

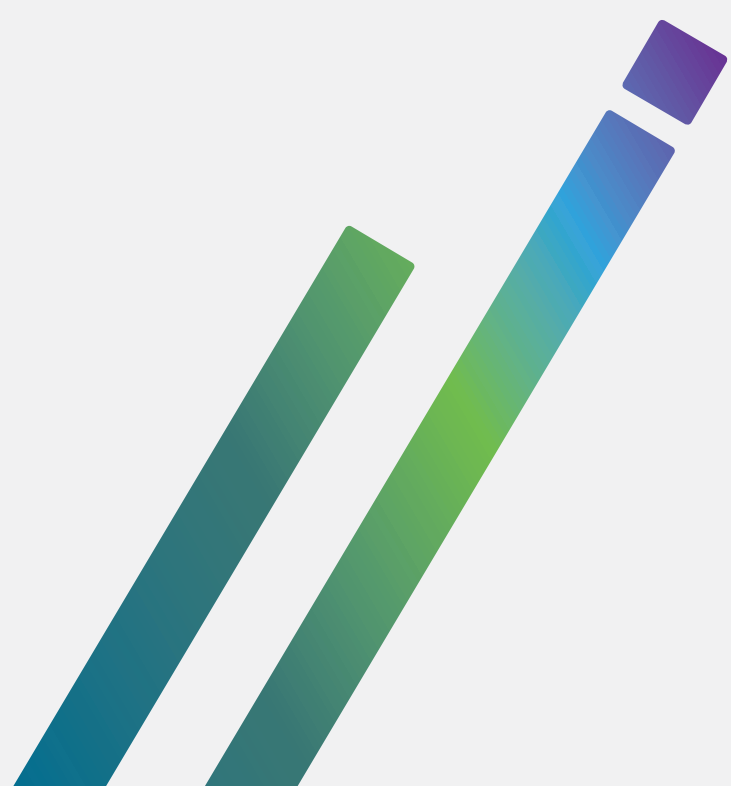


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# Australian Burden of Disease Study: methods and supplementary material 2015



**AIHW**





**Australian Government**  
**Australian Institute of  
Health and Welfare**

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# **Australian Burden of Disease Study**

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# 1 Introduction

Burden of disease analysis produces comparable and concise policy-relevant evidence on the impact of disease, injuries and risks on the population. A key strength of burden of disease is the ability to collate and use data from various sources to develop an internally consistent measure for all diseases. However, as methods used in burden of disease analyses have become increasingly complex over time, the increased complexity makes it much harder to explain the methods, and can result in decreased clarity for stakeholders.

This report describes, as far as practicable, the methods and assumptions used by the Australian Burden of Disease Study (ABDS) 2015 to quantify the fatal and non-fatal effects and causes of diseases and injuries in the Australian population in 2015, 2011 and 2003. It is a companion publication to *Australian Burden of Disease Study: impact and causes of illness and death in Australia 2015* (AIHW 2019a) and *Australian Burden of Disease Study: impact and causes of illness and death in Australia 2015—summary report* (AIHW 2019b) and was developed to provide transparency of data, assumptions and methods. This report supersedes the methods described in the *Australian Burden of Disease Study 2011: Methods and supplementary material* (AIHW 2016a) for the national component of the ABDS 2011.

The ABDS 2015 does not estimate the burden of disease and injuries on the Aboriginal and Torres Strait Islander populations as key data sources were not available at the time of analysis.

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

## Key considerations

The ABDS 2015 methods build on the methodological approach of the ABDS 2011 (AIHW 2014a; AIHW 2016a), along with methodological developments used in recent iterations of the Global Burden of Disease study (GBD 2015 and 2016). Key considerations for the ABDS 2015 were the need for:

- national estimates which were relevant to Australia, while maintaining comparability with global methods as much as possible
- sub-national estimates (state/territory, remoteness and socioeconomic group)
- comparability to 2011 and 2003 estimates to enable valid comparisons over time.

In addition, the following principles were followed to enable improvements and extensions to the methods used in ABDS 2011 (Box 1.1).

### **Box 1.1: Principles for the ABDS 2015 update**

- If changes were made to the ABDS disease list, methods or model inputs, estimates for previous time points were re-generated to enable true comparison over time.
- Changes to key inputs (such as disability weights or reference life table) or methods (such as redistribution or comorbidity bias adjustment) must not introduce bias or compromise the consistent and systematic approach for all diseases which is the foundation of the ABDS.
- Changes to models, model inputs or data sources must:
  - be introduced to improve accuracy and/or defensibility and be evidence-based
  - take into consideration the appropriateness of the change to previous time points. For example, changes in duration of health loss must consider whether it is appropriate to apply that change to all time points, or only the most recent time point. Changes in duration for more recent time points reflect advances in treatment; ultimately reducing the time spent in ill-health.
- Variations to the list of diseases/injuries must:
  - comply with criteria developed for selection of diseases and injuries in the ABDS (see Chapter 2)
  - maintain the existing disease list structure
  - maintain mutual exclusivity
  - be consistent with diseases used in the risk factor component.
- Variations to the risk factors list must:
  - comply with criteria developed for selection of risk factors in the ABDS (see Chapter 7)
  - be consistent with the disease list (including sequelae) in terms of the associated linked diseases.

## **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 H1) throughout the ABDS 2015.

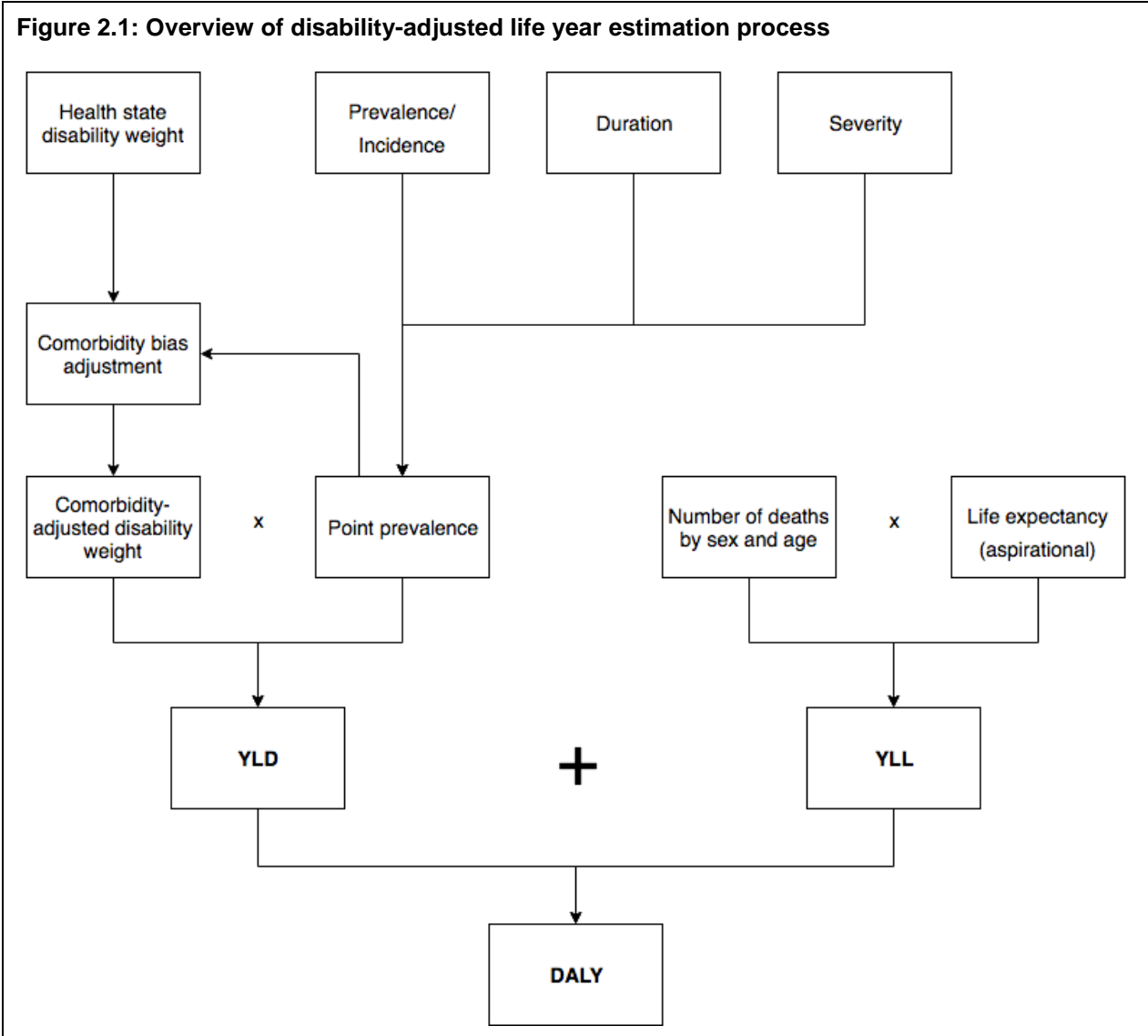
In the ABDS 2011 each disease group had an expert panel of people with relevant clinical and epidemiological expertise. For the ABDS 2015, panel members or key experts provided advice where methods were revised from those used the ABDS 2011. Membership is shown in Appendix tables H2 and H3.



# 2 Overarching methods and choices

The ABDS 2015 measured health loss using a summary measure of health called the disability-adjusted life years (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.



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 2015, 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 reference life table used in the GBD 2010 and 2013 studies, 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 the disabling effects of ill health). 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 which reflects the severity of 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 consequence. The disability weights used in this study were drawn from the GBD 2013 study, hereafter referred to as the GBD 2013 (see GBD 2013 Collaborators 2015), and represented the health loss caused by the consequences of each disease. Disability weights are further adjusted for comorbidity.

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 DALY: 1 DALY represents the loss of 1 year of healthy life.

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

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 the ABDS 2011 and 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.

Constructed this way, DALY 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.

## Reference years 2015, 2011 and 2003

Based on the availability of data at the start of the study, 2015 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 2015 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 2015.

Although 2015 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 estimates for 2011 were originally presented in *Impact and causes of illness and death in Australia 2011* (AIHW 2016b) while estimates for 2003 were originally presented in separate publications by Begg and others (2007) and Vos and others (2007) and revised for the ABDS 2011. While overarching methods for estimating disease burden remained unchanged from the ABDS 2011, some disease-specific methods in the ABDS 2015 differed considerably from the ABDS 2011. Therefore, revision of 2011 and 2003 estimates were 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 previous 2003 and 2011 estimates with new 2015 estimates.

## Reference populations

All Australian population-based rates for 2015 were calculated using populations rebased to the 2016 Census (released 27 June 2017) (ABS 2017a).

Population-based rates for 2011 were calculated using final population estimates from the 2011 Census (released 15 December 2016).

The Australian 2001 standard population (published 15 December 2016) was used for all age-standardisation, as per the Australian Institute of Health and Welfare (AIHW) and ABS standards (ABS 2016).

## 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 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 2015, 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 uses the following hierarchical framework:

**Disease groups:** 17 disease groups of related diseases or conditions—such as cardiovascular diseases, gastrointestinal disorders, or injuries—and one alternative reporting disease group (nature of injury instead of injury by external cause).

**Diseases:** 216 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 conditions are mutually exclusive (non-overlapping) including two perspectives for reporting injuries: by external cause or nature of injury.

The ABDS disease list is collectively exhaustive, meaning it covers the full spectrum of disease and injuries.

## Selection of diseases and injuries

The ABDS disease list is an Australian-specific disease list developed to reflect the needs of health reporting and monitoring in Australia. For this study, the ABDS 2011 disease list was reviewed, and modifications made based on a set of inclusion criteria originally developed and applied in the ABDS 2011.

For inclusion in the ABDS 2015 disease list, the condition or injury must meet at least one of the following guiding principles:

#### **Included in other studies' disease (or cause) lists**

- Have been included in:
  - the GBD study for 2015 or 2016 or
  - the Australian Burden of Disease Study (ABDS 2011) (AIHW 2016b)unless its inclusion in the ABDS 2015 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 a 4-year period, or as having a 'significant' primary care impact, as determined by expert judgment (ensuring the list is not overcome 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 high 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 216 diseases, conditions and injuries (including residual conditions—see 'Residual conditions' section) were selected and agreed on by the Australian Burden of Disease Expert Advisory Group to form the basis of the ABDS 2015. This includes 13 conditions describing the nature of injury used for alternative reporting (see 'Injuries' in Chapter 5). For the full list of diseases, conditions and injuries, see Appendix table A2.

As such, the ABDS 2015 disease list will differ from that used in other studies.

#### **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 32 residual ('other') categories distributed across the 17 disease groups and another 2 in the alternative reporting group for injuries (nature of injury). Diseases assigned to residual categories are listed in Appendix table A2.

In the ABDS 2015, there are new diseases that were previously reported in residual groupings (see Box 2.1).

### **Box 2.1: Key changes in the list of diseases and injuries since the 2011 Australian study**

The following diseases were disaggregated:

- Diabetes into type 1 and type 2 diabetes
- Leukaemia into 5 subtypes
- Mouth & pharyngeal cancer into lip & oral cavity cancer, nasopharyngeal cancer and other oral cavity & pharynx cancers
- Vision loss into 5 disorders—refractive errors, cataract & other lens disorders, glaucoma, age-related macular degeneration and other vision disorders.

The following diseases were removed from residual groupings and estimated separately:

- Road traffic injury – pedestrians
- Road traffic injury – pedal cyclists
- Urinary tract infections
- Mumps
- Interstitial nephritis.

Revised conceptual models for some diseases in line with changes to the disease list or new evidence.

### **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 2015**—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 global studies 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 global studies but was separately estimated in the NZBDS based on hospitalisations (however, it was noted it would be an underestimate). In the ABDS 2015, the burden of FASD experienced by the child was grouped under the disease ‘brain malformations’ in infant & 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). Anti-microbial 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 2015, 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 classifications 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 216 diseases in the ABDS 2015 disease list was based on the ABDS 2011 (AIHW 2016a) with expansion of some diseases. The ABDS 2011 disease list was informed by the code allocation used by the GBD 2010 study (hereafter referred to as the GBD 2010), the NZBDS 2006 and the ABDS 2003 (Begg et al. 2007).

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 is consistent with global studies 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 explored the overlap between these diseases to quantify their indirect impacts and collective burden. Results from these studies were published in the report *Diabetes and chronic kidney disease as risks for other diseases* (AIHW 2016c).
- **Classify diseases according to Australian disease monitoring activities**. Australian disease monitoring classifications were given priority over the GBD to provide better information for Australian health priority setting. For example, the 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 the ABDS 2015 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 GBD 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 & maternal disease group, rather than endocrine disorders, due to this condition only arising during pregnancy, and is consistent with previous national and GBD studies.
- **cerebral palsy**—the burden was allocated to the infant & 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 under 5. As a sequela, cerebral palsy is acquired through several other infant & congenital conditions, such as birth trauma and birth asphyxia.
- **fetal alcohol spectrum disorders (FASD)**—although counted under mental health and substance use disorders in the GBD 2010, the burden was assigned to infant & congenital conditions in the ABDS 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 the ABDS 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 & substance use disorders disease group. This is consistent with previous national and GBD 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 guidelines used to direct data selection). Only data sources that met the guidelines 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.

There are new data sources for many diseases in the ABDS 2015, notably greater use of linked hospital/deaths data.

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 sub-national estimates

Sub-national 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, Western Australia, South 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 (ASGS), 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. This study reported estimates for 4 remotes areas: *Remote* and *Very remote* were combined.
- **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’. The 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 (IRSD) historically being the most commonly used at the AIHW for health-related analyses. For more information on SEIFA, go to [www.abs.gov.au/websitedbs/censushome.nsf/home/seifa?opendocument&navpos=260](http://www.abs.gov.au/websitedbs/censushome.nsf/home/seifa?opendocument&navpos=260). SEIFA was only used for disaggregation of national estimates.

## Sub-national methodology

Sub-national 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 sub-national data sources are combined.

The preferred approach for sub-national estimates was to derive sub-national 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 sub-national 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 sub-national unit. This assumed no difference in disease prevalence rates between sub-national and national populations.

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

### **Key considerations**

The validity of sub-national 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, 1–4, 5–9, ..., 85+ to overcome limitations with data.

## **Methodological choices specific to 2003 and 2011 estimates**

Comparable YLL, YLD, DALY and attributable burden estimates were produced for each disease for the national population. Sub-national estimates for 2003 were not within the scope of this study.

As the 2003, 2011 and 2015 estimates are point-in-time estimates, their comparison with each other does not constitute a time-series analysis. Several issues must be considered before analysing and interpreting time trend data. A key issue is that 3 points in time can provide misleading information about changes over time—assuming that there is a straight-line trend between these 3 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, 2011 and 2015 data that have an impact on the interpretation are highlighted in the relevant chapters in this report.

### **2003 and 2011 methodology**

Where there were no changes in methods or data sources, the 2011 and 2003 estimates from the ABDS 2011 were kept the same. If there were changes in methods or data source for 2015, the estimates for 2011 and 2003 were re-estimated using the new methods to keep comparability across the three years.

Specific details on methods for 2011 and 2003 estimates for mortality, morbidity and risk factors are included in chapters 3–5 and 7–8.

### 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 reference period. In the ABDS 2015, YLL estimates were based on deaths that occurred in the reference years: 2003, 2011 and 2015.

Deriving YLL requires both:

- mortality data – the actual number of deaths and the ages at which those deaths occurred; and
- a reference life table – a measure of life expectancy at each age to derive 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 with 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.

**YLL (years of life lost):** 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 for estimating YLL is to compile all deaths by age and disease. Deaths are aligned to the study's disease list using the cause of death.

YLL is then calculated for each disease using single year of age at death. Each death is weighted according to the remaining potential life expectancy at that age of death using the reference life table.

The weighted deaths are summed, and the result is the total number of years of life lost. For YLL from all causes, this is described mathematically as:

$$YLL = \sum_{ai} D_{ai} \times W_a$$

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).

## Mortality data

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 Marriage in each state and territory and from the National Coronial Information System managed by the Victorian Department of Justice and coded to the International Classification of Disease (ICD) by the Australian Bureau of Statistics (ABS). The AIHW website <https://www.aihw.gov.au/about-our-data/how-we-use-our-data/deaths-data/> provides detailed information on the registration of deaths and coding of causes of death in Australia (AIHW 2018a). The completeness, accuracy and coding of these data are described elsewhere (ABS 2018a, AIHW 2018a). The deaths data are collated by the ABS into an administrative data set for statistical analysis. The AIHW houses a set of these data in the AIHW's National Mortality Database (NMD). The data quality statements underpinning the AIHW NMD 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](http://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](http://www.abs.gov.au/ausstats/abs%40.nsf/mf/3303.0).

All deaths data used in the ABDS 2015 were extracted from the AIHW's NMD. This is a register of all deaths in Australia since 1964, sourced from the cause of death unit record files as described above. 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. Despite completeness, causes of death that do not directly align to the study's disease list need to be reassigned to a disease in the list (see 'Redistribution of deaths').

Cause of death data for deaths occurring in 2003, 2011 and 2015 were used for this analysis. Deaths for the three reference years were extracted from the NMD for deaths registered in 2003 up to and including deaths registered in 2016. As a result, the analysis set includes deaths that occurred in 2015 but were not registered until 2016; on average, between 4% and 7% of deaths that occur in a given year are not registered until a later year—most of these in the following 2 years (ABS 2017c).

Deaths for the 2003 and 2011 reference years are almost all (at least 99%) based on a final version of cause of death data and most (95%) for 2015 are from a revised version of data. Since 2006, deaths certified by a coroner undergo revision and causes of death may be updated, pending the status of coroner investigation. As such, some cause of death information is subject to change. The ABS revisions process is described in detail elsewhere (ABS 2017c).

## 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 are then ascribed to diseases in the ABDS disease list (as described in Chapter 2).

Deaths data used in the ABDS 2015 are coded to the ICD-10 (ABS 2018a; WHO 2016). The procedure for assigning ICD-10 coded deaths records to items in the ABDS disease list is set out in Chapter 2.

Some ICD-10 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 the section '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 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 2015.

## **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 and unspecified diabetes.

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 and were reassigned to digestive cancers only. All deaths assigned to a group were redistributed using the same algorithm.

The redistribution groups used in the ABDS 2015 largely align with those used in the ABDS 2011, except for the addition of unspecified diabetes. Appendix table B1 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. Each death identified for redistribution may be reassigned in portions to 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 coded with inappropriate or invalid codes, by exploiting available evidence of a plausible alternate cause

of death. The ABDS 2015 has extended these methods using Australian-specific data and Australian-specific direct evidence.

Three methods were used for redistribution in the ABDS 2015:

- **Direct evidence:** This method uses direct evidence about particular deaths or causes of death—obtained through data linkage studies or extracted from sources other than the NMD—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 reassigns 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 MCO (or a combination of both). In the ABDS 2015, 86% of redistribution was based on one of these methods. 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 (14%) were redistributed using this method (Appendix table B1).

### 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 2015, 16,433 deaths were identified for redistribution in the 2015 reference year, equating to 204,493 YLL (Table 4.2). This amounted to 11% of deaths and 9% of YLL. The number and per cent of deaths redistributed and the associated YLL for each reference year is in Table 3.1.

**Table 3.1: Number and per cent of deaths and YLL, total and redistributed, by reference year**

Reference year	Total deaths	Deaths for redistribution	Per cent of total deaths	Total YLL	YLL for redistributed deaths	Per cent of YLL redistributed
2003	131,992	13,946	10.6	2,219,535	195,881	8.8
2011	146,756	16,711	11.4	2,271,682	215,906	9.5
2015	157,162	16,433	10.5	2,358,383	204,493	8.7
<b>All years</b>	<b>435,910</b>	<b>47,090</b>	<b>10.8</b>	<b>6,849,600</b>	<b>616,280</b>	<b>9.0</b>

The number of deaths identified for redistribution varied with age (Appendix table B2). They generally followed the patterns of age at death for all causes of death tabulations for Australia. For example, most redistributed deaths occurred among older people.

Appendix table B3 shows the number of deaths classified to disease groups before and after redistribution. The largest numbers of deaths gained by redistribution were for:

- cardiovascular (5,081 more deaths, an increase of 11%)
- cancer (4,956 more deaths, an increase of 11%)
- endocrine (1,648 more deaths, an increase of 46%).

Note the large apparent 'gain' in deaths for endocrine disorders was due to deaths coded to unspecified diabetes being reassigned to Type 1, Type 2 and Other diabetes.

The largest proportional gains, other than described above, were for:

- skin (99 more deaths, an increase of 16%)
- gastrointestinal (986 more deaths, an increase of 16%)
- injuries (1,457 more deaths, an increase 13%).

The impact of redistribution on YLL is also shown in Appendix table B3. The largest number of YLL gained was for:

- cancers (72,505 more YLL, a 9% increase)
- cardiovascular (43,881 more YLL, a 9% increase)
- injuries (26,650 more YLL, an 8% increase).

Other large percentage gains in YLL were for:

- endocrine disorders (22,618 more YLL, a 48% increase)
- skin (862 more YLL, a 14% increase)
- gastrointestinal (10,208 more YLL, a 10% increase).

Note that the majority of these increases were based on targeted redistribution using direct evidence or indirect MCODE. To illustrate the method 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 2015 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 B3 shows 42,083 deaths were coded to a cancer in the ABDS disease list. After redistribution, there were 47,039 cancer deaths, reflecting a gain of 4,956 deaths, or an additional 11%.

Appendix table B1 shows that non-specific cancer deaths were reassigned to specific cancers using the direct evidence method, and that the target diseases were all in the cancer disease group. In 2015, 2,707 deaths were coded to a non-specific type of cancer, and 1,237 deaths were coded to a non-specific digestive cancer. So, in total, 3,944 non-specific cancer deaths were identified for redistribution into a cancer cause.

So far, 80% of the overall gain in cancer deaths (3,944 out of the overall 4,956) has come from deaths initially coded to (non-specific) cancer-related causes, which have been redistributed into (specific) cancers in the ABDS disease list.

Appendix table B1 also shows a further 1,202 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 deaths consistent with the proportion of cancer deaths (identified pre-redistribution) were reassigned to cancers in the ABDS disease list. As can be seen from Appendix table B3, pre-redistribution, 27% of deaths were cancers, so about 27% of the 1,202 deaths (equivalent to around 322 deaths) were also redistributed to a specific cancer.

The foregoing redistribution steps account for around 86% of the overall gain in cancer deaths (3,944 plus 322 deaths).

The remaining 14% of the gain (690 cancer deaths) came 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 B1.

## **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 years 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 tables, which summarise 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, because it can be used across population groups and over time. 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 2015 uses the standard reference life table used in the GBD 2010 and 2013 (Murray et al. 2012) when calculating YLL for the Australian and sub-national populations. The standard reference life table has a life expectancy at birth of 86.0 years.

The most recent global estimates of YLL are based on a newer life table—the Theoretical Minimum Risk Life Table (TMRLT) (GBD 2016 Causes of Death Collaborators 2017). This life table is based on the lowest observed age-specific mortality rates from locations with total populations greater than 5 million. From this life table, life expectancy at birth is 86.6 years and 1.6 years at age 105 (the limit of the standard reference life table) and 1.4 years at age 110.

When preparing this report, the TMRLT was only available in an abridged format; that is, where life expectancy is reported for five-year age groups. YLL estimates are best made using a life table that describes life expectancy at each single year of age. Using an abridged version results in less accurate YLL (unpublished AIHW analysis of the NMD), therefore the standard reference life table was used for calculating YLL in the ABDS 2015.

### **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, the GBD selected the lowest age-specific death rate observed in any of the countries the study covered, excluding 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 B4 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
- 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 residents of major cities and very remote areas, 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 2014–2016 was 80.4 and 84.6 years, respectively—lower than the aspirational life expectancy of 86.0 years used in both the GBD and the ABDS. Life expectancies for Australian males and females were also lower than the GBD standard in 2010–2012 (79.9 and 84.3 years, respectively) and in 2002–2004 (78.1 and 83.0 years, respectively). For comparison, life expectancies in the GBD 2010 standard life table and for the Australian population for 2002–2004, 2010–2012 and 2014–2016 are shown for selected ages in Appendix Table B5.

## Sub-national 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 based on the remoteness area in each death record in the NMD. Remoteness area 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 2014a). In this study, remoteness areas were aligned to the ABS 2011 geography standard, including deaths that were registered in 2016 but occurred in 2015.

Deaths where there was insufficient information to ascribe a remoteness area were excluded from the sub-national analysis. These amounted to less than 0.5% of deaths in any one reference year.

### Socioeconomic group

As discussed in Chapter 2, the ABDS did not have information on socioeconomic status at the individual level. Instead, the ABDS 2015 derived socioeconomic group from the Index of Relative Disadvantage of the SEIFA index, which is 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 excluded from the analysis. These amounted to less than 0.5% of deaths in any one reference year.

## 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 (ill health).

YLD estimates in the ABDS 2015 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 adjustment for 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. Each **sequela** is linked to a specific health state—this may be a single health state or multiple health states to account for severity. For example, heart failure is a sequela of coronary heart disease and has 3 severity levels of mild, moderate and severe. Each health state is mapped to a **disability weight** which reflects the severity of health loss.

**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 is a sequela of 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 the ABDS 2015 are reported for 216 diseases, including two reporting categories for injuries—‘External cause of injury’ and ‘Nature of injury’, that constitute the disease list for the study (see Appendix Table A2).

YLD estimates are achieved using the following steps:

1. Develop a conceptual model for each disease, which includes main sequelae of the disease and severity of sequela (if required).
2. Map each sequela/severity to a health state and disability weight for all diseases
3. Estimate point prevalence by age and sex for each sequela/ severity.
4. Calculate YLD for each disease, which is estimated up from the sequela level (for each age and sex), described as:

$$YLD = \sum_i PP_i \times DW_i$$

where:

$\sum_i$  is the sum over all sequelae

$i$  is an index for sequela

$PP_i$  is the point prevalence of sequela  $i$

$DW_i$  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).

YLD estimates are also adjusted to account for comorbidity. Further detail for each step and the process of adjusting for comorbidity is described below.

## 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 2015 are provided at the health state level, these epidemiological models needed to be converted into simpler conceptual models. These models describe the significant outcomes (sequelae) of each disease, 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 for the ABDS 2015 analysis.

The conceptual models were developed by the AIHW in conjunction with disease experts. In many cases, a conceptual model was based on 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.

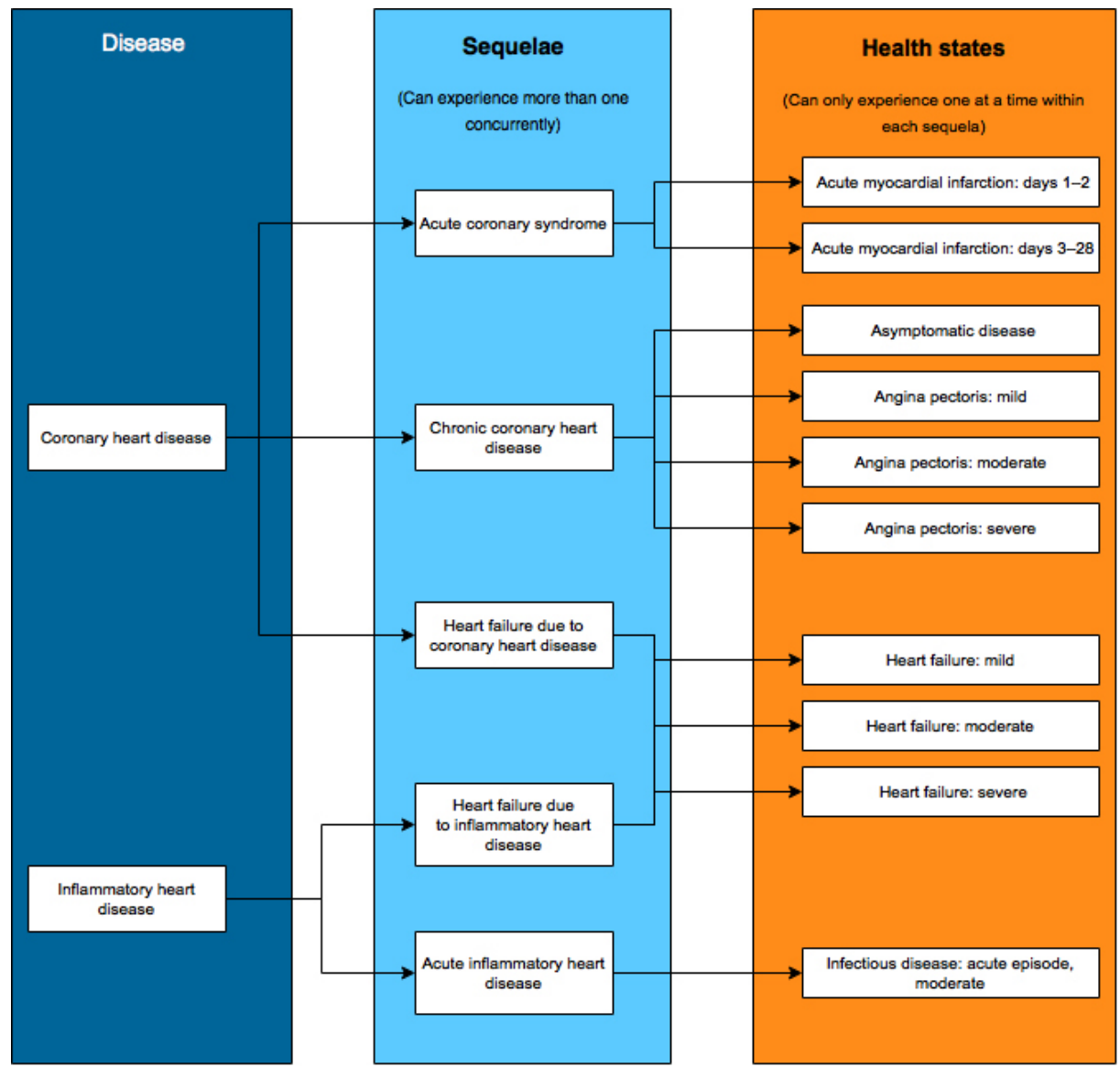
Within a single disease, a person could have any number of sequelae simultaneously—for example, a person experiencing health loss from diabetes might at the same time have health loss from diabetic foot and vision impairment due to diabetes. A person might also have multiple sequelae from multiple diseases 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. Disability weights express the health loss on a scale from 0 (no health loss) to 1 (total health loss).

To provide a set of weights for such large numbers of sequelae, the GBD 2010 pioneered 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. These were originally derived for the GBD 2010 from a large, multinational, cross-cultural study (Salomon 2010; Salomon et al. 2012) and further refined for the GBD 2013 (GBD 2013 Collaborators 2015). The GBD 2013 disability weights were used in the ABDS 2011 and 2015.

The 315 sequelae in the ABDS 2015 were mapped to 196 of the 236 available health states (see Appendix Table C1). This resulted in 675 sequela–health state combinations that included the different severity levels (such as mild, moderate and severe).

## 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 2015 estimated point prevalence as at 30 June 2015, 30 June 2011 and 30 June 2003.

The YLD estimation requires point prevalence at the sequela–health state levels 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 sequela–health state levels 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 2015. This study drew on the findings of meta-analyses done for the 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 (AHS) 2011–12 and the National Health Survey (NHS) 2014–15 held by the ABS. For further information on these data sources, including data quality statements, see <https://www.aihw.gov.au/about-our-data/our-data-collections/national-hospitals>, <https://www.aihw.gov.au/about-our-data/our-data-collections/australian-cancer-database>, [www.abs.gov.au/australianhealthsurvey](http://www.abs.gov.au/australianhealthsurvey) and

<http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/by%20Subject/4363.0~2014-15~Main%20Features~Data%20quality~61>.

Primary data sources used for each disease are summarised in Appendix Table C2.

To estimate point prevalence, the ABDS needed data relating to people rather than clinical events. The NHMD was a key data source for some diseases. However, since it provides counts of the number of hospital separations rather than the number of individual patients, the dataset created for the National Data Linkage Demonstration Project (NDLDP) was used to calculate people-to-hospitalisations ratios using linked New South Wales and Victorian hospital and deaths data for selected sequelae. This ratio was then applied to corresponding hospitalisation counts by sex and age from the NHMD to derive a count of people. This approach assumed that the other states and territories have the same hospital presentation ratio as New South Wales and Victoria combined.

The National Data Linkage Demonstration Project (NDLDP) database was created under the auspices of the Australian Health Ministers' Advisory Council. It includes data for public hospitals from New South Wales and Victoria, and three national data sources—the Medicare Benefits Schedule (MBS), Pharmaceutical Benefits Scheme (PBS) and the National Death Index (NDI). The ABDS 2015 used the hospitals and the NDI data from 2010–11 to 2014–15. It did not use the MBS and PBS components of the database.

Linked hospitals and deaths data from Western Australia were also used to calculate people-to-hospitalisations ratios using linked data for selected sequelae, including cardiovascular diseases, respiratory diseases and amputations due to diabetes. This and other usage of linked data is described in the disease-specific methods (Chapter 5).

## **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 sequela in question.

Where there were no empirical data on the distribution of health states within a sequela, severity distributions were adopted from the NZBDS or the GBD 2013 (where used in the ABDS 2011) or GBD 2015, where available. Severity distributions from the GBD were considered global distributions, however they were 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.

## **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](http://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 prevalence of anaemia due to each of these diseases adjusted to ensure they summed to the overall prevalence (please see Chapter 5 for more detail).

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**

Accounting for comorbidity is an important process in the non-fatal estimation component of burden of disease studies. 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.

Whilst this would enable estimation of population level YLD that accounts for all possible combinations of morbidity, it would not be able to provide comorbidity adjusted YLD for an individual disease.

As comprehensive unit-record level comorbidity data and a full suite of combination disability weights does not exist, available prevalence data for each consequence of disease and derived disability weights are used. The available data are less than ideal, because:

- **prevalence** is 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.

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

- Consider the case of Jane Doe who has metastatic cancer (disability weight = 0.451), episodic 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 (per individual) of non-fatal 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 prevalence 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 prevalence 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 prevalence) that comorbidity-adjusted YLDs are derived.

## Comorbidity bias adjustment in the ABDS 2015

The strategy outlined above has been adopted for the ABDS 2015. 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 prevalence, the ABDS 2015 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 (Vos et al. 2012).
- For disability weights, the ABDS 2015 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 recent iterations of the GBD studies and other recent burden of disease studies.

Because disease prevalence 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 population at points in

time, comorbidity bias adjustment was undertaken separately for each of the reference years—2003, 2011 and 2015—using the prevalence specific to those years.

Assembling the simulated population entailed the following steps:

- 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 2015 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 is 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.
- The available data on single-condition disability weights (and the multiplicative assumption) was used to attach an adjusted disability weight to each combination of comorbid conditions, and, from there, to each population age and sex group.

The adjusted YLD that result from applying adjusted disability weights derived from the simulation are expected to be a reasonable approximation to the ideal aggregate YLD (and comorbidity-adjusted YLD 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 the assumptions regarding independence. Validation studies by the GBD and the New Zealand Ministry of Health suggest that the approximations using a multiplicative model appear reasonable at aggregate level (NZMOH 2012; Vos et al. 2012). 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 YLD:YLL ratio estimated for other conditions in that disease group (at the age and sex level) applied to known YLL. 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, neurological and respiratory 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 to estimate mortality and morbidity for each of the 17 disease groups (below, in alphabetical order). It also describes methods for the following conditions, which are sequela to multiple diseases (referred to as envelopes), within these disease group sections:

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

Detailed information is provided on the methods used for 2015 national estimates. Where these methods differ for sub-national estimates, 2011 or 2003 estimates, this is described separately.

### Blood and metabolic disorders

#### Mortality estimates

Deaths related to blood & 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 all diseases excluding injuries, reproductive & maternal conditions, oral disorders and hearing & vision disorders. Deaths coded to E86 and E87 were proportionally redistributed across all disease groups (excluding reproductive & maternal conditions, oral disorders and hearing & vision disorders) (Appendix Table B1).

#### Morbidity estimates

#### Sequelae and health states

Sequelae and health states assigned to blood & metabolic disorders are shown in Table 5.1. Assumptions are outlined in subsections for individual diseases.

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

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>
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 <sup>(b)</sup>	196, 197

(continued)

**Table 5.1 (continued): Sequelae and health states for blood & metabolic disorders**

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>
Iron-deficiency anaemia	Anaemia due to iron-deficiency anaemia <sup>(b)</sup>	195, 196, 197
Protein-energy deficiency	Stunting due to protein-energy deficiency	211
	Wasting due to protein-energy deficiency	210, 211
Other blood & metabolic disorders	Anaemia due to other blood & metabolic disorders <sup>(b)</sup>	197
	Non-anaemic deficiency due to other blood & metabolic disorders	195
	Immune suppression due to other blood & metabolic disorders	10
	Metabolic dysfunction due to other blood & metabolic disorders	31

(a) See Appendix Table C1.

(b) Part of anaemia envelope.

## Prevalence estimation

### Anaemia envelope

As an envelope in the ABDS 2015, the overall prevalence of anaemia was calculated to ensure the sum of estimates for sequelae do not exceed the total. Diseases that include anaemia as sequelae include iron-deficiency anaemia, haemolytic anaemia, uterine fibroids, chronic kidney disease, gastroduodenal disorders and maternal haemorrhage.

The following section describes the method used to calculate anaemia envelope estimates and anaemia due to iron-deficiency anaemia in the ABDS 2015. Methods for estimating sequelae from the other diseases are found in their respective disease groups.

#### *Prevalence estimation of the anaemia envelope*

Prevalence rate of individuals at risk of anaemia in ages 10 and over, by age and sex, were derived from self-reported data from the NHS 2014–15 (ABS 2017h). To account for individuals with undiagnosed anaemia, sex-specific ratios of self-reported: biomedical anaemia were estimated from the AHS 2011–12 (ABS 2013a) and applied to calculated prevalence rates from the NHS 2014–15. The AHS 2011–12 survey was used as biomedical data were not available from the NHS 2014–15. As well, the NHS did not report on *Very remote* areas, so prevalence was modelled to account for *Very remote* areas.

Similarly to the ABDS 2011, iron-deficiency anaemia was assumed to be prevalent among 4% of children aged under 1 (Oti-Boateng et al. 1998), 2% of children aged 1–4 (Looker et al. 1997; Mackerras et al. 2004), and 1% for children aged 5–10 (Sadler & Blight 1996).

To avoid double counting, the sum of estimates of anaemia as sequelae due to other disease were subtracted from the anaemia envelope estimates. This included estimates for anaemia due to haemolytic anaemia, uterine fibroids, chronic kidney disease and gastroduodenal disorders. Methods for estimating anaemia due to these conditions are described in their respective disease groups. Once all anaemia sequelae has been subtracted, the remainder results 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 NHS 2014–15 results.

### Cystic fibrosis

Prevalence of cystic fibrosis was derived from the *Australian Cystic Fibrosis Data Registry annual report 2015* (Ahern et al. 2017). Registrants by age, sex and severity (lung function) was obtained from the report for ages 0–60+ years.

As the severity age groupings in this report did not align to age groupings used in the ABDS 2015, the following assumptions were made:

1. 0–10 years were assigned the 6–11-year severity level
2. 10–19 years were assigned the 12–17-year severity level
3. 20–29 years were assigned the 18–29-year severity level
4. 30–60 years and over were assigned the 30-years-and-over severity levels.

Disaggregation into finer age groups was based on rates on hospitalisations with a diagnosis of cystic fibrosis.

The report details numerous markers for severity, but these conflict with other components of the ABDS 2015 (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 2015 included haemophilia A and B. Prevalence estimates and severity distribution were derived from the Australian Bleeding Disorders Registry 2015–16 report (National Blood Authority 2017).

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 proportions across all ages. Based on clinical advice, it was assumed 95% of females with haemophilia have mild and 5% have moderate haemophilia (Rowell 2015, pers. comm. 11 September).

### Haemolytic anaemia

The same disabling sequelae for haemolytic anaemia as used in the ABDS 2011 were used in this study (Table 5.2). Table 5.2 lists diagnosis and procedure codes (using the 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 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, data from linked hospitals and NDI components for the NDLDLP database 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 these states 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. Methods to estimate iron-deficiency anaemia are described previously.

Severity was based on haemoglobin level definitions for mild and moderate anemia (WHO 2011). The severity distribution used in the ABDS 2011 (derived from AHS 2011–12 biomedical data) was used for the ABDS 2015.

### **Protein-energy deficiency**

In this study, burden due to protein-energy deficiency was only estimated for elderly individuals and Indigenous children under 5, as these are the population group most likely to be affected in Australia. Burden among Indigenous Australians is out of scope of this study hence estimates for Indigenous children were not required. Prevalence estimates in elderly Australians.

Estimates of protein energy deficiency in elderly Australians are restricted to individuals 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 showed that 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 at a state and remoteness level (AIHW 2012a, AIHW 2012b).

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 overestimated.

### **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, separations based on principal diagnosis 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**

<b>Sequelae</b>	<b>ICD-10-AM codes</b>	<b>Duration</b>
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

### **Sub-national estimates**

State and territory prevalence estimates for blood & 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 2015.

### **2011 and 2003 estimates**

2011 and 2003 estimates were based on the same method as for 2015.

Hospital separations were derived from the 2011 and 2003 calendar year.

Registrant data from 2011 and 2003 were used to estimate haemophilia and cystic fibrosis prevalence in the respective year. Where age and sex or severity distributions were unavailable, these were obtained from reports closest to the reference year that provided this information.

Total iron-deficiency anaemia prevalence estimates for 2011 were derived from the biomedical data from the AHS 2011–2012. Estimates for 2003 were derived from self-reported estimates from the 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 for 2011 and 2003 used the same method as in 2015 but was based on the number of people living in permanent residential aged care facilities or receiving in-home care services in the respective years, by sex.



# 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’.

The same direct evidence algorithms were applied to all three reference periods.

## Morbidity estimates

### Sequelae and health states

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

**Table 5.4: General cancer-related sequelae and health states**

Sequelae	Health state	ABDS 2015 health state identifier <sup>(a)</sup>
Diagnosis and primary therapy phase of < cancer type >	Cancer: diagnosis and primary therapy	18, 208 <sup>(b)</sup>
Controlled phase of < cancer type > <sup>(c)</sup>	Generic uncomplicated disease: worry and daily medication	207
Metastatic phase of < cancer type > <sup>(d)</sup>	Cancer: metastatic	19
Terminal phase of < cancer type > <sup>(e)</sup>	Terminal phase: with medication	22

(a) See Appendix Table C1.

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

(c) Non-melanoma skin cancer model did not include controlled phase health state.

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

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

**Table 5.5: Long-term cancer sequelae and health states**

Disease	Sequelae	ABDS 2015 health state identifier <sup>(a)</sup>
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.

### General sequelae

Average durations for each general sequela for the various cancers were primarily taken from the GBD 2013, though a small number that were developed specifically for the ABDS 2011 based on expert advice were used in the ABDS 2015 (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 2014 ACD and projected to the year 2015. 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 2015 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 Victoria, Queensland and Western Australia, 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 Victoria, Queensland and Western Australia was applied to separations from other jurisdictions, to derive national and sub-national estimates. As no incidence data are available for 2015, incidence data for all jurisdictions was obtained by applying the age-specific ratio of incidence to separations from 2014 to separations for 2015.

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 2015 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 2015, there is no overlap with people who died that year. Prevalence data were sourced from the 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 the reference year was based on people who died from cancer in that year (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. Health loss experienced by people dying early in the following year is equal to health loss experienced in the preceding year by people dying early in the year.

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**

Long-term sequelae were assumed to apply to all survivors (not just those diagnosed in the previous 10 years) consistent with the GBD 2013 onwards. To enable comparison between all three time points, this life-time prevalence was truncated at 20 years as this is the longest prevalence available for 2003 (as cancer data starts in 1982). 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 2015 (derived from the ACD). This was applied to the 20-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 2015 (derived from 2014 ACD and projected to 2015). This ratio was applied to 20-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. The overall colostomy closure rate was obtained from the Western Australian Department of Health using linked hospitals data. This method assumes that the closure rate from Western Australia is consistent across Australia.

### *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 2014 ACD and projected to 2015. Age-specific ratios were applied to the 20-year prevalence of breast cancer for females; an overall ratio was applied for males.

As 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 2005–2015 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 (obtained from the Western Australian Department of Health) 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 20-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 2015 (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, urinary incontinence is assumed not to apply to children aged under 5.

#### *Stoma and urinary incontinence due to bladder cancer*

In the ABDS 2015, 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 20-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 and others 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 2015 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.

### **Sub-national estimates**

State and territory incidence and prevalence data were derived directly from the data—summing to create the national incidence and prevalence counts for each year.

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 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.

### **2011 and 2003 estimates**

Due to method changes for the ABDS 2015, all estimates for the years 2003 and 2011 were re-estimated using cancer incidence and prevalence derived from the ACD and cancer mortality from the NMD, for the reference years in the same way as for 2015.

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 estimated incidence of NMSC for the year 2003. Instead, incidence from the 2002 survey by Staples and others (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 2015.

Long-term sequelae were derived in the same way using 2011 or 2003-specific ratios. Where the NHMD was the primary data source, separations from the respective calendar year was used.

For brain injury due to malignant and benign brain tumours and central nervous system cancer, the same rates were assumed as for 2015 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 and 2015.

## **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 MCODE method to all diseases excluding injuries, reproductive & maternal conditions, oral disorders and hearing & vision disorders, and to selected cardiovascular and infant & congenital conditions, respectively. Deaths coded to cardiac arrest and cardiac conduction disorders were proportionally distributed across all diseases (except reproductive & maternal conditions, oral disorders and hearing & vision disorders), while deaths coded to unspecified atherosclerosis and cardiac signs and symptoms were proportionally distributed across all disease groups excluding cancer, injuries, infectious diseases, reproductive & maternal conditions, oral disorders and hearing & vision disorders.

### **Morbidity estimates**

Methods for cardiovascular diseases mostly remained the same between the ABDS 2011 and the ABDS 2015, with the exception of rheumatic heart disease (and consequently, non-rheumatic heart disease) and aortic aneurysm.

## Sequelae and health states

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 (Table 5.6). As these events are of short duration, point prevalence was estimated by applying the duration of health loss to incidence.

**Table 5.6: ABDS 2015 diseases and sequelae that use the NHMD to estimate point prevalence**

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>	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
Rheumatic heart disease	Acute rheumatic fever	3	84 days (3 months)
Inflammatory heart disease	Acute inflammatory heart disease	2	28 days
Aortic aneurysm	Symptomatic aortic aneurysm requiring repair	193, 194	28 days (ruptured)
			14 days (non-ruptured)
			2 days (endovascular stent/other surgery)

(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 2014b). 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 sub-national 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 the reference year in the NHMD.

Prevalence (incidence times duration) estimates were then split into the 5 severity levels using proportions obtained from the 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 rheumatic fever*

Hospitalisation data were chosen since the national register was in development at the time of analysis.

Incidence was calculated by counting the number of non-fatal separations due to acute rheumatic fever (defined as principal diagnosis of ICD-10-AM I00–I02) in the reference year in the NHMD. A duration of 84 days (or 3 months) was applied to estimate point prevalence. It was assumed that any readmission for acute rheumatic fever within a period of 84 days was likely caused by the same event. Hospitalisation ratios were calculated using linked hospitals and deaths data from the NDLDP database were used to account for potential readmissions within 84-days.

#### *Acute inflammatory heart disease*

Incidence was estimated by counting the number of separations due to acute inflammatory heart disease in the NHMD in the reference year. 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 as a principal diagnosis in a single year (AIHW analysis of Western Australian linked hospitalisation and deaths data sets; AIHW 2014b). 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. The same ratio was used for the 2015 reference year as it is assumed that the ratio would not have significantly changed between 2011 and 2015.

#### *Aortic aneurysm*

The conceptual model for aortic aneurysm was modified for the ABDS 2015 to allow for more specific estimation of the burden due to aortic aneurysm. Aortic aneurysm is an acute condition. Cases of aortic aneurysm are defined as hospitalised patients with a principal diagnosis of aortic aneurysm (ICD-10-AM I71) and having undergone a surgical repair. Point prevalence was estimated by applying the appropriate duration depending on whether it was a ruptured or non-ruptured aortic aneurysm and the kind of surgery (that is, open repair surgery, an endovascular stent or other surgery).

### **Chronic sequelae**

The prevalence 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 2015 diseases and sequelae that use a combination of the NHMD and Western Australian linked hospitalisations and deaths data to estimate prevalence**

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>
Coronary heart disease	Chronic coronary heart disease	26, 27, 28, 262
	Heart failure due to coronary heart disease <sup>(b)</sup>	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 <sup>(b)</sup>	31, 32, 33
Non-rheumatic heart disease	Valvular diseases due to non-rheumatic heart disease	207
	Heart failure due to non-rheumatic heart disease <sup>(b)</sup>	31, 32, 33
Hypertensive heart disease	Heart failure due to hypertensive heart disease <sup>(b)</sup>	31, 32, 33
Inflammatory heart disease	Heart failure due to inflammatory heart disease <sup>(b)</sup>	31, 32, 33
Cardiomyopathy	Heart failure due to cardiomyopathy <sup>(b)</sup>	31, 32, 33
Cardiovascular defects <sup>(c)</sup>	Heart failure due to congenital cardiovascular defects <sup>(b)</sup>	31, 32, 33

(a) See Appendix Table C1.

(b) Part of heart failure envelope.

(c) Included under infant & congenital conditions.

For sequelae that are considered chronic (this includes chronic coronary heart disease, chronic stroke, rheumatic heart disease and 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 the reference year. An 11-year look-back period was used to keep the method consistent with what was done for the ABDS 2011.

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 Western Australian linked hospitalisations and deaths data. These ratios were obtained from the Western Australian Department of Health.

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 the GBD 2013 (Burstein et al. 2015).

The prevalence of chronic stroke was broken down by severity using distributions from the 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.

#### *Atrial fibrillation and flutter*

The prevalence of all atrial fibrillation & 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 was estimated by counting the number of separations with atrial fibrillation listed as the principal diagnosis in the reference year 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.

**Table 5.8: ABDS 2015 diseases and sequelae that use the NZBDS prevalence rates**

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>
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.

### **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 Western Australian linked hospitalisations and deaths data to the national count of separations from the NHMD. These ratios were obtained from the Western Australian Department of Health.

As one of the envelopes in the ABDS 2015, the overall prevalence of heart failure from all diseases was calculated to ensure the sum of estimates for sequelae do not exceed the total. 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 the GBD 2013 (Burstein et al. 2015).

### **Sub-national estimates**

Where prevalence was obtained from the NHMD, sub-national estimates were derived directly by applying 2011 ASGS remoteness areas and 2011 SEIFA population-based IRSD 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 sub-national disaggregation from 2011 separations in the NHMD.

### **2011 and 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 and 2011 were largely similar to those for 2015. However, due to a change in the diagnosis classification and to 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 and 2011 prevalence estimates were the same as those used for 2015 estimates. For acute rheumatic fever, the hospital ratio used to adjust for readmission for 2015 estimates was used for 2011 and 2003 prevalence estimates. For atrial fibrillation and peripheral vascular disease, the NZBDS prevalence rates used for 2015 estimates were also used for 2011 and 2003 estimates.

## Endocrine disorders

### Mortality estimates

Endocrine disorder 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 & maternal conditions. Deaths due to diabetic nephropathy (E10.2, E11.2, E13.2, E14.2) were assigned to kidney and urinary diseases. Deaths due to unspecified diabetes were redistributed across type 1, type 2 and other diabetes.

### Morbidity estimates

#### Sequelae and health states

Unlike in the ABDS 2011, diabetes mellitus was disaggregated into type 1, type 2 and other diabetes for the ABDS 2015. Sequelae and health states assigned to endocrine disorders are shown in Table 5.9.

**Table 5.9: Sequelae and health states for endocrine disorders**

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>
Type 1 diabetes	Amputation due to type 1 diabetes	140
	Diabetic foot ulcer	39
	Diabetic neuropathy	40
	Diagnosed diabetes	207
	Vision impairment due to type 1 diabetes	114, 115, 116
Type 2 diabetes	Amputation due to type 2 diabetes	140
	Diabetic foot ulcer	39
	Diabetic neuropathy	40
	Diagnosed diabetes	207
	Vision impairment due to type 2 diabetes	114, 115, 116
Other diabetes mellitus	Diagnosed other diabetes	207
Other endocrine disorders	Other endocrine disorders	..

(a) See Appendix Table C1.

## Prevalence estimation

### Type 1 diabetes

#### *Diagnosed type 1 diabetes*

The prevalence of type 1 diabetes mellitus was sourced from the linked National Diabetes Services Scheme (NDSS) and Australasian Paediatric Endocrine Group (APEG) data set, which is comprised of data from the NDSS Registrant data, the NDSS Sales data and the APEG's state-based registers. The prevalence estimates were provided by the Cardiovascular, Diabetes and Kidney Unit of the AIHW. For more information on these data sets, refer to the National (insulin-treated) Diabetes Register 2017 Quality statement <https://meteor.aihw.gov.au/content/index.phtml/itemId/714433>.

#### *Diabetic neuropathy and foot ulcer*

The overall prevalences of diabetic neuropathy, diabetic foot syndrome and vision loss due to type 1 diabetes were obtained from phase 2 of the Fremantle Diabetes Study (Davis 2018, pers. comm., 8 March; Sämann et al. 2008). Prevalence estimates by sex and age were modelled using the national sex and age group distribution. Prevalence was modelled to start at age 20; this decision was informed by data from the NHMD.

#### *Amputation due to type 1 diabetes*

The prevalence of amputation due to type 1 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 type 1 diabetes if there was a principal or additional diagnosis of type 1 diabetes accompanying a lower limb amputation in the hospitalisation.

#### *Vision impairment due to type 1 diabetes*

Similar to diabetic neuropathy and diabetic foot syndrome, the prevalence estimates for vision impairment due to diabetes were calculated using results from phase 2 of the Fremantle Diabetes Study (unpublished data). Breakdowns by sex and age were modelled using 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 study by Wong and others (2009).

### Type 2 diabetes

#### *Diagnosed type 2 diabetes*

The prevalence of type 2 diabetes mellitus was sourced from self-report data from the NHS 2014–15, the AHS 2011–13 and the NHS 2004–05. Due to high Relative Standard Errors (RSEs) for a number of the younger and older age groups, prevalence rates by sex and 5-year age group were modelled.

Since the health surveys do not survey the *Very remote* areas, weighted counts of type 2 diabetes were inflated to include prevalence in *Very remote* areas.

#### *Diabetic neuropathy and foot ulcer*

The overall prevalence of diabetic neuropathy, diabetic foot syndrome and vision loss due to type 2 diabetes were obtained from phase 2 of the Fremantle Diabetes Study (Baba et al. 2015; WA Davis 2018, pers. comm., 7 March). Prevalence estimates by sex and age were modelled using the national sex and age group distribution. Prevalence was modelled to start at age 25; this decision was informed by data from the NHMD.

### *Amputation due to type 2 diabetes*

The prevalence of amputation due to type 2 diabetes was estimated using the same method as amputation due to type 1 diabetes—where a principal or additional diagnosis of type 2 diabetes accompanied a lower limb amputation.

### *Vision impairment due to type 2 diabetes*

The prevalence of vision impairment due to type 2 diabetes was estimated using the same method as for vision impairment due to type 1 diabetes. The same severity distribution was used.

### **Other diabetes mellitus**

The prevalence of other diabetes mellitus is difficult to estimate due to lack of robust national-level data. As such, the prevalence of complications due to other diabetes mellitus was not estimated.

The prevalence of other diabetes mellitus was estimated using the proportion of other diabetes from the Fremantle Diabetes Study (Davis et al. 2018). Sex by age distributions were obtained from the NHMD.

### **Other endocrine disorders**

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

### **Sub-national estimates**

Prevalence estimates by state/territory, remoteness area and socioeconomic group were derived from the same data source as the national estimates and modelled similarly.

### **2011 and 2003 estimates**

Type 1 and other diabetes prevalence estimates for 2011 were derived from the same data sources as the estimates for 2015.

For diagnosed type 2 diabetes, prevalence estimates were derived from the AHS 2011–12. The prevalences of diabetic neuropathy, diabetic foot syndrome, vision impairment and amputation due to type 2 diabetes was estimated using the same data sources used for 2015 estimates.

Type 1 and other diabetes prevalence estimates for 2003 were derived from the same data sources as the estimates for 2015.

For diagnosed type 2 diabetes, prevalence estimates were derived from the NHS 2004–05. The overall prevalence for diabetic neuropathy, diabetic foot syndrome and vision impairment due to diabetes were obtained from the AusDiab Study (Tapp et al. 2003a, 2003b). Breakdowns by sex and age were modelled using data from the NHMD. Amputation due to type 2 diabetes was estimated using the same data sources as for the 2015 estimates.

# 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 direct evidence and indirect MCODE method to chronic liver disease, gastroduodenal disorders and diverticulitis. Deaths coded to peritonitis (K65–K66) were also redistributed using direct evidence and indirect MCODE to gastroduodenal disorders, hernias, pancreatitis, gallbladder and bile duct disease, paralytic ileus & intestinal obstruction without hernia and inflammatory bowel disease. Toxic liver disease with acute hepatitis was redistributed proportionally to all causes (except reproductive & maternal conditions, oral disorders and hearing & vision disorders).

## Morbidity estimates

### Sequelae and health states

The sequela and health states assigned to gastrointestinal disorders are shown 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 2015 health state identifier <sup>(a)</sup>	Duration
Gastroduodenal disorders	Anaemia due to gastroduodenal disorder <sup>(b)</sup>	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)
Inflammatory bowel disease	Crohn's disease or ulcerative colitis	46	12 months
Diverticulitis	Diverticulitis	194	2 weeks (medical therapy)
			3 weeks (surgical intervention)
Diverticulitis (continued)	Stoma due to diverticulitis	21	12 months (permanent stoma)
			5.4 months (temporary stoma)
			10 months (with end-stage liver disease)

(continued)

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

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>	Duration
Chronic liver disease	Decompensated cirrhosis of the liver	44	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 (GORD)	Mild symptomatic gastro-oesophageal reflux disease	262	..
	Moderate/severe symptomatic gastro-oesophageal reflux disease	192	approx. 4 days/ week for 12 months
Functional gastrointestinal disorders (FGID)	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 2015, 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. However, 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 from the GBD 2013 were used for gastritis and peptic ulcers.

### **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 procedure—were assumed to be incident cases. The 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. The duration of health loss for patients with symptomatic hernia until repair was assumed to be 12 months. This was based on the NZBDS's estimate of duration which accounts for 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 the GBD 2013), and 2 days for those undergoing minor intervention based on expert advice.

Experts also advised that minor surgery should account for the majority of procedures to relieve intestinal obstruction; however, investigation of inpatient hospitals data showed that major surgery was performed in 5 times as many separations as minor surgery. This may 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. Duration of health loss was assumed to be 6 weeks which is consistent with the GBD 2013.

### **Pancreatitis**

Acute cases of pancreatitis were defined as hospitalised patients with a principal diagnosis of acute pancreatitis (ICD-10-AM K85). Patients with acute pancreatitis are incident cases of short duration. 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). Patients with chronic pancreatitis are prevalent cases. 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, and the duration of temporary stomas.

## **Chronic liver disease**

Chronic liver disease is a progressive disease with different stages and severity (and therefore multiple sequelae). 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.

Data from linked hospitals and NDI components for the NDLDLP database 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.

Data from linked hospitals and NDI components for the NDLDLP database 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. Estimates of the number of individuals that received a liver transplant due to chronic liver disease for the reference year were obtained from the Australian and New Zealand Liver Transplant Registry.

## **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 the GBD 2013, and applied by the ABDS 2015, 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 Sydney inflammatory bowel disease cohort study (Selinger et al. 2013), which derived prevalence using hospitals and gastroenterologists' data. This is the most recently published study that used a similar method to other relevant studies that were done previously (Gearry et al. 2006; Studd 2013). The study draws on a population that is generalisable to the Australian population.

## **Gastro-oesophageal reflux disease**

Gastro-oesophageal reflux disease (GORD) (which includes hiatal hernias) is largely a chronic disease treated in response to symptoms. The major symptoms include heartburn, acid reflux and difficulty swallowing. This condition was first included in the ABDS 2011 due to the reportedly high morbidity.



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

Total prevalence of moderate or severe GORD was based on published estimates by Harrison et al. (2013), which estimated the national prevalence of GORD as 7.5%. This prevalence rate was applied to the population aged 15 and over. Secondary data sources were required to inform age–sex distributions.

Age and sex distributions for GORD in males and females aged 15 and over were derived from the study by Miller and others (2015), which estimated age-specific rates of general practice consultations with a GORD diagnosis in 2012–14.

For GORD in males and females aged under 15, prevalence rates were derived from general practice data for the year 2008-09 (as used in the ABDS 2011) and applied to the 2015 Australian Estimated Residential Population, as more recent data was unavailable at the time of the study.

### **Functional gastrointestinal disorders**

Functional gastrointestinal disorders have not been included in previous Australian or GBD studies until the ABDS 2011 and is also included in the ABDS 2015. Functional gastrointestinal disorders are common disorders characterised by persistent and recurring gastrointestinal symptoms. 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 the Rome III criteria (Rome Foundation 2006), which impose strict criteria that must be met for functional symptoms to be classed as pathological.

As there were limited updated data available on the prevalence rates of functional gastrointestinal disorders, the method used in the ABDS 2015 were similar to that used in the ABDS 2011 (described below).

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, the ABDS 2015 estimates were based on the study by Boyce and others (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 the ABDS 2015, as in the ABDS 2011. Estimates for children and adolescents were based on international studies by Chitkara and others (2005) and Helgeland and others (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 YLD:YLL ratio for all gastrointestinal disorders (except gastro-oesophageal reflux and functional gastrointestinal disorders) combined to the YLL for other gastrointestinal disorders.

### **Sub-national estimates**

Estimates derived directly from the NHMD were broken down by state/territory, and by remoteness area and socioeconomic group by applying the 2011 ASGS remoteness areas and the 2011 SEIFA population-based IRSD 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.

### **2011 and 2003 estimates**

The same methods used for 2015 estimates were used to estimate point prevalence for each of the diseases in the gastrointestinal disorders group for 2011 and 2003, using 2011 and 2003 hospitalisations data and populations.

## **Hearing and vision disorders**

### **Mortality estimates**

Deaths from hearing & vision disorders were treated as implausible causes of death. Deaths in the NMD related to hearing & vision disorders were redistributed proportionally across all diseases, excluding reproductive & maternal conditions and oral disorders.

### **Morbidity estimates**

#### **Disease list**

For the ABDS 2015, vision disorders included in the vision loss envelope were reported separately by the type of vision disorder. This is different from previous Australian burden of disease studies (including the ABDS 2011), where vision loss was reported as a single envelope estimate (excluding vision loss due to diabetic retinopathy and trachoma in the ABDS 2011).

Prevalence estimates for vision loss due to diabetic retinopathy and trachoma were calculated separately under endocrine disorders and infections (see methods for these respective disease groups). Vision loss due to injuries is included in other vision disorders.

#### **Sequelae and health states**

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

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

Disease	Sequela	ABDS 2011 health state identifier <sup>(a)</sup>
Hearing loss	Hearing loss	103, 104, 105, 106, 108, 109, 110, 111
Other hearing & vestibular disorders	Ear pain	15
	Vertiginous symptoms due to other hearing & vestibular disorders	207
Age-related macular degeneration	Vision loss	113, 114, 115, 116
Cataract & other lens disorders	Vision loss	113, 114, 115, 116
Glaucoma	Vision loss	113, 114, 115, 116
Refractive errors	Vision loss	113, 114, 115
Other vision disorders	Vision loss	114, 116, 117

(a) See Appendix Table C1.

## Prevalence estimation

### Hearing loss

In the ABDS 2015, 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, the ABDS 2015 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 in Australia, the overall national prevalence of hearing loss was estimated using 3 main data sources:

- For ages 0–24, prevalence was derived from the Australian Hearing 2015 demographics report summary tables of people aged 26 and under with a clinically diagnosed hearing impairment who were fitted with a hearing aid (Australian Hearing 2015).
- Prevalence for people aged 25–54 was derived from the NHS 2014–15 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) as in the ABDS 2011 as more recent data were unavailable at the time of the study.

#### *Prevalence estimation by age and sex*

Prevalence estimates in 10-year age groups by sex were derived from the Blue Mountains Hearing Study. To derive 5-year age groups, sex-specific proportions of total hearing loss in 5-year age groups from the NHS 2014–15 were applied to ages 55 and over.

#### *Prevalence by severity*

The same severity distribution as used in the ABDS 2011 (derived from the GBD 2010 for high-income regions) was used, as it was the most updated publicly available data at the time of the study.

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 & vestibular disorders**

Other hearing disorders were also calculated using self-reported data from the NHS 2014–15. It was assumed that conditions classified under Meniere disease would result in vertigo, and those classified as other ear diseases would result in ear pain.

Estimates of Meniere disease by sex were obtained from the NHS 2014–15 (age estimates were not available due to high RSEs). To obtain age estimates, the age distribution was obtained using hospitalisations of Meniere disease in 2015 by age and sex from the NHMD, and then applied to the total prevalence derived from the NHS 2014–15. As well, the NHS did not report on *Very remote* areas, so prevalence estimates were adjusted to account for *Very remote* areas.

To estimate burden from ear pain due to other hearing and vestibular disorders, estimates were obtained from the NHS 2014–15 by age and sex. Age groups that had high RSEs (0–14 and 75 and over) were estimated using population sex-specific proportions to obtain 5-year age groups.

### **Refractive error and cataract & other lens disorders**

For mild and moderate cases of uncorrected refractive error and for cataract & other lens disorders, prevalence rates by sex were obtained from the National Eye Health Survey 2016 (Foreman et al. 2016). For severe and blind cases, prevalence rates by 10-year age groups were obtained from the Melbourne Visual Impairment Project, as in the ABDS 2011, due to limited updated data being available. Estimates were modelled in 5-year age groups using age proportions from the ABDS 2003 (Begg et al. 2007). For cataract & other lens disorders only, prevalence was estimated from age 40 and over due to the nature of this condition.

Severity distributions for refractive error were obtained from the Melbourne Visual Impairment Project and modelled to account for inconsistencies. It was assumed that 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.

Severity distributions for vision impairment due to cataract were obtained from published Melbourne Visual Impairment Project 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.

### **Glaucoma**

Prevalence for glaucoma was estimated only 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, as in the ABDS 2011, due to limitations in reliable data. 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 was used across all age groups.

### **Age-related macular degeneration**

Prevalence of age-related macular degeneration was estimated only from age 50 and over, 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, as in the ABDS 2011, due to limitations in reliable data. Prevalence rates in younger age groups (that is, 50–64) were 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 that the ratio of clinical age-related macular degeneration-to-vision loss due to age-related macular degeneration was the same as the ratio of mild vision loss-to-blindness due to age-related macular degeneration. This also assumed the same progression rate through each severity.

### **Other vision disorders**

As in the ABDS 2011, 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 vision loss in Weih and others (2000) is the same as that for near-sighted-to-moderate vision loss for other vision disorders.

### **Sub-national estimates**

Sub-national estimates were apportioned from the national estimates based on age- and sex-specific ratios from the NHS 2014–15 data.

## 2011 and 2003 estimates

Due to limitations in reliable data, the same severity distribution and proportions of individuals with hearing loss and vision loss used in 2015 estimates were used for national 2003 and 2011 estimates.

# Infant and congenital conditions

## Mortality estimates

Deaths related to infant & congenital conditions were assigned from the NMD as defined by the disease list (Appendix Table A2). 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 all non-communicable diseases (that is, excluding infections, cancer and injuries) excluding reproductive & maternal conditions, oral disorders and hearing & vision disorders.

## Morbidity estimates

### Sequelae and health states

The sequelae and health states assigned to infant and congenital disorders are listed in Table 5.12. 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 5.12: Sequelae and health states for infant & congenital conditions**

Disease	Sequela	ABDS 2011 health state identifier <sup>(a)</sup>
Pre-term birth & low birthweight complications	Acute complications due to pre-term & low birthweight complications	54
	Neurodevelopment impairment due to pre-term & low birthweight complications <sup>(b)</sup>	213, 214, 215, 216, 217, 218
Birth trauma & asphyxia	Neurodevelopment impairment due to birth trauma & asphyxia <sup>(b)</sup>	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 <sup>(b)</sup>	218
Brain malformations	Neurodevelopment impairment due to brain malformations <sup>(b)</sup>	216, 217, 218
Cardiovascular defects	Congenital cardiovascular defects untreated	33
	Heart failure due to congenital cardiovascular defects <sup>(c)</sup>	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

(continued)

**Table 5.12 (continued): Sequelae and health states for infant & congenital conditions**

Disease	Sequela	ABDS 2011 health state identifier <sup>(a)</sup>
Down syndrome	Intellectual disability due to Down syndrome <sup>(b)</sup>	99, 100, 101, 102,243
Other chromosomal abnormalities	Intellectual disability due to chromosomal abnormalities <sup>(b)</sup>	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 & congenital conditions are listed in Table 5.13.

**Table 5.13: Key data sources for infant & 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 in 2015. This rate was then applied to the Australian live births in 2015 to derive national estimates.

## DISMOD II

Two groups of congenital abnormalities—neural tube defects and gastrointestinal malformations—used DISMOD II to obtain point prevalence for long-term sequelae. 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 2015**

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 FASD)
- 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 the 'Mental and substance use disorders' section in this chapter.

### **Pre-term birth & low birthweight complications**

Prevalence of neurodevelopmental impairment due to pre-term birth & 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 and others (2013).

Estimates for acute complications due to pre-term births & low birthweight were based on incidence of hospital separations in the 2015 calendar year. Any admissions to hospital that included the corresponding ICD-10-AM codes as diagnosis were counted.

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 2015, by gestational age (Chow et al 2017). The durations were:

- extremely pre-term: 108 days
- very pre-term: 54 days
- late pre-term: 20 days.

### **Birth trauma & asphyxia**

Prevalence of neurodevelopmental impairment due to birth trauma & asphyxia was derived from the intellectual disability envelope. The severity distribution for birth trauma and asphyxia was derived from the NHMD 2014–15 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 2016 (Cerebral Palsy Alliance 2016). Incidence and mortality from cerebral palsy 1913–2015 was used to derive prevalence. Incidence and mortality from cerebral palsy 1913–2015 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 2012).

An Australian-specific severity distribution derived from the Gross Motor Function Classification System was applied to the estimates (Appendix Table D5).



### *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 & congenital disease group (birth trauma & asphyxia and pre-term & low birthweight complications) was excluded after estimation of the YLD. Half (50%) of YLD for neonatally acquired cerebral palsy was distributed to birth trauma & asphyxia (10%) and pre-term & low birthweight complications (40%). The proportional split was determined from the studies by McIntyre and others (2013), Badawi and others (2005) and the NZBDS (NZMOH 2012). The remaining 50% of YLD was assigned to cerebral palsy.

### **Neonatal infections & other disorders of infancy**

Health loss from neonatal infections & other disorders of infancy is short term. Prevalence estimates for neonatal infections & other disorders of infancy were based on hospital separations from the NHMD 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 D6). The life expectancy for people with moderate or severe neural tube defects was assumed to be about 46 years (Oakeshott et al. 2015).

### **Brain malformations**

Prevalence of neurodevelopmental impairment due to brain malformations was derived from the intellectual disability envelope (see 'Mental and substance use disorders' in this chapter). 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% of cases have no residual disability (GBD 2013 Collaborators 2015).

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. 2001).

Live birth prevalence rates of cleft lip and/or palate were derived from published WARDA data for 1980–2015. 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 2015 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 44.6% of people with anorectal malformations experience faecal incontinence (Stenström et al. 2014). 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 2014 (ICBDSR 2014). 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 testis was derived from the NHMD 2015, 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 YLD:YLL ratio was derived using the combined YLD and YLL 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.

## **Sub-national 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 2015 data.

## **2011 and 2003 estimates**

Estimates for infant & congenital conditions used a similar method, with data sourced for 2011 and 2003.

# **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 as follows: some infections of the skin and subcutaneous tissue were allocated to skin conditions, infections of the amniotic sac and membranes were allocated to reproductive & maternal conditions, and some neonatal infections were allocated to infant & congenital conditions (see Appendix Table B1).

Septicaemia (A40, excluding A40.3, and A41) was the largest cause of death requiring redistribution within the infections group. 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 MCODE method.

## **Morbidity estimates**

### **Sequelae and health states**

A list of sequelae and health states assigned to each infectious disease is included in Appendix table D7. As infectious disease data are generally measured in terms of incident cases, prevalence estimates were produced by applying a duration of health loss. These durations were sourced from previous Australian or GBD studies.

### **Prevalence estimation**

The primary data sources used for infectious diseases are listed in Table 5.14. 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 for each disease.

**Table 5.14: 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, mumps, rubella, <i>Haemophilus influenzae</i> type-B (Hib), pneumococcal disease, meningococcal disease, dengue, Ross River virus, Barmah Forest virus, malaria
Bettering the Evaluation and Care of Health survey (BEACH)	Upper respiratory infections, otitis media (acute), varicella, herpes zoster, lower respiratory infections, influenza, other sexually transmitted infections, urinary tract 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 to be an accurate estimate of the incidence of tuberculosis, diphtheria, tetanus, measles, mumps, 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 mean notifications might result in an overestimate in burden in some years (Cashman et al. 2008; Liu et al. 2007; Selvey et al. 2014). 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: 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 programs, outbreak investigation and expert advice (de Greeff et al. 2009; Kirk et al. 2014).

Enhanced disease surveillance and screening programs 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) as used in the ABDS 2011 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 and to distribute national syphilis notifications, by stage of disease.

## **Bettering the Evaluation and Care of Health**

Data from the BEACH survey were used for infectious diseases where no other representative data source was available (including acute otitis media, herpes zoster, influenza, lower respiratory infections, upper respiratory infections, urinary tract infections, varicella and other sexually transmitted infections).

For the year 2011, the number of BEACH 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 extrapolated number of national consultations was used to estimate disease incidence, based on the assumption that 1 GP episode represents 1 incident case.

Where disease prevalence rates were assumed to remain constant in recent years or no other data sources could be identified to inform prevalence, the disease prevalence rates calculated for 2011 were applied to the 2015 population to attain estimates for the year 2015. This was done for otitis media and upper respiratory infections.

Where alternative data sources were available (such as notifications or hospital separations), age–sex or sex-specific ratios were calculated between notifications or hospitalisations and BEACH data in previous years. Estimated ratios were applied to notifications or separations data in the year 2015 to calculate expected disease incidence in the community in 2015. This assumes that the proportion of cases identified via notifications or separations data is consistent with those identified in general practices from 2010–2012. This method was used to estimate varicella, herpes zoster, lower respiratory infections, influenza and urinary tract infections.

## **National Hospital Morbidity Database**

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; these ratios were calculated using data from linked hospitals and NDI components for the NDLDLP database to correct for multiple hospital separations for a single person.

## **Other published data sources**

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

- the incidence of gastrointestinal infectious diseases in 2010 (Kirk et al. 2014)
- the number of individuals living with human immunodeficiency virus/acquired immune deficiency syndrome (HIV/AIDS) by age and sex (Jansson & Wilson 2012; The Kirby Institute 2012)
- the annual incidence of hepatitis B and C infections (The Kirby Institute 2016)
- inflation factors for emergency department presentations for pelvic inflammatory disease that do not get admitted to hospital (Goller et al. 2018)
- estimated proportions of pelvic inflammatory disease due to chlamydia, gonorrhoea and other sexually transmitted infections (Reekie et al. 2014)

- age-specific proportion of newly acquired Hepatitis B infection that are symptomatic (Shepard et al. 2006).

As well, prevalence estimates for infertility were derived as part of the reproductive & maternal conditions disease group. Prevalence estimates for vision loss due to trachoma were estimated as part of the hearing & vision loss disease group.

### Sub-national 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.

### 2011 and 2003 estimates

Prevalence estimates for 2003 and 2011 were calculated from the data sources and method as described for 2015; however, general practice encounters data were used directly for the years 2011 and 2003.

## Injuries

### Injury perspectives for burden of disease analysis

Burden of disease studies traditionally report injury burden by external causes. The functional limitations (health states) caused by injury are described by the nature of the injury or the body part injured.

In the ABDS 2015, injury burden was reported using two perspectives—the **external cause** that led to the injury (for example, a road traffic accident, a fall or an accidental poisoning) and the **nature of the injury** (for example, a hip fracture, a 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 facilitate reporting by both perspectives, the fatal injury burden by external cause was mapped to the nature of injury causes, and the non-fatal burden by nature of injury was mapped to external causes (as described further in the following section).

Injury categories for each perspective are shown in Table 5.15. The ICD-10 codes used to identify injury causes are shown in Appendix Table A2.

**Table 5.15: List of injury categories used in the ABDS 2015 for nature of injury and external cause of injury**

Injury by nature	Injury by external cause
Traumatic brain injury	Road traffic injuries—motorcyclists
Spinal cord injury	Road traffic injuries—motor vehicle occupants
Internal & crush injury	Road traffic injuries—pedal cyclists
Poisoning	Road traffic injuries—pedestrians
Drowning & submersion injuries	Other land transport injuries
Hip fracture	Poisoning
Tibia & ankle fracture	Falls
Humerus fracture	Fire, burns & scalds

(continued)

**Table 5.15 (continued): List of injury categories used in the ABDS 2015 for nature of injury and external cause of injury**

Injury by nature	Injury by external cause
Other fractures	Drowning
Dislocations	Other unintentional injuries
Soft tissue injuries	Suicide & self-inflicted injuries
Burn injuries	Homicide & violence
Other injuries	All other external causes of injury

## Mortality estimates

Injury deaths were identified from the NMD as deaths with an underlying cause coded to an external cause of injury from ICD-10 'Chapter XX: External causes of morbidity and mortality' in the range V01–Y98.

## Redistribution

Some external causes of injury were identified for redistribution, specifically:

- Y10–Y34 (event of undetermined intent)—redistributed across injury causes based on direct evidence informed by the ABS revisions process
- X59 (exposure to unspecified factor)—redistributed across injury causes using proportional allocation
- Y87.2 (non-specific injury deaths)—(sequelae of events of undetermined intent), Y89.9 (sequelae of unspecified external cause), Y90–Y98 (supplementary factors related to causes of morbidity and mortality classified elsewhere)—redistributed across all causes using proportional allocation.

Injury deaths may also arise from other redistribution causes having injuries as the target cause for redistribution. Some examples are septicaemia, pneumonitis, unknown causes and all other non-specific, intermediate and immediate causes. The redistribution groups, methods and target causes are described in Appendix Table B1.

## Conversion to nature of injury

YLL were also estimated for the other injury perspective—nature of injury using codes from ICD-10 'Chapter XIX: Injury poisoning and certain other consequences of external causes' in the range S00–T75, T79–T81 and T88. The external cause of injury was mapped to the nature of injury using information reported in the associated causes of death.

Each death can have more than one associated cause of death (which are not reported in order of severity). Hence, the single most relevant associated cause of death must be identified. We used a hierarchical approach to identify, from each death, the injury most likely to have caused the death (Table 5.16). The hierarchy used in the ABDS is a modified version of that used in the NZBDS (NZMOH, unpublished). In the NZBDS, the likelihood that the injury caused death was based on the nature of the injury, prognosis and clinical knowledge of injury conditions.

For example, if an injury death reports traumatic brain injury (TBI) as an associated cause of death, this will be selected as the injury most likely to have caused the death and is thus ascribed as the nature of injury. Where TBI is not reported as an associated cause, the next injury most likely to have resulted in death is selected as the nature of injury.

**Table 5.16: 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 & 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.

The relationship between external cause and nature of injury was used to develop age- and sex-specific matrices (cross-tabulations) for mapping YLL by external cause to YLL by nature of injury, maintaining internal consistency for YLL.

Nature of injury category was found for more than 96% of injury death records. Only records with an external cause of death code and a nature of injury code were used to develop the mapping algorithm.

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

## Morbidity estimates

The overarching methods for estimating non-fatal burden in the ABDS 2015 are the same as for the ABDS 2011 (AIHW 2016a); however, the epidemiological inputs used to estimate injury prevalence have been updated since the ABDS 2011. The overall differences in the inputs and the impact of these on the results are described further below in Box 5.1.

In the ABDS 2015 it is assumed that all injuries in Australia are treated. Therefore, the GBD disability weights that relate to untreated injuries were not used for estimating non-fatal burden.

## Overview of method for estimating non-fatal injury burden

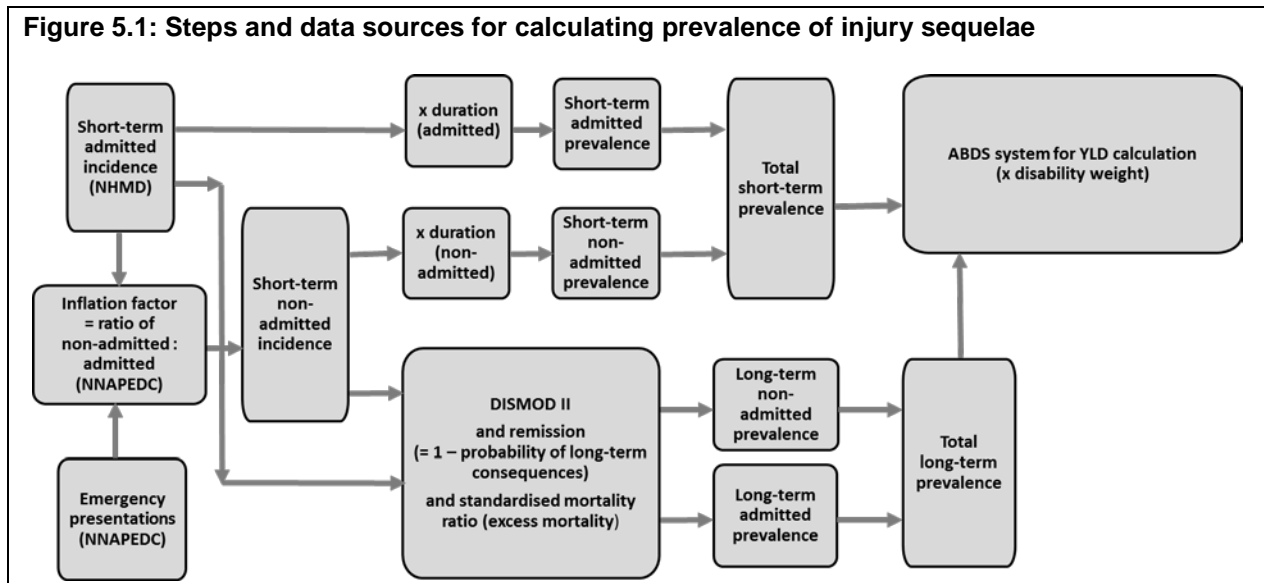
The calculation of non-fatal injury burden requires the estimation of 4 prevalence components: the prevalence of short-term admitted and non-admitted injuries, and of long-term admitted and non-admitted injuries.

Short-term injury burden is directly associated with the incidence of injury and duration of the health consequences, while long-term burden is directly related to the incidence of injury, the remission of the health consequences and the associated excess mortality.

Figure 5.1 describes the calculation of each component for estimating non-fatal injury burden.



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



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

Following on from this example, each injury sustained will have some duration of short-term health loss—based on the duration inputs—followed by long-term health loss—based on remission (or percentage likelihood of sustaining long-term health loss) and the excess mortality associated with the injury.

The starting point for calculating non-fatal injury burden is the incidence of short-term admitted injury. The incidence of short-term injury is then used to derive:

- **short-term admitted prevalence** is the product of the short-term admitted incidence and the duration of each injury sequela
- **short-term non-admitted prevalence** is obtained by inflating the short-term admitted incidence, using an inflation ratio (to account for cases presenting to the emergency department but which were not admitted) and the duration of short-term non-admitted injury sequela
- **long-term admitted prevalence** is derived using DISMOD II and is modelled on the short-term admitted incidence, the remission and excess mortality of each sequela
- **long-term non-admitted prevalence** is derived similarly using incidence, remission and excess mortality for each injury sequela for non-admitted injuries.

The YLD is the prevalence weighted by severity (that is, prevalence multiplied by the disability weight) associated with the short- and long-term health states for each injury sequela. The total YLD for any injury is the sum of the YLD for each of the 4 weighted prevalence components.

The inputs for calculating the prevalence components were updated in the ABDS 2015. The main changes and their overall impact are described in Box 5.1.

### **Box 5.1: Impact of Global Burden of Disease Study 2013 epidemiological inputs and other methodological changes on non-fatal injury burden in the ABDS 2015**

Injury YLD estimates rely on data inputs describing the duration, remission and excess mortality for each cause of injury. In the ABDS 2011, a single comprehensive source of the required epidemiological inputs was not available. As a result, these inputs were sourced from multiple studies—namely, the original ABDS 2003 (Begg et al. 2007), the NZBDS (NZMOH 2013) and information that was available from the GBD study at the time. Since then, details of the GBD 2013 methods for estimating non-fatal injury burden have become available. In the ABDS 2015, we incorporated the majority of the GBD 2013 methods for estimating non-fatal injury burden as described by Haagsma and others (2016).

The overarching approach in the ABDS 2015 to calculating non-fatal injury burden was the same as used for the ABDS 2011, but the epidemiological inputs (durations, remission and excess mortality from injuries) from GBD 2013 are substantially different to those used in ABDS 2011 (AIHW 2016a). Hence, the results for non-fatal injury burden (YLD) in the ABDS 2015 are quite different from those reported in the ABDS 2011.

As described in Chapter 4, YLD for each disease is calculated by summing the severity-adjusted prevalence of the health sequelae arising from the disease or injury.

For short-term consequences of injury, the prevalence is the product of the incidence and duration; for long-term consequences, it is based on the incidence, remission from the health consequences and excess mortality associated with the injury.

The main differences in the epidemiological inputs for calculating prevalence between the ABDS 2015 (based on the GBD 2013) and the ABDS 2011, and their overall impact on the results, are as follows:

- the short-term durations of the health consequences of injury are longer in the ABDS 2015 than those used in the ABDS 2011. **Longer durations** result in higher prevalence as people suffer the consequences for longer
- remission rates in the ABDS 2015 were overall higher than those used in the ABDS 2011. **Higher remission** results in fewer prevalent cases as more people get better
- the excess mortality from injury was overall slightly higher in the ABDS 2015 than in the ABDS 2011. **Higher excess mortality** has the impact of decreasing prevalence as the likelihood of dying is greater for those suffering the consequences of injury compared with the general population.

The severity adjustment (the disability weights) were the same as for the ABDS 2011.

As well, in the ABDS 2015, inflation ratios to calculate the incidence of non-admitted injuries were derived using 3 years of emergency department data (compared with 1 year in the ABDS 2011) and were limited to emergency department presentations only. Overall, the inflation ratios were lower in the ABDS 2015 than in the ABDS 2011 and, as a result, reduced the incidence of short-term non-admitted injuries.

In alignment with GBD 2013 methods, non-admitted injuries were considered to have long-term consequences in the ABDS 2015; these were excluded from the calculation of long-term consequences in the ABDS 2011.

### **Scope of non-fatal injuries**

The scope of injuries is 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 associated with the underlying reason for the specific intervention.

However, while the burden associated with fractures and dislocations is reported in the injury disease group, there are some known associations between physical trauma and the later development of osteoarthritis and other musculoskeletal conditions. As a result, it is likely that some portion of the post-traumatic burden of injuries is double-counted in the injuries disease group and in the musculoskeletal diseases group. This limitation and ways in which it might be dealt with in future iterations of the ABDS are discussed in Chapter 10.

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, such as those presenting only to a GP and those for which no medical care is sought, are not captured. This approach is similar to that used for previous Australian studies, where injuries treated outside the hospital system were assumed to result in insignificant disability to warrant inclusion (Begg et al. 2007). This, however, imposes a limitation on the estimates and may 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 and health states**

All injuries (admitted and non-admitted) were assumed to have short-term consequences. Long-term consequences were included according to the GBD 2013 methods (Haagsma et al. 2016).

The model inputs for durations, remission, and excess mortality by admission status (admitted and non-admitted) for each injury sequela were based on the GBD 2013 methods. These inputs are shown in Appendix Table D8.

Some exceptions were that we used Australian-specific direct evidence to calculate YLD for two injuries:

- spinal cord injury—a severity distribution based on (unpublished) Australian trauma care data
- burns—excess mortality from a study in Western Australia (Duke et al. 2015).

### **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 (NNAPEDC). The prevalence of long-term consequences was estimated using DISMOD II, based on incident cases derived from the NHMD and the NNAPEDC.

Injury cases were identified in the NHMD based on separations in the 2003, 2011 and 2015 calendar years. The NNAPEDC for 2013–14 to 2015–16 was used to estimate incidence of non-admitted cases using information about the diagnosis.

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

### **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 principle 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. For example, if a severe burn and a minor burn were reported in a single episode of care, only the severest injury is counted for estimating YLD.

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

Only separations for acute types of care were counted. This excludes injuries presenting to hospitals, for example, for rehabilitation. It is assumed that the burden associated with injuries requiring rehabilitation is sufficiently estimated using the methods described below 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 of short duration, while the fatal burden was captured in YLL. There was no adjustment for repeat admission for the same injury.

### *Estimating 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 NNAPEDC database for 2013–14 to 2015–16. This data set included a diagnosis variable.

As diagnosis data were provided in a number of classifications, only jurisdictions that had more than 95% of emergency department records coded to an ICD-10 AM classification were included in the analysis. Hence, all records from New South Wales and Western Australian hospitals were excluded. Further exclusions were made for records not coded to an ICD-10 AM classification. In total, around 48.4% of records were found to be useable for the purposes of the ABDS—that is, after excluding records for New South Wales and Western Australia (as stated earlier), other records not coded to a version of ICD-10 AM, non-emergency visits and records not identified as admitted or non-admitted. Of the useable records, 27.1% had a principal diagnosis of injury.

An inflation ratio was used to estimate the number of non-admitted injury cases. The ratio of non-admitted to admitted cases for each injury sequela (by age and sex) was calculated using the NNAPEDC. The ratio reflects the excess or absence of non-admitted cases compared with admitted cases. A ratio of less than 1 suggests that there were fewer non-admitted cases than admitted cases, and a ratio greater than 1 suggests that there were more non-admitted cases than admitted cases. For example, an inflation ratio of 1.2 suggests that for every 10 admitted cases there were 12 non-admitted cases, while a ratio of 0.2 suggests that for every 10 admitted cases there were 2 non-admitted cases. The ratio was applied to cases of admitted injuries (from the NHMD).

Diagnosis information is available in the NNAPEDC database starting from 2013–14. As a result, inflation ratios were calculated using the data having diagnosis information (2013–14 to 2015–16) and applied similarly to all data years in the study. A broad assumption in this method is that admission and non-admission rates over the period 2013–14 to 2015–16 were applicable to 2003, 2011 and 2015.

A limitation of this method is the reliability of the inflation ratios; that is, these data have not been rigorously assessed to understand how well the diagnosis predicts admission. The data were very broadly assessed 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. As well, it should be noted that NNAPEDC 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 2014–15, it was estimated that about 88% of emergency occasions were reported in the NNAPEDC (AIHW 2015a).

### **Long-term sequelae**

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

The values for these inputs for each long-term injury sequela were sourced from Haagsma and others (2015). The inputs estimated from this source are presented in Appendix table D8.

The GBD 2013 used a method to avoid double-counting of injury; for example, where post-trauma effects manifest as other musculoskeletal conditions. This method was not implemented in the ABDS 2015 (see Box 5.2 for more detail).

For each ABDS reference period (2003, 2011 and 2015), the respective national all-cause 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 the amount of extra modelling required in DISMOD II was minimal 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.

### **Box 5.2: ABDS diseases associated with previous injury**

In the GBD 2013, a method was implemented to avoid double-counting of the burden of diseases associated with previous injury, such as the long-term musculoskeletal conditions resulting from previous trauma. Specifically, the method involved deducting the long-term sequelae of fractures, dislocations, and contusions due to injuries from the disease 'other musculoskeletal conditions'.

There was insufficient detail in the GBD methods paper to implement a similar approach in the ABDS 2015. As a result, the ABDS 2015 estimates may include some double-counting of the musculoskeletal sequelae of injury; that is, the burden of the long-term effects of trauma from injury may be counted in injuries and in musculoskeletal conditions.

With limited available detail on the GBD methodology, the literature was explored to better understand the types and extent of injuries associated with musculoskeletal conditions to help inform a suitable process to avoid this double-counting. The literature review was brief but raised further questions around the relevance of deducting the injury sequelae specifically from other musculoskeletal conditions, as opposed to from specific musculoskeletal conditions.

Given the complexity of the relationships between these causes, further work is required to develop methods to suitably reduce potential double-counting of the burden associated with these causes. This was out of scope for this study.

### **Conversion to external cause**

Injury YLD were calculated according to the nature of the injury and 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, and provides a mapping of 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 NNAPEDC), it is assumed that the external cause of non-admitted injuries follows a similar pattern to that for admitted injuries. It is possible that the relationship between external cause and injury differs, depending on whether or not the injury resulted in admission. 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 patterns of external causes giving rise to particular injuries is the same nationally; that is, the matrices have not been calculated specifically for sub-national populations.

### **Sub-national estimates**

Sub-national estimates were largely derived directly using the same methods as those used for national estimates. This was helped by the availability of unit record data in the NHMD.

For injury cases obtained from the NHMD, sub-national estimates were derived by applying the 2011 ASGS remoteness areas and 2011 SEIFA population-based IRSD quintiles to the SA2 recorded in hospital separations data. The same inflation ratios were applied to sub-national data.

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

For all sub-national estimates, particularly for remoteness areas and socioeconomic groups, if there was insufficient information in the admitted injury records for ascribing a remoteness area or socioeconomic group, the record was excluded for generating models to distribute the remaining prevalence components (non-admitted short-term, and admitted and non-admitted long-term prevalence).

### **2011 and 2003 estimates**

The approach used to estimate 2011 and 2003 prevalence was the same as that used for 2015 estimates. The prevalence of short-term and long-term sequelae was calculated using the same methods.

## **Kidney and urinary conditions**

### **Mortality estimates**

Deaths related to kidney & urinary diseases were assigned from the NMD as defined by the disease list (Appendix Table A2). Please note that the ICD-10 codes used to align deaths to chronic kidney disease in the ABDS 2015 are different to the ICD-10 codes used for routine reporting of deaths due to chronic kidney disease in the AIHW. For more information on those ICD-10 codes, see Appendix B of *Indicators of socioeconomic inequalities in cardiovascular disease, diabetes and chronic kidney disease* (AIHW 2019c).

For the kidney & urinary disease group, the relevant ICD-10 codes that need to be redistributed are N17 (acute renal failure) and N19 (unspecified renal failure). Acute kidney failure (N17) was redistributed because it has multiple causes and is generally a consequence of many other diseases—for example, injury, infection, cancer and myocardial infarction. Unspecified renal failure (N19) was redistributed to chronic or acute renal failure.

These codes were redistributed using a 2-step approach. In the first step, deaths due to N19 (unspecified renal failure) were redistributed using direct evidence from information on hospitalisations prior to death in linked data from New South Wales and Western Australia (AIHW 2014c). N19 deaths were then be redistributed to N17 (acute renal failure), N18 (chronic renal failure) according to the proportions obtained from the linked data. This is described in more detail in the section that follows.

In the second step, N17 deaths (including those reassigned from N19) were then redistributed over all disease groups, using the indirect MCODE method.

### **Morbidity estimates**

#### **Sequelae and health states**

Sequelae and health states assigned to kidney and urinary conditions are shown in Table 5.17. 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 envelope**

Anaemia due to chronic kidney disease is part of the anaemia envelope. As anaemia 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 the GBD 2013 used those described in the study by Kassebaum and others (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 & metabolic disorders earlier in this chapter for more information on the methods used to estimate the anaemia envelope.

**Table 5.17: Sequelae, health states and duration for kidney & urinary conditions**

Disease	Sequela	ABDS 2011 health state identifier <sup>(a)</sup>	Duration
Chronic kidney disease	Asymptomatic chronic kidney disease	262	..
	Anaemia due to stage 3 chronic kidney disease <sup>(b)</sup>	195, 196, 197	12 months
	Stage 4 chronic kidney disease	41	12 months
	Anaemia due to stage 4 chronic kidney disease <sup>(b)</sup>	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
Interstitial nephritis	Interstitial nephritis	2, 3	7 days, 14 days
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 biomedical data available in 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 2015 (ANZDATA). Stages of chronic kidney disease in the AHS 2011–12 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 2014d).



### *Stages 1 and 2 chronic kidney disease*

The prevalence of stages 1 and 2 chronic kidney disease was estimated as described in *Cardiovascular disease, diabetes and chronic kidney disease, Australian facts: prevalence and incidence* (AIHW 2014d). The prevalence for these stages were given an asymptomatic health state with a disability weight of 0.

### *Stage 3 chronic kidney disease and anaemia due to stage 3 chronic kidney disease*

The prevalence of stage 3 chronic kidney disease was estimated from measured data from the AHS 2011–12. To estimate prevalence in the year 2015, the AIHW analysis of trends in stages 3–5 chronic kidney disease prevalence from the 1999–2000 AusDiab compared with the AHS 2011–12 in the broad age groups was used (AIHW 2018b). The age and sex distribution was further refined using the age and sex of people who were hospitalised for N18.3 in 2015.

The proportion of people with mild and moderate anaemia was derived from biomedical data available in the AHS 2011–12, with no updated biomedical data available in the NHS 2014–15. Since no severe anaemia due to stage 3 chronic kidney disease was reported in the AHS, the proportion of people with severe anaemia in stage 3 chronic kidney disease from the GBD 2013 (GBD 2013 Collaborators 2015) was used instead. It is important to note that the GBD proportions might not be reflective of 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 number of people with stages 4 and 5 chronic kidney disease. To estimate prevalence in 2015, similarly to stage 3, the estimate was based on trends for stages 3–5 chronic kidney disease from the 1999–2000 AusDiab compared with the AHS 2011–12. To estimate those with only stage 4 chronic kidney disease, the stages 4 and 5 estimate was used minus the number of people with end-stage kidney disease (stage 5 only) sourced from the ANZDATA.

It was 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 the GBD 2013 (GBD 2013 Collaborators 2015).

The age and sex distribution was based on 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 2015 was used to determine the prevalence of end-stage kidney disease treated by dialysis or transplant.

### *Untreated end-stage kidney disease*

People with untreated end-stage kidney disease were those not receiving kidney replacement therapy, although they might be receiving palliative treatments. The prevalence of people with untreated end-stage kidney disease was estimated by projecting incidence rates from 1997 to 2013 ANZDATA/NDI linked data to the reference year 2015. The by-5-year-age-group prevalence was estimated using the distribution in 2013 ANZDATA/NDI linked data.

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 2015 from the NHMD. This includes men admitted for surgery or for other reasons, which are both assumed to indicate substantial health loss, due to hospitalisation's 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 (obtained from the Western Australian Department of Health) were used to adjust national NHMD data for potential readmissions and hospital transfers, to obtain prevalence of the number of men with enlarged prostate in 2015. Health loss was assumed to apply 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 2015 from the NHMD. As this is an acute condition, each separation was assumed to be a case.

## **Interstitial nephritis**

Interstitial nephritis is a condition that can lead to a variety of non-specific systemic symptoms (including vomiting, fever, rashes and malaise) and can cause discomfort and difficulty with daily activities. Interstitial nephritis can be acute or chronic in nature, with untreated chronic conditions ultimately leading to end-stage kidney disease. For the ABDS 2015, burden due to interstitial nephritis was from acute cases only. Burden from chronic interstitial nephritis is captured under chronic kidney disease.

Burden due to nephritis was estimated using a combination of data from the NHMD and the GBD 2016. Point prevalence of severe cases was estimated by applying a duration of 2 weeks to hospitalised cases, with a principal diagnosis of interstitial nephritis (N10–N12) in 2015. Non-hospitalised cases was estimated using the ratio of severe:non-severe cases of interstitial nephritis from the GBD 2016. This indicated that nearly three-quarters of all interstitial nephritis cases are severe, which for the ABDS 2015 were assumed to represent hospitalised cases. Point prevalence for non-hospitalised cases was estimated by applying a 7-day duration of health loss to calculated estimates.

## **Other kidney and urinary diseases**

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

## **Sub-national estimates**

Prevalence estimates by state and territory, remoteness area and socioeconomic group were derived directly from the same data source as the national estimates with the exception of stage 3 chronic kidney disease (with and without anaemia), stage 4 chronic kidney disease with anaemia, and stage 4 chronic kidney disease. For these estimates, data on hospital separations ratios were used as a proxy in 2015.

## **2003 and 2011 estimates**

Estimates of end stage kidney disease, kidney stones, interstitial nephritis and enlarged prostate were taken directly from the same data source, using the same method to produce prevalence estimates for 2003 and 2011.

For stages 3 and 4 chronic kidney disease (without anaemia), prevalence estimates for 2011 were derived from the AHS 2011–12. For 2003, as for 2015, the estimate was based on trends for stages 3–5 chronic kidney disease from the 1999–2000 AusDiab compared with the AHS 2011–12. The same severity distributions used in 2015 for stages 3 and 4 chronic kidney disease with anaemia were also applied to the 2011 and 2003 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 distributions from hospitalisations were applied to the 2003 estimates.

## **Mental and substance use disorders**

### **Mortality estimates**

Deaths related to mental & substance use 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 all diseases (except reproductive & maternal conditions, oral disorders and hearing & vision disorders).

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 injury 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 to be 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**

The majority of morbidity estimates were based on methods used for the ABDS 2011, except for drug use disorders and autism spectrum disorders.

### **Sequelae and health states**

Sequelae and health states assigned to mental and substance use disorders are shown in Table 5.18. Durations (where relevant) and assumptions are outlined in relevant subsections.

**Table 5.18: Sequelae and health states for mental and substance use disorders**

<b>Disease</b>	<b>Sequela</b>	<b>ABDS 2015 health state identifier<sup>(a)</sup></b>
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
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
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
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	Childhood autism	98
Attention deficit hyperactivity disorder	Asymptomatic	262
	Attention deficit hyperactivity disorder	95
Conduct disorder	Asymptomatic	262
	Conduct disorder	96
Intellectual disability	Idiopathic & other intellectual disability <sup>(b)</sup>	100, 101, 102, 243, 99
Other mental & substance use disorders	Other mental & 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 prevalence are shown in Table 5.19.

**Table 5.19: 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)	Opioid use disorders and amphetamine use disorders
Alcohol and other drug treatment services national minimum data set (AODTS-NMDS) (supplemented with article by McKetin et al. 2017)	Amphetamine use disorders (for reference year 2015)
GBD 2010	Anorexia nervosa
2003–04 Te Rau Hinengaro: The New Zealand Mental Health Survey (Wells et al. 2006)	Bulimia nervosa

### **Box 5.3: Key method changes in the estimation of mental and substance use disorders for the ABDS 2015**

#### **Autism spectrum disorders**

The key data source used for the ABDS 2015 was the same as the one used for the ABDS 2011, i.e. the WA IDEA data. However, due to changes in DSM-V wherein Asperger's syndrome is no longer diagnosed separately, data provided from the WA IDEA no longer separates childhood autism and Asperger's syndrome. This change in DSM-V would have been applied in practice in WA since 2014, which affects data for 2015.

Experts advised that the first method (using the WA IDEA data in its entirety without separating childhood autism and Asperger/other autism from each other) provides more accurate results for Australia as it better reflects the increase in ASD prevalence between 2011 and 2015. As a result, prevalence estimates for 2011 and 2003 were revised to follow the method for 2015.

#### **Drug use disorders**

##### *Amphetamine use disorder*

In the ABDS 2011, amphetamine prevalence was based on the analyses by the National Drug and Alcohol Research Centre (Degenhardt et al. 2004). This article used indirect evidence, collected annually between 2002–03 and 2013–14. Multiplier methods were applied to treatment and hospital data for amphetamines to estimate the number of dependent methamphetamine users by year.

##### *Amphetamine use disorder*

For the ABDS 2015, updated multipliers from the study by McKetin and others (2017) were used on treatment services data from the Alcohol and other drug treatment services national minimum data set (AODTS-NMDS).

##### *Cannabis and cocaine use disorders*

Upon advice from the mental and substance use disorders expert panel, prevalence estimates for cannabis and cocaine use disorders were based on the prevalence rates from the GBD 2015 for these disorders for Australia.

## **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 the GBD 2013 distributions published by Burstein and others (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).

## **Intellectual disability**

As an envelope in the ABDS 2015, the overall prevalence of intellectual disability 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 of less than 70, and an indication of developmental delay before the age of 18. Mild, moderate, and severe intellectual disability are defined as an intelligent quotient of 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 & congenital conditions, with the remaining intellectual disability falling under idiopathic/other intellectual disability in the mental and substance use disorders disease group (Table 5.20). 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 5.20: Diseases within the intellectual disability envelope, and data source(s) for severity**

Disease	Source of severity distribution
Pre-term birth & low birthweight complications	Mild prevalence was based on the proportion reported in the WA IDEA database. The relationship between mild, moderate and severe was based on the perinatal data collection.
Birth trauma & asphyxia	Mild prevalence was based on the proportion reported in the WA 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	The severity distribution for birth trauma & asphyxia was used for brain malformations. This decision was informed by data from the WA IDEA database, which showed that the severity distribution for brain malformations and brain trauma & asphyxia were similar.
Down syndrome	All prevalence was based on the proportion reported in the WA IDEA database, adjusted for deaths.
Other chromosomal abnormalities	All prevalence was based on the proportion reported in the WA IDEA database.

The proportions of total intellectual disability that could be attributed to diseases specified in the ABDS 2015 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). For those conditions, 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 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, F38, 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, 2011 or 2015) and age group, but were not created separately for sub-national 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.

## Sub-national 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 rate ratio for Victoria was used for Tasmania, the rate ratio for New South Wales for the Australian Capital Territory, and the rate ratio for South Australia for the Northern Territory).

State and territory rate ratios for opioid use disorders were based on the analysis by Degenhardt and others (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.

## 2011 and 2003 estimates

With a few exceptions, all prevalence rates were considered stable between 2003, 2011 and 2015, 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 and others (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.

## 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 and health states

Sequelae and health states assigned to musculoskeletal conditions are shown in Table 5.21. Durations and assumptions are outlined in subsections for individual diseases.

**Table 5.21: Sequelae and health states for musculoskeletal conditions**

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>
Osteoarthritis	Osteoarthritis of the knee	262, 126, 127,
	Osteoarthritis of the hip	262, 126, 127,
Gout	Musculoskeletal problems caused by gout	132,
Rheumatoid arthritis	Musculoskeletal problems caused by rheumatoid arthritis	262, 130, 131,
Back pain & problems	Back pain & problems	262, 234, 254, 233, 255, 241, 242, 239,
Other musculoskeletal conditions <sup>(b)</sup>	Other musculoskeletal problems	262, 126, 127, 128, 130, 131,

(a) See Appendix Table C1.

(b) Other musculoskeletal conditions excludes symptoms and signs involving musculoskeletal conditions and osteoporosis.

### Prevalence estimation

Prevalence estimates for musculoskeletal conditions were derived from self-reported data in the NHS 2014–15, as it covered all the musculoskeletal conditions of interest.

Though self-reported data is generally not considered as good as clinical data, Peeters and others (2015) found that self-reported data is acceptable for osteoarthritis and rheumatoid arthritis. The NHS 2014–15 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 sub-national estimates, these 5-year age groups were combined to deal with sample size issues from the survey. Modelling was required to redistribute the data into 5-year age groups for analysis. As well, the NHS 2014–15 did not report on *Very remote* areas, so prevalence estimates were adjusted to account for *Very remote* areas.

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 NHS 2014–15. 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.

The GBD 2013 used a method to avoid double counting of disease associated with post-traumatic effects of injury that lead to long-term musculoskeletal conditions. This method was not implemented in the ABDS 2015, and as a result, there is potential overlap between other musculoskeletal conditions and osteoarthritis with selected injuries such as fractures and dislocations (see Box 5.2 under Injuries).

### Osteoarthritis

The NHS 2014–15 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 the GBD 2015.

Severity is based on the distribution of the pain experienced in the previous 4 weeks by people reporting arthritis only (Table 5.22). Health loss is assumed to last for the entire year.

**Table 5.22: ABDS severity distributions (%) for osteoarthritis**

Osteoarthritis	Asymptomatic	Mild	Moderate	Severe
ABDS 2011	14.5	46.9	28.0	10.6
ABDS 2015	10.8	42.7	32.7	13.8

### Gout

As a breakdown of chronic or acute gout was not available in the NHS 2014–15 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 NHS 2014–15 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 NHS 2014–15 (Table 5.23). Health loss is assumed to last for the entire year.

**Table 5.23: ABDS severity distributions (%) for rheumatoid arthritis**

Rheumatoid arthritis	Asymptomatic	Mild	Moderate	Severe
ABDS 2011	28.9	48.3	11.2	11.6
ABDS 2015	16.2	32.1	35.0	16.7

## Back pain and problems

The NHS 2014–15 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 & problems. The distribution of severity for back pain and problems is based on an associated pain data distribution (back pain & problems only) from the NHS 2014–15. 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 the GBD 2015. The resulting severity distribution is provided in Table 5.24.

**Table 5.24: ABDS severity distributions (%) for back pain and problems**

Back pain and problems	Asymptomatic	Mild	Moderate	Severe	Very severe
<b>ABDS 2011</b>					
Without leg pain (%)	15.1	41.9	19.1	5.4	0.9
With leg pain (%)	. .	10.9	5.0	1.4	0.2
<b>Total</b>	<b>15.1</b>	<b>52.8</b>	<b>24.1</b>	<b>6.8</b>	<b>1.1</b>
<b>ABDS 2015</b>					
Without leg pain (%)	14.1	43.2	19.6	6.2	1.4
With leg pain (%)	. .	7.6	5.2	2.1	0.5
<b>Total</b>	<b>14.1</b>	<b>50.8</b>	<b>24.8</b>	<b>8.3</b>	<b>1.9</b>

## Other musculoskeletal conditions

The prevalence of other musculoskeletal conditions was also derived from the NHS 2014–15. It was estimated by combining the prevalence of specific musculoskeletal conditions (excluding osteoarthritis, rheumatoid arthritis, gout, and back pain/problems) including the following: other arthropathies, other soft tissue disorders, other diseases of the musculoskeletal system and connective tissue, rheumatism and arthritis—other and type unknown.

The distribution of severity for other musculoskeletal conditions is based on associated pain data distribution (other musculoskeletal conditions only) from the NHS 2014–15 (Table 5.25).

**Table 5.25: ABDS severity distributions (%) for other musculoskeletal conditions**

	Asymptomatic	Level 1	Level 2	Level 3	Level 4	Level 5	Level 6
ABDS 2011	12.7	30.4	15.9	16.9	16.9	6.2	1.0
ABDS 2015	17.8	23.3	21.8	13.1	13.1	9.0	2.0

It is important to note that the NHS 2014–15 does not distinguish cases of other musculoskeletal conditions or osteoarthritis that were due to injuries; therefore, there may be double counting of prevalence in the musculoskeletal and injuries disease groups (see Box 5.2 under Injuries).

## Sub-national estimates

National prevalence estimates were apportioned based on sex and combined age-specific estimates from the NHS 2014–15 to derive sub-national 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 RSEs of more than 50% for these estimates.

## 2011 and 2003 estimates

The same methods used for the 2015 estimates were used for 2011 non-fatal burden musculoskeletal conditions estimates. The primary data source was the NHS 2011–12. The severity distributions used for 2011 estimates are included in the tables with the distributions used for 2015 estimates so they can be compared. It is important to note that some of the differences between 2015 and 2011 will be due to differences in the severity distributions.

The same methods used for the 2015 and 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.

# 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 and health states

Sequelae and health states assigned to the neurological conditions were shown in Table 5.26. Durations and assumptions were outlined in subsections for individual diseases.

**Table 5.26: Sequelae and health states for neurological conditions**

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>
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 2015, epilepsy is defined as a chronic disorder of the brain characterised by recurrent seizures, as consistent with the GBD 2013. The national sex-specific prevalence estimates of self-reported epilepsy were based on the NHS 2014–15. Age–sex specific distributions couldn't be obtained from the NHS 2014–15 as the RSEs for the epilepsy counts in many age groups were too high. Age and sex specific epilepsy prevalence was calculated from the NHMD by applying the person-to-separation ratios derived from Western Australian linked hospitalisations and deaths data (obtained from the Western Australian Department of Health) to the number of epilepsy separations in 2015. The NHMD 5-year age group proportions of prevalence for each sex was then applied to the NHS 2014–15 national estimates to obtain the national estimates by age and sex. As well, the NHS 2014–15 did not report on *Very remote* areas, so prevalence estimates were adjusted to account for *Very remote* areas.

There was no direct Australian data source to estimate the severity of epilepsy as defined in the ABDS 2015. The severity distribution of epilepsy was based on the European study by Forsgren and others (2005).

### Dementia

Dementia includes Alzheimer's disease (the most common form), vascular dementia, dementia with Lewy bodies and frontotemporal dementia (F00–F03, G30–G31). Prevalence estimates for dementia were calculated by applying the prevalence rates published in the *Dementia in Australia* report (AIHW 2012c) to the Australian estimated resident population. 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), for those aged under 80 and aged over 80 separately.

### Parkinson disease

There was a lack of recently published high quality and population-based Australian studies on Parkinson disease at the time of analysis. Thus, prevalence was estimated using findings from 2 international studies (de Rijk et al. 2000; Willis et al. 2013). Prevalence rates from the de Rijk study was applied to the Australian estimated resident population for people aged 55 and over. Prevalence rates of Parkinson disease in Australians aged 30–55 were modelled based on findings from the Willis study, assuming there is a linear trend of increase in the distribution of Parkinson disease in these age groups. For Australians aged under 30, the prevalence of Parkinson disease was assumed to be zero.

The severity distribution was derived from unpublished data from the Queensland Parkinson Project.

### Multiple sclerosis

Prevalence of multiple sclerosis was estimated based on prevalence rates from an Australian report prepared by the Menzies Health Economic Research Group, Associate Professor Ingrid van der Mei and Professor Bruce Taylor (Menzies Health Research Group, van der Mei & Taylor 2018). Age and sex distributions of multiple sclerosis were modelled based on the 2015 Survey of Disability, Ageing and Carers (SDAC). The 2015 SDAC is a national survey that collects information on people with disabilities, people aged 65 and over and carers of people with disability, long-term health conditions or older people. When finer age distributions were required, 2015 NHMD separations were used.

The severity distribution was obtained from the joint report by Covance Pty Ltd and Professor Andrew 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 neurones. Prevalence was estimated using person-to-separations ratios derived from the linked hospitals and NDI components for the NDLD database, which were applied to the count of motor neurone disease separations from the NHMD.

Since the 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, migraine is an episodic condition characterized by quiescent and relapse phases, known as headaches that typically last 4–72 hours. Period prevalence estimates for migraine (by age and sex) in a 6-month period were based from the NHS 2014–15 self-reported data. Point prevalence was estimated by applying a duration of 9 days of health loss in 6 months to the period prevalence (number of people with migraine). This duration is based on the assumption that a person with self-reported migraine on average, has 12 episodes (about once a month) per year with a duration of 1.5 days for each 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 person-to-separation ratio derived from the linked hospitals and NDI components for the NDLD database, which was applied to the count of separations from the NHMD. This minimized double counting of the number of events of Guillain-Barré syndrome in a reference year, as each event has a duration of 6.7 months based on the GBD 2013 (GBD 2013 Collaborators 2015). This duration was then applied to the number of people with Guillain-Barré syndrome derived from the NHMD 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:YLL ratio for Parkinson disease, multiple sclerosis and motor neurone disease combined to the YLL for other neurological conditions.

### **Sub-national estimates**

For migraine and epilepsy, counts of the number of people with these conditions were provided at the sub-national levels (state, remoteness and socioeconomic group) using self-reported data from the NHS 2014–15. Proportions of the counts for each sub-national group were applied to the national prevalence estimates to obtain the sub-national estimates.

For motor neurone disease and Guillain-Barré syndrome, the number of separations at sub-national levels were derived from the NHMD directly and point prevalence was estimated by applying the person to separation ratios to the count of separations as consistent with the national estimates.

For multiple sclerosis, prevalence rates by state and territory and proportions by remoteness area were available from the 2018 Menzies Health Economic Research Group report.

Prevalence estimates by socioeconomic group were calculated by applying proportions of multiple sclerosis deaths derived from the NMD to the national estimates.

For dementia and Parkinson disease, breakdowns by state/territory, remoteness area and socioeconomic group were derived by applying proportions of deaths from the NMD to the national estimates.

## 2011 and 2003 estimates

The overall methods used to estimate the prevalence of neurological conditions in 2015 was the based on the methods used to produce the 2003 and 2011 estimates.

Where available, the data source used for the 2003 and 2011 estimates was the same as for the 2015 estimates. For motor neurone disease and Guillain-Barré syndrome, 2003 and 2011 separations were derived from the NHMD. However, a different set of people-to-separation ratios was applied to the separations in 2003 and 2011 for Guillain-Barré syndrome than for 2015. For epilepsy and migraine, earlier estimates were based on data from the NHS 2004–05 and the AHS 2011–12 for the 2003 and 2011 reference years.

For multiple sclerosis, the 2003 and 2011 estimates were based on the prevalence rates from an earlier study by Andrew Palmer (Palmer et al. 2013) and age distributions were modelled based on 2010 NHMD separations. Estimates for dementia and Parkinson disease used prevalence rates from the same studies for 2003, 2011 and 2015.

## Oral disorders

### Mortality estimates

Oral disorder deaths were assigned from the NMD as defined by the disease list (Appendix table A2). No deaths due to oral disorders were redistributed. Oral disorders were also not a target cause for redistribution.

### Morbidity estimates

#### Sequelae and health states

Sequelae and health states assigned to mental and substance use disorders are shown in Table 5.27. Durations and assumptions are outlined in subsections for individual diseases.

**Table 5.27: Sequelae and health states for mental and substance use disorders**

Disease	Sequela	ABDS 2011 health state identifier <sup>(a)</sup>
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 under 15 as it is relatively uncommon. Estimates of dental caries in children were modelled with inputs from the National Child Oral Health Study 2012–14, and also past trends from the Child Dental Health Survey 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 under 15, as chronic periodontal disease in children aged under 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 under 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 people 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 2015 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.

### **Sub-national estimates**

Prevalence estimates by state/territory, remoteness and socioeconomic group were calculated from results of the National Survey of Adult Oral Health. For dental caries in children under 15, sub-national results from the Child Dental Health Survey 2009 were used. The National Child Oral Health Study 2012–14 reported caries in deciduous and adult teeth separately and the results were compared with the Child Dental Health Survey 2009, but it did not report by remoteness and socioeconomic group.

Proportions 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 sub-national estimates used the same approach as for national but disaggregated directly according to remoteness area, socioeconomic group and state/territory.

### 2011 and 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 2011 and 2003 population structures to calculate prevalence of dental caries, periodontal disease and severe tooth loss in 2011 and 2003, respectively.

Prevalence of dental caries in children from the Child Dental Health Survey 2009 were incorporated into the 2011 estimates. Differences in the prevalence of dental caries in children between the 2003–04 and 2009 Child Dental Health Surveys were incorporated into the 2003 estimates.

The prevalence of other oral disorders for 2011 and 2003 used the same approach as 2015, but drawn from data in the 2011 and 2003 calendar years, respectively.

## Reproductive and maternal conditions

### Mortality estimates

Deaths related to reproductive & 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 all disease excluding reproductive & maternal conditions, oral disorders and hearing & vision disorders.

Reproductive & maternal conditions were not a target cause for redistribution.

### Morbidity estimates

#### Sequelae and health states

Sequelae, health states and durations for sequelae assigned to reproductive and maternal conditions are shown in Table 5.28.

**Table 5.28: Sequelae, health states and durations for reproductive & maternal conditions**

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>	Duration for acute sequelae
<b>Maternal conditions</b>			
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

*(continued)*

**Table 5.28 (continued): Sequelae, health states and durations for reproductive and maternal conditions**

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>	Duration for acute sequelae
<b>Maternal conditions (continued)</b>			
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
<b>Reproductive conditions</b>			
Endometriosis	Endometriosis	193, 194	3 days per month
Endometriosis	Infertility due to endometriosis <sup>(b)</sup>	50, 51	..
Uterine fibroids	Anaemia due to uterine fibroids <sup>(c)</sup>	195, 196	6 months
	Infertility due to uterine fibroids <sup>(b)</sup>	50, 51	..
	Symptomatic uterine fibroids	192	2–6 weeks
Genital prolapse	Faecal incontinence	48	..
	Genital prolapse	192	..
	Stress incontinence	260	..
Polycystic ovarian syndrome	Infertility due to polycystic ovarian syndrome <sup>(b)</sup>	50, 51	..
	Polycystic ovarian syndrome	207	..
Infertility	Infertility <sup>(b)</sup>	50, 51	..
Other reproductive conditions	Anaemia due to other reproductive conditions <sup>(c)</sup>	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 estimated for men and women aged 20–49 seeking to have a child. As infertility is a sequela of multiple conditions across the 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 human immunodeficiency virus, or 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 cycles in 2015 was derived from the Australian and New Zealand Assisted Reproductive Database (Fitzgerald et al. 2017). Estimates were inflated to account for varying types of assisted reproductive technology.

The number of men and women seeking assistance for infertility in 2015 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 an estimated 19.6% of people who experience difficulty becoming pregnant seek assisted reproductive technology (Marino et al. 2011), the prevalence from the Australian and New Zealand Assisted Reproductive Database was inflated to estimate the overall prevalence of infertility in 2015.

Age by sex distributions were the same as used in the ABDS 2011, originally derived from GP encounters for infertility between April 2000 and March 2011 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 5.29).

**Table 5.29: 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

Population-based data of women who gave birth in 2015 and whether they had previously given birth (at least 20 weeks gestation or 400 grams birthweight) was applied (Table 5.30). 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 5.30: Women who gave birth in 2015, by maternal age and parity (%)**

Maternal age	Primipara (no previous births)	Multipara (one or more previous births)
20–24 years	55.5	44.5
25–29 years	49.8	50.2
30–34 years	39.3	60.6
35–39 years	28.1	71.8
40–44 years	27.1	72.8
45 years and over	40.7	59.1

Source: AIHW analysis of National Perinatal Data Collection.

### 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 90% of tubal factor infertility is caused by sexually transmitted infections. Current literature reports 7.0%–9.8% of female infertility 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%) based on GBD estimates.

## Prevalence estimation

### Maternal conditions

Incidence of maternal conditions in 2015 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 medically or surgically induced) before a gestational age of 20 weeks. Medical abortions performed via use of pharmaceuticals were included for 2015 using PBS data, but not for 2003 or 2011, due to the introduction of 'MS-2 step' pharmaceuticals in 2013.

As maternal conditions are generally measured in terms of incident cases, prevalence estimates were produced by applying a duration of health loss (Table 5.28). Durations to derive prevalence from incidence data were the same as those used in the ABDS 2011, 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. As new evidence suggests 23.3% of ectopic pregnancies are treated in emergency departments and do not go on to be admitted to hospital (Goller et al. 2018), estimates derived from admitted patient data was inflated to account for those that experienced ectopic pregnancy but were not admitted.

Cases of surgically induced early pregnancy loss were derived from public patient hospital admissions for medical abortions, as well as Medicare claims data, where relevant (AIHW National Perinatal Statistics Unit 2005). Queensland provided estimates derived from linked data.

Adjustments for unclaimed procedures in New South Wales, Victoria, Tasmania and the Australian Capital Territory were applied to Medicare Benefits Schedule data (AIHW NPSU 2005). Non-hospital claims for these jurisdictions were inflated by 7.5% to account for unclaimed procedures (Shankar et al. 2017). Public patient admissions were added to adjusted Medicare data and PBS data to derive incidence of abortion in 2015.

Medically induced abortions were included for 2015 only using PBS data for MS-2 Step pharmaceuticals.

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 sequela.

#### *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 hospitals data and obtained from the Western Australian Department of Health 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, GP 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 34–39 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 were born between 1973 and 1978. Age distributions, derived from GP visits were applied to these estimates, to derive prevalence by age.

Endometriosis severity was based on surgical intervention. Hospitalised cases of endometriosis in 2015 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 2015 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. More recent studies suggest the impact of fibroids on fertility is unknown (for example, Purohit & Vigneswaran 2016) and do not report a proportion. However, it was decided to use the 1981 estimate.

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*

**Symptomatic genital prolapse:** The prevalence of genital prolapse in Australia was based on prevalence rates obtained from the NZBDS (NZMOH 2012) applied to the 2015 Australian Estimated Residential Population. Due to limited data, male estimates were calculated using the male-to-female genital prolapse hospitalisations ratio in the year 2015, with procedure codes related to genital prolapse.

**Stress incontinence due to genital prolapse:** Stress incontinence in males was not included as this was assumed to be prostate related. The age-specific proportion of females with genital prolapse who experience stress incontinence was obtained from Lawrence and others (2008) and applied directly to females symptomatic prolapse estimates.

**Faecal incontinence due to genital prolapse:** Estimates of faecal incontinence from Harvie and others (2018) were applied to total female and male symptomatic prolapse estimates. The age-distribution was obtained from Lawrence and others (2008) and applied to the total proportion with faecal incontinence due to genital prolapse.

### *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. Conditions identified as resulting in pain, anaemia or both were included in estimations.

The prevalence rate of 'other reproductive conditions' by age, sex and sequela as estimated in the ABDS 2011, was applied to the 2015 Australian Estimated Residential Population to derive estimates in year 2015. The original estimates from ABDS 2011 were derived from the BEACH survey using the proportion of general practice visits for these conditions between March 2000 and April 2011. Estimates for people aged under 15 were based on population distributions, and estimates for people aged 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 & metabolic disease group).

## **Sub-national estimates**

Sub-national estimates for most reproductive and maternal conditions were derived directly from the NHMD in 2015, 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.

## **2011 and 2003 estimates**

Estimates using hospital separations data used the same method as for 2015, but with 2011 and 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 2015, applied to the 2011 or 2003 population. This is because using earlier Australian Longitudinal Study on Women's Health surveys and BEACH data gave implausible estimates.

# 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 (J96) were redistributed across all diseases (excluding reproductive and maternal conditions, oral disorders and hearing & vision disorders) using proportional allocation. Symptoms and signs involving the respiratory system (R04–R07) were redistributed across all diseases (excluding injuries, reproductive and maternal conditions, oral disorders and hearing & vision disorders) using proportional allocation. Pneumonitis deaths (J69) were redistributed using the indirect MCODE method with all diseases (excluding reproductive and maternal conditions, oral disorders and hearing & vision disorders) in the target range.

## Morbidity estimates

### Sequelae and health states

Sequelae and health states assigned to respiratory conditions are shown in Table 5.31. As most of these conditions (except for upper respiratory conditions) are chronic, health loss was assumed to apply for the whole year.

**Table 5.31: Sequelae and health states for respiratory diseases**

Disease	Sequela	ABDS 2015 health state identifier <sup>(a)</sup>
Asthma	Asthma	52, 53, 54
Chronic obstructive pulmonary disease (COPD)	COPD	55, 56, 57
Sarcoidosis	Sarcoidosis	262, 55, 56, 57
Interstitial lung disease	Interstitial lung disease	55, 56, 57
Pneumoconiosis	Asbestosis	55, 56, 57
	Silicosis	55, 56, 57
	Other pneumoconiosis	55, 56, 57
Upper respiratory conditions	Upper respiratory	262, 207
Other respiratory disease	Other respiratory	207

(a) See Appendix Table C1.

## Prevalence estimation

### Asthma and upper respiratory conditions

The NHS 2014–15 was the main data source used to estimate the national prevalence of asthma and upper respiratory conditions. The NHS did not include people who lived in institutionalised facilities, such as hospitals or aged care facilities, so estimates on the prevalence of asthma and upper respiratory disease (mainly in the older age groups) in these institutions were not included. As well, the NHS 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 et al. 2015). The following proportions were used: 54.4% controlled, 22.6% partially controlled and 23.0% uncontrolled. Health loss was assumed to last for the entire year.

## *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. Health loss was assigned to 33% of cases based on findings from allergic rhinitis studies in the United States and Australia (Meltzer et al. 2012; Tan et al. 2017), with the remainder considered asymptomatic.

## **Chronic obstructive pulmonary disease**

Prevalence for COPD was based on measured data from the Australian arm of the Burden of Obstructive Lung Disease (BOLD) Study (Toelle et al. 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 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).

Prevalence rates determined for 2011 were applied to the 2015 estimated resident population to obtain 2015 prevalence estimates. An assumption was made that the rate of COPD in Australia had not changed significantly between these time points.

## **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 (obtained from the Western Australian Department of Health) were applied to the national hospitalisations to account for repeat admissions per person. Ratios from 2011 were used to determine 2015 prevalence under the assumption that the repeat admissions for these conditions have not changed over time.

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 obtained from GBD 2015 data (GBD 2015 Disease and Injury Incidence and Prevalence Collaborators 2016). The severity distributions used for sarcoidosis and interstitial lung disease are listed in Table 5.32.



**Table 5.32: GBD severity distributions (%) for sarcoidosis**

	Asymptomatic	Mild	Moderate	Severe
GBD 2013	23.9	55.0	16.0	5.1
GBD 2015	22.2	50.8	14.8	12.2

*Notes*

1. In the GBD study, pulmonary sarcoidosis and interstitial lung disease are grouped together.
2. The change in severity distribution between the GBD 2013 and the GBD 2015 is a result of a methodology change in 2015 in which secondary diagnosis data in hospitalisation data were used.

The severity distributions used for pneumoconiosis are listed in Table 5.33.

**Table 5.33: GBD severity distributions (%) for pneumoconiosis**

	Asymptomatic	Mild	Moderate	Severe
<b>Asbestosis</b>				
GBD 2013	30.8	43.9	17.8	7.5
GBD 2015	23.0	32.7	12.9	31.4
<b>Silicosis</b>				
GBD 2013	31.8	44.8	17.2	6.2
GBD 2015	23.4	33.2	13.2	30.2
<b>Other pneumoconiosis</b>				
GBD 2013	29.0	41.6	16.2	13.3
GBD 2015	22.8	32.3	12.8	32.1

*Notes*

1. In the GBD distributions, 'Other pneumoconiosis' did not include 'Coal workers pneumoconiosis', but the proportions were very similar.
2. The change in severity distribution between the GBD 2013 and the GBD 2015 is a result of a methodology change in 2015 in which secondary diagnosis data in hospitalisation data were used.

**Other respiratory conditions**

The prevalence of other respiratory conditions was derived using the YLD:YLL ratio for the following identified conditions: sarcoidosis, pneumoconiosis and interstitial lung disease. The ratio was applied to YLL for other respiratory conditions identified using the ICD-10 codes outlined in Appendix Table B2.

**Sub-national 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. Due to the small number of cases for pneumoconiosis, data from the NHMD and the NMD were used and the proportions applied to national estimates for the sequelae (asbestosis, silicosis and other pneumoconiosis).

**2011 and 2003 estimates**

The same methods used for the 2015 estimates were used for 2011 estimates of non-fatal burden respiratory disease. The severity distributions used for 2011 estimates are included in the tables with the distributions used for 2015 estimates so they can be easily compared. It is important to note that some of the differences between 2015 and 2011 estimates will be due

to differences in the severity distributions, particularly for sarcoidosis, pneumoconiosis and interstitial lung disease.

National 2003 estimates of asthma and upper respiratory conditions used a similar method to that outlined for 2015 and 2011 estimates but drew on the NHS 2004–05. Estimates of COPD 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.

## 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–L68, L71–L85, L87, L90–L92, L94, L98.0, L98.1, L98.8 and L98.9 were redistributed proportionally to all diseases (excluding reproductive and maternal conditions, oral disorders and hearing & vision disorders) (see Appendix table B2).

### Morbidity estimates

#### Sequelae and health states

Sequelae and health states assigned to skin disorders are shown in Table 5.34. Where these conditions are chronic, health loss was assumed to apply for the whole year (12 months).

**Table 5.34: Sequelae, health states and durations for skin conditions**

Disease	Sequela	ABDS 2011 health state identifier <sup>(a)</sup>	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 the Australian estimated resident population for all age groups, including children.

The severity distribution for dermatitis and eczema in adults was based on severity of atopic dermatitis from Plunkett and others (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 severity distribution in children was based on the study by Marks and others (1999a), which investigated atopic eczema in Australian school students (aged 4–18). The study reported that 32.1% of cases were minimal disease, 54.1% mild, 12.6% moderate and 1.2% severe. Based on expert advice, the minimal and mild groups in this study were combined into the no health loss group. 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).

## **Psoriasis**

Prevalence was based on the NHS 2014–15 self-reported psoriasis that had lasted, or was expected to last, at least 6 months. Prevalence rates derived from the NHS 2014–15 psoriasis counts were applied to the Australian estimated resident population to calculate the national prevalence. Although the NHS 2014–15 did not report on *Very remote* areas, prevalence estimates were modelled to account for these areas.

Severity distribution of psoriasis was based on results from a study of GP and dermatologist patients with psoriasis (Jenner et al. 2002). Patients who spent 15 minutes or less on treatment each day were considered asymptomatic (minimal psoriasis). People who spent 15–60 minutes each day for treatment were considered mild cases, and those who spent more than 1 hour each day were considered moderate–severe cases. Proportions of the number of people in each group were then applied to the national 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 (Marks et al. 1999b; Plunkett et al. 1999). Age-specific prevalence rates of acne from this study were applied to the Australian estimated resident population for adults aged 20 and over. The severity distribution was based on proportions that were calculated using scores from the Dermatology Life Quality Index (Marks et al. 1999b) and applied to the adult estimates.

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). Age- and sex-specific prevalence rates were applied to the Australian estimated resident population for students in this age group. For younger students, the prevalence of acne was assumed to be zero (0). The severity distribution was based on proportions calculated using scores from the Acne Disability Index (Marks et al. 1999b) and applied to the student's estimates.

## **Ulcers**

### *Pressure ulcers*

There are 3 main populations at risk of developing pressure ulcers: patients admitted to hospital; people living in residential care facilities (older Australians and people with disability); and people receiving home-based care in the community (a similar cohort to those living in residential care facilities). The prevalence of pressure ulcers was modelled separately for each of these populations based on different data sources (Table 5.35). These figures were added together to produce the total prevalence of pressure ulcers in Australia.

**Table 5.35: Summary of data sources for modelling the prevalence of pressure ulcers, by reference year and key populations**

Population at risk of pressure ulcers	Prevalence	Age distribution	Severity	Duration (if required)
<b>2015</b>				
Hospitals	CEC 2017; 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)
Residential aged care	Edwards et al. 2017	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	
<b>2003 and 2011</b>				
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	

*Pressure ulcers in the hospital:* The prevalence of pressure ulcers in hospitals was based on the proportions of hospital-based pressure ulcers from 3 jurisdictions. The proportion of pressure ulcers in New South Wales (CEC 2017), Queensland (Queensland Health 2012) and Western Australia (Mulligan et al. 2011) were applied to the number of hospitalisations (in the NHMD) in these states in 2015 and extrapolated to the remaining states/territories. This estimated the total national prevalence of pressure ulcers in the hospital.

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 Victoria, Western Australia and South Australia (see Table 5.35), which were applied to the national prevalence estimate.

Durations for each stage were based on the mean expected time to heal from ulcers, as reported by Dealey and others (2012), with more severe ulcers modelled to progress to less severe stages during the healing process. 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.

*Pressure ulcers in residential care:* The prevalence of pressure ulcers in residential care was based on the proportion of pressure ulcers sourced from a study that implemented the Champions for Skin Integrity model in 7 residential aged care facilities across 2 states in Australia (Edwards et al. 2017). Age distributions were modelled based on the findings of Santamaria and others (2009) and applied to the residential aged care population in Australia

as at 30 June 2015 (AIHW 2018c). Severity distributions were also based on the Santamaria study and were applied to the prevalence estimates.

*Pressure ulcers in home-based care:* 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); prevalence rates were applied to the population in home-based care as at 30 June 2015 (AIHW 2015b). The study also reported that a proportion of home care patients had acquired the ulcer during hospitalisation. To avoid double-counting, the proportion of patients with hospital acquired pressure ulcers was removed from the home care prevalence figure.

#### *Chronic skin ulcers*

The prevalence of chronic skin ulcers was based on GP encounters for chronic skin ulcers reported in a BEACH study (Harrison et al. 2013). The crude rates from the survey were weighted and modelled according to the method used by Harrison and others (2013) to estimate the prevalence of chronic conditions. This estimate accounted for the frequency of GP visits in the population and also for those who did not visit a GP in the year, so the prevalence is generalised to the total population in Australia. To avoid double-counting of chronic ulcers caused by diabetes (diabetic foot ulcers), the proportion of diabetic foot ulcers was removed from the chronic skin ulcer prevalence.

### **Skin infections**

The prevalence of skin infections was based on hospital separations (from the NHMD) in 2015. Separations 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. A duration of 2 weeks out of 1 year was applied to the separations to estimate the point prevalence of skin infections.

### **Other skin disorders**

The prevalence of other acute skin disorders was based on hospitalisations from the NHMD in 2015. A duration of 2 weeks out of 1 year was applied to the separations to estimate the point prevalence.

Other chronic skin disorders were based on the NHS 2014–15 count of conditions reported as ‘other diseases of skin and subcutaneous tissue’. It was estimated that about half of these conditions would correspond to ‘other chronic skin disorders’ as defined in the ABDS 2015. Age-specific prevalence rates were calculated from the NHS 2014–15 ‘other skin’ counts and applied to the Australian estimated resident population for both males and females to obtain the prevalence of other chronic skin disorders.

### **Sub-national estimates**

For dermatitis & eczema, acne and skin ulcers, the national prevalence rates by age and sex were applied to the populations for each state/territory, remoteness area and socioeconomic group to obtain the sub-national estimates. This method was used because there was a lack of prevalence data specific to the sub-national levels for these conditions.

For skin infections and other acute skin disorders, hospitalisations by sub-national groups were obtained from the NHMD and a duration of 2 weeks was applied to the separations to obtain the point prevalence.

For psoriasis and other chronic skin disorders, proportions of the prevalence at sub-national levels were derived from the NHS 2014–15 counts and applied to the national estimates to produce the sub-national point prevalence.

## **2011 and 2003 estimates**

The overarching methods used to estimate point prevalence for skin disorders was consistent for 2003, 2011 and 2015. For dermatitis & eczema and acne, the same prevalence rates (sourced from the same studies) were applied to the 2003 and 2011 populations. For skin infections and other acute skin disorders, 2003 and 2011 hospitalisations obtained from the NHMD were used to calculate the point prevalence.

For psoriasis and other chronic skin disorders, the prevalence was based on data sourced from the NHS 2004–05 and the AHS 2011–12. However, the ABDS 2015 methods changed slightly compared with the ABDS 2011 and this was updated for all 3 reference years. (See the AIHW 2016a for the previous methods used).

For pressure ulcers, only data sources applicable to the 2003 and 2011 reference years were used to estimate the prevalence in those years (see Table 5.35). Overall prevalence in hospitals was based on the study by Mulligan and others (2011) and on Queensland Health (2012). Prevalence in residential aged care facilities was estimated using 2 studies (Mulligan et al. 2011; Santamaria et al. 2009) for low-care and high-care facilities, respectively. Before July 2014, aged care places were classified as low-care and high-care and separate prevalence rates were applied to these populations. The same data sources and methods were used to estimate the prevalence in patients receiving home-based care in 2003 and 2011. Aged care and home-based care populations were sourced from the AIHW's population data on those in aged care and home care for the reference years (AIHW 2013).

For other chronic skin ulcers, the same method based on the study by Harrison and others (2013) was used to estimate the prevalence in 2003 and 2011.

## 6 Estimating the health-adjusted life expectancy

Life expectancy measures the average number of years a person can expect to live, without taking into account how healthy those years of life are. During their lifetime, a person spends time in different states of health. Health-adjusted life expectancy (HALE) extends the concept of life expectancy by considering the time spent living with ill health from disease and injury. It reflects the average length of time a person at a specific age lived in full health. HALE is measured using the morbidity and mortality experienced by the population for a particular reference year. Both life expectancy and HALE are summary measures based on experiences of the population.

HALE is typically reported:

- at birth: describing the average number of healthy years that a baby born in a particular year could expect to live, if they experienced the mortality rates and morbidity rates for that year, and
- at age 65: describing the average number of healthy years that a person at this age could expect during their remaining expected lifetime.

HALE, as described here, differs from disability-free life expectancy in that HALE includes the full experience of ill health and the impact of the health-related consequences; disability-free life expectancy as reported by the AIHW (AIHW 2017a) encompasses a broader scope of functional limitations of disability and selected long-term conditions. For a more detailed description of the differences, see AIHW 2017b.

### Method for estimating HALE

HALE is one of a range of measures of health expectancy (another, for example, is disability-free life expectancy).

In the ABDS 2015, Sullivan's method was used to calculate HALE (see Jagger et al. 2014). This method was chosen for its simplicity and suitability for available data. It requires age-specific measures of average health and age-specific mortality information from a life table.

### Estimating morbidity

Years lived with disability is a measure of the years that could have been spent in full health but were instead spent in ill health. YLD rates describe the combined time spent in ill health per 1,000 population; they are an estimate of the average experience of health loss, adjusted for severity. YLD rates expressed per person can be interpreted as the proportion of the year that each person, on average, spent in ill health, thereby providing a measure of average health in the population during that year. These rates are based on the prevalence of all health outcomes, adjusted for the duration and severity of the health consequences.

The YLD rate for men aged 40–44 in 2015 was 96.7 YLD per 1,000 men—on average, each group of 1,000 men in this age group spent a combined time of 96.7 years living in ill health in 2015. Thus, on average, each male in this age group spent 9.67% of 2015 in less than full health and, conversely, 90.3% of the year in full health. Compare this with men aged 90–94 who experienced a YLD rate of 420.4 YLD per 1,000 men; that is, on average, men in this age group spent 42% of the year in less than full health and 58% in full health.

## Estimating mortality

Life tables are statistical models used to describe the mortality of a population. They describe the number of person-years lived at each age (or age group) and the remaining years of life at each age (the life expectancy) for a hypothetical cohort that experienced the mortality rates of the population of interest in that time period.

## Calculating HALE

HALE is calculated by adjusting life table data in proportion to the average health of the population (using YLD rates to estimate the average health of the population).

A HALE calculation using Sullivan's method is shown in Table 6.1. The steps are as follows:

### Step 1: Calculate the healthy years lived by the cohort

Using data from the life table, adjust the total person-years lived ( $L_x$ ) in each age group in the hypothetical population by the average time lived in full health ( $H_x$ ), estimated using the YLD rate; that is,  $L_x$  multiplied by  $H_x$ . The result is an adjusted total person-years lived,  $L'_x$ , reflecting the combined time lived in full health in each age group of the cohort.

### Step 2: Calculate the cumulative healthy years lived by the cohort

Recalculate the cumulative number of years lived by the cohort from age  $x$  to the last age in the life table, considering only the time lived in full health. That is,  $T'_x$  is the cumulative number of healthy years lived by the cohort from age  $x$  to the top age in the life table and is the sum of the total healthy years lived by the cohort (that is, the sum of  $L'_x$  and  $L'_{(x+1)}$ ).  $T'_0$ , for example, represents the total combined healthy years lived by the whole population in the reference year.

### Step 3: Recalculate life expectancy based on healthy years lived

The last step is to recalculate the life expectancy using the adjusted cumulative number of healthy years,  $T'_x$  and the number of people surviving to each age,  $l_x$ . That is, in the same way that  $e_x$  is calculated:  $e'_x = T'_x/l_x$ . However,  $e'_x$  is the adjusted life expectancy, or the average number of healthy years lived by the cohort at age  $x$ .

This calculation is summarised in Box 6.1. The same method was applied to all population groups using life tables and YLD rates specific to all population groups (national, state, remoteness areas and socioeconomic groups) and reference years (2003, 2011 and 2015).



**Table 6.1: Example calculation of HALE, Australia, males, 2014–2016**

Male life table data (2014–2016) <sup>(a)</sup>											Key to variables		
Age <i>x</i> (years)	Population (hypothetical)	Proportion of population dying	Total person- years lived	Life expectancy	YLD rate <sup>(b)</sup>	YLD rate <sup>(b)</sup>	Average health	Total healthy person- years lived	Cumulative total healthy person- years lived	Life expectancy (healthy years)	<i>x</i>	age in years (or age group when using an abridged life table)	
	<i>l<sub>x</sub></i>	<i>q<sub>x</sub></i>	<i>L<sub>x</sub></i>	<i>e<sub>x</sub></i>	(years/ 1,000 people)	<i>p<sub>x</sub></i>	<i>H<sub>x</sub></i>	<i>L'<sub>x</sub></i>	<i>T'<sub>x</sub></i>	<i>e'<sub>x</sub></i>			<i>l<sub>x</sub></i>
	(number)	(rate)	(number)	(years)		(years/ person)	(years/ person)	(number)	(number)	(years)		<i>q<sub>x</sub></i>	the proportion of people who die between age <i>x</i> and <i>x</i> +1
												<i>L<sub>x</sub></i>	the total person-years lived in the age interval <i>x</i> to <i>x</i> +1
												<i>e<sub>x</sub></i>	the life expectancy (that is, the average of cumulative person-years lived by each person in the cohort)
												<b>YLD rate</b>	the average time spent in less than full health by every 1,000 people in the population
												<i>p<sub>x</sub></i>	average ill health; the proportion of the year spent in less than full health (average YLD per person)
												<i>H<sub>x</sub></i>	average health; the proportion of the year spent in full health (1 minus the average ill health)
												<i>L'<sub>x</sub></i>	the total healthy years lived in the age interval <i>x</i> to <i>x</i> +1 (equals <i>H<sub>x</sub></i> x <i>L<sub>x</sub></i> )
												<i>T'<sub>x</sub></i>	the cumulative years lived in full health from age <i>x</i> ; equivalent to the cumulative sum of <i>L'<sub>x</sub></i> from age <i>x</i> to the top age in the life table
												<i>e'<sub>x</sub></i>	the health-adjusted life expectancy (that is, the average of cumulative person-years lived in full health by each person in the cohort); equivalent to <i>T'<sub>x</sub></i> / <i>l<sub>x</sub></i>
0	100,000	0.00357	99,682	80.4	26.62	0.0266	0.9734	97,028	7,144,404	71.4			
1	99,643	0.00029	99,627	79.7	22.56	0.0226	0.9774	97,379	7,047,376	70.7			
2	99,614	0.00016	99,606	78.8	22.56	0.0226	0.9774	97,359	6,949,996	69.8			
3	99,599	0.00014	99,592	77.8	22.56	0.0226	0.9774	97,345	6,852,638	68.8			
4	99,585	0.00012	99,579	76.8	22.56	0.0226	0.9774	97,332	6,755,292	67.8			
5	99,573	0.00011	99,567	75.8	32.74	0.0327	0.9673	96,308	6,657,960	66.9			
6	99,562	0.0001	99,557	74.8	32.74	0.0327	0.9673	96,298	6,561,653	65.9			
7	99,553	0.00009	99,548	73.8	32.74	0.0327	0.9673	96,289	6,465,355	64.9			
97	4,985	0.2743	4,275	2.6	447.49	0.4475	0.5525	2,362	7,274	1.5			
98	3,618	0.29584	3,060	2.5	447.49	0.4475	0.5525	1,691	4,912	1.4			
99	2,547	0.31995	2,120	2.3	447.49	0.4475	0.5525	1,171	3,221	1.3			
100	1,732	0.34389	3,703	2.1	446.48	0.4465	0.5535	2,050	2,050	1.2			

Notes

1. The table was obtained from the ABS Australian Demographic Statistics, Dec 2016 (ABS 2017a).
2. The YLD rates were calculated as part of the ABDS 2015.

### Box 6.1: Calculating HALE using Sullivan's method

$$HALE_{x,s} = \left( \sum_{x=0}^{\text{last age}} (L'_{x,s}) \right) / l_{x,s}$$

and

$$L'_{x,s} = L_{x,s} (1 - p_{x,s})$$

$$L'_{x,s} = L_{x,s} (H_{x,s})$$

where:

HALE is health adjusted-life expectancy;

x is the exact age for which life expectancy or health adjusted-life expectancy is to be estimated;

s refers to sex;

$L_{x,s}$  refers to the number of life-years lived in the age group x;

$L'_{x,s}$  refers to the health-adjusted number of life-years lived in the age group x, for sex, s;

$l_{x,s}$  is the number of survivors at age x (as described above for the life table), for sex, s;

$p_{x,s}$  is the prevalence of ill-health, estimated by YLD rate for each, x, age and sex, s;

$H_{x,s}$  represents the complement of  $p_{x,s}$  and is the average level of health-related quality of life; it has a value between 0 and 1 where a value of 1 indicates full health.

## Data sources

In the ABDS 2015, HALE was calculated at the national level for 2003, 2011 and 2015 and for sub-national populations (state and territory, remoteness areas and socioeconomic groups) for 2011 and 2015. Life table data were sourced from published and customised life tables (see Appendix Table E1).

## Alignment of non-fatal burden and life table data

The life table data and measures of average health must align to calculate HALE. National and state and territory life table data were available for single year age groups capped at 100 or more, while abridged life table data were used for remoteness areas and socioeconomic groups. That is, age groupings were for 5-year groups (with infant and 1–4 age group splits) and capped at age 85 or more.

YLD rates were available at the national level for 5-year age groups to 100 or more (with infant and 1–4 age group splits) and in the same disaggregation for state and territory, remoteness areas and socioeconomic groups, except the age cap was 85 or more.

As a result, YLD rates (expressed as average health) for a 5-year age group were aligned to each single year age group as appropriate. For example, in Table 6.1, the YLD rate of 32.74 YLD per 1,000 children aged 5–9 was used to estimate HALE for each single year age group in the life table (5, 6, 7, 8 and 9 years).

As well, state and territory life table data were collapsed for ages 85 and over to align with the YLD rates for states and territories capped at age 85 or more.

Results presented for age 65 for remoteness areas and socioeconomic groups represent life expectancy and HALE results calculated for the age group 65–69.

## Sub-national estimates

HALE estimates for states were calculated using state-specific life expectancy data (ABS 2007, 2012, 2017e) and state-specific YLD rates.

For remoteness areas, HALE was estimated for 4 remoteness areas: *Major cities*, *Inner regional*, *Outer regional*, and *Remote and Very remote* combined (reported as *Remote and very remote*), based on the ASGS 2011. HALE estimates for remoteness areas are described in the ABDS 2015 to align with the first age of the age group; for example, HALE at age 65 represents HALE for the age group 65–69.

For socioeconomic groups, life table data were available for 5 socioeconomic groups: groups 1 to 5, based on the 2011 SEIFA IRSD. The highest socioeconomic group (group 5) represents the least disadvantaged areas and the lowest socioeconomic group (group 1), the most disadvantaged areas. HALE estimates for socioeconomic groups are described in the ABDS 2015 to align with the first age of the age group; for example, HALE at age 65 represents HALE for the age group 65–69.

The AIHW calculates socioeconomic differences using SEIFA indexes divided into population-based quintiles. With this approach, approximately one-fifth of the population is allocated to each quintile, regardless of the underlying geographical area. In this report, the YLD rates used to estimate the proportion of ill health in the 5 socioeconomic groups were derived this way.

# 7 Overarching methods and choices for 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 methodology allows death and health loss to be attributed to specific underlying (or linked) risk factors. Quantification of the impact of risk factors assists evidence-based decisions about where to direct efforts to prevent disease and injury and to improve population health.

The methods used to quantify the impact of risk factors in the ABDS 2015 are described in this chapter (for specific risk factor methods, see Chapter 8).

## 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 (TMRED)**.

**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 (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.

**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's occurring in the exposed group to the probability of its occurring in the unexposed group.

**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.

**risk factor:** Any factor that causes or increases the likelihood of illness or death due to a disease or injury or other unwanted condition or event.

**theoretical minimum risk exposure distribution (TMRED):** The risk factor exposure distribution that will lead to the lowest conceivable disease burden.

The burden attributable to risk factors is generally estimated using PAFs applied to the disease burden estimated as per the previous chapters.

## Methodological developments since the ABDS 2011

Most of the risk factors methods were the same as those used in the ABDS 2011. However, some methods have changed following recent ABDS extension projects for risk factors, which included reviews of the methods and the diseases linked to each risk factor.

As a result, the diseases diabetes and chronic kidney disease were added to the ABDS 2015 as risk factors, using a diseases-as-risk-factor approach (AIHW 2016c). These diseases act as risk factors, in that exposure is linked to disease outcomes. The GBD approach was used, which included diabetes and chronic kidney disease as part of the exposure to the risk factors high blood plasma glucose and impaired kidney function, respectively.

Other risk factors that had method changes as part of extension projects are:

- alcohol (AIHW 2018d)
- illicit drug use (AIHW 2018d)
- physical inactivity (AIHW 2017c)
- intimate partner violence (Ayre et al. 2016)
- overweight and obesity (previously named high body mass) (AIHW 2017d).

As well, risk factors for dementia were identified as part of an extension project (AIHW 2016d).

New methods have been developed specifically for the ABDS 2015, based on updates from the GBD 2016 and on expert advice. The most notable of these was the changes to dietary risks. Dietary risks were reviewed in detail, including the risk factors included, how exposure is calculated, the linked diseases and the definition of foods. These changes are described in detail in Chapter 8.

The methods for the dietary risk factors diet high in sugar sweetened beverages and diet high in sodium were updated, using new methods developed by the GBD 2016 (GBD 2016 Risk Factors Collaborators 2017). These risk factors mediate through other risks in that they increased exposure in the population to other risk factors. For example, the methods for these risk factors involve estimating how much high blood pressure and overweight and obesity is caused by a diet high in sodium and in sugar sweetened beverages, respectively.

As well, a study from the literature was used to expand the risk factor child sexual abuse to child abuse and neglect. This expanded risk factor includes burden due to physical, sexual and emotional abuse and neglect in Australia (Moore et al. 2015).

## Steps in estimating risk factor attributable burden

The basic steps of estimating risk factor attributable burden are:

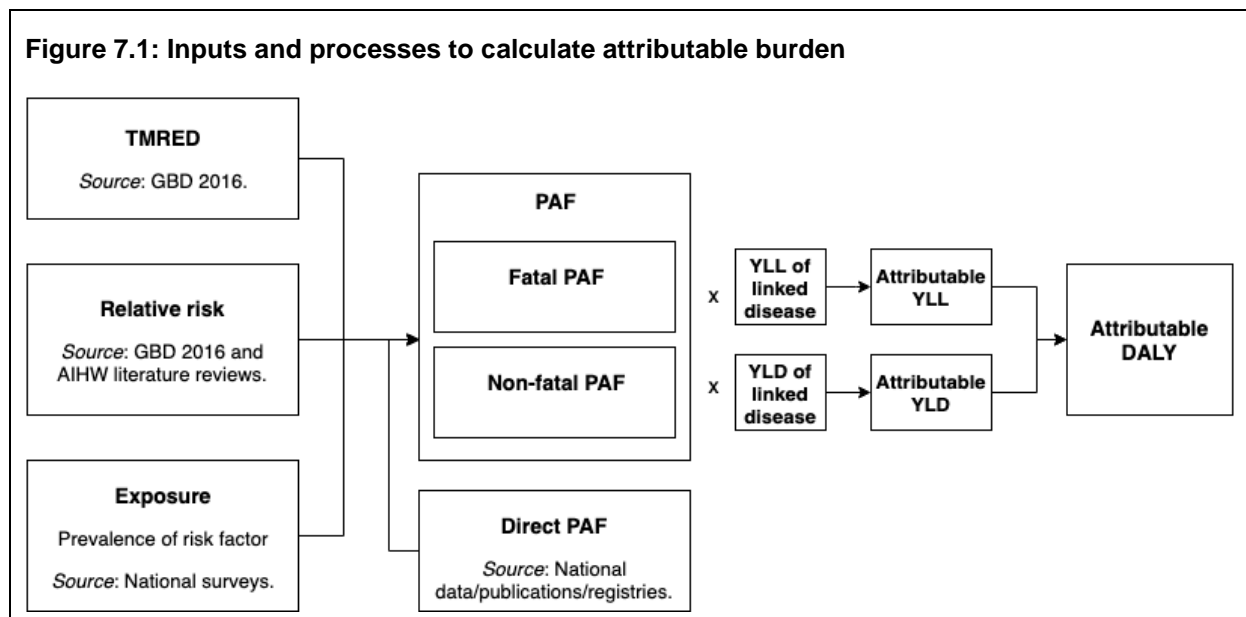
1. select risk factors
2. identify linked diseases based on convincing or probable evidence in the literature that the risk factor has a causal association with increased prevalence or mortality
3. define the exposure to the risk factor that is not associated with increased risk of disease (the theoretical minimum risk exposure distribution, or TMRED, or counterfactual)
4. estimate the PAFs by either a direct method or the comparative risk assessment method:
  - (a) if PAFs appropriate to the disease and population in question are available from a comprehensive data source (such as a disease register), they are estimated directly from this data source (named a direct PAF in this report) and do not require steps 5, 6 and 7

(b) if not, PAFs are created using the comparative risk assessment method, which involves steps 5, 6 and 7

5. define the amount of increased risk (relative risk) of morbidity or mortality for the linked disease due to exposure to the risk factor
6. estimate exposure to each risk factor in the population
7. use these inputs to calculate the PAF. The PAF has a value between 0 and 1, where 0 means there was no burden attributable to the risk factor and 1 means that all the burden for the linked disease was attributable to the risk factor.

The burden attributable to each risk factor is calculated by applying the PAFs for each linked disease to the relevant YLL and YLD.

This process is shown in Figure 7.1.



## Selection of risk factors

The risk factor list describes the specific risk factors considered as underlying causes of health loss through their causal association with particular diseases. In contrast to the disease list, which is exhaustive, and where an established classification system (the International Statistical Classification of Diseases and Related Health Problems) 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 was used to 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 2015, 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 2016
  - the ABDS 2011unless its inclusion in the ABDS 2015 conflicted with other criteria.

## **Substantial impact and policy interest**

- Be of considerable importance to national disease burden based on previous studies (ABDS 2011; GBD 2016).
- Be of substantial Australian 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 measurable, 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 sizes per unit of exposure of outcome-specific impacts
  - evidence to support the ability of effect sizes to be generalised to populations, other than those included in the available studies, or to satisfactory models for extrapolating them.

## **ABDS 2015 risk factor list**

The ABDS 2015 identified 38 risk factor components or exposures (such as cannabis and cocaine use) that combine to 18 individual risk factors (such as illicit drug use). These were broadly grouped into categories (behavioural, metabolic and environmental risks). The risk factors included in the ABDS 2015 are listed in Appendix Table F2.

Due to methodological reasons, many of these 18 risk factors are the sum of estimates from different measures of exposure to risk factors that are in addition to the 38 reported (for example, tobacco use is the sum of current tobacco use and the smoking impact ratio). These measures of exposure are listed for each risk factor in Appendix Table F2. Specific methods, including linked diseases and TMREs, are shown in Appendix Table F2 and described in more detail in Chapter 8.

There is a high and complex degree of interrelatedness between the chosen risk factors, potentially causing biases. For this reason, risk factors were analysed and reported individually. A combined estimate is reported for all risk factors and for all dietary risk factors—using a multiplicative method that adjusts for the co-occurrence of multiple risk factors—to estimate the burden attributable to multiple risk factors (described in the section headed Combined risk factor analysis later in this chapter).

## **Risk factors not included in the ABDS 2015**

Sub-optimal breastfeeding, childhood underweight and exposure to lead (included in the GBD) were not included in the ABDS 2015:

- Sub-optimal 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. This was because the effect sizes available from the GBD 2016 were sourced from developing countries and related to infectious diseases only, failing to capture the increased likelihood of chronic disease in later 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.

- Exposure to lead was also excluded because exposure data were not available for Australia.
- Exposure to per- and poly-fluoroalkyl substances (PFAS) was excluded as a risk factor as there is insufficient evidence in the literature of disease outcomes (Department of Health 2018).

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. This was due to the resources that would have been needed to undertake the large and complex body of work required (such as developing appropriate definitions directly related to health, and sourcing disease-specific relative risks). Estimating exposure to social determinants is further complicated in that their impact can be accumulative over the life course and subsequent 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. Some indication of the effect of social determinants on the health burden is provided by estimating burden by socioeconomic area in this study.

Work was begun as part of this study to estimate the contribution of heatwaves to the burden of disease. This risk factor was found to meet the criteria for inclusion but further methodological developments are needed before it can be included.

## Selection of linked diseases

A linked disease is 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, these diseases are linked to the risk factor high fasting plasma glucose. The risk factors and linked diseases selected for the ABDS 2015 are shown in Appendix Table F2.

Linked diseases were included where there was sufficient evidence of a causal link. This is defined as having convincing or probable evidence measured against criteria 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.

The linked diseases were spread across 15 disease groups. Some risk factors had a single linked disease, while others were paired with many outcomes across the disease groups.

The ABDS 2015 adopted relevant linked diseases used in the GBD 2016 (GBD 2016 Risk Factors Collaborators 2017) and those identified by the AIHW from literature reviews for selected risk factors as part of extension projects (AIHW 2016c, 2017c, 2017d, 2018d). The details of why any additional linked diseases were selected or not included when compared with the GBD is described in the methods chapter of these reports.

The linked diseases for dietary risk factors were reviewed as part of this study, based on the GBD 2016, a literature review and expert advice.

The risk factors diet high in sugar sweetened beverages and diet high in sodium were linked to other risk factors (overweight and obesity and high blood pressure, respectively), which are then linked to diseases. The impact of these risk factors was estimated by the amount of exposure to the linked risk factor that they cause and therefore the diseases linked to that risk factor. They have the linked diseases of the risk factor secondary in the pathway.

Dementia was linked to risk factors where it was identified by the AIHW as having sufficient evidence (Appendix Table F2; AIHW 2016d).

## 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 and 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 2015, 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 BMI), 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 reflects 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 (GBD 2016 Risk Factors Collaborators 2017). 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 20–25 kg/m<sup>2</sup> with a standard deviation of 1.

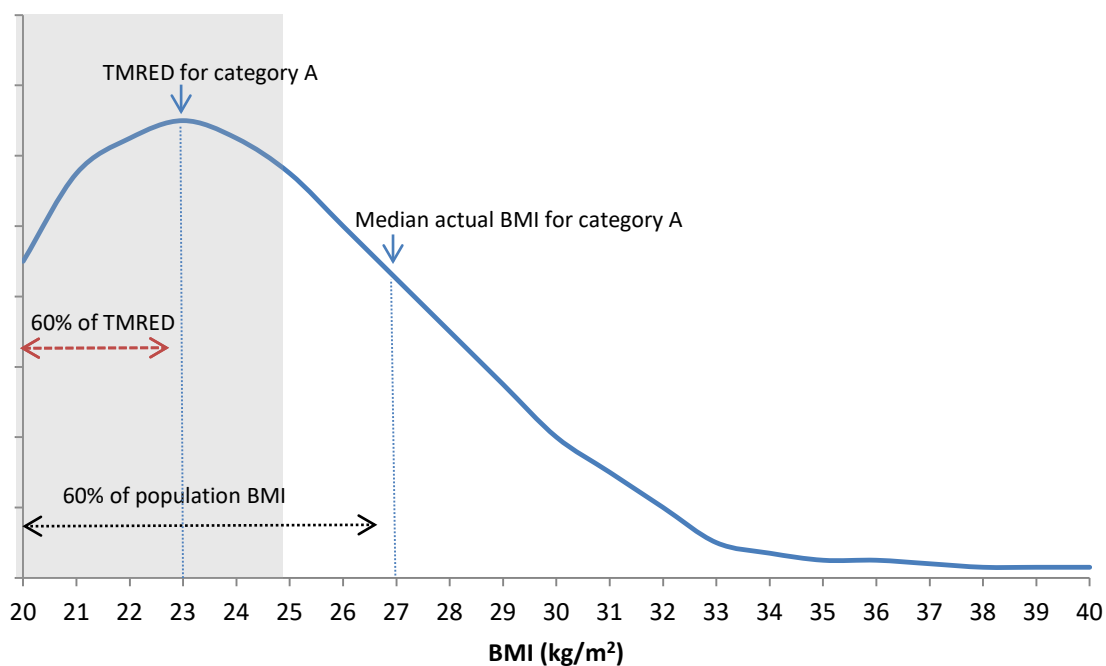
Where the TMRED is a range, exposure to risk is not dichotomous (that is, at risk or not at risk). In this situation, the measure of attributable burden cannot be estimated by simply comparing each level of exposure in the population with the endpoints. Instead, to determine how much burden each exposure level contributes compared with TMRED, the relative position in the range of the level of exposure is compared with its relative position in the range of the TMRED. The appropriate TMRED value for each category of exposure depends on the placement of their

category within the risk factor exposure distribution of the population, starting at the lowest TMRED possible.

An example of estimating a TMRED value from a category of BMI 26.0–27.9 (named A) is shown in Figure 7.2. If the median within category A is a BMI of 27.0 kg/m<sup>2</sup> and this BMI is greater than 60% of the population's BMI, the TMRED value for category A is equal to 60% of the possible TMRED values from within this range (20 kg/m<sup>2</sup> up to 25 kg/m<sup>2</sup>), assuming the TMRED distribution is uniform. The TMRED for category A is then a BMI of 23.

This model assumes that a healthy BMI (the BMI levels not associated with disease outcomes) is a range, as opposed to a single value for the entire population. The level of risk of disease outcomes for each person in the population is then calculated, based on the level of actual BMI compared with the TMRED value from within the range.

**Figure 7.2: Example of estimating the TMRED for a category of overweight and obesity**



*Note:* The shaded range in the figure refers to the TMRED, which is between 20 kg/m<sup>2</sup> and 25 kg/m<sup>2</sup>. Category A is the BMI of 26.0 to 27.9 and the median of this category is 27.0.

## 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 2015, the definitions of risk factor exposures have been adopted where possible from the GBD 2016 (GBD 2016 Risk Factors Collaborators 2017) and the AIHW review of the literature (AIHW 2017c, 2017d, 2018d).

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 F for the data selection criteria and Appendix table F1 for the scoring matrix). Only data sources that met these criteria were included in the study.

Estimates of Australian population distributions of risk factor exposure by age and sex have been based on a variety of data sources:

- ABS apparent consumption of alcohol data
- AHS 2011–12
- Census of population and housing
- Kirby Institute annual surveillance reports
- ABS Labour force survey
- National Drug Strategy Household Survey (NDSHS) 2016
- NHS 2014–15
- National HIV Register
- National Homicide Monitoring Program
- NHMD
- NMD
- Personal Safety Survey (PSS) 2016
- Safe Work Australia
- state-based air monitoring stations.

Some risk factors (such as illicit drug use) had several different measures or definitions of exposure. For illicit drug use, these included opioid use, amphetamine use, cannabis use, cocaine use, other illicit drug use as well as unsafe injecting practices. These different measures of exposure are mutually exclusive for illicit drug use and can be summed.

The risk factor exposure for comparative risk assessment is measured as either a:

- categorical variable (with a set number of mutually exclusive categories), or
- continuous variable.

Some categorical risk factors are measured through relatively straightforward dichotomous descriptions (for example, the proportion of people exposed to second-hand smoke versus the proportion who were 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. This approach was used for the risk factors alcohol use and low bone mineral density.

For the ABDS 2015 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 F2. 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, the AHS 2011–12), they were extracted at such granularity as to ensure that the RSE 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 use estimates with RSEs 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 2015 adopted relative risks estimated by the GBD 2016 or the AIHW review of the literature (AIHW 2017c, 2017d, 2018d; GBD 2016 Risk Factors Collaborators 2017). The GBD relative risks used were judged appropriate to be used globally, in different countries and for different ethnicities.

The relative risks from the GBD 2016 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. They were estimated with direct evidence data as described for each risk factor in Chapter 8.

Effect sizes used were adjusted for confounders ('parallel' risk factors), but not for factors that occur successively along the causal pathway. For example, relative risk of coronary heart disease due to physical inactivity was not adjusted for high blood plasma glucose, 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. The exceptions were:

- diet high in fruit and diet high in vegetables, which had multiplicative curves, as advised by experts, and
- overweight and obesity, which were determined by the literature (AIHW 2017d).

The relevant relative risk to apply to each exposure category was determined as the relative risk for the median survey response of that category. For example, for the proportion of the population who ate 80 g–120 g of fruit, the relative risk for the median, which is 111 g in this example, was applied. When the exposure category included an open-ended range, the median in this range was also used.

## 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 linked disease 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 linked diseases 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.

## Calculating the 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 PAFs to the ABDS disease list

A small number of linked diseases and injuries sourced from the GBD 2016 did not align to the ABDS disease list (Appendix Table F3). This was due to the GBD's disaggregating causes to a further level—for example, stroke, which was estimated as a single disease in the ABDS 2011, had only relative risks from the GBD 2016 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 and others (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 the GBD 2016 was used. Appendix table F3 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 PAFs for haemorrhagic stroke were 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, attenuation factors were used to attenuate the PAFs for the risk factor secondary to the other factor in the same causal pathway. The attenuation factors were sourced from the GBD 2016 (GBD 2016 Risk Factors Collaborators 2017).

For example, to reflect the causal pathway of overweight and obesity's increasing the risk of high blood pressure, which, in turn, increases the risk of coronary heart disease. The amount of mediation (the attenuation factor) of overweight and obesity's causing high blood pressure and then coronary heart disease was estimated to be 31% by the GBD 2016. The PAFs for high blood pressure's causing coronary heart disease is attenuated by 31% to provide the necessary independence assumption required for step 2.

Secondly, to prevent the combined disease burden's 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  $i$

$\prod$  is the product of overall 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, and thus 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.

## Attributable burden estimates by socioeconomic group

The burden attributable to risk factors was estimated by socioeconomic group in 2015. The risk factors were not estimated by state or remoteness as this was not in scope for this project. It was not possible to estimate exposure to the risk factors child abuse and neglect, low bone mineral density, sun exposure and air pollution by socioeconomic group.

For some risk factors, modelling was used to estimate exposure by socioeconomic group from the relevant survey. This is due to high RSEs when trying to estimate directly from the relevant survey. For these risk factors, exposure by socioeconomic group was estimated by comparing the mean estimate of exposure in each quintile and the mean national exposure from the survey by age and sex. The absolute change between these estimates was then used to adjust unit record data to reflect exposure to the risk factor in each socioeconomic group.

The methods for each risk factor are described in Chapter 8.

## 2011 and 2003 estimates

Where possible the burden attributable to risk factors was calculated for 2011 and 2003. Exposure distributions for air pollution was estimated only for 2015 as PM<sub>2.5</sub> (particulate matter 2.5) could not be measured in 2003, and estimates for 2011 were not comparable to those for 2015. Exposure to high plasma glucose could not be estimated in 2003. Child abuse and neglect, low bone mineral density, iron deficiency and sun exposure PAFs were based on data relevant to the whole period of the study and were considered appropriate for 2003, 2011 and 2015.

The way exposure was estimated for risk factors in 2003 and 2011 is described in the individual risk factor section in Chapter 8.

## Changes in risk factor exposure over time

Changes in exposure to the risk factors over time was estimated using the percentage change in *total PAF*.

The total PAF, which is the sum of PAFs for each risk factor, was estimated using the following steps:

- age-specific PAFs were weighted by the 2001 standard population to estimate a PAF for all ages (by risk factor, linked disease, sex and type of burden—PAFs for fatal and non-fatal burden)
- the PAFs for all ages were then summed to estimate the total PAF (by risk factor and type of burden)
- when the total PAF was different by type of burden, the average of these total PAFs was used. These risk factors include alcohol use, illicit drug use, intimate partner violence and occupational exposures & hazards.

The total PAF will vary between risk factors by the number of linked diseases and the size of the PAFs; however, this is the same over time for each risk factor. Any changes over time are risk weighted because, in the PAF calculation exposure, the risk factor is multiplied by the relative risk. Changes in exposures associated with the most increased risk of the linked disease have the highest influence on the estimate.



## 8 Risk factor specific methods

This chapter describes in detail the methods unique to each risk factor included in the ABDS 2015. It is focused on the calculation of exposure estimates, as this was the aspect of risk estimation most influenced by Australia-specific data. The amount of detail described for each risk factor varies; more detail is included for risk factors for which there were new developments in the ABDS 2015, in particular, dietary risks.

Chapter 7 describes the overall method used to calculate the PAFs and attributable burden, including the selection of linked diseases, estimation of effect sizes (relative risks), and assumptions for TMREs (see also Appendix Table F2).

The linked diseases and relative risks were sourced from the GBD 2016 or an AIHW review of the literature as described in this chapter and in Chapter 7. Most TMREs were also sourced from the GBD 2016, with the exceptions described in Chapter 7.

Exposure to risk factors in the lifetime of the individuals in the population can influence the proportion of burden in the reference year. For risk factors such as tobacco use, occupational risks, alcohol use, child abuse and neglect, illicit drug use, and unsafe sex, the burden can continue to exist from past exposure levels. Where evidence of ever being exposed to a risk factor 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 overweight and obesity, current exposure can 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 blood pressure occurs for people aged 25 and over. The choices for population groups and type of burden (fatal or non-fatal) were informed by the GBD 2016 (GBD 2016 Risk Factor Collaborators 2017). The population group for which attributable burden from a given risk factor has been estimated is described in each section.

Also, both fatal and non-fatal burden are relevant for most linked diseases in the study. For others, such as back pain and problems linked to occupational risks, only non-fatal burden has been estimated.

### Tobacco use

The impact of tobacco use captures the burden attributable to current smoking, past smoking (in people aged 30 and over) and exposure to second-hand smoke in the home (in people of all ages). In the GBD 2016, chewing tobacco was added as an exposure to tobacco use. Due to very low prevalence in Australia, chewing tobacco was not included in the ABDS 2015.

### Population attributable fraction estimated using comparative risk assessment

#### Linked diseases and relative risks

Linked diseases and relative risks were sourced from the GBD 2016 (GBD 2016 Risk Factor Collaborators 2017). More detail on the methods will be described further in the report *Burden of tobacco use in Australia: Australian Burden of Disease Study 2015*, which is expected to be published in 2019 (AIHW forthcoming 2019).

## Exposure estimates

The NDSHS 2010 was used to estimate the proportion of the population who are current smokers (5-year lagged). Using these data for current smokers allows for a 5-year lag between exposure and these disease outcomes. Current smoking (5-year lagged) was linked to cardiovascular diseases, diabetes, asthma and respiratory infections. Exposure to current tobacco smoking (5-year lagged) was calculated from the proportion of individuals in the NDSHS 2010 who reported smoking daily, weekly or less than weekly.

The NDSHS 2016 was used to estimate the proportion of non-smokers exposed to environmental tobacco in the home (second-hand smoke).

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 recent prevalence. 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 2015 (by age and sex) from the NMD was compared with lung cancer mortality rates among a cohort of smokers and never-smokers in the United States (Peto et al. 1992). The excess mortality seen in the Australian population, compared with this 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 30–39 resulted in unreliable PAFs.

## Estimates by socioeconomic group

Exposure estimates by socioeconomic group were based directly from the same data source as the national exposure estimates.

## 2011 and 2003 estimates

The NDSHS 2007 was used to estimate the proportion of the population who are current (5-year lagged) smokers for 2011. The NDSHS 2010 was used to estimate the proportion of non-smokers exposed to second-hand smoke. The NMD 2011 was used to estimate lung cancer mortality.

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.

## Alcohol use

The impact of alcohol use was estimated in people aged 15 and over. The burden attributable to this risk factor was calculated as described in detail in the AIHW publication *Impact of alcohol and illicit drug use on the burden of disease and injury in Australia: Australian Burden of Disease Study 2011* (AIHW 2018d). Note that the risk factor is alcohol use while alcohol use disorders is a linked disease.

## Population attributable fraction calculated with direct evidence

In the GBD study, the linked diseases chronic liver disease due to alcohol and liver cancer due to alcohol were entirely attributed to alcohol use, and no relative risks were published for use in the comparative risk assessment approach. In the ABDS 2015, chronic liver disease and liver cancer were not broken down to this level. The PAFs for chronic liver disease were estimated from the proportion represented by chronic liver disease due to alcohol of all chronic liver disease burden, as estimated for Australia by the GBD 2016. The same method was used to estimate the PAFs for liver cancer. The burden of *alcohol dependence* (the linked disease) was fully attributed to alcohol use (the risk factor).

Direct evidence was used to derive the PAFs for accidental poisoning linked to alcohol use, using the mention of specific drugs recorded in the NMD 2015 as described by the AIHW (2018d).

## Population attributable fraction estimated using comparative risk assessment

### Exposure estimates

The proportions of the Australian population who are current drinkers, former drinkers or never drank alcohol were sourced from self-reported data in the NDSHS 2016. However, the amount of alcohol self-reported to be consumed by current drinkers in this and other surveys is known to be an underestimate of actual consumption (Rehm et al. 2010).

To overcome this, alcohol sales data were used to inflate the survey estimates. The total volume of alcohol sold in Australia was sourced from the apparent consumption of alcohol (ABS 2018b). In the ABDS 2015, 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 and others (2010).

The proportion of self-reported lifetime abstainers and ex-drinkers from the NDSHS 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 consumption for that year. In 2015, the inflation factor was estimated to be 1.29.

Following methods used in Rehm and others (2010) and in the GBD 2010, 80% of the alcohol available nationally was assumed to have been consumed (Lim et al. 2012). Only a proportion (80%) of alcohol sold in Australia was used, because the total figure includes 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. This distribution was used to estimate the proportion of the population who consumed alcohol in categories relevant to the relative risks.

### Estimates by socioeconomic group

Exposure estimates by socioeconomic group were based directly from the same data source as the national exposure estimates.

## 2011 and 2003 estimates

Exposure estimates for 2011 were calculated using data from the NDSHS 2010 and alcohol sales data for 2011, while exposure for 2003 was calculated using data from the NDSHS 2004 and alcohol sales data for 2003. Both followed the method for estimating exposure used for 2015. Direct PAFs were calculated using the method for 2015, which was based on the GBD 2016 estimates for 2011 and 2003.

## Physical inactivity

Burden due to physical inactivity was estimated in people aged 20 and over. The burden attributable to this risk factor was calculated as described in detail in the AIHW publication *Impact of physical inactivity as a risk factor for chronic conditions: Australian Burden of Disease Study* (AIHW 2017c).

## Population attributable fraction estimated by comparative risk assessment

### Exposure estimates

Population exposure to physical inactivity was treated as a categorical variable. The categories describe the range of total activity per week, as measured by the total metabolic equivalent of tasks (METs). This measure encompasses the rate of energy expenditure, with one (1) MET equivalent to 1 kcal/kg/hr, which is about the energy expended in sitting. The higher the MET, the greater the energy expended. The calculation of METs requires the input of:

- time spent undertaking the activity in 1 week ( $T$ )
- intensity score for that specific activity ( $I$ ).

The total MET for each activity is calculated as:

$$MET = T \times I$$

In this study, the total MET score describes the total rate of energy expended across 4 activity domains: leisure, transportation, occupational, and household. The categories included:

- Sedentary: fewer than 600 METs per week
- Low levels of activity: 600–3,999 METs per week
- Moderate levels of activity: 4,000–7,999 METs per week
- High levels of activity: 8,000 METs and over per week.

These categories align with relative risks provided by the GBD 2016 and were used in the ABDS 2011.

The National Nutrition and Physical Activity Survey (as part of the AHS 2011–12) was used to obtain the inputs required to calculate METs for occupational activity (ABS 2013a). The METs for leisure were estimated from the trend in METs reported in successive health surveys, including the NHS 2001, the NHS 2004–05, the NHS 2007–08, AHS 2011–12 and the NHS 2014–15. The change over time was used to adjust estimates of exercise for leisure in the AHS 2011–12 to represent 2015. Estimates of METs for transport were based on the AHS 2011–12 as no trend information was available. The number of adjusted self-reported minutes spent in each activity per week was multiplied by the intensity scores as provided by the AHS 2011–12 to calculate the total MET for each individual in the survey.

The AHS 2011–12 and the NHS 2014–15 do not provide information on the time spent and the intensity of activity due to household chores, so this was obtained from alternative data sources. The time taken on specific household chores was obtained from the ABS Time Use Survey 2006 and this estimate was used in 2015 and 2011 (ABS 2008). This survey provides detailed information on daily activity patterns of people in Australia and the time allocated to different activities. The time spent undertaking household chores (excluding meal and drink preparation) by sex in 10-year age groups was extracted and multiplied by the conservative intensity of 3.0. The calculated METs by age and sex were added to the calculated METs from remaining domains to provide the total MET.

Prevalence was estimated from the proportion of people within each activity category once the METs from each domain were summed.

## **Estimates by socioeconomic group**

Exposure estimates by socioeconomic group were estimated for physical activity from leisure in the NHS 2014–15. National estimates were used for all other domains of activity (occupational, household chores and transport).

### **2011 and 2003 estimates**

The number of total METs in 2011 was estimated from the AHS 2011–12 using the same method as used in 2015. METs for occupation and household chores were the same as in 2015 as no further data were available.

The number of total METs in 2003 for leisure was estimated using the same trend calculated to estimate METs in 2015. The AHS 2011–12 data were adjusted based on this trend to represent 2003. Total METs were calculated using the same method as used in 2015. METs for occupation and household chores were the same as in 2015 as no further data were available.

## **Illicit drug use**

The impact of illicit drug use was estimated in people aged 15 and over. The burden attributable to this risk factor was calculated as described in detail in the AIHW publication *Impact of alcohol and illicit drug use on the burden of disease and injury in Australia: Australian Burden of Disease Study 2011* (AIHW 2018d).

## **Population attributable fraction estimated using direct evidence**

### **Unsafe injecting practices**

PAFs for the linked diseases for unsafe injecting practices (chronic liver disease, hepatitis B, hepatitis C, HIV/AIDS and liver cancer) were calculated from the NNDSS data published in the annual surveillance reports by The Kirby Institute (The Kirby Institute 2016).

### **HIV/AIDS**

For HIV/AIDS, direct PAFs were calculated from the proportion of diagnosed AIDS cases in 2015 who were exposed to unsafe injecting practices with or without homosexual contact.

### **Acute hepatitis B and C**

For acute hepatitis B and hepatitis C, the direct PAFs were calculated from the proportion of newly acquired hepatitis B or hepatitis C infections in 2015 who were exposed to unsafe injecting practices with or without homosexual contact.

## **Chronic liver disease and liver cancer**

### *Chronic hepatitis C infection*

The rates of decompensated cirrhosis (chronic liver disease), hepatocellular carcinoma (liver cancer) and liver transplants due to hepatitis C for 2006 to 2015 are published in the 2016 Annual Surveillance Reports (The Kirby Institute 2016). These were multiplied by the earliest year (1997) of exposure data estimates available to determine the proportion of hepatitis C related morbidity due to unsafe injecting practices.

The proportion of chronic liver disease and liver cancer due to unsafe injecting practices was then estimated by quantifying the rate of hepatitis C related morbidity from the total prevalence for liver cancer and chronic liver disease in 2015, as estimated in the ABDS 2011.

### *Chronic hepatitis B infection*

The Kirby Institute reported that 5.7% of people living with chronic hepatitis B in 2015 had acquired this condition through unsafe injecting practices. This is similar to Australian estimates reported by other published studies for the years 2011 (5.7%) and the year 2000 (4.7%) (MacLachlan et al. 2013; O'Sullivan 2004).

The proportion of these chronic outcomes being chronic liver disease or liver cancer was then estimated using total disease prevalence data from the ABDS 2015.

## **Accidental poisoning**

The direct PAFs for accidental poisoning linked to specific illicit drugs was estimated using the number of deaths due to accidental poisoning with a mention of each drug type compared with the total number of accidental poisoning deaths in 2015 in the NMD. These methods are described in more detail in the section on alcohol use (see earlier in this chapter). The PAFs were also applied to non-fatal burden due to accidental poisoning.

## **Illicit drug dependence**

All of the burden due to drug use disorders (including amphetamine, cannabis, cocaine, opioid and other illicit drug use disorders) was attributable to illicit drug use (a PAF of 1).

## **Population attributable fraction estimated using comparative risk assessment**

### **Exposure estimates**

There are 2 types of exposure to drug use estimated for the risk factor illicit drug use: drug dependence and driving under the influence of illicit drugs. Estimates of the exposure to drug dependence are sourced from prevalence estimates for the relevant drug use disorder from the ABDS 2015. Exposure to drug dependence—not drug use—was used in this study.

Exposure to driving under the influence of illicit drugs was estimated from the 2016 NDSHS—specifically, the proportion of the population that responded yes to the question: 'In the last 12 months did you undertake the activity—drove a motor vehicle—while under the influence of or affected by illicit drugs?' However, these data do not provide details on the type of drug used while driving and are likely to be an underestimate.

The type of drug used while driving was sourced by the relative prevalence of the use of different drugs self-reported in the NDSHS. This data source was used as a source of drug type in preference to roadside drug testing, as it included a full range of illicit drugs associated with driving impairment and was not impacted by the ability to measure the presence of the drug in saliva tests.

## Estimates by socioeconomic group

The data source used for the national estimates as described above also provided data by socioeconomic status, except for unsafe injecting practices for which these data were not available. The national PAFs were used for each socioeconomic group for diseases linked to unsafe injecting practices.

### 2011 and 2003 estimates

The burden attributable to illicit drug use in 2011 was estimated using the NDSHS 2010 and The Kirby Institute data for 2011 as described in *Impact of alcohol and illicit drug use on the burden of disease and injury in Australia: Australian Burden of Disease Study 2011* (AIHW 2018d), using the same methods as for 2015.

The burden attributable to illicit drug use in 2003 was estimated using the NDSHS 2004 and The Kirby Institute data for 2003, using the same methods as for 2015.

## Intimate partner violence

The burden of intimate partner violence was estimated in women aged 15 and over.

The burden was estimated as described further in the report *Examination of the burden of disease of intimate partner violence against women in 2011: Final report* (Ayre et al. 2016).

This risk factor was estimated in women only as the evidence in the literature used to inform the linked diseases and relative risks was only available for women (Ayre et al. 2016; GBD 2016 Risk Factor Collaborators 2017).

### Population attributable fraction estimated with direct evidence

Homicide and violence linked to intimate partner violence was estimated using direct evidence from the National Homicide Monitoring Program for fatal burden, which estimated that 54% of homicides in females were due to an intimate partner in 2012–14 (Bryant & Bricknell 2017).

Non-fatal burden from homicide and violence due to an intimate partner was estimated directly from the NHMD, using the proportion of hospitalisations (with any principal diagnosis) with an external cause related to assault by an intimate partner (ICD-10-AM codes X85–Y09 with a fifth digit of 0).

### Population attributable fraction estimated with comparative risk assessment

#### Exposure estimates

Exposure to intimate partner violence data were sourced from the PSS 2016 (ABS 2017i). It was based on survey respondents aged 18 and over who self-reported intimate partner violence from a cohabiting partner from the age of 15 onwards.

Multiple definitions of exposure to intimate partner violence exist to reflect the complexity of violence against women. This study has been able to include emotional, physical and sexual intimate partner violence by a cohabiting current or previous intimate partner. It was not possible to estimate violence by a non-cohabiting current or previous intimate partner. This is because the PSS 2016 did not include an estimate of emotional abuse by non-cohabiting partners (ABS 2017i).

## Estimates by socioeconomic group

Exposure estimates by socioeconomic group were based directly from the same data source as the national exposure estimates.

### 2011 and 2003 estimates

The burden due to intimate partner violence in 2011 was estimated using data from the PSS 2012 (ABS 2013b), NHMD hospitalisations in 2011 and the National Homicide Monitoring Program 2010–2012 (Bryant & Cussen 2015).

The burden due to intimate partner violence in 2003 was estimated using data from the PSS 2005 (ABS 2006), NHMD hospitalisations in 2003 and the *National Homicide Monitoring Program annual report 2003–04* (Mouzos 2005). Prevalence of emotional abuse in 2003 was based on the PSS 2012, assuming no trends, as it was not estimated in the PSS 2005.

## Unsafe sex

This risk factor was estimated in people aged 15 and over using direct evidence. It was not possible to estimate the burden due to this risk factor by socioeconomic group as the data were not available.

### Population attributable fraction estimated using direct evidence

The entire burden of cervical cancer, chlamydia, gonorrhoea, syphilis and other sexually transmitted infections was attributed to unsafe sex; therefore, a PAF of 1 was used.

PAFs were calculated directly for chronic liver disease, hepatitis B, hepatitis C, HIV/AIDS, and liver cancer from the National Notifiable Diseases Surveillance Scheme data published in the annual surveillance reports by The Kirby Institute (The Kirby Institute 2017).

#### Acute hepatitis B and C

For acute hepatitis B and hepatitis C, the direct PAFs were calculated from the proportion of people with newly acquired hepatitis B or hepatitis C infections in 2015 who were exposed to unsafe sex.

#### Chronic liver disease and liver cancer

##### *Chronic hepatitis C infection*

The annual rates of decompensated cirrhosis (chronic liver disease), hepatocellular carcinoma (liver cancer) and liver transplants due to hepatitis C between 2006 and 2015 were published in the 2016 Annual Surveillance Reports (The Kirby Institute 2016). This trend information was used to determine the rate of hepatitis C related morbidity in each reference year (2003, 2011 and 2015).

To determine the rate of hepatitis C related chronic liver disease and liver cancer due to unsafe sex, data on newly acquired hepatitis C infection in men between the years 2000 and 2013 by exposure type was used as a proxy.

The proportion of chronic liver disease and liver cancer due to unsafe sex was estimated by dividing the number of hepatitis C related morbidity cases due to unsafe sex by the total prevalence for liver cancer and chronic liver disease in each reference year.



### *Chronic hepatitis B infection*

There is little data on the proportion of people living with chronic hepatitis B due to unsafe sex; however, there is more data available on the proportion of people living with chronic hepatitis B due to unsafe injecting practices (MacLachlan et al. 2013; O'Sullivan 2004).

Therefore, an indirect method was used to estimate hepatitis C related morbidity due to unsafe sex. The proportion of chronic liver disease and liver cancer due to unsafe sex was estimated by applying an unsafe sex exposure:drug use exposure ratio to the proportion of hepatitis B related chronic outcomes due to unsafe injecting practices in each reference year. Estimates of the number of newly acquired hepatitis B infection in men between 2002 and 2011 by exposure type were used to estimate the unsafe sex exposure: drug use exposure ratio.

Since only a single direct PAF is required for chronic liver disease due to unsafe sex and another for liver cancer due to unsafe sex, the separate PAFs calculated for hepatitis C related and hepatitis B related chronic liver disease and liver cancer due to unsafe sex were summed.

### **HIV/AIDS**

For HIV/AIDS, direct PAFs were calculated from the proportion of diagnosed AIDS cases in 2015 with a relevant exposure category (including homosexual contact only, homosexual contact and injecting drug use or heterosexual contact).

### **2011 and 2003 estimates**

Methods for estimating exposure and calculating the PAFs in 2015 were used to produce 2011 and 2003 estimates. Data from the NNDSS used for 2011 estimates were published in the annual surveillance reports by The Kirby Institute (The Kirby Institute 2012, 2013). PAFs for unsafe sex were calculated from the Annual Surveillance Report (The Kirby Institute 2004).

## **Child abuse and neglect**

Child abuse and neglect included emotional, physical, sexual abuse and neglect. The burden of child abuse and neglect was estimated in people aged 5 and over. The burden for this risk factor was calculated as described in the study by Moore and others (2015). It was not possible to estimate this risk factor by socioeconomic group as this was not reported by Moore and others (2015).

Moore and others (2015) identified Australian studies from the period of 2005 to 2015 that estimated exposure to the different types of abuse in different age groups and by sex. They also identified from the literature an estimate of the proportion of cases that had multiple types of abuse. Using these data together, they estimated the proportion of the Australian population with all types of abuse and neglect, with only a single type, and with different types of abuse in combination.

Moore and others (2015) also reviewed the literature and identified linked diseases and relative risks for child abuse and neglect. Using these data and comparative risk assessment methodology, they estimated the PAF of each linked disease by age and sex for Australia.

## **Population attributable fraction**

### **Exposure estimates**

Exposure and PAFs were estimated by Moore and others (2015) by age and sex. The same PAFs were applied to each reference year of this study: 2003, 2011 and 2015.

# Overweight and obesity

## Population attributable fraction

The burden due to overweight and obesity was estimated in people aged 5 and over. The methods used for this risk factor are described in detail in the AIHW publication *Impact of overweight and obesity as a risk factor for chronic conditions: Australian Burden of Disease Study* (AIHW 2017d).

## Exposure estimates

Age- and sex-specific data were extracted in the finest possible increments from a continuous high body mass distribution for the Australian population based on measurements of height and weight from the NHS 2014–15. For children and adolescents aged 5–14, age- and sex-specific BMI cut-off levels indicating overweight and obesity were derived from the study by Cole and others (2000).

## Relative risks

The relative risks used were based on those published by the AIHW (2017a). Additional linked diseases were included, based on the GBD 2016, including atrial fibrillation and flutter, cataract, non-Hodgkin lymphoma and multiple myeloma.

For dementia and gallbladder and bile duct disease, relative risks from the GBD 2016 were used instead of relative risks from the AIHW 2017a as they were based on a more recent meta-analysis.

## Estimates by socioeconomic group

It was not possible to aggregate risk factor exposure data by socioeconomic group with acceptable RSEs; therefore, exposure was estimated based on the difference in the mean estimate in each quintile as described in Chapter 7.

## 2011 and 2003 estimates

Exposure for 2011 was estimated as described above, using data from the AHS 2011–12.

For people aged 20 and over, prevalence by BMI category, age and sex was estimated for the time-point 2003 by using the trends in the prevalence of the distribution of BMI from the 3 successive health surveys (the NHS 2007–08, the AHS 2011–12 and the NHS 2014–15) as described in AIHW 2017a.

For people aged 5 to 19, prevalence by BMI category, age and sex was estimated for the time-point 2003, using the NHS 2007–08. The estimate of prevalence of obesity in people aged 5 to 19 decreased slightly from the NHS 2007–08 to the AHS 2011–12, and from the AHS 2011–12 to the NHS 2014–15, but these differences were not statistically significant. Due to this, the trend from the 3 successive health surveys (the NHS 2007–08, the AHS 2011–12 and the NHS 2014–15) were not considered accurate for this age group when compared with the 1995 National Nutrition Survey estimates.

## High blood pressure

The burden attributable to high blood pressure was estimated in people aged 25 and over.

### Population attributable fraction

#### Exposure estimates

Age- and sex-specific data were extracted in the finest possible increments from a continuous systolic blood pressure distribution for the Australian population based on blood pressure measurements from the NHS 2014–15 (ABS 2017h).

#### Estimates

Exposure to high blood pressure by socioeconomic group was based on the same data source as for the national exposure estimates. It was not possible to aggregate risk factor exposure data by socioeconomic group to generate acceptable RSEs; therefore, exposure was estimated based on the difference in the mean estimate in each quintile as described in Chapter 7.

#### 2011 and 2003 estimates

Data were sourced directly from the AHS 2011–12, using the same method as for 2015.

For 2003 estimates, the exposure to high blood pressure in 2003 was calculated by comparing the mean exposure from the Australian Diabetes, Obesity and Lifestyle Study (AusDiab) 1999–2000 and the mean exposure from the 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 blood pressure in 2003.

## High blood plasma glucose (including diabetes)

The burden attributable to high blood plasma glucose was estimated in people of all ages. The risk factor includes estimates of the burden due to intermediate hyperglycaemia and diabetes. Burden due to this risk factor was not estimated the 2003 reference year as there were no data on trends of blood plasma glucose between 2003 and 2011.

### Population attributable fraction using direct evidence

All types of diabetes were entirely attributable to high blood plasma glucose (PAF of 1) as high blood plasma glucose is a diagnostic criteria for all types of diabetes.

#### *Chronic kidney disease due to high blood plasma glucose*

The method for attributing the amount of chronic kidney disease due to diabetes is based on the GBD 2016 and involves a 2-step approach:

1. The proportion of the GBD cause 'chronic kidney disease due to diabetes' of the total GBD cause 'chronic kidney disease' in the GBD 2016 (53%) was used to estimate the direct PAF of chronic kidney disease due to high blood plasma glucose.
2. Exposure to high blood plasma glucose is linked to the remaining 47% of chronic kidney disease not attributed in step 1 as described later in this section. Part of this remaining proportion (the GBD causes 'chronic kidney disease due to hypertension, glomerulonephritis or other and unspecified causes') is attributed to high blood plasma glucose, using the comparative risk assessment method.

## Population attributable fraction using comparative risk assessment

### Exposure estimates

Exposure to high plasma glucose included 2 parts: the population distribution of blood plasma glucose levels (continuous risk model) and the proportion of the population with diabetes (categorical risk model). Each of these exposures was linked to different diseases (see Appendix Table F2).

To estimate and report the burden attributable by intermediate hyperglycaemia and diabetes, the continuous distribution of high blood plasma glucose was divided into the following categories:

- exposure to 4.9 to 6.9 mmol/l high plasma glucose was attributable to intermediate hyperglycaemia. This range was defined by the GBD TMRED of 4.9 mmol/L and expert advice for the 6.9 mmol/l cut-off
- burden due to blood plasma glucose of 7 mmol/l or more was attributable to diabetes in addition to the attributable burden estimated from exposure to diabetes.

#### *High blood plasma glucose*

Age- and sex-specific data were extracted in the finest possible increments from a continuous fasting blood plasma glucose distribution for the Australian population from the AHS 2011–12. As no data were available to inform trends, this estimate was also applied in 2015.

#### *Diabetes*

The prevalence of diabetes was based on the prevalence of type 1, type 2 and other diabetes in 2015. All types of diabetes are included because people exposed to all types of diabetes are at risk of the disease outcomes identified, and the risk factor is modifiable.

### Estimates by socioeconomic group

Exposure estimates by socioeconomic group were calculated directly from the same data source as for the national exposure estimates.

For high blood plasma glucose, it was not possible to aggregate risk factor exposure data by socioeconomic group with acceptable RSEs; therefore, exposure was estimated based on the difference in the mean estimate in each quintile as described in Chapter 7.

### 2011 estimates

The prevalence of high blood plasma glucose was estimated using measured data from the AHS 2011–12. The prevalence of diabetes for 2011 was from the ABDS 2015 study.

It was not possible to estimate this risk factor in 2003 because there were no data available to estimate the trend in high blood plasma glucose.

# High cholesterol

The burden attributable to high cholesterol was estimated in people aged 25 and over.

## Population attributable fraction

### Exposure estimates

Age- and sex-specific data were extracted in the finest possible increments from a continuous measured total cholesterol distribution for the Australian population from the AHS 2011–12.

The exposure to high cholesterol in 2015 was calculated by comparing the mean exposure from the AusDiab 1999–2000 and the mean exposure from the 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 that would be expected between the years 2011 to 2015. The adjusted unit record data were used to estimate the distribution of exposure to high cholesterol in 2015.

### Estimates by socioeconomic group

Exposure to high cholesterol was estimated using data from the AHS 2011–12, modelled to 2015, and the difference in the mean estimate in each quintile as described in Chapter 7.

### 2011 and 2003 estimates

The prevalence of total cholesterol for 2011 was estimated using data from the AHS 2011–12.

The same trend described here for 2015 was used to estimate prevalence in total cholesterol in 2003.

# Impaired kidney function

The burden attributable to impaired kidney function was estimated in people aged 25 and over.

## Population attributable fraction

### Exposure estimates

#### *Chronic kidney disease stages 1–3*

Age- and sex-specific data were extracted in the finest possible increments from the estimate of stages 1, 2 and 3 chronic kidney disease for the Australian population from the AHS 2011–12.

To estimate prevalence in the year 2015, the AIHW analysis of trends in stages 3–5 chronic kidney disease prevalence from the 1999–2000 AusDiab compared with the AHS 2011–12 in the broad age groups was used (AIHW 2018b). The age and sex distribution for stage 3 chronic kidney disease were further refined using the age and sex of people who were hospitalised for stage 3 chronic kidney disease (N18.3) in 2015.

#### *Chronic kidney disease stages 4–5*

The prevalence of stage 4 and 5 (end-stage) chronic kidney disease was estimated as the prevalence for the relevant sequelae (stage 4 chronic kidney disease, end-stage chronic kidney disease treated with dialysis or transplant) for the cause chronic kidney disease in the ABDS 2015. The methods for these sequelae are described for the cause chronic kidney disease.

## **Estimates by socioeconomic group**

Exposure to stages 1–3 chronic kidney disease by socioeconomic group was estimated using data from the AHS 2011–12, modelled to 2015 and grouped into broad age- and sex-groups.

Exposure of stages 4–5 chronic kidney disease by socioeconomic group was sourced as described for the cause chronic kidney disease.

### **2011 and 2003 estimates**

#### *Chronic kidney disease stages 1–3*

The prevalence of stages 1, 2 and 3 chronic kidney disease for 2011 was estimated using data from the AHS 2011–12. To estimate prevalence in the year 2003, the AIHW analysis of trends in stages 3–5 chronic kidney disease prevalence from the 1999–2000 AusDiab compared with the AHS 2011–12 in the broad age groups was used (AIHW 2018b).

#### *Chronic kidney disease stages 4–5*

The prevalence of stages 4–5 chronic kidney disease in 2011 and 2003 was sourced as described for the cause chronic kidney disease for the ABDS 2015.

## **Iron deficiency**

These PAFs were estimated using direct evidence in women aged 15 to 45. Iron deficiency anaemia is the only disease linked to iron deficiency and was 100% attributable to this risk factor (PAF of 1). The method was the same in all 3 years. An estimate by socioeconomic group was not included to match other risk factors where exposure does not change by socioeconomic group.

## **Low bone mineral density**

The burden attributable to low bone mineral density was measured in people aged 40 and over. It was not possible to estimate exposure by socioeconomic group as no data were available.

## **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 and are therefore 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 Sánchez-Riera and others (2014).

### **2011 and 2003 estimates**

Methods for estimating exposure and calculating the PAFs for the 2011 reference year were the same as those used for 2015.

Methods for estimating exposure and calculating the PAFs in 2015 and 2011 were used to produce 2003 estimates. Data from the Geelong Osteoporosis Study were applied to the 2003 population.

## **Occupational exposures and hazards**

Occupational exposures and hazards captured the impact of exposure 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, particulate matter, and gases and fumes in the workplace.

### **Population attributable fraction from direct evidence**

The PAFs for injuries 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.

All pneumoconiosis was attributable to occupational exposure as informed by expert advice (T Driscoll 2015, pers. comm., 24 June 2016).

For injuries, direct evidence was sourced from Safe Work Australia, including data on the number of deaths occurring at work (Safe Work Australia 2016) and the number of workers' compensation injury claims annually (Safe Work Australia 2018). Counts of deaths and injuries, with some disaggregation by age, sex and nature or external cause of injury, were used to directly calculate PAFs.

The PAFs for fatal burden were estimated by the number of deaths occurring at work compared with the total number of deaths due to injuries in the broader population.

The data for non-fatal burden are limited in that compensation claims will capture only injuries that require more than 1 week away from work and are fairly severe. They will also not include people who are self-employed. These PAFs were estimated for people aged 15 and over.

The PAFs for non-fatal burden were estimated by the number of injuries reported at work in 2015 from Safe Work Australia (2018) divided by the incidence of admitted and non-admitted hospitalisations and emergency department presentations in the NHMD in 2015.

### **Population attributable fraction by comparative risk assessment**

#### **Exposure estimates**

To estimate the number of people working in Australia—the economically active population—by age, sex and industry or occupation, was estimated from the Labour Force Survey, (ABS 2018c).

#### **Industry**

Exposure to working in type of industry was linked to various cancers, hearing loss and COPD (see Appendix Table F2). This is because working in these industries is known to expose a proportion of the workforce to carcinogens, noise, particulate matter, gases and fumes as estimated by the Carcinogen Exposure (CAREX) research project (Kauppinen et al. 2000).

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 2016 Census of Population and Housing.

A severity distribution from the GBD 2010 was applied to obtain the proportion of people working in these industries exposed to high and low levels of noise, and to high and low levels of particulate matter, gases and fumes. 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 adjusts 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 (CAREX) research project produces estimates of the proportion of workers in each industry who will be exposed to specific carcinogens (Kauppinen et al. 2000). These proportions, which are based on data from the European Union and Canada, are then applied for each of the industries described earlier. The PAFs for carcinogens were calculated for people aged over 15.

### **Occupation**

Exposure to types of occupations was linked to asthma and low back pain (see Appendix table F2). This is because working in these occupations is known to expose a proportion of the workforce to asthmagens and ergonomic stressors.

The number of working people was apportioned by 8 broad occupational groups (professional, technical and related workers; administrative and managerial workers; clerical and related workers; sales workers; service workers; agricultural, animal husbandry and forestry workers; fishermen and hunters; production and related workers; transport equipment operators and labourers) based on the 2016 Census of Population and Housing (ABS 2017d).

Exposure to working in these occupations was used to estimate the PAFs in people aged 15–64 and no severity distribution was applied.

### **Estimates by socioeconomic group**

The estimate of the economically active population by socioeconomic group was adjusted, based on the proportion of the population in each quintile not in the labour force. The proportion in each industry and occupation group was estimated from the same data source as for the national exposure estimates. National estimates for occupational injury were used for each quintile as it was not possible to estimate exposure by socioeconomic group.

### **2011 and 2003 estimates**

Methods for estimating exposure and calculating the PAFs in 2015 were followed for 2011 estimates. The working population was estimated from the Labour Force Survey (ABS 2011) and disaggregated by occupation and industry using the 2011 Census of Population and Housing (ABS 2017d).

Methods for estimating exposure and calculating the PAFs in 2015 were followed to produce 2003 estimates. The working population was estimated from the Labour Force Survey 2003 (ABS 2003) and disaggregated by occupation and industry using the 2006 Census of Population and Housing (ABS 2017d).



## High sun exposure

The burden attributable to sun exposure was estimated in people of all ages using direct evidence. The direct PAFs used here are a proportion of current burden due to past and current sun exposure in the population. An estimate by socioeconomic group was not included to match other risk factors where exposure does not change by socioeconomic group.

### Population attributable fractions using direct evidence

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 PAFs appropriate for Australia were 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 exposure in Australia (F Xiang 2015, pers. comm., 11 November 2015).

### 2011 and 2003 estimates

The same PAFs were used in 2011 and 2003 as they were not specific to 2015 but based on latitude.

## Air pollution

The fatal burden attributable to air pollution was measured by concentration of particulate matter 2.5µg/m<sup>3</sup> (PM2.5) in Australia in people of all ages. It was not possible to estimate exposure to this risk factor in 2011 or 2003 because comparable exposure data were not available. An estimate by socioeconomic group was not included to match other risk factors where exposure does not change by socioeconomic group.

### Population attributable fraction

#### Exposure estimates

PM2.5 is particles suspended in the air with a diameter in a specified size range, 0–2.5 microns. Population exposure to air pollution was estimated from ground-based monitoring stations in 58 locations around Australia that measure 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 7.1). The number of people covered was calculated using population data by state/territory and remoteness area.

Daily maximum air pollution data from each station in 2015 was provided by personal communication (G Pereira 2018, pers. comm., 8 August 2018).

The majority of monitoring stations were located in *Major cities* as the stations are concentrated around population centres. Some stations were located in *Inner regional* areas and *Outer regional* areas. No stations were located in *Remote* or *Very remote* locations. *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 indicate the level of pollution to which people in the region immediately surrounding the station are exposed. 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 larger population. There is also likely to be a substantial amount of variation between sites in

the amount of time that people generally spend outside, being exposed to air pollution; and, due to differences in planning history and population density, the effects of industrial pollution may be greater in some locations than in 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 ways to measure PM<sub>2.5</sub> through satellite imaging rely on a large amount of spatial modelling and could not be done in time for this study. Satellite modelling has the same issue, however, in measuring ambient air pollution levels rather than actual exposure to air pollution, but has the advantage that estimates would be based on measurements from larger areas of Australia and be calibrated by ground monitoring stations.

**Table 7.1: Name and remoteness category of stations that collected data on PM<sub>2.5</sub> in 2015**

State/territory	Station	Remoteness category
NSW	Beresfield, Camden, Carrington, Chullora, Earlwood, Liverpool, Mayfield, Newcastle, Richmond, Wallsend, Wollongong, Woollooware, Wyong, Prospect, Stockton, Kembla grange, Rozelle, Stockton, Albion park south, Prospect	Major cities
	Camberwell, Muswellbrook, Singleton, Wagga Wagga north	Inner regional
Vic	Alphington Footscray	Major cities
	Morwell east, Morwell south, Traralgon,	Inner regional
QLD	Cannon hill, Lytton, Rocklea, South Brisbane, Springwood, Woolloongabba, Wynnum, Wynnum west	Major cities
	Boyne island, Clinton, Jondaryan, South Gladstone	Inner regional
	Boat creek, Targinie	Outer regional
WA	Caversham, Duncraig, Quinn rocks, South lakes	Major cities
	Bunbury, Busselton	Inner regional
SA	Netley, North haven	Major cities
Tas	Devonport, North Hobart, Ti-tree	Inner regional
ACT	Monash, Florey	Major cities
NT	Palmerston, Winnellie	Outer regional

## Dietary risk factors

The burden of most of the dietary risk factors was estimated in people aged 25 and over except for the burden of a diet high in sugar sweetened beverages, which was estimated in children aged 5 and over.

It should be noted that the methods, including the TMREDS, used in the ABDS 2015 to calculate attributable burden due to dietary risk factors do not align with current Australian dietary guidelines as they are used to calculate disease burden (see Appendix Table F2). For information on recommended food choices, see the Australian Dietary Guidelines (NHMRC 2015).

A review of the list of dietary risk factors and the methods used in ABDS 2011 was undertaken as part of this study. The risk factors included were based on the AIHW review of evidence from the GBD 2016 (which included 14 dietary risk factors) and other systematic reviews from authoritative sources that have also assessed the impact of dietary risk factors on health. These other sources included the NHMRC dietary guidelines evidence paper, which provided scientific evidence for healthier Australian diets (NHMRC 2011). Evidence from the continuous

update project by the World Cancer Research Fund International (WCRFI) was used for cancer outcomes (WCRFI 2017). Information on carbohydrates and health (SACN 2015) and the evidence used to create the World Health Organization guideline for sugar intake for adults and children by Moynihan and Kelly (2014) were reviewed while considering the inclusion of diet high in added sugar.

## **Dietary risks included**

The risk factors included by the GBD that were not in this study include diet low in fibre and diet low in calcium. These were mediated entirely through diet low in whole grains and diet low in milk, respectively, which were included in this study. To avoid double-counting, diet low in fibre and diet low in calcium were excluded from this study.

The risk factor diet high in trans-fat was excluded from the study as consumption is low in Australia, on average.

The risk factor diet low in omega-3 seafood fatty acids was replaced by diet low in fish and seafood to align this risk factor with the other whole food risk factors, included using evidence from Zheng and others (2012).

We did identify evidence to expand the risk factor diet low in milk to include cheese and yoghurt, which were also both linked to bowel cancer (WCRFI 2017). We were unable to identify an appropriate method to estimate this risk factor due to the different gram amounts required from the different dairy sources in the time frames for this study.

We did not include the risk factor diet high in added sugar due to insufficient evidence and inputs in the literature; this risk factor was also not included in the GBD 2016. Moynihan and Kelly (2014) did a systematic review on added sugar for the World Health Organization guideline for sugar intake for adults and children (WHO 2015). It identified dental caries as a linked disease with a relative risk of 7.15 (2.82–18.14) for high sugar intake (when sugar intake is greater than 10% of daily caloric energy uptake compared with when it is less than 10%). However, a subsequent systematic review and meta-analysis by the Scientific Advisory Committee on Nutrition for Public Health England was quite critical of this study and was concerned about bias due to the design of some of the studies included (SACN 2015). Excluding these studies, Scientific Advisory Committee on Nutrition for Public Health England did not identify sufficient evidence to link diet high in added sugar with dental caries. Therefore, diet high in added sugar was not included in the ABDS 2015.

Other linked diseases were considered for diet low in vegetables, as well as those estimated by the GBD 2016. The WCRFI did have probable evidence that a diet low in vegetables was linked to a number of cancers. However, there was insufficient data to estimate a relative risk; therefore, these linked diseases could not be included in the study. Sufficient evidence was reported by the WCRFI for processed meat linked to stomach cancer, which was not included in the GBD. This linked disease and the relative risk for it were included in this study. Diet low in whole grains was also linked to bowel cancer, but the available data were not suitable to allow estimation for this study. Note that diet low in whole grains includes the intake of high fibre cereal varieties.

The name of the risk factor diet low in saturated fats estimated in the ABDS 2011 was changed in this study to diet low in poly-unsaturated fats to reflect the methods used to estimate the burden due to this risk factor. In the ABDS 2011, exposure to polyunsaturated fats was considered a proxy for saturated fats, based on the methods from the GBD 2013. The name of this risk factor was changed for this study to more accurately reflect what was measured, as advised by experts and the GBD 2016.

## **Population attributable fractions estimated using comparative risk assessment**

The risk factors estimated using the comparative risk assessment were diet low in fruit, vegetables, wholegrains, milk, fish and seafood, polyunsaturated fats, legumes, nut and seeds; and diet high in red meat and processed meat.

### **Exposure estimate**

The National Nutrition and Physical Activity Survey (NNPAS) part of the AHS 2011–12 collected food intake data (through a 24-hour recall) from participants for 2 days. However, at the time of the ABDS 2011, it was possible to use only the day 1 data because the modelling required to estimate the usual intake (modelled distribution based on 2 days of data) was not available in time for the study

For the ABDS 2015, the amount of each food was adjusted to the usual intake, taking into account reported intake on day 1 and day 2 and using the method developed by the National Cancer Institute. This method was used to estimate the distribution of intake of foods as described in Appendix Table F2 in the Australian population.

To estimate consumption in 2015, unit record level data from the AHS 2011–12 was adjusted by the percentage change from 2011 to 2015, based on the mean exposure from the National Nutrition Survey 1995 component of NHS 1995 and the mean exposure from the AHS 2011–12 by age. The mean exposure in each year was estimated by mean number of serves per 10,000 kJ, as published by the ABS (2017b).

It is important to note that there is significant under-reporting of dietary intake in the AHS 2011–12 (as with all representative dietary surveys) (ABS 2014b). 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 (that is, '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 2015, except for diet high in sodium.

### **Estimates by socioeconomic group**

Exposure to dietary risks was estimated from the AHS 2011–12, modelled to 2015, and the difference in the mean estimate in each socioeconomic group quintile as described in Chapter 7.

### **2011 and 2003 estimates**

The analysis for the year 2011 was based on the methods using the AHS 2011–12 data as described earlier in this section on dietary risks.

The exposure to these risk factors over time was calculated by comparing the mean exposure from the NHS 1995 and the mean exposure from the AHS 2011–12 by age. Unit record level data from the AHS 2011–12 were adjusted by the percentage change from 2011 to 2003 in these data sources to estimate the distribution of dietary intake in 2003. This method is the same as was used for the 2015 study.

## **Dietary risks that are mediated through other risk factors**

Diet high in sodium and diet high in sugar sweetened beverages were measured by the amount they mediated blood pressure and body mass index, respectively. The methods for these risk factors use comparative risk assessment and are based on the GBD 2016.

### **Diet high in sodium**

The attributable burden for diet high in sodium was calculated from a model of the impact of current sodium consumption on blood pressure levels in Australia. The model estimates the blood pressure distribution of Australians if no sodium above the TMRED was consumed.

#### **Population attributable fraction**

This was calculated in 4 steps for 2011:

1. The consumption of sodium self-reported on day 1 in the AHS 2011–12 study was adjusted due to under-reporting. The data from dietary recall studies are known to include an under-reporting of the consumption of discretionary foods that are high in sodium (ABS 2014b). An adjustment factor was calculated by comparing the mean amount of sodium from 24 hour urinary samples for Australia estimated by the Global Burden of Diseases Nutrition and Chronic Disease Expert Group (Powles et al. 2013) with the mean amount by dietary recall in the AHS 2011–12.
2. The prevalence of blood pressure due to high sodium intake was estimated using the effect of sodium consumption on blood pressure. The effect was estimated by an adjustment factor, which varies by age, the presence or absence of hypertension, and race (non-African descent), sourced from the GBD 2016. These adjustment factors were used to calculate the distribution of systolic blood pressure that would be expected from reducing sodium consumption to the TMRED compared with current levels of sodium consumption. Blood pressure was based on measured estimates in the AHS 2011–12.
3. These 2 estimates of the distribution of systolic blood pressure (with and without sodium consumption above the TMRED) were used with the methods for the high blood pressure risk factor (including the TMRED, all linked diseases and relative risks) to estimate the PAFs for both of these scenarios.
4. Finally, the PAFs for diet high in sodium was estimated using the difference between the PAFs from the 2 scenarios by age and sex.

To estimate the impact of sodium in 2015, the distribution of blood pressure prevalence from the NHS 2014–15 was estimated. To calculate the distribution without the consumption of sodium above the TMRED, the blood pressure of each survey respondent was adjusted by the average adjustment per weighted count in each age, sex and blood pressure category calculated in 2011–12. The PAF for diet high in sodium was then calculated as described here for 2011.

### **Estimates by socioeconomic group**

The average adjustment factors estimated for sodium intake and blood pressure for 2011 by socioeconomic quintile were applied to the distribution of blood pressure prevalence in the NHS 2014–15.

### **2011 and 2003 estimates**

The analysis for the year 2011 was based on the methods using the AHS 2011–12 data as described earlier.

The average adjustment factors estimated for sodium intake and blood pressure for 2011 were applied to the distribution of blood pressure prevalence in the NHS 2004–05 to calculate 2003 estimates.

### **Diet high in sugar sweetened beverages**

The attributable burden due to diet high in sugar sweetened beverages was calculated from a model of the impact of current sugar sweetened beverage consumption on body mass index levels in Australia. The model estimates the body mass index distribution of Australians if no sugar sweetened beverages were consumed (TMRED for diet high in sugar sweetened beverages).

### **Population attributable fraction**

This was calculated in 4 steps for 2011:

1. The consumption of sugar sweetened beverages was estimated using the self-report of the relevant food codes on day 1. It was not possible to adjust self-reports of sugar sweetened beverages to account for under-reporting in time for this study. The ABS is working to estimate apparent consumption of these products in Australia. Updated estimates from the NHS 2017–18 of self-reported sugar sweetened beverage consumption in Australia were also not available in time for this study.
2. The prevalence of body mass index due to high sugar sweetened beverage intake was estimated using the effect of sugar sweetened beverage consumption (per 226.8mL) on body mass index. The effect was estimated using an adjustment factor, which varies by age and a BMI of more or less than 25, sourced from the GBD 2016 for adults and for people aged 18 and under (Malik et al. 2013). These adjustment factors were used to calculate the distribution of body mass index that would be expected from reducing sugar sweetened beverage consumption to the TMRED compared with current levels of sugar sweetened beverage consumption. Body mass index was based on measured estimates in the AHS 2011–12.
3. These two estimates of the distribution of body mass index (with and without sugar sweetened beverage consumption above the TMRED) were used with the methods for the overweight and obesity risk factor (including the TMRED, all linked diseases and relative risks) to estimate the PAFs for both of these scenarios.
4. Finally, the PAFs for diet high in sugar sweetened beverages was estimated using the difference between the PAFs from the two scenarios by age and sex.

To calculate the body mass index distribution with and without the consumption of sugar sweetened beverages in 2015, the average adjustment per weighted count in each age, sex and body mass index category was calculated using data from the AHS 2011–12. This adjustment was applied to body mass index estimates in the NHS 2014–15.

### **Estimates by socioeconomic group**

The average adjustment factors estimated for the body mass index due to high sugar sweetened beverage for 2011 by socioeconomic quintile were applied to the distribution of body mass index prevalence in the NHS 2014–15.

### **2011 and 2003 estimates**

The analysis for the year 2011 was based on the methods using the AHS 2011–12 data as described above. The average adjustment factors estimated for the impact of sugar sweetened beverages were applied to the BMI measured in the NHS 2007–08.

## 9 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

As part of the ABDS 2011, a quality framework was developed to report on estimates produced as part of the study. 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.

The AIHW considered the two most commonly used measures of reliability—uncertainty analysis and scenario testing (see Appendix G for more details). However, 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 2015 quality index

In light of the assessments of measuring uncertainty described previously, the Expert Advisory Group for the ABDS 2011 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, the 2-dimensional index developed for the ABDS 2011 was used for the ABDS 2015 burden estimates. This 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. The higher the index the more relevant and accurate the estimate was.

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.

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 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 the GBD 2010 and 2013) was appropriate for use in the Australian context
- 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 the 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 risk factors:
  - the risk–outcome pairs, minimum exposure levels and effect sizes (used in the risk factor analysis) defined by the GBD 2016 and other studies were appropriate for:
    - the particular risk factor
    - the Australian 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 quality guidelines endorsed by the study's Expert Advisory Group and Indigenous Reference Group to ensure that the highest quality data available were included in the study (see Appendix A). However, 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 2015 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 sub-national, 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 hospitalisations 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 G2.

## 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 2015 national 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 G3) and risk factors (Appendix table G4) 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).

### **Sub-national, 2011 and 2003 estimates**

The data and methods used for 2015 estimates underpinned the sub-national, 2011 and 2003 estimates, so the quality of these estimates must be considered together with the broad sub-national, 2011 and 2003 methods described in Chapter 2 ('Overarching methods and choices'), and the specific details described in Chapter 5 ('Disease specific methods') and Chapter 8 ('Risk factor specific 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 G3 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 D or E, which are considered to be somewhat relevant/accurate or of questionable dependability, respectively.

### **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 G4. For each risk factor, it was only possible to rate the quality of the data used to estimate the direct PAFs or the exposure data used to calculate the PAFs. Many other inputs (such as relative risks) were included in these calculations, as described in chapters 7 and 8, 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 G4 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 8.

# Appendix A: Additional information and tables for Chapter 2

## Assessment of data sources

National 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, and be timely, accurate, reliable and credible.

Published and unpublished data sources were assessed according to the guidelines 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 guidelines 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: Guidelines 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. 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): Guidelines 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 (sub-national)
3. a sub or super-regional population (includes New Zealand, United Kingdom, United States of America and Canada)
4. another population.

#### **Currency**

The data source should ideally have been collected within 5 years of each of the ABDS reference years.

#### **Accuracy**

The data source should ideally have more than 90% case ascertainment or coverage of the population of interest, and a 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): Guidelines 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: for example, against 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.

## Additional tables

Table A1: Assessment matrix for data sources to be used in the ABDS 2015

Data source									
Data provider									
Level of disaggregation									
Rating	Comparability	Relevance/representativeness	Currency	Accuracy			Validation	Credibility	Accessibility/ timeliness
				Ascertainment/ coverage	Error (sampling/ non-sampling)	Measurement error			
High	Consistent	National	2011 or later	More than 90%	Less than 25% RSE	Clinically reported or measured	Validated	Published and Peer reviewed	Currently available
Medium	Comparable	Sub-national	2004–2010	60%–90%	25%–50% RSE	Self-reported		Published but not peer reviewed	Expected to be available in time for analysis
		Sub or super-regional (such as New Zealand, United States, Canada)					Not published but peer reviewed		
Low	Inconsistent	Other	Before 2004	Less than 60%	More than 50% RSE	Not known	Not validated	Not published nor peer reviewed	Unlikely to be available in time for analysis.

**Table A2: ABDS 2015 disease list by ICD-10 code**

<b>ABDS 2015 disease/injury</b>	<b>ICD-10 codes<sup>(a)</sup></b>
<b>Blood &amp; metabolic disorders</b>	
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
<b>Cancer &amp; other neoplasms</b>	
Lip and oral cavity cancer <sup>(b)</sup>	C00–C08
Nasopharyngeal cancer	C11
Other oral cavity and pharynx cancers	C09–C10, C12–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

*(continued)*



**Table A2 (continued): ABDS 2015 disease list by ICD-10 code**

ABDS 2015 disease/injury	ICD-10 codes <sup>(a)</sup>
<b>Cancer &amp; other neoplasms (continued)</b>	
Kidney cancer	C64
Brain and central nervous system cancer	C70–C72
Thyroid cancer	C73
Non-Hodgkin lymphoma	C82–C86
Hodgkin lymphoma	C81
Acute myeloid leukaemia (AML)	C92.0, C92.3–C92.6, C92.8, C93.0, C94.0, C94.2, C94.4–C94.5
Chronic myeloid leukaemia (CML)	C92.1
Acute lymphoblastic leukaemia (ALL)	C91.0
Chronic lymphocytic leukaemia (CLL)	C91.1
Other leukaemias	C91.2–C91.9, C92.2, C92.7, C92.9, C93.1–C93.9, C94.1, C94.3, C94.6–C94.7, C95
Myeloma	C90
Other lymphohaematopoietic (blood) cancers	C88, C96, D45, D46, D47.1, D47.3–D47.5
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, C30–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–D38, D41, D44, D47.0, D47.2, D47.4–D47.8, D48
<b>Cardiovascular diseases</b>	
Coronary heart disease	I20–I25
Stroke	I60–I69
Rheumatic heart disease (including acute rheumatic fever)	I00–I06, I08.0, I08.1, I08.3, I09
Non-rheumatic valvular disease	I07, I08.2, I08.8, I08.9, 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, I10, I13, I15, I26–I28, I44–I47, I49, I50–I52, I70.9, I77–I84, I86–I89, I95, I97–I99

*(continued)*

**Table A2 (continued): ABDS 2015 disease list by ICD-10 code**

<b>ABDS 2015 disease/injury</b>	<b>ICD-10 codes<sup>(a)</sup></b>
<b>Endocrine disorders</b>	
Type 1 diabetes mellitus	E10.0–E10.1, E10.3–E10.9, O24.0
Type 2 diabetes mellitus	E11.0–E11.1, E11.3–E11.9, O24.1
Other diabetes mellitus	E12.0–E12.1, E12.3–E12.9, E13.0–E13.1, E13.3–E13.9, O24.2
Other endocrine disorders	E03–E07, E15, E16, E20–E27, E29–E32, E34, E35, E89
<b>Gastrointestinal disorders</b>	
Gastroduodenal disorders	K22.1, K25–K29
Appendicitis	K35–K37
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 <sup>(c)</sup>	. .
Other gastrointestinal diseases	K22.0, K22.2–K22.9, K23, K30, K31, K38, K58–K64, K67, K77, K87, K90, K91, K93
<b>Hearing &amp; vision disorders</b>	
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
<b>Infant &amp; congenital conditions</b>	
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

*(continued)*

**Table A2 (continued): ABDS 2015 disease list by ICD-10 code**

<b>ABDS 2015 disease/injury</b>	<b>ICD-10 codes<sup>(a)</sup></b>
<b>Infant &amp; congenital conditions (continued)</b>	
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–Q45
Urogenital malformations	Q50–Q56, Q60, 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
<b>Infectious diseases</b>	
HIV/AIDS	B20–B24, O98.7
Tuberculosis	A15–A19, B90, N33.0, N74.0, N74.1, O98.0, P37.0
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, N74.3, O98.2
Sexually transmitted chlamydial infections	A55–A56, N74.4
Other sexually transmitted infections	A57–A64, O98.3
Urinary tract infections	N30, N34, N39.0
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

*(continued)*

**Table A2 (continued): ABDS 2015 disease list by ICD-10 code**

ABDS 2015 disease/injury	ICD-10 codes <sup>(a)</sup>
<b>Infectious diseases (continued)</b>	
Pertussis	A37
Tetanus	A33–A35
Measles	B05
Mumps	B26
Rubella	B06, P35.0
Varicella	B01
Herpes zoster	B02
<i>Haemophilus influenzae</i> type-B	G00.0
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, B27–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
<b>Injuries (external cause)</b>	
Road traffic injuries: motorcyclists	V20.3–V20.5, V20.9, V21.3–V21.5, V21.9, V22.3–V22.5, V22.9, V23.3–V23.5, V23.9, V24.3–V24.5, V24.9, V25.3–V25.5, V25.9, V26.3–V26.5, V26.9, V27.3–V27.5, V27.9, V28.3–V28.5, V28.9, V29.4–V29.6, V29.8–V29.9
Road traffic injuries: motor vehicle occupants	V30.4–V30.7, V30.9, V31.4–V31.7, V31.9, V32.4–V32.7, V32.9, V33.4–V33.7, V33.9, V34.4–V34.7, V34.9, V35.4–V35.7, V35.9, V36.4–V36.7, V36.9, V37.4–V37.7, V37.9, V38.4–V38.7, V38.9, V39.4–V39.6, V39.8–V39.9, V40.4–V40.7, V40.9, V41.4–V41.7, V41.9, V42.4–V42.7, V42.9, V43.4–V43.7, V43.9, V44.4–V44.7, V44.9, V45.4–V45.7, V45.9, V46.4–V46.7, V46.9, V47.4–V47.7, V47.9, V48.4–V48.7, V48.9, V49.4–V49.6, V49.8–V49.9, V50.4–V50.7, V50.9, V51.4–V51.7, V51.9, V52.4–V52.7, V52.9, V53.4–V53.7, V53.9, V54.4–V54.7, V54.9, V55.4–V55.7, V55.9, V56.4–V56.7, V56.9, V57.4–V57.7, V57.9, V58.4–V58.7, V58.9, V59.4–V59.6, V59.8–V59.9, V60.4–V60.7, V60.9, V61.4–V61.7, V61.9, V62.4–V62.7, V62.9, V63.4–V63.7, V63.9, V64.4–V64.7, V64.9, V65.4–V65.7, V65.9, V66.4–V66.7, V66.9, V67.4–V67.7, V67.9, V68.4–V68.7, V68.9, V69.4–V69.6, V69.8–V69.9, V70.4–V70.7, V70.9, V71.4–V71.7, V71.9, V72.4–V72.7, V72.9, V73.4–V73.7, V73.9, V74.4–V74.7, V74.9, V75.4–V75.7, V75.9, V76.4–V76.7, V76.9, V77.4–V77.7, V77.9, V78.4–V78.7, V78.9, V79.4–V79.6, V79.8–V79.9, V89.2, Y85.0

*(continued)*

**Table A2 (continued): ABDS 2015 disease list by ICD-10 code**

ABDS 2015 disease/injury	ICD-10 codes <sup>(a)</sup>
<b>Injuries (external cause) (continued)</b>	
Road traffic injuries: pedal cyclists	V10.3–V10.5, V10.9, V11.3–V11.5, V11.9, V12.3–V12.5, V12.9, V13.3–V13.5, V13.9, V14.3–V14.5, V14.9, V15.3–V15.5, V15.9, V16.3–V16.5, V16.9, V17.3–V17.5, V17.9, V18.3–V18.5, V18.9, V19.4–V19.6, V19.8–V19.9
Road traffic injuries: pedestrians	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
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, Y87.9
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
<b>Injuries (nature)</b>	
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 2015 disease list by ICD-10 code**

<b>ABDS 2015 disease/injury</b>	<b>ICD-10 codes<sup>(a)</sup></b>
<b>Injuries (nature) (continued)</b>	
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	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
<b>Kidney and urinary diseases</b>	
Chronic kidney disease	E10.2, E11.2, E12.2, E13.2, E14.2, I12, N02–N07, N08, N13–N16, N18, N39.1, N39.2, Q61.0–Q61.3
Enlarged prostate	N40
Kidney stones	N20, N21
Interstitial nephritis	N10–N12
Other kidney and urinary diseases	N00, N01, N10, N22, N23, N25–N28, N31–N32, N35–N37,
<b>Mental and substance use disorders</b>	
Depressive disorders	F32, F33, F34.1, F34.8, F34.9, 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 2015 disease list by ICD-10 code**

ABDS 2015 disease/injury	ICD-10 codes <sup>(a)</sup>
<b>Mental and substance use disorders (continued)</b>	
Other mental and substance use disorders	F04–F07, F09, F17, F38, F44, F45, F48, F51–F55, F59–F66, F68, F69, F80–F83, F88, F89, F93–F95, F98, F99
<b>Musculoskeletal conditions</b>	
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
<b>Neurological conditions</b>	
Epilepsy	G40, G41
Dementia	F00–F03, G30–G31
Parkinson 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
<b>Oral disorders</b>	
Dental caries	K02, K04
Periodontal disease	K05
Severe tooth loss <sup>(d)</sup>	..
Other oral disorders	K00, K01, K03, K06–K14
<b>Reproductive and maternal conditions</b>	
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 2015 disease list by ICD-10 code**

ABDS 2015 disease/injury	ICD-10 codes <sup>(a)</sup>
<b>Reproductive and maternal conditions (continued)</b>	
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
<b>Respiratory diseases</b>	
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
<b>Skin disorders</b>	
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) This includes salivary gland cancers (C07-C08) which differs to AIHW definition.

(c) Criteria used to diagnose this condition are currently not defined in ICD-10. See 'Gastrointestinal disorders' in Chapter 5 for further details.

(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.



## Appendix B: Additional information and tables for Chapter 3

**Table B1: Number and proportion of deaths by redistribution group, method and target diseases, 2015**

Redistribution group	ICD-10 codes	Method	Scope of target diseases <sup>(a)</sup>	Number	%
Non-specific cancers	C76–C80	Direct evidence	Cancer	2,707	16.5
Non-specific digestive cancers	C26	Direct evidence	Cancer (digestive cancers)	1,237	7.5
Unknown causes	R99	Direct evidence	All diseases	855	5.2
Undetermined intent	Y10–Y34	Direct evidence	All injuries	172	1.0
Heart failure	I50	Indirect MCOD	Cardiovascular, infant/congenital	3,001	18.3
Renal failure	N17, N19	Direct evidence and indirect MCOD	Partial kidney/urinary, all diseases	799	4.9
Unspecified gastrointestinal causes	K92	Direct evidence and indirect MCOD	Gastrointestinal	487	3.0
Peritonitis	K65–K66	Direct evidence and indirect MCOD	Gastrointestinal	79	0.5
Septicaemia, pneumonitis	A40 (excluding A40.3), A41, J69	Indirect MCOD	All diseases	2,373	14.4
Hypertension	I10, I13, I15	Indirect MCOD	All diseases excluding injuries	839	5.1
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–L68, L71–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	1,202	7.3
Unspecified factor	X59	Proportional allocation	Injuries	880	5.4

(continued)

**Table B1 (continued): Number and proportion of deaths by redistribution group, method and target diseases, 2015**

Redistribution group	ICD-10 codes	Method	Scope of target diseases <sup>(a)</sup>	Number	%
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	124	0.8
Unspecified amyloidosis, unspecified respiratory signs and symptoms and cachexia	E85.3–E85.9, R04–R07, R64	Proportional allocation	All diseases excluding injuries	165	1.0
Unspecified diabetes	E14 (excluding E14.2)	Direct evidence	Type 1, Type 2 and Other diabetes	1,513	9.2
<b>All redistribution causes</b>				<b>16,433</b>	<b>100.0</b>

(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 B2: Number of deaths identified for redistribution and associated YLL, by age and sex, 2015**

Age group	Deaths			YLL		
	Males	Females	Persons	Males	Females	Persons
Under 1	45	37	82	3,871	3,183	7,054
1–4	11	6	17	924	505	1,430
5–9	5	3	8	402	233	635
10–14	9	7	16	665	519	1,184
15–19	18	7	25	1,245	484	1,729
20–24	25	7	32	1,612	456	2,068
25–29	34	17	51	2,026	1,010	3,037
30–34	52	19	71	2,831	1,035	3,866
35–39	55	24	79	2,716	1,188	3,904
40–44	90	47	137	4,006	2,098	6,104
45–49	134	66	200	5,337	2,608	7,945
50–54	210	140	350	7,364	4,905	12,269
55–59	290	158	448	8,826	4,790	13,616
60–64	393	215	608	10,173	5,587	15,760
65–69	539	313	852	11,560	6,707	18,267
70–74	657	439	1,096	11,263	7,503	18,766
75–79	980	746	1,726	12,920	9,812	22,732
80–84	1,209	1,212	2,421	11,617	11,520	23,137
85–89	1,550	1,884	3,434	10,237	12,367	22,605
90–94	1,059	2,067	3,126	4,706	9,075	13,781
95–99	335	982	1,317	1,004	2,907	3,911
100 and over	71	252	323	148	523	671
<b>All ages</b>	<b>7,726</b>	<b>8,611</b>	<b>16,337</b>	<b>111,582</b>	<b>85,832</b>	<b>197,417</b>

Source: AIHW analysis of the NMD.

**Table B3: Number and proportion of deaths before and after redistribution and associated change (increase), by disease group: National, 2015**

Disease group	Before redistribution				After redistribution				Increase (before to after)			
	Deaths		YLLs		Deaths		YLLs		Deaths		YLLs	
	Number	%	Number	%	Number	%	Number	%	Number	%	Number	%
Blood/metabolic	1,820	1.2	32,695	1.4	1,918	1.2	34,173	1.4	98	5.1	1,478	4.3
Cancer	42,083	26.8	730,984	31	47,039	29.9	803,489	34.1	4,956	10.5	72,505	9
Cardiovascular	40,532	25.8	463,628	19.7	45,613	29	507,509	21.5	5,081	11.1	43,881	8.6
Endocrine	1,970	1.3	24,870	1.1	3,618	2.3	47,488	2	1,648	45.6	22,618	47.6
Gastrointestinal	5,258	3.3	90,178	3.8	6,244	4	100,386	4.3	986	15.8	10,208	10.2
Infant/congenital	1,157	0.7	79,650	3.4	1,239	0.8	85,121	3.6	82	6.6	5,471	6.4
Infections	5,300	3.4	58,418	2.5	5,691	3.6	62,972	2.7	391	6.9	4,554	7.2
Injuries	9,470	6	307,569	13	10,927	7	334,219	14.2	1,457	13.3	26,650	8
Kidney/urinary	3,689	2.3	43,356	1.8	4,166	2.7	47,593	2	477	11.4	4,237	8.9
Mental	794	0.5	13,258	0.6	845	0.5	14,178	0.6	51	6	920	6.5
Musculoskeletal	1,300	0.8	15,591	0.7	1,407	0.9	16,722	0.7	107	7.6	1,131	6.8
Neurological	16,803	10.7	162,054	6.9	17,481	11.1	168,190	7.1	678	3.9	6,136	3.6
Oral	29	0	249	0	29	0	249	0	0	0	0	0
Reproductive/maternal	34	0	787	0	34	0	809	0	0	0	22	2.7
Respiratory	9,971	6.3	125,236	5.3	10,294	6.5	129,056	5.5	323	3.1	3,820	3
Skin	519	0.3	5,367	0.2	618	0.4	6,229	0.3	99	16	862	13.8
Redistribution	16,433	10.5	204,493	8.7	0	0	0	0	..	..	..	..
<b>All deaths</b>	<b>157,162</b>	<b>100</b>	<b>2,358,383</b>	<b>100</b>	<b>157,162</b>	<b>100</b>	<b>2,358,384</b>	<b>100</b>	<b>..</b>	<b>..</b>	<b>..</b>	<b>..</b>

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 B4: YLL, by age at death used in the ABDS 2015**

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.

**Table B5: Expected years of life remaining at selected ages, GBD standard reference and Australian life tables, by sex, 2003, 2011 and 2015**

Age (years)	GBD 2010 standard	Australia 2003		Australia 2011		Australia 2015	
	Persons	Males	Females	Males	Females	Males	Females
0	86.0	78.1	83.0	79.9	84.3	80.4	84.6
1	85.2	77.5	82.4	79.3	83.5	79.7	83.8
5	81.3	73.6	78.5	75.3	79.6	75.8	79.9
15	71.3	63.7	68.5	65.4	69.7	65.9	70.0
25	61.4	54.1	58.7	55.7	59.8	56.2	60.1
45	41.8	35.2	39.3	36.7	40.4	37.1	40.6
65	23.3	17.8	21.1	19.1	22.0	19.6	22.3
75	14.8	10.8	13.2	11.7	13.8	12.1	14.0
85	7.6	5.7	6.9	6.1	7.2	6.2	7.3
95	3.3	3.1	3.6	3.1	3.4	3.0	3.3
100	2.2	2.5	2.8	2.3	2.5	2.1	2.3
105	1.6	..	..	..	..	..	..

*Notes*

1. Australian life expectancy is calculated by the ABS using multiple years of mortality data: 2002–2004 for 2003, 2010–2012 for 2011 and 2014–2016 for 2015.
2. Australian (2003, 2011 and 2015) life expectancies for age 100 shown here are for all ages 100 or more.

*Sources:* Murray et al. 2012; ABS 2005, 2012, 2017d.

## Appendix C: Additional information and tables for Chapter 4

**Table C1: ABDS 2015 health states and disability weights**

ABDS 2015 health state ID	Health state name	Disability weight	ABDS 2015 health state ID	Health state name	Disability weight
1	Infectious disease: acute episode, mild	0.006	20	Mastectomy	0.036
2	Infectious disease: acute episode, moderate	0.051	21	Stoma	0.095
3	Infectious disease: acute episode, severe	0.133	22	Terminal phase: with medication (for cancers, end-stage kidney or liver disease)	0.540
4	Infectious disease: post-acute consequences (fatigue, emotional lability, insomnia)	0.219	23	Terminal phase: without medication (for cancers, end-stage kidney or liver disease)	0.569
5	Diarrhoea: mild	0.074	24	Acute myocardial infarction: days 1–2	0.432
6	Diarrhoea: moderate	0.188	25	Acute myocardial infarction: days 3–28	0.074
7	Diarrhoea: severe	0.247	26	Angina pectoris: mild	0.033
8	Epididymo-orchitis	0.128	27	Angina pectoris: moderate	0.080
9	Herpes zoster	0.058	28	Angina pectoris: severe	0.167
10	HIV: symptomatic, pre-AIDS	0.274	29	Cardiac conduction disorders and cardiac dysrhythmias	0.224
11	HIV/AIDS: receiving antiretroviral treatment	0.078	30	Claudication	0.014
12	AIDS: not receiving antiretroviral treatment	0.582	31	Heart failure: mild	0.041
13	Intestinal nematode infections: symptomatic	0.027	32	Heart failure: moderate	0.072
14	Lymphatic filariasis: symptomatic	0.109	33	Heart failure: severe	0.179
15	Ear pain	0.013	34	Stroke: long-term consequences, mild	0.019
16	Tuberculosis: without HIV infection	0.333	35	Stroke: long-term consequences, moderate	0.070
17	Tuberculosis: with HIV infection	0.408	36	Stroke: long-term consequences, moderate plus cognition problems	0.316
18	Cancer: diagnosis and primary therapy	0.288	37	Stroke: long-term consequences, severe	0.552
19	Cancer: metastatic	0.451	38	Stroke: long-term consequences, severe plus cognition problems	0.588

(continued)

**Table C1 (continued): ABDS 2015 health states and disability weights**

ABDS 2015 health state ID	Health state name	Disability weight	ABDS 2015 health state ID	Health state name	Disability weight
39	Diabetic foot	0.020	61	Headache: migraine	0.441
40	Diabetic neuropathy	0.133	62	Headache: tension-type	0.037
41	Chronic kidney disease (stage IV)	0.104	63	Multiple sclerosis: mild	0.183
42	End-stage renal disease: with kidney transplant	0.024	64	Multiple sclerosis: moderate	0.463
43	End-stage renal disease: on dialysis	0.571	65	Multiple sclerosis: severe	0.719
44	Decompensated cirrhosis of the liver	0.178	70	Parkinson disease: mild	0.010
45	Gastric bleeding	0.325	71	Parkinson disease: moderate	0.267
46	Crohn's disease or ulcerative colitis	0.231	72	Parkinson disease: severe	0.575
47	Benign prostatic hypertrophy: symptomatic	0.067	73	Alcohol use disorder: mild	0.235
48	Urinary incontinence	0.139	74	Alcohol use disorder: moderate	0.373
49	Impotence	0.017	75	Alcohol use disorder: severe	0.570
50	Infertility: primary	0.008	76	Fetal alcohol syndrome: mild	0.016
51	Infertility: secondary	0.005	77	Fetal alcohol syndrome: moderate	0.056
52	Asthma: controlled	0.015	78	Fetal alcohol syndrome: severe	0.179
53	Asthma: partially controlled	0.036	79	Cannabis dependence	0.266
54	Asthma: uncontrolled	0.133	80	Amphetamine dependence	0.486
55	Chronic obstructive pulmonary disease (COPD) and other chronic respiratory diseases: mild	0.019	81	Cocaine dependence	0.479
56	COPD and other chronic respiratory diseases: moderate	0.225	82	Heroin and other opioid dependence	0.697
57	COPD and other chronic respiratory diseases: severe	0.408	83	Anxiety disorders: mild	0.030
58	Dementia: mild	0.069	84	Anxiety disorders: moderate	0.133
59	Dementia: moderate	0.377	85	Anxiety disorders: severe	0.523
60	Dementia: severe	0.449	86	Major depressive disorder: mild episode	0.145

*(continued)*



**Table C1 (continued): ABDS 2015 health states and disability weights**

ABDS 2015 health state ID	Health state name	Disability weight	ABDS 2015 health state ID	Health state name	Disability weight
87	Major depressive disorder: moderate episode	0.396	110	Hearing loss: severe, with ringing	0.261
88	Major depressive disorder: severe episode	0.658	111	Hearing loss: profound, with ringing	0.277
89	Bipolar disorder: manic episode	0.492	112	Hearing loss: complete, with ringing	0.316
90	Bipolar disorder: residual state	0.032	113	Distance vision: mild impairment	0.003
91	Schizophrenia: acute state	0.778	114	Distance vision: moderate impairment	0.031
92	Schizophrenia: residual state	0.588	115	Distance vision: severe impairment	0.184
93	Anorexia nervosa	0.224	116	Distance vision blindness	0.187
94	Bulimia nervosa	0.223	117	Near vision impairment	0.011
95	Attention deficit hyperactivity disorder	0.045	126	Musculoskeletal problems: legs, mild	0.023
96	Conduct disorder	0.241	127	Musculoskeletal problems: legs, moderate	0.079
97	Asperger syndrome	0.104	128	Musculoskeletal problems: legs, severe	0.165
98	Autism	0.262	129	Musculoskeletal problems: arms, mild	0.028
99	Intellectual disability: mild	0.043	130	Musculoskeletal problems: arms, moderate	0.117
100	Intellectual disability: moderate	0.100	131	Musculoskeletal problems: generalised, moderate	0.317
101	Intellectual disability: severe	0.160	132	Musculoskeletal problems: generalised, severe	0.581
102	Intellectual disability: profound	0.200	133	Gout: acute	0.295
103	Hearing loss: mild	0.010	134	Amputation of finger(s), excluding thumb: long term, with treatment	0.005
104	Hearing loss: moderate	0.027	135	Amputation of thumb: long term	0.011
105	Hearing loss: severe	0.158	137	Amputation of both arms: long term, with treatment	0.123
106	Hearing loss: profound always	0.204	138	Amputation of both arms: long term, without treatment	0.383
107	Hearing loss: complete	0.215	139	Amputation of toe	0.006
108	Hearing loss: mild, with ringing	0.021	140	Amputation of one leg: long term, with treatment	0.039
109	Hearing loss: moderate, with ringing	0.074	141	Amputation of one leg: long term, without treatment	0.173

*(continued)*

**Table C1 (continued): ABDS 2015 health states and disability weights**

ABDS 2015 health state ID	Health state name	Disability weight	ABDS 2015 health state ID	Health state name	Disability weight
142	Amputation of both legs: long term, with treatment	0.088	158	Fracture of foot bones: short term, with or without treatment	0.026
143	Amputation of both legs: long term, without treatment	0.443	159	Fracture of foot bones: long term, without treatment	0.026
144	Burns of <20% total surface area without lower airway burns: short term, with or without treatment	0.141	160	Fracture of hand: short term, with or without treatment	0.010
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	161	Fracture of hand: long term, without treatment	0.014
146	Burns of >=20% total surface area: short term, with or without treatment	0.314	162	Fracture of neck of femur: short term, with or without treatment	0.258
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	163	Fracture of neck of femur: long term, with treatment	0.058
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	164	Fracture of neck of femur: long term, without treatment	0.402
149	Lower airway burns: with or without treatment	0.376	165	Fracture other than neck of femur: short term, with or without treatment	0.111
150	Crush injury: short or long term, with or without treatment	0.132	166	Fracture other than neck of femur: long term, without treatment	0.042
151	Dislocation of hip: long term, with or without treatment	0.016	167	Fracture of patella, tibia or fibula, or ankle: short term, with or without treatment	0.050
152	Dislocation of knee: long term, with or without treatment	0.113	168	Fracture of patella, tibia or fibula, or ankle: long term, with or without treatment	0.055
153	Dislocation of shoulder: long term, with or without treatment	0.062	169	Fracture of pelvis: short term	0.279
154	Other injuries of muscle and tendon (includes sprains, strains, and dislocations other than shoulder, knee, or hip)	0.008	170	Fracture of pelvis: long term	0.182
155	Drowning and non-fatal submersion: short or long term, with or without treatment	0.247	171	Fracture of radius or ulna: short term, with or without treatment	0.028
156	Fracture of clavicle, scapula, or humerus: short or long term, with or without treatment	0.035	172	Fracture of radius or ulna: long term, without treatment	0.043
157	Fracture of face bone: short or long term, with or without treatment	0.067	173	Fracture of skull: short or long term, with or without treatment	0.071

(continued)

**Table C1 (continued): ABDS 2015 health states and disability weights**

ABDS 2015 health state ID	Health state name	Disability weight	ABDS 2015 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	194	Abdominopelvic problem: severe	0.324
175	Fracture of vertebral column: short or long term, with or without treatment	0.111	195	Anaemia: mild	0.004
176	Fractures: treated, long term	0.005	196	Anaemia: moderate	0.052
177	Injured nerves: short term	0.100	197	Anaemia: severe	0.149
178	Injured nerves: long term	0.113	198	Periodontitis	0.007
179	Injury to eyes: short term	0.054	199	Dental caries: symptomatic	0.010
180	Severe traumatic brain injury: short term, with or without treatment	0.214	200	Severe tooth loss	0.067
181	Traumatic brain injury: long-term consequences, minor, with or without treatment	0.094	201	Disfigurement: level 1	0.011
182	Traumatic brain injury: long-term consequences, moderate, with or without treatment	0.231	202	Disfigurement: level 2	0.067
183	Traumatic brain injury: long-term consequences, severe, with or without treatment	0.637	203	Disfigurement: level 3	0.405
184	Open wound: short term, with or without treatment	0.006	204	Disfigurement: level 1 with itch or pain	0.027
185	Poisoning: short term, with or without treatment	0.163	205	Disfigurement: level 2, with itch or pain	0.188
186	Severe chest injury: long term, with or without treatment	0.047	206	Disfigurement: level 3, with itch or pain	0.576
187	Severe chest injury: short term, with or without treatment	0.369	207	Generic uncomplicated disease: worry and daily medication	0.049
188	Spinal cord lesion below neck: treated	0.296	208	Generic uncomplicated disease: anxiety about diagnosis	0.012
189	Spinal cord lesion below neck: untreated	0.623	209	Iodine-deficiency goitre	0.199
190	Spinal cord lesion at neck: treated	0.589	210	Kwashiorkor	0.051
191	Spinal cord lesion at neck: untreated	0.732	211	Severe wasting	0.128
192	Abdominopelvic problem: mild	0.011	212	Speech problems	0.051
193	Abdominopelvic problem: moderate	0.114	213	Motor impairment: mild	0.010

(continued)

**Table C1 (continued): ABDS 2015 health states and disability weights**

ABDS 2015 health state ID	Health state name	Disability weight	ABDS 2015 health state ID	Health state name	Disability weight
214	Motor impairment: moderate	0.061	245	Cocaine dependence, mild	0.116
215	Motor impairment: severe	0.402	246	Concussion	0.110
216	Motor plus cognitive impairments: mild	0.031	247	Distance vision, monocular	0.017
217	Motor plus cognitive impairments: moderate	0.203	248	Epilepsy, less severe (seizures less than once per month)	0.263
218	Motor plus cognitive impairments: severe	0.542	249	Epilepsy, severe (seizures once per month or more)	0.552
219	Rectovaginal fistula	0.501	250	Headache, medication overuse	0.223
233	Low back pain, moderate	0.054	251	Heroin and other opioid dependence, mild	0.335
234	Low back pain, mild	0.020	252	Hyperthyroidism	0.145
235	Alcohol use disorder, very mild	0.123	253	Hypothyroidism	0.019
236	Amphetamine dependence, mild	0.079	254	Mild low back pain with leg pain	0.020
237	Amputation of 1 upper limb (long term, with treatment)	0.039	255	Moderate low back pain with leg pain	0.054
238	Amputation of 1 upper limb (long term, without treatment)	0.118	256	Neck pain, mild	0.053
239	Back pain, most severe, with leg pain	0.384	257	Neck pain, moderate	0.114
240	Back pain, most severe, without leg pain	0.372	258	Neck pain, severe	0.229
241	Back pain, severe, with leg pain	0.325	259	Neck pain, most severe	0.304
242	Back pain, severe, without leg pain	0.272	260	Stress incontinence	0.020
243	Borderline intellectual functioning	0.011	261	Thrombocytopenic purpura	0.159
244	Cannabis dependence, mild	0.039	262	Asymptomatic disease	0.000

Source: GBD 2015.

**Table C2: ABDS 2015 main data sources for YLD estimation**

<b>Disease group</b>	<b>Key national data sources</b>
Blood & metabolic disorders	National Hospital Morbidity Database National Health Survey 2014–15 Australian Health Survey 2011–12 Australian Cystic Fibrosis Data Registry Australian Bleeding Disorders Registry Linked hospitals and NDI components for the NDLDP database Epidemiological studies
Cancer & other neoplasms	Australian Cancer Database National Mortality Database National Hospital Morbidity Database Medicare Benefits Schedule Epidemiological studies
Cardiovascular diseases	National Hospital Morbidity Database Linked hospitals and NDI components for the NDLDP database New Zealand Burden of Disease Study Western Australian linked data
Endocrine disorders	National Diabetes Register National Health Survey 2014–15 Fremantle Diabetes Study
Gastrointestinal disorders	National Hospital Morbidity Database Epidemiological studies
Infant & congenital conditions	National Hospital Morbidity Database National Mortality Database National Perinatal Data Collection Western Australian Intellectual Disability Exploring Answers database Western Australian Register of Developmental Anomalies Australian Cerebral Palsy Register
Infectious diseases	National Notifiable Diseases Surveillance System National Hospital Morbidity Database Australian and New Zealand Assisted Reproductive Database Bettering the Evaluation and Care of Health Epidemiological studies National HIV Register
Injuries	National Hospital Morbidity Database National Non-Admitted Patient Emergency Department Care Database
Hearing & vision disorders	National Health Survey 2014–15 Australian Hearing Database Blue Mountains Hearing Study

*(continued)*

**Table C2 (continued): ABDS 2015 main data sources for YLD estimation**

<b>Disease group</b>	<b>Key national data sources</b>
Hearing & vision disorders (continued)	Melbourne Vision Impairment Project National Eye Health Survey Epidemiological studies
Kidney and urinary diseases	Australian and New Zealand Dialysis and Transplantation Registry National Hospital Morbidity Database Australian Health Survey 2011–12 Western Australian linked data
Mental and substance use disorders	National Survey of Mental Health and Wellbeing Young Minds Matter survey Western Australian Intellectual Disability Exploring Answers database The Australian National Survey of High Impact Psychosis Alcohol and Other Drug Treatment Services National Minimum Dataset Global Burden of Disease Study 2015
Musculoskeletal conditions	National Health Survey 2014–15 Australian Health Survey 2011–12 National Health Survey 2004–05
Neurological conditions	National Hospital Morbidity Database National Health Survey 2014–15 AIHW dementia analyses Epidemiological studies Linked hospitals and NDI components for the NDLDP database
Oral disorders	National Survey of Adult Oral Health National Dental Telephone Interview Survey Child Dental Health Survey
Reproductive and maternal conditions	National Hospital Morbidity Database Australian and New Zealand Assisted Reproduction Database Australian Longitudinal Study on Women's Health Bettering the Evaluation and Care of Health Epidemiological studies
Respiratory diseases	National Mortality Database National Hospital Morbidity Database Western Australian linked data National Health Surveys 2014–15 and 2004–05 Australian Health Survey 2011–12 Burden of Obstructive Lung Disease study National Hospital Morbidity Database Epidemiological studies

*(continued)*

**Table C2 (continued): ABDS 2015 main data sources for YLD estimation**

Disease group	Key national data sources
Skin disorders	National Health Survey 2014–15 National Hospital Morbidity Database Epidemiological studies

# 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 (years) and sex**

Disease	Males				Females			
	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 <sup>(a)</sup>	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) C80 Unknown primary will require further redistribution.

Sources: Pooled Western Australia Cancer Registry data, 2007–2011 and South Australia Cancer Registry data, 2007–2011.



**Table D2: Redistribution proportions of ill-defined cancers (C39, C76–C80, C97), by age (years) and sex**

Disease	Males				Females			
	0–44	45–64	65–84	85+	0–44	45–64	65–84	85+
Unknown primary <sup>(a)</sup>	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
Lip and oral cavity cancer	0.025	0.032	0.021	0.012	0.027	0.004	0.001	0.012
Nasopharyngeal cancer	0.003	0.004	0.003	0.002	0.004	0.001	0.000	0.002
Other oral cavity and pharynx cancers	0.017	0.021	0.014	0.008	0.018	0.003	0.001	0.008
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
Acute myeloid leukaemia	0.000	0.000	0.001	0.000	0.000	0.007	0.001	0.000
Chronic myeloid leukaemia	0.000	0.000	0.000	0.000	0.000	0.001	0.000	0.000
Acute lymphoblastic leukaemia	0.000	0.000	0.000	0.000	0.000	0.001	0.000	0.000
Chronic lymphocytic leukaemia	0.000	0.000	0.001	0.000	0.000	0.003	0.000	0.000
Other leukaemias	0.000	0.000	0.000	0.000	0.000	0.002	0.000	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 sequela duration, by malignant cancer type, ABDS 2015**

Cancer type	Sequelae duration (months)			
	Diagnosis and primary therapy	Controlled phase	Metastatic phase	Terminal phase
Lip and oral cavity cancer <sup>(1)</sup>	5.30	Remainder of the year	9.33	1
Nasopharyngeal cancer <sup>(1)</sup>	5.30	Remainder of the year	13.19	1
Other oral cavity and pharynx cancers <sup>(1)</sup>	5.30	Remainder of the year	7.91	1
Laryngeal cancer <sup>(1)</sup>	5.30	Remainder of the year	8.84	1
Oesophageal cancer <sup>(1)</sup>	5.00	Remainder of the year	4.6	1
Stomach cancer <sup>(1)</sup>	5.20	Remainder of the year	3.88	1
Bowel cancer <sup>(1)</sup>	4.00	Remainder of the year	9.69	1
Liver cancer <sup>(1)</sup>	4.00	Remainder of the year	2.51	1
Gallbladder cancer <sup>(1)</sup>	4.00	Remainder of the year	3.47	1
Pancreatic cancer <sup>(1)</sup>	4.10	Remainder of the year	2.54	1
Lung cancer <sup>(1)</sup>	3.30	Remainder of the year	4.51	1
Mesothelioma <sup>(1)</sup>	4.00	Remainder of the year	7.75	1
Melanoma ( $\leq 1.00$ mm) <sup>(2)</sup>	0.46	Remainder of the year	7.18	1
Melanoma (1.01 mm–2.00 mm) <sup>(2)</sup>	0.61	Remainder of the year	7.18	1
Melanoma (2.01 mm–4.00 mm) <sup>(2)</sup>	0.71	Remainder of the year	7.18	1
Melanoma ( $> 4.00$ mm) <sup>(2)</sup>	1.66	Remainder of the year	7.18	1
Melanoma of the skin (unknown) <sup>(2)</sup>	0.86	Remainder of the year	7.18	1
Non-melanoma skin cancer	0.46	Remainder of the year	17	1
Breast cancer (females) <sup>(3)</sup> (<20 mm)	3.40	Remainder of the year	17.7	1
Breast cancer (females) (20 mm–50 mm) <sup>(3)</sup>	6.80	Remainder of the year	17.7	1
Breast cancer (females) ( $> 50$ mm) <sup>(3)</sup>	8.00	Remainder of the year	17.7	1
Breast cancer (females) (unknown size) <sup>(3)</sup>	6.07	Remainder of the year	17.7	1
Breast cancer (males) <sup>(3)</sup>	6.07	Remainder of the year	17.7	1
Cervical cancer <sup>(1)</sup>	4.80	Remainder of the year	9.21	1
Uterine (endometrium) cancer <sup>(1)</sup>	4.60	Remainder of the year	11.6	1
Ovarian cancer <sup>(1)</sup>	3.20	Remainder of the year	25.6	1
Prostate cancer <sup>(1)</sup>	4.00	Remainder of the year	30.35	1
Testicular cancer <sup>(1)</sup>	3.70	Remainder of the year	19.47	1
Bladder cancer <sup>(1)</sup>	5.10	Remainder of the year	5.8	1
Kidney cancer <sup>(1)</sup>	5.30	Remainder of the year	5.38	1
Brain and central nervous system cancer <sup>(1)</sup>	5.00	Remainder of the year	6.93	1
Thyroid cancer <sup>(1)</sup>	3.00	Remainder of the year	19.39	1
Non-Hodgkin lymphoma <sup>(1)</sup>	3.70	Remainder of the year	7.7	1
Hodgkin lymphoma <sup>(1)</sup>	3.70	Remainder of the year	26	1
ALL <sup>(1)</sup>	12.00	Remainder of the year	7.02	1

*(continued)*

**Table D3 (continued): Average sequela duration by malignant cancer type, ABDS 2015**

Cancer type	Sequelae duration (months)			
	Diagnosis and primary therapy	Controlled phase	Metastatic phase	Terminal phase
AML <sup>(1)</sup>	6.00	Remainder of the year	4.6	1
CLL <sup>(1)</sup>	6.00	Remainder of the year	48	1
CML <sup>(1)</sup>	6.00	Remainder of the year	4.6	1
Other leukaemias <sup>(1)</sup>	6.73	Remainder of the year	15.05	1
Myeloma <sup>(1)</sup>	7.00	Remainder of the year	36.82	1
Other lymphohaematopoietic (blood) cancers <sup>(3)</sup>	7.00	Remainder of the year	36.82	1
Unknown primary cancer <sup>(3)</sup>	3.30	0	Remainder of the year	1
Other malignant neoplasms <sup>(1)</sup>	4.40	Remainder of the year	15.81	1

Sources: GBD 2013 Collaborators 2015; Melanoma Management Guide for GPs and Melanoma Institute Australia; Expert opinion from Dr Catherine Shannon, Senior Medical Oncologist, Mater Cancer Care Centre; and Professor Christobel Saunders, School of Surgery, University of Western Australia.

## Hearing and vision disorders

**Table D4: Proportion of tinnitus in hearing impaired population, by age, sex and severity level**

Age group (years)	Proportion of tinnitus within each severity level of hearing loss (%)			
	Mild	Moderate	Severe	Profound
<b>Males</b>				
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
<b>Females</b>				
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

# Infant and congenital conditions

**Table D5: Severity distribution used for cerebral palsy, by Gross Motor Function Classification System (GMFCS) level**

GMFCS levels	Description	GBD health state	%
Level I	Walks without limitations	Motor impairment: mild	35.3
Level II	Walks with limitations, including long distances, balancing, running or jumping; requires use of mobility devices when first learning to walk, and may rely on wheeled mobility equipment when outside of home for travelling long distances	Motor impairment: moderate	24.2
Level III	Walks with adaptive equipment assistance. Requires mobility assistance to walk indoors, while utilising wheeled mobility outdoors; can sit on own or with limited external support; and has some independence in standing transfers	Motor impairment: moderate	11.8
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	13.3
Level V	Severe head and trunk control limitations. Requires extensive use of assisted technology and physical assistance; and to be transported in a wheelchair.	Motor impairment: severe	15.3

Source: Cerebral Palsy Alliance 2016.

**Table D6: 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 D7: 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 <sup>(a)</sup>	50, 51	..
	Pelvic inflammatory disease due to chlamydia	193, 194	..
Gonorrhoea	Gonococcal infection	1	1–2 weeks
	Infertility due to gonorrhoea <sup>(a)</sup>	50, 51	..
	Pelvic inflammatory disease due to gonorrhoea	193, 194	..
Other sexually transmitted infections	Infertility due to other sexually transmitted infections <sup>(a)</sup>	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	Varicella	1	Chickenpox (children): 1 week Chickenpox(adults): 10 days

(continued)

**Table D7 (continued): Sequelae, health states and durations for infectious diseases**

Disease	Sequela	ABDS 2011 health state identifier	Duration (short-term sequelae)
Herpes zoster	Herpes zoster	4, 9	Shingles: 2 weeks Post-herpetic neuralgia: 3 months
Mumps	Mumps	2, 3	10 days (non-hospitalised), 14 days (hospitalised)
<i>Haemophilus influenzae</i> type b	<i>Haemophilus influenzae</i> 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 <sup>(b)</sup>	115, 116	..
	Low vision due to trachoma <sup>(b)</sup>	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
Urinary tract infections	Urinary tract infections	2, 3	7 days (non-hospitalised), 14 days (hospitalised)

(a) Part of infertility envelope.

(b) Part of vision envelope.

# Injuries

**Table D8: Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and mortality risk ratio for long-term injuries**

Injury (nature)	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years) admitted	Duration of short-term health loss (years) non-admitted	Mortality risk ratio
Traumatic brain injury	Traumatic brain injury short-term, minor	246	S06.00, S06.02	0.101	0.096	..
	Traumatic brain injury short-term, moderate-severe	180	S06 (excl S06.00, S06.02)	0.110	0.074	..
	Traumatic brain injury long-term, minor	181	S06.00, S06.02	..	..	1.00
	Traumatic brain injury long-term, moderate	182	S06 (excl S06.00, S06.02)	..	..	2.18
	Traumatic brain injury long-term, severe	183	S06 (excl S06.00, S06.02)	..	..	2.18
	Skull fracture short-term	173	S020, S021, S027, S029	0.126	0.101	..
	Skull fracture long-term	173	S020, S021, S027, S029	..	..	1.00
Spinal cord injury						2.72 (Under 60 years), 1.89 (60 years or older)
	Below neck – complete severe	188	S24.11	0.500	0.500	2.72 (Under 60 years), 1.89 (60 years or older)
	Below neck – incomplete severe	215	S24.12 (33%)	0.500	0.500	2.72 (Under 60 years), 1.89 (60 years or older)
	Below neck – incomplete moderate	214	S24.12 (67%)	0.500	0.500	2.72 (Under 60 years), 1.89 (60 years or older)
	Below neck – mild	213	S24.0, S24.1 (excluding S24.11, S24.12) S24.7, S34.0, S34.1, S34.7, T06.1, T09.3	0.077	0.077	1.00

(continued)

**Table D8 (continued): Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and mortality risk ratio for long-term injuries**

Injury (nature)	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years) admitted	Duration of short-term health loss (years) non-admitted	Mortality risk ratio
Spinal cord injury (continued)						
	At neck – complete severe	190	S14.11	0.500	0.500	5.03 (Under 60 years), 2.48 (60 years or over)
	At neck – incomplete severe	215	S14.12, S14.13 (33%)	0.500	0.500	5.03 (Under 60 years), 2.48 (60 years or over)
	At neck – incomplete moderate	214	S14.12, S14.13 (67%)	0.500	0.500	5.03 (Under 60 years), 2.48 (60 years or over)
	At neck – mild	213	S14.0, S14.7, T06.0. S14.10, S14.1 (excluding S14.11, S14.12, S14.13)	0.077	0.077	1.00
Internal and crush injury						
	Crush injury	150	S07, S17, S18, S47, S380, S382, S383, S57, S67, S77, S87, S97, T04, T147	0.167	0.030	5.23
	Severe chest injury	187	S110, S224, S225, S25, S26, S27, S28, S297	0.148	0.115	1.00
	Abdominal /pelvic injuries	187	S35–S37, S381, S396, S397, T065	0.057	0.057	1.00
Poisoning						
	Poisoning short-term	185	T36–T65	0.011	0.005	..
	Poisoning long-term	216	T36–T65	..	..	1.00

(continued)



**Table D8 (continued): Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and mortality risk ratio for long-term injuries**

Injury (nature)	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years) admitted	Duration of short-term health loss (years) non-admitted	Mortality risk ratio
Drowning and submersion injuries	Drowning and non-fatal submersion short-term	155	T751	0.011	0.005	..
	Drowning and non-fatal submersion long-term	155	T751	..	..	1.00
Hip fracture	Neck of femur short-term	162	S720, S721, S722	0.216	0.197	..
						1.00 (Under 50 years), 3.97 (50–75 years), 2.42 (75 years or over)
	Neck of femur long-term	163	S720, S721, S722	..	..	
	Other than neck of femur short-term	165	S723–S729, S79	0.233	0.167	..
	Other than neck of femur long-term	176	S723–S729, S79	..	..	1.00
Humerus fracture	Humerus fracture	156	S422, S423, S424, S427	0.175	0.142	
Tibia and ankle fracture	Tibia or fibula fracture	167	S821, S822, S823, S827, S824, S828, S829, T12	0.359	0.257	1.00
	Ankle fracture	167	S825, S826	0.359	0.257	1.00
Other fractures	Patella	167	S820	0.359	0.257	1.00
	Clavicle or scapula	156	S228, S229, S420, S421, S428, S429, S497	0.175	0.142	1.00
	Face bone, short-term	157	S022–S026, S028	0.126	0.101	..
	Face bone, long-term	157	S022–S026, S028	..	..	1.00
	Foot bone	158	S92	0.134	0.099	1.00

(continued)

**Table D8 (continued): Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and mortality risk ratio for long-term injuries**

Injury (nature)	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years) admitted	Duration of short-term health loss (years) non-admitted	Mortality risk ratio
Other fractures (continued)	Hand bone	160	S620–S624, S625–S627, S628, S697	0.099	0.110	1.00
	Pelvis	169	S321, S323–S328, T021	0.167	0.148	1.00
	Coccyx	184	S322	0.233	0.205	1.00
	Radius or ulna	171	S52, S597, T10	0.131	0.112	1.00
	Sternum or one or two ribs	174	S222, S223	0.148	0.115	1.00
	Vertebral column	175	S12, S220–S221, S320, T08	0.233	0.205	1.00
	Other	176	T020, T022–T029	0.167	0.148	1.00
Dislocations	Shoulder joint	153	S430–S433	0.170	0.148	..
	Shoulder other than joint	184	S530–S531	0.178	0.131	..
	Hip	151	S730	0.110	0.085	..
	Knee	152	S830, S831	0.110	0.112	..
	Other	154	S131–S133, S231–S232, S331–S333, S630, S631, S632, S930, S933, S930, S933, S030–S033, S931, T03, T092, T112, T132, T143	0.178	0.131	..
Soft tissue injuries			S130, S46, S832–S837, S860, S932, S934, S96, S134, S230, S233, S235, S290, S335–S337, S034–S035, S135–S136, S234, S390, S532–S534, S56, S633–S637, S66, S731, S76, S861–S869, S935–S936, T064, T095, T115, T135, T146, S16, S330, S334	0.178	0.131	..
	Soft tissue injuries	154				

(continued)

**Table D8 (continued): Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and mortality risk ratio for long-term injuries**

Injury (nature)	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years) admitted	Duration of short-term health loss (years) non-admitted	Mortality risk ratio
Burn injuries	Non-airway burn, short-term, minor	144	(T31.0 or T31.1) and (T20–T30, excluding T20, T23, T26 and T27)	0.077	0.038	..
	Non-airway burn, long-term, minor	145	(T31.0 or T31.1) and (T20–T30, excluding T20, T23, T26 and T27) or (T31.0) and (T20, T23 or T26)	..	..	2.10 (45 years or over)
	Non-airway burn, short-term, severe	146	(T31.2 –T31.9) and (T20–T30, excluding T20, T23, T26 and T27)	0.164	0.164	..
	Non-airway burn, long-term, severe	147	[(T31.2 –T31.9) and (T20–T30, excluding T20, T23, T26 and T27)] or [(T31.1 – T31.9) and (T20, T23 or T26)]	..	..	1.30 (45 years or over)
	Airways burn	149	T27	0.077	0.077	1.00
	Other injuries	Amputation of finger(s), excluding thumb	134	S681, S682	0.500	0.500
	Amputation of thumb	135	S680	0.500	0.500	1.00
	Amputation of one arm	237	S48, S58, S683, S684, S688, S689, T116	0.500	0.500	1.00
	Amputation of both arms	137	T050–T052	0.500	0.500	1.00
	Amputation of toe	139	S981, S982	0.500	0.500	1.00

*(continued)*

**Table D8 (continued): Sequelae, health state identifiers, ICD-10 AM codes, average duration of short-term and mortality risk ratio for long-term injuries**

Injury (nature)	Sequela	ABDS 2011 health state identifier	ICD-10 AM codes	Duration of short-term health loss (years) admitted	Duration of short-term health loss (years) non-admitted	Mortality risk ratio
Other injuries (continued)	Amputation of one leg	140	S78, S88, S980, S983, S984, T136, T056	0.500	0.500	1.00
	Amputation of both legs	142	T053–T055	0.500	0.500	1.00
	Injured nerves, short-term	177	S04, S142–S146, S242–S246, S342–S346, S348, S44, S54, S64, S74, S84, S94, T062, T113, T133, T144, T094	0.170	0.099	..
	Injured nerves, long-term	178	S04, S142–S146, S242–S246, S342–S346, S348, S44, S54, S64, S74, S84, S94, T062, T113, T133, T144, T094	..	..	1.00
	Injury to eyes	179	S05, T15	0.123	0.137	1.00
	Superficial injuries	184	S00, S10, S20, S30, S40, S50, S60, S70, S80, S90, T00, T090, T110, T130, T140	0.115	0.049	1.00
	Open wound	184	S01, S08, S111–S119, S15, S21, S31, S399, S41, S51, S55, S61, S65, S71, S75, S81, S85, S91, S95, T01, T091, T111, T114, T131, T134, T141	0.099	0.049	1.00
	All other injuries	184	S09, S19, S298, S299, S45, S598, S599, S698, S699, S89, S99, T058, T059, T063, T068, T07, T096, T098, T099, T118, T119, T138, T139, T142, T145, T148, T149, T16–T19, T33, T34, T35, T66, T67, T68, T69, T70, T71, T73, T74, T75, T79, T80, T81, T88	0.008	0.005	1.00

# Appendix E: Additional information and tables for Chapter 6

**Table E1: Life table data sources**

Population	Data year	Data source
<b>National and state/territory</b>	2003	<i>Life tables, states, territories and Australia, 2002–2004</i> (ABS 2007)
	2011	<i>Life tables, states, territories and Australia 2010–2012</i> (ABS 2013c)
	2015	<i>Life tables, states, territories and Australia 2014–2016</i> (ABS 2017e)
<b>Remoteness</b>	2011	<i>Life tables by remoteness areas, 2010–2012</i> (ABS 2017f) – customised report
	2015	<i>Life table by remoteness areas, 2014–2016</i> (ABS 2018d) – customised report
<b>Socioeconomic group</b>	2011	<i>Life tables by Index of Relative Socioeconomic Disadvantage (IRSD), 2010–2012</i> (ABS 2017g) – customised report
	2015	<i>Life tables by Index of Relative Socioeconomic Disadvantage (IRSD), 2014–2014</i> (ABS 2018e) – customised report

# Appendix F: Additional information and tables for Chapter 7

## 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 their 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; and be timely, accurate, reliable and credible.

Published and unpublished data sources were assessed according to the criteria in Box F1. These criteria 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 F1: 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 (sub-national)
3. regional population (such as New Zealand, the United States of America, Canada).

*(continued)*

## **Box F1 (continued): Criteria for risk factor exposure data selection**

### **Currency**

The data source should ideally be within 5 years of the reference year. Data sources for 2003–2009 may also be included if no later data sources are available.

### **Accuracy**

The data source should ideally have more than 90% case ascertainment or coverage of the population of interest, and a RSE or 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.

### **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.

*(continued)*

### **Box F1 (continued): Criteria for risk factor exposure data selection**

#### **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 to 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 F1.

- 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 with availability of better data.

A data source scoring predominantly low was not included.



**Table F1: Assessment matrix for exposure data to be used in the ABDS 2015**

Data source										
Data provider										
Level of disaggregation										
Rating	Comparability	Relevance/representativeness		Currency	Accuracy			Validation	Credibility	Accessibility/ timeliness
		Australian	Indigenous		Ascertainment/ coverage	Error (sampling/ non-sampling)	Measurement error			
High	Consistent	National	National	2011 or later	>90%	Less than 25% RSE	Clinically reported	Validated	Published and peer reviewed	Currently available
Medium	Comparable	Sub- national	Sub-national	2003–2010	60%–90%	25%–50% RSE	Self-reported		Published but not peer reviewed	Available in time for analysis
			Total Australian or non-Indigenous					Not validated	Not published but peer reviewed	
Low	Inconsistent	Sub- or super- regional	Other Indigenous population (New Zealand, United States, Canada)	Before 2003	<60%	More than 50% RSE	Not known	Not published nor peer reviewed	Unlikely to be available in time for analysis.	

## Additional tables

**Table F2: Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREDs, data sources and units for calculating relative risks**

Risk factor	Risk factor exposure	Risk factor measure	Disease outcome	TMRED	National data source	Units for effect size calculation		
Air pollution	Air pollution	Particulate matter (2.5 µg/m <sup>3</sup> )	COPD, coronary heart disease, lower respiratory infections, lung cancer, stroke	2.4–5.9 µg/m <sup>3</sup> (PM2.5)	State/territory-based air monitoring stations	Daily maximum atmospheric particulate matter (PM2.5)		
Alcohol use	Alcohol use	Former drinkers	Atrial fibrillation and flutter; bowel cancer; coronary heart disease; epilepsy, hypertensive heart disease; laryngeal cancer; lower respiratory infections; lip and oral cavity cancer; nasopharynx cancer; oesophageal cancer; other oral cavity and pharynx cancers; pancreatitis; stroke	No alcohol use	NDSHS 2016	Former drinker		
		Average daily alcohol consumption by current drinkers	Atrial fibrillation and flutter; bowel cancer; breast cancer; coronary heart disease; drowning; diabetes; epilepsy; falls; fire, burns and scalds; homicide and violence; hypertensive heart disease; laryngeal cancer; lip and oral cavity cancer; lower respiratory infections; nasopharynx cancer; oesophageal cancer; other land transport injuries; other oral cavity and pharynx cancers; other unintentional injuries; pancreatitis; RTI—motor vehicle occupants; RTI—motorcyclists; RTI—pedal cyclists; road traffic injuries—pedestrians; stroke				NDSHS 2016; apparent consumption of alcohol data	Average consumption of pure alcohol (g per day)
		Alcohol use and dependence	Alcohol use disorders, accidental poisoning, liver cancer, chronic liver disease				NMD; GBD 2016	Direct evidence
		Alcohol dependence	Suicide and self-inflicted injuries				ABDS 2015	Prevalence alcohol use disorders

(continued)

**Table F2 (continued): Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREDS, data sources and units for calculating relative risks**

Risk factor	Risk factor exposure	Risk factor measure	Disease outcome	TMRED	National data source	Units for effect size calculation
Child abuse and neglect	Child abuse and neglect	Physical, sexual and emotional abuse and neglect	Anxiety disorders, depressive disorders, suicide and self-inflicted injuries	No child abuse and neglect	Moore et al. 2015	Prevalence of childhood abuse and neglect
Dietary risks	Diet low in fish and seafood	Average daily consumption of fish and seafood	Coronary heart disease	Consumption of fish or seafood 100 g per week	Self-reported from AHS 2011–12	Per 15 g per day intake decrease
	Diet low in fruit	Average daily consumption of fresh, frozen, cooked, canned, or dried fruits (excluding fruit juices)	Coronary heart disease, lung cancer, lip and oral cavity cancer, nasopharynx cancer, other oral cavity and pharynx cancers, stroke, type 2 diabetes	Consumption of fruit between 200 and 300 g per day	Self-reported from AHS 2011–12	Per 100 g per day intake decrease
	Diet low in legumes	Average daily consumption of fresh, frozen, cooked, canned, or dried legumes	Coronary heart disease	Consumption of legumes between 50 and 70 g per day	Self-reported from AHS 2011–12	Per 50 g per day intake decrease
	Diet low in milk	Average daily consumption of milk including non-fat, low-fat, and full-fat milk, excluding soy milk and other plant derivatives	Bowel cancer	Consumption of milk between 350 and 520 g per day	Self-reported from AHS 2011–12	Per 226.8 g per day intake decrease
	Diet low in nuts and seeds	Average daily consumption of nut and seed foods	Coronary heart disease, type 2 diabetes	Consumption of nuts and seeds between 16 and 25 g per day	Self-reported from AHS 2011–12	Per 4.05 g per day intake decrease
	Diet low in polyunsaturated fats	Average daily consumption of polyunsaturated fats	Coronary heart disease	Consumption of polyunsaturated fatty acids between 9% and 13% of total daily energy	Self-reported from AHS 2011–12	Per 5% energy from polyunsaturated fat decrease
	Diet high in processed meats	Average daily consumption of meat preserved by smoking, curing, salting, or addition of chemical preservatives	Bowel cancer, coronary heart disease, type 2 diabetes, stomach cancer	Consumption of processed meat between 0 and 4 g per day	Self-reported from AHS 2011–12	Per 50 g per day intake increase

(continued)

**Table F2 (continued): Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREs, data sources and units for calculating relative risks**

Risk factor	Risk factor exposure	Risk factor measure	Disease outcome	TMRED	National data source	Units for effect size calculation
Dietary risks (continued)	Diet high in red meat	Average daily consumption of red meat (beef, pork, lamb, and goat) (excluding poultry, fish, eggs and all processed meats)	Bowel cancer, diabetes	Consumption of red meat between 18 and 27 g per day	Self-reported from AHS 2011–12	Per 100 g per day intake increase
	Diet high in sodium	Consumption of sodium	High blood pressure-linked diseases: Aortic aneurysm, atrial fibrillation and flutter, cardiomyopathy, chronic kidney disease, coronary heart disease, dementia, hypertensive heart disease, inflammatory heart disease, other cardiovascular diseases, peripheral vascular disease, rheumatic heart disease, stroke	24 hr urinary sodium of 2 g per day	Self-reported from AHS 2011–12; adjusted based on urinary sodium estimate (Powles et al. 2013)	Per 2.3 g per day intake increase
	Diet high in sugar sweetened beverages	Consumption of beverages with ≥50 kcal per 226.8 g serving, including carbonated beverages, sodas, energy drinks and fruit drinks (excluding 100% fruit and vegetable juices)	Overweight- and obesity-linked diseases: Acute lymphoblastic leukaemia, acute myeloid leukaemia, asthma, atrial fibrillation and flutter, back pain and problems, bowel cancer, breast cancer, cataract and other lens disorders, chronic kidney disease, chronic lymphocytic leukaemia, chronic myeloid leukaemia, coronary heart disease, dementia, gallbladder and bile duct disease, gallbladder cancer, gout, hypertensive heart disease, kidney cancer, liver cancer, myeloma, non-Hodgkin lymphoma, oesophageal cancer, osteoarthritis, other leukaemias, ovarian cancer, pancreatic cancer, stroke, thyroid cancer, type 2 diabetes, uterine cancer	No consumption of sugar-sweetened beverages	Self-reported from AHS 2011–12 (day 1 only)	Per 226.8 g per day intake increase
	Diet low in vegetables	Average daily consumption of fresh, frozen, cooked, canned, or dried vegetables, (excluding vegetable juices, legumes and starchy vegetables such as potatoes or corn)	Coronary heart disease, stroke	Consumption of vegetables between 290 and 430 g per day	Self-reported from AHS 2011–12	Per 100 g per day of vegetable intake decrease

(continued)

**Table F2 (continued): Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREDS, data sources and units for calculating relative risks**

Risk factor	Risk factor exposure	Risk factor measure	Disease outcome	TMRED	National data source	Units for effect size calculation
Dietary risks (continued)	Diet low in whole grains (including high fibre cereals)	Average daily consumption of wholegrain or higher fibre breads, cereals, rice, pasta, crumpets, muffins, crispbreads, relevant fortified cereals with 1 g of fibre per 10 g of carbohydrate	Coronary heart disease, stroke, type 2 diabetes	Consumption of whole grains between 100 and 150 g per day	Self-reported from AHS 2011–12	Per 50 g per day intake decrease
High blood plasma glucose	Intermediate hyperglycaemia; diabetes	High fasting plasma glucose	Chronic kidney disease, coronary heart disease, stroke	Blood plasma glucose 4.8–5.4 mmol/L	AHS 2011–12	Per 1 mmol/L of fasting plasma glucose increase
	Diabetes	Self-reported diabetes	Bladder cancer, bowel cancer, breast cancer, cataract and other lens disorders, dementia, glaucoma, liver cancer, lung cancer, ovarian cancer, pancreatic cancer, peripheral vascular disease	No diabetes	ABDS 2015	Prevalence of type 1, type 2 and other diabetes
	Diabetes	Diabetes	Chronic kidney disease, type 2 diabetes, type 1 diabetes, other diabetes	No diabetes	GBD 2016	Direct evidence
High blood pressure	High blood pressure	Systolic blood pressure	Aortic aneurysm, atrial fibrillation and flutter, cardiomyopathy, chronic kidney disease, coronary heart disease, dementia, hypertensive heart disease, inflammatory heart disease, other cardiovascular diseases, peripheral vascular disease, rheumatic heart disease, stroke	Systolic blood pressure between 110–115 mmHg	NHS 2014–15	Per 10 mmHg of systolic blood pressure increase
High cholesterol	High cholesterol	Total cholesterol	Coronary heart disease, stroke	Total cholesterol between 2.8–3.4 mmol/L	AHS 2011–12	Per 1 mmol/L of total cholesterol increase

(continued)

**Table F2 (continued): Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREDS, data sources and units for calculating relative risks**

Risk factor	Risk factor exposure	Risk factor measure	Disease outcome	TMRED	National data source	Units for effect size calculation
High sun exposure	Sun exposure	n.a.	Melanoma, non-melanoma skin cancer	No health outcomes from sun exposure	Lucas et al. 2006; F Xiang 2015, pers. comm., 11 November 2015	Direct evidence
Illicit drug use	Cannabis use	Cannabis dependence	Anxiety disorders, depressive disorders, schizophrenia	No illicit drug use	ABDS 2015	Prevalence illicit drug use disorders
		Driving under the influence of cannabis	Road traffic injuries—motorcyclists and road traffic injuries—motor vehicle occupants		NDSHS 2016	Prevalence driving under the influence of illicit drugs
		Cannabis use and dependence	Accidentals poisoning		NMD	Direct evidence
	Amphetamine, cocaine and opioid use	Amphetamine, cocaine and opioid or dependence	Suicide and self-inflicted injuries		ABDS 2015	Prevalence illicit drug use disorders
		Driving under the influence of amphetamine, cocaine or opioids	Road traffic injuries—motorcyclists and road traffic injuries—motor vehicle occupants		NDSHS 2016	Prevalence driving under the influence of illicit drugs
		Amphetamine or opioid use and dependence	Accidental poisoning		NMD	Direct evidence
Amphetamine, cannabis cocaine, opioid and other illicit drug use	Illicit drug dependence	Drug use disorders (excluding alcohol)	ABDS 2015	Direct evidence		

*(continued)*

**Table F2 (continued): Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREDs, data sources and units for calculating relative risks**

Risk factor	Risk factor exposure	Risk factor measure	Disease outcome	TMRED	National data source	Units for effect size calculation
Illicit drug use	Unsafe injecting practices	Unsafe injecting practices	Chronic liver disease, hepatitis B, hepatitis C, HIV/AIDS, liver cancer	No unsafe injecting practices	National notifiable disease annual surveillance reports (The Kirby Institute)	Direct evidence
Impaired kidney function	Chronic kidney disease stage 1–3	Chronic kidney disease stages 1–2	Coronary heart disease, dementia, gout, peripheral vascular disease, stroke	No chronic kidney disease	AHS 2011–12	Prevalence of chronic kidney disease stages 1–2
		Chronic kidney disease stage 3	Coronary heart disease, dementia, gout, peripheral vascular disease, stroke, chronic kidney disease		AHS 2011–12	Prevalence of chronic kidney disease stage 3
		Chronic kidney disease stages 4–5	Coronary heart disease, dementia, gout, peripheral vascular disease, stroke, chronic kidney disease		AHS 2011–12; ANZDATA as for ABDS 2015	Prevalence of chronic kidney disease stages 4–5
Intimate partner violence	Intimate partner violence	Physical, sexual, emotional abuse from a cohabitating partner	Anxiety disorders, alcohol use disorders, early pregnancy loss, depressive disorders, homicide and violence, suicide and self-inflicted injuries	No exposure to intimate partner violence	ABS Personal Safety Survey 2016; National Homicide Monitoring Program	Ever been exposed to intimate partner violence since the age of 15 years (prevalence)
Iron deficiency	Iron deficiency		Iron deficiency anaemia	No iron deficiency anaemia		All of iron deficiency anaemia is attributable

*(continued)*

**Table F2 (continued): Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREDS, data sources and units for calculating relative risks**

Risk factor	Risk factor exposure	Risk factor measure	Disease outcome	TMRED	National data source	Units for effect size calculation
Low bone mineral density	Low bone mineral density		Hip fracture, humerus fracture, other fractures, tibia and ankle fracture	95th percentile of the Third National Health and Nutrition Examination Survey (NHANES-III) cohort by age (Looker et al. 2012)	Geelong Osteoporosis Study (Barwon Health)	Standardised bone mineral density at the femoral neck
Occupational exposures and hazards	Occupational exposures and hazards	Occupational injuries	Drowning; falls; fire, burns and scalds; homicide and violence; road traffic injuries—motor vehicle occupants; road traffic injuries—motorcyclists; other unintentional injuries; other land transport injuries	No occupational injuries	Work-related Traumatic Injury Fatalities, Australia 2018; Workers Compensation Statistics 2016	Direct evidence: number of workplace fatalities and the number of workers compensation claims for injuries
		Occupational exposure to benzene or formaldehyde	Acute lymphoblastic leukaemia, acute myeloid leukaemia, chronic lymphocytic leukaemia, chronic myeloid leukaemia, other leukaemias, nasopharyngeal cancer	No occupational exposure to benzene or formaldehyde	Census of Population and Housing 2015; ABS Labour force survey, June 2015	Distribution of the labour force by broad industry type
		Occupational exposure to arsenic, beryllium, cadmium chromium, diesel engine exhaust, polycyclic aromatic hydrocarbons, nickel, second-hand smoke, silica	Lung cancer	No occupational exposure to arsenic, beryllium, cadmium chromium, diesel engine exhaust, polycyclic aromatic hydrocarbons, nickel, second-hand smoke, silica	Census of Population and Housing 2015; ABS Labour force survey, June 2015	Distribution of the labour force by broad industry type

(continued)



**Table F2 (continued): Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREDS, data sources and units for calculating relative risks**

Risk factor	Risk factor exposure	Risk factor measure	Disease outcome	TMRED	National data source	Units for effect size calculation
Occupational exposures and hazards (continued)		Occupational exposure to asbestos, silicone and other particulate matter	Pneumoconiosis	No occupational exposure to asbestos, silicone and other particulate matter	GBD 2016	Direct evidence
		Occupational exposure to sulphuric acid	Laryngeal cancer	No occupational exposure to sulphuric acid	Census of Population and Housing 2015; Labour force survey, June 2015	Distribution of the labour force by broad industry type
		Occupational exposure to trichloroethylene	Kidney cancer	No occupational exposure to trichloro-ethylene	Census of Population and Housing 2015; ABS Labour force survey, June 2015	Distribution of the labour force by broad industry type
		Occupational exposure to particulate matter, gas and fumes	COPD	No occupational exposure to particulate matter, gas and fumes	Census of Population and Housing 2015; ABS Labour force survey, June 2015	Distribution of the labour force by broad industry type
		Occupational asbestos	Laryngeal cancer, lung cancer, mesothelioma, ovarian cancer	No occupational exposure to asbestos	Census of Population and Housing 2015; ABS Labour force survey, June 2015	Distribution of the labour force by broad industry type

*(continued)*

**Table F2 (continued): Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREDS, data sources and units for calculating relative risks**

Risk factor	Risk factor exposure	Risk factor measure	Disease outcome	TMRED	National data source	Units for effect size calculation
Occupational exposures and hazards (continued)		Occupational exposure to noise	Hearing loss	Background noise exposure	Census of Population and Housing 2015; ABS Labour force survey, June 2015	Distribution of the labour force by broad occupational group
		Occupational asthmagens	Asthma	Background asthmagen exposure	Census of Population and Housing 2015; ABS Labour force survey, June 2015	Distribution of the labour force by broad occupational group
		Occupational ergonomic factors	Back pain and problems	No occupational exposure to ergonomic factors causing back pain and problems	Census of Population and Housing 2015; ABS Labour force survey, June 2015	Distribution of the labour force by broad occupational group
Overweight and obesity	Overweight, obese	Body mass index	Acute lymphoblastic leukaemia, acute myeloid leukaemia, asthma, atrial fibrillation and flutter, back pain and problems, bowel cancer, breast cancer, cataract & other lens disorders, chronic kidney disease, chronic lymphocytic leukaemia, chronic myeloid leukaemia, coronary heart disease, dementia, gallbladder and bile duct disease, gallbladder cancer, gout, hypertensive heart disease, kidney cancer, liver cancer, myeloma, non-Hodgkin lymphoma, oesophageal cancer, osteoarthritis, other leukaemias, ovarian cancer, pancreatic cancer, stroke, thyroid cancer, type 2 diabetes, uterine cancer	Body mass index between 20 and 25 BMI	NHS 2014–15	Per 5 BMI

*(continued)*

**Table F2 (continued): Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREDs, data sources and units for calculating relative risks**

Risk factor	Risk factor exposure	Risk factor measure	Disease outcome	TMRED	National data source	Units for effect size calculation
Physical inactivity	Physical inactivity	Metabolic equivalent of task (METs)	Breast cancer, bowel cancer, coronary heart disease, dementia, type 2 diabetes, stroke, uterine cancer	All adults experience average 8000 metabolic equivalent of task (METs) per week (highly physically active)	AHS 2011–12; NHS 2014–15	METs of less than 600, 600–3,999, 4,000–7,999
Tobacco use	Second-hand smoke	Second-hand smoke	Breast cancer, coronary heart disease, influenza, lower respiratory infections, lung cancer, otitis media, stroke, type 2 diabetes	No tobacco use	NDSHS 2016	Proportion of the population exposed to second-hand smoke
	Tobacco use	Current smoking (5-year lagged)	Age-related macular degeneration, aortic aneurysm, asthma, atrial fibrillation and flutter, back pain and problems, cataract & other lens disorders, coronary heart disease, dementia, gallbladder & biliary diseases, gastroduodenal disorders, hypertensive heart disease, lower respiratory infections, multiple sclerosis, other cardiovascular diseases peripheral vascular disease, rheumatoid arthritis, stroke, type 2 diabetes		NDSHS 2010	Proportion of the population who smoked 5 years ago
		Smoking impact ratio	Acute lymphoblastic leukaemia, acute myeloid leukaemia, bladder cancer, bowel cancer, breast cancer, cervical cancer, chronic lymphocytic leukaemia, chronic myeloid leukaemia, COPD, kidney cancer, laryngeal cancer, lip and oral cavity cancer, liver cancer, lung cancer, nasopharynx cancer, oesophageal cancer, other leukaemias, other respiratory diseases, pancreatic cancer, prostate cancer, stomach cancer		NMD	Lung cancer mortality rate; Peto et al. 1992

*(continued)*

**Table F2 (continued): Risk factors in the ABDS 2015, measures of exposure, linked diseases, TMREDs, data sources and units for calculating relative risks**

<b>Risk factor</b>	<b>Risk factor exposure</b>	<b>Risk factor measure</b>	<b>Disease outcome</b>	<b>TMRED</b>	<b>National data source</b>	<b>Units for effect size calculation</b>
Unsafe sex		Unsafe sex	Cervical cancer, chlamydia, chronic liver disease, gonorrhoea, hepatitis B, hepatitis C, HIV/AIDS, liver cancer, syphilis, other sexually transmitted infections	No unsafe sex	National notifiable disease annual surveillance reports (The Kirby Institute)	All sexually transmitted infections and cervical cancer attributed to unsafe sex  HIV/AIDS, hepatitis B and hepatitis C from direct evidence

**Table F3: Proportion and method used to align GBD relative risks with the ABDS 2015 diseases**

<b>ABDS 2011 disease</b>	<b>GBD 2013 cause</b>	<b>Source of disaggregation</b>	<b>Proportion of ABDS disease (%)</b>
Stroke	Ischaemic stroke	Thrift et al. 2009	0.776
Stroke	Haemorrhagic stroke	Thrift et al. 2009	0.224
Chronic liver disease	Chronic liver disease due to alcohol	GBD 2016	0.314 (Males) 0.266 (Females)
Liver cancer	Liver cancer due to alcohol	GBD 2016	0.395 (Males) 0.432 (Females)
Inflammatory heart disease	Endocarditis	Separations in the NHMD 2011	0.206
Osteoarthritis	Osteoarthritis of the hip	GBD 2013	0.180
Osteoarthritis	Osteoarthritis of the knee	GBD 2013	0.820
Chronic kidney disease	Diabetic chronic kidney disease	GBD 2016	0.534

# Appendix G: Additional information and tables for Chapter 9

## Measuring the quality of outputs from the ABDS

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% CIs). 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 feasible only in the case of some relatively straightforward transformations and some well-understood input distributions. That is why the 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.

**Table G1: ABDS quality index, Dimension I—Data relevance scores**

Score	Criteria
5	<p>Current data from one of the following: fully enumerated disease register (such as a cancer register) or administrative data, unlinked hospitalisation data for condition with a high likelihood of hospitalisation or national Australian survey (such as the AHS) of either (a) diagnostically confirmed conditions/sequelae or (b) established high correlation between self-report and clinical diagnosis specific to the population with no major variability due to small numbers.</p> <p>No severity distribution needed, or high-quality empirical data on this distribution were available.</p>
4	<p>Same as '5' BUT not fully enumerated with either known gaps in coverage or not diagnostically confirmed or within 2 years of the reference date or there was some variability due to small numbers (for example, a particular age group) or had high RSEs or severity not available.</p> <p>It was also used for estimates with components that scored between 5 and 3.</p>
3	<p>Same as '4' BUT with medium specificity of the data source to the condition/sequela being estimated. For example:</p> <ul style="list-style-type: none"><li>– for survey data, there was known medium correlation between what was collected (for example, measurement, self-report and clinical diagnosis) and the condition</li><li>– for hospitals data, condition had a medium likelihood of hospitalisation (that is, condition only results in hospitalisation in severe or certain cases).</li></ul> <p>Also, data were from a single, large area (more than 1 state/territory) Australian study of very good quality or from a systemic meta-analysis that could be generalised or from a review of Australian studies with medium currency.</p> <p>It was also used for estimates with components that scored between 4 and 2.</p>
2	<p>Data were from one of the following: 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 high-income countries) studies. Additionally, the data source was specific to the condition/sequela being estimated and either the 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 <b>not</b> changing over time.</p> <p>It was also used for estimates with components that scored between 3 and 1.</p>
1	<p>Data were from one of the following: 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 generalisability to the Australian context or a secondary data source for indirect prevalence estimates.</p>



**Table G2: ABDS quality index, Dimension II—Data transformation scores**

Score	Criteria
5	<p>Data were directly applied to the model and minimal or no extra modelling was required.</p> <p>Severity distribution (if required) was obtained directly from the data.</p>
4	<p>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.</p> <p>It was also used for estimates with components that scored between 5 and 3.</p>
3	<p>One of the following transformations was used: 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).</p> <p>It was also used for estimates with components that scored between 4 and 2.</p>
2	<p>One of the following transformations was used: 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, and so on; GBD global severity distribution was used.</p> <p>It was also used for estimates with components that scored between 3 and 1.</p>
1	<p>Transformations were done using one of the following: inference of distributions from other slightly related data sources; based on 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.</p>

**Table G3: National YLD quality ratings**

Disease	Data	Methods	Description
<b>Blood &amp; metabolic disorders</b>			
Cystic fibrosis	A	B	National prevalence estimates, by severity, were obtained from <i>Australian Cystic Fibrosis Data Registry annual report 2015</i> . Hospitals data were used to apportion estimates into 5-year age group for registrants over 65 years.
Haemophilia	A	B	National prevalence estimates by severity were obtained from <i>Australian Bleeding Disorders Registry annual report 2015–2016</i> . 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.
Haemolytic anaemias	A	B	National prevalence estimates by sequela were obtained from the NHMD with a high likelihood of hospitalisation. People-to-separations ratios derived from the linked hospitals and NDI components of the NDLDP database were applied to unlinked hospitalisation data to estimate prevalence.
Iron-deficiency anaemia	B	C	National prevalence estimates for the anaemia envelope, by severity, were obtained from the NHS 2014–15. Transformations were required by using the biomedical and survey data from the AHS 2011–12 since biomedical information was not available in the NHS 2014–15, as well as 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.
Protein-energy deficiency	A	B	National prevalence estimates, by sequela, in elderly Australians was obtained from an Australian community-living based study assessing malnutrition (Rist et al. 2012). Protein-energy deficiency in children was estimated for the Indigenous population only, and obtained from the National Aboriginal and Torres Strait Islander Health Survey 2014–15.  Minimal transformations were required to determine severity distributions specific to remoteness category, which were then summed to provide a national estimate.
Other blood and metabolic disorders	C	A	National 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.
<b>Cancer &amp; other neoplasms</b>			
Laryngeal cancer	A	A	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 20-year prevalence of laryngeal cancer.
Oesophageal cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Stomach cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Bowel cancer	A	A	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 20-year prevalence of survivors.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Cancer &amp; other neoplasms (continued)</b>			
Liver cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Gallbladder cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Pancreatic cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Lung cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Mesothelioma	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Melanoma of the skin	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Non-melanoma skin cancer	B	A	Prevalence of metastatic and terminal sequelae were derived directly from the 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.
Breast cancer	A	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 20-year prevalence of breast cancer, by age and sex.
Cervical cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Uterine cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Ovarian cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Prostate cancer	A	B	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 20-year prevalence of prostate cancer.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Cancer &amp; other neoplasms (continued)</b>			
Testicular cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Bladder cancer	A	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 20-year prevalence of bladder cancer, with rates of incontinence as derived from Gilbert and others (2007).
Kidney cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Brain and central nervous system cancer	A	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.
Thyroid cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Non-Hodgkin lymphoma	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Hodgkin lymphoma	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Myeloma	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Other blood cancers	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Unknown primary	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Cancer &amp; other neoplasms (continued)</b>			
Benign and uncertain brain tumours	E	E	Diagnosis/treatment and controlled phases were derived directly from the ACD for Victoria, Queensland and Western Australia, and incidence-to-separation ratios 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 the estimated lifetime prevalence of benign brain tumours from malignant-to-benign ratios based on the study by Porter and others (2010).
Ductal carcinoma in situ (breast)	A	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.
Lip and oral cavity cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Nasopharyngeal cancer	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Other lip, oral cavity and pharynx cancers	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Acute myeloid leukaemia (AML)	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Chronic myeloid leukaemia (CML)	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Acute lymphoblastic leukaemia (ALL)	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Chronic lymphocytic leukaemia (CLL)	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Other leukaemias	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.
Other malignant neoplasms (cancers)	A	A	Diagnosis/treatment and controlled phases were derived directly from the ACD. Metastatic and terminal phases were derived directly from the NMD.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Cancer and other neoplasms (continued)</b>			
Other benign, in situ and uncertain neoplasms	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 the from the ABDS 2003. Metastatic and terminal phases were derived directly from the NMD.
<b>Cardiovascular diseases</b>			
Coronary heart disease	B	C	National 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.
Stroke	B	C	National 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.
Rheumatic heart disease	B	C	National 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 for the chronic sequela from unlinked hospitalisation data into prevalence. Ratios from the New South Wales and Victorian linked hospitalisations and deaths data were used to transform estimates for the acute sequela from unlinked hospitalisation data into prevalence.
Non-rheumatic valvular disease	B	C	National 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.
Hypertensive heart disease	B	C	National 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.
Atrial fibrillation and flutter	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.
Inflammatory heart disease	B	C	National 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.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Cardiovascular diseases (continued)</b>			
Cardiomyopathy	B	C	National 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.
Aortic aneurysm	A	A	National prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation.
Peripheral vascular disease	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.
<b>Endocrine disorders</b>			
Type 1 diabetes	C	B	National prevalence estimates were obtained from the linked NDSS and APEG data set. 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.
Type 2 diabetes	C	C	National prevalence estimates were obtained from the NHS 2014–15. 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.
Other diabetes	D	C	Overall prevalence was derived from the proportion of other diabetes from the Fremantle Diabetes Study. The sex and age group distribution were modelled using the NHMD.
<b>Gastrointestinal disorders</b>			
Gastroduodenal disorders	C	C	Prevalence based on hospital separations were adjusted for physician-diagnosed disease (Sung et al. 2009). Anaemia estimates were derived from the NHMD. Severity distribution was obtained from the GBD 2013.
Appendicitis	A	A	Derived directly from hospital separations with a high likelihood of hospitalisation adjusted for duration.
Abdominal wall hernia	A	A	Derived directly from hospital separations with a high likelihood of hospitalisation adjusted for duration.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Gastrointestinal disorders (continued)</b>			
Vascular disorders of intestine	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.
Intestinal obstruction (without hernia)	A	A	Derived directly from hospital separations with a high likelihood of hospitalisation, adjusted for duration.
Inflammatory bowel disease	D	C	Rates from a Sydney study (Selinger et al. 2013) applied to national populations.
Diverticulitis	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.
Chronic liver disease	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 hospitals and NDI components of the NDLD database applied to unlinked hospitalisation data. Prevalence of liver transplant patients was based on data from the Australia and New Zealand Liver Transplant Registry.
Gallbladder and bile duct disease	A	A	Derived directly from hospital separations with a high likelihood of hospitalisation adjusted for duration.
Pancreatitis	A	A	Derived directly from hospital separations with a medium–high likelihood of hospitalisation adjusted for duration.
Gastro oesophageal reflux disease	C	D	Estimates for moderate/severe gastro-oesophageal reflux disease for people seeking medical attention were based on the study by Harrison and others (2013).
Functional gastrointestinal disorders	D	C	Derived from small area study in Penrith, New South Wales (Boyce et al. 2006), and applied to populations.
<b>Hearing &amp; vision disorders</b>			
Hearing loss	B	C	National prevalence estimates were based on estimates from Australian Hearing annual reports, the NHS 2014–15, 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 the GBD 2010 for high-income countries.

*(continued)*



**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Hearing &amp; vision disorders (continued)</b>			
Refractive errors	B	D	National prevalence estimates were based on estimates from the National Eye Health Survey 2016, which covers majority of people with this condition but not all. Some transformations were required to calculate estimates by age and severity using the ABDS 2003 estimates and data from the Melbourne Visual Impairment Project (Taylor et al. 2005; Weih et al. 2000), a small Australian study with data more than 5 years from the reference date.
Cataract	C	C	National prevalence estimates for mild and moderate cases were based on the National Eye Health Survey 2016 data, which covers majority of people with this condition but not all. For severe and blind cases, national prevalence estimates were based on the Melbourne Visual Impairment Project (Taylor et al. 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, the ABDS 2003 estimates, or expert advice, when required.
Glaucoma	D	B	National prevalence estimates, by cause of vision loss, were based on estimates from the Melbourne Visual Impairment Project (Taylor et al. 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, the ABDS 2003 estimates, or expert advice when, required.
Age-related macular degeneration	D	B	National prevalence estimates, by cause of vision loss, were based on estimates from the Melbourne Visual Impairment Project (Taylor et al. 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, the ABDS 2003 estimates, or expert advice when, required.
Other vision disorders	D	B	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, the ABDS 2003 estimates, or expert advice, when required.
Other hearing and vestibular disorders	B	C	National prevalence estimates, by sequela, were based on estimates from the NHS 2014–15. Moderate transformations were required to obtain age- and sex-specific distributions using the NHMD or population data.
<b>Infant &amp; congenital conditions</b>			
Pre-term birth and low birthweight complications	D	D	National prevalence for acute complications was derived from the National Perinatal Data Collection. National prevalence estimates for long-term sequelae were derived from intellectual disability envelope.
Birth trauma and asphyxia	D	E	National prevalence estimates were derived from the intellectual disability envelope. The severity distribution for birth trauma and asphyxia was derived from the NHMD.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Infant &amp; congenital conditions (continued)</b>			
Cerebral palsy	B	C	Incidence for cerebral palsy was estimated from the Australia Cerebral Palsy Register. Prevalence estimates (1913–2015) were adjusted for standard background mortality using the Australian life table (ABS 2017e), 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 2016).
Neonatal infections	A	A	National prevalence estimates were obtained directly from the NHMD, and applied a 4-week duration.
Other disorders of infancy	A	A	National prevalence estimates were obtained directly from the NHMD, and applied a 4-week duration.
Neural tube defects	C	D	Live birth prevalence rates were derived from WARDA data 2013–2015. 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).
Cardiovascular defects	C	C	The acute sequela (cardiovascular defects before surgery) was derived from the WARDA with a duration of 1 year. The chronic sequela (heart failure due to congenital cardiovascular defects) was modelled under the heart failure envelope. National 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.
Cleft lip and/or palate	C	C	Live birth prevalence rates were derived from the WARDA data 1980–2015. The prevalence rate for a given age in 2015 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	C	D	Prevalence in babies aged under 1 year was sourced directly from the live birth prevalence rate derived from the 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 2012 (ICBDSR 2014). It was assumed 44.6% people with anorectal malformations experience faecal incontinence (Stenström et al. 2014).
Urogenital malformations	C	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.
Down syndrome	D	E	National 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.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Infant &amp; congenital conditions (continued)</b>			
Brain malformations	D	D	National 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	National prevalence estimates were derived from the intellectual disability envelope.
<b>Infectious diseases</b>			
HIV/AIDS	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.
Tuberculosis	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.
Syphilis	C	D	Incidence was based on notifications to the NNDSS, with some adjustment for under-notification. Minimal modelling was required.
Chlamydia	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. Infertility sequela was based on published estimates (Hafner & Pelzer 2011), expert advice and GBD 2013 proportions.
Gonorrhoea	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. Infertility sequela was based on published estimates (Hafner & Pelzer 2011), expert advice and GBD 2013 proportions.
Other sexually transmitted infections	C	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). Infertility sequela was based on published estimates (Hafner & Pelzer 2011), expert advice and GBD 2013 proportions.
Hepatitis A	B	C	Estimates were based on notifications to the NNDSS. As these are thought to underestimate hepatitis A incidence, these estimates were adjusted for under-notification.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Infectious diseases (continued)</b>			
Hepatitis B (acute)	C	C	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).
Hepatitis C (acute)	C	C	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.
Upper respiratory infections	C	C	Estimates were based on encounters with GPs 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 GP. Some transformations were required for BEACH data.
Otitis media	C	C	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 for BEACH data.
Lower respiratory infections	D	B	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. Some transformations were required for BEACH data.
Influenza	D	B	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 for BEACH data and to overcome variability across age groups.
Diphtheria	A	A	Estimates were based on the number of NNDSS notifications for diphtheria, which is likely to capture all cases of toxigenic diphtheria.
Pertussis	C	D	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.
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.
Measles	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.
Rubella	C	D	Estimates were based on notifications to the NNDSS, which is known to have issues with under-notification.
<i>Haemophilus influenzae</i> type-b	A	A	Estimates were obtained using notifications to the NNDSS, which is thought to capture all cases of <i>Haemophilus influenzae</i> type b.
Pneumococcal disease	A	A	Estimates were obtained using notifications to the NNDSS, which provides a good prevalence estimate of invasive pneumococcal disease. Minimal modelling was required.

(continued)

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Infectious diseases (continued)</b>			
Meningococcal disease	A	A	Estimates were obtained using notifications to the NNDSS, which are thought to capture most severe cases.
Other meningitis and encephalitis	A	A	Person-to-separations ratios derived from linked hospitals components of the NDLDP database applied to unlinked hospitalisation data to estimate prevalence.
Dengue	B	B	Estimates were obtained using notifications to the NNDSS and hospitalisations. Hospitalisations were adjusted using persons-to-separations ratios.
Ross River virus	C	D	Estimates were obtained using notifications to the NNDSS (which has issues with both false positivity and under-notification) and hospitalisations.
Barmah Forest virus	C	D	Estimates were obtained using notifications to the NNDSS, which has issues with both false positivity and under-notification.
Malaria	B	B	Estimates were obtained using notifications to the NNDSS and hospitalisations.
Trachoma	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, the ABDS 2003 estimates, and expert advice.
Campylobacteriosis	B	B	Total incidence was based on estimates produced by Kirk and others (2014). Some transformations were required to fill gaps in age, sex and severity using NNDSS notifications and hospitalisations.
Salmonellosis	B	B	Total incidence was based on estimates produced by Kirk and others (2014). Some transformations were required to fill gaps in age, sex and severity using NNDSS notifications and hospitalisations.
Rotavirus	C	D	Total incidence was based on estimates produced by Kirk and others (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 people-to-separations ratios.
Other gastrointestinal infections	B	D	Total incidence was based on estimates produced by Kirk and others (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).

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Infectious diseases (continued)</b>			
Varicella	C	C	Estimates were based on GP encounters reported in the BEACH data. Moderate transformations were required to overcome known data gaps.
Herpes zoster	C	B	Estimates were based on GP encounters reported in the BEACH data. Moderate transformations were required to overcome known data gaps for herpes zoster and post-herpetic neuralgia.
Mumps	A	B	Estimates were obtained using notifications to the NNDSS and hospitalisations.
Urinary tract infections	C	C	Estimates of moderate cases were based on GP encounters 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 and for BEACH data.
<b>Injuries</b>			
Traumatic brain injury	A	D	Short-term prevalence was estimated from the NHMD, and adjusted to account for non-admitted cases, based on estimates from the National Non-admitted Patient Emergency Department Care Database (NNAPEDCD). Long-term estimates were modelled in DISMOD II using GBD 2013 estimates for remission and mortality risk (see Haagsma et al. 2016).
Spinal cord injury	A	D	Short-term prevalence was estimated from the NHMD, and adjusted to account for non-admitted cases, based on estimates from the NNAPEDCD. Severity distribution was based on a customised report from Monash University (see Monash University, unpublished). Long-term estimates were modelled in DISMOD II using GBD 2013 estimates for remission and mortality risk (see Haagsma et al. 2016).
Internal and crush injury	A	D	Short-term prevalence was estimated from the NHMD, and adjusted to account for non-admitted cases, based on estimates from the NNAPEDCD. Long-term estimates were modelled in DISMOD II using GBD 2013 estimates for remission and mortality risk (see Haagsma et al. 2016).
Poisoning	A	D	Short-term prevalence was estimated from the NHMD, and adjusted to account for non-admitted cases, based on estimates from the NNAPEDCD. Long-term estimates were modelled in DISMOD II using GBD 2013 estimates for remission and mortality risk (see Haagsma et al. 2016).
Drowning and submersion injuries	A	D	Short-term prevalence was estimated from the NHMD, and adjusted to account for non-admitted cases based on estimates from the NNAPEDCD. Long-term estimates were modelled in DISMOD II using GBD 2013 estimates for remission and mortality risk (see Haagsma et al. 2016).

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Injuries (continued)</b>			
Hip fracture	A	D	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from the NNAPEDCD. Long-term estimates were modelled in DISMOD II using GBD 2013 estimates for remission and mortality risk (see Haagsma et al. 2016).
Tibia and ankle fracture	C	D	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from the NNAPEDCD. Long-term estimates were modelled in DISMOD II using GBD 2013 estimates for remission and mortality risk (see Haagsma et al. 2016).
Humerus fracture	B	D	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from the NNAPEDCD. Long-term estimates were modelled in DISMOD II using GBD 2013 estimates for remission and mortality risk (see Haagsma et al. 2016).
Other fractures	B	D	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from the NNAPEDCD. Long-term estimates were modelled in DISMOD II using GBD 2013 estimates for remission and mortality risk (see Haagsma et al. 2016).
Dislocations	C	B	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from the NNAPEDCD.
Soft tissue injuries	C	B	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from the NNAPEDCD.
Burn injuries	A	D	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from the NNAPEDCD. Long-term estimates were modelled in DISMOD II using estimates for remission from Duke and others (2015).
Other injuries	B	B	Short-term prevalence was estimated from the NHMD and adjusted to account for non-admitted cases based on estimates from the NNAPEDCD. Long-term estimates were modelled in DISMOD II using GBD 2013 estimates for remission and mortality risk (see Haagsma et al. 2016).
<b>Kidney and urinary diseases</b>			
Chronic kidney disease	B	B	National prevalence estimates were obtained from the Australia and New Zealand Dialysis and Transplant Registry and the AHS 2011–12. Moderate transformations of the AHS were required to obtain estimates for 2015 using AIHW analysis of the AHS 2011–12 compared with AusDiab 1999–2000 and 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.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Kidney and urinary diseases (continued)</b>			
Enlarged prostate	B	B	National prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation for cases with sufficient severity. People-to-separations ratios derived from Western Australian linked hospitalisations 2011 data were applied to unlinked hospitalisation data to estimate prevalence.
Kidney stones	A	A	National prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation for cases with sufficient severity.
Interstitial nephritis	C	C	National prevalence estimates for severe cases were obtained from the NHMD with a high likelihood of hospitalisation. As the amount non-hospitalised cases was not directly available, inflation factors based on the GBD 2016 global severity distributions were used to determine non-hospitalised cases.
<b>Mental and substance use disorders</b>			
Depressive disorders	B	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 the ones used for the GBD 2013. Moderate transformations were required to overcome gaps in age distribution, and to adjust to point prevalence.
Anxiety disorders	B	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 the ones used for the GBD 2013. Moderate transformations were required to overcome gaps in age distribution, and to adjust to point prevalence.
Bipolar affective disorder	B	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 the ones used for the GBD 2013. Moderate transformations were required to overcome gaps in age distribution, and to adjust to point prevalence.
Alcohol use disorders	B	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 the GBD 2013. Some transformations were required to overcome gaps in age distribution.
Drug use disorders (excluding alcohol)	C	C	National estimates were obtained from a variety of sources depending on the drug. These varied in quality from cannabis and cocaine dependence estimates—which were based on the GBD 2015 estimates—to estimates of amphetamine dependence—which were based on proxy measures. All severity distributions were from the GBD 2013. Moderate transformations were required to overcome data gaps.
Schizophrenia	B	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 the ones developed by Ferrari and others (2012).

*(continued)*



**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Mental and substance use disorders (continued)</b>			
Eating disorders	D	C	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.
Autism spectrum disorders	D	D	National estimates were based on prevalence in Western Australia, and were based on administrative data that would reasonably capture most cases of childhood autism. Substantial transformations were required to overcome data gaps.
Attention deficit hyperactivity disorder	B	C	National estimates were obtained from the 2013–14 Young Minds Matter survey, based on diagnostic criteria and the GBD 2013. Moderate transformations were required to calculate prevalence.
Conduct disorder	B	C	Estimates were obtained from the 2013–14 Young Minds Matter survey based on diagnostic criteria and the GBD 2013. Moderate transformations were required to calculate prevalence.
Intellectual disability	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.
Other mental and substance use disorders	C	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.
<b>Musculoskeletal conditions</b>			
Osteoarthritis	B	B	National prevalence estimates were obtained from the NHS 2014–15. Severity distributions were obtained from the same data source. Moderate transformations were required to overcome data gaps.
Gout	B	B	National prevalence estimates were obtained from the NHS 2014–15. Moderate transformations were required to overcome data gaps.
Rheumatoid arthritis	B	B	National prevalence estimates were obtained from the NHS 2014–15. Severity distributions were obtained from the same data source. Moderate transformations were required to overcome data gaps.
Back pain and problems	B	B	National prevalence estimates were obtained from the NHS 2014–15. Severity distributions were obtained from the same data source. Moderate transformations were required to overcome data gaps.
Other musculoskeletal	B	B	National prevalence estimates were obtained from the NHS 2014–15. Severity distributions were obtained from the same data source. Moderate transformations were required to overcome data gaps.
<b>Neurological conditions</b>			
Epilepsy	B	C	National prevalence estimates were obtained from the NHS 2014–15. People-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	National prevalence estimates were obtained from AIHW 2012c. Severity distribution was obtained from Lucca and others (2015) and Barendregt and others (1998). Moderate transformations were required to produce prevalence estimates by severity.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Neurological conditions (continued)</b>			
Parkinson disease	E	C	National 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.
Multiple sclerosis	C	C	National prevalence estimates were obtained from a current Australian study (Menzies Health Research Group, van der Mei & Taylor 2018). Severity distribution was obtained from another Australian study (Covance Pty Ltd & Palmer 2011). Some transformations were required to produce accurate estimates for the reference year 2015.
Motor neurone disease	B	B	National prevalence estimates were obtained from the NHMD with a moderate likelihood of hospitalisation. People-to-separations ratios derived from New South Wales and Victoria linked hospitalisations and deaths data were applied to unlinked hospitalisation data to estimate prevalence.
Migraine	B	B	National prevalence estimates were obtained from the NHS 2014–15. 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	National prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation. People-to-separations ratios derived from New South Wales and Victoria linked hospitalisations and deaths data were applied to unlinked hospitalisation data to estimate prevalence.
<b>Oral disorders</b>			
Dental caries	D	C	National estimates were obtained from the National Survey of Adult Oral Health 2004–06 for adults, the 2009 Child Dental Health Survey for children and the National Child Oral Health Study 2012–14. These 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.
Periodontal disease	D	C	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.
Severe tooth loss	D	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.
Other oral disorders	C	B	National estimates were based on hospitalisations, which has medium likelihood of hospitalisation. Some transformations were required to overcome gaps in age/sex distribution.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Reproductive and maternal conditions</b>			
Maternal haemorrhage	A	A	National prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation.
Maternal infections	A	A	National prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation.
Hypertensive disorders of pregnancy	A	A	National prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation.
Obstructed labour	A	A	National prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation.
Early pregnancy loss	A	C	National prevalence estimates for both sequelae were obtained from the NHMD with a high likelihood of hospitalisation for ectopic pregnancy. Estimates for ectopic pregnancy were inflated to account for non-admitted cases (Goller et al. 2018). Estimates for early pregnancy losses occurring outside hospital were calculated from Medicare data, and adjusted for unclaimed procedures using an Australian study (Shankar et al. 2017). Early pregnancy loss estimates for Queensland were based directly on data obtained from Queensland Health. Pharmaceutical Benefits Scheme (PBS) data was used for early pregnancy loss using MS-2 Step pharmaceuticals.
Gestational diabetes	A	C	National 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.
Endometriosis	C	D	Overall national estimates were derived from the Australian Longitudinal Study on Women's Health and transformed using BEACH data. National 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. Infertility sequela for the national population was derived from the Australian Longitudinal Study on Women's Health.
Uterine fibroids	C	C	National 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.  Anaemia sequela was based on an international study (Zimmerman et al. 2012). Severity distribution was the same as used for iron-deficiency anaemia. Infertility sequela was based on international estimates (Khaund & Lumsden 2008).
Genital prolapse	D	C	National prevalence estimates for females were obtained from rates from the NZBDS and from 2 international studies (Harvie et al. 2018; Lawrence et al. 2008). 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.
Polycystic ovarian syndrome	B	C	National prevalence estimates were derived from the Australian Longitudinal Study on Women's Health and transformed using BEACH data. Estimates were removed from the total prevalence to derive remaining sequelae.  Infertility sequela was derived from the Australian Longitudinal Study of Women's Health.

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Reproductive and maternal conditions (continued)</b>			
Infertility	D	D	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.
Other maternal conditions	A	A	National prevalence estimates were obtained from the NHMD with a high likelihood of hospitalisation.
Other reproductive conditions	D	C	National prevalence estimates, by sequela, were based on BEACH data, with moderate transformations using population data.
<b>Respiratory diseases</b>			
Asthma	B	C	National prevalence estimates were obtained from the NHS 2014–15. Severity distributions were obtained from an Australian web-based survey (Reddel et al. 2015). Moderate transformations were required to overcome data gaps.
Chronic obstructive pulmonary disease	C	C	National prevalence estimates were based on measured data from a study specific to the Australian population—the BOLD study (Toelle et al. 2013). Prevalence rates from 2011 were applied to the 2015 population. Severity distributions were also obtained from the BOLD study, together with expert advice. Some transformations were required to overcome variation caused by small numbers.
Sarcoidosis	C	D	National prevalence estimates were obtained from the NMD and the NHMD. As these data sources captured only moderate/severe cases, transformations were required to fill gaps due to low data specificity. People-to-separations ratios derived from Western Australian linked hospitalisations and deaths data from the ABDS 2011 were applied to unlinked hospitalisation data to estimate prevalence. Severity distributions were obtained from the GBD 2015.
Interstitial lung disease	C	D	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 from the ABDS 2011 were applied to unlinked hospitalisation data to estimate prevalence. Severity distributions were obtained from the GBD 2015.
Pneumoconiosis	C	D	National prevalence estimates were obtained from the NMD and the NHMD. As these data sources captured only moderate/severe cases, transformations were required to fill gaps due to low data specificity. People-to-separations ratios derived from Western Australian linked hospitalisations and deaths data from the ABDS 2011 were applied to unlinked hospitalisation data to estimate prevalence. Severity distributions were obtained from the GBD 2015.
Upper respiratory conditions	B	C	National prevalence estimates were obtained from the NHS 2014–15. Moderate transformations were required to overcome data gaps. Health loss was assigned to 33% of cases based on findings from allergic rhinitis studies in the United States of America and Australia (Meltzer et al. 2012; Tan et al. 2017).

*(continued)*

**Table G3 (continued): National YLD quality ratings**

Disease	Data	Methods	Description
<b>Skin disorders</b>			
Dermatitis and eczema	D	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 and others (1999a).
Psoriasis	D	B	National prevalence estimates were obtained from the NHS 2014–15. 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.
Acne	D	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).
Ulcers	C	C	National prevalence estimates and severity distributions were obtained from the NHMD, and Australian and international studies (Asimus & Li 2011; CEC 2017; Dealey et al. 2012; Edwards et al. 2017; Mulligan et al. 2011; Queensland Health 2012; SA Health 2007; Santamaria et al. 2009; VQC 2006). Substantial transformations were required to overcome data gaps.
Skin infections (including cellulitis)	C	B	National prevalence estimates were obtained from the NHMD with a moderate likelihood of hospitalisation. Some transformations were required to apply a duration of 2 weeks to estimate the point prevalence.
Other skin disorders	D	D	National prevalence estimates were obtained from the NHMD and the NHS 2014–15. 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 G4: National risk factor ratings**

Risk factor	Data	Methods	Description
Air pollution	D	D	Estimate from ground-based monitoring stations located in populated areas.
Alcohol use	B	B	National exposure estimates were obtained from the NDSHS 2016. The NDSHS data were adjusted for under-reporting of alcohol consumption by estimates of alcohol available for sale in Australia. The direct PAF for accidental poisoning were estimated from the NMD 2015. Alcohol dependency burden were estimated from the prevalence of alcohol use disorders in the ABDS 2015.
Childhood abuse & neglect	C	..	Data were modelled from a range of studies. Information on the quality of these estimates are published in the paper by Moore and others (2015).
Dietary risks	B	B	National exposure estimates were obtained from the AHS 2011–12 modelled to 2015 based on trends
High blood plasma glucose	C	C	National exposure estimates to high plasma glucose were obtained from the 2011–12 AHS, no data was available to inform trends. Exposure to diabetes was estimated by the prevalence for the ABDS 2015 causes type 1, type 2 and other diabetes.
High blood pressure	A	A	National exposure estimates were obtained from the NHS 2014–15.
High cholesterol	B	B	National exposure estimates were obtained from the AHS 2011–12 modelled to 2015 based on trends.
High sun exposure	C	..	Australian appropriate estimate for melanoma was based on the range estimate from the global study on the burden of disease from solar ultraviolet radiation (Lucas et al. 2006). No distribution by age and sex of over time was available.
Illicit drug use	B	C	Direct evidence PAFs for unsafe injecting practices were obtained from the National Notifiable Diseases Surveillance System 2015. Exposure to driving under the influence of drugs was obtained from the NDSHS 2016; however, the type of drug used was approximated. Drug dependency were estimated from the prevalence to relevant drug use disorders estimated in the ABDS 2015. Accidental poisoning PAFs were estimated from the NMD 2015.
Impaired kidney function	B	B	National exposure estimates were obtained from the AHS 2011–12 modelled to 2015 based on trends.
Intimate partner violence	B	B	National exposure estimates were obtained from the Personal Safety Survey 2016. Direct evidence from the National homicide monitoring program and the NHMD 2015.
Iron deficiency	B	C	No exposure or direct PAF estimated. Quality is reflected by the quality of the ABDS 2015 cause iron deficiency anaemia.
Low bone mineral density	E	C	Exposure was estimated from the Geelong osteoporosis study and the data more than 5 years from the reference year; with no known trend.
Occupational exposures and hazards	B	C	Exposure to working in categories of occupations and industries were estimated from the ABS Labour Force Survey and ABS Census of Housing for linked diseases. Exposure was estimate from data collected by Safe Work Australia estimates mapped to the ABDS 2015 causes.
Overweight and obesity	A	A	National exposure estimates were obtained from the NHS 2014–15.
Physical inactivity	A	B	National exposure estimates were estimated from the NHS 2014–15 for activity for leisure and activity from transport and occupation were from the AHS 2011–12. Household chores were obtained from the 2006 ABS time use survey.
Tobacco use	B	B	National exposure estimates were obtained from the National Drug Strategy Household Survey (NDSHS) 2010 to include a 5-year lag. Lung cancer mortality rates were based on national cancer mortality data.

*(continued)*

**Table G4 (continued): National risk factor ratings**

Risk factor	Data	Methods	Description
Unsafe sex	B	C	Direct evidence PAFs for unsafe injecting practices were obtained from the National Notifiable Diseases Surveillance System 2015 and no disaggregation was available by age.

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 H: List of expert advisors

**Table H1: List of the ABDS 2015 Expert Advisory Group members**

<b>Member</b>	<b>Organisation</b>
Ching Choi (Chair)	University of New South Wales
Anthony Barnes	Independent consultant
Antony Blakely	University of Otago/ University of Melbourne
Justine Boland	Australian Bureau of Statistics
Annette Dobson	University of Queensland
Tim Driscoll	University of Sydney
Sonya Glasson	Australian Government Department of Health
John Goss	University of Canberra
Richard Juckes	Australian Institute of Health and Welfare
Paul Kelly	ACT Health
Laura Kirkland	Department of Health Western Australia
Maarit Laaksonen	University of New South Wales
Lynelle Moon	Australian Institute of Health and Welfare
Helen Moore	NSW Ministry of Health
Bernie Towler	Australian Government Department of Health
Harvey Whiteford	University of Queensland
Jeanette Young	Queensland Health



**Table H2: List of the ABDS disease-specific advisors**

<b>Expert (group or person)</b>	<b>Organisation</b>
<b>Blood &amp; metabolic disorders</b>	
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
<b>Cancer and other neoplasms</b>	
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
<b>Cardiovascular diseases</b>	
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
Dr Judith Katzenellenbogen	University of Western Australia
<b>Endocrine disorders</b>	
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
<b>Gastrointestinal disorders</b>	
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

*(continued)*

**Table H2 (continued): List of the ABDS disease-specific advisors**

<b>Expert (group or person)</b>	<b>Organisation</b>
<b>Gastrointestinal disorders (continued)</b>	
Dr Stephen Williams	Westmead Hospital
Prof. Rupert Leong	University of New South Wales
Prof. Geoff McCaughan	Centenary Institute
<b>Hearing and visions disorders</b>	
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
<b>Infant &amp; congenital conditions</b>	
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
<b>Infectious diseases</b>	
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
Prof Brett Mitchell	Avondale College of Higher Education
<b>Injuries</b>	
Prof. James Harrison	Research Centre for Injury Studies, Flinders University
Dr Sophie Pointer	Research Centre for Injury Studies, Flinders University

*(continued)*

**Table H2 (continued): List of the ABDS disease-specific advisors**

<b>Expert (group or person)</b>	<b>Organisation</b>
<b>Injuries (continued)</b>	
Prof. Belinda Gabbe	School of Public Health and Preventive Medicine, Monash University
<b>Kidney and urinary diseases</b>	
Cardiovascular, Diabetes and Kidney Unit	Australian Institute of Health and Welfare
Chronic Kidney Disease Expert Advisory Group— Steven Chadban (Chair), Jeremy Chapman, Bettina Douglas, Stephen McDonald, David Parker	Australian Institute of Health and Welfare advisory group
<b>Mental and substance use disorders</b>	
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
<b>Musculoskeletal conditions</b>	
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
<b>Neurological conditions</b>	
Disability and Ageing Unit	Australian Institute of Health and Welfare
Prof. Kaarin Anstey	Ageing Futures Institute, University of New South Wales
Prof. George Mellick	Griffith University
Prof. Matthew Kiernan	Brain and Mind Research Institute, University of Sydney
Prof. Andrew Palmer	University of Tasmania

*(continued)*

**Table H2 (continued): List of the ABDS disease-specific advisors**

<b>Expert (group or person)</b>	<b>Organisation</b>
<b>Oral disorders</b>	
Assoc. Prof. David Brennan	Australian Research Centre for Population Oral Health
Adjunct Assoc. Prof. Ratilal Laloo	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
<b>Reproductive and maternal conditions</b>	
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
Ms Jane Goller	University of Melbourne
<b>Respiratory diseases</b>	
National Asthma and Other Chronic Respiratory Conditions Monitoring Advisory Group, Chaired by Prof Christine Jenkins	Australian Institute of Health and Welfare advisory group
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
<b>Skin disorders</b>	
Indigenous Modelling & Research Unit	Australian Institute of Health and Welfare
Dr Pamela Brown	Consultant dermatologist
Dr Keng Chen	Skin and Cancer Foundation
Dr Suzanne Kapp	La Trobe University
Dr Monique Kilkeny	Monash University
Dr Rosana Norman	Queensland University of Technology

**Table H3: List of the ABDS risk-specific advisors**

<b>Expert (group or person)</b>	<b>Organisation</b>
Mr Paul Atyeo	ABS
Prof Emily Banks	Australian National University
Cardiovascular, Diabetes and Kidney Unit	AIHW
Chronic Kidney Disease Expert Advisory Group	AIHW advisory group
Prof. Tim Driscoll	Sydney School of Public Health, University of Sydney
Family, Domestic & Sexual Violence Unit	AIHW
Ms Louise Gates	ABS
Assoc. Prof. John Goss	University of Canberra
Ms Tracy Hambridge	Food Standards Australia and New Zealand
Dr Ivan Hanigan	Australian National University
Prof David Johnson	Primary Care Education Advisory Committee for Kidney Health Australia (PEAK)
Dr Grace Joshy	Australian National University
Prof. Amanda Lee	School of Public Health and Social Work and School of Exercise and Nutrition Science, Queensland University of Technology
Prof. Robyn Lucas	National Centre for Epidemiology and Population Health, Australian National University
Prof Dorothea Mackeras	Food Standards Australia and New Zealand
Assoc. Prof. Gavin Pereira	Curtin University
Dr Sarah Perkins-Kirkpatrick	University of New South Wales
Population Health Unit	AIHW
Dr Rosemary Stanton	Nutritionist consultant
Tobacco, Alcohol and Other Drugs Unit	AIHW
Dr Fan Xiang	National Centre for Epidemiology and Population Health, Australian National University
Dr Zhiwei Xu	Queensland University of Technology

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Disease group methods development and analysis were undertaken in consultation with disease experts by:

- Karen Bishop (injuries)
- Melanie Dunford (blood & metabolic disorders, infectious diseases, hearing & vision disorders, reproductive & maternal conditions, kidney & urinary diseases, gastrointestinal disorders)
- Julianne Garcia (cardiovascular diseases, endocrine disorders, mental & substance use disorders, neurological conditions)
- Melissa Goodwin (cancer & other neoplasms, gastrointestinal disorders)
- Michelle Gourley (mental & substance use disorders)
- Wendy Ho (oral disorders, infant & congenital conditions)
- Alise Kha (hearing & vision disorders, gastrointestinal disorders)
- Paula Laws (reproductive & maternal conditions, infant & congenital conditions)
- Vanessa Prescott (kidney & urinary diseases)
- Anna Reynolds (musculoskeletal conditions, respiratory diseases)
- Chenkun Zhao (neurological conditions, skin disorders).

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# Abbreviations

ABDS	Australian Burden of Disease Study
ABS	Australian Bureau of Statistics
ACD	Australian Cancer Database
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 survey
BOLD	Burden of Obstructive Lung Disease
CI	confidence interval
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
GORD	gastro-oesophageal reflux disease
GP	general practitioner
Hib	<i>Haemophilus influenzae</i> 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
IRSD	Index of Relative Socioeconomic Disadvantage
MCOD	multiple causes of death
MET	metabolic equivalent of tasks
mmHg	millimetre of mercury
mmol	millimole

NDLDP	National Data Linkage Demonstration Project
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
YLD	years lived with disability
YLL	years of life lost

## Symbols

>	greater than
<	less than
—	nil or rounded to zero
..	not applicable
n.a.	not available



# 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 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 event 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.

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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 population for 2015, 2011 and 2003, as well as estimates of how much of the burden can be attributed to various risk factors. This report is targeted at those seeking to further understand results provided in the Australian Burden of Disease Study 2015.

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