Risk factors for diabetes and its complications

3

Introduction

Overweight and obesity

Physical inactivity

Impaired glucose tolerance

Nutrition

Tobacco smoking

High blood pressure

High cholesterol and triglycerides

diabetes
australian facts 2002

Australian Institute of Health and Welfare

Introduction

Many factors are thought to contribute to the development of diabetes. Type 1 diabetes is believed to be caused by exposure to environmental factors, possibly toxins or viruses. A genetic component is suspected, although a large proportion of cases occur in people with no family history of the disease. Race and ethnicity are also important factors. No modifiable risk factors for Type 1 diabetes have been clearly identified.

Type 2 diabetes results from a combination of genetic, environmental and behavioural risk factors. The risk of developing Type 2 diabetes increases significantly with age; the incidence of Type 2 diabetes is low before 30 years of age. Twin studies show a strong relationship between family history and Type 2 diabetes, although the actual genetic basis for the condition remains unknown. Family studies show that the presence of Type 2 diabetes in a family member is a risk factor; however, it is difficult to determine whether this represents the influence of genetics or shared environmental factors. Race and ethnic background are also associated with the development of Type 2 diabetes, with the prevalence of the condition being higher among Indigenous Australians and people of Pacific Islander, Asian and Southern European descent. Other risk factors for Type 2 diabetes include impaired glucose tolerance, overweight and obesity, physical inactivity and poor nutrition.

Urbanisation and increased modernisation have also been implicated in increasing the risk of Type 2 diabetes (Rewers & Hamman 1995). These risk factors are linked to lifestyle and behaviour associated with a westernised lifestyle. Westernisation may result in improvements in nutrition and life expectancy, but is also connected with obesity and reduced physical activity.

Another factor which has been associated with later development of Type 2 diabetes is intra-uterine and neonatal nutrition. Poor foetal nutrition leads to low birthweight for birthdate and may predispose individuals to Type 2 diabetes. If such individuals are exposed to other risk factors (obesity, ageing and physical inactivity) the likelihood of developing Type 2 diabetes is greater.

The risk of developing Type 2 diabetes can be decreased through lifestyle changes, hence national initiatives aimed at preventing Type 2 diabetes frequently focus on modifiable factors such as poor nutrition, physical inactivity, and overweight and obesity.

The risk factors for gestational diabetes are similar to those for Type 2 diabetes. Indeed, women who have had gestational diabetes are at greater risk of developing Type 2 diabetes in later life.

Box 3.1: The Metabolic Syndrome

The World Health Organization has classified a specific clustering of risk factors as the Metabolic Syndrome (Syndrome X). Insulin resistance is thought to be the underlying defect in this syndrome. In addition to insulin resistance, a person with the Metabolic Syndrome will usually have two or more of the following: glucose intolerance (impaired glucose tolerance or diabetes), dyslipidaemia, high blood pressure, central obesity and microalbuminuria. The syndrome greatly increases a person's risk of developing Type 2 diabetes or cardiovascular disease.

Source: WHO 1999.

Complications of diabetes may be macrovascular (diseases of the large blood vessels), microvascular (diseases of the small blood vessels), or associated with pregnancy. Macrovascular complications include coronary heart disease, stroke and peripheral vascular disease, and microvascular complications include retinopathy, kidney diseases and neuropathy. Regular screening is essential in detecting the development of complications, as all may progress to an advanced stage without symptoms.

After accounting for age and the duration of diabetes, the risk of microvascular complications is similar for Type 1 and Type 2 diabetes. However, macrovascular complications are more common with Type 2 diabetes. Any form of diabetes in pregnancy increases the risk of complications of pregnancy and childbirth, although gestational diabetes is not known to be associated with foetal malformations. Mothers with diabetes have a significantly higher occurrence of pre-term births compared to mothers without diabetes, and children of mothers with pre-existing or gestational diabetes may develop insulin resistance or impaired glucose tolerance early in life.

Many factors may contribute to the development of complications in people with diabetes, including age and possibly sex and genetic factors. In addition to these, modifiable factors including obesity, physical inactivity, high blood pressure, high cholesterol, tobacco smoking, hyperglycaemia, poor management of diabetes and a lack of access to appropriate care increase the risk of complications. Another important factor in the development of complications is the duration of diabetes. The risks of neuropathy, vision disorders, kidney disease, peripheral vascular disease, foot ulcers, amputations and coronary heart disease are increased with longer duration of diabetes. In fact, duration of diabetes is a far more important risk factor for kidney disease than any other. However, complications may progress without symptoms before diagnosis, especially for Type 2 diabetes which in many cases remains undiagnosed for years.

References and further reading

Hales N & Barker D 2001. The thrifty phenotype hypothesis. British Medical Bulletin 60:5–20.

Rewers M & Hamman R 1995. Risk factors for non-insulin dependent diabetes. In: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health. Diabetes in America. 2nd edn. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health.

WHO (World Health Organization) 1999. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: Diagnosis and classification of diabetes mellitus. Geneva: Department of Noncommunicable Disease Surveillance, WHO.

Overweight and obesity

The health risks associated with overweight and obesity are numerous and have been well documented. Excess weight is not only a risk factor for Type 2 diabetes but also for other diseases and conditions including coronary heart disease, stroke, heart failure, arthritis, reproductive problems and sleep apnoea. It is also associated with other risk factors such as high blood pressure, high blood cholesterol, and reduced life expectancy. Weight loss in those who are overweight reduces the incidence and severity of diabetes, high blood pressure and high cholesterol.

Excess weight, particularly abdominal obesity, is strongly linked to insulin resistance. Being overweight or obese increases the risk of developing Type 2 diabetes and, in those who already have diabetes, increases its severity. Overweight adults are up to three times more likely to develop Type 2 diabetes than those of ideal weight, while for those who are obese the risk is much greater, possibly up to ten times that of persons of ideal weight.

Obesity also increases the risk of developing coronary heart disease and peripheral vascular disease in people with and without diabetes. However, since diabetes is also a risk factor for these diseases, the risk is further increased in those people who have diabetes and are obese.

What is overweight and obesity?

The most common way of defining overweight and obesity is by body mass index (BMI), calculated as weight in kilograms divided by the squared height in metres. Generally, in adults a BMI of 25 or more is considered to indicate overweight, while a BMI of 30 or more indicates obesity. However, these values may not be appropriate for all ethnic groups. Different cutoffs, specific to age and sex, are used in children and adolescents.

In this document, unless otherwise specified, the term 'overweight' refers to people with a BMI of 25 or over, that is, it includes people who are obese.

Measurement of waist circumference may also be used as an indicator of excess abdominal weight. Fat distributed in the abdominal region is particularly associated with an increased risk of diabetes and cardiovascular disease. The World Health Organization reports that waist circumferences greater than 94 cm in men and 80 cm in women indicate increased risk (abdominal overweight), while measurements greater than 102 cm in men and 88 cm in women indicate greatly increased risk (abdominal obesity). Australia currently has no national standard cutoffs for waist circumference.

How many Australians are overweight and obese?

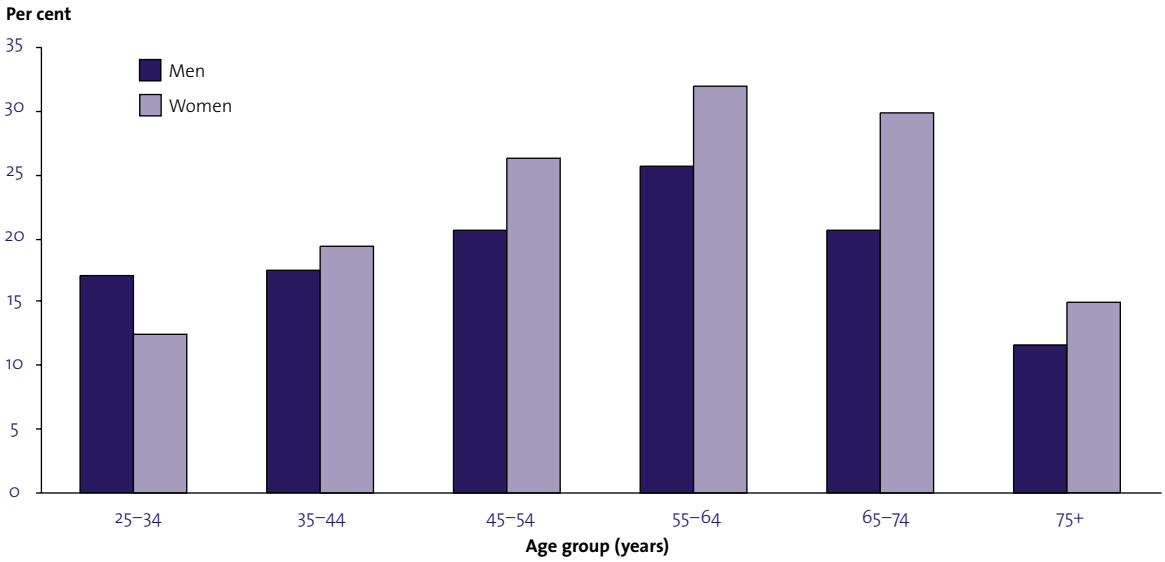
Estimates from the Australian Diabetes, Obesity and Lifestyle Study suggest that in 1999–2000 over seven million Australians aged 25 or over (60%) were overweight, as measured by BMI. Over two million of these (20% of the total population 25 or over) were obese.

In 1999–2000, men were more likely than women to be overweight. The proportion of overweight people (classified by BMI) increased with age and peaked at 55–64 years for men (74%) and at 65–74 years for women (69%). Obesity prevalence peaked at age 55–64 for both sexes (Figure 3.1).

People with Type 2 diabetes were more likely to be overweight than the general population. In particular, obesity was much more common in people with Type 2 diabetes. Among males aged 25 or over, 89% of those with Type 2 diabetes were overweight, with 62% being obese, compared with 67% overweight and 19% obese among all males of the same age. Among females, 64% of those with Type 2 diabetes were overweight with 43% obese, compared with 51% and 21% of all females aged 25 or over.

There are no national data on the prevalence of overweight and obesity among people with Type 1 diabetes. Evidence from other western countries suggests that adolescents, especially girls, with Type 1

Figure 3.1: Prevalence of obesity by age, 1999–2000



Source: 1999–2000 AusDiab.

diabetes are more likely to be overweight than their non-diabetic counterparts. Overweight in adolescents with Type 1 diabetes is often associated with poor metabolic control. However, adults with Type 1 diabetes are less likely to be overweight and obese than those without diabetes.

Using waist circumference measures, in 1999–2000 around 54% of Australians aged 25 or over were abdominally overweight while 29% were abdominally obese. Twenty-six per cent of males and 32% of females were abdominally obese. Women were more likely than men to be considered abdominally obese in all age groups except 40–44 years.

As with BMI, people with Type 2 diabetes are much more likely to be abdominally overweight and obese than the general population. Among people with Type 2 diabetes in 1999–2000, 87% of males and 67% of females were abdominally overweight while 64% of males and 53% of females were abdominally obese.

There has been a significant increase in the prevalence of overweight and obesity over the past 20 years. In men aged 25–64, the prevalence of overweight (measured by BMI) has increased from 48% in 1980 to 65% in 1999–2000 while the prevalence of obesity has risen from 8% to 17%. In women of the same age range, overweight has risen from 27% to 45% over the same period while obesity has more than doubled, from 7% to 19%.

Special population groups

Overweight and obesity is more common among women in lower socioeconomic groups and those living in remote areas. In 1995, around 53% of women in the lowest socioeconomic group were overweight and 24% were obese, compared with 44% and 14% in the highest socioeconomic group. Among women living in remote areas, 53% were overweight and 22% were obese compared with 47% and 18% of women living in urban or rural areas. For men there were no

significant differences in overweight or obesity relating to socioeconomic status or area of residence.

Aboriginal and Torres Strait Islander people

The Indigenous Australian population shows higher rates of overweight and obesity than the general population. While there was little difference in the proportion overweight between Indigenous Australian men in 1994 (62%) and all Australian men in 1995 (63%), Indigenous Australian men were more likely to be obese, at 25% compared with 18% of all Australian men. Among women, Indigenous Australians were much more likely to be overweight and obese than all Australian women, with rates of 60% and 28% among Aboriginal and Torres Strait Islander women compared with 49% and 18% of all Australian women.

Main data sources

1999–2000 Australian Diabetes, Obesity and Lifestyle Study (International Diabetes Institute & Commonwealth Department of Health and Aged Care).

1995 National Nutrition Survey (Australian Bureau of Statistics & Commonwealth Department of Health and Aged Care).

1994 National Aboriginal and Torres Strait Islander Survey (Australian Bureau of Statistics).

1989–90 & 1995 National Health Surveys (Australian Bureau of Statistics).

1980, 1983 & 1989 Risk Factor Prevalence Surveys (National Heart Foundation).

References and further reading

Booth ML, Wake M, Armstrong T, Chey T, Hesketh K & Mathur S 2001. The epidemiology of overweight and obesity among Australian children and adolescents, 1995–97. Australian and New Zealand Journal of Public Health 25:162–9.

Cunningham J & Mackerras D 1998. Overweight and obesity, Indigenous Australians. ABS Cat. No. 4702.0. Canberra: ABS.

Dunstan D, Zimmet P, Welborn T et al. 2001. Diabesity and associated disorders in Australia 2000. The accelerating epidemic. Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Melbourne: International Diabetes Institute.

Eckersley RM 2001. Losing the battle of the bulge: causes and consequences of increasing obesity. Medical Journal of Australia 174:590–2.

NHMRC (National Health and Medical Research Council) 1997. Acting on Australia's weight: a strategic plan for the prevention of overweight and obesity. Canberra: NHMRC.

Physical inactivity

Participation in regular moderate physical activity is known to be an essential factor in maintaining good health. Being physically inactive increases the risk of developing Type 2 diabetes, cardiovascular disease, colon cancer and breast cancer, and can increase the risk of musculoskeletal problems and injuries, particularly falls in the elderly. Physical inactivity also leads to increases in weight, blood pressure and blood cholesterol levels.

People who are physically inactive have increased insulin resistance, a major risk factor for the development of Type 2 diabetes. It is estimated that 30–50% of new cases of Type 2 diabetes could be prevented by appropriate levels of physical activity (Manson & Spelsberg 1994). For people who already have diabetes, being physically inactive increases the risk of developing complications, most notably coronary heart disease. It may also indirectly contribute to other complications such as kidney disease, peripheral vascular disease and retinopathy through its effect on weight, blood pressure and cholesterol levels.

Physical activity significantly improves glucose tolerance in those whose tolerance is impaired. Studies have shown that a program of physical activity and diet modification can delay or prevent progression from impaired glucose tolerance to Type 2 diabetes (Diabetes Prevention Program Research Group 2002; Tuomilehto et al. 2001). For people with diabetes, participation in regular physical activity can improve blood glucose control considerably. While a single bout of exercise can enhance insulin sensitivity in the short term, for longer term health benefits and control of blood glucose levels it is important that physical activity be regular and sustained, since the beneficial effects on glucose metabolism disappear quickly once activity ceases. Regular physical activity, combined with a controlled diet, can control Type 2 diabetes without the need for medication.

What is physical inactivity?

Physical inactivity is defined for this report as participating in less than 150 minutes of moderate-intensity activity per week. This figure is derived from the National Physical Activity Guidelines for Australians (DHAC 1999) which recommend that the accumulation of 30 minutes of moderate physical activity on most days of the week (interpreted here as 5 days) is beneficial for health. Moderate physical activities include brisk walking, social tennis, dancing and swimming. Regular participation in more vigorous activities such as jogging and aerobics also provides important health benefits.

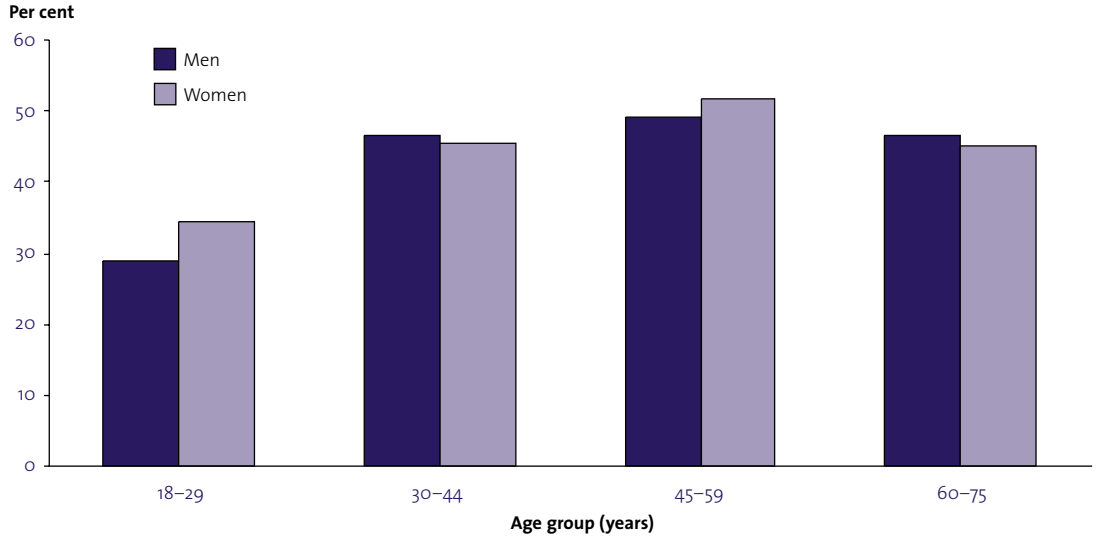
It has been shown that resistance exercise, such as lifting weights, can also have a beneficial effect on health in older people with diabetes. An Australian study (Dunstan 2002) demonstrated that strength training in combination with a healthy eating plan resulted in moderate weight loss, improved control over blood glucose levels, increased muscle strength, and improved emotional and functional wellbeing.

Since physical activity affects glucose metabolism, it is important that people with diabetes monitor their response to exercise and adjust their diet and medication accordingly. It is best to discuss any physical activity program with a general practitioner first.

How many Australians are physically inactive?

In 2000, 44% of Australians aged 18–75 years (around 5.8 million people) did not undertake physical activity at the levels recommended to achieve health benefits (Figure 3.2). Almost 15% of people did no leisure time physical activity at all. There has been a decrease in activity levels since 1997, when the proportion of physically inactive Australians was 38%. This decrease was seen for both sexes and among all age groups with the exception of those aged 60–75 years, in whom activity levels remained constant.

Figure 3.2: People who are physically inactive by age, 2000



Source: 2000 National Physical Activity Survey.

For both men and women, rates of physical inactivity were highest among 45–59-year-olds (49% and 52%, respectively) and lowest among 18–29-year-olds (29% and 34%). Those people with less than 12 years of education had a higher rate of physical inactivity than those with 12 years or more of education.

Data from the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study showed that people with Type 2 diabetes were more likely to be physically inactive than the general population. Among males with Type 2 diabetes aged 25 or over, 29% were undertaking less than 150 minutes of activity per week while a further 28% did no physical activity at all in their leisure time. This compares with 28% and 14% of all males of this age. Among females aged 25 or over, 55% of those with Type 2 diabetes did less than 150 minutes of activity per week while a further 12% were sedentary. For all females aged 25 or over the proportions were 36% and 17%, respectively.

Special population groups

The 1995 National Health Survey showed that people in the lowest socioeconomic group and those living in remote areas were more likely than other Australians to report doing no leisure time physical activity. Thirty-seven per cent of men and 40% of women in the lowest socioeconomic group were sedentary in their leisure time, compared with 27% and 29% of those in the highest group. Among people in remote areas, 37% of people were sedentary compared with 34% of those in urban areas and 32% of those in rural areas.

Aboriginal and Torres Strait Islander people

In 1995, Indigenous Australian women were more likely to be sedentary in their leisure time than other Australian women in all age groups, as were younger Indigenous Australian men (aged 18–44). Overall, 40% of Indigenous Australians reported no leisure time physical activity, as compared with 34% of other Australians.

Main data sources

2000 National Physical Activity Survey (Australian Sports Commission).

1999–2000 Australian Diabetes, Obesity and Lifestyle Study (International Diabetes Institute & Commonwealth Department of Health and Aged Care).

1995 National Health Survey (Australian Bureau of Statistics).

References and further reading

AIHW (Australian Institute of Health and Welfare): Armstrong T, Bauman A & Davies J 2000. Physical activity patterns of Australian adults. Results of the 1999 National Physical Activity Survey. Canberra: AIHW.

DHAC (Commonwealth Department of Health and Aged Care) 1999. National Physical Activity Guidelines for Australians. Canberra: DHAC.

Diabetes Prevention Program Research Group 2002. Reduction in the incidence of Type 2 diabetes with lifestyle intervention or metformin. New England Journal of Medicine 346:393–403.

Dunstan D 2002. Time to be physically active! Diabetes Management Journal 1:9.

Manson JE & Spelsberg A 1994. Primary prevention of non-insulin-dependent diabetes mellitus. American Journal of Preventive Medicine 10:172–84.

Tuomilehto J, Lindstrom J, Eriksson J et al. 2001. Prevention of Type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. New England Journal of Medicine 344(18):1343–50.

USDHHS (United States Department of Health and Human Services) 1996. Physical activity and health: a report of the Surgeon General. Atlanta, GA: National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, USDHHS.

Impaired 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.

What is impaired glucose tolerance?

In people with IGT, blood glucose levels are higher than normal but less than that required for a diagnosis of diabetes. Blood glucose levels normally rise after eating a meal then gradually fall as the meal is digested. However, these levels remain elevated in those with IGT. 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 are 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 (described in Box 2.1). People with IGT have a plasma glucose concentration of less than 7.0 millimoles/litre (mmol/L) before fasting and between 7.8 and 11.1 mmol/L two hours after the oral glucose load.

A new category, 'Impaired fasting glucose'—also considered to be predictive of diabetes—was adopted by the Australasian Working Party on Diagnostic Criteria for Diabetes Mellitus in 1999 (Colman et al. 1999). This category is based on an abnormal blood glucose measurement after fasting. The impaired fasting glucose category covers fasting plasma glucose levels between 6.1 mmol/L and 7.0 mmol/L.

The risk of macrovascular disease is increased in people with IGT, particularly when a person has other cardiovascular risk factors including obesity and high blood pressure. This clustering of interrelated cardiovascular risk factors is also known as Syndrome X (the Metabolic Syndrome, Box 3.1, page 22).

IGT is potentially avoidable. Improvements in glucose tolerance can be achieved through participation in regular physical activity and weight reduction. Clinical studies have shown that physical activity and physical fitness can increase insulin sensitivity and improve glucose tolerance (Takemura et al. 1999; Tuomilehto et al. 2001). In addition to controlling weight, exercise also improves the body's sensitivity to insulin, helping to lower blood glucose. Even a single bout of vigorous physical activity will have an immediate impact on insulin sensitivity.

People with IGT should be advised about lifestyle modifications. Such people are less likely to develop Type 2 diabetes if they maintain a reasonable level of fitness and lose weight if overweight or obese. Screening for other cardiovascular risk factors such as high blood pressure and high cholesterol is also recommended.

Who is affected by impaired glucose tolerance?

IGT is common in people who are physically inactive and obese, particularly those with high fat deposits in the abdominal region, and is more common in older people because 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 (see Table 3.1). Indeed, 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 3.1: Prevalence of impaired glucose tolerance, 1999–2000

Age group	Males (%)	Females (%)
25–34 years	2.1	4.9
35–44 years	4.8	8.5
45–54 years	8.4	11.2
55–64 years	14.8	15.2
65–74 years	20.4	22.9
75 years and over	25.5	20.7

Source: Dunstan et al. 2001.

The Australian Diabetes, Obesity and Lifestyle Study (AusDiab), carried out in 1999–2000, found that more than one in ten Australians aged 25 years or over has IGT—a prevalence of 10.6% (Dunstan et al. 2001). The condition was more common in females (12.0%) than in males (9.2%). Dunstan et al. compared their results with an earlier estimate of the prevalence of IGT from the 1981 Busselton Population Survey. Using the Busselton survey criteria for IGT, they found a substantial increase in the prevalence of IGT (Table 3.2) (Dunstan et al. 2001).

Table 3.2: Trends in the age-standardised prevalence of impaired glucose tolerance, 1981 and 1999–2000[(a)]

Survey	Males (%)	Females (%)
Busselton 1981	3.2	3.0
AusDiab 1999–2000	9.8	12.3

(a) Age-standardised to the 1998 Australian population.

Source: Dunstan et al. 2001.

Dunstan et al. (2001) suggest that a corresponding increase in the prevalence of obesity in Australia has been a significant contributing factor to the increasing prevalence of diabetes. Given the links between obesity and IGT, this trend may also contribute to the escalating prevalence of IGT.

Special population groups

There are limited data to estimate IGT prevalence in population subgroups; however, the risk of IGT is likely to be higher in subgroups that have a greater risk of Type 2 diabetes. Population subgroups at greater risk of Type 2 diabetes include Aboriginal and Torres Strait Islander peoples, and people from the Pacific Islands, the Indian subcontinent and South-East Asia.

Aboriginal and Torres Strait Islander people

De Courten et al. (1998) reviewed literature relating to the epidemiology, aetiology, pathogenesis and preventability of Type 2 diabetes in Aboriginal and Torres Strait Islander populations. The review cites only one study comparing IGT in Indigenous Australians with non-Indigenous Australians (Guest et al. 1992). In that study, prevalence rates of IGT were found to be similar in the two groups, although rates of diabetes in Indigenous Australians were twice those of non-Indigenous Australians.

A study of risk factors among Torres Strait Islander people (Leonard et al. 2002) found that 4.7% of study participants had IGT, while 26.2% of people had diabetes. The authors suggest that the lower prevalence of IGT among Torres Strait Islander people compared with other Australians reflects that these two populations are at different stages in the epidemic of obesity and diabetes.

References and further reading

Colman P, Thomas D, Zimmet P, Welborn T, Garcia-Webb P & Moore M 1999. New classification and criteria for diagnosis of diabetes mellitus. Medical Journal of Australia 170:375–8.

de Courten M, Hodge A, Dowse G, King I, Vickery J & Zimmet P 1998. Review of the epidemiology, aetiology, pathogenesis and preventability of diabetes in Aboriginal and Torres Strait Islander populations. Canberra: Commonwealth Department of Health and Family Services.

Dunstan D, Zimmet P, Welborn T et al. 2001. Diabesity and associated disorders in Australia 2000. The accelerating epidemic. Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Melbourne: International Diabetes Institute.

Guest C, O'Dea K, Hopper J, Nankervis A & Larkins R 1992. The prevalence of glucose intolerance in Aborigines and Europids of south-eastern Australia. Diabetes Research and Clinical Practice 15:227–35.

Harris MI & Zimmet PZ 1992. Classification of diabetes mellitus and other categories of glucose intolerance. In: Keen H, DeFronzo R, Alberti K & Zimmet P (eds). The international textbook of diabetes mellitus. London: John Wiley, 3–18.

Leonard D, McDermott R, O'Dea K et al. 2002. Obesity, diabetes and associated cardiovascular risk factors among Torres Strait Islander people. Australian and New Zealand Journal of Public Health 26(2):144–9.

Takemura Y, Kikuchi S, Inaba Y, Yasuda H & Nakagawa K 1999. The protective effect of good physical fitness when young on the risk of impaired glucose tolerance when old. Preventive Medicine 28:14–19.

Tuomilehto J, Lindstrom J, Eriksson J et al. 2001. Prevention of Type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. New England Journal of Medicine 344(18):1343–50.

Nutrition

Poor nutrition as a risk factor for diabetes

Poor nutrition is a risk factor for Type 2 diabetes and impaired glucose tolerance largely through its influence on body weight, in particular obesity. To date, the evidence linking specific dietary factors with increased risk of Type 2 diabetes is not conclusive. There is some evidence that increased intake of whole grains and dietary fibre may reduce the risk of developing diabetes. Furthermore, reduced intake of total fat, particularly saturated fat, may improve insulin sensitivity thereby reducing risk, independent of weight loss (Franz et al. 2002). When improved nutrition is incorporated into an overall risk reduction strategy, the risk of developing diabetes is further diminished. Dietary modification (such as reducing total and saturated fat intake, and increasing dietary fibre), exercise and weight reduction can effectively delay diabetes in overweight people with impaired glucose tolerance (Tuomilehto et al. 2001). The glycaemic index (see Box 3.2) of the total diet has also been linked to an increased risk of developing Type 2 diabetes (Salmeron et al. 1997).

Poor nutrition as a risk factor for diabetes complications

Poor nutrition is also a risk factor for several of the complications associated with diabetes including cardiovascular disease, kidney disease and dental disease. Improving dietary quality to minimise the risk of developing complications is an integral component in the management of both Type 1 and Type 2 diabetes. The primary aims of dietary therapy are to:

- achieve and maintain a healthy weight
- control blood glucose
- optimise blood lipids.

Weight loss is achieved through dietary energy restriction and increased physical activity, both of which result in decreased insulin resistance and subsequent improved blood glucose control. Regular daily eating patterns, a varied diet and frequent inclusion of low glycaemic index foods (such as lentils, pasta, noodles, multigrain bread and many fruits) also help in the management of blood glucose levels (Wahlqvist 1997). A reduced saturated fat intake is recommended to reduce LDL cholesterol. Reductions in

Box 3.2: Glycaemic index

The glycaemic index (G.I.) is a ranking of foods based on their overall effect on blood glucose levels. Eating foods containing carbohydrate (such as sugars and starches) causes blood glucose levels to rise to different levels depending on the rate of digestion of the food. It is the rise in the blood glucose level which is used to determine the G.I. of a food. The G.I. is expressed as a value between 1 and 100, with glucose being used as the reference value of 100. Foods with a low G.I. (such as grainy breads, porridge, pasta, milk, yoghurt, beans, lentils and fruit) release glucose gradually into the bloodstream whereas foods with a high G.I. (such as potatoes, white and wholemeal bread, processed breakfast cereals and many types of rice) cause a rapid and high rise in blood glucose levels. Selecting foods and planning meals with a lower G.I. is recommended for both the prevention of Type 2 diabetes and in managing blood glucose control in people who already have diabetes.

Source: Brand Miller et al. 1998.

total fat intake remain contentious as low-fat, high-carbohydrate diets increase triglycerides and lower HDL cholesterol, whereas the higher fat (Mediterranean-style) diet promotes lower triglycerides and higher HDL levels (Shrapnel 1994).

Dietary patterns of Australians

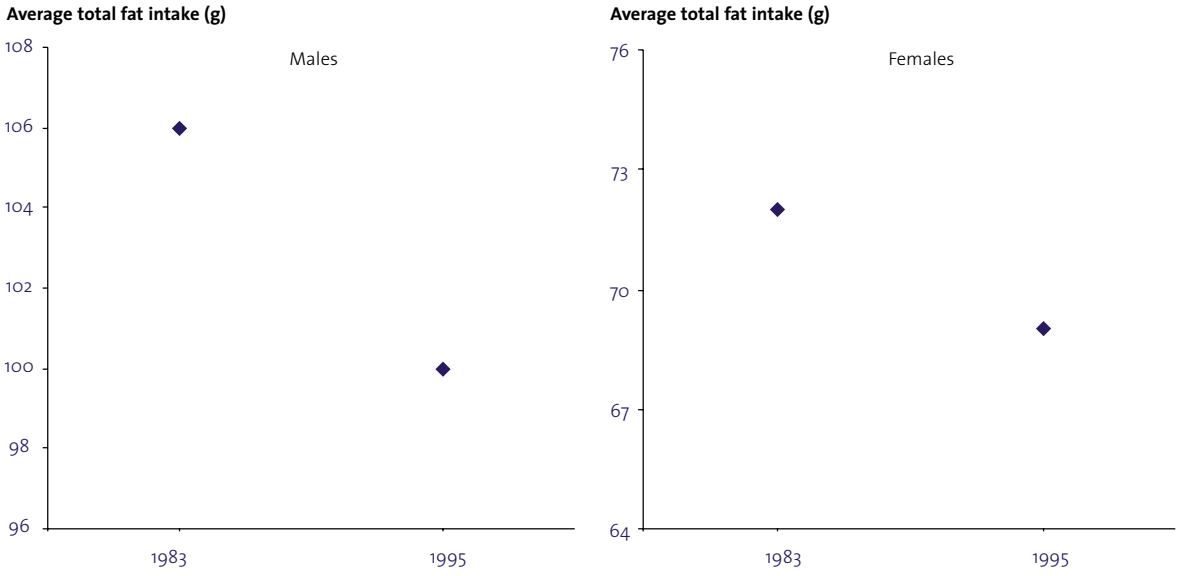
Adults

Among Australian adults, average total fat intake declined significantly between 1983 and 1995 (Figure 3.3). In 1995, saturated fat accounted for around 13% of total energy intake, higher than the recommended maximum level of 10%. The contribution of saturated fat to energy intake declined with age: 13.3% among 19–24 year olds compared with 12.0% among those aged 65 years and over (ABS & DHAC 1998).

Usual consumption of reduced fat or skim milk compared with full cream milk is a good indicator of lower total and saturated fat intake (Rutishauser et al. 2001). Data from the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) show that adult males with Type 2 diabetes are more likely to usually use reduced fat or skim milk (63%) than males without diabetes (46%), whereas the reverse pattern was evident among adult females with and without diabetes (43% and 60%, respectively).

Despite favourable trends in total fat consumption, energy intake among Australian adults increased significantly between 1983 and 1995 (Figure 3.4), a factor contributing to the increase in overweight and obesity among adults observed over the last two decades (see 'Overweight and obesity' in this chapter). The increase in daily energy intake (approximately 350 kJ) equates to an extra slice of bread per day.

Figure 3.3: Average total fat intake among adults aged 25–64 years in capital cities

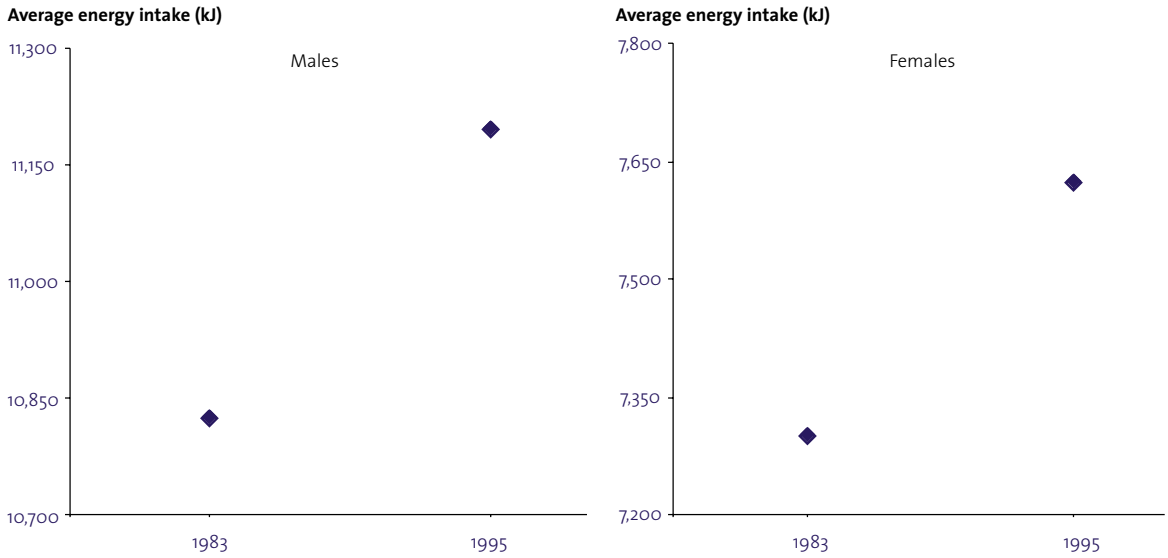


Notes

1. Different scales have been used for males and females due to their differing dietary requirements and intake.
2. Difference between estimated means for 1983 and 1995 is statistically significant at the 0.01 level.

Source: Cook, Coles-Rutishauser & Seelig 2001.

Figure 3.4: Average energy intake among adults aged 25–64 years in capital cities



Notes

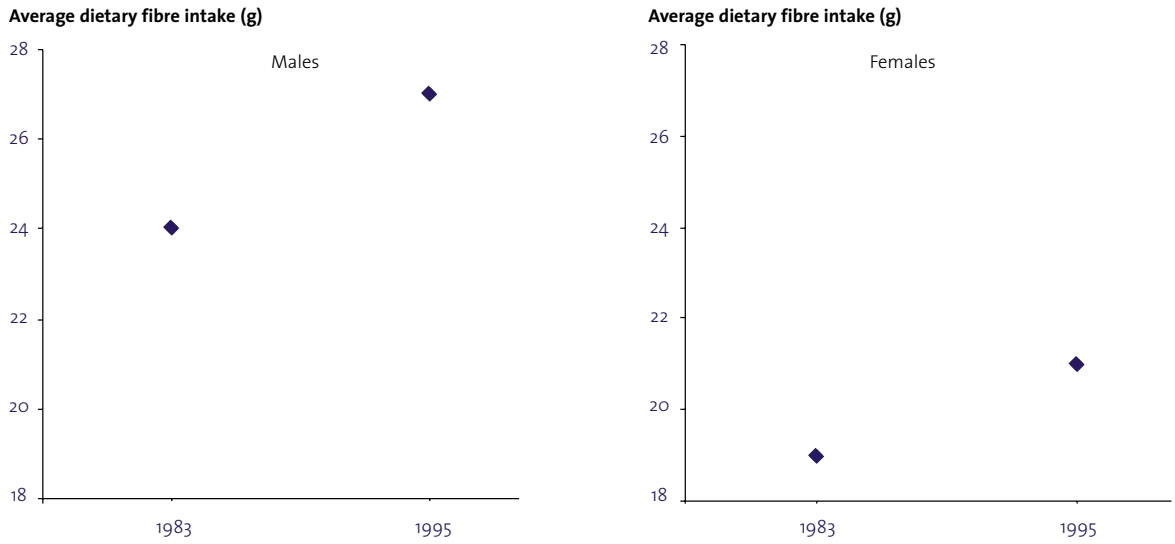
1. Different scales have been used for males and females due to their differing dietary requirements and intake.
2. Difference between estimated means for 1983 and 1995 is statistically significant at the 0.01 level.

Source: Cook, Coles-Rutishauser & Seelig 2001.

Dietary fibre intake is an indicator of the consumption of plant foods. Average dietary fibre intake among Australian adults increased significantly between 1983 and 1995 although it was still well below the recommended 30 g per day in 1995 (Figure 3.5). In 1995, dietary fibre intake was substantially higher among males (27 g) compared with females (21 g).

Data from the 1999–2000 AusDiab study show that a higher proportion of adult males with diabetes usually consume two or more serves of fruit (52%) and four or more serves of vegetables per day (35%) compared with males without diabetes (39% and 28%, respectively). By contrast, fewer adult females with diabetes consumed two or more serves of fruit (35%) and four or more serves of vegetables (22%) than females without diabetes (50% and 41%, respectively).

Figure 3.5: Average dietary fibre intake among adults aged 25–64 years in capital cities



Note: Difference between estimated means for 1983 and 1995 is statistically significant at the 0.01 level.

Source: Cook, Coles-Rutishauser & Seelig 2001.

Children and adolescents

Substantial recent increases in the prevalence of overweight and obesity among children and adolescents highlight the need to track similar dietary factors to those described for adults. Of particular interest is energy intake because of its relationship with overweight. Between 1985 and 1995, there were significant increases in the estimated daily energy intake among both boys and girls aged 10–15 years: 15% and 12%, respectively (Figure 3.6). Possible contributors to the increased energy intake were substantial increases in the consumption of cereal-based foods (biscuits, cakes etc.), confectionery, non-alcoholic beverages and sugar products (honey, jams etc.). There were no significant increases in commonly consumed food groups such as cereals, fruit, vegetables and meats.

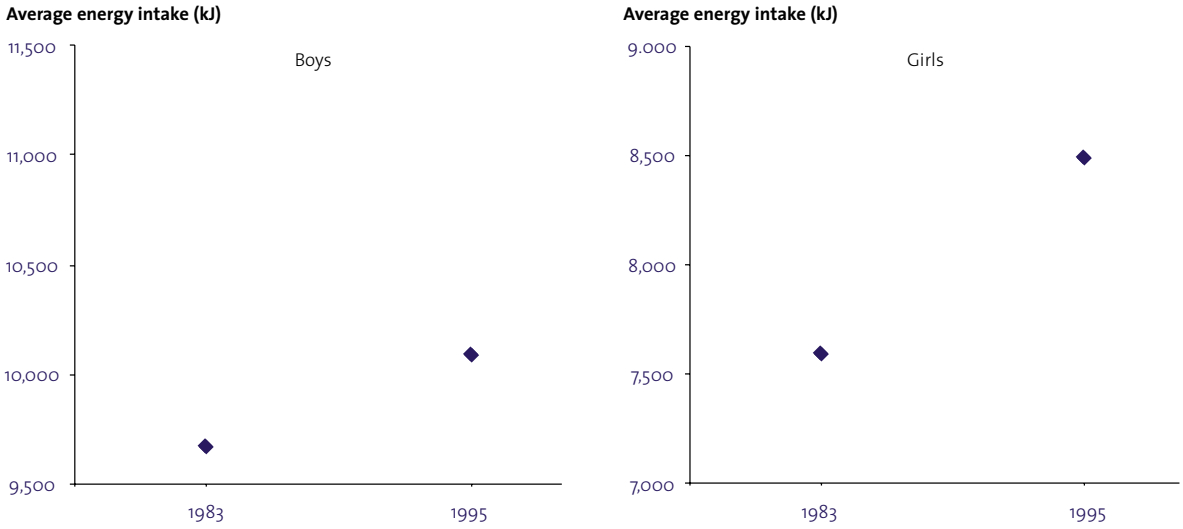
Contrasting the decrease in total fat consumption among adults, total fat intake among children increased during this period, but not significantly. Dietary fibre increased significantly, following a similar trend in adults, despite there being no significant increases in cereal, fruit, vegetable or legume intake (Cook, Coles-Rutishauser & Seelig 2001).

Aboriginal and Torres Strait Islander people

There are limited data available on the dietary patterns of Aboriginal and Torres Strait Islanders. However, there is information available about some of the outcomes associated with poor nutrition.

Poor foetal nutrition leading to low birthweight followed by obesity in adulthood is associated with an increased risk of developing Type 2 diabetes (Forsen et al. 2000). Both low birthweight and obesity are more prevalent in the Aboriginal and Torres Strait Islander population compared with the non-Indigenous population. Aboriginal mothers are twice as likely to give birth to low birthweight babies compared with other Australian mothers. Low birthweight is also an issue among Torres Strait Islanders, yet in lower

Figure 3.6: Average energy intake among children aged 10–15 years



Notes

1. Different scales have been used for males and females due to their differing dietary requirements and intake.
2. Difference between estimated means for 1983 and 1995 is statistically significant at the 0.01 level.

Source: Cook, Coles-Rutishauser & Seelig 2001.

proportions than in Aboriginals (NATSINWP 2001). Data collected in the mid-1990s showed that obesity was more prevalent among Indigenous Australian men (25%) than all Australian men (18%). The trend was similar among Indigenous women and all Australian women, 28% and 18% respectively.

Main data sources

1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) (International Diabetes Institute & Commonwealth Department of Health and Aged Care).

1995 National Nutrition Survey (Australian Bureau of Statistics & Commonwealth Department of Health and Aged Care).

1985 National Dietary Survey of School Children (Commonwealth Department of Health and Aged Care).

1983 National Dietary Survey of Adults (Commonwealth Department of Health and Aged Care & National Heart Foundation).

References and further reading

ABS & DHAC (Australian Bureau of Statistics & Commonwealth Department of Health and Aged Care) 1998. National Nutrition Survey: nutrient intakes and physical measurements, Australia, 1995. ABS Cat. No. 4805.0. Canberra: ABS & DHAC.

Brand Miller J, Foster-Powell K, Colagiuri S & Leeds A 1998. The G.I. factor. Rev. edn. Sydney: Hodder & Stoughton.

Cook T, Coles-Rutishauser I & Seelig M 2001. Comparable data on food and nutrient intake and physical measurements from the 1983, 1985 and 1995 national surveys. Canberra: Commonwealth Department of Health and Aged Care.

Forsen T, Eriksson J, Tuomilehto J et al. 2000. The fetal and childhood growth of persons who develop Type 2 diabetes. Annals of Internal Medicine 133(3):176–82.

Franz MJ, Bantle JP, Beebe CA et al. 2002. Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. Diabetes Care 25:148–98.

NATSINWP (National Aboriginal and Torres Strait Islander Nutrition Working Party) 2001. National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan 2000–2010 and first phase activities 2000–2003. Indigenous component of Eat Well Australia, developed by the Strategic Inter-Governmental Nutrition Alliance (SIGNAL) of the National Public Health Partnership (NPHP). Canberra: SIGNAL & NPHP.

O'Dea K, Colaguiri S, Hepburn A, Holt P & Colaguiri R 2002. Evidence based guidelines for Type 2 diabetes: primary prevention. Canberra: Diabetes Australia & National Health and Medical Research Council.

Rutishauser IHE, Webb K, Abraham B & Allsopp R 2001. Evaluation of short dietary questions from the 1995 NNS. Canberra: Commonwealth Department of Health and Aged Care.

Salmeron J, Ascherio A, Rimm EB et al. 1997. Dietary fiber, glycemic load, and risk of NIDDM in men. Diabetes Care 20:545–50.

Shrapnel WS 1994. Diets, triglycerides and diabetes. Nutrition Issues and Abstracts. No. 2 August 1994.

Tuomilehto J, Lindstrom J, Eriksson JG et al. 2001. Prevention of Type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. New England Journal of Medicine 344:1342–50.

Wahlqvist M 1997. Nutrition and diabetes. Australian Family Physician 26(4):384–9.

Tobacco smoking

Tobacco smoking increases the risk of developing diabetes-related complications such as retinopathy, coronary heart disease, stroke and peripheral vascular disease. Tobacco smoking reduces insulin sensitivity and increases blood cholesterol levels (Mikhailidis et al. 1998). Passive exposure to smoke also has serious health consequences, including increased risk of heart disease among adults.

What is tobacco smoking?

Smoking here refers to the smoking of tobacco products, including packet cigarettes, roll-your-own cigarettes, pipes and cigars. 'Daily smokers' refers to those who smoke daily or on most days.

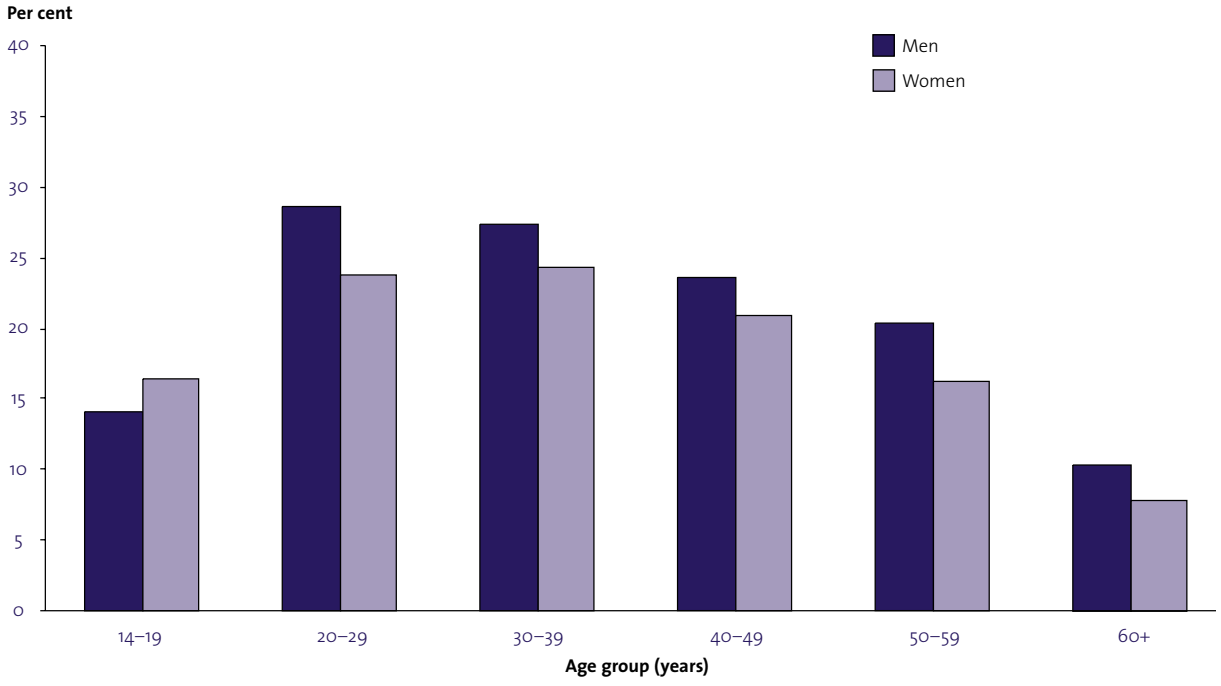
How many Australians smoke?

In 2001, approximately 3.1 million (19.5%) Australians aged 14 years or over smoked on a daily basis, with males more likely to be daily smokers (21%) than females (18%). The highest rates of regular smoking occurred among males aged 20–29 years (28%) and females aged 30–39 years (24%) (Figure 3.7). From these age groups, regular smoking declines with age, with those aged 60 years and over recording the lowest rates at 10% for males and 8% for females.

The prevalence of people with diabetes who smoked was similar to the rest of the Australian population (Nutbeam, Thomas & Wise 1993:55).

In 2001, around 30% of males and 23% of females aged 14 years or over reported that they were ex-smokers, while a further 45% and 56%, respectively, stated that they had never smoked.

Figure 3.7: Proportion of people who are daily smokers, 2001



Source: 2001 National Drug Strategy Household Survey.

Smoking rates among Australian adults have been declining steadily since the 1950s, when it was estimated that 70% of men and 30% of women smoked. This trend has continued into the 1990s and for the first time dropped to less than 20% in 2001 (Figure 3.8).

Special population groups

Smoking is more common among people from lower socioeconomic backgrounds when compared with people from higher socioeconomic backgrounds. The 2001 National Drug Strategy Household Survey found that around 24% of those from the most disadvantaged socioeconomic quintile reported that they smoked daily, compared with approximately 14% of those from the least disadvantaged socioeconomic quintile.

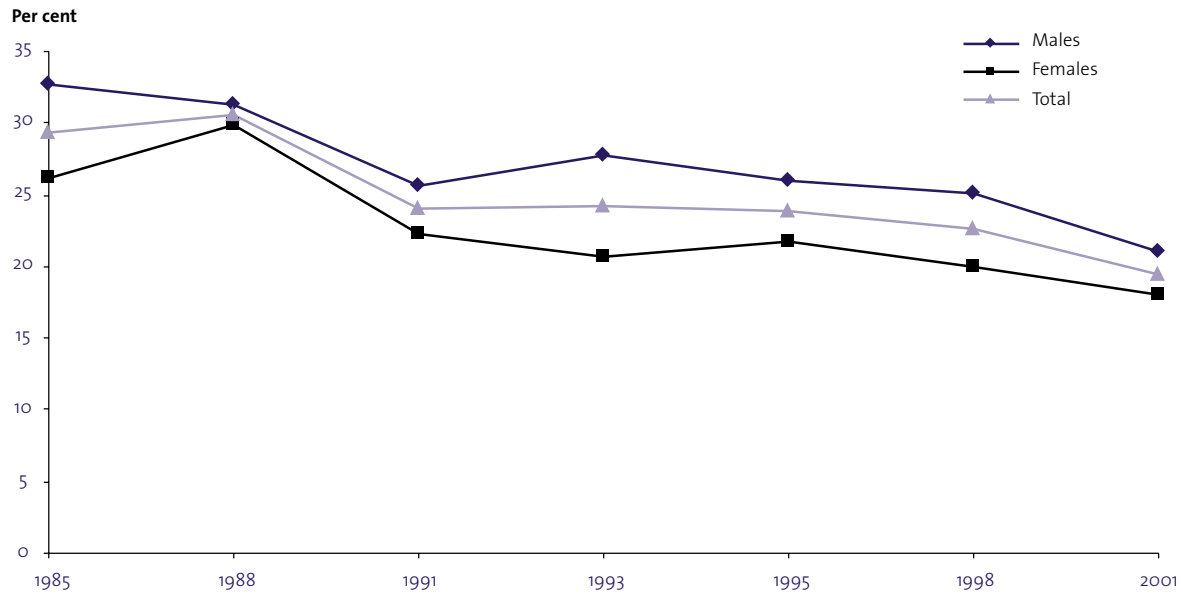
In 1998, a slightly higher percentage of individuals living in rural and remote areas reported that they were daily smokers (26%), compared to those living in urban communities (21%). Further, 35% of urban Australians aged 14 years or over reported that they had never smoked, compared with 31% of Australians in rural and remote areas.

In 1998, the Northern Territory recorded the highest smoking prevalence, with around 31% of those aged 14 years or over indicating that they smoked daily. This contrasted with South Australia, which recorded the lowest rate at around 19%.

Aboriginal and Torres Strait Islander people

In 2001, Indigenous Australians aged 14 years or over were more than twice as likely to smoke when compared with their non-Indigenous counterparts.

Figure 3.8: Proportion of people aged 14 years or over who are daily smokers, 1985–2001



Source: AIHW 2002.

Around 46% of Indigenous Australians aged 14 years or over were current smokers compared with 19% of non-Indigenous Australians. Indigenous Australians were also less likely than non-Indigenous Australians to be former smokers or to have never smoked (Figure 3.9)

Main data sources

2001 National Drug Strategy Household Survey (Commonwealth Department of Health and Ageing).

1995 National Health Survey (Australian Bureau of Statistics).

References and further reading

ABS (Australian Bureau of Statistics) 1999. 1995 National Health Survey: Aboriginal and Torres Strait Islander results. ABS Cat. No. 4806.0. Canberra: ABS.

AIHW (Australian Institute of Health and Welfare) 1996. Tobacco use and its health impact in Australia. AIHW Cat. No. CVD 1. Canberra: AIHW.

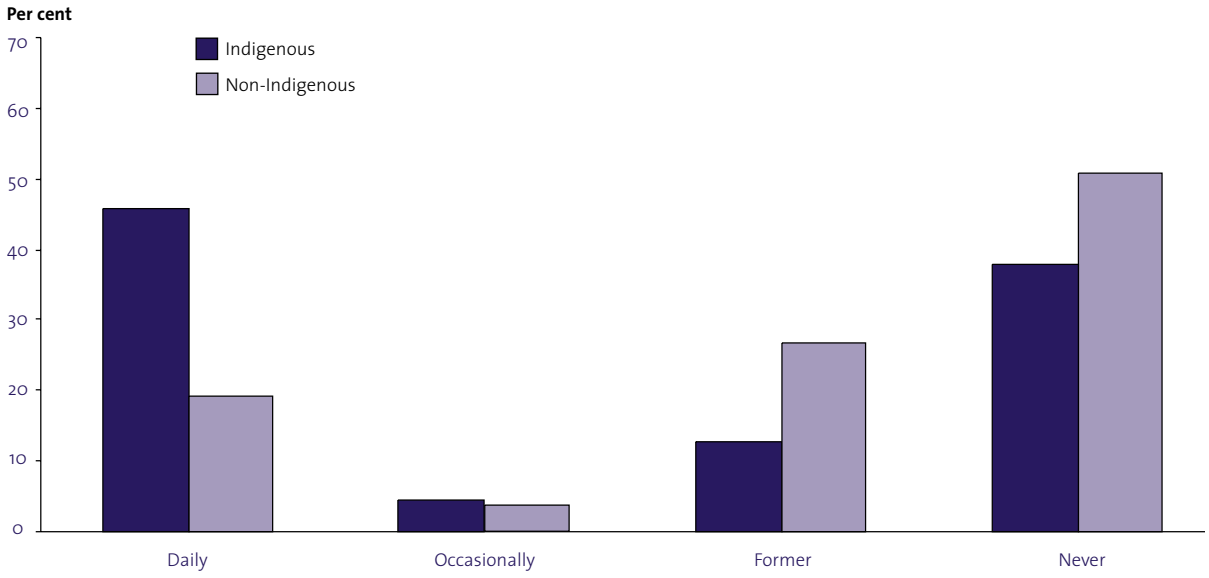
AIHW 1999. 1998 National Drug Strategy Household Survey: first results. Drug Statistics Series. AIHW Cat. No. PHE 15. Canberra: AIHW.

AIHW 2002. Australia's health 2002. Canberra: AIHW.

AIHW: Adhikari P & Summerill A 2000. 1998 National Drug Strategy Household Survey: detailed findings. Drug Statistics Series No. 6. AIHW Cat. No. PHE 27. Canberra: AIHW.

AIHW: Higgins K, Cooper-Stanbury M & Williams P 2000. Statistics on drug use in Australia 1998. Drug Statistics Series. AIHW Cat. No. PHE 16. Canberra: AIHW.

Figure 3.9: Smoking status, Indigenous and non-Indigenous Australians, 2001



Notes

1. 'Occasional' means smokes less than daily; 'former' means (presently) no longer smoking; 'never' means having consumed no more than 100 cigarettes (or equivalent).
2. Includes people aged 14 years or over.

Source: 2001 National Drug Strategy Household Survey.

AIHW: Miller M & Draper G 2001. Statistics on drug use in Australia 2000. Drug Statistics Series No. 8. AIHW Cat. No. PHE 30. Canberra: AIHW.

AIHW: Ridolfo B & Stevenson C 2001. The quantification of drug-caused mortality and morbidity in Australia, 1998. Drug Statistics Series No. 7. AIHW Cat. No. PHE 29. Canberra: AIHW.

Cunningham J 1997. Cigarette smoking among Indigenous Australians. ABS Cat. No. 4701.0. Canberra: ABS.

Hill DJ, White V & Letcher T 1999. Tobacco use among Australian secondary students in 1996. Australian and New Zealand Journal of Public Health 23:252–9.

Mikhailidis DP, Papadakis JA & Ganotakis ES 1998. Smoking, diabetes and hyperlipidaemia. Journal of the Royal Society of Health 118(2):91–3.

Nutbeam D, Thomas M & Wise M 1993. National Action Plan. Diabetes to the year 2000 and beyond. A plan for the prevention and control of non-insulin dependent diabetes mellitus (NIDDM) in Australia. Canberra: Australian Diabetes Society.

High blood pressure

High blood pressure (also referred to as hypertension) is linked to diabetes, existing often with central obesity and high cholesterol levels. It is a major risk factor known to contribute to or lead to the development of complications among people with diabetes. These complications include coronary heart disease, stroke, peripheral vascular disease, nephropathy and retinopathy. Mortality, primarily from coronary heart disease and stroke, is 4–5 times more likely in people with Type 2 diabetes and high blood pressure (Gilbert et al. 1995). The risk of disease increases as the level of blood pressure increases.

People who are overweight, physically inactive, have high dietary salt intake or are under mental stress are more likely to develop high blood pressure.

Data from the 2000–01 study of general practice activity in Australia show that high blood pressure accounted for 6% of all conditions managed by general practitioners (AIHW: Britt et al. 2001). Diabetes was one of the most common conditions managed with high blood pressure at 7.7 per 100 high blood pressure encounters. This rate is above average, indicating a relationship between these conditions (AIHW: Britt et al. 2001).

What is high blood pressure?

Blood pressure represents the forces exerted by blood on the walls of the arteries and is written as systolic/diastolic (e.g. 120/80 mm Hg, stated as '120 over 80').

The continuous relationship between blood pressure levels and cardiovascular disease risk, and the 'arbitrary' nature of the definition of high blood pressure, has contributed to the variation in the definitions issued by various national and international authorities for population surveys and clinical guidelines.

New classifications for the clinical management of high blood pressure have recently been released by the World Health Organization (1999). These new guidelines define high blood pressure as:

- systolic blood pressure (SBP) greater than or equal to 140 mm Hg; and/or
- diastolic blood pressure (DBP) greater than or equal to 90 mm Hg; and/or
- receiving medication for high blood pressure.

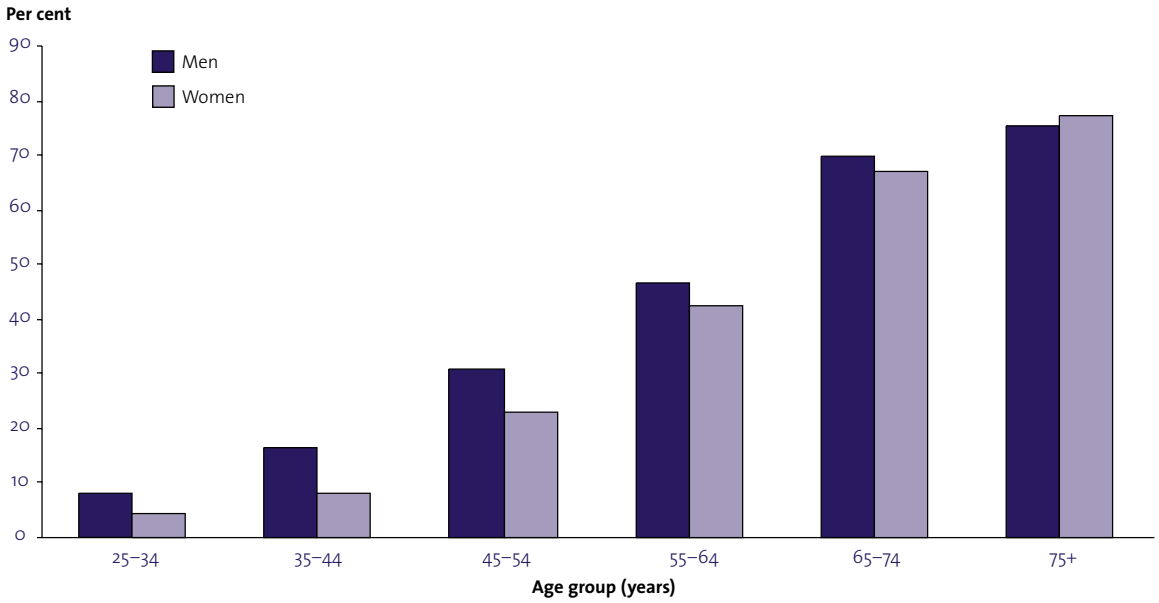
For the purposes of this report, high blood pressure is defined using these guidelines.

Previously, in Australia, high blood pressure was defined as a SBP of 160 mm Hg or greater and/or a DBP of 95 mm Hg or greater and/or receiving medication for high blood pressure. Data published using the old classifications (for example, *Australia's Health 2000*) should not be compared with data using the new guidelines such as in this publication.

How many Australians have high blood pressure?

In 1999–2000, over 3.6 million Australians (29%) aged 25 years or over had high blood pressure or were on medication for that condition. High blood pressure was more common among men aged 25 years or over (31%) than women (26%). The proportion of people with high blood pressure increased with age. Among men aged 65–74 years, 70% had high blood pressure or were on medication for treatment of high blood pressure. Almost 67% of women in that age group had high blood pressure or were on medication for that condition (Figure 3.10).

Figure 3.10: Proportion of people with high blood pressure, 1999–2000



Note: Based on WHO definition of high blood pressure.

Source: 1999–2000 AusDiab.

In 1999–2000, high blood pressure was much more frequent in people with diabetes compared with the general population. Nearly 61% of men aged 25–59 years with diabetes had high blood pressure compared with 19% of non-diabetic men in the same age group. For men with diabetes aged 60 years or over, almost 53% had high blood pressure compared with 45% of non-diabetic men of the same age group. Around 32% of women with diabetes aged 25–59 years had high blood pressure compared with 12% of non-diabetic women. For women with diabetes aged 60 years or over, 55% had high blood pressure compared with 49% of non-diabetic women in the same age group.

There have been significant declines in the proportion of people with high blood pressure and/or receiving treatment since the 1980s. The proportion of men (aged 25–64 years) with high blood pressure has fallen steadily from 45% in 1980 to 22% in 1999–2000. The rate for women (aged 25–64 years) has fallen steadily from 29% in 1980 to 16% in 1995, and has not changed since (Figure 3.11).

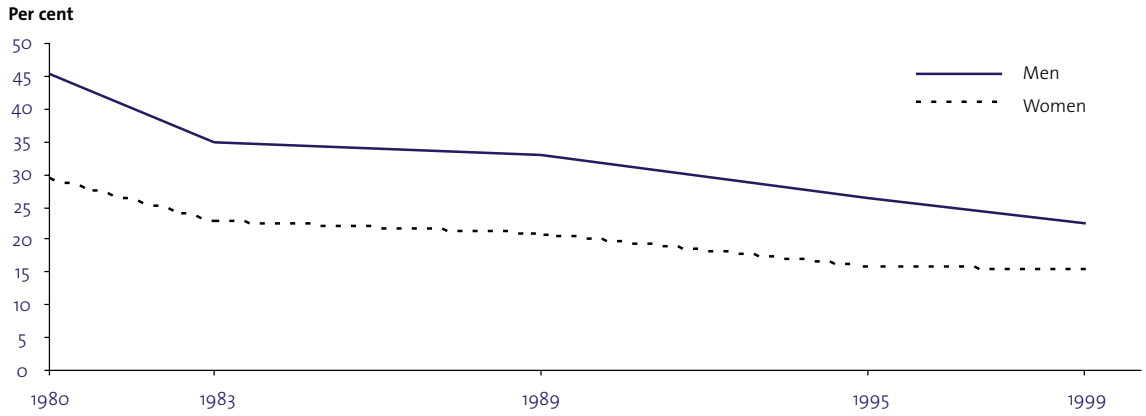
There has also been a significant decline in average blood pressure levels during the same period. This decline occurred equally among those not on medication for high blood pressure as among those on treatment.

Special population groups

In 1995, the prevalence of high blood pressure among women increased with increasing socioeconomic disadvantage. Although not significantly different, 25% of women in the lowest socioeconomic group had high blood pressure compared with 17% of those in the highest group. There was no significant difference in the prevalence of high blood pressure among men in the lowest socioeconomic group (31%) and those in the highest group (29%).

In 1995, there were no significant differences in the prevalence of high blood pressure between urban, rural and remote areas. Around 22% of urban, rural and remote women had high blood pressure. For men, estimated rates were 29–30% in urban, rural and remote regions.

Figure 3.11: Proportion of people aged 25–64 years with high blood pressure, 1980 to 1999–2000



Notes

1. Age-standardised to the 1991 Australian population.
2. Based on WHO definition of high blood pressure.
3. Capital cities only.

Sources: 1980, 1983, 1989 Risk Factor Prevalence Surveys; 1995 National Nutrition Survey; 1999–2000 AusDiab.

Differences in the prevalence of high blood pressure between States were also not significant. The highest rates were in Tasmania and South Australia (around 30%), and the lowest were in the Northern Territory (22%).

Aboriginal and Torres Strait Islander people

There are no measured national data to assess the rates of high blood pressure among Aboriginal and Torres Strait Islander people. However, a study by Smith et al. (1992) found the prevalence of high blood pressure in Indigenous Australians from the Kimberley region to be two to three times higher than among Caucasian Australians.

Main data sources

1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) (International Diabetes Institute & Commonwealth Department of Health and Aged Care).

1995 National Nutrition Survey (Australian Bureau of Statistics & Commonwealth Department of Health and Aged Care).

1980, 1983, 1989 Risk Factor Prevalence Surveys (National Heart Foundation).

References and further reading

AIHW (Australian Institute of Health and Welfare): Britt H, Miller GC, Knox S et al. 2001. General practice activity in Australia 2000–01. General Practice Series No. 8. AIHW Cat. No. GEP 8. Canberra: AIHW.

AIHW 2000. Australia's health 2000. AIHW Cat. No. AUS 19. Canberra: AIHW.

AIHW 2001. Heart, stroke and vascular diseases—Australian facts 2001. Cardiovascular Disease Series No. 14. AIHW Cat. No. CVD 13. Canberra: AIHW, National Heart Foundation of Australia & National Stroke Foundation of Australia.

Dunstan D, Zimmet P, Welborn T et al. 2001. Diabesity and associated disorders in Australia 2000. The accelerating epidemic. Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Melbourne: International Diabetes Institute.

Gilbert D, Jasik M, DeLuise M, O'Callaghan C & Cooper M 1995. Diabetes and hypertension. Australian Diabetes Society position statement. Medical Journal of Australia 163:372–5.

Smith RM, Spargo RM, Hunter EM et al. 1992. Prevalence of hypertension in Kimberley Aborigines and its relationship to ischaemic heart disease. An age stratified random survey. Medical Journal of Australia 156:557–62.

WHO (World Health Organization) 1999. International Society of Hypertension guidelines for the management of hypertension—guidelines subcommittee. Journal of Hypertension 17:151–83.

High cholesterol and triglycerides

People with diabetes, particularly those with Type 2 diabetes, often have blood fat abnormalities and their diabetic condition can intensify the risk that these abnormalities normally carry. Blood fat abnormalities, known as dyslipidaemia, are irregularities in fat metabolism and include high levels of cholesterol and triglycerides. Like many Australians, people with diabetes often have high cholesterol, which is a major risk factor for coronary heart disease. They are also more likely than those without diabetes to have high triglycerides and low high-density lipoprotein (HDL) levels.

Dyslipidaemia is a major risk factor for diabetes-related complications, coronary heart disease and possibly some types of stroke. It is one of the main causes of atherosclerosis, the process that can clog the blood vessels that supply the heart and other parts of the body. This process may be intensified by diabetes. High triglyceride levels and low HDL levels have been repeatedly linked with Type 2 diabetes, impaired glucose tolerance and the Metabolic Syndrome (Syndrome X, Box 3.1) (Rewers & Hamman 1995). However, raised total cholesterol and low-density lipoprotein (LDL) are also common in people with diabetes, with raised LDL often the primary focus of treatment.

What are high cholesterol and triglycerides?

Cholesterol and triglycerides are fats found in the blood. Cholesterol is metabolised by the liver and carried by the blood supply 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 angina, heart attack or stroke.

For most people, saturated fat in the diet is the main factor that raises blood cholesterol levels. Cholesterol in foods can also raise blood cholesterol levels, but less than saturated fat does. Genetic factors can also affect blood cholesterol—some people have high cholesterol levels regardless of their saturated fat and cholesterol dietary intake and are at increased risk of coronary heart disease (Bouchard et al. 1997). Hyperglycaemia is also known to contribute to high blood cholesterol levels (DHAC & AIHW 1999).

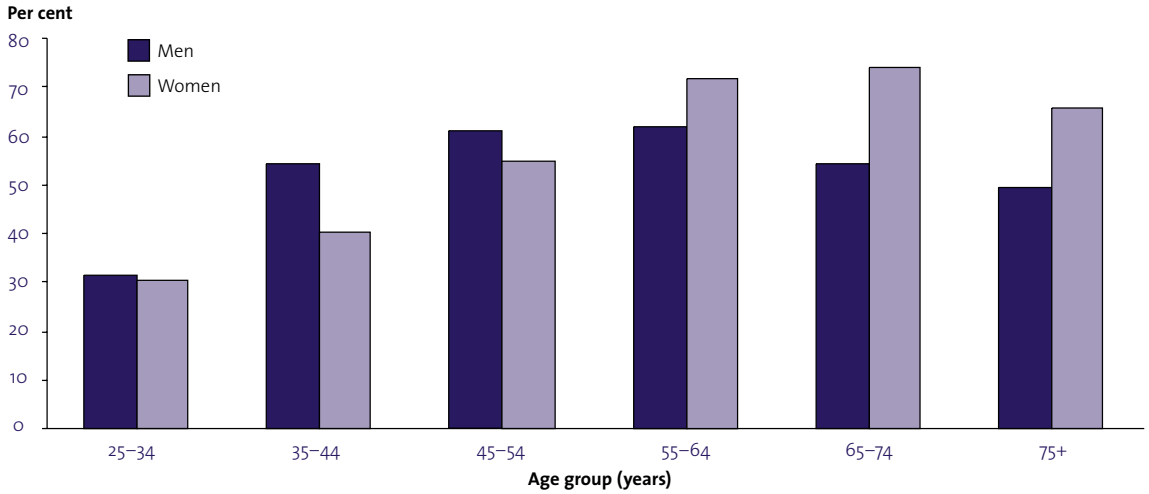
Determining levels of different fats in the blood can include measuring:

- total cholesterol, which includes LDL cholesterol and HDL cholesterol. The risk of heart disease increases steadily from low cholesterol levels.
- LDL cholesterol, often referred to as 'bad' cholesterol, which is the main cause of obstructions in the arteries when in excess. High levels of LDL lead to greater risk of heart disease.
- HDL cholesterol, otherwise known as the 'good' cholesterol, which has a protective effect against heart disease.
- triglycerides, which are formed from the digestion of fats in food. Triglyceride levels can fluctuate according to dietary fat intake. In excess they may contribute to the development of atherosclerosis, but are generally considered less important than excess LDL.

How many people have high cholesterol and triglycerides?

The 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) revealed that 58% of males and 69% of females with Type 2 diabetes (aged 25 years or over) had high total cholesterol (5.5 mmol/L or greater). Among the general population the rate was around 50% in both sexes. Analysis of AusDiab reveals that in the general population high-risk total cholesterol increased with age—to ages 65–74 in women and 55–64 in men (Figure 3.12).

Figure 3.12: People with high blood cholesterol, 1999–2000



Source: 1999–2000 AusDiab.

The prevalence of low HDL (less than 1.0 mmol/L) was also found to be higher among people with Type 2 diabetes—22.4% in males and 24% in females, compared with 18% and 5% respectively in the general population. Taskinen (1999) notes that LDL cholesterol levels are one-and-a-half to three times higher in people with Type 2 diabetes than in people without diabetes.

Prevalence of fasting hypertriglyceridaemia (triglyceride levels greater than 4.0 mmol/L) was four times as high among females with Type 2 diabetes and twice as high among males with Type 2 diabetes compared with males and females in the general population. National Health Priority Areas indicators 2.4 and 2.5 in the Appendixes provide information on total cholesterol and triglycerides.

Average blood cholesterol levels in 1999–2000 were very similar to those 20 years earlier, for men and for women (Table 3.3). Although average cholesterol levels in Australia in 1999–2000 were 5.6 mmol/L for men and 5.5 mmol/L for women, it is biologically achievable for people to have lower cholesterol levels. Cholesterol levels in societies with hunter-gatherer or agriculture-based lifestyles are much lower, as are their rates of cardiovascular disease (Forge 1999).

There has been no marked reduction in the prevalence of people with high blood cholesterol since 1980, when nationwide monitoring began (Figure 3.13) (AIHW 2001).

Table 3.3: Average blood cholesterol levels, 1980 to 1999–2000

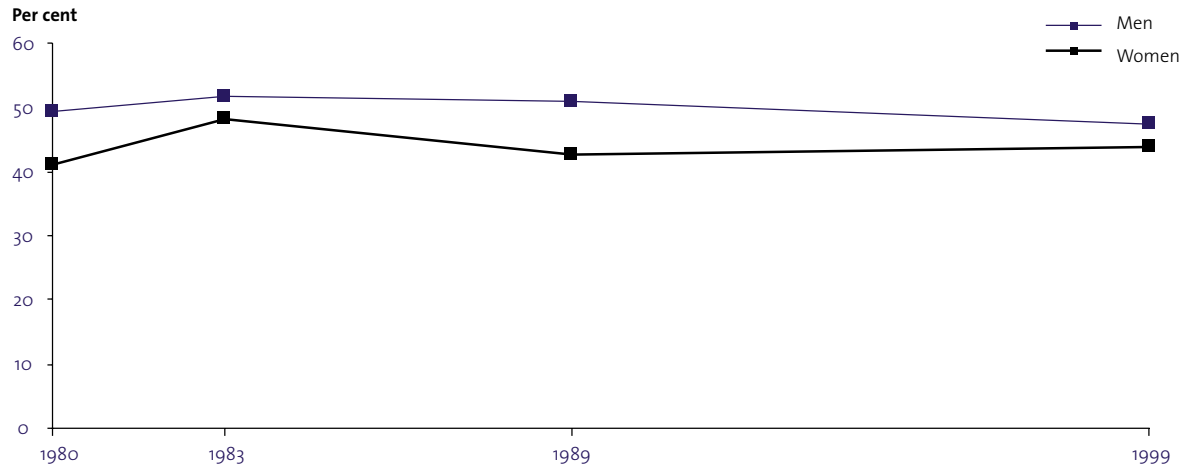
Sex	1980	1983	1989	1999–2000
		mmol/L		
Men	5.6	5.7	5.6	5.6
Women	5.5	5.6	5.5	5.5

Notes

1. Age-standardised to the 1991 Australian population.
2. Includes persons aged 25–64.
3. For capital cities only.

Sources: 1980, 1983 and 1989 Risk Factor Prevalence Surveys; 1999–2000 AusDiab.

Figure 3.13: Proportion of people with high blood cholesterol, 1980 to 1999–2000



Notes

1. Age-standardised to the 1991 Australian population.
2. Capital cities only.
3. Includes people aged 25–64.

Sources: 1980, 1983, 1989 Risk Factor Prevalence Surveys; 1999–2000 AusDiab.

Special population groups

In 1989 there were no strong associations between cholesterol levels and socioeconomic status. However, very high blood cholesterol (≥ 6.5 mmol/L) was more common among unemployed women (25–64 years) than among women in full-time employment. Among men aged 25–64 years, those living alone or previously married had around one-and-a-half times higher rate of very high blood cholesterol (≥ 6.5 mmol/L) than those with partners or dependants.

There are no national data on blood cholesterol levels across urban, rural and remote areas of Australia.

Aboriginal and Torres Strait Islander people

There are no national data on blood cholesterol levels among Aboriginal and Torres Strait Islander people. A New South Wales survey on cardiovascular risk factors in 1987–88 showed that a greater proportion of Indigenous women in Wilcannia had cholesterol levels above 6.5 mmol/L compared with other Australian women. However, other studies have shown no difference in cholesterol levels between Indigenous Australians and other Australians.

Main data sources

1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) (International Diabetes Institute & Commonwealth Department of Health and Aged Care).

1980, 1983, 1989 Risk Factor Prevalence Surveys (National Heart Foundation).

References and further reading

AIHW (Australian Institute of Health and Welfare) 2001. Heart, stroke and vascular diseases—Australian facts 2001. Cardiovascular Disease Series No. 14. AIHW Cat. No. CVD 13. Canberra: AIHW, National Heart Foundation of Australia & National Stroke Foundation of Australia.

AIHW: Britt H, Miller G, Knox S et al. 2001. General practice activity in Australia 2000–01. General Practice Series No. 8. AIHW Cat. No. GEP 8. Canberra: AIHW.

AIHW: Mathers C, Vos T & Stevenson C 1999. The burden of disease and injury in Australia. AIHW Cat. No. PHE 17. Canberra: AIHW.

Barter P 2001. Lipid management guidelines—2001. Medical Journal of Australia 175 supp: s57–s88.

Bouchard C, Malina RM & Perusse L 1997. Genetics of fitness and physical performance. Champaign, Il: Human Kinetics.

Diabetes Australia 2002. Part 7: Evidence based guideline for management of lipid abnormalities in Type 2 diabetes, December 2001. Viewed 16 April 2002, <http://www.diabetesaustralia.com.au/submission-documents.htm>.

DHAC & AIHW (Commonwealth Department of Health and Aged Care & Australian Institute of Health and Welfare) 1999. National Health Priority Areas report: cardiovascular health 1998. AIHW Cat. No. PHE 9. Canberra: DHAC & AIHW.

Dunstan D, Zimmet P, Welborn T et al. 2001. Diabesity and associated disorders in Australia 2000. The accelerating epidemic. Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Melbourne: International Diabetes Institute.

Forge BHR 1999. Cholesterol in perspective. Medical Journal of Australia 170:385–90.

National Heart Foundation of Australia 2002. Cholesterol and fat, May 1995. Viewed 26 September 2001,<http://www.heartfoundation.com.au/docs/hhd3.htm>.

Rewers M & Hamman RF 1995. Risk factors for non-insulin dependent diabetes. In: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health. Diabetes in America. 2nd edn. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, 165–78.

Taskinen M-R 1999. Lipid metabolism in diabetes. In: Turtle J, Kaneko T & Osato S (eds). Diabetes in the new millennium. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney.

Williams G & Pickup J 1999. Handbook of diabetes. 2nd edn. Oxford: Blackwell Science.