

Diabetes & Associated Disorders in Australia - 2000

The Accelerating Epidemic

The Australian Diabetes, Obesity and
Lifestyle Study (AusDiab)



Authors

Dr D Dunstan, Professor P Zimmet, Professor T Welborn, Dr R Sicree, Dr T Armstrong, Professor R Atkins, Mr A Cameron, Dr J Shaw, and Dr S Chadban on behalf of the AusDiab Steering Committee

Contributors

Dr S Bennett
Dr M de Courten
Ms G Hodgson
Mr A Meehan
Ms S Murray
Ms N Watson
Dr J Williams

List of sponsors

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(Minister: The Hon Dr Michael Wooldridge, Federal Minister for Health and Aged Care)

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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 eyes, kidneys, nerves, heart and blood vessels.

- Type 1 diabetes results from autoimmune destruction of the pancreatic beta cells, the cells which produce insulin. In this form of diabetes, insulin is required for survival. It accounts for 10% of all persons with diabetes in Australia. It can appear at any age, although usually before 40 years.
- Type 2 diabetes is characterised by resistance to insulin's action and impaired insulin production by the pancreas, 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 (hence the term "diabesity") and lack of exercise. In most instances, the molecular or metabolic causation is not yet known.



Ministerial foreword



Foreword

International Diabetes Federation



Professor Sir K George MM Alberti

Message from the IDF

This work represents a landmark in the history of diabetes in Australia. Amongst the earliest hints of the emerging diabetes pandemic were the studies by Australians of diabetes in the Pacific Islands and subsequent work with the indigenous populations. Both of these pointed to Westernisation of lifestyle as a crucial factor.

Studies from Western Australia at that time suggested that there was less of a problem in those of European origin. However, no Australia-wide data have been available. This has finally been rectified.

The results should focus attention on the incontrovertible truth: that diabetes is now one of the biggest health problems assailing Australia.

The prevalence rates match or exceed those in most Western countries and are similar to those in the USA. This, together with the presumptive much higher rates in the Indigenous population, should be taken by all as a call to action on two fronts: first, prevention of Type 2 diabetes by tackling the horrendous twin problems of obesity and lack of physical activity; and second, improved treatment facilities to hold at bay the deadly complications.

Australia led the way by early identification of the world pandemic. Now the IDF challenges Australia to lead the way by implementing the first successful nationwide preventive strategy.

Professor Sir K George MM Alberti

President, International Diabetes Federation



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Executive summary

In both human and economic terms, diabetes mellitus is already one of Australia's most costly diseases with the number of new cases and its impact accelerating. It is a major risk factor for heart disease, kidney failure, blindness, amputations and birth defects. Diabetes shortens life expectancy by up to 15 years and its annual cost to the nation exceeds \$1.2 billion. Already in the United States, diabetes related costs account for 12% of the national health budget. The proliferation of studies describing the epidemiology and impact of diabetes over the last 20 years in many countries has been extraordinary. Surprisingly however, there has never been a national study performed in Australia.

Against this background, the National Diabetes Strategy (NDS), launched in 1998 and spearheaded by Dr Michael Wooldridge, the Federal Minister for Health and Aged Care, was a visionary and innovative public health initiative. Arising from the NDS, the Australian Diabetes, Obesity and Lifestyle Study (AusDiab) was conducted to determine the prevalence of diabetes, obesity and other cardiovascular disease risk factors including hypertension and abnormal serum lipid profiles (dyslipidaemia).

Until recent years, our governments and public health planners in Australia were largely unaware of the magnitude of diabetes and its costs and, more importantly, the predicted escalation of the number of persons with diabetes and its serious complications. For this reason, diabetes was not prioritised as a major public health challenge before 1996. However, in recognition of the impact of diabetes on the Australian community, Australian Health Ministers agreed in 1996 that diabetes would become one of the National Health Priorities and a commitment was made to address diabetes in its major forms: Type 1, Type 2 and gestational diabetes. In 1999, the Health Ministers endorsed the National Diabetes Strategy 2000-2004 with the signing of the Australian Diabetes Declaration.

Thus AusDiab was commissioned as the first accurate national study of diabetes prevalence. The findings will provide the baseline data for the evaluation of the success of any intervention programs introduced as part of the NDS.

Diabetes mellitus

The prevalence of diabetes in the Australian population aged 25 years and older was 7.5%: 8.0% for males and 7.0% for females. The prevalence of diabetes rose from 2.5% in people 35 to 44 years to 23.6% in those 75 years and over.

- For every known case of diabetes, there was one undiagnosed case.
- There are about 940,000 people over the age of 25 years with diabetes in Australia.
- The number of adults in Australia with diabetes has trebled since 1981.
- The prevalence of impaired glucose metabolism (being either impaired glucose tolerance or impaired fasting glycaemia) in the population was 16.3%: 17.3% for males and 15.3% for females.

Almost 1 in 4 Australians 25 years and over has either diabetes or a condition of impaired glucose metabolism. This condition is associated with substantially increased immediate risk of heart disease as well as increased risk of diabetes in the future.

In comparison to people with normal glucose tolerance, those with diabetes were more likely to have hypertension (69.3% vs. 21.1%), to be obese (44.4% vs. 15.9%), to have elevated triglycerides (42.9% vs. 16.0%), and to have a depressed HDL-cholesterol (23.1% vs. 10.6%).

Overweight and obesity

- The prevalence of being mildly overweight (BMI 25-29.9 kg/m²) was 39.1%: 48.2% for males and 30.2% for females.
- The prevalence of obesity (BMI ≥ 30 kg/m²) was 20.5%: 19.1% for males and 21.8% for females. This is more than double the rate observed in 1980.
- Thus 59.6% of the participants were mildly overweight or obese. The rate for males was 67.4%, and for females, 52.0%.



Dyslipidaemia (abnormal cholesterol and other blood fats)

- The prevalence of elevated total cholesterol (5.5 mmol/l) was 51.2%: 51.1% for males and 51.2% for females.
- The prevalence of elevated triglycerides (2.0 mmol/l) was 20.5%: 24.5% for males and 16.6% for females.
- Lipid lowering agents were being taken by only 7.3% of the population.

Hypertension

- The prevalence of hypertension was 28.8%: 30.6% for males and 27.1% for females.
- Anti-hypertensive medication was being taken by 13.4% of the population: 11.5% of males, and 15.3% of females.
- Of those with hypertension, only 37.6% of males and 56.5% of females were on medication for hypertension. Thus, for every known case of hypertension, there was at least one untreated case.
- There is a trend to a lowering of prevalence of hypertension over the last two decades.

Smoking

- The rate for current smoking was 15.6%: 18.2% of males and 13.1% of females.
- Of those who had ever smoked, 63.3% had ceased smoking: the same for both males and females.

Physical activity

- Only half (49.8%) of the Australian adults aged 25 years and above were undertaking 'sufficient' physical activity to maintain good health.
- Approximately 1 in 6 people (15.6%) reported no participation in physical activity at all.

Cardiovascular disease risk factors - the “Deadly Quartet”

A major reason for the high rates of cardiovascular disease in persons with diabetes is the co-existence

or clustering with diabetes of other risk factors such as abdominal (central) obesity, dyslipidaemia and hypertension i.e. the Metabolic Syndrome or what is sometimes called the “Deadly Quartet”.

Over 50% of Australian adults have at least one component of the “Deadly Quartet” and the consequent risk of cardiovascular disease. This percentage does not take into account other cardiovascular disease risk factors such as cigarette smoking, sedentary behaviour and family history of heart disease.

Renal disease

- Renal disease is a major health burden, and currently consumes 5.7% of the Australian health care budget.
- Proteinuria was detected in 2.5% of the survey population, haematuria in 6.4% and 1.1% had an elevated serum creatinine.
- Together with diabetes and hypertension, this indicates over one third of Australian adults are at increased risk of renal disease.

A later report will examine the problem of renal disease in greater detail including information on renal disease in people with and without diabetes.

Conclusions

The prevalence of diabetes and its co-morbidities in Australia is very high by world standards for a Western nation. The number of people with diabetes has trebled since 1981.

Apart from the escalating rate of diabetes, there is a high prevalence of impaired glucose metabolism, a condition associated with increased risk of heart disease and future diabetes. The high rates of diabetes and impaired glucose metabolism, coupled with those of obesity, dyslipidaemia and hypertension, constitute a significant threat in terms of the future socio-economic burden of cardiovascular disease and diabetes complications for Australia.

Diabetes and its complications are associated with very high social and economic costs for both the person with diabetes, and State and Federal governments. The high rates of diabetes and cardiovascular disease risk factors represent a



significant public health burden that requires urgent measures both for prevention of diabetes and its associated complications.

The magnitude of the diabetes epidemic in Australia, coupled with the significant premature morbidity and mortality due to the enormous burden associated with diabetic complications, including heart and kidney disease, heralds the need for increased attention and resources. The fact that potent environmental risk factors for Type 2 diabetes such as obesity and exercise are modifiable, points to lifestyle intervention. This involves the incorporation of a healthy diet with an increase in physical activity, as a means of curbing the impact of this epidemic.

Finally, we propose:

- A 5 year follow-up study be undertaken to monitor outcomes in the people who participated in the AusDiab survey to examine the natural history of diabetes, its complications, impaired glucose metabolism as well as cardiovascular disease and stroke.
- A comparable study is urgently needed to assess the magnitude of the challenge at a national level in Australia's Indigenous community given the epidemic rates and huge health burden of diabetes. Such a study will provide the first accurate assessment of the burden of diabetes, diabetes complications and associated chronic diseases in the Indigenous population.



1 Background

Diabetes mellitus is now recognised as a serious global health problem often resulting in substantial morbidity and mortality, primarily from cardiovascular complications, eye and kidney diseases and limb amputations. It will undoubtedly be one of the major health problems facing Australia in the 21st century¹.

World-wide, diabetes is becoming epidemic. Recently, in collaboration with the World Health Organization (WHO) in Geneva, the International Diabetes Institute produced new global predictions of the number of people with diabetes for various countries for the year 2025 (unpublished, April, 2001). It was estimated that in the year 2000 there were approximately 160 million people with diabetes in the world. This will climb to over 280 million people by the year 2025, the majority of them with Type 2 diabetes. It was estimated that for Australia there will be 1.23 million persons with diabetes in the year 2010. From Figure 1.1, the predicted number at the time of this survey is nearly 950,000. This figure illustrates the dramatic rise in diabetes cases over the last two decades and shows the estimated number of persons with diabetes in Australia

The number of studies describing the epidemiology of diabetes over the last twenty years in many countries has been extraordinary, yet there has not been a national study in Australia. For this reason, governments and public health planners in this country had not prioritised diabetes. They remained largely unaware of the current magnitude, or, more

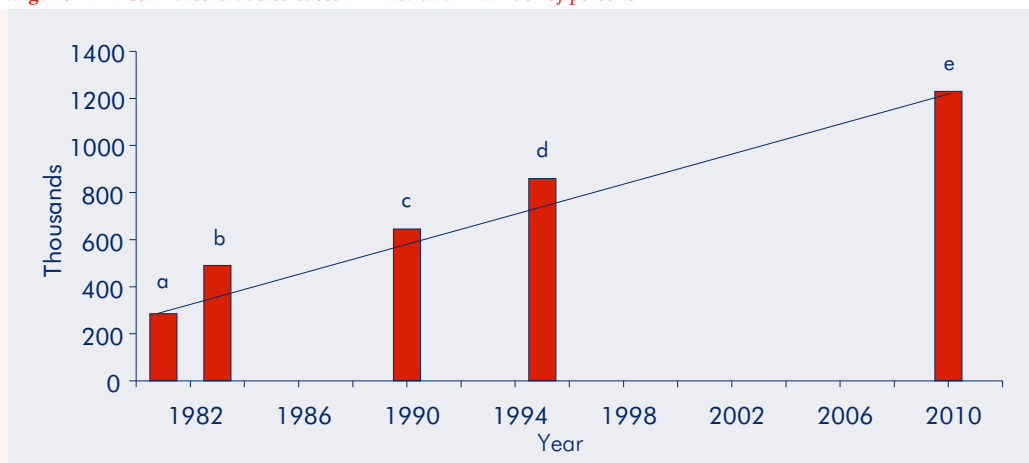
importantly, the future potential for increases in diabetes and its serious complications in Australia.

Australia is a nation that by world standards provides a high proportion of its population with the opportunities for good health. Life expectancy is high, but, increasingly an ageing population and some susceptible groups in the community are suffering from lifestyle diseases. 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, and the unfavourable effects on lifestyle that have come with modernization and industrialization of our society. For example, exercise is being engineered out of our lives with the mechanisation and computerization of our society.

The major causes of death in Australia are currently coronary heart disease, cancer and stroke². Much of this mortality is the result of life-style changes that have led to lower levels of physical activity and unfavourable changes in our diet with consequent increase in obesity³ and detrimental changes to lipid profiles. Indications that diabetes rates were also on the rise led to the AusDiab initiative.

The above factors and the ageing of the Australian population have led to high levels of morbidity from a number of chronic diseases which contribute greatly to the national health costs. Diabetes mellitus and cardiovascular disease (CVD) are two of these conditions^{1,3}. As a result they have been included by the Federal, State and Territory

Figure 1.1: Estimated diabetes cases in Australia: number of persons



For the questionnaire based studies (1983, 1989-90, 1995), the total number of people with diabetes is calculated on the basis of there being one undiagnosed case for every diagnosed case¹⁻³. a. Busselton, 1981⁴; b. NHF, 1983^{5,6}; c. ABS, 1989-90⁷; d. ABS, 1995⁸; e. (Estimate).



governments amongst the six National Health Priority Areas, which also include cancer, injury prevention, asthma and mental health.

As an initiative of the Federal Health Minister, a National Diabetes Strategy and Implementation Plan¹⁰ was produced in 1998. It had as its aims to:

1. Prevent or delay the development of Type 1 and Type 2 diabetes,
2. Improve quality of life and reduce complications and premature mortality in people with diabetes,
3. Achieve maternal and child outcomes for gestational diabetes and for women with pre-existing diabetes equivalent to those of non-diabetic pregnancies,
4. Achieve progress towards a cure for Type 1 diabetes,
5. Advance knowledge and understanding about the prevention, cure and care of diabetes, through a comprehensive research effort.
6. 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.

To achieve these ambitious objectives, reliable nationally representative information on diabetes and related conditions is a prerequisite. Such data will allow the planning of programs to prevent the onset of diabetes and its complications; they will also provide an essential baseline to assess the impact of such programs in the future.

Accurate prevalence data for diabetes in Australia were unavailable or inadequate prior to the AusDiab study, although it was recognised that diabetes is:

- the second most common cause 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, and

- one of the most common chronic diseases in children¹¹.

For Australia (as for many other countries), there has up till now been a paucity of reliable prevalence and incidence data for Type 2 diabetes, its complications and associated conditions.

Diabetes in Australia - pre 1999

What information has been available to date? The most recent estimates of the national prevalence come from the 1995 ABS National Health Survey¹², on which basis 430,700 Australians were determined as being aware of having diabetes. This was an increase on the figure of 309,000 previously established by the 1989-90 survey⁷. It is unlikely that this increase during such a relatively short interval can be solely attributed to the ageing of the population or improved diagnosis.

The most recent figures, although obtained from an essentially representative sample of the urban population, lack the reliability that would be obtained from the taking of blood samples to diagnose diabetes. Type 2 diabetes can be asymptomatic for many years, and self reported diabetes represents only about one half of actual cases. The true prevalence of diabetes can only be established by blood testing. In addition, blood sampling provides important health-related information that is otherwise not available through self-reported data, and it is essential for determining impaired glucose tolerance (IGT), undiagnosed Type 2 diabetes and abnormal lipid profiles. Furthermore, the WHO will only accept survey data on the diagnosis of diabetes that includes laboratory blood glucose measurements¹³.

A number of studies based on blood glucose levels have previously been performed in Australia. In 1981, a study from the rural Western Australia town of Busselton using the oral glucose tolerance test (OGTT) revealed a diabetes prevalence of 3.4% (2.5% known cases and 0.9% newly diagnosed) in subjects 25 years and over⁴. The prevalence of IGT was 2.9%. On the basis of these data it was estimated that nearly 285,000 Australians had diabetes, and a further 241,000 had IGT. Between 1981 and the present, the only population based study which included blood testing using the oral



glucose tolerance test (OGTT) was conducted by Guest et al in rural Victoria². They studied non-Indigenous adults aged 15 years and over, finding a crude diabetes prevalence of 3.4% (1.6% known and 1.8% newly diagnosed). The IGT prevalence was 6.0%. Neither of these studies is adequately representative of the general demographic features of Australia.

By far the largest 'blood surveys' undertaken in Australia have been the three surveys conducted during the 1980s by the National Heart Foundation (NHF) to monitor the prevalence of cardiovascular risk factors in adults in capital cities^{5,14,15}. The surveys, conducted in 1980, 1983 and 1989, each included a questionnaire, physical measurements, blood pressure and a fasting blood sample in adults aged over 25 years. These are the only nationwide surveys of non-communicable disease risk factors previously conducted. The fasting glucose level was only collected in the 1983 survey. The use of only a fasting blood level to diagnose diabetes would result in a substantial underestimate of true diabetes prevalence¹⁶, and also provides no data on IGT.

Therefore, more than a decade has passed since a biomedical risk survey that includes blood samples was undertaken throughout Australia, and there is no definite date for a subsequent survey. As a consequence, there are no current estimates of the prevalence of major non-communicable disease risk factors, such as high levels of cholesterol or triglycerides or blood glucose, and other important factors which require a blood specimen. Furthermore, since the NHF surveys were conducted only in capital cities, there remains a deficiency of such data for rural areas.

Diabetes 1999 and beyond - the AusDiab Study

In recognition of the lack of adequate, recent representative data, the Australian Diabetes, Obesity and Lifestyle Study (AusDiab) is the first national study to provide estimates of the number of people with diabetes (based on blood tests) and its public health and societal impact. This important initiative is an integral component of the National Diabetes Strategy that resulted from the vision and commitment of the Federal Minister, Dr Michael Wooldridge, to tackle the mounting problem of diabetes and its complications in Australia.

Objectives of AusDiab

The specific objectives of the AusDiab Study were to:

1. Estimate the prevalence of diabetes and other forms of abnormal glucose tolerance.
2. Estimate the prevalence of related conditions of the Metabolic Syndrome¹³, including obesity, hypertension, and lipid profile abnormalities.
3. Assess the distribution and relationships of the cardiovascular risk factors indicated above.
4. Assess trends in risk factor levels as compared to those obtained in previous surveys in Australia.

Survey report

This report presents the main findings from the AusDiab Study of 1999-2000 based on data collected from a stratified sample of 11,247 Australians aged 25 years or over, residing in 42 randomly selected urban and non-urban areas (Census Collector Districts) of the six states of Australia and the Northern Territory.

All data for prevalence, unless otherwise stated, were weighted to the Australian population aged 25 years and over as projected to have applied at June 30, 1998¹⁷.

The findings with respect to the key matters of interest (disorders of glucose tolerance, weight and obesity status, lipid profile and blood pressure, smoking, physical activity and renal function) are presented separately. The data are presented according to age and gender. For the conditions of diabetes and hypertension, delineation is made of those who were aware of, or being treated for their condition, and those for whom any positive findings represented a new diagnosis.

More detailed analyses of the data from the survey, with particular reference to the prevalence and associations of the complications of diabetes, will be published subsequently in peer reviewed journals.

2 Diabetes mellitus

Background

The term diabetes mellitus describes a metabolic disorder of multiple causation characterized by chronically elevated blood glucose (hyperglycaemia) 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, lower limb amputations and renal failure.

Definition

The diagnostic criteria for the presence of diabetes, impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) were based on values for the oral glucose tolerance test (OGTT) venous plasma glucose concentration (fasting and 2-hr measurements) outlined in the WHO report on the Diagnosis and Classification of Diabetes Mellitus¹³.

Diabetes was further classified into ‘known’ and ‘undiagnosed’ diabetes.

Known diabetes

Participants were classified as having known diabetes if they satisfied at least one of the following criteria:

1. receiving current treatment in the form of tablets or insulin (or both) at the time of the study, or;

2. having ever been told by a doctor or nurse that they had diabetes, and had a fasting blood glucose or 2-hr post load glucose level over the cut-offs for diabetes mellitus (see Table 2.1).

Newly diagnosed diabetes

Newly diagnosed cases of diabetes consisted of those:

1. not presently receiving pharmacological treatment for diabetes, nor previously diagnosed with diabetes, and
2. who had fasting or 2-hour plasma glucose measurements over the diabetes cut-off range (see Table 2.1).

Table 2.1: Classification of glucose tolerance status

Classification	Plasma glucose (mmol/l)		
	Fasting		2-hr
Diabetes	7.0	or	11.1
IGT	< 7.0	&	7.8 - 11.0
IFG	6.1 - 6.9	&	< 7.8
Normal	< 6.1	&	< 7.8

All participants on hypoglycaemic medication were classified as having diabetes.

Results

Glucose tolerance status

The AusDiab survey found that the prevalence of diabetes was 7.5%: 8.0% for males and 7.0% for females. The prevalence of impaired glucose tolerance (IGT) was 10.6%: 9.2% for males and 12.0% for females. Impaired fasting glucose (IFG) was considerably more prevalent amongst males than females, having rates respectively of 8.1% and 3.3%, a combined rate of 5.7%.

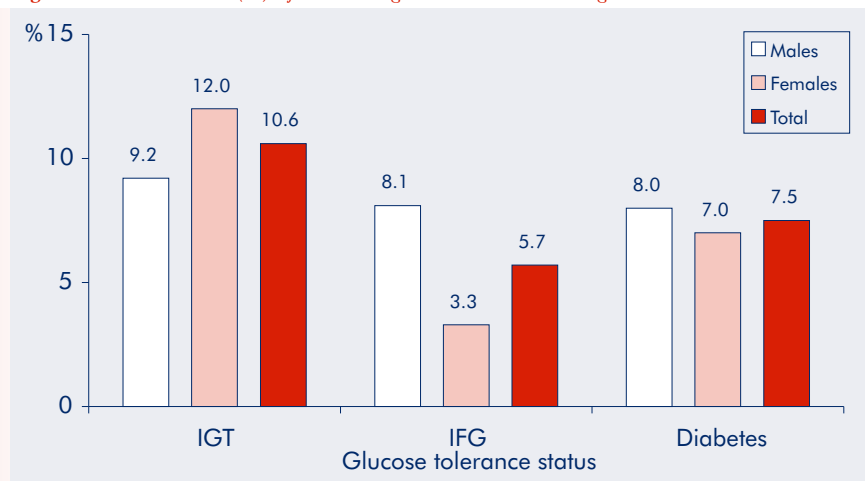
Prevalence of diabetes: known and newly diagnosed

The survey found that only about one half of the persons found to have diabetes were aware of having the condition, having been previously diagnosed.

Overall, for every known case of diabetes, there was one newly diagnosed case: that is, the real diabetes prevalence is twice that “self-reported”. This discrepancy between actual, and self-reported



Figure 2.1: Prevalence (%) of abnormal glucose tolerance among Australian residents



diabetes has been well documented² and described¹¹.

Table 2.2 shows the prevalence of known and newly diagnosed diabetes for the Australian population, and Figure 2.2 the gender combined prevalence of diabetes for the surveyed population. Figure 2.3 shows the age-specific prevalences of diabetes of each gender.

Applying the age specific prevalence of diabetes for each gender to the total population of Australia at June 30 1999¹⁸ produces an estimate of 938,700

people aged over 25 years with diabetes, which is consistent with previous estimates as shown in Figure 1.1, and considerably higher than other previous estimates¹¹. With the inclusion of Australians under the age of 25 years with Type 1 diabetes this represents about one million Australians with diabetes.

Prevalence of impaired glucose tolerance (IGT) and impaired fasting glucose (IFG)
The prevalence of IGT was 10.6%: 9.2% in males and 12.0% in females. Although the prevalence increased with age for both genders, its

Table 2.2: Age-and gender-specific prevalence (%) of known and newly diagnosed diabetes

Classification	Age (years)						Total
	25-34	35-44	45-55	55-64	65-74	75 +	
Males							
Known	0.0	1.1	2.8	8.6	12.8	13.8	4.3
Newly diagnosed	0.1	1.5	4.0	7.5	8.8	8.6	3.8
Females							
Known	0.3	0.9	3.5	4.6	7.3	9.7	3.3
Newly diagnosed	0.1	1.4	2.0	5.3	8.8	14.8	3.8
Persons							
Known	0.2	1.0	3.1	6.6	9.8	11.4	3.8
Newly diagnosed	0.1	1.4	3.0	6.5	8.8	12.3	3.8

Figure 2.2: Age-specific prevalence (%) of diabetes among Australian residents

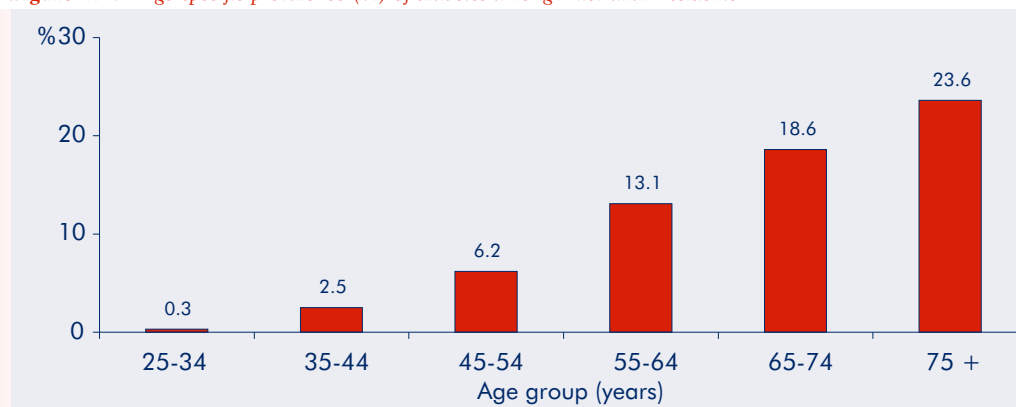
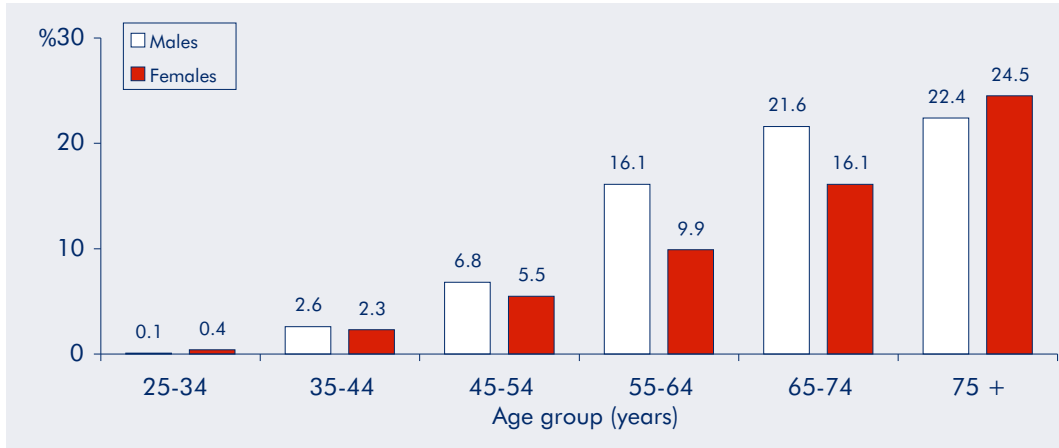


Figure 2.3: Age-and gender-specific prevalence (%) of diabetes among Australian residents



predominance in females decreased with age. The prevalence of IFG was 5.7%: 8.1 % in males and 3.3 % in females. The age distribution is distinctly different from that of IGT and diabetes with a peak in middle age and a lower rate in the older age groups. The prevalence of IFG was found to be considerably higher in males than females except for the oldest participants. Whereas for females the prevalence increased fairly consistently with age, for males it peaked in the 55-64 year group and was somewhat lower for the oldest participants.

The total prevalence of either of these forms of impaired glucose tolerance, for the total Australian population, would be nearly 2.1 million persons over the age of 25 years, which is more than twice that of diabetes. This ratio was markedly higher for the younger groups. The age specific prevalences are indicated in Figures 2.4 and 2.5.

Table 2.3 shows the age specific prevalence of IGT and IFG for the combined male and female population, and the combined prevalence of either of these forms of impaired glucose tolerance.

Table 2.3: Age-specific prevalence (%) of IGT and of IFG

Classification	Age (years)						Total
	25-34	35-44	45-55	55-64	65-74	75 +	
IGT	3.5	6.6	9.8	15.0	21.7	22.7	10.6
IFG	1.9	5.4	7.1	8.6	7.3	6.6	5.7
IGT or IFG	5.4	12.0	16.9	23.6	29.1	29.3	16.3

Figure 2.4: Age-specific prevalence (%) of IGT among Australian residents

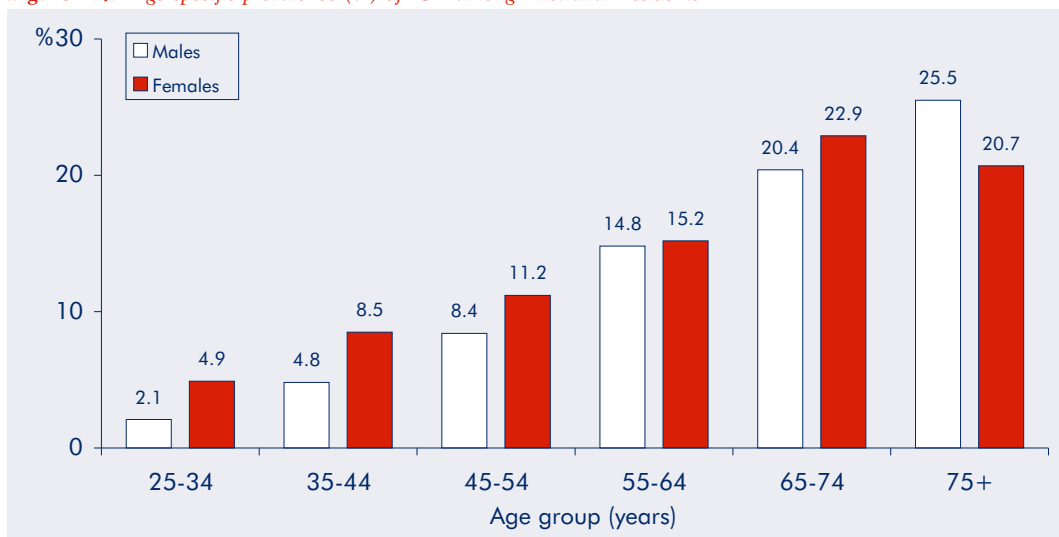
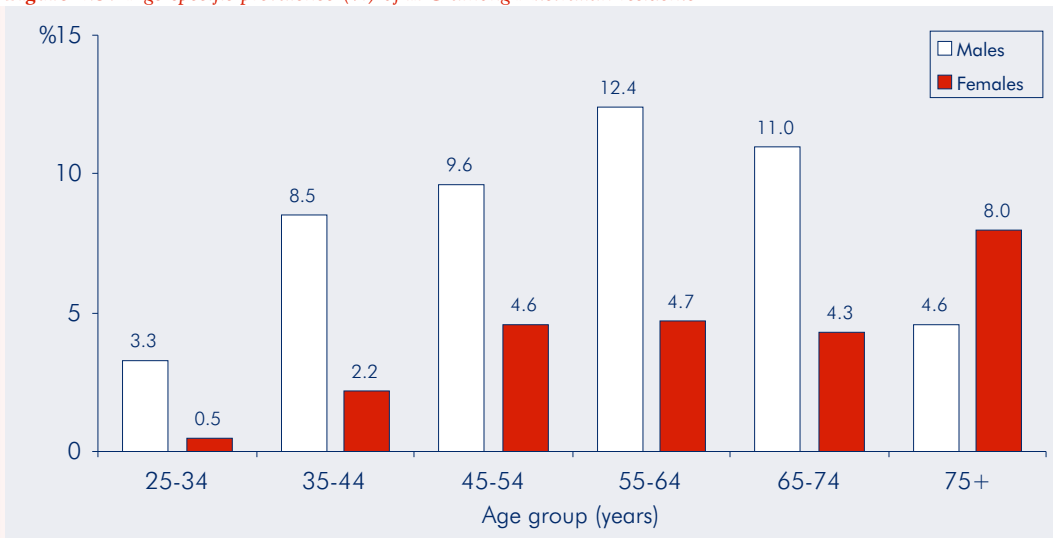




Figure 2.5: Age-specific prevalence (%) of IFG among Australian residents



Trends

Diabetes prevalence

We compared the estimate of diabetes prevalence derived from the 1981 Busselton survey⁴ with that from this survey. The methodologies and age distributions of the surveys were similar. Using the criteria of that survey (which involved a fasting level of 7.8 mmol/l, rather than the current 7.0 mmol/l) and age-standardised to the 1998 population¹⁷, the prevalence rates for diabetes were as shown in Table 2.4.

The age-specific prevalence of diabetes (for males and females combined) for the two survey populations is shown in the Figure 2.6, which highlights the rate at which diabetes prevalence has increased over the last twenty years, and the younger age at which its prevalence starts to increase markedly.

Table 2.4: Trends in the age-standardised prevalence (%) of diabetes: 1981 - 2000^a

Survey	Males	Females
Busselton ^b	4.0	3.2
AusDiab	6.9	6.6

^a Age-standardised to the 1998 Australian population¹⁷.

^b Busselton, 1981⁴.

Figure 2.6: Trends in the age-specific diabetes prevalence^a (%): 1981 - 2000

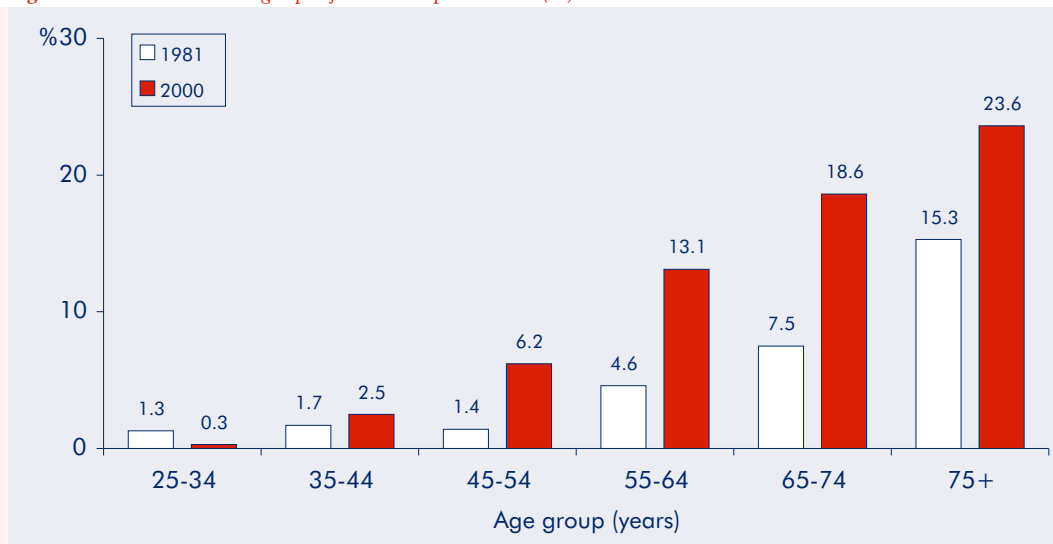


Table 2.5: Trends in the age-standardised prevalence (%) of IGT: 1981 – 2000^a

Survey	Males	Females
Busselton ^b	3.2	3.0
AusDiab	9.8	12.3

^a Age-standardised to the 1998 Australian population¹⁷.
^b Busselton, 1981⁴.

Trends in IGT prevalence

The IGT prevalence detected in the AusDiab survey was over twice that found in the Busselton survey of 1981 (using the Busselton survey criteria) for all age groups of males except those over 75 years. For females it was nearly four times the prevalence up to the age of 64 years, and thereafter more than

twice the prevalence. The prevalences (age-standardised to the 1998 Australian population¹⁷) are shown in Table 2.5.

No adequate data have been published to assess trends in IFG prevalence.

Abnormal glucose metabolism

The overall frequency of impaired glucose metabolism in the AusDiab sample was calculated as the prevalence of individuals with either IGT or IFG, or diabetes. The prevalence of impaired

glucose metabolism was 23.8%: for males 25.3% and for females 22.3%, which represents nearly 3 million Australians over the age of 25 years.

Diabetes and associated conditions

The association of diabetes with a number of other metabolic conditions has been well documented in Australia¹². Table 2.6 shows the prevalence of a number of these conditions among the participants according to glucose tolerance status.

difference in the demographics between the diabetic and non-diabetic populations, some of the differences in prevalence of associated conditions were reduced. However, obesity and hypertriglyceridaemia were approximately three times as frequent, and hypertension and depressed HDL approximately twice as frequent in the diabetic as in the normal population.

Table 2.6 indicates a strong association of abnormal glucose tolerance, in particular diabetes, with a number of other cardiovascular risk factors. After age and sex adjustments to account for the

Table 2.6: Weighted prevalence (%) of associated conditions stratified by glucose tolerance status

Associated condition	Glucose tolerance status			
	Diabetes	IFG	IGT	Normal
Hypertension ^o	69.3	43.5	50.1	21.1
Obesity (BMI ≥ 30 kg/m ²)	44.4	30.1	31.5	15.9
LDL (≥ 3.5 mmol/l)	45.9	59.6	53.0	44.1
HDL (<1.0 mmol/l)	23.1	16.8	11.6	10.6
Triglycerides (≥ 2.0 mmol/l)	42.9	31.4	31.1	16.0

^o On treatment, or systolic pressure ≥ 140 mm Hg, or diastolic pressure ≥ 90 mmHg.

Discussion

Diabetes prevalence

There were four findings of major importance:

- The high absolute prevalence of diabetes - nearly a million people.
- The prevalence detected was twice that detected in Busselton 20 years ago.

- Diabetes remains a condition for which diagnosis and hence therapy and monitoring for complications is only of the order of 50% of the prevalence.
- The onset of diabetes is occurring earlier.

All of these factors have major implications for



public health and the national health burden, in that failure to adequately treat is associated with an increased rapidity of progression to the many complications of diabetes^{19,20}.

IFG and IGT prevalence

In comparison with the Busselton data, this survey detected a substantial increase in the prevalence of

IGT. The data also suggested an increase in the prevalence of IFG in men. In that these conditions are both likely precursors to diabetes^{13,21}, it is hoped that more prompt detection will provide an opportunity for intervention programs to be implemented at an early stage.

3 Overweight and obesity

Background

Since obesity is strongly linked to Type 2 diabetes, the term ‘diabesity’ has been recently used to embrace the two conditions. Obesity is a major risk factor not only for Type 2 diabetes, but also for other chronic conditions such as hypertension, CVD,

dyslipidaemia and arthritis. The distribution of adipose tissue is important in that abdominal rather than peripheral fat is particularly associated with more pronounced risks for diabetes and CVD^{22,23}.

Definition

Weight status was defined using the WHO classification²⁴ for Europids, based on the Body Mass Index (BMI). The BMI = weight (kg) / height (m)².

Measurement of waist circumference (cm) is also used as an indicator of abdominal obesity²⁴.

Table 3.1: Classification of weight status by BMI

Classification	BMI (kg/m ²)
Not Overweight	< 25.0
Overweight	
Pre-obese	25.0 – 29.9
Obese	30.0

Table 3.2: Classification of abdominal obesity by waist circumference

Classification	Waist circumference (cm)	
	Males	Females
Not Overweight	< 94.0	< 80.0
Overweight		
Pre-obese	94.0 – 101.9	80.0 – 87.9
Obese	102.0	88.0

Results

Prevalence of overweight

Almost 60% of Australians were overweight using either BMI or waist circumference to classify weight status. Using BMI as the index, 59.6% of participants were overweight: 67.4% of males and 52.0% of females. For waist circumference, 55.9% of the participants were overweight: 55.2% of males and 56.5% of females. These prevalences are shown in Figure 3.1. The age-specific prevalences of overweight according to the two parameters are shown in Table 3.3.

Over 60% of males were overweight by the index of BMI for all age groups. Younger females had a considerably lower prevalence of overweight than did younger males, but the gender differences became much smaller with increasing age.

Using waist circumference as the index, a similar

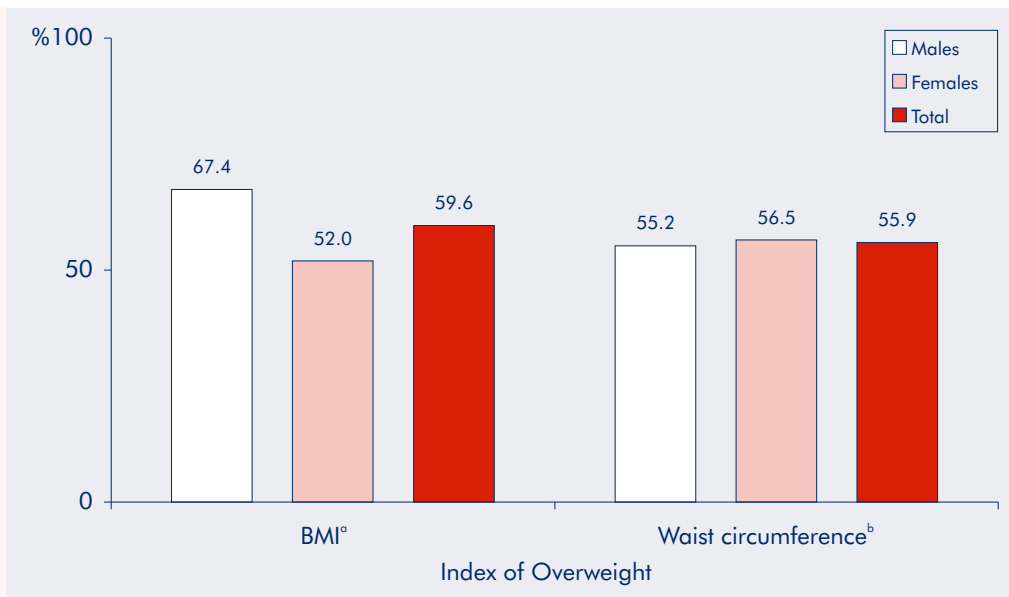
percentage of males and females were overweight. The prevalence was nearly 40% for the youngest subgroup, but increased to about 75%, for those between the ages of 65 and 74 years. The eldest subgroup had a slightly lower prevalence of overweight.

Prevalence of obesity

Obesity, that is a BMI ≥ 30 kg/m², is the more severe category of being overweight. On the basis of BMI, 20.5% of participants were obese: 19.1% of males and 21.8% of females. The prevalence was even higher (30.3%) if waist circumference was used as the index: 26.6% of males, and 33.9% of females. The prevalences of these two indices of obesity are shown in Figure 3.2. Table 3.4 shows that using either index obesity is very prevalent in Australia. The rates increase with age, peaking in the 55-74 year age group.



Figure 3.1: Prevalence (%) of abnormal BMI or waist circumference among Australian residents



^a BMI 25 kg/m².

^b Waist circumference: males 94 cm; females 80 cm.

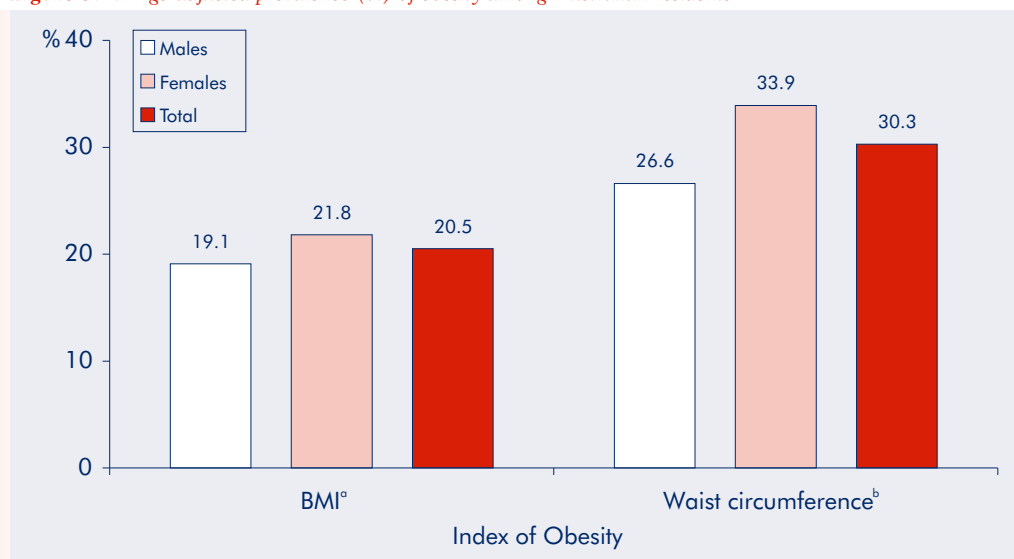
Table 3.3: Age-specific prevalence (%) of overweight by BMI and waist circumference

Classification	Age (years)						Total
	25–34	35–44	45–54	55–64	65–74	75 +	
BMI^a							
Males	60.5	64.2	72.4	74.0	73.1	63.8	67.4
Females	35.1	44.5	58.1	67.6	68.9	52.2	52.0
Persons	48.1	54.4	65.3	70.8	70.8	57.1	59.6
Waist^b							
Males	40.1	51.3	58.3	66.6	71.2	64.8	55.2
Females	36.6	46.9	59.1	72.7	79.7	67.5	56.5
Persons	38.4	49.1	58.7	69.6	75.9	66.3	55.9

^a BMI 25 kg/m².

^b Waist circumference: males 94 cm; females 80 cm.

Figure 3.2: Age-adjusted prevalence (%) of obesity among Australian residents



^a BMI 30 kg/m².

^b Waist circumference: males 102 cm; females 88 cm.

Table 3.4: Age-specific prevalence (%) of obesity by BMI and waist circumference

Classification	Age (years)						Total
	25-34	35-44	45-54	55-64	65-74	75+	
BMI^a							
Males	17.0	17.5	20.5	25.5	20.5	11.6	19.1
Females	12.2	19.4	26.0	31.9	29.7	14.9	21.8
Persons	14.7	18.4	23.2	28.7	25.5	13.5	20.5
Waist^b							
Males	13.6	24.6	27.4	35.8	41.2	36.8	26.6
Females	17.1	25.6	37.6	46.7	52.2	43.0	33.9
Persons	15.3	25.1	32.4	41.2	47.3	40.5	30.3

^a BMI 30 kg/m².

^b Waist circumference: males 102 cm; females 88 cm.

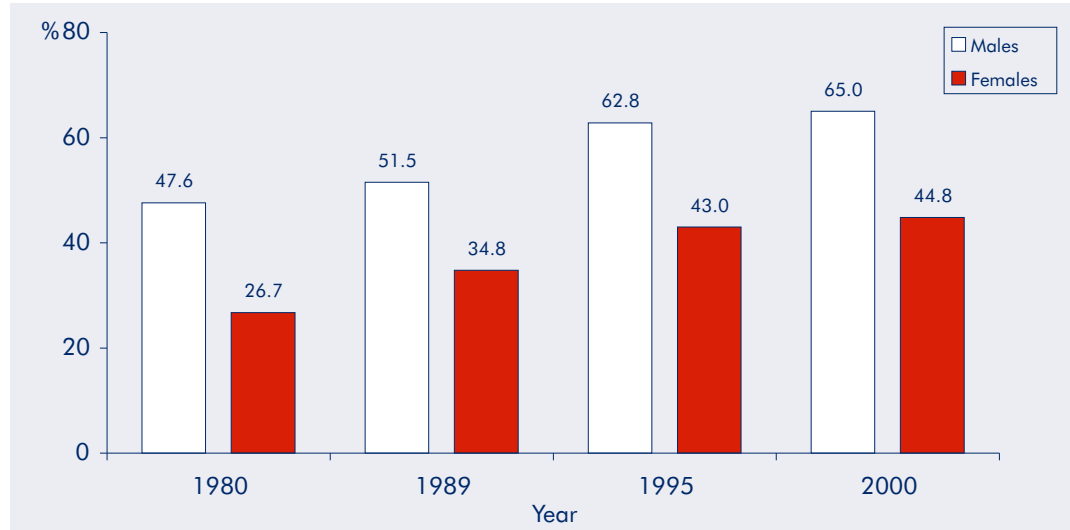
Trends

Overweight

The proportions of participants who were overweight and obese were compared with data obtained during the 1980 and 1989 NHF Risk Factor Prevalence Studies^{14,15} and the ABS National Nutrition Survey of 1995²⁵. For consistency purposes, the data were confined to the capital city participants of these surveys (and urban participants of the National Nutrition Survey), who were aged 25-64 years of age. The levels of

overweight detected in this study were considerably higher than those reported by the NHF surveys, although not markedly different from those of the ABS for 1995. Figure 3.3 indicates the extent to which the prevalence of overweight (BMI ≥ 25 kg/m²) has increased over the past 20 years (age-standardised to the June 30, 1991 population²⁶) and supports the finding of an upward trend of excess weight described in the 1998 AIHW Report "Australia's Health, 1998"³.

Figure 3.3: Trends in the age-standardised^a prevalence (%) of overweight^b: 1980 - 2000



^a Age-standardised to 1991 Australian population²⁶.

^b BMI ≥ 25 kg/m².



Obesity

Table 3.5 and Figures 3.4 and 3.5 highlight the extent to which the higher level of obesity has increased over the past 20 years for both men and women aged 25-64 years for the urban or capital city residents in the 1995 survey, and capital city residents of the other three surveys (age-standardised to the June 30, 1991 population²⁶). Figures 3.4 and 3.5 show the age specific prevalences of obesity, for the participants of the 1980 and 1989 NHF surveys^{14,15} and AusDiab capital city participants.

Whereas Figure 3.3 shows the marked trend for an increase in the prevalence of a BMI ≥ 25 kg/m² from 1980 to 2000, Figures 3.4 and 3.5 suggest that much of this increase has occurred at the higher level of BMI (≥ 30 kg/m²). For males, the prevalence of being at least overweight increased from 1980 to 2000 by 17.5%, while the prevalence of obesity increased by 10%. For females the increase in overweight was 18%, of which 12% was in the obesity component. For men this increase has been a more than doubling, and for women a near tripling, in obesity over the past 20 years.

Table 3.5: Trends in the age-standardised prevalence^a (%) of obesity^b: 1980 – 2000

Gender	Year			
	1980 ^c	1989 ^d	1995 ^e	2000
Males	7.2	9.3	17.6	17.1
Females	7.0	11.1	16.1	18.9

^a Age-standardised to 1991 Australian population²⁶.

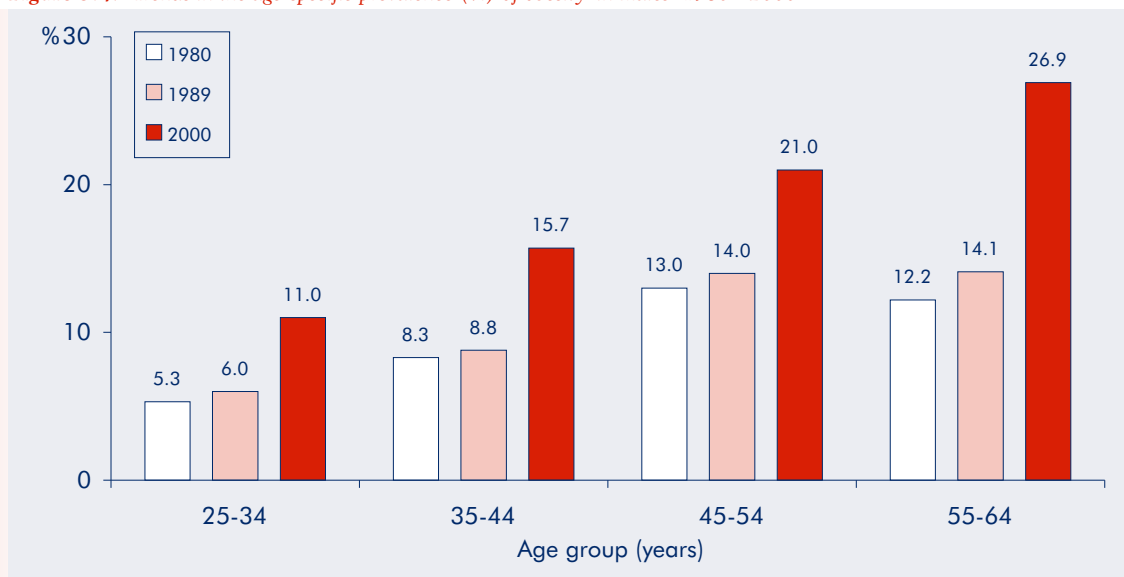
^b BMI ≥ 30.0 kg/m².

^c NHF Risk Factor Prevalence Survey¹⁴.

^d NHF Risk Factor Prevalence Survey¹⁵.

^e ABS National Nutrition Survey²⁵ (includes urban participants).

Figure 3.4: Trends in the age-specific prevalence (%) of obesity^a in males: 1980 - 2000^b



^a BMI ≥ 30 kg/m².

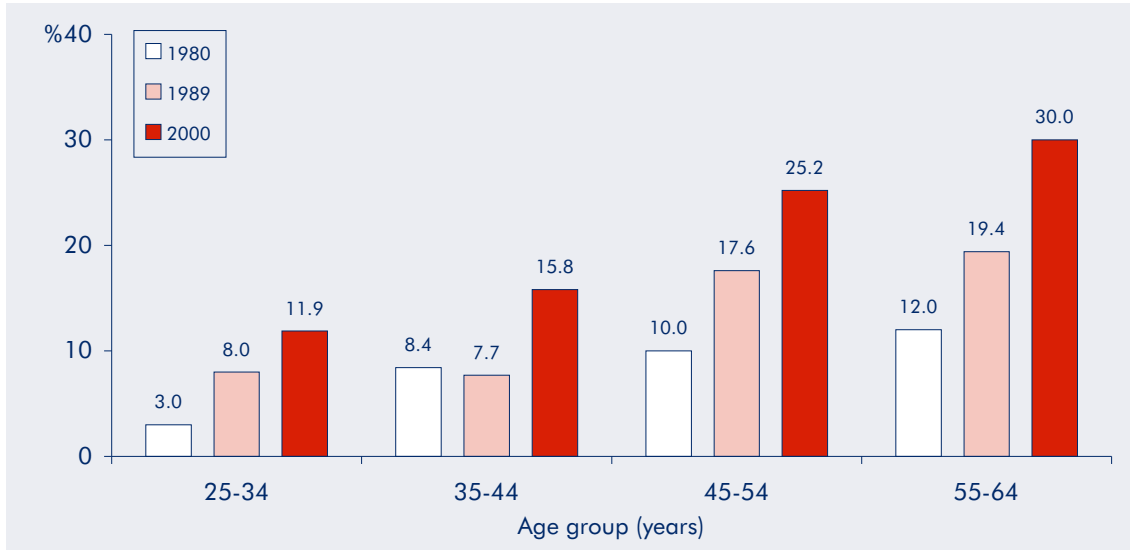
^b Age-standardised to 1991 Australian population¹⁷.

Discussion

The principal finding was the high prevalence of being overweight, both in the pre-obese and obese ranges amongst the AusDiab participants. A comparison with previous data from nationwide surveys^{14,15} shows that there has been a continuation in the clear trend of increasing prevalence of excess weight, previously noted in the Australian Institute of Health and Welfare report of 1998³.

The likelihood is that the increased levels of obesity have been a significant contributing factor in the escalating prevalence of diabetes. The epidemic of obesity must be curtailed in order to reduce the impact on diabetes, as well as other obesity-related conditions.

Figure 3.5: Trends in the age-specific prevalence (%) of obesity^a in females: 1980 - 2000^b



^a BMI ≥ 30 kg/m².

^b Age-standardised to 1991 Australian population¹⁷.



4 Dyslipidaemia

Background

Blood lipid levels reflect genetic background but are modified by diet, obesity and other lifestyle factors. Total cholesterol levels are a crude but consistent measure of CVD risk, particularly for coronary heart disease. The CVD risk relating to high total cholesterol is further refined by considering cholesterol fractions, namely elevated

LDL and depressed HDL cholesterol - both of which are powerful predictors of CVD. Elevated triglycerides are also a CVD risk factor, especially when combined with low HDL cholesterol as often occurs in diabetes and the Metabolic Syndrome.

Definition

The following criteria for abnormal lipid levels are based on recommendations by the National Heart Foundation¹⁵ and the Australian Diabetes Society²⁷.

Classification was made on the basis of plasma level, irrespective of treatment status.

Table 4.1: Classification of lipid values

Classification	Blood lipid concentration (mmol/l)			
	Cholesterol	HDL-cholesterol	LDL-cholesterol	Triglycerides
Normal	< 5.5	1.0	< 3.5	< 2.0
Abnormal	5.5	< 1.0	3.5	2.0

Results

Prevalence of dyslipidaemia

The prevalence of elevated total cholesterol levels (Figure 4.1) was 51.2%: 51.1% for males and 51.2% for females. The prevalence of elevated LDL cholesterol was 45.8%: 49.8% for males and 42.2% for females, and of reduced HDL cholesterol 11.9%: 18.6% for males and 5.5% for females. Males overall had a higher prevalence of elevated triglycerides than females, with rates of 24.5% and 16.6% respectively - a combined rate of 20.5%.

dyslipidaemias (Table 4.2) peaked in the 45-54 age group (apart from total cholesterol, which peaked in the 55-64 age group). In females, however, the peak prevalence was seen uniformly in the 65-74 age group. The prevalence of low HDL was consistently higher in males than females, being 2.5 to 6.1 times greater in males, depending on age group. Elevated LDL and elevated triglycerides were both more prevalent in males than females up to the age of 54; thereafter, these abnormalities were seen slightly more frequently in females.

In males, the prevalence of each of the

Figure 4.1: Prevalence (%) of abnormal lipid levels for Australian residents

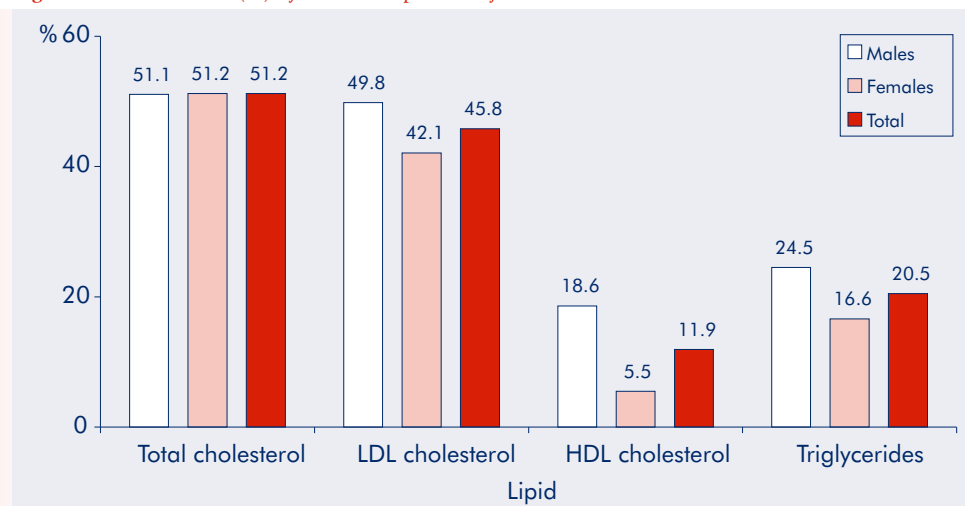


Table 4.2: Age- and gender-specific prevalence (%) of abnormal lipid levels

Lipid	Age (years)						Total
	25–34	35–44	45–54	55–64	65–74	75 +	
Males							
Total cholesterol ^a	31.8	55.3	60.7	61.4	53.8	49.5	51.1
LDL cholesterol ^b	34.2	52.9	58.9	58.1	51.0	48.6	49.8
HDL cholesterol ^c	18.3	18.0	20.3	19.2	17.7	17.1	18.6
Triglycerides ^d	14.5	26.2	31.4	30.0	24.8	19.9	24.5
Females							
Total cholesterol ^a	31.2	39.0	55.0	71.2	75.0	65.4	51.2
LDL cholesterol ^b	22.6	35.3	46.3	57.5	63.7	50.1	42.1
HDL cholesterol ^c	6.3	4.1	5.7	6.0	7.0	2.8	5.5
Triglycerides ^d	9.9	9.4	17.0	25.2	29.9	20.7	16.6
Persons							
Total cholesterol ^a	31.5	47.1	57.9	66.3	65.4	58.8	51.2
LDL cholesterol ^b	28.3	43.9	52.5	57.8	58.0	49.5	45.8
HDL cholesterol ^c	12.2	11.0	13.1	12.7	11.8	8.7	11.9
Triglycerides ^d	12.2	17.8	24.3	27.6	27.6	20.4	20.5

^a Total cholesterol 5.5 mmol/l.
^b LDL cholesterol 3.5 mmol/l.
^c HDL cholesterol < 1.0 mmol/l.
^d Triglycerides 2.0 mmol/l.

Lipid therapy

Lipid lowering agents were being taken by 7.3% of the population, comprising 7.3% of the male and 7.4% of the female populations respectively. Table 4.3 shows the prevalence of any lipoprotein abnormality for those not on lipid therapy, and usage rates of lipid lowering therapy by age and gender. For both genders, the use of lipid therapy

increased markedly with age, from nearly no use for the youngest participants to over 20% for those aged 65-74 years. At all ages therapy occurred at less than 50% of the lipid abnormality rate, and these figures barely changed when an elevated triglyceride level was removed from the combination producing any lipoprotein abnormality.

Table 4.3: Age and gender-specific prevalence (%) of any lipid abnormality, and usage of lipid-lowering therapy

Classification	Age (years)						Total
	25 – 34	35 – 44	45 – 54	55 – 64	65 – 74	75 +	
Males							
Abnormality ^a	50.4	68.0	71.7	66.9	61.1	61.6	63.3
Lipid Therapy	0.2	2.7	6.9	16.1	20.3	11.5	7.3
Total ^b	50.6	70.7	78.6	83.0	81.4	73.1	70.6
Females							
Abnormality ^a	39.9	46.3	59.5	68.2	67.9	62.3	54.3
Lipid Therapy	0.2	0.7	5.7	12.7	22.0	19.8	7.4
Total ^b	40.1	47.0	65.3	80.9	89.9	82.2	61.7
Persons							
Abnormality ^a	45.1	57.2	65.7	67.6	64.8	62.0	58.8
Lipid Therapy	0.2	1.7	6.3	14.4	21.2	16.4	7.3
Total ^b	45.3	58.9	72.0	82.0	86.0	78.4	66.1

^a Abnormality: calculated as % of total population, and comprises participants who were not taking lipid lowering therapy and had any of:
 i. Total cholesterol 5.5 mmol/l
 ii. LDL cholesterol 3.5 mmol/l
 iii. HDL cholesterol < 1.0 mmol/l
 iv. Triglyceride 2.0 mmol/l.
^b Totals may not exactly equal the sum of the two alternatives because of rounding.



Trends

Total cholesterol

Despite the increasing awareness of the importance of lipid control, there has been no reduction in the prevalence of elevated cholesterol levels since the 1980 NHF survey¹⁴. The lack of a clear trend of mean cholesterol during the 1980s has been described previously²⁸.

Table 4.4 shows the proportion of males and females with total cholesterol ≥ 5.5 mmol/l, age-standardised to the 1991 Australian population²⁶, for the NHF^{5,14,15} and capital city participants of the AusDiab surveys aged up to 64 years, in order to ensure comparability with the earlier surveys.

The mean total cholesterol levels for these four survey populations has also changed little since 1980 as can be seen in Table 4.5.

Triglyceride level trends

Table 4.6 shows the change in the prevalence of elevated triglycerides since 1980, for the same

survey populations as for the total cholesterol.

Except for the 1983 data, the prevalence of hypertriglyceridaemia in males has shown little change in the last two decades. In the same time period, the prevalence in females has doubled.

Trends in lipid therapy

The rates of lipid therapy use were similar to those reported by the 1989 NHF study¹⁴ for men up to the age of 64 years and women up to the age of 54 years, but for older persons the current rates of treatment are considerably higher than the rates reported 10 years ago.

The use of such therapy is markedly higher than that reported by the 1980 and 1983 NHF studies^{5,13}, and Table 4.7 shows the age and gender standardised rates of usage of such therapy. Only capital city participants of the AusDiab survey aged less than 65 years were included, for consistency.

Table 4.4: Trends in the age-standardised prevalence^a (%) of total cholesterol ≥ 5.5 mmol/l: 1980 - 2000

Gender	Year			
	1980 ^b	1983 ^c	1989 ^d	2000
Males	49.5	51.6	50.9	47.4
Females	40.9	48.1	42.8	43.8

^a Age-standardised to 1991 Australian population²⁶.

^b NHF Risk Factor Prevalence Survey¹³.

^c NHF Risk Factor Prevalence Survey⁵.

^d NHF Risk Factor Prevalence Survey¹⁴.

Table 4.5: Trends in mean age-standardised total cholesterol levels^a (mmol/l): 1980 - 2000

Gender	Year			
	1980 ^b	1983 ^c	1989 ^d	2000
Males	5.6	5.6	5.7	5.5
Females	5.4	5.6	5.6	5.4

^a Age-standardised to 1991 Australian population²⁶.

^b NHF Risk Factor Prevalence Survey¹³.

^c NHF Risk Factor Prevalence Survey⁵.

^d NHF Risk Factor Prevalence Survey¹⁴.

Table 4.6: Trends in the age-standardised prevalence^a (%) of triglycerides ≥ 2.0 mmol/l: 1980 - 2000

Gender	Year			
	1980 ^b	1983 ^c	1989 ^d	2000
Males	18.2	12.0	18.6	20.8
Females	5.3	5.0	6.8	12.7

^a Age-standardised to 1991 Australian population²⁶.

^b NHF Risk Factor Prevalence Survey¹³.

^c NHF Risk Factor Prevalence Survey⁵.

^d NHF Risk Factor Prevalence Survey¹⁴.

Table 4.7: Trends in the age-standardised usage^a (%) of lipid-lowering therapy: 1980 - 2000

Gender	Year			
	1980 ^b	1983 ^c	1989 ^d	2000
Males	1.3	0.5	3.4	5.1
Females	1.2	0.5	1.9	3.3

^a Age-standardised to 1991 Australian population²⁶.

^b NHF Risk Factor Prevalence Survey¹³.

^c NHF Risk Factor Prevalence Survey⁵.

^d NHF Risk Factor Prevalence Survey¹⁴.

Discussion

The most important findings from AusDiab are the very high prevalence of abnormalities, particularly for total and LDL cholesterol, even amongst the younger participants. As this section of the population ages over the next 20 years, these levels of dyslipidaemia signal the potential for a major burden of cardiovascular disease.

Abnormalities in total cholesterol and LDL cholesterol showed very similar profiles, with over half the population having elevated levels. Nonetheless, the rates of elevation of total cholesterol were similar to those detected in the previous NHF surveys^{5,13,14}.

Hypertriglyceridaemia was more common for males than females, except for the oldest participants, but the increase in its prevalence since 1980 has been especially pronounced for females. Possibly this parameter is more closely related to obesity than the other lipoprotein measures, and the rise in obesity prevalence has unmasked this tendency in women.

The use of medication for lipoprotein abnormalities increased consistently with age. Although the usage of such therapy has increased markedly among those aged over 55 years during the last ten years, over 75% of those not on therapy had abnormal lipoprotein profiles. This figure was considerably higher for the younger participants, with only a small fraction of those with abnormal lipoprotein levels being on drug treatment. Although over 40% of men aged between 25 and 44 years had elevated total cholesterol levels, fewer than 2% were on therapy. In this younger group, awareness of lipid levels should be promoted so as to encourage healthy dietary practices (low saturated fat intake to reduce LDL levels) and lifestyle measures (exercise and weight control to raise HDL and lower triglycerides). In the middle aged and elderly, and especially where vascular disease exists, drug therapy has an important role.



5 Hypertension

Background

Elevated blood pressure represents an important risk factor for cardiovascular and renal disease in the general population. Amongst those with diabetes, it is a risk factor for micro-vascular

complications as well as being an additive risk factor for cardiovascular disease. Thus hypertension is of major significance to the whole population^{29,30}.

Definition

The presence or absence of hypertension was determined for each participant in accordance with

WHO guidelines³¹, as shown in Table 5.1.

Table 5.1: Classification of blood pressure

Classification	Blood pressure (mmHg)		
	Systolic	and	Diastolic
Normal	< 140	and	< 90
Hypertension	140	or	90

Participants were also classified as hypertensive if they were on medication for blood pressure, irrespective of their current hypertension status.

Results

Hypertension status

The age-specific prevalences of hypertension for each gender are shown in Figure 5.1, and demonstrate the discrepancy between male and female rates, particularly at the younger ages, and the extent to which prevalence increases with age.

The overall prevalence of hypertension was 28.8%: 30.6% for males and 27.1% for females (Table 5.2).

Anti-hypertensive medication use

The diagnostic criteria for hypertension recommended by the WHO include both untreated persons with hypertension and those who have been diagnosed and are on treatment. At all ages untreated hypertension was more common among men than women. Overall, for every person being treated for hypertension there was another untreated person. Table 5.2 shows whether the participants were classified as hypertensive on the

Figure 5.1: Age-specific prevalence (%) of hypertension in Australian residents

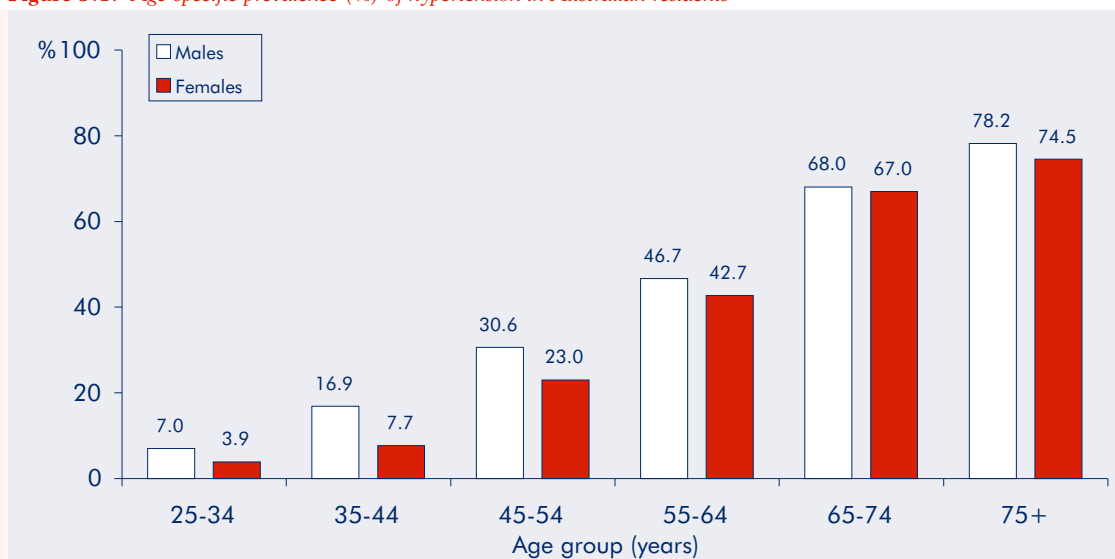


Table 5.2: Age-specific classification by treatment status of hypertensive participants

Hypertension Category	Age (years)						Total
	25 – 34	35 – 44	45 – 54	55 – 64	65 – 74	75 +	
Males							
Untreated ^a	7.0	14.0	20.0	26.3	33.3	44.8	19.2
Treated	0.0	2.9	10.5	20.5	34.5	33.3	11.5
Total hypertensive^b	7.0	16.9	30.6	46.7	67.9	78.1	30.6
Females							
Untreated ^a	2.7	4.3	11.1	21.1	25.1	25.8	11.8
Treated	1.2	3.2	11.9	21.7	41.8	48.7	15.3
Total hypertensive^b	3.9	7.5	23.0	42.7	67.0	74.4	27.1
Persons							
Untreated ^a	4.8	9.2	15.6	23.7	28.8	33.7	15.4
Treated	0.6	3.0	11.2	21.1	38.5	42.3	13.4
Total hypertensive^b	5.4	12.2	26.8	44.7	67.4	76.0	28.8

^a Systolic pressure \geq 140 mmHg, or diastolic pressure \geq 90 mmHg, and not on anti-hypertensive medication.

^b The totals may not exactly equal the sums of the two components because of rounding.

basis of their blood pressure or because of medication use, with the percentage being of the total population.

Medication to control hypertension was being taken by 13.4% of the population: 11.5% of the male and 15.3% of female participants. In both genders the usage of such medication increased with age, from levels of 1% or less for the youngest groups, to nearly 50% for the oldest female group. Of those with hypertension, nearly 40% of males and over 55% of females were taking medication for hypertension, with the remaining 60% of males and 45% of females being untreated cases of hypertension.

The age-specific prevalence of hypertensive

persons who were on medication is shown in Figure 5.2.

As was shown in Figure 5.2 the treatment rate of hypertension tended to increase with age, except for the oldest male group, who had the highest prevalence of untreated hypertension.

Blood pressure status of treated participants

Of the 13.4% of Australians who were on anti-hypertensive medication, 34.3% of males and 46.4% of females were adequately controlled, with blood pressures less than 140/90 mmHg. This percentage, perhaps surprisingly, tended to decrease with age and is shown in Table 5.3.

Figure 5.2: Age-specific percentage (%) of hypertensive participants on treatment

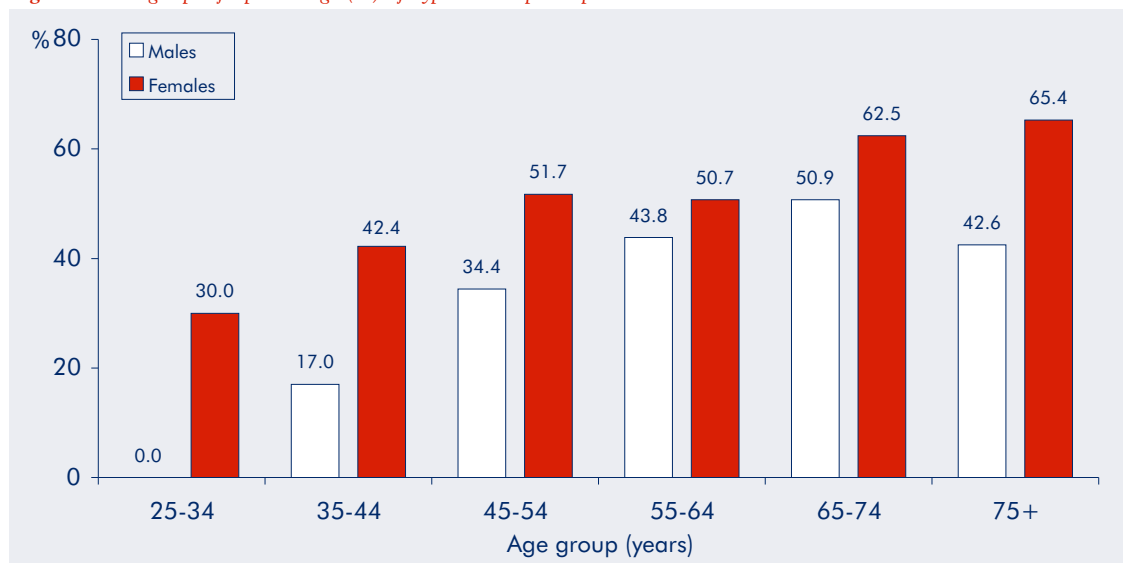




Table 5.3: Prevalence (%) of adequate blood pressure^a control among people on anti-hypertensive therapy

	Age (years)						Total
	25–34	35–44	45–54	55–64	65–74	75 +	
Males		55.8	45.3	37.4	28.7	20.1	34.3
Females	75.5	75.9	59.2	47.6	43.2	33.7	46.4
Persons	75.5	66.4	52.6	42.6	37.3	29.3	41.4

^a Systolic pressure < 140 mmHg, and a diastolic pressure < 90 mmHg, and on anti-hypertensive medication.

Trends

Trends in hypertension status

Figure 5.3 shows the male and female age-standardised prevalence of hypertension, for those participants of the AusDiab study aged 25-64 years, residing in capital cities as well as the results from the three NHF surveys, using the following criteria:

1. Systolic blood pressure \geq 140 mm Hg, or
2. Diastolic blood pressure \geq 90 mm Hg, or
3. Taking medications for blood pressure.

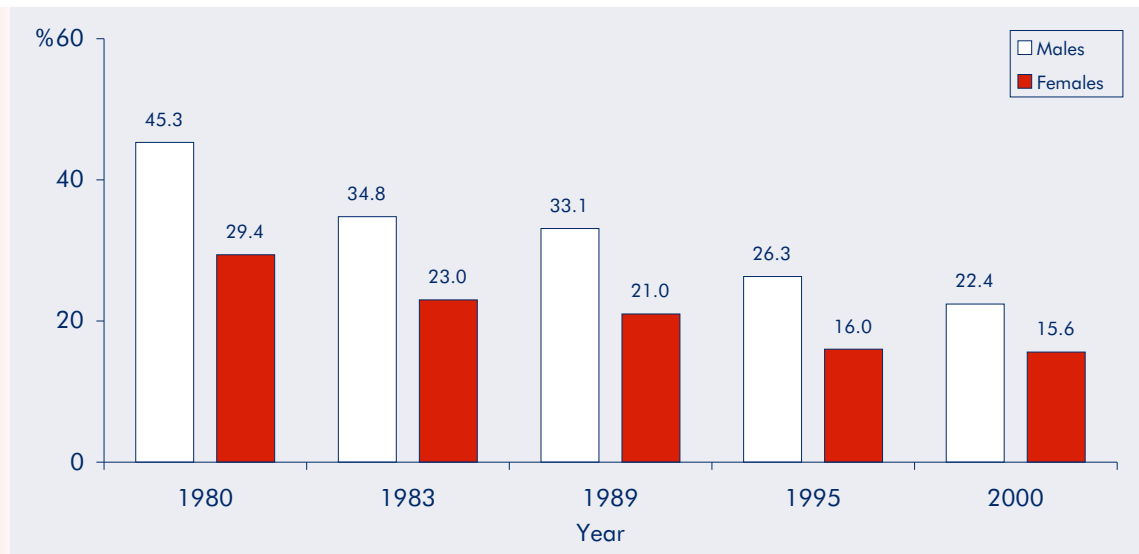
Figure 5.3 demonstrates a consistent and

progressive fall in the prevalence of hypertension in both males and females.

Trends in anti-hypertensive medication use

The current data were compared with that obtained from the three NHF surveys^{5,13,14}, as they all obtained information as to usage of anti-hypertensive medication. The data suggested little change in the rates at which these medications have been being used, other than the higher usage among women in 1980. Table 5.4 shows the extent of usage of such drugs for those studies and the participants of the AusDiab study residing in capital cities.

Figure 5.3: Trends in the age-standardised prevalence^a (%) of hypertension: 1980 - 2000



^a Age-standardised to the 1991 Australian population¹⁷.

Table 5.4: Trends in the age-standardised prevalence^a (%) of anti-hypertension medication use 1980 - 2000

Gender	Year			
	1980 ^b	1983 ^c	1989 ^d	2000
Males	6.6	7.7	7.5	5.9
Females	10.1	7.8	7.7	6.4

^a Age-standardised to 1991 Australian population²⁶.

^b NHF Risk Factor Prevalence Survey¹³.

^c NHF Risk Factor Prevalence Survey⁵.

^d NHF Risk Factor Prevalence Survey¹⁴.



Discussion

As a major risk factor for both coronary heart and cerebrovascular disease, it is critically important that hypertension should be prevented, recognised and controlled. The trend of a lowering prevalence of hypertension reported previously³ appears to be continuing, notwithstanding little change in the usage of anti-hypertensive medication. Such a lack of change in medication use is surprising given the introduction over this period of a number of efficacious agents with fewer side effects than the older medications.

Nonetheless, hypertension remains a major risk factor for most manifestations of vascular disease,

with particular significance for those with co-existing risk factors. As with diabetes, there is nearly one untreated and possibly undiagnosed person with hypertension for every person on treatment.

For those on treatment, a large proportion continued to have elevated blood pressure levels. Notwithstanding that no information was available as to participants' blood pressure prior to treatment, nor the duration or type of therapy, it is of concern that so many treated hypertensive persons remain hypertensive despite treatment.



6 Smoking

Background

Cigarette smoking represents a major risk factor for a large number of respiratory and vascular diseases, and its effects on such conditions increases the risk of these occurring as

complications of other conditions already described, such as diabetes, hypertension and dyslipidaemia.

Definition

The following criteria were used to define smoking status (current smoker, ex-smoker, never smoked)

for the participants, based on self-reported data from the questionnaires.

Table 6.1: Classification of smoking status

Classification	Amount Smoked
Current	At least daily
Ex-smoker	Less than daily for at least the last three months
Never smoked	Smoked < 100 cigarettes during life

Results

The prevalence of being a current smoker was 15.6% (Figure 6.1): for males 18.2% and females 13.1%. The ex-smoker rate was 26.9%: 31.4% for males and 22.6% for females. The remaining 57.5% had never been smokers: 50.4% of males and 64.3% of females.

peaked for males in the 35-44 year age group, for females the highest prevalence was in the youngest age group. For males, the proportion that had never smoked was highest for the youngest groups and decreased with increasing age. However, for females, the highest rates for never having smoked were among the oldest participants, with past or present smoking combined being most prevalent in the 35-44 year age group.

Table 6.2 shows the smoking status of the participants by age for each gender. Current smoking was more common among males than females at all ages, and whereas current smoking

There was a marked discrepancy between the

Figure 6.1: Prevalence (%) of smoking status among Australian residents

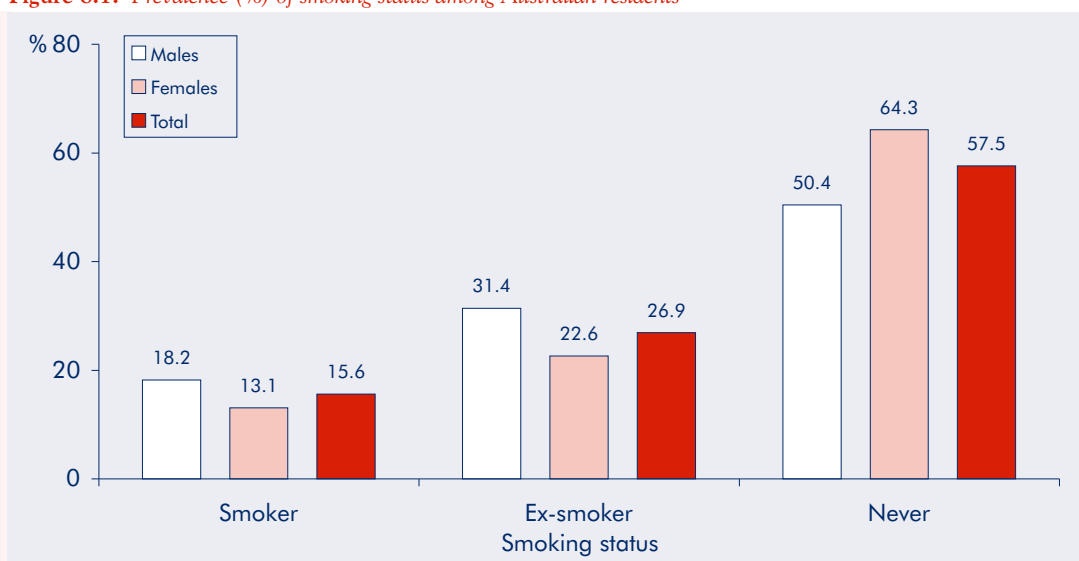


Table 6.2: Age-specific prevalence (%) of smoking

Smoking Status	Age (years)						Total
	25–34	35–44	45–54	55–64	65–74	75+	
Males							
Smoker	22.5	23.6	18.2	13.4	9.2	4.6	18.2
Ex-smoker	15.2	26.3	31.4	43.4	50.7	56.9	31.4
Never smoked	62.2	50.1	50.4	43.2	40.1	38.6	50.4
Females							
Smoker	18.6	16.3	13.6	8.8	5.8	4.5	13.1
Ex-smoker	18.8	26.5	22.4	21.4	25.8	19.0	22.6
Never smoked	62.6	57.2	64.0	69.8	68.3	76.5	64.3
Persons							
Smoker	20.5	20.0	15.9	11.1	7.3	4.5	15.6
Ex-smoker	17.0	26.4	27.0	32.5	37.0	34.6	26.9
Never smoked	62.4	53.7	57.1	56.4	55.6	60.8	57.5

Note - totals for columns may not add to 100% because of rounding.

proportion of males and females who had ever smoked amongst the oldest participants, but this difference was considerably smaller among the younger ages.

More people were ex-smokers than were current smokers for all age and gender groups, except

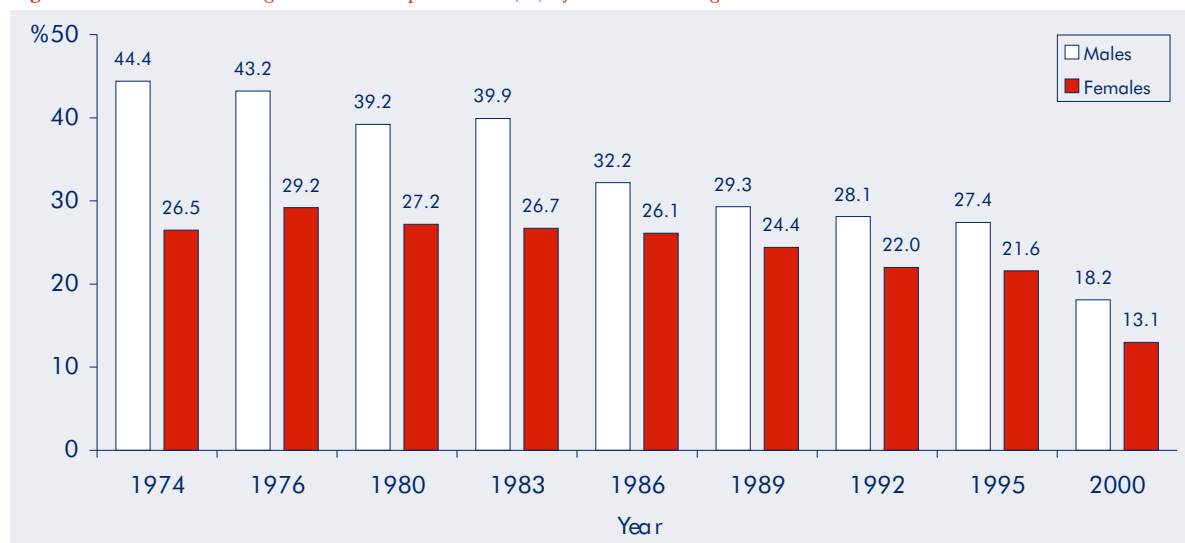
males aged between 25 and 34 years, and the proportion of participants who had ceased smoking increased with age for both genders: from 45% for the youngest males, to 93% for males aged over 75 years, and from 52% for the youngest females, to 82% for those aged over 75 years.

Trends

The Anti-Cancer Council has obtained regular data for the same categories of smoking as were used in this survey, and the 1998 AIHW Report³ noted the declining levels of “current smoking” that had occurred from 1974 to 1995, using data obtained nationwide by the Anti-Cancer Council, and published in 1988³², 1991³³, 1995³⁴ and 1998³⁵.

Figure 6.2 shows current smoking rates for participants in those and the AusDiab surveys, age-standardised to the 1998 population¹⁷ and shows the extent to which smoking prevalence has declined.

Figure 6.2: Trends in the age-standardised prevalence^a (%) of current smoking: 1974 - 2000



^a Age-standardised to the 1998 Australian population.



Discussion

The major finding was the continued decline in the rate of smoking. This has the potential to contribute to a reduction of vascular complications, in both those with low risk levels and those who, because of other risk factors, will derive even greater benefit from such risk reduction.

The less encouraging note is the high rates of having ever smoked for the younger women as compared to the older women. This raises the

possibility of an increase in smoking related diseases for women in the future.

Some degree of caution must be noted in the extent of the apparent decline in smoking prevalence. For both males and females, the admitted smoking prevalence among the participants was 10% less than reported in 1995 by the Anti-Cancer Council³⁵, and it may be that smokers were under represented in the AusDiab sample.

7 Physical activity

Background

Regular physical activity is recognised as being an important component of a healthy lifestyle. It plays a vital role in the prevention and treatment of a number of conditions, including CVD, Type 2 diabetes, some forms of cancer, osteoporosis and mental health conditions. The National Physical

Activity Guidelines for Australians³⁶ recommend that the 'accumulation of 30 minutes of moderate physical activity on most days of the week' is the minimum to obtain health benefits.

Definition

Proposed national standards for the measurement of physical activity developed by the AIHW were used to classify physical activity participation. This classification reflects the amount and type of

physical activity that is 'sufficient' to confer a health benefit. Participants were classified into one of three categories of physical activity, shown in Table 7.1.

Table 7.1: Classification of physical activity

Classification	Physical Activity
Sufficient	At least 150 minutes 'physical activity time' in the previous week.
Insufficient	Less than 150 minutes 'physical activity time' in the previous week.
Sedentary	No participation in physical activity in the previous week.

"Physical activity time" was calculated as the sum of the time spent walking, performing moderate activity plus double the time spent in vigorous activity (to reflect its greater intensity)³⁷.

Results

Physical activity participation status

The study (Figure 7.1) showed that only half (49.8%) of the Australian residents aged 25 years and over were undertaking 'sufficient' physical activity to obtain a health benefit. Participation in this level of activity was more common in males than females. With the exception of the youngest (25-34 years) and oldest (75 years) age-groups, 'sufficient' physical activity consistently represented half of the participants within the respective age groups.

Approximately one-third of the participants (34.5%) were doing some physical activity, but not enough to be categorised as 'sufficient', while another 15.6% reported no participation in physical activity at all in the previous week. Sedentary behaviour and participation in 'insufficient' physical activity were more common in females than males. The combination of sedentary and 'insufficient' physical activity behaviours was lowest in the youngest age group and highest in the oldest age group. Figure

7.1 shows the physical activity status of the participants of this survey.

These results show that just more than half the males, and just less than half the females are partaking of sufficient physical activity to maintain optimal health, and that about one in six Australians is sedentary - not participating in any moderate level physical activity on a weekly basis.

Table 7.2 shows the relationship between age and physical activity patterns according to age for each gender.

The results from the AusDiab participants were similar to those published by Armstrong et al³⁷. The prevalence of sedentary behaviour amongst males peaked in the 55-64 age group, whilst for females, it rose continuously with increasing age.

Figure 7.1: Age-adjusted prevalence (%) of physical activity status among Australian residents

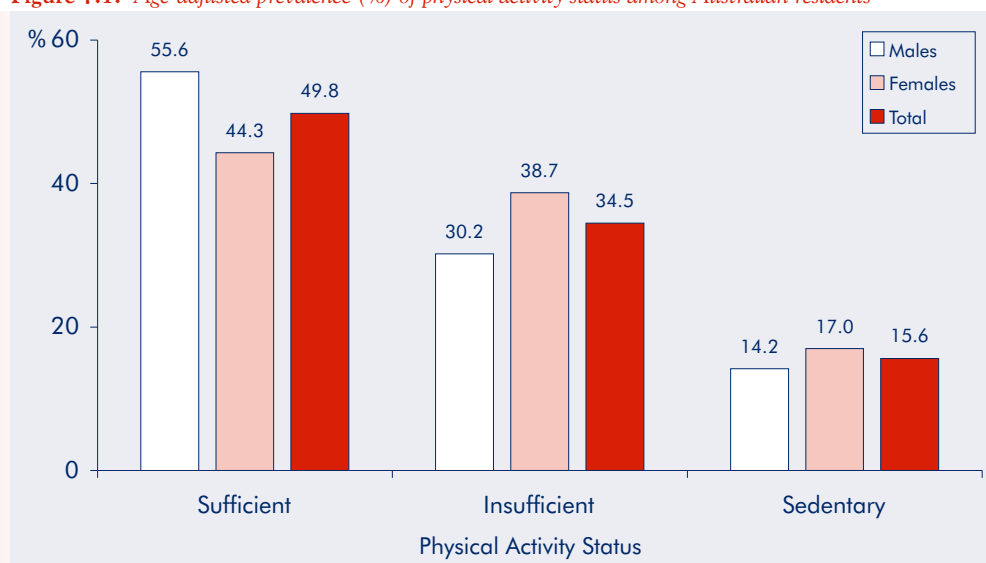


Table 7.2: Age and gender-specific prevalence (%) of physical activity levels

Physical activity status	Age (years)						Total
	25 – 34	35 – 44	45 – 54	55- 64	65 – 74	75+	
Males							
Sufficient	59.3	54.2	53.4	53.9	58.4	53.2	55.6
Insufficient	28.9	32.3	31.4	27.5	27.5	34.0	30.2
Sedentary	11.8	13.6	15.2	18.6	14.1	12.9	14.2
Females							
Sufficient	49.1	44.5	44.0	45.0	39.5	36.4	44.3
Insufficient	38.0	40.3	37.4	37.1	40.8	38.9	38.7
Sedentary	13.0	15.1	18.6	18.0	19.7	24.7	17.0
Persons							
Sufficient	54.2	49.3	48.8	49.5	48.1	43.4	49.8
Insufficient	33.5	36.3	34.4	32.2	34.8	36.8	34.5
Sedentary	12.4	14.4	16.9	18.3	17.1	19.8	15.6

Discussion

The AusDiab findings reveal that half of the Australian residents aged 25 years and over are not sufficiently active for good health as defined by the National Physical Activity Guidelines for Australians³⁶, and concur with a recent report highlighting a decline in recent years in the proportion of the population who undertake 'sufficient' physical activity to achieve a health benefit³⁷.

It is probable that the increase in obesity and many of its related metabolic disorders in Australia over the past few decades is highly associated with a reduction in physical activity during the same period. Dietary surveys show that total caloric consumption has not increased over the same period³⁸. Modernisation, including increased urbanisation, increased technological innovation

and societal changes have contributed to a more sedentary lifestyle at home and at work, while television viewing and other physically inactive pastimes have increasingly dominated leisure-time.

Public health strategies, such as Active Australia, that aim to increase participation in physical activity and to prevent further weight gain in the Australian population continue to emphasise the need to increase physical activity as an integral component of our daily routine. Evidence suggests³⁹ that this may be best achieved by identifying the most important personal, social, and environmental influences to shape integrated approaches from governmental and non-governmental sectors involved in health, sport and recreation, transport and urban design.

8 Cardiovascular disease risk factors

Background

The greater the number of risk factors present, inevitably the greater the likelihood of an adverse outcome. In order to determine the overall impact of cardiovascular risk factors, we have assessed the

frequency with which these risk factors co-exist, and have also calculated the risk of cardiovascular events for each individual.

Definition

A variation of the “Metabolic Syndrome”, as defined by the WHO¹², has been incorporated by including the following criteria, to create what has been described as “The Deadly Quartet”⁴⁰:

1. Glucose intolerance (Diabetes, IGT, or IFG),
2. Dyslipidaemia (triglycerides ≥ 2.0 mmol/l, or HDL cholesterol < 1.0 mmol/l, or treatment for either),

3. Hypertension (SBP ≥ 140, or DBP ≥ 90 mm Hg) or treatment, and
4. Central obesity (waist circumference ≥ 102 cm - males; 88 cm - females).

Results

Table 8.1 shows that over 50% of Australians have at least one of the “Deadly Quartet” and the consequent higher risk of cardiovascular disease.

Prediction of major CVD event

The likelihood within a specific time of the occurrence of a major cardiovascular event, such as a stroke, a myocardial infarction or peripheral vascular disease, has been the subject of research, with formulae based on the Framingham studies used to derive such estimates for individuals⁴⁰. The formula used calculates risk based on age, gender, smoking status, total and HDL cholesterol, diabetes status, systolic blood pressure and the presence or

absence of left ventricular hypertrophy (LVH). For AusDiab, information on LVH is not yet available, and so it has been assumed to be absent in all cases. Furthermore, the risk will be higher than calculated by this method for all people who already have CVD (either symptomatic or asymptomatic). Thus, these figures are likely to represent an underestimate.

Table 8.2 indicates the likelihood over ten years of occurrence of a coronary heart disease (CHD) event (angina, myocardial infarction or CHD death).

Table 8.1: Prevalence (%) of “Deadly Quartet” risk factors

	No. of “Deadly Quartet” risk factors				
	0	1	2	3	4
Males	44.8	26.4	16.9	9.2	2.8
Females	48.1	25.1	14.9	9.3	2.6
Persons	46.5	25.7	15.9	9.3	2.7

Table 8.2: Mean ten-year risk (%) of coronary heart disease for those aged 25-74 years^a

	Age (years)					Total
	25-34	35-44	45-54	55-64	65-74	
Male	1.5	4.9	9.8	15.6	21.4	8.7
Female	0.0	1.3	4.3	8.2	10.8	4.0
Total	0.8	3.1	7.1	11.9	15.6	6.3

^a Based on the Framingham equation⁴¹.



As expected, this table shows the increased risk associated with being male and with increasing age. Based on these estimates, it is likely that over 700,000 Australians, currently aged 25-74 years, will suffer a CHD event over the next 10 years. Taking into account the fact that the risk in the over

75s will be considerably higher, and that stroke and peripheral vascular disease have not been included in these estimates, the total number of people suffering a CVD event will be considerably greater.

9 Indicators of renal disease

Background

Renal disease is a major complication of diabetes and is the second leading cause of death among adults with diabetes¹¹. It is the leading cause of death among the diabetic Indigenous population of Central Australia⁴². The AusDiab study provided the opportunity not only to obtain baseline data for studying the natural history of diabetes but also to undertake a population-based study of indicators of renal disease in the general community of Australia.

The incidence of end-stage renal disease in Australia currently stands at 90/million population

Definitions and methodology

1. Proteinuria - Spot urine protein:creatinine (UPR) ratio 20 (mg/mg) is abnormal.
2. Elevated serum creatinine - serum creatinine > 0.12mmol/L is abnormal.
3. Haematuria - Subjects with haematuria present on dipstick (+) were asked to provide a mid-stream urine for microscopy

Screening for renal disease

Whilst screening has been optimised for the detection of some cancers, such as bowel and breast cancers, little work has been done on the utility of screening for renal disease. Renal disease, when detected early, is often amenable to intervention. Simple and inexpensive tests for the early detection of renal diseases are available. We therefore applied available screening tests to the AusDiab cohort to determine the prevalence of the following indicators of renal disease in Australian adults:

1. Proteinuria (seen in glomerulonephritis, diabetic nephropathy and other renal diseases),

per annum and it is increasing rapidly. Renal disease currently consumes 5.7% of the Australian health care budget. The key causes of renal failure in Australia today are diabetes, glomerulonephritis, inherited kidney diseases and vascular kidney disease relating to atherosclerosis and hypertension. Over the last ten years, diabetes has changed from being the fourth-leading to the second-leading cause of entering end-stage renal disease programs^{11,43}. Screening may offer the best hope of preventing end-stage renal failure.

and repeat dipstick. Samples with 10,000 red cells/ml and/or a repeat positive dipstick of 1 + or greater were considered abnormal.

2. Elevated serum creatinine (an indicator of reduced kidney function), and
3. Haematuria (seen in glomerulonephritis, renal cancers, renal calculi, urinary tract infections).

Diabetes and hypertension are also major risk determinants for renal disease and are addressed elsewhere in this report.



Results and summary

The crude prevalence of each indicator of renal disease in the AusDiab population was as follows:

- proteinuria - 2.5%,
- elevated serum creatinine - 1.1%,
- haematuria - 6.4%.

Thus, indicators of renal disease or conditions leading to renal disease, such as diabetes and hypertension, were present in a large proportion of the AusDiab population. The implications of these findings remain to be determined. This will be the aim of the prospective component of the AusDiab Study.

Published data on the prevalence and implications of indicators of renal disease are scant but have been summarised in the Australian context⁴⁵.

International data allow some predictions to be made. Screening studies in Japan indicate that subjects with proteinuria are 15 times more likely than those without proteinuria to develop renal failure within 10 years⁴⁵. Thus, the finding of significant proteinuria in 2.5% of our population indicates a population clearly at risk for whom intervention should be contemplated. Similarly, the study cited above indicates a relative risk of renal failure of 2.3 for those with haematuria, in addition to their risk of renal cancer, inflammation and calculi. Thus, the finding of haematuria in 6.4% of our population similarly indicates a sub-group of the population at risk of disorders which are potentially life threatening but also amenable to intervention.

The data on renal disease in association with diabetes and hypertension will be part of a future report on the complications of diabetes.

10 Survey methodology

Sample selection

The sample selection was based on a stratified cluster method, with seven strata (6 states and the Northern Territory) used and clusters formed through census collector districts (CDs). For each stratum, six CDs (primary cluster) were randomly selected without replacement and with probability proportional to size (population aged over 25 years). To improve the 'yield' from each primary cluster, we supplemented each (with the exception of the first 3 clusters) with at least one neighbouring CD (defined as one which shares a boundary of

non-zero length with the primary cluster). The number of neighbouring CDs selected was governed by the expected coverage in each area, and following calculations of early response rates after the introduction of household survey activities. The resulting sample was comprised of just the primary cluster in the first three areas and 'clusters' of CD pairs in 22 areas, CD triplets in 16 areas and a CD quadruplet in one area. The localities of the sampled clusters are shown Appendix 2.

Survey protocol and procedures

For logistic and economic purposes, the data collection phase of AusDiab occurred over a 21 month period. Approximately 2 months were allocated to data collection in each state and the Northern Territory.

Preparation of survey tools and training

Experience from previous national and international field surveys of diabetes and other non-communicable diseases was utilized in the preparation of survey tools such as questionnaires, data record forms and survey information documents. The questionnaires were developed in accordance with the objectives of the study, with a specific emphasis placed on having direct comparability with questions used in the 1995 ABS National Health Survey⁴⁶.

Two teams of survey workers were recruited in each state and the Northern Territory to administer the household interview and the physical examination components respectively. Both teams were briefed on the background and methodology of the survey and participated in workshops conducted by the study coordinators to ensure compliance with the interviewing and recording standards and procedures of the survey.

Invitation and publicity

All private dwellings within the sampled cluster were initially approached through a hand-delivered (non-addressed) letter informing them of their selection in the survey and advising them that an AusDiab interviewer would call to arrange a suitable time to conduct the household interview. A brochure providing some background to the survey,

information concerning the interview and examination process, and a guarantee of confidentiality, was included with the initial approach letter.

Local media agencies within and adjacent to the sampled cluster were used to publicise the importance of the study among local residents and to seek their support and co-operation.

Household interview

Details of the number of eligible adults within each household were initially obtained from a single adult in the household. When respondents were unavailable, repeat visits were made to the home. A minimum of two, and up to five calls were made before a household was classified as a non-contact.

Where possible, a personal interview was conducted with each adult aged 25 years and over, who met the eligibility requirements at each participating household. The personal household interview consisted of a short questionnaire that ascertained marital status, level of education, date and country of birth, language spoken at home and history of diabetes or high blood sugar levels.

At the end of the interview respondents were invited to attend a local, temporary test site for the physical examination component. An appointment to attend the test site was arranged by the interviewer and respondents were given verbal and written instructions on the procedures to follow before their physical examination, including instructions to fast for at least 10 hours prior to their test.



Physical examination

The AusDiab physical examination protocol followed closely the WHO recommended model for diabetes and other non-communicable disease field surveys^{13,47,48}. The physical examination was conducted on weekdays (except Friday) and weekend days over an 8 day period in each sampled area. Local survey sites included community halls, scout halls, sporting halls, church halls and schools. Survey activities at the testing site commenced at 7:00 am each morning and typically finished at 2:00 pm. On average, approximately 40 participants attended daily.

All respondents gave informed consent to participate in the survey upon arrival at the testing site. Their particulars were checked against information collected from the household interviewer. Each individual was then assigned a unique nine-digit survey number. Data were entered onto the participant record form at each testing station and checked for completeness prior to their departure. Information recorded on

questionnaires was manually checked for missing values, data-entry errors and consistency. Data anomalies were confirmed with the respective staff member and, where possible, amended through direct verification with the participants on-site or by telephone.

Questionnaires and participant record forms were regularly dispatched to the International Diabetes Institute for electronic scanning using the Teleform Information Capture Software (Cardiff Software Inc., Vista, Ca). To minimise errors in the dataset, all data from the participant record forms were entered both electronically and manually, with inconsistencies between the two methods flagged and verification checks performed by the data manager. The database was subjected to further consistency and verification checks using specially developed data queries. When required, the original document was consulted to overcome inconsistency issues.

Physical examination procedures

Participants moved through the physical examination procedures in a circuit-like manner that took approximately 2.5 to 3 hours to complete. Participants were requested to remain at the testing site until all tests had been completed. Central to the physical examination was the standard 2-hr oral glucose tolerance test, during which period all other procedures were performed.

Blood sampling and OGTT

Blood was collected by venepuncture after an overnight fast of at least 10 hours. Specimens were collected into separate tubes in the following order: a plain tube for measurement of total cholesterol, HDL-Cholesterol, LDL-Cholesterol and triglycerides, fluoride/oxalate tube for plasma glucose; sodium citrate tube for fibrinogen; and an EDTA tube for HbA1c.

All participants, except those with diabetes who were taking medication (including insulin) for diabetes, were then given a 300 ml drink of 75 g of glucose (Biocorp Australia Pty. Ltd., Victoria, Australia) to be consumed within 5 minutes. A second blood sample was taken by venepuncture to determine plasma glucose 2 hours after the glucose load.

Height and weight

Height was measured without shoes using a stadiometer, which had been mounted onto a stable backing board, on a flat surface. Each participant was positioned to be standing fully erect with heels, buttocks and shoulders resting lightly against the board and facing forwards so that the Frankfort plane was in a horizontal position. Readings were taken with the examiner's eyes level with the headpiece. One measurement to the nearest 0.5 cm was recorded. A mechanical beam balance scale was used to measure weight. Participants were instructed to remove shoes, excess clothing (no coats) and items from pockets prior to the measurement. Weight was recorded to the nearest 0.1 kg. Body Mass Index (BMI) was then calculated from the weight and height measurements.

Waist circumference

Waist measurements were taken with a 0.5 cm wide flexible steel measuring tape over the participant's light clothing. Measurements were made halfway between the lower border of the ribs, and the iliac crest on a horizontal plane. Two measurements to the nearest 0.5 cm were recorded and, if the variation between the measurements was greater



than 2 cm, a third measurement was taken. The mean of the two closest measurements was calculated.

Blood pressure

Blood pressure was measured in a seated position after the participant had rested for at least 5 minutes. A cuff of suitable size was applied to the participant's exposed upper arm (the arm not used for blood collection), which was supported by the table at heart level.

In Victoria only, blood pressure was measured using a standard mercury sphygmomanometer. The cuff was inflated to the level upon which the radial pulse disappeared, and then slowly deflated. The pressure at which the first Korotkoff sound was heard was taken as the systolic blood pressure. The diastolic blood pressure was taken as the pressure at which the sounds disappeared (phase V). Two measurements were taken for each participant, with a 30-second interval between them. However, if the difference between the two measurements (either systolic or diastolic) was greater than 10 mm Hg, a third measurement was taken. The mean of the two closest readings was then calculated.

In the remaining states, blood pressure was measured using the Dinamap semi-automatic oscillometric recorder. Once the appropriate cuff size was applied, three readings were taken at one-minute intervals. The first reading was discarded and the average of the final two measurements calculated.

Laboratory procedures

All blood specimens collected in the fluoride/oxalate tubes and the plain tubes were centrifuged on-site to separate out the plasma and

serum respectively. The plasma and serum were then placed in separate tubes, which, in addition to the sodium citrate and EDTA tubes, were transported daily to the central laboratory (HITECH Pathology) in Melbourne. Analyses of plasma glucose, total cholesterol, HDL-cholesterol, triglycerides were performed on the same day.

Plasma glucose levels were measured enzymatically (glucose oxidase) using the Olympus AU600 automated analyser. Serum total cholesterol HDL-cholesterol and triglycerides were also determined enzymatically on the Olympus AU600 analyser. LDL-cholesterol was derived by calculation using the Friedewald formula⁴⁹.

Glycated haemoglobin was measured for all participants with previously diagnosed diabetes and those with plasma glucose levels in the undiagnosed diabetes, impaired glucose tolerance and impaired fasting glucose ranges of the 1999 WHO criteria¹³. Analysis was performed at Monash University with a Bio-Rad Variant Haemoglobin Testing System set up to measure total glycated haemoglobin (Total GHb) in whole blood using boronate affinity high performance liquid chromatography (HPLC). A variant standardization function converted Total GHb values to haemoglobin A1c (HbA1c) values.

Feedback to participants

Participants recorded on their consent form whether they wished their results to be mailed to themselves and/or their doctor. After the laboratory results were available and data had been checked for errors, a letter was posted to participants and their doctors if requested, with attention drawn to those results needing follow-up. Participants were encouraged to discuss their results with their family doctor.

Survey response

In total, 28,033 households were approached throughout the selected clusters. Contact was achieved with 19,214 households. Of the 17,129 eligible households where contact was made, 5,178 (30.0%) refused to participate in the household interview. In the 11,479 households that participated in the household interview, 20,355 people were identified as being eligible to participate in the survey (Table 10.1). Of these, 20,257 people completed the household interview, giving a response rate to the household interview of

99.6%. In total, 11,247 eligible residents participated in the physical examination, yielding a response rate of 55.3%. The response rate for the physical examination ranged from 49.5% in South Australia and Queensland to 61.8% in Western Australia.

Comparison between physical examination respondents and the general population

To assess whether the age and gender profile of the people responding to the physical examination was



Table 10.1: Response rates

State	Eligible residents (n) ^a	Respondents to household interview (n)	Respondents to physical examination (n)	Physical examination response rate (%) ^b
VIC	2,396	2,383	1,434	59.9
WA	2,526	2,475	1,561	61.8
NSW	2,719	2,716	1,515	55.7
TAS	3,339	3,338	1,848	55.4
SA	3,627	3,617	1,796	49.5
NT	2,446	2,442	1,459	59.7
QLD	3,302	3,286	1,634	49.5
Total	20,355	20,257	11,247	55.3

^a Total number of eligible residents of eligible households at which contact was achieved with at least one adult resident.

^b Calculated as percentage of eligible residents who attended for the physical examination.

similar to that of the general population (Table 10.2), a comparison was made to demographic information collected from the 1996 ABS Census (adjusted to give the 1998 resident population estimates¹⁷).

of females, under-represented the young (25-34 years) and over-represented the age groups over 45 years in both males and females. Male respondents to the physical examination were also under-represented in the 35-44 year age group.

Table 10.3 shows that compared to the general population, the sample of respondents to the physical examination had a higher representation

Weighting of the survey sample

Sample weights were applied to those participating in the physical examination to account for the

Table 10.2: Estimated national population (thousands) aged over 25 years, June 30, 1998¹⁷

Gender	Age (years)						Total
	25 – 34	35 – 44	45 – 54	55 – 64	65 – 74	75 +	
Males	1,440.5	1,443.8	1,246.1	815.0	620.3	378.9	5,944.6
Females	1,440.1	1,451.5	1,219.8	801.3	678.6	603.9	6,195.3
Total	2,880.5	2,895.3	2,465.9	1,616.3	1,298.9	982.8	12,139.9

Table 10.3: Comparison of the age and gender distribution between the estimated 1998 Australian population^a and respondents to the physical examination (unweighted and weighted)

Classification	1998 population estimates ^a	Respondents to physical examination	
		unweighted	weighted
Males	49.0	44.9	48.9 (47.9 - 50.0)
Females	51.0	55.1	51.1 (50.0 - 52.1)
Males 25-34	11.9	5.2	11.7 (9.6 - 13.8)
Males 35-44	11.9	9.7	12.0 (10.9 - 13.1)
Males 45-54	10.3	12.0	10.1 (8.6 - 11.7)
Males 55-64	6.7	8.3	6.8 (5.7 - 7.9)
Males 65-74	5.1	6.5	5.5 (4.6 - 6.5)
Males 75+	3.1	3.2	2.7 (2.0 - 3.3)
Females 25-34	11.9	7.1	11.9 (9.8 - 14.0)
Females 35-44	12.0	13.0	12.0 (10.6 - 13.5)
Females 45-54	10.0	13.7	10.0 (8.4 - 11.5)
Females 55-64	6.6	9.7	6.7 (5.7 - 7.6)
Females 65-74	5.6	7.4	6.7 (5.1 - 8.4)
Females 75+	5.0	7.2	3.8 (2.4 - 5.1)

^a June 30, 1998 Australian population¹⁷.

Note - data are percentages (95% confidence intervals).



clustered stratified sample design. The weights were first adjusted to ensure the weighted sample matched the age and gender distribution within each CD as obtained from the 1996 Census. These weights were further adjusted so that the age and gender distribution within each state matched the estimated 1998 residential population.

Weighting of the data rectified the differences in age and gender distribution to yield a physical examination respondent population that was similar to the general population estimate (Table 10.3).

Statistical analysis

Descriptive statistics were generated using SPSS version 10.0.5 for Windows (SPSS Inc., 1999, Chicago, IL). Calculation of estimates and confidence intervals were performed using Stata Statistical Software to account for the stratification and clustering of the survey design: Release 6.0 (StataCorp., 1999 College Station, TX).

Confidence intervals are expressed as indicating the 95% confidence estimate of the range of the described parameter.



Appendix 1

Summary table with confidence intervals

Classification	Male		Female		Persons	
Diabetes mellitus	8.0	(6.1 – 9.9)	7.0	(5.1 – 9.0)	7.5	(5.7 – 9.3)
IFG	8.1	(7.1 – 9.1)	3.3	(2.4 – 4.3)	5.7	(4.81 – 6.6)
IGT	9.2	(7.7 – 10.6)	12.0	(10.9 – 13.0)	10.6	(9.6 – 11.5)
Overweight						
BMI	67.4	(64.3 – 70.4)	52.0	(46.7 – 57.3)	59.6	(56.7 – 62.5)
WC	55.1	(50.4 – 59.7)	56.5	(49.8 – 63.2)	55.8	(50.8 – 60.9)
Obese						
BMI	19.1	(16.6 – 21.6)	21.8	(18.7 – 24.9)	20.5	(18.2 – 22.8)
WC	26.6	(23.4 – 29.7)	33.8	(28.1 – 39.7)	30.3	(26.1 – 34.5)
Total cholesterol	51.1	(48.7 – 53.5)	51.2	(48.4 – 54.0)	51.2	(48.9 – 53.4)
Triglycerides	24.5	(21.3 – 27.7)	16.6	(14.1 – 19.0)	20.5	(18.0 – 22.9)
LDL - cholesterol	49.8	(46.6 – 52.9)	42.1	(39.8 – 44.3)	45.8	(43.7 – 47.9)
HDL - cholesterol	18.6	(16.0 – 21.2)	5.4	(3.6 – 7.3)	11.9	(9.8 – 14.0)
Lipid - medication	7.3	(5.9 – 8.7)	7.4	(5.6 – 9.1)	7.3	(5.9 – 8.7)
Hypertension	30.6	(27.1 – 34.1)	27.1	(22.8 – 31.4)	28.8	(25.3 – 32.4)
Hypertension - medication	11.5	(10.0 – 12.9)	15.3	(12.3 – 18.2)	13.4	(11.4 – 15.4)
Current smoker	18.2	(14.6 – 21.8)	13.1	(10.2 – 15.9)	15.6	(12.6 – 18.5)
Ex-smoker	31.4	(29.4 – 33.4)	22.6	(20.7 – 24.4)	26.9	(25.3 – 28.6)
Never smoked	50.4	(46.1 – 56.7)	64.3	(60.6 – 68.1)	57.5	(53.8 – 61.2)
Sufficient physical activity	55.6	(52.5 – 58.7)	44.3	(40.7 – 47.8)	49.8	(46.9 – 52.8)
Insufficient physical activity	30.2	(28.0 – 32.3)	38.7	(36.8 – 40.6)	34.5	(32.8 – 36.3)
Sedentary	14.2	(12.4 – 16.0)	17.0	(14.5 – 19.5)	15.6	(13.9 – 17.3)

Data are percentages (95% confidence intervals).
Definitions will be found in the text.

Appendix 2

Survey sites		
Victoria	Parkdale	Blackburn North
	Burwood East	Wattle Glen
	Bendigo	Mildura
Western Australia	Trigg	Scarborough
	Kardinya	High Wycombe
	Mt Helena	Oakford
New South Wales	West Pennant Hills	Hurstville
	Auburn	Grays Point
	Orange	Berkeley Vale
Tasmania	Alanvale	Ravenswood
	Georgetown	Ulverstone
	Taroona	Blackmans Bay
South Australia	Hyde Park	Netley
	Glenelg	Port Lincoln
	Millicent	Parafield Gardens
Northern Territory	Driver	Marrara
	Nightcliff	Wagaman
	Larrakeyah	Parap
Queensland	Cairns	Chapel Hill
	Nambour	East Toowoomba
	Stafford Heights	Currumbin



References

1. McCarty DJ, Zimmet P, Dalton A, Segal L, Welborn TA. The rise and rise of diabetes in Australia, 1996: A review of statistics, trends and costs. Canberra: International Diabetes Institute & Diabetes Australia, 1996.
2. Guest C, O'Dea K, Hopper J, Nankervis A, Larkins R. The prevalence of glucose intolerance in Aborigines and Europeans of south-eastern Australia. *Diab Res Clin Pract* 1992;15:227-235.
3. Australian Institute of Health and Welfare. Australia's Health 1998: the sixth biennial health report of the Australian Institute of Health and Welfare. Canberra: AIHW, 1998.
4. Glatthaar C, Welborn TA, Stenhouse NS, Garcia-Webb P. Diabetes and impaired glucose tolerance. A prevalence estimate based on the Busselton 1981 survey. *Medical Journal of Australia* 1985;143:436-440.
5. National Heart Foundation of Australia. Risk Factor Prevalence Study No. 2 - 1983. Canberra: National Heart Foundation, 1983.
6. Welborn T, Glatthaar C, Whittall D, Bennett S. An estimate of diabetes prevalence from a national population sample: a male excess. *Medical Journal of Australia* 1989;150:78-81.
7. Australian Bureau of Statistics. 1989-90 National Health Survey: summary of results, Australia. Canberra: Australian Bureau of Statistics, 1991.
8. Australian Bureau of Statistics. 1995 National Health Survey: summary of results, Australia. Canberra: Australian Bureau of Statistics, 1997.
9. Australian Bureau of Statistics. Causes of Death, Australia, 1996. Canberra: Australian Bureau of Statistics, 1997.
10. Colagiuri S, Colagiuri R, Ward J. National Diabetes Strategy and Implementation Plan. Canberra: Diabetes Australia, 1998.
11. Commonwealth Department of Health and Care & Australian Institute of Health and Welfare. National Health Priority Areas Report: Diabetes Mellitus 1998. Canberra: HEALTH and AIHW, 1999.
12. Australian Bureau of Statistics. 1995 National Health Survey: Diabetes. Canberra: ABS, 1997.
13. 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.
14. National Heart Foundation of Australia. Risk Factor Prevalence Study No. 1 - 1980. Canberra: National Heart Foundation, 1980.
15. National Heart Foundation and Australian Institute of Health. Risk Factor Prevalence Study No. 3 1989. Canberra: National Heart Foundation & Australian Institute of Health, 1990.
16. Taylor R, Zimmet P. Limitation of fasting plasma glucose for the diagnosis of diabetes mellitus. *Diabetes Care* 1981;4:556-558.
17. Australian Bureau of Statistics. Population by Age and Sex, Australian States and Territories. Canberra: Australian Bureau of Statistics, 1999.
18. Australian Bureau of Statistics. Australian Demographic Statistics, December Quarter 1999. Canberra: Australian Bureau of Statistics, 2000.
19. DCCT Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *The New England Journal of Medicine* 1993;329(14):977-986.
20. United Kingdom Prospective Diabetes Study (UKPDS) Group. Effect of intensive blood-glucose control with metformin on complications in overweight patients with Type 2 diabetes: UKPDS 34. *Lancet* 1998;352:854-865.
21. Alberti K. The clinical implications of impaired glucose tolerance. *Diabetic Medicine* 1996;13:927-937.
22. Hartz A, Rupley D, Kalkhoff R, Rimm A. Relationship of obesity to diabetes: influence of obesity level and body fat distribution. *Preventive Med* 1983;12:351-357.
23. Dowse G, Zimmet P, Gareeboo H, et al. Abdominal obesity and physical inactivity as risk factors for NIDDM and impaired glucose tolerance in Indian, Creole and Chinese Mauritians. *Diabetes Care* 1991;14:271-282.
24. World Health Organization. Obesity: Preventing and Managing the Global Epidemic: Report of a WHO Expert Committee. Geneva: WHO, 1998.

25. Australian Bureau of Statistics, Department of Health and Family Services. National Nutrition Survey: selected highlights 1995. Canberra: Australian Bureau of Statistics & Department of Health & Family Services, 1997.
26. Australian Bureau of Statistics. Estimated resident population by sex and age: States and Territories of Australia, June 1987 to June 1992. Canberra: Australian Bureau of Statistics, 1993.
27. Australian Diabetes Society. Diabetic dyslipidaemia - Australian Diabetes Society position statement. *Medical Journal of Australia* 1995;162:91-93.
28. Bennett SA, Magnus P. Trends in cardiovascular risk factors in Australia: results from the National Heart Foundation's Risk Factor Prevalence Study, 1980 - 1989. *Medical Journal of Australia* 1994;161:519-527.
29. United Kingdom Prospective Study (UKPDS) Group. Tight blood pressure control and risk of macrovascular and microvascular complications in Type 2 diabetes: UKPDS 38. *British Medical Journal* 1998;317:703-713.
30. United Kingdom Prospective Diabetes Study (UKPDS) Group. Efficacy of atenolol and captopril in reducing risk of macrovascular and microvascular complications in Type 2 diabetes: UKPDS 39. *British Medical Journal* 1998;317:713-720.
31. Guidelines Subcommittee. 1999 World Health Organization - International Society of Hypertension Guidelines for the Management of Hypertension. *Journal of Hypertension* 1999;17:151-183.
32. Hill DJ, White VM, Gray NJ. Measures of tobacco smoking in Australia 1974-1986 by means of a standard method. *Medical Journal of Australia* 1988;149:10-12.
33. Hill DJ, White VM, Gray NJ. Australian patterns of tobacco smoking in 1989. *Medical Journal of Australia* 1991;154:797-801.
34. Hill DJ, White VM. Australian adult smoking prevalence in 1992. *Australian Journal of Public Health* 1995;19:305-08.
35. Hill DJ, White VM, Scollo MM. Smoking behaviours of Australian adults in 1995: trends and concerns. *Medical Journal of Australia* 1998;168:209-213.
36. Commonwealth Department of Health and Aged Care. National physical activity guidelines for Australians. Canberra: Department of Health & Aged Care, 1999.
37. Armstrong T, Bauman A, Davies J. Physical activity patterns of Australian adults. Results of the 1999 National Physical Activity Survey. Canberra: Australian Institute of Health and Welfare, 2000.
38. Australian Institute of Health and Welfare. Heart, stroke and vascular diseases - Australian facts 2001: Australian Institute of Health & Welfare, National Heart Foundation, & National Stroke Foundation of Australia, 2001 (in press).
39. Sallis J, Owen N. *Physical Activity and Behavioural Medicine*. Thousand Oaks, CA: Sage Publications, 1999.
40. Kaplan N. The deadly quartet: Upper-body obesity, glucose intolerance, hypertriglyceridaemia, and hypertension. *Archives of Internal Medicine* 1989;149:1514-1520.
41. Anderson K, Odell P, Wilson P, Kannel W. Cardiovascular disease risk profiles. *American Heart Journal* 1991;121:293-298.
42. Phillips CB, Patel MS, Weeramanthri TS. High mortality from renal disease and infection in Aboriginal central Australians with diabetes. *Australian Journal of Public Health* 1995;19:482-486.
43. Disney APS (ed). ANZDATA Registry Report 2000. Adelaide: Australian and New Zealand Transplant Registry, 2000.
44. Briganti E, McNeil J, Atkins R. The epidemiology of diseases of the kidney and urinary tract: an Australian perspective: The Australian Kidney Foundation: <http://www.kidney.org.au/survey/front.htm>, 1999.
45. Iseki K, Iseki C, Ikemiya Y, Fukiyama K. Risk of developing end-stage renal disease in a cohort of mass screening. *Kidney International* 1996;49:800-805.
46. Australian Bureau of Statistics. National Health Survey: Users' Guide, Australia 1995. Canberra: Australian Bureau Of Statistics, 1996.
47. World Health Organization. *Cardiovascular Survey Methods*, 2nd Edition. Geneva: WHO, 1982.
48. Dowse G, Zimmet P. A model protocol for a diabetes and other noncommunicable disease field survey. *World Health Statistics Quarterly* 1992;45:360-372.
49. Friedewald W, Levy R, Fredrickson D. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without the use of preparative ultracentrifuge. *Clinical Chemistry* 1972;18:499-502.



Steering committee

The Australian Diabetes, Obesity and Lifestyle Study

Steering committee

Dr BOB ATKINS
Dept. of Nephrology
Monash Medical Centre (VIC)

Dr STAN BENNETT
Australian Institute of Health & Welfare
(ACT)

Dr STEVEN CHADBAN
Department of Nephrology
Monash Medical Centre (VIC)

Prof. STEPHEN COLAGIURI
Centre for Diabetes Strategies
The Prince of Wales Hospital (NSW)

Dr MAX DE COURTEN
International Diabetes Institute (VIC)

Dr MICHAEL D'EMBDEN
Royal Brisbane Hospital (QLD)

Dr DAVID DUNSTAN
International Diabetes Institute (VIC)

Prof. TERRY DWYER
Menzies Centre for Population
Research (TAS)

Dr DAMIEN JOLLEY
Deakin University (VIC)

Dr PAUL MAGNUS
Australian Institute of Health & Welfare
(ACT)

Prof. JOHN MATHEWS
National Centre for Disease Control
(ACT)

Dr DAN McCARTY
Centre for Eye Research Australia (VIC)

Prof. KERIN O'DEA
Menzies School of Health Research (NT)

Dr PAT PHILLIPS
Queen Elizabeth Hospital (SA)

Dr PHIL POPPLEWELL
Flinders Medical Centre (SA)

MR IAN KEMP
Commonwealth Department of Health
& Aged Care (ACT)

Prof. HUGH TAYLOR
Centre for Eye Research Australia (VIC)

Prof. TIM WELBORN
Sir Charles Gairdner Hospital (WA)

Prof. PAUL ZIMMET AM
International Diabetes Institute (VIC)

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Principal investigators

Professor Paul Zimmet AM

Professor Timothy Welborn

Co-ordinating centers

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AusDiab staff

Mr Adrian Cameron (International Diabetes Institute)

Ms Marita Dalton (International Diabetes Institute)

Dr Max de Courten (International Diabetes Institute)

Dr David Dunstan (International Diabetes Institute)

Mr Adam Meehan (International Diabetes Institute)

Mrs Shirley Murray (International Diabetes Institute)

Ms Alison Stewart (International Diabetes Institute)

Ms Robyn Tapp (International Diabetes Institute)

Ms Fay Wilson (International Diabetes Institute)

Ms Annie Allman (HITECH Pathology)

Ms Clare Reid (HITECH Pathology)

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