12.4
Non-Indigenous	0.4	1.0	6.2
Not stated	0.2	1.1	6.6
Geographic location			
Major cities	0.5	1.0	6.2
Inner regional	0.4	1.0	6.4
Outer regional	0.6	1.3	7.1
Remote	0.6	1.2	8.3
Very remote	0.9	2.1	10.2

(a) Extremely low birthweight = birthweight of less than 1,000 g.

(b) Very low birthweight = birthweight of less than 1,500 g.

(c) Low birthweight = birthweight of less than 2,500 g.

Note: In this table, very low is a subset of low birthweight and extremely low is a subset of very low birthweight.

Sources: Australia's mothers and babies 2006 (Laws & Hilder 2008); National Perinatal Statistics Unit additional analysis of the National Perinatal data collection.

Absolute risk

Up until now, preventive treatment decisions for CVD, Type 2 diabetes and CKD have been focused on individual risk factors. However, the relationship between risk factors and disease is integrated and continuous (Chen et al. 2008). Individuals are more likely to develop clusters of risk factors and the assessment of disease risk based on the combined effect of multiple risk factors is more accurate than that based on individual risk factors (NVDPA 2009).

'Absolute risk' is a term used to define the probability of an event occurring, for example a CVD event such as stroke, within a specified period, and takes into account an individual's entire risk factor profile instead of focusing on single risk factors. Tools for health professionals have recently been developed to assess the risk of CVD and Type 2 diabetes in people without known disease. There are no tools available yet to assess the absolute risk of CKD.

For absolute CVD risk, tools have been developed in both electronic and paper-based formats. These are usually based on the Framingham Risk Equation (Anderson et al. 1991). This equation has been adapted for the Australian population and considers an individual's age, sex, systolic blood pressure, total and HDL cholesterol, smoking status and left ventricular hypertrophy (enlargement of the lower left chamber of the heart). The result reflects the individual's risk of developing CVD and is designed to help Australian primary care health professionals to make informed decisions about the individual's clinical care to manage their risk (NVDPA 2009). Guidelines have been developed by the National Vascular Disease Prevention Alliance to help Australian health professionals to use this tool effectively. These guidelines have also been approved by the NHMRC.

In a recent study, Chen and colleagues (2007) used the Framingham Risk Equation to compute 5- and 10-year risks of developing first-time CVD for the AusDiab survey population. The results were weighted to the total Australian population and are shown in Table 2.13.

Table 2.13: Proportion of Australian population aged 25 years and over at CVD risk

	Per cent of Australian population	
	Men	**Women**
5-year CVD risk		
High[(a)]	5.4	0.6
Intermediate[(b)]	13.0	3.0
10-year CVD risk		
High[(a)]	5.4	0.6
Intermediate[(b)]	14.7	3.9

(a) High risk was defined as 5-year CVD risk > 15% or 10-year CVD risk > 30%.

(b) Intermediate risk was defined as 5-year CVD risk of 10–15% or 10-year CVD risk of 20–30%.

Source: Adapted from Chen et al. 2007.

To assess the risk of Type 2 diabetes in people without known disease, the Australian Type 2 Diabetes Risk Assessment Tool, commonly known as 'AUSDRISK'. has been developed (DOHA 2009d). This tool is based on the Finnish Type 2 Diabetes Risk Score, known as 'FINRISC' (Lindstrom & Tuomilehto 2003), but has been adapted for the Australian population. It is used to assess an individual's risk of developing Type 2 diabetes in the next 5 years. In this assessment tool, an individual's age, sex, ethnicity, family history of diabetes, blood glucose level, blood pressure, smoking status, fruit and vegetable intake, level of exercise and waist measurement are used to assign an overall level of risk of the individual developing Type 2 diabetes. It has been designed to be completed by the patient who may then, based on the result, be asked to consult their GP (DoHA 2009d).