THE BURDEN OF DISEASE AND INJURY IN ABORIGINAL AND TORRES STRAIT ISLANDER PEOPLES

2003

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The burden of disease and injury in Aboriginal and Torres Strait Islander peoples 2003

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Foreword

Policies and programs to improve health rely heavily on valid and timely information about the major causes of disease and injury in a population, and how these are changing. Typically, government policy is informed by a variety of data sources about the state of population health, many of them fragmentary and of uncertain quality, and none of them completely capturing the spectrum of diseases and injuries of interest, or indeed the consequences of disease and injury in terms of premature mortality and disability. More than a decade ago, a single summary measure of population health, the Disability-Adjusted Life Year, or DALY, was developed and has been widely applied since to measure disease burden by age, sex, cause and risk factor in numerous populations across the globe, as well as for the world and its major regions. DALYs are now the accepted unit for health accounting worldwide, and the Burden of Disease framework has become the global standard for integrating, adjusting and using available health information to produce policy-relevant and comparable evidence about a population's health.

While there have been numerous burden of disease studies over the past decade in developed countries, including several in Australia, the framework and tools have never before been applied to measure disease burden in Indigenous populations apart from a pilot study in the Northern Territory, although the need for such evidence to guide policies and programs is clear. Given the uncertain quality of many data sources on Indigenous health, and the lack of comprehensive information about the comparative importance of various diseases and injuries, it has been difficult to appreciate the complete set of priorities for Indigenous health development. This report responds to that need by providing the first ever burden of disease and injury estimates for the Aboriginal and Torres Strait Islander population of Australia by leading experts in the field. Detailed estimates are provided of the comparative importance of over 170 diseases and injuries for the health of Indigenous Australians, and in doing so, this report fills an important gap in the evidence base for Indigenous Australian health policy.

By highlighting the main causes of disease burden – and there are many – the report provides clear guidance for intervention strategies, particularly to reduce the unacceptably high risks of death in young adulthood that are still prevalent today. For example, on current rates, one-third of young indigenous men aged 15 will be dead before age 60, compared to 8% in the Australian population. This four-fold increase in risk of death, comparable to parts of Africa today, is largely due to excess mortality from such causes as ischaemic heart disease, suicide and Type 2 diabetes, and its reduction must be a priority for Indigenous health services.

With the publication of this report, a critical gap in the information base for health development for Indigenous Australians has been filled. I strongly urge all health jurisdictions throughout Australia to consider its findings carefully in developing policies for better health of Indigenous Australians.

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Abbreviations and acronyms

ABS	Australian Bureau of Statistics
ACE	Assessing Cost-Effectiveness
A&E	Accident and emergency
ADHD	Attention-deficit hyperactivity disorder
AIDS	Acquired immunodeficiency syndrome
AIHW	Australian Institute of Health and Welfare
ARIA+	Accessibility/Remoteness Index of Australia
ASGC	Australian Standard Geographical Classification
BMI	Body mass index
COPD	Chronic obstructive pulmonary disease
DALY	Disability-adjusted life year
EERP	Experimental estimated resident population
GGB	General growth balance
HALE	Health-adjusted life expectancy
HIV	Human immunodeficiency virus
ICD	International Classification of Disease
IHD	Ischaemic heart disease
IMR	Infant mortality rate
NACCHO	National Aboriginal Community Controlled Health Organisation
NHMRC	National Health and Medical Research Council
OATSIH	Office for Aboriginal and Torres Strait Islander Health
PYLD	Prevalent years lived with disability
RR	Rate ratio
RTA	Road traffic accident
SLA	Statistical local area
STD	Sexually transmitted disease
YLD	Years lived with disability
YLL	Years of life lost

Executive summary

This study is the first complete assessment of the burden of disease and injury for Aboriginal and Torres Strait Islander peoples. It identifies the extent and causes of Indigenous health problems and quantifies 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' in disability-adjusted life years (DALYs). The DALY is a health gap measure that compares the current health status of a population against an 'ideal' in which everyone lives into old age free from disease. Similar estimates for the total Australian population for the same baseline year, 2003, have been published separately in *The Burden of Disease and Injury in Australia, 2003* report (Begg et al. 2007). Results from both studies are quantified in the same measure with comparable methods.

The health problems facing Indigenous Australians are best illustrated as the 'Indigenous health gap', which we calculated as the difference between the burden of disease estimates for Indigenous Australians in 2003 and what these estimates would have been if Indigenous Australians had experienced mortality and disability at the level of the total Australian population. The Indigenous health gap reflects the potential for health gain; in other words, it answers the question 'For which health problem, addressing which age group, in males or females, is a concerted effort most likely to lead to an improvement in the health status of the Indigenous population?' This makes the Indigenous health gap the most useful finding for health policy makers.

The assessment of the health status of Indigenous Australians is not easy. The main challenge is the inaccurate and incomplete identification of who is, and who is not, Indigenous in population health and census data collections. For instance, in order to estimate more accurate rates of mortality for Indigenous Australians we had to use an indirect demographic method which in turn introduces uncertainty. Estimating the cause of death was deemed more accurate because the quality of certification of cause of death for Indigenous people was similar to that for the rest of the population when comparing the proportions of deaths coded to ill-defined codes. We therefore assumed that the cause of death pattern in deaths recorded as Indigenous reflected the pattern of all Indigenous deaths.

Estimates of disability for more than 170 disease and injury categories that were included in this study depended on a combination of methods. If available, data on directly observed Indigenous health events in routine health statistics databases, health surveys or epidemiological studies were included. For many diseases, such information did not exist. Instead, ratios of the differences between Indigenous and total population rates were sought for proxy measures of disease occurrence, such as hospital admissions or mortality records. Consequently, the accuracy of the estimates included in this study vary. The study provides transparency on the data sources consulted, and all the assumptions and judgments on which the results depend.

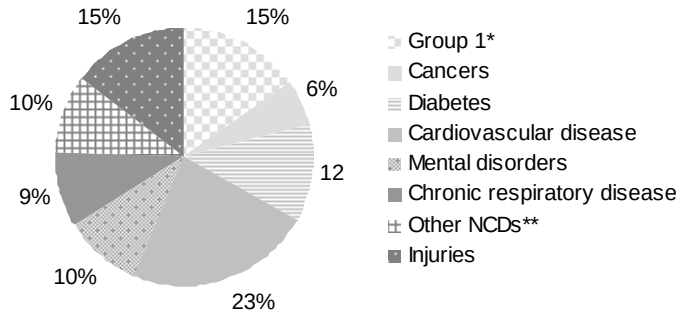
Key findings

The Indigenous Australian population is younger than the total Australian population, therefore where overall rates are presented and comparisons are made with rates in the total

Australian population these are age-standardised to the 2003 Indigenous Australian population.

Indigenous health gap

- If Indigenous Australians had the same level of mortality and disability as the total Australian population, the total burden of disease would have been 59% lower (39,522 compared with 95,976 DALYs); this indicates a very large potential for health gain.
- Non-communicable diseases explained 70% of the health gap, with cardiovascular disease the leading cause group (23%) followed by diabetes (12%), mental disorders (12%) and chronic respiratory diseases (9%) (Figure i).
- Ischaemic heart disease (14%), Type 2 diabetes (12%) and substance use disorders (6%) were the main non-communicable disease categories that contributed to the health gap.
- Injuries and group I conditions (communicable disease, maternal and neonatal conditions) were each responsible for 15% of the gap.
- Communicable diseases (9%) and neonatal conditions (4%) explained almost all of the gap for group I conditions.
- Suicide (4%), road traffic accidents (RTAs) (3%), and homicide & violence (3%) were the major causes responsible for the injury gap.

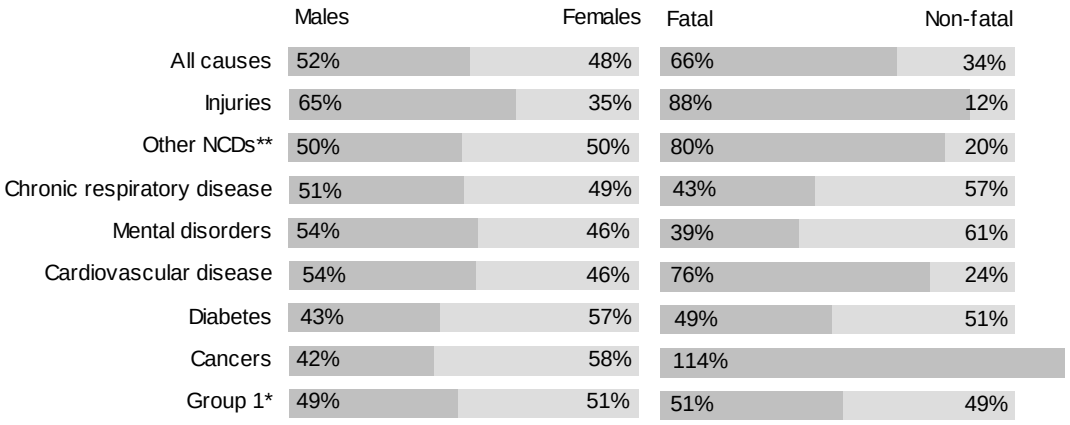


* Communicable diseases, maternal and neonatal conditions
** Other non-communicable diseases

Figure i: Indigenous health gap (DALYs) by selected causes, 2003

- The overall Indigenous health gap was similar in males and females; the gap for injuries was much greater in males while that for diabetes and cancers was greater in females (Figure ii).
- Compared to the total burden of disease estimates for Indigenous Australians, where 54% of burden was due to mortality, two-thirds of the Indigenous health gap was due to mortality (Figure iii). This means that the mortality gap was considerably greater than the disability gap, and reflects in part a higher case fatality: when sick, Indigenous Australians are more likely to die.

- Mortality dominated the gap for cancer, injuries and cardiovascular disease; cancer was the only disease category that had a lower rate of disability (by 14%) than the total population (hence the 114% for fatal cancer in Figure ii).

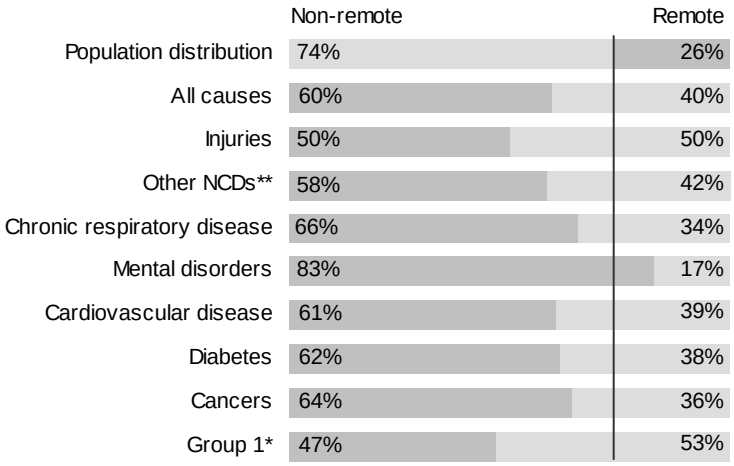


* Communicable diseases, maternal and neonatal conditions
** Other non-communicable diseases

Figure ii: Indigenous health gap (DALYs) by selected causes expressed as proportions by sex, and proportions due to fatal and non-fatal outcomes, 2003

- The largest proportion of Indigenous health gap occurred in the age group of 35–54 years (35%) followed by the 15–34-year age group (25%), the 55-year and older age group (23%) and then children under 15 years (17%).
- Cardiovascular disease, particularly ischaemic heart disease and diabetes, were the main contributors to the health gap at ages 35 years and over.
- Injuries and mental disorders were the main contributors to the health gap in young adults aged 15–34 years; notably at these young ages, cardiovascular disease and diabetes were already responsible for one-fifth of the health gap.
- Suicide explained almost half of the health gap from injuries in young males. In young females, injuries contributed a lesser proportion to the gap but there was still considerable excess health loss from RTAs, suicide and violence.
- Substance use disorders explained most of the gap from mental disorders in young adults.
- Indigenous Australians residing in remote areas represented 26% of the total Indigenous Australian population but contributed 35% of the overall Indigenous disease and injury burden and experienced 40% of the Indigenous health gap (Figure iii). Thus, despite the higher disease rates experienced by Indigenous Australians in remote areas, the majority of burden still occurred in non-remote areas. This indicates that policies need to address the health problems of Indigenous Australians in both non-remote and remote areas.
- Relative to population size Indigenous Australians residing in remote areas experienced a disproportionate amount of the health gap for all major disease areas apart from mental disorders (Figure iii). This latter finding may be an artefact of the methods. For example, the disease models for alcohol dependence & harmful use, and anxiety & depression were derived from the 2004-05 National Aboriginal and Torres Strait Islander Health

Survey Social and Emotional Well-being module which gave proxies rather than diagnostic indicators for ICD-10 defined mental disorders.



* Communicable diseases, maternal and neonatal conditions
** Other non-communicable diseases

Figure iii: Indigenous health gap (DALYs) by selected causes expressed as proportions by remoteness, 2003

Disease and injury burden in DALYs

- The disease burden occurred at a considerably higher rate at each age for Indigenous Australians compared with the total Australian population. In 2003, the Indigenous Australian population made up 2.4% of the total Australian population; however, despite its much younger age structure, the Indigenous Australian population carried 3.6% of the total disease burden.
- Cardiovascular disease and mental disorders were the leading causes of disease burden in the Indigenous Australian population in 2003 (Figure iv). These two broad cause groups together accounted for 32% of the disease burden. Chronic respiratory disease, diabetes mellitus and cancers were the next three leading causes, accounting for an approximately equal proportion of the total Indigenous Australian disease burden at 8% each.
- Cancer was responsible for a much greater proportion of the burden in the total Australian population (19%) than the Indigenous population (8%). Diabetes, and unintentional and intentional injuries were each responsible for a larger proportion of the total burden in Indigenous Australians than in the total Australian population (Figure iv).

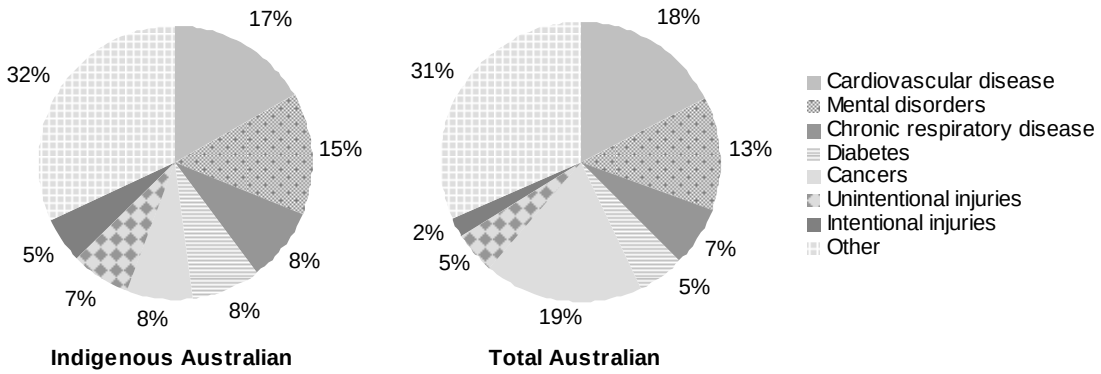
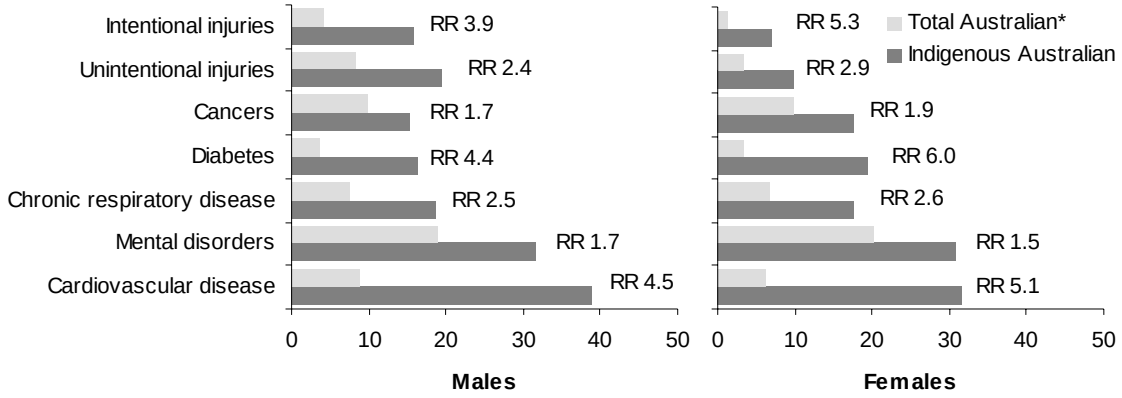


Figure iv: DALYs by broad cause group, Indigenous and total Australian population, 2003

- Among the top seven broad cause categories, the largest differentials in rate of burden between the Indigenous and total Australian population were seen in cardiovascular disease, diabetes mellitus and intentional injuries for both males and females (Figure v).



*Total Australian rates age-standardised to the Indigenous population
RR Indigenous Australian to total Australian rate ratio

Figure v: DALY rate per 1,000 for the leading broad cause groups by sex, Indigenous and total Australian population, 2003

- Ischaemic heart disease was the largest single contributor to the disease burden experienced by Indigenous Australian males, accounting for 12% of the total burden. Type 2 diabetes, anxiety & depression, suicide, and RTAs were the next four leading causes of male burden, together accounting for 22% of the male burden.
- For females, the leading cause was anxiety & depression, causing 10% of the total burden. Type 2 diabetes, ischaemic heart disease, asthma and chronic obstructive pulmonary disease (COPD) were the following four leading causes, accounting for 26% of the total burden for females.
- Among the 20 conditions that caused the greatest burden, the largest differentials in burden rate between the Indigenous and total Australian male population were for homicide & violence (rate ratio, RR 6.8), inflammatory heart disease (RR 6.3), and lower respiratory tract infection (RR 6.1) .For females, the largest differentials were for

rheumatic heart disease (RR 26.4), homicide & violence (RR 11.0), and alcohol dependence & harmful use (RR 7.9).

Health risks

- The 11 risk factors considered (tobacco, alcohol, illicit drugs, high body mass, inadequate physical activity, low intake of fruit and vegetables, high blood pressure, high cholesterol, unsafe sex, child sexual abuse and intimate partner violence) together explained 37% of the total burden of disease experienced by Indigenous Australians.
- If Indigenous Australians experienced the same burden rates as the total Australian population due to these 11 selected risk factors, 29% of the total Indigenous Australian burden of disease could be avoided (Table i). This is half of the overall Indigenous health gap of 59%. This indicates that there is potential to considerably reduce the disease and injury experience of all Indigenous Australians with interventions targeted at these risk factors.

Table i: Indigenous health gap (DALYs) due to selected risk factors, expressed as a proportion of excess burden from each risk factor, 2003

	Total (%)						
Risk factor	**0–14**	**15–34**	**35–54**	**55+**	**Total**	**Health gap (DALYs)**	**% of total Indigenous burden**
Tobacco	6	0	47	47	100	9,816	10
High body mass	0	9	57	34	100	8,953	9
Physical inactivity	0	13	48	39	100	6,554	7
High blood cholesterol	0	10	64	26	100	3,994	4
Alcohol	2	45	40	13	100	3,820	4
High blood pressure	0	5	45	50	100	3,215	3
Low fruit and vegetable intake	0	10	52	37	100	2,873	3
Illicit drugs	4	63	28	5	100	2,150	2
Intimate partner violence	0	48	42	11	100	1,836	2
Child sexual abuse	0	67	28	5	100	869	1
Unsafe sex	4	40	43	12	100	926	1
11 risk factors combined[(a)]	**3**	**21**	**45**	**32**	**100**	**27,383**	**29**

(a) Joint effect of 11 risk factors in Indigenous analysis, and 14 in National Study (Begg et al. 2007) minus the burden from osteoporosis, occupation, and air pollution

- Indigenous Australians residing in remote areas experienced a disproportionate amount of the health gap due to all selected risk factors excluding illicit drugs (Figure vi). The distribution of the gap due to child sexual assault is likely to be an artefact of our methods where, due to limited information, we assumed the same prevalence of child sexual abuse in remote and non-remote areas.

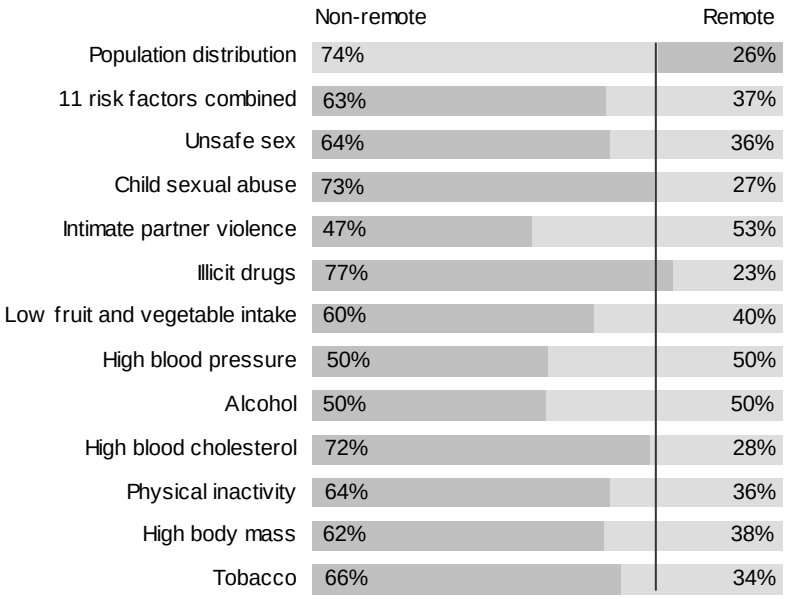


Figure vi: Indigenous health gap (DALYs) by selected risk factors expressed as proportions by remoteness, 2003

- For the total Indigenous population, the 10 risk factors associated with cardiovascular disease together explained 69% of the cardiovascular disease burden. Tobacco contributed most to this cause, followed closely by high body mass, high blood cholesterol, physical inactivity and high blood pressure.
- Eight of the risk factors were associated with cancer and together explained 49% of the total burden from this cause. In contrast, for the burden of disease and injury study in the total Australian population, the 14 risk factors considered explained 33% of the cancer burden. The major difference between the distribution of cancer burden among these risk factors was that a greater proportion of cancer was explained by tobacco in the Indigenous Australian population compared with the total Australian population (35% compared with 21%).
- More than one-third of the mental disorder burden was attributable to four of the risk factors. Alcohol contributed the most to this burden followed by illicit drugs, child sexual abuse and intimate partner violence.
- Five of the risk factors were associated with injury burden and together explained 33% of the burden from this cause. This was similar to the proportion of injury burden explained by these risk factors in the total Australian population (32%) in relative terms, but as the burden of injuries in Indigenous Australians was much larger, it meant that the average risk of an injury due to these risk factors was also much higher. Alcohol was by far the leading risk factor for injury burden in Indigenous Australians, followed by intimate partner violence, illicit drugs and child sexual abuse.
- Indigenous Australians experienced a higher rate of disease burden due to each of the 11 risk factors considered compared with the total Australian population. This resulted from a combination of higher prevalence of exposure to the risk factors, and higher disease levels in the population. The largest relative differences in rates of burden were for low fruit and vegetable consumption, tobacco, and high body mass.

Mortality and health-adjusted life expectancy

- Mortality in young and middle-aged Indigenous adults was particularly high (33% and 23% probability of dying between ages 15 and 60 years in males and females, respectively, compared with 10% and 6% in the total Australian population). A comparable high level of adult mortality was found in only a few countries in the world that are not severely affected by HIV/AIDS mortality. The probability of dying between ages 15 and 60 years was higher still in Indigenous Australians residing remotely: 46% and 31% for males and females, respectively.
- Under-five mortality in Indigenous Australians was also greater than that of the total Australian population but differences were less extreme than they were for adult mortality. The probability of dying before age 5 was 1.6% and 1.4% for males and females, respectively (compared with national figures of 0.7% and 0.6%).
- Using the general growth balance (GGB) methodology and assuming no trend in life expectancy since the 1996 to 2001 intercensal period, health-adjusted life expectancy (HALE) for Indigenous males was 56 years in 2003 (compared with 71 years for males in the total population), and for Indigenous females was 60 years (compared with 75 years for females in the total population); a gap of about 15 years respectively compared with the total population by sex.
- Indigenous Australians not only have a much shorter life span, but the proportion of time lived with disability is greater than that in the total population (13% compared with 10%).

Key implications

It has been known for a long time that Indigenous Australians experience much higher mortality rates and have worse life expectancy than the total Australian population. However, this study is the first comprehensive description of the Indigenous population that takes fatal and non-fatal health outcomes into account and details which conditions at which ages by sex contribute most to the observed enormous health gap. This study provides health policy makers with a wealth of information to identify the greatest potential for health gain by addressing particular diseases and risk factors; by targeting the most affected age groups in males and/or females; and by providing effective interventions to Indigenous people residing in cities, regional towns and remote areas.

To set priorities to achieve these health gains, complementary information is needed on the cost-effectiveness of specific interventions for each of the health problems. The details provided in this study on each disease and risk factor are essential for these economic analyses.

There is great potential to reduce the Indigenous health gap by addressing the 11 risk factors identified in this study as being responsible for half of the total health gap. There is growing evidence to guide choices on what preventive interventions for which risk factors are most likely to be effective and cost-effective in the total Australian population but not yet what the implications are for the Indigenous population. The much higher disease burden makes it more likely that interventions can achieve large health gain in Indigenous Australians. In economic evaluations this needs to be taken into account together with the different costs of interventions that are culturally appropriate and the different challenges that will arise by delivering interventions to Indigenous people residing in non-remote versus remote areas.

In this study, certain diseases and risk factors contributed more to the overall burden of disease in Indigenous Australians, and particularly to the Indigenous health gap. Cardiovascular disease, diabetes and other tobacco-related conditions (e.g. lung cancer and chronic respiratory disease) explained half the gap. Apart from tobacco, these conditions have many lifestyle risk factors in common, including high body mass, physical inactivity, raised blood pressure and cholesterol. These health problems largely affect middle-aged and older Indigenous Australians; however, they start at young ages, and there is already a sizeable burden in the 15–34-year age group. This means that prevention efforts should be targeted at a much younger Indigenous population than would be the case in the rest of the population.

Further health gain (up to 15% of the health gap) can be expected if the excess burden from infectious disease and neonatal conditions can be addressed. We also note that suicide, RTAs, and homicide & violence were the main injuries that explained 15% of the health gap. Most of this excess in injuries occurred at young adult ages. Mental disorders, including substance use disorders and particularly alcohol, also contributed significantly (10%) to the health gap.

When addressing the Indigenous health gap, it is important to note that the focus should not just be on prevention. The higher proportion of the health gap that was due to mortality reflected the greater chance of dying if Indigenous Australians fall sick. Each disease may have specific problems to be addressed; however, it is likely that the higher case fatality for most diseases was influenced by a combination of late presentations, shortcomings in acute surgical and medical management, and poor follow-up during the course of disease. Therefore, reducing the incidence and effects of these diseases requires a multi-pronged approach.

Addressing the multitude of health problems facing Indigenous Australians is complex and will require a wide range of initiatives to increase preventive and curative efforts from mainstream and particularly Indigenous health services. The National Aboriginal Community Controlled Health Organisation (NACCHO) states that:

> It is widely recognised that health solutions lie in assisting Aboriginal people being able to enjoy their right to self-determination. All relevant inquiries and studies have shown conclusively that culturally appropriate, comprehensive primary health care (such as Aboriginal community controlled health services), based on maximum community participation, is the best way of addressing Aboriginal health (NACCHO 2001).

It would be more effective to combine this with approaches outside the health sector to address the social and economic disadvantages that contribute to the poor health status of Indigenous Australians. This is in keeping with the Indigenous concept of health which acknowledges that:

> Improving Aboriginal health is not just about improving the physical well-being of an individual. It is about working towards the social, emotional, and cultural well-being of the whole community in which each individual is able to achieve their full potential as a human being (NACCHO 2001).

However these requirements should not lead to inaction by health policy makers arguing that the social and economic problems should be tackled first. It is within the reach of appropriately resourced health services to reduce a sizeable proportion of the Indigenous health gap.

1 Introduction

1.1 Purpose of the report

In 1993, Harvard University and the World Health Organization collaborated on the *World Development Report 1993 (The World Bank 1993)*. This report provided a detailed assessment of the global burden of disease, injuries and risk factors, to guide policies towards delivering more cost-effective and equitable health care to areas where achievable gains could be made. To help achieve this, a new summary measurement of population health (which combined information on mortality and non-fatal health outcomes into a single number to represent a population's health status) was developed: the disability-adjusted life year (DALY) (Murray & Lopez 1996).

Two Australian (Begg et al. 2007, Mathers et al. 1999), and two Victorian burden of disease and injury studies (Victorian Department of Human Services 1999a, 1999b, 2005) built on the methods outlined in the *World Development Report 1993.* These Australian studies provided a comprehensive overview of the burden of disease for 176 diseases and conditions by age group, sex, national health priority area, risk factors, and by area of socioeconomic disadvantage for all Australians and Victorians respectively. The results of these studies continue to be used widely in population health and health economic-related policy and research environments. In particular, the inclusion of the impact of morbidity has helped raise the profile of health conditions that do not have a significant mortality impact, such as mental disorders, hearing loss, vision loss and musculoskeletal disorders. In the recent Australian study (Begg et al. 2007) these conditions were estimated to contribute 20% to the overall disease burden.

Neither of these studies, nor subsequent smaller studies in other Australian states, quantified the burden of disease and injury for Indigenous Australians, predominantly due to concerns about incomplete Indigenous identification in population datasets. A pilot study sought to overcome these data issues by focusing on Indigenous and non-Indigenous people in the Northern Territory (NT), which has well-documented Indigenous identification practices (Zhao et al. 2004). The NT study used a combination of observed data with relativities derived from health services data on which to develop the disease models that underlie DALYs. The study demonstrated that Indigenous people had rates of DALYs two-and-a-half times as high as the Australian rate, and that the non-Indigenous Northern Territory population was similar to the total Australian population. The pilot study also showed that health inequalities not only affect the length of life (three time more years of life lost due to premature deaths) but also the health-related quality of life (almost a doubling of disability experienced due to non-fatal disease). Finally, most of the extra burden experienced by Indigenous people was found to occur in diseases (e.g. cardiovascular and renal disease, and diabetes mellitus) with preventable risk factors, such as diet, lifestyle, education and physical activity (Zhao et al. 2004).

In 2003, the Australian Government Department of Health and Ageing Office for Aboriginal and Torres Strait Islander Health (OATSIH) funded the University of Queensland to develop, in parallel to the national study, a separate study of the burden of disease in Indigenous Australians in 2003.

This study's objectives were:

- to develop internally consistent estimates of mortality for Indigenous Australians for more than 170 diseases and injuries for the year 2003 by age group, sex and cause
- to develop internally consistent estimates of disease occurrence and duration for more than 170 diseases and injuries for the year 2003 by age group, sex and cause
- to estimate the premature mortality (years of life lost, YLL), non-fatal disability (years of life lost to disability, YLD) and burden of disease and injury (DALY) for more than 170 diseases and injuries for the year 2003 by age group, sex and cause
- to estimate the attributable burden of disease and injury to major disease risk factors for the year 2003 by age group, sex and cause.

Importantly, the Indigenous burden of disease study has been guided by a steering committee of experts and representatives of Indigenous community organisations to ensure the study provides relevant information for policymaking and advocacy. We thank Professor Cindy Shannon and OATSIH for their advice with the selection of this committee. A representative technical subcommittee was also formed to discuss methodological issues. It is also noteworthy that the study operated under the umbrella of the Cooperative Research Centre for Aboriginal Health as an 'in-kind project'.

1.2 Background to the report

Indigenous Australians continue to suffer disproportionately from the consequences of European settlement (AIHW & ABS 2005). The colonisation of Australia was characterised by a legacy of unjust and misguided policies (Steering Committee for the Review of Government Service Provision 2005) that lead to the 'dispossession, physical ill-treatment, social disruption, population decline, economic exploitation, codified discrimination, and cultural devastation' of Indigenous Australians (Gardiner-Garden 1998-99, Gray et al. 2004). Today, the majority of Indigenous Australians live in conditions of clear social and economic disadvantage in terms of poor education, employment and housing outcomes (AIHW & ABS 2005). Many indicators are reported to show 'little or no movement' (Steering Committee for the Review of Government Service Provision 2005:xx) and that 'in some important respects the circumstances of Indigenous people appear to have deteriorated or regressed' (Banks 2003:9). In 2005, worsening indicators included child protection notifications rates, imprisonment rates and victim crime rates (Steering Committee for the Review of Government Service Provision 2005). All the aforementioned factors interact to contribute to the extremely poor health of Indigenous people in Australia. According to the Australian Bureau of Statistics (ABS) and the AIHW, 'Aboriginal and Torres Strait Islander people suffer greater ill-health, are more likely to experience disability and reduced quality of life and to die at younger ages, than other Australians' (AIHW & ABS 2005:91).

While it is beyond any doubt that great differentials in health status exist between Indigenous and non-Indigenous Australians, the evidence is generally limited to traditional population health indicators, such as life expectancy at birth, mortality rates, hospital separation data and reports on the prevalence of infectious diseases and lifestyle disorders (ABS & AIHW 2003, Steering Committee for the Review of Government Service Provision 2005, Zhao et al. 2004). For policy decision making, these disparate measures inadequately indicate where the opportunities for health gain lie. Burden of disease estimates for Indigenous Australians, on the other hand, would help to identify those diseases and risk

factors that are most responsible for the gap in health status between Indigenous Australians and the Australian population overall. The existence of a large gap is indicative of the potential to improve health status.

In addition, there is considerable uncertainty about the correct level of mortality, as well as disease occurrence, among Indigenous Australians. This is because routine data collection systems systematically underestimate true rates due to inadequate identification of Indigenous status, and also because population denominators are rising due to a greater propensity to identify as an Indigenous Australian. There is a lack of comprehensive, Indigenous-specific epidemiological data and little is known about non-fatal health states affecting Indigenous people.

OATSIH recognised the value of funding a study that would improve the evidence base for determining the size and impact of health problems in the Indigenous population, using a 'burden of disease' methodology, which is increasingly being used in Australia and internationally to assess population health outcomes and to evaluate the cost-effectiveness of health interventions. Burden of disease methodology is distinct from other methods of summarising population health in policy-settings, because it incorporates fatal and non-fatal conditions in assessments of health status; it separates epidemiology from advocacy to produce objective, independent and demographically plausible assessments of the burdens of particular conditions and diseases; and it can be used to assess the cost per unit of disease burden averted using a specific health intervention (Murray & Lopez 1996).

This study contributes to the development of such an agenda for Indigenous Australians 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 Australia, as well as the contribution of major risk factors to health. By doing so, this study also provides an unprecedented, comprehensive summary of the magnitude and distribution of health problems for Indigenous Australians. The study is an important foundation for further work on developing the evidence base for improving health outcomes in Indigenous Australians.

It is important to note that the burden of disease and injury method measures health status in terms of disease and infirmity. This method is not capable of incorporating the nuances inherent in the broader definition of health and wellbeing preferred by Indigenous Australians:

> Health does not simply mean the physical well being of an individual but refers to the social, emotional and cultural well being of the whole community. For Aboriginal people this is seen in terms of the whole of life view incorporating the cyclical concept of life – death – and the relationship to the land. Health care services should strive to achieve the state where every individual is able to achieve their full potential as a human being of their community. (NACCHO 2003:11).

Although the World Health Organization advocates a similar, broad definition of health, the whole-of-life view is difficult to quantify. In this study, we have opted to use the same definition of health that has been used in previous burden of disease studies in Australia (Begg et al. 2007, Mathers et al. 1999, Victorian Department of Human Services 1999a, 1999b, 2005), arguing that comparisons of health status between the Indigenous population and the total Australian population are paramount when arguing for investment in Indigenous health. Such comparisons are only valid the methods of assessment are the same.

Further, work is currently being undertaken as part of the ACE-Prevention study to define and quantify a broader concept of Indigenous Australian health benefit. The ACE-Prevention

study is a collaborative effort by the University of Queensland and Deakin University, funded by the National Health and Medical Research Council (NHMRC), and aims to evaluate the cost effectiveness of preventive health interventions for non-communicable disease. An important component of the study focuses on the cost and benefit implications of these intervention options for the health of Indigenous Australians. ACE-Prevention researchers are also collaborating with the Cooperative Research Centre for Aboriginal Health to consider important Indigenous values that extend beyond the traditional notion of health gain to individuals, including community health gain, equity and cultural security.

1.3 Unique features of the Indigenous population

In 2001, the ABS Census of Population and Housing showed that Indigenous Australians represented 2.4% of the total Australian population. The vast majority of Indigenous Australians identified as being 'only Aboriginal' in origin (90%) with small proportions reporting as being of 'only Torres Strait Islander' origin (6%) or having 'both Aboriginal and Torres Strait Islander' origins (4%) (ABS 2003b). For ease in reading, we use the term 'Indigenous Australians' to refer to the Aboriginal and Torres Strait Islander Australian population. Making separate estimates for Torres Strait Islanders was outside the scope of this study. While we recognise that Torres Strait Islander people have their own distinctive cultural identity, we also note that they share many of the characteristics of disadvantage that Indigenous Australians experience generally (AIHW & ABS 2005).

The 2001 Census also showed that the Indigenous population had a younger age profile compared with the total Australian population, with a median age of 21 years compared with 36 years (ABS 2003b). To account for these differences in age structure we use age standardisation when comparing the Indigenous and total Australian populations.

In 2001, 26% of Indigenous Australians lived in remote areas, compared with 2% of non-Indigenous Australians. While 74% of the Indigenous population lived in major cities and regional areas compared to 98% of the non-Indigenous population (ABS 2003b).

Ever since Indigenous peoples were formally counted in census and vital registration data, the number of people identifying as Indigenous has increased in excess of what can be explained by births, deaths and migration (ABS 2003b). We used specific methods to correct for Indigenous identification in Australian mortality and health datasets (Chapter 2, Appendix A and Appendix B).

1.4 Disability-adjusted life years

The measure of 'burden of disease' is the disability-adjusted life year (DALY) — the sum of the years of life lost (YLL), due to premature death, and the years lived with disability (YLD), with time as a common metric:

$$DALY = YLL + YLD$$

One DALY is equivalent to one year of healthy life lost (Zhao et al. 2004). Estimating YLL involves multiplying the number of deaths by a standard life expectancy at age of death. The formula for calculating the YLL is:

$$YLL = \text{number of deaths} \times \text{standard life expectancy (in years)}$$

The loss of healthy life due to non-fatal health conditions requires an estimate of the incidence of the 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 measures the loss of healthy life using an average health state weight (Begg et al. 2007, Murray & Lopez 1996, Victorian Department of Human Services 2005). The basic formula for calculating the YLD is:

YLD = number of incident cases x disability weight (range 0–1) x duration of disability (in years)

Estimates of diseases were first made separately, disease by disease. Adjustments were then made to account for when two or more conditions occur simultaneously in the same person, either by chance or because the conditions are related. Health loss is likely to be over-estimated without such corrections because the weights were originally derived for health states in isolation, without reference to co-existing conditions (Mathers et al. 2006). Due to a lack of appropriate data for Indigenous Australians we apply the same comorbidity adjustments as used in the National Study (Begg et al. 2007).

The great advantage of a detailed burden of disease analysis is that it facilitates meaningful comparisons between populations and identifies the most important diseases and risk factors that contribute to differences in health status and hence differences in health service needs. A detailed burden of disease analysis also indicates areas of health with the greatest potential for change. It is important to realise that burden of disease estimates are calculated for the year of interest (2003), taking into account the impact of all existing health services on levels of disease and injury.

Burden of disease analyses rely on available data sources. Even if data sources for a particular disease are inadequate, provisional estimates are made. To be transparent about these assumptions, the study results are accompanied by a detailed description of the underlying data sources and data manipulations.

1.5 Structure of the report

Chapters 2 and 3 present the methods used to estimate fatal and non-fatal health outcomes, respectively. Chapter 4 provides an overview of the burden of disease results for Indigenous Australians by cause, age and sex compared with the total Australian population. Chapter 5 gives estimates of the burden of disease attributable to selected risk factors in Indigenous Australians. Chapter 6 presents the Indigenous health gap — the difference between the current levels of disease burden in Indigenous people and what it would be if the total Australian population DALY rates applied. In this chapter we also describe the differentials in the burden of disease for Indigenous Australians by broad remoteness areas. Chapter 7 provides a general discussion of the major findings.

Technical notes on the methods for adjusting recorded mortality and hospital rates for under-identification of who is Indigenous are presented in Appendices A and B respectively. Technical notes on the methods for estimating non-fatal health outcomes and attributing risk follow in Appendices C and D.

Annex Table 1 summarises the primary data sources used to estimate disease occurrence. Annex Table 2 summarises the quality of information on disease occurrence for the main disabling conditions. Annex Table 3 summarises assumptions and confidence in the risk factor exposure estimates. Tabulations of the core results are included in Annex Tables 4–8.

2 Estimating fatal burden of disease

2.1 Background

The estimation of fatal health outcomes relies on accurate information on the population size, the numbers of death by age and sex — the mortality envelope — as well as details on the causes of death. There are problems with under-identification of Indigenous people in population censuses and in the death registration system. As a result mortality rates for Indigenous people based on recorded data suffer from bias in the numerator (number of deaths) and denominator (population estimates) and these biases are not necessarily of the same magnitude. There are indirect demographic methods to help correct for these biases. The ABS has used two of these methods to date to produce adjusted mortality estimates. However they caution readers against using their corrected mortality estimates for any purpose other than population projection because of concerns about judgments made in their application of the indirect methods (ABS 2004a). Nevertheless, the ABS life expectancy at birth figures for Indigenous Australians are widely quoted.

For this study, we engaged Professor Kenneth Hill (Professor of Demography, Harvard University), a pioneer and world expert in indirect demographic methods, to advise on the most appropriate method to adjust for under-identification of Indigenous people in census counts and the death registration system. Details of the methods have been published elsewhere and are summarised in Section 2.2, below. Further details regarding adjustment of Australian mortality data is provided in Appendix A.

The cause of death attribution in Australia is of good quality in those identified as Indigenous, as non-Indigenous or with unknown Indigenous status. Thus, the question is whether the cause of death structure in those erroneously not identified as Indigenous is the same as that for deaths identified as Indigenous. This is discussed in greater detail in Section 2.2.3, below.

Once we had estimated the Indigenous mortality envelope by age, sex and cause, we were able to estimate the loss of health for each death, in YLL. We based this estimate on the concept that not all deaths are equivalent: a death at a younger age represents a greater loss than a death at an older age. In keeping with the National Study (Begg et al. 2007) and the Global Burden of Disease study (Murray & Lopez 1996), we quantified YLL as the difference between the age at death and the remaining life expectancy at that age derived from standard life tables with a corresponding life expectancy at birth of 80.0 for males and 82.5 for females.

2.2 Methods for correcting Indigenous mortality rates

The ABS compiles information on Australian deaths from the state and territory offices of the Registrar of Births, Deaths and Marriages. It is considered that most deaths in Australia are registered and that the mortality of Indigenous Australians is substantially higher than that of the total Australian population (ABS & AIHW 2003). Despite this, the exact magnitude of the mortality difference between Indigenous Australians and the total Australian population

is unclear. This uncertainty about the true age- and sex-specific Indigenous mortality is due to:

- failure to identify the Indigenous status of all deaths
- changing propensity to identify as Indigenous in population counts, with errors thus affecting the numerators and denominators required for death and YLL rates.

To a lesser extent, there are also some concerns about incorrect data (e.g. wrong declaration of age).

2.2.1 Indirect demographic methods

The problems in estimating Australian Indigenous mortality are similar to those faced by developing countries with incomplete vital registration data, for which indirect demographic methods were developed. One group of indirect mortality estimation methods designed for post-childhood mortality correction are the 'death distribution methods', such as methods developed by Brass (1975), Preston and Hill (1980), Bennett and Horiuchi (1981), Hill (1987) and Bhat (2002). Essentially, these methods propose that if the completeness of death recording relative to population recording can be estimated, the differential in completeness can be adjusted for. All these methods, apart from Brass (1975) , compare population counts from two successive censuses and subsequently compare these with the deaths recorded in the period between censuses. This approach is based on the principle that the population in a particular age group counted during the first census can either remain alive and shift to an older age group at the next census, or die in the period between censuses (Hill et al. 2005). Physical migration in and out of the population can also affect this comparison. The Bhat method explicitly takes physical migration into account but other methods, such as Hill's (1987) GGB method, have also been adapted to consider migration (Hill & Queiroz 2004).

It is important to note that all the indirect demographic methods rely on subjective expert opinion (ABS 2004a). As there is no direct way of verifying the accuracy of estimates derived from these methods, plausibility is the key when choosing an indirect method and assessing the required assumptions (Hill et al. 2007).

2.2.2 ABS experimental population estimation

Corrected age- and sex-specific mortality rates are an essential component of estimating and projecting the Indigenous population. As a result, since 1998 the ABS has tried applying the Preston-Hill method and more recently the Bhat method to correct for the under-identification of Indigenous status in mortality data, at a national level and for grouped jurisdictions. The Bhat (2002) method reformulates the GGB (Hill 1987) method to make it applicable to populations that are affected by migration. Even though migration of Indigenous Australians in and out of the country is considered negligible, the ABS uses the migration corrections in the Bhat method to adjust for the unexplained growth of the Indigenous population between censuses, which is attributed to a changing propensity to identify as Indigenous. The Bhat method requires an a priori estimate of the population growth in the period between two censuses and this in turn requires an upfront estimate of mortality. This leads to circularity: an estimate of mortality is needed as an input to derive adjusted mortality estimates as an output. The ABS has flagged this as one of their more problematic assumptions (ABS 2004a) and so has an independent reviewer (Brown 2005).

2.2.3 General growth balance method and adult Indigenous mortality

To avoid the problems with the Bhat method, Professor Hill suggested using the GGB method. The main difference between the GGB and Bhat methods is that the former treats 'changing propensity to identify as Indigenous' from one census to the next as a 'change in census coverage' (with an age pattern proportional to the observed population), rather than 'migration' (with an age pattern that can be approximated from prior knowledge). In other respects, the methods are very similar. For instance, both the Bhat and GGB methods assume that errors in reporting deaths are proportionately constant by age, and that errors of census coverage (if there are any) are also proportionately constant by age. If the changing propensity to declare oneself as Indigenous is approximately constant across age groups, the GGB method is preferred on the grounds of simplicity, because it requires no assumption about the rate of natural increase to arrive at an estimate of the extent of the change.

If, on the other hand, the changing propensity to identify as Indigenous has a known age pattern, the Bhat method or the GGB method, adapted for migration, are preferable. Since we do not have data about this age pattern, and migration of the Indigenous population at the national level is thought to be negligible, the GGB method was selected by Professor Hill.

Application of the GGB method requires three data inputs: the initial and final population age distribution from two successive censuses, and an age pattern of deaths for the intervening period from vital registration data. To avoid incorporating the assumptions of other analysts, we opted to use the actual census counts of the 1996 and 2001 censuses (Source: ABS 2005. ABS data available on request) rather than the experimental estimated resident population (EERP). We demonstrated that this choice had little bearing on the results (Hill et al. 2007), which is not surprising because the differences between actual census counts and EERP estimates are of the same magnitude for the 1996 and 2001 censuses, with few differences for age or sex.

The second choice was to use the age structure of mortality rates for the years 2000–02 rather than rates based on recorded deaths in the five-year period between the 1996 and 2001 censuses. The reasons for this were that Queensland only started recording Indigenous status in 1996 and the number of recorded Indigenous deaths in other jurisdictions only stabilised towards the end of the 1990s. The implication of our choice to use the age structure of mortality rates for 2000–02 is that our mortality estimates reflect the mortality experience of the proportion of the population who identified themselves as Indigenous in the 2001 census. It is important to choose a particular year of reference for mortality estimates because of the phenomenon of unexplained growth in population, where more people are inclined to identify as Indigenous over time.

Third, we assumed that the difference in census coverage as estimated in the GGB method (by the intercept of the regression line) is a measure of the unexplained growth. Two assumptions in the baseline application of the GGB method are that the change in census coverage and the under-identification of deaths were the same for all age groups. In a sensitivity analysis, we demonstrated that the results were not influenced much by altering these two assumptions to reflect that both the unexplained population growth and the under-identification of deaths affects young adults more than older adults, and males more than females (Hill et al. 2007). However, it can be argued that change in census coverage and under-identification of deaths may have varied in other age groups than in our sensitivity analysis, and differentially between census and deaths. Additional sensitivity analyses could be undertaken to test the effects of such changes.

2.2.4 GGB method and adult Indigenous mortality by remoteness

We applied the GGB method (Hill 1987) separately to four groupings jurisdictions (a - Northern Territory; b - Western Australia and South Australia; c - Queensland; d – New South Wales, Victoria, Australian Capital Territory and Tasmania) and by broad remoteness areas. Results for the four regional groupings showed rather large differentials and we hypothesised that this was due to different proportions of Indigenous people in these jurisdictions residing in major cities, regional and remote areas as determined by the ARIA+ remoteness classification of usual residence (AIHW 2004b). This classification system is a geographic approach to remoteness based on road distance to five categories of service centre (a surrogate for remoteness) and on the population size of a service centre (a surrogate for the availability of services). When we compared the results from the GGB models by remoteness, we found similar mortality estimates for major cities and regional areas but much higher mortality for Indigenous Australians residing in remote areas. In consultation with this study's steering committee, we decided to restrict our burden of disease calculations to non-remote and remote groupings.

We obtained customised data from the ABS on the migration patterns between remote and non-remote areas between the 1996 and 2001 censuses. We first calculated the net migration from remote to non-remote areas by sex and five-year age group for people with a stated remote or non-remote residence in both censuses. We then scaled the figures by the ratio of the total 2001 population and those with stated area of residence in both censuses. Application of these net migration figures caused an upward adjustment of remote life expectancy by 0.6 years and a downward adjustment of non-remote life expectancy of 0.2 years.

We multiplied the GGB derived non-remote and remote life expectancy estimates by the proportions of the Indigenous population in the four regional groupings of jurisdiction mentioned above (Table 2.1). This gave life expectancy at birth estimates for the grouped jurisdictions within two years of those estimated separately by the GGB method. This supported our hypothesis that differences in life expectancy between jurisdictions were largely explained by the proportion of the population in remote areas.

Table 2.1: Proportion of Indigenous population by broad remoteness areas and grouped jurisdictions, 2001

Grouped jurisdictions	Non-remote (%)	Remote (%)
NSW, Vic, Tas, ACT	95	5
Qld	76	24
SA, WA	61	39
NT	19	81

Source: AIHW (2005b)

2.2.5 Correcting under-five mortality

Death distribution methods, such as the GGB method, do not provide information about the under-identification of deaths in young children. Therefore, different adjustments need to be made. For the life tables for the total Indigenous population, we used infant mortality rates (IMR) based on observed deaths and births, after finding that the IMR in Western Australia (Freemantle et al. 2006), estimated using linked birth and death data, was similar to that

calculated from ABS recorded deaths and births. Furthermore, the neonatal mortality rate from the AIHW perinatal data collection did not suggest that the observed IMR was an underestimate. We adjusted the observed mortality in 1 to 4 year old Indigenous children by the same GGB correction factor used for adults.

In the absence of published information on births for Indigenous children by remoteness, we assumed that IMRs for Indigenous infants residing in non-remote and remote areas for 1998–2001 from Western Australia's linked database held for all Indigenous infants in Australia by remoteness (Freemantle et al. 2006). As these IMR estimates for 1998–2001 were for males and females combined, we applied the published sex ratio from the 1980–2001 period. We adjusted the observed mortality in 1 to 4 year old Indigenous children by the increased likelihood of mortality for 1 to 7 year old Indigenous children by remoteness from Western Australia's linked database for the 1980–1997 period (Freemantle 2003).

2.3 Cause of death data for Indigenous Australians

We examined the ABS death information for Indigenous Australians in four ways. First, plots of the age-and-sex distribution of observed deaths by Indigenous status (i.e. Indigenous, non-Indigenous and not stated groups) showed an almost complete overlap between the non-Indigenous and not-stated proportions, and a very different age pattern for those identified as Indigenous. This indicated to us that the unknown number of unidentified Indigenous deaths could have been coded to either the not-stated or non-Indigenous category. Second, age- and sex-specific mortality rates based on recorded Indigenous deaths showed an expected exponential increase from age 35 onwards. This suggested that there was no obvious underreporting of Indigenous deaths by age. Third, age-standardised proportions of deaths according to burden of disease broad cause groups (group I: communicable disease, maternal and neonatal causes; group II: non-communicable disease; and group III: injuries) were similar by Indigenous status indicating that the broad cause of death structure was similar for Indigenous and non-Indigenous Australians. Fourth, the proportions of ill-defined and residual categories contributing to overall mortality were similar for Indigenous and non-Indigenous deaths.

We concluded that the recording of mortality by age, sex and cause for Indigenous Australians was plausible and of good quality. Thus, we assumed that the cause of death pattern for deaths recorded as Indigenous reflected the cause of death pattern of all Indigenous deaths (including the unknown number of unidentified Indigenous deaths). As numbers of deaths by age, sex and cause can fluctuate from year to year, we assessed the cause of death pattern for the 2001 to 2003 period. In consultation with the study's steering committee and technical advisory panel, we fixed the mortality envelope for Indigenous Australians as the mortality rates predicted from the GGB exercise for the 1996–2001 period multiplied by the 2003 Indigenous population estimates. We then multiplied the resulting numbers of age- and sex-specific deaths by the proportions of deaths for each cause to obtain our final estimates of corrected Indigenous deaths by age, sex and cause.

These calculations assumed no changes in mortality from the 1996 to 2001 period to 2003. This decision was based on consultation with the steering committee, due to the paucity of available trend data. However, recent evidence suggests that this may not be the case, at least for the Northern Territory (Wilson et al. 2007) . Between 1967 and 2004, the life expectancy at birth increased by 8.0 years for Indigenous men and 14.2 years for Indigenous women in the Northern Territory (Wilson et al. 2007). These findings were derived from what is considered to be the most complete and internally consistent database of deaths by Indigenous status

and population counts in Australia for the period 1967–2004 (Wilson et al. 2007). On average, and assuming a stable mortality decline over time, this translates to an increase of 0.22 and 0.38 years per year for males and females respectively, over the 38-year period. This is within the same range of the increase that has been observed for the total Australian population (although with a different sex ratio), which was 0.33 and 0.22 years per year for males and females respectively over the past 10 years (Begg et al. 2007). It is worthwhile noting that despite this increase in life expectancy at birth over time for Indigenous people residing in the Northern Territory, the disparity in life expectancy compared with the total Australian population remained substantial: 15.2 years for females, and 17.7 years for males (Wilson et al. 2007).

3 Estimating non-fatal burden of disease

3.1 Background

The disease and injury models for Indigenous Australians are based on the models developed for the National Study (Begg et al. 2007). This ensures consistent and meaningful comparisons between the Indigenous and the total Australian population. The Indigenous-specific epidemiological information required to complete the disease models were derived using similar methods to the pilot Northern Territory study (Zhao et al. 2004).

3.1.1 The burden of disease and injury in Australia, 2003

The National Study developed internally consistent measures of incidence, prevalence, remission, mortality and duration for more than 170 diseases and injuries that describe the health status of the total Australian population, 2003 (Begg et al. 2007).. These models were based on a critical examination of Australian data sources, including disease registers, surveillance systems, notification systems, vital registration systems, health service use data, population health surveys and epidemiological studies. If Australian data sources were few or absent, international epidemiological studies were used to inform estimates (e.g. dementia) and expert opinion was sought to validate assumptions. A small number of 'other' categories were estimated indirectly based on the YLL to YLD ratio from the rest of the disease category. For further explanation, we encourage readers to refer to Annex Table 2 (which summarises the primary data sources for incidence and prevalence) and Appendix 1 (which discusses each of the models in detail) in the National Study (Begg et al. 2007).

3.1.2 Burden of disease and injury in the Northern Territory

The Northern Territory study estimated disease and injury models for Indigenous and non-Indigenous populations using multiple sources. These sources included observed events based on Northern Territory surveillance systems, disease registries and local epidemiological studies (for 36% of YLD), relativities based on Northern Territory hospital data (29% of YLD), and by assuming the national 'average' when there was no available evidence to suggest otherwise (36% of YLD) (Zhao et al. 2004).

3.2 Methods for calculating YLD

We undertook a comprehensive literature review to identify all potential data sources for calculating YLD for Indigenous Australians. Due to the lack of available data, for the majority of disease models we concentrated on estimating incidence or prevalence and defaulted to using the same assumptions for remission, relative risk of mortality and the proportion of time symptomatic. We estimated the disease occurrence for Indigenous Australians by using observed health events (rates or proportions) and/or applying differentials (rate ratios) in health events to the disease models developed for the National Study (Begg et al. 2007). Data sources for disease occurrence included routine data collections (such as Australian perinatal collection data, notification data, mortality data and hospital data) (covering 53% of YLD); self-report and measured population health surveys (28% of YLD), and epidemiological studies that identified Indigenous Australians (3% of

YLD) (Table 3.1). For a number of conditions, we assumed the same disease occurrence as the National Study because of the lack of data to suggest otherwise (concerning 10% of YLD). Finally, in keeping with the National Study, to estimate rest categories (such as 'other cancers'), we applied a ratio of YLL to YLD from the rest of the disease category to the YLL for the disease in question (6% of YLD). We encourage readers to refer to Annex Table 1 for a summary of the primary data sources for incidence and prevalence; and Appendix C for a summary of each of the explicit disease models.

Table 3.1: Principal source of YLD estimates by percent of total YLD

Source	YLD	Per cent of total YLD
Disease registers, surveillance, notification & vital registration systems	1,880	4
Health service utilisation data	21,895	49
Population health surveys	12,510	28
Epidemiological studies	1,362	3
Indirect estimation	2,477	6
Assume same incidence as the total Australian population	4,378	10

3.3 Precision of estimates

The precision of the YLD estimates is not quantifiable in the usual statistical sense of deriving a confidence interval, because of the diversity of the data sources and the methods used (Zhao et al. 2004).

There are a number of areas of uncertainty in YLD estimation for Indigenous Australians. As already mentioned, the accurate identification of Indigenous people in health data collections is a challenging issue. In the previous chapter, we highlighted the use of an indirect demographic method to adjust the observed mortality rates to give more plausible mortality rates for Indigenous Australians for the purpose of this study. Similarly, we adjusted the Australian hospital data (AIHW 2003b) for under-identification of Indigenous people by state and remoteness using a method developed by the AIHW for their *Expenditures on health for Aboriginal and Torres Strait Islander peoples, 2001–02* report (AIHW 2005b). Broadly, this approach involves applying correction factors by jurisdiction and remoteness across diseases, age and sex. These correction factors were derived by the AIHW, in part, from jurisdictional studies on the quality of Indigenous identification in hospitals. For more information on this method, see Appendix B.

The epidemiological information available for Indigenous Australians is limited (see Annex Table 2). Specifically, data sources allowing direct measurement of disease parameters for Indigenous Australians are few; often not representative for all Indigenous Australians; and/or for a different time period.

In addition to data identification issues and gaps in the literature, when judging the quality of the burden of disease estimates for Indigenous Australians, it is also important to note that the quality of the data sources not only differ from each other but within the source by disease. For example, hospital data can be used reliably for estimating health events, such as injuries and maternal conditions, where hospital admission is an important part of managing these conditions. However, less confidence can be placed in using relativities based on

hospital data for conditions for which hospitalisation is only a small part of management (e.g. asthma, depression and anxiety, osteoarthritis).

This brings us to the underlying assumption of the majority of the disease models: relativities based on health events and self-report population health surveys between the Indigenous population and the non-Indigenous or total Australian populations reflect actual differentials in disease occurrence. The consistency we observed in gradients of several indirect sources for non-fatal outcomes with those found in mortality and population health survey data added credibility to use of relativities. However, for a number of conditions we were unable to triangulate data sources (e.g. Alzheimer and other dementias, Parkinson's disease and migraine). For these conditions, we assumed the same disease occurrence as the total Australian population. For conditions such as adult-onset hearing loss, for which there is considerable uncertainty around the national estimates, the accuracy of the Indigenous-specific estimates will be affected even more. We encourage the reader to refer to Annex Table 2 ('Assessment of quality of disease occurrence information for the main disabling conditions in the Indigenous population') and Section 7.3 ('Precision of estimates') in the National Study for a discussion of these issues (Begg et al. 2007).

Petrol sniffing is a substance use disorder that causes devastating health and social consequences for Indigenous Australian young people and their communities. Unfortunately, we were unable to model the disability related to petrol sniffing as a distinct substance disorder category because of the problems estimating the prevalence of this condition and the uncertainty about the exact nature of long-term harms. According to the literature, the number of Indigenous people who sniff petrol in Australia and in individual communities has fluctuated considerably over time; petrol sniffing does not occur in all Indigenous communities, and occurs in 'waves' in some regions, depending on the time of year (Gray et al. 2004). Furthermore, the increasing uptake of non-sniffable petrols, such as 'Avgas' and 'Opal' in remote areas in recent years also hinders the use of estimates from older studies.

It is clear from the considerations above that there is great scope for refining the models as appropriate data becomes available. Notwithstanding these qualifications, we consider this study to make the most comprehensive and critical use of health information for Indigenous Australians to date. Furthermore, we anticipate that the data gaps and deficiencies identified in this study will contribute to setting priorities for improving health information.

4 Burden of disease and injury in Indigenous Australians

4.1 Overview

This chapter presents the 2003 burden of disease and injury in Indigenous Australians, with comparisons to the results for the total Australian population. This chapter discusses the overall burden, fatal and non-fatal burden, and burden by the six leading broad cause categories: cardiovascular disease, mental disorders, intentional and unintentional injuries combined, chronic respiratory disease, diabetes mellitus, and cancers. Because the Indigenous Australian population is younger than the total Australian population overall rates comparing these groups are age standardised.

4.2 Disability-adjusted life years

In 2003, the Indigenous Australian population made up 2.4% of the total Australian population but, despite its much younger structure, carried 3.6% of the total Australian population disease burden.

The majority of the absolute burden (number of DALYs) for Indigenous Australians occurred in the middle-aged population with a significant peak also occurring in the very young (Figure 4.1). In the total Australian population, the absolute burden continued to increase into old age for females, while for males there was a steady increase peaking at age 70–74 years.

The rate of burden, in DALYs, peaked in both Indigenous Australians and the total Australian population in the very young, followed by a sharp drop then a steady rise to old age (Figure 4.1). In the Indigenous Australian population, the rate of burden increased at a much younger age than for the total Australian population. The rate of burden also occurred at a considerably higher rate at each age for Indigenous Australians compared with the total Australian population.

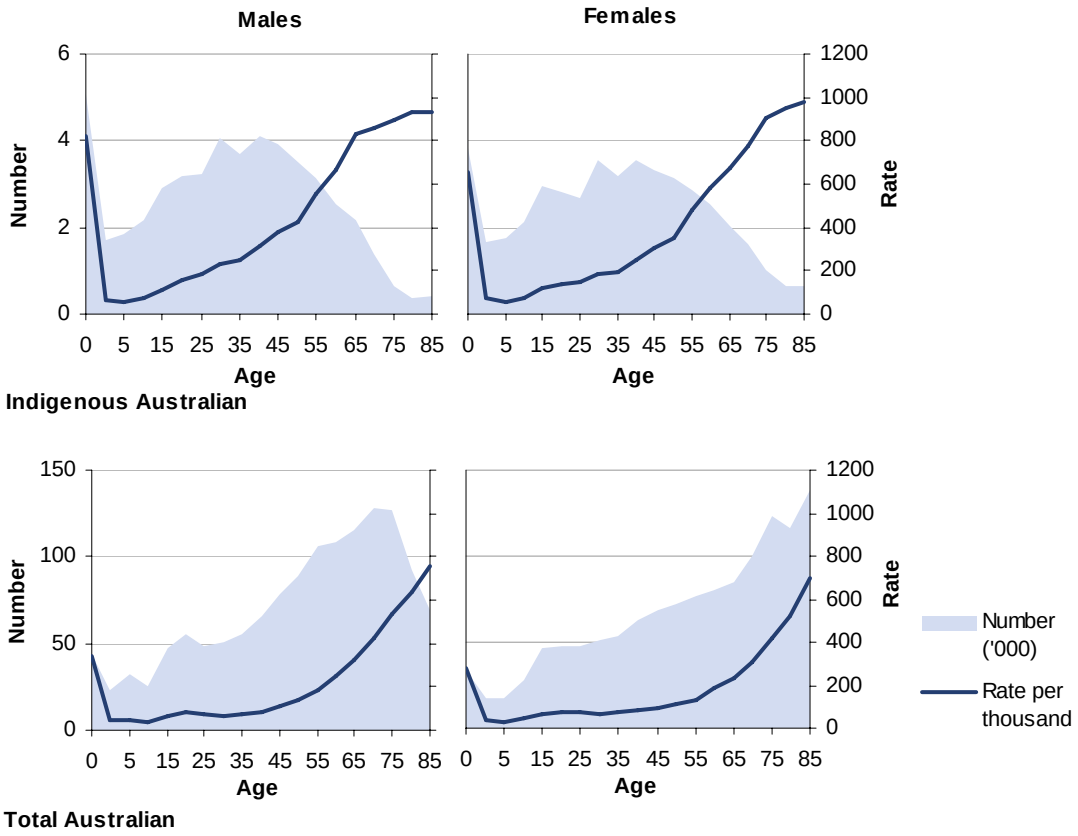


Figure 4.1: DALYs by age and sex expressed as rates and numbers, Indigenous Australian and total Australian populations, 2003

Cardiovascular disease and mental disorders were the leading causes of burden in the Indigenous Australian population in 2003 (Figure 4.2). These two broad cause groups together accounted for 32% of the Indigenous disease burden. Chronic respiratory disease, diabetes mellitus, and cancers were the next three leading causes, accounting for an approximately equal proportion of the disease burden at 8% each.

Cancer was responsible for a greater proportion of the burden in the total Australian population (19%) than the Indigenous population (8%) (Figure 4.2). Diabetes, and unintentional and intentional injuries were each responsible for a larger proportion of the burden in Indigenous Australians than in the total Australian population.

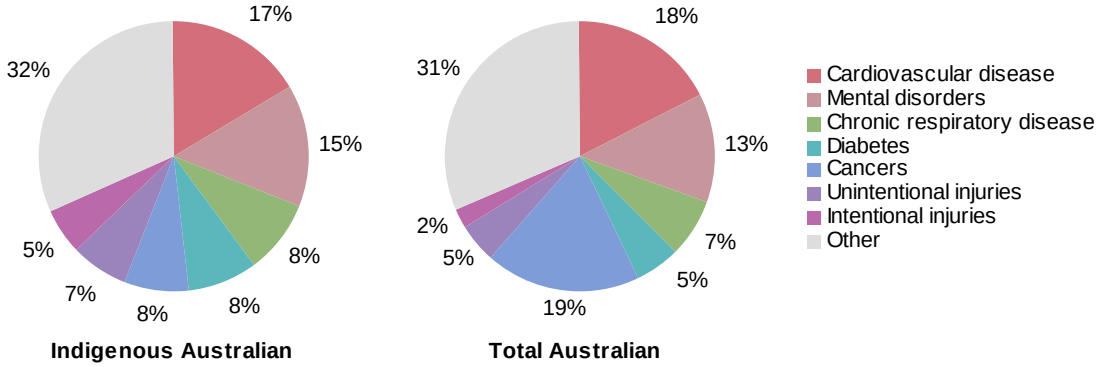


Figure 4.2: DALY by broad cause group, Indigenous Australian and total Australian populations, 2003

There was a reversal in the sex ratios of the burden due to diabetes mellitus and cancers between the total Australian and Indigenous Australian populations, with the majority of the experienced by females in the Indigenous population (Figure 4.3).

A greater proportion of the Indigenous burden was due to premature mortality compared with the total Australian population (Figure 4.3). For five of the seven leading disease groups, the share of the burden caused by mortality was higher in Indigenous people. Meanwhile a greater proportion of the burden from intentional injuries was due to non-fatal disability whereas the proportion of non-fatal disability for cardiovascular disease was similar for the two populations.

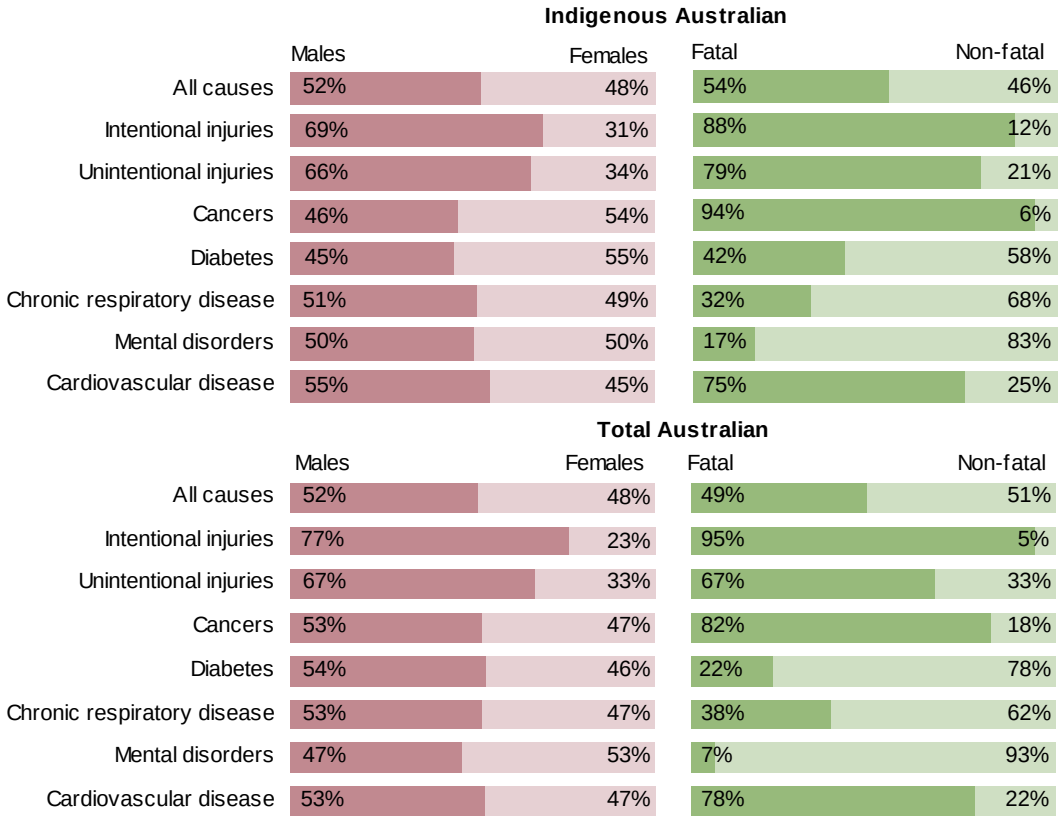
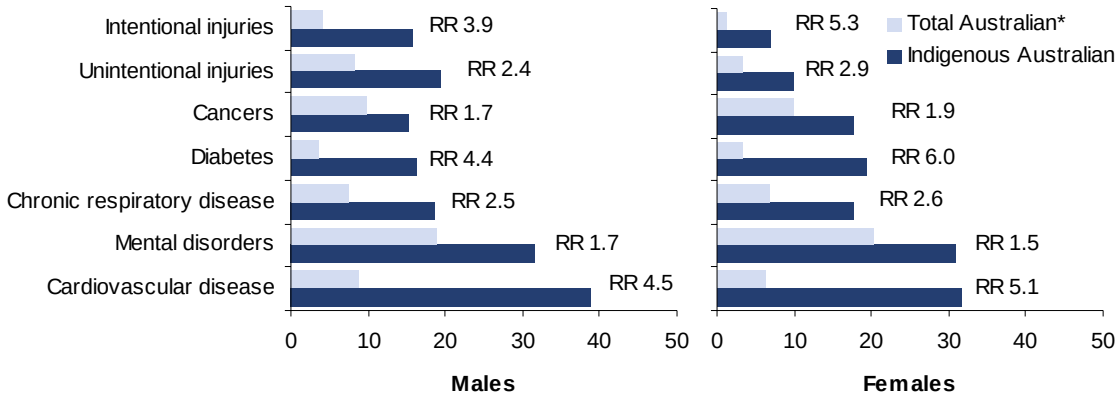


Figure 4.3: DALYs by broad cause group expressed as proportions by sex, and proportions due to fatal and non-fatal outcomes, Indigenous Australian and total Australian populations, 2003

Among the top seven broad cause categories contributing to Indigenous burden, the largest differentials in disease burden rates for the Indigenous and total Australian population were for cardiovascular disease, diabetes mellitus, and intentional injuries in both males and females (Figure 4.4).



* Age standardised to the total Indigenous Australian population, 2003
RR Indigenous Australian to total Australian rate ratio

Figure 4.4: DALY rate per 1,000 and rate ratios for the leading broad cause groups by sex, Indigenous Australian and total Australian populations, 2003

Ischaemic heart disease was the largest single contributor to the disease burden experienced by Indigenous males, accounting for 11.8% of the total Indigenous male burden (Table 4.1). Type 2 diabetes, anxiety & depression, suicide, and RTAs were the next four leading causes of male burden, together accounting for 21.9% of the male Indigenous burden. For females, the leading cause of burden was anxiety & depression, causing 10.0% of the total Indigenous female burden. Type 2 diabetes, ischaemic heart disease, asthma, and COPD were the next four leading causes, accounting for 26.2% of the female Indigenous burden.

Table 4.1: Leading causes of DALYs by sex, Indigenous Australians, 2003

	Males			Females		
Rank	**Condition**	**DALY**	**Per cent of total**	**Condition**	**DALY**	**Per cent of total**
	All causes	**50,107**	**100.0**	**All causes**	**45,869**	**100.0**
1	Ischaemic heart disease	5,899	11.8	Anxiety & depression	4,582	10.0
2	Type 2 diabetes	3,520	7.0	Type 2 diabetes	4,361	9.5
3	Anxiety & depression	2,864	5.7	Ischaemic heart disease	4,074	8.9
4	Suicide	2,644	5.3	Asthma	1,907	4.2
5	Road traffic accidents	1,955	3.9	COPD	1,678	3.7
6	COPD	1,941	3.9	Stroke	1,413	3.1
7	Alcohol dependence & harmful use	1,797	3.6	Road traffic accidents	1,074	2.3
8	Asthma	1,396	2.8	Alcohol dependence & harmful use	1,008	2.2
9	Stroke	1,293	2.6	Lung cancer	945	2.1
10	Homicide & violence	1,102	2.2	Homicide & violence	854	1.9
11	Low birth weight	1,001	2.0	Low birth weight	808	1.8
12	Lung cancer	995	2.0	Pneumonia	798	1.7
13	Pneumonia	878	1.8	Suicide	795	1.7
14	Inflammatory heart disease	799	1.6	Breast cancer	719	1.6
15	Heroin or polydrug dependence	771	1.5	Rheumatic heart disease	660	1.4
16	Schizophrenia	695	1.4	Deficiency anaemia	626	1.4
17	Epilepsy	616	1.2	Schizophrenia	558	1.2
18	Hepatitis	590	1.2	Otitis media	505	1.1
19	Birth trauma & asphyxia	538	1.1	Heroin or polydrug dependence	453	1.0
20	Otitis media	516	1.0	STDs (not HIV/AIDS)	450	1.0

The top 20 causes of burden in Indigenous Australians, and the corresponding ranks for the total Australian population, revealed differences in the distribution of cause of burden in the two populations (Table 4.2). For Indigenous Australian males, otitis media, homicide & violence, birth trauma & asphyxia, and low birth weight were ranked at least 20 places higher than in the total Australian population. For females, sexually transmitted diseases, homicide & violence, otitis media, and rheumatic heart disease showed the largest shift in rank.

Table 4.2: Rank of leading causes of DALYs, Indigenous Australian and total Australian populations, 2003

Males			Females		
Condition	**Indigenous Australian**	**Total Australian**	**Condition**	**Indigenous Australian**	**Total Australian**
Ischaemic heart disease	1	1	Anxiety & depression	1	1
Type 2 diabetes	2	2	Type 2 diabetes	2	4
Anxiety & depression	3	3	Ischaemic heart disease	3	2
Suicide	4	8	Asthma	4	9
Road traffic accidents	5	12	COPD	5	7
COPD	6	6	Stroke	6	3
Alcohol dependence & harmful use	7	14	Road traffic accidents	7	22
Asthma	8	13	Alcohol dependence & harmful use	8	34
Stroke	9	5	Lung cancer	9	8
Homicide & violence	10	46	Homicide & violence	10	75
Low birth weight	11	37	Low birth weight	11	33
Lung cancer	12	4	Pneumonia	12	16
Pneumonia	13	21	Suicide	13	24
Inflammatory heart disease	14	32	Breast cancer	14	6
Heroin or polydrug dependence	15	24	Rheumatic heart disease	15	74
Schizophrenia	16	16	Deficiency anaemia	16	52
Epilepsy	17	36	Schizophrenia	17	19
Hepatitis	18	23	Otitis media	18	81
Birth trauma & asphyxia	19	54	Heroin or polydrug dependence	19	55
Otitis media	20	81	STDs (not HIV/AIDS)	20	86

Among the 20 conditions that caused the greatest burden in Indigenous Australians, the largest differentials in burden rate between Indigenous and total Australian population males were for homicide & violence, inflammatory heart disease, and lower respiratory tract infection (Table 4.3). For females, the largest differentials were for rheumatic heart disease, homicide & violence, and alcohol dependence & harmful use.

Table 4.3: Rate per 1,000 and rate ratio of top 20 leading causes of DALYs, Indigenous Australian and total Australian populations, 2003

	Males				Females			
Rank	Condition	Indigenous Australian	Total Australian[(a)]	RR[(b)]	Condition	Indigenous Australian	Total Australian[(a)]	RR[(b)]
	All causes	**212.4**	**87.6**	**2.4**	**All causes**	**191.5**	**78.0**	**2.5**
1	Ischaemic heart disease	25.0	4.9	5.1	Anxiety & depression	19.1	13.9	1.4
2	Type 2 diabetes	14.9	3.2	4.6	Type 2 diabetes	18.2	2.9	6.3
3	Anxiety & depression	12.1	7.3	1.7	Ischaemic heart disease	17.0	2.6	6.6
4	Suicide	11.2	3.4	3.3	Asthma	8.0	4.8	1.7
5	Road traffic accidents	8.3	3.3	2.5	COPD	7.0	1.4	4.9
6	COPD	8.2	1.9	4.3	Stroke	5.9	1.9	3.1
7	Alcohol dependence & harmful use	7.6	2.1	3.7	Road traffic accidents	4.5	1.2	3.7
8	Asthma	5.9	4.8	1.2	Alcohol dependence & harmful use	4.2	0.5	7.9
9	Stroke	5.5	2.1	2.7	Lung cancer	3.9	1.2	3.3
10	Homicide & violence	4.7	0.7	6.8	Homicide & violence	3.6	0.3	11.0
11	Low birth weight	4.2	1.7	2.5	Low birth weight	3.4	1.5	2.3
12	Lung cancer	4.2	1.7	2.4	Pneumonia	3.3	0.5	6.8
13	Pneumonia	3.7	0.6	6.1	Suicide	3.3	1.0	3.3
14	Inflammatory heart disease	3.4	0.5	6.3	Breast cancer	3.0	2.9	1.0
15	Heroin or polydrug dependence	3.3	1.4	2.3	Rheumatic heart disease	2.8	0.1	26.4
16	Schizophrenia	2.9	1.8	1.6	Deficiency anaemia	2.6	0.4	6.2
17	Epilepsy	2.6	1.1	2.4	Schizophrenia	2.3	1.3	1.7
18	Hepatitis	2.5	0.6	4.0	Otitis media	2.1	0.3	6.2
19	Birth trauma & asphyxia	2.3	1.1	2.2	Heroin or polydrug dependence	1.9	0.5	3.7
20	Otitis media	2.2	0.4	5.4	STDs (not HIV/AIDS)	1.9	0.2	9.2

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

Other conditions that occur at a much higher rate in the Indigenous Australian population, but are not among the leading contributors to Indigenous burden of disease include: trachoma, which we assume only occurs in Indigenous Australians residing in remote areas; rheumatic heart disease (Indigenous to total Australian RR 23.2 in males and 26.4 in females); sexually transmitted diseases (male RR 11.6, female RR 9.2); non-hepatitis liver cancer (male RR 5.5, female RR 6.7); septicaemia (male RR 7.3, female RR 7.9); sudden infant death syndrome (male RR 5.7, female RR 7.3); deficiency anaemia (male RR 7.5, female RR 6.2); and pancreatitis (male RR 11.4, female RR 4.7).

4.3 Years of life lost

Years of life lost (YLL), or fatal burden, accounted for 54% of the total Indigenous Australian disease burden in 2003 (Figure 4.3). This was higher than the total Australian population where 49% of total burden was due to mortality. The bulk of the absolute fatal burden occurred in the very young and middle ages for Indigenous Australians (Figure 4.5), in part reflecting the younger age structure of the population. At every age, the Indigenous Australian rate of fatal burden was higher than that of the total Australian population.

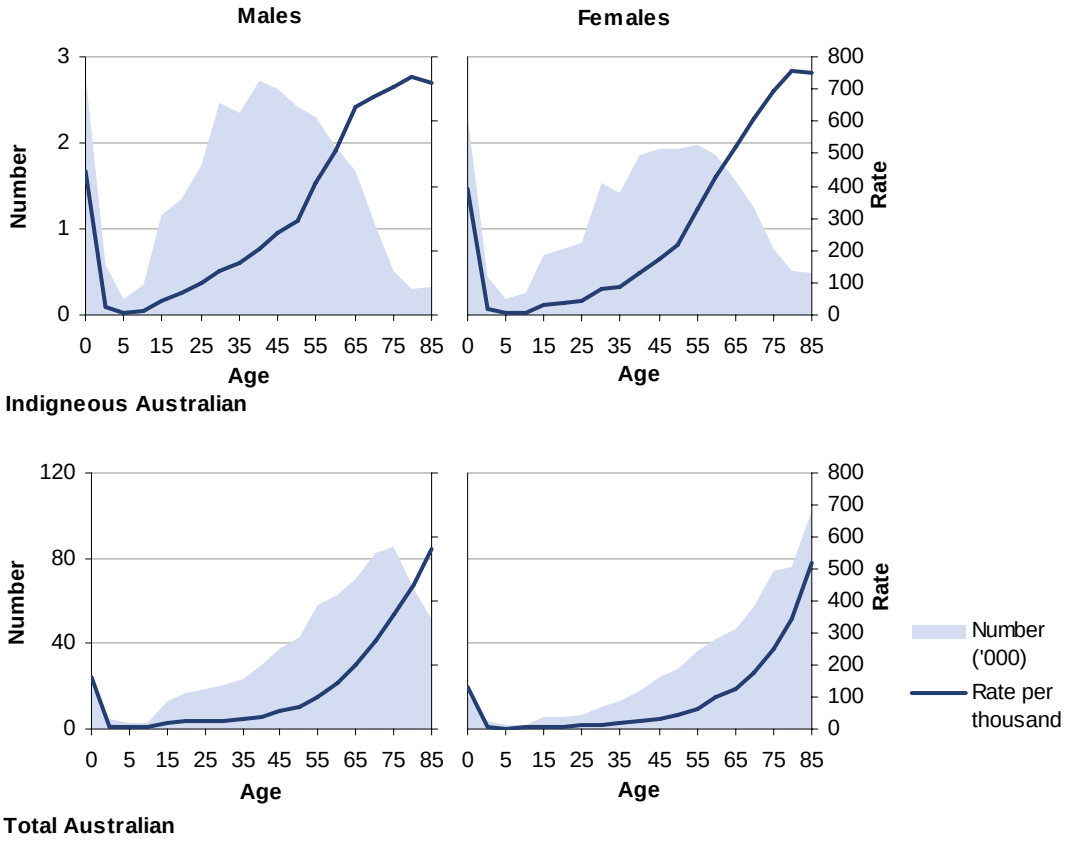


Figure 4.5: YLL by age and sex expressed as rates, and numbers, Indigenous Australian and total Australian populations, 2003

Ischaemic heart disease was the largest disease or injury category contributing to Indigenous Australian fatal burden (Table 4.4). The second leading cause was suicide for males and Type 2 diabetes for females. Road traffic injury was the third leading cause of fatal burden for males and females.

The ranking of suicide in Indigenous males and females (second and seventh respectively) was considerably higher than that for Australian males and females (seventh and twenty-third, respectively). This was partly due to the younger age structure of the Indigenous Australian population, but particularly due to the increased incidence of suicide in the Indigenous Australian population, where it occurred at around three times the total Australian rate.

Table 4.4: Leading causes of YLL by sex, Indigenous Australians, 2003

	Males			Females		
Rank	Condition	YLL	Per cent of total	Condition	YLL	Per cent of total
	All Causes	**28,904**	**100.0**	**All Causes**	**22,571**	**100.0**
1	Ischaemic heart disease	5,026	17.4	Ischaemic heart disease	2,995	13.3
2	Suicide	2,628	9.1	Type 2 diabetes	1,735	7.7
3	Road traffic accidents	1,786	6.2	Road traffic accidents	1,008	4.5
4	Type 2 diabetes	1,336	4.6	Stroke	932	4.1
5	Alcohol dependence & harmful use	1,125	3.9	Lung cancer	923	4.1
6	Lung cancer	971	3.4	COPD	807	3.6
7	Stroke	899	3.1	Suicide	783	3.5
8	COPD	864	3.0	Alcohol dependence & harmful use	758	3.4
9	Homicide & violence	802	2.8	Breast cancer	641	2.8
10	Pneumonia	711	2.5	Pneumonia	618	2.7
11	Inflammatory heart disease	584	2.0	Homicide & violence	561	2.5
12	Low birth weight	584	2.0	Low birth weight	509	2.3
13	Hepatitis	546	1.9	Rheumatic heart disease	455	2.0
14	Epilepsy	469	1.6	Hepatitis	389	1.7
15	SIDS	413	1.4	SIDS	342	1.5
16	Suffocation & foreign bodies	402	1.4	Nephritis & nephrosis	333	1.5
17	Nephritis & nephrosis	297	1.0	Cervical cancer	323	1.4
18	Drowning	290	1.0	Colorectal cancer	290	1.3
19	Poisoning	280	1.0	Inflammatory heart disease	271	1.2
20	Oesophagus cancer	277	1.0	Poisoning	250	1.1

Indigenous Australians experienced a higher rate of fatal burden for all 20 leading conditions compared with the total Australian population (Table 4.5). The Indigenous fatal burden rate was at least double that seen in the total Australian population for all 20 leading fatal conditions in males, and 18 of the top 20 conditions in females. The ratio of the Indigenous population to the total Australian population rates was largest for Type 2 diabetes for both males and females (RR 12.8 males; RR 24.5 females). Indigenous females also experienced a much higher rate of fatal burden from rheumatic heart disease, and alcohol dependence & harmful use compared with the total Australian female population.

Table 4.5: Rate per 1,000 and rate ratio of top 20 leading causes of YLL, Indigenous Australian and total Australian populations, 2003

	Males				Females			
Rank	Condition	Indigenous Australian	Total Australian[(a)]	RR[(b)]	Condition	Indigenous Australian	Total Australian[(a)]	RR[(b)]
	All causes	**122.5**	**34.9**	**3.5**	**All causes**	**94.3**	**23.3**	**4.1**
1	Ischaemic heart disease	21.3	4.1	5.2	Ischaemic heart disease	12.5	1.8	6.8
2	Suicide	11.1	3.4	3.3	Type 2 diabetes	7.2	0.3	24.5
3	Road traffic accidents	7.6	2.8	2.7	Road traffic accidents	4.2	1.0	4.2
4	Type 2 diabetes	5.7	0.4	12.8	Stroke	3.9	1.1	3.7
5	Alcohol dependence & harmful use	4.8	0.6	8.4	Lung cancer	3.9	1.1	3.5
6	Lung cancer	4.1	1.6	2.5	COPD	3.4	0.6	6.0
7	Stroke	3.8	1.0	3.7	Suicide	3.3	1.0	3.4
8	COPD	3.7	0.7	5.3	Alcohol dependence & harmful use	3.2	0.2	20.7
9	Homicide & violence	3.4	0.5	7.1	Breast cancer	2.7	1.8	1.5
10	Pneumonia	3.0	0.4	7.0	Pneumonia	2.6	0.3	8.0
11	Inflammatory heart disease	2.5	0.4	5.8	Homicide & violence	2.3	0.2	9.6
12	Low birth weight	2.5	1.0	2.5	Low birth weight	2.1	0.8	2.8
13	Hepatitis	2.3	0.6	3.9	Rheumatic heart disease	1.9	0.1	24.4
14	Epilepsy	2.0	0.3	5.8	Hepatitis	1.6	0.3	5.4
15	SIDS	1.8	0.3	5.7	SIDS	1.4	0.2	7.3
16	Suffocation & foreign bodies	1.7	0.3	5.0	Nephritis & nephrosis	1.4	0.2	6.5
17	Nephritis & nephrosis	1.3	0.3	4.9	Cervical cancer	1.3	0.2	5.7
18	Drowning	1.2	0.4	3.3	Colorectal cancer	1.2	0.8	1.5
19	Poisoning	1.2	0.6	2.0	Inflammatory heart disease	1.1	0.2	5.4
20	Oesophagus cancer	1.2	0.3	3.5	Poisoning	1.0	0.3	3.3

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

The leading causes of death were similar to the leading causes of fatal burden (Table 4.6). However, where the condition was dominated by mortality in the younger ages, the YLL per death was higher than for a condition that primarily affects older people (i.e. a death at a young age is more heavily weighted than a death in old age). For example, the YLL rate was higher in Indigenous males for RTAs than for Type 2 diabetes (Table 4.65) despite there being more deaths due to Type 2 diabetes (Table 4.6).

Table 4.6: Leading causes of death by sex, Indigenous Australians, 2003

	Males			Females		
Rank	**Condition**	**Deaths**	**Per cent of total**	**Condition**	**Deaths**	**Per cent of total**
	All Causes	**1,574**	**100.0**	**All Causes**	**1,296**	**100.0**
1	Ischaemic heart disease	314	20.0	Ischaemic heart disease	213	16.4
2	Suicide	104	6.6	Type 2 diabetes	115	8.8
3	Type 2 diabetes	85	5.4	Stroke	77	6.0
4	Road traffic accidents	72	4.6	Lung cancer	58	4.5
5	COPD	71	4.5	COPD	57	4.4
6	Stroke	71	4.5	Road traffic accidents	39	3.0
7	Lung cancer	68	4.3	Breast cancer	37	2.9
8	Alcohol dependence & harmful use	59	3.7	Pneumonia	37	2.8
9	Pneumonia	40	2.5	Alcohol dependence & harmful use	35	2.7
10	Homicide & violence	32	2.0	Suicide	29	2.2
11	Inflammatory heart disease	29	1.8	Nephritis & nephrosis	25	1.9
12	Hepatitis	28	1.8	Homicide & violence	21	1.6
13	Epilepsy	21	1.3	Rheumatic heart disease	21	1.6
14	Nephritis & nephrosis	20	1.3	Dementia	20	1.6
15	Low birth weight	19	1.2	Hepatitis	19	1.5
16	Oesophagus cancer	19	1.2	Cervical cancer	18	1.4
17	Colorectal cancer	17	1.1	Colorectal cancer	17	1.3
18	Mouth cancers	17	1.1	Low birth weight	17	1.3
19	Prostate cancer	16	1.0	Type 1 diabetes	15	1.2
20	Suffocation & foreign bodies	16	1.0	Inflammatory heart disease	14	1.1

A comparison of the mortality rates between the Indigenous and total Australian populations for the top 20 leading conditions showed a similar pattern as that described above for YLL rates (Table 4.7).

Table 4.7: Mortality rate per 10,000 and rate ratio of top 20 leading causes, Indigenous Australian and total Australian populations, 2003

	Males				Females			
Rank	Condition	Indigenous Australian	Total Australian[(a)]	RR[(b)]	Condition	Indigenous Australian	Total Australian[(a)]	RR[(b)]
	All causes	**66.7**	**21.5**	**3.1**	**All causes**	**54.1**	**14.2**	**3.8**
1	Ischaemic heart disease	13.3	3.6	3.7	Ischaemic heart disease	8.9	1.8	5.0
2	Suicide	4.4	1.4	3.1	Type 2 diabetes	4.8	0.3	18.9
3	Type 2 diabetes	3.6	0.4	9.2	Stroke	3.2	1.0	3.3
4	Road traffic accidents	3.0	1.1	2.8	Lung cancer	2.4	0.7	3.2
5	COPD	3.0	0.7	4.4	COPD	2.4	0.5	5.2
6	Stroke	3.0	1.0	2.9	Road traffic accidents	1.6	0.4	4.2
7	Lung cancer	2.9	1.3	2.3	Breast cancer	1.6	1.0	1.5
8	Alcohol dependence & harmful use	2.5	0.3	8.0	Pneumonia	1.5	0.2	6.1
9	Pneumonia	1.7	0.4	4.5	Alcohol dependence & harmful use	1.5	0.1	18.7
10	Homicide & violence	1.3	0.2	7.2	Suicide	1.2	0.4	3.1
11	Inflammatory heart disease	1.2	0.2	5.1	Nephritis & nephrosis	1.0	0.2	5.4
12	Hepatitis	1.2	0.3	3.4	Homicide & violence	0.9	0.1	10.0
13	Epilepsy	0.9	0.1	6.3	Rheumatic heart disease	0.9	0.0	18.3
14	Nephritis & nephrosis	0.9	0.3	3.1	Dementia	0.8	0.3	3.0
15	Low birth weight	0.8	0.3	2.5	Hepatitis	0.8	0.2	4.7
16	Oesophagus cancer	0.8	0.2	3.4	Cervical cancer	0.7	0.1	6.1
17	Colorectal cancer	0.7	0.7	1.0	Colorectal cancer	0.7	0.6	1.3
18	Mouth cancers	0.7	0.2	4.1	Low birth weight	0.7	0.2	2.8
19	Prostate cancer	0.7	0.6	1.1	Type 1 diabetes	0.6	0.1	10.2
20	Suffocation & foreign bodies	0.7	0.1	4.7	Inflammatory heart disease	0.6	0.1	5.1

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

4.4 Years lived with disability

Years lived with disability (YLD), or non-fatal burden, is typically the number of years of healthy life lost due to disability accrued into the future from incident cases of disease in the base year. An alternative method uses prevalent cases. Prevalent non-fatal burden (PYLD) is interpreted as the number of years of life 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 study, references to non-fatal burden reflect incident non-fatal burden unless otherwise specified.

4.4.1 Incident YLD

Incident YLD accounted for 46% of total Indigenous Australian disease burden (Figure 4.3). The absolute non-fatal burden peaked in young adulthood for Indigenous Australians, dropping off considerably after 40 years of age (Figure 4.6). For Australian females, absolute non-fatal burden peaked in young adulthood and was reasonably steady to old age. For males, there were peaks in young adulthood, then a decline and steady rise through the middle ages, before the absolute non-fatal burden decreased in the oldest ages. Again, while the absolute number of YLD was considerably smaller for Indigenous Australians compared with the total Australian population, the DALY rate was higher at every age.

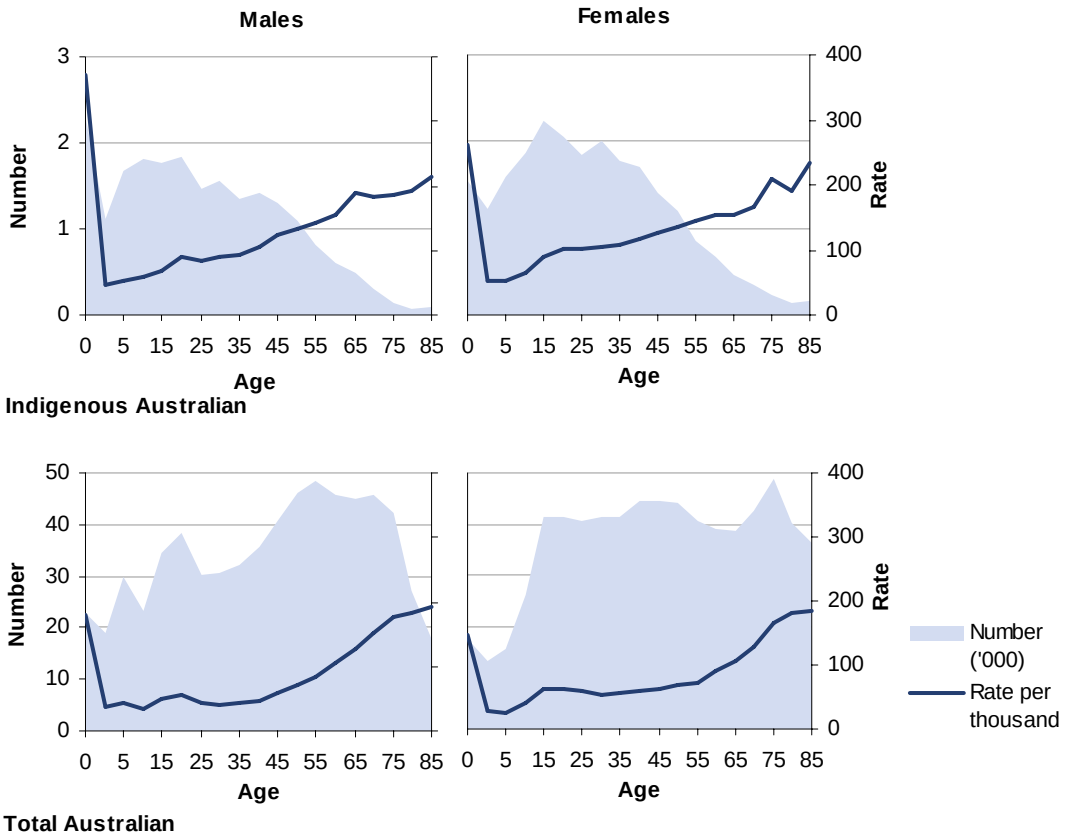


Figure 4.6: Incident YLD by age and sex expressed as rates, and numbers, Indigenous Australian and total Australian populations, 2003

Anxiety & depression, Type 2 diabetes, and asthma were the leading causes of incident non-fatal burden for male and female Indigenous Australians (Table 4.8). The top five conditions accounted for 38.9% and 46.7% of male and female non-fatal burden respectively.

Table 4.8: Leading causes of incident YLD by sex, Indigenous Australians, 2003

	Males			Females		
Rank	Condition	YLD	Per cent of total	Condition	YLD	Per cent of total
	All Causes	**2,1202**	**100.0**	**All Causes**	**23,299**	**100.0**
1	Anxiety & depression	2,855	13.5	Anxiety & depression	4,582	19.7
2	Type 2 diabetes	2,183	10.3	Type 2 diabetes	2,626	11.3
3	Asthma	1,262	6.0	Asthma	1,718	7.4
4	COPD	1,077	5.1	Ischaemic heart disease	1,080	4.6
5	Ischaemic heart disease	872	4.1	COPD	872	3.7
6	Schizophrenia	695	3.3	Deficiency anaemia	619	2.7
7	Alcohol dependence & harmful use	672	3.2	Schizophrenia	558	2.4
8	Otitis media	515	2.4	Otitis media	493	2.1
9	Heroin or polydrug dependence	512	2.4	Stroke	481	2.1
10	Low birth weight	417	2.0	Migraine	426	1.8
11	Stroke	394	1.9	Dental caries	405	1.7
12	Dental caries	388	1.8	STDs (not HIV/AIDS)	373	1.6
13	Adult-onset hearing loss	370	1.7	Personality disorders	309	1.3
14	ADHD	324	1.5	Low birth weight	299	1.3
15	Birth trauma & asphyxia	316	1.5	Homicide & violence	293	1.3
16	Personality disorders	307	1.4	Heroin or polydrug dependence	264	1.1
17	Homicide & violence	300	1.4	Peripheral vascular disease	263	1.1
18	Neonatal infections	285	1.3	Alcohol dependence & harmful use	250	1.1
19	Peripheral vascular disease	280	1.3	Infertility	211	0.9
20	Cannabis dependence	243	1.1	Back pain	210	0.9

For most of the top 20 leading causes of the Indigenous Australian non-fatal burden, Indigenous Australians experienced a higher rate than the total Australian population (Table 4.9). Both male and female Indigenous Australians experienced a much higher rate of non-fatal burden from homicide & violence (RR 6.1 males; RR 15.5 females), and ischaemic heart disease (RR 5.0 males; RR 6.2 females) compared with the total Australian population.

Table 4.9: Rate per 1,000 and rate ratio of top 20 leading causes of incident YLD, Indigenous Australian and total Australian populations, 2003

	Males				Females			
Rank	Condition	Indigenous Australian	Total Australian[(a)]	RR[(b)]	Condition	Indigenous Australian	Total Australian[(a)]	RR[(b)]
	All causes	**89.9**	**52.7**	**1.7**	**All causes**	**97.3**	**54.7**	**1.8**
1	Anxiety & depression	12.1	7.3	1.7	Anxiety & depression	19.1	13.9	1.4
2	Type 2 diabetes	9.3	2.8	3.3	Type 2 diabetes	11.0	2.6	4.2
3	Asthma	5.4	4.7	1.1	Asthma	7.2	4.7	1.5
4	COPD	4.6	1.2	3.7	Ischaemic heart disease	4.5	0.7	6.2
5	Ischaemic heart disease	3.7	0.7	5.0	COPD	3.6	0.9	4.1
6	Schizophrenia	2.9	1.8	1.6	Deficiency anaemia	2.6	0.4	6.2
7	Alcohol dependence & harmful use	2.8	1.5	1.9	Schizophrenia	2.3	1.3	1.7
8	Otitis media	2.2	0.4	5.5	Otitis media	2.1	0.3	6.1
9	Heroin or polydrug dependence	2.2	0.9	2.4	Stroke	2.0	0.9	2.3
10	Low birth weight	1.8	0.7	2.5	Migraine	1.8	1.9	0.9
11	Stroke	1.7	1.0	1.6	Dental caries	1.7	0.5	3.1
12	Dental caries	1.6	0.5	3.1	STDs (not HIV/AIDS)	1.6	0.2	8.2
13	Adult-onset hearing loss	1.6	1.7	0.9	Personality disorders	1.3	1.3	1.0
14	ADHD	1.4	1.4	1.0	Low birth weight	1.2	0.7	1.8
15	Birth trauma & asphyxia	1.3	0.6	2.1	Homicide & violence	1.2	0.1	15.5
16	Personality disorders	1.3	1.4	1.0	Heroin or polydrug dependence	1.1	0.3	3.2
17	Homicide & violence	1.3	0.2	6.1	Peripheral vascular disease	1.1	0.2	5.6
18	Neonatal infections	1.2	0.3	4.8	Alcohol dependence & harmful use	1.0	0.4	2.7
19	Peripheral vascular disease	1.2	0.3	3.9	Infertility	0.9	0.9	1.0
20	Cannabis dependence	1.0	0.5	1.9	Back pain	0.9	0.9	0.9

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

4.4.2 Prevalent YLD

The difference between prevalent and incident non-fatal burden was most apparent for childhood conditions (e.g. 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 was much larger compared with prevalent non-fatal burden, because most incident cases of chronic conditions at young ages were expected to remain prevalent cases at older ages. This explained the shift to the right in the figures of prevalent non-fatal burden (Figure 4.7) compared with incident non-fatal burden (Figure 4.6).

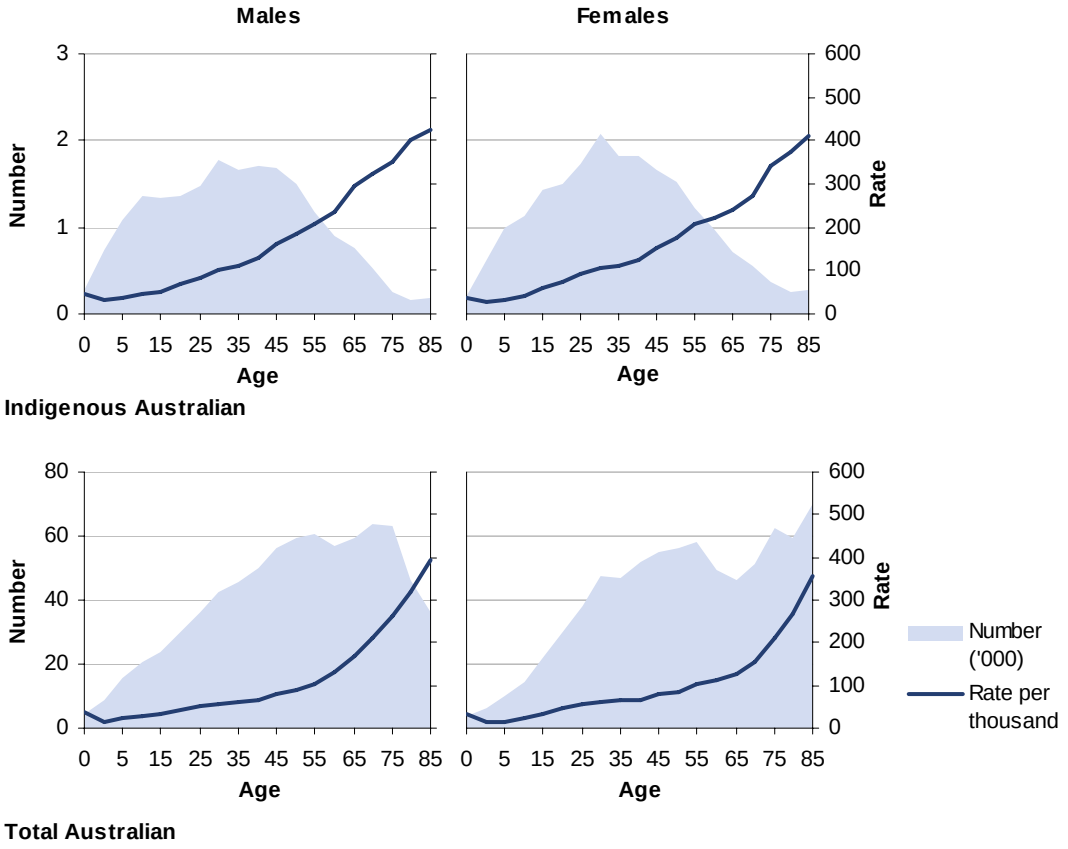


Figure 4.7: Prevalent YLD by age and sex expressed as rates, and numbers, Indigenous Australian and total Australian populations, 2003

4.5 Mortality and life expectancy

Mortality in young and middle-aged Indigenous adults was particularly high (33% and 23% probability of dying between ages 15 and 60 years in males and females, respectively, compared with 10% and 6% in the total Australian population). A comparable high level of adult mortality is found in only a few countries in the world that are not severely affected by HIV/AIDS mortality. Under-five mortality in Indigenous Australians was also greater than that of the total Australian population but differences were less extreme than they were for adult mortality. The probability of dying before age 5 was 1.6% and 1.4% for males and females, respectively (compared with national figures of 0.7% and 0.6%).

For the purpose of this report we estimated the life expectancy at birth in Indigenous Australians for the period 1996 to 2001 to be 64 years for males and 69 years for females, a gap of 12.5 and 13.5 years with life expectancy of the total Australian population, respectively. These life expectancy figures are higher than those reported by the ABS for the same period. There is a scientific debate about the validity of either set of estimates that can only be resolved when new and better data and methods become available. We have endeavoured to make our estimation process as available, transparent and as scientifically rigorous as possible, including having our work peer-reviewed internationally (Hill et al. 2007), to help advance this very important area of Indigenous health. If the ABS mortality figures had been adopted, the total Indigenous population burden of disease estimates would have been greater (since the ABS estimates a larger gap in life expectancy for the total

Indigenous Australian population), with DALY rates comparable to those presented in chapter six for Indigenous people residing in remote areas.

Table 4.10: Life expectancy at birth and probability of dying before age 5 and between ages 15 and 60, Indigenous Australian and total Australian populations, 1996–2001

	Life expectancy (years)		Probability of dying before age 5		Probability of dying between ages 15 and 60	
Area	Males	Females	Males	Females	Males	Females
Indigenous Australian population	64	69	0.016	0.014	0.326	0.231
Total Australian population	77	82	0.007	0.006	0.101	0.057

4.6 Specific disease and injury categories

This section discusses the seven leading broad cause categories in greater detail. These categories together accounted for more than 70% of the total disease burden, three-quarters of the fatal burden, and two-thirds of the non-fatal burden in Indigenous Australians (Table 4.11). In this section, we discuss broad cause categories intentional and unintentional injuries together.

Table 4.11: YLD, YLL and DALYs for top ten broad cause groups, Indigenous Australian population, 2003

Rank	Cause	YLD	Per cent of total	YLL	Per cent of total	DALY	Per cent of total
	All causes	**44,501**	**100.0**	**51,475**	**100.0**	**95,976**	**100.0**
1	Cardiovascular disease	4,214	9.5	12,573	24.4	16,786	17.5
2	Mental disorders	12,335	27.7	2,525	4.9	14,860	15.5
3	Chronic respiratory disease	5,816	13.1	2,771	5.4	8,587	8.9
4	Diabetes	4,946	11.1	3,552	6.9	8,498	8.9
5	Cancers	466	1.0	7,351	14.3	7,817	8.1
6	Unintentional injuries	1,464	3.3	5,524	10.7	6,989	7.3
7	Intentional injuries	622	1.4	4,774	9.3	5,395	5.6
8	Nervous system and sense organ disorders	2,629	5.9	1,485	2.9	4,114	4.3
9	Neonatal causes	1,668	3.7	2,379	4.6	4,047	4.2
10	Infectious and parasitic diseases	1,682	3.8	2,114	4.1	3,796	4.0
	Other	8,660	19.5	6,427	12.5	15,087	15.7

4.6.1 Cardiovascular disease

Cardiovascular disease was the leading broad cause category, responsible for 17.5% of disease burden in Indigenous Australians in 2003 (Table 4.11). This was a similar proportion to that seen in the total Australian population (18.0%). Ischaemic heart disease and stroke dominated the cause group, accounting for more than three-quarters of the cardiovascular burden. Cardiovascular burden was largely fatal, with premature mortality causing 75% of the total burden (Figure 4.8). Males contributed to 55% of the total cardiovascular burden.

However, of the top four specific cardiovascular diseases, the burden of rheumatic heart disease is higher in females.

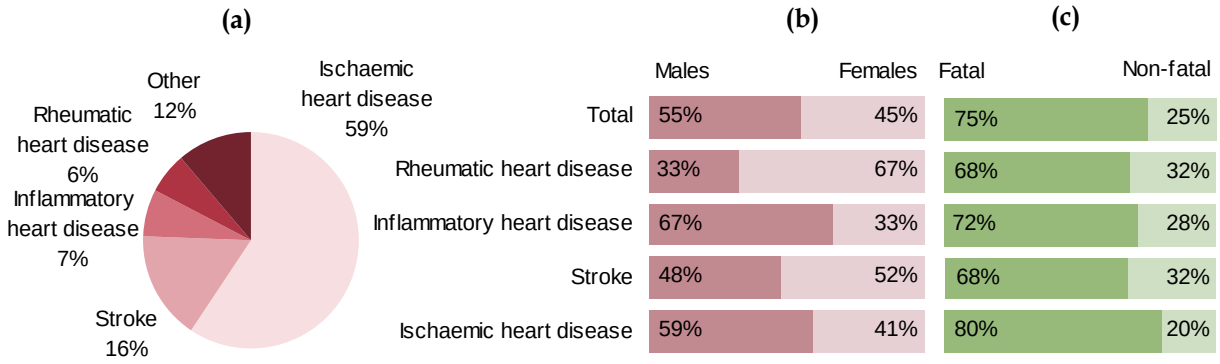


Figure 4.8: Cardiovascular disease DALYs by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

Ischaemic heart disease alone contributed to 10.4% of the total disease burden (Table 4.12). The cardiovascular burden was 4.6 times higher among Indigenous Australians than the total Australian population. At the disease level, the rate ratio was lowest for stroke, and highest for rheumatic heart disease.

Table 4.12: Cardiovascular disease DALYs by specific cause, Indigenous Australian and total Australian populations, 2003

	Indigenous Australian			Total Australian			
Cause	**DALY**	**Per cent of total DALY**	**DALY rate per 1,000**	**DALY**	**Per cent of total DALY**	**DALY rate per 1,000[a]**	**RR[b]**
Ischaemic heart disease	9,973	10.4	21.0	263,497	10.0	3.9	5.4
Stroke	2,706	2.8	5.7	118,462	4.5	2.1	2.8
Inflammatory heart disease	1,193	1.2	2.5	15,904	0.6	0.4	5.9
Rheumatic heart disease	984	1.0	2.1	4 ,091	0.2	0.1	25.1
Other	1,931	2.0	4.1	71,840	2.7	1.2	3.4
Total cardiovascular burden	**16,786**	**17.5**	**35.3**	**473,794**	**18.0**	**7.6**	**4.6**

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

4.6.2 Mental disorders

Mental disorders caused 15.5% of the total disease burden experienced by Indigenous Australians in 2003 (Table 4.11); with anxiety & depression, alcohol dependence & harmful use, and schizophrenia contributing more than three-quarters to this burden (Figure 4.9). Overall, the burden of mental disorders was equally distributed between males and females; however, anxiety & depression was more common in females, while males experienced more substance use and schizophrenia. More than 80% of the mental disorder burden was non-

fatal; although, the burden from alcohol dependence & harmful use had a large fatal component.

The fatal component of the substance use categories only included those deaths directly coded to that use, thereby excluding, for example, injury deaths where alcohol was a contributing factor. In the risk factor analysis, we included all alcohol- and illicit drug-related deaths (see Chapter 5).

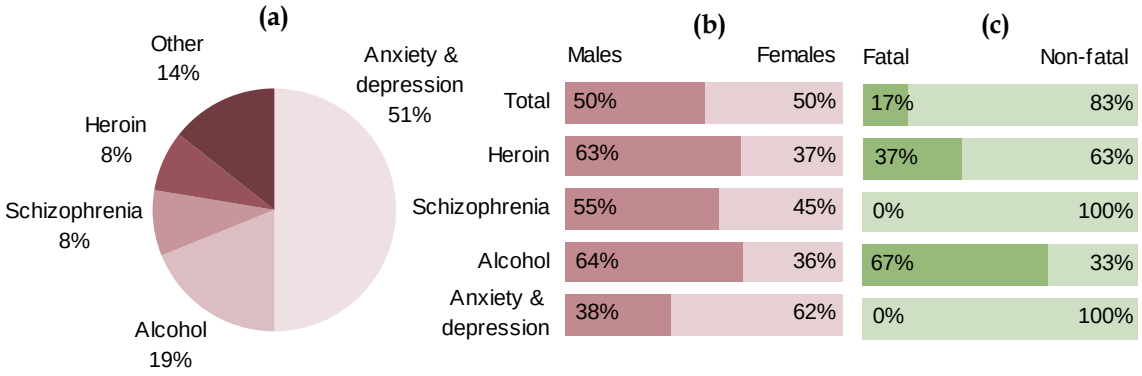


Figure 4.9: Mental disorder DALYs by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

The burden due to mental disorders occurred in Indigenous Australians at 1.6 times the rate of the total Australian population (Table 4.13); with alcohol dependence & harmful use 4.5 times the total Australian population rate.

Table 4.13: Mental disorder DALYs by specific cause, Indigenous Australian and total Australian populations, 2003

	Indigenous Australian			Total Australian			
Cause	**DALY**	**Per cent of total DALY**	**DALY rate per 1,000**	**DALY**	**Per cent of total DALY**	**DALY rate per 1,000[a]**	**RR[b]**
Anxiety & depression	7,446	7.8	15.7	191,786	7.3	10.5	1.5
Alcohol abuse	2,805	2.9	5.9	34,116	1.3	1.3	4.5
Schizophrenia	1,253	1.3	2.6	27,502	1.0	1.6	1.7
Heroin or polydrug dependence	1,223	1.3	2.6	16,839	0.6	1.0	2.7
Other	2,133	2.2	4.5	80,303	3.1	5.2	0.9
Total mental disorder burden	**14,860**	**15.5**	**31.3**	**350,545**	**13.3**	**19.6**	**1.6**

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

4.6.3 Injuries

Together, intentional and unintentional injuries were the third leading broad cause of Indigenous Australian disease burden, causing 12.9% of total burden (5.6% and 7.3% respectively) (Table 4.11). In comparison, injuries were responsible for 7.0% of the disease burden in the total Australian population. Suicide, RTAs, and homicide & violence contributed to more than two-thirds of the Indigenous Australian injury burden (Figure

4.10). Overall, injuries were much more common in males. However, homicide & violence had an almost equal distribution between the sexes. Among the top six specific injury categories, falls was the only category dominated by non-fatal burden, while 83% of the overall injury burden was due to mortality.

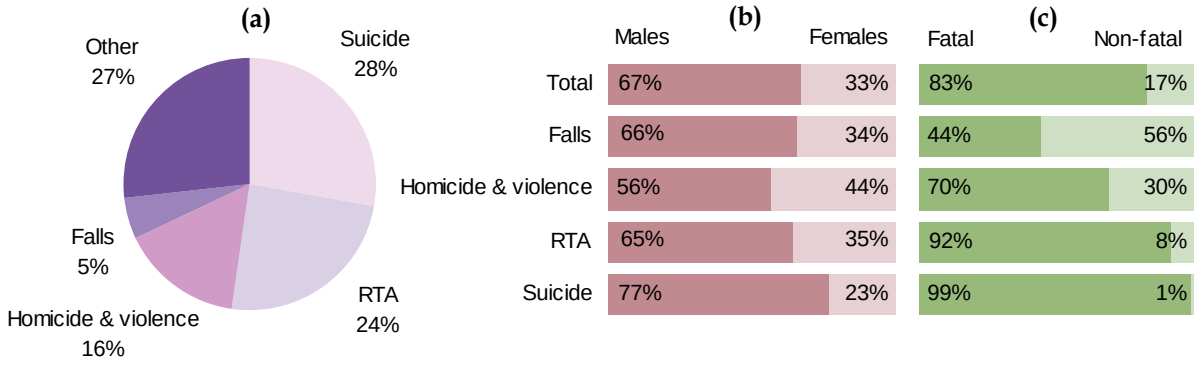


Figure 4.10: Injury DALYs by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

Overall, the injury burden rate was three times higher in Indigenous Australians compared with the total Australian population (Table 4.14); with homicide & violence 8.6 times the total Australian rate.

Table 4.14: Injury DALYs by specific cause, Indigenous Australian and total Australian populations, 2003

	Indigenous Australian			Total Australian			
Cause	**DALY**	**Per cent of total DALY**	**DALY rate per 1,000**	**DALY**	**Per cent of total DALY**	**DALY rate per 1,000[(a)]**	**RR[(b)]**
Suicide	3,439	3.6	7.2	49,916	1.9	2.2	3.2
RTAs	3,030	3.2	6.4	42,425	1.6	2.3	2.8
Homicide & violence	1,956	2.0	4.1	9,221	0.4	0.5	8.1
Falls	655	0.7	1.4	26,386	1.0	0.8	1.7
Other	3,304	3.4	7.0	57,101	2.2	2.8	2.4
Total injury burden	**12,384**	**12.9**	**26.1**	**185,050**	**7.0**	**8.6**	**3.0**

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

4.6.4 Chronic respiratory diseases

Chronic respiratory diseases were responsible for 8.9% of the total disease burden in Indigenous Australians in 2003 (Table 4.11). Chronic obstructive pulmonary disease and asthma caused 43% and 38% of this burden respectively (Figure 4.11). Males experienced the majority of burden due to COPD, while females experienced the majority of asthma burden. The burden due to asthma was largely non-fatal, with mortality only contributing 10%.

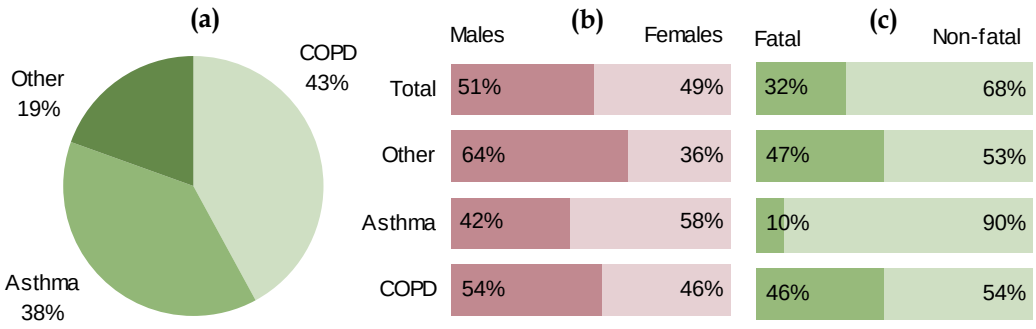


Figure 4.11: Chronic respiratory disease DALYs by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

The burden from COPD and other chronic respiratory diseases occurred at a higher rate in Indigenous Australians than in the total Australian population (Table 4.15). The largest differentials occurred in COPD and other chronic respiratory diseases.

Table 4.15: Chronic respiratory disease DALYs by specific cause, Indigenous Australian and total Australian populations, 2003

	Indigenous Australian			Total Australian			
Cause	**DALY**	**Per cent of total DALY**	**DALY rate per 1,000**	**DALY**	**Per cent of total DALY**	**DALY rate per 1,000[(a)]**	**RR[(b)]**
COPD	3,619	3.8	7.6	86,751	3.3	1.7	4.5
Asthma	3,303	3.4	6.9	63,100	2.4	4.8	1.4
Other	1,664	1.7	3.5	36,887	1.4	0.6	5.8
Total chronic respiratory disease burden	**8,587**	**8.9**	**18.1**	**186,737**	**7.1**	**7.1**	**2.5**

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

4.6.5 Diabetes

Diabetes was responsible for 8.9% of the disease burden in Indigenous Australians in 2003 (Table 4.11); with Type 2 diabetes accounting for 93% (Figure 4.12). Females experienced the majority of burden due to diabetes, and 58% of the burden was non-fatal.

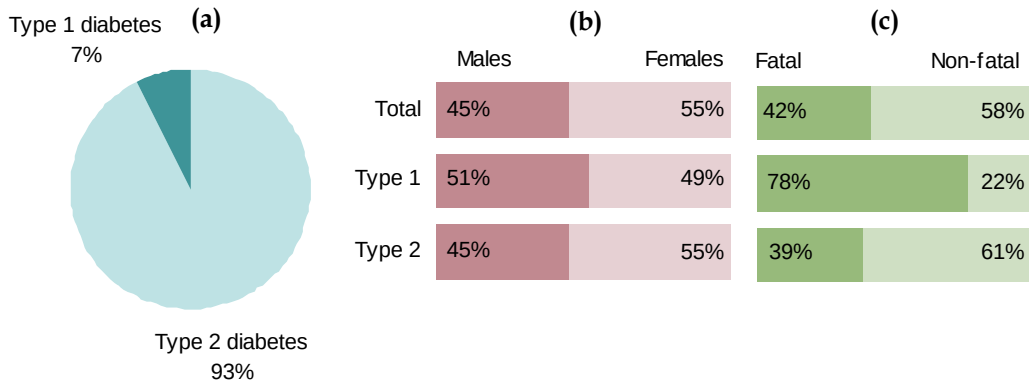


Figure 4.12: Diabetes DALYs by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

Overall, the burden due to diabetes occurred in Indigenous Australians at 5.1 times the rate experienced by the total Australian population (Table 4.16). While we have assumed a similar incidence of Type 1 diabetes in Indigenous and total Australian populations, the rate of burden is elevated by a factor three due to differences in YLL. This may reflect a higher case fatality of Type 1 diabetes in Indigenous Australians but may also be due to coding errors of Type 1 diabetes deaths.

Table 4.16: Diabetes DALYs by specific cause, Indigenous Australian and total Australian populations, 2003

	Indigenous Australian			Total Australian			
Cause	DALY	Per cent of total DALY	DALY rate per 1,000	DALY	Per cent of total DALY	DALY rate per 1,000[(a)]	RR[(b)]
Type 2 diabetes	7,880	8.2	16.6	132,940	5.0	3.1	5.4
Type 1 diabetes	618	0.6	1.3	10,891	0.4	0.4	3.0
Total diabetes burden	**8,498**	**8.9**	**17.9**	**143,831**	**5.5**	**3.5**	**5.1**

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

4.5.6 Cancers

Cancer was responsible for 8.1% of the total disease burden in Indigenous Australians in 2003 (Table 4.11). This was considerably lower than the 19.7% seen in the total Australian population. Despite this, the rate of burden due to cancer was 1.7 times higher in the Indigenous population compared to the total Australian population (Table 4.17). Lung cancer, followed by breast cancer, contributed the most (Figure 4.13). Females experienced 54% of the cancer burden; however, in the total Australian population, females experienced 47% of the cancer burden. Almost 94% of the burden due to cancer in Indigenous Australians was due to mortality.

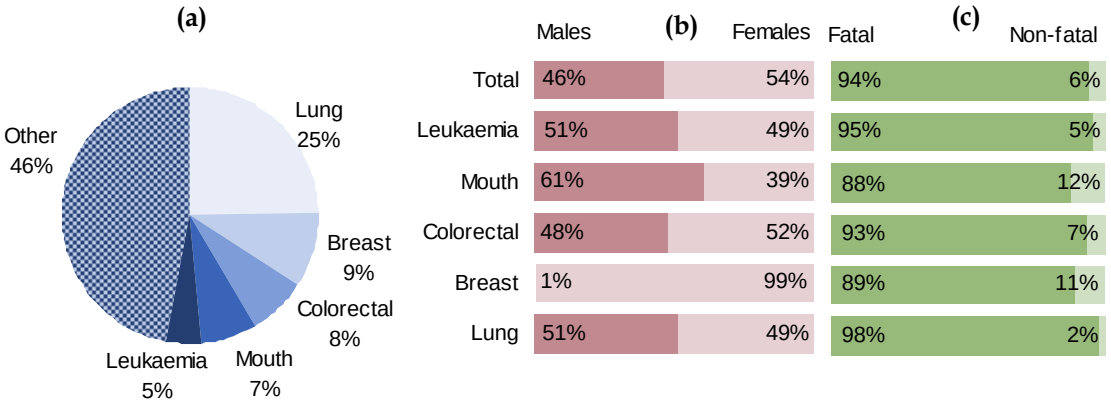


Figure 4.13: Cancer DALYs by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

The burden due to mouth & oropharynx cancer, and lung cancer among Indigenous Australians was 3.8 and 2.7 times as high as the total Australian population (Table 4.17).

Table 4.17: Cancer DALYs by specific cause, Indigenous Australian and total Australian populations, 2003

	Indigenous Australian			Total Australian			
Cause	**DALY**	**Per cent of total DALY**	**DALY rate per 1,000**	**DALY**	**Per cent of total DALY**	**DALY rate per 1,000[a]**	**RR[b]**
Lung cancer	1,940	2.0	4.1	88,904	3.4	1.5	2.7
Breast cancer	725	0.8	1.5	60,654	2.3	1.4	1.1
Colorectal cancer	601	0.6	1.3	63,605	2.4	1.1	1.1
Mouth & oropharynx cancers	530	0.6	1.1	13,464	0.5	0.3	3.8
Leukaemia	358	0.4	0.8	19,956	0.8	0.5	1.4
Other	3,663	3.8	7.7	252,833	9.6	5.0	1.5
Total cancer burden	**7,817**	**8.1**	**16.4**	**499,416**	**19.0**	**9.8**	**1.7**

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

4.5.7 Alternative categories

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 was divided amongst other chromosomal disorders, Down syndrome, low birth weight, infection, epilepsy, other perinatal conditions, autism and foetal alcohol syndrome. 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, trachoma, and other causes of vision loss.

Indigenous Australian males experienced the majority of the intellectual disability burden, while females experienced the majority of vision loss burden (Figure 4.14). The burden from

renal failure is overwhelmingly from years of life lost, while vision loss resulted mainly in non-fatal burden.

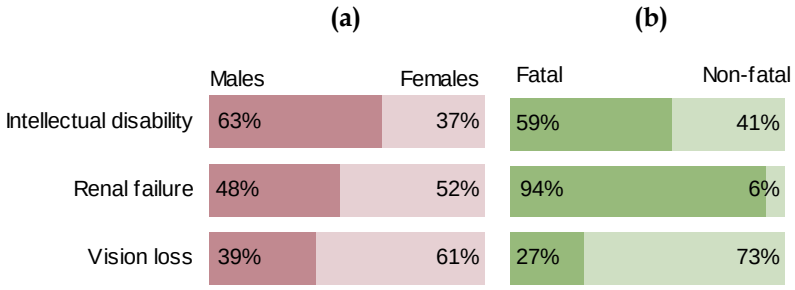


Figure 4.14: Alternative category DALYs by specific cause expressed as (a) proportions by sex; (b) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

Indigenous Australians experienced renal failure at a much higher rate (7.6 times) than the total Australian population (Table 4.18).

Table 4.18: Alternative category DALYs, Indigenous Australian and total Australian populations, 2003

	Indigenous Australian			Total Australian			
Cause	DALY	Per cent of total DALY	DALY rate per 1,000	DALY	Per cent of total DALY	DALY rate per 1,000[(a)]	RR[(b)]
Intellectual disability	5,261	5.5%	11.1	44,187	1.7%	4.1	2.7
Renal failure	4,923	5.1%	10.4	68,721	2.6%	1.4	7.6
Vision loss	859	0.9%	1.8	55,661	2.1%	1.3	1.3

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

5 Risks to health in Indigenous Australians

5.1 Overview

This chapter discusses the contribution of a number of important health risks to the burden of disease and injury in Indigenous Australians in 2003. The choice of which risks to include was based on the following:

1. Good evidence of a causal association between the exposure to the risk and the health outcomes and available relative risk estimates from reputable epidemiological studies.
2. Reliable estimates of exposure to the risk factor for the Indigenous Australian population.
3. Importance of the risk factor to Indigenous health policy making as informed by this study's advisory committees.

The outcome of these considerations was a set of 11 selected health risks (Table 5.1). Lack of data on prevalence and/or outcome prevented the estimation of the burden of intimate partner violence in males, osteoporosis and occupation. Urban air pollution was excluded because of data uncertainty and the expectation that it would not be a very large contributor to the Indigenous burden of disease.

Table 5.1: Eleven selected risks to health discussed in this study

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

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

First, health risks tend to cluster around 'high-risk' individuals who experience more than one exposure (e.g. smokers tend to be drinkers). This combination of exposures may produce higher or lower levels of the combined risk as a result of complex interaction effects. The analyses presented in this chapter for individual factors 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.

Second, the causal paths between a number of related health risks and their eventual health outcomes are complicated. For example, physical inactivity can lead to obesity, which can cause high blood pressure 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, 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 was estimated to be 29.9% of total cardiovascular disease burden, while that for high body mass, high blood cholesterol and high blood pressure was 31.3%, 31.3% and 26.3% of cardiovascular disease, respectively (Table 5.1). The burden attributable to these health risks in combination, however, was not the sum of burden from each risk (that is, the combined burden was not an implausible 118.8%). This is because the combined effect of these risks must 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 11 risk factors, this 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 World Health Organization Comparative Risk Assessment project (Ezzati et al. 2004). Sensitivity analyses indicated that overall results based on this approach were relatively robust to the underlying assumptions; however, apportioning the combined overall risk back to each contributing risk factor was more difficult and was 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 the *Australian Burden of Disease and Injury 2003* study (Begg et al. 2007).

5.2 Combined effect of 11 selected risks to health

The 11 risk factors considered in this chapter together explained 37.4% of the total burden of disease experience of Indigenous Australians (Table 5.2). This indicates the potential to considerably reduce the disease and injury experience of Indigenous Australians with interventions targeted at these risk factors.

Eight of the risk factors were associated with cancer and together explained 48.5% of the total burden from this cause. In the total Australian population, the 14 risk factors considered (including air pollution, osteoporosis and occupational causes, which are risk factors that are not included in this study) explained 32.9% of the cancer burden. The major difference between the distribution of cancer burden among these risk factors is that a greater proportion of cancer was explained by tobacco in the Indigenous Australian population compared with the total Australian population (34.6% versus 21.0%).

The 10 risk factors associated with cardiovascular disease together explained 68.9% of this burden in Indigenous Australians. Tobacco contributed most to this cause, followed closely by high body mass, high blood cholesterol, physical inactivity and high blood pressure.

More than one-third of burden due to mental disorders was attributable to four of the risk factors under consideration. Alcohol, followed by illicit drugs, contributed the most to this burden.

Tobacco smoking was the only risk factor considered that was associated with neurological disorders (it has a small protective effect on Parkinson's disease).

Five of the risk factors were associated with injury burden and together explained 32.6% of the burden from this cause. This was similar to the proportion of injury burden explained by 14 risk factors in the total Australian population (31.7%) in relative terms, but as the burden of injuries in Indigenous Australians was much larger, it meant that the average risk of an

injury due to these risk factors was also much higher. Alcohol was by far the leading cause of injury burden in Indigenous Australians.

Due to the higher prevalence of tobacco smoking among Indigenous Australians, the proportion of burden due to cancer and cardiovascular disease explained by tobacco use was considerably higher in Indigenous Australians than in the total Australian population.

Table 5.2: Individual and joint DALYs attributable to 11 selected risk factors by broad cause group, Indigenous Australian population, 2003

	Broad cause group							
	Cancer	CVD	Mental	Neurological	Injury	Diabetes	Other	All causes
Total burden	7,817	16,786	14,860	4,114	12,384	8,498	31,517	95,976
Attributable burden (%)[(a)]								
Tobacco	34.6	33.0		–0.3	0.7		10.5	12.1
High body mass	3.2	31.3				63.2	0.1	11.4
Physical inactivity	4.7	29.9				31.2		8.4
High blood cholesterol		31.3						5.5
Alcohol								
Harmful effects	6.3	1.6	16.3		22.2		0.2	6.2
Beneficial effects		–4.8					>–0.1	–0.8
Net effects	6.3	–3.2	16.3		22.2		0.2	5.4
High blood pressure		26.3						4.6
Low fruit and vegetable intake	4.2	18.0						3.5
Illicit drugs		<0.1	12.9		3.6		2.8	3.4
Intimate partner violence	2.4	2.4	4.5	<0.1	7.5		0.9	2.6
Child sexual abuse	0.2	<0.1	6.7		2.7		0.1	1.4
Unsafe sex	4.5						2.6	1.2
11 risk factors combined[(b)]	**48.5**	**68.9**	**37.4**	**–0.3**	**32.6**	**68.8**	**16.2**	**37.4**

(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 5.1 for further details

The risk factors selected for this study had a different impact by sex and age (Table 5.3). Four risk factors attributed to disease burden of 0–14 year-olds, and together explained around 5% of total burden for this age group. Tobacco was the largest contributor to disease burden in this age group, due to the association between smoking during pregnancy and increased risk of having a low-birth weight child. Just over 30% of burden among males and females aged 15–34 years was explained by the 11 risk factors. Alcohol and illicit drugs contributed the most to burden among males at this age, while intimate partner violence was the largest contributor to female burden. More than half the burden among people aged 35 years and over was explained by the 11 risk factors. The leading cause of burden among males in the 35–54-year age group was tobacco and high body mass, which had an almost equal contribution. For females, high body mass was the largest contributor to burden in this age group, followed by tobacco. Tobacco was the largest contributor in males and females aged 55 years and over, followed by high body mass and physical inactivity.

Table 5.3: Individual and joint DALYs attributable to 11 selected risk factors by sex and age group, Indigenous Australian population, 2003

	Males					Females				
	0–14	15–34	35–54	55+	All ages	0–14	15–34	35–54	55+	All ages
Total burden	10,811	13,366	15,278	10,652	50,107	9,376	12,004	13,218	11,271	45,869
Attributable burden (%)[(a)]										
Tobacco	3.9	0.1	19.5	26.0	12.3	3.8	0.1	16.8	25.4	11.9
High body mass	–	3.2	19.2	16.1	10.1	–	3.9	23.5	20.1	12.7
Physical inactivity	–	3.5	13.4	15.4	8.3	–	3.8	12.8	15.3	8.5
High blood cholesterol	–	2.4	13.3	9.0	6.6	–	1.0	7.7	7.2	4.2
Alcohol										
Harmful effects	0.8	13.8	10.9	5.7	8.4	0.2	6.4	5.7	2.2	3.9
Beneficial effects	–	–0.5	–1.9	–1.7	–1.1	–	–0.2	–1.0	–1.1	–0.6
Net effects	0.8	13.3	8.9	4.1	7.3	0.2	6.2	4.7	1.1	3.3
High blood pressure	–	1.0	7.6	12.9	5.3	–	0.3	4.2	10.4	3.8
Low fruit and vegetable intake	–	1.6	7.3	7.6	4.3	–	1.0	4.3	4.7	2.6
Illicit drugs	0.5	11.0	3.3	1.0	4.3	0.4	5.7	2.5	0.6	2.4
Intimate partner violence	–	–	–	–	–	–	10.4	7.5	2.0	5.4
Child sexual abuse	–	1.6	0.8	0.2	0.7	–	5.7	2.4	0.4	2.3
Unsafe sex	0.0	0.8	0.8	0.2	0.5	0.4	3.1	2.9	1.1	2.0
11 risk factors combined[(b)]	**5.1**	**32.6**	**53.4**	**55.3**	**37.8**	**4.9**	**31.4**	**52.6**	**51.2**	**36.9**

(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 5.1 for further details

5.3 Individual contribution of 11 selected risks to health

Indigenous Australians experienced a higher rate of burden due to each of the 11 risk factors considered compared with the total Australian population (Table 5.4). This resulted from a combination of higher prevalence of exposure to the risk factors and higher disease levels in the population. The largest relative differences in rates of burden were for low fruit and vegetable consumption, tobacco, and high body mass.

Table 5.4: DALYs attributable to 11 selected risk factors by proportion of total DALY and rate, Indigenous Australian and total Australian populations, 2003

	Indigenous Australian			Total Australian			
Risk	DALY	% of total	Rate per 1,000	DALY	% of total	Rate per 1,000[a]	RR[b]
Tobacco	11,633	12.1	24.5	204,788	7.8	3.9	6.3
High body mass	10,919	11.4	23.0	197,632	7.5	4.2	5.5
Physical inactivity	8,032	8.4	16.9	174,431	6.6	3.1	5.4
High blood cholesterol	5,262	5.5	11.1	163,591	6.2	2.7	4.1
Alcohol							
Harmful effects	5,982	6.2	12.6	87,936	3.3	3.3	3.8
Beneficial effects	–811	–0.8	–1.7	–26,845	–1.0	–0.5	3.7
Net effects	5,171	5.4	10.9	61,091	2.3	2.9	3.8
High blood pressure	4,417	4.6	9.3	199,315	7.6	2.6	3.6
Low fruit & vegetable intake	3,344	3.5	7.0	55,259	2.1	1.0	7.0
Illicit drugs	3,264	3.4	6.9	51,463	2.0	2.4	2.9
Intimate partner violence	2,469	2.6	5.2	29,360	1.1	1.3	4.0
Child sexual abuse	1,390	1.4	2.9	23,513	0.9	1.1	2.7
Unsafe sex	1,174	1.2	2.5	14,897	0.6	0.5	4.7

(a) Age standardised to the Indigenous Australian population, 2003
(b) Indigenous Australian to total Australian rate ratio

5.3.1 Tobacco

Tobacco smoking was responsible for 12.1% of the total burden, and one-fifth of deaths in Indigenous Australians in 2003 (Table 5.5). It was the largest contributing risk factor overall for males, and the second largest contributor for females. Ischaemic heart disease, COPD, and lung cancer attributable to tobacco accounted for almost three-quarters of overall burden due to tobacco. Three-quarters of the burden attributed to tobacco smoking was due to mortality (Figure 5.1).

Indigenous Australian males experienced 53% of the burden from tobacco smoking compared with around two-thirds in the total Australian population. This difference most likely reflects a similarly high prevalence of smoking in Indigenous males and females in recent decades.

Tobacco-related illnesses with a short lag time between exposure and outcome (e.g. cardiovascular diseases) contributed more significantly to the burden in Indigenous Australians than the total Australian population. In the latter, lung cancer and COPD were the leading causes of tobacco burden. The recent favourable trends seen in the prevalence of tobacco smoking in the total Australian population have not occurred in the Indigenous Australian population. Unlike the total Australian population, we therefore do not expect to see any significant downward trend in tobacco smoking-related illnesses that have a long lag time (e.g. cancers and COPD) in the next few decades in Indigenous Australians, and there is evidence that the incidence of smoking-related cancers in Northern Territory Indigenous Australians is still increasing (Condon et al. 2005). However, when there is a reduction in prevalence of smoking, positive effects will been seen in the shorter term through a

reduction in the large burden of cardiovascular disease due to tobacco for which the lag time is a few years rather than decades.

Table 5.5: Deaths and DALYs attributable to tobacco by specific cause, Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Ischaemic heart disease	220	7.7	4,246	4.4
COPD	99	3.5	2,430	2.5
Lung cancer	116	4.0	1,780	1.9
Stroke	59	2.0	1,063	1.1
Low birth weight	13	0.4	632	0.7
Other	68	2.4	1,482	1.5
Total attributable	**574**	**20.0**	**11,633**	**12.1**

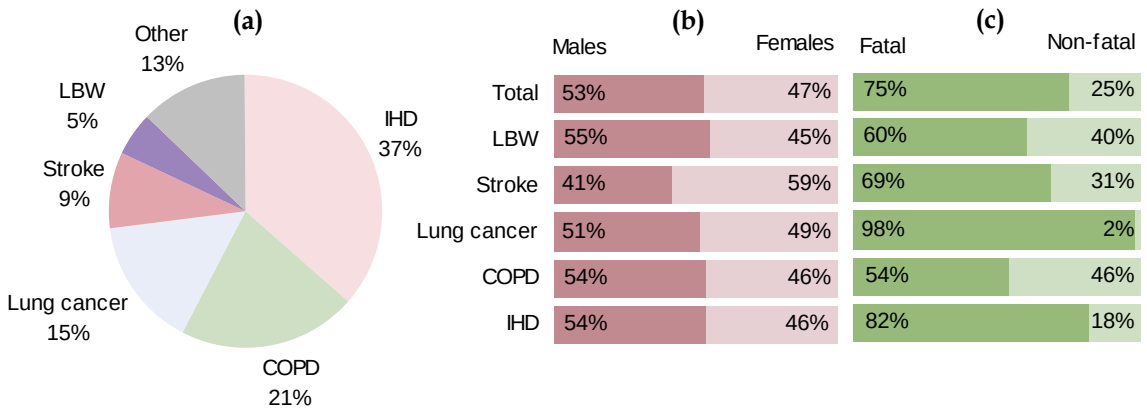


Figure 5.1: DALYs attributable to tobacco by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

5.3.2 High body mass

High body mass was responsible for 11.4% of the total Indigenous Australian burden of disease in 2003, and was the second leading cause of burden among the 11 risk factors examined (Table 5.6). For females aged 35–54 years, high body mass was responsible for the largest amount of burden among the 11 risks examined. Type 2 diabetes and ischaemic heart disease accounted for 89% of the total burden due to high body mass in Indigenous Australians. Fifty-three per cent of the burden was experienced by females, and 60% was due to mortality (Figure 5.2).

Table 5.6: Deaths and DALYs attributable to high body mass by specific cause, Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Type 2 diabetes	135	4.7	5,373	5.6
Ischaemic heart disease	195	6.8	4,359	4.5
Stroke	31	1.1	734	0.8
Other	25	0.9	453	0.5
Total attributable	**386**	**13.4**	**10,919**	**11.4**

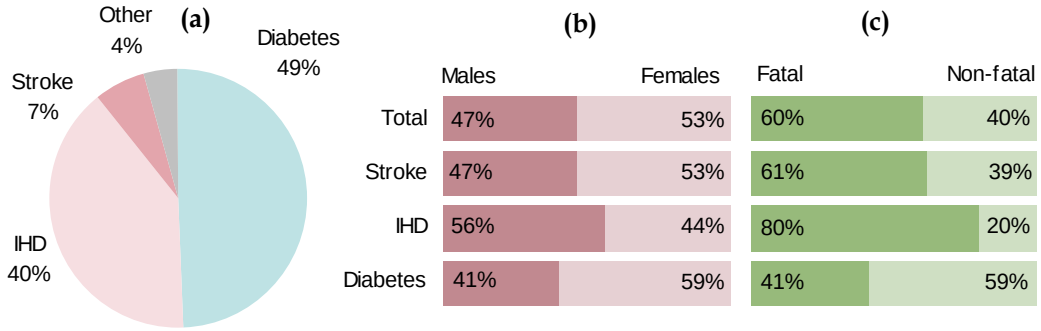


Figure 5.2: DALYs attributable to high body mass by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

5.3.3 Physical inactivity

Physical inactivity was responsible for 8.4% of the total Indigenous Australian burden in 2003 (Table 5.7), and was the third leading cause of burden among the 11 risk factors considered. Ischaemic heart disease and Type 2 diabetes together accounted for 88% of the burden due to this cause. Two-thirds of physical inactivity burden was due to mortality; however, 60% of Type 2 diabetes burden was non-fatal. Overall, Indigenous Australian males and females experienced an equal share of the burden due to physical inactivity (Figure 5.3).

Table 5.7: Deaths and DALYs attributable to physical inactivity by specific cause, Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Ischaemic heart disease	220	7.7	4,336	4.5
Type 2 diabetes	67	2.3	2,655	2.8
Stroke	36	1.3	675	0.7
Colorectal cancer	10	0.4	186	0.2
Breast cancer	9	0.3	180	0.2
Total attributable	**342**	**11.9**	**8,032**	**8.4**

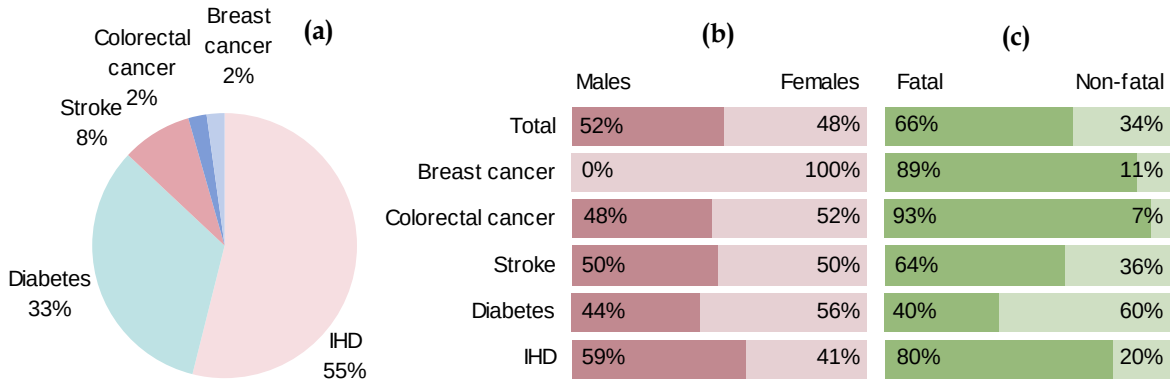


Figure 5.3: DALYs attributable to physical inactivity by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

5.3.4 High blood cholesterol

High blood cholesterol was responsible for 5.5% of the Indigenous Australian burden of disease in 2003 (Table 5.8). Ischaemic heart disease was responsible for 92% of the burden from high cholesterol, with stroke responsible for the remaining 8% of burden. The burden from ischaemic heart disease and stroke was largely due to mortality. Males experienced almost two-thirds of the burden from high blood cholesterol (Figure 5.4).

Table 5.8: Deaths and DALYs attributable to high blood cholesterol by specific cause, Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Ischaemic heart disease	221	7.7	4,862	5.1
Stroke	17	0.6	400	0.4
Total attributable	**238**	**8.3**	**5,262**	**5.5**

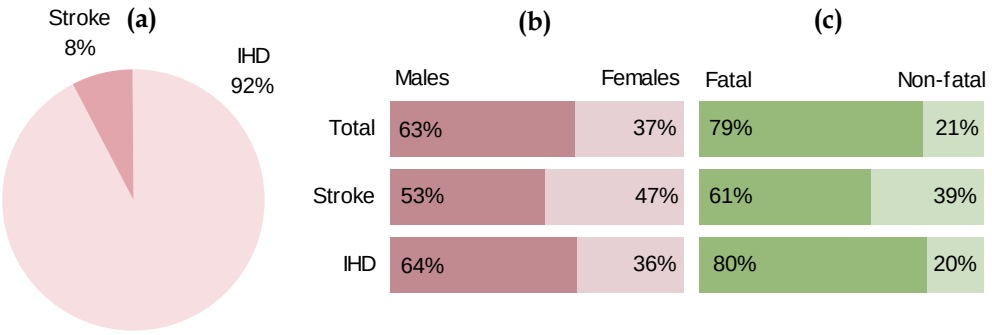


Figure 5.4: DALYs attributable to high blood cholesterol by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

5.3.5 Alcohol

Alcohol has both hazardous and protective effects on health. Among Indigenous Australian males aged 15–34 years, alcohol was responsible for the greatest amount of burden among the 11 risk factors considered (Table 5.3). For females in this age group, alcohol followed intimate partner violence as the second leading cause of burden.

Alcohol harm was responsible for 6.2%, and prevented 0.8% of the total burden in Indigenous Australians in 2003 (Figure 5.5). At each age, alcohol harm far outweighed its beneficial effects. Alcohol abuse and harmful use, homicide & violence, and suicide were the largest contributors to the harm caused by alcohol.

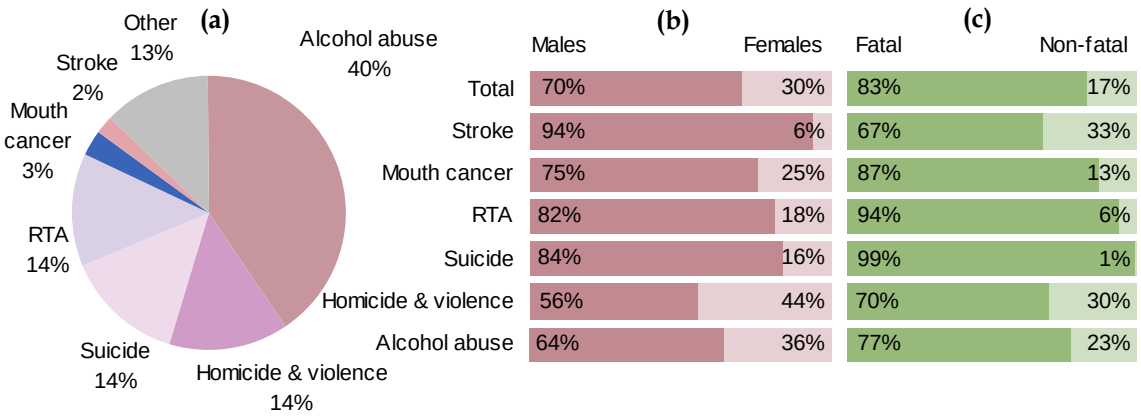


Figure 5.5: DALYs attributable to alcohol (*harm*) by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

Table 5.9: Deaths and DALYs attributable to alcohol by specific cause, Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Harm				
Alcohol abuse	93	3.3	2,419	2.5
Homicide & violence	23	0.8	841	0.9
Suicide	33	1.2	839	0.9
Road traffic accidents	30	1.0	811	0.8
Mouth & oropharynx cancers	9	0.3	168	0.2
Stroke	8	0.3	140	0.1
Other	37	1.3	765	0.8
Total attributable harm	*233*	*8.1*	*5,982*	*6.2*
Benefit				
Ischaemic heart disease	–36	–1.3	–731	–0.8
Stroke	–4	–0.1	–72	–0.1
Other	0	0.0	-8	0.0
Total attributable benefit	*–41*	*–1.4*	*–811*	*–0.8*
Total attributable	**192**	**6.7**	**5,171**	**5.4**

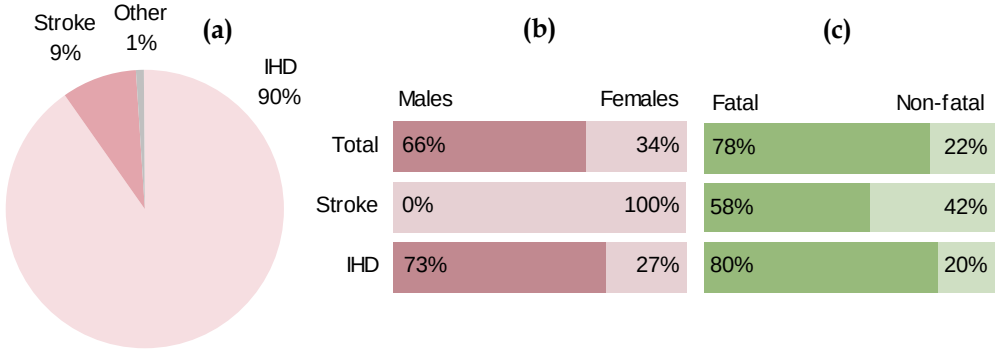


Figure 5.6: DALYs attributable to alcohol (*benefit*) by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

5.3.6 High blood pressure

High blood pressure was responsible for 4.6% of the total burden of disease, and 9.5% of deaths in Indigenous Australians in 2003 (Table 5.10). Ischaemic heart disease and stroke were the primary contributors. Males experienced 60% of the high blood pressure burden and 82% of the burden was due to mortality (Figure 5.7).

Table 5.10: Deaths and DALYs attributable to high blood pressure by specific cause, Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Ischaemic heart disease	184	6.4	3,040	3.2
Stroke	64	2.2	1051	1.1
Other	23	0.8	326	0.3
Total attributable	**272**	**9.5**	**4,417**	**4.6**

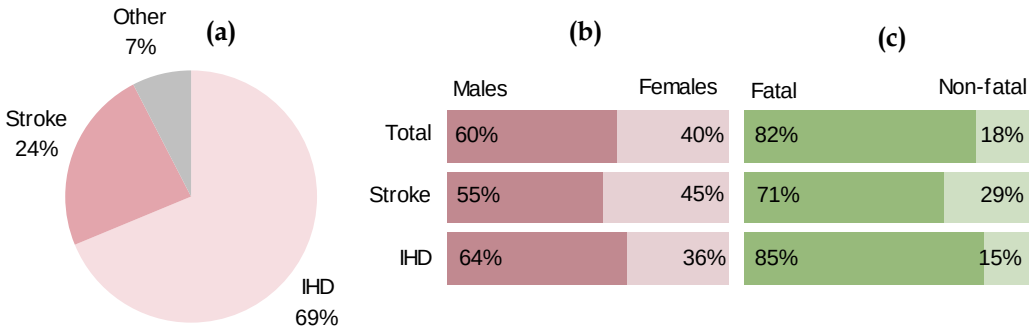


Figure 5.7: DALYs attributable to high blood pressure by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

5.3.7 Low fruit and vegetable consumption

Insufficient fruit and vegetable consumption contributed to 3.5% of the total burden of disease in Indigenous Australians in 2003 (Table 5.11). Ischaemic heart disease was the largest contributor to the burden from this risk factor. Overall, 81% of the burden from insufficient fruit and vegetable consumption was due to mortality, and 64% was experienced by males (Figure 5.8).

Table 5.11: Deaths and DALYs attributable to low fruit and vegetable consumption by specific cause, Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Ischaemic heart disease	130	4.5	2,744	2.9
Stroke	13	0.5	270	0.3
Lung cancer	13	0.5	214	0.2
Other	7	0.2	116	0.1
Total attributable	**163**	**5.7**	**3,344**	**3.5**

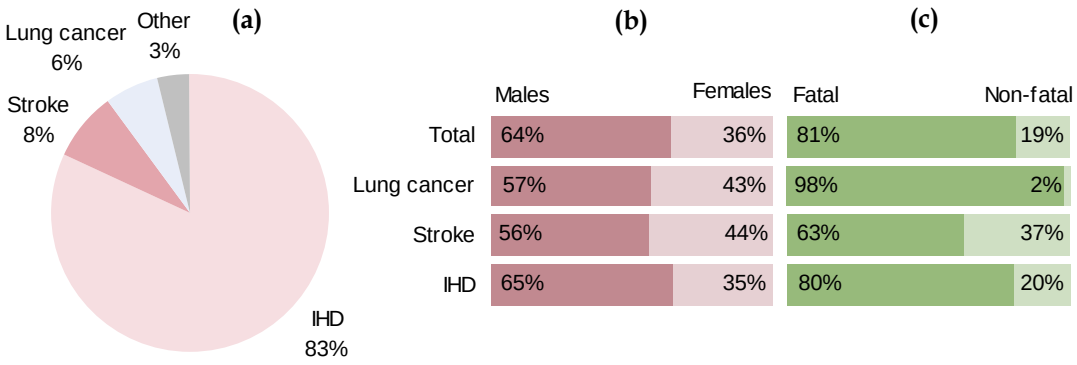


Figure 5.8: DALYs attributable to low fruit and vegetable consumption by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

5.3.8 Illicit drugs

Illicit drug use was responsible for 3.4% of the total burden in Indigenous Australians in 2003 (Table 5.12). Heroin or polydrug dependence was responsible for 37% of the burden due to illicit drugs. Two-thirds of the illicit drug burden was experienced by males, and 56% was due to mortality (Figure 5.9).

Table 5.12: Deaths and DALYs attributable to illicit drugs by specific cause, Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Heroin or polydrug dependence	17	0.6	1,216	1.3
Hepatitis C	28	1.0	554	0.6
Cannabis dependence	0	0.0	302	0.3
Suicide	12	0.4	299	0.3
Hepatitis B	10	0.3	215	0.2
Other	14	0.5	677	0.7
Total attributable	**80**	**2.8**	**3,264**	**3.4**

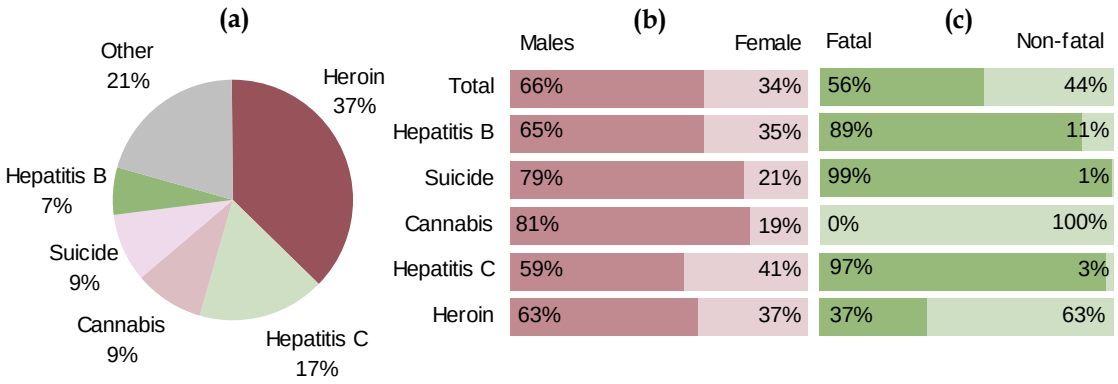


Figure 5.9: DALYs attributable to illicit drug use by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

5.3.9 Intimate partner violence

Due to a lack of data regarding male experience of intimate partner violence and the related health effects, we only attempted to quantify the proportion of female burden due to intimate partner violence.

Intimate partner violence was responsible for 2.6% of the total burden of disease in Indigenous Australians, and 5.4% of the burden among female Indigenous Australians in 2003 (Table 5.13). Homicide & violence, and anxiety & depression were both responsible for 25% of the burden from this risk factor. Just over half of the burden from intimate partner violence was due to mortality (53%) (Figure 5.10).

Table 5.13: Deaths and DALYs attributable to intimate partner violence by specific cause, female Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Homicide & violence	16	0.6	628	0.7
Anxiety & depression	0	0.0	606	0.6
Ischaemic heart disease	11	0.4	287	0.3
Suicide	10	0.4	277	0.3
Other	22	0.7	671	0.7
Total attributable	**58**	**2.0**	**2,469**	**2.6**

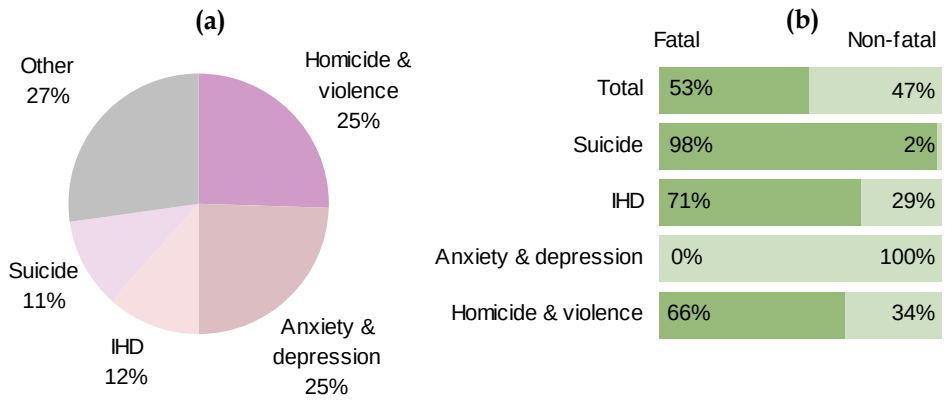


Figure 5.10: DALYs attributable to intimate partner violence by specific cause expressed as (a) proportions of total; (b) proportions due to fatal and non-fatal outcomes, female Indigenous Australian population, 2003

5.3.10 Child sexual abuse

We estimated the health outcomes in adulthood due to exposure to sexual abuse in childhood. Past exposure to child sexual abuse was responsible for 1.4% of the total burden of disease in Indigenous Australians (Table 5.14). Eighty-four per cent of this burden was from anxiety & depression, suicide, and alcohol dependence & harmful use. The burden from child sexual abuse was primarily experienced by females (75%) and was non-fatal (65%) (Figure 5.11). Caution is warranted in interpreting these estimates of the burden attributable to child sexual abuse as they are based on very limited data (see Annex table 3: Assessment of quality of risk factor exposure estimates in the Indigenous population).

Table 5.14: Deaths and DALYs attributable to child sexual abuse by specific cause, Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Anxiety & depression	0	0.0	812	0.8
Suicide	10	0.4	271	0.3
Alcohol abuse	4	0.1	91	0.1
Other	7	0.2	216	0.2
Total attributable	**21**	**0.7**	**1,390**	**1.4**

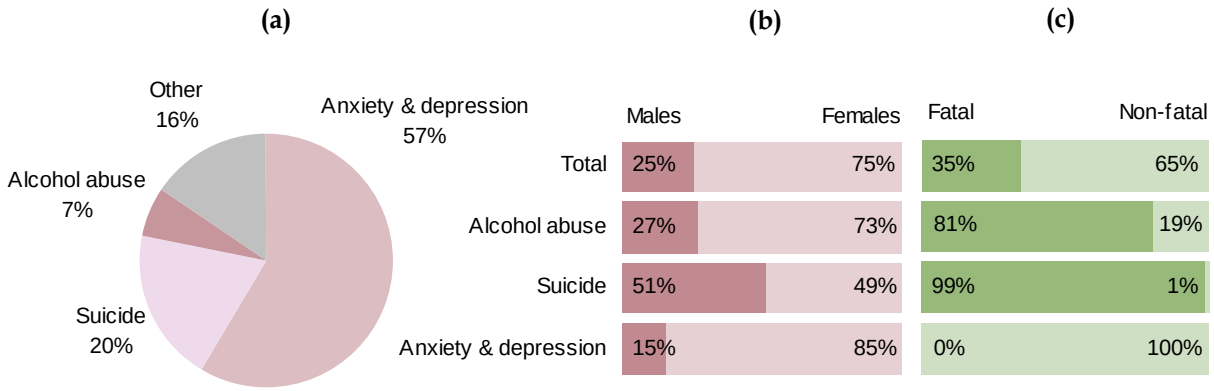


Figure 5.11: DALYs attributable to child sexual abuse by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

5.3.11 Unsafe sex

Unsafe sex was responsible for 1.2% of the total burden in Indigenous Australians in 2003 (Table 5.15). Cervical cancer, chlamydia, and HIV/AIDS accounted for 70% of the burden from this risk factor. The burden from unsafe sex was primarily experienced by females (79%), and was evenly split between fatal and non-fatal burden (Figure 5.12).

Table 5.15: Deaths and DALYs attributable to unsafe sex by specific cause, Indigenous Australian population, 2003

	Deaths		DALYs	
Specific cause	**Number**	**Per cent of total**	**Number**	**Per cent of total**
Cervical cancer	18	0.6	355	0.4
Chlamydia	1	0.0	267	0.3
HIV/AIDS	3	0.1	201	0.2
Other	8	0.3	351	0.4
Total attributable	**31**	**1.1**	**1,174**	**1.2**

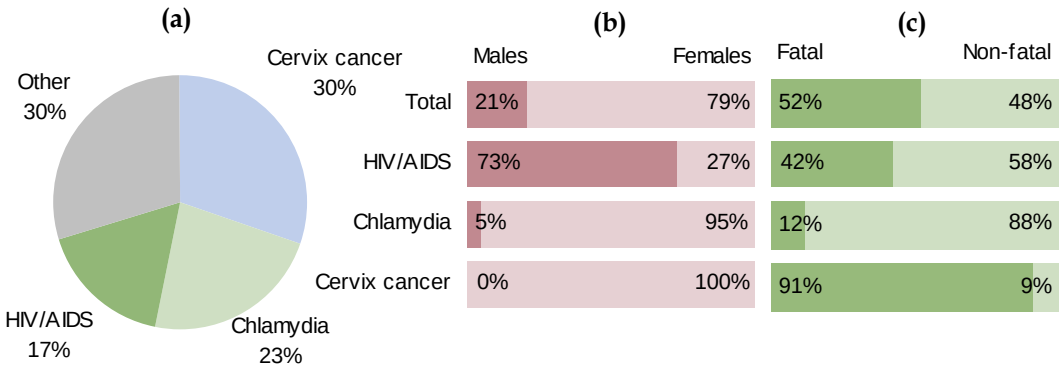


Figure 5.12: DALYs attributable to unsafe sex by specific cause expressed as (a) proportions of total; (b) proportions by sex; (c) proportions due to fatal and non-fatal outcomes, Indigenous Australian population, 2003

6 Indigenous health gap and differentials by remoteness

6.1 Overview

In this chapter we introduce the concept of the 'Indigenous health gap' — the difference between the current levels of disease burden in Indigenous Australians and what the disease burden in Indigenous Australians would be if National Study DALY rates applied. This gap indicates the potential for health gain. We also describe the differentials in burden of disease in Indigenous Australians by remoteness. We discuss these differentials in terms DALYs, risk factors, life expectancy, and HALE.

Indigenous Australians make up 2.4% of the total Australian population (Table 6.1). More than one-quarter of the Indigenous Australian population resides in remote areas (26.5%). Only 2.4% of the total Australian population resides in remote areas. The age structure of the non-remote and remote Indigenous Australian populations is similar, but is considerably younger than that of the total Australian population.

Table 6.1: Selected demographic characteristics of Indigenous Australians by remoteness and the total Australian population, 2003

		Proportions					
Population group	Count	Total	0–14 years	15–34 years	35–54 years	55+ years	Male
Indigenous Australians	475,395	2.4	38.1	34.4	20.5	7.0	49.6
Non-remote	349,600	1.8	39.0	34.1	20.3	6.6	49.4
Remote	125,795	0.6	35.7	35.5	20.9	7.9	50.3
Total Australian population	19,881,469	100.0	20.0	28.3	28.7	23.0	49.7

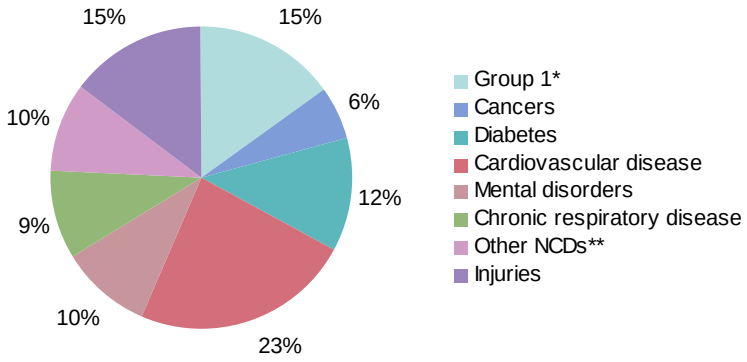
6.2 Indigenous health gap

A useful property of the DALY is that it lends itself to the analysis of which causes and age groups contribute most to the burden of disease or to the difference in health status between populations. Measures such as life expectancy and HALE (sections 6.5 and 6.6), while attractive for summarising the health of a population in a single measure, are less suited for describing underlying causes. We define the 'Indigenous health gap' as the difference between observed and 'achievable' DALYs. The 'achievable' burden is calculated by applying the total Australian rates of burden by age, sex and condition to the Indigenous Australian population. This gap between observed and 'achievable' is a useful measure for identifying health problems at different ages and thus where the greatest potential for Indigenous health gain exists.

6.2.1 Health gap by disease

If Indigenous Australians experienced the fatal and non-fatal burden rate of the total Australian population, a total of 56,455 DALYs could be avoided, equivalent to 59% of the burden of disease estimated for Indigenous Australians in 2003 (Table 6.2).

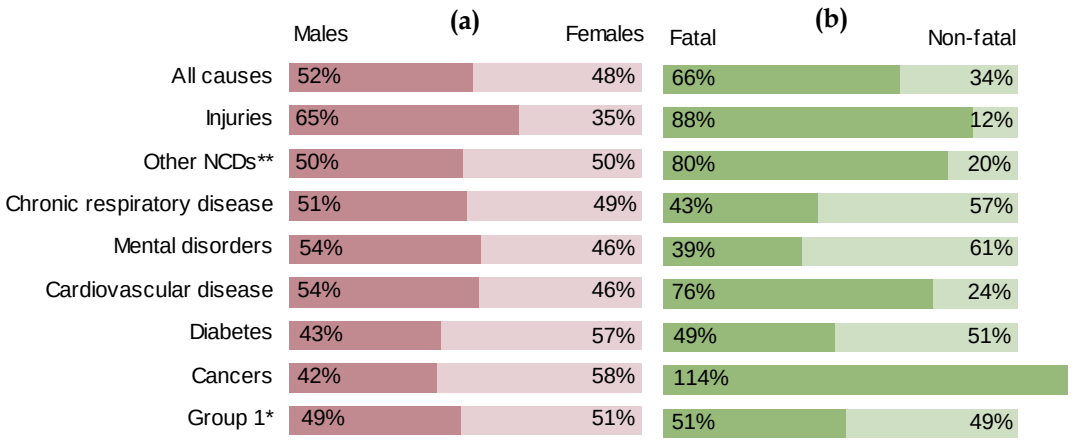
Almost one-quarter of the Indigenous health gap was due to cardiovascular diseases (Figure 6.1). Diabetes, mental disorders and chronic respiratory disease were responsible for a further 31% of the gap. Injuries and the group I conditions (communicable diseases, maternal and neonatal conditions) were each responsible for 15% of the gap. Infectious and parasitic diseases, acute respiratory infections and neonatal conditions contributed most to the gap in group I conditions (Table 6.2). The major contributors to the gap in injury burden were RTAs, suicide, and homicide & violence.



* Communicable diseases, maternal and neonatal conditions
** Other non-communicable diseases

Figure 6.1: Indigenous health gap (DALYs) by selected causes, 2003

Overall, the Indigenous health gap was equally distributed between males and females, and two-thirds of the gap was due to mortality (Figure 6.2). Mortality dominated the gap for cardiovascular disease, injuries and cancer. In fact, mortality explained more than 100% of the gap in cancer. This was because disability rates from cancer in Indigenous Australians were lower than the national rates by 14%, due to the higher case fatality rate in Indigenous people with cancer.



* Communicable diseases, maternal and neonatal conditions
** Other non-communicable diseases

Figure 6.2: Indigenous health gap (DALYs) by selected causes expressed as, (a) proportions by sex, and (b) proportions due to fatal and non-fatal outcomes, 2003

The largest proportion of the Indigenous health gap occurred in people aged 35–54 years (Table 6.2). Non-communicable diseases, particularly cardiovascular disease and diabetes, contributed most to the gap at these ages. The 15–34-year age group accounted for the second largest proportion of the gap. Even at these young adult ages, cardiovascular disease and diabetes contributed one-fifth to the gap although injuries and mental disorders were the largest contributors. Cardiovascular disease, diabetes and tobacco-related cancers and respiratory disease dominated the gap in the oldest age group. In children, group I conditions showed the greatest differences, particularly neonatal conditions — and, among these, low birth weight.

Table 6.2: Indigenous health gap (DALYs) due to selected causes, expressed as a proportion of total Indigenous health gap, 2003

Cause	Total (%)					Health gap (DALYs)	% of total Indigenous burden
	0–14	15–34	35–54	55+	Total		
All causes	**17**	**25**	**35**	**23**	**100**	**56,455**	**59**
Group I[(a)]	**8**	**3**	**3**	**1**	**15**	**8,633**	**9**
Infectious and parasitic diseases	1	1	2	1	5	3,074	3
Acute respiratory infections	2	1	1	1	4	2,358	2
Neonatal causes	4	0	0	0	4	2,354	2
Other group I	1	1	0	0	2	847	1
Non-communicable diseases	**8**	**13**	**28**	**21**	**70**	**39,491**	**41**
Cancers	0	0	2	3	6	3,151	3
Tobacco related cancers[(b)]	*0*	*0*	*1*	*3*	*4*	*2,287*	*2*
Other cancers	*0*	*0*	*1*	*1*	*2*	*865*	*1*
Diabetes	0	2	6	4	12	6,833	7
Cardiovascular disease	0	3	11	9	23	13,208	14
Ischaemic heart disease	*0*	*2*	*7*	*6*	*14*	*8,169*	*9*
Stroke	*0*	*0*	*1*	*2*	*3*	*1,734*	*2*
Other cardiovascular diseases	*0*	*1*	*3*	*1*	*6*	*3,305*	*3*
Mental disorders	3	4	2	1	10	5,542	6
Substance use disorders	*1*	*2*	*2*	*1*	*6*	*3,239*	*3*
Other mental disorders	*2*	*2*	*0*	*0*	*4*	*2,303*	*2*
Chronic respiratory disease	1	1	4	3	9	5,213	5
Other NCDs[(c)]	4	2	3	2	10	5,543	6
Injuries	**2**	**9**	**3**	**1**	**15**	**8,331**	**9**
Road traffic accidents	0	2	1	0	3	1,969	2
Suicide	0	3	1	0	4	2,393	2
Homicide & violence	0	2	1	0	3	1,716	2
Other injuries	1	2	1	0	4	2,252	2

(a) Communicable diseases, maternal and neonatal conditions
(b) Mouth and oropharynx, oesophagus, lung, larynx, pancreas, bladder, kidney, stomach, and uterine cancers
(c) Other non-communicable diseases

While the proportion of the Indigenous health gap in the four age groups was similar between males and females, the causes contributing to the gap varied by sex and age group (Table 6.3). In the 0–14-year age group, the gap had a similar causal distribution. In young adult males, injuries contributed almost as much to the gap as non-communicable diseases and suicide explained almost half of that. In young female adults, injuries contributed a lesser proportion to the gap but there was excess health loss from RTAs, suicide and violence. In both male and female young adults, mental disorders, early-onset diabetes and cardiovascular disease explained most of the gap for non-communicable disease.

Males and females aged 35 years and over experienced most excess health loss from cardiovascular disease, particularly ischaemic heart disease. At older ages, diabetes was responsible for a greater proportion of the gap in females than in males.

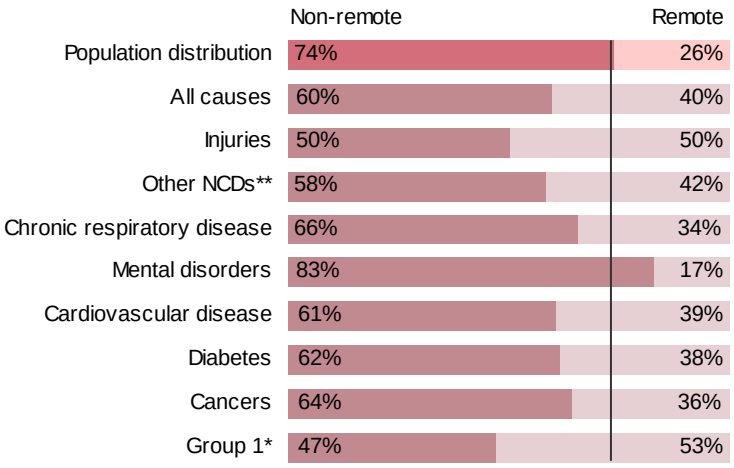
Table 6.3: Indigenous health gap (DALYs) due to selected causes by sex, expressed as a proportion of total Indigenous health gap, 2003

	Males (%)					Females (%)				
Cause	**0–14**	**15–34**	**35–54**	**55+**	**Total**	**0–14**	**15–34**	**35–54**	**55+**	**Total**
All causes	**17**	**26**	**36**	**21**	**100**	**18**	**23**	**33**	**26**	**100**
Group I[(a)]	**8**	**2**	**3**	**1**	**14**	**7**	**4**	**4**	**2**	**16**
Infectious and parasitic diseases	1	1	2	1	5	1	2	2	1	6
Acute respiratory infections	2	1	1	1	4	2	1	1	1	4
Neonatal causes	5	0	0	0	5	4	0	0	0	4
Other group I	1	0	0	0	1	1	1	0	0	2
Non-communicable diseases	**7**	**13**	**29**	**19**	**67**	**9**	**13**	**27**	**24**	**73**
Cancers	0	0	2	3	4	0	1	2	4	7
Tobacco related cancers[(b)]	*0*	*0*	*2*	*2*	*4*	*0*	*0*	*1*	*3*	*4*
Other cancers	*0*	*0*	*0*	*0*	*0*	*0*	*1*	*1*	*1*	*3*
Diabetes	0	2	5	3	10	0	2	6	5	14
Cardiovascular disease	0	3	12	8	24	0	3	10	9	23
Ischaemic heart disease	*0*	*2*	*9*	*5*	*16*	*0*	*1*	*6*	*6*	*13*
Stroke	*0*	*0*	*1*	*2*	*3*	*0*	*0*	*1*	*2*	*3*
Other cardiovascular disease	*1*	*1*	*3*	*1*	*5*	*0*	*2*	*3*	*2*	*6*
Mental disorders	2	5	3	1	10	4	3	2	0	9
Substance use disorders	*1*	*3*	*2*	*1*	*7*	*1*	*2*	*2*	*0*	*5*
Other mental disorders	*1*	*2*	*0*	*0*	*4*	*3*	*1*	*0*	*0*	*5*
Chronic respiratory disease	0	1	4	3	9	2	1	4	3	10
Other NCD[(c)]	4	2	3	1	9	3	3	3	2	10
Injuries	**2**	**11**	**5**	**1**	**18**	**1**	**6**	**2**	**1**	**11**
Road traffic accidents	0	2	1	0	4	0	2	1	0	3
Suicide	0	5	1	0	6	0	2	0	0	2
Homicide & violence	0	2	1	0	3	0	2	1	0	3
Other injuries	1	2	1	0	5	1	1	1	0	3

(a) Communicable diseases, maternal and neonatal conditions
(b) Mouth and oropharynx, oesophagus, lung, larynx, pancreas, bladder, kidney, stomach, and uterine cancers
(c) Other non-communicable diseases

Indigenous Australians residing in remote areas represented 26% of the total Indigenous Australian population and 35% of the total Indigenous burden; however they experienced 40% of the Indigenous health gap (Figure 6.3). This highlights that while the rate of burden is higher in remote areas, the bulk of the burden of disease is experienced by Indigenous Australians residing in non-remote areas.

Relative to population size Indigenous Australians residing in remote areas experienced a disproportionate amount of the health gap for all major disease areas apart from mental disorders (Figure 6.3). This latter finding may be an artefact of the methods. For example, the disease models for alcohol dependence & harmful use, and anxiety & depression were derived from the 2004-05 National Aboriginal and Torres Strait Islander Health Survey Social and Emotional Well-being module which gave proxies rather than diagnostic indicators for ICD-10 defined mental disorders.



* Communicable diseases, maternal and neonatal conditions
** Other non-communicable diseases

Figure 6.3: Indigenous health gap (DALYs) by selected causes expressed as: proportions by remoteness, 2003

Group I conditions were responsible for 20% and 12% of the health gap experienced by Indigenous Australians residing in remote and non-remote areas respectively (Table 6.4). The greater burden of infectious and parasitic diseases in remote areas was responsible for most of the difference in excess health loss in group I conditions. Similarly, injuries were responsible for a greater proportion of the gap in remote areas (18%) compared with non-remote areas (12%). The higher proportion of the health gap that was explained by injuries was due to a combination of higher rates of RTA, suicide, and homicide & violence burden in remote areas. Mental disorders contributed 14% of total health gap in non-remote areas, but only 4% in remote areas. Similar proportions of the health gap in remote and non-remote areas were contributed by other non-communicable diseases, but as the overall gap for remote was greater than would be expected given the population distribution, similar proportions mean a greater gap in numbers of DALYs per person.

The gap between remote and non-remote occurred at similar proportions between the four age groups (Table 6.4). A somewhat greater portion of the Indigenous health gap for those residing in non-remote areas occurred in the 0–14-year age group than in the remote gap.

Table 6.4: Indigenous health gap (DALYs) due to selected causes, expressed as a proportion of total excess burden, by remoteness, 2003

	Non-remote (%)					Remote (%)				
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**
All causes	**19**	**24**	**33**	**24**	**100**	**15**	**26**	**36**	**22**	**100**
Group I[(a)]	**6**	**2**	**3**	**1**	**12**	**9**	**4**	**5**	**2**	**20**
Infectious & parasitic diseases	1	1	2	1	4	2	2	3	1	8
Acute respiratory infections	1	0	1	0	3	3	1	1	1	6
Neonatal causes	4	0	0	0	4	4	0	0	0	4
Other group I	0	1	0	0	1	1	1	0	0	2
Non-communicable diseases	**11**	**15**	**27**	**23**	**76**	**4**	**10**	**28**	**20**	**62**
Cancers	0	0	2	4	6	0	0	3	2	5
Tobacco related cancers[(b)]	0	0	1	3	5	0	0	2	2	3
Other cancers	0	0	1	1	1	0	0	1	1	2
Diabetes	0	3	6	4	12	0	2	5	4	12
Cardiovascular disease	0	3	11	9	24	1	4	11	7	23
Ischaemic heart disease	0	1	8	6	16	0	2	7	4	13
Stroke	0	0	1	2	3	0	0	1	1	3
Other cardiovascular disease	0	1	2	1	5	1	2	3	1	7
Mental disorders	4	6	3	1	14	1	1	2	0	4
Substance use disorders	1	3	2	1	7	0	1	2	0	4
Other mental disorders	3	3	1	0	7	0	0	0	0	0
Chronic respiratory disease	2	1	4	3	10	0	1	4	3	8
Other NCD[(c)]	4	2	2	1	10	3	2	3	2	10
Injuries	**2**	**7**	**3**	**0**	**12**	**2**	**12**	**4**	**1**	**18**
Road traffic accidents	0	1	1	0	2	1	3	2	0	5
Suicide	0	3	1	0	4	0	4	0	0	5
Homicide & violence	0	2	0	0	2	0	3	1	0	4
Other injuries	1	1	1	0	4	1	2	1	0	4

(a) Communicable diseases, maternal and neonatal conditions
(b) Mouth and oropharynx, oesophagus, lung, larynx, pancreas, bladder, kidney, stomach, and uterine cancers
(c) Other non-communicable diseases

6.2.2 Health gap by risk factors

If Indigenous Australians experienced the same burden rates due to the 11 selected risk factors as the total Australian population, 29% of the total Indigenous Australian burden of disease would be avoided (Table 6.5).

The largest proportion of the Indigenous health gap due to risk factors occurred in the 35–54-year age group (Table 6.5). However, the 15–34-year age group experienced the largest proportion of health gap due to alcohol, illicit drugs, intimate partner violence, and child sexual abuse.

Table 6.5: Indigenous health gap (DALYs) due to selected risk factors, expressed as a proportion of excess burden from each risk factor, 2003

Risk factor	Total (%)					Health gap (DALYs)	% of total Indigenous burden
	0–14	15–34	35–54	55+	Total		
Tobacco	6	0	47	47	100	9,816	10
High body mass	0	9	57	34	100	8,953	9
Physical inactivity	0	13	48	39	100	6,554	7
High blood cholesterol	0	10	64	26	100	3,994	4
Alcohol	2	45	40	13	100	3,820	4
High blood pressure	0	5	45	50	100	3,215	3
Low fruit and vegetable intake	0	10	52	37	100	2,873	3
Illicit drugs	4	63	28	5	100	2,150	2
Intimate partner violence	0	48	42	11	100	1,836	2
Child sexual abuse	0	67	28	5	100	869	1
Unsafe sex	4	40	43	12	100	926	1
11 risk factors combined[(a)]	**3**	**21**	**45**	**32**	**100**	**27,383**	**29**

(a) Joint effect of 11 risk factors in Indigenous analysis, and 14 in national model (Begg et al. 2007) minus the burden from osteoporosis, occupation, and air pollution

Males and females experienced equal proportions of the Indigenous health gap due to the 11 risk factors together (Figure 6.4). The health gap due to unsafe sex and child sexual abuse was predominately experienced by females.

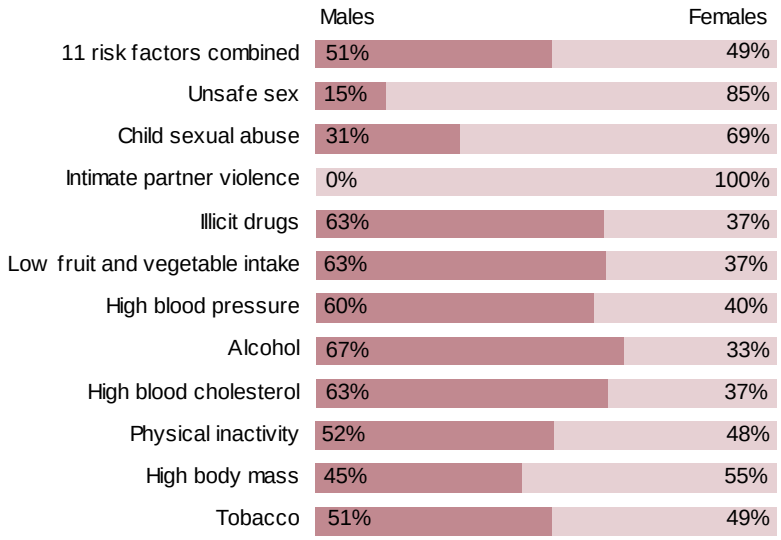


Figure 6.4: Indigenous health gap (DALYs) by selected risk factors expressed as proportions by sex, 2003

Indigenous Australians residing in remote areas experienced a disproportionate amount of the health gap due to all selected risk factors excluding illicit drugs (Figure 6.5).

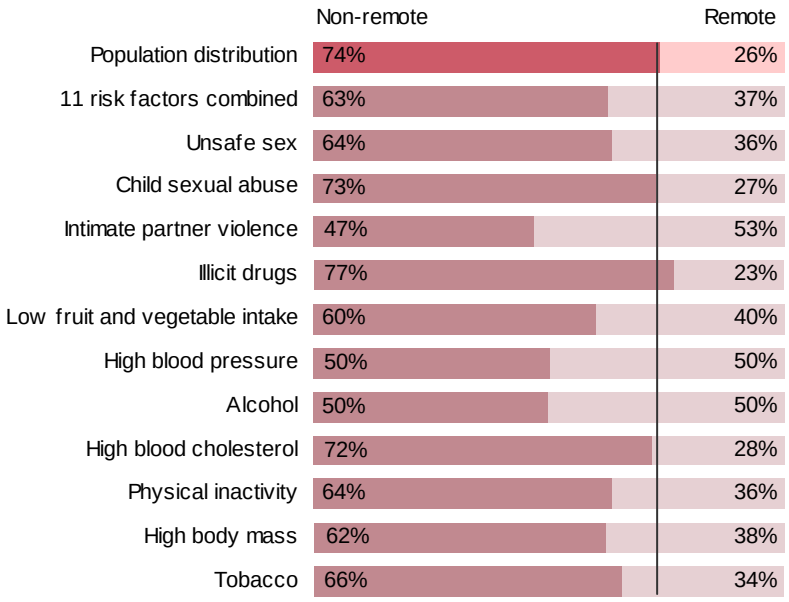


Figure 6.5: Indigenous health gap (DALYs) by selected risk factors expressed as proportions by remoteness, 2003

6.3 Differentials by remoteness

6.3.1 Disease burden by remoteness

Indigenous Australians experienced 3.6% of the total disease burden in Australia in 2003 (Table 6.6). While Indigenous Australians in remote areas made up just over one-quarter of the Indigenous Australian population, they experienced more than one-third of the Indigenous Australian disease burden (35.0%). In remote areas, Indigenous Australian males experienced a greater proportion of the burden than their counterparts in non-remote areas. For Indigenous Australians, a greater proportion of burden was due to years lost from mortality, particularly in remote areas.

Table 6.6: DALYs for remoteness categories by proportions of total, proportions by sex, and proportions due to mortality, Australia, 2003

			Per cent of DALYs	
Population group	**DALYs**	**Per cent of total**	**Males**	**Fatal burden**
Indigenous Australians	95,976	3.6	52.2	53.6
Non-remote	62,357	2.4	50.9	49.2
Remote	33,619	1.3	54.7	61.8
Total Australian population	2,632,770	100.0	51.8	48.6

Compared with Indigenous Australians residing in non-remote areas, Indigenous Australians residing in remote areas experienced a greater rate of burden for each of the 10 leading cause groups except for mental disorders (Table 6.7). The greatest remote to non-

remote differentials were for infectious and parasitic diseases (RR 2.9), intentional injuries (RR 2.0), and unintentional injuries (RR 2.0).

Table 6.7: .DALY rates per 1,000 by remoteness category for the 10 leading broad cause groups, Australia, 2003

Cause	Indigenous Australians	Non-remote Indigenous[(a)]	Remote Indigenous[(a)]	RR[(b)]
All causes	**201.9**	**181.1**	**257.9**	**1.4**
Cardiovascular disease	35.3	31.1	46.9	1.5
Mental disorders	31.3	32.8	27.0	0.8
Chronic respiratory disease	18.1	17.1	20.4	1.2
Diabetes mellitus	17.9	15.8	23.2	1.5
Cancers	16.4	15.8	18.4	1.2
Unintentional injuries	14.7	11.7	22.8	2.0
Intentional injuries	11.3	8.9	17.9	2.0
Nervous system and sense organ disorders	8.7	8.6	8.7	1.0
Neonatal causes	8.5	7.8	10.6	1.3
Infectious and parasitic diseases	8.0	5.3	15.3	2.9
Other	31.7	26.3	46.7	1.8

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Remote to non-remote Indigenous rate ratio

Indigenous Australians residing in remote areas experienced a higher rate of burden for 15 of the top 20 specific causes of Indigenous Australian burden (Table 6.8). The rate of burden from anxiety & depression, asthma, schizophrenia, and heroin or polydrug dependence was lower in Indigenous Australians residing in remote areas than those residing in non-remote areas. For lung cancer, the rate of burden was the almost the same in both areas. For the remaining conditions, the largest remote to non-remote differentials were for otitis media (RR 6.1), and rheumatic heart disease (RR 4.4).

Table 6.8: DALY rates per 1,000 by remoteness category for the 20 leading specific causes, Australia, 2003

Cause	Indigenous	Non-remote Indigenous[(a)]	Remote Indigenous[(a)]	RR[(b)]
Ischaemic heart disease	21.0	19.3	25.5	1.3
Type 2 diabetes	16.6	14.6	21.5	1.5
Anxiety & depression	15.7	16.9	12.1	0.7
Chronic obstructive pulmonary disease	7.6	7.0	9.3	1.3
Suicide	7.2	6.1	10.2	1.7
Asthma	6.9	7.8	4.4	0.6
Road traffic accidents	6.4	4.4	11.6	2.6
Alcohol dependence & harmful use	5.9	5.1	8.0	1.6
Stroke	5.7	5.2	6.9	1.3
Homicide & violence	4.1	2.8	7.6	2.8
Lung cancer	4.1	4.1	4.2	1.0
Low birth weight	3.8	3.4	4.9	1.4
Lower respiratory tract infections	3.5	2.4	6.5	2.7
Schizophrenia	2.6	2.9	2.0	0.7
Heroin or polydrug dependence	2.6	3.3	0.6	0.2
Inflammatory heart disease	2.5	1.7	4.8	2.8
Otitis media	2.1	0.9	5.7	6.1
Hepatitis	2.1	1.8	3.1	1.7
Rheumatic heart disease	2.1	1.1	4.7	4.4
Birth trauma & asphyxia	2.0	1.8	2.4	1.3

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Remote to non-remote Indigenous rate ratio

6.3.2 Risk factors by remoteness

Indigenous Australians residing in remote areas experienced a greater rate of burden due to nine of the 11 risk factors considered in this study and due to the 11 risk factors combined (Table 6.9). The largest differences in rates of burden were for intimate partner violence (RR 2.3), alcohol (RR 2.1), and high blood pressure (RR 2.0). Indigenous Australians residing in remote and non-remote areas experience a similar rate of burden due to high blood cholesterol.

The rate of burden from child sexual abuse was estimated to be similar in remote and non-remote areas. This was an artefact of the methods, where — due to lack of information — we assumed the same prevalence of child sexual abuse among all Indigenous Australians. These results may therefore not be a true reflection of the differentials between these two populations.

Table 6.9: DALYs attributable to 11 selected risk factors by Indigenous status and remoteness, rate per 1,000, Australia 2003

Risk factor	Total Indigenous	Non-remote Indigenous[(a)]	Remote Indigenous[(a)]	RR[(b)]
Tobacco	24.5	22.8	29.1	1.3
High body mass	23.0	20.3	30.0	1.5
Physical inactivity	16.9	15.3	21.0	1.4
High blood cholesterol	11.1	11.0	11.3	1.0
Alcohol				
Harmful effects	*12.6*	*10.1*	*19.2*	*1.9*
Beneficial effects	*–1.7*	*–1.7*	*–1.6*	*0.9*
Net effects	*10.9*	*8.3*	*17.6*	*2.1*
High blood pressure	9.3	7.3	14.4	2.0
Low fruit and vegetable intake	7.0	6.1	9.6	1.6
Illicit drugs	6.9	7.1	6.2	0.9
Intimate partner violence	5.2	3.8	8.8	2.3
Child sexual abuse	2.9	2.9	2.9	1.0
Unsafe sex	2.5	2.2	3.1	1.4
11 risk factors combined[(c)]	**75.5**	**68.2**	**95.0**	**1.4**

(a) Age standardised to the total Indigenous Australian population, 2003
(b) Remote to non-remote Indigenous rate ratio
(c) Figures for joint effects are not column totals; see Section 5.1 for further details

6.3.3 Mortality by remoteness

The probability of dying between ages 15 and 60 years is higher in Indigenous Australians residing in remote areas than in those residing in non-remote areas: 46% and 31% for males and females, respectively (Table 6.10). Also, the probability of dying before age 5 is higher in Indigenous children in remote areas compared to that of children in non-remote areas: 2.5% and 2.1% for males and females, respectively (compared to 1.5% and 1.3%).

Our estimates indicate Indigenous Australians residing in remote areas have a markedly lower life expectancy than Indigenous people residing in non-remote areas (major cities and regional areas combined): 58 years vs. 66 years in males and 65 years vs. 70 years in females. However the extent of the differences in life expectancy for Indigenous Australians by residence of remoteness is a matter of ongoing controversy and debate. Some members of the Steering Committee consider that at least some of the differences may be due to incomplete identification of Indigenous deaths and population counts in non-remote areas despite our attempts to correct for these issues using indirect demographic methods. On the other hand, members of our technical advisory panel believed our findings to be plausible and data on non-fatal health outcomes analysed for this study also indicated substantial differences in the health status of Indigenous Australians by remoteness.

Table 6.10: Expectation of life at birth and probability of dying before age 5 and between ages 15 and 60 for Indigenous Australians by remoteness, 1996–2001

	Life expectancy (years)		Life expectancy gap with total population (years)		Probability of dying before age 5		Probability of dying between ages 15 and 60	
Area	**Males**	**Females**	**Males**	**Females**	**Males**	**Females**	**Males**	**Females**
Indigenous Australians	64	69	13	13	0.016	0.014	0.326	0.231
Non-remote	66	70	11	12	0.015	0.013	0.272	0.199
Remote	58	65	19	17	0.025	0.021	0.460	0.314
Total Australian population	77	82			0.007	0.006	0.101	0.057

6.3.4 Healthy-adjusted life expectancy by remoteness

Health-adjusted life expectancy (HALE) provides an estimate of the average years of equivalent 'healthy' life (without disability) 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 that 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 spend at each age in states less than perfect health, adjusted for the relative severity of those health states. The sum of PYLD across all causes is used to derive this 'severity-weighted' proportion for each age. We assumed no change in life expectancy from 1996–2001 estimates when calculating the HALE estimate for Indigenous Australians in 2003.

The gap in HALE between the Indigenous Australian population and total Australian population was approximately 15 years (Table 6.11); with Indigenous Australians losing a greater proportion of their shorter life expectancy due to disability.

Table 6.11: Health-adjusted life expectancy at birth by area and sex, Indigenous Australian and total Australian populations, 2003

	HALE (years)		Life expectancy at birth lost due to disability (%)	
	Males	**Females**	**Males**	**Females**
Indigenous Australians	56	60	18	13
Non-remote	58	62	12	12
Remote	51	56	13	13
Total Australian population	71	75	10	9

7 Discussion and conclusions

7.1 Policy implications

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 study contributes most obviously by identifying the magnitude of health problems in the Indigenous population and by quantifying the contribution of major modifiable risks to these health problems. In particular, the excess amount of disease burden that Indigenous Australians experience in comparison with the total Australian population indicates where the greatest potential for health gain is. The fact that much lower rates of disease burden are experienced by the majority of Australians means that with existing knowledge and technologies, a reduction of most of the Indigenous health gap is achievable. However, reducing this health gap is a momentous task, because the health disadvantage of Indigenous Australians is apparent in almost all diseases and risk factors, at all ages, in men and women, and in remote and less remote areas. The most important contribution of this study is to comprehensively quantify the health problems that Indigenous Australians face. This helps policy makers identify which health issues are most likely to lead to health improvements if targeted for intervention.

To set priorities for interventions, complementary information is needed on the cost-effectiveness of specific interventions for each of these health problems. The details provided in this study on each disease and risk factor are essential inputs to such economic analyses. This set of results is already being incorporated into economic models that are under development for the 'Assessing Cost-Effectiveness (ACE)—Prevention' project funded by the NHMRC at The University of Queensland and Deakin University. The aim of the ACE-Prevention project is to comprehensively model the cost-effectiveness of preventive intervention options for non-communicable disease in Australia and, explicitly, to examine the implications if these interventions are delivered to Indigenous Australians.

There is great potential to reduce the Indigenous health gap by addressing the 11 risk factors identified in this study. There is growing evidence to guide choices on what preventive interventions for which risk factors are most likely to be effective and cost-effective in the total Australian population but not yet for the Indigenous population. The much higher disease burden makes it more likely that interventions can achieve large health gain in Indigenous Australians. In economic evaluations this needs to be taken into account together with the different costs of interventions that are culturally appropriate and may need to also reach people in remote areas.

In this study, we found that certain diseases and risk factors contributed more to the overall burden of disease in Indigenous Australians, particularly to the Indigenous health gap. These included cardiovascular disease, diabetes and other tobacco-related conditions, such as lung cancer and chronic respiratory disease. Collectively, these diseases accounted for half the Indigenous health gap. Apart from tobacco, these diseases shared additional lifestyle risk factors, including high body mass, physical inactivity, raised blood pressure and cholesterol. These health problems largely affect middle-aged and older Indigenous Australians; however, they start at young ages, and there already is a sizeable burden in the 15–34-year

age group. Therefore, prevention efforts should be targeted at a much younger Indigenous population than in the rest of the population.

Further health gain can be expected if the excess burden from infectious disease and neonatal conditions is addressed. These conditions explained 15% of the Indigenous health gap. Half of this excess burden was experienced by children.

Suicide, RTAs, and homicide & violence were the main injuries that explained another 15% of the health gap. Most of this excess in injuries occurred at young adult ages and there was a particularly high rate of suicide in young Indigenous males. Mental disorders, including substance use disorders — particularly alcohol, also contributed significantly (10%) to the health gap.

When addressing the health gap, it is important to note that the focus should not just be on prevention. The higher proportion of the health gap that was due to mortality reflected the greater chance of dying if Indigenous Australians fall sick. Each disease may have specific problems to be addressed; however, it is likely that the higher case fatality for most diseases was influenced by a combination of late presentations, shortcomings in acute surgical and medical management, and inadequate follow-up during the course of disease. Therefore, a multi-pronged approach is needed.

It is important to note that while the rate of burden of disease may be higher in remote areas, the bulk of the burden and the health gap for Indigenous people is in non-remote areas since the vast majority of Indigenous Australians reside in non-remote areas. The policy implication is that the focus of health service action to reduce inequalities in Indigenous health needs to include culturally appropriate and uniquely targeted approaches for non-remote and remote areas. The value of this report is that it indicates to policy makers where the emphasis may need to vary depending on the health problem being addressed by broad remoteness area.

Addressing the multitude of health problems facing Indigenous Australians is complex and will require a wide range of initiatives to increase preventive and curative efforts from mainstream and particularly Indigenous health services. NACCHO states that:

> It is widely recognised that health solutions lie in assisting Aboriginal people being able to enjoy their right to self-determination. All relevant inquiries and studies have shown conclusively that culturally appropriate, comprehensive primary health care (such as Aboriginal community controlled health services), based on maximum community participation, is the best way of addressing Aboriginal health (NACCHO 2001).

It would be more effective to combine this with approaches outside the health sector to address the social and economic disadvantages that contribute to the poor health status of Indigenous Australians. This is in keeping with the Indigenous concept of health which acknowledges that:

> Improving Aboriginal health is not just about improving the physical well-being of an individual. It is about working towards the social, emotional, and cultural well-being of the whole community in which each individual is able to achieve their full potential as a human being (NACCHO 2001).

However these requirements should not lead to inaction by health policy makers arguing that the social and economic problems should be tackled first. It is within the reach of appropriately resourced health services to reduce a sizeable proportion of the Indigenous health gap.

7.2 Precision of estimates

7.2.1 Mortality estimates and years of life lost

Assessing the health status of Indigenous Australians is not easy. The main challenge is the inaccurate or incomplete identification of Indigenous status in population health and census data collection systems. When estimating mortality in Indigenous Australians, this means that ascertainment of the true rate of death requires adjustments using indirect demographic methods, which inherently introduce an additional degree of uncertainty. We applied the GGB method, an indirect demographic technique frequently used in developing country settings to estimate under-reporting of deaths (Hill 1987). The ABS has applied a similar method developed by Bhat (2002) to correct for under-registration of deaths for the purpose of Indigenous population projections. Ken Hill, a demographer at Harvard University and the world expert on these indirect demographic techniques, recommended that we use the GGB method in preference to the Bhat method, as applied by the ABS, because it requires fewer assumptions. As there is no 'gold standard' against which to compare either method, plausibility is the key issue. The GGB's main assumptions are a constant age pattern of under-identification of the Indigenous population counts at two successive censuses and a constant age pattern of under-identification of deaths in the vital registration system. This may not be the case.

To test these two vital assumptions, we carried out a series of sensitivity analyses using advice from the steering committee members on the likely direction of departure from this constant age pattern by assuming greater proportions of under-identification in younger adults and a greater proportion of males being under-identified. None of the sensitivity scenarios altered our conclusion that the true level of mortality of Indigenous Australians was not as high as the ABS has estimated (a gap in life expectancy at birth of around 13 years rather than 17 years) but was still at unacceptably high levels for a high-income country.

Uniquely, we also introduced a calculation of corrected mortality rates and life expectancy for Indigenous Australians residing in remote and non-remote areas. This showed, for the first time, that there is a considerable difference in mortality for Indigenous Australians residing in remote compared to non-remote areas. We were also able to correct these estimates for the net flow of migration of Indigenous people from remote to non-remote areas based on census data. However, the extent of the differences in life expectancy between remote and non-remote areas is a matter of ongoing controversy and debate. Some members of the Steering Committee consider that at least some of the differences may be due to incomplete identification of Indigenous deaths in non-remote areas. On the other hand, data on non-fatal health outcomes analysed for this study indicate that there also are substantial differences in health status between Indigenous people residing in remote and non-remote areas.

Despite our confidence that we estimated plausible mortality rates, we cannot be certain about the true level of mortality in Indigenous Australians until better data becomes available. The ABS has commenced a project to link 2006 Census with deaths that occurred in the 10 months following the Census. This will greatly assist in determining the completeness of death registration relative to census counts. The longer-term strategy will have to include efforts to improve the recording of Indigenous status in the vital registration system.

Estimating the cause of death was deemed more accurate because the quality of certification of cause of death for Indigenous people was similar to that for the rest of the population when comparing the proportions of deaths coded to ill-defined codes. We therefore assumed that the cause of death pattern in deaths recorded as Indigenous reflected the pattern of all Indigenous deaths. As we discussed earlier, in the National Study (Begg et al. 2007), there are unresolved issues around the validity of cause of death attribution even in the high-quality vital registration systems that we have in Australia. That concern would also apply to cause of death attribution in Indigenous Australians but is unlikely to lead to different conclusions about the main causes of fatal disease.

In this study, we assumed that the level of mortality estimated for the 1996–2001 period between censuses applied to our baseline year of 2003 for the burden of disease estimation. This was a decision made in consultation with our technical advisory panel because there was a lack of reliable information on mortality trends in Indigenous Australians. More recent information has indicated an improvement in life expectancy in Aboriginal people in the Northern Territory that are of the same magnitude as the trends in life expectancy for the total Australian population, although with a reversal of the sex ratio (Wilson et al. 2007). The annual rate of change in life expectancy in Northern Territory Indigenous men was 0.22 years compared with 0.33 years for all Australian men; for Northern Territory Indigenous women it was 0.38 years compared with 0.22 years in all Australian women. If we applied the Northern Territory trend to adjust our mortality estimate downwards, the total amount of YLL in Indigenous Australians would have been lower by 7% and the total amount of DALYs lower by 4%.

7.2.2 Non-fatal burden

Estimates of disability for the more than 170 disease and injury categories included in this study depended on a combination of methods. When available, we included data on directly observed Indigenous health events in routine health statistics databases, health surveys or epidemiological studies. For many diseases, such information did not exist. Instead, ratios of the differences between Indigenous and total population rates, for hospital admissions or mortality, were sought as proxy measures of disease occurrence. The consequence is that there are varying degrees of confidence about the accuracy of these estimates. This study provides transparency on the data sources consulted and all the assumptions and judgments on which the results depend.

Only six of the top 20 disabling conditions had good-quality estimates of their incidence. These conditions included ischaemic heart disease, stroke, homicide & violence, low birth weight, and birth trauma & asphyxia — all of which relied on hospital admission statistics. However, hospital data also suffers from under-reporting of admissions for Indigenous Australians. We made crude adjustments based on judgments by the states and territories on the quality of the recording of Indigenous status in their jurisdiction combined with one study of the quality of Indigenous status identification by remoteness from New South Wales. What we were not able to adjust for was the potential difference in propensity to be admitted for a health problem between the Indigenous and total Australian population. A range of factors, including care-seeking behaviour, severity of disease at presentation, distance to hospital and admission practices, may be different for Indigenous Australians. Some of these factors would lead to higher and some to lower admission rates. It is not clear what the net effect is and how much this varies from disease to disease. We judged that the estimates for cardiovascular events and more severe injuries were more likely to reasonably reflect true differentials in disease occurrence as they were emergency events. For maternal

and neonatal conditions, we also judged the accuracy of hospital-derived estimates as good quality, because almost all deliveries take place in hospital.

The only other condition with good data among the top 20 disabling conditions was dental caries. We judged the accuracy of estimates for alcohol dependence, migraine and personality disorders as particularly poor. The accuracy of estimates for the remaining top 20 conditions were only judged as fair only. This indicated that there was a large gap in knowledge to accurately estimate the disease occurrence of many major diseases contributing to the disability component of the burden of disease. Mental disorders, Type 2 diabetes, asthma and COPD each had good-quality data to estimate the burden in the total Australian population but accurate data in Indigenous Australians was lacking. We resorted to finding differentials between Indigenous Australians and the total Australian population in proxy data sources, or were limited to a few isolated epidemiological studies to generalise to the whole population. This is a key strategy in burden of disease and injury studies. Instead of ignoring health problems with poor data sources, we endeavoured to approximate the size and extent of each health problem even if it meant using less-adequate proxy indicators. The argument for doing so was that this makes it more worthwhile to policy makers than presenting an incomplete quantification of health problems.

A key gap in available data to estimate the large contribution of major risk factors to the overall Indigenous burden of disease and the Indigenous health gap was the lack of representative health measurement data on key risk factors, such as blood pressure, cholesterol and body mass. We strongly recommend that a representative Indigenous health measurement survey is undertaken to measure these risk factors. Such a survey could include also measure a number of other important health problems for which we currently have poor data, such as diabetes, COPD, asthma and hearing loss.

Appendix A Australian mortality data adjustment

A.1 Mortality data

A.1.1 Data access

Throughout this report, we use the term 'Australian mortality data' as shorthand for the AIHW's National Mortality Database (AIHW 2003a). We accessed Australian mortality data under the data sharing agreement put in place between The University of Queensland and the AIHW for the purposes of estimating the burden of disease and injury in Australia in 2003. We were supplied with a unit record file of Australian deaths, which included: underlying and associated causes of death coded according to the 10th revision of the *International Classification of Diseases* (ICD-10), date of death registration, Indigenous status, usual statistical local area (SLA) of residence, age and sex. For burden of disease estimates, we considered all deaths of Indigenous Australians that were registered between 2000 and 2003 (specifically 2000–02 deaths for the GGB method and 2001–03 deaths for the Indigenous cause of death structure).

A.1.2 Indigenous status in mortality records

It is important to note that while an Indigenous identifier has been progressively introduced since 1980, a uniform system of identifying the Indigenous status of all deaths in Australia was only established in the mid to late 1990s. Queensland only included a question on Indigenous status of the deceased person in 1996.

When an Indigenous person dies, their death will only be classified correctly as an Indigenous death in the registrar's database if the following events occur:

- the question about Indigenous status is answered correctly on the death certification form completed by the funeral director (which usually requires that the question is asked of a relative or friend who knows what the deceased person would have answered; and that the answer is recorded correctly on the form)
- the form is completed and submitted to the registrar's office
- the form is processed and the information about Indigenous status is correctly entered into the system
- this information is retained throughout the editing and data processing stages and captured correctly on the file sent by the registrar to the ABS.

A.1.3 Classification of deaths for burden of disease purposes

Deaths were mapped into 'burden of disease and injury' categories and some specific and non-specific causes of death were redistributed. We refer the reader to the following sections — 'Categorising deaths' and 'Redistributing non-specific causes of deaths' (pages 18–21) — within the National Study for additional information on this topic area (Begg et al. 2007).

A.1.4 Classification of data by remoteness

We used the 2001 Australian Standard Geographical Classification (ASGC) Remoteness Areas (based on ARIA+ index values) to code deaths by SLA of usual residence to broad remoteness areas. The ASGC is a geographic approach to remoteness based on road distance to five categories of service centre (as a surrogate for remoteness) and on the population size of a service centre (as a surrogate for the availability of services). A classification based on road distance implies that persons have equal access to road transport (either via car or public transport). However, some population groups (such as persons in less urban areas) have lower levels of access to car and public transport than the general population. Road distance classifications also do not allow for differences in terms of road quality and serviceability (some roads in remote Australia are inaccessible for substantial parts of the year due to flooding). The five categories of remoteness are (AIHW 2004b):

- major cities of Australia
- inner regional Australia
- outer regional Australia
- remote Australia
- very remote Australia.

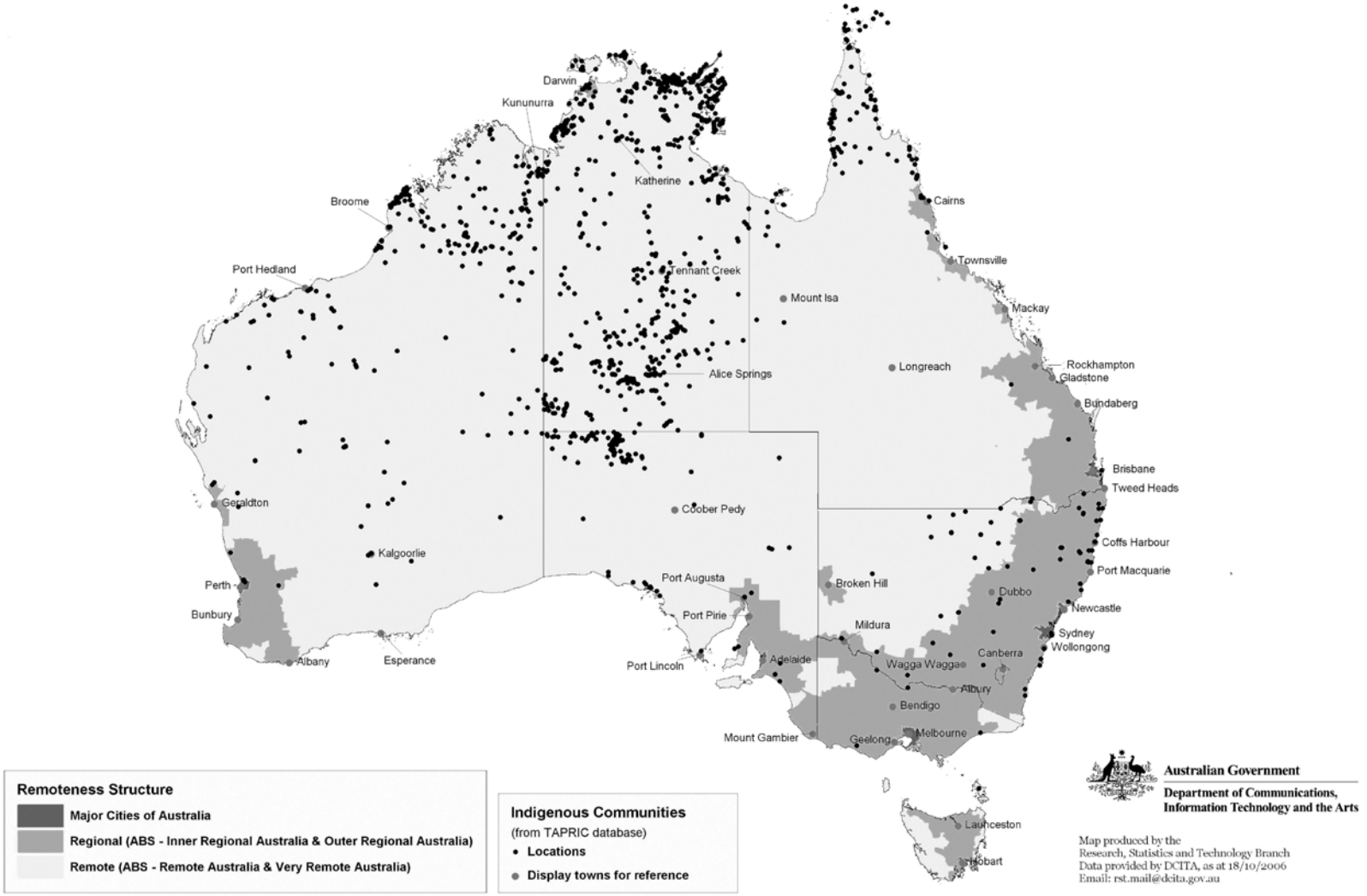
We collaborated with the ABS to develop a whole-of-allocation SLA to broad remoteness area mapping (to account for those SLAs that are not wholly contained within one remoteness area) for the 1991, 1996 and 2001 census data . We then purchased these concordances. To take into account boundary changes in SLAs between censuses, we updated the mapping file using published information (ABS 2001). We initially aggregated the five ASGC categories into three broad remoteness areas: major cities, regional (inner regional and outer regional) and remote (remote and very remote) areas. In 2001, Indigenous Australians were fairly evenly distributed across Australia by remoteness areas with just over one in four Indigenous Australians residing in remote areas, compared with 2% of non-Indigenous Australians; while the vast majority of the non-Indigenous population resided in Major cities (67%) (Table A.1) (ABS 2003b).

Table A.1: Per cent of Australian population by broad remoteness areas and Indigenous status, 2001

Indigenous status	Major cities	Regional	Remote
Indigenous	31	43	26
Non-Indigenous	67	31	2

Source: ABS (2003b)

The following map shows a graphical distribution of the Indigenous Australian communities by the broad remoteness categories of major cities, regional and remote areas (Figure A.1).



Source: Australian Government Department of Communications Information Technology and the Arts (2006)

Figure A.1: Indigenous communities (remoteness structure) map

Following our first application of the GGB method (see section A.2.4) we further refined the ASGC categories into non-remote (major cities, inner regional and outer regional) and remote (remote and very remote) areas.

A.1.5 Estimating the 2003 experimental Indigenous population by remoteness

Rates in this study are calculated from the low series of experimental projections for 2003 based on the 2001 Census of Population and Housing. We extrapolated the Indigenous experimental population for 2003 by broad remoteness areas (non-remote and remote), sex and age (0,1,5–85+) in Stata v. 9.0. First we used the 2001 census data for Indigenous Australians by remoteness to extrapolate the appropriate percentage breakdowns for our age groups of interest: 0,1,5 (by 5-year age groups) to 85+. Secondly we adjusted the projections to give our age groups of interest. Finally we assumed that the proportionate breakdown by age and sex in 2001 still held in 2003 by broad remoteness areas. We used the following ABS files: (a) 30 June 2001 census data for Indigenous Australians by ASGC remoteness areas, sex and age (0,1,5–100+) — customised data request; (b) 30 June 2001 experimental Indigenous population estimates by states and territories, ASGC remoteness areas, sex and age (0, 5–75+) — customised data request; (3) 2001–2009 Experimental Projections of Aboriginal and Torres Strait Islander Australians by state and territory, ATSIC Regions, sex and age (0,4,5–75+) — available as a download (ABS 2004b); and (4) 2001–2009 Experimental Projections of Aboriginal and Torres Strait Islander Australians by state and territory, ATSIC Regions, sex and age (0,1) — customised data request.

A.2 Method for correcting mortality rates

A.2.1 Methods used by the ABS

The first indirect demographic method applied by the ABS to correct for under-identification in Indigenous Australian mortality rates was the Preston and Hill (1980) method In a subsequent review, the ABS determined that the method was not appropriate because the ongoing unexplainable intercensal increases in people identifying as Indigenous violated the method's requirement of a closed population. Following the 2001 census, the ABS utilised a method proposed by Bhat (2002) that allows for migration of known age pattern, but at the cost of requiring information about the rate of natural increase of the population.

A.2.2 ABS results using the Bhat method

Experimental work for the 1996–2001 period by the ABS using the Bhat method indicated a large difference (around 17 years) in the life expectancy at birth of Indigenous Australians at a national level (59.4 years for males and 64.8 years for females) relative to the total Australian population (76.7 years for males and 82.4 years for females) for the 1998–2000 period (ABS 2004a). Estimates by grouped states and territories were made (Table A.2). There was no clear pattern across jurisdictions with Northern Territory males having the lowest life expectancy at birth (57.6 years) by 0.9 years whereas Northern Territory females had the second highest life expectancy at birth (65.2 years) after South Australia. The ABS assumed that the Bhat-derived correction factor, which is based on deaths at ages 5 to 64, applied to deaths at all ages (including under-five mortality) (ABS 2004a).

Table A.2: ABS life expectancy at birth estimates (years) for Indigenous Australians by sex, 1996–2001

Area	Males	Females	Difference
Australia	59.4	64.8	5.4
NSW, Vic, ACT and Tas	60.0	65.1	5.1
Qld	58.9	62.6	3.7
SA and WA	58.5	67.2	8.7
NT	57.6	65.2	7.6

Note: Unadjusted for interstate migration

While the ABS assessed the life-expectancy estimates based on the Bhat method as suitable for the purpose of producing short-range experimental estimates and projections of the Indigenous population, the ABS makes the following important qualifications (ABS 2004a):

1. "There is some degree of circularity in the way the unexplained growth is calculated. The Bhat method is used to estimate the completeness of death registration data and then to produce an 'approximate' life table for Australia. This 'approximate' life table is then used to calculate unexplained growth of the Indigenous population. Therefore, the accuracy of the level and age distribution of unexplained growth obtained this way is very much dependent on the accuracy of the 'approximate' life table this method produced in the first place."
2. "It should be noted that the correction to the registered Indigenous deaths, the level of the unexplained population growth and the age distribution of the unexplained growth are all based on subjective judgements, and any variations in these would influence the outcome as measured by the expectation of life at birth."
3. "Results obtained from the sensitivity analysis show that life expectancy estimates derived under various assumptions vary widely, suggesting that the life expectancy estimates in the life tables will not be robust for some forms of analysis."
4. "Over-precise analysis of these life expectancy estimates as measures of Indigenous health outcomes should be avoided."

A.2.3 UQ results for grouped states using the GGB method

Table A.3 shows our results of the GGB method for 1996 to 2001. The GGB method indicated life expectancy at birth estimates for Indigenous Australians around 13 years lower than that of the total Australian population. Thus, our reassessment suggested that the total Indigenous mortality differential, although still large, was smaller than previously estimated by the ABS. Further, the GGB method indicated a gradient in the mortality experience of grouped jurisdictions. Note, as in the ABS approach, that the life expectancy estimates for grouped states have not been adjusted for interstate migration.

Table A.3: GGB life expectancy at birth estimates (years) for Indigenous Australians by sex, 1996–2001

Area	Males	Females	Difference
Australia	64	69	5
NSW, Vic, ACT and Tas	68	72	4
Qld	64	68	4
SA and WA	64	67	3
NT	56	65	9

Note: Unadjusted for interstate migration

Interestingly, the GGB results are similar to results reported by the ABS prior to their second major adjustment for 'identification migration' (i.e. assuming a population growth rate) (Table A.4). This is because, up to this point, the Bhat method is essentially treating change in identification as change in census coverage, similar to the GGB method. Professor Hill argued that this is not necessary and that correcting again for migration is essentially a double adjustment.

Table A.4: GGB life expectancy at birth estimates (years) for Indigenous Australians by sex versus ABS results with zero unexplained change, 1996–2001

	Males			Females		
Area	ABS[(b)]	GGB	Difference	ABS[(b)]	GGB	Difference
Australia	63	64	1	68	69	1
NSW, Vic, ACT & Tas[(a)]	66	68	2	71	72	1
Qld	62	64	2	68	68	–
SA & WA[(b)]	61	64	3	70	67	-3
NT	57	56	-1	64	65	1

(a) The ABS presented life expectancy results at birth for this stage only by individual jurisdiction the GGB New South Wales grouped states are compared only with ABS New South Wales and GGB South Australia/Western Australia are compared only with South Australia
(b) *Source:* ABS (2004a)
Note: Unadjusted for interstate migration

A.2.4 UQ results by remoteness (major cities, regional and remote) using the GGB method

We hypothesised that the gradient observed in our exploratory life expectancy results by grouped states and territories could largely be explained by the proportions of Indigenous people residing in major cities, regional and remote areas (Table A.5) and the level of life expectancy by area of remoteness. For example, the Northern Territory had the lowest life expectancy and the highest proportion of people residing remotely whereas New South Wales/Victoria had the highest life expectancy and the lowest proportion of Indigenous people residing remotely.

Table A.5: Per cent of Indigenous population by broad remoteness areas and grouped jurisdictions, 2001

Grouped states	Major cities	Regional	Remote
NSW, Vic	40	55	5
Qld	25	51	24
SA, WA	36	25	39
NT	0	19	81

Source: AIHW (2005b)

Applying the GGB method to census and mortality data for Indigenous people by remoteness supported this hypothesis, with remote areas experiencing the lowest life expectancy at birth and major cities/regional areas faring similarly (Table A.6). Note, at the time these estimates were made inter-regional migration was not accounted for. After consultation with our advisory committees it was decided to combine the estimates for major cities and regional areas. Thus mortality differentials and disease burden estimates in this report are presented for Indigenous Australians residing in non-remote and remote areas only.

Table A.6: Preliminary GGB life expectancy at birth estimates (years) for Indigenous Australians by broad remoteness areas and sex, 1996–2001

Area	Males	Females
Australia	*64*	*69*
Major cities	68	71
Regional	67	72
Remote	58	64

A.2.5 Final UQ results by remoteness (non-remote and remote) using the GGB method

Based on the GGB method, Indigenous people residing in remote areas experience a greater gap in life expectancy compared with Indigenous people in non-remote areas (Table A.7).

Table A.7: GGB life expectancy at birth (years) for Indigenous Australians by broad remoteness areas and sex, 1996–2001

Area	Males		Females	
	Life expectancy	Difference with total population	Life expectancy	Difference with total population
Australia	*64*	*13*	*69*	*14*
Australia (fitted)[(a)]	*64*	*13*	*69*	*14*
Non-remote	66	11	70	12
Remote	58	18	65	18

(a) Fitted life expectancies for Australia are based on corrected age- and sex-specific mortality rates for non-remote and remote areas multiplied by population data to give deaths, which are summed and entered into a new life table for Indigenous Australians at a national level

A.2.6 Consistency check

We multiplied and summed the proportions of Indigenous people residing in non-remote and remote areas in each of the grouped states by the corresponding life expectancy estimates for non-remote and remote areas. This gave life expectancy estimates that are reasonably consistent with those predicted by the GGB method for the grouped states (Table A.8). This finding supported our hypothesis that differentials in life expectancy of Indigenous Australians by states and territories are largely a function of remoteness.

Table A.8: GGB life expectancy at birth estimates (years) for Indigenous Australians by grouped jurisdictions and sex versus predicted results, 1996–2001

	Males			Females		
Area	**Predicted**	**GGB**	**Difference**	**Predicted**	**GGB**	**Difference**
NSW, Vic, ACT and Tas	66	68	–2	70	72	–2
Qld	64	64	–	69	68	1
SA and WA	63	64	–1	68	67	1
NT[(a)]	60	56	4	66	65	1

(a) Compared with the 1996–2001 Northern Territory life expectancies at birth based on John Condon's internally consistent dataset, males: 59.8; females: 66.0 (Wilson et al. 2007)
Note: Predicted life expectancy at birth based on life expectancy by remoteness, age and sex and proportion of people residing by remoteness in each grouped jurisdiction by age and sex

A.3 Cause of death structure

Since the recording of mortality statistics is thought to be complete in Australia and Indigenous identification is incomplete, it follows that deaths in the not-stated Indigenous category and the non-Indigenous category include Indigenous deaths. The plotted proportions of deaths by age groups show that the deaths with not-stated Indigenous status have a very similar age distribution to that of the non-Indigenous deaths, with most of the deaths occurring in older age groups (Figure A.2 and Figure A.3). Conversely, the age distribution of Indigenous deaths shows a distinctly different pattern for both males and females. These graphs suggest that the deaths in the not-stated Indigenous category are predominantly non-Indigenous deaths. However, it is important to note that even if there is a small number of deaths within the not-stated Indigenous categories that are Indigenous, this could significantly affect Indigenous mortality rates without influencing the non-Indigenous mortality profile.

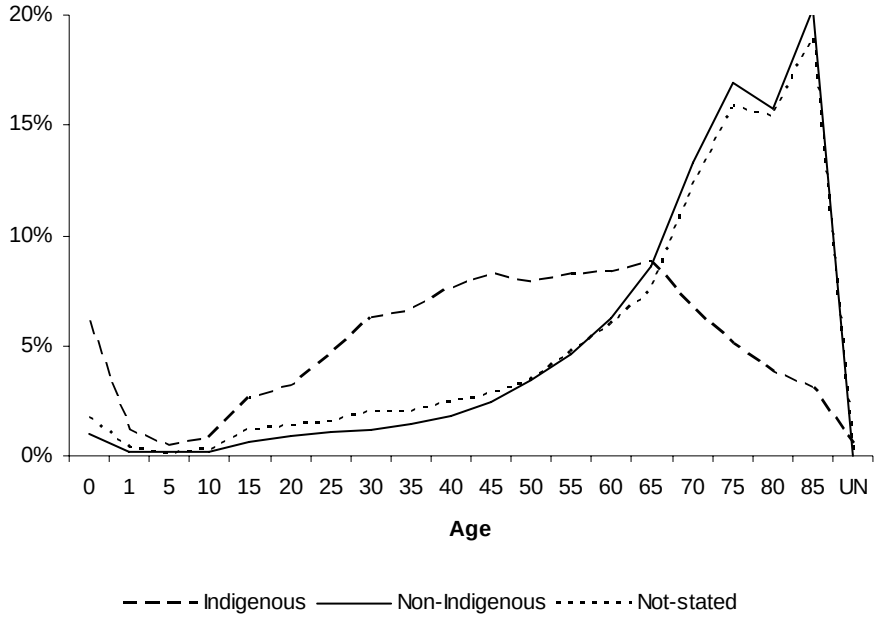


Figure A.2: Proportion of male deaths by recorded Indigenous status and age, 2000–02

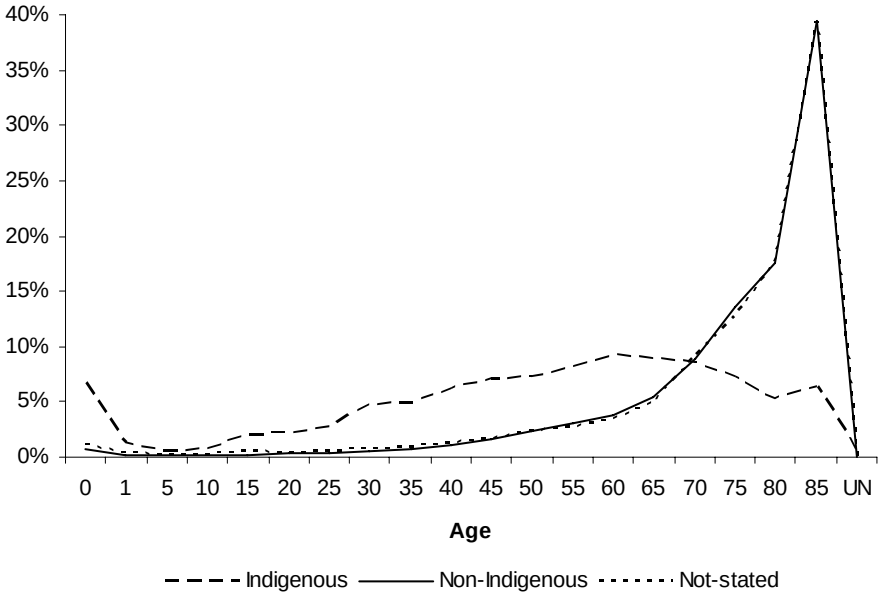


Figure A.3: Proportion of female deaths by recorded Indigenous status and age, 2000–02

Apart from childhood mortality, age-specific mortality rates are expected to increase linearly and exponentially. The logged age- and sex-specific mortality rates for the Indigenous Australian and total Australian populations followed this pattern (Figure A.4 and Figure A.5). This suggested that vital registration data for deaths recorded as Indigenous was not subject to a selection bias by age.

Figure A.4: Logged recorded male Indigenous and total Australian population rates, 2000–02

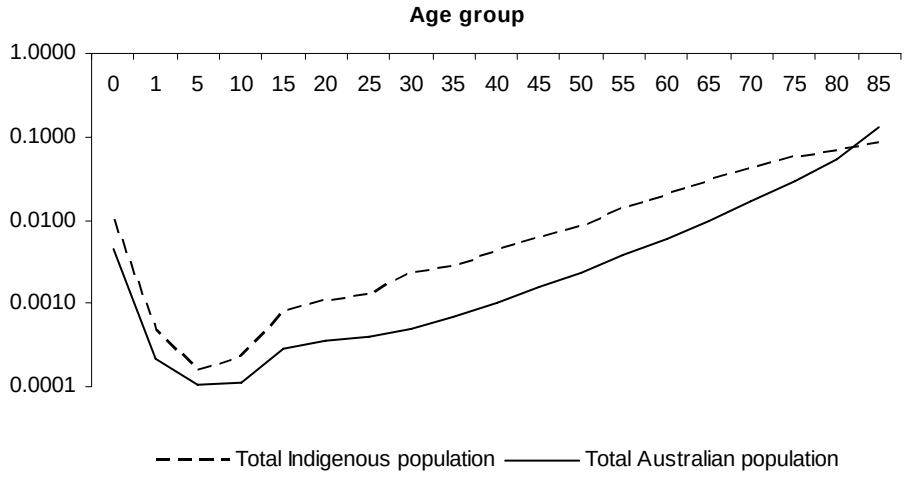


Figure A.5: Logged recorded female Indigenous and total Australian population rates, 2000–02

A.3.1 Analysis of Indigenous cause of death structure

Proportions of ill-defined and residual categories contributing to overall mortality are indicators of the quality of the cause of death coding. Ill-defined mortality as a proportion of overall mortality was less than 2% for both Indigenous and non-Indigenous Australians (Figure A.6). In addition, residual categories (e.g. other neoplasms, other cardiovascular conditions, etc) for Indigenous and non-Indigenous Australians contributed 10% and 8% to overall mortality respectively. (From experience working with mortality data, we tend to say that in a good-quality vital registration system, the contribution of residual categories should not exceed 10%.)

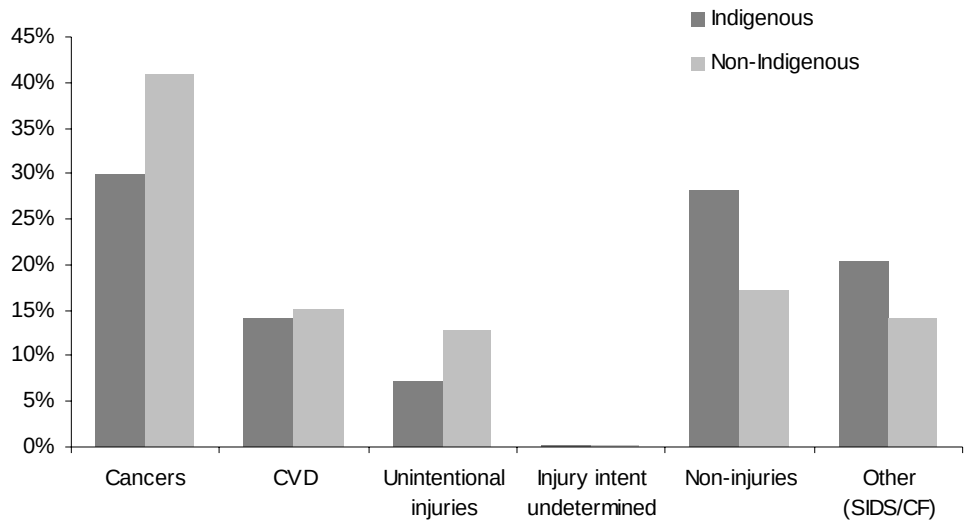


Figure A.6: Burden of disease ill-defined categories as a proportion of overall mortality by Indigenous status 2000–02

Finally, the standardised proportions of deaths by broad cause clusters (group 1, communicable disease, neonatal and maternal causes; group 2, non-communicable diseases; and group 3, injuries) and Indigenous status showed how similar the cause of death structure is for deaths identified as Indigenous and those identified as non-Indigenous (Figure A.7).

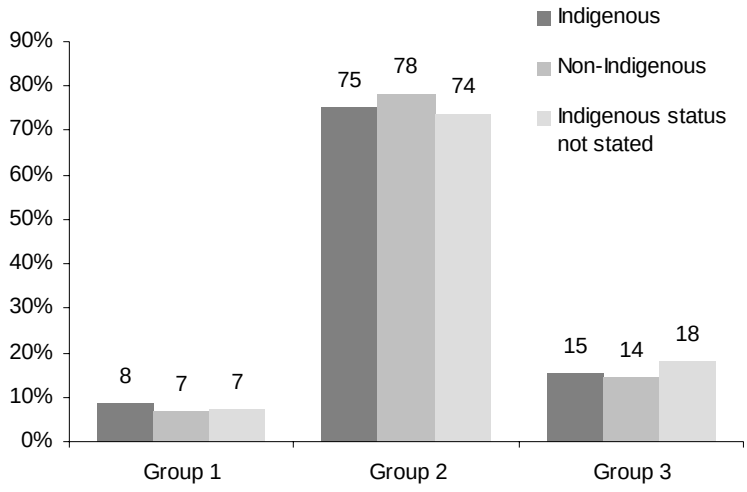


Figure A.7: Proportion of deaths in broad cause categories by Indigenous status, 2000–02

Appendix B Australian hospital data adjustment

Throughout this report, we use the term 'Australian hospital data' as shorthand for the AIHW's National Hospital Morbidity Database (AIHW 2003b). This dataset is a compilation of episodes of care (or 'separations') and includes people who were admitted to a hospital in Australia for a particular condition or procedure at least once in 2001–02 and 2002–03. The following three issues must be taken into account when using Australian hospital data to describe and compare the health status of Indigenous Australians:

- the under-identification of Indigenous Australians in population health datasets
- the utility of hospital data in general as an indicator of disease occurrence
- the plausibility of relativities by remoteness and for major diseases given adjustments for under-identification in Indigenous status.

These three issues are explained in the following sections.

B.1 Quality of Indigenous identification in Australian hospital data

Australia has a universal system of access to hospital services and records all episodes of care in hospital. However, a number of studies have shown that Indigenous identification in hospitals is under-reported and as a result, true hospitalisation rates for Indigenous Australians are likely to be higher than what is observed (AIHW 2005d).

The AIHW has developed correction factors for Indigenous under-identification in hospital data (see Table B.1). These assessments are based on consultation with state and territory health authorities and include a range of inputs, such as review of separation numbers, detailed internal data consistency checking, and formal data quality studies (AIHW 2005d). The Northern Territory and South Australia are thought to have complete Indigenous identification, with Western Australia being very close. The completion of Indigenous identification decreases from Queensland to Victoria to New South Wales. All together, the adjustment factors implied a national under-identification factor of 16% in 2001–02 (AIHW 2005b).

B.2 AIHW correction factors

The AIHW has developed correction factors for Indigenous under-identification in hospital data for each state and territory for use in their *Expenditures on health for Aboriginal and Torres Strait Islander peoples* report (AIHW 2005b). They also have estimates by remoteness from a New South Wales record linkage study. The approach, recommended by the AIHW, was to adjust the correction factors by remoteness (Table B.2) on a pro-rata basis by state and territory to equal the overall state and territory correction factors (Table B.1) (John Goss, email, 17 January 2006).

Table B.1: Under-identification factors by jurisdiction used for estimating hospital expenditure for Indigenous patients by the AIHW (2001–02)

State/territory	Under-identification adjustment[(a)]	Derived completeness (1/under-identification adjustment) (%)
NT	NIL[(b)]	100
SA	NIL[(b)]	100
WA	1.06	94
Qld	1.20	83
Vic	1.25	80
NSW	1.30	77
ACT	1.30	77
Tas	NIL	According to expenditures report, the quality is thought to be too poor to estimate a correction factor

(a) *Source:* Table A5.4 in Appendix A of the *Expenditures on Health for Aboriginal and Torres Strait Islander Peoples 2001–02* report (AIHW 2005b:42)
(b) For those states and territories where no under-identification adjustment was made, the not stated responses were distributed between Indigenous and non-Indigenous patients according to the proportion of identified responses

B.2.1 The New South Wales record linkage study

The New South Wales Health Department assessed the under-identification of Indigenous status by statistically matching individual patients within annual sets of data from its hospital separations data collection. Linked separations were identified for all Indigenous patients, and an estimate of under-identification was derived from Indigenous patients with multiple separations by determining the number of these linked separations that had Indigenous status recorded as 'other than Indigenous'. However, there are several limitations to this work. First, in the absence of a unique and universal patient identifier, comparison of data for multiple patient episodes relies on the precision of the record linkage methods. Second, this method assumes that no individual is incorrectly identified as Indigenous. A Victorian data linkage exercise found that some patients reported as Indigenous were probably incorrectly identified. Potentially, even very low rates of random recording error for non-Indigenous people could outweigh any systematic under-identification of Indigenous patients. Third, the New South Wales methodology assumes that, in theory, recording Indigenous status at admissions is an independent event. However, in practice, this is not the case because once someone is identified as Indigenous at a hospital, they are more likely to have this status marked on their admission record for subsequent admissions (AIHW 2005b, 2005d).

Table B.2 presents the AIHW's unpublished work on correction factors by the ASGC remoteness areas. The second column shows the correction factor (from the New South Wales record linkage study) that is applied to the admissions data in *each hospital's remoteness area* in New South Wales to bring Indigenous admissions up to the 'true levels' of Indigenous admissions. The fourth column shows the correction factor required to adjust for under-identification by the patient's usual residence of remoteness. This correction factor was derived by the AIHW using the 10-year-old linked New South Wales study in conjunction with 2003–04 New South Wales hospital separation data for Indigenous people, assuming that there have been no changes in Indigenous under-identification practices since the New South Wales study was conducted (John Goss, email, 17 January 2006).

Table B.2: Under-identification factors by remoteness used for estimating hospital expenditure for Indigenous patients by the AIHW (2001–02)

Remoteness	Under-identification adjustment by remoteness of hospital	Derived completeness by remoteness of hospital (%)	Transformed under-identification adjustment by patients usual residence of remoteness	Derived completeness by patient's usual residence by remoteness (%)
Major cities	1.83	55	1.79	56
Inner regional	1.25	80	1.35	74
Outer regional	1.21	83	1.27	79
Remote	1.12	89	1.21	83
Very remote	1.04	96	1.15	87

The completeness of Indigenous identification in New South Wales is highest in remote areas and lowest in major cities. This is in keeping with the finding that Indigenous identification has been found to be more accurate in areas where Indigenous Australians make up a larger proportion of the population, and vice versa. The AIHW advise that only 51% of separations of Indigenous Australians residing in remote areas take place in remote or very remote hospitals. Thus the remote adjustment factor changes from 1.12 (when it is based on remoteness of the hospital) to 1.21 (when the additional adjustment is made for remoteness of the patient's usual residence). Table B.3 shows rate ratios for the top three non-fatal conditions for which we use hospital relativities before and after adjustment.

Table B.3: Rate ratios for leading three non-fatal conditions based on observed and adjusted hospital data

	Observed Indigenous versus total Australian population			Adjusted Indigenous versus total Australian population		
Disease category	All	Non-remote	Remote	All	Non-remote	Remote
Type 2 diabetes						
Males	6.1	5.7	7.0	6.7	6.5	7.1
Females	8.8	7.4	12.3	9.7	8.5	12.5
COPD						
Males	3.6	3.1	4.6	4.1	3.8	4.7
Females	4.9	4.2	6.6	5.6	5.1	6.7
Ischaemic heart disease						
Males	2.1	2.1	2.1	2.4	2.6	2.1
Females	3.7	3.7	3.8	4.3	4.8	3.8

B.3 Plausibility of corrected hospital data

The correction factors reflect the average level of under-identification of Indigenous patients across a given group of separations. Therefore, the use of these factors might be problematic in disease specific analyses. Annex Table 1 contains a complete list of diseases and injuries for which hospital relativities have been used to estimate disease occurrence (this table summarises the primary data sources for incidence and prevalence).

There is a paucity of data comparing under-identification for Indigenous status by disease. One disease category that has received attention is injuries. Three studies of injury hospitalisations for Indigenous people have highlighted the issue of under-identification of Indigenous status (Gladman et al. 1997, Hockey et al. 1999, Mid North Coast Aboriginal Health Partnership 2001). For instance, the New South Wales Mid North Coast Aboriginal Injury Surveillance Project found that, based on the information recorded by the accident and emergency department, Indigenous Australians had twice the injury rate of the non-Indigenous population. However, after correcting for under-identification using independent assessors, the increase was six-fold. The Queensland Health validation study of the emergency department information system for injury surveillance showed that, in two Queensland hospitals in 1999, 3% of presentations were Indigenous compared with only 1% of presentations that were coded as Indigenous (Hockey et al. 1999). A 12-month case note audit of daily clinic register from one remote community in the 1996–97 study of injury in five Cape York communities found that only 0.15% of total injury events resulted in death and less than 8% of injury events resulted in a hospital admission. Despite this, routine hospital data showed that the people from Cape York Aboriginal communities were admitted to hospital for injury at 3–4 times the rate of other Queenslanders (Gladman et al. 1997) . However, it is not known how the completeness of Indigenous identification for injuries compares with hospital admissions for other diseases. These studies are described below.

B.4 Using hospital data for burden of disease

Apart from a limited number of 'acute' conditions, for which, it is reasonable to assume that the majority of people seek treatment (such as stroke or heart attack, neonatal and maternal conditions, and injuries requiring immediate intervention), hospital separations say little about the actual incidence or prevalence of a condition in the community (AIHW 2005b). Moreover, hospital separations are influenced by factors such as rates of re-admission (which may differ across conditions, groups of people and geographical areas, depending on admission practices and levels and patterns of service provision), and transfers within and between hospitals (Victorian Department of Human Services 2005). A record is included for each separation, rather than for each patient, so patients who were admitted more than once a year have more than one record in the database. Despite this, hospital data can provide insights into the health of the population who use hospitals (AIHW 2005b). For this study, we assumed that we can use differentials in hospitalisation rates as an estimate of the relative difference in disease occurrence between Indigenous Australians and the total Australian population, even for diseases for which hospitalisation rates do not accurately represent the occurrence of disease within the community. Therefore, this method assumes that the likelihood of being hospitalised for a given disease is similar for Indigenous people and the total Australian population.

The first step in our hospital analyses was to map all mentions of diseases (principal diagnosis and multiple secondary conditions) for an episode of care to burden of disease categories. After adjusting for Indigenous identification issues, decisions about using relativities for the Indigenous population were made on a disease-by-disease basis after comparing the plausibility of the age- and sex-specific graphs of hospital separations between the Indigenous and the total Australian population. If the age- and-sex-specific hospitalisation patterns were similar, an overall age-standardised rate ratio was used. If the patterns were different, age- and sex-specific relativities were applied to the national model

(Begg et al. 2007) instead. Ideally, hospital relativities were used when they appeared plausible or showed consistency with other data we had on these conditions.

Appendix C Methods for estimating non-fatal burden

In this section we describe our methods for calculating YLD for a number of specific diseases and their complications. Only those diseases for which an explicit model was developed are discussed (see Annex Table 1 for an outline of the data sources used for each condition, and whether or not an explicit model was developed). In the event an explicit disease model could not be developed for a condition, rate ratios were applied to the National Study as outlined in Annex Table 1. A more general discussion of the methods for estimating non-fatal health outcomes is provided in Chapter 3.

Unless otherwise specified, all the disease and injury models for Indigenous Australians used the same case definitions, disease stages, disability weights and complications as the National Study, *The Burden of Disease and Injury in Australia, 2003* (Begg et al. 2007).[1] Many of the modelled diseases have relevant information about disease occurrence (i.e. incidence or prevalence) in the Indigenous Australian population, but not for other disease parameters. For these diseases, we used the same assumptions for remission and case-fatality as the National Study.

Rate ratios derived from proportions and rates using Australian hospital, mortality and notification data were standardised for age. Age-standardisation removes the confounding effects of age from comparisons of different populations. This is important because the Indigenous population has a much younger population age distribution than the total Australian population. Because of the paucity of even basic epidemiological information on some of the conditions that were analysed for Indigenous Australians, as new data becomes available the models we present can be refined. Notwithstanding this caveat, the methods described below make what we consider to be the most extensive and critical use of health information for Indigenous Australians to date. Table C.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.

In the following sections, broad cause groups are classified with a number to identify broad cluster groups (communicable diseases, maternal, neonatal and nutritional conditions; non-communicable diseases; and injuries) and letter to identify broad cause group. The same classification is used in the annex tables and in *The Burden of Disease and Injury in Australia, 2003* (Begg et al. 2007).

1 For example, we model new disability weights for 'asthma', 'epilepsy' and 'cataract' and also include the modelling of a new disease 'foetal alcohol syndrome'

Table C.1: List of common name abbreviations

Common name	Full name
1980 National Trachoma Program	1980 National Trachoma and Eye Health Program (Royal Australian College of Ophthalmologists 1980)
1994–96 National Dental Telephone Survey	*1994-96 National Dental Telephone Interview Survey*(Brennan & Carter 1998)
1995–96 Adult dental survey	1995–96 Prospective Adult Dental Programs Survey (Brennan & Carter 1998)
2001/2004–05 National Health Survey	2001 National Health Survey — Indigenous (ABS 2006a) 2004–05 National Aboriginal and Torres Strait Islander Health Survey (NATSIHS)
2004-06 National oral health survey	The National Survey of Adult Oral Health 2004–06 (Slade et al. 2007)
Australian child wish data	Fertility Decision Making Project (Weston et al. 2004)
Australian general practitioner data	2000–01 Bettering the Evaluation and Care of Health (BEACH)
Australian hospital data	2001–03 National Hospital Morbidity Database (AIHW 2003b)
Australian mortality data	2001–03 Cause of Death dataset
Australian notification data	2001–03 National Notifiable Diseases Surveillance System, age- and sex-specific incident cases for Australians by Indigenous status and remoteness provided electronically by Communicable Diseases Australia except for HIV/AIDS, which is from the 2003 National Centre for HIV Epidemiology and Research (National Centre in HIV Epidemiology and Clinical Research 2004)
Disability weight regression model	Regression model of Dutch disability weights which requires inputs of health state description based on the six domains of the Euroqol 5D+
DisMod	DisMod version II
GBD study	*Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factory in 1990 and Projected to 2020* (Murray & Lopez 1996)
National model	Disease model from *The Burden of Disease and Injury in Australia, 2003* (Begg et al. 2007)

1A Infectious and parasitic diseases

We use the term 'Australian notification data' to refer to infectious disease notifications from the Australian Government's Department of Health and Ageing - Communicable Diseases Network Australia National Notifiable Diseases Surveillance System (NNDSS) for 2001-03. The NNDSS consists of the national surveillance of more than 50 communicable diseases. The Australian Government's Department of Health and Ageing website notes that:

> The quality and completeness of data compiled in the National Notifiable Diseases Surveillance System are influenced by various factors. Notifications may be required from treating clinicians, diagnostic laboratories or hospitals. In addition, the mechanism of notification varies between States and Territories and in some cases different diseases are notifiable by different mechanisms. The proportion of cases seen by health care providers which are the subject of notification to health authorities is not known with certainty for any disease, and may vary among diseases, between jurisdictions and over time (Australian Government Department of Health and Ageing 2005).

For non-fatal disease modelling purposes, we calculated incidence rates for the following conditions: chlamydia, gonorrhoea, whooping cough, haemophilus influenzae type b, dengue, other arbovirus infection, hepatitis A, malaria, Ross River virus, Barmah Forest virus and acute hepatitis B. Missing data (such as remoteness, age, sex and Indigenous status) was redistributed proportionately.

Tuberculosis

We estimated the incidence of tuberculosis for the total Indigenous Australian population using published 2003 age- and sex-specific notification rates (Li et al. 2004). We estimated the incidence of tuberculosis for Indigenous Australians by remoteness using rate ratios based on admission rates from Australian hospital data (AIHW 2003b).

Sexually transmitted diseases (excluding HIV/AIDS)

Chlamydia and gonorrhoea

We estimated the incidence of chlamydia and gonorrhoea for Indigenous Australians residing in non-remote and remote areas by applying age and sex specific rate ratios from Australian notification data. We adjusted the incidence of the complication infertility for 'the wish to have a child' from the Fertility Decision Making Project (Weston et al. 2004).

HIV/AIDS

Similar to the national model (Begg et al. 2007), we modelled HIV as a progressive condition with four stages: (1) asymptomatic HIV; (2) symptomatic HIV; (3) AIDS before terminal phase; and (4) terminal AIDS. Using Australian notification data, we assumed that the incidence of HIV and AIDS in the Indigenous population was the same as in the total Australian population (National Centre in HIV Epidemiology and Clinical Research 2004). We modified the incidence of HIV and AIDS to reflect the different sex distribution (HIV — 71.4% males and AIDS — 83.4% males) reported for the Indigenous population. To estimate the duration of HIV and AIDS in the Indigenous population, we adjusted the durations for the total Australian population downwards using a ratio (of the total Australian population life expectancy by age to the Indigenous life expectancy by age) to account for the reduced life expectancy in Indigenous Australians. We assumed that the duration of terminal AIDS was the same as in the total Australian population.

Tetanus

We estimated the incidence of tetanus by applying the published age-standardised rate ratio for the total Indigenous population in 2000–02 to the total Australian population incidence (Menzies et al. 2004). We assumed the same incidence for Indigenous Australians residing in non-remote and remote areas.

Childhood immunisable diseases

Measles

We estimated the incidence of measles by applying the published rate ratio for the total Indigenous population in 2000–02 to the total Australian population incidence (Menzies et al. 2004). We assumed the same incidence for Indigenous Australians residing in non-remote and remote areas. Based on published data and Australian hospital data (AIHW 2003b), we assumed no complications from measles occurred in the Indigenous population in 2003.

Septicaemia

We estimated the incidence of septicaemia for Indigenous Australians residing in non-remote and remote areas using Australian hospital data.

Hepatitis B

We estimated the incidence of acute hepatitis B for Indigenous Australians residing in non-remote and remote areas by applying age-standardised rate ratios, based on Australian notification data, to the total Australian population (National Centre in HIV Epidemiology and Clinical Research 2004). We assumed that all infections reported as incident were symptomatic. Notification data do not capture the proportion of infants infected in the perinatal period by hepatitis B-positive mothers or the larger number of children who become infected in childhood. Therefore, we estimated the incidence of acute hepatitis B infection in infants by applying published probabilities of perinatal transmission for Indigenous mothers to birth data (Kaldor et al. 1996). Based on the literature, we assumed a 40% probability of transmission if exposed. Using this estimate, we calculated the number of infants who would be infected in the absence of vaccination (Kaldor et al. 1996). We then adjusted our estimates downwards on the basis that current vaccination coverage in children born to 'at-risk' mothers is 95% (Menzies et al. 2004). Similarly, we adjusted the number of perinatal infections downwards for the probability of symptomatic infection, which is 5%. Based on expert opinion, we assumed a similar number of infections by casual contact in childhood as from perinatal transmission.

We based our estimates of chronic hepatitis B on a series of DisMod models. First, we estimated the prevalence of adults with chronic hepatitis B by adjusting the age-specific prevalence from the national model (Begg et al. 2007) by the crude rate ratio of Indigenous Australian to Australian prevalence using published figures of 2.3% and 8.2% respectively for Indigenous Australians residing in non-remote and remote areas (Kaldor et al. 1996) and the overall prevalence estimate from the Australian model. We scaled the age-specific estimates by a constant so that our overall prevalence estimates for Indigenous Australians residing in non-remote and remote areas equalled the published estimates. We assumed the same low spontaneous remission (0.5%) and overall relative risk of mortality (1.5) as in the national model (Begg et al. 2007). Then we estimated the prevalence of adults with chronic hepatitis B using incidence estimates from perinatal and casual childhood transmission if no vaccination had occurred. We again assumed the same remission and relative risk of mortality as the national model (Begg et al. 2007). Finally, we subtracted the prevalence of adult carriers from childhood infections (second model) from the prevalence of all adult carriers (first model) so we could 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 only recently an impact of vaccination on 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, we considered this to be the most plausible method of modelling the disease following expert consultation. We assumed the disability weights as per the national model (Begg et al. 2007). The methods we used to derive the incidence of hepatitis B-related cirrhosis and liver cancer are described in the respective sections for these diseases.

Hepatitis C

Due to the asymptomatic nature of hepatitis C infection, we assumed that all disability is a result of complications (i.e. cirrhosis and/or liver cancer). The methods we used to derive the incidence of hepatitis C-related cirrhosis and liver cancer are described in the respective sections for these diseases.

Trachoma

We estimated the prevalence of visual impairment resulting from trachoma infection using data from the 1980 National Trachoma Program (Royal Australian College of Ophthalmologists 1980). Based on expert advice, we assumed that trachoma infection is only a problem in remote Australia and we adjusted the prevalence of related visual impairment downwards by one third to account for observed decreases in the prevalence of the trachoma scarring stage since the national survey was conducted (Landers et al. 2005, Mak & Plant 2001). In the absence of more specific information, we assumed the following:

- trachoma proportions by age were the same as those reported for corneal disease
- mild and moderate vision loss have the same cause distribution by age as severe vision loss
- 'poor' vision is equivalent to our definition of moderate vision loss
- the difference between the pool of people without good vision and those with poor vision and severe vision loss is equivalent to the group of people with mild vision loss.

We made minor adjustments to the prevalence of each stage by age to ensure plausibility and to reflect published estimates. We estimated the incidence and duration of trachoma-related visual impairment in DisMod using our derived prevalence estimates. We initially modelled the prevalence of severe vision loss in DisMod assuming no remission and a relative risk of mortality of 1. We then used 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 (i.e. more accurate average durations than using remission as remitted cases in the DisMod model, which continues to be subject to the hazard of incidence). Similarly, we used the incidence of moderate vision loss as 'mortality' in mild vision loss. Based on expert advice, we assumed remissions of zero for all three stages of trachoma-related vision loss. Based on expert advice, trachoma is only present in remote communities north and west of Port Augusta, South Australia, excluding coastal communities. We therefore may have over-estimated the overall prevalence of trachoma since we apply the estimated prevalence (described above) to the entire population of Indigenous Australians residing in remote areas.

1B Acute respiratory infections

Otitis media

We modelled the following stages of otitis media, acute infection, bilateral chronic infection and lifelong deafness. Indigenous general practice encounters for acute otitis media acute occurred at twice the rate of Australian encounters (Britt et al. 2001). Therefore, we increased the national model (Begg et al. 2007) incidence by a factor of 2. We assumed that Australian general practitioner data is not representative of Indigenous Australians residing in remote areas, and estimated the incidence of acute infection in Indigenous Australians residing in remote areas using a factor of 10, based on findings from the 1980 National Trachoma Program (Royal Australian College of Ophthalmologists 1980). We used the disability weight regression model to derive weight of 0.090 and assumed a duration of one week.

We estimated the prevalence of chronic otitis media in Indigenous Australians residing in non-remote areas based on the number of Indigenous people reporting otitis media as a long-term health problem in the 2001 National Health Survey (ABS 2003a). We adjusted the prevalence of chronic infection downwards by one third to account for bilateral cases only, using a ratio of bilateral to unilateral cases from the 1980 National Trachoma Program (Royal Australian College of Ophthalmologists 1980). We estimated the prevalence of bilateral chronic otitis media in Indigenous Australians residing in remote areas from the 1980 National Trachoma Program and assumed that the epidemiology of bilateral chronic otitis media had not changed since the survey was undertaken. We derived the incidence and duration of bilateral chronic otitis media in DisMod using prevalence, a relative risk of 1 and remission equivalent to 3 years duration for Indigenous Australians based on Australian data (McGilchrist & Hills 1986). For chronic infection, we applied the Dutch weight for early acquired mild to moderate hearing loss (0.110).

We based our estimates for permanent hearing loss resulting from acute infections on the Global Burden of Disease Study (Murray & Lopez 1996), which estimated that 5 in 100 000 cases of acute otitis media in 0–14 year-olds results in permanent hearing loss. For the small number of cases that experience life-long deafness, we used the Dutch weight for early acquired severe hearing loss (0.233).

1C Maternal conditions

We estimated the incidence of maternal conditions for Indigenous Australians residing in non-remote and remote areas by applying age- and sex-specific hospital rate ratios for the combined condition and complications state (e.g. rate ratios for hospital separations with a diagnosis of both maternal haemorrhage and acute post-haemorrhagic anaemia) to the national model (Begg et al. 2007). We adjusted the incidence of infertility as a complication, in the abortion and maternal sepsis models, for 'the wish to have a child' (as described above in section on sexually transmitted diseases).

1D Neonatal causes

Low birth weight

We estimated the incidence of low birth weight in Indigenous infants using published data from the National Perinatal Statistics Unit, and various state and territory perinatal collections. According to National Perinatal Statistics Unit (Laws & Sullivan 2005), 2.4% and 10.6% of infants born to Aboriginal or Torres Strait Islander mothers had a birth weight (<1500 g and 1500–<2500 g respectively), giving an incidence of around twice the non-Indigenous rate. To estimate the sex distribution of low birth weight Indigenous infants, we used Australian hospital data (AIHW 2003b). We excluded the low birth weight neonatal deaths, applying the same sex and birth weight distribution as the national model (Begg et al. 2007) to the published Indigenous neonatal mortality rate (5.6/1,000 live births) (Laws & Sullivan 2005). To estimate the incidence of low birth weight for Indigenous infants residing in non-remote and remote areas, we applied the rate ratios from Australian hospital data to our overall rate of Indigenous low birth weight. We estimated the incidence of low birth weight complications proportionate to the higher incidence of low birth weight in Indigenous infants compared with the total Australian population. The methods used assumed that the probability and distribution of low birth weight complications among low birth weight survivors is the same for Indigenous and non-Indigenous Australians.

Birth trauma & asphyxia

We estimated the incidence of birth trauma-related hearing loss, seizure and cerebral palsy without intellectual disability in the Indigenous population by remoteness by applying indirect age-standardised hospital rate ratios to the incidence of these complications in the total Australian population. We assumed that the remission, case-fatality and disability weights of severe hearing loss, seizure and cerebral palsy without intellectual disability was the same as those with mild intellectual disability. We used the estimates of intellectual disability due to birth trauma from the overall calculations for intellectual disability by all underlying causes (see Section 2K).

Neonatal infections

We estimated the incidence of acute neonatal infections and the associated complications (severe hearing loss and motor deficit) by applying indirect age-standardised rate ratios by remoteness from Australian hospital data (AIHW 2003b) to the national model (Begg et al. 2007). We used the estimates of intellectual disability due to neonatal infections from the overall calculations for intellectual disability by all underlying causes (see Section 2K).

2F Malignant neoplasms

We modelled the incidence of all cancers using rate ratios from Australian hospital data and applied a 50% higher case fatality rate based on the literature.

Previous studies used 1997–2002 Queensland cancer registry data to show that Indigenous people were 1.5 times more likely to die from cancer than non-Indigenous people (after adjusting for age, sex, year of diagnosis, remoteness index, type of cancer and stage of cancer) (Valery et al. 2006). This finding built on earlier studies, which found that Indigenous

people from the Northern Territory were 1.9 times more likely to die from cancer (after adjusting for age at diagnosis and sex) than the total populations of Western Australia and Tasmania for 1991–2001 (Condon et al. 2005).

Liver cancer

We estimated the underlying causes of liver cancer (alcohol, other, hepatitis B and hepatitis C) using proportions derived in the Indigenous cirrhosis model (see Section 2N).

2H Diabetes

Diabetes cases

We based the incidence of 'all diabetes' (Type 1 and 2 diabetes combined) in the Indigenous population on a DisMod model of prevalence (7.2% — males and 9.5% — females), relative risk of all-cause mortality of two, zero remission and a 5% annual incidence trend derived from a systematic review of the literature (Mott 2005). The predicted prevalence was based on a multiple regression analysis of pooled data from 21 studies spanning 1982–2001, after accounting for variance in diabetes diagnostic criteria, study size, year of study, age at study commencement, remoteness and ethnicity. The relative risk of all-cause mortality was based on a 1986–91 cohort study of Indigenous people with diabetes from central Australia compared with the Northern Territory Indigenous population (Phillips et al. 1995). A plausible age distribution of this relative risk was derived by scaling it to age-specific findings from an international meta-analysis (Woodward et al. 2003).

Based on the literature, we assumed that the epidemiology of Type 1 diabetes and its complications for Indigenous Australians is the same as in the total Australian population (Office for Aboriginal & Torres Strait Islander Health Services 1998, Verge et al. 1994). In the absence of specific information, we also assumed that there are no differentials in the incidence of Type 1 diabetes and its complications for Indigenous Australians by remoteness.

We estimated the incidence of Type 2 diabetes in Indigenous Australians by subtracting the incidence of Type 1 diabetes from the incidence of 'all diabetes' in Indigenous Australians. We estimated the incidence of Type 2 diabetes for Indigenous people residing in non-remote areas by applying rate ratios from Australian hospital data (AIHW 2003b) to the total Indigenous population incidence for Type 2 diabetes. We modelled the incidence of Type 2 diabetes for Indigenous Australians residing in non-remote areas in DisMod assuming zero remission, the relative risk of all cause mortality output from the Type 2 diabetes DisMod model for all Indigenous Australians and the 5% annual incidence trend. We made separate estimates for the incidence of Type 2 diabetes for Indigenous people residing in remote areas by applying rate ratios from Australian hospital data.

Complications

We estimated the incidence of the following Type 2 diabetes-related complications for Indigenous Australians residing in non-remote areas

- retinopathy
- neuropathy
- peripheral vascular disease
- diabetic foot, amputations

- renal failure
- cataract
- glaucoma
- ischaemic heart disease
- stroke.

We estimated the incidence of all Type 2 diabetes complications for Indigenous Australians residing in remote areas by applying rate ratios of hospital separations between remote and non-remote areas.

Retinopathy, neuropathy and peripheral vascular disease

To estimate the incidence of Type 2 diabetes-related retinopathy, neuropathy and peripheral vascular disease among Indigenous Australians residing in non-remote areas, we adjusted the total Australian population estimates using rate ratios of the incidence of Type 2 diabetes in the non-remote residing Indigenous population compared with the total Australian population.

Amputations and diabetic foot

We estimated the incidence of diabetes-related amputations using age- and sex-specific Australian hospitalisation rates, assuming all procedures represented incident cases. As per the total Australian model, we assumed that for every amputee there are 20 cases of diabetic 'foot' (where poor circulation and neuropathy lead to gangrene). This assumption was supported by literature from central Australia, which showed that that 7% of separations for diabetic foot required amputation of at least a toe (Ewald et al. 2001). We modelled the incidence of toe and foot/leg amputations using zero remission and assuming a relative risk of all cause mortality two times the excess mortality risk of Type 2 diabetes that Indigenous Australians residing in non-remote areas experience to reflect the risk in the more advanced stages of disease.

Renal failure

We estimated the incidence of Type 2 diabetes-related dialysis and kidney transplants for Indigenous Australians using 2001–03 data from the Australian and New Zealand Register of Dialysis and Renal Transplants (Excell & McDonald 2005).[2] To estimate the comparable incidence for Indigenous Australians residing in non-remote areas we applied rate ratios from Australian mortality data.

For dialysis, we estimated remission as the proportion of cases that go on to receive a transplant: 4.5% for those younger than 65 years for Type 2 diabetes and 0% for those over 65 years with Type 2 diabetes. We assumed that the relative risk of all causes of mortality for Type 2 diabetes-related dialysis was twice the excess mortality risk of Type 2 diabetes that Indigenous Australians residing in non-remote areas experience. For transplants, we estimated the case-fatality for Indigenous Australian diabetics residing in non-remote areas who undergo transplants using the methodology outlined in the nephritis & nephrosis model (see Section C12.1, below). We estimated the remission (8.6%) for cases of kidney transplants by applying a rate ratio for Indigenous Australians from McDonald (McDonald 2004). Case fatality rates for kidney transplants in Indigenous Australians were also from

[2] The data reported here have been supplied by the Australia and New Zealand Dialysis and Transplant Registry. The interpretation and reporting of these data are the responsibility of the editors and in no way should be seen as an official policy or interpretation of the Australia and New Zealand Dialysis and Transplant Registry.

published data (McDonald 2004). We used ANZDATA mortality to adjust for the differences in diabetics and non-diabetics and we used rate ratios from Australian mortality data to estimate the case-fatality rate for Indigenous people residing in non-remote areas.

Other complications

We estimated the proportion of cataract, glaucoma, ischaemic heart disease and stroke YLD respectively in the Indigenous population attributable to Type 2 diabetes using Indigenous prevalence figures and relative risks from the national model (Begg et al. 2007).

2J Mental disorders

Harmful alcohol use & dependence

In the absence of survey data on ICD-10 or DSM-IV-defined alcohol dependence, we used relativities of risky/high-risk alcohol consumption from the 2004–05 National Health Survey as a proxy (ABS 2006a). Alcohol risk levels in this survey were derived from the average daily consumption of alcohol in the seven days before the interview and were grouped into the following levels as defined by the NHMRC:

- low (males <4 drinks; females <2 drinks)
- risky (males 4–5.6 drinks; females 2–4 drinks)
- high risk (males >6 drinks; females >4 drinks).

Risk level as defined by the NHMRC is based on regular consumption levels of alcohol, whereas indicators derived in the 2004–05 National Health Survey *do not take into account whether consumption in the reference week was more, less or the same as usual* (ABS 2006a).

The 2004–05 National Health Survey shows that, after adjusting for age differences, the proportion of Indigenous adults who reported drinking at risky/high-risk levels (15%) was similar to that of non-Indigenous adults (14%), although relative risks based on age-specific proportions showed slightly elevated risks for Indigenous people across most of the age groups (ABS 2006a). This relative relationship was also observed in the 2001 National Health Survey, although the absolute differences were lower for the Indigenous and non-Indigenous population (ABS 2006a). In 2004–05, there was also no statistically significant difference in risky/high-risk alcohol consumption between Indigenous Australians residing in non-remote (17%) and remote (15%) areas (ABS 2006a). This finding was the opposite of what was observed for the 2001 National Health Survey, which found Indigenous people residing in remote areas (18%) had higher levels of risky/high-risk drinking than Indigenous people residing in non-remote areas (10%; age-standardised RR of 1.8) (ABS 2006a).

The age-standardised relative risks from the 2004–05 National Health Survey for risky/high-risk alcohol consumption between Indigenous and non-Indigenous Australians, and Indigenous Australians residing in non-remote and remote areas differ greatly from age-standardised rate ratios derived from Australian hospital (RR 9–10) and mortality (RR 10–13) datasets for alcohol use disorders. Australian general practitioner data also suggest a higher risk (RR 2.6) for Indigenous versus non-Indigenous Australians who drank six or more drinks at least weekly (Proude et al. 2006). The 2004 National Drug Strategy Household Survey, which also used NHMRC alcohol consumption guidelines, found that Indigenous Australians had elevated short-term (RR 1.9) and long-term (RR 2.3) risks for risky/high-risk alcohol consumption compared with the non-Indigenous population (AIHW 2005a). It is not clear why the results from the 2004–05 National Health Survey are so different from these

other potential proxy data sources of alcohol dependence. It would be interesting to further stratify the 2004–05 National Health Survey results using the question, 'Is the amount you drank last week more, about the same, or less compared with most weeks?' and analyse the responses for the 'about the same' group. Conversely, the 2004–05 National Health Survey (ABS 2006a) interviewed nearly 10 500 Indigenous people compared with around 500 Indigenous respondents from the survey, *Statistics on Drug Use in Australia 2004* (ABS 2006a, AIHW 2005e). In addition, the National Drug Survey asked respondents to recall their alcohol consumption for the previous day only. It is unknown which method would provide more reliable results. Some researchers prefer the 2001 or 2004–06 National Health Surveys to the 2004 National Drug Survey for comparative risk factor assessment models in the total Australian population 'because of the more specific nature of the information collected (seven categories of alcoholic drinks) and the fact that adjustments were made for the alcohol content of a variety of brands within these categories' (Ridolfo & Stevenson 2001). Another source of potential uncertainty in self-report surveys may be the sensitive nature of questions about alcohol consumption.

As a compromise between the differing findings, we assumed that Indigenous Australians have a similar occurrence of alcohol dependence as the total Australian population but with different consequences by remoteness. Therefore, we estimated the prevalence of alcohol dependence in Indigenous Australians by applying derived age- and sex-specific rate ratios of risky/high-risk alcohol consumption for Indigenous Australians by remoteness from published 2004–05 National Health Survey data to the national model (Begg et al. 2007). It is likely that the large hospital and mortality rate ratios in part reflected the consequences of acute alcohol intoxication (such as injuries). We believe that the indirect age-standardised mortality rate ratios for cirrhosis are more indicative of the chronic long-term harms associated with alcohol dependence than the rate ratios for alcohol dependence. However, since the cirrhosis mortality risk also reflect hepatitis-related cirrhosis, we adjusted the cirrhosis relative risks downwards by one half (non-remote: male RR 2.4, female RR 4.3; remote: male RR 4.2, female RR 5.7). We then applied these predicted differentials by sex and remoteness to the total Australian population elevated mortality risks (1.8 in males and 3.8 in females) for alcohol dependence, to estimate the relative risk of excess mortality in Indigenous Australians.

Foetal alcohol syndrome

We used the estimates of intellectual disability by severity and underlying cause to model the intellectual disability related to foetal alcohol syndrome (see Section 2K). According to the approach outlined in Section 2K for estimating the epidemiology of intellectual disability in Indigenous Australians (after rescaling and assuming that all intellectual disability due to intoxication is alcohol), the prevalence of foetal alcohol syndrome in Indigenous infants for 1953–2000 was 0.29% (Glasson et al. 2005). This figure is similar to other published estimates of foetal alcohol syndrome among Indigenous infants from Western Australia (Bower et al. 2000, Leonard et al. 2003).

Harmful cannabis use & dependence

Since the hospital rate ratios for cannabis dependence did not seem plausible we derived relative risks based on the proportions of Indigenous people reporting cannabis use in the last 12 months (from the 1994 Indigenous NDSHS) compared with the proportions of the total population reporting cannabis use in the past 12 months from the (2001 NDSHS) by sex,

based on the assumption that patterns of current use have not changed markedly over time. This is supported in the total population with trend data over time from the 1993-2001 National Drug Strategy Household Surveys (AIHW 2003c). For the Indigenous population, the 2002 NATSISS also suggests that patterns of current use have not changed over time since the 1994 NDSHS-ATSI; although it is important to note that the NATSISS is not conducted in remote areas (Gray et al. 2004, Hunter 2006). We then used hospital relativities to estimate the occurrence of cannabis dependence by remoteness. Because the hospital rate ratio of 0.5 for remote areas was not thought to be plausible we applied the hospital rate ratio for remote areas to the excess risk from the national drug survey results.

Harmful other drug use & dependence

For other drug dependence the hospital relativities did not seem plausible. Instead we used a relative risk of 1.6 based on current use of an illicit drug from the National Drug Strategy Household Survey (Gray et al. 2004). We then used hospital relativities to estimate the occurrence of other drug dependence by remoteness. Because the hospital rate ratio of 0.5 for remote areas was not thought to be plausible we applied the hospital rate ratio for remote areas to the excess risk from the national drug survey results.

Schizophrenia

In the absence of survey data for schizophrenia using ICD-10 or DSM-IV-defined criteria, we used adjusted relativities from Australian hospital. For schizophrenia, in keeping with findings for New Zealand Maoris, we halved the excess risk suggested by hospital data by remoteness (Wheeler et al. 2005). We also assumed that the rate ratio for females was the same as males (as a higher risk did not appear plausible).

Anxiety & depression

We used indicators of psychological distress from the Western Australian Aboriginal Child Health Survey (Goodman's Strengths and Difficulties Questionnaire) and the 1997–98 New South Wales Health Surveys (Kessler 10 questionnaire) as proxies for anxiety & depression. To estimate the prevalence of anxiety & depression in Indigenous Australians, we applied derived rate ratios of psychological distress from published data, between the Indigenous and non-Indigenous population, to the national model (NSW Health Department Public Health Division 2000, Zubrick et al. 2005). For 4–17 year-olds, the rate ratio of psychological distress in Indigenous children and young adults was 1.6 times that of the non-Indigenous population. For male and female Indigenous adults, the rate ratios of psychological distress were in the order of 1.9 and 1.5 respectively. We estimated the prevalence of anxiety & depression for Indigenous children residing in non-remote and remote areas using published data (odds ratio=0.7) from the Western Australian Aboriginal Child Health Survey, which showed that children residing in more remote areas fared better in terms of significant emotional difficulties. We estimated the prevalence of anxiety & depression for Indigenous adults residing in non-remote and remote areas using rate ratios derived from published data from the 'Social and Emotional Wellbeing module' in the 2004–05 National Health Survey (ABS 2006a). Averaging rate ratios based on published proportions of responses of 'all the time/most of the time' for selected items in this wellbeing module indicated that Indigenous adults residing in non-remote areas were at slightly higher risk (RR 1.3) of psychological distress than Indigenous adults residing in remote areas.

Autism and Asperger's syndrome

We estimated the incidence of autism and Asperger's syndrome by applying the published odds ratio of autism in Western Australian Indigenous versus non-Indigenous infants (OR=0.3) to the national model incidence estimates for autism and Asperger's syndrome respectively (Leonard et al. 2005). This odds ratio is based on linking Western Australian data on people with intellectual disability from the Maternal and Child Health Research Database with other sources, including the Disability Services Commission, the Department of Education, Catholic Education and the Association of Independent Schools. In the absence of more detailed information, we assumed no differentials by remoteness. We assumed same remission, case-fatality and disability weights as the national model (Begg et al. 2007).

2K Nervous system and sense organ disorders

Epilepsy

A study comparing epilepsy presentations for Indigenous and non-Indigenous people with a far-north Queensland specialist clinic and a local hospital concluded that the similar incidence of epilepsy in Indigenous and non-Indigenous people presenting to the clinic indicated similar incidence in the community (Archer & Bunby 2006). Furthermore, the study argued that the much higher hospital admission rates for epilepsy in Indigenous people reflected inequalities in health care use (including epilepsy management practices, issues relating to access in remote areas, and socioeconomic differentials) rather than differentials in disease occurrence. Therefore we assumed that the incidence of primary epilepsy in the Indigenous Australian population was the same as the total Australian population and that there were no differentials by remoteness. We assumed the same remission and case fatality as the national model (Begg et al. 2007). We accounted for the greater disability associated with primary epilepsy in the Indigenous population by estimating a new disability weight, which assumed that a greater proportion of cases are untreated. The GBD assumes 80% treatment coverage in industrialised countries. Instead, we assumed 50% of cases are treated; and to be consistent with the National Study, we applied the ratio of the disability weights for 50% versus 80% treatment coverage to the Dutch disability weight for epilepsy (0.110). This gave a new disability weight of 0.144.

We used the estimates of intellectual disability by severity and underlying cause to model the epilepsy-related intellectual disability (see Section 2K).

Glaucoma and cataract-related blindness

There has been no systematic assessment of the prevalence of glaucoma or cataract among Indigenous Australians since the National Trachoma Program, which was conducted between 1976 and 1978 in remote Australia (Royal Australian College of Ophthalmologists 1980).

Primary open angle glaucoma is the most common type of glaucoma. The 1980 National Trachoma Program showed that 'Aborigines when compared to non-Aborigines appear to be remarkably spared from primary glaucoma' (Royal Australian College of Ophthalmologists 1980:97). Expert advice confirms that primary open-angle glaucoma is not seen in Indigenous Australians who do not have non-Indigenous ancestors. We estimated the incidence of glaucoma-related visual impairment for Indigenous Australians residing in non-remote and

remote areas by applying hospital rate ratios for glaucoma surgery by sex and remoteness (non-remote: males RR 0.8, females RR 0.9; remote: males RR 0.4, females RR 0.4) to the national model (Begg et al. 2007). We assumed duration was equivalent to the expected life expectancy by sex, age and remoteness. We assumed the same case fatality as the national model.

Expert advice indicated that Indigenous Australians residing in non-remote areas have the same prevalence of cataract as the total Australian population . This advice is consistent with the rate ratio (RR 1.0) of cataract surgeries in Indigenous Australians in non-remote areas and the total Australian population. We assumed that all mild and moderate bilateral vision loss due to cataracts is operated on, based on trends in the total Australian population. We estimated the incidence of mild and moderate cataract-related vision impairment in Indigenous Australians by remoteness using Australian hospital data (AIHW 2003b), and based on the following assumptions:

- 50% of Indigenous cases that were corrected surgically had vision loss in both eyes before the operation for an average of one year
- of these, 70% of cases were mild and 30% were moderate (compared with 90% and 10% split in the Australian model).

In addition to increasing the proportion of cases that were moderate, we also doubled the national model (Begg et al. 2007) remission to account for Indigenous Australians being accepted for cataract surgery at a much more advanced stage of disease (Taylor 1997). We estimated the incidence of severe bilateral vision loss due to cataracts by applying the indirect age-standardised rate ratio of hospital mentions of cataracts by sex and remoteness. We assumed the duration of severe bilateral vision loss due to cataracts was the same as the life expectancy for an Indigenous Australian at that age. We also assumed the same case fatality for all three stages of cataract as the national model (Begg et al. 2007).

Intellectual disability

Similar to the national model (Begg et al. 2007), we categorised intellectual disability into the following levels: mild, moderate, severe and profound. Intelligence quotient ranges were 50–69, 35–49, 20–34, and <20 respectively. To estimate the incidence of intellectual disability in Indigenous Australians, we applied published prevalence ratios (RR 2.4 for mild/moderate and RR 1.6 for severe) from a linked study of several sources[3] of intellectual disability information in Western Australia for 1983–92 to the total Australian population incidence by severity (Leonard et al. 2003). We assumed that the rate ratio of mild/moderate intellectual disability applies to both mild and moderate categories. Likewise, we assumed that the rate ratio of severe intellectual disability holds for Indigenous Australians with profound intellectual disability too. We estimated the duration of intellectual disability by severity for Indigenous Australians by applying total Australian population rate ratios (life expectancy at birth by level of severity of intellectual disability for the total population compared with the total Australian population life expectancy at birth) to Indigenous life expectancy at birth.

[3] The data sources included the Western Australia Disability Services Commission, the Western Australian Department of Education, Western Australian Catholic Education and Western Australian Association of Independent Schools. Record linkage with the Maternal and Child Health Research Database was used to obtain basic demographic information not available from the other sources and to form the denominator of the prevalence ratio.

In the absence of more detailed information, we assumed that the occurrence of intellectual disability does not differ by remoteness. As per the national model (Begg et al. 2007), we estimated the incidence of intellectual disability in DisMod assuming that:

- 90% of intellectual disability starts in the first year of life and the remaining 10% starts in the 1–4-year age group
- there is no remission
- there is a relative risk of mortality that gives an average duration by severity level, as explained above.

We did not include the incidence of intellectual disability as a discrete category in the main listings of this burden study. Instead, we attributed incident cases of intellectual disability to underlying causes (other chromosomal disorders, Down syndrome, low birth weight, infection, epilepsy, other perinatal conditions, autism and foetal alcohol syndrome) using findings from 1953–2000 Western Australian data on intellectual disability by cause among Indigenous Australians (Glasson et al. 2005). Since some Indigenous people had more than one underlying cause recorded, we rescaled the number of causes to reflect the number of cases. We also redistributed cases with unspecified underlying causes proportionately. This gave us the underlying cause distribution of intellectual disability for Indigenous Australians. However, since we had no information on the distribution of underlying causes by severity, we assumed that the proportionate distribution of cases by underlying cause was the same by severity.

2L Cardiovascular disease

Heart diseases resulting in heart failure

The complete descriptive epidemiology in this group of conditions was derived in DisMod from prevalence and case fatality; the third parameter being zero remission. Unlike the national model (Begg et al. 2007), we assumed no favourable trend in case fatality or incidence over time.

We estimated the prevalence of heart failure due to all causes for Indigenous Australians residing in remote and non-remote areas by applying the age-specific (to age 65+) rate ratios of heart failure, from Australian hospital data (AIHW 2003b), to the total Australian population prevalence. We increased the total Australian population case fatality rate for heart failure by 50% based on a report by the AIHW (Mathur et al. 2006). This report found that Indigenous case fatality for major coronary events was 50% higher than non-Indigenous. We assumed that all cardiovascular mortality would be similarly increased.

Underlying cause of heart failure was derived for Indigenous Australians residing in remote and non-remote areas by broad age group and sex from Australian hospital data where both heart failure and cardiovascular disease (rheumatic, hypertensive, ischaemic, pulmonary, inflammatory, and non-rheumatic valvular heart diseases) were mentioned in the same record. Remission for rheumatic heart disease and non-rheumatic valvular disease was estimated as the proportion of age-specific estimated prevalent cases that underwent surgery for valve replacement based on two years of adjusted Australian hospital data divided by two (to estimate the number of replacements in one year).

Ischaemic heart disease

Three health states were modelled separately for ischaemic heart disease: angina pectoris, acute myocardial infarction and heart failure. We estimated the incidence of angina pectoris and acute myocardial infarction by applying the age-specific (to age 65+) total Australian to remote and non-remote rate ratios of hospitalisation. We estimated remission from angina pectoris as the proportion of prevalent cases that underwent revascularisation surgery based on two years of adjusted Australian hospital data (AIHW 2003b). We increased the total Australian population case fatality for angina pectoris by a factor of 1.5 to reflect the 50% higher fatality seen for major cardio-vascular events (Mathur et al. 2006).

Stroke

Judy Katzenellenbogen, a PhD candidate in Perth, provided 28-day survivor stroke models, derived from a linked Western Australian database of hospital and mortality data and from the Perth community stroke data, for Indigenous Australians residing in remote and non-remote areas of Western Australia (Judy Katzenellenbogen, email, 22 September 2006). We estimated the incidence for all Indigenous Australians who reside in remote and non-remote areas by adjusting the Western Australia incidence figures with the age-standardised Western Australia to Australian hospital separation rate ratio for stroke.

Peripheral vascular diseases

We applied the rate ratio of total Australian to Indigenous Australian remote and non-remote peripheral vascular disease hospital separations to the national model incidence (Begg et al. 2007). We used this data to estimate the incidence of peripheral vascular disease among Indigenous Australians residing in remote and non-remote areas. We increased the total Australian population case fatality by 50%, reflecting the higher fatality in Indigenous Australians seen for major cardiovascular events (which we assumed applied to all cardiovascular disease case fatality) (Mathur et al. 2006). Remission for cases was determined as the proportion of prevalent cases (estimated in a first iteration in DisMod with input of incidence, case-fatality rate, and zero remission) that underwent peripheral vascular disease related bypass surgery. We assumed that the hospitalisation rate for peripheral vascular disease-related toe amputations and foot or leg amputations reflected true incidence.

2M Chronic respiratory disease

Chronic obstructive pulmonary disease

We estimated the prevalence of COPD for Indigenous Australians residing in non-remote and remote areas by applying age-specific rate ratios (for COPD) from Australian hospital data (AIHW 2003b) to the total Australian population prevalence. We assumed zero remission and set the case fatality rate to be 30% higher than the national model (Begg et al. 2007). YLD estimates for heart failure due to chronic lung disease and pulmonary heart disease were added to the COPD category (see Heart diseases resulting in heart failure, above).

Asthma

We estimated the prevalence of asthma for Indigenous Australians residing in non-remote and remote areas by applying relative risks of self-reported asthma from the 2004–05 National Health Survey (Source: ABS 2006. ABS data available on request) to the total Australian population prevalence. We assumed no difference in propensity to report asthma between Indigenous Australians and the total Australian population. We assumed remission was the same as in the total Australian population, and entered age- and sex-specific mortality due to asthma into DisMod.

The total Australian to Indigenous Australian rate ratios for asthma-related hospital separations were higher than those suggested in survey and epidemiological study data (Blair et al. 2005, Valery et al. 2001). We assumed that the higher hospitalisation rate for Indigenous Australians reflected, in part, sub-optimal therapy or prevention (Correll et al. 2007, Couzos & Davis 2005). Given this assumption, we derived a higher disability weight for Indigenous Australians. The national model derived a disability weight based on the symptomatic and asymptomatic Dutch disability weight (Begg et al. 2007). The GBD assumes a 95% treatment coverage for established market economies. We adjusted this downwards to 80% treatment coverage for Indigenous Australians. We then adjusted the national model disability weight using the ratio of the GBD weights for 80% and 95% treatment coverage.

2N Diseases of the digestive system

Cirrhosis of the liver

We estimated the incidence of cirrhosis in the Indigenous population by applying hospital rate ratios by remoteness to the national model (Begg et al. 2007). We distributed the incidence of liver cirrhosis by underlying cause for Indigenous Australians using Australian hospital data (AIHW 2003b), which show that alcohol is mentioned in 62% of all admissions for cirrhosis with an underlying cause stated as secondary diagnostic category. Similar to the national model (Begg et al. 2007), we attributed 5% of non-alcohol related cirrhosis to 'other' causes and the remainder to hepatitis (hepatitis b=30% and hepatitis C=70%). This translated to 10% and 23% of all cirrhosis for hepatitis B and C, respectively. In the absence of more detailed information, we assumed the same proportions for the liver cancer model.

2O Genitourinary diseases

Nephritis & nephrosis

For nephritis & nephrosis, we calculated three complications: end-stage renal failure with dialysis, kidney transplants, and untreated end-stage renal failure.

The Australia and New Zealand Dialysis and Transplant Registry collects information from all hospitals treating end-stage renal disease patients in Australia and New Zealand for all people under their care. Ethnicity is self-reported and is not further verified. Information is collected about cause of end-stage renal disease, type of renal replacement therapy, and dose and location, together with placement on the active transplant waiting list (McDonald & Russ 2003). We excluded diabetic cases from our calculations, because they were included in other models.

For end-stage renal failure with dialysis and transplant cases, we estimated the total number of incident cases as the mean number of new Indigenous patients over the years 2001–03 (excluding patients with renal disease related to diabetes) (Excell & McDonald 2005). We distributed incident end-stage renal disease dialysis cases according to the average 2001–03 age distribution for all new cases. Based on a study by McDonald and Russ (McDonald & Russ 2003), we assumed that 43% of cases at each age were male. Transplant cases were distributed by age according to 2001 data (McDonald & Russ 2002) and by sex according to the sex distribution of recipients with a functioning transplant at 31 Dec 2003 (Excell & McDonald 2005). We separated incident end-stage renal disease with dialysis and transplant cases into remote and non-remote by applying the remote to total Indigenous Australian, and non-remote to total Indigenous Australian mortality relative risks. We used the mortality data, because the hospital data may potentially reflect access issues for people residing in remote areas. We estimated incidence of untreated end-stage renal failure as the number of deaths due to nephritis & nephrosis in the mortality data minus the number of deaths estimated after dialysis and/or transplant from DisMod. Any negatives were set to zero.

We estimated remission for end-stage renal disease with dialysis as the proportion of cases that go on to receive a transplant. Remission for cases of kidney transplants (i.e. graft failure) from the national model (Begg et al. 2007) was increased by a factor of 3.1 based on the hazard ratio for graft survival among Indigenous transplant recipients (McDonald 2004). We assumed a duration of one year for untreated end-stage renal disease.

Indigenous-specific case fatality rates for end-stage renal disease with dialysis and kidney transplant are from McDonald (McDonald 2004). To adjust for the differences in diabetics and non-diabetics, we used adjusted case-fatality rates all Australian Australia and New Zealand Dialysis and Transplant Registry mortality data obtained by an all-age rate ratio of diabetics to non-diabetics.

We assumed that graft failure cases become dialysis cases again, and therefore added the remitters from the kidney transplant DisMod model to the end-stage renal disease with dialysis incidence.

2R Congenital anomalies

We used the estimates of intellectual disability by severity, remission and case fatality to model the intellectual disability related to Down syndrome and other chromosomal disorders (see Section 2K).

2S Oral health

Dental caries

We estimated the incidence of caries in Indigenous children using relative risks derived from published data on caries experience between Aboriginal and non-Aboriginal children (6-year-old RR 2.4; 12-year-old RR 1.8) from the 2001 South Australian Child Dental Health Survey (AIHW DSRU 2003). Higher incidence of caries in Indigenous children has also been reported in several other studies (AIHW DSRU 2002, Davies et al. 1997, Popat & Dinnage 2006). It is important to note that the South Australian data are based on children who use the School Dental Service and therefore may not be representative of all Indigenous children.

Using information from this same report, we assumed that there are negligible differentials by remoteness for Indigenous children's caries experience (5–9 years RR 0.9; 10–14 years RR 1.1) (AIHW DSRU 2003). We assumed that the 1–4-year-old Indigenous children have the same decay experience as 5–9–year-old Indigenous children.

Consistent with our findings for children, Indigenous adults also experienced higher rates of caries compared with their non-Indigenous counterparts. We estimated the incidence of caries in Indigenous adults using relative risks derived from published data on untreated dental decay between Indigenous and non-Indigenous adults (15–34 years RR 2.7; 35–54 years RR 2.2; 55+ RR 2.0) from the 2004–06 National Survey of Adult Oral Health (Roberts-Thomson & Do 2007). Using published South Australian data, we assumed that Indigenous adults residing in remote areas had half the risk of dental caries compared with Indigenous adults residing in non-remote areas (AIHW DSRU 2003). We assumed the same remission and case fatality as the total Australian population. We adjusted the national model (Begg et al. 2007) estimate of time symptomatic using a relative risk (RR 1.3) based on published data of the proportion of people by Indigenous status seeking dental help for a problem from the 1994–96 National Dental Telephone Survey (Brennan & Carter 1998).

Periodontal disease

We assumed that the incidence of periodontal disease in the Indigenous population was the same as the national model (Begg et al. 2007). This is because of the lack of converging evidence of the occurrence of periodontal disease in Indigenous Australians. The 2004–06 National Oral Health Survey found no differences in the prevalence of periodontitis. However, the authors state that the lack of differences 'may be due to under-representation and or limited statistical power to detect differences' (since only 75 Indigenous people were examined for periodontal disease) (Roberts-Thomson & Do 2007). A review of hospital data suggests differentials in the order of two times the total Australian population, which increase by remoteness. This increase of periodontal disease is consistent with the relationship between Type 2 diabetes and periodontal disease (Matthews 2002) and the increased risk of Type 2 diabetes among Indigenous people who reside in remote areas. However, hospital data is more likely to reflect symptomatic oral disease rather than asymptomatic oral health. According to expert advice, periodontal disease is asymptomatic the majority of the time. The 1995–96 Adult Dental Survey also showed a two-fold risk of periodontal disease, as indicated by pockets of 6+ mm, for Indigenous Australians (Brennan & Carter 1998). However, the Adult Dental Survey studied patients attending public dental care and therefore may not be representative of all Indigenous Australians. We assumed the same remission, case fatality and time symptomatic as the national model (Begg et al. 2007).

Edentulism

Based on findings from the 2004–06 National Oral Health Survey, we assumed that the incidence of edentulism in the Indigenous population is the same as the total Australian population. Despite reporting that Indigenous people have 'disproportionately elevated rates of tooth loss' (Slade et al. 2007:xvii), the confidence intervals for Indigenous and non-Indigenous prevalence estimates from the national oral health survey mostly overlap (overall RR 1.2) (Roberts-Thomson & Do 2007). In contrast, the 1994–96 National Dental Telephone Survey found that Indigenous people were more likely to be edentulous than non-Indigenous people (RR 1.5), whereas the 1995–96 Adults' Dental Survey found that edentulism was lower among Indigenous than non-Indigenous patients (RR 0.4). Both of

these latter datasets had high associated standard errors (Brennan & Carter 1998). We assumed the same remission and case fatality as the national model (Begg et al. 2007). In the absence of more detailed information, we assumed there were no differentials by remoteness.

Appendix D Methods for attributing risk

This study includes eleven risk factors for disease and injury (tobacco, alcohol, illicit drugs, high body mass index, inadequate physical activity, low intake of fruit and vegetables, high blood pressure, high cholesterol, unsafe sex, child sexual abuse, and intimate partner violence). We excluded three risk factors that were included in the National Study (Begg et al. 2007) (occupation, osteoporosis and air pollution) because no appropriate data existed upon which to estimate the prevalence of exposure and associated risk. Similarly, we did not include other risk factors from the *Comparative quantification of health risks, global and regional burden of disease attributable to selected major risk factors* (Ezzati et al. 2004) such as poverty (because of lack of available data on economic status and health outcomes in individuals), even though it may have considerable impact on the health of Indigenous Australians.

We followed the methods used in the National Study (Begg et al. 2007) and used the same relative risks of disease due to risk factor exposure and direct population-attributable fractions unless otherwise specified. In this section, we describe the methods used to derive exposure estimates, and relative risks or direct population-attributable fractions where relevant. A summary of estimated exposure prevalence is provided in Table D.1, and an assessment of the exposure estimates is in Annex Table 3.

D.1 High blood pressure

High systolic blood pressure is associated with an increased risk of cardiovascular disease, including ischaemic heart disease and stroke. By definition, we attributed all hypertensive heart disease to high blood pressure.

Mean and standard deviation systolic blood pressure for Indigenous Australians residing in non-remote areas was estimated using age group- and sex-specific data from the DRUID Study (diabetes and related conditions in urban Indigenous people in the Darwin region) supplied by the data custodian. This study used non-random techniques to recruit volunteer participants, and was based in one urban area of Australia. This may limit the study's generalisability to all Indigenous Australians residing in non-remote areas; however, it is one of the few studies based in a non-remote area that measured systolic blood pressure and was available for our use. A published description of the cohort and methods used in the DRUID Study has been published elsewhere (Cunningham et al. 2006).

For Indigenous Australians residing in remote areas, estimates of mean and standard deviation systolic blood pressure were based on a study by Wang and Hoy (2003). Data were collected during 1992–98, as part of a community screening program in a remote Northern Territory Aboriginal community (Tiwi Islands). Mean and standard deviation systolic blood pressure were reported by sex and 10-year age groups from 25 years onwards. Similar to the study on which the non-remote estimates are based, this study was conducted in a single remote Indigenous Australian community. The estimates of mean blood pressure may therefore not be representative of the entire Indigenous Australian population that live in remote areas.

The mean blood pressure and standard deviations for both the DRUID and Wang and Hoy studies were presented for age groups that do not match the available relative risk. Therefore, we estimated the mean and standard deviation blood pressure for the required

age groups by fitting a linear equation to the supplied means, and a power equation to the standard deviations.

D.2 High blood cholesterol

High total cholesterol is associated with ischaemic heart disease and stroke. Mean and standard deviation total cholesterol estimates are based on the same studies used to estimate mean blood pressure. For Indigenous Australians residing in non-remote areas, we estimated the mean and SD cholesterol for the required age groups by fitting polynomial equations to the supplied DRUID data. For Indigenous Australians residing in remote areas, we estimated the mean cholesterol for males by fitting a linear equation, and for females by fitting a power equation to the Wang and Hoy data (2003). To estimate SD for both male and female Indigenous Australians residing in remote areas we fit power equations.

D.3 High body mass index

High body mass index (BMI) is associated with an increased risk of cardiovascular disease, Type 2 diabetes, osteoarthritis, and cancers of the breast, bowel, and uterus.

We estimated mean and standard deviation of BMI from NATSIHS 2004–05 (Source: ABS 2006. ABS data available on request). The NATSIHS 2004–05 collected self-reported height and weight information for Indigenous Australians residing in non-remote areas, while in remote areas participants were offered to have their height and weight measured (ABS 2006a). The ABS provided tables of mean self-reported and measured BMI and SDs by age and sex for Indigenous Australians residing in remote areas, and also mean self-reported BMI and SD by age group for Indigenous Australians residing in non-remote areas. We estimated the sex-specific self-reported mean BMI and SD for Indigenous Australians residing in non-remote areas by assuming the same sex ratios as those residing in remote areas. Tables provided by the ABS indicated that for Indigenous Australians residing in remote areas, mean BMI based on self-report data was higher than that based on measured data (i.e. remote residing Indigenous Australians tend to overestimate weight, and/or underestimate height). We assumed that these sex- and age-specific self-reported to measured ratios are the same for Indigenous Australians in non-remote areas and adjusted the non-remote self-reported estimates accordingly. To interpolate from the ABS data to the required age groups, we fitted polynomial equations to the sex- and geographic-specific BMI estimates.

Since BMI is not normally distributed, we assumed that it follows a log-normal distribution and derived parameters of a log-normal distribution that has the same mean as generated above.

D.4 Insufficient intake of fruit and vegetables

Insufficient consumption of fruit and vegetables is related to an increased risk of ischaemic heart disease, stroke, and cancers of the lung, stomach, bowel, and oesophagus.

The National Study (Begg et al. 2007) assumed a normal distribution of grams of fruit and vegetable intake, and therefore used mean and standard deviation of fruit and vegetable intake. In this study, however, we used grams of fruit and vegetable intake as a categorical variable, while the theoretical minimum was still a distribution with a mean of 600 g and

standard deviation of 50 g. Categories of daily fruit and vegetable consumption for Indigenous Australians residing in non-remote areas were derived from the NATSIHS 2004–05 survey (ABS 2006b). In the NATSISH 2004–05 survey, one serve of vegetables was defined as 75 g, while a serve of fruit was defined as 150 g fresh or 50 g dried fruit (ABS 2006a). Given that there was no breakdown of fruit into fresh or dried, and that the inherent assumption from the ABS definition is that 50 g dried fruit is of equivalent value to 150 g fresh fruit, we applied the conversion of 150 g to each serve of fruit (whether fresh or dried). The questions relating to fruit and vegetable consumption in the remote component of the survey did not allow for direct estimation of the number of grams consumed. Therefore, we estimated the proportion of Indigenous people in remote areas who do not usually consume fruit or vegetables, and distributed those that usually eat fruit and/or vegetables each day as per the estimates for Indigenous Australians residing in non-remote areas.

D.5 Physical inactivity

Insufficient level of physical activity was associated with an increased risk of ischaemic heart disease, stroke, Type 2 diabetes, breast cancer, and bowel cancer.

Physical activity data were derived for Indigenous Australians in non-remote areas from the NATSIHS 2004–05 survey (Source: ABS 2006. ABS data available on request). The questions regarding physical activity in the remote component of the survey did not allow for the classification of individuals into the required categories. We therefore estimated the proportion of Indigenous Australians residing in remote areas who are classified as inactive (i.e. did not walk or do moderate or vigorous activity) from the items relating to number of times walked, or participated in moderate or vigorous activity, and distributed the remaining 'active' population to the other three categories as per the non-remote distribution. The exercise-related questions in this survey related to physical exercise undertaken for recreation, sport or health/fitness purposes, conceptually excluding physical activity undertaken as a part of work or for other purposes. This may result in an underestimate of the amount of physical activity undertaken, and therefore overestimate the burden of disease attributable to physical inactivity.

Since the relative risks for conditions due to exercise level apply to different age groupings from those supplied in the tables by the ABS, we estimated the prevalence for relevant age groups by fitting a polynomial equation.

D.6 Tobacco smoking

Tobacco smoking is associated with cardiovascular disease, a range of cancers, chronic respiratory disease, fire injuries, inflammatory bowel disease, age-related vision disorders, low birth weight, sudden infant death syndrome, and otitis media. Tobacco smoking also has a small protective effect on Parkinson's disease.

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. We therefore 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' was 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. Due to the small number

of lung cancer deaths, we calculated a smoking impact ratio for Indigenous Australians by age group, with no sex- or remoteness-specific estimates, with the reference population being adult males from the CPS-II. Compared with cancers and COPD, the mean time between exposure to tobacco and all other adverse health outcomes was considerably shorter. Therefore, for these conditions, we used the prevalence of daily smoking among adults from the NATSIHS 2004–05 survey (ABS 2006b).

We base adult passive smoking estimates on those used by Ridolfo & Stevenson (2001), and therefore assume the proportion of people who are non-smokers and are exposed to spousal tobacco smoke is the same in the Indigenous Australian and total Australian population. Comparisons of proportions of those that have never smoked but live with a smoker in the 1994 NDSHS Urban Indigenous Supplement (Department of Human Services and Health 1995) are very similar to those estimates used for the total population in Ridolfo & Stevenson and therefore has negligible impact on the tobacco smoking results.

Prevalence of smoking during pregnancy (52%) was taken from the *Australia's Mothers and Babies 2003* report (Laws & Sullivan 2005). Data from the Western Australian Aboriginal Child Health Survey suggest a relationship between remoteness and prevalence of smoking during pregnancy, with a non-remote to remote RR of 1.33 (Zubrick et al. 2004). For 2003, we estimated that 32% of Indigenous women aged 15–49 years lived in remote areas of the five states and territories that provided data to the NPSU for *Australia's Mothers and Babies 2003* (Laws & Sullivan 2005). This distribution and the RR from the Western Australian study were used to estimate the prevalence of smoking during pregnancy for Indigenous Australians residing in remote and non-remote areas. Based on the Bibbulung Gnarneep Solid Kid Study (Eades et al. 1999), we assumed that the same proportion of Indigenous Australian women continue to smoke post-partum, and applied the prevalence of smoking during pregnancy to infants exposed to maternal smoking.

D.7 Alcohol

Alcohol abuse and harmful use is associated with stroke, hypertensive heart disease, cancers of the mouth, oesophagus, liver, larynx, and breast, pancreatitis, inflammatory heart disease, and injuries. Alcohol also has some beneficial effect on ischaemic heart disease and cholelithiasis.

To estimate prevalence of alcohol consumption, we used self-report data from the ABS NATSIHS 2004–05 survey (ABS 2006b). These data suggest that a higher proportion of Indigenous Australians in non-remote areas fall into the harmful/hazardous category than remote residing Indigenous Australians (remote 9.0% males, 8.6% females; non-remote 10.7% males, 8.8% females). This relationship is in the opposite direction of that suggested by hospital and mortality data for alcohol dependence, where the rate of hospitalisation and mortality for this cause is higher in remote compared with non-remote areas (NHMD RR 1.5 males, 2.1 females; mortality RR 1.9 males, 3.3 females).

While we recognised that alcohol consumption occurs at ages younger than 18 years, we did not estimate the risk of disease or injury (excluding RTAs and fire/burns/scalds) from alcohol for this age group due to lack of exposure data comparable to that used for adults. Prevalence of alcohol consumption among adults was categorised into the four levels used in English et al.'s analysis of the risks of alcohol consumption (1995). The prevalence of each level of alcohol intake was estimated by age group and sex, from the average weekly

consumption of alcohol. The data was combined to give each day of the week of interview equal weighting.

Based on data from English et al. (1995), the National Study (Begg et al. 2007) attributed 24% of acute pancreatitis to alcohol use. According to a study on admissions to Alice Springs Hospital, 70% of Indigenous and 43% of non-Indigenous admissions for pancreatitis were associated with alcohol (Ah-Tye 2001). We applied this Indigenous to non-Indigenous relativity to the PAF used in the National Study and estimated that 38% of acute pancreatitis in Indigenous Australians was attributable to alcohol.

D.8 Illicit drugs

We estimated the proportion of HIV/AIDS, hepatitis B and C, antepartum haemorrhage, low birth weight, schizophrenia, inflammatory heart disease, suicide, and RTA injuries due to use of illicit drugs.

We used 2003 data from the HIV/AIDS, viral hepatitis and sexually transmissible infections in the *HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report 2005* (National Centre in HIV Epidemiology and Clinical Research 2005) to estimate the proportion of HIV and AIDS cases among Indigenous Australians that are attributable to illicit drug use. We assumed that all cases with exposure category 'male homosexual contact and injecting drug use' were attributable to male homosexual contact. Given a lack of information regarding HIV exposure categories for Indigenous Australian deaths following AIDS, we assumed that the exposure category for death following AIDS was the same as for all AIDS cases over the period 1995–2004.

We had no information regarding hepatitis B and C in Indigenous Australians that is attributable to illicit drugs or sexual transmission. We therefore applied the same proportions as the total Australian population estimates.

Antepartum haemorrhage is a potential outcome of heroin or cocaine use during pregnancy. We estimated the Indigenous Australian to total Australian rate ratio of heroin or cocaine use in past 12 months for females aged 15–49 years from the NATSIHS 2004–05 survey (Source: ABS 2006. ABS data available on request), and the NDSHS 2004 (AIHW & Australian Government Department of Health and Ageing 2005). We applied this rate ratio to the age-specific prevalence estimates for the total Australian population. Given a lack of information, we assumed the same prevalence for Indigenous Australians residing in remote and non-remote areas. For low birth weight, we used prevalence of cannabis and opioid diagnosis during pregnancy in New South Wales and relative risks from Burns et al. (2006).

To estimate the proportion of schizophrenia attributable to cannabis use, we estimated the prevalence of daily cannabis use for Indigenous Australians. According the Chikritzhs and Brady (Chikritzhs & Brady 2006), the *National Drug Strategy Household Survey: Urban Aboriginal and Torres Strait Islander Supplement, 1994* is the most reliable Indigenous substance-use survey published so far, although it is now quite dated. A comparison of prevalence from the 1995 and 2004 NDSHS data indicates that the self-reported prevalence of daily cannabis use in all Australians has risen (AIHW & Australian Government Department of Health and Ageing 2005, Vuksa & Kelly 1996). We assumed a similar increase in prevalence among Indigenous Australians residing in remote and non-remote areas.

The method used to derive Indigenous Australian prevalence of daily cannabis use assumes that the non-remote Indigenous-to-total-Australian differential that existed in 1994–95 was the same as in 2004. We applied the age-standardised ratio of urban Indigenous prevalence

(Department of Human Services and Health 1995) to total Australian prevalence (Vuksa & Kelly 1996), to the total Australian prevalence in 2004 (AIHW & Australian Government Department of Health and Ageing 2005). This gave us a recent estimate of daily cannabis use for Indigenous Australians residing in non-remote areas. In the absence of other information, we resorted to using the rate ratio of hospitalisation for cannabis dependence & harmful use between non-remote and remote residing Indigenous Australians to derive the estimated prevalence of daily cannabis use for remote residing Indigenous Australians.

D.9 Unsafe sex

For HIV, AIDS, AIDS deaths and hepatitis, we calculated the proportion attributable to unsafe sex in the same way as for illicit drug use (described above). We assumed that all sexually transmitted disease, abortion and cervical cancer resulted from unsafe sex.

D.10 Childhood sexual abuse

Sexual abuse in childhood is associated with anxiety & depression, alcohol and illicit drug dependence & harmful use and associated risks, and suicide as an adult.

Studies have suggested that child protection statistics underestimate the actual occurrence of child sexual abuse more so in the Indigenous community than the non-Indigenous community (Stanley et al. 2003). Despite this, child protection statistics are still believed to be the most reliable source of data on child abuse and neglect (Richardson 2005), and therefore probably the best source of information regarding the relative differences in the prevalence of child sexual abuse in Indigenous and non-Indigenous Australians.

We estimated the rate ratio of Indigenous child sexual abuse substantiations to total Australian child sexual abuse substantiations from child protection data for 2002–03 (AIHW 2004a). For non-contact child sexual abuse, we assume the same prevalence among Indigenous Australians as the national model (Begg et al. 2007); for contact-only child sexual abuse, we applied half the rate ratio; and for intercourse child sexual abuse, we applied the whole rate ratio. This gradient of risk was based on the assumption that child sexual abuse substantiations reflect more severe abuse, and if this rate ratio were applied to all categories, the overall prevalence of child sexual abuse would be implausibly high. Due to the lack of empirical data on the prevalence of child sexual abuse in remote versus non-remote communities, we assumed the same prevalence applies to both areas.

Our method assumed that the observed rate ratio of child sexual abuse between Indigenous and non-Indigenous Australian children in 2002–03 has been constant over time. This was necessary as we attributed disease outcomes in adulthood to child sexual abuse in the past.

D.11 Intimate partner violence

Intimate partner violence is associated with anxiety & depression, eating disorders, tobacco, alcohol and other drug use and associated risks, sexually transmitted disease, cervical cancer, fall injuries, suicide, and homicide & violence.

We estimated the prevalence of Indigenous women that had experienced intimate partner violence by applying the Indigenous-to-non-Indigenous ratio of the proportion of women who reported experiencing any violence in the past 12 months, and over their lifetime from

the Australian component of the International Violence Against Women Survey (Mouzos & Makkai 2004) to the national model estimates of 12-month or lifetime prevalence (Begg et al. 2007). The application of these rate ratios assumed that the same proportion of any violence experienced by Indigenous and non-Indigenous women was perpetrated by the intimate partner. Australian hospital data (AIHW 2003b) on assaults suggest that a similar proportion of assaults in all Australian women, and Indigenous Australian women residing in non-remote and remote areas, are perpetrated by a spouse (including ex and defacto) (40%, 44%, and 40% respectively). However, the proportion of female hospital separations for assault that have no perpetrator specified is higher for Indigenous women (22%, 34%, and 44%). Also, in a study of homicide over the period 1989–98, 75.4% of Indigenous femicide victims were killed by their intimate partners, compared with 54% for non-Indigenous femicide victims (Mouzos 1999).

The International Violence Against Women Survey was a computer-assisted telephone interview survey, and only included 91 respondents who identified as being Indigenous (1.4% of the sample). Also, the survey response rate was just 39%. This may limit the generalisability of the survey findings to the entire Indigenous Australian female population. While the hospitalisation and Supported Accommodation Assistance Program Indigenous-to-non-Indigenous ratios are much higher than those found in the International Violence Against Women Survey, we assumed that the institution-based data represented the severe end of the spectrum, and since Indigenous women experience more severe injury as a result of violence (Ferrante et al. cited in Blagg 2000), use of service data could lead to overestimation of total intimate partner violence experienced.

Remote-to-non-remote ratios based on the Supported Accommodation Assistance Program data for closed support periods were used to adjust the total Indigenous prevalence of intimate partner violence. We attempted to adjust support periods to represent people by dividing the non-Indigenous and Indigenous periods by the mean number of support periods per client (1.5 and 1.8 respectively) (AIHW 2005c). While the mean number of support periods per client may differ between Indigenous women residing in remote and non-remote areas, we have no information regarding this.

The population-attributable fraction for homicide & violence due to intimate partner was derived from a study that found that over the period 1989–98, 75.4% of Indigenous women who were victims of homicide were killed by an intimate partner (Mouzos 1999). We adjusted this figure for Indigenous Australian women residing in remote and non-remote areas by applying the remote/non-remote to total Indigenous ratios derived from the proportion of assault hospital separations, where the relationship of the victim of assault to the perpetrator was recorded as spouse or domestic partner.

Table D.1: Prevalence distributions of risk factors by age and sex

	Indigenous Australians in non-remote areas								Indigenous Australians in remote areas							
	Males				Females				Males				Females			
Risk factor	**0–14**	**15–34**	**35–54**	**55+**	**0–14**	**15–34**	**35–54**	**55+**	**0–14**	**15–34**	**35–54**	**55+**	**0–14**	**15–34**	**35–54**	**55+**
High blood pressure (mmHg)[(a)]																
Mean	. .	120	125	134	. .	111	119	137	. .	124	132	148	. .	115	123	140
SD	. .	14	17	23	. .	12	15	21	. .	16	18	20	. .	16	20	28
High cholesterol (mmol/L)[(a)]																
Mean	. .	5.2	5.4	5.3	. .	4.9	5.2	5.2	. .	5	5	5	. .	5	5	5
SD	. .	1.3	1.4	1.0	. .	1.0	1.0	0.9	. .	1	1	1	. .	1	1	1
BMI (kg/m^2)[(a)]																
Mean	. .	24.9	26.0	27.0	. .	26.2	27.9	28.0	. .	26	26	26	. .	27	28	27
SD	. .	5.6	5.6	5.2	. .	6.8	7.0	5.8	. .	6	6	5	. .	7	7	6
Fruit and vegetable intake (g/day)																
0	. .	1%	1%	1%	. .	0%	0%	0%	. .	16%	11%	13%	. .	9%	8%	6%
75	. .	4%	4%	2%	. .	4%	1%	1%	. .	3%	3%	2%	. .	4%	1%	1%
150	. .	2%	4%	4%	. .	2%	2%	1%	. .	2%	4%	4%	. .	2%	1%	1%
225	. .	17%	20%	15%	. .	17%	16%	7%	. .	14%	18%	13%	. .	15%	15%	6%
300	. .	14%	10%	11%	. .	11%	12%	5%	. .	12%	9%	10%	. .	10%	11%	5%
375	. .	22%	19%	11%	. .	21%	18%	18%	. .	19%	17%	10%	. .	19%	16%	17%
450	. .	11%	12%	7%	. .	12%	10%	14%	. .	9%	11%	6%	. .	11%	9%	13%
525	. .	8%	9%	12%	. .	10%	16%	17%	. .	7%	8%	10%	. .	9%	14%	16%
600	. .	8%	4%	11%	. .	7%	6%	8%	. .	6%	4%	10%	. .	7%	6%	7%
675	. .	4%	6%	13%	. .	5%	8%	9%	. .	4%	6%	11%	. .	5%	7%	8%
750	. .	8%	11%	13%	. .	10%	12%	20%	. .	7%	10%	12%	. .	9%	11%	19%
Physical activity (% population in categories)																
Sedentary	. .	35%	49%	59%	. .	47%	52%	59%	. .	28%	44%	67%	. .	37%	45%	66%
Low	. .	46%	42%	31%	. .	46%	44%	37%	. .	51%	45%	23%	. .	55%	51%	30%
Moderate	. .	16%	9%	9%	. .	6%	4%	4%	. .	18%	10%	9%	. .	7%	4%	4%
High	. .	3%	1%	0%	. .	1%	0%	0%	. .	3%	1%	0%	. .	1%	0%	0%

(continued)

Table D.1 (continued): Prevalence distributions of risk factors by age and sex

	Indigenous Australians in non-remote areas								Indigenous Australians in remote areas							
	Males				Females				Males				Females			
Risk factor	**0–14**	**15–34**	**35–54**	**55+**	**0–14**	**15–34**	**35–54**	**55+**	**0–14**	**15–34**	**35–54**	**55+**	**0–14**	**15–34**	**35–54**	**55+**
Tobacco (% population in categories)																
Current smoker	. .	39%	53%	32%	. .	41%	56%	26%	. .	54%	55%	42%	. .	44%	45%	24%
Prenatal exposure	56%	. .	. .	. .	56%	. .	. .	. .	42%	. .	. .	. .	42%	. .	. .	. .
Exposed to maternal smoking	56%	. .	. .	. .	56%	. .	. .	. .	42%	. .	. .	. .	42%	. .	. .	. .
Alcohol (% population in categories)																
Low	. .	35%	40%	30%	. .	19%	23%	15%	. .	24%	24%	18%	. .	10%	15%	12%
Hazardous	. .	8%	8%	8%	. .	8%	12%	6%	. .	7%	6%	5%	. .	6%	6%	7%
Harmful	. .	8%	15%	13%	. .	5%	4%	3%	. .	6%	14%	6%	. .	8%	5%	2%
Illicit drugs (% population in categories)																
Daily cannabis use	. .	9%	7%	0%	. .	11%	7%	0%	. .	5%	3%	0%	. .	3%	2%	0%
Prenatal exposure — opioids	2%	. .	. .	. .	2%	. .	. .	. .	2%	. .	. .	. .	2%	. .	. .	. .
Prenatal exposure — cannabis	3%	. .	. .	. .	3%	. .	. .	. .	3%	. .	. .	. .	3%	. .	. .	. .
Maternal use — heroin	. .	. .	. .	. .	. .	0%	0%	. .	. .	. .	. .	. .	. .	0%	0%	. .
Maternal use — cocaine	. .	. .	. .	. .	. .	1%	0%	. .	. .	. .	. .	. .	. .	1%	0%	. .
Child sexual abuse (% population in categories)																
Non-contact only CSA	1%	2%	2%	2%	4%	7%	9%	9%	1%	2%	2%	2%	4%	7%	9%	9%
Contact only CSA	2%	3%	4%	4%	12%	18%	23%	23%	2%	3%	4%	4%	11%	18%	23%	23%
Intercourse CSA	2%	3%	4%	4%	7%	11%	13%	13%	2%	3%	4%	4%	7%	11%	13%	13%
Intimate partner violence (% population in categories)																
Sexual or physical violence	. .	. .	. .	. .	. .	18%	27%	16%	. .	38%	55%	32%	. .	38%	55%	32%

(a) For high blood pressure, high cholesterol, and BMI mean and standard deviation in 15–34 column refers to persons aged 30–34 years
. . Not applicable

Annex tables

Annex table 1: Principal data sources for epidemiological modelling

Primary data source	Reference period	Disease and injury categories	
A. Disease registers, surveillance, notification and vital registration systems			
National Notifiable Diseases Surveillance System			
Age- and sex-specific rate ratios	2001–03	1A02b	Chlamydia (E)
		1A02c	Gonorrhoea (E)
		1A05b	Whooping cough
		1A05g	*Haemophilus influenzae* type b
		1A08c	Dengue
		1A08d	Other arbovirus infection
		1A09a	Hepatitis A
		1A10	Malaria
Age-standardised rate ratios	2001–03	1A08a	Ross River virus
		1A08b	Barmah Forest virus
		1A09b	Hepatitis B (E)
Annual report	2003	1A01	Tuberculosis (E)
		1A05e	Measles (E)
National HIV Database and National AIDS Registry	2003	1A03	HIV/AIDS (E)
Australia and New Zealand Dialysis and Transplant Registry	2001–03	2O01	Nephritis & nephrosis (E)
Australian mortality data			
Age-standardised mortality rate ratios	2003	2F	Malignant neoplasms[(a)]
	2003	2O01	Nephritis & nephrosis (E)
National Perinatal Data Collection	2003	1D02	Low birth weight (E)
B. Health service utilisation data			
Australian hospital data			
Age- and sex-specific rate ratios	2001–03	1A02a	Syphilis
		1A02d	Other STD
		1A04	Diarrhoeal diseases
		1A06	Meningitis
		1A07	Septicaemia (E)
		1C01	Maternal haemorrhage (E)
		1C02	Maternal sepsis (E)
		1C03	Hypertensive disorders of pregnancy (E)
		1C04	Obstructed labour (E)
		1C05	Abortion (E)

(continued)

Annex table 1 (continued): Principal data sources for epidemiological modelling

Primary data source	Reference period	Disease and injury categories	
		1Coth	Other maternal conditions (E)
		1E02	Deficiency anaemia
		2K08b	Cataract-related blindness (E, P)
		2L02	Ischaemic heart disease (E)
		2L06	Non-rheumatic valvular disease (E) [(c)]
		2L08	Peripheral vascular disease (E)
		2Lhfa	Heart failure[(c)]
		2M01	Chronic obstructive pulmonary disease (E)
		2N07	Pancreatitis
Age-standardised rate ratios		1A01	Tuberculosis (E)
		1B01	Lower respiratory tract infections
		1B02	Upper respiratory tract infections
		1B01	Lower respiratory tract infections
		1B02	Upper respiratory tract infections
		1D01	Birth trauma & asphyxia (E)
		1D02	Low birth weight (E)
		1D03	Neonatal infections (E)
		2G01	Uterine myomas
		2G02	Benign neoplasms of meninges and brain
		2H02	Type 2 diabetes (E)
		2J01d	Cannabis
		2J01e	Other drug dependence
		2J02	Schizophrenia
		2I01a	Haemolytic anaemia
		2I01b	Other non-deficiency anaemia
		2I02	Cystic fibrosis
		2I03	Haemophilia
		2J01b	Heroin or polydrug dependence
		2J04	Bipolar disorder
		2J06a	Anorexia nervosa
		2J06b	Bulimia nervosa
		2J06c	Other eating disorders
		2K04	Multiple sclerosis
		2K08a	Glaucoma-related blindness (E, P)
		2K08b	Cataract-related blindness (E)
		2K08e	Refractive errors

(continued)

Annex table 1 (continued): Principal data sources for epidemiological modelling

Primary data source	Reference period	Disease and injury categories	
		2L03	Stroke (E)
		2L05	Hypertensive heart disease (E)
		2L07	Aortic aneurysm
		2N01	Peptic ulcer disease
		2N02	Cirrhosis of the liver (E, U)
		2N03	Appendicitis
		2N04	Intestinal obstruction
		2N05	Diverticulitis
		2N06	Gallbladder and bile duct disease
		2N09	Vascular insufficiency bowel
		2O02	Benign prostatic hypertrophy
		2Ooth	Other genitourinary diseases
		2P03	Psoriasis
		2P04	Ulcers
		2Q02	Osteoarthritis
		2Q04	Slipped disc
		2Q06	Systemic lupus erythematosus
		2Q07	Gout
		2Qoth	Other musculoskeletal diseases
		2R01	Anencephaly
		2R02	Spina bifida
		2R03	Congenital heart disease
		2R04	Cleft lip and/or palate
		2R05a	Anorectal atresia
		2R05b	Oesophageal atresia
		2R05c	Other digestive system malformations
		2R06a	Renal agenesis
		2R06b	Other urogenital tract malformations
		2R07	Abdominal wall defect
		2S04	Pulpitis
		2Z02	Chronic fatigue syndrome
		3T	Unintentional injuries
		3U	Intentional injuries
Bettering the Evaluation And Care of Health	2000–01	1B03	Otitis media (E)
Western Australian Data Linkage System	1990–03	2L03	Stroke (E)

(continued)

Annex table 1 (continued): Principal data sources for epidemiological modelling

Primary data source	Reference period	Disease and injury categories	
C. Population health surveys			
National Trachoma Eye Health Program	1976–78	1A11	Trachoma (E)
		1B03	Otitis media (E)
National Health Survey			
Customised table	2004–05 and 2001	2M02	Asthma (E)
Main report	2004–05	2J01a	Alcohol dependence (E)
Main report	2004–05	2J03	Anxiety & depression (E)
CURF	2001	1B03	Otitis media
NSW Health Survey	1997–98	2J03	Anxiety & depression (E)
Western Australian Aboriginal Child Health Survey	2001–02	2J03	Anxiety & depression (E)
2004–06 National Oral Health Survey	2004–06	2S01	Dental caries (E)
	2004–06	2S02	Periodontal disease (E)
	2004–06	2S03	Edentulism (E)
South Australian Child and Adult Dental Health Surveys	1999–01	2S01	Dental caries (E)
D. Epidemiological studies			
Menzies et al. (2004)	2000–02	1A05e	Measles (E)
Menzies et al. (2004)	2000–02	1A05c	Tetanus (E)
National Trachoma and Eye Health Program Report (1980)	1998–99	1A11	Trachoma (E)
Kaldor et al. (1996)	1996[(b)]	1A09b	Hepatitis B (E)
Mott (2005)		2H02	Type 2 diabetes
Leonard et al. (2003)	1983–92	2K9	Intellectual disability
Glasson et al. (2005)	1953–2000	2K9	Intellectual disability (U)
Archer and Bunby (2006)	2001–05	2K02	Epilepsy (E)
Leonard et al. (2005)	1983–92	2J07b	Autism and Asperger's (E)
E. Indirect estimation			
YLD to YLL ratio from rest of category		1Aoth	Other infectious and parasitic diseases
		2Foth	Other malignant neoplasms
		2Goth	Other benign neoplasms
		2Ioth	Other endocrine and metabolic disorders
		2Koth	Other nervous system and sense organ disorders
		2Loth	Other cardiovascular disease
		2Moth	Other chronic respiratory diseases
		2Noth	Other digestive system diseases
		2Roth	Other congenital anomalies

(continued)

Annex table 1 (continued): Principal data sources for epidemiological modelling

Primary data source	Reference period	Disease and injury categories	
F. Assume same incidence as the total Australian population			
		1A09d	Other hepatitis
		2H01	Type 1 diabetes
		2J01c	Benzodiazepine dependence
		2J05	Personality disorders (isolated)
		2J07a	Attention-deficit hyperactivity disorder
		2K01	Alzheimer and other dementias
		2K03	Parkinson's disease
		2K05	Motor-neuron disease
		2K06	Huntington's chorea
		2K07	Muscular dystrophy
		2K08c	Macular degeneration
		2K08d	Adult-onset hearing loss
		2K08f	Other vision loss
		2K10	Migraine
		2N08	Inflammatory bowel disease
		2O03	Urinary incontinence
		2O04	Infertility
		2P01	Eczema
		2P02	Acne
		2Q01	Rheumatoid arthritis
		2Q03	Back pain (acute and chronic)
G. Condition assumed absent in Indigenous population			
		1A11	Trachoma (for the non-remote population)
		1A05a	Diphtheria
		1A05d	Poliomyelitis
		1A05f	Rubella
		1Doth	Other conditions arising in the perinatal period
		2Q05	Occupational overuse syndrome

(a) Liver cancer is the only explicit model (as it has different assumptions re: survival)
(b) Review published in 1996 — year of primary data collection is uncertain
(c) Four broad age groups
(U) Proportion by underlying cause
(E) Explicit model
(P) Uses procedures as basis of estimates instead of diagnosis mentions

Annex table 2: Assessment of quality of disease occurrence information for the main disabling conditions in the Indigenous population

		Total Australian population		Indigenous		
YLD rank	Disease condition	Main data source and application	Quality of data	Main data source and application	Assumptions	Quality of data[(a)]
1	Anxiety & depression	Derived from old but good-quality survey data: the 1997 National Survey of Mental Health and Wellbeing.	Good	Apply relative risks of the prevalence of psychological distress in Indigenous Australians (based on Kessler-10 questionnaire) from the 1997 and 1998 NSW Health Survey to the national model of anxiety & depression. Apply relative risks derived from published 2004–05 NATSIHS mental health and wellbeing module to adjust overall Indigenous estimates by remoteness.	Differences in K-10 results are indicative of the difference in the prevalence of the ICD-10 defined depression and anxiety between Indigenous and non-Indigenous Australians. The K-10 screening instrument is culturally appropriate for screening the mental health and wellbeing of Indigenous Australians. The 1997–98 NSW results hold for Australia in 2003. There is no difference in self-reporting errors (due to the sensitive nature of questions) between Indigenous and non-Indigenous people in the 2004–05 NATSIHS. Health survey relativities by remoteness reflect differentials in disease occurrence.	Fair
2	Type 2 diabetes	Based on measurement survey AusDiab 2000.	Good	Predict prevalence of all diabetes in 2003 from regression of Australian studies carried out over the past 20 years. Assume the incidence of Type 1 diabetes is same based on available literature. Estimate the incidence of Type 2 diabetes by taking away the incidence of Type 1 from the overall estimate. Apply rate ratios from Australian hospital data to adjust overall Indigenous estimates by remoteness.	The predicted prevalence (published studies are biased by remoteness) reflects actual disease occurrence in all Indigenous Australians. The epidemiology of Type 1 diabetes in the Indigenous population is the same as the total Australian population. Hospital relativities by remoteness reflect differentials in disease occurrence (i.e. no differential health care access for Indigenous Australians; and the adjustment of hospital data for Indigenous identification is appropriate and does not differ by disease category).	Fair
3	Asthma	Based on Australian studies that used results based on hyper-responsiveness test rather than the higher self-reported figures.	Good	Apply relative risks of self-reported asthma as a long-term health condition by remoteness from the 2004–05 National Health Survey.	Self-report health survey relativities by remoteness reflect differentials in disease occurrence.	Fair
4	Ischaemic heart disease	Based on hospital data for infarction, angina and heart failure.	Very good	Apply age-specific rate ratios of heart failure, from Australian hospital data. Estimate the underlying cause of heart failure from hospital records where both heart failure and cardiovascular disease (in this	Hospital relativities by remoteness reflect differentials in disease occurrence.	Good

(continued)

Annex table 2 (continued): Assessment of quality of disease occurrence information for the main disabling conditions in the Indigenous population

		Total Australian population		Indigenous		
YLD rank	Disease condition	Main data source and application	Quality of data	Main data source and application	Assumptions	Quality of data[(a)]
				case IHD) were mentioned in the same record. Apply age-specific rate ratios from hospital data to estimate angina pectoris and acute myocardial infarction by remoteness.		
5	Chronic obstructive pulmonary disease	Based on community study from WA Busselton.	Good	Apply age-specific rate ratios of COPD from Australian hospital data by remoteness.	Hospital relativities by remoteness reflect differentials in disease occurrence.	Fair
6	Schizophrenia	Derived from 'Low Prevalence Study' section of the 1997 National Survey of Mental Health and Wellbeing	Good	Half the excess risk from Australian hospital data reflects what is generally found for other 'ethnic' groups in the literature.	Large hospital differentials do not reflect disease occurrence. International literature on disease occurrence in 'ethnic' groups is representative of the Indigenous Australian experience.	Fair
7	Otitis media	General practice encounters for non-Indigenous Australians.	Fair	Apply rate ratios from Australian general practice data to estimate acute OM in non-remote areas. Apply rate ratios from the 1980 National Trachoma Program to estimate acute OM in remote areas. Adjust estimates of chronic OM in non-remote areas, based on relative risks using self-report of long term health conditions from the 2001 National Health Survey, to account only for bilateral cases using a ratio from the 1980 survey. Apply estimates from 1980 survey to estimate the prevalence of bilateral chronic OM in remote areas We use the GBD study (Murray & Lopez 1996) estimate of infection-related permanent hearing loss to estimate OM related hearing loss.	General practice data is not representative of Indigenous Australians residing in remote areas. Differentials from the 1980 trachoma survey for acute OM hold in 2003 for Indigenous Australians residing in remote areas. Differentials in the self-report of long-term health conditions reflect differentials in disease occurrence. Prevalence of chronic OM for Indigenous Australians residing in remote areas has not changed since the 1980 National Trachoma Program was undertaken.	Fair
8	Alcohol dependence	Derived from old but good-quality survey data: the 1997 National Survey of Mental Health and Wellbeing.	Good	Apply relative risks of self-reported risky/high risk alcohol consumption for Indigenous Australians from the 2004–05 National Health Survey to the national model. Apply relative risks of risky/high risk alcohol consumption by remoteness from the 2004-05 National Health Survey.	Differences in risky/high-risk average daily alcohol consumption in the seven days before the interview are indicative of the differences in the prevalence of the ICD-10-defined alcohol dependence between Indigenous and non-Indigenous Australians. There is no difference in self-reporting errors (due to the sensitive nature of questions) between Indigenous and non-Indigenous people in the 2004–05 NATSIHS. Health survey relativities by remoteness reflect differentials in disease occurrence.	Poor

(continued)

Annex table 2 (continued): Assessment of quality of disease occurrence information for the main disabling conditions in the Indigenous population

		Total Australian population		Indigenous		
YLD rank	Disease condition	Main data source and application	Quality of data	Main data source and application	Assumptions	Quality of data[(a)]
9	Stroke	Good data from Western Australia linked hospital and mortality data with some adjustments based on epidemiological studies for non-hospitalised cases, and level of severity. Adjust for mortality difference between Australia and Western Australia.	Very good	Judy Katzenellenbogen provided 28-day survivor stroke models for Indigenous Australians residing in remote and non-remote areas of Western Australia. We estimate the incidence for all remote and non-remote residing Indigenous Australians by adjusting the Western Australian incidence figures with the age-standardised Western Australian to Australian hospital separation rate ratio for stroke. The Western Australian rate is standardised to total Indigenous Australian remote or non-remote population.	The age-standardised Indigenous Western Australian to Indigenous Australian hospital separation rate ratio for stroke is an appropriate method for extrapolation to the total Indigenous population.	Good
10	Deficiency anaemia	Estimate from a variety Australian studies, AusDiab and the 1989 National Risk Factor Prevalence Survey.	Fair	Apply age- and sex-specific rate ratios for deficiency anaemia from Australian hospital data by remoteness.	Hospital relativities by remoteness reflect differentials in disease occurrence.	Fair
11	Dental caries	Good South Australian data on prevalence of DMFT.	Good	Apply relative risks derived from measured prevalence of dental caries in: (a) Indigenous children from the 2001 South Australian Child Dental Health Survey; (b) Indigenous adults from the 2004–06 National Oral Health Survey of Australia to the national model. Apply relative risks derived from measured prevalence of dental caries in Indigenous adults by remoteness.	Health survey relativities in the national survey and South Australian data reflect differentials in disease occurrence for Indigenous children and adults. South Australian data is based on children who use the school dental service and therefore may not representative of all Indigenous children. Using information from this same report, we assume that there are negligible differentials by remoteness for Indigenous children caries experience. 1–4-year-old Indigenous children have the same decay experience as 5–9-year-old Indigenous children. Dental caries are only possible in the dentate population.	Good
12	Heroin or polydrug dependence	Reasonable data sources (Degenhardt et al. 2004) due to difficulty in collecting information on a group of people often not included in 'normal' surveys. Estimates are more than 'ballpark' figures.	Fair	Apply rate ratios for heroin dependence from Australian hospital data by remoteness. These relativities are in keeping with findings for Indigenous people who currently inject illegal drugs from the 1994 National Drug Strategy Household Survey: Urban Aboriginal and Torres Strait Islander Peoples Supplement.	Hospital relativities by remoteness reflect differentials in disease occurrence.	Fair

(continued)

Annex table 2 (continued): Assessment of quality of disease occurrence information for the main disabling conditions in the Indigenous population

		Total Australian population		Indigenous		
YLD rank	Disease condition	Main data source and application	Quality of data	Main data source and application	Assumptions	Quality of data[(a)]
13	Low birth weight	Good data on neonatal conditions as almost all babies born in hospital.	Excellent	Apply published incidence data from the National Perinatal Statistics Unit, and various state and territory perinatal collections. Apply distributions by sex from Australian hospital data. Apply rate ratios of LBW from Australian hospital data by remoteness. Apply rate ratio of LBW in Indigenous infants from Australian perinatal data to LBW sequelae.	The differentials in Perinatal data reflect differentials in disease occurrence (i.e. not influenced by Indigenous identification issues). The probability and distribution of LBW sequelae among LBW survivors is the same for Indigenous and non-Indigenous Australians.	Good
14	Migraine	Prevalence is derived from 2001 National Health Survey data and incidence from an international study.	Good	No information.	The disease occurrence in the Indigenous population is the same as the total population.	Poor
15	Personality disorders (isolated)	Derived from the 1997 National Survey of Mental Health and Wellbeing.	Good	No information.	The disease occurrence in the Indigenous population is the same as the total population.	Poor
16	Homicide & violence	Extrapolated from hospitalised cases.	Very good	Apply rate ratios for homicide & violence from Australian hospital data to the national model.	Hospital relativities by remoteness reflect differentials in disease occurrence.	Good
17	Adult-onset hearing loss	Based on South Australian survey.	Fair	Assume the same prevalence as the national model.	The disease occurrence in the Indigenous population is the same as the total population.	Fair
18	Dementia	Based on overseas data but prevalence by age stable across many countries.	Good	No information.	The disease occurrence in the Indigenous population is the same as the total population.	Fair
19	Peripheral vascular disease	Extrapolated from hospitalised cases.	Good	The rate ratio of total Australian to remote and to non-remote Indigenous Australian peripheral vascular disease hospital separations was applied to the national model.	Hospital relativities by remoteness for peripheral vascular disease reflect differentials in disease occurrence. Hospital separations for peripheral vascular disease related toe amputations and foot or leg amputations reflect true incidence.	Good
20	Birth trauma & asphyxia	Good data on neonatal conditions as almost all babies born in hospital.	Very good	Apply rate ratios from Australian hospital data to the birth trauma-related hearing loss, seizure and cerebral palsy without intellectual disability in the Indigenous population by remoteness to the national models. Derive estimates of birth trauma-related intellectual disability from Western Australia data for Indigenous people.	Hospital relativities by remoteness reflect differentials in disease occurrence. Data on intellectual disability for Western Australian Indigenous people are indicative of Australian intellectual disability data for Indigenous Australians. No differentials by remoteness for the incidence of intellectual disability in Indigenous Australians.	Good

(continued)

Annex table 2 (continued): Assessment of quality of disease occurrence information for the main disabling conditions in the Indigenous population

		Total Australian population		Indigenous		
YLD rank	Disease condition	Main data source and application	Quality of data	Main data source and application	Assumptions	Quality of data[(a)]
	Injuries	Australian hospital separations data for 2002–03	Very good	Apply rate ratios for injuries from Australian hospital data to the national model.	Hospital relativities by remoteness reflect differentials in disease occurrence.	Good
	Cancers	Good data from cancer registry	Very good	Apply rate ratios for cancers by remoteness from Australian hospital data to the national model.	Hospital relativities by remoteness reflect differentials in disease occurrence.	Good

(a) Quality of data rated poor to very good

Annex table 3: Assessment of quality of risk factor exposure estimates in the Indigenous population

Risk factor	Data source	Assumptions	Confidence in prevalence estimates[(a)]
High blood pressure	DRUID (Cunningham et al. 2006); Wang & Hoy (Wang & Hoy 2003)	The DRUID and Wang & Hoy studies were both relatively small and based in one region each. We assume that the measured systolic blood pressure mean and standard deviations from these studies are representative of Indigenous Australians residing in non-remote and remote areas respectively.	2
High total cholesterol	DRUID (Cunningham et al. 2006); Wang & Hoy (Wang & Hoy 2003)	The DRUID and Wang & Hoy studies were both relatively small and based in one region each. We assume that the measured total cholesterol mean and standard deviations from these studies are representative of Indigenous Australians residing in non-remote and remote areas respectively.	2
High body mass	NATSIHS (Source: ABS 2006. ABS data available on request)	Measured height and weight information is used to estimate mean BMI for Indigenous Australians residing in remote areas. The relative difference between self-reported and measured BMI are assumed to be the same in Indigenous Australians residing in remote and non-remote areas, and is applied to mean self-reported BMI in Indigenous Australians residing in non-remote areas.	3 (non-remote) 4 (remote)
Insufficient fruit and vegetable consumption	NATSIHS (ABS 2006b)	Self-reported usual serves of fruit and vegetables are used to estimate categories of consumption for Indigenous Australians residing in non-remote areas. We assume that self-reported consumption reflects true levels. The same detail regarding amount of usual fruit and vegetable consumption is not available for Indigenous Australians residing in remote areas. For this group we can only directly estimate the proportion that does not usually consume fruit or vegetables. We distribute the remainder according to the distribution observed in the non-remote population. Therefore, we assume that those Indigenous Australians residing in remote areas that indicate that they usually consume fruit and/or vegetables are distributed the same as non-remote residing Indigenous Australians.	4 (non-remote) 3 (remote)
Physical inactivity	NATSIHS (Source: ABS 2006. ABS data available on request)	Prevalence of physical activity estimates are based on self-report data and we assume that self-reported activity reflects true levels. The survey only allows for the direct estimation of the inactive category in Indigenous Australians residing in remote areas. The level of physical activity among those that usually participate in physical activity in remote areas is distributed the same as Indigenous Australians residing in non-remote areas.	4 (non-remote) 3 (remote)

(continued)

Annex table 3 (continued): Assessment of quality of risk factor exposure estimates in the Indigenous population

Risk factor	Data source	Assumptions	Confidence in prevalence estimates[(a)]
Tobacco			
Past smoking	Peto–Lopez method	We use age-specific lung cancer mortality rates for males and females combined to estimate proportions of long lag-time conditions due to tobacco smoking. We assume that rates of lung cancer mortality are the same in Indigenous males and females residing in remote and non-remote areas, and by implication that levels of smoking were similar in these population groups over the past 20–30 years.	4
Current daily smokers	NATSIHS (ABS 2006b)	Prevalence of current smokers is based on self-report data and the assumption is that that reflects true prevalence.	4
Passive smoking	National Health Survey 1995 (cited in Ridolfo & Stevenson 2001)	We base passive smoking estimates on those used by Ridolfo & Stevenson, and therefore assume the proportion of people who are non-smokers and are exposed to spousal tobacco smoke is the same in the Indigenous Australian and total Australian population.	2
Maternal smoking and smoking while pregnant	Australia's Mothers and Babies 2003 (Laws & Sullivan 2005); WAACHS (Zubrick et al. 2004); Bibbulung Gnarneep ('Solid Kid') study (Eades et al. 1999)	The estimate of prevalence of smoking during pregnancy is based on self-reported smoking, and the assumption is that self-report data reflects true prevalence.	3
Alcohol	NATSIHS 2004–05 (ABS 2006b)	The NATSIHS 2004–05 collected self-report data on alcohol consumption. We assume that self-reported alcohol consumption reflects true prevalence.	4
Illicit drugs			
Drug use and dependence	Drug use and dependence models		
Use of illicit drugs	HIV/AIDS, hepatitis B, hepatitis C, inflammatory heart disease, pancreatitis, suicide, and RTAs population attributable fractions direct from the literature	We assume that hepatitis B and C, RTA, and suicide attributable fractions are the same in Indigenous and total Australian populations.	3

(continued)

Annex table 3 (continued): Assessment of quality of risk factor exposure estimates in the Indigenous population

Risk factor	Data source	Assumptions	Confidence in prevalence estimates[a]
Daily cannabis use	NDSHS: Aboriginal and Torres Strait Islander Supplement 1994 (Department of Human Services and Health 1995); NDSHS 1995 (Vuksa & Kelly 1996); NDSHS 2004 (AIHW & Australian Government Department of Health and Ageing 2005); hospitalisation data 2001–03	We apply the ratio of 2004 to 1995 estimated prevalence of daily cannabis use in the total Australian population to the 1994 estimate of prevalence in the non-remote residing Indigenous Australian population. We therefore assume that the increase seen in daily cannabis use in the National Drug Strategy Household Survey over the period 1995 –2004 also occurred in the Indigenous Australian population residing in non-remote areas. We use the rate ratio of hospital separations for cannabis dependence between Indigenous Australians residing in non-remote and remote areas to derive the estimates of prevalence of daily cannabis use for remote residing Indigenous Australians. The proportion of daily cannabis users hospitalised for reasons relating to harmful or dependant cannabis use are therefore assumed to be the same for Indigenous Australians residing in remote and non-remote areas.	3
Prenatal exposure cannabis and cocaine	Burns and colleagues (Burns et al. 2006)	Burns and colleagues used record linkage to examine illicit drug use in pregnancy in New South Wales. We assume that prevalence of drug use during pregnancy among Indigenous women in New South Wales can be generalised to all Indigenous Australian women.	3
Use heroin and cocaine while pregnant	NATSIHS 2004–05 (Source: ABS 2006. ABS data available on request)	The NATSIHS 2004-05 collected self reported data on illicit drug use. We assume self-reported use of heroin and cocaine reflects true prevalence.	3
Unsafe sex	Sexually transmissible diseases, abortion, and cervical cancer models; PAF direct from the literature (National Centre in HIV Epidemiology and Clinical Research 2005)		4
Child sexual abuse	Global Burden of Disease Comparative Risk assessment prevalence estimates for Australia; Child Protection data 2002–03 (AIHW 2004a)	We use child protection data to estimate the relative difference in prevalence of child sexual abuse between Indigenous and the total Australian population. This caries the assumption that child protection statistics cover the same proportion of abuse cases in the Indigenous and non-Indigenous Australian population. Our method assumes that the observed rate ratio of child sexual abuse between Indigenous and non-Indigenous Australian children in 2002–03 has been constant over time. This is necessary as we attribute disease outcomes in adulthood that are due to child sexual abuse in the past. For non-contact child sexual abuse we assume the same prevalence among Indigenous Australians as the national model; for contact-only child sexual abuse we apply half of the rate ratio; and for intercourse child sexual abuse we apply the whole rate ratio. This gradient of risk is based on the assumption that child sexual abuse substantiations reflect more severe abuse, and if this rate ratio were applied to all categories, the overall prevalence of child sexual abuse would be extremely high and for some age groups reach 100%. Given no data related to relative difference in prevalence of child sexual abuse in remote and non-remote areas, we assume that prevalence of child sexual abuse is the same in Indigenous	2 (non-remote) 1 (remote)

(continued)

Annex table 3 (continued): Assessment of quality of risk factor exposure estimates in the Indigenous population

Risk factor	Data source	Assumptions	Confidence in prevalence estimates[(a)]
		Australians residing in remote areas as those in non-remote areas.	
Intimate partner violence	Women's Safety Survey 1996 (ABS 1996); International Violence Against Women Survey (Mouzos & Makkai 2004); Supported Accommodation Assistance Program (AIHW 2005c)	We estimate the relative difference in experience of intimate partner violence based on prevalence of any violence from the Australian component of the International Violence Against Women Survey. It is therefore assumed that the estimates of proportion of women that have experienced violence from the IVAWS is representative of all Australian women, and non-remote residing Indigenous Australians. Since we apply ratios of any violence to intimate partner violence, we assume the same proportion of any violence experienced by Indigenous and all Australian women is perpetrated by an intimate partner. We use Supported Accommodation Assistance Program data to derive remote estimates. We therefore assume Indigenous women residing in remote and non-remote areas that experience intimate partner violence use supported accommodation at the same rate.	2

(a) 1 least confident tin our prevalence estimates o 5 most confident

Annex table 4: Disability-adjusted life years (DALYs) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
All causes	**10,811**	**13,366**	**15,278**	**10,652**	**50,107**	**9,376**	**12,004**	**13,218**	**11,271**	**45,869**	**95,976**
I. Communicable diseases, maternal and neonatal	**3,546**	**670**	**1,167**	**508**	**5,892**	**2,963**	**1,246**	**1,142**	**552**	**5,903**	**11,794**
A. Infectious and parasitic diseases	356	396	814	277	1,843	390	505	727	331	1,953	3,796
1. Tuberculosis	1	1	30	11	43	1	13	10	14	39	82
2. Sexually transmitted diseases	3	16	3	–	22	41	256	137	16	450	472
3. HIV/AIDS	1	80	79	3	162	–	39	34	1	74	236
9. Hepatitis	3	96	371	120	590	15	58	254	88	414	1,004
Other Infectious and parasitic diseases	348	204	331	143	1,026	334	138	291	213	976	2,002
B. Acute respiratory infections	676	259	335	220	1,490	630	312	291	190	1,423	2,913
1. Lower respiratory tract infections	248	124	292	214	878	224	154	239	181	798	1,676
2. Upper respiratory tract infections	61	24	9	2	96	67	37	13	3	120	216
3. Otitis media	367	111	35	3	516	339	121	39	5	505	1,021
C. Maternal conditions	–	–	–	–	–	1	151	9	–	161	161
D. Neonatal causes	2,327	–	–	–	2,327	1,720	–	–	–	1,720	4,047
2. Low birth weight	1,001	–	–	–	1,001	808	–	–	–	808	1,809
Other neonatal causes	1,326	–	–	–	1,326	912	–	–	–	912	2,238
E. Nutritional deficiencies	186	15	19	12	232	221	278	115	31	646	877
II. Non-communicable diseases	**6,221**	**7,746**	**12,069**	**9,827**	**35,863**	**5,691**	**8,569**	**11,205**	**10,469**	**35,934**	**71,798**
F. Malignant neoplasms	92	190	1,245	2,090	3,616	92	365	1,545	2,199	4,201	7,817
3. Stomach cancer	–	1	48	98	148	–	1	5	50	56	204
5. Liver cancer[(a)]	1	8	84	72	164	–	1	27	62	89	253
8. Lung cancer	–	1	284	709	995	–	1	338	606	945	1,940
12. Breast cancer	–	–	–	5	6	–	64	351	304	719	725
13. Cervical cancer	–	–	–	–	–	–	71	186	98	355	355
16. Prostate cancer	–	–	17	178	195	–	–	–	–	–	195
Other malignant neoplasms	92	179	811	1,026	2,108	91	227	637	1,081	2,036	4,144
G. Other neoplasms	13	2	29	46	89	31	5	42	40	117	206

(continued)

Annex table 4 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
H. Diabetes mellitus	104	713	1,859	1,162	3,837	79	774	2,061	1,747	4,661	8,498
I. Endocrine and metabolic disorders	173	39	130	106	447	48	121	210	171	550	997
J. Mental disorders	1,884	3,668	1,547	379	7,477	1,995	3,484	1,683	221	7,383	14,860
1. Substance use disorders	242	1,499	940	301	2,981	163	725	576	137	1,601	4,582
a. Alcohol dependence & harmful use[(b)]	241	438	818	300	1,797	145	236	492	135	1,008	2,805
b. Heroin or polydrug dependence & harmful use	–	674	95	1	771	7	371	74	1	453	1,223
Other drug dependence & harmful use	–	387	27	–	414	12	118	9	–	140	554
2. Schizophrenia	14	641	40	1	695	14	406	133	4	558	1,253
3. Anxiety & depression	1,149	1,207	456	52	2,864	1,656	2,028	856	42	4,582	7,446
Other mental disorders	479	321	111	26	937	161	325	118	38	643	1580
K. Nervous system and sense organ disorders	393	476	574	572	2,015	337	737	359	665	2,098	4,114
1. Dementia	17	12	22	141	191	1	–	14	292	307	498
2. Epilepsy	155	136	282	42	616	101	71	68	13	254	869
3. Parkinson's disease	–	–	13	60	73	–	–	4	67	71	144
8. Sense organ disorders	11	52	141	268	472	7	36	82	190	316	788
d. Adult-onset hearing loss	–	42	120	208	370	–	29	63	116	207	578
Vision loss	11	10	21	60	102	7	7	19	75	108	211
Other nervous system and sense organ disorders	210	277	116	61	663	228	630	191	102	1151	1,814
L. Cardiovascular disease	209	1,173	4,339	3,477	9,198	180	1,004	3,023	3,382	7,588	16,786
1. Rheumatic heart disease	63	108	121	32	324	24	247	251	138	660	984
2. Ischaemic heart disease	9	621	2,978	2,290	5,899	14	330	1,696	2,035	4,074	9,973
3. Stroke	19	119	449	705	1,293	73	152	495	693	1,413	2,706
4. Inflammatory heart disease	114	209	346	130	799	41	73	205	74	394	1,193
5. Hypertensive heart disease	–	2	28	75	106	1	1	59	81	141	247
Other cardiovascular disease	4	113	418	244	779	27	200	317	362	906	1,684
M. Chronic respiratory disease	1,202	551	1,365	1,277	4,395	1,210	712	1,141	1,129	4,192	8,587
1. Chronic obstructive pulmonary disease	8	228	777	927	1,941	6	179	759	735	1,678	3,619
2. Asthma	1,121	143	84	48	1,396	1,202	468	150	87	1,907	3,303

(continued)

Annex table 4 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
Other chronic respiratory diseases	72	180	504	302	1,058	2	64	232	308	607	1,664
N. Diseases of the digestive system	27	168	306	217	718	26	101	288	199	613	1,331
1. Peptic ulcer disease	–	9	57	28	95	–	1	44	11	56	151
2. Cirrhosis of the liver[c]	–	7	34	12	53	–	7	27	9	43	95
Other digestive system diseases	27	152	215	176	571	26	93	217	179	515	1,085
O. Genitourinary diseases	1	179	177	242	600	97	602	270	329	1,297	1,897
1. Nephritis & nephrosis[d]	–	60	134	147	342	27	17	144	193	381	723
Other genitourinary diseases	1	119	43	95	258	70	584	126	135	916	1,174
P. Skin diseases	88	109	67	58	322	82	131	101	101	416	738
Q. Musculoskeletal diseases	33	113	207	140	493	57	196	265	202	720	1,212
1. Rheumatoid arthritis	13	10	19	15	58	43	30	75	44	191	249
2. Osteoarthritis	–	7	31	33	71	–	2	27	62	91	162
3. Back pain/slipped disc	13	54	92	52	210	10	65	111	49	236	445
Other musculoskeletal diseases	7	42	66	40	154	5	99	52	47	202	356
R. Congenital anomalies	1,458	116	45	11	1,630	989	76	14	9	1,088	2,718
S. Oral conditions	130	230	144	47	551	126	244	165	61	595	1,146
1. Dental caries	85	177	97	29	388	80	185	106	34	405	793
Other oral conditions	45	53	47	18	163	46	59	58	27	190	353
Z. Ill-defined conditions	413	21	35	5	475	342	19	40	14	415	890
1. Sudden infant death syndrome	413	–	–	–	413	342	–	–	–	342	755
Other ill-defined conditions	–	21	35	5	61	–	19	40	14	73	134
III. Injuries	**1,044**	**4,950**	**2,042**	**316**	**8,352**	**722**	**2,190**	**871**	**250**	**4,032**	**12,384**
T. Unintentional injuries	907	2,293	1,157	249	4,605	580	998	573	232	2,383	6,989
1. Road traffic accidents	208	1,158	520	69	1,955	139	595	293	48	1,074	3,030
2. Other transport accidents	107	109	87	21	324	8	42	25	1	76	400
3. Poisoning	12	181	68	24	285	2	155	72	27	256	541

(continued)

Annex table 4 (continued): Disability-adjusted life years (DALYs) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
4. Falls	128	135	97	70	431	68	22	46	88	224	655
5. Fires, burns and scalds	91	56	46	16	210	64	33	34	10	142	352
6. Drowning	61	107	117	5	291	63	28	34	1	126	417
Other unintentional injuries	299	546	222	43	1,109	237	122	69	57	485	1,594
U. Intentional injuries	137	2,657	885	67	3,746	142	1,192	298	18	1,649	5,395
1. Suicide and self-inflicted injuries	29	1,973	583	59	2,644	84	568	143	–	795	3,439
2. Homicide & violence	108	684	302	8	1,102	58	624	155	17	854	1,956
Alternative burden of disease categories											
All intellectual disability	2,884	113	280	40	3,317	1,836	35	62	10	1,943	5,261
All vision loss	26	46	127	134	333	20	45	136	325	526	859
All renal failure	45	355	1,024	920	2,345	45	199	973	1,361	2,578	4,923

(a) Liver cancer (excluding hepatitis B- and C-related)
(b) Alcohol dependence & harmful use (including alcoholic cirrhosis)
(c) Cirrhosis of the liver (excluding alcoholic and hepatic cirrhosis)
(d) Nephritis & nephrosis (excluding diabetic-, congenital- and poisoning-related renal failure)
– nil or rounded down to zero

Annex table 5: Years of life lost (YLL) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
All causes	**3,898**	**6,735**	**10,116**	**8,155**	**28,904**	**3,164**	**3,844**	**7,146**	**8,416**	**22,571**	**51,475**
I. Communicable diseases, maternal and neonatal conditions	**1,707**	**287**	**835**	**398**	**3,228**	**1,466**	**301**	**588**	**371**	**2,727**	**5,954**
A. Infectious and parasitic diseases	210	205	575	196	1,186	196	145	390	198	928	2,114
1. Tuberculosis	–	–	29	11	40	–	12	9	13	35	75
2. Sexually transmitted diseases	–	–	–	–	–	13	26	32	6	76	76
3. HIV/AIDS	–	15	37	–	52	–	23	24	–	48	100
9. Hepatitis	1	83	348	114	546	13	46	246	84	389	935
Other Infectious and parasitic diseases	209	107	161	71	549	170	37	78	94	380	928
B. Acute respiratory infections	188	82	259	198	727	185	99	199	161	645	1,372
1. Lower respiratory tract infections	172	82	259	198	711	170	88	199	161	618	1,329
2. Upper respiratory tract infections	16	–	–	–	16	15	–	–	–	15	31
3. Otitis media	–	–	–	–	–	–	11	–	–	11	12
C. Maternal conditions	–	–	–	–	–	–	57	–	–	57	57
D. Neonatal causes	1,309	–	–	–	1,309	1,070	–	–	–	1,070	2,379
2. Low birth weight	584	–	–	–	584	509	–	–	–	509	1,092
Other neonatal causes	725	–	–	–	725	562	–	–	–	562	1,287
E. Nutritional deficiencies	–	–	–	5	5	15	–	–	12	27	32
II. Non-communicable diseases	**1,527**	**2,182**	**7,429**	**7,476**	**18,614**	**1,250**	**1,711**	**5,802**	**7,846**	**16,608**	**35,223**
F. Malignant neoplasms	82	165	1,176	1,981	3,404	85	330	1,429	2,103	3,947	7,351
3. Stomach cancer	–	1	46	95	142	–	1	4	49	54	196
5. Liver cancer[(a)]	–	8	83	71	162	–	1	26	61	88	250
8. Lung cancer	–	1	279	691	971	–	1	331	591	923	1,894
12. Breast cancer	–	–	–	5	6	–	60	306	276	641	647
13. Cervical cancer	–	–	–	–	–	–	59	170	95	323	323
16. Prostate cancer	–	–	11	151	162	–	–	–	–	–	162
Other malignant neoplasms	81	155	756	969	1,961	85	209	592	1,032	1,917	3,879
G. Other neoplasms	12	1	27	44	84	29	1	23	36	88	172

(continued)

Annex table 5 (continued): Years of life lost (YLL) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
H. Diabetes mellitus	–	93	753	730	1,576	1	18	684	1,272	1,976	3,552
I. Endocrine and metabolic disorders	64	27	87	63	242	6	75	134	101	316	558
J. Mental disorders	1	468	778	291	1,538	27	305	515	139	987	2,525
1. Substance use disorders	1	456	769	284	1,510	27	304	515	129	976	2,485
a. Alcohol dependence & harmful use[(b)]	–	174	667	284	1,125	15	162	452	129	758	1,883
b. Heroin or polydrug dependence & harmful use	–	178	80	–	258	–	126	63	–	189	447
Other drug dependence & harmful use	–	104	22	–	126	12	16	–	–	28	155
2. Schizophrenia	–	–	–	–	–	–	–	–	–	–	–
3. Anxiety & depression	–	–	9	–	9	–	–	–	–	–	9
Other mental disorders	–	12	–	7	19	–	1	–	10	11	30
K. Nervous system and sense organ disorders	156	212	363	144	875	109	160	127	214	610	1,485
1. Dementia	17	10	8	43	78	1	–	9	137	147	225
2. Epilepsy	52	100	277	40	469	26	34	62	10	133	602
3. Parkinson's disease	–	–	–	17	17	–	–	–	15	15	31
8. Sense organ disorders	–	–	–	–	–	–	–	–	–	–	–
d. Adult-onset hearing loss	–	–	–	–	–	–	–	–	–	–	–
Vision loss	–	–	–	–	–	–	–	–	–	–	–
Other nervous system and sense organ disorders	87	101	78	44	311	82	126	55	52	315	626
L. Cardiovascular disease	132	809	3,415	2,910	7,267	83	544	1,997	2,682	5,306	12,573
1. Rheumatic heart disease	39	71	89	19	218	–	174	178	103	455	672
2. Ischaemic heart disease	–	457	2,522	2,048	5,026	–	143	1,152	1,700	2,995	8,021
3. Stroke	12	81	294	511	899	42	60	321	510	932	1,831
4. Inflammatory heart disease	79	119	279	106	584	28	33	159	52	271	855
5. Hypertensive heart disease	–	–	25	73	98	–	–	57	78	135	233
Other cardiovascular disease	1	82	206	153	442	14	134	131	240	519	961
M. Chronic respiratory disease	49	117	439	863	1,469	18	82	451	752	1,303	2,771
1. Chronic obstructive pulmonary disease	3	11	158	692	864	1	13	251	542	807	1,671
2. Asthma	14	26	57	37	134	15	33	82	59	189	323
Other chronic respiratory diseases	32	80	225	134	471	1	36	118	151	306	777

(continued)

Annex table 5 (continued): Years of life lost (YLL) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
N. Diseases of the digestive system	14	125	238	187	564	14	61	197	166	438	1,002
1. Peptic ulcer disease	–	–	45	24	70	–	–	27	9	37	106
2. Cirrhosis of the liver[(c)]	–	7	33	11	51	–	7	27	8	42	92
Other digestive system diseases	14	118	160	152	444	14	54	143	149	360	804
O. Genitourinary diseases	–	53	106	194	353	26	25	142	299	492	845
1. Nephritis & nephrosis[(d)]	–	53	106	138	297	26	10	115	182	333	630
Other genitourinary diseases	–	–	–	56	56	–	15	27	116	159	215
P. Skin diseases	–	13	–	20	33	–	–	53	17	71	104
Q. Musculoskeletal diseases	–	–	10	35	46	–	51	38	46	134	180
1. Rheumatoid arthritis	–	–	–	3	3	–	–	9	14	23	26
2. Osteoarthritis	–	–	–	2	2	–	–	–	–	–	2
3. Back pain/slipped disc	–	–	–	6	6	–	–	–	–	–	6
Other musculoskeletal diseases	–	–	10	24	34	–	50	30	31	111	146
R. Congenital anomalies	603	98	36	10	747	509	58	12	8	587	1,334
S. Oral conditions	–	–	–	–	–	–	–	–	–	–	–
1. Dental caries	–	–	–	–	–	–	–	–	–	–	–
Other oral conditions	–	–	–	–	–	–	–	–	–	–	–
Z. Ill-defined conditions	413	–	–	3	416	342	–	–	11	353	770
1. Sudden infant death syndrome	413	–	–	–	413	342	–	–	–	342	755
Other ill-defined conditions	–	–	–	3	3	–	–	–	11	11	14
III. Injuries	**664**	**4,266**	**1,853**	**281**	**7,063**	**448**	**1,832**	**755**	**200**	**3,235**	**10,298**
T. Unintentional injuries	551	1,847	1,017	217	3,632	332	866	510	185	1,892	5,524
1. Road traffic accidents	186	1,038	496	66	1,786	126	555	281	46	1,008	2,794
2. Other transport accidents	87	74	79	20	260	–	30	23	–	54	314
3. Poisoning	11	178	67	24	280	–	153	71	26	250	530
4. Falls	22	59	68	55	204	–	1	33	53	87	291
5. Fires, burns and scalds	26	26	34	15	101	11	27	22	10	70	171
6. Drowning	61	106	117	5	290	63	28	34	1	126	415
Other unintentional injuries	158	365	156	32	711	132	70	45	50	298	1,009

(continued)

Annex table 5 (continued): Years of life lost (YLL) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
U. Intentional injuries	112	2,419	835	64	3,430	116	967	245	15	1,343	4,774
1. Suicide and self-inflicted injuries	29	1,961	579	59	2,628	84	558	141	–	783	3,411
2. Homicide & violence	83	458	256	5	802	32	409	104	15	561	1,363
Alternative burden of disease categories											
All intellectual disability	1,513	113	280	40	1,947	1,064	35	62	10	1,171	3,118
All vision loss	–	–	–	23	24	–	–	9	202	210	234
All renal failure	44	337	940	881	2,203	44	180	876	1,305	2,405	4,608

(a) Liver cancer (excluding hepatitis B- and C-related)
(b) Alcohol dependence & harmful use (including alcoholic cirrhosis)
(c) Cirrhosis of the liver (excluding alcoholic and hepatic cirrhosis)
(d) Nephritis & nephrosis (excluding diabetic-, congenital- and poisoning-related renal failure)
– nil or rounded down to zero

Annex table 6: Years lived with disability (YLD) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
All causes	**6,913**	**6,631**	**5,162**	**2,497**	**21,202**	**6,212**	**8,160**	**6,072**	**2,854**	**23,299**	**44,501**
I. Communicable diseases, maternal and neonatal conditions	**1,839**	**383**	**333**	**110**	**2,664**	**1,497**	**944**	**553**	**181**	**3,176**	**5,840**
A. Infectious and parasitic diseases	146	191	238	81	657	194	360	337	134	1,025	1,682
1. Tuberculosis	1	1	1	1	3	1	1	1	1	3	7
2. Sexually transmitted diseases	3	16	3	–	22	28	230	105	10	373	396
3. HIV/AIDS	–	65	42	2	110	–	16	10	1	26	136
9. Hepatitis	3	12	23	6	44	2	12	8	3	25	69
Other Infectious and parasitic diseases	139	96	171	72	477	164	101	213	119	596	1,074
B. Acute respiratory infections	488	177	76	22	763	445	213	92	29	778	1,541
1. Lower respiratory tract infections	76	43	32	17	167	54	66	40	20	180	347
2. Upper respiratory tract infections	46	24	9	2	80	52	37	13	3	105	185
3. Otitis media	367	110	35	3	515	339	110	39	5	493	1,009
C. Maternal conditions	–	–	–	–	–	1	94	9	–	104	104
D. Neonatal causes	1,018	–	–	–	1,018	650	–	–	–	650	1,668
2. Low birth weight	417	–	–	–	417	299	–	–	–	299	717
Other neonatal causes	601	–	–	–	601	351	–	–	–	351	951
E. Nutritional deficiencies	186	15	19	7	226	207	278	115	19	619	845
II. Non-communicable diseases	**4,694**	**5,564**	**4,640**	**2,351**	**17,249**	**4,441**	**6,859**	**5,403**	**2,623**	**19,326**	**36,575**
F. Malignant neoplasms	11	25	68	108	212	7	35	115	97	254	466
3. Stomach cancer	–	–	2	4	6	–	–	1	1	1	7
5. Liver cancer[(a)]	–	–	1	1	2	–	–	–	–	1	3
8. Lung cancer	–	–	5	18	24	–	–	7	15	22	46
12. Breast cancer	–	–	–	–	–	–	4	46	28	78	78
13. Cervical cancer	–	–	–	–	–	–	13	17	3	32	32
16. Prostate cancer	–	–	6	28	33	–	–	–	–	–	33
Other malignant neoplasms	10	24	55	58	147	7	17	45	49	119	266

(continued)

Annex table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
G. Other neoplasms	1	–	2	2	5	2	4	19	4	29	34
H. Diabetes mellitus	104	620	1,106	431	2,261	78	756	1,377	475	2,685	4,946
I. Endocrine and metabolic disorders	108	12	43	42	205	43	46	76	70	234	439
J. Mental disorders	1,882	3,199	769	89	5,939	1,968	3,179	1,168	82	6,396	12,335
1. Substance use disorders	241	1,043	171	17	1,472	136	420	61	7	625	2,097
a. Alcohol dependence & harmful use[(b)]	241	264	151	16	672	130	73	40	6	250	922
b. Heroin or polydrug dependence & harmful use	–	496	15	1	512	7	245	11	1	264	776
Other drug dependence & harmful use	–	282	5	–	287	–	102	9	–	111	399
2. Schizophrenia	14	641	40	1	695	14	406	133	4	558	1,252
3. Anxiety & depression	1,149	1,207	447	52	2,855	1,656	2,028	856	42	4,582	7,437
Other mental disorders	479	309	111	19	918	161	324	118	28	632	1,549
K. Nervous system and sense organ disorders	237	264	211	428	1,140	228	577	233	451	1,489	2,629
1. Dementia	–	1	14	98	113	–	–	5	155	160	273
2. Epilepsy	103	36	5	3	147	74	37	6	3	121	267
3. Parkinson's disease	–	–	13	43	56	–	–	4	52	56	112
8. Sense organ disorders	11	52	141	268	472	7	36	82	190	316	788
d. Adult-onset hearing loss	–	42	120	208	370	–	29	63	116	207	578
Vision loss	11	10	21	60	102	7	7	19	75	108	211
Other nervous system and sense organ disorders	122	175	38	16	352	146	504	135	51	836	1,188
L. Cardiovascular disease	77	363	925	566	1,932	97	460	1,026	700	2,282	4,214
1. Rheumatic heart disease	24	37	33	13	106	24	73	73	35	205	311
2. Ischaemic heart disease	9	165	456	242	872	14	187	544	334	1,080	1,952
3. Stroke	7	39	155	194	394	31	92	175	183	481	875
4. Inflammatory heart disease	34	90	67	24	215	14	40	46	23	123	338
5. Hypertensive heart disease	–	2	3	3	8	1	1	2	3	6	14
Other cardiovascular disease	2	31	212	91	336	13	66	186	122	387	723
M. Chronic respiratory disease	1,152	434	926	414	2,926	1,193	630	689	377	2,890	5,816
1. Chronic obstructive pulmonary disease	5	217	619	235	1,077	4	166	508	193	872	1,948
2. Asthma	1,107	117	27	11	1,262	1,187	435	68	27	1,718	2,980

(continued)

Annex table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
Other chronic respiratory diseases	40	100	280	167	587	1	28	114	157	300	887
N. Diseases of the digestive system	14	43	68	30	154	12	40	91	33	175	329
1. Peptic ulcer disease	–	9	12	5	25	–	1	16	2	19	44
2. Cirrhosis of the liver[(c)]	–	–	1	1	2	–	–	1	–	1	3
Other digestive system diseases	14	34	55	24	127	12	39	74	31	155	282
O. Genitourinary diseases	1	126	71	49	247	71	576	128	30	805	1,051
1. Nephritis & nephrosis[(d)]	–	8	28	9	45	–	7	29	11	48	92
Other genitourinary diseases	1	119	43	39	202	70	569	99	19	757	959
P. Skin diseases	88	96	67	38	289	82	131	48	84	345	634
Q. Musculoskeletal diseases	33	112	197	104	447	57	146	226	156	585	1,032
1. Rheumatoid arthritis	13	10	19	13	55	43	30	66	29	168	223
2. Osteoarthritis	–	7	31	31	69	–	2	27	62	91	160
3. Back pain/slipped disc	13	54	92	45	203	10	65	111	49	236	439
Other musculoskeletal diseases	7	41	56	16	120	5	48	22	16	91	211
R. Congenital anomalies	856	17	9	1	883	479	18	2	1	501	1,384
S. Oral conditions	130	230	144	47	551	126	244	165	61	595	1,146
1. Dental caries	85	177	97	29	388	80	185	106	34	405	793
Other oral conditions	45	53	47	18	163	46	59	58	27	190	353
Z. Ill-defined conditions	–	21	35	2	58	–	19	40	2	62	120
1. Sudden infant death syndrome	–	–	–	–	–	–	–	–	–	–	–
Other ill-defined conditions	–	21	35	2	58	–	19	40	2	62	120
III. Injuries	**380**	**684**	**189**	**36**	**1,289**	**274**	**357**	**116**	**50**	**797**	**2,086**
T. Unintentional injuries	355	446	139	33	973	248	132	64	47	491	1,464
1. Road traffic accidents	22	120	24	3	169	13	40	11	2	66	235
2. Other transport accidents	20	35	8	1	64	8	12	2	1	22	87
3. Poisoning	2	3	1	–	5	2	2	1	1	5	11
4. Falls	106	76	28	16	226	68	21	13	36	138	364
5. Fires, burns and scalds	65	31	12	1	109	53	6	12	1	72	181
6. Drowning	–	1	–	–	1	–	–	–	–	–	1

(continued)

Annex table 6 (continued): Years lived with disability (YLD) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
Other unintentional injuries	141	180	66	11	398	104	52	24	7	188	585
U. Intentional injuries	25	239	50	3	316	25	225	53	3	305	622
1. Suicide and self-inflicted injuries	–	12	4	1	16	–	10	2	–	13	29
2. Homicide & violence	25	227	46	3	300	25	215	51	2	293	593
Alternative burden of disease categories											
All intellectual disability	1,370	–	–	–	1,370	772	–	–	–	772	2,142
All vision loss	26	46	127	110	309	20	45	127	124	316	625
All renal failure	1	18	84	39	142	2	19	97	56	173	315

(a) Liver cancer (excluding hepatitis B- and C-related)
(b) Alcohol dependence & harmful use (including alcoholic cirrhosis)
(c) Cirrhosis of the liver (excluding alcoholic and hepatic cirrhosis)
(d) Nephritis & nephrosis (excluding diabetic-, congenital- and poisoning-related renal failure)
– nil or rounded down to zero

Annex table 7: Prevalent years lived with disability (PYLD) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
All causes	**3,491**	**5,952**	**6,561**	**3,959**	**19,964**	**2,956**	**6,748**	**6,831**	**4,381**	**20,917**	**40,880**
I. Communicable diseases, maternal and neonatal conditions	**1,189**	**801**	**634**	**281**	**2,905**	**1,003**	**1,100**	**781**	**316**	**3,200**	**6,105**
A. Infectious and parasitic diseases	112	123	270	171	676	149	259	372	212	993	1,669
1. Tuberculosis	1	1	1	1	3	1	1	1	1	3	7
2. Sexually transmitted diseases	3	15	3	1	22	15	167	165	22	369	392
3. HIV/AIDS	1	25	94	23	142	1	4	9	2	16	159
9. Hepatitis	2	1	16	7	26	1	1	4	4	10	36
Other Infectious and parasitic diseases	105	81	156	139	482	132	85	193	184	594	1,076
B. Acute respiratory infections	438	200	86	24	749	397	233	102	32	764	1,513
1. Lower respiratory tract infections	74	43	32	16	164	53	65	40	20	177	342
2. Upper respiratory tract infections	45	24	9	2	79	50	37	13	3	103	182
3. Otitis media	320	134	46	6	506	294	131	49	9	483	989
C. Maternal conditions	–	–	–	–	–	1	81	26	1	109	109
D. Neonatal causes	525	464	259	79	1,327	334	311	180	51	876	2,202
2. Low birth weight	217	195	108	33	553	155	148	84	23	410	963
Other neonatal causes	308	269	151	46	774	179	163	95	28	465	1,239
E. Nutritional deficiencies	114	14	18	7	153	122	216	101	20	459	612
II. Non-communicable diseases	**2,168**	**4,658**	**5,411**	**3,472**	**15,710**	**1,851**	**5,370**	**5,737**	**3,902**	**16,860**	**32,570**
F. Malignant neoplasms	10	21	59	111	200	6	27	100	102	236	436
3. Stomach cancer	–	–	2	4	6	–	–	–	1	1	7
5. Liver cancer[(a)]	–	–	1	1	2	–	–	–	1	1	3
8. Lung cancer	–	–	4	18	22	–	–	5	15	20	43
12. Breast cancer	–	–	–	–	–	–	3	38	31	72	72
13. Cervical cancer	–	–	–	–	–	–	9	17	3	30	30
16. Prostate cancer	–	–	4	27	30	–	–	–	–	–	30
Other malignant neoplasms	9	21	49	61	140	6	15	39	52	112	252
G. Other neoplasms	1	–	2	2	5	2	3	19	5	28	33

(continued)

Annex table 7 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
H. Diabetes mellitus	23	239	888	686	1,836	20	251	987	783	2,041	3,877
I. Endocrine and metabolic disorders	78	27	42	38	186	25	53	69	60	207	392
J. Mental disorders	742	2,538	1,951	418	5,649	608	2,606	1,934	517	5,664	11,313
1. Substance use disorders	125	794	446	59	1,423	68	349	208	31	655	2,078
a. Alcohol dependence & harmful use[(b)]	125	304	230	39	698	67	119	90	19	294	992
b. Heroin or polydrug dependence & harmful use	–	293	163	17	473	1	154	88	10	253	726
Other drug dependence & harmful use	–	197	53	3	252	–	76	29	3	108	360
2. Schizophrenia	2	320	283	64	669	3	173	264	85	525	1,194
3. Anxiety & depression	292	1,096	1,014	247	2,648	416	1,830	1,275	352	3,873	6,522
Other mental disorders	324	328	208	48	909	121	254	187	48	610	1,519
K. Nervous system and sense organ disorders	140	217	210	453	1,021	159	383	317	469	1,328	2,350
1. Dementia	–	–	6	97	104	–	–	1	139	140	244
2. Epilepsy	48	64	34	10	156	33	53	30	9	125	281
3. Parkinson's disease	–	–	4	47	51	–	–	1	50	51	102
8. Sense organ disorders	9	30	85	272	395	6	19	55	195	275	671
d. Adult-onset hearing loss	–	20	68	212	300	–	11	40	124	176	476
Vision loss	9	10	17	59	95	6	7	15	71	99	194
Other nervous system and sense organ disorders	84	122	81	28	315	120	311	230	75	737	1,052
L. Cardiovascular disease	29	201	691	763	1,684	33	250	752	933	1,968	3,653
1. Rheumatic heart disease	11	33	37	20	101	10	51	79	49	189	290
2. Ischaemic heart disease	3	69	333	337	742	4	74	368	444	889	1,631
3. Stroke	2	21	103	220	346	8	54	143	238	443	789
4. Inflammatory heart disease	12	65	87	40	203	4	27	48	34	113	317
5. Hypertensive heart disease	–	1	2	3	7	–	1	2	3	6	13
Other cardiovascular disease	1	13	129	143	286	7	43	113	165	328	614
M. Chronic respiratory disease	406	527	810	617	2,360	475	625	702	580	2,381	4,741
1. Chronic obstructive pulmonary disease	1	61	413	428	903	1	49	304	354	708	1,611
2. Asthma	371	379	156	46	952	473	550	302	97	1,422	2,373
Other chronic respiratory diseases	34	87	242	143	505	1	26	96	129	252	757

(continued)

Annex table 7 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
N. Diseases of the digestive system	10	34	68	36	148	8	30	88	42	167	315
1. Peptic ulcer disease	–	7	11	6	24	–	1	12	4	17	41
2. Cirrhosis of the liver[c]	–	–	1	1	2	–	–	–	–	1	3
Other digestive system diseases	10	27	57	29	123	8	29	75	37	149	271
O. Genitourinary diseases	–	96	95	53	245	17	434	233	44	728	973
1. Nephritis & nephrosis[d]	–	4	25	13	42	–	4	24	15	43	85
Other genitourinary diseases	–	92	71	40	203	17	430	208	29	685	888
P. Skin diseases	60	106	71	41	278	48	149	53	75	326	604
Q. Musculoskeletal diseases	14	69	163	145	391	24	116	183	191	514	906
1. Rheumatoid arthritis	4	12	15	19	49	17	34	50	48	149	198
2. Osteoarthritis	–	2	22	35	60	–	1	16	60	77	137
3. Back pain/slipped disc	6	35	78	58	179	5	43	94	64	205	384
Other musculoskeletal diseases	4	19	48	33	104	3	39	24	18	83	187
R. Congenital anomalies	533	361	194	56	1,144	307	215	113	34	669	1,813
S. Oral conditions	122	206	134	46	508	119	218	151	59	547	1,055
1. Dental caries	79	156	90	27	352	75	163	98	31	367	719
Other oral conditions	43	50	44	19	156	44	55	53	28	180	336
Z. Ill-defined conditions	–	14	33	7	54	–	11	36	8	55	109
1. Sudden infant death syndrome	–	–	–	–	–	–	–	–	–	–	–
Other ill-defined conditions	–	14	33	7	54	–	11	36	8	55	109
III. Injuries	**135**	**492**	**516**	**206**	**1,349**	**102**	**278**	**313**	**163**	**857**	**2,205**
T. Unintentional injuries	126	362	372	158	1,019	93	177	165	107	542	1,560
1. Road traffic accidents	6	65	78	25	174	3	27	28	12	70	244
2. Other transport accidents	6	26	26	8	66	2	9	9	3	24	90
3. Poisoning	1	2	2	1	5	1	2	1	2	6	11
4. Falls	47	82	72	37	237	32	39	34	44	148	386
5. Fires, burns and scalds	21	45	40	16	122	20	28	26	12	86	207
6. Drowning	–	–	–	–	1	–	–	–	–	–	1
Other unintentional injuries	45	142	155	71	412	35	73	67	34	208	620

(continued)

Annex table 7 (continued): Prevalent years lived with disability (PYLD) by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
U. Intentional injuries	8	130	144	48	330	9	100	149	57	315	645
1. Suicide and self-inflicted injuries	–	6	7	3	16	–	5	6	2	12	28
2. Homicide & violence	8	124	137	45	314	9	96	143	55	303	617

(a) Liver cancer (excluding hepatitis B- and C-related)
(b) Alcohol dependence & harmful use (including alcoholic cirrhosis)
(c) Cirrhosis of the liver (excluding alcoholic and hepatic cirrhosis)
(d) Nephritis & nephrosis (excluding diabetic-, congenital- and poisoning-related renal failure)
– nil or rounded down to zero

Annex table 8: Deaths by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
All causes	**129**	**254**	**468**	**723**	**1,574**	**104**	**142**	**318**	**731**	**1,296**	**2,870**
I. Communicable diseases, maternal and neonatal	**56**	**11**	**39**	**36**	**142**	**48**	**11**	**26**	**37**	**122**	**264**
A. Infectious and parasitic diseases	7	8	27	16	58	6	5	17	16	45	103
1. Tuberculosis	–	–	1	1	2	–	–	–	2	3	5
2. Sexually transmitted diseases	–	–	–	–	–	–	1	1	–	3	3
3. HIV/AIDS	–	1	2	–	2	–	1	1	–	2	4
9. Hepatitis	–	3	16	9	28	–	2	11	6	19	47
Other Infectious and parasitic diseases	7	4	7	7	25	6	1	3	8	18	43
B. Acute respiratory infections	6	3	12	19	41	6	4	9	19	38	78
1. Lower respiratory tract infections	6	3	12	19	40	6	3	9	19	37	77
2. Upper respiratory tract infections	1	–	–	–	1	1	–	–	–	1	1
3. Otitis media	–	–	–	–	–	–	–	–	–	–	–
C. Maternal conditions	–	–	–	–	–	–	2	–	–	2	2
D. Neonatal causes	43	–	–	–	43	35	–	–	–	35	78
2. Low birth weight	19	–	–	–	19	17	–	–	–	17	36
Other neonatal causes	24	–	–	–	24	18	–	–	–	18	42
E. Nutritional deficiencies	–	–	–	–	–	–	–	–	2	2	3
II. Non-communicable diseases	**51**	**83**	**347**	**665**	**1,146**	**41**	**64**	**260**	**677**	**1,042**	**2,188**
F. Malignant neoplasms	3	6	56	167	232	3	12	64	159	239	470
3. Stomach cancer	–	–	2	8	10	–	–	–	3	3	14
5. Liver cancer[(a)]	–	–	4	6	10	–	–	1	5	6	16
8. Lung cancer	–	–	13	55	68	–	–	15	43	58	126
12. Breast cancer	–	–	–	–	–	–	2	14	22	37	38
13. Cervical cancer	–	–	–	–	–	–	2	7	8	18	18
16. Prostate cancer	–	–	1	15	16	–	–	–	–	–	16
Other malignant neoplasms	3	6	36	83	128	3	8	27	79	117	244

(continued)

Annex table 8 (continued): Deaths by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
G. Other neoplasms	–	–	1	5	7	1	–	1	3	5	12
H. Diabetes mellitus	–	4	36	57	97	–	1	31	97	130	227
I. Endocrine and metabolic disorders	2	1	4	5	12	–	3	6	10	19	31
J. Mental disorders	–	18	36	22	76	1	11	22	9	44	120
1. Substance use disorders	–	17	36	21	74	1	11	22	9	43	117
a. Alcohol dependence & harmful use[(b)]	–	7	31	21	59	1	6	20	9	35	94
b. Heroin or polydrug dependence & harmful use	–	7	3	–	10	–	5	3	–	7	17
Other drug dependence & harmful use	–	4	1	–	5	–	1	–	–	1	6
2. Schizophrenia	–	–	–	–	–	–	–	–	–	–	–
3. Anxiety & depression	–	–	–	–	–	–	–	–	–	–	–
Other mental disorders	–	–	–	1	1	–	–	–	1	1	2
K. Nervous system and sense organ disorders	5	8	16	16	46	4	6	6	26	41	86
1. Dementia	1	–	–	7	8	–	–	–	20	20	28
2. Epilepsy	2	4	12	3	21	1	1	3	1	5	26
3. Parkinson's disease	–	–	–	3	3	–	–	–	2	2	4
8. Sense organ disorders	–	–	–	–	–	–	–	–	–	–	–
d. Adult-onset hearing loss	–	–	–	–	–	–	–	–	–	–	–
Vision loss	–	–	–	–	–	–	–	–	–	–	–
Other nervous system and sense organ disorders	3	4	4	4	14	3	5	3	4	13	28
L. Cardiovascular disease	4	31	159	268	462	3	20	89	256	368	831
1. Rheumatic heart disease	1	3	4	2	10	–	6	8	7	21	31
2. Ischaemic heart disease	–	18	118	179	314	–	5	52	156	213	528
3. Stroke	–	3	14	54	71	1	2	14	59	77	149
4. Inflammatory heart disease	3	5	13	9	29	1	1	7	5	14	43
5. Hypertensive heart disease	–	–	1	8	9	–	–	2	8	10	19
Other cardiovascular disease	–	3	10	17	29	–	5	6	21	33	62
M. Chronic respiratory disease	2	5	21	79	106	1	3	21	61	85	191
1. Chronic obstructive pulmonary disease	–	–	8	63	71	–	–	11	45	57	128
2. Asthma	–	1	3	3	7	1	1	4	5	10	18

(continued)

Annex table 8 (continued): Deaths by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
Other chronic respiratory diseases	1	3	10	13	27	–	1	5	11	18	46
N. Diseases of the digestive system	–	5	11	21	37	–	2	9	16	27	64
1. Peptic ulcer disease	–	–	2	3	5	–	–	1	2	3	9
2. Cirrhosis of the liver[(c)]	–	–	2	1	3	–	–	1	1	2	5
Other digestive system diseases	–	4	7	17	29	–	2	6	13	22	51
O. Genitourinary diseases	–	2	5	18	25	1	1	6	30	39	64
1. Nephritis & nephrosis[(d)]	–	2	5	13	20	1	–	5	18	25	45
Other genitourinary diseases	–	–	–	5	5	–	1	1	12	14	19
P. Skin diseases	–	–	–	1	2	–	–	2	2	4	6
Q. Musculoskeletal diseases	–	–	–	4	5	–	2	2	4	8	12
1. Rheumatoid arthritis	–	–	–	–	–	–	–	–	1	1	2
2. Osteoarthritis	–	–	–	1	1	–	–	–	–	–	1
3. Back pain/slipped disc	–	–	–	–	–	–	–	–	–	–	–
Other musculoskeletal diseases	–	–	–	3	3	–	2	1	3	6	9
R. Congenital anomalies	20	4	2	1	26	17	2	1	1	20	46
S. Oral conditions	–	–	–	–	–	–	–	–	–	–	–
1. Dental caries	–	–	–	–	–	–	–	–	–	–	–
Other oral conditions	–	–	–	–	–	–	–	–	–	–	–
Z. Ill-defined conditions	14	–	–	–	14	11	–	–	2	13	27
1. Sudden infant death syndrome	14	–	–	–	14	11	–	–	–	11	25
Other ill-defined conditions	–	–	–	–	–	–	–	–	2	2	2
III. Injuries	**22**	**159**	**82**	**22**	**286**	**15**	**67**	**32**	**18**	**132**	**418**
T. Unintentional injuries	19	69	46	17	150	11	32	22	17	82	232
1. Road traffic accidents	6	38	22	5	72	4	20	12	3	39	111
2. Other transport accidents	3	3	4	1	11	–	1	1	–	2	13
3. Poisoning	–	7	3	2	11	–	6	3	2	11	22
4. Falls	1	2	3	5	11	–	–	2	6	7	18
5. Fires, burns and scalds	1	1	2	1	5	–	1	1	1	4	9
6. Drowning	2	4	5	–	12	2	1	1	–	5	16

(continued)

Annex table 8 (continued): Deaths by age, sex and cause, Indigenous Australians, 2003

	Males					Females					Persons
Cause	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**0–14**	**15–34**	**35–54**	**55+**	**All ages**	**All ages**
Other unintentional injuries	5	14	7	2	28	4	3	2	5	14	42
U. Intentional injuries	4	91	37	5	136	4	35	10	1	50	186
1. Suicide and self-inflicted injuries	1	73	25	5	104	3	20	6	–	29	133
2. Homicide & violence	3	17	11	–	32	1	15	4	1	21	53
Alternative burden of disease categories											
All intellectual disability	50	4	13	3	70	35	1	3	1	39	109
All vision loss	–	–	–	3	3	–	–	–	17	17	20
All renal failure	1	13	45	70	129	1	7	40	103	151	281

(a) Liver cancer (excluding hepatitis B- and C-related)
(b) Alcohol dependence & harmful use (including alcoholic cirrhosis)
(c) Cirrhosis of the liver (excluding alcoholic and hepatic cirrhosis)
(d) Nephritis & nephrosis (excluding diabetic-, congenital- & poisoning-related renal failure)
– nil or rounded down to zero
Note: Due to the mortality data adjustment processes (see Chapter 2 and Appendix A for more details), an entry of a death in the above table does not necessarily represent one actual death from that particular cause or age group in 2003.

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The Burden of Disease and Injury in Aboriginal and Torres Strait Islander peoples 2003 provides the first comprehensive assessment of the burden of disease of Indigenous Australians.

Burden of disease analysis gives a unique perspective on health. Fatal and non-fatal outcomes are combined, but can be examined separately as well. This report provides details of the extent of premature mortality and disability estimated for over 170 disease and injury categories and for Indigenous people living in remote and non-remote areas of Australia. It also presents estimates of the amount of disease and injury caused by 11 major risk factors. More importantly, it measures the *Indigenous Health Gap*, which is the difference between the observed burden of disease in Indigenous Australians and what it would have been if the same rates of burden of disease as in the total Australian population would have applied. This is of major policy interest. The diseases and risk factors that contribute most to the *Indigenous Health Gap* are identified as health areas where appropriately resourced health services, combined with interventions to address the social and economic disadvantages faced by Indigenous Australians, are likely to have the greatest impact on reducing the burden of disease in Aboriginal and Torres Strait Islander peoples.