



The burden of disease and injury in Australia 2003

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The burden of disease and injury in Australia 2003

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The burden of disease and injury in Australia 2003

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May 2007

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Foreword

Exactly a decade ago, the results of the first Global Burden of Disease (GBD) Study were published by Harvard University on behalf of the World Health Organization and the World Bank. These organisations and several countries then became interested in applying the GBD approach to better inform health policy, leading to a series of country studies on all continents. Probably the most technically competent and comprehensive of these were the Australian studies, led by Colin Mathers, for Australia as a whole, and Theo Vos for the state of Victoria. These analyses were based around 1996 data and have been widely used to inform priority setting and health policy debates in Australia.

As a result of these initial studies, governments across Australia have become interested in using the burden of disease framework to help quantify health needs. There have also been advances in methods over the past ten years and greater interest among the health policy community in information about the burden of disease in population subgroups. This has all stimulated the need for a revised Australian burden of disease and injury study to update and extend the initial efforts.

This report responds to that need. Some of the world's leading researchers in burden of disease studies, with extensive experience in national applications of the methods, have joined the University of Queensland to create the great focus of expertise reflected in this study. Building on the analytical framework of previous studies, the report includes a number of important extensions of the framework that are highly relevant for policy. They include disease projections, small area analyses and state-level burden of disease results. Also, better methods around comorbidity and risk factor assessment have much improved the scientific basis of the findings reported here.

This comprehensive study will undoubtedly meet the need for detailed information about the burden of disease and injury in Australia and its jurisdictions, about the principal causes of that burden, and how it is changing. But it alone is not enough. With rising pressure on health budgets, governments will increasingly rely not only on information about the burden of disease and injury, but also on cost-effective ways of reducing that burden. This study is a critical and fundamental step in that policy process and we expect it to be used widely to help improve the health of all Australians.

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Executive summary

Introduction

This report is the first complete assessment of the health of Australians to be released in the new millennium.

The findings in this report identify the extent and distribution of health problems in Australia, and quantify the contribution of key health risk factors to these problems.

Levels of death and disability from a comprehensive set of diseases, injuries and risks to health are combined to measure the total health 'burden'.

This report is the second of this type in Australia, the first having been released in 1999. It expands the scope of that previous report and also presents for the first time:

- the differentials of health burden across areas and population groups in Australia
- the joint contribution of key health risks – including combined lifestyle, physiological, social and environmental factors – on health
- an analysis of past trends of health burden and the likely health of Australians in 20 years from now should those trends continue.

The findings of this report describe the health loss due to disease and injury that is not ameliorated by current treatment, rehabilitative and preventive efforts of the health system and society generally. Thus they represent the 'unmet' challenges of the health system and are best interpreted as opportunities for health gain.

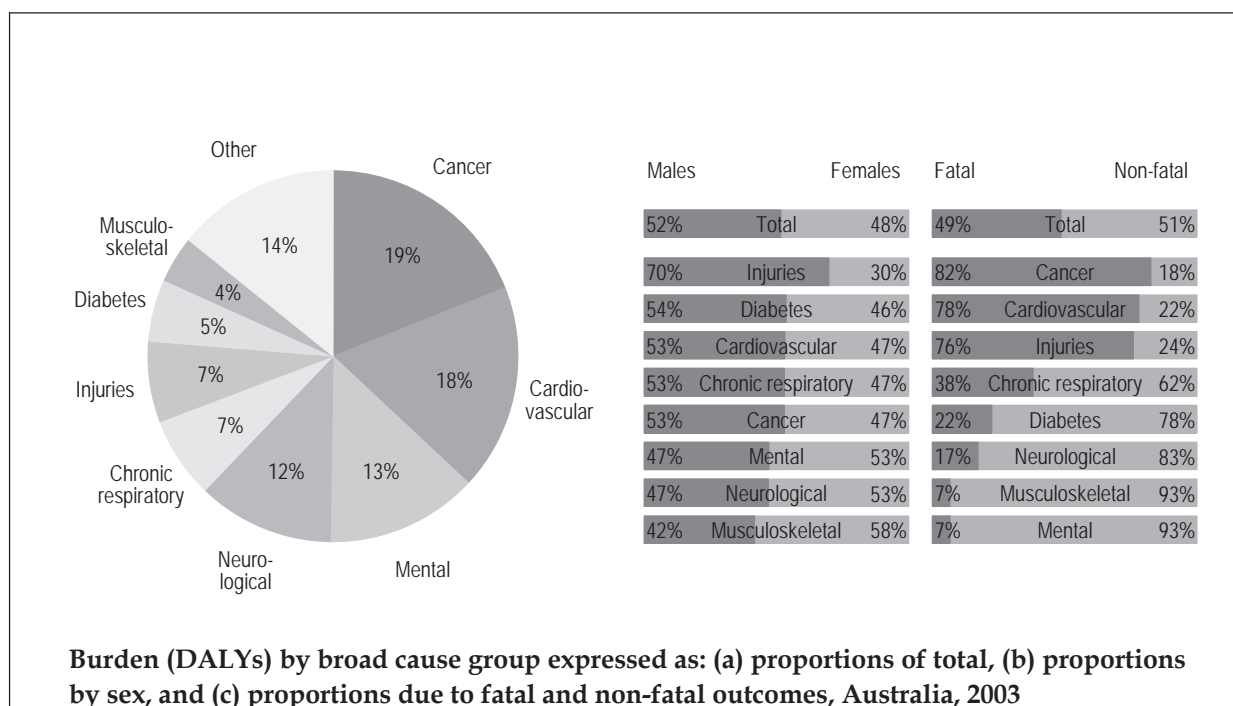
By providing a comprehensive database of all relevant epidemiological and burden parameters through time, the report will benefit health policy development and research in relation to preventive and curative health interventions, health care expenditure projections, and further assessments of health burden in the period before the next major update.

The study upon which the report is based was funded by the Australian Government Department of Health and Ageing. A report specifically examining the burden of disease and injury in Aboriginal and Torres Strait Islander people will be published separately.

Key findings

Total burden of disease and injury

The key measure used in this report to measure the total burden of disease and injury is the 'disability-adjusted life year' (DALY). It describes the amount of time lost due to both fatal and non-fatal events, that is, years of life lost due to premature death coupled with years of 'healthy' life lost due to disability.



- In 2003, more than 2.63 million years of 'healthy' life (that is, DALYs) were lost due to the burden of disease and injury in Australia.
- Cancers (19%) and cardiovascular disease (18%) were the leading causes of the burden of disease and injury in Australia in 2003, accounting for 37% of the total burden. Four-fifths of that burden was from premature deaths. For the first time, cancer has overtaken cardiovascular disease as the greatest cause of burden in Australia.
- Lung, colorectal and breast cancer were the leading specific causes of the burden of cancer.
- Ischaemic heart disease, stroke, and peripheral vascular disease were the leading specific causes of cardiovascular burden.
- Mental disorders and neurological & sense disorders were the next largest contributors, together accounting for a further 25% of the total health burden. Less than one-fifth of that burden was from premature deaths.
- Anxiety & depression, alcohol abuse, and personality disorders dominated the burden of mental disorders.

- Dementia, adult-onset hearing loss, and vision loss were the leading causes of burden due to neurological & sense disorders.
- Anxiety & depression also carries a risk of ischaemic heart disease and suicide, increasing the total burden due to the combined category of anxiety & depression from 7.3% to 8.2%.
- Diabetes also carries a risk of ischaemic heart disease and stroke, increasing the total burden of diabetes from 5.5% to 8.3%, and making it the fourth largest contributor to overall burden after cancer, CVD and mental disorders.
- The eight national health priority conditions – asthma, cancer, cardiovascular disease, diabetes mellitus, injuries, mental health, arthritis and musculoskeletal conditions, and dementia – accounted for 72.8% of the total burden in 2003.
- Distribution of the burden between the sexes was roughly equal except for injuries (70% of the burden in males) and musculoskeletal (58% of the burden in females).
- The five leading specific causes of burden in men were ischaemic heart disease (11.1%), Type 2 diabetes (5.2%), anxiety & depression (4.8%), lung cancer (4.0%) and stroke (3.9%).
- The five leading specific causes of burden in women were anxiety & depression (10.0%), ischaemic heart disease (8.9%), stroke (5.1%), Type 2 diabetes (4.9%) and dementia (4.8%).
- Disability from all diseases and injuries resulted in a loss of 1.5% of healthy time lived by children, increasing with age to 14.7% in those aged 65 to 69 years, to 41.5% in the very aged.

Fatal burden

- ‘Life expectancy’ estimates the average years of life that a person can expect to live given current risks of mortality. In 2003 in Australia, life expectancy at birth was 80.7 years (78.3 years for males and 83.2 years for females).
- Fatal burden – measured in years of life lost (YLL) – accounted for 49% of the total burden of disease and injury in Australia in 2003.
- Cancers (32.0%), cardiovascular disease (29.0%) and injuries (11.0%) were responsible for almost three-quarters of the fatal burden.
- Males experienced 55% of total fatal burden. The five leading specific causes of mortality burden among men were ischaemic heart disease (18.2%), lung cancer (7.3%), suicide & self-inflicted injury (5.4%), stroke (5.1%) and colorectal cancer (3.9%).
- Females experienced 45% of total fatal burden. The five leading specific causes of mortality burden among women were ischaemic heart disease (15.7%), stroke (8.5%), breast cancer (7.0%), lung cancer (5.5%) and colorectal cancer (4.2%).

Non-fatal burden

- ‘Health adjusted life expectancy’ (HALE) estimates the average years of equivalent ‘healthy life’ that a person can expect to live. In 2003 in Australia, the average HALE was 72.9 years (70.6 years for males and 75.2 years for females), with 9.7% of life expectancy at birth lost due to disability.
- Non-fatal burden – measured in years of ‘healthy’ life lost due to disability (YLD) – accounted for 51% of the total burden of disease and injury in Australia in 2003.

- Mental disorders (24%) and neurological & sense disorders (19%) contributed most to non-fatal burden.
- The five leading specific causes of non-fatal burden among men were anxiety & depression (10.0%), Type 2 diabetes (8.5%), adult-onset hearing loss (6.5%), asthma (4.2%) and dementia (3.9%).
- The five leading specific causes of non-fatal burden among women were anxiety & depression (18.1%), Type 2 diabetes (7.2%), dementia (6.4%), asthma (4.5%) and ischaemic heart disease (3.3%).

Age patterns and total burden

Distribution of population and burden (DALYs) by five broad age groups, Australia, 2003

Age group	Population ^(a)	Per cent of total	DALYs	Per cent of total
0–14 years	3,979,410	20.0	221,536	8.4
15–44 years	8,622,610	43.4	633,260	24.1
45–64 years	4,733,808	23.8	681,566	25.9
65–74 years	1,349,949	6.8	428,904	16.3
75 years and over	1,195,692	6.0	667,504	25.4
Total	19,881,469	100.0	2,632,770	100.0

(a) Estimated resident population figures as at 30 June 2003 (ABS cat. no. 3201.0).

- Adults aged 45 to 64 years comprised 23.8% of the population in 2003 and experienced the largest proportion (25.9%) of disease and injury burden across key age groups. Cancer (28%), cardiovascular disease (16%) and neurological disorders (10%) accounted for more than half the total burden in this age group. Almost half of the burden was due to mortality.
- Adults aged over 75 years comprised 6.0% of the population but experienced the second highest proportion of burden (25.4%). Cardiovascular disease (34%) and cancer (19%) accounted for more than half of the burden. Overall, 68% of the burden was due to mortality.
- Adults aged 15 to 44 years represented the largest age group (43.4% of the population) and experienced 24.1% of the burden. Mental disorders (36%) and injuries (17%) accounted for more than half of the total burden in this age group. Mortality contributed 29% to the burden in this age group.
- Children aged 0-14 years comprised 20.0% of the population and experienced 8.4% of the total burden of disease and injury in Australia in 2003. Twenty-three per cent of this burden was due to mental disorders, 18% to chronic respiratory disorders, and 16% to neonatal conditions. About one-quarter of the burden was due to mortality.

Health risks

Individual and joint burden (DALYs) attributable to 14 selected risk factors by broad cause group, Australia, 2003

	Broad cause group							All causes
	Cancer	CVD	Mental	Neuro-logical	Injury	Diabetes	Other	
Total burden ('000)	499.4	473.8	350.5	312.8	185.1	143.8	667.4	2,632.8
Attributable burden (%) ^(a)								
Tobacco	20.1	9.7	—	-0.6	0.5	—	8.9	7.8
High blood pressure	—	42.1	—	—	—	—	—	7.6
High body mass	3.9	19.5	—	—	—	54.7	1.1	7.5
Physical inactivity	5.6	23.7	—	—	—	23.7	>-0.1	6.6
High blood cholesterol	—	34.5	—	—	—	—	—	6.2
Alcohol								
Harmful effects	3.1	0.9	9.7	—	18.1	—	<0.1	3.3
Beneficial effects	—	-5.6	—	—	—	—	>-0.1	-1.0
Net effects	3.1	-4.7	9.7	—	18.1	—	<0.1	2.3
Low fruit & vegetable consumption	2.0	9.6	—	—	—	—	>-0.1	2.1
Illicit drugs	—	<0.1	8.0	—	3.6	—	2.5	2.0
Occupational exposures & hazards	3.1	0.4	—	0.8	4.7	—	3.4	2.0
Intimate partner violence	0.5	0.3	5.5	0.1	2.5	—	0.2	1.1
Child sexual abuse	<0.1	<0.1	5.8	—	1.4	—	<0.1	0.9
Urban air pollution	0.8	2.7	—	—	—	—	0.4	0.7
Unsafe sex	1.0	—	—	—	—	—	1.4	0.6
Osteoporosis	—	—	—	—	2.4	—	—	0.2
Joint effect ^(b)	32.9	69.3	26.9	0.2	31.7	60.1	17.2	32.2

(a) Attributable burden within each column is expressed as a percentage of total burden for that column.

(b) Figures for joint effects are not column totals. See Section 4.1 for further details.

Findings on the amount of burden in 2003 that was attributable to current and past exposures to risks to health considered the following:

- Lifestyle behaviours (tobacco smoking, physical inactivity, alcohol consumption, low fruit and vegetable consumption, use of illicit drugs, and unsafe sex)
- Physiological states (high body mass, high blood pressure, high cholesterol, and osteoporosis)
- Social and environmental factors (occupational exposures and hazards, intimate partner violence, child sexual abuse, and urban air pollution).

The 14 risks together explained 32.2% of the total burden of disease and injury in Australia in 2003.

- Tobacco was responsible for the greatest disease burden in Australia (7.8% of total burden), followed by high blood pressure (7.6%), high body mass (7.5%), physical inactivity (6.6%), and high blood cholesterol (6.2%).

- The five leading risks in males in 2003 were tobacco (9.6%), high blood pressure (7.8%), high body mass (7.7%), high blood cholesterol (6.6%) and physical inactivity (6.4%).
- Among women the leading risks were high blood pressure (7.3%), high body mass (7.3%), physical inactivity (6.8%), high blood cholesterol (5.8%) and tobacco (5.8%).

This report sets out for the first time the combined or 'joint' effect of these risks on health, accounting for the fact that many risks share complex causal pathways. It is difficult to quantify the exact contribution of each risk to the combined totals, but the proportion of total burden that is 'explained' by multiple risks within each disease and injury category can be reported with sufficient accuracy.

- Ten risks were associated with cancer and together explained 32.9% of the cancer burden. The majority was explained by tobacco but also included the effect of physical inactivity, high body mass, and alcohol consumption.
- Twelve risks were associated with cardiovascular disease and together explained 69.3% of the disease burden; for ischaemic heart disease this figure was 85.2%. High blood pressure and high blood cholesterol were the largest contributors.
- Three risks were associated with neurological and sensory disorders and together explained 0.2% of the burden from these disorders. This reflects a lack of knowledge about causation in this group.
- Two risks were associated with Type 2 diabetes and together explained 60.1% of the total burden. High body mass was by far the largest contributor (54.7%) followed by physical inactivity (23.7%).
- The burden associated with harmful alcohol consumption (3.2%) was partially offset by the cardiovascular disease prevented by safe levels of alcohol consumption (-0.9%). This protective factor only becomes apparent after 45 years of age, whereas the harmful effects of alcohol are apparent at all ages.

Differentials in burden across Australia

- This report shows for the first time that there are differentials across Australia in the proportion of life expectancy lost due to disability. There was a strong socioeconomic gradient in this measure, and differentials with respect to remoteness were also apparent but not as large.
- Health-adjusted life expectancy (HALE) in 2003 in Australia was 72.9 years (70.6 for males and 75.2 for females), with an average 9.7% of life expectancy at birth lost due to disability.
- Across states and territories, the proportion of life expectancy lost due to disability ranged from 7.7% in the ACT to 10.6% in South Australia. The NT had almost twice the rate of total burden of the ACT due to a higher rate of burden for most causes, but particularly cardiovascular disease, diabetes, and injury.
- Across socioeconomic quintiles, the proportion of life expectancy lost due to disability ranged from 8.7% in the highest quintile to 10.6% in the lowest. The 31.7% greater burden for the most disadvantaged population compared to the highest was due to higher rates of burden for most causes, but particularly mental disorders and cardiovascular disease.

Health-adjusted life expectancy (HALE) and life expectancy at birth lost due to disability by area and sex, Australia, 2003

Area	Health-adjusted life expectancy (HALE) (years)						Life expectancy at birth lost due to disability (%)		
	At birth			At age 60			Males	Females	Persons
	Males	Females	Persons	Males	Females	Persons			
Jurisdiction									
NSW	70.5	75.3	72.9	17.1	20.6	18.9	9.8	9.5	9.6
Vic	71.1	75.4	73.2	17.5	20.8	19.2	9.6	9.4	9.5
Qld	70.5	75.3	72.8	17.0	20.4	18.7	10.1	9.7	9.9
WA	71.5	75.6	73.5	17.5	20.6	19.1	9.6	9.6	9.6
SA	69.3	74.2	71.7	16.4	20.0	18.3	10.8	10.5	10.6
Tas	68.8	73.7	71.3	16.3	19.7	18.1	10.2	9.8	10.0
NT	65.8	70.2	67.7	12.6	15.1	13.6	10.0	10.6	10.3
ACT	73.9	77.8	75.9	18.9	21.9	20.5	7.8	7.5	7.7
Socioeconomic quintile									
Low	68.7	73.8	71.2	16.1	19.7	17.9	10.7	10.4	10.6
Moderately low	69.5	74.6	72.0	16.4	20.1	18.2	10.2	9.9	10.1
Average	69.9	74.6	72.2	16.6	20.1	18.4	10.0	9.8	9.9
Moderately high	71.4	75.9	73.6	17.6	20.8	19.3	9.7	9.1	9.4
High	73.8	77.2	75.5	19.2	21.9	20.6	8.7	8.7	8.7
Remoteness									
Major cities	71.3	75.6	73.5	17.5	20.8	19.2	9.6	9.4	9.5
Regional	69.6	74.5	72.0	16.5	20.1	18.3	10.3	9.8	10.1
Remote	67.3	72.3	69.5	15.4	18.5	16.8	10.8	11.3	11.0
Australia	70.6	75.2	72.9	17.1	20.5	18.9	9.8	9.6	9.7

- Based on remoteness, the proportion of life expectancy lost due to disability ranged from 9.5% in major cities to 11.0% in remote areas. The 26.5% greater burden for remote areas compared to major cities reflected a higher burden per person from most causes but particularly injuries.

Trends—past, present and future

This report presents an analysis of health trends over a 30-year period, based on the past decade and projected trends of health burden if these trends continued over the next 20 years.

- The average years of 'healthy life' a person can expect to live (HALE) will grow at a slower rate than life expectancy over the next 20 years. If past trends in morbidity and mortality continue, HALE will increase 0.22% annually and life expectancy 0.24% annually. This is partly because declines in mortality will be accompanied by a somewhat smaller decline in time that is lost to disability.

- The rate of disability will actually decline in most age groups, except for those 80 years and over, where it is expected to increase and thereby offset some of the gains for younger age groups. The growing rate of disability in the oldest age group mostly comes from expected increases in diabetes and neurological conditions.

Key implications

Key implications of the report's findings include:

- Ageing of Australia's population will result in increasing numbers of people with disability from diseases more common in older ages such as dementia, Parkinson's disease, hearing and vision loss, and osteoarthritis. This will increase demand for services in the home, community care, residential aged care and palliative care sectors.
- Cardiovascular disease has been overtaken by cancer as the major cause of burden in Australia. This has been largely as a result of programs which have reduced smoking and facilitated the use of therapies to lower cholesterol and blood pressure levels, as well as better treatment of existing cardiovascular disease. It is likely that additional gains could be made through increasing the coverage of these interventions.
- Cancer is expected to retain its share of total health burden. Age-standardised rates of death and disability are expected to fall, but cancer will remain the largest contributor to the health burden in 20 years time.
- There is likely to be strong growth in the burden of diabetes over the next 20 years, mostly as a direct consequence of increasing levels of obesity. The disability consequences of increasing obesity will be magnified as fatality rates for people with diabetes continue to decline. This increased survival will mean an increase in the risk of people developing other largely non-fatal but disabling consequences of diabetes such as renal failure and vision loss.
- Australia is likely to benefit from further efforts towards expanding the range of effective prevention and treatment strategies for all causes of burden, while recognising that the returns for these efforts can take time to be realised. Only in recent years, for example, have smoking-related cancers started to decline as the result of several decades of successful tobacco control programs.

1 Introduction

1.1 Purpose

The study upon which this report is based is the first complete assessment of the health of Australians in the new millennium and the second study in this country with comparable objectives. The original study, the results of which continue to be used widely in policy and research environments, was conducted by the Australian Institute of Health and Welfare (AIHW: Mathers et al. 1999) and provided a comprehensive overview of disease and injury burden for the year 1996. Increasing demand for a contemporary picture of health status in Australia led, in 2003, to an Australian Government-funded collaboration between the University of Queensland and the Australian Institute of Health and Welfare (AIHW), the aim of which was to update and expand the original work.

The objectives of this collaboration were to report on the following:

- full burden of disease and injury results for the year 2003 by age group, sex and cause
- projections of disease and injury burden 20 years into the future
- improved models for attributing disease and injury burdens to risk factors
- subnational estimates of burden for state and territory jurisdictions, socioeconomic quintiles, remoteness categories and small areas
- the burden of disease and injury in Aboriginal and Torres Strait Islander populations.

This report presents the main findings of this collaboration and meets the above objectives, except the last which is covered in a separate report.

1.2 Background

Changes in demography and technology are placing increasing pressure on the health budgets of developed countries around the world. Mortality and fertility rates have decreased consistently over recent decades, resulting in increases in life expectancy and the proportion of total population alive at old and very old ages (AIHW 2006). In addition, developments in knowledge and medical technology are contributing to a growing demand for health services and, in many cases, to higher costs of providing these services. In Australia and elsewhere, these factors have brought into focus the need for more rigorous debate about how health systems can achieve their dual objectives of maximising health gains for given levels of expenditure and maintaining fair and equitable access to health services.

Improving the evidence base that informs this debate is critical if health systems are to be meaningfully held to account. Such an agenda requires contributions from a number of areas, including:

- detailed assessments of the size and impact of health problems in a population, including information on the causes of loss of health in the population (in terms of both diseases and injury, and risk factors or broader determinants)

- information on inequalities in health status, health determinants, and access to and use of health services (including prevention and treatment services)
- information on health expenditure and health infrastructure (a national system of health accounts) detailing the availability of resources for health improvement and the current use of these resources
- information on the cost-effectiveness of available technologies and strategies for improving health
- information on current levels of investment in health research and development, and on the opportunities for investment with the greatest likelihood of developing new or improved interventions that best remedy major health problems.

This report contributes to the development of such an agenda in Australia by providing a detailed and internally consistent assessment of the incidence, prevalence, duration, mortality and burden for an exhaustive and mutually exclusive set of major diseases and injuries experienced in this country. The burden from these causes is quantified for various subpopulations, risks to health and points in time using a summary measure of population health that combines both fatal and non-fatal health outcomes, and includes comorbidity adjustments to account for individuals who simultaneously experience multiple conditions.

This assessment provides an unprecedented level of detail on the magnitude and distribution of health problems in contemporary Australia. Although solutions to these problems are not addressed explicitly in the following chapters, the analyses described encompass a methodology that is increasingly being used in Australia and elsewhere to assess health outcomes both for descriptive purposes and in comparative analyses of the costs and effectiveness of particular health interventions. The report can be regarded, therefore, as an important foundation for further work on improving health system performance in Australia.

1.3 Summary measures of population health

Summary measures of population health are measures that combine information on mortality and non-fatal health outcomes into a single number to represent one or more dimensions of health at a population level (Field & Gold 1998). In the past 15 years, there has been a marked increase in interest in the development, calculation and use of summary measures. The range of potential applications includes:

- comparing health conditions or overall health status between two populations or the same population over time
- quantifying health inequalities
- ensuring that non-fatal health outcomes receive appropriate policy attention
- measuring the magnitude of different health problems using a common currency
- analysing the benefits of health interventions for use in cost-effectiveness studies
- providing information to help set priorities for health planning, public health programs, research and development, and professional training (Murray et al. 1999b).

Most summary measures fall into one of two broad groups: health 'expectancies' and health 'gaps'. Both groups use time (either lived in health states or lost through premature death and illness) as the unifying 'currency' for combining the impact of mortality and non-fatal health outcomes. Another common feature is the requirement for explicit or implicit choices

in their application: mortality-based indicators, for example, exclude considerations regarding non-fatal loss of health; indicators of potential years of life lost ignore deaths beyond an arbitrary age (for example 65 years); and indicators of disability-free life expectancy do not place any positive value on years lived with disability.

Health 'gap' measures, in particular, quantify the gap between a population's actual health status and some 'ideal' or reference status. The most widely known example of such a measure, and the one used in this report, is the disability-adjusted life year or DALY. Another measure commonly used in economic evaluations but not in population health status assessments is the Quality Adjusted Life Year (QALY).

1.4 Disability-Adjusted Life Years

The DALY was first developed to provide information to support health policy and priority setting at a global level. The concept was developed as part of a comprehensive assessment of global health for the year 1990 in what became known as the Global Burden of Disease or GBD study (Murray & Lopez 1996a, 1996b; World Bank 1993). It has since become synonymous with 'burden of disease' and the terms tend to be used interchangeably.

The DALY was originally intended to:

- allow estimates of health effects to be mapped to causes, either in terms of disease and injury, or risk factors and broader social determinants
- provide a common measure for estimating population health effects and cost-effectiveness of interventions
- use common values and health standards for all regions of the world
- provide a common measure for fatal and non-fatal health outcomes.

In this way, the DALY extends the concept of potential years of life lost due to premature death (PYLL) by including equivalent years of 'healthy' life lost by virtue of being in states of poor health or disability. A DALY for a disease or health condition is calculated as the sum of the years of life lost due to premature mortality (YLL) in the population and the equivalent 'healthy' years lost due to disability (YLD) for incident cases of the health condition:

$$\text{DALY} = \text{YLL} + \text{YLD}$$

where YLL = number of deaths x standard life expectancy at age of death and

YLD = incidence x duration x severity weight.

The loss of healthy life due to health conditions (YLD) requires estimation of the incidence of the disabling health condition (disease or injury) in the specified time period. For each new case, the number of years of healthy life lost is obtained by multiplying the average duration of the condition (to remission or death) by a severity weight that quantifies the equivalent loss of healthy years of life due to living with the health condition or its sequelae. The YLD is as an incidence-based measure, therefore, which captures the future health consequences of new cases of disease and injury that occur in the baseline year (2003 in this study). Such a measure, when combined with YLL, enables the full 'health loss' of different diseases and injuries to be compared and has most application in planning.

Alternatively, health loss can be measured by counting it at the age it is lived. This is the 'prevalent burden' or prevalent years lost due to disability (PYLD) and is calculated thus:

$$\text{PYLD} = \text{prevalence} \times \text{severity weight}$$

Prevalent burden is useful from a service utilisation or expenditure perspective and measures the amount of disability (but not the fatal burden) being experienced in a population at a point in time.

From the perspective of the International Classification of Functioning, Disability and Health (ICF) (see <www3.who.int/icf/icftemplate.cfm>) the YLD measures the impact of a health condition on an individual's functioning, now and into the future. Functioning includes the functional and structural integrity of the human body as well as activities undertaken by people and participation in life situations.

Interpreting the DALY

The DALY methodology provides a way to link information on disease causes and occurrence to information on both short-term and long-term health outcomes, including activity limitations and restrictions in participation in usual roles, and death. The burden of disease methodology is designed to inform health policy about the prevention and treatment (cure or reduction in severity) of adverse health outcomes. It is not designed to inform policy for the provision of social support or welfare services for people with long-term disability.

When using the DALY for the first time, Murray and Lopez sought to make explicit the value choices that they had to make in their application of a summary measure at a global level. For example, they chose to use the same life expectancy 'ideal' standard for all population subgroups across the globe, whether or not their current life expectancy was lower than that of other groups. They also excluded all non-health characteristics (such as race, socioeconomic status or occupation), apart from age and sex, from consideration in calculating lost years of healthy life. Most importantly, they used the same severity weight for everyone living a year in a specified health state. These and other aspects of the DALY are described in further detail in Chapter 2.

1.5 Burden of disease analysis in Australia

Since its introduction, burden of disease analysis has been applied in an increasing number of international and national settings; for example, it was used for a period by the World Health Organization (WHO) to inform global health planning (WHO 2002). Burden of disease analysis has a particularly strong history in Australia. The first study by the AIHW assessed the burden of disease and injury in Australia for the year 1996 (AIHW: Mathers et al. 1999). Starting in June 1998, the first study was partly funded by the then Commonwealth Department of Health and Aged Care and was conducted in parallel with a state-level analysis for Victoria by the Victorian Department of Human Services (DHS 1999a, 1999b). Both project teams worked together closely on methods and analyses.

This work represented the first attempt to carry out a systematic and comprehensive analysis of over 170 disease and injury categories in this country. It also substantially extended the

international work on burden of disease in many areas, as shown by the fact that a number of its methodological advancements were subsequently picked up in the GBD 2000 work at WHO (Mathers et al. 2004). Since then, burden of disease analysis has been undertaken in most jurisdictions throughout Australia, at varying levels of detail. The update of the Victorian Burden of Disease study for the year 2001 (DHS 2005) deserves special mention as a number of disability models and data sources were shared between the researchers working on that project and those working on the present study.

This study was conducted in close consultation with relevant jurisdictional stakeholders, and the national and jurisdictional estimates in this report are intended to complement existing estimates from individual State and Territory based burden of disease studies. Because of somewhat different estimating methods and data sources, the jurisdictional estimates in this report may differ somewhat from State and Territory based estimates. This does not mean that one estimate is more correct than the other, but reflects the uncertainties inherent in any analysis which attempts to estimate burden for over 170 conditions.

1.6 Burden in Aboriginal and Torres Strait Islander peoples

Findings about Aboriginal and Torres Strait Islander peoples are not covered in this report, the primary focus of which is on the health status of Australians as a whole. This is not a problem for most of the comparisons presented, although special caution should be taken when interpreting the results of Chapter 5 on health differentials, particularly the estimates for remote areas and the Northern Territory. The higher proportion of Indigenous people in these areas explains most of the greater health loss in these areas compared with those where the proportion of Indigenous people is lower. However, the contribution of Indigenous populations to this loss has not been quantified in this report. Readers seeking to know such comparisons are referred to the companion report on the burden of disease and injury in Aboriginal and Torres Strait Islander peoples.

1.7 Structure of report

Details of the specific methodological developments of this study are presented in Chapter 2. Chapter 3 provides an overview of the total burden of disease and injury in Australia, by cause, age and sex. Chapter 4 provides estimates of the burden of disease and injury attributable to selected risk factors in Australia. Chapter 5 shows how the burden of disease and injury across Australia varies according to where people live and their socioeconomic status. Chapter 6 presents the past, present and projected burden of disease and injury in Australia and Chapter 7 provides a general discussion of the major findings.

Technical notes on the methods used for estimating non-fatal health outcomes and attributing risk are presented in Appendixes 1 and 2, respectively. Annex table 1 summarises the disease and injury categories used and their respective International Classification of Diseases codes. Annex table 2 summarises the primary data sources used to construct the core set of results. Tabulations of the core results are included in Annex tables 3 to 9. More detailed tabulations of the core results are available in Annex tables 10 to 25, which are available on the web at <www.aihw.gov.au/bod>.

Readers should note that every attempt was made to identify the best available information in the preparation of this report, and to consult as widely as possible on decisions about methods, assumptions and data sources. For some aspects of the study, however, it was not possible with the resources available to go beyond simple models and assumptions about some key parameters. For many disease models, not all required information was available and analyses drew on information from overseas or expert opinion. In the projections work, trends in disease occurrence were nonexistent for many conditions. The results presented in the following chapters, therefore, represent a complex synthesis of information, judgment and, in some cases, even speculation. It is hoped that further improvements over time in methods, models and data will result in increasing accuracy and certainty in estimates of burden of disease and injury in Australia. The authors at the University of Queensland and the Australian Institute of Health and Welfare welcome suggestions for such improvements.

2 Methodological developments

This chapter discusses the key methodological considerations that underpin the findings presented throughout the report. Readers who are only interested in these findings can skip to chapters 3 through 7 and return to this chapter at a later time. Those wishing to understand the ways in which the methodological challenges were resolved are encouraged to read on. The chapter begins by outlining some solutions to various methodological issues that are unavoidable in the application of the burden of disease and injury framework, including social value choices, causal attribution and comorbidity. It concludes with a description of the specific methods that were adopted to derive the findings on risks to health, burden across time and differentials in burden.

2.1 Social value choices

The burden of disease and injury framework encompasses some obviously normative characteristics (that is, it incorporates certain value judgments about how things ought to be). This is because its main measure (the disability-adjusted life year or DALY) comprises only a selection of all possible parameters that could be used to characterise health, and the numerical weighting given to each parameter implies a judgment about its relative importance to the total measure. These judgments have come to be known collectively as 'social value choices'. While the implications of certain choices over others are important and sometimes contested, as reflected by the growing literature in this area (Anand & Hanson 1997; Reidpath et al. 2003; Williams 1999), such considerations are beyond the scope of this chapter. The purpose here is to provide a brief discussion of the key choice that differs from the previous study. Readers are referred elsewhere for a more in-depth discussion on the merits of the other social value choices (Murray et al. 2002).

As mentioned in the previous chapter, the DALY is a health gap measure that requires an ideal against which to quantify the gap between current patterns of mortality and a counterfactual scenario in which all mortality is averted until very old age. The steering committee of the previous Australian Burden of Disease and Injury Study requested that projected life expectancy, based on a cohort life table (which takes into account past trends in mortality) for Australia, be used to define the mortality 'gap' for the purposes of calculating the years of life lost due to premature mortality (YLL). Until then, the standard that had been used in all burden of disease studies was based on the Coale and Demeny West level 26 model life table (Coale & Guo 1989), chosen after observing the highest life expectancy recorded for any nation (82.5 years for women in Japan at the time). It was then assumed that the minimum male-female 'biological' difference in survival potential was in the order of 2.5 years, but because there was no male schedule with a life expectancy of 80 years, the standard for males was based on the Coale and Demeny West level 25 schedule for females (Murray & Lopez 1996a).

The cohort life tables for Australia used in the 1996 study and the standard life tables used in other studies are very similar, and the substitution of one for the other would have had little effect on the final results. This is particularly true for discounted YLL, where the small differences in time lost would have been even further reduced by a time discount rate of 3%, although some differences were observable if undiscounted YLL were compared. For the current study, however, the situation is complicated by the fact that life expectancy in

Australia has changed since 1996 (an increase of 0.25 years and 0.3 years per annum for females and males, respectively). If the projected cohort life expectancy were to be used again, the mortality gap would be somewhat different because the projected cohort life expectancy based on changes in mortality rates to 2003 would be different from the old cohort life expectancy, which was based on changes in mortality rates to 1996. While the difference is not great, it does not aid comparisons to have a standard that is continually changing. Thus the current advisory committee has recommended a return to the internationally recognised standard used in most other burden of disease studies.

It is worth noting here that the life table for a population that actually achieves the 'ideal standard' (that is, no mortality until age 82.5 in females and 80 in males) would be very different from the standard life table. It is best to view the choice of the standard life table, therefore, as a weighting for age at death, without reference to the properties of the life table used to derive these weights.

All other social value choices remain as they were in the previous study (Table 2.1): uniform age weights and a discount rate of 3% were applied, and a combination of disability weights from the original GBD study (Murray & Lopez 1996a) and the Disability Weights for Diseases in the Netherlands study (Stouthard et al. 1997) were used. For some health states, there was no equivalent in either the Dutch or GBD set of weights, or the weights that appear in the published material seemed implausible. In these instances, the weights that were specifically derived for the previous Australian studies were applied. Unfortunately, a study to determine local weights for the range of health states most relevant to Australia was not able to be done. The complete list of weights is available at <www.aihw.gov.au/bod>.

Table 2.1: Social value choices used in the calculation of DALYs, 1996 study and present study

Choice	1996 study	Present study
Mortality counterfactual	Projected life expectancy based on cohort life tables for Australia in 1996	International standard first reported in Murray & Lopez 1996a
Age weighting	Uniform	Uniform
Discount rate	3%	3%
Source of disability weights	Murray & Lopez 1996a, Stouthard et al. 1997 and locally derived	Murray & Lopez 1996a, Stouthard et al. 1997 and locally derived

Box 2.1: Interpreting a disability weight

To place a value on the time lived in non-fatal health states, health state weights are used to formalise and quantify social preferences for different states of health. Depending on how these weights are derived, they are referred to as disability weights, quality-adjusted life year (QALY) weights, health state valuations, health state preferences or health state utilities. QALY weights are measured as a number on a scale of 0 – 1, where 0 is assigned to a state comparable to death and 1 is assigned to a state of ideal health. This assignment for the DALY (where 0 = perfect health and 1 = death) is the complement to 1, compared to that used for the QALY, because the QALY measures equivalent healthy years lived, whereas the DALY measures loss of health.

Although the disability weights used in DALY calculations quantify societal preferences for different health states, the weights do not represent the lived experience of any disability or health state, or imply any societal value for the person in a disability or health state. Rather, they quantify societal preferences for health states in relation to the societal ideal of good health. Thus, a weight for paraplegia of 0.57 does not mean that a person in this health state is ‘half-dead’, that they experience their life as halfway between life and death, or that society values them less as a person compared with ‘healthy’ people. It means that, on average, society judges a year with blindness (weight 0.43) to be preferable to a year with paraplegia (weight 0.57), and a year with paraplegia to be preferable to a year with unremitting unipolar major depression (weight 0.76). It also means that, on average, society would prefer a person to have a year in good health followed by death than a year with paraplegia followed by death. Society would also prefer to restore a person with paraplegia to good health rather than restore a person’s sight if the costs of cure are the same for the two interventions.

2.2 Causal attribution

There are two traditions for causal attribution of health outcomes or states: categorical attribution and counterfactual analysis (Mathers et al. 2001). In categorical attribution, an event such as death is attributed to a single cause (such as a disease or risk factor) or group of causes according to a defined set of rules, such as the International Classification of Disease (ICD) system for attributing causes of death (WHO 1992). In counterfactual analysis, the contribution of one or a group of risk factors to disease or mortality is estimated by comparing the current or future disease burden with the levels that would be expected under some alternative hypothetical scenario (referred to as the counterfactual). This study uses both approaches: categorical attribution for attributing burden to diseases and injuries, which is discussed below, and counterfactual analysis for attributing burden to more distal risks to health, which is discussed in a subsequent section.

Estimates of burden are typically attributed to a comprehensive set of disease and injury ‘entities’ (for example ischaemic heart disease or falls). These entities represent the smallest unit of disaggregation in the analysis and are referred to in this report as ‘specific causes’ or ‘conditions’. Each entity is mutually exclusive and belongs to one of a number of ‘broad cause groups’, most of which correspond to chapter-level headings of the ICD (for example cardiovascular disease or intentional injuries). Each broad cause group, in turn, belongs to one of three broad clusters:

- Group I: Communicable, maternal, neonatal and nutritional conditions
- Group II: Non-communicable diseases
- Group III: Injuries.

Annex Table 1 defines the classifications used in this study in terms of ICD-10 codes, most of which are consistent with the classifications used by WHO in the GBD2000 project (Mathers et al. 2004). A comparison of the ICD-10 list and the one based on ICD-9 used in the previous study is available at <www.aihw.gov.au/bod>.

Categorising deaths

The ICD has its origins in the preparation of mortality statistics, and standard death statistics use the categorical approach to causal attribution. While any number of conditions may be recorded on a death certificate, the ICD allows for only one to be selected for primary tabulation purposes. This single cause is referred to as the 'underlying cause of death' and is intended to represent the condition, event or circumstances without the occurrence of which the person would not have died. The concept of underlying cause has been central to mortality coding and comparable international mortality reporting over the 100-year period that the ICD has been used for such purposes.

Box 2.2: Death registration in Australia

Registration of deaths in Australia is the responsibility of the state and territory Registrars of Births, Deaths and Marriages. Information on the cause of death is supplied by the medical practitioner certifying the death or a coroner. Other information about the deceased is supplied by a relative or other person acquainted with the deceased or by an official of the institution where the death occurred. Registration of death is a legal requirement in Australia, and compliance is almost complete. The information is provided by the Registrars to the Health and Vitals Unit at the Queensland office of the Australian Bureau of Statistics (ABS) for coding and compilation into national statistics. The ABS began automated coding of death certificates using software known as the Mortality Medical Data System (MMDS) in 1997 and has made available multiple causes of death data coded in ICD-10 for all years since that time. Before 1997, only underlying cause of death data are available. The MMDS was developed by the National Center for Health Statistics in the United States of America to facilitate the coding of all causes of death reported on death certificates, and the designation of the underlying cause of death according to ICD criteria.

The availability of an unambiguous set of rules, such as can be found in the ICD, does not alter the fact that the accuracy of the information to which these rules are applied is dependent on several factors: the availability and quality of the clinical evidence at the time of certification; the thoroughness and diligence with which physicians and coroners record this information on the death certificate; and the quality of the system used to transcribe information from death certificates and translate this information to ICD codes. Australia is regarded as having a high-quality system of registration by international standards and this is reflected by one measure of quality, the proportion of total deaths coded to non-specific underlying causes of death. The small amount of non-specific coding that does occur is confined mainly to the ill-defined sections of the cardiovascular disease, cancer and injury chapters, with only a very small proportion of deaths being coded to the general signs and symptoms chapter. However, with the exception of a few studies on sensitivity and specificity in relation to specific conditions, relatively little is known about the frequency with which Australian doctors attribute the correct underlying cause to the majority of deaths. It is likely that accuracy varies with the location of the death (for example in an

institutional setting versus at home), but the assumption that inaccuracies tend to cancel each other out at the population level is largely speculative and is an area deserving of further research.

While this study largely followed the ICD concept of ‘underlying cause’ in the categorisation of deaths, in some cases deaths were reallocated to more specific or different categories to ensure consistency with the estimates for years lost due to disability (YLD). For example, the proportion of liver cancer and liver cirrhosis mortality that is attributable to hepatitis was redistributed to the hepatitis B and hepatitis C categories in the core results. Similarly, data on the underlying cause of renal failure from the Australia and New Zealand Dialysis and Transplant Registry (ANZDATA) was used to redistribute renal failure deaths to nephritis & nephrosis, diabetes mellitus, injuries, congenital conditions, cancers and infectious diseases.

It is important to note that for many conditions there is a difference between the number of deaths attributed to the disease and amount of excess mortality that occurs in prevalent cases of the disease. This is often due to comorbidity and the fact that diseases may cluster in people exposed to the same risk factors that also affect the risk of dying from other causes. Examples of this are schizophrenia, where part of the excess risk is due to the high prevalence of smoking and diseases associated with the usually lower socioeconomic status of people with chronic and severe mental disease; and cardiovascular disease, where the main lifestyle risk factors also increase the risk of dying from diabetes and some cancers.

For the overall cause of death structure presented in this report, recorded underlying causes of death were used, subject to the redistribution algorithms discussed below. In the disease modelling discussed in Appendix 1, however, best available estimates of excess mortality were used in order to derive the most accurate estimates of disease duration.

Redistributing non-specific causes of death

In keeping with established ‘burden of disease’ methods, attempts were made to remove possible distortions to the reported overall cause of death structure by reallocating deaths with certain codes known to be problematic to valid and specific underlying causes of death. The rationale for not taking reported causes of death at face value is that policy objectives are best served by information that is corrected for possible sources of systematic bias. By world standards, the extent of distortions in cause of death information in Australia is small (around 6–10%, depending on what codes are included in this definition). In some areas, however, there are obvious anomalies that require specific attention.

Murray and Lopez (1996a) were the first to provide convincing evidence that a significant and varying proportion of ischaemic heart disease deaths are coded in many countries to ill-defined codes such as heart failure. They argued that this, in part, helps to explain the French paradox in which mortality from ischaemic heart disease in France is comparatively low despite high levels of exposure in the French population to risks known to be associated with this disease. In fact, many ischaemic heart disease deaths are most probably being coded to heart failure or other equally non-specific cardiovascular causes. Policy is better served by correcting this misclassification error.

Various redistribution algorithms to correct non-specific cause of death coding have been developed in response to these considerations throughout the world. In the previous Australian Burden of Disease and Injury Study, for example, a number of decisions were made about what to do with problem coding based on local considerations regarding the cause of death collection system at the time. One of the guiding principles of the present

study was not to change past decisions such as these unnecessarily, unless there were compelling reasons to do so, such as new evidence.

In the period since the completion of the previous study, the vital registration system in Australia has changed in two significant respects. First, the ABS moved from the coding of mortality using version 9 of the ICD to version 10 in 1997. Second, at the same time, the ABS implemented automated coding of mortality statistics using software developed in the United States. The use of this system allows multiple cause of death coding (that is, coding of the underlying cause of death as well as all other associated causes recorded on the death certificate by the certifying medical practitioner), significantly enhancing the amount of information on official mortality files (see Box 2.2). To facilitate an assessment of the impact of these changes, the ABS retained the old system of coding for a period of two years, thus providing an invaluable resource for researchers trying to assemble comparable data on causes of death in Australia over time.

The availability of this additional information has allowed known problematic codes to be examined in much greater detail than has been possible in the past. It has also allowed the identification of some areas where possible new coding anomalies are emerging. The most glaring of these is the much greater number of deaths being coded to pneumonia under the new system. In the seven years to 1997, there were around 1,700 deaths from this condition annually. With the advent of automated coding, this number has risen to around 3,300 deaths annually. Such dramatic shifts are not due to changes in underlying disease frequency, but are rather an artefact of a greater preference under the new system to code deaths to this category (manual coders, on the other hand, were probably more likely to attribute an underlying chronic condition). Rather than correcting for this large discontinuity, which would then need to be repeated in the future to ensure comparability, the coding for these deaths was left unchanged. This explains the rapid rise in lower respiratory tract infections from 1993 to 2003 described in Chapter 6.

The other area where a discontinuity of this magnitude is apparent is the greater preponderance under the new system to code deaths due to external causes to 'exposure to unspecified factor' (ICD-10 code X59). Analysis of the dual-coded data revealed that the majority of these deaths in the elderly were in fact coded to 'falls' under the old system. In this instance, an additional allocation algorithm was applied whereby deaths coded to this category (around 0.6% of all deaths) were reallocated to 'falls' if they also had a 'fracture' code in the multiple cause of death data (AIHW: Cripps & Carman 2001). This approach was also used for 'unspecified septicaemia' (ICD-10 code A419), whereby deaths in this category (again, around 0.6% of all deaths) were reallocated to 'nephritis & nephrosis' if they also had an 'acute renal failure' code (ICD-10 code N17).

Another area where the new system may be in error is in the assigning of inappropriate underlying causes where another code would have been more informative. For example, in the 7-year period to 2003, 548 deaths were coded to tobacco dependence as an underlying cause. Likewise, 885 deaths were coded to obesity and 2,072 to hypercholesterolaemia and dyslipidaemia over the same period. These codes are most appropriately regarded as risk factors for more specific underlying disease processes and preferably should not be used in primary underlying cause of death tabulations. The number of deaths coded to these categories is likely to substantially underestimate the true mortality attributable to these risks (which is estimated in this report using very different methods, as discussed in Appendix 2). Deaths coded to tobacco dependence were therefore redistributed across lower respiratory tract infections, mouth and oropharynx cancers, lung cancer, ischaemic heart disease, stroke, other cardiovascular disease, chronic obstructive pulmonary disease (COPD)

and other chronic respiratory diseases based on a probability analysis of multiple-cause information over the period 1997 to 2003. Obesity was allocated to ‘other endocrine & metabolic disorders’ and the other two codes (about 300 deaths per year) to ‘ill-defined cardiovascular disease’, which was ultimately reapportioned to specific cardiovascular diseases (largely ischaemic heart disease).

The probability approach using multiple causes of death information was also applied to two other categories: ‘ill-defined nutritional’ (ICD-10 codes E64 and E639) and ‘essential hypertension’ (ICD-10 code I10). The first (representing 0.1% of all deaths) was redistributed across lower respiratory tract infections, other endocrine & metabolic disorders, dementia, other chronic respiratory diseases, and nephritis & nephrosis. The second (accounting for 0.2% of all deaths) was redistributed across all specific cardiovascular diseases.

Useful though it is, multiple cause of death information provides no new insights about three known problematic areas: ill-defined cancer, ill-defined injury and ill-defined non-injury deaths. It turns out that these deaths are assigned non-specific codes precisely because there is very little other information of relevance either on the death certificate or through coronial investigations (in the case of external causes) to make a more accurate determination. In the previous study these causes were allocated to specific cause groupings on a pro-rata basis on the assumption that the proportional distribution within these groupings reflected the most likely probabilities for causal attribution to a specific cause. There is no new evidence to alter these decisions. These causes and the cause groupings to which they were proportionately redistributed are listed in Table 2.2.

Table 2.2: Ill-defined causes of death and specific cause groupings to which they were allocated on a pro rata basis

Ill-defined cause ^(a)	Per cent of all causes	Specific cause groupings ^(a)
Ill-defined malignant neoplasms ^(b)	1.92	All specific cancer sites
Uterus cancer—unspecified ^(b)	0.04	Cervix cancer Corpus uteri cancer
Other anaemias	0.06	Haemolytic anaemia Other non-deficiency anaemia
Ill-defined non-injuries (i.e. diseases) ^(a)	0.39	All specific non-injury causes
Ill-defined unintentional accidents (no fracture) ^(b)	0.11	All specific unintentional injury causes

(a) Refer to Annex Table 1 for the ICD-10 codes that correspond to these cause categories.

(b) Denotes a redistribution decision derived from the previous Australian Burden of Disease and Injury Study.

Based on an assessment of cause of death statistics in Australia over a 25-year period, including the seven years of multiple causes of death information to 2003, a number of redistribution decisions were retained from the previous study, largely because there was no compelling reason to do otherwise. The list of these causes and the corresponding specific causes to which they are proportionately redistributed is outlined in Table 2.3.

Table 2.3 (continued): Ill-defined causes of death and percentage allocation to specific causes

Ill-defined cause ^(a)	Allocation to specific causes (%) ^(a)																										
	Deaths in ill-defined causes as % of all causes	Chlamydia	Other STD	Hepatitis B	Hepatitis C	Birth trauma & asphyxia	Low birth weight	Neonatal infections	Other perinatal	Type 1 diabetes	Type 2 diabetes	Nephritis & nephrosis	Pepitic ulcer disease	Cirrhosis of the liver	Ischaemic heart disease	Inflammatory heart disease	Hypertensive heart disease	Other cardiovascular disease	Suicide & self-inflicted injuries	Homicide & violence	Road traffic accidents	Poisoning	Falls	Fires/burns/scalds	Drowning	Other unintentional injuries	
Persons 15+ years																			90		10						
<i>Falls—intent undetermined^(b)</i>	0.01																										
Persons 0–14 years																					100						
Persons 15+ years																			90			10					
<i>Poisoning—intent</i>	0.04																										
Persons 0–14 years																					100						
Persons 15+ years																			90				10				
<i>Burns—intent undetermined^(b)</i>	0.00																										
Persons 0–14 years																					100						
Persons 15+ years																			90					10			
<i>Drowning—intent</i>	0.01																										
Persons 0–14 years																					100						
Persons 15+ years																			90								
<i>Other accidents—intent</i>	0.00																										
Persons 0–14 years																					100						
Persons 15+ years																			90							10	
Persons 0–14 years																					100						
Persons 15+ years																			90								

(a) Refer to Annex Table 1 for the ICD-10 codes that correspond to these cause categories.

(b) Denotes a redistribution decision derived from the previous Australian Burden of Disease and Injury Study

(c) Denotes neonatal deaths coded to maternal conditions (ICD-10 codes P00–P02) and subsequently redistributed back to neonatal causes based on an analysis of dual-coded data.

Alternative categories

In order to present the burden for mutually exclusive categories, decisions had to be made on how to classify sometimes closely linked conditions while still adhering to ICD rules. Chapter 3, however, presents alternative calculations of the burden (Table 3.20) due to certain disease entities that otherwise are split across a number of categories in the main disease and injury tabulations. The three entities are intellectual disability, renal failure and vision disorders, although other groupings are also possible (for example heart failure). Underlying causes of intellectual disability are various and include Down syndrome, central nervous system defects, birth trauma, low birth weight, infection, injury, brain tumours, chromosomal causes, epilepsy and autism. Renal failure can be attributed to diabetes, some cancers, congenital conditions and injury.

Alternative calculations are also presented for diabetes and depression & anxiety because these conditions are themselves risk factors for other causes of disability. The alternative estimate for diabetes includes the proportion of burden from ischaemic heart disease and stroke that is due to this disease. Likewise, for depression & anxiety the proportions of ischaemic heart disease and suicide caused by this condition are attributed. A new approach in this study, also, is that suicide is attributed to a range of mental and substance use disorders rather than to depression alone. These alternative calculations appear under the relevant disease or injury group subheading.

2.3 Comorbidity and health

It is not uncommon for two or more conditions to occur simultaneously in a person, either by chance or because the conditions are related to each other. This is referred to as 'comorbidity'. Independent comorbidity is the situation where the probability of having two or more conditions simultaneously equals the product of the probabilities for having each of the conditions. Dependent comorbidity, on the other hand, refers to the situation where the probability of having two or more diseases is greater than the product of the probabilities for each disease, reflecting common causal pathways (for example common risk factors causing both diabetes and heart disease) and also that one disease may increase the risk of another.

Both types of comorbidity are problematic for burden of disease estimation because the available disability weights are almost exclusively derived for a condition as it exists independently from other conditions. Little attention has been directed towards estimating weights for comorbid (or coexisting) conditions due to the enormity of the task. The severity of health states associated with two or more conditions in combination may not simply be the sum of the disability weights for each of the conditions. In many cases it is likely to be less than the sum, but in some cases there may be exacerbating effects on health of having the combined set of conditions. For example, the experience of symptomatic grade 2 osteoarthritis of the hip and severe vision loss together is probably not as disabling as the addition of the two weights for these health states (0.14 and 0.43, respectively). The experience of the latter with profound deafness, however, may be equal to or even more disabling than the summation approach would suggest.

In contrast to the GBD 1990 study, an attempt was made in the original Australian studies to accommodate this phenomenon by adjusting the disability weights for the 21 most common non-fatal conditions of older age (for example hearing loss, osteoarthritis, heart conditions, and diabetes). A multiplicative model was used to estimate weights for comorbid conditions,

and the change in total weight deducted from the weight for the milder of the conditions (see Box 2.3). Mental health problems are less prevalent at older ages, apart from dementia, and no attempt was made to adjust for mental–physical comorbidities, although comorbidity between mental disorders was accounted for.

A key assumption in the implementation of this adjustment procedure was that the prevalence of a set of comorbid conditions is equal to the product of the individual prevalences of these conditions. In other words, dependent comorbidity was not considered. More recent work as part of the GBD 2000 study, however, suggests that dependence is important and has a non-trivial impact on final results (Mathers et al. 2006). As a result, it was decided to incorporate the empirical evidence, limited though it is, on disease dependence into the overall corrections for comorbidity.

Box 2.3: Combining disability weights

The simplest approach to estimating the disability weight for the combined conditions 1 and 2 is to assume that the health state valuations (1 – disability weight) are multiplicative, so that the combined weight is more severe than the weight for either condition on its own but less than if they were simply added together, and remains bounded by 0 and 1. The disability weight for the combined conditions 1 and 2 is given by:

$$DW_{1+2} = 1 - (1 - DW_1) \times (1 - DW_2)$$

This formula can be generalised to deal with more than two causes as follows:

$$DW_{total} = 1 - \prod_i (1 - DW_i)$$

where \prod denotes the product operator.

In the original Australian studies, this method was used to derive a composite weight for comorbid conditions. In the case of two conditions, the weight for the most severe condition remained unchanged, while the weight for the milder condition was deemed to be the balance of the composite weight minus the weight for the more severe condition. For example, if a person has symptomatic grade 2 osteoarthritis of the hip or knee (0.14) and severe vision loss (0.43), the composite weight for both conditions is 0.51 and the adjusted weight for the osteoarthritis is 0.08.

In the current study the disability weights are proportionately reduced for each comorbid state.

The approach taken in this study was to determine the numbers of people for every combination of causes of ill-health measured by the major Australian health surveys and in the National Hospital Morbidity Database. While none of these data sources contained information on every cause of interest, each overlapped in the causes they did provide information on, at least to some degree. This allowed comorbidity to be simulated across the full range of causes by deriving conditional probabilities on causes common to two or more surveys and generating an artificial cohort of people based on these probabilities. The assumption was that the correlations observed in self-report surveys and hospital diagnoses are reasonable proxies for the co-occurrence of disability in these samples, even though these data sources may not accurately reflect the actual levels of disease at the population level.

Unlike the previous study, this study did not incorporate a severity hierarchy of the disability weights by causes. Instead, a proportional downward adjustment was made to the disability weight of each coexisting cause. The proportion used to deflate individual

disability weights was the total adjusted disability weight divided by the total unadjusted disability weight for each cause and all possible combinations. A further consideration that has not been explicitly addressed in previous work is that when a disability weight changes with advancing age (due to comorbidity corrections or for some other reason), incident YLD should be calculated to incorporate these changes. In other words, if the duration of a condition is 20 years, incident YLD should be calculated using the disability weight that is relevant to each age above the age of incidence until the 20-year duration has been reached, rather than using the weight at the age of incidence for the whole 20-year period. This correction was implemented in the present study.

2.4 Risks to health

Reliable and comparable assessments of the impact on population of exposure to health risks are fundamental to prevention and health promotion activities. Until relatively recently, health risk assessment has been conducted in the context of the methodological traditions of individual risk factors, with little regard to achieving consistency between these traditions when combining results. In the original Australian study, for example, the criteria for evaluating the scientific evidence on prevalence, causality and hazard size varied greatly among the 10 health risks assessed, resulting in lack of comparability between the estimated population health impacts of these risks.

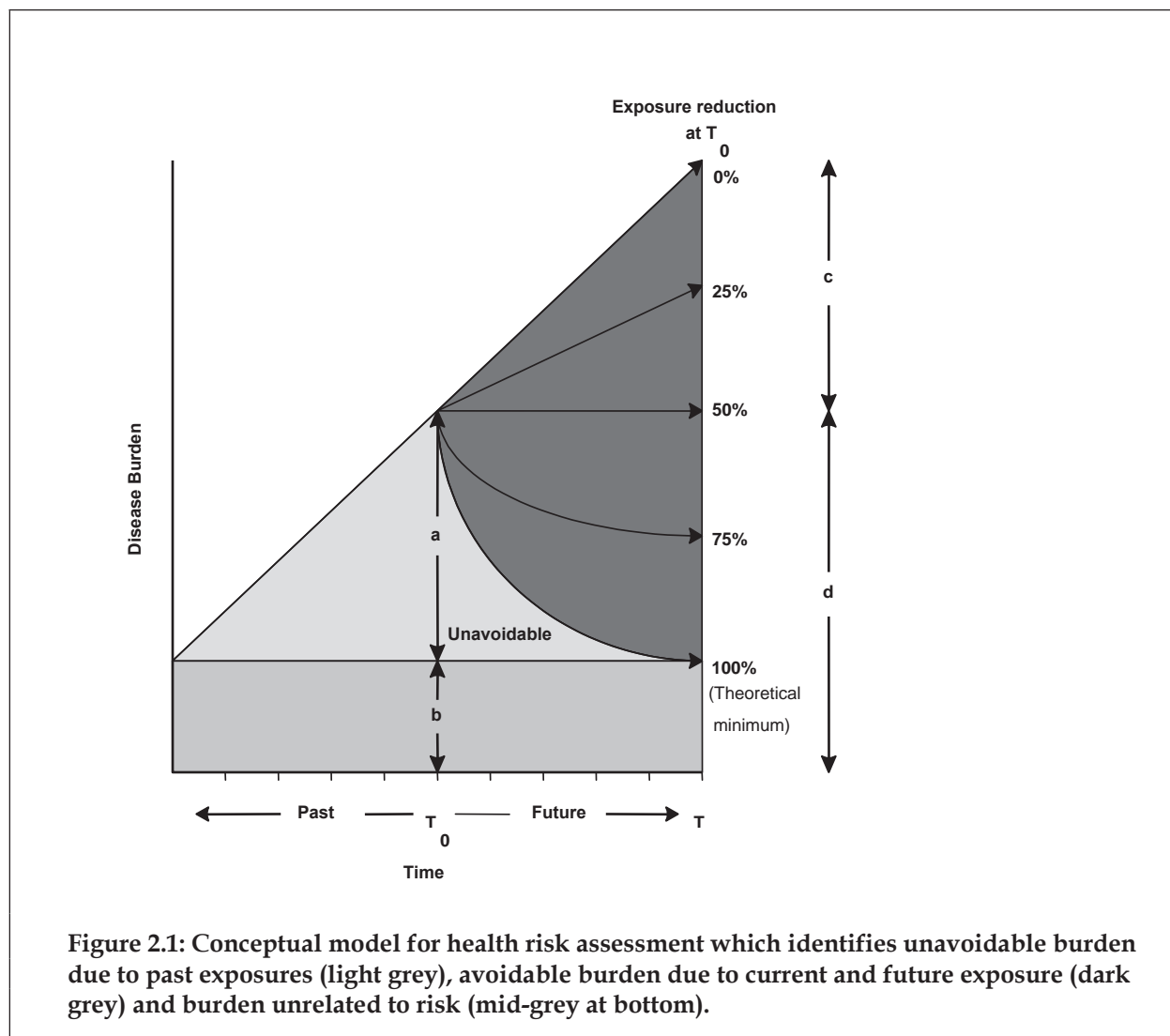
Techniques for attributing outcomes to health risks have advanced considerably in recent times, particularly through the contribution of the Comparative Risk Assessment (CRA) project. This was a large-scale effort by international panels of experts under the direction of the World Health Organization (WHO) to collect the most up-to-date information on the prevalence of exposure to health risks and the relationship between these exposures and health outcomes. WHO dedicated its 2002 World Health Report to describing the results of this effort (WHO 2002), and subsequently published a two-volume book containing detailed information on each of the 22 health risks covered by the project (Ezzati et al. 2004a, 2004b).

The key advances of the CRA approach over previous attempts to attribute burden to health risks are:

1. A consistent theoretical framework that uses the 'hypothetical minimum' as the counterfactual against which burden due to a risk is calculated.
2. Inclusion of continuous risk variables that previously were categorical in nature, that is, taking into account the full range of risk from elevated blood pressure, serum cholesterol, body mass index (BMI) or inadequate fruit and vegetable intake rather than defining thresholds for hypertension, hypercholesterolaemia, underweight/obesity and low fruit and vegetable consumption.
3. A more systematic review of the international literature on the impact of risk factors on health outcomes, including estimates of relative risk for a unit of increase in continuous risk factors.
4. A theoretical framework and provisional methods for estimating the joint effects of multiple risks to health.

Explicit 'counterfactuals'

Estimating the health risks associated with exposure to a particular hazard in a population is typically undertaken with reference to an alternative or 'counterfactual' distribution of exposure (for example exposed versus not exposed). While different counterfactual distributions may be used for different purposes (Murray and Lopez (1999) identify at least four of potential interest), an important contribution of the CRA project was to seek consistency in the definition and use of this distribution across each of the 22 risks analysed. In burden of disease and injury studies, the counterfactual of greatest relevance to the question 'How much of this health outcome is due to that exposure?' is the 'theoretical minimum' risk distribution. This is defined as the distribution of exposure that would yield the lowest possible risk in a population (for example zero tobacco use) and is useful for determining how much of current burden is due to past exposure to a particular hazard (the light grey area of Figure 2.1). This is distinct from intervention analyses, which are typically interested in how much future burden could realistically be avoided by shifting current exposure through the implementation of a particular intervention (various scenarios depicted in the dark grey area of Figure 2.1).



While simple enough to operationalise in the context of hazards for which absence of exposure is indeed the lowest possible risk, the concept of ‘no exposure’ is problematic when lack of exposure is not meaningful, as is the case for blood pressure, cholesterol and body mass. Before the CRA project, this issue was avoided by the categorisation of these hazards into normal and abnormal (for example hypercholesterolaemia, hypertension, overweight or obesity). Although relevant from a clinical management perspective, this approach is likely to underestimate the population-level attributable burden; even though the elevation in risk at levels of exposure below these cut-points may be small, the large numbers of people at these levels contribute substantially to total population-level risk. The approach advocated by the CRA researchers was to respect the continuous nature of these hazards by assessing risk across the full distribution of exposure experienced by a population. This meant defining ‘theoretical minimum’ distributions even for hazards for which lack of exposure is not meaningful, which they did by drawing on evidence from very low-risk populations in the literature (Ezzati et al. 2003).

Joint risk attribution

Another area where the CRA project made an important contribution was the joint attribution of risks. Health risk assessment before this project typically provided information about burden attributable to a hazard in isolation from other hazards. The difficulty with this approach is that if several analyses are added together it can appear as if more than 100% of total burden for any one disease or injury is being accounted for by the hazards in combination. This is not an error in the individual risk attribution method itself but rather it is an issue of interpretation. Individual risk attribution analyses should not be added together, although this can be a difficult message to convey, particularly when they are presented together.

Estimating the joint effects of multiple risks is complex in practice for several reasons. First, some of the effects of the more distal factors (for example physical inactivity) are mediated through more proximal factors (for example via high BMI and from BMI via high blood pressure). Estimating the joint effects of more distal and proximal factors requires knowledge of independent hazards of the distal ones and the amount of risk mediated through proximal risk factors. Second, the hazard due to a risk factor may depend on the presence of other risk factors (effect modification). Third, there may be correlation between exposures to various risk factors, because they are affected by the same distal factors and social dynamics.

The approach used to estimate joint population attributable fractions (PAFs) in this study is based on methods developed for the CRA, in which the assumption is made that health risks are biologically independent and uncorrelated. This is, of course, an over-simplification, as some risks are not biologically independent (for example physical inactivity and BMI), and various exposures are highly correlated (for example smokers also tend to be drinkers). However, it allows the joint PAF for n number of risks to be expressed as:

$$\text{joint PAF} = 1 - \prod_{i=1}^n (1 - \text{PAF}_i)$$

where PAF_i is the PAF of individual risk factors.

The second term in the right-hand side of this equation (that is, the product of all $[1 - \text{PAF}_i]$ terms) is the fraction of burden not attributable to any of the n risk factors. One minus this term is the fraction attributable to the combined effects of the n risk factors.

For instance, inadequate intake of fruit and vegetables and high BMI increase the risk of colon cancer. Assuming there is no dependence or correlation between these two risks, if the PAF for fruit and vegetable intake is 0.20 and the PAF for BMI is 0.10, the burden attributable to the two risks equals $1 - (1 - 0.2) \times (1 - 0.1) = 1 - 0.8 \times 0.9 = 0.28$.

Epidemiological studies on the effects of high BMI, physical inactivity, and low fruit and vegetable consumption on cardiovascular disease risk have illustrated some attenuation of the effects after adjustment for more proximal factors (for example blood pressure or cholesterol) (Berlin & Colditz 1990; Blair et al. 2001; Eaton 1992; Gaziano et al. 1995; Jarrett et al. 1982; Jousilahti et al. 1999; Khaw & Barrett-Connor 1987; Liu & Manson 2001; Manson et al. 1990; Rosengren et al. 1999; Tate et al. 1998). This attenuation confirms that some of the hazard of the more distal factors operates by increasing levels of risk in factors closer in the causal pathway to the disease. The attenuation varies among studies but is consistently less than one-half of the excess risk (that is, $RR - 1$) of the more distal factors. An upper bound of 50% is used in this study as the proportion of the excess risk from BMI, physical activity and fruit and vegetable intake that is mediated through proximal factors that are themselves among the risks being analysed. For example, if the relative risk of BMI for diabetes is 4 for a particular level of BMI exposure, for the joint effects calculation a relative risk of $(4 - 1) \times 0.5 + 1 = 2.5$ is used to calculate the PAF that eventually feeds into the equation on the previous page. Joint risk factor estimates for cardiovascular disease are not very sensitive even to large variations in this assumption of attenuation (Ezzati et al. 2004a).

The burden attributable to both child sexual assault and intimate partner violence is estimated in this study for the first time. Evidence suggests that girls who experience child sexual abuse are more likely than non-abused girls to experience intimate partner violence (Mouzos & Makkai 2004). In the joint effects analysis for these exposures, the burden due to child sexual abuse and intimate partner violence is calculated as the sum of the PAFs for exposure to child sexual abuse only, exposure to intimate partner violence only, and the combined state of exposure to both risks.

2.5 Past, present and future burden

Forecasts about the future play an important role in shaping public policy. For example, an important consequence of economic development has been improvements in health, particularly among the elderly. Better health, in turn, has led to greater economic development and more people surviving to old age. Together with decreasing fertility, this has contributed to 'population ageing'.

There is increasing analysis being undertaken in relation to the long-term sustainability of public finances in the context of these widespread demographic trends across the developed world. Under the Charter of Budget Honesty Act 1998, the Australian Government is required to prepare an Intergenerational Report (IGR) that assesses the long-term sustainability of current Government policies over the next 40 years, and to take account of the financial implications of demographic change. The first IGR was released on 14 May 2002 as part of the 2002-03 Federal Budget (Budget Paper No. 5) and considered future health care costs based on expected demographic trends and projected Australian Government expenditure on health services, represented as a proportion of gross domestic product (GDP), for the period 2002 to 2041.

Likely trends in disease occurrence were not explicitly accounted for in the IGR as the analytical projections were based on historical trends in major health expenditure program

groupings (medical benefits, pharmaceutical benefits and hospitals) at selected ages. It is optimistic to assume that simply because underlying changes in disease occurrence were embedded within the historical data on expenditure that they are therefore plausibly reflected in these analyses. An analysis that explicitly takes account of changes in both disease occurrence and per unit expenditure at the level of individual diseases is likely to provide much firmer ground upon which to base estimates of future health expenditure. Common to both approaches, of course, is the assumption that the rate of change in policy responses to emerging problems in the future is consistent with the rate observed in the historical period upon which the projections are based (that is, 'business as usual'). If these dynamics change, expectations with regard to the future will consequently change.

One objective of the present study was to address the need for comprehensive health projections in Australia by analysing the most likely changes in burden of disease and injury to the year 2023. The past is a good (but far from perfect) predictor of the future and an important by-product of such work is a comprehensive analysis of past trends in disease occurrence. To pre-empt the inevitable requests for information on the past, this part of the study was extended to include 'back-casting' of disease burden as well. This has the logical appeal of ensuring consistency between estimates of past, present and future disease burden. More importantly, it may limit the potential for misinterpretation should people compare these current and future burden estimates with results based on alternative methods. The inevitable comparison that people will make between the results presented in this report and those of the previous Australian Burden of Disease and Injury Study should be regarded in this light.

Australia has an excellent vital registration system by international standards and, with few exceptions (for example pneumonia), observable trends in vital events over time are arguably the most reliable and consistently recorded information on changes in the frequency of diseases and injuries that result in death. Previous work (Barendregt et al. 2003) has shown that the complete epidemiology of a disease is ultimately a function of only three parameters: incidence (the hazard of getting the disease), remission (the 'hazard' of being cured from having the disease) and case-fatality (the hazard of dying as a consequence of having the disease). For most chronic diseases, cause-specific mortality is influenced by only two of these – incidence and case-fatality – with remission having little if any role. It follows, therefore, that any epidemiological parameter of interest for a chronic disease can be 'back-cast' from a point in time for which the complete epidemiology of that disease is known simply by making assumptions about the relative contribution of incidence and case-fatality to the observed changes in mortality.

This idea also applies to projections, providing one is willing to make predictions about cause-specific mortality into the future. Since it has already been argued that cause-specific mortality is a reliable and consistently recorded source of information on changes in disease frequency in many cases, cause-specific mortality is a sound starting point for projecting the epidemiology of a disease. Other approaches that are based on predicting incidence from risk factors may have more intuitive appeal but are more tenuous as they involve multiple assumptions about disease-exposure relationships and future exposure trajectories.

The methods used in this study involved a number of separate analytical or computational steps. A brief outline of the overall approach is presented below. More complete details are provided in subsequent sections of the report as indicated.

1. Baseline models for over 170 diseases and injuries for Australia in 2003 were developed as part of the core set of analyses for the present study. Appendix 1 discusses each of these models in detail.

2. Trends in observed cause-specific mortality over the period 1979 to 2003 were analysed and projected into the future using a combination of regression techniques.
3. For mostly fatal conditions, each baseline disease model was extrapolated backwards and forwards in time based on assumptions about the relative contribution of incidence and case-fatality to changes in mortality. Baseline models for mostly non-fatal conditions were extrapolated based on assumptions about changes in incidence only. The complete epidemiology of each was then estimated separately in a fully dynamic model that accounted for changes in all-cause mortality as well as changes in incidence and case-fatality (where appropriate) so that incidence, prevalence and duration by age, sex and cause was described over the past as well as into the future.
4. Absolute numbers of incident and prevalent cases were derived by applying the rates from the above analyses to the ABS 'Series 8' projection series population estimates (ABS 2003d). This series assumes a high net overseas migration of 125,000 annually, constant improvements in life expectancy (low mortality assumption), and a total fertility rate declining to 1.6 by 2011 and then remaining constant.

Incident and prevalent YLD for each disease were calculated for non-baseline periods by applying durations and extrapolated numbers of incident and prevalent cases from the dynamic model to disability weights that were corrected for probabilities of comorbidity in 2003. Years of life lost (YLL) for non-baseline periods were calculated directly from observed deaths in the past and projected deaths into the future.

Mortality trends and projections

Observed all-cause mortality rates for the period 1979 to 2003 were extrapolated into the future using simple log-linear Poisson regression. Cause-specific mortality data for the same period were then collapsed into 51 clinically meaningful conditions, or groups of conditions. Multinomial logistic regression was used to model changes in the contribution of each group as a proportion of all-cause mortality, with changes in absolute levels of all-cause mortality expressed as the natural log of the rate per unit of population. These models were used to predict the future cause-specific structure of mortality based on projected all-cause mortality rates. Separate analyses were done for each age group and sex.

Among the causes analysed, cardiovascular disease, cancers, chronic obstructive pulmonary disease (COPD), diabetes, alcohol-related conditions, road traffic accidents, falls, suicide and homicide showed significant mortality trends. The apparent trend in dementia mortality was ignored because: (a) there has been a shift in coding practices with more deaths being attributed to dementia; (b) the prevalence data from international epidemiological studies showed no clear change over time; (c) the case-fatality was unlikely to have changed much over time as there are no effective life-saving interventions.

Incidence and case-fatality

Mortality trends for cancers, COPD, diabetes, alcohol-related conditions, road traffic accidents, falls, suicide and homicide were assumed to be fully due to changes in incidence. Incidence trends for these causes were therefore adjusted to reflect changes in mortality over the projection period, with case-fatality being held constant. Findings from Unal et al. (2004) suggest that 58% of the drop in cardiovascular mortality observed in England and Wales was due to a drop in incidence and the remaining 42% due to a reduction in case-fatality. The

same proportions were assumed to apply in this study to all cardiovascular disease over the projection period.

Changes in the diagnostic criteria for Type 2 diabetes in surveys and a paucity of representative survey data meant that there was no direct measurement of trends of Type 2 diabetes in Australia from which to project the incidence of this disease. Body mass index (BMI, defined as body weight in kilograms divided by the square of height in metres), overwhelmingly the main risk factor for Type 2 diabetes, however, has been measured consistently at various points over recent time. The approach taken in this study, therefore, was to translate historical trends in BMI into expected changes in diabetes incidence following the risk attribution methods described in the WHO Comparative Risk Assessment project.

Haby and colleagues (2006) analysed trends in BMI using data from five measurement surveys: the three National Heart Foundation Risk Factor Prevalence studies in the 1980s, the National Nutrition Survey of 1995 and the AusDiab study in 1999 and 2000. Projected mean BMI by age group and sex was derived from Haby and colleagues' regression model of the mean of log-transformed BMI values on age, birth cohort and sex. Similar techniques were applied to the standard deviations of BMI values so as to fully describe the expected change in the distribution of this risk into the future (a change which can be characterised as a broadening of the distribution in the tail towards the highest BMI values rather than at the other end of the distribution with low values).

The population-level risk of diabetes is simply the area under the curve represented by the distribution of BMI multiplied by the relevant relative risk of developing diabetes at each level of BMI. This is easiest to derive using integration techniques. Proportional changes in the size of this area over time represent changes in the incidence of diabetes resulting from changes in BMI. Ni Mhurchu and colleagues (2006) undertook a meta-analysis of results from the Asia-Pacific Cohort Study collaboration and report the relative risk of developing diabetes for each unit increase in BMI by age and sex. Using these relative risks and the predicted BMI distributions derived above, changes in the incidence of diabetes were estimated over the projection period. For consistency with CRA methods, a theoretical minimum distribution of BMI (mean of 21 and standard deviation of 1) was incorporated into the calculations, below which no excess risk of diabetes was assumed.

Information on trends in case-fatality rates amongst people with diabetes is scarce. In the absence of such information, an assumption was made that at least half the mortality in these people is due to vascular causes and is subject to the same factors that influence cardiovascular disease mortality more generally. Changes in case-fatality for diabetes, therefore, were assumed to reflect half the trends in case-fatality for cardiovascular disease, which were estimated to be decreasing over the projection period. The combined effect of increasing BMI and decreasing case-fatality was a considerable increase in the incidence of Type 2 diabetes, and an even greater increase in future prevalence.

Non-fatal conditions

Mortality trend data are not relevant for conditions that are largely non-fatal. These include mental, sense organ and musculoskeletal disorders. The only mental health survey in Australia was carried out in 1997 and hence there are no trend data. Internationally there is no clear evidence of trends due to a paucity of mental health survey data collected using comparable diagnostic tools and criteria. Therefore no trends were assumed. Similarly, no

disease trends were applied to hearing loss (only one community survey), and the various causes of vision loss and musculoskeletal disorders (no evidence for trends).

2.6 Differentials in burden

The high demand for information on health differentials, both between and within populations, is one measure of the obvious public policy implications of such information. For example, knowing that the gap in life expectancy at birth between Aboriginal and Torres Strait Islander Australians and other Australians is demonstrably very large is a sound basis for new initiatives to improve Indigenous health. One of the aims of the original study was to develop estimates of disease burden for different groups within the Australian population. To this end, the final report presented preliminary analyses of inequalities in disease burden by level of socioeconomic disadvantage, although it was not possible to complete a comprehensive analysis of non-fatal burden within the time available. An objective of the current project was to extend these analyses by providing a more complete picture of disease burden for a much greater range of subgroups within the Australian population.

The methods used in this study build on the first comprehensive attempt to describe 'small area' variability in health status across Victoria (DHS 2006), and are in the methodological tradition of describing differences in health across population subgroups. Murray and colleagues (1999a) differentiate this from descriptions of 'health inequalities', a term they reserve for analysis of the variation in health status across individuals in a population (analogous to analyses of income inequality, which measure the distribution of income at the level of individuals). While health inequalities are sometimes regarded as synonymous with subgroup differences in health in the literature, analyses of the latter are based on subgroup averages and as such can mask the true extent of inequalities between individuals.

Categorising geographic areas

The most disaggregated geographic information on place of usual residence for most Australian health data is the Statistical Local Area (SLA), and this geographic entity is used as the unit of analysis for this component of the study. For various reasons, SLA names and boundaries are revised over time, the most substantial revision occurring as a result of local government amalgamations in the early 1990s. To achieve geographic consistency, all data, regardless of year, were analysed in terms of ASGC definitions for the year 2001 (ASGC, or Australian Standard Geographical Classification, being the reference used to define SLAs) (ABS 2001a). Data defined in terms of SLAs fragmented as a result of boundary revisions were reapportioned using information from the 2001 Census on the proportion of each old SLA population residing in each current SLA after the redrawing of the boundaries. Irregular coding in data arising from such revisions was resolved on a case-by-case basis using historic documentation provided by the ABS. Estimated mid-year resident population figures for each SLA by year (1999 to 2003), 5-year age groups (0, 5...85+) and sex were obtained from the ABS.

The ASGC 2001 provides for the classification of SLAs in terms of both socioeconomic status and remoteness. Socioeconomic status can be determined from one of four socioeconomic indexes for areas (SEIFA indexes) developed by the ABS from the 2001 Census using principal component methods on attributes such as low income, low educational attainment,

high levels of public sector housing, high unemployment, and jobs in relatively unskilled occupations (ABS 2001b). This study uses the index of disadvantage that is functionally equivalent to the Index of Relative Socioeconomic Disadvantage derived from the 1996 Census. This index is estimated at a collector-district level to be normally distributed at a national level, and can be population-weighted to derive values for ASGC 2001 SLAs. Socioeconomic quintiles were derived by ranking SLAs in order of disadvantage index then grouping them into five categories such that each category contains approximately 20% of the total Australian population.

Remoteness can be determined from the Accessibility/Remoteness Index of Australia (ARIA+) developed by the Australian Government Department of Health and Ageing and the National Centre for Social Applications of Geographic Information Systems (GISCA), and subsequently incorporated into ASGC 2001 (ABS 2001a). ARIA+ is a continuous varying index with values ranging from 0 (high accessibility) to 15 (high remoteness), and is based on road distance measurements from 11,879 populated localities to the nearest service centre. Index values for each locality have been interpolated to a 1 km grid so that all areas of Australia have an index value and scores for larger areas such as SLAs can be derived. Each SLA was classified into one of three groups based on the following standard cut-points as defined in ASGC 2001: Major cities (0–0.20), Regional (>0.20–5.92) and Remote (>5.92).

Estimating burden for subpopulations

One category of information readily available for disaggregating national estimates of burden to subpopulations is data on observed variations in event frequency for any aggregation from the level of the SLA and above. This includes the National Mortality dataset, the National Hospital Morbidity Database and the National Cancer Statistics Clearing House dataset. The other category comprises information that can be tabulated by state or territory jurisdiction, disadvantage quintile or remoteness category, but cannot be disaggregated below these strata. Most surveys (for example the National Health Survey, the Survey on Disability, Ageing and Carers, the National Survey of Mental Health and Wellbeing, and the Australian Diabetes Obesity and Lifestyle Study (AusDiab)) and published data tabulations fit this description. The primary objective with either category was to derive relativities between whatever level of disaggregation was possible, and to ensure that these relativities were as accurate as possible and not simply an artefact of small numbers. Of less concern was the absolute level of disease occurrence being reported, because these would be constrained by national estimates.

The adopted strategy was intended to ensure consistency in the use of the available information and to ensure sufficient numbers at each level of the analysis. First, all sources were assessed for whether they could provide simple state/territory jurisdiction proportions (preferably by sex, but not necessarily by age) for any condition in the study's list of diseases and injuries. Most sources could provide this information. Next, they were assessed for plausibility as a valid proxy for variability in disease occurrence across a 15-cell matrix comprising five SEIFA categories and three remoteness categories. Not as many sources could provide this information and, of these, a few could provide information on only one dimension (that is, either SEIFA or remoteness, but not both). Age-standardised rates were then calculated for each cell of observed data, and these were divided by the crude rate for the whole matrix to derive 15 cell-specific standardised rate ratios. In matrices with only one dimension, ratios for the observed dimension were held constant across the missing dimension.

This estimation process means that the estimates of deaths of cancer cases in a particular SLA are not the same as the actual deaths or cancer cases in that SLA, but are synthetic estimates which reflect the rates of deaths and cancer in SLAs of similar type.

Having determined possible sources for two pieces of information (state/territory proportions and matrices of rate ratios), an assessment was made for each disease and injury category as to whether there was agreement between sources (if there were more than one) and which information seemed sufficiently robust in terms of underlying numbers. For conditions with a predominantly fatal burden, preference was given to information derived from mortality data. For other conditions, preference was given to the data source upon which the national disability model was based.

Each condition was then assigned a single source to be used to derive the proportion of national incidence cases that would be expected to occur in each state and territory. If no source could be identified, the number of incident cases was unconstrained at this step in the disaggregation. The implied jurisdiction-specific rate (or national rate where jurisdiction numbers were unconstrained) was then distributed to subpopulations within the jurisdiction using one of the matrices of rate ratios derived in the previous step. If no matrix was available, rates were held constant across subpopulations within jurisdictions. Derived incident cases were then rescaled to be consistent with jurisdiction totals where applicable, and ultimately national totals. Deaths were treated in the same way as incident cases.

The final step was to derive prevalent cases and duration for each condition and its sequelae for each subpopulation within jurisdictions. An automated implementation of the equations underlying DisMod (an epidemiological modelling software package) was applied to subpopulation-specific incidence rates and national assumptions regarding remission and case-fatality to derive these parameters. In order to derive accurate durations, one of 15 sets of all-cause mortality rates was used according to the SEIFA and remoteness category of the subpopulation. All subpopulation-specific prevalent cases and YLD (both incident and prevalent) were then rescaled to be consistent with national totals.

Subpopulation comparisons in this report

This report is limited to the following subpopulation comparisons:

1. state and territory jurisdictions
2. remoteness categories
3. socioeconomic quintiles.

While the analyses were aimed at disaggregating national burden estimates to the level of the SLA, there was no intention to disseminate results at this level of detail. In addition to the potential privacy considerations of the data providers, the release of such information may be misleading given the methods used. Rather, the authors and various jurisdictional stakeholders are working to regroup the data into meaningful aggregations of SLAs for specific health policy and planning purposes.

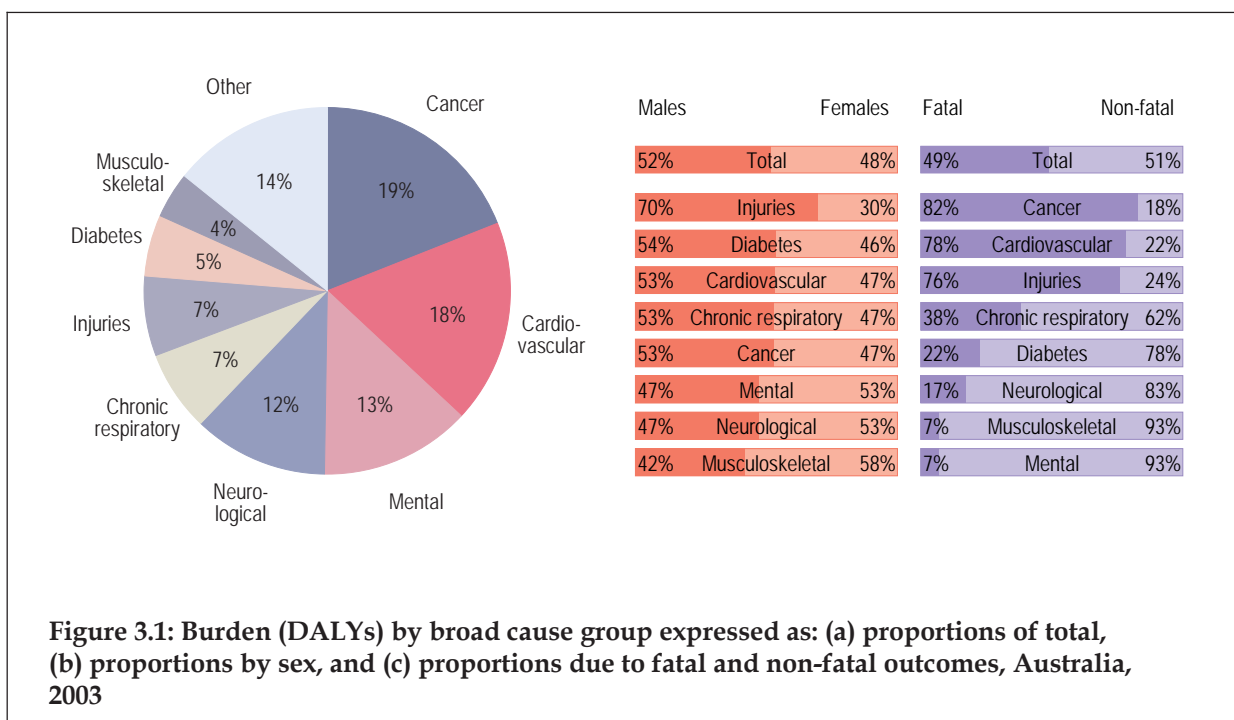
3 Burden of disease and injury in Australia

This chapter discusses the burden of disease and injury in Australia in 2003 by fatal and non-fatal burden, sex, age and leading broad cause group. The numbers presented in this chapter should not be compared with those presented in the previous report (AIHW: Mathers et al. 1999) due to substantially different methods for many of the disability models. Readers who are interested in gaining an understanding of changes in burden over time are referred to Chapter 6 which discusses trends in population health over a 30-year period.

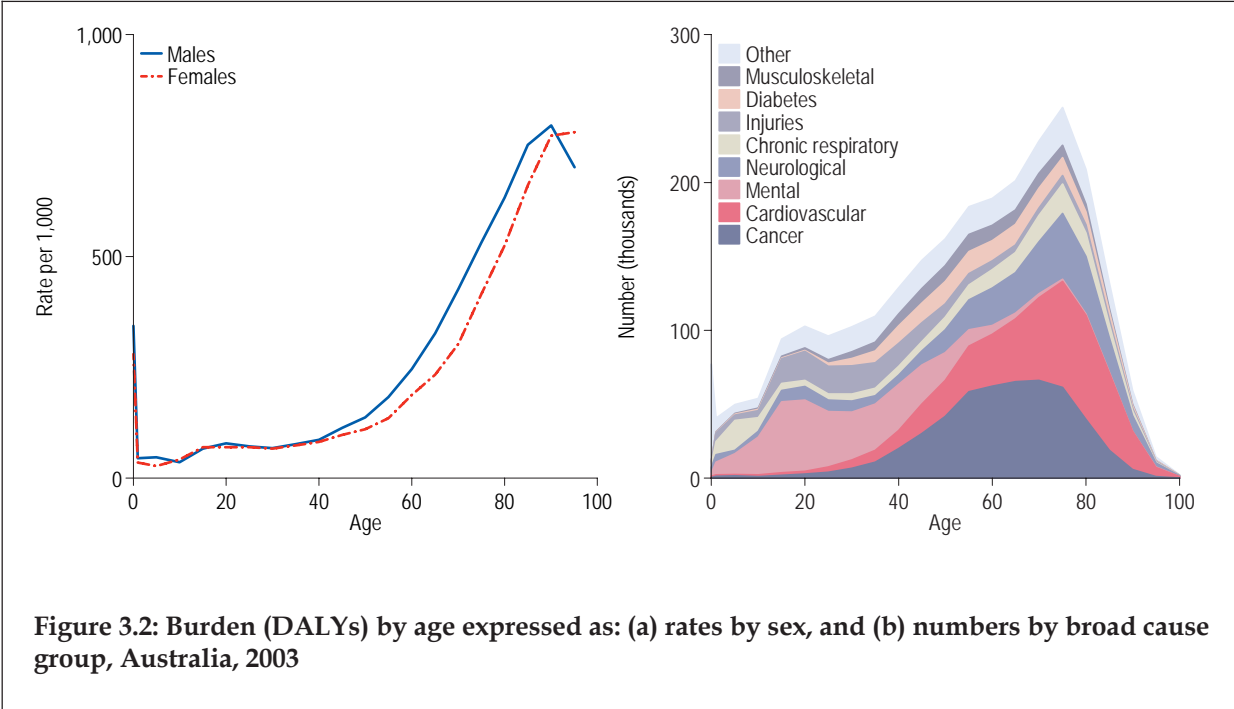
3.1 Disability-adjusted life years

Cancer, cardiovascular disease and mental disorders were the leading causes of total burden of disease and injury in Australia in 2003 (Figure 3.1). Cancer and cardiovascular disease accounted for 37% of the total burden; for both causes, four-fifths of this burden was from mortality. Mental disorders and neurological & sense disorders were the next largest contributors, together accounting for a further quarter of the total burden. The contribution of mortality to the burden from these two groups was small, highlighting the importance of including non-fatal health outcomes in population health measurement.

Overall, half the total burden (49%) was due to mortality and the distribution between the sexes was roughly equal for most causes, with injuries (70% of the burden in males) and musculoskeletal disorders (58% of the burden in females) the exceptions.



Total burden in absolute terms increased at a relatively constant rate until age 75 (Figure 3.2b), while the burden per head of population continued to rise exponentially, with small but significant peaks in childhood and early adulthood (Figure 3.2a). Injuries in males and mental disorders were the main cause groups until middle age and accounted for the majority of total burden in early adulthood, after which cancer, cardiovascular disease and neurological & sense disorders were more prominent. The contribution from cancer peaked at age 70 then declined, leaving cardiovascular disease as the major cause of burden in the elderly (Figure 3.2b).



The burden due to specific disease and injury categories reflected the more general picture at the broad cause group level. Ischaemic heart disease was the largest single cause in males, accounting for 11.1% of the total male burden (Table 3.1). For females, anxiety & depression was the leading cause, accounting for 10.0% of the total female burden. Ischaemic heart disease, stroke, Type 2 diabetes and dementia were the next four leading causes of DALYs in females. In males, Type 2 diabetes, anxiety & depression, lung cancer and stroke were the next four leading causes.

Seven health areas have been identified by the Commonwealth, state and territory governments for priority attention as National Health Priority Areas: asthma, cancer, cardiovascular disease, diabetes mellitus, injuries, mental health, and arthritis and musculoskeletal conditions. In addition, dementia is an Australian Government health priority. In 2003, these eight health groupings accounted for 72.8% of the total burden, 17 of the 20 leading conditions for males and 15 of the 20 leading conditions for females.

Table 3.1: Leading causes of burden (DALYs) by sex, Australia, 2003

Rank	Males	DALYs	Per cent of total	Females	DALYs	Per cent of total
1	Ischaemic heart disease	151,107	11.1	Anxiety & depression	126,464	10.0
2	Type 2 diabetes	71,176	5.2	Ischaemic heart disease	112,390	8.9
3	Anxiety & depression	65,321	4.8	Stroke	65,166	5.1
4	Lung cancer	55,028	4.0	Type 2 diabetes	61,763	4.9
5	Stroke	53,296	3.9	Dementia	60,747	4.8
6	COPD	49,201	3.6	Breast cancer	60,520	4.8
7	Adult-onset hearing loss	42,653	3.1	COPD	37,550	3.0
8	Suicide & self-inflicted injuries	38,717	2.8	Lung cancer	33,876	2.7
9	Prostate cancer	36,547	2.7	Asthma	33,828	2.7
10	Colorectal cancer	34,643	2.5	Colorectal cancer	28,962	2.3
11	Dementia	33,653	2.5	Adult-onset hearing loss	22,200	1.8
12	Road traffic accidents	31,028	2.3	Osteoarthritis	20,083	1.6
13	Asthma	29,271	2.1	Personality disorders	16,339	1.3
14	Alcohol abuse	27,225	2.0	Migraine	15,875	1.3
15	Personality disorders	16,248	1.2	Back pain	15,188	1.2
16	Schizophrenia	14,785	1.1	Lower respiratory tract infections	14,233	1.1
17	Osteoarthritis	14,495	1.1	Falls	13,269	1.0
18	Back pain	14,470	1.1	Parkinson's disease	13,189	1.0
19	Melanoma	13,734	1.0	Schizophrenia	12,717	1.0
20	Parkinson's disease	13,664	1.0	Rheumatoid arthritis	12,062	1.0

Table 3.2 compares burden by broad cause groups in 2003 with total health system expenditures in 2000–01. This table is included to illustrate a misunderstanding about the relationship between health expenditure and health outcomes. It is sometimes argued, for example, that the proportion of total expenditure that is committed to a particular health problem should be commensurate in some way to its contribution to total burden. This is not necessarily the case.

Burden estimates describe the health problems that remain in a population in spite of all currently implemented prevention and treatment strategies. Large expenditure for a cause with a small burden is money well spent if that expenditure reflects an efficient health service response to what otherwise would have been a much larger problem. Oral conditions, for example, account for only 0.9% of total burden but consume 6.7% of total expenditure (\$3.4 billion). This commitment of resources may well represent a good investment if it keeps the burden from oral conditions at low levels and that without it, the burden would be much higher. If, on the other hand, some of this expenditure is not impacting on the burden, because it is being directed towards cosmetic or ineffective services, for example, or there are inefficiencies in the delivery of oral health services, then the conclusion may be less sanguine.

The real test of whether an investment has been worthwhile depends on the change in burden resulting from the expenditure as well as the opportunity cost of that expenditure to investments in other areas of the health sector. Exploring this requires information on the

effectiveness and costs associated with all current prevention and treatment strategies. Such analyses are beyond the scope of this report.

The proportion of burden for a particular health problem vis-a-vis expenditure, therefore, is more appropriately used as one argument amongst others for prioritising research into the development of new treatment and preventive interventions, and into assessing the effectiveness of these interventions. It should not be used to prioritise existing treatment and preventive activities.

Table 3.2: Burden (DALYs) in 2003 and expenditure in 2000–01 by broad cause group, Australia

Cause	DALYs in 2003		Expenditure in 2000–01 ^(a)	
	No. (thousands)	Per cent	\$ (millions)	Per cent
Neoplasms ^(b)	510.3	19.4	2,918	5.8
Cardiovascular disease	473.8	18.0	5,479	10.9
Mental disorders	350.5	13.3	3,741	7.5
Neurological & sense disorders	312.8	11.9	4,942	9.9
Respiratory disease ^(c)	222.2	8.4	3,742	7.5
Injuries	185.1	7.0	4,013	8.0
Diabetes mellitus	143.8	5.5	812	1.6
Musculoskeletal diseases	105.5	4.0	4,634	9.2
Genitourinary diseases	65.2	2.5	2,076	4.1
Diseases of the digestive system	58.0	2.2	2,811	5.6
Infectious & parasitic diseases	44.7	1.7	1,224	2.4
Neonatal causes	34.6	1.3	358	0.7
Congenital anomalies	33.2	1.3	221	0.4
Endocrine & metabolic disorders	28.6	1.1	1,587	3.2
Oral conditions	24.5	0.9	3,372	6.7
Skin diseases	20.3	0.8	1,370	2.7
Maternal conditions	2.2	0.1	1,315	2.6
Other ^(d)	17.5	0.7	5,530	11.0
All causes	2,632.8	100.0	50,146	100.0

(a) Total health system expenditures from AIHW 2005c.

(b) Includes cancers (malignant neoplasms) and other (non-malignant) neoplasms.

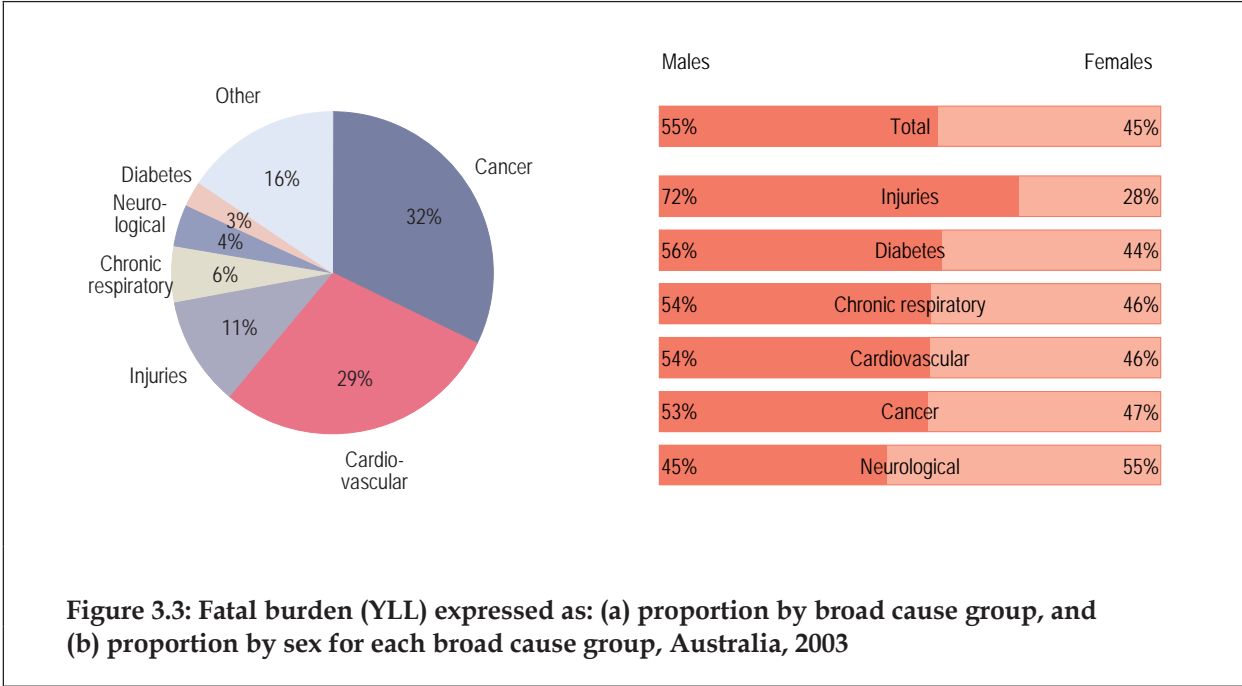
(c) Includes chronic respiratory disease and acute respiratory infections.

(d) Includes 'Signs, symptoms and ill-defined conditions' which includes expenditure on diagnostic and other services for signs, symptoms and ill-defined conditions where the cause of the problem is unknown and includes 'other contact with the health system' such as fertility control, reproduction and development, elective plastic surgery, general prevention, screening and health examination; and treatment and aftercare for unspecified disease.

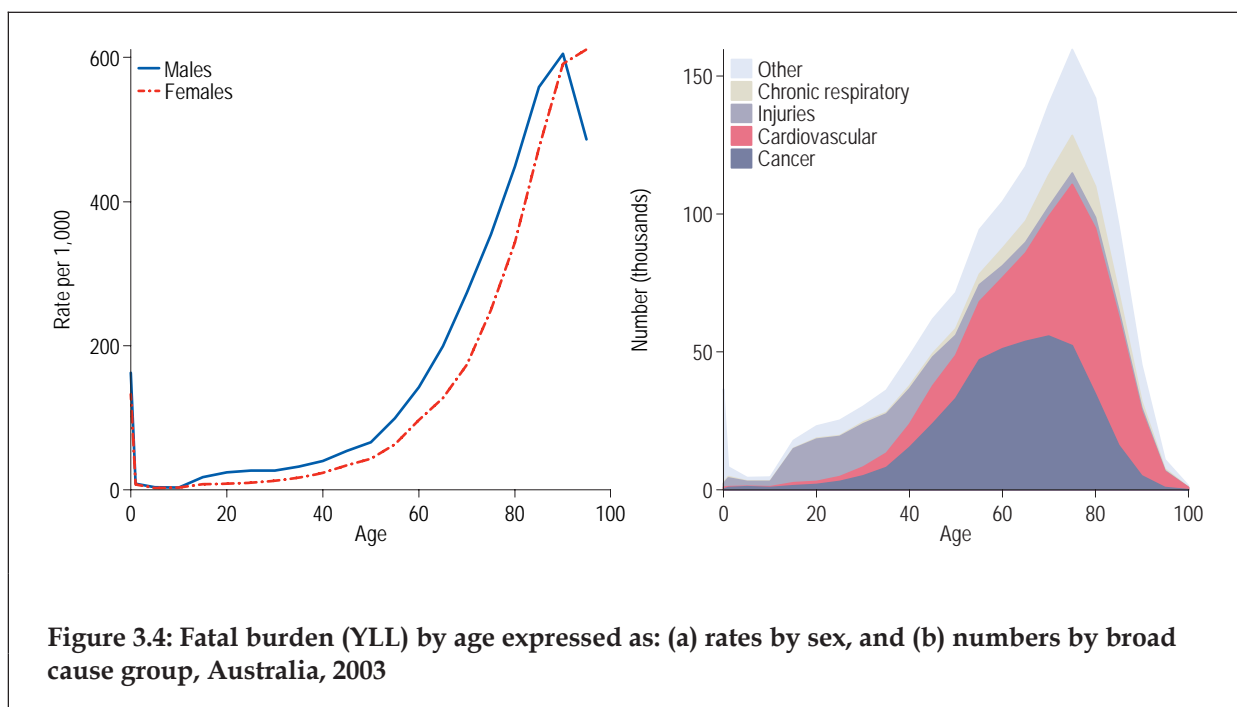
3.2 Years of life lost

Years of life lost (YLL), or fatal burden, accounted for 49% of the total burden of disease and injury in Australia in 2003 (Figure 3.1c). Cancers, cardiovascular disease and injuries were responsible for almost three-quarters of this burden (Figure 3.3a). Since the 1996 study, cancer has overtaken cardiovascular disease as the greatest cause of fatal burden as

cardiovascular mortality has declined much more than cancer mortality over the past three to four decades. Males experienced 55% of the total fatal burden.



As with total burden, fatal burden increased in absolute terms at a relatively constant rate until age 75 (Figure 3.4b), while the burden per head of population continued to increase exponentially, with small but important peaks in childhood and, in males, early adulthood (Figure 3.4a). Injury was the main cause of fatal burden until age 35 and accounted for the majority of fatal burden in early life, after which cancer and cardiovascular disease were more prominent. The contribution from cancer peaked at age 70 then declined, leaving cardiovascular disease as the major cause of fatal burden in the elderly (Figure 3.4b).



Again, the fatal burden due to specific disease and injury categories reflected the more general picture at the broad cause group level. Ischaemic heart disease was the disease contributing most to YLL in both males and females. Stroke was the second largest disease causing YLL in females, followed by breast cancer and lung cancer. In males, lung cancer ranked second, followed by suicide & self-inflicted injuries, stroke, and colorectal cancer (Table 3.3).

Table 3.3: Leading causes of mortality burden (YLL) by sex, Australia, 2003

Rank	Males	Per cent		Females	Per cent	
		YLL	of total		YLL	of total
1	Ischaemic heart disease	128,991	18.2	Ischaemic heart disease	89,152	15.7
2	Lung cancer	51,505	7.3	Stroke	48,548	8.5
3	Suicide & self-inflicted injuries	38,434	5.4	Breast cancer	40,080	7.0
4	Stroke	36,152	5.1	Lung cancer	31,551	5.5
5	Colorectal cancer	27,997	3.9	Colorectal cancer	23,735	4.2
6	Road traffic accidents	26,674	3.8	COPD	21,025	3.7
7	COPD	26,183	3.7	Dementia	16,009	2.8
8	Prostate cancer	23,175	3.3	Lower respiratory tract infections	12,309	2.2
9	Type 2 diabetes	15,273	2.2	Type 2 diabetes	11,751	2.1
10	Hepatitis	12,524	1.8	Pancreas cancer	10,984	1.9
11	Alcohol abuse	11,449	1.6	Ovary cancer	10,946	1.9
12	Lower respiratory tract infections	11,221	1.6	Suicide & self-inflicted injuries	10,945	1.9
13	Pancreas cancer	11,136	1.6	Road traffic accidents	9,678	1.7
14	Brain cancer	10,718	1.5	Nephritis & nephrosis	9,521	1.7
15	Lymphoma	10,474	1.5	Lymphoma	8,324	1.5
16	Melanoma	10,108	1.4	Brain cancer	7,809	1.4
17	Leukaemia	10,039	1.4	Leukaemia	7,468	1.3
18	Oesophagus cancer	9,427	1.3	Hepatitis	6,534	1.1
19	Nephritis & nephrosis	9,336	1.3	Falls	5,845	1.0
20	Stomach cancer	8,209	1.2	Stomach cancer	5,609	1.0

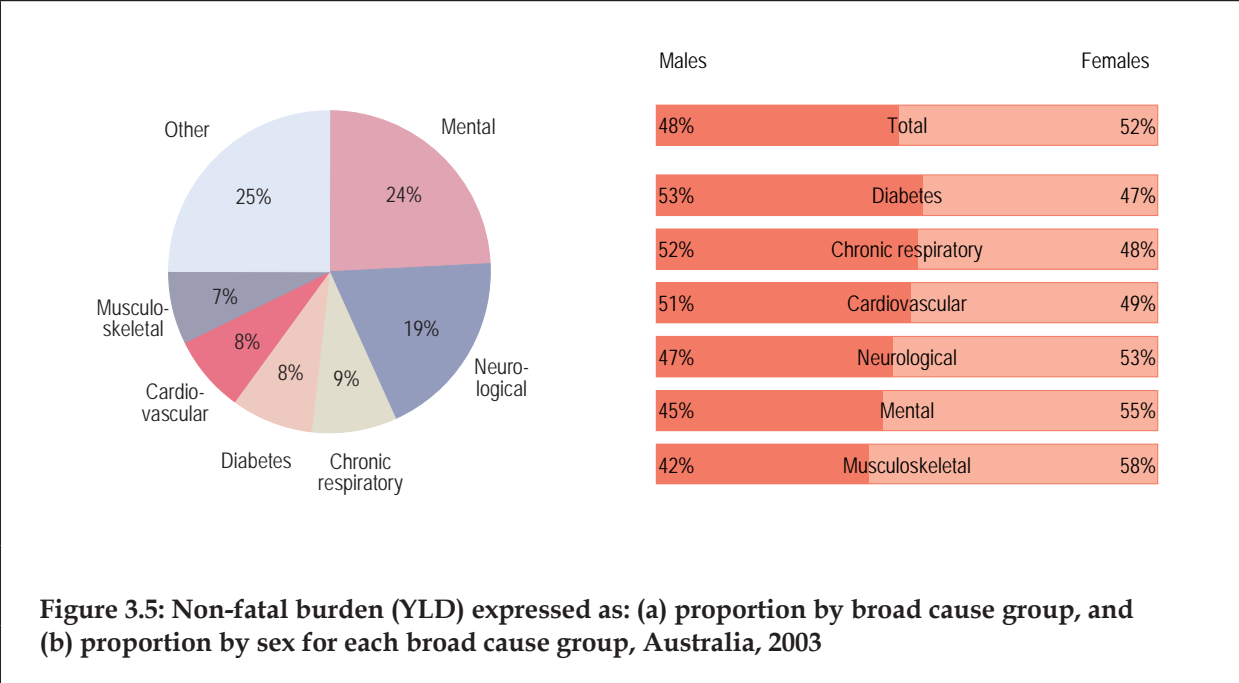
3.3 Years lost due to disability

Years lost due to disability (YLD), or non-fatal burden, are typically calculated from incidence cases in a base year and as such are to be interpreted as the number of healthy years lost due to disability that will accrue into the future from new cases of disease and injury in that base year. Incident non-fatal burden is added to fatal burden (YLL) to derive total burden (DALYs). An alternative way of calculating non-fatal burden uses prevalent cases as the basis. Prevalent non-fatal burden (PYLD) is to be interpreted as the number of healthy years lost due to disability currently experienced by a population. This cannot be added to fatal burden to derive total burden in the same way as incident non-fatal burden. Both methods of calculating non-fatal burden are presented below. For all the other sections of this report, references to non-fatal burden reflect incident non-fatal burden unless otherwise specified.

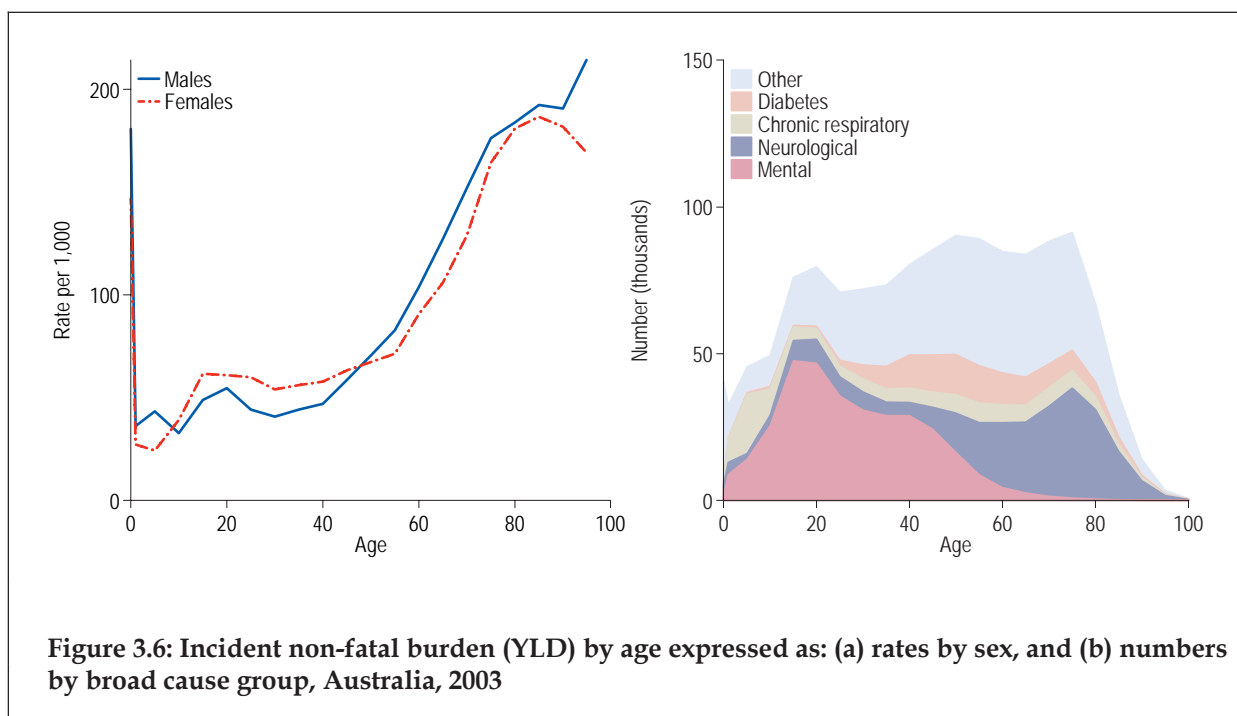
Incident YLD

Incident non-fatal burden accounted for 51% of the total burden of disease and injury in Australia in 2003 (Figure 3.1c). Mental, neurological and sense disorders contributed most,

together accounting for 43% of this burden (Figure 3.5a). While cancer, cardiovascular disease and injuries contributed 72% to the total fatal burden (Figure 3.3a), these causes did not make a similar contribution to incident non-fatal burden. Incident non-fatal burden was distributed equally between the sexes (Figure 3.5b).



Incident non-fatal burden increased rapidly in absolute terms until early adulthood then levelled out, while the rate per head of population continued increasing, but at a slower rate than for fatal burden (Figure 3.6). Mental disorders were the main causes of incident non-fatal burden until middle age and accounted for the majority of fatal burden in early life, after which neurological & sense disorders were more prominent, accounting for the majority of non-fatal burden in the elderly. Chronic respiratory conditions accounted for a small but consistent proportion of incident non-fatal burden, with peaks in childhood due to asthma and at older ages from chronic obstructive pulmonary disease (Figure 3.6).



Anxiety & depression and Type 2 diabetes were the leading causes of incident non-fatal burden in males and females (Table 3.4). Dementia was the third leading cause in females, followed by asthma and ischaemic heart disease. In males, adult-onset hearing loss ranked third, followed by asthma. Mental disorders accounted for six of the 20 leading causes of incident non-fatal burden in males and three in females.

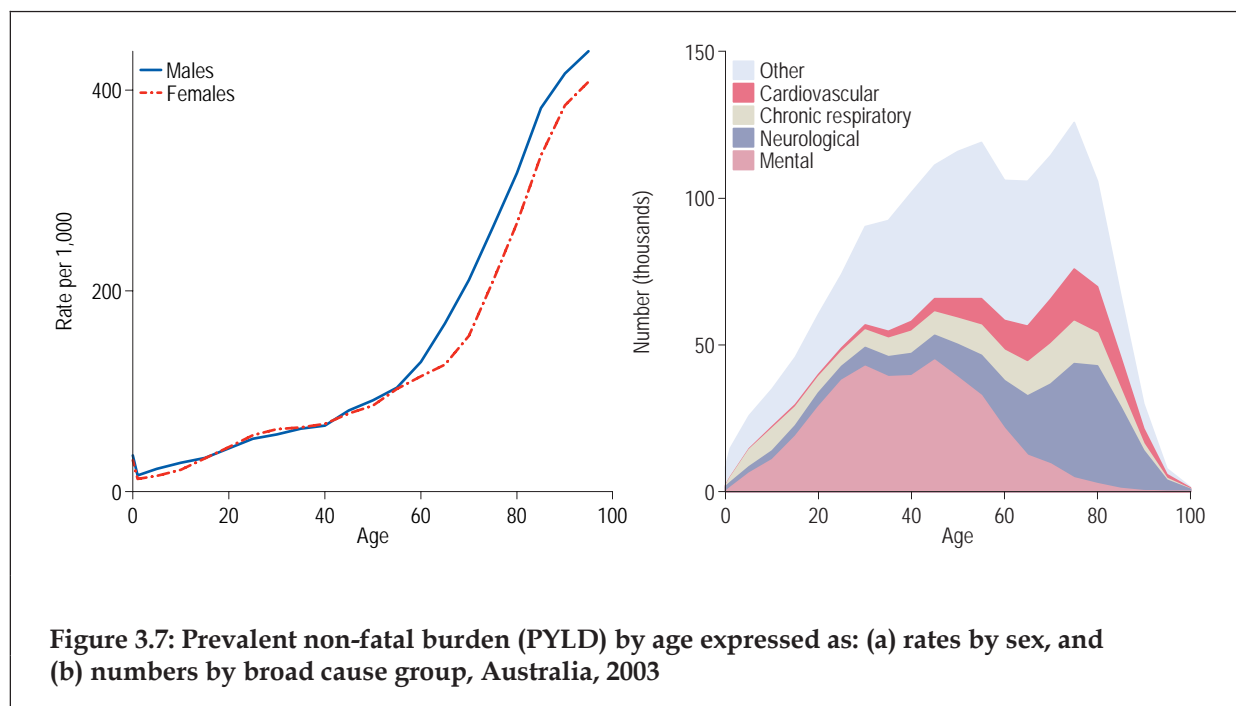
Table 3.4: Leading causes of incident non-fatal burden (YLD) by sex, Australia, 2003

Rank	Males	YLD	Per cent of total	Females	YLD	Per cent of total
1	Anxiety & depression	65,208	10.0	Anxiety & depression	126,244	18.1
2	Type 2 diabetes	55,903	8.5	Type 2 diabetes	50,012	7.2
3	Adult-onset hearing loss	42,653	6.5	Dementia	44,738	6.4
4	Asthma	27,649	4.2	Asthma	31,405	4.5
5	Dementia	25,558	3.9	Ischaemic heart disease	23,238	3.3
6	COPD	23,018	3.5	Adult-onset hearing loss	22,200	3.2
7	Ischaemic heart disease	22,116	3.4	Breast cancer	20,440	2.9
8	Stroke	17,144	2.6	Osteoarthritis	19,775	2.8
9	Personality disorders	16,248	2.5	Stroke	16,619	2.4
10	Alcohol abuse	15,775	2.4	COPD	16,525	2.4
11	Schizophrenia	14,673	2.2	Personality disorders	16,339	2.3
12	Osteoarthritis	14,429	2.2	Migraine	15,868	2.3
13	Back pain	14,355	2.2	Back pain	15,129	2.2
14	Prostate cancer	13,372	2.0	Schizophrenia	12,577	1.8
15	Autism spectrum disorders	11,702	1.8	Rheumatoid arthritis	10,918	1.6
16	Parkinson's disease	10,623	1.6	Parkinson's disease	10,534	1.5
17	Refractive errors	8,241	1.3	Refractive errors	10,520	1.5
18	Peripheral vascular disease	7,965	1.2	Infertility	8,076	1.2
19	Heroin or polydrug abuse	7,498	1.1	Falls	7,424	1.1
20	Benign prostatic hypertrophy	7,378	1.1	Macular degeneration	7,259	1.0

Prevalent YLD

Figure 3.7 illustrates the prevalent non-fatal burden by age. The difference between prevalent and incident non-fatal burden is most apparent for childhood conditions, such as asthma and congenital disorders, and for chronic mental disorders, the incidence of which peaks in childhood and early adulthood. Incident non-fatal burden at these life stages is much larger compared to prevalent non-fatal burden because most incident cases of chronic conditions at young ages are expected to remain prevalent cases at older ages. This explains the shift to the right in the picture of prevalent non-fatal burden (Figure 3.7) compared to incident non-fatal burden (Figure 3.6).

The rate of prevalent burden was lowest in children between 1 and 4 years of age (15 PYLD per thousand) and increased to 147 in people aged 65 to 69 years and then to 415 per thousand in people over the age of 95. In other words, disability from all diseases and injuries resulted in a loss of 1.5% of healthy time lived by young children, increasing with age to 14.7% in those 65 to 69 years and 41.5% in the very old.



3.4 Age and sex patterns

In this section, the size and composition of burden is reported by five broad age groups (Table 3.5).

Table 3.5: Distribution of population and burden (DALYs) by five broad age groups, Australia, 2003

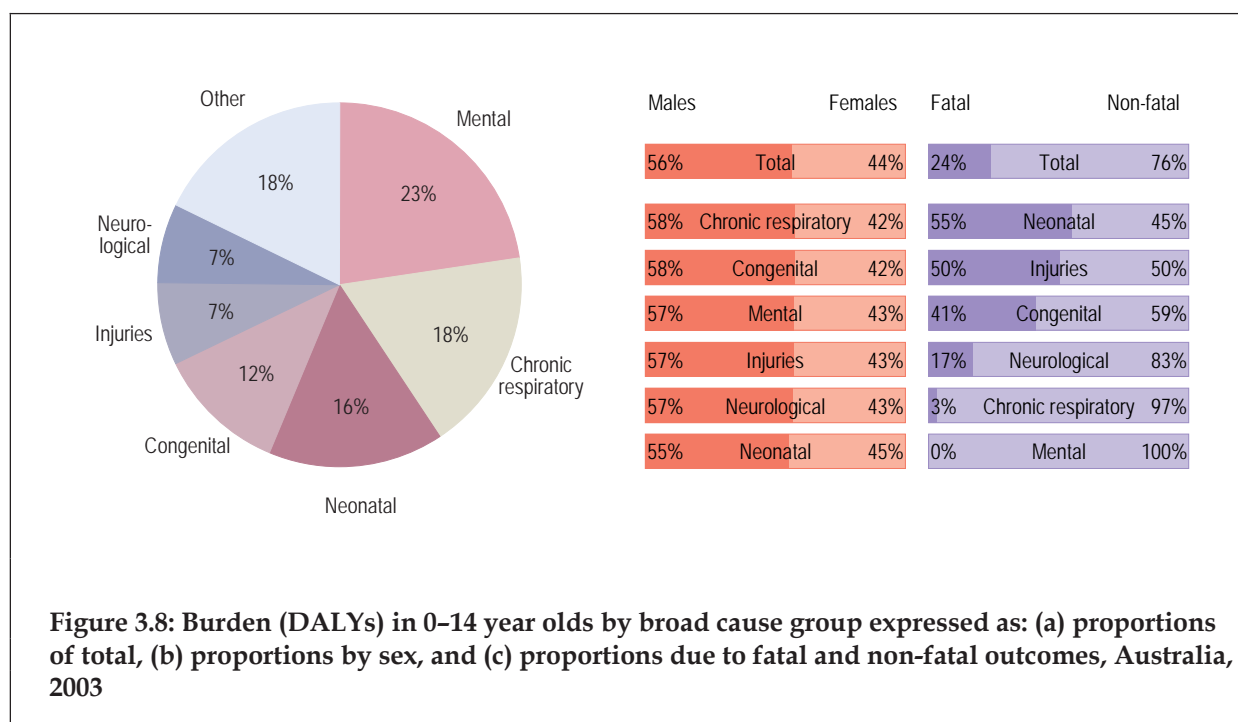
Age group	Population ^(a)	Per cent of total	DALYs	Per cent of total
0–14 years	3,979,410	20.0	221,536	8.4
15–44 years	8,622,610	43.4	633,260	24.1
45–64 years	4,733,808	23.8	681,566	25.9
65–74 years	1,349,949	6.8	428,904	16.3
75 years and over	1,195,692	6.0	667,504	25.4
Total	19,881,469	100.0	2,632,770	100.0

(a) Estimated resident population figures as at 30 June 2003 (ABS cat. no. 3201.0).

Children aged 0–14 years

Children aged 0–14 years comprised 20.0% of the total population and experienced 8.4% of the total burden of disease and injury in Australia in 2003 (Table 3.5). Twenty-three per cent of this burden was due to mental disorders (that is anxiety & depression, attention-deficit hyperactivity disorder and autism spectrum disorders), 18% due to chronic respiratory conditions (mostly asthma) and 16% due to neonatal conditions. Less than a quarter of the

burden was due to mortality (Figure 3.8). Males experienced 56% of the burden in this age group.



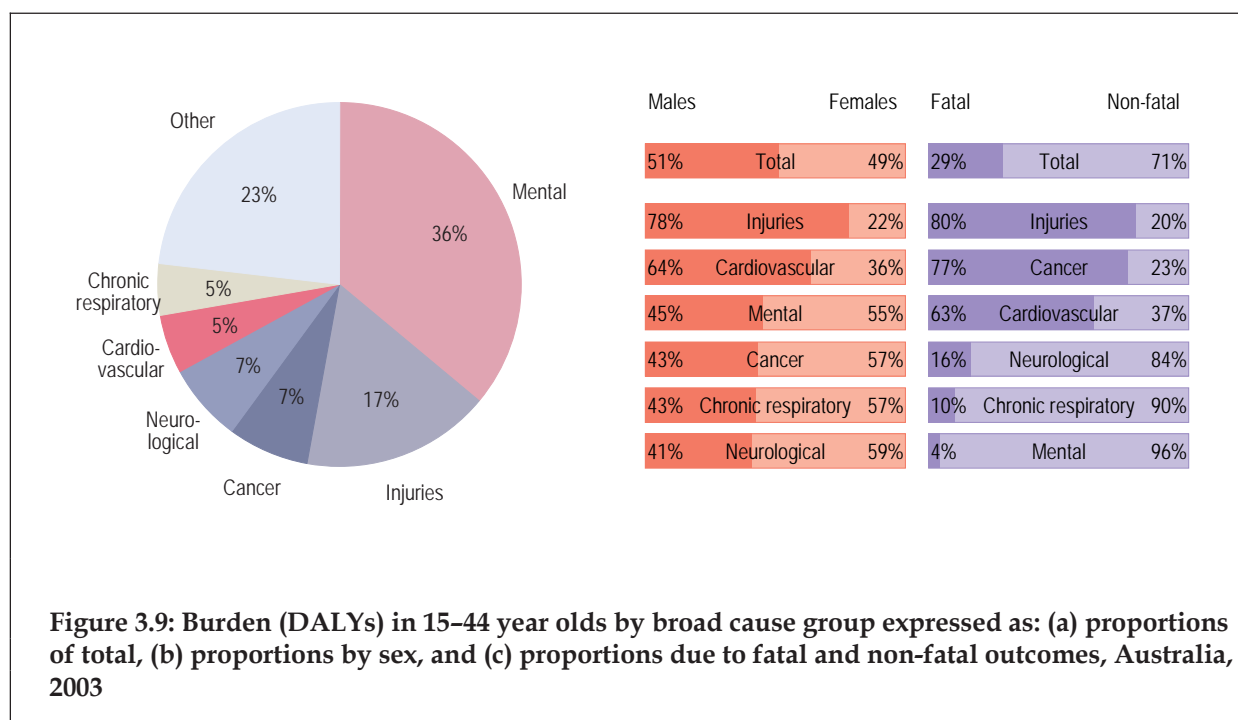
Asthma was the leading cause of burden for both males and females (Table 3.6). This was followed by autism spectrum disorders, anxiety & depression, and low birth weight in males. In females, anxiety & depression, low birth weight and birth trauma & asphyxia were the next leading causes. The leading 10 causes of burden accounted for 58.7% of the total burden in this age group.

Table 3.6: Leading causes of DALYs in 0–14 year olds by sex, Australia, 2003

Rank	Males	DALYs	Per cent of total	Females	DALYs	Per cent of total
1	Asthma	21,953	17.6	Asthma	16,490	17.0
2	Autism spectrum disorders	11,703	9.4	Anxiety & depression	15,507	16.0
3	Anxiety & depression	9,554	7.7	Low birth weight	7,142	7.4
4	Low birth weight	8,281	6.6	Birth trauma & asphyxia	4,221	4.4
5	Attention-deficit hyperactivity disorder	7,082	5.7	Attention-deficit hyperactivity disorder	2,840	2.9
6	Birth trauma and asphyxia	5,086	4.1	Epilepsy	2,446	2.5
7	Congenital heart disease	3,434	2.8	Congenital heart disease	2,202	2.3
8	Epilepsy	3,249	2.6	Autism spectrum disorders	2,056	2.1
9	Neonatal infections	2,156	1.7	Otitis media	1,377	1.4
10	Road traffic accidents	1,991	1.6	Road traffic accidents	1,336	1.4

Older children and adults aged 15–44 years

Older children and adults aged 15–44 years comprised 43.4% of the total population and experienced 24.1% of the total burden of disease and injury in Australia in 2003 (Table 3.5). Over a third of the total burden in this age group was attributable to mental disorders, and another 17% was due to injuries (Figure 3.9). There were considerable sex differences in this age group, with females experiencing a greater share of the burden from neurological disorders, chronic respiratory diseases, cancers and mental disorders than males. Males, on the other hand, experienced more than three-quarters of the injury burden, partly because of their greater inclination for risk taking. Overall, 29% of the burden in this age group was due to mortality.



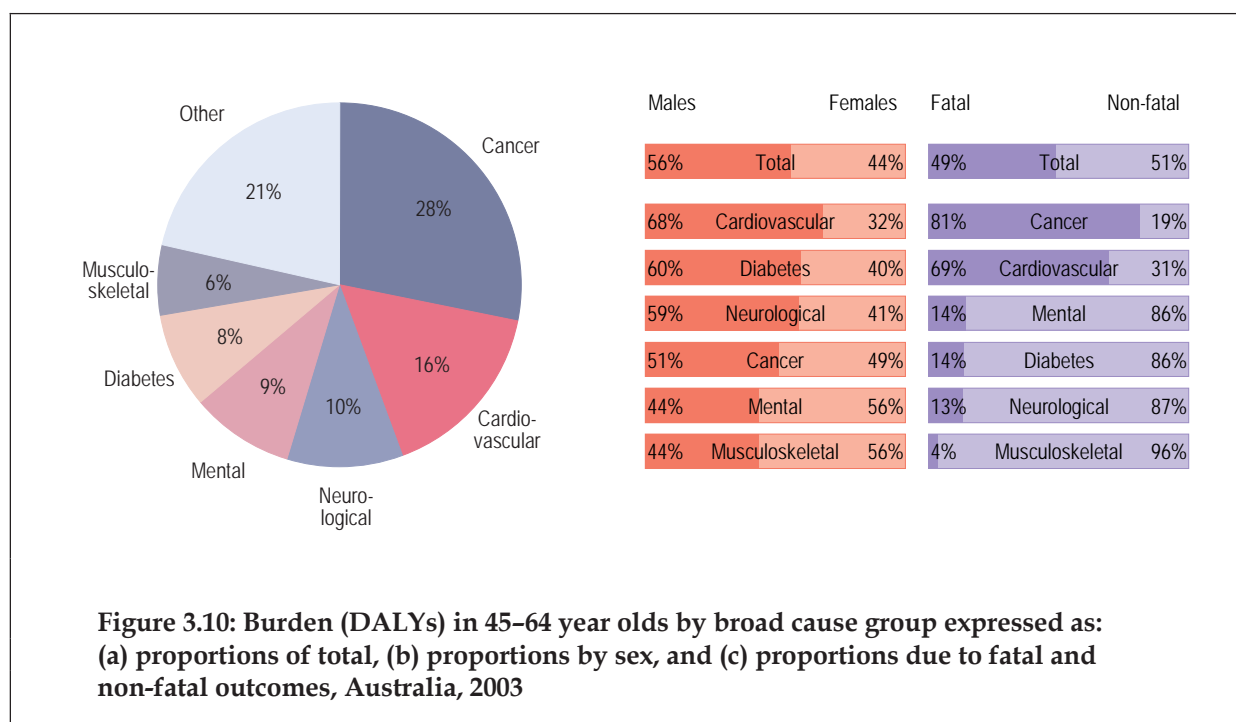
Anxiety & depression was by far the leading single cause of burden in both males and females, followed by suicide & self-inflicted injuries and road traffic accidents in males, and migraine and Type 2 diabetes in females (Table 3.7). Mental disorders made up half of the top 10 leading causes of burden in males and three of the top 10 leading causes of burden in females. The leading 10 ranked conditions accounted for 54.8% of the burden in this age group.

Table 3.7: Leading causes of DALYs in 15–44 year olds by sex, Australia, 2003

Rank	Males	DALYs	Per cent of total	Females	DALYs	Per cent of total
1	Anxiety & depression	42,237	13.0	Anxiety & depression	84,717	27.4
2	Suicide & self-inflicted injuries	27,592	8.5	Migraine	14,105	4.6
3	Road traffic accidents	22,845	7.1	Type 2 diabetes	12,487	4.0
4	Schizophrenia	14,376	4.4	Asthma	11,311	3.7
5	Alcohol abuse	13,953	4.3	Schizophrenia	11,064	3.6
6	Type 2 diabetes	12,868	4.0	Personality disorders	9,389	3.0
7	Heroin abuse	11,882	3.7	Breast cancer	9,068	2.9
8	Personality disorders	10,526	3.2	Infertility	8,057	2.6
9	Ischaemic heart disease	9,750	3.0	Suicide & self-inflicted injuries	7,174	2.3
10	COPD	6,840	2.1	Road traffic accidents	6,751	2.2

Adults aged 45–64 years

Adults aged 45–64 years comprised 23.8% of the total population and experienced 25.9% of the total burden of disease and injury in Australia in 2003 (Table 3.5). Cancer, cardiovascular disease and neurological disorders accounted for more than half of the total burden in this age group. Males experienced a greater share of the burden than females for all causes except mental disorders and musculoskeletal disorders (Figure 3.10). Overall, 49% of the burden in this age group was due to mortality.



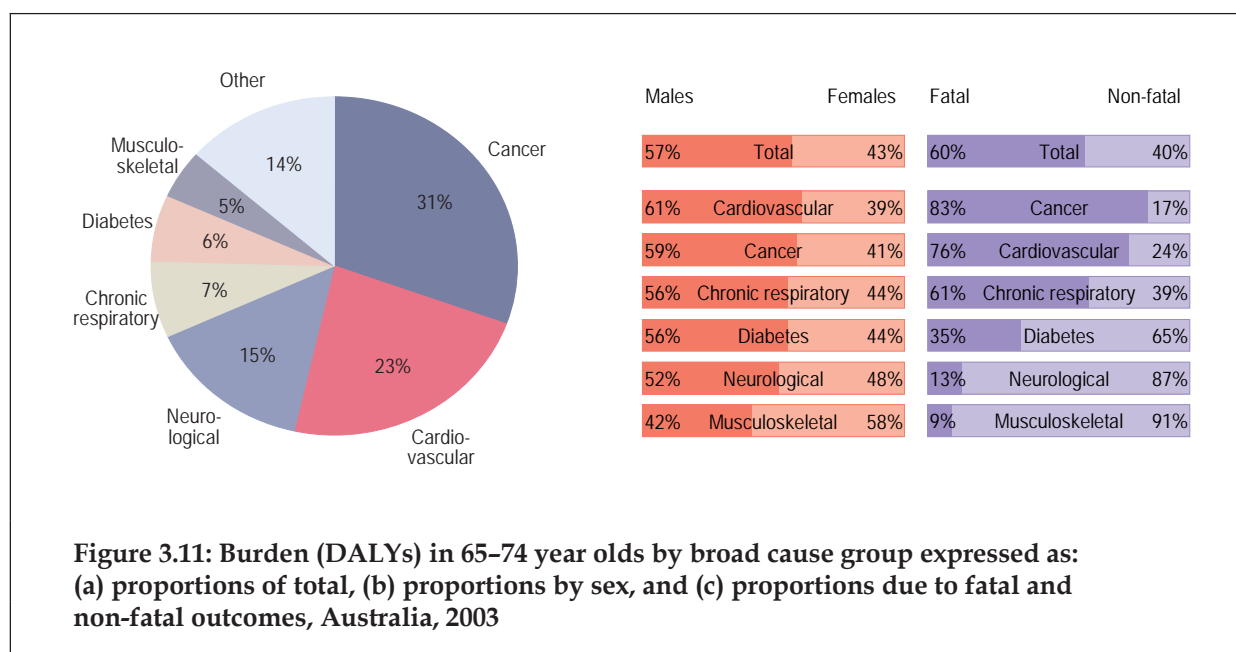
Ischaemic heart disease was the leading cause of burden in males, followed by Type 2 diabetes and lung cancer (Table 3.8). In females, the top three causes were breast cancer, anxiety & depression and Type 2 diabetes. The top 10 conditions accounted for 52.0% of total burden in this age group.

Table 3.8: Leading causes of DALYs in 45–64 year olds by sex, Australia, 2003

Rank	Males	DALYs	Per cent of total	Females	DALYs	Per cent of total
1	Ischaemic heart disease	47,782	12.5	Breast cancer	32,012	10.7
2	Type 2 diabetes	32,741	8.6	Anxiety & depression	25,744	8.6
3	Lung cancer	20,861	5.5	Type 2 diabetes	22,299	7.5
4	Adult-onset hearing loss	20,847	5.5	Ischaemic heart disease	17,489	5.8
5	COPD	15,389	4.0	Lung cancer	13,475	4.5
6	Colorectal cancer	14,130	3.7	Adult-onset hearing loss	10,576	3.5
7	Stroke	13,800	3.6	COPD	10,422	3.5
8	Anxiety & depression	11,757	3.1	Colorectal cancer	9,808	3.3
9	Alcohol abuse	10,077	2.6	Stroke	9,693	3.2
10	Prostate cancer	8,953	2.3	Back pain	6,620	2.2

Adults aged 65–74 years

Adults aged 65–74 years comprised 6.8% of the total population and experienced 16.3% of the total burden of disease and injury in Australia in 2003 (Table 3.5). Cancer and cardiovascular disease accounted for over half of the total burden in this age group (Figure 3.11). Females experienced a greater share of the burden than males from musculoskeletal conditions, while the reverse was true for all other broad cause groups. Overall, 60% of the burden in this age group was due to mortality.



Ischaemic heart disease, lung cancer and Type 2 diabetes were the leading causes of burden in males, together accounting for 29% of total male burden (Table 3.9). In females, ischaemic heart disease, Type 2 diabetes and breast cancer were the leading causes, accounting for 23% of total burden. The top 10 conditions accounted for 56.3% of total burden in this age group.

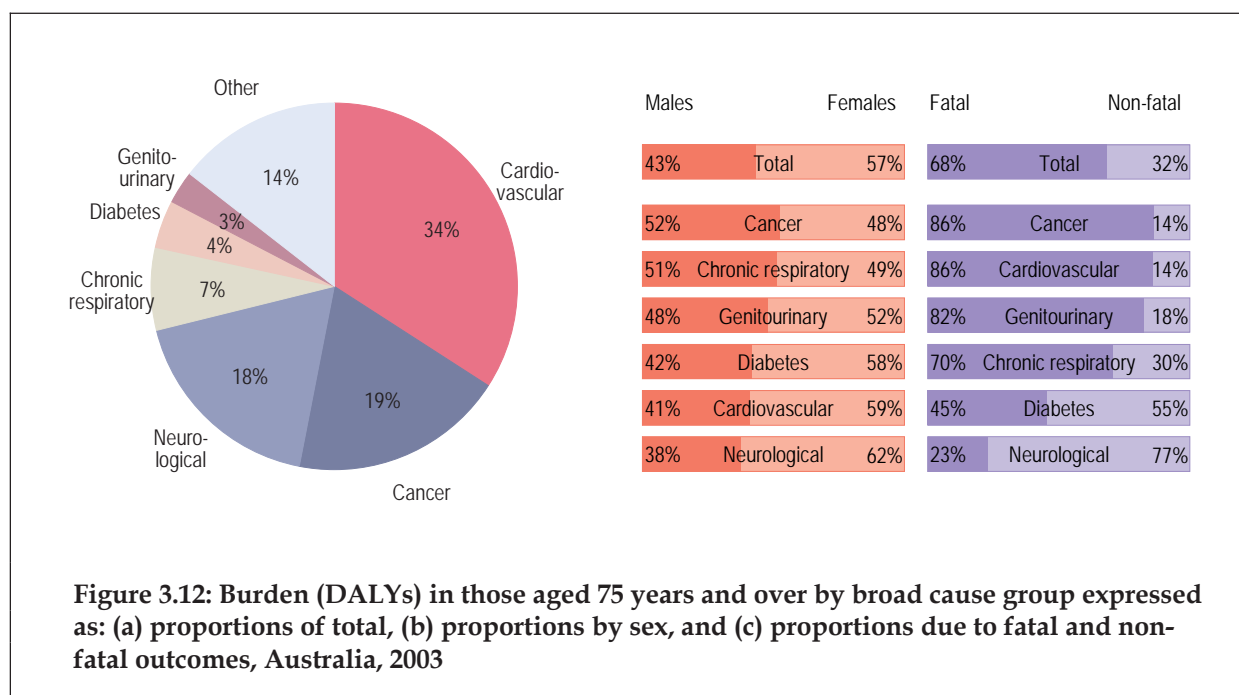
Table 3.9: Leading causes of DALYs in 65-74 year olds by sex, Australia, 2003

Rank	Males	DALYs	Per cent of total	Females	DALYs	Per cent of total
1	Ischaemic heart disease	37,860	15.5	Ischaemic heart disease	21,052	11.4
2	Lung cancer	19,258	7.9	Type 2 diabetes	11,517	6.2
3	Type 2 diabetes	14,203	5.8	Breast cancer	10,445	5.7
4	Prostate cancer	11,950	4.9	Dementia	10,236	5.5
5	Adult-onset hearing loss	11,920	4.9	Lung cancer	9,937	5.4
6	COPD	11,693	4.8	Stroke	9,635	5.2
7	Stroke	10,938	4.5	COPD	8,855	4.8
8	Colorectal cancer	10,531	4.3	Colorectal cancer	7,513	4.1
9	Dementia	7,872	3.2	Osteoarthritis	6,088	3.3
10	Parkinson's disease	3,958	1.6	Adult-onset hearing loss	5,834	3.2

Older people aged 75 years and over

Older people aged 75 years and over comprised 6.0% of the total population and experienced 25.4% of the total burden of disease and injury in Australia in 2003 (Table 3.5).

Cardiovascular disease and cancer accounted for over half of the total burden in this age group (Figure 3.12). Females experienced a greater share of the burden than males overall and for all broad cause groups except chronic respiratory diseases and cancer. Overall, 68% of the burden in this age group was due to mortality.



Ischaemic heart disease, stroke and dementia were the leading causes of burden in males, together accounting for 34% of total male burden (Table 3.10). In females, ischaemic heart disease, dementia and stroke were the leading causes, accounting for 42% of total burden. The top 10 conditions account for 60.9% of the total burden in this age group.

Table 3.10: Leading causes of DALYs in those aged 75 years and over by sex, Australia, 2003

Rank	Males	DALYs	Per cent of total	Females	DALYs	Per cent of total
1	Ischaemic heart disease	55,680	19.3	Ischaemic heart disease	70,853	18.7
2	Stroke	21,834	7.5	Dementia	46,984	12.4
3	Dementia	21,095	7.3	Stroke	39,830	10.5
4	Prostate cancer	15,484	5.4	Type 2 diabetes	15,330	4.1
5	COPD	14,900	5.2	COPD	13,318	3.5
6	Lung cancer	13,533	4.7	Colorectal cancer	9,703	2.6
7	Type 2 diabetes	11,262	3.9	Lower respiratory tract infections	9,137	2.4
8	Colorectal cancer	8,442	2.9	Lung cancer	9,059	2.4
9	Adult-onset hearing loss	7,052	2.4	Breast cancer	8,995	2.4
10	Lower respiratory tract infections	6,395	2.2	Falls	7,814	2.1

3.5 Specific disease and injury categories

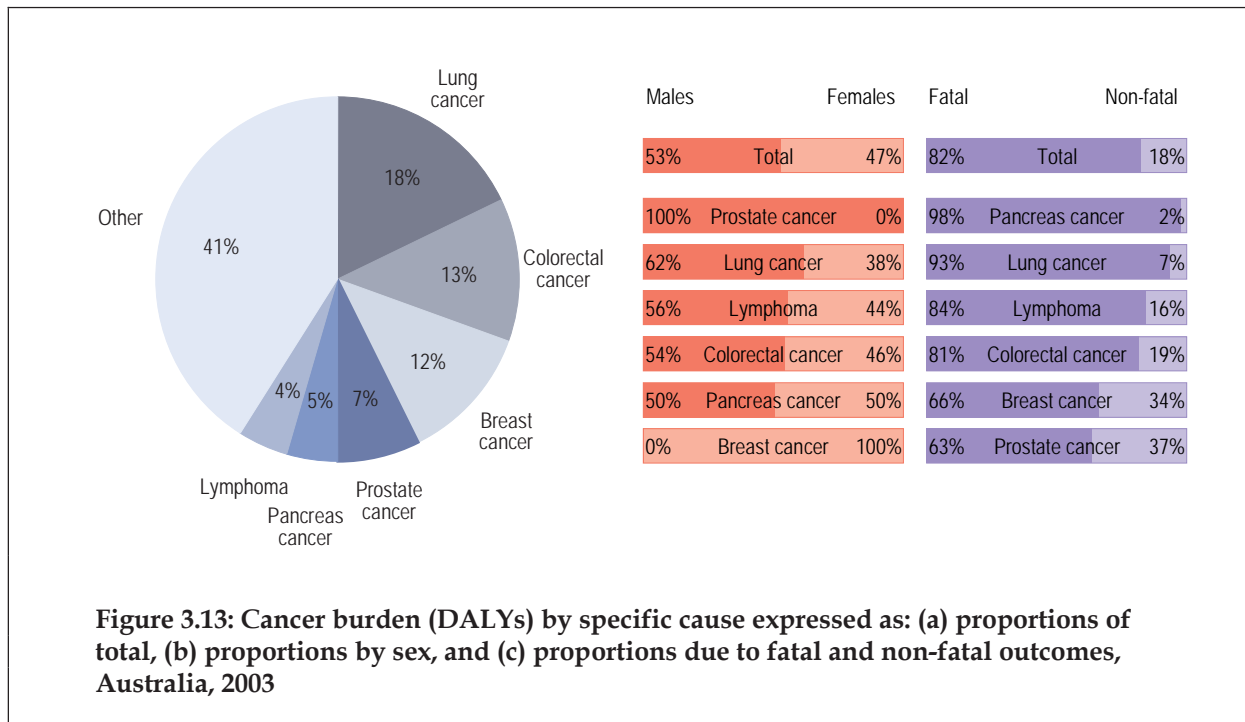
This section presents burden by 22 broad cause groupings (Table 3.11) and discusses the eight largest of these in greater detail. The section ends with a short discussion on three conditions (renal failure, vision loss and intellectual disability), the burden from which in the previous sections is split across multiple subheadings depending on aetiology. However, from a health service planning perspective there is value in presenting the aggregates for these conditions.

Table 3.11: Burden (YLD, YLL and DALYs) by broad cause group, Australia, 2003

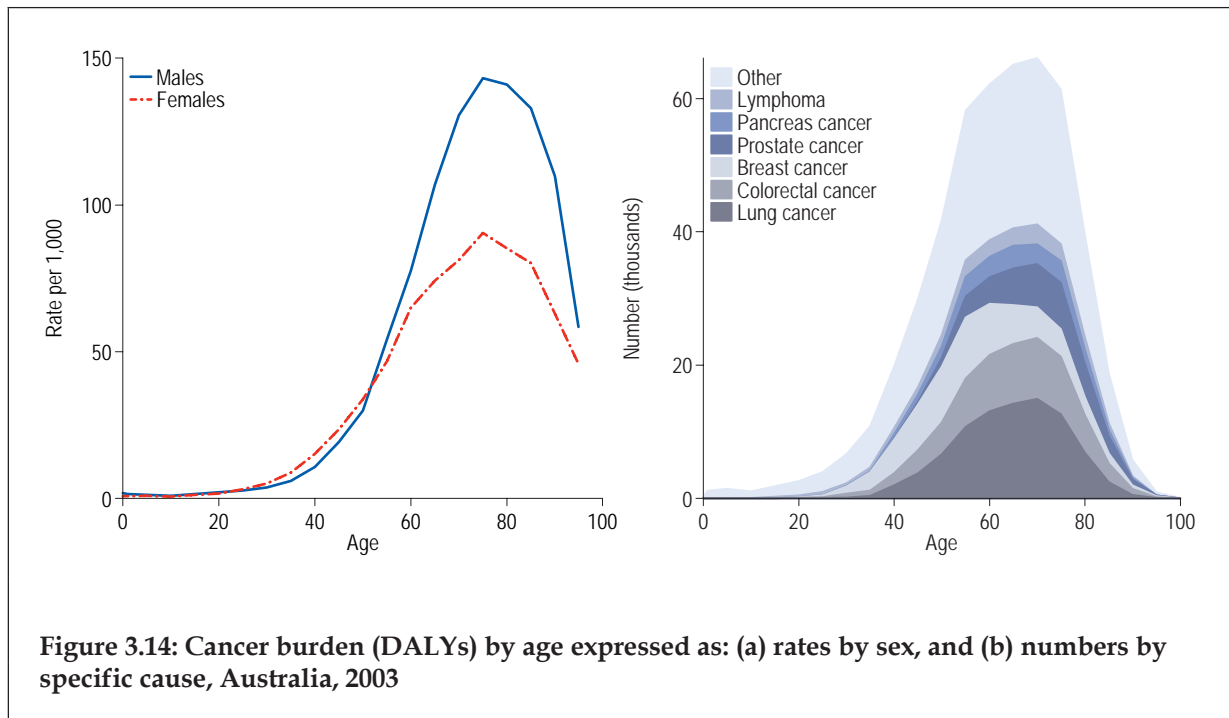
Cause	YLD	Per cent of total	YLL	Per cent of total	DALYs	Per cent of total
Cancers	87,463	6.5	411,953	32.2	499,416	19.0
Cardiovascular disease	104,429	7.7	369,365	28.9	473,794	18.0
Mental disorders	327,391	24.2	23,154	1.8	350,545	13.3
Neurological & sense disorders	258,638	19.1	54,127	4.2	312,766	11.9
Chronic respiratory diseases	115,398	8.5	71,339	5.6	186,737	7.1
Diabetes mellitus	111,536	8.2	32,295	2.5	143,831	5.5
Unintentional injuries	41,263	3.0	84,599	6.6	125,862	4.8
Musculoskeletal diseases	98,481	7.3	7,027	0.5	105,508	4.0
Genitourinary diseases	41,161	3.0	24,087	1.9	65,249	2.5
Intentional injuries	3,139	0.2	56,050	4.4	59,189	2.2
Diseases of the digestive system	30,246	2.2	27,710	2.2	57,957	2.2
Infectious & parasitic diseases	14,021	1.0	30,665	2.4	44,685	1.7
Acute respiratory infections	11,752	0.9	23,750	1.9	35,502	1.3
Neonatal causes	15,584	1.2	18,974	1.5	34,558	1.3
Congenital anomalies	16,331	1.2	16,897	1.3	33,228	1.3
Endocrine & metabolic disorders	14,968	1.1	13,598	1.1	28,565	1.1
Oral conditions	24,406	1.8	102	0.0	24,507	0.9
Skin diseases	18,130	1.3	2,173	0.2	20,302	0.8
Ill-defined conditions	8,781	0.6	2,536	0.2	11,317	0.4
Non-malignant neoplasms	3,209	0.2	7,694	0.6	10,903	0.4
Nutritional deficiencies	5,739	0.4	458	0.0	6,197	0.2
Maternal conditions	1,926	0.1	226	0.0	2,152	0.1
Total burden	1,353,992	100.0	1,278,778	100.0	2,632,770	100.0

Cancers

Cancer was responsible for 19.0% of the total burden of disease and injury in Australia in 2003 (Table 3.11), with lung, colorectal, breast and prostate cancer accounting for half of this burden (Figure 3.13). Apart from the sex-specific cancers (that is, breast, cervical and uterine cancers in females and prostate cancer in males), there were considerable sex differences in the experience of cancer burden, with males having a greater share of the burden from melanoma, colorectal cancer, lymphomas and lung cancer than females. The difference in lung cancer between males and females was largely due to the higher prevalence of smoking in males than females two or more decades ago. More than four-fifths of the total cancer burden was due to mortality.



Total cancer burden, both in absolute terms and when expressed as a rate per head of population, increased exponentially until age 75, then declined (Figure 3.14). The contribution from lung cancer was greatest at this age after which it declined in proportion to other cancers. In males, the contribution from prostate cancer increased until old age, whereas in females the contribution from breast cancer increased until age 60, then declined in proportion to other cancers. The contribution from colorectal cancer was important across all ages.



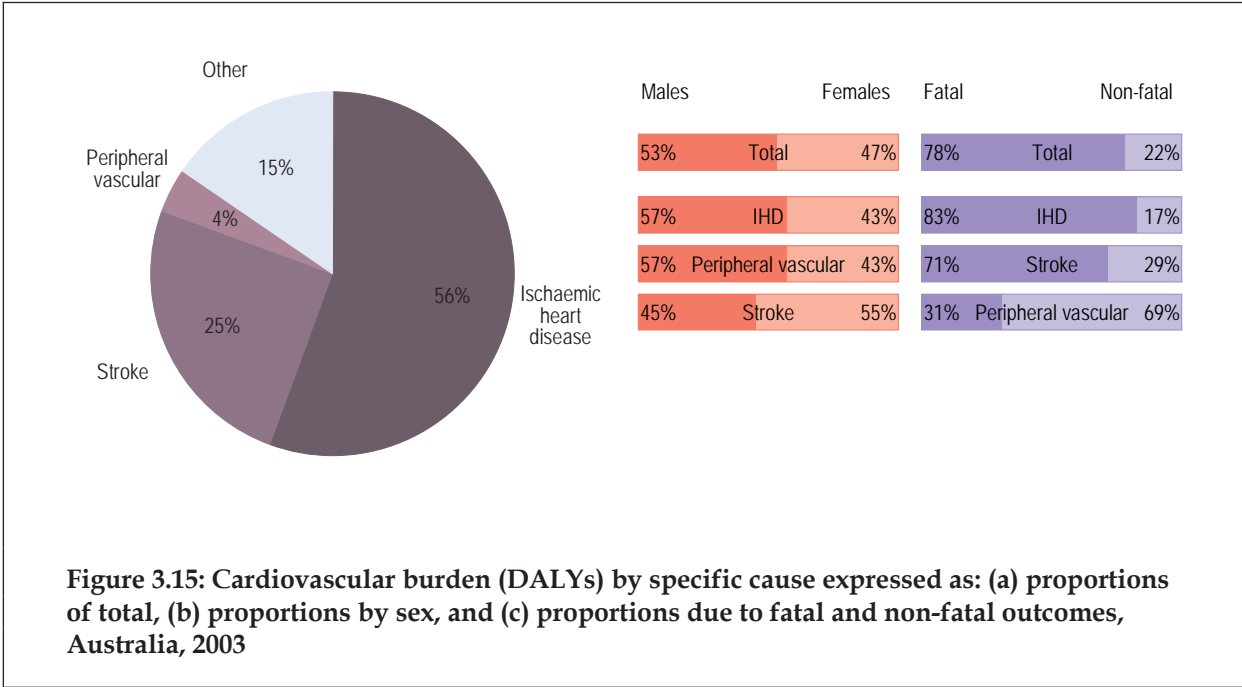
Lung cancer was the fourth leading cause of overall burden in males, while prostate and colorectal cancers were the ninth and tenth, respectively. Breast cancer was the sixth leading cause of overall burden in females, while lung cancer and colorectal cancer were eighth and tenth, respectively (Table 3.1). Although cancer of the cervix was not a leading cause of death in females, it is a cancer priority area because it is one of the few cancers where pre-cancerous lesions can and have been cost-effectively detected and treated through an organised screening program. Moreover, a vaccine against human papilloma virus has become available that has the potential to further reduce the burden of cervical cancer over time. This illustrates that burden information should be used with other evidence to determine health service priorities.

Table 3.12: Cancer burden (YLD, YLL and DALYs) by specific cause, Australia, 2003

Cause	YLD	Per cent of total	YLL	Per cent of total	DALYs	Per cent of total
Lung cancer	5,848	0.4	83,056	6.5	88,904	3.4
Colorectal cancer	11,873	0.9	51,732	4.0	63,605	2.4
Breast cancer	20,440	1.5	40,214	3.1	60,654	2.3
Prostate cancer	13,372	1.0	23,175	1.8	36,547	1.4
Pancreas cancer	561	0.0	22,119	1.7	22,680	0.9
Lymphoma	3,465	0.3	18,798	1.5	22,263	0.8
Other	31,905	2.4	172,859	13.5	204,763	7.8
Total cancer burden	87,463	6.5	411,953	32.2	499,416	19.0

Cardiovascular disease

Cardiovascular disease were responsible for 18.0% the total burden of disease and injury in Australia in 2003 (Table 3.11), with ischaemic heart disease and stroke accounting for over four-fifths of this burden (Figure 3.15). These diseases were also in the five leading causes of overall burden (Table 3.1). The contribution from ischaemic heart disease was greater in males than in females, while the reverse was the case for stroke. Nearly four-fifths of total cardiovascular burden was due to mortality.



In contrast to cancer, the total cardiovascular burden per head of population continued increasing until old age. This resulted in a larger proportion of the absolute burden at older ages than for cancer (Figure 3.16). Ischaemic heart disease dominated across all ages.

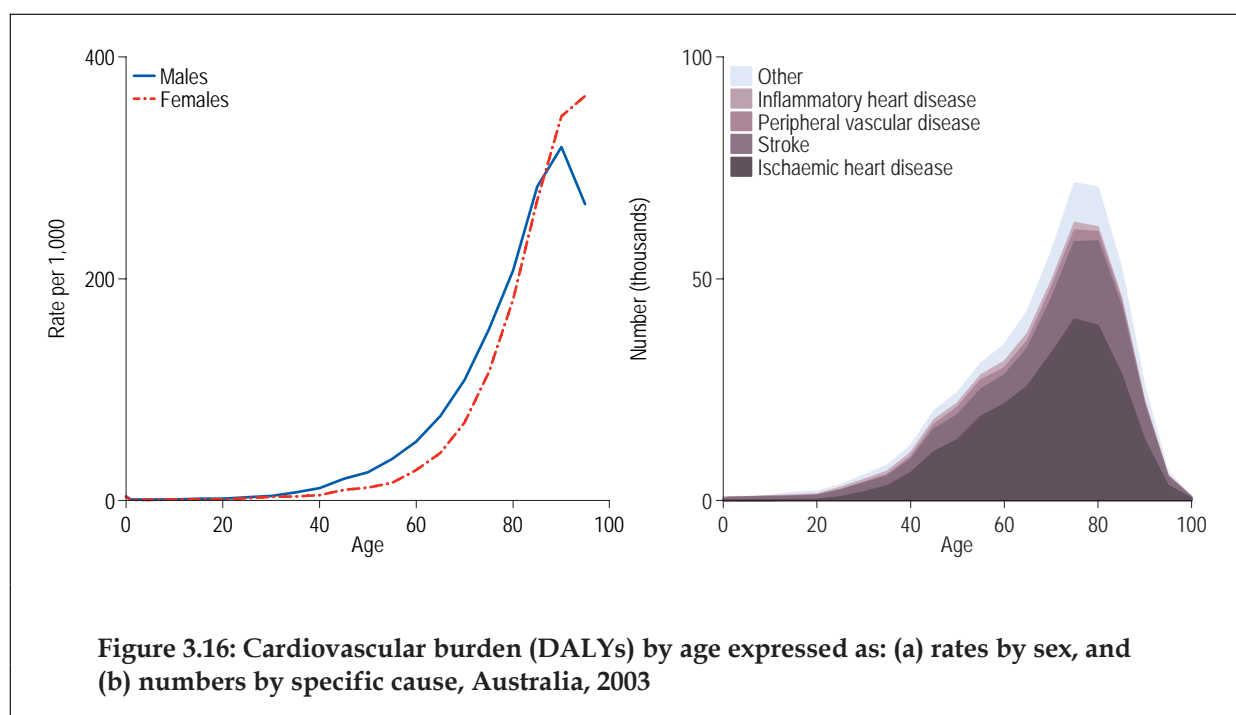


Figure 3.16: Cardiovascular burden (DALYs) by age expressed as: (a) rates by sex, and (b) numbers by specific cause, Australia, 2003

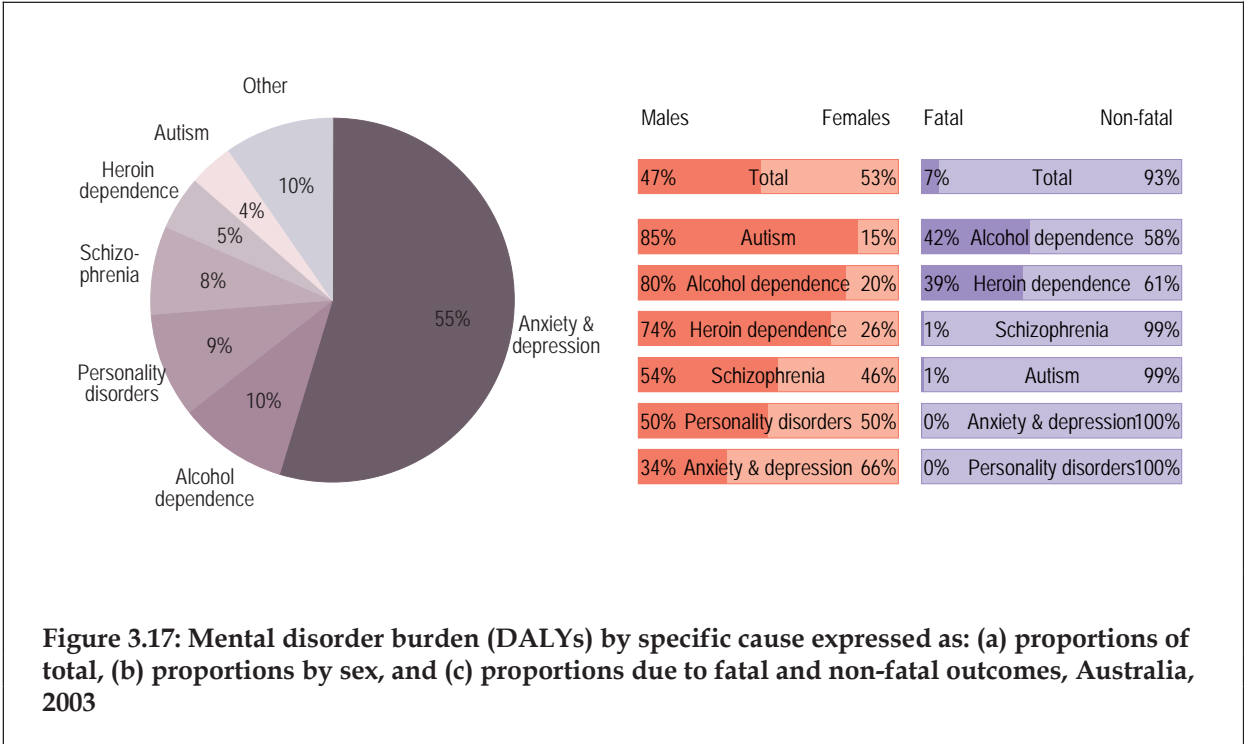
Table 3.13: Cardiovascular burden (YLD, YLL and DALYs) by specific cause, Australia, 2003

Cause	YLD	Per cent of total	YLL	Per cent of total	DALYs	Per cent of total
Ischaemic heart disease	45,354	3.3	218,143	17.1	263,497	10.0
Stroke	33,763	2.5	84,699	6.6	118,462	4.5
Peripheral vascular disease	12,888	1.0	5,718	0.4	18,606	0.7
Inflammatory heart disease	3,689	0.3	12,215	1.0	15,904	0.6
Aortic aneurysm	209	0.0	11,129	0.9	11,338	0.4
Hypertensive heart disease	678	0.1	8,303	0.6	8,982	0.3
Other	7,848	0.6	29,157	2.3	37,005	1.4
Total cardiovascular burden	104,429	7.7	369,365	28.9	473,794	18.0

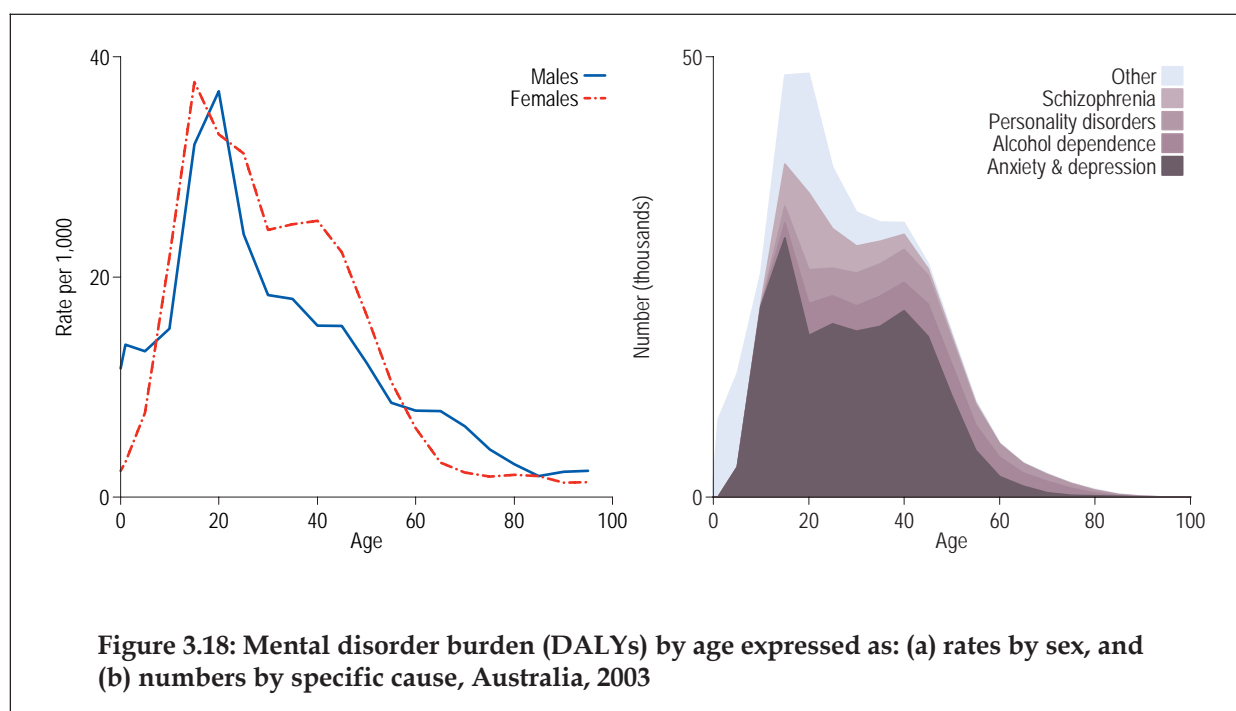
Mental disorders

Mental disorders were responsible for 13.3% of the total burden of disease and injury in Australia in 2003 (Table 3.11), with anxiety & depression, alcohol abuse and personality disorders accounting for almost three-quarters of this burden (Figure 3.17). There were marked sex differences in the mental illness burden for particular disorders. The burden from anxiety & depression was twice as high for females as for males. Conversely, the burden from substance abuse was more than three times as high in males as in females. Eating disorders occurred mainly in females. Autism spectrum disorders were much more common in males, with females having just 15% of the total burden from these conditions.

Seven per cent of the burden from mental disorders was due to mortality, most of which was accounted for by fatal outcomes associated with substance abuse.



The burden from mental disorders both in absolute terms and when expressed as a rate per head of population was greater in early adulthood than at other ages (Figure 3.18). This was partly due to the peak in new cases of chronic mental illnesses at this life stage, the burden of which was experienced throughout adult life. Anxiety & depression contributed most until age 60, after which the contribution from alcohol abuse and personality disorders was more prominent.



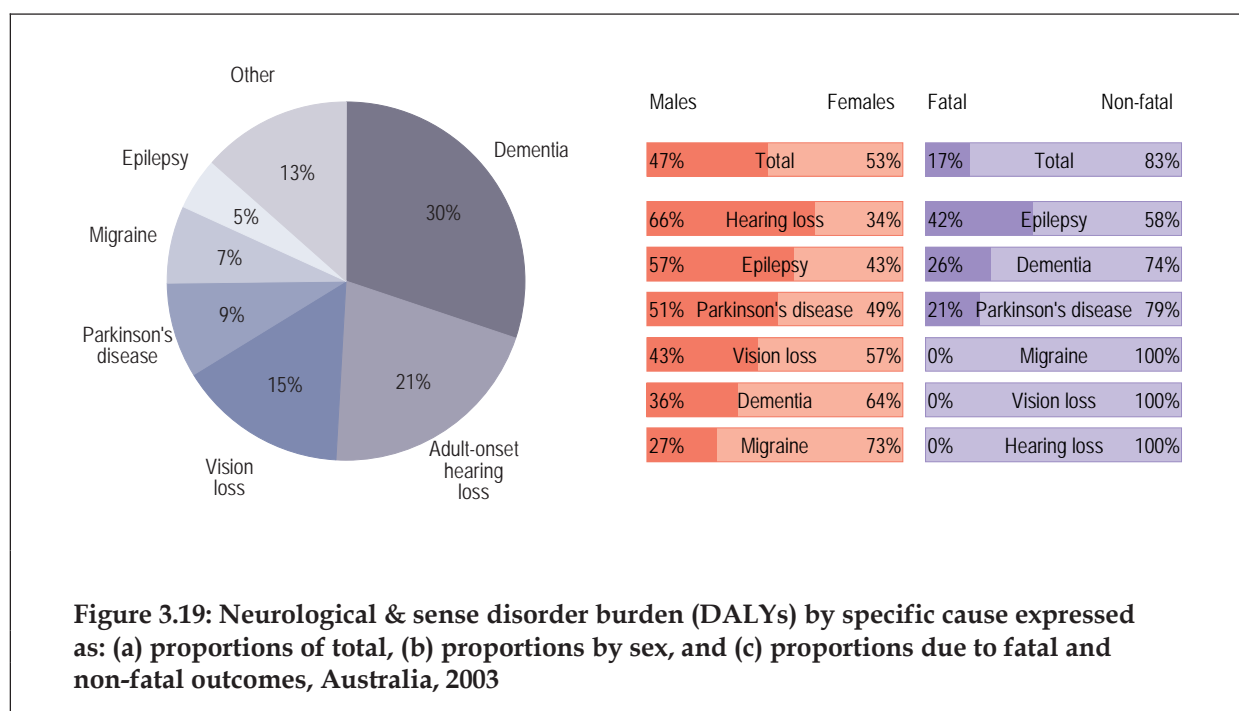
In males, anxiety & depression was the third leading cause of overall male burden, while alcohol abuse was the fourteenth. In females, anxiety & depression was the leading cause of overall female burden, while isolated personality disorders was the thirteenth (Table 3.1). Anxiety & depression also carries with it an increased risk of ischaemic heart disease and suicide. When this risk was accounted for, the burden attributable to anxiety & depression increased from 7.3% to 8.2% of total burden (Table 3.14). The contribution of other mental disorders to the burden of suicide follows in the section on injuries.

Table 3.14: Mental disorder burden (YLD, YLL and DALYs) by specific cause, Australia, 2003

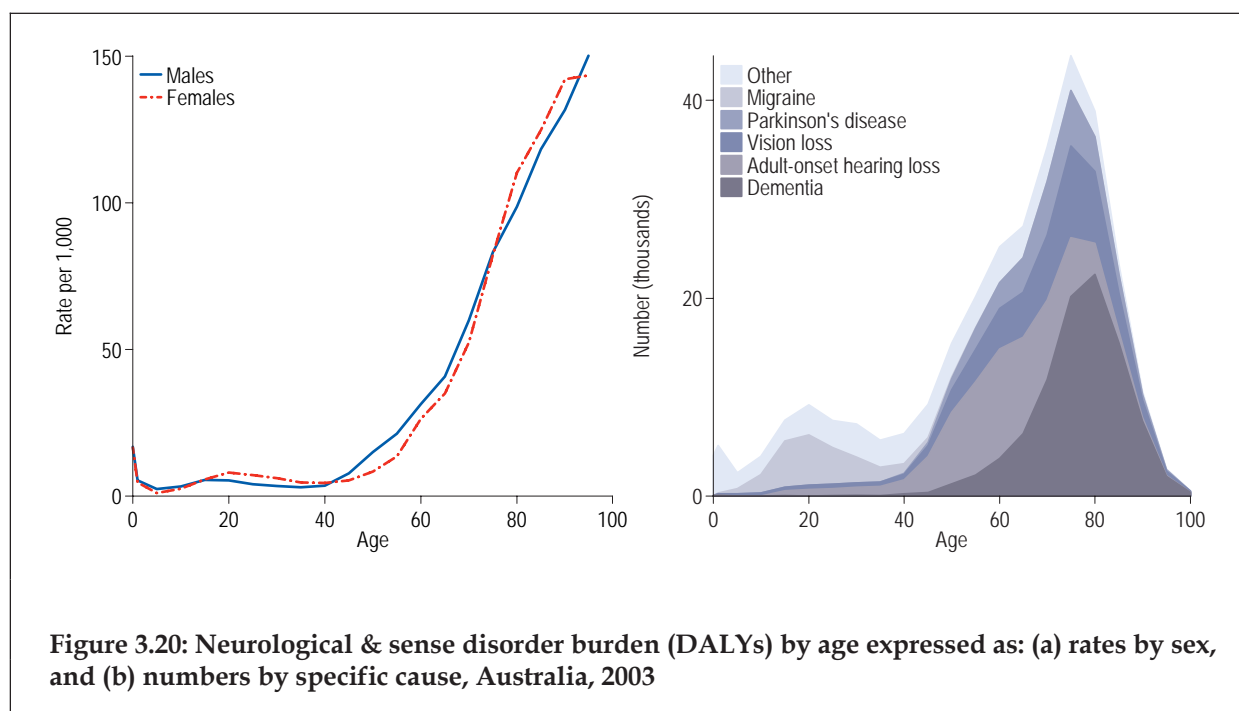
Cause	YLD	Per cent of total	YLL	Per cent of total	DALYs	Per cent of total
Anxiety & depression	191,452	14.1	334	0.0	191,786	7.3
Alcohol abuse	19,861	1.5	14,255	1.1	34,116	1.3
Personality disorders	32,587	2.4	—	0.0	32,587	1.2
Schizophrenia	27,250	2.0	252	0.0	27,502	1.0
Heroin or polydrug abuse	10,287	0.8	6,552	0.5	16,839	0.6
Autism spectrum disorders	13,756	1.0	110	0.0	13,866	0.5
Other	32,198	2.4	1,652	0.1	33,850	1.3
Total mental disorder burden	327,391	24.2	23,154	1.8	350,545	13.3
Ischaemic heart disease attributable to anxiety & depression	1,399	0.1%	5,689	0.4%	7,088	0.3%
Suicide attributable to anxiety & depression	200	0.0%	16,708	1.3%	16,908	0.6%
Total burden attributable to anxiety & depression	193,051	14.3%	22,731	1.8%	215,783	8.2%

Neurological and sense disorders

Neurological & sense disorders were responsible for 11.9% of the total burden of disease and injury in Australia in 2003 (Table 3.11), with dementia, adult-onset hearing loss and vision loss accounting for two-thirds of this burden (Figure 3.19). There were marked sex differentials in the attribution of the neurological & sense disorder burden to particular conditions. Females contributed three times as much to migraine and twice as much to dementia than males. Conversely, the burden from hearing loss was twice as high in males as in females. The greater preponderance of burden from dementia and vision disorders in females was largely due to higher life expectancy in females than males. Only 17% of the burden from neurological & sense disorders was due to mortality.



The burden from neurological & sense disorders, both in absolute terms and when expressed as a rate per head of population, increased with age, with a small but important peak in early adulthood due to the contribution of migraine (Figure 3.20). The contribution from dementia increased from middle age to more than half the burden in the elderly and was more pronounced in females than in males. Conversely, the contribution from hearing loss decreased with age and was more pronounced in males than in females. Vision loss made a smaller but important contribution across all ages.



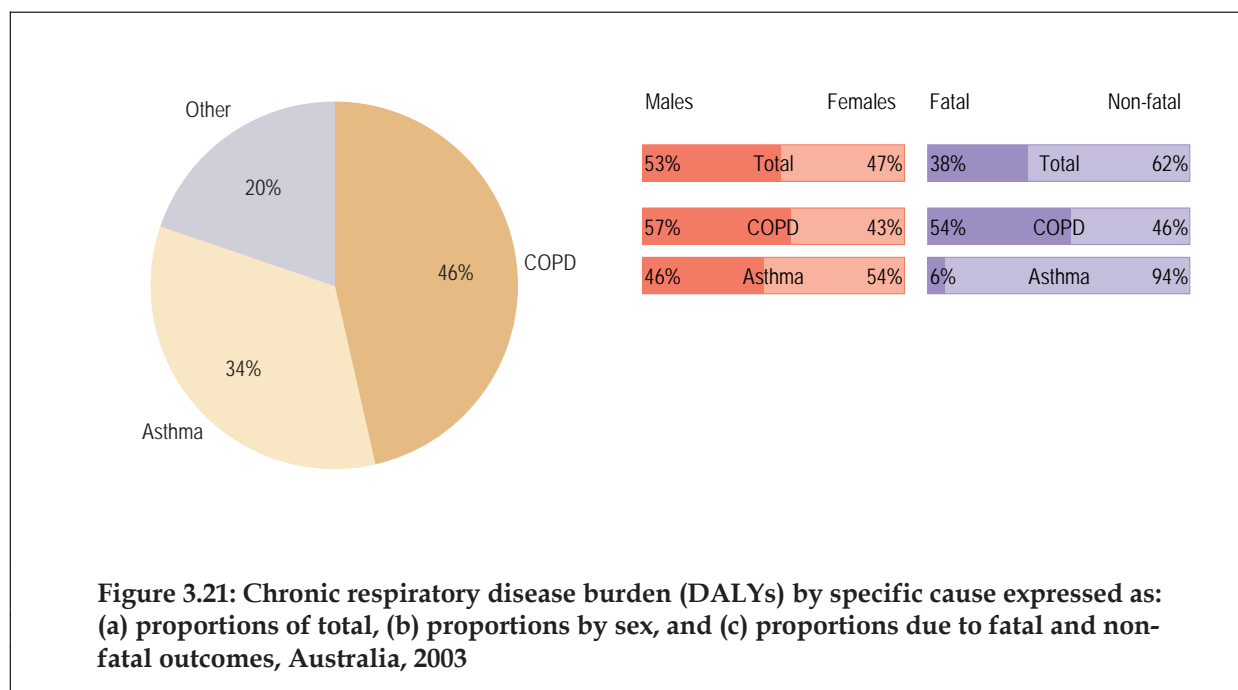
Adult-onset hearing loss, dementia and Parkinson’s disease were the seventh, eleventh and twentieth leading causes of overall male burden. In females, dementia was ranked the fifth leading cause of overall female burden, with hearing loss, migraine and Parkinson’s disease ranked eleventh, fourteenth and eighteenth (Table 3.1).

Table 3.15: Neurological & sense disorder burden (YLD, YLL and DALYs) by specific cause, Australia, 2003

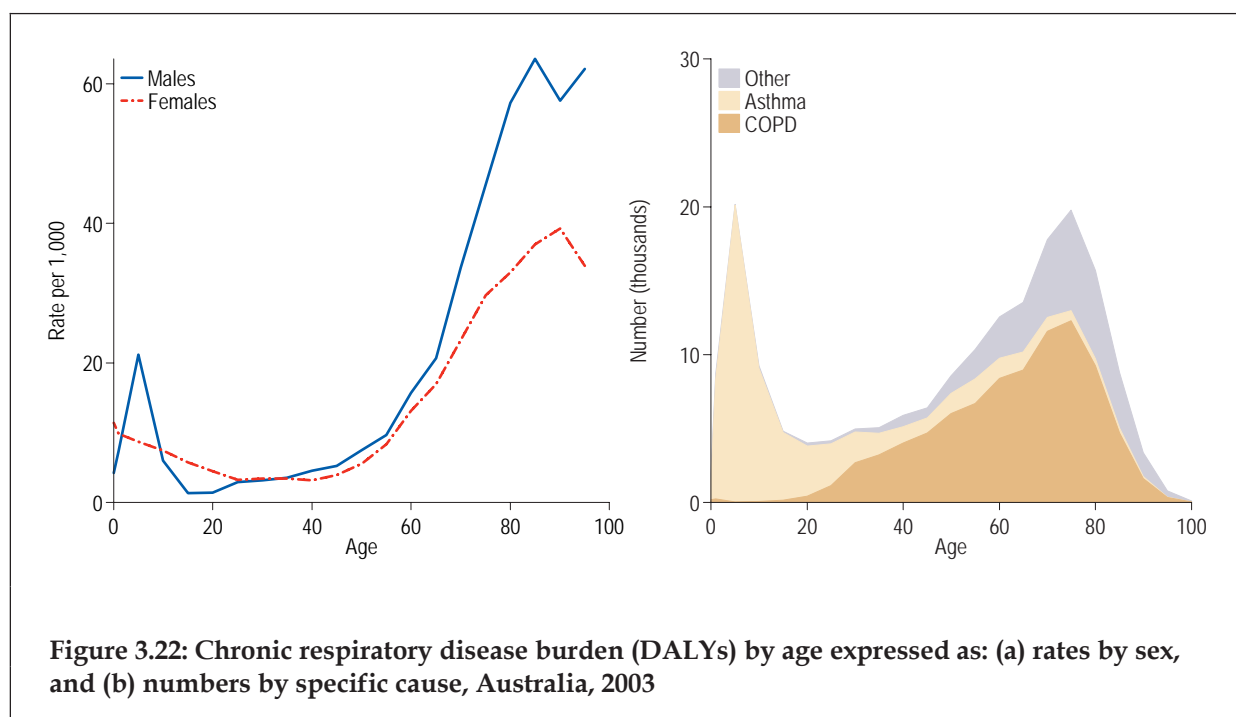
Cause	YLD	Per cent of total	YLL	Per cent of total	DALYs	Per cent of total
Dementia	70,296	5.2	24,103	1.9	94,399	3.6
Adult-onset hearing loss	64,853	4.8	0	0.0	64,853	2.5
Vision loss	47,865	3.5	9	0.0	47,875	1.8
Parkinson’s disease	21,157	1.6	5,695	0.4	26,852	1.0
Migraine	21,841	1.6	7	0.0	21,848	0.8
Epilepsy	8,601	0.6	6,220	0.5	14,821	0.6
Other	24,025	1.8	18,092	1.4	42,118	1.6
Total neurological & sense disorder burden	258,638	19.1	54,127	4.2	312,766	11.9

Chronic respiratory diseases

Chronic respiratory diseases were responsible for 7.1% of total burden of disease and injury in Australia in 2003 (Table 3.11), with chronic obstructive pulmonary disease and asthma accounting for 46% and 34% of this burden, respectively (Figure 3.21). Males had a greater share of chronic obstructive pulmonary disease burden than females because of the greater prevalence of smoking in males 20 to 30 years ago. Fifty-four per cent of the burden from chronic obstructive pulmonary disease was due to mortality, whereas only 6% of the asthma burden was due to mortality.



The burden from chronic respiratory diseases per head of population in both sexes was higher in childhood than in middle age, after which it increased exponentially (Figure 3.22). This was due to the high incidence of asthma in childhood, particularly in boys, and then remission as the body matures. Chronic obstructive pulmonary disease, largely from smoking, was the leading cause of chronic respiratory disease burden from middle age onwards.



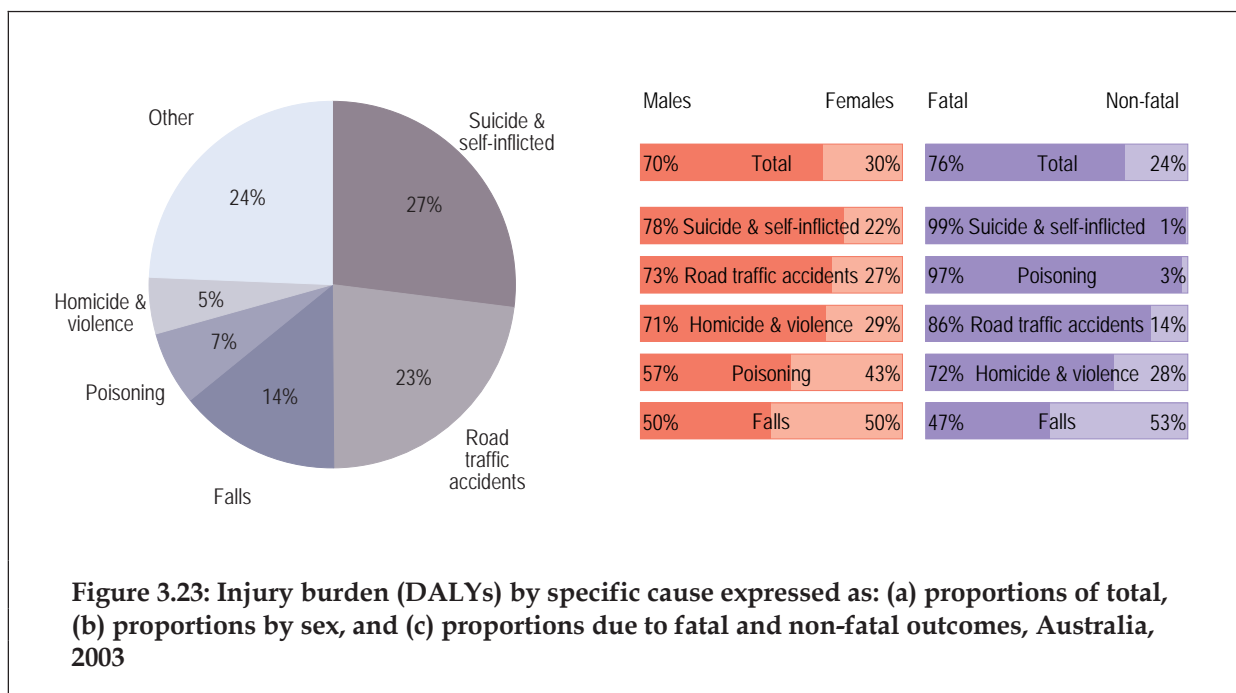
Chronic obstructive pulmonary disease was the sixth leading cause of overall male burden, with asthma at thirteenth. In females, these conditions were ranked seventh and ninth in the leading causes of overall female burden, respectively (Table 3.1).

Table 3.16: Chronic respiratory disease burden (YLD, YLL and DALYs) by specific cause, Australia, 2003

Cause	YLD	Per cent of total	YLL	Per cent of total	DALYs	Per cent of total
COPD	39,543	2.9	47,208	3.7	86,751	3.3
Asthma	59,054	4.4	4,045	0.3	63,100	2.4
Other	16,801	1.2	20,086	1.6	36,887	1.4
Total chronic respiratory disease burden	115,398	8.5	71,339	5.6	186,737	7.1

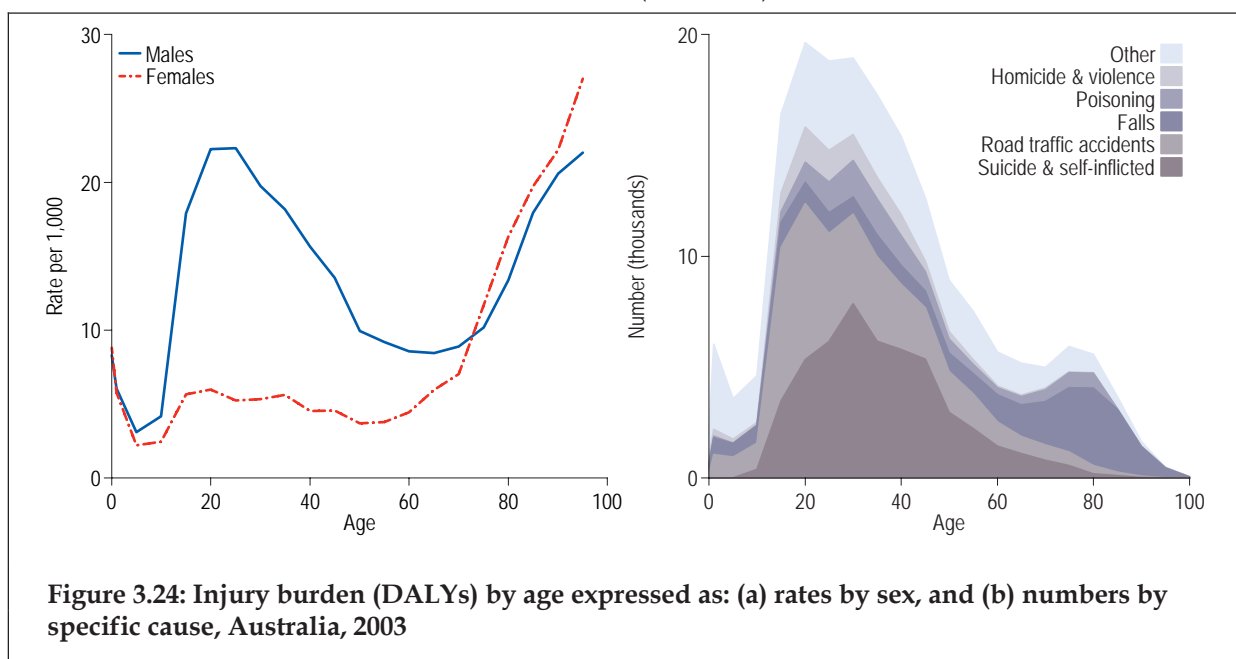
Injuries

Injuries were responsible for 7.0% of the total burden of disease and injury in Australia in 2003 (Table 3.11), with suicide & self-inflicted injuries, road traffic accidents and falls accounting for nearly two-thirds of this burden (Figure 3.23). The burden in males was greater than females for most causes of injury. Males accounted for 73% of the burden due to road traffic accidents and 78% for suicide & self-inflicted injuries. The burden from falls, on the other hand, was equally distributed amongst males and females. Seventy-six per cent of the overall injury burden was due to mortality.



The injury burden in males was greater in early adulthood than at other ages, both in absolute terms and when expressed as a rate per head of population (Figure 3.24). For females, the absolute burden was greatest in the very young, while the rate increased with age from a third of the male rate at early adulthood to similar levels at old age. The peak in absolute burden in early adulthood was due to the high mortality from road traffic accidents and suicide at this life stage. The distribution of injury burden by cause was similar between males and females at all ages.

In males, suicide & self-inflicted injuries and road traffic accidents were the eighth and twelfth leading cause of overall male burden. In females, only falls were in the 20 leading causes of overall female burden at seventeenth (Table 3.1).



Suicide & self-inflicted injuries were responsible for 27% of the total injury burden in Australia in 2003 (Figure 3.23), with anxiety & depression and alcohol abuse accounting for nearly three-quarters of this burden (Figure 3.25). The burden in males was greater than in females for all major causes of suicide & self-inflicted injury.

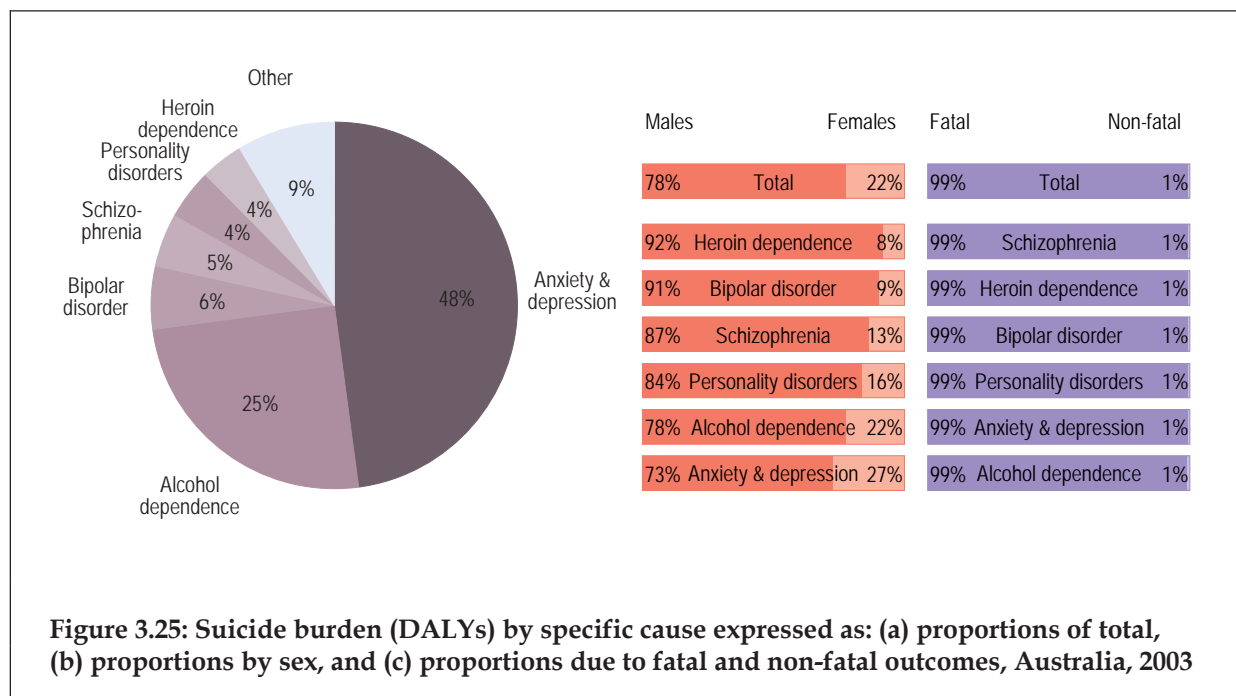


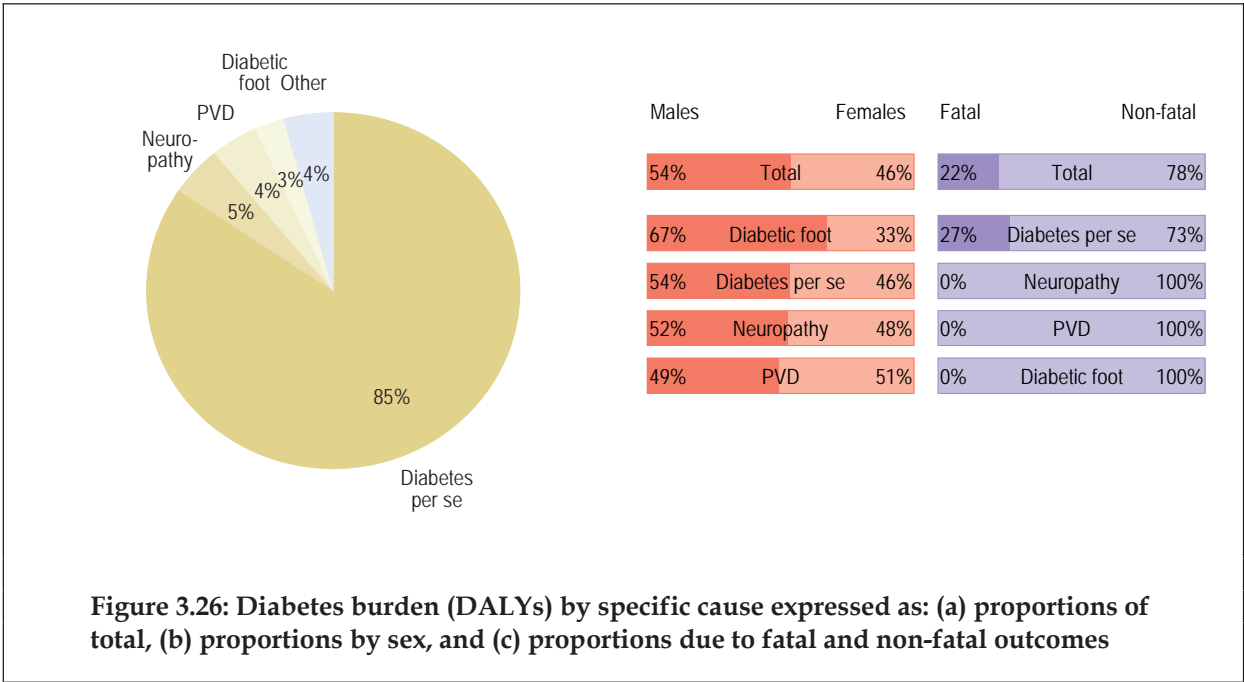
Table 3.17: Injury burden (YLD, YLL and DALYs) by specific cause, Australia, 2003

Cause	YLD	Per cent of total	YLL	Per cent of total	DALYs	Per cent of total
Suicide & self-inflicted injuries	537	0.0	49,379	3.9	49,916	1.9
Road traffic accidents	6,073	0.4	36,352	2.8	42,425	1.6
Falls	13,995	1.0	12,391	1.0	26,386	1.0
Poisoning	326	0.0	11,720	0.9	12,046	0.5
Homicide & violence	2,597	0.2	6,624	0.5	9,221	0.4
Other transport accidents	2,873	0.2	5,728	0.4	8,601	0.3
Other	18,001	1.3	18,454	1.4	36,454	1.4
Total injury burden	44,402	3.3	140,648	11.0	185,050	7.0

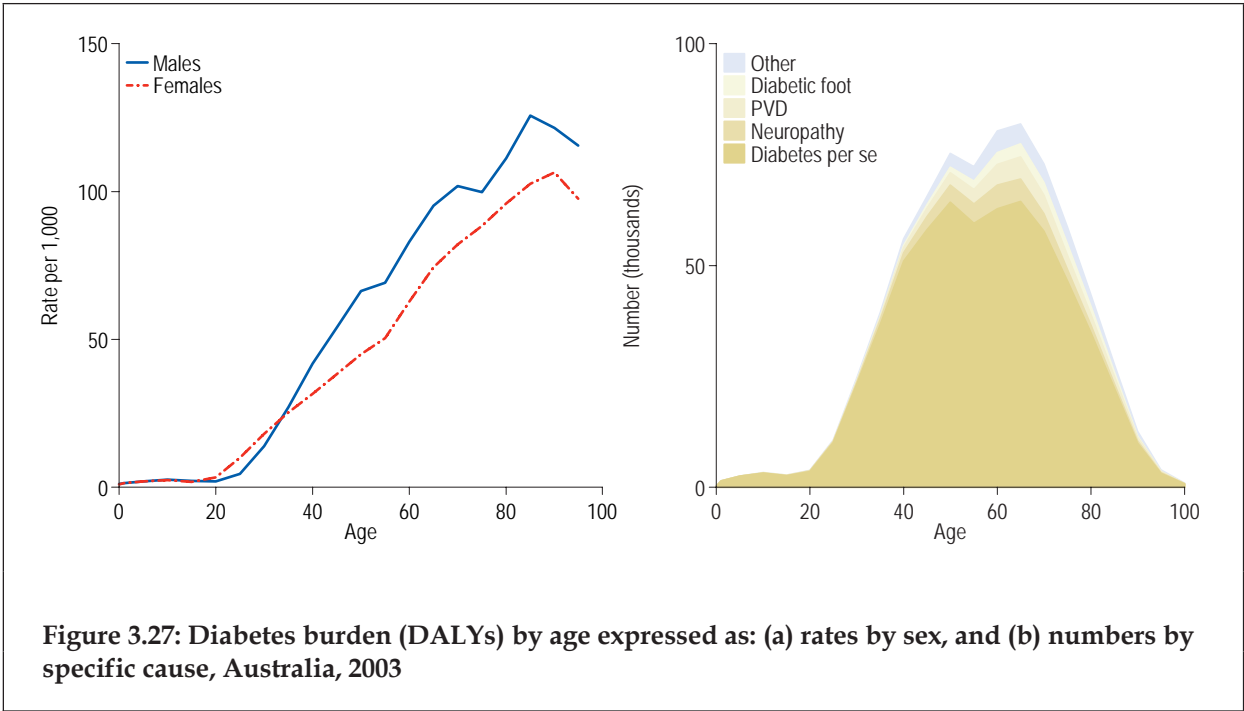
Diabetes

Diabetes was responsible for 5.5% of the total burden of disease and injury in Australia in 2003 (Table 3.11), with Type 2 diabetes accounting for 92% of this burden. Eighty-five per cent of the total diabetes burden was due to diabetes per se (that is, the experience of being diabetic regardless of complications), with the remainder being due to complications such as

neuropathy, peripheral vascular disease (PVD), and diabetic foot (Figure 3.26). Twenty-two per cent of the total diabetes burden was due to mortality.



The risk of burden from diabetes in both sexes increased linearly until age 85 then declined (Figure 3.27). The contribution from diabetes per se dominated at all ages. Diabetes ranked second and fourth in the 20 leading causes of burden for males and females, respectively (Table 3.1).



Diabetes also carries with it an increased risk of ischaemic heart disease and stroke. When this risk was accounted for, the burden attributable to diabetes increased to 8.3% of total burden (Table 3.18).

Table 3.18: Diabetes burden (YLD, YLL and DALYs) by specific cause, Australia, 2003

Cause	YLD	Per cent of total	YLL	Per cent of total	DALYs	Per cent of total
Diabetes per se	89,252	6.6	32,295	2.5	121,547	4.6
Neuropathy	6,500	0.5	—	0.0	6,500	0.2
Peripheral vascular disease	5,917	0.4	—	0.0	5,917	0.2
Diabetic foot	3,672	0.3	—	0.0	3,672	0.1
Amputation	2,455	0.2	—	0.0	2,455	0.1
Retinopathy	1,258	0.1	—	0.0	1,258	0.0
Other ^(a)	2,483	0.2	—	0.0	2,483	0.1
Total diabetes burden	111,536	8.2	32,295	2.5	143,831	5.5
Ischaemic heart disease attributable to diabetes	8,494	0.6	45,948	3.6	54,442	2.1
Stroke attributable to diabetes	3,985	0.3	16,260	1.3	20,245	0.8
Total burden attributable to diabetes	124,015	9.2	94,503	7.4	218,518	8.3

(a) Includes renal failure.

Musculoskeletal diseases

Musculoskeletal diseases were responsible for 4.0% of the total burden of burden and injury in Australia in 2003 (Table 3.11), with osteoarthritis, back pain and rheumatoid arthritis accounting for over three-quarters of this burden (Figure 3.28). Only 7% of the musculoskeletal burden was due to mortality. The sex difference evident in the burden due to osteoarthritis and rheumatoid arthritis was mainly a result of the higher female life expectancy, which in turn allowed for more incident cases of this disease. The lack of a plausible physiological or occupational explanation for the large sex difference in burden from occupational overuse syndrome (OOS) provides support to the notion that this syndrome is not a single entity.

The risk of burden from musculoskeletal diseases in both sexes increased until age 80 then declined (Figure 3.29). The contribution of back pain was relatively constant until age 70 then declined in proportion to the contribution from osteoarthritis.

Osteoarthritis and back pain conditions ranked seventeenth and eighteenth in the 20 leading causes of burden for males, and twelfth and fifteenth for females (Table 3.1).

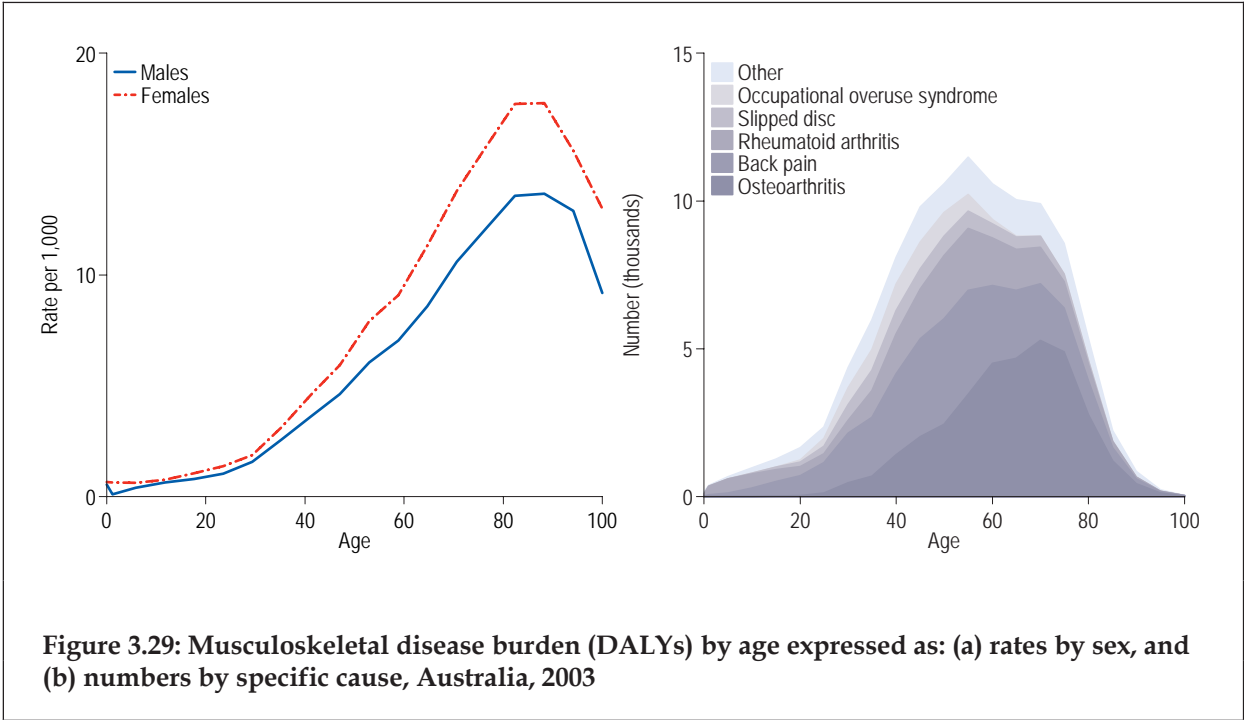
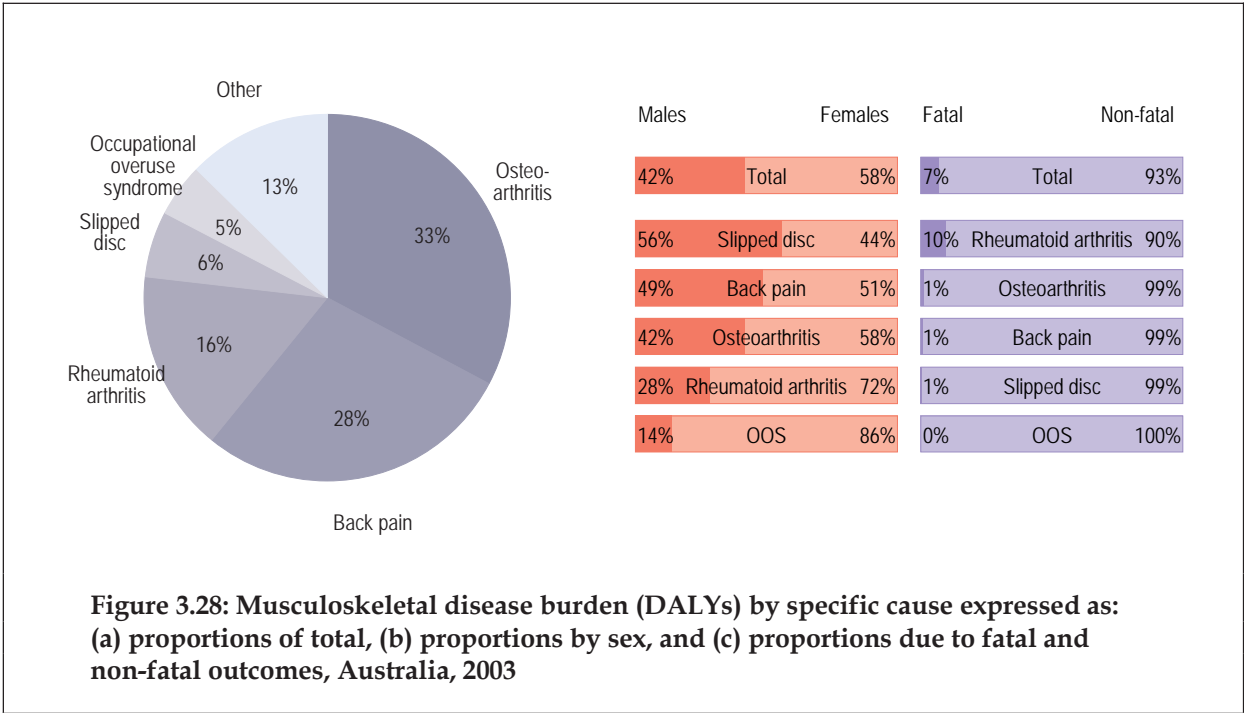


Table 3.19: Musculoskeletal disease burden (YLD, YLL and DALYs) by specific cause, Australia, 2003

Cause	YLD	Per cent of total	YLL	Per cent of total	DALYs	Per cent of total
Osteoarthritis	34,204	2.5	374	0.0	34,578	1.3
Back pain	29,484	2.2	173	0.0	29,658	1.1
Rheumatoid arthritis	15,215	1.1	1,626	0.1	16,841	0.6
Slipped disc	6,089	0.4	31	0.0	6,120	0.2
Occupational overuse syndrome	4,953	0.4	—	0.0	4,953	0.2
Gout	1,813	0.1	175	0.0	1,988	0.1
Other	6,722	0.5	4,647	0.4	11,369	0.4
Total musculoskeletal disease burden	98,481	7.3	7,027	0.5	105,508	4.0

Alternative categories for selected conditions

The burden from intellectual disability, renal failure and vision disorders was attributed to multiple underlying causes in the primary listing of diseases and injuries and is therefore not discussed explicitly in the above sections. The burden from intellectual disability, apart from congenital conditions (for example Down syndrome), was divided among epilepsy, autism spectrum disorders, infectious diseases, injuries, and perinatal conditions. The burden from renal failure was divided among diabetic nephropathy, the injury category of medical misadventure (analgesic nephropathy), and congenital conditions (dysplasia, polycystic kidneys). The burden from total vision loss was divided among diabetic retinopathy, glaucoma, cataract, refraction errors, age-related macular degeneration and other causes of vision loss. The total burden from intellectual disability, renal failure and total vision loss after re-aggregation was 1.7%, 2.6% and 2.1%, respectively (Table 3.20).

Table 3.20: Aggregated burden (YLD, YLL and DALYs) for selected conditions, Australia, 2003

Cause	YLD	Per cent of total	YLL	Per cent of total	DALYs	Per cent of total
Intellectual disability	20,999	1.6	23,189	1.8	44,187	1.7
Renal failure	3,809	0.3	64,912	5.1	68,721	2.6
Total vision loss	50,671	3.7	4,868	0.4	55,539	2.1

4 Risks to health in Australia

4.1 Overview

This chapter discusses the contribution of a number of health risks to the burden of disease and injury in Australia for 2003. The analyses are not meant to be comprehensive since choices had to be made about which risks to include on the basis of the availability of the following:

1. Good evidence of a causal association between the exposure to the risk and the health outcomes
2. Current estimates from reputable epidemiological studies of the relative risk involved
3. Reliable estimates of exposure in the Australian population to the health risk.

The outcome of these considerations was a set of 14 selected health risks as outlined in Table 4.1. Several important dietary factors were considered for inclusion (for example sodium and saturated fat) as part of these deliberations, but were ultimately excluded due to inadequate data on exposure. With the exception of low fruit and vegetable consumption, therefore, the impact of 'poor diet' is measured indirectly through the assessments for high body mass, blood cholesterol and blood pressure. Similarly, lack of data on prevalence and outcome prevented estimation of the burden of intimate partner violence in males.

Table 4.1: Fourteen selected risks to health discussed in this report

Lifestyle behaviours	Physiological states	Social and environmental factors
1. Tobacco	7. High body mass	11. Urban air pollution
2. Alcohol	8. High blood pressure	12. Intimate partner violence
3. Physical inactivity	9. High blood cholesterol	13. Child sexual abuse
4. Illicit drugs	10. Osteoporosis	14. Occupational exposures & hazards
5. Low fruit and vegetable consumption		
6. Unsafe sex		

It is important to remember several points when interpreting the results in the following sections.

Firstly, health risks tend to cluster around 'high risk' individuals who experience more than one exposure (for example smokers tend to be drinkers). This combination of exposures may produce higher or lower levels of overall risk as a result of complex interaction effects. The analyses presented in this chapter do not explicitly account for these interactions, except to the extent to which confounding was controlled for in the studies from which the exposure-outcome relationships were derived.

Secondly, the causal paths between a number of related health risks and their eventual health outcomes can be complicated. For example, physical inactivity can lead to obesity, which can cause hypertension or high blood cholesterol, which can ultimately lead to cardiovascular disease. Most of the analyses presented in this chapter only measure the effect of a risk independent of the other exposures and irrespective of the risk's place in a causal

path. The important implication here is that such analyses are not additive. Using the example above, the burden attributable to physical inactivity is estimated to be 23.7% of total cardiovascular disease burden, while that for high body mass, high blood cholesterol and high blood pressure was 19.5%, 34.5% and 42.1% of cardiovascular disease, respectively (Table 4.2). The burden attributable to these health risks in combination, however, is not the sum of burden from each risk (that is, the combined burden is not 119.9%). This is because the combined effect of these risks has to be expressly calculated rather than derived from the addition of their individual effects. Ignoring shared causal paths in this example leads to obvious over-estimation of the combined effect.

To illustrate the total 'explanatory' power of the 14 risk factors, the chapter begins with an analysis that accounts for many of the overlaps between risks that share causal paths. This is done using the 'joint effects' method developed for the WHO Comparative Risk Assessment project (Ezzati et al. 2004b). Sensitivity analyses indicate that overall results based on this approach are relatively robust to the underlying assumptions; apportioning the combined overall risk back to each contributing risk factor is more difficult, however, and is much more sensitive to assumptions. Therefore, only the former analyses are presented in this report. Further details on the methods used for estimating joint effects are provided in Chapter 2.

4.2 Combined effect of 14 selected risks to health

The 14 selected risk factors presented in this chapter together explained 32.2% of the total burden of disease and injury in Australia in 2003 (Table 4.2). These risk factors explained 35.1% and 29.1% of the total burden in males and females respectively (Table 4.3). This indicates that there is considerable potential to further reduce burden in Australia through interventions that target these health risks, each of which contribute to more than one health outcome. Additional evidence on the (cost-) effectiveness of such interventions may guide the setting of health service priorities to meet this objective.

Key findings about broad cause groups were:

- Ten of the risks were associated with cancer and together explained 32.9% of the total burden from this cause. The majority was explained by tobacco. The contributions of the other risk factors (physical inactivity, high body mass, alcohol, occupational exposure, low fruit and vegetable consumption, air pollution and unsafe sex (through the link between the human papilloma virus and cancer of the cervix)) were comparatively much smaller.
- Twelve of the risks were associated with cardiovascular disease and together explained 69.3% of the burden from this group of causes; for ischaemic heart disease, this figure was 85.2%. High blood pressure and high blood cholesterol were the largest contributors, followed by physical inactivity, high body mass, tobacco, and low fruit and vegetable consumption. The very low prevalence of smoking in elderly Australians, who are most affected by cardiovascular disease, explains the relatively small contribution of tobacco to this disease.
- Four of the risks were associated with mental disorders and together explained 26.9% of the burden from this cause. Alcohol and illicit drugs contributed in roughly equal proportions. Intimate partner violence and child sexual abuse contributed less but were the only risks implicated in the large burden from anxiety and depression.

- Three of the risks were associated with neurological and sense disorders, and together explained only 0.2% of the burden from these disorders. This reflects lack of knowledge about causation in this group. Ultraviolet light, causing cataract, is probably the most obvious omitted risk factor in this disease category but the burden of cataract is small because surgical treatment is widely available.
- Seven of the risks were associated with injury and together explained 31.7% of the burden from this cause. Alcohol was by far the largest contributor, followed by occupational exposures and hazards, illicit drugs, intimate partner violence, osteoporosis, child sexual abuse, and tobacco.
- Two of the risks were associated with Type 2 diabetes (including the proportion of cardiovascular disease caused by diabetes) and together explained 60.1% of the burden from this cause. High body mass was by far the largest contributor to this disease.

Table 4.2: Individual and joint burden (DALYs) attributable to 14 selected risk factors by broad cause group, Australia, 2003

	Broad cause group							All causes
	Cancer	CVD	Mental	Neuro-logical	Injury	Diabetes	Other	
Total burden ('000)	499.4	473.8	350.5	312.8	185.1	143.8	667.4	2,632.8
Attributable burden (%) ^(a)								
Tobacco	20.1	9.7	—	-0.6	0.5	—	8.9	7.8
High blood pressure	—	42.1	—	—	—	—	—	7.6
High body mass	3.9	19.5	—	—	—	54.7	1.1	7.5
Physical inactivity	5.6	23.7	—	—	—	23.7	>-0.1	6.6
High blood cholesterol	—	34.5	—	—	—	—	—	6.2
Alcohol								
Harmful effects	3.1	0.9	9.7	—	18.1	—	<0.1	3.3
Beneficial effects	—	-5.6	—	—	—	—	>-0.1	-1.0
Net effects	3.1	-4.7	9.7	—	18.1	—	<0.1	2.3
Low fruit & vegetable consumption	2.0	9.6	—	—	—	—	>-0.1	2.1
Illicit drugs	—	<0.1	8.0	—	3.6	—	2.5	2.0
Occupational exposures & hazards	3.1	0.4	—	0.8	4.7	—	3.4	2.0
Intimate partner violence	0.5	0.3	5.5	0.1	2.5	—	0.2	1.1
Child sexual abuse	<0.1	<0.1	5.8	—	1.4	—	<0.1	0.9
Urban air pollution	0.8	2.7	—	—	—	—	0.4	0.7
Unsafe sex	1.0	—	—	—	—	—	1.4	0.6
Osteoporosis	—	—	—	—	2.4	—	—	0.2
Joint effect ^(b)	32.9	69.3	26.9	0.2	31.7	60.1	17.2	32.2

(a) Attributable burden within each column is expressed as a percentage of total burden for that column.

(b) Figures for joint effects are not column totals. See Section 4.1 for further details.

The 14 selected risk factors presented in this chapter had a differential impact on health in terms of both sex and age (Table 4.3). In the 0–44 year-old age group, alcohol and illicit drugs

were the leading causes of burden in males, mental disorders (alcohol abuse, and heroin and polydrug abuse) and injuries (suicide and self-inflicted injuries, and road traffic accidents) being the predominant health outcomes from these risks. In this age group, 23.6% of total male burden and 17.9% of total female burden was explained by the 14 risks in combination. In females, intimate partner violence and child sexual abuse were the leading causes in this age group, anxiety and depression and suicide and self-inflicted injuries being the predominant health outcomes from these risks.

In the 45–64 year-old age group, high body mass and tobacco were the leading causes in both sexes, Type 2 diabetes, ischaemic heart disease, stroke, lung cancer and chronic obstructive pulmonary disease (COPD) being the predominant health outcomes from these risks. The proportion of total burden in this age group that is explained by the 14 risks in combination was 43.8% in males and 33.6% in females.

In the 65 years and over age group, high blood pressure was the leading cause in both sexes, followed by tobacco in males and high blood cholesterol in females. The predominant health outcomes from both high blood pressure and high blood cholesterol are ischaemic heart disease and stroke. For tobacco, the predominant health outcomes are lung cancer and COPD. The proportion of total burden in this age group that is explained by the 14 risks in combination was 38.4% and 34.8% in males and females, respectively.

Table 4.3: Individual and joint burden (DALYs) attributable to 14 selected risk factors by sex and age group, Australia, 2003

	Males				Females			
	0–44	45–64	65+	All ages	0–44	45–64	65+	All ages
Total burden ('000)	448.8	382.5	533.4	1,364.6	406.0	299.1	563.0	1,268.2
Attributable burden (%) ^(a)								
Tobacco	1.9	14.7	12.5	9.6	1.1	8.7	7.6	5.8
High blood pressure	0.8	7.8	13.8	7.8	<0.1	4.0	14.2	7.3
High body mass	3.3	13.3	7.5	7.7	2.6	12.1	8.1	7.3
Physical inactivity	1.8	9.0	8.5	6.4	1.9	8.4	9.6	6.8
High blood cholesterol	1.9	9.6	8.3	6.6	0.7	5.1	9.9	5.8
Alcohol								
Harmful effects	8.1	5.5	1.8	4.9	2.2	2.4	0.8	1.6
Beneficial effects	–0.3	–1.5	–1.5	–1.1	–0.2	–0.9	–1.5	–0.9
Net effects	7.8	4.0	0.3	3.8	2.0	1.4	–0.6	0.7
Low fruit & vegetable consumption	0.8	4.1	3.3	2.7	0.3	1.7	2.2	1.5
Illicit drugs	5.7	1.9	0.6	2.7	2.4	1.1	0.4	1.2
Occupational exposures & hazards	2.7	4.2	1.4	2.6	1.6	2.4	0.4	1.3
Intimate partner violence	—	—	—	—	4.8	2.8	0.3	2.3
Child sexual abuse	0.6	0.3	<0.1	0.3	3.4	1.7	<0.1	1.5
Urban air pollution	0.2	0.9	1.2	0.8	0.1	0.6	1.2	0.7
Unsafe sex	0.8	0.4	0.2	0.5	1.0	0.9	0.4	0.7
Osteoporosis	—	<0.1	0.2	<0.1	—	<0.1	0.6	0.3
Joint effect ^(b)	23.6	43.8	38.4	35.1	17.9	33.6	34.8	29.1

(a) Attributable burden within each column is expressed as a percentage of total burden for that column.

(b) Figures for joint effects are not column totals. See Section 4.1 for further details.

4.3 Individual contribution of 14 selected risks to health

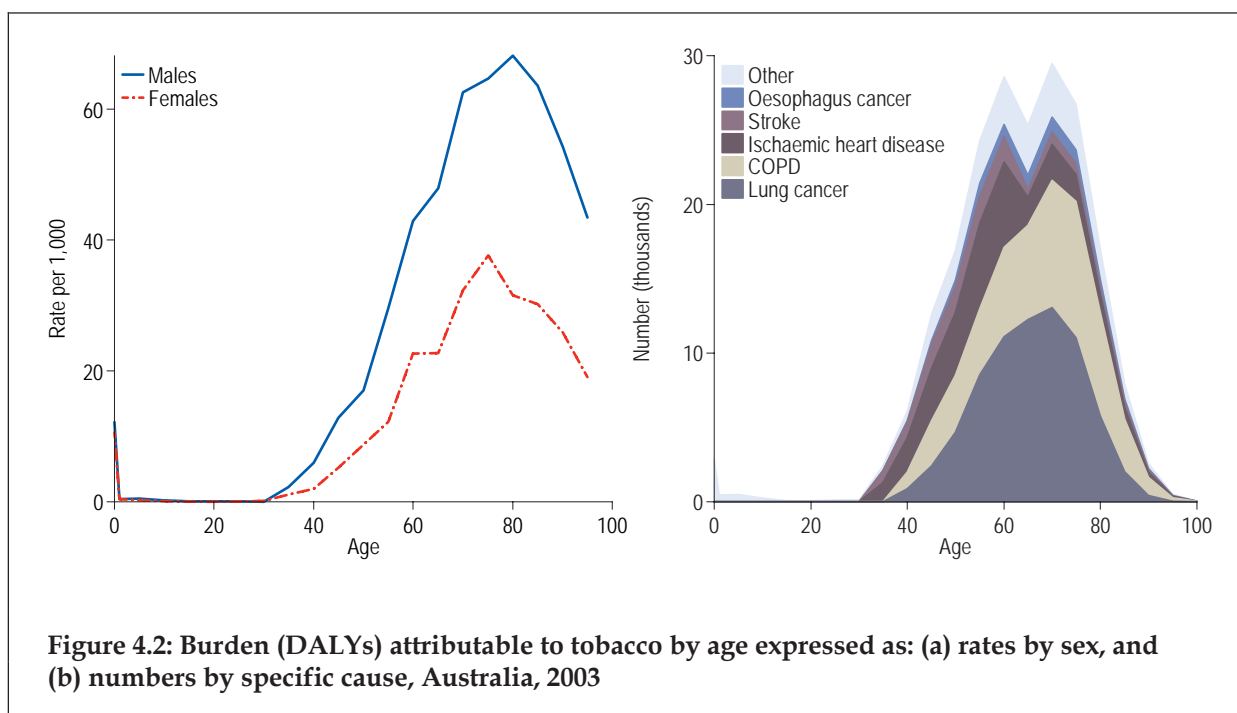
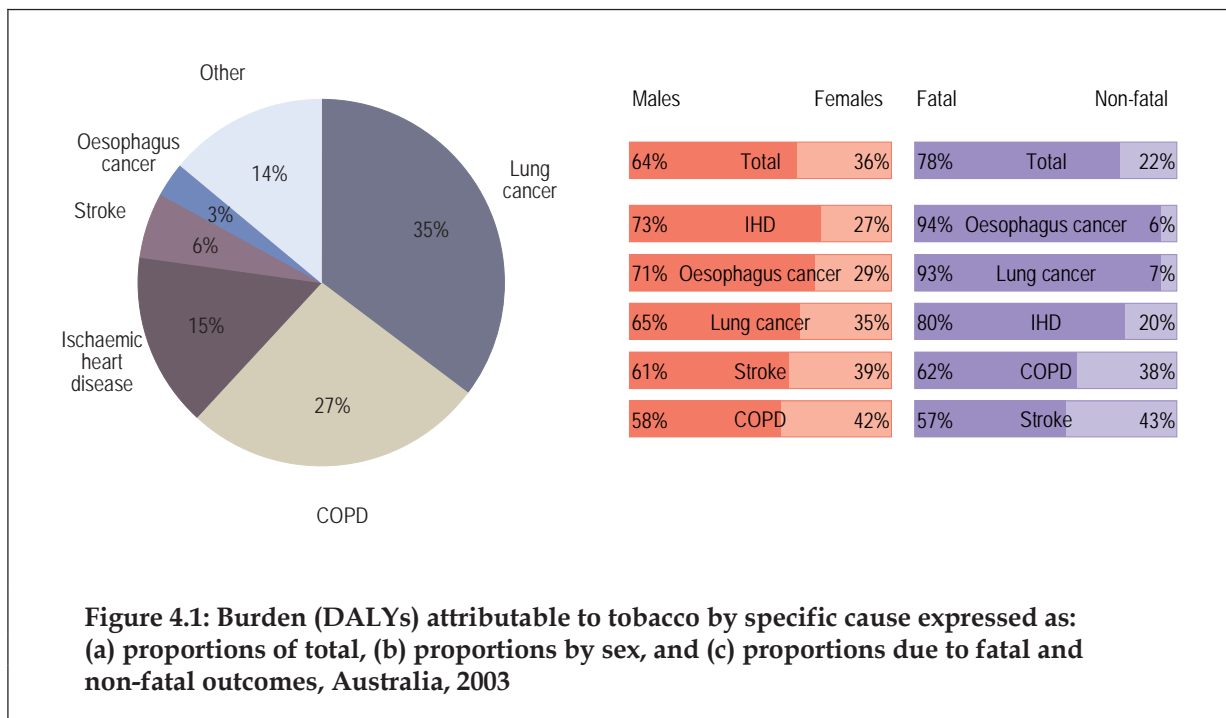
Tobacco

Tobacco was responsible for 7.8% of the total burden of disease and injury in Australia in 2003 (Table 4.4), with lung cancer, COPD and ischaemic heart disease accounting for more than three-quarters of this burden (Figure 4.1). Of the 14 risk factors examined, tobacco was responsible for the largest amount of burden across all ages in males (Table 4.3). Almost two-thirds of the burden from tobacco was experienced by males due to the higher prevalence 20 to 30 years ago of smoking in males compared with females. More than three-quarters of the burden from tobacco was due to mortality (Figure 4.1). Because of the long lag time between smoking and many of its ill effects on health, the health benefits of recent favourable trends in smoking prevalence will not be fully realised until many years in the future.

The rate of burden from tobacco per head of population increased with age until 75 and the absolute burden was concentrated between the ages of 55 and 75. The contribution from lung cancer dominated at most ages but was overtaken by contributions from COPD and ischaemic heart disease in the elderly (Figure 4.2).

Table 4.4: Deaths and burden (DALYs) attributable to tobacco by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Lung cancer	6,309	4.8	72,213	2.7
COPD	4,175	3.2	54,492	2.1
Ischaemic heart disease	1,962	1.5	31,435	1.2
Stroke	577	0.4	11,812	0.4
Oesophagus cancer	572	0.4	6,248	0.2
Other	1,916	1.4	28,588	1.1
Total attributable	15,511	11.7	204,788	7.8



High blood pressure

High blood pressure was responsible for 7.6% of the total burden of disease and injury in Australia in 2003 (Table 4.5), with ischaemic heart disease and stroke accounting for 93% of

this burden (Figure 4.3). Of the 14 risk factors examined, high blood pressure was responsible for the greatest amount of burden in the 65 years or over age group in both sexes (Table 4.3). Overall, the burden from high blood pressure was somewhat greater in males and 81% was due to mortality.

The rate of burden from high blood pressure per head of population increased with age and the absolute burden was concentrated around old age (Figure 4.4). The contributions from ischaemic heart disease and stroke dominated across all ages.

Table 4.5: Deaths and burden (DALYs) attributable to high blood pressure by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Ischaemic heart disease	14,089	10.7	125,461	4.8
Stroke	6,603	5.0	59,962	2.3
Other	1,812	1.4	13,893	0.5
Total attributable	22,504	17.0	199,315	7.6

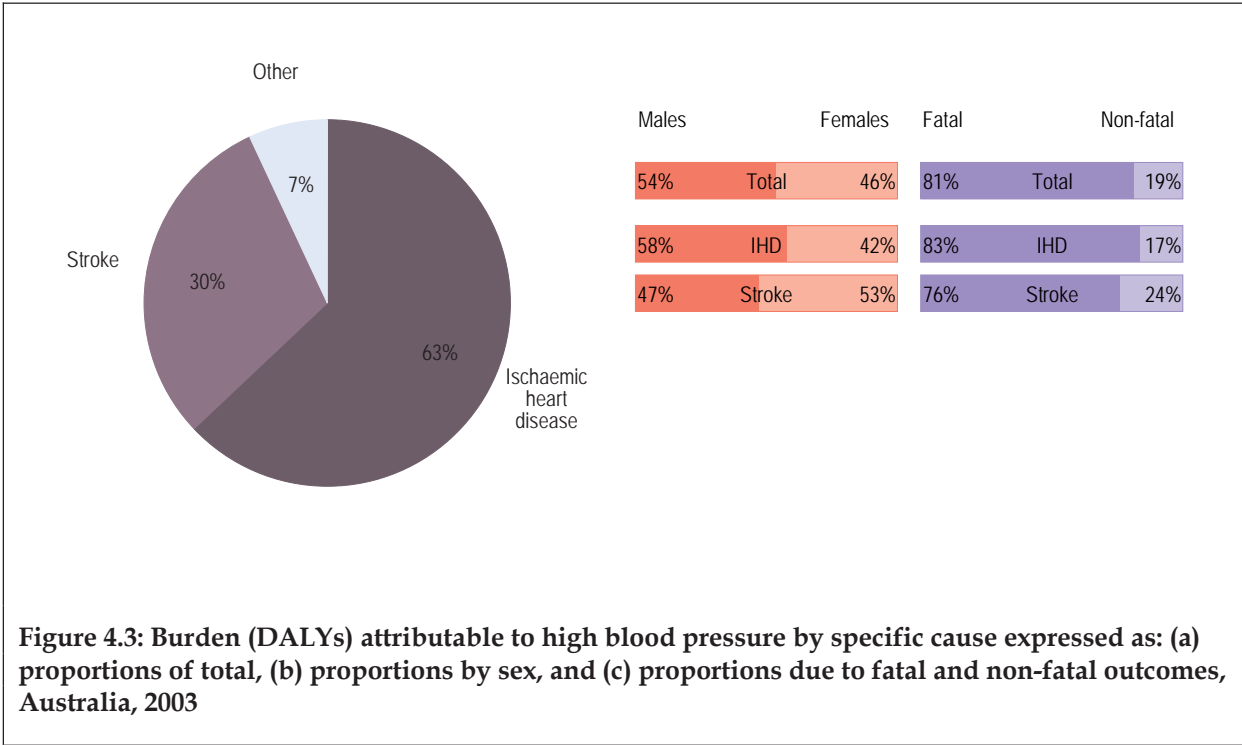
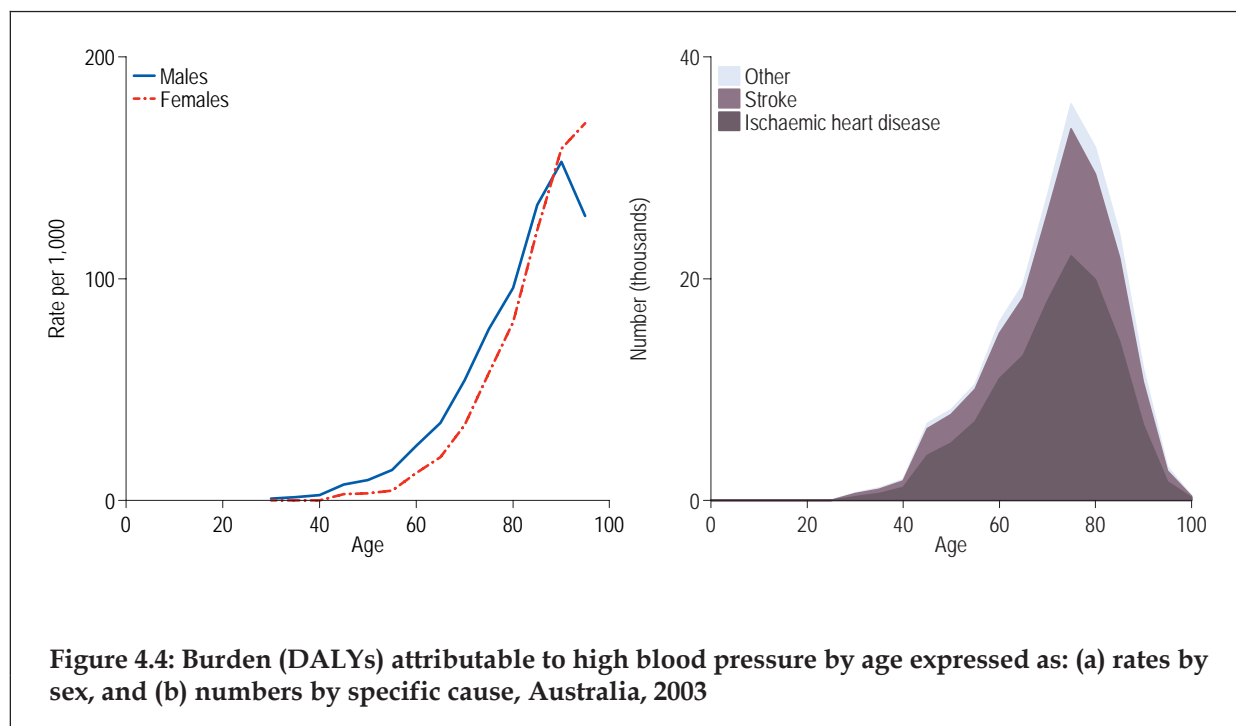


Figure 4.3: Burden (DALYs) attributable to high blood pressure by specific cause expressed as: (a) proportions of total, (b) proportions by sex, and (c) proportions due to fatal and non-fatal outcomes, Australia, 2003



High body mass

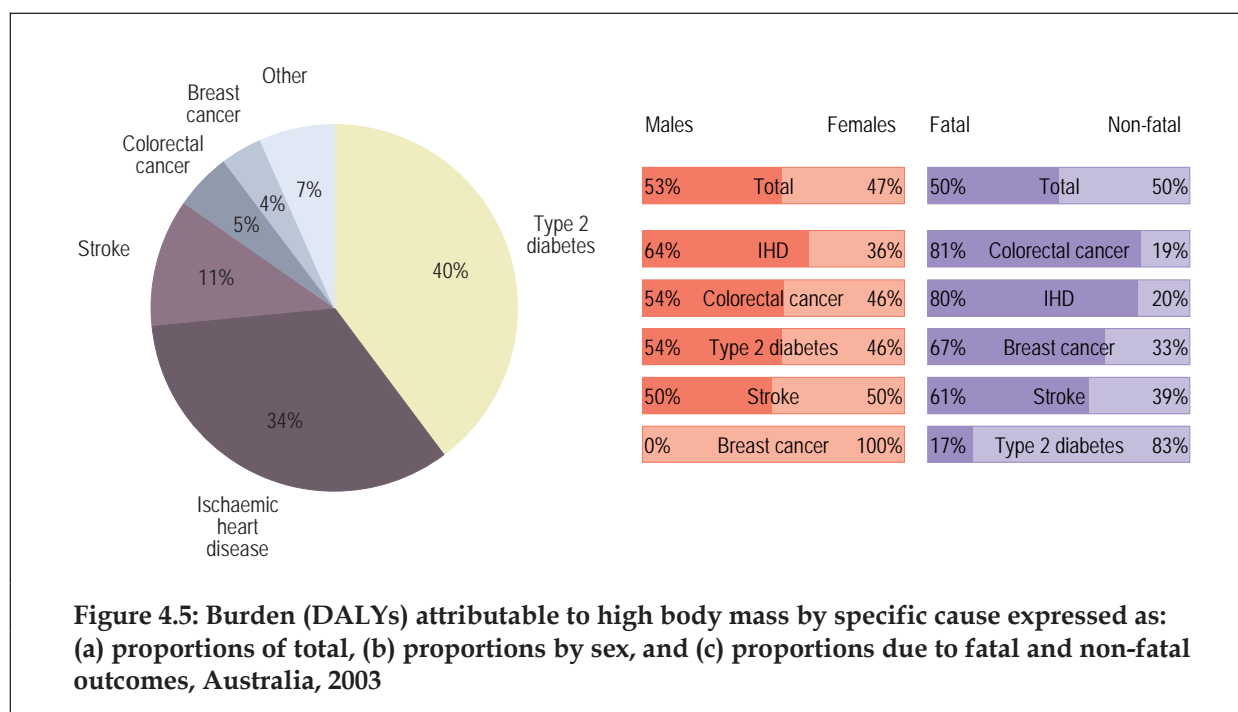
The body mass index (BMI) is a measure of weight in kilograms over height in metres squared and is typically categorised into under weight (BMI<20), normal weight (20≤BMI<25), over weight (25≤BMI<30) and obese (BMI≥30). Rather than use these categories, the health effects of ‘high body mass’ in the following analyses were estimated using new methods in which BMI is measured on a continuous scale and risk is assessed against a minimum counterfactual distribution with a mean of 21 and a SD of 1 (see Appendix 2). This means that risk is attributed to all people in the population with a BMI of greater than 21, with the degree of risk increasing exponentially above this value. The consequence of this approach is that some of the attributable risk from high body mass comes from the large proportion of the population that is not over weight or obese in the conventional sense, but whose risk of disease is elevated, at least to some degree.

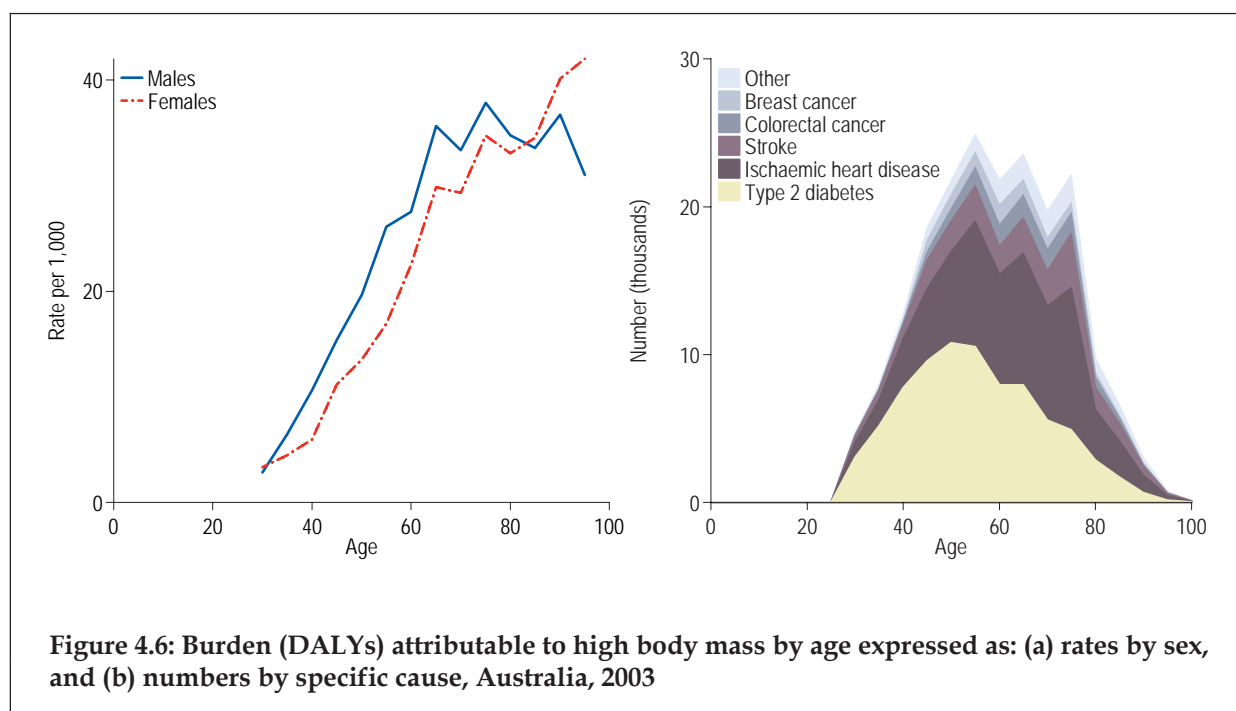
High body mass was responsible for 7.5% of the total burden of disease and injury in Australia in 2003 (Table 4.6), with Type 2 diabetes and ischaemic heart disease (IHD) accounting for almost three-quarters of this burden (Figure 4.5). Of the 14 risk factors examined, high body mass accounted for the greatest amount of burden in the 45–64 year age group in females (Table 4.3). The burden from high body mass was greater in males due to the higher incidence of Type 2 diabetes itself and the associated cardiovascular complications. Half of the burden from high body mass was due to mortality (Figure 4.5).

The rate of burden from high body mass per head of population increased with age; the absolute burden was concentrated between the ages of 55 and 75. The contributions from Type 2 diabetes and ischaemic heart disease dominate across all ages (Figure 4.6).

Table 4.6: Deaths and burden (DALYs) attributable to high body mass by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Type 2 diabetes	1,381	1.0	78,688	3.0
Ischaemic heart disease	4,914	3.7	66,533	2.5
Stroke	1,528	1.2	22,218	0.8
Colorectal cancer	721	0.5	9,920	0.4
Breast cancer	379	0.3	7,125	0.3
Other	602	0.5	13,148	0.5
Total attributable	9,525	7.2	197,632	7.5





Physical inactivity

Physical inactivity was responsible for 6.6% of the total burden of disease and injury in Australia in 2003 (Table 4.7), with ischaemic heart disease, Type 2 diabetes and stroke accounting for more than four-fifths of this burden. Overall, the burden from physical inactivity was shared equally between the sexes. With the exception of diabetes, most of the conditions attributable to physical inactivity were associated with high mortality (Figure 4.7).

The rate of burden from physical inactivity per head of population increased with age and the absolute burden was concentrated around old age. The contributions from ischaemic heart disease and Type 2 diabetes dominated across all ages (Figure 4.8).

Table 4.7: Deaths and burden (DALYs) attributable to physical inactivity by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Ischaemic heart disease	8,739	6.6	88,617	3.4
Type 2 diabetes	704	0.5	34,132	1.3
Stroke	2,390	1.8	23,742	0.9
Colorectal cancer	1,074	0.8	14,978	0.6
Breast cancer	584	0.4	12,962	0.5
Total attributable	13,491	10.2	174,431	6.6

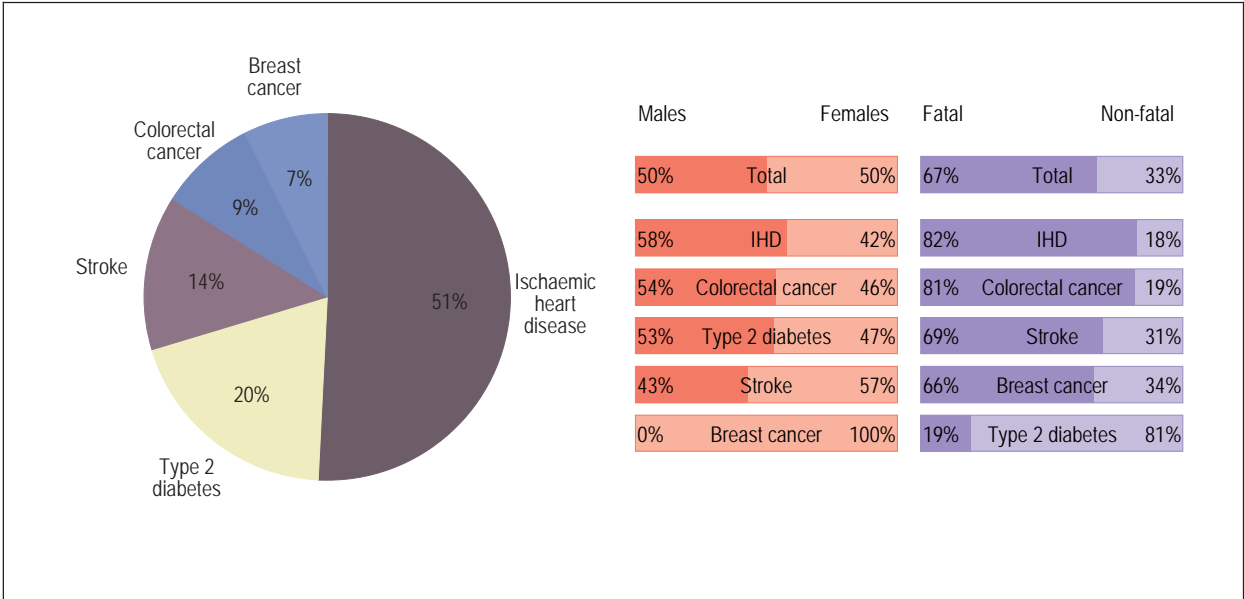


Figure 4.7: Burden (DALYs) attributable to physical inactivity by specific cause expressed as: (a) proportions of total, (b) proportions by sex, and (c) proportions due to fatal and non-fatal outcomes, Australia, 2003

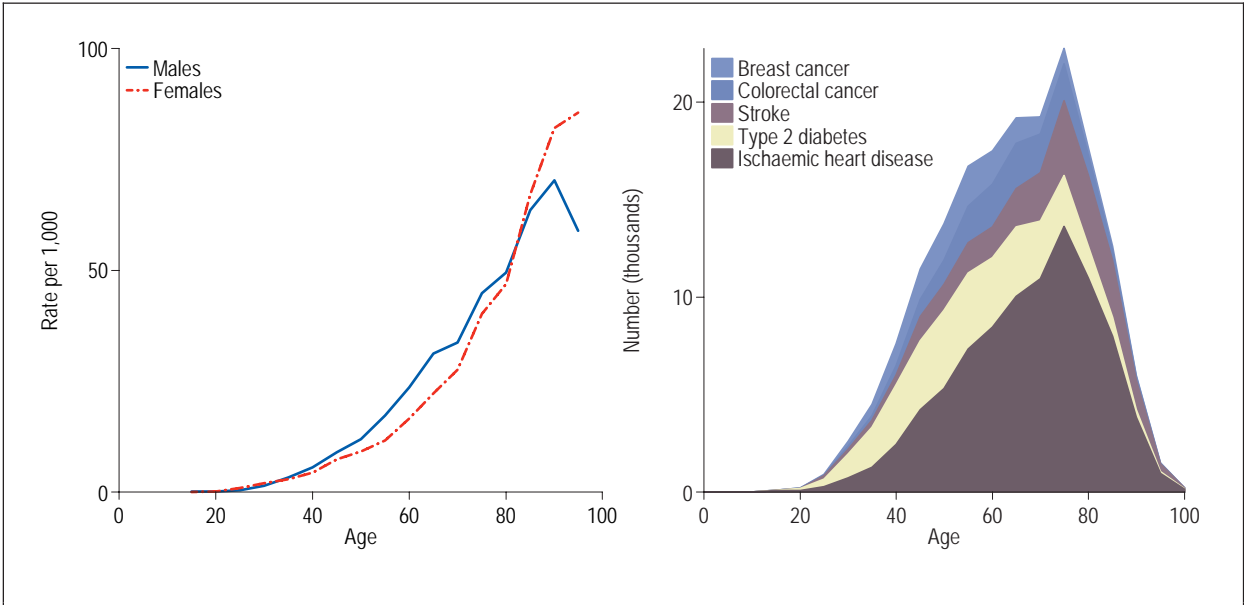


Figure 4.8: Burden (DALYs) attributable to physical inactivity by age expressed as: (a) rates by sex, and (b) numbers by specific cause, Australia, 2003

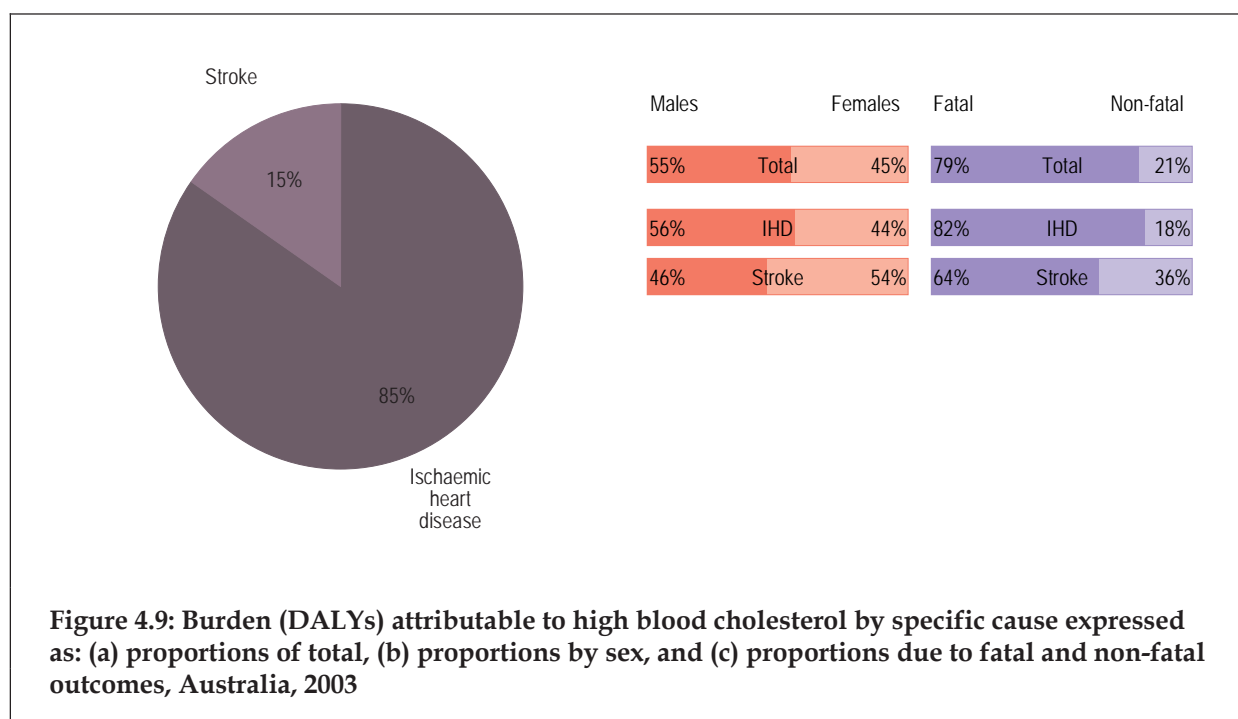
High blood cholesterol

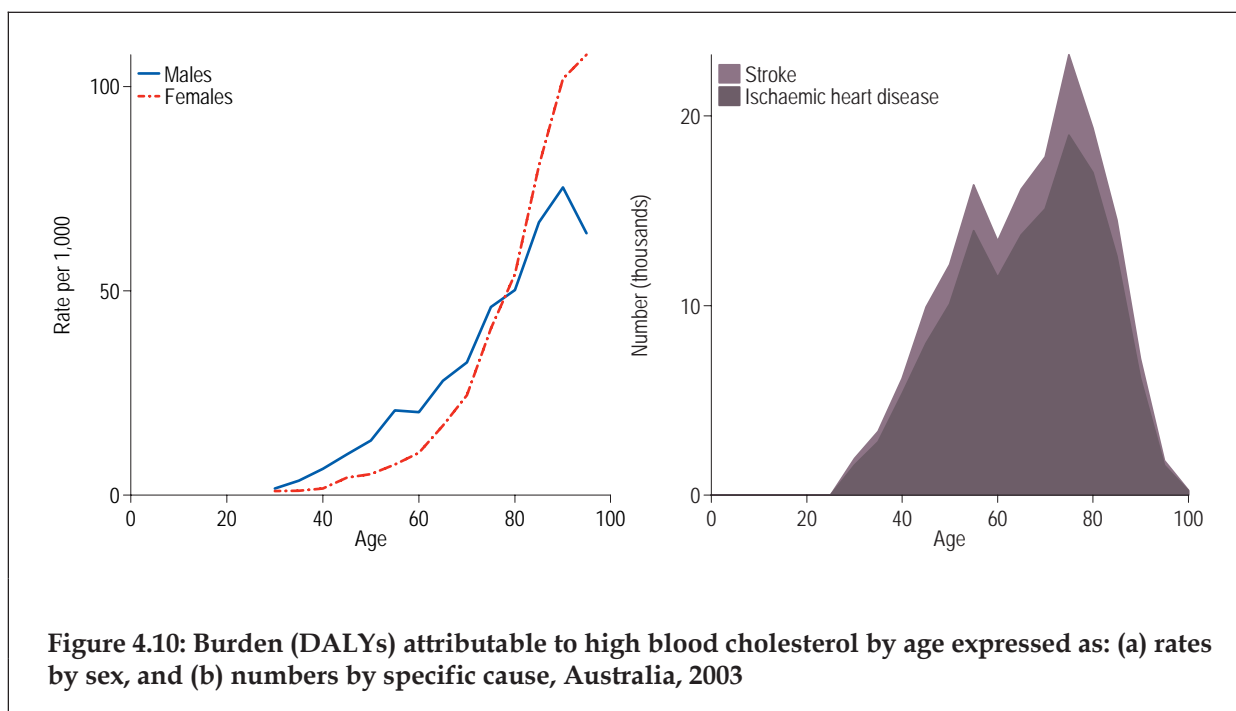
High blood cholesterol was responsible for 6.2% the total burden of disease and injury in Australia in 2003 (Table 4.8), with ischaemic heart disease and stroke accounting for this entire burden. Both ischaemic heart disease and stroke were associated with high mortality. Overall, males experienced a slightly higher burden from high blood cholesterol than females (Figure 4.9).

The rate of burden from high blood cholesterol per head of population increased with age and the absolute burden was concentrated around old age. The contribution from ischaemic heart disease dominated across all ages (Figure 4.10).

Table 4.8: Deaths and burden (DALYs) attributable to high blood cholesterol by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Ischaemic heart disease	13,371	10.1	138,605	5.3
Stroke	1,980	1.5	24,986	0.9
Total attributable	15,351	11.6	163,591	6.2





Alcohol

Alcohol has both hazardous and protective effects on health, and the age and sex distribution of these effects varies in important ways. Of the 14 risk factors examined, alcohol was responsible for the greatest amount of burden in males under the age of 45 (Table 4.3).

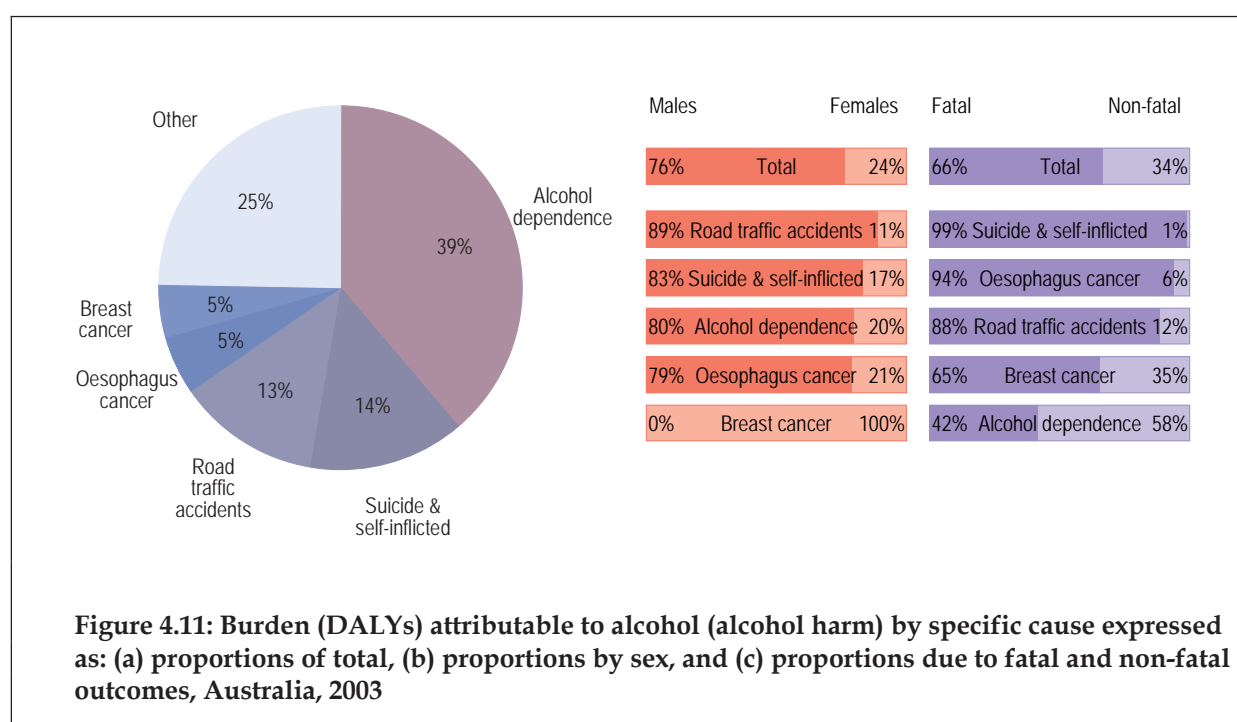
Alcohol harm was responsible for 3.2% of the total burden of disease and injury in Australia in 2003. Alcohol also prevented 0.9% per cent of the total burden in 2003 (Table 4.9). The benefits of alcohol consumption outweigh its harmful effects only in females over the age of 65. Given that the net impact of alcohol was to contribute to 2.3% of total burden, it is important to understand that, even though moderate intake of alcohol may have beneficial effects at middle and older ages, alcohol is harmful when taken in excess at all ages.

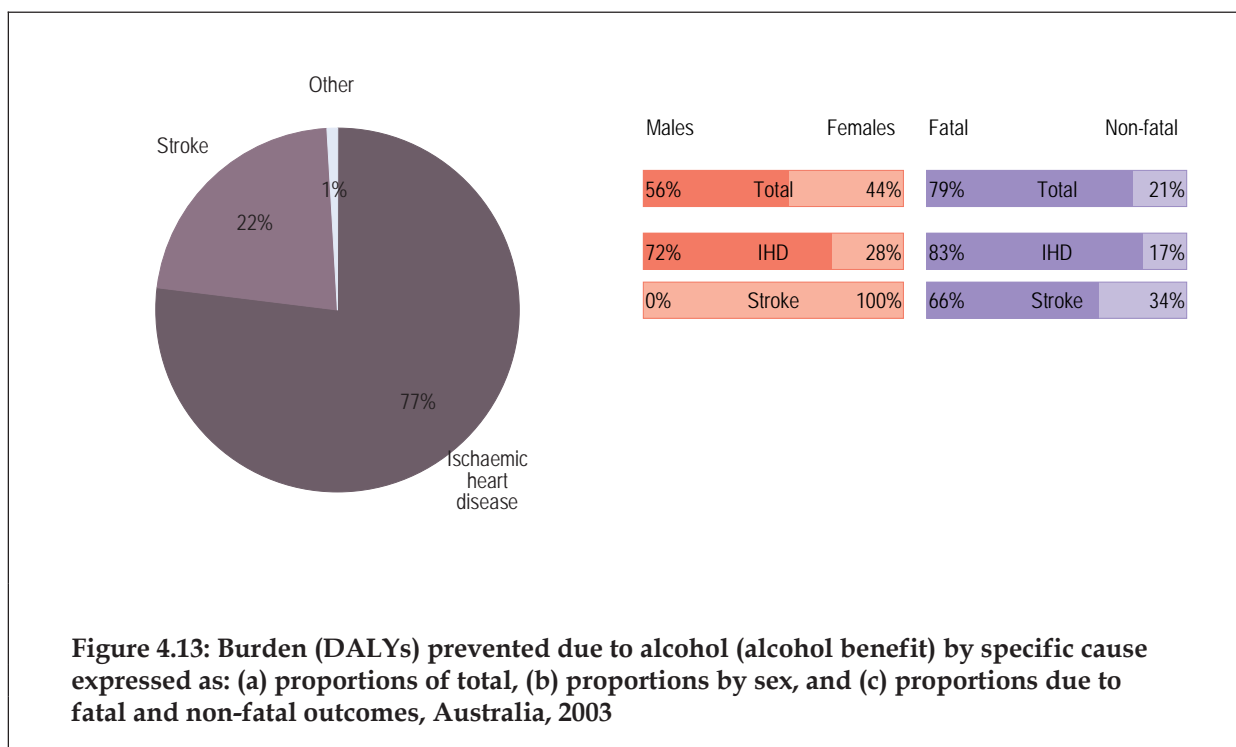
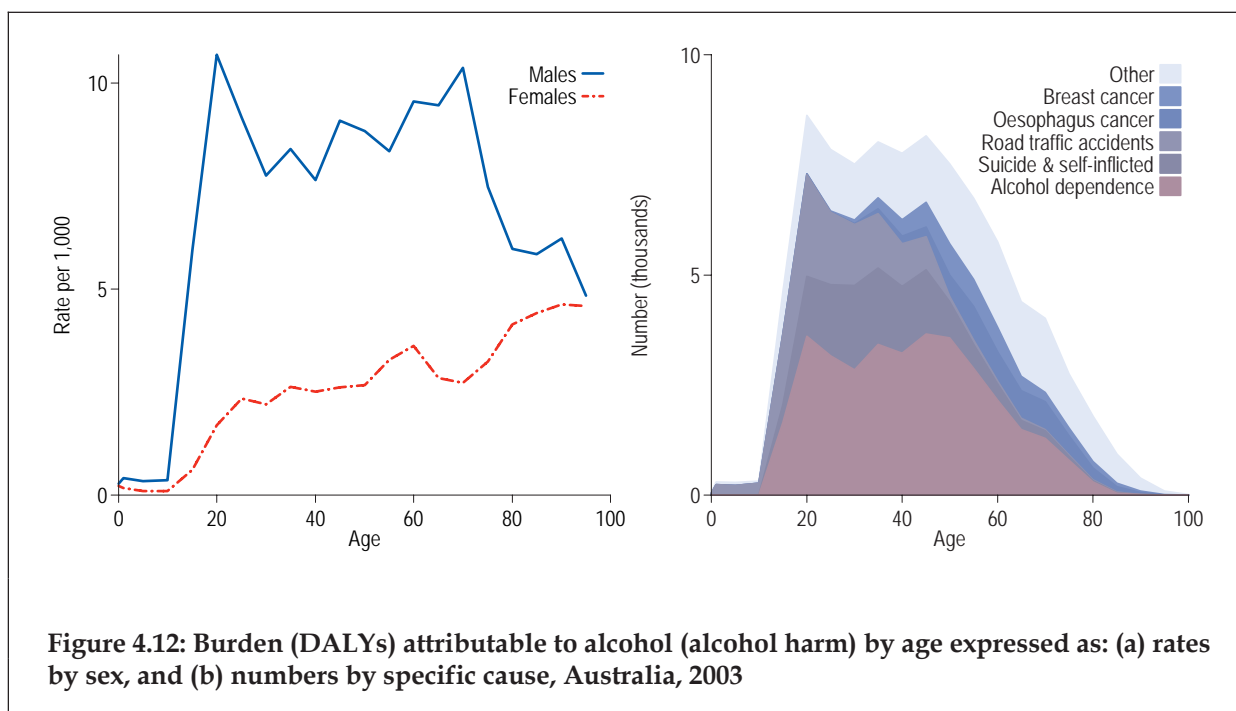
Alcohol abuse, road traffic accidents and suicide contributed two-thirds of the harm attributed to alcohol (Figure 4.11).

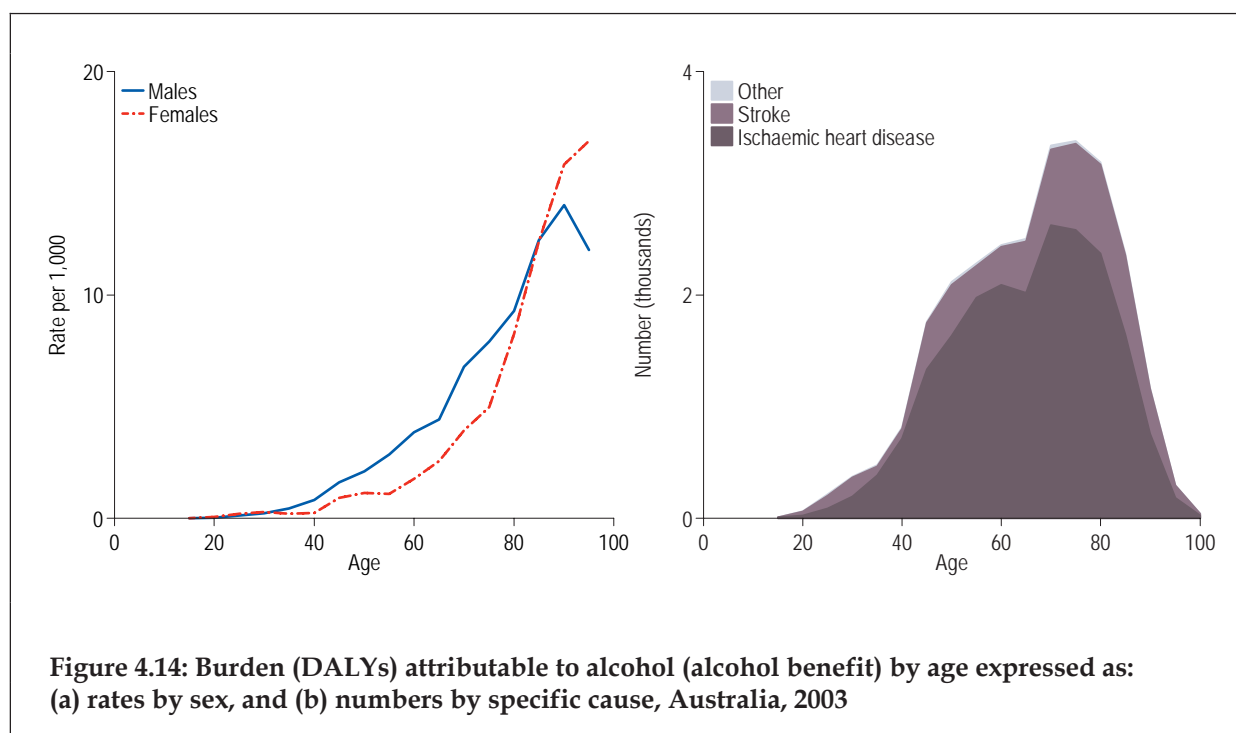
This study reports a substantially lower health benefit due to alcohol compared to the previous Australian burden study (AIHW: Mathers et al. 1999, AIHW: Ridolfo & Stevenson 2001) with only an estimated 2,346 deaths being prevented in 2003 compared to 7,157 deaths in 1996. This is due to the previous study underestimating the number of people who abstain from alcohol or drink less than 0.25 drinks per day.

Table 4.9: Deaths and burden (DALYs) attributable to alcohol by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Harm				
Alcohol abuse	918	0.7	34,116	1.3
Suicide & self-inflicted injuries	553	0.4	12,245	0.5
Road traffic accidents	396	0.3	11,121	0.4
Oesophagus cancer	368	0.3	4,594	0.2
Breast cancer	184	0.1	4,152	0.2
Other	1,012	0.8	19,207	0.7
<i>Total attributable harm</i>	3,430	2.6	85,435	3.2
Benefit				
Ischaemic heart disease	-1,950	-1.5	-20,659	-0.8
Stroke	-380	-0.3	-3,451	-0.1
Other	-16	0.0	-233	0.0
<i>Total attributable benefit</i>	-2,346	-1.8	-24,343	-0.9
Total attributable	1,084	0.8	61,091	2.3







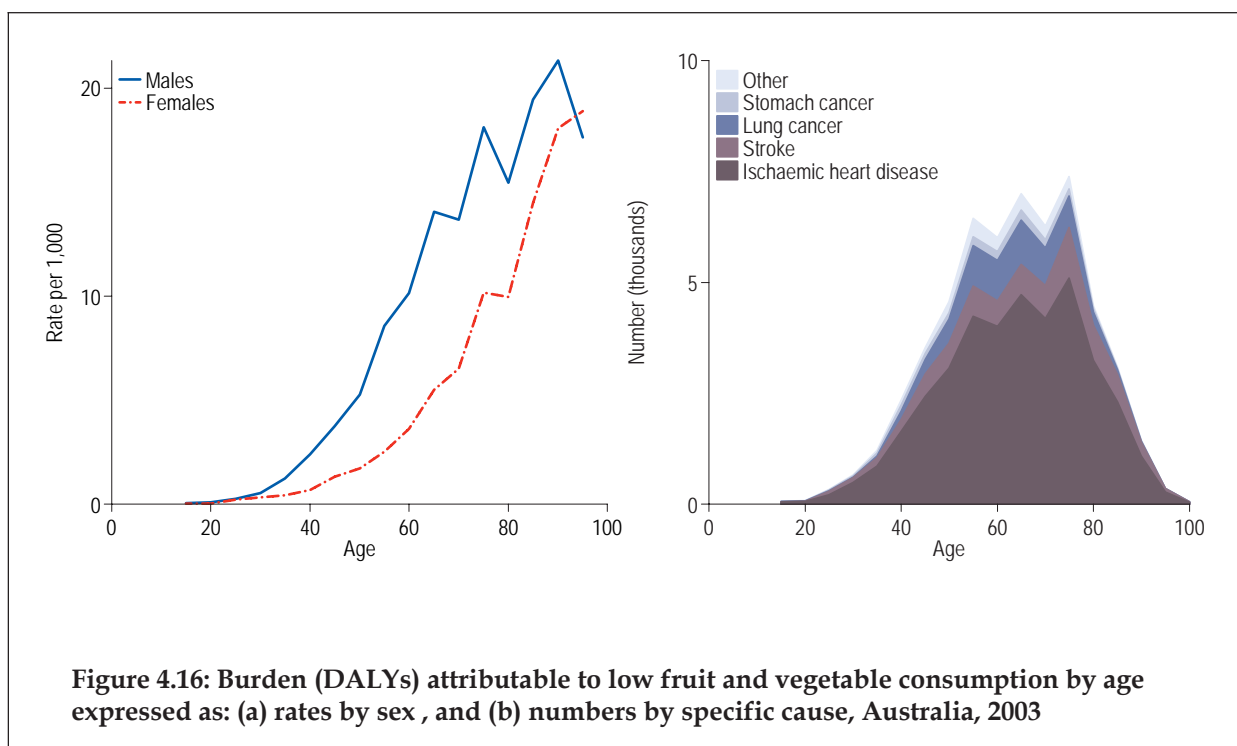
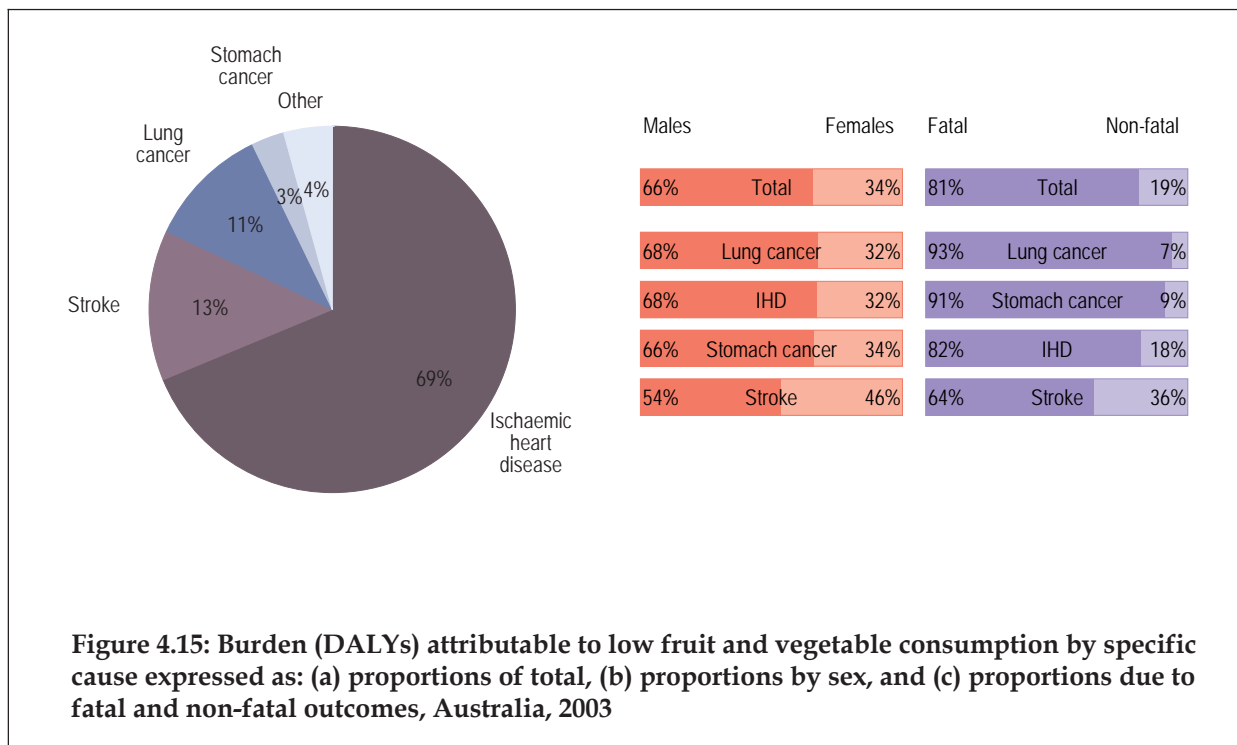
Low fruit and vegetable consumption

Low fruit and vegetable consumption was responsible for 2.1% of the total burden of disease and injury in Australia in 2003 (Table 4.10). Eating enough fruit and vegetables helps to prevent cancers, ischaemic heart disease and, to a lesser extent, stroke. Sixty-nine per cent of the burden from low fruit and vegetable consumption was due to ischaemic heart disease and two-thirds was experienced by males, partly because males tend to eat less fruit and vegetables than females, but also because males have a higher burden from ischaemic heart disease than females. Overall, 81% of the burden from low fruit and vegetable consumption was due to mortality.

The absolute burden from low fruit and vegetable consumption peaked between the age of 60 and 80 while the rate per head of population continued to increase until old age. The contribution from ischaemic heart disease dominated at all ages (Figure 4.16).

Table 4.10: Deaths and burden (DALYs) attributable to low fruit and vegetable consumption by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Ischaemic heart disease	3,219	2.4	37,981	1.4
Stroke	605	0.5	7,346	0.3
Lung cancer	463	0.3	5,956	0.2
Other	281	0.2	3,977	0.2
Total attributable	4,568	3.5	55,259	2.1



Illicit drugs

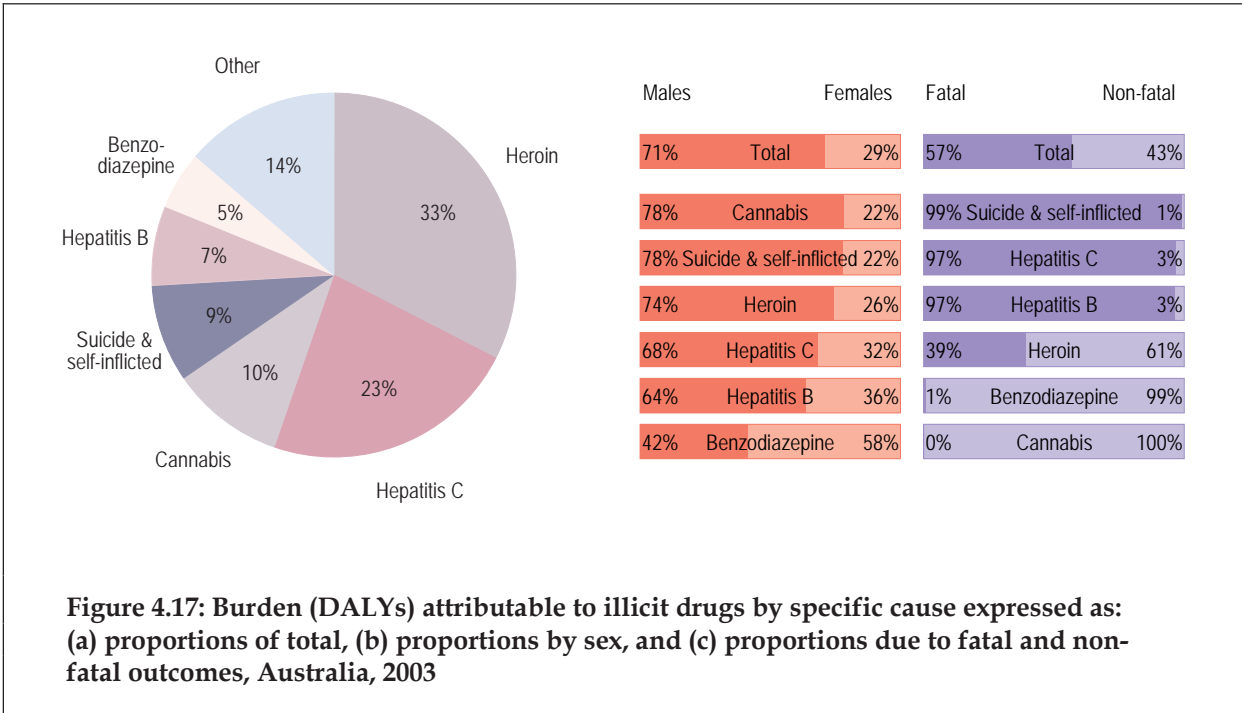
Illicit drugs were responsible for 2.0% of the total burden of disease and injury in Australia in 2003 (Table 4.11). Illicit drugs are a direct cause of death and disability as well as being

risk factors for conditions such as HIV/AIDS, hepatitis, low birth weight, inflammatory heart disease, poisoning, and suicide and self-inflicted injuries. Almost three-quarters of the burden from illicit drugs was experienced by males because males are more likely to both use illicit drugs and adopt drug habits that put them at risk of dying. Overall, fifty-seven per cent of the burden from illicit drugs was due to mortality (Figure 4.17).

The burden from illicit drugs, both in terms of rate per head of population and in absolute terms, peaked in early adulthood when drug addiction usually begins. The contribution from heroin dominated at this age but was overtaken by contributions from hepatitis B and C with increasing age as the long-term effects of drug use begin to manifest (Figure 4.18).

Table 4.11: Deaths and burden (DALYs) attributable to illicit drugs by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Heroin & polydrug abuse	263	0.2	16,758	0.6
Hepatitis C	759	0.6	11,709	0.4
Cannabis abuse	0	0.0	5,206	0.2
Suicide & self-inflicted injuries	204	0.2	4,458	0.2
Hepatitis B	329	0.2	3,637	0.1
Benzodiazepine abuse	1	0.0	2,656	0.1
Other	149	0.1	7,040	0.3
Total attributable	1,705	1.3	51,463	2.0



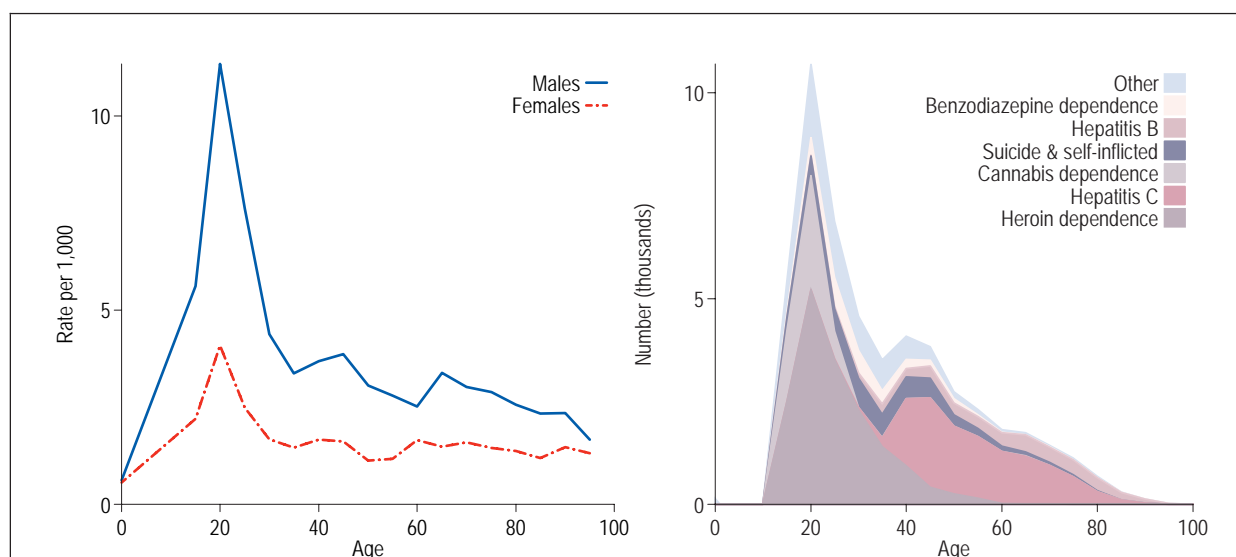


Figure 4.18: Burden (DALYs) attributable to illicit drugs by age expressed as: (a) rates by sex, and (b) numbers by specific cause, Australia, 2003

Occupational exposures and hazards

Occupational exposures and hazards were responsible for 2.0% of the total burden of disease and injury in Australia in 2003 (Table 4.12). More than two-thirds of this burden was experienced by males, mostly because occupational exposures and hazards occur in industries dominated by male employment. Females, however, experienced 86% of the burden from occupational overuse syndrome (OOS). Overall, 43% of the burden from occupational exposures and hazards was due to mortality (Figure 4.19).

The burden from occupational exposures and hazards was concentrated in the working ages and peaked in middle age, both in terms of rate per head of population and in absolute terms (Figure 4.20).

Table 4.12: Deaths and burden (DALYs) attributable to occupational exposures and hazards by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Cancer	1,154	0.9	15,559	0.6
Back pain	1	0.0	7,806	0.3
Occupational overuse syndrome	—	—	4,944	0.2
COPD	111	0.1	4,563	0.2
Road traffic accidents	124	0.1	2,975	0.1
Other	264	0.2	15,515	0.6
Total attributable	1,654	1.3	51,362	2.0

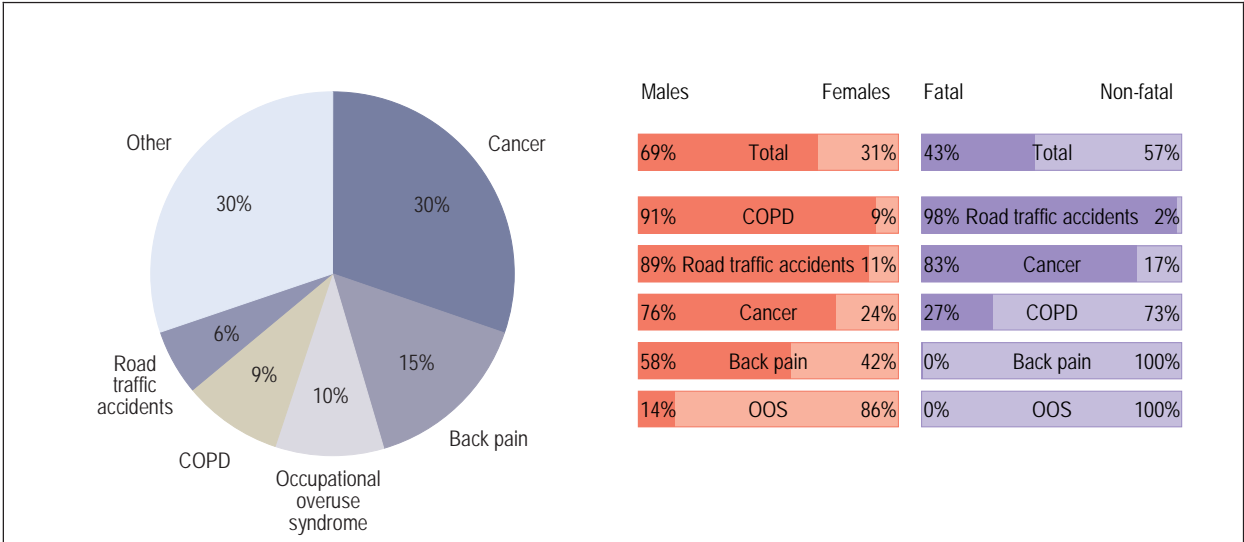


Figure 4.19: Burden (DALYs) attributable to occupational exposures and hazards by specific cause expressed as: (a) proportions of total, (b) proportions by sex, and (c) proportions due to fatal and non-fatal outcomes, Australia, 2003

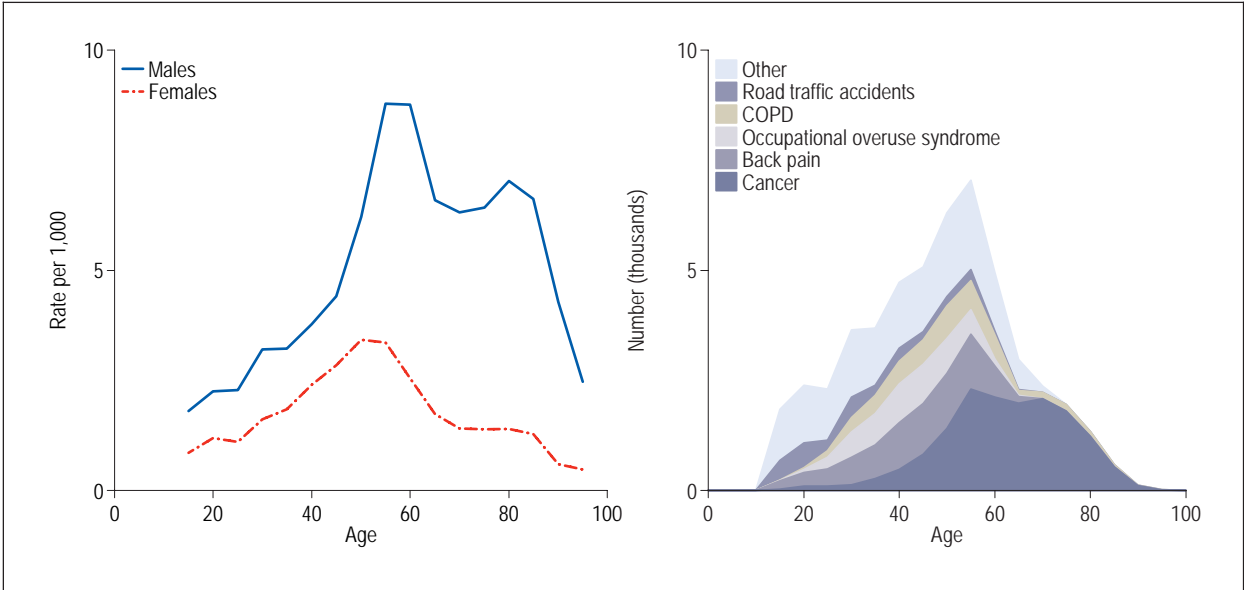


Figure 4.20: Burden (DALYs) attributable to occupational exposures and hazards by age expressed as: (a) rates by sex, and (b) numbers by specific cause, Australia, 2003

Intimate partner violence

The attribution of burden to intimate partner violence was attempted only for females due to insufficient evidence on prevalence and risk among males. While this risk is unlikely to be zero, it is probably small in comparison with the risk experienced by females. Intimate partner violence was responsible for 1.1% of the total burden of disease and injury in Australia in 2003 (Table 4.13). Of the 14 risk factors examined, intimate partner violence contributed most to the burden in females under the age of 45 (Table 4.3). Most of the burden from intimate partner violence was due to anxiety and depression, and conditions arising due to the associated increased use of tobacco, alcohol and illicit substances (Figure 4.21).

The burden from intimate partner violence, both in terms of rate per head of population and in absolute terms, peaked at around age 30 then declined with age (Figure 4.22). The contribution from anxiety and depression dominated throughout adulthood but was overtaken by contributions from tobacco-related disease with increasing age as the effects of higher smoking rates begin to manifest.

Table 4.13: Deaths and burden (DALYs) for females attributable to intimate partner violence by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Anxiety & depression	3	0.0	18,358	0.7
Suicide & self-inflicted injuries	131	0.1	3,099	0.1
Lung cancer	89	0.1	1,477	0.1
Homicide & violence	35	0.0	1,260	0.0
COPD	49	0.0	1,114	0.0
Other	128	0.1	4,051	0.2
Total attributable	435	0.3	29,360	1.1

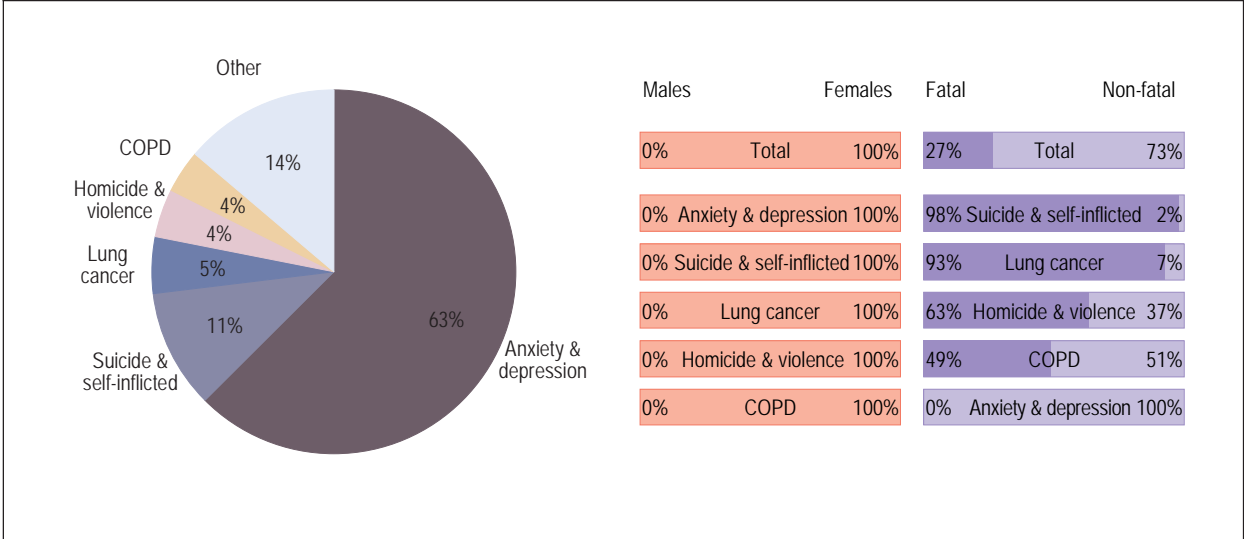


Figure 4.21: Burden (DALYs) attributable to intimate partner violence by specific cause expressed as: (a) proportions of total, (b) proportions by sex, and (c) proportions due to fatal and non-fatal outcomes, Australia, 2003

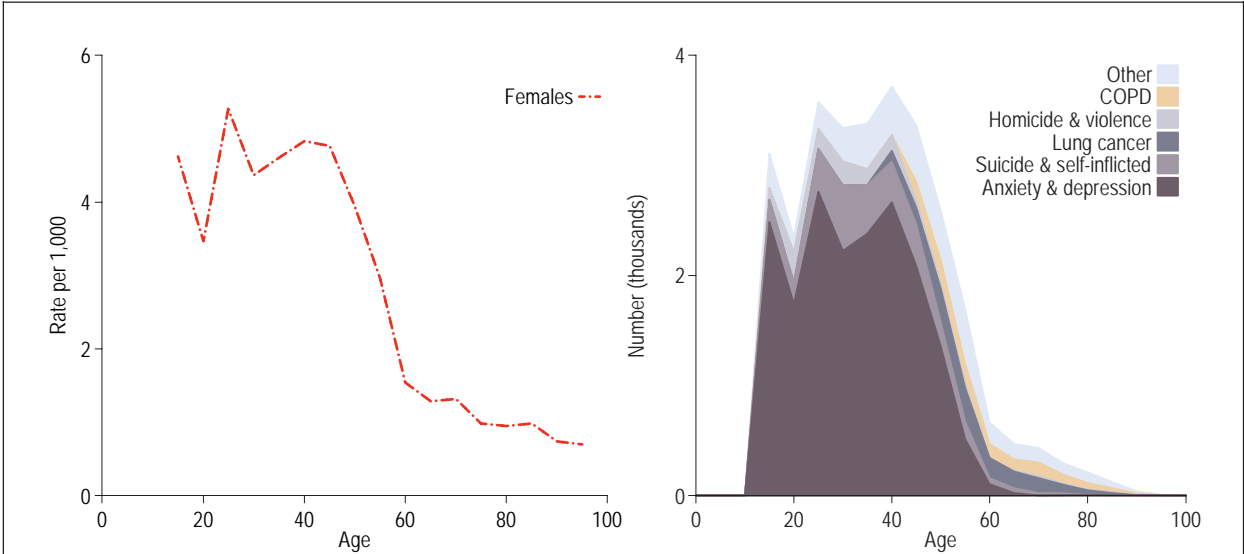


Figure 4.22: Burden (DALYs) attributable to intimate partner violence by age expressed as: (a) rates by sex, and (b) numbers by specific cause, Australia, 2003

Child sexual abuse

Child sexual abuse was responsible for 0.9% of the total burden of disease and injury in Australia in 2003 (Table 4.14). Ninety-four per cent of this burden was due to anxiety and

depression, suicide and self-inflicted injuries, and alcohol abuse. Of the 14 risk factors examined, child sexual abuse was the second leading cause of burden in females under the age of 45 (Table 4.3). Just over four-fifths of the burden from child sexual abuse was experienced by females and 14% was due to mortality (Figure 4.23).

The burden from child sexual abuse, both in terms of rate per head of population and in absolute terms, peaked at around 40 years-old then declined with age. The contribution from anxiety and depression dominated at this age after which contributions from suicide and self-inflicted injuries and alcohol abuse became increasingly important (Figure 4.24).

Table 4.14: Deaths and burden (DALYs) attributable to child sexual abuse by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Anxiety and depression	7	0.0	19,133	0.7
Suicide & self-inflicted injuries	103	0.1	2,258	0.1
Alcohol abuse	24	0.0	730	0.0
Other	62	0.0	1,392	0.1
Total attributable	196	0.1	23,513	0.9

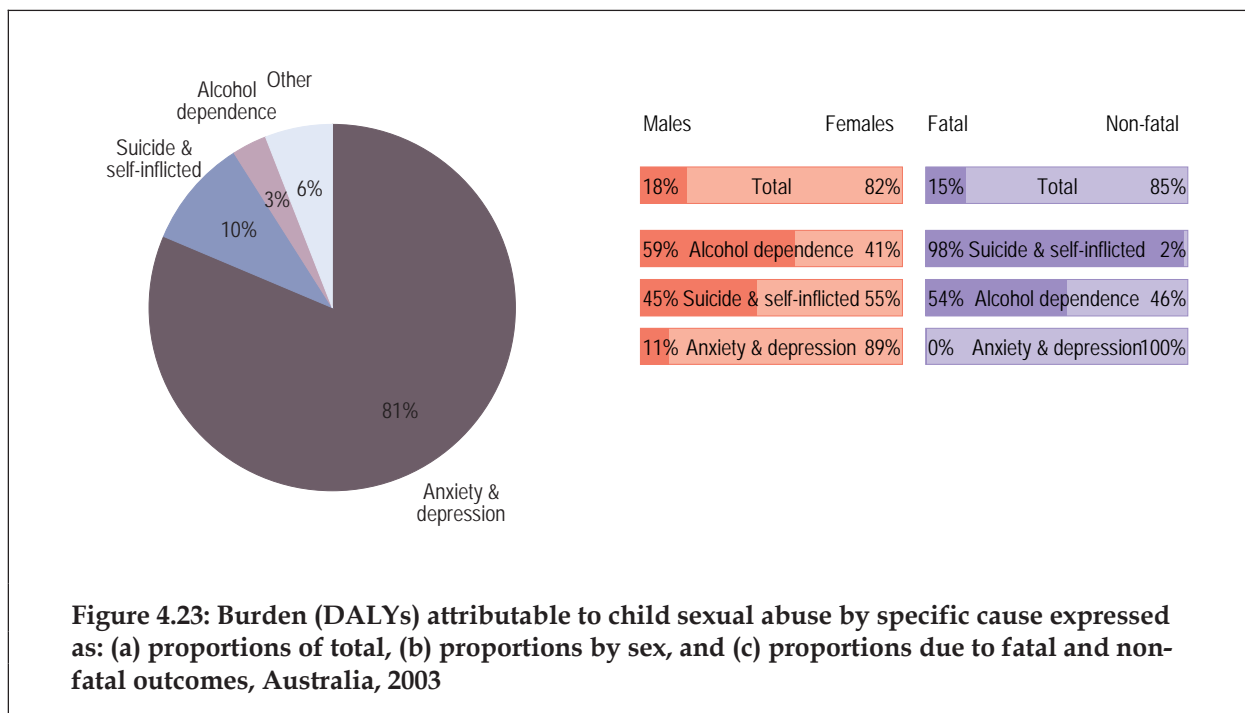
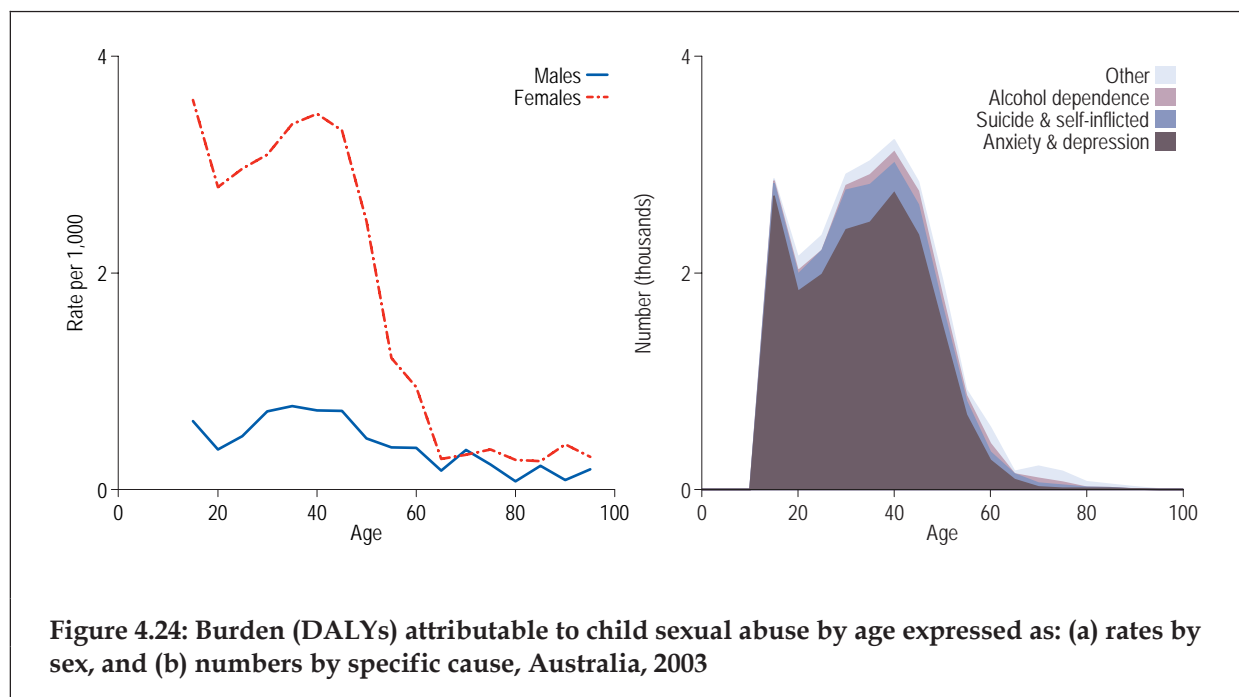


Figure 4.23: Burden (DALYs) attributable to child sexual abuse by specific cause expressed as: (a) proportions of total, (b) proportions by sex, and (c) proportions due to fatal and non-fatal outcomes, Australia, 2003



Urban air pollution

The health effects of urban air pollution are largely chronic conditions (such as ischaemic heart disease, lung cancer and stroke) resulting from long-term exposure to this risk. There may also be an additional burden from short-term exposure to abnormally high levels of urban air pollution, although this risk is more controversial. Table 4.15 provides estimates for both long-term and short-term effects; all other figures in this section reflect the long-term effects only. Urban air pollution was responsible for 1.0% of the total burden of disease and injury in Australia in 2003 (Table 4.15). Sixty-two per cent of the burden from urban air pollution was due to cardiovascular disease (ischaemic heart disease and stroke) and 53% of the burden from urban air pollution was experienced by males. Overall, 80% of the burden from urban air pollution was due to mortality (Figure 4.25).

The absolute burden from urban air pollution peaked at age 80 while the rate per head of population continued to increase until old age. The contribution from cardiovascular disease dominated at all ages (Figure 4.26).

Table 4.15: Deaths and burden (DALYs) attributable to urban air pollution by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Long-term				
Ischaemic heart disease	959	0.7	8,483	0.3
Lung cancer	351	0.3	4,115	0.2
Stroke	432	0.3	3,738	0.1
COPD	184	0.1	2,654	0.1
Other	83	0.1	748	0.0
<i>Total attributable to long-term exposure</i>	<i>2,009</i>	<i>1.5</i>	<i>19,738</i>	<i>0.7</i>
Short-term				
<i>Total attributable to short-term exposure</i>	<i>1,046</i>	<i>0.8</i>	<i>7,781</i>	<i>0.3</i>
Total attributable	3,056	2.3	27,519	1.0

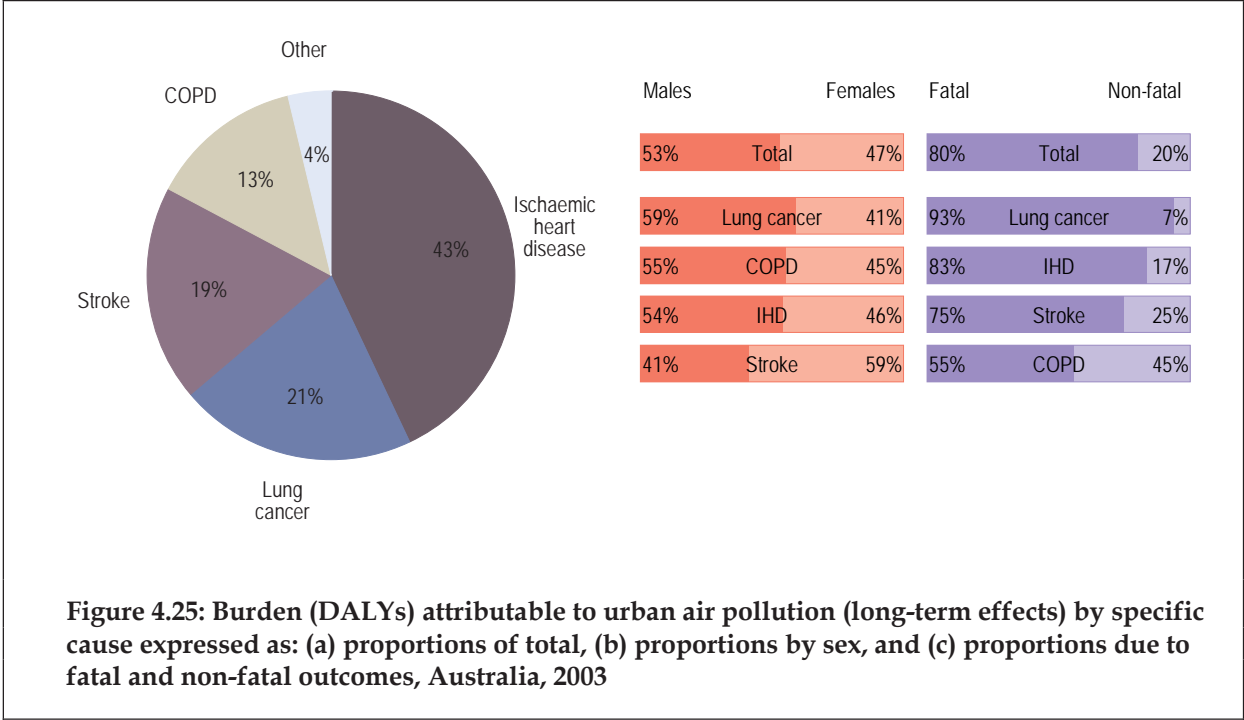


Figure 4.25: Burden (DALYs) attributable to urban air pollution (long-term effects) by specific cause expressed as: (a) proportions of total, (b) proportions by sex, and (c) proportions due to fatal and non-fatal outcomes, Australia, 2003

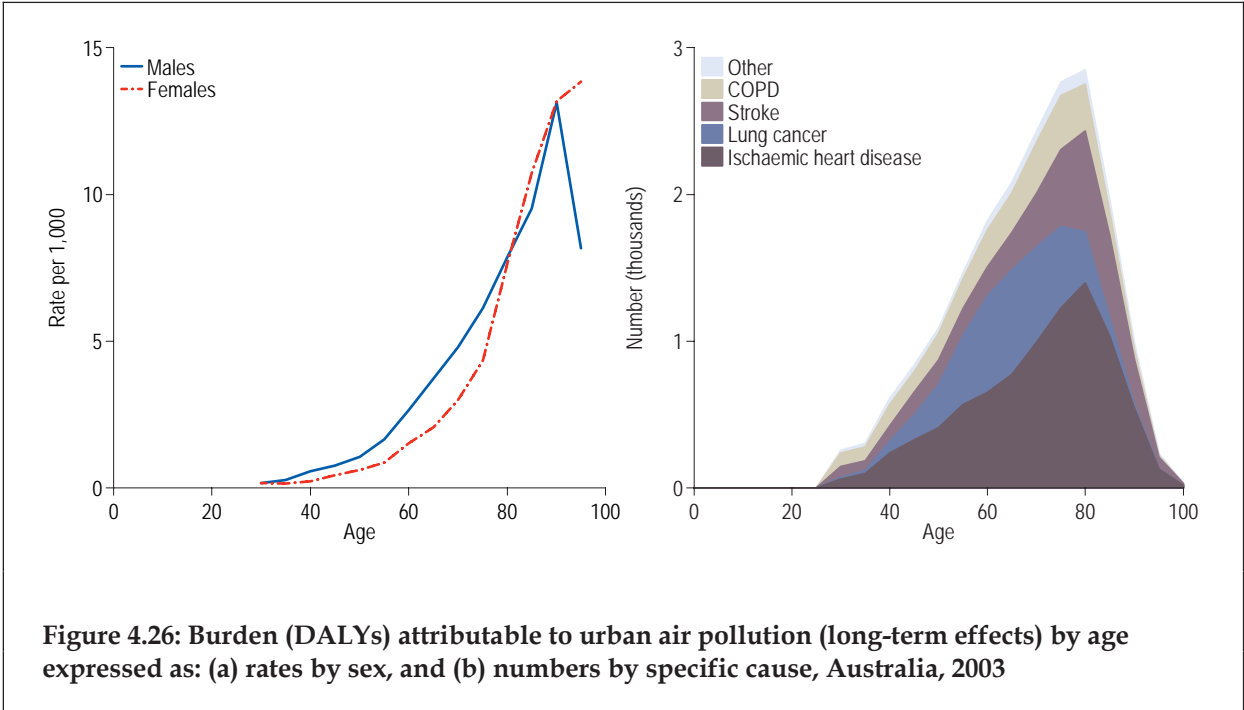


Figure 4.26: Burden (DALYs) attributable to urban air pollution (long-term effects) by age expressed as: (a) rates by sex, and (b) numbers by specific cause, Australia, 2003

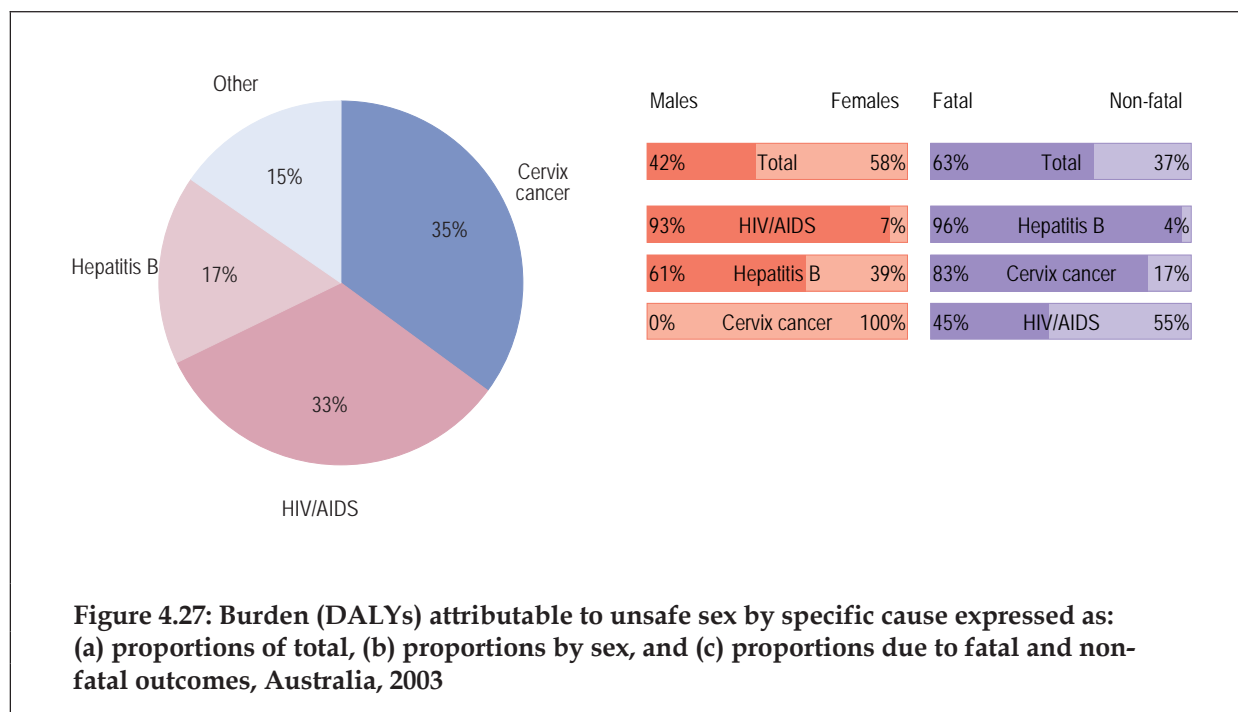
Unsafe sex

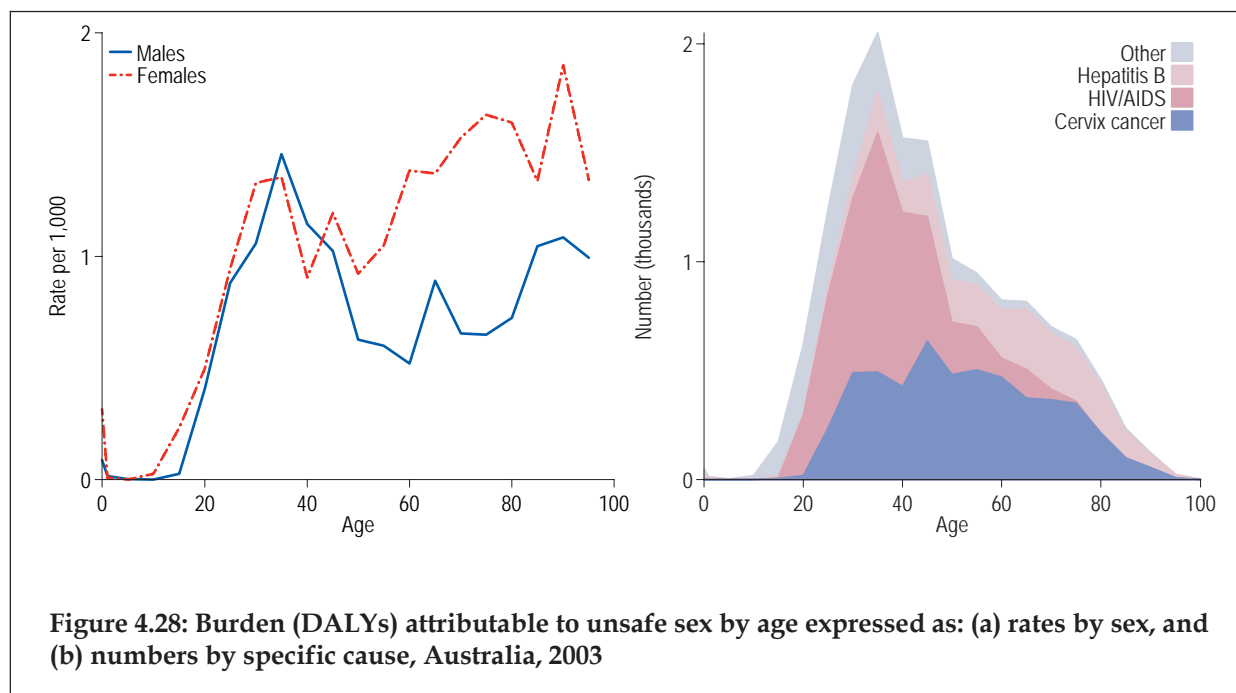
Unsafe sex was responsible for 0.6% of the total burden of disease and injury in Australia in 2003 (Table 4.16). Over two-thirds of this burden was due to cervix cancer and HIV/AIDS. Sixty-three per cent of the burden from unsafe sex was due to mortality (Figure 4.27).

The burden from unsafe sex in males peaked in early adulthood due to the impact of HIV infection, after which it declined and the long-term effects of hepatitis B infection began to manifest. In females, the rate per head of population continued to increase with age and the absolute burden was concentrated around middle age when the contribution from cervix cancer dominated (Figure 4.28).

Table 4.16: Deaths and burden (DALYs) attributable to unsafe sex by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Cervix cancer	298	0.2	5,231	0.2
HIV/AIDS	105	0.1	4,873	0.2
Hepatitis B	225	0.2	2,499	0.1
Other	26	0.0	2,293	0.1
Total attributable	655	0.5	14,897	0.6





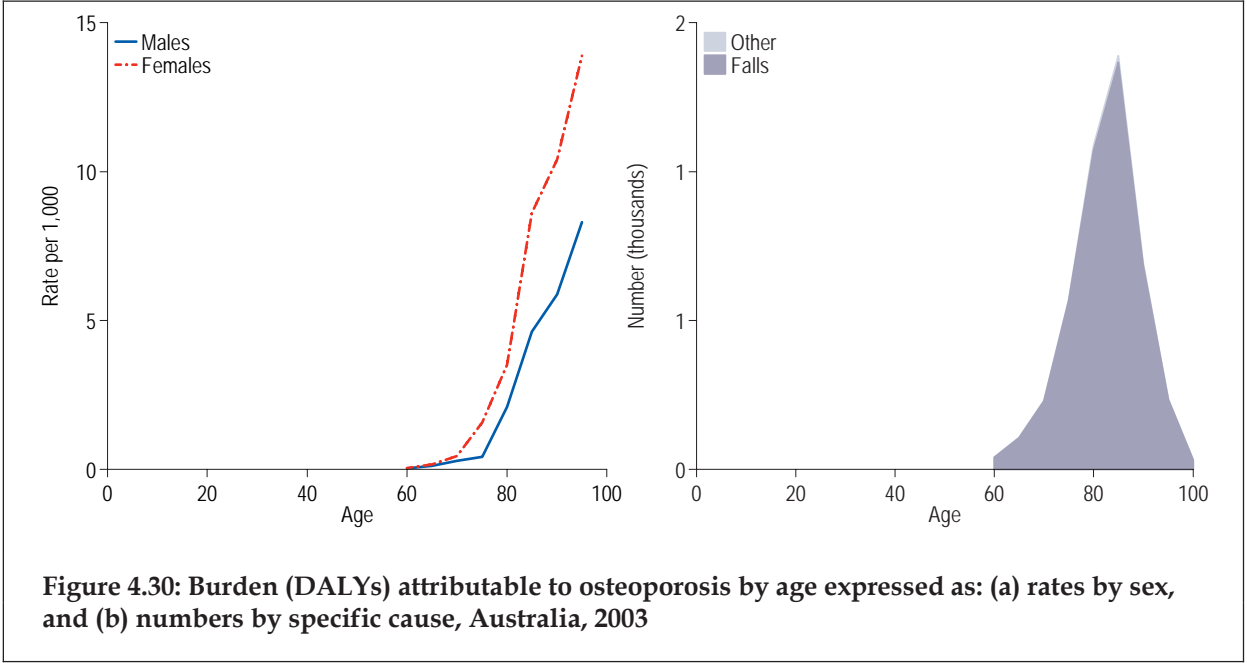
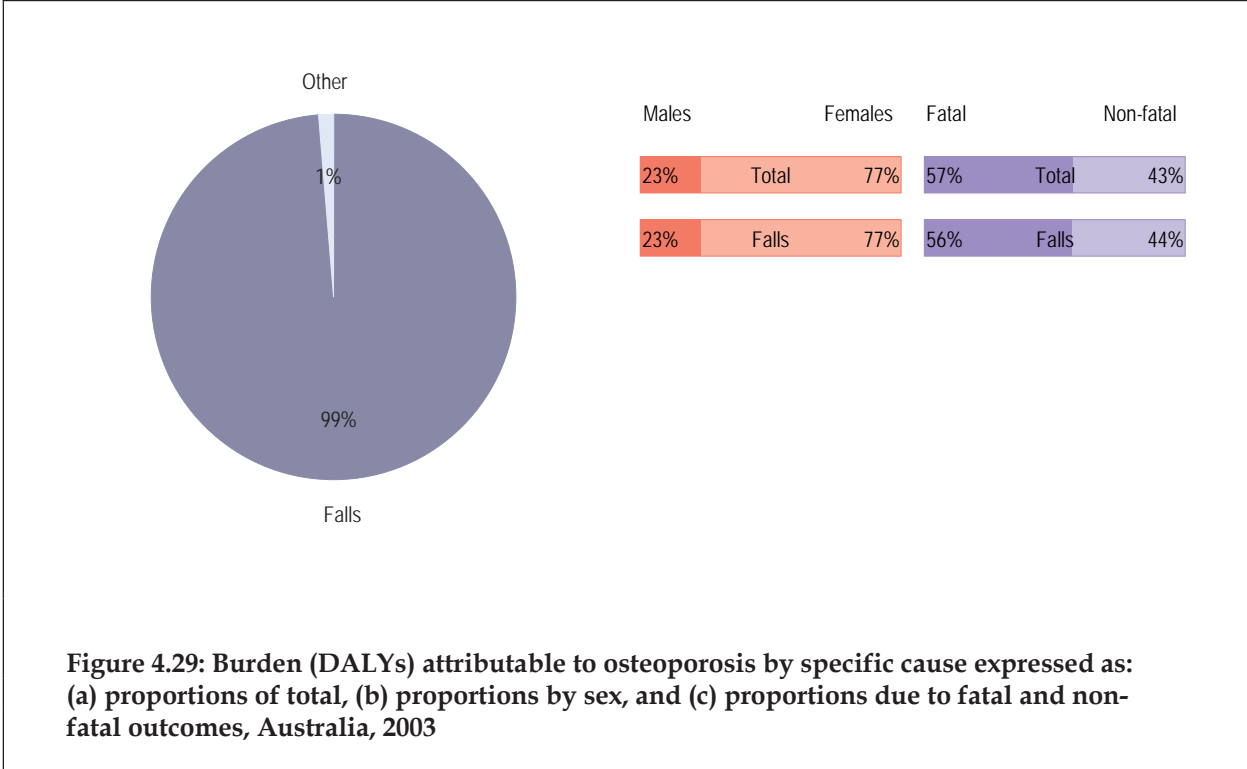
Osteoporosis

Osteoporosis was responsible for 0.2% of the total burden of disease and injury in Australia in 2003 (Table 4.17). Almost all of this burden was due to falls and more than three-quarters was experienced by females. More than half of the burden from osteoporosis was due to mortality (Figure 4.29).

The burden from osteoporosis was experienced from age 60 onwards. The contribution from falls dominated at all ages (Figure 4.30).

Table 4.17: Deaths and burden (DALYs) attributable to osteoporosis by specific cause, Australia, 2003

Specific cause	Deaths		DALYs	
	Number	Per cent of total	Number	Per cent of total
Falls	534	0.4	4,329	0.2
Other	10	0.0	58	0.0
Total attributable	545	0.4	4,386	0.2



5 Differentials in burden of disease and injury across Australia

5.1 Overview

This chapter describes differentials in burden of disease and injury across Australia in terms of the following stratifications of the population: state and territory jurisdictions, socioeconomic quintiles and remoteness categories (major cities, regional and remote). The chapter begins by comparing life expectancy and health-adjusted life expectancy (HALE) across each subpopulation within these strata. It then discusses the main differentials between subpopulations by leading causes of burden. Table 5.1 summarises for each subpopulation the important demographic characteristics that influence these differentials.

Table 5.1: Selected demographic characteristics by area, Australia, 2003

Area	Population ^(a)		Per cent of population for area						
	('000)	Per cent of Australia	Age group (years)				Males	Indigenous ^(b)	Low SES ^(c)
			<15	15–59	60–79	80+			
Jurisdiction									
NSW	6,687.5	33.6	19.9	62.4	14.2	3.5	49.7	2.0	19.0
Vic	4,918.0	24.7	19.5	62.9	14.1	3.4	49.3	0.6	16.0
Qld	3,797.3	19.1	20.8	62.9	13.3	3.0	49.9	3.3	23.9
WA	1,952.5	9.8	20.4	64.0	12.8	2.8	50.0	3.3	22.5
SA	1,527.6	7.7	18.8	61.7	15.4	4.1	49.5	1.6	14.9
Tas	477.2	2.4	20.4	60.6	15.4	3.7	49.3	3.6	55.4
ACT	322.9	1.6	19.8	67.4	10.7	2.2	49.4	1.2	0.3
NT	198.4	1.0	25.4	67.4	6.5	0.7	52.5	28.8	28.4
Socioeconomic quintile									
Low	3,917.1	19.7	21.9	61.5	13.7	2.9	50.0	n.a.	n.a.
Mod. low	3,973.8	20.0	21.2	60.6	14.9	3.3	49.8	n.a.	n.a.
Average	3,747.8	18.8	20.3	61.9	14.3	3.4	49.8	n.a.	n.a.
Mod. high	4,097.3	20.6	19.4	64.5	13.0	3.1	49.6	n.a.	n.a.
High	4,145.4	20.8	17.4	65.4	13.5	3.7	49.1	n.a.	n.a.
Remoteness									
Major cities	13,347.9	66.8	19.1	64.4	13.5	3.3	49.6	1.0	17.2
Regional	6,050.5	30.4	21.7	59.7	15.3	3.4	50.0	3.2	23.7
Remote	483.1	2.4	25.6	63.4	9.2	1.8	53.2	25.7	39.2
Australia	19,894.7	100.0	20.0	62.8	13.9	3.3	49.6	2.3	19.7

(a) Estimated resident population figures as at 30 June 2003 (ABS cat. no. 3201.0).

(b) Based on people identifying as Indigenous in the 2001 Census (ABS cat. no. 2019.0 – 2019.8).

(c) Based on Socio-Economic Indexes for Areas (SEIFA) (ABS cat. no. 2039.0.55.001).

5.2 Health-adjusted life expectancy

HALE provides an estimate of the average years of equivalent 'healthy' life that a person can expect to live at various ages. HALE is related to life expectancy, which provides an estimate of the average years of life a person can expect to live at various ages given current risks of mortality. HALE extends this concept by reducing the estimated duration by the proportion of time spent at each age in states less than perfect health, adjusted for the relative severity of those health states. The sum of prevalent years lost due to disability (PYLD) across all causes is used to derive this 'severity-weighted' proportion for each age. Since the starting point for HALE is a life table, life expectancy at birth for the various subpopulations discussed in this chapter is presented first in Table 5.2.

Table 5.2: Life expectancy at birth by area and sex, Australia, 2003

Area	Life expectancy at birth (years)					
	Males		Females		Persons	
Jurisdiction						
NSW	78.2	(78.0–78.3)	83.1	(83.0–83.3)	80.6	(80.5–80.8)
Vic	78.6	(78.4–78.8)	83.2	(83.0–83.4)	80.9	(80.8–81.0)
Qld	78.4	(78.2–78.6)	83.3	(83.1–83.5)	80.8	(80.7–81.0)
WA	79.0	(78.7–79.3)	83.7	(83.4–84.0)	81.3	(81.1–81.5)
SA	77.7	(77.3–78.0)	82.9	(82.5–83.2)	80.3	(80.0–80.5)
Tas	76.7	(76.1–77.3)	81.7	(81.1–82.2)	79.2	(78.8–79.6)
ACT	80.2	(79.4–80.9)	84.2	(83.4–84.9)	82.3	(81.7–82.8)
NT	73.1	(72.2–74.0)	78.6	(77.6–79.6)	75.5	(74.8–76.1)
Socioeconomic quintile						
Low	76.9	(76.7–77.1)	82.3	(82.1–82.5)	79.6	(79.4–79.7)
Moderately low	77.4	(77.2–77.6)	82.8	(82.6–83.0)	80.0	(79.9–80.2)
Average	77.7	(77.5–77.9)	82.7	(82.5–82.9)	80.2	(80.0–80.3)
Moderately high	79.0	(78.8–79.2)	83.5	(83.3–83.7)	81.2	(81.1–81.4)
High	80.9	(80.6–81.1)	84.5	(84.3–84.7)	82.7	(82.5–82.8)
Remoteness						
Major cities	78.8	(78.7–78.9)	83.5	(83.4–83.6)	81.2	(81.1–81.2)
Regional	77.5	(77.4–77.7)	82.7	(82.5–82.8)	80.0	(79.9–80.1)
Remote	75.4	(74.8–76.1)	81.5	(80.9–82.2)	78.1	(77.6–78.6)
Australia	78.3	(78.2–78.4)	83.2	(83.1–83.3)	80.7	(80.7–80.8)

When interpreting the results presented in this chapter it is important to keep in mind that Indigenous people are a much greater proportion of the total population in the Northern Territory and remote areas of Australia. This accounts for the much greater health loss in these areas, although the contribution of Indigenous populations to this loss is not quantified in this report. Readers seeking such comparisons are referred to the separate report on the Indigenous component of this study. Once the Indigenous results are available separate small area comparisons can be made for non-Indigenous people. This is relevant to health

policy in that there is a raft of Indigenous health issues that is distinct from the health issues of the general population living in remote areas.

HALE was calculated for subpopulations using the PYLD estimated for each population separately, as discussed in Chapter 2. Total HALE at birth across Australia in 2003 was 70.6 years for males, 75.2 years for females and 72.9 years for both sexes combined (Table 5.3). The figures for both sexes ranged from 67.7 to 75.9 years across state and territory jurisdictions, 71.2 to 75.5 years across socioeconomic quintiles and 69.5 to 73.5 years across remoteness categories.

Table 5.3: Health-adjusted life expectancy (HALE) and life expectancy at birth lost due to disability by area and sex, Australia, 2003

Area	Health-adjusted life expectancy (HALE) (years)						Life expectancy at birth lost due to disability (%)		
	At birth			At age 60			Males	Females	Persons
	Males	Females	Persons	Males	Females	Persons			
Jurisdiction									
NSW	70.5	75.3	72.9	17.1	20.6	18.9	9.8	9.5	9.6
Vic	71.1	75.4	73.2	17.5	20.8	19.2	9.6	9.4	9.5
Qld	70.5	75.3	72.8	17.0	20.4	18.7	10.1	9.7	9.9
WA	71.5	75.6	73.5	17.5	20.6	19.1	9.6	9.6	9.6
SA	69.3	74.2	71.7	16.4	20.0	18.3	10.8	10.5	10.6
Tas	68.8	73.7	71.3	16.3	19.7	18.1	10.2	9.8	10.0
NT	65.8	70.2	67.7	12.6	15.1	13.6	10.0	10.6	10.3
ACT	73.9	77.8	75.9	18.9	21.9	20.5	7.8	7.5	7.7
Socioeconomic quintile									
Low	68.7	73.8	71.2	16.1	19.7	17.9	10.7	10.4	10.6
Moderately low	69.5	74.6	72.0	16.4	20.1	18.2	10.2	9.9	10.1
Average	69.9	74.6	72.2	16.6	20.1	18.4	10.0	9.8	9.9
Moderately high	71.4	75.9	73.6	17.6	20.8	19.3	9.7	9.1	9.4
High	73.8	77.2	75.5	19.2	21.9	20.6	8.7	8.7	8.7
Remoteness									
Major cities	71.3	75.6	73.5	17.5	20.8	19.2	9.6	9.4	9.5
Regional	69.6	74.5	72.0	16.5	20.1	18.3	10.3	9.8	10.1
Remote	67.3	72.3	69.5	15.4	18.5	16.8	10.8	11.3	11.0
Australia	70.6	75.2	72.9	17.1	20.5	18.9	9.8	9.6	9.7

When the difference between life expectancy and HALE is expressed as a proportion of life expectancy, this represents the proportion of remaining life that is lost due to disability. Hereafter this is referred to as PLD (proportion of life expectancy lost due to disability). PLD at birth is the most commonly reported figure, although it can be calculated at any age and increases with age (Figure 5.1).

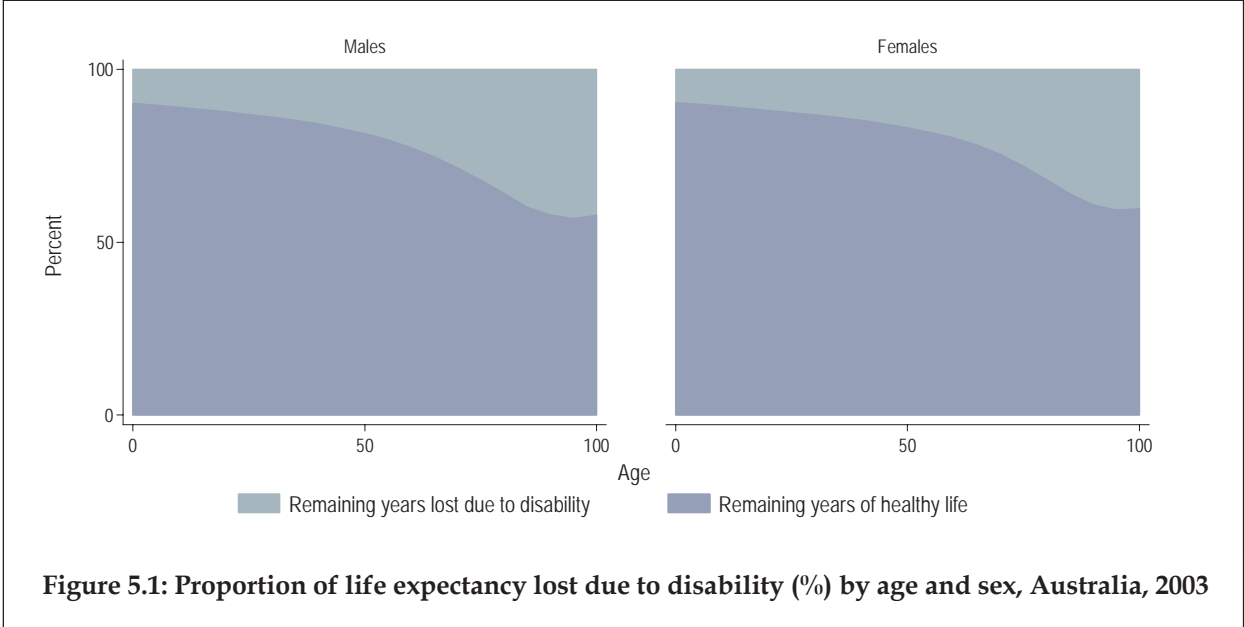


Figure 5.1: Proportion of life expectancy lost due to disability (%) by age and sex, Australia, 2003

This report shows for the first time that there are differentials in PLD at birth across Australia (Table 5.3). There was a strong socioeconomic gradient in this measure, with the lowest socioeconomic quintile losing 10.6% of life expectancy at birth through disability and the highest losing only 8.7%. Differentials with respect to remoteness category were also apparent but not as large, with remote areas losing 11.0% and major cities losing 9.5%. With respect to state and territory jurisdictions, the Australian Capital Territory had the lowest PLD at birth at 7.7% and South Australia had the highest at 10.6%.

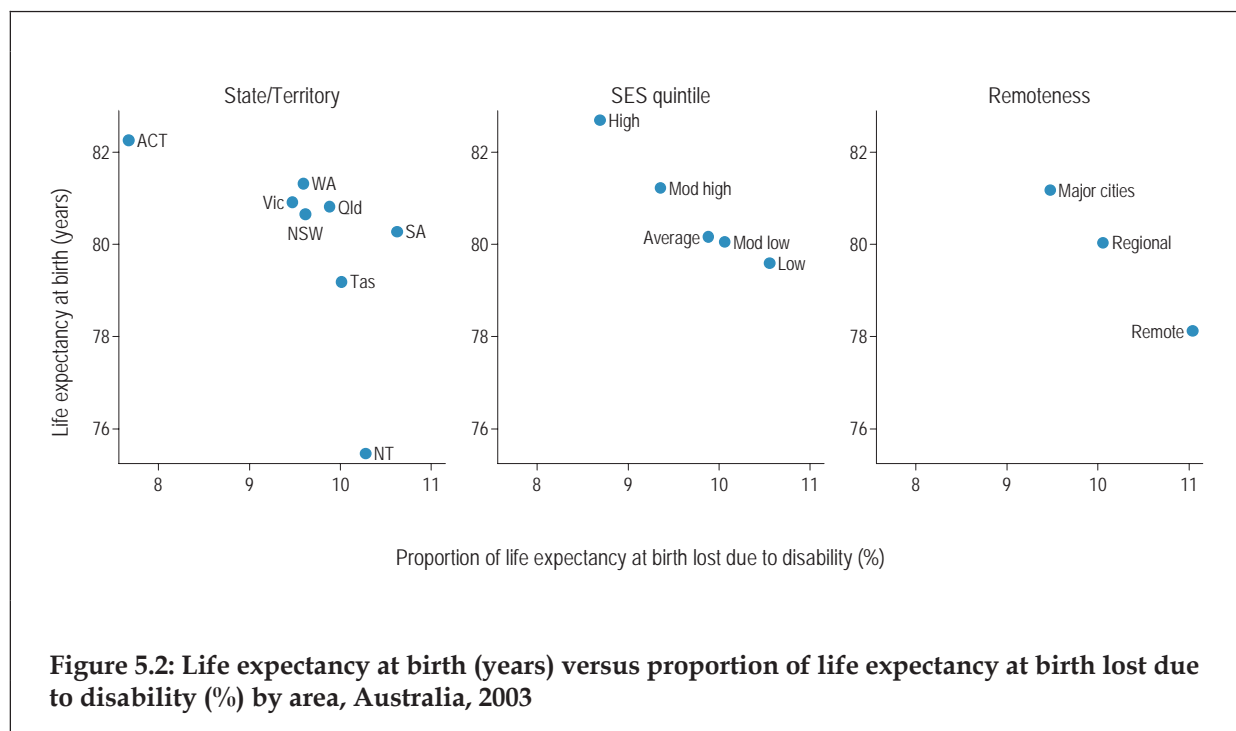


Figure 5.2 shows the inverse relationship between life expectancy at birth and PLD, with subpopulations experiencing the highest life expectancy also having the lowest PLD. In other words, longevity is associated with lower average levels of disability throughout the life span.

The remainder of this chapter presents health differentials across these subpopulations using the standard burden metric of disability-adjusted life years – (DALYs). All rates per head of population were standardised to remove the effect of different age structures between populations. This standard technique is used when comparing populations whereby the age-specific rates of the populations of interest are applied to the age structure of a reference population before comparisons are made.

5.3 State and territory differentials

The proportion of burden experienced by each state and territory jurisdiction was roughly proportional to the population size, with New South Wales accounting for the largest proportion (34.0%), followed by Victoria (24.8%) and Queensland (18.6%) (Table 5.4). Males experienced more of this burden than females in all jurisdictions except the Australian Capital Territory where it was more equally distributed between the sexes. In all jurisdictions except Tasmania, slightly more of total burden was due to non-fatal causes.

Table 5.4: Burden (DALYs) for state/territory jurisdictions by proportions of total, proportions by sex and proportions due to mortality, Australia, 2003

Area	DALYs ('000)	Per cent of total	Per cent male	Per cent fatal burden
NSW	895.8	34.0	51.9	49.5
Vic	651.6	24.8	50.9	48.9
Qld	488.5	18.6	53.0	46.9
SA	234.3	8.9	51.5	48.7
WA	236.8	9.0	51.7	46.6
Tas	73.4	2.8	51.6	51.4
NT	22.9	0.9	58.5	46.6
ACT	29.5	1.1	50.4	47.5
Australia	2,632.8	100.0	51.8	48.6

There were important differentials in burden experienced per head of population between jurisdictions. After age standardisation, the Northern Territory had almost twice the rate of total burden of the Australian Capital Territory for both males and females. This was due to higher rates of burden for most causes, but particularly for cardiovascular disease, diabetes and injuries (Figure 5.3).

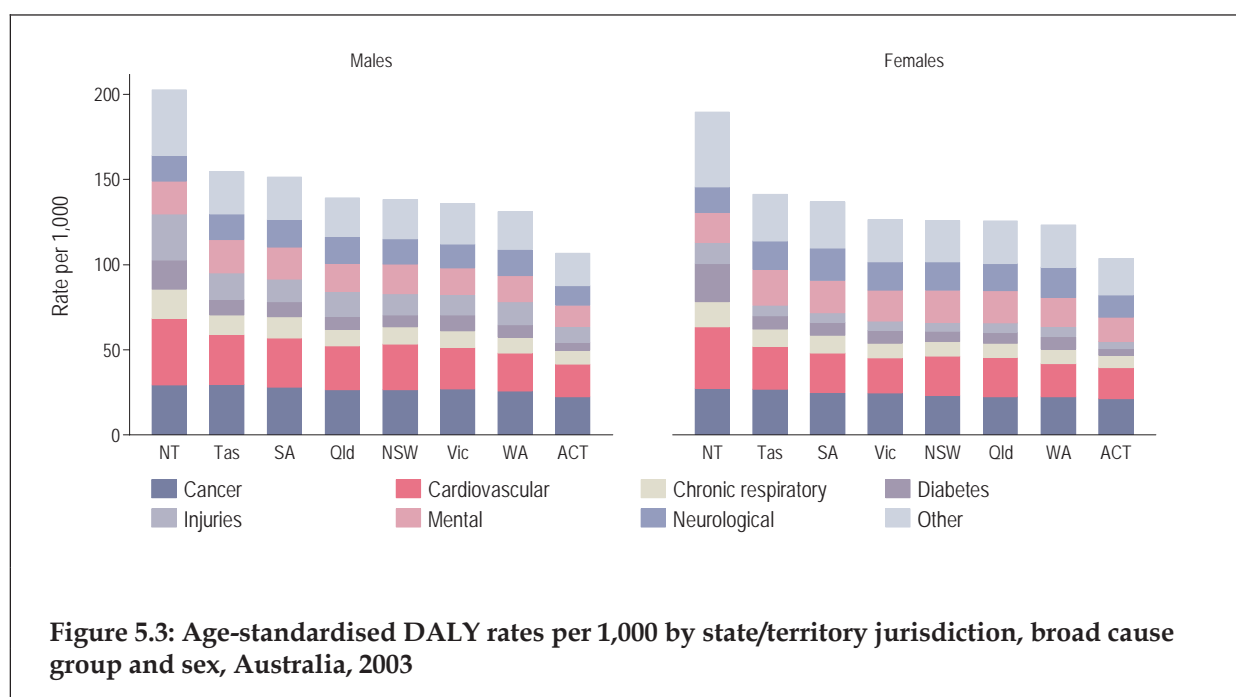


Table 5.5 provides a comparison between burden rates for jurisdictions and the national average for the 10 leading broad causes of burden in Australia for 2003. Of these causes, the greatest difference between jurisdictions with the lowest and highest rates occurred (in order of magnitude of difference) in diabetes, injuries, genitourinary conditions and chronic respiratory diseases. The causes that contributed most in terms of the absolute difference

observed between jurisdictions were cardiovascular disease (19.7%), diabetes (15.5%) and injuries (13.6% for intentional and unintentional combined).

Table 5.5: Differentials in burden (DALYs) by state/territory jurisdiction for the 10 leading broad cause groups, Australia, 2003

Broad cause group	Rate	Standardised rate ratio ^(b)								% diff.	% of
	Aust. ^(a)	NSW	Vic	Qld	SA	WA	Tas	NT	ACT	high/low ^(c)	total diff. ^(d)
Cancer	25.1	1.00	1.02	0.98	1.06	0.96	1.13	1.15	0.87	31.3	6.9
Cardiovascular	23.8	1.04	0.94	1.02	1.09	0.88	1.13	1.61	0.78	104.8	19.7
Mental	17.6	1.03	0.97	1.01	1.06	0.94	1.15	1.05	0.76	52.1	7.0
Neurological	15.7	0.99	0.97	1.00	1.13	1.04	1.02	0.97	0.78	44.3	5.5
Chronic respiratory	9.4	0.99	0.98	0.99	1.20	0.94	1.18	1.72	0.81	111.6	8.6
Diabetes	7.2	0.88	1.15	0.95	1.14	1.00	1.15	2.71	0.57	371.8	15.5
Unintentional injuries	6.3	0.96	0.95	1.08	1.01	1.06	1.13	2.03	0.68	196.7	8.6
Musculoskeletal	5.3	0.96	1.00	1.05	1.02	1.05	1.18	0.96	0.87	35.5	1.7
Genitourinary	3.3	1.01	1.04	0.93	1.06	0.94	1.01	1.76	0.77	127.9	3.3
Intentional injuries	3.0	0.94	0.89	1.11	1.10	1.05	1.18	2.46	0.79	210.3	5.0
All causes	132.4	1.00	0.99	1.00	1.09	0.96	1.12	1.50	0.79	88.7	100.0

(a) DALY rate for Australia per 1,000.

(b) Ratio of age-standardised DALYs per 1,000 population for area to DALYs per 1,000 population for Australia.

(c) Calculated for each cause as the greatest difference in DALY rates between areas as a proportion of lowest rate for that cause.

(d) Calculated for each cause as the greatest difference in DALY rates between areas as a proportion of greatest difference for all causes.

Table 5.6 lists the 10 leading specific causes of burden for Australia and summarises for each jurisdiction these causes in terms of rank order and percentage of total burden. Diseases of old age, such as ischaemic heart disease and dementia, contributed less to the total burden in jurisdictions with younger populations (for example the Northern Territory and the Australian Capital Territory).

Table 5.6: Differentials in burden (DALYs) by state/territory jurisdiction for the 10 leading specific causes, Australia, 2003

Specific cause ^(a)	Rank								Per cent of total							
	NSW	Vic	Qld	SA	WA	Tas	NT	ACT	NSW	Vic	Qld	SA	WA	Tas	NT	ACT
Ischaemic heart disease	1	1	1	1	1	1	3	2	10.4	9.6	10.2	10.8	8.8	10.7	6.5	8.1
Anxiety & depression	2	2	2	2	2	2	1	1	7.2	7.1	7.9	6.0	8.0	7.3	8.4	9.3
Type 2 diabetes	4	3	3	3	3	3	2	4	4.4	5.9	4.8	5.3	5.4	5.0	7.9	3.5
Stroke	3	4	4	4	5	4	11	3	5.0	4.3	4.4	4.5	3.9	4.4	2.0	3.9
Dementia	5	5	7	5	4	8	15	10	3.8	3.4	3.2	4.0	4.3	2.5	1.2	2.5
Lung cancer	7	6	5	7	6	6	10	7	3.4	3.4	3.3	3.2	3.5	3.8	2.3	2.8
COPD	6	7	6	6	7	5	7	8	3.4	3.1	3.3	3.7	2.8	4.0	3.3	2.6
Adult-onset hearing loss	10	10	8	8	10	9	35	12	2.2	2.5	2.9	2.6	2.4	2.4	0.7	2.3
Colorectal cancer	8	8	10	9	9	7	22	11	2.3	2.6	2.3	2.4	2.5	2.7	0.9	2.4
Asthma	11	9	9	11	8	10	8	5	2.2	2.5	2.5	2.3	2.7	2.4	2.3	3.3

(a) Sorted according to the leading specific causes for Australia.

5.4 Differentials by socioeconomic status

Populations in areas with lower socioeconomic status experienced proportionally more burden than populations in areas with higher socioeconomic status (Table 5.7). Females experienced slightly more burden than males in areas with the highest socioeconomic status. Conversely, males experienced more burden than females in areas with the lowest socioeconomic status. The highest proportion of burden that was fatal was in the moderately low and average socioeconomic areas, and the lowest (47.6%) was in the low socioeconomic area.

Table 5.7: Burden (DALYs) for socioeconomic quintiles by proportions of total, proportions by sex, and proportions due to mortality, Australia, 2003

Area	DALYs ('000)	Per cent of total	Per cent male	Per cent fatal burden
Low SES	562.5	21.4	52.8	47.6
Moderately low SES	564.2	21.4	52.7	49.3
Average SES	523.6	19.9	52.1	49.5
Moderately high SES	507.7	19.3	52.0	48.0
High SES	474.8	18.0	49.1	48.4
Australia	2,632.8	100.0	51.8	48.6

Total burden per head of population increased with decreasing socioeconomic status, with the most disadvantaged populations having 31.7% greater burden than the most advantaged populations. Again, this was due to higher rates of burden for most causes, but particularly for mental disorders and cardiovascular disease (Figure 5.4).

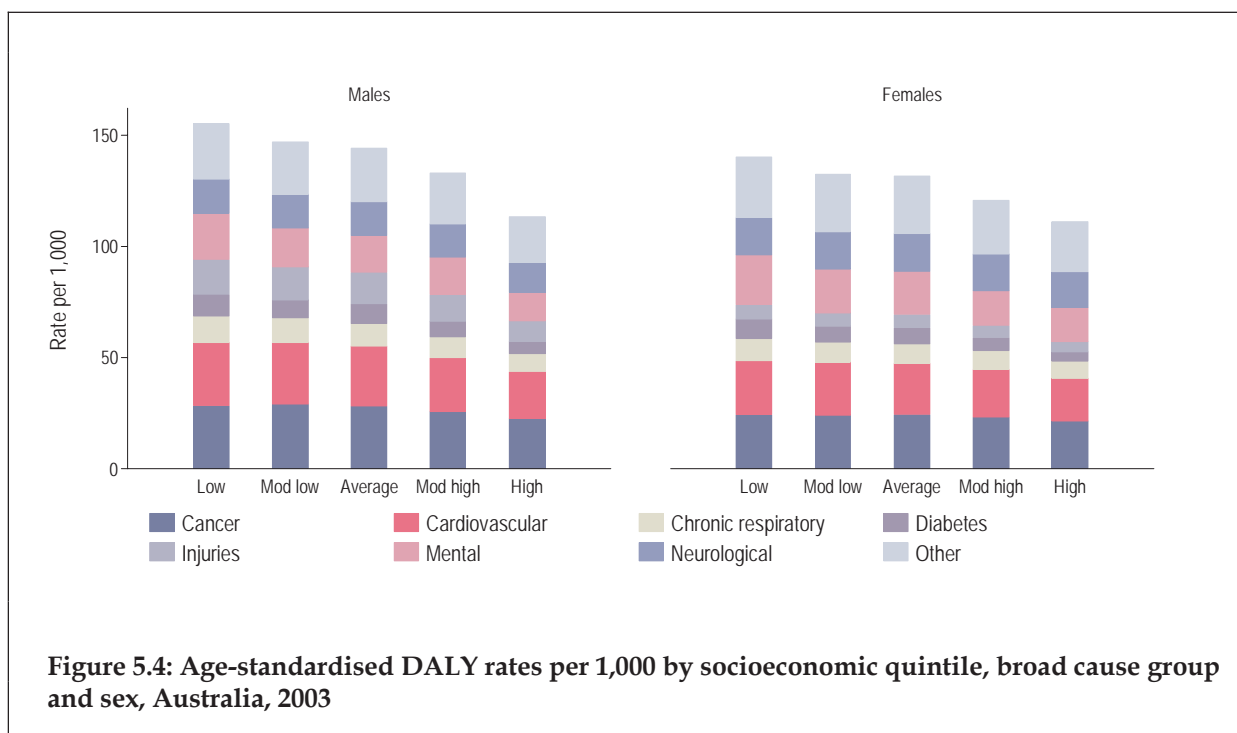


Table 5.8 provides a comparison between burden rates for areas by socioeconomic category and the national average for the 10 leading broad causes of burden in Australia for 2003. Of these causes, the greatest difference between areas with the lowest and highest rates occurred (in order of magnitude of difference) in diabetes, injuries, mental disorders and chronic respiratory diseases. The causes that contributed most in terms of the absolute difference observed between socioeconomic quintiles were mental disorders (20.9%), cardiovascular disease (17.6%) and diabetes (12.2%). Lifestyle-related (that is behavioural) risk factors are important underlying risks for these conditions; the much greater burden from these causes in lower socioeconomic areas is likely to be due to the greater prevalence of lifestyle risk factors in these areas compared with higher socioeconomic areas. Limited data availability on exposures by socioeconomic status, however, prevented further exploration of this association.

Table 5.8: Differentials in burden (DALY rates) by socioeconomic quintile for the 10 leading broad cause groups, Australia, 2003

Broad cause group	Rate	Standardised rate ratio ^(b)					% diff.	% of
	Aust. ^(a)	Low	Mod. low	Average	Mod. high	High	high/low ^(c)	total diff. ^(d)
Cancer	25.1	1.05	1.05	1.05	0.97	0.88	19.3	12.0
Cardiovascular	23.8	1.10	1.08	1.05	0.95	0.84	31.8	17.6
Mental	17.6	1.22	1.05	1.02	0.92	0.80	53.5	20.9
Neurological	15.7	1.02	1.02	1.03	1.00	0.93	10.2	4.2
Chronic respiratory	9.4	1.15	1.07	1.01	0.95	0.83	38.8	8.4
Diabetes	7.2	1.30	1.05	1.09	0.91	0.70	87.2	12.2
Unintentional injuries	6.3	1.14	1.12	1.12	0.93	0.72	57.8	7.3
Musculoskeletal	5.3	1.08	1.02	1.05	0.97	0.89	20.5	2.7
Genitourinary	3.3	1.07	1.02	1.04	0.97	0.92	16.0	1.4
Intentional injuries	3.0	1.28	1.11	1.00	0.91	0.73	75.1	4.6
All causes	132.4	1.12	1.05	1.04	0.96	0.85	31.7	100.0

(a) DALY rate for Australia per 1,000.

(b) Ratio of age-standardised DALYs per 1,000 population for area to DALYs per 1,000 population for Australia.

(c) Calculated for each cause as the greatest difference in DALY rates between areas as a proportion of lowest rate for that cause.

(d) Calculated for each cause as the greatest difference in DALY rates between areas as a proportion of greatest difference for all causes.

Table 5.9 lists the 10 leading specific causes of burden for Australia and summarises for each socioeconomic quintile these causes in terms of rank order and percentage of total burden. Ischaemic heart disease and anxiety & depression were the leading causes of burden across all socioeconomic quintiles.

Table 5.9: Differentials in burden (DALYs) by socioeconomic quintile for the 10 leading specific causes, Australia, 2003

Specific cause ^(a)	Rank					Per cent of total				
	Low	Mod. low	Average	Mod. high	High	Low	Mod. low	Average	Mod. high	High
Ischaemic heart disease	1	1	1	1	1	9.8	10.5	10.2	9.6	9.8
Anxiety & depression	2	2	2	2	2	8.1	7.3	6.8	7.5	6.6
Type 2 diabetes	3	3	3	3	5	5.9	5.0	5.3	4.8	4.2
Stroke	4	4	4	4	3	4.0	4.6	4.6	4.5	4.9
Dementia	7	6	5	5	4	2.9	3.5	3.7	3.7	4.2
Lung cancer	6	5	6	6	6	3.5	3.6	3.5	3.2	3.0
COPD	5	7	7	7	7	3.7	3.5	3.3	3.1	2.8
Adult-onset hearing loss	9	8	9	8	11	2.4	2.5	2.5	2.6	2.4
Colorectal cancer	10	9	8	9	9	2.1	2.4	2.5	2.5	2.6
Asthma	8	10	11	11	10	2.5	2.4	2.2	2.5	2.5

(a) Sorted according to the leading specific causes for Australia.

5.5 Differentials by remoteness

The majority (64.5%) of the burden was experienced by people in the major cities as they account for 67% of the population. Regional areas accounted for 33.1% of the burden and remote areas 2.5% (Table 5.10). Males experienced more of this burden than females in all areas, but particularly in remote areas. Remote areas experienced proportionately slightly less fatal burden than other areas.

Table 5.10: Burden (DALYs) for remoteness categories by proportions of total, proportions by sex, and proportions due to mortality, Australia, 2003

Area	DALYs ('000)	Per cent of total	Per cent male	Per cent fatal burden
Major cities	1,698.0	64.5	51.0	48.2
Regional	870.1	33.1	53.1	49.6
Remote	64.6	2.5	57.5	46.2
Australia	2,632.8	100.0	51.8	48.6

Total burden per head of population increased with remoteness, with remote populations having 26.5% greater burden than populations in major cities. Again, this is due to higher rates of burden for most causes, but particularly for injuries (Figure 5.5).

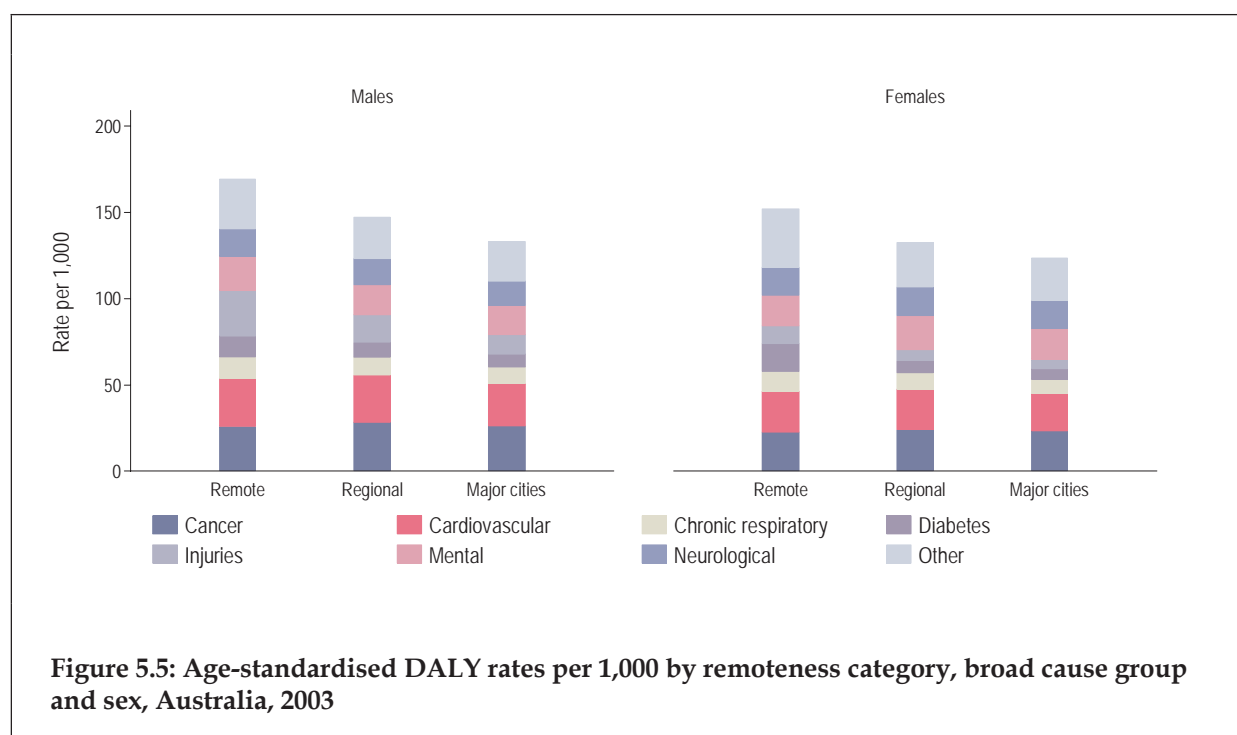


Figure 5.5: Age-standardised DALY rates per 1,000 by remoteness category, broad cause group and sex, Australia, 2003

Table 5.11: Differentials in burden (DALY rates) by remoteness category for the 10 leading broad cause groups, Australia, 2003

Broad cause group	Rate	Standardised rate ratio ^(b)			% diff.	% of
	Aust. ^(a)	Major cities	Regional	Remote	high/low ^(c)	total diff. ^(d)
Cancer	25.1	0.98	1.04	0.98	7.0	4.6
Cardiovascular	23.8	0.96	1.07	1.10	14.6	9.1
Mental	17.6	0.98	1.05	1.06	8.5	4.0
Neurological	15.7	0.99	1.03	1.03	4.2	1.8
Chronic respiratory	9.4	0.97	1.04	1.30	33.6	8.3
Diabetes	7.2	0.94	1.08	1.93	105.6	19.5
Unintentional injuries	6.3	0.87	1.24	1.92	121.3	18.1
Musculoskeletal	5.3	0.95	1.10	0.99	16.0	2.2
Genitourinary	3.3	1.00	0.99	1.11	12.3	1.1
Intentional injuries	3.0	0.90	1.13	2.26	151.5	11.0
All causes	132.4	0.97	1.06	1.22	26.5	100.0

(a) DALY rate for Australia per 1,000.

(b) Ratio of age-standardised DALYs per 1,000 population for area to DALYs per 1,000 population for Australia.

(c) Calculated for each cause as the greatest difference in DALY rates between areas as a proportion of lowest rate for that cause.

(d) Calculated for each cause as the greatest difference in DALY rates between areas as a proportion of greatest difference for all causes.

Table 5.11 provides a comparison between burden rates for areas by remoteness category and the national average for the 10 leading broad causes of burden in Australia for 2003. Of these causes, the greatest difference between areas with the lowest and highest rates occurred (in order of magnitude of difference) in injuries, diabetes, chronic respiratory diseases, musculoskeletal disorders and cardiovascular disease. The cause that contributed by far the greatest proportion in terms of the absolute difference observed between remoteness categories was injuries (29.1% for intentional and unintentional combined), followed by diabetes (19.5%) and cardiovascular disease (9.1%).

Table 5.12 lists the 10 leading specific causes of burden for Australia and summarises for each remoteness category these causes in terms of rank order and percentage of total burden. Type 2 diabetes was the leading cause of burden in remote areas whereas dementia was ranked twelfth, reflecting the younger age structure and higher proportion of Indigenous people in these areas compared with the rest of Australia.

Table 5.12: Differentials in burden (DALYs) by remoteness category for the 10 leading specific causes, Australia, 2003

Specific cause ^(a)	Rank			Per cent of total		
	Major cities	Regional	Remote	Major cities	Regional	Remote
Ischaemic heart disease	1	1	2	9.8	10.6	7.3
Anxiety & depression	2	2	3	7.4	7.1	6.4
Type 2 diabetes	3	3	1	4.9	5.1	7.7
Stroke	4	4	8	4.6	4.4	2.8
Dementia	5	7	12	3.8	3.3	2.0
Lung cancer	6	6	10	3.4	3.5	2.6
COPD	7	5	4	3.1	3.6	3.8
Adult-onset hearing loss	11	8	11	2.3	2.7	2.1
Colorectal cancer	9	9	15	2.4	2.5	1.4
Asthma	8	10	9	2.5	2.3	2.7

(a) Sorted according to the leading specific causes for Australia.

6 Past, present and future burden of disease and injury in Australia

6.1 Overview

This chapter presents trends in population health dynamics over a thirty-year period. The analyses involved consideration of health statistics over the last 25 years or more, although the discussion about the past is linked to health trends over the last decade. Also presented are the projected levels of the burden of disease and injury if these trends were to continue 20 years into the future. Since mortality is the starting point for many of these analyses, observed and projected trends in mortality by broad cause group are summarised in Table 6.1. The methods underlying all analyses presented in this chapter are described in detail in Chapter 2.

Table 6.1: Changes in mortality by broad cause group and sex, Australia, 1993 to 2023

Broad cause group	Rate per 100,000 for 2003		Standardised rate ratio ^(a)							
			Males				Females			
	Males	Females	1993	2003	2013	2023	1993	2003	2013	2023
Infectious	14.8	9.5	0.93	1.00	0.97	0.92	0.95	1.00	0.92	0.83
Acute respiratory ^(b)	16.5	20.9	0.43	1.00	1.00	1.00	0.40	1.00	1.00	1.00
Maternal	—	0.1	—	—	—	—	1.87	1.00	1.26	1.19
Neonatal	3.6	2.7	1.74	1.00	0.63	0.41	1.01	1.00	0.66	0.46
Nutritional	0.2	0.6	2.83	1.00	1.24	1.09	1.72	1.00	0.74	0.65
Cancer	211.1	163.7	1.20	1.00	0.89	0.75	1.12	1.00	0.92	0.82
Other neoplasms	4.3	4.1	1.06	1.00	0.88	0.74	0.92	1.00	0.96	0.91
Diabetes	19.5	16.6	1.01	1.00	0.96	0.90	1.13	1.00	0.88	0.76
Endocrine	5.6	7.0	1.73	1.00	1.00	0.89	0.95	1.00	1.07	1.06
Mental	10.4	3.4	1.18	1.00	0.90	0.75	1.10	1.00	0.91	0.79
Neurological	27.8	41.7	1.10	1.00	0.99	0.91	0.93	1.00	1.04	1.05
Cardiovascular	237.9	252.6	1.61	1.00	0.71	0.47	1.52	1.00	0.76	0.52
Chronic respiratory	48.1	37.7	1.38	1.00	0.82	0.69	1.00	1.00	1.04	1.07
Digestive	14.7	19.4	1.18	1.00	0.74	0.55	1.13	1.00	0.80	0.63
Genitourinary	16.7	20.1	1.01	1.00	0.95	0.87	0.86	1.00	0.98	0.93
Skin	1.2	2.0	1.06	1.00	0.96	0.83	0.88	1.00	1.03	1.04
Musculoskeletal	2.6	5.1	1.29	1.00	0.94	0.79	1.10	1.00	0.95	0.90
Congenital	4.2	3.4	1.17	1.00	0.75	0.60	1.30	1.00	0.75	0.58
Oral	0.0	0.1	1.61	1.00	1.29	1.11	0.39	1.00	0.73	0.74
Ill defined	0.5	0.6	3.13	1.00	0.54	0.25	2.01	1.00	0.69	0.52
Injuries	52.4	27.7	1.13	1.00	0.87	0.73	1.00	1.00	0.87	0.74
All causes	692.1	639.0	1.32	1.00	0.83	0.67	1.22	1.00	0.87	0.73

(a) Ratio of age-standardised mortality rates for year to mortality rates for 2003.

(b) Age-specific rates for pneumonia post-2003 held at 2003 rates due to coding discontinuities between ICD-9 and ICD-10 for this cause.

6.2 Health-adjusted life expectancy

This section begins, as did Chapter 5, by presenting life expectancy and health-adjusted life expectancy, but this time with a temporal dimension rather than with a focus on differentials between subpopulations. Over the last decade, total life expectancy in Australia improved from 78.0 years in 1993 to 80.7 years in 2003. This was an annual growth of 0.35% (or 0.28 of a year per year). If past mortality trends continue into the future as projected (that is, at an exponentially declining rate), life expectancy will increase to 82.6 years in 2013 and 84.6 years in 2023, an increase of 3.9 years from 2003. This represents an annual growth of 0.24% (or 0.20 of a year per year) over the 20-year period (Table 6.2).

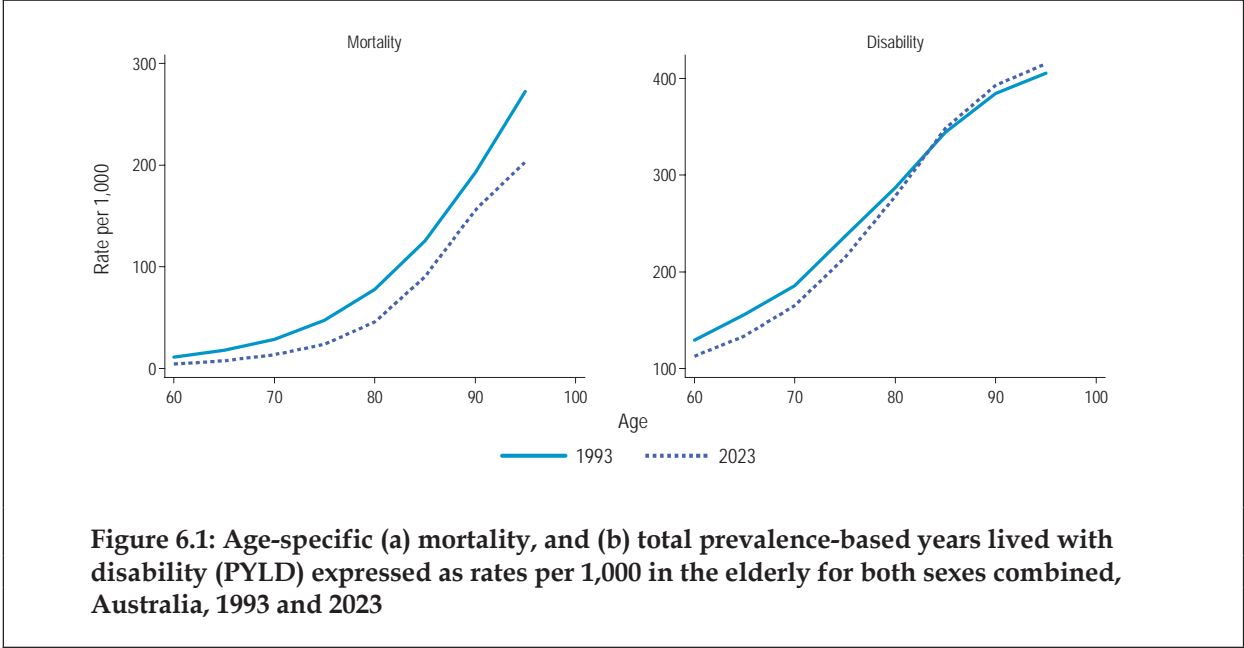
Table 6.2: Life expectancy and health-adjusted life expectancy by sex, Australia, 1993 to 2023

	1993			2003			2013			2023		
	Males	Females	Both	Males	Females	Both	Males	Females	Both	Males	Females	Both
Population ^(a) (millions)	8.8	8.9	17.7	9.9	10.0	19.9	11.0	11.2	22.2	12.1	12.3	24.5
Proportion of population at selected ages (%)												
0–59 years	85.8	82.8	84.3	84.1	81.6	82.8	79.8	77.6	78.7	75.1	72.7	73.9
60–79 years	12.5	14.1	13.3	13.5	14.2	13.9	16.9	17.3	17.1	20.3	21.1	20.7
80+ years	1.6	3.2	2.4	2.4	4.2	3.3	3.4	5.1	4.2	4.6	6.1	5.4
Life expectancy (years)												
At birth	75.0	81.0	78.0	78.3	83.2	80.7	80.6	84.8	82.6	83.3	86.5	84.6
At age 60	19.5	23.8	21.7	22.1	25.6	23.9	23.7	26.8	25.2	25.9	28.1	26.8
At age 80	7.2	9.1	8.4	8.4	9.9	9.3	9.1	10.5	9.8	10.6	11.3	10.7
Health-adjusted life expectancy (years)												
At birth	68.0	73.5	70.7	70.6	75.2	72.9	72.5	76.6	74.5	74.7	78.0	76.2
At age 60	15.2	19.2	17.3	17.1	20.5	18.9	18.4	21.5	19.9	20.1	22.5	21.2
At age 80	4.7	6.3	5.6	5.4	6.8	6.2	5.9	7.2	6.6	6.8	7.7	7.1
Healthy life expectancy lost due to disability (years)												
At birth	7.0	7.5	7.3	7.7	7.9	7.8	8.0	8.2	8.1	8.6	8.5	8.5
At age 60	4.4	4.6	4.5	5.0	5.1	5.0	5.3	5.3	5.2	5.8	5.6	5.6
At age 80	2.5	2.8	2.7	3.0	3.2	3.1	3.2	3.3	3.3	3.7	3.6	3.5
Healthy life expectancy lost due to disability as proportion of total life expectancy (%)												
At birth	9.4	9.2	9.3	9.8	9.6	9.7	9.9	9.6	9.8	10.3	9.9	10.0
At age 60	22.3	19.3	20.6	22.6	19.8	21.1	22.2	19.7	20.8	22.6	19.9	21.0
At age 80	35.2	31.1	32.5	35.8	31.8	33.2	35.3	31.8	33.1	35.4	31.9	33.3

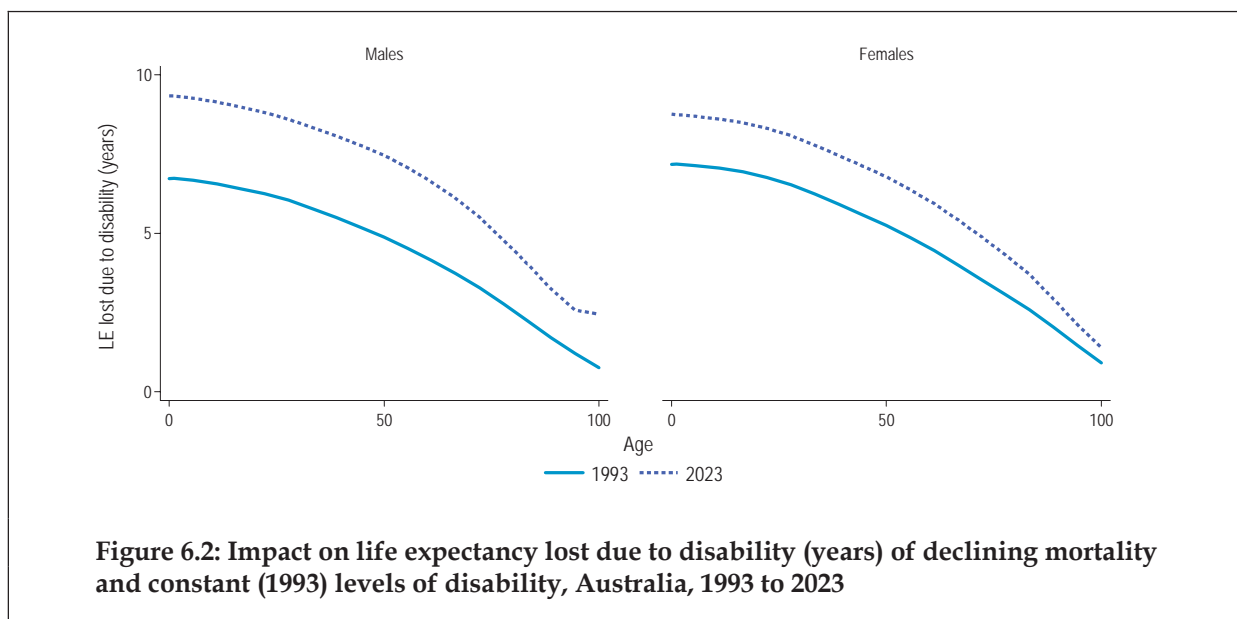
(a) Estimated resident population figures as at 30 June 1993 and 2003 (ABS 2006, Cat. no. 3201.0, Table 9) and ABS population projections series 8 (ABS 2003a, Cat. no. 3222.0).

Health-adjusted life expectancy, on the other hand, increased from 70.7 years to 72.9 years in the decade to 2003, an annual growth of 0.31% (or 0.22 of a year per year). If, in addition to past mortality trends, trends in non-fatal health conditions that give rise to disability continue into the future as projected, health-adjusted life expectancy will increase to 74.5 years in 2013 and 76.2 years in 2023. This represents an annual growth of 0.22% (or 0.16 of a year per year) over the 20-year period.

Complex dynamics in population health will drive the slower gains in health-adjusted life expectancy relative to total life expectancy. The most important of these is the decline in mortality rates between 1993 and 2023 across the life span, but particularly in the elderly (Figure 6.1a). One of the consequences of declining mortality is that, in combination with ongoing declines in fertility, Australia’s population will continue to age. Of particular relevance is the number of people aged 80 years and older. Over the last decade, the proportion of the total population in this age group increased from 2.4% in 1993 to 3.3% in 2003. Based on recent Australian Bureau of Statistics (ABS) projections (ABS 2003a), this is expected to increase to 4.2% in 2013 and 5.4% in 2023 (Table 6.2).

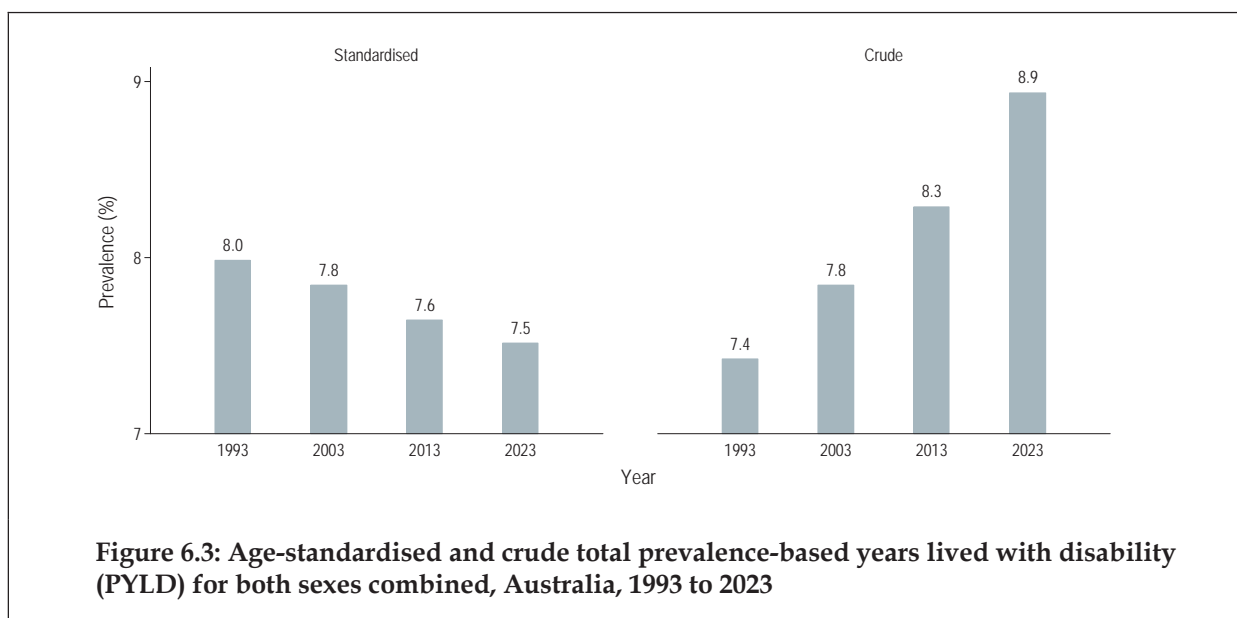


The impact on life expectancy of declining mortality rates is straightforward – it will increase. The impact on health-adjusted life expectancy and its corollary, life expectancy lost due to disability, however, is perhaps less intuitive at first. The key point is that, in most populations, even if the prevalence of disability at each age were to remain at constant levels, a decline in mortality would mean an increase in life expectancy lost due to disability in the future (Figure 6.2). This is because reductions in mortality result in more people surviving through to ages when the probability of being disabled is highest. Ultimately, though, this relationship depends on changes in the rate at which mortality increases with age relative to changes in the rate at which disability increases with age.



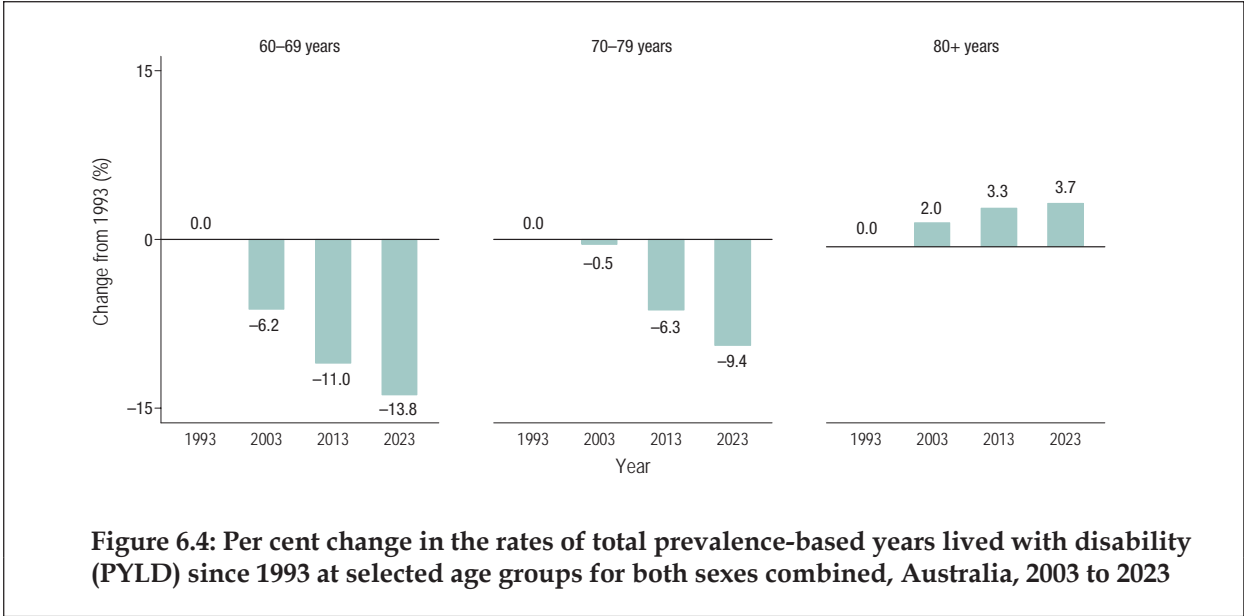
In addition to the increase in the proportion of total life expectancy lost due to disability through reductions in mortality, is the impact of temporal trends in diseases and injuries that give rise to the prevalence of disability. By estimating separately the epidemiology of these causes in a fully temporal model, changes in total prevalence of disability by age, sex and cause can be quantified for the first time over the past as well as into the future.

While the prevalence of overall disability appears to decrease when the effect of population ageing is removed (by standardising for age), it will consistently increase over the next two decades in crude terms (Figure 6.3). In other words, the proportion of overall time lived with disability will increase from 7.8% in 2003 to 8.9% in 2023, an increase of 14.1%.



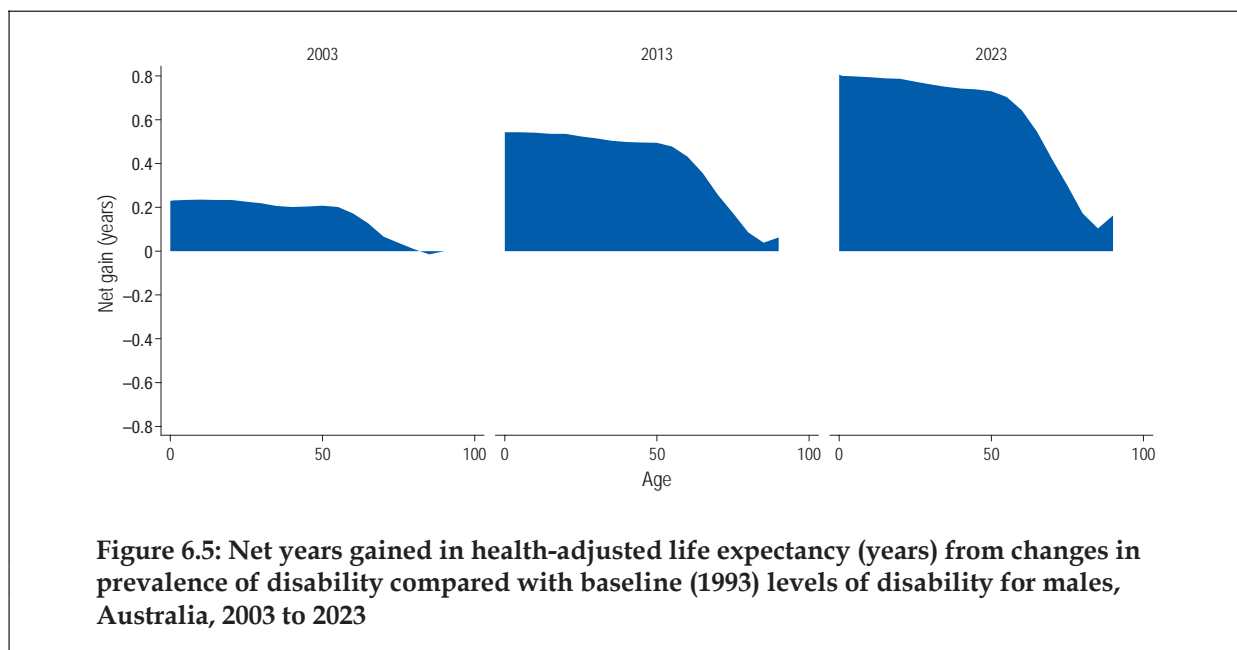
This is for two reasons. First, the number of people aged 80 years and over is set to expand rapidly due to declining mortality (Figure 6.1a). Second, while the prevalence of disability will drop at most ages, it will actually increase in this age group (Figure 6.1b). In the decade

to 2003, disability in people aged 80 years and over increased by 2.0%; if past trends continue, by 2023 disability will have increased a further 1.7% (Figure 6.4). This lends support to the hypothesis which predicts that as population health improves, disability is increasingly concentrated towards the end of the life span.

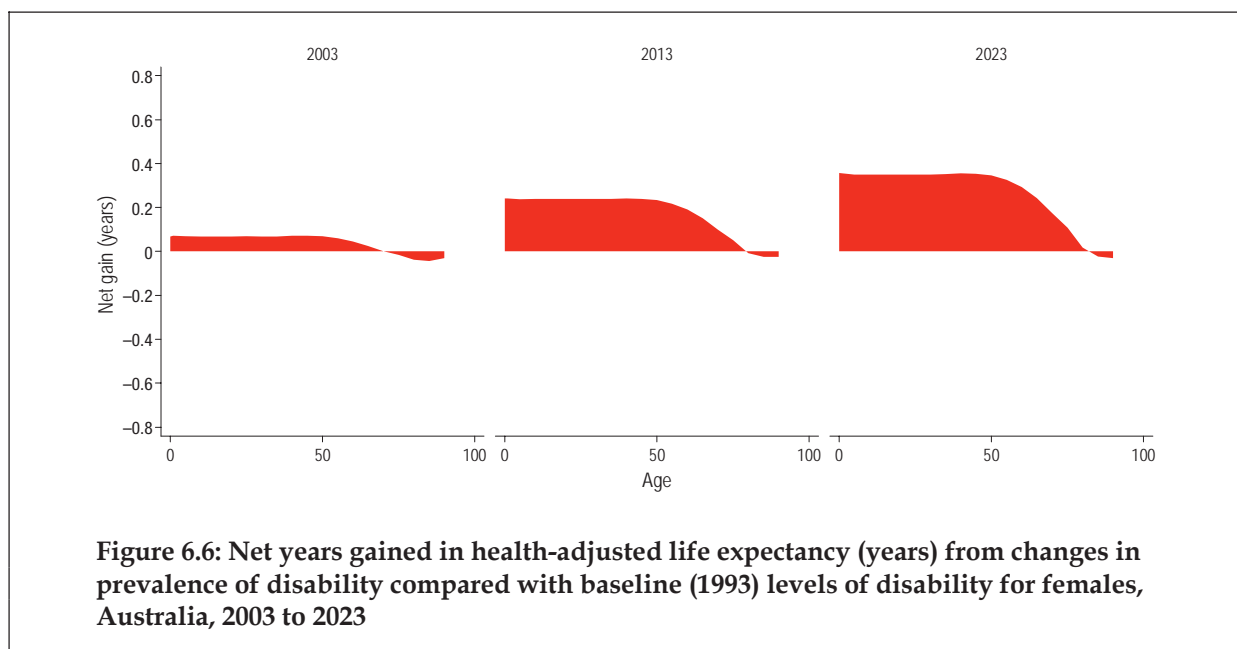


The effect on health-adjusted life expectancy of the increasing concentration of disability towards the end of life, which until now has been largely unexplored using empirical data, can be illustrated by gains in expectation of life in a model in which disability is included as a dynamic force over time, compared with a counterfactual scenario in which the probability of disability is held constant (Figure 6.5 and Figure 6.6). Such a comparison provides insights into the question ‘What impact will concentration of disability towards the end of the lifespan have on health-adjusted life expectancy?’

Health-adjusted life expectancy at birth is the most commonly cited measure, and summarises mortality and disability risks across the life span. In males, this will increase at a rate faster than would have been observed through reductions in mortality alone, with the net effect of morbidity being increasingly concentrated towards the end of life. From 1993 to 2003 the gain was about 0.2 years. Over the longer term, however, the gain will be larger, at around 0.8 years of healthy life for males born in 2023. At adult ages, the gains are less and, in the elderly, where gains in health expectancy due to declines in mortality are more easily offset by increases in disability, there were losses in healthy life due to this dynamic in the decade to 2003, but these disappear in the subsequent decade (Figure 6.5).



In females, the impact of morbidity being increasingly concentrated towards the end of life is not readily apparent in the decade to 2003. Over the next two decades this dynamic will start to have an impact, although the gains in health expectancy at birth will be smaller than for males (around 0.3 years in 2023) and the losses in the elderly will be greater and will be experienced earlier in life.



The correct answer to the question ‘What impact will the concentration of disability in the latter part of the lifespan have on health-adjusted life expectancy?’, therefore, is: ‘It depends’. This is because health expectancy at any particular age is a summary measure based on the

combination of mortality and disability risks at that age and all subsequent ages. While a detailed decomposition of the drivers of this complex dynamic is beyond the scope of this report, growth in prevalent disability in the elderly is likely to come from increases in diabetes and neurological conditions. Disability from diabetes, in particular, grew 10.4% in the decade to 2003, and will grow a further 29.3% over the next two decades if current trends in obesity continue (Figure 6.7). Neurological conditions grew 2.5% in the decade to 2003, and are likely to grow a further 6.6% in the 20 years to 2023. Most other causes of prevalent disability are likely to decline.

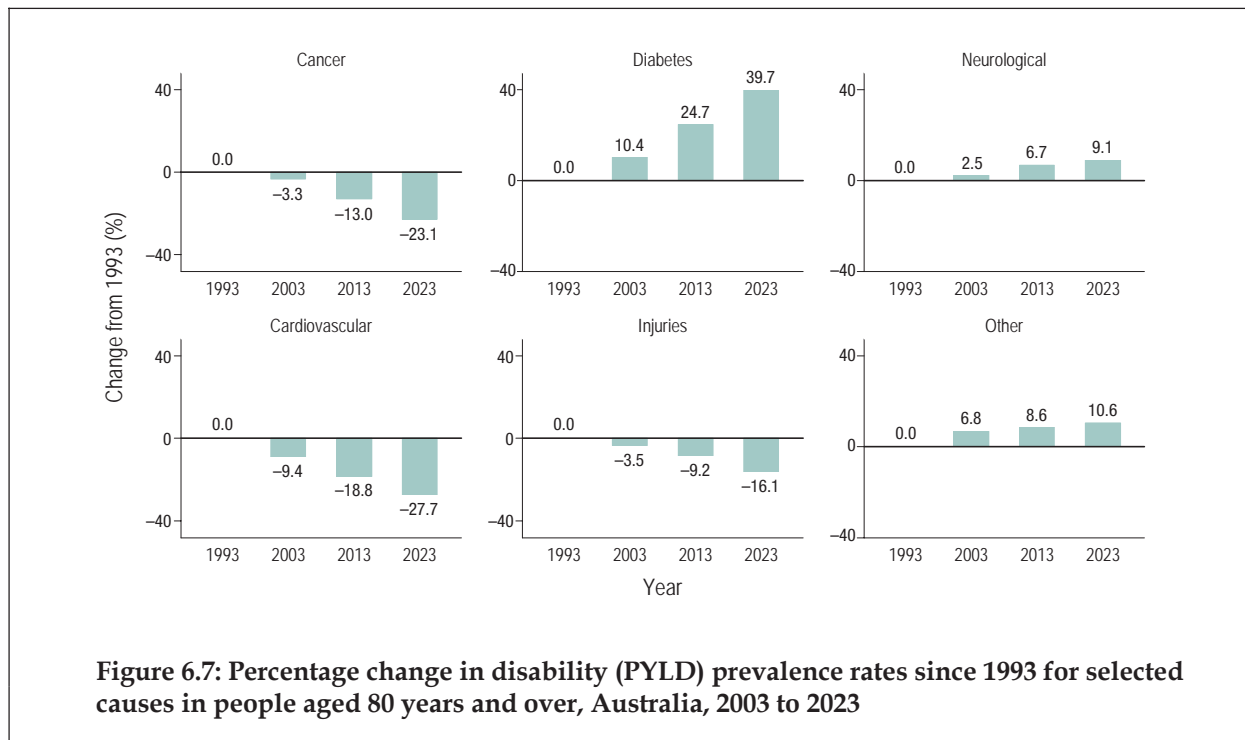
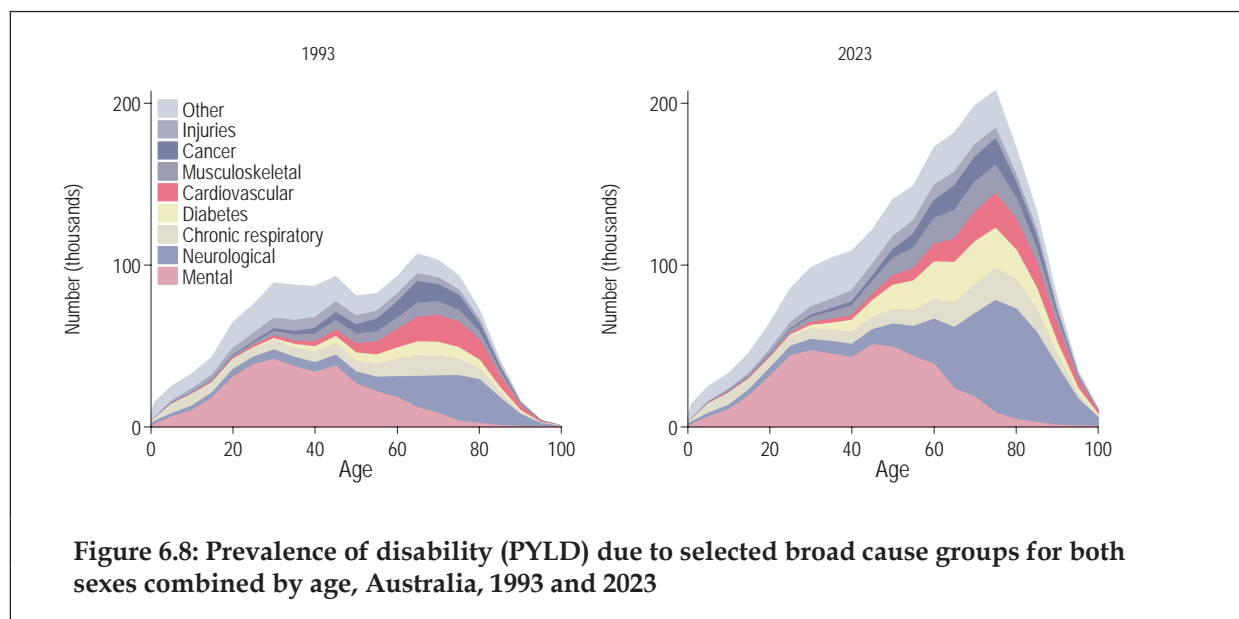


Figure 6.8 shows the number of healthy years lost due to prevalent disability (PYLD) by cause and age for 1993 and 2023. This figure demonstrates the absolute growth in PYLD that is expected to occur over this period due to increases in population size. It also shows the shift in the distribution of PYLD towards older ages that will occur as a result of population ageing. Trends in epidemiology will interact with these demographic factors to influence the composition of causes of prevalent disability at each age. Neurological conditions will grow substantially over the period 1993 to 2023 and will remain the largest contributor to disability prevalence at older ages. Mental disorders, on the other hand, will grow only slightly from 1993 levels but will remain the largest contributor to disability prevalence until age 60. Disability from cardiovascular disease is expected to decline from middle age onwards over this period but this decline will be more than offset by increases from diabetes.



Changes in the age-specific trends described above reflect changes in the prevalence of disability experienced at all ages (Figure 6.9). Mental disorders decreased from 26% to 25% of total prevalence of disability in the decade to 2003. The effects of population ageing will mean that mental disorders, which are largely experienced in early to middle adulthood, will further decline to 22% of total prevalent disability in 2023, although they will remain the leading cause of overall prevalent disability. Neurological & sense disorders, on the other hand, will increase as a consequence of population ageing because they are experienced later in life. In the decade to 2003 this group increased from 15% to 17% of total prevalent disability in 2003, and over the next two decades, through population ageing alone, will increase to 21% in 2023.

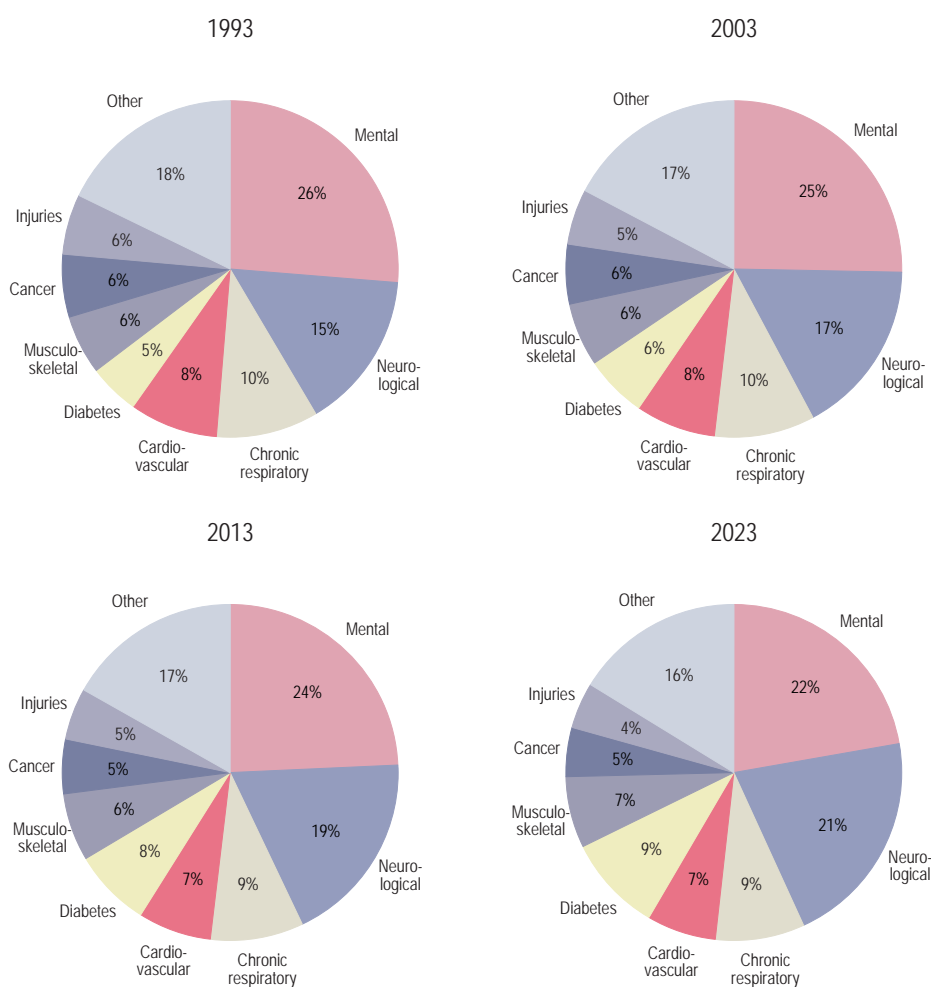


Figure 6.9: Proportion of total prevalence of disability (PYLD) due to selected broad cause groups, Australia, 1993 to 2023

Diabetes was the other strong growth area at all ages, increasing from 5% of total prevalent disability in 1993 to 6% in 2003. If current trends in obesity continue, this figure is set to increase by a further 50% to 9% of total prevalent disability in 2023.

6.3 Burden

The remainder of this chapter presents past, present and future burden using the standard burden measure – DALYs. It is worth reiterating at this point that, unlike prevalent years lived with disability (PYLD), DALYs are incidence-based and include, in addition to non-fatal health outcomes, time lost due to premature mortality. Observed and projected trends in burden (DALYs) by broad cause group are summarised in Table 6.3. The methods underlying these figures are described in detail in Chapter 2. More detailed data on past,

present and future burden by age, sex and cause is available on the web at <www.aihw.gov.au/bod>.

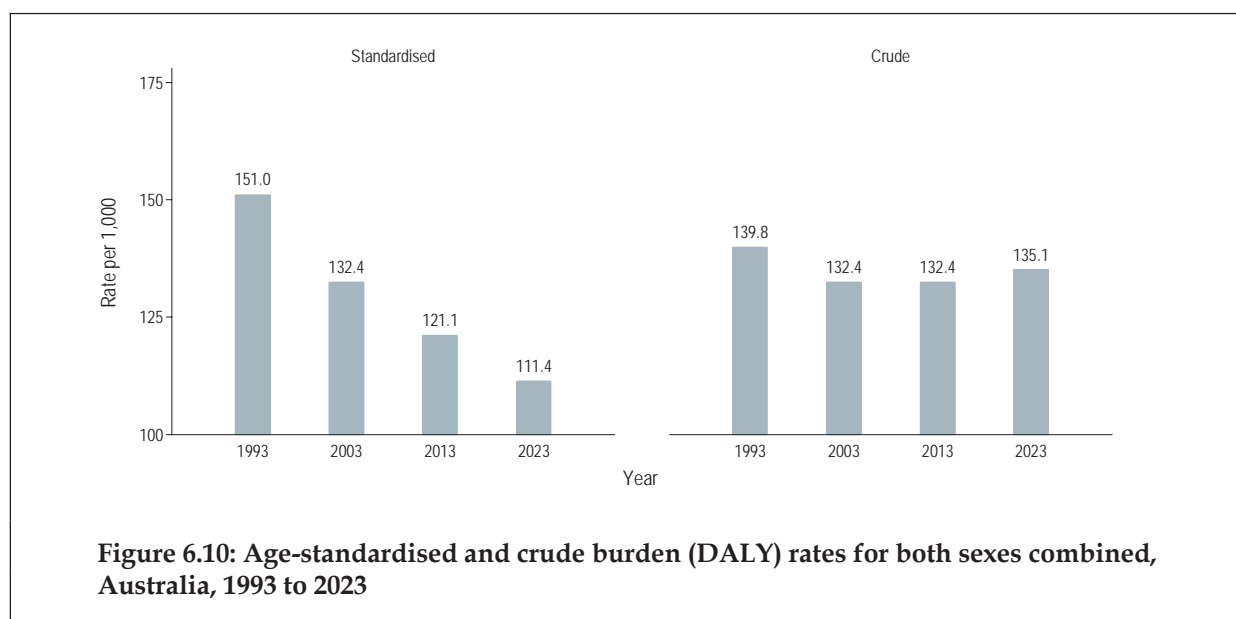
Table 6.3: Changes in burden rates (DALYs) by broad cause group and sex, Australia, 1993 to 2023

Broad cause group	Rate per 1,000 for 2003		Standardised rate ratio ^(a)							
			Males				Females			
	Males	Females	1993	2003	2013	2023	1993	2003	2013	2023
Infectious	2.8	1.7	0.93	1.00	1.02	0.99	0.99	1.00	0.93	0.85
Acute respiratory ^(b)	1.7	1.8	0.67	1.00	1.00	1.00	0.61	1.00	1.00	1.00
Maternal	—	0.2	—	—	—	—	1.09	1.00	1.03	1.02
Neonatal	1.9	1.6	1.32	1.00	0.80	0.68	1.00	1.00	0.82	0.71
Nutritional	0.1	0.5	1.12	1.00	1.03	1.02	1.03	1.00	0.99	0.98
Cancer	26.8	23.5	1.20	1.00	0.85	0.70	1.16	1.00	0.88	0.74
Other neoplasms	0.5	0.6	1.03	1.00	0.83	0.68	0.94	1.00	0.89	0.81
Diabetes	7.8	6.6	0.87	1.00	1.15	1.32	0.89	1.00	1.18	1.40
Endocrine	1.5	1.4	1.88	1.00	1.08	1.03	0.89	1.00	1.16	1.31
Mental	16.8	18.5	1.03	1.00	1.01	0.99	0.99	1.00	1.01	1.01
Neurological	14.9	16.6	0.96	1.00	1.02	1.03	0.96	1.00	1.03	1.05
Cardiovascular	25.6	22.1	1.56	1.00	0.69	0.48	1.51	1.00	0.74	0.53
Chronic respiratory	10.0	8.8	1.22	1.00	0.83	0.73	1.04	1.00	0.96	0.93
Digestive	2.9	2.9	1.01	1.00	0.81	0.71	1.03	1.00	0.85	0.75
Genitourinary	2.9	3.7	0.97	1.00	0.97	0.96	0.97	1.00	0.98	0.95
Skin	1.0	1.0	1.00	1.00	1.00	0.99	1.00	1.00	1.00	0.99
Musculoskeletal	4.5	6.1	0.98	1.00	1.03	1.05	0.97	1.00	1.02	1.02
Congenital	1.9	1.4	1.11	1.00	0.84	0.74	1.19	1.00	0.84	0.72
Oral	1.2	1.3	0.99	1.00	1.02	1.03	0.98	1.00	1.01	1.02
Ill defined	0.5	0.7	1.70	1.00	0.83	0.73	1.31	1.00	0.93	0.89
Injuries	13.1	5.5	1.16	1.00	0.91	0.79	1.08	1.00	0.89	0.76
All causes	138.2	126.7	1.18	1.00	0.90	0.81	1.11	1.00	0.93	0.87

(a) Ratio of age-standardised DALY rates for year to DALY rates for 2003.

(b) Age-specific rates for pneumonia post-2003 held at 2003 rates due to coding discontinuities between ICD-9 and ICD-10 for this cause.

As observed with PYLD, total burden will most likely decrease after the effect of population ageing is removed (that is, age-standardisation) over the next two decades, yet in crude terms it will most likely increase (Figure 6.10). Again, this is due to a larger proportion of the population alive at older ages.



Chapter 3 described the decline of cardiovascular disease relative to cancer as a proportion of overall burden, and stated that for the first time, cancer accounted for the largest share of overall burden experienced by the Australian population in 2003. This is primarily because Australia has been relatively successful at curbing the impact of the cardiovascular disease epidemic, but not nearly as successful to date with cancer. If these trends continue, the burden of cardiovascular disease will further decline to about 13% of the total burden in 2023. The age-standardised rates of cancer mortality and disability are expected to fall somewhat in the future but cancer as a whole will retain its share of around 19% of total burden two decades from now and will remain the largest contributor to total burden in 2023 (Figure 6.11).

Despite the steady decline in cardiovascular disease burden over the next two decades, there is likely to be a strong increase in burden due to diabetes, primarily as a consequence of the obesity epidemic. If current trends in obesity continue unabated, diabetes will account for around 9% of total burden in 2023, up from around 5% in 2003 (Figure 6.11).

A major consequence of population ageing will be the steady growth in burden from neurological & sense disorders, up from 12% in 2003 to around 16% in 2023. The main contributors here will be dementia and adult-onset hearing loss, both causes for which current treatments are largely ineffectual. The economic consequences of the former in terms of the provision of appropriate care services are likely to be significant and will be evident in the home and community sectors before they are felt in the residential aged care sector.

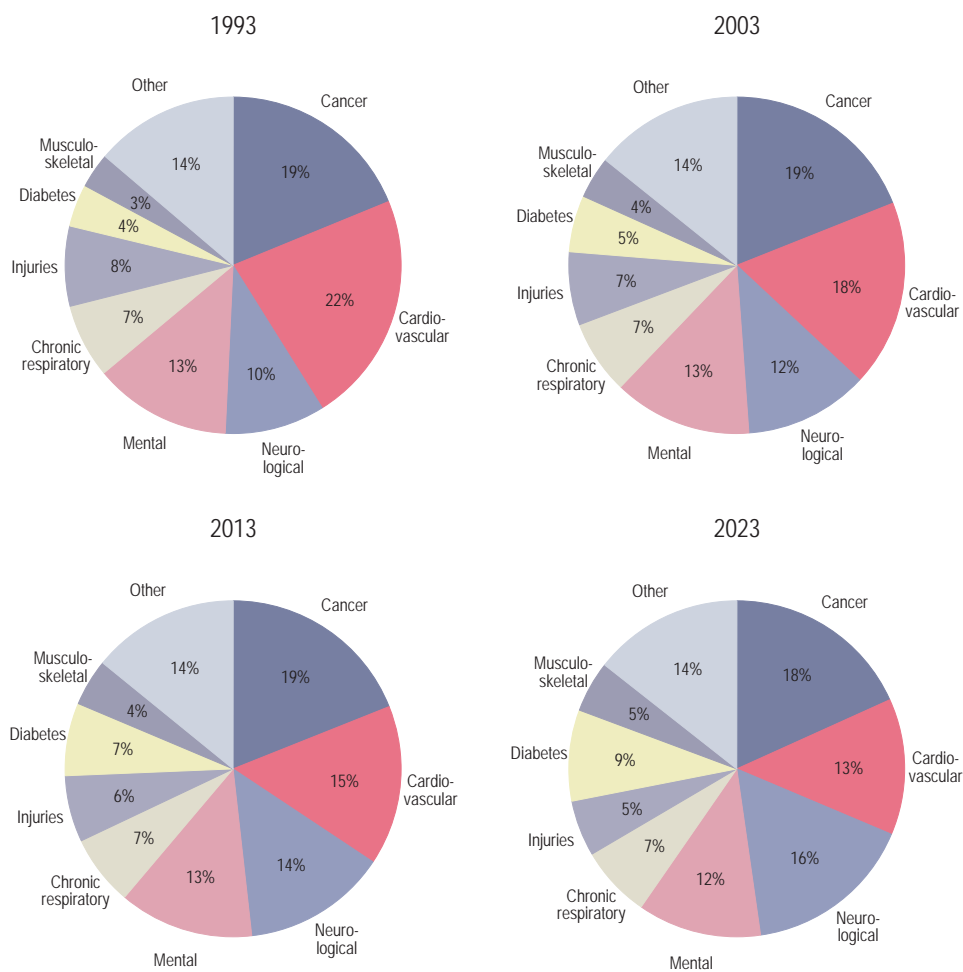
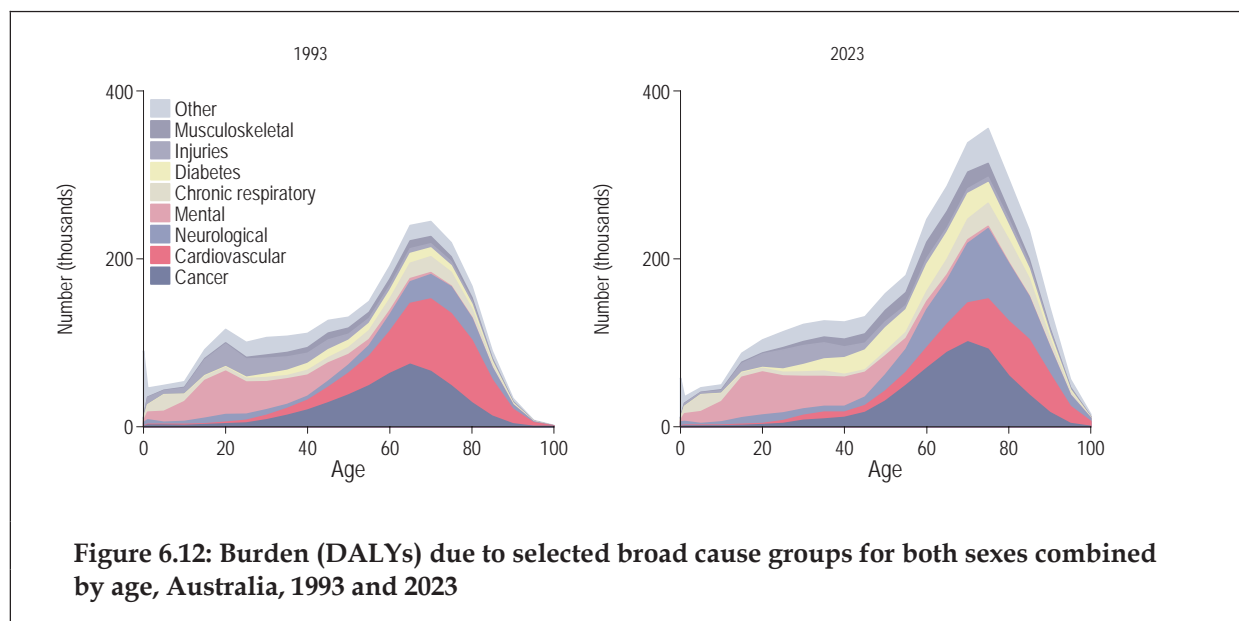


Figure 6.11: Proportion of total burden (DALYs) due to selected broad cause groups, Australia, 1993 to 2023

The proportion of burden due to major causes experienced at different ages throughout the life span is unlikely to change dramatically over the next two decades (Figure 6.12). The decline of cardiovascular disease as a proportion of total burden will be experienced at all ages, although, in absolute terms, most notably in the elderly. This will be partially offset by the increase in the proportion due to diabetes at all ages. The proportion of total burden due to cancer at different ages is unlikely to change.



In terms of specific causes of disease burden, ischaemic heart disease is the leading cause in males across three of the four time periods. Its share of burden declined from 14.7% in 1993 to 11.1% in 2003 (Table 6.4). If this trend continues, ischaemic heart disease will decline a further 36% to 7.1% of the total burden in 2023. Type 2 diabetes, on the other hand, rose from sixth place to second in the decade to 2003, and is likely to increase a further 65% to first place or 8.6% of the total burden in 2023. Anxiety & depression will retain its third place, at around 4.5% of the total burden in 2023, but lung cancer will drop to sixth place, largely because of the dramatic decline in smoking prevalence in males over the last two decades. In its place, dementia will occupy fourth position in 2023, up from 11th place in 2003.

Table 6.4 Leading causes of burden (DALYs) in males, Australia, 1993 to 2023

Specific cause	Rank ^(a)				Per cent of total			
	1993	2003	2013	2023	1993	2003	2013	2023
Ischaemic heart disease	1	1	1	2	14.7	11.1	8.9	7.1
Type 2 diabetes	6	2	2	1	3.6	5.2	6.8	8.6
Anxiety & depression	2	3	3	3	4.5	4.8	4.7	4.5
Lung cancer	3	4	4	6	4.4	4.0	3.8	3.4
Stroke	5	5	6	7	4.2	3.9	3.5	3.2
COPD	4	6	9	11	4.4	3.6	2.9	2.2
Adult-onset hearing loss	11	7	5	5	2.5	3.1	3.7	4.2
Suicide & self-inflicted injuries	8	8	10	10	2.9	2.8	2.8	2.4
Prostate cancer	10	9	8	8	2.5	2.7	3.0	3.1
Colorectal cancer	9	10	11	9	2.6	2.5	2.6	2.4
Dementia	14	11	7	4	1.8	2.5	3.3	4.4
Road traffic accidents	7	12	14	18	3.0	2.3	1.8	1.3
Asthma	12	13	12	12	2.2	2.1	2.0	1.9
Alcohol dependence & harmful use	13	14	13	14	2.0	2.0	1.9	1.6
Personality disorders	16	15	17	19	1.1	1.2	1.2	1.2
Schizophrenia	15	16	20	23	1.1	1.1	1.1	1.0
Osteoarthritis	24	17	15	15	0.8	1.1	1.3	1.6
Back pain	23	18	18	17	0.9	1.1	1.2	1.3
Melanoma	20	19	21	20	0.9	1.0	1.1	1.1
Parkinson's disease	25	20	16	13	0.8	1.0	1.3	1.6

(a) Sorted according to the leading specific causes for Australia in the year 2003.

Anxiety & depression is ranked first in females across three of the four time periods, although in percentage terms its share of the total burden will decrease from 10.0% in 2003 to 8.7% in 2023 (Table 6.5). Ischaemic heart disease will remain in second place over the next decade, but fall to fourth place by 2023. In its place will be dementia, which increased by 1.1 percentage points to 4.8% of the total burden in the decade to 2003, and, if current projections of population ageing eventuate, will be ranked third at 7.4% of the total burden in 2023. As with males, Type 2 diabetes is set to increase steadily and is likely to occupy second position in 2023, at around 8% of the total burden.

Table 6.5 Leading causes of burden (DALYs) in females, Australia, 1993 to 2023

Specific cause	Rank ^(a)				Per cent of total			
	1993	2003	2013	2023	1993	2003	2013	2023
Anxiety & depression	2	1	1	1	9.8	10.0	9.6	8.7
Ischaemic heart disease	1	2	2	4	12.4	8.9	7.5	6.1
Stroke	3	3	5	5	5.9	5.1	4.4	3.8
Type 2 diabetes	6	4	3	2	3.7	4.9	6.4	8.0
Dementia	5	5	4	3	3.7	4.8	5.9	7.4
Breast cancer	4	6	6	6	5.1	4.8	4.3	3.5
COPD	7	7	8	8	3.1	3.0	2.9	2.8
Lung cancer	10	8	7	7	2.3	2.7	3.1	3.5
Asthma	8	9	9	9	2.9	2.7	2.5	2.4
Colorectal cancer	9	10	10	12	2.6	2.3	2.2	1.9
Adult-onset hearing loss	11	11	11	11	1.5	1.8	2.0	2.2
Osteoarthritis	12	12	12	10	1.4	1.6	1.9	2.2
Personality disorders	15	13	14	16	1.2	1.3	1.3	1.3
Migraine	14	14	17	18	1.3	1.3	1.2	1.1
Back pain	16	15	15	15	1.1	1.2	1.3	1.3
Lower respiratory tract infections	38	16	13	13	0.5	1.1	1.3	1.6
Falls	20	17	18	19	0.9	1.0	1.1	1.1
Parkinson's disease	19	18	16	14	0.9	1.0	1.2	1.5
Schizophrenia	17	19	20	25	1.0	1.0	1.0	0.9
Rheumatoid arthritis	21	20	19	20	0.9	1.0	1.0	1.0

(a) Sorted according to the leading specific causes for Australia in the year 2003.

7 Discussion and conclusions

7.1 Potential applications

A detailed description of the burden of disease and injury in a population is not sufficient for setting priorities in public health. It is, however, an important foundation on which to build assessments and evaluations that underpin health policies. This report contributes most obviously by identifying the magnitude of health problems in a population and by quantifying the contribution to these problems of major modifiable risks to health. The present study greatly extends the scope of the previous study in this respect by presenting burden estimates for a greater range of population subgroups in Australia. It also provides a cogent analysis of past trends in burden in this country, and suggests the likely state of the population's health in 20 years from now if these trends were to continue. Furthermore, it quantifies the contribution to overall burden of an expanded set of risks to health.

Equally important, however, is the contribution of burden estimation to down-stream analyses by the creation of a consistent set of epidemiological parameters for a full range of health conditions, and a detailed description of the relationship between these parameters and risks to health. Again, the study upon which this report is based expands the scope of such analyses through the creation of a comprehensive database of all relevant epidemiological and burden parameters both for a number of different subpopulations and through time. The most obvious synergy here is with cost-effectiveness analyses of the potential outcomes of health interventions. Estimates from the previous study have been used extensively in a number of economic evaluation studies to date (for example, Nelson et al. 2005; Stone et al. 2004; Vos et al. 2005). This new set of results has been incorporated into models under development for the project funded by the National Health and Medical Research Council 'Assessing Cost-Effectiveness (ACE) – Prevention', at the University of Queensland and University of Melbourne, the aim of which is to comprehensively model the cost-effectiveness of preventive intervention options for non-communicable disease in Australia.

The essential link between burden of disease and injury data and cost-effectiveness results is acknowledged by the Pharmaceutical Benefits Advisory Committee, which now requires companies to present evidence on the likely uptake of a new drug in the population. This requires knowledge about the number of people with a particular condition or risk profile for whom the drug is intended. This report will be invaluable as a common reference point.

An important new application of the results of this study will be to further improve estimates of future health expenditure in this country. As life expectancy continues to increase and populations continue to age, this is an area of concern to governments, not only in Australia but around the world. Projections for the 2002 Intergenerational Report relied on models that do not take into account major shifts in epidemiology and expenditure for some diseases. The projections in this report have already been linked to health expenditure data (AIHW 2005c), thus enabling more detailed health expenditure projections for these causes (for example, Vos et al. 2007).

7.2 Policy implications

The preceding section illustrated that the analyses underpinning this report are not sufficient on their own as a basis for setting future directions in health policy. A number of other inputs are also necessary, not the least of which is evidence on the costs and effectiveness of available interventions. Nevertheless a number of important implications for policy arise from the findings presented in the preceding chapters.

This report has presented for the first time a comprehensive overview of the likely effects of population ageing on patterns of disease and injury in Australia over the next 20 years. These projections are based on analyses of past trends in health and are presented as a 'business as usual' scenario (that is, the rate of change in policy responses to emerging problems in the future is consistent with the rate observed in the historical period upon which the projections are based). While all projections regarding the future are uncertain, including those presented in this report, some are more uncertain than others. This is particularly true for the projections for diabetes, where information on trends is limited to one cross-sectional survey and some assumptions regarding changes in case-fatality relative to those observed for ischaemic heart disease (assumptions which are corroborated by the second round of the Australian Diabetes, Obesity and Lifestyle study). Notwithstanding these caveats, the general tenor of these analyses is clear.

A key finding of this study is that while life expectancy is likely to continue increasing steadily, growth in health-adjusted life expectancy will not be as rapid. This is because the number of very old people is set to expand rapidly, mostly due to declining mortality. In addition, while the prevalence of disability will drop at most ages, it is expected to increase for people aged 80 years and over.

A major consequence of this population dynamic will be the steady growth in the burden from diseases associated with old age such as dementia, Parkinson's disease, hearing and vision loss, and osteoarthritis, all causes for which current prevention and (with the exception of osteoarthritis and cataract) treatment strategies are largely ineffectual. The impact of increasing disability from these diseases is likely to be significant and will be felt in the home and the community care sectors before it is felt in the residential aged care and palliative care sectors. While future research into prevention and treatment may yield unexpected results, relevant stakeholders should be planning for growth in the number of elderly people requiring appropriate services in each of these care settings. The economic consequences of these changes on future health care expenditure have been quantified in a separate report (Vos et al. 2007), which it is hoped might assist the development of appropriate policy responses in this area.

In addition, cardiovascular disease are likely to continue to decline relative to cancer as a proportion of the overall burden, primarily because current health care has been relatively successful at curbing the effects of the former, but not nearly as successful with the latter. Successful reductions of cardiovascular disease should not obscure the fact that additional gains could be made by further reductions in levels of cholesterol, blood pressure and smoking, the primary risk factors for these diseases (Taylor et al. 2006). Increasing the coverage and targeting of interventions known to be effective (for example dietary modification, cholesterol and blood pressure lowering drugs, and smoking cessation) is one way of achieving this. There is also likely to be scope for increased efficiency through the adoption of a more cost-effective mix of interventions.

Lastly, the projected strong growth in the burden from diabetes over the next 20 years is an area of concern. This is mostly a consequence of increases in body weight. The consequences of increasing obesity will be further magnified by reductions in case-fatality from cardiovascular disease – the major cause of mortality in people with diabetes – through successful tobacco control and cholesterol and blood pressure lowering strategies. This increased survival will mean an increase in the risk of developing other largely non-fatal but disabling consequences of diabetes such as renal failure, retinopathy, neuropathy and peripheral vascular disease. Efforts to find new approaches to stem rising levels of obesity need to continue.

7.3 Precision of estimates

Fatal burden

The calculation of fatal burden (YLL) is relatively straightforward and the precision of these estimates is almost entirely dependent on the quality of information on underlying cause of death in official mortality data. While every effort has been made to remedy likely distortions to the overall reported cause of death structure by reallocating deaths with certain codes known to be non-specific to valid and specific underlying causes of death, by world standards the extent of these distortions is small (around 6% to 10%, depending on the codes included in this definition). Of greater concern are the deaths coded to valid and specific causes of death. With the exception of a few studies on sensitivity and specificity for specific conditions, relatively little is known about the accuracy of causal attribution for the majority of cases. It is likely that accuracy varies with the location of the death due to differential access to diagnostic information (for example in an institutional setting versus at home), but the assumption that these inaccuracies will cancel each other out at the population level is largely speculative. Further research in this area would greatly enhance the integrity of the vital registration system in this country.

Non-fatal burden

The accuracy of estimates of disability is not quantifiable using formal statistical techniques. This is because, in the construction of these estimates, data of widely varying levels of quality, ranging from population level disease registers and high quality research findings at one end to 'guesstimates' and expert opinion at the other, was drawn upon. Precision is likely to vary greatly between different individual estimates, and ultimately depends on the type of model used and the source and nature of the underlying data. Using simulation methods, it is feasible to quantify an uncertainty interval for each estimate that accounts for confidence in the underlying epidemiological data as well as uncertainties associated with the various assumptions and additional information used. Such an analysis has not been possible in the time frame of this report, however, but may constitute the subject of future research.

In the absence of such analyses, it is worth noting where major sources of uncertainty are likely to lie in more qualitative terms. Among major causes of burden presented in this report, uncertainty is probably highest for hearing loss, neurological conditions, osteoarthritis and cirrhosis. The reasons for suspecting higher levels of uncertainty in these conditions are discussed below.

Hearing loss – although population data on measured hearing loss thresholds were used to estimate disability for this condition, there was considerable uncertainty associated with the modelling of the average durations associated with progressing from mild through moderate to severe hearing loss and, to a lesser extent, the effect of hearing aids on reducing the severity of disability from hearing loss.

Osteoarthritis – estimates for this condition were based on the same overseas studies of incidence and severity used in the previous Australian Burden of Disease and Injury Study. These estimates are lower than would be suggested by the Australian self-reported population data on osteoarthritis. Considerable uncertainty remains about the true incidence of this condition at the population level.

Selected neurological conditions – information on dementia and Parkinson’s disease came from meta-analyses of international community-based studies of prevalence. For the estimates presented in this report, these analyses were updated to include all such studies in Western countries. There is uncertainty about the variations in the level of disease among these countries.

Cirrhosis – there is no easy way to measure the prevalence of cirrhosis at a population level. This report relied on a published modelling effort that projects the progression to cirrhosis in people with hepatitis C. These figures were used to extrapolate cirrhosis from hepatitis B, alcohol dependence and other causes. Considerable uncertainty surrounds these extrapolations.

Mental disorders – these estimates were based on the mental health survey conducted in 1997; more recent data on this large cause of non-fatal burden would have been desirable. Because of high levels of comorbidity between depression and anxiety, as well as the largely similar treatment pathways, these conditions were combined into one entity and modelled over a life course. This departs from the approach taken in the previous study where each condition was treated separately and depression was modelled as an episodic condition.

General levels of uncertainty

In more general terms, it is likely that uncertainty in the estimates of burden presented in this report may not be excessive. Overall, about half of the total burden in Australia was attributable to mortality, for which estimates are fairly robust. Of the remainder, half was attributable to non-fatal burden from a small number of diseases (including cardiovascular disease, cancers, diabetes, common mental disorders, and injuries) for which reasonably good Australian data are available. This leaves around a quarter of the total burden with varying and probably higher levels of uncertainty.

What is clear is that a number of key estimates presented in this report are likely to be much more accurate than those of the previous study. This is due to the availability of considerably better quality data in some cases, one source of which deserves special mention. Access to the linked hospital and mortality databases in Western Australia allowed greater accuracy in the modelling of cardiovascular disease, the second leading cause of burden in Australia. As Western Australia adds more health information data sets to its linkage program – including health surveys, disease registries, Medicare and Pharmaceutical Benefits Scheme (PBS) data – this will become an even more valuable resource, both for Western Australia and nationally. Various efforts are in train to encourage data linkage in other jurisdictions and at the national level. For example, the Statistical Information Management Committee (a multi-jurisdictional committee established by health CEOs) has commissioned the development of a framework for national data linkage, which takes careful account of such concerns as

privacy, data protection, and custodianship. These efforts are to be encouraged, as they will underpin improvements in future analyses of the burden of disease and injury in Australia.

Aside from data inputs, it is also worth mentioning the tools used. The second version of the epidemiological modelling software DisMod allowed much more accurate modelling of consistent epidemiological parameters than was possible in the previous study. In using this software, particular attention was directed towards a consistent application of the concept of 'excess risk' (that is, people with a condition are often more at risk of dying than would be indicated by the mortality actually coded to that condition) by accessing information from international cohort studies and the linked hospital and death databases in Western Australia.

DisMod also allows for past trends in incidence and case-fatality to be modelled, which is particularly important for diseases with prevalence as the main observed parameter and for which there have been significant trends in the past. This is because prevalence is a 'stock' variable and is simply a reflection of past trends in incidence and case-fatality. In this study, there was a strong upward trend in the incidence of diabetes, accompanied by an improvement in case-fatality. Ignoring these trends would have led to an underestimate of the true incidence of this disease. One area where DisMod was unable to help, however, was in replicating final epidemiological models to multiple subpopulations and to various points through time. For this application a custom-built routine was developed within a statistical package environment based on DisMod's underlying equations.

A final issue that is relevant to precision considerations is the set of disability weights assigned to each health state. To date, a comprehensive set of weights has not been derived in the Australian context. It was therefore necessary to continue to use an assortment of weights derived largely from a Dutch study supplemented with weights from the original Global Burden of Disease study and weights derived from a regression model on the Dutch weights and the six domains of the EQ5D+ (an instrument to quantify quality of life). Funding requested as part of the current study to validate these disability weights in the Australian context has not been forthcoming. Also, internationally there has not been the expected further development of measuring health state preferences to determine disability weights. The large effort of collecting data in the World Health Surveys by the World Health Organization in the first part of this decade has not yet resulted in any publication.

While this may raise concerns about the construct validity of the non-fatal estimates of burden presented in this report, it should be noted that the rank order of weights for most conditions has strong face validity and has been documented to be reasonably constant for a set of 'tracer conditions' when replicated in different countries. Of greater concern are the disability weights for common but low-severity conditions such as mild hearing loss, mild vision loss, uncomplicated diabetes, asthma and anaemia. Existing health state evaluation methods do not seem to accurately capture differences in severity between such conditions. The lack of valid disability weights for distinguishing between high-prevalence low-severity conditions is more important than it sounds because a small absolute difference in the disability weight for a highly prevalent condition has a major bearing on the size of the burden attributable to that condition. This is an area in need of further development.

Lastly, with respect to disability weights, a major improvement of this study compared with most burden studies has been a comprehensive correction for coexisting health states. In the previous study, an attempt was made to deal with comorbidities within mental disorders, injuries and common causes of burden in the elderly, although each group was treated separately and the latter was not comprehensive. Furthermore, the methods used assumed no dependence between health states (that is, groups of conditions being more likely to

coexist due to common causal pathways). The methods developed for the present study drew upon multiple surveys and hospital data to derive probabilities of coexistence for all possible combinations of health states that were modelled using an innovative microsimulation approach. Dependence is implicitly accounted for in this approach. Corrections for comorbidity in burden estimates are necessary because disability weights are typically derived in isolation from each other, meaning that coexisting disability in the same person is unlikely to be simply additive across two or more health states.

7.4 Access to data

Previous burden estimation work has been done mostly within a spreadsheet environment. The benefits of this approach are that transparency and portability are maximised and, to date, no other approach has achieved the level of flexibility afforded by working in this way. However, accuracy can be a problem in that, in a spreadsheet, location is critical and incorrect cell references are common. More importantly, spreadsheets rapidly become unwieldy when more than several dimensions are being represented. In this study, spreadsheets were retained for all basic disability modelling work, but a statistical package environment was used for all subsequent analyses. The first practical implication of this decision is that a set of spreadsheets containing all disability models exists and will be made available to those who are interested. Users of this resource should note, however, that the disability estimates in these spreadsheets will usually not correspond exactly with final estimates in this report because comorbidity corrections and some trend corrections occur outside this environment.

The other more interesting implication is that having extracted all relevant information from the spreadsheets and derived final burden estimates outside this environment, the results can be reassembled in any requested structure. This is particularly relevant for the estimates for subpopulations and for various points through time. In collaboration with various jurisdictional stakeholders, this information can be grouped into meaningful aggregations for specific health policy and planning purposes. In addition, a web-based interface could be developed whereby users could extract the desired information by cause, age, gender, time and subpopulation. The website developed for disseminating the 1996 and 2001 Victorian Burden of Disease study results is a useful model (see www.dhs.vic.gov.au/health/healthstatus/bod/bod_reg.htm).

7.5 Future directions

This study comes seven years after the original Australian Burden of Disease and Injury Study. It has taken three years to complete, with the equivalent of 2-3 full-time staff and considerable intermittent assistance from researchers from several state health departments and masters students. Such a commitment of resources to this type of research is unlikely to occur again in this country in the short term. One of the aims of the study, therefore, was to develop a less resource-intensive way of retaining an up-to-date set of burden estimates going forward in time. To this end, a database of estimates for each year between 2003 and 2023 has been developed. This will provide an invaluable set of 'base-level' results for those wanting to make assessments of burden in the period prior to the next major update. Such assessments may entail varying levels of sophistication, from simple updates of fatal burden using the most recent mortality data to ad hoc changes to specific non-fatal estimates based

on advances in knowledge and better data. Through the adoption of strategies such as these, a major revision may not be needed for at least another five years.

The final report of the previous Australian Burden of Disease and Injury Study identified seven areas where it was felt priority should be placed in future research. Progress has been made in a number of these areas over the last seven years, four of which are addressed specifically by the present study. These include the development of burden estimates for Indigenous Australians, a more detailed assessment of differentials in burden across Australia, including estimates by socioeconomic status, remoteness and jurisdictional boundaries, and more detailed modelling of National Health Priority Area diseases. A fifth suggestion regarding the value of linking burden research to cost-effectiveness analyses has been picked up in a number of separate studies around the country over this period.

Progress on two recommendations, however, has been less rapid. It has not been possible to estimate and validate a set of disability weights in the Australian context and this remains an important area requiring further development. Finally, there has been no formal evaluation of the usefulness of burden of disease and injury analyses for policy makers and health planners. There has been enough informal feedback from planners, researchers and the media to know that there is consistent demand for this type of information. But a more formal analysis of the impact on health assessment, policy and planning of this research over the last seven years would be welcome.

Appendix 1: Methods for estimating disability burden

In this section we describe our methods for calculating disability for the large number of diseases and injuries and their sequelae for which models were developed, including all those that make significant contributions to the total non-fatal burden. While this list is extensive, it is not exhaustive, and explicit models were not developed for many conditions. Table A1.1 lists the full names of many of the data sources underlying our models and our abbreviations of these names, which we use in this section for ease of reference.

Table A1.1: List of full and abbreviated names of commonly used data sources

Abbreviated name	Full name
AusDiab	The Australian Diabetes, Obesity and Lifestyle Study, 1999–2000 (Dunstan et al. 2001)
Australian dialysis and transplant data	2002 Australian and New Zealand Dialysis and Transplant Registry (McDonald & Russ 2002). The interpretation of this data is the responsibility of the authors of this report and should not be seen as the interpretation of the Australian and New Zealand Dialysis and Transplant Registry.
Australian disability survey	Survey of Disability, Ageing and Carers (1993, 1998 or 2003) (ABS 1993, 1998b, 2003b)
Australian general practitioner data	2000–01 and 2002–03 Bettering the Evaluation and Care of Health (AIHW: Britt et al. 2001)
Australian hospital data	2002–03 National hospital morbidity database (AIHW 2003a)
Australian mortality data	2003 Cause of Death dataset (ABS 2005)
Australian notification data	National Notifiable Infectious Disease Surveillance System (CDA, 2003) except for HIV/AIDS which is from the National Centre for HIV Epidemiology and Research (National Centre in HIV Epidemiology and Clinical Research, 2003)
Australian perinatal data	2002 Australia's mothers and babies and various state and territory perinatal data collections (AIHW: Laws & Sullivan 2004; Queensland Health 2004; Riley & King 2003).
Disability weight regression model	Regression model of Dutch disability weights which requires inputs of health state description based on the six domains of the EQ5D+ (p. 158 of AIHW: Mathers et al. 1999)
DisMod	DisMod version II (Barendregt et al. 2003)
GBD study	Global burden of disease and risk factors, 2000 (Lopez et al. 2006)
Low prevalence study	1997–98 Low Prevalence (Psychotic) Disorders Study (Jablensky et al. 1999)
National Health Survey	2001 National Health Survey (unless otherwise specified as the 1995 National Health Survey) (ABS 1995, 2001c)
National mental health survey	1997 National Survey of Mental Health and Wellbeing (ABS 1997)
National Trachoma Survey	1980 National Trachoma and Eye Health Program (Royal Australian College of Ophthalmologists 1980)
Previous Australian burden study	Australian Burden of Disease and Injury Study, 1996 (AIHW: Mathers et al. 1999)
Victorian birth defect data	2001–02 Victorian Birth Defects Register (Riley & Halliday 2004)
Victorian linked hospital dataset	Analyses of Victorian hospital data 1996–2002 & 2001–02 from the 2001 Victorian Burden of Disease and injury study (DHS, 2005)
Women's health Australia	Australian longitudinal study on women's health (Lee et al. 2005)

1A Infectious and parasitic diseases

Tuberculosis

We estimate the incidence of tuberculosis using Australian notification data on new cases of tuberculosis. We assume that the average duration for tuberculosis is 8 months, reflecting 6 months for the shortest treatment cycle available and another 2 months of symptoms before treatment.

Sexually transmitted diseases (excluding HIV/AIDS)

We base our incidence estimates for syphilis, chlamydia and gonorrhoea on Australian notification data. Following expert advice, we assume that annual notifications for syphilis and gonorrhoea represent all incident cases. We model syphilis using a staged approach applying proportionate distributions for primary, secondary, and tertiary syphilis from the GBD study. We adjust our estimates for chlamydia to account for under-reporting due to asymptomatic infections and the reluctance of some patients to consult general practitioners about sexually transmitted diseases. We base our incidence estimates of pelvic inflammatory disease, a complication of both chlamydia and gonorrhoea in women, on Australian hospital data. Following expert advice we adjust these estimates to account for under-identification. Common sequelae of pelvic inflammatory disease include ectopic pregnancy, chronic pelvic pain, infertility and tubo-ovarian abscess. We base our rates of complications following pelvic inflammatory disease on GBD assumptions. We adjust our incident estimates of infertility resulting from pelvic inflammatory disease for women who do not wish to have a child and therefore do not experience disability. In the absence of Australian data on 'child wish', we make this adjustment using findings from a recent German study (Stobel-Richter et al. 2005). The GBD reports urethral stricture and epididymitis as complications following chlamydial and gonorrhoeal urethritis in men. These complications were thought by experts to be rare, and so have not been included in the Australian estimates. We model disability weights and durations for syphilis, chlamydia and gonorrhoea and their sequelae using the assumptions of the GBD study.

HIV/AIDS

We model HIV as a progressive condition with four stages: (1) asymptomatic HIV; (2) symptomatic HIV; (3) AIDS prior to terminal phase; and (4) terminal AIDS. We assume that the annual number of new HIV diagnoses from Australian notification data represent all incident cases of HIV. We use the Dutch disability weights for each of the stages (stage 1 – 0.2, stage 2 – 0.31, stage 3 – 0.56 and stage 4 – 0.95) and adjust the weight for stage 1 to account for the estimated proportion of undiagnosed asymptomatic HIV cases to whom we assign a disability weight of 0 (Aalen et al. 1997). We calculate the mean durations for stages 1 to 3 using Weibull regressions of published data accounting for background mortality (Kaldor & McDonald 2003; Mocroft et al. 1997; Porter et al. 2003). This gives average durations of 30 years for the combined stages 1 and 2 and 5.5 years for stage 3. We adjust our duration estimates for stage 1 and 2 based on the assumption that an equal amount of time is

spent in each stage based on work by Aalen and colleagues (1997). In the absence of new evidence, we assume that stage 4 lasts an average 0.5 of a year.

Diarrhoeal diseases

Diarrhoeal diseases include a number of notifiable diseases as well as non-notifiable diseases. Given that notifications are generally considered a gross underestimate of the incidence for notifiable diarrhoeal diseases, and that there is often even less reliable information on the incidence of non-notifiable diarrhoeal diseases, we do not model diarrhoeal diseases using notification data or by specific cause. Instead, we derive the incidence of diarrhoea not requiring hospitalisation using annualised self-reported data from the 2001–02 National Gastroenteritis Survey (Hall & OzFoodNet Working Group 2004). We base our duration of 2 days from the findings of this survey and use age-specific weights for uncomplicated diarrhoea (average weight of 0.093) from the GBD study. We use Australian hospital data to estimate the incidence of diarrhoea cases requiring hospitalisation. We use the age-specific GBD weight for diarrhoea (0.093) since the Dutch weight is implausible. We assume 2 weeks duration for complicated diarrhoea and derive an average weight (0.42) based on 1 week of disability equivalent to the regression model of health state (323311) and 1 week of disability for uncomplicated diarrhoea.

Childhood immunisable diseases

We do not model poliomyelitis and diphtheria for 2003. This is because there were no notifications of poliomyelitis from 1993 to 2003 and only one notification of diphtheria in 2001.

Pertussis

We estimate the incidence of pertussis using Australian notification data averaged over 2000–2003, an epidemic cycle. We adjust our incidence estimates for under-reporting based on the literature (Andrews et al. 1997; Torvaldsen et al. 2002). We apply the age-specific GBD disability weights for untreated cases for pertussis (0–4 years: 0.178; 5–14 years: 0.166; 15 years or over: 0.156), since the weight for treated cases is implausible, along with the GBD duration of 1 month. Following expert advice we estimate the incidence of intellectual disability attributable to pertussis as the proportion of intellectual disability cases from the total episodes of infection for 0–4 year olds in the GBD study (that is, 0.3% of pertussis cases). We derive a disability weight (0.58) for pertussis-related intellectual disability by weighting the number of cases of intellectual disability due to infectious diseases by the level of severity (using the Dutch weights for intellectual disability).

Tetanus

We estimate the incidence of tetanus using Australian notification data and apply the GBD disability weight for 60 years or over of 0.612, and duration of 2 weeks.

Measles

We derive the incidence of measles using Australian notification data. We assume annual notifications in 2003 represent all cases of measles due to enhanced surveillance (Brotherton et al. 2004). For acute measles episodes we apply the GBD duration and disability weights (2 weeks, 0.152). We use Australian hospital data to estimate the incidence of measles sequelae. In 2003 there were no hospitalisations for measles encephalitis, and only one for sub-acute sclerosing panencephalitis. For the latter sequelae we apply the Dutch disability weight for end stage disease with a duration of 9 months.

Rubella

We derive the incidence of rubella using Australian notification data which we adjust for over-reporting. Enhanced surveillance of rubella notifications in Victoria found that 27% were laboratory confirmed (Guy et al. 2004). As there is no GBD or Dutch weight for rubella we use the measles disability weight (0.152) with a duration of one week. We use Australian notification data to derive incidence estimates of congenital defects due to rubella; there were only three such cases in 2003. The classic triad of complications associated with congenital rubella infection are cataract, heart disease, and deafness. In the absence of more specific information, we derive an average disability weight and durations to reflect each of these complications.

Haemophilus influenzae type b

We derive the incidence of *Haemophilus influenzae* type b from Australian notification data. We only model the disability associated with the following sequelae – meningitis, epiglottitis, septicaemia, pneumonia and ‘other’ using data from an Australian study (Herceg 1997). Following expert advice we assume that all cases of epiglottitis and meningitis are confined to the 0–14 year age group and pneumonia and septicaemia to the 15 years or older age group. We assume that meningitis from *Haemophilus influenzae* type b is included in the hospitalisation-based estimates of total meningitis and subtract these cases from the total incidence estimates of meningitis to avoid double-counting. We use the same disability weights and durations for these sequelae as per the previous Australian burden study.

Meningitis

We estimate the incidence of meningitis from Australian hospital data which we adjust to avoid double-counting of meningitis from *Haemophilus influenzae* type b. We model meningitis as a progressive condition with acute episodes of one month, after effects lasting up to six months and subsequent lifelong effects, in some, for a range of conditions (including hearing loss, ventriculoperitoneal shunt, seizure disorder, less severe developmental problems, mental retardation and motor deficit and physical deformities). We make minor modifications to the assumptions in the Dutch study regarding proportions of meningitis cases progressing to sequelae and their associated disability using the results of a seven-year follow-up study of meningitis in Melbourne children (Grimwood et al. 1995) and expert opinion.

Septicaemia

We estimate incident cases of septicaemia from Australian hospital data. We do not adjust our estimates to account for meningitis-related septicaemia as Victorian data suggests that less than 2% of cases are due to meningitis. In the absence of a weight for this condition in its uncomplicated state, we use the Dutch weight for meningitis for an average duration of 1 month (Stouthard et al. 1997).

Arbovirus infections

We estimate the incidence of arbovirus infections using Australian notification data. Because there are no specific disability weights for arboviruses we use comparable weights from the Dutch study. For Ross River and Barmah Forest viruses we adjust estimates by 100% to account for under-reporting in endemic areas. We model Ross River and Barmah Forest viruses as a febrile illness in children aged up to 14 years and as an illness with acute and chronic stages for incident cases aged 15 years or over. Based on Australian literature we use the Dutch weight for influenza for children (1 month duration) and the Dutch weights for moderate rheumatoid arthritis (1 month duration) and mild arthritis (3.5 months in Ross River fever and half of this duration for Barmah Forest virus) for acute and chronic stages respectively in adults (Mylonas et al. 2002; Russell 2002). In general, arthralgia persists longer in Ross River virus infection than in Barmah Forest virus infection (Mackenzie et al. 1998; Russell & Dwyer 2000), therefore we halve the duration of the chronic phase in the latter.

We adjust notifications for dengue fever by 10% to account for under-reporting. Based on the literature we use the Dutch weight for malaria with a duration of 6 days (Russell & Doggett 1998; Solomon & Mallewa 2001). We use Australian hospital data to estimate the incidence of the rare and disabling sequelae dengue haemorrhagic fever. There were only two cases in 2003. The GBD weight for this condition appears too low and so we apply the Dutch weight for meningitis for just over 1 week.

We model the following flavivirus infections as 'other arbovirus infections': Murray Valley encephalitis, Kunjin virus infection, Japanese encephalitis, and flavivirus not elsewhere classified. In 2003 there were no notifications for Murray Valley encephalitis and only one case of Japanese encephalitis notified. We apply GDB estimates of the incidence of sequelae (episodes, cognitive impairment and neurologic sequelae), average disability weights, and duration for Japanese encephalitis to all other arbovirus infections.

Hepatitis

Hepatitis A

We estimate the total incidence of hepatitis A using Australian notification data, which we adjust for under-reporting (Amin et al. 1999). We assume that the 10% of incident cases represent prolonged hepatitis A. We assume that Australian hospital data on hepatitis A represent all cases of complicated hepatitis A. We calculate the number of incident cases of uncomplicated hepatitis A by deducting the prolonged and complicated cases from our total estimate. Due to the implausibility of the Dutch weight for uncomplicated hepatitis A we use the average GBD weight of 0.093 with a duration of 3 weeks (Amin et al. 1999). We assume that prolonged hepatitis A cases experience depression or fatigue for 6 months at disability

weight equivalent to the Dutch weight for mild depression (0.14) (McIntyre 1990; Willner et al. 1998). We assume durations of 4 weeks for children and 6 weeks for adults (Melnick 1995). We apply a severe disability weight for half of this time (DW 0.747), and the remaining time at the same weight as uncomplicated cases. This gives an average weight of 0.42.

Hepatitis B

We estimate the incidence of acute hepatitis B using Australian notification data and assume that all infections reported as incident are symptomatic.

We derive incidence estimates for acute symptomatic hepatitis B infection in infants from birth data and probabilities of perinatal transmission for 'at risk' mothers as reported by Kaldor and colleagues (1996). Based on the literature we assume a 40% probability of transmission if exposed. Using this estimate we can calculate the number of infants who would be infected in the absence of vaccination (Kaldor et al. 1996). As current vaccination coverage in children born to mothers 'at risk' is 95% (Menzies et al. 2004), we reduce the number of carriers from perinatal transmission accordingly. Similarly we adjust the number of perinatal infections for the probability of symptomatic infection which is 5% (Kaldor et al. 1996). Based on expert opinion, we assume a similar number of infections by casual contact in childhood and for males and females.

We base our estimates of chronic hepatitis B on a series of DisMod models. First we estimate the prevalence of adult carriers using an overall prevalence of 0.47% (O'Sullivan et al. 2004), a remission of 0.5%, and an overall relative risk of mortality of 1.5. Next we estimate the prevalence of adult carriers using incidence estimates of carriers from perinatal and casual childhood transmission assuming no vaccination had occurred. We then subtract the prevalence of carriers from childhood infections from the first model so we can use DisMod to derive the incidence of chronic hepatitis B infection in adults. This model assumes a steady state of hepatitis B infection in the population, with vaccination only recently affecting perinatal and childhood transmission rates. This is unlikely to reflect the pattern of disease over time, but in the absence of data on the trends over time, this was considered the most plausible method of modelling the disease following expert consultation.

We assume the average duration for an acute symptomatic episode to be 4 weeks (Lee 1997). We use the Dutch disability weight for acute hepatitis infection (0.21). We adjust the Dutch weight for chronic hepatitis B infection with active viral replication (0.36) following expert advice that only 15% of chronic cases have a symptomatic episode for 2 weeks each year (giving an average weight of 0.002). The methods we use to derive YLD for hepatitis B-related cirrhosis and liver cancer are described in the following section on hepatitis C. test

Hepatitis C

Due to the asymptomatic nature of hepatitis C infection we assume that all YLD are a result of hepatitis C sequelae, that is, cirrhosis and liver cancer.

There is a paucity of information on the occurrence of cirrhosis at a population level. Instead, we make use of estimates of hepatitis C-related cirrhosis occurrence from an Australian study which modelled the progression rates to various sequelae from hepatitis C incidence (Law et al. 2003). The major problem in estimating the occurrence of hepatitis C-related cirrhosis is the dramatic change in hepatitis C incidence over the last 5 decades, the relevant time period for the development of current cirrhosis cases. The best available approximation

of the pattern of hepatitis C epidemiology over the last 40 years is based on the pattern of injecting drug use over time (Law et al. 2003).

We make largely the same assumptions in the modelling of hepatitis C-related cirrhosis as in Law and colleagues (2003):

- 75% of people exposed to hepatitis C develop chronic infection
- an annual progression rate of 2% to cirrhosis
- a hepatitis C-related mortality rate of 1.5% following cirrhosis
- mean age of hepatitis C seroconversion among injecting drug users of 25 years
- a male to female ratio of 2:1 for persons who inject drugs and are hepatitis C-infected
- unlike in Law et al. (2003), we assume that 80% of those exposed to the hepatitis C (the estimated proportion of hepatitis C carriers exposed through injecting drug use) have a relative risk of mortality of 13 (Darke & Ross 2002) for an average of 14 years from the moment of exposure (as estimated for heroin dependence)
- background mortality is calculated from life tables constructed from Australian mortality and population data from 1950 to 2003.

Based on this model, we estimate 447 new cases of hepatitis C-related cirrhosis and 5,804 people living with cirrhosis due to hepatitis C in 2003. Next, we examine the Australian hospital data for cirrhosis. In all cases in 2003 with a stated underlying cause, 49.4% are alcohol related and 50.6% non-alcohol related. Based on expert opinion we attribute 5% of non-alcohol related cirrhosis to other causes and the remainder to hepatitis. We estimate the occurrence of cirrhosis due to other causes (that is, hepatitis B, alcohol abuse, and 'other') by adjusting Australian hospital data by the admissions-to-prevalence ratio observed in hepatitis C-related cirrhosis cases. We only give a disability weight for the last 3 years lived with cirrhosis at 0.31 (minus 2 months) and 0.84 for the last 2 months (effectively interpreting the Dutch weight for compensated cirrhosis as relevant for the time spent in decompensated cirrhosis and the decompensated cirrhosis weight of the Dutch as the weight for terminal liver failure).

In the previous study we assumed liver cancer occurred in around 19% of people with hepatitis C and hepatitis B. More recent data, including from a large multi-centre study of liver cancer patients in Europe, indicates that hepatitis B and C are responsible for around 19% and 40% of liver cancer respectively (Brecht et al. 1998; CDC 2001).

Malaria

We derive incidence estimates for malaria from Australian notification data. We model two aspects of malaria, episodes and neurologic sequelae and adopt GBD assumptions for disability weights and durations.

Trachoma

We model the disability associated with mild, moderate and severe vision impairment resulting from trachoma infection. We assume that trachoma related visual impairment is a problem only in remote Australia. We estimate the prevalence of trachoma-related visual impairment using the 1980 National Trachoma and Eye Health Program. Based on expert advice we adjust the prevalence downwards by one-third to account for observed decreases

in the prevalence of scarring stages that follow infectious trachoma since the national survey was conducted (Landers et al. 2005; Mak & Plant 2001). In the absence of more specific information we assume that mild and moderate vision loss have the same cause distribution by age as severe vision loss. We make minor adjustments to the prevalence of each stage by age to ensure plausibility and to reflect published estimates. We estimate the incidence and duration of trachoma in DisMod using our derived prevalence estimates. We initially model the prevalence of severe vision loss in DisMod assuming no remission and a relative risk of mortality of 1. We then use the incidence of severe vision loss from the DisMod output as 'mortality' in the moderate vision loss DisMod model. This takes the cases of severe vision loss out of the pool of susceptible cases for moderate vision loss and therefore gives more accurate average durations than if we were to use remission as remitted cases in the DisMod model, as the cases continue to be subject to the hazard of incidence. Similarly we use the incidence of moderate vision loss as 'mortality' in mild vision loss.

1B Acute respiratory infections

Lower respiratory tract infections

We base our incidence estimates for lower respiratory tract infections, including episodes of influenza, acute bronchitis and pneumonia, on Australian general practitioner data. For pneumonia, general practitioner data was thought to be more representative than hospital data as it should include those who do and do not go to hospital. We use the same assumptions for disability and durations as in the previous Australian burden study. GBD duration estimates were halved to 3.5 days for acute bronchitis, and left at 1 and 2 weeks respectively for influenza and pneumonia. Disability weights were derived using the regression model (influenza 0.047; acute bronchitis 0.132; pneumonia 0.373).

Upper respiratory tract infections

We base our incidence estimates for episodes of acute nasopharyngitis and acute sinusitis on annualised self-report data from the National Health Survey, while we model tonsillitis and pharyngitis using Australian general practitioner data. We use the data from the 1995 National Health Survey because the 2001 survey did not include questions on acute conditions. We adjust the tonsillitis and pharyngitis incidence estimate upwards by twofold to reflect the much higher rate (13 times) for the broader condition of 'sore throat' that was reported in the survey. We use derived weights and assume GBD durations, with minor adjustments where we consider this to be appropriate. For employed adults, the average number of days off work due to upper respiratory tract infections was around 0.5 of a day. The GBD assumed an average duration of 3.5 days. The self-report prevalence data probably includes a considerable number of minor infections with minimal disability. Hence we use days off work plus half a day on either side to give an average duration of 1.5 days for acute nasopharyngitis. For tonsillitis and pharyngitis and sinusitis we use the GDB durations of 3.5 days.

Otitis media

We model the following stages of otitis media; acute infection, bilateral chronic infection, and life-long deafness. We estimate the incidence of acute episodes using Australian general practitioner data. We assume that those who have relatively low disability do not seek treatment and base YLD estimates on treated numbers. We adjust our incidence estimates to allow for a higher rate of acute otitis media in Indigenous Australians in remote areas based on findings from the 1980 National Trachoma and Eye Health Program. We use the disability weight regression model to derive an appropriate weight (0.090) and assume a duration of 1 week.

We estimate the prevalence of chronic otitis media in non-Indigenous and Indigenous Australians from the National Health Survey for those people reporting otitis media as a long-term health problem. We assume that these estimates represent non-Indigenous Australians in all areas and Indigenous Australians in major city or regional areas. We adjust these prevalence estimates downwards to account only for bilateral cases using a ratio of bilateral to unilateral cases from the 1980 National Trachoma survey. We estimate the prevalence of bilateral chronic otitis media in Indigenous Australians in remote areas from the 1980 National Trachoma survey and assume that the epidemiology of bilateral chronic otitis media has not changed since the survey was undertaken. We derive the incidence and duration of bilateral chronic otitis media in DisMod using prevalence, a relative risk of 1 and remissions equivalent to durations of 3 months and 3 years for non-Indigenous and Indigenous Australians, respectively, based on Australian data (McGilchrist & Hills 1986). We base our estimates for permanent hearing loss resulting from acute infections on the GBD study. For chronic infection we apply the Dutch weight for early acquired mild to moderate hearing loss (0.110). For the small number of cases that experience lifelong deafness, we use the Dutch weight for early acquired severe hearing loss (0.233).

1C Maternal conditions

We base our incidence estimates for maternal haemorrhage, maternal sepsis, hypertension in pregnancy, obstructed labour, abortion and other maternal conditions on Australian hospital data. We adopt GBD methods except in the following instances. On expert advice we assume hypertension in pregnancy results in restricted activity (due to advised bed rest or hospitalisation) for 2 months at a derived weight of 0.117 (health state 122111), with 1 in 2,500 cases developing neurological sequelae. We model the sequela caesarean section with 2 weeks of disability at a derived weight of 0.349 (health state 222111). We base our incidence estimates for abortions using South Australian data on terminations of pregnancy as a proportion of total births (Chan et al. 2003). For abortion we model the disability of infertility resulting from the sequela pelvic inflammatory disease. We assume that 20% of hospitalised cases of pelvic inflammatory disease following abortion experience infertility from age at infection to post-reproductive age which we assume to be 45 years. We adjust our incident estimates of infertility, in the abortion and maternal sepsis models, for women who do not wish to have a child and who therefore do not experience disability. In the absence of Australian data on 'child wish', we make this adjustment using findings from a recent German study (Stobel-Richter et al. 2005). Although stress incontinence was considered a sequela of obstructed labour in the GBD study, most stress incontinence occurs in the absence of such a history. We therefore treat this condition as a category in its own right, classified under 'genitourinary conditions'.

1D Neonatal causes

Birth trauma and asphyxia

We estimate the incidence of mild, moderate and severe birth asphyxia using Australian hospital data. We separate the mild and moderate incident cases using data from the GBD study. We base our sequela estimates of neurological disability by severity of birth asphyxia (0% of mild, 25% of moderate and 100% of severe) on the GBD study.

We use the estimates of intellectual disability due to birth trauma from the overall calculations for intellectual disability by all underlying causes (see Section 2K). Stanley and colleagues (1995) estimated that 8% of cerebral palsy is associated with birth trauma. The balance of the incident cases of permanent disability is divided equally between deafness and seizures.

We assume that the duration of cerebral palsy without intellectual disability and severe hearing loss is the same as those with mild intellectual disability. We base the duration of seizure on life expectancy at birth assuming a twofold risk of dying to indicate a greater likelihood of premature mortality.

Low birth weight

We estimate the incidence of low birth weight ($\geq 1500\text{g}$ and $< 2500\text{g}$) and very low birth weight ($< 1500\text{g}$) in neonatal survivors using Australian perinatal data. We apply the sex distribution of low birth weight from the 2002 Victorian perinatal data to the Australian combined proportion for both sexes which we then apply to the total number of live births in Australia in 2003 (ABS 2004). We adjust our estimates of total neonatal deaths in 2003 (ABS 2005) using a proportion for those due to low birth weight. This was derived using an average of 2002 Victorian, Queensland, and South Australian data.

We assume the probability of disability among low birth weight survivors is 25% for very low birth weight ($< 1500\text{g}$) and 5% for low birth weight ($\geq 1500\text{g}$ and $< 2500\text{g}$) as per the GBD study. This corresponds to a total of 1,230 incident cases (596 males, 634 females) of disability in low birth weight survivors in 2003.

For hearing loss, vision loss, epilepsy, and other disability we distribute the incident cases of disability in low birth weight survivors to disability type from the GBD study. We use the estimates of intellectual disability due to low birth weight from the overall estimates of intellectual disability (see Section 2K). In addition we attribute 60% of total incident cerebral palsy cases (at 2.25 per 1,000 live births) to low birth weight.

Just over one half of the low birth weight survivors with permanent disability do not have severe neuro-developmental disability. In the absence of a defined disability weight for this health state we assume that these cases have a level of disability similar to the Dutch weight for permanent early childhood acquired moderate hearing loss. For all other sequelae we apply the relevant Dutch disability weight.

We assume the duration of cerebral palsy without intellectual disability, severe hearing loss, moderate vision loss, and mild permanent disability to be the same as those with mild intellectual disability. We base the duration of epilepsy on life expectancy at birth assuming a twofold risk of dying as compared to the mortality rates of the general population.

Neonatal infections

We estimate the incidence of neonatal infections from Australian hospital data. We assume 1 month of acute disability using the Dutch weight of 0.894 (same as for meningitis) for acute episodes.

The main long-term sequelae are deafness, motor deficit disability and intellectual disability. We estimate intellectual disability attributable to neonatal infections as part of overall estimates for all causes of intellectual disability (see Section 2K).

Other conditions arising in the perinatal period

Here we include YLD for intellectual disability due to other conditions arising in the perinatal period.

1E Nutritional deficiencies

Iron deficiency anaemia

We model the following levels of severity for iron deficiency: non-anaemic, mild anaemia, moderate anaemia and severe anaemia. We define anaemia in terms of blood haemoglobin levels as per the GBD study. We derive our incidence estimates for iron deficiency anaemia using DisMod. We base our prevalence estimates for mild, moderate and severe anaemia for men and women aged 25 years and above from AusDiab. For the younger ages we use a variety of Australian studies, assuming 60% of cases are mild and the remaining 40% moderate (English & Bennett 1990; Karr et al. 1996; Nguyen et al. 2004; Oti-Boateng et al. 1998; Sadler 1996;). Iron deficiency causes anaemia but people can be iron-deficient and not anaemic and vice versa. To calculate iron deficiency without anaemia, we first have to estimate the prevalence of total iron deficiency which includes those with and without anaemia. We assume prevalence estimates of 10% and 1% in children aged 0–4 years and 5–14 years respectively (English & Bennett 1990; Mira et al. 1996; Oti-Boateng et al. 1998; Rangan et al. 1998; Ranmuthugala et al. 1998; Sadler 1996), with figures for other ages taken directly from 1989 National Risk Factor Prevalence Survey – Iron status study. In the absence of population data on the overlap between iron-deficiency and anaemia, we assume half the cases with mild anaemia and all cases with moderate anaemia are also iron-deficient. For adults aged 15 years or over, we subtract the prevalence of iron deficiency combined with anaemia from the total prevalence of iron deficiency to avoid double-counting the disability. We use the same assumptions for disability and duration as in the previous Australian burden study.

2F Malignant neoplasms

As in the previous study, the basis of YLD estimation for malignant neoplasms is a series of models of disease progression developed by the Dutch burden of disease study team for 26 cancers for which they determined disability weights (Stouthard et al. 1997).

The disease model commences with an initial phase of diagnosis and primary therapy, with a duration of up to 12 months. After this, cases are classified as those who will and will not

be cured. Those who will be cured enter a phase of up to 5 years after which they are considered cured and have (with some exceptions as discussed below) no further cancer-related disability. Those who will not be cured enter a phase (of variable length) of remission followed by a phase of disseminated carcinoma (lasting 12 months or less), then a terminal phase (lasting 1 month) and death.

We allocate a Dutch weight to each of these phases. Where no Dutch weights were available for a specific cancer site, we extrapolate weights based on the cancer that it most resembles. The Dutch study did not derive a weight for the terminal phase of any of the cancers, so we use instead the Dutch weight for general end-stage disease.

We modify this general model to each cancer site with results from studies in the peer-reviewed literature and input from local clinicians to reflect local treatment practices.

Disease incidence data

The primary source of cancer incidence data is the AIHW & AACR National Cancer Statistics Clearing House database (AIHW & AACR 2001). This database records all cancer cases (except non-melanoma skin cancer) notified in Australia from 1982 to 2001. Cancer incidence rates in the Australian population change very slowly (AIHW et al. 2005). We apply the 2001 age- and sex-specific cancer incidence rates to the 2003 Australian population counts to estimate the 2003 cancer incidence.

Two exceptions to this approach are breast cancer and non melanoma skin cancer. The breast cancer disease model requires details of size of tumour at diagnosis which are not available from the Clearing House database. Instead, we extrapolate the proportion of new cases in each size category from 2001 BreastScreen Australia data (AIHW 2005a) and all incident cases from the AIHW breast cancer size and nodal status report for 1997 (AIHW et al. 2001). We then apply these proportions to the 2003 incident cases projected from the Clearing House database. Non-melanoma skin cancer is not a notifiable disease in Australia and so is not within the scope of the Clearing House database. Instead, we extrapolate the incidence from the results of a 2002 Australian population survey of the incidence of non-melanoma skin cancer (NCCI 2003).

Cure rate and mean survival time

To estimate the cure rate and mean time to death for those not cured for each cancer we assume a Weibull distribution for the time from diagnosis to death and apply a non-linear model to the survival curves for each cancer (Verdecchia et al. 1998). We base the survival curves on all cases recorded in the Clearing House database with a diagnosis date between 1982 and 1997 which we follow-up for death until the end of 1999 (AIHW & AACR 2001).

We base the durations of the initial treatment, disseminated and terminal stages separately for each cancer, using Dutch study assumptions, peer-reviewed literature and input from local clinicians. For those not cured, we base duration of the remission stage as the total average time to death (estimated from the Weibull model) less the sum of the other stages. For those cured, we base the duration of the stage following initial treatment as 5 years less the duration of the initial treatment stage.

Again, breast cancer and non melanoma skin cancer are the two exceptions to this approach. Since the Clearing House database does not record tumour size, we base the survival times and cure rates on an analysis of breast cancer cases by tumour size published by the South

Australian Cancer Registry (South Australian Cancer Registry 2000). Because there are no national data on non melanoma skin cancer we estimate survival times and cure rates using assumptions modelled from published studies.

Long-term sequelae of cancer

The model for cancer in the previous Australian burden study assumed, with the exception of bone cancer, that cancer sufferers have no further burden following cancer cure. However, there are some cancers that are likely to have major sequelae causing long-term burden following successful treatment. The GBD study included long-term sequelae for colorectal cancer, breast cancer, female reproductive cancers and male genitourinary cancers. In addition, we include removal of one eye for eye cancer, removal of the larynx for larynx cancer, amputation for bone cancer and long-term brain injury for brain cancer. These sequelae and their associated severity weights are listed in the table below (Table A1.2).

We estimate cancer-related rates of amputation, stoma, mastectomy, larynx, eye removal and infertility from Australian hospital data. We estimate infertility rates from cancer-related hysterectomies and assume these only apply to survivors under 40 years of age. We derive impotence and incontinence rates from a review of the literature. Results published in the literature note the similarity between the effects of treatment for brain cancer and other forms of traumatic head injury, so we assume that the rates of long-term brain injury from brain cancers are the same as the equivalent rates for head injury.

We use the GBD disability weights for stoma, mastectomy, infertility, impotence and incontinence. For the disability associated with removal of an eye, amputation, and long-term brain injury we use comparable weights from the Australian study for long-term weight for an injury to an eye, major amputation and long-term effects of a brain injury in a non-fatal accident or injury, respectively. For removal of the larynx we assume that the Dutch weight for mild hearing loss, which is defined as ‘some difficulty in actively participating in a conversation with one or more persons’, is appropriate.

Table A1.2: Extra sequelae for cancer model

Site/sequelae	Proportion of survivors with sequelae (%)	Severity weight
Colorectal cancer—stoma	0.09	0.21
Bone & connective tissue—amputation	0.08	0.30
Breast cancer—mastectomy	0.51	0.09
Female reproductive cancer—infertility	Cervix: 0.46	0.18 (ages under 40)
	Uterus: 1.00	
	Ovary: 0.64	
Male genitourinary cancer—impotence and incontinence	Prostate: 0.53 Bladder: 0.12	0.20
Brain cancer—long-term brain injury	0.05	0.35
Eye cancer—removal of an eye	0.45	0.30
Larynx cancer—removal of the larynx	0.35	0.04

2G Other neoplasms

Benign neoplasms are not notifiable in Australia. As a result we base our incidence estimates for uterine myoma and benign brain tumour on Australian hospital data.

Specifically, for uterine myoma we use the numbers of myomectomies and hysterectomies for fibroids. We assume that surgical treatment is undertaken for all cases of rapidly growing or large tumours and myoma-related symptoms. We assume a six month pre-operative state equivalent to the GBD weight for chronic pelvic pain and an additional three-week post-operative state equivalent to laparotomy (derived weight of 0.349 for health state 222211). Based on expert advice, we assume reproductive disability occurs in 3% of hysterectomy cases to whom we apply the GBD weight for infertility. We assume the additional burden associated with menorrhagia in undiagnosed women is included in our YLD estimates for this condition under the 'other genitourinary' category.

Our model for benign brain tumour is based on the model for malignant brain tumours where we model the disease in stages for survivors (diagnosis and initial treatment, and post-curative treatment) and non-survivors (diagnosis and initial treatment, pre-terminal and terminal). We adjust our incidence estimates on the assumption that 20% of hospitalisations are readmissions (Jaaskelainen 1986; Simoca et al. 1994). We base our survival estimates on Australian mortality data and assume successfully treated cases recover normal efficiency (Steiner et al. 1998) with a period of 'worry' after treatment of 2 years. In the absence of specific disability weights, we use those for malignant brain tumours.

2H Diabetes

Diabetes cases

We estimate the incidence of insulin dependent diabetes mellitus (Type 1) from the National Diabetes Register (AIHW 2003b). We use DisMod to estimate prevalence and duration,

assuming no remission and age-specific risks of dying for all diabetes from the Asia Pacific Cohort Studies Collaboration – a meta-analysis of 24 cohort studies from Asia, Australia, and New Zealand that assessed the effects of diabetes on the risks of major cardiovascular disease and death (Woodward et al. 2003). We estimate the incidence of non-insulin dependent diabetes mellitus (Type 2) for ages less than 25 years from the National Diabetes Register. We estimate the incidence of Type 2 diabetes for ages 25 years and above by subtracting the prevalence of Type 1 diabetes from the total prevalence of diabetes from AusDiab and then deriving incidence and duration in DisMod including an annual trend for the period 1980–1999 for incidence of 2.5% for males and 1.5% for females (Dunstan et al. 2002). There is no direct measurement of the trend in incidence/prevalence of Type 2 diabetes in Australia. Instead, we analyse the historical trend in diabetes mortality (which is relatively ‘flat’) and assume that this reflects the net effect of an increase in incidence and a decrease in case-fatality which in turn we assume to be equivalent to the trend in cardiovascular disease case-fatality (as the main causes of death in people with diabetes are of cardiovascular origin). Thus, we also incorporate a 20 year trend for the case-fatality rate (–2% annual for males and –1% for females). We then project incidence and case-fatality forward to the year 2003 using the same trends as above and enter these into a DisMod model for total diabetes for 2003.

We subtract out those with diabetic nephropathy to avoid double-counting as the Dutch disability weight for diabetic nephropathy includes the disability associated with diabetes per se. We use the Dutch disability weight for an uncomplicated diabetes case (0.070).

Complications from diabetes for which we calculate YLD include retinopathy, cataract, glaucoma, renal failure, neuropathy, peripheral vascular disease, diabetic foot, amputations, ischaemic heart disease and stroke.

Retinopathy

We estimate the prevalence of mild and moderate vision loss from proliferative diabetic retinopathy in the Melbourne Visual Impairment Project (Weih et al. 2000). Experts confirmed that most retinopathy is treated before it leads to more serious vision loss. Therefore we estimate the incidence and duration of diabetic retinopathy in DisMod from the prevalence estimates from the Melbourne project, assuming no remission and twice the excess risk of mortality as for all diabetes. We base the proportion of cases due to Type 1 and Type 2 diabetes on the ratio of expected cases derived from modelling data on the progression of proliferative diabetic retinopathy from time of diagnosis (NHMRC 1997b; Tapp et al. 2003b). The Dutch disability weights for mild and moderate vision loss apply.

Cataract and glaucoma

We estimate the proportion of YLD from cataract and glaucoma attributable to Type 1 and Type 2 diabetes using population attributable fractions. We base the risks of cataract and glaucoma in diabetics from the Blue Mountain Eye Study (Mitchell et al. 1997) and use severity distributions from the Melbourne Visual Impairment Project (Weih et al. 2000).

Renal failure

We estimate the incidence of diabetes-related renal failure using 2002 data from the Australian dialysis and transplant data. We use DisMod to estimate the average duration for people on dialysis, assuming a case-fatality rate reflecting observed deaths from the register. We base our annual remission estimates on observed transplant data: 85% in Type 1 diabetes cases aged 0–85 years or over for males and females combined; 6% in Type 2 cases under 65 years for males and females combined; and 0% in Type 2 cases aged 65 years or over for males and females combined. We use the Dutch disability weight for diabetic nephropathy (0.29). We estimate YLD for transplant patients assuming a case-fatality ratio reflecting observed deaths from the register and 3% ‘remission’ due to graft failure (as these patients return back to the pool of dialysis cases). We assume a high disability weight (0.29) for the first 6 months following the transplant and a GBD weight of 0.11 thereafter.

Neuropathy

Tapp and colleagues provide estimates of diabetic neuropathy prevalence by time since diagnosis (2003a). We estimate by linear regression an annual increment in prevalence, which we then apply to survivors of incident cases of Type 1 and Type 2 diabetes by age as they progress to other age groups. Based on the Rochester Diabetic Neuropathy Study only 15% of Type 1 and 13% of Type 2 cases with diabetic neuropathy are symptomatic, of which 6% of Type 1 and 1% of Type 2 are severely affected (Dyck et al. 1993). The disability weight for Type 1 is 0.099 using the disability weight regression model (health state: 111111 – 85%; 222221 – 9%; and 222331 – 6%) and for Type 2 is 0.074 (using the corresponding percentages of 87%, 12% and 1%).

Peripheral vascular disease

Tapp and colleagues provide estimates of peripheral vascular disease incidence and prevalence (Tapp et al. 2003a). We assume that only those with claudication are symptomatic. We estimate by linear regression an annual increment in the prevalence of diabetes-related peripheral vascular disease in order to derive incidence, similar to the approach for diabetic retinopathy. In the absence of Dutch or GBD disability weights for this condition we derive a weight of 0.19 using the disability weight regression model. Remission from surgery by vascular grafts is assumed to be 20%.

Amputation and diabetic foot

We estimate the incidence of diabetes-related amputations from Australian hospital data. We use GBD disability weights for these conditions and base our durations and proportions treated on expert opinion. We use amputation rate data for diabetics with foot ulcers from the Diabetes Research Foundation (Yue & Molyneaux 2005). From 1994–2005 the amputation rate for diabetics with foot ulcers was 5.3%. We calculate an average duration of 8.9 months after fitting a log normal function to follow-up data on the duration of foot ulcers. As there is no Dutch disability weight, we apply the GBD weight of 0.113.

Ischaemic heart disease and stroke

We estimate the proportion of ischaemic heart disease YLD attributable to Type 1 and Type 2 diabetes using a population attributable fraction based on prevalence and the relative risk (2.0 and 2.5 for males and females respectively) of dying from ischaemic heart disease and stroke (2.0 for both males and females) amongst diabetics from the Asia Pacific Cohort Studies Collaboration (Woodward et al. 2003).

21 Endocrine and metabolic disorders

Haemolytic anaemia

We use Australian hospital data to estimate the incidence of hereditary haemolytic anaemia, assuming that annual admissions at age 0 years represent incidence. We model beta thalassaemia and 'other' haemolytic anaemia separately to account for different durations. We assume that the average duration for beta thalassaemia is 35 years based on a USA review (US Preventive Services Taskforce 1996) and we assume that the life expectancy of persons with 'other' haemolytic anaemia is the same for sickle cell anaemia, that is, around 25 years lower than the population average. In the absence of specific weights we use the GBD weight for very severe anaemia (0.25) and severe anaemia (0.09) for beta thalassaemia and other haemolytic anaemias respectively.

Other non-deficiency anaemia

We model the disability associated with aplastic anaemia and autoimmune anaemia. We base the prevalence of aplastic anaemia on Australian hospital data. We derive incidence and duration in DisMod using prevalence data, Australian mortality data where aplastic anaemia was an underlying condition and a remission of zero. We estimate the incidence of autoimmune anaemia using hospital data and assume that the average duration is 3 months. In the absence of a specific weight for other non-deficiency anaemias we use the GBD weight for very severe anaemia (0.25).

Cystic fibrosis

Massie and colleagues (2000) found the incidence of cystic fibrosis in Victoria over a 9-year period to be 3.5 per 10,000. This estimate is very similar to information from Queensland and Western Australia (Bower et al. 2004; Queensland Health 2004). We apply the Victorian estimate to the whole of Australia. We estimate the duration of cystic fibrosis in DisMod using the above incidence, no remission, and an age- and sex- specific risk of mortality from a patient-based USA study (Kulich et al. 2003). There is no disability weight for cystic fibrosis available. As obstructive lung disease is a major sequela, and the disease is progressive and fatal, we use the disability weight for severe chronic obstructive pulmonary disease (0.53).

Haemophilia

We base our estimate of the incidence of moderate and severe haemophilia on Australian data (Street & Ekert 1996). We do not model mild cases of haemophilia since we assume they have zero disability, as bleeding only occurs as a result of injury. We use the same assumptions about severity distribution, duration and disability weights as the previous Australian burden study.

2J Mental disorders

The 1997 National Survey of Mental Health and Wellbeing, including the child mental health and low prevalence disorder components, remains the only population-based data source for our estimates of most mental disorders (ABS 1998a; Jablensky et al. 1999; Sawyer et al. 2000).

Table A1.3 summarises the mental disorders for which we calculated YLD, along with the sources of data on which our incidence estimates are based.

Table A1.3: Sources of data for mental disorders

Data source	Mental disorder
National Survey of Mental Health and Wellbeing 1997	Depression & anxiety; bipolar disorder; most substance abuse (alcohol, sedative and cannabis drug dependence or abuse); and borderline personality disorder
Low Prevalence (Psychotic) Disorders Study	Psychotic disorders
Child and Adolescent Component of the National Survey of Mental Health and Wellbeing 1997 (Sawyer et al. 2000)	Childhood disorders (separation anxiety disorder, attention-deficit hyperactivity disorder)
Epidemiological Study—National Drug and Alcohol Research Centre Technical Report No. 198 (Degenhardt et al. 2004)	Heroin dependence
Alcohol and Other Drug Treatment Services National Minimum Data Set (AODTS-NMDS) collection < www.aihw.gov.au/drugs/datacubes/index.cfm > (accessed 15 December 2005)	Stimulant dependence
Reviews of epidemiological studies	Eating disorders (anorexia nervosa and bulimia nervosa), autism, and Asperger's syndrome

Depression & anxiety, substance abuse (excluding heroin and stimulant dependence), borderline personality disorder and bipolar disorder

While the data sources have remained mostly the same as were used for the previous Australian burden study, there are a number of key methodological changes. First, we have grouped all anxiety disorders (panic, agoraphobia, social phobia, generalised anxiety disorder, obsessive-compulsive disorder, post-traumatic stress disorder and separation anxiety disorder) and the unipolar depressive disorders (major depression and dysthymia) that were previously modelled separately into a single disease category. This is based on the argument that the high degree of comorbidity and the similarity in psychological and drug treatment means that all these disorders can be considered as part of the same entity, with a continuum between mostly depressed to mostly anxious (for example (Andrews et al. 1990;

Andrews & Slade 2002). The advantage of this approach is that it takes away some of the difficulties of dealing with the frequent comorbidity among these disorders.

Second, disability weights for all conditions derived from the national mental health survey continue to be based on the mental component score of the SF-12 but for this update we calculate a per unit change in disability weight for each unit change in the mental component score and apply this to all disorders. Dutch disability weights exist for mild, moderate and severe depression as well as for six different anxiety disorders for a combined mild-moderate state and a severe state. Assuming that mild, moderate and severe are 1, 2 and 3 standard deviations, respectively, below the population mean of the mental component score we sought a mathematical function that best describes the range of disability weights. A second-order polynomial function gave the best fit. This transformation of categorical weights into a continuous scale allows us to calculate a disability weight for each respondent in the survey. Any mental component score value greater than the population mean of 52 is set to 0 and the weight for a mental component score of 20 is taken as the highest disability weight even if the mental component score is lower (this is done because otherwise the lowest mental component scores would correspond with a disability weight of greater than 1).

Third, to deal with comorbidity, we apportion the disability weights calculated in the National mental health survey equally between the comorbid mental health diagnoses for each individual. In the previous Australian burden study we did the correction for comorbidity at the level of the number of people affected and hence reported lower than actual numbers of incident and prevalent cases.

Our general model for these conditions derives incidence figures from the National mental health survey prevalence figures, using DisMod and assuming appropriate remission rates and relative risks of mortality from a meta-analysis (Harris & Barraclough 1998). We use the proportion of one-year prevalent cases reporting symptoms in the previous two weeks as an approximation of the proportion of time with symptoms and thus assume that all these conditions have a chronic nature with periods of remission in between.

For children aged 5–17 years, we use prevalence estimates for depression and anxiety from the Child and Adolescent Component of the national mental health survey (Sawyer et al. 2000). In DisMod we use a remission rate of 0.043, a pooled estimate from follow-up studies of people with various anxiety disorders (Steketee et al. 1999; Wewetzer et al. 2001; Yonkers et al. 2003) and an increased relative risk of mortality of 1.5, a value in between the range of meta-analysis estimates reported for anxiety and depressive disorders (Harris & Barraclough 1998).

The prevalence estimates for bipolar disorder in the previous Australian burden study were based on the international literature. This was because the prevalence figures from the National mental health survey were considered inaccurate due to a technical problem during the conduct of the survey. Subsequently Mitchell and colleagues (2004) have re-analysed the data and defined the prevalence of 'euphoric hypomanic/manic syndrome'. They argue that with this definition around 95% of cases of bipolar disorder are captured. For the current estimates we use the same definition and adjust the 12-month prevalence by 100/95. In DisMod we use a remission rate of 0.035 calculated from a follow-up study (Angst & Preisig 1995) and an increased relative risk of mortality of 1.96 in men and 1.76 in women (Harris & Barraclough 1998).

In this study we include all personality disorders – rather than borderline personality disorder only – but limit our estimates to those without any comorbid mental disorders. The

proportion of comorbidity between personality disorders and other mental disorders is so high that we argue that in most cases it ought to be seen as a risk factor rather than a separate condition. However, in order to capture all disability from mental disorders we include a category 'isolated personality disorder'. The remission estimate of 17% is consistent between two follow-up studies (Grilo et al. 2004; Zanarini et al. 2003). The relative risk of mortality is 1.84 (Harris & Barraclough 1998).

In the previous Australian burden study, estimates for alcohol use disorder were made separately for alcohol dependence and harmful alcohol use, and then presented as one disease category. In the current update we combine the two categories and create one DisMod model based on 12 month prevalence of any alcohol use disorder in the National mental health survey. The two other parameters in DisMod are a remission rate of 23.7% calculated from a two-year follow-up study (Booth et al. 2001), and an elevated mortality risk of 1.8 in males and 3.84 in females (Harris & Barraclough 1998).

For cannabis dependence, we assume a remission of 8% (Swift et al. 2000) and no excess risk of mortality. There are no follow-up studies of people with sedative dependence. We use the same remission as in the cannabis model and apply an excess mortality risk of 2.1 reported for 'legal' drug use (Harris & Barraclough 1998).

Heroin dependence and harmful use

Household surveys are likely to underestimate the true prevalence of heroin use (differential response between users and non-users and a greater proportion of users not living in households). Instead, we use higher estimates of regular heroin users based on triangulation between five data sources: ABS opioid deaths, ambulance attendances for drug overdose in New South Wales, New South Wales Health heroin pharmacotherapy client database, New South Wales data on arrests for drug offences, and data from the Alcohol and Drug Information Service on calls related to heroin use (Degenhardt et al. 2004). While the detailed comparison of databases was done for New South Wales, extrapolations were made for all jurisdictions by extrapolation of relationship between numbers under treatment or in contact with police and opioid mortality figures from New South Wales and the opioid deaths in each jurisdiction.

In the previous Australian burden study, we assumed very high remission after age 45 years to reflect the low prevalence of heroin use. However, expert advice that this is a cohort effect rather than a high remission effect explains the drop in prevalence at older ages. In current estimates we 'allow' DisMod to build up prevalence figures at older ages.

Back projection methods by the National Drug and Alcohol Research Centre assumes a risk of dying from overdose of 0.8% per year (Law et al. 2001). We assume a case-fatality rate of 1% to account for raised mortality from other causes. The overall relative risk calculated in DisMod is of the same order of magnitude as reported elsewhere (AIHW: Ridolfo & Stevenson 2001; Darke & Ross 2002). The disability weight for heroin dependence of 0.27 was derived by Victorian mental health experts for the previous Australian burden study and is close to the GBD disability weight estimate of 0.252.

Stimulant dependence

We decided to use treatment figures rather than the estimates of prevalence of stimulant dependence from the National mental health survey as there has been a marked increase in

the use of stimulants since 1997 and the survey results show an erratic age pattern as only few cases were identified. Instead we estimate the prevalence of stimulant dependence from the number of closed treatment episodes in 2002–2003 where the principal drug of concern was listed as amphetamines (Alcohol and Other Drug Treatment Services National Minimum Data Set) collection. We inflate these figures by 5.5 as described by McKetin and colleagues (2005).

We estimate remission by first entering prevalence, a relative risk of 0 and a case-fatality rate of 0, into DisMod. We thus get DisMod to produce an estimate of remission that best replicates the age pattern of prevalence. The average remission across all ages was 12%. We then run the DisMod model again with same prevalence, this remission rate and a relative risk of 2.1 for excess mortality as reported for 'legal drug use' (Harris & Barraclough 1998).

We derive a disability weight for stimulant dependence as we have done for all other conditions in the National mental health survey and thus assume that the same average severity found among the lower number of cases with stimulant dependence in the survey reflects that of all cases in the population.

Psychotic disorders

Estimates for psychotic disorders are based on prevalence from the Low Prevalence (Psychotic) Disorders Study conducted in Australia in 1997 as part of the National Survey of Mental Health and Wellbeing. This survey measured an overall estimate of 4.7 per 1,000 population. The low prevalence study suffered from a low response rate by general practitioners contacted in the study areas and therefore under-represented people with psychotic disorders who are solely managed by their general practitioner (Lewin & Carr 1998). Before conducting further analysis, we adjust upwards to one in three the number of people in the survey who are wholly treated by a general practitioner and adjust downwards by a factor of 0.841 to reflect only those with schizophrenia and related diagnoses and not those with a diagnosis of bipolar or affective psychosis. Annual remission is based on a number of longer term studies and is set at the median of the reported rates (1.5%) (Ciompi 1980; Harding et al. 1987; Harrison et al. 2001; Helgason 1990; Huber et al. 1980). We derive incidence and duration figures from DisMod using a 54% higher risk of mortality overall for people with schizophrenia (Harris & Barraclough 1998), with an age pattern imposed by the relative frequency by age that schizophrenia is mentioned in death records. The DisMod incidence output indicates that almost all psychotic disorders have their beginning in late adolescence or early adulthood, with a small second peak in post-menopausal women. We assume that the average time spent in psychosis is 30% (Leff et al 1992). We use a composite weight based on 30% of the GBD weight for psychosis corresponding to the estimated time spent in this state and 70% of the treated weight ($0.3 \times 0.627 + 0.7 \times 0.351 = 0.434$). The low prevalence study reported a higher proportion (61%) of people with a psychotic disorder having current delusions or hallucinations. It also stated that 86% are taking prescribed medication and that 83% of the total reported that their psychotic symptoms respond to pharmacological treatment. The first finding would indicate that our composite disability weight is too low but the second finding would support a lower weight. For the Assessing Cost-Effectiveness (ACE)–Mental Health study, disability weights for each individual in the low prevalence study were estimated using a sliding scale between the highest and lowest of Dutch disability weights for schizophrenia and anchoring individuals on this scale based on their score on the diagnostic interview for psychosis disability module that was included in the survey (Haby et al. 2004). The mean disability weight across the sample using this

method is 0.39. We decided to continue to use the 0.434 disability weight as in the previous Australian burden study.

Eating disorders

Estimates for bulimia are based on a prevalence rate of 0.7% among Swiss 14–17 year old females (Steinhausen et al. 1997). This is the mid-point in the range of prevalence between 0.5% and 1% reported from more rigorous epidemiological studies (Gilchrist et al. 1998). We calculate a remission rate of 0.21 from figures reported in a review of follow-up studies (Keel et al. 1999). We derive incidence and duration estimates for women from these figures using DisMod, assuming the age at onset is between 14 and 29 years with no increased risk of mortality. Estimates for anorexia are based on a 0.5% prevalence among females older than 15 years (Gilchrist et al. 1998; Keel et al. 1999) and a remission rate of 0.11 calculated from a follow-up study (Strober et al. 1997). We use DisMod to derive incidence and duration estimates for women from these figures, assuming the age at onset is between 14 and 29 years with an increased annual risk of mortality of 0.59% (Sullivan 1995). We assume the incidence in males is 10% of the rate in females. We use the Dutch weight of 0.28 for both types of eating disorder.

Childhood disorders

Australian prevalence data for childhood attention deficit with hyperactivity disorder come from the Child and Adolescent Component of the 1997 National Survey of Mental Health and Wellbeing (Sawyer et al. 2000). We define attention deficit with hyperactivity disorder to include children with a diagnosis on the survey and whose parents report the child having more emotional or behavioural problems than have other children of the same age. The estimates of burden of attention deficit with hyperactivity disorder were derived from prevalence rates of 6% in male children, 3% in female children, 2% in male adolescents and 1% in female adolescents. Our incidence figures were derived from DisMod, assuming an age at onset of 3–6 years and a remission rate of 0.15 (Hill & Schoener 1996). To reproduce the prevalence pattern we use a higher remission rate of 0.25 in adolescents aged 10–19 years and 0.3 thereafter. We assume no increased risk of mortality. We use the Dutch weights for both mild and moderate-to-severe attention deficit with hyperactivity disorder (0.02 and 0.15), and weight these by the severity distribution found in the 1997 survey to derive a composite disability weight.

Autism is part of pervasive developmental disorders; the other important condition in that category is Asperger's syndrome, which was described at about the same time as autism. Autism is characterised by the triad of language or communication impairment, social impairment and behavioural impairment (obsessions, rituals). However, Asperger's syndrome has only the latter two components and is not associated with intellectual disability, as is the case with 80% of autistic children. Behavioural problems are a predominant feature in children with Asperger's syndrome.

We derive the incidence of autism and Asperger's syndrome from an Australian study with data from treatment and educational support services in Western Australia and New South Wales. We assume no remission and an elevated risk of mortality as reported by Shavelle and colleagues (2001). We use the average duration of mild intellectual disability and the Dutch disability weight of 0.55 for autism, and for Asperger's syndrome an estimated weight

of 0.25 based on expert advice that the condition is worse than moderate to severe attention deficit with hyperactivity disorder but much less severe than autism.

2K Nervous system and sense organ disorders

Dementia

A door-to-door population-based two-phase investigation method (screening followed by detailed neurological examination by a psychiatrist) is the most accurate epidemiologic approach to estimate the epidemiology of dementia and Parkinson's disease (Benito-Leon et al. 2004).

We base our estimates of the prevalence of dementia for people aged 65 years or over on a recent European meta-analysis of population-based door-to-door studies conducted by the Neurologic Diseases in the Elderly Research Group (Lobo et al. 2000). We proportionately redistribute the one-third of cases that constitute 'other or mixed type' to Alzheimer's and vascular dementia. We estimate the prevalence of dementia below the age of 65 years from a recent UK study of patients aged 30–64 years (Harvey et al. 2003).

We use relative risks of mortality for Alzheimer's disease and vascular dementia from a survival study of incident cases that controlled for comorbidity (Aguero-Torres et al. 1999). The estimated mortality risk for all dementia from this is comparable to the results of the meta-analyses of dementia prevalent cases and survival (Dewey & Saz 2001; Jagger et al. 2000). We prefer using the former because it provides type-specific survival data.

We derive incidence and duration using DisMod, based on the aforementioned representative population-based studies of prevalence, assuming no remission and relative risks from the incident-based survival study. This model gives average durations across all ages for both sexes of around 4 years which was in keeping with the literature on the survival of prevalent cases (Aguero-Torres et al. 1999; Helmer et al. 2001). We model dementia as a progressive illness and discount the latter stages back to incidence of disease. We use the disability weights derived by the previous Australian burden study (which combined the Dutch weights with a severity distribution from a European population-based cohort study).

Epilepsy

We base our incidence estimates for primary epilepsy on the 1980–84 Rochester Epidemiology Project medical record linkage system (Zarrelli et al. 1999). We use these incidence estimates, assuming no differentials by sex, with age-specific remissions (Annegers et al. 1979) and an overall standardised mortality ratio of 1.3 (Tomson 2000) to derive estimates of incidence and duration using DisMod. We use the Dutch disability weight for epilepsy (0.110).

Parkinson's disease

We only explicitly model primary Parkinson's disease (ICD-10 code G20). We assume that secondary Parkinsonism is accounted for under other relevant disease categories.

We base our estimates of the prevalence of Parkinson's disease from a recent European meta-analysis of population-based door-to-door studies conducted by the Neurologic Diseases in the Elderly Research Group (de Rijk et al. 2000). There are no sex differences in the prevalence of Parkinson's disease. We do not use the Australian studies on the prevalence of Parkinson's disease since we consider them to be outliers; they give prevalence estimates two to three times higher than most of the literature (Chan et al. 2001, 2005).

We base our relative risk of mortality on the meta-analysis of prevalent cases of Parkinson's disease and survival undertaken by the Neurologic Diseases in the Elderly Research Group (Berger et al. 2000). We plot and fit the risk of mortality by age using an exponential trendline to smooth the irregular pattern by age.

We derive incidence and duration using DisMod assuming no remission and a relative risk of mortality for males and females of 3.1 and 1.8 respectively, resulting in average durations of 4.5 and 9.8 years. These durations are broadly consistent with durations reported in the literature (Elbaz et al. 2003; Fall et al. 2003; Herlofson et al. 2004; Hughes et al. 2004; Morgante et al. 2000).

We derive disability weights from an Australian patient-based cohort study (Hely et al. 1999) reporting on the distribution of Hoehn and Yahr stages (which corresponds with the descriptions of the severity states of Parkinson's disease for which Dutch disability weights are available) and survival at each 2-year interval. We model Parkinson's disease assuming that all cases start with mild symptoms and progress over time to moderate and then severe symptoms over time. From simple linear regression lines we derive an annual increase in those with moderate and severe symptoms. The proportion of cases over time who are in the moderate category is the balance between those moving from mild to moderate and those exiting moderate by shifting to the severe category. For each age group we calculate the average disability weight during the estimated average duration. As severity progresses with time since incidence and younger age groups have longer durations, disability weights are highest in the younger age groups.

Motor neurone disease

We base our incidence estimates for motor neurone disease on Australian mortality data. We assume that incident cases equal annual deaths due to motor neurone disease. Our estimates for males and females are consistent with international literature for males (Chancellor et al. 1993). We assume average durations of 2.9 years for those aged 0–64 years and 1.9 years for people aged 65 years or over. We base our duration assumptions on Australian and international literature (Forbes et al. 2004; Sach 1995). In the absence of a specific disability weight we use the Dutch weight for progressive multiple sclerosis (0.67).

Multiple sclerosis

We estimate the prevalence of multiple sclerosis using 1981 and 1996 estimates of multiple sclerosis for some Australian states and territories (Barnett et al. 2003; Simmons et al. 2001) with extrapolations based on latitudinal differences for jurisdictions with no estimates. We assume that changes over time represent improvements in identification rather than changes in epidemiology. We derive incidence and duration using DisMod assuming no remission and age and sex specific case-fatality rates based on a 25-year New Zealand cohort study (Miller et al. 1992).

In 10.8% of patients the disease has a progressive course from the onset (Roxburgh et al. 2005). The median time it takes to reach an Expanded Disability Status Scale score of 6 (equivalent to having to use a cane) in those with a relapsing-remitting course is 30 years (Tremlett et al. 2006). We use the Dutch weights for relapsing-remitting (0.33) and progressive (0.67) phases and assume that those with relapsing-remitting disease have 30 years at the lower disability weight and the remainder at the higher disability weight level.

Huntington's chorea

Huntington's chorea is modelled in DisMod using prevalence from the literature (McCusker et al. 2000), assuming no remission and mortality data. We assume a duration of 20 years for the younger age groups and apply the durations from DisMod for ages 65 years or over. Assuming similar progression of disease as in Parkinson's, we adopt the weights for the three stages of this disease.

Muscular dystrophy

For muscular dystrophy in males, we use the average incidence rates from New South Wales, Victoria, Queensland, Western Australia and the Australian Capital Territory (Cowan et al. 1980; Emery 1991). The incidence for females is calculated by applying the sex ratio from mortality data. In the absence of specific weights for this condition, we assume the initial symptomatic phase is similar to the initial stage of Parkinson's disease, the phase in which walking becomes impossible is similar to that of paraplegia, and the final stage is equivalent to quadriplegia.

Vision loss

Our incidence estimates for vision loss are based on the results of the Melbourne Visual Impairment Project, which assessed visual acuity and the prevalence by cause of mild, moderate and severe visual impairment in a sample representative of Victorians (Weih et al. 2000). For glaucoma, refraction errors, macular degeneration and the category 'other vision loss', we derive incidence and duration of related visual impairment using DisMod, assuming no remission and a relative mortality risk of 1. For glaucoma we use Dutch disability weights for mild, moderate and severe vision loss to derive a composite disability weight from the severity pattern across all ages (as the age-specific data are based on small numbers). For macular degeneration, refraction errors and 'other vision loss' we derive age-specific disability weights.

We estimate the incidence of mild and moderate cataract-related vision impairment using Australian hospital data assuming that 50% of surgically corrected cases had vision loss in both eyes prior to operation for 1 year on average and that 90% of cases are mild and 10% are moderate. We estimate the prevalence of un-operated cataracts as the difference between the prevalence of cataract-related visual impairment estimated by the Melbourne study and the number of surgical corrections. This leads to a small estimate of un-operated cataracts in the elderly over 80 years of age. We use this to estimate the incidence of un-operated cases of cataract-related severe vision loss in DisMod, assuming no remission and a relative risk of 1.5. For cataract-related vision loss at ages 0-14 years we assume duration of 2 years and for ages 15 years or over we assume a 1-year duration. Incident cases of un-operated cataract were assumed to be prevalent cases waiting on average 1 year for cataract surgery. We use

Dutch disability weights for mild and moderate cataract-related vision loss. For severe cataract-related vision loss we estimate a combined disability weight using the Dutch weights for each of the stages along with prevalence data from the Melbourne study to derive combined stages age-specific disability weights. The proportion of glaucoma and cataract-related vision loss attributable to diabetes is then determined from relative risks from the Blue Mountain Eye Study (Mitchell et al. 1997) and only non-diabetes-related vision loss is included in the YLD estimates for these categories.

Hearing loss

We model hearing loss as a progressive condition with mild (25–34 dB and 35–44 dB), moderate and severe stages so that prevalent cases with moderate or severe impairment are regarded as incident cases of mild impairment at an earlier age. We use survey prevalence data from South Australia (Wilson et al. 1999), initially modelling the prevalence of severe hearing loss, no remission and a relative risk of 1 in DisMod. We use incidence of severe hearing loss from the DisMod output as ‘mortality’ in the moderate hearing loss DisMod model; this takes the cases of severe hearing loss out of the pool of susceptible cases for moderate hearing loss and hence gives more accurate average durations than if remission were used as remitted cases in the DisMod model, as the cases continue to be subject to the hazard of incidence. Similarly, we use incidence of moderate hearing loss as ‘mortality’ in mild hearing loss (35–44 dB) and incidence of mild hearing loss (35–44 dB) as ‘mortality’ in mild hearing loss (25–34 dB). From examination of the prevalence data by level of severity and age, and assuming that all cases progress from the mildest to most severe category, it seems reasonable to assume that on average progression to the next severity level occurs at 5 year intervals between mild (25–34 dB) and mild (35–44 dB), and at 10 year intervals from mild (35–44 dB) to moderate and moderate to severe. From the cross-sectional data on prevalence it is not possible to estimate these progression times exactly. However, to be consistent with other disease models where subsequent severity levels for the same health state are discounted back to first incidence, we apply a 25-year lag for severe hearing loss, 15 years for moderate and 5 years for the mild (35–44 dB) categories. Dutch weights of 0.04, 0.12 and 0.37 apply for mild (35–44 dB), moderate and severe hearing loss, respectively. For the mild (25–34 dB) category we assume a disability weight of 0.02, half that of the mild (35–44dB) category.

Intellectual disability

Intellectual disability is categorised into the following levels: mild, moderate, severe and profound, with intelligence quotient (IQ) ranges of 50–69, 35–49, 20–34, <20 respectively. This categorisation is based on the Dutch disability weight criteria.

We estimate the incidence of mild-moderate and severe intellectual disability using the Intellectual Disability Exploring Answers Database, a Western Australian population-based dataset of children with intellectual disability identified through disability and educational services between 1983–1996. We adjust the severity distribution of incidence data to account for unspecified cases and redistribute cases so that the severity level as defined by IQ is comparable to the Dutch disability weight criteria. Then we extrapolate incident cases by the two severity levels (mild-moderate and severe) to four levels of severity (mild, moderate, severe and profound) using the average severity distribution from two Australian studies (Einfeld & Tonge 1996; Wellesley et al. 1992). We assume that because neither study

recruited cases from school services, mild cases were underestimated and base our estimation of mild cases on the balance of the mild-moderate category. This gives the following proportionate distribution of incident cases by severity: mild (76%), moderate (14%), severe (7%) and profound (3%).

In order to derive plausible durations of intellectual disability by the four stages of severity we calculate the proportional difference in life expectancy by level of severity of intellectual disability in comparison to the life expectancy of the general population from a 35-year Finnish follow-up study (Patja et al. 2000).

We model the incidence and duration of intellectual disability in DisMod assuming that 90% of intellectual disability, based on Australian population data, occurs in the first year of life and the remaining 10% occurs in the 1–4 age group, no remission, and a relative risk of mortality that gives an average duration by severity level based on the extrapolation of Finnish data to the 2003 Australian life table.

We do not include the YLD for intellectual disability as a discrete category in the main listings of this burden study. Instead, incident cases of intellectual disability are attributed to underlying causes (such as congenital disorders, epilepsy, autism, perinatal conditions, meningitis, brain tumours and cerebral palsy) using findings from the Australian Child to Adult Development Study, a longitudinal study of behavioural and emotional problems in 429 young people with intellectual disabilities. We use data from two publications of this study to produce the proportionate distribution of the underlying cause of intellectual disability by severity level and sex (Mowat et al. unpublished; Partington et al. 2000). We calculate YLD for each underlying cause using incidence and duration derived from DisMod and the Dutch disability weights for mild, moderate, severe, and profound intellectual disability.

Migraine

We base our prevalence estimates for migraine on the National Health Survey data and our incidence estimates for migraine on international data (Stewart et al. 1991). We estimate the incidence and duration of migraine in DisMod using prevalence, incidence, and a case-fatality rate of zero. Within DisMod, we use manual smoothing to extrapolate incidence to older ages. We assume that 20% of cases receive treatment in developed countries. We assume that the average duration for untreated and treated episodes is 24 hours and 6 hours respectively. We derive average disability weights for untreated and treated models using frequency, severity, and disability weight data from *Global burden of migraine in the year 2000* (Leonardi & Mathers 2003).

2L Cardiovascular disease

Ischaemic heart disease

Three health states are modelled separately for ischaemic heart disease: angina pectoris, acute myocardial infarction and heart failure. We model the incidence of angina pectoris as the number of admissions to hospital without any mention of angina in any previous admission in 15 years of linked hospital records in Western Australia (Department of Health of Western Australia et al. 2005; Holman et al. 1999) and adjust by the ratio of admissions for

angina pectoris between Western Australia and the whole country. We model angina pectoris pre- and post-myocardial infarct together. The duration is determined in DisMod, assuming remission estimated from the number of revascularisation procedures from Australian hospital data and age- and sex-specific case-fatality rates calculated over the period 1998–2003 in 'prevalent cases' of angina pectoris (that is, anyone with an admission for angina pectoris since 1988 and still alive over the follow-up period).

Assuming that about half of the declining ischaemic heart disease mortality reflects change in the case-fatality rate rather than incidence (Unal et al. 2004), we apply half of the ischaemic heart disease mortality trend observed over the period 1979–2003 in DisMod to incidence and the other half to case-fatality.

We assume 95% of angina is experienced at the mild-moderate level with the corresponding Dutch disability weight of 0.08, and the remaining 5% with a weight of 0.57.

For people discharged alive following acute myocardial infarction in 2003, we calculate a period of 3 months of disability at the GBD treated disability weight of 0.395.

Heart diseases resulting in heart failure

Population-level prevalence or incidence information on heart failure is absent in Australia and scarce elsewhere. In 2001, by extrapolation from US studies a rough estimate was made of about 300,000 prevalent cases of heart failure in Australia (Krum 2001). Complicating factors in the estimation of heart failure prevalence are that estimates from other countries and different time periods may not apply to the current Australian situation. Ischaemic heart disease is the underlying cause of heart failure in the majority of cases, and there has been a steady decline in the risk of ischaemic heart disease since the early 1970s combined with improved survival due to improvements in therapeutic options. The first would cause a reduction in prevalence, while the latter would lead to higher prevalence. It is not clear what the net effect of these two influences would be on the prevalence of heart failure.

Using hospital data is also not straightforward as the current wisdom is that there has been a change in the case load of people presenting to tertiary health facilities with this condition, following the wider use of improved pharmacological treatment combinations since the 1990s, resulting in a greater proportion of cases being successfully treated in primary care. Nevertheless, our model for heart failure starts with a description of the epidemiology of hospitalised heart failure, for which we have extensive information from Western Australia. From the linked data set of all hospitalisations and deaths in this state, we identify people who presented to hospital with heart failure (either as a primary diagnosis or as an associated condition) at any time in the period 1990–2003. To derive case-fatality, we calculate the number of years lived between 1998 and 2003 by anyone who had ever been admitted with a diagnosis of heart failure since 1990. The case-fatality rate was then taken as the number of deaths over person-years of follow-up in 5-year age groups after subtracting out the background mortality.

The complete descriptive epidemiology in this group is derived in DisMod from incidence and case-fatality, the third parameter being zero remission (that is, people do not recover from heart failure). We include in this model a declining trend in case-fatality over the last 10 years of 3% per year for males and 1% per year for females (derived from our survival model), and a 2% decline in incidence per year for both males and females over the last 35 years. This latter figure is half the annual decline we observe for ischaemic heart disease mortality over this period, ischaemic heart disease being the major driver of heart failure

risk. The other half of the decline in ischaemic heart disease mortality we assume to be due to improvements in case-fatality (see above) (Unal et al. 2004).

There is little information on the incidence of heart failure in the community (that is, not yet diagnosed cases and those diagnosed but treated in the primary care setting without requiring hospitalisation). We assume that this group has less severe disease with better survival compared to their hospitalised counterparts. We also assume that when they die, it is less likely that heart failure will be mentioned as the underlying cause of death. We have data on the number of hospitalised cases of heart failure who died with heart failure as the underlying cause of death and we know the overall number of deaths coded to heart failure. Assuming that the linkage of hospital and death records in Western Australia is complete, we then assume that the balance of heart failure coded deaths occur in never-hospitalised cases of heart failure. In the absence of data to characterise the never-hospitalised cases of heart failure we make two assumptions. First, to account for lower severity we assume that their case-fatality rate is lower by 25%. Second, we assume that deaths in non-hospitalised heart failure cases are 25% less likely to be coded to heart failure.

Among hospitalised cases that die, the probability of receiving an underlying cause of death code of heart failure (428 in ICD-9 and I58 in ICD-10) is 3.6% in males and 5.3% in females. If non-hospitalised cases are 25% less likely to be assigned a code of heart failure the percentage of total excess deaths coded to heart failure would be 2.7% in males and 3.9% in females. From this we can derive the total number of deaths due to heart failure in non-hospitalised cases (3,199 in males and 3,958 in females over the period 2001–2003). By adding in the 3,833 deaths from ever-hospitalised cases of heart failure in males and 4,186 in females, we can calculate the average population mortality rate of heart failure over the period. These rates (calculated by age and sex) are the inputs to a second iteration of DisMod, together with zero remission and the case-fatality rate of the first DisMod model of hospitalised heart failure cases adjusted downwards to reflect the proportion of never-hospitalised cases having 25% lower case-fatality. We continue to use the same assumptions on trends in case-fatality and incidence as in the first model. The output of the second DisMod iteration then gives us the incidence, prevalence and average durations for all heart failure, which feed into our YLD calculations. The total prevalence of heart failure in Australia in 2003 is thus estimated to be 220,000 cases.

We then identify the underlying causes for all heart failure cases – rheumatic heart disease, hypertensive heart disease, ischaemic heart disease, pulmonary heart disease, inflammatory heart disease, non-rheumatic valvular heart disease – in the Victorian linked hospital admission dataset between 1996 and 2002, if any of these were mentioned as a cause in the six years of hospital admission data. We then adjust the proportions, by age and sex, of all underlying causes so they add up to 100% to account for cases with none or more than one underlying cause identified. We use the duration, together with the incidence and prevalence estimates initially obtained from the heart failure model described above, multiplied by the proportion of heart failure cases for each of the above six underlying causes, to calculate the YLD for each of these conditions (including ischaemic heart disease).

Stroke

We model stroke in terms of the following health states: a short period of disability for those who die in the first 28 days, survival beyond 28 days with no permanent impairment at one year after onset, and survival beyond 28 days with permanent impairment. Admissions for stroke in the year 2003 are the starting point for our estimate of incidence. To get an

approximation of first-ever stroke incidence we take the ratio of hospital admission figures from the North-East Melbourne Stroke Incidence Study (NEMESIS) area during the time of the study to the reported NEMESIS first-ever incidence figures (Thrift et al. 2000) and apply this ratio to 2003 Australian hospital admissions for stroke. Next, we subtract a proportion of cases that die, using a 28-day case-fatality rate by stroke subtype as reported by Thrift and colleagues.

The case-fatality rate of stroke comes from the Western Australian linked database using a similar approach to that described above for heart failure. The case-fatality rate for DisMod is the excess mortality in prevalent cases, defined in our analyses as anyone still alive at the beginning of the follow-up period (mid 1998–mid 2004) with a mention of stroke during any admission between 1989 and 1998 as well as any new cases of admitted stroke during follow-up. Follow-up time and numbers of deaths were analysed for each 5-year age group and the overall case-fatality rate reduced by the relevant background mortality.

Analyses by Judy Katzenellenbogen in Western Australia for her PhD indicate that after the first 28 days the case-fatality rate does not vary significantly by type of stroke and that the DisMod assumption of a case-fatality hazard that varies with age but not with time since stroke is plausible.

Disability weights are derived from one-year follow-up data of stroke survivors in the Perth Community Stroke Study analysed by Judy Katzenellenbogen to compare health status information before and at 4 months and 1 year after the stroke event.

Other cardiovascular disease

Heart failure is the main disability from rheumatic heart disease, non-rheumatic valvular disease, hypertensive heart disease and the group of inflammatory heart diseases (including myocarditis, cardiomyopathy, endocarditis and pericarditis). The proportions of heart failure cases for each of these causes are derived as described above for all heart failure. For rheumatic heart disease and non-rheumatic heart disease we do a separate DisMod model based on heart failure prevalence for these causes and taking into account the remission through surgical interventions using Australian hospital data.

For aortic aneurysm, we assume the hospitalisation rate reflects incidence. For peripheral vascular disease, we assume the hospitalisation rate reflects prevalence at all ages. We derive the incidence from DisMod, assuming a relative risk of 2 and a remission rate of 0.1, which approximates the number of surgical interventions as a proportion of total prevalent cases.

For aortic aneurysm, we assume a one-month period of disability during treatment and no residual disability for those who survive treatment. Without a disability weight for this health state, we use the derived weight for laparotomy (0.349). For peripheral vascular disease, we use derived weights of 0.243 and 0.257 for men and women respectively, based on severity distributions from the 1993 Australian disability survey. Weights for amputations are from the GBD study.

2M Chronic respiratory diseases

Chronic obstructive pulmonary disease

We estimate the prevalence of chronic obstructive pulmonary disease for cases with a forced expiratory volume in one second of less than 70% of predicted (excluding those with a doctor defined diagnosis of asthma) using the 1994–95 Busselton Study (Knuiman et al. 1999). While this study sample comprises a selected rural population in Western Australia, we assume the data are representative of prevalence in all areas of Australia. We use DisMod to estimate the incidence of chronic obstructive pulmonary disease in 1994, assuming no remission and a relative risk equivalent to that calculated from death rates attributed to smoking (see section on risk factors). We include a trend of -2% per year for males and 3% per year for females based on trends in chronic obstructive pulmonary disease mortality since 1979. We calculate 2003 incidence estimates by applying age- and sex-specific trends (based on mortality) to the 1994 incidence estimates. We then use DisMod with the same assumptions about remission and relative risk of mortality to model prevalence, age of onset and duration for 2003. We derive a composite average disability weight for males (0.168) and females (0.159) using the Dutch weights for mild, moderate and severe chronic obstructive pulmonary disease and the proportionate distribution by level of severity of dyspnoea from the Busselton Study. We add the proportion of heart failure cases attributed to ‘pulmonary heart disease’ on the basis that chronic lung disease is the underlying cause.

Asthma

We estimate the prevalence of asthma for cases that have a positive airway hyper-responsiveness test and wheezing in the last 12 months from the literature (Bauman et al. 1992a; Peat et al. 1992, 1994, 1995; Toelle et al. 2004). Although these two criteria may underestimate the ‘true’ prevalence of asthma, a reliance on self-reported wheeze alone overestimates figures by up to a third (Toelle et al. 1992; Van Asperen 1995). We estimate the prevalence of asthma in children aged 1–2 years to be 5.75%, using a report of ‘wheeze’ from the US (Martinez et al. 1995) which we adjust by 42% to obtain an estimate that reflects those with wheeze having asthma (Peat et al. 1994, 1995; Toelle et al. 2004). We estimate the prevalence of asthma to be 12.3% in boys and 8.8% in girls aged 3–18 years, using an average of 3 studies from 1992 to 2002 (Peat et al. 1994, 1995; Toelle et al. 2004) and a male-to-female ratio of 1.4:1 (Gergen et al. 1988). For adults, we average the prevalence data from the early 1990 studies (Bauman et al. 1992a; Peat et al. 1992, 1994, 1995) since these were the last studies in adults to have used a positive airway hyper-responsiveness test and assume no change in the prevalence of asthma over time based on the literature and the observed trend in children. We use a male-to-female ratio of 1:1.5 (DHS 2002) to give an estimated 2003 asthma prevalence of 5% and 7.5% in male and female adults. We derive incidence estimates from DisMod assuming age-specific remission rates from a follow-up study in the US (Bronnimann & Burrows 1986), which are consistent with overall remissions reported by Australia studies (Xuan et al. 2002). From findings reported by Bauman and colleagues, we calculate that asthmatics are symptomatic 12% of the time (Bauman et al. 1992b). Rather than use the Dutch weight for this health state (0.36), which we consider to be for a more severe health state than the average for symptomatic asthmatics in the population, we use a derived weight of 0.229 based on the severity distributions found in the 1998 Australian disability survey (ABS 1998b) and the disability weight regression model. The remainder of the time

we assume is spent in a state equivalent to the Dutch weight for asthma controlled by treatment (0.03). This results in a combined weight of 0.054.

2N Diseases of the digestive system

Peptic ulcer disease

In the absence of Australian population data on the frequency of peptic ulcer disease, we assume that all incident cases of peptic ulcer disease visit a general practitioner and base our estimates on the Australian general practitioner data. We assume that 83% of cases are treated by *Helicobacter pylori* eradication therapy, which has a cure rate of 90% (Mollison et al. 1999). We model those who are cured using eradication therapy as being symptomatic for one month, with no residual disability. We assume that the remainder of those who are treated but not cured (including those receiving alternative treatments) receive relief from their treatment but remain with the condition for the GBD duration. Untreated cases we assume to be symptomatic for the same period. Because the annualised Dutch weight for peptic ulcer disease is implausible, we use derived weights from the Dutch study for both symptomatic and treated states.

Cirrhosis of the liver

The methods of deriving estimates of alcohol-related cirrhosis and the category of 'other' cirrhosis have been described in the section on hepatitis.

Inflammatory bowel disease

We model two manifestations of inflammatory bowel disease: Crohn's disease and ulcerative colitis. We estimate the incidence of inflammatory bowel disease in adults from a European study (Shivananda et al. 1996) and for children we pool estimates from a number of international studies based on a recent review (Griffiths 2004). The relative risks of mortality due to the two types of inflammatory bowel disease were based on the findings of a recent large UK study which showed that inflammatory bowel disease was associated with a small overall increase in mortality after controlling for smoking and sex (Card et al. 2003). We assume no remission and derive a composite disability weight (0.224), assuming that 20% of time is spent with active exacerbation and the remainder is in 'remission' (Griffiths 1995; Hendriksen et al. 1985; Stonnington et al. 1987).

For inflammatory bowel disease (and vascular insufficiency of the intestine, diverticulitis and intestinal obstruction), we assume that a proportion of cases have more complicated surgery involving the creation of a stoma (a surgical opening in the skin of the abdomen for excretion of faeces) that can be either permanent or temporary. We estimate the incidence of inflammatory bowel disease cases that receive a temporary or permanent stoma from Australian hospital data. We apply the ratio of stoma for Crohn's disease to stoma for ulcerative colitis from an analysis of Victorian linked hospital data. Similarly the average duration of temporary stoma was estimated from Victorian hospital data from 1998–99 to 2001–02 to determine if they were closed and, if closed, the time to closure. The duration of permanent stoma was taken to be the same as the duration of the respective condition. We

assume stomas not yet closed within this period remain open indefinitely. In the absence of a specific weight for this condition, we derive a weight (0.204) from the disability weight regression model.

Other diseases of the digestive system

We base the incidence estimates for appendicitis, intestinal obstruction, diverticulitis, gall bladder and bile duct disease, pancreatitis and vascular insufficiency of the intestine on the numbers of people with a relevant hospital procedure or diagnosis from Australian hospital data. With the exception of appendicitis, these conditions were not considered in either the GBD or Dutch studies. We adopt a 2-week duration for appendicitis, and a 3-week duration for gall bladder and bile duct disease, intestinal obstruction, vascular insufficiency and pancreatitis. For each of these conditions, we assume the GBD weight for appendicitis. For gall bladder and bile duct disease, we use cholecystectomies or bile duct incisions but ignore people admitted with un-operated cholelithiasis on the assumption that these people are largely asymptomatic.

20 Genitourinary diseases

Nephritis & nephrosis

We base the incidence of dialysis and transplant patients on the Australian dialysis and transplant data from which we derive durations for both categories of patients using DisMod. For dialysis patients, we use case-fatality rates to match observed deaths and remission through transplant, and apply the Dutch weight for diabetic nephropathy (0.290). In the first 6 months after transplant, we assume a health state equivalent to the Dutch weight for diabetic nephropathy (0.290). For the remaining period with the transplant, we use a weight of 0.11, which is equivalent to both the GBD weight for treated renal failure and the Dutch weight for 'uncertain prognosis'. We derive untreated end stage renal failure from the difference between dialysis or transplant deaths and total renal deaths, to which we apply an average duration of 1 year prior to death at the GBD weight for untreated renal failure (0.104). We use Australian dialysis and transplant data on underlying renal disease distribution to attribute YLD from diabetic nephropathy to diabetes, analgesic nephropathy to the injury category of medical misadventure, and congenital dysplasia and polycystic kidney disease to congenital urogenital disease, and retain only those for primary renal disease in the 'nephritis & nephrosis' category.

Benign prostatic hypertrophy

We base the incidence of benign prostatic hypertrophy on Australian hospital data. Based on expert advice we adjust the number of benign prostatic hypertrophy cases upwards to account for the proportion of cases that receive medical instead of surgical treatment. We also assume, based on expert opinion, that half of all benign prostatic hypertrophy cases receive surgical treatment, a proportion of whom experience complications or continuing symptoms following surgery (1% with lifelong incontinence at a derived weight of 0.204, 15% with lifelong impotence at the GBD weight of 0.195, and 5% with urethral stricture for 4 weeks at the GBD weight of 0.151). Of those opting for medical treatment, we assume 70%

use alpha-blocker drugs, of which half are cured. The other half may then try surgery. We assume none of those receiving drugs other than alpha-blockers are cured. We apply the GBD weight for symptomatic benign prostatic hypertrophy to each of these intervention pathways assuming the following durations: 1.5 years for surgery, 1 year for successful medical treatment, 2 years for unsuccessful medical treatment then surgery, and lifelong for unsuccessful medical treatment but no surgery.

Urinary incontinence

We derive incidence rates of incontinence from DisMod using prevalence figures reported in a review of Australian and international literature (AIHW: Lea 1993) and from Women's Health Australia. We assume that a number of diseases and injuries are associated with this condition, most of which are more prevalent at older ages, and that the underlying causes are multi-factorial and interrelated. Based on a multivariate analysis (Chiarelli et al. 1999), we assume that, while all disability from incontinence among younger men and younger and middle-aged women belongs under this category, half that experienced by middle-aged and older men and older women is already captured under other conditions either explicitly (for example, as a sequela for benign prostatic hypertrophy among men) or implicitly as part of the overall weightings for these conditions (for example, severe stroke). For unaccounted incontinence, we apply an average of the GBD weight for moderate incontinence and the derived weight for benign prostatic hypertrophy-related severe incontinence using severity distributions from the 1998 Australian disability survey.

Infertility

We estimate the prevalence of infertility from a 1988 population survey of infertility, surgical sterility and associated reproductive disability in Perth, Western Australia (Webb & Holman 1992). This survey indicates that of the 3.5% of couples with non-surgical infertility, 68% have an associated reproductive disability defined in terms of the couple being unable to achieve a desired level of reproductive function. From a review of patients at an Adelaide infertility clinic indicating that 83% of couples with reproductive disability seek assisted reproductive technologies, 30% of whom achieve a pregnancy within 2 years (Weiss et al. 1992), we derive a net prevalence of 1.02% and 0.73% for short-term reproductive disability and 0.67% and 0.48% for long-term reproductive disability in females and males respectively. The causes of infertility are derived from recent national data on assisted conception and reproduction (AIHW: Dean & Sullivan 2003; AIHW: Ford et al. 2003). For short-term cases, we assume incident cases equal prevalent cases divided by the duration, which we assume is 2 years. For long-term cases, we derive incidence and durations from DisMod assuming non-zero remission rates from ages 45 years or over to account for declining prevalence of reproductive disability reflecting adoptions and changes in reproductive goals. For women, we subtract from the total number of long-term incident cases the estimated incidence of infertility as a sequela to maternal sepsis, abortion and pelvic inflammatory disease, the disability of which is calculated under chlamydia and gonorrhoea. We determine the duration of long-term infertility by subtracting the age at onset estimated in DisMod from 45 years. GBD weights are used for both short- and long-term reproductive disability.

Other genitourinary diseases

For this residual category, we assume the application of a simple YLD to YLL ratio of one across the age groups is sufficient to capture the morbidity from other genitourinary diseases in men. This method, however, does not capture the significant burden experienced by women, particularly at younger ages. We therefore calculate separate models for menstrual disorders and hysterectomies for menorrhagia, genital prolapse and endometriosis.

We base our estimates for menstrual disorders on women who report they often have severe period pain or premenstrual tension in the last 12 months from Women's health Australia. For severe period pain we assume a duration of 1 day per month and a disability weight similar to that for caesarean section. For menstrual tension we assume a duration of 2 days each month and we use the disability weight for mild depression. We use DisMod to model the conditions, assuming no excess mortality and remission of 0.1 for ages less than 50 years.

We model disability from hysterectomies associated with menorrhagia, genital prolapse and endometriosis in terms of disability from both the procedure and the resulting infertility. We derive the number of procedures from hospital data and we assume a 2-week duration at the derived weight for laparotomy of 0.349 (compare with estimates for caesarean section). Following the findings of a survey of surgical sterility in Perth (Webb & Holman 1992), we assume the majority of women who undergo a hysterectomy have completed their reproductive objectives, and that infertility leads to disability in 3.3% of cases with endometriosis. We apply the GBD weight for infertility.

2P Skin diseases

Eczema, acne and psoriasis

We model the incidence of severe eczema (that is, an episode in the past 12 months that disrupts sleep on average one or more nights per week) using self-reported prevalence data from a study of Melbourne school children (Robertson et al. 2004) and from the National Health Survey for adults (ABS 2001c). For other skin conditions we limit our estimates to severe acne and moderate and severe psoriasis. Prevalence figures for acne are based on a study of Australian school children and a study of adults in Central Victoria (Kilkenny et al. 1998; Marks et al. 1999). Prevalence figures for psoriasis were derived from the National Health Survey and from the central Victorian study (Marks et al. 1999; Plunkett et al. 1999). We derive incidence and duration estimates from DisMod assuming no excess mortality and a remission rate of 0.1 for eczema (Thestrup-Pedersen 2003), 0.27 for acne (assuming 70% spontaneous remission after 4 to 5 years) and 0.3 for psoriasis. For eczema we derive a disability weight (0.019) from the disability weight regression model which we adjust for 3 symptomatic episodes per year lasting 6 weeks in total. For acne we use the unadjusted disability weight for eczema from the disability weight regression model (0.056) and for psoriasis we apply the GBD weight for vitiligo.

Other skin diseases

We model the disability associated with chronic leg, skin and varicose ulcers, excluding decubitus and cellulitis which we assume are captured elsewhere. We use the weighted

incident cases of skin ulcers from Australian general practitioner data to estimate the incidence of other chronic skin ulcers. YLD for diabetic foot is included within the diabetes mellitus model. To avoid double-counting diabetic foot we adjust our incident estimates for skin ulcers using Western Australian aetiological data on the proportion of leg ulceration cases that had diabetes (Baker et al. 1992). In the absence of more specific information we use the same assumptions for duration (8.9 months) and disability (0.131) as the diabetic foot model.

2Q Musculoskeletal diseases

Musculoskeletal diseases are highly prevalent in the population. The fair to good test-retest reliability of self-reported musculoskeletal diseases and the consistent correlation with pain make health survey self-reports of some use to measure musculoskeletal conditions. Although the prevalence of most musculoskeletal diseases differs substantially depending on the measurement method, with self-report showing the highest prevalence, the pattern of prevalence in men and women is often similar. A higher prevalence of herniated disc of the back and gout is found in men, whereas for most other musculoskeletal diseases the prevalence is higher among women than among men (Picavet & Hazes 2003).

Rheumatoid arthritis

Given the small numbers in Australian studies on rheumatoid arthritis and problems with proper incidence and remission measurement, we base our incidence estimates for this condition on the international literature. For juvenile chronic arthritis, we use findings from a population study during 1984–1988 in Sweden (Gare & Fasth 1992). For adults, we use results from a 40-year follow-up study of a population-based cohort in Rochester, Minnesota, USA (Doran et al. 2002). We derived durations from DisMod assuming a relative risk of mortality of 1.6 at ages 15 years or over (Pincus et al. 1994), with no increased risk for children, and a remission rate of 0.04 (Prevoe et al. 1996) indicating that, while drug treatment may slow the disease process and remission is the ultimate endpoint of treatment, most therapeutic options have fallen short of achieving this (Sesin & Bingham 2005). Because progression through the three stages of rheumatoid arthritis described by the Dutch weights is relatively rapid, we do not model this condition as progressive. Rather we apply an average of the Dutch weights using severity distributions for American adults (Hakala et al. 1994) and those relating to Swedish children (Gare & Fasth 1992).

Osteoarthritis

While there are a few Australian population-based studies on self-reported osteoarthritis (Jones et al. 1995; March et al. 1998), we prefer to base our estimates for this condition on reported findings of radiographic osteoarthritis (grade 2 and above) by affected joint, age and sex from a large-scale study in Massachusetts, USA (Jones et al. 1995; March et al. 1998). We model hip and knee osteoarthritis only, given the high correlation between osteoarthritis of the hip, hand and fingers (Spector et al. 1997). We used DisMod to derive average durations, assuming a slightly increased risk of mortality (1.1) and the observed remission rate from joint replacement surgery. Because osteoarthritis is a relatively slow progressive disease, with few patients showing symptomatic progression over an 11-year period (Ahern

& Smith 1997), we apply an average of the relevant Dutch weights, assuming a severity distribution based on the Framingham study (Guccione et al. 1990).

Back pain

Back pain is a very common condition, with about 70–90% of people suffering from it in some form at some point in their lives (Hicks et al. 2002). Back pain may be viewed as running either an acute or chronic course. Acute back pain is usually considered to have a short duration and tends to resolve within days to weeks. However, recurrence of acute episodes is common and there is some contention as to the difference between recurring acute back pain and long-term chronic back pain. A duration of back pain lasting at least 3 months commonly underlies the definition of chronic back pain (NINDS 2006), and is often likely to continue indefinitely (Von Korff & Saunders 1996). Our estimates for back pain are based on self-reported prevalence of recent episodes, and long-term back pain from the 2003 Australian disability survey and the 1995 National health survey. We model recent episodes of (acute) back pain and long-term (chronic) back pain separately. Prevalence of long-term back pain resulting in at least mild disability is obtained from the Australian disability survey. Of these, the cases that were due to recent episodes of back pain were not identified separately. We therefore estimated the proportion due to recent episodes by applying the percentage of recent cases of long-term back pain from the 1995 National Health Survey. We use the Dutch weight for low back pain (0.06) as the disability weight for recent episodes of back pain, which applies to an average health state involving some problems in walking about and in usual activities, as well as moderate pain or discomfort. We assume an average duration of 4 days for painful and limiting episodes of back pain. To model chronic back pain, we use the prevalence of long-term back pain (not identified as recent episodes as described above) from the 2003 Australian disability survey. We use DisMod to derive the incidence and duration of chronic back pain, assuming a remission rate of 10% and no increased risk of mortality. For many people, there are few treatment alternatives and complete relief is rare (Atkinson 2004). We assume that 14% of long-term cases experience constant or persistent pain (Quittan 2002), and 86% experience pain 1 day per week. We use the GBD disability weight for chronic intervertebral disc pain of 0.103.

Slipped disc

Our estimates for slipped disc are based on numbers of intervertebral disc procedures from Australian hospital data. We assume only 7.5% of incident cases of disc displacement receive surgery (Deyo et al. 1990), and derive total annual episodes from this proportion. We assume on average an episode of discomfort lasts 4 weeks. For those who receive surgery, we take the median time of 224 days from onset of symptoms to recovery reported in the literature (Rasmussen 1996). In the absence of weights for both these health states, we use the Dutch weight for low back pain (0.06). Based on a 5-year follow-up study (Kurth et al. 1996), we model 14% of operated cases as going on to experience long-term chronic pain with a lifelong duration at the GBD disability weight for chronic intervertebral disc of 0.103.

Occupational overuse syndrome

Occupational overuse syndrome (formerly known as repetition strain injury) is a contentious condition with considerable disagreement within the literature about its aetiology and

pathophysiology (Byrne 1992; Cohen et al. 1992; Helme et al. 1992). Our model uses self-report prevalence data on 'repetition strain injury' from the 2003 Australian disability survey from which we derive incidence figures using DisMod assuming an average duration of 3 years and no mortality. In the absence of Dutch or GBD weights for this condition, we use sex-specific derived weights to account for the fact that all males in the 1993 Australian disability survey had mild or no handicap, whereas 26% of females had moderate handicap and 17% had severe or profound handicap.

Gout

Our estimates for gout are based on self-reported prevalence from the National Health Survey which has the same overall result as found in a general practitioner study in the UK (Mikuls et al. 2005). We assume a slight increased risk of mortality associated with gout (relative risk=1.1) and no remission, based on information that at 1 year 62%, at 2 years 78% and at 10 years 93% has had at least one repeat attack (Alamo Family Foot and Ankle Care 2005). Fitting a Weibull function to these figures gives an average time to the next episode of 2.2 years, but this is rather high because of the skewness of the function. The median time to next episode is 0.44 years. We assume that 10% has chronic symptoms and the remaining 90% has an attack of 1 week every 0.44 years. Given that people may suffer gout at varying levels, from acute attacks of a short duration to chronic gout, we assume on average one attack per 2 months lasting 1 week in 90% of people and the remaining 10% suffer chronic ongoing disease at the GBD disability weight of 0.061.

Other musculoskeletal disorders

Because mortality for musculoskeletal conditions is low and because 49% of deaths from musculoskeletal disorders do not fall within the above categories, a derivation of disability for this rest category by applying a ratio of YLD to YLL for the explicitly modelled musculoskeletal conditions is not plausible. Therefore we try to model disability from all other conditions explicitly. In the absence of detailed information, we define an 'other' category comprising both prevalent minor conditions and more serious diseases (for example joint derangement and disorders; osteopathies; chondropathies and other bone disorders; connective tissue diseases; and soft tissue problems such as rheumatism, ganglions, bunions, bursitis, cramps, tenosynovitis and tennis elbow). We base our estimates for these conditions on the prevalence of other musculoskeletal disorders that have not been accounted for in each of the musculoskeletal models described above from the 2001 National Health Survey. Based on figures from the 2003 Australian disability survey, we assume a proportion of prevalent cases report on refer to musculoskeletal sequelae of other diseases or injuries, which we account for by adjusting overall prevalence figures downwards by 50%.

For recent non-chronic cases, we assume the same duration and weight as for recent episodes of back pain. For chronic cases, we derive incidence rates and durations from DisMod assuming no excess mortality and a remission rate of 0.1. We take the proportion reporting symptoms in the 2 weeks before interview as an approximation of the proportion of time spent symptomatic and assume symptomatic chronic cases experience a health state equivalent to the weight for low back pain.

2R Congenital anomalies

Congenital heart disease

We model the disability associated with four types of congenital heart disease for live-born infants: surgically treated atrial or ventricular septal defect, surgically treated Fallot's tetralogy or transposition of great vessels, surgically treated pulmonary stenosis, and complex but not curatively operable congenital heart disease. We derive the incidence of the first three conditions from Australian hospital data by assuming that all curative procedures represent an incident case with disability. We assume a duration of 1 year before operation with disability equivalent to the Dutch weight for moderate heart failure (0.35) and post-surgery we use relevant Dutch weights and assume reduced life expectancy, except for those with septal defects (Miyamura et al. 1993; Nollert et al. 1997a, 1997b). We assume disability starts at birth and we discount YLD back to birth to account for this. We derive the incidence of other congenital heart malformations from Victorian birth defects data. Following expert advice, we assume that 50% of these cases are complex but not curatively operable. We assume that duration is half of those with surgically treatable conditions and use the relevant Dutch weight (0.72).

Digestive system malformations

We model the disability for anorectal and oesophageal atresia and other digestive system malformations. We estimate the incidence of digestive system atresia for cases surviving 28 days using Victorian birth defects data. We assume 26 weeks of disability from birth at the GBD weight for anorectal atresia (0.85). After this period, we assume that a proportion of both types of atresia cases have lifelong problems (15% and 20% respectively) and decreased life expectancy (by 10 and 5 years respectively) and disability equivalent to health state 111211 for two-thirds of the time (0.037) (Ludman & Spitz 2003). We estimate the incidence of other digestive system malformations using data from the Australian congenital malformations dataset (AIHW: Hurst et al. 2001). We assume no long-term disability, and a 1-month period of disability from birth equivalent to the GBD weight for anorectal atresia.

Renal agenesis

We estimate the incidence of unilateral and bilateral renal agenesis for cases surviving 28 days using Victorian birth defects data. For unilateral cases we assume that 20% of survivors have ongoing problems, with a life expectancy of 70 years and a disability of 0.067. For bilateral cases we assume an average duration of 3.5 days and use the GBD weight for renal agenesis (0.85). We also calculate YLD for renal failure due to renal dysplasia based on attributions from Australian dialysis and transplant data.

Other urogenital tract malformations

We model the disability associated with the following urogenital tract malformations: cystic kidney disease, obstructive defects of renal pelvis and ureter, and other urinary tract malformations. We estimate the incidence of cases of other urogenital tract malformations surviving beyond 28 days from the Victorian, Western Australian and Australian birth

defects data. We assume 30% of cases have chronic lifelong problems, with a life expectancy of 50 years and a disability weight of 0.067. YLD were also calculated for end-stage renal failure due to cystic kidney disease.

Other congenital anomalies

We estimate the incidence of anencephaly using Australian mortality data for newborns, assuming deaths are equivalent to incident cases. We assume a duration of 1 week with a disability weight of 1. For spina bifida, we estimate the average annual number of live births that survive the first 28 days from Victorian birth defect data (Riley & Halliday 2004). We derive an average disability weight (0.52) based on the Dutch weights for each level of severity combined with severity distributions from expert advice. We estimate the incidence of surgically treated cleft lip and cleft palate from Australian hospital data, assuming that all curative procedures represent a case and that all cases are treated within the first year. We assume disability equivalent to the 'treated' GBD weights (0.016, 0.015 respectively). YLD estimates for Down syndrome and 'other chromosomal anomalies' are calculated as described in the section on intellectual disability (see Section 2K).

We estimate the incidence of abdominal wall defects (exomphalos and gastroschisis) in infants surviving >28 days using 2001 Australian birth defects data (AIHW NPSU 2004) and survival data from the Victorian birth defects data. We assume a duration of 4 weeks based on Australian and international literature (Dimitriou et al. 2000; Sharp et al. 2000) and apply the GBD weight for abdominal wall defect. Based on expert advice we assume that 20% of cases have lifelong problems, a shortened life expectancy by 20 years, and disability weight of 0.200 (the Dutch weight for young adult in permanent stage after surgical repair to Fallot's tetralogy).

2S Oral conditions

Caries

The incidence of caries is measured by one or more new dental cavities (caries increment). The occurrence of dental caries in an individual is measured using the DMFT or DMFS index: the number of decayed (D), missing (M) and filled (F) primary or permanent teeth (T) or surfaces (S). A review of the relationship between DMFT and DMFS suggests that DMFS data should be adjusted by a factor of 1/3.5 to be consistent with DMFT data (Carvalho et al. 2004; Hopcraft & Morgan 2005; Rosen et al. 2004).

For children and adults we estimate the incidence from representative Australian caries prevalence data: the 2000 Child Dental Health Survey (AIHW: Armfield et al. 2004) and the 1987–88 National Oral Health Survey of Australia (Barnard 1993). Fitting linear regression lines to the prevalence data gives slopes in children (1–14 years) of 0.25 (AIHW: Armfield et al. 2004; Davies et al. 1997) and in adults (15–59 years) of 0.27 (Barnard 1993). For older adults (60 years or over) and nursing home residents (60 years or over) we estimate the incidence of caries from the South Australia Dental Longitudinal Study (AIHW DSRU 2002) and the 1998 Adelaide Dental Study of Nursing Homes (AIHW: Chalmers et al. 2001), respectively. Based on the 5-year increment of all new carious surfaces, the 1-year increment (assuming that the incidence of carious surfaces over the 5-year period was evenly

distributed) is 0.98 (AIHW DSRU 2002). The 1-year increment of new carious surfaces in nursing home residents is 3.5 (AIHW: Chalmers et al. 2001). We use our DMFT/DMFS adjustment factor to give annual caries increments of 0.28 and 1.0 respectively for older adults in the general population and nursing homes.

The previous Australian burden study assumed a symptomatic duration of 10 weeks based on advice from the Australian Research Centre for Population Oral Health. More recent work, based on patient self-report, by this group suggests durations in the order of 81 weeks (Brennan & Spencer 2004, 2005). However, both of these estimates refer to time spent with and without symptoms. A review of the literature shows that there is a paucity of information on symptomatic caries, specifically mean duration of symptoms and proportion of people who are symptomatic. A patient-based study in children in the UK reported that 78% of the children sampled presented within 1 month of pain onset (Mason et al. 1997) whereas a patient-based study in New Zealand observed that 67% of adults presented within 1 month of pain onset (Whyman et al. 1996). Patient-based samples are biased as they do not reflect all cases of caries in the community. Neither of these studies provided data on the mean durations for those people experiencing symptoms for greater than 1 month. We estimate the average time symptomatic for those people presenting with caries problems by fitting a lognormal distribution to the midpoint of the observed durations. This gives mean durations of symptomatic caries of 28 days in children and 55 days in adults. We base our estimate of people with symptomatic caries (32.4%) on the findings of the 1998 Australian Longitudinal Study of Dentists' Practice Activity (Brennan & Spencer 2002).

Following the first Australian burden of disease study the Australian Research Centre for Population Oral Health developed disability weights for oral disease using a patient-based sample in South Australia (Brennan & Spencer 2004, 2005). Disability weights for caries (0.044), periodontal disease (0.023) and denture problems (0.026) in this study were higher than comparable Dutch weights used in the previous Australian burden study (0.005 for caries involving a filling and 0.014 for caries involving an extraction, 0.007 for periodontal disease, and 0.004 for edentulism). We did not use these Australian-derived disability weights because patient-based samples are likely to under-represent asymptomatic people, and questions with limited response categories are likely to bias results. For instance, the duration-related question was 'During the period that you have had this dental problem, what percentage of the time (0% = none of the time, 50% = half of the time, 100% = all of the time) have you experienced the limitations listed above in relation to: mobility, self-care, usual activities, pain/discomfort, anxiety/depression, cognition?' (Brennan & Spencer 2004, 2005). Both of these limitations are likely to over-estimate the percentage of people reporting problems for each of the health dimensions as well as the duration of their symptoms.

We follow expert advice and derive a disability weight for symptomatic caries (0.057) using the disability weight regression model (health states: 20% – 111211 and 80% – 111111).

Edentulism

We estimate the prevalence of edentulism (loss of all natural teeth) for the general population and nursing home residents using the 2002 National Dental Telephone Interview Survey (AIHW: Carter & Stewart 2003) and the 1998 Adelaide Dental Study of Nursing Homes (AIHW: Chalmers et al. 2001), respectively. We derive incidence and duration using DisMod, based on these studies, assuming no remission and no excess case-fatality. We model a 2% declining time trend to reflect the observed decline of the prevalence of

edentulism from 20.5% in 1979 to 8.0% in 2002 (Sanders et al. 2004). We use the same disability weight as in the previous Australian burden study.

Periodontal disease

We estimate the prevalence of periodontal pockets larger than 6 mm using data from the 1987–88 National Oral Health Survey of Australia (Barnard 1993). Expert advice suggested that periodontal disease is a largely asymptomatic risk factor for tooth loss; pain occurs in around 1% of time when an abscess forms in a periodontal pocket; and the typical duration of periodontal disease is around 15 years. We derive incidence and duration using DisMod based on the Australian prevalence data, remission rates that reflect 15 years average duration and no case-fatality. A new disability weight for periodontal abscess was estimated (0.056) based on the disability weight regression model (health state 111211).

Pulpitis

We estimate the incidence of pulpitis using the proportion of patients sampled in the 1998 Longitudinal Study of Dentists' Practice Activity (AIHW: Spencer & Brennan 2002) who had a main diagnosis of pulpal infection and the total number of dental consultations in Australia in 2003. We assume that most people with pulpal infection will visit a dentist. We estimate the total number of dental consultations by multiplying the proportion of people who visited a dentist in the last 12 months by the mean number of dental visits per person (from the 2002 National Dental Telephone Information Survey (AIHW: Carter & Stewart 2003)) and 2003 population data (excluding the edentulous population). Expert consultation suggests that a symptomatic duration of 1 month is plausible for pulpitis with the first few weeks consisting of intermittent pain and the last week being of more severe and consistent pain. We assume that 71.3% of people with pulpitis presenting in pain, a figure which we derive from the 1998 Longitudinal Study of Dentists' Practice Activity. We use the disability weight regression model to estimate a disability weight for pulpitis assuming that 1 week is spent in moderate pain and 3 weeks are spent at a level of the disability for moderate pain for 10% of the time.

2Z Chronic fatigue syndrome

We base our model for chronic fatigue syndrome on the internationally accepted US Centers for Disease Control and Prevention criteria, which state that for a patient to receive a diagnosis of chronic fatigue syndrome, they must have severe chronic fatigue of 6 months or longer duration with other known medical conditions excluded by clinical diagnosis, and concurrently have four or more of the following symptoms: substantial impairment in short-term memory or concentration; sore throat; tender lymph nodes; muscle pain; multi-joint pain without swelling or redness; headaches of a new type, pattern or severity; unrefreshing sleep; and post-exertional malaise lasting more than 24 hours. The symptoms must have persisted or recurred during six or more consecutive months of illness and must not have predated the fatigue (Fukuda et al. 1994).

Following expert consultation we conceptualise two manifestations of chronic fatigue syndrome: (a) post-infective fatigue syndrome which constitutes between 30–40% of cases and is characterised as an acute outcome of viral and non-viral infections, has a disability starting point of moderate severity, a median duration of 12 months, and around 99%

recovery at 2 years (Hickie et al. submitted 2005; Wilson et al. 2001); and (b) protracted chronic fatigue syndrome, which constitutes the remaining 60–70% of chronic fatigue syndrome cases, where cases have an insidious onset with initially severe disability followed by cases fluctuating around 50–80% of their previous healthy state, and a median duration of around 7 years. We assume that the disability associated with post-infective fatigue syndrome is included within the disability weights and durations in the relevant infectious disease models (explicitly in the arbovirus estimates but not for other viral infections such as Q fever and Epstein-Barr virus which are subsumed in the rest of infectious disease category).

We base our estimates of prevalence for protracted chronic fatigue syndrome on the population-based study of chronic fatigue syndrome conducted in Wichita, Kansas, USA in 1997 (Reyes et al. 2003). In the previous Australian burden study we used prevalence estimates based on an Australian prevalence study of chronic fatigue syndrome (Lloyd et al. 1990). This study's applicability in the current context is limited due to the different diagnostic criteria used and the physician referral sample. The population-based study by Reyes and colleagues (2003) showed that only 16% of people identified with chronic fatigue syndrome had previously been diagnosed as such by a medical practitioner. Although it is not clear how similar the epidemiology of chronic fatigue syndrome is between the US and Australia, the findings from an international multi-centre study of the prevalence of chronic fatigue syndrome in patients lend support to the notion that the epidemiology of chronic fatigue syndrome is similar in the two countries (Wilson et al. 2001).

We model incidence and duration using DisMod, assuming no excess mortality and remission rates which gave an average duration of 7.3 years (Reyes et al. 2003). We assume that 90% of the time people with chronic fatigue syndrome are symptomatic, using findings from the 1993 Australian disability survey. In the absence of an established disability weight for chronic fatigue syndrome we use the disability weight estimated for the previous Australian burden study.

3 Injuries

We model the disability from non-fatal injuries where a person has an injury severe enough to warrant emergency department or inpatient hospital treatment but that does not lead to death. This method assumes that injuries treated outside the hospital system do not result in significant disability. We derive non-fatal incident injuries from Australian hospital data. We classify incident cases according to a matrix of 14 'external cause of injury' categories (12 unintentional and two intentional) and 32 'nature of injury' categories (for example fractures, burns, wounds, brain injury, spinal cord injury). We exclude admissions for the same ICD-10 code within 90 days, on the assumption that these are re-admissions, as well as, those resulting in death. Given that it is not uncommon for multiple sites of the body to be damaged from a single accident, we estimate disability for only the most disabling ICD-10 code associated with each incident, on the assumption that the disability for the other ICD-10 codes is captured in the weight for the more severe injury. We redistribute ill defined injuries and adjust estimates for 'amputated finger' as in the previous Australian burden of disease study. We use disability weights, durations and the risk of mortality as per the GBD study.

Appendix 2: Methods for attributing risk

In this section we describe our methods for assessing the contribution of 14 health risks to the total burden of disease and injury in Australia. For most risks, our analyses are based on methods developed by the WHO CRA project and described in detail elsewhere (Ezzati et al. 2004a). Briefly, the main inputs are the prevalence of exposure to a health risk in a population and information on the risk of disease, injury or death (referred to here as relative risk or hazard) from this exposure, which is typically derive from systematic reviews of the international literature. Our analyses are not comprehensive since choices had to be made about which risks to include on the basis of certain criteria, as outlined at the beginning of Chapter 4. We begin by describing the methodological basis of our analyses, the population attributable fraction.

Estimating population attributable fractions

The population attributable fraction (PAF) is a subtype of a more general measure – the ‘potential impact fraction’ (PIF). The PIF measures the proportional reduction in disease or injury burden experienced by a population that would occur if the population were subjected to an alternative or ‘counterfactual’ distribution of exposure to a particular health risk. If the alternative exposure scenario is set to a level such that it represents the lowest possible risk in a population (no exposure, for example), the PIF represents the total amount of burden that is attributable to that risk; in this instance it is called the ‘population attributable fraction’ (Eide & Heuch 2001; Miettinen 1974). For health risks that are measured on a continuous scale, the PIF can be defined thus:

$$PIF = \frac{\int_{x=0}^m RR(x)P(x) dx - \int_{x=0}^m RR(x)P'(x) dx}{\int_{x=0}^m RR(x)P(x) dx}$$

Where $RR(x)$ = relative risk at exposure level, $P(x)$ = population distribution of exposure, $P'(x)$ = counterfactual distribution of exposure, and m = maximum exposure level

(Equation 1)

When a risk is measured on a categorical scale, the discrete version of the PIF formula is (Eide & Heuch 2001; Walter 1980):

$$PIF = \frac{\sum_c P_c RR_c - \sum_c P_c^* RR_c}{\sum_c P_c RR_c}$$

Where c = an index for category, P = prevalence, and P^* = prevalence after a change, and RR = relative risk

(Equation 2)

The difference between Equation 1 and Equation 2 in practical terms is that the latter can easily be resolved in a spreadsheet environment, whereas the former requires more advanced mathematical techniques. Equation 2 is mathematically the same as the PAF formula for risk factors with multiple categories given by English and colleagues (Equation 3), if the counterfactual is set as the hypothetical minimum distribution (English et al. 1995).

$$PAF = \frac{\sum_c P_c (RR_c - 1)}{\sum_c P_c (RR_c - 1) + 1}$$

(Equation 3)

Choice of theoretical minimum

Calculating a PAF requires the explicit characterisation of an exposure distribution that represents the lowest possible level of risk in a population. This has been termed the ‘theoretical minimum exposure distribution’ and corresponds to zero exposure for some risks (for example smoking). For other risks, however, zero exposure is inappropriate because it is physiologically impossible (for example systolic blood pressure, BMI and cholesterol). In this case the lowest levels observed in specific populations and epidemiological studies described in the literature are used instead. For example, a theoretical minima of 115 mmHg for systolic blood pressure and 3.8 mmol/L for total cholesterol (each with a small standard deviation) are the lowest levels at which the dose-response relationships have been characterised (Chen et al. 1991; Eastern Stroke and Coronary Heart Disease Collaborative Research Group 1998; Law et al. 1994). For factors with protective effects (fruit and vegetable consumption and physical activity), the theoretical minimum exposure distribution is based on information from high exposure populations about the level to which the benefits continue to accrue given current scientific evidence.

Estimating attributable burden

Age- and sex-specific PAFs are calculated for each health risk and health outcome pair using the relationships in Equations 1 and 2. Where a relative risk of disease or injury is different to the relative risk for death, two PAFs are calculated, one for non-fatal burden and the other for fatal burden. PAFs are then multiplied with the relevant burden estimates for that health outcome and the sum of the burden across all outcomes affected by a health risk constitutes the total attributable burden for that risk. For example, if there are 1,000 deaths from ischaemic heart disease and 500 from stroke in a particular age and sex category, and the PAFs for cholesterol leading to ischaemic heart disease and stroke are 0.5 and 0.3 respectively, the mortality attributable to high cholesterol equals $1,000 \times 0.5 + 500 \times 0.3 = 650$. In other words, if the population had been exposed to the hypothetical minimum cholesterol distribution instead of the current distribution, 650 fewer deaths would have occurred.

Table A2.3 summarises the exposure levels, theoretical minima, health outcomes and sources of relative risks for each of the 14 health risks analysed in this report. Table A2.4 summarises

our estimates of exposure in the Australian population to each of these risks. A brief description the specific methods we used for each risk is provided below.

Tobacco

Given the long lag time between exposure to tobacco smoke and the occurrence of cancers and COPD, the attributable burden cannot be estimated from the current prevalence of smoking. Even with good historical information on smoking prevalence, it is not straightforward to determine the current amount of illness that is due to smoking because the lag time between the relevant exposure and disease is variable. Therefore, we used the method of Peto and colleagues, who proposed an artificial compound prevalence measure of the relevant past exposure to tobacco (Peto et al. 1992). This 'smoking impact ratio' is derived from a comparison of lung cancer mortality rates in the population of interest and lung cancer mortality rates among non-smokers and smokers observed in a large long-term follow-up study in the United States. We used this smoking impact ratio instead of the current prevalence in the standard calculation of attributable fractions for the other cancers and COPD. Compared with cancers and COPD, the mean time between exposure to tobacco and all other adverse health outcomes is considerably shorter. We therefore used prevalence estimates of smoking for adults aged 18 years and over in 2001, two years before our baseline year of 2003 (ABS 2001c).

Our previous calculations of attributable mortality burden included only those diseases for which English and colleagues report strong evidence of an association (English et al. 1995). For this report, we added other conditions for which reasonable evidence of an association with tobacco exists (AIHW: Ridolfo & Stevenson 2001): cancer of the stomach, endometrial cancer, peripheral vascular disease, pneumonia, inflammatory bowel disease, injuries from fires and Parkinson's disease. Tobacco has a small protective effect against Parkinson's disease and endometrial cancer. We omitted peptic ulcer disease, given evidence of its largely infectious aetiology. We also added the burden attributable to smoking from macular degeneration (Mitchell et al. 1999; Tomany et al. 2004).

In addition, we calculated the burden from passive smoking using attributable fractions for lung cancer, ischaemic heart disease, and asthma in children (NHMRC 1997a). For lower respiratory tract infection, sudden infant death syndrome and otitis media in children due to passive smoking, we used the prevalence of maternal smoking (Turrell et al. 2002) and relative risks from the US Surgeon General's Report (US Department of Health and Human Services 2006) and NHMRC (NHMRC 1997a). We also estimated the burden of low birth weight due to smoking during pregnancy using the relative risk from Ridolfo and Stevenson (2001) and estimates of smoking during pregnancy from Laws and Sullivan (2005).

High blood pressure

We used the AusDiab study (Dunstan et al. 2002) to estimate distributions of high blood pressure by age and sex in the Australian population. Despite a low response rate AusDiab is the only recent and representative study that has measured this risk in Australia. Relative risks came from Lawes and colleagues (2004a). We used the CRA theoretical minimum distribution for blood pressure (mean 115, SD 6 mmHg) as the counterfactual in this analysis.

High body mass

We used the AusDiab study (Dunstan et al. 2002) to estimate distributions of body mass index (BMI) by age and sex in the Australian population. Relative risk of type 2 diabetes came from the Asia Pacific Cohort Collaboration (2006); the relative risk of all remaining conditions associated with high body mass came from James and colleagues (2004). We used the CRA theoretical minimum distribution for BMI (mean 21, SD 1 kg/m²) as the counterfactual in this analysis.

Physical inactivity

Recent developments have led to the treatment of physical inactivity as a four-level categorical variable by subdividing the exposure group labelled as 'sufficiently active' in the CRA project into those 'meeting current recommendations' and 'highly active'. While physical activity levels equivalent to 2.5 hours per week of moderate-intensity activity (approximately 4000kJ/week) are considered an important target for population health benefits, the protective effects are expected to continue to higher levels. Therefore, the theoretical minimum exposure distribution was chosen to be the whole population in the 'high active' category to increase consistency with the counterfactual exposure distribution of other risk factors (Bull 2003; Murray et al. 2003; Powles & Day 2002) (Table A2.1). The required prevalence data were derived from the NHS 2001 (ABS 2001c). The exercise related questions in this survey relate to physical exercise undertaken for recreation, sport, health or fitness purposes, conceptually excluding physical activity undertaken as a part of work or for other purposes. This may underestimate the amount of physical activity undertaken, and therefore our analyses may overestimate the burden of disease attributable to physical inactivity.

The associated hazards were modified to correspond to the new referent category of 'highly active'. Given no available quantitative meta-analysis with comparable categories, risk estimates were derived from a synthesis of recent reviews (Kelley & Goodpaster 2001; Kesaniemi et al. 2001; Kohl 2001; Oguma et al. 2002; Thune & Furberg 2001; Williams 2001) and findings from several recent studies in which the results were reported separately by intensity of activity as well as total volume of activity (Manson et al. 2002; Sesso et al. 2000). The relative risk of ischaemic heart disease for the inactive group compared to 'high active' was set at 2.0, based on reviews of studies with both physical activity and fitness measures as well as a recent study's differential results for moderate versus vigorous activity. The likely linear dose-response relationship (Kesaniemi et al. 2001) was represented by the arithmetic midpoints for those classified as 'meeting current recommendations' and 'insufficiently active'. For stroke, the mean of nine studies summarised in the systematic review and meta-analysis by Blair and colleagues (2001) was used (relative risk of 2.0). The findings from the review by Thune and Furberg (2001) were used to derive the risk estimate for colon and breast cancer. There has been no quantitative review of diabetes and physical activity; therefore the relative risks from the CRA project were adjusted by the same magnitude as for ischaemic heart disease. It is recognised that these estimates of risk are derived from a synthesis of the available scientific evidence and alternative interpretations are possible.

Table A2.1 Physical activity exposure categories

Physical activity level	Definition
High	3 sessions x at least average 40 minutes vigorous AND total of at least 1500 METmins/week ^(a)
Recommended	3 sessions x at least average 20 minutes vigorous OR 5 x 30 minutes moderate OR 600 METmins/week
Insufficient	Some activity but not meeting recommendation
Inactive	No activity

(a) The standard metabolic equivalent, or MET, level. This unit is used to estimate the amount of oxygen used by the body during physical activity. One MET = the energy (oxygen) used by the body sitting quietly, perhaps while talking on the phone or reading a book. The harder the body works during the activity, the higher the MET.

High blood cholesterol

We used the AusDiab study (Dunstan et al. 2002) to estimate distributions of high blood cholesterol by age and sex in the Australian population. Relative risks came from Lawes and colleagues (2004b). We used the CRA theoretical minimum distribution for serum cholesterol (mean 3.8, SD 0.5 mmol/L) as the counterfactual in this analysis.

Alcohol

There are a number of recent data sources on the prevalence of alcohol consumption in the Australian population, including the 2004 National Drug Strategy Household Survey (NDSHS 2004) (AIHW & DoHA 2005) and the 2001 National Health Survey (NHS 2001) (ABS 2001c). The NHS 2001 focuses on the quantity of alcohol consumption on the three most recent days on which alcohol was consumed in the week prior to interview, while the NDSHS 2004 explicitly quantifies the amount of alcohol drunk on the day prior to interview. Of these, only the NHS 2001 collected information on the type and brand of alcoholic drinks consumed as well as the number. Also, the NHS 2001 gives average daily alcohol consumption over the previous week in millilitres. For this reason, we used the NHS 2001 to estimate the prevalence of alcohol consumption for adults aged 18 years or over.

We categorised the prevalence of alcohol consumption into the four levels used in English and colleagues' analysis of the risks of alcohol consumption (English et al. 1995), and with the NHMRC's recommendations on alcohol consumption (NHMRC 1992) (Table A2.2). The prevalence of each level of alcohol intake was estimated by age and sex from the average weekly consumption of alcohol after conversion to standard drinks per day. Data for people interviewed on each day of the week were reweighted to obtain prevalence of alcohol consumption based on equal samples for each day of the week. Those that last drank alcohol more than 1 week ago were classified as abstainers.

Table A2.2 Classification and prevalence of alcohol intake levels used in this report

Alcohol intake	Average number of standard drinks (= 10 g alcohol) per day	
	Males	Females
Abstinence	0–0.25	0–0.25
Low	0.26–4.00	0.26–2.00
Hazardous	4.01–6.00	2.01–4.00
Harmful	>6	>4

Source: English et al. 1995

We used relative risks and population attributable fractions from Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001) for conditions for which there is evidence of causation by alcohol consumption. English and colleagues (1995) estimated that 44% of fire injuries are attributable to alcohol; this was not updated by Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001). We revised these estimates with the addition of more recent studies, and produced a separate PAF for fire injuries and scalds or other burns for both YLD and YLL. We also updated the drowning PAF of 0.34 from Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001) with age-specific estimates from Driscoll and colleagues (AIHW: Driscoll et al. 2004) who found that 17% of unintentional drownings were attributed to alcohol (blood alcohol content of at least 0.10 g/100 ml). English and colleagues (1995) derived a PAF of 0.07 for alcohol, and occupational and machine injuries. We applied this to all machinery accidents, and to the occupational YLD PAFs for injury codes not already covered elsewhere in alcohol. For YLL we applied a PAF of 0.051 (Driscoll et al. 2001) to the occupational YLL PAFs for injury codes not already covered elsewhere in alcohol.

Low fruit and vegetable consumption

We used the National Health Survey (ABS 2001c) to estimate distributions of fruit and vegetable consumption by age and sex in the Australian population. Relative risks came from Lock and colleagues (2004). We used the CRA theoretical minimum risk distribution for fruit and vegetable consumption (mean 600, SD 50g/day) as the counterfactual in this analysis.

Illicit drugs

In addition to being a direct cause of death, illicit drugs are also risk factors for conditions such as HIV/AIDS, hepatitis, low birth weight, inflammatory heart disease, poisoning, and suicide & self-inflicted injuries. By definition, heroin, benzodiazepine, cannabis and other drug dependence and harmful use are due to illicit drug use; therefore the entire burden due to these conditions was attributed to this risk factor category. For infective endocarditis and suicide we used the attributable fractions for illicit drugs developed by English and colleagues (1995). The proportion of inflammatory heart disease that was due to infective endocarditis was derived from hospital data. The infective endocarditis PAF was then applied to this proportion only.

The proportion of HIV due to injecting drug use was based on diagnosed HIV from the Australian HIV Public Access Dataset (National Centre in HIV Epidemiology and Clinical Research 2005b). We use diagnosed rather than newly acquired HIV, which is in keeping

with YLD estimates, and due to the apparent stabilisation of HIV incidence over recent years. AIDS cases and deaths attributable to injecting drug use were from the Australian AIDS Public Access Dataset (National Centre in HIV Epidemiology and Clinical Research 2005a). Time to death (year of death minus year of AIDS diagnosis) was added to the midpoint of the age at diagnosis range to approximate age range at death. For those age-at-death ranges available in the AIDS Public Access Dataset, we used the age-specific proportion attributable to injecting drug use. For all other ages we applied the all-age proportion. For cases of HIV and AIDS, and AIDS deaths, we assumed that all cases with exposure category 'male homosexual contact and injecting drug use' were attributable to male homosexual contact.

The proportion of newly acquired hepatitis B and C cases due to injecting drug use was from the HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia Annual Surveillance Report 2004 (National Centre in HIV Epidemiology and Clinical Research 2004).

The proportion of road traffic accidents due to illicit drug use was derived from Drummer and colleagues (2004). We applied the methodology used by Ridolfo and Stevenson on earlier data from Drummer (1994).

For low birth weight we used prevalence of cannabis and opioid diagnosis during pregnancy in New South Wales and relative risks from Burns and colleagues (2006). The relative risk for antepartum haemorrhage attributable to illicit drug use was from English and colleagues (1995), with the prevalence being heroin or cocaine use in past 12 months for females aged 15–49 years from the NDSHS 2004.

The odds ratio for schizophrenia attributable to cannabis use was from Semple and colleagues (2005). This odds ratio is the result of a meta-analysis of seven studies with different classifications of psychosis and cannabis use. Despite these differences, there was consistency in the unadjusted odds ratios. We used prevalence of daily cannabis use over the last 12 months from the 2004 NDSHS (AIHW & DoHA 2005) to calculate the PAF to be applied to schizophrenia.

Occupational exposures and hazards

The attributable burden of occupational exposures and hazards was based on the following methods. Work-related fatal injuries were derived from the National Worker's Compensation Statistics Database accessed online via National Occupational Health and Safety Commission (NOHSC) Online Statistics Interactive (NOSI) <www.nosi2.nohsc.gov.au/>. Since compensation statistics do not cover all occurrences of occupational injury deaths, we inflated these figures according to a study carried out by Driscoll and colleagues (AIHW: Driscoll et al. 2004), that investigated the coverage of work-related traumatic deaths by official occupational health and safety and compensation agencies in Australia. Work-related deaths by mechanism, nature and industry from the NOSI database were inflated according to the proportion of all work-related deaths in 1989–92 covered by compensation agencies by industry (Table 3 in AIHW: Driscoll et al. 2004).

In the absence of more reliable information, the attributable fractions for non-fatal injuries were derived from an analysis of the National Hospital Morbidity Database 2002–03. For each age–sex–injury group, the attributable fraction for occupational injuries was estimated as the ratio of hospital episodes where 'workplace' was specified as the place where the injury occurred to the total hospital episodes where a place of occurrence was specified.

Where possible we derived non-injury attributable fractions by following the CRA methods (Concha-Barrientos et al. 2004). This produced age- and sex-specific attributable fractions for

lung cancer, leukaemia, COPD, asthma, adult onset hearing loss, and chronic back pain (which we also applied to slipped disc). For each of the remaining cancer categories, we derived attributable fractions from a study carried out for the National Institute of Occupational Health and Safety (Kerr et al. 1996). This study also provided attributable fractions for a number of other chronic diseases, including neurological disorders, cardiovascular diseases, chronic respiratory diseases and renal disease. Attributable fractions for osteoarthritis were derived separately, based on relative risks of self-reported arthritis for blue collar workers compared to managers, administrators and professionals (AIHW: Turrell et al. 2006).

Child sexual abuse and intimate partner violence

Girls that experience child sexual abuse are more likely to experience intimate partner violence than non-abused girls (Mouzos & Makkai 2004). Women that experience multiple types of abuse, including child sexual abuse and intimate partner violence, have a higher risk of depression than those subject to only one form of abuse (Arias 2004; Messman-Moore et al. 2000; Nicolaidis et al. 2004). The 2001 Victorian Burden of Disease Study produced estimates of the burden attributable to intimate partner violence but did not calculate the burden attributable to child sexual abuse (DHS 2005; Vos et al. in press). Conversely the CRA project (Andrews et al. 2004) produced estimates of the burden attributable to child sexual abuse but not intimate partner violence. In this study we estimated the burden attributable to child sexual abuse and intimate partner violence. Further, to avoid over-estimating the burden when both of these risk factors are present we estimated an adjusted relative risk to account for the combined exposure state of having experienced both child sexual abuse and intimate partner violence.

We estimated the prevalence of 'intimate partner violence without child sexual abuse' and 'child sexual abuse and intimate partner violence combined' from the Women's Safety Survey (ABS 1996). We used two categories of exposure to intimate partner violence, namely physical or sexual violence by a partner in the last 12 months, and physical or sexual violence by a partner more than 12 months ago. Given that the Women's Safety Survey asks only one question regarding child sexual abuse ('Whether experienced sexual abuse when a child') we used the CRA project priors for Australia for the prevalence of child sexual abuse (based upon epidemiological studies) and assumed no trend in prevalence of child sexual abuse. We subtracted the prevalence of 'child sexual abuse and intimate partner violence combined' from the child sexual abuse priors to estimate the prevalence of child sexual abuse without intimate partner violence.

Messman-Moore and colleagues (2000) looked at the mean psychological functioning indices for women who had experienced (a) both contact child sexual abuse and adult victimisation (revictimisation); (b) adult victimisation only (multiple or once only); (c) contact child sexual abuse only; and (d) no abuse history. From these group means and standard errors we calculated an effect size using Hedges' adjusted *g* for standardised mean difference (Egger et al. 2001). We then converted the effect sizes into odds ratios for risk of depression, anxiety and post-traumatic stress disorder by exposure group using the methods described by Hasselblad and Hedges (1995).

These odds ratios, along with relative risks for contact child sexual abuse from the CRA project (Andrews et al. 2004), and relative risks for intimate partner violence from the Women's health Australia study (see DHS 2005: page 29) were then used to derive relative

risks for 'contact child sexual abuse only', 'intimate partner violence only', and 'child sexual abuse and intimate partner violence combined'.

Since Messman-Moore and colleagues (2000) define child sexual abuse as contact only, for non-contact child sexual abuse we used the CRA project relative risks and prevalence for that category unadjusted. In our main results we combined anxiety and depression together into one category. We therefore found the mean relative risk from the derived relative risks for depression, anxiety, and post-traumatic stress disorder symptoms. We applied the same relativities from the anxiety and depression relative risks for child sexual abuse only, intimate partner violence only, and combined child sexual abuse and intimate partner violence, to the intimate partner violence and child sexual abuse relative risks for other conditions (alcohol use disorders, other drug use disorders, and self-inflicted injuries).

For ease of reporting, the population attributable fraction calculated for the 'combined child sexual abuse and intimate partner violence category' was proportionately redistributed to either child sexual abuse or intimate partner violence. To calculate the population attributable fractions for those disease categories that only apply to intimate partner violence (smoking, cervical cancer, sexually transmitted diseases, eating disorders and physical injuries) we used the relative risks for intimate partner violence from the Women's health Australia study, and the prevalence of intimate partner violence (including those women who may have also experience child sexual abuse) from the Women's Safety Survey. The proportion of homicide due to intimate partner violence (52%) was from the 2003–2004 National Homicide Monitoring Program Annual Report (Mouzos 2005). Violence YLD was based on the proportion of hospitalisations for assaults where the relationship of the victim of assault to the perpetrator was recorded as spouse or domestic partner (including ex-spouse and ex-partner). The assaults where this relationship was unspecified were proportionately redistributed.

Due to a lack of data on the prevalence of intimate partner violence among males, and on the related health outcomes, for males we only estimated the burden due to child sexual abuse. Analyses were based on methods developed for the CRA project described elsewhere (Andrews et al. 2004). We used the CRA priors for Australia for the prevalence of male child sexual abuse (based upon epidemiological studies).

Urban air pollution

Numerous studies have documented that urban air pollution has a range of effects on health, from irritated eyes to death. The effects of short-term exposure are generally demonstrated through time-series studies on daily events (for example mortality, hospitalisations, emergency department attendance) (Cohen et al. 2004; Simpson et al. 2005a, 2005b). The effects of long-term exposure have been demonstrated in large cohort and cross-sectional studies, mainly in the US and Europe (Cohen et al. 2004; Pope et al. 2002). We estimated the burden due to both long- and short term exposure to urban air pollution, and present the results for long-term exposure only as a minimum estimate, and the combination of long- and short-term exposure as a more inclusive but less certain higher estimate.

Long-term exposure

For chronic exposure to urban air pollution, our analyses are based on methods developed for the CRA project (Cohen et al. 2004). The main data inputs were: (a) annual 24-hour average particulate matter concentrations (particulate matter with an aerodynamic diameter

of less than 10 and 2.5 micrometres, PM₁₀ and PM_{2.5}) as an indicator of exposure to pollution from combustion sources; and (b) information on the relative risk of mortality. In the CRA method, the population attributable fraction was calculated from these inputs as the difference in disease experience in a population and the hypothetical disease experience if the population were exposed to the hypothetical minimum of particulate matter (PM_{2.5} 7.5µg/m³; PM₁₀ 15µg/m³). However, there is evidence that there may be no safe level of exposure to particulate matter (WHO Europe 2004). We therefore set the theoretical minimum to zero in our analyses.

Our estimates for long-term exposure are based on the contributions of two health outcomes: cardiopulmonary disease and lung cancer in adults aged 30 years and older. Attributable burden was estimated using risk coefficients from a large cohort study of adults in the United States (Pope et al. 2002). We did not use the CRA method of attributing acute respiratory infection in children aged 0–4 years as this method applies a relative risk based on daily exposure to annual exposure levels. Given the availability of daily urban air pollution data in Australia, and more appropriate relative risk estimates from Australian pollution concentration and mortality data, we used the estimates generated using the short-term effects methods described below.

We based exposure on annual mean levels for 2002 in the following urban areas: Sydney, Newcastle, Wollongong, Melbourne, Geelong, Brisbane, Perth, Adelaide, Canberra (including Queanbeyan), and Hobart. Annual concentrations were derived from data supplied by the state and territory environmental protection authorities, except for Adelaide and Hobart where we used published estimates (DPIWE 2004; Gooding & Riordan 2004). PM_{2.5} concentration was not available for Geelong, Hobart or Canberra. For Geelong, we estimated the concentration from Melbourne's PM₁₀:PM_{2.5} ratio. For Hobart and Canberra, we based our estimates on the average PM₁₀:PM_{2.5} ratio for those cities with original data (that is, Brisbane, Melbourne, Perth, Sydney, Adelaide, Newcastle and Wollongong). Due to temporal trends in particulate matter concentration, the linking of current exposure to chronic outcomes may underestimate the attributable burden if exposure levels were higher in the past. However, the use of recent exposure data is in keeping with the CRA methods.

Short-term exposure

Short-term exposure to urban air pollution has been associated with day-to-day variations in hospital admissions and mortality (Simpson et al. 2005a, 2005b). However, translating these findings into burden of disease estimates is not straightforward. The difficulty with estimating attributable morbidity is that published risks are established for the impact on hospitalisations only. An increase in hospitalisations for causes related to urban air pollution is likely to largely reflect exacerbation of existing disease rather than new disease events. Our YLD estimates are based on incident cases and their average duration at a particular level of severity. Thus the impact of urban air pollution on morbidity needs to be estimated as either a proportion of new cases of disease or a worsening of the condition for an undefined period of time. Until these methodological issues can be resolved we consider only a mortality component of the short-term health consequences of urban air pollution.

The problem with attributing mortality to the short-term impact of urban air pollution is that there is equivocal evidence regarding the extent of 'harvesting', that is, imminent deaths brought forward by only a short period of time (less than a month) that were imminent anyway, or 'new deaths' that would not have occurred in the absence of urban air pollution. This has a major bearing on our estimates of YLL: if harvesting occurs, YLL will be only a

fraction of that normally calculated for each death. There is much debate in the literature on this topic. There are some arguments that harvesting does not play a role in the effects of urban air pollution. For instance there is an increase in deaths when longer lags between exposure and outcomes (up to 4 months) of urban air pollution are estimated, rather than a decrease (Schwartz 2001; Zeger et al. 1999). (The need to control for seasonal variation in these analyses makes it difficult to extend these analyses over the longer term as longer lags become strongly correlated with seasonal changes). This finding has been interpreted to indicate that harvesting is not an important issue. However, it could also be the case that urban air pollution exposure leads to chronic rather than acute effects on mortality. A further argument put forward by the same authors is that the largest increase in deaths was seen in people dying outside a hospital, while one would have expected a greater increase in hospital deaths if harvesting were bringing deaths forward in people who were already ill. The authors do not comment, however, on whether this may be due to the protective effect of the hospital environment. We concluded that there is no consensus on the relative contribution of deaths brought forward by urban air pollution nor on the size of the true acute impact on mortality. We therefore present the chronic impact as a lower estimate of the burden due to urban air pollution and add an alternative estimate of the combined short-term and long-term effects, ignoring any harvesting, as an upper bound.

Recent Australian research has provided the most applicable risk coefficients describing the effect of short-term exposure to urban air pollution on mortality (Simpson et al. 2005b). We applied these to daily urban air pollution data to estimate the attributable mortality burden of this risk. Following expert advice, our estimates were based on an averaged 0–1 day lag (that is, exposure to urban air pollution on the day of death and the day before death) of the contributions of two pollutants to two causes of death: all cause mortality (excluding accidental and other external causes of death) due to particle exposure (in units of light scattering by nephelometry, bsp), and respiratory deaths due to exposure to ozone. The choice of including these two pollutants and excluding others was made after discussion with the researchers (Simpson, Williams and Barnett) and justified by the finding that the impact on mortality of NO₂, CO and particles largely overlaps and hence including all three would lead to overestimation. The impact of SO₂ is considered small in Australia but ozone has a significant impact on respiratory mortality independent of that of other pollutants.

Estimates were calculated with a theoretical minimum exposure level of zero. This is based on evidence that at the population level there appears to be no safe level of exposure to particles or ozone (WHO Europe 2004).

A decision was made to work with exposure data from 2002 rather than 2003 (the reference year for our study) because 2003 is considered an outlier year by the environmental protection authorities for pollutant readings. Daily urban air pollution data were supplied by the Victorian, New South Wales, Australian Capital Territory, Queensland and Western Australian environmental protection authorities. We calculated a PAF for each day by urban area, pollutant, and underlying cause of death, with the assumption that the entire population of that urban area was exposed. This was applied to daily 2002 mortality data, and aggregated to age- and sex-specific annual PAFs. We aggregated the number of deaths and YLL attributable to urban air pollution in specific areas (Sydney, Newcastle, Wollongong, Melbourne, Geelong, Brisbane, Perth, Adelaide, Canberra including Queanbeyan, and Hobart), calculated this as a proportion of all deaths or YLL in Australia and, finally, applied this proportion to 2003 mortality estimates.

We did not gain access to daily Tasmanian or South Australian urban air pollution data. Particle levels for Adelaide and Hobart were therefore extrapolated from published annual mean PM₁₀ levels (Air Monitoring Unit, EPA SA 2003; DPIWE 2004), and the average ratio of bsp:PM₁₀ for Brisbane, Sydney and Melbourne from Simpson and colleagues (2005b). The ratio of the extrapolated mean bsp for Adelaide and Hobart to the annual mean bsp level for the cities for which we had detailed exposure data was then applied to the annualised PAF for these cities to extrapolate the PAFs for the two cities with missing exposure data. Ozone levels for Adelaide were based on the published average for 2002 (Gooding & Riordan 2004). Ozone is not routinely monitored in Hobart (DPIWE 2006); we therefore did not include this region in our analysis of respiratory deaths due to ozone exposure.

Unsafe sex

All sexually transmitted diseases were attributed to unsafe sex. The PAFs for HIV/AIDS and hepatitis B and C due to unsafe sex were derived as described in the section on illicit drugs. Previous Australian and Victorian burden of disease studies have used a PAF of 0.90 for cervical cancer. In this study we attributed all cervical cancer to sexual transmission of the human papilloma virus. Munoz and colleagues (2003) found that 90.7% cases had HPV DNA detected. Similarly, in a meta-analysis Clifford and colleagues (2003) found that HPV DNA was present in 80–89% of cases. However, research by Walboomers and colleagues (1999), in which they revisited a previous study, suggests that nearly all cases that were negative for HPV DNA were false negatives. They revised up the estimates of cases testing positive for HPV DNA from 93% to 99.7%. Bosch and Munoz (2002) suggest that in most studies where 5–15% of cases are negative for HPV these are false negatives.

Osteoporosis

Osteoporosis causes no disability or death per se; it does, however, increase the risk of fracture. Therefore we treated osteoporosis as a risk factor in this study rather than as a disease in its own right, as was done in the previous Australian burden study. The WHO Task-Force for Osteoporosis recommends that the condition be defined by level of bone mineral density (BMD). We therefore based our PAF calculations on the population distribution of BMD, and relative risks associated with decreasing BMD.

In Australia there are two large studies that have measured population BMD, one based in Geelong and the other in Dubbo. Both the Geelong Osteoporosis Study and the Dubbo Osteoporosis Epidemiology Study state that the population they cover is representative of the Australian population (Nguyen et al. 2001; Sanders et al. 1998). Mean BMD and standard deviations (SDs) for the Geelong and Dubbo studies were supplied by the study custodians. We used Geelong data for ages <60 and combined Geelong and Dubbo data for 60 years or over by fitting a Weibull distribution. From this distribution we plotted BMD by age for ages 25 years or over and fitted a polynomial distribution ($R^2=0.998$). We then predicted mean BMD from this equation for 5-year age groups from 60 years.

For males, we assumed that the difference between the Dubbo and Geelong BMD means for women would also apply to males if Geelong data were available. We therefore increased Dubbo means by the ratio of female Dubbo sampled mean to the combined mean. We assumed deviations from the line were sampling error, and predicted mean BMD by age group from the fitted quadratic equation ($R^2=0.925$). We assumed the SD for Dubbo applied.

The WHO Task-Force for Osteoporosis recommends that the condition be defined in Caucasian women as a BMD 2.5 SDs or more below the young female reference mean (Genant et al. 1999). The Australia and New Zealand Bone and Mineral Society and Osteoporosis Australia recommended that data from the Geelong Osteoporosis Study be used to establish a standardised reference range for Australia (Henry et al. 2004). We therefore used the mean BMD and SD for young women aged 20–29 from this study as the theoretical minimum, and also used this population for the osteoporosis cut-off (Henry et al. 2004).

There is currently no Australian reference mean BMD and SD for young adult men. We estimated these values by multiplying the Australian young female mean and SD (Henry et al. 2004) by the ratio of male to female mean and SD from the USA's National Health and Nutrition Examination Survey (NHANES) (Looker et al. 1998). The NHANES used Hologic densitometers while both the Dubbo and Geelong studies used Lunar densitometers. These machines do not give standardised results. We therefore converted the Hologic estimates to Lunar by applying the formula available at <www.courses.washington.edu/bonephys/opBMDs.html>.

Relative risks and odds ratios from a number of studies were pooled to estimate the relative risk of low impact fracture per 0.1g/cm² decrease in BMD measured at the femoral neck (EPOS Group 2002; Fujiwara et al. 2003; Kroger et al. 1995; Nguyen et al. 2005a, 2005b; Papaioannou et al. 2005; Schott et al. 2005; Schuit et al. 2004; Stone et al. 2003). Where a study used Hologic or Norland densitometers, and the relative risk was per SD change in BMD, we converted the study's SD estimates to Lunar.

We derived PAFs for a number of fracture sites. Where possible these sites were linked directly to a single nature of injury category. In some cases (for example hip) we applied the PAF to a proportion of a category based on the distribution of fracture sites in the National Hospital Morbidity Database 2002–03. Since most studies that we included in the calculation of relative risks excluded fractures resulting from high impact causes, we applied the PAFs to fractures resulting from falls, striking and crushing accidents, and other unintentional injuries.

For attributable YLL, we applied the site-specific fracture YLD PAFs to the site-specific mortality distribution for vertebral, pelvis and femur fracture to derive a site-specific YLL PAF. This was applied to deaths with an underlying cause of falls, striking and crushing accidents, other unintentional injuries, ill-defined falls or osteoporosis, where a fractured spine, pelvis or femur was mentioned. If more than one fracture was mentioned we applied the larger PAF, that is, for fractured pelvis and femur we applied the PAF for femur. We assumed that all deaths with an underlying cause of osteoporosis but no mention of vertebral, pelvis, or femur fracture, were attributable to osteoporosis. To determine the burden of disease code-specific YLL PAF for osteoporosis we calculated the proportion of burden of disease code-specific deaths attributable to osteoporosis. Osteoporosis and ill-defined fall deaths were redistributed to falls. If we were to limit the deaths attributable to osteoporosis to only those that were coded to osteoporosis, the overall number of deaths would have been considerably smaller.

Table A2.3: Definitions, theoretical minima, health outcomes and data sources for 14 selected health risks

Health risk	Exposure variable	Theoretical minimum	Outcomes	Sources for exposure estimates	Sources for hazard estimates
High blood pressure	Level of usual systolic blood pressure	115 (SD 6) mmHg	Ischemic heart disease, stroke, hypertensive heart disease	AusDiab study (Dunstan et al. 2002)	Meta-analysis of 61 cohort studies with 1,000,000 North American and European participants (Prospective Studies Collaboration (Lawes et al. 2003))
High blood cholesterol	Level of usual total blood cholesterol	3.8 (SD 0.6) mmol/L (147 (SD 23) mg/dL)	Ischemic heart disease, ischemic stroke	AusDiab study (Dunstan et al. 2002)	Meta-analysis of 10 cohorts with 490,000 North American and European participants, and 29 cohorts with 350,000 participants from the Asia-Pacific region
High body mass index (BMI)	Body mass index, BMI (weight over height squared)	21 (SD 1) kg/m ²	Ischemic heart disease, stroke, hypertensive heart disease, diabetes, osteoarthritis, endometrial cancer, kidney cancer, colon cancer, post-menopausal breast cancer	AusDiab study (Dunstan et al. 2002)	Meta-analysis of 33 cohorts with 310,000 participants for cardiovascular disease risks, 27 cohorts for cancer risks, and systematic review of cohort studies for diabetes risk
Low fruit and vegetable consumption	Fruit and vegetable intake per day	600 (SD 50) g intake per day for adults	Ischemic heart disease, stroke, colorectal cancer, gastric cancer, lung cancer, oesophageal cancer	National Health Survey 2001 (ABS 2001c)	Systematic review and new meta-analysis of published cohort studies
Osteoporosis	Bone mineral density of the femoral neck	Males 1.107 (SD 0.140) g/cm ² , Females 1.018 (SD 0.127) g/cm ² With osteoporosis defined 2.5 or more SD below this mean	Fractured hip, femur, humerus, clavicle, forearm/wrist, elbow, spine, rib, pelvis, lower leg, patella, foot, heel, toe, hand, finger from falls, striking and crushing accidents, other unintentional injuries	Dubbo Osteoporosis Epidemiology Study (Nguyen 2005) and Geelong Osteoporosis Study (Kotowicz 2005)	Pooled analysis of 10 studies (EPOS Group 2002; Fujiwara et al. 2003; Kroger et al. 1995; Nguyen et al. 2005a, 2005b; Papaioannou et al. 2005; Schott et al. 2005; Schuit et al. 2004; Stone et al. 2003)

(continued)

Table A2.3 (continued): Definitions, theoretical minima, health outcomes and data sources for 14 selected health risks

Health risk	Exposure variable	Theoretical minimum	Outcomes	Sources for exposure estimates	Sources for hazard estimates
Physical inactivity	Four categories: inactive, insufficient, recommended level and highly active	All in 'highly active' group	Ischemic heart disease, stroke, breast cancer, colon cancer, diabetes	National Health Survey 2001 (ABS 2001c)	Systematic review of published literature and new meta-analysis of cohort studies
Tobacco	Past smoking	No smoking	COPD, cancers of mouth, oesophagus, lung, pancreas, larynx, bladder, kidney, stomach and uterus	Peto-Lopez method	Systematic reviews by English and colleagues (1995) and Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001)
	Current daily smokers	No smoking	Ischemic heart disease, stroke, peripheral vascular disease, Parkinson's disease, pneumonia (adults), fire injuries, macular degeneration	National Health Survey 2001 (ABS 2001c)	Systematic reviews by English and colleagues (1995) and Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001); Tomy and colleagues (2004) for age related macular degeneration
	Passive smoking	No smoking	Ischemic heart disease, stroke	National Health Survey 1995 (ABS 1995)	Systematic reviews by English and colleagues (1995) and Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001)
	Maternal smoking; smoking while pregnant	No smoking	Asthma, pneumonia (children), sudden infant death syndrome, otitis media, low birth weight	National Health Survey 2001 (ABS 2001c), <i>Australia's Mothers and Babies 2003</i> (AIHW: Laws & Sullivan 2005)	Systematic reviews by English and colleagues (1995) and Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001); US Surgeon General's Report on Involuntary exposure to tobacco smoke (US Department of Health and Human Services 2006); systematic review by Anderson and Cook (1997).

(continued)

Table A2.3 (continued): Definitions, theoretical minima, health outcomes and data sources for 14 selected health risks

Health risk	Exposure variable	Theoretical minimum	Outcomes	Sources for exposure estimates	Sources for hazard estimates
Alcohol	Average number of standard drinks per day	Low level of drinking	Cancers of the mouth and oropharynx, oesophagus, liver, larynx and breast; inflammatory heart disease, hypertensive heart disease, ischemic heart disease, stroke, alcohol dependence and harmful use, gallbladder and bile duct disease, pancreatitis, road traffic accidents, falls, fires/burns/scalds, drowning, machinery accidents, suffocation and foreign bodies, suicide and self-inflicted injuries, homicide and violence, occupational injuries	National Health Survey 2001 (ABS 2001c)	Systematic reviews by English and colleagues (1995) and Ridolfo & Stevenson (AIHW: Ridolfo & Stevenson 2001); the National Coroners Information System (Driscoll et al. 2001, 2004) for alcohol-related drownings and occupational YLL; fire injuries and fatalities pooled results from published studies; scalds and burns from Levy and colleagues (2004)
Illicit drug use	Use of illicit drugs	Abstinence	Heroin or polydrug, benzodiazepine, cannabis, and other drug dependence and harmful use	AusBoD drug use and dependence models	PAF = 1 by definition
	Use of illicit drugs	Abstinence	HIV/AIDS, hepatitis B, hepatitis C, inflammatory heart disease, suicide and self-inflicted injuries, road traffic accidents	Population attributable fraction direct from the literature	Incorporated findings from a multi-centre case-control study on 3,398 fatally injured drivers over Victoria, NSW and Queensland (examining psychoactive drugs); viral hepatitis and sexually transmissible infections from <i>Australia Annual Surveillance Report 2004</i> ; and systematic literature reviews by English and colleagues (1995) and Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001)
	Daily cannabis use	No cannabis use, or use less often than daily	Schizophrenia	National Drug Strategy Household Survey 2004	Meta-analysis of 7 published case-control or cohort studies (examining link between psychosis and cannabis use).

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Table A2.3 (continued): Definitions, theoretical minima, health outcomes and data sources for 14 selected health risks

Health risk	Exposure variable	Theoretical minimum	Outcomes	Sources for exposure estimates	Sources for hazard estimates
Unsafe sex	Unprotected sex	Abstinence/protection	Sexually transmissible diseases, abortion, cervical cancer, HIV/AIDS, hepatitis B & C	AusBoD sexually transmissible diseases, abortion, and cervical cancer models; PAF direct from the literature	PAF=1 (sexually transmissible diseases, abortion, cervical cancer); HIV/AIDS proportion from the Australian HIV and AIDS Public Access Datasets (National Centre in HIV Epidemiology and Clinical Research 2005a, 2005b); hepatitis B & C fraction from the National Centre in HIV Epidemiology and Clinical Research (National Centre in HIV Epidemiology and Clinical Research 2004)
Child sexual abuse	Non-contact only, contact intercourse	No abuse	Anxiety & depression, alcohol dependence & harmful use, heroin or polydrug use & dependence, benzodiazepine dependence & harmful use, cannabis dependence & harmful use, other drug dependence & harmful use, suicide and self-inflicted injuries	CRA priors for Australia	Systematic review and new meta-analysis of published studies (Andrews et al. 2004)
Intimate partner violence	Physical or sexual violence by current or previous partner	No history of sexual or physical violence by an intimate partner	Anxiety & depression, alcohol dependence & harmful use, heroin or polydrug use & dependence, benzodiazepine dependence & harmful use, cannabis dependence & harmful use, other drug dependence & harmful use, suicide and self-inflicted injuries, tobacco smoking, cervical cancer, syphilis, chlamydia, gonorrhoea, other sexually transmissible diseases, anorexia nervosa, bulimia nervosa, other eating disorders, falls, other unintentional injuries, homicide & violence	Women's Safety Survey 1996 (ABS 1996)	Australian Longitudinal Study on Women's Health (Brown et al. 1999); 2003–2004 National Homicide Monitoring Program (NHMP) Annual Report (Mouzos 2005); National Hospital Morbidity Database 2002–03 (AIHW 2003a)

(continued)

Table A2.3 (continued): Definitions, theoretical minima, health outcomes and data sources for 14 selected health risks

Health risk	Exposure variable	Theoretical minimum	Outcomes	Sources for exposure estimates	Sources for hazard estimates
Occupational exposures and hazards	Exposure in the workplace to disease-causing agents such as carbon monoxide, dyes, inorganic and organic dusts, pesticides, metals, metal fumes, petrochemicals, plastics, solvents, isocyanate and nitroglycerine or nitroglycerol	No exposure	All accidents, intentional and unintentional injuries, cancers, heart disease, neurological disorders, chronic respiratory disorders, renal disease, osteoarthritis, slipped disc, occupational overuse syndrome	National Worker's Compensation Statistics Database 2003, National Coroners Information system 2003, and Best estimates of the magnitude of health effects of occupational exposure to hazardous substances (Kerr et al. 1996)	Systematic review of published literature, hospital inpatient data, mortality datasets, National Health Survey results, workers compensation data, notified industrial accident reports and special disease registry datasets
Urban air pollution	Exposure to particulate matter and/or oxygen (i.e. total population of cities of interest)	No exposure	Short-term exposure: cardiovascular, respiratory, and other deaths Long-term exposure: lung cancer, ischemic heart disease, stroke, inflammatory heart disease, hypertensive heart disease, COPD	Assume all residing in relevant geographical areas exposed; particulate and ozone levels from state environmental protection agencies	Time series analysis of short-term effects of urban air pollution in four Australian cities (Simpson et al. 2005a); long-term exposure effects from Pope and colleagues (2002)

Table A2.4: Prevalence of health risks by age and sex

Health risk	Category	Males							Females								
		0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+
Blood pressure (mmHg)	mean	124	131	140	148	154	115	126	138	146	150
	SD	11	16	17	19	19	12	17	19	22	21
Blood cholesterol (mmol/L)	mean	5.5	5.8	5.6	5.6	5.3	5.2	5.8	6.0	6.1	5.9
	SD	1.0	1.1	0.9	0.9	1.0	1.0	1.1	0.9	1.0	1.0
BMI (kg/m ²)	mean	26.8	27.5	27.2	27.1	25.8	25.4	27.2	28.5	27.0	24.9
	SD	4.1	4.0	3.7	3.8	3.5	5.4	5.7	5.8	5.2	4.5
Fruit and vegetable consumption (g/day)	mean	445	452	496	538	538	538	484	506	569	602	577	577
	SD	241	235	245	230	219	219	237	228	240	234	217	217
Bone mineral density (BMD) (g/cm ³)	Mean	0.93	0.87	0.77	0.85	0.78	0.66
	SD	0.15	0.14	0.16	0.13	0.12	0.12
Physical activity (% population in categories)	High	10%	3%	3%	1%	1%	0%	4%	2%	1%	1%	0%	0%
	Recommended	47%	37%	37%	41%	44%	30%	37%	32%	35%	38%	27%	17%
Tobacco (% population in categories)	Insufficient	23%	29%	29%	26%	22%	21%	35%	38%	33%	28%	28%	24%
	Inactive	20%	31%	32%	33%	33%	49%	25%	28%	30%	33%	45%	59%
Tobacco (% population in categories)	Current smoker	30%	31%	23%	16%	9%	7%	25%	25%	18%	12%	9%	2%
	Prenatal exposure	16%	16%
Alcohol (% population in categories)	Maternal smoking	27%	27%
	Abstainer	37%	35%	33%	43%	49%	56%	57%	59%	58%	66%	73%	76%
Alcohol (% population in categories)	Low	48%	51%	52%	43%	45%	40%	35%	32%	32%	25%	20%	22%
	Hazardous	7%	7%	8%	8%	4%	2%	7%	7%	7%	7%	6%	2%
Alcohol (% population in categories)	Harmful	7%	7%	7%	6%	2%	2%	1%	2%	3%	2%	1%	0%

(continued)

Table A2.4 (continued): Prevalence of health risks by age and sex

Health risk	Category	Males										Females									
		0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+				
Illicit drugs (% population in categories)	Daily cannabis use	4%	4%	1%	0%	0%	0%	2%	2%	0%	0%	0%	0%				
	Prenatal exposure – opioids	0%	0%				
Prenatal exposure – cannabis	Prenatal exposure – cannabis	1%	1%				
	Maternal use – heroin				
Maternal use – cocaine	Maternal use – cocaine				
	No abuse	100%	96%	96%	94%	94%	94%	94%	94%	98%	79%	79%	71%	71%	71%	71%	71%				
Child sexual abuse (% population in categories)	Non-contact only	0%	1%	1%	2%	2%	2%	2%	2%	1%	6%	6%	9%	9%	9%	9%	9%				
	CSA	0%	2%	2%	3%	3%	3%	3%	3%	1%	11%	12%	16%	16%	16%	16%	16%				
Intimate partner violence (% population in categories)	Intercourse CSA	0%	1%	1%	1%	1%	1%	1%	1%	0%	3%	3%	5%	5%	5%	5%	5%				
	Sexual or physical violence	22%	21%	10%	10%	10%				
Occupational exposure to ergonomic stressors (% population in categories)	Low	14%	9%	8%	3%	1%	1%	34%	25%	22%	5%	1%	1%				
	Moderate	44%	41%	35%	13%	3%	3%	18%	17%	18%	4%	1%	1%				
Occupational exposure to ergonomic stressors (increases risk of osteoarthritis) (% population in categories)	High	2%	3%	4%	4%	2%	2%	0%	0%	0%	0%	0%	0%				
	Blue collar workers	40%	40%	34%	12%	3%	3%	8%	9%	10%	3%	1%	1%				

(continued)

Table A2.4 (continued): Prevalence of health risks by age and sex

Health risk	Category	Males										Females									
		0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+				
Occupational exposure to noise (% population in categories)	85-90 dBA	5%	5%	5%	2%	1%	1%	4%	3%	3%	1%	0%	0%				
	>90 dBA	4%	4%	3%	1%	0%	0%	1%	1%	1%	0%	0%	0%				
Occupational exposure to leukaemogens (% population in categories)	Low	3%	4%	4%	1%	0%	0%	3%	4%	4%	1%	0%	0%				
	High	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%				
Occupational exposure to lung carcinogens (% population in categories)	Low	21%	28%	23%	8%	2%	2%	6%	9%	7%	2%	0%	0%				
	High	2%	3%	3%	1%	0%	0%	1%	1%	1%	0%	0%	0%				
Occupational exposure to agents causing COPD (% population in categories)	Low	18%	27%	25%	11%	4%	4%	7%	11%	10%	3%	1%	1%				
	High	12%	14%	11%	3%	1%	1%	1%	2%	2%	0%	0%	0%				
Occupational exposure to agents causing asthma (% population in categories)	Background	23%	10%	17%	67%	90%	90%	31%	28%	33%	84%	97%	97%				
	Administration	6%	12%	13%	4%	1%	1%	16%	21%	18%	4%	1%	1%				
Technical	Technical	16%	30%	28%	10%	3%	3%	16%	27%	24%	5%	1%	1%				
	Sales	10%	4%	3%	2%	0%	0%	19%	6%	5%	1%	0%	0%				
Agriculture	Agriculture	2%	3%	4%	4%	2%	2%	0%	1%	1%	1%	0%	0%				
	Mining	1%	1%	1%	0%	0%	0%	0%	0%	0%	0%	0%	0%				
Transport	Transport	6%	8%	9%	3%	1%	1%	1%	1%	1%	0%	0%	0%				
	Manufacturing	31%	28%	21%	7%	1%	1%	6%	6%	6%	1%	0%	0%				
Services	Manufacturing	6%	4%	4%	2%	1%	1%	11%	10%	11%	3%	1%	1%				
	Services	6%	4%	4%	2%	1%	1%	11%	10%	11%	3%	1%	1%				

Annex tables

Annex Table 1: Disease and injury categories and ICD-10 codes

Cause	ICD-10 codes
I. Communicable diseases, maternal and neonatal conditions	
A. Infectious and parasitic diseases	
1. Tuberculosis	A15-19;B90;K230,673,930;M011,490,900;N330,7401;O980;P370
2. Sexually transmitted diseases ^(a)	
a. Syphilis	A50-53;I980;K672;M031,731;N290,742
b. Chlamydia	A56;K670;N744
c. Gonorrhoea	A54;K671;M730;N743;O982
d. Other sexually transmitted diseases	A55,57-64
3. HIV/AIDS	B20-24;F024
4. Diarrhoeal diseases	A00-09
5. Childhood immunisable diseases	
a. Diphtheria	A36
b. Whooping cough	A37
c. Tetanus	A33-35
d. Poliomyelitis	A80;B91
e. Measles	B05
f. Rubella	B06;M014;P350
g. <i>Haemophilus influenzae</i> type b (Hib)	A413,492;G000;J051,14,201
6. Meningitis	A39;G001-9,03
7. Septicaemia	A40,410-2,414-8
8. Arbovirus infection	
a. Ross River virus	B331
b. Barmah Forest virus	A92.8
c. Dengue	A90-91
d. Other arbovirus infection	A83-84,852,92-99
9. Hepatitis	
a. Hepatitis A	B15
b. Hepatitis B ^(b)	B16,170,180-1
c. Hepatitis C ^(c)	B171,182
d. Other hepatitis	B172-8,188-9,19;P353
10. Malaria	B50-54
11. Trachoma	A71;B940
12. Other infectious and parasitic diseases	A20-32,38,42-48,490-1,493-9,65-70,74-79,81-82,850-1, 858, 86-89;B00-04,07-09,25-30,330,332-8,34-49,55-89,92 (excluding 92.8),941, 948-9,95-99;G01-02,04-07;K231;M00,010,012-3,015-8, 030
B. Acute respiratory infections	
1. Lower respiratory tract infections	J10-13,15-18,200,202-9,21-22
2. Upper respiratory tract infections	J00-04,050,06
3. Otitis media	H65-66
C. Maternal conditions	
1. Maternal haemorrhage	O441,45-46,67,72
2. Maternal sepsis	O411,85-86
3. Hypertensive disorders of pregnancy	O10-16
4. Obstructed labour	O64-66,711,713
5. Abortion	O00-08

(continued)

Annex Table 1 (continued): Disease and injury categories and ICD-10 codes

Cause	ICD-10 codes
6. Other maternal conditions	O09,20-24,26-40,410,418-9,42-43,440,47-63,68-70, 710,712, 714-9,73-82,87-97,981,983-9,99
D. Neonatal causes	
1. Birth trauma and asphyxia	P03,10-21,24-28
2. Low birthweight	P05-07,22
3. Neonatal infections	P23,351-2,358-9,36,371-9,38-39
4. Other conditions arising in the perinatal period	P04,08,29,50-96
E. Nutritional deficiencies	
1. Protein-energy malnutrition	E40-45,640;M833;O25
2. Deficiency anaemia	D50-53
3. Other nutritional deficiencies	E00-02,031,50,51-1,518-9,52-61,630-8,641-9
II. Non-communicable diseases	
F. Malignant neoplasms	
1. Mouth and oropharynx cancers	C00-14
2. Oesophagus cancer	C15
3. Stomach cancer	C16
4. Colorectal cancer	C18-21
5. Liver cancer ^(d)	C22
6. Gallbladder cancer	C23-24
7. Pancreas cancer	C25
8. Lung cancer	C33-34
9. Bone and connective tissue cancer	C40-41,490-9
10. Melanoma	C43
11. Non-melanoma skin cancers	C44
12. Breast cancer	C50
13. Cervix cancer	C53
14. Corpus uteri cancer	C54
15. Ovary cancer	C56,570-4
16. Prostate cancer	C61
17. Testicular cancer	C62
18. Bladder cancer	C67
19. Kidney cancer	C64-66,68
20. Brain cancer	C71
21. Thyroid cancer	C73
22. Lymphoma	C81-85,96
23. Multiple myeloma	C88-90
24. Leukaemia	C91-95
25. Larynx cancer	C32
26. Eye cancer	C69
27. Other malignant neoplasms	C17,26-31,37-39,45-48,51-52,577-9,58-60,63,70,72,74-75
G. Other neoplasms	
1. Uterine myomas	D25
2. Benign neoplasms of meninges and brain	D32-33
3. Other benign neoplasms	D00-24,26-31,34-48
H. Diabetes mellitus	
1. Type 1 diabetes	E10
2. Type 2 diabetes	E11-13

(continued)

Annex Table 1 (continued): Disease and injury categories and ICD-10 codes

Cause	ICD-10 codes
I. Endocrine and metabolic disorders	
1. Non-deficiency anaemia	
a. Haemolytic anaemia	D55-58
b. Other non-deficiency anaemia	D59-63,640-8
2. Cystic fibrosis	E84
3. Haemophilia	D66-67,681
4. Other endocrine and metabolic disorders	D680,682-9,69-72,730-4,738-9,74-89;E030,032-9,04-07,15-35,65,660-2,67-77,781-4,786-9,79-83,85,873-4,878,88-90;D65,735;E668-9,86,870-2,875-7
J. Mental disorders	
1. Substance use disorders	
a. Alcohol dependence and harmful use ^(e)	E512;F10;G312;X45
b. Heroin or polydrug dependence and harmful use	F11;X42
c. Benzodiazepine dependence and harmful use	F13
d. Cannabis dependence and harmful use	F12
e. Other drug dependence and harmful use	F14-16,18-19
2. Schizophrenia	F20-29
3. Anxiety and depression	F30,32-39,400-1,410-2,42,431,930
4. Bipolar disorder	F31
5. Personality disorders ^(f)	F603
6. Eating disorders	
a. Anorexia nervosa	F500-1
b. Bulimia nervosa	F502-3
c. Other eating disorders	F504-9
7. Childhood conditions	
a. Attention-deficit hyperactivity disorder	F90
b. Autism spectrum disorders	F84
8. Other mental disorders	F05-09, 402-9,413-9,430,432-9,44-48,51-59,600-2,604-9,61-69,80-83,88-89,91-92,931-9,94-99
K. Nervous system and sense organ disorders	
1. Dementia	F00-01,020-1,023,03;G30,310-1,318-9
2. Epilepsy	G40-41
3. Parkinson's disease	G20
4. Multiple sclerosis	G35
5. Motor neurone disease	G122
6. Huntington's chorea	F022;G10
7. Muscular dystrophy	G710
8. Sense organ disorders	
a. Glaucoma-related blindness	H40
b. Cataract-related blindness	H25-27
c. Macular degeneration	H353
d. Adult-onset hearing loss	H90-91
e. Refractive errors	H520-7
f. Other vision loss	H54
9. Migraine	G43
10. Other nervous system and sense organ disorders	F028,04,70-79;G08-09,11,120-1,128-9,13,21-26,32,36-37,44,46-70,711-932,72-92,934-9,94-H22;H28-34,350-2,354-9,36,42-51,53,55-62,67-83,92-95

(continued)

Annex Table 1 (continued): Disease and injury categories and ICD-10 codes

Cause	ICD-10 codes
L. Cardiovascular disease	
1. Rheumatic heart disease	I00-09
2. Ischemic heart disease	I20-25
3. Stroke	G45;I60-69
4. Inflammatory heart disease	I30-33,40-42
5. Hypertensive heart disease	I11,130,15
6. Non-rheumatic valvular disease	I34-39
7. Aortic aneurysm	I71
8. Peripheral vascular disease	I700-8,720-9,73-74
9. Other cardiovascular disease	I26,271,28,43-45,470-1,479,48,491-9,510-4,52,77-84,86-97,981-8,99
M. Chronic respiratory disease	
1. Chronic obstructive pulmonary disease (COPD)	I270,278-9;J40-44
2. Asthma	J45-46
3. Other chronic respiratory diseases	J30-39,47-99
N. Diseases of the digestive system	
1. Peptic ulcer disease	K25-27
2. Cirrhosis of the liver ^(g)	I85;K70,717,721-9,73-74,766-7
3. Appendicitis	K35-37
4. Intestinal obstruction	K400-1,403-4,410-1,413-4,420-1,430-1,440-1,450-8,460-1,56
5. Diverticulitis	K57
6. Gallbladder and bile duct disease	K80-83
7. Pancreatitis	K85,860-1
8. Inflammatory bowel disease	K50-51
9. Vascular insufficiency bowel	K55
10. Other digestive system diseases	K20-22,238,28-31,38,402,409,412,419,429,439,449,469,52,58-66,678,710-6,718-9,720,75,760-5,768-9,77,862-9,87-91,928-9,931-8
O. Genitourinary diseases	
1. Nephritis and nephrosis ^(h)	I12,131;N00-01,03-16,17-19
2. Benign prostatic hypertrophy	N40
3. Urinary incontinence	N393-4
4. Infertility	N46,97
5. Other genitourinary diseases	N02,20-28,291-8,30-32,338-392,34-37,398-9,41-45,47-64,75-96,98-99
P. Skin diseases	
1. Eczema	L20-27
2. Acne	L70
3. Psoriasis	L40
4. Ulcers	L03,088-9,89,97,984
5. Other skin diseases	L00-02,04-05,080-1,10-14,28-30,41-68,71-88,90-95,980-3,985-9,99
Q. Musculoskeletal diseases	
1. Rheumatoid arthritis	M05-06,080,120,465-8
2. Osteoarthritis	M15-19
3. Back pain ⁽ⁱ⁾	M469,47,480-3,488-9,538-9,545-9
4. Slipped disc	M464,50-51,543-4,961
5. Occupational overuse syndrome	
6. Systemic lupus erythematosus (SLE)	M32

(continued)

Annex Table 1 (continued): Disease and injury categories and ICD-10 codes

Cause	ICD-10 codes
7. Gout	M10
8. Other musculoskeletal diseases	M02,032-6,07,081-9,09,11,121-8,13-14,20-31,33-45,460-3,484-5,491-8,530-3,540-2,60-72,738,75-79,830-2,834-9,84-89,901-960,91-95,962-9,99
R. Congenital anomalies	
1. Anencephaly	Q00
2. Spina bifida	Q05
3. Congenital heart disease	Q20-28
4. Cleft lip and/or palate	Q35-37
5. Digestive system malformations	
a. Anorectal atresia	Q42
b. Oesophageal atresia	Q390-1
c. Other digestive system malformations	Q38,392-9,40-41,43-45
6. Urogenital tract malformations	
a. Renal agenesis ⁽ⁱ⁾	Q60
b. Other urogenital tract malformations ^(k)	Q50-56,61-64
7. Abdominal wall defect	Q792-5
8. Down syndrome	Q90
9. Other chromosomal disorders	Q91-99
10. Other congenital anomalies	Q01-04,06-18,30-34,65-78,790-1,796-9,80-89
S. Oral conditions	
1. Dental caries	K02
2. Periodontal disease	K05
3. Edentulism	
4. Pulpitis	K04
5. Other oral conditions	K00-01,03,06-14
Z. Ill-defined conditions	
1. Sudden infant death syndrome	R95
2. Chronic fatigue syndrome	G933;R53
III. Injuries	
T. Unintentional injuries	
1. Road traffic accidents	V011-9,021-9,031-9,041-9,061-9,092-3,104-9,114-9,124-9,134-9,144-9,154-9,164-9,174-9,184-9,194-9,204-9,214-9,224-9,234-9,244-9,254-9,264-9,274-9,284-9,294-9,305-9,315-9,325-9,335-9,345-9,355-9,365-9,375-9,385-9,394-9,405-9,415-9,425-9,435-9,445-9,455-9,465-9,475-9,485-9,494-9,505-9,515-9,525-9,535-9,545-9,555-9,565-9,575-9,585-9,594-9,605-9,615-9,625-9,635-9,645-9,655-9,665-9,675-9,685-9,694-9,705-9,715-9,725-9,735-9,745-9,755-9,765-9,775-9,785-9,794-9,803-5,809,811,821-9,830-3,840-3,850-3,860-4,870-8,892,899;Y85
2. Other transport accidents	V010,020,030,040,05,060,090-1,099,100-3,110-3,120-3,130-3,140-3,150-3,160-3,170-3,180-3,190-3,200-3,210-3,220-3,230-3,240-3,250-3,260-3,270-3,280-3,290-3,300-4,310-4,320-4,330-4,340-4,350-4,360-4,370-4,380-4,390-3,400-4,410-4,420-4,430-4,440-4,450-4,460-4,470-4,480-4,490-3,500-4,510-4,520-4,530-4,540-4,550-4,560-4,570-4,580-4,590-3,600-4,610-4,620-4,630-4,640-4,650-4,660-4,670-4,680-4,690-3,700-4,710-4,720-4,730-4,740-4,750-4,760-4,770-4,780-4,790-3,800-2,806-8,810,812-9,820,834-9,844-9,854-9,865-9,879,88,890-1,893,90-99
3. Poisoning	X40-41,43-44,46-49
4. Falls	W00-19; M80-82
5. Fires, burns and scalds	X00-19

(continued)

Annex Table 1 (continued): Disease and injury categories and ICD-10 codes

Cause	ICD-10 codes
6. Drowning	W65-74
7. Sports injuries	W21;X50
8. Natural and environmental factors	W53-59,64,85-99;X20-39,51-57
9. Machinery accidents	W24,27-31
10. Other unintentional injuries	
Suffocation and foreign bodies	W44, W75-W84
Adverse effects of medical treatment	Y40-Y59, Y60-Y69, Y70-Y84, Y88
Other unintentional injuries n.e.c.	W20, W22-W23, W25-W26, W32-W44, W45, W49, W51, W50, W52, W60, W75-84; X58; Y40-Y59, Y60-Y84, Y86, Y880-Y883
U. Intentional injuries	
1. Suicide and self-inflicted injuries	X60-84;Y870
2. Homicide and violence	X85-Y09;Y871
3. Legal intervention and war	Y35-36,890-1
Redistribution categories	
1. Pelvic inflammatory disease	N70-73,748
2. Unspecified septicaemia	A419
3. Hepatitis sequelae	B942
4. Neonatal causes coded based on maternal condition	P00-02
5. Ill-defined nutritional	E46,639
6. Ill-defined malignant neoplasms	C76-80,97
7. Uterus cancer—unspecified	C55
8. Unspecified diabetes mellitus	E14
9. Other anaemia	D649
10. Smoking listed as cause	F17
11. Hypertensive heart and renal disease	I132-9
12. Heart failure	I50
13. Essential hypertension	I10
14. Ill-defined cardiovascular conditions	E780,785;I46,472,490,515-9,709
15. Gastric haemorrhage	K920-2
16. Ill-defined unintentional accidents (fall if also fracture)	X59;Y90-98
17. Other accidents—intent undetermined	Y20,22-25,28-29,33,34,872,899
18. Road traffic accidents—intent undetermined	Y32
19. Poisoning—intent undetermined	Y10-19
20. Falls—intent undetermined	Y30-31
21. Burns—intent undetermined	Y26-27
22. Drowning—intent undetermined	Y21
23. Ill-defined non-injuries	R00-52,54-94,96-99

Notes

- (a) Excluding HIV/AIDS.
- (b) Including hepatitis B-related liver cancer and cirrhosis.
- (c) Including hepatitis C-related liver cancer and cirrhosis.
- (d) Excluding hepatitis B and C related liver cancer.
- (e) Including alcoholic cirrhosis.
- (f) Excludes those with any other comorbid mental disorders.
- (g) Excluding alcoholic and hepatic cirrhosis.
- (h) Excluding diabetic-, congenital- and poisoning-related renal failure.
- (i) Includes both acute and chronic back pain.
- (j) Including renal failure due to dysplasia.
- (k) Including polycystic renal failure.

Annex Table 2: Principal data sources for epidemiological modelling

Primary data source	Prevalence/ Incidence	Reference period		Disease and injury categories
A. Disease registers, surveillance and notification systems				
National Notifiable Diseases Surveillance System: includes: notifications; and reports: annual report <i>Communicable diseases intelligence</i>	Incidence	2003	A1	Tuberculosis
	Incidence	2003	A2a	Syphilis
	Incidence	2003	A2b	Chlamydia
	Incidence	2003	A2c	Gonorrhoea
	Incidence	2003	A5a	Diphtheria
	Incidence	2000–03	A5b	Pertussis
	Incidence	2003	A5c	Tetanus
	Incidence	2003	A5d	Poliomyelitis
	Incidence	2003	A5e	Measles
	Incidence	2003	A5f	Rubella
	Incidence	2003	A5g	<i>Haemophilus influenzae</i> type B
	Incidence	1993–96	A5g	Hib B sequela
	Incidence	2003	A8	Arbovirus infections
	Incidence	2003	A9a	Hepatitis A
	Incidence	2003	A9b	Hepatitis B
	Incidence	2003	A10	Malaria
HIV/AIDS National Registry	Incidence	2003	A3	HIV/AIDS
National Perinatal Data Collection	Incidence	2003	D2	Low birthweight
Victorian Perinatal Data Collection Unit	Incidence	2001–02	D2	Low birthweight
Queensland Perinatal Data Collection	Incidence	2002	D2	Low birthweight
National Cancer Statistics Clearing House	Incidence	2001	F	Malignant neoplasms
State and territory cancer registries	Incidence	1997	F12	Breast cancer
BreastScreen Australia	Incidence	2001–02, 1997	F12	Breast cancer
National Diabetes Register	Incidence	2001	H	Diabetes mellitus
Australia and New Zealand Dialysis and Transplant Registry	Incidence	2002	H	Diabetes mellitus sequela
	Incidence	2002	O1	Nephritis and nephrosis
Victorian Cystic Fibrosis Screening program	Incidence	1989–1998	I2	Cystic fibrosis
ABS Causes of death data set	Incidence	2003	K5	Motor neurone disease
	Incidence	2003	R1	Anencephaly
Western Australian Intellectual Disability Exploring Answers database	Incidence	1983–1996	K9	Intellectual disability
Victorian Perinatal Data Collection Unit Birth Defects Register	Incidence	2001–02	R2	Spina bifida
	Incidence	2001–02	R5	Digestive system malformation
Congenital malformations, Australia	Incidence	2001–02	R6a	Renal agenesis
	Incidence	2001–02	K9	Intellectual disability
	Incidence	1997	R3	Congenital heart disease
	Incidence	1997	R5	Digestive system malformation
	Incidence	1997	R6b	Other urogenital tract malformations
	Incidence	2001	R7	Abdominal wall defect
	Incidence	2003	R6b	Other urogenital tract malformations

(continued)

Annex Table 2 (continued): Principal data sources for epidemiological modelling

Primary data source	Prevalence/ Incidence	Reference period	Disease and injury categories	
B. Health service utilisation data				
National Hospital Morbidity Database (diagnoses or procedures)	Incidence	2002–03	A2b	Chlamydia sequela
	Incidence		A2c	Gonorrhoea sequela
	Incidence		A4	Diarrhoea
	Incidence		A5e	Measles sequela
	Incidence		A6	Meningitis
	Incidence		A7	Septicaemia
	Incidence		A8c	Dengue fever sequela
	Incidence		A9a	Hepatitis A
	Incidence		A9c	Hepatitis B sequela (D) ^(a)
	Incidence		A9c	Hepatitis C sequela (D)
	Incidence		C1	Maternal haemorrhage (P) ^(b)
	Incidence		C3	Hypertension in pregnancy (P)
	Incidence		C4	Obstructed labour (P)
	Incidence		C5	Abortion (P)
	Incidence		C6	Other maternal conditions (P)
	Incidence		D1	Birth trauma & asphyxia
	Incidence		D3	Neonatal infections
	Incidence		G	Benign neoplasms (P)
	Incidence		H	Diabetes sequela (P)
	Incidence		I1a	Haemolytic anaemia
	Prevalence		I1b	Other non-deficiency anaemia
	Incidence		K8b	Cataract-related blindness (P)
	Incidence		L2	Ischemic heart disease—AMI
	Incidence		L3	Stroke
	Incidence		L7	Aortic aneurysm
	Prevalence		L8	Peripheral vascular disease (P)
	Incidence		L8	Peripheral vascular disease sequela
	Prevalence		N2	Cirrhosis of the liver (D)
	Incidence		N3	Appendicitis (P)
	Incidence		N4	Intestinal obstruction (P)
	Incidence		N5	Diverticulitis (P)
	Incidence		N6	Gall bladder and bile duct disease (P)
	Incidence		N7	Pancreatitis
	Incidence		N8	Inflammatory bowel disease (P)
	Incidence		N9	Vascular insufficiency of intestine (P)
	Incidence		O2	Benign prostatic hypertrophy (P)
	Incidence		Oot	Other genitourinary diseases (P)
	Incidence		Q4	Slipped disc (P)
	Incidence		R3	Congenital heart disease (P)
	Incidence		R4	Cleft lip and or palate (P)
Incidence	T	Unintentional injuries		
Incidence	U	Intentional injuries		
Bettering the Evaluation and Care of Health	Incidence	2000–01	B1	Lower respiratory tract infections
	Incidence	2000–01	B2	Upper respiratory tract infections

(continued)

Annex Table 2 (continued): Principal data sources for epidemiological modelling

Primary data source	Prevalence/ Incidence	Reference period		Disease and injury categories
	Incidence	2000–01	B3	Otitis media
	Incidence	2003–04	N1	Peptic ulcer disease
	Incidence	2003–04	P4	Skin ulcers
Alcohol and Other Drug Treatment Services National Minimum Data Set	Prevalence	2002–03	J1c	Stimulant dependence
Western Australian Data Linkage System	Incidence	1990–2003	L1	Heart failure
	Incidence	1990–2003	L2	Ischemic heart disease
	Incidence	1990–2003	L3	Stroke
Victorian Linked Admitted Episodes Database	Incidence	1996–2002	L	Heart failure
	Incidence	1996–2002	N9	Vascular insufficiency of intestine
C. Population health surveys				
2001–02 National Gastroenteritis Survey	Incidence	2001–02	A4	Diarrhoea
1980 National Trachoma and Eye Health Program	Prevalence	1976–78	A11	Trachoma sequela
	Incidence	1976–78	B3	Otitis media
National Health Survey	Incidence	1995	B2	Upper respiratory tract infections
	Incidence	2001	B3	Otitis media
	Prevalence	2001	K10	Migraine
	Prevalence	2001	P1	Eczema
	Prevalence	2001	Poth	Other skin diseases
	Prevalence	1995	Q3	Chronic back pain (U) ^(c)
	Incidence	2001	Q7	Gout
	Prevalence & incidence	2001	Qot	Other musculoskeletal disorders
	Prevalence & incidence	1995	Qot	Other musculoskeletal disorders
Australian Diabetes, Obesity and Lifestyle Study (AusDiab)	Incidence	1999–2000	E2	Deficiency anaemia
	Prevalence	1999–2000	H	Diabetes mellitus
Risk Factor Prevalence Study, 1989	Incidence	1989	E2	Deficiency anaemia
2002 National non-melanoma skin cancer survey	Incidence	2002	F11	Non-melanoma skin cancer
National Mental Health and Wellbeing Survey, 1997—adult component, Low prevalence (psychotic) disorders component, and child & adolescent component	Prevalence	1997	J1a	Alcohol dependence
	Prevalence	1997	J1c	Benzodiazepine dependence
	Prevalence	1997	J1d	Cannabis dependence
	Prevalence	1997	J2	Psychotic disorders
	Prevalence	1997	J3	Anxiety and depression
	Prevalence	1997	J4	Bipolar disorder
	Prevalence	1997	J5	Personality disorders (isolated)
	Prevalence	1997	J7a	ADHD
Australian Child to Adult Development Study	Incidence	1990–96	K9	Intellectual disability
Australian Longitudinal Study on Women's Health ^(d)	Prevalence	1996–2002	O3	Urinary incontinence
	Prevalence	1996–2002	Oot	Menstrual problems
Survey of Disability, Ageing and Carers	Prevalence	1998	O3	Urinary incontinence
	Prevalence	2003	Q3	Chronic back pain
	Prevalence	2003	Q5	Occupational overuse syndrome
	Prevalence	1993	Qot	Other musculoskeletal disorders
Child Dental Health Survey, Australia	Incidence	2000	S1	Dental caries
National Oral Health Survey of Australia	Prevalence	1987–88	S1	Dental caries
	Prevalence	1987–88	S2	Periodontal disease
South Australian Dental Longitudinal Study	Incidence	1991–1996	S1	Dental caries

(continued)

Annex Table 2 (continued): Principal data sources for epidemiological modelling

Primary data source	Prevalence/ Incidence	Reference period	Disease and injury categories	
The Adelaide Dental Study of Nursing Homes, one year follow up 1999	Incidence	1999	S1	Dental caries
The Adelaide Dental Study of Nursing Homes 1998	Prevalence	1998	S3	Edentulism
The Longitudinal Study of Dentists' Practice Activity	Incidence	1998–99	S4	Pulpal infection
National Dental Telephone Interview Survey	Prevalence	2002	S3	Edentulism
	Incidence	2002	S4	Pulpal infection
D. Epidemiological studies				
GBD study	Incidence		A2	STIs (apart from HIV/AIDS)
	Incidence		A5b	Pertussis sequela
	Incidence		A10	Malaria—sequela
	Incidence		B3	Otitis media—sequela
	Incidence		C2	Maternal sepsis—sequela
	Incidence		C3	Hypertensive disorders in pregnancy—sequela
Australian epidemiological studies	Incidence		A6	Meningitis sequela
	Prevalence		A9b	Hepatitis B
	Prevalence		A9c	Hepatitis C sequela
	Incidence		D4	Other neonatal causes
	Prevalence		E2	Deficiency anaemia
	Incidence		H	Diabetes mellitus sequela
	Prevalence		I3	Haemophilia
	Prevalence		J1b	Heroin dependence
	Prevalence		J6b	Anorexia
	Incidence		J7b	Autism spectrum disorders
	Prevalence		K4	Multiple sclerosis
	Incidence		K6	Huntington's chorea
	Incidence		K7	Muscular dystrophy
	Prevalence		K8	Sense organ disorders
	Incidence		K9	Intellectual disability
	Prevalence		L3	Stroke
	Prevalence		M1	Chronic obstructive pulmonary disease
	Prevalence		M2	Asthma
	Prevalence		N2	Cirrhosis of the liver
Prevalence		O4	Infertility	
Prevalence		P1	Eczema	
International epidemiological studies	Prevalence		Poth	Other skin diseases
	Incidence		A2b	Chlamydia sequela (i.e. childwish)
	Prevalence		A9b	Hepatitis B sequela
	Incidence		A9c	Hepatitis C sequela
	Incidence		D1	Birth trauma & asphyxia—sequela
	Incidence		D2	Low birthweight—sequela
	Incidence		J6a	Bulimia
	Incidence		K2	Epilepsy
	Incidence		K10	Migraine
	Prevalence		M2	Asthma
Incidence		N8	Inflammatory bowel disease	

(continued)

Annex Table 2 (continued): Principal data sources for epidemiological modelling

Primary data source	Prevalence/ Incidence	Reference period	Disease and injury categories
	Prevalence	Q3	Urinary incontinence
	Incidence	Q1	Rheumatoid arthritis
	Incidence	Q2	Osteoarthritis
	Incidence	Q4	Slipped disc
	Prevalence	Z2	Chronic fatigue syndrome
Meta-analyses of epidemiological studies	Prevalence	K1	Dementia
	Prevalence	K3	Parkinson's disease
E. Estimates that are distributed to other models			
K9 Intellectual disability	Incidence		D1 Birth trauma & asphyxia
			D2 Low birthweight
			D3 Neonatal infections
			D4 Other perinatal conditions
			R8 Down syndrome
			R9 Other chromosomal anomalies
L Heart failure	Prevalence	1996–2002	L1 Rheumatic heart disease
	Incidence	1996–2002	L2 Ischemic heart disease
	Incidence	1996–2002	L4 Inflammatory heart disease
	Prevalence	1996–2002	L5 Hypertensive heart disease
	Incidence	1996–2002	L6 Non-rheumatic valvular disease
	Incidence	1996–2002	M1 Chronic obstructive pulmonary disease
F. Indirect estimation			
YLL to YLD ratio from rest of category			A12 Other infectious and parasitic diseases
			D4 Other perinatal conditions
			I4 Other endocrine and metabolic diseases
			L9 Other cardiovascular disease
			M3 Other chronic respiratory diseases
			N10 Other digestive system diseases
			R10 Other congenital anomalies
			Oot Other genitourinary diseases

Notes

- (a) (D) refers to distributions which are used to estimate incidence to underlying causes.
- (b) (P) refers to hospital data on procedures—may or may not be in addition to information on principal diagnosis.
- (c) (U) proportion by underlying cause or type of problem (recent versus long-term).
- (d) The research on which this report is based was conducted as part of the Australian Longitudinal Study on Women's Health, The University of Newcastle and The University of Queensland. We are grateful to the Australian Government Department of Health and Ageing for funding and to the women who provided the survey data.

Annex Table 3: Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
All causes	2,632,770	1,364,614	1,268,156	603,937	244,198	289,190	96,727	94,077	514,332	184,705	378,314
I. Communicable diseases, maternal and neonatal conditions	123,094	64,993	58,101	22,017	6,363	9,970	20,387	3,131	17,027	4,240	13,316
A. Infectious and parasitic diseases	44,685	27,301	17,385	2,004	4,034	3,269	1,644	1,099	8,662	2,318	3,662
1. Tuberculosis	646	330	316	3	150	100	4	11	92	44	166
2. Sexually transmitted diseases ^(a)	2,048	83	1,966	5	40	12	54	437	1,412	21	41
a. Syphilis	102	26	77	4	8	12	36	2	29	—	9
b. Chlamydia	1,188	49	1,139	—	26	—	14	264	830	13	19
c. Gonorrhoea	28	9	19	—	6	—	—	5	13	—	—
d. Other sexually transmitted diseases	730	—	730	—	—	—	4	166	539	8	13
3. HIV/AIDS	6,660	5,960	700	7	346	12	6	62	610	22	—
4. Diarrhoeal diseases	1,858	872	986	334	101	77	348	90	310	63	175
5. Childhood immunisable diseases	557	315	243	99	7	23	121	8	47	39	27
a. Diphtheria	—	—	—	—	—	—	—	—	—	—	—
b. Whooping cough	150	70	80	42	7	1	42	8	26	2	1
c. Tetanus	—	—	—	—	—	—	—	—	—	—	—
d. Poliomyelitis	197	119	78	—	—	22	—	—	21	37	21
e. Measles	1	—	—	—	—	—	—	—	—	—	—
f. Rubella	25	17	8	17	—	—	8	—	—	—	—
g. <i>Haemophilus influenzae</i> type b (Hib)	184	108	76	40	—	—	71	—	—	—	5
6. Meningitis	2,722	1,405	1,317	937	154	29	631	234	389	36	27
7. Septicaemia	3,987	2,244	1,743	224	49	704	144	25	405	231	938
8. Arbovirus infection	1,272	658	614	2	58	13	8	61	506	25	15

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females							
		Males		Females		Males		Females		Males		Females			
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74
a. Ross River virus	649	307	342	1	26	256	18	5	1	27	292	13	8		
b. Barmah Forest virus	253	126	128	—	8	106	8	4	—	9	111	5	2		
c. Dengue	5	3	2	—	1	2	—	—	—	—	1	—	—		
d. Other arbovirus infection	364	222	142	—	23	180	15	4	6	24	101	6	4		
9. Hepatitis	19,889	13,072	6,817	74	36	8,848	2,456	1,659	30	30	3,970	1,305	1,482		
a. Hepatitis A	51	26	25	5	3	11	1	7	3	3	8	1	10		
b. Hepatitis B ^(b)	6,961	4,429	2,532	45	31	2,430	967	956	13	16	1,074	530	899		
c. Hepatitis C ^(c)	12,723	8,509	4,214	15	2	6,308	1,488	696	13	12	2,887	775	528		
d. Other hepatitis	154	108	46	9	—	98	—	—	—	—	—	—	46		
10. Malaria	89	60	29	30	29	1	—	—	—	—	28	—	—		
11. Trachoma	121	55	66	—	1	42	11	1	—	1	49	14	2		
12. Other infectious and parasitic diseases	4,835	2,247	2,588	288	80	694	545	640	297	140	843	518	790		
B. Acute respiratory infections	35,502	17,217	18,285	3,388	833	4,461	2,078	6,456	2,790	851	3,791	1,635	9,219		
1. Lower respiratory tract infections	27,354	13,121	14,233	1,067	298	3,360	2,001	6,395	798	268	2,519	1,511	9,137		
2. Upper respiratory tract infections	3,451	1,614	1,837	615	282	618	56	43	615	359	731	82	50		
3. Otitis media	4,697	2,482	2,215	1,706	254	484	22	17	1,377	223	541	42	33		
C. Maternal conditions	2,152	—	2,152	—	—	—	—	—	1	434	1,716	—	—		
1. Maternal haemorrhage	126	—	126	—	—	—	—	—	—	19	108	—	—		
2. Maternal sepsis	332	—	332	—	—	—	—	—	1	95	236	—	—		
3. Hypertensive disorders of pregnancy	887	—	887	—	—	—	—	—	1	204	683	—	—		
4. Obstructed labour	147	—	147	—	—	—	—	—	—	24	123	—	—		
5. Abortion	25	—	25	—	—	—	—	—	—	12	14	—	—		
6. Other maternal conditions	634	—	634	—	—	—	—	—	—	82	552	—	—		
D. Neonatal causes	34,558	19,027	15,531	19,027	—	—	—	—	15,530	—	—	—	—		
1. Birth trauma and asphyxia	9,308	5,086	4,221	5,086	—	—	—	—	4,221	—	—	—	—		

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
2. Low birthweight	15,423	8,281	—	—	—	—	7,142	—	—	—	—	—
3. Neonatal infections	3,404	2,156	—	—	—	—	1,248	—	—	—	—	—
4. Other conditions arising in the perinatal period	6,424	3,505	—	—	—	—	2,919	—	—	—	—	—
E. Nutritional deficiencies	6,197	1,449	417	73	462	251	421	746	2,858	287	435	48
1. Protein-energy malnutrition	97	33	64	—	1	—	—	1	—	14	48	—
2. Deficiency anaemia	6,011	1,368	4,643	73	461	238	421	746	2,842	259	376	—
3. Other nutritional deficiencies	89	48	42	—	—	12	1	—	17	14	10	—
II. Non-communicable diseases	2,324,625	1,170,116	1,154,509	90,683	72,481	232,155	69,322	83,085	472,356	175,985	353,761	—
F. Malignant neoplasms	499,416	264,382	235,034	2,512	2,530	77,316	1,577	1,926	117,559	53,828	60,144	—
1. Mouth and oropharynx cancers	13,464	9,483	3,981	36	122	2,226	2	53	1,984	910	1,032	—
2. Oesophagus cancer	14,163	9,983	4,180	—	29	2,933	—	—	1,292	1,190	1,698	—
3. Stomach cancer	15,218	9,073	6,145	1	3	2,788	—	31	2,661	1,388	2,064	—
4. Colorectal cancer	63,605	34,643	28,962	2	46	10,531	2	52	11,693	7,513	9,703	—
5. Liver cancer ^(e)	4,716	3,241	1,474	15	2	948	14	12	648	333	468	—
6. Gallbladder cancer	3,549	1,429	2,121	—	—	500	—	—	752	633	735	—
7. Pancreas cancer	22,680	11,434	11,246	—	—	3,413	1	—	4,172	3,023	4,050	—
8. Lung cancer	88,904	55,028	33,876	62	63	19,258	1	30	14,848	9,937	9,059	—
9. Bone and connective tissue cancer	5,879	3,317	2,562	315	666	419	212	357	1,388	276	329	—
10. Melanoma	20,236	13,734	6,501	5	238	2,836	2	53	3,450	1,519	1,478	—
11. Non-melanoma skin cancers	4,734	3,233	1,502	—	2	933	—	—	391	246	864	—
12. Breast cancer	60,654	134	60,520	—	—	23	—	25	41,056	10,445	8,995	—
13. Cervix cancer	5,231	—	5,231	—	—	—	—	24	3,738	741	727	—
14. Corpus uteri cancer	4,663	—	4,663	—	—	—	—	—	2,448	1,174	1,041	—
15. Ovary cancer	11,994	—	11,994	—	—	—	11	164	6,429	2,631	2,758	—

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
16. Prostate cancer	36,547	—	—	9,112	11,950	15,484	—	—	—	—	—
17. Testicular cancer	862	6	123	713	10	11	—	—	—	—	
18. Bladder cancer	10,077	1	4	2,046	2,133	2,827	—	28	598	770	
19. Kidney cancer	12,487	47	6	4,128	2,092	1,521	53	2	1,618	1,429	
20. Brain cancer	19,792	11,515	721	7,617	1,693	970	543	194	4,959	1,538	
21. Thyroid cancer	1,762	640	—	1,122	201	120	4	45	675	159	
22. Lymphoma	22,263	12,375	173	9,888	3,161	2,597	27	318	4,029	2,382	
23. Multiple myeloma	8,925	4,778	30	1,824	1,437	1,487	—	1	1,343	1,216	
24. Leukaemia	19,956	11,393	785	4,841	2,753	2,570	542	342	3,293	1,909	
25. Larynx cancer	3,751	3,263	—	1,644	1,059	560	—	—	237	134	
26. Eye cancer	952	530	39	279	125	76	38	12	197	67	
27. Other malignant neoplasms	22,354	12,945	276	5,455	3,895	3,309	125	181	3,659	2,262	
G. Other neoplasms	10,903	4,615	155	1,377	1,180	1,666	237	45	2,998	1,057	
1. Uterine myomas	1,545	—	—	—	—	—	—	4	1,447	70	
2. Benign neoplasms of meninges and brain	1,451	518	42	218	98	153	21	6	495	203	
3. Other benign neoplasms	7,907	4,097	113	1,159	1,082	1,513	215	35	1,056	784	
H. Diabetes mellitus	143,831	77,437	975	48,711	15,183	11,887	911	802	36,213	12,109	
1. Type 1 diabetes	10,891	6,260	872	3,236	980	625	781	404	1,825	592	
2. Type 2 diabetes	132,940	71,176	103	45,476	14,203	11,262	130	398	34,388	11,517	
I. Endocrine and metabolic disorders	28,565	14,556	3,395	5,470	1,972	2,928	2,162	800	4,600	1,815	
1. Non-deficiency anaemia	5,109	2,739	917	787	399	594	636	36	594	332	
a. Haemolytic anaemia	1,313	774	689	14	13	56	476	1	2	14	
b. Other non-deficiency anaemia	3,797	1,965	228	773	385	538	160	35	592	318	
2. Cystic fibrosis	1,863	926	520	263	—	3	492	244	201	—	
3. Haemophilia	205	169	59	56	11	43	—	—	2	18	

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
4. Other endocrine and metabolic disorders	21,387	10,722	10,665	1,899	608	4,364	1,563	2,288	1,033	520	3,803	1,465	3,844
J. Mental disorders	350,545	165,676	184,869	28,633	48,387	82,282	4,711	1,662	21,492	47,683	112,469	1,878	1,347
1. Substance use disorders	60,782	46,094	14,687	3	14,711	28,148	2,397	836	79	4,180	9,680	417	331
a. Alcohol dependence and harmful use ^(e)	34,116	27,225	6,891	—	4,848	19,181	2,378	817	—	416	5,749	395	331
b. Heroin or polydrug dependence and harmful use	16,839	12,455	4,383	3	5,657	6,776	14	6	78	2,052	2,233	20	—
c. Benzodiazepine dependence and harmful use	2,656	1,102	1,554	—	207	892	3	—	—	362	1,189	2	—
d. Cannabis dependence and harmful use	5,206	4,075	1,131	—	3,520	554	1	—	—	983	148	—	—
e. Other drug dependence and harmful use	1,966	1,237	729	—	478	745	—	14	—	367	361	—	—
2. Schizophrenia	27,502	14,785	12,717	186	9,795	4,719	25	60	181	3,754	8,639	53	90
3. Anxiety and depression	191,786	65,321	126,464	9,554	17,868	36,126	1,430	343	15,507	29,946	80,515	321	175
4. Bipolar disorder	7,770	3,920	3,849	—	2,672	1,246	2	—	—	2,450	1,347	30	23
5. Personality disorders ^(f)	32,587	16,248	16,339	—	3,130	11,955	816	347	—	2,622	12,044	1,032	642
6. Eating disorders	6,062	375	5,687	103	211	52	—	9	828	4,639	200	—	19
a. Anorexia nervosa	2,933	367	2,567	103	211	52	—	—	407	2,063	91	—	5
b. Bulimia nervosa	3,087	—	3,087	—	—	—	—	—	421	2,576	90	—	—
c. Other eating disorders	41	9	33	—	—	—	—	9	—	—	19	—	14
7. Childhood conditions	23,794	18,804	4,990	18,785	—	19	—	—	4,896	93	—	—	—
a. Attention-deficit hyperactivity disorder	9,928	7,082	2,846	7,082	—	—	—	—	2,840	6	—	—	—
b. Autism spectrum disorders	13,866	11,722	2,144	11,703	—	19	—	—	2,056	88	—	—	—
8. Other mental disorders	262	127	135	1	—	17	42	66	—	—	43	26	67

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
K. Nervous system and sense organ disorders	312,766	8,850	7,613	51,886	32,573	45,723	6,744	9,296	45,104	30,004	74,973
1. Dementia	94,399	45	42	4,599	7,872	21,095	155	32	3,340	10,236	46,984
2. Epilepsy	14,821	3,249	1,209	3,430	353	237	2,446	848	2,248	336	464
3. Parkinson's disease	26,852	—	—	3,459	3,958	6,247	—	—	2,759	4,979	5,451
4. Multiple sclerosis	5,252	18	101	1,345	119	27	79	201	3,112	110	140
5. Motor neurone disease	7,088	1	1	1,896	1,124	674	33	—	1,394	1,114	851
6. Huntington's chorea	1,779	—	35	758	103	41	—	7	631	124	81
7. Muscular dystrophy	1,046	221	318	227	35	—	99	29	64	34	18
8. Sense organ disorders	112,728	383	1,073	29,229	17,192	15,439	237	857	18,678	11,675	17,964
a. Glaucoma-related blindness	3,671	—	—	868	586	244	—	—	866	694	414
b. Cataract-related blindness	2,343	5	2	139	228	510	3	1	153	337	966
c. Macular degeneration	11,642	—	—	13	1,132	3,238	—	—	14	1,338	5,906
d. Adult-onset hearing loss	64,853	—	699	22,983	11,920	7,052	—	432	12,315	5,834	3,618
e. Refractive errors	18,761	224	286	2,697	1,941	3,094	90	343	2,861	2,107	5,119
f. Other vision loss	11,457	154	87	2,529	1,386	1,301	143	81	2,470	1,364	1,941
9. Migraine	21,848	1,523	3,539	910	1	—	955	6,217	8,671	15	17
10. Other nervous system and sense organ disorders	26,953	3,411	1,294	6,034	1,815	1,964	2,741	1,104	4,206	1,381	3,004
L. Cardiovascular disease	473,794	2,112	2,414	94,217	59,839	93,822	1,632	1,324	45,697	38,727	134,009
1. Rheumatic heart disease	4,091	5	65	585	284	432	33	63	809	702	1,112
2. Ischaemic heart disease	263,497	35	322	57,210	37,860	55,680	13	120	20,352	21,052	70,853
3. Stroke	118,462	1,436	1,128	17,961	10,938	21,834	984	480	14,237	9,635	39,830
4. Inflammatory heart disease	15,904	419	305	5,207	2,078	2,125	428	156	2,066	1,181	1,939
5. Hypertensive heart disease	8,982	6	5	1,018	885	1,855	7	5	637	708	3,856

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
6. Non-rheumatic valvular disease	8,951	4,367	4,584	29	141	1,390	912	1,896	26	80	887	772	2,820
7. Aortic aneurysm	11,338	7,189	4,149	—	59	1,871	2,187	3,071	31	29	580	916	2,594
8. Peripheral vascular disease	18,606	10,604	8,002	50	74	4,816	2,639	3,026	21	93	2,592	1,519	3,777
9. Other cardiovascular disease	23,962	10,569	13,394	133	315	4,161	2,058	3,903	90	297	3,537	2,242	7,228
M. Chronic respiratory disease	186,737	98,925	87,813	23,093	1,936	31,452	17,475	24,968	16,944	6,925	26,552	13,854	23,537
1. Chronic obstructive pulmonary disease (COPD)	86,751	49,201	37,550	378	294	21,936	11,693	14,900	174	278	14,923	8,855	13,318
2. Asthma	63,100	29,271	33,828	21,953	1,314	4,802	738	465	16,490	6,641	8,069	1,412	1,216
3. Other chronic respiratory diseases	36,887	20,453	16,435	762	329	4,715	5,044	9,603	280	5	3,560	3,587	9,003
N. Diseases of the digestive system	57,957	28,613	29,344	1,204	1,281	14,092	5,083	6,953	799	1,134	11,792	4,535	11,084
1. Peptic ulcer disease	6,358	3,292	3,065	32	39	1,622	662	937	—	6	1,148	334	1,577
2. Cirrhosis of the liver ^(d)	1,524	687	838	31	17	277	112	249	1	3	178	87	569
3. Appendicitis	648	324	323	53	59	135	13	64	41	60	154	30	38
4. Intestinal obstruction	5,019	2,227	2,792	61	18	665	582	902	10	18	927	381	1,455
5. Diverticulitis	6,118	2,829	3,289	—	6	1,373	701	749	—	1	1,072	921	1,296
6. Gallbladder and bile duct disease	3,202	1,212	1,990	2	6	395	359	450	3	55	852	295	785
7. Pancreatitis	2,501	1,464	1,037	2	38	921	232	273	2	37	498	151	348
8. Inflammatory bowel disease	12,176	6,334	5,843	553	1,001	4,369	264	148	523	854	4,044	227	195
9. Vascular insufficiency of bowel	3,982	1,647	2,335	125	29	463	365	664	32	35	557	592	1,119
10. Other digestive system diseases	16,430	8,597	7,832	346	69	3,872	1,792	2,518	186	64	2,362	1,518	3,702

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
O. Genitourinary diseases	65,249	28,163	37,086	127	1,637	11,461	5,791	9,148	1,068	7,860	14,951	3,273	9,935
1. Nephritis and nephrosis ^(b)	21,133	10,688	10,444	106	107	2,808	1,933	5,734	46	146	1,944	1,632	6,677
2. Benign prostatic hypertrophy	7,622	7,622	—	—	—	2,723	2,950	1,949	—	—	—	—	—
3. Urinary incontinence	8,263	1,823	6,440	—	—	898	542	383	1	217	4,271	1,053	898
4. Infertility	14,344	6,268	8,076	21	1,502	4,746	—	—	19	1,822	6,236	—	—
5. Other genitourinary diseases	13,888	1,762	12,126	—	28	286	365	1,082	1,002	5,676	2,500	589	2,360
P. Skin diseases	20,302	9,852	10,451	1,446	1,679	4,555	1,126	1,045	1,593	1,778	2,672	1,408	3,000
1. Eczema	2,730	1,031	1,699	371	47	555	31	27	1,210	42	413	31	2
2. Acne	3,899	1,988	1,910	646	1,013	329	—	—	242	1,198	470	—	—
3. Psoriasis	4,021	3,122	899	206	578	2,059	174	105	58	192	524	76	49
4. Ulcers	9,324	3,620	5,704	222	41	1,575	886	895	82	346	1,177	1,235	2,864
5. Other skin diseases	329	90	238	1	—	37	35	18	—	—	88	65	84
Q. Musculoskeletal diseases	105,508	44,210	61,298	856	1,289	27,639	8,375	6,052	1,305	1,639	35,570	11,574	11,211
1. Rheumatoid arthritis	16,841	4,780	12,062	343	214	2,833	888	502	958	513	7,658	1,710	1,222
2. Osteoarthritis	34,578	14,495	20,083	1	58	7,772	3,863	2,802	—	—	7,356	6,088	6,638
3. Back pain ⁽ⁱ⁾	29,658	14,470	15,188	275	541	9,776	2,227	1,650	206	610	10,704	2,012	1,657
4. Slipped disc	6,120	3,439	2,681	13	144	2,711	386	184	29	84	1,956	401	211
5. Occupational overuse syndrome	4,953	697	4,256	—	9	663	24	—	—	65	4,177	13	1
6. Systemic lupus erythematosus (SLE)	1,609	168	1,441	—	1	43	56	68	1	76	984	186	193
7. Gout	1,988	1,636	352	2	85	1,330	100	119	1	59	131	97	64
8. Other musculoskeletal diseases	9,759	4,525	5,235	222	236	2,511	829	726	109	232	2,605	1,066	1,223
R. Congenital anomalies	33,228	18,770	14,458	14,738	624	2,688	345	374	10,838	528	2,172	439	481
1. Anencephaly	387	102	285	102	—	—	—	—	285	—	—	—	—
2. Spina bifida	812	408	404	307	31	57	12	—	270	30	105	—	—
3. Congenital heart disease	8,394	4,975	3,419	3,434	282	1,091	97	71	2,202	268	723	135	90

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
		Males	Females	Males	Females	Males	Females	Males	Females	Males	Females
4. Cleft lip and/or palate	221	112	109	112	—	—	109	—	—	—	—
5. Digestive system malformations	493	244	248	207	16	12	222	1	—	12	14
a. Anorectal atresia	31	17	14	17	—	—	14	—	—	—	—
b. Oesophageal atresia	31	19	12	19	—	—	12	—	—	—	—
c. Other digestive system malformations	431	208	223	171	16	12	196	1	—	12	14
6. Urogenital tract malformations	2,575	1,560	1,016	568	1	168	144	—	393	191	288
a. Renal agenesis ⁽ⁱ⁾	279	153	126	145	1	7	82	—	29	11	3
b. Other urogenital tract malformations ^(k)	2,296	1,407	890	422	—	168	62	—	364	180	284
7. Abdominal wall defect	312	210	102	210	—	—	102	—	—	—	—
8. Down syndrome	3,808	2,181	1,627	1,668	61	22	1,059	2	470	79	18
9. Other chromosomal disorders	8,493	4,685	3,807	4,682	1	—	3,754	1	52	—	—
10. Other congenital anomalies	7,733	4,293	3,440	3,448	248	32	2,692	226	429	21	71
S. Oral conditions	24,507	11,402	13,105	1,114	1,098	1,186	1,062	1,065	8,490	1,470	1,017
1. Dental caries	12,088	6,026	6,061	665	789	427	631	760	3,819	375	476
2. Periodontal disease	581	280	301	5	20	19	5	19	237	30	9
3. Edentulism	5,264	1,880	3,384	2	7	526	3	12	2,281	836	252
4. Pulpitis	6,497	3,197	3,300	443	283	214	424	274	2,127	228	248
5. Other oral conditions	77	18	59	—	—	—	—	—	26	—	32
Z. Ill-defined conditions	11,317	4,467	6,850	1,470	283	—	958	280	5,517	14	81
1. Sudden infant death syndrome	2,428	1,470	958	1,470	—	—	958	—	—	—	—
2. Chronic fatigue syndrome	8,890	2,997	5,893	—	283	—	—	280	5,517	14	81

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
III. Injuries	185,050	129,504	9,290	28,191	80,233	5,681	7,018	24,949	4,480	11,238	
T. Unintentional injuries	125,862	84,201	8,695	19,148	46,795	4,154	6,293	14,745	3,963	10,997	
1. Road traffic accidents	42,425	31,028	1,397	10,380	17,215	838	1,336	5,253	621	616	
2. Other transport accidents	8,601	6,782	1,819	1,756	3,996	177	556	815	62	70	
3. Poisoning	12,046	6,922	5,124	927	5,501	230	55	2,722	691	1,194	
4. Falls	26,386	13,118	13,269	1,717	5,171	1,490	1,086	2,119	1,870	7,814	
5. Fires, burns and scalds	4,399	2,822	1,577	279	1,499	154	564	775	67	108	
6. Drowning	4,812	3,366	1,447	672	1,854	114	706	532	81	34	
7. Sports injuries	579	344	234	112	147	8	44	96	16	35	
8. Natural and environmental factors	1,927	1,330	597	277	780	53	190	182	54	72	
9. Machinery accidents	5,095	4,725	370	957	3,255	227	37	270	11	4	
10. Other unintentional injuries ^(b)	19,591	13,765	5,827	2,070	7,378	862	1,718	1,981	491	1,050	
Suffocation and foreign bodies	5,727	3,930	1,797	606	2,133	172	734	529	55	298	
Adverse effects of medical treatment	3,695	2,016	1,678	124	812	405	55	580	347	570	
Other unintentional injuries n.e.c.	10,169	7,818	2,351	1,340	4,433	285	929	872	89	182	
U. Intentional injuries	59,189	45,303	13,886	9,043	33,438	1,527	726	10,204	517	240	
1. Suicide and self-inflicted injuries	49,916	38,717	11,199	7,320	29,099	1,437	208	8,854	467	191	
2. Homicide and violence	9,221	6,535	2,686	1,722	4,289	90	518	1,349	50	49	
3. Legal intervention and war	51	51	—	1	50	—	—	—	—	—	
Australian population ('000)	19,881	9,872	10,010	2,041	1,404	5,292	1,938	1,349	5,311	718	
DALYs per 1,000 population	132.4	138.2	126.7	61.2	73.0	372.3	49.9	69.7	96.8	526.9	

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
Risk factors													
Alcohol	61,091	52,180	8,911	737	11,648	38,139	2,867	-1,211	237	1,518	10,704	-296	-3,252
Illicit drugs	51,463	36,515	14,948	78	11,892	21,161	2,109	1,276	67	4,246	8,579	1,067	989
Tobacco	204,788	131,616	73,172	2,238	88	62,414	35,879	30,997	1,757	22	28,699	18,915	23,780
Unsafe sex	14,897	6,217	8,679	22	303	5,022	514	357	60	493	5,990	1,004	1,132
Child sexual abuse	23,513	4,166	19,348	—	704	3,204	173	84	—	4,309	14,597	209	232
Intimate partner violence	29,360	—	29,360	—	—	—	—	—	—	5,455	22,325	901	678
Occupational exposures & hazards	51,362	35,492	15,870	—	2,853	25,291	4,245	3,104	—	1,387	12,458	1,097	928
Physical inactivity	174,431	87,742	86,689	—	147	42,424	21,262	23,909	—	166	32,596	17,172	36,756
High blood pressure	199,315	107,098	92,218	—	—	33,296	28,717	45,085	—	—	12,100	18,236	61,882
High body mass	197,632	105,616	92,017	—	—	65,684	22,683	17,248	—	—	46,520	20,534	24,963
Low fruit and vegetable consumption	55,259	36,429	18,830	—	103	18,804	9,111	8,411	—	38	6,272	4,150	8,370
High blood cholesterol	163,591	89,669	73,922	—	—	45,316	19,718	24,635	—	—	17,979	14,247	41,695
Osteoporosis	4,386	1,019	3,368	—	—	18	128	873	—	—	23	209	3,135
Air pollution - short term	7,781	4,032	3,750	70	37	981	1,010	1,935	54	8	629	702	2,356
Particulates	3,807	1,976	1,831	19	11	590	470	885	12	4	274	323	1,219
Ozone	3,974	2,056	1,918	50	25	391	539	1,050	42	4	355	379	1,138
Air pollution - long term	19,738	10,422	9,316	—	—	4,097	2,768	3,557	—	—	2,280	1,740	5,296
Joint effect of all risk factors	847,307	478,511	368,796	3,075	27,569	242,892	99,049	105,926	2,122	15,726	155,198	63,319	132,432
Alternative burden of disease categories													
Diabetes mellitus (attributable)	218,518	112,615	105,904	977	688	56,122	24,190	30,637	913	805	39,282	17,720	47,183
Anxiety and depression (attributable)	215,783	80,770	135,013	9,560	19,056	48,235	2,620	1,298	15,526	30,441	86,556	1,134	1,356
All intellectual disability	44,187	22,822	21,365	18,743	666	3,043	228	141	18,591	270	1,935	244	326
All vision loss	55,539	26,828	28,711	5,354	404	6,746	5,548	8,775	384	425	6,834	6,109	14,959
All nephritis and nephrosis	68,721	37,691	31,030	519	1,029	15,723	8,514	11,905	215	682	8,480	6,281	15,371

(continued)

Annex Table 3 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Australia, 2003

Notes

- (a) Excludes HIV/AIDS.
- (b) Includes hepatitis B-related liver cancer and cirrhosis.
- (c) Includes hepatitis C-related liver cancer and cirrhosis.
- (d) Excludes liver cancer related to hepatitis B and C.
- (e) Includes alcoholic cirrhosis.
- (f) Excludes those with any other comorbid mental disorders.
- (g) Excludes alcoholic and hepatic cirrhosis.
- (h) Excludes diabetic-, congenital- and poisoning-related renal failure.
- (i) Includes both acute and chronic back pain.
- (j) Includes renal failure due to dysplasia.
- (k) Includes polycystic renal failure.
- (l) Includes suffocation and foreign bodies, adverse effects of medical treatment, other mechanical force injuries and other unintentional injuries.

Annex Table 4: Deaths by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
All causes	132,287	68,325	63,962	1,004	1,071	15,483	14,039	36,728	776	399	8,895	8,295	45,597
I. Communicable diseases, maternal and neonatal conditions	6,847	3,464	3,383	405	15	734	496	1,813	314	14	340	267	2,449
A. Infectious and parasitic diseases	2,416	1,465	952	28	10	606	319	501	23	10	254	150	514
1. Tuberculosis	52	26	26	—	—	5	5	16	—	—	2	3	21
2. Sexually transmitted diseases ^(a)	12	2	10	—	—	—	—	2	1	—	4	—	5
a. Syphilis	5	2	3	—	—	—	—	2	1	—	1	—	1
b. Chlamydia	4	—	4	—	—	—	—	—	—	—	2	—	2
c. Gonorrhoea	—	—	—	—	—	—	—	—	—	—	—	—	—
d. Other sexually transmitted diseases	3	—	3	—	—	—	—	—	—	—	1	—	2
3. HIV/AIDS	119	108	11	—	—	94	12	1	—	—	10	1	—
4. Diarrhoeal diseases	48	18	29	—	1	3	3	11	1	—	—	3	25
5. Childhood immunisable diseases	27	16	11	1	—	5	6	4	2	—	1	3	5
a. Diphtheria	—	—	—	—	—	—	—	—	—	—	—	—	—
b. Whooping cough	—	—	—	—	—	—	—	—	—	—	—	—	—
c. Tetanus	—	—	—	—	—	—	—	—	—	—	—	—	—
d. Poliomyelitis	20	12	8	—	—	2	6	4	—	—	1	3	4
e. Measles	—	—	—	—	—	—	—	—	—	—	—	—	—
f. Rubella	—	—	—	—	—	—	—	—	—	—	—	—	—
g. <i>Haemophilus influenzae</i> type b (Hib)	7	4	3	1	—	3	—	—	2	—	—	—	1
6. Meningitis	61	29	32	11	4	5	5	4	9	6	12	2	3
7. Septicaemia	304	156	149	5	1	23	35	91	3	—	9	11	125
8. Arbovirus infection	—	—	—	—	—	—	—	—	—	—	—	—	—
a. Ross River virus	—	—	—	—	—	—	—	—	—	—	—	—	—

(continued)

Annex Table 4 (continued): Deaths by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females							
		Males	Females	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
b. Barmah Forest virus	—	—	—	—	—	—	—	—	—	—	—	—	—	—
c. Dengue	—	—	—	—	—	—	—	—	—	—	—	—	—	—
d. Other arbovirus infection	—	—	—	—	—	—	—	—	—	—	—	—	—	—
9. Hepatitis	1,455	933	521	2	1	439	212	280	1	1	187	100	233	—
a. Hepatitis A	2	1	1	—	—	—	—	1	—	—	—	—	1	—
b. Hepatitis B ^(b)	625	381	244	1	1	121	83	175	—	—	49	41	154	—
c. Hepatitis C ^(c)	824	550	274	—	—	317	129	104	—	—	138	59	76	—
d. Other hepatitis	3	1	2	—	—	1	—	—	—	—	—	—	2	—
10. Malaria	3	2	1	1	1	—	—	—	—	—	1	—	—	—
11. Trachoma	—	—	—	—	—	—	—	—	—	—	—	—	—	—
12. Other infectious and parasitic diseases	335	174	161	8	2	30	41	92	6	3	28	27	97	—
B. Acute respiratory infections	3,724	1,630	2,095	23	5	128	176	1,297	19	1	78	110	1,886	—
1. Lower respiratory tract infections	3,709	1,624	2,085	21	5	126	176	1,295	18	1	76	110	1,880	—
2. Upper respiratory tract infections	9	3	6	—	—	1	—	2	1	—	1	—	4	—
3. Otitis media	6	3	3	2	—	1	—	—	—	—	1	—	2	—
C. Maternal conditions	9	—	9	—	—	—	—	—	—	2	7	—	—	—
1. Maternal haemorrhage	1	—	1	—	—	—	—	—	—	—	1	—	—	—
2. Maternal sepsis	1	—	1	—	—	—	—	—	—	—	1	—	—	—
3. Hypertensive disorders of pregnancy	1	—	1	—	—	—	—	—	—	1	—	—	—	—
4. Obstructed labour	—	—	—	—	—	—	—	—	—	—	—	—	—	—
5. Abortion	—	—	—	—	—	—	—	—	—	—	—	—	—	—
6. Other maternal conditions	5	—	5	—	—	—	—	—	—	1	4	—	—	—
D. Neonatal causes	624	352	272	352	—	—	—	—	272	—	—	—	—	—
1. Birth trauma and asphyxia	134	68	66	68	—	—	—	—	66	—	—	—	—	—
2. Low birthweight	281	159	122	159	—	—	—	—	122	—	—	—	—	—
3. Neonatal infections	51	31	20	31	—	—	—	—	20	—	—	—	—	—

(continued)

Annex Table 4 (continued): Deaths by age, sex and cause, Australia, 2003

Cause	Persons		Males					Females				
			0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
4. Other conditions arising in the perinatal period	158	93	65	—	—	—	—	65	—	—	—	—
E. Nutritional deficiencies	74	17	56	1	—	—	1	15	—	1	6	49
1. Protein-energy malnutrition	16	6	10	—	—	—	—	6	—	—	1	9
2. Deficiency anaemia	50	8	42	—	—	—	—	8	—	—	4	38
3. Other nutritional deficiencies	7	3	4	—	—	—	1	1	—	1	1	2
II. Non-communicable diseases	117,499	59,690	57,809	456	274	11,835	13,135	33,991	174	7,683	7,801	41,820
F. Malignant neoplasms	37,222	20,835	16,387	68	71	5,371	5,869	9,455	54	4,677	3,573	8,044
1. Mouth and oropharynx cancers	761	516	245	1	3	223	151	138	1	68	54	122
2. Oesophagus cancer	1,222	825	397	—	1	272	252	300	—	60	85	252
3. Stomach cancer	1,288	763	525	—	—	206	229	329	1	122	101	301
4. Colorectal cancer	4,871	2,620	2,251	—	1	740	762	1,117	1	497	482	1,271
5. Liver cancer ^(e)	397	268	128	—	—	87	84	97	—	34	26	68
6. Gallbladder cancer	316	125	192	—	—	31	42	52	—	38	47	106
7. Pancreas cancer	2,063	1,016	1,047	—	—	305	302	410	—	217	232	598
8. Lung cancer	7,549	4,872	2,677	2	2	1,216	1,634	2,018	1	731	733	1,213
9. Bone and connective tissue cancer	308	180	128	10	22	64	33	52	11	55	18	39
10. Melanoma	1,220	814	407	—	6	320	191	297	1	148	84	173
11. Non-melanoma skin cancers	432	281	152	—	—	51	68	162	—	11	12	129
12. Breast cancer	2,955	11	2,944	—	—	5	2	4	—	1,311	555	1,078
13. Cervix cancer	298	—	298	—	—	—	—	—	—	142	54	102
14. Corpus uteri cancer	280	—	280	—	—	—	—	—	—	80	71	129
15. Ovary cancer	869	—	869	—	—	—	—	—	4	297	195	372
16. Prostate cancer	3,075	3,075	—	—	—	233	674	2,168	—	—	—	—
17. Testicular cancer	18	18	—	—	1	16	—	1	—	—	—	—
18. Bladder cancer	951	639	311	—	—	91	145	404	1	25	51	234
19. Kidney cancer	954	575	378	1	—	201	162	211	1	65	96	215
20. Brain cancer	1,211	702	509	19	15	371	149	149	15	227	120	141

(continued)

Annex Table 4 (continued): Deaths by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
21. Thyroid cancer	91	—	—	9	17	16	—	—	8	9	32
22. Lymphoma	1,690	4	5	282	253	381	—	9	167	163	427
23. Multiple myeloma	828	1	—	93	118	230	—	—	66	89	231
24. Leukaemia	1,531	22	14	223	228	387	14	11	134	137	361
25. Larynx cancer	245	—	—	70	74	68	—	—	11	9	13
26. Eye cancer	39	—	—	7	5	8	—	—	4	3	12
27. Other malignant neoplasms	1,758	8	—	258	296	458	3	5	159	146	424
G. Other neoplasms	839	4	7	60	92	263	6	1	57	67	281
1. Uterine myomas	1	—	—	—	—	—	—	—	—	1	—
2. Benign neoplasms of meninges and brain	70	1	—	4	5	17	—	—	11	11	21
3. Other benign neoplasms	768	3	7	56	87	246	6	1	46	55	260
H. Diabetes mellitus	3,590	—	2	366	553	1,005	2	3	161	294	1,204
1. Type 1 diabetes	460	—	2	85	66	85	2	3	39	39	140
2. Type 2 diabetes	3,130	—	—	281	487	921	—	—	122	255	1,063
I. Endocrine and metabolic disorders	1,249	26	14	154	80	279	17	17	120	77	466
1. Non-deficiency anaemia	193	—	—	15	9	51	2	—	8	11	96
a. Haemolytic anaemia	23	—	—	1	1	11	—	—	—	1	9
b. Other non-deficiency anaemia	170	—	—	15	8	40	2	—	8	10	87
2. Cystic fibrosis	33	—	5	11	—	1	—	9	8	—	—
3. Haemophilia	16	—	—	3	1	7	—	—	—	1	3
4. Other endocrine and metabolic disorders	1,007	26	9	125	70	219	15	8	104	64	367
J. Mental disorders	1,371	—	42	654	189	145	1	20	172	39	109
1. Substance use disorders	1,229	—	41	647	184	103	1	17	161	30	44
a. Alcohol dependence and harmful use ^(e)	918	—	2	456	184	101	—	1	100	29	44

(continued)

Annex Table 4 (continued): Deaths by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		Males	Females	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
b. Heroin or polydrug dependence and harmful use	264	200	64	—	32	168	—	—	1	12	50	1	—
c. Benzodiazepine dependence and harmful use	1	—	1	—	—	—	—	—	—	—	1	—	—
d. Cannabis dependence and harmful use	—	—	—	—	—	—	—	—	—	—	—	—	—
e. Other drug dependence and harmful use	45	32	13	—	7	24	—	2	—	4	10	—	—
2. Schizophrenia	31	12	19	—	—	2	1	9	—	—	1	3	15
3. Anxiety and depression	51	17	34	—	—	2	—	15	—	—	2	2	30
4. Bipolar disorder	7	1	6	—	—	1	—	—	—	—	1	2	3
5. Personality disorders ⁽ⁱ⁾	—	—	—	—	—	—	—	—	—	—	—	—	—
6. Eating disorders	11	2	9	—	—	—	—	2	—	—	5	—	4
a. Anorexia nervosa	5	—	5	—	—	—	—	—	—	—	4	—	1
b. Bulimia nervosa	—	—	—	—	—	—	—	—	—	—	—	—	—
c. Other eating disorders	6	2	4	—	—	—	—	2	—	—	1	—	3
7. Childhood conditions	4	1	3	—	—	1	—	—	—	3	—	—	—
a. Attention-deficit hyperactivity disorder	—	—	—	—	—	—	—	—	—	—	—	—	—
b. Autism spectrum disorders	4	1	3	—	—	1	—	—	—	3	—	—	—
8. Other mental disorders	37	20	17	—	—	1	4	15	—	—	2	2	13
K. Nervous system and sense organ disorders	6,922	2,749	4,173	48	49	419	371	1,861	41	23	296	314	3,499
1. Dementia	4,426	1,416	3,009	1	1	40	120	1,254	5	1	39	126	2,837
2. Epilepsy	338	196	143	10	22	122	18	24	7	9	62	13	51
3. Parkinson's disease	845	457	387	—	—	14	66	377	—	—	9	41	337
4. Multiple sclerosis	103	34	70	—	—	18	10	5	—	—	45	8	17
5. Motor neurone disease	533	277	256	—	—	94	93	89	1	—	66	82	106
6. Huntington's chorea	67	34	32	—	—	22	7	5	—	—	13	8	11

(continued)

Annex Table 4 (continued): Deaths by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		Males	Females	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
7. Muscular dystrophy	36	26	10	1	11	10	3	—	1	1	3	3	2
8. Sense organ disorders	3	—	3	—	—	—	—	—	—	—	—	—	3
a. Glaucoma-related blindness	1	—	1	—	—	—	—	—	—	—	—	—	1
b. Cataract-related blindness	—	—	—	—	—	—	—	—	—	—	—	—	—
c. Macular degeneration	—	—	—	—	—	—	—	—	—	—	—	—	—
d. Adult-onset hearing loss	—	—	—	—	—	—	—	—	—	—	—	—	—
e. Refractive errors	—	—	—	—	—	—	—	—	—	—	—	—	—
f. Other vision loss	2	—	2	—	—	—	—	—	—	—	—	—	2
9. Migraine	1	—	1	—	—	—	—	—	—	—	—	—	1
10. Other nervous system and sense organ disorders	571	309	262	36	15	97	53	107	27	11	59	32	132
L. Cardiovascular disease	48,768	23,481	25,287	22	48	3,786	4,367	15,258	21	28	1,351	2,254	21,633
1. Rheumatic heart disease	289	98	191	—	2	26	18	52	1	2	26	41	121
2. Ischaemic heart disease	28,207	14,754	13,453	1	10	2,666	2,922	9,155	—	3	675	1,217	11,558
3. Stroke	12,369	4,882	7,488	7	12	466	735	3,661	5	7	347	583	6,545
4. Inflammatory heart disease	1,115	681	434	10	8	212	146	305	11	3	64	65	290
5. Hypertensive heart disease	1,335	490	845	—	—	49	74	368	—	—	26	50	769
6. Non-rheumatic valvular disease	1,017	464	553	—	4	64	72	324	—	2	30	48	472
7. Aortic aneurysm	1,340	821	520	—	2	104	196	519	1	1	30	75	414
8. Peripheral vascular disease	861	365	496	1	1	29	55	278	—	1	19	38	438
9. Other cardiovascular disease	2,234	927	1,308	3	9	169	149	596	2	8	134	138	1,025
M. Chronic respiratory disease	8,519	4,748	3,770	28	16	482	1,037	3,184	12	3	421	650	2,685
1. Chronic obstructive pulmonary disease (COPD)	5,685	3,291	2,393	11	1	300	769	2,211	3	1	257	455	1,677
2. Asthma	333	116	218	4	9	41	18	44	3	1	59	34	120
3. Other chronic respiratory diseases	2,500	1,341	1,160	14	6	141	251	929	5	—	105	162	887

(continued)

Annex Table 4 (continued): Deaths by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		Males	Females	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
		1,447	1,939	13	4	258	257	915	4	3	178	220	1,534
N. Diseases of the digestive system	3,386	1,939	13	4	258	257	915	4	3	178	220	1,534	
1. Peptic ulcer disease	591	322	1	—	40	44	184	—	—	16	21	284	
2. Cirrhosis of the liver ^(a)	180	119	1	1	14	10	36	—	—	8	7	104	
3. Appendicitis	24	11	—	—	2	1	10	—	—	3	2	6	
4. Intestinal obstruction	485	286	1	—	12	31	155	—	—	16	17	252	
5. Diverticulitis	319	205	—	—	14	20	79	—	—	12	32	161	
6. Gallbladder and bile duct disease	267	115	—	—	10	26	78	—	—	13	16	124	
7. Pancreatitis	198	89	—	1	41	19	48	—	1	21	11	55	
8. Inflammatory bowel disease	54	33	—	—	6	4	11	—	—	6	7	20	
9. Vascular insufficiency of bowel	435	172	4	1	23	29	114	1	1	23	44	194	
10. Other digestive system diseases	832	374	5	1	95	73	198	3	1	59	62	334	
O. Genitourinary diseases	3,667	2,014	2	2	125	201	1,323	1	3	83	167	1,760	
1. Nephritis and nephrosis ^(b)	2,849	1,503	2	1	106	161	1,076	1	3	67	124	1,309	
2. Benign prostatic hypertrophy	39	—	—	—	1	6	32	—	—	—	—	—	
3. Urinary incontinence	—	—	—	—	—	—	—	—	—	—	—	—	
4. Infertility	—	—	—	—	—	—	—	—	—	—	—	—	
5. Other genitourinary diseases	779	511	—	1	17	34	216	—	—	16	43	451	
P. Skin diseases	317	114	—	—	11	26	76	—	—	10	21	172	
1. Eczema	—	—	—	—	—	—	—	—	—	—	—	—	
2. Acne	—	—	—	—	—	—	—	—	—	—	—	—	
3. Psoriasis	14	8	—	—	1	—	5	—	—	—	2	6	
4. Ulcers	270	172	—	—	8	23	67	—	—	6	14	152	
5. Other skin diseases	32	23	—	—	2	3	4	—	—	4	5	14	
Q. Musculoskeletal diseases	769	261	3	—	42	63	152	—	4	73	92	338	
1. Rheumatoid arthritis	185	50	1	—	8	15	26	—	—	9	33	92	
2. Osteoarthritis	76	14	—	—	1	—	13	—	—	1	1	59	
3. Back pain ^(c)	26	16	—	—	2	4	10	—	—	—	1	9	

(continued)

Annex Table 4 (continued): Deaths by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females							
		Males	Females	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
4. Slipped disc	4	3	1	—	—	—	2	1	—	—	—	—	—	1
5. Occupational overuse syndrome	—	—	—	—	—	—	—	—	—	—	—	—	—	—
6. Systemic lupus erythematosus (SLE)	45	7	38	—	—	1	2	4	—	1	18	6	—	12
7. Gout	25	14	11	—	—	2	1	11	—	—	—	—	—	11
8. Other musculoskeletal diseases	408	156	252	2	—	28	39	87	—	3	45	51	—	154
R. Congenital anomalies	757	413	344	192	18	107	28	68	153	15	83	32	61	61
1. Anencephaly	13	3	9	3	—	—	—	—	9	—	—	—	—	—
2. Spina bifida	17	9	8	4	1	2	1	—	3	1	4	—	—	—
3. Congenital heart disease	230	130	99	62	9	43	7	9	44	8	25	9	—	13
4. Cleft lip and/or palate	1	—	1	—	—	—	—	—	1	—	—	—	—	—
5. Digestive system malformations	19	10	9	6	—	1	1	2	6	—	—	1	—	2
a. Anorectal atresia	—	—	—	—	—	—	—	—	—	—	—	—	—	—
b. Oesophageal atresia	—	—	—	—	—	—	—	—	—	—	—	—	—	—
c. Other digestive system malformations	19	10	9	6	—	1	1	2	6	—	—	1	—	2
6. Urogenital tract malformations	175	105	70	13	—	22	15	55	2	—	16	15	—	37
a. Renal agenesis ⁽ⁱ⁾	10	4	5	4	—	—	—	—	2	—	1	1	—	1
b. Other urogenital tract malformations ^(k)	166	101	65	9	—	22	15	55	—	—	15	14	—	36
7. Abdominal wall defect	7	5	2	5	—	—	—	—	2	—	—	—	—	—
8. Down syndrome	77	40	37	13	2	23	2	—	5	—	23	6	—	2
9. Other chromosomal disorders	40	16	23	16	—	—	—	—	21	—	2	—	—	—
10. Other congenital anomalies	178	94	84	70	5	15	2	2	58	5	12	1	—	7
S. Oral conditions	16	4	12	—	—	—	—	4	—	—	1	1	10	10
1. Dental caries	—	—	—	—	—	—	—	—	—	—	—	—	—	—
2. Periodontal disease	1	—	1	—	—	—	—	—	—	—	—	—	—	—
3. Edentulism	—	—	—	—	—	—	—	—	—	—	—	—	—	—

(continued)

Annex Table 4 (continued): Deaths by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females							
		Males	Females	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
4. Pulpitis	3	—	3	—	—	—	—	—	—	—	—	—	—	3
5. Other oral conditions	12	4	8	—	—	—	—	4	—	—	1	—	—	7
Z. Ill-defined conditions	107	52	56	49	—	—	—	3	—	—	—	—	—	23
1. Sudden infant death syndrome	80	49	31	49	—	—	—	—	—	—	—	—	—	—
2. Chronic fatigue syndrome	27	3	24	—	—	—	—	3	—	—	—	—	—	23
III. Injuries	7,940	5,171	2,769	143	782	2,914	408	924	132	211	873	227	1,327	
T. Unintentional injuries	5,382	3,187	2,194	126	483	1,495	272	811	109	142	462	187	1,295	
1. Road traffic accidents	1,662	1,193	469	56	327	646	70	95	38	103	198	45	84	
2. Other transport accidents	259	212	47	12	41	138	13	8	13	4	21	3	6	
3. Poisoning	661	320	341	1	32	236	20	31	1	16	116	52	156	
4. Falls	1,668	710	958	1	25	135	79	470	3	4	37	47	866	
5. Fires, burns and scalds	117	78	39	6	1	42	12	17	2	1	17	4	15	
6. Drowning	213	151	62	21	23	83	10	13	23	3	25	6	5	
7. Sports injuries	—	—	—	—	—	—	—	—	—	—	—	—	—	
8. Natural and environmental factors	69	47	22	2	8	24	4	9	4	1	2	3	11	
9. Machinery accidents	22	22	—	1	2	16	2	1	—	—	—	—	—	
10. Other unintentional injuries ⁽¹⁾	711	455	256	25	25	176	61	169	24	9	46	27	150	
Suffocation and foreign bodies	295	190	105	20	18	82	15	54	20	6	22	4	53	
Adverse effects of medical treatment	249	140	109	1	—	17	29	93	1	2	17	19	70	
Other unintentional injuries n.e.c.	167	126	41	4	6	77	17	22	3	1	7	3	27	
U. Intentional injuries	2,559	1,984	575	17	298	1,420	136	113	23	69	411	40	33	
1. Suicide and self-inflicted injuries	2,279	1,786	493	6	261	1,280	129	110	7	50	375	36	26	
2. Homicide and violence	278	196	82	11	37	138	7	3	16	19	36	4	7	
3. Legal intervention and war	2	2	—	—	—	2	—	—	—	—	—	—	—	

(continued)

Annex Table 4 (continued): Deaths by age, sex and cause, Australia, 2003

Cause	Males						Females						
	Persons	Males	Females	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
Australian population ('000)	19,881	9,872	10,010	2,041	1,404	5,292	656	478	1,938	1,349	5,311	694	718
Deaths per 1,000 population	6.7	6.9	6.4	0.5	0.8	2.9	21.4	76.8	0.4	0.3	1.7	12.0	63.5
Risk factors													
Alcohol	1,084	1,364	-280	21	205	1,167	222	-251	6	32	302	-14	-606
Illicit drugs	1,705	1,185	520	2	81	714	181	208	1	27	257	81	155
Tobacco	15,511	10,116	5,394	35	—	2,599	2,802	4,680	25	—	1,057	1,212	3,101
Unsafe sex	655	237	418	—	—	131	42	64	1	—	175	72	169
Child sexual abuse	196	90	106	—	4	65	10	11	—	7	62	10	27
Intimate partner violence	435	—	435	—	—	—	—	—	—	24	268	56	87
Occupational exposures & hazards	1,654	1,337	317	—	52	562	289	434	—	8	130	62	116
Physical inactivity	13,491	6,434	7,058	—	4	1,396	1,428	3,606	—	2	784	880	5,391
High blood pressure	22,504	10,973	11,531	—	—	1,424	2,156	7,393	—	—	415	1,085	10,031
High body mass	9,525	5,032	4,493	—	—	1,583	1,311	2,138	—	—	752	867	2,874
Low fruit and vegetable consumption	4,568	2,830	1,738	—	3	846	690	1,291	—	1	213	248	1,275
High blood cholesterol	15,351	7,332	8,019	—	—	1,935	1,468	3,930	—	—	545	817	6,656
Osteoporosis	545	136	409	—	—	—	5	131	—	—	—	7	401
Air pollution - short term	1,046	516	530	2	1	55	94	364	2	—	33	57	438
Particulates	515	244	271	1	—	33	43	167	—	—	14	26	230
Ozone	532	273	259	2	1	23	50	197	1	—	19	31	208
Air pollution - long term	2,009	961	1,048	—	—	166	212	583	—	—	77	108	863
Joint effect of all risk factors	57,948	31,558	26,390	58	347	7,797	7,138	16,218	33	89	3,300	3,502	19,465
Alternative burden of disease categories													
Diabetes mellitus (attributable)	13,655	6,169	7,486	—	2	700	1,234	4,233	2	3	265	625	6,591
Anxiety and depression (attributable)	1,390	851	538	—	42	541	96	172	1	17	247	52	222
All intellectual disability	920	445	475	231	24	146	20	24	306	10	87	19	53

(continued)

Annex Table 4 (continued): Deaths by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
All vision loss	163	160	1	—	—	—	—	—	—	—	—	—	3
All nephritis and nephrosis	7,274	3,696	34	730	749	2,167	6	22	360	484	2,706		

Due to the deaths data adjustment processes (see Chapter 2 for details), an entry of one death in the above table does not necessarily represent one actual death from that particular cause/age group.

Notes

- (a) Excludes HIV/AIDS.
- (b) Includes hepatitis B-related liver cancer and cirrhosis.
- (c) Includes hepatitis C-related liver cancer and cirrhosis.
- (d) Excludes liver cancer related to hepatitis B and C.
- (e) Includes alcoholic cirrhosis.
- (f) Excludes those with any other comorbid mental disorders.
- (g) Excludes alcoholic and hepatic cirrhosis.
- (h) Excludes diabetic-, congenital- and poisoning-related renal failure.
- (i) Includes both acute and chronic back pain.
- (j) Includes renal failure due to dysplasia.
- (k) Includes polycystic renal failure.
- (l) Includes suffocation and foreign bodies, adverse effects of medical treatment, other mechanical force injuries and other unintentional injuries.

Annex Table 5: Prevalent years lived with disability (PYLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
All causes	1,559,364	49,137	53,938	407,260	123,094	145,798	34,923	52,334	403,590	97,264	192,026		
I. Communicable diseases, maternal and neonatal conditions	79,607	8,325	4,307	23,897	2,955	1,962	7,131	4,960	21,632	2,237	2,201		
A. Infectious and parasitic diseases	21,386	819	493	10,313	1,241	742	725	580	5,036	633	804		
1. Tuberculosis	170	3	12	50	10	12	3	9	55	7	8		
2. Sexually transmitted diseases ^(a)	2,272	5	23	42	3	1	9	162	1,978	30	18		
a. Syphilis	32	14	—	9	2	1	6	—	6	1	1		
b. Chlamydia	1,365	49	21	28	—	—	3	102	1,183	18	11		
c. Gonorrhoea	31	8	2	6	—	—	—	3	19	—	—		
d. Other sexually transmitted diseases	845	—	—	—	—	—	1	57	769	11	6		
3. HIV/AIDS	9,264	8,474	52	7,642	616	152	12	14	709	43	13		
4. Diarrhoeal diseases	1,470	690	72	250	21	22	313	88	311	28	40		
5. Childhood immunisable diseases	277	141	22	63	7	4	41	21	64	6	4		
a. Diphtheria	—	—	—	—	—	—	—	—	—	—	—		
b. Whooping cough	197	92	15	41	4	2	30	16	51	5	3		
c. Tetanus	—	—	—	—	—	—	—	—	—	—	—		
d. Poliomyelitis	—	—	—	—	—	—	—	—	—	—	—		
e. Measles	1	—	—	—	—	—	—	—	—	—	—		
f. Rubella	59	39	5	20	2	2	5	3	10	1	1		
g. <i>Haemophilus influenzae</i> type b (Hib)	20	9	2	2	—	—	6	2	2	—	—		
6. Meningitis	2,685	1,581	230	874	114	83	166	151	615	86	87		
7. Septicaemia	1,264	711	21	272	152	202	48	23	210	91	180		
8. Arbovirus infection	1,371	711	2	544	77	52	2	42	517	53	47		

(continued)

Annex Table 5 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
a. Ross River virus	647	1	25	256	19	6	1	26	292	14	8
b. Barmah Forest virus	252	—	8	105	8	4	—	8	110	6	3
c. Dengue	5	—	—	2	—	—	—	—	1	—	—
d. Other arbovirus infection	467	—	3	182	49	42	1	7	114	32	37
9. Hepatitis	880	16	6	382	95	54	6	11	194	53	62
a. Hepatitis A	35	5	3	11	1	1	3	3	9	1	1
b. Hepatitis B ^(b)	310	3	3	98	41	28	3	8	84	24	18
c. Hepatitis C ^(c)	424	1	—	198	54	26	1	—	102	28	15
d. Other hepatitis	110	7	—	75	—	—	—	—	—	—	28
10. Malaria	2	—	—	1	—	—	—	—	1	—	—
11. Trachoma	123	—	—	29	16	9	—	—	32	21	15
12. Other infectious and parasitic diseases	1,606	68	19	164	129	151	123	58	351	215	329
B. Acute respiratory infections	12,073	2,525	775	2,253	255	243	2,097	877	2,434	301	314
1. Lower respiratory tract infections	3,788	418	152	977	155	175	263	223	1,034	162	228
2. Upper respiratory tract infections	3,326	599	281	600	56	35	568	355	714	78	40
3. Otitis media	4,960	1,508	342	676	44	32	1,266	298	686	61	46
C. Maternal conditions	2,096	—	—	—	—	—	1	271	1,774	25	25
1. Maternal haemorrhage	96	—	—	—	—	—	—	17	79	—	—
2. Maternal sepsis	379	—	—	—	—	—	—	43	336	—	—
3. Hypertensive disorders of pregnancy	939	—	—	—	—	—	—	138	750	25	25
4. Obstructed labour	146	—	—	—	—	—	—	22	124	—	—
5. Abortion	36	—	—	—	—	—	—	3	33	—	—
6. Other maternal conditions	500	—	—	—	—	—	—	48	452	—	—
D. Neonatal causes	38,446	20,484	2,971	10,871	1,240	802	3,898	2,575	9,568	1,077	845
1. Birth trauma and asphyxia	13,316	7,624	1,122	4,119	476	315	1,099	819	3,103	364	308
2. Low birthweight	17,430	8,702	1,286	4,705	538	349	1,819	1,273	4,701	527	408

(continued)

Annex Table 5 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
3. Neonatal infections	3,620	2,433	1,188	821	307	1,112	121	72	474	132	493	53	37
4. Other conditions arising in the perinatal period	4,079	1,725	2,355	363	256	934	105	67	507	351	1,271	133	92
E. Nutritional deficiencies	5,606	1,303	4,303	380	68	460	219	175	411	658	2,820	202	213
1. Protein-energy malnutrition	—	—	—	—	—	—	—	—	—	—	—	—	—
2. Deficiency anaemia	5,606	1,303	4,303	380	68	460	219	175	411	658	2,820	202	213
3. Other nutritional deficiencies	—	—	—	—	—	—	—	—	—	—	—	—	—
II. Non-communicable diseases	1,396,109	679,995	716,115	38,888	45,468	342,590	113,811	139,238	26,634	45,805	368,817	91,809	183,050
F. Malignant neoplasms	89,060	44,531	44,529	450	468	17,111	13,189	13,312	323	343	22,910	9,917	11,036
1. Mouth and oropharynx cancers	4,012	2,854	1,159	1	19	1,673	646	514	1	11	576	227	344
2. Oesophagus cancer	872	551	321	—	—	207	175	169	—	—	98	109	114
3. Stomach cancer	1,520	933	587	1	—	323	280	329	—	—	197	138	252
4. Colorectal cancer	12,283	6,779	5,504	1	7	2,217	2,161	2,394	1	12	1,743	1,455	2,293
5. Liver cancer ^(d)	82	63	20	1	—	27	17	17	1	—	7	4	8
6. Gallbladder cancer	201	94	107	1	—	30	28	35	—	—	26	29	52
7. Pancreas cancer	550	295	256	—	—	105	91	99	1	—	85	57	113
8. Lung cancer	5,807	3,626	2,181	1	3	1,086	1,395	1,141	—	1	873	713	593
9. Bone and connective tissue cancer	903	440	463	31	48	234	64	63	38	50	234	60	82
10. Melanoma	5,020	3,673	1,347	2	55	2,174	756	686	1	9	307	501	529
11. Non-melanoma skin cancers	1,023	596	426	—	1	241	156	198	—	—	166	83	177
12. Breast cancer	21,032	—	21,032	—	—	—	—	—	—	9	13,091	4,344	3,589
13. Cervix cancer	1,000	—	1,000	—	—	—	—	—	—	10	798	91	101
14. Corpus uteri cancer	1,399	—	1,399	—	—	—	—	—	—	—	728	351	320
15. Ovary cancer	1,062	—	1,062	—	—	—	—	—	5	29	641	186	200
16. Prostate cancer	12,981	12,981	—	—	—	3,539	4,596	4,845	—	—	—	—	—
17. Testicular cancer	560	560	—	5	49	489	11	5	—	—	—	—	—
18. Bladder cancer	2,169	1,702	467	—	2	409	532	760	—	—	97	126	244

(continued)

Annex Table 5 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
19. Kidney cancer	1,962	12	3	475	346	316	19	4	313	210	264
20. Brain cancer	1,305	138	107	501	43	35	76	42	291	34	38
21. Thyroid cancer	936	—	8	171	31	22	2	25	552	68	57
22. Lymphoma	3,640	33	83	1,043	431	443	15	51	699	340	502
23. Multiple myeloma	855	—	—	172	137	174	—	1	102	99	170
24. Leukaemia	2,531	136	72	605	300	284	105	43	458	205	325
25. Larynx cancer	874	—	—	334	271	188	—	—	27	25	30
26. Eye cancer	518	41	9	139	64	40	34	12	108	33	37
27. Other malignant neoplasms	3,963	46	2	919	656	557	24	34	693	429	603
G. Other neoplasms	3,251	25	35	277	174	236	49	12	1,863	236	345
1. Uterine myomas	1,549	—	—	—	—	—	—	2	1,460	61	27
2. Benign neoplasms of meninges and brain	695	244	6	135	41	51	20	5	264	71	90
3. Other benign neoplasms	1,007	14	28	142	133	185	29	5	140	104	228
H. Diabetes mellitus	93,502	224	469	25,226	11,952	12,203	189	402	18,695	8,860	15,283
1. Type 1 diabetes	8,508	203	421	3,171	621	473	164	328	2,265	400	463
2. Type 2 diabetes	84,995	22	48	22,055	11,331	11,730	25	74	16,430	8,460	14,820
I. Endocrine and metabolic disorders	16,424	1,863	930	3,423	1,066	1,413	1,156	691	2,955	888	2,039
1. Non-deficiency anaemia	4,885	455	363	1,228	336	404	274	226	1,020	245	334
a. Haemolytic anaemia	2,315	330	286	677	17	1	210	189	574	26	4
b. Other non-deficiency anaemia	2,570	125	78	551	318	402	64	37	446	220	330
2. Cystic fibrosis	1,810	346	212	355	17	11	326	192	324	16	13
3. Haemophilia	148	31	22	79	9	6	—	—	—	—	—
4. Other endocrine and metabolic disorders	9,581	1,031	333	1,761	704	992	556	273	1,611	628	1,693
J. Mental disorders	394,544	12,127	24,720	139,786	12,279	3,888	6,363	23,049	157,684	9,593	5,054
1. Substance use disorders	42,786	—	5,495	25,069	697	307	8	1,300	9,647	186	76

(continued)

Annex Table 5 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
a. Alcohol dependence and harmful use ^(e)	20,363	—	2,684	12,764	443	209	—	194	4,028	34	8
b. Heroin or polydrug dependence and harmful use	12,681	—	1,343	7,571	211	87	8	504	2,824	89	44
c. Benzodiazepine dependence and harmful use	2,967	—	58	1,165	26	9	—	107	1,517	62	24
d. Cannabis dependence and harmful use	5,820	—	1,289	3,182	16	2	—	382	948	—	—
e. Other drug dependence and harmful use	955	—	122	387	1	—	—	113	330	1	—
2. Schizophrenia	37,974	20	1,924	17,133	1,386	623	33	706	13,346	1,654	1,148
3. Anxiety and depression	223,523	1,598	9,102	65,522	7,407	1,380	2,741	14,060	113,292	5,938	2,483
4. Bipolar disorder	9,984	—	628	4,129	192	69	—	568	4,125	169	102
5. Personality disorders ^(f)	32,937	—	1,466	13,104	1,187	690	—	1,284	12,630	1,448	1,128
6. Eating disorders	6,570	29	175	214	1	—	292	3,749	2,105	4	1
a. Anorexia nervosa	3,248	29	175	214	1	—	117	1,550	1,158	4	1
b. Bulimia nervosa	3,322	—	—	—	—	—	176	2,199	947	—	—
c. Other eating disorders	—	—	—	—	—	—	—	—	—	—	—
7. Childhood conditions	40,771	10,480	5,929	14,615	1,409	819	3,289	1,381	2,540	194	115
a. Attention-deficit hyperactivity disorder	10,613	5,885	1,558	87	—	—	2,455	591	36	—	—
b. Autism spectrum disorders	30,157	4,595	4,371	14,527	1,409	819	833	790	2,503	194	115
8. Other mental disorders	—	—	—	—	—	—	—	—	—	—	—
K. Nervous system and sense organ disorders	263,934	124,029	4,018	37,672	26,724	50,726	3,739	4,240	37,071	20,563	74,292
1. Dementia	64,914	23,615	4	1,881	4,572	17,157	—	—	831	4,131	36,338
2. Epilepsy	14,843	8,194	1,377	4,618	532	412	1,006	972	3,718	443	510
3. Parkinson's disease	20,688	10,015	—	1,654	2,702	5,658	—	—	798	2,868	7,007
4. Multiple sclerosis	4,621	1,441	3	1,254	126	36	18	60	2,586	326	190
5. Motor neurone disease	608	321	—	136	91	94	2	—	92	79	113

(continued)

Annex Table 5 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
6. Huntington's chorea	898	—	7	383	55	21	—	1	310	82	38
7. Muscular dystrophy	381	201	78	—	—	—	73	29	—	—	—
8. Sense organ disorders	113,231	281	552	18,725	17,031	25,809	177	417	11,634	11,005	27,600
a. Glaucoma-related blindness	3,722	—	—	259	583	798	—	—	175	629	1,279
b. Cataract-related blindness	2,220	5	2	127	209	486	3	1	138	305	944
c. Macular degeneration	10,266	—	—	2	320	3,243	—	—	2	338	6,360
d. Adult-onset hearing loss	68,178	—	181	13,995	12,868	16,268	—	70	6,881	6,665	11,250
e. Refractive errors	17,846	146	277	2,241	1,712	3,459	53	259	2,425	1,765	5,510
f. Other vision loss	10,998	131	92	2,100	1,340	1,555	121	86	2,014	1,303	2,256
9. Migraine	24,699	621	1,188	4,764	333	153	520	1,979	14,122	651	368
10. Other nervous system and sense organ disorders	19,051	2,406	913	4,257	1,281	1,385	1,942	782	2,979	978	2,128
L. Cardiovascular disease	119,823	57,802	928	21,804	15,082	19,433	353	546	17,013	12,665	31,444
1. Rheumatic heart disease	1,274	353	4	72	73	200	2	3	219	178	518
2. Ischaemic heart disease	50,644	23,262	13	7,217	6,038	9,989	4	9	5,165	6,128	16,077
3. Stroke	41,409	19,929	442	9,103	5,379	4,303	267	342	7,817	3,986	9,068
4. Inflammatory heart disease	4,220	2,366	81	1,072	538	633	28	53	733	396	645
5. Hypertensive heart disease	735	315	5	111	83	113	2	4	106	95	213
6. Non-rheumatic valvular disease	1,365	574	22	221	110	205	13	18	246	147	368
7. Aortic aneurysm	206	151	—	25	52	74	—	—	6	14	35
8. Peripheral vascular disease	13,798	8,431	28	3,029	2,338	3,023	11	34	1,731	1,094	2,497
9. Other cardiovascular disease	6,172	2,420	72	953	471	894	25	83	991	628	2,025
M. Chronic respiratory disease	150,444	79,342	5,979	31,134	14,833	18,397	6,606	5,973	31,642	10,284	16,597
1. Chronic obstructive pulmonary disease (COPD)	57,130	33,965	17	13,534	9,724	10,630	27	78	8,961	5,551	8,548
2. Asthma	69,057	31,258	8,455	14,346	1,627	1,139	6,406	5,892	20,484	2,521	2,495
3. Other chronic respiratory diseases	24,258	14,119	526	3,255	3,482	6,629	173	3	2,196	2,213	5,554

(continued)

Annex Table 5 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
N. Diseases of the digestive system	37,294	450	529	11,068	3,520	3,557	301	497	9,693	2,985	4,694
1. Peptic ulcer disease	2,180	—	19	881	200	26	—	3	818	99	134
2. Cirrhosis of the liver ^(a)	43	—	—	11	6	6	—	—	10	4	4
3. Appendicitis	419	50	60	92	6	3	38	60	99	6	4
4. Intestinal obstruction	1,906	20	16	320	232	291	10	9	448	229	332
5. Diverticulitis	3,783	—	1	849	541	575	—	1	595	492	730
6. Gallbladder and bile duct disease	1,152	1	5	205	76	59	3	52	578	96	77
7. Pancreatitis	309	2	8	132	23	18	2	8	78	16	22
8. Inflammatory bowel disease	16,533	144	373	5,950	1,197	828	133	323	5,518	1,055	1,012
9. Vascular insufficiency of bowel	374	1	1	41	41	68	1	1	85	49	87
10. Other digestive system diseases	10,596	231	46	2,588	1,198	1,683	115	40	1,463	940	2,292
O. Genitourinary diseases	43,834	18	571	9,102	3,055	4,498	257	3,661	19,215	1,671	1,785
1. Nephritis and nephrosis ^(h)	2,323	18	42	803	248	260	8	33	582	149	181
2. Benign prostatic hypertrophy	6,965	—	—	965	2,249	3,751	—	—	—	—	—
3. Urinary incontinence	8,467	—	—	730	558	487	—	66	3,778	1,371	1,476
4. Infertility	16,572	—	529	6,604	—	—	—	619	8,819	—	—
5. Other genitourinary diseases	9,506	—	—	—	—	—	249	2,941	6,037	151	128
P. Skin diseases	18,513	894	1,808	4,802	879	738	891	1,976	3,249	1,134	2,144
1. Eczema	3,058	231	148	628	62	50	657	494	719	45	24
2. Acne	4,103	314	1,161	604	—	—	123	991	910	—	—
3. Psoriasis	3,947	162	428	2,144	225	116	42	147	581	70	30
4. Ulcers	7,405	186	71	1,426	591	572	69	344	1,038	1,018	2,090
5. Other skin diseases	—	—	—	—	—	—	—	—	—	—	—
Q. Musculoskeletal diseases	94,846	330	826	21,502	8,601	8,972	549	1,176	26,919	10,390	15,580
1. Rheumatoid arthritis	15,782	84	197	2,164	1,032	903	379	510	6,059	2,225	2,229
2. Osteoarthritis	31,137	—	20	5,445	3,457	4,363	—	—	4,052	4,486	9,315

(continued)

Annex Table 5 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
3. Back pain ⁽ⁱ⁾	27,411	143	349	7,973	2,463	2,278	98	408	8,955	2,301	2,443
4. Slipped disc	6,886	4	44	2,386	739	686	8	34	1,555	619	812
5. Occupational overuse syndrome	4,945	—	5	638	50	3	—	36	4,114	92	5
6. Systemic lupus erythematosus (SLE)	898	—	1	23	31	37	1	43	550	104	108
7. Gout	2,101	—	14	1,050	359	308	—	10	122	106	132
8. Other musculoskeletal diseases	5,686	2,704	195	1,824	470	394	64	136	1,511	457	537
R. Congenital anomalies	38,531	7,000	3,023	10,398	1,142	759	4,843	2,113	7,656	866	730
1. Anencephaly	—	—	—	—	—	—	—	—	—	—	—
2. Spina bifida	869	434	68	237	22	8	92	65	240	25	14
3. Congenital heart disease	5,595	2,142	577	1,660	168	122	471	305	1,095	135	136
4. Cleft lip and/or palate	488	281	41	150	18	13	41	29	110	14	14
5. Digestive system malformations	92	53	4	14	1	1	24	3	10	1	1
a. Anorectal atresia	42	22	2	6	—	—	12	1	5	—	—
b. Oesophageal atresia	43	27	2	8	1	—	9	1	5	1	—
c. Other digestive system malformations	7	3	—	—	—	—	3	—	—	—	—
6. Urogenital tract malformations	798	549	76	317	28	14	42	29	144	22	12
a. Renal agenesis ⁽ⁱ⁾	96	61	9	33	3	1	8	6	19	2	1
b. Other urogenital tract malformations ^(k)	702	489	67	284	25	13	34	23	125	20	11
7. Abdominal wall defect	198	113	19	60	3	—	22	14	46	3	1
8. Down syndrome	5,588	3,255	477	1,762	205	136	471	335	1,272	145	110
9. Other chromosomal disorders	18,715	10,617	1,554	5,745	671	449	1,631	1,161	4,411	506	389
10. Other congenital anomalies	6,187	3,567	2,865	453	27	16	2,050	172	327	16	54
S. Oral conditions	23,251	10,884	1,018	6,489	1,221	1,093	1,013	984	6,856	1,529	1,986
1. Dental caries	11,611	5,788	734	3,725	417	281	600	707	3,689	371	457

(continued)

Annex Table 5 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
		Males	Females	Males	Females	Males	Females	Males	Females	Males	Females	
2. Periodontal disease	559	284	1	7	196	43	29	1	7	194	43	40
3. Edentulism	4,692	3,021	—	2	496	545	628	1	3	876	886	1,255
4. Pulpitis	6,389	3,240	431	275	2,073	216	155	412	267	2,096	230	234
5. Other oral conditions	—	—	—	—	—	—	—	—	—	—	—	—
Z. Ill-defined conditions	8,856	5,808	—	145	2,795	95	14	—	143	5,395	229	41
1. Sudden infant death syndrome	—	—	—	—	—	—	—	—	—	—	—	—
2. Chronic fatigue syndrome	8,856	3,049	—	145	2,795	95	14	—	143	5,395	229	41
III. Injuries	83,648	57,787	1,924	4,163	40,773	6,329	4,598	1,158	1,568	13,141	3,218	6,776
T. Unintentional injuries	77,833	53,870	1,893	3,898	37,732	5,929	4,419	1,145	1,484	11,757	2,982	6,595
1. Road traffic accidents	17,947	12,501	116	646	9,978	1,274	487	64	311	3,914	689	468
2. Other transport accidents	4,440	3,305	126	308	2,462	283	126	48	94	784	116	92
3. Poisoning	425	197	13	14	120	26	24	13	13	89	51	63
4. Falls	26,676	17,190	833	1,418	11,460	1,723	1,756	460	379	2,522	1,269	4,856
5. Fires, burns and scalds	4,495	2,667	184	260	1,734	279	210	189	181	1,071	181	206
6. Drowning	75	68	1	6	52	7	2	—	1	5	1	—
7. Sports injuries	848	515	19	59	352	53	32	13	28	192	39	62
8. Natural and environmental factors	1,103	652	30	45	438	79	59	16	37	287	51	59
9. Machinery accidents	6,404	5,885	42	227	3,906	925	785	11	22	316	79	90
10. Other unintentional injuries ^(b)	15,420	10,890	529	913	7,229	1,280	938	331	418	2,577	507	697
Suffocation and foreign bodies	1,311	909	48	65	609	107	80	48	51	230	35	38
Adverse effects of medical treatment	1,792	1,072	20	53	632	190	177	9	21	297	121	272
Other unintentional injuries n.e.c.	12,317	8,909	461	795	5,987	984	681	274	346	12,317	8,909	3,408

(continued)

Annex Table 5 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
U. Intentional injuries	5,815	31	265	3,041	400	179	14	84	1,384	235	181
1. Suicide and self-inflicted injuries	826	1	24	326	58	31	2	28	281	42	33
2. Homicide and violence	4,982	30	241	2,710	341	148	12	57	1,103	194	148
3. Legal intervention and war	7	—	—	6	1	—	—	—	—	—	—
Australian population ('000)	19,881	2,041	1,404	5,292	656	478	1,938	1,349	5,311	694	718
PYLD per 1,000 population	78.4	24.1	38.4	77.0	187.6	305.0	18.0	38.8	76.0	140.1	267.4

Notes

- (a) Excludes HIV/AIDS.
- (b) Includes hepatitis B-related liver cancer and cirrhosis.
- (c) Includes hepatitis C-related liver cancer and cirrhosis.
- (d) Excludes liver cancer related to hepatitis B and C.
- (e) Includes alcoholic cirrhosis.
- (f) Excludes those with any other comorbid mental disorders.
- (g) Excludes alcoholic and hepatic cirrhosis.
- (h) Excludes diabetic-, congenital- and poisoning-related renal failure.
- (i) Includes both acute and chronic back pain.
- (j) Includes renal failure due to dysplasia.
- (k) Includes polycystic renal failure.
- (l) Includes suffocation and foreign bodies, adverse effects of medical treatment, other mechanical force injuries and other unintentional injuries.

Annex Table 6: Years lived with disability (YLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
All causes	1,353,992	94,603	72,722	309,705	90,986	87,002	73,199	82,802	336,416	81,055	125,503		
I. Communicable diseases, maternal and neonatal conditions	49,021	12,578	1,386	7,687	958	808	10,818	2,747	10,054	919	1,067		
A. Infectious and parasitic diseases	14,021	1,149	626	5,182	486	412	948	807	3,405	428	577		
1. Tuberculosis	169	3	13	49	11	11	4	10	54	7	8		
2. Sexually transmitted diseases ^(a)	1,879	5	26	39	—	—	24	437	1,317	21	10		
a. Syphilis	24	4	1	7	—	—	5	2	3	—	—		
b. Chlamydia	1,134	—	22	26	—	—	14	264	789	13	6		
c. Gonorrhoea	28	—	3	6	—	—	—	5	13	—	—		
d. Other sexually transmitted diseases	694	—	—	—	—	—	4	166	511	8	4		
3. HIV/AIDS	4,153	6	341	3,333	40	4	6	61	355	8	—		
4. Diarrhoeal diseases	1,478	328	72	249	22	22	317	90	309	29	41		
5. Childhood immunisable diseases	189	64	7	18	2	1	58	8	26	2	1		
a. Diphtheria	—	—	—	—	—	—	—	—	—	—	—		
b. Whooping cough	147	41	7	18	2	1	41	8	26	2	1		
c. Tetanus	—	—	—	—	—	—	—	—	—	—	—		
d. Poliomyelitis	—	—	—	—	—	—	—	—	—	—	—		
e. Measles	1	—	—	—	—	—	—	—	—	—	—		
f. Rubella	25	16	—	—	—	—	8	—	—	—	—		
g. <i>Haemophilus influenzae</i> type b (Hib)	16	7	—	—	—	—	8	—	—	—	—		
6. Meningitis	1,331	601	56	98	14	7	372	54	112	10	7		
7. Septicaemia	1,291	727	21	279	157	205	48	24	214	94	183		

(continued)

Annex Table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females							
		Males		Females		Males		Females		Males		Females			
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74
8. Arbovirus infection	1,271	657	614	2	58	543	42	13	8	61	506	25	15		
a. Ross River virus	649	307	342	1	26	256	18	5	1	27	292	13	8		
b. Barmah Forest virus	253	126	128	—	8	106	8	4	—	9	111	5	2		
c. Dengue	5	2	2	—	—	2	—	—	—	—	1	—	—		
d. Other arbovirus infection	364	222	142	—	23	180	15	4	6	24	101	6	4		
9. Hepatitis	831	548	283	20	15	400	85	27	11	13	176	42	42		
a. Hepatitis A	35	20	15	5	3	11	1	1	3	3	8	1	1		
b. Hepatitis B ^(b)	256	161	95	7	12	90	37	13	7	10	52	19	7		
c. Hepatitis C ^(c)	430	285	145	1	—	224	46	13	1	—	115	23	7		
d. Other hepatitis	110	82	28	7	—	75	—	—	—	—	—	—	28		
10. Malaria	2	1	1	—	—	—	—	—	—	—	—	—	—		
11. Trachoma	121	55	66	—	1	42	11	1	—	1	49	14	2		
12. Other infectious and parasitic diseases	1,305	424	881	54	15	131	103	121	101	48	287	176	269		
B. Acute respiratory infections	11,752	5,877	5,875	2,681	686	2,044	234	231	2,224	819	2,258	285	289		
1. Lower respiratory tract infections	3,824	1,900	1,924	426	151	986	157	181	265	236	1,035	161	226		
2. Upper respiratory tract infections	3,340	1,577	1,763	611	282	595	56	34	583	359	703	82	36		
3. Otitis media	4,588	2,400	2,188	1,644	253	464	22	17	1,376	223	519	42	27		
C. Maternal conditions	1,926	—	1,926	—	—	—	—	—	1	376	1,549	—	—		
1. Maternal haemorrhage	95	—	95	—	—	—	—	—	—	18	77	—	—		
2. Maternal sepsis	305	—	305	—	—	—	—	—	1	95	209	—	—		
3. Hypertensive disorders of pregnancy	856	—	856	—	—	—	—	—	1	174	681	—	—		
4. Obstructed labour	146	—	146	—	—	—	—	—	—	24	122	—	—		
5. Abortion	25	—	25	—	—	—	—	—	—	12	14	—	—		
6. Other maternal conditions	499	—	499	—	—	—	—	—	—	53	446	—	—		

(continued)

Annex Table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
D. Neonatal causes	15,584	8,361	—	—	—	—	7,223	—	—	—	—
1. Birth trauma and asphyxia	5,220	3,018	—	—	—	—	2,202	—	—	—	—
2. Low birthweight	6,885	3,452	—	—	—	—	3,433	—	—	—	—
3. Neonatal infections	1,844	1,206	—	—	—	—	638	—	—	—	—
4. Other conditions arising in the perinatal period	1,635	686	—	—	—	—	949	—	—	—	—
E. Nutritional deficiencies	5,739	1,324	73	461	164	421	745	2,842	205	202	
1. Protein-energy malnutrition	—	—	—	—	—	—	—	—	—	—	
2. Deficiency anaemia	5,739	1,324	73	461	164	421	745	2,842	205	202	
3. Other nutritional deficiencies	—	—	—	—	—	—	—	—	—	—	
II. Non-communicable diseases	1,260,568	602,698	64,863	286,998	84,981	59,325	78,160	321,317	78,524	120,544	
F. Malignant neoplasms	87,463	44,223	545	20,595	9,650	381	412	25,976	8,733	7,737	
1. Mouth and oropharynx cancers	3,942	2,794	2	1,872	350	2	22	653	215	257	
2. Oesophagus cancer	889	556	—	270	123	—	—	131	116	85	
3. Stomach cancer	1,400	864	1	386	210	—	—	228	136	172	
4. Colorectal cancer	11,873	6,646	12	2,787	1,702	1	18	2,059	1,453	1,695	
5. Liver cancer ^(e)	90	69	—	35	14	1	—	9	4	7	
6. Gallbladder cancer	188	90	—	37	24	—	—	32	31	36	
7. Pancreas cancer	561	299	—	137	74	1	—	109	58	95	
8. Lung cancer	5,848	3,523	4	1,422	771	1	1	1,143	722	458	
9. Bone and connective tissue cancer	863	417	33	219	50	51	46	230	57	62	
10. Melanoma	4,851	3,626	85	2,301	519	2	16	402	446	361	
11. Non-melanoma skin cancers	1,118	673	1	296	200	—	—	185	96	164	
12. Breast cancer	20,440	—	—	—	—	—	22	14,933	3,361	2,123	
13. Cervix cancer	875	—	—	—	—	—	21	736	60	58	

(continued)

Annex Table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+			
3. Haemophilia	59	—	—	—	—	—	—	—	—	—	—	—	—	—
4. Other endocrine and metabolic disorders	10,296	1,132	366	1,933	773	1,089	585	286	1,693	660	1,779	—	—	—
J. Mental disorders	327,391	28,628	47,242	68,849	2,617	736	21,457	47,112	108,693	1,386	671	—	—	—
1. Substance use disorders	38,817	—	13,566	14,865	354	113	46	3,699	6,135	36	2	—	—	—
a. Alcohol dependence and harmful use ^(e)	19,861	—	4,782	10,550	336	107	—	386	3,672	26	2	—	—	—
b. Heroin or polydrug dependence and harmful use	10,287	—	4,763	2,715	14	6	46	1,701	1,033	9	—	—	—	—
c. Benzodiazepine dependence and harmful use	2,627	—	205	891	3	—	—	362	1,163	2	—	—	—	—
d. Cannabis dependence and harmful use	5,203	—	3,520	552	1	—	—	983	147	—	—	—	—	—
e. Other drug dependence and harmful use	839	—	296	156	—	—	—	268	120	—	—	—	—	—
2. Schizophrenia	27,250	186	9,795	4,670	15	7	181	3,754	8,614	19	10	—	—	—
3. Anxiety and depression	191,452	9,554	17,868	36,087	1,430	269	15,507	29,945	80,482	295	14	—	—	—
4. Bipolar disorder	7,679	—	2,672	1,220	2	—	—	2,450	1,328	4	2	—	—	—
5. Personality disorders ^(f)	32,587	—	3,130	11,955	816	347	—	2,622	12,044	1,032	642	—	—	—
6. Eating disorders	5,921	103	211	52	—	—	828	4,636	90	—	—	—	—	—
a. Anorexia nervosa	2,835	103	211	52	—	—	407	2,061	—	—	—	—	—	—
b. Bulimia nervosa	3,087	—	—	—	—	—	421	2,575	90	—	—	—	—	—
c. Other eating disorders	—	—	—	—	—	—	—	—	—	—	—	—	—	—
7. Childhood conditions	23,684	18,784	—	—	—	—	4,894	6	—	—	—	—	—	—
a. Attention-deficit hyperactivity disorder	9,928	7,082	—	—	—	—	2,840	6	—	—	—	—	—	—
b. Autism spectrum disorders	13,756	11,702	—	—	—	—	2,054	—	—	—	—	—	—	—
8. Other mental disorders	—	—	—	—	—	—	—	—	—	—	—	—	—	—

(continued)

Annex Table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
K. Nervous system and sense organ disorders	258,638	7,409	6,247	43,767	28,562	36,084	5,506	8,644	39,044	26,120	57,255		
1. Dementia	70,296	4	11	3,956	6,599	14,988	—	—	2,619	8,683	33,435		
2. Epilepsy	8,601	2,965	605	818	147	96	2,226	581	836	171	156		
3. Parkinson's disease	21,157	—	—	3,238	3,257	4,128	—	—	2,607	4,476	3,451		
4. Multiple sclerosis	3,628	18	100	1,002	5	3	79	201	2,196	10	15		
5. Motor neurone disease	622	—	—	152	96	81	2	—	106	87	98		
6. Huntington's chorea	818	—	35	346	26	10	—	7	356	26	12		
7. Muscular dystrophy	258	189	—	—	—	—	69	—	—	—	—		
8. Sense organ disorders	112,718	383	1,073	29,229	17,192	15,439	237	857	18,678	11,675	17,955		
a. Glaucoma-related blindness	3,668	—	—	868	586	244	—	—	866	694	411		
b. Cataract-related blindness	2,343	5	2	139	228	510	3	1	153	337	966		
c. Macular degeneration	11,642	—	—	13	1,132	3,238	—	—	14	1,338	5,906		
d. Adult-onset hearing loss	64,853	—	699	22,983	11,920	7,052	—	432	12,315	5,834	3,618		
e. Refractive errors	18,761	224	286	2,697	1,941	3,094	90	343	2,861	2,107	5,119		
f. Other vision loss	11,451	154	87	2,529	1,386	1,301	143	81	2,470	1,364	1,935		
9. Migraine	21,841	1,523	3,539	910	1	—	955	6,217	8,671	15	10		
10. Other nervous system and sense organ disorders	18,698	2,327	883	4,117	1,238	1,340	1,938	781	2,974	976	2,124		
L. Cardiovascular disease	104,429	1,440	1,068	25,373	12,354	12,628	1,004	523	19,360	10,865	19,815		
1. Rheumatic heart disease	1,136	5	6	78	85	152	3	4	257	192	355		
2. Ischaemic heart disease	45,354	12	30	9,203	6,049	6,822	12	23	7,520	6,010	9,673		
3. Stroke	33,763	1,220	801	9,509	3,035	2,579	831	271	7,364	2,453	5,700		
4. Inflammatory heart disease	3,689	121	92	1,059	450	350	84	64	764	346	359		
5. Hypertensive heart disease	678	6	5	137	82	62	7	5	144	95	136		
6. Non-rheumatic valvular disease	1,379	28	24	216	124	189	25	22	264	172	315		

(continued)

Annex Table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
7. Aortic aneurysm	209	—	—	27	53	73	—	—	6	14	35		
8. Peripheral vascular disease	12,888	19	46	4,273	2,045	1,583	21	65	2,217	1,061	1,560		
9. Other cardiovascular disease	5,333	28	66	871	431	817	21	69	824	522	1,684		
M. Chronic respiratory disease	115,398	22,246	1,480	23,110	6,332	7,067	16,594	6,851	18,661	5,789	7,267		
1. Chronic obstructive pulmonary disease (COPD)	39,543	61	260	16,935	3,429	2,334	78	247	10,252	3,222	2,726		
2. Asthma	59,054	21,828	1,066	3,970	544	241	16,393	6,603	6,842	989	579		
3. Other chronic respiratory diseases	16,801	357	154	2,206	2,360	4,493	123	2	1,567	1,578	3,962		
N. Diseases of the digestive system	30,246	830	1,175	9,361	2,282	2,038	671	1,041	8,307	1,809	2,732		
1. Peptic ulcer disease	2,196	1	39	901	180	8	—	6	866	75	119		
2. Cirrhosis of the liver ^(a)	44	—	1	12	7	4	—	1	12	4	3		
3. Appendicitis	419	53	58	90	6	3	41	60	97	6	4		
4. Intestinal obstruction	1,815	29	17	448	247	122	10	18	630	172	123		
5. Diverticulitis	3,745	—	6	1,132	486	324	—	1	850	511	435		
6. Gallbladder and bile duct disease	1,166	2	6	209	78	58	3	55	584	96	75		
7. Pancreatitis	313	2	8	134	23	18	2	9	79	16	22		
8. Inflammatory bowel disease	11,589	553	1,001	4,248	219	84	523	854	3,899	136	74		
9. Vascular insufficiency of bowel	359	1	1	64	51	37	1	6	118	40	40		
10. Other digestive system diseases	8,600	190	38	2,123	983	1,381	92	32	1,172	753	1,837		
O. Genitourinary diseases	41,161	53	1,576	9,198	3,633	2,361	1,041	7,777	13,244	1,227	1,051		
1. Nephritis and nephrosis ^(b)	2,275	32	75	849	208	188	20	63	589	124	127		
2. Benign prostatic hypertrophy	7,378	—	—	2,705	2,883	1,790	—	—	—	—	—		

(continued)

Annex Table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
3. Urinary incontinence	8,263	—	—	898	542	383	1	217	4,271	1,053	898
4. Infertility	14,344	21	1,502	4,746	—	—	19	1,822	6,235	—	
5. Other genitourinary diseases	8,901	—	—	—	—	—	1,001	5,676	2,149	50	
P. Skin diseases	18,130	1,445	1,679	4,371	843	651	1,593	1,778	2,472	1,148	2,150
1. Eczema	2,730	371	47	555	31	27	1,210	42	413	31	2
2. Acne	3,899	1,988	1,013	329	—	—	242	1,198	470	—	—
3. Psoriasis	3,923	846	578	2,040	174	80	58	192	523	53	19
4. Ulcers	7,578	2,892	41	1,447	638	544	82	346	1,065	1,063	2,129
5. Other skin diseases	—	—	—	—	—	—	—	—	—	—	—
Q. Musculoskeletal diseases	98,481	41,832	1,287	26,876	7,684	5,222	1,303	1,522	34,108	10,420	9,297
1. Rheumatoid arthritis	15,215	4,296	214	2,702	724	343	958	513	7,491	1,302	654
2. Osteoarthritis	34,204	14,429	1	7,754	3,863	2,754	—	—	7,338	6,077	6,360
3. Back pain ⁽ⁱ⁾	29,484	14,355	541	9,746	2,182	1,610	206	610	10,704	2,000	1,610
4. Slipped disc	6,089	3,415	13	2,711	367	180	29	84	1,956	401	205
5. Occupational overuse syndrome	4,953	697	9	663	24	—	—	65	4,177	13	1
6. Systemic lupus erythematosus (SLE)	949	101	1	26	34	41	1	45	579	110	114
7. Gout	1,813	1,523	2	1,294	90	52	1	59	131	97	2
8. Other musculoskeletal diseases	5,773	3,015	235	1,980	400	242	108	146	1,734	420	350
R. Congenital anomalies	16,331	9,595	120	470	44	23	6,173	109	379	37	38
1. Anencephaly	—	—	—	—	—	—	—	—	—	—	—
2. Spina bifida	351	179	179	—	—	—	171	—	—	—	—
3. Congenital heart disease	2,835	1,753	25	130	20	7	867	32	153	22	8
4. Cleft lip and/or palate	189	111	—	—	—	—	78	—	—	—	—
5. Digestive system malformations	65	37	—	—	—	—	27	—	—	—	—
a. Anorectal atresia	30	16	—	—	—	—	14	—	—	—	—

(continued)

Annex Table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
b. Oesophageal atresia	28	18	—	—	—	—	10	—	—	—	—	—
c. Other digestive system malformations	7	3	—	—	—	—	3	—	—	—	—	—
6. Urogenital tract malformations	506	339	—	131	12	8	73	—	80	8	6	6
a. Renal agenesis ⁽ⁱ⁾	46	28	—	3	—	—	16	—	2	—	—	—
b. Other urogenital tract malformations ^(k)	460	311	—	128	12	8	57	—	78	8	6	6
7. Abdominal wall defect	89	52	—	—	—	—	37	—	—	—	—	—
8. Down syndrome	2,180	1,285	—	—	—	—	895	—	—	—	—	—
9. Other chromosomal disorders	7,297	4,191	—	—	—	—	3,105	—	—	—	—	—
10. Other congenital anomalies	2,820	1,647	1,323	209	12	7	918	77	146	7	24	24
S. Oral conditions	24,406	11,383	1,114	1,098	1,186	626	1,062	1,065	8,464	1,459	972	972
1. Dental caries	12,088	6,026	6,061	789	427	285	631	760	3,819	375	476	476
2. Periodontal disease	570	280	289	20	19	7	5	19	237	19	9	9
3. Edentulism	5,264	1,880	3,384	7	526	179	3	12	2,281	836	252	252
4. Pulpitis	6,484	3,197	3,287	283	214	155	424	274	2,127	228	235	235
5. Other oral conditions	—	—	—	—	—	—	—	—	—	—	—	—
Z. Ill-defined conditions	8,781	2,984	5,797	283	2,701	—	—	280	5,517	—	—	—
1. Sudden infant death syndrome	—	—	—	—	—	—	—	—	—	—	—	—
2. Chronic fatigue syndrome	8,781	2,984	5,797	283	2,701	—	—	280	5,517	—	—	—
III. Injuries	44,402	28,902	15,500	6,474	15,019	1,213	3,055	1,895	5,046	1,612	3,892	3,892
T. Unintentional injuries	41,263	26,729	14,534	5,700	13,726	1,209	3,018	1,642	4,380	1,609	3,886	3,886
1. Road traffic accidents	6,073	4,354	1,719	1,292	2,651	26	202	645	771	59	41	41
2. Other transport accidents	2,873	2,151	722	627	1,056	18	161	195	317	23	26	26
3. Poisoning	326	139	187	36	61	8	22	20	58	51	37	37
4. Falls	13,995	6,572	7,424	1,025	2,400	997	992	262	1,364	1,287	3,518	3,518

(continued)

Annex Table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+			
5. Fires, burns and scalds	2,438	1,491	948	606	249	619	10	75+	6	495	33	395	12	12
6. Drowning	53	49	4	1	20	28	—	—	—	1	2	1	—	—
7. Sports injuries	578	344	234	71	112	146	8	6	6	44	44	96	16	35
8. Natural and environmental factors	711	430	282	104	52	258	8	7	7	66	70	124	11	11
9. Machinery accidents	4,657	4,287	369	184	901	2,933	206	63	63	37	47	270	11	4
10. Other unintentional injuries ⁽ⁱ⁾	9,558	6,914	2,644	1,692	1,386	3,573	185	78	78	997	324	982	139	202
Suffocation and foreign bodies	740	555	185	142	94	309	7	2	2	142	7	33	1	2
Adverse effects of medical treatment	1,417	837	580	65	123	508	87	53	53	22	70	241	93	154
Other unintentional injuries n.e.c.	7,401	5,522	1,879	1,485	1,168	2,755	91	22	22	833	247	7,401	5,522	1,879
U. Intentional injuries	3,139	2,173	966	90	774	1,293	12	4	4	38	253	666	3	5
1. Suicide and self-inflicted injuries	537	283	254	3	76	200	2	3	3	3	67	180	2	2
2. Homicide and violence	2,597	1,885	712	88	697	1,089	10	1	1	35	186	486	1	4
3. Legal intervention and war	5	5	—	—	1	4	—	—	—	—	—	—	—	—
Australian population ('000)	19,881	9,872	10,010	2,041	1,404	5,292	656	478	478	1,938	1,349	5,311	694	718
YLD per 1,000 population	68.1	66.4	69.8	46.4	51.8	58.5	138.7	182.0	182.0	37.8	61.4	63.3	116.8	174.8
Alternative burden of disease categories														
Diabetes mellitus (attributable)	124,015	64,907	59,108	977	626	43,997	10,826	8,481	8,481	852	713	34,284	9,996	13,262
Anxiety and depression (attributable)	193,051	65,924	127,127	9,555	17,880	36,544	1,571	374	374	15,508	29,968	81,006	476	169
All intellectual disability	20,999	11,747	9,252	11,747	—	—	—	—	—	9,252	—	—	—	—
All vision loss	50,671	21,969	28,702	525	374	6,746	5,548	8,775	8,775	384	425	6,834	6,109	14,950
All nephritis and nephrosis	3,809	2,186	1,623	35	75	1,437	388	250	250	25	65	1,035	288	210

(continued)

Annex Table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Australia, 2003

Notes

- (a) Excludes HIV/AIDS.
- (b) Includes hepatitis B-related liver cancer and cirrhosis.
- (c) Includes hepatitis C-related liver cancer and cirrhosis.
- (d) Excludes liver cancer related to hepatitis B and C.
- (e) Includes alcoholic cirrhosis.
- (f) Excludes those with any other comorbid mental disorders.
- (g) Excludes alcoholic and hepatic cirrhosis.
- (h) Excludes diabetic-, congenital- and poisoning-related renal failure.
- (i) Includes both acute and chronic back pain.
- (j) Includes renal failure due to dysplasia.
- (k) Includes polycystic renal failure.
- (l) Includes suffocation and foreign bodies, adverse effects of medical treatment, other mechanical force injuries and other unintentional injuries.

Annex Table 7: Years of life lost (YLL) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
All causes	1,278,778	709,597	569,181	30,206	29,757	294,233	153,213	202,188	23,529	11,275	177,916	103,650	252,812
I. Communicable diseases, maternal and neonatal conditions	74,073	41,577	32,496	12,259	422	14,330	5,405	9,162	9,569	384	6,974	3,321	12,249
A. Infectious and parasitic diseases	30,665	19,446	11,219	854	275	11,912	3,548	2,857	696	292	5,256	1,890	3,085
1. Tuberculosis	477	243	234	—	1	101	52	89	—	—	38	37	158
2. Sexually transmitted diseases ^(a)	169	12	157	—	—	1	—	12	31	—	95	—	31
a. Syphilis	78	12	66	—	—	—	—	12	31	—	26	—	9
b. Chlamydia	54	—	54	—	—	—	—	—	—	—	41	—	13
c. Gonorrhoea	—	—	—	—	—	—	—	—	—	—	—	—	—
d. Other sexually transmitted diseases	36	—	36	—	—	—	—	—	—	—	27	—	9
3. HIV/AIDS	2,507	2,237	270	2	5	2,084	139	8	1	1	255	14	—
4. Diarrhoeal diseases	379	179	200	6	28	60	30	55	31	1	—	35	134
5. Childhood immunisable diseases	369	222	147	34	—	101	65	22	63	—	21	37	26
a. Diphtheria	—	—	—	—	—	—	—	—	—	—	—	—	—
b. Whooping cough	2	2	1	2	—	—	—	—	1	—	—	—	—
c. Tetanus	—	—	—	—	—	—	—	—	—	—	—	—	—
d. Poliomyelitis	197	119	78	—	—	32	65	22	—	—	21	37	21
e. Measles	—	—	—	—	—	—	—	—	—	—	—	—	—
f. Rubella	1	1	—	—	—	—	—	—	—	—	—	—	—
g. <i>Haemophilus influenzae</i> type b (Hib)	168	101	68	32	—	68	—	—	63	—	—	—	5
6. Meningitis	1,392	629	762	335	98	114	60	22	259	180	277	26	21
7. Septicaemia	2,696	1,516	1,180	159	28	440	389	500	96	1	191	137	754

(continued)

Annex Table 7 (continued): Years of life lost (YLL) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
8. Arbovirus infection	1	—	—	—	—	—	—	—	—	—	—	—	—
a. Ross River virus	—	—	—	—	—	—	—	—	—	—	—	—	—
b. Barmah Forest virus	—	—	—	—	—	—	—	—	—	—	—	—	—
c. Dengue	1	—	—	—	—	—	—	—	—	—	—	—	—
d. Other arbovirus infection	—	—	—	—	—	—	—	—	—	—	—	—	—
9. Hepatitis	19,058	12,524	6,534	8,447	2,371	1,631	20	17	3,794	1,263	1,440	—	—
a. Hepatitis A	16	6	10	—	—	6	1	—	—	—	9	—	—
b. Hepatitis B ^(b)	6,705	4,268	2,437	2,339	930	943	6	6	1,022	511	892	—	—
c. Hepatitis C ^(c)	12,293	8,224	4,069	6,084	1,441	683	13	12	2,772	752	521	—	—
d. Other hepatitis	44	25	18	23	—	—	—	—	—	—	18	—	—
10. Malaria	87	59	28	—	—	—	—	—	28	—	—	—	—
11. Trachoma	—	—	—	—	—	—	—	—	—	—	—	—	—
12. Other infectious and parasitic diseases	3,530	1,823	1,707	563	442	519	196	92	556	342	521	—	—
B. Acute respiratory infections	23,750	11,340	12,411	2,417	1,844	6,224	708	147	2,417	1,844	8,931	32	1,533
1. Lower respiratory tract infections	23,530	11,221	12,309	2,374	1,844	6,215	642	147	2,374	1,844	8,911	32	1,483
2. Upper respiratory tract infections	111	37	74	23	—	10	4	—	23	—	14	—	28
3. Otitis media	110	82	28	19	—	—	62	—	19	—	5	—	22
C. Maternal conditions	226	—	226	—	—	—	—	—	—	—	—	59	167
1. Maternal haemorrhage	31	—	31	—	—	—	—	—	—	—	—	—	31
2. Maternal sepsis	27	—	27	—	—	—	—	—	—	—	—	—	27
3. Hypertensive disorders of pregnancy	31	—	31	—	—	—	—	—	—	—	—	30	1
4. Obstructed labour	1	—	1	—	—	—	—	—	—	—	—	—	1
5. Abortion	—	—	—	—	—	—	—	—	—	—	—	—	—
6. Other maternal conditions	135	—	135	—	—	—	—	—	—	—	—	29	106

(continued)

Annex Table 7 (continued): Years of life lost (YLL) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females							
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+			
D. Neonatal causes	18,974	10,666	—	—	—	—	—	—	—	—	—	—	—	—
1. Birth trauma and asphyxia	4,087	2,068	—	—	—	—	—	—	—	—	—	—	—	—
2. Low birthweight	8,538	4,829	—	—	—	—	—	—	—	—	—	—	—	—
3. Neonatal infections	1,560	950	—	—	—	—	—	—	—	—	—	—	—	—
4. Other conditions arising in the perinatal period	4,789	2,819	—	—	—	—	—	—	—	—	—	—	—	—
E. Nutritional deficiencies	458	125	—	—	1	13	1	1	17	82	233	48	174	10
1. Protein-energy malnutrition	97	33	1	—	1	—	—	—	—	—	—	—	—	—
2. Deficiency anaemia	272	44	—	—	—	—	—	—	—	—	—	—	—	—
3. Other nutritional deficiencies	89	48	30	—	—	12	6	—	17	14	10	—	—	—
II. Non-communicable diseases	1,064,057	567,418	13,695	7,619	214,689	143,286	188,129	9,997	4,925	151,039	97,461	233,217	52,407	775
F. Malignant neoplasms	411,953	220,159	2,012	1,985	95,203	64,383	56,576	1,195	1,514	91,583	45,095	52,407	775	775
1. Mouth and oropharynx cancers	9,522	6,689	33	87	4,031	1,690	848	—	31	1,331	696	—	—	—
2. Oesophagus cancer	13,275	9,427	—	29	4,773	2,770	1,854	—	—	1,161	1,074	—	—	—
3. Stomach cancer	13,818	8,209	—	3	3,733	2,522	1,951	—	31	2,433	1,252	—	—	—
4. Colorectal cancer	51,732	27,997	—	34	12,835	8,387	6,740	1	34	9,633	6,060	—	—	—
5. Liver cancer ^(d)	4,626	3,173	14	2	1,598	929	630	13	12	638	329	—	—	—
6. Gallbladder cancer	3,361	1,339	—	—	564	472	304	—	—	721	603	—	—	—
7. Pancreas cancer	22,119	11,136	—	—	5,279	3,325	2,532	—	—	4,064	2,965	—	—	—
8. Lung cancer	83,056	51,505	60	59	20,690	17,933	12,763	—	29	13,705	9,215	—	—	—
9. Bone and connective tissue cancer	5,016	2,900	282	609	1,317	360	331	161	311	1,159	219	—	—	—
10. Melanoma	15,384	10,108	—	153	6,041	2,121	1,794	—	37	3,048	1,073	—	—	—
11. Non-melanoma skin cancers	3,617	2,560	—	1	911	758	890	—	—	207	150	—	—	—
12. Breast cancer	40,214	134	—	—	87	23	24	—	3	26,122	7,084	—	—	—

(continued)

Annex Table 7 (continued): Years of life lost (YLL) by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females								
		Males		Females		Males		Females		Males		Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
2. Cystic fibrosis	864	409	140	263	—	3	455	3	140	263	—	10	244	201	—	—
3. Haemophilia	147	110	—	56	11	43	37	1	—	56	11	—	—	2	18	16
4. Other endocrine and metabolic disorders	11,092	5,430	243	2,431	790	1,199	5,662	768	243	2,431	790	449	234	2,109	805	2,065
J. Mental disorders	23,154	17,604	1,146	13,433	2,095	926	5,549	5	1,146	13,433	2,095	35	571	3,775	492	676
1. Substance use disorders	21,965	17,197	1,144	13,283	2,042	724	4,768	3	1,144	13,283	2,042	32	481	3,545	381	329
a. Alcohol dependence and harmful use ^(e)	14,255	11,449	66	8,631	2,042	710	2,806	—	66	8,631	2,042	—	30	2,077	370	329
b. Heroin or polydrug dependence and harmful use	6,552	4,957	894	4,061	—	—	1,595	3	894	4,061	—	32	351	1,200	12	—
c. Benzodiazepine dependence and harmful use	29	2	2	1	—	—	26	—	2	1	—	—	—	26	—	—
d. Cannabis dependence and harmful use	3	2	—	2	—	—	1	—	—	2	—	—	—	—	—	—
e. Other drug dependence and harmful use	1,126	785	183	589	—	14	341	—	183	589	—	—	99	241	—	—
2. Schizophrenia	252	112	—	49	10	53	139	—	—	49	10	—	—	25	35	80
3. Anxiety and depression	334	113	—	39	—	74	221	—	—	39	—	—	—	34	26	161
4. Bipolar disorder	90	26	—	26	—	—	64	—	—	26	—	—	—	19	26	20
5. Personality disorders ^(f)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
6. Eating disorders	141	9	—	—	—	9	132	—	—	—	—	—	3	110	—	19
a. Anorexia nervosa	98	—	—	—	—	—	98	—	—	—	—	—	2	91	—	5
b. Bulimia nervosa	1	—	—	—	—	—	1	—	—	—	—	—	1	—	—	—
c. Other eating disorders	41	9	—	—	—	9	33	—	—	—	—	—	—	19	—	14
7. Childhood conditions	110	20	—	19	—	—	90	1	—	19	—	2	88	—	—	—
a. Attention-deficit hyperactivity disorder	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
b. Autism spectrum disorders	110	20	—	19	—	—	90	1	—	19	—	2	88	—	—	—

(continued)

Annex Table 7 (continued): Years of life lost (YLL) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
8. Other mental disorders	262	1	—	17	42	66	—	—	43	26	67
K. Nervous system and sense organ disorders	54,127	1,441	1,366	8,119	4,011	9,639	1,238	652	6,059	3,884	17,718
1. Dementia	24,103	41	31	643	1,273	6,108	155	31	721	1,553	13,549
2. Epilepsy	6,220	284	604	2,612	206	141	220	267	1,413	165	308
3. Parkinson's disease	5,695	—	—	221	701	2,119	—	—	152	503	2,000
4. Multiple sclerosis	1,623	—	1	342	114	24	—	1	916	100	125
5. Motor neurone disease	6,466	1	1	1,744	1,028	593	31	—	1,288	1,028	753
6. Huntington's chorea	961	—	—	412	77	31	—	—	274	97	69
7. Muscular dystrophy	788	32	318	227	35	—	30	29	64	34	18
8. Sense organ disorders	9	—	—	—	—	—	—	—	—	—	9
a. Glaucoma-related blindness	3	—	—	—	—	—	—	—	—	—	3
b. Cataract-related blindness	—	—	—	—	—	—	—	—	—	—	—
c. Macular degeneration	—	—	—	—	—	—	—	—	—	—	—
d. Adult-onset hearing loss	—	—	—	—	—	—	—	—	—	—	—
e. Refractive errors	—	—	—	—	—	—	—	—	—	—	—
f. Other vision loss	6	—	—	—	—	—	—	—	—	—	6
9. Migraine	7	—	—	—	—	—	—	—	—	—	7
10. Other nervous system and sense organ disorders	8,255	1,084	411	1,917	577	624	803	323	1,232	404	880
L. Cardiovascular disease	369,365	673	1,346	68,845	47,485	81,195	628	801	26,338	27,862	114,194
1. Rheumatic heart disease	2,955	—	59	507	199	280	30	59	552	510	758
2. Ischaemic heart disease	218,143	23	292	48,007	31,810	48,859	1	96	12,832	15,042	61,180
3. Stroke	84,699	216	327	8,451	7,903	19,254	153	209	6,873	7,182	34,130
4. Inflammatory heart disease	12,215	297	214	4,148	1,628	1,775	344	92	1,303	835	1,581
5. Hypertensive heart disease	8,303	—	—	882	803	1,793	—	—	493	614	3,719

(continued)

Annex Table 7 (continued): Years of life lost (YLL) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
6. Non-rheumatic valvular disease	7,573	—	117	1,174	788	1,707	1	59	623	600	2,505
7. Aortic aneurysm	11,129	—	59	1,844	2,134	2,998	31	29	573	902	2,559
8. Peripheral vascular disease	5,718	31	29	543	594	1,443	—	29	375	458	2,217
9. Other cardiovascular disease	18,629	105	249	3,289	1,627	3,086	69	228	2,713	1,719	5,544
M. Chronic respiratory disease	71,339	847	457	8,342	11,142	17,901	350	73	7,892	8,065	16,270
1. Chronic obstructive pulmonary disease (COPD)	47,208	317	34	5,001	8,265	12,566	97	32	4,671	5,633	10,592
2. Asthma	4,045	124	248	832	194	224	97	39	1,227	424	637
3. Other chronic respiratory diseases	20,086	406	175	2,509	2,684	5,110	157	3	1,993	2,008	5,041
N. Diseases of the digestive system	27,710	375	106	4,731	2,801	4,914	127	93	3,485	2,726	8,352
1. Peptic ulcer disease	4,162	31	—	722	482	929	—	—	283	259	1,458
2. Cirrhosis of the liver ^(a)	1,480	31	16	265	105	246	1	2	166	83	566
3. Appendicitis	228	—	1	44	7	61	—	—	57	24	35
4. Intestinal obstruction	3,203	32	—	217	335	780	—	1	297	209	1,332
5. Diverticulitis	2,373	—	—	241	215	425	—	—	221	410	862
6. Gallbladder and bile duct disease	2,035	—	—	186	281	391	—	—	268	200	710
7. Pancreatitis	2,188	—	30	787	209	255	—	28	419	134	326
8. Inflammatory bowel disease	587	—	—	121	45	64	—	1	146	91	121
9. Vascular insufficiency of bowel	3,623	125	28	399	313	626	32	29	439	552	1,078
10. Other digestive system diseases	7,830	156	31	1,749	810	1,138	94	32	1,190	765	1,865

(continued)

Annex Table 7 (continued): Years of life lost (YLL) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
O. Genitourinary diseases	24,087	11,341	12,746	74	60	2,263	2,157	6,787	83	1,706	2,046	8,884
1. Nephritis and nephrosis ^(b)	18,857	9,336	9,521	74	32	1,959	1,725	5,546	83	1,355	1,508	6,550
2. Benign prostatic hypertrophy	244	244	—	—	—	17	67	159	—	—	—	—
3. Urinary incontinence	—	—	—	—	—	—	—	—	—	—	—	—
4. Infertility	—	—	—	—	—	—	—	—	—	—	—	—
5. Other genitourinary diseases	4,986	1,762	3,225	—	28	286	365	1,082	—	352	538	2,334
P. Skin diseases	2,173	863	1,310	1	—	184	283	394	—	200	260	849
1. Eczema	—	—	—	—	—	—	—	—	—	—	—	—
2. Acne	—	—	—	—	—	—	—	—	—	—	—	—
3. Psoriasis	98	44	53	—	19	—	—	25	—	—	23	30
4. Ulcers	1,746	728	1,018	—	128	248	351	18	—	112	171	735
5. Other skin diseases	329	90	238	1	—	37	35	18	—	88	65	84
Q. Musculoskeletal diseases	7,027	2,377	4,649	92	2	763	691	830	117	1,462	1,154	1,914
1. Rheumatoid arthritis	1,626	483	1,143	30	—	131	164	159	—	167	408	568
2. Osteoarthritis	374	66	308	—	—	17	—	48	—	19	12	278
3. Back pain ⁽ⁱ⁾	173	115	59	—	—	30	45	40	—	—	12	47
4. Slipped disc	31	24	7	—	—	—	20	4	—	—	—	7
5. Occupational overuse syndrome	—	—	—	—	—	—	—	—	—	—	—	—
6. Systemic lupus erythematosus (SLE)	661	67	594	—	—	17	22	27	1	31	77	80
7. Gout	175	113	62	—	—	36	10	67	—	—	—	62
8. Other musculoskeletal diseases	3,986	1,509	2,477	62	2	532	430	484	1	86	646	873
R. Congenital anomalies	16,897	9,175	7,722	5,801	504	2,217	301	352	4,666	419	1,793	442
1. Anencephaly	387	102	285	102	—	—	—	—	285	—	—	—
2. Spina bifida	461	229	232	128	31	57	12	—	98	30	105	—
3. Congenital heart disease	5,559	3,221	2,338	1,864	256	961	77	64	1,334	236	114	83

(continued)

Annex Table 7 (continued): Years of life lost (YLL) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		Males	Females	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
4. Cleft lip and/or palate	32	1	31	1	—	—	—	—	31	—	—	—	—
5. Digestive system malformations	428	207	221	169	—	16	12	8	194	1	—	12	14
a. Anorectal atresia	1	1	—	1	—	—	—	—	—	—	—	—	—
b. Oesophageal atresia	3	1	2	1	—	—	—	—	2	—	—	—	—
c. Other digestive system malformations	424	205	219	168	—	16	12	8	192	1	—	12	14
6. Urogenital tract malformations	2,069	1,220	849	379	1	416	157	268	71	—	313	183	281
a. Renal agenesis ⁽ⁱ⁾	233	125	108	120	1	4	—	—	67	—	27	11	3
b. Other urogenital tract malformations ^(k)	1,836	1,095	741	259	—	412	157	268	4	—	286	172	278
7. Abdominal wall defect	224	158	65	158	—	—	—	—	65	—	—	—	—
8. Down syndrome	1,628	896	732	383	61	429	22	—	163	2	470	79	18
9. Other chromosomal disorders	1,196	494	702	491	1	2	—	—	649	1	52	—	—
10. Other congenital anomalies	4,913	2,646	2,267	2,125	153	336	20	12	1,774	149	283	14	47
S. Oral conditions	102	19	83	—	—	1	—	18	—	—	26	11	45
1. Dental caries	—	—	—	—	—	—	—	—	—	—	—	—	—
2. Periodontal disease	11	—	11	—	—	—	—	—	—	—	—	11	—
3. Edentulism	—	—	—	—	—	—	—	—	—	—	—	—	—
4. Pulpitis	13	1	13	—	—	1	—	—	—	—	—	—	13
5. Other oral conditions	77	18	59	—	—	—	—	18	—	—	26	—	32
Z. Ill-defined conditions	2,536	1,483	1,053	1,470	—	—	—	13	958	—	—	14	81
1. Sudden infant death syndrome	2,428	1,470	958	1,470	—	—	—	—	958	—	—	—	—
2. Chronic fatigue syndrome	108	13	95	—	—	—	—	13	—	—	—	14	81

(continued)

Annex Table 7 (continued): Years of life lost (YLL) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
III. Injuries	140,648	100,602	40,046	4,252	21,717	65,214	4,522	4,897	3,963	5,966	19,903	2,868	7,346
T. Unintentional injuries	84,599	57,472	27,127	3,748	13,448	33,069	3,007	4,199	3,275	4,020	10,366	2,354	7,111
1. Road traffic accidents	36,352	26,674	9,678	1,668	9,088	14,564	776	579	1,134	2,926	4,482	562	574
2. Other transport accidents	5,728	4,631	1,097	355	1,129	2,940	151	56	395	121	498	38	44
3. Poisoning	11,720	6,783	4,937	31	891	5,440	218	202	33	443	2,664	640	1,158
4. Falls	12,391	6,546	5,845	32	692	2,771	860	2,192	94	117	755	582	4,296
5. Fires, burns and scalds	1,960	1,331	629	180	30	880	144	97	69	30	379	55	96
6. Drowning	4,759	3,317	1,442	645	652	1,826	114	79	705	92	530	80	34
7. Sports injuries	1	1	—	—	—	1	—	—	—	—	—	—	—
8. Natural and environmental factors	1,216	901	315	59	225	521	46	49	125	29	57	43	61
9. Machinery accidents	438	438	—	30	56	322	21	8	—	—	—	—	—
10. Other unintentional injuries ⁽¹⁾	10,033	6,851	3,182	748	684	3,805	677	938	721	261	999	353	848
Suffocation and foreign bodies	4,987	3,375	1,612	594	512	1,824	165	280	593	174	496	54	296
Adverse effects of medical treatment	2,278	1,179	1,098	30	—	303	317	528	32	57	339	254	416
Other unintentional injuries n.e.c.	2,768	2,296	472	124	172	1,678	194	130	96	31	2,768	2,296	472
U. Intentional injuries	56,050	43,130	12,919	504	8,269	32,145	1,515	697	688	1,946	9,538	514	235
1. Suicide and self-inflicted injuries	49,379	38,434	10,945	174	7,244	28,898	1,436	682	205	1,411	8,674	465	189
2. Homicide and violence	6,624	4,650	1,975	330	1,025	3,200	79	15	483	534	863	48	46
3. Legal intervention and war	46	46	—	—	—	46	—	—	—	—	—	—	—
Australian population ('000)	19,881	9,872	10,010	2,041	1,404	5,292	656	478	1,938	1,349	5,311	694	718
YLL per 1,000 population	64.3	71.9	56.9	14.8	21.2	55.6	233.6	423.0	12.1	8.4	33.5	149.4	352.1

(continued)

Annex Table 7 (continued): Years of life lost (YLL) by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
Alternative burden of disease categories											
Diabetes mellitus (attributable)	94,503	47,708	46,796	12,125	13,365	22,156	60	92	4,998	7,724	33,921
Anxiety and depression (attributable)	22,731	14,846	7,886	1,176	1,049	924	18	473	5,550	658	1,187
All intellectual disability	23,189	11,075	12,114	666	3,043	141	9,339	270	1,935	244	326
All vision loss	4,868	4,859	9	30	—	—	—	—	—	—	9
All nephritis and nephrosis	64,912	35,505	29,407	484	14,286	8,126	191	617	7,445	5,993	15,161

Notes

- (a) Excludes HIV/AIDS.
- (b) Includes hepatitis B-related liver cancer and cirrhosis.
- (c) Includes hepatitis C-related liver cancer and cirrhosis.
- (d) Excludes liver cancer related to hepatitis B and C.
- (e) Includes alcoholic cirrhosis.
- (f) Excludes those with any other comorbid mental disorders.
- (g) Excludes alcoholic and hepatic cirrhosis.
- (h) Excludes diabetic, congenital- and poisoning-related renal failure.
- (i) Includes both acute and chronic back pain.
- (j) Includes renal failure due to dysplasia.
- (k) Includes polycystic renal failure.
- (l) Includes suffocation and foreign bodies, adverse effects of medical treatment, other mechanical force injuries and other unintentional injuries.

Annex Table 8: Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males				Females				Males					Females				
		Males	Females	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
I. Communicable diseases, maternal and neonatal conditions																			
A. Infectious and parasitic diseases																			
1. Tuberculosis	948	488	460	17	69	267	64	71	19	55	296	41	49						
2. Sexually transmitted diseases ^(a)																			
a. Syphilis	2,466	1,621	845	6	171	1,254	106	84	16	233	485	38	73						
b. Chlamydia	40,621	27,647	12,973	98	12,510	14,934	58	48	226	8,780	3,947	8	13						
c. Gonorrhoea	6,861	4,758	2,103	46	1,496	3,182	26	9	132	1,286	683	1	—						
3. HIV/AIDS	840	753	87	1	64	676	11	1	1	12	72	2	—						
4. Diarrhoeal diseases	17,457,098	7,867,069	9,590,029	2,025,448	1,172,464	4,248,622	246,688	173,847	2,351,078	1,377,993	5,223,546	318,473	318,940						
5. Childhood immunisable diseases																			
a. Diphtheria	—	—	—	—	—	—	—	—	—	—	—	—	—						
b. Whooping cough	8,791	3,972	4,819	1,706	576	1,475	136	80	1,718	659	2,164	173	106						
c. Tetanus	4	3	1	—	—	—	1	2	—	—	—	—	1						
d. Poliomyelitis	—	—	—	—	—	—	—	—	—	—	—	—	—						
e. Measles	98	58	40	22	14	22	—	—	9	12	19	—	—						
f. Rubella	27	17	10	1	7	8	—	—	1	3	6	1	—						
g. <i>Haemophilus influenzae</i> type b (Hib)	23	10	13	7	—	1	2	—	8	—	4	1	—						
6. Meningitis	1,631	876	754	357	139	285	60	35	219	134	327	40	34						
7. Septicaemia	18,982	10,640	8,342	875	287	3,965	2,332	3,181	653	329	3,045	1,410	2,905						
8. Arbovirus infection																			
a. Ross River virus	7,741	3,664	4,077	90	296	2,985	224	68	72	310	3,425	166	103						
b. Barmah Forest virus	2,759	1,370	1,389	32	82	1,124	88	44	22	92	1,190	56	29						

(continued)

Annex Table 8 (continued): Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
c. Dengue	946	490	456	40	82	345	19	4	45	87	307	11	6
9. Hepatitis													
a. Hepatitis A	2,173	1,292	881	385	190	645	40	32	210	175	450	25	21
b. Hepatitis B	2,591	1,407	1,183	136	260	965	26	20	133	227	797	12	14
c. Hepatitis C	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
10. Malaria	605	406	199	58	100	240	8	—	60	29	108	2	—
11. Trachoma	102	53	50	—	1	40	9	2	—	1	37	10	2
B. Acute respiratory infections													
1. Lower respiratory tract infections	2,048,242	991,471	1,056,771	221,955	101,229	520,576	80,926	66,784	166,425	126,302	603,845	86,732	73,466
2. Upper respiratory tract infections	26,237,596	12,310,741	13,926,855	4,743,888	1,980,032	4,955,337	376,366	255,119	4,865,864	2,552,985	5,750,295	408,353	349,358
3. Otitis media	1,174,267	627,022	547,245	455,983	57,643	105,121	3,277	4,998	337,552	52,110	134,082	15,755	7,746
C. Maternal conditions													
1. Maternal haemorrhage	21,543	—	21,543	—	—	—	—	—	11	4,622	16,910	—	—
2. Maternal sepsis	6,793	—	6,793	—	—	—	—	—	8	1,528	5,257	—	—
3. Hypertensive disorders of pregnancy	32,698	—	32,698	—	—	—	—	—	24	6,757	25,917	—	—
4. Obstructed labour	10,871	—	10,871	—	—	—	—	—	3	1,772	9,096	—	—
D. Neonatal causes													
1. Birth trauma and asphyxia	859	473	386	473	—	—	—	—	386	—	—	—	—
2. Low birthweight	1,339	659	680	659	—	—	—	—	680	—	—	—	—
3. Neonatal infections	9,409	5,362	4,047	5,362	—	—	—	—	4,047	—	—	—	—
E. Nutritional deficiencies													
2. Deficiency anaemia	940,777	217,333	723,444	68,594	10,053	73,386	37,777	27,523	69,839	124,138	460,432	33,934	35,101

(continued)

Annex Table 8 (continued): Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females									
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+					
II. Non-communicable diseases																
F. Malignant neoplasms																
1. Mouth and oropharynx cancers	2,822	1,976	846	1,102	460	398	1	16	1,102	460	398	1	10	389	169	276
2. Oesophagus cancer	1,139	746	393	284	215	247	—	—	284	215	247	—	—	82	111	200
3. Stomach cancer	2,008	1,273	734	415	389	468	1	—	415	389	468	—	—	217	175	342
4. Colorectal cancer	13,552	7,383	6,169	2,518	2,351	2,503	1	9	2,518	2,351	2,503	1	14	1,853	1,596	2,704
5. Liver cancer																
a. Liver cancer ^(b)	368	266	101	103	78	82	2	1	103	78	82	2	—	32	23	44
b. All liver cancer	897	650	247	250	190	201	6	2	250	190	201	5	—	78	57	108
6. Gallbladder cancer	626	276	350	82	83	110	1	—	82	83	110	—	—	82	99	169
7. Pancreas cancer	1,967	1,019	948	341	292	385	—	—	341	292	385	1	—	217	197	533
8. Lung cancer	8,734	5,706	3,028	1,613	2,001	2,086	2	4	1,613	2,001	2,086	1	1	1,016	890	1,120
9. Bone and connective tissue cancer	731	386	345	179	68	81	22	36	179	68	81	29	27	158	53	79
10. Melanoma	9,290	5,281	4,008	2,833	1,159	1,186	6	97	2,833	1,159	1,186	13	125	2,478	654	738
11. Non-melanoma skin cancers	382,623	221,190	161,433	111,824	56,202	52,546	—	619	111,824	56,202	52,546	—	—	81,027	37,241	43,164
12. Breast cancer	12,359	—	12,359	—	—	—	—	—	—	—	—	—	9	7,835	2,334	2,181
13. Cervix cancer	760	—	760	—	—	—	—	—	—	—	—	—	9	550	87	114
14. Corpus uteri cancer	1,622	—	1,622	—	—	—	—	—	—	—	—	—	—	868	375	379
15. Ovary cancer	1,355	—	1,355	—	—	—	—	—	—	—	—	4	22	681	256	393
16. Prostate cancer	11,899	11,899	—	3,287	4,194	4,419	—	—	3,287	4,194	4,419	—	—	—	—	—
17. Testicular cancer	615	615	—	479	13	4	6	113	479	13	4	—	—	—	—	—
18. Bladder cancer	3,130	2,401	729	557	759	1,082	—	3	557	759	1,082	—	—	158	193	378
19. Kidney cancer	2,584	1,599	985	654	470	459	12	4	654	470	459	20	1	369	259	337

(continued)

Annex Table 8 (continued): Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females									
		0-14		15-24		25-64		0-14		15-24		25-64		65-74		75+	
		Males	Females	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females
20. Brain cancer	1,404	822	582	59	33	422	172	136	41	15	273	122	131				
21. Thyroid cancer	1,218	309	909	—	17	219	37	37	5	57	695	73	78				
22. Lymphoma	4,088	2,252	1,836	46	87	1,066	507	546	22	72	757	397	589				
23. Multiple myeloma	1,266	735	531	—	—	251	196	288	—	1	138	145	247				
24. Leukaemia	2,336	1,359	977	116	39	508	305	390	97	17	357	174	332				
25. Larynx cancer	619	562	57	—	—	248	173	141	—	—	21	15	21				
26. Eye cancer	244	144	100	10	3	56	44	31	10	3	42	18	27				
G. Other neoplasms																	
1. Uterine myomas	19,239	—	19,239	—	—	—	—	—	1	30	17,913	891	404				
2. Benign neoplasms of meninges and brain	1,788	619	1,169	26	15	335	106	137	46	12	676	182	254				
H. Diabetes mellitus																	
1. Type 1 diabetes	2,202	1,301	901	453	251	533	38	24	371	160	326	24	20				
2. Type 2 diabetes	94,825	48,704	46,122	53	75	29,656	9,005	9,916	68	218	21,634	8,090	16,112				
I. Endocrine and metabolic disorders																	
1. Non-deficiency anaemia																	
a. Haemolytic anaemia	432	243	190	243	—	—	—	—	190	—	—	—	—				
2. Cystic fibrosis	86	44	42	44	—	—	—	—	42	—	—	—	—				
3. Haemophilia	13	13	—	13	—	—	—	—	—	—	—	—	—				
J. Mental disorders																	
1. Substance use disorders																	
a. Alcohol dependence and harmful use	238,731	181,904	56,827	—	59,433	116,143	4,397	1,930	—	6,979	49,135	346	368				

(continued)

Annex Table 8 (continued): Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females							
		Males		Females		Males		Females		Males		Females			
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74
b. Heroin or polydrug dependence and harmful use	3,070	2,238	833	—	1,403	824	7	4	4	13	500	316	4	—	
c. Benzodiazepine dependence and harmful use	4,573	2,306	2,267	—	404	1,891	10	1	1	—	517	1,745	4	1	
d. Cannabis dependence and harmful use	22,108	17,098	5,009	—	14,592	2,494	11	1	1	—	4,274	735	—	—	
2. Schizophrenia	3,028	1,635	1,393	19	1,055	552	4	5	4	17	378	989	4	4	
3. Anxiety and depression	111,064	40,669	70,395	7,220	13,267	18,086	1,266	830	830	9,027	17,609	43,295	407	58	
4. Bipolar disorder	4,100	2,104	1,997	—	1,403	697	3	1	1	—	1,249	739	4	4	
5. Personality disorders ^(c)	76,471	41,329	35,142	—	7,677	29,902	2,421	1,328	1,328	—	5,432	25,389	2,405	1,916	
6. Eating disorders															
a. Anorexia nervosa	1,448	182	1,266	49	105	27	—	—	—	202	1,064	—	—	—	
b. Bulimia nervosa	2,723	—	2,723	—	—	—	—	—	—	349	2,285	88	—	—	
7. Childhood conditions															
a. Attention-deficit hyperactivity disorder	26,095	18,186	7,909	18,186	—	—	—	—	—	7,888	20	—	—	—	
b. Autism spectrum disorders	1,207	1,017	189	1,017	—	—	—	—	—	189	—	—	—	—	
K. Nervous system and sense organ disorders															
1. Dementia	37,064	13,819	23,245	—	1	1,049	2,750	10,019	10,019	—	—	578	2,695	19,973	
2. Epilepsy	3,922	1,925	1,997	602	404	571	159	188	188	571	387	570	168	301	

(continued)

Annex Table 8 (continued): Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
3. Parkinson's disease	7,148	—	—	546	1,115	2,809	—	—	341	921	1,416
4. Multiple sclerosis	568	2	13	168	2	3	8	24	327	3	16
5. Motor neurone disease	533	—	—	94	93	89	1	—	66	82	106
6. Huntington's chorea	110	—	3	40	9	6	—	1	36	7	7
7. Muscular dystrophy	38	28	—	—	—	—	10	—	—	—	—
8. Sense organ disorders											
a. Glaucoma-related blindness	1,343	—	—	241	230	155	—	—	232	244	241
b. Cataract-related blindness	62,701	74	59	4,443	7,567	12,529	54	31	4,904	11,242	21,798
c. Macular degeneration	7,940	—	—	4	533	2,529	—	—	4	552	4,318
d. Adult-onset hearing loss	246,428	—	1,923	84,823	46,161	36,969	—	1,021	40,308	20,932	14,290
e. Refractive errors	86,090	1,171	1,503	14,927	8,888	12,575	471	1,802	15,601	9,112	20,041
9. Migraine	99,607	15,300	10,126	1,955	4	3	20,808	27,309	23,943	79	80
10. Intellectual disability											
a. All mild intellectual disability	1,120	571	—	—	—	—	548	—	—	—	—
b. All moderate intellectual disability	412	220	—	—	—	—	192	—	—	—	—
c. All severe intellectual disability	207	111	—	—	—	—	96	—	—	—	—

(continued)

Annex Table 8 (continued): Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
d. All profound intellectual disability	75	40	—	—	—	—	35	—	—	—	—	—
11. All vision loss												
a. All mild vision loss	178,262	1,746	1,845	24,206	19,751	30,320	1,003	2,108	25,024	23,434	48,825	
b. All moderate vision loss	16,683	7	6	763	1,559	4,355	5	3	767	1,953	7,264	
c. All severe vision loss	3,835	29	—	284	285	830	31	—	262	286	1,828	
12. All hearing loss												
a. All mild hearing loss (25-34 dBHTL)	136,384	—	1,621	53,349	21,270	12,799	—	983	30,489	12,501	3,373	
b. All mild hearing loss (35-44 dBHTL)	67,387	—	302	22,913	14,757	13,497	—	37	6,250	4,417	5,213	
c. All moderate hearing loss	37,111	—	—	7,584	9,138	10,009	—	—	2,672	3,208	4,500	
d. All severe hearing loss	5,641	56	—	978	996	664	39	—	897	807	1,205	
L. Cardiovascular disease												
1. Rheumatic heart disease	1,925	635	6	133	160	329	6	5	306	283	690	
2. Ischaemic heart disease	38,675	24,651	3	11,011	5,992	7,616	1	3	2,844	2,968	8,208	
3. Stroke	19,627	9,129	180	2,995	1,870	3,840	240	96	2,921	1,383	5,858	
4. Inflammatory heart disease	3,123	1,758	37	636	464	590	24	21	423	322	575	
5. Hypertensive heart disease	655	271	2	82	83	103	2	2	82	86	212	

(continued)

Annex Table 8 (continued): Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females							
		Males		Females		Males		Females		Males		Females			
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74
6. Non-rheumatic valvular disease	3,831	1,907	1,924	31	27	588	512	748	22	19	445	448	989		
7. Aortic aneurysm	8,847	6,456	2,391	7	9	1,070	2,222	3,148	3	3	261	599	1,525		
8. Peripheral vascular disease	9,824	6,086	3,738	10	25	2,643	1,629	1,779	10	33	1,257	769	1,669		
9. All heart failure	34,340	15,649	18,691	94	81	3,262	4,016	8,197	79	68	2,861	3,817	11,865		
M. Chronic respiratory disease															
1. Chronic obstructive pulmonary disease (COPD)	20,402	11,772	8,630	4	57	6,483	2,413	2,815	1	49	3,614	1,947	3,019		
2. Asthma	78,493	39,502	38,991	31,376	1,124	4,865	1,247	890	20,415	6,743	7,838	2,016	1,979		
N. Diseases of the digestive system															
1. Peptic ulcer disease	136,579	70,848	65,731	48	2,063	52,990	14,869	878	—	305	48,764	5,931	10,730		
2. Cirrhosis of the liver	—	—	—	—	—	—	—	—	—	—	—	—	—		
a. Cirrhosis of the liver ^(d)	61	34	27	—	1	16	11	6	—	1	15	6	5		
b. All cirrhosis of the liver	1,353	987	365	—	2	714	242	29	—	1	271	80	14		
3. Appendicitis	26,170	13,102	13,068	3,204	3,554	5,685	434	225	2,491	3,698	6,229	390	260		
4. Intestinal obstruction	29,804	14,404	15,400	720	487	6,318	3,067	3,812	385	308	7,124	2,724	4,859		
5. Diverticulitis	102,137	48,950	53,187	3	43	22,200	14,143	12,561	1	29	21,248	15,440	16,469		
6. Gallbladder and bile duct disease	51,256	15,506	35,750	61	230	8,951	3,529	2,735	109	2,279	25,262	4,442	3,658		
7. Pancreatitis	13,620	7,932	5,688	65	326	5,658	1,042	841	71	362	3,429	742	1,084		
8. Inflammatory bowel disease	2,321	1,299	1,022	88	164	891	91	65	82	130	713	45	52		

(continued)

Annex Table 8 (continued): Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
9. Vascular insufficiency of bowel	4,666	2,098	2,568	32	34	667	585	780	19	28	778	550	1,193
O. Genitourinary diseases													
1. Nephritis and nephrosis	3,532	1,873	1,660	30	56	674	219	895	16	44	465	153	981
a. Nephritis and nephrosis ^(e)	4,565	2,392	2,173	33	56	1,003	312	988	20	46	700	257	1,150
b. All nephritis and nephrosis	24,938	24,938	—	—	—	6,381	9,517	9,039	—	—	—	—	—
2. Benign prostatic hypertrophy	43,066	12,106	30,960	—	—	6,315	3,111	2,680	7	1,186	23,225	2,966	3,576
3. Urinary incontinence	33,050	13,822	19,228	7	1,570	12,245	—	—	11	2,162	17,055	—	—
4. Infertility	17,709	7,038	10,671	2,285	297	3,791	302	364	7,419	259	2,693	262	39
P. Skin diseases													
1. Eczema	21,077	10,710	10,366	3,438	5,422	1,851	—	—	1,294	6,447	2,626	—	—
2. Acne	67,687	53,142	14,544	3,340	9,480	35,085	3,369	1,869	954	3,173	8,968	1,013	436
3. Psoriasis	93,757	34,081	59,676	2,382	442	16,328	7,750	7,178	887	3,773	12,315	13,163	29,538
4. Ulcers	6,993	2,207	4,786	92	65	1,168	500	381	276	154	2,939	777	640
Q. Musculoskeletal diseases													
1. Rheumatoid arthritis	45,923	18,588	27,335	1	45	8,172	5,449	4,922	—	—	8,571	7,861	10,903
2. Osteoarthritis	9,045,837	3,840,354	5,205,483	71,652	79,441	1,941,898	740,998	1,006,366	911	222,074	3,119,084	922,055	941,359
3. Back pain ^(f)	230,300	128,523	101,776	318	3,821	91,616	19,742	13,026	709	2,185	65,029	19,973	13,880
4. Slipped disc	12,729	4,672	8,057	—	61	4,421	187	4	1	118	7,908	28	2
5. Occupational overuse syndrome	13,515	11,166	2,349	8	407	8,631	1,088	1,032	5	273	1,027	1,005	40
7. Gout													
R. Congenital anomalies													
1. Anencephaly	13	3	9	3	—	—	—	—	9	—	—	—	—

(continued)

Annex Table 8 (continued): Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
2. Spina bifida	25	13	—	—	—	—	12	—	—	—	—
3. Congenital heart disease	1,455	770	26	163	38	17	419	30	182	16	
4. Cleft lip and/or palate	443	259	—	—	—	—	184	—	—	—	
5. Digestive system malformations											
a. Anorectal atresia	56	30	—	—	—	—	26	—	—	—	
b. Oesophageal atresia	48	30	—	—	—	—	18	—	—	—	
6. Urogenital tract malformations											
a. Renal agenesis	27	19	—	—	—	—	8	—	—	—	
b. Abdominal wall defect	82	48	—	—	—	—	34	—	—	—	
8. Down syndrome	241	136	—	—	—	—	106	—	—	—	
S. Oral conditions											
1. Dental caries	5,011,664	2,507,395	377,931	1,392,170	154,535	104,185	454,515	363,366	1,377,079	173,691	
2. Periodontal disease	81,641	40,824	2,308	31,831	4,021	2,100	538	2,221	31,740	2,773	
3. Edentulism	85,752	33,512	20	16,501	11,046	5,883	23	108	29,447	7,328	
4. Pulpitis	2,208,275	1,088,720	96,240	715,848	72,885	53,100	144,220	93,295	724,013	80,350	
Z. Ill-defined conditions											
2. Chronic fatigue syndrome	4,671	1,607	140	1,466	—	—	—	135	2,930	—	
III. Injuries											
T. Unintentional injuries											
1. Road traffic accidents	25,381	17,618	1,975	9,384	534	493	863	1,933	3,862	611	

(continued)

Annex Table 8 (continued): Incidence by age, sex and cause, Australia, 2003

Cause	Persons	Males										Females				
		Males					Females					Females				
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
2. Other transport accidents	15,770	11,811	3,959	3,146	3,176	5,017	261	211	1,313	699	1,514	158	276			
3. Poisoning	11,412	5,622	5,790	1,659	833	2,560	269	302	1,468	1,048	2,498	271	504			
4. Falls	125,322	62,029	63,293	16,421	9,630	21,729	4,491	9,759	9,790	2,229	15,655	7,895	27,724			
5. Fires, burns and scalds	6,657	4,239	2,419	1,738	647	1,600	133	121	1,134	184	798	107	195			
6. Drowning	76	54	22	5	16	31	1	—	4	6	8	1	3			
7. Sports injuries	10,619	7,026	3,594	723	1,612	4,110	315	265	383	485	1,852	337	537			
8. Natural and environmental factors	8,808	5,264	3,544	1,187	763	2,899	259	157	821	393	1,873	230	228			
9. Machinery accidents	10,812	9,718	1,094	403	1,618	6,788	623	286	166	140	703	46	38			
10. Other unintentional injuries	52,743	37,538	15,206	5,954	8,963	19,626	1,522	1,473	3,029	1,785	6,794	1,149	2,450			
U. Intentional injuries																
1. Suicide and self-inflicted injuries	24,386	9,533	14,852	119	2,089	6,681	213	431	406	4,390	9,671	182	203			
2. Homicide and violence	16,986	13,356	3,631	391	4,704	8,104	118	39	153	855	2,546	31	46			
3. Legal intervention and war	51	46	5	1	7	37	—	1	—	1	4	—	—			

Due to the incidence estimation process, an entry of one case in the above table does not necessarily represent one actual case from that particular cause/age group.

Notes

- (a) Excludes HIV/AIDS.
- (b) Excludes hepatitis B and C related.
- (c) Excludes those with any other comorbid mental disorders.
- (d) Excludes alcoholic and hepatic cirrhosis.
- (e) Excludes diabetic-, congenital- and poisoning-related renal failure.
- (f) Includes both acute and chronic back pain.

Annex Table 9: Prevalence by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
II. Non-communicable diseases													
H. Diabetes mellitus													
1. Type 1 diabetes	97,440	54,532	42,908	2,943	5,954	36,920	5,202	3,513	2,413	4,754	27,583	4,142	4,016
2. Type 2 diabetes	1,073,459	561,587	511,871	300	715	288,597	134,270	137,706	370	1,109	218,952	105,183	186,259
I. Endocrine and metabolic disorders													
1. Non-deficiency anaemia													
a. Haemolytic anaemia	23,123	12,113	11,010	2,754	2,452	6,667	220	20	2,160	1,980	6,491	326	53
2. Cystic fibrosis	3,618	1,879	1,739	676	420	722	36	24	637	380	659	34	29
3. Haemophilia	1,030	1,030	—	208	147	556	69	50	—	—	—	—	—
J. Mental disorders													
1. Substance use disorders													
a. Alcohol dependence and harmful use	903,572	702,969	200,603	—	124,081	547,008	22,152	9,728	—	11,863	184,518	2,562	1,660
b. Heroin or polydrug dependence and harmful use	46,966	34,114	12,852	—	4,973	28,041	780	321	30	1,867	10,461	329	164
c. Benzodiazepine dependence and harmful use	49,560	24,413	25,146	—	1,105	22,618	517	173	—	1,547	22,308	928	363
d. Cannabis dependence and harmful use	230,014	177,112	52,902	—	48,297	127,531	1,064	220	—	14,559	38,309	25	10
2. Schizophrenia	87,538	48,608	38,929	46	4,436	39,495	3,195	1,436	77	1,628	30,765	3,812	2,647
3. Anxiety and depression	1,764,581	662,302	1,102,279	19,487	112,044	469,137	37,785	23,849	26,629	137,869	821,207	72,089	44,484
4. Bipolar disorder	87,775	45,078	42,696	—	5,401	37,094	1,881	703	—	4,730	35,435	1,556	975
5. Personality disorders ^(a)	414,019	231,135	182,884	—	20,608	184,150	16,686	9,691	—	14,245	140,074	16,055	12,510

(continued)

Annex Table 9 (continued): Prevalence by age, sex and cause, Australia, 2003

Cause	Persons	Males	Females	Males					Females				
				0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
6. Eating disorders													
a. Anorexia nervosa	11,601	1,496	10,105	103	625	766	2	1	416	5,534	4,137	13	4
b. Bulimia nervosa	11,863	—	11,863	—	—	—	—	—	628	7,854	3,381	—	—
7. Childhood conditions													
a. Attention-deficit hyperactivity disorder	130,006	92,245	37,761	72,091	19,084	1,070	—	—	30,076	7,239	446	—	—
b. Autism spectrum disorders	80,675	68,127	12,548	10,596	10,660	39,277	4,578	3,016	1,991	1,988	7,272	746	551
K. Nervous system and sense organ disorders													
1. Dementia	167,378	61,310	106,068	3	10	5,641	13,771	41,886	—	—	2,564	12,781	90,723
2. Epilepsy	55,602	26,970	28,632	2,747	3,897	15,432	2,253	2,642	2,604	3,737	15,510	2,428	4,352
3. Parkinson's disease	46,573	22,300	24,273	—	—	3,148	5,820	13,332	—	—	1,465	5,981	16,826
4. Multiple sclerosis	13,622	4,321	9,301	8	57	3,725	406	124	44	150	7,476	1,011	620
5. Motor neurone disease	1,035	544	491	—	—	222	154	167	3	—	150	135	203
6. Huntington's chorea	1,319	683	636	—	10	554	85	33	—	2	449	125	61
7. Muscular dystrophy	821	601	220	431	169	—	—	—	158	62	—	—	—
8. Sense organ disorders													
a. Glaucoma-related blindness	17,479	7,592	9,887	—	—	1,148	2,659	3,786	—	—	786	2,908	6,194
b. Cataract-related blindness	62,025	23,761	38,265	137	58	3,970	6,677	12,919	103	32	4,327	9,845	23,958
c. Macular degeneration	53,639	18,608	35,032	—	—	11	1,596	17,001	—	—	11	1,681	33,340

(continued)

Annex Table 9 (continued): Prevalence by age, sex and cause, Australia, 2003

Cause	Persons	Males						Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+		
d. Adult-onset hearing loss	2,223,138	1,339,155	883,983	—	9,126	510,135	398,812	421,082	—	3,899	299,721	248,491	331,872
e. Refractive errors	266,239	120,597	145,642	2,848	5,452	46,341	28,125	37,831	1,031	5,114	49,997	28,970	60,529
9. Migraine	1,089,376	309,337	780,039	26,448	50,986	209,286	15,336	7,282	22,191	85,142	624,702	30,268	17,735
10. Intellectual disability													
a. All mild intellectual disability	88,076	44,058	44,017	8,911	6,326	23,806	2,928	2,088	8,541	6,130	23,891	2,949	2,507
b. All moderate intellectual disability	31,075	16,353	14,721	3,277	2,416	8,984	1,041	635	2,851	2,129	8,183	931	628
c. All severe intellectual disability	15,081	7,947	7,135	1,646	1,211	4,410	459	220	1,430	1,065	4,018	409	213
d. All profound intellectual disability	4,792	2,525	2,268	593	431	1,403	82	14	512	377	1,291	73	14
11. All vision loss													
a. All mild vision loss	406,508	181,210	225,298	4,576	6,658	63,995	43,306	62,675	2,647	6,251	67,849	47,398	101,154
b. All moderate vision loss	71,465	26,304	45,162	14	7	2,392	3,418	20,473	10	4	2,303	3,835	39,010
c. All severe vision loss	32,728	13,523	19,205	456	324	2,559	3,298	6,887	479	344	2,246	3,447	12,690
12. All hearing loss													
a. All mild hearing loss (25-34 dBHTL)	1,322,036	678,833	643,203	—	7,673	306,856	203,880	160,424	—	3,755	247,922	186,144	205,382
b. All mild hearing loss (35-44 dBHTL)	498,893	389,734	109,159	—	1,453	151,259	116,433	120,589	—	144	33,387	31,111	44,517

(continued)

Annex Table 9 (continued): Prevalence by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
c. All moderate hearing loss	331,805	238,394	93,411	—	45,402	68,413	124,579	—	—	12,324	22,017	59,070
d. All severe hearing loss	77,904	36,567	41,337	882	626	10,379	15,703	607	436	7,786	9,428	23,081
L. Cardiovascular disease												
1. Rheumatic heart disease	9,192	2,520	6,671	24	29	486	1,465	14	19	1,500	1,273	3,865
2. Ischaemic heart disease	309,726	137,468	172,258	—	35	52,206	49,194	—	21	40,051	41,550	90,636
3. Stroke	141,227	61,647	79,581	1,346	2,142	27,740	15,837	1,326	1,701	38,836	14,633	23,085
4. Inflammatory heart disease	26,448	14,708	11,740	243	462	6,462	4,142	161	303	4,460	2,527	4,290
5. Hypertensive heart disease	4,602	1,954	2,647	14	27	669	723	14	24	642	594	1,374
6. Non-rheumatic valvular disease	14,020	5,808	8,212	140	208	2,153	2,180	115	166	2,425	1,523	3,983
7. Aortic aneurysm	723	528	195	1	1	85	263	—	—	21	48	126
8. Peripheral vascular disease	59,501	36,804	22,697	61	125	13,678	12,678	48	143	7,425	4,700	10,381
9. All heart failure	204,854	90,188	114,666	534	920	21,401	45,019	479	839	21,327	22,840	69,181
M. Chronic respiratory disease												
1. Chronic obstructive pulmonary disease (COPD)	387,150	226,650	160,500	9	214	89,222	71,777	2	193	60,470	38,876	60,959
2. Asthma	1,356,620	610,717	745,904	160,831	109,013	282,586	24,527	121,895	112,984	404,303	52,505	54,216
N. Diseases of the digestive system												
1. Peptic ulcer disease	166,784	85,620	81,164	26	1,132	66,715	2,463	—	163	62,474	7,937	10,590
2. Cirrhosis of the liver												
a. Cirrhosis of the liver ^(b)	162	89	73	—	2	41	23	—	2	38	17	17

(continued)

Annex Table 9 (continued): Prevalence by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females						
		Males	Females	0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+
b. All cirrhosis of the liver	3,692	2,685	1,007	—	3	1,780	670	232	—	2	672	231	102
3. Appendicitis	1,002	501	500	115	139	222	17	9	89	142	244	15	10
4. Intestinal obstruction	1,680	811	869	41	27	356	172	215	22	17	401	154	275
5. Diverticulitis	5,750	2,749	3,001	—	2	1,228	795	723	—	2	1,171	870	957
6. Gallbladder and bile duct disease	2,910	875	2,035	3	13	503	198	158	6	122	1,438	255	214
7. Pancreatitis	775	452	323	3	18	322	59	48	4	20	195	42	62
8. Inflammatory bowel disease	71,894	37,348	34,547	650	1,608	25,917	5,368	3,804	615	1,405	23,337	4,615	4,576
9. Vascular insufficiency of bowel	262	117	144	2	2	37	32	44	1	2	43	31	68
O. Genitourinary diseases													
1. Nephritis and nephrosis													
a. Nephritis and nephrosis ^(c)													
	14,222	7,984	6,238	95	246	4,832	1,254	1,557	51	204	3,772	836	1,375
b. All nephritis and nephrosis													
	19,379	11,005	8,374	106	250	6,340	2,081	2,228	62	221	4,791	1,361	1,939
2. Benign prostatic hypertrophy													
	75,959	75,959	—	—	—	11,982	25,333	38,644	—	—	—	—	—
3. Urinary incontinence													
	228,264	32,822	195,442	—	—	15,678	8,993	8,152	14	2,475	146,147	22,034	24,773
4. Infertility													
	113,652	42,429	71,224	21	3,093	39,310	3	2	26	4,300	65,523	675	699
P. Skin diseases													
1. Eczema													
	165,196	61,351	103,845	12,215	7,877	34,561	3,643	3,055	34,593	26,207	39,007	2,621	1,418
2. Acne													
	76,882	38,698	38,184	5,790	21,522	11,383	2	—	2,291	18,549	17,344	—	—
3. Psoriasis													
	211,243	164,281	46,962	8,357	22,237	114,431	12,582	6,674	2,190	7,686	31,254	4,021	1,811
4. Ulcers													
	65,565	24,214	41,351	1,464	565	11,776	5,177	5,232	546	2,749	8,770	9,167	20,119

(continued)

Annex Table 9 (continued): Prevalence by age, sex and cause, Australia, 2003

Cause	Persons	Males					Females					
		0-14	15-24	25-64	65-74	75+	0-14	15-24	25-64	65-74	75+	
Q. Musculoskeletal diseases												
1. Rheumatoid arthritis	80,975	22,399	400	949	10,881	5,365	4,805	1,806	2,463	30,638	11,671	11,998
2. Osteoarthritis	300,655	121,027	2	171	48,322	31,603	40,929	—	—	39,293	44,686	95,649
3. Back pain ^(e)	994,222	484,146	4,589	12,318	290,862	91,892	84,484	3,734	12,779	316,614	83,690	93,259
4. Slipped disc	73,980	41,149	48	570	25,721	7,718	7,092	102	410	17,155	6,721	8,444
5. Occupational overuse syndrome	36,534	13,322	—	94	12,161	999	68	1	193	22,461	527	30
7. Gout	284,413	234,135	19	1,832	139,316	49,439	43,529	12	1,225	16,082	14,444	18,515
S. Oral conditions												
1. Dental caries	631,394	314,742	34,335	39,921	202,565	22,665	15,256	32,608	38,451	200,605	20,162	24,826
2. Periodontal disease	999,064	492,246	1,307	12,936	349,162	76,234	52,607	1,241	12,409	346,483	75,914	70,770
3. Edentulism	1,172,922	417,786	89	424	123,913	136,373	156,986	174	644	219,044	221,457	313,817
4. Pulpitis	167,107	82,372	11,262	7,203	54,211	5,646	4,050	10,781	6,984	54,830	6,013	6,128
Z. Ill-defined conditions												
2. Chronic fatigue syndrome	29,489	10,208	—	464	9,356	337	51	—	447	17,862	816	156

Due to the prevalence estimation process, an entry of one case in the above table does not necessarily represent one actual case from that particular cause/age group.

Notes

- (a) Excludes those with any other comorbid mental disorders.
- (b) Excludes alcoholic and hepatic cirrhosis.
- (c) Excludes diabetic-, congenital- and poisoning-related renal failure.
- (d) Includes both acute and chronic back pain.

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Abbreviations and symbols

ABS	Australian Bureau of Statistics
ACE	Assessing Cost-Effectiveness
ADHD	Attention deficit hyperactivity disorder
AHT	Airway hyper-responsiveness test
AIDS	Acquired immune deficiency syndrome
AIHW	Australian Institute of Health and Welfare
AMI	Acute myocardial infarction
ANZDATA	Australian and New Zealand Dialysis and Transplant Registry
AODTS	Alcohol and other drug treatment services
ART	Assisted reproductive technologies
ARIA+	Accessibility/Remoteness Index of Australia
ASGC	Australian Standard Geographical Classification
AUSDIAB	Australian Diabetes, Obesity, and Lifestyle Study
BCC	Basal cell carcinoma
BEACH	Bettering the Evaluation and Care of Health
BFV	Barmah Forest virus
BMD	Bone mineral density
BMES	Blue Mountains Eye Study
BMI	Body mass index
BPH	Benign prostatic hypertrophy
CEO	Chief Executive Officers
CFS	Chronic fatigue syndrome
COPD	Chronic obstructive pulmonary disease
CRA	Comparative Risk Assessment
CVD	Cardiovascular disease
DALY	Disability-adjusted life year
DHA	Australian Government Department of Health and Ageing
DHS	Department of Human Services (Victoria)
DisMod 2	Disease Modelling Software Package, Version 2
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders – Fourth edition
DW	Disability weight
EME	Established market economies
FEV	Forced expiratory volume

GAD	Generalised anxiety disorder
GBD	Global burden of disease
GDP	gross domestic product
GISCA	National Centre for Social Applications of Geographic Information Systems
HALE	Health-adjusted life expectancy
HCC	Hepatocellular cancer
HIV	Human immunodeficiency virus
ICD-10	International Classification of Diseases – 10th revision
ICD-9	International Classification of Diseases – 9th revision
ICF	International Classification of Functioning, Disability and Health
IDUs	Intravenous drug users
IGR	Intergenerational Report
IHD	Ischaemic heart disease
IOTF	International Obesity Task Force
MDE	Major depressive episodes
MHS	National Mental Health and Wellbeing Survey 1997
MMDS	Mortality Medical Data System
NCSCHE	National Cancer Statistics Clearing House
NEMESIS	North East Melbourne Stroke Incidence Study
NHMRC	National Health and Medical Research Council
NHS	National Health Survey
NIDSS	Notifiable Infectious Diseases Surveillance Systems
NMDS	National minimum data set
NOHS	National Oral Health Survey
NOHSC	National Occupational Health and Safety Commission
NPSU	National Perinatal Statistics Unit, AIHW
NZMOH	New Zealand Ministry of Health
OOS	Occupational overuse syndrome
OCD	Obsessive-compulsive disorder
PAF	Population attributable fractions
PBS	Pharmaceutical Benefits Scheme
PID	Pelvic inflammatory disease
PTO	Person trade-off valuation method
PTSD	Post-traumatic stress disorder
PVD	Peripheral vascular disease

PYLD	Prevalent years lost due to disability
PYLL	Potential years of life lost due to premature death
QALY	Quality-adjusted life year
RR	Relative risk
RRV	Ross River virus
SACR	South Australian Cancer Registry
SCC	Squamous cell carcinoma
SDs	Standard deviations
SDAC	ABS Survey of Disability, Ageing and Carers
SEIFA	Socio-economic Indexes for Areas
SF-12	Medical Outcomes Study 12 item Short-Form Health Survey
SF-36	Medical Outcomes Study 36 item Short-Form Health Survey
SIDS	Sudden infant death syndrome
SLA	Statistical Local Area
SMR	Standardised mortality ratio
STD	Sexually transmitted disease
TB	Tuberculosis
TOP	Termination of pregnancy
VCR	Victorian Cancer Registry
VEMD	Victorian Emergency Inpatient Dataset
VHPSS	Victorian Hospital Pathogens Surveillance Scheme
VAED	Victorian Admitted Episode Dataset
WHO	World Health Organization
YLD	Years lost due to disability
YLL	Years of life lost due to premature mortality
n.a.	not available
..	not applicable
n.e.c.	not elsewhere classified
—	nil or rounded down to zero

References

- Aalen OO, Farewell VT, De Angelis D, Day NE & Gill ON 1997. A Markov model for HIV disease progression including the effect of HIV diagnosis and treatment: application to AIDS prediction in England and Wales. *Statistics in Medicine* 16:2191–210.
- ABS (Australian Bureau of Statistics) 1993. Survey of Disability, Ageing and Carers 1993. Expanded CURF, RADL, ABS data available on request.
- ABS 1995. National Health Survey 1995. Expanded CURF, RADL, ABS data available on request.
- ABS 1996. Women's Safety Australia, 1996. Cat. no. 4128.0. Canberra: ABS.
- ABS 1997. Mental Health and Wellbeing of Adults, Australia, basic, 1997. Expanded CURF, RADL, ABS data available on request.
- ABS 1998a. Mental Health and Wellbeing Profile of Adults, Australia, 1997. Cat. no. 4326.0. Canberra: ABS.
- ABS 1998b. Survey of Disability, Ageing and Carers 1998. Expanded CURF, RADL, ABS data available on request.
- ABS 2001a. Australian Standard Geographical Classification (ASGC), 2001. Cat. no. 1216.0. Canberra: ABS.
- ABS 2001b. Census of Population and Housing: Socio-economic Indexes for Areas (SEIFA). Cat. no. 2033.0.55.001. Canberra: ABS.
- ABS 2001c. National Health Survey 2001. Expanded CURF, RADL, ABS data available on request.
- ABS 2003a. Population projections Australia 2002 to 2101. Cat. no. 3222.0. Canberra: ABS.
- ABS 2003b. Survey of Disability, Ageing and Carers, Australia 2003. Expanded CURF, RADL, ABS data available on request.
- ABS 2004. Births, Australia 2003. Cat. no. 3301.0. Canberra: ABS.
- ABS 2005. Causes of death Australia 2003. Cat. no. 3303.0. Canberra: ABS.
- ABS 2006. Population by age and sex, Australian States and Territories, June 2006. Cat. No. 3201.0, Spreadsheet Table 9, Canberra, ABS.
- Aguero-Torres H, Fratiglioni L, Guo Z, Viitanen M & Winblad B 1999. Mortality from dementia in advanced age: a 5-year follow-up study of incident dementia cases. *Journal of Clinical Epidemiology* 52:737–43.
- Ahern MJ & Smith MD 1997. Rheumatoid arthritis. *Medical Journal of Australia* 166:156–61.
- AIHW (Australian Institute of Health and Welfare) and AACR (Australasian Association of Cancer Registries) 2001. 2001 National Cancer Statistics Clearinghouse Database. Unit record file. Canberra: AIHW.
- AIHW 2003a. 2002–03 National Hospital Morbidity Database. Unit record file. Canberra: AIHW.
- AIHW 2003b. National Diabetes Register. Diabetes series no. 4. Cat. no. CVD 24. Canberra: AIHW.

AIHW 2004. National drug strategy household survey 2004. Unit record file. Canberra: AIHW.

AIHW 2005a. BreastScreen Australia monitoring report 2001–02. Cancer series no. 29. Cat. no. CAN 24. Canberra: AIHW.

AIHW 2005b. Health system expenditures on cancer and other neoplasms in Australia 2000–01. Health and welfare expenditure series no. 22. Cat. no. HWE 29. Canberra: AIHW.

AIHW 2005c. Health system expenditure on disease and injury in Australia 2000–01, second edition. Health and welfare expenditure series no. 21. Cat. no. HWE 28. Canberra: AIHW.

AIHW 2006. Australia's health 2006. Australia's health 10. Cat. no. AUS 73 Canberra: AIHW.

AIHW & AACR (Australasian Association of Cancer Registries) 2001. Cancer survival in Australia, 2001. Part 1. Cancer series no. 18. Cat. no. CAN 13. Canberra: AIHW.

AIHW, AACR & NCSG (National Cancer Strategies Group) 2005. Cancer incidence projections, Australia 2002 to 2011. Cancer series no. 30. Cat. no. CAN 25. Canberra: AIHW, AACR & NCSG.

AIHW, DHAC (Commonwealth Department of Health and Aged Care), AACR, BreastScreen Australia & National Breast Cancer Centre 2001. Breast cancer size and nodal status. Cancer monitoring series no. 2. Canberra: AIHW.

AIHW & DoHA (Australian Government Department of Health and Ageing) 2005. National Drug Strategy Household Survey 2004. Computer file. Canberra: Australian Social Science Data Archives, Australian National University.

AIHW DSRU (Dental Statistics and Research Unit) 2002. The South Australian Dental Longitudinal Study Five-year Follow-up. DSRU research report no. 3. Cat. no. DEN 102. Adelaide: AIHW DSRU.

AIHW NPSU (National Perinatal Statistics Unit) 2004. Australia's babies. AIHW bulletin no. 21. Cat. no. AUS 54. Canberra: AIHW.

AIHW: Armfield JM, Roberts-Thomson KF, Slade GD & Spencer AJ 2004. Dental health differences between boys and girls: the Child Dental Health Survey, Australia 2000. Dental statistics and research series no. 31. Cat. no. DEN 131. Canberra: AIHW.

AIHW: Britt H, Miller G, Knox S, Charles J, Valenti L, Henderson J et al. 2001. General practice activity in Australia 2000–01. General practice series no. 8. Cat. no. GEP 8. Canberra: AIHW.

AIHW: Carter KD & Stewart JF 2003. National Dental Telephone Interview Survey 2002. Cat. no. DEN 128. Adelaide: AIHW DSRU.

AIHW: Chalmers JM, Carter KD, Hodge CP, Fuss JM & Spencer AJ 2001. Adelaide dental study of nursing homes 1998. Dental statistics and research series no. 22. Cat. no. DEN 83. Adelaide: AIHW DSRU.

AIHW: Chalmers JM, Hodge CP, Fuss JM, Spencer AJ, Carter KD 2001. The Adelaide dental study of nursing homes: one-year follow up 1999. Dental statistics and research series no. 22. Cat. no. DEN 84. Adelaide: AIHW DSRU.

AIHW: Cripps R & Carman J 2001. Falls by the elderly in Australia: Trends and data for 1998. Injury research and statistics series 6. Cat. no. INJCAT 35 Canberra: AIHW.

AIHW: Dean J & Sullivan EA 2003. Assisted conception Australia and New Zealand 2000 and 2001. Assisted reproduction series no. 7. Cat. no. PER 22. Canberra: AIHW.

- AIHW: Driscoll T, Henley G & Harrison J 2004. The National Coroners Information System as an information tool for injury surveillance. Injury research and statistics series no. 21. Cat. no. INJCAT 60. Canberra: AIHW.
- AIHW: Ford J, Nassar N, Sullivan EA, Chambers G & Lancaster P 2003. Reproductive health indicators Australia 2002. Cat. no. PER 20. Canberra: AIHW.
- AIHW: Hurst T, Shafir E, Lancaster P & Day P 2001. Congenital malformations, Australia 1997. Birth defects series no. 4. Canberra: AIHW.
- AIHW: Laws P & Sullivan EA 2004. Australia's mothers and babies 2002. Perinatal statistics series no. 15. Cat. no. PER 28. Canberra: AIHW.
- AIHW: Lea A 1993. Management of incontinence: an information paper. Cat. no. AIHW 213. Canberra: AIHW.
- AIHW: Mathers C, Vos T & Stevenson C 1999. The burden of disease and injury in Australia. Cat. no. PHE 17. Canberra: AIHW.
- AIHW: Ridolfo B & Stevenson C 2001. The quantification of drug-caused mortality and morbidity in Australia, 1998. Drug statistics series no. 7. Cat. no. PHE 29. Canberra: AIHW.
- AIHW: Spencer AJ & Brennan DS 2002. Dentist practice activity in Australia 1983-84 to 1998-99. Dental statistics and research series no. 26. Cat. no. DEN 101. Adelaide: AIHW DSRU.
- AIHW: Turrell G, Stanley L, de Looper M & Oldenburg B 2006. Health inequalities in Australia. Health inequalities monitoring series no. 2. Cat. no. PHE 72. Canberra: AIHW.
- Air Monitoring Unit, EPA SA (Environment Protection Authority, South Australia) 2003. Air Monitoring Report 2002: Compliance with the National Environment Protection (Ambient Air Quality) Measure. Adelaide: EPA South Australia.
- Alamo Family Foot and Ankle Care 2005. Gout. Viewed 17 August 2006, <www.podlink.com/pathology/gout.htm>.
- Amin J, Heath T & Morrell S 1999. Hepatitis A in Australia in the 1990s: future directions in surveillance and control. *Communicable Diseases Intelligence* 23:113-20.
- Anand S & Hanson K 1997. Disability-adjusted life years: a critical review. *Journal of Health Economics* 16:685-702.
- Anderson HR & Cook DG 1997. Passive smoking and sudden infant death syndrome: review of the epidemiological evidence. *Thorax* 52:1003-9.
- Andrews G, Corry J, Slade T, Issakidis C & Swanston H 2004. Child sexual abuse. In: Ezzati M, Lopez AD, Rodgers A & Murray CJL (eds). *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Vol. 2. Geneva: World Health Organization, 1851-940.
- Andrews G & Slade T 2002. The classification of anxiety disorders in ICD-10 and DSM-IV: a concordance analysis. *Psychopathology* 35:100-6.
- Andrews G, Stewart G, Allen R & Henderson AS 1990. The genetics of six neurotic disorders: a twin study. *Journal of Affective Disorders* 19:23-9.
- Andrews R, Herceg A & Roberts C 1997. Pertussis notifications in Australia, 1991 to 1997. *Communicable Diseases Intelligence* 21:145-8.
- Angst J & Preisig M 1995. Course of a clinical cohort of unipolar, bipolar and schizoaffective patients: results of a prospective study from 1959 to 1985. *Swiss Archives of Neurology and Psychiatry* 146:5-16.

- Annegers JF, Hauser WA & Elveback LR 1979. Remission of seizures and relapse in patients with epilepsy. *Epilepsia* 20:729-37.
- Arias I 2004. The legacy of child maltreatment: long-term health consequences for women. *Journal of Women's Health* 13:468-73.
- Atkinson JH 2004. Chronic back pain: searching for causes and cures. *Journal of Rheumatology* 31:2323-5.
- Baker SR, Stacey MC, Singh G, Hoskin SE & Thompson PJ 1992. Aetiology of chronic leg ulcers. *European Journal of Vascular Surgery* 6:245-51.
- Barendregt JJ, Van Oortmarssen GJ, Vos T & Murray CJ 2003. A generic model for the assessment of disease epidemiology: the computational basis of DisMod II. *Population Health Metrics* 1:4.
- Barnard PD 1993. National Oral Health Survey of Australia 1987-88. Canberra: AGPS.
- Barnett MH, Williams DB, Day S, Macaskill P & McLeod JG 2003. Progressive increase in incidence and prevalence of multiple sclerosis in Newcastle, Australia: a 35-year study. *Journal of the Neurological Sciences* 213:1-6.
- Bauman A, Mitchell CA, Henry RL, Robertson CF, Abramson MJ, Comino EJ et al. 1992a. Asthma morbidity in Australia: an epidemiological study. *Medical Journal of Australia* 156:827-31.
- Bauman A, Young L, Peat JK, Hunt J & Larkin P 1992b. Asthma under-recognition and under-treatment in an Australian community. *Australian and New Zealand Journal of Medicine* 22:36-40.
- Benito-Leon J, Bermejo-Pareja F, Morales-Gonzalez JM, Porta-Etessam J, Trincado R, Vega S et al. 2004. Incidence of Parkinson disease and parkinsonism in three elderly populations of central Spain. *Neurology* 62:734-41.
- Berger K, Breteler MM, Helmer C, Inzitari D, Fratiglioni L, Trenkwalder C et al. 2000. Prognosis with Parkinson's disease in Europe: a collaborative study of population-based cohorts. Neurologic Diseases in the Elderly Research Group. *Neurology* 54:S24-7.
- Berlin JA & Colditz GA 1990. A meta-analysis of physical activity in the prevention of coronary heart disease. *American Journal of Epidemiology* 132:612-28.
- Blair SN, Cheng Y & Holder JS 2001. Is physical activity or physical fitness more important in defining health benefits? *Medicine and Science in Sports and Exercise* 33:S379-99; discussion S419-20.
- Booth BM, Fortney SM, Fortney JC, Curran GM & Kirchner JE 2001. Short-term course of drinking in an untreated sample of at-risk drinkers. *Journal of Studies on Alcohol* 62:580-8.
- Bosch FX & Munoz N 2002. The viral etiology of cervical cancer. *Virus Research* 89:183-90.
- Bower C, Rudy E, Ryan A & Cosgrove P 2004. Report of the Birth Defects Registry of Western Australia. Perth: Women's and Children's Health Service, Government of Western Australia Department of Health.
- Brechot C, Jaffredo F, Lagorce D, Gerken G, Meyer zum Buschenfelde K, Papakonstantinou A et al. 1998. Impact of HBV, HCV and GBV-C/HGV on hepatocellular carcinomas in Europe: results of a European concerted action. *Journal of Hepatology* 29:173-83.
- Brennan DS & Spencer AJ 2002. Influence of patient, visit, and oral health factors on dental service provision. *Journal of Public Health Dentistry* 62:148-57.

- Brennan DS & Spencer AJ 2004. Disability weights for the burden of oral disease in South Australia. *Population Health Metrics* 2:7.
- Brennan DS & Spencer AJ 2005. Comparison of a generic and a specific measure of oral health related quality of life. *Community Dental Health* 22:11-18.
- Bronnimann S & Burrows B 1986. A prospective study of the natural history of asthma: remission and relapse rates. *Chest* 90:480-4.
- Brotherton J, McIntyre P, Puech M, Wang H, Gidding H, Hull B et al. 2004. Vaccine preventable diseases and vaccination coverage in Australia 2001 to 2002. *Communicable Diseases Intelligence* 28.
- Brown WJ, Dobson AJ, Bryson L & Byles JE 1999. Women's Health Australia: on the progress of the main cohort studies. *Journal of Women's Health and Gender-Based Medicine* 8:681-8.
- Bull F 2003. Defining physical inactivity. *Lancet* 361:258-9.
- Burns L, Mattick RP & Cooke M 2006. The use of record linkage to examine illicit drug use in pregnancy. *Addiction* 101:873-82.
- Byrne E 1992. RSI revisited. *Medical Journal of Australia* 156:372-3.
- Card T, Hubbard R & Logan RF 2003. Mortality in inflammatory bowel disease: a population-based cohort study. *Gastroenterology* 125:1583-90.
- Carvalho JC, D'Hoore W & Van Nieuwenhuysen JP 2004. Caries decline in the primary dentition of Belgian children over 15 years. *Community Dentistry and Oral Epidemiology* 32:277-82.
- CDA (Communicable Diseases Australia) 2003. National Notifiable Diseases Surveillance System (NNDSS). *Communicable Diseases Australia*, Department of Health and Ageing, Canberra. Viewed 26 September 2006, <www9.health.gov.au/cda/Source/CDA-index.cfm>.
- CDC (Centers for Disease Control and Prevention) 2001. National Hepatitis C Prevention Strategy: a comprehensive strategy for the prevention and control of hepatitis c virus infection and its consequences. Division of Viral Hepatitis, National Center for Infectious Diseases, Centers for Disease Control and Prevention. Viewed 17 August 2006, <www.cdc.gov/ncidod/diseases/hepatitis/c/plan/strategy.pdf>.
- Chan A, Scott J, Nguyen AM & Green P 2003. *Pregnancy outcome in South Australia 2002*. Adelaide: Department of Human Services.
- Chan DK, Cordato D, Karr M, Ong B, Lei H, Liu J et al. 2005. Prevalence of Parkinson's disease in Sydney. *Acta Neurologica Scandinavica* 111:7-11.
- Chan DK, Dunne M, Wong A, Hu E, Hung WT & Beran RG 2001. Pilot study of prevalence of Parkinson's disease in Australia. *Neuroepidemiology* 20:112-7.
- Chancellor AM, Slattery JM, Fraser H, Swingler RJ, Holloway SM & Warlow CP 1993. The prognosis of adult-onset motor neuron disease: a prospective study based on the Scottish Motor Neuron Disease Register. *Journal of Neurology* 240:339-46.
- Chen Z, Peto R, Collins R, MacMahon S, Lu J & Li W 1991. Serum cholesterol concentration and coronary heart disease in population with low cholesterol concentrations. *British Medical Journal (Clinical Research Edition)* 303:276-82.
- Chiarelli P, Bower W, Attia J, Sibbritt D & Wilson A 1999. *The prevalence of faecal incontinence within the community: a systematic review*. Canberra: National Continence

Management Strategy, Australian Government Department of Health, Aged and Community Care.

Ciampi L 1980. Three lectures in schizophrenia. the natural history of schizophrenia in the long term. *British Journal of Psychiatry* 136:413–20.

Clifford GM, Smith JS, Plummer M, Munoz N & Franceschi S 2003. Human papilloma virus types in invasive cervical cancer worldwide: a meta-analysis. *British Journal of Cancer* 88:63–73.

Coale A & Guo G 1989. Revised regional model life tables at very low levels of mortality. *Population Index* 55:613–43.

Cohen ML, Arroyo JF, Champion GD & Browne CD 1992. In search of the pathogenesis of refractory cervicobrachial pain syndrome: a deconstruction of the RSI phenomenon. *Medical Journal of Australia* 1516:432:6.

Cohen AJ, Anderson HR, Ostro B, Pandey KD, Krzyzanowski M, Künzli N et al. 2004. Urban air pollution. In: Ezzati M, Lopez AD, Rodgers A & Murray CJL (eds). *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Vol. 2. Geneva: World Health Organization, 1353–433.

Concha-Barrientos M, Nelson DI, Driscoll T, Steenland NK, Punnett L, Fingerhut MA et al. 2004. Selected occupational risk factors. In: Ezzati M, Lopez AD, Rodgers A & Murray CJL (eds). *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Vol. 2. Geneva: World Health Organization, 1651–801.

Cowan J, Macdessi J, Stark A & Morgan G 1980. Incidence of Duchenne muscular dystrophy in New South Wales and Australian Capital Territory. *Journal of Medical Genetics* 17:245–9.

Darke S & Ross J 2002. Suicide among heroin users: rates, risk factors and methods. *Addiction* 97:1383–94.

Davies MJ, Spencer AJ & Slade GD 1997. Trends in dental caries experience of school children in Australia – 1977 to 1993. *Australian Dental Journal* 42:389–94.

de Rijk MC, Breteler MM, Graveland GA, Ott A, Grobbee DE, van der Meche FG et al. 2000. Prevalence of Parkinson's disease in Europe: a collaborative study of population-based cohorts. *Neurology* 54(Suppl 5):S21–3.

Degenhardt L, Rendle V, Hall W, Gilmour S & Law M 2004. *Estimating the number of current regular heroin users in NSW and Australia 1997–2002*. Sydney: National Drug and Alcohol Research Centre, University of New South Wales.

Department of Health of Western Australia, University of Western Australia, Curtin University of Technology & The Telethon Institute for Child Health Research 2005. Customised data from the Epidemiology Branch and Western Australia Data Linkage System 1989–2003.

Dewey ME & Saz P 2001. Dementia, cognitive impairment and mortality in persons aged 65 and over living in the community: a systematic review of the literature. *International Journal of Geriatric Psychiatry* 16:751–61.

Deyo RA, Loeser JD & Bigos SJ 1990. Herniated lumbar intervertebral disk. *Annals of Internal Medicine* 112:598–603.

DHS (Department of Human Services, Victoria) 1999a. *The Victorian Burden of Disease Study: morbidity*. Melbourne: DHS.

DHS 1999b. *The Victorian Burden of Disease Study: mortality*. Melbourne: DHS.

- DHS 2002. Victorian Population Health Survey 2001. Melbourne: DHS.
- DHS 2005. The Victorian Burden of Disease Study: mortality and morbidity in 2001. Melbourne: Victorian Government Department of Human Services.
- DHS 2006. Burden of disease (BoD) – LGAs and regions. Melbourne: DHS. Viewed 16 February 2006, <www.health.vic.gov.au/healthstatus/bod/bod_reg.htm>.
- Dimitriou G, Greenough A, Mantagos JS, Davenport M & Nicolaidis KH 2000. Morbidity in infants with antenatally-diagnosed anterior abdominal wall defects. *Pediatric Surgery International* 16:404–7.
- Doran MF, Pond GR, Crowson CS, O’Fallon WM & Gabriel SE 2002. Trends in incidence and mortality in rheumatoid arthritis in Rochester, Minnesota, over a forty-year period. *Arthritis and Rheumatism* 46:625–31.
- DPIWE (Department of Primary Industries, Water and Environment, Tasmania) 2004. PM10 annual statistics Hobart 2000–2004. Hobart: DPIWE. Viewed 30 May 2006, <www.dpiw.tas.gov.au/inter.nsf/Images/LBUN-63R7UV>.
- DPIWE 2006. Major air pollutants. Hobart: DPIWE. Viewed 30 May 2006, <www.dpiw.tas.gov.au/inter.nsf/WebPages/MCLE-5XF43K>.
- Driscoll T, Mitchell R, Mandryk J, Healey S, Hendrie L & Hull B 2001. Work-related fatalities in Australia, 1989 to 1992: an overview. *Journal of Occupational Health and Safety – Australia and New Zealand* 17:45–66.
- Driscoll TR, Harrison JE & Steenkamp M 2004. Alcohol and drowning in Australia. *Injury Control and Safety Promotion* 11:175–81.
- Drummer O 1994. Drugs in drivers killed in Australian road traffic accidents: the use of responsibility analysis to investigate the contribution of drugs to fatal accidents. Melbourne: Victorian Institute of Forensic Pathology & Department of Forensic Pathology, Monash University.
- Drummer OH, Gerostamoulos J, Batziris H, Chu M, Caplehorn J, Robertson MD et al. 2004. The involvement of drugs in drivers of motor vehicles killed in Australian road traffic crashes. *Accident Analysis and Prevention* 36:239–48.
- Dunstan D, Zimmet P, Welborn T et al. 2001. Diabetes and associated disorders in Australia: the accelerating epidemic. Report of the Australian Diabetes, Obesity and Lifestyle Study. Melbourne: International Diabetes Institute.
- Dunstan DW, Zimmet PZ, Welborn TA, De Courten MP, Cameron AJ, Sicree RA et al. 2002. The rising prevalence of diabetes and impaired glucose tolerance: the Australian Diabetes, Obesity and Lifestyle Study. *Diabetes Care* 25:829–34.
- Dyck PJ, Kratz KM, Karnes JL, Litchy WJ, Klein R, Pach JM et al. 1993. The prevalence by staged severity of various types of diabetic neuropathy, retinopathy, and nephropathy in a population-based cohort: the Rochester Diabetic Neuropathy Study. *Neurology* 43:817–24.
- Eastern Stroke and Coronary Heart Disease Collaborative Research Group 1998. Blood pressure, cholesterol, and stroke in eastern Asia. *Lancet* 352:1801–7.
- Eaton CB 1992. Relation of physical activity and cardiovascular fitness to coronary heart disease. Part I: A meta-analysis of the independent relation of physical activity and coronary heart disease. *Journal of the American Board of Family Practice* 5:31–42.
- Egger M, Smith GD & Altman DG (eds) 2001. Systematic reviews in health care: meta-analysis in context. London: BMJ Publishing Group.

- Eide GE & Heuch I 2001. Attributable fractions: fundamental concepts and their visualization. *Statistical Methods in Medical Research* 10:159–93.
- Einfeld SL & Tonge BJ 1996. Population prevalence of psychopathology in children and adolescents with intellectual disability: II. Epidemiological findings. *Journal of Intellectual Disability Research* 40(Pt 2):99–109.
- Elbaz A, Bower JH, Peterson BJ, Maraganore DM, McDonnell SK, Ahlskog JE et al. 2003. Survival study of Parkinson disease in Olmsted County, Minnesota. *Archives of Neurology* 60:91–6.
- Emery AE 1991. Population frequencies of inherited neuromuscular diseases – a world survey. *Neuromuscular Disorders* 1:19–29.
- English DR, Holman CDJ, Milne E, Winter MG, Hulse GK, Codde JP et al. 1995. The quantification of drug-caused morbidity and mortality in Australia. Canberra: Commonwealth Department of Human Services and Health.
- English RM & Bennett SA 1990. Iron status of Australian children. *Medical Journal of Australia* 152:582–6.
- EPOS Group (European Prospective Osteoporosis Study Group) 2002. The relationship between bone density and incident vertebral fracture in men and women. *Journal of Bone and Mineral Research* 17:2214–21.
- Ezzati M, Hoorn SV, Rodgers A, Lopez AD, Mathers CD & Murray CJ 2003. Estimates of global and regional potential health gains from reducing multiple major risk factors. *Lancet* 362:271–80.
- Ezzati M, Lopez AD, Rodgers A & Murray CJL (eds) 2004a. Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors. Geneva: World Health Organization, 1229–30.
- Ezzati M, Vander Hoorn S, Rodgers A, Lopez AD, Mathers CD & Murray CJL 2004b. Potential health gains from reducing multiple risk factors. In: Ezzati M, Lopez AD, Rodgers A & Murray CJL (eds). *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Vol. 2. Geneva: World Health Organization, 2167–90.
- Fall PA, Saleh A, Fredrickson M, Olsson JE & Granerus AK 2003. Survival time, mortality, and cause of death in elderly patients with Parkinson's disease: a 9-year follow-up. *Movement Disorders* 18:1312–16.
- Field MJ & Gold GM (eds) 1998. *Summarizing population health: directions for the development and application of population metrics*. Washington, D.C.: Institute of Medicine, National Academy Press.
- Forbes RB, Colville S, Cran GW & Swingler RJ 2004. Unexpected decline in survival from amyotrophic lateral sclerosis/ motor neurone disease. *Journal of Neurology, Neurosurgery and Psychiatry* 75:1753–5.
- Fries JF 1980. Aging, natural death, and the compression of morbidity. *New England Journal of Medicine* 303:130–5.
- Fujiwara S, Kasagi F, Masunari N, Naito K, Suzuki G & Fukunaga M 2003. Fracture prediction from bone mineral density in Japanese men and women. *Journal of Bone and Mineral Research* 18:1547–53.

- Fukuda K, Straus SE, Hickie I, Sharpe MC, Dobbins JG & Komaroff A 1994. The chronic fatigue syndrome: a comprehensive approach to its definition and study. *International Chronic Fatigue Syndrome Study Group. Annals of Internal Medicine* 121:953–9.
- Gare BA & Fasth A 1992. Epidemiology of juvenile chronic arthritis in southwestern Sweden: a 5-year prospective population study. *Pediatrics* 90:950–8.
- Gaziano JM, Manson JE, Branch LG, Colditz GA, Willett WC & Buring JE 1995. A prospective study of consumption of carotenoids in fruits and vegetables and decreased cardiovascular mortality in the elderly. *Annals of Epidemiology* 5:255–60.
- Genant HK, Cooper C, Poor G, Reid I, Ehrlich G, Kanis J et al. 1999. Interim report and recommendations of the World Health Organization Task-Force for Osteoporosis. *Osteoporosis International* 10:259–64.
- Gergen PJ, Mullally DI & Evans R, 3rd 1988. National survey of prevalence of asthma among children in the United States, 1976 to 1980. *Pediatrics* 81:1–7.
- Gilchrist PN, Ben-Tovim DI, Hay PJ, Kalucy RS & Walker MK 1998. Eating disorders revisited. I: Anorexia nervosa. *Medical Journal of Australia* 169:438–41.
- Gooding D & Riordan D 2004. Ambient air quality monitoring – South Australia 1979–2003. Adelaide: EPA South Australia.
- Griffiths AM 1995. Inflammatory bowel disease. *Adolescent Medicine* 6:351–68.
- Griffiths AM 2004. Specificities of inflammatory bowel disease in childhood. Best practice and research. *Clinical Gastroenterology* 18:509–23.
- Grilo CM, Sanislow CA, Gunderson JG, Pagano ME, Yen S, Zanarini MC et al. 2004. Two-year stability and change of schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. *Journal of Consulting and Clinical Psychology* 72:767–75.
- Grimwood K, Anderson VA, Bond L, Catroppa C, Hore RL, Keir EH et al. 1995. Adverse outcomes of bacterial meningitis in school-age survivors. *Pediatrics* 95:646–56.
- Guccione AA, Felson DT & Anderson JJ 1990. Defining arthritis and measuring functional status in elders: methodological issues in the study of disease and physical disability. *American Journal of Public Health* 80:945–9.
- Guy RJ, Andrews RM, Kelly HA, Leydon JA, Riddell MA, Lambert SB et al. 2004. Mumps and rubella: a year of enhanced surveillance and laboratory testing. *Epidemiology and Infection* 132:391–8.
- Haby MM, Carter R, Mihalopoulos C, Magnus A, Sanderson K, Andrews G et al. 2004. Assessing cost-effectiveness – mental health: introduction to the study and methods. *Australian and New Zealand Journal of Psychiatry* 38:569–78.
- Haby MM, Vos T, Carter R, Moodie M, Markwick A, Magnus A, Tay-Teo KS, & Swinburn B 2006. A new approach to assessing the health benefit from obesity interventions in children and adolescents: the assessing cost-effectiveness in obesity project. *Int J Obes (Lond)* 30(10): 1463-75.
- Hakala M, Nieminen P & Koivisto O 1994. More evidence from a community based series of better outcome in rheumatoid arthritis. Data on the effect of multidisciplinary care on the retention of functional ability. *Journal of Rheumatology* 21:1432–7.
- Hall G & OzFoodNet Working Group 2004. Results from the National Gastroenteritis Survey 2001 – 2002. Canberra: National Centre for Epidemiology and Population Health, The Australian National University.

- Harding CM, Brooks GW, Ashigawa T, Strauss JS & Breier A 1987. The Vermont Longitudinal Study of Persons With Severe Mental Illness, 1: methodology, study sample, and overall status 32 years later. *American Journal of Psychiatry* 144:718–26.
- Harris EC & Barraclough B 1998. Excess mortality of mental disorder. *British Journal of Psychiatry* 173:11–53.
- Harrison G, Hopper K, Craig T, Laska E, Siegel C & Wanderling J 2001. Recovery from psychotic illness: a 15- and 25-year international follow-up study. *British Journal of Psychiatry* 178:506–17.
- Harvey RJ, Skelton-Robinson M & Rossor MN 2003. The prevalence and causes of dementia in people under the age of 65 years. *Journal of Neurology, Neurosurgery and Psychiatry* 74:1206–9.
- Hasselblad V & Hedges LV 1995. Meta-analysis of screening and diagnostic tests. *Psychological Bulletin* 117:167–78.
- Helgason L 1990. Twenty years' follow-up of first psychiatric presentation for schizophrenia: what could have been prevented? *Acta Psychiatrica Scandinavica* 81:231–5.
- Helme RD, LeVasseur SA & Gibson SJ 1992. RSI revisited: evidence for psychological and physiological differences from an age, sex and occupation matched control group. *Australian and New Zealand Journal of Medicine* 22:23–9.
- Helmer C, Joly P, Letenneur L, Commenges D & Dartigues JF 2001. Mortality with dementia: results from a French prospective community-based cohort. *American Journal of Epidemiology* 154:642–8.
- Hely MA, Morris JG, Traficante R, Reid WG, O'Sullivan DJ & Williamson PM 1999. The Sydney multicentre study of Parkinson's disease: progression and mortality at 10 years. *Journal of Neurology, Neurosurgery and Psychiatry* 67:300–7.
- Hendriksen C, Kreiner S & Binder V 1985. Long term prognosis in ulcerative colitis – based on results from a regional patient group from the county of Copenhagen. *Gut* 26:158–63.
- Henry MJ, Pasco JA, Pocock NA, Nicholson GC & Kotowicz MA 2004. Reference ranges for bone densitometers adopted Australia-wide: Geelong Osteoporosis Study. *Australasian Radiology* 48:473–5.
- Herceg A 1997. The decline of Haemophilus influenzae type b disease in Australia. *Communicable Diseases Intelligence* 21:173–6.
- Herlofson K, Lie SA, Arslan D & Larsen JP 2004. Mortality and Parkinson disease: a community based study. *Neurology* 62:937–42.
- Hickie I, Davenport T, Wakefield D, Vollmer-Conna U, Cameron B, Vernon S et al. submitted 2005. Viral and non-viral pathogens precipitate post-infective and chronic fatigue syndromes: a prospective cohort study. *British Medical Journal*.
- Hicks GS, Duddlestone DN, Russell LD, Holman HE, Shepherd JM & Brown CA 2002. Low back pain. *American Journal of the Medical Sciences* 324:207–11.
- Hill JC & Schoener EP 1996. Age-dependent decline of attention deficit hyperactivity disorder. *American Journal of Psychiatry* 153:1143–6.
- Holman CD, Bass AJ, Rouse IL & Hobbs MS 1999. Population-based linkage of health records in Western Australia: development of a health services research linked database. *Australian and New Zealand Journal of Public Health* 23:453–9.

- Hopcraft MS & Morgan MV 2005. Comparison of radiographic and clinical diagnosis of approximal and occlusal dental caries in a young adult population. *Community Dentistry and Oral Epidemiology* 33:212-18.
- Huber G, Gross G, Schuttler R & Linz M 1980. Longitudinal studies of schizophrenic patients. *Schizophrenia Bulletin* 6:592-605.
- Hughes TA, Ross HF, Mindham RH & Spokes EG 2004. Mortality in Parkinson's disease and its association with dementia and depression. *Acta Neurologica Scandinavica* 110:118-23.
- Jaaskelainen J 1986. Seemingly complete removal of histologically benign intracranial meningioma: late recurrence rate and factors predicting recurrence in 657 patients. A multivariate analysis. *Surgical Neurology* 26:461-9.
- Jablensky A, McGrath J, Herrman H, Castle D, Gureje O, Morgan V et al. 1999. National Survey of Mental Health and Wellbeing Report 4: people living with psychotic illness: an Australian study 1997-98. Canberra: Mental Health Branch, Commonwealth Department of Health and Aged Care.
- Jagger C, Andersen K, Breteler MM, Copeland JR, Helmer C, Baldereschi M et al. 2000. Prognosis with dementia in Europe: a collaborative study of population-based cohorts. Neurologic Diseases in the Elderly Research Group. *Neurology* 54:516-20.
- James WPT, Jackson-Leach R, Ni Mhurchu C, Kalamar E, Shayeghi M, Rigby NJ et al. 2004. Overweight and obesity (high body mass index). In: Ezatti M, Lopez AD, Rodgers A & Murray CJL (eds). *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Vol. 1. Geneva: World Health Organization, 497-596.
- Jarrett RJ, Shipley MJ & Rose G 1982. Weight and mortality in the Whitehall Study. *British Medical Journal (Clinical Research Edition)* 285:535-7.
- Jones G, Nguyen T, Sambrook PN, Lord SR, Kelly PJ & Eisman JA 1995. Osteoarthritis, bone density, postural stability, and osteoporotic fractures: a population based study. *Journal of Rheumatology* 22:921-5.
- Jousilahti P, Vartiainen E, Tuomilehto J & Puska P 1999. Sex, age, cardiovascular risk factors, and coronary heart disease: a prospective follow-up study of 14, 786 middle-aged men and women in Finland. *Circulation* 99:1165-72.
- Kaldor J & McDonald A 2003. HIV/AIDS surveillance systems in Australia. *Journal of Acquired Immune Deficiency Syndromes* 32(Suppl 1):S18-23.
- Kaldor JM, Plant AJ, Thompson SC, Longbottom H & Rowbottom J 1996. The incidence of hepatitis B infection in Australia: an epidemiological review. *Medical Journal of Australia* 165:322-6.
- Karr M, Alperstein G, Causer J, Mira M, Lammi A & Fett MJ 1996. Iron status and anaemia in preschool children in Sydney. *Australian and New Zealand Journal of Public Health* 20:618-22.
- Keel PK, Mitchell JE, Miller KB, Davis TL & Crow SJ 1999. Long-term outcome of bulimia nervosa. *Archives of General Psychiatry* 56:63-9.
- Kelley DE & Goodpaster BH 2001. Effects of exercise on glucose homeostasis in Type 2 diabetes mellitus. *Medicine and Science in Sports and Exercise* 33:S495-501; discussion S28-9.
- Kerr C, Morrell S, Salkeld G, Corbett S, Taylor R & Webster F 1996. Best estimates of the magnitude of health effects of occupational exposure to hazardous substances. Canberra: National Institute of Occupational Health and Safety.

- Kesaniemi YK, Danforth E, Jr, Jensen MD, Kopelman PG, Lefebvre P & Reeder BA 2001. Dose-response issues concerning physical activity and health: an evidence-based symposium. *Medicine and Science in Sports and Exercise* 33:S351-8.
- Khaw KT & Barrett-Connor E 1987. Dietary fibre and reduced ischaemic heart disease mortality rates in men and women: a 12-year prospective study. *American Journal of Epidemiology* 126:1093-102.
- Kilkenny M, Merlin K, Plunkett A & Marks R 1998. The prevalence of common skin conditions in Australian school students: 3. Acne vulgaris. *British Journal of Dermatology* 139:840-5.
- Knuiman MW, James AL, Divitini ML, Ryan G, Bartholomew HC & Musk AW 1999. Lung function, respiratory symptoms, and mortality: results from the Busselton Health Study. *Annals of Epidemiology*. 9:297-306.
- Kohl HW, 3rd 2001. Physical activity and cardiovascular disease: evidence for a dose response. *Medicine and Science in Sports and Exercise* 33:S472-83; discussion S93-4.
- Kotowicz M 2005. Geelong Osteoporosis Study – age-specific bone mineral density means and standard deviations. Email, 16 September.
- Kroger H, Huopio J, Honkanen R, Tuppurainen M, Puntila E, Alhava E et al. 1995. Prediction of fracture risk using axial bone mineral density in a perimenopausal population: a prospective study. *Journal of Bone and Mineral Research* 10:302-6.
- Krum H 2001. Guidelines for management of patients with chronic heart failure in Australia. *Medical Journal of Australia* 174:459-66.
- Kulich M, Rosenfeld M, Goss CH & Wilmott R 2003. Improved survival among young patients with cystic fibrosis. *Journal of Pediatrics* 142:631-6.
- Kurth AA, Rau S, Wang C & Schmitt E 1996. Treatment of lumbar disc herniation in the second decade of life. *European Spine Journal* 5:220-4.
- Landers J, Kleinschmidt A, Wu J, Burt B, Ewald D & Henderson T 2005. Prevalence of cicatricial trachoma in an indigenous population of Central Australia: the Central Australian Trachomatous Trichiasis Study (CATTs). *Clinical and Experimental Ophthalmology* 33:142-6.
- Law MG, Dore GJ, Bath N, Thompson S, Crofts N, Dolan K et al. 2003. Modelling hepatitis C virus incidence, prevalence and long-term sequelae in Australia, 2001. *Internal Journal of Epidemiology* 32:717-24.
- Law MG, Lynskey M, Ross J & Hall W 2001. Back-projection estimates of the number of dependent heroin users in Australia. *Addiction* 96:433-43.
- Law MR, Wald NJ & Thompson SG 1994. By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease? *British Medical Journal (Clinical Research Edition)* 308:367-72.
- Lawes CM, Rodgers A, Bennett DA, Parag V, Suh I, Ueshima H et al. 2003. Blood pressure and cardiovascular disease in the Asia Pacific region. *Journal of Hypertension* 21: 707-16.
- Lawes CMM, Vander Hoorn S, Law MR, Elliott E, MacMahon S & Rodgers A 2004a. High blood pressure. In: Ezzati M, Lopez AD, Rodgers A & Murray CJL (eds). *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Vol. 1. Geneva: World Health Organization, 281-389.

- Lawes CMM, Vander Hoorn S, Law MR & Rodgers A 2004b. High cholesterol. In: Ezzati M, Lopez AD, Rodgers A & Murray CJL (eds). Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors. Vol. 1. Geneva: World Health Organization, 391–496.
- Lee C, Dobson A, Brown W, Bryson L, Byles J, Warner-Smith P et al. 2005. Cohort profile: the Australian Longitudinal Study on Women's Health. *International Journal of Epidemiology* 34:987–91.
- Lee WM 1997. Hepatitis B virus infection. *New England Journal of Medicine* 337:1733–45.
- Leff J, Sartorius N, Jablensky A, Korten A & Ernberg G 1992. The International Pilot Study of Schizophrenia: five-year follow-up findings. *Psychological Medicine* 22:131–45.
- Leonardi M & Mathers C 2003. Global burden of migraine in the year 2000: summary of methods and data sources. GBD 2000 Working Paper. Geneva: World Health Organization.
- Levy DT, Mallonee S, Miller TR, Smith GS, Spicer RS, Romano EO et al. 2004. Alcohol involvement in burn, submersion, spinal cord, and brain injuries. *Medical Science Monitor* 10: CR17–24.
- Lewin TJ & Carr VJ 1998. Rates of treatment of schizophrenia by general practitioners. A pilot study. *Medical Journal of Australia* 168:166–9.
- Liu S & Manson JE 2001. What is the optimal weight for cardiovascular health? *British Medical Journal (Clinical Research Edition)* 322:631–2.
- Lloyd AR, Hickie I, Boughton CR, Spencer O & Wakefield D 1990. Prevalence of chronic fatigue syndrome in an Australian population. *Medical Journal of Australia* 153:522–8.
- Lobo A, Launer LJ, Fratiglioni L, Andersen K, Di Carlo A, Breteler MM et al. 2000. Prevalence of dementia and major subtypes in Europe: a collaborative study of population-based cohorts. Neurologic Diseases in the Elderly Research Group. *Neurology* 54:S4–9.
- Lock K, Pomerleau J, Causer L & McKee M 2004. Low fruit and vegetable consumption. In: Ezzati M, Lopez AD, Rodgers A & Murray CJL (eds). Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors. Vol. 1. Geneva: World Health Organization, 597–728.
- Looker AC, Wahner HW, Dunn WL, Calvo MS, Harris TB, Heyse SP et al. 1998. Updated data on proximal femur bone mineral levels of US adults. *Osteoporosis International* 8:468–89.
- Lopez AD, Mathers CD, Ezzati M, Jamison DT & Murray CJL (eds) 2006. Global burden of disease and risk factors. New York: Oxford University Press & the World Bank.
- Ludman L & Spitz L 2003. Quality of life after gastric transposition for oesophageal atresia. *Journal of Pediatric Surgery* 38:53–7; discussion 57.
- Mackenzie JS, Broom AK, Hall RA, Johansen CA, Lindsay MD, Phillips DA et al. 1998. Arboviruses in the Australian region, 1990 to 1998. *Communicable Diseases Intelligence* 22:93–100.
- Mak DB & Plant AJ 2001. Trichiasis in Aboriginal people of the Kimberley region of Western Australia. *Clinical and Experimental Ophthalmology* 29:7–11.
- Manson JE, Colditz GA, Stampfer MJ, Willett WC, Rosner B, Monson RR et al. 1990. A prospective study of obesity and risk of coronary heart disease in women. *New England Journal of Medicine* 322:882–9.

- Manson JE, Greenland P, LaCroix AZ, Stefanick ML, Mouton CP, Oberman A et al. 2002. Walking compared with vigorous exercise for the prevention of cardiovascular events in women. *New England Journal of Medicine* 347:716–25.
- March LM, Schwarz JM, Carfrae BH & Bagge E 1998. Clinical validation of self-reported osteoarthritis. *Osteoarthritis and Cartilage* 6:87–93.
- Marks R, Plunkett A, Merlin K & Jenner N 1999. Atlas of common skin diseases in Australia. Melbourne: Department of Dermatology, St Vincent's Hospital.
- Martinez FD, Wright AL, Taussig LM, Holberg CJ, Halonen M & Morgan WJ 1995. Asthma and wheezing in the first six years of life. The Group Health Medical Associates. *New England Journal of Medicine* 332:133–8.
- Mason C, Porter SR, Madland G & Parry J 1997. Early management of dental pain in children and adolescents. *Journal of Dentistry* 25:31–4.
- Massie RJ, Olsen M, Glazner J, Robertson CF & Francis I 2000. Newborn screening for cystic fibrosis in Victoria: 10 years' experience (1989–1998). *Medical Journal of Australia* 172:584–7.
- Mathers CD, Bernard C, Iburg KM, Inoue M, Ma Fat D, Shibuya K, Stein C, Tomijima N, Xu H 2004. Global Burden of Disease in 2002: data sources, methods and results. Global Programme on Evidence for Health Policy Discussion Paper no. 54. Geneva: World Health Organization.
- Mathers CD, Iburg KM & Begg S 2006. Adjusting for dependent comorbidity in the calculation of healthy life expectancy. *Population Health Metrics* 4:4.
- Mathers CD, Vos T, Lopez AD, Salomon J & Ezzati M 2001. National Burden of Disease Studies: a practical guide. Edition 2.0. Global Program on Evidence for Health Policy. Geneva: World Health Organization.
- McCusker EA, Casse RF, Graham SJ, Williams DB & Lazarus R 2000. Prevalence of Huntington disease in New South Wales in 1996. *Medical Journal of Australia* 173:187–90.
- McDonald SP & Russ GR (eds) 2002. ANZDATA Registry 2002 report. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.
- McGilchrist CA & Hills LJ 1986. Estimation of cumulative illness using cross-sectional data. *Journal of Chronic Diseases* 39:929–31.
- McIntyre N 1990. Clinical presentation of acute viral hepatitis. *British Medical Bulletin* 46:533–47.
- McKetin R, McLaren J, Kelly E, Hall W & Hickman M 2005. Estimating the number of regular and dependent methamphetamine users in Australia. Technical Report No. 230. Sydney: National Drug & Alcohol Research Centre (NDARC).
- McLeod JG, Hammond SR & Hallpike JF 1994. Epidemiology of multiple sclerosis in Australia. With NSW and SA survey results. *Medical Journal of Australia* 160:117–22.
- Melnick JL 1995. History and epidemiology of hepatitis A virus. *Journal of Infectious Diseases* 171(Suppl 1):S2–8.
- Menzies R, McIntyre P & Beard F 2004. Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia, 1999 to 2002. *Communicable Diseases Intelligence* 28(Suppl 1):S1–45.

- Messman-Moore TL, Long PJ & Siegfried NJ 2000. The revictimization of child sexual abuse survivors: an examination of the adjustment of college women with child sexual abuse, adult sexual assault, and adult physical abuse. *Child Maltreatment* 5:18-27.
- Miettinen OS 1974. Proportion of disease caused or prevented by a given exposure, trait or intervention. *American Journal of Epidemiology* 99:325-32.
- Mikuls TR, Farrar JT, Bilker WB, Fernandes S, Schumacher HR, Jr & Saag KG 2005. Gout epidemiology: results from the UK General Practice Research Database, 1990-1999. *Annals of the Rheumatic Diseases* 64:267-2.
- Miller DH, Hornabrook RW & Purdie G 1992. The natural history of multiple sclerosis: a regional study with some longitudinal data. *Journal of Neurology, Neurosurgery and Psychiatry* 55:341-6.
- Mira M, Bawden-Smith J, Causer J, Alperstein G, Karr M, Snitch P et al. 1996. Blood lead concentrations of preschool children in central and southern Sydney. *Medical Journal of Australia* 164:399-402.
- Mitchell P, Chapman S & Smith W 1999. Smoking is a major cause of blindness. *Medical Journal of Australia* 171:173-4.
- Mitchell P, Smith W, Chey T & Healey PR 1997. Open-angle glaucoma and diabetes: the Blue Mountains Eye Study, Australia. *Ophthalmology* 104:712-18.
- Mitchell PB, Slade T & Andrews G 2004. Twelve-month prevalence and disability of DSM-IV bipolar disorder in an Australian general population survey. *Psychological Medicine* 34:777-85.
- Miyamura H, Eguchi S & Asano K 1993. Long-term results of the intracardiac repair of tetralogy of Fallot: a follow-up study conducted over more than 20 years on 100 consecutive operative survivors. *Surgery Today* 23:1049-52.
- Mocroft A, Youle M, Morcinek J, Sabin CA, Gazzard B, Johnson MA et al. 1997. Survival after diagnosis of AIDS: a prospective observational study of 2625 patients. Royal Free/Chelsea and Westminster Hospitals Collaborative Group. *British Medical Journal (Clinical Research Edition)* 314:409-13.
- Mollison LC, Jamrozik KD & Plant AJ 1999. Australian doctors' beliefs and practice regarding *Helicobacter pylori*. *Medical Journal of Australia* 170:354-7.
- Morgante L, Salemi G, Meneghini F, Di Rosa AE, Epifanio A, Grigoletto F et al. 2000. Parkinson disease survival: a population-based study. *Archives of Neurology* 57:507-12.
- Mouzos J 2005. Homicide in Australia: 2003-2004 National Homicide Monitoring Program (NHMP) annual report. Research and public policy series no. 66. Canberra: Australian Institute of Criminology.
- Mouzos J & Makkai T 2004. Women's experiences of male violence: findings from the Australian component of the International Violence Against Women Survey (IVAWS). Research and public policy series no. 56. Canberra: Australian Institute of Criminology.
- Mowat D, Partington M, Turner G, Tonge B, Einfeld S, Gray K et al. In: The Australian Child and Adolescent Developmental (ACAD) study: genetic assessment of an epidemiological sample of young people with mental retardation. Sydney: Department of Medical Genetics, Sydney Children's Hospital. Unpublished.

- Munoz N, Bosch FX, de Sanjose S, Herrero R, Castellsague X, Shah KV et al. 2003. Epidemiologic classification of human papilloma virus types associated with cervical cancer. *New England Journal of Medicine* 348:518–27.
- Murray CJ, Ezzati M, Lopez AD, Rodgers A & Vander Hoorn S 2003. Comparative quantification of health risks: conceptual framework and methodological issues. *Population Health Metrics* 1:1.
- Murray CJL, Salomon JA, Mathers CD & Lopez AD 2002. *Summary Measures of Population Health: Concepts, Ethics, Measurement and Applications*. Geneva: World Health Organization.
- Murray CJ, Gakidou EE & Frenk J 1999a. Health inequalities and social group differences: what should we measure? *Bulletin of the World Health Organization* 77:537–43.
- Murray CJ & Lopez AD 1999. On the comparable quantification of health risks: lessons from the Global Burden of Disease Study. *Epidemiology* 10:594–605.
- Murray CJ, Salomon J & Mathers C 1999b. A critical examination of summary measures of population health. GPE discussion paper no. 2. Geneva: World Health Organization.
- Murray CJL & Lopez AD (eds) 1996a. *The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020*. Vol I. Cambridge, MA: Harvard School of Public Health on behalf of the World Health Organization & the World Bank.
- Murray CJL & Lopez AD 1996b. *Global health statistics: a compendium of incidence, prevalence and mortality estimates for over 200 conditions*. Cambridge, MA: Harvard School of Public Health on behalf of the World Health Organization & the World Bank.
- Mylonas AD, Brown AM, Carthew TL, McGrath B, Purdie DM, Pandeya N et al. 2002. Natural history of Ross River virus-induced epidemic polyarthritides. *Medical Journal of Australia* 177:356–60.
- National Centre in HIV Epidemiology and Clinical Research 2003. *HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report 2003*. Sydney: National Centre in HIV Epidemiology and Clinical Research.
- National Centre in HIV Epidemiology and Clinical Research 2004. *HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report 2004*. Sydney, Canberra: National Centre in HIV Epidemiology and Clinical Research, AIHW.
- National Centre in HIV Epidemiology and Clinical Research 2005a. *Australian AIDS Public Access Dataset*. Sydney: National Centre in HIV Epidemiology and Clinical Research. Viewed 19 October 2005, <web.med.unsw.edu.au/nchecr/>.
- National Centre in HIV Epidemiology and Clinical Research 2005b. *Australian HIV Public Access Dataset*. Sydney: National Centre in HIV Epidemiology and Clinical Research. Viewed 19 October 2005, <web.med.unsw.edu.au/nchecr/>.
- NCCI (National Cancer Control Initiative) 2003. *The 2002 national non-melanoma skin cancer survey. A report by the NCCI Non-melanoma Skin Cancer Working Group*. Melbourne: NCCI.
- Nelson MR, Liew D, Bertram M & Vos T 2005. Epidemiological modelling of routine use of low dose aspirin for the primary prevention of coronary heart disease and stroke in those aged > or =70. *British Medical Journal (Clinical Research Edition)* 330:1306.

- Nguyen ND, Allen JR, Peat JK, Beal P, Webster BH & Gaskin KJ 2004. Iron status of young Vietnamese children in Australia. *Journal of Paediatrics and Child Health* 40:424-9.
- Nguyen ND, Pongchaiyakul C, Center JR, Eisman JA & Nguyen TV 2005a. Abdominal fat and hip fracture risk in the elderly: the Dubbo Osteoporosis Epidemiology Study. *BMC Musculoskeletal Disorders* 6:11.
- Nguyen ND, Pongchaiyakul C, Center JR, Eisman JA & Nguyen TV 2005b. Identification of high-risk individuals for hip fracture: a 14-year prospective study. *Journal of Bone and Mineral Research* 20:1921-8.
- Nguyen TV, 2005. Dubbo Osteoporosis Epidemiology Study – Age and sex-specific bone mineral density mean and standard deviation. Email, 12 October.
- Nguyen TV, Center JR, Sambrook PN & Eisman JA 2001. Risk factors for proximal humerus, forearm, and wrist fractures in elderly men and women: the Dubbo Osteoporosis Epidemiology Study. *American Journal of Epidemiology* 153:587-95.
- NHMRC (National Health and Medical Research Council) 1992. Is there a safe level of daily consumption of alcohol for men and women? Canberra: NHMRC.
- NHMRC 1997a. The health effects of passive smoking. Canberra: NHMRC.
- NHMRC 1997b. Management of diabetic retinopathy: clinical practice guidelines. Canberra: Commonwealth of Australia.
- Ni Mhurchu C, Parag V, Nakamura M, Patel A, Rodgers A & Lam TH 2006. Body mass index and risk of diabetes mellitus in the Asia-Pacific region. *Asia Pac J Clin Nutr* 15(2): 127-33.
- Nicolaidis C, Curry M, McFarland B & Gerrity M 2004. Violence, mental health, and physical symptoms in an academic internal medicine practice. *Journal of General Internal Medicine* 19:819-27.
- NINDS (National Institute of Neurological Disorders and Stroke) 2006. Low back pain fact sheet. Viewed 22 August 2006, <www.ninds.nih.gov/disorders/backpain/detail_backpain.htm>.
- Nollert G, Fischlein T, Bouterwek S, Bohmer C, Dewald O, Kreuzer E et al. 1997a. Long-term results of total repair of tetralogy of Fallot in adulthood: 35 years follow-up in 104 patients corrected at the age of 18 or older. *The Thoracic and Cardiovascular Surgeon* 45:178-81.
- Nollert G, Fischlein T, Bouterwek S, Bohmer C, Klinner W & Reichart B 1997b. Long-term survival in patients with repair of tetralogy of Fallot: 36-year follow-up of 490 survivors of the first year after surgical repair. *Journal of the American College of Cardiology* 30:1374-83.
- Oguma Y, Sesso HD, Paffenbarger RS, Jr & Lee IM 2002. Physical activity and all cause mortality in women: a review of the evidence. *British Journal of Sports Medicine* 36:162-72.
- O'Sullivan BG, Gidding HF, Law M, Kaldor JM, Gilbert GL & Dore GJ 2004. Estimates of chronic hepatitis B virus infection in Australia, 2000. *Australian and New Zealand Journal of Public Health* 28:212-16.
- Oti-Boateng P, Seshadri R, Petrick S, Gibson RA & Simmer K 1998. Iron status and dietary iron intake of 6-24-month-old children in Adelaide. *Journal of Paediatrics and Child Health*. 34:250-3.
- Papaioannou A, Joseph L, Ioannidis G, Berger C, Anastassiades T, Brown JP et al. 2005. Risk factors associated with incident clinical vertebral and nonvertebral fractures in postmenopausal women: the Canadian Multicentre Osteoporosis Study (CaMos). *Osteoporosis International* 16:568-78.

- Partington M, Mowat D, Einfeld S, Tonge B & Turner G 2000. Genes on the X chromosome are important in undiagnosed mental retardation. *American Journal of Medical Genetics* 92:57-61.
- Patja K, Iivanainen M, Vesala H, Oksanen H & Ruoppila I 2000. Life expectancy of people with intellectual disability: a 35-year follow-up study. *Journal of Intellectual Disability Research* 44(Pt 5): 591-9.
- Peat JK, Gray EJ, Mellis CM, Leeder SR & Woolcock AJ 1994. Differences in airway responsiveness between children and adults living in the same environment: an epidemiological study in two regions of New South Wales. *European Respiratory Journal* 7:1805-13.
- Peat JK, Haby M, Spijker J, Berry G & Woolcock AJ 1992. Prevalence of asthma in adults in Busselton, Western Australia. *British Medical Journal (Clinical Research Edition)* 305:1326-9.
- Peat JK, Toelle BG, Gray EJ, Haby MM, Belousova E, Mellis CM et al. 1995. Prevalence and severity of childhood asthma and allergic sensitisation in seven climatic regions of New South Wales. *Medical Journal of Australia* 163:22-6.
- Peto R, Lopez AD, Boreham J, Thun M & Heath C, Jr 1992. Mortality from tobacco in developed countries: indirect estimation from national vital statistics. *Lancet* 339:1268-78.
- Picavet HS & Hazes JM 2003. Prevalence of self reported musculoskeletal diseases is high. *Annals of the Rheumatic Diseases* 62:644-50.
- Pincus T, Brooks RH & Callahan LF 1994. Prediction of long-term mortality in patients with rheumatoid arthritis according to simple questionnaire and joint count measures. *Annals of Internal Medicine* 120:26-34.
- Plunkett A, Merlin K, Gill D, Zuo Y, Jolley D & Marks R 1999. The frequency of common non-malignant skin conditions in adults in central Victoria, Australia. *International Journal of Dermatology* 38:901-8.
- Pope CA, 3rd, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K et al. 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association* 287:1132-41.
- Porter K, Babiker A, Bhaskaran K, Darbyshire J, Pezzotti P & Walker AS 2003. Determinants of survival following HIV-1 seroconversion after the introduction of HAART. *Lancet* 362:1267-74.
- Powles J & Day N 2002. Interpreting the global burden of disease. *Lancet* 360:1342-3.
- Prevoo ML, van Gestel AM, van T Hof MA, van Rijswijk MH, van de Putte LB & van Riel PL 1996. Remission in a prospective study of patients with rheumatoid arthritis. American Rheumatism Association preliminary remission criteria in relation to the disease activity score. *British Journal of Rheumatology* 35:1101-5.
- Queensland Health 2003. Incidence data – Queensland newborn screening. Brisbane: Health Information Centre. Viewed 22 August 2006, <www.health.qld.gov.au/hic/peri03_final/NSC03.pdf>.
- Queensland Health 2004. Perinatal statistics Queensland 2002: baby details. Brisbane: Health Information Centre. Viewed 22 August 2006, <www.health.qld.gov.au/hic/peri2002/BABY.pdf>.
- Quittan M 2002. Management of back pain. *Disability and Rehabilitation* 24:423-34.

- Rangan AM, Blight GD & Binns CW 1998. Iron status and non-specific symptoms of female students. *Journal of the American College of Nutrition* 17:351-5.
- Ranmuthugala G, Karr M, Mira M, Alperstein G, Causer J & Jones M 1998. Opportunistic sampling from early childhood centres: a substitute for random sampling to determine lead and iron status of pre-school children? *Australian and New Zealand Journal of Public Health* 22:512-14.
- Rasmussen C 1996. Lumbar disc herniation: social and demographic factors determining duration of disease. *European Spine Journal* 5:225-8.
- Reidpath DD, Allotey PA, Kouame A & Cummins RA 2003. Measuring health in a vacuum: examining the disability weight of the DALY. *Health Policy and Planning* 18:351-6.
- Reyes M, Nisenbaum R, Hoaglin DC, Unger ER, Emmons C, Randall B et al. 2003. Prevalence and incidence of chronic fatigue syndrome in Wichita, Kansas. *Archives of Internal Medicine* 163:1530-6.
- Riley M & Halliday J 2004. Birth defects in Victoria 2001-2002. Melbourne: Victorian Perinatal Data Collection Unit, Victorian Government Department of Human Services.
- Riley M & King J 2003. Births in Victoria 2001-2002. Melbourne: Victorian Perinatal Data Collection Unit, Victorian Government Department of Human Services.
- Robertson CF, Roberts MF & Kappers JH 2004. Asthma prevalence in Melbourne schoolchildren: have we reached the peak? *Medical Journal of Australia* 180:273-6.
- Rosen B, Olavi G, Birkhed D, Edvardsson S & Egelberg J 2004. Effect of different frequencies of preventive maintenance treatment on dental caries: five-year observations in general dentistry patients. *Acta Odontologica Scandinavica* 62:282-8.
- Rosengren A, Stegmayr B, Johansson I, Huhtasaari F & Wilhelmsen L 1999. Coronary risk factors, diet and vitamins as possible explanatory factors of the Swedish north-south gradient in coronary disease: a comparison between two MONICA centres. *Journal of Internal Medicine* 246:577-86.
- Roxburgh RH, Seaman SR, Masterman T, Hensiek AE, Sawcer SJ, Vukusic S et al. 2005. Multiple Sclerosis Severity Score: using disability and disease duration to rate disease severity. *Neurology* 64:1144-51.
- Royal Australian College of Ophthalmologists 1980. National Trachoma and Eye Health Program. Sydney: Royal Australian College of Ophthalmologists.
- Russell RC 2002. Ross River virus: ecology and distribution. *Annual Review of Entomology* 47:1-31.
- Russell RC & Doggett SL 1998. Dengue. Sydney: Department of Medical Entomology, University of Sydney. Viewed 17 August 2006, <medent.usyd.edu.au/fact/dengue.htm>.
- Russell RC & Dwyer DE 2000. Arboviruses associated with human disease in Australia. *Microbes and Infection* 2:1693-704.
- Sach L 1995. People living with motor neurone disease in Victoria: profiles and strategies with application to other neurological conditions. Melbourne: Motor Neurone Disease Association of Victoria.
- Sadler S 1996. Iron deficiency in eight-month-old babies. *Professional Care of Mother and Child* 6:65, 8-9.

- Sanders AE, Slade GD, Carter KD & Stewart JF 2004. Trends in prevalence of complete tooth loss among Australians, 1979–2002. *Australian and New Zealand Journal of Public Health* 28:549–54.
- Sanders KM, Pasco JA, Ugoni AM, Nicholson GC, Seeman E, Martin TJ et al. 1998. The exclusion of high trauma fractures may underestimate the prevalence of bone fragility fractures in the community: the Geelong Osteoporosis Study. *Journal of Bone and Mineral Research* 13:1337–42.
- Sawyer MM, Arney FM, Baghurst PA, Clark JJ, Graetz BW, Kosky RJ et al. 2000. The mental health of young people in Australia. Canberra: Mental Health and Special Programs Branch, Commonwealth Department of Health and Aged Care.
- Schott AM, Hans D, Duboeuf F, Dargent-Molina P, Hajri T, Breart G et al. 2005. Quantitative ultrasound parameters as well as bone mineral density are better predictors of trochanteric than cervical hip fractures in elderly women. Results from the EPIDOS study. *Bone* 37:858–63.
- Schuit SC, van der Klift M, Weel AE, de Laet CE, Burger H, Seeman E et al. 2004. Fracture incidence and association with bone mineral density in elderly men and women: the Rotterdam Study. *Bone* 34:195–202.
- Schwartz J 2001. Is there harvesting in the association of airborne particles with daily deaths and hospital admissions? *Epidemiology* 12:55–61.
- Semple DM, McIntosh AM & Lawrie SM 2005. Cannabis as a risk factor for psychosis: systematic review. *Journal of Psychopharmacology* 19:187–94.
- Sesin CA & Bingham CO, 3rd 2005. Remission in rheumatoid arthritis: wishful thinking or clinical reality? *Seminars in Arthritis and Rheumatism* 35:185–96.
- Sesso HD, Paffenbarger RS, Jr & Lee IM 2000. Physical activity and coronary heart disease in men: The Harvard Alumni Health Study. *Circulation* 102:975–80.
- Sharp M, Bulsara M, Gollow I & Pemberton P 2000. Gastroschisis: early enteral feeds may improve outcome. *Journal of Paediatrics and Child Health* 36:472–6.
- Shavelle RM, Strauss DJ & Pickett J 2001. Causes of death in autism. *Journal of Autism and Developmental Disorders* 31:569–76.
- Shivananda S, Lennard-Jones J, Logan R, Fear N, Price A, Carpenter L et al. 1996. Incidence of inflammatory bowel disease across Europe: is there a difference between north and south? Results of the European Collaborative Study on Inflammatory Bowel Disease (EC-IBD). *Gut* 39:690–7.
- Simmons RD, Hall CA, Gleeson P, Everard G, Casse RF & O'Brien ED 2001. Prevalence survey of multiple sclerosis in the Australian Capital Territory. *Internal Medicine Journal* 31:161–7.
- Simoca I, Olarescu AA, Jipescu I & Lisievici M 1994. Postoperative outcome of intracranial meningiomas; long-term prognosis. *Romanian Journal of Neurology and Psychiatry* 32:237–51.
- Simpson R, Williams G, Petroschevsky A, Best T, Morgan G, Denison L et al. 2005a. The short-term effects of air pollution on hospital admissions in four Australian cities. *Australian and New Zealand Journal of Public Health* 29:213–21.
- Simpson R, Williams G, Petroschevsky A, Best T, Morgan G, Denison L et al. 2005b. The short-term effects of air pollution on daily mortality in four Australian cities. *Australian and New Zealand Journal of Public Health* 29:205–12.

Solomon T & Mallewa M 2001. Dengue and other emerging flaviviruses. *Journal of Infection* 42:104–15.

South Australian Cancer Registry 2000. *Epidemiology of cancer in South Australia: incidence, mortality and survival 1977–1999*. Adelaide: South Australian Cancer Registry, Epidemiology Branch.

Spector TD, Nandra D, Hart DJ & Doyle DV 1997. Is hormone replacement therapy protective for hand and knee osteoarthritis in women?: The Chingford Study. *Annals of the Rheumatic Diseases* 56:432–4.

Stanley F, Blair E, Rice G, Stone P, Robinson J, Henderson-Smart D et al. 1995. The origins of cerebral palsy – a consensus statement: The Australian and New Zealand Perinatal Societies. *Australian College of Midwives Incorporated Journal* 8:19–25.

Steiner HH, Herold-Mende C, Bonsanto M, Geletneky K & Kunze S 1998. Zur prognose von hirntumoren: epidemiologie, uberlebenszeit und klinischer verlauf [Prognosis of brain tumors: epidemiology, survival time and clinical course]. *Versicherungsmedizin* 50:173–9.

Steinhausen HC, Winkler C & Meier M 1997. Eating disorders in adolescence in a Swiss epidemiological study. *International Journal of Eating Disorders* 22:147–51.

Steketee G, Eisen J, Dyck I, Warshaw M & Rasmussen S 1999. Predictors of course in obsessive-compulsive disorder. *Psychiatry Research* 89:229–38.

Stewart WF, Linet MS, Celentano DD, Van Natta M & Ziegler D 1991. Age- and sex-specific incidence rates of migraine with and without visual aura. *American Journal of Epidemiology* 134:1111–20.

Stobel-Richter Y, Beutel ME, Finck C & Braehler E 2005. The ‘wish to have a child’, childlessness and infertility in Germany. *Human Reproduction* 20:2850–7.

Stone CA, Carter RC, Vos T & John JS 2004. Colorectal cancer screening in Australia: an economic evaluation of a potential biennial screening program using faecal occult blood tests. *Australian and New Zealand Journal of Public Health* 28:273–82.

Stone KL, Seeley DG, Lui LY, Cauley JA, Ensrud K, Browner WS et al. 2003. BMD at multiple sites and risk of fracture of multiple types: long-term results from the Study of Osteoporotic Fractures. *Journal of Bone and Mineral Research* 18:1947–54.

Stonnington CM, Phillips SF, Melton LJ, 3rd & Zinsmeister AR 1987. Chronic ulcerative colitis: incidence and prevalence in a community. *Gut* 28:402–9.

Stouthard ME, Essink-Bot M, Bonsel GJ, Barendregt JJ, Kramers PGN, van de Water HPA et al. 1997. Disability weights for diseases in The Netherlands. Rotterdam: Department of Health, Erasmus University Rotterdam.

Street AM & Ekert H 1996. Haemophilia – darkest hours before the dawn. *Medical Journal of Australia* 164:453–5.

Strober M, Freeman R & Morrell W 1997. The long-term course of severe anorexia nervosa in adolescents: survival analysis of recovery, relapse, and outcome predictors over 10–15 years in a prospective study. *International Journal of Eating Disorders* 22:339–60.

Sullivan PF 1995. Mortality in anorexia nervosa. *American Journal of Psychiatry* 152:1073–4.

Swift W, Hall W & Copeland J 2000. One year follow-up of cannabis dependence among long-term users in Sydney, Australia. *Drug and Alcohol Dependence* 59:309–18.

- Tapp RJ, Shaw JE, de Courten MP, Dunstan DW, Welborn TA, Zimmet PZ et al. 2003a. Foot complications in Type 2 diabetes: an Australian population-based study. *Diabetic Medicine* 20:105-13.
- Tapp RJ, Shaw JE, Harper CA, de Courten MP, Balkau B, McCarty DJ et al. 2003b. The prevalence of and factors associated with diabetic retinopathy in the Australian population. *Diabetes Care* 26:1731-7.
- Tate RB, Manfreda J & Cuddy TE 1998. The effect of age on risk factors for ischaemic heart disease: the Manitoba Follow-Up Study, 1948-1993. *Annals of Epidemiology* 8:415-21.
- Taylor R, Dobson A & Mirzaei M 2006. Contribution of changes in risk factors to the decline of coronary heart disease mortality in Australia over three decades. *European Journal of Cardiovascular Prevention and Rehabilitation*. 13:760-8
- Thestrup-Pedersen K 2003. Atopic eczema. What has caused the epidemic in industrialised countries and can early intervention modify the natural history of atopic eczema? *Journal of Cosmetic Dermatology* 2:202-10.
- Thrift AG, Dewey HM, Macdonell RA, McNeil JJ & Donnan GA 2000. Stroke incidence on the east coast of Australia: the North East Melbourne Stroke Incidence Study (NEMESIS). *Stroke* 31:2087-92.
- Thune I & Furberg AS 2001. Physical activity and cancer risk: dose-response and cancer, all sites and site-specific. *Medicine and Science in Sports and Exercise* 33:S530-50; discussion S609-10.
- Toelle BG, Ng K, Belousova E, Salome CM, Peat JK & Marks GB 2004. Prevalence of asthma and allergy in schoolchildren in Belmont, Australia: three cross sectional surveys over 20 years. *British Medical Journal (Clinical Research Edition)* 328:386-7.
- Toelle BG, Peat JK, Salome CM, Mellis CM & Woolcock AJ 1992. Toward a definition of asthma for epidemiology. *American Review of Respiratory Disease* 146:633-7.
- Tomany SC, Wang JJ, Van Leeuwen R, Klein R, Mitchell P, Vingerling JR et al. 2004. Risk factors for incident age-related macular degeneration: pooled findings from 3 continents. *Ophthalmology* 111:1280-7.
- Tomson T 2000. Mortality in epilepsy. *Journal of Neurology* 247:15-21.
- Torvaldsen S, Hull BP & McIntyre PB 2002. Using the Australian Childhood Immunisation Register to track the transition from whole-cell to acellular pertussis vaccines. *Communicable Diseases Intelligence* 26:581-3.
- Tremlett H, Paty D & Devonshire V 2006. Disability progression in multiple sclerosis is slower than previously reported. *Neurology* 66:172-7.
- Unal B, Critchley JA & Capewell S 2004. Explaining the decline in coronary heart disease mortality in England and Wales between 1981 and 2000. *Circulation* 109:1101-7.
- US Department of Health and Human Services 2006. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. Atlanta, GA: US Department of Health and Human Services & Centers for Disease Control and Prevention (CDC).
- US Preventive Services Task Force 1996. Guide to clinical preventive services: report of the US Preventive Services Taskforce, second edition. Baltimore: Williams & Williams.
- Van Asperen PP 1995. Towards a better understanding of childhood asthma. *Journal of Paediatrics and Child Health* 31:272-5.

- Verdecchia A, De Angelis R, Capocaccia R, Sant M, Micheli A, Gatta G et al. 1998. The cure for colon cancer: results from the EURO CARE study. *International Journal of Cancer* 77:322-9.
- Von Korff M & Saunders K 1996. The course of back pain in primary care. *Spine* 21:2833-7.
- Vos T, Astbury J, Piers LS, Magnus A, Heenan M, Stanley L et al. 2006. Measuring the impact of intimate partner violence on the health of women in Victoria, Australia. *Bulletin of the World Health Organization*, 84:739-44.
- Vos T, Haby MM, Magnus A, Mihalopoulos C, Andrews G & Carter R 2005. Assessing cost-effectiveness in mental health: helping policy-makers prioritize and plan health services. *Australian and New Zealand Journal of Psychiatry* 39:701-12.
- Vos T, Goss J, Begg S & Mann N 2007. Projection of health care expenditure by disease: a case study from Australia. Brisbane: School of Population Health, University of Queensland.
- Walboomers JM, Jacobs MV, Manos MM, Bosch FX, Kummer JA, Shah KV et al. 1999. Human papilloma virus is a necessary cause of invasive cervical cancer worldwide. *Journal of Pathology* 189:12-9.
- Walter SD 1980. Prevention for multifactorial diseases. *American Journal of Epidemiology* 112:409-16.
- Webb S & Holman D 1992. A survey of infertility, surgical sterility and associated reproductive disability in Perth, Western Australia. *Australian Journal of Public Health* 16:376-81.
- Weih LM, VanNewkirk MR, McCarty CA & Taylor HR 2000. Age-specific causes of bilateral visual impairment. *Archives of Ophthalmology* 118:264-9.
- Weiss TJ, Meffin EM, Jones RG & Jones WR 1992. Trends in causes and treatment of infertility at Flinders Medical Centre, Adelaide, 1976-1989. *Medical Journal of Australia* 156:308-11.
- Wellesley DG, Hockey KA, Montgomery PD & Stanley FJ 1992. Prevalence of intellectual handicap in Western Australia: a community study. *Medical Journal of Australia* 156:94-6, 100, 2.
- Wewetzer C, Jans T, Muller B, Neudorfl A, Bucherl U, Remschmidt H et al. 2001. Long-term outcome and prognosis of obsessive-compulsive disorder with onset in childhood or adolescence. *European Child and Adolescent Psychiatry* 10:37-46.
- WHO (World Health Organization) 1992. *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision*. Geneva: WHO.
- WHO 2002. *World health report 2002 – reducing risks, promoting healthy life*. Geneva: WHO.
- WHO Europe 2004. *Health aspects of air pollution results from the WHO project 'Systematic review of health aspects of air pollution in Europe'*. Copenhagen: WHO Europe.
- Whyman RA, Treasure ET & Ayers KM 1996. Dental disease levels and reasons for emergency clinic attendance in patients seeking relief of pain in Auckland. *New Zealand Dental Journal* 92:114-17.
- Williams A 1999. Calculating the global burden of disease: time for a strategic reappraisal? *Health Economics* 8:1-8.
- Williams PT 2001. Physical fitness and activity as separate heart disease risk factors: a meta-analysis. *Medicine and Science in Sports and Exercise* 33:754-61.

- Willner IR, Uhl MD, Howard SC, Williams EQ, Riely CA & Waters B 1998. Serious hepatitis A: an analysis of patients hospitalized during an urban epidemic in the United States. *Annals of Internal Medicine* 128:111-4.
- Wilson A, Hickie I, Hadzi-Pavlovic D, Wakefield D, Parker G, Straus SE et al. 2001. What is chronic fatigue syndrome? Heterogeneity within an international multicentre study. *Australian and New Zealand Journal of Psychiatry* 35:520-7.
- Wilson D, Sanchez L & Read P 1998. Hearing impairment in an Australian population. Adelaide: Centre for Population Studies in Epidemiology, Department of Human Services.
- Wilson DH, Walsh PG, Sanchez L, Davis AC, Taylor AW, Tucker G et al. 1999. The epidemiology of hearing impairment in an Australian adult population. *International Journal of Epidemiology* 28:247-52.
- Woodward M, Zhang X, Barzi F, Pan W, Ueshima H, Rodgers A et al. 2003. The effects of diabetes on the risks of major cardiovascular disease and death in the Asia-Pacific region. *Diabetes Care* 26:360-6.
- World Bank 1993. *World development report: investing in health*. New York: Oxford University Press.
- Xuan W, Marks GB, Toelle BG, Belousova E, Peat JK, Berry G et al. 2002. Risk factors for onset and remission of atopy, wheeze, and airway hyperresponsiveness. *Thorax* 57:104-9.
- Yonkers KA, Bruce SE, Dyck IR & Keller MB 2003. Chronicity, relapse, and illness – course of panic disorder, social phobia, and generalized anxiety disorder: findings in men and women from 8 years of follow-up. *Depression and Anxiety* 17:173-9.
- Yue D & Molyneaux L 2005. Diabetes Research Foundation, Royal Prince Alfred Hospital. Email, June.
- Zanarini MC, Frankenburg FR, Hennen J & Silk KR 2003. The longitudinal course of borderline psychopathology: 6-year prospective follow-up of the phenomenology of borderline personality disorder. *American Journal of Psychiatry* 160:274-83.
- Zarrelli MM, Beghi E, Rocca WA & Hauser WA 1999. Incidence of epileptic syndromes in Rochester, Minnesota: 1980-1984. *Epilepsia* 40:1708-14.
- Zeger SL, Dominici F & Samet J 1999. Harvesting-resistant estimates of air pollution effects on mortality. *Epidemiology* 10:171-5.
- Zhao Y, Guthridge S, Magnus A & Vos T 2004. Burden of disease and injury in Aboriginal and non-Aboriginal populations in the Northern Territory. *Medical Journal of Australia* 180:498-502.

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