Main types of diabetes 2

Type 1 diabetes

Type 2 diabetes

Gestational diabetes

diabetes
australian facts 2002

Australian Institute of Health and Welfare

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

	Males		Females	
Age at diagnosis	**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
Total	**387**	**19.2**	**356**	**18.6**

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 (≥ 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 (polydypsia)
- 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

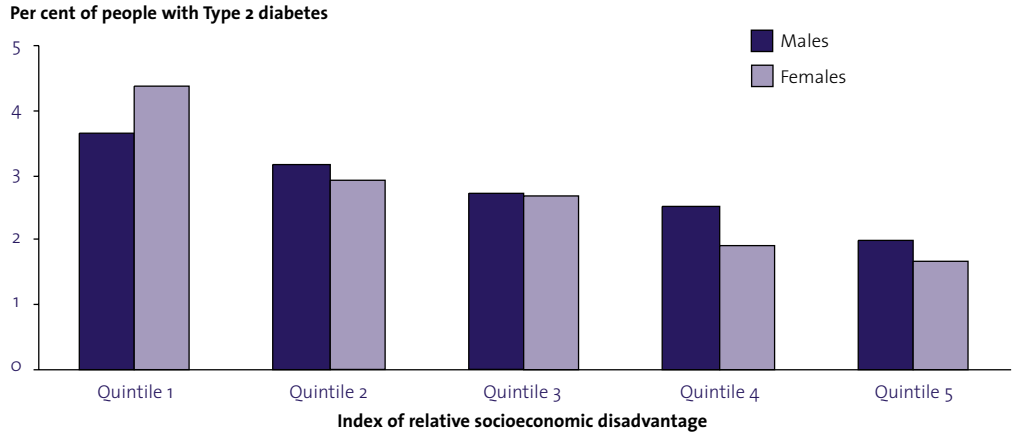
Table 2.3: Prevalence of Type 2 diabetes for persons aged 25 years or over, 1999–2000

Age group	Males	Females	Persons
		Per cent	
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
Total	**7.6**	**6.7**	**7.2**

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. Diabesity 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 Europids 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 ≥ 7.8 millimoles/litre (mmol/L) after a 50 g glucose drink or ≥ 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 ≥ 5.5 mmol/L at 0 hours and/or ≥ 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.