Australian Government
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Australian Burden of Disease Study 2011

Methods and supplementary material

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Authoritative information and statistics to promote better health and wellbeing

AUSTRALIAN BURDEN OF DISEASE STUDY SERIES
Number 5

Australian Burden of Disease Study 2011

Methods and supplementary material

Australian Institute of Health and Welfare
Canberra
Cat. no. BOD 6

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This publication is part of the Australian Institute of Health and Welfare's Australian Burden of Disease series. A complete list of the Institute's publications is available from the Institute's website <www.aihw.gov.au>.

ISSN 2204-4108 (PDF)
ISSN 2006-4508 (Print)
ISBN 978-1-76054-002-9 (PDF)
ISBN 978-1-76054-003-6 (Print)

Suggested citation
Australian Institute of Health and Welfare 2016. Australian Burden of Disease 2011: methods and supplementary material. Australian Burden of Disease Study series no. 5. Cat. no. BOD 6. Canberra: AIHW.

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Published by the Australian Institute of Health and Welfare

This publication is printed in accordance with ISO 14001 (Environmental Management Systems) and ISO 9001 (Quality Management Systems). The paper is sourced from sustainably managed certified forests.

Please note that there is the potential for minor revisions of data in this report. Please check the online version at <www.aihw.gov.au> for any amendments.

Contents

Acknowledgments

The Australian Burden of Disease Study 2011 was undertaken by members of the Australian Burden of Disease Unit and the Indigenous Modelling and Research Unit of the Australian Institute of Health and Welfare (AIHW) under the guidance of Lynelle Moon (lead analyst) and Michelle Gourley (lead analyst, Indigenous), and project management of Miriam Lum On.

The Australian Burden of Disease Expert Advisory Group provided advice on overarching methods. All methods pertaining to Indigenous analyses were overseen by the Australian Burden of Disease Indigenous Reference Group. Project oversight was provided by the Australian Burden of Disease Project Governance Committee. Membership of these groups is provided in Appendix G.

Ken Tallis and Anthony Barnes provided insightful methodological advice and wisdom throughout the project, which is gratefully acknowledged.

Methods development and implementation for specific areas of analysis were overseen by Karen Bishop (mortality), Melissa Goodwin (morbidity), Vanessa Prescott (risk factor analysis) and Jessica Zhang (Indigenous mortality).

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- Julie Ayre (mental and substance use disorders, oral disorders)
- Karen Bishop (injuries)
- Ilona Brockway (respiratory diseases)
- Melanie Dunford (blood and metabolic disorders, hearing and vision disorders, reproductive and maternal conditions)
- Julianne Garcia (neurological conditions, cardiovascular diseases, endocrine disorders)
- Melissa Goodwin (cancer and other neoplasms, gastrointestinal disorders)
- Michelle Gourley (mental and substance use disorders)
- Wendy Ho (hearing and vision disorders, infant and congenital conditions)
- Miriam Lum On (infant and congenital conditions)
- Nick Mann (musculoskeletal conditions)
- Michael McGrath (infectious diseases)
- Lynelle Moon (cardiovascular diseases, endocrine disorders, kidney and urinary diseases)
- Vanessa Prescott (kidney and urinary diseases)
- Nancy Stace-Winkles (skin disorders).

Vanessa Prescott and Michael McGrath developed and analysed individual risk factor methods, in consultation with experts.

Systems development was undertaken by Nick Mann and Melissa Goodwin.

Fadwa Al-Yaman, Tracy Dixon, Lisa McGlynn, Louise York and David Whitelaw from the AIHW provided constructive methodological advice, while Jennifer Kerrigan,

Simon Margrie, Laura Pritchard, Naila Rahman, Anna Reynolds, Marianna Stylianou and Jeanine Wilson (also of AIHW) gave additional analytical assistance.

Methods specific to diseases and risk factors were reviewed by expert panels comprised of relevant clinical and epidemiological experts. A full list of contributors to disease and risk factor work is provided in Appendix G.

The Australian Burden of Disease Study 2011 was funded by the Australian Government Department of Health and the former Australian National Preventive Health Agency.

This report was peer reviewed by Department of Health, Western Australia.

Abbreviations

ABDS	Australian Burden of Disease Study
ABS	Australian Bureau of Statistics
ACD	Australian Cancer Database
AATSIHS	Australian Aboriginal Torres Strait Islander Health Survey
ACHI	Australian Classification of Health Interventions
AHS	Australian Health Survey
AIHW	Australian Institute of Health and Welfare
ASGS	Australian Statistical Geography Standard
AusDiab	Australian Diabetes, Obesity and Lifestyle Study
BEACH	Bettering the Evaluation and Care of Health
BOLD	Burden of Obstructive Lung Disease
CI	confidence interval
CIMHA	Consumer Integrated Mental Health Application
COPD	chronic obstructive pulmonary disease
DALY	disability-adjusted life years
FASD	fetal alcohol spectrum disorders
GBD	Global Burden of Disease
GMFCS	Gross Motor Function Classification System
GP	general practitioner
Hib	Haemophilus influenzae type b
HIV	human immunodeficiency virus
HIV/AIDS	human immunodeficiency virus/acquired immune deficiency syndrome
ICD	International Statistical Classification of Diseases and Related Health Problems
ICD-10	International Statistical Classification of Diseases and Related Health Problems, 10th Revision
ICD-10-AM	International Statistical Classification of Diseases and Related Health Problems, 10th revision, Australian modification
IDEA	Intellectual Disability Exploring Answers

IQ	intelligence quotient
MCOD	multiple causes of death
MET	metabolic equivalent of tasks
mmol	millimole
NATSIHS	National Aboriginal and Torres Strait Islander Health Survey
NATSISS	National Aboriginal and Torres Strait Islander Social Survey
NDSHS	National Drug Strategy Household Survey
NHMD	National Hospital Morbidity Database
NHS	National Health Survey
NMD	National Mortality Database
NMSC	non-melanoma skin cancer
NNDSS	National Notifiable Diseases Surveillance System
NNAPEDCD	National Non-admitted Patient Emergency Department Care Database
NZBDS	New Zealand Burden of Disease Study
PAF	population attributable fraction
PM	particulate matter
RSE	relative standard error
SEIFA	Socio-Economic Indexes for Areas
TMRED	Theoretical minimum risk exposure distribution
WARDA	Western Australian Registry of Developmental Anomalies
WHO	World Health Organization
YLD	years lived with disability
YLL	years of life lost

Symbols

–	nil or rounded to zero
. .	not applicable
n.a.	not available

1 Introduction

Burden of disease analysis is a standard method for collating data of acceptable quality on causes of health loss, to produce comparable and concise policy-relevant evidence. Being able to use data from various sources to develop an internally consistent measure for all diseases is a key strength of a burden of disease study. However, methods used in burden of disease studies have become more complex over time, and the number of diseases and risk factors specificially analysed has increased. This increased complexity makes it much harder to explain the methods, and can result in decreased clarity for stakeholders.

One of the central principles for the Australian Burden of Disease Study (ABDS) 2011 is transparency of data, assumptions and methods. This report describes, as far as practicable, the methods and assumptions used by the ABDS 2011 to quantify the fatal and non-fatal effects and causes of diseases and injuries in the Australian and Aboriginal and Torres Strait Islander populations in 2011 and 2003. It is a companion publication to *Impact and causes of illness and death in Australia 2011* (AIHW 2016b) and *Impact and causes of illness and death in Aboriginal and Torres Strait Islander people 2011* (AIHW 2016c).

The report is divided into 3 main sections.

The first presents the general methods used to estimate disease burden, followed by disease-specific methods.

The second presents the general methods used to estimate the burden attributable to various risk factors, followed by more specific methods for each risk factor.

The third presents methods used to account for quality and accuracy.

Where they differ from the national methods, methods used to estimate the burden in the Aboriginal and Torres Strait Islander population, subgroups (state/territory, remoteness and socioeconomic group) and in 2003 are explained as necessary within each section.

To make the report easier to read, large tables and additional information are presented in appendixes A to F.

Key considerations

The ABDS 2011 methods build on the methodological approach of the Global Burden of Disease (GBD) 2010 and 2013. Detail on key considerations of the methods used in GBD 2010 and their applicability to the Australian context have been previously published in *Assessment of Global Burden of Disease 2010 methods for the Australian context* (AIHW 2014b).

Key considerations in the development of the methods for ABDS 2011 were the need for:

- national estimates which were relevant to Australia, while maintaining comparability with global methods as much as possible
- Indigenous estimates, including measures of the gap in disease burden between the Indigenous and non-Indigenous populations
- subnational estimates (state/territory, remoteness and socioeconomic group)
- comparability to 2003 estimates to enable valid comparisons over time.

Expert advice and review

An Expert Advisory Group provided oversight and detailed advice on key technical issues, including the overall methods and inputs (Appendix Table G2) throughout the ABDS 2011.

An Indigenous Reference Group provided advice on methods relevant to the Aboriginal and Torres Strait Islander population (Appendix Table G3).

Methods specific to diseases and risk factors were reviewed by expert panels comprised of relevant clinical and epidemiological experts (Appendix tables G4 and G5).

Ken Tallis provided overall methodological advice and review, while Tony Barnes provided advice on Indigenous methods.

Section I: Estimating the disease burden

This section describes the methods used in the ABDS 2011 to quantify the burden due to all diseases, conditions and injuries.

- Chapter 2 explains some key methodological choices made during the study.
- Chapters 3 and 4 provide an overview of the methods used to estimate fatal burden and non-fatal burden.
- Chapter 5 provides greater detail of the specific methods used to estimate mortality and prevalence for each disease group.

Key terms used in this section

age weighting: A method that is sometimes used to adjust the relative 'value' of years lived at different ages—for example, to value a year lived by a young adult more highly than a year lived at older ages. If applied, age weighting results in some age groups having an increased influence on the estimates of disease burden relative to other age groups.

disability-adjusted life year (DALY): A year of healthy life lost, either through premature death or living with disability due to illness or injury.

disability weight: A factor that reflects the severity of health loss from a condition on a scale from 0 (perfect health) to 1 (equivalent to death).

discounting: A method that is sometimes used to adjust the relative 'value' of years lived (or lost) in the future. It is based on the assumption that a year lived in the future is of less 'value' than a year lived now. Discounting for future benefits is standard practice in some economic analyses.

reference life table: A table that shows, for each age, the number of remaining years a person could potentially live—used as a measure of the years of life lost from dying at that age.

sequelae: Health consequences of diseases and injuries, such as anaemia due to chronic kidney disease.

years lived with disability (YLD): Measures the years of what could have been a healthy life that were instead spent in states of less than full health. YLD represents non-fatal burden.

years of life lost (YLL): Measures years of life lost due to premature death. YLL represents fatal burden.

2 Overarching methods and choices

The ABDS 2011 measured health loss using a summary measure of health called the disability-adjusted life year (DALY). One DALY represents 1 lost year of 'healthy life' due to premature death, illness or disability, or a combination of these factors. This measure quantifies the gap between a population's actual health and an ideal level of health in the given year—that is, every individual living in full health for his or her ideal or potential life span—and includes both fatal and non-fatal components.

A broad overview of the process for estimating DALY is shown in Figure 2.1.

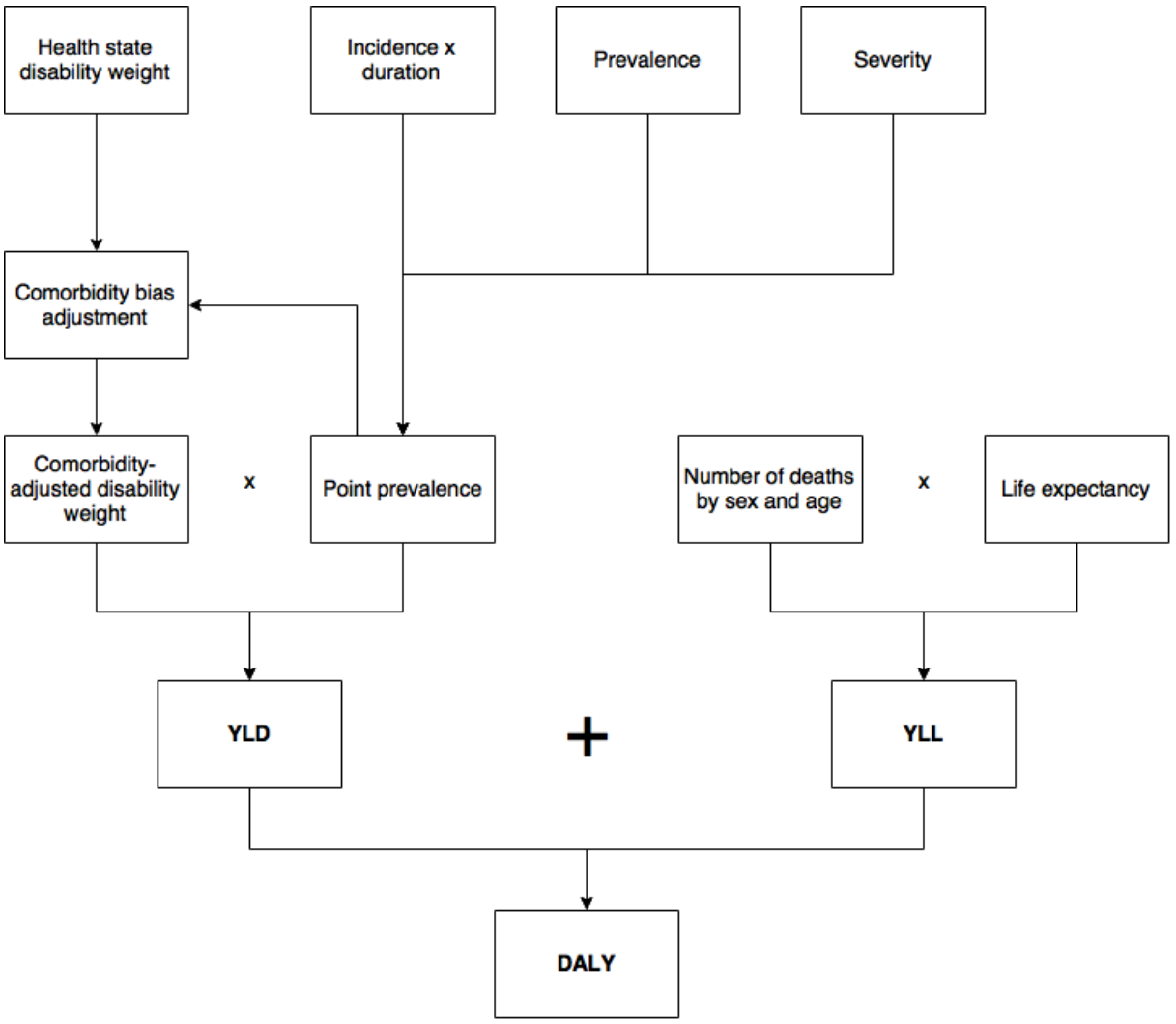

Figure 2.1: Overview of disability-adjusted life year estimation process

The fatal component is measured using years of life lost (YLL)—1 YLL represents 1 year of life lost (due to premature death). YLL measures the years lost between the age at which a person dies and an ideal life span according to a reference life table. Total YLL are influenced by both the total number of deaths, and the ages at which those deaths occur.

In the ABDS 2011, the ideal remaining expectancy varied at each age, but started with a life expectancy at birth of 86.0 years for both males and females. This ideal life span was drawn from the Global Burden of Disease (GBD) 2010 reference life table, and was based on the lowest observed death rates at each age group from multiple countries (Murray et al. 2012).

See Chapter 3 for more detail on YLL estimation.

The non-fatal component is measured using years lived with disability (YLD)—1 YLD represents 1 year of life lost (due to ill health or disability). YLD measures the number of healthy years of life lost due to disease in the reference year. This is calculated by estimating the amount of person-time spent with a condition, multiplied by a disability weight, indicating the severity of the health loss associated with the condition. Total YLD are influenced by the number of people with each disease, the time spent in less than full health, and the disability weights defined for each disease. The disability weights used in this study were drawn from the GBD 2013 (see GBD 2013 Collaborators 2015b), and represented the health loss caused by the consequences of each disease.

See Chapter 4 for further detail on YLD estimation and use of disability weights.

As they use time as a common currency, the YLL and YLD can be summed to measure DALYs: 1 DALY represents the loss of 1 year of healthy life.

$$DALY = YLL + YLD$$

When DALY are used to measure the burden of disease in a population in a time interval, they can be calculated in various ways: from an incidence, prevalence, or hybrid perspective. Each method produces a measurement of a different quantity. This study used the hybrid perspective for calculating DALY consistent with recent global studies. This calculates YLL from an incidence perspective (see Chapter 3 for details) and YLD from a prevalence perspective (see Chapter 4). The main advantage of this approach is that all data needed to calculate DALY can be measured in the period in question (whereas incidence-based DALY require a projection of the future duration of health loss, and prevalence DALY require knowledge of deaths that occurred before the period in question).

Constructed this way, DALYs can be thought of as an index of population health in a given year, providing a summary measure of the overall population health for the year being reported. This enables diseases, population groups and points in time to be compared.

No age weighting or discounting

Consistent with the methodological approach used by GBD 2010, no age-weighting (assigning larger relative value or quantitative influence to certain age groups compared with others) or discounting (valuing healthy years lived in the present more than those lived in the future) was applied in ABDS 2011. This differs to previous Australian burden of disease studies. Further information about the rationale behind this decision is in *Assessment of Global Burden of Disease 2010 methods for the Australian context* (AIHW 2014b).

Reference years 2011 and 2003

Based on the availability of data at the start of the study, 2011 was considered the most suitable choice for the primary reference year. It should be noted that some data used in the ABDS (mainly from surveys or epidemiological studies) related to periods earlier than 2011 as this was when the most recent survey or the most relevant epidemiological study was done. In such cases, modelling was required to adjust the counts or rates to 2011.

Although 2011 was used as the reference year of the study, more than 1 year of data was compiled and analysed in some cases to overcome small numbers or to smooth variability. For some estimations, it was also informative to look at trends over time.

Australian and Indigenous estimates for 2003 were originally presented in separate publications by Begg et al. (2007) and Vos et al. (2007). As the ABDS 2011 methods differed considerably from these studies, revision of 2003 estimates was required to provide comparable Australian burden of disease estimates to assess changes over time, and also to reduce the risk of users making erroneous comparisons between these 2003 estimates and new 2011 estimates from ABDS.

Reference populations

All Australian population-based rates were calculated using populations rebased to the 2011 Census (released 20 June 2013). Rates for the Aboriginal and Torres Strait Islander population were also calculated using population estimates rebased to the 2011 Census for both 2003 and 2011 estimates (released April 2014). For more information on the choice of backcast population series for 2003 Indigenous estimates see 'Methodological choices specific to Indigenous estimates' in this chapter.

The Australian 2001 standard population (published 20 June 2013) was used for all age-standardisation, as per AIHW and ABS standards (ABS 2013a).

Age groups

Analysis was done using as fine an age disaggregation as was supported by the data. For fatal burden, YLL were calculated using single year of age. For non-fatal and total burden, construction of YLD (and hence DALY) estimates were based on 5-year age groups of 0, 1–4, 5–9, …, 100+ for the national component, and collapsed age groups for 0–4 and 85 and over for Indigenous estimates. Where the available data could not directly support 5-year age groups, modelling was used to derive estimates at the required level of age disaggregation.

The reporting age groups were aligned to fit with existing reporting practices by age and sex to enable comparisons with other data, within the constraints of the quality of the underlying data.

Selection and classification of diseases

The list of diseases and injuries (referred to as the ABDS disease list)—and their organisation into disease groups—forms the analytical framework of the ABDS 2011, and underpins all estimates of deaths, YLL, YLD, DALY and risk-attributable burden. As the burden of each disease is estimated relative to every other disease specified in the study, this list forms the foundation of all analysis and reporting.

The ABDS disease list used the following hierarchical framework:

Disease groups: 17 disease groups of related diseases or conditions, such as cardiovascular diseases, gastrointestinal disorders, or injuries

Diseases: 188 specific conditions or sets of conditions, such as coronary heart disease, appendicitis, or poisoning, for which estimates of deaths, YLL, YLD, DALY and risk-attributable burden were produced. These 188 conditions have been devised to be mutually exclusive (non-overlapping), and collectively exhaustive (covering the full spectrum of disease and injuries).

Selection of diseases and injuries

An Australian-specific disease list was developed for the ABDS 2011 to reflect the needs of health reporting and monitoring in Australia. As such, this list will differ from that used in other studies.

To be included in the ABDS 2011, a condition was required to satisfy one or more of the following criteria.

Included in other studies' disease (or cause) lists

- Have been included in:
  - the Global Burden of Disease (GBD) Study 2010 or
  - the 2003 Australian Burden of Disease Study (ABDS 2003) (Begg et al. 2007)

  unless its inclusion in ABDS 2011 conflicted with other criteria.

Significant burden

- Be of significant burden to at least 1 age group or sex—defined as either more than 25 deaths or more than 500 inpatient events averaged annually over 2008–2011, or as having a 'significant' primary care impact, as determined by expert judgment (ensuring the list is not cluttered with very minor conditions, for which it might be difficult or costly to assemble data).

Policy interest

- Be of substantial Australian or Indigenous health policy interest—defined as being the focus of current policy or professional attention, or thought to be increasing substantively in impact (which might be signalled by large increases in incidence or prevalence), or
- be the subject of an existing health monitoring activity within Australian or Indigenous populations, or
- be required for the analyses of risk factors that are of strong policy interest.

Be able to be measured

- High-quality, relevant and recent epidemiological data needed to be available for at least 2 out of these key epidemiological variables: incidence, prevalence, survival or mortality of/from the condition.

Using these criteria, a final list of 188 mutually exclusive diseases, conditions and injuries (including residual conditions—see 'Residual conditions') were selected and agreed on by the Australian Burden of Disease Expert Advisory Group to form the basis of the ABDS 2011. A further 12 conditions describing the nature of injury were also included for alternative

reporting (see 'Injuries' in Chapter 5). For the full list of diseases, conditions and injuries, see Appendix Table A2.

Residual conditions

The disease list is collectively exhaustive. Conditions that could not be individually specified are included in a residual category for each disease group. For example, the residual category 'other musculoskeletal conditions' are those musculoskeletal conditions not included in arthritis, gout, rheumatoid arthritis and back pain and problems. There are 30 residual categories distributed across the 17 disease groups. These residual diseases are listed in Appendix Table A2.

Conditions not included as specific diseases in the disease list

There were 3 key reasons for not including some conditions as specific diseases in the ABDS 2011 disease list:

- **Scarcity of recent and/or robust data to reliably estimate prevalence in Australia in 2011**—these conditions could be incorporated into future burden of disease analyses should more recent or robust data become available. Examples include:
  - myalgic encephalomyelitis/chronic fatigue syndrome—although believed to be of significant impact, this condition is not monitored in Australia and recent robust data on incidence and/or prevalence are scarce. Although this was included in the ABDS 2003 as a separate disease, the data underpinning these estimates are now outdated. Myalgic encephalomyelitis/chronic fatigue syndrome was not separately estimated in GBD 2010, GBD 2013 or the New Zealand Burden of Disease Study (NZBDS) 2006 (NZMOH 2013). In this study the burden of this condition is included in 'other neurological conditions'.
  - fetal alcohol spectrum disorders (FASD)—although FASD is of policy interest, no national data source was identified. FASD was not separately estimated in GBD 2010, but was separately estimated in the NZBDS based on hospitalisations (however, it was noted it would be an underestimate). In the ABDS 2011, the burden of FASD experienced by the child was grouped under the disease 'brain malformations' in infant and congenital conditions.
- **The condition is the result of other underlying causes, or its burden is captured under other sequelae**—these conditions do not fit within the mutually exclusive disease structure required for burden of disease analysis. Future analyses of these conditions might be possible by selecting corresponding diseases or sequelae. Examples include:
  - antimicrobial resistance—antimicrobial resistance includes many types of organisms (for example, staphylococcus) and types of resistance (for example, penicillin). Antimicrobial resistance was not included in previous burden of disease studies. Although it is of policy interest, and there are sufficient data for modelling, its outcomes were captured by other diseases already included in the study (for example, infectious diseases).
  - septicaemia—this is considered an intermediate, rather than underlying, cause of burden, and its impact was captured through the sequelae and the severity distributions for relevant diseases (for example, selected infectious, neonatal and maternal diseases).

- heart failure—this is also considered an intermediate cause of burden, and its impact was captured through the sequelae and the severity distributions for relevant diseases (for example, cardiovascular disease, congenital heart disease).

- **The condition was conceptualised as a risk factor**—these conditions might not have been associated with health loss themselves, but place individuals at greater risk of other health conditions. Their impact is captured as burden attributable to various risk factors. Examples include:
  - osteoporosis—the health loss from osteoporosis is captured under falls in the injury disease group. The risk factor low bone mineral density was used in this study to estimate the proportion of falls attributable to osteoporosis (see chapters 6 and 7)
  - nutritional deficiencies—in the ABDS 2011, protein-energy deficiency and iron-deficiency anaemia are included as specific nutritional deficiencies in the disease list. Other nutritional deficiencies (such as diet low in calcium) are not included as diseases, but instead as risk factors for other diseases (see Chapter 6).

Classification of diseases and injuries

To ensure that the disease list was both comprehensive and mutually exclusive, each included disease and injury had to be carefully defined. To ensure consistency between YLL and YLD estimation, the classification of each disease had to be suitable for both mortality and morbidity components.

As the internationally recognised and definitive set of codes to describe all health conditions, the International Classification of Diseases and Related Health Problems, 10th Revision (ICD-10) (2010 version) (WHO 2016) was used to broadly define each disease in the disease list. To estimate YLL, ICD-10 classifcations were used, but for YLD, classifications were adapted as necessary depending on the data that were available and appropriate for analysis (for example, the Australian modification ICD-10-AM was used for hospital separations data).

See Chapter 5 for details of the specific classifications used for each disease group.

Mapping of ICD-10 codes to the disease list

The allocation of more than 12,000 ICD-10 codes to the 200 diseases in the ABDS disease list was informed by the corresponding allocation of codes for GBD 2010, NZBDS 2006 and the ABDS 2003. Priority was given to GBD 2010 allocation, based on the project principle to align with GBD methods as much as possible, unless it conflicted with the requirements of Australian clinical or policy concerns.

To promote internal consistency and objectivity, the following principles were applied:

- **Attribute the burden to the condition where the health loss was experienced ('prevalence principle').** This principle was used mostly when mapping diseases or conditions that can be a long-term result of an earlier condition; diseases that are risk factors or sequelae for other diseases; or diseases that can be counted in more than one disease group**.** Examples include:
  - The burden from liver cancer or chronic liver disease due to hepatitis was counted where the condition manifested or was experienced (that is, in cancer or gastrointestinal conditions), not as a long-term sequelae of hepatitis. This was

consistent with GBD 2010 and with the mapping practice for other conditions that are now known to be the result of previous infectious diseases.

  - The overlap in cardiovascular disease, chronic kidney disease and diabetes was dealt with by attributing the health loss to the condition experienced, rather than the underlying cause (for example, renal complications due to diabetes mellitus was counted under chronic kidney disease). The AIHW is exploring the overlap between these diseases to quantify their indirect impacts and collective burden. Results from these studies will be available in forthcoming reports from the AIHW to be released in late 2016.

- **Classify diseases according to Australian disease monitoring activities**. Australian disease monitoring classifications were given priority over GBD to provide better information for Australian health priority setting. For example, GBD classified all neoplasms together, regardless of malignancy. In Australia, monitoring of neoplasms is restricted to malignant neoplasms, so they were classified separately to other neoplasms.

The proposed mappings of ICD-10 codes to diseases in the ABDS disease list were reviewed by disease specific expert groups before being finalised.

Assigning diseases to disease groups

Under the ABDS disease hierarchy, each disease is allocated to a single disease group. The allocation of particular diseases to a disease group affects the estimates of burden and ranking by disease group that are reported in the published analyses. Alternative disease group presentations of ABDS 2011 results can be readily developed from the existing disease list. For example, gastrointestinal disorders do not include gastrointestinal infections, or gastrointestinal cancers, but the estimates for these diseases could be added to the gastrointestinal disorders group to obtain a broader picture of the burden for this area of interest.

For the most part, assigning diseases to disease groups relied heavily on the chapter structure of ICD-10. However, for a small number of diseases it was less straightforward, as they appeared potentially to bear some characteristics of more than one group. These diseases were allocated after discussion with experts from both potential disease groups, and, as with the prevalence principle, assigned according to where the health loss is actually experienced.

Major decisions referred to experts for advice included:

- **suicide and self-inflicted injuries**—the burden was included under injuries, consistent with ICD-10 coding and previous national and global burden of disease studies
- **accidental poisonings involving drugs and alcohol (ICD-10 codes X41, 42 and 45)**—the burden was included under injuries rather than substance use disorders, consistent with coronial assessment, on the basis that where the coroner found evidence of an underlying dependence, the cause of death would reflect this and be assigned to substance use disorders. The drug and alcohol experts expressed concerns about the reliability of distinctions between opioid overdose fatalities that are due to accidental overdose or those due to opioid dependence. There is evidence in Australian studies that most overdose deaths occur among people with a history of dependence, and very few deaths are deliberate. However, as the coding for X42 (Accidental poisoning by and exposure to narcotics and psychodysleptics [hallucinogens], not elsewhere classified)

includes several drugs, not just opioids, this assumption would have to be made for those other drugs as well

- **gestational diabetes**—the burden was counted in the reproductive and maternal disease group, rather than endocrine disorders, due to this condition only arising during pregnancy, and is consistent with previous national and global burden of disease studies
- **cerebral palsy**—the burden was allocated to the infant and congenital conditions disease group, rather than neurological conditions, as, in most cases, cerebral palsy is acquired in the prenatal and perinatal period and emerges as a leading cause of death for children aged less than 5. As a sequela, cerebral palsy is acquired through several other infant and congenital conditions, such as birth trauma and birth asphyxia
- **fetal alcohol spectrum disorders (FASD)**—although counted under mental health and substance use disorders in GBD 2010, the burden was assigned to infant and congenital conditions in ABDS 2011 as the main sequelae are learning difficulties and disfigurement, and the burden is experienced by the child (not the mother)
- **postnatal depression**—the burden was not included as a separate disease in ABDS 2011 due to data limitations. As available data did not distinguish whether the depressive disorder was associated with childbirth, postnatal depression was included in estimates for depressive disorders, within the mental and substance use disorders disease group. This is consistent with previous national and global burden of disease studies.

Selection and assessment of data sources

All potential data sources to estimate disease burden (whether published or unpublished) were assessed for comparability, relevance, representativeness, currency, accuracy, validation, credibility and accessibility/timeliness (see Appendix A for the criteria used to guide data selection). Only data sources that met the criteria were included in the study.

Potential data sources were required to: have case definitions appropriate to the disease or risk factor being analysed; be relevant to the Australian population; and be timely, accurate, reliable and credible. Where possible, national data sources, rather than sources relating to particular regions or subpopulations, were used.

Administrative data sources (for example, disease registers, hospitalisations) were evaluated for their level of ascertainment (how well the data correspond to the disease or sequela in question) and coverage (the proportion of the population included in the data).

Surveys were evaluated for their representativeness, potential selection bias, and measurement bias (validity and reliability of measurement).

Epidemiological studies were evaluated for the quality of their study design, their timeliness, credibility, representativeness, and sources of bias or error.

The key data source used in estimating mortality is described in Chapter 3, and key data sources used in estimating morbidity are listed in Chapter 4.

Methodological choices specific to Indigenous estimates

Additional factors needed to be considered when calculating burden of disease estimates for Aboriginal and Torres Strait Islander people. As a general principle in the ABDS, the methods used to produce Indigenous burden of disease estimates were consistent with those used to produce national estimates. For example, the same reference life table, disability weights and disease list were used. However, it was not always possible to adopt completely consistent methods due to differences in data availability, data quality and population size and characteristics.

Indigenous under-identification

While in recent decades major improvements have been made to the quality and availability of information about Indigenous Australians, existing data are subject to several limitations regarding data quality and availability. These include under-identification of Indigenous Australians in administrative data sets (and changes in people's inclination to identify as Indigenous over time), and lack of available data on the prevalence of certain diseases in the Indigenous population. Methods employed to address these issues in the ABDS are discussed in the relevant chapters of this report on fatal and non-fatal burden (chapters 3 and 4).

Dealing with small numbers

An important consideration for Indigenous burden of disease is the robustness and reliability of estimates produced, and the level of disaggregation supported by the data, given the small size of the Indigenous population compared with the much larger non-Indigenous population.

To ensure validity of the results, the AIHW combined several years of data and/or age groups as necessary to produce Indigenous estimates. Additionally, the level of disaggregation used to report Indigenous estimates was broader than that reported for the total Australian population. This included collapsed age groups for those aged 0–4 and 85 and over. However, where numbers supported the use of more detailed age groups (such as figures presenting total YLL rather than YLL by disease group), Indigenous YLL estimates were reported using the same age groups as reported for the total Australian population.

Measuring the gap between Indigenous and non-Indigenous Australians

Direct age-standardisation was used to compare rates between Indigenous and non-Indigenous Australians, and to measure the gap in burden between the 2 populations. The direct method was chosen, following a series of sensitivity analyses undertaken by the AIHW, which looked at the impact and robustness of using the direct method compared with the indirect method on resulting Indigenous YLL estimates (see AIHW 2015b for more information). The direct method enables multiple comparisons (for example, disease by sex) and can be used for comparisons over time. A limitation of the direct method is that less reliable estimates can be produced when it is applied to a small number of deaths and prevalent cases; this should be kept in mind when interpreting gap results for less common diseases and conditions.

Age-standardised rate differences and rate ratios were reported as measures of the gap. Rate differences provide a measure of the absolute gap between 2 populations, while rate ratios are a measure of the relative gap between 2 populations.

For the most accurate estimate of the gap in disease burden between Indigenous and non-Indigenous Australians, comparisons have been made to estimates calculated for the non-Indigenous population. Estimates for the total Australian population should not be compared with Indigenous population.

Choice of population denominator for 2003 Indigenous estimates

In estimating the Aboriginal and Torres Strait Islander population for the years prior to each Census, a number of assumptions are made regarding past mortality rates, migration, improvements in life expectancy, and changes in Indigenous identification. As such several population backcast and projection series have been produced in addition to the Estimated Resident Population for each Census year.

Following sensitivity analyses by the AIHW to look at the impact of using different Indigenous population denominators in 2003 burden of disease rate calculations, it was agreed to use the backcast population series based on the 2011 Census, which applies the Indigenous identification level in 2011 to earlier years. Using this backcast population for the 2003 estimates provides consistency between the denominators used for the 2003 and 2011 Indigenous burden of disease estimates in the ABDS 2011.

For more information on these choices, see *Impact and causes of illness and death in Aboriginal and Torres Strait Islander people 2011* (AIHW 2016c).

Methodological choices specific to subnational estimates

Subnational estimates include state/territory, remoteness categories and socioeconomic groups. These are defined as:

- **state and territory classifications**—the 8 Australian jurisdictions: New South Wales, Victoria, Queensland, South Australia, Western Australia, Tasmania, Northern Territory and the Australian Capital Territory. Disaggregation by state/territory is well supported by the data, with the majority of data sources (except for epidemiological studies and small surveys) defining and reporting state or territory in a standard way
- **remoteness categories**—based on the 2011 Australian Statistical Geographic Standard, which is divided into 5 remoteness areas: *Major cities*, *Inner regional*, *Outer regional*, *Remote* and *Very remote*. Remoteness areas aggregate to states and territories and cover the whole of Australia. Most major data sources, except for epidemiological studies and small surveys, were able to be broken down by remoteness area
- **socioeconomic groups**—presented as quintiles of lowest to highest socioeconomic position. Ideally, it would be better if detailed individual-level measures of socioeconomic characteristics were available in key data sources. But the most consistently available approach across the national data sources was the geographically-based proxy of socioeconomic group based on the relative socioeconomic characteristics of the area of residence, known as SEIFA (Socio-Economic Indexes for Areas). SEIFA is a measure of socioeconomic disadvantage developed by the ABS that ranks geographic areas in Australia according to relative socioeconomic advantage and disadvantage. The

ABS broadly defines relative socioeconomic advantage and disadvantage in terms of 'people's access to material and social resources and their ability to participate in society'. AIHW generally reports analyses of socioeconomic differences using SEIFA divided into population-based quintiles. It is also the standard for the majority of national agreement indicators. This approach ensures that, regardless of the underlying geographical unit, about 20% of the population is allocated to each quintile. SEIFA contains 4 indexes, with the Index of Relative Socioeconomic Disadvantage historically being the most commonly used at AIHW for health-related analyses. For more information on SEIFA, go to <www.abs.gov.au/websitedbs/censushome.nsf/home/seifa?opendocument&navpos=260>. SEIFA was only used for disaggregation of national estimates. For disaggregation of Indigenous estimates, see Indigenous subnational estimates.

Subnational methodology

Subnational estimates were based on breaking down national estimates at a level of disaggregation (disease, sex and broad age group) supported by the underlying data, rather than being derived using separate data sources. This ensured that comparisons across each disaggregation were based on common data definitions, which is often not the case when subnational data sources are combined.

The preferred approach for subnational estimates was to derive subnational disaggregation directly from the primary data source using geographical identifiers. When this was not available, secondary data sources were used to identify health loss gradients between the subnational regions that could then be applied to the national data. Lastly, when neither of these approaches were possible, the national sex/age prevalence rates were applied to the population structure of the subnational unit. This assumed no difference in disease prevalence rates between subnational and national populations.

Specific details on the methods used for subnational estimates for mortality and morbidity are included in chapters 3–5.

Key considerations

The validity of subnational results is influenced by the availability and quality of data at the level of disaggregation, and by the population size in the various groups.

For state and territory estimates, analyses used the same age groups as the national analysis. For remoteness and socioeconomic group analyses, age groups were restricted to 5-year age groups 0–4, 5–9, …, 85+ to overcome limitations with data.

Indigenous subnational estimates

Indigenous subnational estimates were considered reliable to calculate and report at the disease group level, but not at the specific disease level. This was due to:

- limited availability of Indigenous data for individual diseases at the geographical levels of interest
- limited availability of Indigenous identification adjustment factors at subnational levels for relevant administrative data collections
- small numbers if Indigenous estimates were broken down at subnational levels.

Indigenous subnational estimates were considered adequate to report for 4 states and territories (New South Wales, Queensland, Western Australia, and the Northern Territory). Estimates were not calculated for Victoria, South Australia, Tasmania or the Australian Capital Territory due to small numbers of Indigenous deaths in these jurisdictions, and lack of suitable mortality adjustment factors (see Chapter 3).

Estimates for all 5 categories of remoteness were reported (*Major cities, Inner regional, Outer regional*, *Remote* and *Very remote*.

For Indigenous burden estimates by level of socioeconomic disadvantage, an Indigenous-specific index (the Indigenous Relative Socioeconomic Outcomes Index) (Biddle 2013) was used. This was considered to more accurately reflect levels of disadvantage in the Indigenous population than what SEIFA used for the national component. As such, the Indigenous estimates by socioeconomic disadvantage were not compared with national estimates by socioeconomic disadvantage.

Indigenous subnational estimates of YLL were calculated directly from mortality data (adjusted for Indigenous under-identification) using state/territory and remoteness specific adjustment factors.

Hospitalisation data (adjusted for under-identification), ABS health survey data (2012–13 Australian Aboriginal and Torres Strait Islander Health Survey), or population proportions (depending on the disease group) were used to break down the national-level Indigenous YLD into subnational categories. Hospitalisation data were used for 10 disease groups, and health survey data were used for 6 disease groups for state/territory and remoteness estimates. The subnational Indigenous population structure was used for one disease group (skin disorders). For estimates by socioeconomic group, hospitalisation data were used for all disease groups, as Statistical Area Level 2 data (required to calculate the Indigenous Relative Socioeconomic Outcomes Index) were available from this data collection.

The data sources used to break down Indigenous YLD into subnational categories can be found in Appendix Table A3. The proportions used to break down Indigenous YLD estimates for each disease group can be found in appendix tables A4 to A6.

State-level data were not generally used to build the national burden of disease estimates for the Indigenous population (that is, fatal burden estimates were calculated using national mortality adjustment factors, and non-fatal burden estimates were largely calculated using national prevalence estimates sourced from national data collections). As a result, Indigenous estimates reported at the national level are not subject to the same data quality issues as the state and territory estimates.

For more information on the methods used for Indigenous subnational estimates see AIHW 2016c.

Methodological choices specific to 2003 estimates

Comparable YLL, YLD, DALY and attributable burden estimates were produced for each disease for both the Australian and Indigenous populations. Subnational estimates for 2003 were not within the scope of this study.

As the 2003 estimates are point-in-time estimates, their comparison with the 2011 estimates does not constitute a time-series analysis. Several issues must be addressed before analysing and interpreting time trend data. A key issue is that 2 points in time can provide misleading

information about changes over time—assuming that there is a straight-line trend between these 2 points might mask variation that exists but is not measured in this analysis, and results must be interpreted with this in mind. In addition, interpretation of changes over time also needs to take into account other aspects, such as the impact of confounders over time related to the estimates, and changes in metadata between reference periods. Any major changes between the 2003 and 2011 data that have an impact on the interpretation are highlighted in the relevant chapters in this report.

2003 methodology

Where possible, the same (or comparable) primary data source was used for 2003 as for the 2011 estimates. If this was unavailable, secondary data sources were used to derive age- or sex-specific rate ratios that could be applied to national data. If these approaches were not possible, the 2011 age/sex prevalence rates for 2011 were applied to the population structure for 2003. This assumed no difference in disease prevalence rates between 2003 and 2011.

Specific details on methods for 2003 estimates for mortality, morbidity and risk factors are included in chapters 3–7.

Indigenous 2003 estimates

Issues relating to changing Indigenous identification over time and potential inconsistencies in identification in numerator data and population denominators have an impact on the comparability of Indigenous burden of disease rates over time. These issues also have implications on the choice of population denominator used for 2003 Indigenous burden of disease estimates.

Where possible, adjustments have been made to account for changes in Indigenous identification over time in the numerator data used for rate calculations of disease burden. For example, Indigenous deaths and hospitalisations for both 2003 and 2011 estimates were adjusted using factors based on identification levels relevant to these reference years.

The population denominator used for 2003 Indigenous burden of disease estimates was consistent in terms of Indigenous identification with that used for 2011 estimates, which is important for assessing rate changes over time. Indigenous population estimates based on the 2011 Census were used, which applies the Indigenous identification level in 2011 to earlier years in the series, including for 2003.

3 Estimating the fatal burden

Expressed as years of life lost (YLL), fatal burden is a measure of years lost due to premature death. Analysis of fatal burden takes into account all deaths that occur in a population during a specified period. The YLL estimates in ABDS 2011 were based on deaths that occurred in 2003 and 2011.

Deriving YLL requires both:

- mortality data—the actual number of deaths and the ages at which those deaths occurred
- a reference life table—a measure of the years of life lost at each age.

Key terms used in this chapter

redistribution: A method in a burden of disease study for reassigning deaths with an underlying cause of death that is not in the study's disease list. Typically, the deaths reassigned include: those with a cause that is implausible as an underlying cause of death; those that relate to an intermediate cause in the chain of events leading to death; or those for which there is insufficient detail to ascertain a specific cause of death.

reference life table: A table that shows, for each age, the number of remaining years a person could potentially live—used to measure the years of life lost from dying at that age.

years of life lost (YLL): Measures years of life lost due to premature death.

Overview of methods

YLL measures the impact of dying prematurely; that is, the fatal component of burden of disease. YLD (discussed in Chapter 4) represents the non-fatal component.

The first step to estimate YLL is to compile the total number of deaths by age and disease (cause of death). Key methodological decisions include:

- the most appropriate mortality data to use
- how to assign deaths in the data set to diseases in the study's disease list.

The process for this is described fully in 'Aligning causes of death to the ABDS disease list'.

YLL for each disease is then calculated at the disease-specific level (for each age). Using single year of age at death, each death is weighted according to the remaining or potential life expectancy at that age of death using the reference life table—this becomes the years of life lost. Key methodological decisions include:

- whether potential life expectancy should vary according to sex or the population being analysed
- which reference life table to use.

These weighted deaths are then summed, and the result is the total number of years of life lost from all deaths. This is described mathematically as:

$$\text{YLL} = \sum_{ai} D_{ai} \text{ x } W_a$$

where:

- $\sum_{ai}$ is the sum over all ages and diseases
- a is an index for age
- i is an index for disease
- D_{ai} is the number of deaths due to disease i at age a
- W_a is the weight for deaths at age a (in practice, the number of expected remaining years at that age, according to a reference life table).

The same broad methods were used to derive YLL estimates for Aboriginal and Torres Strait Islander people, states and territories, remoteness areas and socioeconomic groups. The rest of this chapter details these and other methodological decisions that underpin the ABDS 2011 estimates of fatal burden.

Mortality data

Data on the deaths that occurred in the reference year underpin any YLL calculation.

Australian deaths data are collected through a vital registrations system. This is a system collecting and maintaining records of life events—such as births, deaths and marriages—by a government authority. In Australia, this is done by the Registrars of Births, Deaths and Marriages in each state and territory.

Information on causes of deaths nationally is sourced from the Registrars of Births, Deaths and Marriages in each state and territory, and from the National Coronial Information System managed by the Victorian Department of Justice, coded to the International Classification of Disease (ICD) by the Australian Bureau of Statistics (ABS). The AIHW website < www.aihw.gov.au/deaths/about-deaths-data/> provides detailed information on the registration of deaths and coding of causes of death in Australia (AIHW 2016a). The completeness, accuracy and coding of these data are described elsewhere (AIHW 2016a, ABS 2015). The deaths data are collated by the ABS into an administrative data set for statistical analysis.

All deaths data used in ABDS 2011 were extracted from the AIHW's National Mortality Database (NMD). This is a register of all deaths in Australia since 1964, sourced from the cause of death unit record files created by the ABS as described previously. The database comprises information about the causes of death and other characteristics about the person, such as sex, age at death, Indigenous status and area of usual residence.

Australian mortality data are believed to be virtually complete*, so no adjustment needs to be done to account for missing death records. However, in other respects, cause of death data are not immediately ideal for the ABDS 2011 analysis. In particular:

- some adjustments were required to account for under-identification of Aboriginal and Torres Strait Islander people
- causes of death that did not directly align to the study's disease list needed to be reassigned to a disease in the list (see 'Redistribution of deaths').

The data set used for the analysis comprises different versions of mortality data. The versions used have the most up–to-date, and hence most accurate, cause of death information.

2011 mortality data

ABDS 2011 includes all deaths that occurred in 2011 that were captured in cause of death unit record files for 2011 through 2013 (corresponding to deaths registered during 2011 to 2013). These were the latest data available at the time of analysis. Using all files up to 2013 improved the accuracy of the fatal burden estimates in 2 ways:

- **The number of deaths is more accurate.** Sometimes a death registration is delayed—on average, between 4% and 6% of deaths that occur in a given year are not registered until a subsequent year—most of these in the following 2 years (ABS 2013b). By including cause of death unit record files to 2013, any deaths that occurred in 2011 but were not registered until 2012 or 2013, were included in the analysis. It is possible for some deaths that occurred in 2011 to have been registered after 2013, and these are not included. However, judging from previous years of data, the impact of any outstanding deaths registrations on burden estimates is likely to be very small.
- **The causes of death are more accurate.** As a result of new processes implemented by the ABS in recent years, causes of death are subject to a revisions process. When data for a given year are first released, the cause of death information is deemed preliminary. Over the following 24 months, these data undergo 2 revisions: the first results in a revised version of cause of death, and the second in the final version of the cause of death. The following should be noted regarding the revisions process:
  - only death records that are subject to coronial investigation undergo such revisions; doctor-certified causes of death are not affected by the revisions process
  - the only information affected by these revisions is the cause of death—no changes are made to the number of deaths or the year or the age/sex composition
  - as the data move from being preliminary to final, fewer deaths have an unspecified cause, mechanism or intent, so a greater proportion can be readily aligned to the ABDS disease list.

The ABDS 2011 used the final version of 2011 deaths that had been registered in 2011. These data have undergone both ABS rounds of revision, so incorporated the most specific cause of death that can be obtained. For deaths that occurred in 2011, but were not registered until

* The data quality statements underpinning the AIHW National Mortality Database can be found in the ABS's quality declaration summary for Deaths, Australia at <www.abs.gov.au/ausstats/abs%40.nsf/mf/3302.0> and Causes of death, Australia at <www.abs.gov.au/ausstats/abs%40.nsf/mf/3303.0>.

2012 and were open to coronial investigation, the data had undergone the first ABS round of revision (revised version). Deaths that occurred in 2011 but were not registered until 2013 were used in their preliminary version.

The process of revisions and the resulting versions of the cause of death unit record files are described in more detail elsewhere (ABS 2015).

2003 mortality data

For the 2003 reference year, the ABDS used all available cause of death unit record files from 2003 onwards to identify deaths that occurred in 2003.

Cause of death unit record files were subject to the new ABS revision process from 2007 onwards (although 2006 data were revised, the process was different). As no ABS revisions have been made for data before 2006, all such files (including the file for deaths that occurred in 2003) are considered final.

Indigenous mortality data

Indigenous mortality data were sourced from the NMD in the same way using records identified as Aboriginal, Torres Strait Islander or both.

Dealing with small numbers

The number of deaths due to any particular cause varies from year to year. Fluctuations are more noticeable for diseases that are less common, and the instability is yet more severe for Indigenous deaths.

To reduce the impact of random fluctuations, Indigenous YLL estimates were based on the annual average of 3 years of deaths data. For the 2003 reference year, deaths were averaged from deaths occurring in 2002, 2003 and 2004. For the 2011 reference year, deaths were averaged from 2010, 2011 and 2012.

Adjusting for Indigenous under-identification

Every year, a number of deaths of Aboriginal and Torres Strait Islander people are not identified as such when registered (ABS 2013b). This might arise from the non-reporting of a deceased person's Indigenous status on the death registration form, or from incorrect identification of a deceased person's Indigenous status (recording a person as non-Indigenous when they are Indigenous, and vice versa). The net effect is an under-identification of Aboriginal and Torres Strait Islander people in the deaths data. The degree of under-identification can vary by age, by state/territory, by remoteness area and over time. Such under-identification means the number of deaths recorded as Aboriginal and Torres Strait Islander is an underestimate of the true levels of mortality in that population, and, unless some adjustment is made, this would result in an underestimate of the fatal burden (YLL) for Indigenous Australians.

The AIHW and ABS have both assessed under-identification in mortality data using direct methods based on national data linkage studies. The ABS's Census Data Enhancement Indigenous Mortality Study (2011–12) linked Census records with death registration records (ABS 2013c). The AIHW's Enhanced Mortality Database project (2008–2010) linked registered deaths with Indigenous death records from administrative data sources, including residential aged care data, hospital data and perinatal data (AIHW 2012a). Both of these

studies produced mortality adjustment factors that can be used to adjust for Indigenous under-identification in Australian mortality data.

Sensitivity analyses by the AIHW looked at the impact of applying the ABS and AIHW adjustment factors on resulting Indigenous YLL estimates, and on measures of the resulting gap. The analyses suggest that, at the national level, the age patterns and disease rankings would remain consistent regardless of which set of adjustment factors were used (see AIHW 2015b for more detail).

Adjustment of 2010–2012 deaths

The ABDS 2011 used the official mortality adjustment factors from the ABS's Census Data Enhancement Indigenous Mortality Study (2011–12) to adjust Indigenous deaths in 2010–2012 (for 2011 YLL estimates) for under-identification in mortality data. This included applying national age-specific adjustment factors for Indigenous estimates at the national level and by level of socioeconomic disadvantage (for which adjusted deaths from all states and territories were included in the analyses).

The ABS study did not provide adjustment factors for all 5 remoteness areas. The ABDS 2011 has used adjustment factors from the AIHW's Enhanced Mortality Data Collection (2008–2010) when compiling Indigenous mortality estimates by remoteness (Appendix Table B1).

Adjustment of 2002–2004 deaths

The same mortality adjustment factors as described for 2011 estimates were applied in the calculation of Indigenous YLL estimates for the 2003 reference year (which used deaths from 2002–2004). The AIHW assessed this as the most suitable approach to produce 2003 estimates, following sensitivity analyses. This approach assumes no change between 2003 and 2011 in the overall quality or pattern of Indigenous identification when Indigenous deaths are recorded. This view was supported by results from phase 2 of the AIHW's Enhanced Mortality Data Collection, which analysed deaths data from 2001 through 2010.

Aligning causes of death to the ABDS disease list

Having first assembled the deaths that are to be counted when calculating YLL, the causes of those deaths must then be ascribed to diseases in the ABDS disease list (as described in Chapter 2).

For the mortality data used in ABDS 2011 analyses, underlying cause of death was recorded using the International Classification of Diseases and Related Health Problems, 10th Revision (ICD-10) (WHO 2016, ABS 2014e). The procedure for assigning ICD-10 coded deaths records to items in the ABDS disease list is set out in Chapter 2.

Some ICD codes could not be classified directly to a specific disease in the ABDS disease list. To include these deaths in the calculation of YLL, they were redistributed using methods described in 'Redistribution of deaths'.

It is important to note that the alignment of ICD-10 codes to diseases in the ABDS disease list might not be the same as alignment to the disease lists used in other burden of disease studies.In particular, a disease in the ABDS disease list might have the same label, but comprise different (or fewer or more) ICD-10 codes compared with other studies' disease lists. Appendix Table A2 provides a list of ICD-10 codes for each disease used for the estimates of fatal burden in the ABDS 2011, noting that some diseases include codes marked for redistribution.

Redistribution of deaths

Identifying deaths for redistribution

Some ICD-10 codes are not appropriate or valid causes of death for burden of disease analysis. Some examples are:

- causes considered implausible as the underlying cause of death (such as hypertension and paraplegia)
- intermediate causes that have a precipitating cause (such as septicaemia and pneumonitis)
- immediate causes that occur in the final stages of dying (such as cardiac arrest and respiratory failure)
- causes that are ill-defined or unspecified, such as ill-defined digestive diseases.

Despite their overall high quality, Australian deaths data are affected by these issues. To quantify their contribution to the fatal burden, deaths coded to these underlying causes must be reassigned to one or more of the diseases (target diseases) according to what could be a more probable underlying cause. This process, referred to as 'redistribution' ensures that all the deaths in the reference year, hence all years of life lost, are counted in calculating YLL and is undertaken using the methods described.

Redistribution groups

The ICD-10 codes identified for redistribution were firstly assigned to redistribution groups. Each group was redistributed as a whole to the same range of target diseases. For example, non-specific digestive cancers formed one redistribution group redistributed to digestive cancers only. All deaths assigned to a group were redistributed using the same algorithm.

The redistribution groups used in ABDS 2011 generally align with those used by GBD 2010 (Lozano et al. 2012). Appendix Table B2 shows the ABDS redistribution groups, target diseases and method for redistribution. The method by which each group was redistributed depended upon the level of available evidence.

Methods for redistribution

Deaths identified for redistribution were reassigned to one or more diseases in the disease list using statistical algorithms. A portion of each death identified for redistribution may be reassigned across multiple diseases.

The redistribution methods used in burden of disease studies have been refined over time, and algorithms have been developed and improved to redistribute deaths that bear inappropriate or invalid codes, by exploiting available evidence of a plausible alternate cause of death. ABDS 2011 has extended these methods using Australian-specific data and Australian-specific direct evidence.

Three methods were used for redistribution in the ABDS 2011:

- **Direct evidence:** This method uses direct evidence about particular deaths or causes of death—developed through data linkage studies or extracted from sources other than the National Mortality Database—to ascertain probabilities of a more plausible cause of death.

- **Indirect multiple causes of death (MCOD):** This method uses tabulations of the underlying cause of death where the cause to be redistributed is reported as an associated cause of death. The frequency distribution of the corresponding underlying causes of death informs the redistribution algorithm. For example, the algorithm for pneumonitis redistribution was provided by the frequency distribution of the underlying cause of death for all deaths that included pneumonitis as an associated cause of death. This method was used for frequently occurring causes of death, and where supported by the mortality data (for example, septicaemia, pneumonitis and hypertension).
- **Proportional redistribution:** This method assigns deaths across a specified range of target diseases according to patterns of causes of death observed in the mortality data set for the disease list. Target ranges can be prescribed (for example, by narrowing the range of target diseases to injuries only). This method has the advantage of being conceptually simple and easy to implement, but it is relatively blunt, as the patterns of causes observed in the mortality data set might not reflect which underlying causes of death are more or less probable for the particular redistribution cause under consideration.

Direct evidence was preferred where it was available, followed by indirect MCOD (or a combination of both). In ABDS 2011, 85% of redistribution was based on some form of empirical evidence. Proportional allocation was used only when neither of these methods could generate sufficient information to develop an algorithm; only a small proportion of redistributed deaths (15%) were redistributed using this method (Appendix Table B2).

Impact of redistribution

Disease-specific YLL are influenced by the causes of death identified for redistribution, and by the methods used to reassign these to another disease. Redistribution can have an impact on the number of deaths classified to a disease, as well as the number of YLL from that disease. In the ABDS 2011, 14,761 deaths were identified for redistribution, equating to 189,345 YLL. Overall, this amounted to 10% of deaths and 8% of YLL.

The number of deaths identified for redistribution varied with age (Appendix Table B3). They generally followed the patterns of causes exhibited for other cause of death tabulations for Australia. For example, unspecified cancer deaths largely reflect the age patterns for cancer deaths.

Appendix Table B4 shows the number of deaths classified to disease groups before and after redistribution. The largest numbers of deaths gained by redistribution were for:

- cardiovascular (5,021 more deaths, an increase of 12%)
- cancer (4,783 more deaths, an increase of 12%)
- injuries (1,388 more deaths, an increase of 17%).

As a proportion of deaths before redistribution, large gains were apparent for:

- kidney/urinary (717 more deaths, an increase of 23%)
- gastrointestinal (910 more deaths, an increase of 19%).

The impact of redistribution on YLL is also shown in Appendix Table B4. The largest number of YLL gained was for:

- cancers (73,106 more YLL, a 10% increase)
- cardiovascular (44,710 more YLL, a 9% increase)
- injuries (27,573 more YLL, a 10% increase).

As a proportion of YLL before redistribution, the largest gains were for:

- kidney/urinary (6,458 more YLL, a 20% increase)
- skin (721 more YLL, a 16% increase)
- gastrointestinal (10,048 more YLL, a 13% increase).

To illustrate the reasoning underlying the redistribution of deaths and its impact, Box 3.1 steps through the number and type of deaths that were redistributed into the cancer disease group for 2011 YLL estimates.

Box 3.1: How redistribution works

This box steps through the redistribution process, showing, as an example, where additional cancer deaths came from as a result of redistribution.

Appendix Table B4 shows 39,974 deaths were coded to a cancer in the ABDS disease list. After redistribution, there were 44,757 cancer deaths, reflecting a gain of 4,783 deaths, or an additional 12%.

Appendix Table B2 shows 2,442 deaths were coded to a non-specific type of cancer, and 1,247 deaths were coded to a non-specific digestive cancer. So, in total, 3,689 non-specific cancer deaths were identified for redistribution. The same table shows that these non-specific cancer deaths were reassigned to a specific cancer using the direct evidence method, and that the target diseases were all in the cancer disease group.

So far, 77% of the overall gain in cancer deaths (3,689 out of the overall 4,783) has come from deaths initially coded to (non-specific) cancer-related causes, which have been redistributed into (specific) cancer-related diseases in the ABDS disease list.

Appendix Table B2 also shows a further 1,289 deaths (initially coded to 'all other non-specific, intermediate and immediate causes') were identified for redistribution that would be reassigned using the proportional allocation method across the whole range of ABDS diseases. A proportion of those 1,289 deaths—consistent with the proportion of cancer deaths (identified pre-redistribution)—were reassigned to cancers specified in the ABDS disease list. As can be seen from Appendix Table B4, pre-redistribution, 27% of deaths were cancers, so about 27% of the 1,289 deaths (equivalent to around 351 deaths) were also redistributed to a specific cancer.

The foregoing redistribution steps account for around 84% of the overall gain in cancer deaths (3,689 plus 351 deaths).

The remaining 16% of the gain (743 cancer deaths) were from other redistribution causes where cancer was in scope as a target disease. For example, a proportion of septicaemia and pneumonitis deaths could be reassigned to a specific cancer in the ABDS disease list, provided there was evidence in the multiple-causes-of-death data of a combination of septicaemia or pneumonitis with a specific cancer cause. The redistribution groups and methods that have cancer in scope of target diseases are shown in Appendix Table B2.

Redistribution of Indigenous deaths

The same redistribution methods and algorithms used to redistribute total Australian deaths in the ABDS 2011 were used to redistribute Indigenous deaths to maintain comparability. A total of 636 Indigenous deaths (8.3%) were identified for redistribution in 2011 (85% were redistributed using empirical evidence, and 15% using proportional redistribution). The impact of redistribution by disease group is shown in Appendix Table B5.

Missing age and sex

Age at death is missing from some records in the mortality database. As age at death is required to estimate YLL, death records missing this data item were coded according to the median age at death for all deaths in the same sex-cause group.

There were no deaths with missing sex information for the reference period used in YLL calculations.

Reference life table

Life expectancy and life tables

The measure of life expectancy shows how long, on average, a person is expected to live, based on current age- and sex-specific death rates in the population. It is a summary measure commonly used to describe the health of a population. It specifies the remaining life expectancy at each age, with life expectancy at birth (the number of years of life that a person born today can expect to live) being the most commonly used. For a given country, estimates of life expectancy are derived from its actual life table, which summarises the observed pattern of mortality and survival in the population.

YLL is an estimate of years of life lost due to premature death, and so has the character of a 'health gap' measure. As such, it requires an aspirational or potential life span to be able to quantify the gap between the current observed mortality and the counterfactual scenario where all mortality is averted until very old age.

Burden of disease studies use a reference life table, which corresponds to the aspirational or maximum life span for an individual in good health. It is typically more favourable than the actual life table of the population being studied. It is used to produce estimates of life expectancy at each age, so that the number of years of life that are lost from dying at a specific age can be derived. For example, if the remaining potential life expectancy of a person aged 55 is 30 years (that is, at 55 a person could potentially, based on the reference life table, live to 85), then a death at 55 represents a loss of 30 years of life.

Choice of reference life table

The choice of reference life table will affect burden of disease estimates. Other things being equal, a reference life table with longer potential life expectancies at all or most ages will result in greater YLL. Applying the same reference life table across multiple settings enables comparison between population groups and across time.

The ABDS 2011 uses the standard reference life table used in GBD 2010 and 2013 (Murray et al. 2012) when calculating YLL for the Australian, subnational and Indigenous populations.

GBD standard reference life table

The GBD 2010 standard reference life table was derived from worldwide experience of mortality rates (Murray et al. 2012). For each age, GBD selected the lowest age-specific death rate observed in any of the countries the study covered, except those with very small populations. The result is a hypothetical life table based on the most favourable age-specific mortality experienced anywhere. It shows potential life expectancy at any age; in particular, it shows potential life expectancy at birth to be 86.0 years for both males and females. Appendix Table B6 shows the GBD standard life expectancies for each age at death.

Important features of this reference life table are that it:

- is aspirational—that is, it reflects the lowest observed death rates to construct a measure of potential maximum life span (exceeding, as a rule, the actual life expectancy observed in any country)
- applies to all population groups—that is, it assumes the same aspirational life expectancy for any population group. It is the same for males and females, and for Indigenous and non-Indigenous Australians, assuming no difference in the survival potential of any of those groups.

The estimates of potential life expectancy in the GBD standard reference life table are different to that for the Australian population derived by the ABS from actual Australian mortality rates.

The GBD life table represents a longer life span than the Australian life tables. The life expectancy for Australian males and females at birth in 2010–2012 was 79.9 and 84.3 years, respectively—lower than the aspirational life expectancy of 86.0 years used in both GBD and ABDS. Similarly, the life expectancies for Australian males and females in 2002–2004 were lower than the GBD standard at 78.1 and 83.0 years, respectively. For comparison with the GBD 2010 standard life table, the life expectancies for the Australian population for 2010–2012 and 2002–2004 are shown for selected ages in Appendix Table B7.

Indigenous considerations

The same life table as used for national estimates (GBD 2010) was used for Indigenous estimates in the ABDS to maintain comparability in the YLL estimates produced.

The choice of standard life table will not onlyhave an impact on the size of Indigenous and non-Indigenous YLL estimates, but also on estimates of the gap in YLL rates between Indigenous and non-Indigenous Australians. This is because the 2 populations have different distributions of ages at death, so the choice of life span will affect each population's age-specific YLL estimates differently.

Sensitivity analyses by the AIHW showed that using the GBD standard life table results in a greater YLL for Indigenous Australians, and a greater YLL rate difference between Indigenous and non-Indigenous Australians, than using life tables with a lower life expectancy (as used in previous burden of disease studies). But this analysis also found that YLL rate ratios remained stable regardless of which life table was used, and showed no difference in the ranking of diseases in Indigenous YLL or in the gap in fatal burden (AIHW 2015b).

Subnational estimates

State and territory

YLL estimates by state and territory were derived directly from the NMD. Deaths were classified to state and territory according to the state of usual residence of the deceased. YLL were calculated accordingly.

The state and territory analyses used the national redistribution algorithms.

Remoteness

Analysis for remoteness was carried out using the remoteness code shown for each death record in the NMD. In the NMD, remoteness refers to the level of remoteness of each deceased person's usual residence, and is derived using the Australian Statistical Geography Standard (ASGS): Volume 5—Remoteness Areas July 2011 (ABS 2014d).

Where information about remoteness was missing from the death record, remoteness was ascribed according to the overall pattern of remoteness by state (using state of registration of the death) and sex. For example, female deaths registered in South Australia with missing remoteness information were apportioned across remoteness areas based on the proportions of the female population across the remoteness areas for South Australia.

Socioeconomic group

As discussed in Chapter 2, ABDS did not have information on socioeconomic status at the individual level. Instead, for national estimates, the ABDS 2011 derived socioeconomic group from the Index of Relative Disadvantage of the SEIFA index, which was based on the socioeconomic characteristics of the deceased person's area of usual residence.

Death records with an unknown or non-specific geographical location were assigned to socioeconomic groups according to the overall pattern of socioeconomic status by sex within each state. For example, male deaths registered in Victoria with missing area of usual residence were apportioned across socioeconomic groups based on the proportions of the male population across socioeconomic groups in Victoria.

Indigenous subnational estimates

Indigenous YLL estimates by selected state and territory, remoteness and socioeconomic group were derived directly from the NMD according to the deceased's place of usual residence.

For Indigenous YLL estimates by state and territory (reported for New South Wales, Queensland, Western Australia and the Northern Territory), deaths were adjusted for Indigenous under-identification using state/territory specific adjustment factors from the ABS's Census Data Enhancement study.

For Indigenous YLL estimates by remoteness, remoteness specific adjustment factors from the AIHW's Enhanced Mortality Database project (Appendix Table B1) were used to adjust Indigenous deaths.

For Indigenous YLL estimates by level of socioeconomic disadvantage, deaths were adjusted using the national age-specific adjustment factors from the ABS's Census Data Enhancement study. The Indigenous Relative Socioeconomic Outcomes Index was used to classify Indigenous deaths into socioeconomic groups (Biddle 2013).

4 Estimating the non-fatal burden

Expressed as years lived with disability (YLD), non-fatal burden is a measure of healthy years lost due to ill health. YLD estimation captures the frequency, severity, comorbidities and consequences of each disease in the disease list, and quantifies their joint impact on the population in terms of the difference between time lived in full health and time lived with one or more health problems. YLD estimation is the most complex and time consuming aspect of calculating disease burden.

YLD estimates in ABDS 2011 are based on prevalent cases (the number of people experiencing each disease) at a given point in time. YLD are calculated from the **point prevalence** (the number of people experiencing health loss from the condition on a given day) multiplied by a disability weight (which reflects the severity of the disease). As such, YLD should be interpreted as the total number of years spent in less than full health by the population **in the reference year**, weighted according to the health loss associated with each disease.

YLD estimation requires some important methodological decisions, including, but not limited to, the choice of conceptual disease models, severity distributions, disability weights, and the treatment of comorbidity. Also, some complex estimation problems result from the fact that the available data are often not in the form or at the granularity required.

Key terms used in this chapter

comorbidity: A health problem/disease that exists at the same time as (an)other health problem(s).

conceptual disease model: Representation of clinical conditions designed to summarise what is known about the disease epidemiology, the nature of the disease (that is, whether it is chronic, acute, episodic or progressive), and its treatment.

disability weight: A factor that reflects the severity of health loss from a particular condition on a scale from 0 (perfect health) to 1 (equivalent to death).

envelope: The total prevalence of a condition present in the population that is used to constrain the combined prevalence of sequelae common to a number of diseases.

health state: Reflects a combination of signs and symptoms that result in health loss, and are not necessarily unique to a particular disease. A health state can be a severity level of a **sequela** (typically mild, moderate and severe levels are distinguished). For example, the health state 'mild heart failure' is used as a sequela of coronary heart disease, hypertensive heart disease, congenital heart disease and several other conditions. Each health state is associated with a **disability weight**.

incidence: Refers to the occurrence of a disease or event. The incidence rate is the number of new cases occurring during a specified time period.

prevalence: Refers to the existence of a disease or event, whether or not it is newly occurring; the prevalence rate is the number of cases existing at a point in time (point prevalence) or over a specified time period (period prevalence).

sequelae: Health consequences of diseases and injuries. For example, heart failure due to coronary heart disease.

Overview of methods

YLD measures the impact of living with ill health—that is, the non-fatal component of burden of disease. YLL (discussed in Chapter 3) represents the fatal component.

The findings of ABDS 2011 are reported at the level of the 200 diseases (including the alternate reporting category 'Nature of injury') that constitute the disease list for the study. This is achieved using the following steps:

Step 1

The first step to estimate YLD is to define the major disabling sequelae associated with each disease in the disease list, and attribute disability weights that express the health loss on a scale from 0 (no health loss) to 1 (total health loss) associated with each sequelae. Several sequelae may be associated with each disease. A total of 291 sequelae have been defined in ABDS 2011.

To provide a set of weights for this many sequelae, GBD 2010 pioneered (and ABDS 2011 has followed) the practice of using estimates of the health losses associated with a smaller set of health states to which each of the sequelae can be mapped. This approach provides greater flexibility in capturing the health loss from a variety of diseases.

The health states and disability weights used in this study for both national and Indigenous estimates are drawn from GBD 2013 (see GBD 2013 Collaborators 2015b). These were originally derived for GBD 2010 from a large, multinational, cross-cultural study (Salomon 2010; Salomon et al. 2012) and were further refined for GBD 2013. The 291 sequalae in ABDS 2011 were mapped to 196 of the 236 available health states (see Appendix Table C1). This resulted in 641 sequela-health state combinations that included the different severity levels (such as mild, moderate and severe) of some health states.

Step 2

The next step is to compile estimates of point prevalence for each of the 641 sequela-health state combinations. The available data are not always ideal to estimate prevalence, and modelling must often be applied to convert them to the form and granularity needed.

An adjustment must then be made for the potentially biasing effect of comorbidity; in ABDS 2011, the bias adjustment has been effected through altering the suite of disability weights.

Step 3

The final step is to calculate YLD for each disease, which is calculated up from the sequela level (for each age and sex), described as:

$$\text{YLD} = \sum_i PP_i \text{ x } DW_i$$

where:

$\sum_i$ is the sum over all sequelae

i is an index for sequela

PPi is the point prevalence of sequela i

DWi is the disability weight for sequela i (in practice, a weighted average of the disability weights for the component health states associated with each sequela).

The estimation of point prevalence and adjustment for comorbidity are described more fully in following sections. The rest of this chapter details the methodological decisions that underpin estimates of non-fatal burden.

The same or broadly similar methods were used to derive YLD estimates for Aboriginal and Torres Strait Islander people, states and territories, remoteness areas, and socioeconomic groups.

Conceptual disease models

Fundamental to YLD estimation are epidemiological models that describe the evolution of a disease (for example, onset, duration, remission and case fatality) and its relationship with epidemiological variables (such as incidence, prevalence and mortality).

As the disability weights adopted for the ABDS 2011 are provided at the health state level, these epidemiological models needed to be converted into simpler conceptual models describing the significant outcomes (sequelae) of each disease, and the health states that best represent the health loss from each outcome, as well as the time spent in this state. These conceptual models underpin all YLD estimates.

The conceptual models were developed in conjunction with disease experts. In many cases, a conceptual model was based on similar models used in previous burden of disease studies.

Defining sequelae and health states

One or more sequelae were defined for each disease in the disease list. Due to the difficulty of assembling data with the granularity and dimensions required for YLD estimation, only sequelae causing significant health loss were included in the conceptual models.

Within a single disease, a person could have any number of sequelae simultaneously—for example, a person experiencing health loss from diabetes might at the same time have health loss from diabetic foot and vision impairment due to diabetes. A person might also have multiple sequelae from multiple diseases simultaneously—for example, a person with health loss from diabetic foot might also have heart failure due to coronary heart disease. The impact of multiple sequelae are adjusted for in the comorbidity bias adjustment.

Each sequela is then mapped to one or more health states. Health states are the functional consequences or symptoms experienced by people with each disease sequela—for example, heart failure is the functional consequence of heart failure regardless of whether it is due to coronary heart disease, cardiomyopathy or rheumatic heart disease. Multiple states within a sequela indicate its severity (for example, mild, moderate, severe heart failure) or disease progression (such as diagnosis and treatment, controlled, metastatic and terminal phases of cancer). As a result, within each sequela, a person can only be in one health state at any given point in time.

An example showing how coronary heart disease and inflammatory heart disease map through sequelae to health states is provided in Figure 4.1. The list of sequelae for each disease and resultant health states are summarised in the disease-specific sections in Chapter 5.

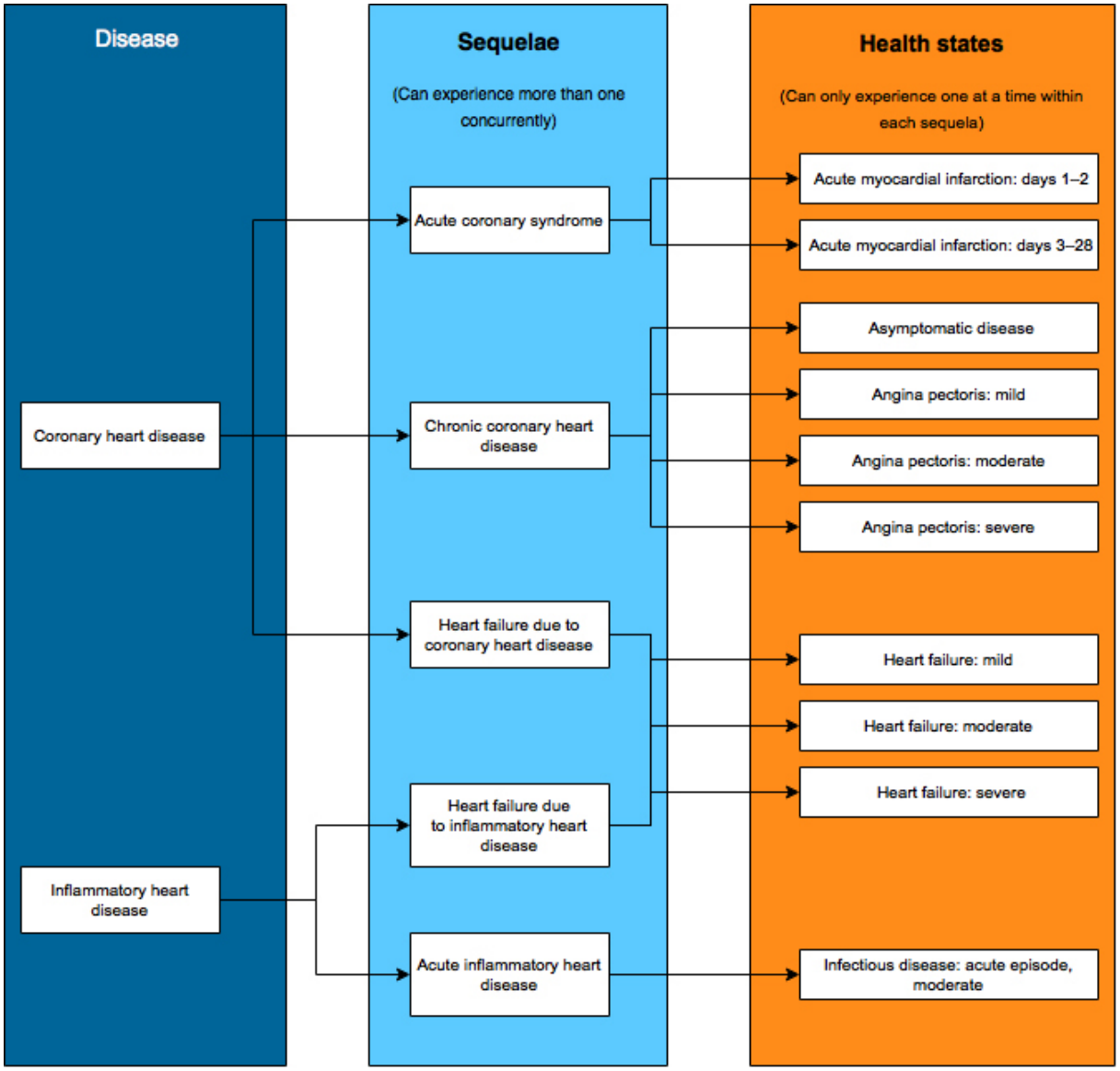

Figure 4.1: Example mapping coronary heart disease and inflammatory heart disease to component health states

Disability weights

Sequelae map to one or more health states, which each have an associated disability weight reflecting the health loss experienced by a person while in that health state.

The health states and disability weights used in the ABDS 2011 were drawn from GBD 2013 (see GBD 2013 Collaborators 2015b).

The same disability weights were used for national and Indigenous YLD estimates.

Estimating point prevalence of each sequela

Point prevalence is the number of cases at a given point in time. This differs from period prevalence, which refers to the number of cases during a period of time, such as 1 year. The ABDS 2011 estimated point prevalence as at 30 June 2011 and 30 June 2003.

The YLD estimation requires point prevalence at the sequelae–health state level for every disease at the age-sex level. In practice, such rich data rarely exist. The data may be expressed in other forms (such as period prevalence or incidence). Further, the measures that might be used to model point prevalence (such as incidence, period prevalence or mortality) are usually available only at the disease level, rather than at the finer sequela or health state level. As a result, point prevalence at the sequelae–health state level was generally modelled from those broader data sources, or, where no empirical data existed, was based on assumptions validated by disease experts.

Data sources

Unlike mortality data, there is no single comprehensive and reliable source of data on the incidence, prevalence, severity and duration of all non-fatal health conditions. Instead, morbidity estimates were drawn from a wide variety of existing sources of epidemiological measures (such as incidence, prevalence and mortality) from disease registers, administrative data, surveys and epidemiological studies.

In many cases, a single primary source provided enough information, but multiple sources were often needed to provide a complete set of data for each disease—for example, for all ages, for population subgroups or for the different sequelae.

No new surveys or meta-analyses of the epidemiological or clinical literature were undertaken as part of the ABDS 2011. This study drew on the findings of meta-analyses done for GBD or by other investigators.

Major data sources used to estimate prevalence, incidence or other epidemiological parameters included the National Hospital Morbidity Database (NHMD) and the Australian Cancer Database (ACD) held by the AIHW, and the Australian Health Survey 2011–12 (AHS) and the Australian Aboriginal and Torres Strait Islander Health Survey 2012–13 (AATSIHS) held by the ABS. For further information on these data sources, including data quality statements, see <www.aihw.gov.au/hospitals-data/national-hospital-morbidity-database>, <www.aihw.gov.au/australian-cancer-database> and <www.abs.gov.au/australianhealthsurvey>.

Primary data sources used for each disease for both national and Indigenous prevalence estimates are summarised in Appendix Table C2.

To estimate point prevalence, ABDS needed data relating to people rather than clinical events. The NHMD was a key data source for some diseases, but as it provides counts of the number of hospital separations rather than the number of individual patients, the Western Australian Department of Health calculated persons–to–hospitalisations ratios using linked Western Australian hospital and deaths data for a number of sequelae. This ratio was then applied to corresponding hospitalisation counts by sex and age from the NHMD to derive a count of persons. This approach assumed that the other states and territories have the same hospital presentation ratio as Western Australia. Further detail on when and how this method was used is available in disease-specific sections of Chapter 5.

Indigenous considerations

Adjusting for Indigenous under-identification in hospitals data

Indigenous Australians are under-identified in hospitals data to varying degrees across state and territory and remoteness areas. This results in an underestimate of hospitalisations of Indigenous Australians. In the ABDS 2011, hospitalisation data used to calculate Indigenous YLD estimates were adjusted for Indigenous under-identification using adjustment factors from hospital data quality studies done by the AIHW (Appendix tables C3 and C4). These studies were undertaken on admitted patients in public hospitals only, and estimates were not adjusted for the casemix of patients or private hospitals.

Adjusting for poor quality of Indigenous data

When the quality of Indigenous data was considered to be adequate for reporting in only some jurisdictions and no adjustment factors were available, analysis was restricted to only those jurisdictions with acceptable data quality, and combined rates from these jurisdictions applied to the populations of the remaining jurisdictions to complete the national Indigenous data. Instances where this occurs are detailed in Chapter 5 under relevant diseases.

Indirect methods for deriving Indigenous prevalence

Where no data were available to provide a reliable Indigenous prevalence estimate, indirect methods were needed to derive prevalence estimates. Such methods included applying rate ratios (such as Indigenous-to-non-Indigenous ratio) from proxy data sources (for example, hospitalisations) to the total population prevalence.

Potential indirect methods were assessed against a set of guidelines developed by the AIHW, which covered dimensions relating to the data source used in the indirect method (for example, comparability, relevance and representativeness, currency, accuracy, coverage, statistical uncertainty, measurement error and credibility). This assessment was used in conjunction with expert advice to determine the most appropriate indirect method to derive an Indigenous prevalence estimate for each disease.

Indirect methods were used to derive Indigenous prevalence for either the whole or part of the disease for 40 diseases across 8 disease groups. Of these, 12 (30%) used hospitalisation rate ratios, 29 (73%) used rate ratios from other data sources, and 2 (5%) used Maori prevalence rates. A list of these diseases and sequelae, and the indirect methods used, can be found in Appendix Table C5. Mental and substance use disorders represented the large majority (85%) of the Indigenous YLD produced based on indirect methods (and accounted for 9 of the diseases).

A further 11 diseases used national prevalence rates to derive Indigenous prevalence for the whole disease—representing 5% of total Indigenous YLD in 2011—and an additional 11 diseases used national ratios applied to Indigenous hospitalisations or cancer incidence rates to derive Indigenous prevalence for particular sequelae (Appendix Table C6).

Severity distributions

The overall prevalence of a sequela that maps to more than one health state was distributed across those health states using Australian empirical data or epidemiological studies, where possible. The proportion of prevalent cases in each health state at a point in time is referred to as the severity distribution for the sequelae in question.

Where there were no empirical data on the distribution of health states within a sequela, severity distributions were adopted from NZBDS or GBD 2013. GBD 2013 were global

severity distributions, but generally derived from data from developed countries (predominantly the United States of America and/or Australia), and so were considered appropriate to the Australian context.

Indigenous severity distributions

Where data were available, Indigenous-specific severity distributions were used for estimates for the Aboriginal and Torres Strait Islander population. Where such data were not available, the severity distributions used for national estimates in the study were adopted.

Modelling practices

Modelling of point prevalence from epidemiological measures—such as period prevalence or incidence—required different approaches, depending on the type of condition being modelled and the nature of the data available. For consistency across the ABDS, the following practices were applied in the circumstances described.

Acute versus chronic sequelae

For chronic conditions or conditions that last for at least 1 year, point prevalence is equal to annual prevalence. Prevalent age (the age associated with the disease case, which is carried into YLD calculations) is the person's age in the reference year.

For sequelae with short duration (such as appendicitis), acute events within a chronic disease (such as acute coronary syndrome) and the acute phase of injuries, point prevalence must take into account the duration of the health loss. Where health loss is less than 1 year, point prevalence is numerically equal to incidence multiplied by duration, where duration is expressed as a fraction of a year. As duration is less than 1 year, the prevalent age at which health loss occurs is the same as the incident age.

Episodic diseases

Episodic diseases are characterised by relapse and quiescent phases.

Where the quiescent phase remained as background health loss during an acute phase (for example, chronic pancreatitis during an episode of acute pancreatitis), the phases were treated as separate sequelae, and the prevalence of the quiescent phase was assigned for the whole year.

The prevalence of the acute phase was estimated using the same approach as for acute conditions. The combined health loss of co-existing sequelae was adjusted for in the comorbidity bias adjustment (described in 'Dealing with comorbidity').

Where the quiescent phase was not evident during an acute phase (for example, migraine), the phases were treated as severity levels, and the prevalence distributed according to the frequency and duration of the relapse using the same approach as for acute sequelae.

Progressive diseases

Progressive diseases are characterised by disease progression through various phases.

Where these phases generally lasted less than 1 year, and could not co-exist (such as the progression through cancer from diagnosis, metastases and terminal phase), these were treated as severity levels, and prevalence was distributed according to the duration of the phase.

Where the progressive phases could co-exist (such as amputation due to diabetes), these were generally treated as separate sequelae, and estimated separately. The combined health loss of co-existing sequelae was adjusted for in the comorbidity bias adjustment.

Data transformation

Where data sources used a different case definition, or a period prevalence (for example 1-month and 6-month prevalence), the data needed to be adjusted to be consistent, which was done using expert advice. Details of such adjustments are included in the relevant disease-specific section in Chapter 5.

Use of DISMOD

DISMOD II is a freely available statistical software tool commonly used in burden of disease studies to calculate missing epidemiological estimates, or to refine them. It requires epidemiological estimates (such as measures of incidence, prevalence, remission and mortality) as inputs to calculate related epidemiological measures. For example, to estimate the prevalence of the long-term sequelae of injury, estimates were available for the incidence, remission of the injury sequelae and mortality (in this case, the mortality rate ratio). Using these measures as inputs, DISMOD II produces an estimate of prevalence that is consistent with the input parameters.

DISMOD II was only used to produce estimates for those sequelae for which limited data sources for prevalence were available, such as long-term sequelae for injuries and congenital abnormalities. More direct methods of estimating prevalence were used where adequate data were available.

Further information on DISMOD II is available at <www.who.int/healthinfo/global_burden_disease/tools_software/en>.

Estimating the total prevalence of conditions that are sequela to many disease

There were a small number of conditions (heart failure, vision loss, anaemia, infertility, intellectual disability and cerebral palsy) that were sequelae of many different diseases. For each of these conditions, the combined prevalence of the different sequelae must equal the total prevalence of the condition present in the population.

For example, anaemia is a sequela of iron-deficiency anaemia, haemolytic anaemia, uterine fibroids, chronic kidney disease, gastroduodenal disorders and maternal haemorrhage. If the prevalence of anaemia due to each of these diseases were estimated independent of each other, there is a risk of either under-estimating the total prevalence of anaemia (as there might be a source of anaemia not counted), or over-estimating the total health loss as the combined prevalence may exceed the total anaemia present in the population.

To overcome this problem, the total anaemia present in the population was treated as fixed (referred to as an 'envelope'), and the individual prevalences of anaemia due to each of these diseases adjusted to ensure they summed to the overall prevalence.

Envelopes were used for heart failure, vision loss, anaemia, infertility, intellectual disability and cerebral palsy. The details of prevalence estimation and the methods for adjustment for each envelope are described in Chapter 5.

Dealing with comorbidity

Comorbidity occurs when a person experiences several diseases or injuries simultaneously. This might arise by coincidence (known as independent comorbidity), such as when someone has both asthma and dental caries. Or it might reflect systematic influences, such as when: a single risk factor (for example, an environmental pollutant or physical inactivity) gives rise to several health conditions; multiple conditions are associated genetically; or when one condition (or its treatment) gives rise to another condition. The clinical and epidemiological literature offers multiple views, causal pathways and taxonomies of comorbidity.

Comorbidity is of interest in its own right. The preferred clinical treatment of a person experiencing comorbidity might not be just the simultaneous application of treatments for the co-conditions. An understanding of comorbidity might be important to assess and ameliorate risk factors. Patterns of comorbidity may differ markedly between subpopulations of interest (for example, between young and old, Indigenous and non-Indigenous, urban and rural) and such differences affect health policies, programs and practice.

Comorbidity in burden of disease studies

Comorbidity poses a particular problem in estimating burden of disease. To estimate burden inclusive of comorbidity, we would need both a full suite of:

- unit records for every person in the population, showing what combination of (comorbid) conditions that person experienced in the reference period
- disability weights associated with every observed combination of comorbid conditions.

While this would enable the estimation of the ideal aggregate YLD (by summing all the health losses implied by the observed pattern of comorbidity), this hypothesised data set would not enable the compilation of a table of the comorbidity-adjusted YLDs for individual diseases consistent with the total YLD.

If the hypothesised data set were available, and if disability weights were additive (that is, if the health loss from a comorbid combination of conditions were just the sum of the health losses from the component conditions), then comorbidity might prove relatively unproblematic for a burden of disease study. But those ideal circumstances are not realised.

First, the available data are less than ideal, because:

- **prevalence** derived from a wide variety of data sources and models is generally restricted to a single health condition, not combinations of conditions, and there is no data on the pattern of all possible comorbidities
- available suites of **disability weights** refer to single health conditions, rather than all possible combinations of conditions.

Second, it is implausible to assume that disability weights are additive:

- Consider the case of Jane Doe who has metastatic cancer (disability weight=0.451), migraine headache (disability weight=0.441) and severe epilepsy (disability weight=0.552). If we ignore comorbidity, Jane would contribute 1.444 person-years to aggregate YLD, which exceeds the ceiling of 1 person-year's health loss on any individual's contribution.

As a result, the total of the (unadjusted) condition-by-condition estimates of YLDs created using the available prevalences and disability weights will not coincide with the ideal aggregate YLD described above. This discrepancy is termed 'comorbidity bias', and must be adjusted for.

In the absence of comprehensive data sets, adjusting for comorbidity bias in burden of disease estimation has relied on modelling both the prevalences and the disability weights for comorbid conditions. The modelled data are then used to compute a rescaled (comorbidity-adjusted) disability weight for each individual disease—and it is from these adjusted weights (applied to the original prevalences) that comorbidity-adjusted YLDs are derived.

Comorbidity bias adjustment in ABDS 2011

The strategy outlined above has been adopted for ABDS 2011. The key idea underpinning the adjustment procedure was to simulate a population with comorbidities and their associated health losses (disability weights) that mimics the ideal data set hypothesised earlier, to support the compilation of comorbidity-adjusted disability weights.

- For prevalences, the ABDS 2011 assumed independent ('multiplicative') comorbidity—that is, the probability of having a specific combination of conditions is simply the product of the probability of having each of the constituent conditions. In reality, the pattern of comorbidities is likely to be more complex, but there is evidence that this assumption provides an approximation acceptable for the purposes of burden of disease estimation.
- For disability weights, the ABDS 2011 assumed a multiplicative relationship between the health loss suffered by a person with specific combinations of sequelae and the losses associated with the constituent sequelae. The combined disability weight for a comorbid combination of conditions is equal to:

  1 *minus* {the *product* of {1 *minus* the disability weight for each constituent sequela}}.

  This assumption puts a maximum value of 1 on the disability weight that can arise from any combination of conditions.

Assumptions of these kinds have been used in GBD 2010, GBD 2013 and other recent burden of disease studies.

Because disease prevalences are known to vary by age and sex (and to support results to be broken down), the procedure was undertaken at the sequela level for each age and sex. To account for known differences in disease prevalence in the Australian and Indigenous populations, and in points in time, comorbidity bias adjustment was undertaken separately for the Australian and Indigenous populations, and for each of the reference years—2003 and 2011—using the prevalences specific to those years and populations.

Assembling the simulated population entailed the following steps.

1. The available data on single-condition prevalence (and the independence assumption) were used to simulate a population that shows all possible combinations of 1, 2, 3 or 4 comorbid conditions selected from the ABDS 2011 list of sequelae. The frequency of a given combination within the simulated population depends on the probabilities (taken as the per-capita prevalence) of individual conditions. In reality, a person may experience 5 or more conditions, but the approximation error from capping the number

of conditions in the synthetic population at 4 might be expected to be negligible. The probability (expected prevalence) associated with a combination of conditions shrinks rapidly toward 0 as the number of co-present sequelae increases. For example, the impact of any change on the calculated YLD of the fifth co-present sequelae is minimal, because the comorbidity-bias-adjusted disability weight is stable to the fifth decimal point. Any change in the fifth decimal place will only affect the YLD calculated for prevalence estimates greater than 100,000 in a particular age-sex cohort.

2. The available data on single-condition disability weights (and the multiplicative assumption) was used to attach a disability weight to each combination of comorbid conditions, and, from there, to each population age and sex group.

The adjusted YLDs that result from applying adjusted weights that have been derived from the simulation are expected to be a reasonable approximation to the ideal aggregate YLD (and comorbidity-adjusted YLDs for individual conditions) described earlier. The closeness of the approximation and whether an adjusted YLD has over-compensated or under-compensated for comorbidity bias depends on how reasonable the assumptions regarding independence and multiplicativity are. Validation studies by GBD and the New Zealand Ministry of Health suggest that the approximations appear reasonable at aggregate level. Further validation or improvement of the methods await the availability of richer data sets.

Estimating YLD for residual diseases

Where possible, the prevalence of the residual group of diseases within each disease group (for example, other malignant neoplasms) was estimated or modelled directly from data.

Where this was not possible, either due to the variety of conditions that it encompassed, or through lack of available data, the YLD for the residual diseases was calculated using the ratio of YLDs to YLLs estimated for other conditions in that disease group (at the age and sex level) applied to known YLLs. The YLL-to-YLD ratio was limited to those conditions in the disease group that were similar in nature to those included in the residual.

This method was used to generate estimates for other cardiovascular, endocrine, gastrointestinal, infectious, congenital, kidney and neurological diseases.

Further information on the diseases included in the YLL-to-YLD ratio for each disease group is included in Chapter 5.

5 Disease specific methods

This chapter provides information on the methods used for mortality and morbidity estimates specific to each of the 17 disease groups, in alphabetical order. It also provides details on estimating the prevalence of the following conditions, which are sequela to many diseases (referred to as envelopes) enforced in the study within these sections:

- anaemia—blood and metabolic disorders
- cerebral palsy—infant and congenital conditions
- heart failure—cardiovascular conditions
- infertility—reproductive and maternal disorders
- intellectual disability—mental and substance use disorders
- vision loss—hearing and vision disorders (visions loss).

Detailed information is provided on the methods used for 2011 national estimates. Where these methods differ for subnational estimates, 2003 estimates or Indigenous estimates, this is described separately.

The methods described in Chapter 3 for mortality estimates (specifically coding, redistribution and Indigenous-specific methods) are standard to all estimates produced in the ABDS 2011, and are not repeated here.

Blood and metabolic disorders

Mortality estimates

Deaths related to blood and metabolic disorders were assigned from the NMD as defined by the disease list (Appendix Table A2). Deaths coded to E85.3, E85.4, E85.8, E85.9, were proportionally redistributed to infectious diseases, cancer and other neoplasms, respiratory diseases, gastrointestinal disorders, neurological disorders, blood and metabolic disorders and musculoskeletal conditions, based on Australian mortality data. Deaths coded to E86 and E87 were proportionally redistributed across all disease groups (except oral and skin disorders) (Appendix Table B2).

Morbidity estimates

Sequelae and health states

Sequelae and health states assigned to blood and metabolic disorders are included in Table 5.1. All diseases were assigned health loss for the entire year. Assumptions are outlined in subsections for individual diseases.

Table 5.1: Sequelae and health states for blood and metabolic disorders

Disease	Sequela	ABDS 2011 health state identifier[a]
Cystic fibrosis	Non-respiratory complications due to cystic fibrosis	207
	Respiratory complications due to cystic fibrosis	55, 56, 57
Haemophilia	Haemophilia	128, 207, 262
Haemolytic anaemia	Haemolytic anaemia	207
	Acute, severe event due to haemolytic anaemia	194, 2
	Anaemia due to haemolytic anaemia[b]	196, 197
Iron-deficiency anaemia	Anaemia due to iron-deficiency anaemia[b]	195, 196, 197
Protein-energy deficiency	Stunting due to protein-energy deficiency	211
	Wasting due to protein-energy deficiency	210, 211
Other blood and metabolic disorders	Anaemia due to other blood and metabolic disorders[b]	197
	Non-anaemic deficiency due to other blood and metabolic disorders	195
	Immune suppression due to other blood and metabolic disorders	10
	Metabolic dysfunction due to other blood and metabolic disorders	31

(a) See Appendix Table C1.

(b) Part of anaemia envelope.

Prevalence estimation

Anaemia envelope

As anaemia is a sequela of multiple conditions across the ABDS, its overall prevalence was calculated to ensure the sum of estimates for sequelae do not exceed the total—referred to as an 'envelope'. To avoid double-counting, and adhere to mutual exclusivity for each disease, the proportion of anaemia due to each disease was estimated.

Diseases that include anaemia as sequelae include iron-deficiency anaemia, haemolytic anaemia, uterine fibroids, chronic kidney disease, gastroduodenal disorders and maternal haemorrhage.

Prevalence estimation of the anaemia envelope

Prevalence rate of individuals at risk of anaemia in ages 10 and over, by sex, were derived from the AHS 2011–12 and converted to 5-year age groups using DISMOD II, assuming no remission and no excess mortality. Iron-deficiency anaemia was assumed to be in 4% of children aged under 1 (Oti-Boateng et al. 1998), 2% for children aged 1–4 (Mackerras et al. 2004; Looker et al. 1997), and 1% for children aged 5–10 (Sadler & Blight 1996), based on epidemiological studies.

Estimates for diseases with anaemia as sequelae were subtracted from the anaemia envelope estimates. See the relevant disease groups for estimation of anaemia due to specific diseases. Remaining estimates resulted in the prevalence of iron-deficiency anaemia.

Maternal haemorrhage estimates were not included in this subtraction, as this condition is short term. It is also not included in AHS 2011–12 results.

Cystic fibrosis

Prevalence of cystic fibrosis was derived from the Australian Cystic Fibrosis Data Registry 2011 annual report (Cystic Fibrosis Australia 2012). Registrants by age, sex and severity (lung function) was obtained from the report for ages 0–60.

As the severity age groupings in this report did not align to age groupings used in the ABDS 2011, the following assumptions were made:

- 0–10 years were assigned the 6–11-year severity level
- 10–19 years were assigned the 12–17-year severity level
- 20–29 years were assigned the 18–29-year severity level
- 30–60 years and over were assigned the 30-years-and-over severity levels.

Prevalence for people aged 0–60 was extrapolated to 100 years and over, or until it could be assumed as 0. Resulting rates were then compared against hospital separation rates for 2011, to assess slope with increasing age. The slope for those aged 40 and over appeared similar for prevalence and hospital separation rates, so trend analyses were used for ages over 60, by sex. As the estimates for the age ranges beyond 70 were increasingly small, it was assumed prevalence was 0 from 85 years onwards, consistent with hospital data.

The report details numerous markers for severity, but these conflict with other components of the ABDS 2011 (risk factor and comorbidity analyses), or are captured elsewhere (for example, in respiratory infections). Therefore, lung function was used to attribute the proportion and severity of respiratory complications due to cystic fibrosis; however in the report, there were a proportion of cystic fibrosis registrants with normal lung function when tested. These registrants had other consequences from cystic fibrosis, so a disability weight similar to mild lung function was applied to registrants with normal lung function to ensure the burden was adequately estimated for this group.

Haemophilia

Haemophilia in the ABDS 2011 included haemophilia A and B. Prevalence estimates and severity distribution were derived from the Australian Bleeding Disorders Registry 2011–12 report (National Blood Authority 2012).

The report provided severity estimates by haemophilia type, in broad age groups. The total male proportions for haemophilia A and B severity were applied to male prevalence estimates, assuming similar proportion across all ages. Based on clinical advice, it was assumed 95% of females with haemophila are classified as mild, and 5% as moderate (Rowell J 2015, pers. comm. 11 September).

Haemolytic anaemia

The disabling sequelae for heamolytic anaemia were mapped to the ABDS 2011 health states shown in Table 5.1. Table 5.2 lists diagnosis and procedure codes (using the International Statistical Classification of Diseases and Related Health Problems, 10th revision, Australian modification (ICD-10-AM) or Australian Classification of Health Interventions (ACHI) codes) for sequelae and severity distributions.

Table 5.2: Sequelae, severity and descriptions for haemolytic anaemia

Sequelae	Severity	Diagnosis/procedure descriptions	ICD-10-AM/ ACHI code
Haemolytic anaemia	Haemolytic anaemia	All haemolytic anaemias	D55–D58
Acute, severe event due to haemolytic anaemia	Acute haemolytic crisis	Sickle cell crisis	D57.0
	Surgical intervention: splenectomy	Haemolytic anaemias with splenectomy procedure code	Block: 815
Anaemia due to haemolytic anaemia	Moderate anaemia	Haemolytic anaemias excluding beta-thalassaemia	D55–D58, excluding D56.1
	Severe anaemia	Beta-thalassaemia	D56.1

Prevalence estimates for haemolytic anaemia in the ABDS 2011 were derived from the NHMD. Separations were ranked according to severity, if separations included more than 1 haemolytic anaemia diagnosis.

As a person can have multiple hospital separations in a single year, linked Western Australian hospitalisations data were used to derive persons-to-separations ratios by sex and haemolytic anaemia type. These ratios were applied to national separations to estimate the number of people admitted. The number of people admitted to hospital in Western Australia for haemolytic anaemia is assumed to be representative of all other states and territories.

Duration of health loss for haemolytic anaemia and anaemia was assumed to be for the entire year. Duration for individuals with splenectomy and acute sickle cell episodes was assumed to be 2 weeks and 7 days, respectively.

Iron-deficiency anaemia

Iron-deficiency anaemia in this study is inclusive of anaemia caused by iron deficiency and by unspecified causes. Severity was based on haemoglobin level definitions for mild and moderate anemia (WHO 2011). Proportions within each level were derived from the AHS 2011–12 biomedical data.

The prevalence of iron-deficiency anaemia is the remaining anaemia once estimates for diseases with anaemia as sequelae were subtracted from the anaemia envelope.

Protein-energy deficiency

Burden due to protein-energy deficiency was only estimated for elderly individuals and Indigenous children under 5, as these are the subpopulations most likely affected in Australia.

Prevalence estimates in elderly Australians

Burden from protein-energy deficiency in elderly Australians was not included in previous burden of disease studies. It was included in the ABDS 2011 morbidity calculations, due to current literature addressing this as a key issue in this population (Banks et al. 2007; Kaiser et al. 2010; Rist et al. 2012). Estimates are restricted to elderly Australians residing in nursing homes and those receiving at-home care.

Estimates were derived from an Australian community-living based study assessing malnutrition using the gold standard Mini Nutritional Assessment. This study identified 35% of residents were at risk of malnutrition and 8.1% were malnourished (Rist et al. 2012).

People at risk of malnutrition were considered to have mild malnutrition (based on the Mini Nutritional Assessment score highlighting nutritional decline in the previous 3 months and intervention required), and people who were malnourished were considered to have moderate/severe malnutrition. These proportions were applied to the number of people living in permanent residential aged care facilities or receiving in-home care services, by sex in 2011 at a state and remoteness level (AIHW 2012c, AIHW 2012d).

It is acknowledged that a proportion of malnutrition in the elderly population might include individuals who are in the end stages of life. As it is not possible to distinguish the cause of malnutrition, estimates in this population might be slightly over-represented.

Prevalence estimates in Indigenous children

Data from the AATSIHS 2012–13 was used to estimate the prevalence of protein-energy deficiency in Indigenous children. As advised by experts, underweight status is indicative of mild malnutrition in the Indigenous population. Population proportions were used to divide estimates into children aged under 1 year and 1–4.

Severity distributions were derived from the AATSIHS underweight class, with levels 2 and 3 identified as wasting (Cole et al. 2007). To align state distributions to remoteness area estimates, the severity distribution for the Northern Territory was used for *Outer regional*, *Remote* and *Very remote* prevalence estimates. National severity distributions were used for *Major cities* and *Inner regional areas*.

Other blood and metabolic conditions

This group includes deficiency anaemia, acquired haemolytic anaemias, coagulation defects, immune mechanism disorders, nutritional deficiencies and metabolic disorders.

To estimate prevalence, principal diagnosis separations in the NHMD were used. The ICD-10-AM codes were grouped according to the main disabling sequelae, and durations applied to the number of separations to derive prevalence (Table 5.3).

Durations were based on hospital analyses of length of stay, or durations used for conditions considered of similar burden.

Table 5.3: Definitions and durations for other blood and metabolic conditions

Sequelae	ICD-10-AM codes	Duration
Anaemia due to other blood and metabolic disorders	D51.0–D53.9, D59.0–D65, D68.0–D69.9	56 days
Immune suppression due to other blood and metabolic disorders	D70–D77, D80.0–D84.9, D86.1–D86.3, D86.8, D89.0–D89.9	2.4 days
Non-anaemic deficiency due to other blood and metabolic disorders	E00.0–E02, E50.0–E56.9, E58–E61.9, E63–E65, E67–E68	6 months
Metabolic dysfunction due to other blood and metabolic disorders	E70.0–E80.7, E83.0–E83.9, E85.0–E85.2, E88.0–E89	7 days

Subnational estimates

State and territory prevalence estimates for blood and metabolic disorders were based directly from the data source for each condition used to derive national prevalence. Prevalence estimates by remoteness and socioeconomic group were derived from hospital separations data in 2011.

2003 estimates

2003 estimates were based on the same method as for 2011.

Hospital separations were derived from the 2003 calendar year.

Registrant data from 2003 were used to estimate haemophilia and cystic fibrosis prevalence in 2003. Where age and sex or severity distributions were unavailable, these were obtained from reports closest to 2003 that provided this information.

Total iron-deficiency anaemia prevalence estimates in 2003 were derived from self-reported estimates from the National Health Survey (NHS) 2004–2005 adjusted for under-reporting. Adjustment factors were based on the difference between self-reported and biomedical measures of anaemia in the AHS 2011–12. Age- and sex-specific severity distributions from 2011 were applied to the 2004 estimate to obtain age- and sex-specific prevalence rates, and applied to the 2003 population to attain estimates for 2003.

Estimates for protein-energy deficiency in elderly Australians in 2003 used the same method as in 2011, but was based on the number of people living in permanent residential aged care facilities or receiving in-home care services in 2003, by sex.

Indigenous estimates

Where possible, the same general methods and data sources were used to derive Indigenous estimates for blood and metabolic disorders for 2011 and 2003.

Indigenous estimates based on hospital separations data were adjusted for under-identification using standard adjustment factors (see Chapter 4 and Appendix tables C3 and C4).

Registrant data for cystic fibrosis and haemophilia did not contain reliable Indigenous identifiers, so hospital separations data were used to estimate prevalence. Based on expert advice, these conditions are not as prevalent in the Indigenous population.

Biomedical data from the AATSIHS was used to estimate iron-deficiency anaemia using the same method as used for the national population.

The same data source and method used to estimate protein-energy deficiency in the national population was used for the 2011 Indigenous population. The prevalence rate of protein-energy deficiency in Indigenous children in 2011 was applied to the 2003 Indigenous population, due to lack of biomedical measurement data consistent with the 2011 method.

Cancer and other neoplasms

Mortality estimates

Cancer-related deaths were assigned from the NMD as defined by the disease list (Appendix Table A2). Deaths coded to other and ill-defined digestive organs (C26) and other and ill-defined cancers, secondary malignant neoplasms and cancers of unknown primary site (C76–C80) were redistributed based on direct evidence from the Western Australian and South Australian cancer registries (Appendix tables B2, D1 and D2).

Although also a candidate for redistribution, there were insufficient deaths due to other and ill-defined respiratory organs (C39) in the Western Australian and South Australian cancer registries to develop a redistribution algorithm. Deaths coded to C39 were instead assigned to 'unknown primary'.

Similarly, cancers of multiple independent primary sites (C97) could not be redistributed using this method, as a single cancer cannot be assigned by cancer registries. Consequently, deaths coded to C97 were also assigned directly to 'unknown primary'.

As there were insufficient data available in the Western Australian and South Australian cancer registries to form Indigenous-specific redistribution algorithms, the national redistribution algorithms were also applied to Indigenous deaths.

The same direct evidence algorithms were also applied to 2003 cancer deaths.

Morbidity estimates

Sequelae

Sequelae and health states for cancer and other neoplasms are based on the progression through 4 phases from diagnosis through metastases to potential death (Table 5.4) and long-term sequelae (usually as a result of curative treatment) for selected cancers (Table 5.5).

Table 5.4: General cancer-related sequelae and health states

Sequelae	Health state	ABDS 2011 health state identifier[a]
Diagnosis and primary therapy phase of <*cancer type*>	Cancer: diagnosis and primary therapy	18, 208[b]
Controlled phase of <*cancer type*>[c]	Generic uncomplicated disease: worry and daily medication	207
Metastatic phase of <*cancer type*>[d]	Cancer: metastatic	19
Terminal phase of <*cancer type*>[e]	Terminal phase: with medication	22

(a) See Appendix Table C1.

(b) For uncomplicated non-melanoma skin cancer only.

(c) Non-melanoma skin cancer and cancer of unknown primary site models did not include controlled phase health state.

(d) Benign and uncertain brain tumours and breast ductal carcinoma in situ models did not include metastatic phase.

(e) Breast ductal carcinoma in situ models did not include terminal phase.

Table 5.5: Long-term cancer sequelae and health states

Disease	Sequelae	ABDS 2011 health state identifier[(a)]
Laryngeal cancer	Laryngectomy due to laryngeal cancer	212
Bowel cancer	Stoma due to bowel cancer	21
Breast cancer	Mastectomy due to breast cancer	20
Prostate cancer	Impotence due to prostate cancer	49
	Urinary incontinence due to prostate cancer	48
Bladder cancer	Stoma due to bladder cancer	21
	Urinary incontinence due to bladder cancer	48
Brain and central nervous system cancer	Brain injury (mild, moderate, severe) due to brain and central nervous system cancer	181, 182, 183
Benign brain tumours	Brain injury (mild, moderate, severe) due to benign brain tumours	181, 182, 183
Ductal carcinoma in situ	Mastectomy due to ductal carcinoma in situ	20

(a) See Appendix Table C1.

Prevalence estimation

General sequelae

Average durations for each general sequela for the various cancers were primarily taken from GBD 2010, though a small number were developed specifically for the ABDS 2011 based on expert advice (Appendix Table D3). Durations were applied to the relevant epidemiological measure for each sequela to derive point prevalence.

Principal diagnosis and primary therapy

Health loss due to diagnosis and treatment of malignant cancer (except non-melanoma skin cancer—NMSC) and ductal carcinoma in situ (DCIS) was based on incidence data from the 2011 Australian Cancer Database (ACD). This assumes that people will undergo primary treatment at the time of diagnosis.

The diagnosis and primary therapy health state for NMSC was divided into 2 severity levels, depending on whether the cancer was treated in community settings (uncomplicated NMSC) or hospital settings (complex NMSC).

Uncomplicated NMSC diagnoses and treatments were sourced from Medicare Benefits Schedule claims for first surgical excision of keratinocyte cancers, and adjusted for histological confirmation. Histological confirmation is based on information from the QSkin Study by QIMR Berghofer Medical Research Institute (Thompson et al. 2014).

Complex NMSC diagnoses and treatments were sourced from separations in the NHMD with a principal diagnosis of NMSC in 2011 that underwent a skin-related surgery.

As benign and uncertain tumours of the brain and central nervous system are only reported to cancer registries in Western Australia, Queensland and Victoria, the number of incident cases undergoing diagnosis and primary therapy was not directly obtainable. Instead, the age-specific ratio of benign or uncertain brain tumours in the ACD to separations in the NHMD for Western Australia, Queensland and Victoria was applied to separations from other jurisdictions, to derive national and subnational estimates.

Incident cases for other non-malignant neoplasms were sourced from the NHMD (acknowledging that this will be the more severe end of the spectrum) using principal diagnosis, adjusted for repeat admissions.

Controlled phase

Health loss due to controlled phase of cancer was based on those people who were alive at the end of 2011 with a diagnosis of cancer in the previous 5 years—this assumes an effective cure rate of 5 years for all cancers.

Health loss is assumed for the full year for each prevalent case, minus the total person-time spent in diagnosis and primary therapy. As prevalent cases must have been alive on 31 December 2011, there is no overlap with people who died in 2011. Prevalence data for 2011 were also sourced from the 2011 ACD, which includes a linkage to the National Death Index to estimate prevalence.

Metastatic and terminal phases

Health loss due to metastatic cancer and terminal cancer in 2011 was based on people who died from cancer in 2011 (regardless of when they were diagnosed). This assumes that the number of people with metastatic and terminal phases who die of something *other* than cancer is small. To ensure that person-time does not exceed 12 months for each death, the duration of health loss in a given year for metastases was capped at 11 months.

Deaths from cancer were sourced directly from the NMD. To ensure consistency with the fatal component of the study, deaths due to unknown primary and unknown digestive cancers were redistributed before prevalence was estimated.

Long-term sequelae

As the number of people alive in 2011 who have long-term sequelae from cancer is not directly available, health loss due to long-term sequelae was generally based on proportions of cases that have undergone long-term surgery, or are otherwise known to experience health loss applied to either 10-year prevalence (that is, people alive at the end of 2011 diagnosed with cancer in the previous 10 years) or lifetime prevalence (defined for this study as people alive at the end of 2011 diagnosed with cancer any time since 1982, the start of the ACD). Health loss for long-term sequelae is assumed to apply for the full year.

Laryngectomy due to laryngeal cancer

Prevalence was based on the ratio of the number of partial or total laryngectomies with a principal diagnosis of laryngeal cancer (derived from the NHMD) to new cases of laryngeal cancer in 2011 (derived from the ACD). This was applied to the 10-year prevalence of laryngeal cancer derived from the ACD.

Stoma due to bowel cancer

Prevalence was based on the ratio of hospitalisations for permanent colostomies due to bowel cancer (derived from the NHMD) to new cases of bowel cancer in 2011 (derived from the ACD). This ratio was applied to 10-year prevalence of bowel cancer.

As individuals cannot be ascertained in the NHMD it was not possible to determine which stomas were temporary or permanent. Instead, permanent stomas were estimated using the overall colostomy closure rate for any disease derived from the NHMD.

Mastectomy due to breast cancer or ductal carcinoma in situ

Prevalence of mastectomies due to breast cancer was based on the ratio of the number of mastectomies with a principal diagnosis of breast cancer (derived from the NHMD) to new cases of breast cancer in 2011 (ACD). Age-specific ratios were applied to the 10-year prevalence of breast cancer for females; an overall ratio was applied for males.

As 10-year prevalence for ductal carcinoma in situ was not available in the ACD to support using the same method as for breast cancer, data from the NHMD were used directly to derive prevalence of mastectomies due to ductal carcinoma in situ. Hospital separations for mastectomies with a principal diagnosis of ductal carcinoma in situ from 2001–2011 were extracted from the NHMD. To derive prevalence from separations, a 10-year prevalence-to-separations ratio was derived from Western Australian linked hospitalisations and deaths data, and applied to the number of national separations. This assumes that the survival of women undergoing mastectomy for ductal carcinoma in situ in Western Australia is consistent across Australia.

Impotence and urinary incontinence due to prostate cancer

Prevalence was based on the proportions of men diagnosed with localised prostate cancer experiencing impotence and/or urinary incontinence at 3-year follow-up, according to treatment type (Smith et al. 2009) adjusted for background proportion of urinary incontinence and impotence. These were applied to the 10-year prevalence of prostate cancer derived from the ACD.

As radical treatment is not generally offered to men over the age of 70, the proportion of men likely to have undergone different treatments in the previous 10 years was only applied to men aged under 80 in 2011 (to allow for 10 years since treatment). It was also assumed there was no health loss from impotence in males aged under 15. To ensure consistency across the ABDS 2011, urinary incontinence is assumed not to apply to children aged under 5.

Stoma and urinary incontinence due to bladder cancer

In the ABDS 2011, urinary incontinence due to bladder cancer refers to the long-term effects of primary therapy for bladder cancer—that is, removal of the bladder (radical cystectomy). It does not refer to urinary incontinence experienced as a symptom of bladder cancer, which is assumed to be short term until seeking treatment.

Radical cystectomy usually results in a stoma or a neobladder being fitted in the patient, and long-term effects depend on the diversion type. Hospitalisations for radical cystectomy were used to estimate incidence hazard ratios for stomas and neobladders following bladder cancer. This was applied to the 10-year prevalence of bladder cancer from the ACD to obtain point prevalence estimates of stoma for each diversion type.

Proportions of patients with incontinence by diversion type were obtained from Gilbert et al. 2007.

Brain injury due to malignant and benign brain tumours and central nervous system cancer

Due to the scarcity of data sources on the long-term impacts of cancer and other tumours of the brain, the ABDS 2011 assumed the proportion of all brain cancer survivors with long-term sequelae was the same as the proportion of brain injury survivors with long-term sequelae (that is, 8% mild, 10% moderate, 5% severe), derived by the NZBDS (NZMOH 2013).

For brain cancer, these proportions were applied to the lifetime prevalence of brain cancer derived from the ACD. As prevalence of survivors of benign and uncertain brain tumours

was not directly available, rate ratios of age-specific prevalence rates for malignant and non-malignant tumours from a United States study (Porter et al. 2010) were applied to the lifetime prevalence of malignant tumours from the ACD to derive lifetime non-malignant prevalence.

Subnational estimates

Remoteness breakdowns of national estimates were derived by applying 2011 ASGS remoteness areas to the Statistical Area Level 2 recorded in hospitals and cancer mortality data, and postcode recorded in cancer incidence data. Deaths/cases with missing data (including data that could not be mapped) were proportionally assigned to remoteness groups based on the proportion of the population in each group, by state and sex.

Socioeconomic group breakdowns of national estimates were derived by applying 2011 SEIFA population-based Index of Relative Socio-economic Disadvantage (IRSD) quintiles to the Statistical Area Level 2 recorded in hospitals and cancer mortality data, and postcode recorded in cancer incidence data. Deaths/cases with missing data (including data that could not be mapped) were proportionally assigned to socioeconomic groups based on the proportion of the population in each group, by state and sex.

2003 estimates

2003 cancer incidence and prevalence were derived from the ACD (which contains all cancer cases up to 2011), and cancer mortality from the NMD, in the same way as for 2011.

As Medicare Benefits Schedule item codes might have changed over time, the positive predictive value provided from the QSkin Study could not be assumed to apply to estimate incidence of NMSC. Instead, incidence from the 2002 survey by Staples et al. (2006) was used for the incidence of simple NMSC, on the assumption that most would have had a simple excision prior to any complex treatment. Hospital separations data were used for health loss due to complex treatment as for 2011.

Long-term sequelae were derived in the same way using 2003-specific ratios. Where the NHMD was the primary data source, separations from the 2003 calendar year were used.

For brain injury due to malignant and benign brain tumours and central nervous system cancer, the same rates were assumed as for 2011 estimates; however, as the ACD only contains data from 1982, the lifetime prevalence for 2003 has a much shorter look-back period, and so will be lower than for 2011.

Indigenous estimates

The same general methods were used to derive 2011 and 2003 Indigenous estimates with the following exceptions:

- The 2011 cancer incidence and prevalence for the Indigenous population were derived from the average cancer incidence recorded in the 2011 ACD for 2009–2011 for the Northern Territory, Western Australia, Victoria and Queensland, and for 2007–2009 for New South Wales—these are the states with cancer incidence data considered of sufficient quality for reporting. Rates from these states combined were applied to the Australian Capital Territory, Tasmania and South Australia populations to determine national Indigenous incidence. Mortality for 2011 was derived from the average number of deaths in 2009–2011, adjusted for under-identification using ABS mortality adjustment factors as per fatal estimates (see Appendix Table B1).

- The 2003 cancer incidence and prevalence for the Indigenous population were derived from the average cancer incidence recorded in the ACD for 2002–2004 for the Northern Territory, Western Australia, New South Wales and Queensland. Rates from these states combined were applied to the Australian Capital Territory, Victoria, Tasmania and South Australia populations to determine national Indigenous incidence. Mortality for 2003 was derived from the average number of deaths in 2002–2004, adjusted for under-identification using ABS mortality adjustment factors as per fatal estimates (see Appendix Table B1).

Indigenous estimates for complex treatment of NMSC were identified from hospitals data for the relevant years, adjusted for under-identification using AIHW standard hospital adjustment factors described in Chapter 4 (see also Appendix tables C3 and C4). As no Indigenous data were available for simple NMSC, the Indigenous-to-national ratio of complex NMSC was applied to the national simple NMSC estimates, to derive Indigenous estimates for both 2011 and 2003.

National hazard-to-incidence ratios for long-term sequelae were applied to Indigenous prevalence estimates, due to insufficient data to derive Indigenous-specific ratios. The exception to this was mastectomy where there were sufficient data to derive female Indigenous-specific (but not age-specific) ratios.

As the proportion of brain injury survivors is not currently available by Indigenous status, it was assumed to be the same as the national proportion.

The number of cases of ductal carcinoma in situ diagnosed in Indigenous women was estimated by applying the ratio of small (less than 2 centimetres) breast tumours in national-to-Indigenous women to the national incidence of ductal carcinoma in situ for both 2011 and 2003.

Cardiovascular diseases

Mortality estimates

Cardiovascular disease-related deaths were assigned from the NMD as defined by the disease list (Appendix Table A2). Deaths coded to hypertension (I10, I13, I15) and heart failure (I50) were redistributed using the indirect MCOD method to all diseases except injuries. Using proportions derived from Australian all-cause mortality data, deaths coded to cardiac arrest and cardiac conduction disorders were proportionally distributed across all causes of death, while deaths coded to atherosclerosis and cardiac signs and symptoms were proportionally distributed across all disease groups excluding cancer, injuries and infectious diseases.

Morbidity estimates

Sequelae

Sequelae and health states assigned to the cardiovascular diseases are divided into acute and chronic. Heart failure is a sequela to a number of the cardiovascular diseases, and is treated as an envelope condition. Sequelae, health states and durations are detailed in 'Prevalence estimation'.

Prevalence estimation

Acute sequelae

The NHMD was the main data source used to estimate prevalence of acute sequelae listed in Table 5.6. As these events are of short duration, point prevalence was estimated by assigning duration of health loss to incidence.

Table 5.6: ABDS 2011 diseases and sequelae that use the NHMD to estimate point prevalence

Disease	Sequela	ABDS 2011 health state identifier[(a)]	Duration
Coronary heart disease	Acute coronary syndrome	24, 25	2 days (more severe) 26 days (less severe)
Stroke	Acute stroke	34, 35, 36, 37, 38	28 days
Inflammatory heart disease	Acute inflammatory heart disease	2	28 days
Aortic aneurysm	Aortic aneurysm	194	28 days

(a) See Appendix Table C1.

Acute coronary syndrome

As health loss from acute coronary syndrome is short term, point prevalence was estimated using incidence (events) multiplied by the duration for each of the 2 severity levels (2 and 26 days, respectively).

As there is no national data source, acute coronary syndrome incidence was estimated using AIHW analyses of Western Australian linked hospitalisations and deaths data to determine the number of non-fatal acute coronary syndrome events in the reference year (AIHW 2014a). Acute coronary syndrome hospitalisations were defined as separations with a principal diagnosis of ICD-10-AM I20.0 and I21. Transfers and readmissions within 28 days were excluded to avoid double-counting of the same event. This incidence rate (based on the Western Australia population) was then applied to the national population to determine national incidence. This assumed that the incidence rate for Western Australia applies nationally.

Acute stroke

Hospitalisation data were chosen over data from epidemiological studies due to the currency, national coverage and ability to provide estimates at the subnational level.

Incidence was calculated by counting the number of non-fatal separations due to stroke (defined as principal diagnosis of ICD-10-AM I60–I64) in 2011 in the NHMD.

Prevalence estimates were then split into the 5 severity levels using proportions obtained from GBD 2013 (Burstein et al. 2015), which were reapportioned to exclude asymptomatic acute stroke since it was not included in the estimates from the NHMD.

Acute inflammatory heart disease

Incidence was estimated by counting the number of separations due to acute inflammatory heart disease in the NHMD in 2011. These were defined as separations with a principal diagnosis of ICD-10-AM: I30–I33, I40–I41.

A considerable number of people have more than one hospitalisation record with inflammatory heart disease listed as a principal diagnosis in a single year (AIHW analysis of

Western Australian linked hospitalisation and deaths data sets; AIHW 2014a). Therefore, an adjustment factor from Western Australian linked data was applied to the count of inflammatory heart disease separations obtained from the NHMD to produce an incidence estimate for 2011.

Aortic aneurysm

Incidence was assumed to be the number of separations due to aortic aneurysm (principal diagnosis of ICD-10-AM: I71) in 2011 in the NHMD.

Chronic sequelae

The prevalences of chronic sequelae were estimated using NHMD, Western Australian linked hospitalisations and deaths data, and the NZBDS.

The sequelae for which a combination of NHMD and linked Western Australian hospitals and deaths data were used are listed in Table 5.7. Heart failure is discussed separately from the other chronic sequelae as it is an envelope condition.

Table 5.7: ABDS 2011 diseases and sequelae that use a combination of the NHMD and Western Australian linked hospitalisations and deaths data to estimate prevalence

Disease	Sequela	ABDS 2011 health state identifier[a]
Coronary heart disease	Chronic coronary heart disease	26, 27, 28, 262
	Heart failure due to coronary heart disease[b]	31, 32, 33
Stroke	Chronic stroke	34, 35, 36, 37, 38, 262
Rheumatic heart disease	Valvular diseases due to rheumatic heart disease	207
	Heart failure due to rheumatic heart disease[b]	31, 32, 33
Non-rheumatic heart disease	Valvular diseases due to non-rheumatic heart disease	207
	Heart failure due to non-rheumatic heart disease[b]	31, 32, 33
Hypertensive heart disease	Heart failure due to hypertensive heart disease[b]	31, 32, 33
Inflammatory heart disease	Heart failure due to inflammatory heart disease[b]	31, 32, 33
Cardiomyopathy	Heart failure due to cardiomyopathy[b]	31, 32, 33
Cardiovascular defects[c]	Heart failure due to congenital cardiovascular defects[b]	31, 32, 33

(a) See Appendix Table C1.

(b) Part of heart failure envelope.

(c) Included under infant and congenital conditions.

For sequelae that are considered chronic (this includes chronic coronary heart disease, chronic stroke, rheumatic heart disease, non-rheumatic valvular disease), it was assumed that people who have these diseases are hospitalised at least once within the 11 years leading up to 2011. An 11-year look-back period was used due to the available data.

Repeat hospitalisations are not discernible in national hospitalisation admission data. To adjust for repeat hospitalisations, the ratio of people alive at the reference date who had at least 1 hospital separation due to the chronic sequela to the number of separations by broad age group and sex was derived from AIHW analyses of Western Australian linked hospitalisations and deaths data (AIHW 2015c, AIHW 2015e, AIHW 2015f).

These ratios were then applied to the count of hospital separations from the NHMD, by age and sex. As the ratios were derived from linked data for only one state, it was assumed that the other states and territories have the same persons-to-separations ratio as Western Australia.

The prevalence of chronic coronary heart disease was broken down by severity using severity distributions from GBD 2013 (Burstein et al. 2015).

The prevalence of chronic stroke was broken down by severity using distributions from GBD 2013 (Burstein et al. 2015). This distribution was adjusted for age differences using the age gradient of health experienced by stroke survivors 12 months after their first stroke from the Perth Community Stroke Study 1989–1990 (Katzenellenbogen et al. 2010).

Due to a lack of robust population-based Australian data, the NZBDS was used to estimate prevalence of the sequelae listed in Table 5.8. These rates were considered appropriate for Australia in the absence of local data as they were derived from linked administrative data.

Table 5.8: ABDS 2011 diseases and sequelae that use the NZBDS prevalence rates

Disease	Sequela	ABDS 2011 health state identifier[(a)]
Atrial fibrillation and flutter	Symptomatic atrial fibrillation and flutter	207, 29
Peripheral vascular disease	Intermittent claudication due to peripheral vascular disease	30

(a) See Appendix Table C1.

Atrial fibrillation and flutter

The prevalence of all atrial fibrillation and flutter (referred to as atrial fibrillation for the rest of this section) in Australia was estimated using the non-Maori prevalence rates from the NZBDS.

The prevalence of moderate/severe atrial fibrillation and flutter was estimated by counting the number of separations with atrial fibrillation listed as the principal diagnosis in 2011 in the NHMD. It was assumed that each separation represented 1 person.

The prevalence of mild atrial fibrillation was estimated by subtracting the prevalence of moderate/severe atrial fibrillation from the overall atrial fibrillation prevalence in Australia.

Peripheral vascular disease

The prevalence of peripheral vascular disease was estimated using the non-Maori prevalence rates from the NZBDS.

Heart failure envelope

Similar to the other chronic conditions mentioned previously (such as chronic coronary heart disease and chronic stroke), the prevalence of heart failure was estimated by applying prevalence-to-separations ratios from AIHW analyses of Western Australian linked hospitalisations and deaths data to the national count of separations from the NHMD (AIHW 2015d).

As heart failure is a sequela of multiple diseases (Table 5.7), the overall prevalence of heart failure from all diseases was calculated to ensure the sum of estimates for sequelae do not exceed the total—referred to as an 'envelope'. To avoid double-counting, and adhere to mutual exclusivity for each disease, weights were created for each disease using results from

Western Australian linked data. Where heart failure was diagnosed with no other accompanying cardiovascular disease diagnosis, these were redistributed to other diseases using proportional allocation.

Heart failure has 3 severity levels: mild, moderate, severe. Severity distributions were obtained from GBD 2013 (Burstein et al. 2015).

Subnational estimates

Where prevalence was obtained from the NHMD, subnational estimates were derived directly by applying 2011 ASGS remoteness areas and 2011 SEIFA population-based Index of Relative Socio-economic Disadvantage quintiles to the Statistical Area Level 2 recorded in hospital separations data.

For atrial fibrillation and peripheral vascular disease, prevalence by state or territory, remoteness area, and socioeconomic group were obtained by applying proportions for these conditions by subnational disaggregation from 2011 separations in the NHMD.

2003 estimates

For chronic sequelae where prevalence was estimated from a combination of the NHMD and ratios and rates derived from AIHW analyses of Western Australian linked data, methods for 2003 were largely similar to the methods for 2011. However, due to a change in the diagnosis classification and the absence of available linked data before 1 July 1999, the look-back period from 2003 was limited to 4 years. To achieve comparable estimates, 2003 estimates were derived from 2005 prevalence rates (which provided more stable age-specific numbers) adjusted using a 6:11-year look-back ratio from 2011 to compensate for the shorter look-back period.

For acute coronary syndrome, acute stroke and acute inflammatory heart disease, the methods used for 2003 prevalence estimates were the same methods used for the 2011 estimates. For atrial fibrillation and peripheral vascular disease, the NZBDS prevalence rates for 2006 were applied to the 2003 population.

Indigenous estimates

The general approach and method used for national estimates were used for both 2011 and 2003 Indigenous estimates. The severity distribution used for national estimates was also used for Indigenous estimates. For diseases and sequelae where the NHMD was used to estimate point prevalence (Table 5.6), hospital separations data were adjusted for under-identification using standard adjustment factors (see Chapter 4, and Appendix tables C3 and C4).

For diseases and sequelae where a combination of ratios from Western Australian linked data and the NHMD were used to estimate prevalence (Table 5.7), Indigenous-specific ratios were obtained from the Western Australian Department of Health. However, for heart failure sequelae estimates, the national weights and redistribution proportions for prevalence was used for the Indigenous estimates.

For atrial fibrillation and flutter and peripheral vascular disease, where non-Maori prevalence rates from the NZBDS were used for the national prevalence estimates, the Maori prevalence rates were applied to 2011 and 2003 Indigenous populations to derive Indigenous prevalence estimates.

Endocrine disorders

Mortality estimates

Endocrine disorder-related deaths were assigned from the NMD as defined by the disease list (Appendix Table A2). Deaths coded to gestational diabetes (O24.4) were assigned to reproductive and maternal conditions. Deaths due to diabetic nephropathy (E10.2, E11.2, E13.2, E14.2) were assigned to kidney and urinary diseases. No deaths due to endocrine disorders were redistributed.

Morbidity estimates

Sequelae

Sequelae and health states assigned to endocrine disorders are included in Table 5.9. All diseases were assigned health loss for the entire year. Assumptions are outlined in subsections for individual diseases.

Table 5.9: Sequelae and health states for endocrine disorders

Disease	Sequela	ABDS 2011 health state identifier[(a)]
Diabetes	Amputation due to diabetes	140
	Diabetic foot ulcer	39
	Diabetic neuropathy	40
	Diagnosed diabetes	207
	Undiagnosed diabetes	262
	Vision impairment due to diabetes	114, 115, 116
Other endocrine disorders	Other endocrine disorders	. .

(a) See Appendix Table C1.

Prevalence estimation

Diabetes

Diabetes includes type 1, type 2 and other diabetes types, with the exception of gestational diabetes (included in reproductive and maternal conditions).

Undiagnosed and diagnosed diabetes

Prevalence estimates for undiagnosed and diagnosed diabetes in people aged 18 and over were derived from the biomedical component of the AHS. For those aged under 18, diagnosed diabetes prevalence estimates were obtained from the National Diabetes Registry. It was assumed there were no people with undiagnosed diabetes aged under 18.

Undiagnosed diabetes was given an asymptomatic health state, which has a disability weight of 0.

Diabetic neuropathy and diabetic foot

The prevalence estimates for diabetic neuropathy and diabetic foot were calculated using the results from phase II of the Fremantle Diabetes Study (Baba et al. 2015). Prevalence estimates by sex and age were modelled using hospitalisations data from the NHMD.

Amputation due to diabetes

The prevalence of amputation due to diabetes was estimated using the NHMD and persons-to-separations ratios derived from Western Australian linked hospitalisations and deaths data. This was used to adjust the count of separations from the NHMD to better estimate prevalence. An amputation was determined as being due to diabetes if there was a principal or additional diagnosis of diabetes accompanying that amputation hospitalisation, and it was a lower-limb amputation.

Vision impairment due to diabetes

Similar to diabetic neuropathy and diabetic foot, the prevalence estimates for vision impairment due to diabetes were calculated using results from phase II of the Fremantle Diabetes Study (unpublished data). Breakdowns by sex and age were modelled using hospitalisations data from the NHMD.

This sequela has 3 severity levels: moderate, severe and blindness. The severity distribution used for the prevalence was obtained from the NZBDS.

Other endocrine disorders

The prevalence of other endocrine disorders is the prevalence of all other endocrine disorders that are not diabetes. The YLD was estimated by applying a YLD-to-YLL ratio of diabetes to the YLL of the other endocrine disorders.

Subnational estimates

For diagnosed diabetes, subnational estimates for those aged less than 15 were derived from the National Diabetes Registry, and from the AHS for those aged over 15.

For diabetic complications (that is, diabetic neuropathy and foot, amputation due to diabetes and vision impairment due to diabetes), subnational estimates were derived by applying 2011 ASGS remoteness areas and 2011 SEIFA population-based Index of Relative Socio-economic Disadvantage quintiles to the Statistical Area Level 2 recorded in hospital separations data.

2003 estimates

The 2003 prevalence estimates for amputation due to diabetes was obtained from the same data source as the 2011 estimates (the NHMD) using the same method.

For diagnosed diabetes, since the health surveys before the AHS did not have biomedical components, the 2003 prevalence estimates were modelled using the self-reported data from the 2001, 2004–05, 2007–08 NHS and the AHS.

The overall prevalence for diabetic neuropathy, diabetic foot and vision impairment due to diabetes were obtained from the AusDiab Study (Tapp et al. 2003a; Tapp et al. 2003b). Breakdowns by sex and age were modelled using data from the NHMD.

Indigenous estimates

Indigenous prevalence for diagnosed diabetes in 2011 was estimated using data from the biomedical component of the AATSIHS 2012–13. Since earlier ABS Indigenous health surveys did not have biomedical components, 2003 Indigenous prevalence estimates were modelled using trend data published by the ABS, which were based on self-reported prevalence of diabetes in the 2001, 2004–05, 2007–08 NHS and the AHS (ABS 2014a).

The prevalence for each of the diabetic complications for Indigenous Australians (with the exception of amputation due to diabetes) were estimated using published results from Davis et al. 2012, and unpublished results from the Fremantle Diabetes Study.

Amputations due to diabetes prevalence were estimated using the NHMD. Hospital separations data were adjusted for under-identification using standard adjustment factors (see Chapter 4 and Appendix tables C3 and C4).

The residual category of other endocrine disorders was estimated using the same method as used for national estimates (by applying the YLD-to-YLL ratio for diabetes to the YLL for other endocrine disorders).

Gastrointestinal disorders

Mortality estimates

Deaths related to gastrointestinal disorders were assigned from the NMD as defined by the disease list (Appendix Table A2). Deaths coded to unspecified digestive diseases (K92) were redistributed using the indirect MCOD method (see Chapter 3) to chronic liver disease, gastroduodenal disorders and diverticulitis. Deaths coded to peritonitis (K65–K66) were redistributed proportionately to gastroduodenal disorders, hernias, pancreatitis, gallbladder and bile duct disease, paralytic ileus and intestinal obstruction without hernia, and inflammatory bowel disease.

Morbidity estimates

Sequelae

Sequela and health states assigned to gastroduodenal disorders are included in Table 5.10. Durations and assumptions are outlined in subsections for individual diseases.

Table 5.10: Sequelae, health states and durations for gastrointestinal disorders

Disease	Sequela	ABDS 2011 health state identifier[a]	Duration
Gastroduodenal disorders	Anaemia due to gastroduodenal disorder[b]	195, 196, 197	8 weeks
	Symptomatic episodes of gastroduodenal disorder	193	1 week (inflammation)
			3 weeks (ulcers)
Appendicitis	Symptomatic appendicitis requiring appendectomy	194	2 weeks
Abdominal wall hernia	Symptomatic hernia requiring repair	192	12 months
Vascular disorders of intestine	Stoma due to vascular disorder of intestine	21	12 months (permanent stoma)
			5.4 months (temporary stoma)
	Vascular disorders of the intestine	194	6 weeks
Intestinal obstruction (without hernia)	Intestinal obstruction	194	2 weeks (major surgery)
			2 days (minor surgery)

(continued)

Table 5.10 (continued): Sequelae, health states and durations for gastrointestinal disorders

Disease	Sequela	ABDS 2011 health state identifier[a]	Duration
Inflammatory bowel disease	Crohn's disease or ulcerative colitis	46	12 months
Diverticulitis	Diverticulitis	194	2 weeks (medical therapy)
			3 weeks (surgical intervention)
	Stoma due to diverticulitis	21	12 months (permanent stoma)
			5.4 months (temporary stoma)
Chronic liver disease	Decompensated cirrhosis of the liver	44	10 months (with end-stage liver disease)
			12 months (no end-stage liver disease)
	End-stage liver disease	22	2 months
	Liver transplant	42	12 months
Gallbladder and bile duct disease	Gallbladder and bile duct disease	193	6 weeks
Pancreatitis	Acute episode of pancreatitis	194	6 weeks
	Chronic pancreatitis	193	12 months
Gastro-oesophageal reflux disease	Mild symptomatic gastro-oesophageal reflux disease	262	. .
	Moderate/severe symptomatic gastro-oesophageal reflux disease	192	12 months
Functional gastrointestinal disorders	Functional heartburn due to functional gastrointestinal disorders	192, 193	12 months
	Irritable bowel syndrome	192, 193	12 months
Other gastrointestinal disorders	Other gastrointestinal disorders	. .	. .

(a) See Appendix Table C1.

(b) Part of anaemia envelope.

Prevalence estimation

The NHMD was the major data source to estimate prevalence of gastrointestinal disorders, unless otherwise stated. Separations for acute conditions were derived from the 2011 and 2003 calendar years, as applicable. The durations used for each sequela are presented in Table 5.10.

Patients hospitalised due to the specified gastrointestinal disorders experience significant health loss, especially if they undergo surgical intervention. A hospitalisation that requires surgery is considered more severe.

Gastroduodenal disorders

Gastroduodenal disorder includes duodenal and gastric ulcers (also referred to as peptic ulcer disease) as well as gastritis and duodenitis. The term 'gastritis' used here refers

specifically to abnormal inflammation in the stomach lining, and is a pathological diagnosis, not clinical.

Endoscopic diagnosis of gastroduodenal disease is generally considered an under-count of total disease as it does not account for physician-diagnosed and treated disease. Estimates for uncomplicated gastroduodenal disease (which is generally diagnosed by a physician and successfully treated without hospitalisation) were derived by applying the rate ratio of physician-diagnosed peptic ulcer disease to hospitalised incidence (Sung et al. 2009) to the incidence of complicated gastroduodenal disorders. Complicated gastroduodenal disorders (which generally results in hospitalisation and endoscopic diagnosis) and resultant anaemia, were sourced from hospital separations for gastroduodenal disease. Separate durations were applied to prevalence estimates for gastritis/duodenitis (inflammation) and gastric/duodenal ulcers (Table 5.10).

Prevalence of anaemia due to gastroduodenal disorders was sourced from the NHMD. As data in the NHMD could not be used to estimate the severity of anaemia due to gastroduodenal disease, the global severity distributions of anaemia for gastritis and peptic ulcers from GBD 2013 were used.

Appendicitis

Appendicitis is an acute condition. Cases of appendicitis—defined as hospitalised patients with a principal or additional diagnosis of appendicitis having undergone an appendectomy—were assumed to be incident cases. Duration of health loss was assumed to be 2 weeks.

Abdominal wall hernia

Incident cases of abdominal wall hernia were defined as hospitalised patients with a principal or additional diagnosis of hernia having undergone a hernia-related procedure. Duration of health loss for patients with symptomatic hernia until repair was based on NZBDS estimate of the time between presentation of symptoms, referral and surgery (NZMOH 2012, unpublished documents).

Intestinal obstruction (without hernia)

Incident cases were defined as hospitalised patients with a principal or additional diagnosis of intestinal obstruction with surgical intervention. The duration of health loss for patients with intestinal obstruction (without hernia) varied depending on the type of surgery. Duration was assumed to be 2 weeks for those undergoing major surgery (consistent with GBD 2013), and 2 days for those undergoing minor intervention based on expert advice.

Investigation of inpatient hospitals data showed that major surgery was performed in 5 times as many separations as minor surgery. This might be due to minor surgery being performed in an outpatient setting, resulting in a potential undercount of minor surgery.

Gallbladder and bile duct disease

Incident cases were defined as hospitalised patients with a principal or additional diagnosis of gallbladder and/or bile duct disease having undergone a cholecystectomy and/or incision of bile ducts. Patients admitted with diagnosis of gallbladder disease and/or cholelithiasis who did not undergo surgery have much milder symptoms which do not result in health loss for burden of disease analysis, and were not included in this analysis.

Pancreatitis

Acute cases of pancreatitis were defined as hospitalised patients with a principal diagnosis of acute pancreatitis (ICD-10-AM K85). This diagnosis code includes acute episodes within a diagnosis of chronic pancreatitis (NCCH 2010, as described in ICD-10-AM, seventh edition by Australian Coding Standard 0001).

Chronic cases were defined as hospitalised patients with a principal or additional diagnosis of chronic pancreatitis (ICD-10-AM K86.0, K86.1). Since individuals cannot be identified using national hospitalisations data, it was assumed that 1 separation was equal to 1 person. This might have resulted in an overestimation of chronic pancreatitis prevalence, which could be improved using linked hospitals data.

Vascular disorder of the intestine

Incident cases were defined as hospitalised patients with a principal diagnosis of vascular insufficiency with or without surgical intervention. Additional health loss was assigned to cases with a stoma opening procedure in either the small or large intestine.

Duration of health loss varied according to whether a stoma was permanent or temporary. It is not possible to tell from national hospitals data which of these patients' stomas were subsequently closed. Instead, overall closure rates of stomas regardless of underlying disease derived from national hospitals data were used to estimate the number of permanent stomas.

Chronic liver disease

As a progressive disease, people might experience different stages of chronic liver disease (and therefore multiple sequelae) in 1 year. The burden allocated to each individual included their most severe sequela, with the remaining time allocated to less severe sequelae (Table 5.10). For example, a person with end-stage liver disease would be allocated 2 months for this sequela. Any remaining time prior to end-stage disease would be allocated as decompensated cirrhosis.

Western Australian linked hospitalisations and deaths data were used to determine the prevalence rate of liver transplants due to chronic liver disease in that state, which was then applied to the national population, based on the assumption that the prevalence rate is the same across all states and territories.

Western Australian linked hospitalisations data were also used to estimate a persons-to-separations ratio for chronic liver disease, by stage of disease progression. These ratios were applied to national hospital separations, by broad age group, to derive national prevalence.

Chronic liver disease patients were identified as those with a principal or additional diagnosis of the condition or from procedures particular to chronic liver disease, based on expert advice.

Inflammatory bowel disease

Inflammatory bowel disease is a chronic condition predominantly comprised of 2 diseases: Crohn's disease and ulcerative colitis, with a small proportion as unclassified inflammatory bowel disease. The health state devised by GBD 2013, and applied by ABDS 2011, is inclusive of the remittent and recurring nature of the disease, surgery and any potential long-term effects such as stoma. The health loss was assumed to apply for the whole year.

Hospitalisations data were not used to estimate the prevalence of inflammatory bowel disease as it only captures patients undergoing procedures related to the condition. Instead,

estimates were based on results of the Barwon inflammatory bowel disease study by Studd (2013), which derived prevalence using hospital and gastroenterologist data. This study reported similar results to other recent relevant studies that used a similar method (Gearry et al. 2006).

Gastro-oesophageal reflux

Gastro-oesophageal reflux (which includes hiatal hernias) is largely a chronic disease treated in response to symptoms. This condition was not included in the previous ABDS 2003, or in GBD 2010 or 2013, but was included in ABDS 2011 due to the reportedly high morbidity and because it is an identified risk factor for oesophageal and junctional adenocarcinoma. The major symptoms include heartburn, acid reflux and difficulty swallowing.

No health loss is assigned to mild symptomatic gastro-oesophageal reflux as it is of short duration. It is assumed that people with moderate or severe gastro-oesophageal reflux (that is, those experiencing symptoms more than once a week) will seek medical help from a general practitioner.

Total prevalence of moderate or severe gastro-oesophageal reflux was based on the Bettering the Evaluation and Care of Health (BEACH) substudy data from 2008–09 by Harrison et al. (2013), which estimated the national prevalence of gastro-oesophageal reflux disease as 7.5%. Age and sex distributions were derived from the 2011 BEACH study and applied to the overall prevalence.

Functional gastrointestinal disorders

Functional gastrointestinal disorders have not been included in previous Australian or global burden of disease studies. Functional gastrointestinal disorders are common disorders characterised by persistent and recurring gastrointestinal symptoms. The ABDS 2011 aimed to estimate burden that caused substantial health loss from these disorders. To avoid bias and over-counting in morbidity estimates, only medically confirmed cases, or cases determined using a validated collection instrument, experiencing health loss were counted.This is best captured through Rome III criteria (Rome Foundation 2006), which impose strict criteria that must be met for functional symptoms to be classed as pathological.

There are no robust community-based data on prevalence classified by the Rome III criteria for Australia, and overseas studies based on Rome III have been based on specific populations that cannot be generalised to Australia. As a result, ABDS 2011 estimates were based on Boyce et al. (2006) which provided adult prevalence rates for specific functional gastrointestinal disorders in the Penrith region in New South Wales. This used a validated questionnaire for the Rome II criteria, which are very similar to the criteria for the 2 sequelae modelled in ABDS 2011. Estimates for children and adolescents were based on international studies by Chitkara et al. (2005) and Helgeland et al. (2009).

Distribution of the severity for each sequela were based on the European Disability Weight Study (Haagsma et al. 2015) which estimated disability weights consistent with the GBD 2010 health states and disability weights for functional heartburn, reflux and irritable bowel syndrome for use in European burden of disease studies.

Other gastrointestinal disorders

YLD was derived indirectly by applying the YLL-to-YLD ratio for all gastrointestinal disorders (except gastro-oesophageal reflux and functional gastrointestinal disorders) combined to the YLL for other gastrointestinal disorders.

Subnational estimates

Estimates derived directly from the NHMD were broken down by state/territory, and by remoteness area and socioeconomic group by applying 2011 ASGS remoteness areas and 2011 SEIFA population-based Index of Relative Socio-economic Disadvantage quintiles to the Statistical Area Level 2 recorded in hospital separations data.

For estimates based on epidemiological studies (gastro-oesophageal reflux, inflammatory bowel disease, functional gastrointestinal disorders), breakdowns were derived by applying prevalence rates to the relevant population.

2003 estimates

The same methods used for 2011 estimates were used to estimate 2003 point prevalence for each of the diseases in the gastrointestinal disorders group using 2003 hospitalisations data and populations.

Indigenous estimates

Indigenous estimates were derived using the same methods and data sources as for national estimates for both 2011 and 2003. Estimates based on hospital separations data were adjusted for under-identification using standard adjustment factors (see Chapter 4 and Appendix tables C3 and C4).

Due to lack of evidence on the rates of gastroduodenal disorders in Indigenous Australians compared with non-Indigenous Australians, the same rate ratio used to derive physician diagnosed gastroduodenal disorders for the national population (Sung et al. 2009) was also used for the Indigenous population.

Indigenous prevalence for inflammatory bowel disease, gastro-oesophageal reflux and functional gastrointestinal disorders was obtained by applying the national distribution directly to the Indigenous population for 2011 and 2003. This assumes the underlying rate is the same between the Indigenous and non-Indigenous populations, and between the 2 time points.

Hearing and vision disorders

Mortality estimates

Deaths from hearing and vision disorders were treated as implausible causes of death. Deaths in the NMD related to hearing and vision disorders were proportionally redistributed across infectious diseases, cancer, cardiovascular diseases, chronic respiratory diseases, and unintentional injuries using proportions derived from Australian mortality data.

Morbidity estimates

Sequelae

Sequelae and health states for hearing and vision disorders are listed in Table 5.11. As only permanent hearing and vision disorders are estimated, health loss is assumed to apply for the whole year.

Table 5.11: Sequelae and health states for hearing and vision disorders

Disease	Sequela	ABDS 2011 health state identifier[a]
Hearing loss	Hearing loss	103, 104, 105, 106, 108, 109, 110, 111
Other hearing and vestibular disorders	Ear pain	15
	Vertiginous symptoms due to other hearing and vestibular disorders	207
Vision loss	Vision loss due to age-related macular degeneration[b]	113, 114, 115, 116
	Vision loss due to cataract and other lens disorders[b]	113, 114, 115, 116
	Vision loss due to glaucoma[b]	113, 114, 115, 116
	Vision loss due to refractive errors[b]	113, 114, 115, 116
Other vision disorders	Vision loss due to other vision disorders[b]	114, 116, 117

(a) See Appendix Table C1.

(b) Part of vision loss envelope.

Prevalence estimation

Hearing loss

In the ABDS 2011, hearing loss refers to all clinically confirmed, chronic hearing loss, irrespective of the cause. Short-term hearing loss for otitis media is included under infectious diseases.

Data sources

Where possible, ABDS 2011 gave priority to clinically confirmed data over self-reported surveys. As there is no single source of clinically confirmed hearing loss for all age groups for Australia, the overall national prevalence of hearing loss was estimated using 3 main data sources:

- For ages 0–14, prevalence was derived from the Australian Hearing 2011 demographics report summary tables of people aged under 21 with a clinically diagnosed hearing impairment, fitted with a hearing aid (Australian Hearing 2012).
- Prevalence for people aged 15–54 was derived from the AHS 2011–12 for the number of people reporting hearing loss, and partial or complete deafness.
- For ages 55 and over, prevalence was derived from published estimates of clinically assessed hearing loss in the Blue Mountains Hearing Study (Mitchell et al. 2011).

Prevalence estimation by age and sex

Prevalence estimates in 10-year age groups by sex were derived from the AHS and Blue Mountains Hearing Study. To derive 5-year age groups, non sex-specific proportions of total hearing loss in 5-year age groups from the AHS was applied to those aged 15–54. Proportions used in Wilson et. al (1998) and trend analyses were used for ages 55 and over. Sex distribution was derived from the ABDS 2003 (Begg et al. 2007).

Prevalence by severity

Severity distributions were derived from GBD 2010 for high-income regions.

Due to limited Australian data for tinnitus prevalence by hearing severity, analyses of self-reported results from a United States National Health Interview Survey were used

(Hoffman & Reed 2004). This was favoured as prevalence was obtained specifically from hearing impaired individuals. As this was a self-reported study, hearing levels were not clinically assessed. To determine severity, the Gallaudet Hearing Scale (used in the survey) was mapped to the GBD 2010 lay descriptions for each health state. The severity distribution for tinnitus is in Appendix Table D4.

The tinnitus estimates were subtracted from the total hearing loss estimates, to calculate estimates for hearing impairment without tinnitus.

Other hearing and vestibular disorders

Other hearing disorders were also calculated using the AHS 2011–12. It was assumed that Meniere's disease would result in vertigo, and conditions classified as other ear diseases would result in ear pain.

Estimates of Meniere's disease by sex were obtained from the AHS—age estimates were not available due to high relative standard errors. To obtain age estimates, the age distribution of hospitalisations of Meniere's disease in 2011 by age and sex in the NHMD were applied to the total AHS count.

To estimate burden from ear pain due to other hearing and vestibular disorders, estimates were obtained from the AHS by age and sex. Age groups which had high relative standard errors (0–14 and 75 and over) were estimated using population sex-specific proportions to obtain 5-year age groups.

Vision loss

Vision loss estimates comprise vision loss due to refractive error, cataract and other lens disorders, glaucoma, age-related macular degeneration and other vision disorders. Other diseases that include vision loss as sequelae include diabetic retinopathy and trachoma. Vision loss due to injuries is included in other vision disorders.

Prevalence estimation of the vision loss envelope

As vision loss is a sequela of multiple conditions across ABDS, the overall prevalence of vision loss was calculated to ensure the sum of estimates for sequelae do not exceed the total vision loss in the population.

Total vision loss in the populations was estimated from projections to 2010 in the Melbourne Visual Impairment Project (Taylor 2005; Weih et al. 2000), which provided estimates of vision loss by various causes. Vision loss due to refractive error, cataract and other lens disorders, glaucoma and age-related macular degeneration were derived directly from this source as described for each cause. Estimates for other vision disorders could not be directly derived as this category in the Melbourne Visual Impairment Project also included trachoma.

Trachoma was calculated separately and estimated in the Indigenous population only (see separate section on trachoma in Indigenous estimates). To ensure mutual exclusivity, prevalence estimates for vision loss due to trachoma were removed from the national prevalence estimates of other vision disorders.

While the Melbourne Visual Impairment Project provided estimates for diabetic retinopathy, estimates for this condition were estimated separately (see methods for endocrine disorders). This was because more recent measured data were available.

As vision loss from diabetic retinopathy was estimated separately, the sum and proportion of all diseases included in the vision loss envelope were then compared with the 2010

projections estimates of vision loss described earlier to ensure estimates were comparable with published results. The final proportions of vision loss due to specific diseases are shown in Table 5.12.

Table 5.12: Proportion of vision loss in ABDS 2011, by cause

Cause of vision loss	Proportion of vision loss envelope (%)
Age-related macular degeneration	12.1
Glaucoma	5.5
Cataract and other lens disorders	15.2
Refractive error	51.7
Other vision disorders	9.9
Trachoma	0.1
Diabetic retinopathy	5.5
Total	**100.0**

Refractive error and cataract and other lens disorders

The prevalence rate of uncorrected refractive error and cataract and other lens disorders, by 10-year age groups from age 40, was obtained from the Melbourne Visual Impairment Project. Estimates were modelled in 5-year age groups using proportions from the ABDS 2003 (Begg et al. 2007).

Prevalence was estimated from age 40 and over for cataract and other lens disorders only, due to the nature of this condition. Due to limited information on refractive error prevalence in people aged under 60, prevalence rates from the ABDS 2003 were used. These rates were originally obtained from estimates from Weih et al. (2000) and modelled using DISMOD II. The sex distribution was based on the Australian population, assuming no sex differentiation in these conditions.

Severity distributions for refractive error was obtained from the Melbourne Visual Impairment Project and modelled to account for inconsistencies. It was assumed there was no differentiation by sex, and that refractive error would not be the primary cause of blindness (< 3/60) in individuals with severe visual impairment, based on expert advice (see Appendix Table D5).

Severity distributions for vision impairment due to cataract were obtained from published Melbourne Visual Impairment Project data analyses. The average population-weighted prevalence estimates by severity across each age group from the Melbourne Visual Impairment Project estimates were applied to all age groups (see Appendix Table D5).

Glaucoma

Prevalence for glaucoma was only estimated from age 40, as primary open angle glaucoma is rare in people aged under 40.

The prevalence rate of vision impairment due to glaucoma for people aged 60–89 in 10-year age groups was obtained from the Melbourne Visual Impairment Project. Extrapolation based on the exponential curve was used to determine rates in younger age groups. Trend analysis was used to determine prevalence rates in 5-year age groups.

Sex distribution was based on the Australian population, assuming no sex differentiation in glaucoma. The severity distribution of glaucoma, by age, was derived from published

Melbourne Visual Impairment Project based estimates (Weih et al. 2000). Due to sampling artefacts in the study, proportions were considered inconsistent with the disease model of glaucoma severity by age. Instead, estimates by age were pooled, and the pooled severity distribution used across all age groups (see Appendix Table D5).

Age-related macular degeneration

Prevalence of age-related macular degeneration was estimated from age 50 and over only, due to the nature of this condition.

The prevalence rate of age-related macular degeneration for people aged 65–89 was obtained from the Melbourne Visual Impairment Project. Prevalence rates in younger age groups (that is, 50–64) was obtained through extrapolation and trend analyses. Proportions in 5-year age groups were obtained from estimates in the Access Economics vision loss reports of prevalence of bilateral age-related macular degeneration in the better eye, based on prevalence derived from the Blue Mountains Eye Study (Deloitte Access Economics 2011).

Sex distribution was based on the Australian population, assuming no sex differentiation in age-related macular degeneration. Severity distributions were obtained from published Melbourne Visual Impairment Project data analyses. Based on expert advice, it was assumed the ratio of clinical age-related macular degeneration-to-vision loss due to age-related macular degeneration was the same as the mild vision loss-to-blindness due to age-related macular degeneration ratio. This also assumed the same progression rate through each severity.

Other vision disorders

Vision loss due to other vision disorders was based on the proportions of vision loss caused by residual disorders described in *Vision loss in Australia* (Taylor et al. 2005). The prevalence of vision loss due to trachoma was subtracted from the estimate to avoid double-counting.

The age and sex distribution from the AHS 2011–12 for visual disturbances and blindness was then applied to the overall estimate. Estimates for people aged 0–9 and 90 and over were attained using population proportions.

Estimates for blindness were based on the proportion in *Vision loss in Australia*, adjusted for trachoma and diabetic retinopathy. Experts advised that most of these are probably due to trauma.

Estimates for moderate and near sighted vision loss were based on the assumption that the ratio of mild-to-moderate in Weih et al. (2000) is the same for near vision-to-moderate vision loss for other vision disorders.

Subnational estimates

Subnational estimates were apportioned from the national estimates based on sex-specific ratios from the AHS 2011–12. Age- and sex-specific proportions were not used due to a high degree of uncertainty in this data source, with relative standard errors over 50% for these estimates.

2003 estimates

Due to limitations in reliable data, the same severity distribution and proportions of individuals with hearing loss and vision loss used in 2011 were used for national 2003 estimates.

Indigenous estimates

Hearing loss

Indigenous estimates for hearing loss for 2011 were directly obtained from the AATSIHS 2011–12. Age distributions were modelled for people aged 80 and over to estimate prevalence in older age groups. The severity distribution used in GBD 2013 for high-income countries (used for national estimates) was adjusted based on 2012–13 data from the Northern Territory Hearing Health Outreach Services, and differences in the age and sex distribution in the Indigenous populations. The adjusted severity distribution was applied to the derived prevalence estimates to attain hearing loss in the Indigenous population by age, sex and severity.

Indigenous estimates for hearing loss in 2003 were obtained from the National Aboriginal and Torres Strait Islander Health Survey (NATSIHS) 2004–05. The derived prevalence rate was applied to the 2003 Indigenous population to estimate prevalence of hearing loss in 2003.

Vision loss

Estimates for vision loss in the Indigenous population were estimated from published results from the National Indigenous Eye Health Survey 2008 for both 2011 and 2003. These estimates provide robust data for vision loss specific to the Indigenous population. Published estimates were reported by state, remoteness area and broad age categories, and apportioned into 5-year age groups and sex, either using Indigenous population or national age and sex distributions for each disease. It was assumed the prevalence rate in 2008 was applicable to both 2011 and 2003. This assumes changes to disease prevalence are due to population growth and ageing only.

Broad severity distributions were obtained from the National Indigenous Eye Health Survey and applied to the Indigenous prevalence estimates by age and sex. The total number of people with vision loss by disease was compared with the proportion of total vision loss in the Indigenous population by disease, to ensure the derived prevalence estimates were consistent with that expected in the Indigenous population.

Trachoma

Vision loss caused by trachoma was measured in the Indigenous population only. The proportion of total vision loss due to trachoma by broad severity groups was obtained from the National Indigenous Eye Health Survey 2008 for both 2011 and 2003. The age distribution of trachomatous scarring prevalence was applied to low vision estimates, and the prevalence of trichiasis was applied to blindness.

The progression from mild vision loss to blindness occurs quickly in individuals with persistent trachoma infection, so expert advice on the appropriate severity distribution was sought on modelled estimates. In the absence of other data, the ratio of moderate-to-severe vision loss by age from the Indigenous ABDS 2003 (Begg et al. 2007) was applied to the broad severity categories.

Indigenous estimates for all causes of visions loss used the same rates or proportions as for 2011, applied to the 2003 population.

Infant and congenital conditions

Mortality estimates

Deaths related to infant and congenital conditions were assigned from the National Mortality Database as defined by the disease list. Deaths due to congenital malformations with ICD-10 codes Q10–Q18, Q38.1, Q54, Q65–Q74, Q82–Q84, Q89.9, Q99.9 were considered implausible causes of death, and were redistributed proportionally to target diseases that were non-communicable (that is, excluding infections, cancer and injuries) based on Australian mortality data.

Morbidity estimates

The sequelae and health states assigned to infant and congenital disorders are listed in Table 5.13. The majority of sequelae are chronic, so health loss was assumed to apply for the whole year. Durations for acute sequelae are described in the relevant sections.

Table 5.13: Sequelae and health states for infant and congenital conditions

Disease	Sequela	ABDS 2011 health state identifier[a]
Pre-term birth and low birthweight complications	Acute complications due to pre-term and low birthweight complications	54
	Neurodevelopment impairment due to pre-term and low birthweight complications[b]	213, 214, 215, 216, 217, 218
Birth trauma and asphyxia	Neurodevelopment impairment due to birth trauma and asphyxia[b]	216, 217, 218
Cerebral palsy	Neurodevelopment impairment due to cerebral palsy	213, 214, 215
Neonatal infections	Acute complications due to neonatal infections	3
Other disorders of infancy	Other disorders of infancy	54
Neural tube defects	Incontinence due to neural tube defects	48
	Motor impairment due to neural tube defects	213, 214, 215
	Neurodevelopment impairment due to neural tube defects[b]	218
Brain malformations	Neurodevelopment impairment due to brain malformations[b]	216, 217, 218
Cardiovascular defects	Congenital cardiovascular defects untreated	33
	Heart failure due to congenital cardiovascular defects[c]	31, 32, 33
Cleft lip and/or palate	Disfigurement due to cleft lip/palate	201, 202
	Speech problems due to cleft lip/palate	212
Gastrointestinal malformations	Acute complications due to gastrointestinal malformations	194
	Incontinence due to anorectal atresia	48
Urogenital malformations	Urogenital malformations	192, 262
Down syndrome	Intellectual disability due to Down syndrome[b]	99, 100, 101, 102,243
Other chromosomal abnormalities	Intellectual disability due to chromosomal abnormalities[b]	99, 100, 101, 102, 243
Other congenital conditions	Other congenital conditions	YLL:YLD ratio

(a) See Appendix Table C1.

(b) Part of intellectual disability envelope.

(c) Part of heart failure envelope.

Prevalence estimation

The key data sources to estimate prevalence of infant and congenital conditions are listed in Table 5.14.

Table 5.14: Key data sources for infant and congenital conditions

Data source	Related diseases
National Hospital Morbidity Database	Neonatal infections, other disorders of infancy, pre-term low birthweight complications (acute)
National Mortality Database	Cerebral palsy
Western Australian Registry of Developmental Anomalies (WARDA)	Neural tube defects (acute), cardiovascular defects (acute), gastrointestinal malformations (acute), urogenital malformations (acute)
Cerebral Palsy Register	Cerebral palsy
Intellectual Disability Exploring Answers (IDEA) database	Intellectual disability envelope conditions
National Perinatal Data Collection	Pre-term low birthweight complications
DISMOD II	Neural tube defects, gastrointestinal malformations

Western Australian Registry of Developmental Anomalies

For congenital abnormalities, prevalent cases for the acute sequelae were obtained from the Western Australian Registry of Developmental Anomalies (WARDA). The live birth prevalence rate for Western Australia was estimated by dividing the number of cases by Western Australia live births at 30 June 2011. This rate was then applied to the Australian live births in 2011 to derive national estimates.

DISMOD II

Some congenital abnormalities used DISMOD II to obtain point prevalence for long-term sequelae. This included neural tube defects and gastrointestinal malformations. Parameters were used as inputs to DISMOD were:

- an incidence rate derived from WARDA for live births
- an assumed remission rate of 0
- a case fatality rate obtained from previous burden of disease studies or derived from incidence and the NMD.

Intellectual disability in the ABDS 2011

Intellectual disability (also referred to as cognitive impairment) is a sequela of multiple conditions in the infant and congenital disease group, including for:

- pre-term birth and low birthweight complications
- birth trauma and asphyxia
- brain malformations (including fetal alcohol syndrome)
- neural tube defects
- Down syndrome
- other chromosomal abnormalities
- other congenital abnormalities.

Details on the methods for prevalence and severity distribution of the intellectual disability envelope are provided in 'Mental and substance use disorders' in this chapter.

Pre-term birth and low birthweight complications

Prevalence of neurodevelopmental impairment due to pre-term birth and low birthweight complications was derived from the intellectual disability envelope. For each severity, 50% of cases were modelled with motor impairment and 50% of cases with motor and cognitive impairment, based on assumptions by Blencowe et al. (2013).

All incident cases of pre-term births and low birthweight were allocated acute complications. Pre-term births were determined from the National Perinatal Data Collection 2011, inflated to account for births due to low birthweight only. These inflation factors were derived from the NHMD.

The duration of acute complications was derived from the median length of stay for level III neonatal intensive care units for Australian and New Zealand Neonatal Network registrants in 2011, by gestational age (Chow 2013). The durations were:

- extremely pre-term: 97 days
- very pre-term: 50 days
- late pre-term: 22 days.

Birth trauma and asphyxia

Prevalence of neurodevelopmental impairment due to birth trauma and asphyxia was derived from the intellectual disability envelope. The severity distribution for birth trauma and asphyxia was derived from NHMD 2013–14 using specific severity codes for hypoxic ischaemic encephalopathy of newborn (P91.61–P91.63).

Cerebral palsy

The key data source for cerebral palsy was the Australian Cerebral Palsy Register Report 2013 (Cerebral Palsy Alliance 2013). Incidence and mortality from cerebral palsy 1913–2011 was used to derive prevalence. Incidence and mortality from cerebral palsy 1913–2011 was estimated from the Australian Cerebral Palsy Register report and the NMD, respectively. Prevalence was adjusted for standard background mortality using the Australian life table (ABS 2012b).

An Australian-specific severity distribution derived from the Gross Motor Function Classification System was applied to the estimates (Appendix Table D6).

Overlaps with other diseases

Cerebral palsy can be caused by a number of related conditions. Health loss due to infection, traumatic brain injuries and other cerebral accidents caused by cerebral palsy acquired post-neonatally were captured under other disease groups (for example, injuries, infections).

The total prevalence of cerebral palsy from neonatal conditions was first determined. To ensure the total health loss due to cerebral palsy was neither over- nor under-estimated, the proportion of cerebral palsy caused by other conditions in the infant and congenital disease group (birth trauma and asphyxia and pre-term and low birthweight complications) was excluded after estimation of the YLD. Half (50%) of YLD for neonatally acquired cerebral palsy was distributed to birth trauma and asphyxia (10%) and pre-term and low birthweight complications (40%). The proportional split was determined from McIntyre et al. (2013),

Badawi et al. (2005) and NZBDS (NZMOH 2012). The remaining 50% of YLD was assigned to cerebral palsy.

Neonatal infections and other disorders of infancy

Health loss from neonatal infections and other disorders of infancy is short term. Prevalence estimates for neonatal infections and other disorders of infancy were based on hospital separations from the NHMD 2011 where these diseases were listed as either the principal or additional diagnosis. It was assumed that cases lasted on average 4 weeks.

Neural tube defects

Prevalence of neural tube defects in babies less than 1 year was sourced directly from the live birth prevalence rate derived from WARDA. DISMOD II was used to model prevalence for those aged over 1 using incidence, remission and case fatality inputs. Prevalence estimates were then distributed into different health states using proportions from Hunt & Oakeshott (2003) (Appendix Table D7). The life expectancy for people with moderate or severe neural tube defects was assumed to be about 40 years (Oakeshott et al. 2010).

Brain malformations

Prevalence of neurodevelopmental impairment due to brain malformations was derived from the intellectual disability envelope. For moderate and severe brain malformations, prevalence rates were modelled to account for a life expectancy of about 40 years.

Congenital cardiovascular defects

Congenital cardiovascular defects were modelled to include an acute sequela (cardiovascular defects prior to surgery) with a duration of 1 year, and a chronic sequela (heart failure due to congenital cardiovascular defects). Heart failure due to congenital cardiovascular defects was modelled under the heart failure envelope (see 'Cardiovascular diseases' in this chapter).

Cleft lip and/or palate

It was assumed that all children born in Australia with cleft lip and/or palate are treated surgically (or at least have commenced a first surgical intervention) within the first year of life (Royal Children's Hospital Melbourne 2010). As such, it was assumed all cases have disfigurement (level 2) until surgery at about 9 months. Post-surgical treatment, it was estimated that 5% of cases continue to have moderate disfigurement (level 2) and 10% mild disfigurement (level 1). It was assumed that 85% cases have no residual disability (GBD Collaborators 2013).

Post-surgery, it was estimated that 19% of cases aged 1–9, and 4% of cases aged 10–14 will experience speech problems, and these are largely resolved by age 15 (Sell et al. 2009).

Live birth prevalence rates of cleft lip and/or palate were derived from published WARDA data for 1980–2011. People born with cleft lip and/or palate were assumed to have the same life expectancy as the general population. Therefore, as an enduring condition, the prevalence rate for a given age in 2011 was obtained from live birth prevalence rate during the relevant birth year. Where WARDA data were unavailable for an age cohort, the prevalence rate from the closest reference year was used.

Gastrointestinal malformations

Gastrointestinal malformations include various congenital anomalies, but anorectal and oesophageal atresia were chosen as the primary sequel for inclusion. An untreated

(pre-surgical) health state in the first year of life was assumed to be equivalent to the GBD 2010 health state: severe abdominopelvic problems.

DISMOD II was used to model prevalence for those aged over 1 using incidence, remission and case fatality inputs. It was assumed 62.5% of people with anorectal malformations experience faecal incontinence (Peña & Hong 2000). The proportion of anorectal malformations was derived from WARDA data published in the annual report of the International Clearinghouse for Birth Defects Surveillance and Research for 2011 (ICBDSR 2013). For the first year of life, it was assumed faecal incontinence only occurred for 6 months after surgical intervention.

Urogenital malformations

The sequelae for urogenital malformations included hypospadias, undescended testicles, and other urogenital malformations.

Children with hypospadias often have surgery at 6–18 months, after which the associated health burden is negligible. As such, hypospadias was assumed to be asymptomatic. For other urogenital malformations, it was proposed the health burden is equivalent to the health state for mild abdominopelvic pain. The proportion of hypospadias and undescended testicles was derived from the NHMD 2011, and it was assumed 30% of other urogenital malformations were symptomatic (mild abdominopelvic pain).

It was assumed people born with urogenital malformations have the same life expectancy as the general population and zero remission; therefore, the live birth prevalence rate (from WARDA) was held constant and applied to the national population by sex and age groups.

Down syndrome

The major sequela for Down syndrome was intellectual disability, which was modelled as part of the intellectual disability envelope. Due to the reduced life expectancy in people with Down syndrome (Day et al. 2005; Glasson et al. 2003), prevalence rates were modelled to account for a life expectancy of about 70 years.

Other chromosomal abnormalities

The major long-term disabling sequela for other chromosomal abnormalities was intellectual disability, which was modelled as part of the intellectual disability envelope.

Other congenital conditions

A YLL-to-YLD ratio was derived using the combined YLL and YLD from cardiovascular defects, cleft lip and/or palates, gastrointestinal malformations and urogenital malformations. This ensured there was no overlap with the health loss captured for conditions under the intellectual disability envelope. This ratio was applied to the fatal burden of other congenital conditions to derive the corresponding YLD.

Subnational estimates

National estimates were apportioned into each remoteness area, socioeconomic group and state/territory based on proportions of the respective disease obtained from the NHMD 2011 data.

2003 estimates

Estimates for infant and congenital conditions used a similar method, with data sourced for 2003.

Indigenous estimates

Where possible, prevalence estimates for the Indigenous population for 2011 and 2003 were obtained from the same data sources as used for national prevalence estimates, using the same methods. Exceptions to this are described elsewhere.

Indigenous estimates based on hospital separations data (that is, neonatal infections, other disorders of infancy, acute pre-term low birthweight complications) were adjusted for under-identification using standard adjustment factors (see Chapter 4 and Appendix tables C3 and C4).

For congenital abnormalities, Indigenous-to-total population rate ratios were derived from WARDA (for birth anomalies, such as neural tube defects) or the NHMD (where surgical interventions occurred, such as for cleft lip/palate) applied to national prevalence rates.

The Australian Cerebral Palsy Register (Cerebral Palsy Alliance 2013) reported 3.5% of people with cerebral palsy were born from mothers of Aboriginal and/or Torres Strait Islander status. This proportion was applied to national estimates to derive the Indigenous prevalence for cerebral palsy for both 2011 and 2003.

For conditions included in the intellectual disability envelope, Indigenous prevalence estimates were calculated using Indigenous-to-non-Indigenous rate ratios from the Western Australian Intellectual Disability Exploring Answers (IDEA) database for the most recent period (see Indigenous estimates section 'Mental and substance use disorders' in this chapter).

Infectious diseases

Mortality estimates

Deaths from infectious diseases were assigned from the NMD as defined by the disease list (Appendix Table A2). A small number of ICD–10 codes relating to infectious diseases were assigned to other disease groups, including: some infections of the skin and subcutaneous tissue allocated to skin conditions; infections of the amniotic sac and membranes allocated to reproductive and maternal conditions; and some neonatal infections allocated to infant and congenital conditions.

Septicaemia (A40, excluding A40.3, and A41) was the largest cause of death requiring redistribution within the infections group, accounting for around 1,200 deaths. While septicaemia is a clearly defined clinical entity, other underlying causes would have led to the chain of events culminating in the death (Naghavi et al. 2010). Deaths coded to septicaemia were redistributed using the indirect MCOD method.

Morbidity estimates

Sequelae

A list of sequelae and health states assigned to each infectious disease is included in Appendix Table D8. As infectious disease data are generally measured in terms of incident cases, prevalence estimates were produced by applying a duration of health loss (also provided in Appendix Table D8). These durations were sourced from previous Australian or global burden of disease studies.

Prevalence estimation

The primary data sources used for infectious diseases are listed in Table 5.15. These data sources were often supplemented by a secondary data source (particularly the NHMD) to help estimate either the severity distribution or the age and sex distribution within each disease.

Table 5.15: Key data sources for infectious diseases

Data source	Disease
National Notifiable Diseases Surveillance System (NNDSS)	Tuberculosis, syphilis, chlamydia, gonorrhoea, hepatitis A, diphtheria, pertussis, tetanus, measles, rubella, Haemophilus influenza type-B (Hib), pneumococcal disease, meningococcal disease, dengue, Ross River virus, Barmah Forest virus, malaria
Bettering the Evaluation and Care of Health (BEACH)	Upper respiratory infections, otitis media (acute), varicella-zoster, lower respiratory infections, influenza, other sexually transmitted infections
National Hospital Morbidity Database (NHMD)	Other meningitis and encephalitis, otitis media (chronic)
Foodborne illness in Australia: annual incidence circa 2010 (Kirk et al. 2014)	Campylobacteriosis, salmonellosis, rotavirus, other gastrointestinal infections
Modelled prevalence estimates produced by the Kirby Institute (University of New South Wales)	HIV/AIDS, hepatitis B, hepatitis C

The methods for prevalence estimation are presented here by primary data source, rather than by disease as in other sections, due to the large number of individual diseases being estimated and similarities in approaches.

National Notifiable Diseases Surveillance System

Notifications to the National Notifiable Diseases Surveillance System (NNDSS) were considered an accurate estimate of the incidence of tuberculosis, diphtheria, tetanus, measles, rubella, Haemophilus influenzae type-b (Hib), pneumococcal disease, meningococcal disease, dengue, Ross River virus, Barmah Forest virus and malaria. However, over-diagnosis and possible false positive diagnostic test results for Ross River virus and Barmah Forest virus means notifications might result in an overestimate in burden in some years. The case definitions for these 2 infections were revised, effective from 1 January 2016, so future studies should take this into consideration (Knope et al. 2016).

For other conditions, disease notifications represent only a proportion of the total incidence (referred to as the 'notified fraction'). The notified fraction varies by disease, jurisdiction and period due to the influence of several factors, including the pathogenicity of the organism, disease severity, changing case definitions, specificity and sensitivity of diagnostic tests, and differences in testing and reporting practices between primary care practices, laboratories and hospitals. As a result, notifications for pertussis and hepatitis A were inflated in an attempt to estimate the true community incidence. These adjustment factors were based on a variety of evidence, including enhanced surveillance programmes, outbreak investigation and expert advice.

Enhanced disease surveillance and screening programmes in target populations (particularly for sexually transmitted diseases) might result in the notification of asymptomatic infection. For burden of disease purposes, individuals who are asymptomatic are assumed to experience no health loss, and are excluded from analysis. Therefore, published data from

state annual surveillance reports (SA Health 2012) and enhanced surveillance studies (Fagan et al. 2013; Ressler et al. 2013) were used to determine sex-specific adjustment factors to correct for asymptomatic notification of chlamydia and gonorrhoea. State annual surveillance reports were similarly used to determine to distribute national syphilis notifications, by stage of disease.

Bettering the Evaluation and Care of Health

Data from Bettering the Evaluation and Care of Health (BEACH) were used for infectious diseases where no other representative data source was available (including upper respiratory infections, lower respiratory infections, influenza, acute otitis media, varicella-zoster and other sexually transmitted infections). The number of BEACH general practitioner (GP) encounters observed by age and sex in 2010–11 was compared with the corresponding number of national GP consultations (based on Medicare Benefits Scheme claims).

From these data, inflation factors were calculated for each age and sex group. This factor was then applied to the weighted number of GP consultations with specific International Classification of Primary Care Version 2+ (ICPC–2+) diagnosis codes to estimate an expected number of national GP consultations for a particular disease. The number of extrapolated number of national consultations was used to estimate disease incidence, based on the assumption that 1 GP episode represents 1 incident case.

National Hospital Morbidity Database (NHMD)

The NHMD was used to estimate the incidence of other meningitis and encephalitis and chronic otitis media (based on myringotomy with tube insertion procedures).

Across most infectious diseases included in the study, the NHMD was also used to estimate the number of severe cases. Hospital separations were adjusted using age-specific persons-to-separations rate ratios calculated from Western Australian linked data to correct for multiple hospital separations for a single person.

Other published data sources

Published estimates were used for the remaining infectious diseases, including:

- the incidence of gastrointestinal infectious diseases in 2010 (Kirk et al. 2014)
- the number of individuals living with HIV/AIDS by age and sex (Kirby Institute 2012; Jansson et al. 2010)
- the annual incidence of hepatitis B and C infections (Kirby Institute 2013).

In addition, prevalence estimates for infertility were derived as part of the reproductive and maternal conditions disease group, and vision loss from trachoma were estimated as part of the hearing and vision loss disease group.

2003 estimates

Prevalence estimates for 2003 were calculated from the same data source and using the same method as for 2011.

Subnational estimates

Prevalence estimates by state and territory were calculated from proportions obtained from the NNDSS (when notifications were considered a good estimate of incidence) or the NHMD. Estimates by remoteness area and socioeconomic group were calculated by applying proportions from the NHMD to national estimates.

Indigenous estimates

Where possible, prevalence estimates for the Indigenous population were obtained from the same data source as used for national prevalence estimates. Where this was not possible, indirect methods were used by applying rate ratios from a secondary data source to the national prevalence estimates to derive Indigenous prevalence. Table 5.16 lists the primary data source or indirect method used for Indigenous prevalence estimates for each of the infectious diseases. Similar to the approach used for national estimates, these data sources were often supplemented by a secondary data source (particularly the NHMD) to help estimate either the severity distribution or the age and sex distribution within each disease.

The same durations were applied for Indigenous estimates as used for national estimates.

Table 5.16: Key data sources for Indigenous prevalence estimates of infectious diseases

Data source	Disease
National Notifiable Diseases Surveillance System (NNDSS)	Tuberculosis, syphilis, chlamydia, gonorrhoea, hepatitis A, diphtheria, tetanus, measles, rubella, Haemophilus influenza type-B (Hib), pneumococcal disease, meningococcal disease, dengue, Ross River virus, Barmah Forest virus, malaria, hepatitis A (moderate-mild)
National Hospital Morbidity Database (NHMD)	Other meningitis and encephalitis, campylobacteriosis (severe), salmonellosis (severe), other gastrointestinal infections (severe), hepatitis A (severe), pertussis (severe)
Modelled prevalence estimates produced by the Kirby Institute (University of New South Wales)	HIV/AIDS
Indirect methods: rate ratios from NNDSS	Varicella–zoster, influenza, other sexually transmitted infections, hepatitis c
Indirect methods: rate ratios from hospitalisations data	Rotavirus, upper respiratory infections, lower respiratory infections
Indirect methods: rate ratios from NNDSS and hospitalisations	Hepatitis B
Indirect methods: rate ratios from AATSIHS and AHS	Otitis media

National Notifiable Diseases Surveillance System (NNDSS)

The completeness of recording of Indigenous status on infectious disease notifications varies by year, jurisdiction and disease. Work to improve the Indigenous identification in notifiable communicable disease registries is ongoing. Consistent with current AIHW practices in reporting Indigenous communicable disease notification rates, only jurisdictions with completeness of Indigenous status exceeding 50% were included in the analysis (AHMAC 2012). Rates produced from these jurisdictions were applied nationally, with consideration of their likely representativeness of the national disease burden.

Table 5.17 outlines the states and territories used in estimating Indigenous prevalence from the NNDSS. Indigenous estimates were based on the average of 3 years of notifications (2010–2012).

Table 5.17: States and territories included in analysis of Indigenous prevalence estimates sourced from the NNDSS

Disease	State/territory
Tuberculosis, hepatitis B, meningococcal, Haemophilus influenza type b (Hib), pneumococcal, malaria, measles, rubella, hepatitis A	All states/territories
Chlamydia	Vic, Qld, WA, SA, Tas, NT
Gonococcal infection	Vic, Qld, WA, SA, Tas, ACT, NT
Ross river virus	WA, SA, ACT, NT
Dengue	NSW, Vic, WA, SA, ACT, NT
Barmah forest virus	WA, SA, Tas, ACT, NT

For tetanus and diphtheria, no Indigenous notifications were reported in the 3-year period 2010–2012, and therefore zero prevalence was assumed.

For measles, a hospitalisation-to-notification ratio was applied to estimate the number of severe cases relative to the number of moderate cases (the latter based on NNDSS notifications).

National Hospital Morbidity Database

Indigenous estimates based on hospitalisation data (other meningitis and encephalitis, chronic otitis media) were adjusted for Indigenous under-identification using standard adjustment factors outlined in Chapter 4 (see also Appendix tables C3 and C4).

Indigenous estimates for the gastrointestinal infections (campylobacteriosis, salmonellosis, other gastrointestinal infections) and pertussis were based on adjusted hospitalisations for severe cases, and then the same relative proportions of mild-to-severe and moderate-to-severe as used for national estimates were applied to derive Indigenous prevalence for mild and moderate cases.

Kirby institute reports

Indigenous estimates for HIV/AIDS were based on prevalence estimates by the Kirby Institute (2014) reporting for 2013. Age distribution was based on the national age distribution from the same publication. Sequelae were distributed in the same proportions as nationally.

Indirect methods

The BEACH sample has not been designed to produce statistically significant results for Indigenous Australians, and Indigenous identification is incomplete, as confirmed in a BEACH sub-study (Deeble et al. 2008). Given this uncertainty, producing Indigenous estimates from BEACH data was not considered reliable. Instead, age-specific Indigenous-to-national rate ratios were used to calculate prevalence. These rate ratios were calculated from:

- disease notifications (for varicella-zoster, influenza—moderate cases)
- Indigenous hospitalisations adjusted for under-identification (for lower respiratory infections, influenza—severe cases, upper respiratory infections and other sexually transmitted infections)
- the AATSIHS 2012–13 and AHS 2011–13 (for acute otitis media).

For hepatitis B, Indigenous estimates were based on rate ratios calculated from notifications for 2009–11 for Western Australia, South Australia, Tasmania, the Australian Capital Territory, and the Northern Territory, and hospitalisations with a principal diagnosis of hepatitis B (B16, B17.0).

For Hepatitis C, Indigenous estimates were based on Indigenous-to-non-Indigenous notification rate ratios for 2009–11 (Western Australia, South Australia, Tasmania, and the Northern Territory only).

Injuries

Injury perspectives for burden of disease analysis

Burden of disease studies traditionally report injury burden according to specific sequelae (or functional limitations) of external causes. The resulting functional limitations from injury (or health states) are determined by the nature of the injury.

In the ABDS 2011, injury burden was reported using 2 perspectives—the external cause that resulted in the injury (for example, a road traffic accident, falls or poisoning), and the nature of the injury (for example, hip fracture, traumatic brain injury or poisoning). The total burden from injury is the same for each reporting perspective, and each perspective is equally comparable with the estimates for other disease groups in this study. To help report both perspectives, the fatal burden was mapped to the nature of injury causes, and the non-fatal burden was mapped to external causes.

Both perspectives are shown in Table 5.18. The ICD-10 codes used to identify external causes are and nature of injury are shown in Appendix Table A2.

Table 5.18: ABDS 2011 disease list for injuries, by nature and external cause of injury

Nature of injury	External cause
Traumatic brain injury	Road traffic injuries - motorcyclists
Spinal cord injury	Road traffic injuries - motor vehicle occupants
Internal and crush injury	Other road traffic injuries
Poisoning	Other land transport injuries
Drowning and submersion injuries	Poisoning
Hip fracture	Falls
Tibia and ankle fracture	Fire, burns and scalds
Humerus fracture	Drowning
Other fractures	Other unintentional injuries
Dislocations	Suicide and self-inflicted injuries
Soft tissue injuries	Homicide and violence
Burn injuries	All other external causes of injury
Other injuries	

Mortality estimates

Injury deaths were identified from the NMD as those defined by the external cause of injury from ICD-10 Chapter XX 'External causes of morbidity and mortality' in the range V01–Y98.

Redistribution

Deaths coded to event of undetermined intent (Y10–Y34) were redistributed based on direct evidence from the ABS revisions process. Death records coded to exposure to unspecified factor (X59) were redistributed across injury deaths using proportional allocation.

The remaining non-specific injury deaths (Y87.2 sequelae of events of undetermined intent; Y89.9 sequelae of unspecified external cause; Y90 evidence of alcohol involvement determined by blood alcohol level; Y91 evidence of alcohol involvement determined by level of intoxication; Y95 nosocomial condition; and Y96 work-related condition) were redistributed across all causes using proportional allocation. Proportional allocation algorithms aligned with the deaths data for each reference period.

Conversion to nature of injury

YLL were also estimated for deaths by the nature of injury (codes from Chapter XIX 'Injury poisoning and certain other consequences of external causes' with a code range S00–T98). Deaths with an external cause of injury were mapped to the nature of injury using information reported in the associated causes of death.

As there can be multiple associated causes of death in a single death record in no order of severity, a single relevant associated cause of death was selected using a hierarchical approach to identify, from each record, the injury most likely to have caused the death (Table 5.19). The hierarchy in the ABDS is a modified version of that used in the NZBDS. In the NZBDS, the likelihood of the injury causing death was based on the nature of the injury, prognosis and clinical knowledge of injury conditions (NZBDS, unpublished documents).

For example, for an injury death that has traumatic brain injury reported as an associated cause of death, traumatic brain injury is the injury most likely to cause death, and would then be the injury ascribed as the nature of injury. Otherwise, the associated causes of death are assessed for the next subsequent injury in the hierarchy.

The relationship between external cause and nature of injury was used to develop age- and sex-specific matrices (cross-tabulations) to convert YLL by external cause to YLL by nature of injury. This ensures internal consistency for YLL is maintained.

Nature of injury category was found for more than 95% of injury death records. Only records with a nature of injury code were used to develop the algorithm.

The matrices were applied to all deaths by external cause following redistribution.

Table 5.19: Priority of nature of injury categories for assigning a single injury cause of death, for deaths with an external cause of injury as the underlying cause

Likelihood of causing death	Nature of injury
Most	Traumatic brain injury
	Spinal cord injury
	Drowning
	Burn injury
	Poisoning
	Internal and crush injury
	Hip fracture
	All other fractures
Least	All other injuries

Notes

1. Soft tissue injuries and dislocations are excluded as injuries that lead to death.
2. Tibia and ankle fractures and humerus fractures are grouped with other fractures for this purpose.

Indigenous estimates

Indigenous mortality estimates by external cause of injury used the same methods (including redistribution) as used for national estimates.

The same age- and sex-specific matrices (cross-tabulations) to convert YLL by external cause to nature of injury as used for national estimates were used for Indigenous injury deaths.

Indigenous deaths were adjusted for under-identification using mortality adjustment factors from the ABS Census Data Enhancement data quality study (see Appendix Table B1).

Morbidity estimates

YLD was estimated for each injury sustained in an incident. That is, where a person sustained multiple injuries—for example, a traumatic brain injury, plus a fractured pelvis, plus an arm amputation from a road traffic accident—the YLD associated with each injury in the disease list was counted. To maintain consistency for YLD, the total sum of these YLD were attributed to a single external cause.

In the ABDS 2011 it is assumed that all injuries in Australia are treated, so the GBD disability weights that relate to untreated injuries were not considered relevant.

Scope of non-fatal injuries

The scope of injuries was limited to those incurred from trauma. That is, for example, disability associated with surgical amputations due to a disease, or chronic conditions, such as carpal tunnel syndrome, and chronic back pain or poisoning due to infections, are out of scope. Similarly the consequences of some medical injuries are likely captured in other disease groups.

Non-fatal injuries were identified as all injuries admitted to hospital (admitted) or presented to an emergency department without hospital admission (non-admitted).

Other injuries, for example, those presenting only to a general practitioner and those for which no medical care was sought, were not captured. This approach is similar to previous

Australian studies where injuries treated outside the hospital system were assumed to result in disability too insignificant to warrant being included (Begg et al. 2007). But this imposes a limitation on the estimates, and might warrant further investigation in future iterations if appropriate data were available.

Due to the nature of identifying injuries in the ABDS, some cases of insignificant injury will be included where they have co-occurred with injuries warranting hospital care.

Sequelae

All injuries were assumed to have short-term consequences. Long-term consequences were assigned consistent with previous studies. Where none of the ABDS 2003, NZBDS or GBD studies report having long-term consequences, the sequelae were assumed to be short-term only.

Sequelae, health states and the average duration of short- and long-term functional limitations were assigned to injuries following the published methods from GBD 2013 as closely as possible. These parameters are shown in Appendix Table D9.

Prevalence estimation

Prevalence estimation is undertaken separately for short- and long-term consequences.

Key data sources to estimate prevalence of injuries were the NHMD and the National Non-admitted Patient Emergency Department Care Database (NNAPEDCD). The prevalence of long-term consequences was estimated using DISMOD II based on incident cases derived from the NHMD and the NNAPEDCD.

Injury cases were identified in NHMD based on separations in the 2003 and 2011 calendar years. The NNAPEDCD for 2013–14 was used as this was the only year available at the time of the analysis that included information about the diagnosis.

The steps and data sources to estimate health loss due to injury are summarised in Figure 5.1.

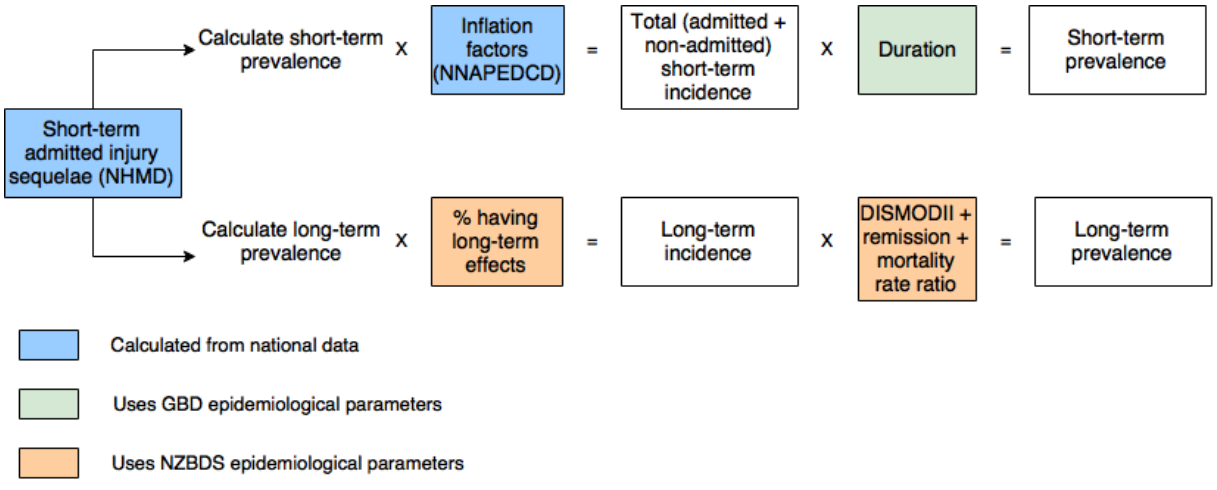

Figure 5.1: Steps and data sources for calculating prevalence of injury sequelae

Short-term sequelae

To capture all injuries that presented to a hospital, both admitted cases and non-admitted cases were counted.

Admitted cases

Short-term admitted injury cases were identified as all separations where the primary reason for admission was injury. All diagnoses of injury in that separation were used to calculate the burden as each diagnosis represents an injury that has resulted in health loss. Injuries reported as additional diagnoses in records where the principal diagnosis was not an injury were excluded.

Injury separations were identified from records in the NHMD where the principal diagnosis was in the ICD-10-AM range S00–T75, T79, T80, T81 and T88. Burden was derived from all injuries in this range of codes recorded in these separations, either as the principal diagnosis or the additional diagnoses.

Multiple mentions of the same injury were counted only once per episode of care. Where there were multiple reports of different levels of severity in the same hospital episode of care, the most severe injury was counted over the less severe mentions of injury.

Burden due to medical injuries in the ICD-10-AM range T82–T87 were assumed to be captured in other disease groups by the underlying reason for the transplant or amputation.

Separations for acute types of care only were counted. This excludes injuries presenting to hospitals, for example, for rehabilitation. It was assumed that the burden associated with injuries requiring rehabilitation are sufficiently estimated using the methods described for long-term consequences of injuries.

Hospital separations where the person died were excluded, as the non-fatal burden from these injuries was assumed to be short term, while the fatal burden was captured in YLL.

Repeat admissions by way of transfers were accounted for by excluding inward transfers of admitted patients from other hospitals. Otherwise, no further adjustment was made for repeat admission for the same injury.

Adjusting for non-admitted injuries

To quantify injury cases presenting to emergency departments but not admitted to hospital, injuries presenting to emergency departments were sourced from the NNAPEDCD database for 2013–14. This data set included a diagnosis variable.

As diagnosis data were provided in several classifications, only jurisdictions that had more than 95% of emergency department records coded to an ICD-10-AM classification (when estimates were prepared in October 2015) were included in the analysis. Consequently, all records from New South Wales and Western Australia hospitals were excluded. Further exclusions were made for records not coded to an ICD-10 AM classification. In total, about 52.2% of records were found to be usable for the purposes of ABDS. From the useable records, 20.1% had a principal diagnosis of injury.

For each injury sequela, an inflation factor by age and sex was calculated to upwardly adjust the number of admitted cases to represent the total (admitted and non-admitted) incident cases. The inflation factors reflect the number of non-admitted cases that occurred for each admitted case (as per the NNAPEDCD). The factors were applied to admitted cases from the NHMD to reflect the total sum of admitted and non-admitted cases having short-term sequelae.

As not enough diagnosis information was available in the NNAPEDCD prior to 2013–14, the same inflation factors were applied to both 2003 and 2011. A broad assumption in this method was that admission and non-admission rates in 2013–14 were applicable to 2003 and 2011.

A limitation of this method is the reliability of the inflation factors—that is, these data have not been rigorously assessed to understand how well the diagnosis predicts admission. Very broad assessment of the data was done for limited types of injuries to determine some level of consistency with expectation. For example, the proportion of all hip fractures that resulted in admission was high (above 95%) as would be expected. In addition, it should be noted that NNAPEDCD data are not necessarily representative of presentations to emergency departments that are not in scope for the collection—for example, in small hospitals or remote areas. In 2013–14, it was estimated that about 88% of emergency occasions were reported in the NNAPEDCD (AIHW 2014d).

Long-term sequelae

Long-term consequences of injury reflect the consequences that persist more than 1 year after the injury. For injuries with long-term functional consequences, the point prevalence was estimated using DISMOD II based on: the proportion of admitted incident cases expected to have long-term consequences; the expected extent of health loss (defined as the annual remission); and expected patterns of mortality (mortality risk ratio).

The values for these parameters for each long-term injury sequelae were sourced from the NZBDS. The parameters used to estimate long-term prevalence are presented in Appendix Table D9.

For each ABDS reference period (2003 and 2011) the respective national mortality rates and populations were used for DISMOD II calculations. The DISMOD II output of prevalent number of cases for each year was used to represent the likely current prevalence of long-term injury sequelae. Note that little additional modelling was required in DISMOD II, as the availability of unit record level data in Australia and its use as the single source for injury prevalence enabled highly accurate data inputs at very fine levels.

Conversion to external cause

Injury YLD were calculated according to the nature of the injury, then converted to external cause using matrices that describe the relationship between the injury and the external cause.

The matrices were derived directly from the NHMD using the principal diagnosis and the first reported external cause. Each matrix was calculated using age- and sex-specific cross-tabulations of injury diagnosis and external cause, providing a set of weights to redistribute the total YLD by nature of injury categories to external cause categories.

As the matrix is derived using admitted cases only (there is no external cause in the NNAPEDCD), it is assumed that the external cause of non-admitted injuries follows a similar pattern to admitted injuries. It is possible that the relationship between external cause and injury is different, depending on whether the injury resulted in admission or not. This method could be further refined using state-based non-admitted data comprising external cause and injury to develop more accurate matrices for non-admitted injuries

It was also assumed that pattern of external causes giving rise to particular injuries is the same nationally. That is, the matrixes have not been calculated specifically for subnational populations.

2003 estimates

The approach used to estimate 2003 prevalence was the same as that used for 2011 estimates. The prevalence of short-term sequelae and long-term sequelae were calculated using the same methods. However, the inflation factors used for 2011 were used to estimate 2003 total short-term incidence due to lack of NNAPEDCD data for 2003.

Subnational estimates

Subnational estimates were largely derived directly using the same methods as for national estimates. This was facilitated by the availability of unit record data in the NHMD.

For injury cases obtained from the NHMD, subnational estimates were derived by applying the 2011 ASGS remoteness areas and 2011 SEIFA population-based Index of Relative Socioeconomic Disadvantage quintiles to the Statistical Area Level 2 recorded in hospital separations data. The non-admitted inflation factors were then applied to these cases.

The long-term national prevalence derived from DISMOD II was apportioned into each state/territory, remoteness area and socioeconomic group based on the age and sex distribution of the short-term admitted cases used to estimate long-term prevalence.

Indigenous estimates

Indigenous estimates of non-fatal injury burden used the same methods as for the national estimates for both 2011 and 2003.

For short-term prevalence, Indigenous cases of short-term injury from the NHMD were adjusted for under-identification using standard adjustment factors from hospital data quality studies by the AIHW (see Chapter 4 and Appendix tables C3 and C4). The national inflation factors used to adjust for non-admitted injuries were applied to the adjusted Indigenous separations (see 'Adjusting for non-admitted injuries').

Long-term injury prevalence was estimated by apportioning national long-term prevalence estimates from DISMOD II according to the age and sex patterns of short-term admitted cases for Indigenous Australians.

The conversion of YLD by nature of injury to external cause used the same age and sex matrices as used for the national estimates.

Kidney and urinary conditions

Mortality estimates

Deaths related to kidney and urinary conditions were assigned from the NMD as defined by the disease list (Appendix Table A2). Deaths due to acute renal failure (N17) and unspecified renal failure (N19) were redistributed. Acute kidney failure was redistributed because it has multiple causes and is generally a consequence of many other diseases, including injury, infection, cancer, and myocardial infarction. Unspecified renal failure was redistributed because it might be due to chronic or acute renal failure.

Deaths coded to these codes were redistributed using a 2-step approach:

1. Deaths coded to unspecified renal failure (N19) were redistributed using direct evidence to acute renal failure (N17) and chronic renal failure (N18) according to the proportions

obtained from information on hospitalisations prior to death in linked data from New South Wales and Western Australia (AIHW 2014c).

2. Deaths coded to acute renal failure (N17, including those reassigned from N19) were redistributed over all disease groups using the indirect MCOD method.

Morbidity estimates

Sequelae and health states

Sequelae and health states assigned to kidney and urinary conditions are included in Table 5.20. Asymptomatic chronic kidney disease is defined as chronic kidney disease stages 1–2 and stage 3 (without anaemia). End-stage kidney disease is defined as stage 5 chronic kidney disease.

Anaemia due to chronic kidney disease is part of the anaemia envelope—as it can result from several conditions, the sum of anaemia from various diseases cannot exceed the total experienced within the population. The definitions for the severity of anaemia in GBD use those described in Kassebaum et al. 2014. These were applied to people with chronic kidney disease *and* anaemia. Specifically the definitions used were for all those aged 5 and over (excluding pregnant women). See the section on methods for blood and metabolic disorders for more information on the methods used to estimate the anaemia envelope.

Table 5.20: Sequelae, health states and duration for kidney and urinary conditions diseases

Disease	Sequela	ABDS 2011 health state identifier[a]	Duration
Chronic kidney disease	Asymptomatic chronic kidney disease	262	. .
	Anaemia due to stage 3 chronic kidney disease[b]	195, 196, 197	12 months
	Stage 4 chronic kidney disease	41	12 months
	Anaemia due to stage 4 chronic kidney disease[b]	195, 196, 197	12 months
	End-stage kidney disease on dialysis	43	12 months
	End-stage kidney disease after transplant	42	12 months
	Untreated end-stage kidney disease	22	5.5 months
Enlarged prostate	Enlarged prostate	47	12 months
Kidney stones	Kidney stones	193	2 weeks
Other kidney and urinary diseases	Other kidney and urinary diseases	. .	. .

(a) See Appendix Table C1.

(b) Part of anaemia envelope.

Prevalence estimation

Chronic kidney disease

The primary data source used to estimate prevalence of chronic kidney disease (with and without anaemia) was the AHS 2011–12, while the primary data source to estimate prevalence of end-stage kidney disease was the Australia and New Zealand Dialysis and Transplant Registry 2011.

Asymptomatic chronic kidney disease

The prevalence of asymptomatic chronic kidney disease (stages 1–3) was estimated using measured data from the AHS 2011–12. Stages were determined by combining the participants' estimated glomerular filtration rate results with their albumin creatinine ratio results as described in *Cardiovascular disease, diabetes and chronic kidney disease, Australian facts: prevalence and incidence* (AIHW 2014e).

Asymptomatic chronic kidney disease was given an asymptomatic health state; therefore, it was given a disability weight of 0.

Anaemia due to stage 3 chronic kidney disease

The AHS 2011–12 was used to calculate the proportion of people with stage 3 chronic kidney disease by broad age group and sex. The age and sex distribution was further refined using the age and sex of people who were hospitalised for N18.3 in 2011.

No severe anaemia due to stage 3 chronic kidney disease was reported in the AHS. Instead, we have used the global estimate of people with stage 3 chronic kidney disease severe anaemia in GBD 2013 (GBD 2013 Collaborators 2015b), though the situation might be different in high-income countries such as Australia.

Stage 4 chronic kidney disease and anaemia due to stage 4 chronic kidney disease

The prevalence of stage 4 chronic kidney disease was also estimated from measured data from the AHS 2011–12, using the estimate of people with stage 4 and 5 chronic kidney disease minus those with end-stage kidney disease (stage 5 only) sourced from the Australia and New Zealand Dialysis and Transplant Registry.

It is not possible to break down the combined chronic kidney disease stages 4 and 5 data in the AHS 2011–12 by anaemia status, due to small numbers. The severity distribution of mild, moderate and severe anaemia due to stage 4 chronic kidney disease was sourced from GBD 2013 (GBD 2013 Collaborators 2015b).

The age and sex distribution was based on the AHS 2011–12 results, but broken down further by the age and sex of people who were hospitalised for N18.4 in 2011.

End-stage kidney disease treated with dialysis or transplant

Registry data from the Australia and New Zealand Dialysis and Transplant Registry in 2011 was used to determine the prevalence of end-stage kidney disease treated by dialysis or transplant.

Untreated end-stage kidney disease

Untreated end-stage kidney disease refers to people who were not receiving kidney replacement therapy, although they might be receiving palliative treatments. The prevalence of people with untreated end-stage kidney disease was estimated from an analysis of the 2010 Australia and New Zealand Dialysis and Transplant Registry linked with the AIHW National Mortality Database and National Death Index, to identify people who died from end-stage kidney disease who were not treated with kidney replacement therapy or were not in the registry (AIHW 2011). The prevalence for 2010 was assumed to be the same as 2011.

Survival was estimated using an analysis of New South Wales and Western Australian linked hospital and mortality data, by age and sex (AIHW 2014c), which indicated that the mean survival time for people with untreated end-stage kidney disease was approximately 5.5 months.

Enlarged prostate

Enlarged prostate includes cases of benign prostatic hypertrophy, and excludes prostate cancer.

Prevalence was estimated using hospitalisations with a diagnosis of enlarged prostate diagnosis (N40) in 2011 from the NHMD. This includes people admitted for surgery or for other reasons, which are both assumed to indicate significant health loss, due to hospitalisation being required. Admissions where there is also a diagnosis of prostate cancer (C61) were excluded. Ratios of persons-to-separations derived from Western Australian linked hospitalisations and deaths data were used to adjust national NHMD data for potential readmissions and hospital transfers.

The people receiving hospital treatment for enlarged prostate are assumed to be symptomatic for the entire year.

Kidney stones

Kidney stones include cases of urolithiasis of the kidney, ureter and lower urinary tract.

Point prevalence was estimated by applying a duration of 2 weeks, based on the NZBDS, to the incident cases of kidney stones—that is, the number of hospitalisations with a diagnosis of kidney stones (N20–N21) in 2011 from the NHMD. As this is an acute condition, each separation was assumed to be a case.

Other kidney and urinary diseases

YLD was derived indirectly by applying the YLD-to-YLL ratio for kidney stones to the YLL for other kidney and urinary diseases.

Subnational estimates

Prevalence estimates by state and territory, remoteness and socioeconomic group were derived directly from the data source, with the exception of stage 3 chronic kidney disease with anaemia, stage 4 chronic kidney disease with anaemia, and stage 4 chronic kidney disease. For these estimates, hospital separations ratios data were used as a proxy in 2011.

2003 estimates

Estimates of end-stage kidney disease, kidney stones, and enlarged prostate were taken directly from the same data source using the same method as used for the 2011 estimates.

The ratio of the prevalence of end-stage kidney disease treated by dialysis or transplant to the prevalence of stage 3 chronic kidney disease and stage 4 chronic kidney disease in 2011 was used to estimate prevalence in 2003, due to lack of biomedical measurement data consistent with the 2011 method.

As the codes used to estimate the age and sex distributions from hospitalisations data were not in use in 2003, the 2011 age and sex distribution from hospitalisations were applied to the 2003 estimates. The same anaemia severity distributions were applied as in 2011.

Indigenous estimates

The same methods and data sources were used to derive Indigenous estimates for kidney and urinary diseases for both 2011 and 2003. Indigenous data were directly available from the Australia and New Zealand Dialysis and Transplant Registry and the NHMD.

Biomedical data for stage 3 chronic kidney disease with anaemia, and stage 4 chronic kidney disease was sourced from the AATSHIS 2012–13.

Estimates based on hospital separations data (enlarged prostate and kidney stones) were adjusted for under-identification using standard adjustment factors (see Chapter 4 and Appendix tables C3 and C4).

The national severity distributions for anaemia were used for both 2011 and 2003 Indigenous estimates.

Mental and substance use disorders

Mortality estimates

Mental and substance use-related deaths were assigned from the NMD as defined by the disease list (Appendix Table A2). Deaths due to mental disorder, unspecified (F99) were proportionally redistributed to other conditions in the mental and substance use disorders disease group.

Codes for accidental poisoning by and exposure to drugs/alcohol (X41, X42, X45) were not included in estimates of fatal burden for substance use disorders. Instead, these deaths are included in estimates for poisoning under the injuries disease group. This approach is consistent with the determinations made by coroners for such deaths in Australia.

As part of the ABS revisions process for mortality data, deaths that are confirmed as being accidental are coded under injuries. Deaths that are initially coded as poisoning with 'undetermined intent' and are determined by the coroner as being due to a drug dependence, were recoded under alcohol or substance use disorders. As such these deaths were included in estimates of fatal burden for substance use disorders in the ABDS (as the study uses the ABS revised version of mortality data for 2011).

Morbidity estimates

Sequelae

Sequelae and health states assigned to mental and substance use disorders are included in Table 5.21. Durations (where relevant) and assumptions are outlined in relevant subsections.

Table 5.21: Sequelae and health states for mental and substance use disorders

Disease	Sequela	ABDS 2011 health state identifier[(a)]
Depressive disorders	Dysthymia	262, 86
	Major depressive disorder	262, 86, 87, 88
Anxiety disorders	Anxiety disorders	262, 83, 84, 85
Bipolar affective disorder	Bipolar disorder	87, 89, 90
Alcohol use disorders	Alcohol dependence	235, 262, 73, 74, 75

(continued)

Table 5.21 (continued): Sequelae and health states for mental and substance use disorders

Disease	Sequela	ABDS 2011 health state identifier[a]
Drug use disorders (excluding alcohol)	Amphetamine dependence	236, 262, 80
	Cannabis dependence	244, 262, 79
	Cocaine dependence	245, 262, 81
	Opioid dependence	251, 262, 82
Schizophrenia	Schizophrenia	91, 92
Eating disorders	Anorexia nervosa	93
	Bulimia nervosa	94
Autism spectrum disorders	Asperger syndrome	97
	Childhood autism	98
Attention deficit hyperactivity disorder	Asymptomatic	262
	Attention deficit hyperactivity disorder	95
Conduct disorder	Asymptomatic	262
	Conduct disorder	96
Intellectual disability	Idiopathic and other intellectual disability[b]	100, 101, 102, 243, 99
Other mental and substance use disorders	Other mental and substance use disorders	83

(a) See Appendix Table C1.

(b) Part of intellectual disability envelope.

Prevalence estimation

Data sources

Key data sources to estimate mental and substance use disorder prevalences are shown in Table 5.22.

Table 5.22: Key data sources for mental and substance use disorder morbidity estimates

Data source	Related diseases
2007 National Survey of Mental Health and Wellbeing	Depressive disorders, anxiety disorders, alcohol use disorders, cannabis use disorders and bipolar disorders
2013–14 Child and Adolescent Survey of Mental Health and Wellbeing (Young Minds Matter survey)	Depressive disorders, anxiety disorders, attention deficit hyperactivity disorder, conduct disorder
National Psychosis Survey (Survey of High Impact Psychosis) 2010	Schizophrenia
Intellectual Disability Exploring Answers (IDEA) database	Idiopathic intellectual disability and autism
National Drug and Alcohol Research Centre analyses (see Degenhardt et al. 2004; Degenhardt et al. 2016)	Amphetamine use disorders and opioid use disorders
GBD 2010	Anorexia nervosa
2003–04 Te Rau Hinengaro: The New Zealand Mental Health Survey (Wells et al. 2006)	Bulimia nervosa

Estimating point prevalence

Adult estimates obtained from the 2007 National Survey of Mental Health and Wellbeing are for 12-month prevalence. To estimate point prevalence, it was assumed that 30-day prevalence would approximate point prevalence, given the long-term nature of the disorders reflected in diagnostic criteria.

As the 30-day prevalence in this survey did not reflect diagnostic criteria as closely, a 30-day-to-12-month prevalence adjustment factor applied to the 12-month estimates was derived from the 1997 National Survey of Mental Health and Wellbeing, based on expert advice.

For major depressive disorder, this ratio was 0.51, and for anxiety disorders it was 0.67. Experts advised that 12-month prevalence would be similar to 30-day prevalence for drug use disorders and dysthymia, so no ratio was applied. These ratios were also applied to estimates for children obtained from the 2013–14 Child and Adolescent Survey of Mental Health and Wellbeing (Young Minds Matter survey).

Idiopathic intellectual disability and autism were considered chronic conditions, so point prevalence was assumed to be the same as period prevalence. Similarly, eating disorders were estimated to result in health loss, on average, for more than 12 months.

Severity distributions and other health states

Severity distributions for depressive disorders, anxiety disorders and drug use disorders (excluding alcohol) were based on GBD 2013 distributions published by Burstein et al. (2015). Severity for alcohol use disorders was based on the (self-reported) extent that alcohol use interfered across various aspects of life in the 2007 National Survey of Mental Health and Wellbeing.

For bipolar disorders, the health states included mania, depression and residual states. For schizophrenia, these were acute (psychotic) and residual states. The distributions of these health states were based on meta-analyses undertaken for GBD 2010 (Ferrari et al. 2012).

No asymptomatic health state was attributed to eating disorders as the health states themselves reflected the intermittent and ongoing nature of these conditions.

The distribution of symptomatic and asymptomatic health states for attention deficit hyperactivity disorder and conduct disorder were based on findings from the Great Smoky Mountain study (Erskine et al. 2014).

Estimates of childhood autism were based on IDEA data. The prevalence of other autism spectrum disorders (including Asperger syndrome) was based on the other autism spectrum disorders-to-childhood autism ratio, as published by GBD 2010 (Baxter et al. 2015). As chronic conditions, these health states were applied for the full year.

Intellectual disability

As intellectual disability is a sequela of multiple conditions across ABDS (primarily in the infant and congenital disease group), its overall prevalence was calculated to ensure the sum of estimates for sequelae did not exceed the total. To avoid double-counting, and adhere to mutually exclusivity for each disease, the proportion of intellectual disability due to each disease was estimated.

Prevalence and severity distribution of the intellectual disability envelope

The total prevalence rate for intellectual disability due to any cause was based on analysis of the IDEA database. IDEA is a Western Australian database of people with intellectual

disability who receive: services from the Disability Services Commission; education support from the state's Department of Education; or, if they were born between 1983 and 1999, support through the Catholic or independent school systems. The database is also linked to registries of births and deaths. In this database, intellectual disability is defined as an intelligence quotient (IQ) of less than 70, and an indication of developmental delay before the age of 18. Mild, moderate, and severe intellectual disability are defined as IQ 55–69, 40–54 and less than 40, respectively. Estimates were based on births between 1983 and 2005, and followed through to 2010. IDEA data were available for people up to the age of 27.

The overall severity distribution of intellectual disability was based on an international meta-analysis (King et al. 2009, as cited by Maulik et al. 2011). Borderline intellectual functioning in children aged 0–14 was based on the borderline intellectual functioning-to-intellectual disability ratio (using cognitive scores) observed in the Longitudinal Study of Australian Children (Emerson et al. 2010).

Prevalence of intellectual disability by sequelae

The intellectual disability envelope is made up of several infant and congenital conditions, with the remaining intellectual disability falling under idiopathic/other intellectual disability in the mental and substance use disorders disease group (Table 5.23). Cases of comorbid intellectual disability and autism were not attributed an intellectual disability health state, as it was assumed that the burden of these conditions would be captured under the autism health states.

Table 5.23: Diseases within the intellectual disability envelope, and data source(s) for severity

Disease	Source of severity distribution
Pre-term birth and low birthweight complications	Mild prevalence was based on the proportion reported in the IDEA database. The relationship between mild, moderate and severe was based on the perinatal data collection
Birth trauma and asphyxia	Mild prevalence was based on the proportion reported in the IDEA database. Moderate and severe were based on severity distributions shown in NHMD analysis
Neural tube defects	Based on severity distribution reported by Hunt & Oakeshott (2003), and modelled in DISMOD II
Brain malformations	Mild prevalence was based on the proportion reported in the IDEA database. Moderate and severe were based on severity distributions shown in IDEA (Petterson et al. 2007)
Down syndrome	All prevalence was based on the proportion reported in the IDEA database, adjusted for deaths
Other chromosomal abnormalities	All prevalence was based on the proportion reported in the IDEA database

The proportions of total intellectual disability that could be attributed to diseases specified in the ABDS 2011 were mostly derived from the IDEA database. This was available separately for mild/moderate and severe/profound severity categories. For Down syndrome and other chromosomal abnormalities, prevalence was estimated directly by applying these proportions to the total.

In some cases the severity distribution was obtained from another source (Table 5.23). In this case, IDEA was used to estimate the number of mild cases, and the remaining severity estimates were calculated relative to the mild estimate.

Motor/cognitive impairment due to neural tube defects was modelled entirely in DISMOD II.

Idiopathic intellectual disability

Intellectual disability sequelae from other diseases (including motor-cognitive sequelae) were subtracted from the intellectual disability envelope. The remaining estimates were the prevalence of idiopathic intellectual disability (which also includes other underlying conditions resulting in intellectual disability not captured elsewhere). All borderline intellectual disability was attributed to the idiopathic/other category.

Other mental and substance use disorders

This residual group includes delirium, personality disorders, and any remaining child disorders such as specific learning disorders, developmental disorders and sleep disorders.

The prevalence of other mental and substance use disorders was estimated by analysing hospitalisations for the corresponding ICD-10-AM codes (F04–09, F17, F44–49, F51–69, F80–83, F85–89, F93–99).These separations were then compared with those for depression, anxiety, bipolar, schizophrenia, conduct disorder, and attention deficit hyperactivity disorder (that is, conditions with some similar aspects and conceivably similar rates of hospitalisation).

Rate ratios were specific to the reference year (2003 or 2011), age group, and Indigenous status, but were not created separately for subnational estimates. Separation rate ratios were then applied to the combined point prevalence estimates, by age and sex (excluding asymptomatic estimates) of the compared conditions to calculate the prevalence of other mental and substance use disorders. This assumes a similar hospitalisation rate for other mental and substance use disorders and the identified conditions.

Subnational estimates

The 2007 National Survey of Mental Health and Wellbeing was analysed to calculate total prevalence rate ratios for each socioeconomic group, remoteness area (*Very remote* areas were not sampled), and state/territory. These were then applied to the national prevalence rates for depressive disorders, anxiety disorders, bipolar disorder, alcohol use disorders and drug use disorders. Where these rate ratios were unreliable due to small sample sizes, a proxy rate ratio was used, usually from a nearby state/territory (the Victorian rate ratio was used for Tasmania, New South Wales for the Australian Capital Territory, and South Australia for the Northern Territory).

State and territory rate ratios for opioid use disorders were based on the analysis by Degenhardt et al. (2004). The relative rate of hospitalisations for these disorders in *Outer regional*, *Remote* and *Very remote* areas was applied to provide rate ratios for *Very remote* areas, which was not sampled in the 2007 National Survey of Mental Health and Wellbeing.

The socioeconomic group rate ratios calculated for bipolar disorder were also applied to schizophrenia, due to lack of specific schizophrenia data. Schizophrenia prevalence rates were modelled as consistent across remoteness areas and state/territory.

For attention deficit hyperactivity disorder and conduct disorder, rate ratios were available by remoteness area and socioeconomic group, but not state/territory from the 2013–14 Child and Adolescent Survey of Mental Health and Wellbeing (Young Minds Matter survey). Consistent prevalence rates were assumed across states and territories for these 2 conditions.

For eating disorders, autism and intellectual disability, the same prevalence rates were assumed to be consistent across socioeconomic groups, remoteness areas and states/territories due to lack of data.

2003 estimates

With a few exceptions, all prevalence rates were considered stable between 2003 and 2011, based on expert advice or lack of available evidence to suggest a significant change. The 2003 opioid prevalence estimates were based on estimates of prevalence in 2002, as reported by Degenhardt et al. (2004). These estimates were then adjusted for change over time, based on data from the National Opioid Pharmacotherapy Statistical Annual Data collection.

The data source for amphetamine disorders (Degenhardt et al. 2016) included estimates for 2003–04 and 2011–12, so each of these was used for the corresponding reference year.

Prevalence estimates for other drug use disorders and other mental and substance use disorders were based on hospitalisation ratios, so for 2003 these were based on hospitalisations during the 2003 calendar year.

Some of the specific causes of intellectual disability that contributed to the intellectual disability envelope were adjusted for differences in rates reported by WARDA for 2003.

Indigenous estimates

All Indigenous estimates were calculated using indirect methods, which involved applying rate ratios from secondary data sources to national prevalence rates. This method was used as no Indigenous-specific data sources were identified that provided adequate information on the prevalence of mental and substance use disorders in the Aboriginal and Torres Strait Islander population. Rate ratios used for selected mental and substance use disorders can be found in Appendix Table D10.

For depressive disorders, anxiety disorders, bipolar disorder and schizophrenia, Indigenous prevalence estimates were calculated using Indigenous-to-total population rate ratios from data provided by Queensland Health from their Consumer Integrated Mental Health Application (CIMHA). These data are ICD-10-AM coded inpatient separation data linked with community mental health services data, and provide a measure of the number of people accessing Queensland public mental health services.

For alcohol use disorders, for which prevalence estimates are required by level of severity, hospitalisation rate ratios were used to derive Indigenous prevalence estimates for asymptomatic, very mild and mild cases of alcohol dependence. Rate ratios from Queensland's CIMHA data were applied to derive Indigenous prevalence estimates for moderate and severe cases of alcohol dependence.

For drug use disorders, CIMHA rate ratios were used to derive Indigenous prevalence for opioid, cannabis and amphetamine dependence (for all levels of severity). For cocaine dependence, rate ratios from self-reported survey data from the 2013 National Drug Strategy Household Survey (NDSHS) were used to derive Indigenous prevalence (for all levels of severity).

For attention deficit hyperactivity disorder and conduct disorder, Indigenous prevalence estimates were based on the rate ratios from the Longitudinal Survey of Australian Children and CIMHA. An average of these rate ratios was applied to national prevalence estimates.

For eating disorders and autism spectrum disorders, total population prevalence rates were applied to the Indigenous population. Due to significant issues with the availability and quality of data on autism spectrum disorders in the Indigenous population, the AIHW did not report Indigenous autism spectrum disorders estimates separately in the final ABDS 2011

report (they were used for estimates of total non-fatal burden for the mental and substance use disorders disease group).

For intellectual disability, Indigenous prevalence estimates were calculated using Indigenous-to-non-Indigenous rate ratios from the IDEA database for the most recent period. Indigenous severity distributions were based on a combination of the meta-analysis by King et al. (2009, as cited by Maulik et al. 2011), which was used for the national severity distribution, and a comparison of Indigenous and non-Indigenous severity distributions reported by the IDEA database.

Similar to national estimates, for most mental and substance use disorders, Indigenous prevalence rates were considered stable between 2003 and 2011, based on expert advice or lack of available evidence. One main exception was for depressive disorders and anxiety disorders, in which prevalence was modelled as 10% lower in 2003 than in 2011. This decrease was based on results from the 2004–05 NATSIHS and 2012–13 AATSIHS indicating a significant increase in high/very high psychological distress for Indigenous Australians between 2004–05 and 2012–13. Other exceptions were for alcohol use disorders (severe), other drug use disorders and other mental and substance use disorders, which were all based on hospitalisation rate ratios specific to 2003.

Musculoskeletal conditions

Mortality estimates

Deaths related to musculoskeletal conditions were assigned from the NMD as defined by the disease list (Appendix Table A2). No musculoskeletal condition deaths were redistributed.

Morbidity estimates

Sequelae

Sequelae and health states assigned to musculoskeletal conditions are included in Table 5.24. Durations and assumptions are outlined in subsections for individual diseases.

Table 5.24: Sequelae and health states for musculoskeletal conditions

Disease	Sequela	ABDS 2011 health state identifier[(a)]
Osteoarthritis	Osteoarthritis of the knee	126, 127, 128, 262,
	Osteoarthritis of the hip	126, 127, 128, 262,
Gout	Musculoskeletal problems caused by gout	132, 133
Rheumatoid arthritis	Musculoskeletal problems caused by rheumatoid arthritis	130, 131, 132, 262
Back pain and problems	Back pain and problems	233, 234, 239, 240, 241, 242, 254, 255, 262
Other musculoskeletal conditions[(b)]	Other musculoskeletal problems	126, 127, 128, 130, 131, 132, 262

(a) See Appendix Table C1.

(b) Other musculoskeletal conditions excludes symptoms signs involving musculoskeletal conditions and osteoporosis

Prevalence estimation

Prevalence estimates for musculoskeletal conditions were derived from self-reported data in the NHS component of the AHS 2011–12, as it covered all the musculoskeletal conditions of interest. After consultation with ABS about a specific data quality issue with published musculoskeletal data, the ABS provided the AIHW with revised musculoskeletal data for analysis. The revised data are currently unpublished by the ABS, but available on request.

Prevalence rates were derived from the AHS estimates and the survey population. These rates were applied to the national 2011 population to generate prevalence estimates for each condition.

Though self-reported data is generally not considered as good as clinical data, Peeters et. al. (2015) found that self-reported data is acceptable for osteoarthritis and rheumatoid arthritis.

The AHS 2011–12 was used to provide an overall prevalence for all musculoskeletal conditions, as well as prevalence estimates for individual diseases within the disease group.

Data derived from the survey was available for 5-year age groups (0–85 and over). For individual diseases and subnational estimates, these 5-year age groups were combined to address sample size issues from the survey. Modelling was required to redistribute the data into 5-year age groups for analysis.

The severity distribution for each of the musculoskeletal conditions, except for gout, is based on the distribution across the 6 pain categories (none, very mild, mild, moderate, severe, or very severe) in the preceding 4 weeks, as used in the AHS 2011–12. The pain categories were mapped to the relevant health states, as described in the following individual sections.

For each condition, the severity distribution analysis was limited to those who only reported experiencing the condition of interest (that is, not multiple conditions) to ensure that the severity distribution was specific for each condition. This distribution was then applied to all cases of the condition.

A key assumption from this method was that the proportion of people who report no pain in the preceding 4 weeks was equivalent to the proportion of people with the condition who are asymptomatic at any point in time.

Osteoarthritis

The AHS data for osteoarthritis cannot be broken down into the sequelae osteoarthritis of the hip and osteoarthritis of the knee; this was split (for risk factor analysis) using proportions from GBD 2013.

Severity is based on the distribution of the pain experienced in the previous 4 weeks by people reporting arthritis only (Table 5.25). Health loss is assumed to last for the entire year.

Table 5.25: ABDS 2011 severity distribution for osteoarthritis

Osteoarthritis	Asymptomatic	Mild	Moderate	Severe
ABDS 2011 health state identifier	262	126	127	128
Proportion (%)	14.5	46.9	28.0	10.6

Gout

As a breakdown of chronic or acute gout was not available in the AHS data, the distribution of severity and the average number and duration of gout episodes was based on the GBD 2010 pain method (Hoy et al. 2014). This method assigned 1.4% of cases as chronic (with 12 months duration) and the remaining 98.6% of cases as acute, with an average 3.9 episodes of 6.8 days duration per year.

Rheumatoid arthritis

The AHS 2011–12 does not collect information on the affected joints or the severity of rheumatoid arthritis. The distribution of severity for rheumatoid arthritis is based on the distribution of pain reported by people reporting rheumatoid arthritis only in the AHS 2011–12 (Table 5.26). Health loss is assumed to last for the entire year.

Table 5.26: ABDS 2011 severity distribution for rheumatoid arthritis

Rheumatoid arthritis	Asymptomatic	Mild	Moderate	Severe
ABDS 2011 health state identifier	262	130	131	132
Proportion (%)	28.9	48.3	11.2	11.6

Back pain and problems

The NHS data only collected information on back pain as a long-term (chronic) condition. Health loss is assumed to last for the entire year. No estimates are provided for short-term back pain and problems.

The distribution of severity for back pain and problems is based on an associated pain data distribution (back pain and problems only) from the AHS 2011–12. Because this variable did not distinguish between those with or without leg pain, the proportion of people experiencing pain at each severity level was divided into with and without leg pain according to proportions from GBD 2013. The resulting severity distribution is provided in Table 5.27.

Table 5.27: ABDS 2011 severity distribution for back pain and problems

Back pain and problems	Asymptomatic	Mild	Moderate	Severe	Very severe
ABDS 2011 health state identifier	262	234	233	242	240
Proportion without leg pain (%)	15.1	41.9	19.1	5.4	0.9
ABDS 2011 health state identifier	. .	254	255	241	239
Proportion with leg pain (%)	. .	10.9	5.0	1.4	0.2

Other musculoskeletal disorders

The prevalence of other musculoskeletal disorders was also derived from the AHS 2011–12. It was estimated by subtracting the prevalence of specific musculoskeletal conditions (osteoarthritis, rheumatoid arthritis, gout, and back pain/problems) from the prevalence of all musculoskeletal conditions combined.

The distribution of severity for other musculoskeletal disorders is based on associated pain data distribution (other musculoskeletal conditions only) from the AHS 2011–12 (Table 5.28).

Table 5.28: ABDS 2011 severity distribution for other musculoskeletal disorders

Other musculoskeletal conditions	Asymptomatic	Level 1	Level 2	Level 3	Level 4	Level 5	Level 6
ABDS 2011 health state identifier	262	126	127	130	128	131	132
Proportion (%)	12.7	30.4	15.9	16.9	16.9	6.2	1.0

Subnational estimates

National prevalence estimates were apportioned based on sex and combined age-specific estimates from the AHS 2011–12 to derive subnational estimates. Sex and 5-year age-specific proportions were not used due to a high degree of uncertainty in some 5-year age groups, with relative standard errors of more than 50% for these estimates.

2003 estimates

The same methods used for the 2011 estimates were used for 2003 non-fatal burden musculoskeletal conditions estimates. The primary data source was the NHS 2004–05. Since the data were not specific to 2003, a survey prevalence rate (that is, rates generated from the survey population) was applied to the 2003 Estimated Resident Population to estimate the 2003 population prevalence of each disease.

As no equivalent pain variable was available for the NHS 2004–05, the same severity distributions used for 2011 were assumed for each disease.

Indigenous estimates

The methods used to estimate the non-fatal musculoskeletal conditions burden for the Indigenous population was similar to the method used for national estimates.

The AATSIHS 2012–13 was the primary data source for 2011 estimates. After consultation with ABS regarding a specific data quality issue with published musculoskeletal data from the survey, the ABS provided the AIHW with revised data for back pain and problems and other musculoskeletal conditions for analysis. The revised data are currently unpublished by the ABS, but available on request. For 5-year age groups with high relative standard error, the national 5-year age distribution was applied to the Indigenous prevalence estimate specific for those age groups.

As the musculoskeletal data items from previous ABS Indigenous health surveys were not comparable with those from the AATSIHS 2012–13, and there was little evidence to suggest a recent change in prevalence of musculoskeletal conditions in the Indigenous population, Indigenous estimates for 2003 were derived from rates from the AATSIHS 2012–13 applied to the 2003 Indigenous population.

The severity distribution for each condition was assumed to be the same as for the national estimates for both 2011 and 2003.

Neurological conditions

Mortality estimates

Neurological conditions-related deaths were assigned from the NMD as defined by the disease list (Appendix Table A2). Deaths coded to ICD-10 codes G81–G83 were proportionally distributed across all diseases using proportions derived from Australian all-cause mortality data.

Morbidity estimates

Sequelae

Sequelae and health states assigned to the neurological conditions are included in Table 5.29. Durations and assumptions are outlined in subsections for individual diseases.

Table 5.29: Sequelae and health states for neurological conditions

Disease	Sequela	ABDS 2011 health state identifier[(a)]
Epilepsy	Epilepsy	207, 248, 249
Dementia	Dementia	58, 59, 60
Parkinson disease	Parkinson disease	70, 71, 72
Multiple sclerosis	Multiple sclerosis	63, 64, 65
Motor neurone disease	Motor neurone disease	65
Migraine	Migraine	61
Guillain-Barré syndrome	Guillain-Barré syndrome	188

(a) See Appendix Table C1.

Prevalence estimation

Epilepsy

For the ABDS 2011, epilepsy is defined consistent with GBD 2013 as a chronic disorder of the brain characterised by recurrent seizures. Sex-specific prevalence estimates of self-reported epilepsy were obtained from the AHS 2011–12. Because the relative standard errors by age and sex were too high for many age groups, proportions of prevalence by 5 year age group by sex were estimated using prevalence-to-separation ratios derived from Western Australian linked hospitalisations and deaths data, applied to the NHMD.

As there was no direct Australian data source to estimate the severity of epilepsy as defined in the ABDS 2011, the epilepsy severity distribution was based on the European study by Forsgren et al. (2005).

Dementia

Dementia includes Alzheimer disease (the most common form), vascular dementia, dementia with Lewy bodies and frontotemporal dementia (F00–F03, G30–G31). Prevalence estimates for dementia were calculated using the prevalence rates published in *Dementia in Australia* (AIHW 2012b). For more information on the methods used to derive dementia prevalence estimates, see Note 2.2 in Appendix D of that report.

The severity distribution of dementia was estimated using 2 European studies (Barendregt & Bonneux 1998; Lucca et al. 2015).

Parkinson disease

Due to a lack of recent population-based Australian studies on Parkinson disease at the time of analysis, prevalence was estimated using findings from 2 international studies (de Rijk et al. 2000; Willis et al. 2013).

The severity distribution was derived from unpublished data from the Queensland Parkinson's Project.

Multiple sclerosis

Prevalence estimates for multiple sclerosis were based on the Australian study by Palmer et al. 2013.

The severity distribution was obtained from the joint report by Covance Pty Ltd and Professor Palmer (Covance Pty Ltd & Palmer 2011).

Motor neurone disease

Motor neurone diseases are a group of progressive neurological disorders (including amyotrophic lateral sclerosis) that destroy motor neurons. Motor neurone disease prevalence was estimated using prevalence-to-separations ratios derived from Western Australian linked hospitalisations and deaths data applied to the count of separations from NHMD.

Since GBD 2013 did not have a disability weight specific to motor neurone disease, the disability weight for severe multiple sclerosis was assumed to apply.

Migraine

According to Headache Australia, migraines are headaches that typically last 4–72 hours. Period prevalence estimates for migraine in a 6 month period were obtained from the AHS. Point prevalence was estimated by applying a duration of 18 days in a year based on 12 episodes (about once a month) per year at 1.5 days per episode (NZBDS, unpublished documents).

Guillain-Barré syndrome

Guillain-Barré syndrome is a disease of the peripheral nervous system that might develop spontaneously or after a systemic infection or other stress. Guillain-Barré syndrome prevalence was estimated using a prevalence-to-separation ratio derived from Western Australian linked hospitalisations and deaths data. This was used to adjust the count of separations from NHMD, to better estimate the prevalence. A duration of 6.7 months, based on GBD 2013 (GBD 2013 Collaborators 2015a), was applied to obtain point prevalence estimates.

Other neurological conditions

The prevalence of other neurological conditions is the prevalence of the remaining neurological conditions that are not listed above. The prevalence for other neurological conditions was estimated by applying a YLD-to-YLL ratio for Parkinson disease, multiple sclerosis and motor neurone disease combined to the YLL for other neurological conditions.

2003 estimates

Where available, the same data source was used for 2003 estimates. Motor neurone disease and Guillain-Barré syndrome were derived from NHMD for 2003, and epilepsy and migraine were based on data from the NHS 2004–05. Estimates for the remaining diseases (dementia, Parkinson disease and multiple sclerosis) used the 2011 prevalence rates applied to the 2003 population due to a lack of more suitable data to estimate the prevalence of these conditions for the 2003 reference year.

Subnational estimates

Where prevalence was obtained from the AHS (epilepsy, migraine) or NHMD (motor neurone disease, Guillain-Barré syndrome), national estimates by state and territory, remoteness area and socioeconomic group were derived directly from the data source.

For multiple sclerosis, prevalence rates by state and territory and remoteness area were available from the main data source. Prevalence estimates by socioeconomic group were derived by applying proportions derived from the NMD onto the national estimates.

For dementia and Parkinson disease, breakdowns by state/territory, remoteness area and socioeconomic group were derived by applying proportions from the NMD onto the national estimates.

Indigenous estimates

Indigenous estimates based on hospital separations data (epilepsy, motor neurone disease and Guillain-Barré syndrome) were adjusted for under-identification using standard adjustment factors (see Chapter 4 and Appendix tables C3 and C4).

Indigenous prevalence for epilepsy and migraine were derived from the AATSIHS 2012–13 for 2011 estimates and the NATSIHS 2004–05 for 2003 estimates, using a similar method as for the national estimates.

Indigenous dementia prevalence was obtained using 2 Australian studies (Radford et al. 2015; Smith et al. 2008). The severity distribution was obtained from the Koori Growing Old Well Study (Radford et al. 2015) and the Barendregt & Bonneux (1998) studies. The same methods to derive prevalence rates and severity distributions were applied for 2003 Indigenous estimates.

Due to the lack of Indigenous-specific data on Parkinson disease and multiple sclerosis, national prevalence rates were applied to the Indigenous population to derive Indigenous prevalence for both 2011 and 2003 estimates.

Oral disorders

Mortality estimates

Oral disorder-related deaths were assigned from the NMD as defined by the disease list (Appendix Table A2). No deaths due to oral disorders were redistributed.

Morbidity estimates

Sequelae

Sequelae and health states assigned to mental and substance use disorders are included in Table 5.30. Durations and assumptions are outlined in subsections for individual diseases.

Table 5.30: Sequelae and health states for mental and substance use disorders

Disease	Sequela	ABDS 2011 health state identifier[(a)]
Dental caries	Untreated dental caries (including failed restorations)	199, 262
Periodontal disease	Chronic periodontal disease	198, 262
Severe tooth loss	Severe tooth loss	200, 262
Other oral disorders	Other oral disorders	200

(a) See Appendix Table C1.

Prevalence estimation

The prevalence of dental caries, periodontal disease and severe tooth loss in adults was based on analysis of the National Survey of Adult Oral Health 2004–06. This survey reported on dental caries apparent during a dental examination, which were measured as part of the DMFT (decayed, missing and filled teeth) index. For this index, DT (decayed teeth) scores indicate the number of dental caries, MT (missing teeth scores), the number of missing teeth, and FT (filled teeth) scores, the number of fillings. The number of adults with complete tooth loss was based on a self-report component of this survey.

Periodontal disease and severe tooth loss was not estimated in children aged less than 15 as it is relatively uncommon. Estimates of dental caries in children were based on analysis of the Child Dental Health Survey 2009, also using the DMFT measure (caries in deciduous and adult teeth were both counted).

Dental caries

Prevalence of dental caries was based on the proportion of people with a DT score greater than 1. This was then inflated to account for failed restorations (failed fillings) based on findings reported by Brennan & Spencer (2004).

Periodontal disease

Periodontal disease prevalence was based on cases of moderate–severe periodontal disease according to definitions developed by the Centers for Disease Control and Prevention/American Academy of Periodontology.

No periodontal disease was estimated in children aged less than 15, as chronic periodontal disease in children aged less than 15 years is relatively rare (Conway et al. 2014), and developmental changes reduce the accuracy of assessment of the disease in children (Jenkins & Papapanou 2001). A review of periodontal disease in children concluded that the prevalence and severity was very low in deciduous teeth (Jenkins & Papapanou 2001). Therefore the prevalence of chronic periodontal disease in children aged less than 15 was assumed to be 0.

All cases of periodontal disease were considered symptomatic. The health state reflects the intermittent nature of the symptoms.

Severe tooth loss

Severe tooth loss was based MT scores on the DMFT measure indicating fewer than 10 teeth remaining, or self-report for persons with complete tooth loss (edentulism).

For severe tooth loss, it was estimated that about 30% of cases were symptomatic, based on the proportion of people with no teeth or wearing dentures who had avoided food in the preceding 12 months (AIHW Dental Statistics and Research Unit 2008).

Other oral disorders

Estimates for other oral disorders were based on incidence of hospital separations in the 2011 calendar year. Any admissions to hospital that included the corresponding ICD-10-AM codes as principal diagnosis were counted. It was assumed that cases lasted an average of 4 weeks.

Subnational estimates

State/territory, remoteness and socioeconomic group-to-rate ratios from the National Survey of Adult Oral Health and Child Dental Health Survey were applied to national age and sex distributions for dental caries, periodontal disease and severe tooth loss. New South Wales and Victoria were not sampled in the Child Dental Health Survey 2009, so the national rates were applied to estimate the prevalence of children with dental caries in these states.

The prevalence of other oral disorders for subnational estimates used the same approach as for national but disaggregated directly according to remoteness area, socioeconomic group and state/territory.

2003 estimates

As the National Survey of Adult Oral Health data were collected in 2004–06, the same prevalence rates have been applied to the 2003 population structure to calculate prevalence of dental caries, periodontal disease and severe tooth loss in 2003. Differences in the prevalence of dental caries in children between the 2003–04 and 2009 Child Dental Health Surveys were incorporated into the estimates.

The prevalence of other oral disorders for 2003 used the same approach as 2011, but drawn from data in the 2003 calendar year.

Indigenous estimates

Due to the small sample size, age and sex-specific prevalence estimates for the Indigenous population were not directly available from the data sources used for national prevalence.

For 2011 and 2003, estimates for adult dental caries and periodontal disease were based on Indigenous-to-national rates ratios from the National Survey of Adult Oral Health 2004–06 applied to national age and sex distributions.

For 2011 prevalence of dental caries in Indigenous children, Indigenous-to-national rate ratios from the Child Dental Health Survey 2009 were applied to national age and sex distributions. For 2003, prevalence of dental caries in Indigenous children was based on Indigenous-to-national rate ratios from the Child Dental Health Survey 2003–04.

For severe tooth loss, Indigenous prevalence for 2011 and 2003 was based on data from AATSIHS 2012–13.

The 2011 and 2003 prevalence of other oral disorders among Indigenous Australians was based on analysis of the NHMD, adjusted for Indigenous under-identification using the standard adjustment factors described in Chapter 4 (see also Appendix tables C3 and C4).

Reproductive and maternal conditions

Mortality estimates

Deaths related to reproductive and maternal conditions were assigned from the NMD as defined by the disease list (Appendix Table A2). Deaths coded to N60, N61, N84–N90 and O94 were redistributed proportionately to cardiovascular diseases, cancer and other neoplasms, infectious diseases, respiratory diseases, and gastrointestinal disorders.

Morbidity estimates

Sequelae and health states

Sequelae, health states and durations for acute sequelae assigned to reproductive and maternal conditions are included in Table 5.31.

Table 5.31: Sequelae, health states and durations for reproductive and maternal conditions

Disease	Sequela	ABDS 2011 health state identifier[a]	Duration for acute sequelae
Maternal conditions			
Maternal haemorrhage	Anaemia due to maternal haemorrhage	195, 196	1–3 months
	Surgical intervention: caesarean section	194	2 weeks
Maternal infections	Maternal sepsis	194	2 weeks
	Other maternal infections	2	1 week
Hypertensive disorders of pregnancy	Hypertensive disorder	194, 207	2 weeks–2 months
Obstructed labour	Surgical intervention: caesarean section	194	2 weeks
Early pregnancy loss	Early pregnancy loss due to ectopic pregnancy	194	2 weeks
	Early pregnancy loss due to other causes	193	1 week
Gestational diabetes	Diagnosed gestational diabetes	207	4 months
Other maternal conditions	Surgical intervention: caesarean section	193	2 weeks
Reproductive conditions			
Endometriosis	Endometriosis	193, 194	3 days per month
Endometriosis	Infertility due to endometriosis[b]	50, 51	. .
Uterine fibroids	Anaemia due to uterine fibroids[c]	195, 196	6 months
	Infertility due to uterine fibroids[b]	50, 51	. .
	Symptomatic uterine fibroids	192	2–6 weeks
Genital prolapse	Faecal incontinence	48	. .
	Genital prolapse	192	. .
	Stress incontinence	261	. .
Polycystic ovarian syndrome	Infertility due to polycystic ovarian syndrome[b]	50, 51	. .
	Polycystic ovarian syndrome	207	. .
Infertility	Infertility[b]	50, 51	. .
Other reproductive conditions	Anaemia due to other reproductive conditions[c]	195, 196	. .
	Pain due to reproductive conditions	192	2 weeks

(a) See Appendix Table C1.

(b) Part of infertility envelope.

(c) Part of anaemia envelope.

Infertility envelope

Infertility was measured in males and females aged 20–49 seeking to have a child. As infertility is a sequela of multiple conditions across ABDS, the overall prevalence of infertility was calculated to ensure the sum of estimates for sequelae did not exceed the total—referred to as the 'infertility envelope'. To avoid double-counting, and adhere to mutually exclusivity for each disease, the total prevalence of infertility was estimated first, then the envelope was used to estimate prevalence of infertility sequelae by other diseases.

Diseases with infertility as sequelae include endometriosis, polycystic ovarian syndrome, uterine fibroids and sexually transmitted diseases (excluding HIV). The methods used to

estimate infertility due to these conditions are outlined in subsections for individual reproductive conditions.

Infertility sequelae estimates from other diseases were subtracted from this envelope. The remaining estimates were the prevalence of infertility as a disease.

Prevalence of infertility envelope, by sex

The number of women who underwent autologous fresh cycles in 2011 was derived from the Australian and New Zealand Assisted Reproductive Database. Estimates were inflated to account for varying types of assisted reproductive technology.

The number of men and women seeking assistance for infertility in 2011 was adjusted to account for individual people (rather than couples) using proportions of infertility due to the female, male or both partners published in the annual report.

As only 19.6% of people with infertility will seek assisted reproductive technology (Marino et al. 2011), the prevalence from the Australian and New Zealand Assisted Reproductive Database was inflated to derive the overall prevalence of infertility in 2011.

Age distributions, by sex, were derived from general practitioner encounters for infertility from the BEACH survey.

Prevalence of infertility envelope by subtype

Infertility was separated into primary and secondary infertility. These are definitions used by the GBD for health states and not clinical definitions of infertility (Table 5.32).

Table 5.32: GBD health states and lay descriptions for infertility

GBD health state	Lay description
Infertility: primary	Wants to have a child and has a fertile partner, but the couple cannot conceive
Infertility: secondary	Has at least 1 child, and wants to have more children. The person has a fertile partner, but the couple cannot conceive

A population-based estimate of the proportion of women with and without children by age who gave birth in 2010 was applied (Table 5.33), as it was unavailable for 2011. It is acknowledged that the distribution might slightly overestimate secondary infertility. As there is limited information on men with infertility, the same proportion as women was applied.

Table 5.33: Proportion of women who gave birth in 2010, by age and parity (per cent)

Age group (years)	No children (%)	1 or more children (%)
20–24	59.0	41.0
25–29	50.2	49.8
30–34	38.5	61.5
35–39	28.3	71.7
40–44	27.4	72.6
45–49	34.8	65.2

Infertility due to sexually transmitted infections (excluding HIV)

Due to the limited information on male infertility, infertility due to sexually transmitted infections was estimated in females only.

Based on clinical advice, it was assumed that about 90% of tubal factor infertility is caused by sexually transmitted infections. Current literature reports 7.0–9.8% of female infertility is attributable to tubal disease (Hafner & Pelzer 2011). This estimate (average 8.4%) was proportioned from the total infertility envelope to be due to other sexually transmitted infections (excluding HIV).

This disease was further proportioned based on GBD estimates into chlamydia (30%), gonorrhoea (20%) and other sexually transmitted infections (50%).

Prevalence estimation

Maternal conditions

Incidence of maternal conditions in 2011 were obtained from the NHMD (unless otherwise stated), with definitions based on ICD-10-AM or ACHI codes or from the Medicare Benefits Schedule. Early pregnancy loss was defined as losses (both spontaneous and surgically induced) before a gestational age of 20 weeks. Medical abortions performed via use of pharmaceuticals were not included due to data limitations.

As maternal conditions are generally measured in terms of incident cases, prevalence estimates were produced by applying a duration of health loss (Table 5.31). Durations to derive prevalence from incidence data were from the previous Australian burden of disease study (originally advised by the AIHW National Perinatal Statistics Unit), unless otherwise stated.

Maternal haemorrhage

Incidence of maternal haemorrhage was assumed to result in acute anaemia. Moderate anaemia was defined as cases of maternal haemorrhage including post-haemorrhagic anaemia (ICD-10-AM: D62), whereas mild cases did not indicate post-haemorrhagic anaemia.

It was assumed it would take 3 months to return to full health from mild anaemia. Severe cases would be treated with blood transfusion, with resulting anaemia lasting at most 1 month. Cases resulting in a caesarean section were given 2-week duration, consistent with surgical interventions with the same health state.

Maternal infections

Cases of maternal sepsis (defined as separations with a diagnosis of O41.1 and O85) were assumed to have health loss of 2 weeks. Other maternal infections—urinary tract infections, vaginitis and wound infections post-delivery—were assumed to have 1 week's health loss.

Hypertensive disorders of pregnancy

Moderate/severe hypertensive disorders (eclampsia and pre-eclampsia) were assumed to have 2 weeks health loss. Remaining hypertensive disorder estimates were given a duration of 2 months. If multiple hospitalisations occurred for this condition, this could have overestimated hypertensive disorders incidence.

Early pregnancy loss

Cases of early pregnancy loss due to ectopic pregnancy were derived from the NHMD.

Cases of surgically induced early pregnancy loss were derived from public patient hospital admissions for medical abortions, as well as relevant Medicare claims.

Adjustments for unclaimed procedures in New South Wales, Victoria, Tasmania and the Australian Capital Territory were applied to Medicare Benefits Schedule data (AIHW National Perinatal Statistics Unit 2005). Non-hospital claims were inflated by 13.1% to account for unclaimed procedures (Nickson et al. 2004). Public patient admissions were added to adjusted Medicare data to derive incidence of abortion in 2011.

It was assumed abortion was performed at 20 weeks or less, but as some state regulations allow this to be performed after 20 weeks, this might have resulted in a slight overestimate.

Due to data limitations, cases of spontaneous early pregnancy loss were restricted to hospitalised instances. This might result in an underestimate of health loss due to this sequelae.

Gestational diabetes

The incidence of gestational diabetes was estimated using the number of hospital separations where gestational diabetes (O24.4) was a diagnosis alongside a delivery (O80–O84). Prevalence-to-separations ratios derived from Western Australian linked data were used to adjust for multiple admissions per person.

Other maternal conditions

Remaining maternal conditions included placental disorders, labour complications and maternal care. An average duration of 2 weeks was applied to derive prevalence.

Reproductive conditions

Hospital data, longitudinal studies, general practitioner visits and epidemiological studies were used to derive prevalence. These sources require a diagnosis; therefore undiagnosed conditions were not included.

Endometriosis and polycystic ovarian syndrome

The prevalence of endometriosis and polycystic ovarian syndrome in women aged 35–39 were derived from the Australian Longitudinal Study on Women's Health, a longitudinal cohort study that collects data on the health of 40,000 women across Australia. The cohort used for prevalence estimates was born between 1973 and 1978. Age distributions, derived from general practitioner visits were applied to these estimates, to derive prevalence by age.

Endometriosis severity was based on surgical intervention. Hospitalised cases of endometriosis in 2011 with a relevant procedure were derived from the NHMD. Duration of health loss was assumed to be 36 days (based on the average duration of secondary dysmenorrhea of 3 days per month). Surgical cases were subtracted from the total prevalence to derive non-surgical cases.

Infertility estimates were derived from the Australian Longitudinal Study on Women's Health, with an estimated 11.7% of women with endometriosis and 14.5% with polycystic ovarian syndrome reported infertility issues. These estimates were subtracted from the infertility envelope, and this is further discussed in the infertility section.

Uterine fibroids

It was assumed people with burdensome uterine fibroids in 2011 would be hospitalised to remove fibroids. Therefore, incidence was derived from the NHMD based on ICD-10-AM codes with a relevant procedure.

Durations were based on surgical procedures. Abdominal hysterectomies received a duration of 6 weeks—due to more extensive recovery—while all other procedures received a duration of 2 weeks.

An estimated 2.5% of infertility was assumed to be due to uterine fibroids (Khaund & Lumsden 2008), and this was subtracted from the infertility envelope as previously described.

The proportion of women with uterine fibroids who had anaemia was based on the Uterine Bleeding and Pain Women's Research Study (Zimmerman et al. 2012). The average of the proportion of women with prolonged or heavy bleeding symptoms was used to apportion women with uterine fibroids experiencing anaemia. This proportion was applied to the burdensome uterine fibroids estimate, to derive the prevalence of anaemia due to uterine fibroids. The same severity distribution used for iron-deficiency anaemia was used to apportion mild anaemia and moderate anaemia.

Genital prolapse

Previous burden of disease studies estimated burden of genital prolapse in females only. The ABDS 2011 had a broader definition of genital prolapse, and included burden from rectal and anal prolapse, to estimate burden in both males and females.

Symptomatic genital prolapse

Prevalence rates of symptomatic prolapse in women from Tegerstedt et al. (2005) were applied to the Australian population, with modification to ages 20–29 and ages 85 and over, based on trends in hospitals data. Due to limited data, male estimates were calculated using the male-to-female genital prolapse hospitalisations ratio, with procedure codes related to genital prolapse.

Stress and faecal incontinence due to genital prolapse

Stress incontinence in males was assumed to be prostate related, so was not included. Estimates of stress incontinence from Tegerstedt et al. (2005) were applied directly to females symptomatic prolapse estimates.

Estimates of faecal incontinence from Jackson et al. (1997) were applied to female symptomatic prolapse estimates. Male estimates were derived using the age-specific male-to-female hospital separations ratio in 2011 for diagnosis of eneterocele or rectocele with surgical repair.

Other reproductive conditions

Remaining ICD-10 codes were categorised into whether they resulted in anaemia, pain, or both anaemia and pain, were captured elsewhere, or did not cause burden (Appendix Table D11). Conditions identified as resulting in pain, anaemia or both were included in estimations.

The BEACH study was used to derive prevalence by age, sex and sequela (pain and/or anaemia) using the proportion of general practice visits with these conditions. Ages under 15 were based on population distributions, and ages 75 and over were modelled on trend

analyses. The severity distribution of iron-deficiency anaemia was applied to anaemia (see anaemia envelope discussion in the blood and metabolic disease group).

Subnational estimates

Subnational estimates for most reproductive and maternal conditions were derived directly from the NHMD in 2011, or from age and sex ratios in the NHMD where direct derivation was not possible. State and territory estimates for abortions performed in non-hospital settings were derived from Medicare claims data, and adjusted to account for legislative differences.

2003 estimates

Estimates using hospital separations data used the same method as 2011, but with 2003 NHMD data. For gestational diabetes, however, due to changes in ICD-10 coding between 2003 and 2011, a different ICD-10 code (Z37) was used to identify deliveries.

Estimates using Australian Longitudinal Study on Women's Health, BEACH and epidemiological studies used the same rates or proportions as for 2011, applied to the 2003 population. This is because using earlier Australian Longitudinal Study on Women's Health surveys and BEACH data gave implausible estimates.

Indigenous estimates

The same methods and data sources were used to derive Indigenous estimates, except where noted. Indigenous estimates based on hospital separations data were adjusted for under-identification using standard adjustment factors (see Chapter 4 and Appendix tables C3 and C4).

Estimates for polycystic ovarian syndrome and endometriosis were based on Indigenous-to-national rate ratios applied to national prevalence rates. The rate ratio for endometriosis was obtained from hospital separations data, and from epidemiological studies for polycystic ovarian syndrome.

Due to lack of data, Indigenous prevalence of genital prolapse and infertility was obtained by applying the national distribution directly to the Indigenous population. This assumes the underlying rate is the same between the Indigenous and non-Indigenous populations.

Respiratory diseases

Mortality estimates

Deaths due to respiratory diseases were assigned from the NMD as defined by the disease list, and were based on the ICD-10 codes shown in Appendix Table A2.

Deaths due to respiratory failure were redistributed across all diseases using proportional allocation. Pneumonitis deaths were redistributed using the indirect MCOD method with all diseases in the target range. These redistribution methods are described in Chapter 3.

Morbidity estimates

Sequelae

Sequelae and health states assigned to respiratory conditions are included in Table 5.34. As most of these conditions (except for upper respiratory conditions) are chronic, health loss was assumed to apply for the whole year.

Table 5.34: Sequelae, health states and durations for respiratory diseases

Disease	Sequela	ABDS 2011 health state identifier[(a)]
Asthma	Asthma	52, 53, 54
Chronic obstructive pulmonary disease	Chronic obstructive pulmonary disease	55, 56, 57
Sarcoidosis	Sarcoidosis	262, 55, 56, 57
Interstitial lung disease	Interstitial lung disease	55, 56, 57
Pneumoconiosis	Asbestosis	55, 56, 57
	Other pneumoconiosis	55, 56, 57
	Silicosis	55, 56, 57
Upper respiratory conditions	Upper respiratory	207, 262
Other respiratory disease	Other respiratory	207, 262

(a) See Appendix Table C1.

Prevalence estimation

Asthma and upper respiratory conditions

The AHS 2011–12 and AATSIHS 2012–13 were the main data sources used to estimate the national and Indigenous prevalence of asthma and upper respiratory conditions. The AHS did not include people who lived in institutionalised facilities, such as hospitals or aged care facilities, so estimates for asthma and upper respiratory disease (mainly in the older age groups) were not possible. Additionally, the AHS did not report on *Very remote* areas, so a small proportion of the population is not covered.

To generate prevalence for the national population, rates derived from the surveys were applied to the national population.

Asthma

Prevalence of asthma was based on self-reported symptoms of diagnosed asthma in the previous 12 months. As this data source did not provide levels of control of asthma consistent with the available health states, the severity distribution was based on an Australian cross-sectional web-based survey (Reddel 2015).

Upper respiratory conditions

Upper respiratory conditions include hayfever, sinusitis and other upper respiratory tract disorders. Prevalence was derived from the proportion of participants who reported having an upper respiratory condition that had lasted or was expected to last at least 6 months. The total duration of health loss from upper respiratory conditions was assumed to be 3 months in the year.

Chronic obstructive pulmonary disease

Prevalence for chronic obstructive pulmonary disease was based on measured data from the Australian arm of the Burden of Obstructive Lung Disease (BOLD) Study (Toelle 2013), provided by the Woolcock Institute of Medical Research. This study involved a prevalence survey of nearly 3,500 randomly selected men and women aged 40 years and over. It was done in 6 locations around Australia between 2007 and 2010, and measured spirometric lung function after an inhaled bronchodilator was administered. Severity distributions were based on spirometric function in accordance with the BOLD study procedure (Buist et al. 2007).

Sarcoidosis, pneumoconiosis and interstitial lung disease

These conditions are rare and so their prevalence cannot be reliably estimated in population health surveys. Instead, prevalence estimates were based on mortality and hospitalisation data. Persons-to-separations ratios derived from Western Australian linked data were applied to the national hospitalisations to account for repeat admissions per person.

Mortality and hospitalisation data were used to estimate the prevalence of moderate and severe cases for these diseases. Prevalence for the mild or asymptomatic health states was then derived, by extrapolating these estimates for moderate and severe disease based on severity distributions used in GBD 2013 (GBD 2013 Collaborators 2015b).

Other respiratory conditions

The prevalence of other respiratory conditions was derived from the ratio of hospitalisations for other respiratory conditions relative to hospitalisations for identified conditions (asthma, upper respiratory conditions, chronic obstructive pulmonary disease, sarcoidosis, pneumoconiosis and interstitial lung disease) applied to the combined prevalence of the identified conditions. This assumes a similar hospitalisation rate for other respiratory conditions and the identified conditions.

Subnational estimates

National estimates were apportioned into each state/territory, remoteness area, and socioeconomic group, based on the proportions obtained from either survey or NHMD data.

2003 estimates

National 2003 estimates of asthma and upper respiratory conditions used a similar method as outlined for 2011, but drew on the 2004–05 NHS. Estimates of chronic obstructive pulmonary disease were also based on the BOLD study, with rates applied to the 2003 population. The remaining conditions used a similar method, but drew on 2003 hospital data.

Indigenous estimates

Indigenous estimates of asthma and upper respiratory conditions for 2011 and 2003 were based on self-reported data from the 2012–13 and 2004–05 National Aboriginal and Torres Strait Islander Health Surveys using similar methods as for national estimates. As there were no Indigenous-specific severity distributions, the national severity distributions were assumed.

Indigenous prevalence estimates and severity distributions for chronic obstructive pulmonary disease for 2011 and 2003 were based on the results of a cross-sectional BOLD study of the Indigenous population conducted in the Kimberley region of Western Australia (Cooksley et al. 2015) using age and sex distributions from hospitalisations data. As

hospitalisations and survey data indicate that Indigenous Australians experience chronic obstructive pulmonary disease at a younger age, estimates were adjusted to include prevalence in ages less than 40.

Sarcoidosis and intestinal lung disease are very rare in the Indigenous population (MacGinley & Allen 1997). Indigenous prevalence estimates for these conditions in 2001 and 2003 were based on hospitalisations and mortality data adjusted for under-identification using standard adjustment factors described in chapters 3 and 4.

Similarly, expert advice indicated that pneumoconiosis is also rare in the Indigenous population, and there are no data which show prevalence. Therefore, prevalence of pneumoconiosis for Indigenous Australians was assumed to be zero for both 2011 and 2003.

Skin disorders

Mortality estimates

Deaths related to skin disorders were assigned from the NMD as defined by the disease list (Appendix Table A2). Deaths coded to L04, L21–L25, L27–L30, L41–L45, L52–L53, L55–L60, L63–L85, L87, L90–L92, L94, L98.0, L98.1, L98.8 and L98.9 were redistributed proportionally to all diseases in the ABDS 2011.

Morbidity estimates

Sequelae and health states

Sequelae and health states assigned to skin disorders are included in Table 5.35. Where these conditions are chronic, health loss was assumed to apply for the whole year.

Table 5.35: Sequelae, health states and durations for skin conditions

Disease	Sequela	ABDS 2011 health state identifier[(a)]	Duration
Dermatitis and eczema	Eczema	204, 205, 262	12 months
Psoriasis	Psoriasis	204, 205, 262	12 months
Acne	Acne	201, 202, 262	12 months
Ulcers	Decubitus ulcer (pressure ulcer)	204, 205, 206, 262	Various, depending on stage of ulcer
	Other chronic skin ulcer	39	12 months
Skin infections (including cellulitis)	Severe skin infection	3	2 weeks
Other skin disorders	Other skin disorder: acute	3	2 weeks
	Other skin disorder: chronic	202	12 months

(a) See Appendix Table C1.

Prevalence estimation

Dermatitis and eczema

The prevalence of eczema was based on a study that conducted clinical examinations for non-malignant skin conditions in Australian adults living in central Victoria (Plunkett et al. 1999). The overall age-and-sex adjusted prevalence rate (31.6%) was applied to all age groups including children.

The severity distribution for dermatitis and eczema in adults was based on severity of atopic dermatitis from Plunkett et al. (1999), taking into account that severe atopic dermatitis was likely to be the only dermatitis or eczema condition that would correspond to the more severe health state.

The Marks et al. (1999a) study on atopic eczema in Australian school children (aged 4–18) was used to inform the severity distribution for children. The study reported 32.1% of cases were minimal, 54.1% mild, 12.6% moderate and 1.2% severe disease. Based on expert advice, the minimal and mild groups from Marks et al. were not considered to cause any health loss. Moderate and severe disease were aligned to the GBD health states using the same approach as outlined for adults (severe atopic dermatitis corresponds to the more severe health state).

Dermatitis and eczema was modelled as a chronic condition lasting the whole year.

Psoriasis

Prevalence was based on AHS 2011–12 self-reported psoriasis that had lasted, or was expected to last at least 6 months.

Severity was based on results from a study of GP and dermatologist patients with psoriasis (Jenner et al. 2002). People who spent 15–60 minutes each day treating their psoriasis were considered mild cases, and those who spent more than 1 hour each day were considered moderate–severe cases. Patients who spent 15 minutes or less on treatment each day were considered asymptomatic (minimal psoriasis). These proportions were then applied to the total prevalence estimates.

Acne

The prevalence of acne in adults was based on a study that conducted clinical examinations of non-malignant skin conditions in Australian adults living in central Victoria (Plunkett et al. 1999; Marks et al. 1999b). Plunkett et al. (1999) reported that the age- and sex-adjusted prevalence of acne was 12.8% in adults. The severity distribution was based on scores from the Dermatology Life Quality Index (Marks et al. 1999b).

The prevalence of acne in children was based on a study of clinical examination of Australian school students (aged 4–18) (Kilkenny et al. 1998). The prevalence of acne of the head and neck for students aged 4–18 was 36.1%. The severity distribution was based on scores from the Acne Disability Index (Marks et al. 1999b).

Ulcers

Pressure ulcers

There are 3 main populations at risk of developing pressure ulcers: patients admitted to hospital; older Australians and people with disability living in residential care facilities; and older Australians and people with disability receiving home-based support in the community. The prevalence of pressure ulcers was modeled separately for each of these populations based on different data sources (Table 5.36).

Table 5.36: Summary of data sources for modelling the prevalence of pressure ulcers, by key populations

Population at risk of pressure ulcers	Prevalence	Age distribution	Severity	Duration (if required)
Hospitals	Mulligan et al. 2011 Queensland Health 2012	SA Health 2007; VQC 2006	Mulligan et al. 2011; SA Health 2007; VQC 2006	Dealey et al. 2012 (adjusted for healing process with progressively reduced severity)
Low-care residential aged care	Mulligan et al. 2011	Santamaria et al. 2009	Santamaria et al. 2009	. .
High-care residential aged care	Santamaria et al. 2009	Santamaria et al. 2009	Santamaria et al. 2009	. .
Home-based care	Asimus & Li 2011 (adjusted for ulcers acquired in hospital)	Asimus & Li 2011	Asimus & Li 2011	. .

The prevalence of pressure ulcers in hospitals was based on the proportions of hospital-based pressure ulcers from the 2011 WoundsWest Wound Prevalence Survey report (Mulligan et al. 2011) and the State of Queensland (Queensland Health) 2011–12 annual report applied to the number of hospitalisations for Western Australia, New South Wales and Queensland in 2011 in the NHMD, and extrapolated to the remaining states/territories.

Age distributions were based on pressure ulcer point prevalence surveys (SA Health 2007; VQC 2006) and severity distributions were based on pressure ulcer stages reported for Western Australia, South Australia and Victoria (see Table 5.36).

Durations for each stage were based on mean expected time to heal, as reported by Dealey et al. (2012), with more severe ulcers modelled to include progression to less severe stages during healing. For example, it was estimated that a stage 4 ulcer would take 155 days to heal, and that this was made up of time spent in stages 3, 2 and 1 as healing progressed.

The prevalence of pressure ulcers in residential aged care was based on the proportions of pressure ulcers among low-care residential aged care residents (Mulligan et al. (2011) and high-care residential aged care residents (Santamaria et al. 2009) applied to the population in low and high-level residential care as at 30 June 2011, respectively. The age and severity distributions for both low-care and high-care residents were based on findings from Santamaria et al. (2009).

The prevalence of pressure ulcers in home-based care was based on an Australian study of patients receiving care from community nurses (Asimus & Li 2011) applied to the population in home-based care as at 30 June 2011. The study also reported the proportion of home-care patients who had acquired the ulcer during hospitalisation. This overlap with hospital prevalence was incorporated by reducing estimates by the proportion of home-care patients who had acquired the ulcer during hospitalisation to avoid double counting.

The number of people in residential care and home-based care were based on counts of people in community aged care (this included Community Aged Care Packages, Extended Aged Care at Home, and Extended Aged Care at Home Dementia) (AIHW 2014f).

Chronic skin ulcers

The prevalence of chronic skin ulcers was based on general practitioner encounters for chronic skin ulcers reported in the BEACH study. The crude rates from the study were

weighted and modelled according to the method by Harrison et al. (2013) to estimate the prevalence of chronic conditions. This estimate took into account the frequency of GP visits in the population, and people who did not visit a general practitioner, so that results could be generalised to the total population. This estimate also accounted for potential double-counting of ulcers caused by diabetes.

Skin infections

The prevalence of skin infections was based on hospital admissions (NHMD) in 2011. Admissions with a principal diagnosis for skin infections (ICD-10-AM: A46, B08.1, B08.4, B86, H00.0, H60.0-H60.1, J34.0, L00–L04, L08.0–L08.9) were included, with an assumed duration of 2 weeks.

Other skin disorders

Other chronic skin disorders were based on prevalence from AHS 2011–12 conditions reported as 'other disease of skin and subcutaneous tissue' or 'symptoms, signs involving skin and subcutaneous tissue'. It was estimated that about half of these conditions would correspond to other skin disorders as defined in the ABDS 2011. Age distribution was not sex specific, and the conditions were considered to last the entire year.

Other acute skin disorders were based on hospitalisations (NHMD), with an assumed duration of 2 weeks.

Subnational estimates

The national prevalence rates by age and sex were applied to each state/territory, remoteness area and socioeconomic group for dermatitis and eczema, psoriasis, acne, and ulcers. Due to the low number of hospitalitalisations and high relative standard errors in each age group, subnational estimates for skin infections and other skin disorders were derived from smoothed national data using proportions contributed by each group from hospitalisations data.

2003 estimates

The 2011 prevalence rates for dermatitis and eczema, and acne were applied to the 2003 population. For the remaining conditions, the corresponding 2003 data source was used, including the 2004–05 NHS, 2002–03 BEACH, and NHMD analysis from 2003 data.

Indigenous estimates

Due to a lack of available data, the national prevalence rates were applied to the Indigenous population to produce Indigenous prevalence estimates of dermatitis and eczema, and acne for 2003 and 2011.

For psoriasis, Indigenous estimates were obtained using the AATSIHS 2012–13 for 2011 estimates, and the NATSIHS 2004–05 for 2003 estimates.

Prevalence for skin infections for the Indigenous population in 2011 and 2003 were estimated using the NHMD, and adjusted for Indigenous under-identification using adjustment factors described in Chapter 4 (see also Appendix tables C3 and C4).

For ulcers, hospitalisation rate ratios (Indigenous-to-national) based on Indigenous hospitalisations adjusted for under-identification, were applied to the national prevalence rates. To estimate the prevalence of other skin disorders in the Indigenous population, the

same methods were used as for the national estimates using the AATSIHS 2012–13 and NATSIHS 2004–05.

Section II: Estimating the burden due to key risk factors

A risk factor is any determinant that causes (or increases the likelihood of) one or more diseases or injuries. As well as providing estimates of fatal and non-fatal burden, burden of disease method allows death and disability to be attributed to specific underlying (or linked) risk factors. Quantification of the impact of risk factors assists in making evidence-based decisions about where to direct efforts to improve population health and prevent disease and injury.

This section describes the method used to quantify the impact of risk factors in the ABDS 2011.

Key terms used in this chapter

attributable burden: The disease burden attributed to a particular risk factor. It is the reduction in burden that would have occurred if exposure to the risk factor had been avoided or had been reduced to its **theoretical minimum risk exposure distribution**.

counterfactual: An alternative risk factor exposure distribution chosen for comparison with the observed distribution, in order to estimate the alterable contribution of that risk factor to the burden of disease. The most commonly used counterfactual in burden of disease studies is the theoretical minimum risk exposure distribution (TMRED).

effect modification: A change in the observed magnitude or direction of an association between a risk exposure and an outcome when a third variable (such as age or sex) is included in the analysis.

effect size: A statistical measure of the strength of the relationship between 2 variables (in this context, between a risk exposure and a disease outcome), expressed, for example, as a relative risk or odds ratio.

risk exposure distribution: The measure of the spread or distribution of exposure to the risk factor in the population that have encountered or experienced, or have the risk factor.

linked disease: A disease or injury for which there is evidence that its likelihood is increased by the risk factor in question.

population attributable fraction (PAF): For a particular risk factor and causally linked disease or injury, the percentage reduction in burden that would occur for a population if exposure to the risk factor was avoided or reduced to its theoretical minimum.

relative risk (RR): The risk of an event relative to exposure, calculated as the ratio of the probability of the event occurring in the exposed group to the probability of it occurring in the non-exposed group.

risk factor: Any factor that causes or increases the likelihood of a health disorder or other unwanted condition or event.

risk–outcome pair: Associates a condition in the disease list with a known risk factor for that condition.

theoretical minimum risk exposure distribution (TMRED): The risk factor exposure distribution that will lead to the lowest conceivable disease burden.

6 Overarching methods and choices for risk factors

The burden attributable to selected risk factors is generally estimated using population attributable fractions (PAFs) applied to the disease burden estimated, as per the previous chapters. If PAFs appropriate to the disease and population in question are available from a comprehensive data source (such as a disease register), they are applied directly. If not, PAFs are created using the comparative risk assessment method that has become standard practice in burden of disease risk factor analysis globally (Lim et al. 2012).

The comparative risk assessment method is a 5-step process:

1. Select risk–outcome pairs.
2. Estimate the population-level distribution of risk factor exposure.
3. Estimate the effect of risk factors on disease outcomes.
4. Define the counterfactual (theoretical minimum risk exposure distribution—TMRED).
5. Calculate the population attributable fraction.

Selection of risk factors

The risk factor list details the specific risk factors to be quantified as underlying causes of the estimated burden through their causal association with particular diseases. In contrast to the disease list, which is exhaustive, and where an established classification system (the ICD) exists, the list of potential risk factors is near limitless, and there is often no consensus in the literature on what level(s) of exposure constitute 'risk'. A predetermined set of criteria is used to help develop the list, taking into account the potential for modification of exposure in the population, the availability of data on exposure, and the quality of evidence about the presence and magnitude of causal effects.

To be included in the ABDS 2011, a risk factor had to satisfy one or more of the following criteria:

Included in other studies' risk factor lists

- Have been included in:
  - the GBD 2010
  - the ABDS 2003

  unless its inclusion in ABDS 2011 conflicted with other criteria.

Significant impact and policy interest

- Be of significant importance to national or Indigenous disease burden based on previous studies (GBD 2010; ABDS 2003).
- Be of substantial Australian or Indigenous health policy interest—defined as currently being the focus of policy concern, professional attention or monitoring activity.
- Be modifiable, and able to be prevented or modified through policy intervention.

Be able to be measured

- Be measureable, including having:
  - sufficient evidence for causal association between exposure and health outcomes based on high-quality epidemiological studies
  - enough data and methods to enable exposure distributions to be estimated
  - enough data to estimate outcome-specific effects sizes per unit of exposure
  - evidence to support the ability of effect sizes to be generalised to populations, other than those included in the available studies, or satisfactory models for extrapolating them
  - resources required to compile the required data.

ABDS 2011 risk factor list

The ABDS 2011 identified 30 risk factors for analysis, which were broadly grouped into categories (behavioural, metabolic, environmental and dietary risks). They included sun exposure, which was not included in either GBD 2010 or ABDS 2013 as per the criteria, but has significant impact and is of policy interest. The risk factors included in ABDS 2011 are listed in Appendix Table E2.

Because of the high and complex interrelatedness of the risk factors within these groups causing bias, risk factors were analysed and reported individually; however, a combined estimate was included in this study for all risk factors and for all dietary risk factors using the multiplicative method to estimate the effect of multiple risk factors (described in section 'Combined risk factor analysis' in this chapter).

Risk factors not included in ABDS 2011

Suboptimal breastfeeding, childhood underweight and exposure to lead (included in GBD 2010) were not included in ABDS 2011. Suboptimal breastfeeding was linked in the global studies to intestinal infection diseases that are not common in Australia. Childhood underweight, although considered important in the Aboriginal and Torres Strait Islander population, was not included as the effect sizes available from GBD 2010 were sourced from developing countries and related to infectious diseases only, failing to capture the increased likelihood of chronic disease later in life due to low birthweight (Hoy et al. 2010). Low birthweight was included as a disease ('pre-term birth and low birthweight complications') rather than as a risk factor in this study, which is consistent with recent global studies. Exposure to lead was also excluded because exposure data were not available for Australia.

Social determinants of health (the economic and social conditions—such as income, level of education and employment status—that influence health status) could not be included as risk factors in the current study, due to the resources needed to undertake the large and complex body of work that would be required (such as developing appropriate definitions directly related to health, and sourcing disease-specific relative risks). Estimating exposure to social determinants is further complicated by the fact that their impact can accumulate over the life course, and their effect might continue to be felt throughout a person's life and even across generations (Atkinson et al. 2010; Zubrick et al. 2010). The AIHW recognises this as an important area of work for future burden of disease studies.

Selection of risk–outcome pairs

A risk–outcome pair associates a condition in the disease list with a known risk factor for that condition. For example, high fasting plasma glucose is a risk factor for diabetes, coronary heart disease, cerebrovascular disease and chronic kidney disease. In this report, such associations are described as diseases or injuries being 'linked to' that risk factor. Thus, high fasting plasma glucose and diabetes constitute a risk–outcome pair; high fasting plasma glucose and coronary heart disease constitute another pair. The risk factors and linked diseases selected for inclusion in the ABDS 2011 are shown in Appendix Table E2.

Risk–outcome pairs were included where there was sufficient evidence of a causal link. This is defined as having convincing or probable evidence measured against the same criteria employed in GBD 2010. This was based on World Health Organization modifications to the World Cancer Research Fund grading system:

- Convincing evidence—evidence based on epidemiological studies showing consistent associations between exposure and disease, with little or no evidence to the contrary. The available evidence is based on a substantial number of studies including prospective observational studies, and, where relevant, randomised controlled trials of sufficient size, duration, and quality, showing consistent effects. The association should be biologically plausible.
- Probable evidence—evidence based on epidemiological studies showing fairly consistent associations between exposure and disease, but for which there are perceived shortcomings in the available evidence, or some evidence to the contrary, which preclude a more definite judgment. Shortcomings in this evidence might be any of the following: insufficient duration of trials (or studies); insufficient trials (or studies) available; inadequate sample sizes; or incomplete follow-up. Laboratory evidence is usually supportive. The association should be biologically plausible.
- Possible evidence—evidence based mainly on findings from case-control and cross-sectional studies. Insufficient randomised controlled trials, observational studies, or non-randomised controlled trials are available. Evidence based on non-epidemiological studies, such as clinical or laboratory investigations, is supportive. More trials are needed to support the tentative associations, which should be biologically plausible.
- Insufficient evidence—evidence based on findings of a few studies that are suggestive, but insufficient to establish an association between exposure and disease. Little or no evidence is available from randomised controlled trials. More well-designed research is needed to support the tentative association.

Source of risk–outcome pairs

For those risk factors selected for inclusion in this study, the ABDS 2011 has adopted the available relevant risk–outcome pairs used in the GBD 2010 (United States Burden of Disease Collaborators 2013). No additional linked diseases were included from the GDB 2013 because the release of that report was too late for inclusion in this study. Risk–outcome pairs for sun exposure (not included in GBD 2010) were included, after consulting Australian experts.

The risk–outcome pairs were spread across 13 disease groups. Some risk factors had only a single disease risk–outcome pair, while others had many outcomes within the 13 disease groups.

Population distribution of exposure

A clear and consistent definition of risk factor exposure is key to estimating the proportion of the population 'at risk'. For the ABDS 2011, the definitions of risk factor exposures have been adopted where possible from the GBD 2010 (Lim et al. 2012).

All potential data sources to estimate exposure (whether published or unpublished) were assessed for comparability, relevance and representativeness, currency, accuracy, validation, credibility and accessibility/timeliness (see Appendix E for the data selection criteria and Appendix Table E1 for scoring matrix). Only data sources that met these criteria were included in the study.

Estimates of Australian and Indigenous population distributions of risk factor exposure by age and sex have been based on a variety of data sources:

- Australian Health Survey 2011–12 (AHS) (national estimates only)
- Australian Aboriginal and Torres Strait Islander Health Survey (AATSIHS) 2012–13 (Indigenous estimates only)
- National Drug Strategy Household Survey 2010
- ABS apparent consumption of alcohol data (national estimates only)
- Kirby Institute annual surveillance reports
- National HIV Register
- AIHW Cancer Registry
- State-based air monitoring stations
- National Homicide Monitoring Program
- ABS Personal Safety Survey 2012 (national estimates only).

Some risk factors (such as tobacco smoking) had several different measures or definitions of exposure. For tobacco smoking these included current smoking, exposure to second hand smoke, and past smoking. These 3 measures of exposure are mutually exclusive for tobacco, and can be summed. Other risk factors with more than 1 measure of exposure that can be summed include alcohol use, drug use and occupational risks.

The risk factor exposure is measured as either a:

- categorical variable (with a set number of mutually exclusive categories)
- continuous variable.

Some categorical risk factors are measured through relatively straightforward dichotomous descriptions (for example, the proportion of mothers who smoked during pregnancy versus the proportion who did not). For other risk factors, broad categories are used, such as the proportion of the population (by age and sex) falling into standardised categories of physical activity.

However, the majority of risk factors are measured as continuous variables, and the PAF calculations require the population prevalence per unit of exposure (for example, the

observed population distribution of systolic blood pressure per millimetre of mercury), by age and sex.

Some previous burden of disease studies used a modelled risk exposure distribution rather than the empirical data themselves. They have, for example, taken the observed mean and standard deviation of exposure to a risk factor in the population, then modelled the exposure distribution using a normal or a lognormal function with that mean and standard deviation.

For the ABDS 2011 study, empirical survey data were used where possible to determine the distribution of exposure to risk factors. The data were derived from the sources described in Appendix Table E3. The proportion of the population exposed to each risk factor level was estimated in accordance with the finest exposure increments supported by the data source.

Where data were sourced directly from a survey (for example, AHS 2011–12), they were extracted at such granularity as to ensure that the relative standard error for the majority of cells was 25% or less. Sex, age groups or exposure categories were combined into larger cells to conform to this principle as necessary; however, for a small number of age and sex categories, it was necessary to accept estimates with relative standard errors of 25–50%.

Estimates of effect size (relative risks)

Burden of disease studies use relative risks to measure the strength of causal association between risk factors and the linked disease outcomes. The ABDS 2011 adopted relative risks released by the GBD 2010, except when they were inappropriate or not available (US Burden of Disease Collaborators 2013). The GBD relative risks used were judged appropriate for the United States of America, and mostly appropriate for Australia, as both are high-income countries.

Some relative risks for dietary risk factors were sourced from the GBD 2013, but only for diseases linked in the GBD 2010 (GBD 2013 Risk Factors Collaborators 2015). The relative risks for injuries linked to alcohol use were from Taylor et al. (2010).

The relative risks from the GBD 2010 for infectious diseases such as hepatitis C, hepatitis B, HIV/AIDS and tuberculosis were not considered appropriate for Australia because control mechanisms exist in Australia for these conditions. There is also direct evidence data available from the Kirby Institute that details the number of cases of these conditions caused by the risk factor (unsafe sex or drug use) (Kirby Institute 2012). These data were used directly to inform estimates of effect size instead of the comparative risk assessment method.

Direct evidence from the National Homicide Monitoring Program (Bryant & Cussen 2015) was used for homicides linked to intimate partner violence. The AIHW is doing further work to review and refine the risk–outcome pairs and estimates of effect sizes used to measure attributable burden due to intimate partner violence in Australian women (Lum On et al. 2016; Ayre et al. forthcoming).

Effect sizes used in the GBD 2010 were adjusted for confounders ('parallel' risk factors), but not for factors that occur successively along the causal pathway. For example, relative risk of ischaemic heart disease due to physical inactivity was not adjusted for high blood pressure, as these risk factors occur along the same causal pathway. This means the estimates of their effects cannot be added together.

For continuous risk factors, the distribution of relative risks across the required levels of exposure were determined by applying a linear relationship to the available units of measure

for each risk factor and the published relative risks by age and sex, except for diet high in fruit and diet high in vegetables, which had multiplicative curves, as advised by experts.

The relevant relative risk to apply to each exposure category was determined as the relative risk for the mid-point of that category. For example, for the proportion of the population who ate 80–120 grams of fruit, the relative risk for 100 grams was applied. When the exposure category included an open-ended range, the limit of the exposure range was used. For example, for the proportion of the population who ate 400 grams or more of fruit, the relative risk for 400 grams was applied.

Theoretical minimum risk exposure distribution

The estimated contribution of a risk factor to disease burden is calculated by comparing the observed risk factor distribution with an alternative, hypothetical distribution (the counterfactual scenario). This could be an increase or decrease in levels of exposure, or changes in behaviour compared with what is currently observed in the population. In the ABDS 2011, as in previous burden of disease studies, a TMRED scenario was adopted. This involved determining the hypothetical exposure distribution that would lead to the lowest conceivable disease burden.

For some risk factors the choice of TMRED is obvious, as it involves no exposure to risk—for example, all people are lifelong non-smokers, or all people are highly active. However, for many risk factors, no exposure is not appropriate, either because it is physiologically impossible (for example, blood pressure or body mass index), or because there are lower limits beyond which exposure cannot feasibly be reduced (for example, air pollution). In these cases, epidemiological evidence is used to determine the optimal level of exposure, which reflect either the lowest level at which a dose–response relationship can be observed within a meta-analysis of cohort studies, or the lowest risk factor exposure distribution observed globally (Lim et al. 2012). The counterfactual then becomes a narrow distribution around the optimal level. For example, based on a meta-analysis of global studies, the counterfactual distribution for high body mass index is based on a population mean of a body mass index of 21–23 kilograms/metre^2 with a standard deviation of 1.

Where the TMRED is a range, exposure to risk is not dichotomous (that is, at risk or not at risk). In this situation, the measure of attributable burden cannot be estimated by simply comparing each level of exposure in the population with the endpoints. Instead, to determine how much burden each exposure level contributes compared with TMRED, the range of the level of exposure is compared with its relative position in the range of the TMRED. The level of exposure is then adjusted to incorporate the range of TMRED.

For example, the TMRED of fasting blood plasma glucose is 4.9 to 5.3 mmol/L. The fasting blood plasma glucose exposure in the population is adjusted by 0 at the lowest level of exposure and 0.4 (the range of the TMRED) at the highest level of exposure. At 3.1 mmol/L (point A) the exposure is adjusted by 0.015, from 3.1 to 3.065 mmol/L, while at 10.0 mmol/L (point B), exposure is adjusted by 0.38 (from 10.0 to 9.62 mmol/L). The adjusted exposure is then compared with a point in the TMRED relative to its position in the TMRED range. In this case, point A is compared with a TMRED of 4.915 (4.9+0.015) mmol/L, while point B is compared with a TMRED of 5.28 (4.0+0.38) mmol/L. This example is shown in Figure 6.1.

The TMREDs developed as part of the GBD 2010 study have been adopted for the ABDS 2011 (Lim et al. 2012), except for a diet high in sodium, where a higher TMRED was used (1.6 grams instead of 1 gram), based on new evidence in the literature and advice from

nutrition experts (R Stanton 2016, pers. comm., 5 February; A Lee 2016, pers. comm., 8 February). A panel of dietary experts provided advice on an appropriate TMRED for this risk factor.

Additionally, the TMRED for low bone mineral density was adopted from the more recent GBD 2013 (US Burden of Disease Collaborators 2013).

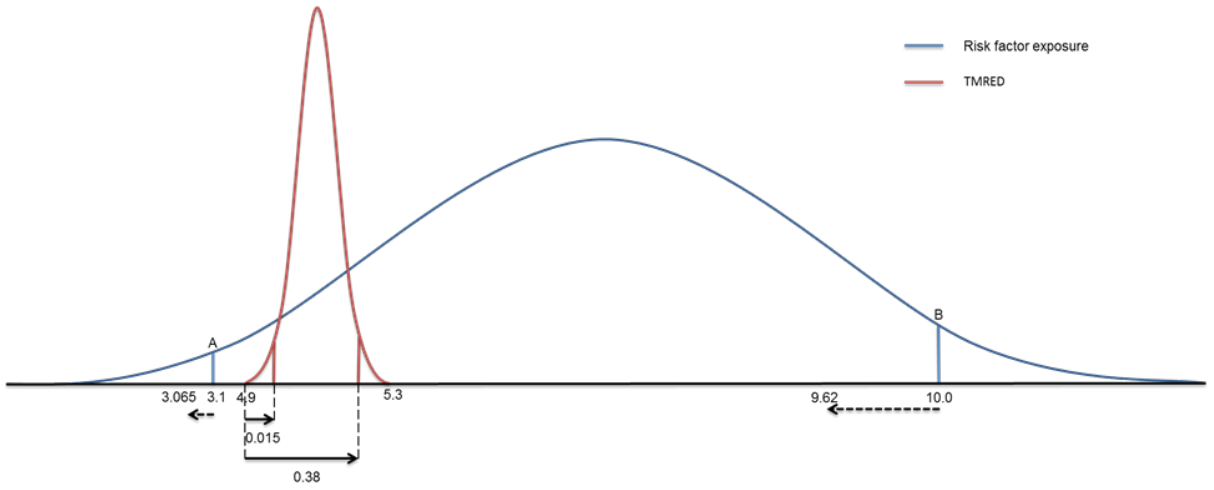

Figure 6.1: Comparing exposure with TMRED for fasting blood plasma glucose

Calculation of population attributable fractions

PAFs determine the proportion of a particular disease that could have potentially been avoided if the population had never been exposed to a risk factor (or, rather, had been exposed to TMRED levels). PAFs were calculated for each risk–outcome pair by sex and age group.

The calculation of PAFs requires the input of the relative risk (RR) and prevalence of exposure in the population (P):

$$PAF = \frac{P(RR - 1)}{P(RR - 1) + 1} \times 100$$

When the risk factor has multiple categories of relative risks and exposure levels, the following formula is used:

$$PAF = \frac{\sum_c P_c\,(RR_c - 1)}{\sum_c P_c\,(RR_c - 1) + 1} \times 100$$

where:

$\sum_c$ is the sum over all categories

c is an index for category

P is prevalence

RR is relative risk.

Direct population attributable fractions

For some risk–outcome pairs, direct evidence is used to calculate the PAF. This is used:

- for risk–outcome pairs where there is evidence from high-quality data sources to attribute a disease outcome to a risk factor in Australia. It is important that the estimate captures all cases of the disease outcome in Australia. An example is the HIV register which collects data on the risk factor exposures that cause HIV (unsafe sex and/or drug use). The direct PAF is calculated as the proportion of the outcome caused by the risk factor
- when exposure to the risk factor is necessary to have the outcome—for example, all of the disease outcome 'alcohol use disorders' is attributable to the risk factor 'alcohol use'. In this case the PAF is 1, where all of the disease outcome is attributed to the risk factor.

Change in PAF over time

To calculate the change in PAF between 2003 and 2011, the percentage change in median age-adjusted PAF is calculated by weighting the age and sex-specific PAF for each risk factor and linked disease by the 2001 standard population. The median PAF from all the linked disease PAFs is determined for each risk factor. The change in PAF from 2003 to 2011 is then calculated as the percentage change in the median PAF.

Calculating the attributable burden

The burden attributable to each risk factor is calculated by applying the PAF for each risk–outcome pair to the relevant YLL and YLD. This is shown in Figure 6.2.

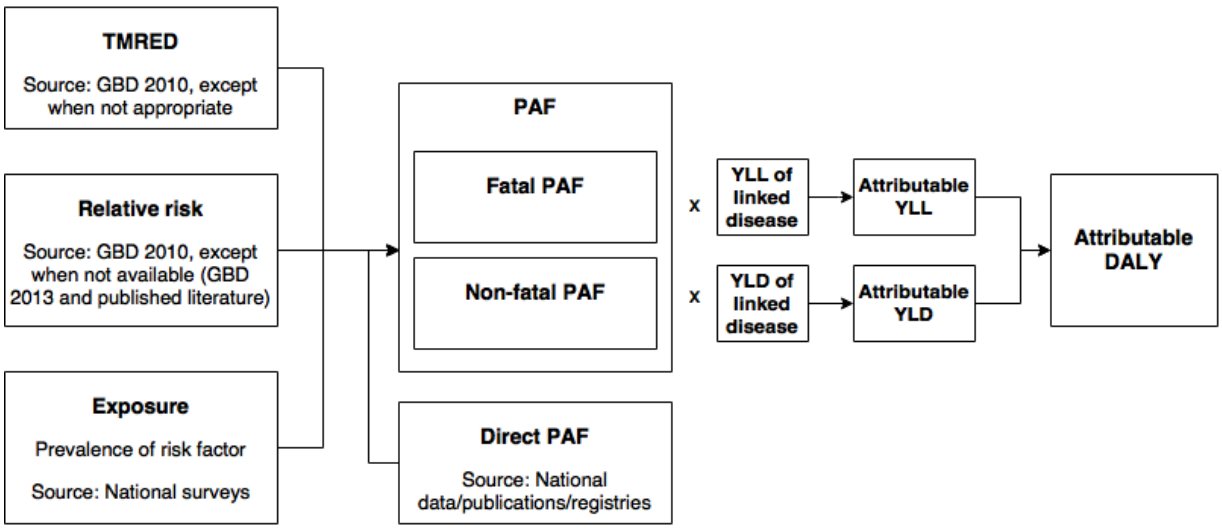

Figure 6.2: Inputs and processes to calculate attributable burden

Attributable DALY for each risk factor and linked disease is calculated at the disease level (for each age and sex), described mathematically as:

$$\text{Attributable DALY} = \sum_i (PAF_{YLDi} \times YLD_i) + (PAF_{YLLi} \times YLL_i)$$

where:

$\sum_i$ is the sum over all diseases linked with that risk factor

PAF_{YLDi} is the morbidity population attributable fraction for disease i

YLD_i is the non-fatal burden of the linked disease i

PAF_{YLLi} is the mortality population attributable fraction for disease i

YLL_i is the fatal burden of the linked disease i

for each risk factor.

Applying PAF to the ABDS disease list

A small number of linked diseases and injuries sourced from GBD 2010 did not align to the ABDS disease list (Appendix Table E4). This was due to GBD disaggregating causes to a further level—for example, stroke, which was estimated as a single disease in ABDS 2011, only had relative risks from GBD 2010 for ischaemic and haemorrhagic stroke. These could not be applied directly to the single stroke burden.

To adjust for this, data were used from a range of sources to identify the proportion of the prevalence of the ABDS disease corresponding to the available relative risk. For example, Thrift et al. (2009) found haemorrhagic stroke to be 22.4% of strokes in Australia. Where such disaggregation was unavailable from published literature, the proportion of fatal/non-fatal burden for these diseases in Australia from GBD 2010 was used. Appendix Table E4 describes the source of any such disaggregation and the proportion used.

A limitation of this approach is that the proportion of prevalence does not always equate to the proportion of the burden represented by the GBD cause, and this might vary by fatal and non-fatal burden.

The PAFs for each risk factor were calculated at the GBD cause level (disaggregated level). The PAFs were multiplied by the proportion of the ABDS disease it represented and applied at the ABDS disease level to calculate the attributable DALY. For example, the PAF for haemorrhagic stroke was multiplied by 0.224 before being used to calculate the attributable DALY.

Combined risk factor analysis

The burden from different risk factors for a particular disease cannot simply be added together, because:

- some risk factors are on the same causal pathway—for example, a diet high in sweetened beverages increases the likelihood of high body mass
- the PAFs are estimated independently—similar to issues with comorbidity, the burden due to each risk factor for a given disease might exceed the total burden of that disease.

The combined effect of multiple risk factors must account for the bias introduced by the complex pathways and interactions between many risk factors.

Firstly, to account for risk factors on the same causal pathway, an attenuation factor of 50% was used (as specified by the WHO) for risk factors that are secondary to other factors in the same causal pathway (Ezzati et al. 2004).

For example, to reflect the causal pathway of high intake of sweetened beverages increasing the risk of high body mass (which, in turn, increases the risk of stroke), the PAF for high body mass causing stroke is attenuated by 50%. This provides the necessary independence assumption required for step 2.

Secondly, to prevent the combined disease burden exceeding the total burden for a given disease, the combined burden of more than 1 risk factor was estimated using the following formula:

$$PAF_i = 1 - \prod_r (1 - PAF_{ir})$$

where

- PAF_i is the population attributable fraction of burden attributable to a particular disease from those risk factors being combined, such as all risk factors or all dietary risk factors
- i is the linked disease
- r is the individual risk factor for a linked disease being combined
- PAF_{ir} is the population attributable fraction for risk factor r for linked disease i
- Π is the product over all risk factors r.

This formula, which has been used in several other studies, has the desirable property of placing a cap on the estimated combined attributable burden, so avoids the possibility of exceeding 100% of the total burden of disease. However, it assumes that risk factors are independent—that is, it does not take into account risk factors that are in the same causal pathway.

A combined estimate was included in this study for all risk factors and dietary risk factors. The risk factors and outcome pairs that were adjusted can be found in Appendix Table E5.

Combined estimates were not calculated for other risk factors categories, such as metabolic risk factors, because within these categories there is an increase in the complex interrelation of the risk factors causing bias compared with all risk factors combined.

Further evidence and investigations are needed to account for the interactions between separate risk factors.

2003 estimates

Where possible the burden attributable to risk factors was calculated for 2003. Exposure distributions for air pollution, childhood sexual abuse, dietary risk factors (except fruit and vegetables), high fasting plasma glucose, iron deficiency and sun exposure could not be measured for the 2003 estimates due to lack of available input data comparable with the methods used for the 2011 estimates.

The way exposure was estimated for these risk factors in 2003 is described under the individual risk factors in Chapter 7. The same relative risk–outcome pairs were included as for the 2011 estimates.

Indigenous estimates

For the Indigenous population, the same risk factor list was used as for the total Australian population, with 2 exceptions: unimproved sanitation, which was included for Indigenous estimates only; and high sun exposure, which was included for national estimates only. The burden from unimproved sanitation was not estimated for the non-Indigenous population due to lack of available exposure data, and was assumed to be close to 0. The burden from sun exposure was not estimated for the Indigenous population as it was not possible to account for the impact of differences in skin melanin levels.

The same risk–outcome pairs, relative risks and TMREDs as used for national estimates were used for Indigenous risk factor estimates. Relative risks specific to the Indigenous population were not available.

Exposure distributions for some risk factors could not be measured for the 2003 Indigenous estimates due to lack of available input data comparable with the methods used for the 2011 estimates. These were air pollution, dietary risk factors (except fruit and vegetables), high fasting plasma glucose, iron deficiency, childhood sexual abuse, and unimproved sanitation. As a result, these risk factors were not included in the 2003 Indigenous estimates.

7 Individual risk factor methods

This chapter describes in detail the methods unique to each risk factor included in the ABDS 2011, with a focus on the calculation of exposure estimates, as this was the aspect of risk estimation most influenced by Australia-specific data and other evidence.

Chapter 6 describes the overall method used to calculate the PAF and attributable burden, including the selection of risk–outcome pairs (linked diseases), estimation of effect sizes (relative risks), and assumptions for TMREDs (See also Appendix tables E2 and E3).

The same methods used to calculate the 2011 national estimates were also generally used to calculate attributable burden in 2003 and for the Indigenous population. Any exceptions to this approach are described within each risk factor's methods.

Most risk–outcome pairs and relative risks were sourced from GBD 2010. The few exceptions are noted in Chapter 6.

Most TMREDs were also sourced from GBD 2010, with exceptions described in Chapter 6.

Exposure to risk factors in the past can influence the proportion of burden in the reference year. For risk factors such as tobacco use, occupational risks, alcohol use, childhood sexual abuse, drug use, and unsafe sex, the burden might continue to exist from past high exposure levels. Where evidence of past exposure can be linked to current burden, this is included in the analyses, and described under the individual risk factor.

For some risk factors, such as high body mass, current exposure might have an impact on future burden. This is not accounted for in this study as the burden pertains to the reference year.

Not all risk factors are relevant to all population (age and sex) groups. For example, the bulk of the burden from high cholesterol occurs for people aged 25 and over, and the bulk of the burden from intimate partner violence occurs in females aged 15 and over. The population relevant to each risk factor was informed by GBD 2010. The population group for which attributable burden from a given risk factor has been estimated is described in each section.

Also, for most risk–outcome pairs in the study, both fatal and non-fatal burden are relevant. For others such as air pollution only fatal burden has been estimated. The choices for population groups and type of burden (fatal or non-fatal) were informed by GBD 2010.

Tobacco use

The impact of tobacco use was measured in people aged 25 and over; it captures the burden attributable to current smoking, past smoking and exposure to second-hand smoke in the home.

Population attributable fraction

Exposure estimates

The National Drug Strategy Household Survey (NDSHS) 2007 was used to estimate the proportion of the population who are current and former smokers. The NDSHS 2010 was used to estimate the proportion of non-smokers exposed to environmental tobacco in the home.

For cardiovascular diseases, diabetes, asthma and respiratory infections, exposure to tobacco was calculated from the proportion of individuals in the 2007 NDSHS who reported smoking daily, weekly or less than weekly. Using these data allows for a 5-year lag between exposure and these disease outcomes.

Due to the much longer lag between smoking and the incidence of cancers and chronic respiratory conditions, as well as consistent reductions in smoking rates over recent decades, the tobacco attributable burden for those disease outcomes cannot be estimated from data on the current or relatively recent prevalence of smoking. For these conditions, the 'smoking impact ratio' (described by Peto et al. 1992) was used as an indirect method to estimate the accumulated risk from tobacco smoking. Lung cancer mortality in both references years (by age and sex) was compared with lung cancer mortality rates among a cohort of smokers and never-smokers in a long-term study conducted in the United States (Peto et al. 1992). The excess mortality seen in the Australian population, compared with the cohort of non-smokers, is used to determine the proportion of the population living with accumulated tobacco risk. The burden attributable to past smoking was estimated in people aged 40 and over, because the small number of lung cancer deaths observed in those aged 25–39 meant that PAFs produced for these age groups would have been unreliable.

2003 estimates

National exposure estimates for 2003 were calculated from the earlier iterations of the same surveys used for the 2011 estimates (applied to the 1998 NDSHS), and followed the same method.

Indigenous estimates

The same general methods were used to estimate exposure to tobacco use in the Indigenous population. However, there were some differences in the data sources used to estimate the proportion of the Indigenous population who are current and former smokers, as well as the proportion of non-smokers exposed to environmental tobacco in the home.

Due to the small Aboriginal and Torres Strait Islander sample in the NDSHS (about 460 respondents in 2010), estimates of tobacco exposure were not considered reliable when broken down by age, sex and smoking status. Instead, the National Aboriginal and Torres Strait Islander Social Surveys (NATSISS) (2002 and 2008) were used for 2003 and 2011 estimates, respectively. While these survey dates do not directly align with the 5-year lagged smoking prevalence used for national estimates, analysis of Indigenous smoking rates in consecutive ABS Indigenous health and social surveys (2001, 2002, 2004–05, 2008, 2011–12) showed no discernible trends up to the AATSIHS 2011–12. Therefore, the choice of the 2002 and 2008 NATSISS surveys is likely to have had little impact on the proportion of the population exposed.

Similar to national estimates, a smoking impact ratio was used as an indirect method to estimate the accumulated risk from tobacco smoking for cancers and respiratory diseases. Reporting of Indigenous status on the Australian Cancer Database is only considered sufficiently complete for New South Wales, Queensland, Western Australia and the Northern Territory (AIHW & Cancer Australia 2013). Thus, lung cancer mortality data for only those 4 states and territory were used to calculate an Indigenous smoking impact ratio.

Alcohol use

The attributable burden due to alcohol use was measured in people aged 15 and over. This includes the burden attributable to:

- single occasion risk (or binge drinking), resulting in injury
- average daily consumption, leading to mental health and chronic disease outcomes.

Population attributable fraction

In the GBD study, chronic liver disease due to alcohol and liver cancer due to alcohol were entirely attributed to alcohol use, and no relative risks were published to use in the comparative risk assessment approach. The ABDS diseases of chronic liver disease and liver cancer were not broken down to this level. The PAF for chronic liver disease was estimated from the proportion represented by chronic liver disease due to alcohol of all chronic liver disease burden, as estimated for Australia by GBD 2010 (Appendix Table E4). The same method was used to estimate the PAF for liver cancer.

Exposure estimates

The NDSHS 2010 was identified as the best Australian data source to measure alcohol exposure. However, self-reported alcohol consumption is known to be an underestimate of actual consumption (Rehm et al. 2010). ABS data provide an estimate of the amount of alcohol available for consumption from beer, wine, spirits, pre-mixed beverages, and cider in a given year, based on excise, import and sales figures. While these data are a better measure of the overall volume of alcohol consumed annually, they cannot be broken down by age and sex. So in ABDS 2011, self-reported daily consumption from the NDSHS, by age and sex, was inflated to match alcohol sales data in each reference year, based on the methods described by Rehm et al. 2010 and GBD 2013 Risk Factors Collaborators 2015.

From the NDSHS, the proportion of self-reported lifetime abstainers and ex-drinkers was assumed to be correct. Among current drinkers, the mean number of standard drinks self-reported per day was converted into litres of self-reported alcohol for that year. In 2010, this amounted to almost 114 million litres of alcohol (Table 7.1). By comparison, 182 million litres of alcohol was available for consumption in Australia in the financial year 2010–11 (ABS 2012a).

Table 7.1: Self-reported annual alcohol consumption compared with national alcohol sales figures

Year	Self-reported alcohol consumption (litres)	Alcohol sales (litres)	Alcohol assumed consumed (80% of sales) (litres)	Adjustment factor
2003	97,595,127	163,620,000	130,896,000	1.34
2011	113,987,831	184,907,000	147,925,600	1.30

Source: NDSHS 2004 & 2010; AIHW analysis of ABS data.

Following methods used in GBD 2010, 80% of the alcohol available nationally was assumed to have been consumed (that is, almost 148 million litres). The same approach was used to inflate the 2003 self-reported alcohol data. Self-reported alcohol consumption was inflated by more than 30% in both reference years to account for under-reporting. Only a proportion (80%) of alcohol sold in Australia was used because these figures include alcohol discarded

due to changes in stocks, alcohol consumed by overseas travellers, alcohol that has been stored or cellared, and alcohol that has been used to prepare food or discarded as waste.

The adjusted litres of alcohol consumed nationally were distributed among self-reported current drinkers using a 2-parameter gamma distribution, which has been found to be the best model to shift the distribution of survey data to fit sales data (Rehm et al. 2010). While this approach brings self-reported alcohol consumption in line with known alcohol sales, a limitation is that it assumes the degree of under-reporting of alcohol consumption is uniform across all age and sex groups.

2003 estimates

Exposure estimates for 2003 were calculated from the 2004 NDSHS and 2003 alcohol sales data and followed the same method as for 2011.

Indigenous estimates

Due to the small Aboriginal and Torres Strait Islander sample in the NDSHS (about 460 respondents in 2010), estimates were not considered reliable when broken down by age, sex and amount of alcohol consumed. Instead, the NATSIHS 2004–05 and the AATSIHS 2012–13 were used. As alcohol excise, sales and import figures published by the ABS represent a single national figure, it is not possible to calculate Indigenous-specific factors to correct for under-reporting. Therefore, national factors were applied to Indigenous estimates from the NATSIHS and AATSIHS.

Physical inactivity

The burden attributable to physical inactivity was measured as the metabolic equivalent of tasks (METs) done by people aged 25 and over. This is a categorical risk factor, and the categories are sedentary (less than 600 METs), low levels of activity (600–3,999 METs), moderate levels of activity (4,000–7,999 METs), and high levels of activity (8,000 METs and over).

Population attributable fraction

Exposure estimates

Exposure was estimated from the AHS 2011–12 as self-reported METs in the previous week. Included in the estimates were exercise due to stretching, gardening and walking for transport, as well as recreational exercise.

2003 estimates

For 2003 national estimates of physical inactivity, data were extracted from the NHS 2004–05, and adjusted by the proportion of additional METs from gardening and stretching, which were not included in the survey.

Indigenous estimates

Exposure estimates of physical inactivity for the Indigenous population was estimated from the AATSIHS 2012–13 (for 2011 estimates) and the NATSIHS 2004–05 (for 2003 estimates).

It was not possible to adjust these estimates to include stretching and gardening, as this information was not available from the relevant surveys used.

Drug use

The impact of illicit drug use captures both the infectious disease outcomes (HIV/AIDS, acute hepatitis B, acute hepatitis C) and their associated chronic conditions (liver cancer and liver disease) arising from injecting drug use. It also captures other outcomes, such as injury and mental and substance use disorders, caused by drug dependency in people of all ages.

Population attributable fraction

Two methods were used to estimate PAFs for injecting drug use and drug dependency.

Injecting drug use

PAFs were calculated directly for HIV/AIDS, hepatitis B, hepatitis C, chronic liver disease and liver cancer from the NNDSS published in the annual surveillance reports by the Kirby Institute (Kirby Institute 2012 and 2013).

For HIV/AIDS, the direct PAF was from the proportion of diagnosed AIDS cases in 2011 with an exposure category of injecting drug use with or without homosexual contact. For acute hepatitis B and C the direct PAF was from the proportion of newly acquired hepatitis B or C infections in 2011 with an exposure of injecting drug use with or without homosexual contact.

For chronic liver disease and liver cancer the direct PAF was from the proportion of newly acquired hepatitis B and C infections with an exposure of injecting drug use from the earliest available data. For hepatitis C, the earliest available exposure data were (on average) between 2000 and 2001. For hepatitis B the earliest available exposure data were from 1997. These were applied to the proportion of chronic liver disease or liver cancer due to hepatitis B and C, estimated from causes in GBD 2010 as described in Table E4.

Drug dependency

All burden due to amphetamine, cannabis, cocaine and opioid dependence was attributable to drug use; therefore a PAF of 1 was attributed to the prevalence estimates. For more information on estimating burden due to these conditions see 'Drug use disorders' in Chapter 5.

2003 estimates

Similar methods for estimating exposure and calculating the PAF in 2011 were used to produce 2003 estimates. Injecting drug use PAFs were calculated from the annual surveillance reports by the Kirby Institute for the year 2003 (Kirby Institute 2004). The PAF for chronic liver disease and liver cancer had a shorter look-back period, and the same years of data were used as for the 2011 estimates.

Indigenous estimates

For Indigenous risk factor estimates for drug use for 2011 and 2003, the same data sources and methods were used as for national estimates. The quality of Indigenous data in the

NNDSS varies by disease and state/territory, and is described in the annual surveillance reports published by the Kirby Institute.

Intimate partner violence

The burden attributable to intimate partner violence was estimated in females aged 15 and over. The attributable burden reflects short-term health outcomes, such as injuries, as well as longer-term health outcomes, such as mental and substance use disorders, as a result of exposure to intimate partner violence.

The AIHW, in collaboration with Australia's National Research Organisation for Women's Safety, is doing further work to assess the methodological inputs to calculate attributable burden due to exposure to intimate partner violence in Australian women. This work will result in revised estimates, which are anticipated to be published in late 2016.

Population attributable fraction

Attributable burden due to intimate partner violence used a direct PAF for fatal burden due to homicide and violence, while the remaining burden was estimated using the comparative risk assessment method.

Fatal burden due to homicide and violence

For the fatal burden due to homicide and violence, direct evidence of attributable burden was available from the National Homicide Monitoring Program. In 2010–2012, 46% of female homicides were classified as perpetrated by an intimate partner (Bryant & Cussen 2015).

Remaining burden

Exposure to intimate partner violence was estimated from the ABS Personal Safety Survey 2012. Females exposed were estimated as those aged 18 and over who answered yes to experiencing physical or sexual violence from a current or previous partner from the age of 15. A partner is defined as a person the respondent currently lives with, or lived with ever, in a married or de facto relationship. The same rate in females aged 18 was applied to those aged 15 and over.

2003 estimates

The ABS found no change in exposure to intimate partner violence between the 2005 and 2012 Personal Safety Surveys, so rates from the 2012 survey were used to estimate exposure to intimate partner violence in 2003.

For 2003, 52% of female homicides were classified as perpetrated by an intimate partner (Mouzos 2005).

Indigenous estimates

For fatal burden due to homicide and violence, direct evidence for Indigenous women was used from the National Homicide Monitoring Program. In 2010–2012, 65% of Indigenous female homicides were classified as perpetrated by an intimate partner (Cussen & Bryant 2015); while for 2003, this was assumed to be 59% based on estimates from 2006–07 (Dearden & Jones 2010).

For the remaining burden, the ABS Personal Safety Survey 2012 did not include an Indigenous identifier, so indirect methods were used to estimate Indigenous exposure to intimate partner violence. A rate ratio of 2.5 was applied to national exposure estimates (AIHW 2015a). This rate ratio is based on age-standardised rates for 12-month prevalence of physical or threatened violence victimisation reported by females from the 2006 General Social Survey (for national estimates) and the 2008 NATSISS (for Indigenous estimates). The same rate ratio was applied to the national exposure estimates to derive Indigenous exposure for both 2003 and 2011.

Unsafe sex

The burden of unsafe sex was estimated in people of all ages using direct evidence.

Population attributable fraction

The entire burden of syphilis, chlamydia, gonorrhoea, cervical cancer and other sexually transmitted infections was attributed to unsafe sex, so a PAF of 1 was used.

PAFs were calculated directly for HIV/AIDS, hepatitis B, hepatitis C, chronic liver disease and liver cancer from the NNDSS published in the annual surveillance reports by the Kirby Institute (Kirby Institute 2012 and 2013).

For HIV/AIDS the direct PAF was from the proportion of diagnosed AIDS cases in 2011 with an exposure category of unsafe sex (including homosexual contact only, homosexual contact and injecting drug use, or heterosexual contact). For acute hepatitis B and C, the direct PAF was from the proportion of newly acquired hepatitis B or C in 2011 with exposure to unsafe sex.

For chronic liver disease and liver cancer, the direct PAF was from the proportion of newly acquired hepatitis B and C infections with an exposure to unsafe sex from the earliest available data. For hepatitis C, the earliest available exposure data is for 2000–2001. For hepatitis B, the earliest data available exposure is 1997. These were applied to the proportion of chronic liver disease or liver cancer due to hepatitis B and C, estimated from causes in GBD 2010, as described in Appendix Table E4.

2003 estimates

The same methods used to estimate exposure and calculate the PAF in 2011 were used to produce 2003 estimates. PAFs for unsafe sex were calculated from the annual surveillance reports by the Kirby Institute for 2003 (Kirby Institute 2004).

Indigenous estimates

For Indigenous estimates for unsafe sex for 2011 and 2003, the same methods and exposure data sources were used as for national estimates. The quality of Indigenous data in the NNDSS varies by disease and state/territory, and is described in the annual surveillance reports published by the Kirby Institute.

Childhood sexual abuse

The burden attributable to childhood sexual abuse was estimated in people aged 15 and over due to limitations in data available to estimate prevalence of exposure in childhood. The attributable burden reflects the health outcomes experienced later in life as a result of sexual abuse during childhood, such as mental and substance use disorders.

Population attributable fraction

Exposure estimates

Exposure was estimated from the ABS Personal Safety Survey 2012 (ABS 2013d), from people aged 18 and over who answered yes to being exposed to childhood sexual abuse before the age of 15. Sexual abuse was defined as any act by an adult involving a child (before the age of 15) in sexual activity beyond their understanding or contrary to currently accepted community standards.

Indigenous estimates

As the Personal Safety Survey 2012 did not include an Indigenous identifier, indirect methods were used to estimate Indigenous exposure to childhood sexual abuse.

Data on victims of sexual assault from ABS recorded crime data were used to estimate the relative difference in prevalence of child sexual abuse between the Indigenous and the total Australian populations, based on the Overcoming Indigenous Disadvantage 2014 tables (Steering Committee for the Review of Government Service Provision 2014). The ratio was based on 2013 data from 4 jurisdictions (New South Wales, Queensland, South Australia and the Northern Territory), and assumed that these jurisdictions were representative of the national situation. This method also assumed that the recorded crime statistics covered the same proportion of child sexual assault victims in the Indigenous and non-Indigenous Australian populations.

High body mass

The burden attributable to high body mass was estimated in people aged 25 and over. This is the population for which relative risks are available, and for which burden was estimated in recent global burden of disease studies. The AIHW is currently doing further work funded by the Australian Government Department of Health to extend this analysis to estimate the burden attributable to high body mass in people younger than 25. A report on the findings of this work is expected to be published in the first half of 2017.

Population attributable fraction

Exposure estimates

Exposure was estimated as the distribution of body mass index in participants from the AHS 2011–12. Exposure was adjusted by the range of the TMRED, as described in Chapter 6.

2003 estimates

The exposure to body mass index over time was calculated by comparing the trend in mean exposure from NHS 1995, NHS 2007 and AHS 2011–12, by sex. Record level data from the AHS 2011–12 were adjusted by the percentage change from 2011 to 2003, calculated using the trend in these data. The adjusted unit record data were used to estimate the distribution of exposure to body mass index in 2003.

Indigenous estimates

Exposure for 2011 was estimated as the distribution of body mass index in Indigenous Australians from the AATSIHS 2012–13. The 2003 estimates were calculated using the same method as described for national estimates by comparing the trend in mean body mass index in 2011 to that estimated for Indigenous Australians in the 2003 Indigenous Australian Burden of disease study (Vos et al. 2007).

The 2003 estimates were based on data from the 2001 NATSIHS, which used measured height and weight information to estimate mean body mass index for Indigenous Australians living in remote areas. The relative difference between self-reported and measured body mass index were assumed to be the same in Indigenous Australians living in remote and non-remote areas, and was applied to the mean self-reported body mass index for Indigenous Australians living in non-remote areas (Vos et al. 2007).

High blood pressure

The burden attributable to high blood pressure was estimated in people aged 25 and over.

Population attributable fraction

Exposure estimates

Exposure was estimated as the distribution of systolic blood pressure in participants from the AHS 2011–12. Exposure was adjusted by the range of the TMRED, as described in Chapter 6.

2003 estimates

The exposure to blood pressure over time was calculated by comparing the mean exposure from Australian Diabetes, Obesity and Lifestyle Study (AusDiab) 1999–2000 and the mean exposure from AHS 2011–12, by age and sex. The percentage change (or absolute change) from 2011 to 2003 was calculated using these data. Unit record level data from the AHS 2011–12 was adjusted by the percentage change to estimate the distribution of exposure to blood pressure in 2003.

Indigenous estimates

Exposure for 2011 was estimated as the distribution of blood pressure in Indigenous Australians from the AATSIHS 2012–13. The 2003 estimates were calculated using the same method as described for national estimates by comparing the trend in mean exposure in 2011 to exposure estimated in the 2003 Indigenous Australian burden of disease study (Vos et al. 2007).

The 2003 estimates were based on data published in the 2003 Australian burden of disease study (Vos et al. 2007). These data are from relatively small studies covering 2 regions (the DRUID study by Cunningham et al. 2006; Wang & Hoy 2003 in Vos et al. 2007), in which it was assumed that the measured systolic blood pressure mean and standard deviations were representative of Indigenous Australians living in non-remote and remote areas.

High blood plasma glucose

The burden attributable to high blood plasma glucose was estimated in people aged 25 and over. It was not possible to calculate burden attributable to this risk factor in 2003 because there were no comparable biological data on exposure to the risk factor before 2011 to enable estimation of trends.

Population attributable fraction

Exposure estimates

Exposure was estimated as the distribution of fasting plasma glucose levels in participants from the AHS 2011–12. Exposure was adjusted by the range of the TMRED, as described in Chapter 6.

Indigenous estimates

Exposure was estimated as the distribution of fasting plasma glucose levels in Indigenous Australians from the AATSIHS 2012–13.

High cholesterol

The burden attributable to high cholesterol was estimated in people aged 25 and over.

Population attributable fraction

Exposure estimates

Exposure was estimated as the distribution of blood total cholesterol in participants from the AHS 2011–12. Exposure was adjusted by the range of the TMRED, as described in Chapter 6.

2003 estimates

The exposure to high cholesterol over time was calculated by comparing the mean exposure from the AusDiab 1999–2000 and the mean exposure from AHS 2011–12, by age and sex (Begg et al. 2007). Record level data from the AHS 2011–12 were adjusted by the percentage change in the mean from 2011 to 2003. The adjusted unit record data were used to estimate the distribution of exposure to high cholesterol in 2003.

Indigenous estimates

Exposure for 2011 was estimated as the distribution of total blood cholesterol in Indigenous Australians from the AATSIHS 2012–13. The 2003 estimates were calculated using the same method as described for national estimates by comparing the trend in mean exposure in 2011

to exposure estimated in the 2003 Indigenous Australian burden of disease study (Vos et al. 2007).

The 2003 estimates were based on data from relatively small studies covering 2 regions (the DRUID study by Cunningham et al. 2006; Wang & Hoy 2003 in Vos et al. 2007), in which it was assumed that the measured total cholesterol mean and standard deviations were representative of Indigenous Australians living in non-remote and remote areas (Vos et al. 2007).

Iron deficiency

The burden attributable to iron deficiency was estimated in females aged 15–45 for 2011. It was not possible to estimate exposure to this risk factor in 2003, as comparable biological exposure data for previous years were not available to estimate trends.

Population attributable fraction

Exposure estimates

Exposure was estimated as the distribution of blood haemoglobin levels in participants from the AHS 2011–12. Exposure was adjusted by the range of the TMRED, as described in Chapter 6.

Indigenous estimates

Exposure was estimated as the distribution of blood haemoglobin levels in Indigenous Australians from the AATSIHS 2012–13.

Low bone mineral density

The burden attributable to low bone mineral density was measured in people aged 40 and over.

Population attributable fraction

Exposure estimates

Self-reported prevalence of osteoporosis underestimates the true community prevalence of the condition, as many individuals with low bone mineral density display no overt symptoms, so will remain undiagnosed.

Exposure data were sourced from the 2001–06 wave of the Geelong Osteoporosis Study (Henry et al. 2010). Mean bone mineral density at the femoral neck, by age and sex, was used to model exposure distributions, assuming a normal distribution and following methods described by Sànchez-Riera et al. (2014).

2003 estimates

The same methods used to estimate exposure and calculate the PAFs in 2011 were used to produce 2003 estimates. Data from the Geelong Osteoporosis Study were applied to the 2003 population.

Indigenous estimates

Standardised bone mineral density measurements at the femoral neck were not available for the Indigenous population. National rates of low bone mineral density, by age and sex, were applied to the Indigenous population to calculate Indigenous exposure estimates. This approach was supported by the same rates of self-reported osteoporosis for the Indigenous and national populations reported in the AATSIHS 2012–13 and AHS 2011–12.

Occupational exposures and hazards

Occupational exposures and hazards capture the impact of exposures to 13 carcinogens (asbestos, arsenic, benzene, beryllium, cadmium, chromium, diesel engine exhaust, second-hand smoke, formaldehyde, nickel, polycyclic aromatic hydrocarbons, silica and sulphuric acid), asthmagens, noise, ergonomic stressors, injury, and particulate matter, gases and fumes in the workplace. The burden is ascribed from past as well as current exposure. The burden attributable to these exposures was calculated for different age groups as described in each section.

Population attributable fraction

The PAF for injury were estimated directly from data collected by Safe Work Australia. For all other disease outcomes, the PAFs were estimated from exposure to working in various industries or occupations.

Exposure estimates

To estimate the number of people working in Australia, the economically active population, by age and sex, was estimated from the Labour Force Survey, 30 June 2011 (ABS 2011a). This was then broken down by industry or occupation.

Industry

Exposure to working in various industries was linked to various cancers, hearing loss and chronic obstructive pulmonary disease (Table E2). This is because working in these industries is known to expose a proportion of the workforce to carcinogens, noise, particulate matter, gases and fumes.

The working population was distributed across 9 broad industry types (agriculture, hunting, forestry and fishing; mining and quarrying; wholesale, retail trade, restaurants and hotels; manufacturing; electricity, gas and water; transport, storage and communication; construction; finance, insurance, real estate and business services; community, social and personal services) based on the 2011 Census of Population and Housing.

A severity distribution was then applied as the proportion of people working in these industries exposed to high and low levels of noise, and particulate matter, gases and fumes. This was sourced from GBD 2010. The PAFs were calculated for people aged 15–74.

To account for the latency period between exposure and the symptoms of cancer, an 'occupational turnover rate' was applied to the number of people working in these industries. The occupational turnover rate is an adjustment factor that accounts for annual worker turnover, mortality rates, and past trends by industry, to estimate past exposure to carcinogens in each industry. These factors are based on trends observed in the United Kingdom.

Data from the Carcinogen Exposure research project produces estimates of the proportion of workers in each industry who will be exposed to specific carcinogens. These proportions are based on data from the European Union and Canada. They were then applied for each industry as described previously. The PAF for carcinogens were calculated for people aged over 15.

Occupation

Exposure to various occupations was linked to asthma and low back pain (Table E2). This is because working in these occupations is known to expose a proportion of the workforce to asthmagens and ergonomic stressors.

The working population was apportioned by broad occupational groups based on the 2011 Census of Population and Housing. These groupings were designed to best match the available relative risks specific to each linked disease, based on the correspondence between the Australian and New Zealand Standard Classification of Occupations (ANZCO) First Edition Revision 1 and the International Standard Classification of Occupations 1988 (ISCO-88) (ABS 2009), and vary between the linked diseases.

For asthma, occupations were broadly grouped as: administrative, clerical and managerial work; agriculture, forestry and commercial fishing; manufacturing and related work; mining and quarrying; sales work; service work; transport and communications work; and technical work.

For back pain, occupations were broadly grouped as: administrative and managerial work; clerical and related work; agricultural, animal husbandry, forestry, fishers and hunters; production and related work, transport equipment operators and labourers; professional, technical and related work; sales work; and service work.

Exposure from working in these occupations was used to estimate the PAF in people aged 15–64, and no severity distribution was applied.

Direct evidence

All of pneumoconiosis was attributable to occupational exposure, as informed by expert advice (T Driscoll, 2015. pers. comm., 24 June).

For injuries, direct evidence was sourced from:

- *Work-related traumatic injury fatalities, Australia, 2010–11* (Safe Work Australia 2012) (for the fatal burden)
- *Compendium of workers' compensation statistics Australia, 2010–11* (Safe Work Australia 2013) (for the non-fatal burden)

These publications report the number of deaths occurring at work and the number of workers' compensation claims annually. Counts of deaths and injuries, with some disaggregation by age, sex and nature/external cause of injury, were used to directly calculate PAFs. Due to the relatively small number of female workplace deaths, a 3-year average was used for female mortality PAF estimates.

These data are limited by the fact that compensation claims will only capture injuries that require more than 1 week away from work and are fairly severe. They will also not include people who are self-employed. To correct for the undercount of self-employed individuals, rates of claims reported among administrative and managerial workers were applied to the self-employed working population. These PAFs were estimated for people aged 15 and over.

2003 estimates

The same methods used to estimate exposure and calculate the PAFs in 2011 were used to produce 2003 estimates. The working population was estimated from the Labour Force Survey as at 30 June 2003 (ABS 2003), and broken down by occupation and industry using the 2006 Census of Population and Housing.

Indigenous estimates

The attributable burden in Aboriginal and Torres Strait Islander population was calculated in the same way as for the national population with the following changes.

The estimates of the number of Indigenous Australians working were sourced from the labour force survey 2011 (ABS 2011b). The national estimates of the working population include long-term unemployed people, as they make up only a small proportion of the national population (1.3% in 2003 and 1.0% in 2011) (ABS 2011a). As long-term unemployed people represent a much higher proportion of the Indigenous population (5.7% in 2003 and 6.0% in 2011) (AIHW analysis of the NATSISS 2002 and AATSIHS 2012–13), the estimate of economically active Indigenous population was adjusted down by the difference between these rates in each year.

Estimates of the number of Indigenous Australians working in 2003 were sourced from the Labour Force Survey 2006 (ABS 2007). These estimates were broken down by occupation and industry using estimates from the 2001 Census of Population and Housing (ABS 2001).

National PAFs were used to estimate attributable burden due to carcinogens for Indigenous Australians, because the occupational turnover rates used in this calculation are not appropriate for the Indigenous population.

The Safe Work Australia data sets do not include an Indigenous identifier, so the direct evidence sourced from these publications was not available for the Indigenous population. Instead, an Indigenous to non-Indigenous rate ratio was calculated for all injury hospitalisations with an ICD-10-AM activity code of U73 ('While working for income'), by sex. This ratio was applied to the national exposure rates to derive Indigenous exposure estimates for injuries.

Sun exposure

The burden attributable to sun exposure was estimated in people of all ages. The burden from this risk factor was not estimated for 2003. The attributable burden was not estimated for the Indigenous population as it was not possible to account for the impact of differences in skin melanin levels. The direct PAFs used were a proportion of the current burden that is due to past and current sun exposure in the population.

Population attributable fractions

The PAFs for sun exposure were calculated by collaborating experts Robyn Lucas and Fan Xiang from the National Centre for Epidemiology and Population Health at the Australian National University.

The melanoma PAF appropriate for Australia was advised to be the upper estimate of 0.9 from the global study on the burden of disease from solar ultraviolet radiation (Lucas et al. 2006). The squamous cell carcinoma and basal cell carcinoma PAFs were

calculated using the comparative risk assessment approach based on levels of ultraviolet radiation exposure in Australia (F Xiang 2015, pers. comm., 11 November; Xiang et al 2014).

Air pollution

The fatal burden attributable to air pollution was measured by concentration of particulate matter (PM) of 2.5 micrograms per cubic metre (PM2.5) in Australia in people of all ages. It was not possible to estimate exposure to this risk factor in 2003 because comparable exposure data were not available.

Population attributable fraction

Exposure estimates

PM2.5 is particles suspended in the air with a diameter of 0–2.5 microns. Population exposure to air pollution was estimated from ground-based monitoring stations in 40 locations around Australia measuring PM2.5. Each station provides data on exposure relative to the proportion of the Australian population in the remoteness area and state/territory covered, and the number of stations in the area (Table 7.2). The number of people covered was calculated using population data by state/territory and remoteness area.

Each state and territory provided data upon request (A Grieco, Department of Environment Regulation Western Australia 2015, pers. comm., 29 June; J Innis, Environment Protection Authority Tasmania 2015, pers. comm., 6 August; J Choi Environment Protection Authority Victoria 2015, pers. comm., 13 July; S Gerrity, Environment Protection Authority South Australia 2015, pers. comm., 13 July; J Zhang, Environment Protection and Water Regulation Australian Capital Territory 2015, pers. comm., 16 July) or from their website (Queensland Government 2015, New South Wales Office of Environment and Heritage 2015, Northern Territory Environment Protection Authority 2015).

The majority of monitoring stations were located in major cities, as they are concentrated around population centres. Some stations were located in *Inner regional* and *Outer regional* areas. No stations were located in *Remote* or *Very remote* areas. *Remote* and *Very remote* areas were assumed to have the same exposure as the regional areas in each state where data were available.

Monitoring stations only provide an indication of the level of pollution that people in the region immediately surrounding the station are exposed to. As PM2.5 monitoring stations are sparsely located around Australia, it is likely that the pollution levels recorded differ from the actual levels experienced by the wider population. There is also likely to be significant variation between sites in the amount of time that people generally spend outside being exposed to air pollution. Further, due to differences in planning history and population density, the effects of industrial pollution might be greater in some locations compared with others.

The relative altitude above sea level and the impact of local weather events can also affect the monitoring of air pollution. Other factors that lead to regional variations are possible variations in health practices and the composition of the particulate mix.

Other methods of measuring PM2.5 through satellite imaging rely on a large amount of spatial modelling, and were not possible for this study. They have the same issue of measuring ambient air pollution levels and not actual exposure to air pollution; however,

they would have the advantage that the estimates would be based on measurements from larger areas of Australia.

States and territories report the readings for PM concentrations in various ways (hourly and daily measurements). Where possible, daily data were used; where those were unavailable, hourly data were converted to daily data. Daily average was calculated as the daily average of hourly observations (days with less than 75% of hours were excluded).

Although data from regional Tasmania were obtained using a Nephelometer method, which is not a direct method of measuring PM2.5, these data were still used as they represented only a very small proportion of the Australian population.

Table 7.2: Name and remoteness category of stations that collected data on PM2.5 in 2011

State/territory	Station	Remoteness category
NSW	Beresfield, Chullora, Earlwood, Liverpool, Richmond, Wallsend, Wollongong	Major cities
Vic	Alphington, Brooklyn, Footscray	Major cities
Qld	Arundel, Springwood,	Major cities
	South Gladstone	Inner regional
WA	Caversham, Duncraig, Quinn Rocks, South lakes	Major cities
	Bunbury, Busselton	Inner regional
SA	Netley	Major cities
Tas	Clearys Gates, South Launceston	Inner regional
	Bryn Estyn, Dearby, Emu River, Exeter, Fingal, Gretna, Geeveston, Huonville, Judbury, Lilydale, Scottsdale, Sheffield, Smithton, St Helens, West Ulverstone	Outer regional
ACT	Monash	Major cities
NT	Palmerston	Outer regional

Indigenous estimates

To estimate exposure to air pollution in Aboriginal and Torres Strait Islander people, each monitoring station provides data on exposure relative to the proportion of the Australian Indigenous population living in the state/territory and remoteness area covered, as well as data on the number of stations in the area.

The number of Indigenous Australians exposed to air pollution was calculated by applying these proportions to the 2011 Aboriginal and Torres Strait Islander estimated resident population data by state/territory and remoteness area.

Unimproved sanitation

The burden from unimproved sanitation was not estimated for the non-Indigenous population due to lack of available exposure data, and was assumed to be close to 0.

Indigenous estimates

Exposure was estimated from the AATSIHS 2012–13. The estimate was based the number of Indigenous Australians living in the households that self-reported not having working sewerage facilities.

Dietary risk factors

A total of 13 dietary risk factors were included in the study, and the burden attributable to these was measured in people aged 25 and over.

It should be noted that the methods, including the TMREDs, used in the ABDS 2011 to calculate attributable burden due to dietary risk factors do not align with current Australian dietary guidelines as they are used for the purpose of calculating disease burden. For information of recommended food choices please see the Australian Dietary Guidelines (NHMRC 2015).

Population attributable fraction

Exposure estimates

Some of the dietary risk factors were for the consumption of whole foods such as a diet low in fruit, a diet low in vegetables, a diet high in sweetened beverages, a diet low in milk, a diet high in red meat, a diet low in nuts and seeds, a diet low in whole grains, and a diet high in processed meat.

The other risk factors were for the consumption of micronutrients, such as a diet low in omega-3, a diet high in saturated fat, a diet low in fibre, a diet high in sodium, and a diet low in calcium.

Micronutrients

Exposure was estimated as the distribution of dietary intake of micronutrients (excluding supplements), as reported by survey respondents on day 1 of the 24-hour dietary recall questionnaire from the AHS 2011–12.

These data had not been adjusted to the usual intake by further analysis to include food consumed on both day 1 and day 2 of the survey because these data were not available by age and sex. These data have less variability than the day-1 data, but the same mean.

The risk–outcome pair of calcium and colorectal cancer due to a diet high in calcium was not included in this study because it was a protective relative risk, and it was not possible to establish an appropriate method to measure the attributable burden. All other risk–outcome pairs were adopted from GBD 2010.

Whole foods

The whole foods included for each risk factor are in Table 7.3. The average proportion of these foods types from within each food classified in the AHS 2011–12 was determined from the AHS Australian Dietary Guidelines database (FSANZ 2016; P Atyeo, ABS, 2016, pers. comm., 6 January). Exposure was estimated as the distribution of dietary intake of each of these classified foods on day 1 of the dietary recall from the AHS 2011–12.

These day-1 data are likely to have more variability than the modelled usual dietary intakes, which were not available from ABS in time for this project. ABS has since published key findings on the consumption of food groups from the Australian Dietary Guidelines, which should be taken into account (ABS 2016).

There is significant under-reporting of dietary intake in the AHS 2011–12 (as with all representative dietary surveys) (ABS 2014c). There is a tendency for survey respondents to either change their behaviour or misrepresent their consumption (whether consciously or

subconsciously) to report a lower energy or food intake. This under-reporting is unlikely to affect all foods and nutrients equally (unhealthy, discretionary foods are most likely to be under-reported, and healthy foods, such as fruit and vegetables, are likely to be over-reported). The AIHW was unable to adjust for under-reporting in the ABDS 2011.

Table 7.3: Whole foods included in dietary risk factor analysis

Dietary risk factor	Included foods
Diet low in fruit	Intact fruits (whole or cut) and dried fruit (excluding juice)
Diet low in vegetables	Green and brassica, green leafy, orange, starchy, other vegetables and legumes (excluding juice)
Diet high in sweetened beverages	Sugar sweetened soft drink, cordial base drinks, milk, fruit drink and sports and energy drinks
Diet low in milk	Milk (with and without milk powder)
Diet high in red meat	Unprocessed red meat
Diet low in nuts and seeds	Nuts, seeds, peanut or almond butter or tahini or other nut or seed pastes
Diet low in whole grains	All wholegrain or higher fibre breads, cereals, rice, pasta, crumpets, English muffins, crispbreads, relevant fortified cereals with 1 gram of fibre per 10 grams of carbohydrate[(a)]
Diet high in processed meat	Processed meat and poultry

(a) The FSANZ Australian Dietary Guidelines classification of grain foods into wholegrain (high fibre) was based on description of the food (for example, described as wholemeal) and the amount of fibre. Bread and grains were 5 grams or more per 100 grams, while other products (crackers, crispbreads, muffins and crumpets) were 10 grams or more per 100 grams. See Appendix 1 at: <www.foodstandards.gov.au/science/monitoringnutrients/australianhealthsurveyandaustraliandietaryguidelines/appendices/Pages/default.aspx>.

Relative risks

Where relative risks for some dietary risk factors were not published in GBD 2010 they were sourced from GBD 2013 (GBD 2013 Risk Factors Collaborators 2015), but only for those diseases linked in the GBD 2010. No additional linked diseases were included from GDB 2013 as they were not available in time for analysis due to timing of the release of the ABDS 2011 reports.

2003 estimates

The ABS provided estimates for 1995 that sought to put the 1995 consumption of fruit and vegetables on the same basis as in 2011–12. However, the adjustment for vegetables in particular was based on incomplete information, so the results for vegetables should be treated with caution (P Atyeo 2016, pers. comm., 25 January).

The exposure to these risk factors over time was calculated by comparing the mean exposure from the NHS 1995 with that from the AHS 2011–12, by age. Unit record level data from the AHS 2011–12 was adjusted by the percentage change (or absolute change) from 2011 to 2003 in these data sources, to estimate the distribution of fruit and vegetable intake in 2003.

Indigenous estimates

Exposure estimates for each of the 13 dietary risk factors for the Indigenous population was estimated from the AATSIHS 2012–13 using the same methods as used for national estimates. For example, day 1 dietary recall data were used for the micronutrients. For whole

foods, the AHS whole food database was used to estimate the average proportion of the whole foods from within each food classified in the AATSIHS 2012–13.

For 2003 estimates of diet low in fruit and diet low in vegetables in Indigenous Australians, the changes in mean inadequate consumption of fruit and vegetables was sourced from the difference between the NATSIHS 2004–05 and AATSIHS 2012–13 (ABS 2014b).

Section III: Accounting for quality and accuracy

This section summarises the approach to quality and accuracy of each of the components in the ABDS 2011 (that is, fatal, non-fatal, and attributable burden), and provides context for the interpretation of the results.

8 ABDS quality framework

In an ideal world, burden of disease estimates would be based on a fully enumerated set of data of all health loss and risk exposure experienced by every person in the population of interest. But in reality, burden of disease estimates are based on models of disease and risk factor epidemiology applied to existing sources of data of varying completeness and quality.

In some instances, these 2 components are perfectly matched, but in many cases, there can be differences between the data required by the model and the data available to be analysed, leading to various levels of uncertainty around the estimate.

Ensuring quality of inputs to the ABDS 2011

Several steps were taken to ensure the accuracy and relevance of the estimates in the ABDS:

- All standard inputs (such as the reference life table, disability weights and relative risks) were reviewed and assessed as appropriate by the Australian Burden of Disease Expert Advisory Group and Indigenous Reference Group for relevance and applicability in the Australian and Indigenous contexts.
- All data used in the ABDS were required to meet strict inclusion criteria via protocols endorsed by the Australian Burden of Disease Expert Advisory Group and Indigenous Reference Group.
- All models and inputs used in YLL, YLD and risk factor estimates were reviewed by clinical and other relevant experts to ensure their appropriateness for Australian and Indigenous populations.
- Where there were competing methods or data sources, sensitivity analyses were undertaken to compare the impact of the different choices. Final decisions were made in consultation with the Expert Advisory Group and Indigenous Reference Group.

Measuring the quality of outputs from ABDS 2011

Two commonly used measures of reliability considered by the study to describe the overall quality of estimates were:

- **uncertainty analysis**—this provides a measure of the 'precision' of the estimate, including how much the true value might differ from the estimate (for example, by using 95% confidence intervals). These are estimated based on the underlying data using

well-established statistical techniques that measure random variation in the data, but do not measure variation in the model and assumptions to which the data are applied

- **scenario testing**—this provides a measure of how much the estimate might vary if certain parameters in the model underpinning the estimate differed (for example, if the duration of a disease was longer or shorter) or if the data applied to the model varied, but it does not measure differences that might be due to random variation in the underlying data.

Uncertainty analysis

Using case studies of mortality (national and Indigenous), cancer and chronic kidney disease, the ABDS project team considered 2 approaches to estimate uncertainty: direct calculation and simulation.

Both the direct-calculation approach and the simulation approach required some information about the uncertainty around the input data. The information might take various forms, ranging from an explicitly estimated statistical distribution to a general indication of, for example, the variance (breadth of scatter) around the input data. If only the latter were available, then some plausible statistical distribution (consistent with that variance) needed to be assumed or imposed.

Obtaining information about uncertainty for the inputs (even for a single disease or injury) might require a complex investigation or brave assumptions, particularly for input data drawn from registries or administrative data. Obtaining such information across the whole spectrum of diseases and injuries is a major research problem requiring subject matter expertise, and was outside the scope of this project.

Direct-calculation approach

In concept, this approach entails 4 steps:

1. Ascertain (or assume) the statistical distributions around the inputs.
2. Describe the YLL or YLD estimation process as a mathematical transformation of those inputs.
3. Apply analytical methods (textbook theory) to work out the statistical distribution of the output (YLL or YLD) that results from the transformation.
4. Compute the resultant uncertainty intervals around the output.

Even if the information for the first step were obtainable, the third step is tractable only in the case of some relatively straightforward transformations and some well-understood input distributions. That is why GBD and other investigators that have provided uncertainty intervals have generally relied upon simulation.

Simulation approach

In concept, this approach requires 5 steps, although the actual sequence of computations is generally different, but has been laid out this way for clarity:

1. Ascertain (or assume) the statistical distribution of each data input as outlined above.
2. Draw samples from the input distributions to generate a synthetic population of cases.

3. Put each hypothetical case through the first data transformation (in, for example, the YLD estimation process). This generates a first-transformed synthetic population of cases.
4. Repeat Step 3 for each subsequent data transformation, to eventually obtain a synthetic population of the estimate of interest (for example, YLD).
5. Read off the uncertainty interval from the result of Step 4.

Subject to accomplishing the large prior task of ascertaining statistical distributions for the inputs, this was considered a feasible approach. The methods are pretty well understood and software tools can be used for the computations (such as WinBUGS, a statistical software for Bayesian analysis using Markov chain Monte Carlo methods, developed by the BUGS Project, a team of United Kingdom researchers at the MRC Biostatistics Unit, Cambridge, and Imperial College School of Medicine, London). Nevertheless, implementing the approach across the whole of ABDS, and validating the findings, was estimated to involve a large volume of work that might have exceeded what was required to generate the actual estimates.

It became clear from the case-study assessments that the amount of work required to develop a reasonable and defensible method of uncertainty estimation that could be used across all parts of the ABDS was not within the resources of the project.

In addition, the assessments confirmed that the amount of error that could be encapsulated within an uncertainty interval will generally be only a (possibly minor) part of the total error or uncertainty attached to an estimate. Ignoring or concealing the error that might arise from epidemiological or methodological choices could mislead users into placing unjustified reliance on patterns and differences that they see in estimates of burden.

ABDS 2011 quality index

In light of the assessments of measuring uncertainty described previously, the ABDS 2011 Expert Advisory Group concluded that this was beyond the scope and resources of the project. However, they supported the need for clearly defined indicators to accompany each set of estimates (DALY, YLL, YLD and attributable burden) to provide users with guidance on the quality of the data underpinning the estimate, and to inform interpretation. Such indicators should inform users not only of the type of data used to derive the estimate, but also its coverage and any transformations required to produce inputs suitable to the YLL, YLD, DALY and risk factor attribution estimation process.

To help users understand the potential sources of uncertainty associated with the estimates from the ABDS 2011, a 2-dimensional index was derived based on:

- the relevance of the underlying epidemiological data
- the methods used to transform that data into a form required by this analysis.

These dimensions are explained in greater detail in the following section.

The index was designed to help users understand the reliability and limitations of the estimates, especially which patterns and differences were likely to be genuine, and which could be influenced by uncertainties in the data or methods that made them less reliable. Generally, the higher the index the more relevant and accurate the estimate was.

For it to be useful in assessing the impact of different data sources and transformation methods, the final index also took into account the contribution of the underlying data to the

overall estimate. For example, a particular data source might have contributed a large proportion of the overall YLD for a single disease, while another might have only contributed a small proportion.

This index was developed to assess national estimates from the ABDS. An expanded version of the index was developed to the assess Indigenous estimates, which incorporated Indigenous-specific criteria to address additional data quality issues relevant to Indigenous estimates.

Based on the processes required to produce the various estimates for burden of disease, and the experience of the ABDS project team in collating and analysing data for this purpose, the following key assumptions and core dimensions were developed to provide users with a succinct and coherent assessment of the quality of the estimates.

Key assumptions

To create the index, all standard inputs, methods and assumptions underpinning the estimates were referred to the Australian Burden of Disease Expert Advisory Group, Indigenous Reference Group and/or disease and risk factor experts for review. Assumptions on which this framework was based include:

- for YLL:
  - the reference life table (defined by GBD 2010) was appropriate for use in both the Australian and Indigenous contexts
  - for Indigenous estimates, the adjustment factors used to account for Indigenous under-identification in mortality data were appropriate to use
- for YLD:
  - the conceptual models mapping sequelae to health states that form the basis of estimates were appropriate as per expert review
  - the health states and disability weights (defined by GBD 2013) were appropriate to:
    - the conditions being estimated
    - the Australian and Indigenous populations.
  - the assigned average durations of health loss for sequelae that last for less than 1 year were an accurate reflection of the time spent in a particular health state. Duration has a direct impact on the point prevalence of each sequela (for these sequelae, prevalence = incidence x duration). Durations used in the ABDS were based on accepted clinical research or judgment, and were supplied or reviewed by the expert panels as part of the model.
  - for Indigenous estimates, the adjustment factors used to account for Indigenous under-identification in morbidity (hospitalisations) data were appropriate to use
- for risk factors:
  - the risk–outcome pairs, minimum exposure levels and effect sizes (used in the risk factor analysis) defined by GBD 2010 and other studies were appropriate for:
    - the particular risk factor
    - the Australian and Indigenous contexts.

Index dimensions

Dimension I: Relevance of the underlying epidemiological data

This dimension refers to the data used to generate the estimate, and includes concepts of data quality, currency and coverage, and suitability to the model being used. These were drawn together into a single score of 5 to 1, as outlined in Appendix Table F1. The higher the score the more relevant, current and complete the data.

Data source

All input data to the ABDS were required to meet strict quality criteria endorsed by the study's Expert Advisory Group and Indigenous Reference Group to ensure that only data of suitable quality were included in the study. However, within these criteria, there was still a wide variability of data reliability. This approach facilitated comparison between data sourced from:

- disease registers, administrative data, large national surveys, meta-analyses, modelled estimates and single epidemiological studies
- Australian compared with international sources.

Generally, higher scores were given to Australia-wide unit record or survey data, and lower scores to small surveys and epidemiological studies or international data of limited generalisability.

Data currency and coverage

Data currency refers to how close in time the data were to the reference year. The ABDS 2011 aimed to source data as close to the reference year as possible. While this was possible for most key data sources, it was not possible for all data sources. Data for conditions that are known to be stable over short periods of time were considered current if referring to within 2 years of the reference date (for example, cancer incidence data). Data for conditions that varied from year to year, such as some infectious diseases, were considered current if specific to the reference year.

Data coverage refers to the proportion of the population covered by the data. For example, national versus subnational, or all age groups versus particular age groups. Generally, the wider the coverage, the higher the score.

Data specificity

Data specificity refers to the suitability of the data to the condition and measure being analysed. Specificity depended very much on the relationship between the condition and the data source. For example:

- hospitals data for conditions with a high hospitalisation rate (such as appendicitis, amputation) scored higher than conditions with a medium or low hospitalisation rate (such as soft tissue injuries) when separations were used to estimate prevalence
- for survey data, clinically diagnosed conditions scored higher than self-reported conditions.

Dimension II: Methods of data transformation

This dimension refers to the methods used to transform the data to generate the estimate. It included processes used to fill data gaps, such as:

- projecting data from 1 year to the reference year to overcome issues of currency
- applying age and sex distributions or rate ratios from a secondary data source to overcome data gaps
- applying adjustment factors to overcome issues of data specificity
- smoothing or combining data to overcome variability in the source data due to sampling or small numbers.

As for Dimension I, these were also drawn together into a single score of 5 to 1, as outlined in Appendix Table F2.

Deriving the ABDS quality index

The ABDS quality index operated at the disease or risk factor level, and was applied to the YLL, YLD and attributable burden for the 2011 national and Indigenous estimates. The quality of DALY estimates is the weighted average of the YLL and YLD estimate.

The index was built from the lowest level of estimate using the 2 dimensions outlined previously, weighted for the contribution to the overall disease-level estimate, as follows:

- for YLL, it was applied at the disease level
- for YLD, it was applied at the sequelae level, weighted by the contribution to the overall YLD, and summed to produce an index at the disease level
- for risk factors, it was applied at the measure of exposure level (for example, second-hand smoking), then summed to produce an index at the risk factor level (for example, tobacco use).

The index for each dimension is derived and reported separately for YLD (Appendix Table F3) and risk factors (Appendix Table F4) to help interpret results.

Scoring

Each dimension was scored from 5 to 1. Although these are linear units, it should not be assumed that each score is proportionally equal. This was dealt with by scaling, as follows:

Each score was weighted by the proportion it contributed to the estimate in question. As the maximum score for a disease was 500 (that is, score of 5 contributing to 100% of the estimate) and the minimum 100 (a score of 1 contributing 100%), this was divided by 5 to give an overall score in the range 20–100.

This overall score was then divided into an **index** (A–E) for Dimension I/Dimension II, as follows:

A. 90 or more (highly relevant/accurate—estimate was derived from comprehensive and highly relevant data/little or no data transformation was required)
B. 75 to less than 90 (relevant/accurate)
C. 45 to less than 75 (moderately relevant/accurate—estimate was derived from reasonably comprehensive and relevant data/moderate transformations required, taking into account known trends in the underlying data, such as over time or age-distributions)

D. 30 to less than 45 (somewhat relevant/accurate)

E. Less than 30 (questionable relevance/accuracy—use with caution, as estimate was derived from less comprehensive or relevant data/moderate transformations required with trends unknown or unaccounted for).

Subnational and 2003 estimates

The data and methods used for 2011 estimates underpinned the subnational and 2003 estimates, so the quality of subnational and 2003 estimates must be considered together with the broad subnational and 2003 methods described in Chapter 2 ('Overarching methods and choices'), and the specific details described in Chapter 5 ('Disease specific methods') and Chapter 7 ('Individual risk factor methods').

Derived ratings

Fatal estimates

Using the ABDS quality index, the mortality data were considered to be comprehensive and relevant with little or no transformation required other than the redistribution of a small proportion of deaths that were not considered appropriate for burden of disease analyses (see section on impact of redistribution in Chapter 3). Therefore, all fatal burden estimates are highly indicative of the YLL due to these diseases. One exception to this is the fatal injury burden by nature of injury, as injury-related deaths are classified by the external cause—subsequent mapping was needed to estimate the fatal burden by nature.

Non-fatal estimates

Appendix Table F3 lists the quality index for YLD assigned to each disease, and a concise summary of any data issues. Each rating must be interpreted carefully together with the statement accompanying the index and the disease specific methods described in Chapter 5. Care is needed when using estimates that have a rating of E, which are considered to be of questionable dependability.

Attributable burden estimates

The quality index ratings for risk factor estimates, and a summary of key data issues and gaps are listed in Appendix Table F4. For each risk factor, it was only possible to rate the quality of the data used to estimate the direct PAF or the exposure data used to calculate the PAF. Many other inputs (such as relative risks) were included in these calculations, as described in chapters 6 and 7, but it was not feasible in the scope of this project to determine the quality of these inputs.

For risk factors with multiple measures of exposure such as tobacco use, the quality measures in Appendix Table F4 have been summarised to reflect the measures with the most attributable burden. Each rating should be interpreted together with the statement accompanying the index and the risk factor-specific methods described in Chapter 7.

Appendix A: Additional information and tables for Chapter 2

Assessment of data sources

National and Indigenous-specific data sources were used to compile mortality and morbidity data for YLL and YLD calculations. Administrative data sets and surveys were primary sources of data, supplemented by epidemiological studies.

Administrative data sources (for example death registers, disease registers, hospitalisations) were evaluated for their level of ascertainment and coverage. Surveys were evaluated for their representativeness, potential selection bias and measurement bias (validity and reliability of measurement).

Epidemiological studies were assessed for the quality of the study design, their timeliness, credibility, representativeness, and sources of bias or error.

Potential sources for morbidity data were required to have a comparable case definition, be relevant to the Australian population (or the Indigenous population in the case of Indigenous estimates), and be timely, accurate, reliable and credible.

Published and unpublished data sources were assessed according to the criteria in Box A1. These were largely based on the ABS's Data Quality Framework, but modified in some areas to better suit the range of data sources used for burden of disease analyses, including epidemiological studies. Note that not all of the criteria were applicable to all types of data sources assessed, and not all dimensions were weighted equally, as the importance of each dimension depended on the type of data source.

Box A1: Criteria for data selection for burden of disease estimates

Comparability

The data source should use a case definition that is comparable with that used for the study. The case definition will be decided on a case-by-case basis for each disease in the disease list. The 3 levels of comparability are:

1. consistent if the case definition is the same as the reference definition
2. comparable if the case definitions can be aligned
3. inconsistent if the case definitions are different, and cannot be aligned.

Relevance and representativeness

Consideration should be given to the relevance and representativeness of the study population to the target population. Estimates should ideally use a national data source that includes Australians (in the case of national estimates), and/or Indigenous Australians (in the case of Indigenous estimates). If these are not available for a particular condition, data sources specific to a subpopulation or region within Australia, or data sources for another country with similar economic or cultural characteristics (such as New Zealand, United Kingdom, United States of America and Canada) can be used, provided that the data can be adjusted so that the estimates are representative of the whole population of interest.

(continued)

Box A1 (continued): Criteria for data selection for burden of disease estimates

The 4 options for relevance/representativeness of national estimates are:

1. the Australian population (national)
2. the Australian population (subnational)
3. a sub or super-regional population (includes New Zealand, United Kingdom, United States of America and Canada)
4. another population.

The 4 options for relevance/representativeness for Indigenous estimates are:

1. the Indigenous Australian population (national)
2. the Indigenous Australian population (subnational)
3. the Australian population (total or non-Indigenous)
4. another population (Indigenous populations in New Zealand, the United State of America, Canada).

Currency

The data source should ideally have been collected within 5 years of the ABDS reference year, with the 3 options for currency being:

1. 2007 or later
2. 2000–2006
3. Before 2000.

Accuracy

The data source should ideally have more than 90% case ascertainment or coverage of the population of interest, and a relative standard error (RSE) or confidence interval (CI) of less than 25%.

Ascertainment/coverage

The 3 options for ascertainment/coverage are:

1. more than 90% or above ascertainment or coverage
2. 60–90% ascertainment or coverage
3. below 60% ascertainment or coverage

Error (sampling/non-sampling)

The 3 options for sources of error are:

1. RSE or CI width of less than 25% of the estimate
2. RSE or CI width 25–50% of the estimate
3. RSE or CI width greater than 50% of the estimate.

Measurement error

Data surrounding physiological and biomedical risk factors should ideally be collected and reported by clinical tests, or using similar tests in a survey setting. Self-reported data may be used, but need to be assessed for validity. The 2 options for measurement error are:

1. clinically reported or measurement data
2. self-reported data.

(continued)

Box A1 (continued): Criteria for data selection for burden of disease estimates

Validation

Validated data sources are preferred. In the case of surveys, the questionnaire should have been validated against a gold standard measurement. In the case of administrative data, the data should have been validated by the agency or organisation that manages the data collection. In the case of epidemiological studies, the results should have been validated against results from other studies to determine whether they were plausible. The 2 options for validation are:

1. validated
2. not validated.

Data sources that could not be validated, or were validated but showed poor results should be scored the same as 'Not validated'.

Credibility

The data source should be collected and/or managed by a credible institution such as a national or state/territory statistical agency or a recognised university or research organisation. For epidemiological studies, ideally, estimates from the data source are preferred to have been published and peer-reviewed. The 4 options for credibility of the estimates are:

1. published and peer-reviewed
2. published but not peer-reviewed
3. not published but peer-reviewed
4. not published and not peer-reviewed.

Accessibility/timeliness

The data source at the required level of disaggregation must be available to the AIHW with enough time for analysis. The 3 options for availability of data are:

1. currently available
2. available with enough time for analysis
3. unlikely to be available with enough time for analysis.

Scoring

Each data source was scored against the matrix in Table A1:

- Any data source scoring predominantly high was included in the ABDS, provided that:
  - components of comparability, relevance/representativeness, currency and accuracy (ascertainment/coverage) were high or medium for administrative data
  - components of comparability, relevance/representativeness, currency and accuracy (non-random error) were scored high or medium for survey data
  - components of comparability, relevance/representativeness, currency and credibility were scored high or medium for epidemiological studies.

  In some circumstances, some data were incorporated from a data source that was rated low against, for example, currency or accuracy if that source scored highly against other criteria, and its characteristics complemented another data source.
- A data source scoring predominantly medium was used if no other data sources for the relevant condition existed, or if there were issues of availability of better data.

A data source scoring predominantly low was not included.

Table A1: Assessment matrix for data sources to be used in ABDS 2011

<table>
<tr><td colspan="2">Data source</td><td colspan="9"></td></tr>
<tr><td colspan="2">Data provider</td><td colspan="9"></td></tr>
<tr><td colspan="2">Level of disaggregation</td><td colspan="9"></td></tr>
<tr><td rowspan="2">Rating</td><td rowspan="2">Comparability</td><td colspan="2">Relevance/representativeness</td><td rowspan="2">Currency</td><td colspan="3">Accuracy</td><td rowspan="2">Validation</td><td rowspan="2">Credibility</td><td rowspan="2">Accessibility/ timeliness</td></tr>
<tr><td>Australian</td><td>Indigenous</td><td>Ascertainment/ coverage</td><td>Error (sampling/ non-sampling)</td><td>Measurement error</td></tr>
<tr><td>High</td><td>Consistent</td><td>National</td><td>National</td><td>2007 or later</td><td>More than 90%</td><td>Less than 25% RSE</td><td>Clinically reported or measured</td><td rowspan="2">Validated</td><td>Published and Peer reviewed</td><td>Currently available</td></tr>
<tr><td rowspan="2">Medium</td><td rowspan="2">Comparable</td><td>Subnational</td><td>Subnational</td><td rowspan="2">2000–2006</td><td rowspan="2">60–90%</td><td rowspan="2">25–50% RSE</td><td rowspan="2">Self-reported</td><td>Published but not peer reviewed</td><td rowspan="2">Expected to be available in time for analysis</td></tr>
<tr><td>Sub or super-regional (such as New Zealand, United States, Canada)</td><td>Total Australian or non-Indigenous</td><td rowspan="2">Not validated</td><td>Not published but peer reviewed</td></tr>
<tr><td>Low</td><td>Inconsistent</td><td>Other</td><td>Other Indigenous population (New Zealand, United States, Canada)</td><td>Before 2000</td><td>Less than 60%</td><td>More than 50% RSE</td><td>Not known</td><td>Not published nor peer reviewed</td><td>Unlikely to be available in time for analysis.</td></tr>
</table>

Additional tables

Table A2: ABDS 2011 disease list by ICD-10 code

ABDS 2011 disease/injury	ICD-10 codes[a]
Blood and metabolic disorders	
Cystic fibrosis	E84
Haemophilia	D66, D67
Haemolytic anaemias	D55–D58
Iron deficiency anaemia	D50.1–D50.9
Protein-energy deficiency	E40, E41, E42, E43, E44, E45, E46
Other blood and metabolic disorders	D50.0, D51–D53, D59.0–D59.2, D59.4–D59.9,D60–D61, D62–D65, D68–D77,D80–D84, D86.1, D86.3, D86.8, D89, E00–E02, E50–E68, E70–E80, E83, E85.0–E85.9, E86–E88, E90
Cancer and other neoplasms	
Mouth and pharyngeal cancer	C00–C14
Laryngeal cancer	C32
Oesophageal cancer	C15
Stomach cancer	C16
Bowel cancer	C18–C20
Liver cancer	C22
Gallbladder cancer	C23, C24
Pancreatic cancer	C25
Lung cancer	C33, C34
Mesothelioma	C45
Melanoma of the skin	C43
Non-melanoma skin cancers	C44
Breast cancer	C50
Cervical cancer	C53
Uterine cancer	C54, C55
Ovarian cancer	C56
Prostate cancer	C61
Testicular cancer	C62
Bladder cancer	C67
Kidney cancer	C64
Brain and central nervous system cancer	C70–C72

(continued)

Table A2 (continued): ABDS 2011 disease list by ICD-10 code

ABDS 2011 disease/injury	ICD-10 codes[a]
Cancer and other neoplasms (continued)	
Thyroid cancer	C73
Non-Hodgkin lymphoma	C82–C86
Hodgkin lymphoma	C81
Leukaemia	C91–C95
Myeloma	C90
Other lymphohaematopoietic (blood) cancers	C88, C96, D45, D46, D47.1, D47.3
Unknown primary	C26, C39, C76–C79, C80, C97
Benign and uncertain brain tumours	D32, D33, D42, D43
Breast in situ	D05
Other malignant neoplasms (cancers)	C17, C21, C26–C31, C37–C38, C40-C41, C46–C49, C51–C52, C57–C60, C63, C65–C66, C68–C69, C74–C75
Other benign, in situ and uncertain neoplasms	D00–D04, D06–D31, D34–D48
Cardiovascular diseases	
Coronary heart disease	I20–I25
Stroke	I60–I69
Rheumatic heart disease	I01–I09
Non-rheumatic valvular disease	I34–I39
Hypertensive heart disease	I11
Atrial fibrillation and flutter	I48
Inflammatory heart disease	I30–I33, I40–I41
Cardiomyopathy	I42–I43
Aortic aneurysm	I71
Peripheral vascular disease	I70.0–I70.8, I72–I74
Other cardiovascular diseases	G45, I00, I10, I13, I15, I26–I28, I44–I47, I49, I50–I52, I70.9, I77–I84, I86–I89, I95, I97–I99
Endocrine disorders	
Diabetes	E10–E14 (excluding E10.2, E11.2, E13.2, E14.2), O24.0–24.3
Other endocrine disorders	E03–E07, E15, E16, E20–E27, E29–E32, E34, E35, E89
Gastrointestinal disorders	
Gastroduodenal disorders	K22.1, K25–K27, K29
Appendicitis	K35–K37

(continued)

Table A2 (continued): ABDS 2011 disease list by ICD-10 code

ABDS 2011 disease/injury	ICD-10 codes[a]
Gastrointestinal disorders (continued)	
Hernia	K40–K43, K45, K46
Vascular disorders of intestine	K55
Intestinal obstruction without hernia	K56
Inflammatory bowel disease	K50–K52
Diverticulitis	K57
Chronic liver disease	B18, I85, K70–K76
Gall bladder and bile duct disease	K80–K83
Pancreatitis	K85, K86
Gastro-oesophageal reflux disorder	K20, K21, K44
Functional gastrointestinal disorders(b)	. .
Other gastrointestinal diseases	K22.0, K22.2-K22.9, K23, K28, K30, K31, K38, K58–K61, K63–K67, K77, K87, K90, K91, K92, K93
Hearing and vision disorders	
Refractive disorders	H49–H52
Cataract and other lens disorders	H25–H27
Glaucoma	H40, H42
Age-related macular degeneration	H35.3
Other vision disorders	H30–H35 (excluding H35.3), H43–H48, H53–H59
Hearing loss	H90–H91
Other hearing and vestibular disorders	H60.2–H60.9, H61, H68–H69, H71–H74, H80–H83, H92–H93
Infant and congenital conditions	
Pre-term birth and low birthweight complications	P01.0, P01.1, P05, P07, P22, P25– P28, P52, P61.2, P77
Birth trauma and asphyxia	P01.7, P01.8, P01.9, P02, P03, P08, P10–P15, P20, P21, P24, P90, P91
Cerebral palsy	G80
Neonatal infections	P23, P35.1–P35.9, P36, P37.1, P37.2, P37.5, P37.8, P37.9, P38, P39
Sudden infant death syndrome	R95
Neural tube defects	Q00, Q01, Q05
Brain malformations	Q02, Q03, Q04, Q86.0
Congenital cardiovascular defects	Q20–Q28
Cleft lip and/or palate	Q35–Q37
Gastrointestinal malformations	Q38.1, Q38.2–Q45

(continued)

Table A2 (continued): ABDS 2011 disease list by ICD-10 code

ABDS 2011 disease/injury	ICD-10 codes[(a)]
Infant and congenital conditions (continued)	
Urogenital malformations	Q50–Q54, Q56–Q60, Q61.4–Q61.9, Q62–Q64
Down syndrome	Q90
Other disorders of infancy	P00, P01.2–P01.6, P04, P29, P50, P51, P53–P60, P61.0–P61.1, P61.3–P61.9, P70–P72, P74–P76, P78–P81, P83, P92–P96
Other chromosomal abnormalities	Q91–Q93, Q95–Q98, Q99.0–Q99.8
Other congenital conditions	Q06, Q07, Q10–Q18, Q30–Q34, Q65–Q87, Q89.0–Q89.8, Q89.9, Q99.9
Infectious diseases	
HIV/AIDS	B20–B24, O98.7
Tuberculosis	A15–A19, B90, N33.0, N74.0, N74.1, P37.0, O98.0
Hepatitis A	B15
Hepatitis B (acute)	B16, B17.0
Hepatitis C (acute)	B17.1, B17.8, B17.9
Syphilis	A50–A53, N29.0, N74.2, O98.1
Gonococcal infection	A54, O98.2, N74.3
Sexually transmitted chlamydial infections	A55–A56, N74.4
Other sexually transmitted infections	A57–A64, O98.3
Campylobacteriosis	A04.5
Salmonellosis	A02
Rotavirus	A08.0
Other gastrointestinal infections	A00–A01, A03–A09 (excluding A04.5 and A08.0), D59.3
Upper respiratory tract infections	J00–J06
Otitis media	H65–H68, H70
Lower respiratory infections	J12, J14–J18, J20–J22, J85–J86
Influenza	J09–J11
Diphtheria	A36
Pertussis	A37
Tetanus	A33–A35
Measles	B05
Rubella	B06, P35.0
Varicella-zoster	B01–B02
Haemophilus influenzae type-B	G00.0

(continued)

Table A2 (continued): ABDS 2011 disease list by ICD-10 code

ABDS 2011 disease/injury	ICD-10 codes[a]
Infectious diseases (continued)	
Pneumococcal disease	G00.1, A40.3, J13
Meningococcal disease	A39
Other meningitis and encephalitis	A83–A87, G00.2–G00.9, G01–G05
Dengue	A90–A91
Ross River virus	B33.1
Barmah Forest virus	A92.8
Malaria	B50–B54, P37.3–P37.4
Trachoma	A71, B94.0
Other infections	A20–A32, A38, A40–A44, A48, A49.0–A49.1, A49.3–A49.9, A65–A70, A74–A82, A88–A89, A95–A99, B00–B04, B07–B09, B17.2, B25–B30, B33.0, B33.2–B33.8, B34–B49, B55–B85,B87–B89, B91, B92 (excluding B92.8), B94.1, B94.8–B94.9, B95–B99, G06
Injuries (external cause)	
Road traffic injuries: motorcyclists	V20.3–V20.9, V21.3–V21.9, V22.3–V22.9, V23.3–V23.9, V24.3–V24.9, V25.3–V25.9, V26.3–V26.9, V27.3–V27.9, V28.3–V28.9, V29.4–V29.9
Road traffic injuries: motor vehicle occupants	V30.4–V30.9, V31.4–V31.9, V32.4–V32.9, V33.4–V33.9, V34.4–V34.9, V35.4–V35.9, V36.4–V36.9, V37.4–V37.9, V38.4–V38.9, V39.4–V39.9, V40.4–V40.9, V41.4–V41.9, V42.4–V42.9, V43.4–V43.9, V44.4–V44.9, V45.4–V45.9, V46.4–V46.9, V47.4–V47.9, V48.4–V48.9, V49.4–V49.9, V50.4–V50.9, V51.4–V51.9, V52.4–V52.9, V53.4–V53.9, V54.4–V54.9, V55.4–V55.9, V56.4–V56.9, V57.4–V57.9, V58.4–V58.9, V59.4–V59.9, V60.4–V60.9, V61.4–V61.9, V62.4–V62.9, V63.4–V63.9, V64.4–V64.9, V65.4–V65.9, V66.4–V66.9, V67.4–V67.9, V68.4–V68.9, V69.4–V69.9, V70.4–V70.9, V71.4–V71.9, V72.4–V72.9, V73.4–V73.9, V74.4–V74.9, V75.4–V75.9, V76.4–V76.9, V77.4–V77.9, V78.4–V78.9, V79.4–V79.9, V87.0–V87.8, V89.2
Road traffic injuries: other	V01.1, V01.9, V02.1, V02.9, V03.1, V03.9, V04.1, V04.9, V05.1, V05.9, V06.1, V06.9, V09.2, V09.3, V09.9, V10.3, V10.4, V10.5, V10.9, V11.3, V11.4, V11.5, V11.9, V12.3, V12.4, V12.5, V12.9, V13.3, V13.4, V13.5, V13.9, V14.3, V14.4, V14.5, V14.9, V15.3, V15.4, V15.5, V15.9, V16.3, V16.4, V16.5, V16.9, V17.3, V17.4, V17.5, V17.9, V18.3, V18.4, V18.5, V18.9, V19.4, V19.5, V19.6, V19.8, V19.9, V87.9

(continued)

Table A2 (continued): ABDS 2011 disease list by ICD-10 code

ABDS 2011 disease/injury	ICD-10 codes[a]
Injuries (external cause) (continued)	
Other land transport injuries	V01.0, V02.0, V03.0, V04.0, V05.0, V06.0, V09.0, V09.1, V10.0–V10.2, V11.0–V11.2, V12.0–V12.2, V13.0– V13.2, V14.0–V14.2, V15.0–V15.2, V16.0–V16.2, V17.0–V17.2, V18.0–V18.2, V19.0–V19.3, V20.0–V20.2, V21.0–V21.2, V22.0–V22.2, V23.0–V23.2, V24.0– V24.2, V25.0–V25.2, V26.0–V26.2, V27.0–V27.2, V28.0–V28.2, V29.0–V29.3, V30.0–V30.3, V31.0–V31.3, V32.0–V32.3, V33.0–V33.3, V34.0–V34.3, V35.0–V35.3, V36.0–V36.3, V37.0–V37.3, V38.0–V38.3, V39.0–V39.3, V40.0–V40.3, V41.0–V41.3, V42.0–V42.3, V43.0–V43.3, V44.0–V44.3, V45.0–V45.3, V46.0–V46.3, V47.0–V47.3, V48.0–V48.3, V49.0–V49.3, V50.0–V50.3, V51.0–V51.3, V52.0–V52.3, V53.0–V53.3, V54.0–V54.3, V55.0–V55.3, V56.0–V56.3, V57.0–V57.3, V58.0–V58.3, V59.0–V59.3, V60.0–V60.3, V61.0–V61.3, V62.0–V62.3, V63.0–V63.3, V64.0–V64.3, V65.0–V65.3, V66.0–V66.3, V67.0–V67.3, V68.0–V68.3, V69.0–V69.3, V70.0–V70.3, V71.0–V71.3, V72.0–V72.3, V73.0–V73.3, V74.0–V74.3, V75.0–V75.3, V76.0–V76.3, V77.0–V77.3, V78.0–V78.3, V79.0–V79.3, V80–V86, V88, V89.0, V89.1, V89.3, V89.9, Y85.9
Poisoning	X40–X49
Falls	W00–W19
Fire, burns and scalds	X00–X06, X08–X19
Drowning	V90, V92, W65–W74
Other unintentional injuries	V91, V93–V99, W20–W64, W75–W99, X20–X39, X50–X58, Y35, Y36, Y86, Y89.0, Y89.1
Suicide and self-inflicted injuries	X60–X84, Y87.0
Homicide and violence	X85–Y09, Y87.1
All other external causes of injury	Y40–Y84, Y88
Injuries (nature)	
Traumatic brain injury	S02.0, S02.1, S02.7, S02.9, S06
Spinal cord injury	S14.0, S14.1, S14.7, S24.0, S24.1, S24.7, S34.0, S34.1, S34.7, T06.0, T06.1, T09.3
Internal and crush injuries	S07, S17, S18, S22.4, S22.5, S25, S26, S27, S28, S29.7, S35, S36, S37, S38.0, S38.1, S39.6, S39.7, S47, S57, S67, S77, S87, S97, T04, T06.5, T14.7
Poisoning	T36–T65
Hip fracture	S72
Tibia and ankle fracture	S82
Humerus fracture	S42.2, S42.3, S42.4, S42.7
Other fractures	S02.2– S02.6, S02.8, S12, S22.0– S22.3, S22.8, S22.9, S32, S42.0–S42.1, S42.8–42.9, S49.7, S52, S59.7, S62.0–62.8, S69.7, S82.0, S92, T02, T08, T10, T12, T14.2

(continued)

Table A2 (continued): ABDS 2011 disease list by ICD-10 code

ABDS 2011 disease/injury	ICD-10 codes[(a)]
Injuries (nature)(continued)	
Drowning and submersion injuries	T75.1
Dislocations	S03.0–S03.3–S13.3–S23.2, S33.1, S33.3, S43.0–S43.3, S53.0, S53.1, S63.0–S63.2, S73.0, S83.0 ,S83.1, S93.0, S93.1, S93.3, T03, T09.2, T11.2, T13.2, T14.3
Soft tissue injuries(c)	S03.4, S03.5, S13.4S13.6, S16, S23.0, S23.3S23.5, S29.0, S33.5S33.7, S39.0, S43.4, S43.5, S43.6, S43.7, S46, S53.2, S53.3, S53.4, S56, S63.3, S63.4, S63.5, S63.6, S63.7, S66, S73.1, S76, S83.2, S83.3, S83.4, S83.5, S83.6, S83.7, S86, S93.2, S93.4, S93.5, S93.6, S96, T06.4, T09.5, T11.5, T13.5, T14.6
Burns	T20–T31
Other injuries	S00, S01, S04, S05, S08–S11, S13.0, S14.2–S14.6, S15, S19, S20, S21, S24.2–S24.6, S29.8, S29.9, S30, S31, S33.0, S33.4, S34.2–S34.6, S34.8, S38.2, S38.3, S39.8, S39.9, S40, S41, S44, S45, S48, S50, S51, S54, S55, S58, S59.8, S59.9, S60, S61, S64, S65, S68, S69.8, S69.9, S70, S71, S74, S75, S78, S80, S81 S84, S85, S88–S91, S94, S95, S98, S99, T00, T01, T05, T06.2, T06.3, T06.8, T07, T09.0, T09.1, T09.4, T09.6, T09.8, T09.9, T11.0, T11.1, T11.3, T11.4, T11.6, T11.8, T11.9, T13.0, T13.1, T13.3, T13.4, T13.6, T13.8, T13.9, T14.0, T14.1, T14.4, T14.5, T14.8, T14.9, T15–T19, T33–T35, T66–T75,T79, T80, T81, T88
Kidney and urinary diseases	
Chronic kidney disease	E10.2, E11.2, E12.2, E13.2, E14.2, I12, N02–N07, N08, N11–N16, N18, N39.1, N39.2, Q61.0–Q61.3
Enlarged prostate	N40
Kidney stones	N20, N21
Other kidney and urinary diseases	N00, N01, N10, N22, N23, N25–N28, N30–N32, N34–N37,
Mental and substance use disorders	
Depressive disorders	F32, F33, F34.1, F34.8–F39
Anxiety disorders	F40–F43
Bipolar affective disorder	F30, F31, F34.0
Alcohol use disorders	F10
Drug use disorders (excluding alcohol)	F11–F16, F18, F19
Schizophrenia	F20–F25, F28, F29
Eating disorders	F50
Autism spectrum disorders	F84
Attention deficit hyperactivity disorder	F90
Conduct disorder	F91, F92
Intellectual disability	F70–F73, F78, F79

(continued)

Table A2 (continued): ABDS 2011 disease list by ICD-10 code

ABDS 2011 disease/injury	ICD-10 codes[a]
Mental and substance use disorders (continued)	
Other mental and substance use disorders	F04–F07, F09, F17, F44, F45, F48, F51–F55, F59–F66, F68, F69, F80–F83, F88, F89, F93–F95, F98, F99
Musculoskeletal conditions	
Osteoarthritis	M15–M19
Gout	M10
Rheumatoid arthritis	M05, M06, M08
Back pain and problems	M40, M41, M45–M51, M53, M54, M99
Other musculoskeletal conditions	M00–M03, M07, M09, M11–M14, M20–M25, M30–M36, M42, M43, M60–M63, M65–M68, M70–M73, M75–M77, M79–M96
Neurological conditions	
Epilepsy	G40, G41
Dementia	F01–F03, G30–G31
Parkinson's disease	G20
Multiple sclerosis	G35
Migraine	G43
Motor neurone disease	G12.2
Guillain-Barré syndrome	G61.0
Other neurological conditions	G08–G09, G11, G12.0, G12.1, G12.8, G12.9, G13, G21–G26, G31–G32, G36–G37, G44, G46–G47, G50–G60, G61.1–G61.9, G62–64, G70–G73, G81–83, G90–G99
Oral disorders	
Dental caries	K02, K04
Periodontal disease	K05
Severe tooth loss[d]	. .
Other oral disorders	K00, K01, K03, K06–K14
Reproductive and maternal conditions	
Maternal haemorrhage	O44.1, O45–O46, O67, O72
Maternal infections	O41.1, O85–O86
Hypertensive disorders of pregnancy	O10–O16
Obstructed labour	O64–O66
Early pregnancy loss	O00–O08
Gestational diabetes	O24.4

(continued)

Table A2 (continued): ABDS 2011 disease list by ICD-10 code

ABDS 2011 disease/injury	ICD-10 codes[(a)]
Reproductive and maternal conditions (continued)	
Other maternal conditions	O20–O23, O25–O26, O28–O36, O40, O42–O43, O44.0, O47–48, O60–O63, O68–O71, O73–O75, O80–O84, O87–O92, O95–O97, O98.4–O98.6, O98.8–O98.9, O99
Endometriosis	N80
Uterine fibroids	D25
Genital prolapse	N81, K62.2, K62.3
Polycystic ovarian syndrome	E28.2
Infertility	N46, N97
Other reproductive conditions	N43–N45, N47–N50, N60, N62–N64, N70–73, N74.8, N75–N77, N82–N83, N84–N90, N91–N96, N98–N99, O94
Respiratory diseases	
Asthma	J45, J46
Chronic obstructive pulmonary disease	J40– J44
Interstitial lung disease	J84
Sarcoidosis	D86.0, D86.2, D86.9
Pneumoconiosis	J60–J65
Upper respiratory diseases	J30–J33, J34.1–J34.9,J35–J39
Other chronic respiratory diseases	J47, J66–J68, J70, J80–J82, J90–J96, J98–J99
Skin disorders	
Dermatitis and eczema	L20, L21–L25, L26, L27, L30
Psoriasis	L40
Acne	L70
Ulcers	L89, L97, L98.4
Skin infections (including cellulitis)	A46, B08.1, B08.4, B86, H00.0, H60.0, H60.1, J34.0, L00–L04, L08
Other skin disorders	L05, L10–L13, L28, L29, L41–L45, L50–L60, L62, L63–L68, L71–L88, L90–L95, L98.0, L98.1, L98.2, L98.3, L98.5, L98.6, L98.8, L98.9

(a) The ICD codes shown here describe the ABDS diseases generally. They include some codes that were used to redistribute deaths. See Appendix Table B2 for a full list of ICD-10 codes used to identify deaths for redistribution. ICD codes were not necessarily the basis of the morbidity (non-fatal) estimates, as this depended on the data source used. Codes have only been specified to the fourth or fifth digit where necessary.

(b) Criteria used to diagnose this condition are currently not defined in ICD-10. See 'Gastrointestinal disorders' in Chapter 5 for further details.

(c) A small portion of soft tissue injuries were inadvertently omitted from the ABDS 2011 study. The omission relates to ICD codes S43.4–S43.7 and comprises around 7% of soft tissue injury YLD and around 0.1% of all injury YLD.

(d) Criteria used to diagnose this condition are currently not defined in ICD-10. See 'Oral disorders' in Chapter 5 for further details.

Source: WHO 2016.

Table A3: Data source used for subnational distribution of Indigenous non-fatal burden estimates

	State/territory	Remoteness	Socioeconomic group
Infections	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Infant/congenital	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Cancer	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Cardiovascular	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Respiratory	2012–13 AATSIHS	2012–13 AATSIHS	Adjusted hospitalisations
Gastrointestinal	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Neurological	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Mental/substance use	2012–13 AATSIHS	2012–13 AATSIHS	Adjusted hospitalisations
Endocrine	2012–13 AATSIHS	2012–13 AATSIHS	Adjusted hospitalisations
Kidney/urinary	2012–13 AATSIHS	2012–13 AATSIHS	Adjusted hospitalisations
Reproductive/maternal	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Musculoskeletal	2012–13 AATSIHS	2012–13 AATSIHS	Adjusted hospitalisations
Hearing/vision	2012–13 AATSIHS	2012–13 AATSIHS	Adjusted hospitalisations
Skin	Population distribution	Population distribution	Adjusted hospitalisations
Oral	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Blood/metabolic	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Injuries	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations

Table A4: Proportions used for distribution of non-fatal burden estimates, by state/territory and Indigenous status (%)

	Indigenous				Non-Indigenous			
	NSW	Qld	WA	NT	NSW	Qld	WA	NT
Infections	23.2	25.3	17.0	21.4	29.7	21.7	9.7	0.6
Infant/congenital	31.4	27.2	11.6	10.0	32.6	18.5	10.8	0.6
Cancer	30.5	27.6	11.7	7.5	27.5	23.1	10.6	0.4
Cardiovascular	26.8	27.6	15.0	12.1	30.5	20.3	9.6	0.6
Respiratory	38.5	24.9	11.7	4.7	30.7	19.9	10.9	0.7
Gastrointestinal	30.0	24.8	14.5	10.1	30.9	20.5	9.7	0.6
Neurological	29.9	27.6	13.8	8.7	25.0	22.4	10.6	0.4
Mental/substance use	32.3	28.6	12.8	8.3	31.7	19.5	10.0	0.6
Endocrine	35.4	20.9	19.0	12.0	36.4	22.9	9.8	1.1
Kidney/urinary	25.8	27.2	17.1	20.1	34.7	20.2	10.9	0.7
Reproductive/maternal	27.8	29.7	13.5	12.4	30.3	21.3	10.6	1.0
Musculoskeletal	33.4	28.2	10.9	6.3	33.4	19.4	10.5	0.6
Hearing/vision	32.3	28.6	12.8	8.3	33.4	19.8	10.0	0.7
Skin	31.1	28.2	13.2	10.3	32.3	19.8	10.5	0.7
Oral	22.4	27.4	14.0	13.1	25.3	18.8	13.8	0.4
Blood/metabolic	20.8	25.5	14.4	22.5	23.8	21.0	11.9	0.4
Injuries	24.1	24.7	19.4	16.7	30.1	20.4	10.5	0.8

Note: Proportions for respiratory diseases, endocrine disorders, kidney and urinary disorders, musculoskeletal conditions, mental and substance use disorders, and hearing and vision disorders were calculated from the AATSIHS 2012–13 (Indigenous) and AHS 2011–12 (non-Indigenous). Proportions for skin disorders were based on the Indigenous and non-Indigenous population distributions. All other disease group proportions were calculated from the NHMD.

Table A5: Proportions used for distribution of non-fatal burden, by remoteness and Indigenous status (%)

	Indigenous					Non-Indigenous				
	Major cities	Inner regional	Outer regional	Remote	Very remote	Major cities	Inner regional	Outer regional	Remote	Very remote
Infections	24.3	16.2	20.9	15.4	23.2	66.3	21.3	10.2	1.5	0.7
Infant/congenital	35.2	23.8	20.4	7.9	12.7	71.4	19.1	8.0	1.1	0.4
Cancer	35.7	23.7	20.0	9.0	11.5	67.6	22.1	8.9	1.1	0.3
Cardiovascular	27.1	20.4	22.6	13.6	16.3	64.9	23.5	10.0	1.2	0.4
Respiratory	42.5	25.4	19.8	6.2	6.1	69.4	20.3	8.7	1.2	0.4
Gastrointestinal	33.7	20.7	22.0	11.4	12.2	68.1	21.6	8.9	1.1	0.4
Neurological	34.6	21.7	20.4	12.2	11.1	68.2	21.3	9.1	1.1	0.4
Mental/substance use	37.7	21.0	23.2	6.7	11.1	70.0	19.8	8.1	1.5	0.6
Endocrine	28.8	17.4	25.6	9.8	18.4	67.7	22.0	8.8	1.1	0.4
Kidney/urinary	23.2	12.2	20.8	12.2	31.4	74.0	19.7	5.4	0.7	0.2
Reproductive/maternal	29.9	20.6	23.7	10.7	15.2	71.2	17.9	8.9	1.4	0.5
Musculoskeletal	38.3	24.9	20.8	7.3	8.6	67.4	21.8	9.0	1.4	0.4
Hearing/vision	35.2	23.1	22.7	7.9	11.2	69.6	20.1	8.8	1.2	0.4
Skin	34.8	22.0	21.8	7.7	13.7	71.3	18.3	8.7	1.2	0.5
Oral	29.9	22.6	19.3	11.6	16.6	69.4	19.7	9.4	1.1	0.3
Blood/metabolic	25.1	18.9	23.2	11.7	21.2	68.7	20.9	9.0	1.1	0.3
Injuries	27.5	16.9	20.0	15.2	20.4	66.1	21.6	10.1	1.6	0.6

Note: Proportions for respiratory diseases, endocrine disorders, kidney and urinary disorders, musculoskeletal conditions, mental and substance use disorders, and hearing and vision disorders were calculated from the AATSIHS 2012–13 (Indigenous) and AHS 2011–12 (non-Indigenous). Proportions for skin disorders were based on the Indigenous and non-Indigenous population distributions. All other disease group proportions were calculated from the NHMD.

Table A6: Proportions used for distribution of non-fatal burden in Indigenous Australians, by socioeconomic group (%)

	Indigenous (Indigenous Relative Socioeconomic Outcomes Index)				
	Q1 (lowest)	Q2	Q3	Q4	Q5 (highest)
Infections	13.3	19.6	28.9	16.1	22.1
Infant/congenital	17.9	25.7	29.3	16.8	10.3
Cancer	25.0	25.5	25.1	15.4	9.0
Cardiovascular	14.9	21.0	27.0	20.2	17.0
Respiratory	15.7	23.5	28.8	16.9	15.1
Gastrointestinal	17.3	24.4	28.1	17.9	12.3
Neurological	20.6	24.6	26.3	18.5	10.0
Mental/substance use	22.8	22.0	28.7	15.4	11.2
Endocrine	12.3	20.1	27.2	21.0	19.5
Kidney/urinary	17.0	24.3	27.3	15.6	15.9
Reproductive/maternal	16.3	23.8	29.3	17.1	13.6
Musculoskeletal	20.6	24.3	25.8	15.6	13.7
Hearing/vision	14.8	21.1	26.6	16.3	21.1
Skin	12.5	18.3	27.8	16.2	25.2
Oral	16.8	20.3	26.9	18.5	17.6
Blood/metabolic	14.8	20.9	24.6	17.7	22.0
Injuries	14.7	21.0	27.6	16.6	20.1

Note: All proportions were calculated from the NHMD

Appendix B: Additional information and tables for Chapter 3

This appendix provides the additional tables and information for the methods used to estimate the fatal burden described in Chapter 3.

Table B1: Indigenous mortality adjustment factors used for 2011 YLL estimates

Level of reporting	Disaggregation	ABS Census Data Enhancement Study (2011–12) adjustment factor	AIHW Enhanced Mortality Database project (2008–2010) adjustment factor
National and socioeconomic group estimates	0–14 years	1.21	. .
	15–59 years	1.12	. .
	60 years and over	1.29	. .
State/territory estimates	NSW	1.42	. .
	Qld	1.24	. .
	WA	1.14	. .
	NT	0.96	. .
Remoteness estimates	Major cities	. .	1.25
	Inner regional	. .	1.22
	Outer regional	. .	1.12
	Remote	. .	1.04
	Very remote	. .	1.02

Sources: ABS 2013c; AIHW 2012a.

Table B2: Number and proportion of deaths by redistribution group, method and target diseases

Redistribution group	ICD-10 codes	Method	Scope of target diseases[a]	Number	%
Non-specific cancers	C76–C80	Direct evidence	Cancer	2,442	16.5
Non-specific digestive cancers	C26	Direct evidence	Cancer (digestive cancers)	1,247	8.4
Unknown causes	R99	Direct evidence	All diseases	636	4.3
Undetermined intent	Y10–Y34	Direct evidence	All diseases	269	1.8
Heart failure	I50	Direct evidence and indirect MCOD	Cardiovascular, infant/congenital	2,903	19.7
Renal failure	N17, N19	Direct evidence and indirect MCOD	Partial kidney/urinary, all diseases	1,117	7.6
Unspecified gastrointestinal causes	K92	Direct evidence and indirect MCOD	Gastrointestinal	407	2.8
Peritonitis	K65–K66	Direct evidence and indirect MCOD	Gastrointestinal	93	0.6
Septicaemia, pneumonitis	A40 (excluding A40.3), A41, J69	Indirect MCOD	All diseases	2,765	18.7
Hypertension	I10, I13, I15	Indirect MCOD	All diseases excluding injuries	599	4.1
All other non-specific, intermediate and immediate causes	A48.0, A48.3, B19, B94.2, E86–E87, F99, G81–G83, H00.1–H59.9, H60.2–H62.8, H67, H69, H71–H95, I46, J96, K712, L04, L21–L25, L27–L30, L41–L45, L52–L53, L55–L60, L63–L85, L87, L90–L92, L94, L98.0, L98.1, L98.8, L98.9, N51, N60–N61, N70–N73, N748, N84–N90, O94, R09–R63, R65–R94, R96–R98, Y87.2, Y89.9, Y90–Y98	Proportional allocation	All diseases	1,289	8.7
Unspecified factor	X59	Proportional allocation	Injuries	704	4.8
Cardiac signs and symptoms, unspecified digestive diseases and congenital anomalies	I70.9, Q10–Q18, Q38.1, Q54, Q65–Q74, Q82–Q84, Q89.9, Q99.9, R00–R03	Proportional allocation	All diseases excluding infections, cancer and injuries	152	1.0
Unspecified amyloidosis, unspecified respiratory signs and symptoms and cachexia	E85.3–E85.9, R04–R07, R64	Proportional allocation	All diseases excluding injuries	138	0.9
All redistribution causes				**14,761**	**100.0**

(a) Reproductive/maternal, oral and vision/hearing are excluded from the scope of target diseases, due to small numbers of deaths in these disease groups.

Table B3: Number of deaths identified for redistribution and associated YLL, by age and sex, 2011

	Deaths			YLL		
	Males	**Females**	**Persons**	**Males**	**Females**	**Persons**
Under 1	42	26	68	3,613	2,237	5,849
1–4	12	6	18	1,013	510	1,523
5–9	2	6	8	162	475	636
10–14	3	1	4	219	72	291
15–19	13	5	18	904	344	1,248
20–24	26	10	36	1,679	641	2,319
25–29	31	20	51	1,838	1,179	3,017
30–34	53	28	81	2,881	1,533	4,414
35–39	66	36	102	3,278	1,788	5,066
40–44	88	47	135	3,921	2,071	5,992
45–49	135	74	209	5,364	2,936	8,299
50–54	210	126	336	7,370	4,424	11,794
55–59	265	158	423	8,107	4,835	12,942
60–64	369	199	568	9,556	5,130	14,685
65–69	413	277	690	8,839	5,944	14,784
70–74	630	464	1,094	10,783	7,932	18,715
75–79	813	624	1,437	10,628	8,162	18,790
80–84	1,242	1,195	2,437	11,921	11,362	23,283
85–89	1,317	1,819	3,136	8,732	11,931	20,663
90–94	877	1,581	2,458	3,948	6,997	10,945
95–99	301	896	1,197	905	2,665	3,570
100 and over	39	216	255	82	440	522
All ages	**6,947**	**7,814**	**14,761**	**105,741**	**83,605**	**189,345**

Source: NMD.

Table B4: Number and proportion of deaths before and after redistribution and associated change (increase), by disease group: National

	Before redistribution				After redistribution				Increase (before to after)			
	Deaths		YLLs		Deaths		YLLs		Deaths		YLLs	
Disease group	Number	%	Number	%	Number	%	Number	%	Number	%	Number	%
Blood/metabolic	1,661	1.1	29,095	1.3	1,761	1.2	30,704	1.4	100	6.0	1,609	5.5
Cancer	39,974	27.2	709,243	31.2	44,757	30.5	782,349	34.5	4,783	12.0	73,106	10.3
Cardiovascular	41,123	28.0	478,314	21.1	46,144	31.5	523,024	23.0	5,021	12.2	44,710	9.3
Endocrine	4,187	2.9	53,836	2.4	4,420	3.0	56,499	2.5	233	5.6	2,663	4.9
Gastrointestinal	4,730	3.2	77,062	3.4	5,640	3.8	87,110	3.8	910	19.2	10,048	13.0
Infant/congenital	1,267	0.9	95,229	4.2	1,384	0.9	101,060	4.5	117	9.2	5,831	6.1
Infections	3,418	2.3	43,265	1.9	3,625	2.5	46,418	2.0	207	6.1	3,153	7.3
Injuries	8,277	5.6	282,621	12.4	9,665	6.6	310,194	13.7	1,388	16.8	27,573	9.8
Kidney/urinary	3,111	2.1	33,114	1.5	3,828	2.6	39,572	1.7	717	23.0	6,458	19.5
Mental	763	0.5	16,542	0.7	828	0.6	17,853	0.8	65	8.5	1,311	7.9
Musculoskeletal	1,177	0.8	14,391	0.6	1,281	0.9	15,613	0.7	104	8.8	1,222	8.5
Neurological	13,330	9.1	134,316	5.9	14,062	9.6	141,523	6.2	732	5.5	7,207	5.4
Oral	30	0.0	345	0.0	30	0.0	345	0.0	0	0.0	0	0
Reproductive/maternal	38	0.0	1,132	0.0	39	0.0	1,179	0.1	1	2.6	47	4.1
Respiratory	8,439	5.8	107,696	4.7	8,743	6.0	111,382	4.9	304	3.6	3,686	3.4
Skin	426	0.3	4,555	0.2	506	0.3	5,276	0.2	80	18.8	721	15.8
Redistribution	14,761	10.1	189,345	8.3	0	0.0	0	0.0	. .	. .	. .	. .
All deaths	**146,712**	**100.0**	**2,270,101**	**100.0**	**146,712**	**100.0**	**2,270,101**	**100.0**	**. .**	**. .**	**. .**	**. .**

Notes

1. Hearing/vision is not shown, as there were no deaths due to these causes.
2. Reproductive/maternal, oral and vision/hearing are excluded from the scope of target diseases, due to small numbers of deaths in these disease groups.

Table B5: Number and proportion of deaths before and after redistribution and associated change (increase), by disease group: Indigenous

	Before redistribution				After redistribution				Increase (before to after)			
	Deaths		YLLs		Deaths		YLLs		Deaths		YLLs	
Disease group	Number	%	Number	%	Number	%	Number	%	Number	%	Number	%
Blood/metabolic	39	1.3	1,202	1.2	42	1.4	1,312	1.3	3	7.7	110	9.2
Cancer	596	19.5	15,334	15.2	678	22.2	17,370	17.3	82	13.8	2,036	13.3
Cardiovascular	714	23.4	19,614	19.5	775	25.4	20,970	20.8	61	8.5	1,356	6.9
Endocrine	206	6.7	5,244	5.2	210	6.9	5,359	5.3	4	1.9	115	2.2
Gastrointestinal	154	5.1	5,279	5.2	171	5.6	5,676	5.6	17	11.0	397	7.5
Infant/congenital	108	3.5	8,874	8.8	119	3.9	9,734	9.7	11	10.2	860	9.7
Infections	84	2.7	3,011	3.0	89	2.9	3,270	3.2	5	6.0	259	8.6
Injuries	416	13.6	22,274	22.1	459	15	24,267	24.1	43	10.3	1,993	8.9
Kidney/urinary	112	3.7	2,811	2.8	124	4.1	3,081	3.1	12	10.7	270	9.6
Mental	35	1.1	1,180	1.2	37	1.2	1,255	1.2	2	5.7	75	6.4
Musculoskeletal	18	0.6	513	0.5	20	0.7	576	0.6	2	11.1	63	12.3
Neurological	116	3.8	2,636	2.6	124	4	2,863	2.8	8	6.9	227	8.6
Oral	0	0	16	0.0	0	0	16	0.0	0	0.0	0	0.0
Reproductive/maternal	2	0	92	0.1	2	0.1	98	0.1	0	0.0	6	6.5
Respiratory	191	6.3	4,480	4.5	197	6.4	4,632	4.6	6	3.1	152	3.4
Skin	6	0.2	160	0.2	7	0.2	183	0.2	1	16.7	23	14.4
Redistribution	257	8.4	7,945	7.9					. .	. .	. .	. .
All deaths	**3,054**	**100**	**100,663**	**100**	**3,054**	**100**	**100,663**	**100.0**	**. .**	**. .**	**. .**	**. .**

Table B6: YLL, by age at death used in ABDS 2011

Age at death	YLL	Age at death	YLL	Age at death	YLL	Age at death	YLL
0	86.02	27	59.43	54	33.32	81	10.32
1	85.21	28	58.44	55	32.38	82	9.65
2	84.22	29	57.45	56	31.47	83	8.98
3	83.23	30	56.46	57	30.55	84	8.31
4	82.24	31	55.48	58	29.64	85	7.64
5	81.25	32	54.49	59	28.73	86	7.12
6	80.25	33	53.50	60	27.81	87	6.61
7	79.26	34	52.52	61	26.91	88	6.09
8	78.26	35	51.53	62	26.00	89	5.57
9	77.27	36	50.56	63	25.10	90	5.05
10	76.27	37	49.58	64	24.20	91	4.70
11	75.28	38	48.60	65	23.29	92	4.35
12	74.28	39	47.62	66	22.42	93	4.00
13	73.29	40	46.64	67	21.55	94	3.66
14	72.29	41	45.67	68	20.68	95	3.31
15	71.29	42	44.71	69	19.80	96	3.09
16	70.30	43	43.74	70	18.93	97	2.88
17	69.32	44	42.77	71	18.10	98	2.66
18	68.33	45	41.80	72	17.28	99	2.44
19	67.34	46	40.85	73	16.45	100	2.23
20	66.35	47	39.90	74	15.62	101	2.11
21	65.36	48	38.95	75	14.80	102	1.99
22	64.37	49	38.00	76	14.04	103	1.87
23	63.38	50	37.05	77	13.27	104	1.75
24	62.39	51	36.12	78	12.51	105	1.63
25	61.40	52	35.19	79	11.75		
26	60.41	53	34.25	80	10.99		

Source: Murray et al. 2012

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Table B7: Expected years of life remaining at selected ages using GBD standard reference and Australian life tables, by sex, 2003 and 2011

	GBD 2010 standard	Australia 2011		Australia 2003		Indigenous 2011		
Age (years)	Persons	Males	Females	Males	Females	Age (years)	Males	Females
0	86.0	79.9	84.3	78.1	83.0	0	69.1	73.7
1	85.2	79.3	83.5	77.5	82.4	1–4	68.7	73.2
5	81.3	75.3	79.6	73.6	78.5	5–9	64.9	69.3
15	71.3	65.4	69.7	63.7	68.5	15–19	55.0	59.4
25	61.4	55.7	59.8	54.1	58.7	25–29	45.7	49.8
45	41.8	36.7	40.4	35.2	39.3	45–49	28.4	31.5
65	23.3	19.1	22.0	17.8	21.1	65–69	13.9	15.8
75	14.8	11.7	13.8	10.8	13.2	75–79	8.7	9.6
85	7.6	6.1	7.2	5.7	6.9	85 and over	4.2	4.4
95	3.3	3.1	3.4	3.1	3.6	. .	. .	. .
100	2.2	2.3	2.5	2.5	2.8	. .	. .	. .
105	1.6	. .	. .	. .	. .	. .	. .	. .

Notes

1. Australian life expectancy is calculated by the ABS using multiple years of mortality data: 2002–2004 for 2003 and 2010–2012 for 2011.
2. Australian (2003 and 2011) life expectancies for age 100 shown here are for all ages 100 or more.
3. Indigenous 2011 life expectancies are calculated by the ABS by age group; the youngest boundary has been selected to align with the single year age for the other life expectancies.

Sources: Murray et al. 2012; ABS 2012b; ABS 2005, ABS 2013c.

Appendix C: Additional information and tables for Chapter 4

Table C1: ABDS 2011 health states and disability weights

ABDS 2011 health state ID	Health state name	Disability weight
1	Infectious disease: acute episode, mild	0.006
2	Infectious disease: acute episode, moderate	0.051
3	Infectious disease: acute episode, severe	0.133
4	Infectious disease: post-acute consequences (fatigue, emotional lability, insomnia)	0.219
5	Diarrhoea: mild	0.074
6	Diarrhoea: moderate	0.188
7	Diarrhoea: severe	0.247
8	Epididymo-orchitis	0.128
9	Herpes zoster	0.058
10	HIV: symptomatic, pre-AIDS	0.274
11	HIV/AIDS: receiving antiretroviral treatment	0.078
12	AIDS: not receiving antiretroviral treatment	0.582
13	Intestinal nematode infections: symptomatic	0.027
14	Lymphatic filariasis: symptomatic	0.109
15	Ear pain	0.013
16	Tuberculosis: without HIV infection	0.333
17	Tuberculosis: with HIV infection	0.408
18	Cancer: diagnosis and primary therapy	0.288
19	Cancer: metastatic	0.451
20	Mastectomy	0.036
21	Stoma	0.095
22	Terminal phase: with medication (for cancers, end-stage kidney or liver disease)	0.540
23	Terminal phase: without medication (for cancers, end-stage kidney or liver disease)	0.569
24	Acute myocardial infarction: days 1–2	0.432
25	Acute myocardial infarction: days 3–28	0.074
26	Angina pectoris: mild	0.033
27	Angina pectoris: moderate	0.080
28	Angina pectoris: severe	0.167
29	Cardiac conduction disorders and cardiac dysrhythmias	0.224
30	Claudication	0.014
31	Heart failure: mild	0.041
32	Heart failure: moderate	0.072
33	Heart failure: severe	0.179
34	Stroke: long-term consequences, mild	0.019
35	Stroke: long-term consequences, moderate	0.070
36	Stroke: long-term consequences, moderate plus cognition problems	0.316
37	Stroke: long-term consequences, severe	0.552
38	Stroke: long-term consequences, severe plus cognition problems	0.588

(continued)

Table C1 (continued): ABDS 2011 health states and disability weights

ABDS 2011 health state ID	Health state name	Disability weight
39	Diabetic foot	0.020
40	Diabetic neuropathy	0.133
41	Chronic kidney disease (stage IV)	0.104
42	End-stage renal disease: with kidney transplant	0.024
43	End-stage renal disease: on dialysis	0.571
44	Decompensated cirrhosis of the liver	0.178
45	Gastric bleeding	0.325
46	Crohn's disease or ulcerative colitis	0.231
47	Benign prostatic hypertrophy: symptomatic	0.067
48	Urinary incontinence	0.139
49	Impotence	0.017
50	Infertility: primary	0.008
51	Infertility: secondary	0.005
52	Asthma: controlled	0.015
53	Asthma: partially controlled	0.036
54	Asthma: uncontrolled	0.133
55	Chronic obstructive pulmonary disease (COPD) and other chronic respiratory diseases: mild	0.019
56	COPD and other chronic respiratory diseases: moderate	0.225
57	COPD and other chronic respiratory diseases: severe	0.408
58	Dementia: mild	0.069
59	Dementia: moderate	0.377
60	Dementia: severe	0.449
61	Headache: migraine	0.441
62	Headache: tension-type	0.037
63	Multiple sclerosis: mild	0.183
64	Multiple sclerosis: moderate	0.463
65	Multiple sclerosis: severe	0.719
70	Parkinsons disease: mild	0.010
71	Parkinsons disease: moderate	0.267
72	Parkinsons disease: severe	0.575
73	Alcohol use disorder: mild	0.235
74	Alcohol use disorder: moderate	0.373
75	Alcohol use disorder: severe	0.570
76	Fetal alcohol syndrome: mild	0.016
77	Fetal alcohol syndrome: moderate	0.056
78	Fetal alcohol syndrome: severe	0.179
79	Cannabis dependence	0.266
80	Amphetamine dependence	0.486
81	Cocaine dependence	0.479
82	Heroin and other opioid dependence	0.697
83	Anxiety disorders: mild	0.030
84	Anxiety disorders: moderate	0.133
85	Anxiety disorders: severe	0.523
86	Major depressive disorder: mild episode	0.145

(continued)

Table C1 (continued): ABDS 2011 health states and disability weights

ABDS 2011 health state ID	Health state name	Disability weight
87	Major depressive disorder: moderate episode	0.396
88	Major depressive disorder: severe episode	0.658
89	Bipolar disorder: manic episode	0.492
90	Bipolar disorder: residual state	0.032
91	Schizophrenia: acute state	0.778
92	Schizophrenia: residual state	0.588
93	Anorexia nervosa	0.224
94	Bulimia nervosa	0.223
95	Attention deficit hyperactivity disorder	0.045
96	Conduct disorder	0.241
97	Asperger syndrome	0.104
98	Autism	0.262
99	Intellectual disability: mild	0.043
100	Intellectual disability: moderate	0.100
101	Intellectual disability: severe	0.160
102	Intellectual disability: profound	0.200
103	Hearing loss: mild	0.010
104	Hearing loss: moderate	0.027
105	Hearing loss: severe	0.158
106	Hearing loss: profound always	0.204
107	Hearing loss: complete	0.215
108	Hearing loss: mild, with ringing	0.021
109	Hearing loss: moderate, with ringing	0.074
110	Hearing loss: severe, with ringing	0.261
111	Hearing loss: profound, with ringing	0.277
112	Hearing loss: complete, with ringing	0.316
113	Distance vision: mild impairment	0.003
114	Distance vision: moderate impairment	0.031
115	Distance vision: severe impairment	0.184
116	Distance vision blindness	0.187
117	Near vision impairment	0.011
126	Musculoskeletal problems: legs, mild	0.023
127	Musculoskeletal problems: legs, moderate	0.079
128	Musculoskeletal problems: legs, severe	0.165
129	Musculoskeletal problems: arms, mild	0.028
130	Musculoskeletal problems: arms, moderate	0.117
131	Musculoskeletal problems: generalised, moderate	0.317
132	Musculoskeletal problems: generalised, severe	0.581
133	Gout: acute	0.295
134	Amputation of finger(s), excluding thumb: long term, with treatment	0.005
135	Amputation of thumb: long term	0.011
137	Amputation of both arms: long term, with treatment	0.123
138	Amputation of both arms: long term, without treatment	0.383
139	Amputation of toe	0.006
140	Amputation of one leg: long term, with treatment	0.039
141	Amputation of one leg: long term, without treatment	0.173

(continued)

Table C1 (continued): ABDS 2011 health states and disability weights

ABDS 2011 health state ID	Health state name	Disability weight
142	Amputation of both legs: long term, with treatment	0.088
143	Amputation of both legs: long term, without treatment	0.443
144	Burns of <20% total surface area without lower airway burns: short term, with or without treatment	0.141
145	Burns of <20% total surface area or <10% total surface area if head or neck, or hands or wrist involved: long term, with or without treatment	0.016
146	Burns of >=20% total surface area: short term, with or without treatment	0.314
147	Burns of >=20% total surface area or >=10% total surface area if head or neck, or hands or wrist involved: long term, with treatment	0.135
148	Burns of> =20% total surface area or >=10% total surface area if head or neck, or hands or wrist involved: long term, without treatment	0.455
149	Lower airway burns: with or without treatment	0.376
150	Crush injury: short or long term, with or without treatment	0.132
151	Dislocation of hip: long term, with or without treatment	0.016
152	Dislocation of knee: long term, with or without treatment	0.113
153	Dislocation of shoulder: long term, with or without treatment	0.062
154	Other injuries of muscle and tendon (includes sprains, strains, and dislocations other than shoulder, knee, or hip)	0.008
155	Drowning and non-fatal submersion: short or long term, with or without treatment	0.247
156	Fracture of clavicle, scapula, or humerus: short or long term, with or without treatment	0.035
157	Fracture of face bone: short or long term, with or without treatment	0.067
158	Fracture of foot bones: short term, with or without treatment	0.026
159	Fracture of foot bones: long term, without treatment	0.026
160	Fracture of hand: short term, with or without treatment	0.010
161	Fracture of hand: long term, without treatment	0.014
162	Fracture of neck of femur: short term, with or without treatment	0.258
163	Fracture of neck of femur: long term, with treatment	0.058
164	Fracture of neck of femur: long term, without treatment	0.402
165	Fracture other than neck of femur: short term, with or without treatment	0.111
166	Fracture other than neck of femur: long term, without treatment	0.042
167	Fracture of patella, tibia or fibula, or ankle: short term, with or without treatment	0.050
168	Fracture of patella, tibia or fibula, or ankle: long term, with or without treatment	0.055
169	Fracture of pelvis: short term	0.279
170	Fracture of pelvis: long term	0.182
171	Fracture of radius or ulna: short term, with or without treatment	0.028
172	Fracture of radius or ulna: long term, without treatment	0.043
173	Fracture of skull: short or long term, with or without treatment	0.071

(continued)

Table C1 (continued): ABDS 2011 health states and disability weights

ABDS 2011 health state ID	Health state name	Disability weight
174	Fracture of sternum or fracture of 1 or 2 ribs: short term, with or without treatment	0.103
175	Fracture of vertebral column: short or long term, with or without treatment	0.111
176	Fractures: treated, long term	0.005
177	Injured nerves: short term	0.100
178	Injured nerves: long term	0.113
179	Injury to eyes: short term	0.054
180	Severe traumatic brain injury: short term, with or without treatment	0.214
181	Traumatic brain injury: long-term consequences, minor, with or without treatment	0.094
182	Traumatic brain injury: long-term consequences, moderate, with or without treatment	0.231
183	Traumatic brain injury: long-term consequences, severe, with or without treatment	0.637
184	Open wound: short term, with or without treatment	0.006
185	Poisoning: short term, with or without treatment	0.163
186	Severe chest injury: long term, with or without treatment	0.047
187	Severe chest injury: short term, with or without treatment	0.369
188	Spinal cord lesion below neck: treated	0.296
189	Spinal cord lesion below neck: untreated	0.623
190	Spinal cord lesion at neck: treated	0.589
191	Spinal cord lesion at neck: untreated	0.732
192	Abdominopelvic problem: mild	0.011
193	Abdominopelvic problem: moderate	0.114
194	Abdominopelvic problem: severe	0.324
195	Anaemia: mild	0.004
196	Anaemia: moderate	0.052
197	Anaemia: severe	0.149
198	Periodontitis	0.007
199	Dental caries: symptomatic	0.010
200	Severe tooth loss	0.067
201	Disfigurement: level 1	0.011
202	Disfigurement: level 2	0.067
203	Disfigurement: level 3	0.405
204	Disfigurement: level 1 with itch or pain	0.027
205	Disfigurement: level 2, with itch or pain	0.188
206	Disfigurement: level 3, with itch or pain	0.576
207	Generic uncomplicated disease: worry and daily medication	0.049
208	Generic uncomplicated disease: anxiety about diagnosis	0.012
209	Iodine-deficiency goitre	0.199
210	Kwashiorkor	0.051
211	Severe wasting	0.128
212	Speech problems	0.051
213	Motor impairment: mild	0.010

(continued)

Table C1 (continued): ABDS 2011 health states and disability weights

ABDS 2011 health state ID	Health state name	Disability weight
214	Motor impairment: moderate	0.061
215	Motor impairment: severe	0.402
216	Motor plus cognitive impairments: mild	0.031
217	Motor plus cognitive impairments: moderate	0.203
218	Motor plus cognitive impairments: severe	0.542
219	Rectovaginal fistula	0.501
233	Low back pain, moderate	0.054
234	Low back pain, mild	0.020
235	Alcohol use disorder, very mild	0.123
236	Amphetamine dependence, mild	0.079
237	Amputation of 1 upper limb (long term, with treatment)	0.039
238	Amputation of 1 upper limb (long term, without treatment)	0.118
239	Back pain, most severe, with leg pain	0.384
240	Back pain, most severe, without leg pain	0.372
241	Back pain, severe, with leg pain	0.325
242	Back pain, severe, without leg pain	0.272
243	Borderline intellectual functioning	0.011
244	Cannabis dependence, mild	0.039
245	Cocaine dependence, mild	0.116
246	Concussion	0.110
247	Distance vision, monocular	0.017
248	Epilepsy, less severe (seizures less than once per month)	0.263
249	Epilepsy, severe (seizures once per month or more)	0.552
250	Headache, medication overuse	0.223
251	Heroin and other opioid dependence, mild	0.335
252	Hyperthyroidism	0.145
253	Hypothyroidism	0.019
254	Mild low back pain with leg pain	0.020
255	Moderate low back pain with leg pain	0.054
256	Neck pain, mild	0.053
257	Neck pain, moderate	0.114
258	Neck pain, severe	0.229
259	Neck pain, most severe	0.304
260	Stress incontinence	0.020
261	Thrombocytopenic purpura	0.159
262	Asymptomatic disease	0.000

Source: GBD 2013.

Table C2: ABDS 2011 main data sources for YLD estimation

Disease group	Key national data sources	Key Indigenous data sources
Blood and metabolic disorders	National Hospital Morbidity Database	National Hospital Morbidity Database
	Australian Health Survey 2011–12	Australian Aboriginal and Torres Strait Islander Health Survey 2012–13
	Australian Cystic Fibrosis Data Registry	
	Australian Bleeding Disorders Registry	
	Western Australian linked data	
Cancer and other neoplasms	Australian Cancer Database	Australian Cancer Database
	National Mortality Database	National Mortality Database
	National Hospital Morbidity Database	National Hospital Morbidity Database
	Medicare Benefits Schedule	Epidemiological studies
	Epidemiological studies	
Cardiovascular diseases	National Hospital Morbidity Database	National Hospital Morbidity Database
	Western Australian linked data	Western Australian linked data
	New Zealand Burden of Disease Study	New Zealand Burden of Disease Study
Endocrine disorders	National Diabetes Register	Australian Aboriginal and Torres Strait Islander Health Survey 2012–13
	Australian Health Survey 2011–12	Western Australian linked data
	Fremantle Diabetes Study	Fremantle Diabetes Study
Gastrointestinal disorders	National Hospital Morbidity Database	National Hospital Morbidity Database
	Epidemiological studies	Epidemiological studies
Infant and congenital conditions	National Hospital Morbidity Database	National Hospital Morbidity Database
	National Mortality Database	National Mortality Database
	National Perinatal Data Collection	National Perinatal Data Collection
	Western Australian Intellectual Disability Exploring Answers database	Indirect methods (rate ratios from Western Australian Intellectual Disability Exploring Answers database, Western Australian Register of Developmental Anomalies, Australian Cerebral Palsy Register)
	Western Australian Register of Developmental Anomalies	
	Australian Cerebral Palsy Register	

(continued)

Table C2 (continued): ABDS 2011 main data sources for YLD estimation

Disease group	Key national data sources	Key Indigenous data sources
Infectious diseases	National Notifiable Diseases Surveillance System	National Notifiable Diseases Surveillance System
	National Hospital Morbidity Database	National Hospital Morbidity Database
	Australian and New Zealand Assisted Reproductive Database	National HIV Register
	Bettering the Evaluation and Care of Health	Indirect methods (hospitalisation and notification rate ratios)
	Epidemiological studies	
	National HIV Register	
Injuries	National Hospital Morbidity Database	National Hospital Morbidity Database
	National Non-Admitted Patient Emergency Department Care Database	National Non-Admitted Patient Emergency Department Care Database
Hearing and vision disorders	Australian Health Survey 2011–12	Australian Aboriginal and Torres Strait Islander Health Survey 2012–13
	Australian Hearing Database	National Indigenous Eye Health Survey
	Blue Mountains Eye Study	Epidemiological studies
	Blue Mountains Hearing Study	
	Melbourne Vision Impairment Project	
	Epidemiological studies	
Kidney and urinary diseases	Australian and New Zealand Dialysis and Transplantation Registry	Australian and New Zealand Dialysis and Transplantation Registry
	National Hospital Morbidity Database	National Hospital Morbidity Database
	Australian Health Survey 2011–12	Australian Aboriginal and Torres Strait Islander Health Survey 2012–13
	Western Australian linked data	Western Australian linked data

(continued)

Table C2 (continued): ABDS 2011 main data sources for YLD estimation

Disease group	Key national data sources	Key Indigenous data sources
Mental and substance use disorders	National Survey of Mental Health and Wellbeing	Indirect methods based on rate ratios from Queensland mental health care data, hospitalisations data and the Western Australian Intellectual Disability Exploring Answers database
	Young Minds Matter survey	
	Western Australian Intellectual Disability Exploring Answers database	
	The Australian National Survey of High Impact Psychosis	
Musculoskeletal conditions	Australian Health Survey 2011–12	Australian Aboriginal and Torres Strait Islander Health Survey 2012–13
Neurological conditions	National Hospital Morbidity Database	National Hospital Morbidity Database
	Australian Health Survey 2011–12	Australian Aboriginal and Torres Strait Islander Health Survey 2012–13
	AIHW dementia analyses	Epidemiological studies
	Epidemiological studies	Indirect methods (using Western Australian linked data and hospital data)
	Western Australian linked data	
Oral disorders	National Survey of Adult Oral Health	National Survey of Adult Oral Health
	National Dental Telephone Interview Survey	Australian Aboriginal and Torres Strait Islander Health Survey 2012–13
	Child Dental Health Survey	Child Dental Health Survey
Reproductive and maternal conditions	National Hospital Morbidity Database	National Hospital Morbidity Database
	Australian and New Zealand Assisted Reproductive Database	Australian and New Zealand Assisted Reproductive Database
	Australian Longitudinal Study on Women's Health	Indirect methods (rate ratios from hospitalisations data)

(continued)

Table C2 (continued): ABDS 2011 main data sources for YLD estimation

Disease group	Key national data sources	Key Indigenous data sources
Respiratory diseases	National Mortality Database	National Mortality Database
	National Hospital Morbidity Database	National Hospital Morbidity Database
	Australian Health Survey	Australian Aboriginal and Torres Strait Islander Health Survey 2012–13
	Burden of Obstructive Lung Disease Initiative	Burden of Obstructive Lung Disease Initiative
	Western Australian linked data	National Hospital Morbidity Database
	National Hospital Morbidity Database	Epidemiological studies
	Epidemiological studies	
Skin disorders	Australian Health Survey 2011–12	Australian Aboriginal and Torres Strait Islander Health Survey 2012–13
	National Hospital Morbidity Database	National Hospital Morbidity Database
	Epidemiological studies	Epidemiological studies

Table C3: Hospitalisation adjustment factors used for 2011 Indigenous YLD estimates

State/territory	Remoteness area	Adjustment factor
NSW	Major cities	1.37
	Inner regional	1.09
	Outer regional	1.08
	Remote/Very remote	1.02
Vic	Major cities	1.41
	Inner regional	1.06
	Outer regional	1.09
Qld	Major cities	1.17
	Inner regional	1.12
	Outer regional	1.04
	Remote/Very remote	0.97
WA	Major cities	0.99
	Inner regional	1.02
	Outer regional	1.00
	Remote	1.07
	Very remote	1.00
SA	Major cities	1.16
	Inner regional/Outer regional	1.03
	Remote/Very remote	1.00
Tas	Inner regional	1.37
ACT	Major cities	1.69
NT	Outer regional	1.03
	Remote	0.99
	Very remote	1.00
Total		**1.09**

Source: AIHW 2013.

Table C4: Hospitalisation adjustment factors used for 2003 Indigenous YLD estimates

Remoteness area	Adjustment factor
Major cities	1.25
Inner regional	1.11
Outer regional	1.06
Remote/Very remote	1.03
Total	**1.12**

Source: AIHW 2010.

Table C5: Diseases for which Indigenous prevalence estimates for 2011 were derived using indirect methods

Disease[a]	Data source and indirect method
Mental and substance use disorders	
Depressive disorders	Major depressive disorder: Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
	Dysthymia: sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
Anxiety disorders	Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
Bipolar affective disorder	Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
Alcohol use disorders	Asymptomatic/very mild/mild: Age- and sex-specific hospitalisation rate ratios (Indigenous to national)
	Moderate/severe: Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
Drug use disorders	Cannabis dependence: Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
	Amphetamine dependence and opioid dependence: Sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
	Cocaine dependence: sex-specific rate ratios (Indigenous non-Indigenous) from National Drug Strategy survey data
	Other drug dependence: sex-specific hospitalisation rate ratios (Indigenous to national)
Schizophrenia	Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
Attention deficit hyperactivity disorder	Average of age-specific rate ratios (Indigenous to non-Indigenous) based on Longitudinal Study of Indigenous Children and Queensland linked mental health care data
Conduct disorder	Average of age-specific rate ratios (Indigenous to non-Indigenous) based on Longitudinal Study of Indigenous Children and Queensland linked mental health care data
Intellectual disability	Age-specific rate ratios (Indigenous to non-Indigenous) from IDEA database
Cancer and other neoplasms	
Non-melanoma skin cancer[b]	Diagnosis and primary therapy of simple non-melanoma skin cancer: Applied Indigenous-to-national ratio of complex non-melanoma skin cancer
Ductal carcinoma in situ[b]	Mastectomy due to ductal carcinoma in situ: ratio of Indigenous to national diagnosed breast cancer of less than 2 centimetres applied to national ductal carcinoma in situ incidence
Cardiovascular diseases	
Peripheral vascular disease	New Zealand Maori rates applied to the Indigenous population
Atrial fibrillation and flutter	Applied the New Zealand Maori rates to the Indigenous population
Infectious diseases	
Syphilis	Age- and sex-specific hospital separation and notification rate ratios (Indigenous to national)
Other sexually transmitted infections[a]	Genital herpes: Rate ratio of Indigenous to national herpes simplex virus II sero-prevalence.
Hepatitis B (acute)	Age- and sex-specific notification rate ratios (Indigenous to national)
Hepatitis C (acute)	Age- and sex-specific notification rate ratios (Indigenous to national)
Upper respiratory tract infections	Age- and sex-specific hospital separation rate ratios (Indigenous to national)
Otitis media	Age- and sex-specific rate ratios (Indigenous to national) of self-reported diseases of the ear and mastoid process (AHS 2011–13)
Lower respiratory tract infections	Age- and sex-specific hospital separation rate ratios (Indigenous to national)
Influenza	Age- and sex-specific hospital separation rate ratios (Indigenous to national)

(continued)

Table C5 (continued): Diseases for which Indigenous prevalence estimates for 2011 were derived using indirect methods

Disease[(a)]	Data source and indirect method
Infectious diseases (continued)	
Varicella-zoster	Age- and sex-specific chickenpox and shingles notification rate ratios (Indigenous to national)
Campylobacteriosis	Age- and sex-specific hospital separation rate ratios (Indigenous to national)
Salmonellosis	Age- and sex-specific hospital separation rate ratios (Indigenous to national)
Rotavirus	Age- and sex-specific hospital separation rate ratios (Indigenous to national)
Reproductive and maternal conditions	
Early pregnancy loss	Indigenous-to-national age-specific rate ratios from hospital separations for medical abortions applied to national rate of Medicare data, in addition to adjusted hospitalisations data
Genital prolapse	Indigenous-to-national rate ratios from hospital separations for genital prolapse applied to national rate
Infant and congenital conditions	
Pre-term birth and low birthweight complications	Age-specific rate ratios (Indigenous to non-Indigenous) from IDEA database
Birth trauma and asphyxia	Age-specific rate ratios (Indigenous to non-Indigenous) from IDEA database
Cerebral palsy	Sex-specific rate ratios (Indigenous to non-Indigenous) from the Australian Cerebral Palsy Register, age distribution obtained from national estimates
Neural tube defects	Applied Indigenous birth prevalence rate obtained from WARDA to national estimates
Cardiovascular defects	Applied Indigenous birth prevalence rate obtained from WARDA to national estimates
Cleft lip and/or palate	Applied Indigenous birth prevalence rate obtained from WARDA to national estimates
Gastrointestinal malformations	Applied Indigenous birth prevalence rate obtained from WARDA to national estimates
Urogenital malformations	Applied Indigenous birth prevalence rate obtained from WARDA to national estimates
Down syndrome	Age-specific rate ratios (Indigenous to non-Indigenous) from IDEA database
Brain malformations	Age-specific rate ratios (Indigenous to non-Indigenous) from IDEA database
Oral disorders	
Dental caries and pulpitis	Indigenous-to-national rate ratios from National Survey of Adult Oral Health 2004–06 (15 and over) and Child Dental Health Survey 2009 (less than 15) were applied to national age and sex distributions
Periodontal disease	Indigenous-to-national rate ratios from National Survey of Adult Oral Health 2004–06 and Child Dental Health Survey 2009 were applied to national age and sex distributions
Skin disorders	
Ulcers	Other chronic skin ulcers : hospital rate ratio used to determine prevalence start point, then applied national pattern of prevalence by age Pressure ulcers (skin): hospital rate ratios applied to total population prevalence, by age and sex

(a) Excludes residual ('other') diseases within each disease group, which also used indirect methods such as hospitalisation rate ratios in many instances (6 in total).

(b) Applicable to listed sequelae only.

Table C6: Diseases for which national rates or ratios were assumed to derive Indigenous prevalence estimates for 2011

Disease	Data source and indirect method
Cancer and other neoplasms	
Breast cancer[(a)]	Mastectomy due to breast cancer: national incidence-to-hazard ratio applied for males only
Prostate cancer[(a)]	Impotence/incontinence due to prostate cancer: national rates of treatments and outcomes for prostate cancer applied to the Indigenous 10-year prevalence of prostate cancer
Laryngeal cancer[(a)]	Laryngectomy due to laryngeal cancer: national sex-specific laryngectomy incidence hazard rates applied to the Indigenous 10-year prevalence
Bowel cancer[(a)]	Stoma due to bowel cancer: due to the small number of cases and hospitalisations, Indigenous incidence was assumed to be the same as for the national population
Bladder cancer[(a)]	Stoma/urinary incontinence due to bladder cancer: insufficient data to produce Indigenous-specific rates for the various urinary diversions, so national rates were assumed. The proportion of people experiencing incontinence due to various diversion types was assumed to be the same for the Indigenous population as the national
Brain and central nervous system cancer[(a)]	Brain injury due to brain cancer: national rates assumed
Ductal carcinoma in situ[(a)]	Mastectomy due to ductal carcinoma in situ: ratio of Indigenous to national diagnosed breast cancer less than 2cm applied to national ductal carcinoma in situ incidence
Benign and uncertain brain tumours[(a)]	Brain injury due to benign and uncertain brain tumours: national rates assumed
Infectious diseases	
Other sexually transmitted infections[(a)]	Genital warts: assumed no difference in Indigenous prevalence (based on analysis of BEACH, NHMD and the results of 2 epidemiological studies of human papillomavirus)
Mental and substance use disorders	
Eating disorders	Assumed same prevalence rate as national
Autism spectrum disorders	Assumed same prevalence rate as national
Gastrointestinal disorders	
Gastroduodenal disorders	Assumed same prevalence rate and inflation factor as national
Inflammatory bowel disease	Assumed same prevalence rate as national
Gastro-oesophageal reflux disease	Assumed same prevalence rate as national
Functional gastrointestinal disorders	Assumed same prevalence rate as national
Neurological conditions	
Parkinsons disease	National prevalence rates and severity distribution were applied to the Indigenous population
Multiple sclerosis	The national prevalence: separation ratio was applied to the count of Indigenous multiple sclerosis hospital separations. The severity distribution used for Indigenous is the same as used for national estimates
Guillain-Barré syndrome	The national persons-to-separation ratio was applied to the count of Indigenous Guillain-Barré syndrome hospital separations
Skin disorders	
Acne	Assumed same prevalence rate as national
Dermatitis and eczema	Assumed same prevalence rate as national
Reproductive and maternal conditions	
Endometriosis	Assumed same prevalence as total Australian population for endometriosis. Adjusted hospital separations used for severe endometriosis and subtracted from total endometriosis estimates to inform mild estimates
Infertility	Assumed same prevalence rate as national (including all sequelae of the infertility envelope)

(a) Applicable to listed sequelae only.

Appendix D: Additional information and tables for Chapter 5

Cancer and other neoplasms

Table D1: Redistribution proportions of other and ill-defined digestive organs (C26), by age and sex

	Males				Females			
Disease	**0–44**	**45–64**	**65–84**	**85+**	**0–44**	**45–64**	**65–84**	**85+**
Bowel cancer	0.750	0.868	0.860	0.800	1.000	0.862	0.838	0.774
Stomach cancer	0.125	0.017	0.051	0.038	0.000	0.011	0.022	0.019
Pancreatic cancer	0.000	0.000	0.017	0.013	0.000	0.023	0.013	0.026
Liver cancer	0.000	0.017	0.003	0.000	0.000	0.000	0.004	0.000
Bladder cancer	0.000	0.000	0.003	0.013	0.000	0.000	0.000	0.000
Lung cancer	0.000	0.000	0.010	0.000	0.000	0.000	0.013	0.019
Oesophageal cancer	0.000	0.008	0.007	0.000	0.000	0.000	0.009	0.000
Breast cancer	0.000	0.000	0.000	0.000	0.000	0.000	0.013	0.013
Ovarian cancer	0.000	0.000	0.000	0.000	0.000	0.000	0.004	0.006
Prostate cancer	0.000	0.000	0.007	0.013	0.000	0.000	0.000	0.000
Unknown primary[(a)]	0.125	0.050	0.024	0.100	0.000	0.057	0.035	0.077
Other malignant neoplasms (cancers)	0.000	0.041	0.017	0.025	0.000	0.046	0.048	0.065

(a) Unknown primary (C80) will require further redistribution.

Sources: Pooled Western Australia Cancer Registry data, 2007–2011; South Australia Cancer Registry data, 2007–2011.

Table D2: Redistribution proportions of ill-defined cancers (C39, C76–C80, C97), by age and sex

	Males				Females			
ABDS 2011 disease	**0–44**	**45–64**	**65–84**	**85+**	**0–44**	**45–64**	**65–84**	**85+**
Unknown primary[(a)]	0.364	0.667	0.604	0.691	0.619	0.695	0.676	0.747
Other malignant neoplasms (cancers)	0.318	0.039	0.051	0.026	0.095	0.061	0.059	0.024
Lung cancer	0.000	0.121	0.094	0.037	0.000	0.053	0.080	0.028
Bowel cancer	0.091	0.014	0.033	0.042	0.048	0.015	0.047	0.059
Mouth and pharyngeal cancer	0.045	0.058	0.038	0.021	0.048	0.008	0.002	0.021
Bladder cancer	0.000	0.034	0.028	0.037	0.048	0.031	0.016	0.007
Pancreatic cancer	0.045	0.014	0.015	0.005	0.048	0.015	0.006	0.003
Non-melanoma skin cancers	0.000	0.029	0.049	0.058	0.000	0.000	0.008	0.035
Gallbladder cancer	0.000	0.000	0.005	0.010	0.048	0.015	0.016	0.007
Melanoma of the skin	0.000	0.005	0.015	0.021	0.048	0.000	0.004	0.014
Breast cancer	0.000	0.000	0.000	0.000	0.000	0.038	0.027	0.014
Kidney cancer	0.045	0.005	0.011	0.000	0.000	0.000	0.002	0.007
Brain and central nervous system cancer	0.045	0.000	0.000	0.005	0.000	0.008	0.002	0.000
Testicular cancer	0.045	0.005	0.003	0.000	0.000	0.000	0.000	0.000
Prostate cancer	0.000	0.005	0.016	0.026	0.000	0.000	0.000	0.000
Liver cancer	0.000	0.000	0.005	0.010	0.000	0.015	0.008	0.003
Ovarian cancer	0.000	0.000	0.000	0.000	0.000	0.015	0.023	0.007
Stomach cancer	0.000	0.000	0.013	0.005	0.000	0.008	0.004	0.010
Oesophageal cancer	0.000	0.005	0.008	0.005	0.000	0.000	0.006	0.000
Leukaemia	0.000	0.000	0.003	0.000	0.000	0.015	0.002	0.000
Uterine cancer	0.000	0.000	0.000	0.000	0.000	0.008	0.002	0.007
Non-Hodgkin lymphoma	0.000	0.000	0.003	0.000	0.000	0.000	0.006	0.003
Laryngeal cancer	0.000	0.000	0.005	0.000	0.000	0.000	0.002	0.003

(a) Assumes that deaths coded to C80 by cancer registries are valid clinical classifications of unknown primary, rather than classified due to insufficient information.

Sources: Pooled Western Australia Cancer Registry data, 2007–2011 and South Australia Cancer Registry data, 2007–2011.

Table D3: Average sequelae duration for cancer and other neoplasms

	Sequelae duration (months)			
Cancer type	Diagnosis and primary therapy	Controlled phase	Metastatic phase	Terminal phase
Mouth and pharyngeal cancers[1]	3.0	Remainder of year	9.2	1.0
Laryngeal cancer[1]	3.0	Remainder of year	9.8	1.0
Oesophageal cancer[1]	2.0	Remainder of year	4.2	1.0
Stomach cancer[1]	6.0	Remainder of year	2.7	1.0
Bowel cancer[1]	9.0	Remainder of year	5.0	1.0
Liver cancer[1]	2.0	Remainder of year	1.8	1.0
Gallbladder cancer[1]	2.0	Remainder of year	2.5	1.0
Pancreatic cancer[1]	1.0	Remainder of year	3.1	1.0
Lung cancer[1]	6.0	Remainder of year	5.0	1.0
Mesothelioma[a]	6.0	Remainder of year	5.0	1.0
Melanoma (<=1.00mm)[3]	0.5	Remainder of year	6.7	1.0
Melanoma (1.01–2.00mm)[3]	0.9	Remainder of year	6.7	1.0
Melanoma (2.01–4.00mm)[3]	1.2	Remainder of year	6.7	1.0
Melanoma (>4.00mm)[3]	1.7	Remainder of year	6.7	1.0
Non-melanoma skin cancer[b]	0.5	0	6.7	1.0
Breast cancer (males)[2]	6.0	Remainder of year	10.8	1.0
Breast cancer (females)[2] (<20mm)	3.4	Remainder of year	10.8	1.0
Breast cancer (females) (20–50mm)[2]	6.8	Remainder of year	10.8	1.0
Breast cancer (females) (>50mm)[2]	8.0	Remainder of year	10.8	1.0
Breast cancer (females) (unknown size)[2]	6.0	Remainder of year	10.8	1.0
Cervical cancer[1]	3.0	Remainder of year	8.2	1.0
Uterine cancer[1]	3.0	Remainder of year	9.0	1.0
Ovarian cancer[1]	3.0	Remainder of year	10.3	1.0
Prostate cancer[1]	2.0	Remainder of year	12.1	1.0
Testicular cancer[1]	3.0	Remainder of year	9.2	1.0
Bladder cancer[1]	1.5	Remainder of year	5.6	1.0
Kidney cancer[1]	2.0	Remainder of year	6.0	1.0
Brain and central nervous system cancer[1]	3.0	Remainder of year	19.0	1.0
Thyroid cancer[1]	2.0	Remainder of year	6.4	1.0
Non-Hodgkin lymphoma[1]	4.0	Remainder of year	7.6	1.0
Hodgkin lymphoma[1]	4.0	Remainder of year	7.5	1.0
Leukaemia[1]	8.0	Remainder of year	7.1	1.0
Myeloma[1]	9.0	Remainder of year	10.6	1.0
Other lymphohaematopoietic (blood) cancers[c]	9.0	Remainder of year	10.6	1.0
Unknown primary cancer[d]	6.0	0	Remainder of year	1.0
Other malignant neoplasms[1]	2.0	Remainder of year	7.5	1.0

(continued)

Table D3 (continued): Average sequela duration for cancer and other neoplasms

Cancer type	Sequelae duration (months)			
	Diagnosis and primary therapy	Controlled phase	Metastatic phase	Terminal phase
Brain tumours (benign and uncertain)[e]	3.0	Remainder of year	. .	1.0
Breast ductal carcinoma in situ[f]	3.4	Remainder of year	. .	. .

(a) All phases assumed to be the same as lung cancer.

(b) All phases assumed to be the same as melanoma.

(c) All phases assumed to be the same as myeloma.

(d) Diagnosis and primary therapy phase assumed to be the same as lung cancer. Duration of metastatic phase assumed to be the remainder of the year.

(e) Diagnosis and primary therapy phase and terminal phase assumed to be the same as brain cancer.

(f) Diagnosis and primary therapy phase assumed to be the same as small (<20mm) breast cancer.

Sources

(1) GBD course notes 2013, Breast cancer case study: Naghavi.

(2) Expert opinion from Dr Catherine Shannon, Senior Medical Oncologist, Mater Cancer Care Centre, and Professor Christobel Saunders, School of Surgery, University of Western Australia.

(3) Melanoma Management Guide for GPs and Melanoma Institute Australia.

Hearing and vision disorders

Table D4: Proportion of tinnitus in hearing impaired population, by age, sex and severity level

	Proportion of tinnitus within each severity level of hearing loss (%)			
Age group (years)	**Mild**	**Moderate**	**Severe**	**Profound**
Males				
18–24	38.9	29.2	54.1	0.0
25–44	21.8	35.2	35.3	37.2
45–64	29.9	29.7	39.4	42.6
65 and over	16.5	21.9	26.7	26.4
Females				
18–24	18.8	47.0	73.3	0.0
25–44	35.6	40.4	54.8	45.3
45–64	29.8	34.2	47.2	32.2
65 and over	21.7	28.9	33.6	30.1

Table D5: Proportions of vision loss, by disease, age and severity level

	Severity level of vision loss (%)			
Age group (years)	**Mild**	**Moderate**	**Severe**	**Blindness**
Refractive error				
0–49	68.7	31.3	0.0	0.0
50–59	100.0	0.0	0.0	0.0
60–69	74.9	16.7	8.4	0.0
70–79	80.0	15.0	5.0	0.0
80–89	60.1	29.1	10.9	0.0
90 and over	29.0	41.9	29.0	0.0
Cataract and other lens disorders				
70–79	50.0	25.0	0.0	25.0
80–89	69.0	13.8	3.4	13.8
90 and over	72.0	12.0	4.0	12.0
Average	70.7	12.8	3.7	12.8
Glaucoma				
60–69	20.0	25.0	50.0	5.0
70–79	0.0	16.7	33.3	50.0
80–89	0.0	93.33	3.3	3.3
90 and over	0.0	33.3	0.0	66.7

Infant and congenital conditions

Table D6: Severity distribution used for cerebral palsy, by Gross Motor Function Classification System level

GMFCS levels	Description	GBD health state	%
Level I	Walks without limitations	Motor impairment: mild	38.5
Level II	Walks with limitations, including long distances, balancing, run or jumping; require use of mobility devices when first learning to walk, and may rely on wheeled mobility equipment when outside of home for traveling long distances.	Motor impairment: moderate	24.5
Level III	Walks with adaptive equipment assistance. Requires mobility assistance to walk indoors, while utilizing wheeled mobility outdoors; can sit on own or with limited external support; and has some independence in standing transfers.	Motor impairment: moderate	10.7
Level IV	Self-mobility with use of powered mobility assistance. Is supported when sitting; self-mobility is limited; and likely to be transported in wheelchair.	Motor impairment: severe	12.2
Level V	Severe head and trunk control limitations. Requires extensive use of assisted technology and physical assistance; and transported in a wheelchair.	Motor impairment: severe	14.1

Source: Cerebral Palsy Alliance 2013.

Table D7: Distribution of health states for neural tube defects

Health state	Proportion of neural tube defects cases (%)
Incontinence	80.0
Mild motor impairment	30.0
Moderate motor impairment	27.0
Severe impairment	
Motor impairment only	21.5
Motor plus cognitive impairment	21.5

Source: Hunt & Oakeshott 2003.

Infectious diseases

Table D8: Sequelae, health states and durations for infectious diseases

Disease	Sequela	ABDS 2011 health state identifier	Duration (short-term sequelae)
HIV/AIDS	HIV/AIDS	10, 11, 12, 208	. .
Tuberculosis	Tuberculosis	16	8 months
Syphilis	Congenital syphilis	3	2–5 weeks
	Primary syphilis	1	. .
	Secondary syphilis	2	. .
	Tertiary syphilis	217	. .
Chlamydia	Chlamydial infection	1	1–2 weeks
	Infertility due to chlamydia(a)	50, 51	. .
	Pelvic inflammatory disease due to chlamydia	193, 194	. .
Gonorrhoea	Gonococcal infection	1	1–2 weeks
	Infertility due to gonorrhoea[(a)]	50, 51	. .
	Pelvic inflammatory disease due to gonorrhoea	193, 194	. .
Other sexually transmitted infections	Infertility due to other sexually transmitted infections[(a)]	50, 51	5 days (per episode)
	Other sexually transmitted infections	1	. .
	Pelvic inflammatory disease due to other sexually transmitted infections	193, 194	. .
Hepatitis A	Acute hepatitis A	1, 2, 3	1 week (children) 3 weeks (adults)
	Hepatitis A, relapsing	4	4 months
Hepatitis B (acute)	Acute hepatitis B	2, 3	4–6 weeks
Hepatitis C (acute)	Acute hepatitis C	2, 3	4–6 weeks
Upper respiratory infections	Upper respiratory infections	1, 2	5 days
Otitis media	Otitis media: acute	15	1 week (per episode)
	Otitis media: chronic	103	3 months
Lower respiratory infections	Lower respiratory infections	2, 3	1–3 weeks
Influenza	Influenza	2, 3	2 weeks
Diphtheria	Diphtheria	2, 3	2 weeks
Pertussis	Pertussis, acute	1, 2, 3	7 weeks
Tetanus	Tetanus	3	2 weeks
Measles	Measles	2, 3	2–3 weeks
Rubella	Rubella	1	1 week
Varicella-zoster	Varicella-zoster	1, 4, 9	Chickenpox (children): 1 week Chickenpox(adults): 10 days Shingles: 2 weeks Post-herpetic neuralgia: 3 months

(continued)

Table D8 (continued): Sequelae, health states and durations for infectious diseases

Disease	Sequela	ABDS 2011 health state identifier	Duration (short-term sequelae)
Haemophilus influenzae type b	Haemophilus influenza type b disease	3	4 weeks
Pneumococcal disease	Invasive pneumococcal disease	3	2–4 weeks
Meningococcal disease	Meningococcal disease	3	4 weeks
Other meningitis and encephalitis	Other meningitis and encephalitis	3	2–4 weeks
Dengue	Dengue fever	1, 2, 3, 4	1–2 weeks (acute), 2 months (post-acute consequences)
Ross River virus	Ross River virus infection	131, 4	4 weeks , 3 months (prolonged cases)
Barmah Forest virus	Barmah Forest virus infection	131, 26	3 weeks, 2 months (prolonged cases)
Malaria	Malaria	2, 3	1–2 weeks
Trachoma	Blindness due to trachoma[(b)]	115, 116	. .
	Low vision due to trachoma[(b)]	113, 114	. .
Campylobacteriosis	Gastrointestinal infection	5, 6, 7	3–14 days
Salmonellosis	Gastrointestinal infection	5, 6, 7	6–16 days
Rotavirus	Gastrointestinal infection	5, 6, 7	5–8 days
Other gastrointestinal infections	Gastrointestinal infection	43, 5, 6, 7	2–7 days
Other infections	Other infections, moderate	2	. .
	Other infections, severe	3	. .

(a) Part of infertility envelope.

(b) Part of vision envelope.

Injuries

Table D9: Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and parameters for long-term injuries

Injury	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years)	Proportion with long-term consequences (%)	Proportion entering remission in year (%)	Mortality risk ratio
Traumatic brain injury	Traumatic brain injury, short term, minor	246	S06.00, S06.02	0.067	. .	. .	. .
	Traumatic brain injury, short term, moderate–severe	180	S06 (excluding S06.00, S06.02)	0.067	. .	. .	. .
	Traumatic brain injury, long term, minor	181	S06.00, S06.02	. .	10	1	2.50
	Traumatic brain injury, long term, moderate	182	S06 (excluding S06.00, S06.02)	. .	67	1	2.50
	Traumatic brain injury, long term, severe	183	S06 (excluding S06.00, S06.02)	. .	100	1	2.50
	Skull fracture, short term	173	S02.0, S02.1, S02.7, S02.9	0.107			
	Skull fracture, long term	173	S02.0, S02.1, S02.7, S02.9	. .	15	1	1.10
Spinal cord injury	Below neck	188	S24.0, S24.1, S24.7, S34.0, S34.1, S34.7, T06.1, T09.3	. .	100	1	3.00
	At neck	190	S14.0, S14.1, S14.7, T06.0	. .	100	1	3.00
Internal and crush injury	Crush injury	150	S07, S17, S18, S47, S38.0, S57, S67, S77, S87, S97, T04, T14.7	0.094	. .	. .	. .
	Severe chest injury	187	S22.4, S22.5, S25, S26, S27, S28, S29.7	0.042	. .	. .	. .
	Abdominal /pelvic injuries	187	S35–S37, S38.1, S39.6, S39.7, T06.5	0.042	. .	. .	. .
Poisoning	Poisoning, short term	185	T36-T65	0.008	. .	. .	. .
	Poisoning, long term	216	T36-T65	. .	5	1	2.00

(continued)

Table D9 (continued): Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and parameters for long-term injuries

Injury	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years)	Proportion with long-term consequences (%)	Proportion entering remission in year (%)	Mortality risk ratio
Drowning and submersion injuries	Drowning and non-fatal submersion, short term	155	T751	0.019			
	Drowning and non-fatal submersion, long term	155	T751	. .	15	1	2.00
Hip fracture	Neck of femur, short term	162	S72.0, S72.1, S72.2	0.140	. .	. .	. .
	Neck of femur, long term	163	S72.0, S72.1, S72.2	. .	10	0	1.50
	Other than neck of femur, short term	165	S72.3–S72.9, S79	0.140	. .	. .	. .
	Other than neck of femur, long term	176	S72.3–S72.9, S79	. .	10	0	1.50
Tibia and ankle fracture	Tibia or fibula fracture	167	S82.1, S82.2, S82.3, S82.7, S82.4, S82.8, S82.9	0.090	. .	. .	. .
	Ankle fracture	167	S82.5, S82.6	0.096	. .	. .	. .
Humerus fracture	Humerus fracture	156	S42.2, S42.3, S42.4, S42.7	0.112	. .	. .	. .
Other fractures	Patella	167	S82.0	0.090	. .	. .	. .
	Calvicle or scapula	156	S22.8, S22.9, S42.0, S42.1, S42.8, S42.9, S49.7	0.112	. .	. .	. .
	Face bone, short term	157	S02.2–S02.6, S02.8	0.118	. .	. .	. .
	Face bone, long term	157	S02.2–S02.6, S02.8	. .	15	1	1.10
	Foot bone	158	S92	0.073	. .	. .	. .
	Hand bone	160	S62.0–S62.4, S62.5–S62.7, S62.8, S69.7	0.070	. .	. .	. .
	Pelvis	169	S32.1, S32.3–S32.8, T02.1	0.126	. .	. .	. .
	Coccyx	184	S32.2	0.126	. .	. .	. .
	Radius or ulna	171	S52, S59.7, T10	0.112	. .	. .	. .

(continued)

Table D9 (continued): Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and parameters for long-term injuries

Injury	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years)	Proportion with long-term consequences (%)	Proportion entering remission in year (%)	Mortality risk ratio
Other fractures (continued)	Sternum or 1 or 2 ribs	174	S22.2, S22.3	0.115	. .	. .	. .
	Vertebral column	175	S12, S22.0–S22.1, S32.0, T08	0.140	. .	. .	. .
	Other	176	T02.0, T02.2–T02.9, T12, T14.2	0.070	. .	. .	. .
Dislocations	Shoulder joint	153	S43.0–S43.3	0.035	. .	. .	. .
	Shoulder other than joint	184	S53.0–S53.1	0.019	. .	. .	. .
	Hip	151	S73.0	0.034	. .	. .	. .
	Knee	152	S83.0, S83.1	0.034	. .	. .	. .
	Other	154	S03.0–S03.3, S13.1–S13.3, S23.1, S23.2, S33.1–S33.3, S63.0– S63.2, S93.0, S93.1, S93.3, T03, T09.2, T11.2, T13.2, T14.3	0.019	. .	. .	. .
Soft tissue injuries	Soft tissue injuries	154	S03.4, S03.5, S13.4–S13.6, S16, S23.0, S23.3–S23.5, S29.0, S33.5–S33.7, S39.0, S46, S53.2–S53.4, S56, S63.3–S63.7, S66, S73.1, S76, S83.2–S83.7, S86.0, S86.1–S86.9, S93.2, S93.4–S93.6, S96, T06.4, T09.5, T11.5, T13.5, T14.6,	0.038	. .	. .	. .
Burn injuries	Non-airway burn, short term, minor	144	(T31.0 or T31.1) and (T21, T22, T24, T25, T28, T29, T30)	0.083	. .	. .	. .
	Non-airway burn, long term, minor	145	[(T31.0 or T31.1) and (T21, T22, T24, T25, T28, T29, T30)] or [(T31.0) and (T20, T23 or T26)]	. .	10	0	1.01
	Non-airway burn, short term, severe	146	(T31.2 –T31.9) and (T21, T22, T24, T25, T28, T29, T30)	0.279	. .	. .	. .

(continued)

Table D9 (continued): Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and parameters for long-term injuries

Injury	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years)	Proportion with long-term consequences (%)	Proportion entering remission in year (%)	Mortality risk ratio
Burn injuries (continued)	Non-airway burn, long term, severe	147	[(T31.2–T31.9) and (T21, T22, T24, T25, T28, T29, T30)] or [(T31.1–T31.9) and (T20, T23 or T26)]	. .	100	0	1.10
	Airways burn	149	T27	0.279	. .	. .	. .
Other injuries	Amputation of finger(s), excluding thumb	134	S68.1, S68.2	. .	100	0	1.02
	Amputation of thumb	135	S68.0	. .	100	0	1.05
	Amputation of 1 arm	237	S48, S58, S68.3, S68.4, S68.8, S68.9, T11.6	. .	100	0	1.10
	Amputation of both arms	137	T05.0–T05.2	. .	100	0	1.10
	Amputation of toe	139	S98.1, S98.2	. .	100	0	1.05
	Amputation of 1 leg	140	S78, S88, S98.0, S98.3, S98.4, T13.6, T05.6	. .	100	0	1.20
	Amputation of both legs	142	T05.3–T05.5	. .	100	0	1.20
	Injured nerves, short term	177	S04, S14.2–S14.6, S24.2–S24.6, S34.2–S34.6, S34.8, S44, S54, S64, S74, S84, S94, T06.2, T11.3, T13.3, T14.4, T09.4	0.048	. .	. .	. .
	Injured nerves, long term	178	S04, S14.2–S14.6, S24.2–S24.6, S34.2–S34.6, S34.8, S44, S54, S64, S74, S84, S94, T06.2, T11.3, T13.3, T14.4, T09.4	. .	20	1	1.05
	Injury to eyes	179	S05, T15	0.019	. .	. .	. .
	Superficial injuries	184	S00, S10, S20, S30, S40, S50, S60, S70, S80, S90, T00, T090, T110, T13.0, T14.0	0.019	. .	. .	. .

(continued)

Table D9 (continued): Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and parameters for long-term injuries

Injury	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years)	Proportion with long-term consequences (%)	Proportion entering remission in year (%)	Mortality risk ratio
Other injuries (continued)	Open wound, minor	184	S01, S08, S11.1–S11.9, S15, S21, S31, S39.9, S41, S51, S55, S61, S65, S71, S75, S81, S85, S91, S95, T01, T09.1, T11.1, T11.4, T13.1, T13.4, T14.1	0.024	. .	. .	. .
	Open wound, severe	187	S11.0	0.019	. .	. .	. .
	All other injuries, minor	184	S09, S13.0, S19, S29.8, S29.9, S33.0, S33.4, S45, S59.8, S59.9, S69.8, S69.9, S89, S99, T05.8, T05.9, T06.3, T06.8, T07, T09.6, T09.8, T09.9, T11.8, T11.9, T13.8, T13.9, T14.5, T14.8, T14.9, T16, T17, T33, T34, T35, T66, T67, T68, T69, T70, T71, T73, T74, T75, T79, T80, T81, T88	0.019	. .	. .	. .
	All other injuries, severe	150	S38.2, S38.3, S39.8, T18, T19	0.019	. .	. .	. .

Mental and substance use disorders

Table D10: Rate ratios used to calculate Indigenous prevalence estimates in the ABDS, selected mental and substance use disorders, by age and sex

		Sex-specific rate ratios		Age-specific rate ratio pattern[a][b]		
Disease	**Description**	**Male**	**Female**	**Age group (years)**		**Ratio**
Major depressive disorder	All severities: age- and sex- specific rate ratios (15-year age groups), based on CIMHA data[a]	1.8	1.9	0–14		1.23
				15–29		1.96
				30–44		2.68
				45–59		2.38
				60 and over		1.23
Dysthymia	All severities: sex-specific rate ratios, based on CIMHA data	1.7	1.5	. .		. .
Anxiety disorders	All severities: age-specific rate ratios (15-year age groups), based on CIMHA data[a]	2.2	2.2	0–14		1.59
				15–29		2.14
				30–44		2.62
				45–59		2.19
				60 and over		1.19
Bipolar affective disorder	All health states: age- and sex- specific rate ratios (15-year age groups), based on CIMHA data[a]	1.2	1.6	0–14		1.41[c]
				15–29		1.49
				30–44		1.75
				45–59		2.06
				60 and over		2.58
Alcohol use disorders	Asymptomatic/very mild/mild: age- and sex-specific hospitalisation rate ratios	. .	. .	(2011)	M	F
				15–24	2.4	2.9
				25–34	4.1	4.6
				35–44	6.2	3.8
				45–54	4.9	3.5
				55–64	2.5	2.3
				65 and over	2.3	1.0
	Moderate and severe severities: age- and sex- specific rate ratios (15-year age groups), based on CIMHA data[a]	4.2	4.2	0–14		5.38
				15–29		4.47
				30–44		5.35
				45–59		4.60
				60 and over		4.21[c]
Cannabis dependence	All severities: age- and sex- specific rate ratios (15-year age groups), based on CIMHA data[a]	5.2	6.0	0–14		6.29
				15–29		4.26
				30–44		6.27
				45–59		5.61
				60 and over		5.40[c]
Schizophrenia	All health states: age- and sex-specific rate ratios (15-year age groups), based on CIMHA data[a]	3.0	2.8	0–14		2.91[c]
				15–29		3.28
				30–44		3.59
				45–59		2.82
				60 and over		2.43

(a) Indigenous-to-total population rate ratios are from data provided by Queensland Health from the Consumer Integrated Mental Health Application (CIMHA). This is ICD-10-AM coded linked inpatient separation and community mental health services data, which provides a measure of the number of people accessing Queensland public mental health services.

(b) Applied relative to sex rate ratios if both were available.

(c) When a rate ratio from CIMHA data had relative standard error of more than 0.25, an all-ages rate ratio was used.

Reproductive and maternal conditions

Table D11: Other reproductive conditions, by ICD-10 code and relevant sequelae category

ICD-10 code	Description
Anaemia related sequela	
N92	Excessive, frequent and irregular menstruation
N93	Other abnormal uterine and vaginal bleeding
Pain related sequela	
N43	Hydrocele and spermatocele
N44	Torsion of testis
N47	Redundant prepuce, phimosis and paraphimosis
N48	Other disorders of penis
N49	Inflammatory disorders of male genital organs, not elsewhere classified
N50	Other disorders of male genital organs
N62	Hypertrophy of breast
N63	Unspecified lump in breast
N64	Other disorders of breast
N75	Diseases of Bartholin's gland
N76	Other inflammation of vagina and vulva
N77	Vulvovaginal ulceration and inflammation in diseases classified elsewhere
N82	Fistulae involving female genital tract
N83	Non-inflammatory disorders of ovary, fallopian tube and broad ligament
N99	Postprocedural disorders of genitourinary system, not elsewhere classified
Anaemia and pain related sequela	
N94	Pain and other conditions associated with female genital organs and menstrual cycle
N95	Menopausal and other perimenopausal disorders
Burden captured elsewhere	
N96	Habitual aborter
N98	Complications associated with artificial fertilisation
Experienced no pain or anaemia	
N91	Absent, scanty and rare menstruation

Appendix E: Additional information and tables for Chapter 6

Assessment of data sources

National and Indigenous-specific data sources were used to compile risk factor exposure distributions. Survey and administrative data sets were primary sources of exposure data. In the absence of good quality survey or administrative data, epidemiological studies were used to determine exposures distributions. Administrative data sources were evaluated for their level of ascertainment and coverage. Surveys were evaluated for their representativeness, potential selection bias and measurement bias (validity and reliability of measurement). Epidemiological studies were assessed for the quality of the study design, their timeliness, credibility, representativeness, and sources of bias or error.

Potential sources of data needed to have had comparable exposure definition, be relevant to the Australian population (or the Indigenous Australian population in the case of Indigenous estimates), and be timely, accurate, reliable, and credible.

Published and unpublished data sources were assessed according to the criteria in Box E1. These are largely based on the ABS's Data Quality Framework, but have been modified in some areas to better suit the range of data sources used for burden of disease analyses, including epidemiological studies.

Not all of the criteria were applicable to all types of data sources assessed, and not all dimensions were weighted equally as the importance of each dimension depends on the type of data source.

Box E1: Criteria for risk factor exposure data selection

Comparability

The data source should use an exposure definition that is comparable with that used for both the effect size and the counterfactual distribution. This definition is decided on a case-by-case basis for each risk factor on the risk factor list. The 3 options of comparability are:

1. consistent if the exposure definition is the same as the reference definition
2. comparable if the exposure definitions can be aligned
3. inconsistent if the exposure definitions are different and cannot be aligned.

Relevance and representativeness

Exposure distributions should ideally be drawn from Australian studies. If these are not available, they may be sourced from populations comparable with the Australian population. Care will need to be taken to ensure data are representative of both the Indigenous and non-Indigenous population. The 3 options of relevance for national estimates are:

1. Australian population (national)
2. Australian population (subnational)
3. regional population (such as New Zealand, the United States of America, Canada).

(continued)

Box E1 (continued): Criteria for risk factor exposure data selection

The 4 options of relevance for Indigenous estimates are:

1. the Indigenous Australian population (national)
2. the Indigenous Australian population (subnational)
3. the Australian population (total or non-Indigenous)
4. another Indigenous population (Indigenous populations in New Zealand, the United States of America and Canada).

Currency

The data source should ideally be within 5 years of the reference year. Data sources for 2000–2006 may also be included if no later data sources are available. The 3 options for currency are:

1. 2007 or later
2. 2000–2006
3. before 2000.

Accuracy

The data source should ideally have more than 90% case ascertainment or coverage of the population of interest, and a relative standard error (RSE) or confidence interval (CI) of less than 25%.

The 3 options for ascertainment/coverage are:

1. more than 90% ascertainment or coverage
2. 60–90% ascertainment or coverage
3. below 60% ascertainment or coverage

The 3 options for sources of error (sampling/non-sampling) are:

1. RSE or CI width of less than 25%of the estimate
2. RSE or CI width of 25–50% of the estimate
3. RSE or CI width greater than 50% of the estimate.

Measurement error

Data surrounding physiological and biomedical risk factors should ideally be collected and reported by clinical tests, or using similar tests in a survey setting. Self-reported data may be used, but will need to be assessed for validity. The 2 options for measurement error are:

1. clinically reported or measurement data
2. self-reported.

(continued)

Box E1 (continued): Criteria for risk factor exposure data selection

Validation

The data source should have been validated. In the case of surveys, the questionnaire should have been validated against a gold standard measurement. In the case of administrative data, the data should have been validated by the agency or organisation that manages the data collection. In the case of epidemiological studies, the results should have been validated against results from other studies to determine whether they are plausible. The 2 options for validation are:

1. validated
2. not validated.

Credibility

The data source should be collected and/or managed by a credible institution, such as a national or state/territory statistical agency or a recognised university or research organisation. For epidemiological studies, ideally, estimates from the data source will have been published and peer-reviewed. The 4 options for credibility of the estimates are:

1. published and peer-reviewed
2. published but not peer-reviewed
3. not published but peer-reviewed
4. not published and not peer-reviewed.

Accessibility/timeliness

The data source at the required level of disaggregation must be available to the AIHW with sufficient time for analysis. This criterion will identify issues of accessibility, and help prioritise data sources where such issues exist. The 3 options for availability of data are:

1. currently available
2. available with enough time for burden of disease analysis
3. unlikely to be available with enough time for burden of disease analysis.

Scoring

Each data source was scored against the matrix in Table E1.

- Any data source scoring predominantly high was included in the ABDS 2011, provided that:
  - components of comparability, relevance/representativeness, currency, and accuracy (ascertainment/coverage) were high or medium for administrative data
  - components of comparability, relevance/representativeness, currency, and accuracy (non-random error) were scored high or medium for survey data
  - components of comparability, relevance/representativeness, currency, and credibility were scored high or medium for epidemiological studies.
- A data source scoring predominantly medium was used if no other data sources for the relevant condition existed, or if there were issues of availability of better data.

A data source scoring predominantly low was not included.

Table E1: Assessment matrix for exposure data to be used in ABDS 2011

<table>
<tr><td colspan="2">Data source</td><td colspan="9"></td></tr>
<tr><td colspan="2">Data provider</td><td colspan="9"></td></tr>
<tr><td colspan="2">Level of disaggregation</td><td colspan="9"></td></tr>
<tr><td rowspan="2"></td><td rowspan="2"></td><td colspan="2">Relevance/representativeness</td><td rowspan="2"></td><td colspan="3">Accuracy</td><td rowspan="2"></td><td rowspan="2"></td><td rowspan="2"></td></tr>
<tr><td></td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td>Rating</td><td>Comparability</td><td>Australian</td><td>Indigenous</td><td>Currency</td><td>Ascertainment/ coverage</td><td>Error (sampling/ non-sampling)</td><td>Measurement error</td><td>Validation</td><td>Credibility</td><td>Accessibility/ timeliness</td></tr>
<tr><td>High</td><td>Consistent</td><td>National</td><td>National</td><td>2007 or later</td><td>>90%</td><td>Less than 25% RSE</td><td>Clinically reported</td><td rowspan="2">Validated</td><td>Published and peer reviewed</td><td>Currently available</td></tr>
<tr><td rowspan="2">Medium</td><td rowspan="2">Comparable</td><td rowspan="2">Subnational</td><td>Subnational</td><td rowspan="2">2000–2006</td><td rowspan="2">60–90%</td><td rowspan="2">25–50% RSE</td><td rowspan="2">Self-reported</td><td>Published but not peer reviewed</td><td rowspan="2">Available in time for analysis</td></tr>
<tr><td>Total Australian or non-Indigenous</td><td rowspan="2">Not validated</td><td>Not published but peer reviewed</td></tr>
<tr><td>Low</td><td>Inconsistent</td><td>Sub or super-regional</td><td>Other Indigenous population (New Zealand, United States, Canada)</td><td>Before 2000</td><td><60%</td><td>More than 50% RSE</td><td>Not known</td><td>Not published nor peer reviewed</td><td>Unlikely to be available in time for analysis.</td></tr>
</table>

Additional tables

Table E2: Risk factors in the ABDS 2011, measures of exposure and their disease outcome pairs

Number	Risk factor/cluster	Exposure description	Disease outcome
1	Air pollution	Particulate matter (2.5μg/m^3)	Chronic obstructive pulmonary disease, coronary heart disease, lower respiratory infections, lung cancer, stroke
2	Alcohol use	Daily intake	Alcohol use disorders, atrial fibrillation and flutter, breast cancer, bowel cancer, chronic liver disease, coronary heart disease, diabetes, drowning, epilepsy, oesophageal cancer, pancreatitis, stroke, influenza, laryngeal cancer, liver cancer, lower respiratory infections, falls, fire, burns and scalds, mouth and pharyngeal cancer, poisoning, suicide and self- inflicted injuries, homicide and violence, road traffic injuries—motor vehicle occupants, road traffic injuries—motorcyclists, other unintentional injuries, other land transport injuries, other road traffic injuries
		Binge drinking	
3	Childhood sexual abuse		Alcohol use disorders, suicide and self-inflicted injuries, depressive disorders
4	Dietary risk factors	Diet low in calcium	Prostate cancer
5		Diet low in fibre	Bowel cancer, coronary heart disease
6		Diet low in fruit	Coronary heart disease, laryngeal cancer, lung cancer, mouth and pharyngeal cancer, oesophageal cancer, stroke
7		Diet low in milk	Bowel cancer
8		Diet low in nuts and seeds	Coronary heart disease, diabetes
9		Diet high in processed meats	Bowel cancer, coronary heart disease, diabetes
10		Diet high in red meat	Diabetes
11		Diet high in saturated fat	Coronary heart disease
12		Diet high in sodium	Stomach cancer, stroke
13		Diet high in sugar sweetened beverages	Diabetes
14		Diet low in seafood omega 3 fatty acids	Coronary heart disease
15		Diet low in vegetables	Coronary heart disease, laryngeal cancer, mouth and pharyngeal cancer, stroke
16		Diet low in whole grains	Coronary heart disease, stroke, diabetes
17	Drug use	Illicit drug use: cocaine	Drug use disorders, chronic liver disease, HIV/AIDS, hepatitis B, hepatitis C, liver cancer, suicide and self-inflicted injuries
		Illicit drug use: opioids	
		Illicit drug use: amphetamines	
		Illicit drug use: cannabis	
		Illicit drug use: injecting drug use	

(continued)

Table E2 (continued): Risk factors in the ABDS 2011, measures of exposure and their disease outcome pairs

Number	Risk factor/cluster	Exposure description	Disease outcome
18	High blood pressure		Aortic aneurysm, atrial fibrillation and flutter, cardiomyopathy, chronic kidney disease, coronary heart disease, hypertensive heart disease, inflammatory heart disease, other cardiovascular diseases, peripheral vascular disease, rheumatic heart disease, stroke
19	High body mass		Atrial fibrillation and flutter, back pain and problems, breast cancer, bowel cancer, cardiomyopathy, chronic kidney disease, coronary heart disease, diabetes, oesophageal cancer, gallbladder cancer, hypertensive heart disease, kidney cancer, osteoarthritis, other cardiovascular diseases, pancreatic cancer, peripheral vascular disease, stroke, uterine cancer
20	High fasting blood plasma glucose		chronic kidney disease, coronary heart disease, diabetes, stroke
21	High total cholesterol		Coronary heart disease, stroke
22	Intimate partner violence		Early pregnancy loss, homicide and violence, suicide and self-inflicted injuries, depressive disorders
23	Iron deficiency		Iron-deficiency anaemia
24	Low bone mineral density		Falls
25	Occupational risks	Injuries Exposure by occupation Exposure by industry	Asthma, back pain and problems, chronic obstructive pulmonary disease, drowning, falls, fire, burns and scalds, hearing loss, homicide and violence, leukaemia, laryngeal cancer, lung cancer, mesothelioma, mouth and pharyngeal cancer, other unintentional injuries, road traffic injuries—motor vehicle occupants, road traffic injuries—motorcyclists, other land transport injuries, other road traffic injuries, ovarian cancer, pneumoconiosis, poisoning, suicide and self-inflicted injuries, all other external causes of injury
26	Physical inactivity		Breast cancer, bowel cancer, coronary heart disease, diabetes, stroke
27	Sun exposure[(a)]		Melanoma, non-melanoma skin cancer
28	Tobacco smoking cluster	Smoking: current Smoking: past Smoking: second hand	Aortic aneurysm, asthma, atrial fibrillation and flutter, bladder cancer, bowel cancer, chronic obstructive pulmonary disease, cervical cancer, coronary heart disease, diabetes, oesophageal cancer, stroke, hypertensive heart disease, interstitial lung disease, kidney cancer, leukaemia, liver cancer, lower respiratory infections, lung cancer, mouth and pharyngeal cancer, other cardiovascular diseases, other respiratory diseases, otitis media, pancreatic cancer, peripheral vascular disease, stomach cancer, tuberculosis
29	Unsafe sex		Cervical cancer, chlamydia, chronic liver disease, gonorrhoea, hepatitis B, hepatitis C, HIV/AIDS, liver cancer, syphilis, other sexually transmitted infections
30	Unimproved sanitation[(b)]		Gastrointestinal infections

(a) Only measured for the national population.

(b) Only measured for the Indigenous population.

Table E3: Risk factors, data source, units of measurement and theoretical minimum risk exposure distribution for ABDS 2011

Risk factor/cluster	Definition of exposure	National data source	Indigenous data source	Units for effect size calculation	TMRED
Air pollution	Particulate matter (2.5μg/m^3)	State/territory-based air monitoring stations	State/territory-based air monitoring stations	High atmospheric particulate matter pollution (PM2.5) levels	8.8 μg/m^3 (PM2.5)
Alcohol use	Daily intake	NDSHS 2010; Apparent consumption of alcohol data	The NATSISS 2008	Lifetime risk: average consumption of pure alcohol (grams per day)	No alcohol consumption
	Binge drinking			Single occasion risk: proportion of the population reporting consumption of 0.06kg or more of pure alcohol on a single occasion	Less than 0.06kg of pure alcohol on a single occasion
Childhood sexual abuse	. .	Personal Safety Survey 2012	Indirect methods based on rate ratios	Exposed to childhood sexual abuse (prevalence)	No childhood sexual abuse
Diet low in calcium	. .	AHS 2011–12	AATSIHS 2012–13	Per 1,000mg per day intake decrease	1,600mg per day
Diet low in fibre	. .	AHS 2011–12	AATSIHS 2012–13	Per 20g per day of fibre intake decrease	30g/day
Diet low in fruit	. .	AHS 2011–12	AATSIHS 2012–13	Per 100g per day of fruit intake decrease	300g per day
Diet low in milk	. .	AHS 2011–12	AATSIHS 2012–13	Per 226.8g per day intake decrease	450g per day
Diet low in nuts and seeds	. .	AHS 2011–12	AATSIHS 2012–13	Per 4.05g per day intake decrease	16.3g per day
Diet high in processed meats	. .	AHS 2011–12	AATSIHS 2012–13	Per 50g per day intake increase	0g per day
Diet high in red meat	. .	AHS 2011–12	AATSIHS 2012–13	Per 100g per day intake increase	100g per day
Diet high in saturated fat	. .	AHS 2011–12	AATSIHS 2012–13	Per 5% energy from polyunsaturated fat increase	12% of energy

(continued)

Table E3 (continued): Risk factors, data source, units of measurement and theoretical minimum risk exposure distribution for ABDS 2011

Risk factor/cluster	Definition of exposure	National data source	Indigenous data source	Units for effect size calculation	TMRED
Diet high in sodium	. .	AHS 2011–12	AATSIHS 2012–13	Per 2.3g per day intake increase	1,600mg
Diet high in sweetened beverages	. .	AHS 2011–12	AATSIHS 2012–13	Per 226.8g per day intake increase	0g per day
Diet low in seafood omega 3 fatty acids	. .	AHS 2011–12	AATSIHS 2012–13	Per 100mg per day of omega 3 intake decrease	250mg per day
Diet low in vegetables	. .	AHS 2011–12	AATSIHS 2012–13	Per 100g per day of vegetable intake decrease	400g per day
Diet low in whole grains	. .	AHS 2011–12	AATSIHS 2012–13	Per 50g per day intake decrease	125g per day
Drug use	Illicit drug use: cocaine	. .	. .	Direct evidence: proportion of the population cocaine use disorder	. .
	Illicit drug use: opioids	. .	. .	Direct evidence: proportion of the population opioid use disorder	. .
	Illicit drug use: cannabis	. .	. .	Direct evidence: proportion of the population cannabis use disorder	. .
	Illicit drug use: amphetamines	. .	. .	Direct evidence: proportion of the population amphetamine use disorder	. .
	Illicit drug use: injecting drug use	Kirby annual surveillance reports	Kirby annual surveillance reports	Injecting drug use: direct evidence from Kirby Institute publications (which disaggregated notifications for HIV/AIDS and bloodborne viruses by exposure category).	. .
High blood pressure	. .	AHS 2011–12	AATSIHS 2012–13	Per 10mmHg of systolic blood pressure increase	Mean 110–115mmHg (SD 6mmHg)

(continued)

Table E3 (continued): Risk factors, data source, units of measurement and theoretical minimum risk exposure distribution for ABDS 2011

Risk factor/cluster	Definition of exposure	National data source	Indigenous data source	Units for effect size calculation	TMRED
High body mass	. .	AHS 2011–12	AATSIHS 2012–13	Per 5kg/m^2 of body mass index increase	Mean body mass index 21–23kg/m^2 (SD 1 kg/m^2)
High fasting blood plasma glucose	. .	AHS 2011–12	AATSIHS 2012–13	Per 1 mmol/L of fasting plasma glucose increase	Mean 4.9–5.3 mmol/L (SD 0.3 mmol/L)
High total cholesterol	. .	AHS 2011–12	AATSIHS 2012–13	Per 1 mmol/L of total cholesterol increase	Mean 3.8–4.0 mmol/L (SD 0.9 mmol/L)
Intimate partner violence	. .	ABS Personal Safety Survey 2012; National Homicide Monitoring Program	Indirect methods based on rate ratios	Exposed to intimate partner violence (prevalence)	No intimate partner violence
Iron deficiency	. .	AHS 2011–12	AATSIHS 2012–13	Per 1g/dL decrease in haemoglobin	13.51g/dL haemoglobin
Low bone mineral density	. .	Geelong Osteoporosis Study (Barwon Health)	Geelong Osteoporosis Study (Barwon Health)	Standardised bone mineral density at the femoral neck	95th percentile of the Third National Health and Nutrition Examination Survey (NHANES-III) cohort by age (CDC 2012)
Occupational risks	Injuries	Work-related Traumatic Injury Fatalities, Australia 2010–11; Workers' Compensation Statistics 2010–11	Indirect methods based on rate ratios from NHMD	Direct evidence: number of workplace fatalities and the adjusted number of workers' compensation claims for injuries	. .
	Occupation	Census of Population and Housing 2011; ABS Labour force survey, June 2011	Census of Population and Housing 2011	Distribution of the labour force by industry type	. .
	Industry	Census of Population and Housing 2011; ABS Labour force survey, June 2011	Census of Population and Housing 2011; ABS Labour force survey, June 2011	Distribution of the labour force by broad occupation group	. .
Physical inactivity	. .	AHS 2011–12	AATSIHS 2012–13	METs of less than 600, 600–3,999, 4,000–7,999	All individuals are highly active with METs of 8,000 or more.

(continued)

Table E3 (continued): Risk factors, data source, units of measurement and theoretical minimum risk exposure distribution for ABDS 2011

Risk factor/cluster	Definition of exposure	National data source	Indigenous data source	Units for effect size calculation	TMRED
Tobacco use	Smoking	NDSHS 2010	AATSIHS 2012–13	Current smokers: proportion of the population who currently smoke	No exposure to tobacco smoking
	Smoking: second hand	NDSHS 2010	AATSIHS 2012–13	Second-hand smokers: proportion of the population exposed to second-hand smoke	No exposure to second hand smoke
	Past smoking	. .	. .	Past smokers: Peto et al. 1992	. .
Unsafe sex	. .	Kirby Institute annual surveillance reports National HIV Register	Kirby Institute annual surveillance reports National HIV Register	Direct evidence: all sexually transmitted infections and cervical cancer attributed to unsafe sex Cases of HIV/AIDS, hepatitis B and hepatitis C reported due to unsafe sex	. .
Unimproved sanitation	. .	. .	AATSIHS 2012–13	Exposed to unimproved sanitation (prevalence)	Improved sanitation

Table E4: Proportion and method used to align GBD relative risks with ABDS 2011 diseases

ABDS 2011 disease	GBD 2013 cause	Source of disaggregation	Proportion of ABDS disease (%)
Stroke	Ischaemic stroke	Thrift et al. 2009	0.776
Stroke	Haemorrhagic stroke	Thrift et al. 2009	0.224
Mouth and pharyngeal cancer	Mouth cancer	Australia Cancer Database Incidence 2011	0.670
Mouth and pharyngeal cancer	Nasopharynx cancer	Australia Cancer Database Incidence 2011	0.243
Chronic liver disease	Chronic liver disease due to hepatitis B	GBD 2010	0.232
Chronic liver disease	Chronic liver disease due to hepatitis C	GBD 2010	0.421
Chronic liver disease	Chronic liver disease due to alcohol	GBD 2010	0.269
Liver cancer	Liver cancer due to hepatitis B	GBD 2010	0.206
Liver cancer	Liver cancer due to hepatitis C	GBD 2010	0.476
Liver cancer	Liver cancer due to alcohol	GBD 2010	0.299
Inflammatory heart disease	Endocarditis	Separations in NHMD 2011	0.206
Osteoarthritis	Osteoarthritis of the hip	Prevalence GBD 2013	0.180
Osteoarthritis	Osteoarthritis of the knee	Prevalence GBD 2013	0.820
Chronic kidney disease	Diabetic chronic kidney disease	Australia and New Zealand Dialysis and Transplantations analysis	0.400
Non-melanoma skin cancer	Basal cell cancer	Average incidence from Australian cohort studies 1978–2002 (F Xiang 2015, pers. comm., 9 November)	0.570
Non-melanoma skin cancer	Squamous cell cancer	Average incidence from Australian cohort studies 1978–2002 (F Xiang 2015, pers. comm., 9 November)	0.430

Table E5: Population attributable fractions adjusted because they are in a causal pathway

Risk factor 2nd in pathway	ABDS 2011 disease
High body mass	Bowel cancer, coronary heart disease, oesophageal cancer, other cardiovascular diseases, peripheral vascular disease, stroke
High blood pressure	Atrial fibrillation, cardiomyopathy
High fasting plasma glucose	Coronary heart disease, stroke
High total cholesterol	Coronary heart disease
Diet low in fruit	Coronary heart disease
Diet low in vegetables	Coronary heart disease
Diet low in whole grains	Coronary heart disease
Alcohol use	Homicide and violence, suicide and self-inflicted injuries
Drug use	Homicide and violence

Appendix F: Additional information and tables for Chapter 8

Table F1: ABDS quality index, Dimension I—Data relevance scores

Score	Criteria
5	Current data from: fully enumerated disease register (such as cancer register) or administrative data/unlinked hospitalisation data for condition with a high likelihood of hospitalisation/national Australian survey (such as the Australian Health Survey) of either diagnostically confirmed conditions/sequelae; or established high correlation between self-report and clinical diagnosis specific to the population with no major variability due to small numbers. For Indigenous estimates: data had an Indigenous identifier/no known Indigenous under-identification issues/appropriate adjustment factors available where there were known under-identification issues. No severity distribution needed, or high-quality empirical data on this distribution were available.
4	Same as '5' BUT not fully enumerated with either known gaps in coverage/not diagnostically confirmed/within 2 years of the reference date/some variability due to small numbers (for example, a particular age group) or had high RSEs/severity not available. For Indigenous estimates: data had a known issue with Indigenous under-identification but there were no adjustment factors available—however, a subset (for example, certain jurisdictions) of the primary data provided adequate Indigenous identification (such as the Australian Cancer Database). It was also used for estimates with components that scored between 5 and 3.
3	Same as '4' BUT with medium specificity of the data source to the condition/sequela being estimated. For example: - for survey data, there was known medium correlation between what was collected (for example, measurement, self-report and clinical diagnosis) and the condition - for hospitals data, condition had a medium likelihood of hospitalisation (that is, condition only results in hospitalisation in severe or certain cases). Also, data were from: a single, large area (more than 1 state/territory) Australian study of very good quality/a systemic meta-analysis that could be generalised/review of Australian studies with medium currency. For Indigenous estimates: there was a known issue with Indigenous under-identification, but no adjustment factors available (for example, Non-admitted Patient Emergency Department Care Database). It was also used for estimates with components that scored between 4 and 2.

(continued)

Table F1 (continued): ABDS quality index, Dimension I—Data relevance scores

Score	Criteria
2	Data were from: small Australian studies of good quality/small international area study with good sampling that could be generalised to the Australian population/a systematic and meta-analysis that could be generalised/a review of Australian and/or international (for example, other highincome countries) studies, AND data source was specific to the condition/sequela being estimated AND data were collected less than 5 years previously for a disease or condition that had a known trend of changing over time OR data were collected more than 5 years previously for a disease or condition that had a known trend of **not** changing over time. For Indigenous estimates: secondary data source with no known significant gaps was used to indirectly estimate prevalence. It was also used for estimates with components that scored between 3 and 1.
1	Data were from: a small Australian study and refers to data more than 5 years from the reference year for a disease or condition that has a known or unknown trend of changing over time/a small number of overseas research studies of questionable generalizability to the Australian context/a secondary data source for indirect prevalence estimates. For Indigenous estimates: proxy data source with significant gaps in age/sex was used to indirectly estimate prevalence (for example, rate ratios obtained from 2 data sources without the appropriate sex/age breakdowns).

Table F2: ABDS quality index, Dimension II—Data transformation scores

Score	Criteria
5	Data were directly applied to the model and minimal or no extra modelling was required. For Indigenous estimates: where there were known Indigenous under-identification issues, agreed adjustment factors were applied. Severity distribution (if required) was obtained directly from the data.
4	Rates were projected to the reference year, taking into account changes in underlying trend, and applied to reference population / broad sex or age distributions were converted to 5-year age groups using trend analyses/pooled data from multiple years or sources with comparable definitions/ratios of related and primary data (for example, incidence-to-separations ratio from 1 state) applied to primary data (for example, applied to national separations data). Severity distribution (if required) was obtained from an Australian study. For Indigenous estimates: under-identification issues were overcome by basing data on a subset of jurisdictions with adequate Indigenous identification. It was also used for estimates with components that scored between 5 and 3.
3	Rates from another year were applied to the same population for the reference year not accounting for any change in the underlying trend/rates from another population were applied to the reference population for the reference year where there was evidence or expert advice supporting no difference in the underlying prevalence between populations/age or sex distribution from alternative (but relevant) data source applied to the base data/pooled data from multiple sources with differing definitions after standardisation/applied New Zealand Burden of Disease prevalence rates or severity distributions based on linked data/severity distribution obtained from international studies similar to Australia (such as other high-income countries or GBD high-income severity distribution)/ratios of related and similarly defined secondary data (for example, incidence-to-separations ratio) applied to primary data (for example, prevalence). For Indigenous estimates: prevalence was modelled using data for another population (for example, the national population) where there was no evidence of a difference in the prevalence rates between the 2 populations. It was also used for estimates with components that scored between 4 and 2.
2	Other epidemiological measures were modelled to produce the estimates / indirect modelling methods were used, including indirect modelling of prevalence from other measures, such as incidence, mortality, etc./GBD global severity distribution was used. For Indigenous estimates: prevalence was modelled using data for a different population where there was a known difference between that population and the Indigenous population but no advice/method on how to model the difference. It was also used for estimates with components that scored between 3 and 1.
1	Inference of distributions from: other slightly related data sources/expert advice only/indirect modelling methods where the data source had an inconsistent definition of the condition, had a low coverage factor or data were not within 5 years of the reference year OR the severity distribution from another disease or condition was applied as a proxy.

Table F3: National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Blood and metabolic disorders					
Cystic fibrosis	A	B	B	B	National prevalence estimates, by severity, were obtained from Australian Cystic Fibrosis Data Registry annual report. Hospitals data were used to apportion estimates into 5-year age group for registrants over 65 years. Indigenous prevalence estimates were based on NHMD separations. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Haemophilia	A	B	B	B	National prevalence estimates by severity were obtained from Australian Bleeding Disorders Registry annual report. Hospitals data were used to apportion estimates into 5-year age groups. Severity distributions were obtained from the annual report for males, and based on expert advice for females. Indigenous prevalence estimates were based on NHMD separations. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Haemolytic anaemias	A	B	B	B	National and Indigenous prevalence estimates by sequela were obtained from the NHMD with a high likelihood of hospitalisation. Person-to-separations ratios derived from Western Australian linked hospitalisations data were applied to unlinked hospitalisation data to estimate prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Iron-deficiency anaemia	A	B	B	B	National prevalence estimates for the anaemia envelope, by severity were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Some transformations were required to overcome variability in the data source caused by small numbers. Anaemia sequelae found in other diseases were subtracted from the anaemia envelope to avoid double-counting. The national severity distribution was applied to Indigenous prevalence estimates, due to variability in the data source caused by small numbers.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Blood and metabolic disorders (continued)					
Protein-energy deficiency	A	B	B	B	National and Indigenous prevalence estimates, by sequela, in elderly Australians was obtained from an Australian community-living based study assessing malnutrition (Rist et al. 2012). Indigenous prevalence estimates in children aged under 5 were obtained from the AATSIHS 2012–13. Minimal transformations were required to determine severity distributions specific to remoteness category.
Other blood and metabolic disorders	C	A	B	C	National and Indigenous prevalence estimates by sequela were obtained from the NHMD with a mix of medium and high likelihood of hospitalisation. Durations to derive point prevalence were determined based on NHMD data or durations from diseases with comparable symptoms. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Cancer and other neoplasms					
Mouth and pharyngeal cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Laryngeal cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Laryngectomy prevalence was derived from hospitals data and applied to the 10-year prevalence of laryngeal cancer Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD and NHMD were used to adjust for Indigenous under-identification.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Cancer and other neoplasms (continued)					
Oesophageal cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Stomach cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Bowel cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Stoma hazard rates were derived from hospitals data and applied to the 10-year prevalence of bowel cancer. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification. National stoma hazard rates were assumed to apply, due to insufficient Indigenous-specific data.
Liver cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Cancer and other neoplasms (continued)					
Gallbladder cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Pancreatic cancer	A	B	B	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Lung cancer	A	A	B	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Mesothelioma	A	A	B	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Cancer and other neoplasms (continued)					
Melanoma of the skin	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland. Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Non-melanoma skin cancer	B	A	B	B	Prevalence of metastatic and terminal sequelae were derived directly from NMD. Diagnosis and treatment were based on Medicare Benefits Schedule claims for first excision, adjusted for histological confirmation and hospital separations of people undergoing skin related procedures. Validated adjustment factors for the NMD and NHMD were used to adjust for Indigenous under-identification. Indigenous Medicare Benefits Schedule claims were based on Indigenous-to-national ratio of complex non-melanoma skin cancers from hospitals data.
Breast cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Mastectomy hazard rates were derived from hospitals data and applied to the 10-year prevalence of breast cancer, by age and sex. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD and NHMD were used to adjust for Indigenous under-identification. Indigenous-specific mastectomy hazard rates were derived from hospitals data and applied to the 10-year prevalence of breast cancer for females.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Cancer and other neoplasms (continued)					
Cervical cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Uterine cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Ovarian cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Prostate cancer	A	C	B	C	Diagnosis/treatment and controlled phases derived were directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Treatment and impotence ratios from a New South Wales study (Smith et al. 2009) were applied to 10-year prevalence of prostate cancer. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification. National treatment and impotence ratios were assumed to apply.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Cancer and other neoplasms (continued)					
Testicular cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Bladder cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Radical cystectomy with stoma or neobladder ratios were derived from hospitals data and applied to the 10-year prevalence of bladder cancer, with rates of incontinence as per Gilbert et al. (2007). Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD and NHMD were used to adjust for Indigenous under-identification. National radical cystectomy ratios were assumed to apply.
Kidney cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Brain and central nervous system cancer	A	C	B	C	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Proportion of traumatic brain injury survivors with long-term effects were applied to lifetime prevalence of brain and central nervous system cancer. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Cancer and other neoplasms (continued)					
Thyroid cancer	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Non-Hodgkin lymphoma	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Hodgkin lymphoma	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Leukaemia	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Myeloma	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Other lympho-haematopoietic (blood) cancers	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Unknown primary cancer	A	A	B	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Benign and uncertain brain tumours	E	E	D	D	Diagnosis/treatment and controlled phases were derived directly from the ACD for Victoria, Queensland and Western Australia, and incidence-to-separation ratio for Victoria, Queensland and Western Australia were applied to hospital separations for remaining jurisdictions. Prevalence of metastatic and terminal sequelae were derived directly from the NMD. Proportions of traumatic brain injury survivors with long-term effects were applied to estimated lifetime prevalence of benign brain tumours from malignant-to-benign ratios based on Porter et al. 2010. Validated adjustment factors for the NHMD and NMD were used to adjust for Indigenous under-identification. The higher rating for Indigenous estimates is due to the burden from long-term effects contributing a smaller proportion to the total YLD for this disease in the Indigenous population.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Cancer and other neoplasms (continued)					
Ductal carcinoma in situ (breast)	A	B	B	B	Diagnosis/treatment phase was derived directly from the ACD. Mastectomy prevalence was derived from the NHMD from 2001–2011, adjusted using a prevalence-to-separations ratio from Western Australian linked data. Indigenous cancer incidence and prevalence estimates were based on the ratio of small (less than 2 centimetres) breast tumours in national-to-Indigenous women ratio to the national incidence of ductal carcinoma in situ. Validated adjustment factors for the NHMD and NMD were used to adjust for Indigenous under-identification. Indigenous-specific mastectomy hazard rates were derived from hospitals data and applied to the 10-year prevalence of breast cancer for females.
Other malignant neoplasms (cancers)	A	B	B	B	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD. Indigenous cancer incidence and prevalence estimates were based on data from New South Wales, Victoria, Queensland, Western Australia and the Northern Territory. Validated adjustment factors for the NMD were used to adjust for Indigenous under-identification.
Other benign, in situ and uncertain neoplasms	C	D	C	D	Diagnosis/treatment phase was derived directly from the NHMD (acknowledging that this will be the more severe end of the spectrum) using principal diagnosis, and adjusted for repeat admissions using ratio from ABDS 2003. Metastatic and terminal phases were derived directly from the NMD. Validated adjustment factors for the NHMD and NMD were used to adjust for Indigenous under-identification.
Cardiovascular diseases					
Coronary heart disease	B	B	B	C	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Severity distribution was obtained from the GBD 2013. Ratios from Western Australian linked hospitalisations and deaths data were used to transform estimates from unlinked hospitalisation data into prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. National distributions (including severity distributions) were used to estimate prevalence for some sequelae for the Indigenous population.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Cardiovascular diseases (continued)					
Stroke	B	C	B	C	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Severity distribution was obtained from the GBD 2013, and transformed into broad age-specific severity distributions. Ratios from Western Australian linked hospitalisations and deaths data were used to transform estimates from unlinked hospitalisation data into prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. National severity distributions were used for the Indigenous population.
Rheumatic heart disease	A	C	A	B	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Severity distribution was obtained from the GBD 2013. Ratios from Western Australian linked hospitalisations and deaths data were used to transform estimates from unlinked hospitalisation data into prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. National distributions were used to estimate prevalence for some sequelae for the Indigenous population.
Non-rheumatic valvular disease	A	B	A	C	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Severity distribution was obtained from the GBD 2013. Ratios from Western Australian linked hospitalisations and deaths data were used to transform estimates from unlinked hospitalisation data into prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. National distributions were used to estimate prevalence for some sequelae for the Indigenous population.
Hypertensive heart disease	B	C	B	C	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Severity distributions were obtained from the GBD 2013. Ratios from Western Australian linked hospitalisations and deaths data were used to transform estimates from unlinked hospitalisation data into prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. National distributions were used to estimate prevalence for some sequelae for the Indigenous population.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Cardiovascular diseases (continued)					
Atrial fibrillation and flutter	D	C	D	C	Overall national prevalence was based on non-Maori NZBDS prevalence rates applied to the Australian population, due to lack of recent and robust Australian data at the time of analysis. Overall Indigenous prevalence was based on the Maori NZBDS prevalence rates applied to the Indigenous Australian population.
Inflammatory heart disease	B	C	B	C	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Severity distribution was obtained from GBD 2013. Ratios from Western Australian linked hospitalisations and deaths data were used to transform estimates from unlinked hospitalisation data into prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. National distributions were used to estimate prevalence for some sequelae for the Indigenous population.
Cardiomyopathy	B	C	B	C	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Severity distributions were obtained from GBD 2013. Ratios from Western Australian linked hospitalisations and deaths data were used to transform estimates from unlinked hospitalisation data into prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. National distributions were used to estimate prevalence for some sequelae for the Indigenous population.
Aortic aneurysm	A	A	A	A	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Peripheral vascular disease	D	C	D	C	Overall national prevalence was based on non-Maori NZBDS prevalence rates applied to the Australian population, due to lack of recent and robust Australian data at the time of analysis. Overall Indigenous prevalence was based on the Maori NZBDS prevalence rates applied to the Indigenous Australian population.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Endocrine disorders					
Diabetes	C	C	C	C	National prevalence estimates were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Moderate transformations were required to overcome variability in the data source caused by small numbers. For some sequelae, prevalence estimates were obtained from the Fremantle Diabetes Study), therefore, moderate transformations were required to produce national prevalence estimates. For other sequelae (amputation due to diabetes), prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Ratios from Western Australian linked hospitalisations and deaths data were used to transform estimates from unlinked hospitalisation data into prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Gastrointestinal disorders					
Gastroduodenal disorders	B	D	C	B	Prevalence based on hospitals separations were adjusted for physician-diagnosed disease (Sung et al. 2009). Anaemia estimate were derived from NHMD. Severity distribution was obtained from GBD 2013. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. The higher rating for Indigenous methods is due to the contribution of different sequelae to the total YLD for the Indigenous population.
Appendicitis	A	A	A	A	Derived directly from hospital separations with a high likelihood of hospitalisation adjusted for duration. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Abdominal wall hernia	A	A	A	A	Derived directly from hospital separations with a high likelihood of hospitalisation adjusted for duration. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Vascular disorders of intestine	A	A	A	A	Derived directly from hospital separations with a high likelihood of hospitalisation adjusted for duration. Stomas were assumed from a stoma incidence hazard derived from the NHMD. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Gastrointestinal disorders (continued)					
Intestinal obstruction (without hernia)	A	A	A	A	Derived directly from hospital separations with a high likelihood of hospitalisation adjusted for duration. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Inflammatory bowel disease	D	C	D	C	Rates from Barwon study (Studd 2013) applied to national and Indigenous populations.
Diverticulitis	A	A	A	A	Derived directly from hospital separations with a high likelihood of hospitalisation adjusted for duration. Stomas were assumed from a stoma incidence hazard derived from the NHMD. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification
Chronic liver disease	B	B	B	B	Prevalence of liver disease, by stage, was based on hospitals separations with a medium–high likelihood of hospitalisation, and adjusted using person-to-separation presentation ratio from linked Western Australian data. Prevalence of liver transplant patients was based on ratio from linked Western Australian hospitals/mortality data and applied to the population of each jurisdiction. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Gallbladder and bile duct disease	A	A	A	A	Derived directly from hospital separations with a high likelihood of hospitalisation adjusted for duration. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Pancreatitis	A	A	B	B	Derived directly from hospital separations with a medium–high likelihood of hospitalisation adjusted for duration. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Gastro-oesophageal reflux disease	C	D	C	D	Estimates for moderate/severe gastro-oesophageal reflux disease for people seeking medical attention were based on Harrison et al. (2013). National estimates were applied to Indigenous populations.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Gastrointestinal disorders (continued)					
Functional gastrointestinal disorders	D	C	D	C	Derived from small area study in Penrith, New South Wales (Boyce et al. 2006), and applied to populations.
Hearing and vision disorders					
Vision loss	D	B	B	D	National prevalence estimates, by cause of vision loss, were based on estimates from the Melbourne Visual Impairment Project (Taylor 2005; Weih et al. 2000), a small Australian study with data more than 5 years from the reference date. Some transformations were required to calculate estimates by age, sex and severity using population data, ABDS 2003 estimates, or expert advice when, required. Indigenous prevalence estimates were calculated from the National Indigenous Eye Health Survey. Moderate transformations were required to calculate estimates by age, sex and severity.
Hearing loss	B	C	B	D	National prevalence estimates were based on estimates from Australian Hearing annual reports, AHS 2011–12, and the Blue Mountains Hearing Study. Minimal transformations were required to calculate estimates by age and sex. Estimates for hearing loss with tinnitus were based on a United States National Health Interview Survey (Hoffman & Reed 2004). Severity distribution was based on GBD 2013 for high-income countries. Indigenous prevalence estimates were calculated from the AATSIHS 2011–12. Considerable transformations were required to determine the severity distribution, based on the GBD 2013 adjusted using data from the Northern Territory Hearing health outreach services, and differences in the age and sex distribution in the Indigenous populations.
Other vision disorders	D	B	D	D	National prevalence estimates were based on estimates from the Melbourne Visual Impairment Project, and calculated prevalence estimates for causes of vision loss in the ABDS 2011. Some transformations were required to calculate estimates by age, sex and severity using population data, ABDS 2003 estimates, or expert advice, when required. Indigenous prevalence estimates were calculated from the National Indigenous Eye Health Survey. Considerable transformations were required to calculate estimates by age, sex and severity.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Hearing and vision disorders (continued)					
Other hearing and vestibular disorders	C	C	C	D	National prevalence estimates, by sequela, were based on estimates from the AHS 2011–12. Moderate transformations were required to obtain age- and sex-specific distributions using the NHMD or population data. Indigenous prevalence estimates were based on estimates from the AHS 2011–12. Considerable transformations were required to obtain age- and sex-specific distributions, by sequela, using the NHMD or population data.
Infant and congenital conditions					
Pre-term birth and low birthweight complications	C	D	D	D	National and Indigenous prevalence for acute complications was derived from the National Perinatal Data Collection. National and Indigenous prevalence estimates for long-term sequelae were derived from intellectual disability envelope.
Birth trauma and asphyxia	D	E	D	D	National and Indigenous prevalence estimates were derived from the intellectual disability envelope. The severity distribution for birth trauma and asphyxia was derived from the NHMD. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Cerebral palsy	B	C	D	D	Incidence for cerebral palsy was estimated from the Australia Cerebral Palsy Register. Prevalence estimates (1913–2011) were adjusted for standard background mortality using the Australian life table (ABS 2012b), and mortality estimates from the NMD. An Australian-specific severity distribution derived from the Gross Motor Function Classification System was applied to the estimates (Cerebral Palsy Alliance 2013). The Australia Cerebral Palsy Register (Cerebral Palsy Alliance 2013) reported 3.5% of people with cerebral palsy were born from Aboriginal and/or Torres Strait Islander mothers. This proportion was applied to national estimates to derive the Indigenous prevalence.
Neonatal infections	A	A	A	A	National and Indigenous prevalence estimates were obtained directly from the NHMD, and applied a 4-weeks duration. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Infant and congenital conditions (continued)					
Other disorders of infancy	A	A	A	A	National and Indigenous prevalence estimates were obtained directly from the NHMD, and applied a 4-weeks duration. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Neural tube defects	C	D	D	D	Live birth prevalence rates were derived from WARDA data 1980–2011. DISMOD II was used to model prevalence for those aged over 1. Prevalence estimates were distributed into different health states using proportions from Hunt & Oakeshott (2003). Indigenous estimates were obtained by applying rate ratios, derived from WARDA, to national estimates.
Cardiovascular defects	C	C	C	C	The acute sequela (cardiovascular defects prior to surgery) was derived from WARDA with a duration of 1 year. Indigenous estimates were obtained by applying rate ratios derived from WARDA to national estimates. The chronic sequela (heart failure due to congenital cardiovascular defects) was modelled under the heart failure envelope. National and Indigenous heart failure prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Severity distributions were obtained from the GBD 2013. Moderate transformations were required to produce prevalence estimates, including using ratios from Western Australian linked hospitalisations and deaths data and validated adjustment factors for Indigenous under-identification in the NHMD.
Cleft lip and/or palate	C	B	D	D	Live birth prevalence rates were derived from WARDA data 1980–2011. The prevalence rate for a given age in 2011 was obtained from live birth prevalence rate during the relevant birth year. Where WARDA data were unavailable for an age cohort, the prevalence rate from the closest reference year was used. Indigenous estimates were obtained by applying rate ratios, derived from the NHMD, to national estimates.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Infant and congenital conditions (continued)					
Gastrointestinal malformations	C	D	D	D	Prevalence in babies less than 1 year was sourced directly from the live birth prevalence rate derived from WARDA. DISMOD II was used to model prevalence for those aged over 1. The proportion of anorectal malformations was derived from WARDA data published in the International Clearinghouse for Birth Defects Surveillance and Research for 2011 (ICBDSR 2013). It was assumed 62.5% people with anorectal malformations experience faecal incontinence (Peña & Hong 2000). Indigenous estimates were obtained by applying rate ratios, derived from WARDA, to national estimates.
Urogenital malformations	C	B	D	D	It was assumed people born with urogenital malformations have the same life expectancy as the general population and 0 remission; therefore, the live birth prevalence rate from WARDA was held constant and applied to the national population by sex and age groups. Indigenous estimates were obtained by applying rate ratios, derived from WARDA, to national estimates.
Down syndrome	D	E	D	E	National and Indigenous prevalence estimates were derived from the intellectual disability envelope. Prevalence rates were modelled to account for a reduced life expectancy for people with Down syndrome.
Brain malformations	D	D	D	D	National and Indigenous prevalence estimates were derived from the intellectual disability envelope. Prevalence rates were modelled to account for a reduced life expectancy for people with moderate and severe brain malformations.
Other chromosomal abnormalities	D	E	D	E	National and Indigenous prevalence estimates were derived from the intellectual disability envelope.
Infectious diseases					
HIV/AIDS	B	B	B	B	National prevalence estimates were based on modelled prevalence and treatment coverage estimates produced by the Kirby Institute, for which there are known gaps in coverage. Some modelling was required to estimate severity and age distributions. Indigenous estimates were modelled using the same approach and distributions as for national estimates.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Infectious diseases (continued)					
Tuberculosis	A	A	A	A	Incidence was based on notifications to the NNDSS, which are thought to be good estimates of the incidence of tuberculosis. Minimal modelling was required. Indigenous estimates were of high quality, based on average Indigenous notification rates over 3 years.
Syphilis	B	B	B	C	Incidence was based on notifications to the NNDSS, with some adjustment for under-notification. Minimal modelling was required. Indigenous estimates were calculated using the proportion of cases that identified as Indigenous in selected states, which were then applied to national rates.
Chlamydia	C	C	C	C	Incidence and severity were based on notifications to the NNDSS, BEACH, linked hospitals data, state surveillance reports and an epidemiological study (Reekie et al. 2014). Moderate transformations were required to derive point prevalence estimates by sex, age and severity. Indigenous estimates were based on notification rates in selected states with adequate Indigenous data quality. Infertility sequela was based on published estimates (Hafner & Pelzer 2011), expert advice and GBD 2013 proportions.
Gonorrhoea	C	C	C	C	Incidence and severity were based on notifications to the NNDSS, BEACH, linked hospitals data, state surveillance reports and an epidemiological study (Reekie et al. 2014). Moderate transformations were required to derive point prevalence estimates by sex, age and severity. Indigenous estimates were based on notification rates in selected states with adequate Indigenous data quality. Infertility sequela was based on published estimates (Hafner & Pelzer 2011), expert advice and GBD 2013 proportions.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Infectious diseases (continued)					
Other sexually transmitted infections	C	C	D	C	Estimates were based on BEACH encounters for pelvic inflammatory disease, genital herpes and genital wart, so only some 'other sexually transmitted infections' were captured. Considerable transformations were required to overcome data gaps. Estimates for pelvic inflammatory disease due to other sexually transmitted infections were based on GBD distributions and an epidemiological study (Reekie et al. 2014). For Indigenous estimates, rate ratios were applied based on findings in epidemiological studies. Infertility sequela was based on published estimates (Hafner & Pelzer 2011), expert advice and GBD 2013 proportions.
Hepatitis A	B	C	A	B	Estimates were based on notifications to the NNDSS. As these are thought to underestimate hepatitis A incidence, these estimates were adjusted for under-notification. Indigenous estimates were based on severity distributions relative to the number of hospitalisations.
Hepatitis B (acute)	C	C	D	B	Estimates were based on the estimated annual hepatitis B incidence modelled by the Kirby Institute. The age distribution was drawn from NNDSS notifications for newly acquired hepatitis B. Severity distributions were based on a combination of epidemiological findings (Shepard et al. 2006) (moderate symptomatic cases) and hospitalisations (severe cases). Indigenous estimates were based on rate ratios (Indigenous to total population) from NNDSS notifications and hospitalisations.
Hepatitis C (acute)	C	C	D	B	Estimates were based on the estimated annual hepatitis C incidence modelled by the Kirby Institute. The age distribution was drawn from NNDSS notifications for newly acquired hepatitis C. The proportion symptomatic was based on Victorian annual communicable diseases surveillance reports, and severe cases were identified as admissions to hospital for hepatitis C. Indigenous estimates were based on rate ratios (Indigenous to total population) from NNDSS notifications.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Infectious diseases (continued)					
Upper respiratory infections	C	C	D	C	Estimates were based on encounters with general practitioners for mild and moderate upper respiratory infections, as reported in the BEACH data, which has low to medium specificity for upper respiratory infection, as some people with the condition may not visit a general practitioner. An Indigenous-to-national hospitalisation rate ratio was applied to national rates for Indigenous estimates.
Otitis media	B	B	C	B	Estimates of acute cases were based on GP encounters for otitis media reported in the BEACH data. Chronic estimates were based on hospitalisations with a myringotomy with tube insertion. Some transformations were required using adjustment factors for BEACH data. For Indigenous estimates, a rate ratio was applied based on comparison of ear and mastoid process conditions self-reported in the NHS 2011–12 and AATSIHS 2012–13.
Lower respiratory infections	B	B	C	C	Estimates of moderate cases (including pneumonia) were based on GP encounters for pneumonia and other lower respiratory infections reported in the BEACH data. Hospitalisations were used to estimate the number of severe cases. Some transformations were required to overcome gaps in age distribution. For Indigenous estimates, an Indigenous-to-national rate ratio was applied to national rates, based on hospitalisation rates.
Influenza	B	B	C	C	Estimates of moderate cases were based on GP encounters for influenza reported in the BEACH data. Hospitalisations were used to estimate the number of severe cases. Some transformations were required to overcome variability across age groups. For Indigenous estimates, an Indigenous-to-national rate ratio was applied to national rates, based on analysis of hospitalisations based on NNDSS for moderate cases and hospitalisations for severe cases.
Diphtheria	A	A	. .	. .	Estimates were based on the number of NNDSS notifications for diphtheria, which is likely to capture all cases of toxigenic diphtheria. This was not estimated in the Indigenous population as there were no notifications.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Infectious diseases (continued)					
Pertussis	C	C	D	C	Estimates were based on notifications for pertussis. Age distribution was based on GP encounters for pertussis in BEACH data. Severe cases were identified using hospitalisations for pertussis. Moderate transformations were required to overcome known data gaps. Indigenous estimates were based on hospitalisations for pertussis with the same severity distribution as national estimates.
Tetanus	A	A	. .	. .	Estimates were based on the number of NNDSS notifications for tetanus. Estimates were based on the number of NNDSS notifications for tetanus. This was not estimated in the Indigenous population as there were no notifications.
Measles	A	A	A	A	Estimates were based on notifications to the NNDSS, which are thought to be good estimates of the incidence of measles. Severe cases were identified through hospitalisations. Indigenous estimates were based on notifications averaged over 3 years and Indigenous-to-national hospitalisation ratios.
Rubella	C	D	D	C	Estimates were based on notifications to the NNDSS, which is known to have issues with under-notification. Indigenous estimates were based on notifications averaged over 3 years.
Varicella-zoster	C	C	C	C	Estimates were based on GP encounters reported in the BEACH data. Moderate transformations were required to overcome known data gaps for varicella-zoster and post-herpetic neuralgia. Indigenous estimates were based on Indigenous-to-national rate ratios from NNDSS notification rates.
Haemophilus influenzae type b	A	A	. .	. .	Estimates were obtained using notifications to the NNDSS, which is thought to capture all cases of haemophilus influenzae type b. No notifications were identified as Indigenous.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Infectious diseases (continued)					
Pneumococcal disease	A	A	A	B	Estimates were obtained using notifications to the NNDSS, which provides a good prevalence estimate of invasive pneumococcal disease. Minimal modelling was required. Indigenous estimates were based on notifications averaged over 2009–2011. Some modelling was required to overcome variability in age.
Meningococcal disease	A	A	A	A	Estimates were obtained using notifications to the NNDSS, which are thought to capture most severe cases.
Other meningitis and encephalitis	A	A	A	A	Person-to-separations ratios derived from Western Australian linked hospitalisations data were applied to unlinked hospitalisation data to estimate prevalence. Indigenous estimates were also obtained from hospitalisation data.
Dengue	B	B	C	B	Estimates were obtained using notifications to the NNDSS and hospitalisations. Hospitalisations were adjusted using persons-to-separations ratios. Indigenous estimates were based on notifications from selected states.
Ross River virus	C	D	D	C	Estimates were obtained using notifications to the NNDSS (which has issues with both false positivity and under-notificatication) and hospitalisations. Indigenous estimates were based on notifications from selected states and territories.
Barmah Forest virus	C	D	D	C	Estimates were obtained using notifications to the NNDSS, which has issues with both false positivity and under-notification. Indigenous estimates were based on notifications from selected states and territories.
Malaria	B	B	A	B	Estimates were obtained using notifications to the NNDSS and hospitalisations. Indigenous estimates were based on notifications from all states and territories.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Infectious diseases (continued)					
Trachoma	D	D	D	D	Indigenous prevalence estimates were based on estimates from the National Indigenous Eye Health Survey. Moderate transformations were required to determine age, sex and severity distributions using National Indigenous Eye Health Survey data, ABDS 2003 estimates, and expert advice.
Campylo-bacteriosis	B	B	B	C	Total incidence was based on estimates produced by Kirk et al. 2014. Some transformations were required to fill gaps in age, sex and severity using NNDSS notifications and hospitalisations. Indigenous estimates were based on hospitalisations for severe cases, and then the same relative proportions of mild–severe and moderate–severe were applied.
Salmonellosis	B	B	B	C	Total incidence was based on estimates produced by Kirk et al. 2014. Some transformations were required to fill gaps in age, sex and severity using NNDSS notifications and hospitalisations. Indigenous estimates were based on hospitalisations for severe cases, and then the same relative proportions of mild–severe and moderate–severe were applied.
Rotavirus	B	C	C	C	Total incidence was based on estimates produced by Kirk et al. 2014. Moderate transformations were required to fill gaps in age and sex using New South Wales notifications, and in severity distributions using hospitalisations adjusted using Western Australian person-to-separations ratios. Indigenous estimates were based on hospitalisations for severe cases, and then the relative proportions of mild–severe and moderate–severe for national estimates were applied.
Other gastrointestinal infections	B	D	D	C	Total incidence was based on estimates produced by Kirk et al. 2014, with age and sex distribution based on results from the National Gastroenteritis Survey II. The proportion of severe cases was estimated using the NHMD. Mild and moderate severity distributions were based on those for other gastrointestinal diseases (excluding hepatitis A). Indigenous estimates were based on hospitalisations for severe cases, and then the same relative proportions of mild–severe and moderate–severe were applied.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Injuries					
Traumatic brain injury	A	D	A	E	Short-term prevalence was estimated from the NHMD, and adjusted to account for non-admitted cases, based on estimates from NNPEDCD. Severity distribution was obtained from the GBD 2013. Long-term estimates were modelled in DISMOD II using NZBDS parameters for the probability of these injuries having long-term consequences, annual remission and excess mortality (see NZMOH 2013). Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors. Long-term estimates were derived from national estimates according to age and sex patterns of short-term admitted cases.
Spinal cord injury	A	D	A	E	Short-term prevalence was estimated from the NHMD, and adjusted to account for non-admitted cases, based on estimates from NNPEDCD. Severity distribution was obtained from the GBD 2013. Long-term estimates were modelled in DISMOD II using NZBDS parameters for the probability of these injuries having long-term consequences, annual remission and excess mortality (see NZMOH 2013). Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors. Long-term estimates were derived from national estimates according to age and sex patterns of short-term admitted cases.
Internal and crush injury	A	B	A	C	Short-term prevalence was estimated from the NHMD, and adjusted to account for non-admitted cases, based on estimates from NNPEDCD. Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors.
Poisoning	A	D	A	E	Short-term prevalence was estimated from the NHMD, and adjusted to account for non-admitted cases, based on estimates from NNPEDCD. Severity distribution was obtained from GBD 2013. Long-term estimates were modelled in DISMOD II using NZBDS parameters for the probability of these injuries having long-term consequences, annual remission and excess mortality (see NZMOH 2013). Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors. Long-term estimates were derived from national estimates according to age and sex patterns of short-term admitted cases.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Injuries (continued)					
Drowning and submersion injuries	A	D	A	E	Short-term prevalence was estimated from the NHMD, and adjusted to account for non-admitted cases based on estimates from NNPEDCD. Severity distribution was obtained from GBD 2013. Long-term estimates were modelled in DISMOD II using NZBDS parameters for the probability of these injuries having long-term consequences, annual remission and excess mortality (see NZMOH 2013). Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors. Long-term estimates were derived from national estimates according to age and sex patterns of short-term admitted cases.
Hip fracture	A	C	A	C	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from NNPEDCD. Severity distribution was obtained from GBD 2013. Long-term estimates were modelled in DISMOD II using NZBDS parameters for the probability of these injuries having long-term consequences, annual remission and excess mortality (see NZMOH 2013). Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors. Long-term estimates were derived from national estimates according to age and sex patterns of short-term admitted cases.
Tibia and ankle fracture	C	B	C	C	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from NNPEDCD. Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors.
Humerus fracture	B	B	B	C	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from NNPEDCD. Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Injuries (continued)					
Other fractures	B	C	A	D	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from NNPEDCD. Severity distribution was obtained from GBD 2013. Long-term estimates were modelled in DISMOD II using NZBDS parameters for the probability of these injuries having long-term consequences, annual remission and excess mortality (see NZMOH 2013). Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors. Long-term estimates were derived from national estimates according to age and sex patterns of short-term admitted cases.
Dislocations	C	B	C	C	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from NNPEDCD. Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors.
Soft tissue injuries	C	B	C	C	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from NNPEDCD.
Burn injuries	A	C	B	D	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from NNPEDCD. Severity distribution was obtained from GBD 2013. Long-term estimates were modelled in DISMOD II using NZBDS parameters for the probability of these injuries having long-term consequences, annual remission and excess mortality (see NZMOH 2013). Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors. Long-term estimates were derived from national estimates according to age and sex patterns of short-term admitted cases.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Injuries (continued)					
Other injuries	A	C	A	E	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from NNPEDCD. Severity distribution was obtained from GBD 2013. Long-term estimates were modelled in DISMOD II using NZBDS parameters for the probability of these injuries having long-term consequences, annual remission and excess mortality (see NZMOH 2013). Indigenous estimates for short-term sequelae were adjusted for under-identification using validated adjustment factors. Long-term estimates were derived from national estimates according to age and sex patterns of short-term admitted cases.
Kidney and urinary diseases					
Chronic kidney disease	A	B	A	B	National prevalence estimates were obtained from the Australia and New Zealand Dialysis and Transplant Registry and the AHS 2011–12. Indigenous prevalence estimates were obtained from the Australia and New Zealand Dialysis and Transplant Registry and the AATSIHS 2012–13. Moderate transformations of the AHS and AATSIHS data were required to obtain age- and sex-specific distributions using the NHMD, and the severity distribution was adjusted based on those used by the GBD 2013. No transformation of data from the Australia and New Zealand Dialysis and Transplant Registry was required.
Enlarged prostate	B	B	B	B	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation for cases with sufficient severity. Person-to-separations ratios derived from Western Australian linked hospitalisations data were applied to unlinked hospitalisation data to estimate prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Kidney stones	A	A	A	A	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation for cases with sufficient severity. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Mental and substance use disorders					
Depressive disorders	B	C	D	C	National prevalence estimates were obtained from the 2007 National Survey of Mental Health and Wellbeing and the 2013–14 Young Minds Matter Survey, which used diagnostic criteria to assess for mental health conditions. Severity was not directly available, and was based on those used by the GBD 2013. Moderate transformations were required to overcome gaps in age distribution, and to adjust to point prevalence. Indigenous prevalence estimates were based on rate ratios comparing Indigenous and total Australian rates of people diagnosed with depressive disorders accessing community mental health and inpatient services in Queensland. These were then applied to national prevalence rates. National severity distributions were applied.
Anxiety disorders	B	C	D	C	National prevalence estimates were obtained from the 2007 National Survey of Mental Health and Wellbeing and the 2013–14 Young Minds Matter Survey, which used diagnostic criteria to assess for mental health conditions. Severity was not directly available, and was based on those used by GBD 2013. Moderate transformations were required to overcome gaps in age distribution, and to adjust to point prevalence. Indigenous prevalence estimates were based on rate ratios comparing Indigenous and total Australian rates of people diagnosed with anxiety disorders accessing community mental health and inpatient services in Queensland. These were then applied to national prevalence rates. National severity distributions were applied.
Bipolar affective disorder	B	C	D	C	National prevalence estimates were obtained from the 2007 National Survey of Mental Health and Wellbeing and the 2013–14 Young Minds Matter Survey, which used diagnostic criteria to assess for mental health conditions. Severity was not directly available, and was based on those used by GBD 2013. Moderate transformations were required to overcome gaps in age distribution, and to adjust to point prevalence. Indigenous prevalence estimates were based on rate ratios comparing Indigenous and total Australian rates of people diagnosed with bipolar affective disorder accessing community mental health and inpatient services in Queensland. These were then applied to national prevalence rates. National severity distributions were applied.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Mental and substance use disorders (continued)					
Alcohol use disorders	B	B	D	C	National prevalence estimates were obtained from the 2007 National Survey of Mental Health and Wellbeing and the 2013–14 Young Minds Matter Survey, which used diagnostic criteria to assess for mental health conditions. Severity was partially available from the study, partially from GBD 2013. Some transformations were required to overcome gaps in age distribution. Indigenous prevalence estimates were based on rate ratios comparing Indigenous and total Australian rates of people diagnosed with alcohol use disorders accessing community mental health and inpatient services in Queensland (moderate–severe), and on rate ratios from national hospitalisation data (asymptomatic–mild), These were then applied to national prevalence rates.
Drug use disorders (excluding alcohol)	C	C	D	C	National estimates were obtained from a variety of sources depending on the drug. These varied in quality from cannabis dependence estimates—which were based on the 2007 National Survey of Mental Health and Wellbeing—to estimates of cocaine—which were based on proxy measures. All severity distributions were from the GBD 2013. Moderate transformations were required to overcome data gaps. Indigenous estimates were obtained by applying rate ratios to national prevalence rates for all levels of severity. These were largely based on comparing Indigenous and total Australian rates of people diagnosed with the relevant drug use disorder accessing community mental health and inpatient services in Queensland, or hospitalisation rate ratios. For cocaine dependence, rate ratios from self-reported data from the 2013 NDSHS were used.
Schizophrenia	B	C	D	C	National estimates were obtained from the Survey of High Impact Psychosis (2010), which was based on clinical diagnosis. Moderate transformations were required to overcome data gaps. Health state distribution was based on those used in GBD 2013. Indigenous estimates were obtained by applying rate ratios to national prevalence rates, based on comparing Indigenous and total Australian rates of people diagnosed with schizophrenia accessing community mental health and inpatient services in Queensland.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Mental and substance use disorders (continued)					
Eating disorders	D	B	E	D	National estimates were obtained from the GBD 2010 estimated prevalence for Australia and an epidemiological study conducted in New Zealand. Some modelling was required, particularly for bulimia nervosa due to large age groups. Indigenous prevalence estimates for eating disorders were assumed to be the same rate as the total population prevalence, based on expert advice.
Autism spectrum disorders	D	E	E	E	National estimates were based on prevalence in Western Australia, and were based on administrative data that would reasonably capture most cases of childhood autism. Estimates of other autism spectrum disorders were based on childhood autism-to-other autism spectrum disorders ratio observed in GBD 2013. Substantial transformations were required to overcome data gaps. Due to lack of available data, prevalence of autism spectrum disorders in the Indigenous population is unknown. As such, total population prevalence was assumed for the Indigenous population, based on expert advice. Due to the low data and method rating, the Indigenous estimates for autism spectrum disorders have not been reported separately in the ABDS 2011.
Attention deficit hyperactivity disorder	B	C	E	C	National estimates were obtained from the 2013–14 Young Minds Matter survey, based on diagnostic criteria and GBD 2013. Moderate transformations were required to calculate prevalence. Indigenous estimates were obtained by applying a rate ratio to national prevalence rates, based on behavioural indicators for attention deficit hyperactivity disorder in the Longitudinal Study of Australian Children and attention deficit hyperactivity disorder diagnosis from community and inpatient service use in Queensland.
Conduct disorder	B	C	E	C	Estimates were obtained from the 2013–14 Young Minds Matter survey based on diagnostic criteria and GBD 2013. Moderate transformations were required to calculate prevalence. Indigenous estimates were obtained by applying a rate ratio to national prevalence rates, which were based on behavioural indicators for attention deficit hyperactivity disorder in the Longitudinal Study of Australian Children, and conduct disorder diagnosis from community and inpatient service use in Queensland.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Mental and substance use disorders (continued)					
Intellectual disability	D	E	D	D	National estimates were based on prevalence in Western Australia, and on administrative data that would reasonably capture most cases of intellectual disability. Severity distribution was based on an international meta-analysis. Substantial transformations were required to overcome data gaps. Indigenous estimates were obtained by applying a rate ratio to national estimates, and adjusting for differences in severity (this rate ratio was based on the same key data source used for national estimates—the IDEA database).
Other mental and substance use disorders	C	D	D	D	Estimates were based on a hospitalisation-to-prevalence rate ratio for diseases with a mix of low and medium likelihood of hospitalisation that corresponded to the types of diseases in this residual category. Considerable transformations were required to estimate prevalence. The same approach was used for Indigenous estimates. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Musculoskeletal conditions					
Osteoarthritis	B	A	B	B	National prevalence estimates were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Severity distributions were obtained from the same data source. Minimal transformations were required to overcome variability caused by small numbers in some age groups.
Gout	B	A	B	B	National prevalence estimates were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Minimal transformations were required to overcome variability caused by small numbers in some age groups.
Rheumatoid arthritis	B	A	B	B	National prevalence estimates were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Severity distributions were obtained from the same data source. Minimal transformations were required to overcome variability caused by small numbers in some age groups.

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Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Musculoskeletal conditions (continued)					
Back pain and problems	B	A	B	B	National prevalence estimates were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Severity distributions were obtained from the same data source. Minimal transformations were required to overcome variability caused by small numbers in some age groups.
Other musculoskeletal	B	A	B	B	National prevalence estimates were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Severity distributions were obtained from the same data source. Minimal transformations were required to overcome variability caused by small numbers in some age groups.
Neurological conditions					
Epilepsy	B	C	B	C	National prevalence estimates were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Severity distributions were obtained from Forsgren et al. 2005. Person-to-separations ratios derived from Western Australian linked hospitalisations and deaths data were applied to unlinked hospitalisation data to overcome variability in the data source caused by small numbers.
Dementia	C	C	D	D	National prevalence estimates were obtained from AIHW 2012b. Severity distribution was obtained from Lucca et al. 2015 and Barendregt et al. 1998. Moderate transformations were required to produce prevalence estimates by severity. Indigenous prevalence estimates were obtained from 2 small Australian studies (Radford et al. 2015; Smith et al. 2008). Severity distribution was obtained from the study by Radford et al. (2015) and from Barendregt et al. (1998). Considerable transformations were required to derive prevalence estimates by severity.
Parkinson disease	E	C	E	C	National and Indigenous prevalence estimates were obtained from 2 international studies (de Rijk et al. 2000; Willis et al. 2013). Severity distribution was obtained from unpublished data from the Queensland Parkinson's Project. Due to the low data rating, the estimates must be interpreted with caution.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Neurological conditions (continued)					
Multiple sclerosis	C	B	D	C	National and Indigenous prevalence estimates were obtained from an Australian study (Palmer et al. 2013) with medium currency. Severity was obtained from another Australian study (Covance Pty Ltd & Palmer 2011). These data sources were not Indigenous-specific. Some transformations were required to overcome variability in sampling.
Motor neurone disease	B	B	B	B	National and Indigenous prevalence estimates were obtained from the NHMD with a moderate likelihood of hospitalisation. Person-to-separations ratios derived from Western Australian linked hospitalisations and deaths data were applied to unlinked hospitalisation data to estimate prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. National distributions were used to transform estimates into prevalence for some sequelae for the Indigenous population.
Migraine	B	B	B	B	National prevalence estimates were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Some transformations were required to produce point prevalence estimates using durations obtained from the NZBDS, and to overcome variability in the data source caused by small numbers.
Guillain-Barré syndrome	B	B	B	B	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Person: separations ratios derived from Western Australian linked hospitalisations and deaths data were applied to unlinked hospitalisation data to estimate prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. National distributions were used to transform estimates into prevalence for some sequelae for the Indigenous population.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Oral disorders					
Dental caries	D	C	D	D	National estimates were obtained from the National Survey of Adult Oral Health 2004–06 for adults, and the 2009 Child Dental Health Survey for children. Both were clinical surveys of oral health, but the National Survey of Adult Oral Health had low currency for a condition that can change in prevalence over time. Moderate transformations were required to overcome gaps in age distribution, and prevalence was not adjusted for change over time. For Indigenous estimates, an Indigenous-to-national rate ratio from the National Survey of Adult Oral Health and Child Dental Health Survey was applied to national rates.
Periodontal disease	D	C	D	D	National estimates were obtained from the National Survey of Adult Oral Health 2004–06. Periodontal disease was not estimated in children. This was a clinical survey, but had low currency. Some transformations were required to overcome gaps in age distribution, and prevalence was not adjusted for change over time. For Indigenous estimates, an Indigenous-to-national rate ratio from the National Survey of Adult Oral Health was applied to national rates.
Severe tooth loss	D	C	C	C	National estimates were obtained from the National Survey of Adult Oral Health 2004–06 including self-reported edentulism. Moderate transformations were required to overcome gaps in age distribution, and prevalence was not adjusted for change over time. Indigenous estimates were based on self-reported severe tooth loss in 2011 AATISHS. These data were current, but the condition was self-reported, requiring a respondent to know how many teeth they had lost. Moderate transformations were required to overcome gaps in age distribution.
Other oral disorders	C	B	C	B	National and Indigenous estimates were based on hospitalisations, which has medium likelihood of hospitalisation. Some transformations were required to overcome gaps in age/sex distribution.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Reproductive and maternal conditions (continued)					
Maternal haemorrhage	A	A	A	A	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Maternal infections	A	A	A	A	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Hypertensive disorders of pregnancy	A	A	A	A	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Obstructed labour	A	A	A	A	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Early pregnancy loss	B	B	B	C	National and Indigenous prevalence estimates for both sequelae were obtained from the NHMD with a high likelihood of hospitalisation for ectopic pregnancy. Estimates for early pregnancy losses occurring outside hospital were calculated from Medicare data, and adjusted for unclaimed procedures using an Australian study (Nickson et al. 2004). Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. Medicare data were adjusted based on an Indigenous-to-national rate ratio for early pregnancy losses using the NHMD, and adjusted for unclaimed procedures using the same study as for national estimates.
Gestational diabetes	A	B	A	B	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of being detected at hospital. Person-to-separations ratios derived from Western Australian linked hospitalisations and deaths data were applied to unlinked hospitalisation data to estimate prevalence. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. National distributions were used to transform estimates into prevalence for some sequelae for the Indigenous population.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Reproductive and maternal conditions (continued)					
Endometriosis	C	C	D	D	Overall national estimates were derived from the Australian Longitudinal Study on Women's Health, and transformed using BEACH data. Overall Indigenous estimates were based on an Indigenous-to-national rate ratio for hospitalised cases, applied to the national prevalence rate. National and Indigenous prevalence estimates for severe cases were obtained from the NHMD with a high likelihood of hospitalisation. Estimates were subtracted from the total prevalence to derive remaining sequelae. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. Infertility sequela for the national population was derived from the Australian Longitudinal Study on Women's Health, and the same rate was used for Indigenous estimates.
Uterine fibroids	C	C	C	D	National and Indigenous prevalence estimates for symptomatic uterine fibroids were obtained from the NHMD with a high likelihood of hospitalisation. Severity was based on type of surgical procedures. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. Anaemia sequela was based on an international study (Zimmerman et al. 2012), and the same rate was used for Indigenous estimates. Severity distribution was the same as used for iron-deficiency anaemia. Infertility sequela was based on international estimates (Khaund & Lumsden 2008), and the same rate was used for Indigenous estimates.
Genital prolapse	D	C	E	D	National prevalence estimates for females were obtained from 2 international studies (Tegerstedt et al. 2005; Jackson et al. 1997), and the same rate was used for Indigenous estimates. Moderate transformations were required to derive age-specific prevalence. Male prevalence estimates were calculated using the ratio of male and female genital prolapse hospitalisations from the NHMD. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Reproductive and maternal conditions (continued)					
Polycystic ovarian syndrome	B	C	D	D	National prevalence estimates were derived from the Australian Longitudinal Study of Women's Health, and transformed using BEACH data. Indigenous estimates were based on an Indigenous-to-national rate ratio for hospitalised cases, applied to the national prevalence rate. National and Indigenous prevalence estimates for severe cases were obtained from the NHMD with a high likelihood of hospitalisation. Estimates were removed from the total prevalence to derive remaining sequelae. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. Infertility sequela was derived from the Australian Longitudinal Study of Women's Health, and the same rate was used for Indigenous estimates.
Infertility	D	D	E	E	National prevalence estimates were based on Australian and New Zealand Assisted Reproduction Database estimates, with extensive transformations using information from the database's annual report, BEACH data and an Australian study (Marino et al. 2011). Infertility sequela found in other diseases were subtracted from the infertility envelope to avoid double-counting. Indigenous estimates were based on an Indigenous-to-national rate ratio for hospitalised cases, applied to the national prevalence rate.
Other maternal conditions	A	A	A	A	National and Indigenous prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Other reproductive conditions	C	C	D	D	National prevalence estimates, by sequela, were based on BEACH data, with moderate transformations using population data. Indigenous estimates were assumed to be the same as the national prevalence rate.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Respiratory diseases					
Asthma	B	B	B	B	National prevalence estimates were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Severity distributions were obtained from an Australian web-based survey (Reddel 2015). Some transformations were required to overcome variability caused by small numbers.
Chronic obstructive pulmonary disease	B	B	B	B	National prevalence estimates were based on measured data from a study specific to the Australian population—the Burden of Obstructive Lung Disease (BOLD) study (Toelle 2013). Severity distributions were also obtained from the BOLD study, together with expert advice. Some transformations were required to overcome variation caused by small numbers. Indigenous prevalence estimates and severity distributions were based on measure data from a small cross-sectional BOLD study undertaken in the Kimberly region (Cooksley et al. 2015). Some transformations using the NHMD, were required to obtain age and sex distributions, to overcome variations caused by small numbers.
Sarcoidosis	D	C	D	C	National and Indigenous prevalence estimates were obtained from the NMD and the NHMD. As these data sources only captured moderate/severe cases, transformations were required to fill gaps due to low data specificity. Person-to-separations ratios derived from Western Australian linked hospitalisations and deaths data were applied to unlinked hospitalisation data to estimate prevalence. Validated adjustment factors for the NMD and NHMD were used to adjust for Indigenous under-identification in these data collections.
Interstitial lung disease	C	D	C	D	National and Indigenous prevalence estimates were obtained from the NMD and the NHMD. As these data sources only captured moderate/severe cases, transformations were required to fill gaps due to low data specificity. Person-to-separations ratios derived from Western Australian linked hospitalisations and deaths data were applied to unlinked hospitalisation data to estimate prevalence. Validated adjustment factors for the NMD and NHMD were used to adjust for Indigenous under-identification in these data collections.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Respiratory diseases (continued)					
Pneumoconiosis	D	C	. .	. .	National prevalence estimates were obtained from the NMD and the NHMD. As these data sources only captured moderate/severe cases, transformations were required to fill gaps due to low data specificity. Person-to-separations ratios derived from Western Australian linked hospitalisations and deaths data were applied to unlinked hospitalisation data to estimate prevalence. Prevalence of pneumoconiosis was assumed absent in the Indigenous population, based on expert advice.
Upper respiratory conditions	B	A	B	A	National prevalence estimates were obtained from the AHS 2011–12. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. No additional transformations were required outside of applying a duration to the incidence to derive prevalence.
Other respiratory disease	E	E	E	E	National and Indigenous prevalence estimates were obtained from the NHMD with mixed likelihood of hospitalisation. Substantial transformations were required to fill data gaps. Due to the low data and method ratings, the estimates must be interpreted with caution.
Skin disorders					
Dermatitis and eczema	D	C	E	C	National prevalence estimates were obtained from an older small area Australian study (Plunkett et al. 1999). Severity distributions for children were obtained from Marks et al. 1999a. Overall Indigenous prevalence was based on national prevalence applied to the Indigenous Australian population. Due to the low data rating, the Indigenous estimate must be interpreted with caution
Psoriasis	D	B	D	B	National prevalence estimates were obtained from the 2011–12 AHS. Indigenous prevalence estimates were obtained from the AATSIHS 2012–13. Severity distributions were obtained from a small Australian study (Jenner et al. 2002). Some transformations were required to overcome variability in the data source caused by small numbers.

(continued)

Table F3 (continued): National and Indigenous YLD quality ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Skin disorders (continued)					
Acne	D	C	E	C	National prevalence estimates and severity distributions were obtained from older small area Australian studies (Kilkenny et al. 1998; Marks et al. 1999b; Plunkett et al. 1999). Overall Indigenous prevalence was based on national prevalence applied to the Indigenous Australian population. Due to the low data rating, the Indigenous estimate must be interpreted with caution.
Ulcers	C	D	D	D	National prevalence estimates and severity distributions were obtained from the NHMD, and Australian and international studies (Asimus & Li 2011; Dealey et al. 2012; Mulligan et al. 2011; Queensland Health 2012; Santamaria et al. 2009; SA Health 2007; VQC 2006). Substantial transformations were required to overcome data gaps. Overall Indigenous prevalence was derived from national prevalence and Indigenous-to-national ratios for ulcers from the NHMD. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification.
Skin infections (including cellulitis)	C	B	C	B	National and Indigenous prevalence estimates were obtained from the NHMD with a moderate likelihood of hospitalisation. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. Some transformations were required to overcome variability in the data source caused by small numbers.
Other skin disorders	D	D	D	D	National prevalence estimates were obtained from the NHMD and AHS 2011–12. Indigenous prevalence estimates were obtained from the NHMD and AATSIHS 2012–13. Validated adjustment factors for the NHMD were used to adjust for Indigenous under-identification. Substantial transformations were required to fill data gaps resulting from poor data specificity.

A: Highly relevant/accurate—estimate was derived from comprehensive and highly relevant data/little or no data transformation was required.

B: Relevant/accurate.

C: Moderately relevant/accurate—estimate was derived from reasonably comprehensive and relevant data/moderate transformations required, taking into account known trends in the underlying data, such as over time or age-distributions.

D: Somewhat relevant/accurate.

E: Questionable relevance/accuracy—use with caution, as estimate was derived from less comprehensive or relevant data/moderate transformations required with trends unknown or unaccounted for.

Table F4: National and Indigenous risk factor ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Behavioural					
Tobacco use	A	A	B	B	National exposure estimates were obtained from the NDSHS 2007 to include a 5-year lag. Lung cancer mortality rates were based on national cancer incidence and mortality data. Indigenous exposure estimates were obtained from the NATSISS 2008 to include a 5-year lag. Lung cancer mortality rate was based on 4 states only.
Alcohol use	B	A	C	A	National exposure estimates were obtained from the NDSHS 2011. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. The NDSHS and AATSIHS were adjusted for under-reporting of alcohol consumption by estimates of alcohol available for sale in Australia.
Physical inactivity	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Drug use	B	C	B	C	Direct evidence for injecting drug use was obtained from the Kirby Institute. Burden attributable to other types of drug use were based on 'Drug use disorders' described in Chapter 7. The same method was used for national and Indigenous estimates.
Intimate partner violence	A	B	D	D	National exposure estimates were obtained from the Personal Safety Survey 2012 adjusted to account for ages 85 and over. Indigenous exposure estimates were derived from national exposure estimates using an Indigenous-to-national rate ratio derived from the 2008 NATSISS and the 2006 General Social Survey (AIHW 2015a).
Unsafe sex	A	A	A	A	Direct evidence was obtained from the Kirby Institute for both national and Indigenous exposure estimates.
Childhood sexual abuse	C	B	D	D	National exposure estimates were obtained from the Personal Safety Survey 2012; however this data source was not designed to estimate exposure to childhood sexual abuse in Australia. Estimates were adjusted to account for ages 85 and over. For Indigenous exposure, an Indigenous-to-total Australians reporting victims of sexual assault rate ratio from ABS recorded crime data was applied to national exposure estimates.

(continued)

Table F4 (continued): National and Indigenous risk factor ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Metabolic risks					
High body mass	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
High blood pressure	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
High blood plasma glucose	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
High cholesterol	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Iron deficiency	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Low bone mineral density	D	B	D	B	National exposure was modelled from data from the Geelong Osteoporosis Study using established modelling methods. Indigenous exposure was assumed to be the same as national exposure, based on self-reported prevalence of osteoporosis from the 2011–12 AHS and AATSIHS 2012–13.
Environmental					
Occupational exposure and hazards	A	C	A	C	Exposure to occupations and industries were derived from the Census of Population and Housing 2011 and the ABS Labour Force Survey (June 2011). The occupations from the Census were aligned with international standard classification of occupations using concordance files.
Occupational injuries	A	C	C	C	National exposure estimates were obtained from the Workers' Compensation Statistics 2010–11 (Safe Work Australia 2013) and Work-related Traumatic Injury Fatalities, Australia 2010–11 (Safe Work Australia 2012). Indigenous exposure estimates were obtained by applying rate ratios from the NHMD. Injuries from these sources were mapped to injury causes in this study.

(continued)

Table F4 (continued): National and Indigenous risk factor ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Environmental (continued)					
High sun exposure	n.a.	n.a.	. .	. .	Information on the quality of these estimates will be published in an upcoming paper by the above authors.
Air pollution	D	C	D	C	Exposure estimates were from state/territory air monitoring stations. Australian population data were used to estimate the proportion of people exposed in the total Australian population and in the Indigenous population.
Unimproved sanitation	. .	. .	A	B	Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Dietary risk factors					
Diet low in fruit	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Diet low in vegetables	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Diet high in processed meat	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Diet low in nuts and seeds	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Diet low in whole grains	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Diet low in omega 3 fatty acids	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.

(continued)

Table F4 (continued): National and Indigenous risk factor ratings

	National		Indigenous		
Disease	**Data**	**Methods**	**Data**	**Methods**	**Statement**
Dietary risk factors (continued)					
Diet high in sweetened beverages	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Diet high in sodium	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Diet low in milk	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Diet high in red meat	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.
Diet low in calcium	A	B	A	B	National exposure estimates were obtained from the 2011–12 AHS. Indigenous exposure estimates were obtained from the AATSIHS 2012–13. Estimates were adjusted to account for ages 85 and over.

A: Highly relevant/accurate—estimate was derived from comprehensive and highly relevant data/little or no data transformation was required.

B: Relevant/accurate.

C: Moderately relevant/accurate—estimate was derived from reasonably comprehensive and relevant data/moderate transformations required, taking into account known trends in the underlying data, such as over time or age-distributions.

D: Somewhat relevant/accurate.

E: Questionable relevance/accuracy—use with caution, as estimate was derived from less comprehensive or relevant data/moderate transformations required with trends unknown or unaccounted for.

Appendix G: List of contributors

Table G1: List of ABDS 2011 Project Governance Committee members

Member	Organisation
Andrew Kettle (Chair)	Australian Institute of Health and Welfare
David Kalisch (Chair)	Australian Institute of Health and Welfare
Fadwa Al-Yaman	Australian Institute of Health and Welfare
Bobbie Campbell	Australian Government Department of Health
Ian Crettenden	Australian Government Department of Health
David Cullen	Australian Government Department of Health
Teresa Dickinson	Australian Institute of Health and Welfare
Elizabeth Flynn	Australian Government Department of Health
Sally Goodspeed	Australian Government Department of Health
Paul Jelfs	Australian Bureau of Statistics
Maria Jolly	Australian Government Department of Health
Danielle Klar	Australian National Preventive Health Agency
Lisa McGlynn	Australian Institute of Health and Welfare
Lynelle Moon	Australian Institute of Health and Welfare
Samantha Palmer	Australian Government Department of Health
Jack Quinane	Australian National Preventive Health Agency
Lisa Studdert	Australian National Preventive Health Agency
Bernie Towler	Australian Government Department of Health

Table G2: List of ABDS 2011 Expert Advisory Group members

Member	Organisation
Ching Choi (Chair)	University of New South Wales
Fadwa Al-Yaman	Australian Institute of Health and Welfare
Emily Banks	Australian National University
Anthony Barnes	Independent consultant
Justine Boland	Australian Bureau of Statistics
Annette Dobson	University of Queensland
Ian Crettenden	Australian Government Department of Health
David Cullen	Australian Government Department of Health
Tim Driscoll	University of Sydney
Louisa Jorm	University of New South Wales
Paul Kelly	ACT Health
Siew-Ean Khoo	Australian National University
John Lynch	University of Adelaide
Michelle Marquardt	Australian Bureau of Statistics
Lisa McGlynn	Australian Institute of Health and Welfare
Lynelle Moon	Australian Institute of Health and Welfare
David Roder	University of South Australia
Colin Sindall	Department of Health Victoria
Peter Somerford	Department of Health Western Australia
Bernie Towler	Australian Government Department of Health
Harvey Whiteford	University of Queensland
Jeanette Young	Queensland Health
Project support	
Sonya Glasson	Australian Government Department of Health
Holly Jones	Australian Government Department of Health

Table G3: List of ABDS 2011 Indigenous Reference Group members

Member	Organisation
Len Smith (Chair)	Australian National University
Jason Agostino	National Aboriginal Community Controlled Health Organisation
Anthony Barnes	Independent consultant
Alex Brown	South Australian Health and Medical Research Institute
Daniel Christensen	Telethon Kids Institute
Steve Guthridge	Department of Health Northern Territory
Kirrily Harrison	Australian Government Department of the Prime Minister and Cabinet
Wendy Hoy	University of Queensland
April Lawrie-Smith	Department of Health South Australia
Vanessa Lee	University of Sydney
Julie Nankervis	Australian Bureau of Statistics
Hope Peisley	Australian Government Department of Health
Debra Reid	Independent consultant
Shahidullah	Australian Bureau of Statistics
Fiona Shalley	Australian Bureau of Statistics
Rob Starling	National Aboriginal Community Controlled Health Organisation
Daniel Willamson	Department of Health Queensland
Yuejen Zhao	Department of Health Northern Territory
Project support	
Alice Church	Australian Government Department of Health

Table G4: List of ABDS 2011 disease-specific contributors

Expert (group or person)	Organisation
Blood and metabolic disorders	
Assoc. Prof. Scott Bell	The Prince Charles Hospital, University of Queensland
Prof. Amanda Lee	Queensland University of Technology
Dr Simon McRae	Royal Adelaide Hospital; The Queen Elizabeth Hospital
Dr John Rowell	Royal Brisbane and Women's Hospital
Cancer and other neoplasms	
Cancer and Screening Unit	Australian Institute of Health and Welfare
Cancer Monitoring Advisory Group	Australian Institute of Health and Welfare advisory group
Prof. James Bishop AO	Victorian Comprehensive Cancer Centre
Dr Pamela Brown	Consultant dermatologist
Dr Keng Chen	Skin and Cancer Foundation
Assoc. Prof. Rosemary Knight	Australian Government Department of Health (former)
Prof. David Roder	University of South Australia
Dr Timothy Threlfall	Western Australian Cancer Registry
Prof. Christobel Saunders	Harry Perkins Institute of Medical Research
Dr Catherine Shannon	Mater Cancer Care Centre
Assoc. Prof. James St John AM	Cancer Council Victoria (retired)
Assoc. Prof. Chris Stephenson	Deakin University
Cardiovascular diseases	
Cardiovascular, Diabetes and Kidney Unit	Australian Institute of Health and Welfare
Cardiovascular Disease Expert Advisory Group—Andrew Tonkin (Chair), Tom Briffa, Derek Chew, Annette Dobson, John Lynch, Mandy Thrift	Australian Institute of Health and Welfare advisory group
Endocrine disorders	
Cardiovascular, Diabetes and Kidney Unit	Australian Institute of Health and Welfare
Diabetes Expert Advisory Group—Jonathan Shaw (Chair), Stephen Colagiuri, Maria Craig, Wendy Davis, Mark Harris, Greg Johnson, Glynis Ross, Sophia Zoungas	Australian Institute of Health and Welfare advisory group
Gastrointestinal disorders	
Prof. Jane Andrews	Royal Adelaide Hospital
Dr Paul Clark	University of Queensland
Clinical Assoc. Prof. Peter Katelaris	University of Sydney
Dr Suzanne Mahady	University of Sydney
Dr Stephen Williams	Westmead Hospital

(continued)

Table G4 (continued): List of ABDS 2011 disease-specific contributors

Expert (group or person)	Organisation
Hearing and vision disorders	
Office of Hearing Services	Australian Government Department of Health
Prof. Robert Cowan	University of Melbourne; Macquarie University; HEARing Cooperative Research Centre; HearWorks
Prof. Harvey Dillon	Australian Hearing; The HEARing Cooperative Research Centre
Prof. Louise Hickson	University of Queensland; Communication Disability Centre
Prof. Hugh Taylor	University of Melbourne
Infant and congenital conditions	
Maternal Health, Children, Youth and Families Unit	Australian Institute of Health and Welfare
Prof. Nadia Badawi	University of Sydney; Westmead Children's Hospital; Cerebral Palsy Alliance
Clinical Assoc. Prof. Gareth Baynam	Western Australian Department of Health; University of Western Australia
Prof. Carol Bower	Telethon Kids Institute
Dr Adrienne Gordon	University of Sydney
Dr Lisa Hilder	National Perinatal Epidemiology and Statistics Unit, University of New South Wales
Assoc. Prof. Alison Kent	Australian National University; The Canberra Hospital
Dr Karen Walker	Grace Centre for Newborn Care, University of Sydney
Infectious diseases	
Office of Health Protection	Australian Government Department of Health
Dr Frank Beard	National Centre for Immunisation, Research and Surveillance
Dr Paul Kelly	Australian Capital Territory Health
Assoc. Prof. Martyn Kirk	National Centre for Epidemiology and Population Health, Australian National University
Assoc. Prof. David Wilson	The Kirby Institute, University of New South Wales
Dr Jeannette Young	Queensland Health
Injuries	
Prof. James Harrison	Research Centre for Injury Studies, Flinders University
Dr Sophie Pointer	Research Centre for Injury Studies, Flinders University
Kidney and urinary diseases	
Cardiovascular, Diabetes and Kidney Unit	Australian Institute of Health and Welfare
Chronic Kidney Disease Expert Advisory Group—Tim Mathew (Chair), Alan Cass, Steven Chadban, Jeremy Chapman, Joan Cunningham, Bettina Douglas, Wendy Hoy, Stephen McDonald, David Parker	Australian Institute of Health and Welfare advisory group

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Table G4 (continued): List of ABDS 2011 disease-specific contributors

Expert (group or person)	Organisation
Mental and substance use disorders	
Mental Health and Palliative Care Unit	Australian Institute of Health and Welfare
Ms Jenny Bourke	Telethon Kids Institute
Prof. Louisa Degenhardt	National Drug and Alcohol Research Centre
Dr Alize Ferrari	University of Queensland
Prof. Wayne Hall	University of Queensland
Assoc. Prof. Helen Leonard	Telethon Kids Institute
Prof. John McGrath	University of Queensland
Prof. George Patton	Royal Children's Hospital Melbourne
Prof. Harvey Whiteford	University of Queensland
Musculoskeletal conditions	
Population Health and Primary Care Unit	Australian Institute of Health and Welfare
National Centre for Monitoring Arthritis and Other Musculoskeletal Conditions Advisory Group	Australian Institute of Health and Welfare advisory group
Prof. Chris Maher	University of Sydney
Prof. Lyn March	University of Sydney
Mr Matthew Montgomery	Australian Bureau of Statistics
Prof. Tania Winzenberg	Menzies Research Institute Tasmania, School of Medicine, University of Tasmania
Neurological conditions	
Disability and Ageing Unit	Australian Institute of Health and Welfare
Prof. Kaarin Anstey	Dementia Collaborative Research Centre—Early Diagnosis and Prevention, Australian National University
Prof. George Mellick	Griffith University
Prof. Matthew Kiernan	Brain and Mind Research Institute, University of Sydney
Prof. Andrew Palmer	University of Tasmania
Oral disorders	
Assoc. Prof. David Brennan	Australian Research Centre for Population Oral Health
Adjunct Assoc. Prof. Ratilal Lalloo	Australian Research Centre for Population Oral Health
Dr Liana Luzzi	Australian Research Centre for Population Oral Health
Prof. Marco Peres	Australian Research Centre for Population Oral Health
Dr John Rogers	Victorian Department of Health
Reproductive and maternal conditions	
Assoc. Prof. Georgina Chambers	National Perinatal Epidemiology and Statistics Unit, University of New South Wales
Prof. Caroline Homer	University of Technology, Sydney
Assoc. Prof. Michael Nicholl	University of Sydney; Maternal, Neonatal and Women's Health Network for Northern Sydney Local Health District
Prof. Jeremy Oats	University of Melbourne

(continued)

Table G4 (continued): List of ABDS 2011 disease-specific contributors

Expert (group or person)	Organisation
Respiratory diseases	
Australian Centre for Asthma Monitoring	Australian Institute of Health and Welfare collaborating centre
Prof. Tim Driscoll	University of Sydney
Prof. Guy Marks	Woolcock Institute of Medical Research, University of Sydney
Assoc. Prof. Helen Reddel	Woolcock Institute of Medical Research, University of Sydney
Skin disorders	
Dr Pamela Brown	Consultant dermatologist
Dr Keng Chen	Skin and Cancer Foundation
Dr Suzanne Kapp	La Trobe University
Dr Monique Kilkenny	Monash University
Dr Rosana Norman	Queensland University of Technology

Table G5: List of ABDS 2011 risk-specific contributors

Expert (group or person)	Organisation
Cardiovascular, Diabetes and Kidney Unit	Australian Institute of Health and Welfare
Tobacco, Alcohol and Other Drugs Unit	Australian Institute of Health and Welfare
Mr Paul Atyeo	Australian Bureau of Statistics
Ms Janis Baines	Food Standards Australia and New Zealand
Prof. Tim Driscoll	Sydney School of Public Health, University of Sydney
Ms Louise Gates	Australian Bureau of Statistics
Dr Ivan Hanigan	Australian National University
Prof. Amanda Lee	Queensland University of Technology
Prof. Robyn Lucas	National Centre for Epidemiology and Population Health, Australian National University
Ms Leanne Luong	Australian Bureau of Statistics
Assoc. Prof. Peter Somerford	Western Australian Department of Health
Dr Rosemary Stanton	Nutritionist consultant
Assoc. Prof. David Wilson	The Kirby Institute, University of New South Wales
Dr Fan Xiang	National Centre for Epidemiology and Population Health, Australian National University

Glossary

additional diagnosis: A condition or complaint either coexisting with the principal diagnosis, or arising during the episode of admitted patient care, episode of residential care, or attendance at a health-care establishment. METeOR identifier: 514271.

admitted patient: A patient who undergoes a hospital's admission process to receive treatment and/or care. This treatment and/or care is provided over a period of time, and can occur in hospital and/or in the person's home (for hospital-in-the-home patients). METeOR identifier: 268957.

age weighting: A method sometimes used to adjust the relative 'value' of years lived at different ages—for example, to value a year lived by a young adult more highly than a year lived at older ages. If applied, age weighting results in some age groups having an increased influence on the estimates of disease burden relative to other age groups.

age-standardisation: A set of techniques used to remove, as far as possible, the effects of differences in age when comparing 2 or more populations.

age-standardised rate: Rate that takes into account the age structure of the population.

attributable burden: The disease burden attributed to a particular risk factor. It is the reduction in fatal and non-fatal burden that would have occurred if exposure to the risk factor had been avoided or reduced to its **theoretical minimum risk exposure distribution**.

avoidable burden: The reduction in future burden that would occur if current and/or future exposure to a particular risk factor were avoided. Compare with **attributable burden**.

burden of disease (and injury): The quantified impact of a disease or injury on a population using the **disability-adjusted life year** (DALY) measure.

chronic: Persistent and long-lasting.

comorbidity: A health problem/disease that exists at the same times as (an)other health problem(s).

conceptual disease model: A representation of clinical conditions designed to summarise what is known about the disease epidemiology, the nature of the disease (that is, whether it is chronic, acute, episodic or progressive), and its treatment.

condition (health condition): A broad term that can be applied to any health problem, including symptoms, diseases and certain risk factors, such as high blood cholesterol and obesity. Often used synonymously with disorder or problem.

counterfactual: An alternative risk factor exposure distribution chosen for comparison with the observed distribution, to estimate the alterable contribution of that risk factor to the burden of disease. The most commonly used counterfactual in burden of disease studies is the **theoretical minimum risk exposure distribution**.

disability: In burden of disease analysis, any departure from an ideal health state.

disability-adjusted life years (DALY): A year of healthy life lost, either through premature death or living with disability due to illness or injury.

disability weight: A factor that reflects the severity of health loss from a particular **health state** on a scale from 0 (perfect health) to 1 (equivalent to death).

discounting: A method sometimes used to adjust the relative 'value' of years lived (or lost) in the future. It is based on the assumption that a year lived in the future is of less 'value' than a year lived now. Discounting for future benefits is standard practice in some economic analyses.

disease: A broad term that can be applied to any health problem, including symptoms, diseases, injuries and certain risk factors, such as high blood cholesterol and obesity. Often used synonymously with condition, disorder or problem.

effect modification: A change in the observed magnitude or direction of an association between a risk exposure and an outcome when a third variable (such as age or sex) is included in the analysis.

effect size: A statistical measure of the strength of the relationship between 2 variables (in this context, between a risk exposure and a disease outcome), expressed, for example, as a relative risk or odds ratio.

envelope: The total prevalence of a condition present in the population that is used to constrain the combined prevalence of sequelae common to a number of diseases.

excess burden: The reduction that would occur in overall disease burden if all groups had the same rate of burden as the least burdened group.

external cause: The environmental event, circumstance or condition that causes injury, poisoning and other adverse effect. METeOR identifier: 514295.

fatal burden: The burden from dying prematurely as measured by years of life lost. Often used synonymously with **years of life lost**, and also referred to as 'life lost'.

health state: Reflects a combination of signs and symptoms that result health loss, and are not necessarily unique to 1 particular disease. A health state might also be a severity level of a **sequela** (typically mild, moderate and severe levels are distinguished). For example, the health state 'mild heart failure' is used as a sequela of coronary heart disease, hypertensive heart disease, congenital heart disease and several other conditions. Each health state is associated with a **disability weight**.

hospitalisation: An episode of hospital care that starts with the formal admission process and ends with the formal separation process (synonymous with admission and separation).

incidence: Refers to the occurrence of a disease or event. The incidence rate is the number of new cases occurring during a specified time period.

International Classification of Diseases (ICD): The World Health Organization's internationally accepted classification of diseases and related health conditions. The 10th revision, Australian modification (ICD-10-AM) is currently in use in Australian hospitals for admitted patients.

linked disease: A disease or injury for which there is evidence that its likelihood is increased by the risk factor in question.

morbidity: Ill health in an individual, and levels of ill health in a population or group.

mortality: Death.

non-admitted patient: A patient who does not undergo a hospital's formal admission process. There are 3 categories of non-admitted patient: emergency department patient, outpatient, and other non-admitted patient (treated by hospital employees off the hospital site, including community/outreach services). METeOR identifier: 268973.

non-fatal burden: The burden from living with ill health as measured by years lived with disability. Often used synonymously with **years lived with disability**, and also referred to as 'health loss' in this ABDS 2011 reports.

population attributable fraction (PAF): For a particular risk factor and causally linked disease or injury, the percentage reduction in burden for a population that would occur if exposure to the risk factor was avoided or reduced to its theoretical minimum.

premature death: Deaths that occur at a younger age than a selected cut-off.

prevalence: Refers to the existence of a disease or event, whether or not it is newly occurring; the prevalence rate is the number of cases existing at a point in time (point prevalence) or over a specified time period (period prevalence).

principal diagnosis: The diagnosis established after study to be chiefly responsible for an episode of admitted patient care, an episode of residential care, or an attendance at the health care establishment. METeOR identifier: 514273.

rate: A rate is one number (the numerator) divided by another number (the denominator). The numerator is commonly the number of events in a specified time. The denominator is the population at risk of the event. Rates (crude, age-specific and age-standardised) are generally multiplied by a number such as 100,000 to create whole numbers.

redistribution: A method in a burden of disease study for reassigning deaths with an underlying cause of death that is not in the study's disease list. Typically, the deaths reassigned include: those with a case that is implausible as an underlying cause of death; those that relate to an intermediate cause in the chain of events leading to death; or those for which there is insufficient detail to ascertain a specific cause of death.

reference life table: A table that shows, for each age, the number of remaining years a person could potentially live, to measure the **years of life lost** from dying at that age.

relative risk: The risk of an event relative to exposure, calculated as the ratio of the probability of the even occurring in the exposed group to the probability of it occurring in the non-exposed group.

risk exposure distribution: The measure of the spread or distribution of exposure to the risk factor in the population that have encountered, experienced, or have the risk factor.

risk factor: Any factor that causes or increases the likelihood of a health disorder or other unwanted condition or event.

risk–outcome pair: Associates a condition in the disease list with a known risk factor for that condition.

sequelae: Health consequences of diseases and injuries, such as heart failure due to coronary heart disease. Each sequela may be mapped to one or more **health states**.

theoretical minimum risk exposure distribution (TMRED): The risk factor exposure distribution that will lead to the lowest conceivable disease burden.

years lived with disability (YLD): Measures the years of what could have been a healthy life that were instead spent in states of less than full health. YLD represent non-fatal burden.

years of life lost (YLL): Measures years of life lost due to premature death, defined as dying before the global ideal life span at the age of death. YLL represent fatal burden.

References

ABS (Australian Bureau of Statistics) 2001. Population characteristics, Aboriginal and Torres Strait Islander Australians, 2001, August 2003. ABS cat. no. 4713.0. Canberra: ABS. Viewed 22 June 2016, <www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/4713.0Main+Features12001?OpenDocument>.

ABS 2003. Labour force, Australia, June 2003. ABS cat. no. 6202.0. Canberra: ABS. Viewed 22 June 2016, <www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/6202.0Jun%202003?OpenDocument>.

ABS 2005. Deaths, Australia, 2004. ABS cat. no. 3302.0. Canberra: ABS. Viewed 24 August 2016, <www.abs.gov.au/AUSSTATS/abs@.nsf/allprimarymainfeatures/F69F652EDC86B05CCA257235007865FB?opendocument>.

ABS 2007. Labour force characteristics of Aboriginal and Torres Strait Islander Australians, experimental estimates from the Labour Force Survey, 2006. ABS cat. no. 6287.0. Canberra: ABS. Viewed 22 June 2016, <www.abs.gov.au/ausstats/abs@.nsf/mf/6287.0?OpenDocument>.

ABS 2009. Correspondence Tables -- ANZSCO First Edition Revision 1 to ISCO-88. ABS cat. no. 1220.0. Canberra: ABS. Viewed 31 August 2016, <www.abs.gov.au/ausstats/subscriber.nsf/log?openagent&12200 anzsco first edition revision 1 to isco-88 correspondence tables.xls&1220.0&Data Cubes&D458EA2435AFE28CCA2575DF001C7A57&0&First Edition, Revision 1&25.06.2009&Latest>.

ABS 2011a Labour force, Australia, June 2011. ABS cat. no. 6202.0. Canberra: ABS. Viewed 22 June 2016, <www.abs.gov.au/AUSSTATS/abs@.nsf/allprimarymainfeatures/B8BB5C6E29337405CA2578E8001F0C8C?opendocument>.

ABS 2011b. Labour force characteristics of Aboriginal and Torres Strait Islander Australians, estimates from the Labour Force Survey, 2011. ABS cat. no. 6287.0. Canberra: ABS. Viewed 22 June 2016, <www.abs.gov.au/ausstats/abs@.nsf/mf/6287.0?OpenDocument>.

ABS 2012a. Apparent consumption of alcohol, Australia, 2010–11. ABS cat. no. 4307.0.55.001. Canberra: ABS. Viewed 22 June 2016, <www.abs.gov.au/ausstats/abs@.nsf/mf/4307.0.55.001>.

ABS 2012b. Life tables, states, territories and Australia, 2010–2012. ABS cat. no 3302.055.001 DO001_20102012. Canberra: ABS.

ABS 2013a. Australian demographic statistics, June 2013. ABS cat. no. 3101.0. Canberra: ABS.

ABS 2013b. Causes of death, Australia, 2011. ABS cat. no. 3303.0. Canberra: ABS. Viewed 3 November 2014. <www.abs.gov.au/ausstats/abs@.nsf/Lookup/E8402CEA79A62C3BCA257B2E000D78CC?opendocument>.

ABS 2013c. Life tables for Aboriginal and Torres Strait Islander Australians 2010–2012. ABS cat. no. 3302.0.55.003. Canberra: ABS.

ABS 2013d. Personal safety, Australia, 2012. ABS cat. no. 4906.0. ABS: Canberra. Viewed 15 August 2015, <www.abs.gov.au/ausstats/abs@.nsf/mf/4906.0>.

ABS 2014a. Australian Aboriginal and Torres Strait Islander Health Survey: first results, 2012–13, Australia. ABS cat. no. 47270DO001_20122013. Canberra: ABS. Viewed 21 April 2016,<http://www.abs.gov.au/ausstats/abs@.nsf/mf/4727.0.55.001>.

ABS 2014b. Australian Aboriginal and Torres Strait Islander Health Survey: updated results, 2012–13, Australia. ABS cat. no. 4727.0.55.006. Canberra: ABS. Viewed 22 June 2016, <www.abs.gov.au/ausstats/abs@.nsf/Lookup/4727.0.55.006main+features12012-13>.

ABS 2014c. Australian Health Survey: users' guide, 2011–13: under-reporting in nutrition surveys. ABS cat. no. 4363.0.55.001. Canberra: ABS. Viewed 22 June 2016, <www.abs.gov.au/ausstats/abs@.nsf/Lookup/4363.0.55.001Chapter651512011-13>.

ABS 2014d. Remoteness structure. ABS cat. no. 1270.0.55.005. Canberra: ABS. Viewed 30 May 2016. <www.abs.gov.au/websitedbs/d3310114.nsf/home/remoteness+structure>.

ABS 2014e. Cause of death, 2014. ABS cat. no. 3303.0. Canberra: ABS. Viewed 30 May 2016, <www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/3303.0Explanatory%20Notes32014?Open Document>.

ABS 2015. Causes of death, Australia, 2013. ABS cat. no. 3303.0. Canberra: ABS. Viewed 30 May 2016. <www.abs.gov.au/AUSSTATS/abs@.nsf/Previousproducts/3303.0Quality%20Declaration02013?opendocument&tabname=Notes&prodno=3303.0&issue=2013&num=&view=>.

ABS 2016. Australian Health Survey: consumption of food groups from the Australian Dietary Guidelines, 2011–12. ABS cat. no. 4364.0.55.012. Canberra: ABS. Viewed 22 June 2016, <www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/4364.0.55.012Main+Features12011-12?OpenDocument>.

AHMAC (Australian Health Ministers' Advisory Council) 2012. Aboriginal and Torres Strait Islander Health Performance Framework 2012 report. Canberra: AHMAC.

AIHW (Australian Institute of Health and Welfare) 2008. Expenditure on health for Aboriginal and Torres Strait Islander people 2004–05. Health and welfare expenditure series no. 33. Cat no. HWE 40. Canberra: AIHW.

AIHW 2010. Indigenous identification in hospital separations data: quality report. Health services series no. 35. Cat. no. HSE 85. Canberra: AIHW.

AIHW 2011. End-stage kidney disease in Australia: total incidence, 2003–2007. Cat. no. PHE 143. Canberra: AIHW.

AIHW 2012a. An enhanced mortality database for estimating Indigenous life expectancy: a feasibility study. Cat. no. IHW 75. Canberra: AIHW.

AIHW 2012b. Dementia in Australia. Cat. no. AGE 70. Canberra: AIHW.

AIHW 2012c. Residential aged care in Australia 2010–11: a statistical overview. Aged care statistics series no. 36. Cat. no. AGE 68. Canberra: AIHW.

AIHW 2012d. Aged care packages in the community 2010–11: a statistical overview. Aged care statistics series no. 37. Cat. no. AGE 69. Canberra: AIHW.

AIHW 2013. Indigenous identification in hospital separations data: quality report. Cat. no. IHW 90. Canberra: AIHW.

AIHW 2014a. Acute coronary syndrome: validation of the method used to monitor incidence in Australia. Cat. no. CVD 68. Canberra: AIHW.

AIHW 2014b. Assessment of Global Burden of Disease 2010 methods for the Australian context. Australian Burden of Disease Study working paper no. 3. Cat. no. WEB 035. Canberra: AIHW.

AIHW 2014c. Assessment of the coding of ESKD in deaths and hospitalisation data: a working paper. Cat. no. PHE 182. Canberra: AIHW.

AIHW 2014d. Australian hospital statistics 2013–14: emergency department care. Health services series no. 58. Cat. no. HSE 153. Canberra: AIHW.

AIHW 2014e. Cardiovascular disease, diabetes and chronic kidney disease, Australian facts: prevalence and incidence. Cardiovascular, diabetes and chronic kidney disease series no. 2. Cat. no. CDK 2. Canberra: AIHW.

AIHW 2014f. Residential and community aged care supplementary data. Viewed 19 May 2015, <www.aihw.gov.au/aged-care/residential-and-community-2012-13/data>.

AIHW 2015a. Aboriginal and Torres Strait Islander health performance framework 2014 report: detailed analyses. Cat. no. IHW 167. Canberra: AIHW. Viewed 21 February 2016, <www.aihw.gov.au/publication-detail/?id=60129550779>.

AIHW 2015b. Australian Burden of Disease Study: fatal burden of disease in Aboriginal and Torres Strait Islander people 2010. Australian Burden of Disease Study series no. 2. Cat. no. BOD 2. Canberra: AIHW.

AIHW 2015c. Cardiovascular disease fact sheet: prevalence of coronary heart disease in Western Australia. Cat. no. CVD 71. Canberra: AIHW. Viewed 29 July 2016, <www.aihw.gov.au/publication-detail/?id=60129552846>.

AIHW 2015d. Cardiovascular disease fact sheet: prevalence of heart failure in Western Australia. Cat. no. CVD 72. Canberra: AIHW. Viewed 29 July 2016, <www.aihw.gov.au/publication-detail/?id=60129552813>.

AIHW 2015e. Cardiovascular disease fact sheet: prevalence of stroke in Western Australia. Cat. no. CVD 73. Canberra: AIHW. Viewed 29 July 2016, <www.aihw.gov.au/publication-detail/?id=60129552853>.

AIHW 2015f. Cardiovascular disease fact sheet: prevalence of valvular heart disease in Western Australia. Cat. no. CVD 74. Canberra: AIHW. Viewed 29 July 2016, <www.aihw.gov.au/publication-detail/?id=60129552856>.

AIHW 2016a. About deaths data. Canberra: AIHW. Viewed 30 May 2016, <www.aihw.gov.au/deaths/about-deaths-data>.

AIHW 2016b. Australian Burden of Disease Study: impact and causes of illness and death in Australia 2011. Australian Burden of Disease Study series no. 3. Cat. no. BOD 4. Canberra: AIHW.

AIHW 2016c. Australian Burden of Disease Study: impact and causes of illness and death in Aboriginal and Torres Strait Islander people 2011. Australian Burden of Disease Study series no. 6. Cat. no. BOD 7. Canberra: AIHW.

AIHW & Cancer Australia 2013. Cancer in Aboriginal and Torres Strait Islander peoples of Australia: an overview. Cancer series no. 78. Cat. no. CAN 75. Canberra: AIHW. Viewed 22 June 2016, <www.aihw.gov.au/publication-detail/?id=60129544700>.

AIHW Dental Statistics and Research Unit 2008. Patterns of tooth loss in the Australian population 2004–06. AIHW Dental Statistics and Research Unit research report no. 38. Cat. no. DEN 185. Canberra: AIHW.

AIHW National Perinatal Statistics Unit: Grayson N, Hargreaves J & Sullivan EA 2005. Use of routinely collected national data sets for reporting on induced abortion in Australia. Perinatal statistics series no. 17. Cat. no. PER 30. Sydney: AIHW Perinatal Statistics Unit.

Atkinson J, Nelson J & Atkinson C 2010. Trauma, transgenerational transfer and effects of community wellbeing. In Purdie N, Dudgeon P & Walker R (eds.). Working together: Aboriginal and Torres Strait Islander mental health and wellbeing principles and practice. Canberra: Australian Government Department of Health and Ageing, 135–144.

Asimus M & Li P 2011. Pressure ulcers in home care settings: is it overlooked? Wound Practice and Research 19(2):88–97.

Australian Hearing 2012. Demographic details of persons under the age of 21 years with a hearing impairment who are fitted with a hearing aid or cochlear implant, 2011. Sydney: Australian Hearing. Viewed 15 August 2014, <www.hearing.com.au/>.

Ayre J, Lum On M, Webster K & Moon L forthcoming. Examination of the burden of disease of intimate partner violence against women: horizons report, 2011. Sydney: Australian National Research Organisation for Women's Safety.

Baba M, Davis WA, Norman PE & Davis TME 2015. Temporal changes in the prevalence and associates of foot ulceration in type 2 diabetes: the Fremantle Diabetes Study. Journal of Diabetes and its Complications 29:356–61.

Badawi N, Felix JF, Kurinczuk JJ, Dixon G, Watson L, Keogh JM et al. 2005. Cerebral palsy following term newborn encephalopathy: a population-based study. Developmental Medicine and Child Neurology 47(5):293–8.

Banks M, Ash S, Bauer J & Gaskill D 2007. Prevalence of malnutrition in adults in Queensland public hospitals and residential aged care facilities. Nutrition and Dietetics 64(3):172–8.

Barendregt JJM & Bonneux LGA 1998. Degenerative disease in an aging population models and conjectures. Rotterdam: Erasmus University. Viewed 16 October 2014, <http://hdl.handle.net/1765/16984>.

Baxter AJ, Brugha TS, Erskine HE, Scheurer RW, Vos T & Scott, JG 2015. The epidemiology and global burden of autism spectrum disorders. Psychological Medicine 45(03):601–13.

Begg S, Vos T, Barker B, Stevenson C, Stanley L & Lopez AD 2007. The burden of disease and injury in Australia 2003. Cat. no. PHE 82. Canberra: AIHW.

Biddle N 2013. Socioeconomic outcomes. Paper 13. Indigenous population project: 2011 census report. Canberra: Centre for Aboriginal Economic Policy Research.

Blencowe H, Lee ACC, Cousens S, Bahalim A, Narwal R, Zhong N et al. 2013. Pre-term birth: associated neurodevelopmental impairment estimates at regional and global levels for 2010. Pediatric Research 74:17–34.

Boyce P, Talley N, Burke C & Koloski N 2006. Epidemiology of the functional gastrointestinal disorders diagnosed according to Rome II criteria: an Australian population-based study. Internal Medicine Journal 36:28–36.

Brennan DS & Spencer AJ 2004. Disability weights for the burden of oral disease in South Australia. Pop Health Metrics 2:7.

Bryant W & Cussen T 2015. Homicide in Australia, 2010–11 to 2011–12: National Homicide Monitoring Program report (monitoring report no.23). Canberra: Australian Institute of Criminology. Viewed 22 June 2016, <www.aic.gov.au/publications/current%20series/mr/21-40/mr23.html>.

Buist AS, McBurnie MA, Vollmer WM, Gillespie S, Burney P, Mannino DM et al. 2007. International variation in the prevalence of COPD (the BOLD Study): a population-based prevalence study. Lancet 370:741–50.

Burstein R, Fleming T, Haagsma J, Salomon JA, Vos T & Murray CJL 2015. Estimating distributions of health state severity for the global burden of disease study. Population Health Metrics 13:31.

CDC (Centers for Disease Control) 2012. Lumbar spine and proximal femur bone mineral density. Vital and Health Statistics 11(251).

Cerebral Palsy Alliance 2013. Australian Cerebral Palsy Register report 2013. Viewed 19 July 2016, <www.cpregister.com/pubs/pdf/ACPR-Report_Web_2013.pdf>.

Chitkara D, Rawat D & Talley N 2005. The epidemiology of childhood recurrent abdominal pain in western countries: a systematic review. The American Journal of Gastroentorology 100:1868–75.

Chow SSW 2013. Report of the Australian and New Zealand Neonatal Network 2011. Sydney: ANZNN. Viewed 9 February 2015, <https://npesu.unsw.edu.au/sites/default/files/npesu/data_collection/Report%20of%20the%20Australian%20and%20New%20Zealand%20Neonatal%20Network%202011.pdf>.

Cole T, Flegal K, Nicholls D & Jackson A 2007. Body mass index cut offs to define thinness in children and adolescents: international survey. British Medical Journal 335:194.

Conway D, McMahon A, Robertson D & Macpherson L 2014. Epidemiology of dental diseases. In Ahrens W & Pigeot I (eds). Handbook of epidemiology, 2nd edition. New York: Spring Science+Business 2321–46.

Cooksley N, Atkinson D, Marks G, Toelle B, Reeve D, Johns D et al. 2015. Prevalence of airflow obstruction and reduced forced vital capacity in an Aboriginal Australian population: the cross-sectional BOLD study. Respirology 20(5):766–774. doi:10.111/resp.12482.

Covance Pty. Ltd. & Palmer A 2011. Economic impact of multiple sclerosis in 2010: Australian Multiple Sclerosis Longitudinal Study. North Ryde: Covance Pty. Ltd. Viewed 16 July 2014, <www.msra.org.au/files/msra/docs/Economic%20Impact%20of%20MS%20in%202010%20Full%20Report%20v2.pdf>.

Cunningham J, O'Dea K, Dunbar T, Weeramanthri T, Zimmet P & Shaw J 2006. Study protocol—diabetes and related conditions in urban Indigenous people in the Darwin,

Australia region: aims, methods and participation in the DRUID Study. BMC Public Health 6:8.

Cussen T & Bryant W 2015. Research in practice: Indigenous and non-Indigenous homicide in Australia. Monitoring report no. 37. Viewed 22 June 2016, <www.aic.gov.au/media_library/publications/rip/rip37/rip37.pdf>.

Cystic Fibrosis Australia 2012. Cystic Fibrosis in Australia 2011: 14th annual report, Australian Cystic Fibrosis Data Registry. Baulkham Hills: Cystic Fibrosis Australia.

Davis TME, Hunt K, McAullay D, Chubb SA, Sillars BA, Bruce DG et al. 2012. Continuing disparities in cardiovascular risk factors and complications between Aboriginal and Anglo-Celt Australians with type 2 diabetes: the Fremantle Diabetes Study. Diabetes Care 35(10):2005–11.

Day SM, Strauss DJ, Shavelle RM & Reynolds RJ 2005. Mortality and causes of death in persons with Down syndrome in California. Developmental Medicine and Child Neurology 47(03):171–6.

De Rijk MC, Launer LJ, Berger K, Breteler MMB, Fartigues JF, Baldereschi M et al. 2000. Prevalence of Parkinson's disease in Europe: a collaborative study of population-based cohorts. Neurology 54(Suppl 5):S21–3.

Dealey C, Posnett J &Walker A 2012. The cost of pressure ulcers in the United Kingdom. Journal of Wound Care 21(6): 261–6.

Dearden J & Jones W 2010. Homicide in Australia: 2006–07 National Homicide Monitoring Program annual report. Monitoring report no. 01. Viewed 22 June 2016, <www.aic.gov.au/media_library/publications/mr/mr01/mr01.pdf>.

Degenhardt L, Larney S, Chan G, Dobbins T, Weier M, Roxburgh A et al. 2016. Estimating the number of regular and dependent methamphetamine users in Australia, 2002–2014. Medical Journal of Australia 204(4):1.e2–6.

Degenhardt L, Rendle V, Hall W, Gilmour S & Law M 2004. Estimating the number of current regular heroin users in NSW and Australia 1997–2002. Sydney: National Drug and Alcohol Research Centre.

Deloitte Access Economics 2011. Eyes on the future: a clear outlook on age-related macular degeneration. Sydney: Macular Degeneration Foundation. Viewed 24 September 2014, <www.mdfoundation.com.au/LatestNews/MDFoundationDeloitteAccessEconomicsReport%202011ExecSummary.pdf>.

Emerson E, Einfeld S & Stancliffe R.J 2010. The mental health of young children with intellectual disabilities or borderline intellectual functioning. Social Psychiatry and Psychiatric Epidemiology 45: 579. doi:10.1007/s00127-009-0100-y.

Erskine HE, Ferrari AJ, Polanczyk GV, Moffitt TE, Murray CJ, Vos T et al. 2014. The global burden of conduct disorder and attention-deficit/hyperactivity disorder in 2010. Journal of Child Psychology and Psychiatry 55(4):328–36.

Ezzati M, Lopez AD, Rodgers AA & Murray CJL 2004. Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors. Geneva: World Health Organization.

Fagan PS, Downing SG, McCall BJ, Carroll HJ, Howard TM & Palmer CM 2013. Enhanced surveillance for gonorrhoea in two diverse settings in Queensland in the 2000s. Communicable Disease Intelligence 37(3):E253–9.

Ferrari AJ, Saha S, McGrath JJ, Norman R, Baxter AJ, Vos T et al. 2012. Health states for schizophrenia and bipolar disorder within the Global Burden of Disease 2010 Study. Population Health Metrics 10(1):16.

Forsgren L, Beghi E, Õun A & Sillanpää M 2005. The epidemiology of epilepsy in Europe: a systematic review. European Journal of Neurology 12:245–53.

FSANZ (Food Standards Australia New Zealand) 2016. Assessing the 2011–13 AHS against the Australian Dietary Guidelines. Canberra: FSANZ. Viewed 22 June 2016, <www.foodstandards.gov.au/science/monitoringnutrients/australianhealthsurveyandaustraliandietaryguidelines/Pages/default.aspx>.

GBD (Global Burden of Disease Study) 2013 Collaborators 2015a. Global, regional, and national incidence, prevalence, and years lived with disability for 301 acute and chronic diseases and injuries in 188 countries, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. The Lancet 2015. 386(995):743–800. doi.org/10.1016/S0140-6736(15)60692-4.

GBD 2013 Collaborators 2015b. Supplement to: Global, regional, and national incidence, prevalence, and years lived with disability for 301 acute and chronic diseases and injuries in 188 countries, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013: The Lancet 2015. 386(10010): S1–1868. doi.org/10.1016/S0140-6736(15)60692-4.

GBD 2013 Risk Factors Collaborators 2015. Supplement to: Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. The Lancet 2015. 386(10010): S1–711. doi.org/10.1016/S0140-6736(15)00128-2.

Gearry RB, Richardson A, Frampton CM, Collett JA, Burt MJ, Chapman BA et al. 2006. High incidence of Crohn's disease in Canterbury, New Zealand: results of an epidemiologic study. Inflammatory Bowel Disease. 12(10):936–43.

Gilbert S, Wood D, Dunn R, Weizer A, Lee C, Montie J et al. 2007. Measuring health-related quality of life outcomes in bladder cancer patients using the bladder cancer index. Michigan: American Cancer Society. 109(9):1756–62.

Glasson EJ, Sullivan SG, Hussain R, Petterson BA, Montgomery PD & Bittles AH 2003. Comparative survival advantage of males with Down syndrome. American Journal of Human Biology 15(2):192–5.

Haagsma J, Maertens de Noordhout C, Poliner S, Vos T, Havelaar A, Cassini A et al. 2015. Assessing disability weights based on the responses of 30,660 people from four European countries. Population Health Metrics 13:10.

Hafner LM & Pelzer ES 2011. Tubal damage, infertility and tubal ectopic pregnancy: chlamydia trachomatis and other microbial aetiologies, ectopic pregnancy. In: Kamrava M (ed.). Modern diagnosis and management. InTech; online. Viewed 24 April 2015, <www.intechopen.com/books/ectopic-pregnancy-modern-diagnosis-and-management>.

Harrison C, Britt H, Miller G & Henderson J 2013. Prevalence of chronic conditions in Australia. PLoS ONE 8(7):e67494. doi:10.1371/journal.pone.0067494.

Helgeland H, Flagstad G, Grøtta J, Vandvik P, Kristensen H & Markestad T 2009. Diagnosing pediatric function abdominal pain in children (4–15 years old) according to the Rome III criteria: results from a Norwegian prospective study. Journal of Pediatric Gastroenterology and Nutrition 49(3):309–15.

Henry MJ, Pasco JA, Korn S, Gibson JE, Kotowicz MA & Nicholson GC 2010. Bone mineral density reference ranges for Australian men: Geelong Osteoporosis Study. Osteoporosis International 21(6):909–17.

Hoffman H & Reed G 2004. Epidemiology of Tinnitus In: Snow J (ed). Tinnitus: theory and management. BC Decker: Ontario Canada 16–41.

Hoy D, Smith E, Cross M, Sanchez-Riera L, Burchbinder R, Blyth F et al. 2014. The global burden of musculoskeletal conditions for 2010: an overview of methods. Annals of the Rheumatic Diseases 73(6)982–9.

Hoy W & Nicol JL 2010. Birthweight and natural deaths in a remote Australian Aboriginal community. Medical Journal of Australia 192(1):14–9.

Hunt GM & Oakeshott P 2003. Outcome in people with spina bifida at age 35: prospective community based cohort study. BMJ 326:1365–6.

ICBDSR (International Clearinghouse for Birth Defects Surveillance and Research) 2013. Annual report 2013. Italy: ICBDSR. Viewed 27 October 2015, <www.icbdsr.org/filebank/documents/ar2005/Report2013.pdf>.

Jackson SL, Weber AM, Hull TL, Mitchinson AR & Walters MD 1997. Fecal incontinence in women with urinary incontinence and pelvic organ prolapse. Obstetrics and Gynecology 89(3):423–7.

Jansson J, Wilson D & Watson J 2010. Mapping HIV outcomes: geographical and clinical forecasts of numbers of people living with HIV in Australia. Darlinghurst: National Centre in HIV Epidemiology and Clinical Research.

Jenkins WMM & Papapanou PN 2001. Epidemiology of periodontal disease in children and adolescents. Periodontology 2000 26:16–32.

Jenner N, Campbell J, Plunkett A & Marks R 2002. Cost of psoriasis: a study on the morbidity and financial effects of having psoriasis in Australia. Australasian Journal of Dermatology 43(4):255–61.

Kaiser M, Bauer J, Ramsch C, Uter W, Guigoz Y, Cederholm T et al. 2010. Mini nutritional assessment international 2010. Frequency of malnutrition in older adults: a multinational perspective using the mini nutritional assessment. Journal of the American Geriatrics Society 58(9):1734–8.

Kassebaum NJ, Jasrasaria R, Naghavi M, Wulf SK, Johns N, Lozano R et al. 2014. A systematic analysis of global anemia burden from 1990 to 2010. Blood 123:615–24.

Katzenellenbogen JM, Begg S, Somerford P, Anderson CS, Semmens JB, Codde JP et al. 2010. Disability burden due to stroke in Western Australia: new insights from linked data sources. International Journal of Stroke 5:269–77.

Khaund A & Lumsden MA 2008. Impact of fibroids on reproductive function. Best Practice and Research Clinical Obstetrics and Gynaecology 22(4):749–60.

Kilkenny M, Merlin K, Plunkett A & Marks R 1998. The prevalence of common skin conditions in Australian school students: acne vulgaris. British Journal of Dermatology 139(5):840–5.

Kirby Institute 2004. HIV, viral hepatitis and sexually transmitted infections in Australia: annual surveillance report 2004. Sydney: Kirby Institute, UNSW.

Kirby Institute 2012. HIV, viral hepatitis and sexually transmitted infections in Australia: annual surveillance report 2012. Sydney: Kirby Institute, UNSW.

Kirby Institute 2013. National blood-borne virus and sexually transmissible infections surveillance and monitoring report 2013. Sydney: Kirby Institute, UNSW.

Kirby Institute 2014. HIV, viral hepatitis and sexually transmitted infections in Australia: annual surveillance report 2014. Sydney: Kirby Institute, UNSW.

Kirk M, Glass K, Ford L, Brown K & Hall G 2014. Foodborne illness in Australia: annual incidence circa 2010. Canberra: Department of Health.

Knope K, Kurucz N, Doggett S, Muller M, Johansen C, Feldman R et al. 2016. Arboviral diseases and malaria in Australia, 2012–13: annual report of the National Arbovirus and Malaria Advisory Committee. CDI vol 40. no. 1, 2016. <www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdi4001d.htm>.

Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H et al. 2012. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 380(9859):2224–60.

Looker A, Dallman P, Carroll M, Gunter E & Johnson C 1997. Prevalence of iron-deficiency in the United States. The Journal of the American Medical Association 277:(12); 973–6.

Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V et al. 2012. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. The Lancet 380:2095–128.

Lucas R, McMichael T, Smith W & Armstrong B 2006. Solar ultraviolet radiation: global burden of disease from solar ultraviolet radiation. Environmental burden of disease series. Geneva: World Health Organization.

Lucca U, Tettamanti M, Logroscino G, Tiraboschi P, Landi C, Sacco L et al. 2015. Prevalence of dementia in the oldest old: The Monzino 80-plus population based study. Alzheimer's and Dementia 2015 11:258–70.

Lum On M, Ayre J, Webster K & Moon L 2016. Examination of the health outcomes of intimate partner violence against women: state of knowledge paper (2016). Sydney: Australian National Research Organisation for Women's Safety.

MacGinley R & Allen R 1997. Sarcoidosis in an Australian Aborigine and a Torres Strait Islander. Sarcoidosis Vasculitis and Diffuse Lung Diseases 14:83–5.

McIntyre S, Blair E, Badawi N, Keogh J &Nelson K 2013. Antecedents of cerebral palsy and perinatal death in term and late pre-term singletons. Obstetrics and Gynaecology 122(4):869–77

Mackerras D, Hutton S & Anderson P 2004. Haematocrit levels and anaemia in Australian children aged 1–4 years. Asia Pacific Journal of Clinical Nutrition 13(4):330–5.

Marino JL, Moore VM, Rumbold AR & Davies MJ 2011. Fertility treatments and the young women who use them: an Australian cohort study. Human Reproduction 26(2):473–9.

Marks R, Kilkenny M, Plunkett A & Merlin K 1999a. The prevalence of common skin conditions in Australian school students: atopic dermatitis. British Journal of Dermatology 140(3):468–73.

Marks R, Plunkett A, Merlin K & Jenner N 1999b. Atlas of common skin diseases in Australia. Melbourne: Department of Dermatology, St Vincent's Hospital.

Maulik PK, Mascarenhas MN, Mathers CD, Dua T & Saxena S 2011. Prevalence of intellectual disability: a meta-analysis of population-based studies. Research in Developmental Disabilities 32(2):419–36.

Mitchell P, Gopinath B, Wang J, McMahon C, Schneider J, Rochtchin E et al. 2011. Five-year incidence and progression of hearing impairment in an older population. Ear and Hearing 32(2):251–7.

Mouzos J 2005. Homicide in Australia: 2003–2004 National Homicide Monitoring Program (NHMP) Annual Report. Canberra: Australian Institute of Criminology. Viewed 23 August 2016, <www.aic.gov.au/media_library/publications/rpp/66/rpp066.pdf>.

Mulligan S, Prentice J & Scott L 2011. WoundsWest Wound Prevalence Survey 2011: state-wide overview report. Perth: Ambulatory Care Services, Department of Health.

Murray CJ, Ezzati M, Flaxman AD, Lim S, Lozano R, Michaud C et al. 2012. GBD 2010: design, definitions and metrics. The Lancet 380:2063–6.

Naghavi M, Makela S, Foreman K, O'Brien J, Pourmalek F & Lozano R 2010. Algorithms for enhancing public health utility of national causes-of-death data. Population Health Metrics 20108:9. doi:10.1186/1478-7954-8-9.

National Blood Authority 2012. Australian Bleeding Disorders Registry Annual Report 2011–2012. Canberra: National Blood Authority. Viewed 27 February 2015, <www.blood.gov.au/system/files/documents/abdr-annual-report-2011-12-20140423.pdf>.

NCCH (National Centre for Classification in Health) 2010. The International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification (ICD-10-AM), Australian Classification of Health Interventions (ACHI) and Australian Coding Standards (ACS). Seventh Edition. Sydney: NCCH, University of Sydney.

New South Wales Office of Environment and Heritage 2015. Search air quality data. Sydney: OEH. Viewed 15 July 2015, <www.environment.nsw.gov.au/AQMS/search.htm>.

NHMRC (National Health and Medical Research Council) 2015. Australian Dietary Guidelines 1–5. Canberra: NHMRC. Viewed 15 August 2015, <www.eatforhealth.gov.au/guidelines/australian-dietary-guidelines-1-5>.

Nickson C, Smith AMA & Shelley JM 2004. Intention to claim a Medicare rebate among women receiving private Victorian pregnancy termination services. Australian and New Zealand Journal of Public Health 28(2):120–3.

Northern Territory Environment Protection Authority 2015. Northern Territory Air Quality Network. Envitech, Envista (Version 5.2.7.). Darwin: EPA. Viewed 15 July 2015, <http://ntepa.webhop.net/NTEPA/Default.ltr.aspx>.

NZMOH (New Zealand Ministry of Health) 2012. Ways and means: a report on methodology from the New Zealand Burden of Diseases, Injuries and Risk Factors Study, 2000–2016. Wellington: New Zealand Ministry of Health.

NZMOH 2013. New Zealand Burden of Diseases, Injuries and Risk Factors Study 2006–2016. Wellington: New Zealand Ministry of Health. Viewed 19 July 2016, <www.health.govt.nz/nz-health-statistics/health-statistics-and-data-sets/new-zealand-burden-diseases-injuries-and-risk-factors-study-2006-2016>.

Oakeshott, P, Hunt G, Poulton A & Reid F 2010. Expectation of life and unexpected death in open spina bifida: a 40-year complete, non-selective, longitudinal cohort study. Developmental Medicine and Child Neurology 52:749–53.

Oti-Boateng P, Seshadri R, Petrick S, Gibson RA & Simmer K et al. 1998. Iron status and dietary iron intake of 6–24-month-old children in Adelaide. Journal of Paediatric Child Health 34(3):250–3.

Palmer AJ, Hitchens PL, Simpson Jr. S, O'Leary B, Colman S & Taylor BV 2013. A novel method for calculating prevalence of multiple sclerosis in Australia. Multiple Sclerosis Journal. Viewed 15 April 2013, <http://msj.sagepub.com/content/early/2013/04/15/1352458513479841>.

Peeters GMEE, Alshurafa M, Schaap L & de Vet H 2015. Diagnostic accuracy of self-reported arthritis in the general adult population is acceptable. Journal of Clinical Epidemiology 68:452–9.

Peña A & Hong A 2000. Advances in the management of anorectal malformations. The American Journal of Surgery 180(5):370–6.

Petterson B, Bourke J, Leonard H, Jacoby P & Bower C 2007. Co-occurrence of birth defects and intellectual disability. Paediatric and perinatal epidemiology 21(1):65–75.

Peto R, Boreham J, Lopez AD, Thun M & Heath C 1992. Mortality from tobacco in developed countries: indirect estimation from national vital statistics. The Lancet, 339(8804):1268–78.

Plunkett A, Merlin K, Gill D, Zuo Y, Jolley D & Marks R 1999. The frequency of common non-malignant skin conditions in adults in central Victoria, Australia. International Journal of Dermatology 38(12):901–8.

Porter KR, McCarthy BJ, Freels S, Kim Y & Davis FG 2010. Prevalence estimates for primary brain tumors in the United States by age, gender, behavior and histology. Neuro-oncology 12(6):520–7.

Queensland Government 2015. Air quality monitoring 2011. Brisbane: Queensland Government. Viewed 15 July 2015, <https://data.qld.gov.au/dataset/air-quality-monitoring-2011>.

Queensland Health 2012. The State of Queensland (Queensland Health) annual report 2011–12. Brisbane: Queensland Health. Viewed 22 June 2016, <https://publications.qld.gov.au/storage/f/2014-06-06T04%3A05%3A23.024Z/annual-report-2011-12.pdf>.

Radford K, Mack HA, Draper B, Chalkle S, Daylight G, Cumming R et al. 2015. Prevalence of dementia in urban and regional Aboriginal Australians. Alzheimer's and Dementia 11:1–9.

Reddel HK, Sawyer SM, Everett PW, Flood PV & Peters MJ 2015. Asthma control in Australia: a cross-section web-based survey in a nationally representative population. Medical Journal of Australia 202(9):492–6.

Reekie J, Donovan B, Guy R, Hocking JS, Jorm L, Kaldor JM et al. 2014. Hospitalisations for Pelvic Inflammatory Disease Temporally Related to a Diagnosis of Chlamydia or Gonorrhoea: A Retrospective Cohort Study. PLoS ONE 9(4):e94361.

Rehm J, Kehoe T, Gmel G, Stinson F, Grant B & Gmel G 2010. Statistical modelling of volume of alcohol exposure for epidemiological studies of population health: the US example. Population Health Metrics 8(3):1–12.

Ressler KA, Smedley E, Spokes P, Hockey G, Nurkic A & Ferson MJ 2013. Enhanced surveillance of gonorrhoea in South Eastern Sydney. Sydney: NSW Health.

Rist, G, Miles G & Karimi L 2012. The presence of malnutrition in community-living older adults receiving home nursing services. Nutrition & Dietetics 69(1):46–50.

Rome Foundation 2006. Appendix A: Rome III diagnostic criteria for FGIDs. Rome: Rome Foundation. Viewed 9 December 2014, <www.romecriteria.org/assets/pdf/19_RomeIII_apA_885-898.pdf>.

Royal Children's Hospital Melbourne 2010. Cleft lip and palate. Melbourne: Royal Children's Hospital. Viewed 15 April 2015, <http://www.rch.org.au/uploadedFiles/Main/Content/kidsinfo/6_Cleft_lip_and_palate_booklet.pdf>.

SA Health (South Australian Department of Health) 2007. South Australian Pressure Ulcer Point Prevalence Survey report 2007: increasing validity and reliability of findings, category A1. Adelaide: SA Health.

SA Health 2012. Surveillance of sexually transmitted infections and blood-borne viruses in South Australia 2011. Adelaide: SA Health.

Sadler S & Blight G 1996. Iron status and dietary iron intake of young women. Proceedings of the Nutrition Society of Australia 20:216.

Safe Work Australia 2012. Work-related traumatic injury fatalities, Australia, 2010–11. Canberra: Safe Work Australia. Viewed 22 October 2015, <www.safeworkaustralia.gov.au/sites/SWA/about/Publications/Documents/730/WorkRelatedTraumaticInjuryFatalities2010-11.pdf>.

Safe Work Australia 2013. Compendium of workers' compensation statistics Australia, 2010–11. Canberra: Safe Work Australia. Viewed 22 October 2015, <www.safeworkaustralia.gov.au/sites/SWA/about/Publications/Documents/766/Compendium%202010-11.pdf>.

Salomon J 2010. New disability weights for the global burden of disease. Bulletin of the World Health Organization 88:879.

Salomon JA, Vos T, Hogan DR, Gagnon M, Naghavi M, Mokdad A et al. 2012. Common values in assessing health outcomes from disease and injury: disability weights measurement study for the Global Burden of Disease Study 2010. The Lancet 15;380(9859):2129–43. doi:10.1016/S0140-6736(12)61680-8.

Sànchez-Riera L, Carnahan E, Vos T, Veerman L, Norman R, Lim SS et al. 2014. The global burden attributable to low bone mineral density. Annals of Rheumatic Diseases. 73:1635–45.

Santamaria N, Carville K, Prentice J, Ellis I, Ellis T, Lewin G et al. 2009. Reducing pressure ulcer prevalence in residential aged care: results from phase II of the PRIME trial. Wound Practice and Research: Journal of the Australian Wound Management Association 17(1):12–22.

Sell D, Grunwell P, Mildinhall S, Murphy T, Cornish TA, Bearn D et al. 2009. Cleft lip and palate care in the United Kingdom—the Clinical Standards Advisory Group (CSAG) Study. Part 3: speech outcomes. Cleft Palate–Craniofacial Journal 38(1):30–7.

Shepard CW, Simard EP, Finelli L, Fiore AE & Bell BP 2006. Hepatitis B virus infection: epidemiology and vaccination. Epidemiologic Reviews 28:112–25.

Smith DP, King MT, Egger S, Berry MP, Stricker PD, Cozzi P et al. 2009. Quality of life three years after diagnosis of localised prostate cancer: population based cohort study. BMJ 339:b4817.

Smith K, Flicker L, Lautenschlager NT, Almeida OP, Atkinson D, Dwyer A et al. 2008. High prevalence of dementia and cognitive impairment in Indigenous Australians. Neurology 71:1470–3.

Staples MP, Elwood M, Burton RC, Williams JL, Marks R & Giles GG 2006. Non-melanoma skin cancer in Australia: the 2002 national survey and trends since 1985. MJA 184(1):6–10.

Steering Committee for the Review of Government Service Provision 2014. Overcoming Indigenous disadvantage: key indicators 2014. Canberra: Productivity Commission. Viewed 22 June 2016, <www.pc.gov.au/research/ongoing/overcoming-indigenous-disadvantage/key-indicators-2014#thereport>.

Studd C 2013. Assessment of inflammatory bowel disease epidemiology in Barwon, Victoria: an observational prospective population-based study. Melbourne: University of Melbourne. Viewed 24 November 2014, <https://minerva-access.unimelb.edu.au/bitstream/handle/11343/38454/303645_Studd.pdf>.

Sung J, Kuipers E & El-Serag H 2009. Systematic review: the global incidence and prevalence of peptic ulcer disease. Alimentary Pharmacology and Therapeutics. 29(9):938–46. doi:10.1111/j.1365–2036.2009.03960.x.

Tapp RJ, Shaw JE, de Courten MP, Dunstan DW, Welborn TA & Zimmet PZ 2003a. Foot complications in type 2 diabetes: an Australian population-based study. Diabetic Medicine 20:105–13.

Tapp RJ, Shaw JE, Harper CA, de Courten MP, Balkau B, McCarty DJ et al. 2003b. The prevalence of and factors associated with diabetic retinopathy in the Australian population. Diabetes Care 26:1731–7.

Taylor B, Irving H, Kanteres F, Room R, Borges G, Cherpitel C et al. 2010. The more you drink, the harder you fall: a systematic review and meta-analysis of how acute alcohol consumption and injury or collision risk increase together. Drug and Alcohol Dependence 110:108–16.

Taylor H, Keeffe J, Vu H, Wang J, Rochtchina E, Pezzullo M & Mitchell P 2005. Vision loss in Australia. Medical Journal of Australia 182:565–8.

Tegerstedt G, Maehle-Schmidt M, Nyrén M & Hammarström M 2005. Prevalence of symptomatic pelvic organ prolapse in a Swedish population. International Urogynaecology Journal 16:495–503.

Thompson B, Olsen C, Subramaniam P, Neale R & Whiteman P 2014. Medicare data for cancer follow-up studies. Paper presented at the Australasian Epidemiological Association Annual Scientific Meeting, Auckland, 2014.

Thrift AG, Dewey HM, Sturm JW, Srikanth VK, Gilligan AK, Gall SL et al. 2009. Incidence of stroke subtypes in the North East Melbourne Stroke Incidence Study (NEMESIS): differences between men and women. Neuroepidemiology 32(1):11–8.

Toelle BG, Xuan W, Bird TE, Abramson MJ, Atkinson SN, Burton DL et al. 2013. Respiratory symptoms and illness in older Australians: the Burden of Obstructive Lung Disease (BOLD) Study. Medical Journal of Australia 198(3):144–8.

US Burden of Disease Collaborators 2013. The state of US health, 1990–2010: burden of diseases, injuries, and risk factors. The Journal of the American Medical Association 210(6):591–606.

Vos T, Barker B, Stanley L & Lopez AD 2007. The burden of disease and injury in Aboriginal and Torres Strait Islander peoples 2003. Brisbane: University of Queensland.

VQC (Victorian Quality Council) 2006. Pressure Ulcer Point Prevalence Survey: statewide report 2006. Melbourne: Victorian Government Department of Human Services.

Wang Z & Hoy WE 2003. Hypertension, dyslipidemia, body mass index, diabetes and smoking status in Aboriginal Australians in a remote community. Ethnicity and Disease 13(3):324–30.

Weih LM, VanNewkirk MR, McCarty CA & Taylor HR 2000. Age-specific causes of bilateral visual impairment. Archives of Ophthalmology 118:264–9.

Wells JE, Browne MAO, Scott KM, McGee MA, Baxter J & Kokaua J 2006. Prevalence, interference with life and severity of 12 month DSM–IV disorders in Te Rau Hinengaro: the New Zealand Mental Health Survey. Australian and New Zealand Journal of Psychiatry 40(10):845–54.

WHO (World Health Organization) 2011. Haemoglobin concentrations for the diagnosis of anaemia and assessment of severity. Vitamin and Mineral Nutrition Information System. Geneva: WHO. Viewed 26 August 2014, <www.who.int/vmnis/indicators/haemoglobin.pdf>.

WHO 2016. International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10). Viewed 19 July 2016, <http://apps.who.int/classifications/icd10/browse/2016/en>.

Willis AW, Schootman M, Kung N & Racette BA 2013. Epidemiology and neuropsychiatry manifestations of young onset Parkinson's disease in the United States. Parkinsonism and Related Disorders 19(2):202–6.

Wilson D, Walsh P, Sanchez L & Read P 1998. Hearing impairment in an Australian population. Adelaide: Department of Human Services.

Xiang F, Lucas R, Hales S, Neale R 2014. Incidence of Nonmelanoma Skin Cancer in Relation to Ambient UV Radiation in White Populations, 1978-2012: Empirical Relationships. JAMA Dermatology 150(10):1063-1071. doi:10.1001/jamadermatol.2014.762.

Zimmerman A, Bernuit D, Gerlinger C, Schaefers M & Geppert K 2012. Prevalence, symptoms and management of uterine fibroids: an international internet-based survey of 21,746 women. BMC Women's Health 12(6):1–11.

Zubrick SR, Dudgeon P, Gee G, Glaskin B, Kelly K, Paradies Y et al. 2010. Social determinants of Aboriginal and Torres Strait Islander social and emotional wellbeing. In Purdie N, Dudgeon P & Walker R (eds.). Working together: Aboriginal and Torres Strait Islander mental health and wellbeing principles and practice. Canberra: Australian Government Department of Health and Ageing.

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Related publications

This report is complementary to:

- AIHW 2016. Australian Burden of Disease Study: impact and causes of illness and death in Australia 2011. Australian Burden of Disease Study series no. 3. Cat. no. BOD 4. Canberra: AIHW.
- AIHW 2016. Australian Burden of Disease Study: impact and causes of illness and death in Aboriginal and Torres Strait Islander people 2011. Australian Burden of Disease Study series no. 6. Cat. no. BOD 7. Canberra: AIHW.

All reports, online supplementary material and working papers associated with the Australian Burden of Disease Study 2011 can be downloaded for free from the AIHW website <www.aihw.gov.au/burden-of-disease/>.

The website also provides information on ordering printed copies.

This document provides a detailed description of the methods used to derive the fatal and non-fatal burden of disease (using the disability-adjusted life years, years lived with disability and years of life lost measures) for the Australian and Aboriginal and Torres Strait Islander populations for 2011 and 2003, as well as estimates of how much of the burden can be attributed to various risk factors . The report is targeted at researchers and epidemiologists, and those seeking to further understand results provided in the Australian Burden of Disease Study 2011.