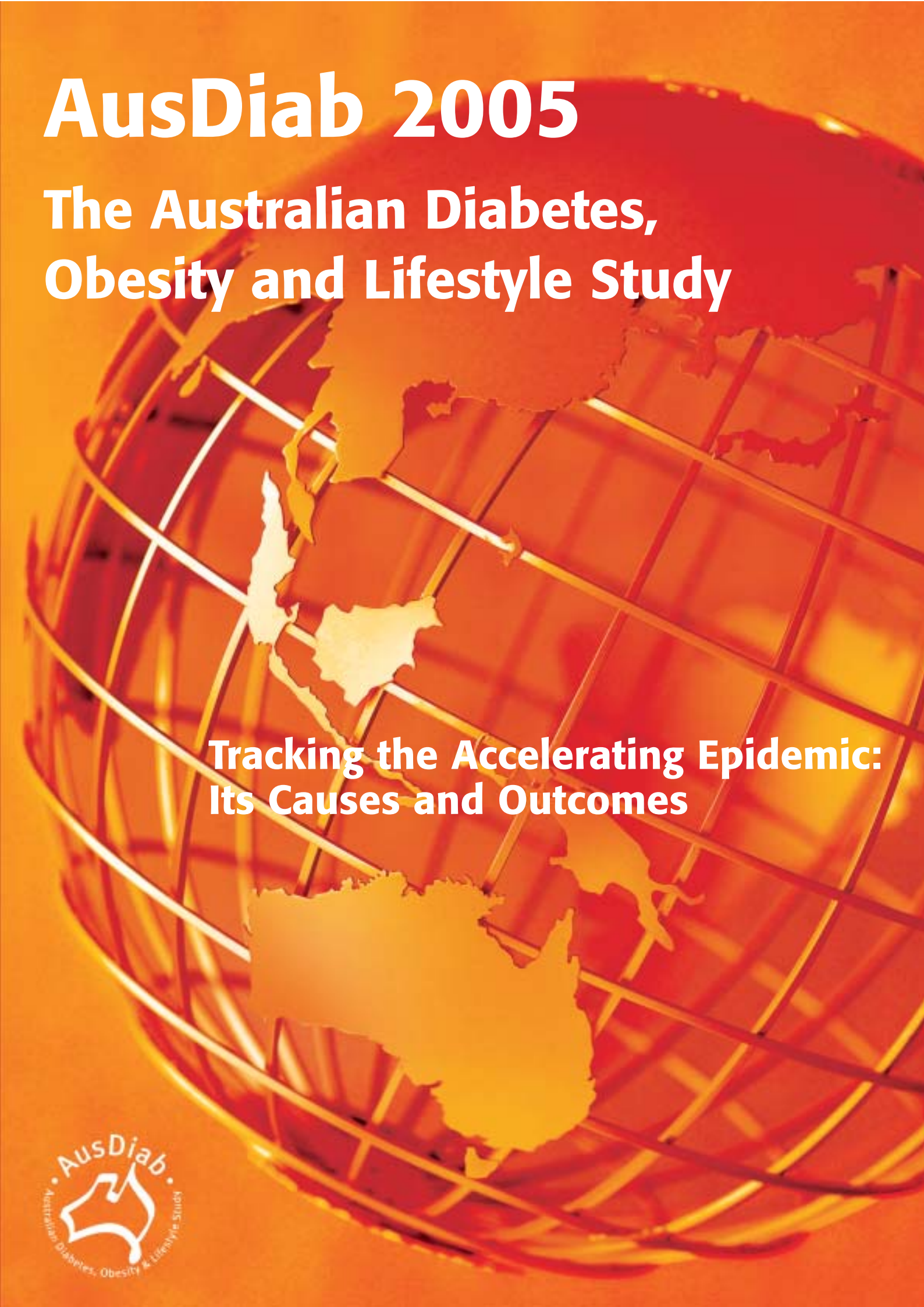
AusDiab 2005

The Australian Diabetes, Obesity and Lifestyle Study



Tracking the Accelerating Epidemic: Its Causes and Outcomes

AusDiab
Australian Diabetes, Obesity & Lifestyle Study

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The Australian Diabetes, Obesity and Lifestyle Study

Tracking the Accelerating Epidemic: Its Causes and Outcomes

AusDiab Report

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What is diabetes?

Diabetes mellitus is a metabolic disease characterised by high blood glucose levels (hyperglycaemia) resulting from defects in insulin secretion, insulin action or both. The chronic hyperglycaemia of diabetes is associated with long-term damage, dysfunction and failure of virtually every body organ, especially the heart and blood vessels, eyes, kidneys and nerves.

- Type 1 diabetes results from autoimmune destruction of the pancreatic beta cells – the cells that produce insulin. In this form of diabetes, insulin injections are required for survival. Type 1 diabetes accounts for approximately 10% of all persons with diabetes in Australia. Type 1 diabetes can occur at any age, although usually before 40 years.

- Type 2 diabetes is characterised by insulin resistance and/or abnormal insulin secretion, either of which may predominate. It is the most common form of diabetes accounting for more than 85% of persons with diabetes in Australia. It has a strong genetic (familial) propensity, which is unmasked by lifestyle factors such as obesity and lack of exercise. In most instances the cause is not yet known.

AusDiab Report

Ministerial foreword

Tony Abbott

In 1999, all Australian Health Ministers endorsed the National Diabetes Strategy. The Strategy aims to coordinate the wide range of activities being undertaken across Australia to improve the prevention, early detection and management of diabetes.

A central component of the strategy was the need to better understand the burden of diabetes faced by Australians. To that end, the Commonwealth Government supported the initial Australian Diabetes, Obesity and Lifestyle (AusDiab) study which undertook, for the first time ever, the task of determining how many Australians were affected by diabetes.

Professor Paul Zimmet and his team at the International Diabetes Institute in Melbourne, who co-ordinated this project, concluded that one in thirteen adult Australians had diabetes and a further one in six were at high risk of developing diabetes.

The information derived from AusDiab has contributed to the Government's understanding of the problem and facilitated the planning and delivery of services around the country to ease the burden of diabetes. This has included funding through the *National Diabetes Strategy and the National Integrated Diabetes Program and Support for Diabetes Research* budget measures.

The second stage of this ground-breaking study has now been completed. The five-year follow-up of the individuals who took part in the first stage will provide further information about the risks of developing diabetes. This report provides the first-ever national data on the rate at which diabetes is developing, and who faces the greatest risks. The development of kidney disease, obesity, and hypertension, as well as mortality risks, are also covered in this wide-ranging study. The information contained in this report will inform health care planners, clinicians and citizens in the battle against diabetes and related disorders.

The Honourable Tony Abbott MHR
Minister for Health and Ageing

Foreword

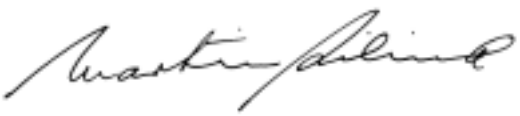
International Diabetes Federation

Professor Martin Silink

In 1999, Professors Paul Zimmet and Tim Welborn assembled a team from across Australia, spearheaded by the International Diabetes Institute, for the ambitious task of conducting a nationwide survey of diabetes and related disorders. The Australian Diabetes, Obesity and Lifestyle (AusDiab) study not only met, but exceeded, the initial expectations, and with over 30 publications in national and international peer-reviewed journals, has become one of the premier epidemiological studies in the field of diabetes anywhere in the world. Australia now stands tall as one of the few nations that has a clear understanding of the burden of diabetes (as well as obesity, kidney and heart disease) that it faces.

The AusDiab team has now completed an important second phase of the study. The follow-up, over five years, of the individuals who took part in the original study, allows an accurate assessment of the risks associated with each of the diabetes risk factors. For example, it will provide much-needed information on the exact risk faced by a person with obesity. The longitudinal nature of the study is the gold standard for understanding how diseases develop and progress, and will provide the AusDiab researchers with the necessary data with which to describe rates of disease progression, and explore new ideas about the development of diabetes.

The International Diabetes Federation, which represents the needs of people with diabetes around the world, is a strong supporter of research into the burden of the disease, and recognises that the AusDiab study is now one of the leading sources of information on diabetes. It has also made a vital contribution to understanding kidney disease, as well as to facilitating a more holistic approach to chronic disease prediction. This report will be the first of many publications from this second phase of AusDiab, as AusDiab continues to provide crucial information for the tracking of the diabetes epidemic in Australia and globally.



Professor Martin Silink
President Elect, International Diabetes Federation

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Executive summary

Diabetes mellitus has become one of the most common non-communicable diseases in the world. It results in substantial morbidity and mortality, primarily from cardiovascular complications, eye and kidney diseases and limb amputations. It now represents one of the most challenging public health problems of the 21st century.

Australia is a nation that by world standards provides its population with the opportunities for good health. Life expectancy is high; however, modernisation and industrialisation have led to a reduction in physical activity and an increase in the consumption of energy-rich foods. Consequently, lifestyle diseases such as diabetes, heart disease and kidney disease are impacting increasingly upon the health of many Australians.

The Australian Diabetes, Obesity and Lifestyle study (AusDiab) is the largest Australian longitudinal population-based study established to examine the natural history of diabetes, pre-diabetes (in which glucose metabolism is impaired but not to the level to cause diabetes), heart disease and kidney disease. Key findings from the baseline survey conducted during 1999–2000 provided benchmark national data on the prevalence (or number of people) with diabetes, obesity, hypertension, and kidney disease in Australia. The second phase of AusDiab, a 5-year follow-up survey of people who participated in the baseline study, now provides a unique opportunity to determine the incidence (or number of new cases) of diabetes, cardiovascular disease and kidney disease, in order to improve our understanding of the factors that increase the risk of developing these conditions.

This report presents the main findings from the AusDiab 5-year follow-up, incorporating data collected from participants in the 1999–2000 baseline survey and the 5-year follow-up survey in 2004–05. Chapters 2–6 present the annual incidence of diabetes, pre-diabetes, obesity, hypertension, and chronic kidney disease. Chapter 7 presents the total mortality data for this 5-year follow-up. The following summarises the key findings for each of these chapters.

Diabetes and pre-diabetes

- Every year 0.8% of adults developed diabetes.
- Every day in Australia approximately 275 adults develop diabetes.
- Those with pre-diabetes were 10 – 20 times more likely to develop diabetes than were those with normal blood glucose levels.
- Obesity, hypertension, dyslipidaemia, physical inactivity and the metabolic syndrome each increased the risk for developing diabetes.

Obesity

- Those aged less than 65 years showed an average weight increase of 1.8 kg over five years. People aged 65 years and older showed a loss in weight of 0.8 kg over five years.
- The average change in waist circumference was greater in females than in males for all age groups.
- Younger people gained more weight and had a greater increase in waist circumference than did older people.
- Twice as many overweight people became obese as reverted to normal.

Executive summary

Blood pressure

- Every year, 3.0% of adults developed high blood pressure.
- The risk of developing high blood pressure was 1.0% per year for people aged 25–34 years and increased to 8.4% per year for people aged 65–74 years.
- The risk of developing high blood pressure was greater for people with pre-diabetes and diabetes, and for those who were overweight or obese. Each of these risk factors (diabetes and obesity) had a greater impact on the development of hypertension for females than for males.

Metabolic syndrome

- People who were obese were six times more likely to develop the metabolic syndrome, than were those of normal weight.
- People with diabetes were twice as likely to develop the metabolic syndrome, than were those with normal blood glucose levels.
- Physically inactive people were also at increased risk of developing the metabolic syndrome.
- Increasing age was associated with an increased risk of developing the metabolic syndrome.
- Across all ages and all weight and physical activity categories, males were at a higher risk of developing the metabolic syndrome than were females.

Kidney disease

- Every year, almost 1.0% of adults developed chronic kidney disease, manifested by a reduction in kidney function (impaired glomerular filtration rate). The risks were higher in females and in older people.
- Every year, almost 1.0% of adults developed evidence of kidney damage as manifested by the leakage of albumin into the urine (albuminuria). The risks were higher in males and in older people.
- Having high blood pressure increased the risk of developing impaired glomerular filtration rate and albuminuria three-fold.
- Having diabetes increased the risk of developing albuminuria five-fold and of developing a reduction in kidney function two-fold.

Mortality

- Over five years, people with previously known diabetes were twice as likely to die as were those with normal glucose tolerance.
- Pre-diabetes was associated with a 45–55% increase in mortality risk over five years.
- People with previously known diabetes had a similar risk of mortality to smokers and to people with previous cardiovascular disease.
- Over two-thirds of all cardiovascular disease mortalities in the AusDiab cohort occurred in people with diabetes or pre-diabetes.

Executive summary

Conclusion

The AusDiab study is the first national longitudinal study to investigate the prevalence and incidence of diabetes, heart disease, kidney disease, and related risk factors such as obesity, hypertension and dyslipidaemia in Australians. It is the only national study in the developed world to incorporate an oral glucose tolerance test. This has enabled a comprehensive examination of the impact of all levels of abnormal glucose metabolism on the development of diabetes, heart disease and kidney disease. Findings from the 5-year AusDiab study have indicated that every year eight out of every 1,000 people in Australia developed diabetes. This, together with the increasing number of new cases of pre-diabetes, obesity, the metabolic syndrome, and kidney disease, has demonstrated that abnormal glucose metabolism is having a major impact on the health of Australians. This is further exemplified by the mortality risk associated with diabetes, where people with previously diagnosed diabetes at baseline were twice as likely to die compared to those with normal glucose tolerance.

Plans are now under way for a 10-year follow-up for AusDiab. In 2009-10, in addition to inviting all AusDiab participants to return for a third visit, another cohort of new participants will be recruited from the general population. Ongoing follow-up of the AusDiab study will provide Australians with the opportunity to continue mapping the changing impact that diabetes, heart disease and kidney disease have on the wider community.

1: Background

Diabetes and associated conditions

Diabetes mellitus has become one of the most common non-communicable diseases in the world and results in substantial morbidity and mortality, primarily from cardiovascular complications, eye and kidney diseases and limb amputations. It now represents one of the most challenging public health problems of the 21st century.[1]

Throughout the world, diabetes is reaching epidemic levels. Recently, in collaboration with the International Diabetes Federation, the International Diabetes Institute contributed epidemiological data to the 2003 *Diabetes Atlas* which presented current and future estimates for the prevalence of diabetes for various countries. It was estimated that in the year 2003 there were approximately 194 million people with diabetes in the world. This is predicted to climb to over 333 million people by the year 2025, the majority of these developing type 2 diabetes. Furthermore, in 2003 it was estimated that 314 million people in the world had impaired glucose tolerance (IGT) – an asymptomatic condition defined by elevated (though not diabetic) blood glucose levels. Individuals with IGT are at high risk of progressing to type 2 diabetes. It is predicted that the number of people with IGT in the world will increase to 472 million by the year 2025. The epidemiological evidence suggests that without effective prevention and control programs, diabetes will likely continue to increase at alarming rates worldwide.[2]

Australia is a nation that by world standards provides a high proportion of its population with excellent opportunities for good health. This is the result of the public health triumphs of the 20th century with the near elimination of the infectious diseases that were the major causes of death in the 19th century. Despite these advances, lifestyle diseases such as diabetes and heart disease are having a greater impact on the health of Australians, with Indigenous Australians, Pacific Islanders, and those of Asian origin being particularly susceptible.[3-5] The major causes of death in Australia are now cancer, heart disease and stroke.[5] This has been attributed both to the effects of an ageing population and to the adverse effects on lifestyle that have come with the modernisation and industrialisation of society. Physical activity is being engineered out of our lives due to increasing mechanisation and computerisation. Lifestyle changes have also had an unfavourable influence on our diet, as the consumption of energy-rich foods has increased. Together with a reduction in physical activity, this is contributing to an increase in obesity.[6]

The national strategy

The above factors, together with the ageing of the Australian population, have led to high levels of morbidity from a number of chronic diseases, which contribute greatly to national health costs. Diabetes and cardiovascular disease are two of these conditions.[1,6] As a result, they have been included by the Australian Government, State and Territory governments as two of the seven National Health Priority Areas, which also include arthritis and musculoskeletal conditions, asthma, cancer control, injury prevention and control, and mental health.

In Australia, it is acknowledged that diabetes contributes to many diseases.[3] Diabetes is acknowledged to be:

- the most common reason for commencing renal dialysis;
- the most common cause of blindness in people under the age of 60 years;
- the most common cause of non-traumatic lower-limb amputation;
- a major cause of cardiovascular disease; and
- one of the most common chronic diseases in children.

In 1998, Dr Michael Wooldridge, as Health Minister, created a National Diabetes Strategy and Implementation Plan.[7] Its aims were to:

- prevent or delay the development of type 1 and type 2 diabetes;
- improve quality of life and reduce complications and premature mortality in people with diabetes;
- achieve maternal and child outcomes for gestational diabetes and for women with pre-existing diabetes equivalent to those of non-diabetic pregnancies;
- achieve progress towards a cure for type 1 diabetes;
- advance knowledge and understanding about the prevention, cure and care of diabetes, through a comprehensive research effort; and
- improve the capacity of the health system to deliver, manage and monitor services for the prevention of diabetes and the care of people with diabetes.

Despite having these ambitious objectives, it was widely acknowledged in the late 1990s that considerable gaps in knowledge existed with respect to the prevalence and incidence of diabetes, its complications and associated conditions in Australia. Previous prevalence estimates had been based on studies that had used self-reported data such as the 1995 ABS National Health Survey.[8] However, the true prevalence of diabetes can only be established by taking blood samples, because type 2 diabetes can be asymptomatic for many years, and self-reported diabetes represents about one half of actual cases.[9] Previous attempts to document the prevalence of known and unknown cases of diabetes and also IGT through an oral glucose tolerance test had been restricted to studies such as those undertaken in the 1980s in the rural Western Australian town of Busselton.[10] These studies were not considered to be adequately representative of the general population of Australia. Consequently, the need to obtain nationally representative estimates of the prevalence of diabetes and related conditions was given high priority within the National Diabetes Strategy. Such data were considered vital for the planning of programs to prevent the onset of diabetes and its complications, and to provide an essential baseline upon which to assess the impact of such programs in the future.

In addition, as there have not been any nationally representative longitudinal studies on diabetes undertaken in Australia, the incidence of these conditions within the Australian population has not been determined. Gaining an understanding of the natural history of diabetes in Australia through a longitudinal study is a highly valuable tool in determining the extent to which hypertension, dyslipidaemia, obesity and various levels of glucose intolerance, such as IGT and impaired fasting glucose (IFG) (collectively known as pre-diabetes), contribute to the future development of diabetes in the Australian setting. Information gathered from nationally representative longitudinal data is considered to be essential for the planning of public health initiatives and for advising and treating individuals at personal risk of diabetes and associated conditions.

1: Background

The AusDiab study

The Australian Diabetes, Obesity and Lifestyle study (AusDiab) is the first national Australian longitudinal population-based study established to examine the natural history of diabetes and its complications, as well as heart disease and kidney disease. Identified as being the only national longitudinal study of its kind to have been undertaken in a developed nation, the AusDiab study consists of two distinct phases: (i) the AusDiab baseline survey conducted during 1999 – 2000, and (ii) the AusDiab 5-year follow-up conducted in 2004 – 05. Each phase provides unique information relating to the prevalence and incidence of diabetes and associated conditions in Australia.

(i) The baseline AusDiab study

The baseline component of the AusDiab study was a cross-sectional population-based study conducted during 1999–2000. The study consisted of a nationally representative sample of 11,247 adults aged 25 years and older who underwent a detailed physical examination, which included glucose tolerance testing, measurement of cardiovascular risk factors, assessment of diet, physical activity and other lifestyle parameters, and investigation for kidney disease. The first AusDiab report[11] of findings from the baseline study was published in 2001, and revealed:

- a diabetes prevalence of 7.4%, one of the highest for a Western nation;
- that the number of people with diabetes had more than doubled since 1981;
- that a further 16.3% had pre-diabetes – either IGT or IFG;
- 60% were overweight or obese;
- 29% had hypertension;
- 66% had dyslipidaemia; and
- 2.5% had proteinuria, 6.4% had haematuria and 1.7% had renal impairment.

(ii) The 5-year AusDiab follow-up study

Although the baseline AusDiab study provided data on the number of people in Australia with diabetes and related conditions, it was not designed to provide estimates on the rate at which people were developing these conditions. In recognition of this, the 5-year follow-up to AusDiab was established, in which all participants of the baseline study were invited to return for re-testing. The 5-year follow-up provides the first population-based Australian data on the risk factors for, and predictors of, diabetes and associated conditions, and will supply much needed data to determine Australia's health burden from type 2 diabetes, cardiovascular disease, obesity and kidney disease.

The main aims of the AusDiab 5-year follow-up were to:

- describe the natural history of type 2 diabetes, and pre-diabetes (IGT and IFG), and associated cardiovascular disease risk factors and complications;
- identify risk factors associated with worsening glucose tolerance status and the development of diabetic complications including cardiovascular disease; and
- measure the progression of renal disease in both the diabetic and the non-diabetic populations.

The difference between prevalence and incidence

When investigating the patterns of disease within a population, it is useful to describe both the prevalence and the incidence.

Prevalence:

The proportion of people within a population who have a certain disease or condition at a particular time.

Incidence:

Among those without the disease or condition, the number of new cases that develop over a period of time.

In the context of the AusDiab study, the 1999–2000 baseline survey collected information about a number of conditions and diseases including diabetes and its complications, cardiovascular disease, obesity and kidney disease. The baseline survey enabled the measurement of prevalence; that is, the number of Australians with these diseases or conditions at that point in time. It provided an estimate of how many people in Australia in 1999–2000 had diabetes and other conditions.

The second survey was undertaken five years after the baseline study (2004–05) and provided the opportunity to investigate the incidence, that is, the number of *new cases*, arising in the Australian population for each of these conditions. This is possible because participants in the baseline survey have been followed up to investigate who did and who did not develop these conditions.

Structure of this report

This report presents the main findings from the AusDiab 5-year follow-up, incorporating data collected from participants in the 1999–2000 baseline survey and the 5-year follow-up survey in 2004–05.

Annual incidence was estimated from the numbers of people developing each of the diseases and conditions studied over the five-year period between surveys. Key findings relating to diabetes and pre-diabetes, obesity, blood pressure, the metabolic syndrome, and chronic kidney disease are presented in Chapters 2–6. Each chapter presents the annual incidence for each of these conditions according to age, sex and other risk factors.

Chapter 7 presents total mortality data for diabetes, pre-diabetes, heart disease and kidney disease for this 5-year follow-up period. Detailed survey methods, including data collection and statistical analyses, response rates, and definitions for each of the conditions are described in Chapters 8–10.

As for the first report on the baseline survey, more detailed analyses of the data from the follow-up survey, in particular the incidence and associations of the complications of diabetes, eye disease and cardiovascular disease, will be subsequently published in peer-reviewed journals.

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2: Diabetes and pre-diabetes

Background

The term diabetes mellitus describes a metabolic disorder with multiple causes characterised by chronically elevated blood glucose (hyperglycaemia) levels, with disturbances of carbohydrate, fat and protein metabolism. The effects of diabetes include long-term damage, dysfunction and failure of various organs and tissues. It predisposes those suffering from it to many severe conditions, including cardiovascular disease, as well as visual loss, amputations and renal failure.

Diabetes is a disease with mixed aetiology. There are many risk factors for the development of the disease including obesity, hypertension, sedentary lifestyle, dyslipidaemia and the metabolic syndrome, many of which are also risk factors for cardiovascular disease. This chapter presents the incidence (% per year) of diabetes, and examines the association between risk factors and the development of diabetes.

Definitions

Diabetes and pre-diabetes

The diagnostic criteria for diabetes, impaired glucose tolerance (IGT) and impaired fasting glucose (IFG), were based on the values for venous plasma glucose concentration (fasting and two-hour measurements) outlined in the World Health Organization report on the Diagnosis and Classification of Diabetes Mellitus (Table 2.1).[1] People who reported taking oral hypoglycaemic medication and/or insulin were classified as having diabetes regardless of their plasma glucose levels. The term 'pre-diabetes' is used to include all those with either IGT or IFG. In this report, results for type 1 and type 2 diabetes have not been reported separately, as the vast majority of cases were classified as type 2.

Table 2.1. Classification values for the oral glucose tolerance test.

	Plasma glucose (mmol/l)		
Glucose tolerance	Fasting glucose		2-hour glucose
Diabetes	≥7.0	or	≥11.1
Impaired glucose tolerance (IGT)	<7.0	and	7.8–11.0
Impaired fasting glucose (IFG)	6.1–6.9	and	<7.8
Normal glucose tolerance (NGT)	<6.1	and	<7.8

All participants on oral hypoglycaemic medication or insulin were classified as having diabetes.

2: Diabetes and pre-diabetes

Incident diabetes

New (incident) cases of diabetes were defined as individuals who had either normal glucose tolerance (NGT) or IFG or IGT at baseline, but had developed diabetes at follow-up.

Incident cases of impaired fasting glucose and impaired glucose tolerance

New (incident) cases of IFG were defined as people who had NGT at baseline, but had developed IFG at follow-up. New (incident) cases of IGT were defined as people who had NGT or IFG at baseline, but had developed IGT at follow-up.

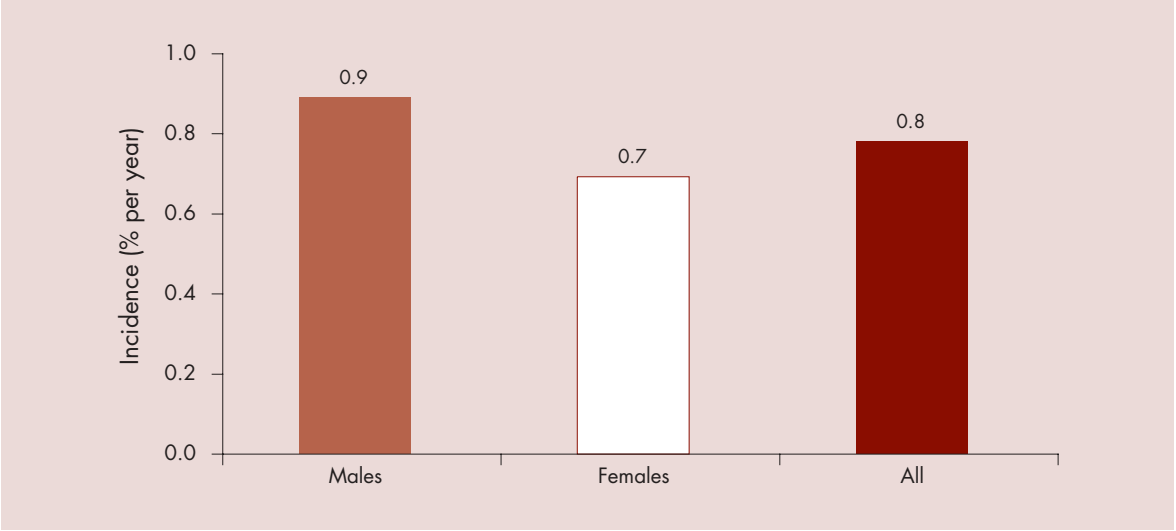
Results

Incidence of diabetes and pre-diabetes

The overall incidence (% per year) of diabetes is shown in Figure 2.1, and indicates that there were slightly more new cases of diabetes per year in males than in females. When the figures were projected to the whole Australian population, they indicated that approximately 275 Australian adults develop diabetes every day.

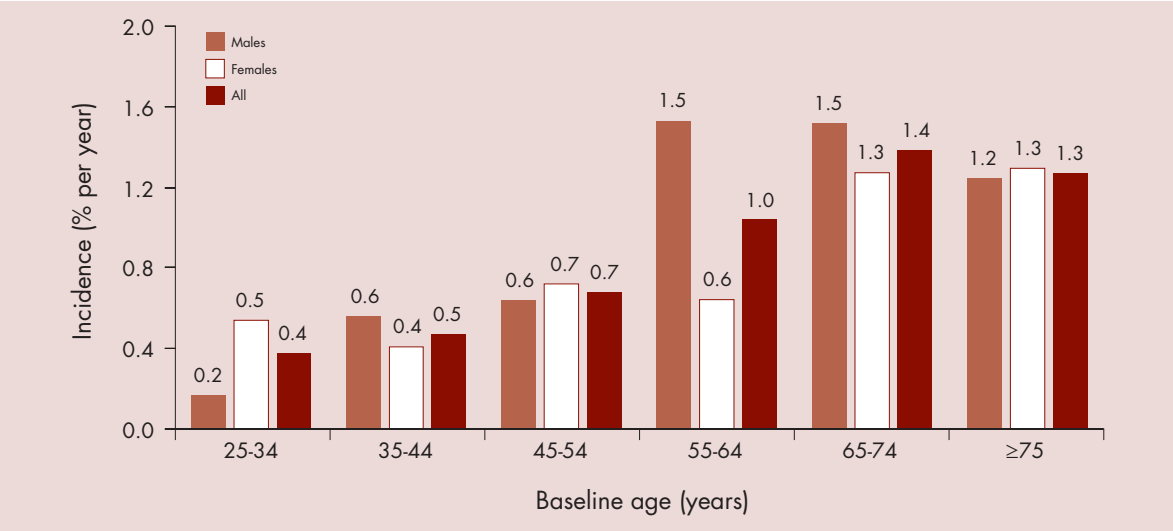
The annual incidence of diabetes increased with age, peaking between the ages of 65 and 74 years, and then decreased after the age of 75 years (Figure 2.2). For males, the incidence of diabetes increased until the 55–74 age groups, and then decreased in those aged 75 years and older. In females, the incidence of diabetes increased with age, until it plateaued after the age of 65.

Figure 2.1. Incidence of diabetes according to sex: the AusDiab study.



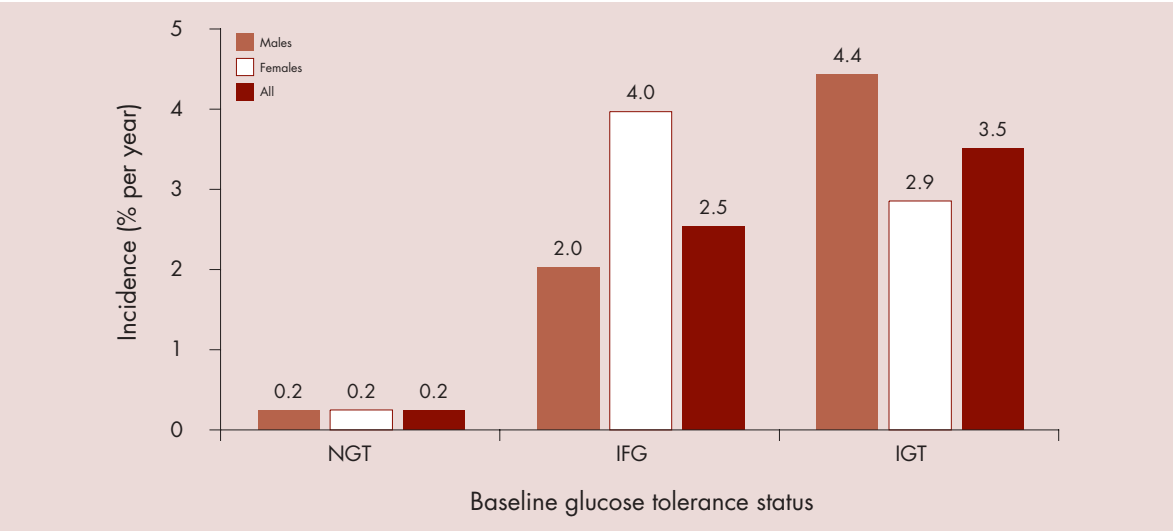
The heights of bars on all graphs are accurate to two decimal places, but data labels are rounded to one decimal place.

Figure 2.2. Incidence of diabetes according to baseline age: the AusDiab study.



The annual incidence of diabetes among those with NGT, IFG and IGT is shown in Figure 2.3. In both males and females, the incidence of diabetes in IGT and IFG was 10–20 times greater than in those with NGT at baseline.

Figure 2.3. Incidence of diabetes according to baseline glucose tolerance status: the AusDiab study.

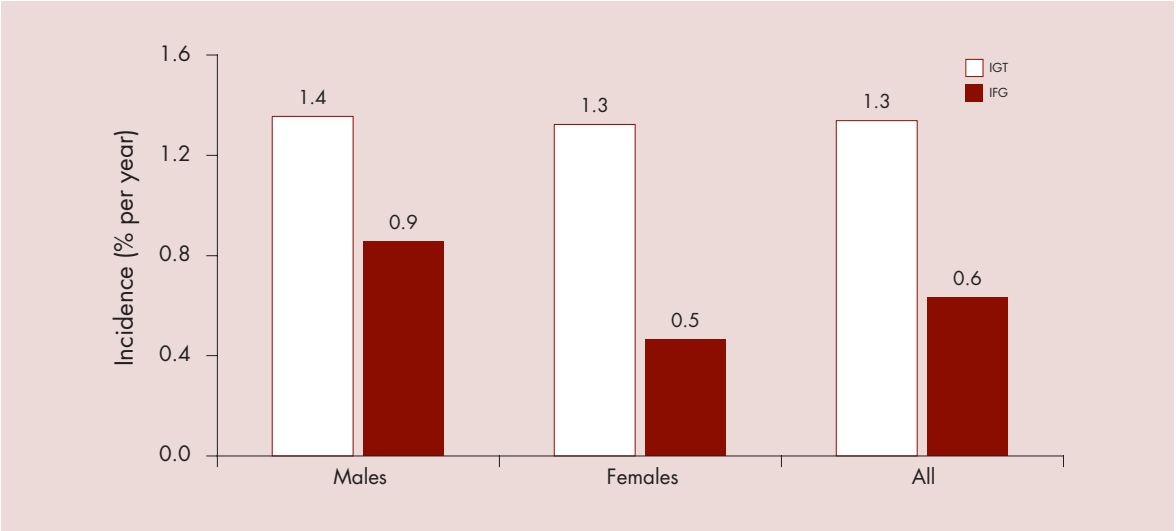


NGT – normal glucose tolerance; IFG – impaired fasting glucose; IGT – impaired glucose tolerance.

The annual incidence of IFG was higher in males than in females, while the annual incidence of IGT was similar in both sexes (Figure 2.4).

2: Diabetes and pre-diabetes

Figure 2.4. Incidence of impaired glucose tolerance and impaired fasting glucose: the AusDiab study.



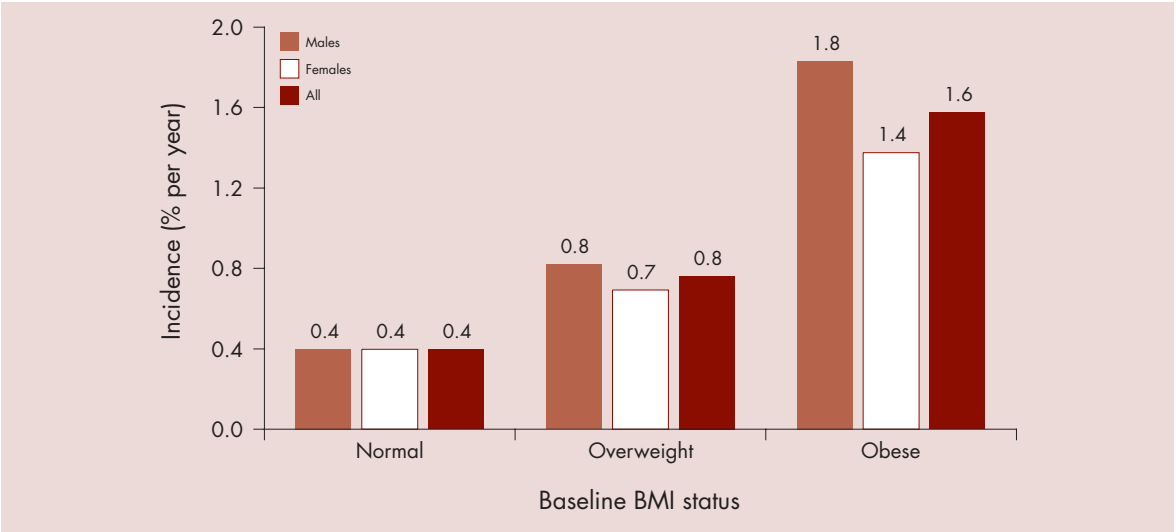
IGT - impaired glucose tolerance; IFG - impaired fasting glucose.

Risk factors for diabetes

Obesity

Compared to those with a body mass index (BMI) in the normal range at baseline, those classified as overweight and obese had an almost two- and four-fold increase, respectively, in the annual incidence of diabetes (Figure 2.5).

Figure 2.5. Incidence of diabetes according to baseline body mass index status: the AusDiab study.

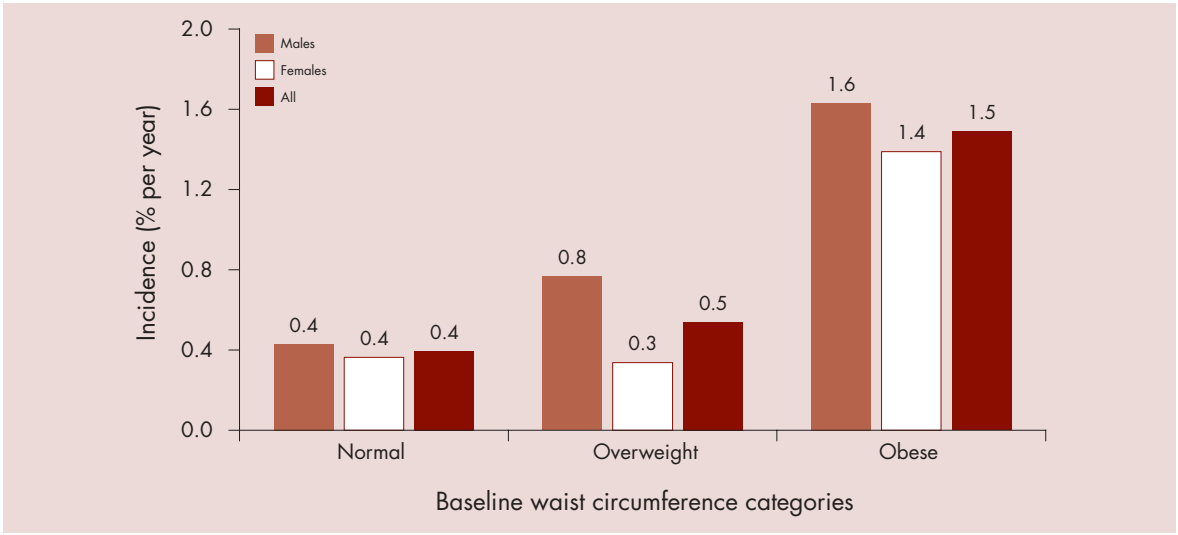


Body mass index (BMI: weight/height^2) was categorised into three groups: (i) normal: BMI<25 kg/m^2; (ii) overweight: 25–29.9 kg/m^2; and (iii) obese: ≥30 kg/m^2.

Males who were overweight or obese at baseline (defined by either BMI or waist circumference) had a higher annual incidence of diabetes than did overweight or obese females (Figures 2.5 and 2.6).

Both males and females who were classified as obese at baseline (using either waist circumference or BMI) had at least double the annual incidence of diabetes compared to those who were overweight at baseline (Figures 2.5 and 2.6).

Figure 2.6. Incidence of diabetes according to baseline waist circumference categories: the AusDiab study.



Waist circumference: (i) normal: <94.0 cm for males, <80.0 cm for females; (ii) overweight: 94–101.9 cm for males, 80.0–87.9 cm females; (iii) obese: ≥102 cm for males, ≥88.0 cm for females.

Physical activity

The annual incidence of diabetes increased in those who reported doing insufficient or sedentary levels of physical activity at baseline compared to those who reported sufficient levels of physical activity (Figure 2.7).

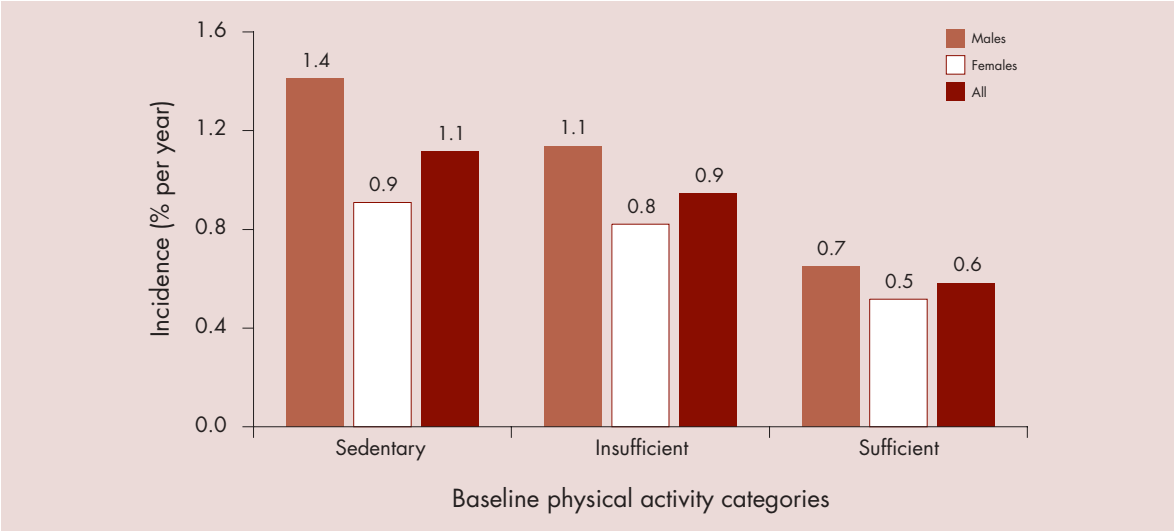
With all levels of physical activity reported at baseline, the annual incidence of diabetes was greater in males than in females (Figure 2.7).

Hypertension

The annual incidence of diabetes was three times greater in those with high blood pressure at baseline compared to those with normal blood pressure at baseline (Figure 2.8).

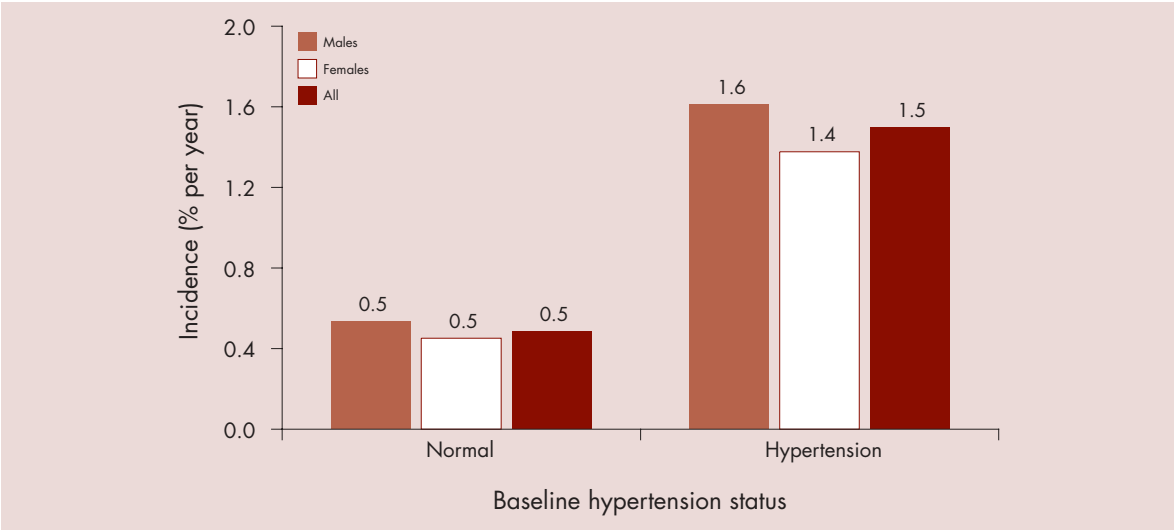
2: Diabetes and pre-diabetes

Figure 2.7. Incidence of diabetes according to baseline physical activity: the AusDiab study.



'Physical activity time' for the previous week was calculated as the sum of the time spent performing moderate activity (e.g. walking) plus double the time spent in vigorous activity (to reflect its greater intensity). Sedentary – no participation in physical activity in the previous week; insufficient – 1 to 149 minutes of physical activity in the previous week; sufficient – at least 150 minutes of physical activity in the previous week.

Figure 2.8. Incidence of diabetes according to baseline hypertension status: the AusDiab study.



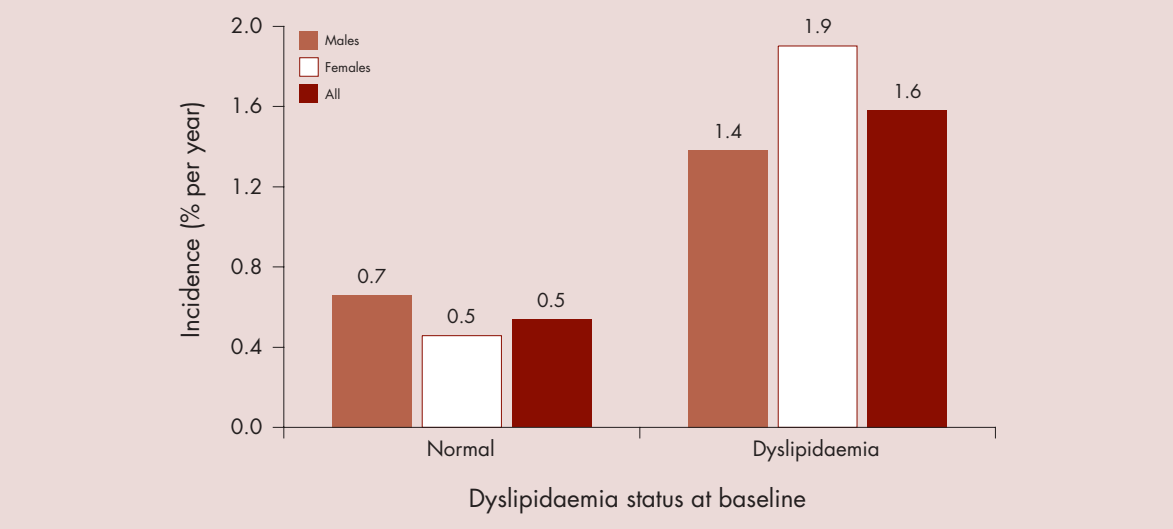
Hypertension (high blood pressure) was defined as having a blood pressure ≥140/90 mmHg and/or taking blood pressure-lowering medication.

Dyslipidaemia

Females who had dyslipidaemia at baseline had a higher annual incidence of diabetes than males with dyslipidaemia (Figure 2.9).

Males classified as having dyslipidaemia at baseline, had a two-fold greater annual incidence of diabetes compared to those with normal levels of triglycerides or high-density lipoprotein cholesterol (HDL-C). While, females with dyslipidaemia at baseline, had more than three times the annual incidence of diabetes compared to those with no dyslipidaemia at baseline (Figure 2.9).

Figure 2.9. Incidence of diabetes according to baseline dyslipidaemia status: the AusDiab study.



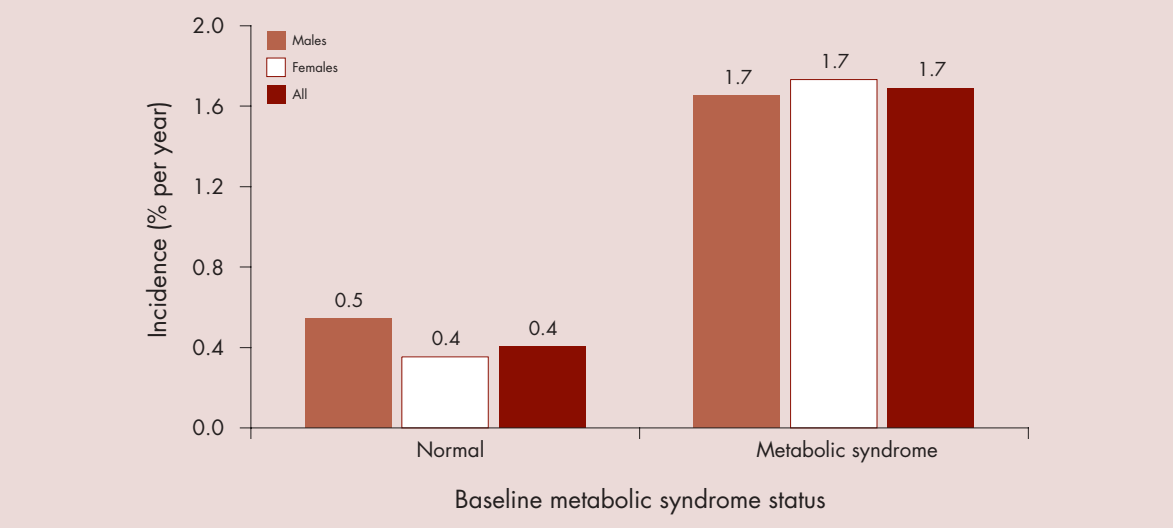
Dyslipidaemia was defined as those with triglycerides ≥2.0 mmol/l or high-density lipoprotein cholesterol levels <1.0 mmol/l.

Metabolic syndrome

The annual incidence of diabetes in those who had the metabolic syndrome according to the definition developed by the International Diabetes Federation[2] was greater than those who did not have the metabolic syndrome (Figure 2.10).

Females who were identified as having the metabolic syndrome at baseline had an annual incidence of diabetes that was approximately four times that of those who did not have the metabolic syndrome at baseline. Males who had the metabolic syndrome at baseline had an annual incidence of diabetes approximately three times that of those who did not have the metabolic syndrome at baseline (Figure 2.10).

Figure 2.10. Incidence of diabetes according to baseline metabolic syndrome status: the AusDiab study.



Metabolic syndrome was defined according to the definition by the International Diabetes Federation.

Key findings

- Every year 0.8% of Australian adults developed diabetes.
- Every day in Australia approximately 275 adults develop diabetes.
- Those with pre-diabetes were 10–20 times more likely to develop diabetes than were those with normal blood glucose levels.
- Obesity, hypertension, dyslipidaemia, physical inactivity and the metabolic syndrome each increased the risk for developing diabetes.

References

1. World Health Organization. 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, 1999.
2. Alberti KG, Zimmet PZ, Shaw J. The metabolic syndrome – a new worldwide definition. Lancet 2005;366:1059–1062.

3: Obesity

Background

Obesity is strongly linked to type 2 diabetes, and is a major risk factor not only for type 2 diabetes, but other chronic conditions such as hypertension, cardiovascular disease, dyslipidaemia, some cancers and arthritis. The most serious form of obesity is the central (abdominal) rather than peripheral form, as it is associated with substantially higher risks for diabetes and cardiovascular disease.[1,2] This chapter presents: (i) the changes in weight, body mass index (BMI) and waist circumference, and (ii) the incidence (% per year) of obesity.

Definition

Overweight and obesity were defined using the World Health Organization classification[3] for Europids, based on BMI (weight/height^2), and waist circumference. While the BMI (kg/m^2) is used as a measure of overall adiposity (Table 3.1), the waist circumference is a more accurate measure of central adiposity (Table 3.2).

Table 3.1. Body mass index classification.

	Body mass index (kg/m^2)
Normal	<25.0
Overweight	25.0–29.9
Obese	≥30.0

Table 3.2. Classification of abdominal obesity by waist circumference.

	Waist circumference (cm)	
	Males	Females
Normal	<94.0	<80.0
Overweight	94.0–101.9	80.0–87.9
Obese	≥102.0	≥88.0

Incident obesity

New (incident) cases of obesity were defined as people who were not obese (BMI <30 kg/m^2) at baseline, but were obese (BMI ≥30 kg/m^2) at follow-up.

3: Obesity

Results

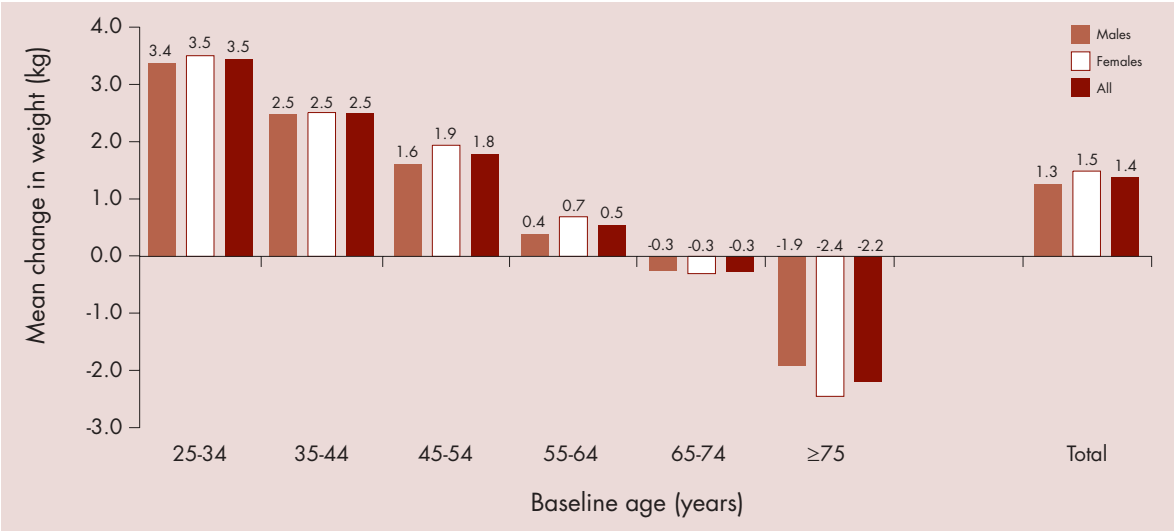
Over the period of follow-up, there was an increase in average weight, BMI and waist circumference in males and females (Figures 3.1–3.3).

For people aged 25–64 years at baseline, weight, BMI and waist circumference increased over the five years of follow-up. These increases became less with increasing age. In those aged 65–74 years at baseline, weight decreased while BMI and waist circumference increased. In those aged 75 years and older at baseline, weight and BMI decreased while waist circumference remained virtually unchanged. Those aged 25–34 years at baseline showed the greatest increase in weight, BMI and waist circumference, compared to the other age groups (Figures 3.1–3.3).

On average, those aged less than 65 years at baseline showed a weight increase of 1.8 kg, while those aged 65 years and older at baseline showed a loss in weight of 0.8 kg. The weight loss observed in the older age group may represent a loss of muscle mass, as similar losses were not observed for waist circumference over the same period (Figure 3.1).

Although the pattern of weight change was very similar in females and males, females in all age groups had slightly greater average weight changes than males. Thus, in people aged 64 years and younger at baseline, females gained slightly more weight than males, whereas, in people aged 65 years and older at baseline females lost slightly more weight than males (Figure 3.1).

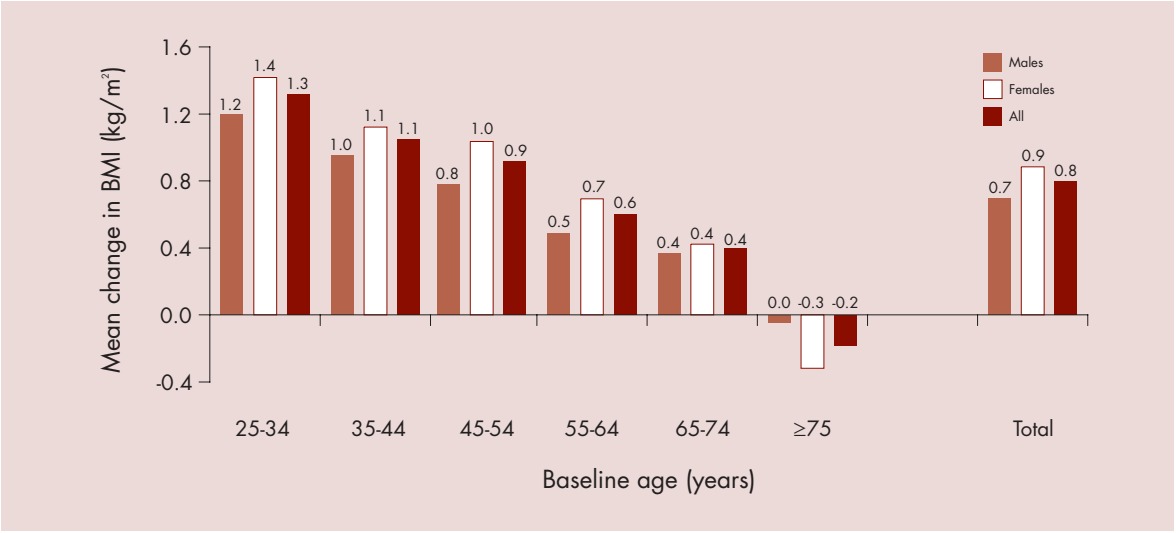
Figure 3.1. Mean weight change over five years according to baseline age: the AusDiab study.



The heights of bars on all graphs are accurate to two decimal places, but data labels are rounded to one decimal place.

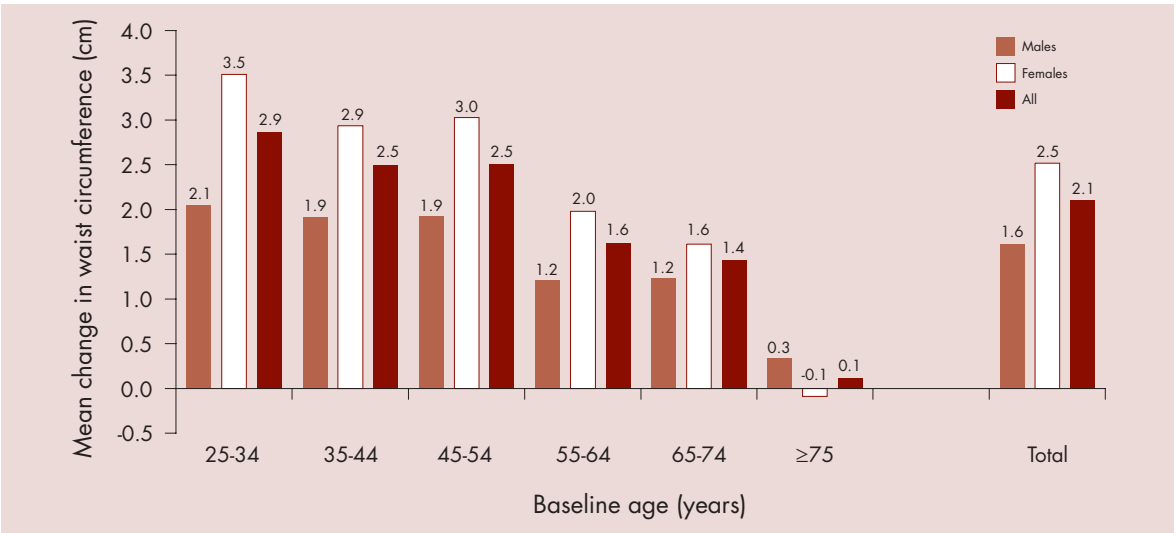
The observed changes in BMI closely followed the weight changes. The only exception was for people aged 65–74 years at baseline who showed a slight weight loss but still had an increase in BMI. This is accounted for by the slight loss in height in this age group (Figure 3.2).

Figure 3.2. Mean body mass index change over five years according to baseline age: the AusDiab study.



Greater waist circumference changes were observed in younger individuals compared with those who were older. On average, the waist circumference increase was approximately 50% greater in females than it was in males (Figure 3.3).

Figure 3.3. Mean waist circumference change over five years according to baseline age: the AusDiab study.

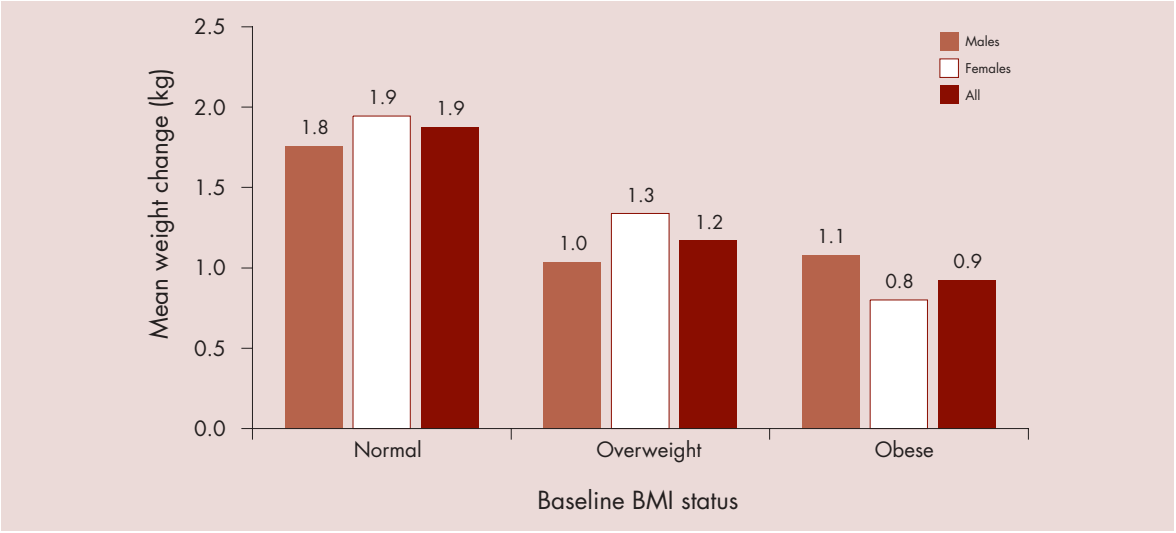


All BMI groups showed an increase in weight over the follow-up period, with females gaining more weight than males (Figure 3.4).

The greatest mean weight increases were observed in those who had a normal BMI at baseline, with lesser weight gains observed in those who were initially overweight or obese (Figure 3.4).

3: Obesity

Figure 3.4. Mean weight change over five years according to baseline body mass index status: the AusDiab study.

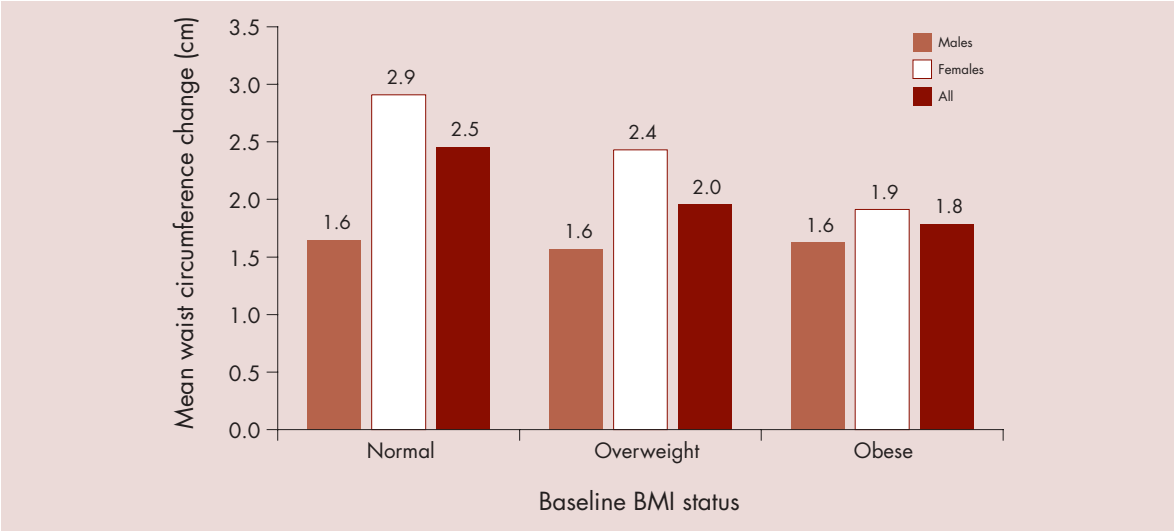


Body mass index (BMI: weight/height^2) was categorised into three groups: (i) normal: BMI<25 kg/m^2; (ii) overweight: 25–29.9 kg/m^2; and (iii) obese: ≥30 kg/m^2.

Over the period of follow-up, an increase in waist circumference was observed in all BMI groups. Compared with those with a normal BMI at baseline, overweight and obese individuals had a smaller increase in waist circumference (Figure 3.5).

For females, the pattern of increase in weight was similar to the pattern of increase in waist circumference, whereby the greatest increases were observed in the normal BMI group, with progressively lesser increases observed in the overweight and obese groups (Figures 3.4–3.5).

Figure 3.5. Mean waist circumference change over five years according to baseline body mass index status: the AusDiab study.



Body mass index (BMI: weight/height^2) was categorised into three groups: (i) normal: BMI<25 kg/m^2; (ii) overweight: 25–29.9 kg/m^2; and (iii) obese: ≥30 kg/m^2.

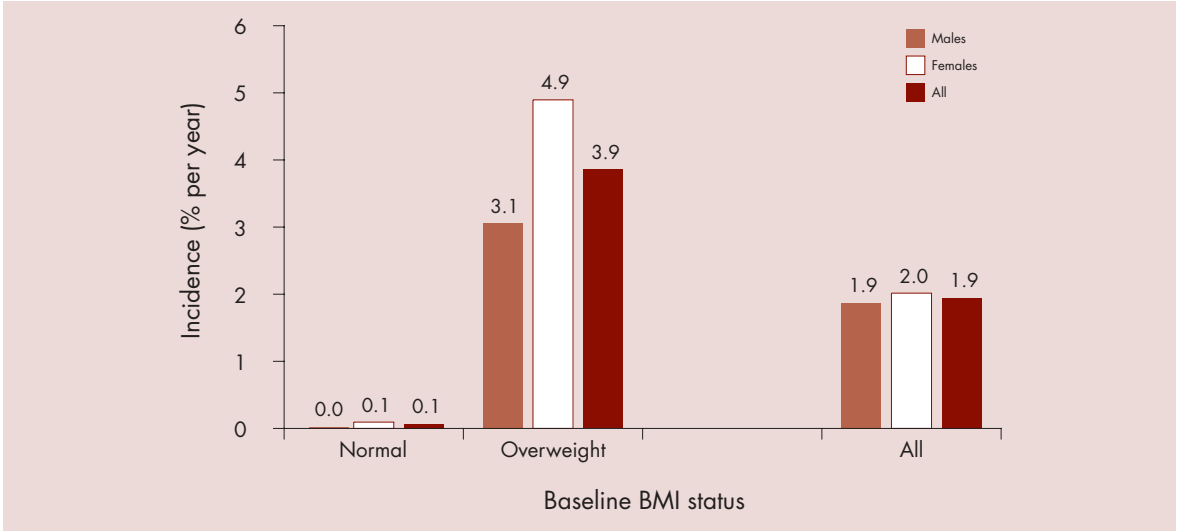
In males, however, the increase in waist circumference over the period of follow-up was unrelated to baseline BMI status, such that, the mean increase in waist circumference was 1.6 cm for all baseline BMI groups (Figures 3.4–3.5).

Incident obesity

The annual incidence of obesity is shown in Figure 3.6. Compared to those with a normal BMI at baseline, those classified as overweight were at a much higher risk of developing obesity (approximately 40 times). As shown in Figure 3.4, this is not because weight gain did not occur in those whose BMI at baseline was normal. For example, if an individual had a BMI of 24 kg/m^2, and a weight of 71 kg, they would need to gain 17 kg in weight in order to reach the obesity cut-point of 30 kg/m^2. Hence, with an average weight gain of 2 kg over five years, the chances of becoming obese from normal would have been very low.

The annual incidence of obesity in those who were overweight at baseline was higher in females than in males (Figure 3.6).

Figure 3.6. Incidence of obesity according to baseline body mass index status: the AusDiab study.



Body mass index (BMI: weight/height^2) was categorised into three groups: (i) normal: BMI<25 kg/m^2; (ii) overweight: 25–29.9 kg/m^2; and (iii) obese: ≥30 kg/m^2.

For those who were normal or overweight at baseline, 20.0% (987/4,929) had progressed to a higher weight category during follow-up.

However, for those who were obese at baseline, only 9.3% (126/1,356) had moved to a lower weight category after five years (Table 3.3).

3: Obesity

Table 3.3. Proportion of individuals classified by body mass index in 2004–05 according to baseline body mass index status: the AusDiab study.

BMI status at baseline		BMI status in 2004–05		
	n	Normal	Overweight	Obese
Normal	2,369	1,831 (77.3)	530 (22.4)	8 (0.34)
Overweight	2,560	194 (7.6)	1,917 (74.9)	449 (17.5)
Obese	1,356	6 (0.4)	120 (8.9)	1,230 (90.7)
Total	6,285	2,031	2,567	1,687

Data are n (%). Body mass index (BMI: weight/height^2) was categorised into three groups: (i) normal: BMI<25 kg/m^2; (ii) overweight: 25–29.9 kg/m^2; and (iii) obese: ≥30 kg/m^2.

For those who were normal or overweight according to waist circumference at baseline, 31.7% (1,309/4,133) had progressed to a higher waist circumference category at follow-up. However, for those who were obese at baseline, only 13.0% (282/2,163) had moved to a lower weight category after five years (Table 3.4).

Table 3.4. Proportion of individuals classified by waist circumference categories in 2004–05 according to baseline waist circumference categories: the AusDiab study.

Waist circumference categories at baseline		Waist circumference categories in 2004–05		
	n	Normal	Overweight	Obese
Normal	2,496	1,752 (70.2)	628 (25.2)	116 (4.7)
Overweight	1,637	301 (18.4)	771 (47.1)	565 (34.5)
Obese	2,163	44 (2.0)	238 (11.0)	1,881 (87.0)
Total	6,296	2,097	1,637	2,562

Data are n (%). Waist circumference: (i) normal: <94.0 cm for males, <80.0 cm for females; (ii) overweight: 94–101.9 cm for males, 80.0–87.9 cm females; (iii) obese: ≥102 cm for males, ≥88.0 cm for females.

Key findings

- Those aged less than 65 years showed an average weight increase of 1.8 kg over five years. People aged 65 years and older showed a loss in weight of 0.8 kg over the same period.
- The average gain in waist circumference in Australians over five years was 2.1 cm.
- The average change in waist circumference was greater in females than in males for all age groups.
- Younger people gained more weight and had a greater increase in waist circumference than did older people.
- Twice as many overweight people became obese as reverted to normal.

References

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2. Alberti K. The clinical implications of impaired glucose tolerance. Diabetic Medicine 1996;13:927–937.
3. World Health Organization. Obesity – Preventing and Managing the Global Epidemic: Report of a WHO Expert Committee. Geneva: World Health Organisation, 1998.

4: Blood pressure

Background

High blood pressure (hypertension) represents an important risk factor for cardiovascular and renal disease in the general population. Amongst those with diabetes, high blood pressure is a risk factor for microvascular complications as well as for cardiovascular disease. Thus, high blood pressure is of major significance to the whole population.[1,2] The AusDiab study conducted in 1999–2000 revealed that one in three Australians aged 25 years and older were classified as being hypertensive (either having a blood pressure ≥140/90 mmHg or taking blood pressure-lowering medication). The 5-year follow-up AusDiab study provided an opportunity to measure the development of hypertension among Australians.

This chapter presents: (i) the incidence (% per year) of hypertension, and (ii) whether the incidence of hypertension differed between males and females, people of different ages, people with and without diabetes, people who were overweight or obese and for smokers and non-smokers.

Definitions

Hypertension

Hypertension (high blood pressure) was defined as having a blood pressure ≥140/90 mmHg and/or taking blood pressure-lowering medication in accordance with the World Health Organization guidelines.[3] Classification of blood pressure is outlined in Table 4.1.

Table 4.1. Classification of blood pressure.

	Systolic blood pressure (mmHg)		Diastolic blood pressure (mmHg)		Blood pressure-lowering medication
Normal	<140	and	<90	and	No
Hypertension	≥140	or	≥90	or	Yes

Incident hypertension

New (incident) cases of hypertension were defined as people who were classified with normal blood pressure at baseline, but had developed hypertension at follow-up.

Results

Blood pressure

The incidence of hypertension was 3.0% per year (3.4% per year for males and 2.7% per year for females).

There was a mean 4.6 mmHg decrease in systolic blood pressure between 1999–2000 and 2004–05.

For those classified with normal blood pressure (<140/90 mmHg and not taking blood pressure medication) at baseline, 13.9% had developed hypertension at follow-up.

For those classified with hypertension at baseline, 18.0% were classified as having normal blood pressure at follow-up (Table 4.2).

Table 4.2. Proportion of individuals classified with hypertension in 2004–05 according to baseline hypertension status: the AusDiab study.

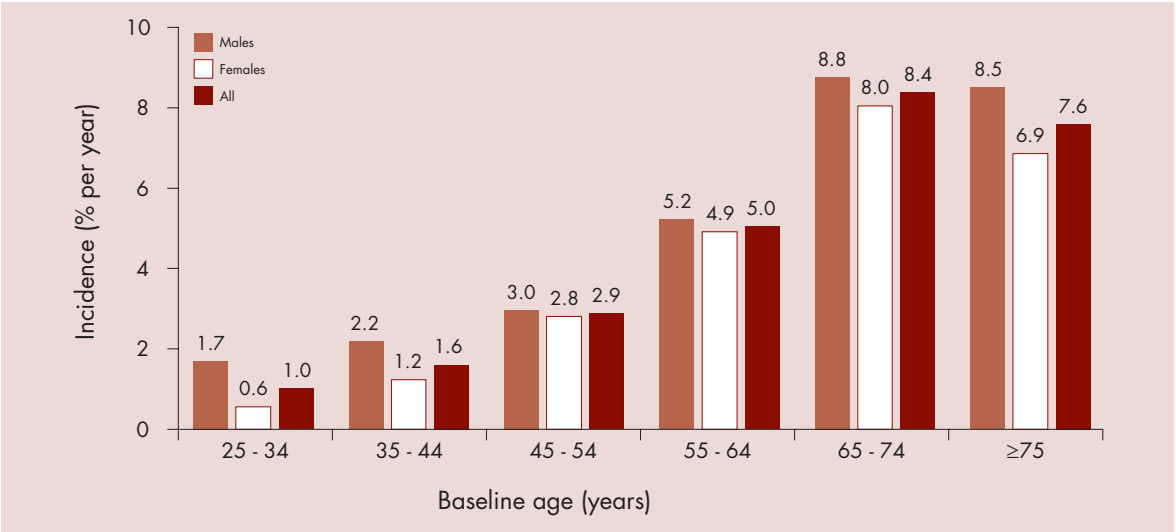
Hypertension status at baseline		Hypertension status in 2004–05	
	n	Normal blood pressure	Hypertension
Normal blood pressure	4,353	3,749 (86.1)	604 (13.9)
Hypertension	1,965	354 (18.0)	1,611 (82.0)
Total	6,318	4,103	2,215

Data are n (%). Hypertension (high blood pressure) is defined as having a blood pressure ≥140/90 mmHg and/or taking blood pressure-lowering medication.

The incidence of hypertension increased according to age, with the incidence of hypertension ranging from 1.0% per year for people aged 25–34 years at baseline to 8.4% per year for people aged 65–74 years at baseline (Figure 4.1).

In each age group, males had a higher incidence of hypertension compared to females, and these differences were particularly evident for those aged 25–44 years and 75 years and older at baseline. There was little difference between males and females for the annual incidence of hypertension in those aged 45–64 years at baseline (Figure 4.1).

Figure 4.1. Incidence of hypertension according to baseline age: the AusDiab study.



The heights of bars on all graphs are accurate to two decimal places, but data labels are rounded to one decimal place.

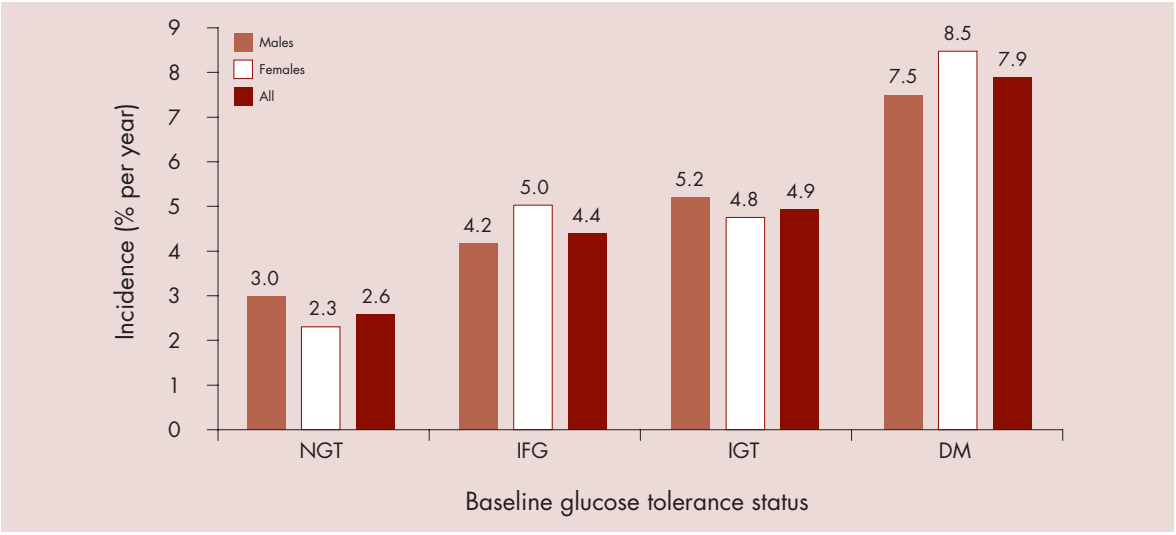
4: Blood pressure

Compared to people with normal glucose tolerance (NGT) at baseline, the annual incidence of hypertension was greater for people with impaired fasting glucose (IFG), impaired glucose tolerance (IGT) and diabetes; with the annual incidence of hypertension being approximately three times greater among those with diabetes at baseline than among those with NGT at baseline (Figure 4.2).

Among those with IFG or diabetes at baseline, males had a lower annual incidence of hypertension compared to females. However, among those with NGT or IGT at baseline, the annual incidence of hypertension was higher in males than in females (Figure 4.2).

The impact of diabetes on the incidence of hypertension was greater for females than for males. For females, the annual incidence of hypertension was nearly four times greater among those with diabetes at baseline compared to those with NGT at baseline (8.5% vs 2.3%). However, for males, the annual incidence among those who had diabetes at baseline was only a little over twice the incidence of those who had NGT at baseline (7.5% vs 3.0%) (Figure 4.2).

Figure 4.2. Incidence of hypertension according to baseline glucose tolerance status: the AusDiab study.



NGT – normal glucose tolerance; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; DM – diabetes mellitus.

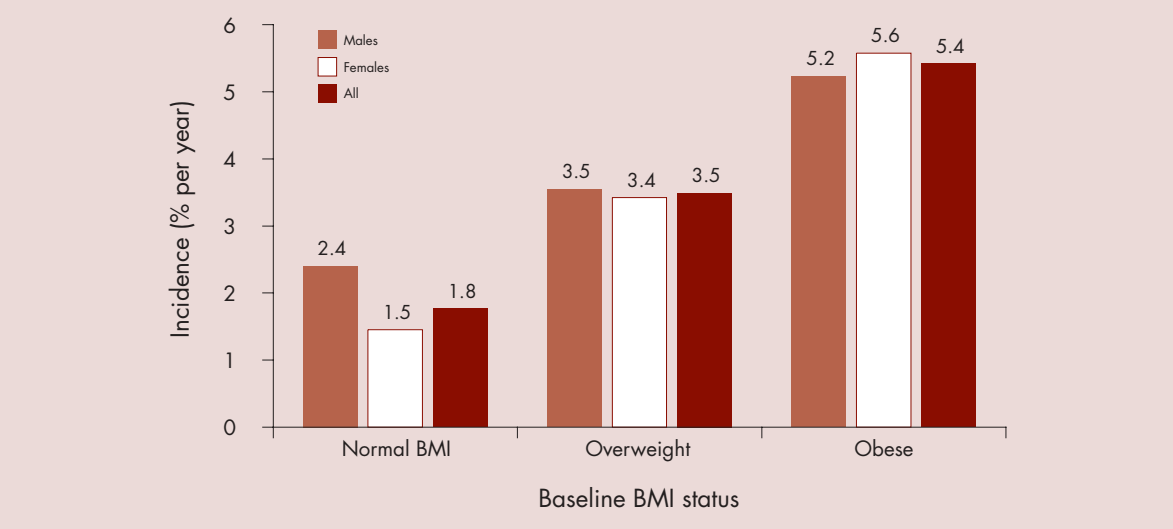
The incidence of hypertension was higher in people who were overweight (3.5% per year) or obese (5.4% per year) at baseline, compared to those who had a normal body mass index (BMI) at baseline (1.8% per year) (Figure 4.3).

For those who had a normal baseline BMI, males had a higher incidence of hypertension compared to females (2.4% vs 1.5% per year) (Figure 4.3).

For those who were obese at baseline, males had a lower incidence of hypertension compared to females (5.2% vs 5.6% per year) (Figure 4.3).

The impact of obesity on the incidence of hypertension was greater for females than for males. For females, the annual incidence of hypertension was nearly four times greater among those who were obese at baseline compared to those who had a normal BMI at baseline (5.6% vs 1.5%). However, for males, the annual incidence among those who were obese at baseline was only a little over twice the incidence of those who had a normal BMI at baseline (5.2% vs 2.4%) (Figure 4.3).

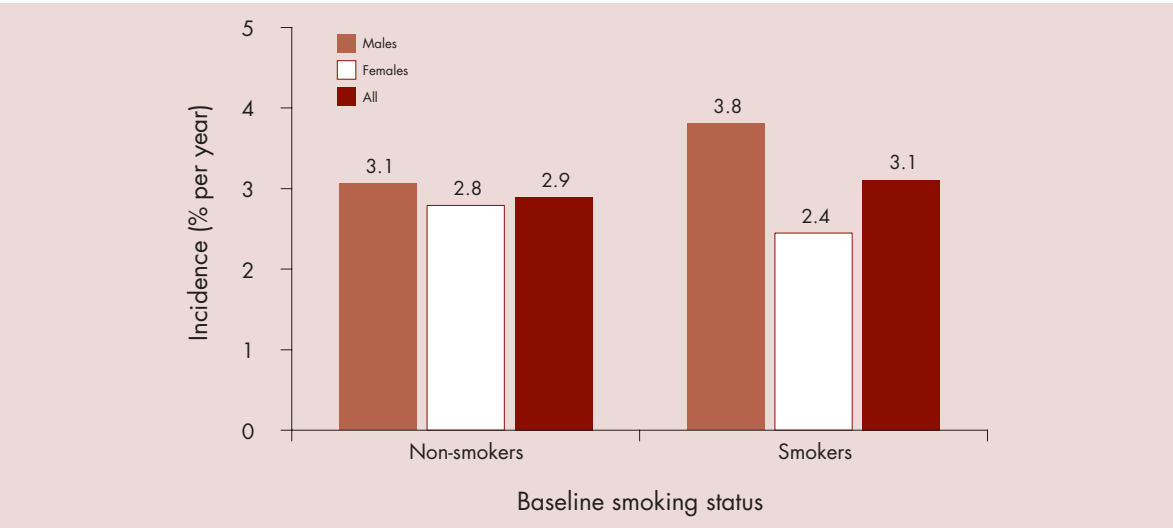
Figure 4.3. Incidence of hypertension according to baseline body mass index status: the AusDiab study.



BMI – body mass index; where, (i) normal was a BMI of <25 kg/m^2, (ii) overweight was a BMI of 25–29.9 kg/m^2 and (iii) obese was a BMI of ≥30 kg/m^2.

The incidence of hypertension was higher in smokers than it was in non-smokers (3.1% *vs* 2.9% per year). Among males, the incidence of hypertension was higher for smokers compared to non-smokers (3.8% vs 3.1% per year). However, among females the incidence of hypertension was lower in smokers compared to non-smokers (2.4% vs 2.8% per year) (Figure 4.4).

Figure 4.4. Incidence of hypertension according to baseline smoking status: the AusDiab study.



'Smokers' included people who were either current smokers or ex-smokers at baseline.

Key findings

- Every year, 3.0% of adults developed high blood pressure.
- The risk of developing high blood pressure was 1.0% per year for people aged 25–34 years and increased to 8.4% per year for people aged 65–74 years.
- The risk of developing high blood pressure was greater for people with pre-diabetes and diabetes, and for those who were overweight or obese. Each of these risk factors (diabetes and obesity) had a greater impact on the development of hypertension for females than for males.
- For smokers, the risk of high blood pressure was greater in males than in females.

References

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5: Metabolic syndrome

Background

The metabolic syndrome is characterised by central or abdominal (visceral and retroperitoneal) obesity and clustering of other cardiovascular risk factors including abnormal glucose tolerance (diabetes, impaired fasting glucose (IFG) or impaired glucose tolerance (IGT)), raised triglycerides, decreased high-density lipoprotein cholesterol (HDL-C), elevated blood pressure, and hyperinsulinaemia with underlying insulin resistance. The clustering of these risk factors together confers a higher risk of diabetes and cardiovascular disease. This chapter presents the incidence (% per year) of the metabolic syndrome and the impact of various risk factors on the development of the metabolic syndrome.

Definition

Metabolic syndrome

The metabolic syndrome was defined according to the International Diabetes Federation definition.[1] Classification of the metabolic syndrome is outlined in Table 5.1.

Table 5.1. Classification of the metabolic syndrome.

Component	Threshold
Waist circumference	Europids: ≥94 cm males, ≥80 cm females South and South-East Asians: ≥90 cm males, ≥80 cm females
Plus two or more of the following:	
• Raised triglycerides	≥1.7 mmol/l or specific treatment of this lipid abnormality
• Reduced HDL-cholesterol	<1.03 mmol/l in males; <1.29 mmol/l in females or specific treatment for this lipid abnormality
• Raised blood pressure	Systolic ≥130 mmHg or diastolic ≥85 mmHg or treatment of previously diagnosed hypertension
• Raised plasma glucose	Fasting plasma glucose ≥5.6 mmol/l or previously diagnosed type 2 diabetes

Incident metabolic syndrome

New (incident) cases of the metabolic syndrome were defined as people who did not meet the criteria for the metabolic syndrome at baseline, but satisfied the criteria at follow-up.

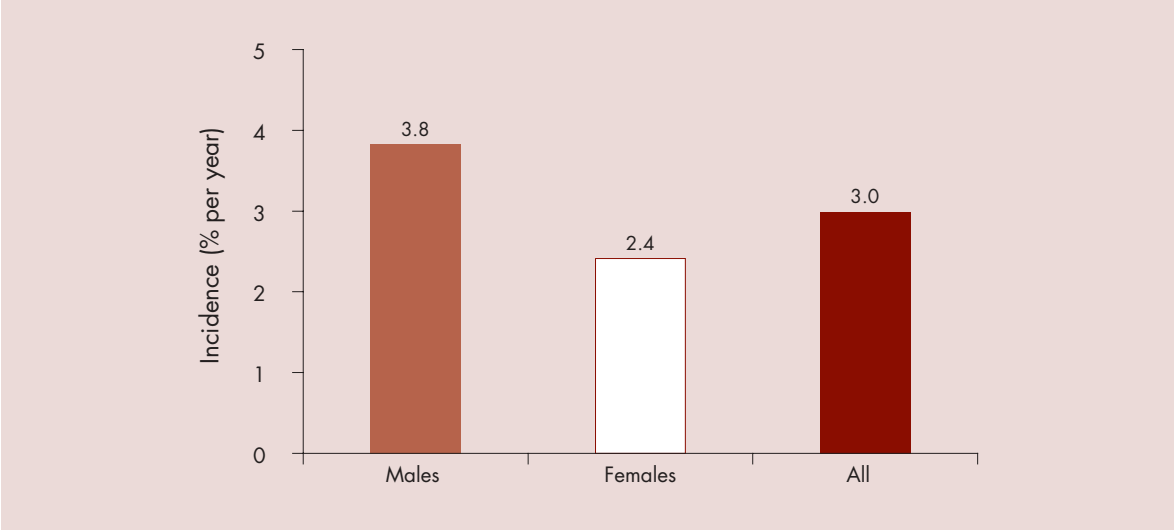
5: Metabolic syndrome

Results

The prevalence of the metabolic syndrome at baseline was 28.6%.

The annual incidence of the metabolic syndrome in those who did not meet the criteria for the metabolic syndrome at baseline was one and a half times higher in males than in females (Figure 5.1).

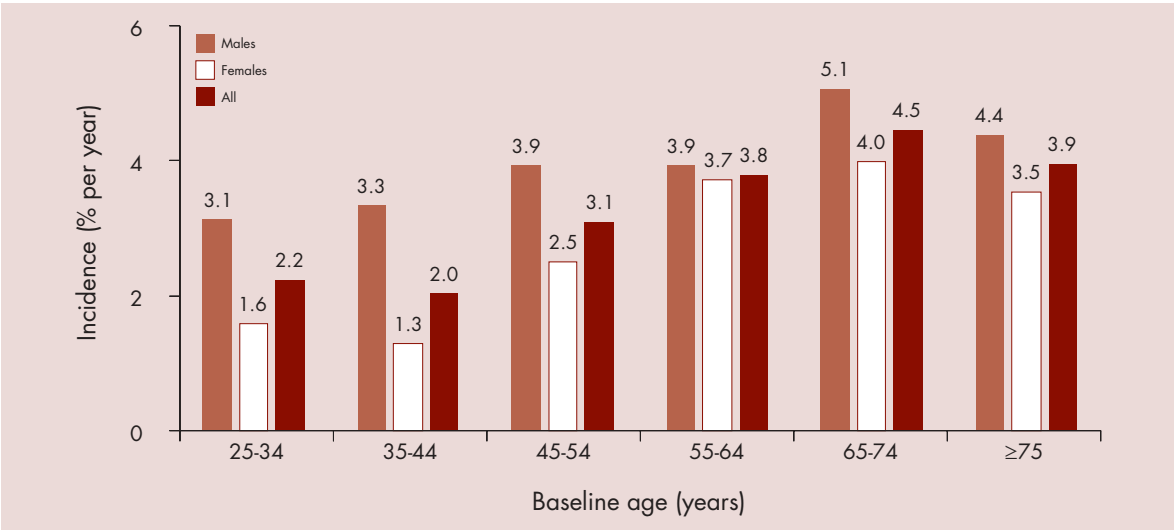
Figure 5.1. Incidence of the metabolic syndrome according to sex: the AusDiab study.



The heights of bars on all graphs are accurate to two decimal places, but data labels are rounded to one decimal place.

In males, the annual incidence of the metabolic syndrome increased with age up to 74 years. In females, the incidence of the metabolic syndrome was more variable and was lowest in those aged 35–44 and highest in those aged 65–74 at baseline, compared to other age groups (Figure 5.2).

Figure 5.2. The incidence of the metabolic syndrome according to baseline age: the AusDiab study.



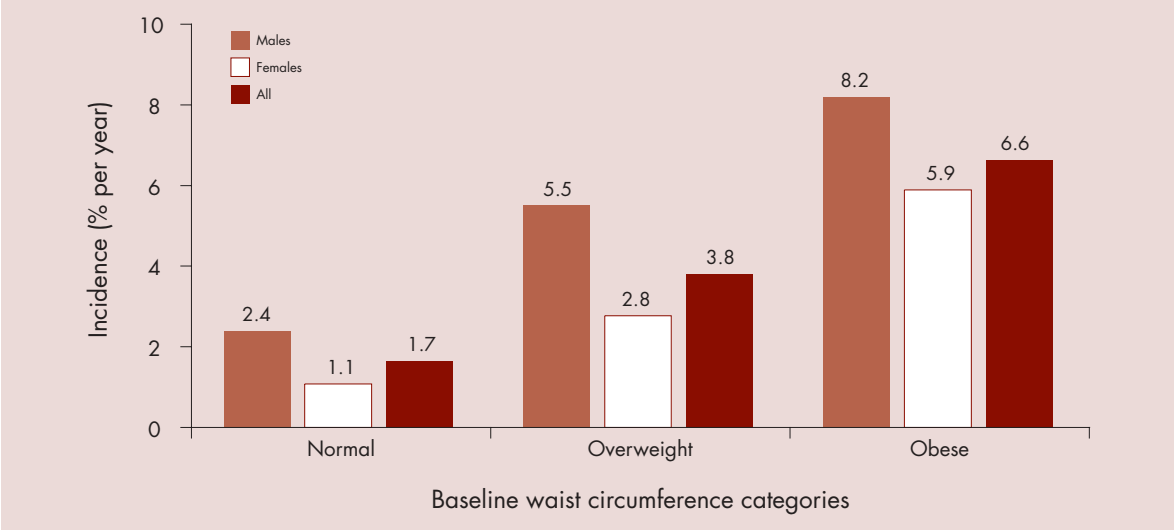
Within each age group the annual incidence of the metabolic syndrome was greater in males than in females (Figure 5.2).

The annual incidence of the metabolic syndrome increased as waist circumference at baseline increased (Figure 5.3).

Compared to those who had a normal waist circumference at baseline, those who were overweight were between two and three times more likely to develop the metabolic syndrome, while those who were obese were three times more likely to develop the metabolic syndrome over five years (Figure 5.3).

Within each of the waist circumference categories, males had a greater annual incidence of the metabolic syndrome than did females (Figure 5.3).

Figure 5.3. Incidence of the metabolic syndrome according to baseline waist circumference categories: the AusDiab study.



Waist circumference: (i) normal: <94.0 cm for males, <80.0 cm for females; (ii) overweight: 94–101.9 cm for males, 80.0–87.9 cm females; (iii) obese: ≥102 cm for males, ≥88.0 cm for females.

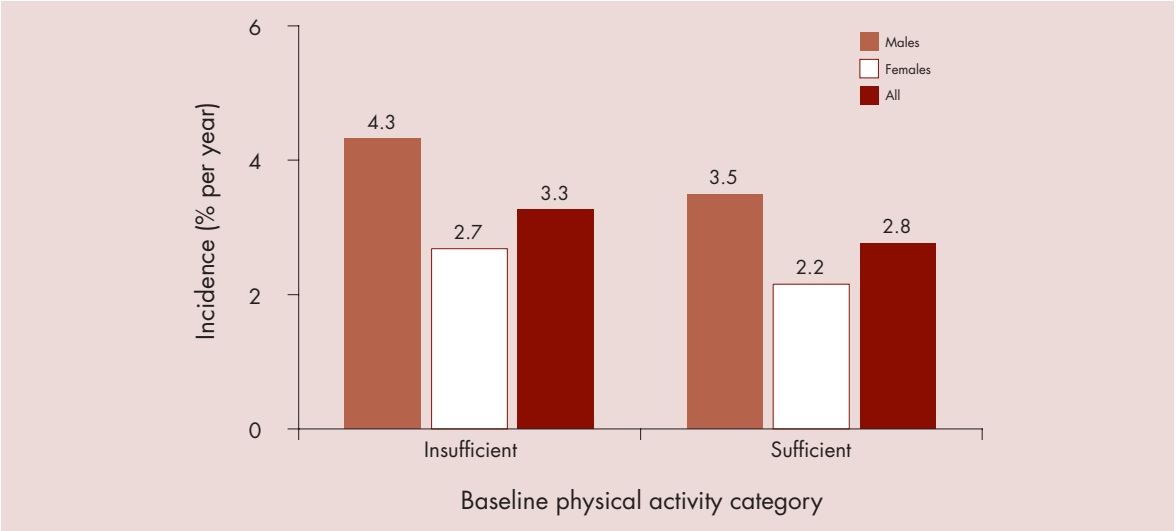
A similar pattern was observed when the incidence of the metabolic syndrome was examined according to baseline body mass index (BMI). The incidence of the metabolic syndrome in those who were categorised as normal (BMI <25 kg/m^2), overweight (BMI 25–29.9 kg/m^2) and obese (BMI ≥30 kg/m^2) at baseline was 1.2%, 4.6% and 7.2% per year, respectively.

The incidence of the metabolic syndrome was slightly higher among those who reported insufficient physical activity at baseline compared to those who reported sufficient physical activity at baseline (Figure 5.4).

The annual incidence of the metabolic syndrome was greater in those with diabetes at baseline than in those with normal glucose tolerance at baseline. For those with pre-diabetes, the incidence of the metabolic syndrome was midway between the incidence in those with normal glucose tolerance at baseline and those with diabetes at baseline (Figure 5.5).

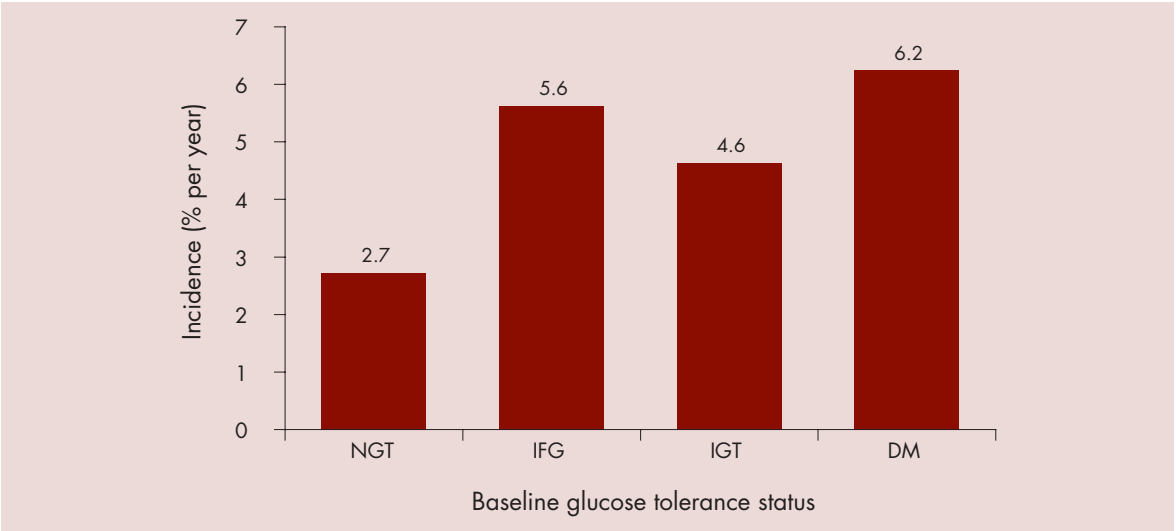
5: Metabolic syndrome

Figure 5.4. The incidence of the metabolic syndrome according to baseline physical activity: the AusDiab study.



'Physical activity time' for the previous week was calculated as the sum of the time spent performing moderate activity (e.g. walking) plus double the time spent in vigorous activity (to reflect its greater intensity). Insufficient – less than 149 minutes of physical activity in the previous week; sufficient – at least 150 minutes of physical activity in the previous week.

Figure 5.5. Incidence of the metabolic syndrome according to baseline glucose tolerance status: the AusDiab study.



NGT – normal glucose tolerance; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; DM – diabetes mellitus.
The graph was not presented according to sex as there were too few individuals in each of the categories.

Key findings

- People who were obese were six times more likely to develop the metabolic syndrome than were those of normal weight.
- People with diabetes were twice as likely to develop the metabolic syndrome than were those with normal blood glucose levels.
- Physically inactive people were also at increased risk of developing the metabolic syndrome.
- Increasing age was associated with an increased risk of developing the metabolic syndrome.
- Across all ages and all weight and physical activity categories, males were at a higher risk of developing the metabolic syndrome than were females.

Reference

1. Alberti KG, Zimmet P, Shaw J. The metabolic syndrome – a new worldwide definition. Lancet 2005;366:1059–1062.

6: Chronic kidney disease

This reprinted version of the report includes changes from the original version and these are highlighted by bold text.

Background

Chronic kidney disease is common in the general community and causes significant physical and mental disability.[1,2] Individuals with chronic kidney disease are at risk of experiencing end-stage kidney failure requiring dialysis or transplantation and are also predisposed to develop premature cardiovascular disease with an increased risk of death due to heart attack or stroke.[3,4]

The number of new cases (incidence) of end-stage kidney disease in Australia is currently 95/million population per annum, with diabetes being the leading cause.[5] Currently 30% of all new end-stage kidney disease is due to diabetes,[5] compared with 17% in 1994.[6] The other common causes of end-stage kidney disease include glomerulonephritis (25%) and vascular kidney disease related to hypertension and/or atherosclerosis (13%).[5]

In the first phase of AusDiab, we demonstrated that 16% of Australian adults have chronic kidney disease.[1] The second phase of AusDiab has enabled, for the first time, the opportunity to determine the rate at which new cases of chronic kidney disease emerge among the 84% of Australian adults who were free from chronic kidney disease at the time of the initial survey at baseline – known as the incidence. By determining the incidence of chronic kidney disease, the AusDiab study will provide information unique in the world, and crucial in our bid to prevent the steady growth in chronic kidney disease and end-stage kidney disease that is currently evident in Australia and around the globe. This chapter presents: (i) the incidence (% per year) of both chronic kidney disease and early kidney damage, and (ii) the risk factors associated with these conditions.

Definitions

Impaired glomerular filtration rate

Chronic kidney disease is defined as present when there is impaired kidney function. The standard measure of kidney function is the glomerular filtration rate (GFR). GFR can be estimated from the results of a blood test (so called 'estimated' GFR or eGFR) and an impaired eGFR is defined as an eGFR of <60 ml/min/1.73m^2.[7] In the AusDiab study, the eGFR has been calculated using the abbreviated MDRD formula.[8]

The abbreviated MDRD formula utilises the results of a simple blood test – serum creatinine – in addition to gender and age to calculate the estimated GFR. The laboratory examinations in the baseline and follow-up surveys were performed by different laboratories: HITECH Pathology (Melbourne, Victoria, Australia) in 1999–2000 and Gribbles Pathology (Melbourne, Victoria, Australia) in 2004–05. A random selection of frozen blood samples that were collected during the baseline survey were re-analysed in 2004 by Gribbles Pathology. While there was a strong correlation between the two samples (r=0.88), the Gribbles assay resulted in a slightly higher serum creatinine compared to HITECH (mean difference 6.8 μmol/L, 95% confidence intervals 5.6 - 8.0). While this difference is small, the difference is magnified when the GFR is estimated due to the logarithmic nature of the MDRD formula. Therefore using a regression analysis of the paired samples, the serum creatinine results obtained by Gribbles in 2004–05 were adjusted to correspond with the HITECH assay before estimation of GFR.

Incident impaired glomerular filtration rate

New (incident) cases of impaired GFR were defined as individuals who had a normal eGFR (>60 ml/min/1.73m^2) at baseline, but had an eGFR of <60 ml/min/1.73m^2 at follow-up.

Albuminuria

Early kidney disease can manifest as the leakage of protein into the urine without any impairment of kidney function. The earliest manifestation of an excessive leakage of protein into the urine can be detected by measuring the urinary albumin levels and is called albuminuria. We considered albuminuria to be present if the spot urine albumin:creatinine ratio was ≥2.5 mg/mmol for males and ≥3.5 mg/mmol for females. Albuminuria is a recognised early risk factor for the development of chronic kidney disease and additionally is an important risk factor for cardiovascular disease and mortality.[9-11]

Incident albuminuria

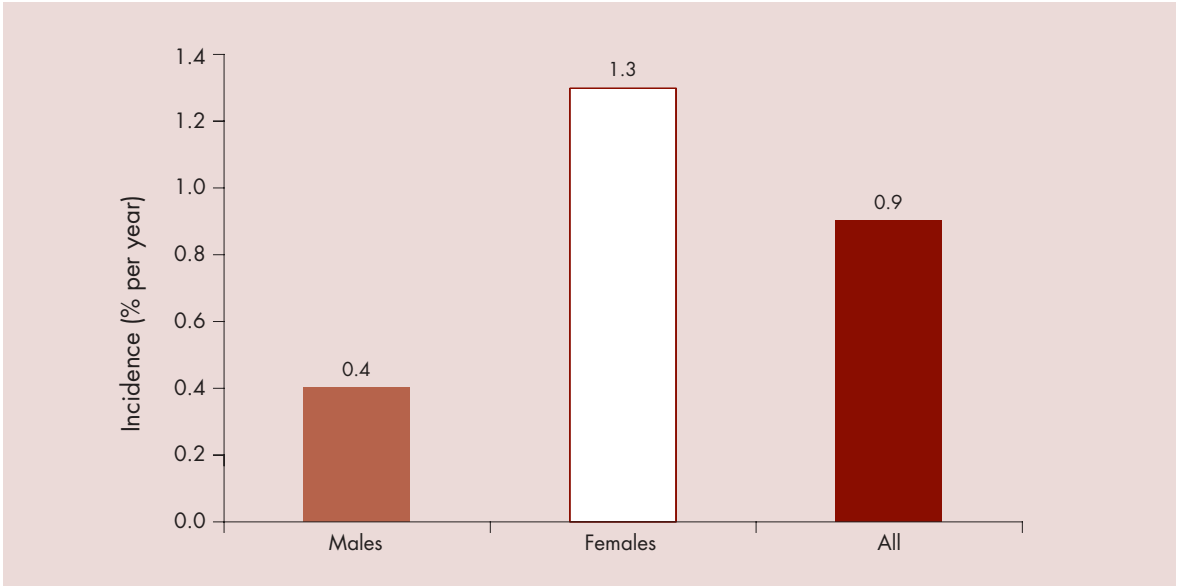
New (incident) cases of albuminuria were defined as people who had normal albumin:creatinine levels in the urine at baseline, but had an abnormal spot urine albumin:creatinine ratio (≥2.5 mg/mmol for males and ≥3.5 mg/mmol for females) at follow-up.

Results

Incidence of impaired glomerular filtration rate

The incidence of impaired GFR was **0.9%** per year, with the incidence in females triple that of males (**1.3%** vs **0.4%** per year) (Figure 6.1).

Figure 6.1. Incidence of impaired glomerular filtration rate according to sex: the AusDiab study.



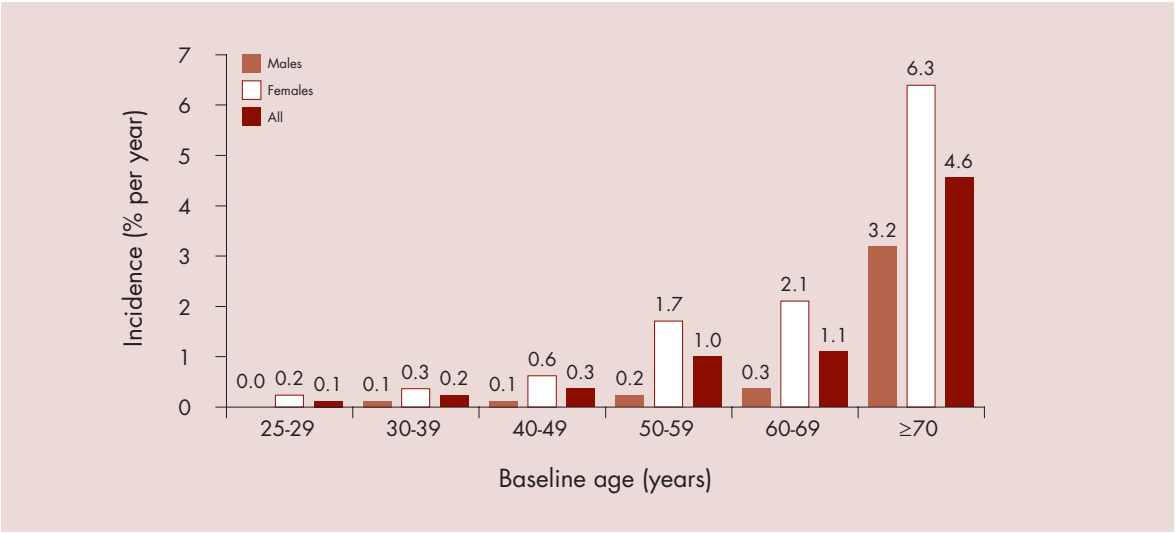
The heights of bars on all graphs are accurate to two decimal places, but data labels are rounded to one decimal place.

The annual incidence of impaired GFR according to sex and age group at baseline are presented in Figure 6.2. In males, **the incidence began to rise over the age of 70 years.**

6: Chronic kidney disease

In females, the annual incidence for impaired GFR changed little with age until the age of **50 years**, where there was a three-fold increase in those aged **50–59 years**, compared to those aged less than **50 years**. **There was a further large increase in incidence for impaired GFR in females aged 70 years and older**. In all age groups, the incidence of impaired GFR was higher in females than in males.

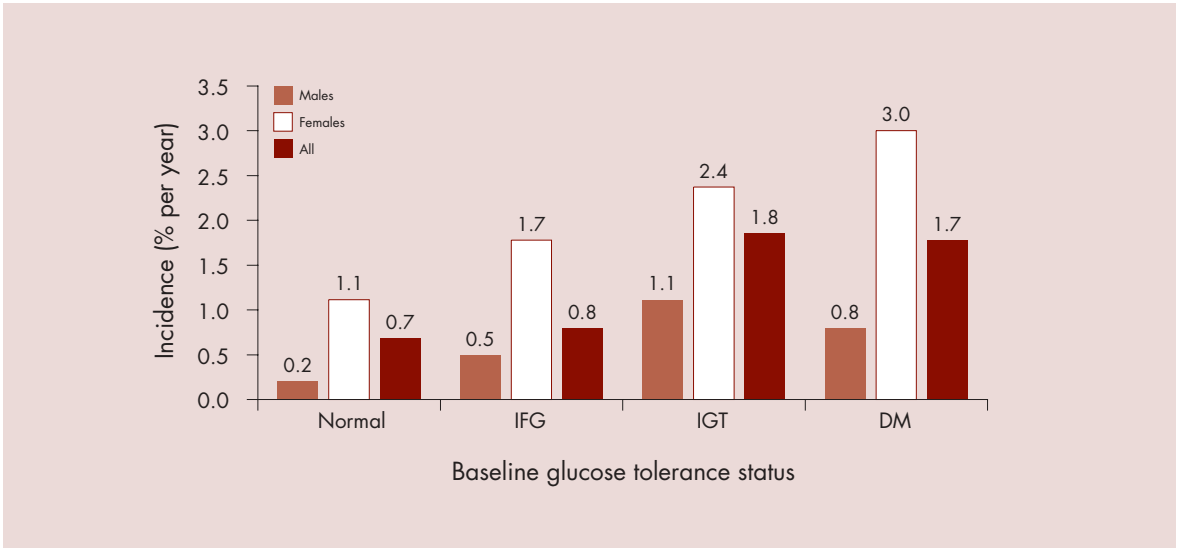
Figure 6.2. Incidence of impaired glomerular filtration rate according to baseline age: the AusDiab study.



The annual incidence of impaired GFR was higher in those with diabetes at baseline compared to those with normal glucose tolerance (NGT) at baseline (**1.7%** vs **0.7%**). **For those with impaired fasting glucose (IFG) at baseline, the incidence of impaired GFR was similar to normal subjects while in those with impaired glucose tolerance (IGT) the incidence was similar to those with diabetes at baseline** (Figure 6.3). In all groups the annual incidence of impaired GFR in females was **at least** double that observed in males.

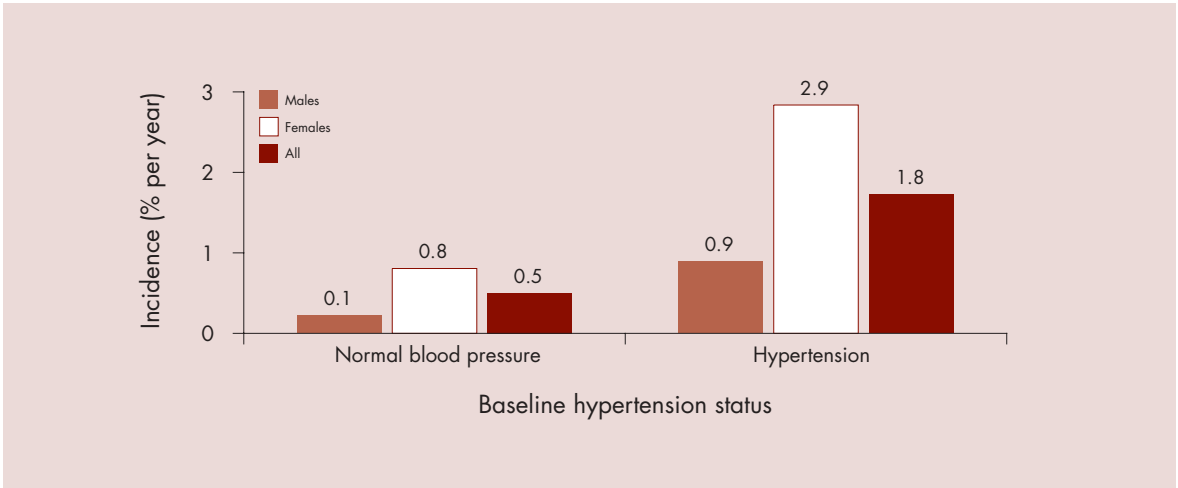
The annual incidence of impaired GFR in persons with or with out hypertension is presented in Figure 6.4. More than **three times** as many people with hypertension developed impaired GFR than did people with normal blood pressure. In both groups the number of new cases in females was **more than triple** that seen in males.

Figure 6.3. Incidence of impaired glomerular filtration rate according to baseline glucose tolerance status: the AusDiab study.



IFG – impaired fasting glucose; IGT – impaired glucose tolerance; DM – diabetes mellitus.

Figure 6.4. Incidence of impaired glomerular filtration rate according to baseline hypertension status: the AusDiab study.



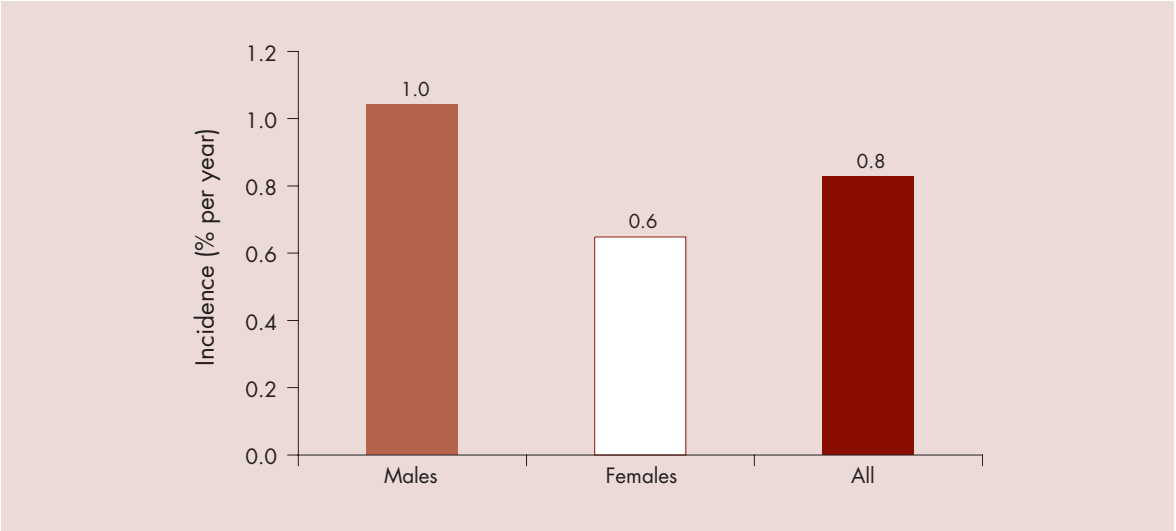
Hypertension (high blood pressure) was defined as having a blood pressure ≥140/90 mmHg and/or taking blood pressure-lowering medication.

6: Chronic kidney disease

Incidence of albuminuria

The incidence of albuminuria was 0.83% per year, with the incidence of albuminuria in males almost double that of females (Figure 6.5).

Figure 6.5. Incidence of albuminuria according to sex: the AusDiab study.



The annual incidence of albuminuria according to baseline age and sex is presented in Figure 6.6. In males, the annual incidence of albuminuria increased according to baseline age, with a substantial increase in incidence in those aged 70 years and older. In females, the annual incidence of albuminuria was higher in those aged 25–29 years compared with those aged 30–39 years, and then in those aged older than 40 years, the incidence increased steadily with age. In all age groups, except those aged 25–29 years, the annual incidence of albuminuria was higher in males compared with females.

The annual incidence of albuminuria was one and a half to two times higher in people with pre-diabetes (IFG or IGT) at baseline compared with people with NGT at baseline. People with diabetes at baseline had an incidence of albuminuria of 3.1% per year, which was five times higher than that observed in people with NGT at baseline (0.6% per year). In people with IGT and diabetes at baseline, males had a higher incidence of albuminuria compared with females, whereas for those with IFG at baseline, males had a lower incidence of albuminuria compared with females (Figure 6.7).

Figure 6.6. Incidence of albuminuria according to baseline age: the AusDiab study.

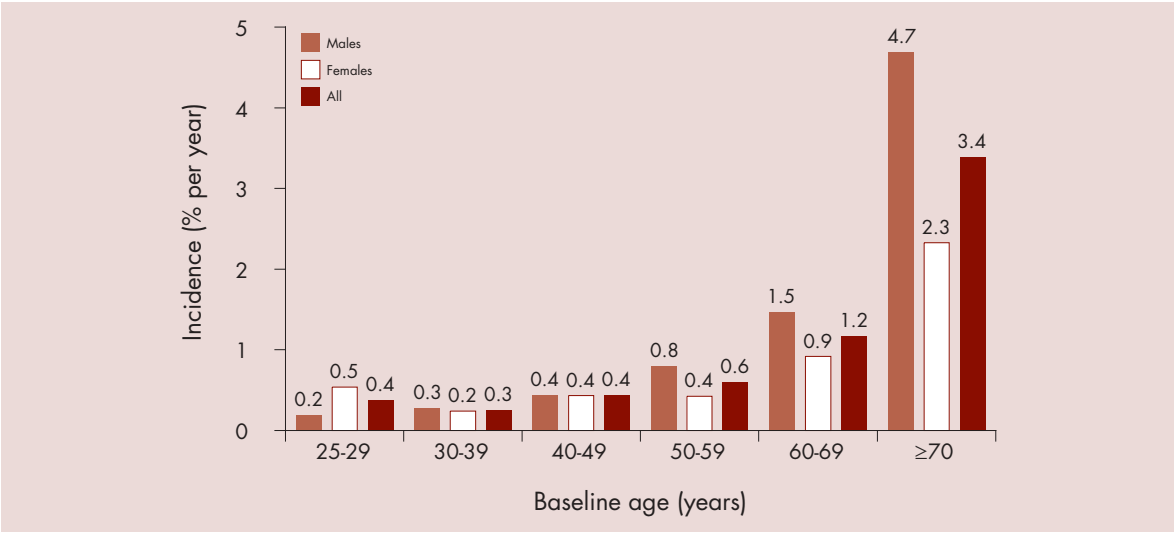
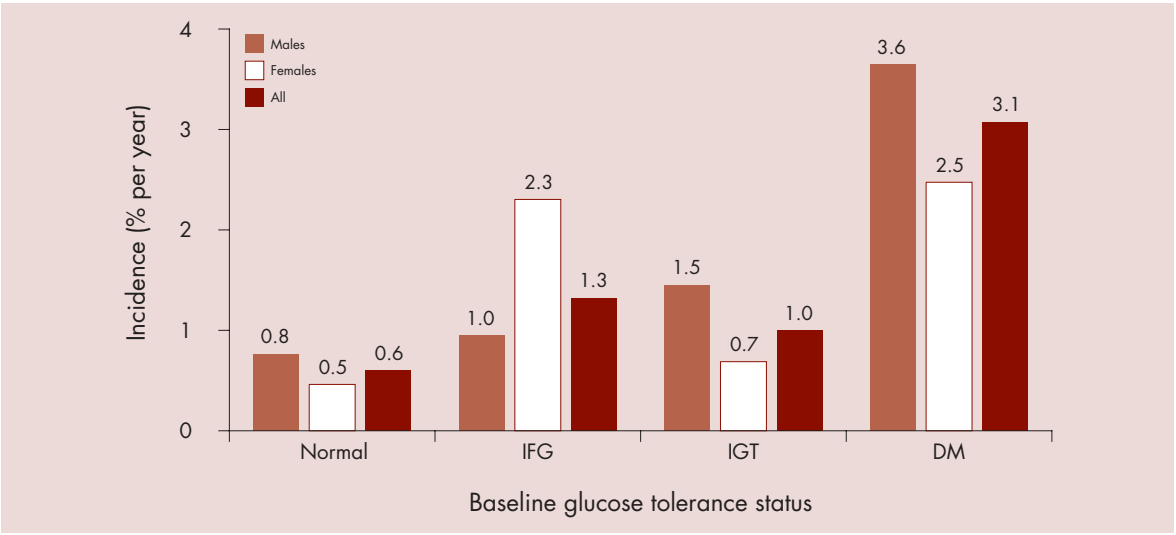


Figure 6.7. Incidence of albuminuria according to baseline glucose tolerance status: the AusDiab study.

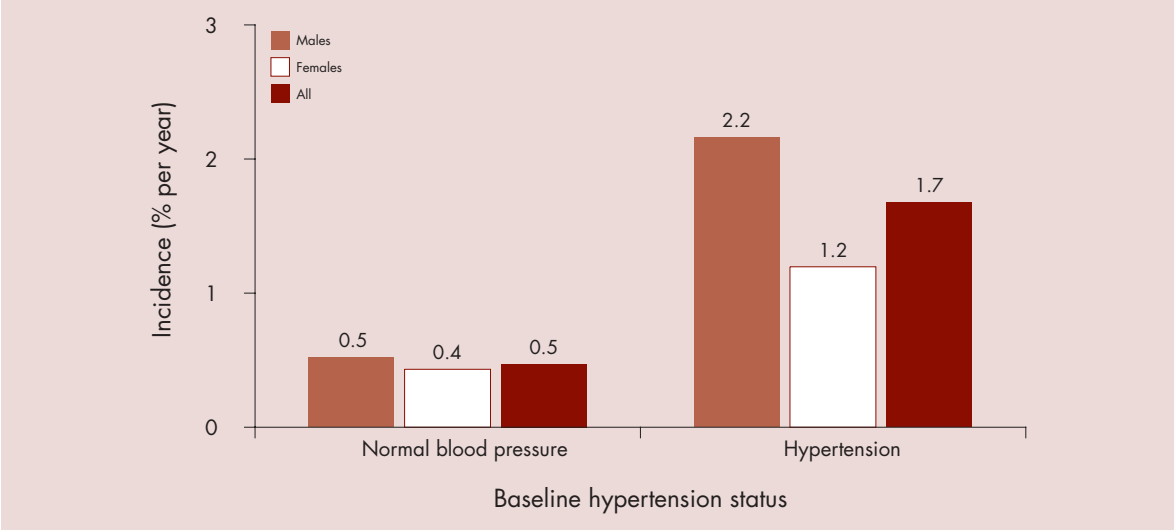


IFG – impaired fasting glucose; IGT – impaired glucose tolerance; DM – diabetes mellitus.

The annual incidence of albuminuria in people with or without hypertension at baseline is presented in Figure 6.8. More than three times as many people with hypertension developed albuminuria each year compared to those with normal blood pressure. The annual incidence of albuminuria in males with hypertension was double that of females with hypertension.

6: Chronic kidney disease

Figure 6.8. Incidence of albuminuria according to baseline hypertension status: the AusDiab study.



Hypertension (high blood pressure) was defined as having a blood pressure ≥140/90 mmHg and/or taking blood pressure-lowering medication.

Key findings

- Every year, almost **1.0%** of adults developed chronic kidney disease, manifested by a reduction in kidney function (impaired glomerular filtration rate). The risks were higher in females and in older people.
- Every year almost 1.0% of adults developed evidence of kidney damage as manifested by the leakage of albumin into the urine (albuminuria). The risks were higher in males and in older people.
- Having high blood pressure increased the risk of developing impaired glomerular filtration rate and albuminuria three-fold.
- Having diabetes increased the risk of developing albuminuria five-fold and of developing a reduction in kidney function **two-fold**.

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7: Mortality

Diabetes, cardiovascular disease, smoking, and kidney disease have a significant impact on death rates. By investigating the mortality associated with each of these conditions, we develop a much better understanding of the natural history of chronic diseases in Australia. This will in turn assist in the development of preventative strategies that can be implemented to help improve the health outcomes for people with these risk factors. This chapter presents the five-year mortality rates for males and females, for people of different ages and for those with different levels of glucose tolerance. The relative mortality risk associated with diabetes and pre-diabetes (impaired glucose tolerance (IGT) or impaired fasting glucose (IFG)), chronic kidney disease, cardiovascular disease, hypertension and smoking, after adjusting for other risk factors is also presented. Unlike the information in the rest of this report which is drawn from the 6,400 participants who attended the baseline and the follow-up surveys, the mortality data relates to all of the 11,247 participants who attended the baseline survey.

Definitions

All-cause mortality refers to death from any cause. The National Death Index, which is a catalogue of all deaths registered in Australia, was used to determine which of the AusDiab study participants had died, and what the cause of death was.

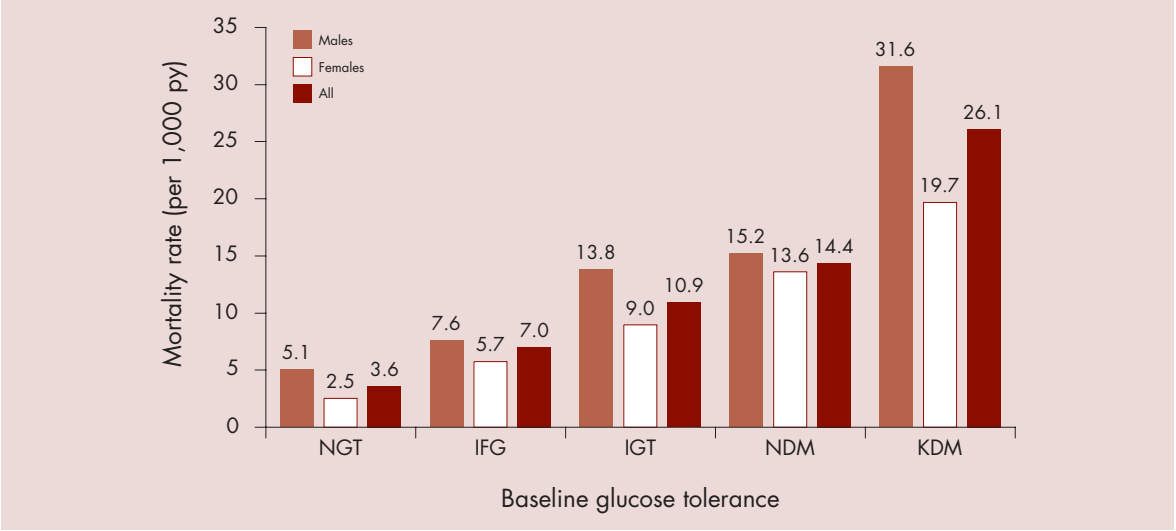
Results

Over a median follow-up of 5.2 years, there were 355 deaths (208 males and 147 females), which represents a mortality rate of 6.1 per 1,000 person-years.

Of those who had diabetes at baseline (either previously known or newly diagnosed), 10% had died within five years of follow-up. By comparison, 5.5% who had IGT, 3.7% who had IFG and 1.9% who had normal glucose tolerance (NGT) at baseline had died after five years.

Figure 7.1 shows the mortality rates (per 1,000 person-years) for males and females in each glucose tolerance category. Males had a higher mortality rate than females for all categories. However, the difference between males and females was particularly marked for people with previously known diabetes (31.6 *vs* 19.7 deaths per 1,000 person-years). Furthermore, the impact of diabetes on mortality was greater for females than males. In females, those with previously known diabetes had a mortality rate that was eight times greater than females with NGT (19.7 *vs* 2.5 deaths per 1,000 person-years). However, in males, there was a six-fold difference between previously diagnosed diabetes and normal glucose tolerance (31.6 *vs* 5.1 deaths per 1,000 person-years).

Figure 7.1. Total mortality according to baseline glucose tolerance status: the AusDiab study.

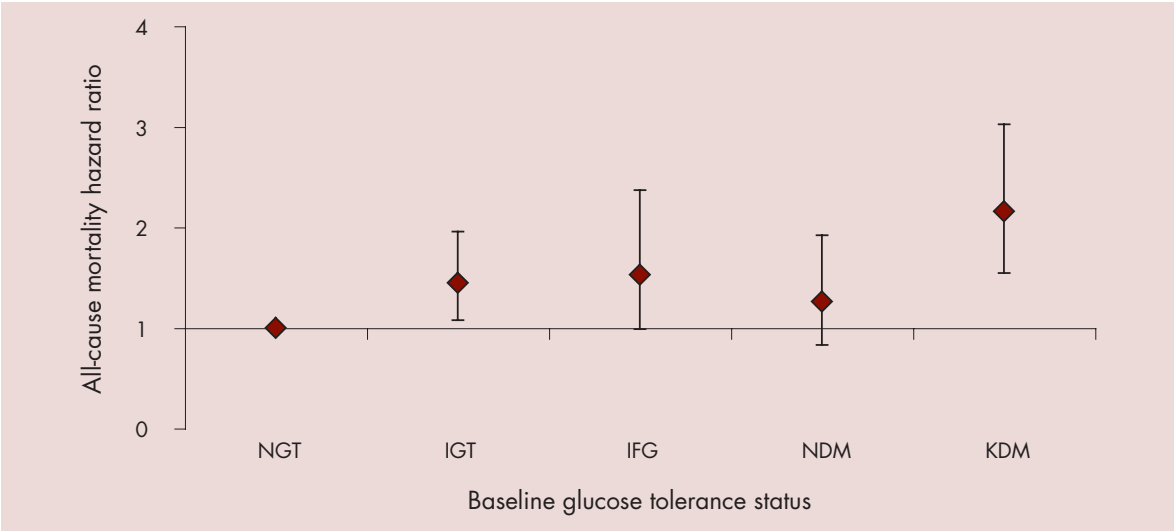


NGT – normal glucose tolerance; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; NDM – newly diagnosed diabetes; KDM – previously diagnosed diabetes.

The heights of bars on all graphs are accurate to two decimal places, but data labels are rounded to one decimal place.

After accounting for the influence of age, sex, blood pressure, smoking, previous cardiovascular disease, cholesterol levels and waist:hip ratio on mortality risk, those individuals who were classified as having previously known diabetes at baseline were twice as likely to die within the five years of follow-up compared with people who had NGT at baseline (Figure 7.2).

Figure 7.2. The relative risk of mortality for people with pre-diabetes and diabetes compared to people with normal glucose tolerance, after accounting for other risk factors: the AusDiab study.



NGT – normal glucose tolerance; IGT – impaired glucose tolerance; IFG – impaired fasting glucose; NDM – newly diagnosed diabetes; KDM – previously diagnosed diabetes.

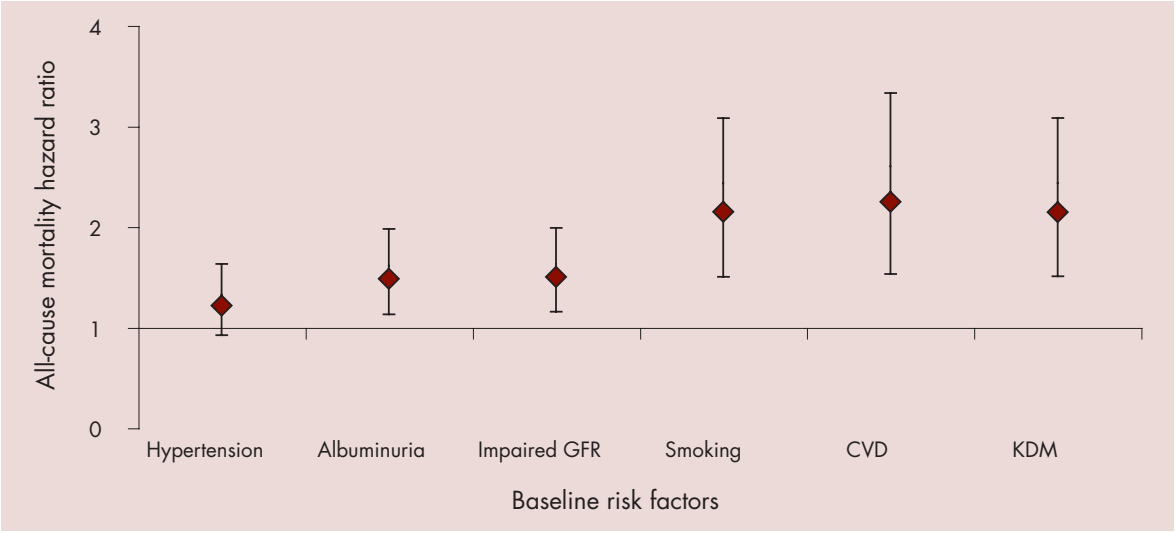
Cox proportional hazards model adjusting for age, sex, previous cardiovascular disease, hypertension, total cholesterol, triglycerides, taking lipid-lowering medication, waist:hip ratio, and smoking. Bars represent 95% confidence intervals.

7: Mortality

IGT and IFG were associated with a 45–55% increase in mortality risk over five years (Figure 7.2).

An increased risk of mortality was also associated with a number of other risk factors, including hypertension, impaired kidney function (glomerular filtration rate <60 ml/min/1.73m^2), smoking and previous cardiovascular disease (Figure 7.3).

Figure 7.3. The relative risk of mortality associated with hypertension, impaired kidney function, smoking, previous cardiovascular disease and diabetes, after accounting for other risk factors: the AusDiab Study.



Hypertension is defined as having a blood pressure ≥140/90 mmHg or taking blood pressure medication.

Albuminuria is defined as albumin:creatinine ratio ≥2.5 mg/mmol for males and ≥3.5 mg/mmol for females.

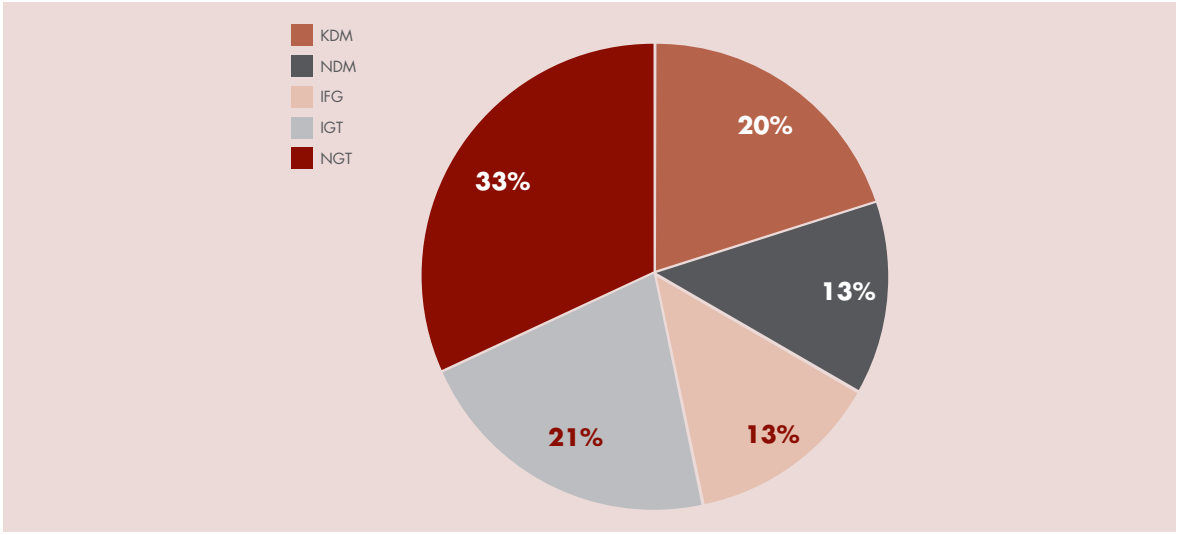
GFR (glomerular filtration rate) provides a measure of kidney function. People with a GFR <60 ml/min/1.73m^2 have impaired kidney function.

CVD – cardiovascular disease; KDM – previously known diabetes.

All risk factors were included in the one Cox proportional hazards model. The model was adjusted for age, sex, total cholesterol, triglycerides, taking cholesterol-lowering medication and waist:hip ratio. Bars represent 95% confidence intervals. The hazard rate for each risk factor was compared to people without that risk factor.

Cause of death was available for 232 of the 355 deaths. Of the 75 deaths that were due to cardiovascular disease, 68% (n=51) occurred in people with diabetes or pre-diabetes at baseline (Figure 7.4). Death due to cardiovascular disease was responsible for 40% of deaths among those with diabetes, 50% among those with IFG, 33% among those with IGT and 25% among those with NGT.

Figure 7.4. Cardiovascular disease mortality (%) according to baseline glucose tolerance status: the AusDiab study.



NGT – normal glucose tolerance; IGT – impaired glucose tolerance; IFG – impaired fasting glucose; NDM – newly diagnosed diabetes; KDM – previously diagnosed diabetes.

Key findings

- Over five years, people with previously known diabetes were twice as likely to die as were those with normal glucose tolerance.
- Pre-diabetes was associated with a 45–55% increase in mortality risk over five years.
- People with previously known diabetes had a similar risk of mortality to smokers and to people with previous cardiovascular disease.
- Over two-thirds of all cardiovascular disease deaths occurred in people with diabetes or pre-diabetes.

8: Survey methods

The 5-year follow-up AusDiab study involved inviting all eligible participants from the baseline study to another survey and physical examination during 2004–05. The survey closely replicated the baseline AusDiab survey that was conducted in 1999–2000, and involved a team travelling around Australia to test participants for diabetes, heart disease and kidney disease.

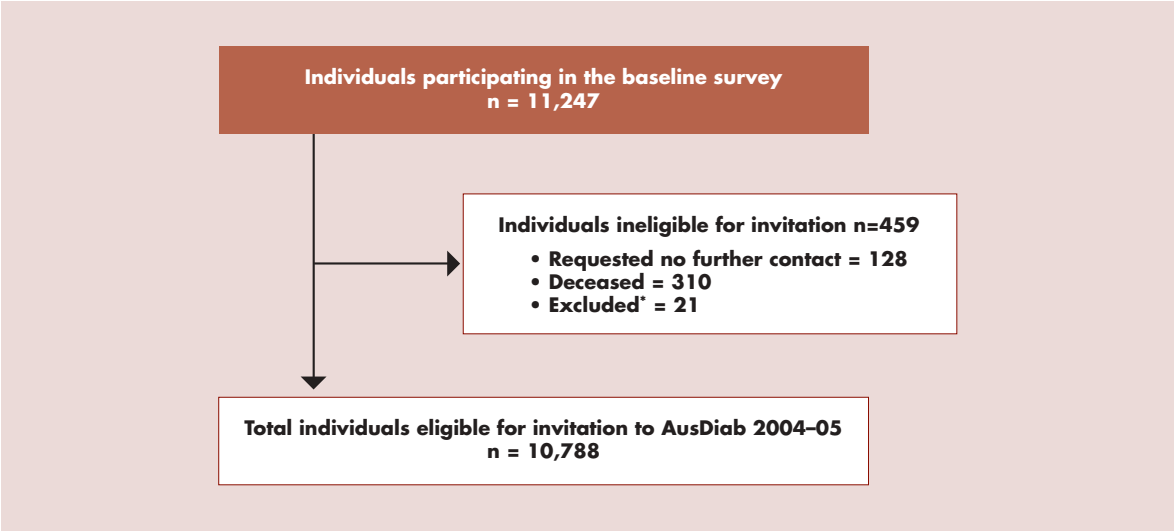
Sample selection and eligibility

The baseline AusDiab (1999–2000) study was a population-based national survey of the Australian general population aged 25 years or older residing in 42 randomly selected urban and rural areas (census collector districts) in six states and the Northern Territory (Appendix A). Full details of the sampling frame, methods and response rates for the baseline AusDiab study have been published previously.[1]

Over the five years since the completion of the baseline study, some participants had moved from their original testing site. These individuals were assigned a new testing site that was located, where possible, in close proximity to their new home. Furthermore, in order to accommodate participants who had moved to the Australian Capital Territory, an additional survey site in Canberra was added to the 42 sites used in the 1999–2000 survey.

Individuals considered ineligible for invitation to the follow-up study included: (i) participants who refused further contact, (ii) participants who were known to be deceased, (iii) participants who had moved overseas, or (iv) participants who had moved into a nursing facility classified for high-care or were ineligible due to chronic or terminal illness. Figure 8.1 summarises the number of individuals eligible for invitation to the follow-up study.

Figure 8.1. Sampling frame for the AusDiab follow-up in 2004–05.



*'Excluded' included participants who had moved into a nursing facility classified for high-care or were ineligible due to chronic or terminal illness.

Survey protocol and procedures

Participants were tested at each of the 43 sites. Of those who could not attend a survey, 137 attended a local pathology laboratory and 2,261 provided self-report data using standard survey forms. On-site testing commenced on 5 June 2004 and finished on 24 November 2005. Where possible, testing took place in the same order as originally conducted for the baseline study, which ensured that most participants had the same follow-up period between 1999–2000 and 2004–05. Appendix A outlines the dates of testing for each of the 43 sites in 2004–05.

Invitation and recruitment

To ensure maximum participation in the 2004–05 survey, annual contact has been maintained with the AusDiab participants via letters and telephone calls. Thus, contact details of survey participants have been updated by using a range of resources including next-of-kin, the Australian Electoral Roll database, online telephone directories and the Telstra White Pages directory on compact disk.

Invitation to the 5-year follow-up involved:

- letters of invitation, sent six and four weeks prior to testing for each site;
- follow-up telephone calls for those who did not reply to the initial letters;
- follow-up telephone calls for all individuals who declined the invitation in order to obtain a reason for non-attendance and/or to re-schedule another appointment time if possible;
- reminder telephone calls two to three days prior to appointments for all individuals who had confirmed an appointment;
- telephone calls to all participants who did not attend their appointment, with the aim of re-scheduling another appointment.

Preparation of survey methods and training

The preparation of the survey methods, including both the physical examination equipment and questionnaires, was undertaken in accordance with the study aims and objectives, with a specific emphasis on the direct comparability with the methods utilised in the baseline study.

A team of survey staff was recruited to administer the survey in each state. All staff attended a three-day training workshop, which was conducted by the project manager and study coordinators prior to collecting data. Staff were briefed on the survey's background, objectives and methodology to ensure accurate and consistent data collection.

8: Survey methods

Physical examination

The AusDiab physical examination procedures closely follow the study protocol as recommended by the World Health Organization for the study of diabetes and other non-communicable diseases.[2] The physical examination was conducted on both weekdays and weekends over a four to seven day period in each of the sampled areas. Local survey sites included community halls, scout halls, sporting halls, church halls and schools. Survey activities at the testing site commenced at 7am and typically finished at 2pm. On average approximately 30 participants attended daily.

All participants gave written informed consent to participate in the survey upon arrival at the testing site. Personal information was verified on-site and entered into a computer database, and each individual was given a barcode based on their original AusDiab unique identification number. Participants were moved through the physical examination procedures in a circuit-like manner that took approximately 2–2.5 hours to complete. Participants were asked to remain on site until all tests were performed. Central to the physical examination was the standard two-hour oral glucose tolerance test (OGTT), during which time all other procedures were performed.

Blood sampling, oral glucose tolerance test and laboratory procedures

Blood was collected by venepuncture after an overnight fast (nine hours or more). In both 1999–2000 and 2004–05, specimens were collected into separate tubes in the following order: a plain tube for measurement of total cholesterol, high-density lipoprotein cholesterol (HDL-C), and triglycerides, and a fluoride/oxalate tube for plasma glucose. All blood specimens collected in the fluoride/oxalate tubes and the plain tubes were centrifuged on-site to separate out the plasma and serum, which was placed in separate tubes. In 1999–2000 all samples were transported daily to a central laboratory. In 2004–05 samples were transported daily to a central laboratory where possible, however, when daily transportation was not possible, all samples were immediately stored on-site in a freezer at –20°C and then transferred to a –70°C storage facility within one to two weeks following collection. All analyses were conducted at a central laboratory (HITECH Pathology, Clayton, Victoria in 1999–2000 and Gribbles Pathology, Clayton, Victoria in 2004–05). Serum triglycerides, total cholesterol and HDL-C were measured by enzymatic methods. In 1999–2000 an Olympus AU600 analyser (Olympus Optical Co. Ltd, Tokyo, Japan) was used, and in 2004–05 the Roche Modular (Roche Diagnostics, Indianapolis, USA), was used. Low-density lipoprotein cholesterol was derived by calculation using the Friedewald formula.[3] A 75g OGTT was performed on all participants, except those on insulin or oral hypoglycaemic drugs, those who were pregnant or those who failed to fast. In 1999–2000, fasting and two-hour plasma glucose levels were determined by a glucose oxidase method using an Olympus AU600 automated analyser (Olympus Optical Co. Ltd, Tokyo, Japan), and in 2004–05 a spectrophotometric-hexokinase method utilising a Roche Modular (Roche Diagnostics, Indianapolis, USA) was used. Laboratory analysis methods were comparable across both the 1999–2000 and 2004–05 surveys (Appendix B).

Urine collection and laboratory procedures

A morning spot urine sample was taken. In 1999–2000, urine creatinine was measured by the modified kinetic Jaffe reaction using the Olympus AU600 auto-analyser (Olympus Optical Co. Ltd). Urine albumin was measured by rate nephelometry with the Beckman Array (Beckman Coulter, Inc., California, USA).

In 2004–05 urine creatinine was measured using spectrophotometric–jaffe alkaline picrate method on a Roche Modular (Roche Diagnostics). Urine microalbumin was measured using nephelometry on a Beckman Immage (Beckman Coulter, Inc.).

Anthropometry

Height was measured to the nearest 0.5 cm without shoes using a stadiometer. Weight was measured without shoes and excess clothing to the nearest 0.1 kg using a mechanical beam balance in 1999–2000 and digital weighing scales in 2004–05. Body mass index (BMI: kg/m^2) was calculated.

Blood pressure

Blood pressure measurements in 2004–05 were performed in a seated position after resting for five minutes or more using an automated blood pressure that was regularly calibrated (Dinamap® Pro-series Monitor Model DP 101–NIBP, Pulse and recorder, GE Medical Systems Information Technologies, Milwaukee, USA). A cuff of suitable size was applied on the participant's exposed upper arm (the arm not used for blood collection), which was supported on a table at heart level. Three sequential measurements were taken, with a 30-second interval between them. All measurements were documented on the participant's form. The mean of the first two measurements were taken. However, if the difference between the first and second measurement was greater than 10 mmHg, for either systolic or diastolic blood pressure, the third measurement was considered, and the mean of the two closest readings was used.

At baseline, blood pressure was measured similarly with an automatic Dinamap machine in all states except for Victoria where a manual sphygmomanometer was used. The two methods at baseline were tested for comparability, and as a result the manual diastolic measurements were adjusted to account for small discrepancies between the manual and automatic methods.

Questionnaires

In both the 1999–2000 and 2004–05 surveys, a series of interviewer-administered questionnaires were used to ascertain a range of health and social information including, previous diagnosis of diabetes and cardiovascular disease, exercise, and smoking.

8: Survey methods

Feedback to participants

All participants who attended either the survey site or a pathology laboratory received a letter outlining some of their survey results. Participants were given the opportunity to request that their results also be sent to their general practitioner.

Participants were sent letters approximately six to eight weeks following the completion of testing, and they were encouraged to seek advice and follow-up where required from their doctor.

Mortality

Vital status was determined by linking the AusDiab cohort to the National Death Index (NDI) maintained by the Australian Institute of Health and Welfare. A recent study by Magliano *et al.*[4] found that the NDI was very accurate in identifying vital status in the Australian population. Linkage to the NDI occurred in May 2004 and again in May 2005. This provided all-cause mortality data for a median follow-up period of 5.2 years. Various parameters including names, date of birth, sex, date of last contact or date of death and geographic code were used to match AusDiab participants to the NDI. Only high level matches between the NDI database and the AusDiab participants were accepted as confirmed deaths. Verification of vital status was greatly assisted by the maintenance of up-to-date contact details of the participants. Cardiovascular disease mortality was determined by reviewing International Classification of Diseases (ICD-10) codes (I10–I99) for the underlying cause of death obtained from the death certificate. The median follow-up for the cardiovascular disease mortality was 3.7 years.

Statistical analysis

All anthropometric analyses in this report are based upon the 6,400 participants who attended both the 1999–2000 and 2004–05 surveys. Given that a further 137 participants were able to give blood at an external pathology laboratory, analyses based on pathology data include 6,537 individuals. Some analyses are based on smaller sample sizes reflecting variables with missing data. Missing data occurred in a random fashion and was not influenced by the study's protocols.

Annual cumulative incidence rates were calculated from the five-year incidence rates by applying the following formula: $-\ln(1-S)/t$; where S is the proportion of new cases (number of new cases at follow-up / number of cases at risk at baseline) over t years, and t equals the time of follow-up which was five years. Confidence intervals (95% CI) for proportions were calculated according to $p \pm (1.96 \times \sqrt{pq/n})$, where p equals the incidence proportion, q equals $1-p$ and n equals the sample size. In circumstances where the number of new cases was small (i.e less than 5), then the following equation for 95% CI was used: $(2r+1.96^2) \pm [(1.96\sqrt{1.96^2 + 4rq})2(n+1.96^2)]$, where r equals the number of new cases and q equals the proportion who did not develop the condition or disease (i.e $1-p$).

Incidence rates (95%CI) and mean differences (±SD) relating to each chapter are summarised in Appendix C. Differences in the baseline characteristics between non-attendees, on-site attendees, and external pathology laboratory attendees of the 5-year follow-up survey were explored with analysis of variance (ANOVA), Kruskall-Wallis tests or Pearson's chi-square test where appropriate. The mean change between 1999–2000 and 2004–05 for weight (kg), BMI (kg/m^2), waist circumference (cm) and systolic blood pressure (mmHg) was calculated.

Total mortality rates were calculated on person-years of follow-up. The period of follow-up was determined by subtracting the date of testing at baseline from either the date of death or the censoring date of the 1 June 2005.

The analyses included all participants, except for three people who were lost to follow-up (n=11,244). Cox proportional hazard models were used to determine the independent effects of a number of different baseline risk factors. Predictor variables were checked for colinearality and interaction terms were assessed. Only participants who were not pregnant, who fasted for nine hours or more and who had complete data for the variables of interest were included in the models (n=10,428).

Analyses were conducted with SPSS version 14.0 (SPSS, Chicago, Illinois, USA) and Stata Statistical Software version 9 (StataCorp, College Station, Texas, USA).

Abbreviations

Abbreviations used in this report are listed in Appendix D.

References

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4. Magliano D, Liew D, Pater H, Kirby A, Hunt D, Simes J, Sundararajan V, Tonkin A. Accuracy of the Australian National Death Index: comparison with adjudicated fatal outcomes among Australian participants in the Long-term Intervention with Pravastatin in Ischaemic Disease (LIPID) study. Australian and New Zealand Journal of Public Health 2003;27:649–653.

9: Response rates

Of the 10,788 participants eligible for testing in 2004–05, 6,400 (59.3%) attended one of the allocated testing sites. A further 137 (1.3%) people attended an external pathology laboratory for blood and urine tests (analysed at the same central laboratory as all other samples) and another 2,261 (21.0%) completed a telephone questionnaire only, which gathered information on a range of health conditions including diabetes. Therefore, 81.6% (8,798/10,788) of the original AusDiab participants took part in some way in the 5-year follow-up survey (Figure 9.1.)

Figure 9.1. Response to AusDiab survey 2004–05.

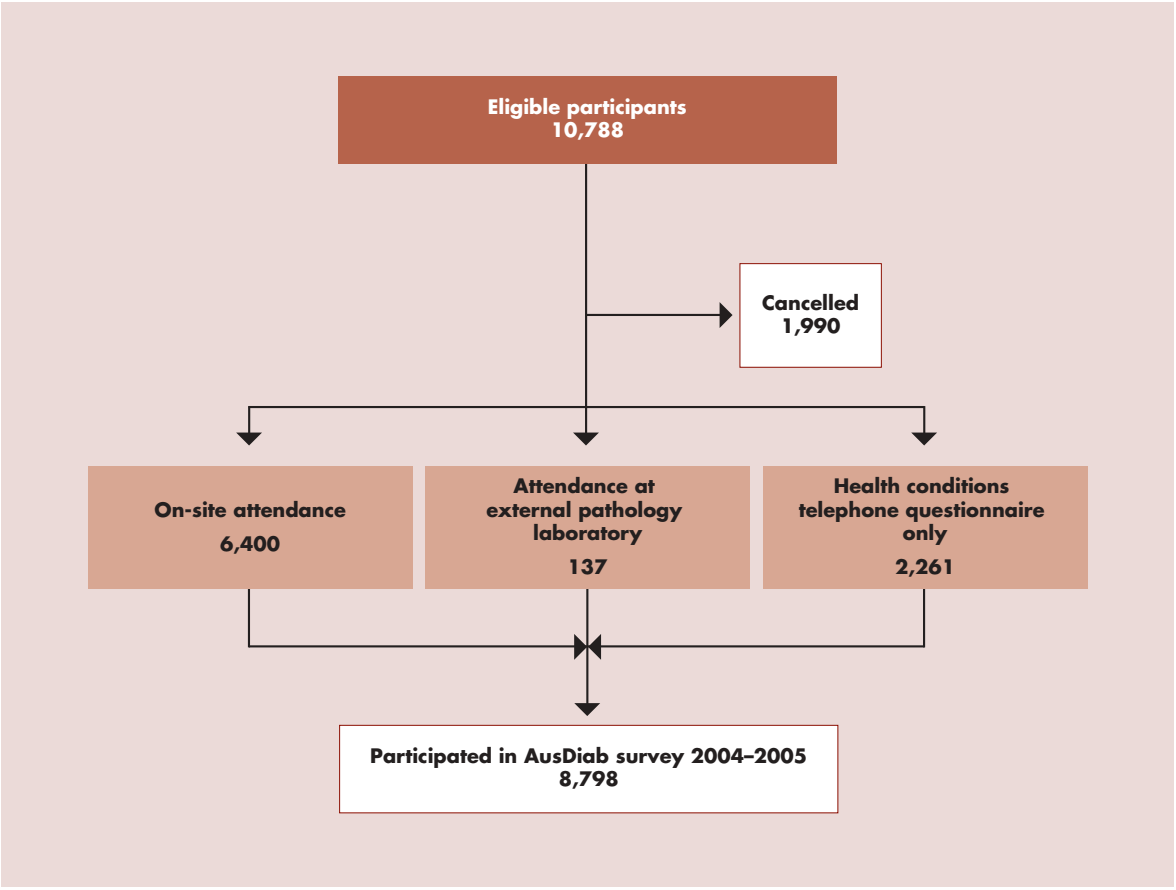

Table 9.1. Response rates for those eligible for testing in 2004–05 according to state or territory: the AusDiab study.

State	Number eligible	Onsite - testing	Pathology laboratory attendance*	Self - reported medical conditions only	Overall responders
Victoria	1429	821 (57.5)	52 (3.6)	337 (23.6)	1210 (84.7)
Western Australia	1526	990 (64.9)	28 (1.8)	210 (13.8)	1228 (80.5)
New South Wales	1458	871 (59.7)	14 (1.0)	323 (22.1)	1209 (82.9)
Tasmania	1700	1102 (64.8)	2 (0.1)	296 (17.4)	1400 (82.4)
South Australia	1700	945 (55.6)	29 (1.7)	467 (27.5)	1441 (84.8)
Northern Territory	1202	702 (58.4)	5 (0.4)	189 (15.7)	895 (74.5)
Queensland	1748	954 (54.6)	7 (0.4)	433 (24.8)	1394 (79.7)
Australian Capital Territory	25	15 (60.0)	0 (0)	6 (24.0)	21 (84.0)
Total	10,788	6,400 (59.3)	137 (1.3)	2,261 (21.0)	8798 (81.6)

Data are n (%). *External pathology laboratory facilities were either not available or were limited in Tasmania, South Australia, Northern Territory and Queensland.

The AusDiab cohort is linked to the National Death Index on an annual basis in order to ascertain the numbers of deceased participants. As of the 1 June 2005, 355 participants were identified as deceased. When the deceased are included, a total of 9,153 (84.8%) participants have been followed up. Three participants were lost to follow-up.

Characteristics of attendees and non-attendees

The baseline physiological and socio-demographic characteristics of participants were compared among those who attended site (n=6,400), those who attended an external Gribbles Pathology centre (n=137), those who only completed a telephone questionnaire on self-reported medical conditions (n=2,261) and those who cancelled (n=1,990) (Table 9.2). People who were not eligible were excluded (n=459). The annual incidence (95% CI) of self-reported diabetes was 0.5 (0.4–0.6) for those who attended site compared with 0.3 (0.2–0.5) for those who only completed a telephone questionnaire. The age and sex profiles of the two groups did not influence these results. (The data presented throughout this report relate only to the 6,537 participants who had blood test results.)

9: Response rates

Table 9.2. Baseline physiological characteristics according to attendance status in 2004-05: the AusDiab study.

	Onsite-testing	Pathology laboratory attendance	Self-reported medical conditions only	Non-attendees	*P*-value
n	6,400	137	2,261	1,990	
Age (years)	51.5 (±12.8)	51.1 (±13.8)	51.7 (±15.7)	48.0 (±15.7)	<0.001
Male (%)	2893 (45.2)	62 (45.3)	944 (41.8)	891 (44.8)	0.041
Systolic blood pressure (mmHg)	128.7 (±17.8)	129.5 (±17.2)	130.4 (±20.0)	127.8 (±19.2)	<0.001
Diastolic blood pressure (mmHg)	70.3 (±11.6)	71.7 (±12.2)	69.9 (±11.9)	69.5 (±11.9)	0.018
BMI (kg/m^2)	26.9 (±4.8)	26.7 (±4.3)	27.1 (±5.2)	27.1 (±5.2)	0.203
Fasting plasma glucose (mmol/l)[†]	5.4 (5.0, 5.8)	5.5 (5.1, 5.9)	5.4 (5.0, 5.8)	5.4 (5.0, 5.7)	0.186
Two-hour plasma glucose (mmol/l)[*†]	5.8 (4.9, 7.0)	5.6 (4.7, 6.9)	6.0 (5.0, 7.2)	5.8 (4.9, 7.2)	<0.001
Total cholesterol (mmol/l)[†]	5.6 (1.0)	5.8 (1.0)	5.7 (1.2)	5.6 (1.1)	0.001
LDL–C (mmol/l)[†]	3.5 (0.9)	3.6 (0.9)	3.6 (1.0)	3.5 (0.9)	0.012
HDL–C (mmol/l)[†]	1.4 (0.4)	1.5 (0.4)	1.4 (0.4)	1.4 (0.4)	0.047
Triglycerides (mmol/l)[†]	1.2 (0.9,1.9)	1.3 (0.9, 1.8)	1.3 (0.9, 2.0)	1.3 (0.9, 1.9)	<0.001
Glucose tolerance status (%)					0.006
• NGT	4761 (75.2)	104 (76.5)	1578 (71.4)	1440 (73.8)	
• IFG	367 (5.8)	8 (5.9)	138 (6.2)	108 (5.5)	
• IGT	750 (11.9)	19 (14.0)	272 (12.3)	241 (12.3)	
• NDM	222 (3.5)	3 (2.2)	112 (5.1)	88 (4.5)	
• KDM	227 (3.6)	2 (1.5)	110 (5.0)	75 (3.8)	

Percentages may not be exact due to missing data.

BMI – body mass index; LDL-C – low-density lipoprotein cholesterol; HDL-C – high-density lipoprotein cholesterol; NGT – normal glucose tolerance; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; NDM – newly diagnosed diabetes at baseline; KDM – previously diagnosed diabetes mellitus at baseline .

Data are mean (±SD), median (25th, 75th percentile) or n (%).

[*]Excluded people who were pregnant at baseline.

[†]Excluded people who fasted for less than nine hours.

Table 9.3. Baseline socio-demographic characteristics according to attendance status in 2004-05: the AusDiab study.

	Onsite-testing	External pathology attendance	Self-reported medical conditions only	Non-attendees	*P*-value
n	6,400	137	2,261	1,990	
Country of birth (%)					<0.001*
• Australia/New Zealand	4904 (76.6)	119 (86.9)	1746 (77.3)	1452 (73.0)	
• United Kingdom/Ireland	721 (11.3)	11 (8.0)	242 (10.7)	221 (11.1)	
• Other countries	773 (12.1)	7 (5.1)	272 (12.0)	315 (15.8)	
Language spoken at home (%)					<0.001*
• English	6190 (96.7)	135 (98.5)	2171 (96.1)	1854 (93.3)	
• Italian	43 (0.7)	0 (0.0)	12 (0.5)	16 (0.8)	
• Greek	24 (0.4)	0 (0.0)	26 (1.2)	17 (0.9)	
• Cantonese	15 (0.2)	0 (0.0)	16 (0.7)	15 (0.8)	
• Mandarin	14 (0.2)	0 (0.0)	4 (0.2)	2 (0.1)	
• Other	112 (1.8)	2 (1.5)	31 (1.4)	84 (4.2)	
Aboriginal/Torres Straight Islander (%)	51 (0.8)	0 (0.0)	23 (1.0)	39 (2.0)	<0.001*
Marital Status					<0.001*
• Married	4858 (75.9)	95 (69.3)	1644 (72.7)	1163 (58.5)	
• De Facto	267 (4.2)	4 (2.9)	96 (4.2)	161 (8.1)	
• Separated	141 (2.2)	5 (3.6)	48 (2.1)	92 (4.6)	
• Divorced	358 (5.6)	8 (5.8)	127 (5.6)	154 (7.7)	
• Widowed	336 (5.3)	12 (8.8)	180 (8.0)	151 (7.6)	
• Never married	437 (6.8)	13 (9.5)	165 (7.3)	267 (13.4)	
Education					<0.001*
• Never attended school or attended primary school only	325 (5.1)	5 (3.6)	167 (7.4)	158 (7.9)	
• Some high school	2263 (35.4)	53 (38.7)	924 (40.9)	782 (39.3)	
• Completed high school	1189 (18.6)	25 (18.2)	464 (20.5)	394 (19.8)	
• University/Technical and further education (TAFE)	2621 (41.0)	54 (39.4)	705 (31.2)	654 (32.9)	
Smokers (%)	2589 (41.2)	51 (37.8)	1063 (48.0)	1025 (52.5)	<0.001*
Exercise					0.002*
• Sufficient (≥150 minutes/day)	3406 (53.5)	70 (51.1)	1141 (50.9)	1023 (51.9)	
• Insufficient (1–149 minutes/day)	1965 (30.9)	50 (36.5)	679 (30.3)	581 (29.5)	
• Sedentary	990 (15.6)	17 (12.4)	421 (18.8)	366 (18.6)	

Percentages may not be exact due to missing data.

*Pearson's Chi-square test

10: Definitions

Diabetes and pre-diabetes

The diagnostic criteria for the presence of diabetes and pre-diabetes are outlined in Table 10.1. Pre-diabetes is a general term that includes both impaired fasting glucose (IFG) and impaired glucose tolerance (IGT). Criteria were based on values for venous plasma glucose concentration (fasting and two-hour measurements) outlined in the World Health Organization report on the Diagnosis and Classification of Diabetes Mellitus.[1] People who reported taking oral hypoglycaemic medication and/or insulin were classified as having diabetes regardless of their plasma glucose levels. In this report, results for type 1 and type 2 diabetes have not been reported separately, as the vast majority of cases were classified as type 2. Women who reported having physician-diagnosed diabetes only during the term of their pregnancy were classified as having had gestational diabetes, and were classified according to the blood glucose results in the survey.

Table 10.1. Classification values for the oral glucose tolerance test.

	Plasma glucose (mmol/l)		
Glucose tolerance	Fasting glucose		2-hour glucose
Diabetes	≥7.0	or	≥11.1
Impaired glucose tolerance (IGT)	<7.0	and	7.8–11.0
Impaired fasting glucose (IFG)	6.1–6.9	and	<7.8
Normal glucose tolerance (NGT)	<6.1	and	<7.8

All participants on oral hypoglycaemic medication or insulin were classified as having diabetes.

Diabetes was further classified into previously "known" diabetes (KDM) and "newly" diagnosed diabetes (NDM). Participants were classified as having known diabetes if they satisfied at least one of the following criteria: (i) receiving oral hypoglycaemic medication and/or insulin, or; (ii) having ever been told by a doctor or nurse that they had diabetes, and had a fasting blood glucose or two-hour plasma glucose levels over the cut-offs for diabetes (Table 10.1). People with newly diagnosed diabetes consisted of those not reporting a diagnosis of diabetes, and who had fasting or two-hour plasma glucose measurements within the diabetes range (Table 10.1).

Obesity

Overweight and obesity were defined using the World Health Organization classification[2] for Europids, based on the body mass index (BMI: weight/height^2), and waist circumference. Table 10.2 outlines classification for the BMI and waist circumference.

Table 10.2. Body mass index and waist circumference classifications.

	Normal	Overweight	Obese
Body mass index (kg/m^2)	<25.0	25.0–29.9	≥30.0
Waist circumference (cm)			
• Males	<94.0	94.0–101.9	≥102.0
• Females	<80.0	80.0–87.9	≥88.0

Hypertension

Hypertension (high blood pressure) was defined as having blood pressure ≥140/90 mmHg and/or taking blood pressure-lowering medication according to the World Health Organization guidelines (Table 10.3).[3]

Table 10.3. Classification of blood pressure.

	Systolic blood pressure (mmHg)		Diastolic blood pressure (mmHg)		Blood pressure-lowering medication
Normal	<140	and	<90	and	No
Hypertension	≥140	or	≥90	or	Yes

10: Definitions

Dyslipidaemia

Dyslipidaemia was defined as having triglycerides ≥2.0 mmol/l or high-density lipoprotein cholesterol (HDL-C) <1.0 mmol/l use. This is based upon the recommendations by the National Heart Foundation[4] and the Australian Diabetes Society[5] (Table 10.4).

Table 10.4. Classification of dyslipidaemia.

	Triglycerides (mmol/l)		HDL-C (mmol/l)
Normal	<2.0	and	≥1.0
Dyslipidaemia	≥2.0	or	<1.0

HDL-C – high-density lipoprotein cholesterol.

Metabolic syndrome

The metabolic syndrome was defined according to the definition by the International Diabetes Federation.[6] Classification of the metabolic syndrome is outlined in Table 10.5.

Table 10.5. Classification of the metabolic syndrome.

Component	Threshold
Waist circumference	Europids: ≥94 cm males, ≥80 cm females South and South-East Asians: ≥90 cm males, ≥80 cm females
Plus any two or more of the following:	
• Raised triglycerides	≥1.7 mmol/l or specific treatment of this lipid abnormality
• Reduced HDL-cholesterol	<1.03 mmol/l in males; <1.29 mmol/l in females or specific treatment for this lipid abnormality
• Raised blood pressure	Systolic ≥130 mmHg or diastolic ≥85 mmHg or treatment of previously diagnosed hypertension
• Raised plasma glucose	Fasting plasma glucose ≥5.6 mmol/l or previously diagnosed type 2 diabetes

Physical activity

'Physical activity time' for the previous week was calculated as the sum of the time spent performing moderate physical activity (e.g. walking) plus double the time spent in vigorous activity.[7] Classification of physical activity is outlined in Table 10.6.

Table 10.6. Classification of physical activity.

	Physical activity
Sufficient	At least 150 minutes of 'physical activity time' in the previous week
Insufficient	1-149 minutes 'physical activity time' in the previous week
Sedentary	No participation in physical activity in the previous week

Kidney disease

Impaired glomerular filtration rate

Chronic kidney disease is defined as present when there is impaired kidney function. The standard measure of kidney function is the glomerular filtration rate (GFR). GFR can be estimated from the results of a blood test (so called 'estimated' GFR or eGFR) and an impaired eGFR is defined as an eGFR of <60 ml/min/1.73m^2.[8] In the AusDiab study, the eGFR has been calculated using the abbreviated MDRD formula.[9]

Albuminuria

Early kidney disease can manifest as the leakage of protein into the urine without any impairment of kidney function. The earliest manifestation of an excessive leakage of protein into the urine can be detected by measuring the urinary albumin levels and is called albuminuria. We considered albuminuria to be present if the spot urine albumin:creatinine ratio was ≥2.5 mg/mmol for males and ≥3.5 mg/mmol for females. Albuminuria is a recognised early risk factor for the development of chronic kidney disease and is also an important risk factor for cardiovascular disease and mortality.[10-12]

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Appendix A

Testing sites and dates

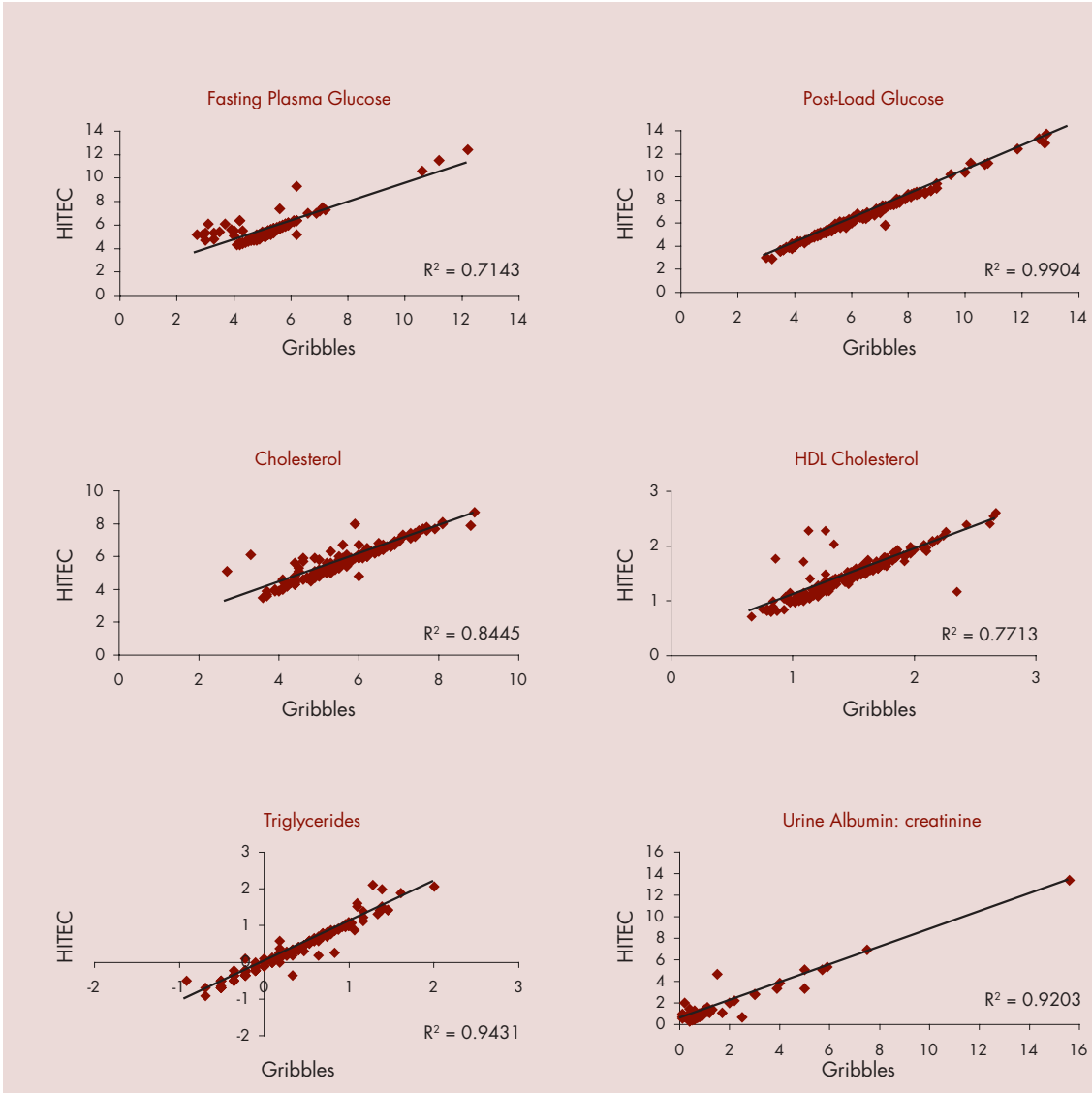
	AusDiab site	Place of testing	Period of testing
Victoria	Parkdale	The Shirley Burke Theatre, 64 Parkers Road, Parkdale	5/06/2004 – 9/06/2004
	Blackburn	Box Hill City Band Hall, 411 Middleborough Road, Box Hill	17/06/2004 – 21/06/2004
	Burwood	Girl Guide Hall, Eley Park, Eley Road, Blackburn South	24/06/2004 – 28/06/2004
	Wattle Glen	Wattle Glen Sporting Club, Wilson Road, Wattle Glen	2/07/2004 – 6/07/2004
	Bendigo	Mac Gillivray Hall, Bendigo TAFE, 136 McCrae Street, Bendigo	10/07/2004 – 18/07/2004
	Mildura	Midura Brass Band Hall, Eighth Street, Mildura	21/07/2004 – 26/07/2004
Western Australia	Scarborough and Trigg	Trigg Island Surf Life Saving Club, West Coast Highway, Trigg Island	21/08/2004 – 30/08/2004
	Kardinya	Willagee Community Centre, Cnr Winnacott & Archibald Street, Willagee	4/09/2004 – 13/09/2004
	High Wycombe	Community & Recreation Hall, Cyril Road, High Wycombe	17/09/2004 – 22/09/2004
	Mount Helena	Mt Helena Scout & Guide Hall, Chidow Street, Mt Helena	25/09/2004 – 30/09/2004
	Oakford	Wandi Community Centre, 302 De-Haer Road, Wandi	6/10/2004 – 10/10/2004
New South Wales	Grays Point	Grays Point Community Centre, 118 Grays Point Road, Grays Point	26/10/2004 – 31/10/2004
	Hurstville	Illawarra Catholic Club, 13–17 Woodville Street, Hurstville	5/11/2004 – 9/11/2004
	Orange	Orange City Bowling Club, 61 Warrendine Street, Orange	13/11/2004 – 21/11/2004
	Berkeley Vale	Berkeley Vale Sports Club, 3–7 Berkeley Road, Glenning Valley	24/11/2004 – 30/11/2004
	West Pennant Hills	Castle Hill Guide Hall, 52 Bounty Avenue, Castle Hill	3/12/2004 – 9/12/2004
	Auburn	Auburn / Lidcombe RSL Youth Centre, Church Street, Lidcombe	12/12/2004 – 15/12/2004
ACT	Canberra	City Health Centre, Diabetes Service, Moore and Alinga Streets, Canberra	2/11/2004 – 3/11/2004
Tasmania	Alanvale	TAFE Tasmania – Alanvale Campus, Alanvale Road, Newnham	23/01/2005 – 30/01/2005
	Ulverstone	Ulverstone Civic Centre, 16 Patrick Street, Ulverstone	3/02/2005 – 9/02/2005
	Ravenswood	Ravenswood Memorial Hall, Vermont Road, Ravenswood	12/02/2005 – 19/02/2005
	George Town	George Town Council Office Building, 16–18 Anne Street, George Town	23/02/2005 – 28/02/2005
	Taroona	Taroona Community Hall, Batchelor Way, Taroona	4/03/2005 – 12/03/2005
	Blackmans Bay	Kingborough Salvation Army Hall, Lot 3, Opal Drive, Blackmans Bay	17/03/2005 – 22/03/2005
South Australia	Unley	Western Youth Centre, 79 Marion Road, Cowandilla	14/04/2005 – 19/04/2005
	Netley	Western Youth Centre, 79 Marion Road, Cowandilla	22/04/2005 – 1/05/2005
	Millicent	Millicent Civic Centre, George Street, Millicent	5/05/2005 – 11/05/2005
	Glenelg	Glenelg Golflands Hall, Aroona Place, Novar Gardens	18/05/2005 – 24/05/2005
	Port Lincoln	RSL Memorial Hall, 14 Hallett Place, Port Lincoln	3/06/2005 – 9/06/2005
	Parafield Gardens	The Dutch Community Social & Welfare Centre, Lot 102 Salisbury Hwy, Greenfields	27/05/2005 – 31/05/2005
Northern Territory	Driver	YMCA of Palmerston, 1st Floor, Satepak Building, 11 Palmerston Crescent, Palmerston	10/09/2005 – 14/09/2005
	Marrara	Italian Club Darwin, Culture Resources Centre, 131 Abala Road, Marrara	5/08/2005 – 14/08/2005
	Wagaman	Italian Club Darwin, Culture Resources Centre, 131 Abala Road, Marrara	17/08/2005 – 22/08/2005
	Nightcliff	Italian Club Darwin, Culture Resources Centre, 131 Abala Road, Marrara	25/08/2005 – 30/08/2005
	Parap	Italian Club Darwin, Culture Resources Centre, 131 Abala Road, Marrara	3/09/2005 – 4/09/2005
	Larrakeya	Greek Orthodox Hall, 92 Cavenagh Street, Darwin	6/09/2005 – 8/09/2005
Queensland	Cairns	Sisters of Mercy Conference & Retreat Centre, 35 Bavhinia Avenue, Earlville	6/10/2005 – 10/10/2005
	Stafford Heights	Chermside Bowls Club, 468 Rode Road, Chermside	21/10/2005 – 29/10/2005
	Chapel Hill	Kenmore Scout Hall, 301 Bielby Road, Chapel Hill	14/10/2005 – 18/10/2005
	Nambour	Nambour Bowls Club, Cnr Coronation Avenue and School Street, Nambour	11/11/2005 – 15/11/2005
	Toowoomba	Queensland Railway Institute Hall, 32 Bellevue Street, Toowoomba	3/11/2005 – 7/11/2005
	Currumbin	Currumbin/Tugun Junior Rugby League Clubrooms, Merv Craig Sporting Complex, Galleon Way, Elanora	19/11/2005 – 24/11/2005

Appendix B

Comparison of laboratory methods

The laboratory methods used in the 1999–2000 (HITECH Pathology, Melbourne, Victoria, Australia) and 2004–05 (Gribbles Pathology, Melbourne, Victoria, Australia) surveys were compared. A random selection of frozen blood samples that were collected during the baseline survey were re-analysed in 2004 by Gribbles Pathology. The following figures outline the results obtained from the original analysis (HITECH Pathology) in 1999–2000 plotted against the results obtained from Gribbles. The triglyceride values are plotted on a logarithmic scale. The data suggest that the laboratory methods used in both surveys were comparable.

Comparison of laboratory methods used in 1999–2000 and 2004–05 AusDiab surveys.



Appendix C

Summary tables

Summary table of incidence for diabetes, obesity, blood pressure, the metabolic syndrome, chronic kidney disease and all-cause mortality

Annual incidence for obesity and hypertension

	Incidence (% per year) (95% confidence intervals)		
	Males	**Females**	**All**
Diabetes and pre-diabetes			
Overall incidence			
Diabetes	0.9 (0.7 – 1.1)	0.7 (0.6 – 0.8)	0.8 (0.7 – 0.9)
IGT	1.4 (1.2 – 1.6)	1.3 (1.1 – 1.5)	1.3 (1.2 – 1.5)
IFG	0.9 (0.7 – 1.1)	0.5 (0.4 – 0.6)	0.6 (0.5 – 0.7)
Incidence of diabetes according to baseline risk factors			
Age (years)			
25-34	0.2 (0.0 – 0.6)	0.5 (0.3 – 1.1)	0.4 (0.2 – 0.7)
35-44	0.6 (0.3 – 0.9)	0.4 (0.3 – 0.7)	0.5 (0.3 – 0.7)
45-54	0.6 (0.4 – 0.9)	0.7 (0.5 – 1.0)	0.7 (0.5 – 0.9)
55-64	1.5 (1.1 – 2.1)	0.6 (0.4 – 1.0)	1.0 (0.8 – 1.3)
65-74	1.5 (1.0 – 2.2)	1.3 (0.8 – 1.9)	1.4 (1.0 – 1.8)
75+	1.2 (0.6 – 2.5)	1.3 (0.6 – 2.6)	1.3 (0.8 – 2.1)
Glucose tolerance status			
NGT	0.2 (0.2 – 0.4)	0.2 (0.2 – 0.3)	0.2 (0.2 – 0.3)
IFG	2.0 (1.4 – 3.0)	4.0 (2.5 – 6.2)	2.5 (1.9 – 3.4)
IGT	4.4 (3.5 – 5.6)	2.9 (2.2 – 3.7)	3.5 (2.9 – 4.2)
BMI* status			
Normal	0.4 (0.2 – 0.6)	0.4 (0.3 – 0.6)	0.4 (0.3 – 0.5)
Overweight	0.8 (0.6 – 1.1)	0.7 (0.5 – 1.0)	0.8 (0.6 – 0.9)
Obese	1.8 (1.4 – 2.5)	1.4 (1.0 – 1.8)	1.6 (1.3 – 1.9)
Waist circumference categories†			
Normal	0.4 (0.3 – 0.6)	0.4 (0.2 – 0.5)	0.4 (0.3 – 0.5)
Overweight	0.8 (0.5 – 1.1)	0.3 (0.0 – 0.6)	0.6 (0.4 – 0.7)
Obese	1.6 (1.3 – 2.1)	1.4 (1.1 – 1.7)	1.5 (1.3 – 1.8)
Physical activity‡			
Sedentary	1.4 (1.0 – 2.1)	0.9 (0.6 – 1.4)	1.1 (0.8 – 1.5)
Insufficient	1.1 (0.8 – 1.6)	0.8 (0.6 – 1.1)	0.9 (0.8 – 1.2)
Sufficient	0.7 (0.5 – 0.9)	0.5 (0.4 – 0.7)	0.6 (0.5 – 0.7)
Hypertension			
Normal	0.5 (0.4 – 0.7)	0.5 (0.3 – 0.6)	0.5 (0.4 – 0.6)
Hypertension§	1.6 (1.3 – 2.0)	1.4 (1.1 – 1.8)	1.5 (1.3 – 1.8)
Dyslipidaemia			
Normal	0.7 (0.5 – 0.9)	0.5 (0.4 – 0.6)	0.5 (0.4 – 0.6)
Dyslipidaemia‖	1.4 (1.1 – 1.8)	1.9 (1.4 – 2.5)	1.6 (1.3 – 1.9)
Metabolic syndrome			
Normal	0.5 (0.4 – 0.7)	0.4 (0.3 – 0.5)	0.4 (0.3 – 0.5)
Metabolic syndrome††	1.7 (1.3 – 2.1)	1.7 (1.4 – 2.2)	1.7 (1.4 – 2.0)

NGT – normal glucose tolerance; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; DM – diabetes mellitus. *BMI - body mass index (weight/height^2) was categorised into three groups: (i) normal: BMI<25 kg/m^2; (ii) overweight: 25–29.9 kg/m^2; and (iii) obese: ≥30 kg/m^2. †Waist circumference: (i) normal: <94.0 cm for males, <80.0 cm for females; (ii) overweight: 94–101.9 cm for males, 80.0–87.9 cm females; (iii) obese: ≥102 cm for males, ≥88.0 cm for females. ‡'Physical activity time' was calculated as the sum of the time spent performing moderate activity (e.g. walking) plus double the time spent in vigorous activity (to reflect its greater intensity). Sedentary – no participation in physical activity in the previous week; insufficient – 1 to 149 minutes of physical activity in the previous week; sufficient – at least 150 minutes of physical activity in the previous week. §Hypertension (high blood pressure) was defined as having a blood pressure ≥140/90 mmHg and/or taking blood pressure-lowering medication. ‖Dyslipidaemia was defined as those with triglycerides ≥2.0 mmol/l or high-density lipoprotein cholesterol (HDL-C) levels <1.0 mmol/l. ††Metabolic syndrome was defined according to the definition by the International Diabetes Federation.

Appendix C

Annual incidence for obesity and hypertension

	Incidence (% per year) (95% confidence intervals)		
	Males	**Females**	**All**
Obesity			
Overall incidence of obesity (BMI ≥30 kg/m^2)	1.9 (1.6 – 2.1)	2.0 (1.8 – 2.3)	1.9 (1.8 – 2.1)
Incidence of obesity according to baseline BMI* status			
Normal	0.0 (0.0 – 0.1)	0.1 (0.0 – 0.2)	0.1 (0.0 – 0.1)
Overweight	3.1 (2.7 – 3.5)	4.9 (4.3 – 5.5)	3.9 (3.5 – 4.2)
Hypertension†			
Overall incidence	3.4 (3.0 – 3.8)	2.7 (2.4 – 3.0)	3.0 (2.8 – 3.2)
Incidence of hypertension according to baseline risk factors			
Age (years)			
25-34	1.7 (1.1 – 2.7)	0.6 (0.3 – 1.1)	1.0 (0.7 – 1.5)
35-44	2.2 (1.7 – 2.8)	1.2 (0.9 – 1.6)	1.6 (1.3 – 2.0)
45-54	3.0 (2.4 – 3.7)	2.8 (2.3 – 3.4)	2.9 (2.5 – 3.3)
55-64	5.2 (4.1 – 6.5)	4.9 (4.0 – 6.0)	5.0 (4.3 – 5.9)
65-74	8.8 (6.6 – 11.5)	8.0 (6.1 – 10.5)	8.4 (6.9 – 10.2)
75+	8.5 (4.3 – 15.4)	6.9 (3.5 – 12.5)	7.6 (4.7 – 11.8)
Glucose tolerance status			
NGT	3.0 (2.6 – 3.4)	2.3 (2.0 – 2.6)	2.6 (2.3 – 2.8)
IFG	4.2 (2.9 – 5.9)	5.0 (2.8 – 8.6)	4.4 (3.2 – 5.9)
IGT	5.2 (3.7 – 7.3)	4.8 (3.6 – 6.3)	4.9 (4.0 – 6.1)
DM	7.5 (5.0 – 10.9)	8.5 (5.3 – 13.0)	7.9 (5.8 – 10.5)
BMI* status			
Normal	2.4 (1.9 – 3.0)	1.5 (1.2 – 1.8)	1.8 (1.5 – 2.1)
Overweight	3.5 (3.0 – 4.2)	3.4 (2.9 – 4.1)	3.5 (3.1 – 3.9)
Obese	5.2 (4.1 – 6.6)	5.6 (4.6 – 6.8)	5.4 (4.7 – 6.3)
Smoking status			
Non-smoker	3.1 (2.6 – 3.6)	2.8 (2.4 – 3.2)	2.9 (2.6 – 3.2)
Smoker‡	3.8 (3.2 – 4.5)	2.4 (2.0 – 3.0)	3.1 (2.7 – 3.5)

NGT – normal glucose tolerance; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; DM – diabetes mellitus. *BMI - body mass index (weight/height^2) was categorised into three groups: (i) normal: BMI<25 kg/m^2; (ii) overweight: 25–29.9 kg/m^2; and (iii) obese: ≥30 kg/m^2. †Hypertension (high blood pressure) was defined as having a blood pressure ≥140/90 mmHg and/or taking blood pressure-lowering medication. ‡Smokers included people who were either current smokers or ex-smokers at baseline.

Annual incidence for the metabolic syndrome

	Incidence (% per year) (95% confidence intervals)					
	Males		Females		All	
Metabolic syndrome*						
Overall incidence	3.8	(3.4 – 4.3)	2.4	(2.1 – 2.7)	3.0	(2.8 – 3.2)
Incidence of metabolic syndrome according to baseline risk factors						
Age (years)						
25-34	3.1	(2.2 – 4.5)	1.6	(1.0 – 2.4)	2.2	(1.7 – 2.9)
35-44	3.3	(2.6 – 4.3)	1.3	(1.0 – 1.7)	2.0	(1.7 – 2.5)
45-54	3.9	(3.2 – 4.8)	2.5	(2.0 – 3.1)	3.1	(2.7 – 3.6)
55-64	3.9	(3.0 – 5.1)	3.7	(2.9 – 4.7)	3.8	(3.2 – 4.5)
65-74	5.1	(3.7 – 6.8)	4.0	(2.9 – 5.4)	4.5	(3.6 – 5.5)
75+	4.4	(2.5 – 7.4)	3.5	(1.9 – 6.2)	3.9	(2.6 – 5.8)
Waist circumference categories†						
Normal	2.4	(2.0 – 2.9)	1.1	(0.8 – 1.4)	1.7	(1.4 – 1.9)
Overweight	5.5	(4.5 – 6.8)	2.8	(2.2 – 3.5)	3.8	(3.3 – 4.4)
Obese	8.2	(6.6 – 10.1)	5.9	(4.9 – 7.0)	6.6	(5.8 – 7.6)
BMI‡ status						
Normal	1.5	(1.1 – 1.9)	1.1	(0.8 – 1.3)	1.2	(1.0 – 1.4)
Overweight	5.4	(4.6 – 6.2)	3.7	(3.1 – 4.5)	4.6	(4.1 – 5.1)
Obese	9.5	(7.2 – 12.1)	6.1	(4.8 – 7.6)	7.2	(6.1 – 8.6)
Physical activity§						
Insufficient	4.3	(2.6 – 6.3)	2.7	(1.4 – 4.1)	3.3	(2.2 – 4.4)
Sufficient	3.5	(2.1 – 5.0)	2.2	(1.0 – 3.5)	2.8	(1.9 – 3.7)
Glucose tolerance status						
NGT	–	– –	–	– –	2.7	(2.0 – 3.5)
IFG	–	– –	–	– –	5.6	(1.6 – 11.4)
IGT	–	– –	–	– –	4.6	(2.3 – 7.5)
DM	–	– –	–	– –	6.2	(0.3 – 16.5)

NGT – normal glucose tolerance; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; DM – diabetes mellitus. *Metabolic syndrome was defined according to the definition by the International Diabetes Federation. †Waist circumference: (i) normal: <94.0 cm for males, <80.0 cm for females; (ii) overweight: 94–101.9 cm for males, 80.0–87.9 cm females; (iii) obese: ≥102 cm for males, ≥88.0 cm for females. ‡BMI - body mass index (weight/height^2) was categorised into three groups: (i) normal: BMI<25 kg/m^2; (ii) overweight: 25–29.9 kg/m^2; and (iii) obese: ≥30 kg/m^2. §'Physical activity time' was calculated as the sum of the time spent performing moderate activity (e.g. walking) plus double the time spent in vigorous activity (to reflect its greater intensity). Sedentary – no participation in physical activity in the previous week; insufficient – 1 to 149 minutes of physical activity in the previous week; sufficient – at least 150 minutes of physical activity in the previous week.

Appendix C

Annual incidence for impaired glomerular filtration rate and albuminuria, and mortality rate (per 1,000 person-years) according to baseline glucose tolerance status

	Incidence (% per year) (95% confidence intervals)					
	Males		Females		All	
Impaired glomerular filtration rate*						
Overall incidence	0.4	(0.3 - 0.5)	1.3	(1.2 - 1.5)	0.9	(0.8 - 1.0)
Incidence of impaired GFR according to baseline risk factors						
Age						
25-29	0.0	(0.0 - 0.7)	0.2	(0.0 - 0.9)	0.1	(0.0 - 0.5)
30-39	0.1	(0.0 - 0.3)	0.3	(0.1 - 0.5)	0.2	(0.1 - 0.3)
40-49	0.1	(0.0 - 0.2)	0.6	(0.4 - 0.8)	0.3	(0.2 - 0.5)
50-59	0.2	(0.1 - 0.4)	1.7	(1.3 - 2.1)	1.0	(0.7 - 1.2)
60-69	0.3	(0.1 - 0.5)	2.1	(1.5 - 2.7)	1.1	(0.8 - 1.5)
≥70	3.2	(2.1 - 4.3)	6.3	(4.7 - 8.1)	4.6	(3.6 - 5.6)
Glucose tolerance status						
Normal	0.2	(0.1 - 0.3)	1.1	(0.9 - 1.3)	0.7	(0.6 - 0.8)
IFG	0.5	(0.1 - 0.9)	1.7	(0.5 - 3.0)	0.8	(0.4 - 1.2)
IGT	1.1	(0.6 - 1.6)	2.4	(1.7 - 3.1)	1.8	(1.3 - 2.2)
DM	0.8	(0.3 - 1.4)	3.0	(1.8 - 4.2)	1.7	(1.1 - 2.3)
Hypertension						
Normal	0.1	(0.1 - 0.2)	0.8	(0.7 - 1.0)	0.5	(0.4 - 0.6)
Hypertension	0.9	(0.6 - 1.2)	2.9	(2.3 - 3.4)	1.8	(1.5 - 2.1)
Albuminuria‡						
Overall incidence	1.0	(0.9 – 1.2)	0.6	(0.5 – 0.8)	0.8	(0.7 – 0.9)
Incidence of albuminuria according to baseline risk factors						
Age (years)						
25-29	0.2	(0.0 – 1.1)	0.5	(0.2 – 1.6)	0.4	(0.0 – 0.7)
30-39	0.3	(0.0 – 0.5)	0.2	(0.0 – 0.4)	0.3	(0.1 – 0.4)
40-49	0.4	(0.2 – 0.7)	0.4	(0.2 – 0.6)	0.4	(0.3 – 0.6)
50-59	0.8	(0.5 – 1.1)	0.4	(0.2 – 0.6)	0.6	(0.4 – 0.8)
60-69	1.5	(1.0 – 2.0)	0.9	(0.5 – 1.3)	1.2	(0.9 – 1.5)
70+	4.7	(3.4 – 6.0)	2.3	(1.5 – 3.2)	3.4	(2.7 – 4.1)
Glucose tolerance status						
NGT	0.8	(0.6 – 0.9)	0.5	(0.3 – 0.6)	0.6	(0.5 – 0.7)
IFG	1.0	(0.4 – 1.5)	2.3	(0.9 – 3.8)	1.3	(0.8 – 1.9)
IGT	1.5	(0.8 – 2.1)	0.7	(0.3 – 1.1)	1.0	(0.7 – 1.4)
DM	3.6	(2.4 – 5.0)	2.5	(1.4 – 3.6)	3.1	(2.2 – 3.9)
Hypertension status						
Normal	0.5	(0.4 – 0.7)	0.4	(0.3 – 0.6)	0.5	(0.4 – 0.6)
Hypertension†	2.2	(1.7 – 2.6)	1.2	(0.9 – 1.5)	1.7	(1.4 – 2.0)
Mortality						
Mortality rate (per 1,000 person-years) according to baseline glucose tolerance status						
NGT	5.1	(4.1 – 6.2)	2.5	(2.0 – 3.3)	3.6	(3.1 – 4.3)
IFG	7.6	(4.8 – 12.0)	5.7	(2.6 – 12.8)	7.0	(4.7 – 10.5)
IGT	13.8	(10.1 – 18.9)	9.0	(6.5 – 12.4)	10.9	(8.7 – 13.7)
NDM	15.2	(9.6 – 24.1)	13.6	(8.3 – 22.2)	14.4	(10.3 – 20.1)
KDM	31.6	(23.2 – 43.0)	19.7	(12.8 – 30.2)	26.1	(20.3 – 33.6)

NGT – normal glucose tolerance; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; DM – diabetes mellitus. * Impaired glomerular filtration rate (GFR) defined as having an estimated GFR of <60 ml/min/1.73m^2. †Hypertension (high blood pressure) was defined as having a blood pressure ≥140/90 mmHg and/or taking blood pressure-lowering medication. ‡Abnormal albumin:creatinine ratio was defined as ≥2.5 mg/mmol for males and ≥3.5 mg/mmol for females.

Summary of mean change (±SD) in weight, BMI and waist circumference over five years according to baseline age and sex: the AusDiab study.

Age (years)	Males	Females	All
Mean weight (kg) difference			
25-34	3.4 (±6.0)	3.5 (±7.7)	3.5 (±7.0)
35-44	2.5 (±5.3)	2.5 (±6.1)	2.5 (±5.8)
45-54	1.6 (±5.0)	1.9 (±5.7)	1.8 (±5.4)
55-64	0.4 (±4.7)	0.7 (±4.6)	0.5 (±4.6)
65-74	– 0.3 (±4.1)	– 0.3 (±5.0)	– 0.3 (±4.6)
75+	– 1.9 (±4.1)	– 2.4 (±4.8)	– 2.2 (±4.5)
Total	1.3 (±5.1)	1.3 (±5.8)	1.4 (±5.5)
Mean BMI (kg/m^2) difference			
25-34	1.2 (±1.9)	1.4 (±2.9)	1.3 (±2.5)
35-44	1.0 (±1.7)	1.1 (±2.3)	1.1 (±2.1)
45-54	0.8 (±1.6)	1.0 (±2.1)	0.9 (±1.9)
55-64	0.5 (±1.5)	0.7 (±1.8)	0.6 (±1.7)
65-74	0.4 (±1.4)	0.4 (±2.0)	0.4 (±1.7)
75+	0.0 (±1.4)	– 0.3 (±1.9)	– 0.2 (±1.7)
Total	0.7 (±1.6)	0.9 (±2.2)	0.8 (±1.9)
Mean waist circumference (cm) difference			
25-34	2.1 (±6.5)	3.5 (±8.1)	2.9 (±7.5)
35-44	1.9 (±6.0)	2.9 (±6.8)	2.5 (±6.5)
45-54	1.9 (±5.8)	3.0 (±6.7)	2.5 (±6.3)
55-64	1.2 (±5.6)	2.0 (±6.8)	1.6 (±6.3)
65-74	1.2 (±5.2)	1.6 (±6.6)	1.4 (±6.0)
75+	0.3 (±5.2)	– 0.1 (±7.5)	0.1 (±6.5)
Total	1.6 (±5.8)	2.5 (±7.0)	2.1 (±6.5)

SD – standard deviation. *BMI – body mass index (weight/height^2). Data are mean difference (±SD).

Summary of mean change (±SD) in weight, and waist circumference over five years according to baseline body mass index status: the AusDiab study.

	Males	Females	All
Mean weight (kg) difference according to baseline BMI			
Normal	1.8 (±4.3)	1.9 (±4.3)	1.9 (±4.3)
Overweight	1.0 (±4.8)	1.3 (±5.9)	1.2 (±5.4)
Obese	1.1 (±6.5)	0.8 (±7.9)	0.9 (±7.3)
Total	1.3 (±5.1)	1.5 (±5.8)	1.4 (±5.5)
Mean waist circumference (cm) difference according to baseline BMI			
Normal	1.6 (±5.4)	2.9 (±6.1)	2.5 (±5.9)
Overweight	1.6 (±5.7)	2.4 (±7.3)	2.0 (±6.5)
Obese	1.6 (±6.5)	1.9 (±7.9)	1.8 (±7.3)
Total	1.6 (±5.8)	2.5 (±7.0)	2.1 (±6.5)

Body mass index (BMI: weight/height^2) was categorised into three groups: (i) normal: BMI<25 kg/m^2; (ii) overweight: 25–29.9 kg/m^2; and (iii) obese: ≥30 kg/m^2. Data are mean difference (±SD).

Appendix D

Abbreviations

AusDiab	Australian Diabetes Obesity and Lifestyle Study
BMI	Body mass index
CVD	Cardiovascular disease
DM	Diabetes mellitus
GFR	Glomerular filtration rate
eGFR	Estimated glomerular filtration rate
HDL-C	High-density lipoprotein cholesterol
IFG	Impaired fasting glucose
IGT	Impaired glucose tolerance
KDM	Previously known diabetes
LDL-C	Low-density lipoprotein cholesterol
NDI	National death index
NDM	Newly diagnosed diabetes
NGT	Normal glucose tolerance
OGTT	Oral glucose tolerance test
SD	Standard deviation

Appendix E

Contributors

The following people contributed to the design and running of the 5-year follow-up AusDiab study.

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Appendix E

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Notes

Notes

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