

# Diabetes: Australian Facts

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National Centre for Monitoring Diabetes

November 2002



**2002**

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# Preface

Diabetes affects the health of many Australians. The disease may cause a range of complications that lead to disabilities, reduced quality of life and shortened life expectancy. As well as the personal health costs, the disease inflicts a large public health burden.

It is estimated that around a million Australians (7.5% of Australians aged 25 years or over) have diabetes and the number is expected to increase over the coming decades. Rates of Type 2 diabetes in some Aboriginal and Torres Strait Islander communities may be among the highest in the world.

Factors such as ageing and genetic predisposition increase the risk of developing Type 2 diabetes, which is the most common form of diabetes. However, prevention of Type 2 diabetes is possible, as lifestyle factors, such as obesity and physical inactivity, contribute to the development of the disease.

*Diabetes: Australian Facts 2002* is the first report by the National Centre for Monitoring Diabetes to present available data across the spectrum of the disease: its levels in the population, the factors that contribute to it, and treatment and preventive programs. Importantly, the information presented aims to represent the challenges posed to the health system by the growing diabetes epidemic. It informs the interested public, academics, health professionals and policy makers about:

- Type 1 diabetes, Type 2 diabetes and gestational diabetes, and associated health burden;
- risk factors for the disease and its complications;
- complications of diabetes; and
- management and care.

A variety of data sources have been used in this report. However, at times limitations in the data have restricted the content and coverage of issues that are essential to a comprehensive understanding of diabetes. Therefore, this report also highlights the need for further investigation and data development in many areas.

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# Highlights

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## **Diabetes is one of the leading threats to the health of Australians...**

- It is estimated that almost a million Australians (940,000 people aged 25 years or over) have diabetes, yet half of these people are unaware of it—there is evidence that for every case of diabetes there is an undetected case.
- In 2000, diabetes was the underlying cause of 3,006 deaths (2.3% of all deaths) and an associated cause in a further 7,124 deaths. Diabetes is twice as likely to be regarded as an associated cause of death rather than the underlying cause. There were 10,130 deaths (7.9% of all deaths) with diabetes as either the underlying cause of death or as an associated cause.
- In terms of premature deaths, diabetes was estimated to be responsible for almost 70,000 years of life lost in 1996—5.3% of the estimated years of life lost by all causes in Australia that year.

## **whether it be Type 1 diabetes...**

- Type 1 diabetes accounts for around 10–15% of people with diabetes.
- Based on self-reported information, in 1995 there were about 39,400 people of all ages with Type 1 diabetes in Australia (220 per 100,000 population). In persons aged 25 years or over, the prevalence of Type 1 diabetes was estimated at 298 per 100,000 for 1999–2000. This corresponds to 37,000 people aged 25 years or over.
- Incidence rates are around 19 per 100,000 population for both males and females aged 0–14 years, based on 743 new cases diagnosed in 2000.

## **Type 2 diabetes...**

- Type 2 diabetes represents about 85–90% of cases of diabetes. In 1999–2000, it is estimated that 7.2% of Australians aged 25 or over had Type 2 diabetes. This represents more than 850,000 Australians 25 years or over.

## **or gestational diabetes.**

- The incidence of gestational diabetes is estimated to be around 5%. The incidence rates may be as high as 20% in Aboriginal and Torres Strait Islander women and in women from high-risk populations such as from India, Asia and the Pacific Islands.

## **The rates of diabetes in the Australian community are increasing...**

- The number of adults with the condition has risen significantly since 1981.



### **and the epidemic is fed mostly by Type 2 diabetes which is potentially preventable.**

- Lifestyle factors such as obesity, physical inactivity, high blood pressure and poor diet are major modifiable risk factors for development of the disease or its complications.
- In 1999–2000 over seven million Australians aged 25 or over (60%) were overweight or obese. These people were at increased risk of developing Type 2 diabetes.
- In 2000, 44% of Australians aged 18–75 years (around 5.8 million people) did not undertake physical activity at high enough levels to get health benefits. Almost 15% of people did no leisure time physical activity at all, increasing their risk of developing Type 2 diabetes.

### **People with diabetes are more prone to certain problems and diseases...**

- People with diabetes are two to four times more likely to develop cardiovascular disease. In 1999–2000, 12% of Australians aged 25 or over with diabetes had had a heart attack and 9% had had a stroke. These proportions were much greater than among people without diabetes (3% and 2%, respectively).
- Diabetic retinopathy is the most common cause of blindness in people aged 30–69 years. In 1999–2000, 15.4% of people with diabetes had retinopathy. In 1995, 9.9% of people who reported diabetes also reported cataracts, 3.2% reported glaucoma and 4.9% reported blindness. This was more than six times the rate of cataracts, four times the rate of glaucoma and five times the rate of blindness reported among people without diabetes.
- In 1995, 6.1% of people who reported diabetes also reported having kidney disease, more than four times the rate among persons without diabetes. In 1999–2000, 11.2% of Australians aged 25 or over with self-reported diabetes reported being treated for or suffering from kidney disease. In 2000, diabetes was the second most common cause of kidney disease among patients receiving dialysis or a kidney transplant, accounting for more than one in five (22%) new patients.
- In 1999–2000, 10.3% of males and 9.4% of females with diabetes had neuropathy. Also, 30.2% of men with self-reported diabetes reported suffering from or receiving treatment for impotence.
- Among people with diabetes, 19.4% were at risk of foot ulcer in 1999–2000. Diabetes complications may also result in amputation: 2.1% of people with self-reported diabetes in 1995 had limbs absent. This was more than four times the rate reported among persons without diabetes, despite the higher mortality of amputees with diabetes.
- People with Type 2 diabetes are about three times more likely to have destructive periodontal disease than those without diabetes.
- Caesarean delivery is three to four times more frequent in pregnancies involving diabetes.

### **resulting in disability.**

- In 1998, almost 64,000 Australians had a disability caused mainly by diabetes.
- Diabetes and its complications were responsible for more than 53,000 years of equivalent 'healthy' life lost to disability (4.6% of all years of life lost to disability) in 1996.

### **Diabetes consumes substantial health resources.**

- The direct health system cost of diabetes in 1993–94 was estimated at \$372 million. When the complications of diabetes were taken into account, the total direct health system costs rose to around \$681 million.
- In 2000–01, general practitioners managed diabetes problems (excluding gestational diabetes) at a rate of 2.8 per 100 encounters, representing 1.9% of all problems. This equates to almost 2.9 million consultations for diabetes each year and makes diabetes the seventh most common problem managed in general practice.
- In March 2002, 526,631 people with diabetes were registered for National Diabetes Services Scheme (NDSS) benefits; 169,585 (32%) of these required insulin. The NDSS distributed over 2.2 million packets of test strips and almost 430,000 boxes of syringes and pen needles in 2000–01.
- In 1999–00, diabetes as a principal or additional diagnosis accounted for 336,976 hospitalisations (5.7% of all hospital separations), with an average length of stay of 7.0 days compared with 3.6 days for people without diabetes.
- In 1998, the cost of drugs, to patients and the government, to treat diabetes was \$119 million; that is, 4% of government and patient costs for all prescription drugs listed in the Pharmaceutical Benefits Scheme.
- In 1999–00, there were 494,611 diabetes patients identified in the Medicare population (3.4%). Of these patients, 27.0% had two glycosylated haemoglobin tests in 1999–00 (that is, one test in each 6-month period), 18.1% had a microalbumin test in 1999–00, 62.9% had a lipid test in 1999–00 and 70.3% had an eye examination between 1998–99 and 1999–00.



### **Certain Australians are at greater risk of diabetes.**

- The prevalence of Type 2 diabetes is considerably higher among Aboriginal and Torres Strait Islander peoples than for the whole Australian population. It may be as high as 30% in some Aboriginal communities, compared with 7% in the general population.
- During 1998–00, Aboriginal and Torres Strait Islander males died from diabetes as the underlying cause at more than seven times the rate of non-Indigenous males, based on available data. The difference in death rates is even larger for females—Aboriginal and Torres Strait Islander females were more than 14 times as likely to die from diabetes as the underlying cause as non-Indigenous females.
- People from the most socioeconomically disadvantaged areas are more likely to have Type 2 diabetes. Males in the lowest socioeconomic group were almost twice as likely to report Type 2 diabetes than those in the highest socioeconomic group. For females, the rate in the lowest socioeconomic group is 2.5 times that in the highest socioeconomic group.
- In 1995, rates of Type 2 diabetes among people from culturally and linguistically diverse backgrounds were more than 40% higher than rates in the general population.
- During 2000, males from culturally and linguistically diverse backgrounds were almost 30% more likely to die from diabetes as the underlying cause than other males. The difference in death rates was larger for females—females from culturally and linguistically diverse backgrounds were 50% more likely to die from diabetes as the underlying cause than other females.





# Introduction

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## diabetes australian facts 2002

Australian Institute of Health and Welfare

## Background

Diabetes has become one of the leading threats to the health of Australians. There is now a growing epidemic due to the recent great rise of Type 2 diabetes, which contributes 85–90% of cases of diabetes. It is estimated that almost a million Australians have diabetes, yet half of these people are unaware of it. Among some Aboriginal and Torres Strait Islander communities as many as one-third of the community have diabetes. It is a costly disease, associated with substantial morbidity and mortality, especially if undetected or poorly controlled. Complications can be life-threatening and include heart disease, stroke, kidney failure, blindness and lower limb amputation.

The rates of diabetes in the Australian community are increasing. Analysis of estimates from a number of surveys has led Dunstan et al. (2001) to conclude that the number of adults with diabetes has trebled since 1981. In an attempt to contain this growing epidemic, in 1996 the Australian Health Ministers agreed that diabetes should be a National Health Priority Area (NHPA). The NHPA initiative focuses public attention on diseases that present a significant health burden, where there is a potential for health gain through prevention and treatment programs.

Type 1 and Type 2 diabetes share many of the same complications. However, unlike Type 1 diabetes, Type 2 diabetes is potentially preventable. Although the disease occurs more commonly in people with a family history of diabetes, its onset is linked to lifestyle factors such as overweight, obesity and physical inactivity. Such risk factors are becoming increasingly common and, together with the ageing of the Australian population, are contributing to the rise of diabetes.

### Purpose and structure of this report

This report was compiled by the National Centre for Monitoring Diabetes, at the Australian Institute of Health and Welfare. It aims to provide health professionals, policy makers, academics and other interested readers with a concise summary of the latest available data and trends relating to diabetes in Australia. It is not designed to be a source of personal medical advice.

This is the Centre's first report to produce data on a broad scope, from population levels of the disease through prevention and risk factors to treatment and health services. However, there is a lack of good quality national data in many areas. This limits a full understanding of the true impact of the disease on Australian society and has also influenced the structure and content of this report. For example, current quality data measuring the human and financial costs of diabetes are not available. Nor have there been any comprehensive Australian studies examining the psychosocial effect of the disease on people with diabetes and their carers.

The report is intended to complement information produced in earlier reports by the Australian Institute of Health and Welfare: *Heart, Stroke and Vascular Diseases—Australian Facts 2001* and a 1999 version of the same report (AIHW 2001; AIHW 1999). As such, the structure of these reports is similar; each topic is presented as a stand-alone information sheet and these are assembled into chapters.

This introduction describes diabetes and its main forms, summarises its overall level and impact, and outlines national action to combat the disease. Chapter 2 describes the main types of diabetes and Chapter 3 presents information on risk factors for diabetes, together with risk factors for complications of the disease. The major complications of diabetes are included in Chapter 4 and Chapter 5 presents available data on the management and care of diabetes. Chapter 6 describes the impact of diabetes, including information on disability, mortality and economic costs. Each of the chapters includes a list of publications for further reading.

An outline of methods, data sources and their limitations is included at the back of this report. Some epidemiological concepts are also described in that section and in the glossary.

### What is diabetes?

Diabetes is a long-term (chronic) condition in which blood glucose levels become too high because the

body produces little or no insulin, or cannot use insulin properly. Insulin is a hormone produced by the pancreas that helps the body to use glucose. Diabetes can have both short-term and long-term effects, the latter through damage to various parts of the body, especially the heart and blood vessels, eyes, kidneys and nerves. Diabetes also contributes to many pregnancy-related complications for the mother and the baby, both before and after birth.

Because the common feature of diabetes is high blood glucose, it is often mistakenly thought to be a single disease. There are actually several types of diabetes, with different causal mechanisms. The three main types of diabetes are Type 1, Type 2 and gestational diabetes.

**Type 1 diabetes** is marked by a total or near-total lack of insulin. It results from the body destroying its own insulin-producing cells in the pancreas. People with this form of diabetes require daily insulin therapy to survive. It is the most common cause of childhood diabetes and accounts for 10–15% of all people with diabetes. Surveys indicate that around 0.2–0.3% of the Australian population have Type 1 diabetes.

**Type 2 diabetes** is marked by reduced levels of insulin, or the inability of the body to use insulin properly (insulin resistance). The disease is most common among people aged 40 years and over, and accounts for 85–90% of all people with diabetes. A 1999–2000 survey estimated that more than 7% of Australians aged 25 years or over have Type 2 diabetes. Many people with this form of diabetes eventually need insulin therapy to control their blood glucose levels.

**Gestational diabetes** occurs during pregnancy in about 3–8% of females not previously diagnosed with diabetes. Screening tests for gestational diabetes are usually performed around the 24th–28th weeks of pregnancy. It is a temporary form of diabetes and usually disappears after the baby is born. However, it is a marker of increased risk of developing Type 2 diabetes later in life.

There are other, less common, causes of diabetes that are not covered in this report. They include those in which a disease, drug or genetic defect causes the onset of diabetes. These types of diabetes have been estimated to account for 1–2% of all diagnosed cases (CDC 1998).

## Scale and impact of diabetes

Diabetes may result in disability for many people, particularly those who suffer complications. The disease also significantly shortens life expectancy. Due to the early onset of Type 1 diabetes (50% of cases before age 18) complications are evident at an earlier age than Type 2 diabetes, creating a greater burden on individuals and their families. The management of diabetes requires dramatic modifications to lifestyle. These may be through adherence to a strict diet, timing of meals, blood glucose monitoring and medication use. The disease also affects quality of life and psychological health, although there are limited data to assess its impact.

A survey conducted in 1999–2000 revealed approximately 940,000 Australians aged 25 or over with either Type 1 or Type 2 diabetes. This survey, the Australian Diabetes, Obesity and Lifestyle Study (AusDiab), collected information on the prevalence of diabetes in Australia measured objectively. It corroborated earlier evidence that for every case of diabetes there exists an undiagnosed Type 2 case (Guest et al. 1992).

Figure 1.1 provides estimates of the number of adults with diabetes in Australia over the last two decades. This information is an adaptation of similar information presented in studies by McCarty et al. (1996) and Dunstan et al. (2001).

These data have been compiled from a number of surveys with different methodologies. For instance, the estimate of population prevalence from the 1981 Busselton Study is based on a sample selected from Busselton in Western Australia and does not include

Indigenous Australians. The 1983 Risk Factor Prevalence Study took place in seven capital cities and Newcastle; it did not sample any rural or remote areas. The method of diabetes detection also varied between the studies. Estimates from the surveys that were based on self-reports of diabetes have been adjusted for undiagnosed cases, based on evidence from Guest et al. (1992) and Dunstan et al. (2001). Although not strictly comparable, these data do suggest a rise in the number of people with diabetes in Australia, leading to the estimate by Dunstan et al. of a trebling in the number of adults with diabetes in the two decades to 2000.

### National action to combat the disease

In recent years the growing effects of diabetes have been well recognised. Commonwealth, State and Territory governments have been working together to identify ways to prevent and manage the disease.

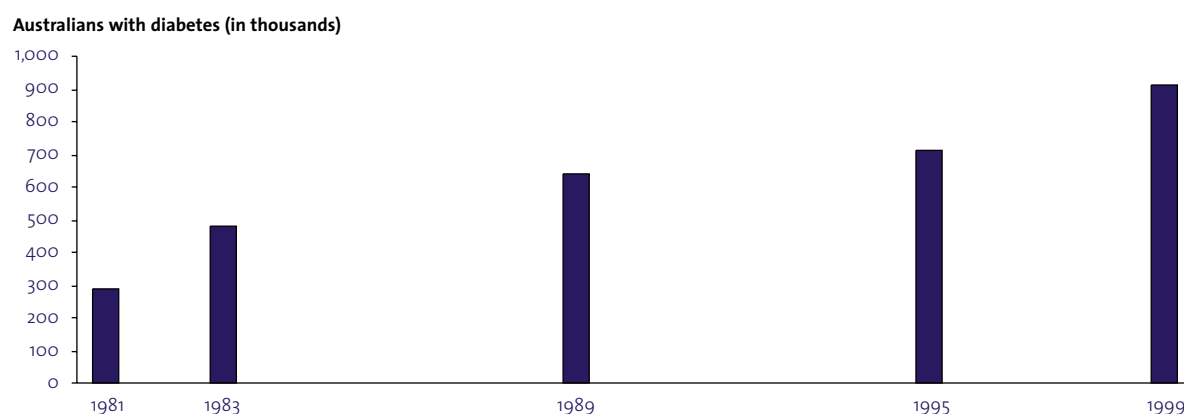
In 1996 diabetes became the fifth NHPA. The NHPA initiative focuses national attention and effort on health conditions which cause the greatest burden and where the greatest health gains can be achieved. Subsequently, a set of priority indicators covering

prevention, screening and early intervention, treatment and management of the condition was developed (DHAC & AIHW 1999). These indicators, which guide ongoing monitoring of the disease, are reported in the appendix.

Following the inclusion of diabetes in the NHPA initiative, the Commonwealth, State and Territory governments agreed to the National Diabetes Strategy 2000–2004 (DoHA 2001). The strategy covers diabetes prevention and management, helping governments and service providers to identify key areas for action to improve the health of Australians with, or at risk of, diabetes. It aims to:

- ensure appropriate attention is given to primary prevention, including risk reduction, effective high-quality management of diabetes and research;
- establish an effective partnership between governments, health care professionals, non-government organisations, consumers and carers; and
- build on experience and successes to date.

**Figure 1.1: Diabetes in Australians aged 25 years and over, 1981–1999**



Note: Estimates based on self-reports (1983, 1989, 1995) have been adjusted for undiagnosed cases of diabetes based on the assumption that for every diagnosed case there exists an undiagnosed case (Guest et al. 1992; Dunstan et al. 2001).

Sources: Adapted from Dunstan et al. 2001, using data from 1981 Busselton Study, Western Australia; 1983 National Heart Foundation Risk Factor Prevalence Study; 1989 & 1995 National Health Survey (ABS); and 1999–2000 AusDiab.



## Prevention and management

Many of the Commonwealth, State and Territory programs described above are intended to help prevent the growing prevalence of Type 2 diabetes, in addition to building awareness of Type 1 and Type 2 complications. Some of the specific prevention initiatives in Australia addressing lifestyle risk factors include the Active Australia campaign, Eat Well Australia and Acting on Australia's Weight.

The SNAP (smoking, nutrition, alcohol, physical activity) framework is an initiative of the Joint Advisory Group on General Practice and Population Health. The framework encourages general practitioners to adopt an integrated approach to modify behavioural risk factors, focusing on smoking, nutrition, alcohol, and physical activity, and supports them in managing these risk factors with their patients.

There are also various programs aimed at reducing the burden of Type 1 diabetes. One example is the proposed establishment of a centre to develop and test a vaccine to prevent the development of Type 1 diabetes, a project jointly managed by the National Health and Medical Research Council (NHMRC) and the Juvenile Diabetes Research Foundation. Other examples include education campaigns and diet counselling.

A consortium led by Diabetes Australia is well-advanced in the preparation of national evidence based guidelines for Type 2 diabetes. The NHMRC have endorsed two of these (primary prevention and case detection and diagnosis). Others have been released as consultation drafts (diagnosis and management of hypertension, prevention and detection of macrovascular disease, identification and management of diabetic foot disease, and guidelines for the management of lipid abnormalities).

## National Integrated Diabetes Program

As part of the 2001–02 Federal Budget, the Government announced funding of \$43.4 million over 4 years to ensure a national approach to improving the prevention, earlier diagnosis and management of

people with diabetes. The National Integrated Diabetes Program consists of four components that will:

- provide incentives for general practice for earlier diagnosis and best practice management of people with diabetes;
- provide infrastructure and support for Divisions of General Practice to work with general practitioners and other health professionals to remove barriers to better care for people with diabetes;
- engage consumers with diabetes to enable appropriate self-care and support partnerships with health professionals; and
- support changes in the practices of health professionals.

More information on the National Integrated Diabetes Program can be found at <<http://www.health.gov.au/pq/diabetes>>.

## References and further reading

AIHW (Australian Institute of Health and Welfare) 1999. Heart, stroke and vascular diseases—Australian facts. Cardiovascular Disease Series No. 10. AIHW Cat. No. CVD 7. Canberra: AIHW & National Heart Foundation of Australia.

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.

CDC (Centers for Disease Control and Prevention) 1998. National diabetes fact sheet: national estimates and general information on diabetes in the United States. Atlanta, GA: CDC.

Colagiuri S, Colagiuri R & Ward J 1998. National Diabetes Strategy and implementation plan. Canberra: Diabetes Australia.

DoHA (Department of Health and Ageing) 2001. National Diabetes Strategy 2000–2004. Viewed 28 November 2001, <<http://www.health.gov.au/hsdd/nhpq/pubs/diabsyn/diab2000.htm>>.

DHAC & AIHW (Commonwealth Department of Health and Aged Care & Australian Institute of Health and Welfare) 1999. National Health Priority Areas report: diabetes mellitus 1998. AIHW Cat. No. PHW 10. Canberra: DHAC & AIHW.

Dunstan D, Zimmet P, Welborn T et al. 2001. Diabetes 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 Europeans of south-eastern Australia. *Diabetes Research in Clinical Practice* 15:227–35.

McCarty D, Zimmet P, Dalton A, Segal L & Welborn T 1996. The rise and rise of diabetes in Australia, 1996: a review of statistics, trends and costs. Canberra: International Diabetes Institute & Diabetes Australia.

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.



# Main types of diabetes

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# 2

Type 1 diabetes

Type 2 diabetes

Gestational diabetes

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## Type 1 diabetes

In Type 1 diabetes the body's immune system destroys the insulin-producing beta cells in the pancreas, resulting in a failure of insulin production. The destruction of beta cells usually occurs in early life but many cases occur in adults as well. Type 1 diabetes has previously been known as 'juvenile-onset diabetes' or 'insulin-dependent diabetes mellitus' (IDDM).

Type 1 diabetes accounts for around 10–15% of people with diabetes. It is one of the most serious and common chronic diseases of childhood, with about half of the people with Type 1 diabetes developing the disease before age 18. About 98% of childhood diabetes is Type 1 (Silink 1994). Type 1 diabetes is thought to be caused by a combination of genetic and environmental factors. Several genes associated with a greater risk for diabetes have been identified—about half of the lifetime risk of Type 1 diabetes is thought to be genetic. Environmental factors, such as viruses, diet or chemicals, may also trigger the disease in genetically predisposed people. No modifiable risk factors have been clearly identified for the disease.

A person with Type 1 diabetes requires insulin injections for survival. Without insulin, glucose cannot be processed in the tissues and builds up in the bloodstream, causing high blood glucose levels (hyperglycaemia). As the body has no means of converting glucose into energy it instead breaks down fat or muscle for energy. The chemicals produced from this process are called ketones. When ketones build up in the body they can lead to nausea and vomiting—symptoms that are often present before a diagnosis of Type 1 diabetes. This complication is known as ketoacidosis.

To maintain normal blood glucose levels, a person with Type 1 diabetes needs to carefully balance food intake, physical activity and insulin dose. If poorly balanced, hyperglycaemia or hypoglycaemia may occur. Hypoglycaemia occurs when blood glucose drops too low and may result from too much insulin, strenuous exercise or insufficient food for the amount of insulin taken. Symptoms include shakiness and confusion. In undiagnosed cases a person may suffer seizures or lose consciousness. Hypoglycaemia is more common

in persons with Type 1 diabetes than in those with Type 2 diabetes. People with Type 1 diabetes are at risk of the full range of diabetes complications. These include blindness, kidney failure, peripheral vascular disease and foot problems.

Type 1 diabetes can have an emotional toll on the patient, their family and carers. A supportive environment, including access to counselling and education, helps patients and their carers to better manage the condition.

### Detection

The signs and symptoms of Type 1 diabetes are often rapid in onset (days to weeks) and include:

- excessive thirst
- frequent urination
- excessive hunger
- nausea and vomiting
- weight loss
- blurred vision.

In childhood and adolescent/young adult cases very high blood glucose levels, often with ketone bodies in the urine, are sufficient to establish diagnosis. An oral glucose tolerance test (described in Box 2.1, page 13, and commonly used in diagnosing Type 2 diabetes) is neither necessary nor appropriate to diagnose Type 1 diabetes in the vast majority of cases (WHO 1999).

### Risk factors

No modifiable risk factors have been clearly identified for Type 1 diabetes. People who have an immediate relative with the disease are at greater risk of developing Type 1 diabetes, although approximately 80% of people with Type 1 diabetes have no family history of the disease (Dorman et al. 1995). The disease is also more prevalent in Caucasian children. Some viruses may trigger the body's immune response,

causing the destruction of the insulin-producing beta cells. They include rubella virus, Coxsackie B virus, Epstein-Barr virus (glandular fever) and mumps virus. Maternal viral infection during pregnancy is also a risk factor for the development of Type 1 diabetes in childhood—12–20% of individuals infected in utero with rubella usually develop Type 1 diabetes within 5–20 years (Yoon et al. 1999).

## How many Australians have Type 1 diabetes?

### Prevalence

Accurate epidemiological data on Type 1 diabetes in Australia are limited. Surveys undertaken to estimate diabetes prevalence in Australia have used a variety of methodologies, including differing diagnostic criteria. As a result, direct comparisons cannot usually be made between such studies.

Based on self-reported information in the 1995 National Health Survey, the prevalence of Type 1 diabetes in Australia is about 39,400 people of all ages (220 per 100,000 population).

The National Heart Foundation Risk Factor Prevalence Study conducted in 1983 provided an estimate of prevalence of Type 1 diabetes in certain age groups based on plasma glucose levels. Among people aged between 25 and 64 years, the prevalence rate for Type 1 diabetes was 317 per 100,000 (Welborn et al. 1989).

The Australian Diabetes, Obesity and Lifestyle Study collected diabetes prevalence information using an oral glucose tolerance test in persons aged 25 years or older only. The age-standardised prevalence of Type 1 diabetes was estimated at 298 per 100,000 for 1999–2000. This corresponds to 37,000 people aged 25 years or over.

Due to the relatively low prevalence of Type 1 diabetes, there is no reliable information on differences in its occurrence among socioeconomic groups, or in urban, rural and remote areas.

## Aboriginal and Torres Strait Islander people

Type 1 diabetes is generally thought to be rare among Aboriginal and Torres Strait Islander people. De Courten et al. (1998) examined the limited information on the incidence of Type 1 diabetes among Aboriginal and Torres Strait Islander children. Very few cases were reported among the surveys cited; however, their results are inconclusive. Moreover, de Courten et al. suggest that some cases of Type 1 diabetes are subject to misclassification. Conversely, a study by Verge et al. (1994) found self-reported Type 1 diabetes in Aboriginal and Torres Strait Islander children at a rate comparable to that in the rest of the population.

Given the extremely high prevalence of Type 2 diabetes in Aboriginal and Torres Strait Islander people, de Courten et al. (1998) conclude that Type 1 diabetes will only account for 1–2% of all cases of diabetes among this group.

### Incidence

The National Diabetes Register collects information on insulin-treated diabetes in Australia. Coverage for 2000 is close to 100% among children with diabetes, who suffer predominantly from Type 1 diabetes. Incidence rates are around 19 per 100,000 population for both males and females aged 0–14 years (Table 2.1). There were 743 new cases diagnosed in 2000.

There are some previous estimates of the incidence of Type 1 diabetes based on surveys. These measures, in the 0–14 age group, range from 12.3 per 100,000 in 1983 (Glatthaar et al. 1988) to 14.9 per 100,000 in 1985–92 (Kelly et al. 1994). Craig et al. (2000) undertook an analysis of trends in the incidence of Type 1 diabetes in New South Wales over the period 1990–96, concluding that the incidence has increased by an average of 3.2% per year since 1990. When more national incidence data from the National Diabetes Register become available, a further measure of the change in the occurrence of the disease will be possible.

**Table 2.1:** Incidence of diabetes among 0–14-year-olds, Australia, 2000

Age at diagnosis	Males		Females	
	Number	Rate per 100,000	Number	Rate per 100,000
0–4 years	84	13.0	75	12.2
5–9 years	138	20.2	133	20.5
10–14 years	165	24.3	148	22.9
<b>Total</b>	<b>387</b>	<b>19.2</b>	<b>356</b>	<b>18.6</b>

Source: National Diabetes Register, AIHW, 2001.

## International comparisons

The WHO DiaMond project was established in 1990 to survey the incidence of Type 1 diabetes among children aged 0–14 years worldwide. Using standardised incidence registries in 100 centres spanning 50 countries, the study sample covered 4.5% of the world's population in that age group.

Over the period 1990–94, there was a 350-fold variation in the incidence of Type 1 diabetes worldwide, from 0.1 per 100,000 per year in China and Venezuela to 36.5 per 100,000 per year in Finland (Karvonen et al. 2000). One-third of the populations had an intermediate incidence rate range (5–9.9 per 100,000 per year). In general, China and South America had the lowest incidence (< 1 per 100,000 per year), while Finland, Sweden, Norway, Portugal, United Kingdom, Canada and New Zealand had a very high incidence ( $\geq 20$  per 100,000 per year). Australia came in at 14.5 per 100,000 per year, although more recent data estimate the incidence to be around 19 per 100,000 per year among Australian children (AIHW 2001). The reasons for such differences in disease rates among populations are not known but genetic factors making certain ethnic groups more prone to diabetes are probably involved.

The incidence of Type 1 diabetes appears to be increasing in almost all populations worldwide, with the increase being larger in those populations with a low incidence. However, it is not clear if this is a true increase in cases of the disease or simply the result of better detection.

## Main data sources

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).

1983 Risk Factor Prevalence Survey (National Heart Foundation).

National Diabetes Register, Australian Institute of Health and Welfare.

## References and further reading

AIHW (Australian Institute of Health and Welfare) 2001. National diabetes register statistical profile December 2000. Diabetes Series No. 2. AIHW Cat. No. CVD 18. Canberra: AIHW.

Akerblom H 1999. Pathogenesis of Type 1 diabetes: environmental factors. In: Turtle J, Kaneko T & Osato S (eds). Diabetes in the new millennium. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney.

Craig M, Howard N, Silink M & Chan A 2000. The rising incidence of childhood Type 1 diabetes in New South Wales, Australia. Journal of Pediatric Endocrinology and Metabolism 13:363–72.

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.

Dorman J, McCarthy B, O'Leary L & Koehler A 1995. Risk factors for 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.

Glatthaar C, Whittall D, Welborn T et al. 1988. Diabetes in Western Australian children: descriptive epidemiology. Medical Journal of Australia 148:117–23.

Karvonen M, Viik-Kajander M, Moltchanova E, Libman I, LaPorte R, Tuomilehto J 2000. Incidence of childhood Type 1 diabetes worldwide. Diabetes Care 23; 10:1516–26.

Kelly H, Russell M, Jones T & Byrne G 1994. Dramatic increase in incidence of insulin dependent diabetes mellitus in Western Australia. Medical Journal of Australia 161:426–9.

McCarty D, Zimmet P, Dalton A, Segal L & Welborn T 1996. The rise and rise of diabetes in Australia, 1996: a review of statistics, trends and costs. Canberra: Diabetes Australia.

Norris J, Beaty B, Klingensmith G et al. 1996. Lack of association between early exposure to cow's milk protein and beta-cell autoimmunity: Diabetes Autoimmunity Study in the Young. Journal of the American Medical Association 276:609–14.

Norris J & Scott F 1996. A meta-analysis of infant diet and insulin-dependent diabetes mellitus. Do biases play a role? Epidemiology 7:87–92.

Silink M 1994. Childhood diabetes and hypoglycaemia. In: Robertson M & Robertson D (eds). Practical paediatrics. 3rd edn. Melbourne: Churchill Livingstone.

Verge C, Silink M & Howard N 1994. The incidence of childhood IDDM in New South Wales, Australia. Diabetes Care 17(7):693–6.

Welborn T, Glatthaar C, Whittall D & Bennett S 1989. An estimate of diabetes prevalence from a national population sample: a male excess. Medical Journal of Australia 150:78–81.

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.

Yoon J-W, Kim A & Jun H-S 1999. Role of viruses in Type 1 diabetes. In: Turtle J, Kaneko T & Osato S (eds). Diabetes in the new millennium. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney.



## Type 2 diabetes

Type 2 diabetes is characterised by reduced levels of insulin and an inability of the body to use insulin properly (insulin resistance). Many people with Type 2 diabetes eventually need insulin therapy to control their blood glucose levels.

Type 2 diabetes is the most common form of diabetes, estimated to account for 85–90% of diabetes. The disease, previously known as non-insulin dependent diabetes, occurs more commonly in people who are aged over 40 years.

Both genetic and environmental factors contribute to the development of Type 2 diabetes. Non-modifiable risk factors for the disease include age, ethnicity and family history. Although increased age enhances susceptibility, the disease may even present in adolescence and childhood. Lifestyle factors such as excess weight, physical inactivity and poor diet are major modifiable risk factors for development of the disease.

A number of minority populations, including Indigenous Australians, Pacific Islanders, people of Chinese descent and those from the Indian subcontinent, are at higher risk. The influence of modernisation on diet and physical activity habits has been implicated in their increased risk. Other people at increased risk include women who have had gestational diabetes, and their children.

A growing body of evidence suggests that half of the people with Type 2 diabetes are not aware that they have the condition (Guest et al. 1992; McCarty et al. 1996; Dunstan et al. 2001). Early diagnosis of the condition is important as careful diabetes management can reduce morbidity from long-term complications. If diabetes is not managed well, damage to small and large blood vessels throughout the body may lead to complications such as blindness, kidney failure, cardiovascular disease, limb amputation and impotence.

### Detection

Type 2 diabetes usually has a relatively insidious onset. It may be detected during a routine medical examination or when a patient seeks medical care for other reasons. Common symptoms include:

- urinating more frequently (polyuria)
- increased thirst (polydipsia)
- feeling tired and lethargic
- blurred vision
- frequent infections
- unexplained weight loss.

The National Health and Medical Research Council has recently endorsed guidelines for the detection and diagnosis of Type 2 diabetes (Colagiuri et al. 2002). These encourage active case detection, particularly among people identified at high risk of the disease. Information about diagnostic tests for the disease is provided in Box 2.1.

### Risk factors

A number of risk factors are implicated in the development of Type 2 diabetes. These may act alone, but often act together in complex interplay. Consideration of combinations of risk factors is important as it may explain why some population subgroups have higher rates of diabetes than others. Table 2.2 outlines the risk factors for Type 2 diabetes.



**Box 2.1:** How is Type 2 diabetes detected?

*Diabetes can be detected using a variety of tests including a fasting blood glucose test, a random blood glucose test and the oral glucose tolerance test (OGTT). The test used may vary depending on whether it is performed for clinical or population survey purposes. All of the tests involve the measurement of glucose in the blood. This is expressed as the concentration of glucose in plasma (the fluid component of blood after blood cells are removed). A test may be performed in people with classical symptoms of diabetes, such as excessive thirst and urination. It is also used for screening for Type 2 diabetes among people identified at increased risk of the disease.*

*A random blood glucose test is a test applied to persons without specifying the need to fast; these persons may include individuals who have not fasted and who have.*

*Recent guidelines endorsed by the National Health and Medical Research Council recommend laboratory testing of a plasma glucose sample taken after fasting; however, a random sample may be used if fasting is impractical (Colagiuri et al. 2002). Diabetes is likely in people with a fasting plasma glucose concentration of at least 7.0 millimoles/litre (mmol/L), or more than 11.1 mmol/L based on a random sample. According to the guidelines, an OGTT should be performed if fasting results fall between 5.5 and 6.9 mmol/L or random results between 5.5 and 11.0 mmol/L.*

*In the OGTT a blood glucose measurement is taken after a period of fasting. An additional measurement is then taken two hours after consuming 75 g of glucose. The blood glucose levels should have returned to normal by this time, but they will remain elevated in a person with diabetes. The results of the OGTT should be interpreted according to the 1999 World Health Organization criteria (WHO 1999).*

*The guidelines state that two positive tests on separate days are necessary for diagnosis, with the exception of cases where plasma glucose is unequivocally elevated with obvious symptoms or signs.*

*Readers should consult Evidence Based Guidelines for Type 2 Diabetes: Case Detection and Diagnosis (Colagiuri et al. 2002) for full details.*

**Table 2.2:** Risk factors for Type 2 diabetes

Demographic	Genetic	Lifestyle and behavioural	Biomedical/Metabolic
Age	Ethnicity	Diet	Intra-uterine growth retardation
Urbanisation	Family history	Obesity (especially abdominal)	Previous gestational diabetes
		Physical inactivity	Impaired glucose metabolism
		Foetal nutrition	(i.e. impaired fasting glucose, impaired glucose tolerance)



## How many Australians have Type 2 diabetes?

The 1999–2000 Australian Diabetes, Obesity and Lifestyle Study estimated the prevalence of Type 2 diabetes in Australians aged 25 or over to be 7.2% (7.6% in males and 6.7% in females, see Table 2.3). This represents more than 850,000 Australians aged 25 years or over. The prevalence of Type 2 diabetes rises with age—a person aged 75 years or older is at least ten times more likely to have diabetes than someone aged 35–44 years.

Available evidence suggests that there has been a rise in prevalence between 1995 and 1999–2000 (refer to National Health Priority Areas indicator 1.1 (b) in Appendixes). Trend information is difficult to generate given variations in the criteria for reporting cases of diabetes; the surveys cited in the appendix include estimates of prevalence based on self-reported data and measured data.

Type 2 diabetes is more prevalent among people from lower socioeconomic backgrounds. In the lowest socioeconomic group (quintile 1) 3.6% of males and 4.3% of females reported having been diagnosed with Type 2 diabetes. A significantly lower proportion of people in the highest socioeconomic group (quintile 5) reported Type 2 diabetes: 2.0% of males and 1.7% of females (Figure 2.1).

## Aboriginal and Torres Strait Islander people

The prevalence of Type 2 diabetes is considerably higher among Aboriginal and Torres Strait Islander peoples than for the whole of the Australian population. Studies suggest that the prevalence may be as high as 30% in some Aboriginal communities (de Courten et al. 1998), compared with 7% in the general population. Unfortunately there are limited national data to accurately measure the extent of the disease among Aboriginal and Torres Strait Islander peoples.

The two most recent national surveys to investigate diabetes prevalence in Aboriginal and Torres Strait Islander peoples are based on self-reports of the disease. Given earlier evidence of the high number of undiagnosed cases of diabetes, these surveys are likely to underestimate the true prevalence of the disease. The results reported here include Type 1 and Type 2 diabetes; however, it is notable that around 98–99% of diabetes in Aboriginal and Torres Strait Islander peoples is thought to be Type 2 diabetes (de Courten et al. 1998).

According to the 1994 National Aboriginal and Torres Strait Islander Survey, 3.5% of males and 4.7% of females reported having diabetes. Corresponding rates after the age of 45 years were 17% and 23%. The 1995 National Health Survey's estimates of the prevalence

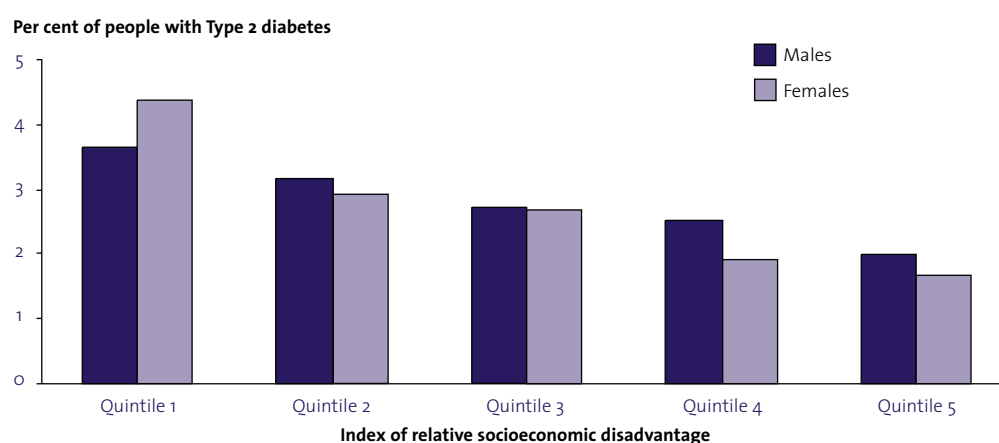
**Table 2.3:** Prevalence of Type 2 diabetes for persons aged 25 years or over, 1999–2000

Age group	Males	Females	Persons
		<b>Per cent</b>	
25–34 years	0.1	0.1	0.1
35–44 years	2.4	1.9	2.1
45–54 years	6.0	5.2	5.6
55–64 years	16.0	9.9	13.0
65–74 years	21.2	15.5	18.1
75+ years	20.9	24.4	23.0
<b>Total</b>	<b>7.6</b>	<b>6.7</b>	<b>7.2</b>

Note: Estimates are based on cases where the type of diabetes could be derived.

Source: 1999–2000 AusDiab.

**Figure 2.1:** Age-standardised prevalence of Type 2 diabetes by socioeconomic disadvantage of area and sex, Australians aged 18 years and over, 1995



Note: Quintile 1 is the most disadvantaged.

Source: 1995 National Health Survey.

of diabetes prevalence exclude persons living in remote areas of Australia. In some age groups diabetes was reported at rates more than eight times those for the non-Indigenous population (Table 2.4).

**Table 2.4:** Self-reported diabetes prevalence among Indigenous and non-Indigenous Australians by age, 1995

Age group	Indigenous (%)	Non-Indigenous (%)
15–24 years	1.7	0.4
25–44 years	7.3	0.9
45–54 years	23.9	2.9
55 years or over	17.3	7.3

Notes

1. Excludes persons living in remote areas.
2. Includes Type 1 and Type 2 diabetes.

Source: ABS 1999.

There are limited data comparing the rates of diabetic complications among Indigenous and non-Indigenous Australians. However, available data suggest higher rates of complications among Aboriginal and Torres Strait Islander peoples. Most remarkable are data from the Australia and New Zealand Dialysis and Transplant Registry; in 2000, 46% of Aboriginal and Torres Strait Islander patients had diabetic nephropathy compared with around 14% of non-Indigenous patients.

The basis of apparent ethnic differences in susceptibility to diabetes complications is unclear, but probably reflects a combination of genetic and environmental factors (de Courten et al. 1998).

### International comparisons

Over the past 20 years, the number of people diagnosed with diabetes worldwide has increased dramatically. This rise primarily reflects increasing prevalence of Type 2 diabetes in both developed and



developing countries. In 1998 the global prevalence of people with diabetes was estimated to be 150 million; this figure is projected to increase to 300 million people in 2025 (King et al. 1998).

### Trends

Estimates of Type 2 diabetes prevalence are difficult to compare between countries as data were collected in different years, using different sampling methods and diagnostic criteria. Nevertheless, epidemiological studies in different populations have highlighted the increasingly epidemic nature of Type 2 diabetes. The highest prevalence rates, ranging from 25% to 50%, have been reported in migrant or urbanised populations, such as the Pima Indians of Arizona, Pacific Islanders of Nauru, urban Indians of Fiji and Australian Aboriginal communities, that have experienced considerable lifestyle change (King & Rewers 1993). In contrast, very low prevalence rates of 1–2% are seen among rural populations in the least developed countries such as Tanzania and Cameroon. In general, these communities have traditional hunter-gatherer or agriculture-based lifestyles incorporating high levels of physical activity.

The increasing prevalence of Type 2 diabetes may be partially attributed to changing patterns in some risk factors over the last few decades. Available data indicate that environmental, social and behavioural changes (particularly adoption of a sedentary lifestyle, rising obesity and changing nutrition) combined with genetic susceptibility are closely associated with diabetes prevalence. With further urbanisation and increased longevity, the number of people with Type 2 diabetes worldwide is projected to increase exponentially (King et al. 1998).

The prevalence of Type 2 diabetes is also reported to be on the rise in children and adolescents. However, epidemiological data on the magnitude of this problem are limited, particularly in parts of the world where the disease is prevalent among adults. Studies of selected populations suggest that Type 2 diabetes among children is associated with obesity, physical inactivity, a family history of Type 2 diabetes, exposure to diabetes in utero, and signs of insulin resistance.

### Main data sources

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).

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

Australian and New Zealand Dialysis and Transplant Registry.

### References and further reading

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

ABS & AIHW (Australian Bureau of Statistics & Australian Institute of Health and Welfare) 2001. The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples. ABS Cat. No. 4704.0. AIHW Cat. No. IHW 6. Canberra: ABS & AIHW.

Colagiuri S, Zimmet P, Hepburn A & Colagiuri R 2002. Evidence based guidelines for Type 2 diabetes: case detection and diagnosis. Canberra: Diabetes Australia & National Health and Medical Research Council.

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.

Diabetes Control and Complications Trial Research Group 1993. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *New England Journal of Medicine* 329:977–86.

Dowse GK, Gareebo H & Zimmet PZ 1990. High prevalence of NIDDM and impaired glucose tolerance in Indian, Creole and Chinese Mauritians. *Diabetes* 30:390–6.

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

Federation of Ethnic Communities' Councils of Australia 1997. Dispelling the myth—a little bit of sugar. Canberra: Diabetes Australia.

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

King H, Aubert RE & Herman WH 1998. Global burden of diabetes, 1995–2025. Prevalence, numerical estimates, and projections. *Diabetes Care* 21(9):1414–31.

King H & Rewers M 1993. Global estimates for prevalence of diabetes mellitus and impaired glucose tolerance in adults. *Diabetes Care* 6(1):157–77.

Krans H 1999. Type 2 diabetes: overview and genetics. In: Turtle J, Kaneko T & Osato S (eds). *Diabetes in the new millennium*. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney.

McCarty DJ, Zimmet P, Dalton A, Segal L & Welborn TA 1996. The rise and rise of diabetes in Australia, 1996: a review of statistics, trends and costs. Canberra: International Diabetes Institute & Diabetes Australia.

New South Wales Health Department 1996. The principles of diabetes care and guidelines for the clinical management of diabetes mellitus in adults. North Sydney: NSW Health Department.

Royal Australian College of General Practitioners & Diabetes Australia 2001. Diabetes management in general practice. 7th edn, rev. Canberra: Diabetes Australia.

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.

## Gestational diabetes

Gestational diabetes is a form of diabetes that may develop during pregnancy. It involves glucose intolerance in pregnant women who have not previously been diagnosed with diabetes. Usually glucose metabolism returns to normal after the baby is born.

During pregnancy the placenta produces hormones that may block the normal action of insulin, a problem known as insulin resistance. As a result blood glucose levels become elevated because additional time is taken for the muscles to absorb glucose.

When gestational diabetes is not controlled, the mother's high blood glucose levels affect the baby. Glucose and other nutrients in the mother's blood pass through the placenta to the baby, affecting the baby's blood glucose levels. The baby's pancreas reacts to the high blood glucose levels making extra insulin. This extra insulin promotes disproportionate growth and body fat, resulting in macrosomia (large or 'fat' babies). Macrosomia is associated with complications of labour and delivery.

Other implications of gestational diabetes for both the mother and the foetus include hypertension, pre-term birth, pre-eclampsia, uterine bleeding, foetal distress, neonatal hypoglycaemia, respiratory distress and jaundice. Women who have had gestational diabetes are at increased risk of developing Type 2 diabetes: at least 10% will have diabetes mellitus 5 years after the birth of their child, and 50% will develop diabetes 25 years following the birth (Martin 1991; O'Sullivan 1991). Their babies are at increased risk of developing obesity and diabetes later in life. The complications experienced by women with gestational diabetes are explored in greater depth in the 'Complications in pregnancy' section of Chapter 4.

### Detection

The Australian Diabetes in Pregnancy Society recommends that all pregnant women undergo screening for gestational diabetes. Where resources are limited, or in areas of low incidence, selective screening based on risk factors may be appropriate

(Hoffman et al. 1998). Screening is normally done through a glucose challenge test, performed between 26 and 28 weeks of gestation. This test involves measuring a person's plasma glucose levels an hour after a glucose drink. If the plasma glucose is  $\geq 7.8$  millimoles/litre (mmol/L) after a 50 g glucose drink or  $\geq 8.0$  mmol/L if the drink was 75 g, this is considered a positive screen. After a positive screen, an oral glucose tolerance test (OGTT—see Box 2.1) should be performed to confirm the diagnosis, but note that the criteria for a diagnosis of gestational diabetes differ from those for other forms of diabetes. Venous plasma glucose levels  $\geq 5.5$  mmol/L at 0 hours and/or  $\geq 8.0$  mmol/L at 2 hours are the criteria for a diagnosis of gestational diabetes.

After giving birth, women who have had gestational diabetes should have their glucose levels tested to check whether diabetes is still present. The Australian Diabetes in Pregnancy Society currently recommends that a repeat OGTT be performed 6–8 weeks post-partum. To prevent the onset of Type 2 diabetes in the future they should be careful to maintain a healthy weight and ensure that subsequent screening of blood glucose levels occurs at least every couple of years.

### Risk factors for gestational diabetes

Pregnant women who are at higher risk of developing gestational diabetes include:

- those who are 25 years or older
- those with a history of glucose intolerance or previous gestational diabetes
- those from certain high-risk ethnic groups, such as Indigenous Australians, and people from the Indian subcontinent, the Pacific Islands, Asia or the Middle East
- women with a family history of diabetes
- women with a history of 'large for gestational age' babies

- those who are overweight or obese before their pregnancy.

It is important to note that gestational diabetes may occur in women who have no identifiable risk factors.

### How many Australian women are affected by gestational diabetes?

The Australasian Diabetes in Pregnancy Society estimates the incidence of gestational diabetes to be around 5% (ADIPS 2002). The incidence rates may be as high as 20% in Aboriginal and Torres Strait Islander women and women from high-risk populations such as from India, Asia and the Pacific Islands (Colagiuri et al. 1998).

The Australian Diabetes, Obesity and Lifestyle Study revealed that 3.6% of women who had been pregnant reported having been told that they had gestational diabetes.

### Aboriginal and Torres Strait Islander people

More research is needed into the incidence of gestational diabetes in Aboriginal and Torres Strait Islander women because reliable national data in this area are limited. However, available data suggest that Indigenous Australian women have a higher prevalence of gestational diabetes than non-Indigenous Australians. As mentioned above, the incidence rate in Aboriginal and Torres Strait Islander women may be as high as 20% compared with a rate of 3–5% in Caucasian women (Colagiuri et al. 1998).

Gestational diabetes is a risk factor for the development of Type 2 diabetes in the mother. Indigenous Australians who have had gestational diabetes have a higher rate of conversion to Type 2 diabetes, up to 5% per year, compared with the Caucasian conversion rate of around 2% per year (Colagiuri et al. 1998).

### Main data source

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

### References and further reading

ADIPS (Australasian Diabetes in Pregnancy Society) 2002. Consumer centre. Viewed 7 March 2002, <<http://www.adips.org/consumer.htm>>.

Colagiuri S, Colagiuri R & Ward J 1998. National diabetes strategy and implementation plan. Canberra: Diabetes Australia.

Hoffman L, Nolan C, Wilson J, Oats J & Simmons D 1998. Gestational diabetes mellitus—management guidelines. Medical Journal of Australia 169:93–7.

Martin FIR 1991. The diagnosis of gestational diabetes. Medical Journal of Australia 155:112.

O'Sullivan JB 1991. Diabetes after GDM. Diabetes 29(2):131–5.





# 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?

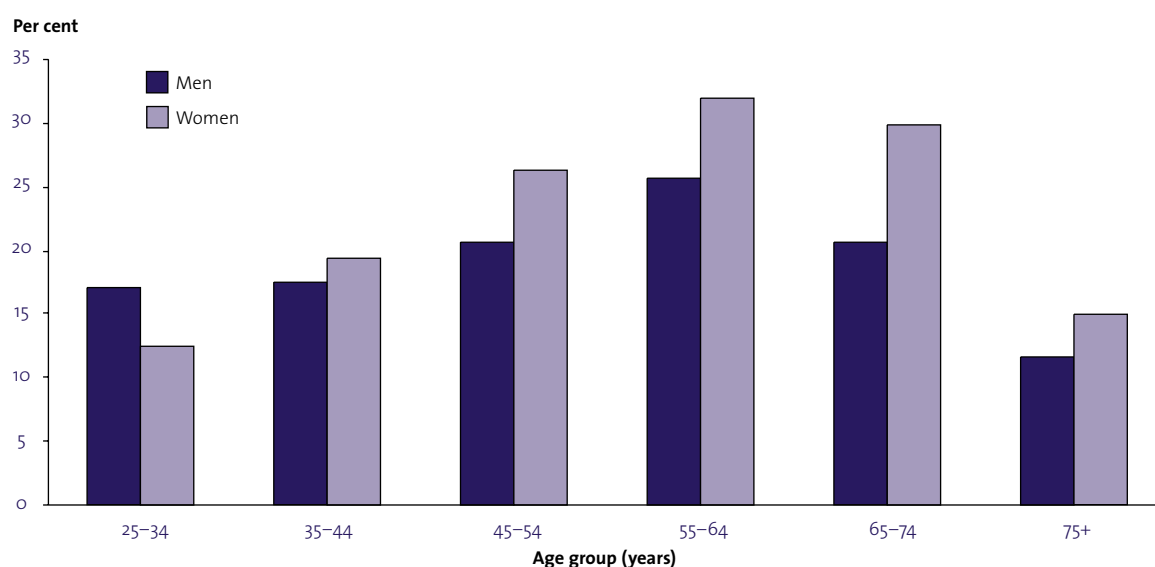
Estimates from the Australian Diabetes, Obesity and Lifestyle Study suggest that in 1999–2000 over seven million Australians aged 25 or over (60%) were overweight, as measured by BMI. Over two million of these (20% of the total population 25 or over) were obese.

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. Diabetes 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?

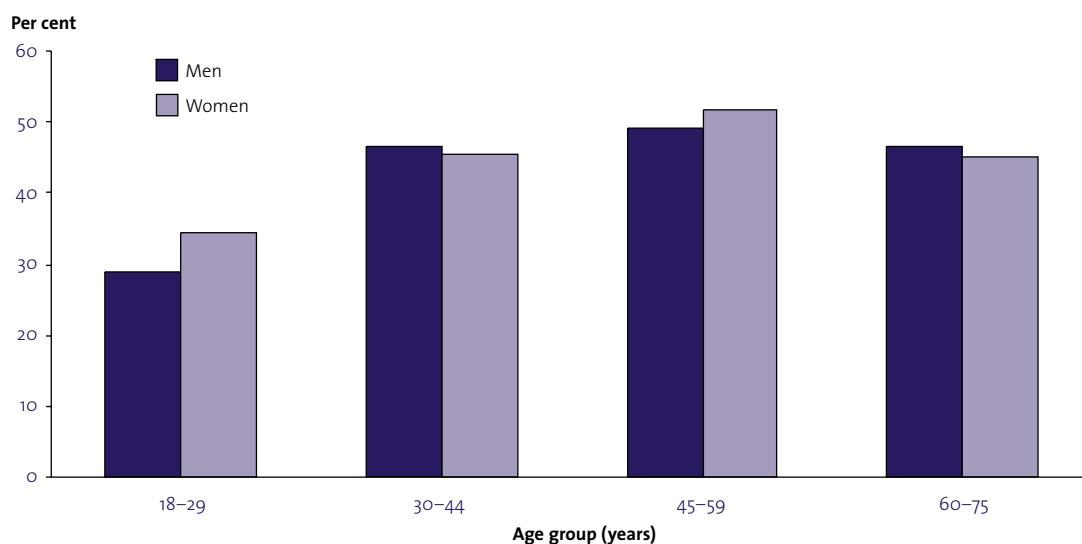
Physical inactivity is defined for this report as participating in less than 150 minutes of moderate-intensity activity per week. This figure is derived from the National Physical Activity Guidelines for Australians (DHAC 1999) which recommend that the accumulation of 30 minutes of moderate physical 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<sup>(a)</sup>

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. Diabetes 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 Europeans 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

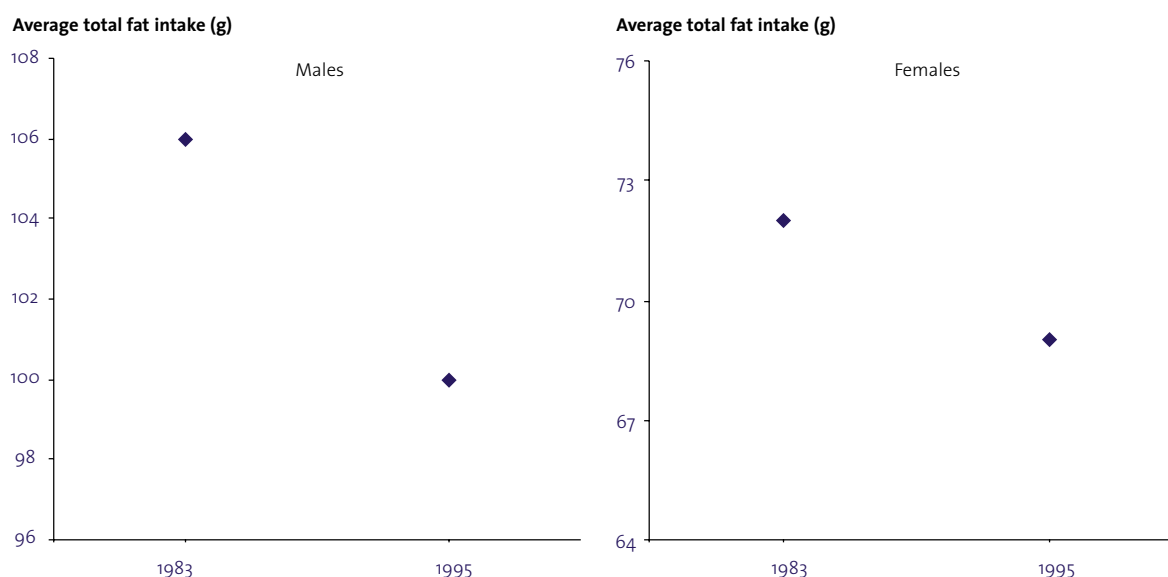
### Adults

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

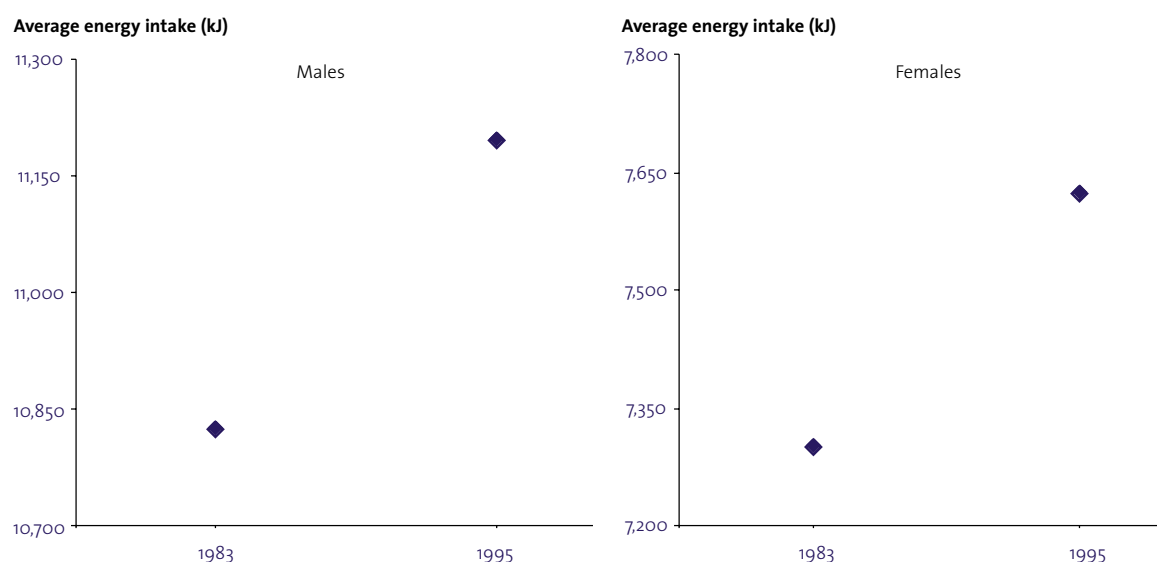


#### Notes

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

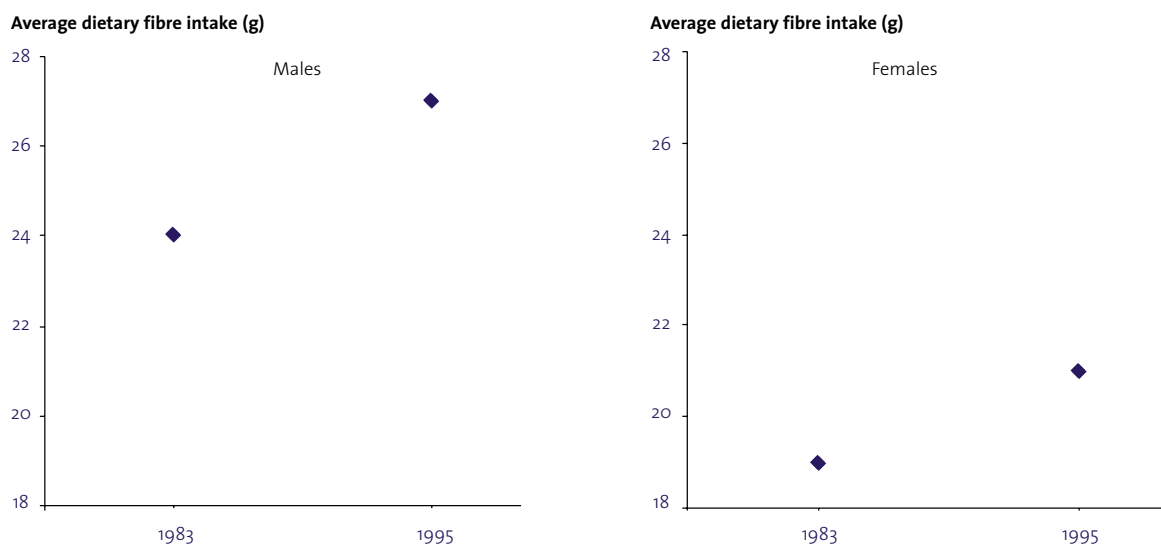
1. Different scales have been used for males and females due to their differing dietary requirements and intake.
2. Difference between estimated means for 1983 and 1995 is statistically significant at the 0.01 level.

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).

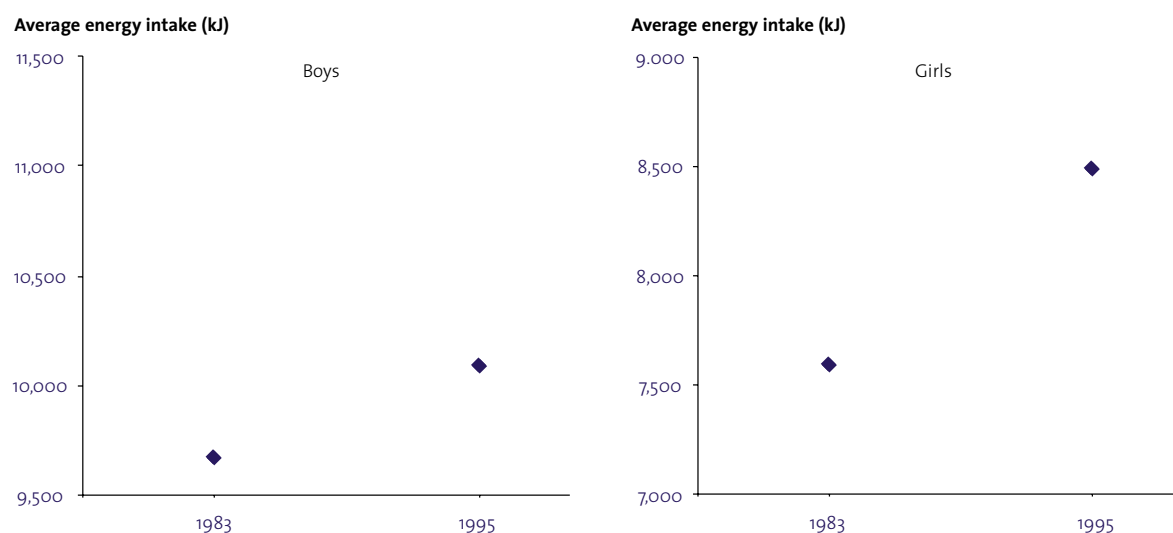
### Aboriginal and Torres Strait Islander people

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, Colaguiro S, Hepburn A, Holt P & Colaguiro 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.

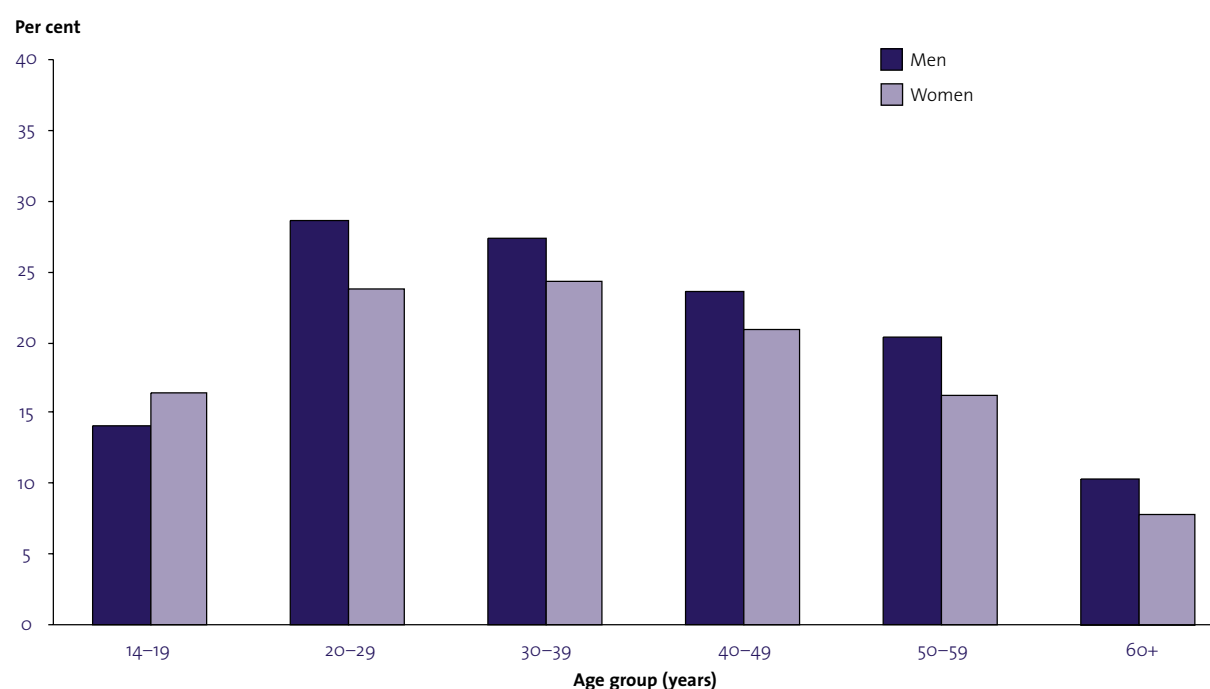
### How many Australians smoke?

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.

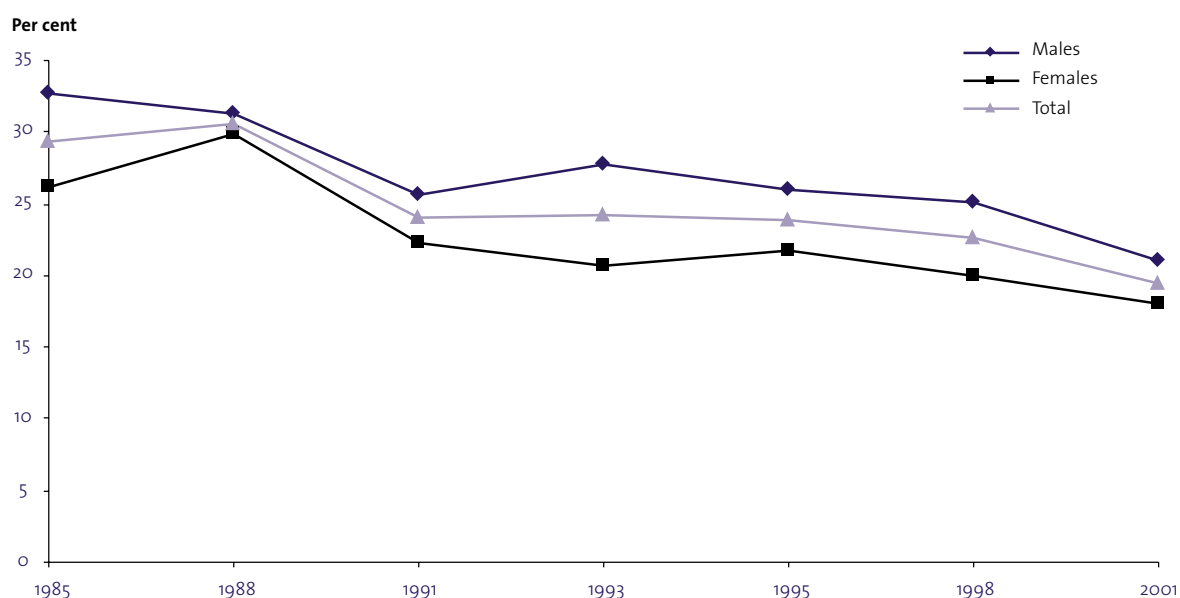
In 1998, a slightly higher percentage of individuals living in rural and remote areas reported that they were daily smokers (26%), compared to those living in urban communities (21%). Further, 35% of urban Australians aged 14 years or over reported that they had never smoked, compared with 31% of Australians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

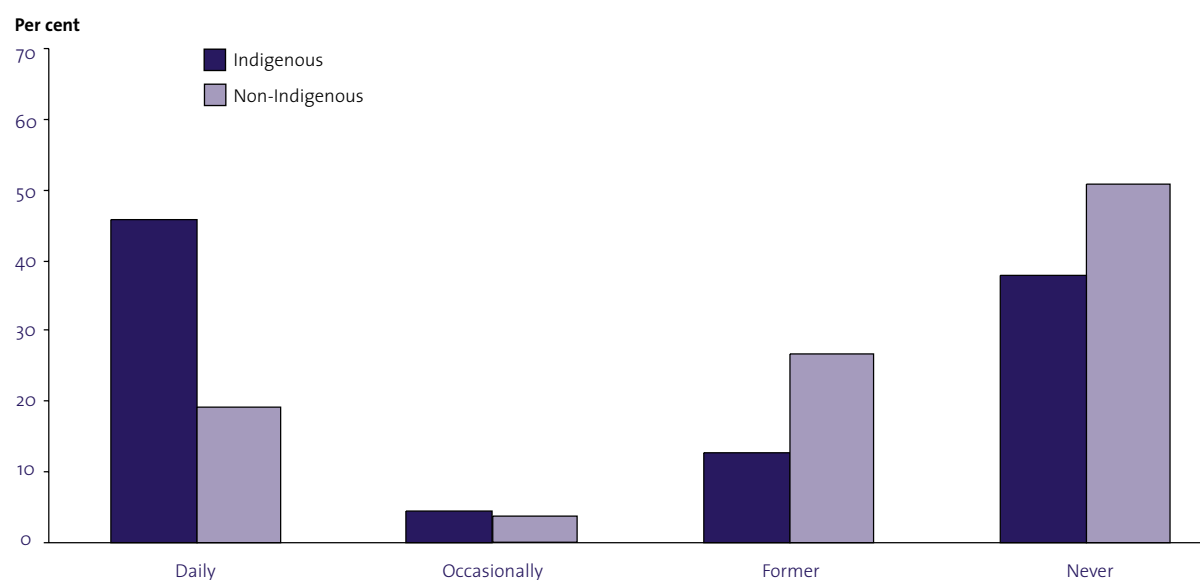
AIHW 1999. 1998 National Drug Strategy Household Survey: first results. Drug Statistics Series. AIHW Cat. No. PHE 15. Canberra: AIHW.

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:

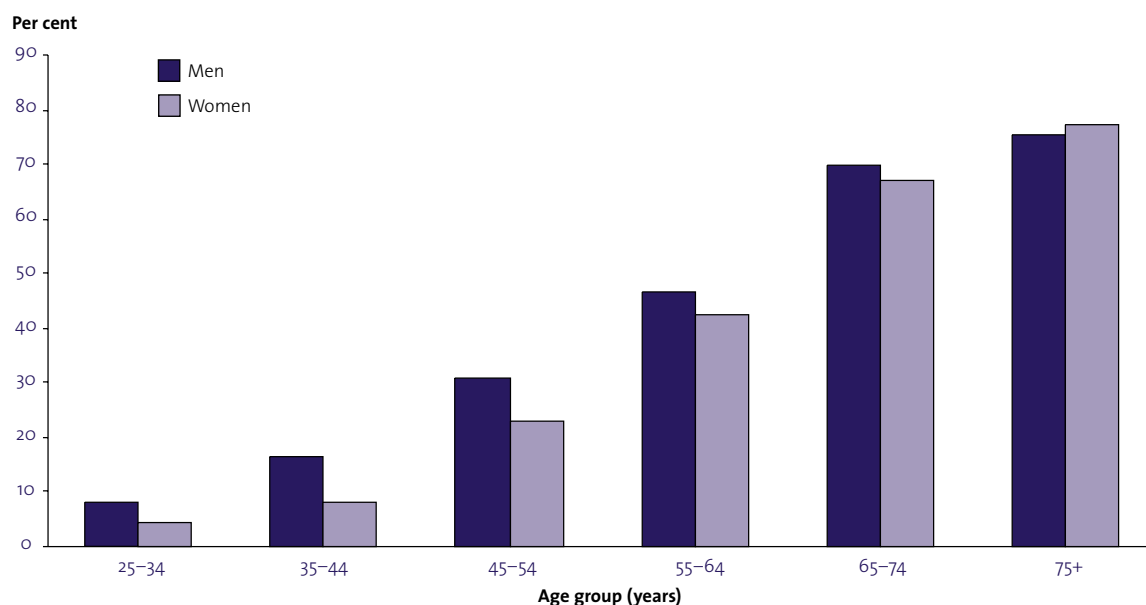
- systolic blood pressure (SBP) greater than or equal to 140 mm Hg; and/or
- diastolic blood pressure (DBP) greater than or equal to 90 mm Hg; and/or
- receiving medication for high blood pressure.

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.

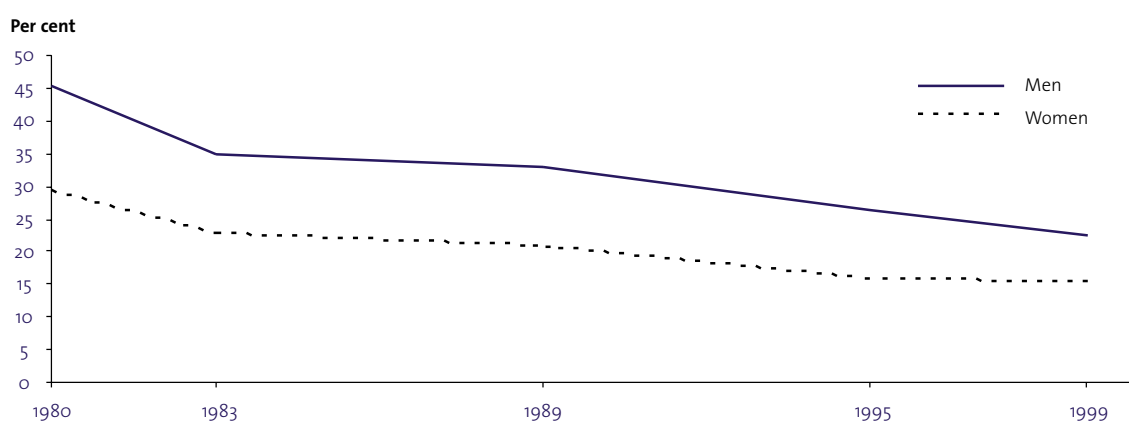
### Special population groups

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. Diabetes 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

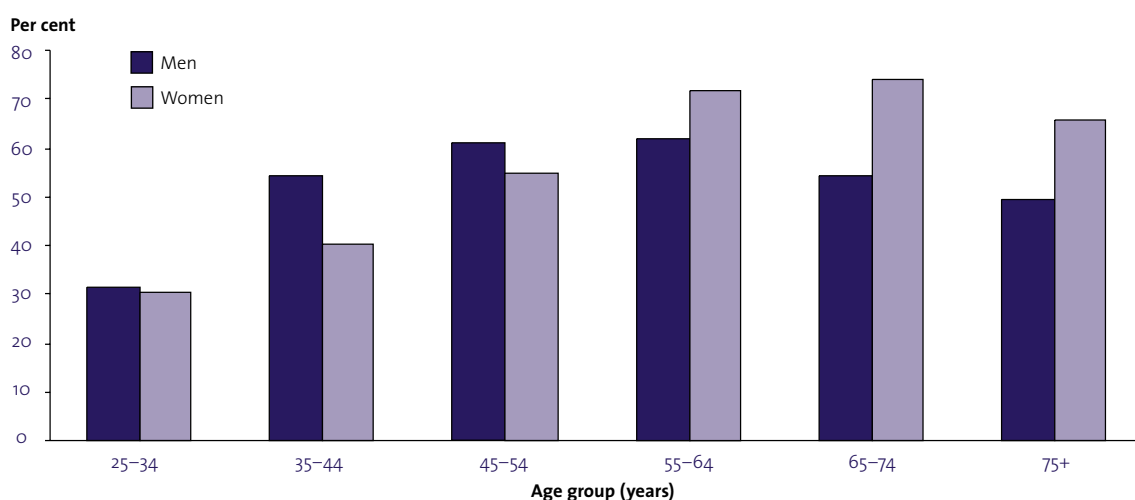
blood cholesterol—some people have high cholesterol levels regardless of their saturated fat and cholesterol dietary intake and are at increased risk of coronary heart disease (Bouchard et al. 1997). Hyperglycaemia is also known to contribute to high blood cholesterol levels (DHAC & AIHW 1999).

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

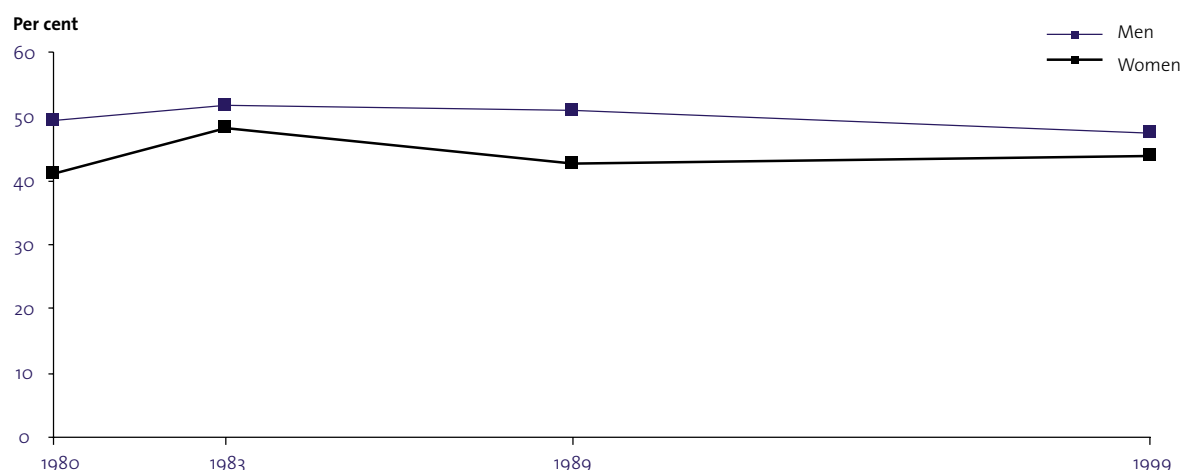
Sex	1980	1983	1989	1999–2000
mmol/L				
Men	5.6	5.7	5.6	5.6
Women	5.5	5.6	5.5	5.5

#### 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 ( $\geq 6.5$  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 ( $\geq 6.5$  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. Diabetes 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.



# Complications of diabetes

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# 4

Introduction

Cardiovascular disease

Eye disease

Kidney disease

Neuropathy

Foot complications

Oral complications

Complications in pregnancy

diabetes  
australian facts 2002

Australian Institute of Health and Welfare

## Introduction

Diabetes can result in a range of long-term complications in addition to causing acute metabolic reactions (e.g. ketoacidosis and diabetic coma). These complications are responsible for loss of working ability, invalidism, shortened life expectancy and reduced quality of life among people with diabetes.

People with diabetes are more prone to diseases of the large blood vessels (macrovascular disease) such as coronary heart disease, stroke and peripheral vascular disease as well as diseases of the small blood vessels (microvascular disease) such as retinopathy, kidney diseases and neuropathy (peripheral nerve disease). Other complications of or conditions associated with diabetes include digestive diseases (ulcers, coeliac disease, cancer of the pancreas, constipation, diarrhoea, liver disease and gallstones), infections, oral diseases, mental problems (depression and anxiety) and problems in pregnancy.

Complications arising from treatment can also occur in diabetes. These include hypoglycaemia from insulin or oral hypoglycaemic agents, side-effects of hypoglycaemic agents (liver toxicity, lactic acidosis, death from heart problems, allergic skin reactions), allergic reactions to insulin, and insulin resistance due to antibodies in the bloodstream binding the given insulin.

The underlying causes of diabetes complications remain controversial, although persistent high blood glucose and other consequences of insulin deficiency have been implicated. The Diabetes Control and Complications Trial, involving participants with Type 1 diabetes, showed that keeping blood glucose levels as close to normal as possible slows the onset and progression of eye, kidney and nerve diseases (NIDDK 2002). The United Kingdom Prospective Diabetes Study of people with Type 2 diabetes found that tight blood glucose control reduces the risk of major diabetic eye disorders by one-quarter and early kidney damage by one-third. Moreover, tight blood pressure control in people with high blood pressure reduces the risk of:

- death from long-term complications of diabetes by one-third;
- strokes by more than one-third; and
- serious deterioration of vision by more than a third (UKPDS 2002).

Improving the management and care of diabetes, particularly the early identification and reduction of risk factors, can delay the onset or slow the progression of complications.

Australian data on the complications of diabetes are limited. Those conditions for which data are currently available are discussed in more detail in this chapter.

## References and further reading

NIDDK (National Institute of Diabetes and Digestive and Kidney Diseases) 2002. Diabetes Control and Complications Trial. Viewed 2 May 2002, <<http://www.niddk.nih.gov/health/diabetes/pubs/dcct1/dcct.htm>>.

UKPDS (UK Prospective Diabetes Study) 2002. United Kingdom Prospective Diabetes Study. Viewed 2 May 2002, <<http://www.dtu.ox.ac.uk/index.html?maindoc=/ukpds/>>.



## Cardiovascular disease

Cardiovascular disease is a major complication of diabetes. People with diabetes are two to four times more likely to develop cardiovascular disease, their prognosis following a cardiovascular event (such as a heart attack) is not as good, nor do they fare as well after cardiac revascularisation procedures compared with those without diabetes (Wu, Brooks & Yue 1999). This section focuses on coronary heart disease, stroke and peripheral vascular disease, as they are the most common cardiovascular complications associated with diabetes.

### Coronary heart disease

Coronary heart disease is the most common cause of sudden death in Australia. It consists mainly of acute myocardial infarction (heart attack) and angina. A heart attack occurs when a vessel supplying blood to the heart muscle suddenly becomes blocked by a blood clot whereas angina is a temporary chest pain or discomfort caused by a reduced blood supply to the heart muscle.

### Stroke

Stroke (also referred to as cerebrovascular disease) includes ischaemic stroke, haemorrhagic stroke and transient ischaemic attack. The two main types of stroke are ischaemic, which occurs when an artery supplying blood to a part of the brain suddenly becomes blocked, and haemorrhagic, which is when an artery supplying blood to a part of the brain suddenly bleeds. These can damage part of the brain, which in turn can impair a range of functions including movement and speech. People who have had a transient ischaemic attack, which is a temporary cerebrovascular event that leaves no permanent damage, are at high risk for an acute ischaemic stroke attack.

### Peripheral vascular disease

Peripheral vascular disease occurs due to a reduced arterial blood supply to the legs. It ranges from asymptomatic disease, through pain on walking, to pain at rest. It can lead to amputation if blood supply

is significantly reduced. Although this is a significant cause of disability among people with peripheral vascular disease, the major cause of death in people with peripheral vascular disease is coronary heart disease.

### How does diabetes increase the risk of developing cardiovascular disease?

The reasons why diabetes increases the risk of cardiovascular disease are only partially understood. It is not clear, for example, whether the development of cardiovascular disease is the same in Type 1 and Type 2 diabetes, although both types are associated with an increased risk. It is also not clear why the increased risk of cardiovascular disease associated with diabetes is greater among women than men.

The prevailing explanation is that diabetes increases atherosclerosis (thickening of the walls of a blood vessel with deposits of plaque). However, improved blood glucose control by itself may not be sufficient to eliminate the excess risk of diabetic cardiovascular complications (Wu, Brooks & Yue 1999), despite its effectiveness in reducing the progression of diabetic microvascular complications such as eye and kidney disease (Diabetes Control and Complications Trial Research Group 1993).

Other factors possibly contributing to the excess risk of cardiovascular disease in diabetes include high blood pressure and dyslipidaemia (low levels of HDL cholesterol and high levels of LDL cholesterol and triglycerides). Both are risk factors for cardiovascular disease and their prevalence is higher among people with diabetes (Wu, Brooks & Yue 1999).

### Risk factors

The risk of developing cardiovascular disease increases when diabetes is present with other risk factors such as tobacco smoking, physical inactivity, high blood pressure, high blood cholesterol, and overweight and obesity.



## How many Australians with diabetes also have cardiovascular disease?

According to the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study, 12% of Australians aged 25 or over with diabetes had had a heart attack (approximately 113,000 people). Nine per cent had had a stroke (approximately 85,000 people). These proportions were much greater than among people without diabetes (3% and 2%, respectively).

There are limited national data on the number of Australians who have diabetes and peripheral vascular disease. Data collected through the Australian National Diabetes Information Audit and Benchmarking survey (refer to Appendixes for more information on data sources) in 2000 revealed that 14.5% of adults with diabetes also had peripheral vascular disease.

## General practice consultations

Data from the 1998–99 study of general practice activity in Australia show that the rate of diabetes in patients managed for coronary heart disease, stroke or peripheral vascular disease is far higher than the average, indicating a clear association between diabetes and cardiovascular disease.

## Hospitalisations

Diabetes increases the likelihood of being hospitalised for cardiovascular disease and of this occurring at a younger age.

In 1999–00, there were nearly 30,000 hospitalisations for coronary heart disease where diabetes was also present (19% of all hospitalisations for coronary heart disease). There were nearly 9,000 hospitalisations for stroke where diabetes was also present (17% of all hospitalisations for stroke) and approximately 1,700 hospitalisations for peripheral vascular disease where diabetes was also present (13% of all hospitalisations for peripheral vascular disease).

Males are much more likely to be hospitalised for cardiovascular disease than females irrespective of whether diabetes is present, although the presence of diabetes increases the likelihood of hospitalisation among females. The rate of hospitalisation for

coronary heart disease or stroke increased over the period 1993–94 to 1999–00 among both males and females with diabetes. The disparity between the sexes increased over this period also (refer to NHPA indicator 4.2 in Appendixes).

## Mortality

In 2000, diseases of the circulatory system were listed as the underlying cause of death in 55.7% of deaths where diabetes was an associated cause of death. Coronary heart disease accounted for almost two-thirds of these deaths while stroke accounted for one in five of these deaths. An examination of cardiovascular mortality associated with diabetes, such as this, is dependent on diabetes being recorded as a contributing cause of death on death certificates. This may not occur when death is sudden, as might occur with a heart attack.

Coronary heart disease (mainly heart attacks) was the leading cardiovascular cause of death in 2000, accounting for 26,521 deaths (21% of all deaths). Of these, 2,508 (9.5% of coronary heart disease deaths) recorded diabetes as a contributing cause of death. Stroke was the second most common cause of death, accounting for 12,354 deaths. Of these, 807 (6.5% of stroke deaths) recorded diabetes as a contributing cause of death. Peripheral vascular disease accounted for 2,046 deaths and of these, 32 (1.6% of deaths due to peripheral vascular disease) recorded diabetes as a contributing cause of death.

Males are more likely than females to die from cardiovascular disease. Death rates for coronary heart disease and peripheral vascular disease are almost twice as high among males but the difference between the sexes is not as great for stroke (Table 4.1). This pattern emerges irrespective of whether diabetes is recorded as a contributing cause.

Death rates for coronary heart disease or stroke where diabetes was recorded as a contributing cause of death fell slightly between 1997 and 2000 among both males and females (2.4% and 2.6%, respectively) (refer to NHPA indicator 5.2 in Appendixes). By comparison, over the same period, falls in the death rate for all cardiovascular disease were 4.4% and 3.8% for males and females, respectively.

**Table 4.1:** Death rate for coronary heart disease, stroke and peripheral vascular disease where diabetes was recorded as a contributing cause, by sex, 2000

	Coronary heart disease	Stroke	Peripheral vascular disease
	Rate (per 100,000 persons)		
Males	14.7	3.8	0.2
Females	8.3	3.1	0.1

#### Notes

1. The rates are age-standardised using the Australian population as at 30 June 1991.
2. The disease groupings are classified according to the ICD-10 codes: I20–I25 for coronary heart disease, G45, G46 and I60–I69 for stroke, I71–I74 for peripheral vascular disease and E10–E14 for diabetes as a contributing cause of death.

Source: AIHW National Mortality Database.

The higher prevalence of diabetes among people from different culturally and linguistically diverse backgrounds is also reflected in higher death rates for cardiovascular disease among this population group. Among males the death rate is nearly 25% higher and among females it is 35% higher compared with the death rates for coronary heart disease or stroke in the general population where diabetes was recorded as a contributing cause of death.

### Main data sources

2000 Australian National Diabetes Information Audit and Benchmarking (ANDIAB) (National Association of Diabetes Centres).

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

1998–2000 Bettering the Evaluation and Care of Health Study (University of Sydney & Australian Institute of Health and Welfare).

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

National Mortality Database (Australian Institute of Health and Welfare).

### 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 2002. Australia's health 2002: the eighth biennial health report of the Australian Institute of Health and Welfare. AIHW Cat. No. AUS 25. Canberra: AIHW.

Diabetes Control and Complications Trial Research Group 1993. The effect of intensive therapy of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *New England Journal of Medicine* 329:977–86.

Wingard DL & Barrett-Connor E 1995. Heart disease and 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, 429–48.

Wu T, Brooks B & Yue D 1999. Macrovascular disease: the sword of Damocles in diabetes. In: Turtle J, Kaneko T & Osato S (eds). *Diabetes in the new millennium*. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney.

## Eye disease

People with diabetes are at an increased risk of developing eye disease, particularly diabetic retinopathy (retinal disease), cataract and glaucoma. Diabetic retinopathy is the most common cause of blindness in people aged 30–69 years (Constable 1999; Donnelly et al. 2000). Cataracts and glaucoma are also major causes of vision impairment and blindness among adults.

### Diabetic retinopathy

Diabetic retinopathy is a microvascular complication of diabetes caused by damage to the capillaries of the retina (the light-sensitive tissues at the back of the eye). It is a progressive disorder classified as non-proliferative or proliferative according to the presence of various clinical abnormalities (NHMRC 1997).

In the early stages, known as non-proliferative diabetic retinopathy (NPDR), retinal capillaries swell and leak fluid. NPDR is not usually associated with visual impairment. As the disease progresses (known as proliferative diabetic retinopathy or PDR) abnormal new capillaries grow on the surface of the retina. Without treatment, these capillaries can bleed causing cloudy vision or blindness. Abnormal fibrous tissue can also develop, leading to retinal detachment with severe vision loss. Blurred central vision may occur when the macular (the central part of the retina that gives the sharpest vision) swells from leaking fluid (called macular oedema).

Diabetic retinopathy is symptomless in its early phases. However, it can be treated successfully by laser surgery if identified early. It is estimated that early detection and timely treatment can prevent nearly all of severe vision loss and blindness due to diabetic retinopathy (Lee et al. 2001).

### Cataracts and glaucoma

A cataract is a clouding of the normally clear lens of the eye, leading to vision loss. A cloudy lens prevents light from entering the eye. Cataracts are more common and progress more rapidly in people with diabetes (Klein & Klein 1995).

Glaucoma is a condition where pressure builds up in the eye, pinching the capillaries that carry blood to the retina and optic nerve. Over time, the retina and optic nerve become damaged and vision is lost. People with diabetes are significantly more likely to develop glaucoma than people without diabetes (Klein & Klein 1995).

### Risk factors

Age at onset and duration of diabetes are key factors influencing the prevalence of diabetic retinopathy. In young people with diabetes (aged less than 30 years at diagnosis), the prevalence is as high as 25% during the first 5 years after diagnosis, increasing to 50% after 15 years since diagnosis. In older people (aged 30 years or more at diabetes diagnosis), up to 20% may have signs of retinopathy, rising to 60% after 15 years with diabetes (Mensah & Kohner 2002).

In addition to duration of diabetes, the risk of developing eye complications and visual impairment increases with coexisting medical problems or complications (such as high blood pressure and nephropathy), poor blood glucose control, pregnancy, elevated blood lipids and smoking (Cohen et al. 1998; NHMRC 1997).

### How many Australians with diabetes also have eye disease?

#### Diabetic retinopathy

The Australian Diabetes, Obesity and Lifestyle Study, carried out in 1999–2000, found that 15.4% of people with diabetes (known and newly diagnosed) had retinopathy. The prevalence among men was 14.0% and among women was 16.6%. The prevalence of retinopathy increased dramatically with duration of diabetes (duration 0–4 years 7.4%, 5–9 years 25.6%, 10–19 years 33.8%, and  $\geq 20$  years 60.5%). A similar rate of retinopathy was reported in a South Australian survey—19% of Type 2 diabetes sufferers had retinopathy (Phillips et al. 1998).

The National Divisions Diabetes Program Data Collation Project found that 11.5% of patients examined had retinopathy detected in at least one eye during 1999–00. Data on the prevalence of diabetic retinopathy among patients attending diabetes clinics are also available from the Australian National Diabetes Information Audit and Benchmarking (ANDIAB) survey. According to ANDIAB, of those patients who had a retinal assessment in 2000, 27.3% had retinopathy in the right eye and 27.1% had retinopathy in the left eye (NADC 2000). These latter estimates are not derived from population-based surveys. ANDIAB data reports on persons with diabetes requiring specialist clinical management, in particular those who have had poor control of their diabetes. Thus, ANDIAB figures may overstate the true rate of this condition among all people with diabetes. For further information on data sources refer to the 'Methods and data sources' section in the Appendixes.

### Cataracts and glaucoma

During the 1995 National Health Survey, 9.9% of respondents who reported ever having had diabetes also reported cataracts, and 3.2% reported glaucoma. These proportions were considerably higher than in people without diabetes (more than six times the rate of cataracts and more than four times the rate of glaucoma reported among persons without diabetes). A slightly larger proportion of females with diabetes reported cataracts than males (12.4% compared with 7.3% respectively). In contrast, more males than females reported glaucoma (4.6% compared with 1.8% respectively).

### Blindness

During the 1995 National Health Survey (NHS), 4.9% of respondents who reported ever having had diabetes also reported blindness. This was five times the rate reported among persons without diabetes. Similar proportions of males and females with diabetes reported blindness—5.6% for females and 4.2% for males.

Based on ANDIAB data for 2000, the incidence rate for blindness is estimated to be around 7 per 1,000 among persons with clinically diagnosed diabetes (refer to National Health Priority Areas indicator 3.2 in Appendixes). The ANDIAB figures are not directly comparable with the NHS figures because the NHS data on blindness was self-reported whereas the ANDIAB data was based on visual acuity measures.

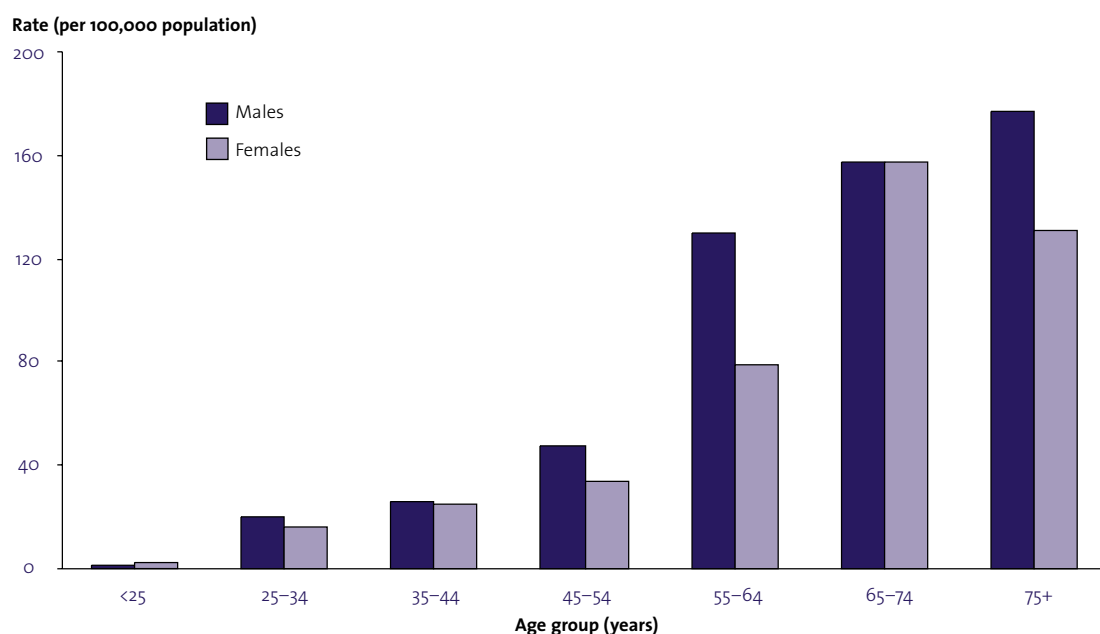
### Hospitalisations

In 1999–00, there were 7,733 hospitalisations for diabetes-related eye complications (including retinopathy, glaucoma and cataract). These diabetes-specific disorders are discretely coded and accounted for 2.3% of all diabetes hospitalisations. Males were more likely to be hospitalised for diabetes-related eye complications than females. Hospital use for diabetes with eye complications tends to increase with age, with those aged 65 and over accounting for almost half of all hospitalisations for diabetes with eye complications in 1999–00 (Figure 4.1).

The average length of stay in hospital for diabetes with eye complications in 1999–00 was 7.5 days. Males and females tended to have a similar length of stay, 7.5 days and 7.4 days respectively.

### Aboriginal and Torres Strait Islander people

There are limited data on the prevalence of diabetic retinopathy in Aboriginal and Torres Strait Islander people. In a Western Australian study of diabetes, 31% of Aboriginal and Torres Strait Islander people were found to have diabetic retinopathy, compared with 20% of non-Indigenous Australians (Stanton et al. cited in OATSIH 2001). More recently, in a study of Indigenous Australians in a rural community, the prevalence of diabetic retinopathy was found to be 14% among those with diabetes (Keefe et al. cited in OATSIH 2001).

**Figure 4.1:** Hospitalisations for diabetes with eye complications, 1999–00

Source: AIHW National Hospital Morbidity Database.

The available data are likely to underestimate the magnitude of this problem among Aboriginal and Torres Strait Islander people. Aboriginal and Torres Strait Islander people often have compounding factors such as high blood pressure and diabetic nephropathy, both of which are associated with the development and severity of diabetic retinopathy (OATSIH 2001). It is also suggested that diabetic retinopathy may be more severe at the time of diagnosis among Aboriginal and Torres Strait Islander people as a result of delayed diagnosis of Type 2 diabetes (OATSIH 2001).

### Main data sources

2000 Australian National Diabetes Information Audit and Benchmarking (ANDIAB) (National Association of Diabetes Centres).

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

1999–2000 National Divisions Diabetes Program (NDDP) Data Collation Project.

1995 National Health Survey (Australian Bureau of Statistics).

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

### References and further reading

Carter S, Bonney M, Flack J, Burns J, Powell Davies PG & Harris MF 2000. National Divisions Diabetes Program Data Collation Project. Volume 5: Divisions of General Practice—Diabetes profiles. Quality of care and health outcomes—collated CARDIAB data. Sydney: Centre for General Practice Integration Studies, School of Community Medicine, University of New South Wales.

Cohen O, Norymberg K, Neumann E & Dekel H 1998. Complication-free duration and the risk of development of retinopathy in elderly diabetic patients. Archives of International Medicine 158:641–4.

Constable I 1999. Diabetic retinopathy: pathogenesis, clinical features and treatment. In: Turtle J, Kaneko T & Osato S (eds). Diabetes in the new millennium. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney, 365–85.

Donnelly R, Emslie-Smith AM, Gardner ID & Morris AD 2000. ABC of arterial and venous disease. Vascular complications of diabetes. British Medical Journal 320:1062–66.

Klein R & Klein B 1995. Vision disorders in 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, 293–338.

Lee SJ, McCarty CA, Taylor HR & Keefe JE 2001. Costs of mobile screening for diabetic retinopathy: a practical framework for rural populations. Australian Journal of Rural Health 9:186–92.

Mensah E & Kohner EM 2002. Diagnosis and management of diabetic retinopathy. Topical Endocrinology 19:14–18.

NADC (National Association of Diabetes Centres) 2000. ANDIAB 2000. Australian National Diabetes Information Audit & Benchmarking. Canberra: National Association of Diabetes Centres.

NHMRC (National Health and Medical Research Council) 1997. Management of diabetic retinopathy. Clinical practice guidelines, February 2002. Viewed 29 April 2002, <<http://www.health.gov.au/nhmrc/publications/pdfcover/cp53covr.htm>>.

OATSIH (Office for Aboriginal and Torres Strait Islander Health) 2001. Specialist eye health guidelines for use in Aboriginal and Torres Strait Islander populations: cataract, diabetic retinopathy, trachoma. Canberra: OATSIH.

Phillips P, Wilson D, Beilby J et al. 1998. Diabetes complications and risk factors in an Australian population. How well are they managed? International Journal of Epidemiology 27:853–9.

Taylor HR 1997. Eye health in Aboriginal and Torres Strait Islander Communities. Canberra: Commonwealth Department of Health and Aged Care.



## Kidney disease

Diabetes can affect the kidneys in a variety of ways, leading to serious and even life-threatening conditions. This section focuses on diabetic nephropathy and end-stage renal disease (ESRD), as these are the most common kidney complications associated with diabetes.

### Diabetic nephropathy

Diabetic nephropathy results from high blood glucose levels damaging the blood-filtering capillaries (glomeruli) in the kidneys. The glomeruli's filtering efficiency declines and blood proteins such as albumin leak into the urine (albuminuria).

In the early stages of diabetic nephropathy, small quantities of albumin leak into the urine (called microalbuminuria). As diabetic nephropathy progresses, the kidneys leak larger amounts of albumin (called macroalbuminuria or proteinuria). Microalbuminuria is a strong predictor of developing proteinuria, ESRD, high blood pressure and cardiovascular disease. Proteinuria indicates a substantial decline in kidney function and is associated with high mortality, particularly from ESRD. Individuals with proteinuria are also known to be at an increased risk of developing high blood pressure, coronary heart disease, peripheral vascular disease and retinopathy.

Diabetic nephropathy is often symptomless until late in the disease when therapeutic interventions are ineffective. However, early detection and intervention may slow or halt its progression.

Diabetic nephropathy can be readily detected by urine testing for albumin. Identifying and treating individuals with microalbuminuria, before there is a substantial decline in kidney function, is very important. Tight control of blood glucose and blood pressure may prevent microalbuminuria progressing to proteinuria or ESRD.

### End-stage renal disease (ESRD)

ESRD is the final stage in the worsening of kidney function, when the kidneys lose the ability to remove waste products such as creatinine and urea from the blood. Thereafter, dialysis (filtering of the blood by machine) or kidney transplantation is necessary to maintain life. Diabetic nephropathy is the second most common cause of ESRD in Australia (Russ 2001).

Measuring the glomerular filtration rate and the quantity of creatinine in the blood can indicate the severity of kidney damage.

### Risk factors

Factors that may determine whether diabetic nephropathy develops and progresses to ESRD include long duration of diabetes, poor blood glucose control, high blood pressure, genetic susceptibility to diabetic kidney disease and smoking.

### How many Australians with diabetes also have kidney disease?

During the 1995 National Health Survey, 6.1% of respondents who reported ever having had diabetes also reported having kidney disease, more than four times the rate reported among persons without diabetes. Similar proportions of males and females with diabetes reported kidney disease—6.3% for females and 5.9% for males.

According to the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab), 11.2% of Australians aged 25 or over with self-reported diabetes reported being treated for or suffering from kidney disease. Significantly more women with self-reported diabetes reported being treated for or suffering from kidney disease than men (18.4% compared with 5.2%).



## Diabetic nephropathy

Examining the prevalence of diabetic nephropathy in populations is problematic due to differences in methods of measurement for albuminuria and lack of standardised terminology. Health outcomes can only be assessed when the type of urine collection is recorded, allowing the appropriate units and reference limits to be applied.

Data on the prevalence of diabetic nephropathy based on urinary albumin measurements are available from the 1999–2000 AusDiab study. The prevalence of proteinuria was found to be more than four times higher in those with diabetes compared with those without (8.7% versus 1.9% respectively) (Chadban et al. unpub.). The prevalence of low glomerular filtration rate was also three times higher in those with diabetes compared with those without (27.6% compared with 9.8%) (Chadban et al. unpub.).

Data on the prevalence of albuminuria among patients attending diabetes clinics are available from the Australian National Diabetes Information Audit and Benchmarking collection (ANDIAB). Of those who had a urinary albumin assessment in 2000 (41.2% of patients) 66.4% had normal albumin levels (normoalbuminuria), 27.9% had microalbuminuria and 5.7% had macroalbuminuria (NADC 2000).

These data are also available from the National Divisions Diabetes Program (NDDP) Data Collation Project. In 1999–00, 1,284 of the 4,359 registered NDDP patients had albuminuria assessed. Of those, 76% had normoalbuminuria, 20.9% had microalbuminuria and 3.1% had macroalbuminuria.

## End-stage renal disease (ESRD)

Evidence of the burden of ESRD caused by diabetes is available from the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry, a registry of people receiving kidney dialysis or a kidney transplant. New

cases of ESRD with diabetic nephropathy as the primary cause have increased dramatically over the past decade (refer to National Health Priority Areas indicator 3.1 in Appendixes). This increase has been most evident among patients with Type 2 diabetes.

In Australia, during 2000, diabetes was the second most common cause of primary kidney disease among ANZDATA patients, accounting for more than one in five (22%) new patients (Russ 2001). This represents a considerable increase in the proportion of ESRD cases with diabetes—from one in eight cases, or 12.5%, in 1991. The burden of ESRD from diabetes, particularly Type 2 diabetes, is likely to increase further as both the age of the population and prevalence of diabetes are projected to rise dramatically.

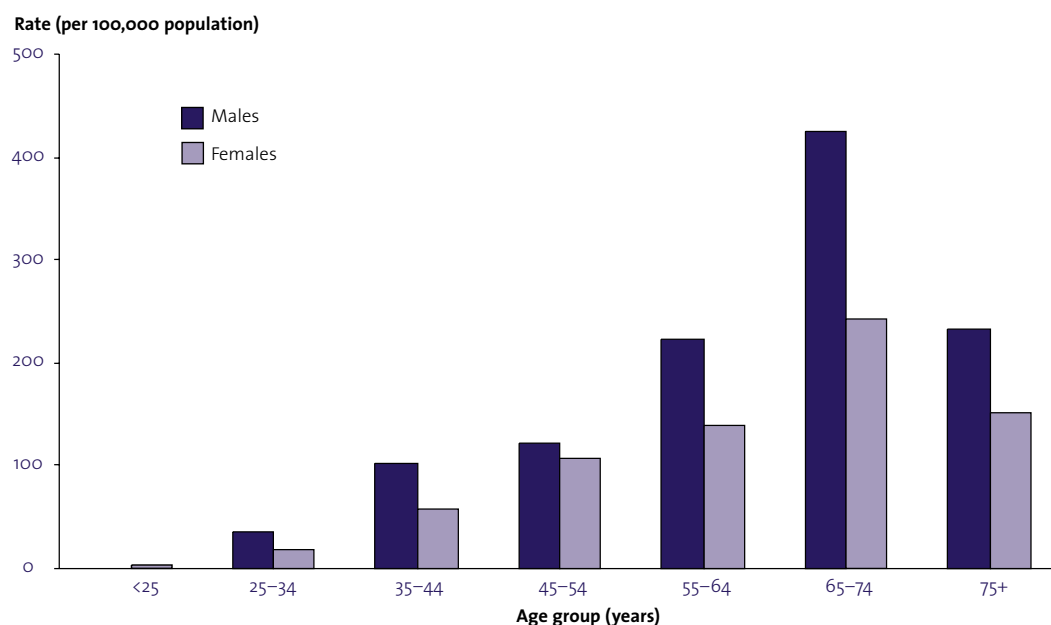
## Hospitalisations

### Kidney (renal) complication

In 1999–00, there were 15,236 hospitalisations for diabetes with kidney complication; these accounted for 4.5% of hospital admissions for diabetes. Males with diabetes were much more likely to be hospitalised for kidney complication than females. Hospital use for diabetes with kidney complication increased with age and peaked at 65–74 years for both men and women (Figure 4.2).

The average length of stay in hospital for diabetes with kidney complication was 5.3 days. Females tended to have longer average lengths of stay than males (5.8 days compared with 4.9 days respectively).

Day-stay admissions for regular dialysis are extremely common for people with kidney disease (Australian Kidney Foundation 1999). After removal of day-stay admissions, the average length of stay in hospital for diabetes with kidney complication increased considerably to 10.1 days (10.2 days for males and 9.9 days for females).

**Figure 4.2:** Hospitalisations for diabetes with kidney complication, 1999–00

Source: AIHW National Hospital Morbidity Database.

### End-stage renal disease (ESRD)

In 1999–00 there were 2,691 admissions where ESRD was the principal diagnosis and diabetes was an additional diagnosis. Males with diabetes were more likely to be hospitalised for ESRD than females. The average length of stay in hospital for people with ESRD as the principal and diabetes as an additional diagnosis was 9.5 days. Length of stay in hospital was generally higher for females than for males (10.3 days compared with 8.8 days).

### Deaths

Kidney-related diseases were listed as an associated cause of death in 22.6% of cases where diabetes was listed as the underlying cause of death in 2000. In addition, diabetes was associated with 7% of deaths where kidney failure was the underlying cause of death.

### Aboriginal and Torres Strait Islander people

There are limited data on diabetic kidney complications among Aboriginal and Torres Strait Islander people. However, the available data indicate that Aboriginal and Torres Strait Islander people are proportionately much more likely to develop kidney disease and ESRD as a result of diabetes than the rest of the population.

Data from the ANZDATA Registry indicate that the yearly incidence of ESRD associated with diabetes is considerably higher among Aboriginal and Torres Strait Islander peoples. During 2000, 46% of Indigenous Australian patients had diabetic nephropathy compared with around 14% of non-Indigenous patients (Russ 2001). Nevertheless, the size of the problem of ESRD in Indigenous Australians is

likely to be underestimated by this register as a result of their poorer access to dialysis and transplant programs (Disney 1992; Russ 2001).

In a study in the Northern Territory, the incidence of ESRD associated with diabetes was 26.5 times higher in Aboriginal and Torres Strait Islander people compared with non-Indigenous Australians (90.2 per million cases in Indigenous Australians and 3.4 per million in non-Indigenous Australians) (Hoy et al. cited in de Courten et al. 1998). Moore et al. (1996) also found diabetic kidney disease to be significantly more common among Aboriginal and Torres Strait Islander patients than non-Indigenous patients (22% compared with 6% of non-Indigenous patients).

## Main data sources

2000 Australian National Diabetes Information Audit and Benchmarking (ANDIAB) (National Association of Diabetes Centres).

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

1999–2000 National Divisions Diabetes Program (NDDP) Data Collation Project.

1995 National Health Survey (Australian Bureau of Statistics).

Australia and New Zealand Dialysis and Transplant Registry (ANZDATA).

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

National Mortality Database (Australian Institute of Health and Welfare).

## References and further reading

ABS (Australian Bureau of Statistics) 2001. Causes of death, Australia, 2000. Cat. No. 3303.0. Canberra: ABS.

Australian Kidney Foundation 1999. The Australian kidney. National Epidemiological Survey of Diseases of the Kidney and Urinary Tract. Adelaide: Australian Kidney Foundation.

Chadban S, Briganti E, Kerr P, Dunstan D, Welborn T & Zimmet P (unpublished). Prevalence of kidney damage in Australian adults—The AusDiab Kidney Study.

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.

Disney A (ed.) 1992. ANZDATA Registry Report 1992. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

Donnelly R, Emslie-Smith AM, Gardner ID & Morris AD 2000. ABC of arterial and venous disease. Vascular complications of diabetes. *British Medical Journal* 320:1062–66.

Hoy WE, Mathews JD & Pugsley DJ 1995. Treatment of Australian Aboriginals with end-stage renal disease in the top end of the Northern Territory: 1978–93. *Nephrology* 1:307–13.

Jerums G, Gilbert RE & Panagiotoulos S 1999. Diabetic nephropathy: recent concepts in mechanisms and management. In: Turtle J, Kaneko T & Osato S (eds). *Diabetes in the new millennium*. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney, 365–85.

Moore L, Lloyd MS, Pugsley DJ & Seymour AE 1996. Renal disease in the Australian Aboriginal population: a pathological study. *Nephrology* 2:315–21.

NADC (National Association of Diabetes Centres) 2000. ANDIAB 2000. Australian National Diabetes Information Audit & Benchmarking. Canberra: National Association of Diabetes Centres.

Russ GR (ed.) 2001. ANZDATA Registry Report 2001. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

## Neuropathy

Neuropathy (nerve damage) is a frequent complication of diabetes. Diabetic neuropathy usually manifests as either peripheral neuropathy, most commonly causing damage to the nerves in the feet, or autonomic neuropathy. The sequelae of diabetic neuropathy include pain, digestive problems, muscle weakness, non-healing ulcers and lower extremity amputation, and are associated with reduced quality of life and increased mortality. Diabetic neuropathy is generally a result of chronically high blood glucose levels which affect the metabolism of nerves. This in turn causes the accumulation of toxins which damage nerve structure and function.

### Peripheral neuropathy

Peripheral neuropathy is the presence of symptoms and/or signs of damage to the peripheral nerves (the nerves outside the brain and spinal cord) (Oyibo et al. 2002). Peripheral neuropathy can cause a diverse range of symptoms, depending on the nerve(s) affected, although some people will experience no obvious symptoms.

There are two broad types of peripheral neuropathy:

- Sensory neuropathy—affects the nerves that carry information to the brain about sensations from various parts of the body. Symptoms may include pain, tingling in the limbs or absence of feeling in the feet (which predisposes people with diabetes to foot trauma).
- Motor neuropathy—affects the nerves that carry signals to muscles to allow the muscles to move. Motor neuropathy can lead to muscle weakness, particularly in the feet, which may become deformed as a result.

### Autonomic neuropathy

Autonomic neuropathy affects the nerves that control involuntary body functions such as heart rate, blood pressure, sweating, and the action of the stomach, intestine and bladder. Symptoms may include dizziness and fainting, nausea, vomiting and diarrhoea, loss of bladder control and impotence in men.

### Risk factors

The risk of developing neuropathy increases with duration of diabetes, poor blood glucose control and age. Strict glycaemic control has been shown to reduce or prevent the development of neuropathy, and may alleviate neuropathic symptoms. Early identification is essential, especially in people with no obvious symptoms, to prevent the late sequelae of neuropathy. A combination of clinical observations and complex nerve function tests are often required to confirm the presence of diabetic neuropathy.

### How many Australians with diabetes also have neuropathy?

According to the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study, 10.3% of males and 9.4% of females with diabetes (known and newly diagnosed) had clinical signs of neuropathy. Also, 30.2% of men with self-reported diabetes reported suffering from or receiving treatment for impotence (difficulty getting or sustaining an erection).

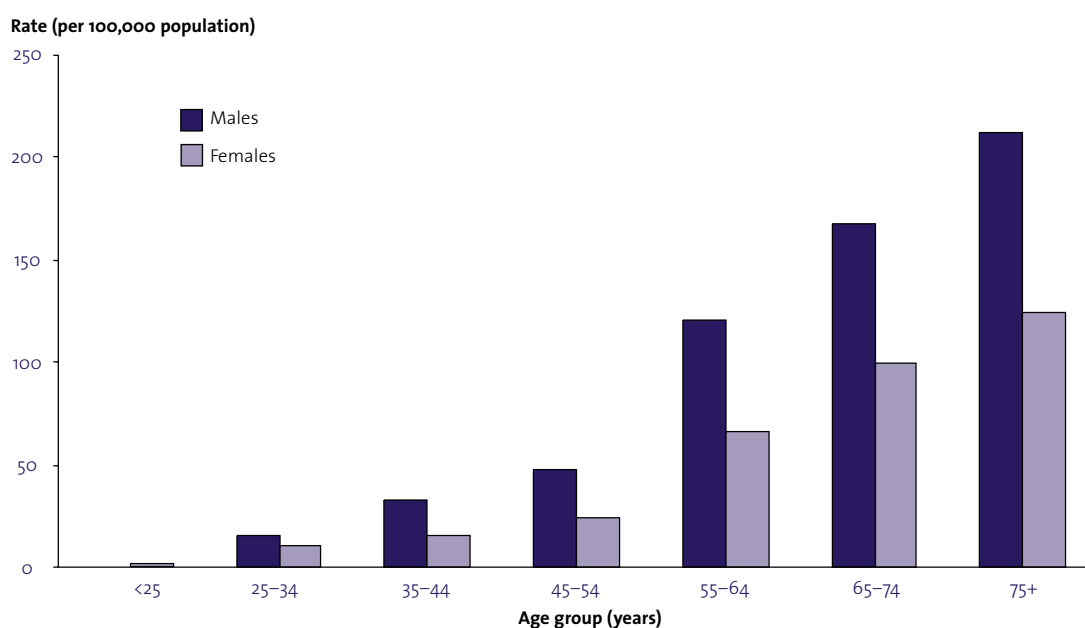
In the 2000 Australian National Diabetes Information and Benchmarking (ANDIAB) study, almost one-quarter (24.2%) of adult patients were recorded as having peripheral neuropathy following clinical assessment. However, it should be noted that ANDIAB data are obtained from specialist diabetes clinics that are likely to see more patients with complications. Impotence during the previous 12 months was also reported by 2.4% of male adult patients, with 25.7% reporting impotence before the previous 12 months.

### Hospitalisations

In 1999–00, there were 6,954 hospitalisations for diabetes-related neurological complications. These diabetes-specific neuropathies accounted for 2.1% of hospital admissions for diabetes.

Males were almost twice as likely as females to be admitted to hospital for diabetes with neurological complication. Hospital use for neurological complication increased dramatically among older people with diabetes, with around half of such cases being aged 65 and over in 1999–00 (Figure 4.3).

**Figure 4.3:** Hospitalisations for diabetes with neurological complication, 1999–00



Source: AIHW National Hospital Morbidity Database.

During 1999–00 the average length of stay in hospital for people with diabetes and neurological complication was 9.4 days. Males tended to have shorter average length of stay than females; 9.0 days compared with 10.1 days.

### Main data sources

2000 Australian National Diabetes Information Audit and Benchmarking (ANDIAB) (National Association of Diabetes Centres).

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

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

National Mortality Database (Australian Institute of Health and Welfare).

### References and further reading

American Diabetes Association & American Academy of Neurology 1988. Consensus statement. Report and recommendations of the San Antonio Conference on Diabetic Neuropathy. *Diabetes Care*, 11(7):592–7.

Feldman EV, Stevens MJ & Greene DA 1999. Diabetic neuropathy. In: Turtle J, Kaneko T & Osato S (eds). *Diabetes in the new millennium*. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney, 365–85.

Oyibo SO, Dang CN & Boulton AJM 2002. Diagnosis and management of diabetic neuropathy. *Topical Endocrinology* 19:10–13.

Vinik AI, Holland MT, Le Beau JM, Liuzzi FJ, Stansberry KB & Colen LB 1992. Diabetic neuropathies. *Diabetes Care* 15(12):1926–75.

## Foot complications

Diabetes is associated with nerve damage (peripheral neuropathy) and poor circulation (peripheral vascular disease—PVD) in the lower limbs. These factors increase the risk of developing foot ulcers and infections. Progression of these conditions in people with diabetes often leads to lower extremity amputations. Amputations are associated with increased morbidity and mortality and high treatment costs. Diabetes is estimated to account for approximately half of all non-traumatic amputations (DHAC & AIHW 1999).

### Foot ulcer

Over time, diabetes can damage the nerves in the feet, resulting in a loss of sensation. Reduced sensation of pain and discomfort from foreign bodies, injury or even tightly fitting shoes can predispose people to foot trauma and ulceration. Damage to nerves also causes wasting of the foot muscles, reduced joint mobility and foot deformities such as claw or hammer toes that are vulnerable to ulceration.

High blood glucose can also damage blood vessels in the lower limbs. Without a healthy supply of oxygen and nutrients, feet are predisposed to ulceration and infection.

Foot ulceration is a common reason for hospital admission for people with diabetes and is estimated to precede more than half of all diabetes-related amputations.

### Lower extremity amputation

The combination of diabetic neuropathy, PVD and foot deformity increases the risk of lower limb ulcers. Non-healing ulcers can result in gangrene (chronic infection resulting in tissue death). Amputation of the affected area may be necessary as a limb-salvaging procedure if medical treatment is unsuccessful.

Amputation is estimated to be 15 times more common in people with diabetes. Nearly half of the amputations in people with diabetes are minor

(involving toes, feet and ankles); the other half are major (below knee or above knee) (Campbell et al. 2000). Major amputations are associated with greater loss of limb function and require greater rehabilitation following amputation (Oyibo et al. 2002).

Many patients with diabetes who undergo amputation will have a subsequent amputation on the other side within a few years. The remaining limb becomes more vulnerable to ulceration and infection because it has to bear extra pressure.

### Risk factors

The risk of lower limb ulcers and amputations is higher in people who have had diabetes for 10 years or more, are male, have poor blood glucose control, have cardiovascular, visual or kidney complications, or smoke. Certain foot-related conditions are associated with an increased risk of foot ulcer and amputation: peripheral neuropathy (particularly loss of protective sensation), PVD, foot deformity, and prior history of foot ulcers or amputation.

Improved glycaemic control can prevent or reduce the development of diabetic neuropathy. Regular monitoring of the feet for early signs of diabetic neuropathy, peripheral vascular disease and foot deformities are essential. Appropriate therapeutic footwear, combined with podiatry care and footcare education, may also reduce the risk of serious foot ulcers and amputation.

### How many Australians with diabetes also have foot complications?

#### Foot ulcer

The 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) assessed the prevalence of various diabetes-related complications in Australia. Among people with known and newly diagnosed diabetes, 19.4% were found to be at risk of foot ulcer (defined by the presence of any one of neuropathy, PVD or history of foot ulceration). The greatest risk was

evident in those with a diabetes duration of 20 years or more (< 5 years 16.4%, 5–9 years 20.4%, 10–19 years 26.1% and ≥ 20 years 46.5%).

Based on Australian National Diabetes Information Audit and Benchmarking (ANDIAB) data for 2000, the prevalence of current foot ulcers among adult patients attending diabetes clinics was 3.0%. In addition, 6.2% of patients had a past history of foot ulcers. The vast majority (86.5%) of patients with a current foot ulcer had a past history of foot ulceration (NADC 2000). Also indicative of potential foot problems, peripheral neuropathy, PVD and foot deformity were recorded for a total of 24.2%, 12.6% and 5.6% of adult patients, respectively.

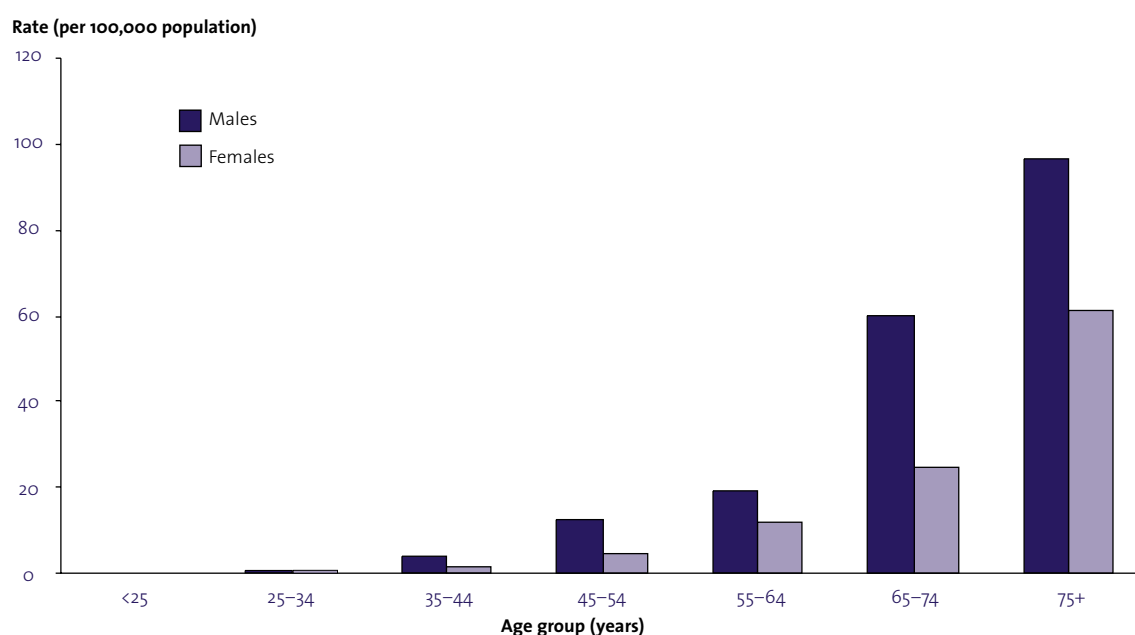
Among patients registered in the National Divisions Diabetes Program (NDDP) Data Collation Project during 1999–00, 24.9% were identified with foot risk (indicated by a history of foot problems and/or presence of peripheral neuropathy, PVD or foot deformity on examination).

### Lower limb amputation

Absence of limbs was reported by 2.1% of respondents with self-reported diabetes during the 1995 National Health Survey. This was more than four times the rate reported among persons without diabetes, despite the higher mortality of amputees with diabetes. The proportion of males with diabetes reporting absence of limbs was 3.3% compared with 0.97% of females with diabetes.

In 2000, the incidence of lower limb amputation among ANDIAB patients was estimated to be 0.8%. Further analysis revealed that 90% of patients undergoing a lower limb amputation in the previous 12 months had a past history of foot ulceration (NADC 2000).

**Figure 4.4:** Hospitalisations for lower limb ulcer as principal and diabetes as additional diagnosis, 1999–00



Source: AIHW National Hospital Morbidity Database.



## Hospitalisations

The majority of foot ulcers are treated in outpatient settings, which limits effective surveillance of the problem (DHAC & AIHW 1999). However, some information on the extent of lower limb ulcer and amputation is available from hospital separation data.

### Lower limb ulcer

In 1999–00 there were 1,859 admissions to hospital where lower limb ulcer was the principal diagnosis and diabetes was an additional diagnosis. Males were almost twice as likely to be hospitalised for lower limb ulcer as the principal and diabetes as an additional diagnosis than females. Hospital use for lower limb ulcer increased with age, with more than 70% of such cases being aged 65 and over in 1999–00 (Figure 4.4).

For people admitted to hospital with lower limb ulcer as principal and diabetes as an additional diagnosis in 1999–00, the average length of stay was 13.4 days. Females tended to have a much longer average length of stay than males, 16.5 days compared with 11.4 days.

### Lower extremity amputation

During 1999–00 a total of 3,404 amputations of lower extremities and/or limbs were performed for a diagnosis of diabetes. Males with diabetes were more than twice as likely to have a lower extremity amputation than females. Hospital use for diabetes-related amputation increases with age. For example, although men and women aged 65 and over represent only 12% of the total population, they accounted for almost 65% of hospitalisations for diabetes-related lower extremity and limb amputations in 1999–00.

Those hospitalised for lower extremity amputation tended to stay considerably longer than those hospitalised for other diabetes-related conditions. The average length of stay in hospital for diabetes-related lower extremity amputation was 27.5 days. Females tended to have a shorter average length of stay than males, 25.4 days compared with 28.4 days.

## Main data sources

2000 Australian National Diabetes Information Audit and Benchmarking (ANDIAB) (National Association of Diabetes Centres).

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

1999–2000 National Divisions Diabetes Program (NDDP) Data Collation Project.

1995 National Health Survey (Australian Bureau of Statistics).

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

National Mortality Database (Australian Institute of Health and Welfare).

## References and further reading

Campbell LV, Graham AR, Kidd RM, Molloy HF, O'Rourke SR & Colagiuri S 2000. The lower limb in people with diabetes. Position statement of the Australian Diabetes Society. *Medical Journal of Australia* 173:369–71.

Carter S, Bonney M, Flack J, Burns J, Powell Davies PG & Harris MF 2000. National Divisions Diabetes Program Data Collation Project. Volume 5: Divisions of General Practice—Diabetes profiles. Quality of care and health outcomes—collated CARDIAB data. Sydney: Centre for General Practice Integration Studies, School of Community Medicine, University of New South Wales.

Colman PG & Beischer AD 2000. Lower-limb amputation and diabetes: the key is prevention. *Medical Journal of Australia* 173:341–342.

DHAC & AIHW (Commonwealth Department of Health and Aged Care & Australian Institute of Health and Welfare) 1999. National Health Priority Areas report: diabetes mellitus 1998. AIHW Cat. No. PHW 10. Canberra: DHAC & AIHW.



Diabetes Australia 2002. Evidence based guidelines. Identification and management of diabetic foot disease. Viewed 12 April 2002, <<http://www.diabetesaustralia.com.au/docs/Foot-Part-6.pdf>>.

NADC (National Association of Diabetes Centres) 2000. ANDIAB 2000. Australian National Diabetes Information Audit & Benchmarking. Canberra: National Association of Diabetes Centres.

Oyibo SO, Dang CN & Boulton AJM 2002. Diagnosis and management of diabetic neuropathy. *Topical Endocrinology* 19:10–13.

Payne CB 2000. Diabetes-related lower-limb amputations in Australia. *Medical Journal of Australia* 173:352–4.

Williams G & Pickup JC 1999. *Handbook of diabetes*. 2nd edn. Oxford: Blackwell Science, 159–64.

## Oral complications

Diabetes can lead to oral complications. Diabetes may manifest initially with oral symptoms other than thirst. For instance, burning tongue, gum bleeding and excessive salivation have been found in undiagnosed people with diabetes and resolved on treatment to improve glycaemic control. Oral complications are very uncommon in westernised societies but are more common in underdeveloped countries or in lower socioeconomic groups, especially where there is poor hygiene and delayed diagnosis of diabetes.

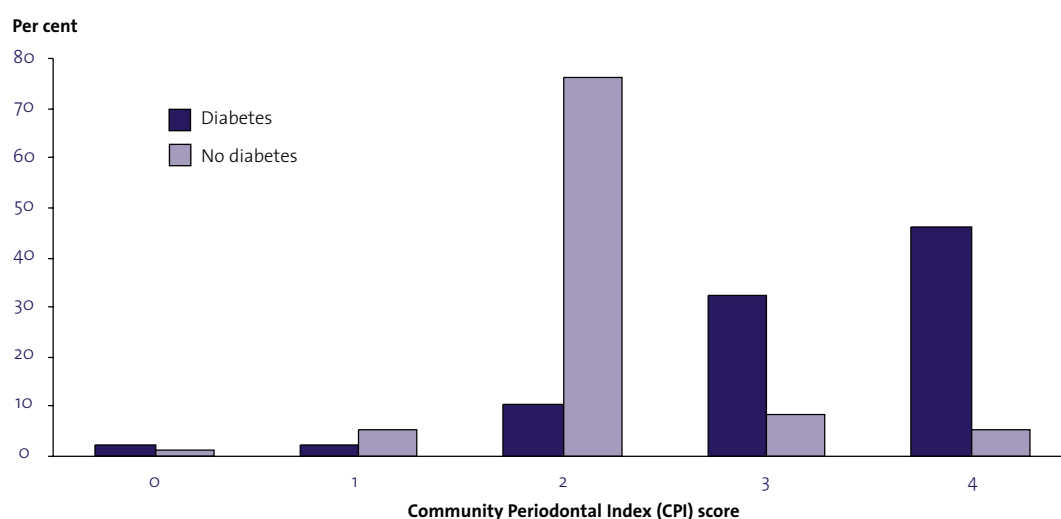
### Periodontal disease

There is growing acceptance that diabetes is associated with increased occurrence, extent and severity of periodontitis (inflammation of the tissues supporting the teeth). The risk is independent of whether the diabetes is Type 1 or Type 2. Some researchers point to a two-way connection between diabetes and periodontal disease, proposing that not only are patients with diabetes more prone to periodontal disease, but the presence of periodontal disease affects control of blood glucose.

International studies have shown that people with Type 2 diabetes are about three times more likely to have destructive periodontal disease than those without diabetes. Among Indigenous Australians in remote communities, having diabetes is significantly associated with a maximum score in the Community Periodontal Index (CPI) (Figure 4.5). CPI is a measure of periodontal disease suggested for use by the World Health Organization that combines three indicators of periodontal health: gum bleeding, calcium deposits on teeth and depth of periodontal pockets, with a score of 4 indicating worst health.

Diabetes can affect the tissues supporting the teeth (periodontium) and the treatment of periodontal diseases. Patients with long-term poor control of diabetes have increased extent and severity of periodontal disease, whereas those who maintain good metabolic control have minimal periodontal problems. Integrated medical and dental management of these conditions is essential for the general health and quality of life of patients. Treatment of periodontal infections with systemic antibiotics can contribute to the control of diabetes.

**Figure 4.5:** Community Periodontal Index score by diabetes status in remote Indigenous Australians, 2000



Source: Dental Statistics Data Collection.

## Tooth loss

Indigenous Australians with diabetes in remote areas of Australia have significantly more missing teeth than those without diabetes, indicating that periodontal disease associated with diabetes may have contributed to tooth loss (Figure 4.6). Missing teeth in older Indigenous Australians are also associated with high rates of diabetes and advanced periodontal disease.

## Other oral problems

Caries (tooth decay) in the crowns of teeth appear to be more frequent in adults with poor control of insulin-dependent diabetes. Oral infections other than dental caries and periodontal disease are often more severe in people with diabetes. Examples of these are life-threatening deep neck infections and fatal ulcers of the palate.

## Risk factors

Poor oral hygiene, poor control of blood glucose levels, smoking and inadequate nutrition increase the risk of oral complications in people with diabetes.

## Main data source

Dental Statistics Data Collection (Australian Institute of Health and Welfare).

## References and further reading

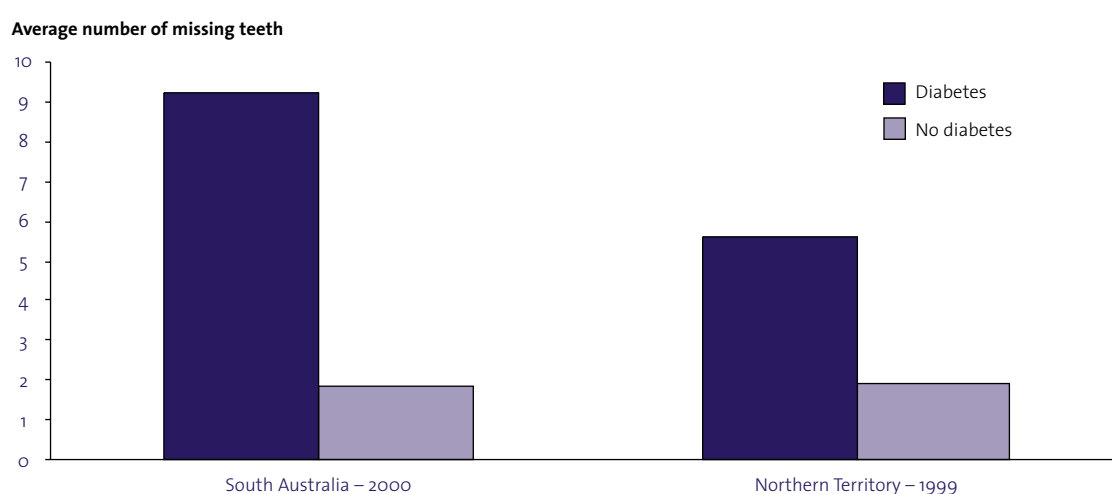
Fenesy KE 1998. Periodontal disease: an overview for physicians. *Mount Sinai Journal of Medicine* 65 (5–6):362–9.

National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health 1995. *Diabetes in America*. 2nd edn. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health.

USDHHS (United States Department of Health and Human Services) 2000. *Oral health in America: a report of the Surgeon General*. Rockville, MD: National Institute of Dental and Craniofacial Research, National Institutes of Health, USDHHS.

WHO (World Health Organization) 1997. *Oral health surveys—basic methods*. Geneva: WHO.

**Figure 4.6:** Missing teeth by diabetes status in remote Indigenous Australians, 1999 and 2000



Source: *Dental Statistics Data Collection*.



## Complications in pregnancy

Two forms of maternal diabetes may occur during pregnancy: pre-existing diabetes and gestational diabetes. Pre-existing diabetes represents about 10% of cases of maternal diabetes.

### Effects of maternal diabetes

In women with pre-existing diabetes, glycaemic control worsens during pregnancy and insulin requirements increase in Type 1 diabetes. This is because the pregnancy hormones induce insulin resistance. Maternal diabetes affects the foetus and newborn as well as the mother.

For the mother with diabetes, pregnancy can worsen kidney function in those with established nephropathy (kidney disease). Increased protein in the urine and pregnancy-induced hypertension are three to four times more common in women with pre-existing diabetes than in women without diabetes. Retinopathy may also deteriorate rapidly during gestation. Caesarean delivery is three to four times more frequent in pregnancies involving diabetes.

Pre-existing diabetes can cause major congenital malformations in the foetus, particularly in the first 8 weeks of gestation, when the major organs are forming, as well as spontaneous abortions. Defects include absence of brain, malformations of the spine, skeleton and kidneys, and heart and great blood vessel abnormalities. The malformation rate is related to the degree of hyperglycaemia (about 7–30% in poorly controlled patients) but tight metabolic control before and during early pregnancy can reduce the rate. International studies indicate that spontaneous abortions occur in 7–17% of diabetic pregnancies if diabetes is not well managed. Women with good control do not appear to have increased rates of spontaneous abortions compared with women without diabetes.

The perinatal death rate (stillbirths and newborn deaths within the first week of life) is also increased by 1.5 to 2-fold in pregnancies with pre-existing diabetes compared with those without diabetes, according to international data. The main causes of this are:

- death in the uterus in the third trimester of pregnancy;
- prematurity due to a high incidence of spontaneous premature labour and of elective premature delivery in an attempt to avoid death in the uterus late in the pregnancy;
- low birth weight due to foetal growth retardation in the uterus in some cases where the mother has diabetic nephropathy;
- congenital malformations; and
- birth trauma due to a high incidence of excessively large babies.

International studies show that large babies occur at a rate of 30% in diabetic pregnancies compared with 10% in non-diabetic pregnancies. Data from Queensland indicate that 12.3% of all babies born in 1997 weighed 4,000 g or more, compared with 17.7% when mothers had gestational diabetes and 17.9% when mothers had pre-existing diabetes.

Accelerated foetal growth, leading to large-for-gestational-age babies, is due to increased delivery of glucose and other nutrients from mother to foetus. This stimulates the pancreas in the foetus to produce extra insulin, which promotes abdominal fat deposition, growth of the skeleton and large size organs. Complications for these babies include birth trauma, and jaundice, hypoglycaemia and low levels of calcium in the newborn. Poor glycaemic control also leads to impaired lubrication of the lungs and respiratory distress in the newborn. These babies may also have a long-term greater risk of obesity.

Women with gestational diabetes may have a greater risk of foetal perinatal death and disease, and are themselves at increased risk of developing Type 2 diabetes and perhaps cardiovascular disease later in life. Infants of women who develop gestational diabetes may have newborn hypoglycaemia, jaundice, respiratory distress and birth trauma resulting from being excessively large babies, much the same as those of women with pre-existing diabetes.

### How many Australian women are affected by maternal diabetes?

Although there are no national figures on how many Australian women are affected by maternal diabetes, data from States and Territories indicate that gestational diabetes occurs in 3.0–4.5% of all women giving birth and pre-existing diabetes in 0.4%.

Reliable data on the prevalence of gestational diabetes among Aboriginal and Torres Strait Islander mothers are scarce. There are varying estimates of prevalence

ranging from less than 1% to up to 20%. However, de Courten et al. (1998) suggest that Indigenous women experience a higher risk of gestational diabetes than non-Indigenous women.

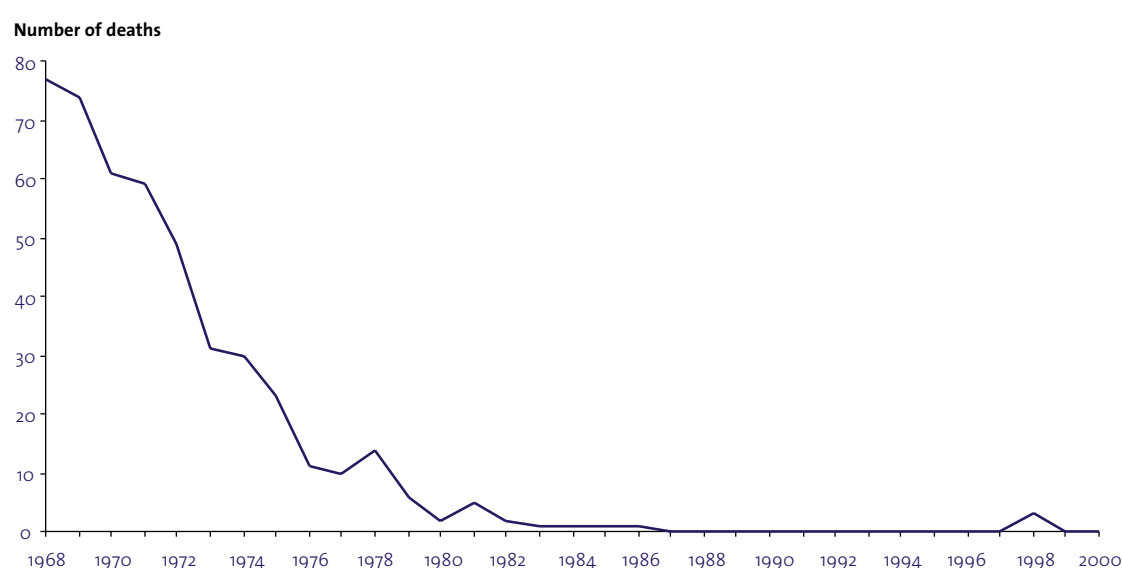
### Hospitalisations

In Australia, in 1999–00 there were 13,901 hospitalisations for gestational diabetes as a principal or additional diagnosis, and 2,330 hospitalisations for pre-existing diabetes in pregnancy as a principal or additional diagnosis.

### Infant deaths

The frequency of foetal disease and mortality in diabetic pregnancies has been dropping over the past few decades, possibly as a result of better medical care and control of diabetes in the mother (Figure 4.7).

**Figure 4.7:** Deaths among infants of a diabetic mother, 1968–2000



Source: AIHW Mortality Database.

## **Main data source**

States and Territories Perinatal Data Collections.

National Mortality Database (Australian Institute of Health and Welfare).

## **References and further reading**

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.

Edwards CRW, Bouchier IAD, Haslett C & Chilvers ER (eds.) 1998. Davidson's principles and practice of medicine. 17th edn. Edinburgh: Churchill Livingstone.

National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health 1995. Diabetes in America. 2nd edn. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health.

Williams G & Pickup JC 1999. Handbook of diabetes. 2nd edn. Oxford: Blackwell Science, 191–6.

# Management and care

5

Health professionals

Services for people with diabetes

Hospitalisations

Medication use

Pathology and other tests

diabetes  
australian facts 2002

Australian Institute of Health and Welfare

## Health professionals

General practitioners (GPs) have a natural role in managing and monitoring diabetes and in coordinating the services that are needed because the condition and its complications affect several parts of the body. Patients and their carers also need education and support. Thus, a range of health professionals may be involved.

### Visits to GPs

In 2000–01 GPs managed diabetes problems (excluding gestational diabetes) at a rate of 2.8 per 100 encounters, representing 1.9% of all problems. This equates to almost 2.9 million consultations for diabetes each year and makes diabetes the seventh most common problem managed in general practice.

GPs managed almost 32% of diabetes problems with clinical treatment and treated 55% of problems without medications. This included mainly providing advice on nutrition or weight, glucose tests done in the GP's rooms, and advice on treatment. Diabetes problems were referred relatively frequently to other

health professionals. About 2% of diabetes problems were referred to medical specialists (mainly ophthalmologists and endocrinologists) and 3% to allied health professionals (dietitians, diabetes education, diabetes clinics, podiatrists). GPs requested pathology tests in 26% of diabetes encounters, at a rate of 227 pathology orders per 100 diabetes problems with pathology. This means that when a diabetes problem required pathology tests, 2.3 tests were ordered on average.

### Other health professionals

Diabetes complications may affect a number of the body's organs, necessitating treatment by specialists in areas such as endocrinology, cardiology, nephrology, obstetrics and ophthalmology.

Information from the 1995 National Health Survey indicates that people with diabetes were 2.8 times more likely than people without diabetes to have visited a specialist in the 2 weeks before the survey; 10.8% of people with diabetes visited a specialist, compared with 3.9% of those without diabetes.

#### Box 5.1: National Integrated Diabetes Program

*In 2001 the Federal Government introduced the National Integrated Diabetes Program, which provides funding to ensure a national approach to improving the prevention, earlier detection and management of people with diabetes. The program includes:*

- *incentives for GPs to develop systems of care to help detect diabetes earlier and manage it better, including monitoring blood pressure, HbA<sub>1c</sub>, blood lipids, eyes, feet, overweight and microalbuminuria;*
- *infrastructure and support for Divisions of General Practice to work with GPs and other health professionals to remove barriers to better care of people with diabetes, such as inadequate access to community services and multidisciplinary care;*
- *information, education resources and tools for people at risk of or with diabetes, and their families, to learn self-care skills and enable them to better manage the condition; and*
- *support for changes in the practices of health professionals that improve the health outcomes of their patients with diabetes.*



Information is also available on referrals made by general practitioners to specialists and other health care professionals (Table 5.1). People with Type 1 diabetes were most frequently referred to endocrinologists, while those with Type 2 diabetes were referred most frequently to ophthalmologists.

**Table 5.1:** GP referrals for Type 1 and Type 2 diabetes, 1998–99

Referrals	Type 1 diabetes Rate <sup>(a)</sup>	Type 2 diabetes Rate <sup>(a)</sup>
Endocrinologist	3.8	0.8
Hospital admission	1.4	0.5
Ophthalmologist	1.2	1.9
Paediatrician	0.5	—
Diabetes clinic	0.4	0.8
Dietitian/nutrition	n.a.	1.2
Diabetes education	n.a.	0.9
Physician	n.a.	0.4
Podiatrist/chiroprapist	n.a.	0.4
Specialist	n.a.	0.2
Optometrist	n.a.	0.1

(a) Rate per 100 diabetes problems.

Source: AIHW: Senes and Britt 2001.

In addition to the care provided by medical specialists, people with diabetes may also seek the advice of diabetes educators, nutritionists and podiatrists. The services of physiotherapists, chiropractors, opticians, naturopaths, osteopaths, acupuncturists, herbalists, psychologists and chemists may also be used.

Results from the 1995 National Health Survey indicate that people with diabetes were more likely than people without diabetes to have sought advice from other health professionals (16.7% reported such a consultation, compared with 9.8% of people without diabetes).

## Main data sources

1998–2000 Bettering the Evaluation and Care of Health Study (BEACH) (University of Sydney & Australian Institute of Health and Welfare).

1995 National Health Survey (Australian Bureau of Statistics).

## References and further reading

AIHW (Australian Institute of Health and Welfare): Senes S & Britt H 2001. A general practice view of cardiovascular disease and diabetes in Australia. Cardiovascular Disease Series No. 18. AIHW Cat. No. CVD 17. Canberra: AIHW.

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.



## Services for people with diabetes

Organisations in Australia that provide services and support to people with diabetes and coordinate diabetes management include consumer, professional, research and education organisations. A number of agencies or programs central to the provision of services for diabetes management are described below. In addition to these organisations there are numerous others that are crucial to the provision of support and care for people with diabetes in Australia.

### Diabetes Australia

Diabetes Australia is a not-for-profit organisation offering a range of advocacy and support services to people with diabetes and their carers. A federation of eight State and Territory member organisations, the diabetes professional organisations and two foundations, Diabetes Australia has a significant involvement in research and in the development of national policies on diabetes care.

The National Diabetes Services Scheme (NDSS) provides important support for many people with diabetes. The NDSS is a Commonwealth government program that provides products for the self-management of diabetes, such as blood and urine testing strips, syringes and needles for special injection systems, at subsidised prices. Diabetes Australia has administered the NDSS for the Commonwealth since it was introduced in 1987. As at 7 March 2002, 526,631 people with diabetes were registered for NDSS benefits; 169,585 (32%) of these required insulin. The NDSS distributed more than 2.2 million packets of test strips and almost 430,000 boxes of syringes and pen needles during 2000–01.

From 2002, the NDSS will also provide a range of information and education services to people with diabetes. A variety of electronic and interpersonal communication strategies will be used to deliver programs to communities and individuals throughout Australia.

### Diabetes centres and educators

Diabetes centres, often referred to as diabetes ambulatory care centres, provide services such as diabetes education, nutrition advice and complications assessment to adults and children. As well as clinical management of the disease, centres generally aim to improve personal management of diabetes to minimise the effect of diabetes on daily living. Most patients attending diabetes centres are referred by general practitioners (GPs) to receive specialist assessment and treatment, generally these are people whose diabetes is less likely to be managed well.

The staff in most diabetes centres include an endocrinologist, diabetes nurse educators, dietitians and podiatrists. Many centres also provide training in diabetes care to other health professionals, and may conduct research into medical or social aspects of diabetes.

In 2001, 71 diabetes centres were members of the National Association of Diabetes Centres (NADC), with an estimated total of 80 centres throughout Australia. The NADC promotes effective health care practice for people with diabetes. Over the past few years a number of the centres in the collective have participated in data collection, enabling assessment and review of diabetes management. Some of these data have been reported in other sections of this report.

### National Divisions Diabetes Program

The National Divisions Diabetes Program (NDDP) is a coordinated national approach to diabetes care in Australian general practice. This program was set up to help national diabetes organisations and Divisions of General Practice share information on policy, best practice, quality assurance and other matters relating to diabetes. The NDDP represents the Divisions of General Practice and GPs on national diabetes

programs, and aims to improve awareness of the importance of the divisions and general practice in maintaining quality health care for people with diabetes.

As part of the NDDP Data Collation Project, 63 of 123 Divisions of General Practice reported having a diabetes program in 1999–00. At least 27% of GPs nationally participated in some aspect of a diabetes program (the 2000–01 annual survey of divisions reported that 84% of divisions had a diabetes program or activity).

These programs reported features such as register/recall systems (48%), GP education (56%), patient education (32%) and shared care (32%). Divisions also reported on services in their area, program highlights and barriers, advice to other divisions and future plans.

### Juvenile Diabetes Research Foundation Australia

The Juvenile Diabetes Research Foundation (JDRF) is the world's largest international non-profit, non-government contributor to diabetes research. Its mission is to find a cure for diabetes and its complications through the support of medical research. The JDRF also provides support for those families affected by diabetes.

The JDRF was founded in 1970 by the parents of children with juvenile diabetes. Volunteers help define research priorities, select research grant recipients, lead advocacy efforts and provide guidance to overall operations. In 2002, over \$10 million of funding helps support Australian projects on:

- restoration of normal blood glucose levels
- reversal of diabetes-related complications
- prevention of diabetes and its recurrence.

### References and further reading

Centre for General Practice Integration Studies 2002. National Divisions Diabetes Program. Viewed 17 April 2002, <<http://www.commed.unsw.edu.au/cgpris/>>.

Diabetes Australia 2002. Diabetes Australia National. Viewed 20 May 2002, <[http://www.diabetesaustralia.com.au/da\\_national.htm](http://www.diabetesaustralia.com.au/da_national.htm)>.

Effective Healthcare Australia & Australian Centre for Diabetes Strategies 2000. A plan for the dissemination and implementation of national evidence based guidelines for the prevention and management of Type 2 diabetes. Viewed 8 March 2002, <<http://www.diabetesaustralia.com.au/submission-documents.htm>>.

Juvenile Diabetes Research Foundation (JDRF) 2002. About JDRF. Viewed 1 May 2002, <[http://www.jdrf.org.au/about\\_jdrf/aboutjdrf.html](http://www.jdrf.org.au/about_jdrf/aboutjdrf.html)>.

## Hospitalisations

In 1999–00, diabetes as a principal or additional diagnosis occurred in 336,976 hospitalisations, that is 5.7% of all hospital separations. Diabetes as a principal diagnosis accounted for 24,417 hospitalisations, or 0.4% of all hospital separations. However, it is important to note that often it is the condition responsible for the hospitalisation, not diabetes, that is recorded as the principal diagnosis, even when it is a complication of diabetes. Diabetes is more frequently recorded as an additional diagnosis, particularly when it is associated with coronary heart disease, stroke or kidney disease. The most frequent primary diagnosis with diabetes as an additional diagnosis was unstable angina with 11,773 hospitalisations.

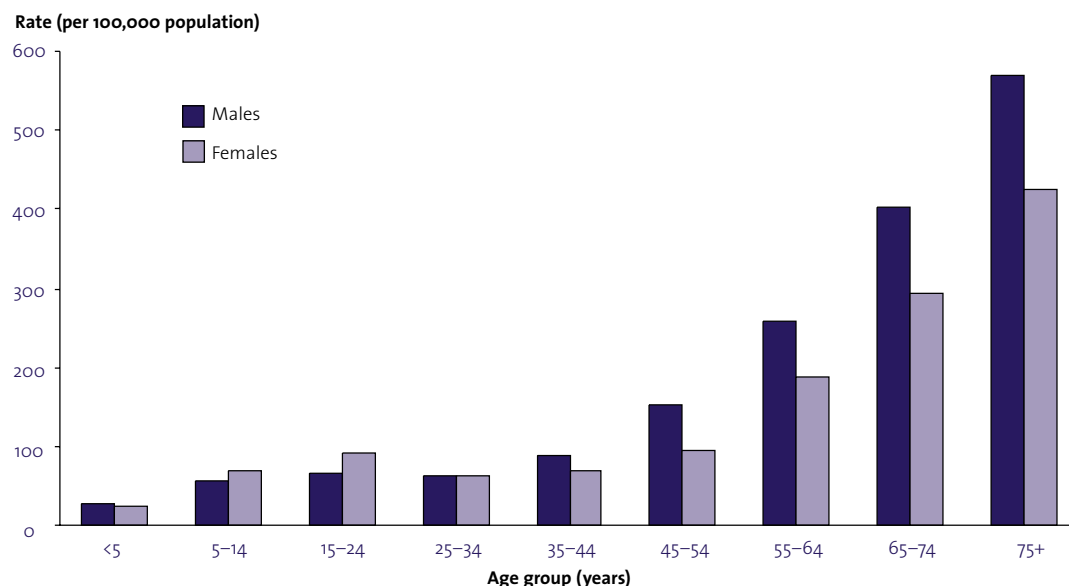
### Age and sex

Males are more likely to be admitted to hospital for diabetes than females. Figure 5.1 shows that hospital use for a primary or additional diagnosis of diabetes increases with age.

### Length of stay in hospital

The average length of stay in hospital when diabetes was the principal diagnosis was 6.6 days in 1999–00, compared with 3.8 days for people without diabetes as a principal diagnosis. When diabetes as an additional diagnosis was considered, the average length of stay increased to 7.0 days compared with 3.6 days for people without diabetes.

**Figure 5.1:** Hospitalisations for diabetes, 1999–00



Note: Includes principal and additional diagnoses of diabetes.

Source: AIHW National Hospital Morbidity Database.

Males with diabetes as a principal diagnosis tended to stay in hospital longer than females: on average, 6.8 days compared with 6.4 days.

Same-day separations for a principal diagnosis of diabetes represented 19% of the total.

### Deaths in hospital

In 1999–00, there were 335 hospitalisations for diabetes as a principal diagnosis where the patient died in hospital (1.4% of all diabetes hospitalisations). There were no significant differences between males and females in this respect.

In the same year, there were 10,280 hospital deaths when diabetes was either the principal or additional diagnosis (3.1% of hospitalisations with a principal or additional diagnosis of diabetes).

### Main data source

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

## Medication use

This section gives an overview of the use of prescription medicines for diabetes in Australia. According to the 1995 National Health Survey, 57% of adults who reported having diabetes were being treated for the condition, 18% were using insulin and 41% were on tablets to control their blood glucose levels. The survey also showed that adults with diabetes were more likely than those without diabetes to use certain medications such as aspirin, frusemide (a diuretic), ACE-inhibitors and digoxin but the problem for which the drugs were taken was not recorded.

The data in Figure 5.2 refer to the use of prescription drugs in the community only (non-public hospital). Medication use is expressed in the World Health Organization approved measurement unit—defined daily doses (DDDs) per 1,000 population per day (DDD/1,000/day). This is based on the assumed average adult dose per day of a drug taken for its main purpose. The DDD enables valid comparisons between drugs independently of differences in price, preparation and quantity per prescription.

### Insulins and insulin analogues

Insulin helps the body use or store the glucose it gets from food. People whose pancreas does not make insulin (Type 1 diabetes) need insulin injections to survive. Some people with Type 2 diabetes also require insulin injections to improve diabetes control. Giving suitable doses of insulin to people with diabetes temporarily restores their ability to process carbohydrates, fats and proteins, to store glycogen in the liver, and to convert glucose to fat.

Insulin is available in several types that differ in how soon the insulin starts working (onset), when it works most (peak time) and how long it lasts in the body (duration).

Fast-acting insulin reaches the blood within 15 minutes of injection, peaks 30–90 minutes later and may last for up to 5 hours. Human insulin was the fast-

acting type of insulin dispensed most commonly in 1998 (1.9 DDD/1,000/day), followed by insulin lispro (0.7 DDD/1,000/day).

Intermediate-acting insulin reaches the blood 2–6 hours after injection, peaks 4–14 hours later and stays in the blood for about 14–20 hours. Human intermediate-acting insulin was dispensed at a rate of 3.1 DDD/1,000/day in 1998.

Long-acting insulin takes 6–14 hours to start working. It has no peak or a very small peak 10–16 hours after injection and stays in the blood between 20 and 24 hours. Long-acting insulin was the type dispensed least frequently (0.2 DDD/1,000/day).

Some insulins come mixed together to make it easier to inject two kinds of insulin at the same time. Human intermediate-acting insulin combined with fast-acting insulin was the most common insulin dispensed overall in 1998 (3.1 DDD/1,000/day).

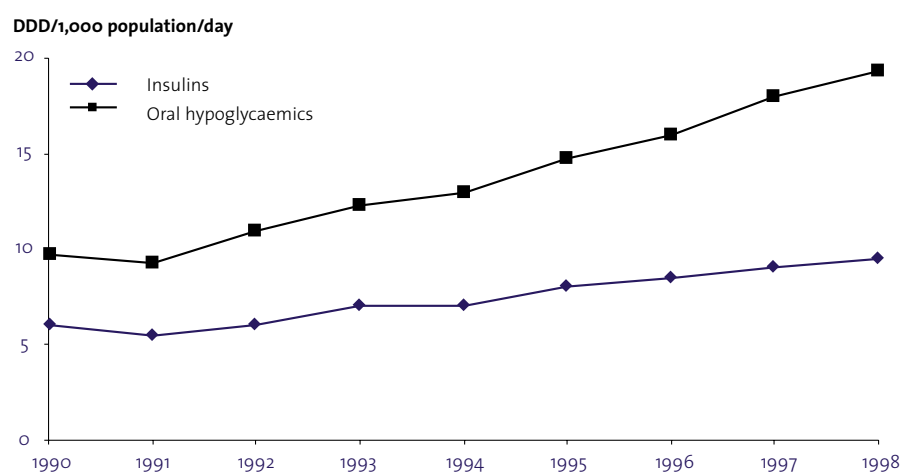
The use of insulins and oral blood glucose lowering drugs has increased during the 1990s, reflecting the increase in the number of people being diagnosed with diabetes.

### Oral blood glucose lowering drugs

Only people with Type 2 diabetes can benefit from the use of pills to treat their diabetes. There are several classes of oral drugs that can lower blood glucose. They work in different ways so they may be used in combination if needed. Figure 5.3 shows community use of oral blood glucose lowering drugs from 1990–1998.

Sulphonylurea drugs stimulate the beta cells in the pancreas to release more insulin. Chlorpropamide, glipizide, glibenclamide, gliclazide and tolbutamide are members of this class. Gliclazide was the most commonly dispensed sulphonylurea in 1998 (6.2 DDD/1,000/day), followed by glibenclamide (4.1 DDD/1,000/day).

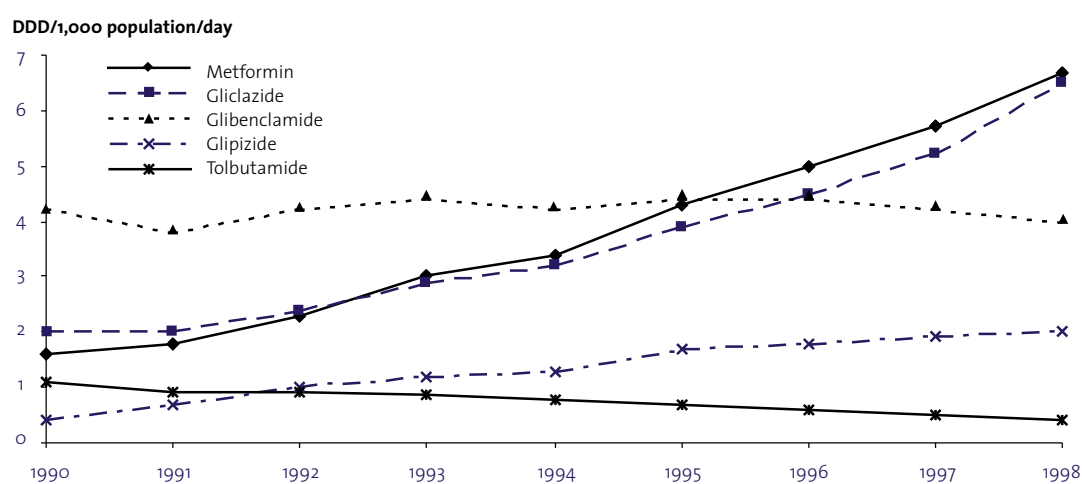
**Figure 5.2:** Community use of antidiabetic drugs, 1990–98



Note: DDD = defined daily dose.

Source: Department of Health and Aged Care 1999.

**Figure 5.3:** Community use of oral blood glucose lowering drugs, 1990–98



Note: DDD = defined daily dose.

Source: Department of Health and Aged Care 1999.



Biguanides lower blood glucose by suppressing glucose production in the liver. Metformin belongs to this class of drugs. It has become increasingly popular in the 1990s and in 1998 it was the most frequently used oral hypoglycaemic drug overall (6.6 DDD/1,000/day). Metformin is among the top 30 most commonly prescribed medications in general practice and in 2000 it accounted for 0.9% of all prescriptions issued by general practitioners.

Alpha glucosidase inhibitors help the body lower blood glucose by blocking the gut enzymes that break down starches (such as bread, potatoes and pasta) and certain sugars into glucose. Their action slows the rise in blood glucose levels after a meal. Acarbose is a member of this class and was dispensed at a rate of 0.1 DDD/1,000/day in 1998.

Thiazolidinedione antidiabetic agents lower blood glucose by improving cell response to insulin. Rosiglitazone and pioglitazone belong in this class. There are no figures on the use of these drugs in Australia because they are not listed on the Pharmaceutical Benefits Scheme.

## Main data sources

1998–2000 Bettering the Evaluation and Care of Health Study (BEACH) (University of Sydney & Australian Institute of Health and Welfare).

1995 National Health Survey (Australian Bureau of Statistics).

## References and further reading

2000 MIMS Annual. 24th edn, June 2000. St Leonards, NSW: MIMS Australia.

Australian medicines handbook 1998. 1st edn. Adelaide: Australian Medicines Handbook.

DHAC (Commonwealth Department of Health and Aged Care) 1999. Australian statistics on medicines 1998. Canberra: AGPS.



## Pathology and other tests

There are a variety of pathology tests used in the diagnosis and management of diabetes. The more common tests include:

- glucose tolerance test, which is a diagnostic test to assess absorption of glucose after a dose is given;
- glycosylated haemoglobin (HbA<sub>1c</sub>) and fructosamine, which monitor glucose control;
- microalbuminuria, which tests for amounts of protein (albumin) in the urine; and
- blood lipids tests, which include total cholesterol, triglycerides and HDL cholesterol.

### Pathology tests processed by Medicare for people with diabetes

This section reports the tests undertaken by pathology labs and billed to Medicare. The figures do not include services provided to public patients in public hospitals, diabetes clinics or services that qualify for a benefit under the Department of Veterans' Affairs National Treatment Account.

Pathology tests billed to Medicare are subject to 'coning', that is, pathology companies charge Medicare for the three most expensive tests undertaken even where more tests were undertaken. Where a patient with diabetes is likely to receive multiple tests for monitoring the disease and its complications, the less expensive test may not be recorded in the Medicare data, i.e. it may 'drop' off the billing process due to coning (AIHW: Britt et al. 2001). The number of HbA<sub>1c</sub> tests is likely to be underestimated in the Medicare data for this reason.

In 1999–00, there were 494,611 diabetes patients identified in the Medicare population (3.4%). Of these diabetes patients, 27.0% had an HbA<sub>1c</sub> test in each of the last two 6 months of 1999–00, 18.1% had a microalbumin test in 1999–00, 62.9% had a lipid test in 1999–00 and 70.3% had an eye examination between 1998–99 and 1999–00.

The Health Insurance Commission's Diabetes Clinical Advisory Group has determined minimum testing frequencies for selected tests based on the New South Wales Health Department's *Principles of Diabetes Care and Guidelines for the Clinical Management of Diabetes Mellitus in Adults* (1996) and the National Health and Medical Research Council's *Management of Diabetic Retinopathy Clinical Practice Guidelines* (1997). Minimum testing frequencies reported in Figure 5.4 are:

- HbA<sub>1c</sub>: two tests in the reporting period (one test in each 6 month period)
- blood lipids (total cholesterol, triglycerides and HDL cholesterol): once a year
- microalbumin: once a year
- eye examination: once every 2 years.

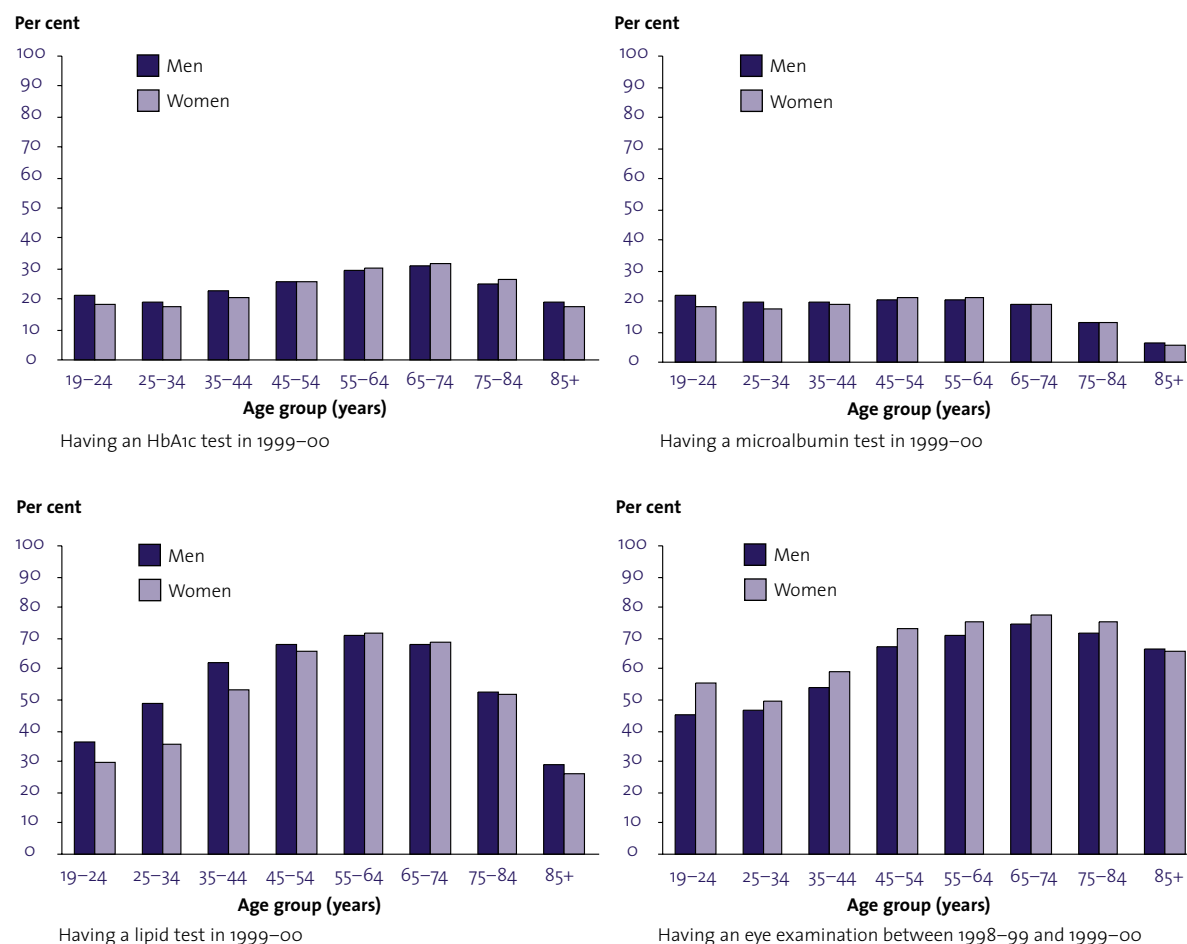
### Pathology tests ordered by GPs

A survey of general practice activity (BEACH) found that, in 1998–99, general practitioners (GPs) ordered pathology tests for Type 1 diabetes problems relatively often (37.3 per 100 problems) compared with the average (17.0 per 100). Tests for HbA<sub>1c</sub>, which monitor glucose control, were the most widely requested (11.9 per 100), followed by glucose tolerance test (7.2 per 100), electrolytes/urea/creatinine (4.2 per 100) and lipids tests (3.8 per 100).

Pathology tests were frequent in the management of Type 2 diabetes, at a rate of 48.5 per 100 problems compared with the average (17.0 per 100). As with Type 1 diabetes, GPs requested tests for HbA<sub>1c</sub> (13.5 per 100), glucose tolerance (12.7 per 100), lipids (6.6 per 100) and electrolytes/urea/creatinine (3.8 per 100) (AIHW: Senes & Britt 2001).

The BEACH data do not capture whether a pathology test ordered by the GP is performed (AIHW: Britt et al. 2001).

**Figure 5.4:** Adults identified with diabetes meeting the minimum testing frequency for selected tests



Source: Health Insurance Commission 2001.

## Pathology tests at diabetes clinics

According to the Australian National Diabetes Information Audit and Benchmarking survey in 2000, 83.2% of patients visiting specialist diabetes clinics had an HbA1c measurement in that year, and 48.5% had a microalbumin (or urinary protein level) recorded. Of the patients attending specialist diabetes clinics, 67.2% had a cholesterol level recorded, 43.4% had an HDL cholesterol level recorded and 64.1% had a triglyceride level recorded. A total of 53.1% of patients were recorded as having seen an ophthalmologist and 17.9% as having seen an optometrist in the last 12 months; 61.9% had seen either an ophthalmologist or an optometrist.

## Main data sources

1998-2000 Bettering the Evaluation and Care of Health Study (BEACH) (University of Sydney & Australian Institute of Health and Welfare).

1998-2000 Australian National Diabetes Information Audit and Benchmarking (National Association of Diabetes Centres).

Health Insurance Commission General Practice Statistics—Diabetes (Health Insurance Commission).

## References and further reading

AIHW (Australian Institute of Health and Welfare):  
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: Senes S & Britt H 2001. A general practice view of cardiovascular disease and diabetes in Australia. Cardiovascular Disease Series No. 18. AIHW Cat. No. CVD 17. Canberra: AIHW.

Colman PG, Thomas DW, Zimmet PZ, Welborn TA, Garcia-Webb P & Moore MP 1999. New classification and criteria for diagnosis of diabetes mellitus. Position statement from the Australian Diabetes Society, New Zealand Society for the Study of Diabetes, Royal College of Pathologists of Australasia and the Australasian Association of Clinical Biochemists. Medical Journal of Australia 170:375–8.

HIC (Health Insurance Commission) 2001. HIC—Professional—Statistics—General Practice Statistics—Diabetes. Viewed 15 November 2001, <<http://www.hic.gov.au/cgi-bin/broker.exe>>.

NADC (National Association of Diabetes Centres) 2000. ANDIAB 2000. Australian National Diabetes Information Audit & Benchmarking. Canberra: National Association of Diabetes Centres.

NHMRC (National Health and Medical Research Council) 1997. Management of diabetic retinopathy. Clinical practice guidelines. Canberra: AGPS.

New South Wales Health Department 1996. The principles of diabetes care and guidelines for the clinical management of diabetes mellitus in adults. North Sydney: NSW Health Department.

Royal College of Pathologists of Australasia 2002. Manual of use and interpretation of pathology tests: 3rd edn. Viewed 27 February 2002, <<http://www.rcpa.edu.au/pathmanu/index.htm>>.



# Impact of diabetes



# 6

Social impact

Mortality

Economic impact

diabetes  
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## Social impact

Diabetes affects the daily lives of not only those who have the disease, but also their friends and family. It affects what people with the disease can eat and drink, their lifestyle and many other areas of their lives. They may need to ensure that either they have quick access to food or they carry food with them at all times. There may be difficulties in obtaining driver's licences as well as getting some jobs. There may also be psychological impacts from being diagnosed with diabetes and from changes to daily life. Friends and family need to adjust to the changes as well, which may affect their own lives and relationships.

There is also the impact and burden of the complications of diabetes. Losing partial or total sight or losing a limb due to amputation may affect a person's ability to participate in social activities and their independence in performing daily activities such as self-care, mobility and communication. Kidney failure means constant trips to the hospital for dialysis or access to a personal dialysis machine, which may consume large amounts of time. However, complications do not have only direct impacts. They can also have psychological impacts, and impacts on the social life and wellbeing of the person. As well, the struggle of learning to cope with the changes may cause stress. The complications may be severe enough to cause the loss of ability to work, which has its own consequences.

### Health perception

People with diabetes are less likely to rate their health as excellent, very good or good compared with people without diabetes. In 1999–2000, among people aged 25 years or over it was estimated that 65.7% of males with diabetes rated their health as excellent, very good or good, compared with 85.9% of males without diabetes. In the same age group, 68.4% of females with diabetes rated their health as excellent, very good or good compared with 87.0% of females without diabetes.

## Disability

Diabetes can result in disability for some people, particularly those who develop complications of the disease. For an individual, a disability may take the form of a functional impairment such as loss of vision, activity limitations such as walking, or participation restrictions such as community involvement. For society, it can have an impact on family, friends and the wider community.

In 1998, it was estimated that almost 64,000 Australians had a disability caused mainly by diabetes. Of these people, 85% were aged 45 or over. For many more people, diabetes may play a role in limiting core activities, although the disease may not be the principal cause. For instance, among those aged 65 or over, it was estimated that about 109,000 Australians with diabetes had limitations on activities such as self-care, mobility and communication. It was estimated that more than 113,000 people aged 65 or over who had diabetes needed assistance with self-care, mobility, communication, health care, housework, meal preparation, house maintenance or transport.

It is estimated that diabetes and its complications were responsible for more than 53,000 years of equivalent 'healthy' life lost to disability (YLD) (4.6% of all YLDs) in 1996 (AIHW: Mathers et al. 1999). The impact of diabetes in terms of chronic disability was higher among males than females—over 29,000 years of 'healthy' life lost to disability (5.1% of all YLDs) estimated for males compared with almost 24,000 years (4.1% of all YLDs) for females in 1996.

### Main data sources

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

1998 Disability, Ageing and Carers Survey (Australian Bureau of Statistics).

## References and further reading

ABS (Australian Bureau of Statistics) 2000. Disability, ageing and carers: disability and long term health conditions, Australia 1998. ABS Cat. No. 4433.0. Canberra: ABS.

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

## Mortality

Diabetes is the sixth leading underlying cause of death among Australians. However, often the condition directly responsible for the death, not diabetes, is regarded as the underlying cause of death, even when it is a complication of diabetes. Diabetes is twice as likely to be regarded as an associated cause of death, rather than the underlying cause, with diabetes being listed as an associated cause of 7,124 deaths.

### Diabetes as the underlying cause of death

In 2000, diabetes was listed as the underlying cause of 3,006 deaths (2.3% of all deaths). Of these deaths, 33.8% (1,015 deaths) were due to Type 2 diabetes, 11.5% (346 deaths) were due to Type 1 diabetes and the rest were due to an unknown or unspecified type of diabetes.

### Sex and age

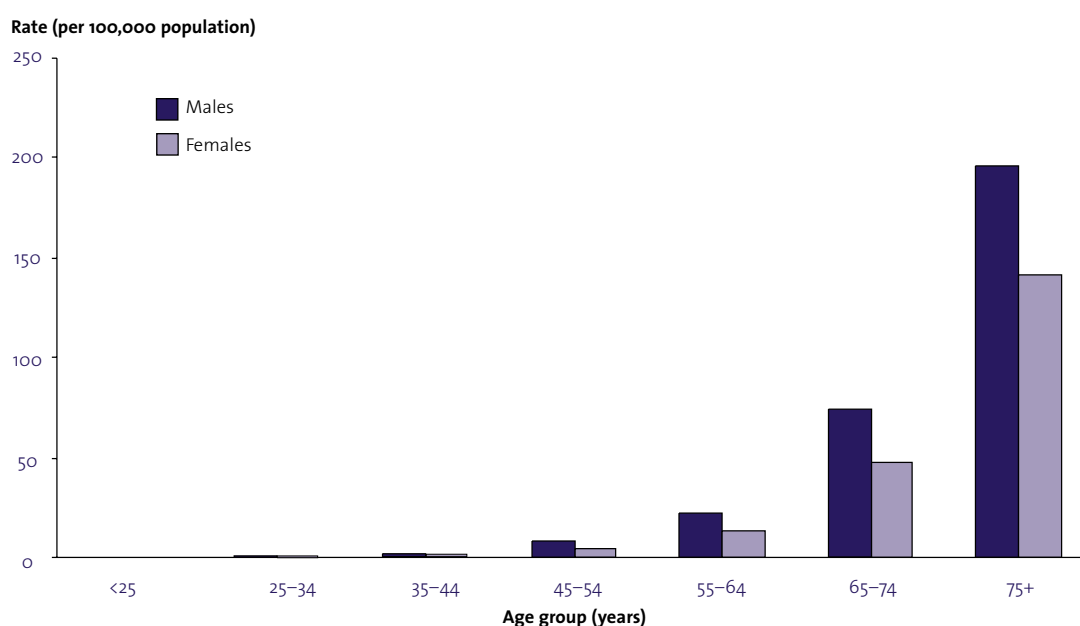
In 2000, Australian males were more than one-and-a-half times as likely to die from diabetes, as the underlying cause of death, as Australian females (Figure 6.1).

Diabetes mortality increases dramatically with age, with almost 90% of deaths where diabetes is the underlying cause occurring in those aged 60 years and over in 2000.

### Trends

The death rate for diabetes (as an underlying cause of death) has shown an increase for males, with an average annual increase of 1.2% between 1989 and 2000. For females there has been no consistent trend in the death rate for diabetes over this same period.

**Figure 6.1:** Death rates from diabetes as the underlying cause, 2000



Source: AIHW National Mortality Database.



## States and Territories

Death rates from diabetes as the underlying cause of death during 1998–00 were highest in the Northern Territory and lowest in the Australian Capital Territory and New South Wales. Among the remaining States and Territories there were no significant differences in death rates from diabetes.

## Urban, rural and remote areas

Death rates from diabetes as the underlying cause of death increase with increasing rurality. During 1998–00, death rates were highest in remote areas, followed by rural areas, with death rates lowest in urban areas for both males and females.

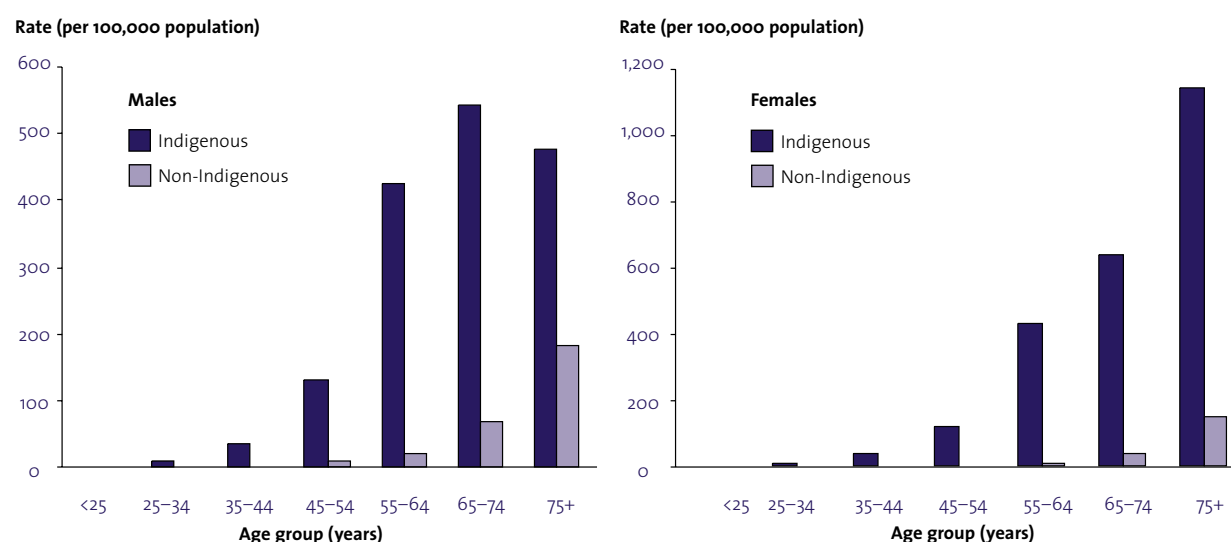
During 1998–00, male Aboriginal and Torres Strait Islander deaths from diabetes as the underlying cause of death accounted for 60% of deaths from diabetes in

remote areas in Queensland, Western Australia, South Australia and the Northern Territory. In comparison, only 3% of male deaths from diabetes in urban areas were Aboriginal and Torres Strait Islander. Similarly, among females, Indigenous deaths were 71% of deaths from diabetes in remote areas compared with 5% in urban areas.

## Aboriginal and Torres Strait Islander People

During 1998–00, Aboriginal and Torres Strait Islander males died from diabetes as the underlying cause of death at more than seven times the rate of non-Indigenous males in Queensland, Western Australia, South Australia and the Northern Territory. The difference in death rates is even larger for females where, during 1998–00, Indigenous females were more than 14 times as likely to die from diabetes as the underlying cause of death as non-Indigenous females (Figure 6.2).

**Figure 6.2:** Death rates from diabetes as an underlying cause, 1998–00



Note: Includes deaths only for Queensland, Western Australia, South Australia and the Northern Territory.

Source: AIHW National Mortality Database.

Diabetes mortality increases with age in both the Indigenous and non-Indigenous Australian populations. However, mortality from diabetes starts at an earlier age among Indigenous Australians. In 1998–00, more than 45% of deaths with diabetes as an underlying cause occurred in those aged less than 60 years in the Indigenous population, compared with 10% among the non-Indigenous population.

### Culturally and linguistically diverse background

During 2000, males from culturally and linguistically diverse backgrounds were almost 30% more likely to die from diabetes as the underlying cause of death than other males. The difference in death rates is larger for females where, during 2000, females from culturally and linguistically diverse backgrounds were 50% more likely to die from diabetes as the underlying cause of death than other females.

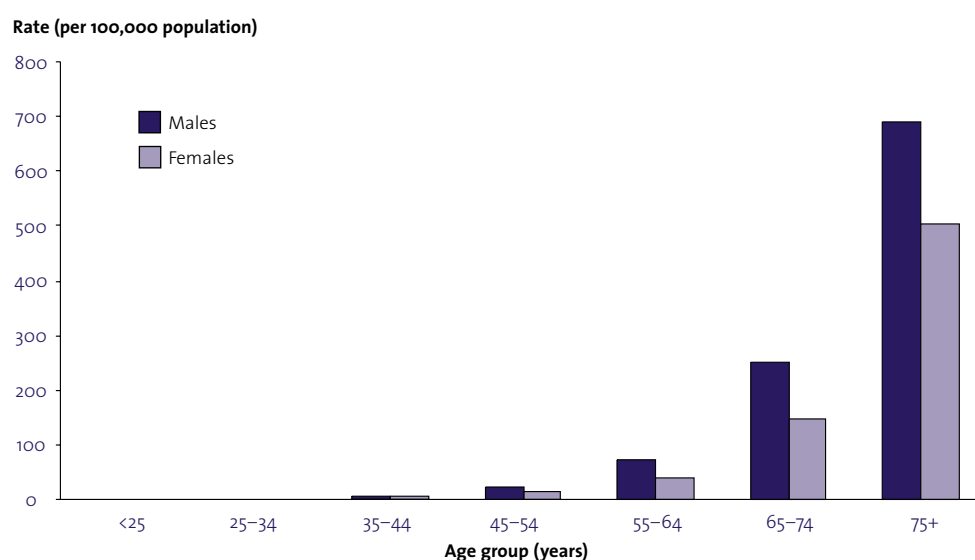
### Socioeconomic groups

People from lower socioeconomic groups are more likely to die from diabetes as the underlying cause of death than those from higher socioeconomic groups. In 1997, males from the most disadvantaged group were almost 50% more likely to die from diabetes as the underlying cause of death than those from the least disadvantaged group. Among females this difference is even greater, with females from the most disadvantaged group more than twice as likely to die from diabetes as those from the least disadvantaged group.

### Diabetes as the underlying or an associated cause of death

The underlying cause of death is the disease or injury initiating the sequence of events leading to death. However, more than one cause of death can be listed on the death certificate. This means that for each

**Figure 6.3:** Death rates from diabetes as the underlying cause or an associated cause, 2000



Source: AIHW National Mortality Database.

death, both underlying and associated causes of death can be listed and are available for analysis after 1996. For diabetes this is particularly important, as it may not be diabetes but one of its many complications which directly leads to death. A more complete picture of the mortality burden of diabetes can be obtained by examining both diabetes as the underlying cause and diabetes as an associated cause of death.

In 2000, diabetes was twice as likely to be regarded as an associated cause of death rather than as the underlying cause. A total of 10,130 deaths, 7.9% of all deaths, were due to diabetes as either the underlying cause of death or as an associated cause. Mortality due to diabetes as either the underlying or an associated cause of death increases dramatically with age, with 91% of deaths in 2000 occurring in those aged 60 years and over (Figure 6.3).

### Causes of death commonly listed with diabetes

Diabetes is rarely listed as the only cause of death on death certificates. Diabetes was listed as the only cause in less than 1.5% of deaths where diabetes was given as the underlying cause of death in 2000. Where diabetes was listed as the underlying cause of death, conditions listed as associated causes of death included coronary heart disease (in 51.0% of the cases), kidney-related diseases (22.6%), stroke (21.4%) and heart failure (18.6%).

Of the deaths in 2000 where diabetes was listed as an associated cause of death, coronary heart disease was recorded as the underlying cause of death in 35.2% of cases. Other prominent underlying causes of death with which diabetes was associated included cancer (20.0%) and stroke (11.3%).

### Years of life lost

In terms of premature deaths, diabetes was estimated to be responsible for almost 70,000 years of life lost (YLL) in 1996—almost 5.3% of the estimated YLL in Australia that year for all causes (AIHW: Mathers et al.

1999). The impact of diabetes on premature deaths was similar in both sexes, with around 37,000 years of life lost (4.9% of all YLLs) for males and more than 32,000 years of life lost (5.4% of all YLLs) for females.

### Main data source

National Mortality Database (Australian Institute of Health and Welfare).

### References and further reading

ABS (Australian Bureau of Statistics) 2001. Causes of death, Australia, 2000. Cat. No. 3303.0. Canberra: ABS.

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

## Economic impact

The monetary cost of diabetes impacts on people who have diabetes, their friends and family, non-government organisations and the government. A person with diabetes may have to pay out-of-pocket costs for medications, pathology tests, supplies and equipment. The government pays subsidies on medications and pathology tests. There are also hospital costs, general practitioner and specialist costs, the cost of public awareness campaigns and more. These costs are due not only to the diagnosis, control and care of diabetes but also, quite significantly, to the diagnosis, treatment and care of its complications.

### Direct costs of diabetes

The Australian Institute of Health and Welfare in 1999 examined the direct health system costs of diabetes in 1993–94; these were estimated to be \$372 million. Type 1 diabetes was estimated to account for 42% of this amount even though only 10–15% of people with diabetes have Type 1. When the complications of diabetes were taken into account, the total direct health system costs were estimated to be around \$681 million in 1993–94.

McCarty et al. (1996) estimated the costs of diabetes for 1995, producing a similar estimate of the direct costs. In producing their estimate of \$561 million, the authors indicated that this was likely to be an underestimate of the true direct costs of diabetes. Despite the limitations of these costing studies, a major issue to be considered is the impact that the rising prevalence of diabetes will have on health system costs in Australia.

### Hospital costs

The average total expenditure per hospital separation with a principal diagnosis of diabetes was \$3,820 in 1999–00. This figure includes overhead and administrative costs, which account for 31% of the average total expenditure per separation.

For separations with diabetes as the principal or an additional diagnosis, the average total expenditure per separation was \$3,864. The overhead and administrative costs included in this cost account for 29% of the average total expenditure per separation.

### Drug costs

In 1998, the cost of drugs, to patients and the government, to treat diabetes was \$119 million; that is, 4% of government and patient costs for all prescription drugs listed in the Pharmaceutical Benefits Scheme. The following table shows the cost of prescription drugs, insulins and analogues as well as oral blood glucose lowering drugs, used to treat diabetes in the community in Australia during 1998.

### Main data sources

Department of Health and Aged Care 1999. Australian statistics on medicines 1998. Canberra: AGPS.

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

### References and further reading

AIHW (Australian Institute of Health and Welfare): Mathers C & Penm R 1999. Health system costs of cardiovascular diseases and diabetes in Australia 1993–94. Health and Welfare Expenditure Series No. 5. AIHW Cat. No. HWE 11. Canberra: AIHW.

McCarty D, Zimmet P, Dalton A, Segal L & Welborn T 1996. The rise and rise of diabetes in Australia, 1996: a review of statistics, trends and costs. Canberra: International Diabetes Institute & Diabetes Australia.

**Table 6.1:** Antidiabetic drugs used in the community, 1998

Drug	No. scripts ('000) <sup>(a)</sup>	Cost (\$m) <sup>(b)</sup>
<b>Insulins and analogues</b>		
Insulins and analogues, fast-acting	122.3	22.1
Insulins and analogues, intermediate-acting	151.3	23.6
Insulins and analogues, intermediate-acting combined with fast-acting	133.7	24.0
Insulins and analogues, long-acting	11.1	1.4
<i>Total insulins and analogues</i>	<i>418.3</i>	<i>71.2</i>
<b>Oral blood glucose lowering drugs</b>		
Biguanides		
Metformin HCl	1,638.2	24.8
Sulphonylurea drugs		
Chlorpropamide	3.2	0.01
Glibenclamide	519.9	5.1
Gliclazide	788.3	12.2
Glipizide	216.4	3.2
Tolbutamide	55.6	0.8
<i>Total sulphonylurea drugs</i>	<i>1,583.4</i>	<i>21.2</i>
Alpha glucosidase inhibitors		
Acarbose	48.6	1.6
<i>Total oral blood glucose lowering drugs</i>	<i>3,270.3</i>	<i>47.6</i>
<b>Total antidiabetic drugs</b>	<b>3,688.6</b>	<b>118.7</b>

**Notes**

(a) Includes drugs subsidised under the Pharmaceutical Benefits and Repatriation Pharmaceutical Benefits Schemes and non-subsidised drugs.

(b) Includes government and patient costs for drugs listed in the Pharmaceutical Benefits Scheme only.

*Source: Department of Health and Aged Care 1999.*





# Appendixes

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Methods, definitions and data sources

Statistical tables

National Health Priority Areas—diabetes indicators

## diabetes australian facts 2002

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## Methods, definitions and data sources

### Rates

Age-standardised rates are used to remove the influence of age when comparing populations with different age structures. In this report the Australian population as at 30 June 1991 is used as the standard population in all Australian comparisons, unless otherwise stated.

### Burden of disease

Information on the burden of disease due to diabetes and its risk factors is taken from the results of the Australian Burden of Disease and Injury Study. The burden of disease refers to the effects of premature mortality, disability, impairment, illness and injury on a 'healthy' life. The burden is described by a summary measure of population health, the disability-adjusted life year or DALY, that combines information on the impact of premature mortality and of disability and other non-fatal health outcomes due to a disease.

### Classifications used in this report

#### Cause of death and hospital diagnosis

The classification of cause of death data is based upon the International Classification of Diseases, Tenth Revision (ICD-10). Hospital diagnosis is classified using the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification (ICD-10-AM). Table A.1 provides the codes used in this report.

**Table A.1: Disease codes**

Disease	ICD-10 code
Cardiovascular disease	G45, G46 & I00–I99
Coronary heart disease	I20–I25
Peripheral vascular disease	I71–I74
Stroke	G45, G46 & I60–I69
Diabetes	E10–E14
Neurological complications	E104, E114, E134, E144
Ophthalmic complications	E103, E113, E133, E143
Renal complications	E102, E112, E132, E142
End-stage renal disease	N17, N18
Lower limb ulcers	L97

#### Medicare Benefits Schedule (MBS) codes

Table A.2 lists the MBS items used in the Pathology section of this report.

**Table A.2: MBS codes**

Test	Item numbers
Glycosylated haemoglobin (HbA1c)	66319, 66551
Microalbumin	66361, 66560
Lipid	66317, 66334, 66335, 66337, 66339, 66341, 66521, 66524, 66527, 66530, 66533, 66536
Eye examination	10900, 10905, 10907, 10914, 10918, 104, 105, 106, 107, 108



## Aboriginal and Torres Strait Islander people

'Indigenous Australians' refers to people who identify themselves as being of Aboriginal and/or Torres Strait Islander origin. Data quality issues exist in the identification of Indigenous Australians across population surveys and administrative data collections. In the 1996 census, the number of people who identified themselves as Indigenous Australians was about a third higher than the number who did so in 1991, a difference much larger than can be explained by natural increase.

Deficiencies in health data for Indigenous Australians occur in the National Mortality Database, the National Health Survey and the National Hospital Morbidity Database. For the years 1998–00, mortality data for only Queensland, Western Australia, South Australia and the Northern Territory are considered to have sufficient coverage of Indigenous Australian deaths. No data are available from the National Health Survey on Indigenous Australians living in remote areas, due to concerns about data quality. In many States the accuracy of Aboriginal and Torres Strait Islander identification in hospital data is questionable, limiting the reliability of Indigenous morbidity statistics. No hospital data have been reported by Indigenous status in this report.

## Culturally and linguistically diverse background

In this publication the term 'culturally and linguistically diverse background' (CaLDB) has been used to classify a special population group. A number of the National Health Priority Area indicators for diabetes stipulate analysis of CaLDB. However, the information needed to properly define CaLDB is not yet available. Therefore, in this publication CaLDB is based on country of birth.

Unless otherwise noted, CaLDB is defined as those people whose country of birth was not one of the following: Australia and its external territories, New

Zealand, the United Kingdom, Ireland, the United States of America, Canada or South Africa. This selection of countries is based on an analysis of the main countries from which Australia receives overseas settlers who are likely to speak English.

The Australian Bureau of Statistics developed CaLDB as a means of standardising the collection and dissemination of information relating to the origins of individuals and cultural diversity. It is done through a system of measuring certain aspects of peoples' language and cultural background. Following the endorsement of the new standard by the Ministerial Council of Immigration and Multicultural Affairs, CaLDB is being implemented across all levels of government. However, it will take some time for data collection agencies to fully implement CaLDB.

## Socioeconomic groups

The Australian Bureau of Statistics has constructed a number of socioeconomic indexes to classify areas on the basis of social and economic information collected in the Census of Population and Housing.

This report uses the index of relative socioeconomic disadvantage. This is derived from the social and economic characteristics of the local area such as levels of income, educational attainment, public sector housing, unemployment and jobs in relatively unskilled occupations.

Individual records were classified into quintiles of socioeconomic disadvantage according to the value of this index for the statistical local area of usual residence. Quintile 1 includes the most disadvantaged households and quintile 5 the least disadvantaged households. Statistical local areas were grouped into quintiles so that each quintile contained approximately 20% of the total Australian population.

It is important to note that the index of socioeconomic disadvantage relates to the average disadvantage of all people living in the statistical local area. These

measures of socioeconomic inequality thus generally understate the true inequality in health at the individual level in Australia.

### Urban, rural and remote areas

Urban, rural and remote areas are identified in this report using the rural, remote and metropolitan areas (RRMA) classification, developed in 1994 by the Department of Primary Industries and Energy and the Department of Human Services and Health.

The RRMA classification assigns each statistical local area in Australia into one of seven categories—two metropolitan, three rural and two remote zones. These can be regrouped into three larger zones: urban (metropolitan), rural and remote. The classification is based primarily on population numbers and an index of remoteness.

This report examines data for the three larger areas (urban, rural and remote), as cell sizes are too small for accurate estimation in a more detailed classification.

### Main data sources

*2000 National Physical Activity Survey* was funded by the Australian Sports Commission, NSW Health and the ACT Department of Health and Community Services. The survey was conducted to give an assessment of physical activity patterns and knowledge of the benefits of physical activity among adult Australians after the Olympics in Sydney (September 2000) and following on from the 1997 and 1999 national physical activity surveys. The survey collected information from a national sample of 3,590 people aged 18–75 years during November and December 2000.

*1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab)*, conducted by the International Diabetes Institute and partially funded by the Commonwealth Department of Health and Aged Care, is the most comprehensive study to date on the prevalence and impact of diabetes. The survey collected information on self-reported and measured diabetes and cardiovascular risk factors, features of the Metabolic Syndrome, health knowledge, attitudes, and

health services utilisation and practices. The study collected information from approximately 11,247 adults aged 25 years and over throughout Australia (excluding the Australian Capital Territory).

*1999 National Physical Activity Survey*, funded by the Commonwealth Department of Health and Aged Care and the Australian Institute of Health and Welfare, was conducted to assess patterns of physical activity and the impact of the Active Australia campaign. The survey collected information from a national sample of 3,841 people in November and December 1999. Comparisons are made with the 1997 Active Australia baseline physical activity survey.

*1998 Disability, Ageing and Carers Survey*, conducted by the Australian Bureau of Statistics, collected national information on the disability levels of Australians, their current and future care needs and the role of carers. It can be used with previous national disability surveys to monitor trends over time. The survey collected information from a sample of about 42,100 people, from March to May 1998.

*1998 National Drug Strategy Household Survey* was conducted between June and September 1998, with 10,030 Australians aged 14 years and older participating. This was the sixth survey in a series that began in 1985. Respondents were asked about their knowledge of drugs, their attitudes towards drugs, their drug consumption histories and related behaviours.

*1995 National Health Survey*, conducted by the Australian Bureau of Statistics, was designed to obtain national information on the health status of Australians, their use of health services and facilities, and health-related aspects of their lifestyle. It can be used with previous health surveys to monitor trends in health over time. The survey collected information from a sample of 57,600 people, from January 1995 to January 1996. In this report the derived type of diabetes is used to determine the type of diabetes. This variable more accurately assesses the presence and type of diabetes based on information collected in the survey.

*1995 National Nutrition Survey*, a joint project between the Australian Bureau of Statistics and the Commonwealth Department of Health and Aged Care, is the largest and most comprehensive Australian survey of food and nutrient intake, dietary habits and body measurements. The survey collected information from a subsample of respondents from the 1995 National Health Survey, approximately 13,800 people from urban and rural areas of Australia. The National Nutrition Survey was conducted from January 1995 to January 1996.

*1983 National Dietary Survey of Adults* was conducted as a component of the second Risk Factor Prevalence Survey, by the Commonwealth Department of Health in collaboration with the National Heart Foundation. The survey was designed to obtain national information on dietary intake to determine the food composition and nutrient intake of Australians aged 25–64. The survey collected information from a sample of 5,950 people living in six of Australia's capital cities.

*Australia and New Zealand Dialysis and Transplant Registry (ANZDATA)* is a register of the delivery of kidney dialysis and transplantation to Australian and New Zealand patients. The Registry contains data on all patients receiving kidney replacement therapy where the intention to treat is long term, i.e. kidney function will not recover. Cases of acute kidney failure are excluded. The Registry is coordinated within the Queen Elizabeth Hospital and is funded by the Commonwealth Department of Health and Ageing.

*Australian National Diabetes Information Audit and Benchmarking (ANDIAB)* is a collection by the National Association of Diabetes Centres (NADC) based on an audit of patients attending a selection of specialist diabetes centres and specialist endocrinologists in private practice. It reports data on 5,680 persons with diabetes requiring specialist clinical management, in particular those who have had poor control of their diabetes. The surveys have been conducted over 1-month periods in 1998, 1999 and 2000. A major limitation of the sample is that it does not accurately reflect the conditions prevailing in the general community.

*Bettering the Evaluation And Care of Health (BEACH)*, an ongoing national survey looking at the clinical activities of general practitioners, is conducted by the General Practice Statistics and Classification Unit (an Australian Institute of Health and Welfare collaborating unit within the Family Medicine Research Centre, University of Sydney). BEACH began in April 1998 and involves a random sample of approximately 1,000 general practitioners per year.

*Burden of Disease and Injury in Australia Study*, a study that assessed the total 'burden' of disease/injury, uses a common measure developed by the Global Burden of Disease Study.

*Dental statistics* are held by the Dental Statistics and Research Unit (an Australian Institute of Health and Welfare collaborating unit within the University of Adelaide). The collection includes data on the oral health of children under the care of State and Territory school dental services, and that of adults receiving public-funded dental care. Information on access to dental care, the dental labour force and dentists' practice activity are also available.

*Drug Utilization Sub-Committee Database*, held at the Commonwealth Department of Health and Ageing, monitors the community (i.e. non-public hospital) use of prescription medicines in Australia. This database combines information on prescriptions subsidised by the Pharmaceutical Benefits Scheme (PBS) and the Repatriation Pharmaceutical Benefits Scheme with an estimate from the Pharmacy Guild Survey of those prescriptions that are not subsidised (i.e. private prescriptions and PBS prescriptions priced under the general patient copayment). Each month the Pharmacy Guild Survey collects dispensing information from a random sample of about 250 pharmacies throughout Australia. Information on drugs prescribed in public hospitals and highly specialised drugs available for outpatients through public hospital pharmacies under section 100 of the National Health Act are not included in this database.

*General practice diabetes statistics*, held at the Health Insurance Commission's web site <<http://www.hic.gov.au>> during 2001, contained a cohort of identified diabetes patients, defined as having an



HbA<sub>1c</sub> test within the preceding 2 years. The patient was allocated to a Division of General Practice based on the major practice postcode of their principal provider. In this report, pathology and other tests meeting the minimum recommended testing frequency (derived from the best practice guidelines of NSW Health, 1996, and the National Health and Medical Research Council, 1997) processed by Medicare are presented for adults aged 19 and over.

*National Aboriginal and Torres Strait Islander Survey (NATSI)* was conducted by the Australian Bureau of Statistics in 1994. The survey contains national information on the social, demographic, economic and health status of Indigenous Australians. A total of 15,700 Indigenous people were interviewed for the survey.

*National Hospital Morbidity Database*, held at the Australian Institute of Health and Welfare, contains demographic, diagnostic, procedural and duration of stay information on episodes of care for patients admitted to hospital. The data items are supplied to the Australian Institute of Health and Welfare by the State and Territory health authorities. The database provides information on the number of hospitalisations for a particular condition or procedure. It is not possible to count patients individually. In this report, disease data may relate to either the principal diagnosis or additional diagnosis of hospitalisations while procedures data relate to principal and additional procedures.

*National Diabetes Register* is a database that collects information about people who use insulin as part of their treatment of diabetes. It includes persons who began to use insulin from 1 January 1999. Data for the register are obtained from two main sources: the National Diabetes Services Scheme, administered by Diabetes Australia, and the Australasian Paediatric Endocrine Group (APEG) State-based registers. APEG registers collect information about children with diabetes aged less than 15 years. At December 2001, the register contained information about 23,179 persons.

*National Divisions Diabetes Program (NDDP) Data Collation Project* was conducted in 1999–00. The project comprised several components with participation of divisions dependent on the nature of their program and data availability. Data from the seven Divisions of General Practice piloting electronic collation of data from CARDIAB—a cardiovascular disease and diabetes database—have been used in this report. Data were collected on 4,359 patients. Quality of care and health outcomes were analysed in the areas of glycaemic control, blood pressure, weight, foot status, lipids and microalbumin level. These data indicate the status of care in environments that promote structured care and cannot be generalised to the entire general practice population.

*National Mortality Database*, held at the Australian Institute of Health and Welfare, contains information on the cause of death supplied by the medical practitioner certifying the death or by a coroner. Registration of deaths is the responsibility of the State and Territory Registrars of Births, Deaths and Marriages. Registrars provide the information to the Australian Bureau of Statistics for coding of cause of death and compilation into aggregate statistics. On 1 January 1997 the Australian Bureau of Statistics introduced new automatic coding software, which identifies multiple causes of deaths within Australia. In this report, unless otherwise specified, death data relate only to the principal cause of death.

*Risk Factor Prevalence Study*, a series of surveys conducted by the National Heart Foundation in 1980, 1983 and 1989, was designed to obtain national information on biomedical and behavioural risk factors in Australia and to monitor trends over time. While the data are somewhat dated, it remains an important source of national data for biomedical risk factors. The study collected information from a sample of around 22,000 adults living in capital cities of Australia (Canberra and Darwin were not included in the 1980 and 1983 surveys), between May/June and December of the survey year.

## Statistical tables

**Table A.3:** Death rates for diabetes by age, 2000

Disease	Sex	Age group							All ages <sup>(a)</sup>
		< 25	25–34	35–44	45–54	55–64	65–74	75+	
Rate (per 100,000 population)									
Diabetes as the underlying cause of death	Males	**	0.5*	1.6	8.1	21.5	73.9	195.1	16.9
	Females	0.2*	0.5*	0.6*	4.1	12.3	47.3	141.5	10.7
Diabetes as the underlying or an associated cause of death	Males	0.2*	1.0*	2.2	12.1	33.6	125.0	416.2	57.2
	Females	0.1*	1.0*	4.4	21.5	71.8	231.8	439.5	35.6
All causes of death	Males	67.8	127.5	167.4	310.0	812.0	2,404.9	8,234.0	712.7
	Females	39.9	48.3	88.8	197.9	484.5	1,342.2	6,574.8	450.7

\* Rates should be interpreted with caution as the relative standard errors are between 25% and 50%.

\*\* Rates are not presented as relative standard errors are greater than 50%.

(a) Age-standardised to the 1991 Australian population.

Source: AIHW National Mortality Database.

**Table A.4:** Hospitalisation rates for diabetes as a principal diagnosis or an additional diagnosis by age, 1999–00

Disease	Sex	Age group							
		< 25	25–34	35–44	45–54	55–64	65–74	75+	All ages <sup>(a)</sup>
		Rate (per 100,000 population)							
Diabetes as the principal diagnosis	Males	52.2	62.5	85.7	150.6	257.0	399.6	567.6	133.7
	Females	68.5	61.9	68.3	93.7	186.1	291.6	425.0	111.3
Diabetes as the principal or additional diagnosis	Males	94.5	249.2	652.5	1,641.7	4,216.6	8,625.3	12,675.1	1,864.9
	Females	138.6	338.9	591.3	1,247.4	2,978.6	6,222.7	8,953.7	1,407.3
Coronary heart disease or stroke with diabetes as additional diagnosis	Males	**	6.1	42.1	229.0	681.5	1,245.1	1,551.3	244.2
	Females	0.2*	2.0	23.8	88.5	292.6	697.7	1,009.0	130.7
End-stage renal disease with diabetes as additional diagnosis	Males	**	1.7	4.6	14.3	29.9	71.1	109.0	14.8
	Females	0.2*	2.0	7.0	10.4	24.5	56.3	64.9	11.5
Lower limb ulcer with diabetes as additional diagnosis	Males	**	0.3*	3.5	12.2	18.9	59.9	96.2	12.0
	Females	**	**	1.1	4.1	11.7	24.4	60.6	7.5

\* Rates should be interpreted with caution as the relative standard errors are between 25% and 50%.

\*\* Rates are not presented as relative standard errors are greater than 50%.

(a) Age-standardised to the 1991 Australian population.

Source: AIHW National Hospital Morbidity Database.

**Table A.5:** Hospitalisation rates for diabetes with various complications by age, 1999–00

		Age group							
Diabetes with complication	Sex	< 25	25–34	35–44	45–54	55–64	65–74	75+	All ages <sup>(a)</sup>
Rate (per 100,000 population)									
Diabetes with ophthalmic (eye) complication	Males	0.9	19.5	25.1	46.9	129.6	157.2	176.3	43.2
	Females	2.1	16.0	24.8	33.1	78.2	157.3	130.7	38.0
Diabetes with renal (kidney) complication	Males	0.4	33.8	100.7	121.6	220.7	423.0	232.6	95.2
	Females	1.9	17.3	55.4	105.8	136.8	240.7	150.5	64.9
Diabetes with neuropathic complication	Males	0.5	15.1	32.7	47.0	119.7	167.4	211.4	44.8
	Females	1.3	9.5	14.9	23.7	65.0	99.2	123.6	28.3

\*Rates should be interpreted with caution as the relative standard errors are between 25% and 50%.

\*\*Rates are not presented as relative standard errors are greater than 50%.

(a) Age-standardised to the 1991 Australian population.

Source: AIHW National Hospital Morbidity Database.

**Table A.6:** Risk factors for diabetes and its complications by age, 1999 to 2000

Risk factor	Sex	Age group							
		18–24	25–34	35–44	45–54	55–64	65–74	75+	25+ <sup>(a)</sup>
		Rate (per 100,000 population)							
Overweight <sup>(b)</sup>	Males	n.a.	60.2	65.5	72.5	74.0	73.7	64.3	67.3
	Females	n.a.	35.8	45.6	58.1	67.2	70.7	56.4	51.7
Insufficient physical activity <sup>(c)</sup>	Males	18.1	46.7	44.5	47.6	50.1	45.8 <sup>(d)</sup>	n.a.	42.0 <sup>(e)</sup>
	Females	32.4	40.1	46.3	52.0	46.9	46.1 <sup>(d)</sup>	n.a.	43.5 <sup>(e)</sup>
High blood pressure <sup>(f)</sup>	Males	n.a.	7.9	16.2	30.5	46.5	69.7	75.1	30.7
	Females	n.a.	4.1	7.9	22.8	42.3	66.7	77.2	25.6
High blood cholesterol <sup>(g)</sup>	Males	n.a.	31.0	54.2	60.7	61.8	54.1	49.2	49.9
	Females	n.a.	30.1	39.8	54.7	71.6	74.0	65.2	49.7
Tobacco smoking <sup>(h)</sup>	Males	24.5	29.0	26.7	22.0	15.2	11.0	4.8	22.0 <sup>(i)</sup>
	Females	23.7	23.8	23.8	17.5	13.5	6.6	4.4	18.4 <sup>(i)</sup>

n.a. Not available.

\* Rates should be interpreted with caution as the relative standard errors are between 25% and 50%.

\*\* Rates are not presented as relative standard errors are greater than 50%.

(a) Age-standardised to the 1991 Australian population.

(b) Overweight is defined as body mass index (BMI)  $\geq 25$ .

(c) Insufficient physical activity is defined as less than 150 minutes of physical activity for recreation or exercise (including walking for transport) in the previous week.

(d) Data for ages 65–75.

(e) Data for ages 18–75.

(f) High blood pressure is defined as systolic blood pressure  $\geq 140$  mm Hg and/or diastolic blood pressure  $\geq 90$  mm Hg and/or receiving treatment for high blood pressure.

(g) High blood cholesterol is defined as above 5.5 mmol/L.

(h) The daily smoking of tobacco products, including packet cigarettes, roll-your-own cigarettes, pipes and cigars.

(i) Data for ages 18 or over.

Sources: 1999–2000 AusDiab; 2000 National Physical Activity Survey; AIHW 2001 National Drug Strategy Household Survey.



# National Health Priority Areas

## —diabetes indicators

In 1996 the Australian Health Ministers agreed that diabetes become a National Health Priority Area (NHPA). The NHPA initiative focuses public attention and health policy on health conditions that contribute most to the burden of illness in the community, particularly areas where it is possible to reduce that burden through prevention and treatment programs.

A set of priority indicators covering prevention, screening and early intervention, treatment and management of the condition was developed. A complete list of the diabetes indicators outlined in the 1998 National Health Priority Areas report on diabetes mellitus appears over the page. Data on the indicators are provided in the following pages, with the exceptions of 1.3, 7.1 and 7.2 where there are no suitable data available. For some indicators, available data have been reported despite limitations in addressing the indicator definition. Notes below the indicator title are provided to alert readers to these data limitations.

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## National Health Priority Area—diabetes indicator set

### 1 Disease incidence and prevalence

- 1.1 Prevalence rates for Type 1 and Type 2 diabetes in:
  - general population
  - Indigenous population
  - people from culturally and linguistically diverse backgrounds
- 1.2 Incidence rates for Type 1 and Type 2 diabetes in:
  - general population
  - Indigenous population
  - people from culturally and linguistically diverse backgrounds
- 1.3 Gestational diabetes among women aged 20–44 years, by parity

### 2 Risk factors for diabetes and associated complications

- 2.1 Prevalence rates for obesity and overweight (as measured by BMI) among people with Type 2 diabetes and in the general population
- 2.2 Rates for non-participation in regular, sustained, moderate aerobic exercise among people with Type 2 diabetes and in the general population
- 2.3 Prevalence rates for high blood pressure among people with Type 2 diabetes:
  - $\geq 140$  mm Hg systolic and/or 90 mm Hg diastolic and aged  $< 60$  years;
  - $\geq 160$  mm Hg systolic and/or 90 mm Hg diastolic and aged  $\geq 60$  years; and/or
  - those on medication for high blood pressure
- 2.4 Prevalence rates for high levels of lipoproteins among people with Type 1 and Type 2 diabetes:
  - total cholesterol above 5.5 mmol/L; and
  - high-density lipoproteins below 1.0 mmol/L
- 2.5 Prevalence rates for fasting hypertriglyceridaemia among people with Type 1 and Type 2 diabetes

### 3 Diabetes complications

- 3.1 Proportion of people with end-stage renal disease with diabetic nephropathy as a causal factor
  - 3.2 Incidence rate for eye disease among people with clinically diagnosed diabetes
  - 3.3 Prevalence rate for foot problems among people with clinically diagnosed diabetes
  - 3.4 Incidence rates for coronary heart disease and stroke among people with clinically diagnosed diabetes and in the general population
-

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## National Health Priority Area—diabetes indicator set (continued)

### 4 Hospital separations for diabetes complications

- 4.1 Hospital separation rates for end-stage renal disease as the principal diagnosis with diabetes as an additional diagnosis
- 4.2 Hospital separation rates for coronary heart disease or stroke as the principal diagnosis with diabetes as an additional diagnosis
- 4.3 Hospital separation rates for conditions other than end-stage renal disease and coronary heart disease/stroke among people with diabetes as a principal diagnosis or an additional diagnosis

### 5 Mortality

- 5.1 Death rates for diabetes in:
  - general population
  - Indigenous population
  - people from culturally and linguistically diverse backgrounds
- 5.2 Death rates for coronary heart disease and stroke among people with diabetes in:
  - general population
  - Indigenous population
  - people from culturally and linguistically diverse backgrounds

### 6 Health status

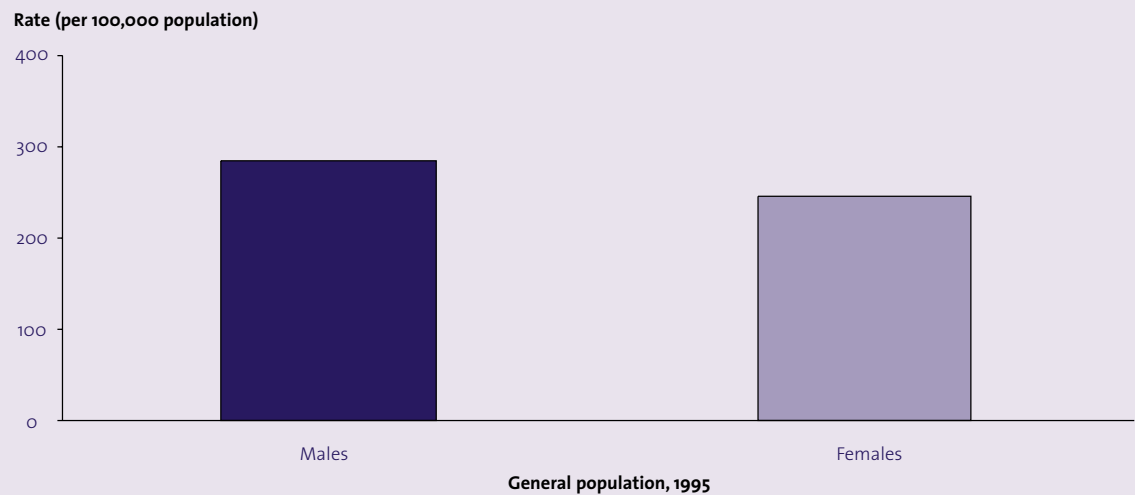
- 6.1 Self-assessed health status of people with and without diabetes

### 7 Screening and management

- 7.1 Proportion of people with diabetes tested for glycosylated haemoglobin (HbA<sub>1c</sub>) level at least every 6 months
  - 7.2 Proportion of pregnant women being tested for gestational diabetes
-

**Indicator 1.1 (a):** Prevalence rates for Type 1 diabetes in the general population, the Indigenous population, and among people from culturally and linguistically diverse backgrounds (CaLDB)

*Note: Data are not available to report prevalence for the Indigenous population or for people from culturally and linguistically diverse backgrounds.*



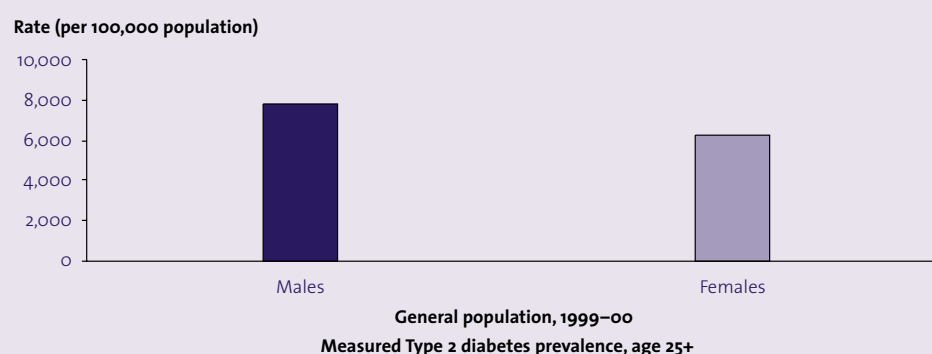
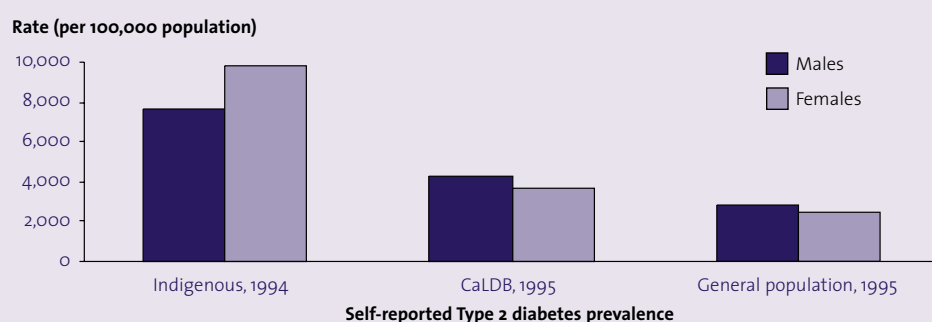
Sex	Rate (per 100,000 population)
Males	282
Females	244

Notes

- 1. Data are not available by diabetes type for the Indigenous population or for people from culturally and linguistically diverse backgrounds.
- 2. Rates are age-standardised to the Australian population as at 30 June 1991.

Source: 1995 National Health Survey (ABS).

**Indicator 1.1 (b):** Prevalence rates for Type 2 diabetes in the general population, the Indigenous population, and among people from culturally and linguistically diverse backgrounds (CaLDB)



Population group		Self-reported diabetes, all ages 1994	Self-reported Type 2 diabetes, all ages 1995	Measured Type 2 diabetes, aged 25+ 1999–2000
		Rate (per 100,000 population)		
Indigenous	Males	7,597	n.a.	n.a.
	Females	9,763	n.a.	n.a.
CaLDB	Males	n.a.	4,174	n.a.
	Females	n.a.	3,565	n.a.
General	Males	n.a.	2,772	7,753
	Females	n.a.	2,461	6,157

#### Notes

1. Data are not available by diabetes type for the Indigenous population; however, most diabetes in the Indigenous population is Type 2 diabetes.
2. Rates are age-standardised to the Australian population as at 30 June 1991.
3. CaLDB = Culturally and linguistically diverse background.

Sources: 1994 National Aboriginal and Torres Strait Islander Survey (ABS); 1995 National Health Survey (ABS); 1999–2000 AusDiab.

**Indicator 1.2:** Incidence rates for Type 1 and Type 2 diabetes in the general population, the Indigenous population, and among people from culturally and linguistically diverse backgrounds (CaLDB)

*Note: Data are not available to report the incidence of Type 2 diabetes. Nor are data available to report the incidence of Type 1 diabetes for the total population, Indigenous population or for people from culturally and linguistically diverse backgrounds. An estimate can be made for the general population aged less than 15 years; these data are provided as the best estimate available.*

Rate (per 100,000 population aged < 15 years)



Sex	Rate (per 100,000 population)
Males aged < 15 years	19.2
Females aged < 15 years	18.6

#### Notes

1. Data are sourced from the National Diabetes Register containing information on insulin-treated diabetes, predominantly Type 1 in persons aged less than 15 years.
2. Data are not available for the total population, the Indigenous population or for people from culturally and linguistically diverse backgrounds.
3. Rates refer to the incidence per 100,000 population (not age-standardised).

Source: 2000 National Diabetes Register (AIHW).

**Indicator 2.1:** Prevalence rates for obesity and overweight (as measured by BMI) among people with Type 2 diabetes and in the general population

*Note: Data presented are for people aged 25 years or over.*



Sex		1999–2000
		Per cent of persons aged 25+
Males, Type 2 diabetes (measured)	Overweight (not obese)	26.6
	Obese	62.2
Males, general population	Overweight (not obese)	48.0
	Obese	18.7
Females, Type 2 diabetes (measured)	Overweight (not obese)	21.6
	Obese	42.8
Females, general population	Overweight (not obese)	29.4
	Obese	21.4

**Notes**

1. BMI is a person's weight in kilograms divided by the square of the person's height in metres. Overweight (not obese) refers to persons with a BMI of 25.0 to less than 30.0. Obese refers to persons with a BMI of 30.0 or greater.
2. Rates are age-standardised to the Australian population as at 30 June 1991.

Source: 1999–2000 AusDiab.

**Indicator 2.2:** Rates for non-participation in regular, sustained, moderate aerobic exercise among people with Type 2 diabetes and in the general population

*Note: Data presented are for people aged 25 years or over.*



Sex		1999–2000
		Per cent of persons aged 25+
Males, Type 2 diabetes (measured)	Insufficient	29.2
	Sedentary	28.2
Males, general population	Insufficient	28.2
	Sedentary	14.0
Females, Type 2 diabetes (measured)	Insufficient	55.3
	Sedentary	12.4
Females, general population	Insufficient	35.7
	Sedentary	16.8

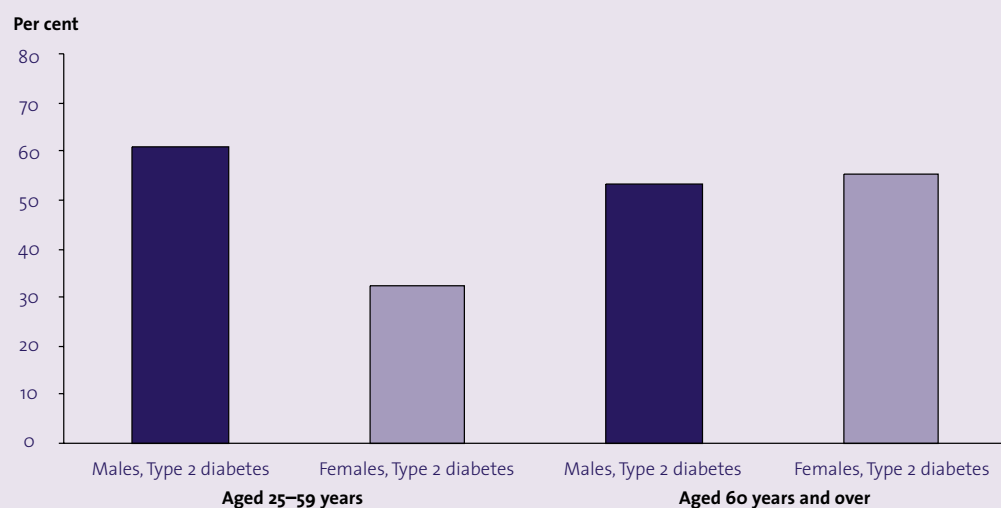
Notes

1. Sedentary refers to persons reporting no leisure-time physical activity in the week before interview. Insufficient refers to persons reporting some activity but less than 150 minutes in total in the week before interview.
2. Rates are age-standardised to the Australian population as at 30 June 1991.

Source: 1999–2000 AusDiab.



**Indicator 2.3:** Prevalence rates for high blood pressure among people with Type 2 diabetes, aged less than 60 years or aged 60 years and over



Sex	Age	1999-2000
		Per cent
Males, Type 2 diabetes (measured)	25-59 years	60.7
Females, Type 2 diabetes (measured)	25-59 years	32.2
Males, Type 2 diabetes (measured)	60 years and over	52.8
Females, Type 2 diabetes (measured)	60 years and over	55.2

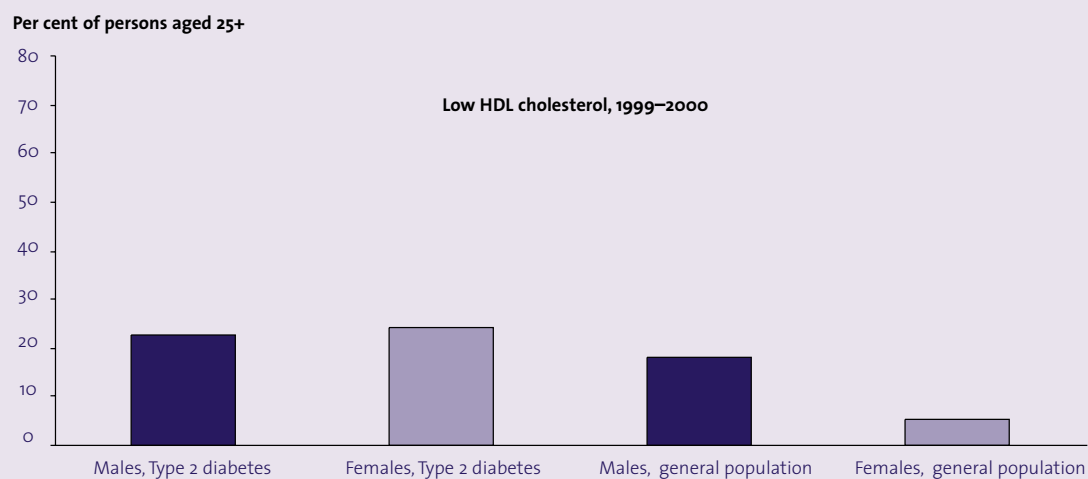
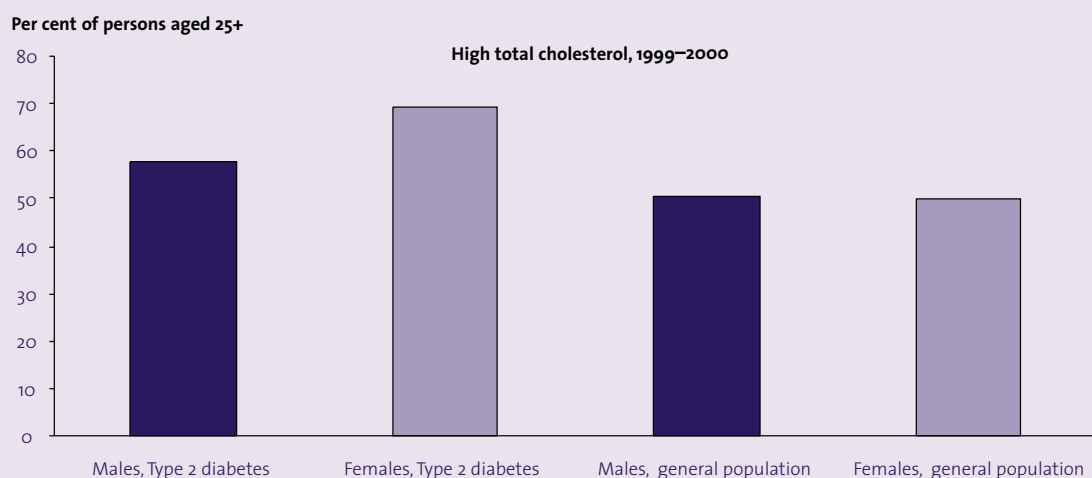
#### Notes

1. High blood pressure is defined as:  $\geq 140$  mm Hg systolic and/or 90 mm Hg diastolic, or taking anti-hypertensive medication for people aged less than 60 years;  $\geq 160$  mm Hg systolic and/or 90 mm Hg diastolic, or taking anti-hypertensive medication, for people aged 60 years and over.
2. Rates are age-standardised to the Australian population as at 30 June 1991.

Sources: 1995 National Nutrition Survey and National Health Survey (ABS), 1999-2000 AusDiab.

**Indicator 2.4:** Prevalence rates for high levels of lipoproteins among people with Type 1 and Type 2 diabetes

*Note: Data are presented separately for high total cholesterol and low HDL cholesterol. Data are not available to report prevalence rates for people with Type 1 diabetes. Data presented are for people aged 25 years and over.*



**Indicator 2.4:** (continued)

<b>High total cholesterol</b>		<b>1999–2000</b>	<b>Low HDL cholesterol</b>		<b>1999–2000</b>
		<b>Per cent of persons aged 25+</b>			<b>Per cent of persons aged 25+</b>
Type 2 diabetes (measured)	Males	57.7	Type 2 diabetes (measured)	Males	22.4
	Females	69.2		Females	24.1
General population	Males	50.2	General population	Males	17.7
	Females	49.6		Females	5.2

## Notes

1. High total cholesterol is defined as  $\geq 5.5$  mmol/L.
2. Low HDL (high density lipoprotein) cholesterol is defined as  $< 1.0$  mmol/L.
3. Rates are age-standardised to the Australian population as at 30 June 1991.
4. Estimates are not provided for people with Type 1 diabetes due to inadequate sample size.

Source: 1999–2000 AusDiab.

**Indicator 2.5:** Prevalence rates for fasting hypertriglyceridaemia among people with Type 1 and Type 2 diabetes

*Note: Data are not available to report prevalence rates for people with Type 1 diabetes. Data presented are for people aged 25 years and over.*



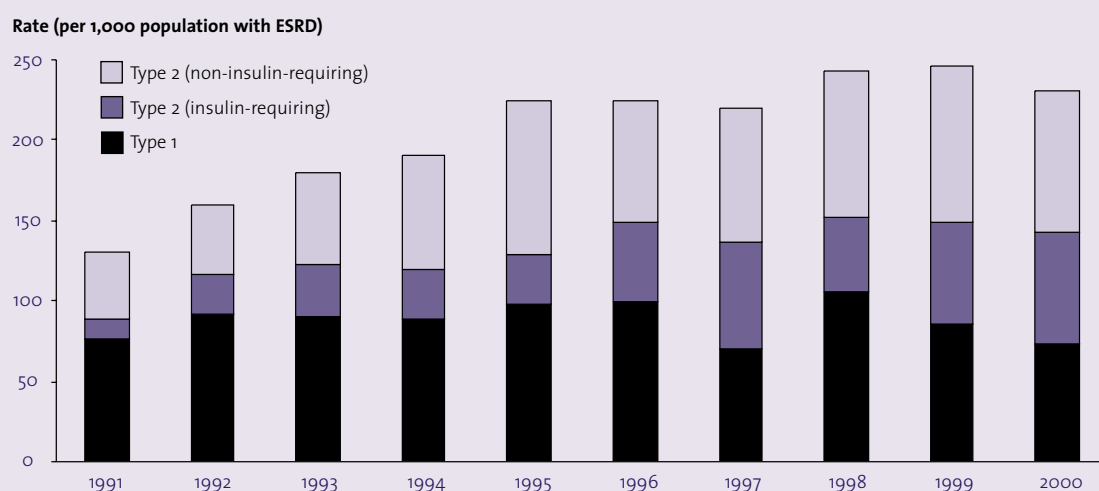
High triglycerides		Per cent of persons aged 25 +
Type 2 diabetes (measured)	Males	7.7
	Females	6.5
General population	Males	3.8
	Females	1.5

## Notes

1. Consistent with the operational definition for this indicator, fasting hypertriglyceridaemia is defined here as  $> 4.0$  mmol/L. Note that the recommended level of triglycerides is  $< 2.0$  mmol/L.
2. Rates are age-standardised to the Australian population as at 30 June 1991.
3. Estimates are not provided for people with Type 1 diabetes due to inadequate sample size.

Source: 1999–2000 AusDiab.

**Indicator 3.1:** Proportion of people with end-stage renal disease (ESRD) with diabetic nephropathy as a causal factor



Diabetes type	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Rate (per 1,000 population with ESRD)										
Type 1	76.0	91.0	90.0	88.0	97.7	97.9	68.0	104.3	84.2	72.6
Type 2 insulin-requiring	11.5	24.0	31.0	29.0	30.8	50.0	64.5	46.6	62.0	68.7
Type 2 non-insulin-requiring	42.0	44.0	57.0	70.9	94.9	71.0	83.5	90.1	95.1	88.6
<b>Total</b>	<b>130.0</b>	<b>158.8</b>	<b>178.5</b>	<b>188.0</b>	<b>223.5</b>	<b>219.1</b>	<b>216.0</b>	<b>241.0</b>	<b>241.3</b>	<b>229.9</b>

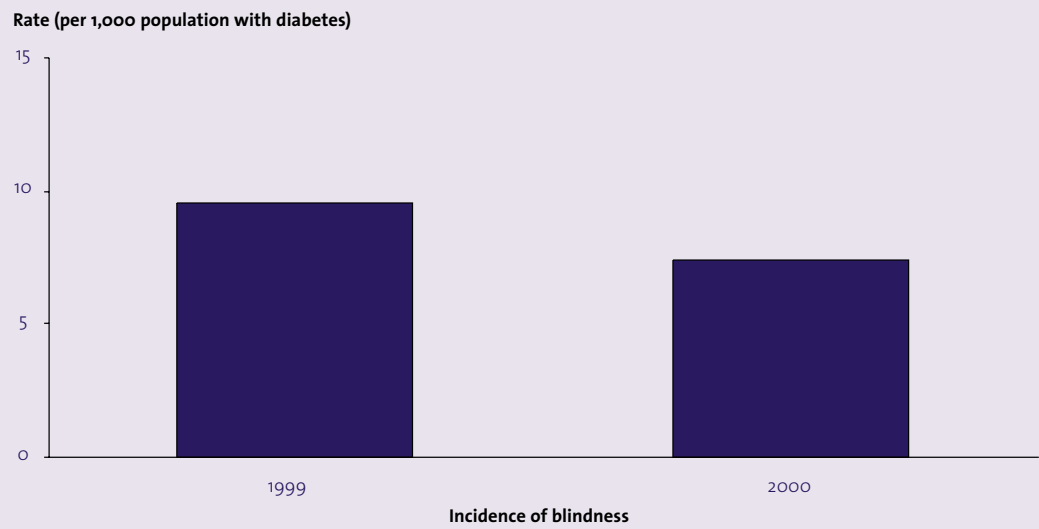
#### Notes

1. Data are presented for new ANZDATA patients only.
2. Rates are age-standardised to the Australian population as at 30 June 1991.

Sources: Estimates from the 1996, 1997, 1998, 1999, 2000 and 2001 ANZDATA Registry Annual Reports.

Indicator 3.2: Incidence rate for eye disease among people with clinically diagnosed diabetes

*Note: Data are not available to report the incidence rate for eye disease. Data presented are for incidence of blindness among patients with diabetes attending diabetes centres.*



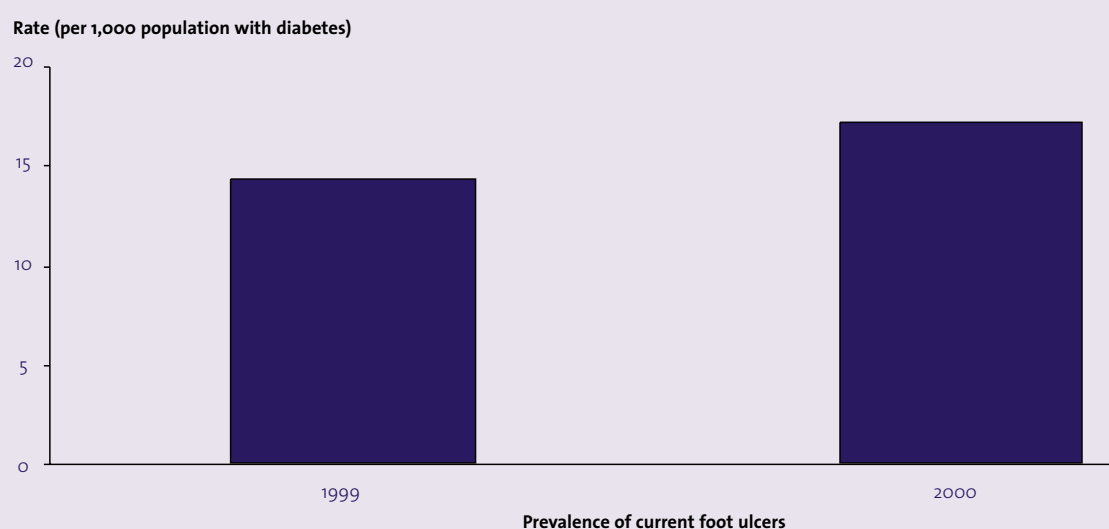
	1999	2000
Rate of blindness (per 1,000 population with diabetes)		
Persons with diabetes	9.5	7.4

Note: Rates are age-standardised to the Australian population as at 30 June 1991.

Source: 1999 and 2000 Australian National Diabetes Information Audit and Benchmarking (NADC).

**Indicator 3.3:** Prevalence rate for foot problems among people with clinically diagnosed diabetes

*Note: Data presented are for prevalence of current foot ulcers among patients with diabetes attending diabetes centres.*



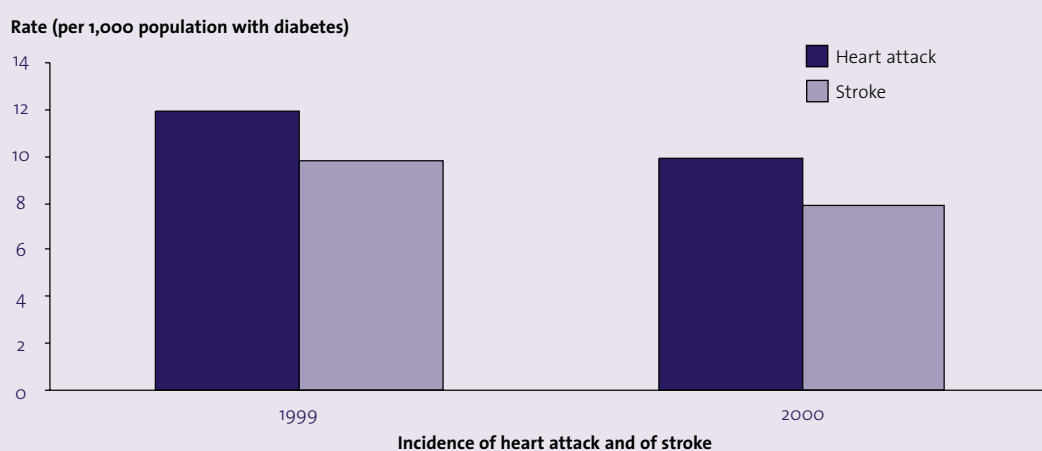
	1999	2000
Rate (per 1,000 population with diabetes)		
Persons with diabetes	14.3	17.2

Note: Rates are age-standardised to the Australian population as at 30 June 1991.

Sources: 1999 and 2000 Australian National Diabetes Information Audit and Benchmarking (NADC).

**Indicator 3.4:** Incidence rates for coronary heart disease and stroke among people with clinically diagnosed diabetes and in the general population

*Note: Data are not available to report incidence rates for coronary heart disease and stroke in the general population and among people with diabetes. Data presented are for incidence rates of heart attack and stroke among patients with diabetes attending diabetes centres.*



	Heart attack		Stroke	
	1999	2000	1999	2000
Rate (per 1,000 population with diabetes)				
Males with diabetes	15.7	11.4	11.2	10.5
Females with diabetes	7.4	7.9	8.1	5.0
Persons with diabetes	11.8	9.9	9.8	7.9

Note: Rates are age-standardised to the Australian population as at 30 June 1991.

Sources: 1999 and 2000 Australian National Diabetes Information Audit and Benchmarking (NADC).



**Indicator 4.1:** Hospital separation rates for end-stage renal disease as the principal diagnosis with diabetes as an additional diagnosis



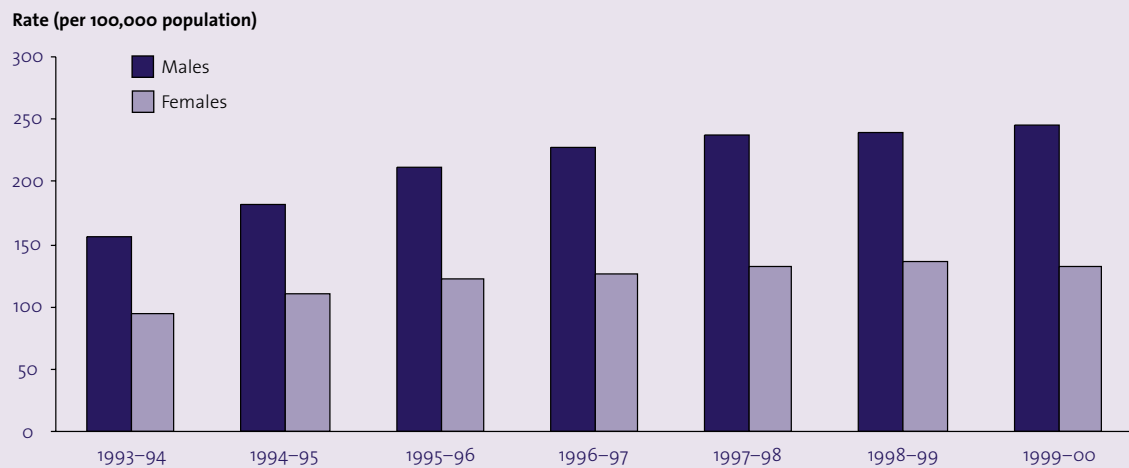
	1993-94	1994-95	1995-96	1996-97	1997-98	1998-99	1999-00
Rate (per 100,000 population)							
Males	7	8	10	11	15	15	15
Females	6	7	9	10	13	12	11
<b>Persons</b>	<b>6</b>	<b>7</b>	<b>10</b>	<b>10</b>	<b>14</b>	<b>13</b>	<b>13</b>

#### Notes

1. Rates are age-standardised to the Australian population as at 30 June 1991.
2. For 1993-94 to 1998-99 the disease groupings are classified according to the ICD-9-CM codes: 584-585 for end-stage renal disease and 250 for diabetes.
3. For 1999-00 the disease groupings are classified according to the ICD-10-AM codes: N17-N18 for end-stage renal disease and E10-E14 for diabetes.

Source: AIHW National Hospital Morbidity Database.

**Indicator 4.2:** Hospital separation rates for coronary heart disease or stroke as the principal diagnosis with diabetes as an additional diagnosis



	1993-94	1994-95	1995-96	1996-97	1997-98	1998-99	1999-00
Rate (per 100,000 population)							
Males	156	181	210	226	236	239	244
Females	93	109	122	125	131	134	131
<b>Persons</b>	<b>123</b>	<b>143</b>	<b>164</b>	<b>173</b>	<b>181</b>	<b>184</b>	<b>184</b>

Notes

1. Rates are age-standardised to the Australian population as at 30 June 1991.
2. For 1993-94 to 1998-99 the disease groupings are classified according to the ICD-9-CM codes: 410-414 for coronary heart disease, 430-438 for stroke and 250 for diabetes.
3. For 1999-00 the disease groupings are classified according to the ICD-10-AM codes: I20-I25 for coronary heart disease, G45, G46 and I60-I69 for stroke and E10-E14 for diabetes.

Source: AIHW National Hospital Morbidity Database.

**Indicator 4.3:** Hospital separation rates for conditions other than end-stage renal disease and coronary heart disease/stroke among people with diabetes as a principal diagnosis or an additional diagnosis



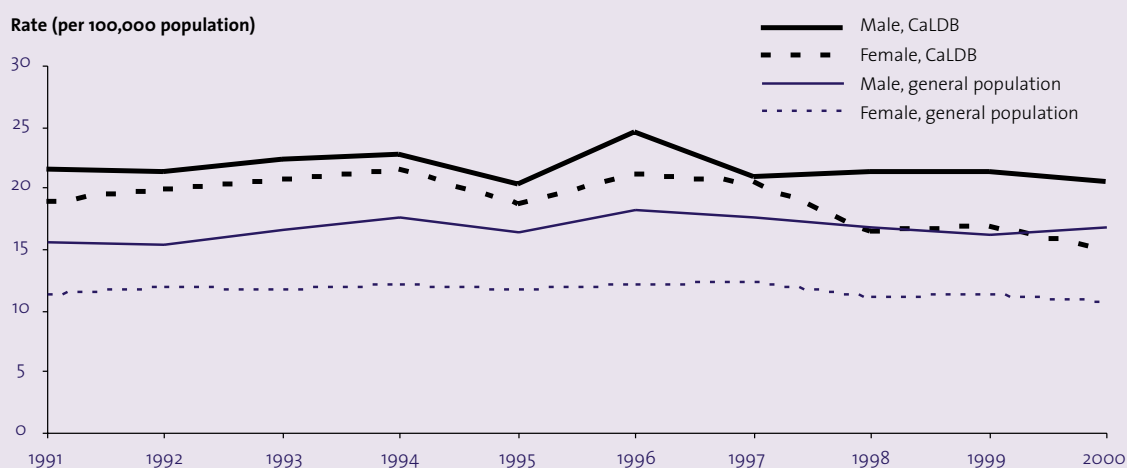
	1993-94	1994-95	1995-96	1996-97	1997-98	1998-99	1999-00
Rate (per 100,000 population)							
Males	858	1,023	1,188	1,322	1,469	1,577	1,606
Females	703	835	986	1,089	1,190	1,251	1,265
<b>Persons</b>	<b>770</b>	<b>916</b>	<b>1,073</b>	<b>1,190</b>	<b>1,312</b>	<b>1,396</b>	<b>1,418</b>

#### Notes

1. Rates are age-standardised to the Australian population as at 30 June 1991.
2. For 1993-94 to 1998-99 the disease groupings are classified according to the ICD-9-CM codes: 584-585 for end-stage renal disease, 410-414 for coronary heart disease, 430-438 for stroke and 250 for diabetes.
3. For 1999-00 the disease groupings are classified according to the ICD-10-AM codes: N17-N18 for end-stage renal disease, I20-I25 for coronary heart disease, G45, G46 and I60-I69 for stroke and E10-E14 for diabetes.

Source: AIHW National Hospital Morbidity Database.

**Indicator 5.1:** Death rates for diabetes in the general population, Indigenous population and among people from culturally and linguistically diverse backgrounds (CaLDB)



	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	
Rate (per 100,000 population)											
Males, CaLDB population	21.5	21.4	22.4	22.7	20.2	24.4	20.9	21.3	21.4	20.5	
Females, CaLDB population	18.9	20.0	20.6	21.6	18.6	21.1	20.5	16.5	16.8	14.9	
Males, general population	15.6	15.4	16.7	17.6	16.4	18.2	17.7	16.8	16.2	16.9	
Females, general population	11.4	11.9	11.8	12.1	11.8	12.2	12.4	11.1	11.3	10.7	
	1998–2000				1998–2000						
Males, Indigenous population	116.5				Males, non-Indigenous population						15.1
Females, Indigenous population	152.4				Females, non-Indigenous population						10.6

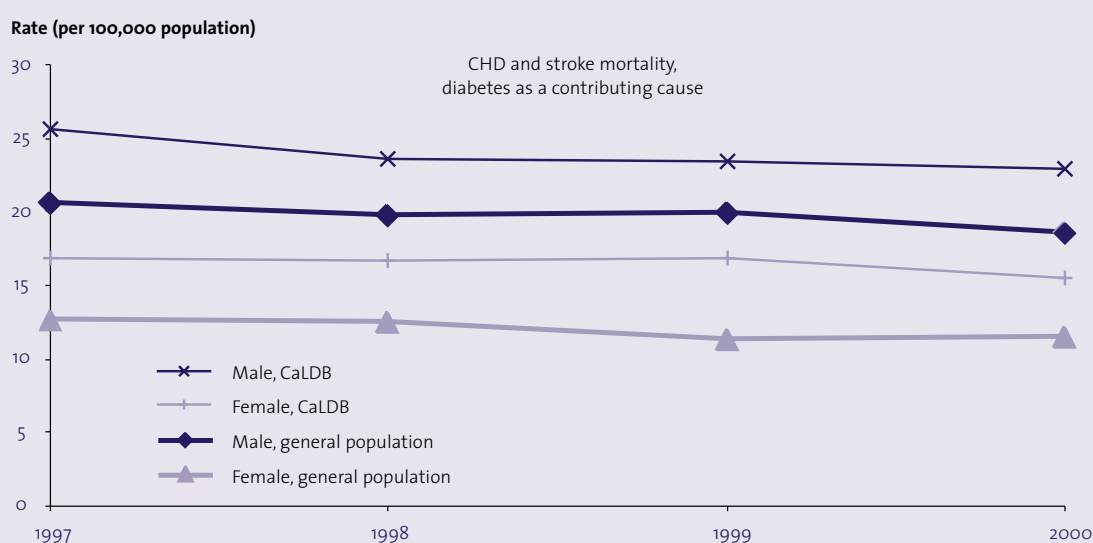
#### Notes

1. Rates are age-standardised to the Australian population as at 30 June 1991.
2. CaLDB = Culturally and linguistically diverse background. CaLDB is based on a country of birth other than Australia, New Zealand, United States of America, Canada, South Africa, United Kingdom or Ireland.
3. Aboriginal and Torres Strait Islander is based on only Queensland, Western Australia, South Australia and the Northern Territory.
4. For 1991 to 1996 the disease grouping is classified according to the ICD-9 code: 250 for diabetes.
5. For 1997 to 2000 the disease grouping is classified according to the ICD-10 codes: E10–E14 for diabetes.

Source: AIHW National Mortality Database.

**Indicator 5.2:** Death rates for coronary heart disease (CHD) and stroke among people with diabetes in the general population, the Indigenous population and among people from culturally and linguistically diverse backgrounds (CaLDB)

*Note: Data are not available to report death rates for coronary heart disease and stroke for the Indigenous population.*



	1997	1998	1999	2000
Rate (per 100,000 population)				
<b>General population</b>				
Males	21	20	20	19
Females	13	12	11	11
<b>People from culturally and linguistically diverse backgrounds</b>				
Males	26	24	23	23
Females	17	17	17	15

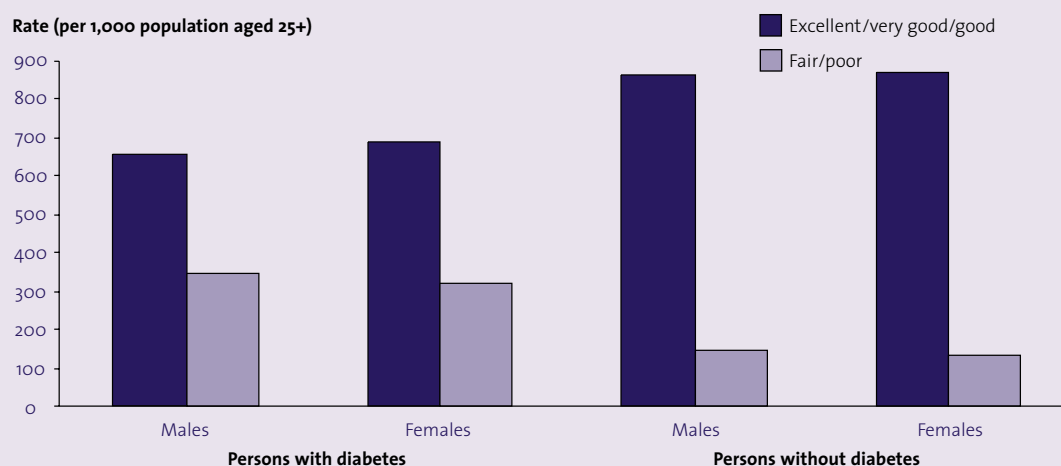
#### Notes

1. Rates are age-standardised using the Australian population as at 30 June 1991.
2. 'People from culturally and linguistically diverse backgrounds' is based on a country of birth other than Australia, New Zealand, United States of America, Canada, South Africa, United Kingdom or Ireland.
3. For 1997 and 1998, the disease groupings are classified according to the ICD-9-CM codes: 410–414 for coronary heart disease, 430–438 for stroke and 250 for diabetes as a contributing cause of death.
4. For 1999 and 2000, the disease groupings are classified according to the ICD-10-AM codes: I20–I25 for coronary heart disease, G45, G46 and I60–I69 for stroke and E10–E14 for diabetes as a contributing cause of death.

Source: AIHW National Mortality Database.

**Indicator 6.1:** Self-assessed health status of people with and without diabetes

*Note: Data presented are for people aged 25 years or over.*

**Self-assessed health status****1999–2000**

Rate (per 1,000 population aged 25+)

*Good, very good or excellent*

Males, with diabetes	657
Males, without diabetes	859
Females, with diabetes	684
Females, without diabetes	870

*Fair or poor*

Males, with diabetes	343
Males, without diabetes	141
Females, with diabetes	316
Females, without diabetes	130

## Notes

1. Rates are age-standardised to the Australian population as at 30 June 1991.
2. Health status is assessed using the question 'In general, would you say that your health is: excellent / very good / good / fair / poor?'

Source: 1999–2000 AusDiab.



# Abbreviations

<b>ABS</b>	Australian Bureau of Statistics
<b>AGPS</b>	Australian Government Publishing Service
<b>AIHW</b>	Australian Institute of Health and Welfare
<b>ANDIAB</b>	Australian National Diabetes Information Audit and Benchmarking
<b>ANZDATA</b>	Australia and New Zealand Dialysis and Transplant Registry
<b>APEG</b>	Australasian Paediatric Endocrine Group
<b>AusDiab</b>	The Australian Diabetes, Obesity and Lifestyle Study
<b>BEACH</b>	Bettering the Evaluation And Care of Health
<b>BMI</b>	Body mass index
<b>CaLDB</b>	Culturally and linguistically diverse backgrounds
<b>CHD</b>	Coronary heart disease
<b>CPI</b>	Community Periodontal Index
<b>DALY</b>	Disability-adjusted life year
<b>DBP</b>	Diastolic blood pressure
<b>DDD</b>	Defined daily dose
<b>DHAC</b>	Department of Health and Aged Care
<b>ESRD</b>	End-stage renal disease
<b>GI</b>	Glycaemic Index
<b>GP</b>	General practitioner
<b>HbA1c</b>	Glycosylated haemoglobin
<b>HDL</b>	High-density lipoprotein
<b>ICD-9</b>	International Classification of Diseases, 9th Revision
<b>ICD-10</b>	International Classification of Diseases, 10th Revision
<b>ICD-10-AM</b>	International Classification of Diseases, 10th Revision Australian Modification
<b>IDDM</b>	Insulin-dependent diabetes mellitus
<b>IGT</b>	Impaired glucose tolerance
<b>JDRF</b>	Juvenile Diabetes Research Foundation
<b>LDL</b>	Low-density lipoprotein
<b>MBS</b>	Medicare Benefits Schedule
<b>NADC</b>	National Association of Diabetes Centres
<b>NDDP</b>	National Divisions Diabetes Program
<b>NDSS</b>	National Diabetes Services Scheme
<b>NHPA</b>	National Health Priority Area
<b>NHMRC</b>	National Health and Medical Research Council
<b>NHS</b>	National Health Survey
<b>NIDP</b>	National Integrated Diabetes Program

<b>NPDR</b>	Non-proliferative diabetic retinopathy
<b>OGTT</b>	Oral glucose tolerance test
<b>PVD</b>	Peripheral vascular disease
<b>RRMA</b>	Rural, Remote and Metropolitan Areas classification
<b>SBP</b>	Systolic blood pressure
<b>WHO</b>	World Health Organization
<b>YLL</b>	Years of life lost to premature mortality
<b>YLD</b>	Years of life lost to disability





# Symbols

\$	Australian dollars, unless otherwise specified
–	nil or rounded to zero
%	per cent
g	gram
kJ	kilojoule
mm Hg	millimetres of mercury
mmol/L	millimoles per litre
n.a.	not available
>	more than
≥	more than or equal to
<	less than
≤	less than or equal to





# Glossary

**abdominal obesity:** Excess fat around the trunk of the body, also called central obesity. Technically defined as a waist circumference  $\geq 102$  cm for males or  $\geq 88$  cm for females.

**albuminuria:** More than normal amounts of a protein called albumin in the urine.

**angina:** Temporary chest pain or discomfort when the heart's own blood supply is inadequate to meet extra needs, as in exercise.

**associated causes of death:** All morbid conditions, diseases and injuries (separate from the *underlying cause of death* recorded on the death certificate) contributing to death. See *cause of death*.

**atherosclerosis:** A process that gradually clogs arteries, through fatty and fibre-like deposits building up on the inner walls of the arteries, and can lead to *cardiovascular disease*.

**blood cholesterol:** Fatty substance produced by the liver and carried by the blood to supply the rest of the body. Its normal function is to provide material for cell walls and for steroid hormones, but if levels in the blood are too high it can lead to *atherosclerosis*.

**blood pressure:** The force exerted by blood against the walls of the arteries. The force is created by the pumping action of the heart, at contraction (systolic) and at relaxation (diastolic).

**body mass index (BMI):** The most commonly used method of assessing whether a person is normal weight, underweight, overweight or obese. Calculated by dividing the person's weight (in kilograms) by their height (in metres) squared, i.e.  $\text{kg/m}^2$ .

**cardiovascular disease:** Any disease of the heart or blood vessels, including *heart attack*, *angina*, *stroke* and *peripheral vascular disease*.

**cause of death:** The disease or factor contributing to the death. When used technically, this term is usually applied to the 'underlying cause' listed on the medical certificate issued at death. The *underlying cause of death* is defined as the main disease that initiated the train of events leading directly to death, distinct from *associated causes of death* which are conditions, diseases or injuries that contributed to the death, directly or indirectly.

**cataract:** Clouding of the lens of the eye.

**central obesity:** See *abdominal obesity*.

**cerebrovascular:** Of or relating to blood vessels and the supply of blood to the brain. See *stroke*.

**chronic disease:** A disease persisting for a long period (at least 3 to 6 months).

**complications:** Conditions and illness resulting directly or indirectly from another disease or condition.

**coronary heart disease (CHD):** *Heart attack* and *angina* (chest pain). Also known as ischaemic heart disease.

**creatinine:** A chemical found in the blood and passed in the urine. A test of the amount of creatinine in blood or in blood and urine indicates functioning of the kidneys.

**dental caries:** Tooth decay.

**dialysis:** A method of removing excess waste substances from the blood when the kidneys are unable to work effectively.



**disability:** When used technically, disability refers to the presence of one or more of a defined set of limitations, restrictions or impairments.

**disability-adjusted life year (DALY):** A summary statistic to describe years of healthy life lost through disability and/or premature mortality.

**dyslipidaemia:** Unhealthy levels of fats (lipids) in the blood.

**encounter (general practitioner):** Any professional interchange between a patient and a general practitioner.

**endocrinologist:** A doctor who treats people who have problems with their endocrine glands. Diabetes is an endocrine disorder.

**glaucoma:** An eye disease associated with increased pressure within the eye.

**glomeruli:** The primary filtration units of the kidney.

**glucose:** The main sugar that the body uses for energy. Glucose comes from the breakdown of carbohydrates in the diet as well as from the breakdown of glycogen (the storage form of glucose) in the liver.

**HDL cholesterol:** Cholesterol packaged in high-density lipoprotein particles. The HDLs are good acceptors of membrane-free cholesterol and transport it back from tissues to the liver.

**health risk factor:** Any factor that represents a greater risk of a health disorder or other unwanted condition. Some risk factors are regarded as causes of disease, others are regarded as contributors.

**heart attack:** Life-threatening emergency that occurs when a vessel supplying blood to the heart muscle is suddenly blocked completely. The event may lead to the death of a part of the heart muscle. The medical term commonly used for a heart attack is myocardial infarction.

**heart failure:** When the heart cannot pump strongly enough to keep the blood circulating around the body at an adequate rate.

**hospital separation:** The formal process by which a hospital records the completion of treatment and/or care for an admitted patient. The episode of care may be completed by an admitted patient's discharge, death, transfer to another hospital, or change in the type of care.

**hyperglycaemia:** High blood *glucose* levels.

**hypertension:** High *blood pressure*.

**hypertriglyceridemia:** High levels of *triglycerides*; a marker of lipid abnormalities.

**hypoglycaemia:** A low blood *glucose* level.

**impaired glucose tolerance:** Slower metabolism of *glucose* due to *insulin* deficiency or resistance. Classified as fasting plasma glucose less than 7.0 mmol/L and 2-hour plasma glucose 7.8–11.0 mmol/L after oral glucose tolerance testing (OGTT).

**incidence:** The number of new cases (of a disease, condition or event) occurring during a given period. Compare with *prevalence*.

**insulin:** A hormone produced in the *pancreas* that helps *glucose* to enter body cells for energy metabolism.

**insulin resistance:** A condition in which *insulin* works inefficiently and the body compensates by producing an excess supply.

**International Classification of Diseases (ICD):** The World Health Organization's internationally accepted statistical classification of disease and injury.

**ischaemic heart disease:** See *coronary heart disease*.

**LDL cholesterol:** Cholesterol packaged in low-density lipoprotein particles. LDLs carry cholesterol to the various tissues for use.

**Metabolic Syndrome:** Also called Syndrome X, is a symptom cluster associated with a high risk of *coronary heart disease* and *stroke*. Central to Metabolic Syndrome is *insulin resistance*. Other common signs include *impaired glucose tolerance*, excessively high blood insulin levels, high *blood pressure* and abnormal blood cholesterol levels (specifically high levels of triglycerides and low levels of HDL cholesterol).

**morbidity:** Refers to ill-health in an individual and to levels of ill-health in a population or group.

**myocardial infarction:** See *heart attack*.

**neuropathy:** A disease of the system that results in damage to nerves.

**nephropathy:** A disease of the kidneys.

**obesity:** Increased adiposity or fat mass, associated with several chronic diseases and their risk factors. Technically defined as *body mass index*  $\geq 30$ . Also see *abdominal obesity* and *central obesity*.

**ophthalmologist:** A doctor who sees and treats people with eye problems or diseases.

**pancreas:** An organ that produces digestive substances and hormones, including *insulin*.

**periodontal:** Refers to the supporting structures of the teeth, including the gums, connective tissue and bone.

**peripheral vascular disease:** Pain in the legs due to an inadequate blood supply to them.

**prevalence:** The number or proportion (of cases, instances, etc.) present in a population at a given time. Compare with *incidence*.

**principal diagnosis:** The diagnosis established after study to be chiefly responsible for occasioning the patient's episode of care in hospital (or attendance at the health care facility).

**retinopathy:** A disease of the small blood vessels in the retina of the eye.

**risk factor:** See *health risk factor*.

**Rural, Remote and Metropolitan Areas classification:** A classification that assigns geographic areas into one of seven categories: capital cities, other metropolitan centres, large rural centres, small rural centres, other rural areas, remote centres and other remote areas.

**saturated fats:** Fats that are solid and are found in the diet mostly from animal sources. In excess, they tend to raise *blood cholesterol*.

**separation:** See *hospital separation*.

**sleep apnoea:** Cessation or prolonged break in breathing during sleep.

**stroke:** When an artery supplying blood to the brain suddenly becomes blocked or bleeds, often causing paralysis of parts of the body or speech problems.

**triglycerides:** A hydrophobic, neutral lipid, packaged with proteins and cholesterol in various lipoprotein particles.

**underlying cause of death:** The main disease or injury initiating the sequence of events leading directly to death. See *cause of death*.