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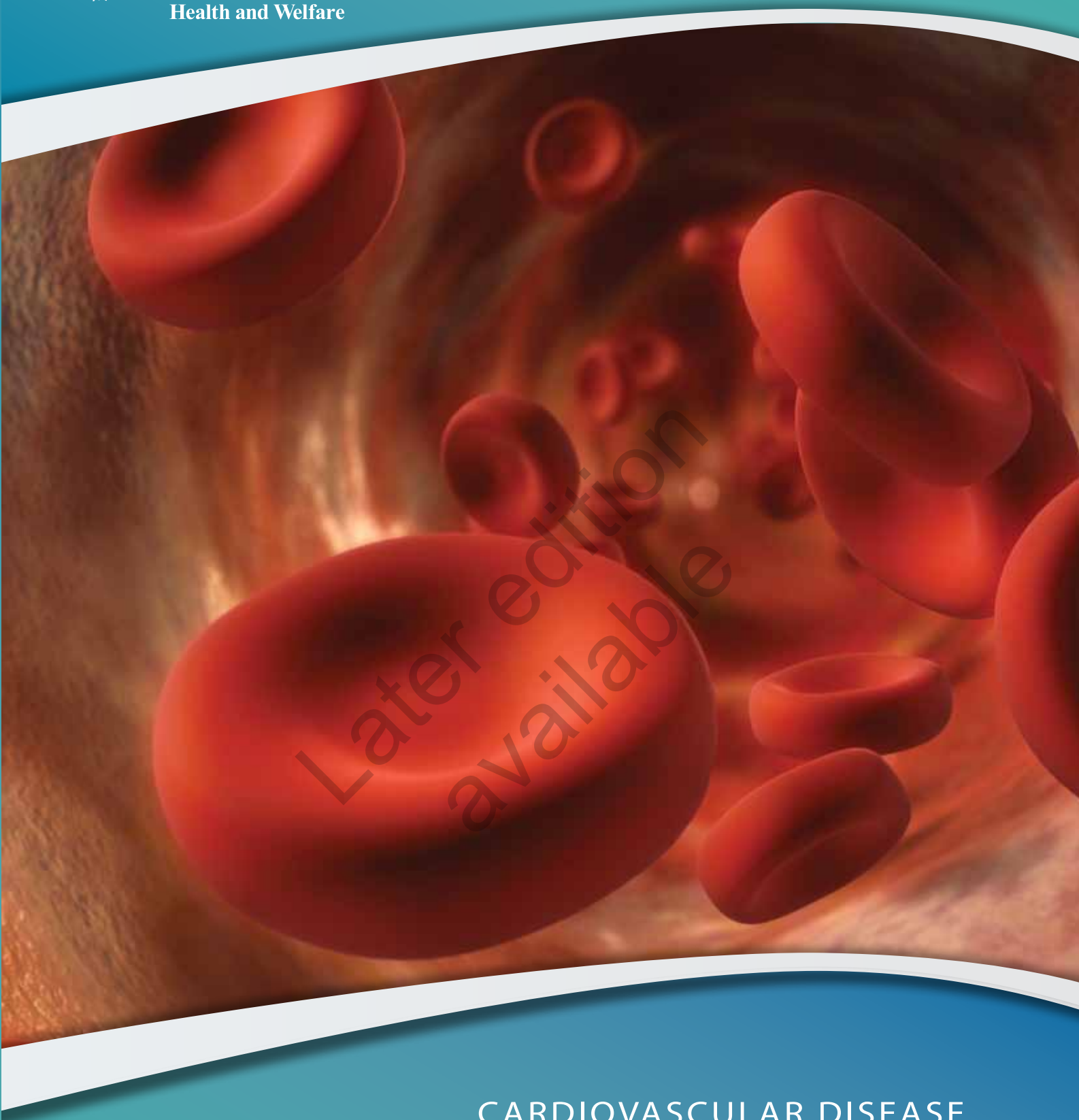

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CARDIOVASCULAR DISEASE
Australian**facts** 2011

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Abbreviations

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ABS	Australian Bureau of Statistics
ACHI	Australian Classification of Health Interventions
AIHW	Australian Institute of Health and Welfare
ALOS	average length of stay
ARF	acute rheumatic fever
ATC	Anatomic Therapeutic Chemical
AusDiab	Australian Diabetes, Obesity and Lifestyle Study
BEACH	Bettering the Evaluation and Care of Health
BMI	body mass index
CABG	coronary artery bypass grafting
CHD	coronary heart disease
CKD	chronic kidney disease
CVD	cardiovascular disease
DALY	disability-adjusted life year
DDD	defined daily dose
DoHA	Australian Government Department of Health and Ageing
ESKD	end-stage kidney disease
ERP	estimated resident population
HDL	high-density lipoprotein
ICD	International Classification of Diseases
ICD-9	International Classification of Diseases, 9th revision
ICD-10	International Classification of Diseases, 10th revision
ICD-10-AM	International Statistical Classification of Diseases and Related Health Problems, 10th revision, Australian Modification
IRSD	Index of Relative Socioeconomic Disadvantage

NATSIHS	National Aboriginal and Torres Strait Islander Health Survey
NATSISS	National Aboriginal and Torres Strait Islander Social Survey
NDSHS	National Drug Strategy Household Survey
NEMESIS	North East Melbourne Stroke Incidence Study
NHFA	National Heart Foundation Australia
NHMRC	National Health and Medical Research Council
NHS	National Health Survey
NNS	National Nutrition Survey
NSF	National Stroke Foundation
NSMHWB	National Survey of Mental Health and Wellbeing
NSW	New South Wales
NT	Northern Territory
OECD	Organisation for Economic Co-operation and Development
Qld	Queensland
PBS	Pharmaceutical Benefits Scheme
PCSS	Perth Community Stroke Study
PYLL	potential years of life lost
RHD	rheumatic heart disease
RPBS	Repatriation Pharmaceutical Benefits Scheme
PVD	peripheral vascular disease
SA	South Australia
SEIFA	Socioeconomic Indexes for Areas
SDAC	Survey of Disability, Ageing and Carers
TIA	transient ischaemic attack
Vic	Victoria
WA	Western Australia
WHO	World Health Organization
YLD	years lost due to disability
YLL	years of life lost (due to premature mortality)

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Symbols

—	nil or rounded to zero
%	per cent
g	gram
cm	centimetre
kg	kilogram
'000	thousands
m	million
mmHg	millimetres of mercury
mmol/L	millimoles per litre
. .	not applicable
<	less than
$	Australian dollars, unless otherwise specified

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Summary and key findings

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In many ways cardiovascular disease (CVD) can be considered Australia's most costly disease. It costs more lives than any other disease and has the greatest level of health expenditure. It also imposes a burden of disease, measured in terms of disability and premature death, second only to cancer.

However as this report illustrates, there are many areas where progress is being made in reducing the impact of CVD in the community but areas remain where there is still room for further improvement.

Cardiovascular disease: Australian facts 2011 is the fourth in a series of national reports by the National Centre for Monitoring Cardiovascular Disease, providing an overview of cardiovascular disease in Australia. It aims to present information and statistics about the number of people with CVD, trends for hospitalisations and deaths, key risk factors and treatment and care.

Key findings

The impact of cardiovascular disease

- In 2007–08, about 3.5 million Australians had a long-term cardiovascular disease.
- Nearly 50,000 deaths were attributed to CVD in Australia in 2008. It was responsible for more deaths than any other disease group—34% of the total.
- CVD was the main cause for 475,000 hospitalisations in 2007–08 and played a secondary role in a further 797,000.
- CVD accounted for about 18% of the overall burden of disease in Australia in 2003, with coronary heart disease and stroke contributing over 80% of this burden.
- CVD remains the most expensive disease group in Australia, costing about $5.9 billion in 2004–05 with just over half of this money spent on patients admitted to hospital.

Trends

- The overall death rate for CVD has fallen by about 80% since the 1960s and continues to fall.
- Death rates for the major types of CVD, such as coronary heart disease, stroke, heart failure, rheumatic heart disease and peripheral vascular disease, have all fallen markedly in the past 20 years.

- There appears to be some recent slowing of the decline in the coronary heart disease death rates in younger age groups.
- CVD hospitalisation rates have declined slowly over the past decade.

Who does it affect most?

- On the whole lower socioeconomic groups, Aboriginal and Torres Strait Islander people and those living in the remote areas of Australia had the highest rates of hospitalisation and death resulting from CVD.
- CVD has its greatest impact on the elderly where hospitalisation and death rates are usually much higher than for others. The main exception to this is congenital heart disease where the impact is greatest on infants.
- For most cardiovascular conditions male death rates are clearly higher than female rates—in some cases twice as high.
- CVD is the cause of more female deaths than male deaths. This is because females usually live longer than males and the risk of a cardiovascular condition increases rapidly with age, particularly among the elderly.

Risk factors for cardiovascular disease—how much can we reduce them?

- The main risk factor for CVD is age.
- Many risk factors for CVD, such as sex, ethnicity and a family history of the disease, cannot be changed.
- Risk factors such as smoking, lack of exercise, being overweight, excessive alcohol use and a poor diet can all be changed and improving them can greatly reduce the impact of CVD.

Where are risk factors headed?

- Smoking rates in Australia have fallen by about 32% over the past two decades with about 17% of Australians now daily smokers. However, smoking remains the single most important cause of ill health and death in Australia.
- The prevalence of obesity rose by 6 percentage points between 1995 and 2007, with about 60% of adult Australians now being overweight or obese and about 70% not getting enough exercise.
- Alcohol use has remained stable in the past decade but around 10% of males and females over 14 years of age drink at risky levels.

What is happening with treatment?

- CVD patients are staying in hospital for shorter periods and the rate of deaths in hospital is declining.
- The rates for most procedures to diagnose and treat people with CVD have increased in the past decade.
- Prescription rates for medicines to control high blood pressure and blood cholesterol levels have also increased substantially in the past decade.

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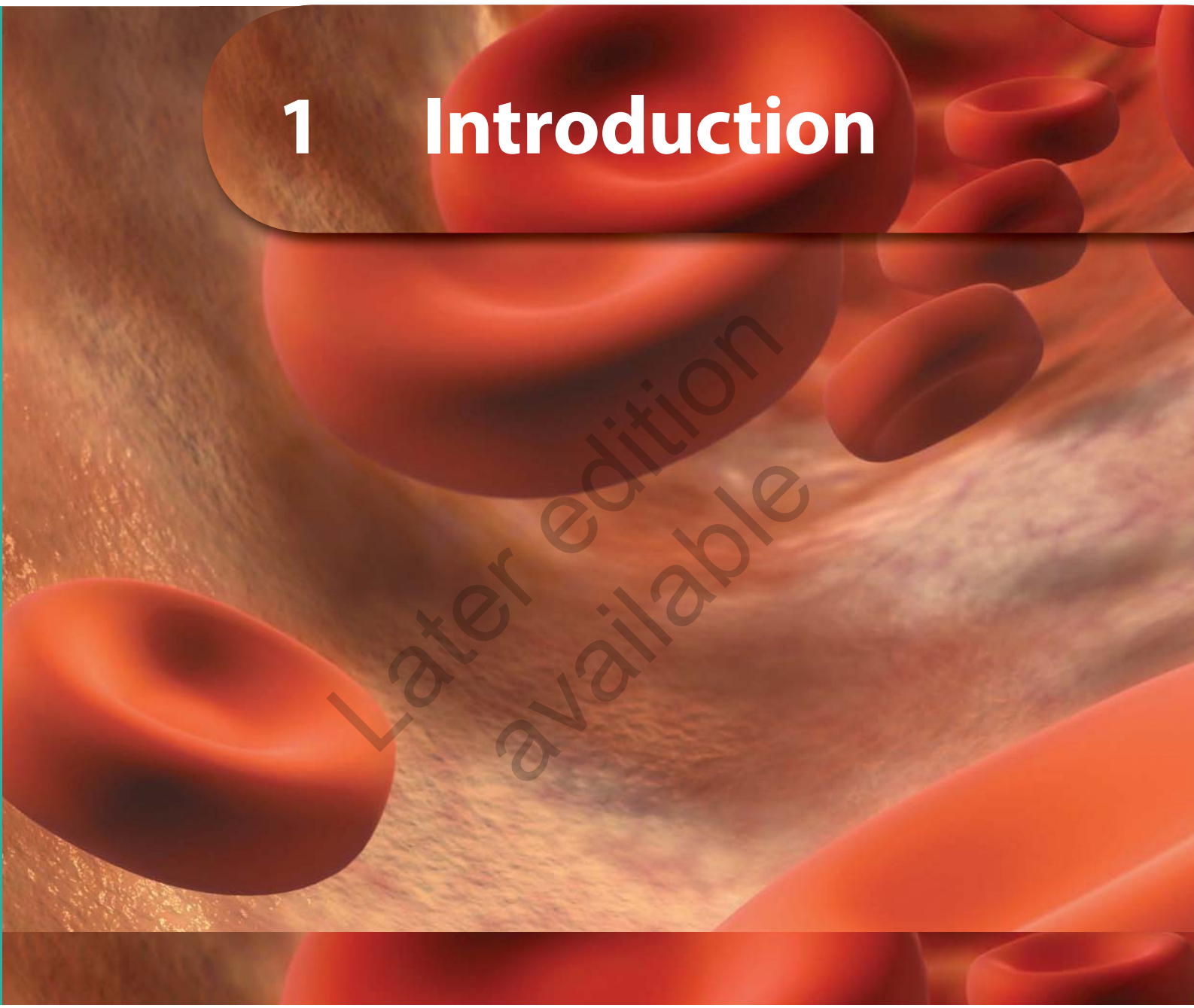

1 Introduction

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1 Introduction

Background

Significant progress has been made in recent years in improving the cardiovascular health of Australians. Death rates have fallen markedly, levels of some risk factors have improved and there have been major advances in treatment. Nevertheless, cardiovascular disease (CVD) continues to impose a heavy burden on Australians in terms of illness, disability and premature death, and the associated direct health care expenditure exceeds that for any other disease group. Also, the prevalence of CVD is expected to increase over the coming decades as the number of elderly Australians, among whom it is the most common, continues to grow.

Some groups have much higher rates of illness and death from CVD than others, particularly Aboriginal and Torres Strait Islander people, those from the most socioeconomically disadvantaged groups and those living in remote areas of Australia.

A large part of the premature death, disability and illness that CVD causes is preventable. Many Australians remain at higher risk of CVD because of behavioural risk factors that can be modified: tobacco smoking, insufficient exercise, being overweight, a diet high in saturated fats and a high alcohol intake level. Largely as a result of these risk factors many Australians also have the major biological risk factors of high blood pressure and blood cholesterol levels.

Psychosocial factors, such as depression and social isolation can also affect the development of CVD. Wider circumstances influence many risk factors and the importance of people's economic resources, education, living and working conditions, social support and access to health care and social services, is widely recognised.

The information presented in this report depends on many statistics which are derived from a large pool of data (see Box 1.1) compiled by many people throughout Australia and it's extended health system.

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Box 1.1: Why some statistics appear old

Although this report is published in 2011, the statistics refer to 2008 or earlier. There are a number of reasons for this. First, some data, such as population-based surveys, are collected every 3 or 5 years, or even less often. Second, whether data are collected recently or not, it can often take a year or more before they are fully processed and released to the AIHW. Finally, the AIHW in turn often needs some months to ensure the quality and accuracy of statistics and their analysis before they are released.

Purpose of the report

Against the background discussed above, this report aims to provide policy makers, health professionals and the community with a comprehensive summary of the latest available data describing CVD in Australia.

What are cardiovascular diseases?

The term cardiovascular disease covers all diseases and conditions of the heart and blood vessels. The main types of CVD in Australia are coronary heart disease, stroke and heart failure/cardiomyopathy. These conditions are described separately in later chapters. In developed countries such as Australia, the main underlying cause of CVD is a process known as *atherosclerosis*. This is a condition where abnormal deposits of fat, cholesterol and other substances build up in the inner lining of the arteries to form *plaque*. Atherosclerosis is most serious when it leads to reduced or blocked blood supply to the heart (causing angina or heart attack) or to the brain (causing a stroke). The process leading to atherosclerosis is slow and complex, often starting in childhood and progressing with age.

Structure of the report

The report covers three main areas: risk factors for CVD, major cardiovascular diseases and CVD health services—including a chapter on health expenditure.

The report comprises 12 chapters in total, including separate chapters for those cardiovascular diseases which have the greatest impact on the Australian population. Using the most recent national data, trends, prevalence, hospitalisation and mortality are described for each disease with additional analysis by Indigenous status, remoteness area and socioeconomic group. For each disease, tables are included in Appendix D to allow a quick comparison of hospitalisation and death rates by age and population subgroup.

The largest chapter in the report, Chapter 2, examines the major risk factors for CVD, many of which are common, not only to most CVD, but to other diseases as well. The latest risk factor information is presented for prevalence, trends, and age and sex breakdowns. Population subgroups are included where possible.

After the disease chapters, 'Chapter 11 Health services for cardiovascular disease' presents information on hospital procedures, medicines and rehabilitation and 'Chapter 12 Expenditure on cardiovascular disease' examines expenditure used to address CVD in Australia: the amounts spent, where they are spent and how expenditure has changed over time.

International comparisons are also made where possible and the methods, definitions, classifications and data sources used are included at the end of the report.

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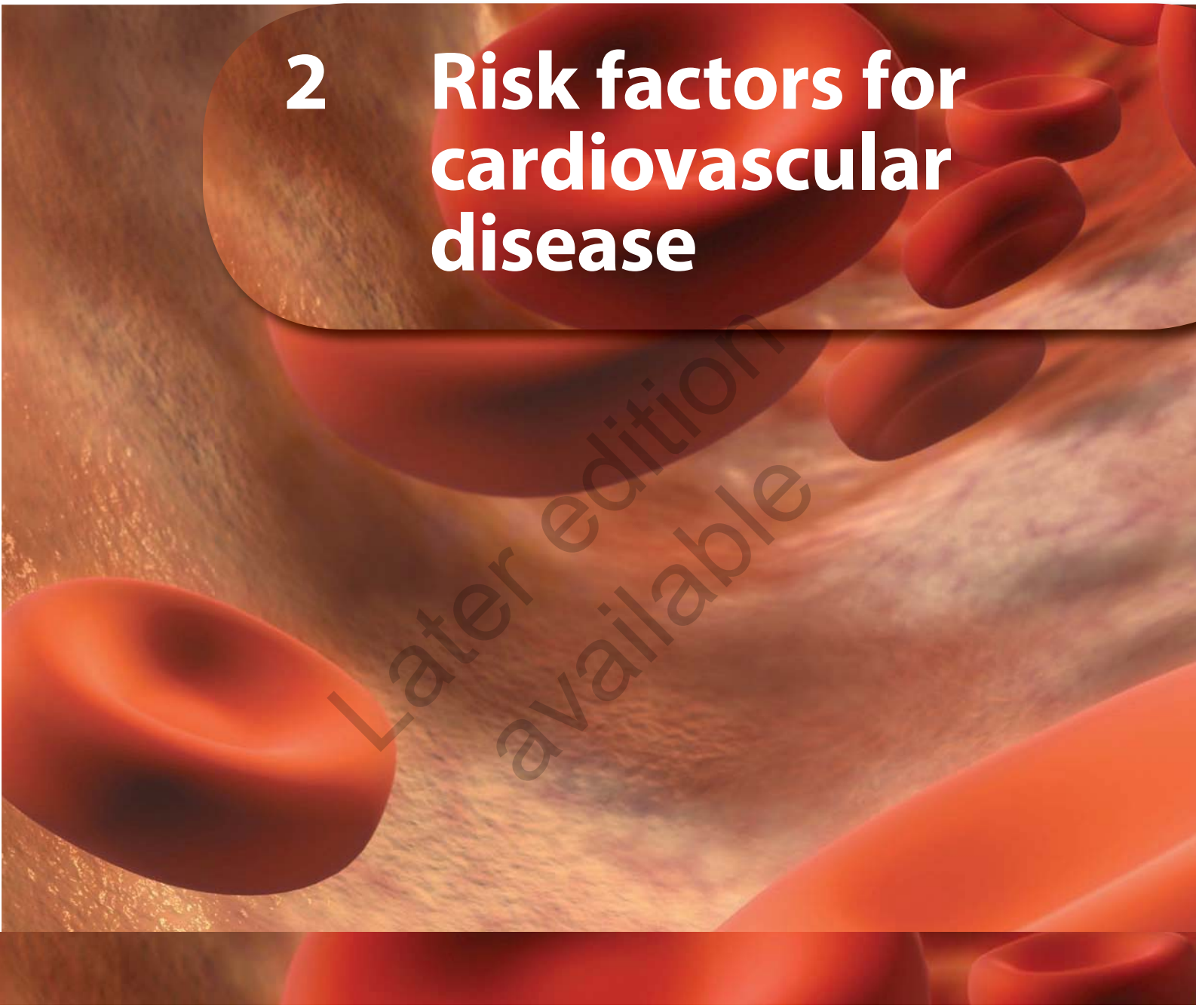
New in the 2011 edition

Earlier editions of this report were published in 1999, 2001 and 2004 (AIHW 2001, 2004; AIHW & Heart Foundation of Australia 1999).

New in the 2011 edition is a chapter examining the comorbidity of CVD, diabetes and chronic kidney disease (CKD). Hospitalisation data are used to highlight the fact that often CVD is diagnosed in conjunction with other diseases and complex relationships exist, not only between CVD and conditions such as diabetes and CKD, but between CVDs themselves.



2 Risk factors for cardiovascular disease

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2 Risk factors for cardiovascular disease

What is a risk factor?

A risk factor is any factor which increases the likelihood of a person developing a health disorder or health condition. Along with their opposites, protective factors, risk factors are known as *determinants* of disease or illness (AIHW 2008b).

There are different types of risk factors, some of which can be altered (modifiable risk factors) and some that cannot (non-modifiable risk factors).

Non-modifiable risk factors such as age, sex, family history and ethnicity, can affect the incidence of cardiovascular disease (CVD). The World Health Organization (WHO) recognises ageing as the most powerful risk factor for CVD with the risk of stroke doubling every decade after the age 55 years. Other non-modifiable risk factors are also important. For instance, males have higher rates of coronary heart disease (CHD) than women; CVD risk increases if a first-degree blood relative has had CHD or stroke before age 55 years (for a male relative) or 65 years (for a female relative); and some ethnic groups show higher rates of CVD than others (Mackay & Mensah 2004).

Modifiable risk factors can have a marked effect on the prevalence of CVD in the community. *The burden of disease and injury in Australia 2003* (Begg et al. 2007) quantified 12 risk factors associated with CVD, which together explained 69% of the burden from this group of causes. High blood pressure and high cholesterol were the largest contributors followed by physical inactivity, high body mass, tobacco use and low fruit and vegetable consumption.

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Multiple risk factors

Individuals with CVD often have multiple risk factors, resulting in an increased risk of illness, a lower life expectancy and greater health care costs than those with only one risk factor (Daviglus et al. 1998). The risk of death increases as the number of risk factors (defined as high blood glucose, high blood pressure, low high density lipoprotein (HDL) cholesterol, and high triglycerides) increase (Trevisan et al. 1998).

Many risk factors interact with each other in complex ways. The development of one risk factor may lead to another occurring, or occurring in a more severe form. For example, physical inactivity can lead to an individual being overweight or obese, a condition which in turn can affect blood pressure and cholesterol levels. Poor nutrition may also affect blood pressure and cholesterol levels regardless of weight.

Continuous and absolute risk

For almost all risk factors there is no known threshold at which risk begins. Rather, there is an increasing effect as the exposure increases and the relationship between risk and disease is described as *continuous*. Although the increase in risk often starts at relatively low levels, the usual practice when monitoring is to focus on the riskier end of the spectrum. However, there is also value in monitoring moderate risk to assess trends in the wider population and to identify people who may benefit from preventative interventions that will help reduce or maintain their risk profile (AIHW 2009d).

In addition to being continuous, it is now recognised that the relationship between risk factors and disease is also integrated (Chan et al. 2008). Individuals are frequently likely to develop clusters of risk factors and the assessment of disease risk based on the combined effect of multiple risk factors is more accurate than that based on individual risk factors (National Vascular Disease Prevention Alliance 2009).

Absolute risk is a term used to define the probability of an event, for example a CVD event such as a stroke, occurring within a specified period, and takes into account an individual's entire risk factor profile rather than focusing on single risk factors. Tools for health professionals have recently been developed to assess the risk in people without known disease (National Vascular Disease Prevention Alliance 2009).

Comorbidity

When a person has two or more health problems at the same time it is known as *comorbidity*. Several of the risk factors for CVD also increase the risk of developing many other chronic diseases. For example, tobacco smoking increases the risk for CVDs such as CHD and stroke, as well as for cancer, respiratory disease, Type 2 diabetes, chronic kidney disease (CKD) and a variety of other conditions. Comorbid conditions can be both risk factors and associated conditions for CVD. People with Type 2 diabetes are more likely to develop CVD, and CVD can increase the risk of developing CKD. The two conditions also share a number of risk factors, resulting in many people developing both conditions together. These relationships are complicated and are explored in more detail in 'Chapter 10 Comorbidity of cardiovascular disease, diabetes and chronic kidney disease'.

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Protective factors

While the focus of this chapter is on modifiable risk factors, there is a range of factors, known as *protective factors*, which can reduce the likelihood of CVD developing or slow the progression and severity of the disease. Protective factors are discussed in more detail at the end of this chapter.

Risk factors for cardiovascular disease

The modifiable risk factors presented in this chapter are:

Behavioural factors

- tobacco smoking
- insufficient physical activity
- dietary behaviour
- excessive alcohol consumption

Biomedical factors

- high blood pressure
- high blood cholesterol
- overweight and obesity
- depression

These are the key modifiable risk factors for CVD. They can increase the likelihood of developing CVD and affect the severity, course and progression of the disease once it has developed. (Non-modifiable risk factors are not addressed in detail in this chapter.)

Aboriginal and Torres Strait Islander people

It is important to note that, although each risk factor is examined individually in this chapter, the presence of risk factors for CVD and the increased risk this brings is more common among Indigenous Australians. Self-reported results for CVD from the 2004–05 National Aboriginal and Torres Strait Islander Health Survey (NATSIHS) show that 53% of those surveyed were exposed to three or four of these risk factors (ABS & AIHW 2005).

In addition, Indigenous Australians are more likely to experience poor housing, low income, poverty, psychosocial stressors (such as the death of a family member or close friend), serious illness or disability and unemployment, which are all recognised as important contributors to the development of CVD (AIHW 2010a; Marmot 2005; van Holst Pellekaan & Clague 2005).

Tobacco smoking

What is tobacco smoking?

In this report, smoking refers to the smoking of tobacco products, including packet cigarettes, roll-your-own cigarettes, pipes and cigars. Smoking contributes to more hospitalisations and deaths than alcohol and illicit drug use combined (AIHW 2008b). It is the single most important preventable cause of ill health and death in Australia (AIHW 2010a).

In this chapter *Daily smokers* refers to those who smoke at least one cigarette per day, and *Occasional smokers* refers to those who smoke less often than daily (AIHW 2008a).

Passive smoking also has serious health consequences. It is defined as the breathing in of second-hand tobacco smoke, consisting of either smoke directly from burning tobacco or exhaled smoke from the smoker. Passive smoking is associated with health problems such as lower respiratory tract infections, lung cancer, CHD and childhood asthma (NHFA 2009).

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Smoking and cardiovascular disease

As its components are absorbed into the bloodstream, tobacco smoke increases the risk of CVD through many mechanisms. It damages blood vessels, increases the risk of plaques, increases the risk of clots at the site of plaques and reduces the blood's oxygen levels. Giving up smoking is associated with substantially improved cardiovascular function and reduced risk of cardiovascular morbidity and mortality (Gratziou 2009). The risk of a coronary event among ex-smokers declines rapidly after quitting. After 1 year of smoking cessation, the risk of CHD is halved compared to those who continue to smoke (DoHA 2004) and within 2–6 years the risk is similar to that of non-smokers, although some studies have found there is a residual increased risk for up to 10 years (Dobson et al. 1991; McElduff et al. 1998).

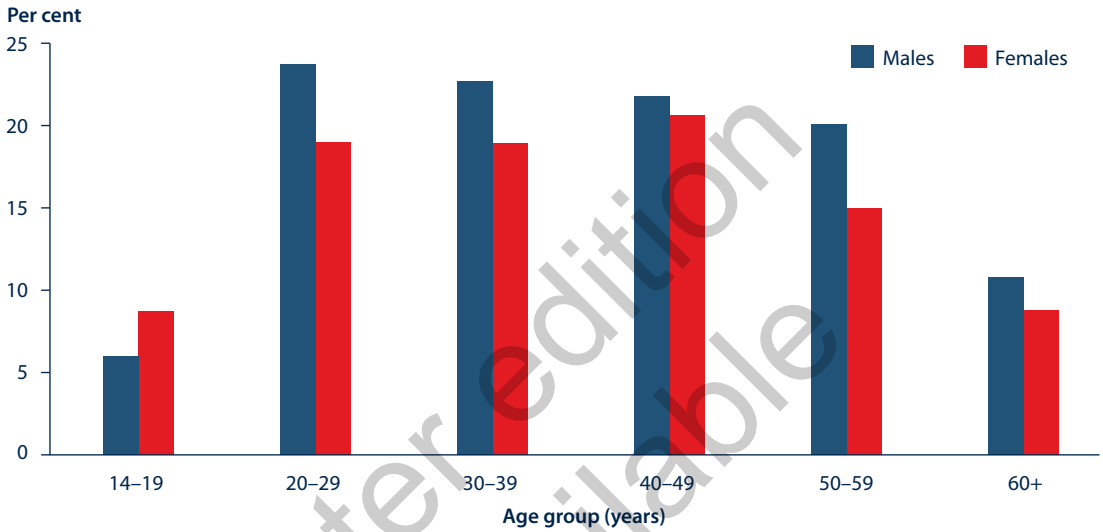
How many Australians smoke?

Based on the 2007 National Drug Strategy Household Survey (NDSHS), 17% of Australians aged 14 years and over smoked tobacco daily in 2007, equating to 2.9 million people. A further 1% smoked tobacco weekly and 2% smoked less than weekly. In total, just over 19% of Australians aged 14 years and over were current smokers in 2007 (AIHW 2008a).

Age and sex

Overall, males aged 14 years and over were more likely to be daily smokers than females (18% compared with 15% of females).

The proportion of daily tobacco smoking was lowest in the 14–19 year age group and highest among those aged between 20 and 59 years. Males were more likely to be daily smokers in all age groups except those aged 14–19 years, where more females smoked (9% of females compared to 6% of males) (Figure 2.1).



Note: Daily tobacco smokers only.

Source: AIHW analysis of 2007 National Drug Strategy Household Survey.

Figure 2.1: Prevalence of daily smokers, by age and sex, 2007

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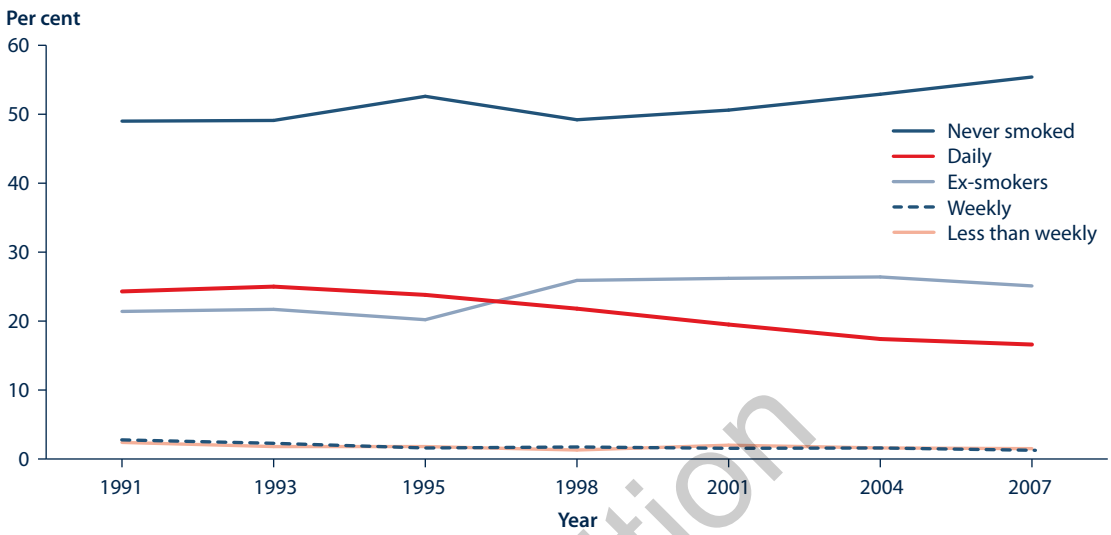
Trends

Smoking rates among Australian adults have declined steadily since the early 1970s, and this trend continues. The proportion of daily smokers among those aged 14 years and over declined to 17% in 2007, down from 24% in 1991 (Figure 2.2). Between 1991 and 2007, the percentage of people never having smoked increased from 49% to 55% and the proportion of ex-smokers increased from 21% to 25%.



Note: Rates not age-standardised.

Source: AIHW analysis of National Drug Strategy Household Survey, 1991–2007.

Figure 2.2: Percentage of the population aged 14 years and over who are daily smokers, 1991–2007

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Aboriginal and Torres Strait Islander people

Results from the 2008 National Aboriginal and Torres Strait Islander Social Survey (NATSISS), which surveyed people aged 15 years and over, showed that 47% of Indigenous people were current smokers, 20% ex-smokers and 34% never smoked (ABS 2009). After adjusting for differences in age structure, using results from the NATSISS and NHS, 45% of Indigenous Australians and 20% of non-Indigenous Australians were current smokers—that is they smoked one or more cigarettes a day. Similar percentages of the Indigenous and non-Indigenous populations were ex-smokers, 24% and 28% (Table 2.1).

Table 2.1: Smoking among Indigenous and non-Indigenous people aged 15 years and over, 2007–08

	Per cent of population	
	Indigenous persons[a]	**Non-Indigenous persons[b]**
Current smoker[c]	45.1	20.1
Ex-smoker	23.7	28.2
Never smoked	31.3	51.7
Total[d]	**100.0**	**100.0**

(a) Data from the ABS 2008 National Aboriginal and Torres Strait Islander Social Survey.

(b) Data from the ABS 2007–08 National Health Survey.

(c) Includes daily, weekly and current smokers.

(d) Totals may not sum to 100 due to rounding.

Source: ABS 2009a, 2009b.

Remoteness

There is a relationship between the proportion of the population that smoke and the remoteness area in which they live. For example, 25% of people living in *Remote and very remote* areas were smokers compared with 18% of people in *Major cities*. This difference may partly reflect the high proportion of Indigenous people living in remote areas.

Socioeconomic group

Smoking is related to socioeconomic group. In 2007, of those aged 14 years and over, 26% of people in the lowest socioeconomic group (the lowest fifth) smoked tobacco, compared with 14% of people in the highest socioeconomic group. (Note: socioeconomic group is defined using the Socio-Economic Indexes for Areas (SEIFA) Index of Relative Disadvantage, based on where people live. See Appendix A for further information.)

Burden of smoking

Overall, tobacco smoking was responsible for 8% of the total burden of disease and injury in Australia in 2003—10% for males and 6% for females (Begg et al. 2007). In 2003, tobacco smoking was estimated to account for 10% of the total burden of CVD in Australia (Begg et al. 2007). The monetary cost of tobacco use in Australia in 2004–05 was estimated to be $12 billion (Collins & Lapsley 2008).

International comparisons

Data on smoking from the Organisation for Economic Co-operation and Development (OECD) member countries places Australia among those with the lowest percentage of daily smokers (17%). Of those countries with 2007 data available, Sweden and the United States had the lowest proportion of daily smokers over 15 years old (14% and 15% respectively). Ireland and the Netherlands had the highest proportion, with almost one-third of the population aged 15 years and over smoking daily (Figure 2.3).

With few exceptions, OECD countries saw a decline in the prevalence of daily smoking between 1966 and 2007, with major reductions in the early part of this period and a slowing of the decline in the past decade.

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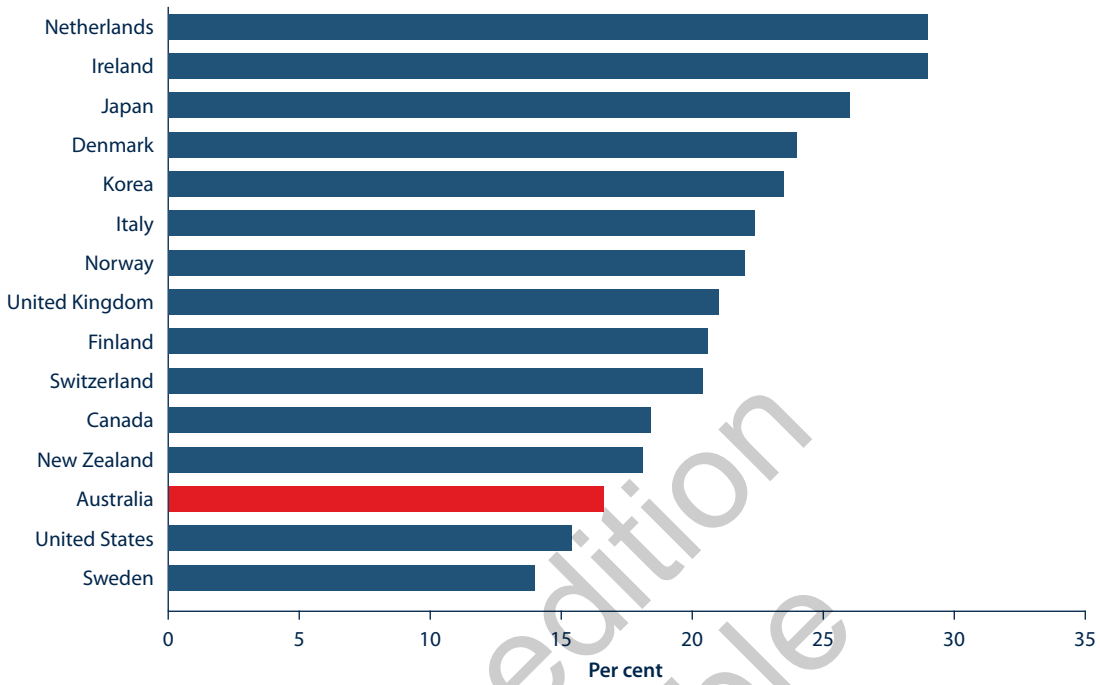

Note: Only countries with 2007 data have been included.

Source: OECD 2010.

Figure 2.3: Percentage of population aged 15 years and over smoking daily, selected countries, 2007

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Insufficient physical activity

What is physical activity?

Physical activity is any bodily movement that the muscles produce which results in energy expenditure. This activity can be in the form of deliberate activity in leisure time (exercise or sport) or other forms of non-leisure activity, such as walking or cycling for transport, or activity associated with a person's job. Even the activity associated with everyday tasks such as shopping and housework—known as incidental activity—can contribute to overall physical activity and its associated health benefits (AIHW 2008b).

If the energy going into the body (via food and drink) is not balanced by energy expenditure (via activity and internal bodily functions) over a sustained period of time, the excess food energy is stored as body fat. This can result in a person becoming overweight or obese. However, regardless of a person's weight, low levels of physical activity are a risk factor on their own (AIHW 2008b).

Sufficient physical activity

The *National Physical Activity Guidelines for Australians* (DoHA 1999) recommend that people get at least 30 minutes of moderate-intensity physical activity on most, preferably all, days of the week. This corresponds to the notion of sufficient activity—the amount needed to obtain health benefits. Examples of moderate-intensity activity include brisk walking, swimming, tennis and cycling. The most recent physical activity data available for Australia are from the 2007–08 National Health Survey (NHS). In the results that follow, *insufficient activity* refers to the NHS categories *Low* and *Sedentary*. It is also important to note that the NHS only measures physical activity related to fitness, recreation and sport (leisure time activities), and therefore does not measure incidental physical activity, such as moving about at work (ABS 2009a).

Physical activity/inactivity and cardiovascular disease

Physical inactivity is associated with an increased risk of ill health and death, particularly relating to CVD (AIHW 2008b). People who do not participate in regular physical activity are almost twice as likely to die from coronary heart disease as those who do participate (Thompson et al. 2003).

In addition to its direct effect on cardiovascular health, insufficient physical activity is linked to other CVD risk factors such as being overweight or obesity, high blood pressure, unfavourable levels of high-density lipoprotein and total blood cholesterol, and Type 2 diabetes.

Regular physical activity, whether deliberate or incidental, has a protective effect, lowering the risk of developing CVD and other CVD risk factors. More information about this is given in the section 'Protective factors' at the end of this chapter.

Later edition available

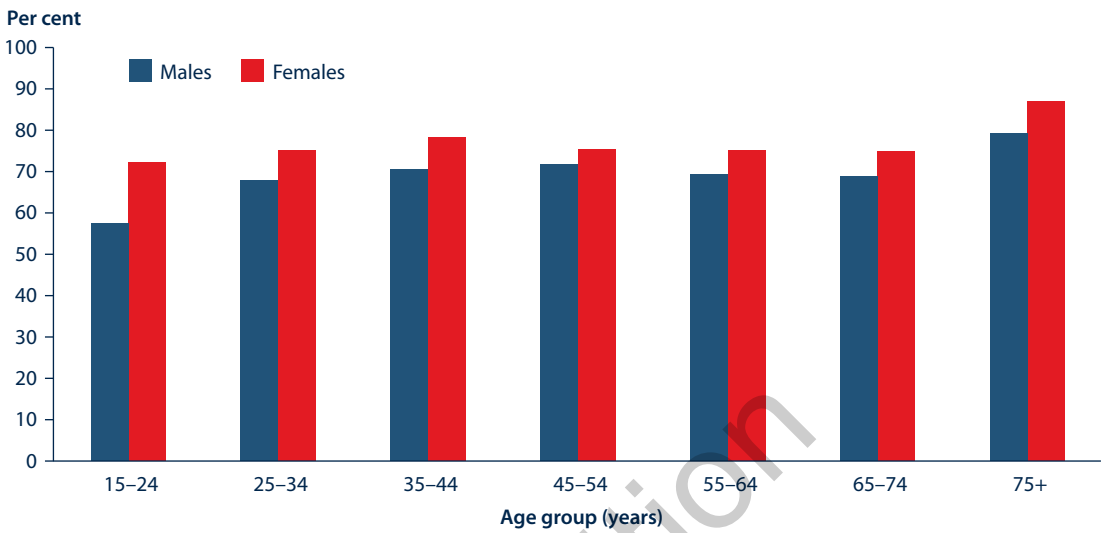
How many Australians are not sufficiently active?

Based on NHS definitions, in 2007–08 about 72% of Australians aged 15 years and over did not undertake sufficient physical activity, being either sedentary or having low levels of activity .

Age and sex

In 2007–08, around 73% of those aged between 25 and 65 years had *Low* or *Sedentary* levels of physical activity. The proportion of people with *Low* or *Sedentary* levels of physical activity was lowest in those aged 15–24 years (65%) and highest in those aged 75 years and over (84%) (Figure 2.4). Some of this pattern may reflect a reduced capacity among older people to undertake leisure activities because of frailty or illness.

Physical inactivity also varied by sex. Females were more likely than males to be physically inactive at all ages. Overall, 76% of Australian females did not get sufficient physical exercise compared to 68% of males.



Notes

1. Based on self-reports.
2. Insufficient physical activity includes the NHS categories *Sedentary* and *Low*.

Source: AIHW analysis of ABS 2007–08 National Health Survey.

Figure 2.4: Percentage of the population aged 15 years and over who were insufficiently physically active, by age and sex, 2007–08

Later edition available

Trends

Trend information suggests that the proportion of people not sufficiently physically active is increasing slowly. In 1995, 69% of those surveyed were in the *Low* or *Sedentary* group; in 2004–05 this had increased to just over 70% (ABS 2006b) and by 2007–08 the proportion was 72%.

Aboriginal and Torres Strait Islander people

Results from the 2004-05 NATSIHS indicate that of Indigenous people aged 15 years and over, 75% (crude rate) were sedentary or had low physical activity levels. Among non-Indigenous Australians, the comparable figure was 69%. After age-standardising the rate for Indigenous Australians was 80% and the non-Indigenous rate remained at 69%.

Remoteness

Physical inactivity levels were quite similar in all remoteness areas. After adjusting for differences in population age structure, *Major cities* had a similar proportion of people with low or sedentary physical activity levels (72%) as *Inner regional* areas (73%) and *Outer regional and remote* areas (74%).

Socioeconomic group

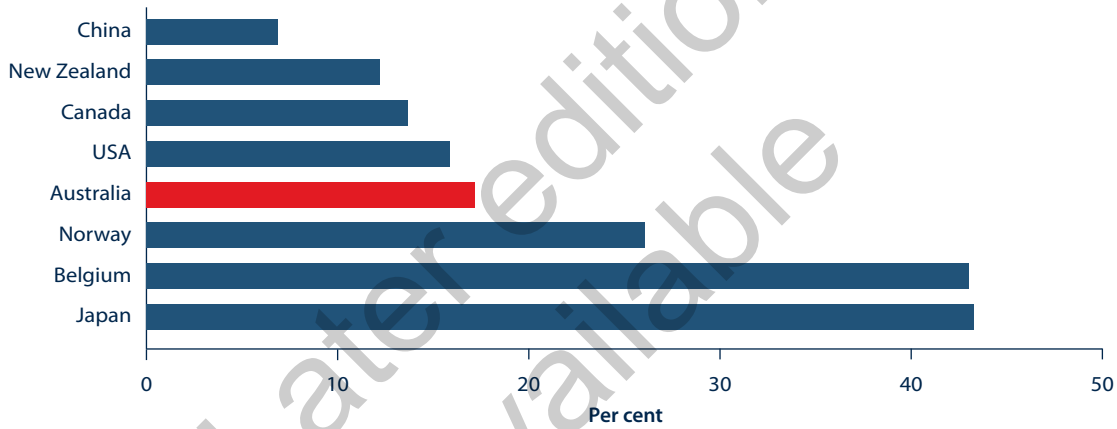
In 2007–08, people in the lowest socioeconomic group were more likely to have *Sedentary* or *Low* physical activity levels (79%) than those in the highest socioeconomic group (65%).

Burden of physical inactivity

Around 7% of the overall burden of disease and injury in Australia in 2003 was attributed to physical inactivity, placing it fourth out of the 14 risk factors studied by Begg and others (2007). In terms of CVD however, physical inactivity accounted for 24% of the burden, ranking it third behind high blood pressure (42%) and high blood cholesterol (35%) (Begg et al. 2007).

International comparisons

The 2005 International Physical Activity Questionnaire is a survey of international populations, used to determine health-related physical activity levels (Bauman et al. 2009). The category of *Low* physical activity in this report is similar to that used in the NHS. Levels of low physical activity in Australia were similar to those in the United States (17% and 16%, respectively), and higher than in other comparable countries, such as New Zealand (12%) and Canada (14%) (Figure 2.5).



Note: Percentages are age-standardised to the 2002 world population.

Source: Bauman et al. 2009.

Figure 2.5: Percentage of the population aged 18–65 years with low levels of physical activity, selected countries, 2002–2004

Later edition available

Poor dietary behaviour

What is poor dietary behaviour?

Poor dietary behaviour refers to a diet which does not support good health outcomes, or which could contribute to poor health. In contrast, good dietary behaviour supports and contributes to good health. Dietary behaviour plays an important role in health and wellbeing and can either reduce or increase the risk of various diseases, including the development of CVD (NHMRC 2003a).

Poor dietary behaviour and CVD

In individuals, the effect of dietary behaviour on CVD risk results from the combined effects of individual dietary factors, as well as total energy intake. Usually no single dietary component is responsible for the development of CVD. Excessive energy intake which results in people becoming overweight or obese is a considerable risk factor for CVD (NHMRC 2001b; WHO 2003). Poor dietary behaviour contributes to several biomedical risk factors, including high blood pressure and blood cholesterol levels, as well as Type 2 diabetes. The National Health and Medical Research Council (NHMRC) *Dietary guidelines for all Australians* (2003a) recommend that people eat a wide variety of nutritious foods and limit the intake of sugar, salt, alcohol and fat, especially saturated fat.

How many Australians have dietary behaviour risk factors for CVD?

The NHS is used in conjunction with state and territory health surveys to determine the proportion of Australians who are at risk of developing chronic disease as a result of poor diet. Data on the intake of fat (via whole milk), fruit and vegetables and salt are reported in this section because of their importance to the development of CVD.

Later edition available

Fat intake

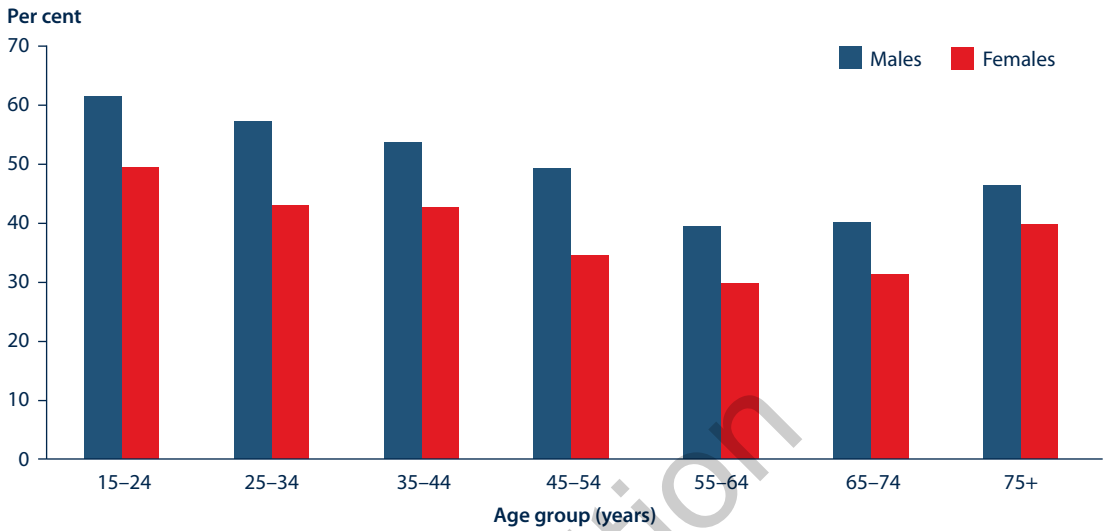
There are several different types of fats, including saturated, polyunsaturated, monounsaturated and trans fats. Diets high in saturated or trans fat increase the risk of CHD by raising blood cholesterol levels, notably the low-density lipoprotein form of cholesterol (NHMRC 2003c). The NHMRC recommends that the overall intake of fat should be limited to help prevent overweight and obesity.

Dairy products provide a major source of nutrients in the Australian diet. However, they contribute to over a quarter of the saturated fat in the diet of Australian adults and reduced or low-fat dairy products are generally recommended (NHMRC 2003c). Because dairy products contribute considerably to saturated fat intake, the proportion of people who usually consume whole (full-cream) milk can be used as an indicator of saturated fat intake (Marks et al. 2001).

Age and sex

Data from the 2007–08 NHS showed that 46% of the Australian population aged 5 years and over years usually drank whole milk (51% of males and 40% of females). The highest level of whole milk consumption was among 15–24 year olds (55%). The lowest consumption level, of 35%, was in the 55–64 age group (Figure 2.6).

Of the remainder of the Australian population, 49% consumed other types of milk (which included low or reduced fat and skim milk) and around 5% did not drink milk at all.



Source: AIHW analysis of ABS National Health Survey 2007–08.

Figure 2.6: Percentage of the population consuming whole (full-cream) milk, by age and sex, 2007–08

Later edition available

Consumption of fruit and vegetables

There is evidence that increased consumption of fruit, vegetables and legumes reduces the risk of cardiovascular conditions such as CHD, stroke, and high blood pressure, as well as other diseases such as diabetes and some cancers (Department of Agriculture, Fisheries and Forestry 2008; NHMRC 2003b).

The beneficial components of fruit and vegetables are not yet fully understood, but there is evidence that antioxidant phytochemicals, antioxidants and other vitamins, minerals and fibre found in fruit and vegetables may contribute to lower levels of blood cholesterol, blood pressure and atherosclerosis. Fruit and vegetable consumption may also take the place of less beneficial foods in daily food intake.

The *Australian Guide to Healthy Eating* recommends that adults eat at least two to four serves of fruit and four to eight serves of vegetables and legumes each day (NHMRC 2003a). The majority of Australians do not meet these recommendations. According to the 2007–08 NHS, 51% of the population did not consume sufficient amounts of fruit and 91% did not consume sufficient amounts of vegetables each day.

Age and sex

Younger people were less likely to consume an adequate amount of fruit than those in older age groups, but the pattern for vegetable consumption across age groups was less clear.

Self-reported 2007–08 NHS data show that overall females had a slightly higher fruit and vegetable intake than males. The data indicated that 56% of males and 46% of females did not consume the recommended levels of fruit, and 92% of males and 90% of females did not consume the recommended levels of vegetables.

Aboriginal and Torres Strait Islander people

Aboriginal and Torres Strait Islander people had higher proportions of inadequate fruit and vegetable consumption than other Australians. From the NATSIHS, 69% of Indigenous people did not eat the recommended serving of fruit and 82% did not have the recommended vegetable intake. The comparable figures for non-Indigenous Australians were 61% and 77%.

Remoteness

People in *Major cities* (50%) were more likely to consume an adequate amount of fruit than those in *Outer regional and remote* areas (43%), but were less likely (7%) than those in regional and remote areas (10%) to consume an adequate amount of vegetables.

Socioeconomic group

Results from the 2007–08 NHS showed some association between inadequate fruit consumption and socioeconomic group, but there was little association for inadequate vegetable consumption. The lowest socioeconomic group had the highest percentage of inadequate fruit consumption (54%) while the highest socioeconomic group had the lowest (47%). For inadequate vegetable consumption, levels were much higher (around 90%), but the differences between the highest and lowest socioeconomic groups were relatively small—about 2%.

High consumption of salt

A high dietary intake of salt may contribute to increased blood pressure, which is an important CVD risk factor. Foods high in salt include bread, cheese, processed meats and snack food.

No national data currently exist to assess levels of salt consumption among Australians, but in one study conducted in Hobart in the mid-1990s, 94% of males aged 18–70 years and 64% of females consumed more than the maximum intake for salt of 100 mmol/day, as recommended by NHMRC (Beard et al. 1997).

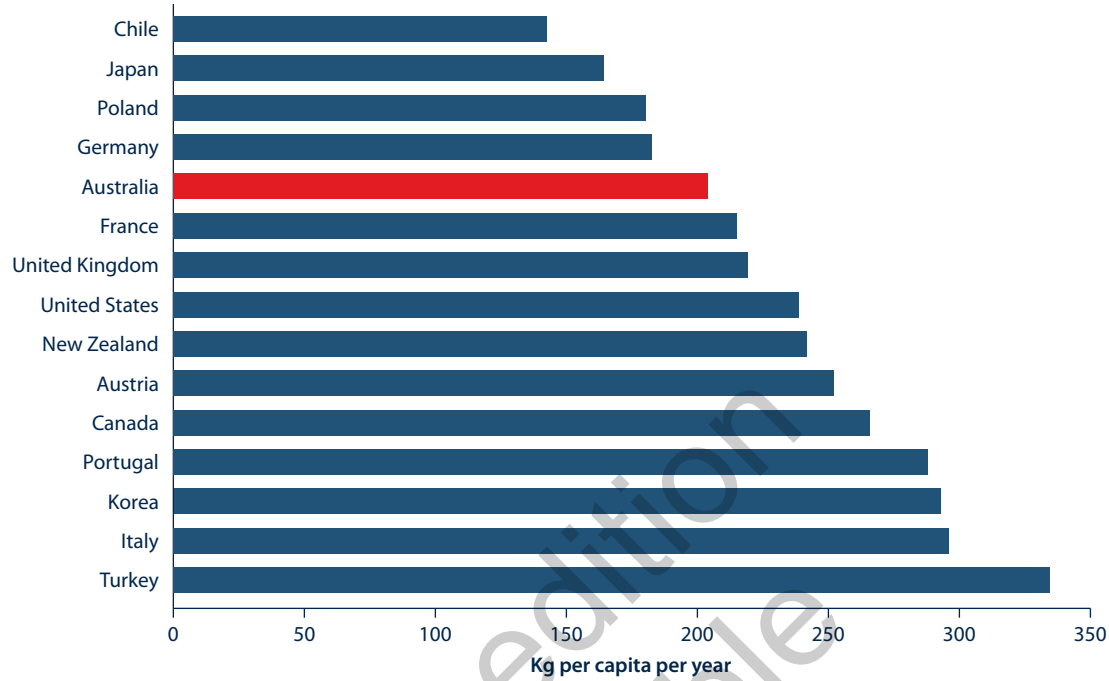
Later edition available

Burden of dietary behaviour

It has been estimated that inadequate fruit and vegetable consumption accounted for 2% of the disease and injury burden among females and 3% among males, placing it seventh out of 14 risk factors in *The burden of disease and injury in Australia 2003* (Begg et al. 2007). It was the sixth largest contributor to the burden of CVD in 2003—accounting for 10%—almost equal to tobacco smoking.

International comparisons

A rudimentary measure for comparing dietary behaviour between countries is to compare the total amount of dietary components consumed by a given country, based on sales divided by the total population. Using this measure, in 2007 Australians consumed less fruit and vegetables than many other OECD countries, including comparable nations such as the United States, the United Kingdom and New Zealand. Australians had 204 kg of fruit and vegetables per capita available to them, while Canadians had 266 kg and the United Kingdom had 219 kg (Figure 2.7) (OECD 2009b).



Source: OECD 2010.

Figure 2.7: Per capita intake of fruit and vegetables (per year), selected OECD countries, 2007

Later edition available

Excessive alcohol consumption

What is alcohol consumption?

Alcohol consumption refers to the consumption of drinks containing *ethanol*, commonly referred to as *alcohol*. The quantity, frequency or regularity with which alcohol is drunk provides a measure of the levels of alcohol consumption.

In 2009, the NHMRC released revised guidelines for the safe consumption of alcohol, recommending lower levels of alcohol consumption than those in the 2001 guidelines (Table 2.2). However, in this report the 2001 guidelines are used because they were still in place when the 2007 NDSHS data used here were collected and reported on.

Table 2.2: 2001 National Health and Medical Research Council safe alcohol consumption guidelines

	Males	Females
	Standard drinks per week	
Low risk	Up to 29	Up to 15
Risky	29–42	15–28
High risk	43 or more	29 or more

Source: NHMRC 2001a.

The risk of alcohol-related harm can be measured as either short-term or long-term risk. Short-term risk is associated with the level of drinking on a particular occasion whereas long-term risk of harm is associated with regular daily patterns of drinking. While this section focuses on the long-term risk of alcohol-related harm and its relationship to cardiovascular disease, it is important to note that binge drinking, also known as 'short-term risky drinking', is associated with higher blood pressure and increased risk of death from stroke.

Alcohol and CVD

Long-term excessive drinking is associated with CVDs such as stroke, heart disease, hypertension, heart failure and congenital heart disease (Begg et al. 2007; English et al. 1995; Mann et al. 2004). The lifetime risk of harm from drinking alcohol increases with the amount consumed (NHMRC 2009). Additionally, alcohol is a source of energy and therefore it must be considered for its potential to increase body mass and lead to overweight and obesity. The 10 grams of alcohol in a standard drink contain 290 kilojoules, around the same amount of energy as in 8 grams of fat. Alcohol can also affect blood triglyceride levels, complicating the effects of high blood cholesterol (AIHW 2004).

Although the consumption of alcohol should never be encouraged on health grounds, low levels of alcohol have been thought to provide some protection against CVD. However, the most recent Australian Burden of Disease Study reported that the only group for whom the benefits of small amounts of alcohol outweighed the harmful effects were females over the age of 65 years (AIHW 2010a) and others have suggested that any benefits from alcohol consumption are restricted to middle-aged and older adults in countries with high rates of CVD (Beaglehole & Bonita 2009). Other research advises that benefits from alcohol consumption only occur at very low levels of drinking, or that there is no protective factor at all (NHMRC 2009).

Later edition available

How many Australians drink alcohol?

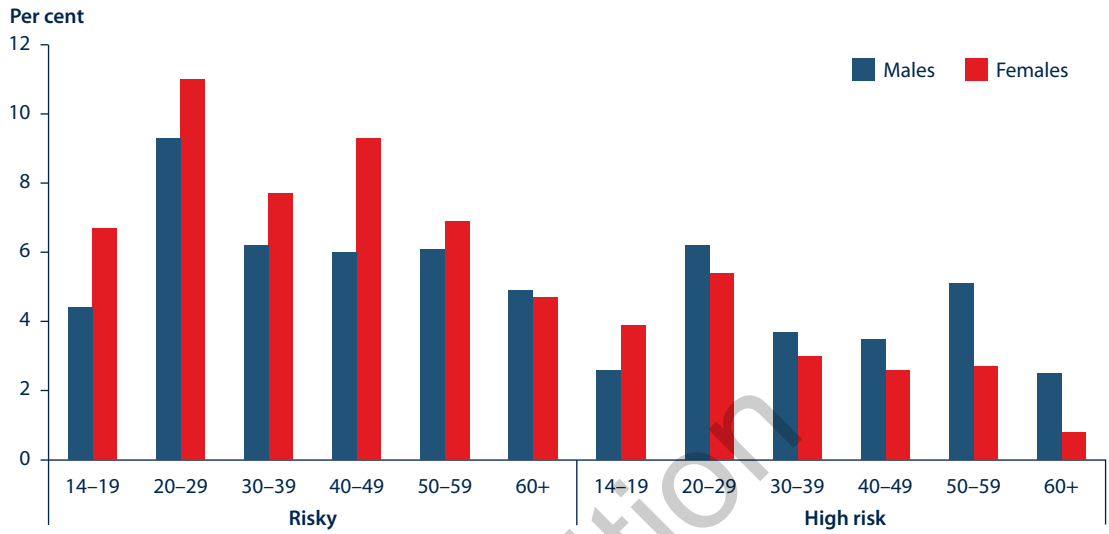
Results from the 2007 NDSHS, indicate that 73% of Australians aged 14 years and over consumed alcohol at *Low risk* levels and a further 10% drank at levels considered to be harmful (*Risky* or *High risk*—see Table 2.2). Seventeen per cent of people aged 14 years and over had abstained from alcohol in the 12 months before the survey.

Sex and age

In 2007, an estimated 7% of Australians aged 14 years and over consumed alcohol at *Risky* levels and a further 3% at *High risk* levels.

The proportions of males and females consuming alcohol at risky levels were similar—around 10%, however males were slightly more likely than females to drink at *High risk* levels—4% of males and 3% of females. Females were more likely to abstain from drinking alcohol than males, 20% compared with 14%.

The group most likely to consume alcohol at harmful levels were those aged 20–29 years with 16% drinking at *Risky* and *High risk* levels. The high level of drinking for this age group was seen in both males and females with 9% of males and 11% of females drinking at *Risky* levels and 6% of males and 5% of females drinking at *High risk* levels (Figure 2.8).



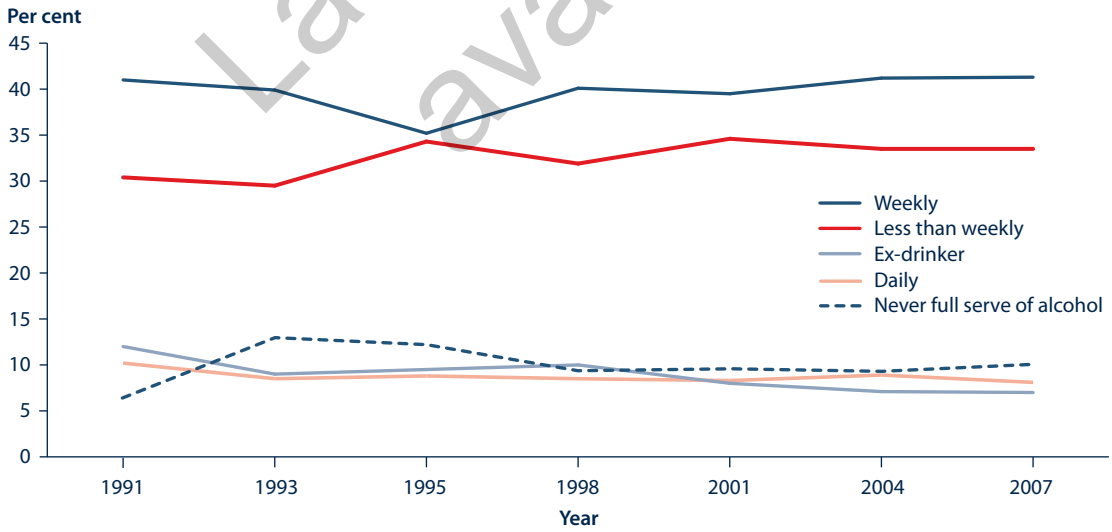
Source: AIHW 2008a.

Figure 2.8: At risk alcohol consumption, by risk category, age and sex, 2007

Later edition available

Trends

There has been little change in the proportion of Australians aged 14 years and over who consumed alcohol between 1991 and 2007 (Figure 2.9).



Source: AIHW analysis of the National Drug Strategy Household Survey, 1991–2007

Figure 2.9: Percentage of the population by drinking status, 1991–2007

Aboriginal and Torres Strait Islander people

Based on data from the self-reported 2004–05 NATSIHS, an estimated 49% of Indigenous Australians consumed alcohol in the week before the survey. Sixteen per cent of Indigenous people drank at *Risky* or *High risk* levels. Ten per cent of Indigenous Australians had never consumed alcohol and a further 14% had not consumed alcohol in the 12 months leading up to the survey.

Remoteness

Consumption of harmful levels of alcohol increased with remoteness. Among people aged 14 years and over, a higher percentage living in *Remote and very remote* areas consumed alcohol at harmful levels (15%) than those living in *Major cities* (10%). This difference was more pronounced for females than for males with a comparatively high proportion of females living in *Remote and very remote* areas drinking to harmful levels. While 9% of males in *Major cities* and 13% in *Remote and very remote* locations drank to harmful levels, the corresponding figures for females were 10% and 18%.

Socioeconomic group

There was no relationship between socioeconomic group and the consumption of alcohol at *Risky* and *High risk* levels. Eleven per cent of people in the lowest socioeconomic group consumed alcohol at harmful levels compared with 12% in highest socioeconomic group.

Burden of alcohol

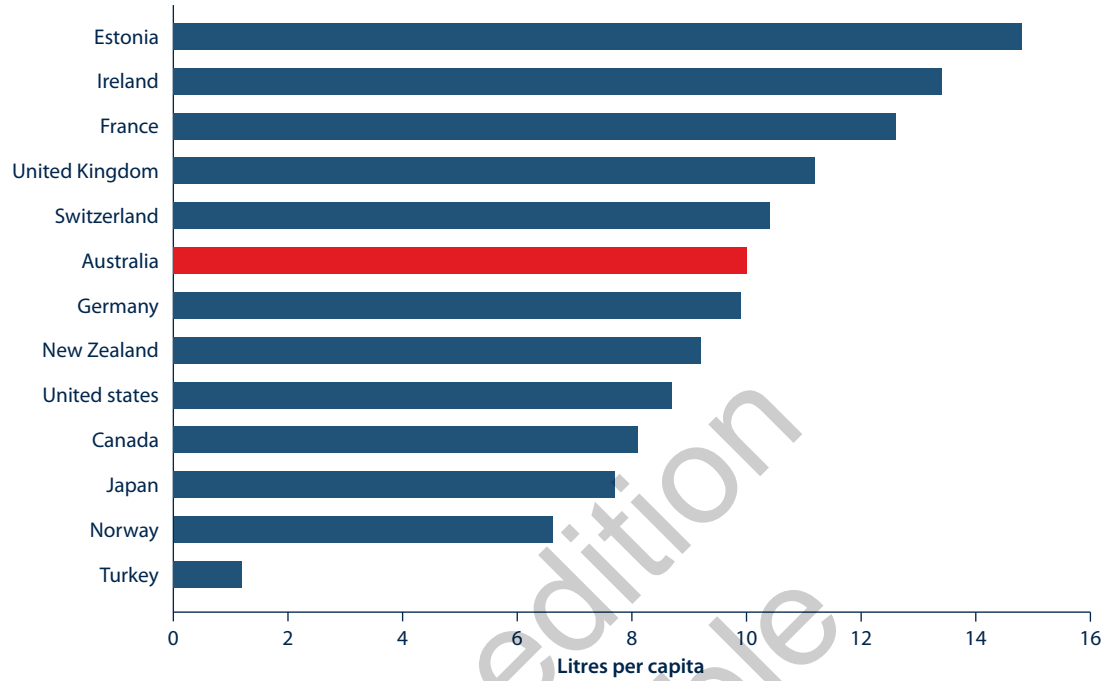
Alcohol has both hazardous and possibly protective effects on health, and the age and sex distribution of these effects varies in important ways. In Australia in 2003, it was estimated that alcohol caused 4% of the total burden of disease and injury for males and 1% for females. These estimates represent a balance between alcohol's harmful and beneficial effects: 5% harm and 1% benefit for males and 2% harm and 1% benefit for females. (Begg et al. 2007).

Later edition available

International comparisons

A useful measure when making international comparisons is to use per person consumption of pure alcohol, which is based on annual sales. This method takes into account the differing alcohol content of drinks. For example, a 'light' Australian beer contains about 2.7% alcohol by volume, meaning that a 375 ml bottle contains about 10 ml of pure alcohol and a 150 ml glass of wine containing 11.5% alcohol will have about 17 ml of pure alcohol.

Australia had a slightly higher annual per capita consumption of alcohol than the average for the 24 OECD countries that provided data for 2007. The average amount of alcohol consumed by people in these countries was 9.8 litres per person aged of 15 years and over—for Australia it was 10.0 litres per person (Figure 2.10). Consumption ranged from 1.2 litres per person per year in Turkey to 14.8 litres in Estonia.



Source: OECD 2010.

Figure 2.10: Per capita alcohol consumption among people aged 15 years and over, selected OECD countries, 2007

Later edition available

High blood pressure

Worldwide, high blood pressure has been found to be responsible for more deaths and disease than any other biomedical risk factor (Lopez et al. 2006). For purposes of population health monitoring, the WHO defines high blood pressure using specified levels of systolic and diastolic pressure (and receiving medication for high blood pressure). However, there is a *continuous* relationship between blood pressure levels and CVD risk. This means that CVD risk increases with each increase in blood pressure from the 'ideal level' of a systolic blood pressure of 115 mmHg (Lawes et al. 2003), making cut-off levels for high blood pressure somewhat arbitrary.

What is high blood pressure?

High blood pressure, often referred to as *hypertension*, is prolonged elevation of the blood pressure.

Blood pressure represents the forces that blood exerts on the wall of the arteries and is written as systolic/diastolic (for example, 120/80 mmHg, stated as '120 over 80' where mmHg is a unit of pressure described as *millimetres of mercury (mmHg)*). Systolic blood pressure reflects the maximum pressure in the arteries when the heart muscle contracts to pump blood; diastolic blood pressure reflects the minimum pressure in the arteries when the heart muscle relaxes before its next contraction.

The WHO defines high blood pressure as any of the following:

- systolic blood pressure of 140 mmHg or more
- diastolic blood pressure of 90 mmHg or more
- receiving medication for high blood pressure.

In this report, high blood pressure is defined using the WHO guidelines, although it is noted that these are not the only guidelines used to define the condition (Whitworth et al. 2003).

High blood pressure and cardiovascular disease

High blood pressure is a major risk factor for stroke, coronary heart disease, heart failure, peripheral vascular disease and kidney failure. It has also been considered a CVD in its own right. The risk factors for high blood pressure are largely the same as those for other forms of CVD. They include age, poor diet (particularly a high salt intake), obesity, excessive alcohol consumption, and insufficient physical activity.

Studies have shown there is a relationship between blood pressure and risk of cardiovascular disease, chronic kidney disease and death (NHFA 2009). When high blood pressure is controlled, the risk is reduced, but not necessarily to the levels of unaffected people (WHO International Society of Hypertension 1999).

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How many people have high blood pressure?

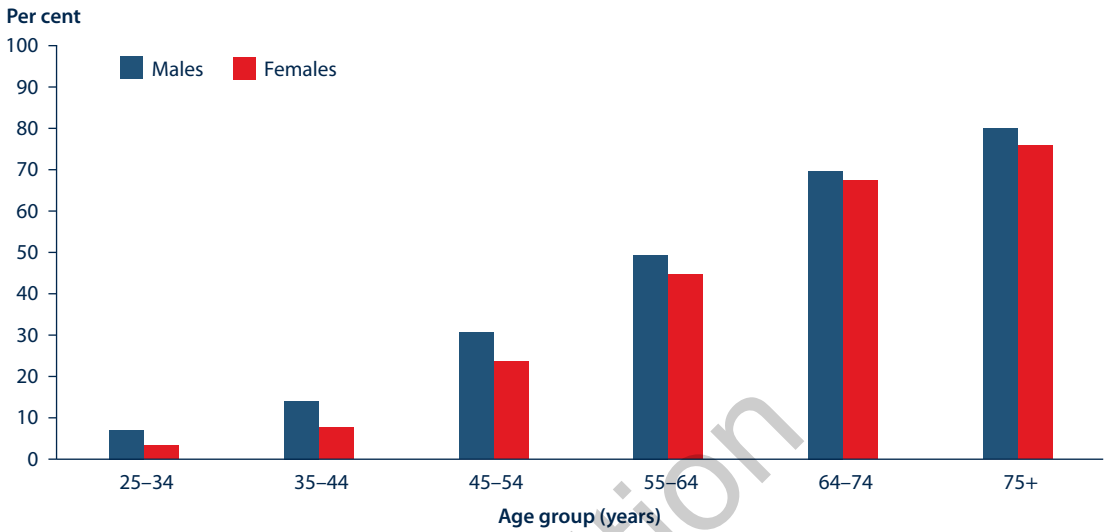
High blood pressure is common among both males and females in Australia. Based on measured data from the 1999–2000 Australian Diabetes, Obesity and Lifestyle (AusDiab) Study, which provides the most recent national information about the prevalence of measured high blood pressure in Australia, it was estimated that almost 30% of Australians aged 25 years and over had the disorder—about 3.7 million Australians.

Sex and age

The age-standardised high blood pressure rates estimated from the 1999–2000 AusDiab Study were 32% for males and 27% for females. The proportion of those with high blood pressure increased with age for both males and females with only 5% recording high blood pressure in the 25–34 year age group, but rising to 78% among those aged 75 years and over. Males had consistently higher blood pressure than females across all age groups (Figure 2.11).

Trends

Trends in Australian blood pressure levels may differ substantially according to where people live and to the age span of those examined. Over the two-decade period from 1980 to 1999–2000, the proportion of those with high blood pressure at least halved among those aged 25–65 years who lived in Australia's urban areas. However, if the group is widened to all Australians aged 25 years and over, the prevalence remained fairly steady over the shorter period between 1995 and 1999–2000 (Figure 2.12) (AIHW 2004).



Source: AIHW analysis of 1999–2000 AusDiab Study.

Figure 2.11: Percentage of the population aged 25 years and over with measured high blood pressure, by age and sex, 1999–2000

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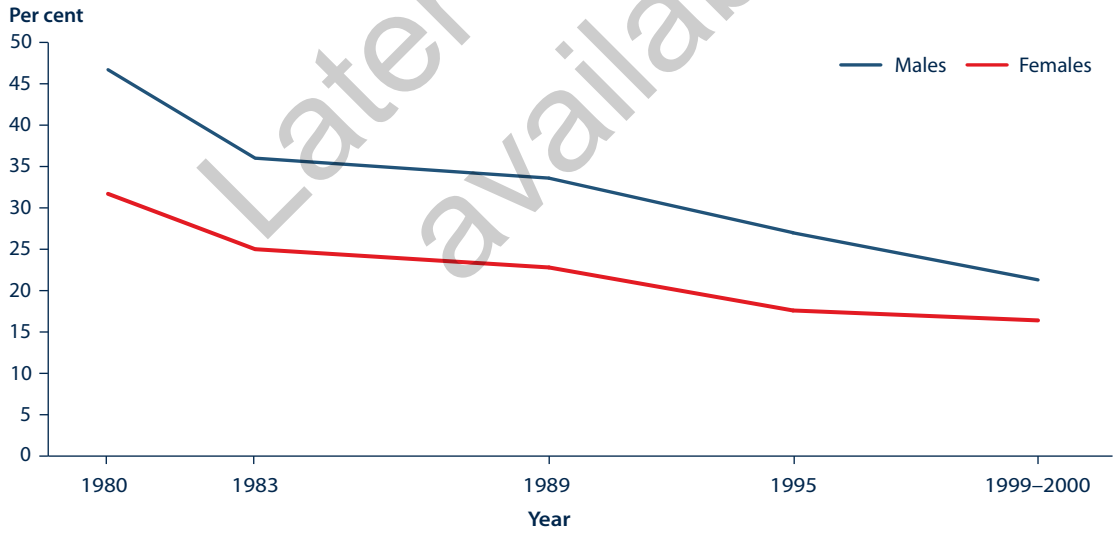

Notes

1. Age-standardised to the 2001 Australian population.
2. Urban areas only.

Sources: AIHW analysis of 1980, 1983, and 1989 Risk Factor Prevalence Studies; 1995 National Nutrition Survey; 1999–2000 AusDiab Study.

Figure 2.12: Prevalence of measured high blood pressure among people aged 25–64 years, by sex, 1980 to 1999–2000

Aboriginal and Torres Strait Islander people

There have been no national surveys that measured blood pressure among Indigenous people. However, self-reported data from the 2004–05 NATSIHS indicated that 7% of all Indigenous Australians and 42% of those aged 55 years and over, had high blood pressure as a long-term condition. After adjusting for differences in the age structure of the Indigenous and non-Indigenous populations, the prevalence of high blood pressure among Indigenous Australians was 1.6 times as high as for non-Indigenous Australians (AIHW: Penm 2008).

Remoteness

The proportion of the population with high blood pressure in 1999–2000 was similar for those living in *Rural* and *Urban* areas, 31% and 30% respectively.

Socioeconomic group

When the highest level of education attained was used as an indicator of socioeconomic position, results from the AusDiab Study indicated that people who had not completed secondary school were more likely to have high blood pressure than those who had completed high school. Among those who had not completed secondary school, 33% had high blood pressure. The comparative rates were 27% for those whose highest level of education was secondary school and 29% for those with higher levels of education.

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Burden of high blood pressure

In Australia, nearly 8% of the overall burden of disease in 2003 was attributed to high blood pressure, ranking it closely behind the leader—tobacco use.

However, high blood pressure accounted for 42% of the burden of CVD, making it the biggest contributor to this disease. Four-fifths of this burden related to premature death and the remainder to disability (Begg et al. 2007).

International comparisons

The WHO produces country-level estimates for average systolic blood pressure using data collated in the *WHO Global InfoBase* (WHO 2005). The data indicate that Australia has had a lower mean systolic blood pressure than the United Kingdom, New Zealand and Canada since at least 2002. International comparisons must however be treated with caution because the methods used to collect the information may vary between countries.

High blood cholesterol

What is cholesterol?

Cholesterol is a fatty substance produced by the liver and carried by the blood to supply the rest of the body. Its natural function is to supply material for cell walls and hormones, but if levels in the blood become too high this can lead to atherosclerosis and heart disease.

Two of cholesterol's components can play an important role in CVD:

- low-density lipoprotein (LDL) cholesterol, often known as 'bad' cholesterol
- high-density lipoprotein (HDL) cholesterol, often known as 'good' cholesterol.

High levels of LDL can contribute to atherosclerosis, the build-up of plaques within the arteries, a condition that may lead to the development of CVD. In contrast, high levels of HDL can have a protective effect and help reduce atherosclerosis.

Although LDL and HDL have opposing effects, the total cholesterol level is often used as an indicator of CVD risk and, as a general principle, the lower it is the better. For clinical and population monitoring purposes a level of 5.5 mmol/L is often labelled as 'increased' and one of 6.5 or more as 'high', but these are arbitrary levels and desirable levels may be below 4 mmol/L.

High cholesterol and CVD

High blood cholesterol is a major risk factor for coronary heart disease, ischaemic stroke and peripheral vascular disease. For most people, saturated fat in the diet is the most important factor associated with elevated blood cholesterol levels although genetic factors can also affect blood cholesterol levels severely in some individuals. Sufficient physical activity and a healthy diet play an important role in maintaining healthy blood cholesterol levels (NHMRC 2003a).

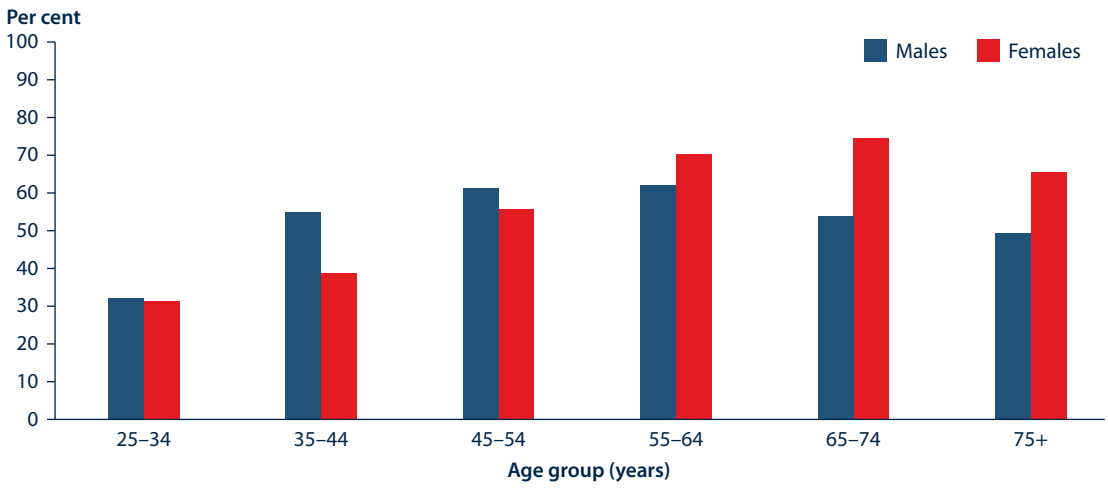
How many people have high blood cholesterol?

From the results of the 1999–2000 AusDiab Study, in which blood samples were obtained from a random sample of people, it was estimated that around 51% of Australians aged 25 years and over—about 6.4 million people—had blood cholesterol levels of 5.5 mmol/L or more.

Later edition available

Sex and age

Results from the 1999–2000 AusDiab Study showed that the prevalence of high blood cholesterol varied with both age and sex. For females, there was a steady increase in prevalence to 65–74 years, after which it declined. For males, it increased steadily with age until 55–64 years and then fell. Between the ages 25 years and 54 years, a higher proportion of males than females had high blood cholesterol, however for age 55 years and over the situation was reversed and a higher proportion of females had high levels (Figure 2.13).

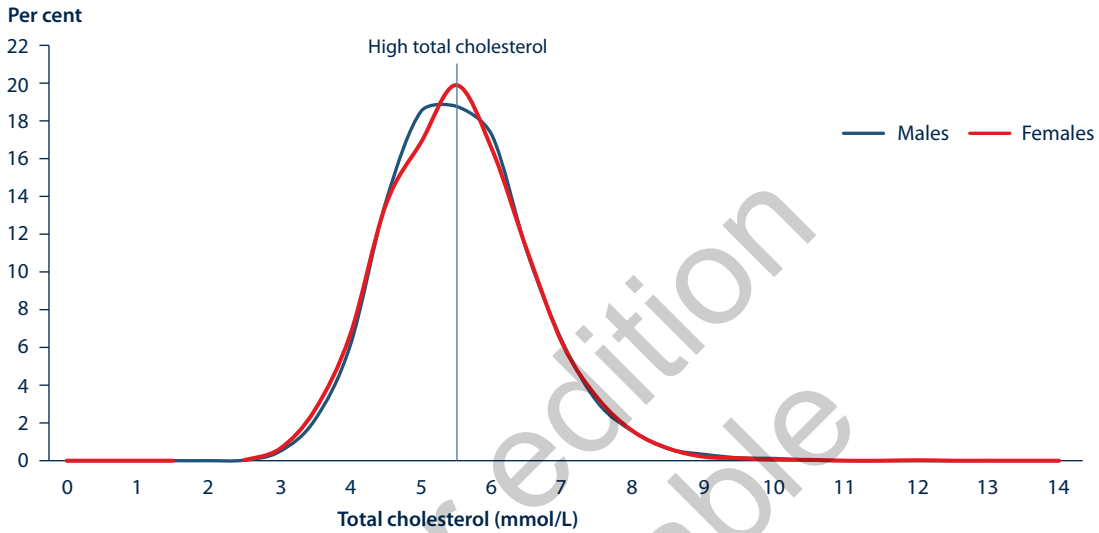


Source: AIHW analysis of 1999–2000 AusDiab Study.

Figure 2.13: Percentage of the population aged 25 years and over with high blood cholesterol, by age and sex, 1999–2000

High blood cholesterol population distribution

Figure 2.14 shows the distribution of total cholesterol levels for the Australian population based on the AusDiab Study. The mean total blood cholesterol level for males was 5.5 mmol/L and for females it was 5.4 mmol/L. Roughly half of all Australians adults aged 25 years and over had a total blood cholesterol level that was considered to be 'increased' or 'high'.



Note: Missing values were excluded from the numerator and the denominator.

Source: AIHW analysis of 1999-2000 AusDiab Study.

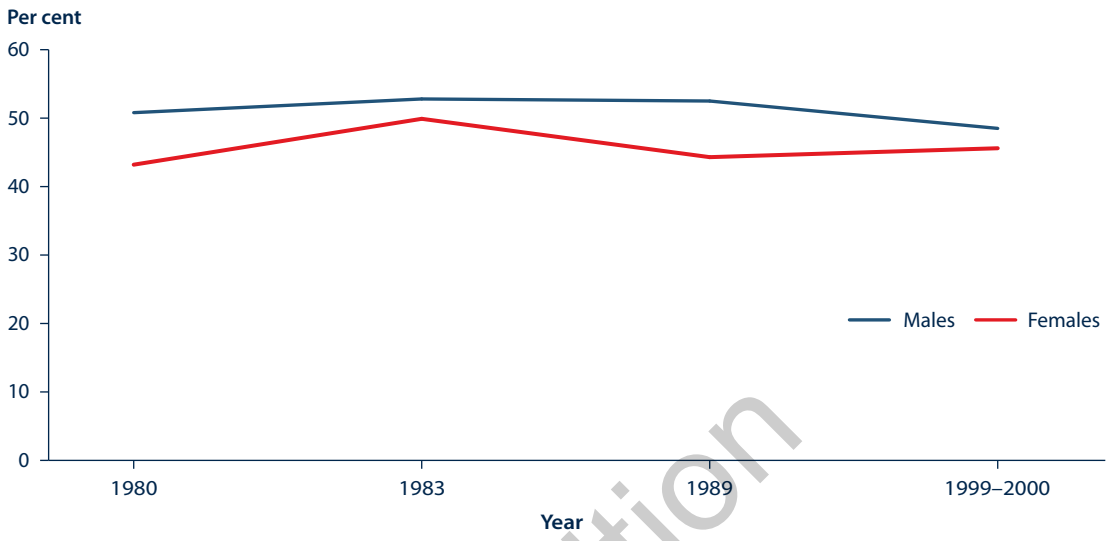
Figure 2.14: Distribution of total measured cholesterol, people aged 25 years and over, 1999-2000

Later edition available

Trends

As for trends in blood pressure, the only trend data available for high blood cholesterol levels are for people aged 25–64 years, living in capital cities, for the period 1980 to 1999–2000.

There were no marked changes in age-adjusted prevalence for high blood cholesterol in this group between 1980 and 1999–2000. In 1980, 51% of males and 43% of females had high blood cholesterol compared with 49% of males and 46% of females in 1999–2000 (Figure 2.15).



Notes

1. Age-standardised to the 2001 Australian population.
2. Urban areas only.
3. High blood cholesterol is total cholesterol level of 5.5mmol/L or more.

Source: AIHW analysis of 1980, 1983, and 1989 Risk Factor Prevalence Study and 1999–2000 AusDiab Study.

Figure 2.15: Prevalence of measured high blood cholesterol among people aged 25 to 64 years, by sex, 1980 to 1999–2000

Later edition available

Aboriginal and Torres Strait Islander people

There are no national data on measured blood cholesterol levels for Indigenous Australians.

Remoteness

When comparing geographic areas, the 1999–2000 AusDiab Study showed that people living in *Rural* areas were slightly more likely to have high blood cholesterol than their *Urban* counterparts—50% compared with 47% respectively. Blood cholesterol levels for females did not vary by region with both *Urban* and *Rural* areas both showing 48% with high levels. However, for males living in *Urban* areas, 45% had high blood cholesterol levels compared with 51% of those living in *Rural* areas.

Socioeconomic group

When educational level was used as an indicator of socioeconomic group, the AusDiab Study data showed that in 1999-2000 those with lower levels of education were somewhat more likely to have high blood cholesterol levels. For instance, half of those who did not complete secondary school had high blood cholesterol levels compared with 46% of those who undertook tertiary, technical or further education.

Burden of high blood cholesterol

Around 6% of the burden of disease and injury in Australia in 2003 was attributed to high blood cholesterol, placing it fifth out of the 14 risk factors examined (Begg et al. 2007). High blood cholesterol, at 35%, was the second highest contributor to the burden of CVD after high blood pressure (Begg et al. 2007).

Overweight and obesity

What are overweight and obesity?

Overweight and obesity are conditions in which a person is considered to carry an unhealthy amount of excess body weight. Highly prevalent in Australia, these conditions occur when the amount of energy consumed through eating and drinking is greater than the amount of energy that the body uses. This results in weight gain over time as the unused energy is stored as body fat. Obesity is a severe form of being overweight and is a well-established risk factor for CVD (NHMRC 2003a; Wang et al. 2008).

The two main methods for monitoring body weight at the population level are the body mass index (BMI) and waist circumference.

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Body mass index

BMI is the most commonly used measure for monitoring overweight and obesity in a population. It is calculated by dividing a person's weight in kilograms by the square of their height in metres (kg/m^2). A BMI of 25 or more indicates that a person is overweight and a BMI of 30 or more indicates they are obese. In this report, unless stated otherwise, being overweight refers to a BMI of 25 or more and BMI is the main measure used in this section. The classifications of overweight and obesity are based primarily on the association between BMI and mortality and are the standards recommended by WHO and outlined in the National Health Data Dictionary (NHDD). These classifications may not be suitable for all ethnic groups, some of whom may have equivalent levels of risk at lower BMI (for example, Asians) or higher BMI (for example, Polynesians). For children and adolescents aged 2–17 years, a separate classification of body weight, based on age and sex, has been developed.

Waist circumference

The NHDD defines waist circumferences of 94 cm or more in men and 80 cm or more in women as indicating an increased risk of ill health and, 102 cm or more in men and 88 cm or more in women indicating a substantially increased risk.

Overweight and obesity, CVD and other disorders

Being overweight and in particular being obese, are risk factors for diseases and conditions that include CHD, high blood pressure, high blood cholesterol, Type 2 diabetes, certain cancers, psychosocial problems and musculoskeletal conditions. As the level of excess weight increases, so does the risk of developing these conditions. Additionally, being overweight can hamper the ability to control or manage chronic disorders. Weight loss reduces the incidence and severity of the majority of these disorders.

How many people are overweight?

Rates of overweight and obesity are continuing to increase in Australia and overseas (OECD 2009b). Based on data measured in the 2007–08 NHS, the majority of adults (60%) had a BMI indicating they were overweight or obese; just under a quarter of these fell into the obese category. A larger proportion of males (68%) than females (55%) were overweight or obese (ABS 2009a). Over one-third (37%) of the population was considered to be of a normal weight; that is neither underweight, overweight nor obese.

Based on measures of waist circumference in the NHS, over half of adults were at increased risk, or substantially increased risk or ill health (AIHW 2009d). As with BMI, this classification may not be suitable for use with people aged 18 years or below, the elderly or with all ethnic groups.

Sex and age

The measured results from the 2007–08 NHS showed that 42% of males aged over 18 years were overweight but not obese and a further 26% were obese. For females, the equivalent figures were 31% overweight and 24% obese. The proportion of the population who were overweight or obese increased steadily with age, from 38% for those aged 18–24 years to 75% for those aged 65–74 years. After this, the proportion declined with 65% of those aged 75 years or over being overweight or obese. For both males and females the 65–74 years age group had the highest proportion of people who were overweight or obese (Figure 2.16). Seventy-nine per cent of males were in this category and 71% of females.

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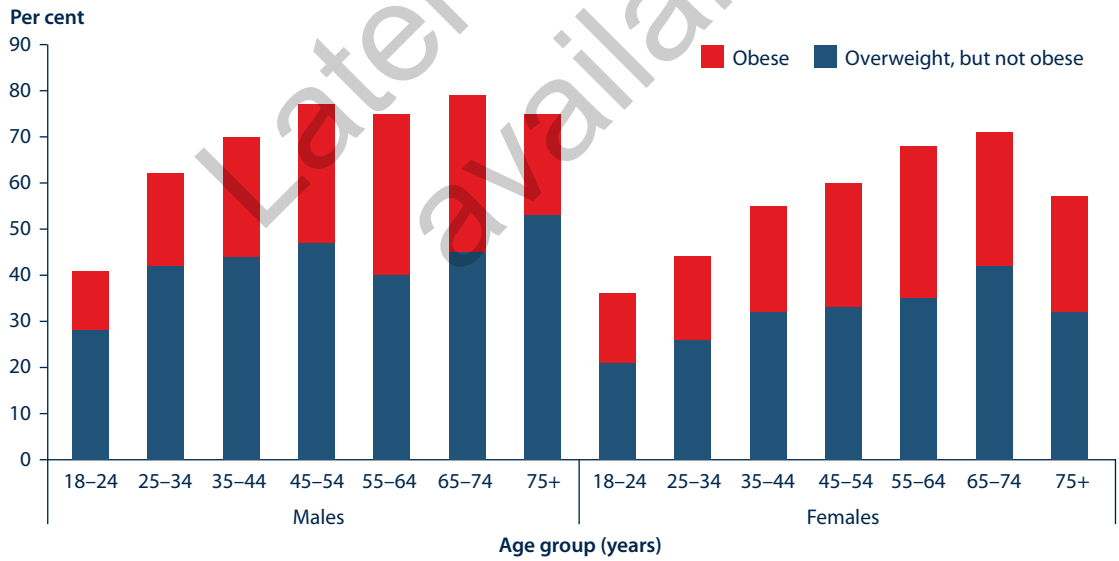


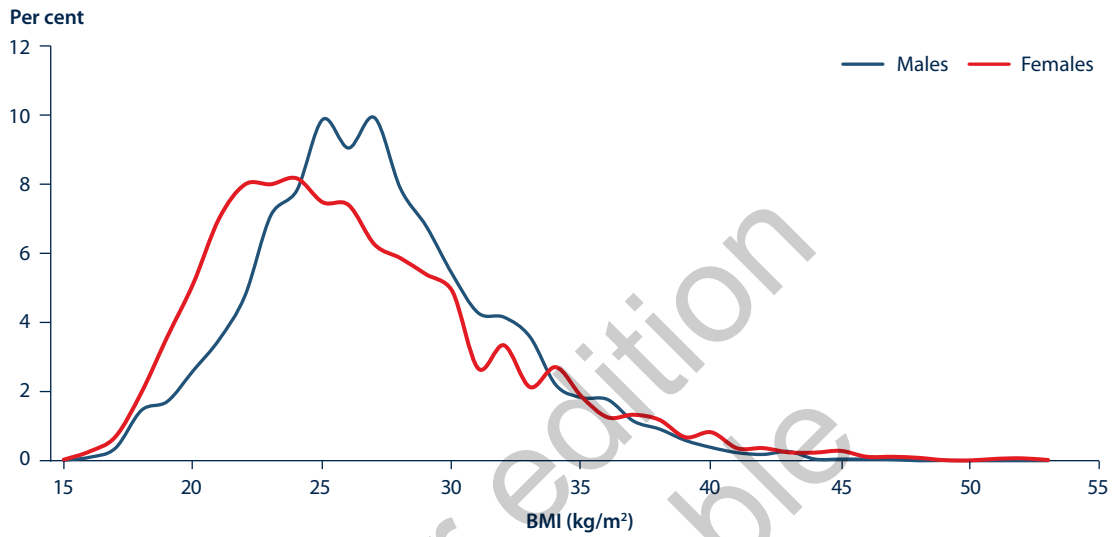
Notes

1. Based on measured data.
2. Overweight is defined as a person having a body mass index of 25 or more. Obese is defined as a person having a body mass index of 30 or more.

Source: AIHW analysis of ABS 2007–08 National Health Survey.

Figure 2.16: Percentage of adults who are overweight or obese, by sex and age, 2007–08

The full BMI distribution, in Figure 2.17, shows a large proportion of people who were not overweight had a BMI of 24 and therefore was at risk of moving to the overweight category. Additionally, 16% of those overweight (but not obese) had a BMI of 29, putting them close to the obesity category. In contrast, 19% of people had a BMI of 30, and were therefore on the borderline of being classified as overweight rather than obese.



Note: Missing values were excluded from the numerator and the denominator.

Source: AIHW analysis of ABS 2007-08 National Health Survey.

Figure 2.17: Distribution of measured body mass index, people aged 15 years and over, by sex, 2007–08

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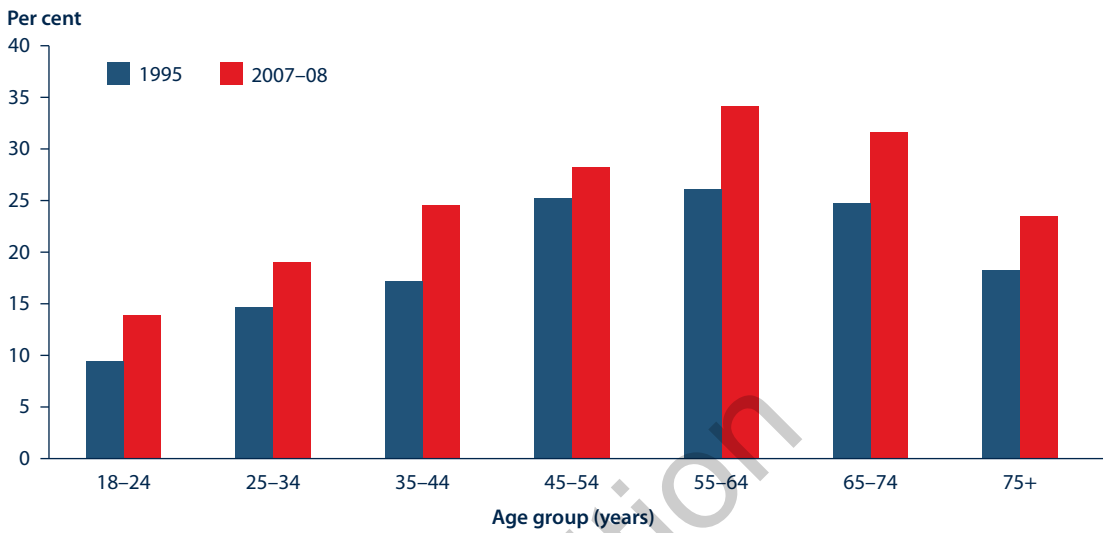
Trends

Levels of overweight and obesity were still increasing in Australia as recently as 2007–08. When measured results from the 1995 National Nutrition Survey (NNS) and the 2007–08 NHS are compared they indicate an increase in the percentage of people aged 18 years and over who were classified as overweight or obese. The proportion of males classified as overweight or obese rose from 64% in 1995 to 68% in 2007–08. For females, the proportion rose from 49% to 55% over the same period.

Overall, the proportion of people classified as obese in the population aged 18 years and over increased by 6 percentage points from 1995 to 2007–08. The most marked increases in obesity during this period were among those aged 35–44 years and 55–64 years where the proportion who were considered obese increased by more than 7 percentage points in both groups (Figure 2.18).



Note: Data from the ABS 1995 National Nutrition Survey and ABS 2007–08 National Health Survey

Source: ABS 2009a.

Figure 2.18: Prevalence of measured obesity, by age, 1995 to 2007–08

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Aboriginal and Torres Strait Islander people

Self-reported data from the 2004–05 NATSIHS indicate that 28% of Indigenous Australians aged 15 years and over were overweight but not obese and a further 29% were obese (ABS 2006a). After adjusting for differences in the age structures of the Indigenous and non-Indigenous populations, Indigenous Australians had lower rates of being overweight (but not obese) than other Australians but obesity rates that were almost twice as high.

Remoteness

Results from the 2007–08 NHS indicate that both males and females aged 15 years and over in *Outer regional and remote* areas had higher rates of obesity (32% of males and 27% of females) than their counterparts in *Major cities* and *Inner regional* areas. Twenty-six per cent of males and 26% of females in *Inner regional* areas were obese. In *Major cities*, 23% of males and 21% of females were obese.

The highest rates of being overweight (but not obese) were recorded for both males and females in *Inner regional* areas with 43% of males and 35% of females in this category.

Socioeconomic group

Results from the 2007–08 NHS for those aged 15 years and over, show that obesity increases with decreasing socioeconomic position. Thirty-three per cent of males and 31% of females in the lowest socioeconomic group were obese. The comparable figures for the highest socioeconomic group were 19% for males and 15% for females.

The relationship between socioeconomic group and being overweight was less clear. For males, the lowest proportion who were overweight but not obese (34%) was recorded for the lowest socioeconomic group while in the highest socioeconomic group, 44% of males were overweight.

Among females, the highest proportion who were overweight but not obese (34%) was found in the middle-ranked group with the lowest proportion (28%) recorded for the highest socioeconomic group (AIHW 2009d).

Burden of overweight and obesity

About 8% of the burden of disease and injury in Australia in 2003 was attributed to high body mass (defined as having a BMI greater than 21), placing it third of the 14 risk factors studied (Begg et al. 2007). After high blood pressure, high blood cholesterol and physical inactivity, it was the fourth largest contributor to the burden of CVD, accounting for 20% (Begg et al. 2007).

International comparisons

The proportion of the Australian population aged 18 years and over that was obese in 2007–08 (25%) was similar to the proportions in the United Kingdom (24%), Canada (24%) and New Zealand (26%). For those countries with measured data for 2007 or 2008, the proportion of obese people varied from 3% in Japan to 34% in the United States.

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Depression

What is depression?

Depression is a mental illness within the affective spectrum which affects many Australians. Depression is a mood disorder which is characterised by prolonged feelings of sadness, hopelessness and inadequacy. Symptoms can include a loss of interest or pleasure in activities, feelings of worthlessness or inappropriate guilt, disturbed sleep or appetite, low energy and poor concentration (ICD-10-AM). If these problems become chronic or recurrent they can impair an individual's ability to take care of their everyday responsibilities. At its most severe, depression can lead to suicide (WHO 2009).

How many people have depression?

Depression is the most commonly diagnosed mental illness in Australia (AIHW 2008b). A depressive episode is defined as a 'state of gloom, dependency or sadness lasting at least two weeks' (ABS 2008). The 2007 National Survey of Mental Health and Wellbeing (ABS 2008) found that of all people aged 16–85 years (16 million), almost 1.9 million (12%) had experienced a depressive episode at some stage in their lives and 650,000 (4%) had experienced a depressive episode in the previous 12 months. These results equated to just over 1 in 25 Australians having experienced a depressive episode in the preceding 12 months (ABS 2008).

Females were more likely to experience depression than males. Overall, 9% of males (694,000 people) and 15% of females (1,150,700 people) aged 18–85 years had experienced a depressive episode at some point in their lives. Females were also more likely than males to have experienced a depressive episode in the previous 12 months (5% of females compared with 3% of males).

Burden of depression

Anxiety and depression were estimated to be responsible for 7% of the total burden of disease in 2003. The burden from anxiety and depression was twice as high for females as for males (Begg et al. 2007).

Depression and CVD

There is evidence that depression can independently lead to medical conditions such as heart disease without any intermediary behavioural effects such as increased rates of smoking or poor diet. The strength of the association between depression and CVD is similar to that of other standard risk factors, such as high cholesterol (Bunker et al. 2003). While depression is a risk factor for CVD, CVD in turn is also a strong risk factor for depression (Clarke & Currie 2009).

Depression can also influence other risk factors for CVD and often co-exists with them. For example, depression is a major barrier to the adoption of healthy lifestyle behaviours (Hayes 2006). Further, people with depression are more likely to smoke and be physically inactive, compared with those without depression (Bunker et al. 2003).

A 2003 review by the National Heart Foundation Australia (NHFA) concluded that depression, social isolation and lack of quality social support were independent risk factors for the onset and prognosis of some CVD. In particular, this was true for the two leading causes of death of Australians—coronary heart disease and stroke (Clarke & Currie 2009; Hayes 2006; Jonas & Mussolino 2000).

Australian and international recommendations state that patients with coronary heart disease should be assessed for depression and treated where necessary and patients diagnosed with depression should be assessed for other coronary heart disease risk factors (Bunker et al. 2003; Mosca et al. 2007).

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Protective factors

What is a protective factor?

Risk factors are defined as those factors which make it more likely that a person will develop a health problem or disorder. Protective factors on the other hand, reduce the likelihood of a person suffering a disease and/or improve their capacity to respond should a disease occur. A protective factor can therefore be described as one that contributes positively to an individual's health and wellbeing. Factors which protect against CVD can vary from levels of HDL in the bloodstream sufficient to help protect against arterial plaque formation to behavioural factors, such as getting regular physical activity or eating plenty of fresh fruit and vegetables. Socioeconomic protective factors, such as having a strong social support network or an adequate level of income, are also important. This section discusses the protective effects of desirable levels of cholesterol, physical activity and alcohol use on CVD.

Sufficient physical activity

Physical activity is a protective factor for good health. Regular moderate to vigorous physical activity helps protect against a range of diseases and conditions, including heart disease, Type 2 diabetes and some forms of cancer (DoHA 2010). Recognising this, the NHMRC recommends at least 30 minutes of moderate–intensity physical activity on most, preferably all days to reduce the risk of these conditions (NHMRC 2001b).

Low alcohol consumption

It may be that some health benefits can be attributed to the moderate use of alcohol, but the harmful effects of overuse in the community far outweigh these benefits and the consumption of alcohol should never be encouraged on health grounds (Beaglehole & Bonita 2009). Low levels of alcohol consumption are thought to be protective against CVD because they raise the level of HDL cholesterol in the blood. Alcohol can also have a mild anti-coagulating effect (NHMRC 2009). Long-term, low to moderate alcohol consumption (one to two drinks per day for men and less than one per day for women), is thought to reduce the risk of stroke, CHD and hypoglycaemia (low blood glucose levels); mostly in men aged over 40 and post-menopausal women (AIHW 2008b, 2008c; Mann et al. 2004; Single et al. 2000; WHO 2003).

High HDL cholesterol

As discussed in the section on high blood cholesterol, LDL and HDL are of primary interest in the consideration of CVD, especially for CHD and ischaemic stroke. High levels of HDL have a protective effect against both diseases by helping to reduce atherosclerosis. HDL is one of five lipoprotein groups that allow cholesterol to be transported in the blood and it is suggested that HDL removes cholesterol from plaques and transports it back to the liver for excretion or re-use.

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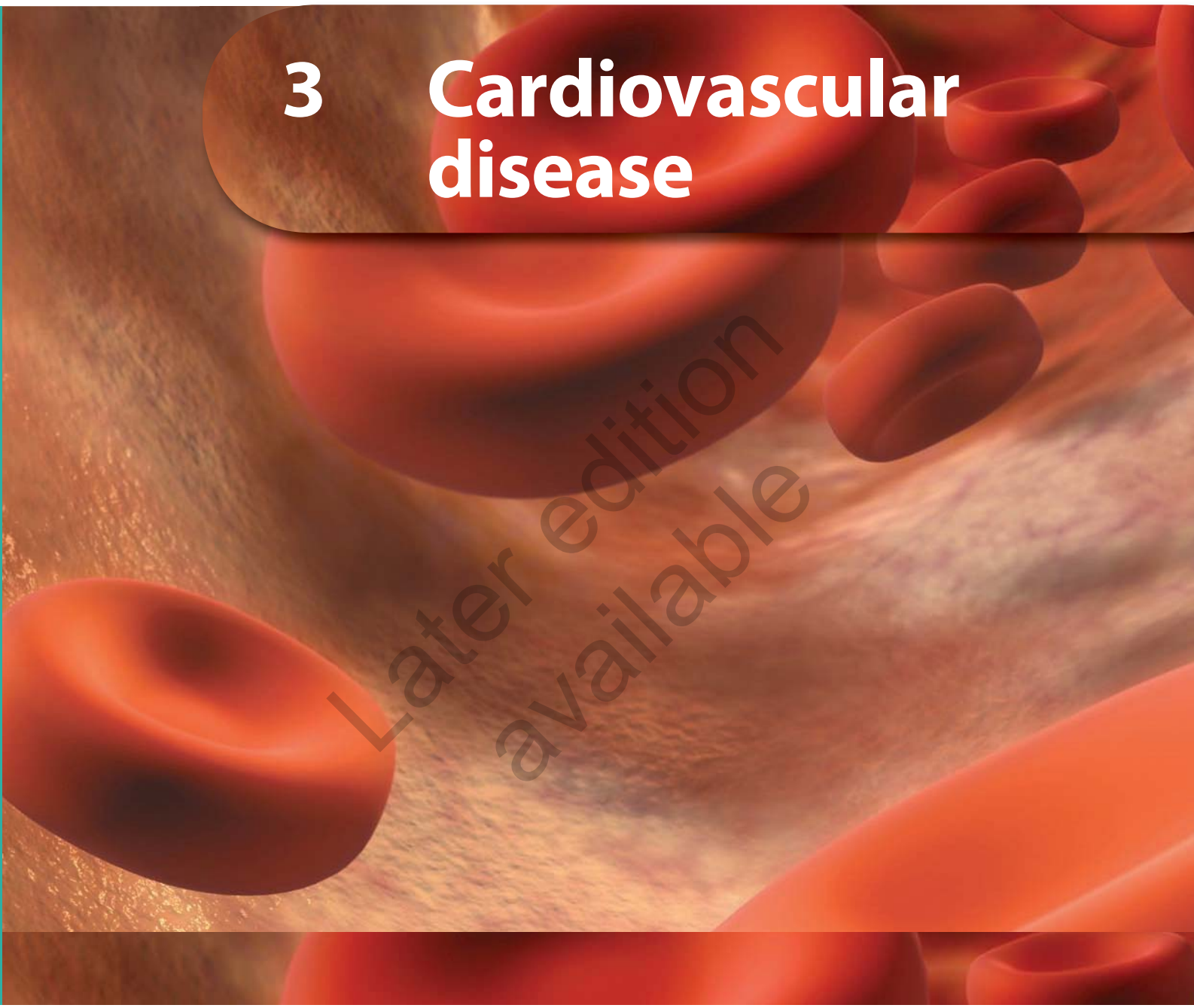

3 Cardiovascular disease

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3 Cardiovascular disease

This chapter examines the levels of cardiovascular disease (CVD) in the Australian community by focusing on CVD-related prevalence, hospitalisations and deaths. Similar chapters follow for those conditions prominent in the overall make-up of CVD in Australia.

How many Australians have cardiovascular disease?

Based on self-reports from the 2007–08 National Health Survey (NHS), an estimated 3.4 million Australians (17% of the population) had one or more long-term cardiovascular diseases (AIHW 2010a). Similarly, estimates from the 2007 National Survey of Mental Health and Wellbeing (NSMHWB) show that 3.5 million Australians aged 16-85 years had a chronic CVD condition. The NHS ranked CVD fourth in prevalence after diseases of the eye and adnexa, the musculoskeletal system, connective tissue and the respiratory system.

It should be noted that the NHS is the major source of prevalence information used in this report but it did not include within its scope people in institutionalised care facilities, such as hospitals and nursing homes, thereby excluding an elderly sector of the population where high levels of CVD are expected. As a result, the NHS undercounts the number of people with CVD, particularly elderly people, and care should be taken when interpreting the results. It is also important to note that the NHS and the NSMHWB are self-reported surveys, further emphasising the need to treat the results with caution because some respondents may not have known or been able to accurately report the state of their cardiovascular health at the time the survey was conducted, while others may tend to over-report some conditions. It should also be noted that changes to methods between the 2001 and 2004–05 National Health Surveys mean that reliable trend data on CVD prevalence are not available.

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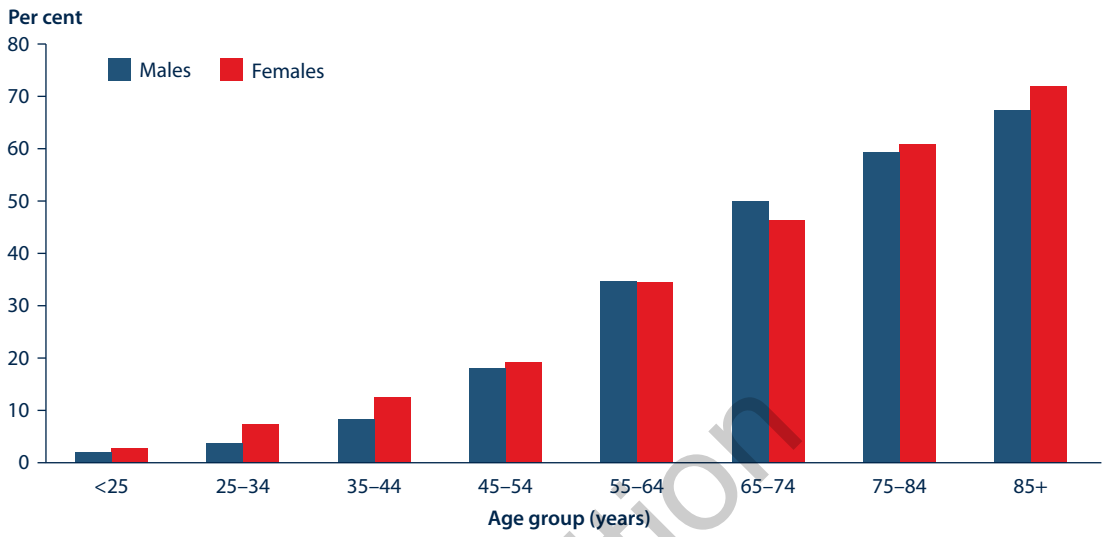
Sex and age

After adjusting the results from the self-reported 2007–08 NHS for age, a slightly higher percentage of females (16%) than males (15%) were estimated to have CVD. CVD occurs more commonly among the elderly with 62% of those aged 75 years and over estimated to have the condition. Among those aged 45–54 years, 19% had CVD and the estimate was 5% for those aged less than 45 years (Figure 3.1).



Source: AIHW analysis of ABS 2007–08 National Health Survey.

Figure 3.1: Cardiovascular disease prevalence, by age and sex, 2007–08

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Aboriginal and Torres Strait Islander people

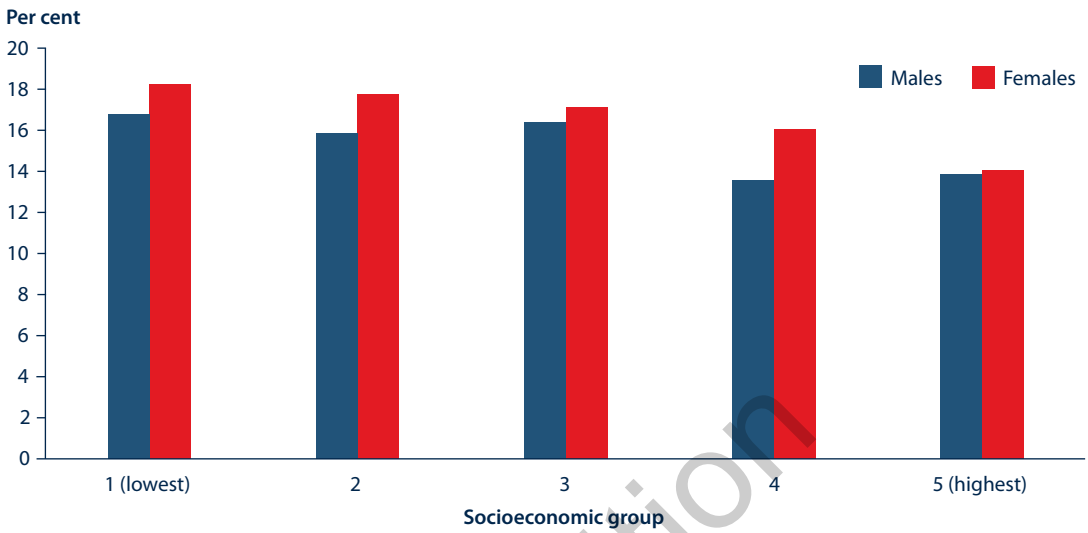
According to self-reported data from the 2004–05 National Aboriginal and Torres Strait Islander Heath Survey (NATSIHS), an estimated 55,900 Indigenous people (12%) had a long-term cardiovascular condition. Overall, 14% of the Indigenous respondents in remote areas had a cardiovascular condition, while for non-remote areas the proportion was 11%. After adjusting for differences in the age structure of the Indigenous and non-Indigenous populations, Aboriginal and Torres Strait Islander people were 1.3 times as likely to have CVD as non-Indigenous Australians. The differences between the two populations are starkly illustrated by observing that the pattern of CVD prevalence among Indigenous Australians is equivalent to that of non-Indigenous Australians who are 10 years older (AIHW: Penm 2008).

Remoteness

Self-reported data from the 2007–08 NHS show that age-standardised CVD prevalence rates differed across the areas of *Major cities*, *Inner regional* and *Other regions*. (In order to overcome small sample numbers, *Other regions* is a grouping of *Outer regional*, *Remote* and *Very remote* areas.) Prevalence rates for CVD were lowest in *Major cities* (15%) and highest in *Other regions* (18%). Prevalence rates were also higher for females than males for each remoteness area. Refer to Appendix A for information about reporting data by remoteness areas.

Socioeconomic group

In 2007–08, Australians in the lowest socioeconomic groups experienced a higher prevalence of CVD than those in the highest socioeconomic groups. Rates were higher for females than males across all groups (Figure 3.2). See Appendix A for an explanation of how socioeconomic group is derived from the Socio-Economic Indexes for Areas (SEIFA) index.



Note: Age-standardised to the 2001 Australian population.

Source: ABS 2009a.

Figure 3.2: Cardiovascular disease prevalence, by socioeconomic group and sex, 2007–08

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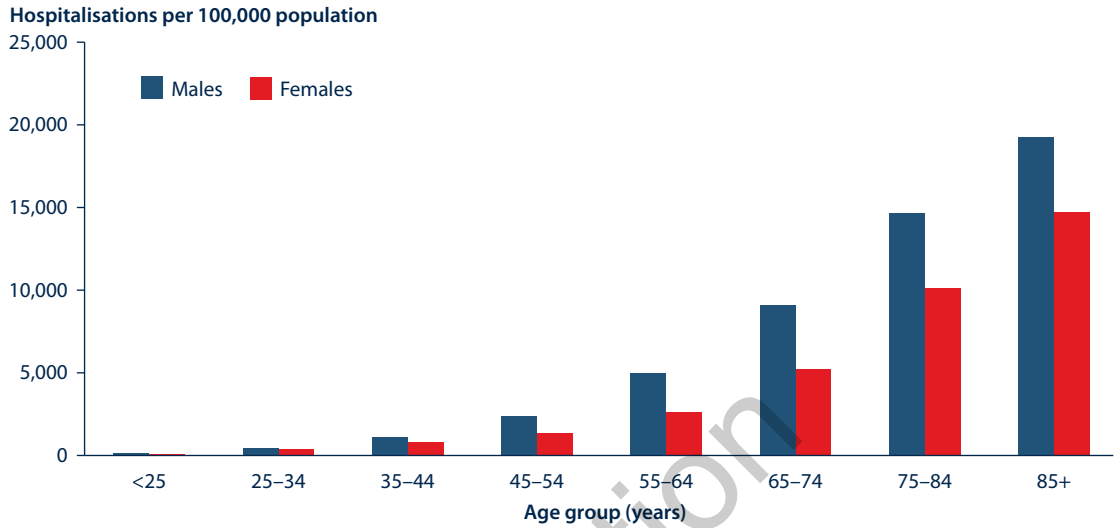
Hospitalisations

In 2007–08, around one in 16 hospitalisations had a principal diagnosis of CVD and in a further one in 10 admissions CVD was recorded as an additional diagnosis. The principal diagnosis is that which is listed in hospital records to describe the problem that was chiefly responsible for the patient's episode of care. An additional diagnosis is a condition or complaint, either co-existing with the principal diagnosis or arising during the episode of admitted patient care (Appendix A). Note that hospitalisations data in this report are based on 'episodes of care' rather than the number of people hospitalised with a condition (Appendix A).

Of all hospitalisations for CVD, 34% were for CHD, followed by heart failure and cardiomyopathy (10%), stroke (7%), peripheral vascular disease (PVD) (5%), transient ischaemic attack (3%), hypertensive heart disease (2%) and rheumatic heart disease (RHD) (1%). Transient ischaemic attack (TIA) is a condition which produces temporary stroke-like symptoms that are important predictors of stroke. In order to maintain comparability with the most recent edition (2004) of this report, TIA is not included in the stroke classification unless indicated otherwise.

Sex and age

The rate of CVD hospitalisations increases rapidly with age and in 2007–08 almost eight in 10 (78%) were for people aged 55 years and over. Males recorded higher rates of CVD hospitalisations than females in all age groups (Figure 3.3). The age-standardised rate for males (2,599 per 100,000 population) was 1.6 times as high as that for females (1,651 per 100,000 population).



Source: AIHW National Hospital Morbidity Database.

Figure 3.3: Cardiovascular disease hospitalisation rates, principal diagnosis, by age and sex, 2007–08

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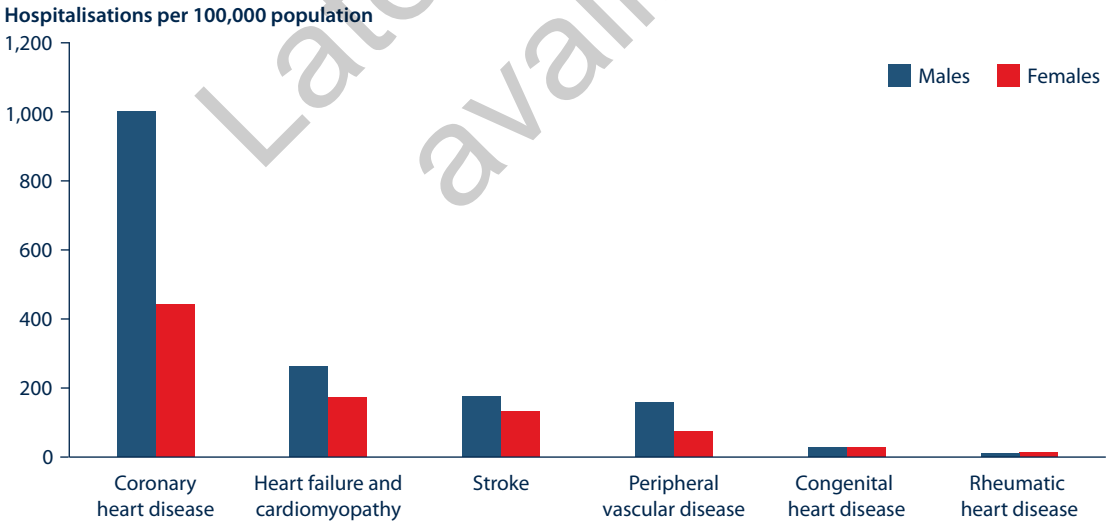
After standardising for age differences, male hospitalisation rates were higher than female rates for all major CVDs, with the male rate for CHD nearly twice as high as the female rate (Figure 3.4).



Note: Age-standardised to the 2001 Australian population.

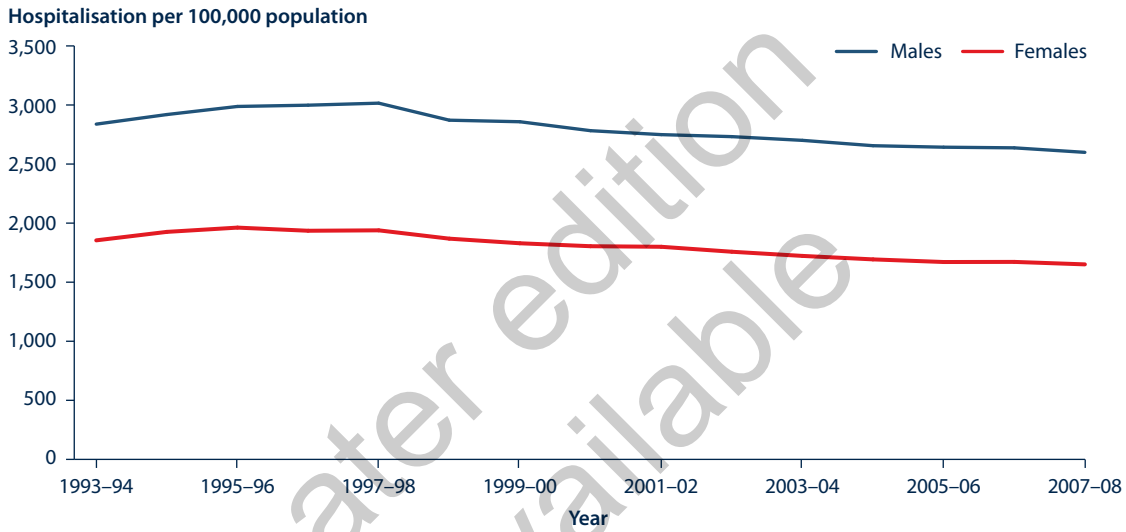
Source: AIHW National Hospital Morbidity Database.

Figure 3.4: Cardiovascular disease hospitalisation rates, principal diagnosis, by disease type and sex, 2007–08

Trends

The age-standardised rate of hospitalisations with CVD has declined over the past 15 years from the 1993–94 rate of 2,312 per 100,000 population to 2,099 in 2007–08. The highest rates (2,441 per 100,000) were reached in 1995–96 and 1997–98. Male rates were consistently higher than those for females over the same period, with both showing similar declines (Figure 3.5).

However, despite the fall in rates, the rate of many hospital procedures performed to diagnose and treat people with CVD has continued to rise, meaning people are now less likely to be hospitalised with CVD but more likely to have a procedure when they are (see 'Chapter 11 Health services for cardiovascular disease' for more information on procedures).



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Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 3.5: Cardiovascular disease hospitalisation rates, principal diagnosis, by sex, 1993–94 to 2007–08

Health inequalities

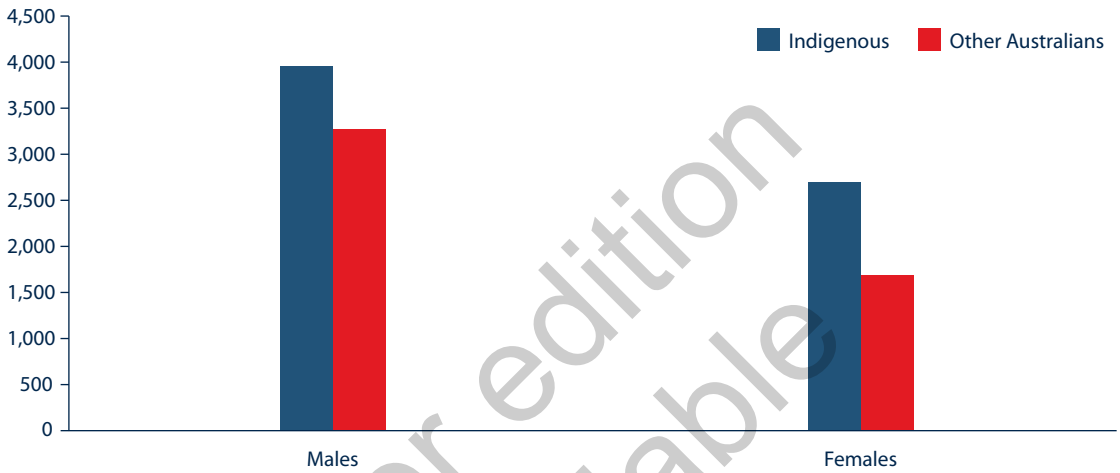
The following section looks at CVD hospitalisations in subgroups of interest in the Australian population. Hospitalisation rates are presented for Indigenous and other Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2007–08, there were 8,547 hospitalisations with a principal diagnosis of CVD where the patient was identified as Indigenous (in the jurisdictions with adequate Indigenous identification). After adjusting for age, hospitalisation rates with CVD among Aboriginal and Torres Strait Islander people were almost twice as high as those for other Australians (3,588 per 100,000 population compared with 2,160 per 100,000, respectively).

The rate of CVD hospitalisations for Indigenous males was 1.5 times as high as that for other Australian males. Similarly, the Indigenous female rate was 1.9 times as high as the other Australian female rate (Figure 3.6).

It is important to note that identification of Indigenous status in hospital data is only considered reliable in New South Wales, Victoria, Queensland, Western Australia, South Australia and the public hospitals of the Northern Territory and that all analyses of Indigenous hospitalisations in this report are based on these states and territories only (AIHW 2009a). The exclusion of some jurisdictions from the analyses will lead to an undercount of the total number of hospitalisations of Aboriginal and Torres Strait Islander people. For further information refer to 'Reporting of Indigenous data' in Appendix A.



Notes

1. Age-standardised to the 2001 Australian population.
2. Data only from hospitals in NSW, Vic, Qld, WA, SA; and for NT public hospitals.

Source: AIHW National Hospital Morbidity Database.

Figure 3.6 Cardiovascular disease hospitalisation rates, principal diagnosis, by sex and Indigenous status, 2007–08

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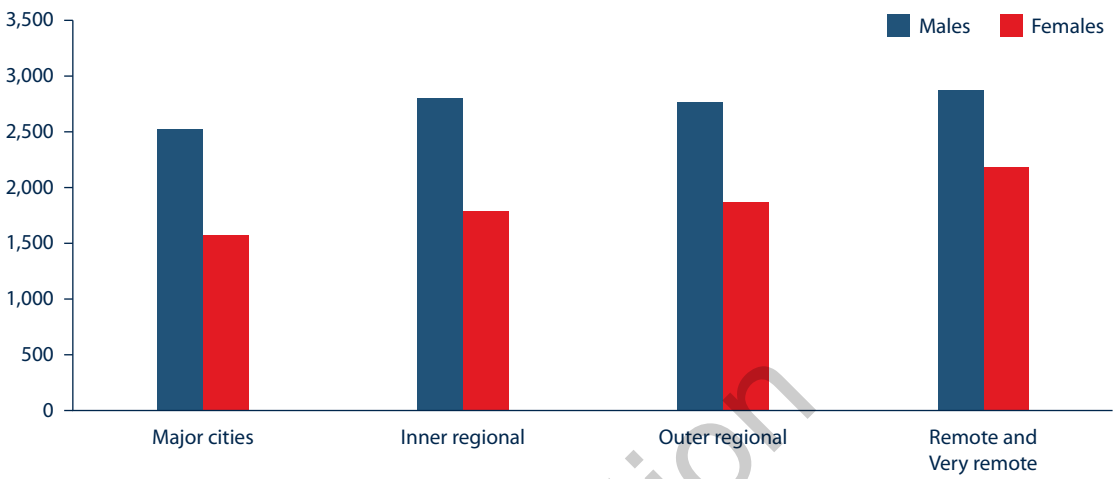
Remoteness

In 2007–08, age-standardised rates of hospitalisations with a principal diagnosis of CVD differed across remoteness areas. The rate for *Major cities* was lowest at 2,019 hospitalisations per 100,000 population and highest for *Remote and very remote* areas at 2,557 per 100,000.

The male rate of CVD hospitalisation was higher than the female rate in all remoteness areas. Rates for both males and females were highest in *Remote and very remote* areas, in part because of the high proportion of Indigenous people living in these areas (Figure 3.7).

It is also important to note that 20% of people who are admitted to hospital with a principal diagnosis of CVD in *Remote* areas are transferred to another hospital. In *Very remote* areas this rises to 23%, whereas for those admitted to *Inner regional* or *Major city* hospitals, 16% and 11% are transferred. The likely explanation for this is that procedures such as angiograms can only be conducted in hospitals located in the *Major city* and *Inner regional* areas. Transfers between hospitals will increase hospitalisation rates because a separate hospitalisation is counted each time a patient is discharged from a hospital.



Notes

1. Age-standardised to the 2001 Australian population.
2. Based on area of usual residence of patient.

Source: AIHW National Hospital Morbidity Database.

Figure 3.7: Cardiovascular disease hospitalisation rates, principal diagnosis, by remoteness and sex, 2007–08

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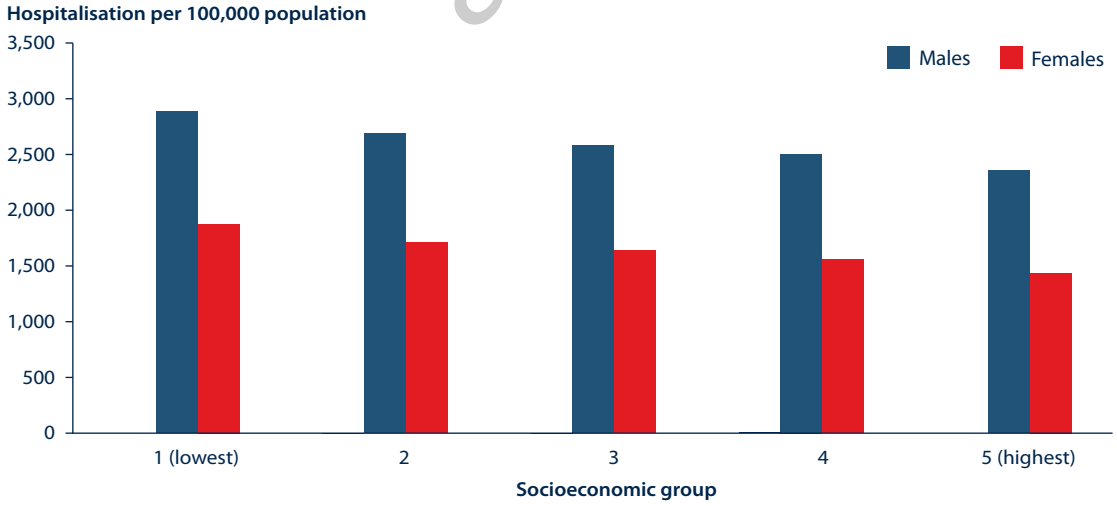
Socioeconomic group

In 2007–08, the age-standardised hospitalisation rates with a principal diagnosis of CVD were highest among those in the lowest socioeconomic group (2,358 per 100,000 population) and lowest for those in the highest socioeconomic group (1,859 per 100,000 population). CVD hospitalisation rates were also higher for males than females in each group (Figure 3.8).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 3.8: Cardiovascular disease hospitalisation rates, principal diagnosis, by socioeconomic group and sex, 2007–08

Length of stay in hospital

The length of time people spend in hospital for CVD is decreasing. In 2007–08, almost half (46%) of people admitted to hospital with CVD were discharged the same day. In 1993–94, the comparable figure was about a quarter (27%). There are likely to be a number of reasons for the increase in 'same-day' hospitalisations, including changes to treatment, diagnosis and practice, and the transfer of cardiac patients to hospitals able to offer more specialised care.

Among those hospitalised with CVD for one night or more, the average length of stay declined steadily from 9.6 days in 1993–94 to 7.9 days in 2007–08. In 2007–08, 70% of all stays were for 4 days or less, while in 1993–94 that figure was 55%.

Of those hospitalised with CVD in 2007–08, patients with stroke tended to stay longest, an average of 12.3 days, followed by patients with congenital heart disease (11 days) peripheral vascular disease (10.8 days), and CHD (6.3 days).

Deaths in hospital

Between 1993–94 and 2007–08 the proportion of hospitalisations with CVD that ended in death underwent a steady decline, from 4.9% to 3.6%.

Deaths

Cardiovascular disease is still Australia's biggest killer

In 2008, CVD was the cause of 48,456 (34%) of all deaths—responsible for more deaths than any other disease group. CVD was followed as a cause of death by cancer (29%), diseases of the respiratory system (8%) and external causes (6%), with mental and behavioural problems and diseases of the nervous and endocrine systems each accounting for a further 4% (Figure 3.9).

It should be noted that although Figure 3.9 presents deaths data relating to 2008, corresponding detailed data were not available when the further analyses in the report were undertaken and subsequently 2007 data are used in the remainder of the report.

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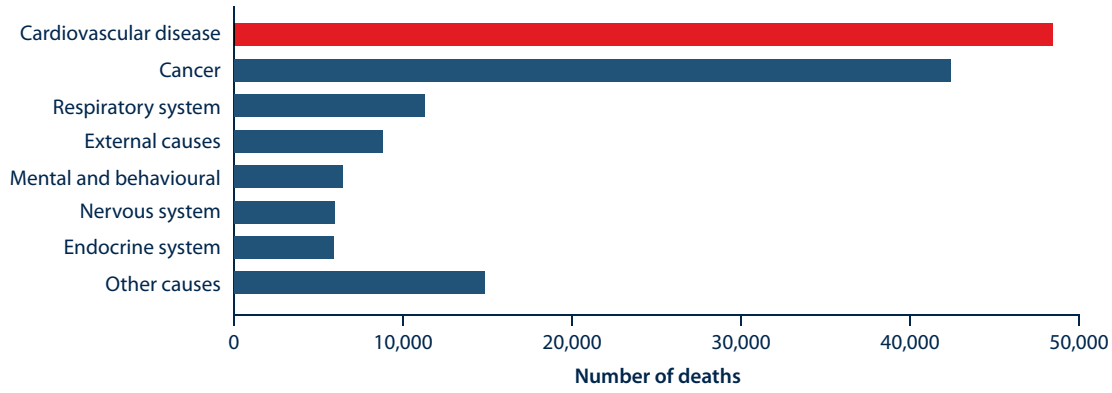


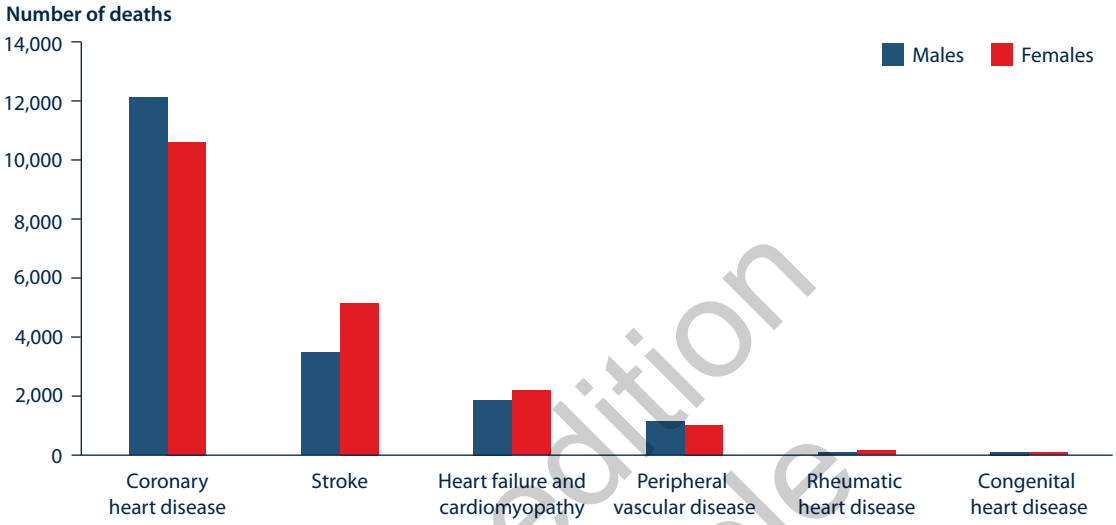
Note: Based on underlying cause of death.

Source: ABS: Causes of Death, Australia, 2008

Figure 3.9: Deaths in Australia, by disease group, 2008

Major causes of cardiovascular death

CHD is by far the main cause of cardiovascular death (49%), followed by stroke (18%). Other major causes of death are heart failure and cardiomyopathy and PVD (Figure 3.10).



Note: Based on underlying cause of death.

Source: AIHW National Mortality Database.

Figure 3.10: Major causes of cardiovascular disease death, 2007

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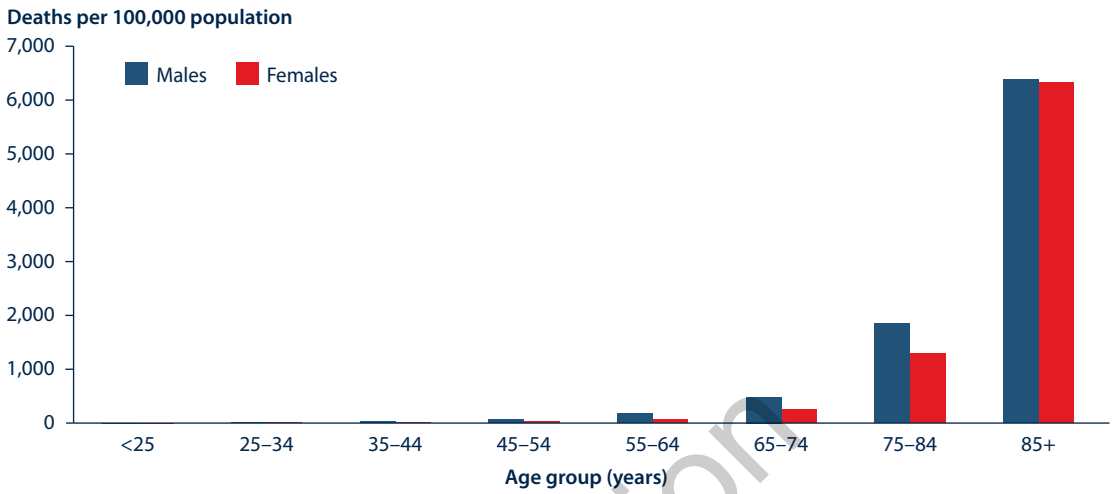
Sex and age

Age-specific CVD death rates increase sharply with age. In 2007, there was a four-fold increase between the 65–74 and 75–84 year age groups and then a similar increase for those aged 85 years and over. Male rates were higher than female rates across all age groups, with males aged 45–64 years experiencing death rates almost 3 times as high as those for females of the same age (Figure 3.11).

A similar number of males and females die from CVD even though male CVD death rates are much higher than female rates. The reason for this is that a higher proportion of the female population lives to older ages and around 78% of CVD deaths occur among those aged 75 years and over.



Source: AIHW National Mortality Database.

Figure 3.11: Cardiovascular disease death rates, by age and sex, 2007

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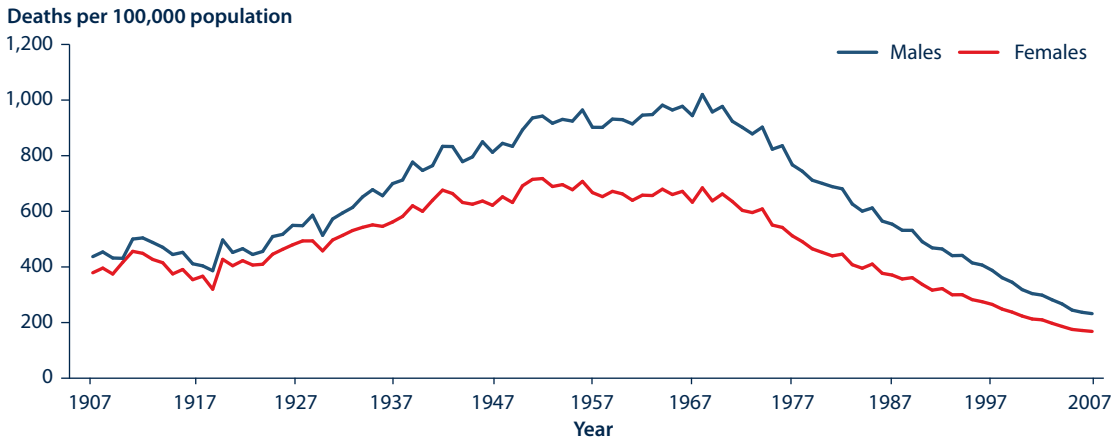
Trends

CVD death rates have fallen dramatically since their peak in the late 1960s when CVD was responsible for around 60,000 deaths annually.

Male CVD death rates (1,020 per 100,000) peaked higher than female rates (718 per 100,000) and began to decline a few years later. Between 1987 and 2007, male rates fell at an average of 4.2% per annum to 232 deaths per 100,000 population and female rates fell at 3.8% per annum to 170 deaths per 100,000 (Figure 3.12).

Much of the decline in CVD death rates can be attributed to improvements in the prevention, detection and management of CVD that has occurred in the past 60 years (AIHW 2009c).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database, AIHW GRIM (General Record of the Incidence of Mortality) Books.

Figure 3.12: Cardiovascular disease death rates, by sex, 1907–2007

If CVD death rates had remained at their 1968 peak, there would have been 195,500 deaths for CVD in 2007—more than the number of deaths from all causes in that year. The actual number of CVD deaths that occurred in 2007 was 46,626 (AIHW 2009b).

Health inequalities

The following section looks at CVD deaths in subgroups of interest in the Australian population. Death rates are presented for Indigenous and non-Indigenous Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

For the period 2005–2007, there were 1,810 deaths with an underlying cause of CVD recorded for Indigenous Australians in jurisdictions with adequate Indigenous identification. The age-adjusted CVD death rate for Indigenous Australians was 1.4 times as high as that for non-Indigenous Australians (404 per 100,000 population compared to 284 for non-Indigenous Australians). Indigenous males and females had CVD death rates 1.4 times and 1.5 times as high respectively, as their non-Indigenous counterparts (Figure 3.13).

Indigenous identification in deaths data is considered of sufficient quality for national reporting for New South Wales, Queensland, Western Australia, South Australia and the Northern Territory only. For further information refer to 'Reporting of Indigenous data' in Appendix A.

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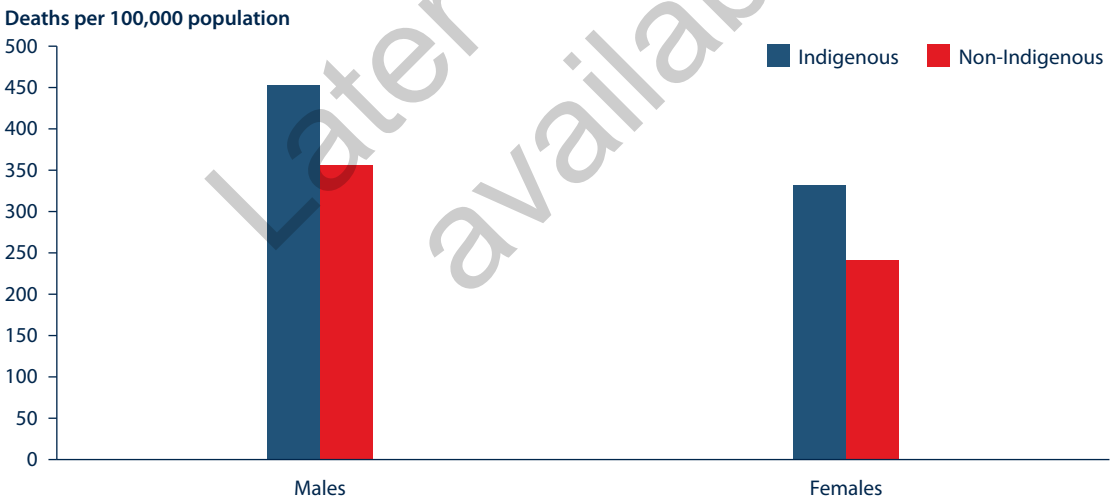


Notes

1. Age-standardised to the 2001 Australian population.
2. Data from NSW, Qld, WA, SA and NT only.

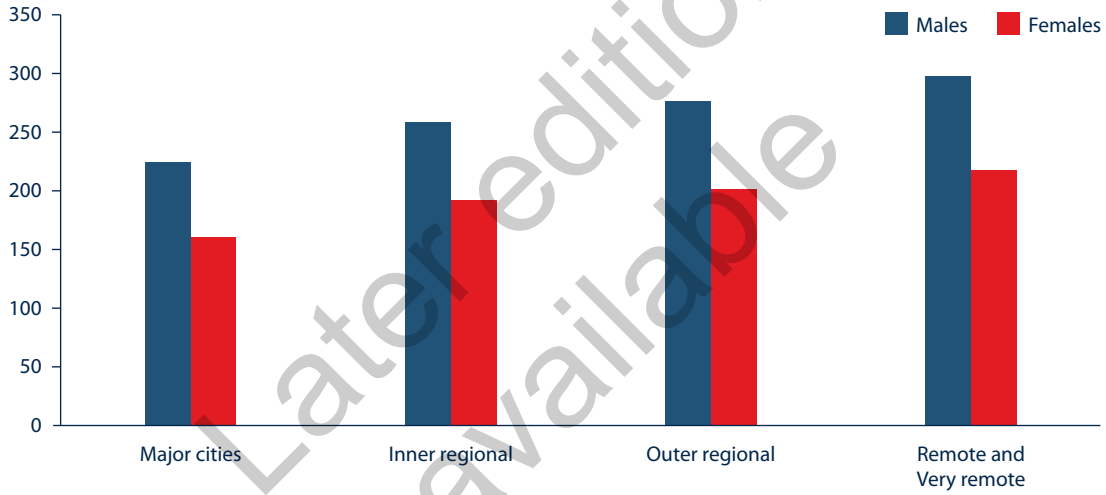
Source: AIHW National Mortality Database.

Figure 3.13: Cardiovascular disease death rates, by sex and Indigenous status, 2005–2007

Remoteness

In 2005–2007, the overall CVD death rate in *Remote and very remote* areas (262 deaths per 100,000 population) was 1.4 times as high as that in *Major cities* (190 per 100,000 population). Male death rates were about 1.4 times as high as those for females across all remoteness areas and were highest in *Remote and very remote* areas (298 deaths per 100,000 population). For females, the highest CVD death rates were also recorded in *Remote and very remote* areas (218 deaths per 100,000) (Figure 3.14).

Age-adjusted CVD death rates are generally higher among Indigenous Australians than non-Indigenous Australians and the higher rates reported for remote areas may reflect the fact that a higher proportion of Indigenous people live in these areas (AIHW 2005). In addition, people in remote areas often belong to lower socioeconomic groups and have lower levels of access to health services, both of which are related to higher CVD death rates.



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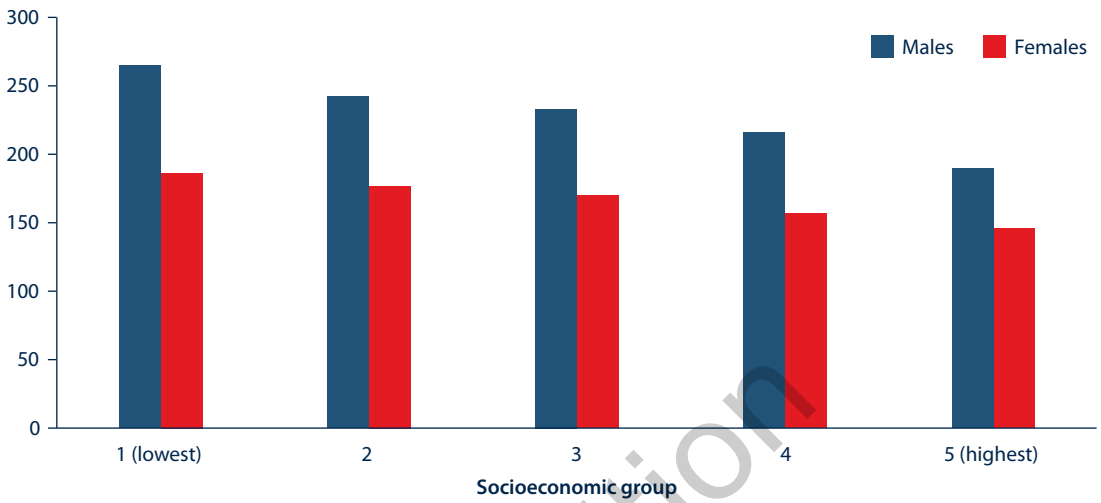
Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Figure 3.14: Cardiovascular disease death rates, by remoteness and sex, 2005–2007

Socioeconomic group

In 2007, for both males and females, the age-adjusted CVD death rate was highest among those in the lowest socioeconomic groups and lowest for those living in the highest socioeconomic groups. For all socioeconomic groups male rates were about 1.4 times as high as female rates (Figure 3.15).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Figure 3.15: Cardiovascular disease death rates, by socioeconomic group and sex, 2007

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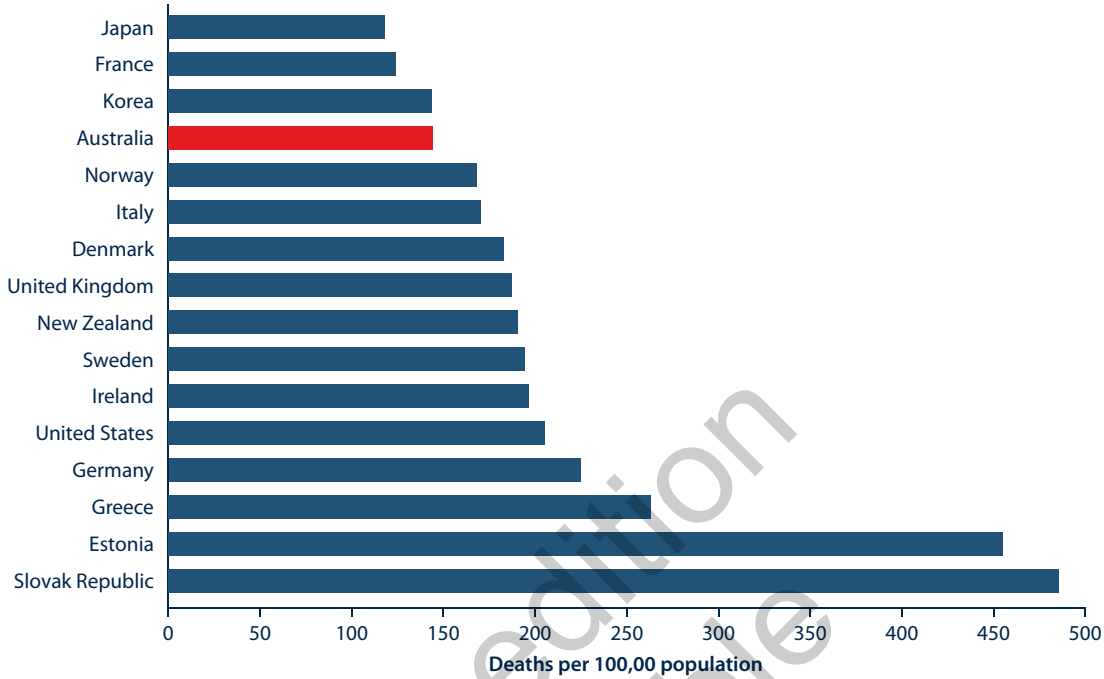
International comparisons

The OECD publishes CVD mortality rates for a range of its member countries. The latest available data for Australia were for 2006 so, in this report, 2006 data were used to make comparisons between countries.

Of those OECD countries for which comparable data were available, the Slovak Republic had the highest CVD death rate and Japan had the lowest. Australia's CVD death rate was low compared with many OECD countries. It was about 1.4 times as high as that of Japan but less than one-third that of the Slovak Republic (Figure 3.16).

Since 1980 most OECD countries have witnessed a substantial reduction in CVD death rates with Australia having one of the greatest decreases, from 444 deaths per 100,000 population in 1980 to 144 per 100,000 in 2006.



Notes

1. Rates have been age-standardised to the 1980 OECD population.
2. Rates for the Slovak Republic and the United States are for 2005.
3. 2006 data have been used where possible for comparability between countries.

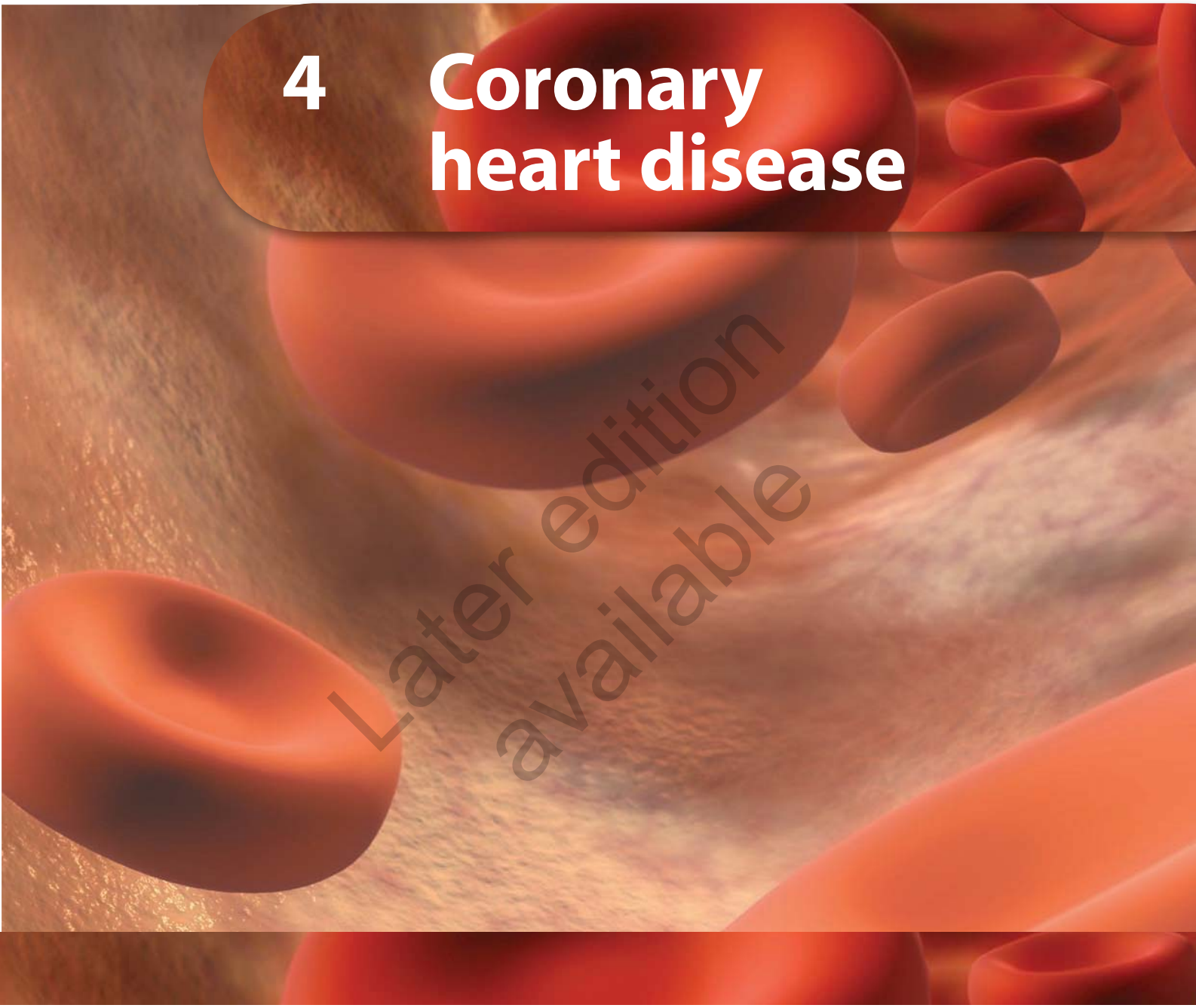
Source: OECD 2010.

Figure 3.16: Cardiovascular death rates, selected countries, 2006

Later edition available

Burden of cardiovascular disease

The burden of disease is a measure of the years of healthy life that an individual, or population, loses as the result of disease or injury. CVD was responsible for 18% of the overall burden of disease and injury in Australia in 2003, with coronary heart disease (CHD) and stroke contributing over four-fifths of this. The contribution from CHD was greater in males (53% of total CVD burden) than females (47%) while the reverse was the case for stroke (45% for males and 55% for females). In 2003, most of the CVD burden (78%) came from years of life lost to premature death and the remainder from disability (AIHW 2010a; Begg et al. 2007).



4 Coronary heart disease

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4 Coronary heart disease

What is coronary heart disease?

Coronary heart disease (CHD), or ischaemic heart disease as it is often referred to, is the most common form of cardiovascular disease. There are two major clinical forms—heart attack (often known as acute myocardial infarction or AMI) and angina. A heart attack is a life-threatening event that occurs when a blood vessel supplying the heart itself is suddenly blocked completely, threatening to damage the heart muscle and its functions. Angina is a chronic condition in which short episodes of chest pain can occur periodically when the heart has a temporary deficiency in its blood supply.

Angina is generally not life-threatening on its own, but those with the condition are more likely to have a heart attack or experience sudden cardiac death than those without it (AIHW: Penm 2008). However, a form known as unstable angina is the more dangerous and less predictable form and is medically treated in a similar manner to heart attack.

All the major risk factors for CVD discussed in Chapter 2 also increase the risk of developing CHD.

How many Australians have coronary heart disease?

Prevalence

Estimates from the self-reported 2007–08 National Health Survey (NHS) indicate that about 3% of the Australian population had had CHD (around 685,000 people). Of those with CHD, 353,000 had experienced angina and 449,000 other ischaemic heart diseases (AIHW 2010a) (note that a person may report more than one disease).

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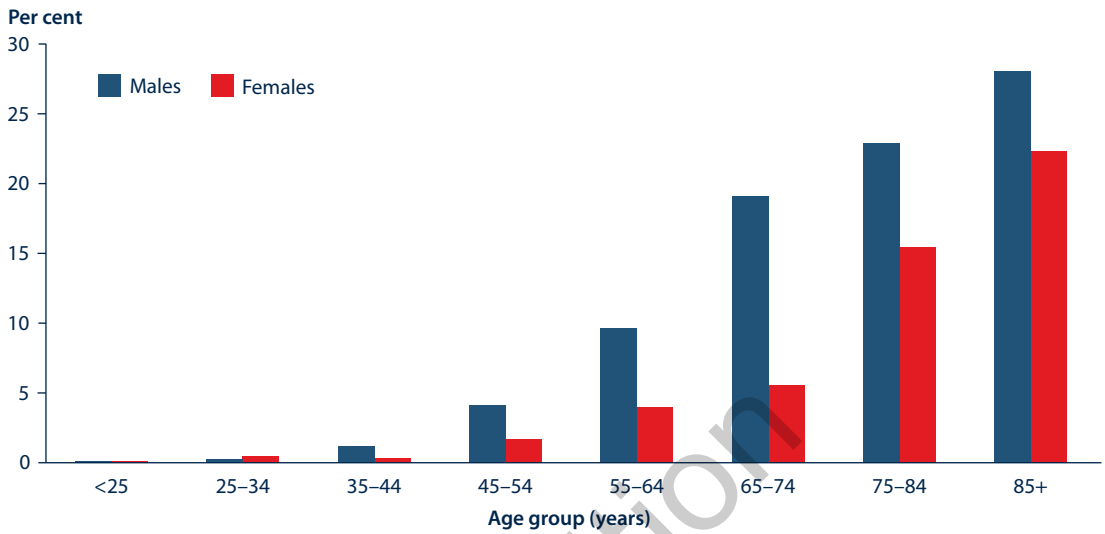
Sex and age

The prevalence of CHD was higher among males than females in all age groups older than 35 years. Overall, after adjusting for age, 4% of males were estimated to have CHD, compared to 2% of females.

The prevalence of CHD increases markedly with age. In 2007–08, around 7% of Australians aged 55–64 years were estimated to have CHD, increasing to 24% among those aged 85 years and over. The proportion of females with CHD increased from 6% in the 65–74 year age group to 15% among those aged 75–84 years. The comparable rates for males were 19% and 23%. At 85 years and over the rates for both males and females were at their highest, 28% and 22% respectively (Figure 4.1).



Source: AIHW analysis of ABS 2007–08 National Health Survey.

Figure 4.1: Coronary heart disease prevalence, by age and sex, 2007–08

Later edition available

Aboriginal and Torres Strait Islander people

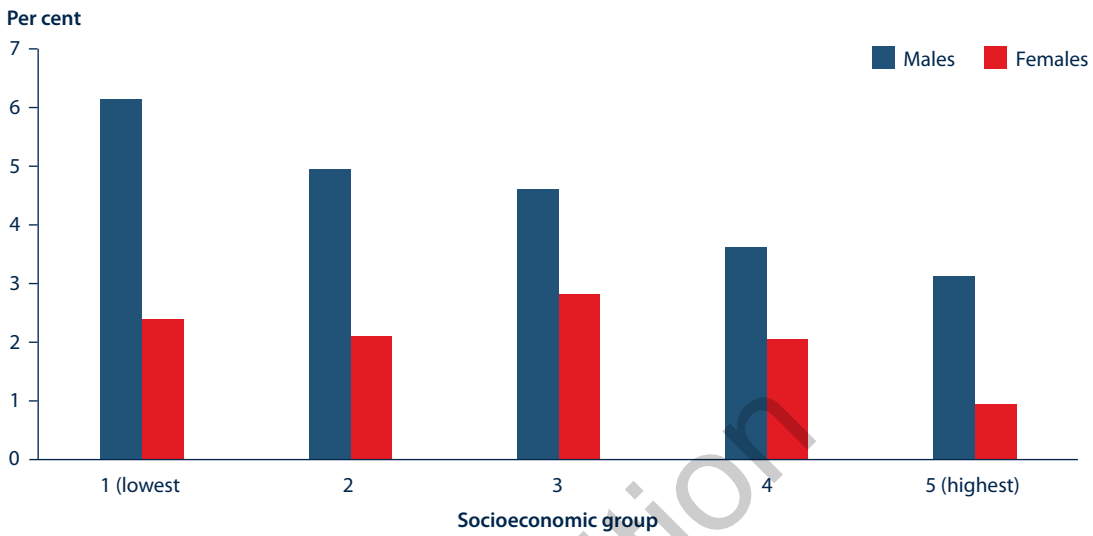
From the 2004–05 National Aboriginal and Torres Strait Islander Heath Survey (NATSIHS) it was estimated that 1% of Indigenous Australians (5,800 people) had CHD. Of these, 48% (2,800) were males and 52% (3,000) were females. When adjusted for age differences, the prevalence rate for Indigenous Australians was approximately twice as high as that for non-Indigenous Australians.

Remoteness

No substantive differences in the overall prevalence of CHD were found across remoteness areas. However, the prevalence among males in more remote areas (*Outer regional* and *Remote and very remote* areas) was higher (5.6%) than in *Major cities* and *Inner regional* areas (both 4.2%).

Socioeconomic group

In 2007–08, overall CHD prevalence was highest in the lowest socioeconomic group and lowest in the highest socioeconomic group. The decline in prevalence with increasing socioeconomic position was evident among males, although for females it was less clear, with the highest level of prevalence in the middle socioeconomic group (Figure 4.2).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW analysis of ABS 2007–08 National Health Survey.

Figure 4.2: Coronary heart disease prevalence, by socioeconomic group and sex, 2007–08

Later edition available

Incidence

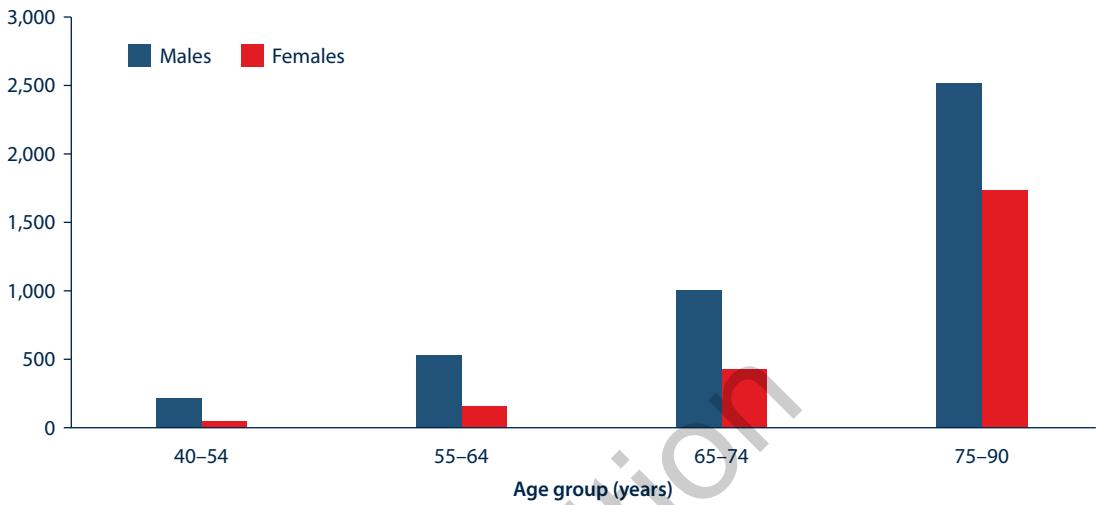
There are no national data sources for measuring incidence (the number of new cases over a given period) of CHD. However, a related measure, the number of major coronary events or heart attacks, can be derived from the national hospitals and mortality databases and is used in this section of the report. At the time of development of the methodology, a decade ago, the estimates were shown to provide a reasonable approximation of the true incidence of heart attack in the population (Jamrozik et al. 2001). Due to changes over time in clinical practice, diagnostic techniques and coding of major coronary events this method is now under review.

Sex and age

Using the method outlined above, in 2007 there were an estimated 49,391 major coronary events in Australia among 40–90 year olds (31,036 men and 18,355 women)—about 135 per day. Nearly 40% of these events were fatal (18,265 cases). The overall rate of major coronary events was twice as high among males as it was among females. After adjusting for age, there were 703 major coronary events per 100,000 population among males, compared with 331 per 100,000 among females.

The rate of major coronary events increased with age—rates among 75–90 year olds were over 16 times as high as among 40–54 year olds. The rate was higher among males for every age group.

The rate for women aged 65–74 years was similar to that of men aged 55–64 years, indicating that men, on average, suffer from coronary heart disease at younger ages than women (Figure 4.3).



Sources: AIHW National Hospital Morbidity Database and AIHW National Mortality Database.

Figure 4.3: Incidence of major coronary events for people aged 40–90 years, by age and sex, 2007

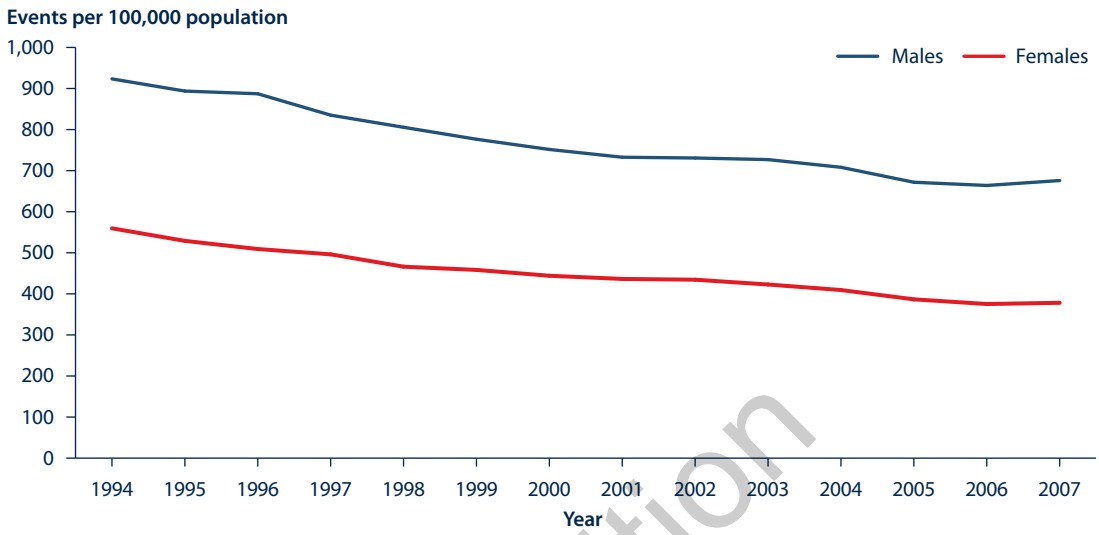
Later edition available

Trends

Incidence rates of major coronary events among 40–90 year olds have declined by almost one–third over the past 14 years. The age-standardised incidence rate for males fell from 923 per 100,000 population in 1994 to 676 per 100,000 in 2007. The comparable rates for females were 560 per 100,000 in 1994, declining to 378 in 2007 (Figure 4.4).

Survival rates from major coronary events have improved steadily over time (AIHW 2010a) and an analysis of long-term survival from heart attacks among people in Perth also shows this improvement (Briffa et al. 2009a; Briffa et al. 2009b).

Some care should be taken in interpreting trends in the estimates of event and fatality rates due to changes in clinical practice over time (resulting in increased rates of hospital transfers and declining average length of stay for CHD patients) and the ongoing introduction of more sensitive diagnostic tests for CHD.



Note: Age-standardised to the 2001 Australian population.

Sources: AIHW National Hospital Morbidity Database and AIHW National Mortality Database.

Figure 4.4: Rate of major coronary events for people aged 40–90 years, by sex, 1994–2007

Later edition available

Aboriginal and Torres Strait Islander people

Previous analysis demonstrated that Aboriginal and Torres Strait Islander people have considerably higher rates of major coronary events than other Australians—3 times as high in 2002–03 (AIHW: Mathur et al. 2006). Higher event rates among Indigenous Australians were also found in more recent studies in Western Australia (Bradshaw et al. 2010) and the Northern Territory where the incidence of AMI (acute myocardial infarction or 'heart attack') in the Indigenous population was found to have increased by 60% between 1992 and 2004 but to have decreased by 20% in the non-Indigenous population over the same period (You et al. 2009).

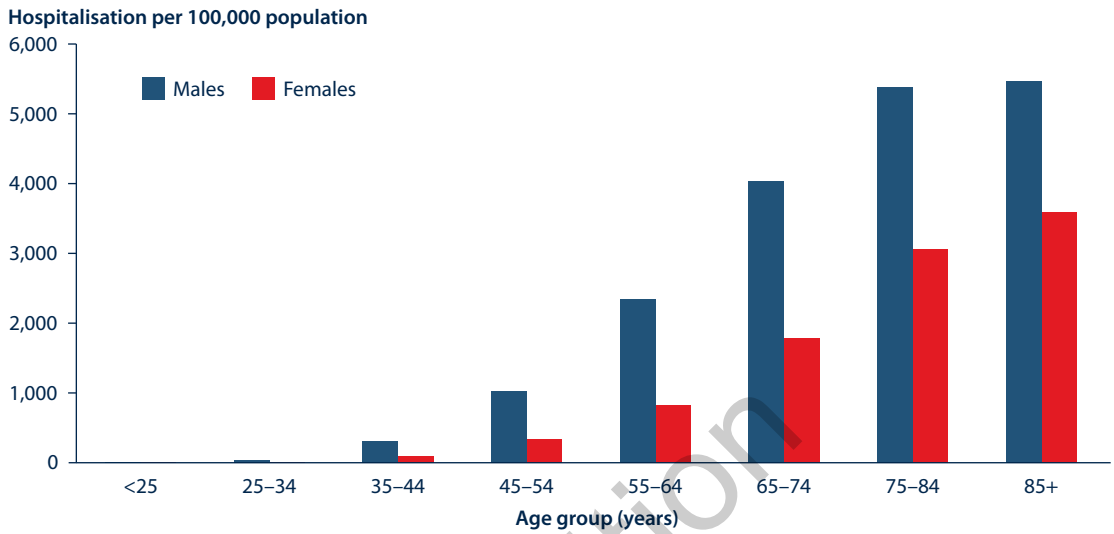
Hospitalisations

In 2007–08, there were 161,417 hospitalisations with a principal diagnosis of CHD, 2% of all hospitalisations, and 34% of hospitalisations for CVD. Of hospitalisations for CHD, angina accounted for 44% (71,801) and AMI for 35% (55,676). Note that hospitalisations data in this report are based on 'episodes of care' rather than the number of people hospitalised with a condition (Appendix A).

Sex and age

In 2007–08, the CHD hospitalisation rate was nearly twice as high for males as it was for females in each age group. Around 60% of hospitalisations with CHD occurred among those aged 65 years and over (Figure 4.5).

The pattern of hospitalisations for both AMI and angina was similar to that for CHD overall, with a higher rate among males than females and rates increasing markedly with increasing age.



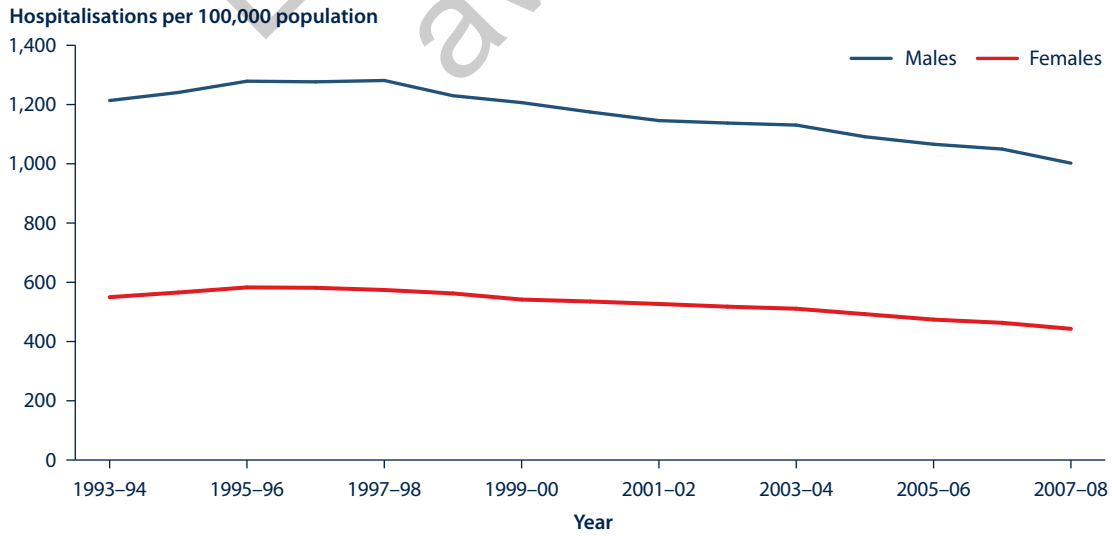
Source: AIHW National Hospital Morbidity Database.

Figure 4.5: Coronary heart disease hospitalisation rates, principal diagnosis, by age and sex, 2007–08

Later edition available

Trends

Between 1993–94 and 2007–08, there was steady reduction in the rate of hospitalisations with a principal diagnosis of CHD, from 867 hospitalisations per 100,000 population in 1993–94 to 709 per 100,000 in 2007–08. This downward trend was more pronounced for males than females, although males continued to be hospitalised at much higher rates than females (Figure 4.6).



Note: Age standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 4.6: Coronary heart disease hospitalisation rates, principal diagnosis, by sex, 1993–94 to 2007–08

There are a number of recent developments that may have influenced the recording of CHD hospitalisations. See Box 4.1 for more information.

Box 4.1: Recent developments that may influence recording of CHD hospitalisations

Hospital data in this report are sourced from the AIHW National Hospital Morbidity Database (NHMD). Hospitalisations with acute myocardial infarction (AMI) comprise a high proportion of CHD hospitalisations and while many factors can affect the consistency of AMI coding in the NHMD, one of the most important has been changes in the sensitivity and specificity of diagnostic tests (Chan et al. 2008; Jamrozik et al. 2001).

Of particular importance in the past ten years has been the introduction and widespread adoption in clinical practice of tests for 'troponins' which are chemicals the body produces when heart tissue dies. The tests are both highly specific and sensitive and recent work by the University of Western Australia suggests that they may lead to an increase in the number of people who are diagnosed with an AMI.

This has implications for the interpretation of AMI hospitalisation rates because if the threshold for AMI diagnosis is being lowered, more hospitalisations with AMI will be recorded than would otherwise have been the case. Additionally, testing for troponins may result in a decrease in the number of admissions with diagnosed angina as an increasing proportion of patients presenting with chest pain are classified as admissions with AMI.

Hospitalisation rate changes may also have been influenced by an increased use of specialised treatment for AMI which, coupled with an improved ability to transport patients for treatment, has led to an increase in the rate of transfer between hospitals. This in turn may have led to an increase in the number of AM1 hospital 'separations' recorded in the NHMD.

The AIHW is investigating these issues in more detail, and intends to publish its findings in 2011. It is important that these issues are taken into consideration when interpreting CHD and AMI hospitalisation trends presented in this report.

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Health inequalities

The following section looks at the impact of CHD hospitalisations on subgroups in the Australian population. Hospitalisation rates are presented for Indigenous and other Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2007–08, there were 3,742 hospitalisations with a principal diagnosis of CHD where the patient was identified as Indigenous. After adjusting for age, hospitalisation rates with CHD among Aboriginal and Torres Strait Islander people were almost twice as high as those for other Australians (1,566 per 100,000 population compared with 727 per 100,000). Indigenous identification in hospitalisation data is considered of sufficient quality for national reporting for New South Wales, Victoria, Queensland, Western Australia, South Australia and the Northern Territory (public hospitals) only. For further information refer to 'Reporting of Indigenous data' in Appendix A.

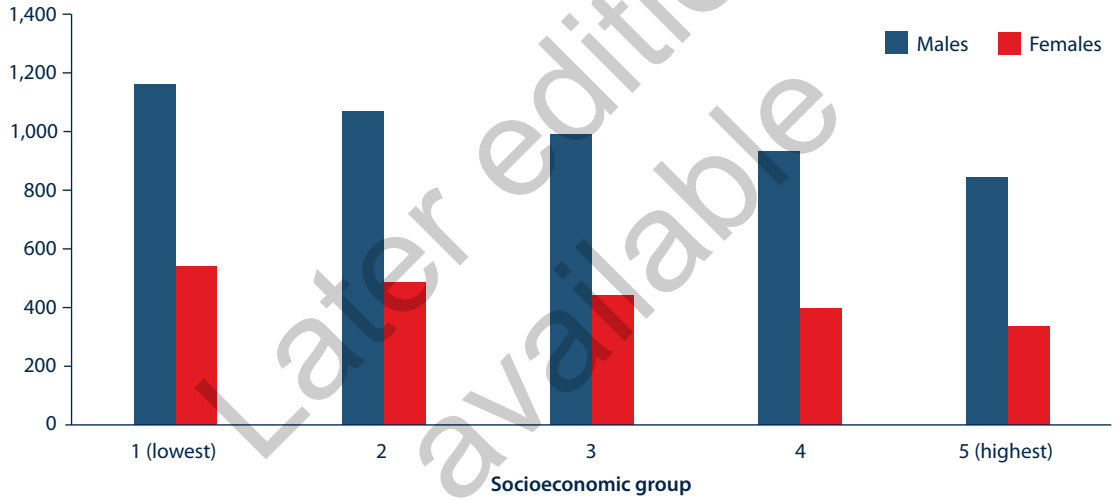
Remoteness

In 2007–08, the age-adjusted rate of hospitalisations with CHD increased with increasing remoteness. For people living in *Major cities* the rate was 666 per 100,000 population compared to 925 per 100,000 in *Remote and very remote* areas.

Across all remoteness areas, male hospitalisation rates were approximately twice as high as female rates.

Socioeconomic group

In 2007-08, for both males and females, the CHD hospitalisation rate was highest among those in the lowest socioeconomic group (840 per 100,000 population) and lowest for those in the highest socioeconomic group (573 per 100,000). Male rates were more than twice as high as female rates in each group (Figure 4.7).



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Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

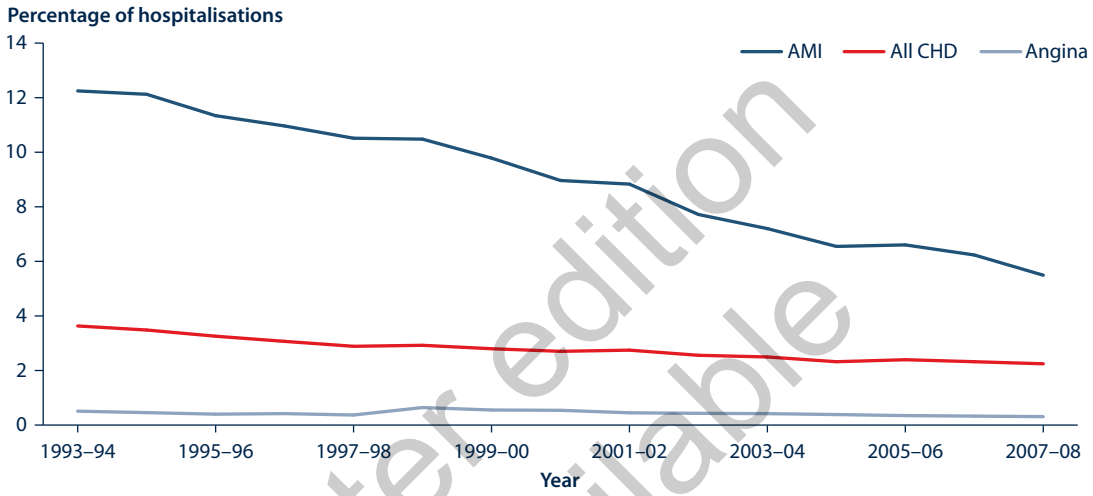
Figure 4.7: Coronary heart disease hospitalisation rates, principal diagnosis, by socioeconomic group and sex, 2007–08

Length of stay in hospital

The average length of stay for hospitalisations with CHD (excluding same day hospitalisations) declined steadily from 7.4 days in 1993–94 to 6.4 days in 1999–2000, after which it remained virtually unchanged until 2007–08. The average length of stay for AMI fell evenly, from 9.1 days in 1993–94 to 6.9 days in 2007–08, while for angina it changed very little over the period, rising unevenly from 4.8 days in 1993-94 to 5.8 in 1998-99 and then falling slightly over the next decade to 5.3 days.

Deaths in hospital

The proportion of CHD hospitalisations ending in death has declined steadily from 3.6% in 1993–94 to 2.2% in 2007–08. Hospitalisations with AMI saw the largest fall, from 12.2% in 1993–94 to 5.5% in 2007–08 (Figure 4.8). The fall in hospital death rates for CHD is probably related to major advances in diagnosis and treatment that have occurred in the past 2 decades. Improved diagnostic sensitivity may also affect the rates if the number of people diagnosed, and subsequently hospitalised with the disease, increases as a result. At this stage it is unknown to what extent hospital death rates may have been affected by changing levels in risk factors in the community.



Source: AIHW National Hospital Morbidity Database.

Figure 4.8: Coronary heart disease hospitalisations ending in death, 1993–94 to 2007–08

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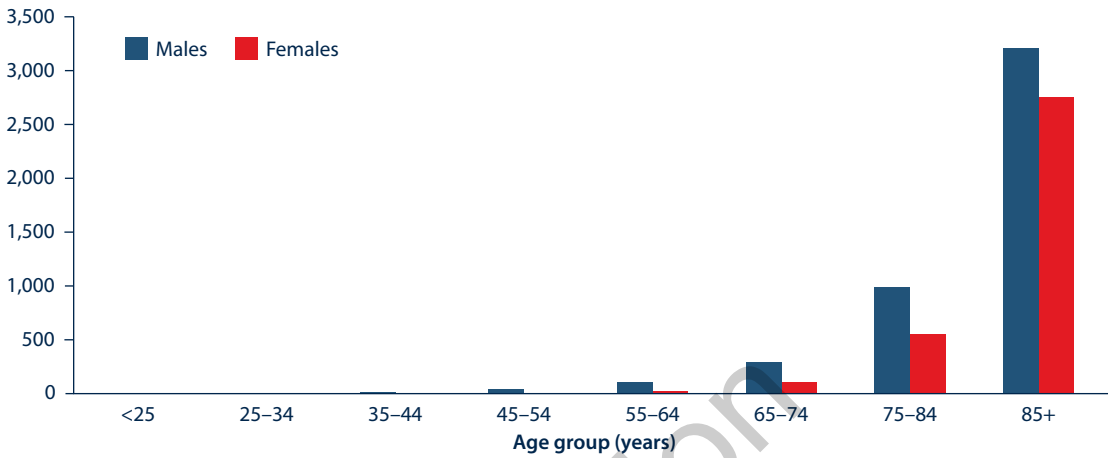
Deaths

In 2007, CHD accounted for more deaths (22,729) than any other disease in Australia—17% of all deaths and 49% of deaths from cardiovascular disease. Approximately half of CHD deaths resulted from AMI (heart attacks).

Sex and age

The CHD death rate increases markedly with age. In 2007, nearly 44% of all CHD deaths occurred among those aged 85 years and over, while fewer than 5% occurred among those less than 55 years of age.

Overall, the age-standardised 2007 male death rate from CHD (126 deaths per 100,000 population) was 1.7 times as high as the female rate (74 per 100,000). Between the ages of 25 and 64 years males had death rates around 3 to 5 times as high as those of females. The death rate remained higher for males aged over 85 years (3,208 per 100,000 population compared to 2,751 per 100,000 for women), even though more women in this age group died from CHD. And, as with CVD overall, the explanation rests with the fact that women, on average, live longer than men and the CHD death rate is closely associated with increasing age (Figure 4.9).



Source: AIHW National Mortality Database.

Figure 4.9: Coronary heart disease death rates, by age and sex, 2007

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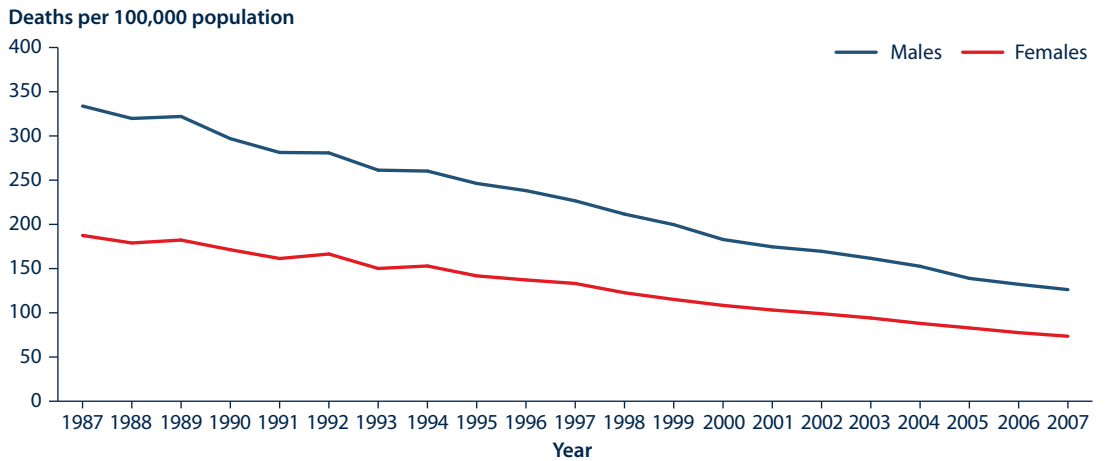
Trends

Between 1987 and 2007, the age-standardised CHD death rate more than halved in Australia, falling from 251 deaths per 100,000 population to 98 per 100,000. CHD death rates fell similarly for males and females (Figure 4.10). On the whole, this downward trend occurred in each age group although the rate of decline for males between the ages of 35 and 54 years has slowed in the past decade, as it has done for females in the 45–54 years age group (AIHW 2010b).

The decline in Australian CHD death rates can be attributed to a number of factors; prominent among them are a decline in the levels of tobacco smoking and the availability of better medical care. Evidence from other countries attributes improvements in risk factors and treatments in about equal proportions (Bennett et al. 2006; Ford et al. 2007; Laatikainen et al. 2005; Unal et al. 2004).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Figure 4.10: Coronary heart disease death rates, by sex, 1987–2007

Health inequalities

The following section looks at the impact of CHD deaths on subgroups in the Australian population. Death rates are presented for Indigenous and non-Indigenous Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2005–2007, CHD was the underlying cause of death for 959 Indigenous people in New South Wales, Queensland, Western Australia, South Australia and the Northern Territory. These are the only jurisdictions for which the Indigenous identification is considered to be sufficiently reliable for national reporting. For further information refer to 'Reporting of Indigenous data' in Appendix A. The age-standardised CHD death rate for Indigenous Australians estimated from data for these jurisdictions was 203 per 100,000 population—1.4 times as high as the rate of non-Indigenous Australians (142 per 100,000). For Indigenous males and females, death rates were 1.4 and 1.5 times greater than those of their non-Indigenous counterparts (Figure 4.11).

The CHD death rate among Indigenous males was higher than the female rate across all age groups except those aged 75 years and over, where the rates were similar. CHD death rates increased markedly with age for all people. For both Indigenous and non-Indigenous people the CHD death rate increased about three-fold between age groups 80–84 years and 85 years and over.

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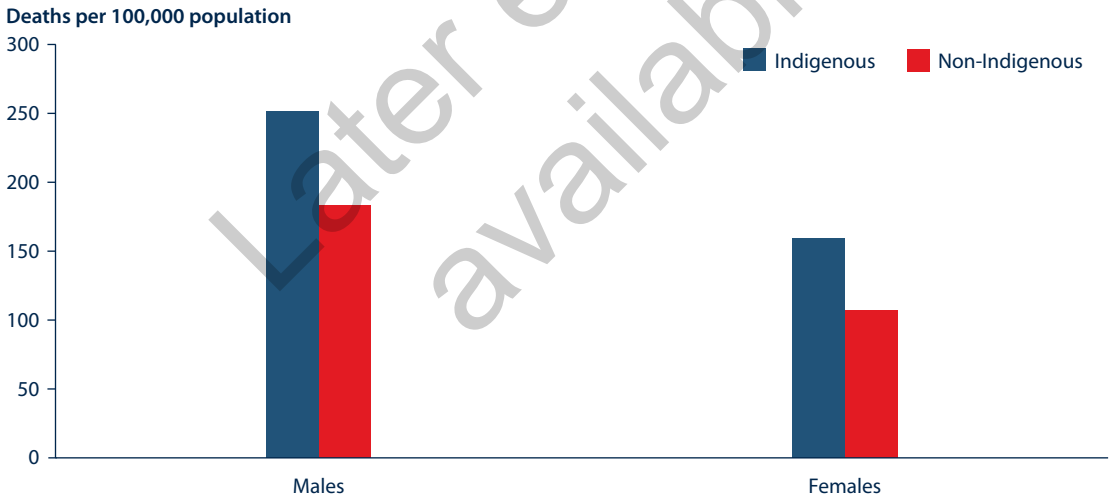


Notes

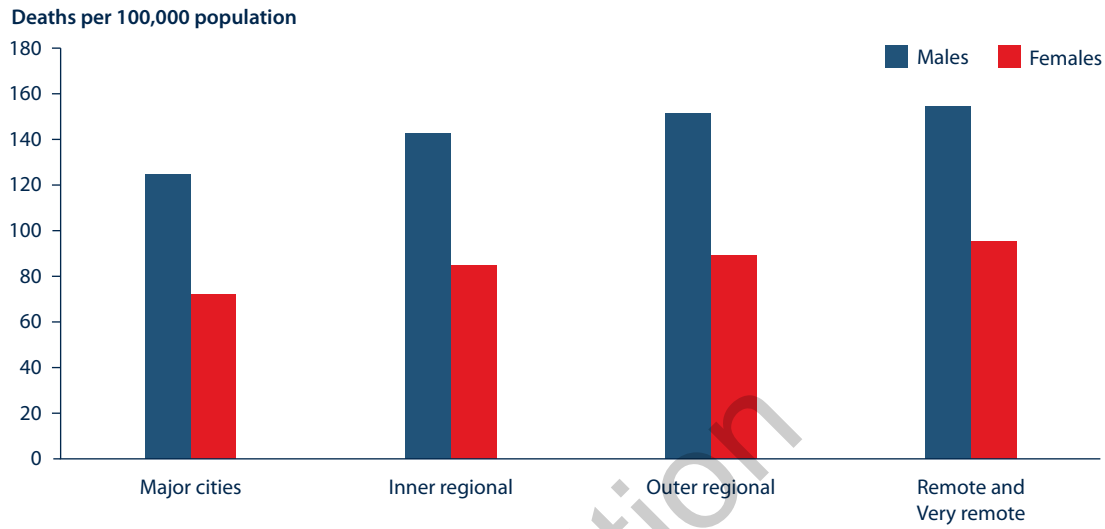
1. Age-standardised to the 2001 Australian population.
2. Data for NSW, Qld, WA, SA and NT only.

Source: AIHW National Mortality Database.

Figure 4.11: Coronary heart disease death rates, by sex and Indigenous status, 2005–2007

Remoteness

In 2005–2007, *Remote and very remote* areas had the highest age-standardised CHD death rate (128 deaths per 100,000 population) and *Major cities* the lowest (96 deaths per 100,000 population). The male rate appeared to be higher than the female rate in all remoteness areas (Figure 4.12).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Figure 4.12: Coronary heart disease death rates, by remoteness and sex, 2005–2007

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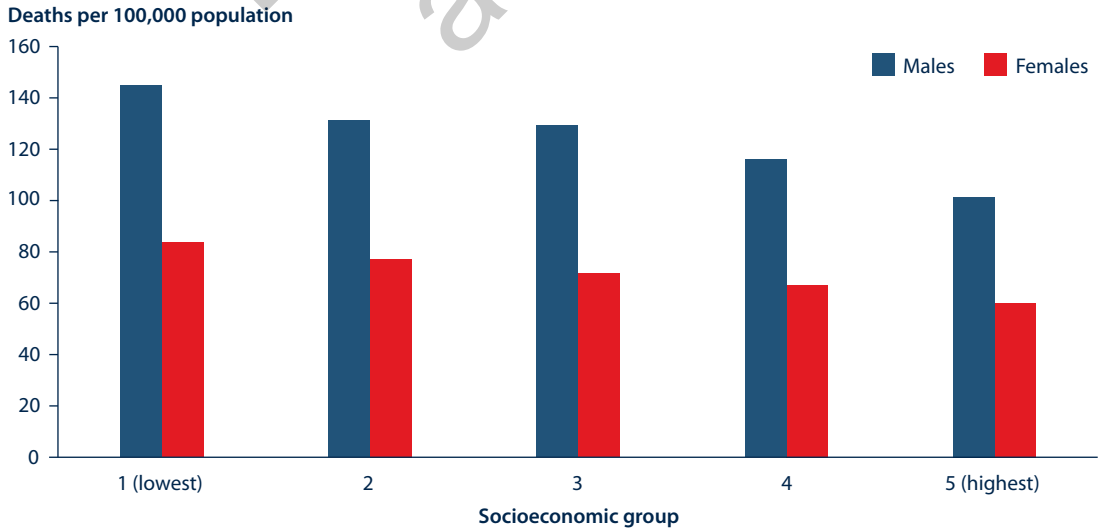
Socioeconomic group

In 2007, for both males and females, the age-standardised CHD death rate was highest in the lowest socioeconomic group (112 deaths per 100,000 population) and lowest in the highest socioeconomic group (78 deaths per 100,000). The overall death rate declined evenly with rising socioeconomic position (Figure 4.13).



Note: Age-standardised to the 2001 Australian population.

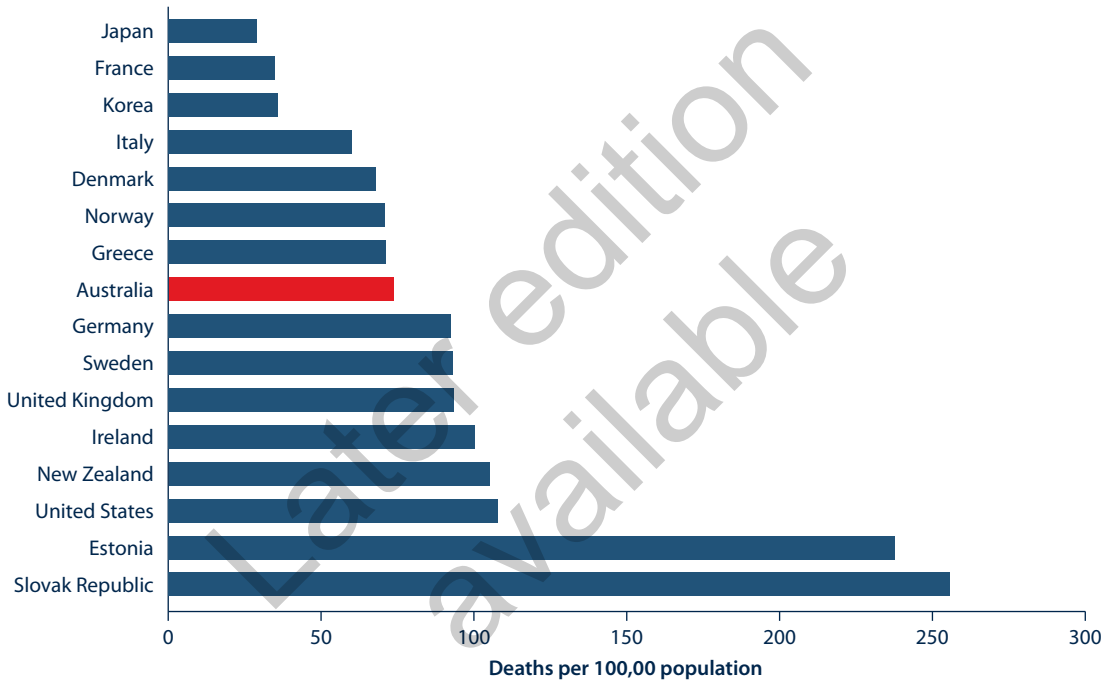
Source: AIHW National Mortality Database.

Figure 4.13: Coronary heart disease death rates, by socioeconomic group and sex, 2007

International comparisons

The Australian CHD death rate was over 3 times as high as that of Japan, but only about a third of that of the Slovak Republic—the country with the highest CHD death rate in the OECD (Figure 4.14). The Slovakian death rate was nearly 9 times as high as that of Japan.

Since 1980, CHD mortality rates have declined in nearly all OECD countries. The declines have been greatest in Estonia, Denmark, Sweden, Norway, New Zealand and Australia, where CHD mortality rates have been cut by two–thirds or more. A small number of countries, however, have seen little or no decline since 1980 and in some countries, such as Korea, mortality rates have increased.



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Notes

1. Rates have been age-standardised to the 1980 OECD population.
2. Rates for the Slovak Republic and the Unites States are for 2005.

Source: OECD 2010.

Figure 4.14: Coronary heart disease death rates for selected countries, 2006

The burden of coronary heart disease

The burden of CHD (including heart failure) in 2003 was estimated to be the leading specific cause of overall disease burden for males (11% of burden) and the second leading specific cause for females (9% of burden), after anxiety and depression (Begg et al. 2007). In 2003, 79% of the overall CHD burden for females, and 85% for males, was due to premature death. Years of healthy life lost due to poor health or disability accounted for the remainder—15% for males and 21% for females.

While disability associated with CHD is a smaller component of the overall burden of CHD than premature death, it remains a major cause of disability in Australia. From self-reported information in the 2003 Survey of Disability, Ageing and Carers, an estimated 1.5% of Australians have one or more disabling conditions associated with CHD, corresponding to about 303,500 people. Of these, almost half needed help or had difficulties with self-care, mobility or communication (AIHW 2010a).

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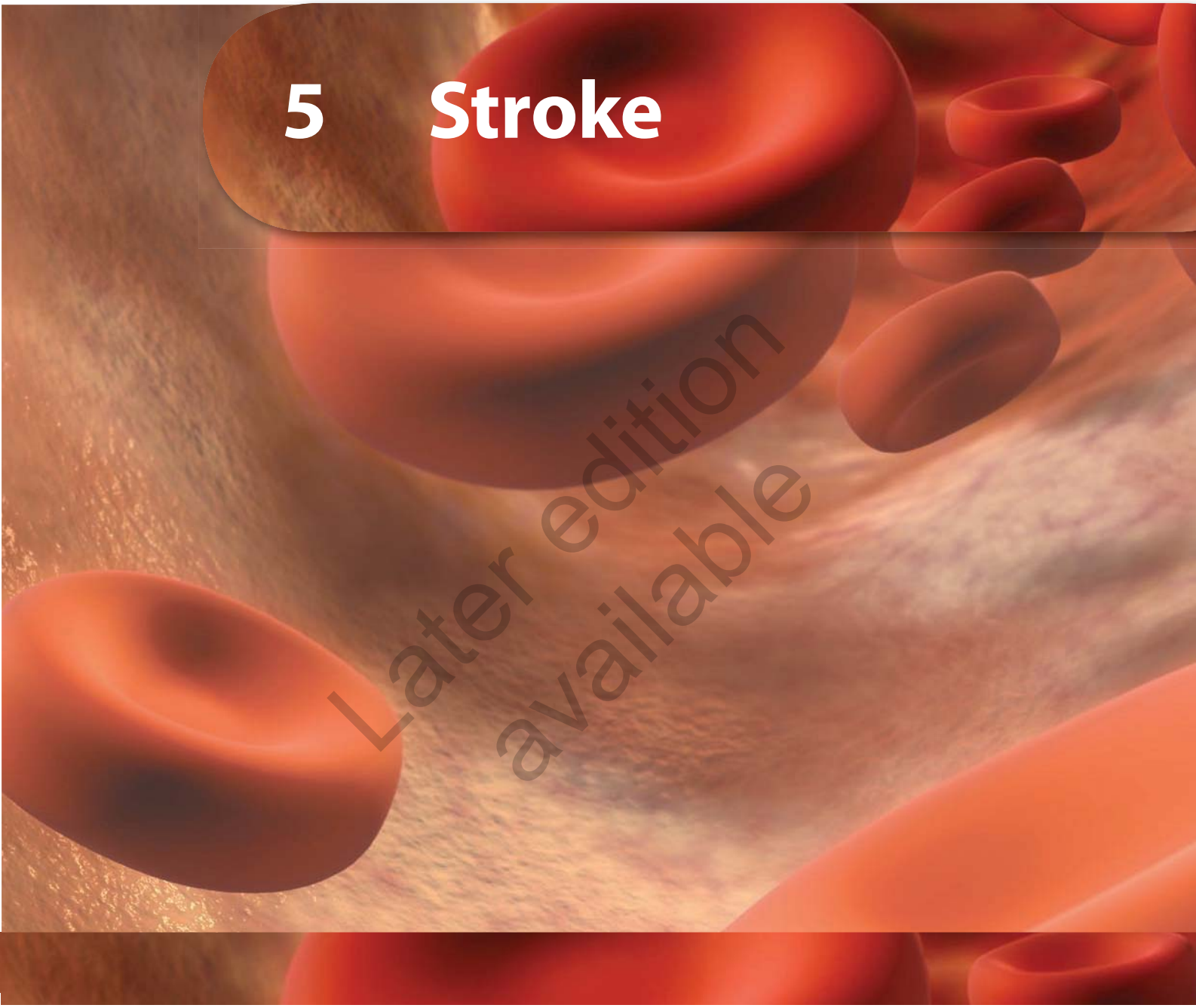
5 Stroke



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5 Stroke

What is stroke?

Stroke occurs when an artery supplying blood to the brain either suddenly becomes blocked or begins to bleed. This may result in part of the brain dying, leading to a sudden impairment that can include a range of activities such as speaking, thinking, movement and communication. Stroke is often fatal.

There are two main types of stroke: a blood clot or other particles blocking a blood vessel causes one type (ischaemic stroke) and the rupturing and subsequent bleeding of a blood vessel causes the other (haemorrhagic stroke). In this report both types of stroke are reported.

A condition related to stroke is transient ischaemic attack (TIA), which results from a temporary blockage of the blood supply to the brain, usually lasting only a few minutes, and producing stroke-like symptoms that disappear within 24 hours (AIHW: Senes 2006). It is a very important predictor of stroke. In this report, TIA is not included in the stroke classification unless indicated.

Stroke is sometimes referred to as cerebrovascular disease, but cerebrovascular disease is actually a broad category of diseases which include stroke and other disorders of the blood vessels supplying the brain or its covering membranes. Stroke is the most common form of cerebrovascular disease, accounting for 75% of its deaths. Where the terms stroke and cerebrovascular disease are used interchangeably in this report, this is noted.

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Risk factors for stroke

Risk factors for stroke include transient ischaemic attack, high blood pressure, tobacco smoking, diabetes, high alcohol consumption, high blood cholesterol, atrial fibrillation, other heart disease and narrowing of the carotid arteries.

How many Australians have had a stroke?

Prevalence

The 2003 Survey of Disability, Ageing and Carers (SDAC) is thought to provide more reliable estimates of stroke prevalence than the National Health Survey (NHS) because it includes non-private dwellings, such as nursing homes and aged-care facilities—which the NHS does not. This is particularly important because stroke is strongly associated with increasing age, and many survivors of stroke require the special care these facilities provide. It is important to note that both surveys are self-reported and that neither the NHS nor the SDAC sampled *Very remote* areas. Based on the strengths and weaknesses of the two surveys, data from both have been included in this section.

Based on the 2003 SDAC, an estimated 346,700 Australians had suffered a stroke at some time in their lives. The comparable figure from the 2007–08 NHS was 244,649.

Sex and age

In 2003, an estimated 1.7% of females (168,400 people) and 1.8% of males (178,300 people) had had a stroke at some time in their lives (SDAC).

The prevalence of stroke increased markedly with age, affecting:

- 8.1% of men and 5.3% of women aged 65–74 years
- 14.7% of men and 11.4% of women aged 75–84years
- 15.1% of men and 17.1% of women aged 85 years and over.

Aboriginal and Torres Strait Islander people

Based on self-reported data from the 2004–05 National Aboriginal and Torres Strait Islander Health Survey (NATSIHS), an estimated 1,400 Aboriginal and Torres Strait Islander people (0.3% of the Indigenous population) had experienced cerebrovascular disease (of which it is expected stroke would be the most common condition). After adjusting for age, there was a similar level of cerebrovascular disease prevalence for Indigenous men and women and an overall prevalence that was nearly twice as high as that for non-Indigenous Australians (AIHW: Penm 2008).

(Note that prevalence data from the NATSIHS were available for cerebrovascular disease only and these should be interpreted with caution because of the high (25–50%) relative standard errors associated with the estimates.)

Remoteness

Results from the 2007–08 NHS show little correlation between the prevalence of stroke and remoteness apart from a slight increase among males the further they lived from *Major cities*.

Socioeconomic group

The 2007–08 NHS data indicate that those in the lowest socioeconomic group are 1.8 times as likely to have had a stroke as those in the highest socioeconomic group. For both sexes the likelihood of stroke increased with decreasing socioeconomic position, apart from in the middle-ranked socioeconomic group, where the male rate was lowest of all (Figure 5.1).

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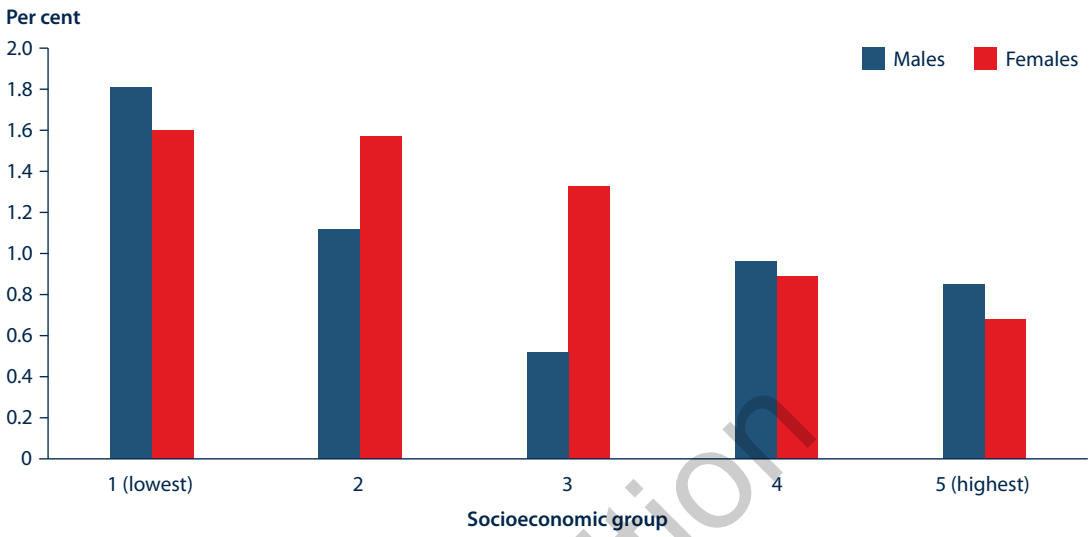

Note: Age-standardised to the 2001 Australian population.

Source: ABS 2009a.

Figure 5.1: Stroke prevalence, by socioeconomic group and sex, 2007–08

Later edition available

Incidence

There are no national data on the incidence (new cases) of stroke. The data used in this section are drawn from the two studies that provide the most up-to-date Australian stroke incidence data. It is acknowledged that neither study is very recent, with the Perth Community Stroke Study (PCSS) having been conducted in 1989–1990 and the North East Melbourne Stroke Incidence Study (NEMESIS) in 1999.

If it is assumed that the incidence rate has not changed since the NEMESIS was conducted, then an estimated 62,000 stroke events would have occurred in Australia in 2010 (45,000 of these would have been first-ever in a lifetime strokes) (NSF 2010). This equates to a stroke occurring every 10 minutes. If it is assumed that the incidence rate has fallen by one per cent since 2000, then about 54,000 strokes would have been expected in 2010 (and 39,500 would have been first-ever in a lifetime strokes). The majority of strokes (around 70%) are first-ever strokes (AIHW 2004) and of those who reported having had a stroke, 72% were aged 60 to 85 years (AIHW 2010a).

Sex and age

Stroke rates increase markedly from about 65 years of age. Results from the PCSS show that the median age of patients having a stroke is about 75 years (Anderson et al. 1993). The comparable figure from NEMESIS is also 75 years (Thrift et al. 2000). In the PCSS the median age of males having a first-ever stroke was 72 years and for females it was 78 years.

Estimates from the PCSS data showed that the age-standardised stroke incidence rate for males was 74% as high as that for females. Investigators in both the Melbourne and Perth studies found that males tended to have strokes at younger ages than females, with between 50% and 58% of strokes occurring in males aged less than 75 years. For females in both studies the corresponding proportion was around 35% (Anderson et al. 1993; Thrift et al. 2000).

Hospitalisations

In 2007–08, there were 34,945 hospitalisations in Australia with a principal diagnosis of stroke (0.4% of all hospitalisations). Around half were treated in a specialised stroke unit (NSF 2009a). Stroke accounted for 7% of all CVD hospitalisations in 2007–08, down from 9% in 1993–94, but the decline was not steady over this period with the lowest proportion of stroke hospitalisations (7%) occurring in both 1998–99 and 2006–07. Note that hospitalisation data in this report are based on 'episodes of care' rather than the number of people hospitalised with a condition (Appendix A).

Sex and age

In 2007–08, the age-standardised hospitalisation rate for males with a principal diagnosis of stroke was 1.3 times as high as that for females. Stroke hospitalisation rates increased rapidly among the most elderly with rates for those aged 85 years and over almost twice as high as for the 75–84 year age group and 12 times the rate among those aged 55 to 64 years. Approximately 66% of stroke hospitalisations occurred among those aged 70 years and over.

Between the ages 45 and 84 years, males had higher rates of stroke hospitalisation than females, after which their rates were similar to females. Beyond 85 years females accounted for 60% of hospitalisations, reflecting the greater proportion living into old age (Figure 5.2).

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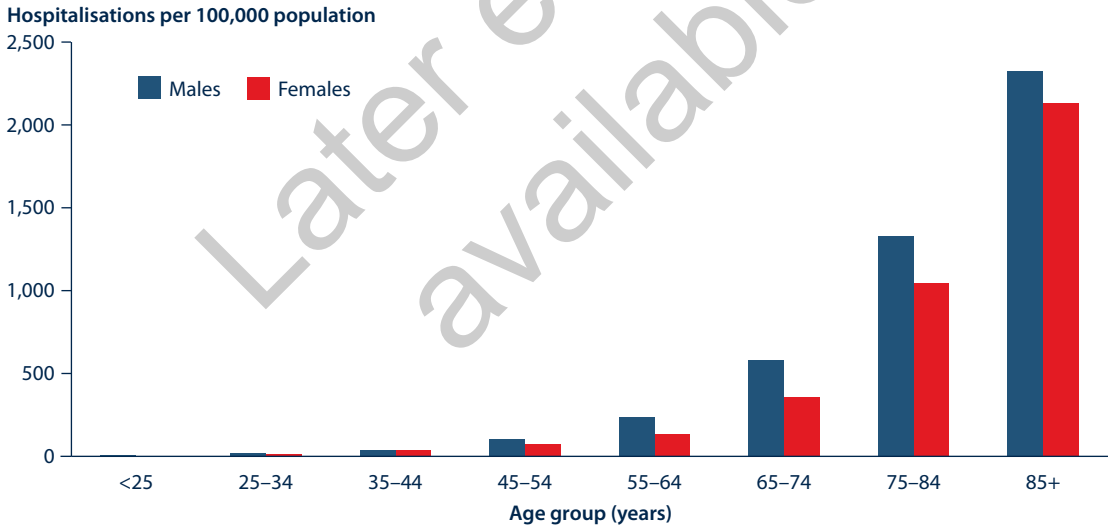
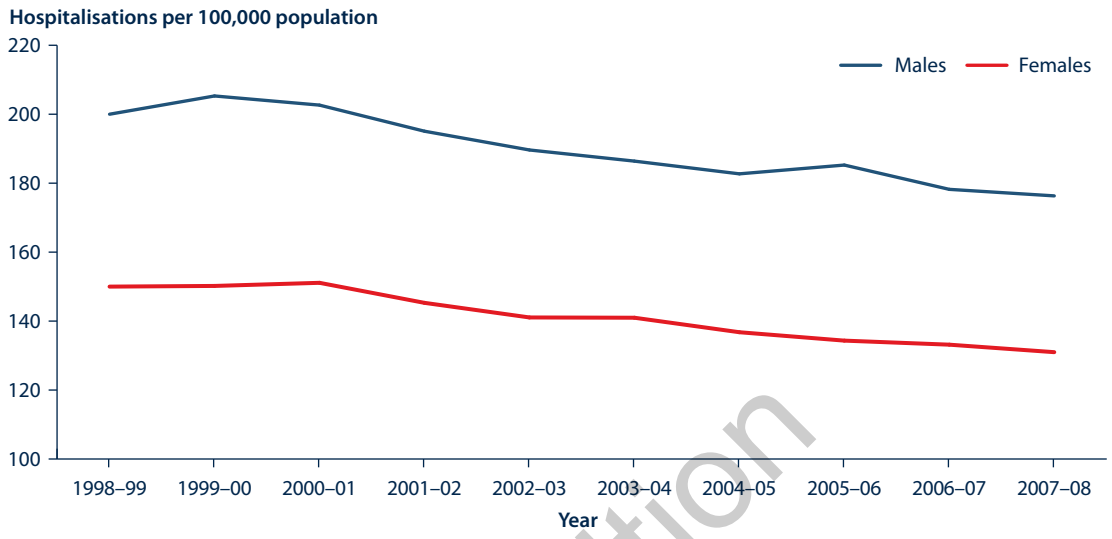


Source: AIHW National Hospital Morbidity Database.

Figure 5.2: Stroke hospitalisation rates, principal diagnosis, by age and sex, 2007–08

Trends

Between 1998–99 and 2007–08, there was a 12% decrease in the age-standardised rate of hospitalisations for stroke (Figure 5.3). The male rate fell from 200 to 176 hospitalisations per 100,000 population and the female rate from 150 to 131 per 100,000. Figure 5.3 begins at 1998–99 for stroke, coinciding with the implementation of ICD-10-AM for hospital coding.



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 5.3: Stroke hospitalisation rates, principal diagnosis, by sex, 1998–99 to 2007–08

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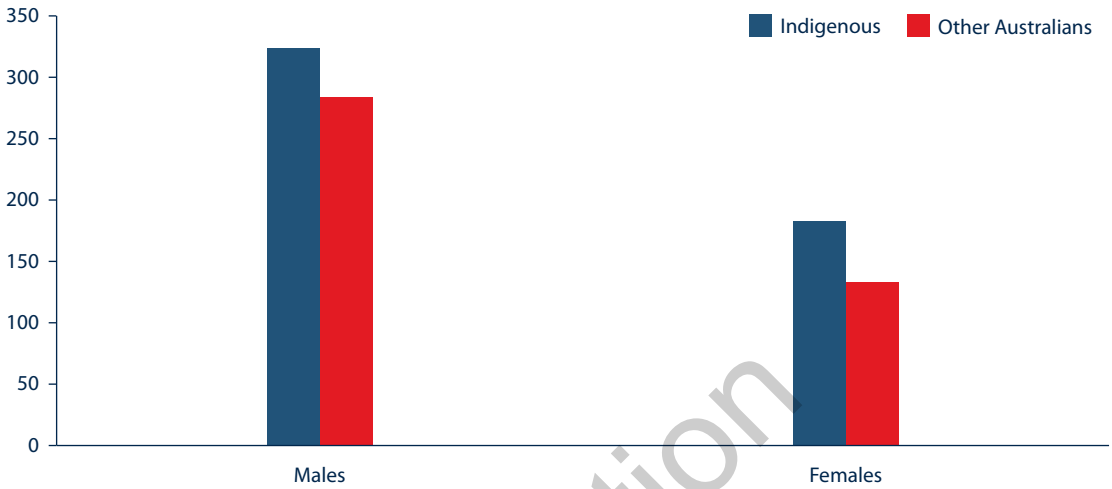
Health inequalities

The following section examines stroke hospitalisations in subgroups of interest in the Australian population. Hospitalisation rates are presented for Indigenous and other Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2007–08 there were 596 hospitalisations for stroke among people identified as Indigenous in hospital records in the jurisdictions with adequate Indigenous identification. The stroke hospitalisation rate for Indigenous males (324 per 100,000 population) was about twice as high as the rate for other Australian males (183 per 100,000 population). Similarly, Indigenous females were hospitalised with stroke at much higher rates (284 per 100,000 population) than other Australian females (133 per 100,000 population) (Figure 5.4). Indigenous identification in hospitalisation data is considered of sufficient quality for national reporting for New South Wales, Victoria, Queensland, Western Australia, South Australia and the Northern Territory (public hospitals) only. For further information refer to 'Reporting of Indigenous data' in Appendix A.



Note:s

1. Age-standardised to the 2001 Australian population.
2. Data from NSW, Vic, Qld, WA, SA and NT (public hospitals only).

Source: AIHW National Hospital Morbidity Database.

Figure 5.4: Stroke hospitalisation rates, principal diagnosis, by sex and Indigenous status, 2007–08

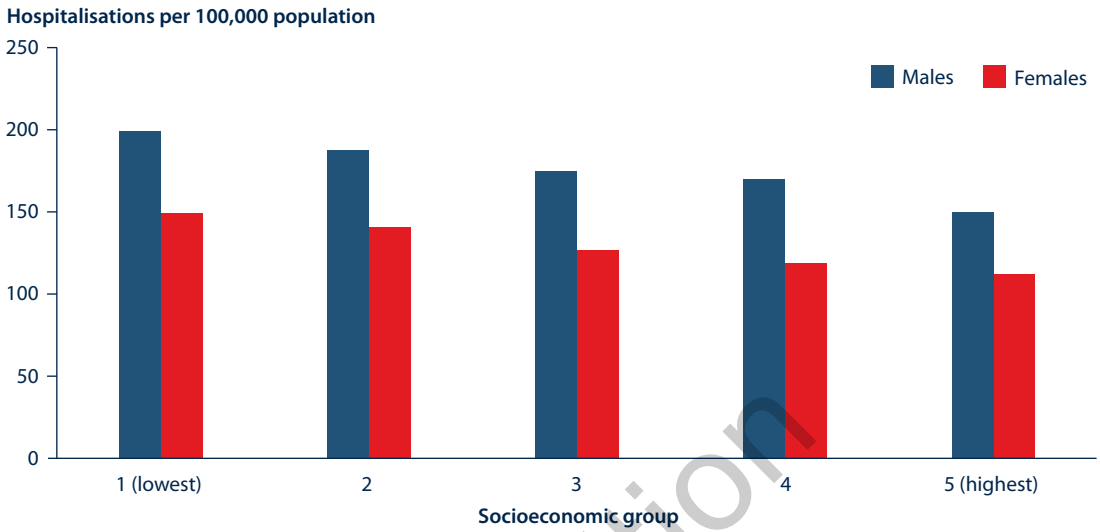
Later edition available

Remoteness

In 2007–08 stroke hospitalisation rates were highest in *Remote and very remote* areas (186 hospitalisations per 100,000 population and lowest in *Major cities* (146 per 100,000). Male rates were higher than female rates in all remoteness areas (Table A5.1).

Socioeconomic group

For both males and females in 2007-08, the stroke hospitalisation rate was highest for those in the lowest socioeconomic group (173 hospitalisations per 100,000 population) and lowest for those in the highest socioeconomic group (130 per 100,000 population) (Figure 5.5). Male rates were higher than female rates within each socioeconomic group.



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 5.5: Stroke hospitalisation rates, principal diagnosis, by socioeconomic group and sex, 2007–08

Later edition available

Length of stay in hospital

In 2007–08, 26% of people admitted to hospital with stroke (or TIA) were discharged the same day. This was an increase from 1993–94, when the proportion of same-day hospitalisations was 15%. The number of same-day hospitalisations increased by an average of 0.8% per year over the period 1993–94 to 2007–08.

Among those hospitalised with stroke for at least one night, the average length of stay was 10.7 days in 2007–08, a decline from the 1993–94 figure of 16.7 days. The decline over this period was fairly steady apart from a small rise between 1999–00 and 2001–02. In 2007–08 nearly half of all stays were for 4 days or less. In 1993–94 it was just over a third.

On average, those hospitalised with stroke or TIA tended to stay twice as long as those hospitalised with CHD. (TIA is included in hospitalisations here to ensure comparability with the published 1993–94 rates.)

Deaths in hospital

In 2007–08, 11% of hospitalisations with stroke (including TIA) ended in death, a small change from the 12% reported in 1993–94.

Deaths

Stroke claimed 8,623 lives in 2007, nearly 6% of all deaths and 19% of CVD deaths, making it the second most common cause of CVD death in Australia. Of those having a first-ever stroke, one in five die as a result within the first month and one in three die within 12 months (AIHW: Senes 2006; Thrift et al. 2000).

Sex and age

In 2007, stroke accounted for approximately 5% (3,466) of all male deaths in Australia and 8% (5,157) of all female deaths. Stroke death rates increase greatly with age, with 82% occurring among those aged 75 years and over. Although the age-specific death rates from stroke are generally higher among males than females (the main exception being the 85 years and over age group) (Figure 5.6), more females than males die from stroke.

In 2007, cerebrovascular disease was the third leading cause of death among males, behind CHD and lung cancer, and the second leading cause of death among females, behind CHD. Ninety-six per cent of cerebrovascular disease deaths in 2007 were the result of stroke (AIHW 2010a).

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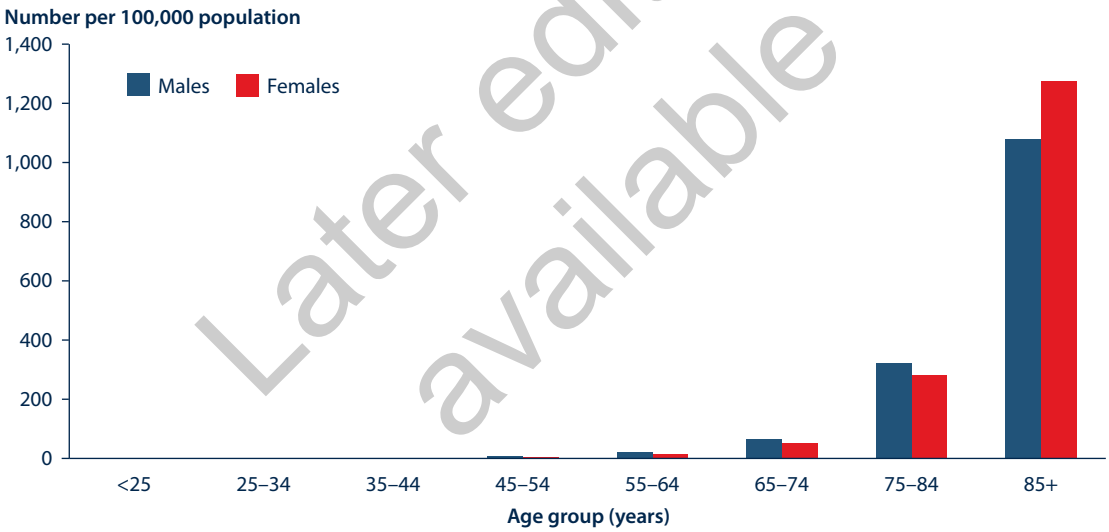


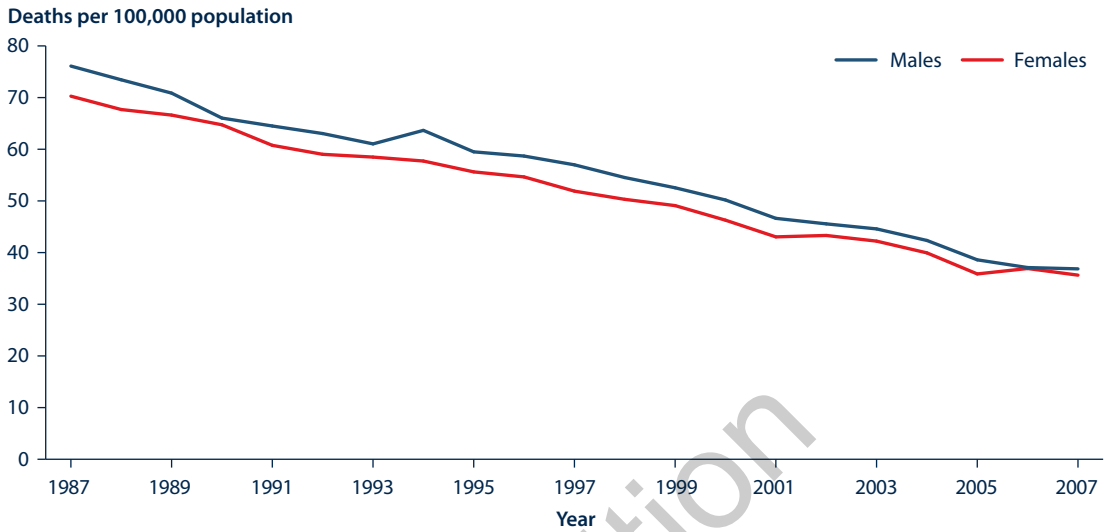
Source: AIHW National Mortality Database.

Figure 5.6: Stroke death rates, by age and sex, 2007

Trends

The steady decline in stroke mortality, which began in the early 1970s, has continued. Age-standardised stroke death rates fell from 74 deaths per 100,000 population in 1987 to 37 deaths per 100,000 in 2007. The average annual rate of decline for both males and females over this period was 4% (Figure 5.7).

Falling stroke death rates appear to have been largely driven by improvements in the levels of risk factors, such as tobacco smoking, and an increased use of blood pressure lowering drugs, treatment to prevent blood clots and other advances in medical care (Gillum 1997).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database

Figure 5.7: Stroke death rates, by sex, 1987–2007

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Health inequalities

The following section looks at stroke deaths in subgroups of interest in the Australian population. Death rates are presented for Indigenous and non-Indigenous Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

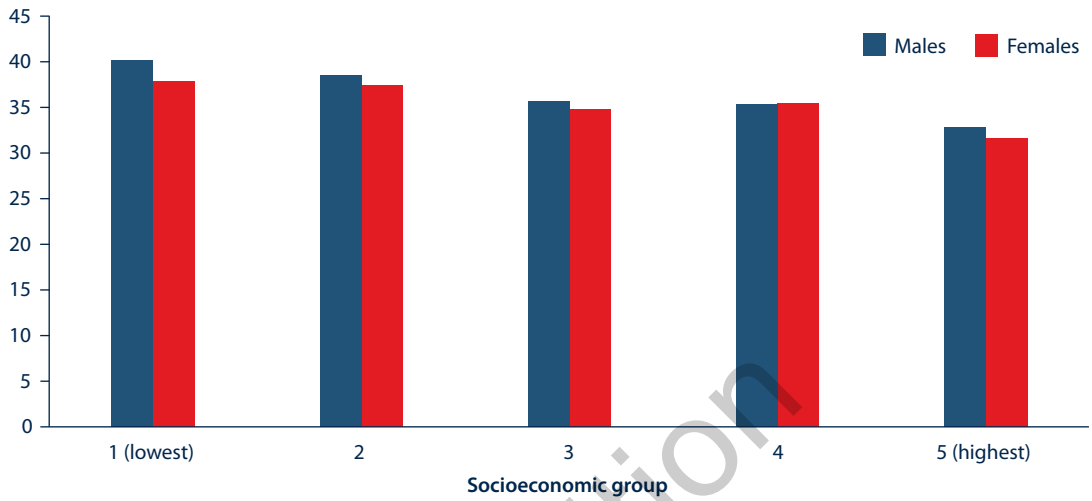
Over the period 2005–2007 there were 274 deaths from stroke among Aboriginal and Torres Strait Islander people in the jurisdictions with adequate Indigenous identification. The age-standardised stroke death rate for Indigenous Australians was 1.4 times as high as the rate of non-Indigenous Australians (72 deaths per 100,000 population compared with 52 per 100,000). The greatest disparity between the stroke death rates for Indigenous and non-Indigenous people was in the 25–29 year age group where the Indigenous rate (4 per 100,000 population) was nearly 7 times as high as the non-Indigenous rate (0.4 per 100,000 population). Indigenous identification in deaths data is considered of sufficient quality for national reporting for New South Wales, Queensland, Western Australia, South Australia and the Northern Territory only. For further information refer to 'Reporting of Indigenous data' in Appendix A.

Remoteness

In 2005–2007, the stroke death rate for those living in *Major cities* appeared to be higher than the rate for people living in *Inner regional*, *Outer regional* and *Remote and very remote* areas, where death rates showed little variation.

Socioeconomic group

In 2007, for both males and females, the stroke death rate was highest for those in the lowest socioeconomic groups and lowest for those in the highest socioeconomic groups (Figure 5.8). There was little difference between male and female death rates in any socioeconomic group.



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Figure 5.8: Stroke death rates, by socioeconomic group and sex, 2007

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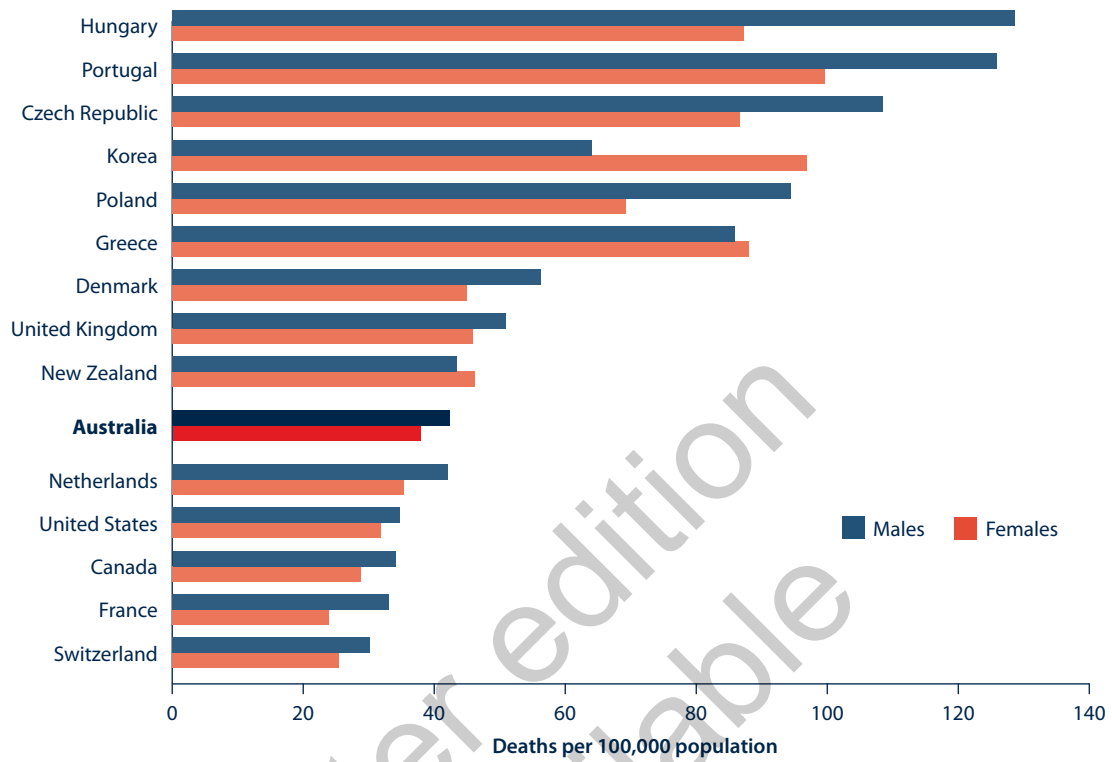
International comparisons

Among OECD countries, Australia had the tenth highest stroke death rate (OECD 2009a). It was low compared with countries such as Hungary, Portugal, Czech Republic and Korea, but 40% higher than the rate in Switzerland, which was lowest overall (OECD 2009a).The stroke death rate for Australian males was 1.4 times as high as the rate for Swiss males. The rate among Australian females was around 1.5 times as high as that in France, where the female rate was the lowest in the OECD. Overall, Portugal had the highest stroke death rate—4 times as high as that of Switzerland and 3 times that of Australia (Figure 5.9).



Note: Rates have been age-standardised to the 1980 OECD population.

Source: OECD Health Data 2009.

Figure 5.9: Stroke death rates for selected countries, 2006 (or latest year available)

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The burden of stroke

In 2003, stroke accounted for 4.5% of the total burden of disease in Australia. It accounted for 5% of the overall disease burden for females, making it the third major contributor behind anxiety and depression and CHD. Among males, stroke was the fifth leading contributor, accounting for 4% of the overall disease burden (Begg et al. 2007).

In 2003, 75% of the stroke burden for females, and 68% for males, resulted from years of life lost to premature death. The balance, 25% for females and 32% for males, was the result of years of healthy life lost because of poor health or disability.

2003 SDAC results indicate that about 282,600 (82%) persons with a history of stroke also had a disability and, in about half of these cases the disability was mainly attributed to stroke. The most common types of disability resulting from stroke were restriction in physical activities, incomplete use of limbs, difficulty gripping or holding items and speech difficulties. Generally, people with stroke and disability were more severely limited or restricted in their activities than all people with a disability (AIHW: Senes 2006).

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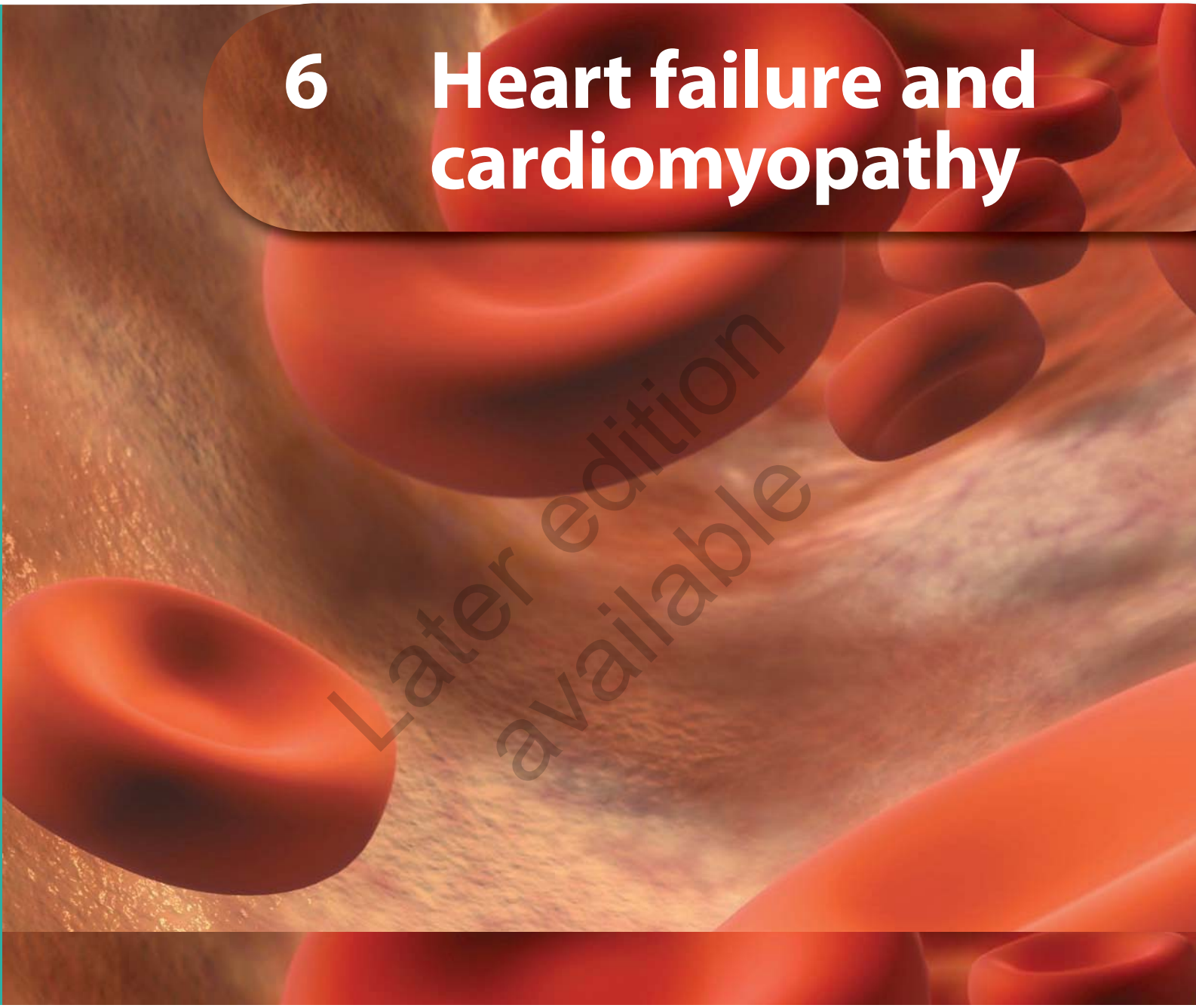

6 Heart failure and cardiomyopathy

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6 Heart failure and cardiomyopathy

What is heart failure?

Heart failure is a condition where the heart is unable to maintain a strong enough blood flow to meet the body's needs. Although it can occur suddenly, it usually develops slowly over many years, as the heart becomes gradually weaker and works less effectively (AIHW: Field 2003). Heart failure can result from a variety of diseases and conditions that impair or overload the heart, notably heart attack, high blood pressure, primary heart muscle weakness (known as cardiomyopathy) or a damaged heart valve.

People with mild heart failure may have few symptoms, but in more severe cases it can result in chronic tiredness, reduced capacity for physical activity and shortness of breath. Heart failure is life-threatening and usually associated with poor survival.

There are different forms of heart failure. Generally heart failure cannot be cured because the heart muscle has been irreversibly damaged although some forms, caused by particular impairments such as heart valve defects or certain effects of a heart attack, may be cured if treated early enough (AIHW 2004). In either case, treatment may improve quality of life, reduce hospital admissions and extend a person's life. In certain end-stage patients, heart transplantation may be used.

Cardiomyopathy is a disease where the heart muscle becomes enlarged, thickened or stiff. As these effects can reduce the effectiveness of the heart, cardiomyopathy and heart failure commonly occur together. In this chapter, cardiomyopathy has been included with heart failure when reporting deaths and hospitalisations. See Appendix B for the codes used to identify heart failure and cardiomyopathy in the analysed data for this report.

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Risk factors for heart failure and cardiomyopathy

Heart failure and cardiomyopathy share risk factors with a number of other chronic diseases and as a result often occur with them. The most important risk factors for heart failure and cardiomyopathy are CHD and high blood pressure. Others are diseases of the heart muscle that result from alcohol abuse or infections; diseases of the heart valves (such as rheumatic heart disease); and diabetes and obesity.

The co-occurrence of multiple diseases in an individual can make the diagnosis of separate diseases, such as heart failure, more difficult.

How many Australians have heart failure?

Prevalence

Estimating the prevalence of heart failure in Australia from the National Health Survey (NHS) is difficult because the information collated is self-reported. In other words, respondents must be aware they have a condition before they can report it and, because heart failure often presents in the initial stages with mild symptoms, many patients may be unaware that they have the condition. As a result NHS estimates are likely to undercount the number of heart failure cases.

A further limitation is that the NHS reports heart failure as a component of the group: *Oedema and heart failure,* rather than as a separate condition. Oedema is a condition where fluid is not properly removed from the body's tissues and while it can be a symptom of heart failure, it also occurs with a range of other conditions, making an accurate measure of heart failure prevalence difficult to obtain from the NHS.

For the reasons mentioned above, estimates of heart failure prevalence presented in this section should be interpreted with caution.

Sex and age

In 2007–08, it is estimated from the NHS that 1.3% of Australians had heart failure or oedema, equating to approximately 277,800 people. More females (177,200) had heart failure or oedema than males (100,500). The prevalence of Australians with heart failure or oedema increased with age, with over a third with the condition aged 75 years or over. It was more prevalent among females than males in every age group, except the very youngest, and especially so among females aged 85 years and over (Figure 6.1).

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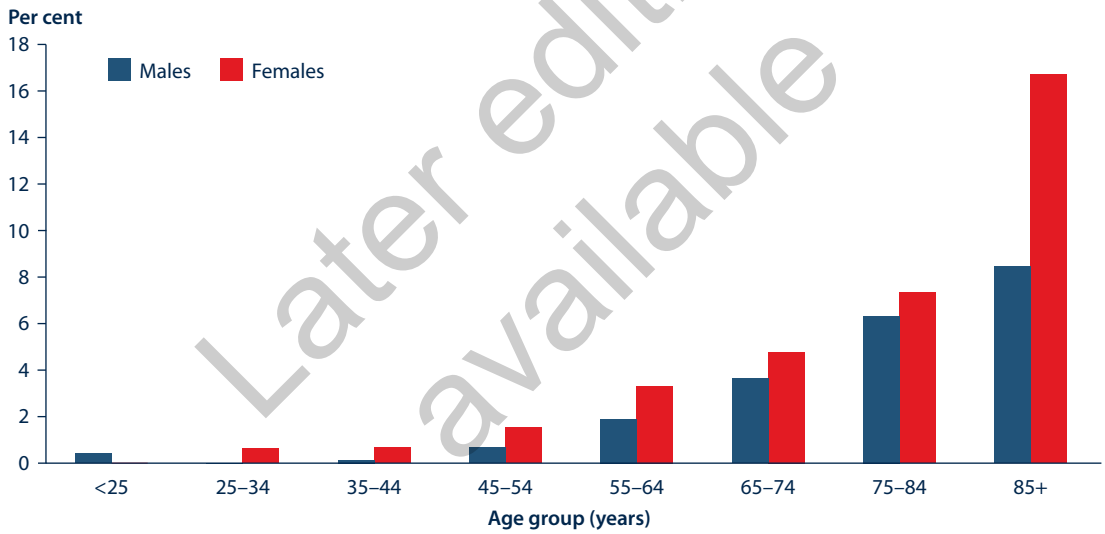

Source: AIHW analysis of ABS 2007–08 National Health Survey.

Figure 6.1: Heart failure and oedema prevalence, by age and sex, 2007–08

Trends

The estimated prevalence of heart failure and oedema in the Australian population (1.3%, 263,000 people) remained largely unchanged between the 2004–05 and 2007–08 National Health Surveys (AIHW 2008b).

Recent estimates suggest that between 1995 and 2005 the number of heart failure patients aged 55 years and over fell by 1.6% (Najafi et al. 2009).

Aboriginal and Torres Strait Islander people

Based on self-reports from the 2004–05 NATSIHS, an estimated 1% of Indigenous Australians, equating to around 4,500 people, have heart failure. After adjusting for differences in age-structure, the Indigenous prevalence rate was 1.7 times as high as the rate for non-Indigenous Australians (AIHW: Penm 2008).

Remoteness

Estimates from the 2007–08 NHS suggest that the prevalence of heart failure and oedema was lowest in *Major cities* for both males and females and highest for males in *Outer regional* and *Remote* areas. For females it was highest in *Inner regional* areas.

Socioeconomic group

In 2007–08, according to estimates from the NHS, heart failure and oedema prevalence in the lowest socioeconomic group (1.6%) was 1.6 times as high as that for the highest socioeconomic group (1%). For males, there was a downward gradient from lowest to highest socioeconomic groups, but for females there are two distinct groups: quintiles 1–2 and quintiles 3–5, within which prevalence rates were similar (Figure 6.2).

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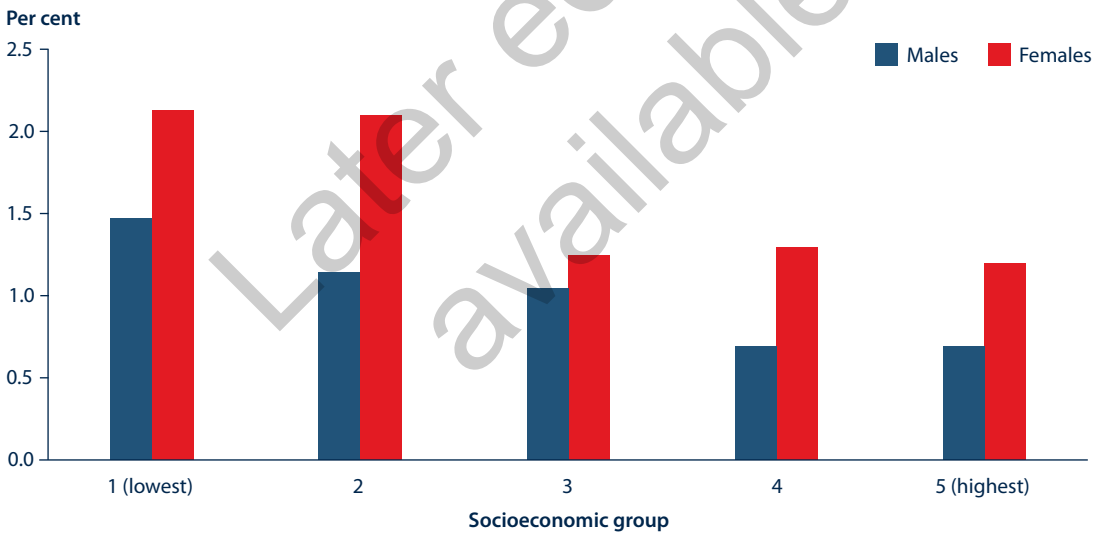

Note: Age-standardised to the 2001 Australian population.

Source: AIHW analysis of ABS 2007–08 National Health Survey.

Figure 6.2: Heart failure and oedema prevalence, by socioeconomic group and sex, 2007–08

International comparisons

Data for the United Kingdom indicate that the prevalence of heart failure is about 3% in people aged 45 years and over (AIHW: Field 2003), compared to an estimated 3.2% in Australia for heart failure and oedema. In the United States, according to the US National Health and Nutrition surveys, heart failure affected an estimated 2% of people aged 40–59 years, 5% aged 60–69 years and 10% aged 70 years or more (AIHW: Field 2003). The estimated prevalence of heart disease and oedema in Australia in 2007–08 for the same age groups is 1.1% of 40–59 year olds, 4% of 60–69 year olds, and 7% of people aged over 70 years. It is important to note that comparing the prevalence estimates of different countries should be done with caution because they may have been arrived at using different methods.

Incidence

Although there are no national data on the incidence (new cases) of heart failure in Australia, by applying overseas findings to the Australian population it has been estimated that around 30,000 new cases of heart failure are diagnosed here each year (AIHW: Field 2003). From another study, it was estimated that 22% of those hospitalised in Australia between 1984 and 1993 with a first heart attack went on to develop heart failure within 28 days of admission (Najafi et al. 2007).

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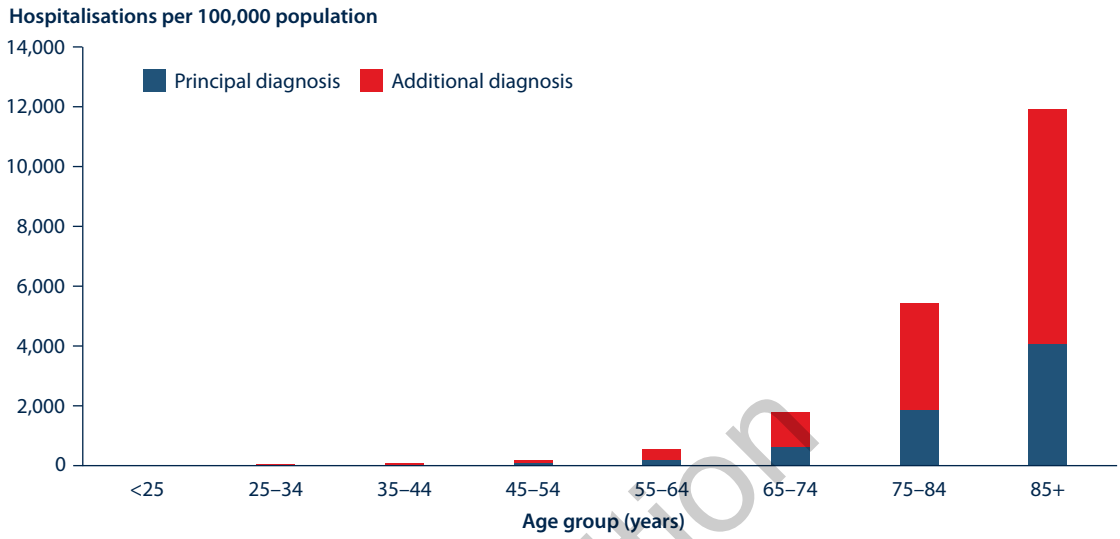
Hospitalisations

Heart failure and cardiomyopathy often occur alongside other chronic diseases, so it is important to count both the principal and the additional diagnoses of heart failure or cardiomyopathy when estimating the true contribution to hospitalisations in Australia. The principal diagnosis is that which is listed in hospital records to describe the problem that was chiefly responsible for the patient's episode of care in the hospital. An additional diagnosis is a condition or complaint, either co-existing with the principal diagnosis or arising during the episode of admitted patient care.

In 2007–08, there were 49,307 hospitalisations in Australia with a principal diagnosis of heart failure or cardiomyopathy, 0.6% of all hospitalisations. Heart failure was the principal diagnosis in 45,212 (92%) of these hospitalisations, with cardiomyopathy accounting for the other 4,095 (8%). Heart failure or cardiomyopathy was listed as an additional diagnosis in a further 94,599 hospitalisations, demonstrating the high frequency with which they occur with other diseases (Figure 6.3). It is important to remember that these figures refer to episodes of hospitalisation, not numbers of people, and an individual with heart failure or cardiomyopathy may be hospitalised more than once with the disease.



Source: AIHW National Hospital Morbidity Database.

Figure 6.3: Heart failure and cardiomyopathy hospitalisation rates, principal and additional diagnosis, by age, 2007–08

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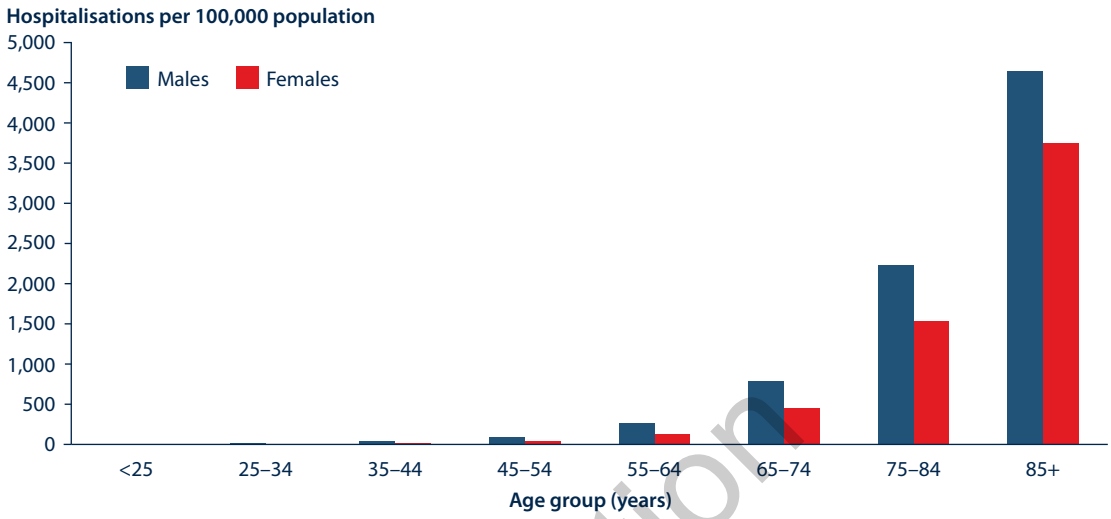
In those cases where heart failure or cardiomyopathy was listed as an additional diagnosis, almost half had either a cardiovascular or respiratory disease listed as the principal diagnosis. The most common principal diagnoses in these cases were CHD (14% of hospitalisations); chronic lower respiratory diseases, including bronchitis and chronic pulmonary obstructive disease (9%); influenza or pneumonia (8%); and, atrial fibrillation or flutter (7%).

Sex and age

Males were hospitalised with a principal diagnosis of heart failure or cardiomyopathy at a higher rate than females in all age groups. The rate of hospitalisations increased rapidly with age, particularly among the elderly. The rate for those aged 85 years and over (4,048 per 100,000 population) was more than twice as high as that for 75–84 year olds (1,847 per 100,000). For males, almost 80% of hospitalisations with heart failure or cardiomyopathy occurred among those aged 65 years or more. For females this proportion was almost 90% (Figure 6.4).



Source: AIHW National Hospital Morbidity Database.

Figure 6.4: Heart failure and cardiomyopathy hospitalisation rates, principal diagnosis, by age and sex, 2007–08

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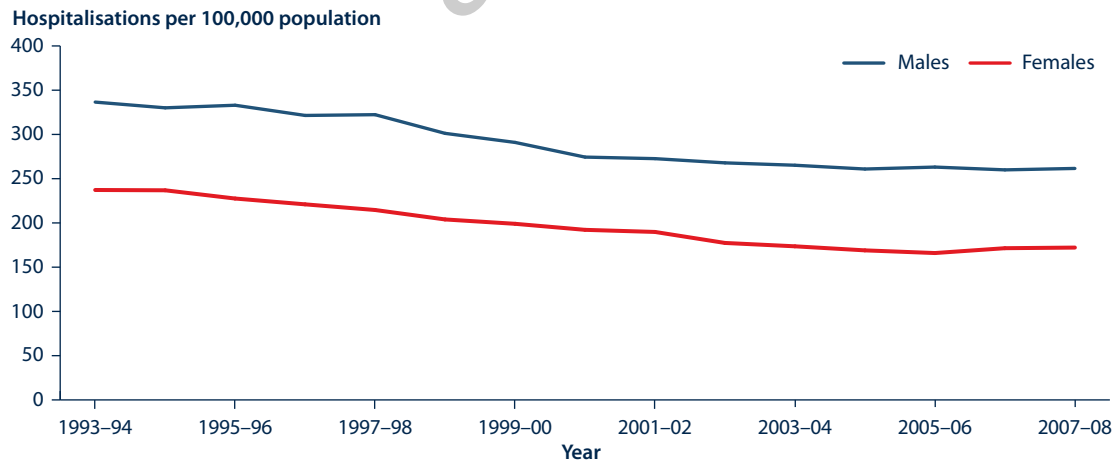
Trends

The number of people hospitalised with a principle diagnosis of heart failure or cardiomyopathy increased from 43,408 in 1993–94 to 49,307 in 2007–08. However, over the same period the population, especially the elderly population, increased at a greater rate, and as a result the age-adjusted hospitalisation rate with heart failure or cardiomyopathy has fallen— from 282 hospitalisations per 100,000 population in 1993–94 to 213 per 100,000 in 2007–08. Over this period, the age-adjusted rate for males was consistently higher than that for females (Figure 6.5).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 6.5: Heart failure and cardiomyopathy hospitalisation rates, principal diagnosis, by sex, 1993–94 to 2007–08

Health inequalities

The following section looks at heart failure and cardiomyopathy hospitalisations in subgroups of interest in the Australian population. Hospitalisation rates are presented for Indigenous and other Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2007–08 in the jurisdictions with adequate Indigenous identification, there were 1,274 hospitalisations with a principal diagnosis of heart failure or cardiomyopathy where the patient was recorded as Indigenous. The age-adjusted rate of 613 hospitalisations per 100,000 population for Indigenous Australians was almost 3 times as high as the rate for other Australians (218 per 100,000).

Indigenous identification in hospitalisation data is considered of sufficient quality for national reporting for New South Wales, Victoria, Queensland, Western Australia, South Australia and the Northern Territory (public hospitals) only. For further information refer to 'Reporting of Indigenous data' in Appendix A.

Remoteness

In 2007–08, the rate of hospitalisation with heart failure or cardiomyopathy was highest in *Remote and very remote* areas (340 hospitalisations per 100,000 population) and lowest in *Major cities* (205 per 100,000). Male rates were higher than female rates in all remoteness areas.

Socioeconomic group

In 2007–08, the age-adjusted rate of hospitalisation with heart failure or cardiomyopathy fell as socioeconomic position rose. The rate of hospitalisation in the lowest socioeconomic group was 1.5 times as high as that in the highest socioeconomic group—262 hospitalisations per 100,000 population compared with 171 per 100,000. The hospitalisation rate was higher for males than females across all socioeconomic groups (Figure 6.6).

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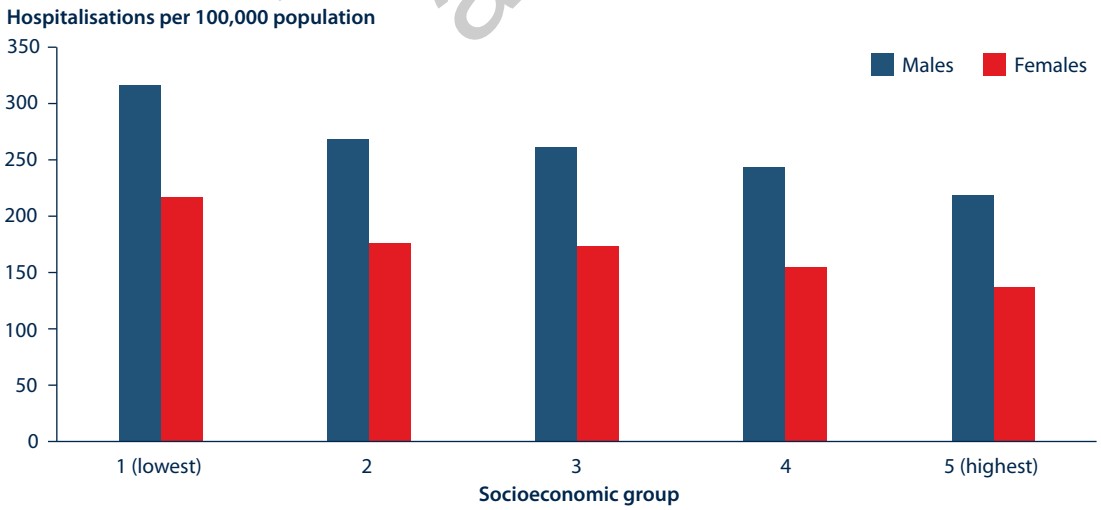

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 6.6: Heart failure and cardiomyopathy hospitalisation rates, principal diagnosis, by socioeconomic group and sex, 2007–08

Length of stay in hospital

The average length of stay for hospitalisations with a principal diagnosis of heart failure or cardiomyopathy has decreased steadily over time. Among those hospitalised for at least 1 night with heart failure or cardiomyopathy, the average length of stay decreased from 11.2 days in 1993–94 to 8.9 days in 2007–08.

Deaths in hospital

The rate of hospitalisations with a principal diagnosis of heart failure or cardiomyopathy, which ended in death, fell from 9% in 1993–94 to 8% in 2007–08. This death rate is similar to those for life-threatening and acute events, such as heart attack and stroke, indicating the severity of a diagnosis of heart failure or cardiomyopathy.

Deaths

In 2007, there were 4,055 deaths in Australia where heart failure or cardiomyopathy was the underlying (main) cause. However, when kidney failure, CHD, diabetes or chronic lower respiratory disease were recorded as the underlying cause of death, heart failure was frequently recorded as an associated cause—one that was considered to have contributed to the death but was not the main cause (AIHW 2008b). To maintain consistency throughout this report, the mortality data used are based on the underlying cause of death only.

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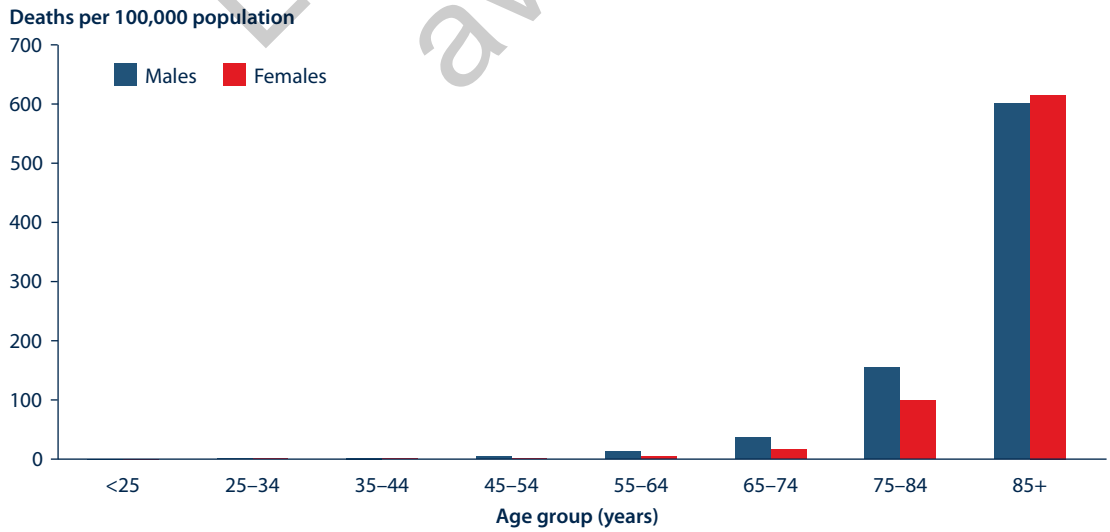
Sex and age

In 2007, nearly 90% of heart failure or cardiomyopathy deaths occurred among those aged 75 years and over. Age-specific death rates were higher among males than females across all age groups, except those aged 85 years and over (Figure 6.7).

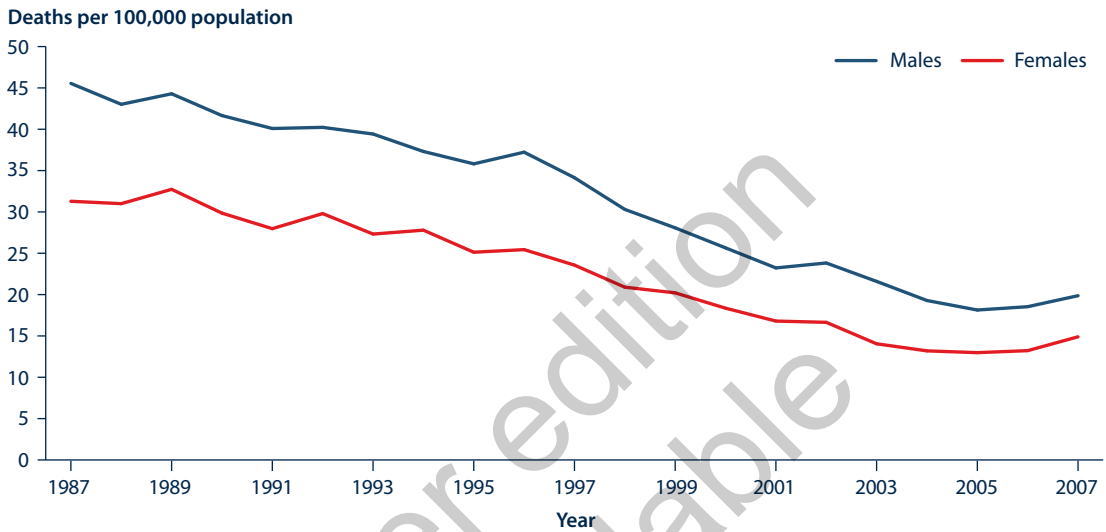


Source: AIHW National Mortality Database.

Figure 6.7: Heart failure and cardiomyopathy death rates, by age and sex, 2007

Trends

Since 1987, the age-standardised death rate from heart failure or cardiomyopathy has declined by almost half, falling from 38 deaths per 100,000 population to 17 per 100,000 in 2007. In more recent years this decline appears to have slowed slightly. Death rates from heart failure or cardiomyopathy were consistently higher for males than females for all years between 1987 and 2007 (Figure 6.8).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Figure 6.8: Heart failure and cardiomyopathy death rates, by sex, 1987–2007

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Health inequalities

The following section looks at heart failure and cardiomyopathy deaths in subgroups of interest in the Australian population. Death rates are presented for Indigenous and non-Indigenous Australians, and by remoteness areas and socioeconomic groups.

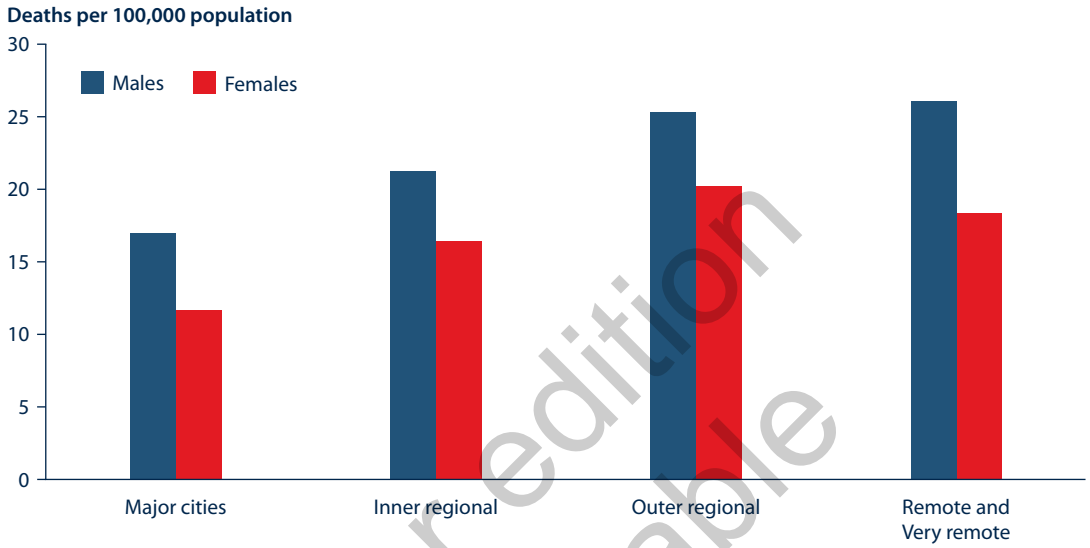
Aboriginal and Torres Strait Islander people

In 2005–2007, heart failure and cardiomyopathy was the underlying cause of death for 136 Indigenous Australians in the jurisdictions with adequate Indigenous identification. The age-standardised Indigenous death rate (32 per 100,000 population) was 1.4 times as high as the non-Indigenous rate (22 per 100,000). Indigenous males and females had heart failure and cardiomyopathy death rates 1.6 and 1.3 times as high as their non-Indigenous counterparts.

Indigenous identification in deaths data is considered of sufficient quality for national reporting for New South Wales, Queensland, Western Australia, South Australia and the Northern Territory only. For further information refer to 'Reporting of Indigenous data' in Appendix A.

Remoteness

In 2005–2007, age-standardised heart failure and cardiomyopathy death rates were lowest for those living in *Major cities* (14 deaths per 100,000 population) and highest for those in *Outer regional* areas (23 per 100,000). Males had higher heart failure and cardiomyopathy death rates than females in all remoteness areas (Figure 6.9).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Figure 6.9: Heart failure and cardiomyopathy death rates, by remoteness and sex, 2005–2007

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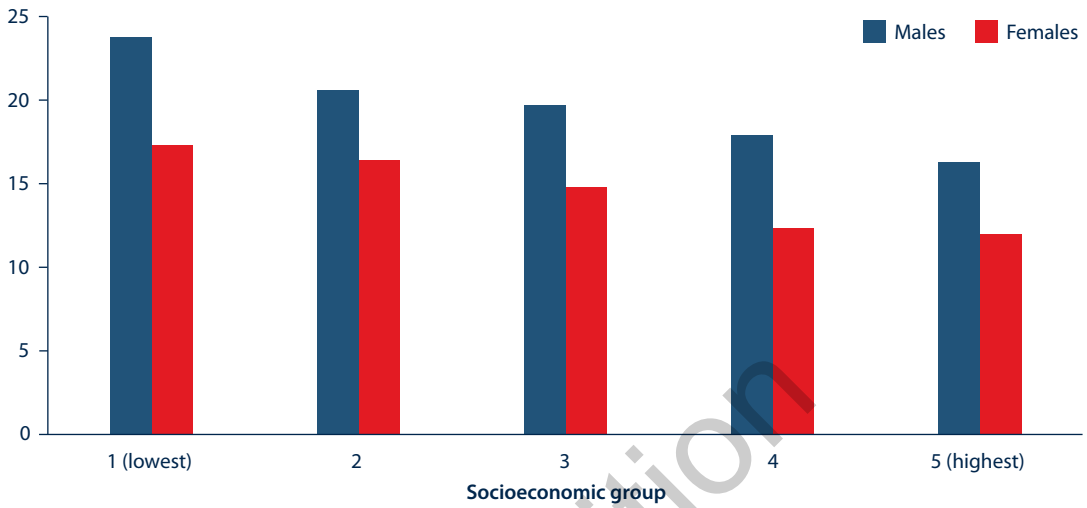
Socioeconomic group

In 2007, the age-standardised death rate for heart failure and cardiomyopathy for the lowest socioeconomic group (21 deaths per 100,000 population) was 1.5 times as high as the rate for the highest socioeconomic group (14 deaths per 100,000). The death rate among males was higher than for females across all socioeconomic groups (Figure 6.10).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database

Figure 6.10: Heart failure and cardiomyopathy death rates, by socioeconomic group and sex, 2007

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Burden of heart failure and cardiomyopathy

The contribution of heart failure and cardiomyopathy to the burden of disease and injury in Australia is included in the estimates for CHD because CHD is commonly the main cause of heart failure (Begg et al. 2007). For more information about the burden of CHD, refer to 'Chapter 4 Coronary heart disease'.

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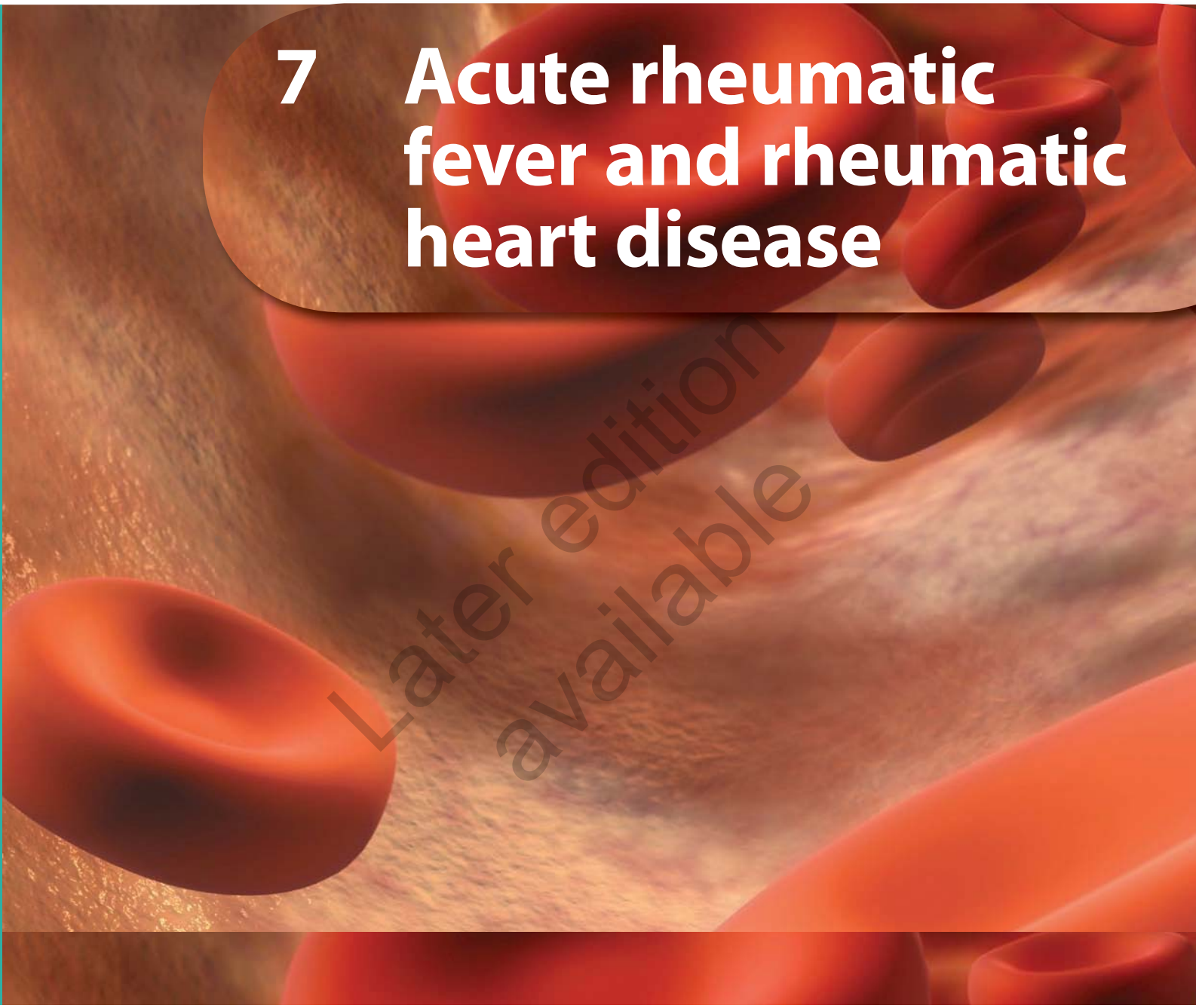

7 Acute rheumatic fever and rheumatic heart disease

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7 Acute rheumatic fever and rheumatic heart disease

What are acute rheumatic fever and rheumatic heart disease?

Acute rheumatic fever

Acute rheumatic fever (ARF) is a condition caused by an untreated infection of group A streptococcus (GAS) bacteria. The untreated infection can cause inflammation throughout the body including the heart, brain, skin and joints. Although the onset of ARF is dramatic and often painful, it causes no lasting damage to the brain, joints or skin. However, it can cause permanent damage to the heart and when this occurs it is known as rheumatic heart disease (RHD). Although ARF is now a rare disease among most Australians it still has a substantial impact on Aboriginal and Torres Strait Islander communities.

ARF usually develops from a GAS infection of the throat, but recent evidence suggests that it can also occur from skin infections in some populations, including Aboriginal and Torres Strait Islander people (McDonald et al. 2004). An untreated GAS infection leads to ARF in about 3% of cases (United States National Library of Medicine & National Institutes of Health 2003). Early detection and treatment of the GAS bacterial infection can prevent it progressing to ARF. The risk of ARF recurrence is high following an initial episode and repeated episodes of ARF increase the chance of long-term heart valve damage (NHFA & Cardiac Society of Australia and New Zealand 2006).

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Rheumatic heart disease

Rheumatic heart disease (RHD) manifests as permanent damage to the heart muscle or heart valves as a result of ARF. Such damage can reduce the ability of the heart to pump blood effectively around the body, leading to symptoms such as shortness of breath after exercise and feelings of fatigue and weakness. Severe forms of the disease can result in serious incapacity or even death.

Heart valve damage is a common presentation of RHD, often in the form of *stenosis* or *regurgitation*. Stenosis occurs when a heart valve becomes smaller and stiffer, obstructing the flow of blood. Regurgitation occurs when a valve fails to close properly and some blood moves back into the heart instead of around the body, reducing the output of blood from the heart.

These symptoms of RHD can also occur with other heart conditions, making a diagnosis more difficult. Signs of damage detected by echocardiography and a history of ARF are both important clinical indicators for RHD diagnosis.

Risk factors and prevention of acute rheumatic fever and rheumatic heart disease

Conditions such as poverty, overcrowding and poor sanitation, which are not uncommon in some Aboriginal and Torres Strait Islander communities, can contribute to the spread of GAS bacterial infections and so potentially to the development of ARF. In addition, a lack of access to medical care, which can also typify some remote communities, increases both the likelihood of recurrent episodes of ARF and of ARF progressing to RHD. The development of RHD can be prevented by treating ARF with antibiotics, an approach which has seen both conditions practically disappear in the 20th century among the non-Indigenous populations of developed countries. The cases that remain in these populations occur mostly as RHD among the elderly—a legacy of the higher childhood rates of ARF that were present in developed countries before the 1960s.

Implementing the intensive treatment regimens required for long-term prevention of ARF and RHD has proven difficult in Australia's remote Indigenous communities. Current guidelines recommend that secondary prevention of RHD requires a monthly injection of penicillin to be given for at least 10 years (NHFA & Cardiac Society of Australia and New Zealand 2006). In communities where socioeconomic disadvantage and lack of access to medical services are commonplace, effective prevention, diagnosis and treatment of the disease remain fundamental problems for their members.

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How many Australians have acute rheumatic fever and rheumatic heart disease?

It should be noted that it is standard practice to measure *prevalence* of RHD (the number of existing cases) and *incidence* of ARF (the number of new cases reported for a given period).

Prevalence of rheumatic heart disease

RHD is at low levels within Australia's non-Indigenous population which is why national prevalence estimates are available for the Indigenous population only. Results from the 2004–05 National Aboriginal and Torres Strait Islander Health Survey (NATSIHS) suggest that around 3,500 Indigenous Australians, or about 0.7% of the Indigenous population, had RHD in 2004–05.

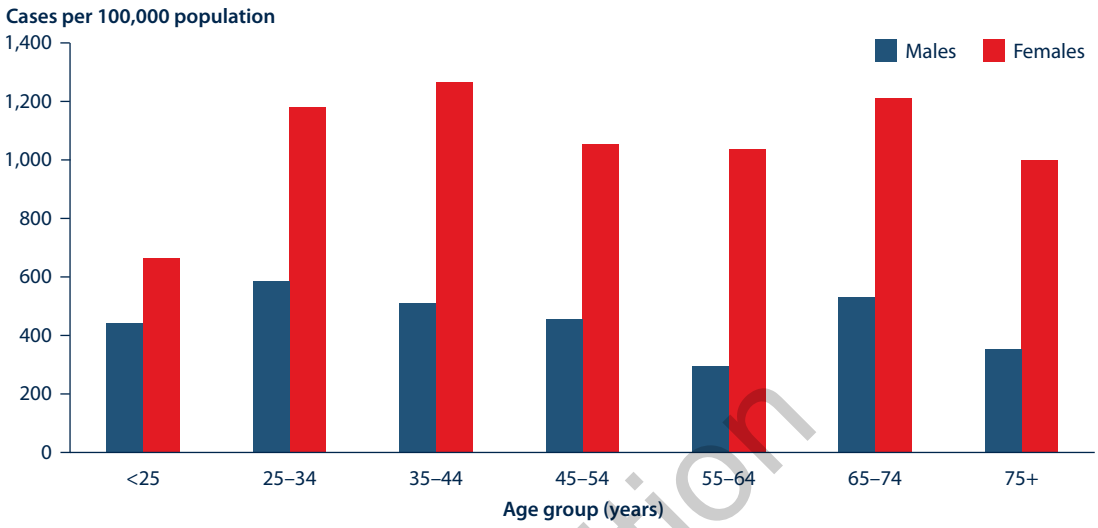
At the time of writing, the only state or territory level information on the prevalence of RHD was available from the Northern Territory RHD registry. It is anticipated, however, that more data will become available as register and control programs are developed in Western Australia and Queensland under the Australian Government's *Rheumatic Fever Strategy* announced in January 2010.

The prevalence data presented in this section are from the Northern Territory registry and it cannot be assumed that they are representative of the rest of Australia.

Rheumatic heart disease prevalence in the Northern Territory

Sex and age

In 2009, there were 1,480 cases of RHD in the Northern Territory—969 females and 511 males. Prevalence rates did not vary much with age for either sex and were higher among females than males in all age groups (Figure 7.1).



Source: Northern Territory RHD registry.

Figure 7.1: Rheumatic heart disease prevalence rates in the Northern Territory, by age and sex, 2009

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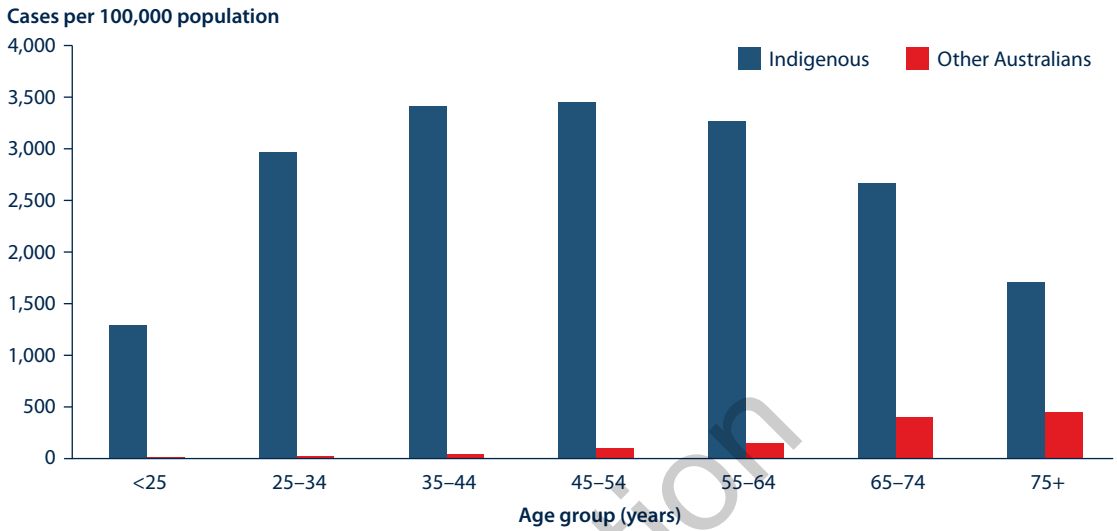
Trends

The prevalence of RHD in the Northern Territory appears to be increasing. Between 2004 and 2009 the rate increased from 565 cases per 100,000 population to 658. It is possible that this increase is the result of improved diagnosis and data collection rather than a true increase in the prevalence of the disease.

Aboriginal and Torres Strait Islander people

In 2009, RHD was much more prevalent among Indigenous Australians in the Northern Territory (1,374 cases) than it was among other Australians (105 cases). After adjusting for the different age structures of the populations, the prevalence rate among Indigenous Australians was 25 times as high as for other Australians—2,466 compared to 97 cases per 100,000 population.

Among Indigenous Australians, RHD prevalence rates were high across all age groups, with the highest being among those aged 35–64 years. In contrast, prevalence rates among other Australians increased steadily with age with the highest rate found among those aged 75 years and over. The most marked differences between Indigenous and other Australians occurred in younger age groups, where RHD was rare for other Australians (Figure 7.2).



Source: Northern Territory RHD registry.

Figure 7.2: Rheumatic heart disease prevalence rates, Northern Territory, by age and Indigenous status, 2009

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International comparisons

Worldwide, it is estimated there are at least 15.6 million people with RHD (Carapetis et al. 2005). In developing countries it has been estimated that acute rheumatic fever and rheumatic heart disease are responsible for almost half of the cardiovascular disease in all age groups and are among the leading causes of death in the first 5 decades of life (Limbu & Maskey 2002).

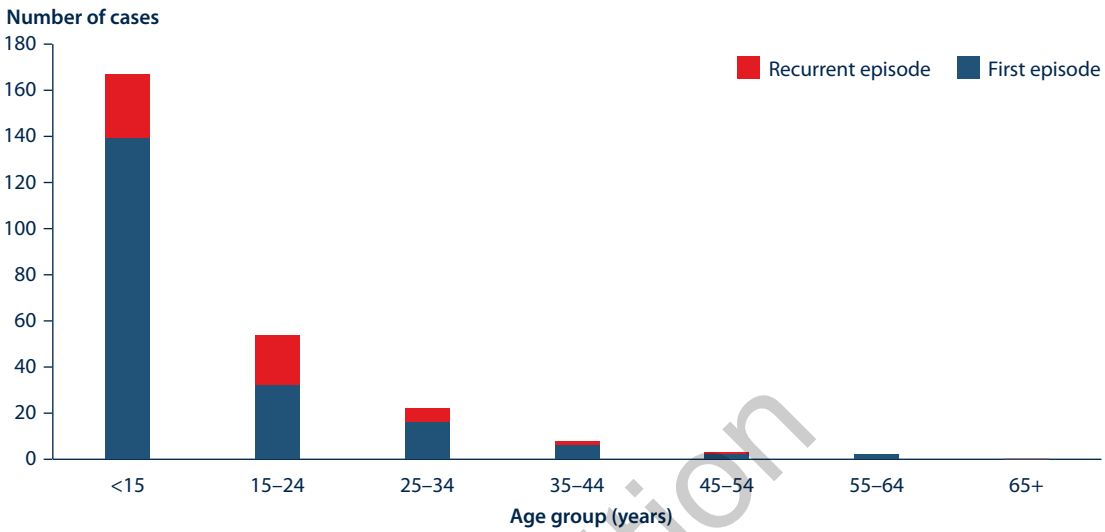
Internationally, the highest regional RHD prevalence rates were found in sub-Saharan Africa (570 cases per 100,000 population), the Pacific and the Indigenous populations of Australia and New Zealand (350 cases per 100,000 population) and south Central Asia (220 cases per 100,000 population) (Carapetis et al. 2005).

Incidence of acute rheumatic fever

No national data on ARF incidence (new cases of the disease) are available. The only jurisdictional data come from the Northern Territory RHD registry which collects information on new and recurrent cases and other demographic variables. Virtually all new cases of ARF in the Northern Territory involve Indigenous people so the results that follow refer to the incidence of the disease in that population only.

ARF incidence in the Northern Territory

In the Northern Territory, 259 cases of ARF were recorded between 2005 and 2009. All but three of these cases were for Indigenous people. Most of these (199) were new episodes of ARF, while the remaining 60 were recurrent episodes. New cases of ARF outnumber recurrent cases in all age groups (Figure 7.3). The number of recurrent cases among those aged below 25 years suggests that the long-term prevention of ARF has not been successful for a large number of those people who have been treated for the disease. To treat ARF successfully and prevent its recurrence, antibiotics must be taken monthly over a 10-year period. Such a treatment regime relies on the provision of continuous medical support that may not always be available in remote Indigenous communities.



Note: Indigenous Australians only.

Source: Northern Territory RHD registry.

Figure 7.3: Acute rheumatic fever incidence, Northern Territory, by age and episode type, 2005–2009

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The Northern Territory results closely resemble findings from a recent study of ARF incidence in North Queensland. In that study, 94% of people with ARF were Indigenous and the majority of cases (63%) were new episodes of ARF (Hanna & Heazlewood 2005).

Age and sex

In 2009, the Northern Territory RHD registry recorded more ARF cases among Indigenous females (27) than Indigenous males (18). ARF disproportionately affected the young, with 65% of cases occurring in people aged less than 15 years and a further 21% occurring in people aged 15–24 years.

Trends

Between 2005 and 2009, the number of new and recurrent cases of ARF in the Northern Territory varied little. More Indigenous females were diagnosed with the disease than Indigenous males in each of those years.

International comparisons

International comparisons of ARF incidence are difficult to make and for many regions (including China and sub-Saharan Africa) no studies are available. Recent estimates report over 470,000 new cases of ARF occur each year, with 95% of new cases coming from developing countries (Carapetis et al. 2005).

Based on these estimates, the highest ARF incidence rates are among children aged 5–14 years in the grouped Pacific Island and Indigenous Australian and New Zealand populations, with 374 cases per 100,000 people. By comparison, the lowest rates are in non-Indigenous Australian and New Zealand populations, with 10 cases per 100,000 population (Carapetis et al. 2005).

Hospitalisations

Because hospital records may not always distinguish between ARF and RHD, the two diseases are grouped together in this section. In 2007–08, there were 2,701 hospitalisations with a principal diagnosis of ARF and RHD—0.6% of all CVD hospitalisations and equating to an age-standardised rate of 12 hospitalisations per 100,000 population. Note that hospitalisations data in this report are based on 'episodes of care' rather than the number of people hospitalised with a condition (see 'Reporting hospitalisations' in Appendix A).

Sex and age

With the exception of those aged 85 years and over, females had higher rates of hospitalisation with ARF and RHD than males in 2007–08. Hospitalisation rates increased sharply with age with the rate among those aged 65–74 years nearly three times as high as those aged 55–64 years. The highest rates for both males and females were in those aged 75–84 years—63 and 69 hospitalisations per 100,000 population (Figure 7.4).

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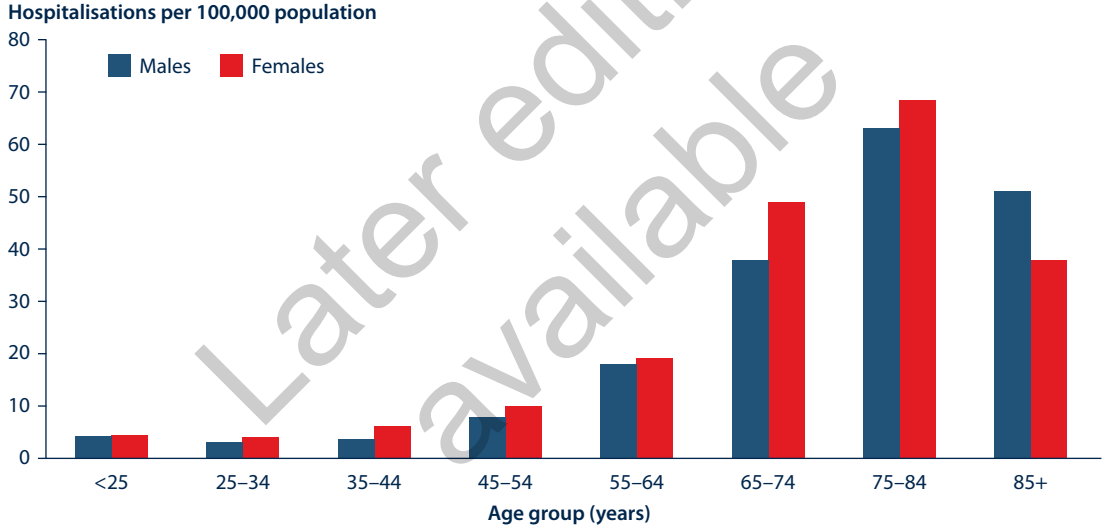
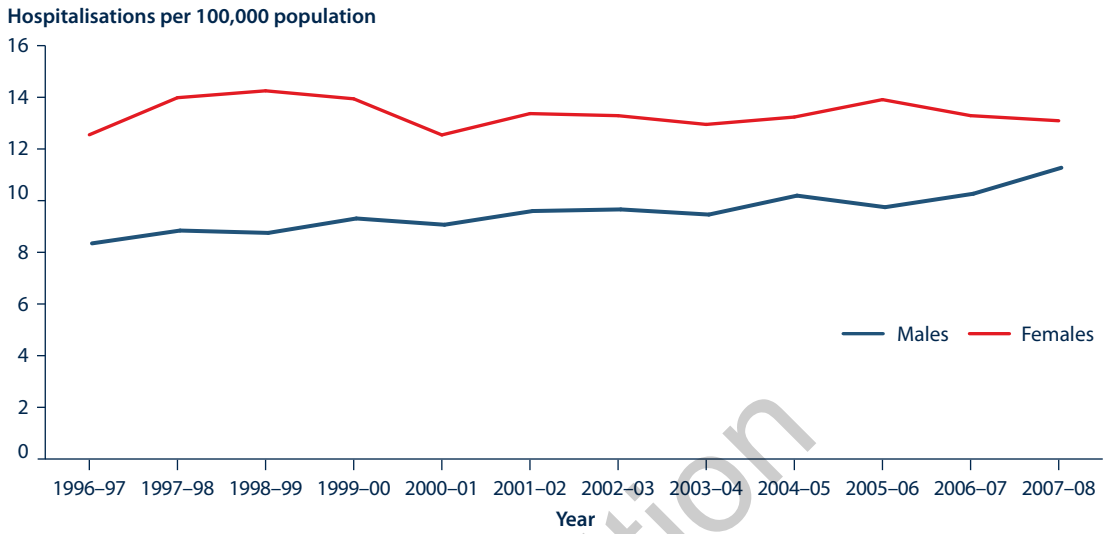


Source: AIHW National Hospital Morbidity Database.

Figure 7.4: Acute rheumatic fever and rheumatic heart disease hospitalisation rates, principal diagnosis, by age and sex, 2007–08

Trends

Between 1996–97 and 2007–08 the number of hospitalisations with ARF and RHD increased from 1,864 to 2,701. Over this period the age-standardised hospitalisation rate for ARF and RHD increased slightly, from 11 to 12 per 100,000 and females had consistently higher rates than males (Figure 7.5).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 7.5: Acute rheumatic fever and rheumatic heart disease hospitalisation rates, principal diagnosis, by sex, 1996–97 to 2007–08

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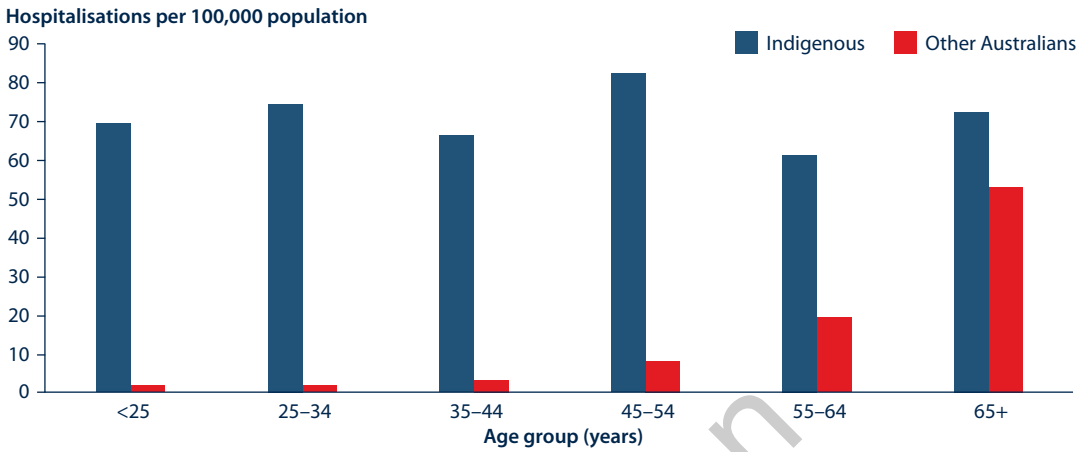
Health inequalities

The following section looks at acute rheumatic fever and rheumatic heart disease hospitalisations in subgroups of interest in the Australian population. Hospitalisation rates are presented for Indigenous and other Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2007–08 in the jurisdictions with adequate Indigenous identification, there were 347 hospitalisations with ARF and RHD where the patient was recorded as Indigenous. The overall hospitalisation rate for the Indigenous population (67 per 100,000 people) was eight times as high as the rate for other Australians and was higher in all age groups, although the rates were much closer after 65 years of age (Figure 7.6).

Indigenous identification in hospitalisation data is considered of sufficient quality for national reporting for New South Wales, Victoria, Queensland, Western Australia, South Australia and the Northern Territory (public hospitals) only. For further information refer to 'Reporting of Indigenous data' in Appendix A.



Note: Data from NSW, Vic, Qld, WA, SA and NT (public hospitals) only.

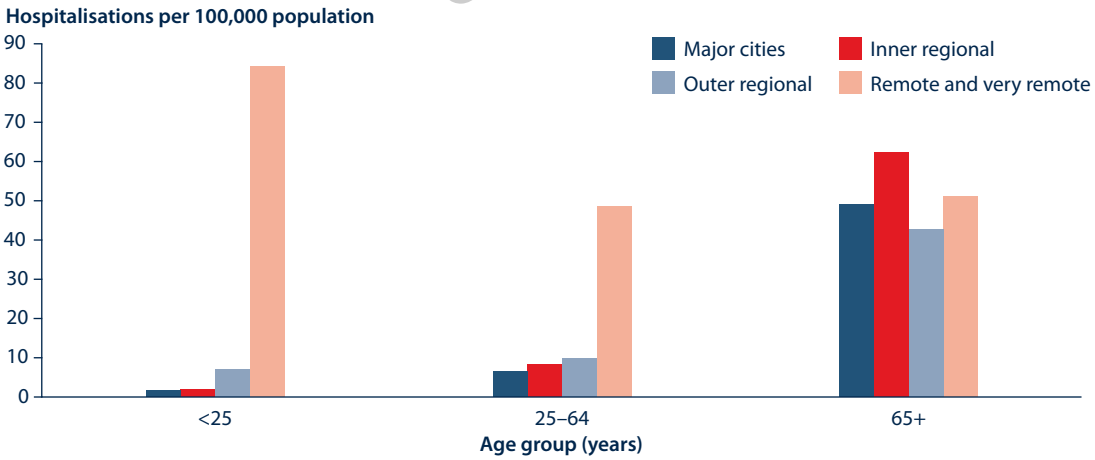
Source: AIHW National Hospital Morbidity Database

Figure 7.6: Acute rheumatic fever and rheumatic heart disease hospitalisation rates, principal diagnosis, by age and Indigenous status, 2007–08

Later edition available

Remoteness

In 2007, the age-specific hospitalisation rate with ARF and RHD for those aged less than 25 years was 84 per 100,000 population in *Remote and very remote* areas but only 2 per 100,000 in *Major cities* (Figure 7.7). The *Remote and very remote* rates reflect both the high proportion of Indigenous Australians living in these areas and that Indigenous Australians in remote areas continue to experience new cases of ARF and RHD, often at a young age. In non-remote areas, hospitalisations with ARF and RHD occur mostly among older people.



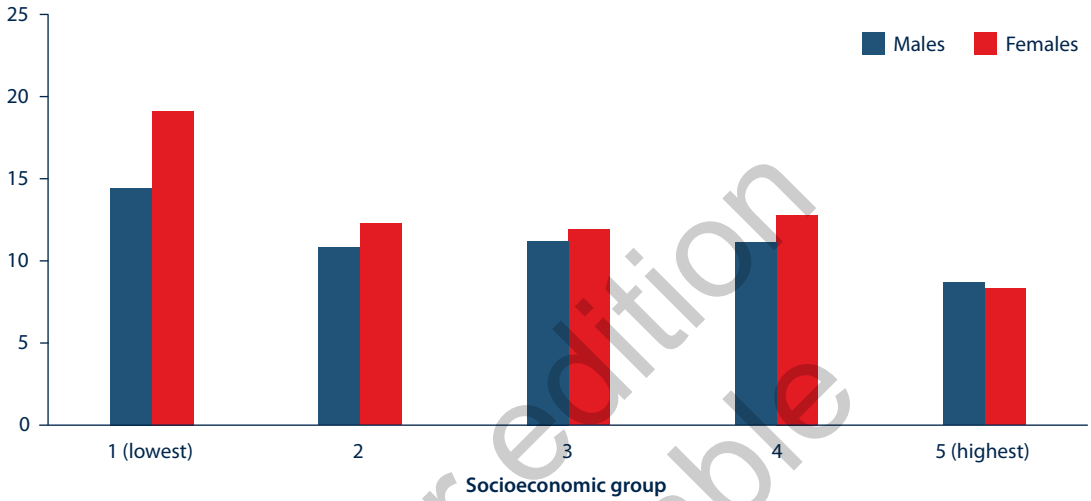
Note: Based on area of usual residence of patient.

Source: AIHW National Hospital Morbidity Database.

Figure 7.7: Acute rheumatic fever and rheumatic heart disease hospitalisation rates, principal diagnosis, by age and remoteness, 2007–08

Socioeconomic group

In 2007–08, the ARF and RHD hospitalisation rate for both males and females was highest in the lowest socioeconomic groups and lowest in the highest socioeconomic groups. Male and female rates did not vary greatly except in the lowest group where the female rate (19 per 100,000 population) was higher than that for males (14 per 100,000 population) (Figure 7.8).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 7.8: Acute rheumatic fever and rheumatic heart disease hospitalisation rates, by socioeconomic group and sex, 2007–08

Later edition available

Length of stay in hospital

In 2007–08, 29% of people admitted to hospital with ARF and RHD were discharged the same day—a substantial decrease from 48% in 1993–94.

Deaths in hospital

In 2007–08, 2.4% of hospitalisations with ARF and RHD ended in death. This proportion has remained stable since 1993–94.

International comparisons

There is limited recent information on ARF and RHD hospitalisations in other countries. New Zealand data from 2002–06 show differences in hospitalisation rates between some ethnic groups. For example, among people aged 0–24 years, there were 1.3 hospitalisation admissions for ARF and RHD per 100,000 population of European New Zealanders, 29 per 100,000 for Maori New Zealanders and 62 per 100,000 for Pacific Islander New Zealanders (Craig et al. 2007).

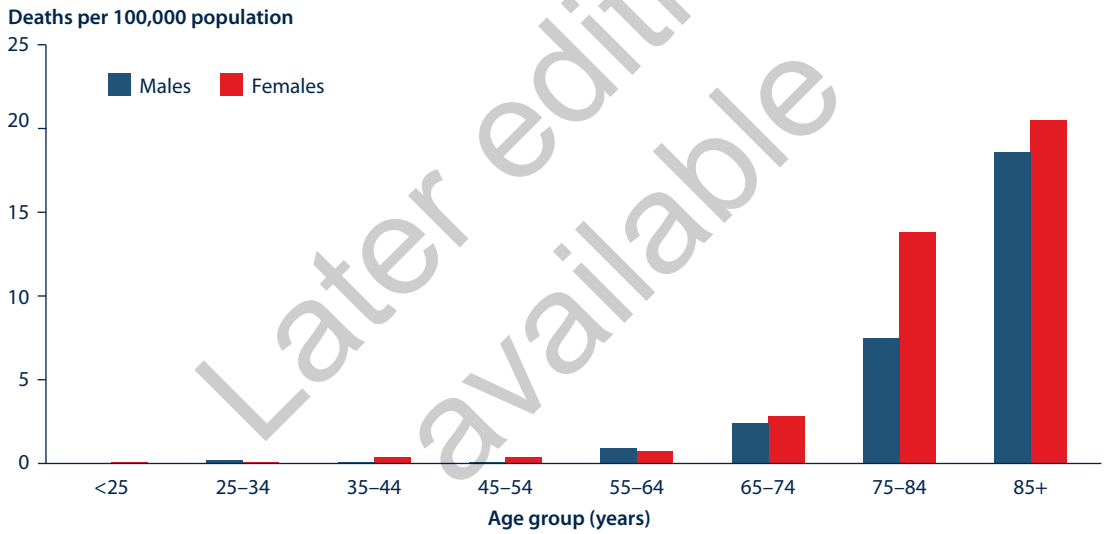
Deaths

Deaths from ARF and RHD are uncommon in Australia and, as with hospitalisations, death records may not distinguish well between ARF and RHD so the two conditions are presented here together. In 2007, 255 people in Australia died from ARF and RHD, representing 0.2% of all deaths and 0.5% of CVD deaths.

Sex and age

Unlike many other forms of CVD, more females die from ARF and RHD than males. In 2007, females accounted for almost two-thirds of ARF and RHD deaths—167 compared with 88 for males. The age-adjusted death rate for females (1.3 per 100,000 population) was 44% higher than the male rate (0.9 per 100,000).

Death rates increased rapidly with age, with the highest rates among those aged 85 years and over (Figure 7.9). Almost 85% of deaths occurred among people aged 65 years and over.



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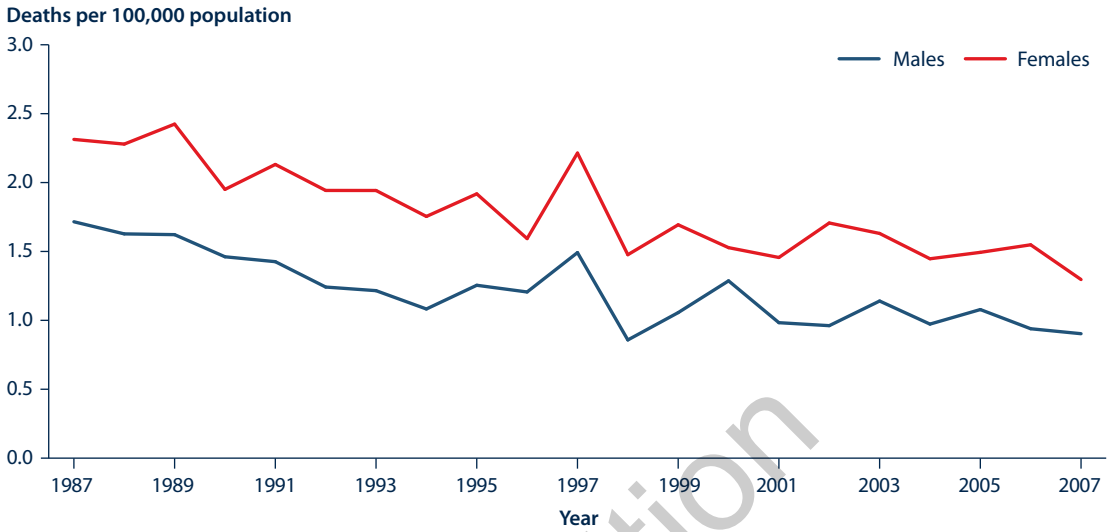
Source: AIHW National Mortality Database.

Figure 7.9: Acute rheumatic fever and rheumatic heart disease death rates, by age and sex, 2007

Trends

Deaths rates from ARF and RHD have continued to decline strongly in recent years. In 2007, age-adjusted death rates for both males and females were just over a third as high as those in 1987. Death rates were consistently higher for females than males between 1987 and 2007 (Figure 7.10).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Figure 7.10: Acute rheumatic fever and rheumatic heart disease death rates, by sex, 1987–2007

Later edition available

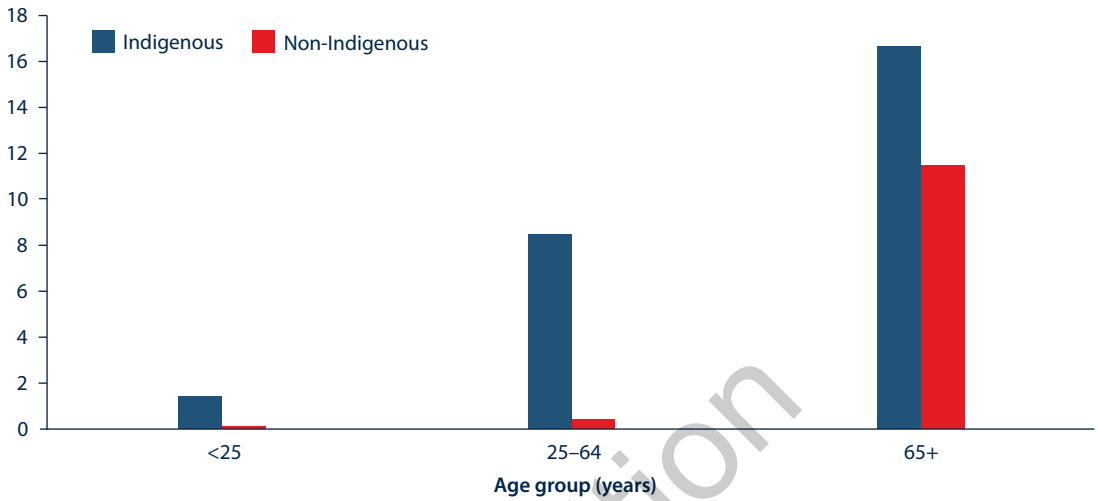
Health inequalities

The following section looks at acute rheumatic fever and rheumatic heart disease deaths in subgroups of interest in the Australian population. Death rates are presented for Indigenous and non-Indigenous Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2005–2007, there were 65 ARF or RHD deaths among Indigenous Australians. The age-adjusted death rate for Indigenous Australians (8 deaths per 100,000 population) was four times as high as the non-Indigenous rate. The difference in death rates is especially evident among females, where the Indigenous rate was over six times as high as the non-Indigenous rate. Among males, the Indigenous rate was about three times as high. Eleven Indigenous Australians aged less than 25 years died from ARF and RHD in 2005–2007 while among non-Indigenous Australians of the same age there were no deaths. In the 25–64 year age group the Indigenous death rate (9 per 100,000) was 20 times greater than the non-Indigenous rate (Figure 7.11). For both Indigenous and non-Indigenous Australians, death rates were highest for people aged 85 years and over—69 and 33 deaths per 100,000. These rates are sharply higher than for those aged 65 years or over—17 Indigenous deaths and 12 non-Indigenous per 100,000.

Indigenous identification in deaths data is considered of sufficient quality for national reporting for New South Wales, Queensland, Western Australia, South Australia and the Northern Territory only. For further information refer to 'Reporting of Indigenous data' in Appendix A.



Note: Data for NSW, Qld, WA, SA and NT only.

Source: AIHW National Mortality Database.

Figure 7.11: Acute rheumatic fever and rheumatic heart disease death rates, by age and Indigenous status, 2005–2007

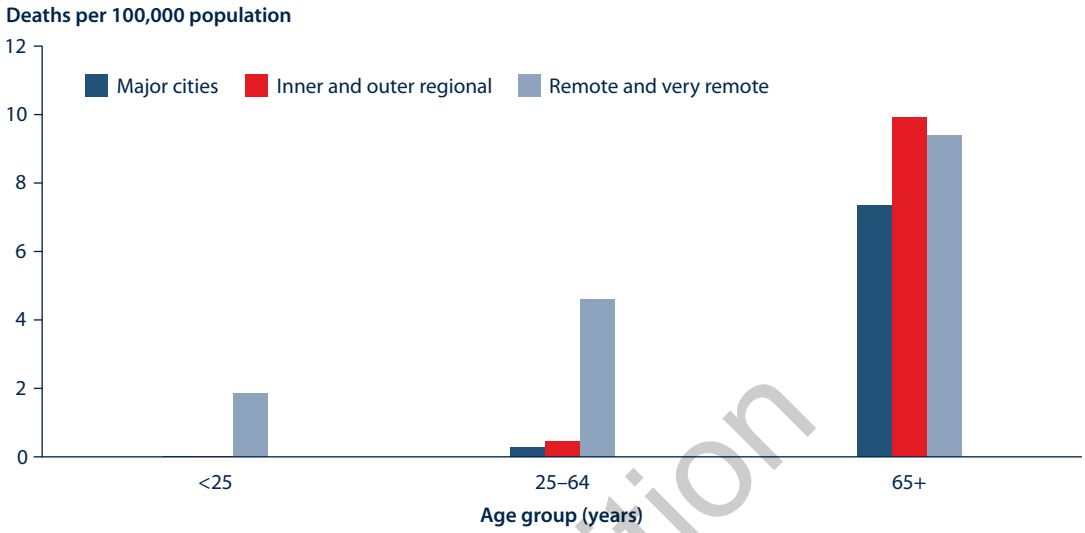
Later edition available

Remoteness

In 2005–2007, age-standardised death rates for ARF and RHD increased with remoteness, from 1 death per 100,000 population in *Major cities* to 4 per 100,000 in *Remote and very remote* areas. (Note, because of the small number of ARF and RHD deaths, the *Inner regional* and *Outer regional* areas have been combined and separate rates for males and females have not been presented here.)

ARF and RHD death rates varied markedly with age between regions. In *Major cities* and *Inner and outer regional* areas, death rates for people younger than 65 years were much lower than those in *Remote and very remote* areas. While in all remoteness areas, death rates were highest for those aged 65 years and over, in *Remote and very remote* areas 18% of deaths occurred among those aged below 25 years. In *Major cities* this figure was only 0.7% (Figure 7.12).

The differences between regions are closely related to Indigenous status. In *Remote and very remote* areas, Aboriginal and Torres Strait Islander people comprise a high proportion of the overall population. As a result, the patterns of deaths across age groups seen among Indigenous Australians resemble those of the most remote areas, especially in the form of elevated death rates in younger age groups.



Note: Inner and Outer regional areas and Remote and Very remote areas have been combined due to small numbers.

Source: AIHW National Mortality Database.

Figure 7.12: Acute rheumatic fever and rheumatic heart disease death rates, by age and remoteness, 2005–2007

Later edition available

Socioeconomic group

ARF and RHD death rates did not differ between socioeconomic groups or between males and females within socioeconomic groups.

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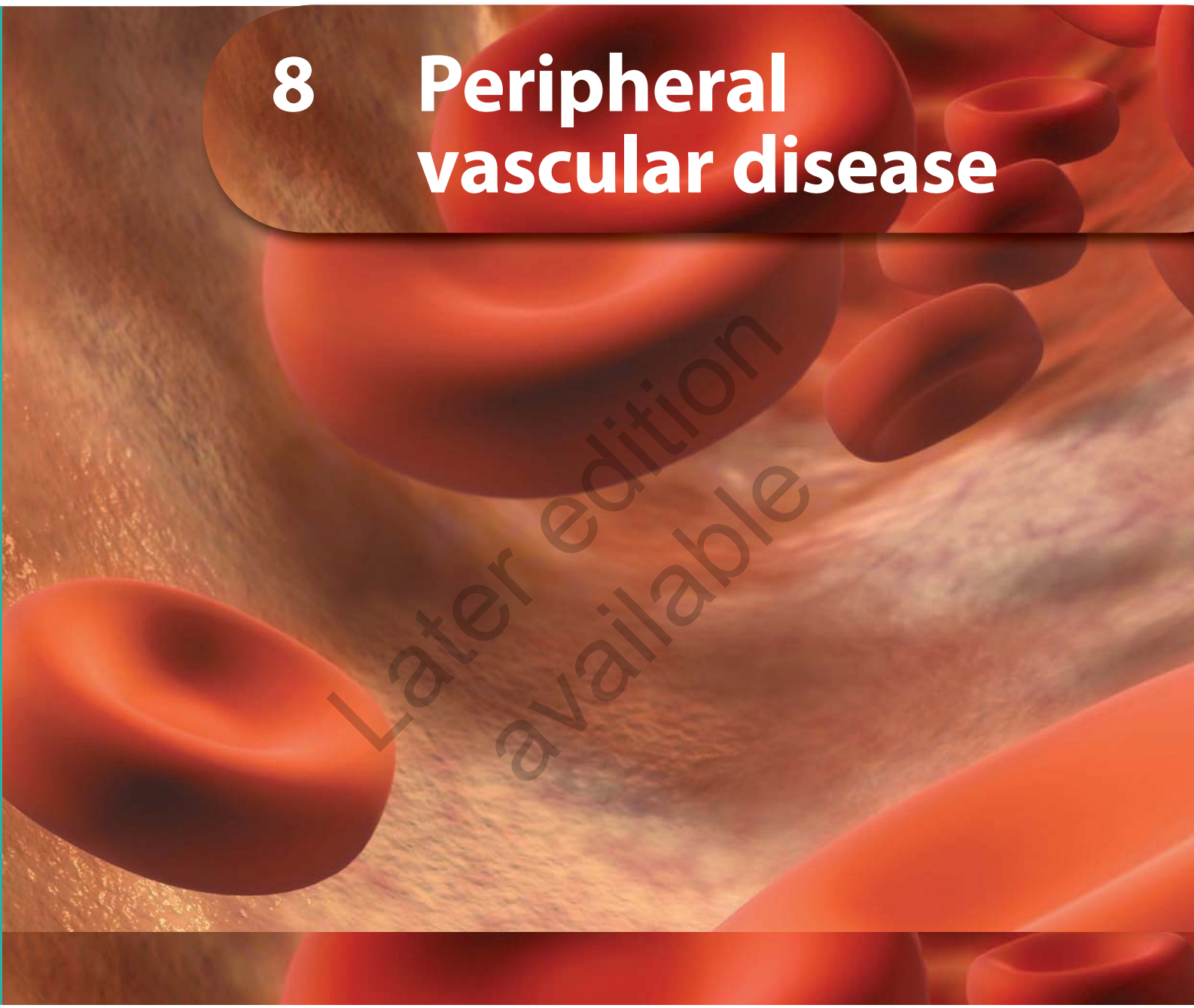

8 Peripheral vascular disease

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8 Peripheral vascular disease

What is peripheral vascular disease?

Peripheral vascular disease (PVD), also known as *peripheral arterial disease*, refers to the obstruction of large arteries that supply blood to the peripheries, which excludes the central organs of the heart and brain. Two important forms of PVD are *atherosclerosis of the peripheral arteries* (APA) and *abdominal aortic aneurysm* (AAA).

APA is a condition whereby peripheral arteries are narrowed by *atherosclerosis*—the deposit of fatty substances within the blood vessels—resulting in reduced blood flow to the peripheral areas of the body. APA most commonly affects the arteries supplying blood to the legs and feet. In some people it does not present any symptoms, while others may experience pain at rest or while walking. In severe cases APA can lead to the amputation of a limb.

AAA is described as the abnormal widening of the aorta (the main artery leading from the heart) below the level of the diaphragm. It can be a life-threatening condition if the arterial wall ruptures. Surgery is necessary in some cases.

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Risk factors for peripheral vascular disease

Tobacco smoking is the primary and most important risk factor for PVD (Stevens et al. 2006). Other PVD risk factors include diabetes, high blood cholesterol levels, high blood pressure, and overweight and obesity, all of which are common to most forms of CVD (See 'Chapter 2 Risk factors for cardiovascular disease').

How many Australians have peripheral vascular disease?

Currently, there are no national data available on the number of Australians who have PVD, although a study published in 2002 (Fowler et al. 2002) showed an age-standardised prevalence of 16% among men aged 65–83 years, living in the Perth metropolitan area of Western Australia.

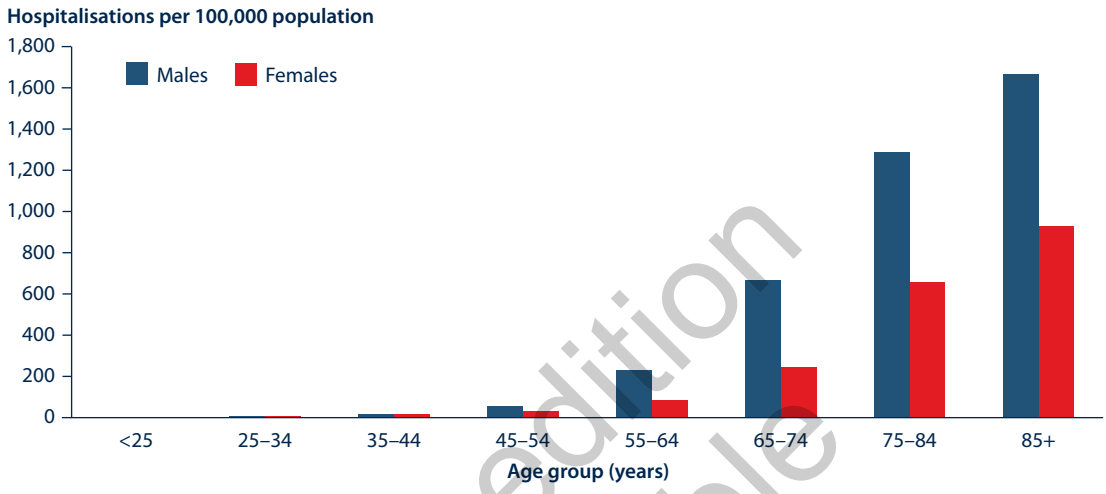
Hospitalisations

In 2007–08, there were 25,796 hospitalisations in Australia with PVD. This equated to 0.3% of all hospitalisations and 5% of CVD hospitalisations. Over half of all PVD hospitalisations (56%) were for APA while AAA accounted for a further 18%. The remainder was comprised largely of embolisms and other aneurysms. Note that hospitalisations data in this report are based on 'episodes of care' rather than the number of people hospitalised with a condition (Appendix A).

Sex and age

In 2007–08, the age-standardised rate of PVD hospitalisations for males (159 hospitalisations per 100,000 population) was more than twice as high as the rate for females (74 per 100,000). The male rate was higher than the female rate in all age groups, and rates increased markedly with age. The rate for people aged 75 years and over was twice as high as the rate for those aged 65–74 years and six times as high as for those aged 55–64 years. Over three-quarters of all hospitalisations for PVD were for those aged 65 years and over (Figure 8.1).

The age-standardised male hospitalisation rate with a primary diagnosis of AAA (37 per 100,000 population) was over five times as high as the female rate (7 per 100,000) and 89% of all AAA hospitalisations occurred among those aged 65 years and over. Similarly, the male hospitalisation rate with APA (85 per 100,000) was twice that for females (44 per 100,000) and 80% of hospitalisations with APA were for those aged 65 years and over.



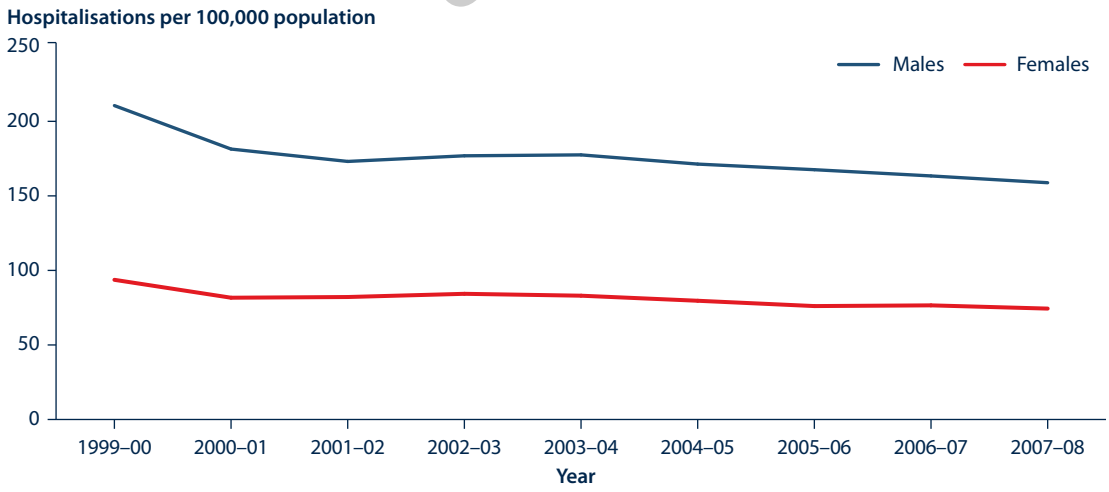
Source: AIHW National Hospital Morbidity Database.

Figure 8.1: Peripheral vascular disease hospitalisation rates, principal diagnosis, by age and sex, 2007–08

Later edition available

Trends

Between 1999–00 and 2007–08, age-standardised hospitalisation rates for PVD decreased by 23%. Male rates remained twice as high as female rates over the period (Figure 8.2).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database

Figure 8.2: Peripheral vascular disease hospitalisation rates, principal diagnosis, by sex, 1999–00 to 2007–08

Health inequalities

The following section examines PVD hospitalisations in subgroups of interest in the Australian population. Hospitalisation rates are presented for Indigenous and other Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2007–08, there were 160 hospitalisations with PVD in the jurisdictions with adequate Indigenous identification where the patient was identified as an Aboriginal or Torres Strait Islander. Overall, the age-standardised hospitalisation rate for other Australians (118 per 100,000 population) was 1.5 times as high as the rate for Indigenous Australians (80 per 100,000). The difference between the rates was almost exclusively the result of the high rate for other Australian males (168 per 100,000). PVD hospitalisation rates for Indigenous males and females and other Australian females were quite similar—around 80 per 100,000 (Figure 8.3). Indigenous identification in hospitalisation data is considered of sufficient quality for national reporting for New South Wales, Victoria, Queensland, Western Australia, South Australia and the Northern Territory (public hospitals) only. For further information refer to 'Reporting of Indigenous data' in Appendix A.

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Notes

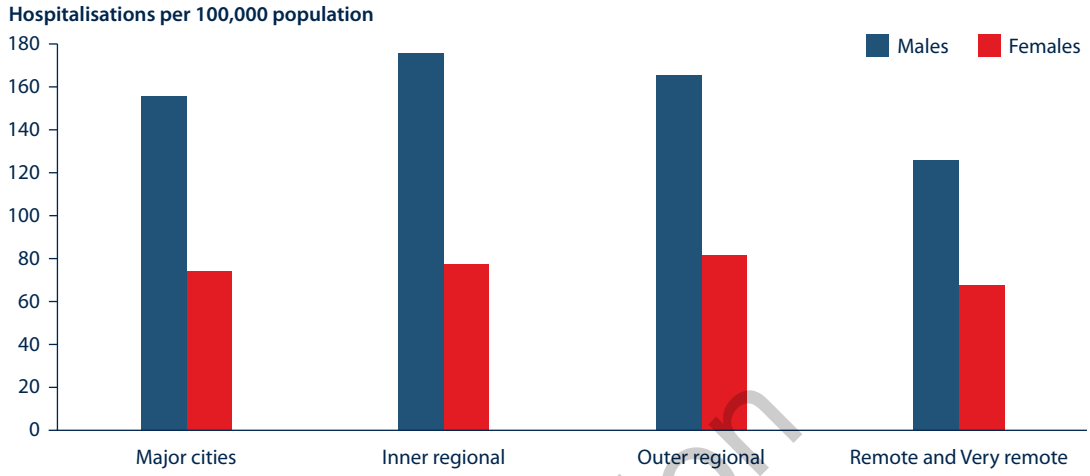
1. Age-standardised to the 2001 Australian population.
2. Data from NSW, Vic, Qld, WA, SA and NT (public hospitals) only.

Source: AIHW National Hospital Morbidity Database.

Figure 8.3: Peripheral vascular disease hospitalisation rates, principal diagnosis, by sex and Indigenous status, 2007–08

Remoteness

In 2007–08, hospitalisation rates for PVD were highest in *Inner regional* and *Outer regional* areas (123 per 100,000 population) and lowest in *Remote and very remote* areas (99 per 100,000). Male hospitalisation rates for PVD were about twice as high as female rates in all remoteness areas (Figure 8.4).



Notes:

1. Age-standardised to the 2001 Australian population.
2. Based on area of usual residence of patient.

Source: AIHW National Hospital Morbidity Database.

Figure 8.4: Peripheral vascular disease hospitalisation rates, principal diagnosis, by remoteness and sex, 2007–08

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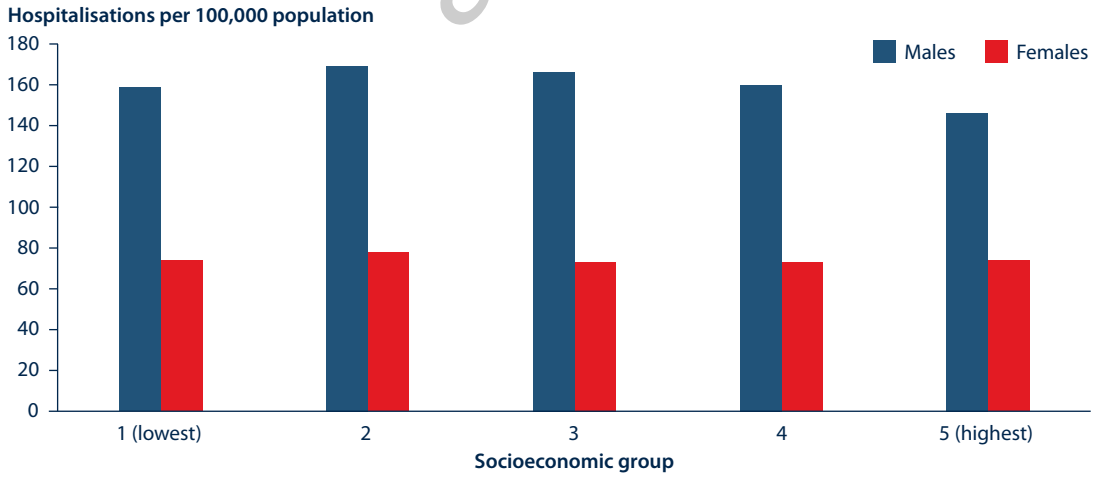
Socioeconomic group

In 2007–08, the PVD hospitalisation rate in the highest socioeconomic group (105 hospitalisations per 100,000 population) was lower than that for other socioeconomic groups, which showed little variation between each other. The male rate was at least twice as high the female rate across all socioeconomic groups (Figure 8.5).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 8.5: Peripheral vascular disease hospitalisation rates, principal diagnosis, by socioeconomic group and sex, 2007–08

Length of stay in hospital

In 2007–08, the average length of stay for those hospitalised with PVD for at least one night was 10.8 days—down from 12.8 days in 1993–94. The decrease in duration over this period was steady apart from small increases in 2002–03 and 2005–06. Hospitalisation data indicate that the average length of stay for a diagnosis of PVD was twice as long as for a diagnosis of CHD.

Deaths in hospital

In 2007–08, 5% of hospitalisations with PVD as the principal diagnosis ended in death— a steady decline from the 1993–94 rate of 6.4%. Between 1993–94 and 2007–08, PVD deaths comprised 8% of all CVD deaths in hospital. Around 38% of PVD deaths occurring in 2007–08 were among people admitted with AAA, with the in-hospital death rate for this condition twice as high as that for PVD overall. Despite this, the in-hospital death rate for AAA fell from 17% in 1993–94 to 10% in 2007–08.

Deaths

PVD was the cause of 2,160 deaths in 2007—equating to 1.6% of all deaths, and 4.6% of all CVD deaths. AAA accounted for 32% of PVD deaths with the remainder resulting from APA, other aneurysms, embolisms and unspecified PVD. Age remained a substantial risk factor with 92% of PVD deaths occurring among those aged 65 years and over. The major cause of death in people diagnosed with PVD was, however, CHD (AIHW 2008c).

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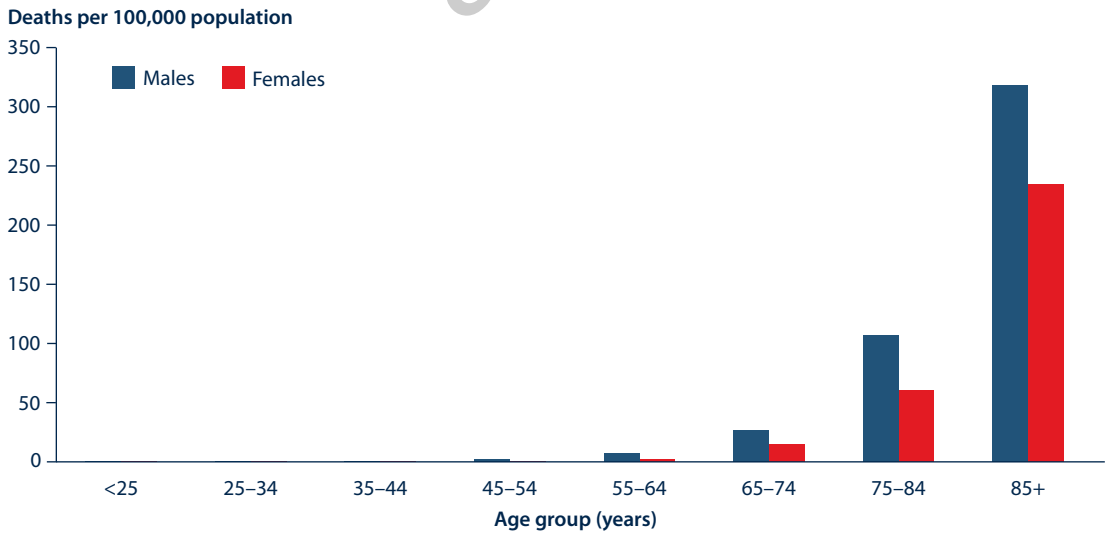
Sex and age

In 2007, the age-standardised death rate for PVD was higher among males (12 per 100,000 population) than females (7 per 100,000). For those aged over 65 years, the male death rate (80 deaths per 100,000) was 1.3 times as high as the female rate (64 per 100,000) (Figure 8.6).

Deaths from PVD increase greatly with age, with 78% occurring among those aged 75 years and over.

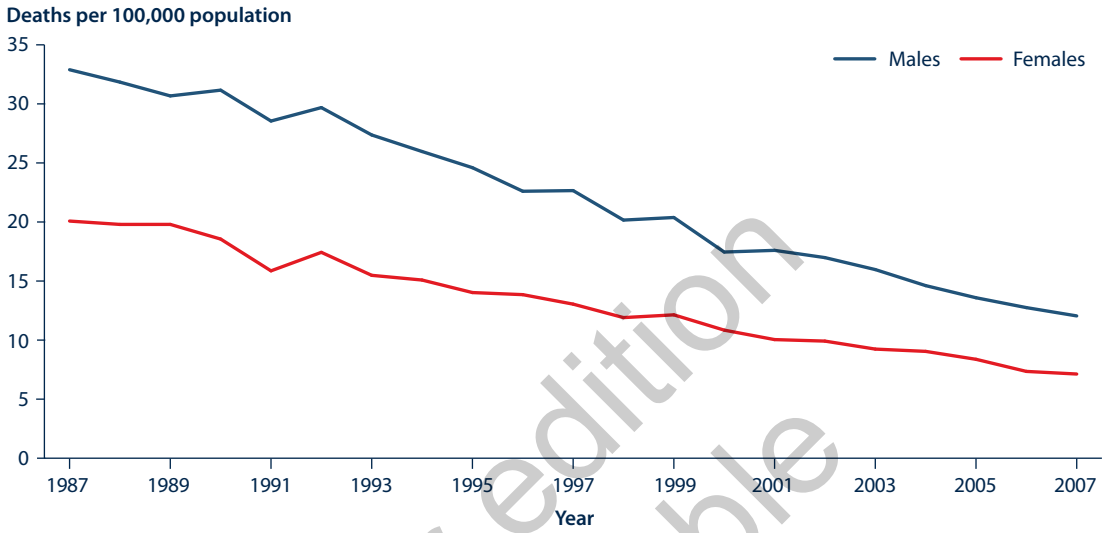


Source: AIHW National Mortality Database.

Figure 8.6: Peripheral vascular disease death rates, by age and sex, 2007

Trends

Between 1987 and 2007, the overall PVD death rate fell from 25 to 9 deaths per 100,000 population. The male and female death rates fell by 63% and 64% respectively (Figure 8.7). Male rates have remained about 1.7 times as high as female rates for the past 2 decades.



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Figure 8.7: Peripheral vascular disease death rates, by sex, 1987–2007

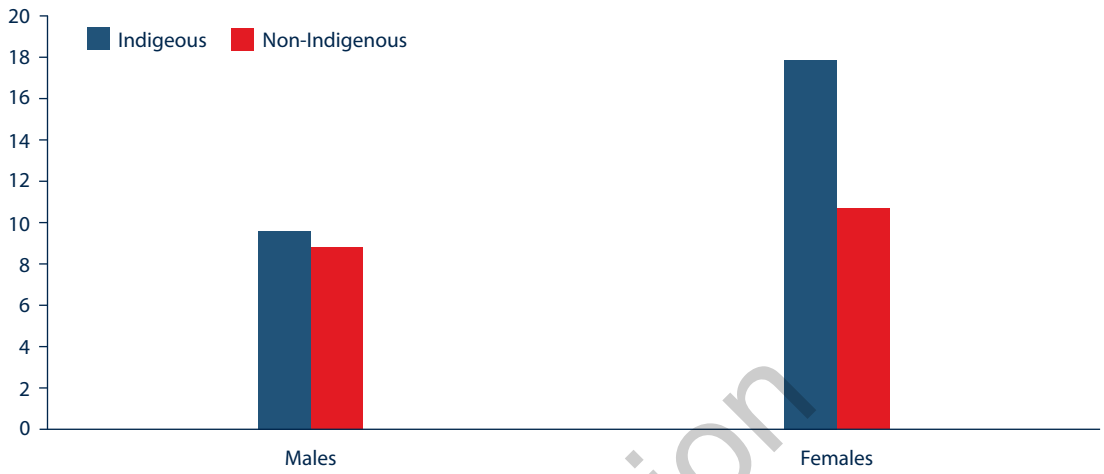
Later edition available

Health inequalities

The following section looks at PVD deaths in subgroups of interest in the Australian population. Death rates are presented for Indigenous and non-Indigenous Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2005–2007, PVD was the cause of death for 43 Indigenous Australians in jurisdictions with adequate Indigenous identification. The overall age-standardised PVD death rate for non-Indigenous Australians (14 deaths per 100,000 population) was 1.4 times as high as that for Indigenous Australians (10 per 100,000). Indigenous and non-Indigenous females had similar death rates for PVD (9 per 100,000 and 11 per 100,000 respectively). However, the difference between the Indigenous and non-Indigenous populations for males was more substantial, with 18 deaths per 100,000 people for non-Indigenous males and 10 per 100,000 for Indigenous males (Figure 8.8). Indigenous identification in deaths data is considered of sufficient quality for national reporting for New South Wales, Queensland, Western Australia, South Australia and the Northern Territory only. For further information refer to 'Reporting of Indigenous data' in Appendix A.



Notes

1. Age-standardised to the 2001 Australian population.
2. Data for NSW, Qld, WA, SA and NT only.

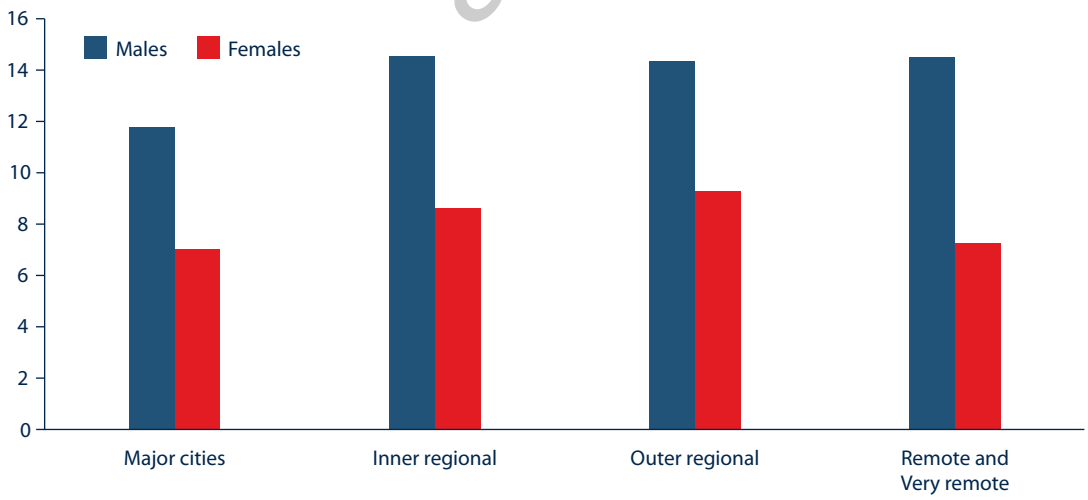
Source: AIHW National Mortality Database.

Figure 8.8: Peripheral vascular disease death rates, by sex and Indigenous status, 2005–2007

Later edition available

Remoteness

In 2005–2007, the lowest age-standardised PVD death rate was in *Major cities* (9 deaths per 100,000 population) and the highest in *Outer regional* areas (12 deaths per 100,000). Male PVD death rates were higher than female rates in all remoteness areas (Figure 8.9).



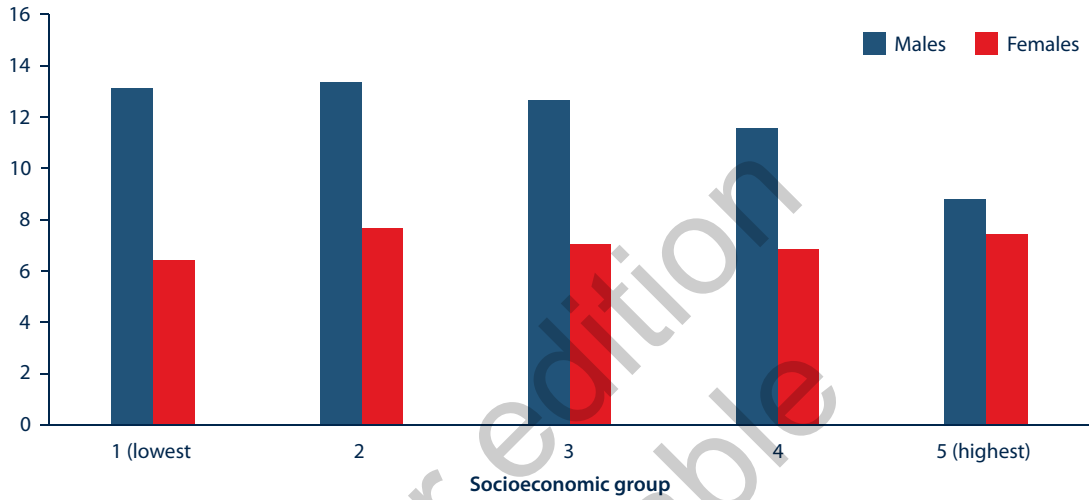
Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Figure 8.9: Peripheral vascular disease death rates, by remoteness and sex, 2005–2007

Socioeconomic group

In 2007, the overall PVD death rate was lowest in the highest socioeconomic groups. Female rates showed little variation by socioeconomic group. The male rate, which was more than twice the female rate in the lowest socioeconomic group, was similar to the female rate in the highest socioeconomic group (Figure 8.10).



Note: Age-standardised to the 2001 Australian population.

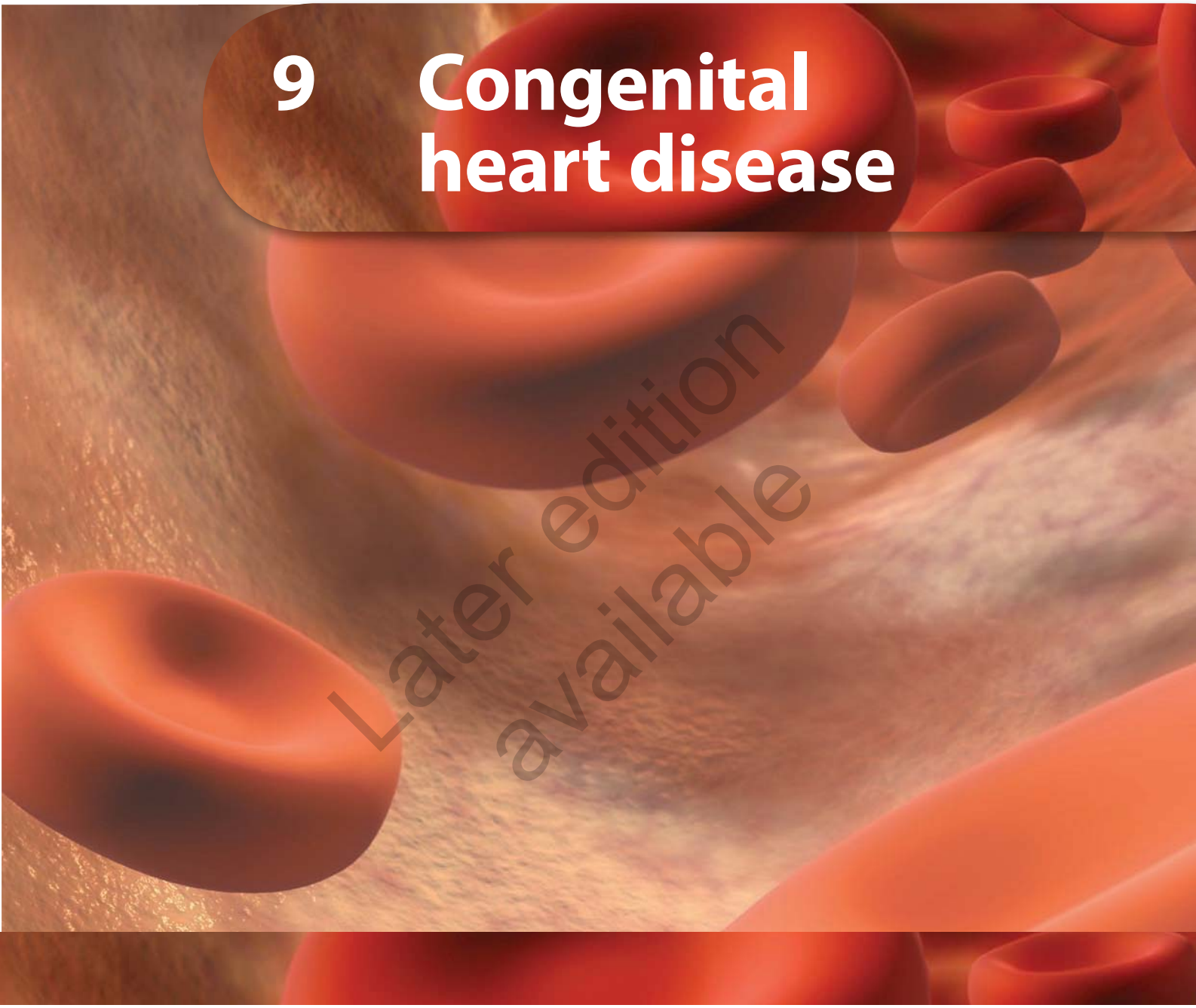
Source: AIHW National Mortality Database.

Figure 8.10: Peripheral vascular disease death rates, by socioeconomic group and sex, 2007

Later edition available

The burden of peripheral vascular disease

PVD was responsible for 0.7% of the total burden of disease and injury in Australia in 2003. Most of the PVD burden (69%) was due to years of healthy life lost due to poor health or disability. The remainder (31%) was due to years of life lost to premature death (Begg et al. 2007).



9 Congenital heart disease

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9 Congenital heart disease

What is congenital heart disease?

Congenital heart disease is any disorder of the heart or central blood vessels that is present at birth. It can take many forms and includes abnormalities of the heart or heart valves, such as holes between the pumping chambers of the heart, or valves that do not open or close properly; defects, such as the narrowing of major blood vessels, like the aorta and pulmonary artery; or combinations of disorders. Symptoms may appear at birth, or sometime thereafter, and can include breathlessness or a failure to attain normal growth and development. Most children with congenital heart disease are treated with surgery or catheter-based techniques, usually in infancy or early childhood (Hurst et al. 2001).

Congenital heart disease constitutes one of the leading causes of death in the first year of life (AIHW 2010a).

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Risk factors for congenital heart disease

In most cases the cause of congenital heart disease is unknown. Some known risk factors are an individual's genetic disposition to the disease; viral infections, such as rubella (German measles); maternal use of illicit drugs, alcohol abuse or over-the-counter and prescription medications during pregnancy; and maternal health factors, such as poorly controlled diabetes or poor nutrition (Hoffman 2006; Kalter & Warkany 1983a, 1983b).

How many Australians have congenital heart disease?

Among babies born in 2003, ventricular septal defect was the most commonly reported congenital heart condition, with 630 cases recorded, followed by patent ductus arteriosus (406), atrial septal defect (402) and pulmonary stenosis (134) (Table 9.1). These data are drawn from the AIHW Australian Congenital Anomalies Monitoring System which obtains information about babies with one or more congenital anomalies, for live births and fetal deaths (of at least 20 weeks gestation or at least 400 grams birthweight) from all states and territories except the Northern Territory (from which congenital anomaly data are not yet available). It should be noted that data collection methods vary between jurisdictions, in particular the upper age for congenital anomaly notifications which ranges from 15 years in Victoria to discharge from hospital after birth in Queensland, Tasmania and the Australian Capital Territory. Thus there will be additional babies born in 2003 with congenital heart disease that are not reported here. For further information on the methods employed by jurisdictions, refer *to Congenital anomalies in Australia 2002–2003* (Abeywardana & Sullivan 2008).

Table 9.1: Number and rate of congenital heart conditions among births[a], Australia[b], 2003

Condition	New cases in 2003	Rate[c]
Ventricular septal defect	630	24.9
Patent ductal arteriosus	406	16.0
Artrial septal defect	402	15.9
Pulmonary stenosis	134	5.3
Transposition of the great vessels	103	4.1
Coarctation of the aorta	92	3.6
Tetralogy of Fallot	82	3.2
Aortic stenosis	34	1.3
Hypoplastic left heart syndrome	37	1.5

Notes

(a) Includes live births and still births with at least 20 weeks of gestational age or at least 400 grams birth weight.

(b) For all states and territories except the Northern Territory.

(c) For all births, the rate is per 10,000 live births and fetal deaths.

Source: Australian Congenital Anomalies Monitoring System.

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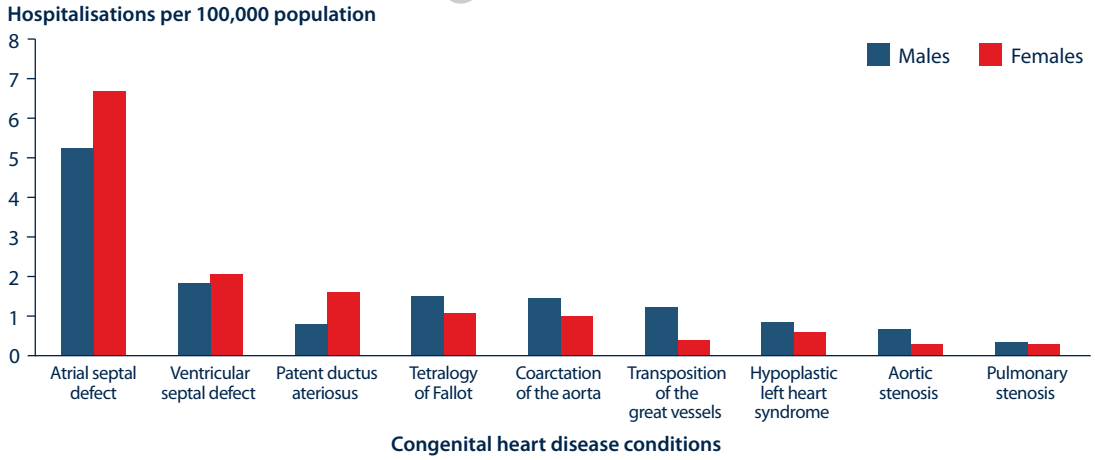
Hospitalisations

In 2007–08, there were 5,802 hospitalisations in Australia with congenital heart disease as a principal diagnosis. The highest rate of hospitalisation was for atrial septal defects (6 hospitalisations per 100,000 persons), followed by ventricular septal defects (2 per 100,000), Tetralogy of Fallot (1.3 per 100,000) and patent ductus arteriosus (1.2 per 100,000). The lowest hospitalisation rate was for pulmonary stenosis, with 0.3 hospitalisations per 100,000 people (Figure 9.1). Note that hospitalisations data in this report are based on 'episodes of care' rather than the number of people hospitalised with a condition (Appendix A).



Note: Age-standardised to the 2001 Australian population.

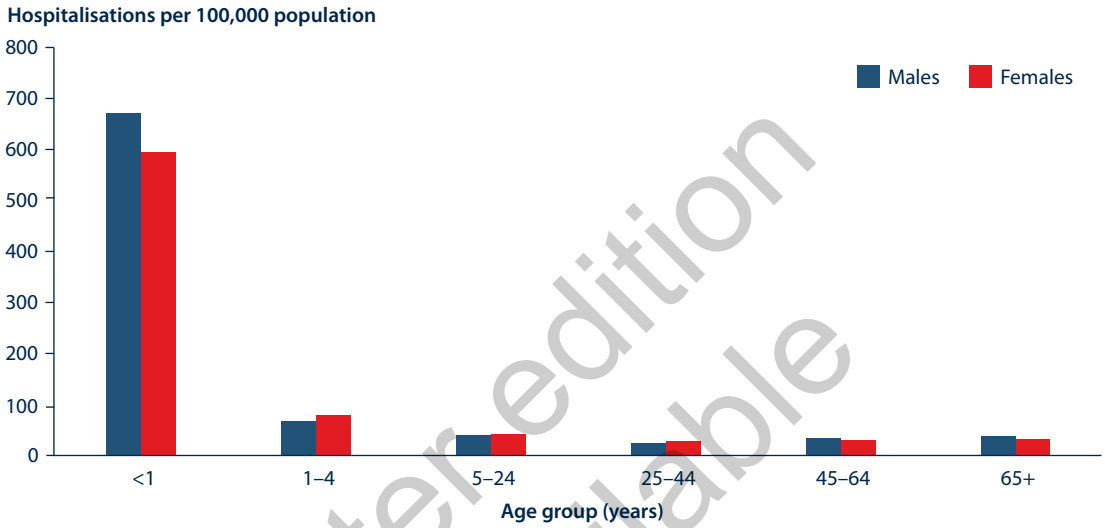
Source: AIHW National Hospital Morbidity Database.

Figure 9.1: Congenital heart disease hospitalisation rates, principal diagnosis, by congenital condition and sex, 2007–08

Sex and age

In 2007–08, the age-standardised hospitalisation rate for congenital heart disease was almost the same for both males and females (around 28 hospitalisations per 100,000 population).

Unlike other cardiovascular conditions, the number and rate of hospitalisations declined with age. Over half of congenital heart disease hospitalisations were for infants aged less than one year (56%), and just over half of these (54%) were for males (Figure 9.2).



Source: AIHW National Hospital Morbidity Database.

Figure 9.2: Congenital heart disease hospitalisation rates, by age and sex, 2007–08

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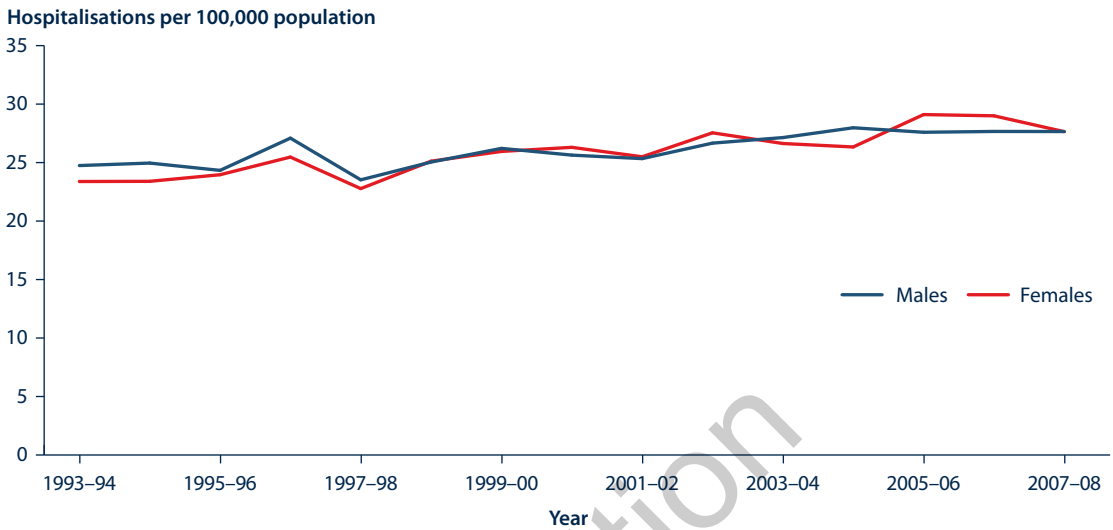
Trends

Between 1993–94 and 2007–08, the age-standardised hospitalisation rate for congenital heart disease increased by 13% from 24 to 28 per 100,000 population. Both male and female age-standardised hospitalisation rates showed similar upwards trends (Figure 9.3), which may reflect the earlier and more accurate detection of the disease, as a result of improvements in clinical procedures, such as foetal cardiac diagnosis (Billett et al. 2008; Bolisetty et al. 2004).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 9.3: Congenital heart disease hospitalisation rates, by sex, 1993–94 to 2007–08

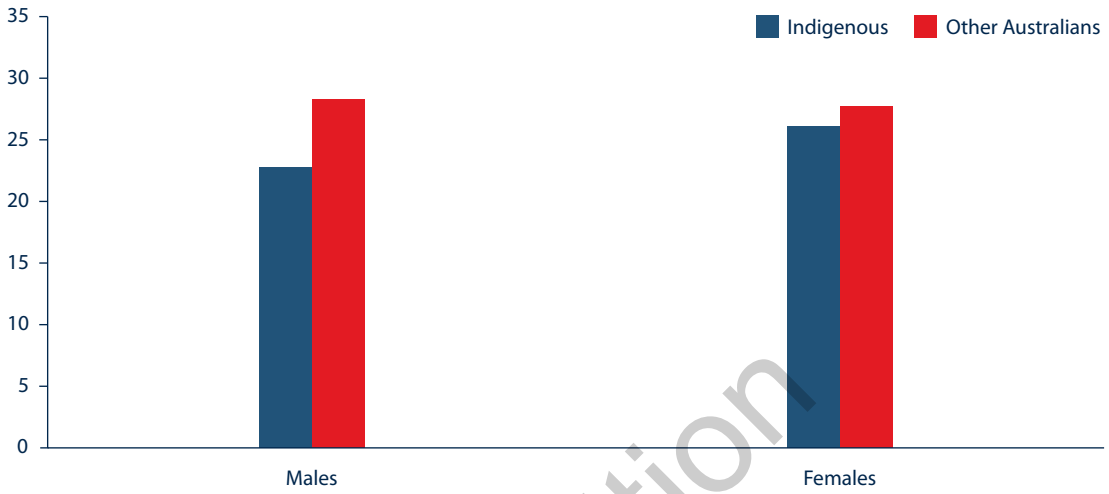
Later edition available

Health inequalities

The following section looks at congenital heart disease hospitalisations in subgroups of interest in the Australian population. Hospitalisation rates are presented for Indigenous and other Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2007–08, there were 199 hospitalisations with congenital heart disease where the patient was identified as Indigenous. The hospitalisation rate for Indigenous Australians (27 per 100,000 population) was a little lower than the rate for other Australians (30 per 100,000) (Figure 9.4). Indigenous identification in hospitalisation data is considered of sufficient quality for national reporting for New South Wales, Victoria, Queensland, Western Australia, South Australia and the Northern Territory (public hospitals) only. For further information refer to 'Reporting of Indigenous data' in Appendix A.



Notes

1. Age-standardised to the 2001 Australian population.
2. Data from NSW, Vic, Qld, WA, SA and NT (public hospitals) only.

Source: AIHW National Hospital Morbidity Database.

Figure 9.4: Congenital heart disease hospitalisation rates, by sex and Indigenous status, 2007–08

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Remoteness

In 2007–08, congenital heart disease hospitalisation rates showed little variation across remoteness areas.

Socioeconomic group

Similarly, in 2007–08, congenital heart disease hospitalisation rates varied little between socioeconomic groups.

Length of stay

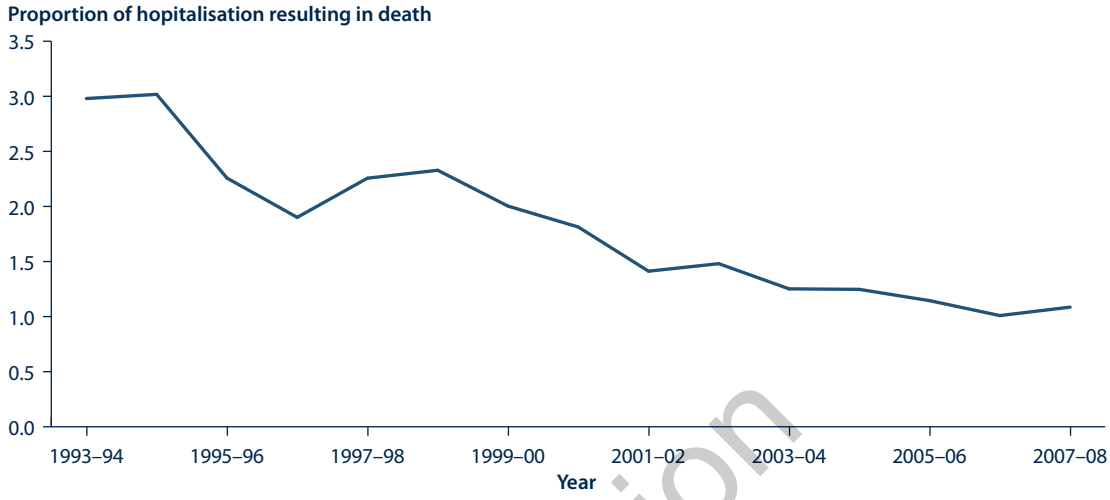
In 2007–08, just over 54% of those hospitalised with congenital heart disease were discharged the same day. In 1993–94 the figure was around 32%.

Among those hospitalised with congenital heart disease for one night or more, the average length of stay rose from 9.2 days in 1993–94 to 11.0 days in 2007–08, with a low of 8.7 days in 1999–00. Congenital heart disease was the CVD with the longest average length of hospital stay.

Deaths in hospital

Of all deaths in hospital in 2007–08, only 0.1% were attributed to congenital heart disease.

Of those patients admitted to hospital with a principal diagnosis of congenital heart disease, 1% died in hospital—down from 3% in 1993–94 (Figure 9.5).



Source: AIHW National Hospital Morbidity Database.

Figure 9.5: Congenital heart disease deaths in hospital, 1993–94 to 2007–08

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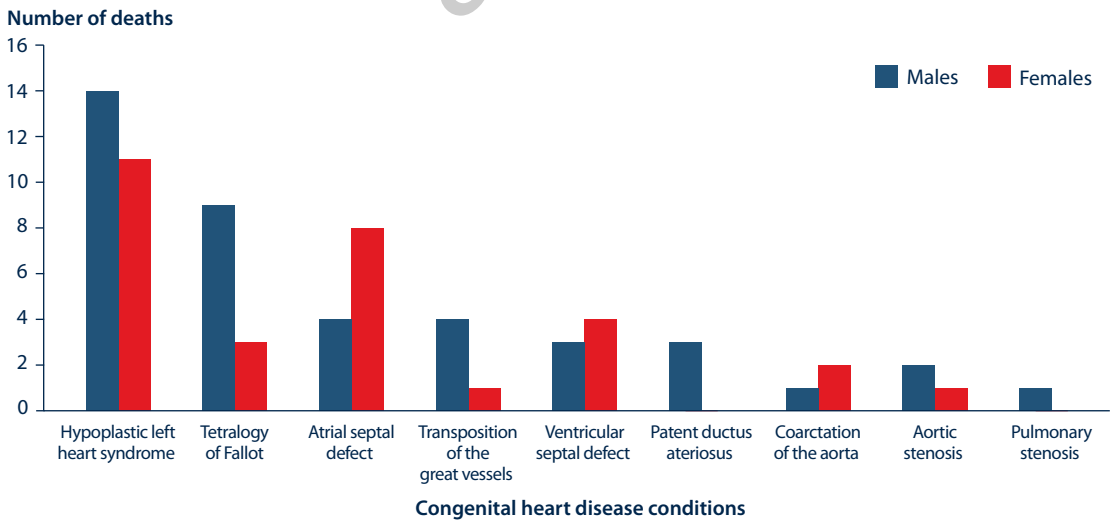
Deaths

In 2007, congenital heart disease was the main cause of 189 deaths. After perinatal conditions, it was responsible for more deaths among infants aged less than one year (26%), than any other condition.

Among congenital heart conditions, hypoplastic left heart syndrome resulted in the highest number of deaths (25), followed by Tetralogy of Fallot (12), atrial septal defects (12), ventricular septal defects (7) and transposition of the great vessels (5). Only one death resulted from pulmonary stenosis in 2007 (Figure 9.6).



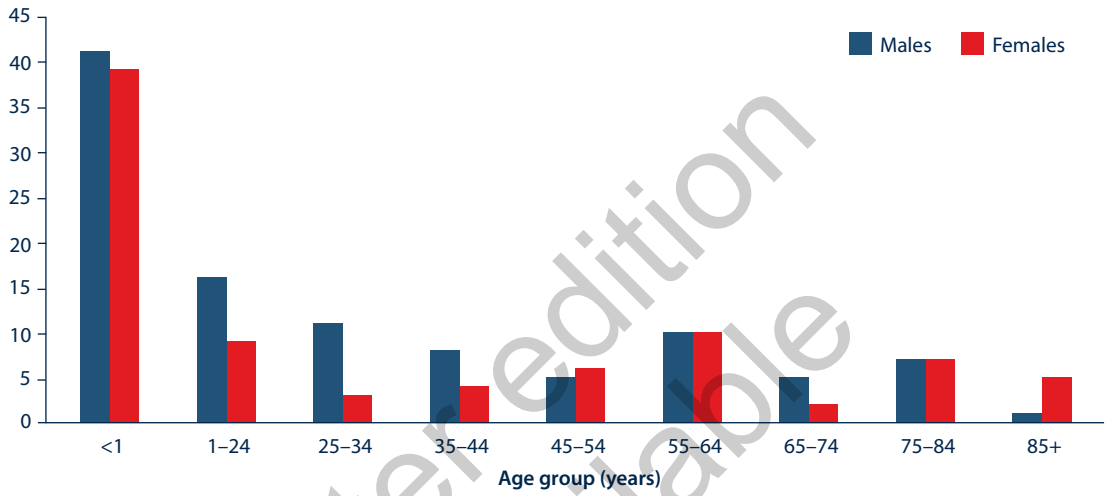
Source: AIHW National Mortality Database.

Figure 9.6: Number of deaths from congenital heart diseases, by condition and sex, 2007

Sex and age

In 2007, 104 males and 85 females died as a result of congenital heart disease. Forty-two per cent were aged one year or less (41 males and 39 females) and 13% (25) were aged 1–24 years (Figure 9.7).

Congenital heart disease affects a relatively small number of people so differences between age groups, sexes and other population subgroups, should be treated with caution.



Source: AIHW National Mortality Database.

Figure 9.7: Number of deaths from congenital heart disease, by age and sex, 2007

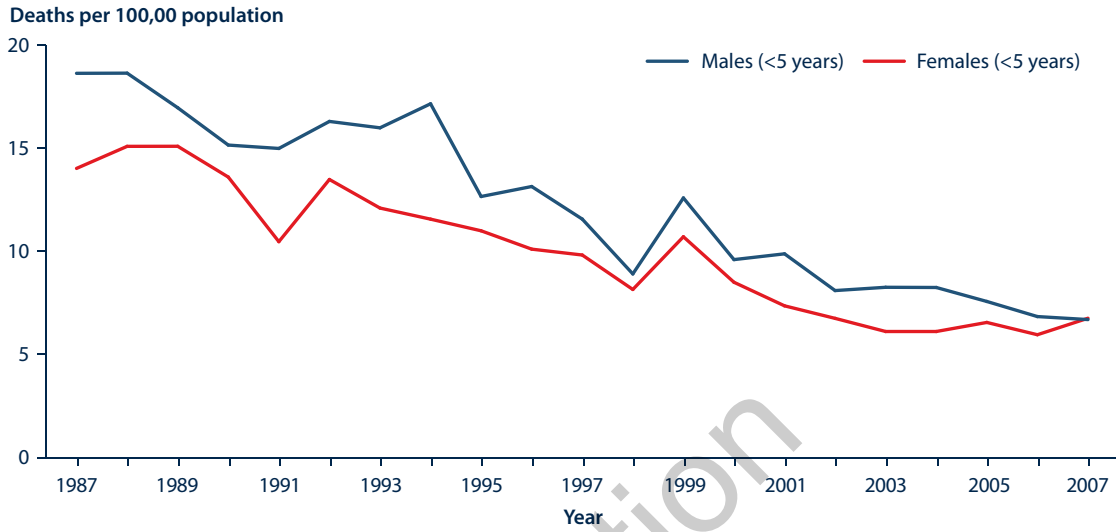
Later edition available

Trends

Over time, advances in medical care, leading to better diagnostic tests, new medications and improved surgical techniques, have increased survival rates for those with congenital heart disease (Billett et al. 2008; Bolisetty et al. 2004).

Reflecting this, between 1987 and 2007, the age-standardised death rate for males fell from 1.8 to 1.0 per 100,000 population and for females, from 1.2 to 0.8 per 100,000.

The age-specific death rate for children less than five years also fell—from 16 deaths per 100,000 population in 1987, to 7 in 2007 (Figure 9.8). The trend over this period reflects both the volatility that is introduced when relatively small numbers underlie the calculated rate and the change in disease coding that took place in 1998–99. Despite these issues, the downward trend in congenital heart disease death rates between 1987 and 2007 is clear for both males and females.



Source: AIHW National Mortality Database.

Figure 9.8: Congenital heart disease death rates for children aged less than 5 years, by sex, 1987–2007

Later edition available

Health inequalities

The following section looks at congenital heart disease deaths in subgroups of interest in the Australian population. Death rates are presented for Indigenous and non-Indigenous Australians, and by remoteness areas and socioeconomic groups.

Aboriginal and Torres Strait Islander people

In 2005–2007, congenital heart disease was the cause of death for 80 Indigenous people in the jurisdictions with adequate Indigenous identification. The age-adjusted death rate for Indigenous Australians was 3.4 times as high as that for non-Indigenous Australians (4.4 per 100,000 population compared with 1.3). It is important to note that the actual number of Indigenous deaths from congenital heart disease is small and results should be treated with caution.

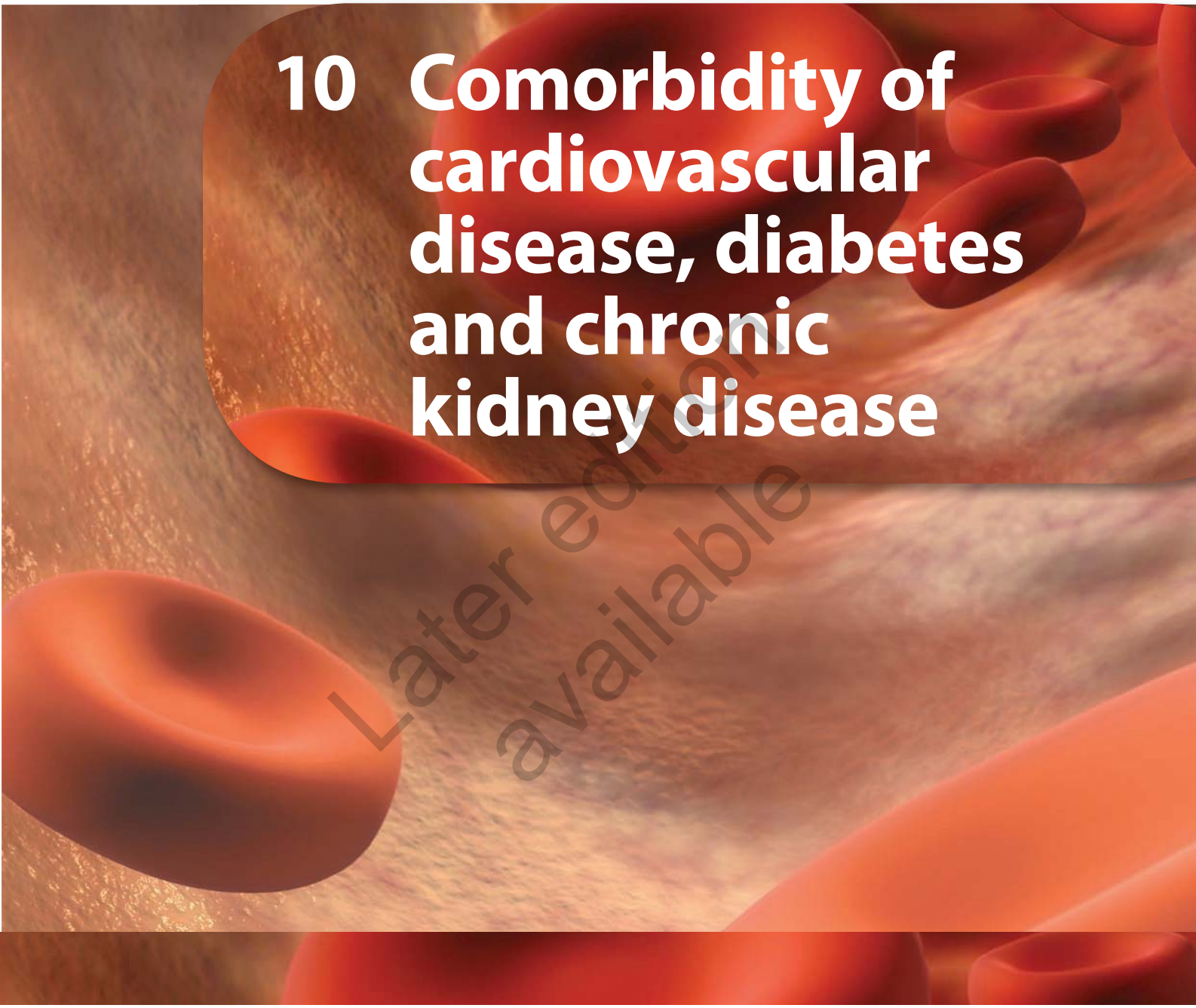
Indigenous identification in deaths data is considered of sufficient quality for national reporting for New South Wales, Queensland, Western Australia, South Australia and the Northern Territory only. For further information refer to' Reporting of Indigenous data' in Appendix A.

Remoteness

In 2005–2007, congenital heart disease death rates showed little variation between remoteness areas.

Socioeconomic group

In 2007, congenital heart disease death rates varied little between socioeconomic groups, or between sexes within socioeconomic groups.



10 Comorbidity of cardiovascular disease, diabetes and chronic kidney disease

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10 Comorbidity of cardiovascular disease, diabetes and chronic kidney disease

What is comorbidity?

Comorbidity is said to exist when a person has two or more health problems at the same time (AIHW 2010a). In this report the particular comorbidity examined is the presence of cardiovascular disease (CVD) in combination with either diabetes, chronic kidney disease (CKD) or both. CVD, diabetes and CKD are referred to as the *index conditions* and in this chapter it is predominantly data from the AIHW National Hospital Morbidity Database that have been used to assess the levels of comorbidity between CVD and the other two index conditions.

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What is diabetes?

Diabetes is a chronic condition marked by an abnormal build-up of glucose in the blood. This occurs either because the body produces little or no insulin, or because it is unable to use its insulin properly (insulin is a hormone that the body produces to help the body to use glucose for energy). There are three main types of diabetes: Type 1 diabetes; Type 2 diabetes and gestational diabetes. All three types of the disease are grouped together in this chapter as *diabetes* (AIHW 2008c).

Comorbidity of diabetes and cardiovascular disease

Among people with diabetes, CVD is a major complication and leading cause of death (Ali & Maron 2006). Diabetes greatly increases the risk of CVD, but the reasons for this are only partly understood. The general explanation is that diabetes increases the process of atherosclerosis (AIHW 2002; Ali & Maron 2006; Buse et al. 2007).

People with diabetes often also tend to have high blood pressure and elevated blood cholesterol levels, both of which are factors that increase the risk of atherosclerosis. People with diabetes have twice the risk of developing CVD, including stroke and heart attack as the general population. They have a higher rate of mortality as a result of their first cardiovascular event and poorer outcomes in the months and years following such an event (Buse et al. 2007). Among people with diabetes, CVD has an earlier onset, and is more resistant to treatment and therapies, compared to those without diabetes (Weisfeldt & Zieman 2007).

What is chronic kidney disease?

Chronic kidney disease (CKD) is described as kidney damage, and/or reduced kidney function, that lasts at least three months. Up to 90% of kidney function can be lost before symptoms appear, although most cases can be detected in the early stages using simple blood tests (AIHW 2010a).

It has been estimated that as many as one in seven Australians aged 25 years and over have some degree of CKD (Chadban et al. 2003). In severe cases, kidney function may deteriorate to the extent that it is no longer sufficient to sustain life and, if untreated, will cause death. Disease of this severity is called *end-stage kidney disease* (ESKD) and to survive those with ESKD require *kidney replacement therapy* (KRT)—either dialysis, to remove wastes or excess fluids from the body, or a kidney transplant (AIHW 2010d). In 2007–08, around 82% of adult hospitalisations with a diagnosis of CKD were attributed to regular dialysis. Because hospitalisations for regular dialysis are numerous, have unique characteristics and admissions are almost always for part of a day only, they have been excluded from the analysis in this chapter.

Comorbidity of chronic kidney disease and cardiovascular disease

CKD often presents in combination with another disease. Other diseases can cause CKD or it can be the cause of them. In particular, diabetes often causes CKD, which in turn increases the risk of cardiovascular events such as heart attacks and stroke (AIHW 2010a).

CVD, especially hypertension (high blood pressure), is one of the major causes of CKD. Untreated hypertension can damage the blood vessels in the kidneys. The blood vessel walls thicken, narrowing the internal diameter, thereby reducing blood supply and decreasing kidney function. Among people with CKD, the presence of CVD is associated with a faster decline of kidney function and the need for dialysis (Levin et al. 2001).

The reasons for excess risk of CVD among people with CKD are not clearly understood. While CKD and CVD share several risk factors, CKD has been found to independently increase the risk of hypertension and other CVDs, including heart attack, angina, coronary artery disease, stroke and heart failure (Go et al. 2004). In addition, CKD complications, such as anaemia and disturbed mineral metabolism, also contribute to an increased risk of CVD (Levin et al. 2001).

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Shared risk factors for cardiovascular disease, diabetes and chronic kidney disease

The interactions between CVD, diabetes and CKD are complex, not only because these diseases have common risk factors, but because each disease is itself a risk factor for each other disease.

A number of risk factors for CKD, such as tobacco smoking, abnormal blood cholesterol levels and high blood pressure, are also risk factors for CVD and diabetes, and these conditions are in turn also risk factors for CKD.

Similarly, the risk of developing CVD increases when diabetes is present with other risk factors, such as tobacco smoking, physical inactivity, high blood pressure, high blood cholesterol, and overweight and obesity, but each of them independently increases the risk of developing CVD whether diabetes is present or not (AIHW 2008c). When more than one of these risk factors is present in an individual, known as having *multiple risk factors*, the level of risk is further increased. For example, people who are overweight and have diabetes are at a greater risk of developing CVD than those who are just overweight or just have diabetes (Eckel et al. 2006).

Box 10.1: Data sources

National information on comorbidities between CVD, diabetes and CKD is available from four main data sources: the National Health Survey (NHS), the National Survey of Disability, Ageing and Carers (SDAC), the AIHW National Mortality Database, and the AIHW National Hospital Morbidity Database (AIHW: Tong & Stevenson 2007).

In the past, the NHS and SDAC have only included information on CVD and diabetes (and not CKD). This information can be used to estimate the number of people with both CVD and diabetes. Data from the mortality database can be used to count the number of deaths where any of CVD, diabetes and CKD was listed on the death certificate, meaning that the condition contributed to the death.

Other people may die with one of these diseases present but, if it did not contribute to their death, it will not be included on the death certificate. The hospital database includes information on both principal and additional diagnoses, recorded in relation to a particular hospitalisation.

Essentially, this is where the condition contributed to the care received in hospital. Similar to the deaths information, there may be other people hospitalised with these conditions but, if they did not contribute to the care the person received in hospital then they will not be recorded on the hospital database.

The following overview section provides summary information from the data sources mentioned above. Further details using the hospital data then follows in the subsequent section.

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Overview

In 2004–05, based on self-reported information from the NHS, nearly 3% of adults not living in institutions such as nursing homes and aged care facilities had a comorbidity of CVD and diabetes (AIHW: Tong & Stevenson 2007). On the other hand, estimates from the 2003 SDAC suggest that about 8% of people who lived in these institutions did have this comorbidity.

In 2007–08, nearly a third of hospitalisations with any diagnosis of CVD had a co-existing diagnosis of diabetes or CKD, and 6% had a diagnosis of all three. Additionally, in 2007, a fifth of deaths involving CVD had diabetes or CKD listed as a contributing factor, with 3% listing all three (Table 10.1).

Table 10.1: Comorbidity of cardiovascular disease with chronic kidney disease and diabetes: hospitalisations and deaths (per cent)

	Hospitalisations 2007–08	Deaths 2007
CVD only	62.5	76.7
CVD and CKD (without diabetes)	3.8	9.9
CVD and diabetes (without CKD)	27.2	10.5
CVD, CKD and diabetes	6.4	2.9
Total CVD	**100**	**100**

Sources: AIHW National Hospital Morbidity Database; AIHW National Mortality Database.

Hospitalisations

In 2007–08, around 38% of hospitalisations with CVD (excluding regular dialysis) had either diabetes, CKD, or both, recorded as co-existing diagnoses; that is, they were comorbid with CVD. Of the three index conditions, CVD had the lowest level of comorbidity with either one or two of the others, followed by diabetes (67% of cases) and CKD (73% of cases). Note that hospitalisations data in this report are based on 'episodes of care' rather than the number of people hospitalised with a condition (Appendix A).

Hospitalisations with cardiovascular disease and diabetes

In 2007–08, there were 321,091 hospitalisations with both CVD and diabetes but not CKD (5% of all hospitalisations). The age-standardised hospitalisation rate for the comorbidity of CVD and diabetes was 1,412 persons per 100,000 population.

The hospitalisation rate increased with age until the 75–85 years age group (11,059 males and 8,658 females per 100,000 population), with almost 70% of those hospitalised with a comorbidity for CVD and diabetes aged 65 years and over. After 85 years of age, the rate began to decline.

The overall male rate (1,629 per 100,000 population) was 1.3 times as high as the female rate (1,220). The greatest difference between males and females occurred between the ages 55 years and 74 years, where the male rate was almost 1.5 times as high as the female rate (Figure 10.1).

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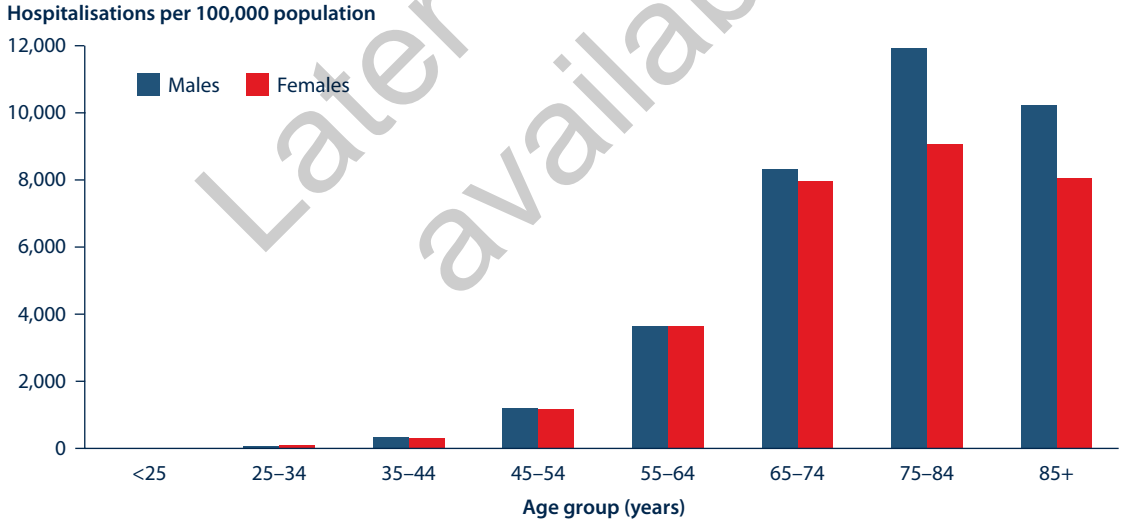


Source: AIHW National Hospital Morbidity Database.

Figure 10.1: Cardiovascular disease and diabetes hospitalisation rates, by age and sex, 2007–08

In 2007–08, the CVD conditions most commonly recorded as a co-existing diagnosis with diabetes were hypertension (63%), CHD (13%), and heart failure or cardiomyopathy (8%).

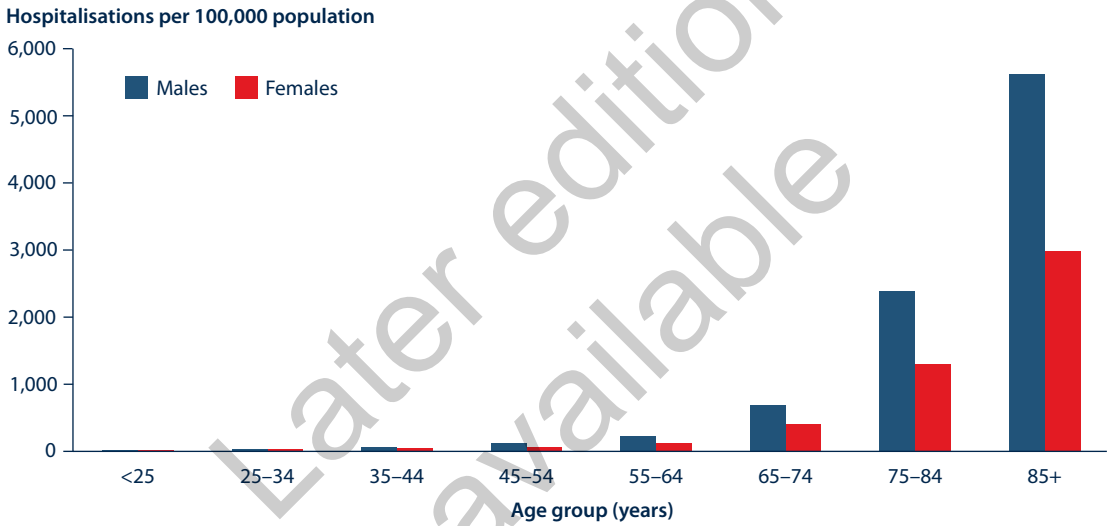
About 18.0% of hospitalisations with other forms of CVD also had diabetes recorded as a comorbid condition.

Hospitalisations with cardiovascular disease and chronic kidney disease

In 2007–08, there were 48,826 hospitalisations with both CVD and CKD (but not diabetes) (0.8% of all hospitalisations). The age-standardised hospitalisation rate for the comorbidity of CVD and CKD was 212 per 100,000 population.

Hospitalisations for the comorbidity of CVD and CKD increased sharply with age, reaching its highest rate in the 85 and over age group (5,646 males and 2,983 females per 100,000). Overall, this rate was over twice as high as that for the 75–84 year age group (1,787 per 100,000). Almost 80% of hospitalisations for CVD, with a co-existing diagnosis of CKD, occurred among those aged 65 years and over (Figure 10.2).

Overall, the age-standardised male hospitalisation rate for the comorbidity was almost twice as high as female rate (283 males and 159 females per 100,000 population).



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Source: AIHW National Hospital Morbidity Database.

Figure 10.2: Cardiovascular disease and chronic kidney disease hospitalisation rates, by age and sex, 2007–08

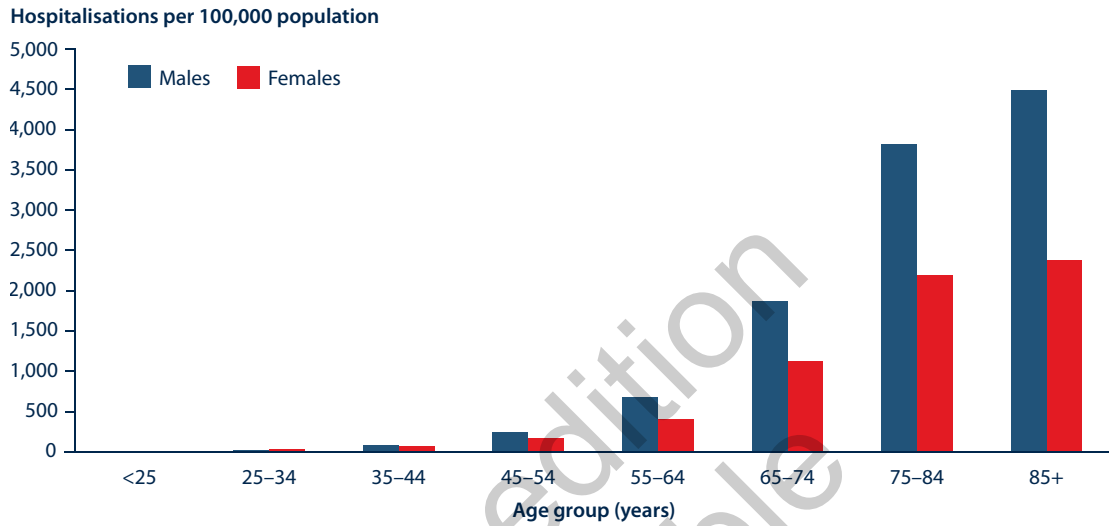
The cardiovascular conditions that occurred most commonly with CKD were hypertension (49%), CHD (17%), PVD (9%) and heart failure or cardiomyopathy (9%). About 11% of hospitalisations with other forms of CVD also had CKD recorded as a comorbid condition.

Hospitalisations with cardiovascular disease, diabetes and chronic kidney disease

In 2007–08, there were 83,168 hospitalisations with CVD, diabetes and CKD present (1.3% of all hospitalisations). The age-standardised hospitalisation rate with all three diseases present was 366 per 100,000 population.

The hospitalisation rate increased with age, with almost 75% of those hospitalised with a comorbidity of CVD, diabetes and CKD aged 65 years and over. The 85 years and over age group experienced the highest hospitalisation rates at 4,498 per 100,000 population for males and 2,378 for females (Figure 10.3).

Overall, the age-standardised male hospitalisation rate was over one and a half times as high as the female rate (469 males compared with 282 females, per 100,000 population), with the greatest difference between the sexes occurring among those aged 75 years and over, where the male rate was almost twice as high as the female rate.



Source: AIHW National Hospital Morbidity Database.

Figure 10.3: Cardiovascular disease, diabetes and chronic kidney disease comorbidity, hospitalisation rates, by age and sex, 2007–08

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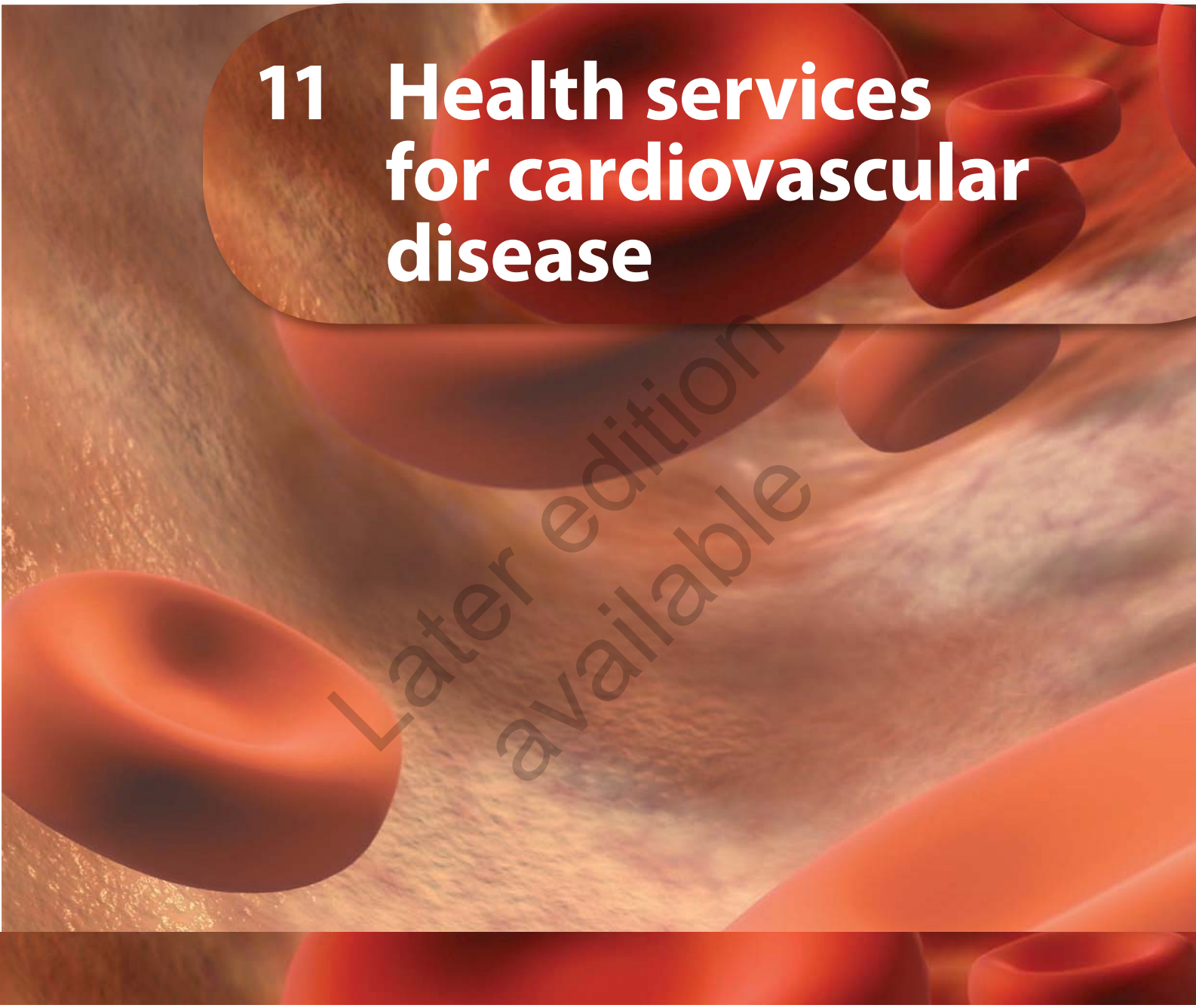
Comorbidity in the Indigenous population

In 2007–08, Indigenous Australians were 12 times more likely than Other Australians to be hospitalised with a comorbidity of all three index conditions: CVD, diabetes and CKD. Similarly, in 2003–2007 Indigenous Australians died with all three conditions present at 13 times the rate of non-Indigenous Australians, and in both periods had much higher comorbidity rates of two of these diseases (AIHW 2010a). For further information on the reporting of Indigenous hospitalisation and deaths data, refer to 'Reporting of Indigenous data' in Appendix A.



11 Health services for cardiovascular disease

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11 Health services for cardiovascular disease

Managing cardiovascular disease is a complex process because patients with CVD need to access the health system at a number of different points, and for a range of different reasons. Broadly, however, the management of the disease can be regarded as having three phases:

- prevention
- acute care
- secondary prevention.

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Prevention

Health care services to prevent CVD can be aimed at both individuals and the general population. The main focus of this report is on individual-level prevention where services in Australia are most commonly delivered by general practitioners (GPs), as well as nurses, pharmacists and Indigenous health workers. For comprehensive information about population-level initiatives and interventions, refer to the 2009 AIHW publication *Prevention of cardiovascular disease, diabetes and chronic kidney disease: Targeting risk factors*.

The prevention activities discussed in this chapter are those aimed at identifying and helping people at risk of CVD before symptoms appear or a cardiovascular event occurs.

The use of cardiovascular medicines and the adoption of lifestyle measures, such as regular exercise and a healthy diet, can reduce important CVD risk factors such as high blood cholesterol and high blood pressure.

Acute care

Acute care is the treatment given during and immediately after an acute event, one that comes on sharply and is often brief, intense and severe, such as a heart attack or stroke. It includes both the emergency care that paramedics provide before a patient reaches hospital and care given in the hospital, including that given in the emergency department and specialised care units. Care at this level is aimed at both improving survival and reducing the damaging effects of the event. Most acute care takes place in hospitals. This is not unexpected because, as reported in Chapter 3, hospitalisations with CVD are common—in 2007–08, there were 475,122 hospitalisations with a principal diagnosis of CVD.

Many patients who are hospitalised with acute CVD events will be cared for in a specialist unit. Coronary care units are well-established in Australia, having been first introduced in the 1960s. There is strong evidence to support their effectiveness in the care of patients with a coronary condition (McElduff et al. 2000). In 2008–09, there were 109 coronary care units in Australian hospitals and a further 32 cardiac surgery units, facilities dedicated to the operative care of patients with cardiac diseases (AIHW 2010c). Specialised stroke units have also proved to be effective, resulting in approximately a 20% reduction in death and disability when compared to general medical care (Stroke Unit Trialists' Collaboration 2007).

Secondary prevention

In this report, secondary prevention refers to health care designed to prevent a recurrence of cardiovascular events or complications of CVD in patients with diagnosed CVD. Recent research shows that, in Australia, more than 40% of major coronary heart disease events occur in people with established CHD (Briffa et al. 2011). Secondary prevention involves medical treatment, modification of behavioural risk factors, psychosocial care, education and support for self-management. Cardiac rehabilitation services are an example of an evidence-based secondary prevention strategy (NHFA 2010).

In this chapter

The three main phases in CVD management described above are all important, but often the data sources needed to describe them are limited. The remainder of this chapter is structured around those categories of CVD management for which data are available:

- GP services
- cardiovascular medicines
- hospital procedures
- rehabilitation.

Most of these categories will affect more than one of the phases of care described earlier. For example, GP services are important in the prevention, ongoing treatment and secondary prevention of CVD.

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General practice care

GPs are often the first port of call for a patient with a health problem. They provide a range of services, from diagnosis and treatment to prevention and rehabilitation activities. GPs also play an important role in directing patients through the health system, including referring them to more specialised or appropriate care when necessary.

This section reports on GP care for cardiovascular and lipid (cholesterol-related) problems in Australia. It shows the types of problems most commonly managed, the risk factors present in patients with CVD, and the treatment provided to these patients. Lipid problems and cardiovascular problems are reported together here because this is how data are reported from the prime GP source, the *Bettering the Evaluation and Care of Health* (BEACH) survey (See Appendix A for further information).

Prevention activities that GPs undertake, such as health checks and health advice, are aimed at a number of risk factors and are therefore relevant to a number of diseases, including CVD (AIHW 2009d).

What problems do GPs manage?

Cardiovascular and lipid problems

Cardiovascular and lipid problems are some of those most commonly encountered in general practice. In 2007–08, cardiovascular and lipid problems were the third most commonly managed problem by GPs after respiratory and general/unspecified problems. In this period, 17.6 cardiovascular and lipid problems were managed per 100 GP encounters, equating to approximately 19.3 million encounters, partly subsidised through Medicare (Britt et al. 2009).

Cardiovascular and lipid problems can be divided into three main groups: cardiac problems, vascular/lipid problems, and cerebrovascular problems.

Cardiac problems

Cardiac problems refer to problems of the heart. In 2007–08, around 22% of the cardiovascular and lipid problems managed were cardiac problems. The most common of these were coronary heart disease (CHD), heart failure and atrial fibrillation/flutter.

Between 1998–99 and 2007–08, the management rate for CHD decreased from 1.5 problems per 100 encounters to 1.1 per 100. For heart failure, there was a decrease from 0.9 to 0.6 problems per 100 encounters. The management rate for atrial fibrillation/flutter increased from 0.6 to 1.0 problem per 100 encounters in the same period.

Vascular and lipid problems

Vascular problems refer to problems of the blood vessels. Lipid problems refer to problems associated with high levels of blood fats, notably blood cholesterol. Three–quarters of cardiovascular and lipid problems managed in 2007–08 were vascular problems, the most common of all being hypertension (7%). Lipid disorders accounted for 2% of all problems that GPs managed.

The management rates for hypertension and lipid disorders have increased over the past decade. In 1998–99, there were 8.3 hypertension problems managed per 100 encounters, compared to 9.9 in 2007–08. Similarly, the management rate for lipid disorders increased from 2.5 problems per 100 encounters in 1998–99 to 3.7 in 2007–08.

Cerebrovascular problems

Cerebrovascular problems refer to problems of the blood vessels which supply the brain. These are not problems that GPs commonly managed—in 2007–08, only 3% of the cardiovascular and lipid problems that GPs managed were cerebrovascular problems.

The main forms of cerebrovascular problems managed were either stroke or transient ischaemic attack (TIA) (both around 0.2 of problems per 100 GP encounters in 2007–08). The management rates for these conditions have remained steady since 1998–99.

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What risk factors do cardiovascular patients have?

Overweight and obesity

In 2006–08, almost one in three adult male (30%) and female patients (32%) attending GPs for cardiovascular/lipid encounters were obese. A further 44% of males and 35% of females at these encounters were overweight but not obese. These figures are higher than the prevalence found in the population of patients who visit GPs. (AIHW: Britt & Miller, 2009).

The proportion of overweight and obesity among patients at cardiovascular/lipid encounters has increased somewhat over time. From 2001–03 to 2006–08, the proportion of adult male patients who were overweight or obese at cardiovascular/lipid encounters increased from 70% to 73%. Among female patients, there was an increase from 63% to 67%.

Smoking

In 2006–08, 12% of adult males and 9% of adult female patients at cardiovascular/lipid encounters were daily smokers. The prevalence of daily smoking was significantly lower among these patients than among adult patients for all GP encounters, where 20% of males and 14% of females were daily smokers.

Alcohol

In 2006–08, 28% of adult males and 19% of females at cardiovascular/lipid encounters drank at risky levels. However, the prevalence of at-risk drinking was lower among cardiovascular/lipid encounters than for all GP encounters, where 32% of adult males and 23% of adult females reported risky drinking levels.

How do GPs manage cardiovascular problems?

When managing cardiovascular and lipid problems, the most common action by GPs is supplying a prescription for a medicine, followed by ordering a pathology test, referring to a specialist, and ordering imaging (Table 11.1). This pattern was similar across different types of cardiovascular disease, although for stroke and heart failure GPs ordered imaging tests at a higher rate than for other CVDs (AIHW 2010c).

Table 11.1: Actions taken to manage cardiovascular and lipid problems, April 2007–March 2008

	Number per 100 problems (95% CI)	
Action	**Males**	**Females**
Medicines	81.9 (78.4–85.5)	77.2 (73.9–80.5)
Referral		
– Specialist	3.5 (3.0–4.0)	3.5 (3.1–3.9)
– Allied health services	0.3 (0.2–0.5)	0.5 (0.3–0.6)
Pathology ordered	41.7 (38.3–45.1)	36.1 (33.2–39.0)
Imaging ordered	2.3 (2.0–2.7)	2.6 (2.2–3.0)

Source: AIHW analysis of BEACH data.

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How is management changing?

Cardiac problems

Between 1998–99 and 2007–08, the overall rate of prescription of medicines by GPs to manage cardiac problems decreased significantly. However, the prescription of certain medicines, in particular statins and some blood-thinning medicines, increased over the same period. The increase in the use of blood-thinning medicines was associated with an increased use of the *International Normalised Ratio* (INR) test. The INR tests blood clotting in patients who have been prescribed *warfarin*, a key blood-thinning medicine. The INR test accounted for 45% of procedural treatments for all cardiac conditions in 2007–08, the increase in its use occurring with the increased use of warfarin.

Referral to a cardiologist was common in 1998–99 and has increased over time. Referral to allied health professionals was not common and has decreased over time (Table 11.2).

Table 11.2: Management of cardiac problems, 1998–99 and 2007–08

Medication type	Effect	Common name, or example	1998–99 (*n* = 3,785) Number per 100 cardiac problems (95% CI)	2007–08 (*n* = 3,658) Number per 100 cardiac problems (95% CI)
Prescribed medications (total)	—	—	106.1 (99.9–112.3)	84.2 (79.0–89.4)
Vitamin K antagonist	Anticoagulant	Warfarin	7.6 (6.6–8.6)	16.6 (14.1–19.1)
Sulfonmaides, plain	Diuretic (blood-pressure-lowering)	Frusemide	15.3 (13.7–16.8)	9.8 (8.5–11.1)
Organic nitrates	Vasodilation	Glyceryl trinitrate	18.3 (16.5–20.1)	6.3 (5.4–7.3)
HMG CoA reductase inhibitor	Lipid-lowering	Statin	3.4 (2.6–4.2)	6.5 (5.4–7.5)
ACE inhibitors (plain)	Blood-pressure-lowering	—	10.7 (9.3–12.0)	6.3 (5.4–7.3)
Other procedures	—	—	5.3 (4.4–6.3)	12.1 (10.0–14.2)
INR test	—	—	[a]	5.5 (3.8–7.2)
Referrals—2000–01[b]				
Specialist—cardiologist	—	—	6.6 (5.6–7.6)	8.7 (7.6–9.8)
Allied health services	—	—	1.0 (0.6–1.4)	0.3 (0.1–0.5)

(a) Insufficient numbers to calculate estimate.

(b) 2000–01 data used for comparison due to coding change since 1998–99.

Note: Only the 5 most commonly prescribed medicines in 2007–08 are displayed here.

Source: Henderson & Pan 2009.

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Vascular and lipid problems

GPs commonly prescribed pathology tests and medicines to manage vascular and lipid problems in 2007–08. The ordering of pathology tests increased from being ordered in 11% of GP-managed vascular/lipid problems in 1998–99 to 15% in 2007–08.

While the overall rate at which medicines were prescribed for vascular and lipid did not change greatly between 1998–99 and 2007–08, the rates at which some blood-pressure-lowering medications were prescribed showed considerable variation (Table 11.3).

Table 11.3: Management of vascular and lipid problems, 1998–99 and 2007–08

Medication type	Effect	Common name, or example	1998–99 (*n* = 12,470) Number per 100 vascular and lipid problems (95% CI)	2007–08 (*n* = 14,365) Number per 100 vascular and lipid problems (95% CI)
Prescribed medications (total)	—	—	81.5 (78.5–84.6)	78.3 (75.1–81.4)
HMG CoA reductase inhibitor	Lipid lowering	Statin	10.8 (10.0–11.6)	14.4 (13.4–15.4)
ACE inhibitors	Blood-pressure-lowering (acting on angiotensin system)	—	20.4 (19.3–21.5)	12.5 (11.7–13.3)
Angiotensin II antagonists (plain)	Blood-pressure-lowering (acting on angiotensin system)	—	4.1 (3.6–4.6)	11.9 (11.0–12.8)
Dihydropyridine derivatives	Blood-pressure-lowering (acting on calcium channels)	—	11.3 (10.3–11.9)	8.5 (7.8–9.1)
Angiotensin II antagonists and diuretics	Blood-pressure-lowering (combination of agents acting on angiotensin system and diuretic)	—	N/A	7.0 (6.4–7.7)
Other treatments	—	—	20.9 (19.2–22.5)	22.7 (20.9–24.6)
Clinical	—	—	19.6 (18.0–21.2)	20.1 (18.3–21.9)
Advice/ education	—	—	1.5 (1.2–1.8)	0.3 (0.2–0.4)
Counselling	—	—	1.5 (1.2–1.8)	2.4 (1.9–2.8)
Procedures	—	—	1.3 (1.1–1.5)	2.6 (2.2–3.1)

Note: Only the five most commonly prescribed medicines in 2007–08 are presented.

Source: Henderson & Pan 2009.

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Cerebrovascular problems

The prescription of medicines to treat cerebrovascular problems increased significantly between 1998–99 and 2007–08. In particular, there were large increases in the prescription of blood-thinning medicines (including aspirin), statins, and some antipsychotic medicines. Rates of referrals to specialists and allied health professionals did not change significantly. As with the management of cardiac problems, the use of the INR test increased significantly and accounted for 35% of all procedural treatments for cerebrovascular disease in 2007–08 (Table 11.4).

Table 11.4: Management of cerebrovascular problems, 1998–99 and 2007–08

Medication type	Effect	Common name, or example	1998–99 (*n* = 12,470) Number per 100 cerebrovascular problems (95% CI)	2007–08 (*n* = 14,365) Number per 100 cerebrovascular problems (95% CI)
Prescribed medications (total)	—	—	46.6 (39.6–53.6)	63.3 (54.5–72.0)
Platelet aggregation inhibitors, excluding heparin	Blood-thinning	Aspirin	0.9 (0.0–1.9)	22.7 (18.2–27.3)
HMG CoA reductase inhibitor	Lipid-lowering	Statin	1.1 (0.1–2.1)	5.1(2.7–7.5)
Phenothiazines with piperazine structure	Antipsychotic	—	0.3 (*)	1.3 (0.3–2.4)
Other treatments	—	—	20.3 (15.7–24.8)	20.4 (16.3–24.5)
Clinical	—	—	17.5 (13.3–21.7)	16.4 (12.6–20.1)
Procedures	—	—	2.7 (0.7–4.8)	4.0 (2.1–6.0)
INR test	—	—	*	1.4 (0.1–2.6)

* Insufficient numbers to calculate estimate.

Source: Henderson & Pan 2009.

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Medicines for cardiovascular disease

Cardiovascular medicines are key elements in preventing and treating CVD and its risk factors. With appropriate medication patients with CVD, or those at risk of the disease, can improve their quality of life and increase their life expectancy.

In this section, CVD medicines have been grouped using the *Anatomic Therapeutic Chemical* (ATC) classification of medicines. The ATC classification groups medicines according to the body organ or system on which they act, their therapeutic characteristics, and their chemical characteristics.

The ATC classification is the Australian standard for classifying medicines and is commonly used in reporting. However, it does not always align well with clinical practice. Note also that some of the medicines reported here may be used for a range of different conditions, including some non-cardiovascular diseases.

The counts of medicines in this section include both government-subsidised and non-subsidised prescription medicines listed on the Pharmaceutical Benefits Scheme and Repatriation Pharmaceutical Benefits Scheme (PBS/RPBS). Non-subsidised medicines listed on the PBS/RPBS are those for which the cost is below the level of patient contribution, capped at $31.30 in 2008. Estimates of the supply of non-subsidised PBS/RPBS medicines are produced from the Pharmacy Guild Survey. (For further information, see Appendix A.)

Note that the estimates in this section refer to medicine supply rather than usage. The reason for this is that patients may not fill all prescriptions or use all the medicines prescribed. In addition, there are some medicines not included in the PBS/RPBS and those are not included in this section.

Medicine supply is expressed as 'defined daily dose per 1,000 population per day (DDD/1,000/day)'. This measure is based on an assumed average dose per day of a medicine used for its main indication (the main reason for which the medicine was prescribed) in adults. The DDD allows comparisons between medicines to be made independently of differences in price, preparation and quantity per prescription.

This section comprises details of the supply of CVD medicines, including antithrombotic medicines, blood-pressure-lowering medicines and lipid-lowering medicines.

Medicine supply and costs

In 2008, over 137 million prescriptions for cardiovascular diseases medicines were supplied to the community at a cost of $4.2 billion. Blood-pressure-lowering medicines were the most commonly dispensed, followed by lipid-modifying medicines and antithrombotics (Table 11.5).

Table 11.5: Supply of cardiovascular prescription medicines, 2008

Medicine	No. scripts ('000)	Cost ($million)
Antithrombotic medicines	8,168	335
Blood-pressure-lowering medicines (excludes antihypertensives and peripheral vasodilators)		
Diuretics	2,926	38
Beta-blockers	8,036	161
Calcium-channel blockers	9,739	213
Renin-angiotensin system agents	29,817	758
Total blood-pressure-lowering medicines	*50,518*	*1,170*
Lipid-modifying medicines	23,280	1,458
Other medicines		
Nitrates	2,055	41
Antiarrhythmics	593	17
Total other medicines	*2,647*	*58*
Total cardiovascular medicines	**137,779**	**4,249**

Note: Columns do not sum to totals because not all medicines supplied are included in the table.

Source: AIHW analysis of data supplied by the DoHA from the Pharmaceutical Benefits Data System.

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Antithrombotic medicines

Antithrombotic medicines act either by preventing the formation of blood clots or by dissolving existing blood clots. These medicines are usually taken over a long period and act to reduce the risk of heart attack and death among people with CHD. They also reduce the risk of subsequent strokes and disability among patients with a history of ischaemic stroke.

In 2008, the DDD per 1,000 population per day for antithrombotic medicines was 37.8, up from 34.5 in 2006.

Blood-pressure-lowering medicines

Blood pressure-lowering medicines, or antihypertensives, are used to treat high blood pressure and have been shown to significantly reduce the number of deaths from heart attacks and stroke (Cutler et al. 2007).

Diuretics

Diuretic medicines increase the rate of urination, leading to a reduction in blood volume. This is a useful technique for reducing blood pressure when treating the symptoms of heart failure. Diuretics are popular medicines because they can be equally as effective in lowering blood pressure as other medicine classes, are inexpensive and have few side-effects (AIHW 2010c).

The prescription rate for diuretics increased slightly between 2006 (44.3 DDD/1,000/day) and 2008 (45.6 DDD/1,000/day). However, since the early nineties the use of diuretics has decreased markedly, reflecting the increased use of other medicines to lower blood pressure. In 1990, diuretics were dispensed in Australia at the rate of almost 90 DDD/1,000/day, but by 2000 this rate had almost halved (AIHW 2004; AIHW: Senes & Penm 2007).

Beta-blockers

Beta-blocking agents reduce the heart's activity by suppressing certain signals to it that cause it to beat faster and harder. These medicines are useful in treating patients with high blood pressure. They can also reduce pain and deaths among people with angina, or a history of heart attacks, and can prevent further strokes and heart attacks (AIHW 2010c).

There was a small increase in the supply of beta-blockers between 2006 and 2008 (25.8 to 26.8 DDD/1,000/day). Beta-blockers were the least prescribed of the blood-pressure-lowering medicines in 2008 (AIHW 2004; AIHW: Senes & Penm 2007).

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Calcium-channel blockers

Different calcium-channel blockers act on particular calcium ion channels in different parts of the heart and circulation, having the effect of reducing the force of contraction of the heart, reducing both blood pressure and the effects of angina (AIHW 2010c).

The rate at which calcium-channel blockers were prescribed, increased from 48.4 DDD/1,000/day in 2006 to 53.8 in 2008, consistent with the rapid increase in their use, witnessed between 1990 and 2000 (AIHW 2004; AIHW: Senes & Penm 2007). Calcium-channel blockers are the second most commonly prescribed blood-pressure-lowering medicines.

Renin-angiotensin system agents

Renin-angiotensin system agents are used to reduce blood pressure by blocking the effects of the *renin-angiotensin system*, a hormone system of the body which regulates blood pressure. These medicines are commonly used to treat people with high blood pressure or heart failure. A common type is the angiotensin-converting enzyme (ACE) inhibitor, which in some cases is the first choice treatment for high blood pressure (AIHW 2010c).

Between 2006 and 2008, the supply of renin-angiotensin system agents increased from 165.6 to 193.6 DDD/1,000/day. This trend continues the sharp increase in the supply of these medicines since the early 1990s (AIHW 2004; AIHW: Senes & Penm 2007). Renin-angiotensin system agents were the most commonly prescribed cardiovascular medicine in 2008.

Lipid-modifying medicines

Lipid-modifying medicines are used to control blood cholesterol level, which is often an important risk factor for those with, or at risk of developing, CVD. Lipid-modifying medicines may act to reduce the levels of blood LDL cholesterol (low-density lipoprotein, the so-called 'bad' cholesterol) or to increase the levels of blood HDL cholesterol (high-density lipoprotein, the so-called 'good' cholesterol). They may also be used to reduce high levels of blood triglyceride, a fat that can be associated with the development of heart disease (AIHW 2010c).

Lipid-modifying medicines are commonly prescribed—in 2008 they were prescribed at the rate of 132.2 DDD/1,000/day, one of the highest prescription rates for cardiovascular medicines, reflecting the importance of cholesterol control in the management of cardiovascular health (AIHW: Senes & Penm 2007).

There was a large increase in the use of lipid-modifying medicines between 2006 and 2008 (101.0 to 132.2 DDD/1,000/day). This increase is consistent with the surge in the use of statins (a commonly used lipid-modifying medicine) between 1990 and 2000 (AIHW 2004; AIHW: Senes & Penm 2007).

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Other medicines

Nitrates

Nitrates are commonly prescribed for angina. They relieve and prevent angina symptoms by dilating heart blood vessels and reducing the work done by the heart (AIHW 2010c). The prescription rate of nitrates remained fairly steady between 2006 and 2008 (13.0 and 12.7 DDD/1,000/day) (AIHW: Senes & Penm 2007).

Antiarrhythmics

Antiarrhythmic medicines are given either to restore normal heart rhythm or prevent serious abnormal heart rhythms (arrhythmias) (AIHW 2010c). The supply of these medicines increased from 0.6 DDD/1,000/day in 2006 to 1.9 in 2008 (AIHW: Senes & Penm 2007).

Medicine supply to Indigenous Australians

Many of the medicines supplied to Indigenous Australians in remote areas are done so under the Section 100 arrangements of the *National Health Act 1953*.

Section 100 arrangements allow any person, Indigenous or otherwise, attending an approved remote area Aboriginal or Torres Strait Islander Health Service, to receive PBS/RPBS medicines without the need for a prescription form, and without cost.

In 2006, 25% of Indigenous Australians lived in *Remote* or *Very remote* areas meaning Section 100 medicines represents only the supply of medicines for a specific subset of the Indigenous population. Other Indigenous people are supplied medicines through the PBS/RPBS in the same way as the rest of the population. However, the number of patients prescribed cardiovascular medicines who identified as Indigenous in the PBS/RPBS cardiovascular population is substantially lower than would be expected from the Australian population. Whether this is the result of under-identification of Indigenous Australians or under-utilisation of the PBS/RPBS is unclear (AIHW 2010c).

In 2008, 366,424 packs of cardiovascular medicines were supplied under Section 100 arrangements.

This represents an increase of 25% since 2005. Comparatively, in 2007–08, 932,768 PBS/RPBS prescriptions were dispensed in *Remote* and *Very remote* areas. Because a Section 100 medicine pack is equivalent to a PBS/RPBS prescription in terms of the medicine supplied, this suggests that Section 100 medicines accounted for 28% of the total CVD medicine supply to *Remote and Very remote* areas. It is important to note that these data represent the supply of medicines to clinics and do not represent the number of items dispensed to patients.

Hospital procedures for cardiovascular disease

What hospital procedures are used for cardiovascular disease?

Hospital procedures for CVD in this report are those procedures performed in hospital on admitted patients to diagnose or treat CVD. Diagnostic procedures aim to identify the type, severity and location of CVD problems and the therapeutic procedures to address the problem once it is identified.

Data in this section are sourced from the AIHW National Hospital Morbidity Database and refer only to procedures performed in hospitals, although it is possible for procedures to be performed elsewhere. In most cases, the data reported in this section are rates of all procedures among the Australian population. However, for computerised tomography (CT) brain scans and Magnetic Resonance Imaging (MRI) brain scans, where a large proportion of procedures are undertaken for non-CVD related illness and disease, the analysis counted procedures only where the principal diagnosis was stroke or transient ischemic attack (TIA).

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Diagnostic procedures

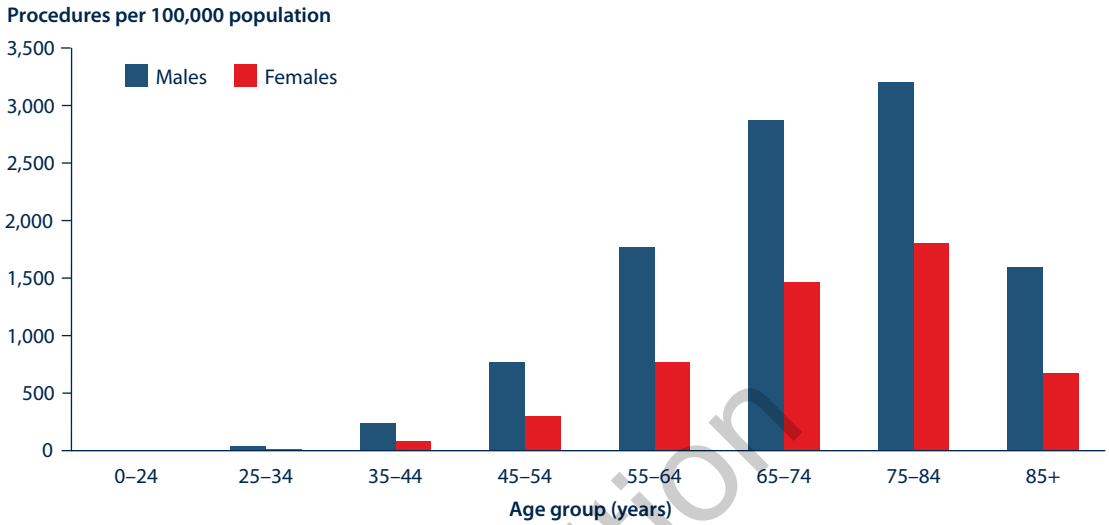
The diagnostic procedures reported in this section are coronary angiography, echocardiography, CT scans and MRI scans.

Coronary angiography

Coronary angiography gives a picture of the heart's arteries, known as the coronary arteries, to find out if and where they are narrowed or blocked. A catheter is guided to the heart where a special dye is released into the coronary arteries before X-rays are taken. The resulting X-ray images provide detailed information about the health of the heart and arteries. This is an important diagnostic test for CHD, providing medical professionals with the information they need to decide upon treatment options.

In 2007–08, a total of 110,611 coronary angiography procedures where performed in hospital. The majority of these procedures were performed on males—73,011 procedures (66%) compared with 37,600 (34%) on females.

The rate of coronary angiography procedures was also higher for males than for females—in 2007–08, the age-adjusted rate of coronary angiography procedures was 676 per 100,000 people for males, and 319 per 100,000 for females. The rate of coronary angiography procedures increased with age until 75–84 years, after which the rate decreased (Figure 11.1).



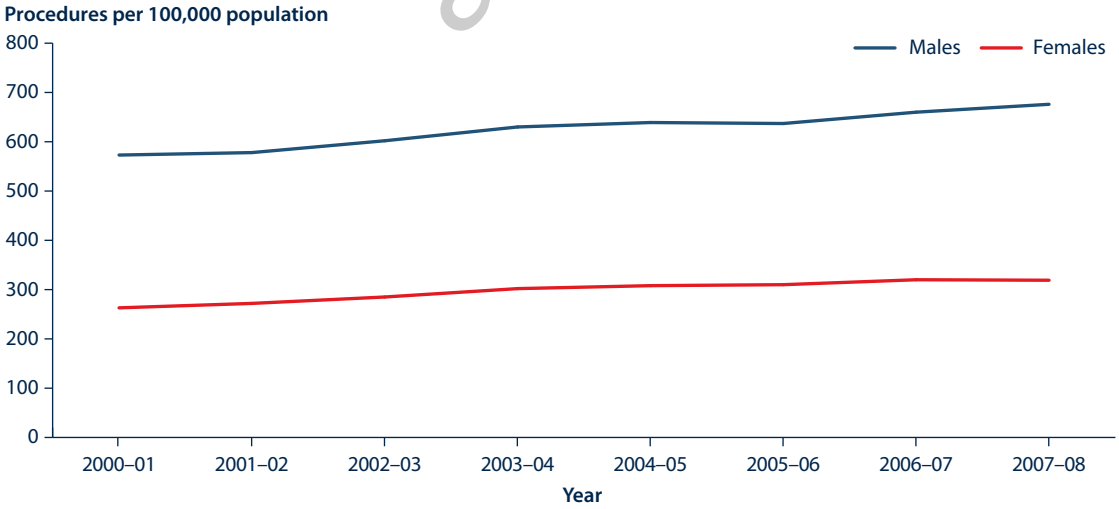
Source: AIHW National Hospital Morbidity Database.

Figure 11.1: Coronary angiography procedure rates, by age and sex, 2007–08

Later edition available

The overall number of coronary angiography procedures increased from 78,981 in 2000–01 to 110,614 in 2007–08, a 40% increase—38% for males and 42% for females.

The age-standardised rate of coronary angiography procedures increased from 412 per 100,000 population to 491 per 100,000 from 2000–01 to 2007–08. In 2000–01, there were 574 coronary angiography procedures per 100,000 people for males and 263 for females. By 2007–08, this rate had increased to 676 procedures for males and 319 for females (Figure 11.2).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 11.2: Coronary angiography procedure rates, by sex, 2000–01 to 2007–08

Echocardiography

Echocardiography is a procedure which takes moving pictures of the heart using high frequency sound waves (ultrasound). With these it is possible to measure the size of the various heart chambers, to study the appearance and motions of the heart valves, and to assess blood flow through the heart.

A total of 28,428 echocardiography procedures were performed in 2007–08. More of these procedures (18,383 or 65%) were performed on males than on females (10,045 or 35%). This difference is also reflected in the age-standardised rate of the procedures, which were higher for males (172 procedures per 100,000 people) than for females (85 procedures per 100,000 people).

Computerised Tomography (CT) brain scan

A computerised tomography (CT) scan of the brain uses cross-sectional X-rays to generate an image of the brain. This image is used to distinguish between the major stroke types—blockage or bleeding—to guide treatment. A CT brain scan can be used for a number of other conditions, such as injury to the head. To exclude such cases, only those procedures where the principal diagnosis was stroke or TIA are reported here.

In 2007–08, 34,441 CT brain scan procedures were performed on patients with a principal diagnosis of stroke or TIA. The procedures were almost evenly divided between males and females, with males receiving 17,457 procedures (51%) and females 16,984 (49%). However, the age standardised rates showed that there were 171 procedures per 100,000 population for males and 131 per 100,000 for females.

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Magnetic Resonance Imaging (MRI) brain scan

An MRI (Magnetic Resonance Imaging) brain scan uses magnets and radio waves to generate an image of the brain. These images show a higher level of detail in soft tissue than CT brain scans. As with a CT brain scan, an MRI brain scan can be used for a number of other conditions. To exclude such cases, this section only reports on those procedures where the principal diagnosis was stroke or TIA.

MRI scans of the brain were used far less often than CT scans. In 2007–08, there were 7,786 MRI brain scan procedures performed on patients with a diagnosis of stroke or TIA. More males (4,490, 58%) received the procedure than did females (3,296, 42%). This is also reflected in the age-standardised rate, which is higher for males (43 procedures per 100,000 people), than for females (27 procedures per 100,000 people).

Therapeutic procedures

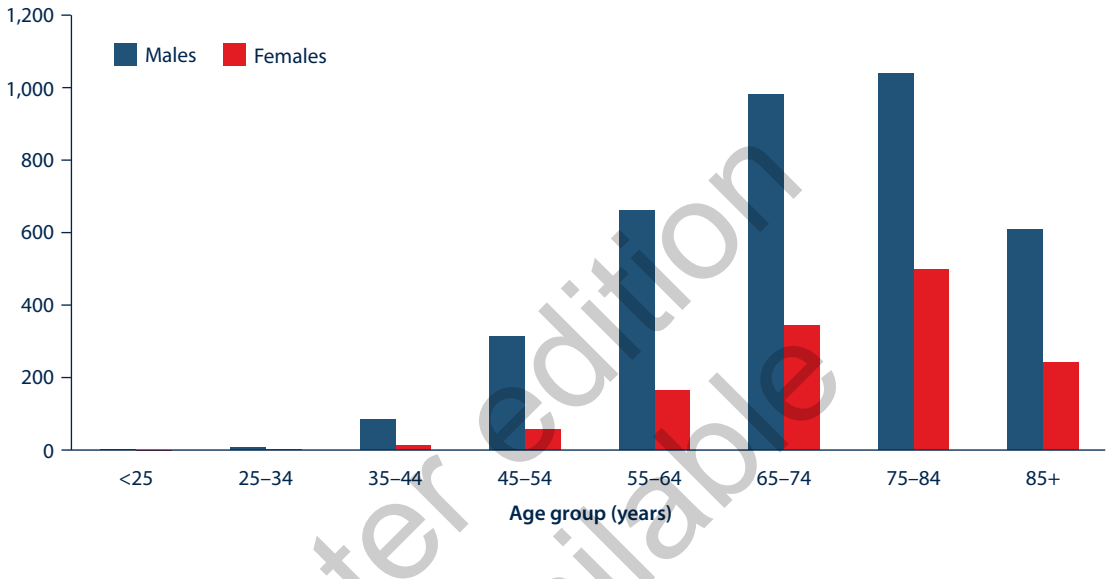
The therapeutic procedures reported in this section are percutaneous coronary interventions, coronary artery bypass grafting, heart valve repair or replacements, pacemaker insertions, cardiac defibrillator implants, heart transplants and carotid endarterectomies.

Percutaneous coronary interventions

Percutaneous coronary interventions (PCIs) are used to restore adequate blood flow to blocked coronary arteries. Two types of procedure are used: coronary angioplasty without stent, and coronary stenting. Coronary angioplasty involves inserting a catheter with a small balloon into a coronary artery which is inflated to clear the blockage. Coronary stenting is similar, but involves the insertion of stents (expandable mesh tubes) into the affected coronary arteries. Of all PCIs performed in 2007–08, nearly 94% involved stenting.

In 2007–08, a total of 34,972 PCIs were performed, three-quarters of which (26,109) were for males. The age-standardised rate for PCIs for males was much higher than that for females (241 compared with 74 per 100,000 people).

For males and females, the rate of PCIs increased steadily with age until the 75–84 years age group and then declined for those 85 years and over (Figure 11.3).



Source: AIHW National Hospital Morbidity Database.

Figure 11.3: Percutaneous coronary intervention rates, by age and sex, 2007–08

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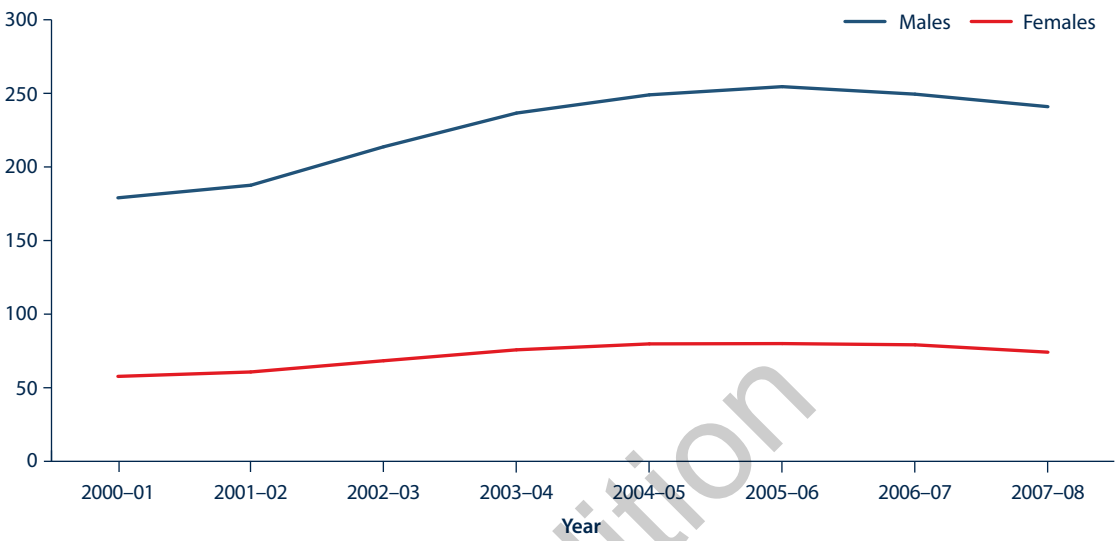
Between 2000–01 and 2007–08, the number of PCIs performed increased by 57%. This increase was greater for males than for females—58% compared with 52% respectively.

In 2000–01 the age-standardised rate was 117 per 100,000 population and in 2007–08 it was 155 per 100,000, which represented a decrease from the highest rate of 164 per 100,000 in 2005–06. The rates for both males and females followed a similar trend over time, although the procedure rates were higher for males than for females in all years (Figure 11.4).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 11.4: Percutaneous coronary intervention rates, by sex, 2000–01 to 2007–08

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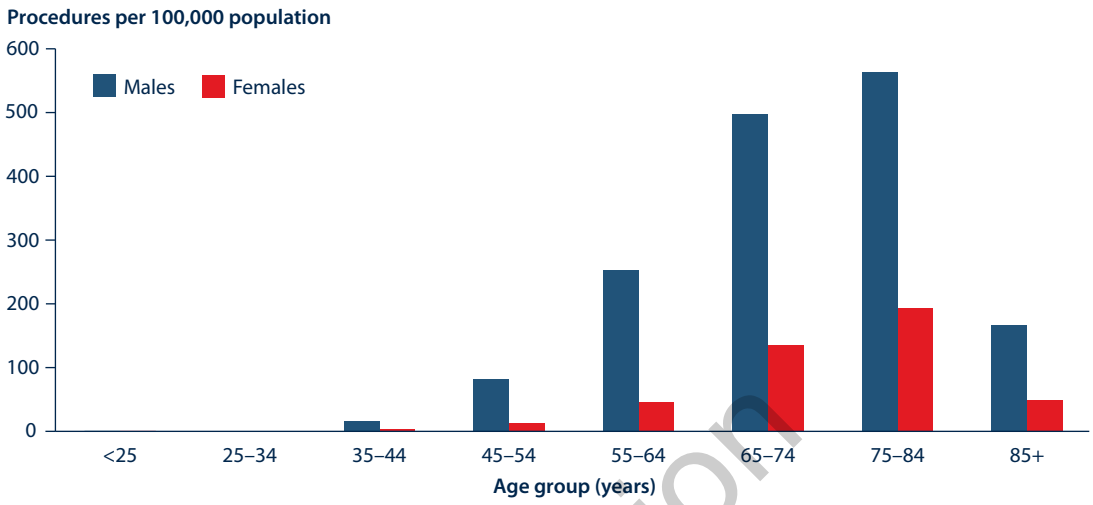
Coronary artery bypass grafting

Coronary artery bypass grafting (CABG) is a surgical procedure using blood vessel grafts to bypass blockages in the coronary arteries and restore adequate blood flow to the heart muscle. The surgery involves taking a blood vessel from a patient's inner chest, arm or leg and attaching it to vessels on the outside of the heart to bypass a blocked artery.

In 2007–08, 13,612 CABG procedures were performed. These procedures were performed much more often on males (10,648 or 78%) than females (2,964 or 22%). This difference is also reflected in the age-standardised procedure rate, which was higher for males (99 per 100,000 people) than for females (25 per 100,000 people).

The rate of CABG procedures increased until the age of 75–84 years for both males and females. Age specific procedure rates were higher for males than for females across all age groups (Figure 11.5).



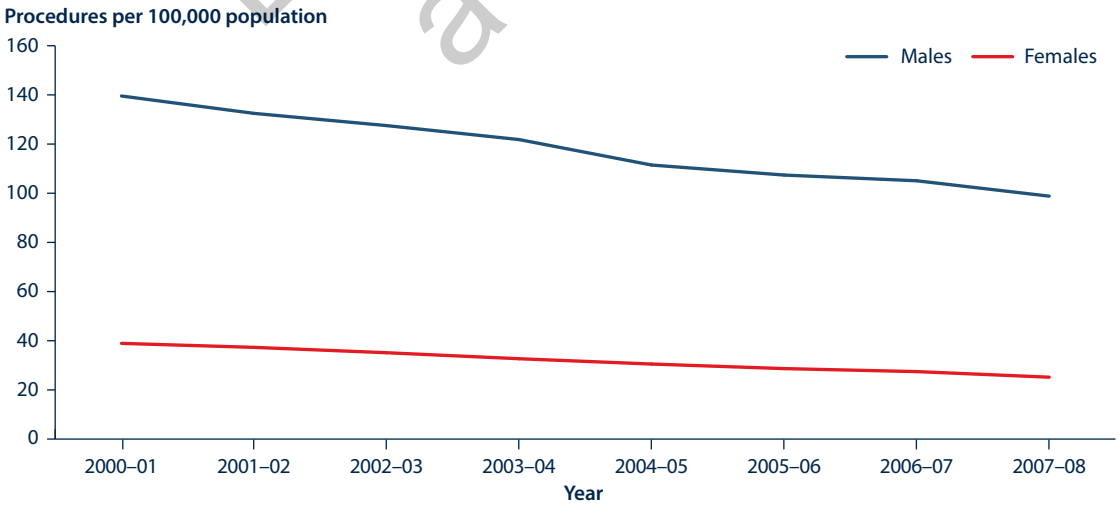
Source: AIHW National Hospital Morbidity Database.

Figure 11.5: Coronary artery bypass graft rates, by age and sex, 2007–08

Later edition available

Overall, there was a 19% reduction in the number of CABGs performed between 2000–01 and 2007–08, from 16,696 to 13,612. The fall in the number of procedures was higher for females (25%) than for males (16%).

The age-standardised rate of CABG procedures decreased steadily between 2000–01 and 2007–08, from 87 per 100,000 population to 61 per 100,000. In 2007–08, the rate of CABG procedures for males was 99 per 100,000, compared with 140 in 2000–01. For females, the 2007–08 rate was 25 per 100,000 population compared with 39 in 2000–01 (Figure 11.6).



Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Figure 11.6: Coronary artery bypass graft rates, by sex, 2000–01 to 2007–08

Heart valve repair and/or replacement

Heart valve repair or replacement procedures are performed when the normal flow of blood through the heart is disrupted by damaged valves, making it harder for the heart to pump blood around the body effectively. This can lead to heart failure. The damage to heart valves may be caused by acute rheumatic fever or rheumatic heart disease, coronary heart disease, or forms of congenital heart disease.

In 2007–08, 8,073 heart valve repair or replacement procedures were performed on hospitalised patients. More than half (61%) of these procedures were performed on males (4,963), and 39% were performed on females (3,110). For males the age-standardised rate for the heart valve repair and/or replacement was 47 per 100,000 in the population. For females, it was 27 per 100,000 population.

Pacemaker insertion

A pacemaker electrically stimulates the heart to contract when the heart's electrical system is not working properly. Once the electrical leads are correctly positioned, as confirmed by X-ray, the pacemaker device is placed under the skin and the leads are connected to the pacemaker box.

In 2007–08, 17,010 pacemaker insertion procedures were performed among hospitalised patients. More patients were male (10,050 or 59%) than female (6,960 or 41%). The age-standardised procedure rate for males (101 procedures per 100,000 people) was about double that for females (53 per 100,000 people).

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Cardiac defibrillator implant

A cardiac defibrillator implant is a device implanted into a patient's chest that monitors the heart rhythm and delivers electrical shocks to the heart when required to eliminate abnormal rhythms. They are effective in preventing sudden cardiac death in people at high risk of the life-threatening cardiac arrhythmia known as ventricular fibrillation.

In 2007–08, 3,318 cardiac defibrillator implantation procedures were performed. Most procedures (2,605 or 79%) were performed on males, with 713 (21%) performed on females. The age-standardised procedure rate among males (24 procedures per 100,000 people) was higher than for females (6 per 100,000 people).

Carotid endarterectomy

Carotid endarterectomy is a procedure where atherosclerotic plaques are surgically removed from the carotid arteries in the neck, which supply blood to the brain. This procedure is used to reduce the risk of stroke caused by blockage.

In 2007–08 there were 2,441 carotid endarterectomy procedures performed on hospitalised patients. More patients were male (1,740 or 71%) than female (701 or 29%). Similarly, the age-standardised rate of procedures was higher for males (17 procedures per 100,000 of the population) than for females (6 per 100,000 population).

Heart transplants

A heart transplant involves implanting a working heart from a recently deceased organ donor into a patient. It is usually used to treat severe forms of heart failure or coronary artery disease.

Heart transplants are uncommon, with only 76 procedures performed in 2007–08 in Australia. Most of these procedures were performed on male patients (54 or 71% of procedures). No procedures were performed on patients aged more than 74 years, and a sizeable proportion of patients were young— more than one quarter of procedures were performed on patients less than 35 years of age.

Rehabilitation

Cardiac rehabilitation

Cardiac rehabilitation refers to all measures designed to help people who have recently had an acute coronary event or heart surgery return to a normal and productive life. The broad aims of cardiac rehabilitation are to minimise recovery time after an event such as a heart attack, to maximise the patient's physical, psychological and social functioning, and to introduce and encourage behaviours to minimise the risk of further cardiac events.

There is good evidence that cardiac rehabilitation confers beneficial effects, above usual medical care, on a wide range of risk factors including blood pressure and blood cholesterol, smoking rates, excess weight and insulin insensitivity. Including a counselling component with cardiac rehabilitation can reduce depression and improve the patient's quality of life.

Cardiac rehabilitation often begins in hospital (described as inpatient rehabilitation) and usually includes the provision of basic information and reassurance, supportive counselling, mobilisation and discharge planning. Discharge planning is a process that includes coordination of care with the patient's GP and/or cardiologist, scheduling follow-up reviews and referral to an outpatient rehabilitation program (NHFA & Australian Cardiac Rehabilitation Association 2004).

Outpatient cardiac rehabilitation, which consists of supervised programs, usually commences soon after discharge from hospital (ideally within a few days) and may continue for two to three months. There are no national data on episodes of outpatient cardiac rehabilitation, although a Melbourne study found that around half of patients admitted to hospital with an AMI, or for revascularisation or cardiac bypass surgery, attended a cardiac rehabilitation program (Worcester et al. 2004).

More recently, another Melbourne study of patients admitted for cardiac bypass surgery found that 72% attended outpatient rehabilitation—a substantially higher percentage than in the previous study which was attributed to an automatic referral system and vigorous follow-up by hospital staff (Higgins et al. 2008).

The final phase of cardiac rehabilitation is the maintenance phase, where exercise and risk factor control occurs with minimal or no supervision. The content and structure of maintenance programs vary greatly and there are no national data available regarding their uptake or effectiveness.

Stroke rehabilitation

Stroke rehabilitation encompasses a range of measures designed to help improve a patient's functioning after a stroke and/or to prevent deterioration of functioning. It aims to maximise a patient's physical, psychological, social and financial independence and ideally begins the first day after a stroke.

Various elements of stroke rehabilitation have been shown to significantly improve outcomes for patients. The use of stroke units in hospital is an important part of stroke rehabilitation.

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Patients who were cared for in a specialised stroke unit were more likely to be alive, independent and living at home a year after their stroke than patients cared for in a general ward (Stroke Unit Trialists' Collaboration 2007). In 2007, there were 4,937 episodes of stroke rehabilitation in Australian hospitals. In over 80% of rehabilitation episodes, the patient was later discharged to the community.

Other elements of rehabilitation involving support at hospital discharge and rehabilitation in the community have also been shown to significantly improve patient outcomes. Discharge planning is an important part of long-term care. The destination of the patient after discharge (home versus residential care) is an important decision and is influenced by the patient's level of functioning, the availability of supportive services and the patient's and their families' preferences (NSF 2009b).

After discharge from hospital a patient may receive rehabilitation services in the community which are either centre-based, where patients travel to a hospital or other community facilities to receive rehabilitation services, or the services may be provided in the patient's home or residential facility.

Of the 2,359 stroke patients in the National Stroke Audit who were discharged from hospital, 2% were referred to an outpatient or community-based rehabilitation program.

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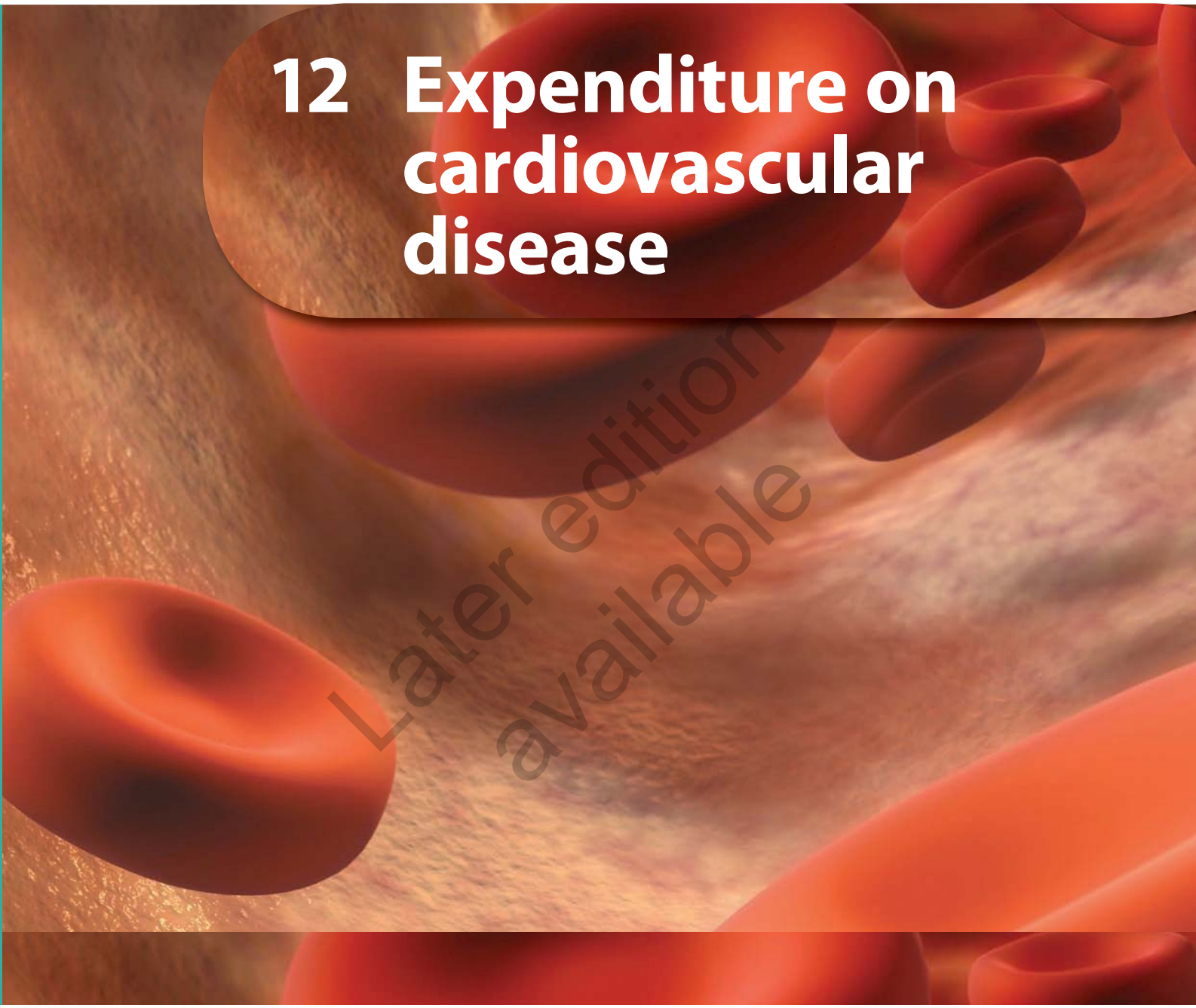

12 Expenditure on cardiovascular disease

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12 Expenditure on cardiovascular disease

This chapter presents the most recent health care expenditure estimates for CVD, including estimates by area of expenditure, age and sex and changes in expenditure over time. It includes expenditure by the Australian Government, state, territory and local governments and the non-government sector (including private health insurance and individual contributions).

Estimates are sourced from the AIHW Disease Expenditure Database (See Appendix C). These estimates report *direct*, *allocated* and *recurrent* expenditure only and do not account for the total amount spent on health.

Direct expenditure

Direct expenditure refers to health care expenses that are immediately related to the diagnosis, treatment or prevention of the condition or disease. Some examples of direct expenditure include spending on medicines and doctors' consultations. Direct expenditure excludes costs that the health system does not accrue, such as travel costs for patients, costs associated with the social and economic burden on carers and families and costs relating to lost productivity or quality and length of life, for which there are only limited data available.

Allocated expenditure

Allocated expenditure refers to spending on health goods and services which can be allocated to a specific disease type. Some areas where expenditure cannot be allocated by disease include hospital non-admitted patient services, over-the-counter medications and health administration.

Recurrent expenditure

Recurrent expenditure refers to expenditure that organisations accrue on a recurring basis for the provision of health services consumed within a year. It includes expenditure on health goods (such as medicines), health services (such as hospital, dental and medical services), public health activities and other activities that support health systems (such as research and administration). It excludes capital expenditure—the expenditure on fixed assets such as new buildings and equipment.

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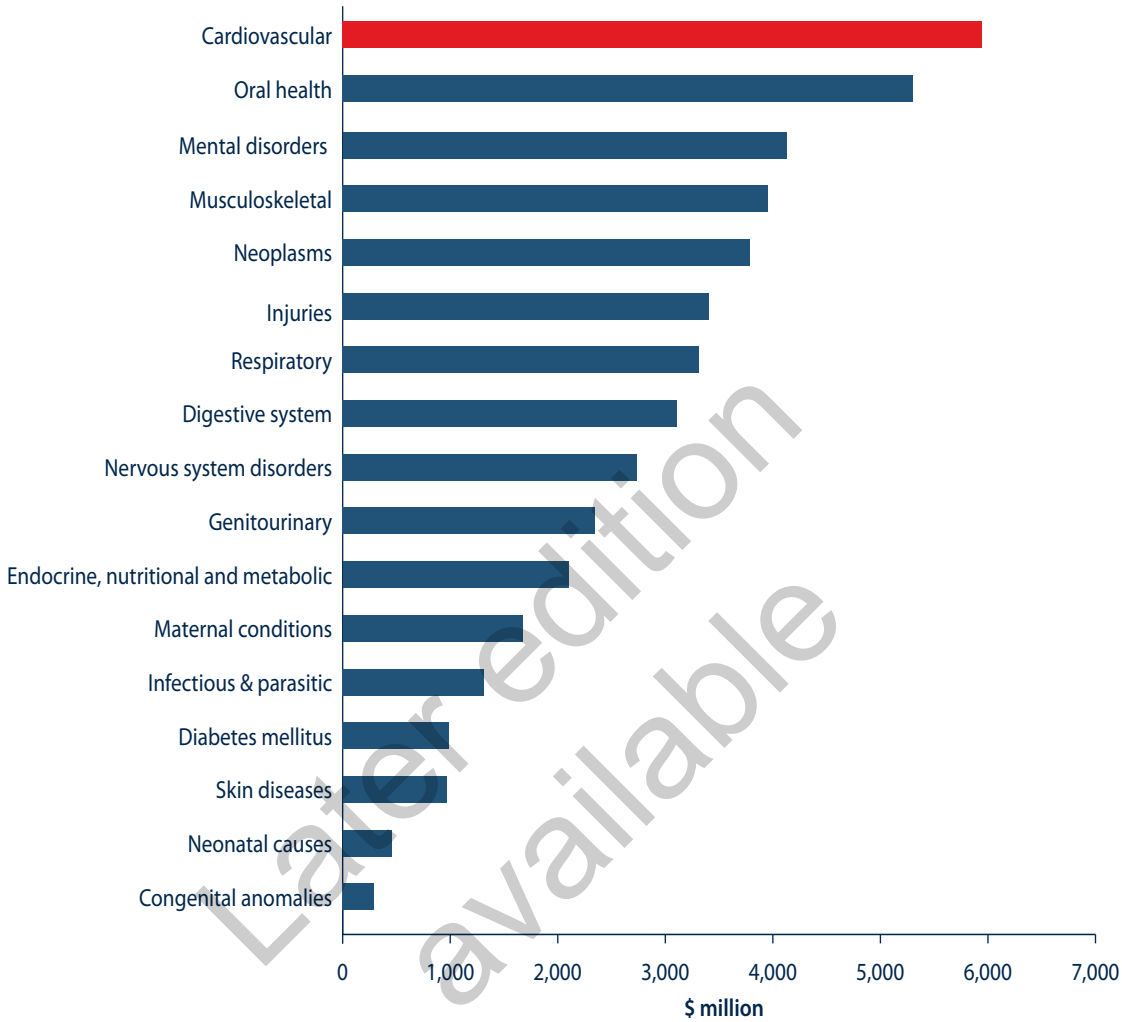
How much is spent on cardiovascular disease?

In 2004–05, CVD accounted for 11% ($5,942 million) of heath care expenditure—more than any other disease group. The high expenditure on CVD reflects its position as the leading cause of death and a major contributor to the overall burden of disease in Australia. Oral health (10% of health expenditure), mental disorders (8%), musculoskeletal diseases (8%) and neoplasms (7%) were the four next most expensive disease groups (Figure 12.1).

The most expensive CVDs were CHD and stroke. Thirty-one per cent ($1,813 million) of CVD expenditure was spent on CHD, while a further 9% ($546 million) was spent on stroke.



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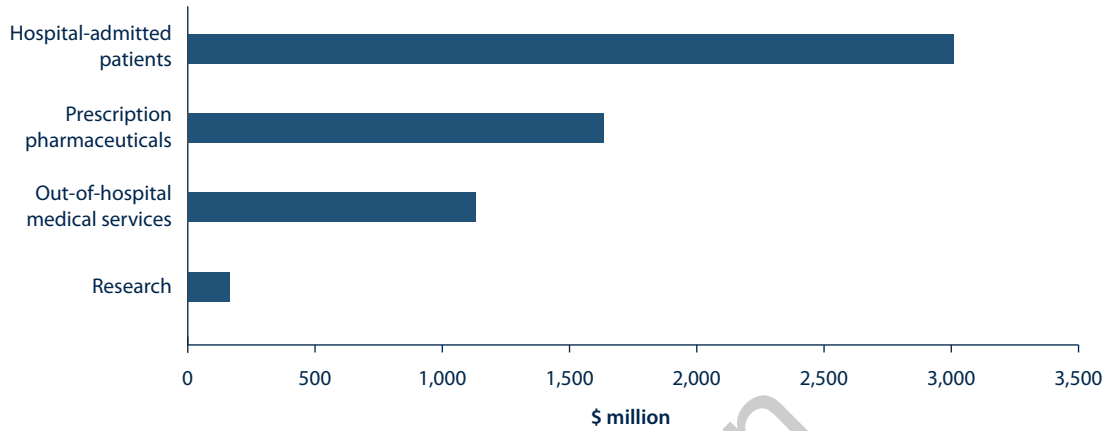
Source: AIHW Disease Expenditure Database.

Figure 12.1: Health care expenditure by disease group, 2004–05

Where is the money being spent?

In 2004–05, just over half ($3,009 million) of CVD expenditure was for hospital-admitted patients. Another 28% ($1,636 million) was spent on prescription pharmaceuticals (including both prescribed medicines subsidised through PBS/RPBS arrangements and prescribed medicines that patients paid for directly. The remainder was spent on out-of-hospital medical services, ($1,133 million) and research ($164 million) (19% and 3% respectively) (Figure 12.2).



Source: AIHW Disease Expenditure Database.

Figure 12.2: Health care expenditure on cardiovascular diseases, by area of expenditure, 2004–05

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Expenditure was distributed differently for each cardiovascular disease. In 2004–05, prescription pharmaceuticals represented 16% of total CHD expenditure, with the comparable figure for stroke being 11%.

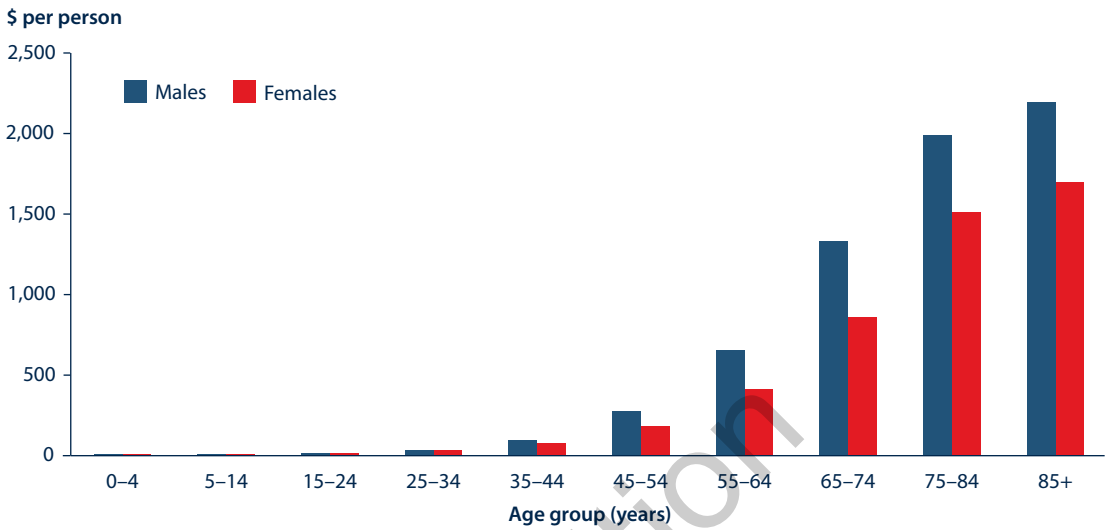
However, these figures exclude much of the expenditure on lipid-lowering medicines, which is allocated to the endocrine, nutritional and metabolic disease group (AIHW 2008d). Given that over $1.4 billion was spent on lipid-lowering medicines in 2008, and these medicines are commonly prescribed for cardiovascular conditions, it is likely that the amount spent on prescription pharmaceuticals for CVD is greatly underestimated (DoHA 2008).

In addition, expenditure on blood-pressure-lowering medicines is allocated to the *Other CVD* category—which includes all CVDs and conditions other than stroke and CHD. But one of the key reasons for treating high blood pressure is to reduce the risk of CHD or stroke so it seems likely that this allocation will add to the underestimate of the true expenditure on prescription pharmaceuticals for CHD and stroke.

Who is it spent on?

Expenditure on CVD in 2004–05 was low among young people but increased sharply from about age 45 years and was highest among those aged 85 years and over (Figure 12.3). For those in the 85 years and over age group, the average annual per person expenditure on CVD was $1,858, compared to $229 for people aged 45-54 years.

Average expenditure per person on CVD was higher among males than females across all age groups, reflecting the higher prevalence of CVD among males. Most of this difference was related to expenditure on hospital-admitted patients where a total of $1,795 million was spent on males compared with $1,214 million on females. Expenditure in the areas of out-of-hospital medical services, prescription pharmaceuticals and research did not vary greatly between males and females and there is some evidence that the higher level of overall CVD expenditure on males may be attributable to differences in the in-hospital diagnosis and treatment of CVD. This is consistent with the fact that in 2007–08, after allowing for the different age structures of the populations, females received most key hospital procedures for CVD at lower rates than males.



Source: AIHW Disease Expenditure Database.

Figure 12.3: Average annual health care expenditure for cardiovascular diseases per person, by age and sex, 2004–05

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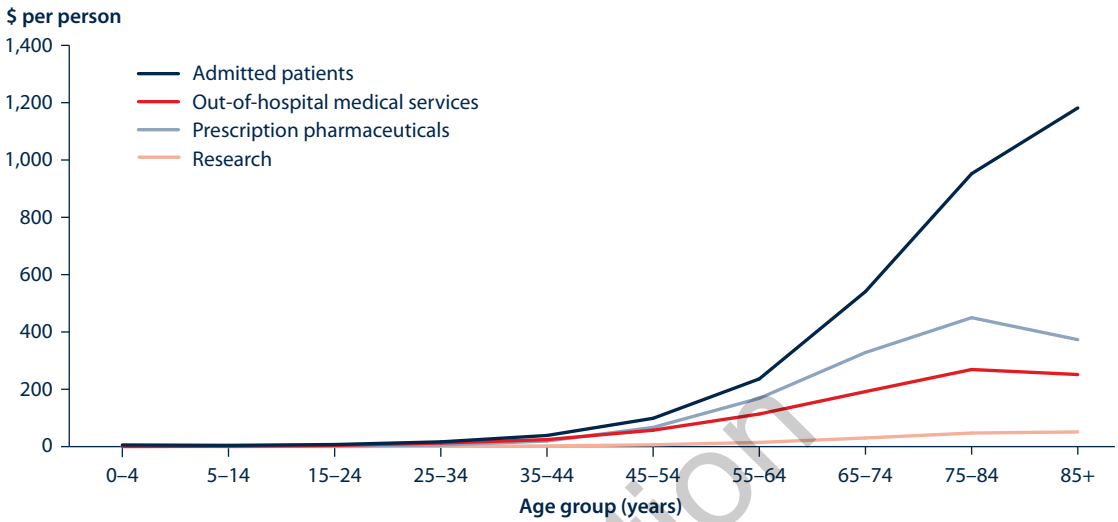
Aboriginal and Torres Strait Islander people

In 2006–07, expenditure on hospitalisations for Indigenous people with CVD was $86.8 million, just under 3% of all expenditure for this area. Per person, this equated to $166 compared with $160 for non-Indigenous people (AIHW 2010e). Rheumatic heart disease (RHD) accounted for 13% of expenditure on hospitalisations for Aboriginal and Torres Strait Islander people—almost six times the per person expenditure for the non-Indigenous population.

Levels of expenditure

The highest levels of health care expenditure, per person, were for patients admitted to hospital, with levels increasing sharply with age. Expenditure for prescription pharmaceuticals and out-of-hospital medical services also increased steadily with age before decreasing slightly for those aged 85 years or over. This decline in the cost of out-of-hospital services for those aged over 85 could indicate that CVD care is increasingly being delivered to the most elderly in hospital, rather than in the community. Expenditure per person on research was small ($8) and did not change much with age (Figure 12.4).



Source: AIHW Disease Expenditure Database.

Figure 12.4: Health care expenditure for all cardiovascular diseases per person, by age and area of expenditure, 2004–05

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Changes in expenditure over time

Between 2000–01 and 2004–05, after adjusting for inflation in health prices and changes in classifications, total expenditure for CVD increased by 18%—slightly less than the increase for all diseases (20%).

For CVD, the largest increase in spending was for out-of-hospital medical services (24%), followed by prescription pharmaceuticals (21%) and hospital-admitted patients (15%). In contrast, expenditure on CVD research decreased by 8% over this period, although it rose by 24% for all diseases (Table 12.1).

Table 12.1: Change in inflation adjusted allocated health expenditure, constant prices, 2000–01 to 2004–05 (per cent)

	Hospital-admitted patients[a]	Prescription pharmaceuticals[b][c]	Out-of-hospital medical services	Research	Total
Disease group		**Per cent change**			
Cardiovascular disease	15	21	24	-8	18
Total allocated health expenditure	20	18	20	24	20

(a) Includes private medical services provided in hospital and expenditure on Highly Specialised Drugs.

(b) Includes all pharmaceuticals for which a prescription is needed, including private prescriptions and under-copayment prescriptions but excludes Highly Specialised Drugs.

(c) Excludes over-the-counter medications such as vitamins and minerals, patent medicines, first aid and wound care products, analgesics, feminine hygiene products, cold sore preparations, and a number of complementary health products that are sold in both pharmacies and other retail outlets.

Note: Excludes optometric services, dental, community mental health and public health cancer screening.

Source: AIHW Disease Expenditure Database.

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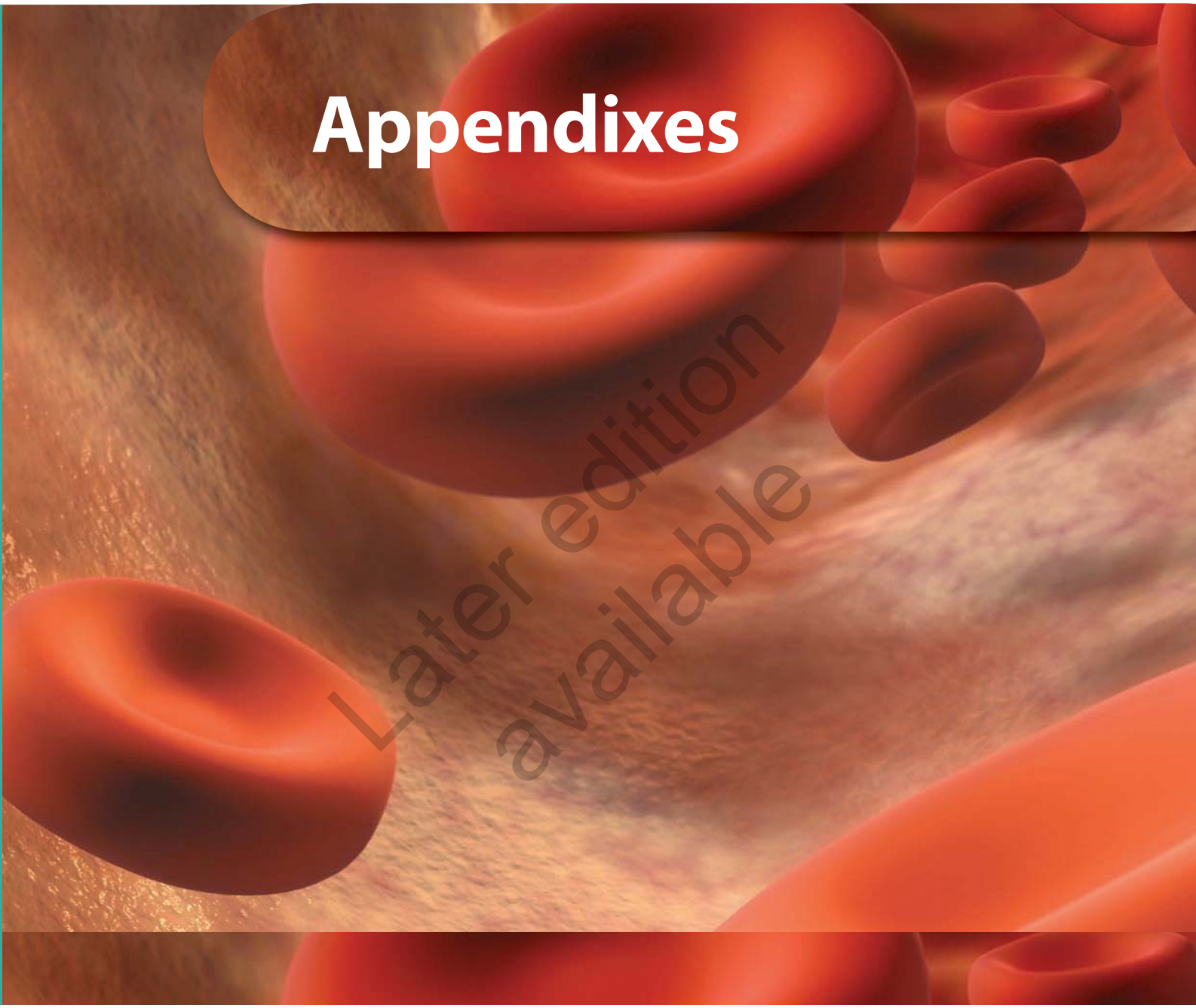
Appendixes



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Appendix A

Methods and definitions

Age-specific rates

Age-specific rates are calculated by dividing the number of cases occurring in each specified age group by the corresponding population in the same age group, expressed as a rate (for example, number per 100,000 persons). Information on the populations used in this report in provided in the section on populations below.

Age-standardised rates

Age-standardisation is a method used to eliminate the effect of differences in population age structures when comparing populations with different age structures, and where age affects the variable being compared. This is the case with CVD, which is occurs more often among the elderly. Age-standardisation is used in this report when comparing rates across different periods of time, different geographical areas, different socioeconomic groups, or other different populations. The direct method of age-standardisation is used throughout this report.

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Direct age-standardisation

This method of age-standardisation is used when the population under study is large and the age-specific rates can be reliably estimated. The calculation of direct age-standardised rates consists of three steps:

Step 1: Calculate the age-specific rate for each age group.

Step 2: Calculate the expected number of cases in each age group by multiplying the age-specific rates by the corresponding standard population for each age group.

Step 3: Sum the expected number of cases in each age group and divide this sum by the total of the standard population to give the age-standardised rate.

For most of the age-standardised rates, the standard population used is the Australian estimated resident population as at 30 June 2001. See the section on population for more information.

Significance testing

The observed value of a rate may vary because of the influence of chance and natural variation. Therefore to provide an approximate indication of whether two rates are statistically different, 95% confidence intervals can be calculated, and significant differences highlighted.

A 95% confidence interval describes a span of numbers around the estimate which has a 95% chance of including the true value. When comparing two groups, if the two confidence intervals do not overlap, the reader can be confident that the difference between the groups is real, and not due to chance.

Confidence intervals were calculated for survey data in this report.

Calculation of confidence intervals for survey data

This method has been used where the available data are weighted estimates based on survey data.

The standard error of the estimated for O/E by Kendall and Stuart (1969) is calculated as:

$$SE = \sqrt{\left[\left(\frac{O^2}{E}\right) \times VAR_e\right] + VAR_o/E^2]}$$

where:

- O/E = ratio of the observed to expected number of cases
- O = the number of synthetic observed rates. The ABS provided weighted estimates of the total number of cases (synthetic numbers), based on the number of cases in the survey and a weighting factor
- E = the number of synthetic expected cases (based on the numbers of synthetic observed cases)
- VAR_o = = the variance for the synthetic total number of observed cases.

The variance is the square of the standard error associated with the observed or expected number, calculated by the ABS and provided with the base data they had provided:

$$VAR_e = \sum (pop/POP)^2 \times (SE_e)^2$$

where:

- pop = the study population in a specific age group
- POP = the standard population in a specific age group
- SE_e = the standard error of the expected synthetic number of cases in the area in a specific age group.

The lower 95% confidence limit = (O/E) – (1.96 x SE).

The upper 95% confidence limit = (O/E) + (1.96 x SE).

As with all statistical comparisons, care should be exercised in interpreting the results. A non-significant difference between two rates may indicate no true difference, or could indicate that numbers of observations are too small to detect a true statistically significant difference. Judgment should be exercised in deciding whether the size of the difference observed is of practical importance.

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Reporting deaths

Data on deaths in this report are sourced from the AIHW National Mortality Database. (See 'Main data sources' in Appendix C) Unless otherwise specified, the cause of death identified in this report is based on the 'underlying cause'—the condition believed to be the primary cause of death. Any other condition or event that is not the underlying cause, but is still considered to contribute to the death, is known as an associated cause. In Australia, the underlying cause is derived from information on the death certificate, using an automated process.

In this report, death data are collated according to the year of registration of the death and not the year of death. While for the most part, year of death and registration coincide, deaths at the end of each calendar year may be held over until the following year, as will deaths in which the cause requires further examination by a coroner. In recent years, less than 5% of deaths were held over from one year to the next for processing.

See the section below for information on the population used to calculate death rates in this report.

Comparability factors

In processing deaths from 1 January 1997, Australia adopted the use of the automated coding system and introduced ICD-10 codes (see 'Classifications' in Appendix B). As a result, there is a break in the underlying cause of death series between 1996 and 1997. Comparability factors have been calculated that can be applied to death counts before 1996 to make them comparable with data from 1997 onwards. Comparability factors close to 1.0 indicate there were no substantial differences between automated ICD-10 coding and manual ICD-9 coding (Table A1).

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Table A1: Comparability factors for the ICD-9 to ICD-10 transition for various cardiovascular diseases

Condition	Comparability factor
Cardiovascular disease	1.00
Coronary heart disease	1.01
Stroke	0.83
Heart failure/ cardiomyopathy	0.98
Peripheral vascular disease	0.97[(a)]
Acute rheumatic fever and rheumatic heart disease	0.69
Congenital heart disease	1.03[(b)]

(a) Comparability factor for diseases of arteries, arterioles and capillaries (ICD-10 codes I70–I79).

(b) Comparability factor for all congenital malformations (ICD-10 codes Q00–Q99).

No adjustment was made to the number of deaths where the comparability factor was close to 1.0 (±0.05). Comparability factors of 0.83 and 0.69 were applied to observed numbers of deaths for stroke and acute rheumatic fever/rheumatic heart disease, respectively, for the period 1987–1996. These comparability factors were calculated at the population level—that is, the same comparability factor was applied to the number of deaths for each age and sex group combination.

Reporting hospitalisations

Information on hospitalisations in Australia is contained in the AIHW National Hospital Morbidity Database (NHMD). See the 'Main data sources' section for more information. In this report a 'hospitalisation' refers to an episode of admitted care, which can be a total hospital stay (from admission to discharge, transfer or death) or a portion of a hospital stay beginning or ending in a change of type of care (for example from acute to rehabilitation). The same person can have multiple 'separations' within the same hospitalisation period and, as the database is event based, it is currently not possible to track individuals. For this reason the data presented in this report do not represent the number or proportion of people admitted to hospital in Australia with CVD. Note also that some care types such as unqualified neonates, hospitalised boarders and organ procurement hospitalisations are not included in the count of hospital separations.

There are two distinct types of diagnoses recorded in the database—principal and additional. The principal diagnosis is the diagnosis established after study to be chiefly responsible for occasioning an episode of admitted patient care. The principal diagnosis is usually a disease, injury or poisoning, but can also be the specific care or service provided for a current condition (for example, dialysis for kidney disease), or other reasons for hospitalisation. Unless stated otherwise, hospitalisation rates presented in this report refer to the principal diagnosis. The additional diagnosis is a condition or complaint that coexists with the principal diagnosis or arises during the episode of care.

Additional diagnoses should be interpreted as conditions that affect patient management in terms of requiring any of the following:

- Commencement, alteration or adjustment of therapeutic treatment
- Diagnostic procedures
- Increased clinical care and/or monitoring.

Additional diagnoses form the basis of the analyses of comorbidity presented in this report.

Also note that the principal diagnosis is determined at the end of the hospitalisation episode. It is therefore possible for the principal diagnosis to be different from the complaint which caused a person to present to hospital. Hospitals data therefore report the condition a patient was hospitalised *with*, rather than the condition they were hospitalised *for*.

See the section below for information on the population used to calculate hospitalisation rates in this report.

Comparability factors

The NHMD adopted the use of the automated coding system and introduced ICD-10 coding in the 1998–99 financial year (see 'Appendix C Classifications'). As a result, there is a break in the underlying cause of death series between 1997–98 and 1998–99. Unlike for deaths data, no comparability factors are available for hospitalisation data. It is important to be aware of the potential for coding changes to have affected the trends in hospitalisations presented in this report.

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Reporting Indigenous data

Reporting Indigenous death and hospitalisation data are problematic because of the under-identification of Indigenous Australians in these databases. Not all states and territories in Australia are able to fully report the Indigenous status of patients in their death and hospitalisation data accurately. In this report, data on Indigenous deaths and hospitalisations were limited to specific jurisdictions (based on the patient's state of usual residence) as these are the ones that have been judged to have sufficiently accurate Indigenous identification.

Deaths

The ABS has assessed the quality of Indigenous deaths in death registration data by state and territory in the Census Data Enhancement Indigenous Mortality Quality Study. This study involved linking Census records with death registration records to examine differences in reporting of Indigenous status across two data sets. This assessment indicates that the Indigenous identification rate is 87% or higher in New South Wales, Queensland, Western Australia and the Northern Territory, and around 65% for the remaining jurisdictions. Historically, Indigenous identification in South Australia, Western Australia and the Northern Territory has been of sufficient quality to include in analyses from 1991 onwards. Queensland was included in analyses from 1998 onwards and in 2010 a decision was made to include data from New South Wales from 2001 onwards.

Hospitalisations

The AIHW has completed an assessment of the level of Indigenous under-identification in hospital data in all states and territories. Results of this assessment indicate that from 2004-05 onwards New South Wales, Victoria, Queensland, Western Australia, South Australia and the Northern Territory (public hospitals only) hospitals data have adequate Indigenous identification (80% or higher overall levels of Indigenous identification) for national reporting. The proportion of the Indigenous population covered by these jurisdictions is 96%.

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Reporting data by remoteness

Comparisons of region in this report use the Australian Standard Geographical Classification (ASGC). The ASGC is a classification system developed by the ABS which groups Australian regions into six areas, called remoteness areas, based on their distance from major population centres and services. The six remoteness areas are:

- *Major cities*
- *Inner regional*
- *Outer regional*
- *Remote*
- *Very remote*
- *Migratory*

Data from *Migratory* areas are not analysed in this report. Also, throughout the report *Remote* and *Very remote* areas are grouped together to allow sufficient numbers for analysis. The boundaries of the different remoteness areas are re-drawn after each Census to account for changes to available services and population change. The remoteness areas used in this report are based on the 2006 Census.

Reporting data by socioeconomic group

The ABS has constructed a number of socioeconomic indexes to classify areas on the basis of social and economic information collected in the Census of Population and housing.

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

For analysis, the population was divided into five groups with roughly equal populations (each around 20% of the total) based on the level of disadvantage of the statistical local area of their usual residence. So the first group includes the 20% of the population living in areas with the highest levels of relative disadvantage, while the last group includes the 20% of the population living in areas with the lowest levels of relative disadvantage.

It is important to note that the index of relative socioeconomic disadvantage relates to the average disadvantage of all people living in a statistical local area, not to the level of disadvantage of a specific individual. As the population of many areas covers a broad range of socioeconomic disadvantage, these measures will generally underestimate the true effect of disadvantage on health.

The index of relative socioeconomic disadvantage values used in this report are based on the 2006 Census. These values have been mapped to the 2007 year statistical local area boundaries for both death and hospitalisation analyses.

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Populations used in this report

Population data are used throughout this report to calculate rates. The population data used are estimated resident populations (ERPs) derived from the ABS Census of Population and Housing. ERPs adjust Census data to add people missed by the Census and people overseas on census night, and to remove overseas visitors. In between census years, the ERPs are updated using indicators of population change such as deaths, births and net migration. The ERPs used in this report are based on the 2006 Census.

Where a rate is calculated for a calendar year, for example with death data, the population used is the ERP as reported at 30 June of that year. Where a rate is calculated for a financial year, as with hospitalisation data, the population used is the ERP for 31 December of the year in the middle of the span. For example, to calculate the hospitalisation rate of the 2007–08 financial year, the population from 31 December 2007 is used.

Throughout this report, rates of deaths and hospitalisations are age-standardised. In these cases, the standard population used to calculate the age-standardised rate is the Australian ERP as at 30 June 2001.

Indigenous population

Australia's Indigenous population is calculated from the Census, and uses ERPs as described above. However, because of the smaller Indigenous population it is not possible to accurately estimate Indigenous populations by age, sex and remoteness area between census years. Therefore, all calculations of Indigenous rates in this report use the Indigenous populations as at 30 June 2006, the census year.

Remoteness area population

The remoteness area populations used to calculate rates in this report are the ERPs at 30 June for the given year, for both deaths and hospitalisations. In the case of hospitalisations, which are reported by financial year, the population used is from the beginning of the relevant financial year. For example, hospitalisation rates by region in 2007–08 would use the remoteness area ERP for 30 June 2007.

Population of socioeconomic areas

Populations of socioeconomic areas used in this report are derived from 2007 estimates of Statistical Local Area (SLAs) populations (as at 30 June of that year). SLAs are ranked according to their score on the index of relative socioeconomic disadvantage, and then divided into 5 roughly equal groups based on their population. That is, the 20% of the population with the highest relative disadvantage score are allocated to the first group, the next 20% to the second, and so on. The same populations were used to calculate rates for both deaths and hospitalisations.

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Appendix B

Classifications

Anatomical Therapeutic Chemical (ATC) classification

ATC codes are used in this report to classify medicines. This classification groups medicines according to the body organ or system they act upon, their therapeutic characteristics, and their chemical characteristics. A complete list of the medicine classes included in this report is shown in Table A2.

Table A2: Anatomic Therapeutic Chemical medicine classes included in this report

ATC code	Description
B01	Antithrombotic agents
C01	Cardiac therapy
C01B	Antiarrythmics
C01DA	Nitrates
C02	Antihypertensives
C03	Diuretics
C04	Peripheral vasodilators
C07	Beta-blocking agents
C08	Calcium-channel blockers
C09	Renin-angiotensin system agents
C10	Lipid-modifying agents

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International Classification of Disease (ICD) codes

Australia uses the International Statistical Classification of Diseases and Related Health Conditions, for coding causes of death. Analyses in this report of the years between 1987 and 1996 used the Ninth Revision (ICD-9); analyses after 1996 used the Tenth Revision (ICD-10).

For hospital diagnoses and procedures, a slightly different classification, modified for Australia, is used. However, this classification does not affect the codes used in this report. In this report, hospital data up to 1997–98 used the ICD-9-CM (International Classification of Diseases and Related Health Conditions, Ninth Revision, Clinical Modification) classification. After 1997–98, the ICD-10-AM classification (International Statistical Classification of Diseases and Related Health Conditions, Tenth Revision, Australian Modification) was used. The Australian Classification of Health Interventions (ACHI) is Australia's intervention classification and is used in conjunction with ICD-10-AM to code interventions. Procedure codes used in this report are from the ACHI, noting that not all procedures are surgical. The National Centre for Classification in Health (NCCH) issues new editions of ICD and ACHI codes every two years. Details of the codes used for diagnosis and procedures in this report are given below (Tables A3 and A4).

Table A3: International Classification of Disease (ICD) codes used in this report

Disease codes	ICD-9 and ICD-9-CM codes	ICD-10 and ICD-10-AM codes
Cardiovascular disease	390–459	I00–I99
Coronary heart disease	410–414	I20–I25
Acute myocardial infarction	410	I21
Angina	413	I20
Cerebrovascular disease	430–438	I60–I69
Stroke	430–434, 436	I60–I64
TIA	435	G45
Heart failure and cardiomyopathy	414.8, 428.0, 428.1, 428.9, 425.2, 425.4, 425.5, 425.7, 425.8, 425.9	I50, I25.5, I42.0, I42.5–I42.9, I43
Heart failure	428	I50
Peripheral vascular disease	440–444	I70–I74
Atherosclerosis of peripheral arteries	..	I70.2
Abdominal aortic aneurysm	..	I71.3–I71.4
Acute rheumatic fever and Rheumatic heart disease	390–398	I00–I09
Congenital heart disease	745–747	Q20–Q28
Transposition of the great vessels	..	Q20.3
Tetralogy of Fallot	..	Q21.3
Hypoplastic left heart syndrome	..	Q23.4
Coarctation of the aorta	..	Q25.1
Diabetes	..	E10–E11, E13–E14, O24.0–O24.4, O24.9
Chronic kidney disease	..	N00–N09, N11–N12, N14–N16, N18–N19, N25–N28, N39.1–N39.2, Q60–Q63, T82.4, T86.1, Z49, Z94.0, Z99.2, E10.2, E11.2, E13.2, E14.2, I12–I13, I15.0–I15.1

.. Codes not included in this report.

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Table A4: Australian Classification of Health Interventions (ACHI) codes for procedures in hospital

Procedure codes	ACHI code
Coronary angiography	38215-00, 38218-00, 38218-01, 38218-02 (block:668) and diagnosis codes I20–I25, I30–I52
Percutaneous coronary interventions	35304-00, 35305-00 (block:670) 35304-01, 35305-01 (block:670) 35310-00, 35310-01, 35310-02 (block: 671) 35310-03, 35310-04, 35310-05 (block: 671) and diagnosis codes I20–I25
Coronary artery bypass grafting	38497-00 to 38497-07, 38500-00 to 38500-04, 38503-00 to 38503-04, 90201-00 to 90201-03 (blocks: 672–679) and diagnosis codes I20–I25, I34, I35
Heart transplant	90205-00, 90205-01 (block: 660)
Cardiac defibrillator implants	38524-00, 38521-01, 38521-02, 38521-03 (block:653) and diagnosis codes I20, I21,I25, I42–I51
Valve replacement, repair or reconstruction	• Aortic Valve (Blocks 621, 622, 623, 624): 38456-10, 38483-00, 38270-01, 38480-00, 38481-00, 38488-00, 38488-01, 38489-00, 38489-01, 38456-15, 38653-04, 38475-02, 38477-02. • Mitral valve (Blocks 625, 626, 627, 628, 629, 630): 38487-00, 38485-01, 38270-02, 38480-01, 38481-01, 38475-00, 38477-00, 38488-02, 38488-03, 38489-02, 38485-00, 38456-16, 38653-05. • Tricuspid valve (Blocks 631, 632, 633, 634, 635): 38456-11, 38480-02, 38481-02, 38475-01, 38477-01, 38488-04, 38488-05, 38489-03, 38456-17, 38653-06. • Pulmonary valve (Blocks 636, 637, 638): 38456-01, 38270-03, 38488-06, 38488-07, 38489-04, 38489-05, 38456-18, 38653-07. and diagnosis codes I01–I09, I20–I25, I33–I39, I42–I52, Q20–Q25, T82
Pacemaker insertion	38281-00, 38281-01, 38281-02, 38281-03, 38281-04, 38281-05, 38281-06, 38281-07, 38281-08, 38281-09, 38281-10, 38281-11, 38281-12, 38281-13 (blocks: 650, 651, 652) and diagnosis codes I08, I20, I21, I25, I34–I39, I42–I52
Echocardiography	55112-00, 55118-00, 55130-00 (block: 1942) and diagnosis codes I00–I52
Carotid endarterectomy	33500–00 (block: 700) and diagnosis codes G45, I63–I67
Computerised tomographic (CT) brain scan in hospitalisations	56001-00, 56007-00, 56010-02, 56010-03 (blocks: 1952, 1953) and diagnosis codes G45, I60–I64
Magnetic resonance imaging (MRI) brain scan in hospitalisations	90901-00 (block: 2015) and diagnosis codes G45, I60–I64

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Appendix C

Main data sources

AIHW Disease Expenditure Database

A comprehensive database that allows expenditure estimates to be produced by source of funds (that is, Australian Government, state government or private) for each area of expenditure.

This report provides direct health expenditure on cardiovascular diseases under four categories:

- admitted patient hospital services covering the expenditure on services provided to an admitted patient, including expenditure on medical services delivered to private admitted patients in hospitals
- prescription pharmaceuticals including prescriptions subsidised under government schemes (for example, Pharmaceutical Benefits Scheme) and private prescriptions
- out-of-hospital medical services comprising medical services funded under the Medical Benefits Schedule, such as primary health visits, pathology and specialist services. Practice Incentive Payments are also included in this category
- research including health socioeconomic research funded by tertiary institutions, private non-profit organisations and government. Commercial research funded by private business is not included.

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AIHW GRIM (General Record of Incidence of Mortality) Books

The National GRIM Books are a collection of dynamic and interactive workbooks comprising cause-specific Australian mortality information for the most recent years (currently to 2007) and historically, for many causes, to 1907. Individual workbooks have been created for over 150 causes (or combination of causes) of death. The mortality data are tabulated by cause of death, age, sex and year of registration of death. These data together with population estimates are used to calculate annual age-specific and age-standardised mortality rates, and other summary measures of mortality. They are constructed to allow deaths data to be analysed over the period 1907-2010, however, data for all years and every cause may not always be available.

AIHW National Hospital Morbidity Database

The National Hospital Morbidity Database (NHMD) is compiled from data supplied by the state and territory health authorities. It is a collection of electronic confidentialised summary records for separations (that is, episodes of admitted patient care) in essentially all public and private hospitals in Australia. The data include demographic, administrative and clinical information, including patient diagnoses and other procedures. The Nation Minimum Dataset for Admitted Patient Care forms the basis of the database, ensuring a high standard of data comparability. Data presented in this report are for the period July 1993 to June 2008.

AIHW National Mortality Database

Contains information on the cause of death supplied by the medical practitioner certifying the death or by a coroner. Registration of deaths is the responsibility of the state and territory Registrars of Births, Deaths and Marriages. These data are then collated and coded by the ABS. In this report, unless otherwise specified, deaths data relate only to the underlying cause of death. Data presented in this report are for deaths registered during the period January 1987 to December 2007.

Australian Congenital Anomalies Monitoring System

Contains data based on notifications of major congenital anomalies to births defects registers in New South Wales, Victoria, Western Australia and South Australia and on data collected on congenital anomalies in Queensland, Tasmania and the Australian Capital Territory. The Northern Territory is currently unable to provide data in a format enabling it to be compiled with data from the other states and territories.

Drug Utilization Sub-Committee Database

Held at the Australian Government Department of Health and Ageing (DoHA), monitors the community (i.e. non-public hospital) use of prescription medicines in Australia. This database combines information on prescriptions subsidised by the PBS, the Repatriation Pharmaceutical Benefits Scheme and an estimate from the Pharmacy Guild Survey of those prescriptions that are not subsidised (i.e. private prescriptions and PBS prescriptions priced under the general patient copayment threshold). The Pharmacy Guild Survey collects dispensing information each month from a random sample of about 250 pharmacies throughout Australia. Information on drugs prescribed in public hospitals and on highly specialised drugs available to outpatients through public hospital pharmacies under section 100 of the *National Health Act* are not included in this database. The data used in this report are sourced from the DoHA publication Australian Statistics on Medicines (DoHA 2008).

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National Aboriginal and Torres Strait Islander Health Survey (NATSIHS)

The NATSIHS is conducted by the ABS to obtain national information on the health of Indigenous Australians, their use of health services and health-related aspects of their lifestyle. The 2004–05 survey collected information from a sample of 10,439 people (about 1 in 45 of the total Indigenous population) from all states and territories, including *Remote and Very remote areas*, from August 2004 to July 2005.

National Drug Strategy Household Survey (2007)

Was conducted between July and November 2007. This was the ninth survey in a series which began in 1985, and was the fourth to be managed by the AIHW. Almost 25,000 Australians aged 12 years and over participated in the survey, in which they were asked about their knowledge of and attitudes towards drugs, their drug consumption histories, and related behaviours. Most of the analyses in this report are based on the population aged 14 years and over, as this allows consistent comparison with earlier survey results.

National Health Surveys (2004–05 and 2007–08)

Were conducted by the ABS in all states and territories and across all age groups. One adult (aged 18 years or over) and one child (where applicable) for each sampled dwelling were included in the survey. The surveys were designed to obtain national benchmarks on a wide range of health issues, and to enable change in health to be monitored over time. Information was collected about: the health status of the population; health-related aspects of lifestyle and other health risk factors; and the use of health services and other actions people had recently taken for their health

National Nutrition Survey (1995)

Conducted by the ABS, this survey was the largest and most comprehensive Australian survey of food and nutrient intake, dietary habits and body measurements ever undertaken. The survey collected information from a sub-sample of respondents from the 1995 National Health Survey, about 13,800 people from urban and rural areas of Australia. The National Nutrition Survey was conducted over a 13-month period from February 1995 to March 1996. This survey provided prevalence estimates of high blood pressure and obesity based on measured data.

Northern Territory Rheumatic Heart Disease Register

Includes data related to rheumatic heart disease and acute rheumatic fever diagnosis, hospitalisations, compliance with prophylactic antibiotic treatment, clinical progress, surgery and mortality. The register is run by Territory Health. Confidentialised data from the register were provided to AIHW.

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Organisation for Economic Co-operation and Development (OECD) Health Data

OECD Health Data is a comprehensive source of comparable statistics on health and health systems across industrialised countries. The database contains information on health status, health care services and health care spending for member countries. The information is provided by OECD member countries, and while efforts are made to ensure comparability, in many cases diverse reporting definitions and standards mean comparisons across countries should be made cautiously.

Pharmaceutical Benefits Database

The database holds personal and other information based on prescriptions recorded in the PBS and is one of the main sources of information on medicines prescribed in Australia.

Risk Factor Prevalence Surveys (1980, 1983, 1989)

This series of surveys were conducted by the National Heart Foundation of Australia to get national information on biomedical and behavioural risk factors in Australia and to monitor trends. The overall study collected information from a sample of about 22,000 adults living in capital cities of Australia (Canberra and Darwin were not included in the 1980 and 1983 surveys) between May/June and December of the survey year. These surveys provided prevalence estimates of high blood pressure and high blood cholesterol based on measured data.

Survey of Disability, Ageing and Carers (2003)

Collects information about people of all ages with a disability, older people (aged 60 years and over) and people who provide assistance to older people with disabilities. The 2003 Survey of Disability, Ageing and Carers was conducted throughout Australia, from June to November 2003. The survey included people in both private and non-private dwellings, including people in establishments where care is provided, but excluded those in correctional institutions.

Survey of Mental Health and Wellbeing (2007)

Was conducted by the ABS from August to December 2007. The survey collected information from about 8,800 Australians aged 16–85 years. The survey provides information on the prevalence of selected lifetime and 12-month mental disorders, by the major disorder groups:

- anxiety disorders (for example, social phobia)
- affective disorders (for example, depression)
- substance use disorders (for example, alcohol harmful use).

The survey also provides information on the level of impairment, health services used for mental health problems, physical conditions, social networks and care-giving, as well as demographic and socioeconomic characteristics.

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The Australian Diabetes, Obesity and Lifestyle (AusDiab) Study (1999–2000)

Was conducted by the International Diabetes Institute, and designed to provide national estimates of the prevalence of diagnosed and undiagnosed diabetes. It also provided national measurements of blood pressure, blood lipids, blood glucose, body fat, height, weight, and waist and hip circumference, as well as self-reported information on CVD, anti-hypertensive and lipid lowering medication use, diet, smoking, alcohol consumption, physical activity, and general health and wellbeing. The study collected information in urban and non-urban areas in all states and the Northern Territory for more than 11,000 people aged 25 years and over who underwent a physical examination. This represents a response rate of 37% (Dunstan et al. 2002-08). Analysis of these data by the AIHW included only those people for whom all relevant data were available. In this report, measured prevalence data on high blood pressure, high blood cholesterol and diabetes were obtained from this source.

The Bettering the Evaluation and Care of Health (BEACH) program

Is a continuous national study of general practice activity in Australia. Data are collected by the Australian General Practice Statistics and Classification Centre, the University of Sydney, in collaboration with the AIHW. BEACH began in April 1998 and involves changing random samples of about 1,000 GPs per year, each of whom records details about 100 consecutive patient encounters. Data include information about the GP encounter, the problems managed and the treatments delivered. BEACH encounter data are weighted to be representative of all encounters and adjustments are made in analyses to allow for the tendency for patients to cluster around an individual GP or practice.

Appendix D

Detailed statistical tables

Table A3.1: Cardiovascular disease hospitalisation rates, 2007–08

	Hospitalisations per 100,000 population		
Population subgroup	**Males**	**Females**	**Persons**
Age group (years)			
<25	134	115	125
25–34	450	366	408
35–44	1,088	802	944
45–54	2,379	1,381	1,876
55–64	4,961	2,603	3,780
65–74	9,122	5,211	7,124
75–84	14,692	10,157	12,162
85 and over	19,239	14,707	16,219
Age-standardised rate	*2,599*	*1,651*	*2,099*
Socioeconomic group			
1st quintile (lowest)	2,884	1,877	2,358
2nd quintile	2,761	1,759	2,235
3rd quintile	2,583	1,639	2,088
4th quintile	2,505	1,557	2,003
5th quintile (highest)	2,355	1,436	1,859
Aboriginal and Torres Strait Islander status[(a)]			
Indigenous	3,956	3,273	3,588
Other Australians	2,695	1,686	2,160
Remoteness			
Major cities	2,528	1,578	2,019
Inner regional	2,799	1,791	2,272
Outer regional	2,766	1,870	2,316
Remote and very remote	2,873	2,185	2,557

(a) Data from NSW, Vic, Qld, WA, SA and NT (public hospitals only).

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

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Table A3.2: Cardiovascular disease death rates

Year	Population subgroup	Deaths per 100,000 population		
		Males	Females	Persons
2007	**Age group (years)**[1]			
	<25	2	2	2
	25–34	6	4	5
	35–44	23	10	16
	45–54	69	24	46
	55–64	172	59	115
	65–74	484	245	362
	75–84	1,856	1,285	1,536
	85 and over	6,388	6,321	6,343
	Age-standardised rate	*232*	*171*	*200*
2007	**Socioeconomic group**			
	1st quintile (lowest)	265	186	223
	2nd quintile	242	177	208
	3rd quintile	233	170	200
	4th quintile	216	157	184
	5th quintile (highest)	190	146	166
2005–2007	**Aboriginal and Torres Strait Islander status**[a]			
	Indigenous	453	356	404
	Non-Indigenous	332	241	284
2005–2007	**Remoteness**			
	Major cities	224	161	190
	Inner regional	259	192	223
	Outer regional	276	202	239
	Remote and very remote	298	218	262

(a) Data from NSW, Qld, WA, SA and NT only.

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Later edition available

Table A4.1: Coronary heart disease hospitalisation rates, 2007–08

	Hospitalisations per 100,000 population		
Population subgroup	**Males**	**Females**	**Persons**
Age group (years)			
<25	3	1	2
25–34	40	13	26
35–44	302	90	195
45–54	1,029	332	677
55–64	2,344	821	1,581
65–74	4,038	1,784	2,887
75–84	5,379	3,056	4,083
85+	5,463	3,589	4,215
Age-standardised rate	*1,000*	*443*	*709*
Socioeconomic group			
1st quintile (lowest)	1,162	540	840
2nd quintile	1,102	504	791
3rd quintile	990	442	705
4th quintile	932	399	653
5th quintile (highest)	843	336	573
Aboriginal and Torres Islander status[(a)]			
Indigenous	1,867	1,316	1,567
Other Australians	1,034	448	727
Remoteness			
Major cities	959	407	666
Inner regional	1,122	511	806
Outer regional	1,060	524	793
Remote and very remote	1154	662	925

(a) Data from NSW, Vic, Qld, WA, SA and NT (public hospitals only).

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

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Table A4.2: Coronary heart disease death rates

Year	Population subgroup	Deaths per 100,000 population		
		Males	Females	Persons
2007	**Age group (years)**			
	<25	—	—	—
	25–34	3	1	2
	35–44	13	3	8
	45–54	45	8	26
	55–64	111	26	69
	65–74	296	109	201
	75–84	987	557	746
	85 and over	3,208	2,751	2,902
	Age-standardised rate	*126*	*74*	*98*
2007	**Socioeconomic group**			
	1st quintile (lowest)	145	84	112
	2nd quintile	131	77	102
	3rd quintile	129	72	98
	4th quintile	116	67	89
	5th quintile (highest)	101	60	78
2005–2007	**Aboriginal and Torres Strait Islander status**[(a)]			
	Indigenous	251	160	203
	Non-Indigenous	184	107	142
2005–2007	**Remoteness**			
	Major cities	125	72	96
	Inner regional	143	85	111
	Outer regional	152	89	119
	Remote and very remote	154	95	128

(a) Data from NSW, Qld, WA, SA and NT only.

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

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Table A5.1: Stroke hospitalisation rates, 2007–08

Population subgroup	Hospitalisations per 100,000 population		
	Males	Females	Persons
Age group (years)			
<25	6	4	5
25–34	18	15	16
35–44	37	38	38
45–54	102	73	87
55–64	239	133	186
65–74	582	355	466
75–84	1,330	1,044	1,170
85 and over	2,323	2,134	2,197
Age-standardised rate	*176*	*131*	*152*
Socioeconomic group			
1st quintile (lowest)	199	149	173
2nd quintile	193	144	167
3rd quintile	175	127	149
4th quintile	170	119	143
5th quintile (highest)	150	112	130
Aboriginal and Torres Strait Islander status[(a)]			
Indigenous	324	284	303
Other Australians	183	133	156
Remoteness			
Major cities	171	125	146
Inner regional	183	143	162
Outer regional	196	154	174
Remote and very remote	198	170	186

(a) Data from NSW, Vic, Qld, WA, SA and NT (public hospitals only).

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

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Table A5.2: Stroke death rates

Year	Population subgroup	Deaths per 100,000 population		
		Males	Females	Persons
2007	**Age group (years)**			
	<25	—	—	—
	25–34	1	1	1
	35–44	3	2	3
	45–54	9	6	7
	55–64	20	13	17
	65–74	64	53	58
	75–84	323	283	300
	85 and over	1,078	1,276	1,211
	Age-standardised rate	*37*	*36*	*37*
2007	**Socioeconomic group**			
	1st quintile (lowest)	40	38	39
	2nd quintile	39	38	38
	3rd quintile	36	35	36
	4th quintile	36	35	36
	5th quintile (highest)	33	32	33
2005–2007	**Aboriginal and Torres Strait Islander status**[(a)]			
	Indigenous	73	70	72
	Non-Indigenous	52	51	52
2005–2007	**Remoteness**			
	Major cities	36	34	35
	Inner regional	40	41	41
	Outer regional	41	41	41
	Remote and very remote	46	42	45

(a) Data from NSW, Qld, WA, SA and NT only.

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

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Table A6.1: Heart failure and cardiomyopathy hospitalisation rates, 2007–08

	Number per 100,000 population		
Population subgroup	**Males**	**Females**	**Persons**
Age group (years)			
<25	5	4	4
25–34	14	7	10
35–44	36	18	27
45–54	90	46	68
55–64	266	124	195
65–74	790	446	614
75–84	2,234	1,541	1,847
85 and over	4,648	3,748	4,048
Age-standardised rate	*262*	*172*	*213*
Socioeconomic group			
1st quintile (lowest)	317	215	262
2nd quintile	274	179	223
3rd quintile	262	171	212
4th quintile	244	153	194
5th quintile (highest)	219	135	171
Aboriginal and Torres Strait Islander status[(a)]			
Indigenous	686	553	613
Other Australians	271	175	218
Remoteness			
Major cities	256	165	205
Inner regional	261	172	213
Outer regional	305	218	261
Remote and very remote	374	302	340

(a) Data from NSW, Vic, Qld, WA, SA and NT (public hospitals only).

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Later edition available

Table A6.2: Heart failure and cardiomyopathy death rates

Year	Population subgroup	Deaths per 100,000 population		
		Males	Females	Persons
2007	**Age group (years)**			
	<25	—	—	—
	25–34	—	—	1
	35–44	2	1	2
	45–54	5	2	3
	55–64	13	5	9
	65–74	37	17	27
	75–84	155	99	123
	85 and over	601	615	611
	Age-standardised rate	*20*	*15*	*17*
2007	**Socioeconomic group**			
	1st quintile (lowest)	24	17	21
	2nd quintile	21	16	18
	3rd quintile	20	15	17
	4th quintile	18	12	15
	5th quintile (highest)	16	12	14
2005–2007	**Aboriginal and Torres Strait Islander status**[(a)]			
	Indigenous	42	25	32
	Non-Indigenous	26	19	22
2005–2007	**Remoteness**			
	Major cities	17	12	14
	Inner regional	21	16	19
	Outer regional	25	20	23
	Remote and very remote	26	18	22

(a) Data from NSW, Qld, WA, SA and NT only.

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

Later edition available

Table A7.1: Acute rheumatic fever or rheumatic heart disease hospitalisation rates, 2007–08

	Hospitalisations per 100,000 population		
Population subgroup	**Males**	**Females**	**Persons**
Age group (years)			
<25	4	5	4
25–34	3	4	4
35–44	4	6	5
45–54	8	10	9
55–64	18	19	19
65–74	38	49	44
75–84	63	69	66
85+	51	38	42
Age-standardised rate	*11*	*13*	*12*
Socioeconomic group			
1st quintile (lowest)	14	19	17
2nd quintile	11	12	12
3rd quintile	11	12	12
4th quintile	11	13	12
5th quintile (highest)	9	8	8
Aboriginal and Torres Strait Islander status[(a)]			
Indigenous	54	78	67
Other Australians	7	8	8
Remoteness			
Major cities	9	11	10
Inner regional	11	13	12
Outer regional	12	13	12
Remote and very remote	54	69	61

(a) Data from NSW, Vic, Qld, WA, SA and NT (public hospitals only).

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Later edition available

Table A7.2: Acute rheumatic fever or rheumatic heart disease death rates

Year	Population subgroup	Deaths per 100,000 population		
		Males	Females	Persons
2007	**Age group (years)**			
	<25	—	—	—
	25–34	—	—	—
	35–44	—	—	—
	45–54	—	—	—
	55–64	1	1	1
	65–74	2	3	3
	75–84	8	14	11
	85 and over	19	21	20
	Age-standardised rate	*1*	*1*	*1*
2007	**Socioeconomic group**			
	1st quintile (lowest)	1.3	1.5	1.4
	2nd quintile	1.0	1.3	1.2
	3rd quintile	0.9	1.4	1.2
	4th quintile	0.6	0.8	0.7
	5th quintile (highest)	0.6	1.2	0.9
2005–2007	**Aboriginal and Torres Strait Islander status**[(a)]			
	Indigenous	3.6	11.2	7.9
	Non-Indigenous	1.3	1.8	1.6
2005–2007	**Remoteness**			
	Major cities	0.7	1.2	1.0
	Inner and outer regional	1.4	1.6	1.5
	Remote and very remote	2.8	6.2	4.4

(a) Data from NSW, Qld, WA, SA and NT only.

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

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Table A8.1: Peripheral vascular disease hospitalisation rates, 2007–08

	Hospitalisations per 100,000 population		
Population subgroup	**Males**	**Females**	**Persons**
Age group (years)			
<25	2	2	2
25–34	8	6	7
35–44	19	17	18
45–54	56	31	43
55–64	231	84	157
65–74	667	245	452
75–84	1,286	658	936
85 and over	1,666	929	1,175
Age-standardised rate	*159*	*74*	*113*
Socioeconomic group			
1st quintile (lowest)	159	74	113
2nd quintile	172	80	122
3rd quintile	166	73	116
4th quintile	160	73	112
5th quintile (highest)	146	74	105
Aboriginal and Torres Strait Islander status[(a)]			
Indigenous	82	76	80
Other Australians	168	77	118
Remoteness			
Major cities	156	74	110
Inner regional	176	78	123
Outer regional	165	82	123
Remote and very remote	126	68	99

(a) Data from NSW, Vic, Qld, WA, SA and NT (public hospitals only).

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

Later edition available

Table A8.2: Peripheral vascular disease death rates

Year	Population subgroup	Deaths per 100,000 population		
		Males	Females	Persons
2007	**Age group (years)**			
	<25	—	—	—
	25–34	—	—	—
	35–44	—	—	—
	45–54	2	1	2
	55–64	7	3	5
	65–74	27	15	21
	75–84	107	60	81
	85 and over	318	234	262
	Age-standardised rate	*12*	*7*	*9*
2007	**Socioeconomic group**			
	1st quintile (lowest)	13	6	9
	2nd quintile	13	8	10
	3rd quintile	13	7	10
	4th quintile	12	7	9
	5th quintile (highest)	9	7	8
2005–2007	**Aboriginal and Torres Strait Islander status**[(a)]			
	Indigenous	10	9	10
	Non-Indigenous	18	11	14
2005–2007	**Remoteness**			
	Major cities	12	7	9
	Inner regional	15	9	11
	Outer regional	14	9	12
	Remote and very remote	15	7	11

(a) Data from NSW, Qld, WA, SA and NT only.

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

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Table A9.1: Congenital heart disease hospitalisation rates, 2007–08

Population subgroup	Hospitalisations per 100,000 population		
	Males	Females	Persons
Age group (years)			
<1	671	615	644
1–4	68	80	74
5–14	28	26	27
15–25	13	16	14
25–34	11	13	12
35–44	13	17	15
45–54	16	16	16
55–64	18	15	17
65–74	17	18	18
75–84	14	9	11
85+	8	7	7
Age-standardised rate	*28*	*28*	*28*
Socioeconomic group			
1st quintile (lowest)	30	29	29
2nd quintile	30	30	30
3rd quintile	31	29	30
4th quintile	28	31	29
5th quintile (highest)	28	31	29
Aboriginal and Torres Strait Islander status[(a)]			
Indigenous	24	29	27
Other Australians	30	30	30
Remoteness			
Major cities	30	30	30
Inner regional	30	32	31
Outer regional	26	28	27
Remote and very remote	24	27	26

(a) Data from NSW, Vic, Qld, WA, SA and NT (public hospitals only).

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Hospital Morbidity Database.

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Table A9.2: Congenital heart disease death rates

Year	Population subgroup	Deaths per 100,000 population		
		Males	Females	Persons
2007	**Age group (years)**			
	<1	28.8	29.0	28.9
	1–24	0.5	0.3	0.4
	25–34	0.7	0.2	0.5
	35–44	0.5	0.3	0.4
	45–54	0.3	0.4	0.4
	55–64	0.9	0.9	0.9
	65–74	0.7	0.3	0.5
	75–84	1.6	1.3	1.4
	85+	0.9	2.2	1.8
	Age-standardised rate	*1.0*	*0.8*	*0.9*
2007	**Socioeconomic group**			
	1st quintile (lowest)	1.3	0.8	1.1
	2nd quintile	0.9	1.0	0.9
	3rd quintile	1.1	0.8	0.9
	4th quintile	0.8	0.7	0.7
	5th quintile (highest)	0.8	0.7	0.8
2005–2007	**Aboriginal and Torres Strait Islander status[(a)]**			
	Indigenous	4.0	4.6	4.4
	Non-Indigenous	1.4	1.2	1.3
2005–2007	**Remoteness**			
	Major cities	0.9	0.7	0.8
	Inner regional	1.3	0.9	1.1
	Outer regional	0.9	0.9	0.9
	Remote and very remote	1.2	1.4	1.3

(a) Data from NSW, Qld, WA, SA and NT only.

Note: Age-standardised to the 2001 Australian population.

Source: AIHW National Mortality Database.

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Glossary

Acute coronary syndrome Describes an *acute myocardial infarction (AMI)* and *unstable angina* when they first present as clinical emergencies with chest pain or other features.

Acute myocardial infarction (AMI) Term still commonly used to mean a heart attack, but more correctly refers only to those heart attacks that have caused some death of heart muscle.

Age-specific rate A rate for a specific age group. The numerator and denominator relate to the same age group.

Age standardisation A method of removing the influence of age when comparing populations with different age structures. This is usually necessary because the rates of many diseases vary strongly (usually increasing) with age. The age structures of the different populations are converted to the same 'standard' structure, the disease rates that would have occurred with that structure are then calculated and compared.

Angina Temporary chest pain or discomfort when the heart's own blood supply is inadequate to meet extra needs, as can occur during exercise. See also *unstable angina* and *cardiovascular disease*.

Angioplasty A method of reducing a blockage in an artery by opening out a balloon placed inside the artery at the point of narrowing.

Associated cause(s) of death Any condition(s), diseases and injuries—other than the *underlying cause*—considered to contribute to a death. See also *cause of death*.

Atherosclerosis A process in which fatty and fibre-like deposits build up on the inner walls of arteries, often forming *plaques* that can then cause blockages. It is the main underlying condition in *heart attack, angina, stroke* and *peripheral vascular disease.*

Average length of stay (ALOS) The average of the length of stay for admitted patient episodes.

Blood cholesterol Fatty substance produced by the liver and carried by the blood to supply the rest of the body. Its natural function is to supply material for cell walls and for steroid hormones, but if levels in the blood become too high this can lead to *atherosclerosis* and heart disease.

Blood pressure The force exerted by the blood on the walls of the arteries as it is pumped around the body by the heart. It is written, for example, as 134/70 mmHg, where the upper number is the systolic pressure (the maximum force against the arteries as the heart muscle contracts to pump the blood out) and the lower number is the diastolic pressure (the minimum force against the arteries as the heart relaxes and fills again with blood). Levels of blood pressure can vary greatly from person to person and from moment to moment in the same person. See also *high blood pressure/hypertension*.

Body mass index (BMI) The most commonly used method of assessing whether a person is of normal weight, underweight, overweight or obese. It is calculated by dividing the person's weight (in kilograms) by their height (in metres) squared; that is, kg ÷ m^2. For both men and women, underweight is a BMI below 18.5, acceptable weight is from 18.5 to less than 25, overweight is 25 and above (includes obese), and obese is 30 and over.

Burden of disease and injury The term that refers to the quantified impact of a disease or injury on an individual or population, using the *disability-adjusted life year* (DALY) measure.

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Cancer A large range of diseases whose common feature is that some of the body's cells become defective, begin to multiply out of control, can invade and damage the area around them, and can also spread to other parts of the body to cause further damage.

Capital expenditure Expenditure on large-scale fixed assets (for example new buildings and equipment with a useful life extending over a number of years).

Cardiomyopathy A condition in which there is direct and widespread damage to the heart muscle, weakening it. The condition can be due to various causes such as viral infections and severe alcohol abuse, and it can lead to an enlarged, thickened and dilated heart as well as *heart failure*.

Cardiovascular disease (CVD) Any disease of the *circulatory system*, namely the heart (cardio) or blood vessels (vascular). Includes *heart attack, angina, stroke* and *peripheral vascular disease*. CVD is also known as *circulatory disease*.

Cause of death From information reported on the medical certificate of cause of death, each death is classified by the underlying cause of death according to rules and conventions of the 10th revision of the International Classification of Diseases. The underlying cause is defined as the disease that initiated the train of events leading directly to death. Deaths from injury or poisoning are classified according to the circumstances of the violence that produced the fatal injury, rather than to the nature of the injury. See also *underlying cause of death*.

Cerebrovascular disease Any disorder of the blood vessels supplying the brain or its covering membranes. A notable and major form of cerebrovascular disease is *stroke*.

Chronic diseases Term applied to a diverse group of diseases, such as heart disease, cancer and arthritis, that tend to be long-lasting and persistent in their symptoms or development. Although these features also apply to some *communicable diseases*, the term is usually confined to non-communicable diseases.

Circulatory disease Alternative name for *cardiovascular disease*.

Circulatory system The heart and the blood vessels, comprising the system that circulates blood around the body to supply oxygen and nutrients to all body tissues and to carry away waste products from them. Also known as the cardiovascular system.

Communicable diseases (infectious diseases) Diseases or illnesses due to infectious organisms or their toxic products. Communication may occur directly or indirectly through contact with other humans, animals or other environments that harbour the organism.

Comorbidity When a person has two or more health problems at the same time.

Confidence interval (CI) A statistical term describing a range (interval) of values within which we can be 'confident' that the true value lies, usually because it has a 95% or higher chance of doing so.

Congenital A condition that is recognised at birth, or that is believed to have been present since birth, including conditions that are inherited or caused by environmental factors.

Coronary artery bypass graft (CABG) Surgical procedure using blood vessel grafts to bypass blockages in the coronary arteries and restore adequate blood flow to the heart muscle.

Coronary artery disease Disease of the coronary arteries, typically meaning *atherosclerosis*. When this leads to symptoms such as chest pain the result is known as *coronary heart disease*.

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Coronary heart disease (CHD) Disease due to blockages in the heart's own (coronary) arteries, expressed as *angina* or a *heart attack*. Also known as *ischaemic heart disease*.

Crude death rate The number of deaths in a given period divided by the size of the corresponding population indexed to 100,000.

Depression A mood disorder with prolonged feelings of being sad, hopeless, low and inadequate, with a loss of interest or pleasure in activities and often with suicidal thoughts or self-blame.

Diabetes (diabetes mellitus) A chronic condition in which the body cannot properly use its main energy source, the sugar glucose. This is due to a relative or absolute deficiency in insulin, a hormone that is produced by the pancreas and helps glucose enter the body's cells from the bloodstream and then be processed by them. Diabetes is marked by an abnormal build-up of glucose in the blood, and it can have serious short- and long-term effects. For the three main types of diabetes see *Type 1 diabetes*, *Type 2 diabetes* and *gestational diabetes*.

Disability-adjusted life year (DALY) A year of healthy life lost, either through premature death or equivalently through living with disability due to illness or injury. It is the basic unit used in *burden of disease and injury* estimates.

Disease A physical or mental disturbance involving *symptoms* (such as pain or feeling unwell), dysfunction or tissue damage, especially if these *symptoms* and *signs* form a recognisable clinical pattern.

Fetal death Birth of a fetus weighing at least 400 grams (or, where birth weight is unavailable, of at least 20 weeks' gestation), which shows no signs of life. Commonly referred to as stillbirth.

Heart attack Life-threatening emergency that occurs when a vessel supplying blood to the heart muscle is suddenly blocked completely by a blood clot. The medical term commonly used for a heart attack is *acute myocardial infarction*.

Heart failure When the heart functions less effectively in pumping blood around the body. It can result from a wide variety of diseases and conditions that can impair or overload the heart, such as heart attack, other conditions that damage the heart muscle directly (see *cardiomyopathy*), *high blood pressure*, or a damaged heart valve.

High blood pressure/hypertension The definition of high blood pressure (also known as hypertension) can vary but a well-accepted one is from the World Health Organization: a systolic blood pressure of 140 mmHg or more or a diastolic blood pressure of 90 mmHg or more, or [the person is] receiving medication for high blood pressure. Also see *blood pressure*.

Highly specialised medicines Under Section 100 of the National Health Act, certain medicines can be supplied to community patients only through hospitals because only hospitals can provide the facilities and staff necessary for the appropriate use of the drugs. These drugs are funded by the Australian Government separately from the Pharmaceuticals Benefits Scheme.

Hypertension See *high blood pressure*.

Incidence The number of new cases (of an illness or event, and so on) occurring during a given period. Compare with *prevalence*.

International Classification of Diseases The World Health Organization's internationally accepted classification of death and disease. The 10th Revision (ICD-10) is currently in use. In this report, causes of death classified before 1979 under previous revisions have been reclassified to ICD-10 by the AIHW. ICD-10-AM is the Australian modification of ICD-10, used for diagnoses and procedures recorded for patients admitted to hospitals.

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

Length of stay Duration of hospital stay, calculated by subtracting the date the patient is admitted from the day of separation. All leave days, including the day the patient went on leave, are excluded. A same-day patient is allocated a length of stay of 1 day.

Low birth weight The weight of a baby at birth that is less than 2,500 grams.

Medicare A national, government-funded scheme that subsidises the cost of personal medical services for all Australians and aims to help them afford medical care.

Mental illness Disturbances of mood or thought that can affect behaviour and distress the person or those around them, so the person has trouble functioning normally. They include anxiety disorders, *depression* and schizophrenia.

Morbidity Refers to ill health in an individual and to levels of ill health in a population or group.

Obesity Marked degree of overweight, defined for population studies as a *body mass index* of 30 or over. See also *overweight*.

Organisation for Economic Co-operation and Development (OECD) An organisation of 30 developed countries, including Australia.

Other Australians People who are not of Aboriginal or Torres Strait Islander descent, or whose status is not known.

Out-of-pocket costs The total costs incurred by individuals for health-care services over and above any refunds from Medicare and private health insurance funds.

Overweight Defined for the purpose of population studies as a *body mass index* of 25 or over. See also *obesity*.

Patient days The number of full or partial days of stay for patients who were admitted for an episode of care and who underwent separation during the reporting period. A patient who is admitted and separated on the same day is allocated one patient day.

Perinatal Pertaining to or occurring in the period shortly before or after birth (usually up to 28 days after).

Peripheral vascular disease Characterised by pain in the extremities, often the legs, due to an inadequate blood supply to them.

Pharmaceutical Benefits Scheme (PBS) A national, government-funded scheme that subsidises the cost of a wide range of pharmaceutical drugs, and that covers all Australians to help them afford standard medications.

Plaque (atherosclerotic) A localised area of *atherosclerosis*, especially when raised or built up, and that may cause blockages in arteries.

Potential years of life lost (PYLL) Number of potential years of life lost in a population as a result of premature death.

Prevalence The number or proportion (of cases, instances, and so forth) present in a population at a given time. Compare with *incidence*.

Principal diagnosis The diagnosis listed in hospital records to describe the problem that was chiefly responsible for the patient's episode of care in hospital.

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Private hospital A privately owned and operated institution, catering for patients who are treated by a doctor of their own choice. Patients are charged fees for accommodation and other services provided by the hospital and relevant medical and allied health practitioners.

Public hospital A hospital controlled by a state or territory health authority. In Australia public hospitals offer free diagnostic services, treatment, care and accommodation to all Australians who need them.

Quintile A group derived by ranking the population of people or elements according to specified criteria and dividing it into five equal parts. The term can also mean the cut-points that make these divisions—that is, the 20th, 50th and 75th percentiles—but the first use is the more common one.

Repatriation Pharmaceutical Benefits Scheme (RPBS) provides pharmaceuticals and dressings at a concession rate for the treatment of eligible veterans, war widows/widowers and their dependants.

Recurrent expenditure Expenditure on goods and services that are used up during the year—for example, salaries. It may be contrasted with *capital expenditure*.

Revascularisation ('re-vesselling') Restoring adequate blood flow to the heart or other part of the body, usually after the supply has been reduced or blocked, as in angina or a *heart attack*. Revascularisation includes methods such as *angioplasty* and *coronary artery bypass graft* surgery.

Rheumatic fever An acute, serious disease that affects mainly children and young adults and can damage the heart valves, the heart muscle and its lining, the joints and the brain. Is brought on by a reaction to a throat infection by a particular bacterium. Now very rare in the non-Indigenous population, it is still at unacceptably high levels among Indigenous Australians living in remote areas. See *rheumatic heart disease*.

Rheumatic heart disease (RHD) Chronic disease from damaged heart valves caused by earlier attack(s) of *rheumatic fever*.

Risk factor Any factor which represents a greater risk of a health disorder or other unwanted condition or event. Some risk factors are regarded as causes of disease, others are not necessarily so. Along with their opposites, protective factors, risk factors are known as *determinants*.

Same-day patients Admitted patients who are admitted to hospital and separated on the same day.

Section 100 medicines See *highly specialised medicines*.

Separation The formal process by which a hospital records the completion of an episode of treatment and/or care for an admitted patient.

Statistical significance An indication from a statistical test that an observed difference or association may be significant or 'real' because it is unlikely to be due just to chance. A statistical result is usually said to be 'significant' if it would occur by chance only once in 20 times or less often.

Stent A metal mesh tube that is expanded within an artery at a point of narrowing and left there to hold the artery open.

Stillbirth See *fetal death*.

Stress Poorly defined term referring to when a person is under significant psychological or physical pressure—real or perceived, acute or chronic. Among the many examples are illness or injury, bereavement, family problems, work demands or job loss.

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Stroke When an artery supplying blood to the brain suddenly becomes blocked or bleeds. Often causes paralysis of parts of the body normally controlled by that area of the brain, or speech problems and other symptoms.

Thrombolysis Emergency 'clot-busting' drug treatment for a *heart attack*.

Thrombosis Clotting of blood, with the term usually applied to clotting within a blood vessel due to disease, as in a *heart attack* or *stroke*.

Transient ischaemic attack (TIA) A 'mini' *stroke*, with temporary problems in speech or paralysis that last for 24 hours or less, often only minutes. It is a strong warning sign of a more severe stroke.

Type 1 diabetes A form of *diabetes* mostly arising among children or younger adults, marked by a complete lack of insulin and needing insulin replacement for survival.

Type 2 diabetes The most common form of *diabetes*, occurring mostly in people aged 40 years or over, and marked by reduced or less effective insulin.

Underlying cause of death The condition, disease or injury initiating the sequence of events leading directly to death; that is, the primary or main cause. Compare with *associated cause(s) of death*.

Underweight Defined for population studies as a *body mass index* less than 18.5.

Unstable angina A form of *angina* that is more dangerous than normal angina but less so than a *heart attack*. It can feature chest pain that occurs at rest; and in someone who already has angina it can be marked by new patterns of onset with exertion or by pain that comes on more easily, more often or for longer than previously.

Ventricular septal defect A congenital defect of the heart that occurs as an opening in the wall that separates the left and right main pumping chambers (the ventricles).

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