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Australian Burden of Disease Study: Methods and supplementary material 2018

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About

Burden of disease analysis aims to quantify health loss for all health outcomes, both fatal and non-fatal, and attribute it to a disease or injury category. This is achieved by separately estimating the fatal (YLL) and non-fatal (YLD) burden, according to a defined list of diseases, and summing them. This burden can then be attributed to risk factors selected for inclusion in that part of the analysis.

Detailed methods for estimating each component of burden of disease analysis for the Australian Burden of Disease Study 2018 are described in this report.

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In this report

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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) 2018 to quantify the fatal and non-fatal effects and causes of diseases and injuries in the Australian population in 2018, 2015, 2011 and 2003; and the Indigenous Australian population in 2018, 2011 and 2003.

It was developed to provide transparency of data, assumptions and methods and is a companion publication to:

- *Australian Burden of Disease Study 2018—key findings* and
- *Australian Burden of Disease Study: impact and causes of illness and death in Australia 2018* and
- *Australian Burden of Disease Study: impact and causes of illness and death in Australia 2018—summary report* and
- *Australian Burden of Disease Study 2018: key findings for Aboriginal and Torres Strait Islander people* and
- *Australian Burden of Disease Study: impact and causes of illness and death in Aboriginal and Torres Strait Islander people 2018* and
- *Australian Burden of Disease Study: impact and causes of illness and death in Aboriginal and Torres Strait Islander people 2018—summary report*.

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Introduction

Key considerations

The ABDS 2018 methods build on the methodological approach of the ABDS 2015 (AIHW 2016; AIHW 2019), along with methodological developments used in recent iterations of the Global Burden of Disease study (GBD 2017 and 2019). Key considerations for the ABDS 2018 were the need for:

- national estimates which were relevant to Australia, while maintaining comparability with global methods as much as possible
- Indigenous estimates which were comparable with national estimates
- sub-national estimates (state/territory, remoteness and socioeconomic group)
- comparability to 2015, 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 the ABDS 2011 and ABDS 2015 (Box 1.1).

Box 1.1: Principles for the ABDS 2018 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
- 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 Overarching methods and choices for risk factors)
- be consistent with the disease list (including sequelae) in terms of the associated linked diseases.

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Introduction

Expert advice and review

An Expert Advisory Group provided oversight and detailed advice on key technical issues, including the overall methods and inputs throughout the ABDS 2018. The Indigenous Reference Group provided guidance on inputs and issues specifically related to estimating burden in Aboriginal and Torres Strait Islander people.

In the ABDS 2011 each disease group had an expert panel of people with relevant clinical and epidemiological expertise to review and inform the methods being developed. This involvement was extended to ABDS 2015. For the ABDS 2018, panel members or key experts provided advice where methods were revised from those used in the ABDS 2015.

Expert Advisory Group members

Member	Organisation
Assoc. Prof. Ching Choi (Chair)	University of New South Wales
Prof. Anthony Barnes	Independent consultant
Prof. Antony Blakely	University of Otago/ University of Melbourne
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Indigenous Reference Group

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Disease-specific contributors

This list includes experts consulted for ABDS 2018 only. Methodological advice received from Expert Advisory Group members, the Indigenous Reference Group and other experts for ABDS 2011 and 2015, and development work undertaken by former ABDS staff is gratefully acknowledged. Please see ABDS 2015 (AIHW 2019) for a full list of expert advisors for previous Studies.

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Population Health Unit	AIHW
Tobacco, Alcohol and Other Drugs Unit	AIHW
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Overarching methods

Overarching methods and choices for ABDS 2018

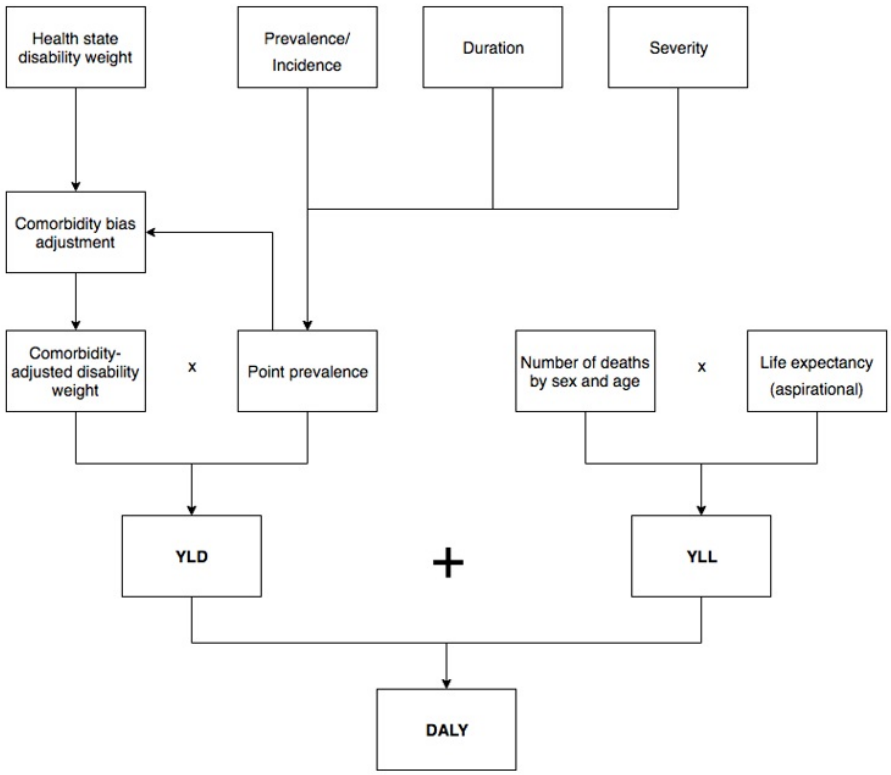
On this page:

Overarching methods and choices for ABDS 2018

The ABDS 2018 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.

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



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

In the ABDS 2018, 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 Estimating the fatal burden 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 Estimating the non-fatal burden 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.

DALY = YLL + YLD

When DALY are used to measure the burden of disease in a population in a time interval, they can be calculated in various ways: from an incidence, prevalence, or hybrid perspective. Each method produces a measurement of a different quantity. This study used the hybrid perspective for calculating DALY consistent with the ABDS 2011, ABDS 2015 and recent global studies. This calculates YLL from an incidence perspective (see Estimating the fatal burden for details) and YLD from a prevalence perspective (see Estimating the non-fatal burden for details). 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 2018, 2015, 2011 and 2003

Based on the availability of data at the start of the study, 2018 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 2018 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 2018.

Although 2018 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.

There have been 4 previous Australian burden of disease studies with estimates published in ABDS 1996, ABDS 2003, ABDS 2011 and ABDS 2015. While overarching methods for estimating disease burden remained unchanged from the ABDS 2011, revision of some disease-specific methods in ABDS 2015 and ABDS 2018 lead to estimates that differed considerably from the ABDS 2011. Therefore, revision of 2015, 2011 and 2003 estimates were required to provide comparable Australian burden of disease estimates to assess changes over time. These revisions reduce the risk of users making erroneous comparisons between previous 2003, 2011 and 2015 estimates with those produced in ABDS 2018.

Reference populations

All Australian population-based rates for 2018 and 2015 were calculated using populations rebased to the 2016 Census (released 27 June 2017) (ABS 2017).

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 2018, 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: 219 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 (ABDS 2018 list of diseases, conditions and injuries and ICD-10 codes).

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 2015 disease list was reviewed, and modifications made based on a set of inclusion criteria originally developed and applied in the ABDS 2015.

For inclusion in the ABDS 2018 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 2017 or the ABDS 2015 (AIHW 2016a) unless its inclusion in the ABDS 2018 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 219 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 2018. This includes 13 conditions describing the nature of injury used for alternative reporting (see Injuries for more detail).

As such, the ABDS 2018 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). In the ABDS 2018, 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 for the 2018 Australian study

- A more comprehensive list of diseases,
- disaggregation of pneumoconiosis into silicosis, asbestosis and other pneumoconiosis, and
- the addition of scabies, which was previously reported under skin infections.

For reporting purposes, Lower respiratory infections and influenza, which includes pneumonia, are combined under Lower respiratory infections (including influenza and pneumonia).

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 2018 disease list:

- **Scarcity of recent and/or robust data to reliably estimate prevalence in Australia in 2018**—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 2018, 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 low bone mineral density for more detail)
    - nutritional deficiencies—in the ABDS 2018, 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 iron deficiency and dietary risk factors for more information).

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 Disease specific methods - morbidity 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 219 diseases in the ABDS 2018 disease list was based on the ABDS 2011 (AIHW 2016b) 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).

Table 2.1: ABDS 2018 list of diseases, conditions and injuries by ICD-10 code

ABDS Code	ABDS Name	ICD10 codes
Blood and metabolic disorders		
P01	Cystic Fibrosis	E84
P02	Haemophilia	D66, D67
P03	Haemolytic anaemias	D55, D56, D57, D58
P04	Iron-deficiency anaemia	D50.1, D50.8, D50.9
P05	Protein-energy deficiency	E40, E41, E42, E43, E44, E45, E46
P99	Other blood and metabolic disorders	D50.0, D51, D52, D53, D59.0, D59.1, D59.2, D59.4, D59.5, D59.6, D59.8, D59.9, D60, D61, D62, D63, D64, D65, D68, D69, D70, D71, D72, D73, D74 , D75, D76, D77, D80, D81, D82, D83, D84.0, D84.1, D84.8, D84.9, D86.1, D86.3, D86.8, D89, E00, E01, E02, E50, E51, E52, E53, E54, E55, E56 , E58, E59, E60, E61, E63, E64, E65, E66, E67, E68, E70, E71, E72, E73, E74, E75, E76, E77, E78, E79, E80, E83, E85.0, E85.1, E85.2 , E88, E90
Cancer and other neoplasms		

C02	Laryngeal cancer	C32
C03	Oesophageal cancer	C15
C04	Stomach cancer	C16
C05	Bowel cancer	C18, C19, C20, C26.0
C06	Liver cancer	C22
C07	Gallbladder cancer	C23, C24
C08	Pancreatic cancer	C25
C09	Lung cancer	C33, C34
C10	Mesothelioma	C45
C11	Melanoma of the skin	C43
C12	Non-melanoma skin cancers	C44
C13	Breast cancer	C50
C14	Cervical cancer	C53
C15	Uterine cancer	C54, C55
C16	Ovarian cancer	C56
C17	Prostate cancer	C61
C18	Testicular cancer	C62
C19	Bladder cancer	C67
C20	Kidney cancer	C64
C21	Brain and central nervous system cancer	C70, C71, C72
C22	Thyroid cancer	C73
C23	Non-Hodgkin lymphoma	C82, C83, C84, C85, C86
C24	Hodgkin lymphoma	C81
C26	Myeloma	C90
C27	Other blood cancers	C88, C96, D45, D46, D47.1, D47.3, D47.4, D47.5
C28	Unknown primary	C39, C97
C31	Benign and uncertain brain tumours	D32, D33, D42, D43
C32	Ductal carcinoma in situ (breast)	D05
C41	Lip and oral cavity cancer	C00, C01, C02, C03, C04, C05, C06, C07, C08
C42	Nasopharyngeal cancer	C11
C43	Other oral cavity and pharynx cancers	C09, C10, C12, C13, C14
C44	Acute myeloid leukaemia (AML)	C92.0, C92.3, C92.4, C92.5, C92.6, C92.8, C93.0, C94.0, C94.2, C94.4, C94.5

C45	Chronic myeloid leukaemia (CML)	C92.1
C46	Acute lymphoblastic leukaemia (ALL)	C91.0
C47	Chronic lymphocytic leukaemia (CLL)	C91.1
C48	Other leukaemias	C91.2, C91.3, C91.4, C91.5, C91.6, C91.7, C91.8, C91.9, C92.2, C92.7, C92.9, C93.1, C93.2, C93.3, C93.7, C93.9, C94.1, C94.3, C94.6, C94.7, C95
C98	Other malignant neoplasms (cancers)	C17, C21, C30, C31, C37, C38, C40, C41, C46, C47, C48, C49, C51, C52, C57, C58, C60, C63, C65, C66, C68, C69, C74, C75
C99	Other benign, insitu and uncertain neoplasms	D00, D01, D02, D03, D04, D06, D07, D09, D10, D11, D12, D13, D14, D15, D16, D17, D18, D19, D20, D21, D22, D23, D24, D26, D27 , D28, D29, D30, D31, D34, D35, D36, D37, D38, D39, D40, D41, D44, D47.0, D47.2, D47.7, D47.9, D48
Cardiovascular diseases		
D01	Coronary heart disease	I20, I21, I22, I23, I24, I25
D02	Stroke	I60, I61, I62, I63, I64, I65, I66, I67, I68, I69
D03	Rheumatic heart disease (including acute rheumatic fever)	I00, I01, I02, I05, I06, I08.0, I08.1, I08.3, I09
D04	Non-rheumatic valvular disease	I07, I08.2, I08.8, I08.9, I34, I35, I36, I37, I38, I39
D05	Hypertensive heart disease	I11
D06	Atrial fibrillation and flutter	I48
D07	Inflammatory heart disease	I30, I31, I32, I33, I40, I41
D08	Cardiomyopathy	I42, I43
D09	Aortic aneurysm	I71
D10	Peripheral vascular disease	I70.0, I70.1, I70.2, I70.8, I72, I73, I74
D99	Other cardiovascular diseases	G45, I26, I27, I28, I44, I45, I47, I49.1, I49.2, I49.3, I49.4, I49.5, I49.8, I49.9, I51, I52, I77, I78, I79, I80, I81, I82, I83, I84, I86 , I87, I88, I89, I95, I97, I98, I99
Endocrine disorders		
I02	Type 1 diabetes	E10.0, E10.1, E10.3, E10.4, E10.5, E10.6, E10.7, E10.8, E10.9, O24.0
I03	Type 2 diabetes	E11.0, E11.1, E11.3, E11.4, E11.5, E11.6, E11.7, E11.8, E11.9, O24.1
I04	Other diabetes	E12.0, E12.1, E12.3, E12.4, E12.5, E12.6, E12.7, E12.8, E12.9, E13.0, E13.1, E13.3, E13.4, E13.5, E13.6, E13.7, E13.8, E13.9, O24.2
I99	Other endocrine disorders	E03, E04, E05, E06, E07, E15, E16, E20, E21, E22, E23, E24, E25, E26, E27, E28.0, E28.1, E28.3, E28.8, E28.9, E29, E30, E31, E32, E34 , E35, E89

Gastrointestinal disorders		
F01	Gastroduodenal disorders	K22.1, K25, K26, K27, K29
F02	Appendicitis	K35, K36, K37
F03	Abdominal wall hernia	K40, K41, K42, K43, K45, K46
F04	Vascular disorders of intestine	K55
F05	Intestinal obstruction (without hernia)	K56
F06	Inflammatory bowel disease (IBD)	K50, K51, K52
F07	Diverticulitis	K57
F08	Chronic liver disease	B18, I85, K70, K71.0, K71.1, K71.3, K71.4, K71.5, K71.6, K71.7, K71.8, K71.9, K72, K73, K74, K75, K76
F09	Gallbladder and bile duct disease	K80, K81, K82, K83
F10	Pancreatitis	K85, K86
F11	Gastro Oesophageal Reflux Disease (GORD)	K20, K21, K44
F12	Functional gastrointestinal disorders (FGID)	
F99	Other gastrointestinal diseases	K22.0, K22.2, K22.3, K22.4, K22.5, K22.6, K22.7, K22.8, K22.9, K23, K28, K30, K31, K38, K58, K59, K60, K61, K62.0, K62.1, K62.4, K62.5, K62.6, K62.7, K62.8 , K62.9, K63, K64, K67, K77, K87, K90, K91, K93
Hearing and vision disorders		
M02	Hearing loss	H90, H91
M03	Refractive errors	H49–H52
M04	Cataract and other lens disorders	H25–H27
M05	Glaucoma	H40, H42
M06	Age-related macular degeneration	H35.3
M98	Other vision disorders	H30–H35 (excluding H35.3), H43–H48, H53–H59
M99	Other hearing and vestibular disorders	H60.2–H60.9, H61, H68–H69, H71–H74, H80–H83, H92–H93
Infant and congenital conditions		
B01	Pre-term birth and low birth weight complications	P01.0, P01.1, P05, P07, P22, P25, P26, P27, P28, P52, P61.2, P77
B03	Birth trauma and asphyxia	P01.7, P01.8, P01.9, P02, P03, P08, P10, P11, P12, P13, P14, P15, P20, P21, P24, P90, P91
B04	Cerebral palsy	G80

B05	Neonatal infections	P23, P35.1, P35.2, P35.3, P35.8, P35.9, P36, P37.1, P37.2, P37.5, P37.8, P37.9, P38, P39
B06	Sudden infant death syndrome	R95
B07	Other disorders of infancy	P00, P01.2, P01.3, P01.4, P01.5, P01.6, P04, P29, P50, P51, P53, P54, P55, P56, P57, P58, P59, P60, P61.0, P61.1, P61.3, P61.4, P61.5, P61.6, P61.8 , P61.9, P70, P71, P72, P74, P75, P76, P78, P80, P81, P83, P92, P93, P94, P95, P96
B08	Neural tube defects	Q00, Q01, Q05
B09	Cardiovascular defects	Q20, Q21, Q22, Q23, Q24, Q25, Q26, Q27, Q28
B10	Cleft lip and/or palate	Q35, Q36, Q37
B12	Gastrointestinal malformations	Q38.0, Q38.2, Q38.3, Q38.4, Q38.5, Q38.6, Q38.7, Q38.8, Q39, Q40, Q41, Q42, Q43, Q44, Q45
B13	Urogenital malformations	Q50, Q51, Q52, Q53, Q55, Q56, Q60, Q62, Q63, Q64
B14	Down syndrome	Q90
B15	Brain malformations	Q02, Q03, Q04, Q86.0
B97	Other chromosomal abnormalities	Q91, Q92, Q93, Q95, Q96, Q97, Q98, Q99.0, Q99.1, Q99.2, Q99.8
B99	Other congenital conditions	Q06, Q07, Q30, Q31, Q32, Q33, Q34, Q75, Q76, Q77, Q78, Q79, Q80, Q81, Q85, Q86.1, Q86.2, Q86.8, Q87, Q89.0, Q89.1, Q89.2, Q89.3, Q89.4, Q89.7 , Q89.8
Infectious diseases		
A01	HIV/AIDS	B20, B21, B22, B23, B24, O98.7
A02	Tuberculosis	A15, A16, A17, A18, A19, B90, N33.0, N74.0, N74.1, O98.0, P37.0
A03	Syphilis	A50, A51, A52, A53, N29.0, N74.2, O98.1
A04	Chlamydia	A55, A56, N74.4
A05	Gonorrhoea	A54, N74.3, O98.2
A06	Other sexually transmitted infections	A57, A58, A59, A60, A63, A64, O98.3
A07	Hepatitis A	B15
A08	Hepatitis B (acute)	B16, B17.0
A09	Hepatitis C (acute)	B17.1, B17.8, B17.9
A11	Upper respiratory infections	J00, J01, J02, J03, J04, J05, J06
A12	Otitis media	H65, H66, H68, H70
A13	Lower respiratory infections	J12, J14, J15, J16, J17, J18, J20, J21, J22, J85, J86
A14	Influenza	J09, J10, J11
A15	Diphtheria	A36
A16	Pertussis	A37
A17	Tetanus	A33, A34, A35

A18	Measles	A81.1, B05
A19	Rubella	B06, P35.0
A21	Haemophilus influenzae type-b	G00.0
A22	Pneumococcal disease	A40.3, G00.1, J13
A23	Meningococcal disease	A39
A24	Other meningitis and encephalitis	A83, A84, A85, A86, A87, B94.1, G00.2, G00.3, G00.8, G00.9, G01, G02, G03, G04, G05
A25	Dengue	A90, A91
A26	Ross River virus	B33.1
A27	Barmah Forest virus	A92.8
A28	Malaria	B50, B51, B52, B53, B54, P37.3, P37.4
A29	Trachoma	A71, B94.0
A30	Campylobacteriosis	A04.5
A31	Salmonellosis	A02
A32	Rotavirus	A08.0
A33	Other gastrointestinal infections	A00, A01, A03, A04.0, A04.1, A04.2, A04.3, A04.4, A04.6, A04.7, A04.8, A04.9, A05, A06, A07, A08.1, A08.2, A08.3, A08.4, A08.5, A09, D59.3
A41	Varicella	B01
A42	Herpes zoster	B02
A43	Mumps	B26
A44	Urinary tract infections	N30, N34, N39.0
A99	Other infections	A20, A21, A22, A23, A24, A25, A26, A27, A28, A30, A31, A32, A38, A42, A43, A44, A48.1, A48.2, A48.4, A48.8, A49, A65, A66, A67, A68 , A69, A70, A74, A75, A77, A78, A79, A80, A81.0, A81.2, A81.8, A81.9, A82, A88, A89, A92.0, A92.1, A92.2, A92.3, A92.4, A92.9, A93, A94, A95, A96 , A98, A99, B00, B03, B04, B07, B08.0, B08.2, B08.3, B08.5, B08.8, B09, B17.2, B25, B27, B30, B33.0, B33.2, B33.3, B33.4, B33.8, B34, B35, B36, B37 , B38, B39, B40, B41, B42, B43, B44, B45, B46, B47, B48, B49, B55, B56, B57, B58, B59, B60, B64, B65, B66, B67, B68, B69, B70 , B71, B72, B73, B74, B75, B76, B77, B78, B79, B80, B81, B82, B83, B85, B87, B88, B89, B91, B92, B94.8, B94.9, B95, B96, B97, B98 , B99, G06, G07, O98.4, O98.5, O98.6, O98.8, O98.9
Injury (external cause)		
Q01	Road traffic injuries - motorcyclists	V20.3, V20.4, V20.5, V20.9, V21.3, V21.4, V21.5, V21.9, V22.3, V22.4, V22.5, V22.9, V23.3, V23.4, V23.5, V23.9, V24.3, V24.4, V24.5, V24.9, V25.3, V25.4, V25.5, V25.9, V26.3 , V26.4, V26.5, V26.9, V27.3, V27.4, V27.5, V27.9, V28.3, V28.4, V28.5, V28.9, V29.4, V29.5, V29.6, V29.8, V29.9

Q02	Road traffic injuries - motor vehicle occupants	V30.4, V30.5, V30.6, V30.7, V30.9, V31.4, V31.5, V31.6, V31.7, V31.9, V32.4, V32.5, V32.6, V32.7, V32.9, V33.4, V33.5, V33.6, V33.7, V33.9, V34.4, V34.5, V34.6, V34.7, V34.9 , V35.4, V35.5, V35.6, V35.7, V35.9, V36.4, V36.5, V36.6, V36.7, V36.9, V37.4, V37.5, V37.6, V37.7, V37.9, V38.4, V38.5, V38.6, V38.7, V38.9, V39.4, V39.5, V39.6, V39.8, V39.9 , V40.4, V40.5, V40.6, V40.7, V40.9, V41.4, V41.5, V41.6, V41.7, V41.9, V42.4, V42.5, V42.6, V42.7, V42.9, V43.4, V43.5, V43.6, V43.7, V43.9, V44.4, V44.5, V44.6, V44.7, V44.9 , V45.4, V45.5, V45.6, V45.7, V45.9, V46.4, V46.5, V46.6, V46.7, V46.9, V47.4, V47.5, V47.6, V47.7, V47.9, V48.4, V48.5, V48.6, V48.7, V48.9, V49.4, V49.5, V49.6, V49.8, V49.9 , V50.4, V50.5, V50.6, V50.7, V50.9, V51.4, V51.5, V51.6, V51.7, V51.9, V52.4, V52.5, V52.6, V52.7, V52.9, V53.4, V53.5, V53.6, V53.7, V53.9, V54.4, V54.5, V54.6, V54.7, V54.9 , V55.4, V55.5, V55.6, V55.7, V55.9, V56.4, V56.5, V56.6, V56.7, V56.9, V57.4, V57.5, V57.6, V57.7, V57.9, V58.4, V58.5, V58.6, V58.7, V58.9, V59.4, V59.5, V59.6, V59.8, V59.9 , V60.4, V60.5, V60.6, V60.7, V60.9, V61.4, V61.5, V61.6, V61.7, V61.9, V62.4, V62.5, V62.6, V62.7, V62.9, V63.4, V63.5, V63.6, V63.7, V63.9, V64.4, V64.5, V64.6, V64.7, V64.9 , V65.4, V65.5, V65.6, V65.7, V65.9, V66.4, V66.5, V66.6, V66.7, V66.9, V67.4, V67.5, V67.6, V67.7, V67.9, V68.4, V68.5, V68.6, V68.7, V68.9, V69.4, V69.5, V69.6, V69.8, V69.9 , V70.4, V70.5, V70.6, V70.7, V70.9, V71.4, V71.5, V71.6, V71.7, V71.9, V72.4, V72.5, V72.6, V72.7, V72.9, V73.4, V73.5, V73.6, V73.7, V73.9, V74.4, V74.5, V74.6, V74.7, V74.9 , V75.4, V75.5, V75.6, V75.7, V75.9, V76.4, V76.5, V76.6, V76.7, V76.9, V77.4, V77.5, V77.6, V77.7, V77.9, V78.4, V78.5, V78.6, V78.7, V78.9, V79.4, V79.5, V79.6, V79.8, V79.9 , V89.2, Y85.0
Q04	Other land transport injuries	V01.0, V02.0, V03.0, V04.0, V05.0, V06.0, V09.0, V09.1, V10.0, V10.1, V10.2, V11.0, V11.1, V11.2, V12.0, V12.1, V12.2, V13.0, V13.1, V13.2, V14.0, V14.1, V14.2, V15.0, V15.1 , V15.2, V16.0, V16.1, V16.2, V17.0, V17.1, V17.2, V18.0, V18.1, V18.2, V19.0, V19.1, V19.2, V19.3, V20.0, V20.1, V20.2, V21.0, V21.1, V21.2, V22.0, V22.1, V22.2, V23.0, V23.1 , V23.2, V24.0, V24.1, V24.2, V25.0, V25.1, V25.2, V26.0, V26.1, V26.2, V27.0, V27.1, V27.2, V28.0, V28.1, V28.2, V29.0, V29.1, V29.2, V29.3, V30.0, V30.1, V30.2, V30.3, V31.0 , V31.1, V31.2, V31.3, V32.0, V32.1, V32.2, V32.3, V33.0, V33.1, V33.2, V33.3, V34.0, V34.1, V34.2, V34.3, V35.0, V35.1, V35.2, V35.3, V36.0, V36.1, V36.2, V36.3, V37.0, V37.1 , V37.2, V37.3, V38.0, V38.1, V38.2, V38.3, V39.0, V39.1, V39.2, V39.3, V40.0, V40.1, V40.2, V40.3, V41.0, V41.1, V41.2, V41.3, V42.0, V42.1, V42.2, V42.3, V43.0, V43.1, V43.2 , V43.3, V44.0, V44.1, V44.2, V44.3, V45.0, V45.1, V45.2, V45.3, V46.0, V46.1, V46.2, V46.3, V47.0, V47.1, V47.2, V47.3, V48.0, V48.1, V48.2, V48.3, V49.0, V49.1, V49.2, V49.3 , V50.0, V50.1, V50.2, V50.3, V51.0, V51.1, V51.2, V51.3, V52.0, V52.1, V52.2, V52.3, V53.0, V53.1, V53.2, V53.3, V54.0, V54.1, V54.2, V54.3, V55.0, V55.1, V55.2, V55.3, V56.0 , V56.1, V56.2, V56.3, V57.0, V57.1, V57.2, V57.3, V58.0, V58.1, V58.2, V58.3, V59.0, V59.1, V59.2, V59.3, V60.0, V60.1, V60.2, V60.3, V61.0, V61.1, V61.2, V61.3, V62.0, V62.1 , V62.2, V62.3, V63.0, V63.1, V63.2, V63.3, V64.0, V64.1, V64.2, V64.3, V65.0, V65.1, V65.2, V65.3, V66.0, V66.1, V66.2, V66.3, V67.0, V67.1, V67.2, V67.3, V68.0, V68.1, V68.2 , V68.3, V69.0, V69.1, V69.2, V69.3, V70.0, V70.1, V70.2, V70.3, V71.0, V71.1, V71.2, V71.3, V72.0, V72.1, V72.2, V72.3, V73.0, V73.1, V73.2, V73.3, V74.0, V74.1, V74.2, V74.3 , V75.0, V75.1, V75.2, V75.3, V76.0, V76.1, V76.2, V76.3, V77.0, V77.1, V77.2, V77.3, V78.0, V78.1, V78.2, V78.3, V79.0, V79.1, V79.2, V79.3, V80, V81, V82, V83, V84 , V85, V86, V87, V88, V89.0, V89.1, V89.3, V89.9, Y85.9
Q05	Poisoning	X40, X41, X42, X43, X44, X45, X46, X47, X48, X49

Q06	Falls	W00, W01, W02, W03, W04, W05, W06, W07, W08, W09, W10, W11, W12, W13, W14, W15, W16, W17, W18, W19
Q07	Fire, burns and scalds	X00, X01, X02, X03, X04, X05, X06, X08, X09, X10, X11, X12, X13, X14, X15, X16, X17, X18, X19
Q08	Drowning and submersion	V90, V92, W65, W66, W67, W68, W69, W70, W73, W74
Q09	Other unintentional injuries	V91, V93, V94, V95, V96, V97, V98, V99, W20, W21, W22, W23, W24, W25, W26, W27, W28, W29, W30, W31, W32, W33, W34, W35, W36 , W37, W38, W39, W40, W41, W42, W43, W44, W45, W46, W49, W50, W51, W52, W53, W54, W55, W56, W57, W58, W59, W60, W64, W75, W76 , W77, W78, W79, W80, W81, W83, W84, W85, W86, W87, W88, W89, W90, W91, W92, W93, W94, W99, X20, X21, X22, X23, X24, X25, X26 , X27, X28, X29, X30, X31, X32, X33, X34, X35, X36, X37, X38, X39, X50, X51, X52, X53, X54, X57, X58, Y35, Y36, Y86, Y89.0, Y89.1
Q10	Suicide and self-inflicted injuries	X60, X61, X62, X63, X64, X65, X66, X67, X68, X69, X70, X71, X72, X73, X74, X75, X76, X77, X78, X79, X80, X81, X82, X83, X84 , Y87.0
Q11	Homicide and violence	X85, X86, X87, X88, X89, X90, X91, X92, X93, X94, X95, X96, X97, X98, X99, Y00, Y01, Y02, Y03, Y04, Y05, Y06, Y07, Y08, Y09 , Y87.1
Q21	Road traffic injuries - pedal cyclists	V10.3, V10.4, V10.5, V10.9, V11.3, V11.4, V11.5, V11.9, V12.3, V12.4, V12.5, V12.9, V13.3, V13.4, V13.5, V13.9, V14.3, V14.4, V14.5, V14.9, V15.3, V15.4, V15.5, V15.9, V16.3 , V16.4, V16.5, V16.9, V17.3, V17.4, V17.5, V17.9, V18.3, V18.4, V18.5, V18.9, V19.4, V19.5, V19.6, V19.8, V19.9
Q22	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
Q99	All other external causes of injury	Y40, Y41, Y42, Y43, Y44, Y45, Y46, Y47, Y48, Y49, Y50, Y51, Y52, Y53, Y54, Y55, Y56, Y57, Y58, Y59, Y60, Y61, Y62, Y63, Y64 , Y65, Y66, Y69, Y70, Y71, Y72, Y73, Y74, Y75, Y76, Y77, Y78, Y79, Y80, Y81, Y82, Y83, Y84, Y88
Injury (nature)		
R01	Traumatic brain injury	S02.0, S02.1, S02.7, S02.9, S06
R02	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
R03	Internal and crush injury	S07, S11.0, 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
R04	Poisoning	T36, T37, T38, T39, T40, T41, T42, T43, T44, T45, T46, T47, T48, T49, T50, T51, T52, T53, T54, T55, T56, T57, T58, T59, T60 , T61, T62, T63, T64, T65
R05	Drowning and submersion injuries	T75.1
R06	Hip fracture	S72
R07	Tibia and ankle fracture	S82.1, S82.2, S82.3, S82.4, S82.5, S82.6, S82.7, S82.8, S82.9
R08	Humerus fracture	S42.2, S42.3, S42.4, S42.7
R09	Other fractures	S02.2, S02.3, S02.4, S02.5, S02.6, S02.8, S12, S22.0, S22.1, S22.2, S22.3, S22.8, S22.9, S32, S42.0, S42.1, S42.8, S42.9, S49.7, S52, S59.7, S62, S69.7, S82.0, S92 , T02, T08, T10, T12, T14.2

R10	Dislocations	S03.0, S03.1, S03.2, S03.3, S13.1, S13.2, S13.3, S23.1, S23.2, S33.1, S33.2, S33.3, S43.0, S43.1, S43.2, S43.3, S53.0, S53.1, S63.0, S63.1, S63.2, S73.0, S83.0, S83.1, S93.0 , S93.1, S93.3, T03, T09.2, T11.2, T13.2, T14.3
R11	Soft tissue injuries	S03.4, S03.5, S13.4, S13.5, S13.6, S16, S23.0, S23.3, S23.4, S23.5, S29.0, S33.5, S33.6, S33.7, 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
R12	Burn injuries	T20, T21, T22, T23, T24, T25, T26, T27, T28, T29, T30, T31, T32
R99	Other injuries	S00, S01, S04, S05, S08, S09, S10, S11.1, S11.2, S11.7, S11.8, S11.9, S13.0, S14.2, S14.3, S14.4, S14.5, S14.6, S15, S19, S20, S21, S24.2, S24.3, S24.4 , S24.5, S24.6, S29.8, S29.9, S30, S31, S33.0, S33.4, S34.2, S34.3, S34.4, S34.5, S34.6, S34.8, S38.2, S38.3, S39.8, S39.9, S40, S41, S44, S45, S48, S49.8, S49.9 , S50, S51, S54, S55, S58, S59.8, S59.9, S60, S61, S64, S65, S68, S69.8, S69.9, S70, S71, S74, S75, S78, S79, S80, S81, S84, S85, S88 , S89, S90, 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, T16, T17, T18, T19, T33, T34, T35, T66, T67 , T68, T69, T70, T71, T73, T74, T75.0, T75.2, T75.3, T75.4, T75.8, T79, T80, T81, T82, T83, T84, T85, T86, T87, T88, T89
Kidney and urinary diseases		
J01	Chronic kidney disease	E10.2, E11.2, E12.2, E13.2, E14.2, I12, N02, N03, N04, N05, N06, N07, N08, N13, N14, N15, N16, N18, N39.1, N39.2, Q61
J04	Enlarged prostate	N40
J05	Kidney stones	N20, N21
J06	Interstitial Nephritis	N10, N11, N12
J99	Other kidney and urinary diseases	N00, N01, N22, N23, N25, N26, N27, N28, N29.1, N29.8, N31, N32, N33.8, N35, N36, N37, N39.3, N39.4, N39.8, N39.9, N41, N42
Mental and substance use disorders		
H01	Depressive disorders	F32, F33, F34.1, F34.8, F34.9, F39
H02	Anxiety disorders	F40, F41, F42, F43
H03	Bipolar affective disorder	F30, F31, F34.0
H04	Alcohol use disorders	F10
H05	Drug use disorders (excluding alcohol)	F11, F12, F13, F14, F15, F16, F18, F19
H06	Schizophrenia	F20, F21, F22, F23, F24, F25, F28, F29
H07	Eating disorders	F50
H08	Autism spectrum disorders	F84
H09	Attention deficit hyperactivity disorder	F90
H10	Conduct disorder	F91, F92
H11	Intellectual disability	F70, F71, F72, F73, F78, F79

H99	Other mental and substance use disorders	F04, F05, F06, F07, F09, F17, F38, F44, F45, F48, F51, F52, F53, F54, F55, F59, F60, F61, F62, F63, F64, F65, F66, F68, F69 , F80, F81, F82, F83, F88, F89, F93, F94, F95, F98
Musculoskeletal disorders		
L01	Osteoarthritis	M15, M16, M17, M18, M19
L02	Gout	M10
L03	Rheumatoid arthritis	M05, M06, M08
L05	Back pain and problems	M40, M41, M45, M46, M47, M48, M49, M50, M51, M53, M54, M99
L99	Other musculoskeletal	M00, M01, M02, M03, M07, M09, M11, M12, M13, M14, M20, M21, M22, M23, M24, M25, M30, M31, M32, M33, M34, M35, M36, M42, M43 , M60, M61, M62, M63, M65, M66, M67, M68, M70, M71, M72, M73, M75, M76, M77, M79, M80, M81, M82, M83, M84, M85, M86, M87, M88 , M89, M90, M91, M92, M93, M94, M95, M96
Neurological conditions		
G01	Epilepsy	G40, G41
G02	Dementia	F00, F01, F02, F03, G30, G31
G03	Parkinson disease	G20
G04	Multiple sclerosis	G35
G05	Motor neurone disease	G12.2
G06	Migraine	G43
G07	Guillain-Barre Syndrome	G61.0
G99	Other neurological conditions	G08, G09, G10, G11, G12.0, G12.1, G12.8, G12.9, G13, G14, G21, G22, G23, G24, G25, G26, G32, G36, G37, G44, G46, G47, G50, G51, G52 , G53, G54, G55, G56, G57, G58, G59, G60, G61.1, G61.8, G61.9, G62, G63, G64, G70, G71, G72, G73, G90, G91, G92, G93, G94, G95, G96 , G97, G98, G99
Oral disorders		
O01	Dental caries	K02, K04
O02	Periodontal disease	K05
O03	Severe tooth loss	
O99	Other oral disorders	K00, K01, K03, K06, K07, K08, K09, K10, K11, K12, K13, K14
Reproductive and maternal conditions		
K01	Maternal haemorrhage	O44.1, O45, O46, O67, O72
K02	Maternal infections	O41.1, O85, O86
K03	Hypertensive disorders of pregnancy	O10, O11, O13, O14, O15, O16
K04	Obstructed labour	O64–O66
K05	Early pregnancy loss	O00–O08
K06	Gestational diabetes	O24.4
K08	Endometriosis	N80

K09	Uterine fibroids	D25
K10	Genital prolapse	N81, K62.2, K62.3
K11	Polycystic ovarian syndrome	E28.2
K12	Infertility	N46, N97
K98	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
K99	Other reproductive conditions	N43–N45, N47–N50, N60, N62–N64, N70–73, N74.8, N75–N77, N82–N83, N84–N90, N91–N96, N98–N99, O94
Respiratory diseases		
E01	Asthma	J45, J46
E02	Chronic obstructive pulmonary disease (COPD)	J40–J44
E03	Sarcoidosis	D86.0, D86.2, D86.9
E04	Interstitial lung disease	J84
E06	Upper respiratory diseases	J30–J33, J34.1–J34.9, J35–J39
E07	Silicosis	J62
E08	Asbestosis	J61
E09	Other pneumoconiosis	J60, J63-J65
E99	Other respiratory disease	J47, J66–J68, J70, J80–J82, J90–J96, J98–J99
Skin disorders		
N01	Dermatitis and eczema	L20–L27, L30
N02	Psoriasis	L40
N03	Acne	L70
N04	Ulcers	L89, L97, L98.4
N05	Skin infections (including cellulitis)	A46, B08.1, B08.4, H00.0, H60.0, H60.1, J34.0, L00–L04, L08
N06	Scabies	B86
N99	Other skin disorders	L05, L10–L13, L28–L29, L41–L45, L50–60, L62–L68, L71–L75, L80–L88, L90–L95, L98.0–L98.3, L98.5–L98.9, L99.0, L99.9

(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' for further details.

(d) Criteria used to diagnose this condition are currently not defined in ICD-10. See 'Oral disorders' for further details.

Source: WHO 2016.

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 2018 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 Additional material 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 2018, notably greater use of linked hospital/deaths data.

The key data source used in estimating mortality is described in Estimating fatal burden, and key data sources used in estimating morbidity are listed in Estimating non-fatal burden.

Methodological choices specific to Indigenous estimates

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

Indigenous under-identification

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

Dealing with small numbers

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

To ensure validity of the results, the AIHW combined several years of data and/or age groups as necessary to produce Indigenous estimates. Additionally, the level of disaggregation used to report Indigenous estimates was broader than that reported for the total Australian population. This included collapsed age groups for those aged 0–4 and 85 and over.

Measuring the gap between Indigenous and non-Indigenous Australians

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

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

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

Choice of population denominator for Indigenous estimates

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

Following sensitivity analyses by the AIHW to look at the impact of using different Indigenous population denominators in burden of disease rate calculations, it was agreed to use the backcast population series based on the 2016 Census, which applies the Indigenous identification level in 2016 to earlier years. Using this backcast population for all reference years provides consistency between the denominators used for the Indigenous burden of disease estimates in the ABDS 2018.

For more information on these choices, see *Impact and causes of illness and death in Aboriginal and Torres Strait Islander people 2018* (AIHW forthcoming 2022).

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, Australian Capital Territory and Northern 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 2016 Australian Statistical Geographic Standard (ASGS) for 2018 and 2015 estimates, or the 2011 ASGS for 2011 estimates. The ASGS 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. 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 Disease specific methods – mortality and Disease specific methods – morbidity.

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.

Indigenous sub-national estimates

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

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

Indigenous sub-national estimates were considered adequate to report for 4 states and territories (New South Wales, Queensland, Western Australia, and the Northern Territory). Estimates were not calculated for Victoria, South Australia, Tasmania or the Australian Capital Territory due to small numbers of Indigenous deaths in these jurisdictions, and lack of suitable mortality adjustment factors. However, these jurisdictions account for the majority of burden in most cases (see Table 2.3).

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

For Indigenous burden estimates by level of socioeconomic disadvantage, an Indigenous-specific index (the Indigenous Relative Socioeconomic Outcomes Index) (Biddle & Markham 2017) was used. This was considered to more accurately reflect levels of disadvantage in the Indigenous population than the SEIFA index used for the national component. As such, the Indigenous estimates by socioeconomic disadvantage were not compared with national estimates by socioeconomic disadvantage.

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

Hospitalisation data (adjusted for under-identification), ABS health survey data (2018–19 Australian Aboriginal and Torres Strait Islander Health Survey; 2012–2013 AATSIHS biomedical data), or population proportions (depending on the disease group) were used to break down the national-level Indigenous YLD into subnational categories. Hospitalisation data were used for 10 disease groups, and health survey data

were used for 5 disease groups for state/territory and remoteness estimates. A combination of Indigenous health survey data and data from the Australia and New Zealand Dialysis and Transplant Registry (ANZDATA) was used for 1 disease group (kidney & urinary diseases), and the subnational Indigenous population structure distribution data was used for the final disease group (skin disorders). For estimates by socioeconomic group, hospitalisation data were used for all disease groups, as Statistical Area Level 2 data (required to calculate the Indigenous Relative Socioeconomic Outcomes Index) were available from this data collection.

The data sources used to break down Indigenous YLD into subnational categories can be found in Table 2.2. The proportions used to break down Indigenous YLD estimates for each disease group can be found in tables 2.3 to 2.5.

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

For more information on the methods used for Indigenous subnational estimates see *Australian Burden of Disease Study: impact and causes of illness and death in Aboriginal and Torres Strait Islander people 2018* (AIHW forthcoming 2022).

Table 2.2: Data source used for subnational distribution of 2018 Indigenous non-fatal burden estimates

	State/territory	Remoteness	Socioeconomic group
Blood/metabolic	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Cancer	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Cardiovascular	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Endocrine	2012–13 AATSIHS	2012–13 AATSIHS	Adjusted hospitalisations
Gastrointestinal	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Hearing/vision	2018–19 NATSIHS	2018–19 NATSIHS	Adjusted hospitalisations
Infant/congenital	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Infections	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Injuries	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Kidney/urinary	2012–13 AATSIHS and ANZDATA	2012–13 AATSIHS and ANZDATA	Adjusted hospitalisations
Mental & substance use	2018–19 NATSIHS	2018–19 NATSIHS	Adjusted hospitalisations
Musculoskeletal	2018–19 NATSIHS	2018–19 NATSIHS	Adjusted hospitalisations
Neurological	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Oral	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Reproductive/maternal	Adjusted hospitalisations	Adjusted hospitalisations	Adjusted hospitalisations
Respiratory	2018–19 NATSIHS	2018–19 NATSIHS	Adjusted hospitalisations

Skin	Population distribution	Population distribution	Adjusted hospitalisations

Note: AATSIHS = Australian Aboriginal and Torres Strait Islander Health Survey, ANZDATA = Australia and New Zealand Dialysis and Transplant Registry, NATSIHS = National Aboriginal and Torres Strait Islander Health Survey.

Table 2.3: Sub-national proportions used for distribution of 2018 non-fatal burden estimates by state/territory, by Indigenous status

	Indigenous					Non-Indigenous				
	NSW	Qld	WA	NT	Remainder	NSW	Qld	WA	NT	Remainder
Blood/metabolic	20.8	25.5	14.4	22.5	16.8	23.8	21.0	11.9	0.4	42.9
Cancer	30.5	27.6	11.7	7.5	22.7	27.5	23.1	10.6	0.4	38.4
Cardiovascular	26.8	27.6	15.0	12.1	18.5	30.5	20.3	9.6	0.6	39.0
Endocrine	32.8	25.0	17.2	14.8	10.2	35.6	21.7	9.6	0.9	32.2
Gastrointestinal	30.0	24.8	14.5	10.1	20.6	30.9	20.5	9.7	0.6	38.3
Hearing/vision	33.4	27.9	10.8	6.8	21.1	31.3	20.2	10.0	0.6	37.9
Infant/congenital	31.4	27.2	11.6	10.0	19.8	32.6	18.5	10.8	0.6	37.5
Infections	23.2	25.3	17.0	21.4	13.1	29.7	21.7	9.7	0.6	38.3
Injuries	24.1	24.7	19.4	16.7	15.1	30.1	20.4	10.5	0.8	38.2
Kidney/urinary	16.1	24.6	17.2	27.4	14.7	56.7	3.4	1.5	1.5	36.9
Mental & substance use	32.7	26.9	12.7	8.3	19.4	31.2	19.6	9.6	0.6	39.0
Musculoskeletal	37.6	25.1	10.5	5.0	21.8	31.8	19.4	9.9	0.5	38.4
Neurological	29.9	27.6	13.8	8.7	20.0	25.0	22.4	10.6	0.4	41.6
Oral	22.4	27.4	14.0	13.1	23.1	25.3	18.8	13.8	0.4	41.7
Reproductive/maternal	27.8	29.7	13.5	12.4	16.6	30.3	21.3	10.6	1.0	36.8
Respiratory	38.0	26.2	10.9	3.8	21.1	29.3	19.7	9.9	0.5	40.6
Skin	33.2	27.8	12.6	9.2	17.2	31.9	19.8	10.3	0.7	37.3

Note: See Table 2.2 for data sources used for proportional splits.

Table 2.4a: Sub-national proportions used for distribution of 2018 non-fatal burden by remoteness, by Indigenous status

	Indigenous				
	Major cities	Inner regional	Outer regional	Remote	Very remote
Blood/metabolic	28.0	19.7	20.2	11.2	20.9
Cancer	37.5	26.4	20.4	6.7	9.0
Cardiovascular	29.0	20.0	23.0	10.9	17.1
Endocrine	27.3	16.4	17.2	11.7	28.1
Gastrointestinal	32.6	24.5	23.5	8.8	10.6
Hearing/vision	40.9	25.2	18.8	6.1	9.0
Infant/congenital	37.5	26.1	19.9	6.5	10.0
Infectious diseases	26.4	17.4	20.3	14.0	21.9
Injuries	30.7	18.9	20.4	11.4	18.6

Kidney/urinary	9.2	11.2	35.6	22.3	21.7
Mental	35.7	27.0	19.6	6.3	11.2
Musculoskeletal	41.7	24.8	20.6	6.0	7.1
Neurological	38.4	26.1	18.5	8.3	8.7
Oral	34.4	22.7	19.9	8.7	14.4
Reproductive/maternal	35.0	22.6	23.2	7.5	11.8
Respiratory	44.6	27.3	18.5	4.5	5.1
Skin	37.7	23.9	20.2	6.6	11.6

Note: See Table 2.2 for data sources used for proportional splits.

Table 2.4b: Sub-national proportions used for distribution of 2018 non-fatal burden by remoteness, by Indigenous status

	Non-Indigenous				
	Major cities	**Inner regional**	**Outer regional**	**Remote**	**Very remote**
Blood/metabolic	70.4	19.5	8.8	1.0	0.3
Cancer	68.3	21.6	8.9	0.9	0.3
Cardiovascular	67.2	22.1	9.3	1.0	0.4
Endocrine	64.8	22.2	11.1	1.4	0.5
Gastrointestinal	69.6	20.6	8.5	0.9	0.4
Hearing/vision	70.8	19.2	8.2	1.3	0.5
Infant/congenital	74.1	17.6	7.1	1.0	0.3
Infectious diseases	69.7	19.9	8.9	1.1	0.4
Injuries	69.6	19.9	8.9	1.1	0.5
Kidney/urinary	77.0	12.1	10.7	0.1	0.0
Mental	71.6	18.0	9.0	0.9	0.3
Musculoskeletal	68.4	20.9	8.9	1.4	0.5
Neurological	71.6	20.0	7.4	0.7	0.3
Oral	72.2	18.6	8.0	0.9	0.3
Reproductive/maternal	74.6	15.8	8.2	1.1	0.4
Respiratory	72.2	18.2	7.8	1.2	0.5
Skin	73.2	17.6	7.8	1.0	0.4

Table 2.5: Subnational proportions used for distribution of 2018 non-fatal burden by socioeconomic group (IRSEO Index), Indigenous Australians

	1 (most disadvantaged)	**2**	**3**	**4**	**5 (least disadvantaged)**
Blood/metabolic	19.2	20.0	24.9	16.9	19.0
Cancer	25.5	27.6	25.0	14.5	7.4
Cardiovascular	18.6	21.9	25.3	18.9	15.4
Endocrine	17.3	22.8	23.9	19.2	16.8
Gastrointestinal	22.4	25.3	26.0	17.5	8.9

Hearing/vision	21.0	24.5	22.6	17.4	14.5
Infant/congenital	21.9	27.3	27.7	14.9	8.3
Infections	15.9	20.5	24.1	19.0	20.5
Injuries	20.4	22.5	22.8	17.4	16.9
Kidney/urinary	22.6	24.4	25.0	16.3	11.8
Mental & substance use	27.7	24.0	23.8	15.1	9.4
Musculoskeletal	27.2	26.5	23.1	14.1	9.2
Neurological	26.8	25.6	25.3	14.9	7.5
Oral	23.1	25.8	23.9	16.5	10.6
Reproductive/maternal	20.6	26.4	27.2	15.9	10.0
Skin	15.6	21.2	22.9	20.5	19.8

Note: All proportions calculated from the NHMD.

Methodological choices specific to 2003, 2011, 2015 and 2018 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, 2015 and 2018 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 4 points in time can provide misleading information about changes over time—assuming that there is a straight-line trend between these 4 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 previous studies and 2018 data that have an impact on the interpretation are highlighted in the relevant chapters in this report.

2015, 2011 and 2003 estimates

Where there were no changes in methods or data sources, the 2015 estimates from the ABDS 2015 were adjusted because the underlying population estimates were updated following the release of the 2016 Census. In contrast, the 2011 and 2003 estimates were kept the same because the underlying populations are based on 2011 census. If there were changes in methods or data source for 2018, the estimates for previous years were re-estimated using the new methods to keep comparability across all four years.

Specific details on methods for previous years estimates for mortality, morbidity and risk factors are included in disease specific methods – mortality, disease specific methods – morbidity and risk factor specific methods.

Indigenous 2011 and 2003 estimates

Issues relating to changing Indigenous identification over time and potential inconsistencies in identification in numerator data and population denominators have an impact on the comparability of Indigenous burden of disease rates over time. These issues also have implications on the choice of population denominator used for 2003 and 2011 Indigenous burden of disease estimates.

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

The population denominator used for 2003 and 2011 Indigenous burden of disease estimates were consistent in terms of Indigenous identification with that used for 2018 estimates, which is important for assessing rate changes over time. Indigenous population estimates based on the 2016 Census were used, which applies the Indigenous identification level in 2016 to earlier years in the series, including for 2011 and 2003.

Additional material

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 2.2. 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 2.2: 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.

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.

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 assessment matrix below (Table 2.6).

- 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.

Table 2.6: Assessment matrix for data sources to be used in the ABDS 2018

Rating	High	Medium	Low
Comparability	Consistent	Comparable	Inconsistent
Relevance – National study	National	Sub-national or sub/super-regional (such as New Zealand, United States, Canada)	Other
Relevance – Indigenous study	Indigenous	National or sub-national	Other indigenous population
Currency	2013 or later	2007–2013	Before 2007
Accuracy – coverage	More than 90%	60%–90%	Less than 60%
Accuracy – sampling	Less than 25% RSE	25%–50% RSE	More than 50% RSE
Accuracy – measurement	Clinically reported or measured	Self-reported	Not known
Validation	Validated	Validated or not validated	Not validated

Credibility	Published and Peer reviewed	Published but not peer reviewed or Not published but peer reviewed	Not published nor peer reviewed
Accessibility/timeliness	Currently available	Expected to be available in time for analysis	Unlikely to be available in time for analysis

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Overarching methods

Overarching methods and choices for risk factors

On this page:

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 2018 are described in this chapter (see specific risk factor methods for detail on each risk factor).

Box 2.3: 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 described in the disease burden sections.

Methodological developments in the ABDS 2018

Most of the risk factors methods were the same as those used in the ABDS 2015. However, some methods have changed following new risk factor methods by GBD 2019 and expert advice (GBD 2019 Risk Factors Collaborators 2020).

The most notable of these was the changes to physical inactivity and the dietary risk factors which both had changes to the categories of exposure that increased risk and the minimum exposure associated with increased risk (TMRED). These changes are described in detail in Risk factor specific methods section, a summary of the major changes are outlined below.

The methods for the dietary risk factor Diet high in sugar sweetened beverages changed to be directly linked to type 2 diabetes and coronary heart disease instead of mediating through overweight (including obesity).

Exposure to air pollution was also estimated from satellites calibrated with ground monitoring stations, instead of ground monitoring stations alone, improving the coverage of estimates for Australia.

Two new risk factors have been included: bullying victimisation and low birthweight & short gestation. Both risk factors were included in GBD 2019; however, the methods used in ABDS 2018 for bullying victimisation were based on Australian specific studies (Jadambaa 2019a, Jadambaa 2019b).

Steps in estimating risk factor attributable burden

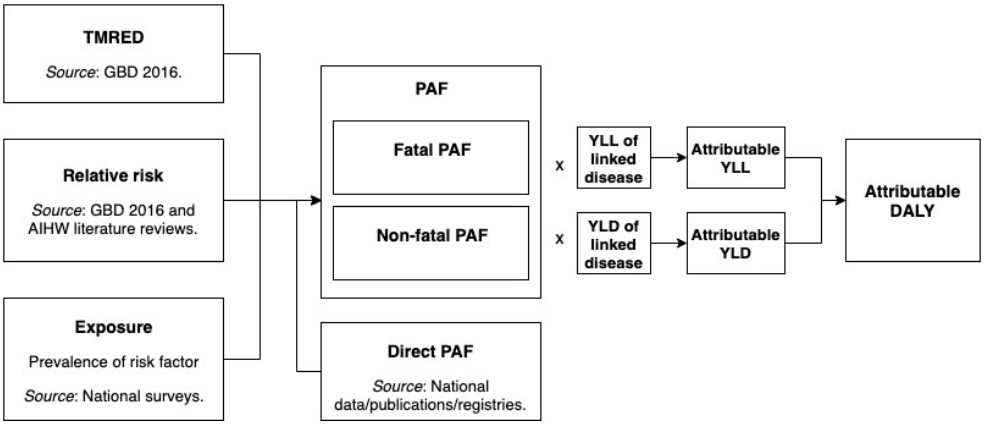
The basic steps of estimating risk factor attributable burden are:

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:
    1. 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
    2. 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 the figure below.

Figure 2.2: Inputs and processes to calculate attributable burden



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 2018, 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 2019
    - the ABDS 2011 or ABDS 2015 unless its inclusion in the ABDS 2018 conflicted with other criteria.

Substantial impact and policy interest

- Be of considerable importance to national or Indigenous disease burden based on previous studies (ABDS 2011, ABDS 2015; GBD 2019).
- Be of substantial Australian or Indigenous health policy interest—defined as currently being the focus of policy concern, professional attention or monitoring activity.
- Be modifiable, and able to be prevented or modified through policy intervention.

Be able to be measured

- Be 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 effect 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 2018 risk factor list

The ABDS 2018 identified 40 risk factor components or exposures (such as cannabis and cocaine use) that combine to 20 individual risk factors (such as illicit drug use). These were broadly grouped into categories (behavioural, metabolic/biomedical and environmental risks).

Table 2.7: List of risk factors 2018

Behavioural
Tobacco use
Tobacco use
Second-hand smoke
Dietary risks
Diet low in legumes
Diet low in whole grains & high fibre cereal
Diet high in sodium
Diet high in red meat
Diet low in fruit
Diet low in nuts & seeds
Diet low in vegetables
Diet high in processed meat
Diet low in polyunsaturated fat
Diet low in fish & seafood
Diet high in sugar-sweetened beverages
Diet low in milk
Alcohol use
Illicit drug use
Opioid use
Amphetamine use
Cocaine use
Cannabis use
Other illicit drug use
Unsafe injecting practices
Physical inactivity
Child abuse & neglect
Intimate partner violence
Unsafe sex
Bullying victimisation

Environmental
Occupational exposures & hazards
Air pollution
High sun exposure
Metabolic/Biomedical
Overweight (including obesity)
Overweight but not obese
Obesity
High blood pressure
High blood plasma glucose
Intermediate hyperglycaemia
Diabetes
High cholesterol
Impaired kidney function
Chronic kidney disease stage 1–3
Chronic kidney disease stage 4–5
Low birthweight & short gestation
Low bone mineral density
Iron deficiency

Due to methodological reasons, many of these 20 risk factors are the sum of estimates from different measures of exposure to risk factors that are in addition to the 40 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 Estimating attribution to risk factors.

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).

Risk factors not included in the ABDS 2018

Sub-optimal breastfeeding and exposure to lead (included in the GBD) were not included in the ABDS 2018 as sub-optimal breastfeeding was linked in the global studies to intestinal infection diseases that are not common in Australia. Exposure to lead was also excluded because exposure data (estimates of bone lead levels) were not available for Australia.

Social determinants of health (the economic and social conditions—such as income, level of education and employment status—that influence health status) could not be included as risk factors in the current study. 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 and data 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 blood 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 blood plasma glucose. The risk factors and linked diseases selected for the ABDS 2018 risk factors are shown in risk factor specific methods.

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 2018 adopted relevant linked diseases used in the GBD 2019 (GBD 2019 Risk Factors Collaborators 2020) and those identified by the AIHW from literature reviews for selected risk factors as part of extension projects (AIHW 2016a, 2017a, 2017b, 2018). 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 2019, a literature review and expert advice. The risk factor diet high in sodium was linked to other risk factors (high blood pressure), which then linked to diseases. The impact was estimated by the amount sodium consumption influences blood pressure and therefore the diseases linked to high blood pressure.

Dementia was linked to risk factors where it was identified by the AIHW as having sufficient evidence (risk factor specific methods, AIHW 2016b).

Additional material

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 2.4. 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 2.4: 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).

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.

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 assessment matrix below.

- 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.
- 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 2.8: Assessment matrix for exposure data to be used in the ABDS 2018

Rating	High	Medium	Low
Comparability	Consistent	Comparable	Inconsistent
Relevance - National study	National	Sub-national	Sub- or super-regional
Relevance - Indigenous study	Indigenous	Sub-national or Total Australian or non-Indigenous	Other Indigenous population (New Zealand, United States, Canada)
Currency	2013 or later	2007–2013	Before 2007
Accuracy - coverage	More than 90%	60%–90%	Less than 60%
Accuracy - sampling	Less than 25% RSE	25%–50% RSE	More than 50% RSE
Accuracy - measurement	Clinically reported or measured	Self-reported	Not known
Validation	Validated	Validated or not validated	Not validated
Credibility	Published and Peer reviewed	Published but not peer reviewed or not published but peer reviewed	Not published nor peer reviewed
Accessibility/ timeliness	Currently available	Expected to be available in time for analysis	Unlikely to be available in time for analysis.

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Estimating burden of disease measures

Years of life lost (YLL)

On this page:

Estimating the fatal burden

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 2018, YLL estimates were based on deaths that occurred in the reference years: 2003, 2011, 2015 and 2018.

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.

Box 3.1: 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 *Estimating the non-fatal burden*) 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:

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

where:

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

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 About 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). 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 and Causes of death, Australia.

All deaths data used in the ABDS 2018 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').

Mortality data in ABDS 2018

Cause of death data for deaths occurring in 2003, 2011, 2015 and 2018 were used for this analysis. Deaths for the four reference years were extracted from the NMD for deaths registered in 2003 up to and including deaths registered in 2019. As a result, the analysis set includes deaths that occurred in 2018 but were not registered until 2019; 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 2019).

Deaths for the 2003, 2011 and 2015 reference years are almost all (at least 99%) based on a final version of cause of death data and most (95%) for 2018 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 2019).

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.

Indigenous identification

Due to small numbers, analysis of indigenous mortality was an average of the three years around the reference year. A separate calculation of non-indigenous burden was calculated with the three years of data.

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 Overarching methods and choices for ABDS 2018).

Deaths data used in the ABDS 2018 are coded to the ICD-10 (ABS 2019; WHO 2016). The procedure for assigning ICD-10 coded death records to items in the ABDS disease list is set out in the next section.

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. Table 2.1 provides a list of ICD-10 codes for each disease used for the estimates of fatal burden in the ABDS 2018.

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 2018 largely align with those used in the ABDS 2015. The table below 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.

Table 3.1: Number and proportion of deaths by redistribution group, method and target diseases, 2018

Redistribution group	ICD-10 codes	Method	Scope of target diseases(a)	Number	Proportion (%)
All other non-specific, intermediate and immediate causes	B19.9, E86, E87.0, E87.1, E87.2, E87.5, E87.6, E87.7, E87.8, G81.9, G82.1, G82.2, G82.4, G82.5, G83.0, G83.4, G83.5, G83.8, G83.9, H05.0, H16.9, H40.9, H44.0, H49.8, H57.8 , H60.9, H81.1, H91.1, H91.9, I46.1, I46.9, I49.0, J96.0, J96.1, J96.9, L27.0, L43.9, L53.8, L53.9, L73.9, L90.5, L92.9, L98.9, N61, N70.9, N73.5, N73.9, N84.0, N85.9.	Proportional allocation	All diseases	1,506	9.6
Cardiac signs and symptoms, unspecified digestive diseases and congenital anomalies	I70.9, Q66.0, Q67.5, Q67.6, Q82.0, Q89.9, Q99.9, R02	Proportional allocation	All diseases excluding infections, cancer and injuries	100	0.6
Heart failure	I50.0, I50.1, I50.9	Direct evidence and indirect MCOD	Cardiovascular, infant/congenital	2,742	17.5
Hypertension	I10, I13.0, I13.1, I13.2, I13.9	Indirect MCOD	All diseases excluding injuries	875	5.6
Non-specific cancers	C76.0, C76.1, C76.2, C76.3, C76.4, C76.5, C80.0, C80.9	Direct evidence	Cancer	2,917	18.6
Non-specific digestive cancers	C26.1, C26.8, C26.9	Direct evidence	Cancer (digestive cancers)	159	1.0
Peritonitis	K65.0, K65.8, K65.9, K66.0, K66.1, K66.8, K66.9	Direct evidence and indirect MCOD	Gastrointestinal	79	0.5
Pneumonitis	J69.0	Indirect MCOD	All diseases	960	6.1
Renal failure	N17.0, N17.9, N19	Direct evidence and indirect MCOD	Partial kidney/urinary, all diseases	777	5.0
Septicaemia	A40.0, A40.2, A40.8, A40.9, A41.0, A41.2, A41.4, A41.5, A41.8, A41.9	Indirect MCOD	All diseases	1,436	9.2
Undetermined intent	Y10, Y11, Y12, Y13, Y14, Y15, Y17, Y19, Y20, Y21, Y23, Y24, Y26, Y28, Y29, Y30, Y31, Y32, Y33, Y34	Direct evidence	All injuries	226	1.4
Unknown causes	R99	Direct evidence	All diseases	1,281	8.2
Unspecified amyloidosis, unspecified respiratory signs and symptoms and cachexia	E85.4, E85.8, E85.9, R05, R06.0, R06.8, R64	Proportional allocation	All diseases excluding injuries	231	1.5
Unspecified diabetes	E14.0, E14.1, E14.4, E14.5, E14.6, E14.7, E14.8, E14.9	Direct evidence	Type 1, Type 2 and Other diabetes	1,637	10.4
Unspecified factor	X59.0, X59.9	Proportional allocation	Injuries	233	1.5
Unspecified gastrointestinal causes	K92.0, K92.1, K92.2, K92.8, K92.9	Direct evidence and indirect MCOD	Gastrointestinal	516	3.3
All redistribution causes				**15,675**	**100**

(a) Reproductive/maternal, oral and hearing/vision disease groups are excluded from the scope of target diseases, due to small numbers of deaths in these disease groups.

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 2018 has extended these methods using Australian-specific data and Australian-specific direct evidence.

Three methods were used for redistribution in the ABDS 2018:

- **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 MCOD (or a combination of both). In the ABDS 2018, 87% 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 (13%) were redistributed using this method (Table 3.1).

Changes made to ABDS 2018 YLL calculations since ABDS 2015 include:

1. C26.0 deaths were assigned to bowel cancer, instead of being redistributed as part of the ABDS algorithm for ill-defined digestive cancers. This aligns with cancer mortality reporting and practice by the ABS.
2. Updated algorithms were used for cancer of other and ill-defined digestive organs (C26–excluding C26.0) and cancers of ill-defined, secondary unknown primary sites (C76–C80). Their redistributed were based on direct evidence from the Western Australian Cancer Registry.
3. Updated algorithm for septicaemia. Changes to selection rules for coding causes of death in recent years have allowed more chronic conditions, such as cancers, coded to Part 2 of the death certificate (associated causes), to be selected as the underlying cause of death when septicaemia appears in Part 1 (underlying cause) of the death certificate. Following discussion with mortality data experts, deaths recorded with septicaemia as the underlying cause were not redistributed to selected chronic conditions, but instead to more acute conditions, such as urinary tract infections.
4. Redistribution of ICD-10 code X59 Exposure to unspecified factor. Previously these were redistributed proportionately across injuries. Using similar methods to AIHW injury reports, we used associated causes of death (fracture codes) to identify additional falls (2,766 deaths), thereby resulting in less X59 deaths being needed to be redistributed (727 deaths).

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 2018, 15,675 deaths were identified for redistribution in the 2018 reference year, equating to 201,561 YLL. This amounted to 9.8% of deaths and 8.5% of YLL. The number and per cent of deaths redistributed and the associated YLL for each reference year are in the table below.

Table 3.2: 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	131,994	12,557	9.5	2,219,586	178,926	8.1
2011	146,784	15,011	10.2	2,272,748	195,654	8.6
2015	157,285	14,514	9.2	2,363,686	179,083	7.6
2018	159,300	15,675	9.8	2,370,341	201,561	8.5
All years	**595,363**	**57,757**	**9.7**	**9,226,360**	**755,224**	**8.2**

The number of deaths identified for redistribution varied with age (see table below). These 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.

Table 3.3: Number of deaths identified for redistribution and associated YLL, by age and sex, 2018

Age group	Female deaths	Male deaths	Person deaths	Female YLL	Male YLL	Person YLL
Under 1	31	47	78	2,667	4,043	6,710
1–4	8	13	21	680	1,099	1,779
5–9	1	4	5	79	312	391
10–14	2	7	9	147	518	665
15–19	6	22	28	414	1,510	1,924
20–24	19	21	40	1,220	1,350	2,570
25–29	23	53	76	1,368	3,143	4,510
30–34	33	57	90	1,792	3,109	4,901
35–39	33	73	106	1,636	3,596	5,232
40–44	67	94	161	2,972	4,191	7,163
45–49	89	161	250	3,518	6,399	9,917
50–54	117	178	295	4,117	6,235	10,352
55–59	179	288	467	5,470	8,779	14,249
60–64	209	398	607	5,398	10,297	15,695
65–69	322	500	822	6,868	10,698	17,566
70–74	463	685	1,148	7,899	11,881	19,780
75–79	641	815	1,456	8,412	10,757	19,168
80–84	1,045	1,149	2,194	10,000	11,002	21,003
85–89	1,585	1,403	2,988	10,349	9,249	19,597
90–94	1,894	1,155	3,049	8,281	5,105	13,386
95–99	1,001	451	1,452	2,977	1,352	4,330
100 and over	266	67	333	536	139	675
All ages	**8,034**	**7,641**	**15,675**	**86,798**	**114,763**	**201,561**

Source: AIHW National Mortality Database

Table 3.4 shows the number of deaths classified to disease groups before and after redistribution. The largest numbers of deaths gained by redistribution were for:

- cardiovascular (5,163 more deaths, an increase of 14%)
- cancer (3,893 more deaths, an increase of 8.7%)
- endocrine (1,762 more deaths, an increase of 76%).

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 (145 more deaths, an increase of 25%)
- gastrointestinal (1,038 more deaths, an increase of 19%)
- kidney and urinary diseases (449 more deaths, an increase of 14%).

The impact of redistribution on YLL is also shown in Table 3.4. The largest number of YLL gained was for:

- cancers (54,912 more YLL, a 7.3% increase)
- cardiovascular (47,763 more YLL, an 11% increase)
- injuries (29,023 more YLL, a 9.2% increase).

Other large percentage gains in YLL were for:

- endocrine disorders (24,187 more YLL, a 78% increase)
- skin (1,282 more YLL, a 23% increase)

- mental and substance use disorders (1,366 more YLL, a 14% increase).

Note that the majority of these increases were based on targeted redistribution using direct evidence or indirect MCOD. To illustrate the method underlying the redistribution of deaths and its impact, Box 3.2 steps through the number and type of deaths that were redistributed into the cancer disease group for 2018 YLL estimates.

Table 3.4: Number and proportion of deaths before and after redistribution and associated change (increase), by disease group: National

Disease group		Deaths	% deaths	YLLs	% YLLs
Blood/metabolic	Before redistribution	1,669	1.0	30,035	1.3
	After redistribution	1,797	1.1	32,064	1.4
	Increase (before to after)	128	7.6	2,029	6.8
Cancer	Before redistribution	44,777	28.1	756,812	31.9
	After redistribution	48,670	30.6	811,723	34.2
	Increase (before to after)	3,893	8.7	54,912	7.3
Cardiovascular	Before redistribution	37,895	23.8	440,121	18.6
	After redistribution	43,058	27	487,884	20.6
	Increase (before to after)	5,163	13.6	47,763	10.9
Endocrine	Before redistribution	2,311	1.5	31,227	1.3
	After redistribution	4,073	2.6	55,414	2.3
	Increase (before to after)	1,762	76.3	24,187	77.5
Gastrointestinal	Before redistribution	5,504	3.5	88,924	3.8
	After redistribution	6,542	4.1	99,735	4.2
	Increase (before to after)	1,038	18.9	10,811	12.2
Infant/congenital	Before redistribution	1,211	0.8	82,371	3.5
	After redistribution	1,297	0.8	88,091	3.7
	Increase (before to after)	86	7.1	5,720	6.9
Infections	Before redistribution	5,381	3.4	55,033	2.3
	After redistribution	5,899	3.7	61,324	2.6
	Increase (before to after)	518	9.6	6,291	11.4
Injuries	Before redistribution	10,607	6.7	315,632	13.3
	After redistribution	11,607	7.3	344,655	14.5
	Increase (before to after)	1,000	9.4	29,023	9.2
Kidney/urinary	Before redistribution	3,113	2	34,956	1.5
	After redistribution	3,562	2.2	38,822	1.6
	Increase (before to after)	449	14.4	3,867	11.1
	Before redistribution	485	0.3	9,926	0.4

Mental health and substance use disorders	After redistribution	551	0.3	11,292	0.5
	Increase (before to after)	66	13.7	1,366	13.8
Musculoskeletal	Before redistribution	1,375	0.9	16,973	0.7
	After redistribution	1,524	1	18,616	0.8
	Increase (before to after)	149	10.9	1,643	9.7
Neurological	Before redistribution	18,666	11.7	173,269	7.3
	After redistribution	19,599	12.3	181,612	7.7
	Increase (before to after)	933	5	8,343	4.8
Oral	Before redistribution	34	0	360	0
	After redistribution	34	0	360	0
	Increase (before to after)	0	0	0	0
Reproductive/maternal	Before redistribution	50	0	1,214	0.1
	After redistribution	50	0	1,234	0.1
	Increase (before to after)	0	0.8	20	1.6
Respiratory	Before redistribution	9,972	6.3	126,308	5.3
	After redistribution	10,316	6.5	130,614	5.5
	Increase (before to after)	344	3.5	4,306	3.4
Skin	Before redistribution	575	0.4	5,620	0.2
	After redistribution	720	0.5	6,902	0.3
	Increase (before to after)	145	25.3	1,282	22.8
Redistribution	Before redistribution	15,675	9.8	201,561	8.5
	After redistribution	0	0	0	0
	Increase (before to after)	. .	. .	. .	. .
All deaths	Before redistribution	159,300	100	2,370,341	100
	After redistribution	159,300	100	2,370,341	100
	Increase (before to after)	. .	. .	. .	. .

Box 3.2: How redistribution works

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

Table 3.4 shows 44,777 deaths were coded to a cancer in the ABDS disease list. After redistribution, there were 48,670 cancer deaths, reflecting a gain of 3,893 deaths, or an additional 8.7%.

Table 3.1 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 2018, 2,917 deaths were coded to a non-specific type of cancer, and 159 deaths were coded to a non-specific digestive cancer. So, in total, 3,076 non-specific cancer deaths were identified for redistribution into a cancer cause.

So far, 79% of the overall gain in cancer deaths (3,076 out of the overall 3,893) has come from deaths initially coded to (non-specific) cancer-related causes, which have been redistributed into (specific) cancers in the ABDS disease list.

Table 3.1 also shows a further 1,506 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 Table 3.4, pre-redistribution, 28% of deaths were cancers, so about 28% of the 1,506 deaths (equivalent to around 423 deaths) were also redistributed to a specific cancer.

The foregoing redistribution steps account for around 90% of the overall gain in cancer deaths (3,076 plus 423 deaths).

The remaining 10% of the gain (393 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 Table 3.1.

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 2018 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.

More recent global estimates of YLL are based on a newer life table—the Theoretical Minimum Risk Life Table (TMRLT) (GBD 2017 Causes of Death Collaborators 2018). This life table is based on the lowest observed age-specific mortality rates from locations with total populations greater than 5 million in 2016. From this life table, life expectancy at birth is 87.9 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 2018. This is consistent with the previous ABDS.

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. Table 3.5 shows the GBD standard life expectancies for each age at death.

Table 3.5: YLL, by age at death used in the ABDS 2018

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.

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 2017–2019 was 80.9 and 85.0 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.

Table 3.6: Expected years of life remaining at selected ages, GBD standard reference and Australian life tables, by sex

Age (years)	GBD 2010 standard - Persons	Australia 2003 - Males	Australia 2003 - Females	Australia 2011 - Males	Australia 2011 - Females	Australia 2015 - Males	Australia 2015 - Females	Australia 2018 - Males	Australia 2018 - Females
0	86.0	78.1	83.0	79.9	84.3	80.4	84.6	80.9	85.0
1	85.2	77.5	82.4	79.3	83.5	79.7	83.8	80.1	84.3
5	81.3	73.6	78.5	75.3	79.6	75.8	79.9	76.2	80.3
15	71.3	63.7	68.5	65.4	69.7	65.9	70.0	66.3	70.4
25	61.4	54.1	58.7	55.7	59.8	56.2	60.1	56.6	60.5
45	41.8	35.2	39.3	36.7	40.4	37.1	40.6	37.5	41.1
65	23.3	17.8	21.1	19.1	22.0	19.6	22.3	20.0	22.7
75	14.8	10.8	13.2	11.7	13.8	12.1	14.0	12.4	14.4
85	7.6	5.7	6.9	6.1	7.2	6.2	7.3	6.4	7.6

95	3.3	3.1	3.6	3.1	3.4	3.0	3.3	3.2	3.6
100	2.2	2.5	2.8	2.3	2.5	2.1	2.3	2.2	2.7
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, 2014–2016 for 2015 and 2017–2019 for 2018.
2. Australian (2003, 2011, 2015 and 2018) life expectancies for age 100 shown here are for all ages 100 and over.

Sources: Murray et al. 2012; ABS 2005, 2013b, 2017, 2020.

Indigenous mortality data

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

Dealing with small numbers

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

To reduce the impact of random fluctuations, Indigenous YLL estimates were based on the annual average of 3 years of deaths data. For the 2003 reference year, deaths were averaged from deaths occurring in 2002, 2003 and 2004. For the 2011 reference year, deaths were averaged from 2010, 2011 and 2012. For the 2018 reference year, deaths were averaged from 2016, 2017 and 2018.

Adjusting for Indigenous under-identification

Every year, a number of deaths of Aboriginal and Torres Strait Islander people are not identified as such when registered (ABS 2013a). This might arise from the non-reporting of a deceased person's Indigenous status on the death registration form, or from incorrect identification of a deceased person's Indigenous status (recording a person as non-Indigenous when they are Indigenous, and vice versa). The net effect is an under-identification of Aboriginal and Torres Strait Islander people in the deaths data.

Adjustment factors to account for Indigenous under-identification in death registration records have been produced from national and state/territory data linkage studies. These studies include the ABS Census Data Enhancement Study (CDE) (ABS 2013c; 2018c) and the AIHW Enhanced Mortality Database (EMD) study (AIHW 2012; 2017).

Based on the results from a series of sensitivity analyses on the impact of using the different mortality adjustment factors, and discussion with the study reference group, the approach taken for the 2018 reference year was to use adjustment factors from the ABS 2016 CDE Study (ABS 2018c) to adjust Indigenous deaths for YLL estimates and gap measures. The ABS adjustment factors take into account under-identification in both mortality and population data and therefore, in theory, provide consistency in the numerator and denominator used in Indigenous YLL calculations. It should be noted, however, that while the ABS Australia-level adjustment factors are provided for 3 age groups, the state and remoteness factors are not provided by age. Given that both the ABS and AIHW studies have shown that under-identification of deaths does vary by age (and to a lesser extent, by sex), the lack of age- and sex-specific adjustment factors is a limitation of this approach.

The adjustment factors used for the 2003 and 2011 reference years in the ABDS 2018 were the same as those used in the ABDS 2011, that is, factors from the ABS 2011 CDE Study for the national, state/territory and socioeconomic levels, and factors from the AIHW EMD study for remoteness levels.

Table 3.7: Indigenous mortality adjustment factors used in the ABDS 2018

	ABS 2010–2012 CDE study adjustment factor	ABS 2015–2017 CDE study adjustment factor	AIHW EMD 2008–2010 study adjustment factor
2018 Indigenous national and 2018 Indigenous socioeconomic group estimates			
0–14 years	. .	1.19	. .
15–59 years	. .	1.07	. .
60 years and over	. .	1.09	. .
Total	. .	1.08	. .
2018 Indigenous state/territory estimates			
New South Wales	. .	1.46	. .
Queensland	. .	0.97	. .
Western Australia	. .	1.06	. .
Northern Territory	. .	0.96	. .
2018 Indigenous remoteness estimates			
Major cities	. .	1.33	. .
Inner regional	. .	1.30	. .

Outer regional	. .	1.30	. .
Remote	. .	1.05	. .
Very remote	. .	1.05	. .
2003 and 2011 Indigenous national and 2011 Indigenous socioeconomic group estimates			
0–14 years	1.21	. .	. .
15–59 years	1.12	. .	. .
60 years and over	1.29	. .	. .
Total	1.21	. .	. .
2011 Indigenous state/territory estimates			
New South Wales	1.42	. .	. .
Queensland	1.24	. .	. .
Western Australia	1.14	. .	. .
Northern Territory	0.96	. .	. .
2011 Indigenous remoteness estimates			
Major cities	. .	. .	1.25
Inner regional	. .	. .	1.22
Outer regional	. .	. .	1.12
Remote	. .	. .	1.04
Very remote	. .	. .	1.02

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 2016 (ABS 2018a). In this study, remoteness areas were aligned to the ABS 2016 geography standard, for deaths that occurred in 2015 or 2018. Deaths that occurred in 2011 were aligned to the ABS 2011 geography standard.

Deaths where there was insufficient information to ascribe a remoteness area were excluded from the sub-national analysis. These amounted to less than 0.8% of deaths in any one reference year.

Socioeconomic group

As discussed in Overarching methods and choices for ABDS 2018, the ABDS did not have information on socioeconomic status at the individual level. Instead, the ABDS 2018 derived population-based socioeconomic quintiles from the 2016 Index of Relative Socio-Economic Disadvantage of the SEIFA index (ABS 2018b), which is based on the socioeconomic characteristics of the deceased person's area of usual residence. These were applied for deaths that occurred in 2015 and 2018. For deaths in 2011, the 2011 Index of Relative Socio-Economic Disadvantage was used.

Death records with an unknown or non-specific geographical location were excluded from the analysis. These amounted to less than 0.8% of deaths in any one reference year.

Indigenous subnational estimates

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

For Indigenous YLL estimates by state and territory (reported for New South Wales, Queensland, Western Australia and the Northern Territory), and for estimates by remoteness in 2018, deaths were adjusted for Indigenous under-identification using state/territory specific adjustment factors from the ABS Census Data Enhancement study.

For Indigenous YLL estimates by remoteness in 2011 and 2003, remoteness specific adjustment factors from the AIHW's Enhanced Mortality Database project were used to adjust Indigenous deaths.

For Indigenous YLL estimates by level of socioeconomic disadvantage, deaths were adjusted using the national age-specific adjustment factors from the Census Data Enhancement study. The Indigenous Relative Socioeconomic Outcomes Index was used to classify Indigenous deaths into socioeconomic groups (Biddle & Markham 2017).

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Estimating burden of disease measures

Years lived with disability (YLD)

On this page:

Estimating the non-fatal burden

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 2018 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.

Box 3.3: 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 Years of life lost (YLL)) represents the fatal component.

The findings of the ABDS 2018 are reported for 218 diseases, including two reporting categories for injuries—'External cause of injury' and 'Nature of injury', that constitute the disease list for the study (see ABDS 2018 list of diseases).

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 sequelae/ severity.
4. Calculate YLD for each disease, which is estimated up from the sequela level (for each age and sex), described as:

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

where:

$\sum_i$ is the sum over all sequelae

i is an index for sequela

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 2018 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 2018 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

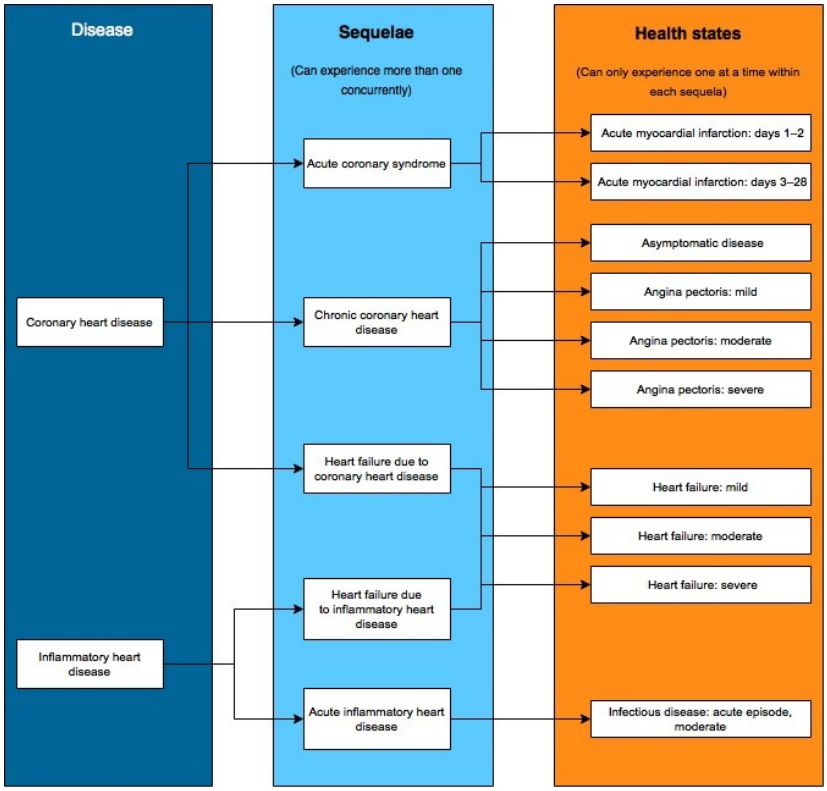
One or more sequelae were defined for each disease in the disease list. Due to the difficulty of assembling data with the granularity and dimensions required for YLD estimation, only sequelae causing significant health loss were included in the conceptual models.

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 the figure below. The list of sequelae for each disease and resultant health states are summarised in the disease-specific sections.

Figure 3.1: 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, 2015 and 2018.

The 351 sequelae in the ABDS 2018 were mapped to 195 of the 236 available health states (see ABDS 2018 health states and disability weights). This resulted in 745 sequela–health state combinations that included the different severity levels (such as mild, moderate and severe).

Table 3.8: ABDS 2018 health states and disability weights

ABDS 2018 health state ID	Health state name	Disability weight
1	Infectious disease: acute episode, mild	0.006
2	Infectious disease: acute episode, moderate	0.051
3	Infectious disease: acute episode, severe	0.133
4	Infectious disease: post-acute consequences (fatigue, emotional lability, insomnia)	0.219
5	Diarrhoea: mild	0.074
6	Diarrhoea: moderate	0.188
7	Diarrhoea: severe	0.247
8	Epididymo-orchitis	0.128
9	Herpes zoster	0.058
10	HIV: symptomatic, pre-AIDS	0.274
11	HIV/AIDS: receiving antiretroviral treatment	0.078
12	AIDS: not receiving antiretroviral treatment	0.582

13	Intestinal nematode infections: symptomatic	0.027
14	Lymphatic filariasis: symptomatic	0.109
15	Ear pain	0.013
16	Tuberculosis: without HIV infection	0.333
17	Tuberculosis: with HIV infection	0.408
18	Cancer: diagnosis and primary therapy	0.288
19	Cancer: metastatic	0.451
20	Mastectomy	0.036
21	Stoma	0.095
22	Terminal phase: with medication (for cancers, end-stage kidney or liver disease)	0.54
23	Terminal phase: without medication (for cancers, end-stage kidney or liver disease)	0.569
24	Acute myocardial infarction: days 1–2	0.432
25	Acute myocardial infarction: days 3–28	0.074
26	Angina pectoris: mild	0.033
27	Angina pectoris: moderate	0.08
28	Angina pectoris: severe	0.167
29	Cardiac conduction disorders and cardiac dysrhythmias	0.224
30	Claudication	0.014
31	Heart failure: mild	0.041
32	Heart failure: moderate	0.072
33	Heart failure: severe	0.179
34	Stroke: long-term consequences, mild	0.019
35	Stroke: long-term consequences, moderate	0.07
36	Stroke: long-term consequences, moderate plus cognition problems	0.316
37	Stroke: long-term consequences, severe	0.552
38	Stroke: long-term consequences, severe plus cognition problems	0.588
39	Diabetic foot	0.02
40	Diabetic neuropathy	0.133
41	Chronic kidney disease (stage IV)	0.104
42	End-stage renal disease: with kidney transplant	0.024
43	End-stage renal disease: on dialysis	0.571
44	Decompensated cirrhosis of the liver	0.178
45	Gastric bleeding	0.325
46	Crohn's disease or ulcerative colitis	0.231
47	Benign prostatic hypertrophy: symptomatic	0.067
48	Urinary incontinence	0.139

49	Impotence	0.017
50	Infertility: primary	0.008
51	Infertility: secondary	0.005
52	Asthma: controlled	0.015
53	Asthma: partially controlled	0.036
54	Asthma: uncontrolled	0.133
55	Chronic obstructive pulmonary disease (COPD) and other chronic respiratory diseases: mild	0.019
56	COPD and other chronic respiratory diseases: moderate	0.225
57	COPD and other chronic respiratory diseases: severe	0.408
58	Dementia: mild	0.069
59	Dementia: moderate	0.377
60	Dementia: severe	0.449
61	Headache: migraine	0.441
62	Headache: tension-type	0.037
63	Multiple sclerosis: mild	0.183
64	Multiple sclerosis: moderate	0.463
65	Multiple sclerosis: severe	0.719
70	Parkinson disease: mild	0.01
71	Parkinson disease: moderate	0.267
72	Parkinson disease: severe	0.575
73	Alcohol use disorder: mild	0.235
74	Alcohol use disorder: moderate	0.373
75	Alcohol use disorder: severe	0.57
76	Fetal alcohol syndrome: mild	0.016
77	Fetal alcohol syndrome: moderate	0.056
78	Fetal alcohol syndrome: severe	0.179
79	Cannabis dependence	0.266
80	Amphetamine dependence	0.486
81	Cocaine dependence	0.479
82	Heroin and other opioid dependence	0.697
83	Anxiety disorders: mild	0.03
84	Anxiety disorders: moderate	0.133
85	Anxiety disorders: severe	0.523
86	Major depressive disorder: mild episode	0.145
87	Major depressive disorder: moderate episode	0.396
88	Major depressive disorder: severe episode	0.658
89	Bipolar disorder: manic episode	0.492

90	Bipolar disorder: residual state	0.032
91	Schizophrenia: acute state	0.778
92	Schizophrenia: residual state	0.588
93	Anorexia nervosa	0.224
94	Bulimia nervosa	0.223
95	Attention deficit hyperactivity disorder	0.045
96	Conduct disorder	0.241
97	Asperger syndrome	0.104
98	Autism	0.262
99	Intellectual disability: mild	0.043
100	Intellectual disability: moderate	0.1
101	Intellectual disability: severe	0.16
102	Intellectual disability: profound	0.2
103	Hearing loss: mild	0.01
104	Hearing loss: moderate	0.027
105	Hearing loss: severe	0.158
106	Hearing loss: profound always	0.204
107	Hearing loss: complete	0.215
108	Hearing loss: mild, with ringing	0.021
109	Hearing loss: moderate, with ringing	0.074
110	Hearing loss: severe, with ringing	0.261
111	Hearing loss: profound, with ringing	0.277
112	Hearing loss: complete, with ringing	0.316
113	Distance vision: mild impairment	0.003
114	Distance vision: moderate impairment	0.031
115	Distance vision: severe impairment	0.184
116	Distance vision blindness	0.187
117	Near vision impairment	0.011
126	Musculoskeletal problems: legs, mild	0.023
127	Musculoskeletal problems: legs, moderate	0.079
128	Musculoskeletal problems: legs, severe	0.165
129	Musculoskeletal problems: arms, mild	0.028
130	Musculoskeletal problems: arms, moderate	0.117
131	Musculoskeletal problems: generalised, moderate	0.317
132	Musculoskeletal problems: generalised, severe	0.581
133	Gout: acute	0.295
134	Amputation of finger(s), excluding thumb: long term, with treatment	0.005
135	Amputation of thumb: long term	0.011

137	Amputation of both arms: long term, with treatment	0.123
138	Amputation of both arms: long term, without treatment	0.383
139	Amputation of toe	0.006
140	Amputation of one leg: long term, with treatment	0.039
141	Amputation of one leg: long term, without treatment	0.173
142	Amputation of both legs: long term, with treatment	0.088
143	Amputation of both legs: long term, without treatment	0.443
144	Burns of <20% total surface area without lower airway burns: short term, with or without treatment	0.141
145	Burns of <20% total surface area or <10% total surface area if head or neck, or hands or wrist involved: long term, with or without treatment	0.016
146	Burns of >=20% total surface area: short term, with or without treatment	0.314
147	Burns of >=20% total surface area or >=10% total surface area if head or neck, or hands or wrist involved: long term, with treatment	0.135
148	Burns of> =20% total surface area or >=10% total surface area if head or neck, or hands or wrist involved: long term, without treatment	0.455
149	Lower airway burns: with or without treatment	0.376
150	Crush injury: short or long term, with or without treatment	0.132
151	Dislocation of hip: long term, with or without treatment	0.016
152	Dislocation of knee: long term, with or without treatment	0.113
153	Dislocation of shoulder: long term, with or without treatment	0.062
154	Other injuries of muscle and tendon (includes sprains, strains, and dislocations other than shoulder, knee, or hip)	0.008
155	Drowning and non-fatal submersion: short or long term, with or without treatment	0.247
156	Fracture of clavicle, scapula, or humerus: short or long term, with or without treatment	0.035
157	Fracture of face bone: short or long term, with or without treatment	0.067
158	Fracture of foot bones: short term, with or without treatment	0.026

159	Fracture of foot bones: long term, without treatment	0.026
160	Fracture of hand: short term, with or without treatment	0.01
161	Fracture of hand: long term, without treatment	0.014
162	Fracture of neck of femur: short term, with or without treatment	0.258
163	Fracture of neck of femur: long term, with treatment	0.058
164	Fracture of neck of femur: long term, without treatment	0.402
165	Fracture other than neck of femur: short term, with or without treatment	0.111
166	Fracture other than neck of femur: long term, without treatment	0.042
167	Fracture of patella, tibia or fibula, or ankle: short term, with or without treatment	0.05
168	Fracture of patella, tibia or fibula, or ankle: long term, with or without treatment	0.055
169	Fracture of pelvis: short term	0.279
170	Fracture of pelvis: long term	0.182
171	Fracture of radius or ulna: short term, with or without treatment	0.028
172	Fracture of radius or ulna: long term, without treatment	0.043
173	Fracture of skull: short or long term, with or without treatment	0.071
174	Fracture of sternum or fracture of 1 or 2 ribs: short term, with or without treatment	0.103
175	Fracture of vertebral column: short or long term, with or without treatment	0.111
176	Fractures: treated, long term	0.005
177	Injured nerves: short term	0.1
178	Injured nerves: long term	0.113
179	Injury to eyes: short term	0.054
180	Severe traumatic brain injury: short term, with or without treatment	0.214
181	Traumatic brain injury: long-term consequences, minor, with or without treatment	0.094
182	Traumatic brain injury: long-term consequences, moderate, with or without treatment	0.231
183	Traumatic brain injury: long-term consequences, severe, with or without treatment	0.637

184	Open wound: short term, with or without treatment	0.006
185	Poisoning: short term, with or without treatment	0.163
186	Severe chest injury: long term, with or without treatment	0.047
187	Severe chest injury: short term, with or without treatment	0.369
188	Spinal cord lesion below neck: treated	0.296
189	Spinal cord lesion below neck: untreated	0.623
190	Spinal cord lesion at neck: treated	0.589
191	Spinal cord lesion at neck: untreated	0.732
192	Abdominopelvic problem: mild	0.011
193	Abdominopelvic problem: moderate	0.114
194	Abdominopelvic problem: severe	0.324
195	Anaemia: mild	0.004
196	Anaemia: moderate	0.052
197	Anaemia: severe	0.149
198	Periodontitis	0.007
199	Dental caries: symptomatic	0.01
200	Severe tooth loss	0.067
201	Disfigurement: level 1	0.011
202	Disfigurement: level 2	0.067
203	Disfigurement: level 3	0.405
204	Disfigurement: level 1 with itch or pain	0.027
205	Disfigurement: level 2, with itch or pain	0.188
206	Disfigurement: level 3, with itch or pain	0.576
207	Generic uncomplicated disease: worry and daily medication	0.049
208	Generic uncomplicated disease: anxiety about diagnosis	0.012
209	Iodine-deficiency goitre	0.199
210	Kwashiorkor	0.051
211	Severe wasting	0.128
212	Speech problems	0.051
213	Motor impairment: mild	0.01
214	Motor impairment: moderate	0.061
215	Motor impairment: severe	0.402
216	Motor plus cognitive impairments: mild	0.031
217	Motor plus cognitive impairments: moderate	0.203
218	Motor plus cognitive impairments: severe	0.542

219	Rectovaginal fistula	0.501
233	Low back pain, moderate	0.054
234	Low back pain, mild	0.02
235	Alcohol use disorder, very mild	0.123
236	Amphetamine dependence, mild	0.079
237	Amputation of 1 upper limb (long term, with treatment)	0.039
238	Amputation of 1 upper limb (long term, without treatment)	0.118
239	Back pain, most severe, with leg pain	0.384
240	Back pain, most severe, without leg pain	0.372
241	Back pain, severe, with leg pain	0.325
242	Back pain, severe, without leg pain	0.272
243	Borderline intellectual functioning	0.011
244	Cannabis dependence, mild	0.039
245	Cocaine dependence, mild	0.116
246	Concussion	0.11
247	Distance vision, monocular	0.017
248	Epilepsy, less severe (seizures less than once per month)	0.263
249	Epilepsy, severe (seizures once per month or more)	0.552
250	Headache, medication overuse	0.223
251	Heroin and other opioid dependence, mild	0.335
252	Hyperthyroidism	0.145
253	Hypothyroidism	0.019
254	Mild low back pain with leg pain	0.02
255	Moderate low back pain with leg pain	0.054
256	Neck pain, mild	0.053
257	Neck pain, moderate	0.114
258	Neck pain, severe	0.229
259	Neck pain, most severe	0.304
260	Stress incontinence	0.02
261	Thrombocytopenic purpura	0.159
262	Asymptomatic disease	0

Source:
GBD
2015.

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 2018 estimated point prevalence as at 30 June 2018, 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. For a list of disease-specific experts, see Expert advice and review.

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 2018. 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 and NHS 2017–18 held by the ABS. For further information on these data sources, including data quality statements, see Australian Health Survey: First results, 2011–12, National Health Survey: First results, 2014–15 and National Health Survey: First results, 2017–18.

Primary data sources used for each disease are summarised in the table below.

Table 3.9: ABDS 2018 main data sources for YLD estimation

Disease group	Key national data sources
Blood & metabolic disorders	National Hospital Morbidity Database
	National Health Survey 2017–18
	Australian Health Survey 2011–12
	Australian Cystic Fibrosis Data Registry
	Australian Bleeding Disorders Registry
	Linked hospitals and NDI components for the NIHSI AA v0.5 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 NIHSI AA v0.5 database
	New Zealand Burden of Disease Study

	Epidemiological studies
Endocrine disorders	National Diabetes Register
	National Health Survey 2017–18
	Fremantle Diabetes Study
Gastrointestinal disorders	National Hospital Morbidity Database
	Linked hospitals and NDI components for the NIHSI AA v0.5 database
	Australian and New Zealand Liver Transplant Registry
	New Zealand Burden of Disease Study
	Epidemiological studies
Hearing & vision disorders	National Health Survey 2017–18
	Australian Health Survey 2011–12
	Australian Hearing Database
	Blue Mountains Hearing Study
	Melbourne Vision Impairment Project
	National Eye Health Survey
	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
Kidney and urinary diseases	Australian and New Zealand Dialysis and Transplantation Registry
	National Hospital Morbidity Database
	Australian Health Survey 2011–12
	Linked hospitals and NDI components for the NIHSI AA v0.5 database
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 2017 and 2019
Musculoskeletal conditions	National Health Survey 2017–18
Neurological conditions	National Hospital Morbidity Database
	National Health Survey 2014–15
	AIHW dementia analyses
	Epidemiological studies
	Linked hospitals and NDI components for the NIHSI AA v0.5 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 Survey 2017–18
	Burden of Obstructive Lung Disease study
	Global Burden of Disease Study
	Epidemiological studies
Skin disorders	National Health Survey 2017–18
	National Hospital Morbidity Database
	Bettering the Evaluation and Care of Health
	AIHW GEN Aged Care data
	Epidemiological studies

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, AIHW analyses of linked hospitalisations and deaths data from the NIHSI AA v0.5 was used to calculate people-to-hospitalisations ratios using linked hospital and deaths data for New South Wales, Victoria, South Australia and Tasmania for selected sequelae. These ratios were 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, Victoria, South Australia and Tasmania combined.

The NIHSI AA is a deidentified enduring linked data asset holding data from 2010–11 onwards on admitted patient care services (APC) (in public and private hospitals where available), emergency department (ED) services and outpatient (NAP) services in public hospitals for all participating states and territories, along with Medicare Benefits Schedule MBS data, Pharmaceutical Benefits Scheme PBS and Repatriation Pharmaceutical Benefits Scheme (RPBS) data, Residential Aged Care Services (RACS) data and National Deaths Index (NDI) data. The ABDS 2018 used the hospitals and the NDI data from 2010–11 to 2016–17. It did not use the MBS, PBS, RPBS or RACS components of the database. In addition to people-to-hospitalisations ratios, data from the NIHSI AA were used to derive rates to estimate point prevalence for specific sequela; details for these can be found in Disease-specific methods - morbidity.

Linked hospitals and deaths data from Western Australia were also used to calculate people-to-hospitalisations ratios using linked data for selected sequelae and for Indigenous-specific estimates. This and other usage of linked data is described in Disease-specific methods - morbidity.

Indigenous considerations

Adjusting for Indigenous under-identification in hospitals data

Indigenous Australians are under-identified in hospitals data to varying degrees across state and territory and remoteness areas. This results in an underestimate of hospitalisations of Indigenous Australians. In the ABDS 2018, hospitalisation data used to calculate Indigenous YLD estimates were adjusted for Indigenous under-identification using adjustment factors from hospital data quality studies done by the AIHW (Indigenous hospital adjustment factors in Years lived with disability (YLD)). These studies were undertaken on admitted patients in public hospitals only, and estimates were not adjusted for the casemix of patients or private hospitals. Data used for reference year 2018 were adjusted based on evidence from the 2011–12 data quality study (AIHW 2013), there being no evidence available with which to construct adjustment factors for more recent years. A sensitivity analysis undertaken by the AIHW showed that assuming changes in identification levels between 2011 and 2018 similar to those seen between the two published quality studies would result in negligible impacts on estimates of non-fatal burden derived from these data.

Table 3.10: Indigenous hospital adjustment factors by state and remoteness

State	Inner Regional	Major Cities	Outer Regional	Remote	Very Remote	ALL
2018 and 2011						
ACT	1.69	1.69	1.69	1.69	1.69	1.69
NSW	1.09	1.37	1.08	1.02	1.02	1.2
NT	1	1	1.03	0.99	1	1
Qld	1.12	1.17	1.04	0.97	0.97	1.08
SA	1.03	1.16	1.03	1	1	1.09
Tas	1.37	1.37	1.37	1.37	1.37	1.37
Vic	1.06	1.41	1.09	1.23	1.23	1.23
WA	1.02	0.99	1	1.07	1	1.01
Australia	1.11	1.21	1.04	1	1	1.09
2003						
Australia	1.11	1.25	1.06	1.03	1.03	1.12

Adjusting for poor quality of Indigenous data

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

Indirect methods for deriving Indigenous prevalence

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

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

Indirect methods were used to derive Indigenous prevalence for either the whole or part of the disease for 61 diseases across 10 disease groups. Of these, 32 (52%) used hospitalisation rate ratios, 28 (46%) used rate ratios from other data sources, and 1 (<0.5%) used Maori prevalence rates. A list of these diseases and sequelae, and the indirect methods used, can be found in the table below.

Table 3.11: Diseases for which Indigenous prevalence estimates for 2018 were derived using indirect methods

Disease[a]	Data source and indirect method
Cancer and other neoplasms	
Non-melanoma skin cancer[b]	Diagnosis and primary therapy of simple non-melanoma skin cancer: Applied Indigenous-to-national ratio of complex non-melanoma skin cancer
Ductal carcinoma in situ[b]	Mastectomy due to ductal carcinoma in situ: ratio of Indigenous to national diagnosed breast cancer of less than 2 centimetres applied to national ductal carcinoma in situ incidence
Cardiovascular diseases	
Atrial fibrillation and flutter	Applied the New Zealand Maori rates to the Indigenous population to estimate total cases
Gastrointestinal disorders	

Chronic liver disease	Liver transplant: Indigenous to non-Indigenous ratios from NHMD. Decompensated cirrhosis: person:separations ratios from NIHSI and age specific ratios from WA. Terminal chronic liver disease: person:separations ratios from NIHSI and age specific ratios from WA.
Infant and congenital conditions	
Pre-term birth and low birthweight complications[(b)]	Neurodevelopment impairment due to pre-term and low birthweight complications: Age-specific rate ratios (Indigenous to non-Indigenous) from IDEA database
Birth trauma and asphyxia	Age-specific rate ratios (Indigenous to non-Indigenous) from IDEA database
Cerebral palsy	Sex-specific rate ratios (Indigenous to non-Indigenous) from the Australian Cerebral Palsy Register, age distribution obtained from national estimates
Neural tube defects	Applied Indigenous birth prevalence rate obtained from WARDA to national estimates
Cardiovascular defects	Applied Indigenous birth prevalence rate obtained from WARDA to national estimates
Cleft lip and/or palate	Applied Indigenous birth prevalence rate obtained from WARDA to national estimates
Gastrointestinal malformations[(b)]	Acute complications due to gastrointestinal malformations: Applied Indigenous birth prevalence rate obtained from WARDA to national estimates. Incontinence due to anorectal atresia: Sex-specific hospital separation rate ratios (Indigenous to national)
Urogenital malformations	Applied Indigenous birth prevalence rate obtained from WARDA to national estimates
Down syndrome	Age-specific rate ratios (Indigenous to non-Indigenous) from IDEA database
Brain malformations	Age-specific rate ratios (Indigenous to non-Indigenous) from IDEA database
Infectious diseases	
HIV	Indigenous proportion derived from Kirby Institute modelling and applied to national estimates
Tuberculosis	Sex-specific hospital separation rate ratios (Indigenous to national)
Syphilis	Sex-specific hospital separation rate ratios (Indigenous to national)
Chlamydia	Sex-specific hospital separation rate ratios (Indigenous to national)
Gonorrhoea	Sex-specific hospital separation rate ratios (Indigenous to national)

Other sexually transmitted infections[a]	Sex-specific hospital separation rate ratios (Indigenous to national)
Hepatitis A	Sex-specific hospital separation rate ratios (Indigenous to national)
Hepatitis B (acute)	Age- and sex-specific notification rate ratios (Indigenous to national)
Hepatitis C (acute)	Age- and sex-specific notification rate ratios (Indigenous to national)
Upper respiratory tract infections	Age- and sex-specific hospital separation rate ratios (Indigenous to national)
Otitis media	Age- and sex-specific rate ratios (Indigenous to national) of self-reported chronic otitis media in children (AHS 2011–13)
Lower respiratory tract infections	Age- and sex-specific hospital separation rate ratios (Indigenous to national)
Influenza	Age- and sex-specific hospital separation rate ratios (Indigenous to national)
Pertussis	Sex-specific hospital separation rate ratios (Indigenous to national)
Measles	Sex-specific hospital separation rate ratios (Indigenous to national)
Invasive pneumococcal disease	Sex-specific hospital separation rate ratios (Indigenous to national)
Meningococcal disease	Sex-specific hospital separation rate ratios (Indigenous to national)
Other meningitis and encephalitis	Sex-specific hospital separation rate ratios (Indigenous to national)
Dengue	Sex-specific hospital separation rate ratios (Indigenous to national)
Ross River virus	Sex-specific hospital separation rate ratios (Indigenous to national)
Barmah forest virus	Sex-specific hospital separation rate ratios (Indigenous to national)
Malaria	Sex-specific hospital separation rate ratios (Indigenous to national)
Campylobacteriosis	Sex-specific hospital separation rate ratios (Indigenous to national)
Salmonellosis	Sex-specific hospital separation rate ratios (Indigenous to national)
Rotavirus	Sex-specific hospital separation rate ratios (Indigenous to national)
Other gastrointestinal infections	Age- and sex-specific hospital separation rate ratios (Indigenous to national)
Varicella	Age- and sex-specific hospital separation rate ratios (Indigenous to national)

Herpes zoster (shingles)	Age- and sex-specific hospital separation rate ratios (Indigenous to national)
Mumps	Sex-specific hospital separation rate ratios (Indigenous to national)
Urinary tract infections	Sex-specific hospital separation rate ratios (Indigenous to national)
Mental and substance use disorders	
Depressive disorders	Major depressive disorder: Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data Dysthymia: sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
Anxiety disorders	Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
Bipolar affective disorder	Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
Alcohol use disorders	Asymptomatic/very mild/mild: Age- and sex-specific hospitalisation rate ratios (Indigenous to national) Moderate/severe: Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
Drug use disorders	Cannabis dependence: Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data Amphetamine dependence and opioid dependence: Sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data Cocaine dependence: sex-specific rate ratios (Indigenous non-Indigenous) from National Drug Strategy survey data Other drug dependence: sex-specific hospitalisation rate ratios (Indigenous to national)
Schizophrenia	Age- and sex-specific rate ratios (Indigenous to national) based on Queensland linked mental health care data
Attention deficit hyperactivity disorder	Average of age-specific rate ratios (Indigenous to non-Indigenous) based on Longitudinal Study of Indigenous Children and Queensland linked mental health care data
Conduct disorder	Average of age-specific rate ratios (Indigenous to non-Indigenous) based on Longitudinal Study of Indigenous Children and Queensland linked mental health care data
Intellectual disability	Age-specific rate ratios (Indigenous to non-Indigenous) from IDEA database
Neurological conditions	

Parkinson disease	National prevalence rates and severity distribution were applied to the Indigenous population, and adjusted by a ratio based on a New Zealand study
Multiple sclerosis	National prevalence rates and severity distribution were applied to the Indigenous population, and adjusted by a ratio based on a New Zealand study
Guillain-Barré syndrome	The national persons-to-separation ratio was applied to the count of Indigenous Guillain–Barré syndrome hospital separations
Oral disorders	
Dental caries and pulpitis	Indigenous-to-national rate ratios from National Survey of Adult Oral Health 2004–06 (15 and over) and Child Dental Health Survey 2009 (less than 15) were applied to national age and sex distributions
Periodontal disease	Indigenous-to-national rate ratios from National Survey of Adult Oral Health 2004–06 were applied to national age and sex distributions.
Reproductive and maternal conditions	
Early pregnancy loss	Indigenous-to-national age-specific rate ratios from hospital separations for medical abortions applied to national rate of Medicare data, in addition to adjusted hospitalisations data
Genital prolapse	Indigenous-to-national rate ratios from hospital separations for genital prolapse applied to national rate
Skin disorders	
Ulcers	Other chronic skin ulcers: hospital rate ratio used to determine prevalence start point, then applied national pattern of prevalence by age and sex Pressure ulcers (skin): hospital rate ratios applied to total population prevalence, by age and sex

(a) Excludes residual ('other') diseases within each disease group, which also used indirect methods such as hospitalisation rate ratios in many instances (6 in total).

(b) Applicable to listed sequelae only.

A further 10 diseases used national prevalence rates to derive Indigenous prevalence for the whole disease, and an additional 10 diseases used national ratios applied to Indigenous hospitalisations or cancer incidence rates to derive Indigenous prevalence for particular sequelae (see table below).

Table 3.12: Diseases for which national rates or ratios were assumed to derive Indigenous prevalence estimates for 2018

Disease	Data source and indirect method
Cancer and other neoplasms	
Breast cancer[(a)]	Mastectomy due to breast cancer: national incidence-to-hazard ratio applied for males only
Prostate cancer[(a)]	Impotence/incontinence due to prostate cancer: national rates of treatments and outcomes for prostate cancer applied to the Indigenous 10-year prevalence of prostate cancer
Laryngeal cancer[(a)]	Laryngectomy due to laryngeal cancer: national sex-specific laryngectomy incidence hazard rates applied to the Indigenous 10-year prevalence
Bowel cancer[(a)]	Stoma due to bowel cancer: due to the small number of cases and hospitalisations, Indigenous incidence was assumed to be the same as for the national population

Bladder cancer[(a)]	Stoma/urinary incontinence due to bladder cancer: insufficient data to produce Indigenous-specific rates for the various urinary diversions, so national rates were assumed. The proportion of people experiencing incontinence due to various diversion types was assumed to be the same for the Indigenous population as the national
Brain and central nervous system cancer[(a)]	Brain injury due to brain cancer: national rates assumed
Ductal carcinoma in situ[(a)]	Mastectomy due to ductal carcinoma in situ: ratio of Indigenous to national diagnosed breast cancer less than 2cm applied to national ductal carcinoma in situ incidence
Benign and uncertain brain tumours[(a)]	Brain injury due to benign and uncertain brain tumours: national rates assumed
Endocrine disorders	
Type 1 diabetes mellitus	National age- and sex-specific rates applied in people aged 30 and over
Gastrointestinal disorders	
Inflammatory bowel disease	Assumed same prevalence rate as national
Gastro-oesophageal reflux disease	Assumed same prevalence rate as national
Functional gastrointestinal disorders	Assumed same prevalence rate as national
Mental and substance use disorders	
Eating disorders	Assumed same prevalence rate as national
Autism spectrum disorders	Assumed same prevalence rate as national
Reproductive and maternal conditions	
Endometriosis	Assumed same prevalence as total Australian population for endometriosis. Adjusted hospital separations used for severe endometriosis and subtracted from total endometriosis estimates to inform mild estimates
Infertility	Assumed same prevalence rate as total Australian population for Infertility (including all sequelae of the infertility envelope)
Other reproductive conditions	Assumed same prevalence as total Australian population for other reproductive conditions.
Polycystic ovarian syndrome	Assumed same prevalence as total Australian population for polycystic ovarian syndrome.
Skin disorders	
Acne	Assumed same prevalence rate as national
Dermatitis and eczema	Assumed same prevalence rate as national

Severity distributions

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

Where there were no empirical data on the distribution of health states within a sequela, severity distributions were adopted from the NZBDS or the GBD 2013 (where used in the ABDS 2011), GBD 2015 or GBD 2017, 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 methods section.

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

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 Disease-specific methods 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 disease-specific methods.

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.

Whist 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 YLD are derived.

Comorbidity bias adjustment in the ABDS 2018

The strategy outlined above has been adopted for the ABDS 2018. 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 2018 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 2018 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 kind have been used in recent iterations of the GBD studies and other 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, 2015 and 2018—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 2018 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 YLL-to-YLD 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 disease-specific methods.

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Estimating burden of disease measures

Risk factor attributable burden

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.

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Estimating attribution to risk factors

Estimating attribution to risk factors

Theoretical minimum risk exposure distribution

In the ABDS 2018, as in previous burden of disease studies, a theoretical minimum risk exposure distribution (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 2019 Risk Factors Collaborators 2020). 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^2 with a standard deviation of 1.

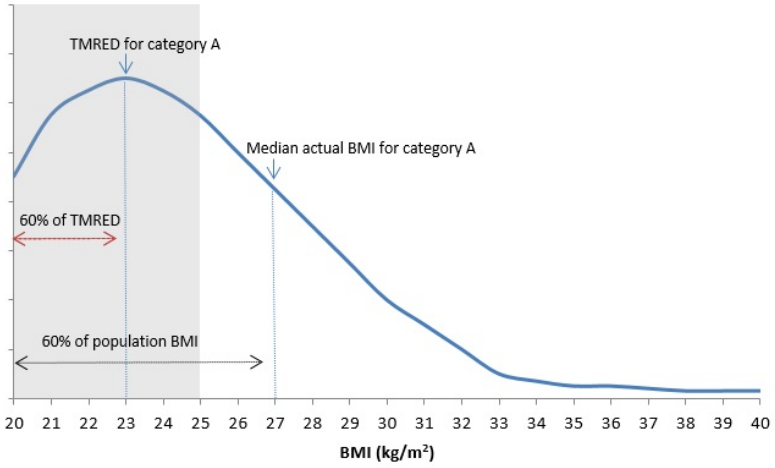
In the ABDS 2018, the TMRED for physical activity was reduced to 4,200 METs (Metabolic Equivalent of Tasks) from 8,000. The TMRED for many of the dietary risks and air pollution also changed to be a single value instead of a range of values due to different methods for GBD 2019 (see Risk factor specific methods for TMREDs).

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.

For example, if a persons' BMI is in the range 26.0–27.9 kg/m^2 (category A) and the TMRED for that person is in the range 20–25 kg/m^2 then a TMRED value is estimated by comparing the median from category A with the proportion of the population with a BMI less than that median value. That is, the median within category A is a BMI of 27.0 kg/m^2 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^2 up to 25 kg/$m^{2)}$. Assuming the TMRED distribution is uniform then the TMRED for category A is 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 3.2: 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^2 and 25 kg/m^2. 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 2018, the definitions of risk factor exposures have been adopted where possible from the GBD 2019 (GBD 2019 Risk Factors Collaborators 2020) and the AIHW review of the literature (AIHW 2017a, 2017b, 2018).

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 the data selection criteria and the scoring matrix). Only data sources that met these criteria were included in the study.

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

- ABS apparent consumption of alcohol data (national estimates only)
- ABS Labour force survey
- Australian Health Survey (AHS) 2011–12 (national estimates only)
- Australian Aboriginal and Torres Strait Islander Health Survey (AATSIHS) 2012–13 and National Aboriginal and Torres Strait Islander Health Survey (NATSIHS) 2018–19 (Indigenous estimates only)
- Census of population and housing
- National Drug Strategy Household Survey (NDSHS) 2019 (national estimates only)
- National Health Survey (NHS) 2017–18 (national estimates only)
- National HIV Register
- National Homicide Monitoring Program
- National Hospital Morbidity Database (NHMD)
- National Mortality Database (NMD)
- National Perinatal Data Collection (NPDC)
- National Perinatal Mortality Data Collection (NPMDC)
- Personal Safety Survey (PSS) 2016 (national estimates only)
- Safe Work Australia
- satellite modelled data calibrated to ground-based air monitoring stations
- The Kirby Institute annual surveillance reports
- epidemiological studies.

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 a 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 2018 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 the risk factor specific methods. 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 there were no relevant updates to the data (such as data sourced from the AHS 2011–12) the survey data was modelled where possible to reflect the change over time to estimate exposure in 2018 as described for each risk factor.

Where data were extracted directly from a survey (for example, the National Health Survey 2017–18), sex, age and exposure categories were extracted at the finest possible level of granularity within an exposure distribution. Where necessary, categories were aggregated into larger cells to conform with requirements for the clearance of minimum cell sizes.

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 2018 adopted relative risks estimated by the GBD 2019 or the AIHW review of the literature (AIHW 2017a, 2017b, 2018; GBD 2019 Risk Factors Collaborators 2020). 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 2019 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 Risk factor specific methods.

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 some 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. However, exceptions were made for overweight (including obesity), which were determined by the literature (AIHW 2017b).

Where categories of relative risk did not correspond to an equivalent exposure category, 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 had a blood pressure between 115–120 mmHg, the relative risk for the median, which is 117 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 year, sex and age group.

For most risk factors the calculation of PAF remained the same as previous ABDS studies. 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.

This formula is modified to produce separate estimates for risk factor exposures which are part of a continuous distribution, such as overweight, which is part of the risk factor overweight (including obesity). The PAF for overweight (including obesity) is the sum of the PAF for overweight (numerator includes the categories of exposure classified as overweight but not obese) and a PAF for obesity (numerator includes the categories of exposure classified as obese). The denominators for the PAFs includes categories for both overweight and obesity.

$$PAF = \frac{\sum_{ca} P_{ca}\,(RR_{ca} - 1)}{\sum_{cab} P_{cab}\,(RR_{cab} - 1) + 1} + \frac{\sum_{cb} P_{cb}\,(RR_{cb} - 1)}{\sum_{cab} P_{cab}\,(RR_{cab} - 1) + 1} \times 100$$

where:

$\sum_c$	is the sum over all categories
c	is an index for category
a	an index of categories that are within risk factor exposure a
b	an index of categories that are within risk factor exposure b
P	is prevalence
RR	is relative risk.

For selected risk factors, the PAF calculation formula was changed based on GBD 2019. This formula allows the relative risks to be protective and therefore less than 1. The risk factors using this formula are physical inactivity; diets low in vegetables, fruit, wholegrains, milk, nuts, and legumes; diets high in red meat, processed meat and polyunsaturated fats; and low birthweight & short gestation.

$$PAF = \frac{\sum_c RR_c P_c - RR_{TMRED}}{\sum_c RR_c P_c} \times 100$$

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 the PAF is sourced from the GBD study where no relative risks are published because the GBD study has a cause which is the disease due to a risk factor, such as chronic liver disease due to alcohol use. The PAF is calculated using the burden for chronic liver disease due to alcohol use as a proportion of all burden due to chronic liver disease.
- when the PAF is sourced from ABDS disease burden estimates where sequela level burden is due to a risk factor, such as drug use disorders with components made up from individual drug dependence estimates. The PAF is calculated using the sequela burden from dependence for each drug (for example cannabis dependence) as a proportion of all burden due to drug use disorders.
- 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:

$\sum_j$	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 2019 did not align to the ABDS disease list. This was due to the GBD disaggregating causes to a further level—for example, stroke, which was estimated as a single disease in the ABDS 2018, had only relative risks from the GBD 2019 for 3 sub-types of stroke (ischaemic stroke, subarachnoid haemorrhage and intracerebral haemorrhage). These could not be applied directly to the single stroke burden.

To adjust for this, data were used from a range of sources to identify the proportion of the prevalence of the ABDS disease corresponding to the available relative risk. For example, Thrift et al. (2009) found ischaemic stroke to be 77.6% 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 2019 was used. The table below describes the source of any such disaggregation and the proportion used.

Table 3.13: Proportion and method used to align GBD relative risks with the ABDS 2018 diseases

ABDS 2018 disease	GBD 2019 cause	Source of disaggregation	Proportion of ABDS disease (%)
Stroke	Ischaemic stroke	Thrift et al. 2009	0.776
Stroke	Subarachnoid haemorrhage	Thrift et al. 2009	0.607
Stroke	Intracerebral haemorrhage	Thrift et al. 2009	0.164
Chronic liver disease	Chronic liver disease due to alcohol	GBD 2019	0.216 (Males) 0.146 (Females)
Liver cancer	Liver cancer due to alcohol	GBD 2019	0.481 (Males) 0.209 (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 2019	0.138 (Males) 0.135 (Females)

Note: Percentages may not sum to 1 due to rounding.

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 YLD, YLL and DALY. For example, the PAFs for ischaemic stroke were multiplied by 0.776 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:

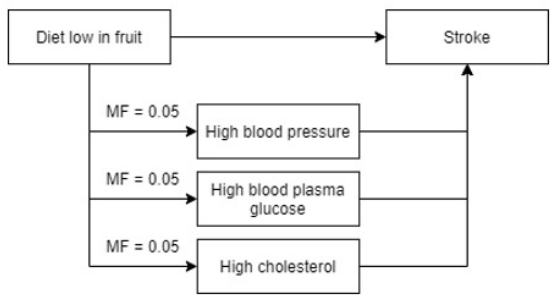
- some risk factors are on the same causal pathway—for example, a diet high in sodium increases the likelihood of high blood pressure
- the PAFs are estimated independently—similar to issues with comorbidity, the burden due to each risk factor for a given disease might exceed the total burden of that 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, mediation factors were used to attenuate the relative risk for the risk factor first in the pathway which mediate through the second risk factor in the same causal pathway for the linked disease. The relative risks for the first risk factor and the linked disease are reduced as the burden is already attributed to the risk factor/s secondary in the causal pathway. When multiple risk factors are possible secondary causal pathways for the linked disease, the attenuation factors were summed (see figure below). The attenuation factors were sourced from the GBD 2019 (GBD 2019 Risk Factors Collaborators 2020).

For example, to reflect the causal pathway of a diet low in fruit increasing the risk of stroke, mediation accounted for the diet's causal pathway towards increasing the risk of three other risk factors, which each, in turn, increase the risk of stroke.

Figure 3.3: Example of associations between a primary risk factor and linked diseases mediated by secondary risk factors in a causal pathway



Source: GBD 2019.

The amount of mediation of a diet low in fruit causing high blood pressure and then stroke was estimated to be 5% by the GBD 2019. This was summed with the mediation factors for high blood plasma glucose and high cholesterol. The relative risk for a diet low in fruit causing stroke was therefore mediated by 15%. These were then used to calculate adjusted PAFs to provide the necessary independence assumption required for the next step.

$$RR_m = \left(1 - \sum_i MF\right) \times (RR - 1) + 1$$

Where:

RR_m	is the adjusted relative risk between the primary risk factor and linked disease
RR	is the unadjusted relative risk between the primary risk factor and linked disease
MF	is the mediation factor(s) by which the secondary risk factor in the causal pathway mediates the primary risk factor
i	is the association between the first risk factor and linked disease in a causal pathway

Following mediation, 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 joint effect formula:

$$PAF_i = 1 - \prod_r (1 - PAF_{ir})$$

Where:

PAF_i	is the population attributable fraction of burden attributable to a particular disease from those risk factors being combined, such as all risk factors or all dietary risk factors
i	is the linked disease
r	is the individual risk factor for a linked disease being combined
PAF_{ir}	is the population attributable fraction for risk factor r for linked disease i
Π	is the product 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.

The use of both the joint effect and mediation formulae therefore adjusts for the interrelatedness between risk factors in the same causal pathway as well as the combined impact of all risk factors and dietary risk factors included in the study.

Attributable burden estimates by socioeconomic group

The burden attributable to risk factors was estimated by socioeconomic group in 2015 and 2018. 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 bullying victimisation, child abuse & neglect, low birth weight & short gestation, low bone mineral density, iron deficiency, sun exposure and unsafe sex 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 Risk factor specific methods.

2015, 2011 and 2003 estimates

Where possible the burden attributable to risk factors was calculated for 2015, 2011 and 2003. Exposure distributions for air pollution was estimated only for 2018 and 2015 as PM2.5 (particulate matter 2.5) estimates from satellite data was not available for the other years. Exposure to high plasma glucose could not be estimated in 2003. Bullying victimisation, child abuse & 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 new risk factor low birth weight & short gestation was only estimated in 2018.

The way exposure was estimated for risk factors in 2015, 2011 and 2003 is described in the individual risk factor section in Risk factor specific methods.

Changes in risk factor exposure over time

The Das Gupta method was used to decompose the changes in burden attributable to each risk factor into 4 additive components (Das Gupta 1993). Using a series of scenarios, this method calculates the effect of each factor on the changes over time by assuming that all other factors, except the factor under consideration, remain the same at both time points.

The change in overall attributable burden is decomposed into changes due to:

- population growth—in Australia population size is increasing over time
- population ageing—in Australia the proportion of older people is increasing over time
- risk factor exposure—changes in the prevalence of exposure to the risk factor in Australia.
- Changes in linked disease burden— changes in the overall burden for those diseases or injuries that are linked to the selected risk factor. This may be influenced by changes in diagnosis, treatment or health intervention (resulting in changes in disease prevalence or severity), as well as changes in other risk factors. For example, increases in overweight (including obesity) may have some impact on coronary heart disease burden which is also linked to tobacco use.

Attributable burden is estimated as the product of these 4 factors using the formula when examining burden by **type of exposure** to the risk factor:

$$B_t = \sum_{i=1}^{n} \sum_{j=1}^{m} P_t \times S_{jt} \times R_{ijt} \times F_{ijt}$$

where

B_t	is the amount of burden (DALY, YLL or YLD) attributable to a particular risk factor at time point *t*.
i	is a type of exposure to the risk factor such as current tobacco use
n	is all types of exposure included in the estimate for the risk factor
j	is an age and sex group
m	is all age and sex groups included (males and females aged 0 to 100+)
t	is a time point
P_t	is the total population size at time *t*
S_{jt}	is the share of the population in age and sex group *j* at the time *t*
R_{ijt}	is the rate of burden of diseases linked to exposure *i* in the age and sex group *j* at the time *t*
F_{ijt}	is the population attributable fraction of diseases linked to exposure *i* in age and sex group *j* at the time *t*
$\sum$	is the sum of all of the types of exposures *i* and all of the age and sex groups *j*

Attributable burden is estimated as the product of these 4 factors using the formula when examining burden by **linked disease group**:

$$B_t = \sum_{k=1}^{o} \sum_{j=1}^{m} P_t \times S_{jt} \times R_{kjt} \times F_{kjt}$$

where

B_t	is the amount of burden (DALY, YLL or YLD) attributable to a particular risk factor at time point *t*.
k	is a disease group of the burden linked to the risk factor
o	is all disease groups of diseases linked to the risk factor
j	is an age and sex group
m	is a age and sex groups included (males and females aged 0 to 100+)
t	is a time point
P_t	is the total population size at time *t*
S_{jt}	is the share of the population in age and sex group *j* at the time *t*
R_{kjt}	is the rate of burden of disease group *k* linked to the risk factor in the age and sex group *j* at the time *t*
F_{kjt}	is the population attributable fraction for disease group *k* in age and sex group *j* at the time *t*.
$\sum$	is the sum of all of the disease groups *k* and all of the age and sex groups *j*

The effect of each of the 4 factors—population size, population ageing, linked disease burden and risk factor exposure—using this method on the change in attributable burden between 2003 and 2018 is calculated as:

$$E_A = (B_{03} - B_{18})\left(\frac{P_{03}S_{03}R_{03}F_{03} + P_{18}S_{18}R_{18}F_{18}}{5}\right.$$
$$+ \frac{\begin{array}{c}P_{03}S_{03}R_{03}F_{18} + P_{03}S_{03}R_{18}F_{03} + P_{03}S_{18}R_{03}F_{03} + P_{18}S_{03}R_{03}F_{03} + \\ P_{18}S_{18}R_{18}F_{03} + P_{18}S_{18}R_{03}F_{18} + P_{18}S_{03}R_{18}F_{18} + P_{03}S_{18}R_{18}F_{18}\end{array}}{20}$$
$$\left. + \frac{\begin{array}{c}P_{03}S_{03}R_{18}F_{18} + P_{03}S_{18}R_{03}F_{18} + P_{03}S_{18}R_{18}F_{03} + P_{18}S_{18}R_{03}F_{03} + \\ P_{18}S_{03}R_{18}F_{03} + P_{18}S_{03}R_{03}F_{18}\end{array}}{30}\right)$$

where

E_A	is the effect of factor *A* (population size, population ageing, linked disease burden and risk factor exposure)
B	is the amount of burden (DALY) attributable to the risk factor in 2003 (B_{03}) in 2018 (B_{18})
P	is the population size in 2003 (P_{03}) or in 2018 (P_{18})
S	is the population age structure in 2003 (S_{03}) or in 2018 (S_{18})
R	is the rate burden of diseases linked to risk factor in 2003 (R_{03}) or in 2018 (R_{18})
F	is the population attributable fraction of diseases linked to exposure in 2003 (F_{03}) or in 2018 (F_{18})

Indigenous estimates

For the Indigenous population, the same risk factor list was used as for the total Australian population, with 3 exceptions: unimproved sanitation, which was included for Indigenous estimates only; and high sun exposure and bullying victimisation, which were included for national estimates only. The burden from unimproved sanitation was not estimated for the non-Indigenous population due to lack of available exposure data, and was assumed to be close to 0. The burden from sun exposure was not estimated for the Indigenous population as it was not possible to account for the impact of differences in skin melanin levels. The burden from bullying victimisation was not estimated for the Indigenous population as data were not available to estimate exposure in a comparable way. The additional impacts of racism for Aboriginal and Torres Strait Islander people would also need to be taken into account. The AIHW is exploring this issue further with the view to incorporating a measure of bullying and/or racism in future Indigneous burden of disease studies.

The same risk–outcome pairs, relative risks and TMREDs as used for national estimates were used for Indigenous risk factor estimates. Relative risks specific to the Indigenous population were not available.

Exposure distributions for some risk factors could not be measured for the 2003 Indigenous estimates due to lack of available input data comparable with the methods used for the 2011 and 2018 estimates. These were air pollution, high blood plasma glucose, unimproved sanitation, and low birthweight & short gestation. As a result, these risk factors were not included in the 2003 Indigenous estimates.

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Estimating burden of disease measures

Health adjusted life-expectancy (HALE)

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.

On this page:

Estimating the health-adjusted life expectancy

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 2021) encompasses a broader scope of functional limitations of disability and selected long-term conditions.

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 2018, 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.

For example, if the YLD rate for men aged 40–44 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 per year. Thus, on average, each male in this age group spent 9.67% of the year 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.

Data sources

In the ABDS 2018, HALE was calculated at the national level for 2003, 2011, 2015 and 2018 and for sub-national populations (state and territory, remoteness areas and socioeconomic groups) for 2011, 2015 and 2018. Life table data were sourced from published and customised life tables (see Table 3.14 below).

Table 3.14: Life table data sources

Population	Data year	Data source
National and state/territory	2003	*Life tables, states, territories and Australia, 2002–2004* (ABS 2005)
	2011	*Life tables, states, territories and Australia 2010–2012* (ABS 2013)
	2015	*Life tables, states, territories and Australia 2014–2016* (ABS 2017b)
	2018	*Life tables, 2016–2018* (ABS 2019)
Remoteness	2011	*Life tables by remoteness areas, 2010–2012* (ABS 2017d) – customised report
	2015	*Life table by remoteness areas, 2014–2016* (ABS 2018b) – customised report
	2018	*Life table by remoteness areas, 2016–2018* (ABS 2020b) – customised report
Socioeconomic group	2011	*Life tables by Index of Relative Socioeconomic Disadvantage (IRSD), 2010–2012* (ABS 2017c) – customised report
	2015	*Life tables by Index of Relative Socioeconomic Disadvantage (IRSD), 2014–2016* (ABS 2018a) – customised report
	2018	*Life tables by Index of Relative Socioeconomic Disadvantage (IRSD), 2016–2018* (ABS 2020a) – customised report

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 and over, 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 and over.

YLD rates were available at the national level for 5-year age groups to 100 and over (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 and over.

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, 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 and over.

Sub-national estimates

HALE estimates for states were calculated using state-specific life expectancy data (ABS 2007, 2013, 2017b) 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 for 2011 and ASGS 2016 for 2015 and 2018. HALE estimates for remoteness areas are described in the ABDS 2018 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 for 2011 and 2016 SEIFA IRSD for 2015 and 2018. 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 2018 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.

Indigenous estimates

HALE for Indigenous and non-Indigenous Australians was estimated at the national level, for 4 states and territories (New South Wales, Queensland, Western Australia and the Northern Territory) and by remoteness area. Estimates for Indigenous Australians were not derived by socioeconomic group as life expectancy data were not available using the same socioeconomic index used for deriving YLD. HALE for Indigenous and non-Indigenous Australians was estimated for 2018 only, as comparable life expectancy estimates for other reference years were not available.

Estimates were calculated using Indigenous- and non-Indigenous-specific life expectancy data (ABS 2018c, 2021 unpublished) and YLD rates for Indigenous and non-Indigenous Australians. HALE estimates for Indigenous and non-Indigenous Australians are described in the ABDS 2018 to align with the first age of the age group; for example, HALE at age 65 represents HALE for the age group 65–69.

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). In the ABDS 2018, HALE is calculated using **Sullivan's method**:

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

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.

A HALE calculation using Sullivan's method (with an example from the ABDS 2015) is shown below. 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.

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, 2015 and 2018).

Table 3.15: Example calculation of HALE, Australia, males, 2014–2016

Male life table data (2014–2016)[a]											Key to variables
											x age in years (or age group when using an abridged life table)
	Population (hypothetical)	Proportion of population dying	Total person-years lived	Life expectancy	YLD rate[b]	YLD rate[b]	Average health	Total healthy person-years lived	Cumulative total healthy person-years lived	Life expectancy (healthy years)	l_x the number of people surviving to exact age x, according to mortality rates
	l_x	q_x	L_x	e_x		p_x	H_x	L'_x	T'_x	e'_x	q_x the proportion of people who die between age x and x+1
											L_x the total person-years lived in the age interval x to x+1
Age x (years)	(number)	(rate)	(number)	(years)	(years/ 1,000 people)	(years/ person)	(years/ person)	(number)	(number)	(years)	e_x the life expectancy (that is, the average of cumulative person-years lived by each person in the cohort
0	100,000	0.00357	99,682	80.4	26.62	0.0266	0.9734	97,028	7,144,404	71.4	YLD rate the average time spent in less than full health by every 1,000 people in the population
1	99,643	0.00029	99,627	79.7	22.56	0.0226	0.9774	97,379	7,047,376	70.7	p_x average ill health; the proportion of the year spent in less than full health (average YLD per person)
2	99,614	0.00016	99,606	78.8	22.56	0.0226	0.9774	97,359	6,949,996	69.8	H_x average health; the proportion of the year spent in full health (1 minus the average ill health)
3	99,599	0.00014	99,592	77.8	22.56	0.0226	0.9774	97,345	6,852,638	68.8	L'_x the total healthy years lived in the age interval x to x+1 (equals H_x x L_x)
4	99,585	0.00012	99,579	76.8	22.56	0.0226	0.9774	97,332	6,755,292	67.8	T'_x the cumulative years lived in full health from age x; equivalent to the cumulative sum of L'_x from age x to the top age in the life table
5	99,573	0.00011	99,567	75.8	32.74	0.0327	0.9673	96,308	6,657,960	66.9	e'_x 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 T'x / lx
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.

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Disease and risk factor specific models and methods

Disease specific methods - mortality

This chapter provides information on the methods used to estimate mortality (fatal burden or YLL) for each of the 17 disease groups (below, in alphabetical order). It includes information on the redistribution methods applied where applicable.

Blood and metabolic disorders

Deaths related to blood & metabolic disorders were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). 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) (Table 3.1).

Cancer and other neoplasms

Cancer-related deaths were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list).

Deaths coded to intestinal tract, part unspecified (C26.0) were assigned to bowel cancer, instead of being redistributed as part of the ABDS algorithm for ill-defined digestive cancers. This aligns with the AIHW cancer mortality reporting and the ABS reporting practice.

Updated algorithms were used for cancer of other and ill-defined digestive organs (C26–excluding C26.0) and cancers of ill-defined, secondary unknown primary sites (C76–C80). Their redistributed were based on direct evidence from the Western Australian cancer registry (see redistribution tables below).

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

Similarly, cancers of multiple independent primary sites (C97) could not be redistributed using direct evidence, as a specific cancer cannot be assigned to these by cancer registries. Consequently, deaths coded to C97 were also assigned directly to ‘cancer of unknown primary site’.

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

Table 4.1: Redistribution proportions of other and ill-defined digestive organs (C26-excluding C26.0), by age (years) and sex

Disease	0-9	10-14	15-24	25-44	45-64	65-84	85-94	95+
Males								
Oesophageal cancer	0.000	0.000	0.000	0.000	0.000	0.000	0.053	0.000
Stomach cancer	0.571	0.571	0.571	0.571	0.198	0.094	0.210	0.000
Bowel cancer	0.429	0.429	0.429	0.429	0.307	0.341	0.317	0.000
Pancreatic cancer	0.000	0.000	0.000	0.000	0.000	0.047	0.000	0.000
Lung cancer	0.000	0.000	0.000	0.000	0.050	0.024	0.105	0.000
Melanoma of the skin	0.000	0.000	0.000	0.000	0.000	0.024	0.000	0.000
Non-melanoma skin cancer (NMSC)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Breast cancer	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Ovarian cancer	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Other lymphohaematopoietic (blood) cancers	0.000	0.000	0.000	0.000	0.000	0.000	0.053	0.000
Cancer of unknown primary site	0.000	0.000	0.000	0.000	0.396	0.329	0.158	1.000

Other malignant neoplasms (cancers)	0.000	0.000	0.000	0.000	0.050	0.141	0.105	0.000
Females								
Oesophageal cancer	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Stomach cancer	0.000	0.000	0.000	0.000	0.139	0.128	0.195	0.000
Bowel cancer	1.000	1.000	1.000	1.000	0.444	0.332	0.268	0.414
Pancreatic cancer	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Lung cancer	0.000	0.000	0.000	0.000	0.000	0.077	0.000	0.000
Melanoma of the skin	0.000	0.000	0.000	0.000	0.000	0.026	0.000	0.000
Non-melanoma skin cancer (NMSC)	0.000	0.000	0.000	0.000	0.000	0.000	0.049	0.000
Breast cancer	0.000	0.000	0.000	0.000	0.000	0.026	0.000	0.586
Ovarian cancer	0.000	0.000	0.000	0.000	0.000	0.051	0.000	0.000
Other lymphohaematopoietic (blood) cancers	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Cancer of unknown primary site	0.000	0.000	0.000	0.000	0.278	0.257	0.439	0.000
Other malignant neoplasms (cancers)	0.000	0.000	0.000	0.000	0.139	0.103	0.049	0.000

Source: Western Australia Cancer Registry data, 2013–2017; AIHW National Mortality Database.

Table 4.2: Redistribution proportions of ill-defined cancers (C39, C76-C80, C97), by age (years) and sex

Disease	0-9	10-14	15-24	25-44	45-64	65-84	85-94	95+
Males								
Laryngeal cancer	0.000	0.000	0.000	0.000	0.010	0.000	0.008	0.000
Oesophageal cancer	0.000	0.000	0.000	0.000	0.000	0.003	0.000	0.000
Stomach cancer	0.000	0.000	0.000	0.000	0.030	0.014	0.008	0.000
Bowel cancer	0.000	0.000	0.000	0.167	0.059	0.045	0.033	0.000
Liver cancer	0.000	0.000	0.000	0.000	0.020	0.021	0.008	0.000
Gallbladder cancer	0.000	0.000	0.000	0.000	0.010	0.000	0.000	0.000
Pancreatic cancer	0.000	0.000	0.000	0.000	0.050	0.007	0.008	0.000
Lung cancer	0.000	0.000	0.000	0.083	0.089	0.110	0.066	0.000
Mesothelioma	0.000	0.000	0.000	0.000	0.010	0.003	0.000	0.000
Melanoma of the skin	0.000	0.000	0.000	0.000	0.000	0.003	0.008	0.000
Non-melanoma skin cancer (NMSC)	0.000	0.000	0.000	0.000	0.089	0.076	0.148	0.300
Prostate cancer	0.000	0.000	0.000	0.000	0.000	0.003	0.025	0.000
Bladder cancer	0.000	0.000	0.000	0.000	0.010	0.014	0.008	0.100
Kidney cancer	0.000	0.000	0.000	0.000	0.000	0.007	0.008	0.000
Brain and central nervous system cancer	0.000	0.000	0.000	0.000	0.010	0.003	0.008	0.000
Non-Hodgkin lymphoma	0.000	0.000	0.250	0.083	0.000	0.000	0.000	0.000

Myeloma	0.000	0.000	0.000	0.000	0.000	0.000	0.008	0.000
Other lymphohaematopoietic (blood) cancers	0.000	0.000	0.000	0.000	0.000	0.003	0.000	0.000
Cancer of unknown primary site	1.000	1.000	0.250	0.500	0.475	0.579	0.607	0.600
Lip and oral cavity cancer	0.000	0.000	0.000	0.000	0.059	0.024	0.016	0.000
Nasopharyngeal cancer	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Other lip, oral cavity and pharynx cancers	0.000	0.000	0.000	0.000	0.010	0.014	0.008	0.000
Acute myeloid leukaemia (AML)	0.000	0.000	0.000	0.000	0.000	0.003	0.000	0.000
Chronic myeloid leukaemia (CML)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Chronic lymphocytic leukaemia (CLL)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Other leukaemias	0.000	0.000	0.000	0.000	0.030	0.003	0.000	0.000
Other malignant neoplasms (cancers)	0.000	0.000	0.500	0.167	0.069	0.062	0.025	0.000
Females								
Laryngeal cancer	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Oesophageal cancer	0.000	0.000	0.000	0.000	0.026	0.009	0.006	0.000
Stomach cancer	0.000	0.000	0.000	0.000	0.013	0.009	0.006	0.000
Bowel cancer	0.125	0.125	0.125	0.125	0.077	0.032	0.040	0.000
Liver cancer	0.000	0.000	0.000	0.000	0.013	0.005	0.000	0.000
Gallbladder cancer	0.000	0.000	0.000	0.000	0.000	0.009	0.006	0.000
Pancreatic cancer	0.000	0.000	0.000	0.000	0.013	0.018	0.006	0.000
Lung cancer	0.000	0.000	0.000	0.000	0.141	0.100	0.034	0.050
Mesothelioma	0.000	0.000	0.000	0.000	0.000	0.009	0.000	0.000
Melanoma of the skin	0.000	0.000	0.000	0.000	0.000	0.005	0.017	0.000
Non-melanoma skin cancer (NMSC)	0.125	0.125	0.125	0.125	0.051	0.027	0.051	0.100
Breast cancer	0.000	0.000	0.000	0.000	0.064	0.041	0.006	0.050
Cervical cancer	0.000	0.000	0.000	0.000	0.000	0.005	0.000	0.000
Uterine cancer	0.000	0.000	0.000	0.000	0.000	0.018	0.006	0.000
Ovarian cancer	0.125	0.125	0.125	0.125	0.026	0.018	0.017	0.050
Bladder cancer	0.000	0.000	0.000	0.000	0.013	0.018	0.006	0.000
Kidney cancer	0.000	0.000	0.000	0.000	0.000	0.005	0.006	0.000
Brain and central nervous system cancer	0.000	0.000	0.000	0.000	0.026	0.005	0.000	0.000
Non-Hodgkin lymphoma	0.000	0.000	0.000	0.000	0.000	0.000	0.006	0.000
Myeloma	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Other lymphohaematopoietic (blood) cancers	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Cancer of unknown primary site	0.625	0.625	0.625	0.625	0.462	0.609	0.751	0.650

Lip and oral cavity cancer	0.000	0.000	0.000	0.000	0.013	0.018	0.011	0.000
Nasopharyngeal cancer	0.000	0.000	0.000	0.000	0.000	0.005	0.000	0.000
Other lip, oral cavity and pharynx cancers	0.000	0.000	0.000	0.000	0.000	0.005	0.000	0.000
Acute myeloid leukaemia (AML)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Chronic lymphocytic leukaemia (CLL)	0.000	0.000	0.000	0.000	0.000	0.000	0.011	0.000
Other leukaemias	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Other malignant neoplasms (cancers)	0.000	0.000	0.000	0.000	0.064	0.032	0.017	0.100

Source: Western Australia Cancer Registry data, 2013–2017; AIHW National Mortality Database.

Cardiovascular diseases

Cardiovascular disease-related deaths were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). Deaths coded to hypertension (I10, I13, I15) and heart failure (I50) were redistributed using the indirect MCOD 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.

Endocrine disorders

Endocrine disorder deaths were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). 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.

Gastrointestinal disorders

Deaths related to gastrointestinal disorders were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). Deaths coded to unspecified digestive diseases (K92) were redistributed using the direct evidence and indirect MCOD method to chronic liver disease, gastroduodenal disorders and diverticulitis. Deaths coded to peritonitis (K65–K66) were also redistributed using direct evidence and indirect MCOD 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).

Hearing and vision disorders

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.

Infant and congenital conditions

Deaths related to infant & congenital conditions were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). Deaths due to congenital malformations with ICD-10 codes Q10–Q18, Q38.1, Q54, Q65–Q74, Q82–Q84, Q89.9, Q99.9 were considered implausible causes of death, and were redistributed proportionally to all non-communicable diseases (that is, excluding infections, cancer and injuries) excluding reproductive & maternal conditions, oral disorders and hearing & vision disorders.

Infectious diseases

Deaths from infectious diseases were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). 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 Table 3.1).

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 MCOD method.

Changes to selection rules for coding causes of death in recent years have allowed more chronic conditions, such as cancers, coded to Part 2 of the death certificate (associated causes), to be selected as the underlying cause of death when septicaemia appears in Part 1 (underlying cause) of the death certificate. Following discussion with mortality data experts, deaths recorded with septicaemia as the underlying cause were not redistributed to selected chronic conditions, but instead to more acute conditions, such as urinary tract infections.

References

Naghavi M, Makela S, Foreman K, O'Brien J, Pourmalek F & Lozano R 2010. Algorithms for enhancing public health utility of national causes-of-death data. Population Health Metrics 20108:9.

Injuries

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 (see Mapping of ICD-10 codes to the disease list).

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.
- Redistribution of ICD-10 code X59 *Exposure to unspecified factor.* Previously these were redistributed proportionately across injuries. Using similar methods to AIHW injury reports, we used associated causes of death (fracture codes) to identify additional deaths from falls (2,766 deaths), thereby resulting in less X59 deaths being needed to be redistributed (727 deaths).
    - Additional fall deaths were identified as those having X59 as the UCOD with a fracture code in the ACOD:
    - the UCOD was an Unintentional fall (W00–W19); or
    - the UCOD was coded as Exposure to unspecified factor (X59) and the MCODs included a code for Fracture (the codes for fractures are S02, S12, S22, S32, S42, S52, S62, S72, S82, S92, T02, T08, T10, T12 and T14.2).

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 Table 3.1.

Table 4.3: 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.

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 4.3). 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.

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.

Table 4.4: List of injury categories used in the ABDS 2018 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
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

Kidney and urinary conditions

Deaths related to kidney & urinary diseases were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). Please note that the ICD-10 codes used to align deaths to chronic kidney disease in the ABDS 2018 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 2019).

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 2014). N19 deaths were then redistributed to N17 (acute renal failure) and N18 (chronic renal failure) according to the proportions obtained from the linked data.

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

References

AIHW 2014. Assessment of the coding of ESKD in deaths and hospitalisation data: a working paper. Cat. no. PHE 182. Canberra: AIHW.

AIHW 2019. Indicators of socioeconomic inequalities in cardiovascular disease, diabetes and chronic kidney disease. Cat. no. CDK 12. Canberra: AIHW.

Mental and substance use disorders

Deaths related to mental & substance use were assigned from the NMD, as defined by the disease list (Mapping of ICD-10 codes to the disease list). 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.

Musculoskeletal conditions

Deaths related to musculoskeletal conditions were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). No musculoskeletal condition deaths were redistributed.

Neurological conditions

Neurological conditions-related deaths were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). Deaths coded to ICD-10 codes G81–G83 were proportionally distributed across all diseases using proportions derived from Australian all-cause mortality data.

Oral disorders

Oral disorder deaths were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). No deaths due to oral disorders were redistributed. Oral disorders were also not a target cause for redistribution.

Reproductive and maternal conditions

Deaths related to reproductive & maternal conditions were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). 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.

Respiratory diseases

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 Mapping of ICD-10 codes to the disease list. Deaths due to respiratory failure (J96) were redistributed across all diseases (excluding reproductive & 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 & maternal conditions, oral disorders and hearing & vision disorders) using proportional allocation. Pneumonitis deaths (J69) were redistributed using the indirect MCOD method with all diseases (excluding reproductive & maternal conditions, oral disorders and hearing & vision disorders) in the target range.

Skin disorders

Deaths related to skin disorders were assigned from the NMD as defined by the disease list (Mapping of ICD-10 codes to the disease list). 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 & maternal conditions, oral disorders and hearing & vision disorders) (see Table 3.1).

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Disease and risk factor specific models and methods

Disease specific methods - morbidity

This chapter provides information on the methods used to estimate morbidity (non-fatal burden or YLD) for each of the 17 disease groups (below, in alphabetical order). It includes information on the sequela and health states used, prevalence and severity data sources used, and details on the methods used to calculate prevalence estimates for each disease.

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
- heart failure—cardiovascular conditions
- infertility—reproductive & maternal conditions
- intellectual disability—mental & substance use disorders
- vision loss—hearing & vision disorders.

Detailed information is provided on the methods used for 2018 national estimates. Where these methods differ for sub-national estimates, 2015, 2011 or 2003 estimates, this is described separately.

Blood and metabolic disorders

Sequelae and health states

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

Table 4.5: Sequelae and health states for blood & metabolic disorders

Disease	Sequela	ABDS 2018 health state identifier[a]
Cystic fibrosis	Non-respiratory complications due to cystic fibrosis	207
	Respiratory complications due to cystic fibrosis	55, 56, 57
Haemophilia	Haemophilia	128, 207, 262
Haemolytic anaemia	Haemolytic anaemia	207
	Acute, severe event due to haemolytic anaemia	194, 2
	Anaemia due to haemolytic anaemia[b]	196, 197
Iron deficiency anaemia	Anaemia due to iron-deficiency anaemia[b]	195, 196, 197
Protein-energy deficiency	Stunting due to protein-energy deficiency	211
	Wasting due to protein-energy deficiency	210, 211
Other blood & metabolic disorders	Anaemia due to other blood & metabolic disorders[b]	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 ABDS 2018 health states.

(b) Part of anaemia envelope.

Prevalence estimation

Anaemia envelope

As an envelope in the ABDS 2018, 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 2018. 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 2017–18 (ABS 2019). 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 2017–18. The AHS 2011–12 survey was used as biomedical data were not available from the NHS 2017–18. Additionally, the NHS did not report on *Very remote* areas, so prevalence was modelled to account for *Very remote* areas.

Similarly to the ABDS 2015, 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 have 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 the NHS 2017–18 results.

Cystic fibrosis

Prevalence of cystic fibrosis was derived from the Australian Cystic Fibrosis Data Registry (ACFDR) (Ruseckaite 2020, pers. comm. 18 June). Registrants by age, sex and severity (lung function) was obtained for all ages.

The ACFDR includes numerous markers for severity, but these conflict with other components of the ABDS 2018 (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 ACFDR, 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 2018 included haemophilia A and B. Prevalence estimates and severity distribution were derived from the Australian Bleeding Disorders Registry 2017–18 report (National Blood Authority 2018).

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 haemophila have mild and 5% have moderate haemophilia (Rowell 2015, pers. comm. 11 September).

Haemolytic anaemia

The same disabling sequelae for heamolytic anaemia as used in the ABDS 2015 were used in this study. The table below lists diagnosis and procedure codes (using the ICD-10-AM or Australian Classification of Health Interventions (ACHI) codes) for sequelae and severity distributions.

Table 4.6: 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, linked data from the NIHSI AA v0.5 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. It is assumed that the hospital presentation ratios for haemolytic anaemia are the same for all states/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 were 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 2018.

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.

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 2020).

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.

Data from the AATSIHS 2012–13 was used to estimate the prevalence of protein-energy deficiency in Indigenous children in 2011. As advised by experts, underweight status is indicative of mild malnutrition in the Indigenous population. Severity distributions were derived from the AATSIHS underweight class, with levels 2 and 3 identified as wasting (Cole et al. 2007). To align state distributions to remoteness area estimates, the severity distribution for the Northern Territory was used for *Outer regional*, *Remote* and *Very remote* prevalence estimates. National severity distributions were used for *Major cities* and *Inner regional* areas. Estimates for 2003, 2015 and 2018 were derived by applying the 2011 Indigenous rate to the Indigenous ERPs for 2003, 2015 and 2018 (from the 2016 Census).

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 (see *Definitions and durations for other blood and metabolic conditions* below).

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

Table 4.7: Definitions and durations for other blood and metabolic conditions

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

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 2018.

2015, 2011 and 2003 estimates

2015, 2011 and 2003 estimates were based on the same method as for 2018.

Hospital separations were derived from the 2015, 2011 and 2003 calendar year.

Registrant data from 2015, 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 and 2015 were derived from self-reported estimates from the NHS 2004–2005 and NHS 2014–15 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 self-reported estimates to obtain age- and sex-specific prevalence rates and applied to population estimates to attain prevalence for 2003 and 2015.

Estimates for protein-energy deficiency in elderly Australians for 2015, 2011 and 2003 used the same method as in 2018 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.

Indigenous specific estimates

Where possible, the same general methods and data sources were used to derive Indigenous estimates for blood & metabolic disorders for 2018, 2011 and 2003.

Indigenous estimates based on hospital separations data were adjusted for under-identification using standard adjustment factors (see Years lived with disability (YLD)).

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

NHMD separations, biomedical data from the AATSIHS 2012–13 (ABS 2020a) and self-report data from NATSIHS 2018–19 (ABS 2020b) were used to estimate iron-deficiency anaemia in 2018, using the same method as used for the national population. The 2011 estimates were calculated using NHMD anaemia separations applied to sex-specific AATSIHS totals to create the anaemia envelope. The 2003 estimates were created by applying the 2011 envelope rates to the 2003 Indigenous population. For all years, other sequela were removed from the envelope to leave IDA. The national severity splits were applied.

The same data source and method used to estimate protein-energy deficiency in the national population was used for the 2018, 2011 and 2003 Indigenous population.

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Cancer and other neoplasms

Methods for cancer and other neoplasms mostly remained the same between the ABDS 2015 and the ABDS 2018. The most notable changes are in the use of linked data from the NIHSI AA v0.5 instead of linked data from the Western Australian Department of Health.

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 (see *General cancer-related sequelae and health states* below). For select cancers, it also includes long-term sequelae —usually as a result of curative treatment (see *Long-term cancer sequelae and health states* below).

Table 4.8: General cancer-related sequelae and health states

Sequelae	Health state	ABDS 2018 health state identifier[(a)]
Diagnosis and primary therapy phase of <*cancer type*>	Cancer: diagnosis and primary therapy	18, 208[(b)]
Controlled phase of <*cancer type*> (c)	Generic uncomplicated disease: worry and daily medication	207
Metastatic phase of <*cancer type*> (d)	Cancer: metastatic	19
Terminal phase of <*cancer type*> (e)	Terminal phase: with medication	22

(a) See ABDS 2018 health states.

(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 4.9: Long-term cancer sequelae and health states

Disease	Sequelae	ABDS 2018 health state identifier[(a)]
Laryngeal cancer	Laryngectomy due to laryngeal cancer	212
Bowel cancer	Stoma due to bowel cancer	21
Breast cancer	Mastectomy due to breast cancer	20
Prostate cancer	Impotence due to prostate cancer	49
	Urinary incontinence due to prostate cancer	48
Bladder cancer	Stoma due to bladder cancer	21
	Urinary incontinence due to bladder cancer	48
Brain and central nervous system cancer	Brain injury (mild, moderate, severe) due to brain and central nervous system cancer	181, 182, 183

Benign brain tumours	Brain injury (mild, moderate, severe) due to benign brain tumours	181, 182, 183
Ductal carcinoma in situ	Mastectomy due to ductal carcinoma in situ	20

(a) See ABDS 2018 health states.

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 and ABDS 2018 (see Supplementary table S4.1). 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 2016 ACD. This assumes that people will undergo primary treatment at the time of diagnosis.

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

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

Complex NMSC diagnoses and treatments were sourced from separations in the NHMD with a principal diagnosis of NMSC in 2018 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 2018 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 2018, 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.

For consistency, deaths from cancer were sourced from the fatal component of the study.

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 2018 (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 2018. 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 2018 ACD. 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 2008–2018 were extracted from the NHMD. To derive prevalence from separations, a 7-year prevalence-to-separations ratio was derived from NIHSI and applied to the number of national separations.

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 2018 (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 2018, 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 2018 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 2016 ASGS remoteness areas (for 2018 and 2015) and 2011 ASGS remoteness areas (for 2011 and 2003) to the Statistical Area Level 2 or Statistical Local Area 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 2016 SEIFA population-based IRSD quintiles (for 2018 and 2015) and 2011 SEIFA population-based IRSD quintiles (for 2011 and 2003) to the Statistical Area Level 2 or Statistical Local Area 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.

2015, 2011 and 2003 estimates

All estimates for the years 2003, 2011 and 2015 were 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 2018.

As Medicare Benefits Schedule item codes might have changed over time, the positive predictive value (PPV) 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. For 2011 and 2015 estimates are produced by using MBS data with a PPV of 68% and a duration of 2 weeks applied to the MBS services for selected items. As the services are provided in 10–15 year age groups, estimates for 5-year age groups are created by applying the overall 10-year prevalence rate to its constituent 5-year age groups.

Hospital separations data were used for health loss due to complex treatment as for 2018.

Long-term sequelae were derived in the same way using year-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, 2015 and 2018.

Indigenous specific estimates

The same general methods were used to derive 2018, 2011 and 2003 Indigenous estimates with the following exceptions:

The 2011 cancer incidence and prevalence for the Indigenous population were derived from the average cancer incidence recorded in the 2011 ACD for 2009-2011 for NT, WA, Victoria and Queensland, and from 2007–2009 for NSW—these are the states with cancer incidence data considered of sufficient quality for reporting. Rates from these states combined were applied to the ACT, Tasmania and SA populations to determine national Indigenous incidence. The 2011 mortality was derived from the average number of deaths in 2009–2011, adjusted for under-identification using ABS mortality adjustment factors as per fatal estimates (see Years of life lost (YLL)).

2003 cancer incidence and prevalence for the Indigenous population were derived from the average cancer incidence recorded in the 2011 version of the ACD for 2002–2004 for NT, WA, NSW and Queensland. Rates from these states combined were applied to the ACT, Victoria, Tasmania and SA populations to determine national Indigenous incidence. 2003 mortality was derived from the average number of deaths in 2002–2004, adjusted for under-identification using ABS mortality adjustment factors as per fatal estimates (see Years of life lost (YLL)).

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

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

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

The number of cases of ductal carcinoma in situ diagnosed in Indigenous women in was estimated by applying the ratio of small (i.e. < 2 cm) breast tumours in national:Indigenous women to the national incidence of ductal carcinoma in situ for both 2011 and 2003

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Cardiovascular diseases

Methods for cardiovascular diseases mostly remained the same between the ABDS 2015 and the ABDS 2018. The most notable changes are in the use of linked data from the NIHSI AA v0.5 instead of linked data from the Western Australian Department of Health.

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 the next section.

Prevalence estimation

Acute sequelae

The NHMD was the main data source used to estimate prevalence of acute sequelae (see table below). As these events are of short duration, point prevalence was estimated by applying the duration of health loss to incidence.

Table 4.10: ABDS 2018 diseases and sequelae that use the NHMD to estimate point prevalence

Disease	Sequela	ABDS 2018 health state identifier[(a)]	Duration
Coronary heart disease	Acute coronary syndrome	24, 25	2 days (more severe)
			26 days (less severe)
Stroke	Acute stroke	34, 35, 36, 37, 38	28 days
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 ABDS 2018 health states.

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 linked hospitalisations and deaths data from the NIHSI AA v0.5 to determine the number of non-fatal acute coronary syndrome events in the reference year, using methods previously published by the AIHW (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. State-specific incidence rates were available for New South Wales, Victoria, South Australia and Tasmania, i.e. the States that are available in the NIHSI AA v0.5. Incidence rates combining the 4 jurisdictions were used to estimate the incidence in the remaining States and Territories.

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

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 NIHSI AA v0.5 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.

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, linked hospitalisations and deaths data from the NIHSI AA v0.5 and the NZBDS.

The sequelae for which a combination of NHMD and linked hospitals and deaths data from the NIHSI AA v0.5 were used are listed in the table below. Heart failure is discussed separately from the other chronic sequelae as it is an envelope condition.

Table 4.11: ABDS 2018 diseases and sequelae that use a combination of the NHMD and the NIHSI AA v0.5 to estimate prevalence

Disease	Sequela	ABDS 2018 health state identifier[(a)]
Coronary heart disease	Chronic coronary heart disease	26, 27, 28, 262
	Heart failure due to coronary heart disease[(b)]	31, 32, 33
Stroke	Chronic stroke	34, 35, 36, 37, 38, 262
Rheumatic heart disease	Valvular diseases due to rheumatic heart disease	207
	Heart failure due to rheumatic heart disease[(b)]	31, 32, 33
Non-rheumatic heart disease	Valvular diseases due to non-rheumatic heart disease	207
	Heart failure due to non-rheumatic heart disease[(b)]	31, 32, 33
Atrial fibrillation and flutter	Moderate/severe atrial fibrillation and flutter	29
Hypertensive heart disease	Heart failure due to hypertensive heart disease[(b)]	31, 32, 33
Inflammatory heart disease	Heart failure due to inflammatory heart disease[(b)]	31, 32, 33
Cardiomyopathy	Heart failure due to cardiomyopathy[(b)]	31, 32, 33
Cardiovascular defects[(c)]	Heart failure due to congenital cardiovascular defects[(b)]	31, 32, 33
Peripheral vascular disease	Intermittent claudication due to peripheral vascular disease	30

(a) See ABDS 2018 health states.

(b) Part of the 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, non-rheumatic valvular disease and peripheral vascular disease), it was assumed that people who have these diseases are hospitalised at least once within the 7 years leading up to the reference year. A 7-year look-back period was used as it was the longest time period available for hospitals data in the NIHSI AA v0.5.

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 linked hospitalisations and deaths data from the NIHSI AA v0.5.

These ratios were then applied to the count of hospital separations from the NHMD, by age and sex. Admitted hospitals data are available from the NIHSI AA v0.5 for New South Wales, Victoria, South Australia and Tasmania. Where numbers allow, that is, the numbers are not low enough to cause volatility in estimates, state-specific ratios are calculated. Otherwise, data from all 4 jurisdictions are used for the ratios. For States/Territories that do not have hospitals data in the NIHSI AA v0.5, ratios calculated using all 4 jurisdictions combined are used. This assumes that persons-to-separations ratios are similar across all jurisdictions.

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 the overall prevalence of atrial fibrillation and flutter. 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. The number of people were estimated by applying persons-to-separations ratios to the count of separations.

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.

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 linked hospitalisations and deaths data from the NIHSI AA v0.5 to the national count of separations from the NHMD.

As one of the envelopes in the ABDS 2018, 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 linked data from the NIHSI AA v0.5. 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 2016 (for 2018 and 2015 estimates) and 2011 SEIFA population-based IRSD quintiles to the Statistical Area Level 2 recorded in hospital separations data.

For atrial fibrillation, prevalence by State or Territory, remoteness area, and socioeconomic group were obtained by applying proportions by sub-national disaggregation from separations in the NHMD.

2015, 2011 and 2003 estimates

For chronic sequelae where prevalence was estimated from a combination of the NHMD and ratios and rates derived from the NIHSI AA v0.5, methods for 2003, 2011 and 2015 were largely similar to those for 2018. 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 2006 prevalence rates. It was assumed that rates were relatively stable between 2003 and 2006.

For acute coronary syndrome, acute stroke and acute inflammatory heart disease, the methods used for 2003, 2011 and 2015 prevalence estimates were the same as those used for 2018 estimates. For acute rheumatic fever, the hospital ratio used to adjust for readmission for 2011 estimates was used for 2003 prevalence estimates. For atrial fibrillation and flutter, the NZBDS prevalence rates used for 2018 estimates were also used for 2015, 2011 and 2003 estimates.

Indigenous specific estimates

The general approach and method used for national estimates were used for the Indigenous estimates. The severity distribution used for national estimates was also used for Indigenous estimates. For diseases and sequelae (Table 4.10) where the NHMD was used to estimate point prevalence, hospital separations data were adjusted for under-identification using standard adjustment factors (see Years lived with disability (YLD)).

For diseases and sequelae (Table 4.11) where ratios from the NIHSI AA were used to estimate prevalence, Indigenous-specific ratios from the Western Australian Linked Data Set were obtained from the WA Department of Health.

For atrial fibrillation and flutter, where non-Maori prevalence rates from the NZBDS were used for the national prevalence estimates, the Maori prevalence rates were applied to Indigenous populations for the relevant reference years to derive Indigenous prevalence estimates.

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Endocrine disorders

Sequelae and health states

Sequelae and health states assigned to endocrine disorders are shown in the table below

Table 4.12: Sequelae and health states for endocrine disorders

Disease	Sequela	ABDS 2018 health state identifier[(a)]
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 ABDS 2018 health states.

Prevalence estimation

Type 1 diabetes

Diagnosed type 1 diabetes

The prevalence of type 1 diabetes mellitus was sourced from the National (insulin-treated) Diabetes Register (NDR). Data for the NDR are sourced from the National Diabetes Services Scheme (NDSS) Registrant data, the NDSS Sales data, the Australasian Paediatric Endocrine Group (APEG) state-based registers and the National Death Index (NDI). 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 2018 Quality statement.

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 linked hospitalisations and deaths data from the NIHSI AA v0.5. 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 et al. (2009).

Type 2 diabetes

Diagnosed type 2 diabetes

The prevalence of type 2 diabetes mellitus was sourced from self-report data from the NHS 2017–18, 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; WA Davis 2020, pers. comm., 16 April). 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.

2015, 2011 and 2003 estimates

Type 1 and other diabetes prevalence estimates for 2015, 2011 and 2003 were derived from the same data sources as the estimates for 2018.

For diagnosed type 2 diabetes, prevalence estimates were derived from the NHS 2014–15, the AHS 2011–12 and the NHS 2004–05 for 2015, 2011 and 2003, respectively. For 2015 and 2011, 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 2018 estimates.

For 2003, 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 2018 estimates.

Indigenous specific estimates

Indigenous prevalence for type 1 diabetes was derived from the linked National Diabetes Services Scheme (NDSS) and Australasian Paediatric Endocrine Group (APEG) data set. Indigenous-specific prevalence rates were used in people aged under 30 years, and national rates in people aged 30 and over, due to data quality issues. The prevalence of type 2 diabetes was estimated by subtracting the estimated number of Indigenous Australians with type 1 diabetes from estimated total diabetes prevalence estimated using data from the biomedical component of the AATSIHS 2012–13 and self-reported data from the 2018–19 NATSIHS.

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

Amputations due to diabetes prevalence were estimated using the NHMD. Hospital separations data were adjusted for under-identification using standard adjustment factors (see Years lived with disability (YLD)).

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

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Gastrointestinal disorders

Sequelae and health states

The sequela and health states assigned to gastrointestinal disorders are shown in the table below. Durations and assumptions are outlined in subsections for individual diseases.

Table 4.13: Sequelae, health states and durations for gastrointestinal disorders

Disease	Sequela	ABDS 2018 health state identifier[(a)]	Duration
Gastroduodenal disorders	Anaemia due to gastroduodenal disorder[(b)]	195, 196, 197	8 weeks
	Symptomatic episodes of gastroduodenal disorder	193	1 week (inflammation) 3 weeks (ulcers)
Appendicitis	Symptomatic appendicitis requiring appendectomy	194	2 weeks
Abdominal wall hernia	Symptomatic hernia requiring repair	192	12 months
Vascular disorders of intestine	Stoma due to vascular disorder of intestine	21	12 months (permanent stoma) 5.4 months (temporary stoma)
	Vascular disorders of the intestine	194	6 weeks
Intestinal obstruction (without hernia)	Intestinal obstruction	194	2 weeks (major surgery) 2 days (minor surgery)
Inflammatory bowel disease	Crohn's disease or ulcerative colitis	46	12 months

Diverticulitis	Diverticulitis	194	2 weeks (medical therapy) 3 weeks (surgical intervention)
	Stoma due to diverticulitis	21	12 months (permanent stoma) 5.4 months (temporary stoma)
Chornic liver disease	Decompensated cirrhosis of the liver End-stage liver disease	44 22	10 months (with end-stage liver disease) 2 months (terminal stage)
	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 ABDS 2018 health states.

(b) Part of the 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 2018, 2015, 2011 and 2003 calendar years, as applicable. The durations used for each sequela are presented in Table 4.13.

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 4.13).

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 2017), 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 (Sequelae, health states and durations for gastrointestinal disorders.). 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.

The sex proportions of liver transplants due to chronic liver disease in that state used the same proportions as ABDS 2015, which were derived from Western Australian liver transplant data, 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 database linked hospitalisations and deaths data from the NIHSI AA v0.5 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 2017, and applied by the ABDS 2018, 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 2016). 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. (2017), which estimated the national prevalence of GORD as 6.9%. 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 et al. (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 ABDS 2015) and applied to the 2018 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 and ABDS 2018. 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 2018 was similar to that used in the ABDS 2011 and in the ABDS 2015 (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 2018 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 2018, 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 for 2015 and 2018 derived directly from the NHMD were broken down by state/territory, and by remoteness area and socioeconomic group by applying the 2016 ASGS remoteness areas and the 2016 SEIFA population-based IRSD quintiles to the Statistical Area Level 2 recorded in hospital separations data. For 2011 and 2003 estimates, 2011 ASGS remoteness areas and SEIFA population-based quintiles were used.

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.

2015, 2011 and 2003 estimates

The same methods used for 2018 estimates were used to estimate point prevalence for each of the diseases in the gastrointestinal disorders group for 2015, 2011 and 2003, using 2015, 2011 and 2003 hospitalisations data and populations.

Indigenous specific estimates

Indigenous estimates were derived using the same methods and data sources as described above for national estimates for 2018, 2011 and 2003. Estimates based on hospital separations data were adjusted for under-identification using standard adjustment factors (see Years lived with disability (YLD)).

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

For chronic liver disease, terminal and decompensated estimates were obtained by applying ratios derived from the NIHSI AA v0.5 and Western Australian linked data to national estimates.

Indigenous prevalence for inflammatory bowel disease, gastro-oesophageal reflux and functional gastrointestinal disorders was obtained by applying the national distribution directly to the Indigenous population for 2018, 2011 and 2003. This assumes the underlying rate is the same between the Indigenous and non-Indigenous populations, and between the two time points.

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Hearing and vision disorders

Disease list

For the ABDS 2015 and the ABDS 2018, 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 the table below. As only permanent hearing & vision disorders are estimated, health loss is assumed to apply for the whole year.

Table 4.14: Sequelae and health states for hearing & vision disorders

Disease	Sequela	ABDS 2018 health state identifier[(a)]
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 ABDS 2018 health states.

Prevalence estimation

Hearing loss

In the ABDS 2018, 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 2018 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 2018 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 2018).
- Prevalence for people aged 25–54 was derived from the NHS 2017–18 (ABS 2019) 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 and the ABDS 2015 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 2017–18 (ABS 2019) were applied to ages 55 and over.

Prevalence by severity

The same severity distribution as used in the ABDS 2011 and the ABDS 2015 (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 Table 4.15.

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

Table 4.15: Proportion of tinnitus in hearing impaired population, by age, sex and severity level

	Proportion of tinnitus within each severity level of hearing loss (%)			
Age group (years)	**Mild**	**Moderate**	**Severe**	**Profound**
Males				
18–24	38.9	29.2	54.1	0.0
25–44	21.8	35.2	35.3	37.2
45–64	29.9	29.7	39.4	42.6
65 and over	16.5	21.9	26.7	26.4
Females				
18–24	18.8	47.0	73.3	0.0
25–44	35.6	40.4	54.8	45.3
45–64	29.8	34.2	47.2	32.2
65 and over	21.7	28.9	33.6	30.1

Other hearing & vestibular disorders

Other hearing disorders were also calculated using self-reported data from the NHS 2017–18 (ABS 2019). 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 2017–18 (ABS 2019) (age estimates were not available due to high RSEs). To obtain age estimates, the age distribution was obtained using hospitalisations of Meniere disease in 2018 by age and sex from the NHMD, and then applied to the total prevalence derived from the NHS 2017–18 (ABS 2019). 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 2017–18 (ABS 2019) by age and sex. Age groups that had high RSEs (0–19 and 70 and over) were estimated using population sex-specific proportions to obtain 5-year age groups.

Refractive error and cataract & other lens disorders

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

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

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

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

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 (AIHW 2016), 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 (VanNewkirk et al. 2001). 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 and ABDS 2015, 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 and the ABDS 2015, 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 (ABS 2013) 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 et al. (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 2017–18 data.

2015, 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 2018 estimates were used for national 2003, 2011 and 2015 estimates.

Indigenous specific estimates

Hearing loss

The overall Indigenous prevalence of hearing loss was estimated using 2 main data sources:

- For ages 0–24, prevalence was derived from the Australian Hearing 2018 report summary tables of Indigenous Australians aged 26 and under with a clinically diagnosed hearing impairment who were fitted with a hearing aid (Australian Hearing 2018).
- Prevalence for Indigenous Australians aged 25 and over was derived from measured hearing loss data from the 2018–19 NATSIHS.

The overall severity distribution for Indigenous estimates was derived from data in the Northern Territory outreach audiology data collection report (AIHW 2020), with the national tinnitus splits then applied.

The Indigenous prevalence rates for 2018 were applied to the 2003 and 2011 Indigenous ERPs to derive prevalence estimates for 2003 and 2011.

Vision loss

Estimates for vision loss in the Indigenous population were estimated from published results from the National Eye Health Survey 2016 for 2018, and the Indigenous Eye Health Survey 2008 for both 2011 and 2003. These estimates provide robust data for vision loss specific to the Indigenous population. Published estimates were reported by state, remoteness category and broad age categories and apportioned into 5 year age groups and sex either using Indigenous population or national age-sex distributions for each cause.

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

Trachoma

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

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

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Infant and congenital conditions

Sequelae and health states

The sequelae and health states assigned to infant and congenital disorders are listed in the table below. 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 4.16: Sequelae and health states for infant & congenital conditions

Disease	Sequela	ABDS 2018 health state identifier[a]
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[b]	213, 214, 215, 216, 217, 218
Birth trauma & asphyxia	Neurodevelopment impairment due to birth trauma & asphyxia[b]	216, 217, 218
Cerebral palsy	Neurodevelopment impairment due to cerebral palsy	213, 214, 215
Neonatal infections	Acute complications due to neonatal infections	3
Other disorders of infancy	Other disorders of infancy	54
Neural tube defects	Incontinence due to neural tube defects	48
	Motor impairment due to neural tube defects	213, 214, 215
	Neurodevelopment impairment due to neural tube defects[b]	218
Brain malformations	Neurodevelopment impairment due to brain malformations[b]	216, 217, 218
Cardiovascular defects	Congenital cardiovascular defects untreated	33
	Heart failure due to congenital cardiovascular defects[c]	31, 32, 33
Cleft lip and/or palate	Disfigurement due to cleft lip/palate	201, 202
	Speech problems due to cleft lip/palate	212
Gastrointestinal malformations	Acute complications due to gastrointestinal malformations	194
	Incontinence due to anorectal atresia	48

Urogenital malformations	Urogenital malformations	192, 262
Down syndrome	Intellectual disability due to Down syndrome[(b)]	99, 100, 101, 102, 243
Other chromosomal abnormalities	Intellectual disability due to chromosomal abnormalities[(b)]	99, 100, 101, 102, 243
Other congenital conditions	Other congenital conditions	YLL:YLD ratio

(a) See ABDS 2018 health states.

(b) Part of the intellectual disability envelope.

(c) Part of the heart failure envelope.

Prevalence estimation

The key data sources to estimate prevalence of infant & congenital conditions are listed below.

Table 4.17: 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) for 2015, 2011 and 2003. Linear regression was used to derive the prevalent cases for 2018. The live birth prevalence rate for Western Australia was estimated by dividing the number of cases by Western Australia live births. This rate was then applied to the Australian live births 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, the same outputs from DisMod II were used for 2015 and 2018, adjusted for population change. Parameters used as inputs to DisMod II 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 2018

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.

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

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. Data on the number of live births by gestational age was obtained from the National Perinatal Data Collection (NPDC) for 2016–2018 combined. This was used to derive the proportion of live births in each preterm category—extremely, very and late pre-term.

For each severity, 50% of cases were modelled with motor impairment and 50% of cases with motor and cognitive impairment, based on assumptions by Blencowe et al. (2013).

Estimates for acute complications due to pre-term births & low birthweight were based on incidence of hospital separations in the 2018 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 2017, by gestational age (Chow et al. 2019). The durations were:

- extremely pre-term (20–27 weeks): 108 days
- very pre-term (28–31 weeks): 55 days
- late pre-term (32–36 weeks): 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 & asphyxia was derived from the NHMD 2018–19 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 2018 (Cerebral Palsy Alliance 2018). Incidence and mortality from cerebral palsy in 1913–2018 were estimated from the Australian Cerebral Palsy Register report and the NMD, respectively. Prevalence was adjusted for standard background mortality using the Australian life table 2016–2018 (ABS 2019).

Estimated Australian-specific severity distribution derived from the Gross Motor Function Classification System was applied to the estimates.

Table 4.18: Estimated severity distribution used for cerebral palsy, by Gross Motor Function Classification System (GMFCS) level

GMFCS levels	Description	GBD health state	Per cent
Level I	Walks without limitations	Motor impairment: mild	37.0
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	25.4
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	11.5
Level IV	Self-mobility with use of powered mobility assistance. Is supported when sitting; self-mobility is limited; and likely to be transported in wheelchair	Motor impairment: severe	12.2
Level V	Severe head and trunk control limitations. Requires extensive use of assisted technology and physical assistance; and to be transported in a wheelchair.	Motor impairment: severe	14.0

Source: Cerebral Palsy Alliance 2018.

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 et al. (2013), Badawi et al. (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 for 2015, 2011 and 2003. Linear regression was used to derive the prevalent cases for 2018. DisMod II was used to model prevalence for those aged over 1 using incidence, remission and case fatality inputs. The same outputs from DisMod II were used for 2015 and 2018, adjusted for population change. Prevalence estimates were then distributed into different health states using proportions from Hunt & Oakeshott (2003) (Table 4.19). The life expectancy for people with moderate or severe neural tube defects was assumed to be about 46 years (Oakeshott et al. 2015).

Table 4.19: 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.

Brain malformations

Prevalence of neurodevelopmental impairment due to brain malformations was derived from the intellectual disability envelope (see 'Mental and substance use disorders'). 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 Disease specific methods - morbidity).

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 2020). 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 2018 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. The same outputs from DisMod II were used for 2015 and 2018, adjusted for population change. 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 2018–19, 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 2018–19 data.

2015, 2011 and 2003 estimates

Estimates for infant & congenital conditions used a similar method, with data sourced for 2015, 2011 and 2003.

Indigenous specific estimates

Where possible, prevalence estimates for the Indigenous population for 2018, 2011 and 2003 were obtained from the same data sources as used for national prevalence estimates, using the same methods. Exceptions to this are described below.

Indigenous estimates based on hospital separations data (that is, neonatal infections, other disorders of infancy, acute preterm low birth weight complications) were adjusted for under-identification using standard adjustment factors (see Years lived with disability (YLD)).

For congenital abnormalities, Indigenous:total population rate ratios were derived from the WA Registry of Developmental Anomalies (for birth anomalies, e.g. for neural tube defects) or the NHMD (where surgical interventions, e.g. for cleft lip/palate) applied to national prevalence rates.

The Australian Cerebral Palsy Register (Cerebral Palsy Alliance 2018) reported 5.8% of people with cerebral palsy were born from mothers of Aboriginal and/or Torres Strait Islander status. This proportion was 3.5% in 2011 and 2003. These proportions were applied to national estimates to derive the Indigenous prevalence for cerebral palsy.

For conditions included in the intellectual disability envelope (see above), Indigenous prevalence estimates were calculated using Indigenous:non-Indigenous rate ratios (see Indigenous estimates section in ‘Mental and substance use disorders’). Due to lack of data, 2003 and 2018 estimates were calculated using 2011 rates, applied to reference populations.

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Infectious diseases

Sequelae and health states

A list of sequelae and health states assigned to each infectious disease is included in the table below. 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.

Table 4.20: Sequelae and health states for infectious diseases

Disease	Sequela	ABDS 2018 health state identifier[a]
HIV/AIDS	HIV/AIDS	10, 11, 12, 208
Tuberculosis	Tuberculosis	16
Syphilis	Congenital syphilis	3
	Primary syphilis	1
	Secondary syphilis	2
	Tertiary syphilis	217
Chlamydia	Chlamydial infection	1
	Infertility due to chlamydia[b]	50, 51
	Pelvic inflammatory disease due to chlamydia	193, 194
Gonorrhoea	Gonococcal infection	1
	Infertility due to gonorrhoea[b]	50, 51
	Pelvic inflammatory disease due to gonorrhoea	193, 194
Other sexually transmitted infections	Infertility due to other sexually transmitted infections[b]	50, 51
	Other sexually transmitted infections	1
	Pelvic inflammatory disease due to other sexually transmitted infections	193, 194
Hepatitis A	Acute hepatitis A	1, 2, 3
	Hepatitis A, relapsing	4
Hepatitis B (acute)	Acute hepatitis B	2, 3
Hepatitis C (acute)	Acute hepatitis C	2, 3
Upper respiratory infections	Upper respiratory infections	1, 2
Otitis media	Otitis media: acute	15
	Otitis media: chronic	103
Lower respiratory infections	Lower respiratory infections	2, 3
Influenza	Influenza	2, 3

Diphtheria	Diphtheria	2, 3
Pertussis	Pertussis, acute	1, 2, 3
Tetanus	Tetanus	3
Measles	Measles	2, 3
Rubella	Rubella	1
Varicella	Varicella	1
Herpes zoster	Herpes zoster	4, 9
Mumps	Mumps	2, 3
***Haemophilus influenzae* type b**	Haemophilus influenza type b disease	3
Pneumococcal disease	Invasive pneumococcal disease	3
Meningococcal disease	Meningococcal disease	3
Other meningitis and encephalitis	Other meningitis and encephalitis	3
Dengue	Dengue fever	1, 2, 3, 4
Ross River virus	Ross River virus infection	131, 4
Barmah Forest virus	Barmah Forest virus infection	131, 26
Malaria	Malaria	2, 3
Trachoma	Blindness due to trachoma[c]	115, 116
	Low vision due to trachoma[c]	113, 114
Campylobacteriosis	Gastrointestinal infection	5, 6, 7
Salmonellosis	Gastrointestinal infection	5, 6, 7
Rotavirus	Gastrointestinal infection	5, 6, 7
Other gastrointestinal infections	Gastrointestinal infection	43, 5, 6, 7
Urinary tract infections	Urinary tract infections	2, 3

(a) See ABDS 2018 health states.

(b) Part of infertility envelope.

(c) Part of vision envelope.

Prevalence estimation

The primary data sources used for infectious diseases are listed below. 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 4.21: Key data sources for infectious diseases

Data source	Disease
National Notifiable Diseases Surveillance System (NNDSS)	Tuberculosis, syphilis, chlamydia, gonorrhoea, hepatitis A, hepatitis B, diphtheria, pertussis, tetanus, measles, mumps, rubella, *Haemophilus influenzae* 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 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.

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, hepatitis A and hepatitis B 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; 2019) and enhanced surveillance studies (Fagan et al. 2013; Ressler et al. 2013) as used in the ABDS 2011 and ABDS 2015 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).

The number of BEACH GP encounters observed by age and sex was compared with the corresponding number of national GP consultations in each of the reference years (based on Medicare Benefits Scheme claims). 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 (using methods described by Britt et al. 2016). 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 2015 were applied to the 2018 population to attain estimates for the year 2018. This was done for otitis media, varicella, herpes zoster, lower respiratory infections, influenza and upper respiratory infections.

For urinary tract infections, BEACH data for 2015 were not available. Instead, age/sex-specific ratios were calculated between hospitalisations and BEACH data in 2011. Estimated ratios were then applied to separations data in the years 2015 and 2018 to calculate expected disease incidence in the community in those years. This assumes that the proportion of cases identified via notifications or separations data is consistent with those identified in general practices from 2010–2012.

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.

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 (The Kirby Institute 2020)
- the annual incidence of hepatitis B and C infections (The Kirby Institute 2020)
- age-specific proportion of newly acquired Hepatitis B infection that are symptomatic (Shepard et al. 2006)
- 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).

Additionally, 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.

Other infections

YLD was derived indirectly by applying the YLL-to-YLD ratio for all specified infectious diseases combined to the YLL for other unspecified infectious diseases.

Sub-national estimates

Prevalence estimates by state and territory as well as by remoteness area and socioeconomic group were calculated by applying proportions from the NHMD to national estimates.

2015, 2011 and 2003 estimates

Prevalence estimates for 2003, 2011 and 2015 were calculated from the data sources and method as described for 2018.

Indigenous specific estimates

Where possible, prevalence estimates for the Indigenous population were obtained from the same data source as used for national prevalence estimates. However, for most causes, indirect methods were used by applying rate ratios from the NHMD to the national prevalence estimates to derive Indigenous prevalence.

The same durations were applied for Indigenous estimates as used for national estimates, with the exception of untreated chronic otitis media.

National Hospital Morbidity Database (NHMD)

Indigenous estimates based on hospitalisation data were adjusted for Indigenous under-identification using standard adjustment factors outlined in Chapter 4 (see Years lived with disability (YLD)).

The Kirby Institute reports

Indigenous estimates for HIV/AIDS were based on prevalence estimates by the Kirby Institute (2020).

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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 2018, 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 the table below. The ICD-10 codes used to identify injury causes are shown in ABDS 2018 list of diseases, conditions and injuries.

Table 4.22: List of injury categories used in the ABDS 2018 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
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

The overarching methods for estimating non-fatal burden in the ABDS 2018 are the same as for the ABDS 2011 and the ABDS 2015; however, the epidemiological inputs used to estimate injury prevalence have been updated since the ABDS 2015. The overall differences in the inputs and the impact of these on the results are described further below.

In the ABDS 2018, 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.

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.

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 the table below.

Table 4.23a: Model inputs for short-term injuries, health states, and durations by admission status (admitted and non-admitted)

Cause sequelae	Short-term health state identifier[(a)]	Short-term admitted duration (years)	Short-term non-admitted duration (years)
Burn (non-airway) - minor	144	0.077	0.038
Burn (non-airway) - severe	146	0.164	0.164
Airways burn	149	0.077	0.077
Dislocation - shoulder joint	153	0.170	0.148
Dislocation - shoulder other	184	0.178	0.132
Dislocation - hip	151	0.110	0.085
Dislocation - knee	152	0.110	0.112
Dislocations - other	154	0.178	0.132
Drowning short-term	155	0.011	0.005
Fracture - neck of femur	162	0.216	0.197
Fracture - other than neck of femur	165	0.233	0.167
Crush injury	150	0.167	0.030
Severe chest injury	187	0.148	0.115
Abdominal /pelvic injuries	187	0.058	0.058
Fracture - humerus	156	0.175	0.142
Fracture - patella	167	0.359	0.258
Fracture - clavicle or scapula	156	0.175	0.142
Fracture - face bone	157	0.126	0.101
Fracture - foot bone	158	0.134	0.099
Fracture - hand bone	160	0.099	0.110
Fracture - pelvis	169	0.167	0.148
Fracture - pelvis (coccyx)	184	0.233	0.205
Fracture - radius or ulna	171	0.132	0.112
Fracture - sternum / ribs	174	0.148	0.115
Fracture - vertebral column	175	0.233	0.205
Fracture - other	176	0.167	0.148
Amputation of finger/s excl thumb	134	0.500	0.500
Amputation of thumb	135	0.500	0.500
Amputation of both arms	137	0.500	0.500

Amputation of one arm	237	0.500	0.500	
Amputation of toe	139	0.500	0.500	
Amputation of one leg	140	0.500	0.500	
Amputation of both legs	142	0.500	0.500	
Injured nerves	177	0.170	0.099	
Injury to eyes	179	0.123	0.137	
Superficial injuries	184	0.115	0.049	
Open wound	184	0.099	0.049	
All other injuries	184	0.008	0.005	
Poisoning short-term	185	0.011	0.005	
Soft tissue injuries	154	0.178	0.132	
SCI at neck - complete severe	190	0.500	0.500	
SCI at neck - incomplete severe	215	0.500	0.500	
SCI at neck - incomplete moderate	214	0.500	0.500	
SCI at neck - mild	213	0.077	0.077	
SCI below neck - complete severe	188	0.500	0.500	
SCI below neck - incomplete severe	215	0.500	0.500	
SCI below neck - incomplete moderate	214	0.500	0.500	
SCI below neck - mild	213	0.077	0.077	
Fracture - tibia or fibula	167	0.359	0.258	
Fracture - ankle	167	0.359	0.258	
TBI ST minor	246	0.101	0.096	
TBI ST moderate-severe	180	0.110	0.074	
TBI skull fracture	173	0.126	0.101	

(a) See ABDS 2018 health states.

Table 4.23b: Model inputs for long-term injuries, proportions and excess mortality by admission status (admitted and non-admitted)

Cause sequelae	Long-term health state identifier[(a)]	Long-term admitted proportion	Long-term non-admitted proportion	Long-term Mortality risk ratio[(b)]
Burn (non-airway) - minor	145	1	1	45+ 2.1
Burn (non-airway) - severe	147	1	1	45+ 1.3
Dislocation - hip	151	1	1	1
Dislocation - knee	152	1	0	1
Dislocations - other	154	1	0	1
Drowning short-term	155	1	0	1
Fracture - neck of femur	163	1	1	Under 50 1.0 51 to 75 3.97 75+ 2.42

Fracture - other than neck of femur	176	1	1	1
Crush injury	150	1	1	5.23
Severe chest injury	187	1	0	1
Abdominal /pelvic injuries	187	1	1	1
Fracture - humerus	156	1	1	1
Fracture - patella	167	1	1	1
Fracture - clavicle or scapula	156	1	0	1
Fracture - face bone	157	1	0	1
Fracture - foot bone	158	0	1	1
Fracture - hand bone	160	1	0	1
Fracture - pelvis	169	1	1	1
Fracture - pelvis (coccyx)	184	1	1	1
Fracture - radius or ulna	171	1	0	1
Fracture - sternum / ribs	174	1	1	1
Fracture - vertebral column	175	1	1	1
Fracture - other	169	1	1	1
Amputation of finger/s excl thumb	134	1	1	1
Amputation of thumb	135	1	1	1
Amputation of both arms	137	1	1	1
Amputation of one arm	237	1	1	1
Amputation of toe	139	1	1	1
Amputation of one leg	140	1	1	1
Amputation of both legs	142	1	1	1
Injured nerves	178	1	0	1
Poisoning short-term	216	1	0	1
SCI at neck - complete severe	190	1	1	Under 60 5.03 60+ 2.48
SCI at neck - incomplete severe	215	1	1	Under 60 5.03 60+ 2.48
SCI at neck - incomplete moderate	214	1	1	
SCI below neck - complete severe	188	1	1	Under 60 2.72 60+ 1.89
SCI below neck - incomplete severe	215	1	1	Under 60 2.72 60+ 1.89
SCI below neck - incomplete moderate	214	1	1	Under 60 5.03 60+ 2.48
Fracture - tibia or fibula	167	1	1	1
Fracture - ankle	167	1	1	1
TBI minor	181	1	1	1

TBI moderate-severe	182 183	0.75 0.25	0.75 0.25	2.18 2.18
TBI skull fracture	173	1	1	1

(a) See ABDS 2018 health states.

(b) Sourced from Haagsma and others (2015).

Some exceptions were that Australian-specific direct evidence was used 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 modelled for ABDS 2015 using DisMod II, based on incident cases derived from the NHMD and the NNAPEDC. Estimates for 2018 were generated from the 2015 modelled estimates using incidence and mortality for the years 2015 to 2017.

Injury cases were identified in the NHMD based on separations in the 2003, 2011, 2015 and 2018 calendar years. The NNAPEDC for 2013–14 to 2018–19 was used to estimate incidence of non-admitted cases using information about the diagnosis.

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.

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 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 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.

- **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 and non-admitted prevalence:** The modelling done for ABDS 2015 using DisMod II (modelled on the short-term admitted incidence, the remission and excess mortality of each sequela) has been adapted for the ABDS 2018 study because, of the inputs for DisMod II, only the admitted incidence may have changed. The ratio of long term to short-term prevalence from 2003, 2011 and 2015 is used to calculate ABDS 2018 estimates for long-term prevalence from updated short-term prevalence for those years. Estimates for long-term prevalence in 2018 are calculated by adding the incidence from 2015, 2016 and 2017 (adjusted for all-cause mortality) to the long-term prevalence in 2015. The same methods are used for **long-term non-admitted prevalence**.

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 2018–19. This data set included a diagnosis variable.

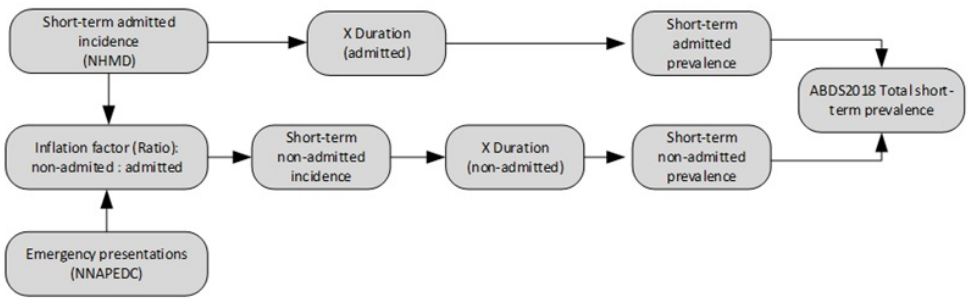
As diagnosis data were provided in a number of classifications, only jurisdictions that had more than 95% of emergency department records coded to an ICD-10 AM classification were included in the analysis. Hence, all records from New South Wales and Western Australian hospitals were excluded. Further 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 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 2018–19) 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 2018–19 were applicable to 2003, 2011, 2015 and 2018.

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 2015).

Figure 4.1: ABDS 2018 Short-term injury prevalence – Steps and data sources for calculating prevalence of injury sequelae



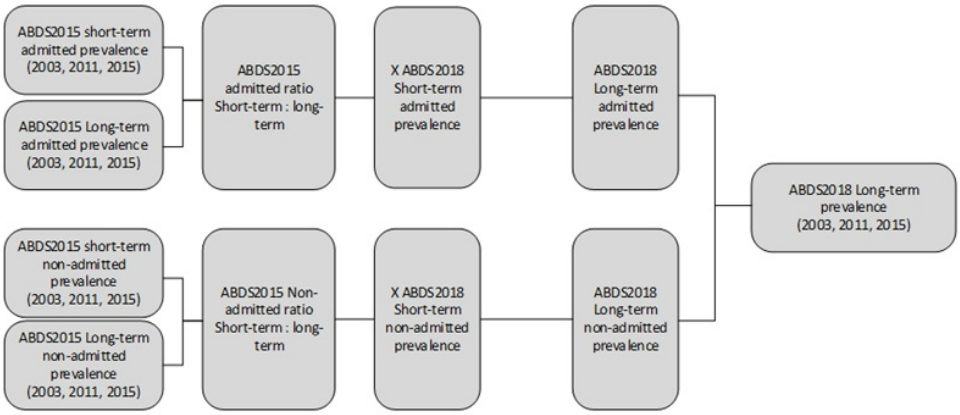
Long-term sequelae

Long-term consequences of injury reflect the functional consequences that persist more than 1 year after the injury. In the ABDS 2015, the respective national all-cause mortality rates and populations were used for DisMod II calculations for each ABDS reference period (2003, 2011 and 2015) 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) for the ABDS 2015. 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.

The ratio between short and long term prevalence calculated in the ABDS 2015 was applied to the short term injury prevalence calculated in the ABDS 2018 to estimate the long term injury prevalence for the 2003, 2011 and 2015 reference years.

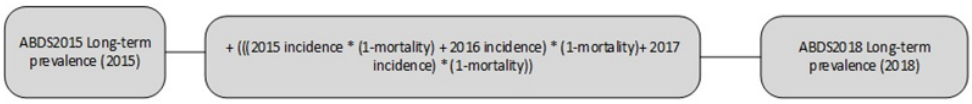
Figure 4.2: ABDS 2018 long-term injury prevalence for 2003, 2011, 2015 – Steps and data sources for calculating prevalence of injury sequelae



The values for these inputs for each long-term injury sequela were sourced from Haagsma et al. (2015). The inputs estimated from this source are presented in model inputs for durations, remission, and excess mortality by admission status (admitted and non-admitted).

Estimates for long-term prevalence in 2018 are calculated by adding the incidence from 2015, 2016 and 2017 (adjusted for all-cause mortality) to the long-term prevalence in 2015.

Figure 4.3: ABDS 2018 long-term injury prevalence for 2018 – Steps and data sources for calculating prevalence of injury sequelae



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 or the ABDS 2018 (see box below for more detail).

Box 4.1: 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' (GBD 2013 Collaborators 2015).

There was insufficient detail in the GBD methods paper to implement a similar approach in the ABDS 2018. As a result, the ABDS 2018 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 for 2015 and 2018 by applying the 2016 ASGS remoteness areas and 2016 SEIFA population-based IRSD quintiles to the SA2 recorded in hospital separations data. For 2011 and 2003 estimates, 2011 ASGS remoteness areas and SEIFA population-based quintiles were used. The same inflation ratios were applied to sub-national data.

The long-term national prevalence 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).

2015, 2011 and 2003 estimates

The approach used to estimate 2015, 2011 and 2003 prevalence was the same as that used for 2018 estimates. The prevalence of short-term and long-term sequelae was calculated using the same methods.

Indigenous specific estimates

Indigenous estimates of non-fatal injury burden used the same methods as for the national estimates for 2018, 2011 and 2003.

For short-term prevalence, Indigenous cases of short-term injury from the NHMD were adjusted for under-identification using standard adjustment factors from hospital data quality studies undertaken by the AIHW (see Methodological choices specific to Indigenous estimates). The national inflation factors used to adjust for non-admitted injuries were applied to the adjusted Indigenous separations (see *Adjusting for non-admitted injuries* above).

Long-term injury prevalence was estimated using long-term/short-term ratios derived from the national study and applying them to the indigenous short-term injury estimates.

The conversion of YLD by nature of injury to external cause used indigenous specific age-sex matrices.

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Kidney and urinary conditions

Sequelae and health states

Sequelae and health states assigned to kidney and urinary conditions are shown in the table below. 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.

Table 4.24: Sequelae, health states and duration for kidney & urinary conditions

Disease	Sequela	ABDS 2018 health state identifier[a]	Duration
Chronic kidney disease	Asymptomatic chronic kidney disease	262	. .
	Anaemia due to stage 3 chronic kidney disease[b]	195, 196, 197	12 months
	Stage 4 chronic kidney disease	41	12 months
	Anaemia due to stage 4 chronic kidney disease[b]	195, 196, 197	12 months
	End-stage kidney disease on dialysis	43	12 months
	End-stage kidney disease after transplant	42	12 months
	Untreated end-stage kidney disease	22	5.5 months
Enlarged prostate	Enlarged prostate	47	12 months
Kidney stones	Kidney stones	193	2 weeks
Interstitial nephritis	Interstitial nephritis	2, 3	7 days, 14 days
Other kidney and urinary diseases	Other kidney and urinary diseases	. .	. .

(a) See ABDS 2018 health states.

(b) Part of the anaemia envelope.

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 et al. (2014). These were applied to people with chronic kidney disease *and* anaemia. Specifically, the definitions used were for all those aged 5 and over (excluding pregnant women). See the section on methods for blood & metabolic disorders for more information on the methods used to estimate the 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 2018 (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 2014).

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 2014). 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 2018, 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 2018). The age and sex distribution was further refined using the age and sex of people who were hospitalised for N18.3 in 2018.

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 2018, 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 2018.

End-stage kidney disease treated with dialysis or transplant

Registry data from the Australia and New Zealand Dialysis and Transplant Registry in 2018 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 2018. The 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 2014a), 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 2018 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 the National Health Services Information Analysis Asset (NIHSI AA v0.5) 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 2018. The ratios were determined using 7 years of data (admitted patient data between 1 July 2010 and 30 June 2017), assuming that the condition is chronic. Health loss was assumed to apply for the entire year. The same ratios were also applied to NHMD data for 2003, 2011 and 2015 prevalence estimates using a 7-year lookback.

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 2018 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 2018, 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 2018. 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 2018 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.

2015, 2011 and 2003 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, 2011 and 2015.

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 and 2018, 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 2018 for stages 3 and 4 chronic kidney disease with anaemia were also applied to the 2015, 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.

Indigenous specific estimates

The same methods and data sources were used to derive Indigenous estimates for kidney and urinary diseases, except that trend data for chronic kidney disease prevalence were not available for Indigenous Australians. Instead, ratios of treated end-stage kidney disease to stage 4 and stage 3 CKD for 2011 were applied to treated end-stage kidney disease data for 2003 and 2018 to estimate the prevalence of stage 3 and 4 CKD for those reference years. Indigenous data were directly available from the Australia and New Zealand Dialysis and Transplant Registry and the NHMD. Biomedical data for stage 3 CKD and anaemia and stage 4 CKD was sourced from the AATSHIS 2012–13.

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

The national severity distributions for anaemia were used for 2018, 2011 and 2003 Indigenous estimates.

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Mental and substance use disorders

The majority of morbidity estimates were based on methods used for the ABDS 2015, except for eating disorders.

Sequelae and health states

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

Table 4.25: Sequelae and health states for mental and substance use disorders

Disease	Sequela	ABDS 2018 health state identifier[(a)]
Depressive disorders	Dysthymia	262, 86
	Major depressive disorder	262, 86, 87, 88
Anxiety disorders	Anxiety disorders	262, 83, 84, 85
Bipolar affective disorder	Bipolar disorder	87, 89, 90
Alcohol use disorders	Alcohol dependence	235, 262, 73, 74, 75
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[(b)]	100, 101, 102, 243, 99
Other mental & substance use disorders	Other mental & substance use disorders	83

(a) See ABDS 2018 health states.

(b) Part of the intellectual disability envelope.

Prevalence estimation

Data sources

Key data sources to estimate mental and substance use disorder prevalence are shown below.

Table 4.26: 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, and bipolar disorder
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 and 2018)
GBD 2017	Anorexia nervosa, bulimia nervosa, cannabis use disorders

GBD 2019	Cocaine use disorders

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 disorder, 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 2018, 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 2013 and followed through to 2018. IDEA data were available for people up to the age of 35.

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 (see table below). 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 4.27: 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 National 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 2018 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 (see table above). 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, 2015 or 2018) 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.

2015, 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 et al. (2004). These estimates were then adjusted for change over time, based on data from the National Opioid Pharmacotherapy Statistical Annual Data collection.

The data source for amphetamine disorders (Degenhardt et al. 2016) included estimates for 2003–04 and 2011–12, so each of these was used for the corresponding reference year. The data source for 2015 estimates was the same as the 2018 estimates but 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, 2011 and 2015 these were based on hospitalisations during the 2003, 2011 and 2015 calendar year, respectively.

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, 2011 and 2015.

Indigenous specific estimates

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

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

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

For drug use disorders, CIMHA modelling rate ratios were used to derive Indigenous prevalence for opioid, cannabis and amphetamine dependence (for all levels of severity). For cocaine dependence, rate ratios from self-reported survey data (AIHW 2019) were used to derive Indigenous prevalence (for all levels of severity).

For attention-deficit/hyperactivity disorder and conduct disorder, Indigenous prevalence estimates were based on the rate ratios from the Longitudinal Survey of Australian Children and CIMHA. An average of these rate ratios was applied to national prevalence estimates.

For eating disorders and autism spectrum disorders total population prevalence rates were applied to the Indigenous population.

For intellectual disability, Indigenous prevalence estimates were calculated using rate ratios derived from the original 2011 ABDS Indigenous estimates and population. The rate ratios were applied to the 2018 ABDS national rate, which was then applied to the 2018 Indigenous population.

Other drug use disorders and other mental and substance use disorders were all based on hospitalisation rate ratios specific to 2018.

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Musculoskeletal conditions

Sequelae and health states

Sequelae and health states assigned to musculoskeletal conditions are shown in the table below. Durations and assumptions are outlined in subsections for individual diseases.

Table 4.28: Sequelae and health states for musculoskeletal conditions

Disease	Sequela	ABDS 2018 health state identifier[(a)]
Osteoarthritis	Osteoarthritis of the knee	262, 126, 127, 128
	Osteoarthritis of the hip	262, 126, 127, 128
Gout	Musculoskeletal problems caused by gout	132, 133
Rheumatoid arthritis	Musculoskeletal problems caused by rheumatoid arthritis	262, 130, 131, 132
Back pain & problems	Back pain & problems	262, 234, 254, 233, 255, 241, 242, 239, 240
Other musculoskeletal conditions[(b)]	Other musculoskeletal problems	262, 126, 127, 128, 130, 131, 132

(a) See ABDS 2018 health states.

(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 2017–18, 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. 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 2017–18 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 2017–18. 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 study 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 2018, and as a result, there is potential overlap between other musculoskeletal conditions and osteoarthritis with selected injuries such as fractures and dislocations (see ABDS diseases associated with previous injury).

Osteoarthritis

The NHS 2017–18 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 2017—85% to knee and 15% to hip.

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

Table 4.29: ABDS severity distributions (%) for osteoarthritis

Reference year (Data source)	Asymptomatic	Mild	Moderate	Severe
2003 (2011-12 AHS)	14.5	46.9	28.0	10.6
2011 (2011-12 NHS)	14.5	46.9	28.0	10.6
2015 (2014-15 NHS)	10.8	42.7	32.7	13.8

2018 (2017-18 NHS)	13.6	41.9	30.9	13.5

Gout

As a breakdown of chronic or acute gout was not available in the NHS 2017–18 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.

Note that the AIHW has changed how they report gout prevalence using the 2017–18 NHS data and now counts any person who reported ever having gout (AIHW 2020).

Rheumatoid arthritis

The NHS 2017–18 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 2017–18. Health loss is assumed to last for the entire year.

Table 4.30: ABDS severity distributions (%) for rheumatoid arthritis

Reference year (Data source)	Asymptomatic	Mild	Moderate	Severe
2003 (2014–15 NHS)	16.2	32.1	35.0	16.7
2011 (2014–15 NHS)	16.2	32.1	35.0	16.7
2015 (2014–15 NHS)	16.2	32.1	35.0	16.7
2018 (2017–18 NHS)	13.6	38.1	30.4	18.0

Back pain and problems

The NHS 2017–18 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 2017–18. 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 distributions are provided below.

Table 4.31: ABDS severity distributions (%) for back pain and problems

Reference year (Data source)	**Asymptomatic**	**Mild**	**Moderate**	**Severe**	**Very severe**
2003 and 2011 (2014–15 NHS)					
Without leg pain (%)	14.1	43.2	19.6	6.2	1.4
With leg pain (%)	. .	7.6	5.2	2.1	0.5
Total	14.1	50.8	24.8	8.3	1.9
2015 (2014-15 NHS)					
Without leg pain (%)	14.1	43.2	19.6	6.2	1.4
With leg pain (%)	. .	7.6	5.2	2.1	0.5
Total	14.1	50.8	24.8	8.3	1.9
2018 (2017-18 NHS)					
Without leg pain (%)	13.5	39.4	23.1	6.5	1.6
With leg pain (%)	. .	7.0	6.1	2.2	0.6

Total	13.5	46.3	29.2	8.6	2.2

Other musculoskeletal conditions

The prevalence of other musculoskeletal conditions was also derived from the NHS 2017–18. 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 2017–18

Table 4.32: ABDS severity distributions (%) for other musculoskeletal conditions

Reference year (Data source)	Asymptomatic	Level 1	Level 2	Level 3	Level 4	Level 5	Level 6
2003 (2014-15 NHS)	17.8	23.3	21.8	13.1	13.1	9.0	2.0
2011 (2014-15 NHS)	17.8	23.3	21.8	13.1	13.1	9.0	2.0
2015 (2014-15 NHS)	17.8	23.3	21.8	13.1	13.1	9.0	2.0
2018 (2017-18 NHS)	14.5	24.2	21.3	14.6	14.6	9.5	1.4

It is important to note that the NHS 2017–18 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 ABDS diseases associated with previous injury).

Sub-national estimates

National prevalence estimates were apportioned based on sex and combined age-specific estimates from the NHS 2017–18 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. Given that the NHS did not include people living in *Very remote* areas an adjustment based on population size was performed to inflate prevalence estimates to account for these people.

2015, 2011 and 2003 estimates

Due to some data issues with the coding of musculoskeletal conditions in the 2011–12 NHS (ABS 2015) the 2011–12 survey data for back pain & problems, rheumatoid arthritis, gout and other musculoskeletal conditions were not used. Instead, the prevalence for 2011 for these diseases was estimated using linear spline interpolation using the age/sex prevalence data for the other reference years. For other musculoskeletal conditions, estimates for 2003 and 2011 prevalence rates from 2015 were applied to the 2011 and 2003 populations. The severity distribution from the 2014–15 NHS was used for both 2003 and 2011 estimates for other musculoskeletal conditions, rheumatoid arthritis and back pain & problems.

The same methods used for the 2018, 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.

It is important to note that some of the differences between the reference years will be due to differences in the severity distributions

Indigenous specific estimates

The methods used for estimating non-fatal musculoskeletal conditions burden for the Indigenous population was similar to the method used for national estimates.

The AATSIHS 2012–13 was the primary data source for 2011 estimates. After consultation with ABS regarding a specific data quality issue with published musculoskeletal data from the survey, the ABS provided the AIHW with revised data for back pain and problems and other musculoskeletal conditions for our analysis. The revised data are currently unpublished by the ABS, but available on request. Where the RSE was high for certain 5-year age groups, the national 5-year age distribution was applied to the Indigenous prevalence estimate specific for those age groups.

As the musculoskeletal data items from previous ABS Indigenous health surveys were not comparable to those from the AATSIHS 2012–13, and there was little evidence to suggest a recent change in prevalence of musculoskeletal conditions in the Indigenous population, Indigenous estimates for 2003 were derived from rates from the AATSIHS 2012–13 applied to the 2003 Indigenous population.

The severity distribution for each condition was assumed to be the same as for the national estimates for both 2011 and 2003.

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Neurological conditions

Sequelae and health states

Sequelae and health states assigned to the neurological conditions are shown below. Durations and assumptions are outlined in subsections for individual diseases.

Table 4.33: Sequelae and health states for neurological conditions

Disease	Sequela	ABDS 2018 health state identifier[(a)]
Epilepsy	Epilepsy	207, 248, 249
Dementia	Dementia	58, 59, 60
Parkinson disease	Parkinson disease	70, 71, 72
Multiple sclerosis	Multiple sclerosis	63, 64, 65
Motor neurone disease	Motor neurone disease	65
Migraine	Migraine	61
Guillain-Barré syndrome	Guillain-Barré syndrome	188

(a) See ABDS 2018 health states.

Prevalence estimation

Epilepsy

For the ABDS 2018, epilepsy is defined as a chronic disorder of the brain characterised by recurrent seizures, as consistent with the GBD 2017. National sex-specific prevalence estimates of self-reported epilepsy were obtained from the NHS 2017–18. The NHS 2017–18 did not report on *Very remote* areas, so prevalence estimates were adjusted to account for *Very remote* areas. Age–sex specific distributions couldn't be obtained from the NHS 2017–18 as the RSEs for the epilepsy counts in many age groups were too high. Instead, an age distribution by state and sex was calculated using hospitalisation data from the NHMD. Counts of hospital separations from the NHMD were adjusted to account for readmissions using linked hospitalisation and deaths data from the NIHSI AA v0.5. Admitted hospitalisations data were available from the NIHSI AA v0.5 for New South Wales, Victoria, South Australia and Tasmania. Where numbers allowed, that is, the numbers were not low enough to cause volatility in estimates, state-specific ratios were calculated. Otherwise, data from all 4 jurisdictions were used for the ratios. For states and territories that did not have hospitals data in the NIHSI AA v0.5, ratios using all 4 jurisdictions combined were used. This assumes that persons-to-separations ratios were similar across all jurisdictions. The age and state/territory distributions for each sex were then applied to the NHS 2017–18 national sex-specific prevalence estimates to obtain the estimates by age, sex and state/territory. State and territory estimates were summed to obtain final national prevalence estimates. There was no direct Australian data source to estimate the severity of epilepsy as defined in the ABDS 2018, therefore the severity distribution of epilepsy was based on a study conducted in the United Kingdom by Moran et al. (2004).

Dementia

Dementia includes Alzheimer's disease (the most common form), vascular dementia, dementia with Lewy bodies and frontotemporal dementia (ICD-10-AM: F00–F03, G30–G31). Prevalence estimates for dementia were calculated by applying the prevalence rates used in the *Dementia in Australia* report (AIHW 2021) to the Australian estimated resident population. The *Dementia in Australia* report uses rates reported by the 2015 World Alzheimer Report for adults aged 60 and older. For adults under 60 years, the report uses rates published in a community-based study conducted in Sydney, Australia in 2008 (Withall et al. 2014), with sex ratios from an international epidemiological study applied (Harvey et al., 2003). 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.

For more information on the methods used to derive dementia prevalence estimates, see the methods of estimating the number of Australians living with dementia in the AIHW *Dementia in Australia* report (AIHW 2021).

Parkinson disease

There was a lack of recently published, high quality, population-based Australian studies on Parkinson disease at the time of analysis. Thus, prevalence was estimated using rates from a number of international studies (de Rijk et al. 1995; de Rijk et al. 2000; Willis et al. 2013). Prevalence rates from the de Rijk studies were 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 an increasing linear trend in the rates of Parkinson disease in these age groups. For Australians aged under 30, the prevalence of Parkinson disease was assumed to be zero.

A severity distribution by broad age groups was derived from unpublished data from the Queensland Parkinson Project.

Multiple sclerosis

Prevalence of multiple sclerosis was estimated using 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). A sex ratio of 3:1 (female: male) was applied to the national prevalence based on data from the Multiple Sclerosis Australia database (Covance Pty Ltd & Palmer 2011). The age distribution of multiple sclerosis was modelled based on the 2018 Survey of Disability, Ageing and Carers (SDAC). The 2018 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, 2018 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 (MND) are a group of progressive neurological disorders (including amyotrophic lateral sclerosis) that destroy motor neurones. National prevalence of MND was estimated by applying person-to-separation ratios calculated using linked hospitalisations and deaths data from the NIHSI AA v0.5 to hospital separation counts from the NHMD. Admitted hospitalisation data were available from the NIHSI AA v0.5 for New South Wales, Victoria, South Australia and Tasmania, however the numbers were low enough to cause volatility in estimates and therefore the data from all 4 jurisdictions were combined. These combined ratios were applied to all states and territories, assuming that persons-to-separations ratios were similar across all jurisdictions.

Since the GBD 2017 did not have a disability weight specific to motor neurone disease, the disability weight for severe multiple sclerosis was applied.

Migraine

According to Headache Australia, migraine is an episodic condition characterised by quiescent and relapse phases, known as headaches that typically last 4–72 hours. Age-and-sex specific prevalence estimates for migraine were based on the NHS 2017–18 self-reported data. As data was not available from the NHS at the required age group disaggregation, some modelling was undertaken to obtain estimates for the older age groups. The NHS 2017–18 did not report on *Very remote* areas, so prevalence estimates were adjusted to account for *Very remote* areas. A duration of 27 days per year was applied to the national estimates to obtain point prevalence. This is the duration used in GBD 2017 which was based on two meta-analyses which reported an average frequency of 43.8 episodes per year and an average duration per episode of 14.87 hours (GBD 2017 Disease and Injury Incidence and Prevalence Collaborators 2018).

Guillain-Barré syndrome

Guillain-Barré syndrome (GBS) is a disease of the peripheral nervous system that might develop spontaneously or after a systemic infection or other stress. National age- and sex-specific prevalence of GBS were estimated by applying person-to-separation ratios calculated using linked hospitalisations and deaths data from the NIHSI AA v0.5 to hospital separation counts from the NHMD. Admitted hospitalisation data were available from the NIHSI AA v0.5 for New South Wales, Victoria, South Australia and Tasmania, however the numbers for Tasmania were low enough to cause volatility in estimates and were therefore not used in isolation. For Tasmania, Western Australia, the Australian Capital Territory and the Northern Territory, the data from New South Wales, Victoria, South Australia and Tasmania were combined and these ratios applied, assuming that persons-to-separations ratios were similar across the jurisdictions. Additionally, ratios were only available by sex, therefore the same ratio was applied to each age group within each sex. The person counts were then summed to obtain national estimates. A duration of 6.7 months was based on the GBD 2013 (GBD 2013 Collaborators 2015) and was applied to the national prevalence estimates to obtain national 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, sex-specific proportions of people who self-reported this condition were obtained at all the sub-national levels (state and territory, remoteness and socioeconomic group) from the NHS 2017–18. These proportions were applied to the national prevalence estimates to obtain the sub-national estimates.

For epilepsy, the proportions of people who self-reported this condition were obtained from the NHS 2017–18 by socioeconomic group, and by sex and remoteness area. These proportions were applied to the national prevalence estimates to obtain the sub-national estimates. State and territory prevalence estimates were generated during the process of obtaining national estimates, therefore see methods for national epilepsy estimates.

For motor neurone disease (MND) and Guillain-Barré syndrome (GBS), the number of separations at the state and territory level were derived from the NHMD directly and person-to-separation ratios obtained from the NIHSI AA v0.5 were applied to these separation counts, as consistent with the national estimates. For MND, state and territory-specific ratios were not available, therefore ratios calculated using data from the 4 jurisdictions for which hospitalisations data was available in the NIHSI AA v0.5 were applied. For GBS state and territory-specific ratios were available for South Australia, Victoria and New South Wales and for all other jurisdictions combined state and territory ratios were used. For both MND and GBS, remoteness and socioeconomic group estimates were calculated by applying proportions of NHMD separations by age, sex and remoteness/socioeconomic group to 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 (Menzies Health Research Group, van der Mei & Taylor 2018). 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, estimates by state and territory, remoteness area and socioeconomic group were derived by applying proportions of deaths due to the condition from the NMD to the national estimates.

2015, 2011 and 2003 estimates

Where available, the methods and data sources used to estimate prevalence in 2003, 2011 and 2015 were largely similar to those for 2018, however different years of data were used. For motor neurone disease and Guillain-Barré syndrome, prevalence estimates for 2015, 2011 and 2003 were also largely derived from the NHMD using data relevant to each reference year. However, since the NIHSI AA v0.5 only contains hospitalisations data from 2010–17, persons-to-separations ratios calculated for 2011 were also used for estimating prevalence in 2003.

For epilepsy, data were available from the NHS 2004–05, AHS 2011–12 and the NHS 2014–2015 and used for the 2003, 2011 and 2015 estimates respectively. Age, sex and state/territory distributions were calculated using NHMD separations data relevant to each reference year. The same person-to-separation ratios were applied to the count of hospital separations for each reference year.

For migraine, data was available from the NHS 2004–05, AHS 2011–12 and the NHS 2014–2015 and used for the 2003, 2011 and 2015 estimates respectively.

For multiple sclerosis, the same prevalence rates that were used for the 2018 estimates were also used for the 2015 estimates. The 2003 and 2011 estimates were based on the prevalence rates from an earlier study by Andrew Palmer (Palmer et al. 2013). The age distribution for the 2015 estimates were modelled based on 2015 NHMD and SDAC data, whereas the 2011 and 2003 estimates were modelled based on 2009 NHMD and SDAC data. The same sex ratio that was applied to the 2018 estimates was also applied to the 2015, 2011 and 2003 estimates.

For dementia and Parkinson disease the same prevalence rates which were used for the 2018 estimates were also used for the 2003, 2011 and 2015 estimates.

Indigenous specific estimates

Indigenous estimates based on hospital separations data (epilepsy and Guillain-Barré syndrome) were adjusted for under-identification using standard adjustment factors (see Years lived with disability (YLD)).

Indigenous estimates for motor neurone disease were based on mortality data and were adjusted for under-identification using standard adjustment factors (see Years of life lost (YLL)).

Indigenous prevalence for epilepsy and migraine were derived from the AATSIHS 2012–13 for 2011 estimates and the NATSIHS 2004–05 for 2003 estimates using a similar method as national estimates.

Indigenous dementia prevalence was obtained using three Australian studies (Li et al 2014, Radford et al. 2015, Smith et al. 2008). Severity distribution was obtained from the Koori Growing Old Well Study and the Barendregt & Bonneux (1998) studies. The same prevalence rates and severity distributions were applied for 2003 Indigenous estimates.

Due to the lack of Indigenous-specific data on Parkinson disease and multiple sclerosis, national prevalence rates were applied to the Indigenous population to derive Indigenous prevalence for both 2011 and 2003 estimates. For multiple sclerosis, prevalence rates were also adjusted to reflect the lower rates in Indigenous populations (compared to National populations) as found in a NZ study 2014 (Pearson JF et

al 2014) and found in comparisons of Australian hospitalisation and mortality data.

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Oral disorders

Sequelae and health states

Sequelae and health states assigned to oral disorders are shown below. Durations and assumptions are outlined in subsections for individual diseases.

Table 4.34: Sequelae and health states for oral disorders

Disease	Sequela	ABDS 2018 health state identifier[(a)]
Dental caries	Untreated dental caries (including failed restorations)	199, 262
Periodontal disease	Chronic periodontal disease	198, 262
Severe tooth loss	Severe tooth loss	200, 262

Other oral disorders	Other oral disorders	200

(a) See ABDS 2018 health states.

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 2017–18 (ARCPOH 2019). 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 (Do & Spencer 2016), and also past trends from the Child Dental Health Survey 2009 using the DMFT measure (caries in deciduous and adult teeth were both counted)(ARCPOH 2015, Ha et al. 2013).

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).

Prevalence for severe tooth loss originated from NSAOH dental examinations. The NSAOH 2017–18 no longer capture severe tooth loss, where only people with less than 21 teeth were captured. Hence transformations were made to deduce the dentate counts of severe tooth loss in ABDS 2018. Using NSAOH 2004–06 data for severe tooth loss, together with data published in NSAOH 2017–18 for people with edentulism and less than
21 teeth, severe tooth loss was estimated to be about 30% of those in these categories.

Other oral disorders

Estimates for other oral disorders were based on incidence of hospital separations in the 2018 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.

2015, 2011 and 2003 estimates

As the National Survey of Adult Oral Health data were collected in 2017–18 and 2004–06, the 2003 prevalence rates were from NSAOH 2004–06, and the 2018 prevalence rates were from NSAOH 2017–18. Between these time points, linear regression was used to derive the prevalence rates for 2011 and 2015. Prevalence rates were applied to the 2015, 2011 and 2003 population structures to calculate prevalence of dental caries, periodontal disease and severe tooth loss in 2015, 2011 and 2003, respectively.

Prevalence of dental caries in children from the Child Dental Health Survey 2009 were incorporated into the 2015 and 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 2015, 2011 and 2003 used the same approach as 2018, but drawn from data in the 2015, 2011 and 2003 calendar years, respectively.

Indigenous specific estimates

Due to the small sample size, age and sex-specific prevalence estimates for the Indigenous population were not directly available from the data sources used for national prevalence.

For 2018, 2011 and 2003, estimates for adult dental caries and periodontal disease were based on rate ratios of Indigenous:national rates from the National Survey of Adult Oral Health 2004–06 (Slade et al. 2007) applied to national age and sex distributions.

For 2018, 2011 and 2003 estimates for dental caries in Indigenous children were based on rate ratios of Indigenous:national rates from the Child Dental Health Survey 2009 applied to national age and sex distributions.

For severe tooth loss, Indigenous prevalence for 2018, 2011 and 2003 was based on data from AATSIHS 2012–13.

The 2018, 2011 and 2003 prevalence of other oral disorders among Indigenous Australians was based on analysis of NHMD adjusted for Indigenous under-identification using the standard adjustment factors (see Years lived with disability (YLD)).

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Reproductive and maternal conditions

Sequelae and health states

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

Table 4.35: Sequelae, health states and durations for reproductive & maternal conditions

Disease	Sequela	ABDS 2018 health state identifier[(a)]	Duration for acute sequelae
Maternal conditions			
Maternal haemorrhage	Anaemia due to maternal haemorrhage[(b)]	195, 196	1–3 months
	Surgical intervention: caesarean section	194	2 weeks
Maternal infections	Maternal sepsis	194	2 weeks
	Other maternal infections	2	1 week
Hypertensive disorders of pregnancy	Hypertensive disorder	194, 207	2 weeks–2 months
Obstructed labour	Surgical intervention: caesarean section	194	2 weeks
Early pregnancy loss	Early pregnancy loss due to ectopic pregnancy	194	2 weeks
	Early pregnancy loss due to other causes	193	1 week
Gestational diabetes	Diagnosed gestational diabetes	207	4 months

Other maternal conditions	Surgical intervention: caesarean section	193	2 weeks
Reproductive conditions			
Endometriosis	Endometriosis	193, 194	3 days per month
	Infertility due to endometriosis[c]	50, 51	. .
Uterine fibroids	Anaemia due to uterine fibroids[b]	195, 196	6 months
	Infertility due to uterine fibroids[c]	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[c]	50, 51	. .
	Polycystic ovarian syndrome	207	. .
Infertility	Infertility[c]	50, 51	. .
Other reproductive conditions	Anaemia due to other reproductive conditions[b]	195, 196	. .
	Pain due to reproductive conditions	192	2 weeks

(a) See ABDS 2018 health states.

(b) Part of anaemia envelope.

(c) Part of infertility 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 mutual 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 2018 was derived from the Australian and New Zealand Assisted Reproductive Database (Newman et al. 2019). Estimates were inflated to account for varying types of assisted reproductive technology.

The number of men and women seeking assistance for infertility in 2018 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 2018.

Age by sex distributions were the same as used in the ABDS 2011 and ABDS 2015, 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 (see table below).

Table 4.36: 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 2018 and whether they had previously given birth (at least 20 weeks gestation or 400 grams birthweight) was applied (see table below). 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 4.37: Women who gave birth in 2018, by maternal age and parity (%)

Maternal age	Primipara (no previous births)	Multipara (one or more previous births)
20–24 years	56.7	43.3
25–29 years	49.6	50.3
30–34 years	39.6	60.3
35–39 years	28.4	71.6
40–44 years	27.1	72.8
45 years and over	40.3	59.5

Source: 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 2018 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 2018 and 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 (see Sequelae, health states and durations for reproductive & maternal conditions). 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 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 were 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, Western Australia and the Australian Capital Territory were applied to Medicare Benefits Schedule data (method from 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 2018.

Medically induced abortions were included for 2018 and 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, Z37). Gestational diabetes is identified using the AIHW's matrix for assigning diabetes in pregnancy status (AIHW 2019b).

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

The prevalence of endometriosis in women aged 40–44 was derived from the Australian Longitudinal Study on Women's Health (ALSWH), a longitudinal cohort study, linked with MBS, PBS and/or admitted patient hospital data (AIHW 2019a). This prevalence was previously derived from ALSWH data that had not been linked. The cohort used for prevalence estimates was born between 1973 and 1978. Age distributions derived from the NHMD were applied to these estimates, to derive prevalence by age. Age distribution has been derived from GP visits previously.

Endometriosis severity was based on surgical intervention. Hospitalised cases of endometriosis in 2018 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 ALSWH, with an estimated 11.7% of women with endometriosis reporting infertility issues. These estimates were subtracted from the infertility envelope. This is further discussed in the infertility section.

Polycystic ovarian syndrome

Polycystic ovarian syndrome in women aged 34–39 was derived from the ALSWH. The cohort used for prevalence estimates were born between 1973 and 1978. Age distributions were derived from BEACH data and applied to these estimates, to determine prevalence by age.

Infertility estimates were derived from the ALSWH, with 14.5% of women with polycystic ovarian syndrome reporting infertility issues. These estimates were subtracted from the infertility envelope (see Infertility section).

Uterine fibroids

It was assumed people with burdensome uterine fibroids in 2018 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 (Buttram & Reiter 1981), 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 2018 Australian Estimated Residential Population. Due to limited data, male estimates were calculated using the male-to-female genital prolapse hospitalisations ratio in the year 2018, 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 et al. (2018) were applied to total female and male symptomatic prolapse estimates. The age-distribution was obtained from Lawrence et al. (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 2018 Australian Estimated Residential Population to derive estimates in year 2018. 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 2018, 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.

2015, 2011 and 2003 estimates

Estimates using hospital separations data used the same method as for 2018, but with 2015, 2011 and 2003 NHMD data.

Estimates using ALSWH, BEACH and epidemiological studies used the same rates or proportions as for 2018, applied to the 2011 or 2003 population, unless otherwise stated. This is because using earlier Australian Longitudinal Study on Women's Health surveys and BEACH data gave implausible estimates.

For endometriosis, prevalence derived from ALSWH linked data vary by year: 11.4% of women aged 40–44 in 2018 and 2015, and 9.6% of women aged 35–39 in 2011 and 2003 (AIHW 2019a).

Indigenous specific estimates

The same methods and data sources were used to derive Indigenous estimates, except where noted. Indigenous estimates based on hospital separations data (see above) were adjusted for under-identification using standard adjustment factors (see Years lived with disability (YLD)).

Estimates for polycystic ovarian syndrome were based on Indigenous: national rate ratios applied to national prevalence rates. The rate ratio for endometriosis was obtained from hospital separations data, and from epidemiological studies for polycystic ovarian syndrome.

Due to lack of data, Indigenous prevalence of endometriosis, genital prolapse and infertility was obtained by applying the national distribution directly to the Indigenous population. This assumes the underlying rate is the same between the Indigenous and non-Indigenous populations.

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Respiratory diseases

Sequelae and health states

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

Table 4.38: Sequelae and health states for respiratory diseases

Disease	Sequela	ABDS 2018 health state identifier[(a)]
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
Asbestosis	Asbestosis	55, 56, 57
Silicosis	Silicosis	55, 56, 57
Other pneumoconiosis	Other pneumoconiosis	55, 56, 57
Upper respiratory conditions	Upper respiratory	262, 207
Other respiratory disease	Other respiratory	207

(a) See ABDS 2018 health states.

Prevalence estimation

Asthma and upper respiratory conditions

The NHS 2017–18 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.

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 calculated using revised data weighted to the 2016 Census population supplied by the BOLD study team (Toelle et al. 2021) were applied to the 2018 ERP to generate prevalence estimates for the 2018 reference year. An assumption was made that the rate of COPD in Australia had not changed significantly between these time points.

The BOLD study experts advised that the rate of COPD in older people should increase with age, and the rates from GBD support this, so the data from BOLD was modelled using a polynomial curve for each sex to estimate rates for people aged 75 and over. This was done for the BOLD data weighted to the 2016, 2011 and 2006 populations supplied to the AIHW by the BOLD investigators.

Sarcoidosis, asbestosis, silicosis, other 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 using linked hospitalisations and deaths data from the NIHSI AA v0.5 were applied to the national hospitalisations to account for repeat admissions per person.

Mortality and hospitalisation data were used to estimate the prevalence of moderate and severe cases for these diseases. Prevalence for the mild or asymptomatic health states was then derived, by extrapolating these estimates for moderate and severe disease, based on severity distributions 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 below.

Table 4.39: GBD severity distributions (%) for sarcoidosis

	Asymptomatic	Mild	Moderate	Severe
Sarcoidosis - GBD 2013	23.9	55.0	16.0	5.1
Sarcoidosis - 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 asbestosis, silicosis and other pneumoconiosis are listed below.

Table 4.40: GBD severity distributions (%) for asbestosis, silicosis and other pneumoconiosis

	Asymptomatic	Mild	Moderate	Severe
Asbestosis - GBD 2013	30.8	43.9	17.8	7.5
Asbestosis - GBD 2015	23.0	32.7	12.9	31.4
Silicosis - GBD 2013	31.8	44.8	17.2	6.2
Silicosis - GBD 2015	23.4	33.2	13.2	30.2

Other pneumoconiosis – GBD 2013	29.0	41.6	16.2	13.3
Other pneumoconiosis – 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, asbestosis, silicosis and other pneumoconiosis and interstitial lung disease. The ratio was applied to YLL for other respiratory conditions identified using the ICD-10 codes outlined in Table 2.1.

Sub-national estimates

National estimates were apportioned into each state/territory, remoteness area and socioeconomic group, based on the proportions obtained from either survey (NHS) or hospitalisation (NHMD) data. Due to the small number of cases for asbestosis, silicosis and other pneumoconiosis, data from the NHMD and the NMD were used for all these diseases and the proportions applied to national estimates for each disease.

Given that the NHS did not include people living in *Very remote* areas an adjustment based on population size was performed to inflate prevalence estimates to account for these people for asthma and upper respiratory conditions.

2015, 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, asbestosis, silicosis, other pneumoconiosis and interstitial lung disease.

National 2003 estimates of asthma and upper respiratory conditions used a similar method to that used for 2018, 2015 and 2011 estimates but drew on the NHS 2004–05. Estimates of COPD were also based on the BOLD study, with rates weighted to the 2006 Census population applied to the 2003 population. The remaining conditions used a similar method but drew on 2003 hospital data.

Indigenous specific estimates

Indigenous estimates of asthma and upper respiratory conditions for 2018, 2011 and 2003 were based on self-reported data from the 2018–19, 2012–13 and 2004–05 National Aboriginal and Torres Strait Islander Surveys, using similar methods as for national estimates. As there were no Indigenous-specific severity distributions, the national severity distributions were assumed. Severity distributions for asthma were further adjusted in those aged 30 and over to reflect higher severities in older age groups. These adjustments were based on hospitalisations data.

Indigenous prevalence estimates and severity distributions for chronic obstructive pulmonary disease for 2018, 2011 and 2003 were based on the results of a cross-sectional BOLD study of the Indigenous population conducted in the Kimberley region of Western Australia (Cooksley 2013) and using age-sex distributions from hospitalisations data. As hospitalisations and survey data indicate that Indigenous Australians experience chronic obstructive pulmonary disease at younger age groups, estimates were adjusted to include prevalence in ages less than 40 years.

Sarcoidosis and intestinal lung disease are very rare in the Indigenous population (MacGinley & Allen 1997). Indigenous prevalence estimates for these conditions in 2018, 2011 and 2003 were based on hospitalisations and mortality data adjusted for under-identification using standard adjustment factors.

Prevalence estimates for asbestosis, silicosis and other pneumoconiosis (including coal worker's lung) were also based on hospitalisations and mortality data adjusted for under-identification using standard adjustment factors.

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Skin disorders

Sequelae and health states

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

Table 4.41: Sequelae, health states and durations for skin conditions

Disease	Sequela	ABDS 2018 health state identifier[(a)]	Duration
Dermatitis and eczema	Eczema	204, 205, 262	12 months
Psoriasis	Psoriasis	204, 205, 262	12 months
Acne	Acne	201, 202, 262	12 months
Ulcers	Decubitus ulcer (pressure ulcer)	204, 205, 206, 262	Various, depending on stage of ulcer
	Other chronic skin ulcer	39	12 months
Skin infections (including cellulitis)	Severe skin infection	3	2 weeks
Scabies	Scabies	204, 205	4.5 months
Other skin disorders	Other skin disorder: acute	3	2 weeks
	Other skin disorder: chronic	202	12 months

(a) See ABDS 2018 health states.

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 modified based on the severity of atopic dermatitis reported by Plunkett et al. (1999). The modified severity distribution takes into account that severe atopic dermatitis is likely to be the only dermatitis or eczema condition that would correspond to the most severe health state.

The severity distribution in children was based on the study by Marks et al. (1999a), which investigated atopic eczema in Australian school students (aged 4–18). The study reported four severities (minimal, mild, moderate and severe). Based on expert advice, the minimal and mild groups were combined into the least severe health state. Moderate and severe severities were aligned to the other health states, using the same approach as outlined for adults (only severe atopic dermatitis corresponds to the most severe health state).

Psoriasis

Prevalence was based on the NHS 2017–18 self-reported psoriasis that had lasted, or was expected to last, at least 6 months. Age-and-sex specific prevalence rates were modelled using the NHS 2017–18 psoriasis counts and applied to the Australian estimated resident population to calculate the national prevalence. Although the NHS 2017–18 did not report on *Very remote* areas, prevalence estimates were modelled to account for these areas.

The severity distribution of psoriasis was based on results from a study of 1,700 adults from several European countries who self-reported having had a diagnosis of psoriasis. Severity of psoriasis was assessed using the Dermatological Life Quality Index (Puig et al. 2017). Participants who were classified as ‘no impact’ in the study were considered to have minimal psoriasis. Participants who were classified as

'small' or 'moderate' impact were considered to have mild psoriasis. Participants who were classified as 'very large' or 'extremely large' impact were considered to have moderate/severe psoriasis. Proportions of the number of people in each severity were 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 for adults was based on proportions that were calculated using scores from the Dermatology Life Quality Index (Marks et al. 1999b) and applied to the 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 those aged 5–19 years. For younger children (0–4 years), the prevalence of acne was assumed to be zero (0). The severity distribution for children was based on proportions calculated using scores from the Acne Disability Index (Marks et al. 1999b) and applied to the estimates.

Ulcers

Pressure ulcers

There are 3 main settings where people are 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 settings based on different data sources (see table below). These figures were added together to produce the total prevalence of pressure ulcers in Australia.

Table 4.42: Summary of data sources for modelling the prevalence of pressure ulcers by setting, 2018

Setting	Prevalence	Age distribution	Severity	Duration (if required)
Hospitals	NSW 2018 Pressure Injury Point Prevalence Survey (CEC 2019) Qld 2018 Bedside Audit (Qld Health 2019) WA 2014 PI Point Prevalence Survey (Ferguson et al. 2019)	NHMD 2018	NHMD 2018	Dealey et al. 2012
Residential aged care	NSW 2018 Pressure Injury Point Prevalence Survey (CEC 2019)	Santamaria et al. 2009	GEN Aged care data 2019 (AIHW 2020)	No duration required
Home-based care	NSW 2018 Pressure Injury Point Prevalence Survey (CEC 2019)	Asimus & Li 2011	Asimus & Li 2011	No duration required

Pressure ulcers in the hospital: The prevalence of pressure ulcers acquired in hospitals was based on the proportions of hospital-acquired pressure ulcers from 3 jurisdictions. The proportions of pressure ulcers in New South Wales (CEC 2019), Queensland (Queensland Health 2019) and Western Australia (Ferguson et al. 2019) were applied to the number of hospitalisations (from the NHMD) in these states in 2018 and extrapolated to the remaining states/territories. This estimated the total national prevalence of pressure ulcers in the hospital setting. The age-sex distribution and the severity distribution were both obtained from NHMD hospitalisations data for pressure ulcers (ICD-10-AM: L89) in 2018.

Durations for each stage were based on the mean expected time to heal from ulcers, as reported by Dealey et al. (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 age care: The national prevalence of pressure ulcers in residential aged care was based on the proportion of pressure ulcers acquired while in NSW residential aged care facilities in 2018 (CEC 2019). This proportion was applied to the residential aged care population in Australia as at 30 June 2018 (AIHW 2019). The age distribution was modelled based on the findings of Santamaria and others (2009). The severity distribution was based on Residential Aged Care Quality Indicator data pertaining to the July–September 2019 period (AIHW 2020).

Pressure ulcers in home-based care: The national prevalence of pressure ulcers in home-based care was based on the proportion of pressure ulcers acquired while in NSW community and outpatient facilities in 2018 (CEC 2019). This proportion was applied to the home care population in Australia as at 30 June 2018 (AIHW 2019). The age distribution was modelled using data from an Australian study of patients receiving care from community nurses (Asimus & Li 2011). The severity distribution was also obtained from this study (Asimus & Li 2011).

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 et al. (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 2018. Separations with a principal diagnosis for skin infections (ICD-10-AM: A46, B08.1, B08.4, 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.

Scabies

Two data sources were used in the estimation of scabies prevalence—the NHMD and a study that measured scabies prevalence before and after two ivermectin mass drug administrations in a remote Australian Aboriginal island community (Kearns et al. 2015). Hospitalisations data were inflated to account for non-hospitalised cases in the community with a duration of 4.5 months applied to each case. Prevalence estimates derived using data from Kearns et al. (2015) were added to the hospitalisation estimates to account for a higher prevalence of scabies in *Very remote* areas.

Other skin disorders

The prevalence of other acute skin disorders was based on hospital separations (from the NHMD) in 2018. Separations with a principal diagnosis of other acute skin disorders (ICD-10-AM: L05, L10–L13, L28–L29, L41–L45, L50–60, L62–L68, L71–L75, L80–L88, L90–L95, L98.0–L98.3, L98.5–L98.9 and L99) were included. A duration of
2 weeks out of 1 year was applied to the separations to estimate the point prevalence.

Age-and-sex specific prevalence rates for other chronic skin disorders were modelled using the NHS 2017–18 counts 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 2018. The rates were applied to the Australian estimated resident population to obtain the national prevalence of other chronic skin disorders.

Sub-national estimates

For dermatitis & eczema, acne and skin ulcers, the proportions of the 2018 Australian estimated resident population in each state/territory, remoteness area and socioeconomic group were applied to the national estimates in order 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 in 2018 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 2017–18 counts and applied to the national estimates to produce the sub-national point prevalence.

For scabies, national prevalence was split into state/territory and socioeconomic sub-national estimates based on observed splits in hospitalisations data. For scabies remoteness sub-national estimates, national scabies prevalence based on hospitalisations was split based on observed splits in hospitalisations data and all of the Kearns et al. estimates were assigned to *Very remote* areas.

2015, 2011 and 2003 estimates

Where available and appropriate, the data sources used for the 2003, 2011 and 2015 estimates were the same as those used for the 2018 estimates.

For dermatitis & eczema and acne, the same prevalence rates (sourced from the same studies) were applied to the 2003, 2011 and 2015 populations. For skin infections and other acute skin disorders, 2003, 2011 and 2015 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, AHS 2011–12 and NHS 2014–15 and used for the 2003, 2011 and 2015 estimates respectively.

For pressure ulcers, the methods of estimating prevalence for 2003, 2011 and 2015 were largely the same as the methods used for the 2018 reference year, however, different data sources were used (see below). The prevalence of pressure ulcers was not always available for the specific reference year, in which case the closest year was used.

There was a notable change in the methods used to estimate prevalence in the ABDS 2018 compared to in the ABDS 2015 and this was updated for all reference years. This change was to use the prevalence of pressure ulcers acquired within each setting (hospital, residential aged care, home care) instead of using the overall prevalence of pressure ulcers reported for each setting. This change was made to avoid double counting of pressure ulcers which may be present within one setting, but were acquired in another. Population counts for residential aged care and home-based care were sourced from AIHW GEN Aged care data for each reference year.

Table 4.43: Summary of data sources for modelling the prevalence of pressure ulcers by setting, 2015, 2011 and 2003

Setting	Prevalence	Age distribution	Severity	Duration (if required)

For other chronic skin ulcers, the same method based on the study by Harrison et al. (2013) was used to estimate the prevalence. BEACH data from 2003 was used for the 2003 estimates, whereas BEACH data from 2011 was used for the 2011 and 2015 estimates.

Hospitals 2015	NSW 2016 Pressure Injury Point Prevalence Survey (CEC 2017) Qld 2015 Bedside Audit (ASCQHC 2018) WA 2014 Pressure Injury Point Prevalence Survey (Ferguson et al. 2019)	NHMD 2015	NHMD 2015	Dealey et al. 2012
Residential aged care 2015	NSW 2016 Pressure Injury Point Prevalence Survey (CEC 2017)	Santamaria et al. 2009	GEN Aged care data 2019 (AIHW 2020)	No duration required
Home-based care 2015	NSW 2016 Pressure Injury Point Prevalence Survey (CEC 2017)	Asimus & Li 2011	Asimus & Li 2011	No duration required
Hospitals 2011	Mulligan et al. 2011 (includes data for WA and NSW) Qld 2011 Bedside Audit (Qld Health 2012)	NHMD 2011	Pressure ulcer point prevalence surveys (SA Health 2007, VQC 2006, Mulligan et al. 2011)	Dealey et al. 2012
Residential aged care 2011	Santamaria et al. 2009 (adjusted to exclude ulcers acquired elsewhere)	Santamaria et al. 2009	GEN Aged care data 2019 (AIHW 2020)	No duration required
Home-based care 2011	Asimus & Li 2011 (adjusted to exclude hospital-acquired ulcers)	Asimus & Li 2011	Asimus & Li 2011	No duration required
Hospitals 2003	Mulligan et al. 2011 (includes data for Qld, WA, NSW, Vic and SA)	NHMD 2003	Pressure ulcer point prevalence surveys (SA Health 2007, VQC 2006, Mulligan et al. 2011)	Dealey et al. 2012
Residential aged care 2003	Santamaria et al. 2009 (adjusted to exclude ulcers acquired elsewhere)	Santamaria et al. 2009	GEN Aged care data 2019 (AIHW 2020)	No duration required
Home-based care 2003	Asimus & Li 2011 (adjusted to exclude hospital-acquired ulcers)	Asimus & Li 2011	Asimus & Li 2011	No duration required

For scabies, inflated hospitalisations (from NHMD) from the relevant reference year were used for the 2003, 2011 and 2015 estimates with the addition of estimates derived from rates from Kearns et al. study (2015). Estimates derived from Kearns et al. (2015) were based on rates applied to the Indigenous population for *Very remote* areas for each year.

Indigenous specific estimates

Due to a lack of available data on Indigenous prevalence, the national prevalence rates were applied to the Indigenous population to produce Indigenous prevalence estimates of dermatitis & eczema and acne, for 2003, 2011 and 2018.

For psoriasis, Indigenous estimates were obtained using the AATSIHS 2018–19 for 2018 estimates, the AATSIHS 2012–13 for 2011 estimates and the NATSIHS 2004–05 for 2003 estimates.

Prevalence for skin infections for the Indigenous population in 2018, 2011 and 2003 were estimated using the NHMD and adjusted for Indigenous under-identification using adjustment factors (see Years lived with disability (YLD)).

For ulcers, hospitalisation rate ratios (Indigenous: national) based on Indigenous hospitalisations adjusted for under-identification using standard adjustment factors, were applied to the national prevalence rates.

To estimate the prevalence of other skin disorders in the Indigenous population, the same methods were used as for national estimates using the AATSIHS 2018–19, AATSIHS 2012–13 and NATSIHS 2004–05.

Indigenous prevalence for scabies were estimated by summing estimates from the NHMD adjusted for Indigenous under-identification using adjustment factors (see Years lived with disability (YLD)) and estimates based on Kearns et al. (2015) for *Very Remote* areas.

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Disease and risk factor specific models and methods

Risk factor specific methods

This page describes in detail the methods unique to each risk factor included in the ABDS 2018. 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 2018, in particular, dietary risks.

Overarching methods and choices for risk factors 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 TMREDs (see risk factor specific methods).

The linked diseases and relative risks were sourced from the GBD 2019 or an AIHW review of the literature as described here and in Overarching methods and choices for risk factors. Most TMREDs were also sourced from the GBD 2019, with the exceptions described in the Risk factor-specific methods.

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 (including 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 2019 (GBD 2019 Risk Factor Collaborators 2020). 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 & problems linked to occupational risks, only non-fatal burden has been estimated.

A supplementary table contains detailed definitions, data sources and linked diseases for all risk factors (Table S4.2).

Behavioural risk factors

Dietary risk factors

The burden attributable to dietary risk factors was estimated in people aged 25 and over.

It should be noted that the methods, including the TMREDs, used in the ABDS 2018 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 Dietary risk model parameters in the table below). For information on recommended food choices, see the Australian Dietary Guidelines (NHMRC 2015).

A literature review of the list of dietary risk factors and the methods used in ABDS 2015 was undertaken as part of this study to identify any additional dietary risk factors or update existing methods and linked diseases. The review looked into updates on reviews undertaken to inform dietary guidelines in Australia or internationally, published meta-analyses on dietary risk factors and associated linked diseases, and studies that specifically estimate relative risks of outcomes such as the World Cancer Research Fund International (WCRFI) continuous update project for linked cancers and the GBD 2019 (GBD 2019 Risk Factors Collaborators 2020).

The risk factors included were based on the AIHW review of evidence from the GBD 2019 (which included 15 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.

Due to methodological differences, methods for diet high in sodium are discussed in a separate sub-section.

Dietary risks included

The same dietary risk factors were included as in the ABDS 2015.

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 et al. (2012).

Population attributable fractions estimated using comparative risk assessment

The risk factors estimated using the comparative risk assessment were diet low in fruit, vegetables, wholegrains, legumes, nuts and seeds, milk, fish and seafood, and polyunsaturated fats; and diet high in red meat, processed meat and sugar-sweetened beverages.

The models for the risk factors in GBD 2019 changed to have different levels of exposure which are not associated with increased risk (TMRED). The updated TMRED are listed in Dietary risk model parameters above and indicate for some risk factors levels of consumption above which there is a conferred protective impact against linked diseases. In the ABDS 2015, TMREDs were made up of a range of exposure categories (e.g. 200 to 300g of fruit); however, now the increased risk has been grouped into categories where it is only possible to include a single cut off value for the TMRED (e.g. 300g of fruit).

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. As with 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 Dietary risk model parameters in the Australian population.

To estimate consumption in 2018, unit record level data from the AHS 2011–12 was adjusted by the percentage change from 2011 to 2018, 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 (2017).

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 2014). 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 2018, except for diet high in sodium.

Table 4.44: Dietary risk model parameters

Risk factor	Diet low in fruit – Average daily consumption of fresh, frozen, cooked, canned, or dried fruits (excluding fruit juices)
Disease outcome	Coronary heart disease, lung cancer, oesophageal cancer, stroke, type 2 diabetes
TMRED	Consumption of at least 300 g of fruit per day
National data source	Self-reported from AHS 2011–12
Units for effect size calculation	Per 100 g per day intake decrease
Risk factor	Diet low in legumes – Average daily consumption of fresh, frozen, cooked, canned, or dried legumes
Disease outcome	Coronary heart disease
TMRED	Consumption of at least 150 g of legumes per day
National data source	Self-reported from AHS 2011–12
Units for effect size calculation	Per 50 g per day intake decrease
Risk factor	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
Disease outcome	Bowel cancer
TMRED	Consumption of at least 240 g of milk per day

National data source	Self-reported from AHS 2011–12
Units for effect size calculation	Per 60 g per day intake decrease
Risk factor	Diet low in nuts and seeds – Average daily consumption of nut and seed foods
Disease outcome	Coronary heart disease, type 2 diabetes
TMRED	Consumption of at least 14 g of nuts and seeds per day
National data source	Self-reported from AHS 2011–12
Units for effect size calculation	Per 7 g per day intake decrease
Risk factor	Diet low in polyunsaturated fats – Average daily consumption of polyunsaturated fats
Disease outcome	Coronary heart disease
TMRED	Consumption of polyunsaturated fatty acids at least 8% of total daily energy
National data source	Self-reported from AHS 2011–12
Units for effect size calculation	Per 2% energy from polyunsaturated fat decrease
Risk factor	Diet high in processed meats – Average daily consumption of meat preserved by smoking, curing, salting, or addition of chemical preservatives
Disease outcome	Bowel cancer, coronary heart disease, type 2 diabetes
TMRED	Consumption of less than 24 g of processed meat per day
National data source	Self-reported from AHS 2011–12
Units for effect size calculation	Per 25 g per day intake increase
Risk factor	Diet high in red meat – Average daily consumption of red meat (beef, pork, lamb, and goat) (excluding poultry, fish, eggs and all processed meats)
Disease outcome	Bowel cancer, breast cancer, coronary heart disease, stroke, type 2 diabetes
TMRED	Consumption of less than 50 g of red meat per day
National data source	Self-reported from AHS 2011–12
Units for effect size calculation	Per 50 g per day intake increase
Risk factor	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)
Disease outcome	Coronary heart disease, oesophageal cancer, stroke
TMRED	Consumption of at least 300 g of vegetables per day
National data source	Self-reported from AHS 2011–12
Units for effect size calculation	Per 100 g per day of vegetable intake decrease
Risk factor	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
Disease outcome	Bowel cancer, coronary heart disease, stroke, type 2 diabetes
TMRED	Consumption of at least 150 g of wholegrains per day

National data source	Self-reported from AHS 2011–12
Units for effect size calculation	Per 50 g per day intake decrease
Risk factor	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)
Disease outcome	Coronary heart disease, type 2 diabetes
TMRED	Consumption of less than 60 g of sugar-sweetened beverages per day
National data source	Self-reported from AHS 2011–12
Units for effect size calculation	Per 60 g per day intake increase
Risk factor	Diet low in fish and seafood - Average daily consumption of fish and seafood
Disease outcome	Coronary heart disease
TMRED	Consumption of fish or seafood 100 g per week
National data source	Self-reported from AHS 2011–12 (day 1 only)
Units for effect size calculation	Per 15g per day intake decrease

Estimates by socioeconomic group

Exposure to dietary risks was estimated from the AHS 2011–12, modelled to 2018, and the difference in the mean estimate in each socioeconomic group quintile as described in Overarching methods and choices for risk factors.

2015, 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 and 2015 in these data sources to estimate the distribution of dietary intake in those reference years. This method is the same as was used for the 2018 study.

Indigenous specific estimates

Exposure estimates for each of the 13 dietary risk factors for the Indigenous population was estimated from the AATSIHS 2012–13 using the same methods as used for national estimates. For example, day 1 dietary recall data were used for the micronutrients. For whole foods, the AHS whole food database was used to estimate the average proportion of the whole foods from within each food classified in the AATSIHS 2012–13. Exposure for 2003 and 2018 were estimated by assuming the same trends as in the national population.

Dietary risks mediated through other risk factors - Diet high in sodium

Diet high in sodium was measured by the amount it mediated blood pressure. The methods for this risk factor use comparative risk assessment and are based on the GBD 2019.

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 2014). 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 2018, the distribution of blood pressure prevalence from the NHS 2017–18 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.

Table 4.45: Diet high in sodium risk model parameters

Risk factor	Diet high in sodium – Consumption of sodium
Disease outcome	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, non-rheumatic valvular disease, peripheral vascular disease, rheumatic heart disease, stroke
TMRED	24 hr urinary sodium of 2 g per day
National data source	Self-reported from AHS 2011–12; adjusted based on urinary sodium estimate (Powles et al. 2013)
Units for effect size calculation	Per 2.3g per day intake increase

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 2017–18.

2015, 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 and 2014–15 to calculate 2003 and 2015 estimates, respectively.

Indigenous specific estimates

The average adjustment factors estimated for sodium intake and blood pressure for 2011 and 2018 were applied to the distribution of blood pressure prevalence in the NATSIHS 2004–05 and NATSIHS 2018–19 to calculate 2003 and 2018 respectively.

References

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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 estimated directly for chronic liver disease, hepatitis B, hepatitis C, HIV/AIDS, and liver cancer from the National Notifiable Diseases Surveillance Scheme data published in annual surveillance reports by The Kirby Institute (The Kirby Institute 2018).

Acute hepatitis B and C

For acute hepatitis B and hepatitis C, the direct PAFs were calculated from estimated proportions of people with newly acquired hepatitis B or hepatitis C infections in 2018 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, 2015 and 2018).

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 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 estimated proportions of diagnosed AIDS cases in 2018 with a relevant exposure category (including homosexual contact only, homosexual contact and injecting drug use or heterosexual contact).

Table 4.46: Unsafe sex risk model parameters

Risk factor	Unsafe sex – Unsafe sex
Disease outcome	Cervical cancer, chlamydia, chronic liver disease, gonorrhoea, hepatitis B, hepatitis C, HIV/AIDS, liver cancer, syphilis, other sexually transmitted infections
TMRED	No unsafe sex
National data source	National notifiable disease annual surveillance reports (The Kirby Institute)
Units for effect size calculation	All sexually transmitted infections and cervical cancer attributed to unsafe sex HIV/AIDS, hepatitis B and hepatitis C from direct evidence

2015, 2011 and 2003 estimates

Methods for estimating exposure and calculating the PAFs in 2018 were used to produce 2015, 2011 and 2003 estimates. Data from the NNDSS published in the annual surveillance reports by The Kirby Institute were used to calculate PAFs for unsafe sex (The Kirby Institute 2004, 2012, 2013, 2016).

Indigenous specific estimates

For Indigenous estimates for unsafe sex, the same methods and exposure data sources were used as for national estimates. The quality of Indigenous data in the NNDSS varies by disease and state/territory, and is described in the annual surveillance reports published by the Kirby Institute.

References

MacLachlan JH, Allard N, Towell V & Cowie BC 2013. The burden of chronic hepatitis B virus infection in Australia, 2011. Australian and New Zealand Journal of Public Health 37(5):416–22.

O'Sullivan BG, Gidding HF, Law M, Kaldor JM, Gilbert GL & Dore GJ 2004. Estimates of chronic hepatitis B virus infection in Australia, 2000. Australian and New Zealand Journal of Public Health 28(3):212–16.

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The Kirby Institute 2016. HIV, viral hepatitis and sexually transmissible infections in Australia: Annual surveillance report 2016. Sydney: The Kirby Institute, UNSW.

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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 2018.

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 are described further in the report *Burden of tobacco use in Australia: Australian Burden of Disease Study 2015* (AIHW 2019).

Exposure estimates

The NDSHS 2013 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 2013 who reported smoking daily, weekly or less than weekly.

The NDSHS 2019 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 2018 (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.

Table 4.47: Tobacco risk model parameters

Risk factor	Tobacco use – Second-hand smoke
Disease outcome	Breast cancer, coronary heart disease, influenza, lower respiratory infections, lung cancer, otitis media, stroke, type 2 diabetes
TMRED	No tobacco use
National data source	NDSHS 2019

Units for effect size calculation	Proportion of the population exposed to second-hand smoke

Risk factor	Tobacco use – Current smoking (5-year lagged)
Disease outcome	Age-related macular degeneration, aortic aneurysm, asthma, atrial fibrillation & flutter, back pain & 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
TMRED	No tobacco use
National data source	NDSHS 2013
Units for effect size calculation	Proportion of the population who smoked 5 years ago

Risk factor	Tobacco use – Smoking impact ratio
Disease outcome	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 & oral cavity cancer, liver cancer, lung cancer, nasopharynx cancer, oesophageal cancer, other leukaemias, other respiratory diseases, pancreatic cancer, prostate cancer, stomach cancer
TMRED	No tobacco use
National data source	NMD
Units for effect size calculation	Lung cancer mortality rate; Peto et al. 1992

Estimates by socioeconomic group

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

2015, 2011 and 2003 estimates

The NDSHS 2010 was used to estimate the proportion of the population who are current (5-year lagged) smokers for 2015. The NDSHS 2016 was used to estimate the proportion of non-smokers exposed to second-hand smoke. The NMD 2015 was used to estimate lung cancer mortality.

National exposure estimates for 2011 and 2003 were calculated from the earlier iterations of the same surveys used for the 2015 estimates and followed the same method.

Indigenous specific estimates

The same general methods were used to estimate exposure to tobacco use in the Indigenous population. However, there were some differences in the data sources used to estimate the proportion of the Indigenous population who are current and former smokers, as well as the proportion of non-smokers exposed to environmental tobacco in the home.

Due to the small Aboriginal and Torres Strait Islander sample in the NDSHS (about 460 respondents in 2010), estimates of tobacco exposure were not considered reliable when broken down by age, sex and smoking status. Instead, the National Aboriginal and Torres Strait Islander Social Surveys (NATSISS) (2002 and 2008) were used for 2003 and 2011 estimates, respectively, and the Australian Aboriginal and Torres Strait Islander Health Survey (AATSIHS) 2012–13 was used for 2018 estimates. While the earlier two survey dates do not directly align with the 5-year lagged smoking prevalence used for national estimates, analysis of Indigenous smoking rates in consecutive ABS Indigenous health and social surveys (2001, 2002, 2004–05, 2008, 2011–12) showed no discernible trends up to the AATSIHS 2011–12. Therefore, the choice of the 2002 and 2008 NATSISS surveys is likely to have had little impact on the proportion of the population exposed.

Similar to national estimates, a smoking impact ratio was used as an indirect method to estimate the accumulated risk from tobacco smoking for cancers and respiratory diseases.

References

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Peto R, Boreham J, Lopez AD, Thun M & Heath C 1992. Mortality from tobacco in developed countries: indirect estimation from national vital statistics. The Lancet 339(8804):1268–78.

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 2018).

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 2018).

HIV/AIDS

For HIV/AIDS, direct PAFs were calculated from the estimated proportion of diagnosed AIDS cases in 2018 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 estimated proportion of newly acquired hepatitis B or hepatitis C infections in 2018 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 are published in the annual surveillance reports by The Kirby Institute. These were multiplied by the earliest year 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 2018.

Chronic hepatitis B infection

The Kirby Institute reported that 5.7% of people living with chronic hepatitis B in 2017 and 2015 had acquired this condition through unsafe injecting practices (The Kirby Institute 2016, 2018). 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 2018.

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 2018 in the NMD. These methods are described in more detail in the section on alcohol use. 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 2018. 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 (as this question was not available in the 2019 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.

Table 4.48: Illicit drug use risk model parameters

Risk factor	Cannabis use – Cannabis dependence
Disease outcome	Anxiety disorders, depressive disorders, schizophrenia

TMRED	No illicit drug use
National data source	ABDS 2018
Units for effect size calculation	Prevalence of illicit drug use disorders

Risk factor	Cannabis use – Driving under the influence of cannabis
Disease outcome	Road traffic injuries—motorcyclists and road traffic injuries—motor vehicle occupants
TMRED	No illicit drug use
National data source	NDSHS 2016
Units for effect size calculation	Prevalence of driving under the influence of illicit drugs

Risk factor	Cannabis use – Cannabis use and dependence
Disease outcome	Accidental poisoning
TMRED	No illicit drug use
National data source	NMD
Units for effect size calculation	Direct evidence

Risk factor	Amphetamine, cocaine and opioid use – Amphetamine, cocaine and opioid use or dependence
Disease outcome	Suicide & self-inflicted injuries
TMRED	No illicit drug use
National data source	ABDS 2018
Units for effect size calculation	Prevalence of illicit drug use disorders

Risk factor	Amphetamine, cocaine and opioid use – Driving under the influence of amphetamine, cocaine or opioids
Disease outcome	Road traffic injuries—motorcyclists and road traffic injuries—motor vehicle occupants
TMRED	No illicit drug use
National data source	NDSHS 2016
Units for effect size calculation	Prevalence of driving under the influence of illicit drugs

Risk factor	Amphetamine, cocaine and opioid use – Amphetamine or opioid use and dependence
Disease outcome	Accidental poisoning
TMRED	No illicit drug use
National data source	NMD
Units for effect size calculation	Direct evidence

Risk factor	Amphetamine, cannabis cocaine, opioid and other illicit drug use – Illicit drug dependence

Disease outcome	Drug use disorders (excluding alcohol)
TMRED	No illicit drug use
National data source	ABDS 2018
Units for effect size calculation	Direct evidence

Risk factor	Unsafe injecting practices – Unsafe injecting practices
Disease outcome	Chronic liver disease, hepatitis B, hepatitis C, HIV/AIDS, liver cancer
TMRED	No unsafe injecting practices
National data source	National notifiable disease annual surveillance reports (The Kirby Institute)
Units for effect size calculation	Direct evidence

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.

2015, 2011 and 2003 estimates

The burden attributable to illicit drug use in 2015 was estimated using the NDSHS 2016 and The Kirby Institute data 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 2018), using the same methods as for 2018.

The burden attributable to illicit drug use in 2011 and 2003 was estimated using the NDSHS 2010 and 2004 and The Kirby Institute data for 2011 and 2003, respectively, using the same methods as for 2018.

Indigenous specific estimates

For Indigenous risk factor estimates for drug use, the same data sources and methods were used as for national estimates. The quality of Indigenous data in the NNDSS varies by disease and state/territory, and is described in the annual surveillance reports published by the Kirby Institute. For drug driving data, due to a lack of suitable data for Indigenous Australians, the prevalence of drug driving was assumed to be the same as for the general population.

References

AIHW 2018. Impact of alcohol and illicit drug use on the burden of disease and injury in Australia: Australian Burden of Disease Study 2011. Cat. no. BOD 19. Canberra: AIHW.

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Alcohol use

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 2018). 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 2018, 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 2019. The same method was used to estimate the PAFs for liver cancer. The burden of *alcohol dependence* (the linked disease) was entirely 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 2018 as described by the AIHW (2018).

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 2019. 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 2019). In the ABDS 2018, self-reported daily consumption from the NDSHS, by age and sex, was inflated to match alcohol sales data in each reference year, based on the methods described by Rehm et al. (2010).

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 2018, the inflation factor was estimated to be 1.46.

Following methods used in Rehm et al. (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.

Table 4.49: Alcohol use risk model parameters

Risk factor	Alcohol use – former drinkers
Disease outcome	Atrial fibrillation & flutter, bowel cancer, breast cancer, coronary heart disease, epilepsy, hypertensive heart disease, laryngeal cancer, lower respiratory infections, lip & oral cavity cancer, nasopharynx cancer, oesophageal cancer, other oral cavity & pharynx cancers, pancreatitis, stroke
TMRED	No alcohol use
National data source	NDSHS 2019
Units for effect size calculation	Former drinker

Risk factor	Alcohol use – Average daily alcohol consumption by current drinkers
Disease outcome	Atrial fibrillation & flutter, bowel cancer, breast cancer, coronary heart disease, drowning, epilepsy, falls, fire, burns and scalds, homicide and violence, hypertensive heart disease, laryngeal cancer, lip & oral cavity cancer, lower respiratory infections, nasopharynx cancer, oesophageal cancer, other land transport injuries, other oral cavity & pharynx cancers, other unintentional injuries, pancreatitis, road traffic injuries (RTI)—motor vehicle occupants, RTI—motorcyclists, RTI—pedal cyclists, RTI—pedestrians, stroke
TMRED	No alcohol use
National data source	NDSHS 2019 apparent consumption of alcohol data;
Units for effect size calculation	Average consumption of pure alcohol (g per day)

Risk factor exposure	Alcohol use – Alcohol use and dependence
Disease outcome	Alcohol use disorders, accidental poisoning, liver cancer, chronic liver disease
TMRED	No alcohol use

National data source	NMD; GBD 2019
Units for effect size calculation	Direct evidence

Risk factor	Alcohol use – Alcohol dependence
Disease outcome	Suicide & self-inflicted injuries
TMRED	No alcohol use
National data source	ABDS 2018
Units for effect size calculation	Prevalence alcohol use disorders

Estimates by socioeconomic group

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

2015, 2011 and 2003 estimates

Exposure estimates for 2015 were calculated using data from the NDSHS 2016 and alcohol sales data for 2015, while exposure for 2011 and 2003 were calculated using data from the NDSHS 2010 and 2004 and alcohol sales data for 2011 and 2003, respectively. These followed the method for estimating exposure used for 2018. Direct PAFs were calculated using the method for 2018, which were based on the GBD 2019 estimates for 2015, 2011 and 2003.

Indigenous-specific estimates

Due to the small Aboriginal and Torres Strait Islander sample in the NDSHS (about 460 respondents in 2010), estimates were not considered reliable when broken down by age, sex and amount of alcohol consumed. Instead, the NATSIHS 2004–05, AATSIHS 2012–13 and NATSIHS 2018–19 were used. As alcohol excise, sales and import figures published by the ABS represent a single national figure, it is not possible to calculate Indigenous-specific factors to correct for under-reporting. Therefore, national factors were applied to Indigenous estimates from the NATSIHS and AATSIHS.

References

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Rehm J, Kehoe T, Gmel G, Stinson F, Grant B & Gmel G 2010. Statistical modelling of volume of alcohol exposure for epidemiological studies of population health: the US example. Population Health Metrics 8(3):1–12.

Physical inactivity

Burden due to physical inactivity was estimated in people aged 20 and over.

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:

$$MET = T \; x \; I$$

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:

- Fewer than 600 METs per week

- 600–1,199 METs per week
- 1,200–1,799 METs per week
- 1,800–2,399 METs per week
- 2,400–2,999 METs per week
- 3,000–3,599 METs per week
- 3,600–4,199 METs per week
- 4,200 METs and over per week.

These categories align with relative risks provided by the GBD 2019 and were used in the ABDS 2018.

The METs for leisure, walking for transport and occupational activity were estimated from the trend in METs reported in successive health surveys, including the NHS 2001, the NHS 2004–05, the NHS 2007–08, the AHS 2011–12, the NHS 2014–15 and the NHS 2017–18. The change over time was used to adjust estimates of exercise in the NHS 2017–18 to represent all other reference years. 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 2017–18 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 all reference years (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.

Average time spent gardening and strengthening and toning were estimated by age and sex using the National Nutrition and Physical Activity Survey (NNPAS) as part of the AHS 2011–12. A trend for the proportion of individuals doing at least some strengthening and toning was estimated using successive health surverys (AHS 2011–12, the NHS 2014–15 and the NHS 2017–18). This trend was used to randomly allocate time spent strengthening and toning to individuals who responded having said they completed at least one day of the activity in the past week. As informing a trend wasn't possible for gardening (due to data limitations between surveys), proportions of those doing any gardening from the NNPAS in 2011 were applied to all reference years with average time spent gardening then being randomly allocated by age and sex.

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

Table 4.50: Physical inactivity risk model parameters

Risk factor	Physical inactivity – Metabolic equivalent of task (METs)
Disease outcome	Breast cancer, bowel cancer, coronary heart disease, dementia, type 2 diabetes, stroke, uterine cancer
TMRED	All adults experience average 4200 metabolic equivalent of task (METs) per week (highly physically active)
National data source	AHS 2011–12; NHS 2017–18
Units for effect size calculation	METs of less than 600, 600–1,999, 1,200–1,799, 1,800–2,399, 2,400–2,999, 3,000–3,599, 3,600–4,199

Estimates by socioeconomic group

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

2015, 2011 and 2003 estimates

The number of total METs in 2015, 2011 and 2003 was estimated using the same trend analyses used to estimate METs in 2018. The NHS 2017–18 data were adjusted based on this trend to represent these METs in earlier reference years. Average METs for household chores were the same as in 2018 as no further data were available.

Indigenous specific estimates

Exposure estimates of physical inactivity for the Indigenous population was estimated from the NATSIHS 2018–19 (for 2018 estimates), AATSIHS 2012–13 (for 2011 estimates) and the NATSIHS 2004–05 (for 2003 estimates). It was not possible to adjust these estimates to include stretching and gardening, as this information was not available from the relevant surveys used.

References

ABS 2008. How Australians use their time, 2006. ABS cat. no. 4153.0 Canberra: ABS. Viewed 2 July 2017.

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 not available for men (AIHW unpublished, Ayre et al. 2016; GBD 2019 Risk Factor Collaborators 2020).

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 (NHMP) for fatal burden, which estimated that 58% of homicides in females were due to an intimate partner in 2018.

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 2017). 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 2017).

Table 4.51: Intimate partner violence risk model parameters

Risk factor	Intimate partner violence – Physical, sexual, emotional abuse from a cohabitating partner
Disease outcome	Anxiety disorders, alcohol use disorders, early pregnancy loss, depressive disorders, homicide and violence, suicide and self-inflicted injuries
TMRED	No exposure to intimate partner violence
National data source	ABS Personal Safety Survey 2016; National Homicide Monitoring Program
Units for effect size calculation	Ever been exposed to intimate partner violence since the age of 15 years (prevalence)

Estimates by socioeconomic group

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

2015, 2011 and 2003 estimates

The burden due to intimate partner violence in 2015 was estimated using data also from the PSS 2016 (ABS 2017), NHMD hospitalisations in 2015 and the NHMP 2012–2014 (Bryant & Bricknell 2017).

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

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 (ABS 2006).

Indigenous specific estimates

For fatal burden due to homicide and violence, direct evidence for Indigenous women was used from the National Homicide Monitoring Program. In 2010–2012, 65% of Indigenous female homicides were classified as perpetrated by an intimate partner (Bryant & Cussen 2015); while for 2003, this was assumed to be 59% based on estimates from 2006–07 (Dearden & Jones 2008).

For the remaining burden, the ABS Personal Safety Survey 2012 did not include an Indigenous identifier, so indirect methods were used to estimate Indigenous exposure to intimate partner violence. A rate ratio of 3.1 was applied to national exposure estimates (AIHW & NIAA 2020). This rate ratio is based on age-standardised rates for 12-month prevalence of physical or threatened violence victimisation reported by females aged 15 years and over, from the 2014 General Social Survey (for national estimates) and the 2014–15 NATSISS (for Indigenous estimates). The same rate ratio was applied to the national exposure estimates to derive Indigenous exposure for both 2003 and 2011.

References

ABS 2006. Personal safety, Australia, 2005 (reissue). ABS cat. no. 4906.0. Canberra: ABS. Viewed 22 March 2018.

ABS 2013. Personal safety, Australia, 2012. ABS cat. no. 4906.0. ABS: Canberra. Viewed 15 August 2015.

ABS 2017. Personal Safety Survey, Australia: user guide, 2016. ABS cat. no. 4906.0.55.003. Canberra: ABS. Viewed 15 March 2018.

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Ayre J, Lum On M, Webster K & Moon L 2016. Examination of the burden of disease of intimate partner violence against women in 2011: final report. Sydney: Australian National Research Organisation for Women's Safety.

Bryant W & Bricknell S 2017. Homicide in Australia, 2012–13 to 2013–14: National Homicide Monitoring Program report. Statistical report no. 02. Canberra: Australian Institute of Criminology. Viewed 19 September 2018.

Bryant W & Cussen T 2015. Homicide in Australia, 2010–11 to 2011–12: National Homicide Monitoring Program report. Monitoring report no. 23. Canberra: Australian Institute of Criminology. Viewed 22 June 2016.

Dearden J and Jones W 2008. Homicide in Australia: 2006-07 National Homicide Monitoring Program annual report (monitoring reports no. 1). Canberra: Australian Institute of Criminology. Accessed 17 November 2020.

GBD 2019 Risk Factors Collaborators 2020. Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. The Lancet 396:1223–49.

Mouzos J 2005. Homicide in Australia: 2003–2004 National Homicide Monitoring Program (NHMP) annual report. Canberra: Australian Institute of Criminology. Viewed 23 August 2016.

Child abuse & neglect

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

Moore et al. (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 et al. (2015) also reviewed the literature and identified linked diseases and relative risks for child abuse & 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 et al. (2015) by age and sex. The same PAFs were applied to each reference year of this study: 2003, 2011, 2015 and 2018.

Table 4.52: Child abuse & neglect risk model parameters

Risk factor	Child abuse & neglect – Physical, sexual and emotional abuse and neglect
Disease outcome	Anxiety disorders, depressive disorders, suicide & self-inflicted injuries
TMRED	No child abuse and neglect
National data source	Moore et al. 2015
Units for effect size calculation	Prevalence of childhood abuse and neglect

Indigenous specific estimates

Child protection data on victims of sexual, emotional, physical and neglect abuse were used to estimate the relative difference in the prevalence of child abuse between the Indigenous and the total Australian populations, based on the Overcoming Indigenous Disadvantage 2020 tables (SCRGSP 2020). The ratio was based on nationwide data and applied to national prevalence estimates. PAFs were calculated.

References

Moore SE, Scott JG, Ferrari AJ, Mills R, Dunne MP, Erskine HE et al. 2015. Burden attributable to child maltreatment in Australia. Child Abuse & Neglect 48:208–20.

SCRGSP (Steering Committee for the Review of Government Service Provision) 2020. Overcoming Indigenous Disadvantage: Key Indicators 2020. Canberra: Productivity Commission.

Bullying victimisation

The burden for bullying victimisation was estimated in people aged 10 to 24. It was not possible to estimate burden by socioeconomic group as exposure data by socioeconomic quintile was not available.

Population attributable fraction estimated by comparative risk assessment

Prevalence and relative risks were taken from a recent systematic review and meta-analysis of bullying victimisation among Australian children and adolescents.

Jadambaa et al. (2019a) identified a number of studies estimating exposure of Australian children to bullying victimisation between 1991 and 2015. Various types of bullying were identified, including both traditional and cyberbullying. A meta-analysis was conducted estimating that 15% of Australian children and adolescents were exposed to bullying within the past 12 months. However, this estimate did not distinguish between the types of bullying with the authors noting a strong overlap between the two types, even with cyberbullying only more recently gaining prominence.

This prevalence was applied to relative risks estimated via another meta-analysis (Jadambaa et al 2019b) using the comparative risk assessment methodology to estimate PAFs for anxiety disorders and depressive disorders. The same PAFs were applied to each reference year of this study.

Table 4.53: Bullying victimisation risk model parameters

Risk factor	Bullying victimisation - Exposure to bullying within the past 12 months
Disease outcome	Anxiety disorders, depressive disorders
TMRED	No bullying victimisation
National data source	Jadambaa et al. 2019a, Jadambaa et al. 2019b
Units for effect size calculation	Prevalence of bullying victimisation

Indigenous specific estimates

This risk factor was not estimated for the Indigenous population due to a lack of suitable data to measure exposure. Investigations are underway as to how this measure may be included in future studies.

References

Jadambaa A, Thomas HJ, Scott JG, Graves N, Brain D & Pacella R 2019a. Prevalence of traditional bullying and cyberbullying among children and adolescents in Australia: A systematic review and meta-analysis. Australian & New Zealand Journal of Psychiatry 53:878–888.

Jadambaa A, Thomas HJ, Scott JG, Graves N, Brain D & Pacella R 2019b. The contribution of bullying victimisation to the burden of anxiety and depressive disorders in Australia. Epidemiology and Psychiatric Sciences 29:1–23.

Metabolic/biomedical risk factors

Overweight (including obesity)

The burden due to overweight (including 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 2017).

Population attributable fraction

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 2017–18. For children and adolescents aged 5–14, age- and sex-specific BMI cut-off levels indicating overweight (including obesity) were derived from the study by Cole et al. (2000).

Relative risks

The relative risks used were largely based on those published by the GBD 2019, including atrial fibrillation & flutter, cataract, non-Hodgkin lymphoma and multiple myeloma. Other relative risks were based on work by the AIHW (AIHW 2017). For dementia and gallbladder and bile duct disease, relative risks from the GBD 2019 were used instead of relative risks from the AIHW as they were based on a more recent meta-analysis.

Table 4.54: Overweight (including obesity) risk model parameters

Risk factor	Overweight, obese – Body mass index BMI
Disease outcome	Acute lymphoblastic leukaemia, acute myeloid leukaemia, asthma, atrial fibrillation & flutter, back pain & 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

TMRED	Body mass index between 20 and 25 BMI
National data source	NHS 2017–18
Units for effect size calculation	Per 5 BMI

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 Overarching methods and choices for risk factors.

2015, 2011 and 2003 estimates

Exposure for 2011 and 2015 were estimated as described above, using data from the AHS 2011–12 and NHS 2014–15, respectively.

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 2017.

For people aged 5–19, 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 5–19 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.

Indigenous specific estimates

Exposure for 2018 was estimated as described for the national estimates and was based on measurements of height and weight from the NATSIHS 2018–19.

Exposure for 2011 was estimated as the distribution of body mass index in Indigenous Australians from the AATSIHS 2012–13. The 2003 estimates were calculated using the same method as described for national estimates by comparing the trend in mean body mass index in 2011 to that estimated for Indigenous Australians in the 2003 Indigenous Australian Burden of disease study (Vos et al. 2007).

The 2003 estimates were based on data from the 2001 NATSIHS, which used measured height and weight information to estimate mean body mass index for Indigenous Australians living in remote areas. The relative difference between self-reported and measured body mass index were assumed to be the same in Indigenous Australians living in remote and non-remote areas, and was applied to the mean self-reported body mass index for Indigenous Australians living in non-remote areas (Vos et al. 2007).

References

AIHW 2017. Impact of overweight and obesity as a risk factor for chronic conditions: Australian Burden of Disease Study. Australian Burden of Disease Study series no. 11. Cat. no. BOD 12. Canberra: AIHW.

Cole TJ, Bellizzi MC, Flegal KM & Dietz WH 2000. Establishing a standard definition for child overweight and obesity worldwide: international survey. British Medical Journal 320:1240–3.

Vos T, Barker B, Stanley L & Lopez AD 2007. The burden of disease and injury in Aboriginal and Torres Strait Islander peoples 2003. Brisbane: University of Queensland.

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 2017–18 (ABS 2019).

Table 4.55: High blood pressure risk model parameters

Risk factor	High blood pressure – Systolic blood pressure
Disease outcome	Aortic aneurysm, atrial fibrillation & flutter, cardiomyopathy, chronic kidney disease, coronary heart disease, dementia, hypertensive heart disease, inflammatory heart disease, non-rheumatic valvular disease , peripheral vascular disease, rheumatic heart disease, stroke
TMRED	Systolic blood pressure between 110–115 mmHg
National data source	NHS 2017–18

Units for effect size calculation	Per 10 mmHg of systolic blood pressure increase

Estimates by socioeconomic group

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 Overarching methods and choices for risk factors.

2015, 2011 and 2003 estimates

Exposure data for 2015 was sourced from the NHS 2014–15 using the same method as for 2018. For 2011, data were sourced directly from the AHS 2011–12.

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.

Indigenous specific estimates

Exposure for 2018 was estimated using the same methods as described for the national estimates using systolic blood pressure distribution extracted from the NATSIHS 2018–19.

Exposure for 2011 was estimated as the distribution of blood pressure in Indigenous Australians from the AATSIHS 2012–13. The 2003 estimates were calculated using the same method as described for national estimates by comparing the trend in mean exposure in 2011 to exposure estimated in the 2003 Indigenous Australian burden of disease study (Vos et al. 2007).

The 2003 estimates were based on data published in the 2003 Australian burden of disease study (Vos et al. 2007). These data are from relatively small studies covering 2 regions (the DRUID study by Cunningham et al. 2006; Wang & Hoy 2003), in which it was assumed that the measured systolic blood pressure mean and standard deviations were representative of Indigenous Australians living in non-remote and remote areas.

References

ABS 2019. National Health Survey: users' guide, 2017–18. ABS cat. no. 4363.0. Canberra: ABS. Viewed 21 August 2017.

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Wang Z & Hoy WE 2003. Hypertension, dyslipidemia, body mass index, diabetes and smoking status in Aboriginal Australians in a remote community. Ethnicity & disease 13(3):324–30.

High cholesterol

The burden attributable to high cholesterol was estimated in people aged 25 and over. In ABDS 2018, the risk factor was updated to be as a measure of low-density lipoprotein (LDL) cholesterol to align with the methods for GBD 2019 (GBD 2019 Risk Factors Collaborators 2020).

Population attributable fraction

Exposure estimates

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

The exposure to high cholesterol in 2018 was calculated by comparing the mean exposure of total cholesterol 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 2018. The adjusted unit record data were used to estimate the distribution of exposure to high cholesterol in 2018.

Table 4.56: High cholesterol risk model parameters

Risk factor	High cholesterol – Low-density lipoprotein (LDL) cholesterol
Disease outcome	Coronary heart disease, stroke
TMRED	LDL cholesterol between 0.7–1.3 mmol/L

National data source	AHS 2011–12
Units for effect size calculation	Per 1 mmol/L of LDL cholesterol increase

Estimates by socioeconomic group

Exposure to high cholesterol was estimated using data from the AHS 2011–12, modelled to 2018, and the difference in the mean estimate in each quintile as described in Overarching methods and choices for risk factors.

2015, 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 2018 was used to estimate prevalence in total cholesterol in 2015 and 2003.

Indigenous specific estimates

Exposure in 2003, 2011 and 2018 was estimated as the distribution of total blood cholesterol levels in Indigenous Australians from the AATSIHS 2012–13.

References

Begg S, Vos T, Barker B, Stevenson C, Stanley L & Lopez AD 2007. The burden of disease and injury in Australia 2003. Cat. no. PHE 82. Canberra: AIHW.

GBD 2019 Risk Factors Collaborators 2020. Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. The Lancet 396:1223–49.

High blood plasma glucose

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 for 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 2019 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 2019 (14%) was used to estimate the direct PAF of chronic kidney disease due to high blood plasma glucose (GBD 2019 Risk Factors Collaborators 2020).
2. Exposure to high blood plasma glucose is linked to the remaining 86% 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 High blood plasma glucose risk model parameters below).

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 and 2018.

Diabetes

The prevalence of diabetes was based on the prevalence of type 1, type 2 and other diabetes in 2018. 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.

Table 4.57: High blood plasma glucose risk model parameters

Risk factor	Intermediate hyperglycaemia; diabetes – High fasting plasma glucose
Disease outcome	Chronic kidney disease, coronary heart disease, stroke
TMRED	Blood plasma glucose 4.8–5.4 mmol/L
National data source	AHS 2011–12
Units for effect size calculation	Per 1 mmol/L of fasting plasma glucose increase

Risk factor	Diabetes – Diabetes prevalence
Disease outcome	Bladder cancer, bowel cancer, breast cancer, cataract & other lens disorders, chronic kidney disease, coronary heart disease, dementia, glaucoma, liver cancer, lung cancer, ovarian cancer, pancreatic cancer, peripheral vascular disease
TMRED	No diabetes
National data source	ABDS 2018
Units for effect size calculation	Prevalence of type 1, type 2 and other diabetes

Risk factor	Diabetes – Direct PAFs
Disease outcome	Chronic kidney disease, type 2 diabetes, type 1 diabetes, other diabetes
TMRED	No diabetes
National data source	GBD 2019
Units for effect size calculation	Direct evidence

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 Overarching methods and choices for risk factors.

2015, 2011 estimates

The prevalence of high blood plasma glucose in 2011 was estimated using measured data from the AHS 2011–12. As mentioned above, these estimates were also applied for 2015.

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.

Indigenous specific estimates

Exposure in 2011 and 2018 was estimated as the distribution of fasting plasma glucose levels in Indigenous Australians from the AATSIHS 2012–13.

References

GBD 2019 Risk Factors Collaborators 2020. Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. The Lancet 396:1223–49.

Iron deficiency

These PAFs were estimated using direct evidence for people of all ages. 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 4 years.

Table 5.58: Iron deficiency risk model parameters

Risk factor	Iron deficiency
Disease outcome	Iron deficiency anaemia

TMRED	No Iron deficiency anaemia
National data source	n.a.
Units for effect size calculation	All of iron deficiency anaemia is attributable

Estimates by socioeconomic group

An estimate by socioeconomic group was not included to match other risk factors where exposure does not change by socioeconomic group.

Indigenous specific estimates

The same methods as for the national population were used.

Low birthweight & short gestation

Burden due to low birthweight & short gestation was estimated in people of all ages. The risk factor represents the combined impact of being born of low weight and prematurely and not as separate risk factors. Due to data limitations, this risk factor was only estimated for the reference year 2018.

Population attributable fraction estimated by comparative risk assessment

Exposure estimates

Exposure estimates were obtained using the National Perinatal Data Collection (NPDC), which contains data on all live births and stillbirths of at least 20 weeks gestation or 400 grams birthweight, and National Perinatal Mortality Data Collection (NPMDC) which contains data on all stillbirths and neonatal deaths of at least 20 weeks gestation or 400 grams birthweight. The number of deaths was obtained within categories of birthweight and gestational age, and also disaggregated by whether they occurred within the early or late neonatal period so as to correspond with relative risks provided by the GBD 2019 (see Table 4.59).

Table 4.59: Categories of exposure to gestation and birthweight and TMREDs from GBD 2019 study

Gestation (weeks)	Birthweight (grams)
[0, 24)	0–499, 500–999
[24, 26)	500–999
[26, 28)	500–999, 1000–1499
[28, 30)	500–999, 1000–1499, 1500–1999, 2000–2499, 2500–2999, 3000–3499
[30, 32)	500–999, 1000–1499, 1500–1999, 2000–2499, 2500–2999, 3000–3499, 3500–3999
[32, 34)	1000–1499, 1500–1999, 2000–2499, 2500–2999, 3000–3499, 3500–3999
[34, 36)	1000–1499, 1500–1999, 2000–2499, 2500–2999, 3000–3499, 3500–3999, 4000–4499
[36, 37)	1000–1499, 1500–1999, 2000–2499, 2500–2999, 3000–3499, 3500–3999, 4000–4499
[37, 38)	1000–1499, 1500–1999, 2000–2499, 2500–2999, 3000–3499, 3500–3999, 4000–4499
[38, 40)	1000–1499, 1500–1999, 2000–2499, 2500–2999, 3000–3499, 3500–3999, 4000–4499
[40, 42)	1500–1999, 2000–2499, 2500–2999, 3000–3499, 3500–3999, 4000–4499
TMRED	
[38, 40]	3000–3499, 3500–3999, 4000–4499
[40-42]	3000–3499, 3500–3999, 4000–4499

Source: GBD 2019.

PAFs were estimated using the comparative risk assessment method using NPDC and NPMDC exposure data for the reference year 2018. Relative risks and linked diseases were obtained from the GBD 2019 though not all were deemed appropriate within the Australian context following expert advice. Pre-term birth and low birthweight complications was the only linked disease that was attributed entirely to the risk factor and applied to people of all ages.

Table 4.60: Low birthweight & short gestation risk model parameters

Risk factor	Low birthweight & short gestation – Birthweight, gestational age

Disease outcome	Birth trauma & asphyxia, haemophilus influenza type-B, lower respiratory infections, meningococcal disease, neonatal infections, other disorders of infancy, other gastrointestinal diseases, other meningitis and encephalitis, otitis media, pneumococcal disease, pre-term birth & low birthweight complications, rotavirus, salmonellosis, sudden infant death syndrome, upper respiratory infections

TMRED	Birthweight ≥ 3000 g and gestational age ≥ 38 weeks
National data source	NPDC and NPMDC 2018
Units for effect size calculation	Prevalence of neonatal deaths by birthweight and gestational age categories

Estimates by socioeconomic group

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

Indigenous specific estimates

For Indigenous estimates for low birthweight & short gestation, the same methods and exposure data sources were used as for national estimates.

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 2018, 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 2018). 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 2018.

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 2018. The methods for these sequelae are described for the cause chronic kidney disease.

Table 4.61: Impaired kidney function risk model parameters

Risk factor	Chronic kidney disease stage 1–3 – stages 1–2
Disease outcome	Coronary heart disease, dementia, gout, peripheral vascular disease, stroke
TMRED	No chronic kidney disease
National data source	AHS 2011–12
Units for effect size calculation	Prevalence of chronic kidney disease stages 1–2

Risk factor	Chronic kidney disease stage 1–3 – stage 3
Disease outcome	Coronary heart disease, dementia, gout, peripheral vascular disease, stroke, chronic kidney disease
TMRED	No chronic kidney disease
National data source	AHS 2011–12
Units for effect size calculation	Prevalence of chronic kidney disease stage 3

Risk factor	Chronic kidney disease stage 4–5

Disease outcome	Coronary heart disease, dementia, gout, peripheral vascular disease, stroke, chronic kidney disease
TMRED	No chronic kidney disease
National data source	AHS 2011–12; ANZDATA for ABDS 2018
Units for effect size calculation	Prevalence of chronic kidney disease stage 4–5

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 2018 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.

2015, 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 years 2003 and 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 2018).

Chronic kidney disease stages 4-5

The prevalence of stages 4–5 chronic kidney disease was sourced as described for the cause chronic kidney disease for the ABDS 2018 (see Disease-specific methods - morbidity).

Indigenous specific estimates

Exposure to stages 1–3 chronic kidney disease for Indigenous Australians was estimated using data from the AATSIHS 2012–13, modelled to 2003 and 2018 and grouped into broad age- and sex-groups.

Exposure of stages 4–5 chronic kidney disease for Indigenous Australians was sourced as described for the cause chronic kidney disease.

References

AIHW 2018. Chronic kidney disease prevalence among Australian adults over time. Cardiovascular, diabetes and chronic kidney disease series no. 6. Cat. no. CDK 6. Canberra: AIHW.

Low bone mineral density

The burden attributable to low bone mineral density was measured in people aged 40 and over. Exposure by socioeconomic group was not estimated due to lack of available data.

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 Sànchez-Riera et al. (2014).

Table 4.62: Low bone mineral density risk model parameters

Risk factor	Low bone mineral density
Disease outcome	Falls
TMRED	95th percentile of the Third National Health and Nutrition Examination Survey (NHANES-III) cohort by age (Looker et al. 2012)
National data source	Geelong Osteoporosis Study (Barwon Health)
Units for effect size calculation	Standardised bone mineral density at the femoral neck

2015, 2011 and 2003 estimates

Methods for estimating exposure and calculating the PAFs for the 2015, 2011 and 2003 reference year were the same as those used for 2018.

Indigenous specific estimates

Standardised bone mineral density measurements at the femoral neck were not available for the Indigenous population. National rates of low bone mineral density, by age and sex, were applied to the Indigenous population to calculate Indigenous exposure estimates. This approach was supported by the same rates of self-reported osteoporosis for the Indigenous and national populations reported in the AATSIHS 2012–13 and the AHS 2011–12.

References

Henry MJ, Pasco JA, Korn S, Gibson JE, Kotowicz MA & Nicholson GC 2010. Bone mineral density reference ranges for Australian men: Geelong Osteoporosis Study. Osteoporosis International 21(6):909–17.

Looker AC, Borrud LG, Hughes JP, Fan B, Shepherd JA, Melton LJ et al. 2012. Lumbar spine and proximal femur bone mineral density, bone mineral content, and bone area: United States 2005–2008. National Center for Health Statistics, Vital Health Stat 11(251):1-132.

Sànchez-Riera L, Carnahan E, Vos T, Veerman L, Norman R, Lim SS et al. 2014. The global burden attributable to low bone mineral density. Annals of Rheumatic Diseases 73:1635–45.

Environmental risk factors

Air pollution

The fatal burden attributable to air pollution was measured by concentration of particulate matter 2.5 $\mu g/m^3$ (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 are particles suspended in the air with a diameter in a specified size range, 0–2.5 microns. Average annual PM2.5 data by mesh block geography from the Centre for Air pollution, energy and health Research (CAR) was estimated using satellite-calibrated ground monitoring stations for 2015 and 2018 and was provided via personal communication (L Knibbs 2020, pers. comm., 24 July 2020).

Geographic correspondence files were used to convert this satellite-modelled data by mesh blocks to SA2 geography. This was then aggregated and population-weighted to estimate national exposure to air pollution by age and sex.

Satellite modelling is limited in that it measures ambient air pollution levels rather than actual exposure to air pollution, but has the advantage over previous methods using monitoring stations only in that estimates are based on measurements from larger areas of Australia and are calibrated by ground monitoring stations. However, there are the same issues in that there can be variation in estimated levels of air pollution and actual levels experienced by the 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.

Table 4.63: Air pollution risk model parameters

Risk factor	Air pollution – Particulate matter (2.5 $\mu g/m^3$)
Disease outcome	COPD, coronary heart disease, lower respiratory infections, lung cancer, stroke, type 2 diabetes
TMRED	2.4–5.9 $\mu g/m^3$ (PM2.5)
National data source	Satellite-based model data
Units for effect size calculation	Daily maximum atmospheric particulate matter (PM2.5)

Indigenous specific estimates

For Indigenous estimates for air pollution, the same methods and exposure data sources were used as for national estimates though aggregated exposure data was weighted to the Indigenous population instead.

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).

Table 4.64: High sun exposure risk model parameters

Risk factor	Sun exposure
Disease outcome	Melanoma, non-melanoma skin cancer
TMRED	No health outcomes from sun exposure
National data source	Lucas et al. 2006; F Xiang 2015, pers. comm., 11 November 2015
Units for effect size calculation	Direct evidence

2015, 2011 and 2003 estimates

The same PAFs were used in 2015, 2011 and 2003 as they were not specific to 2018 but based on latitude.

Indigenous specific estimates

The burden from high sun exposure was not estimated for the Indigenous population as it was not possible to account for the impact of differences in skin melanin levels.

References

Lucas R, McMichael T, Smith W & Armstrong B 2006. Solar ultraviolet radiation: global burden of disease from solar ultraviolet radiation. Environmental burden of disease series. Geneva: World Health Organization.

Occupational exposures & 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). As per the disease group methods, pneumoconiosis was split into its component seqeulae of silicosis, asbestosis and other pneumoconiosis for ABDS 2018.

For injuries, direct evidence was sourced from Safe Work Australia, including data on the number of deaths occurring at work (Safe Work Australia 2019) and the number of workers' compensation injury claims annually (Safe Work Australia 2020). 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 2018 from Safe Work Australia (2019) divided by the incidence of admitted and non-admitted hospitalisations and emergency department presentations in the NHMD in 2018.

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 2020).

Industry

Exposure to working in certain types of industry was linked to various cancers, hearing loss and COPD (see Occupational exposure risk model parameters below). 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 Occupational exposure risk model parameters below). 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 2017).

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

Table 4.65: Occupational exposure risk model parameters

Risk factor	Occupational exposures and hazards – Occupational injuries
Disease outcome	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
TMRED	No occupational injuries
National data source	Work-related Traumatic Injury Fatalities, Australia 2018; Workers Compensation Statistics 2017–18
Units for effect size calculation	Direct evidence: number of workplace fatalities and the number of workers compensation claims for injuries

Risk factor	Occupational exposures and hazards – Occupational exposure to benzene or formaldehyde
Disease outcome	Acute lymphoblastic leukaemia, acute myeloid leukaemia, chronic lymphocytic leukaemia, chronic myeloid leukaemia, other leukaemias, nasopharyngeal cancer
TMRED	No occupational exposure to benzene or formaldehyde
National data source	Census of Population and Housing 2016; ABS Labour force survey, April 2020
Units for effect size calculation	Distribution of the labour force by broad industry type

Risk factor	Occupational exposures and hazards – Occupational exposure to arsenic, beryllium, cadmium, chromium, diesel engine exhaust, polycyclic aromatic hydrocarbons, nickel, second-hand smoke, silica
Disease outcome	Lung cancer
TMRED	No occupational exposure to arsenic, beryllium, cadmium chromium, diesel engine exhaust, polycyclic aromatic hydrocarbons, nickel, second-hand smoke, silica
National data source	Census of Population and Housing 2016; ABS Labour force survey, April 2020
Units for effect size calculation	Distribution of the labour force by broad industry type

Risk factor	Occupational exposures and hazards – Occupational exposure to asbestos, silicone and other particulate matter
Disease outcome	Asbestosis, silicosis, other pneumoconiosis
TMRED	No occupational exposure to asbestos, silicone and other particulate matter
National data source	GBD 2019

Units for effect size calculation	Direct evidence

Risk factor	Occupational exposures and hazards – Occupational exposure to sulphuric acid
Disease outcome	Laryngeal cancer
TMRED	No occupational exposure to sulphuric acid
National data source	Census of Population and Housing 2016; ABS Labour force survey, April 2020
Units for effect size calculation	Distribution of the labour force by broad industry type

Risk factor	Occupational exposures and hazards – Occupational exposure to trichloroethylene
Disease outcome	Kidney cancer
TMRED	No occupational exposure to trichloroethylene
National data source	Census of Population and Housing 2016; ABS Labour force survey, April 2020
Units for effect size calculation	Distribution of the labour force by broad industry type

Risk factor	Occupational exposures and hazards – Occupational exposure to particulate matter, gas and fumes
Disease outcome	COPD
TMRED	No occupational exposure to particulate matter, gas and fumes
National data source	Census of Population and Housing 2016; ABS Labour force survey, April 2020
Units for effect size calculation	Distribution of the labour force by broad industry type

Risk factor	Occupational exposures and hazards – Occupational exposure to asbestos
Disease outcome	Laryngeal cancer, lung cancer, mesothelioma, ovarian cancer
TMRED	No occupational exposure to asbestos
National data source	Census of Population and Housing 2016; ABS Labour force survey, April 2020
Units for effect size calculation	Distribution of the labour force by broad industry type

Risk factor	Occupational exposures and hazards – Occupational exposure to noise
Disease outcome	Hearing loss
TMRED	Background noise exposure
National data source	Census of Population and Housing 2016; ABS Labour force survey, April 2020
Units for effect size calculation	Distribution of the labour force by broad industry type

Risk factor	Occupational exposures and hazards – Occupational exposure to asthmagens
Disease outcome	Asthma
TMRED	Background asthmagen exposure

National data source	Census of Population and Housing 2016; ABS Labour force survey, April 2020
Units for effect size calculation	Distribution of the labour force by broad industry type

Risk factor	Occupational exposures and hazards – Occupational ergonomic factors
Disease outcome	Back pain and problems
TMRED	No occupational exposure to ergonomic factors causing back pain and problems
National data source	Census of Population and Housing 2016; ABS Labour force survey, April 2020
Units for effect size calculation	Distribution of the labour force by broad industry type

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. Exposure by socioeconomic group was not estimated due to lack of available data.

2015, 2011 and 2003 estimates

Methods for estimating exposure and calculating the PAFs in 2018 were followed for 2015 and 2011 estimates. The working population was estimated from the Labour Force Survey (ABS 2003, 2011, 2018) and disaggregated by occupation and industry using the 2016, 2011 and 2006 Census of Population and Housing (ABS 2017).

Indigenous specific estimates

The attributable burden in Aboriginal and Torres Strait Islander population was calculated in the same way as for the national population with the following changes.

The estimates of the number of Indigenous Australians working were sourced from the labour force survey (ABS 2020). The national estimates of the working population include long-term unemployed people, as they make up only a small proportion of the national population (1.3% in 2003 and 1.0% in 2011) (ABS 2011). As long-term unemployed people represent a much higher proportion of the Indigenous population (5.7% in 2003 and 6.0% in 2011) (AIHW analysis of the NATSISS 2002 and AATSIHS 2012–13), the estimate of economically active Indigenous population was adjusted down by the difference between these rates in each year.

Estimates of the number of Indigenous Australians working in 2003 were sourced from the Labour Force Survey 2006 (ABS 2007). These estimates were broken down by occupation and industry using estimates from the 2001 Census of Population and Housing.

National PAFs were used to estimate attributable burden due to carcinogens for Indigenous Australians, because the occupational turnover rates used in this calculation are not appropriate for the Indigenous population.

The Safe Work Australia data sets do not include an Indigenous identifier, so the direct evidence sourced from these publications was not available for the Indigenous population. Instead, an Indigenous to non-Indigenous rate ratio was calculated for all injury hospitalisations with an ICD-10-AM activity code of U73 ('While working for income'), by sex. This ratio was applied to the national exposure rates to derive Indigenous exposure estimates for injuries.

References

ABS (Australian Bureau of Statistics) 2003. Labour force, Australia, June 2003. ABS cat. no. 6202.0. Canberra: ABS. Viewed 22 June 2016.

ABS 2007. Labour force characteristics of Aboriginal and Torres Strait Islander Australians, experimental estimates from the Labour Force Survey, 2006. ABS cat. no. 6287.0. Canberra: ABS.

ABS 2011. Labour force, Australia, June 2011. ABS cat. no. 6202.0. Canberra: ABS. Viewed 22 June 2016.

ABS 2017. Census of Population and Housing: TableBuilder Pro, Australia, 2016. ABS cat. no. 2073.0 Canberra: ABS. Viewed 25 September 2018.

ABS 2018. Labour force, Australia, Jan 2018. ABS cat. no. 6202.0. Canberra: ABS. Viewed 25 September 2018.

ABS 2020. Labour force, Australia, Apr 2020. ABS cat. no. 6202.0. Canberra: ABS. Viewed 25 September 2018.

Kauppinen T, Toikkanen J, Pedersen D, Young R, Ahrens W, Boffetta P et al. 2000. Occupational exposure to carcinogens in the European Union. Occupational and Environmental Medicine 57(1):10–18.

Safe Work Australia 2019. Work-related traumatic injury fatalities, Australia 2018. Canberra: Safe Work Australia.

Safe Work Australia 2020. Australian Workers' Compensation Statistics 2017–18. Canberra: Safe Work Australia.

Unimproved sanitation

The burden from unimproved sanitation was not estimated for the non-Indigenous population due to lack of available exposure data, and was assumed to be close to 0.

Indigenous specific estimates

Exposure was estimated from the NATSIHS 2018–19 (for 2018) and the AATSIHS 2012–13 (for 2011). The estimate was based the number of Indigenous Australians living in the households that self-reported not having working sewerage facilities.

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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.

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ABDS quality framework

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 ABDS quality index 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.

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ABDS quality framework

ABDS 2018 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 2018 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 national 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 2019 and other studies were appropriate for:
    - the particular risk factor
    - the Australian context.

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 Table 5.1. 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 Additional material in Overarching methods and choices for ABDS 2018). 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 2018 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.

Table 5.1: ABDS quality index, Dimension I—Data relevance scores

Score	Criteria
5	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. No severity distribution needed, or high-quality empirical data on this distribution were available.
4	Same as '5' BUT not fully enumerated with either known gaps in coverage 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. It was also used for estimates with components that scored between 5 and 3.
3	Same as '4' BUT with medium specificity of the data source to the condition/sequela being estimated. For example: • for survey data, there was known medium correlation between what was collected (for example, measurement, self-report and clinical diagnosis) and the condition • for hospitals data, condition had a medium likelihood of hospitalisation (that is, condition only results in hospitalisation in severe or certain cases). Also, data were from a single, large area (more than 1 state/territory) Australian study of very good quality or from a systemic meta-analysis that could be generalised or from a review of Australian studies with medium currency. It was also used for estimates with components that scored between 4 and 2.
2	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 not changing over time. It was also used for estimates with components that scored between 3 and 1.
1	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.

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 Table 5.2.

Table 5.2: ABDS quality index, Dimension II—Data transformation scores

Score	Criteria
5	Data were directly applied to the model and minimal or no extra modelling was required. Severity distribution (if required) was obtained directly from the data..
4	Rates were projected to the reference year, taking into account changes in underlying trend, and applied to reference population/broad sex or age distributions were converted to 5-year age groups using trend analyses/pooled data from multiple years or sources with comparable definitions/ratios of related and primary data (for example, incidence-to-separations ratio from 1 state) applied to primary data (for example, applied to national separations data). Severity distribution (if required) was obtained from an Australian study. It was also used for estimates with components that scored between 5 and 3.
3	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). It was also used for estimates with components that scored between 4 and 2.
2	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. It was also used for estimates with components that scored between 3 and 1.
1	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.

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 2018 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 and risk factors (see tables below) 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, 2015, 2011 and 2003 estimates

The data and methods used for 2018 estimates underpinned the sub-national, 2015, 2011 and 2003 estimates, so the quality of these estimates must be considered together with the broad sub-national, 2015, 2011 and 2003 methods described in Overarching methods and choices for ABDS 2018, and the specific details described in Disease specific methods and 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 Years of life lost (YLL)). 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

The table below 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 Disease specific methods. 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 the table below. 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, 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 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 Risk factor specific methods.

This interactive data visualisation reports on the quality information regarding the non-fatal burden estimates of each disease and injury for the national population and for the Aboriginal and Torres Strait Islander population. The specific disease or injury can be selected by the user. There are 2 sections – the first section displays the quality information of the estimates for the national Australian population, the second section displays the quality information of the estimates for the Aboriginal and Torres Strait Islander population. For each disease and injury, there are two scores – one for data and one for methods. Each score is a whole number out of 5. There is also a description of the data and methods used to obtain the non-fatal burden estimate.

Visualisation not available for printing

Guide to quality scoring YLD data source or method used in ABDS 2018

Rating	Data score	Method score
5 stars	Recent, relevant, fully enumerated data of high quality data specific to the Australian population. Where severity is required, this is derived from the same data source.	Minimal or no extra modelling; estimate was derived directly from source data
4 stars	Relevant, high quality data however data is either not fully enumerated, is non-specific to the population, has high variability, is not derived from the reference year or where severity is required it is not available. This may also be a combination of a 5 and 3 star rating.	Modelling such as disaggregating broad age groups into finer age groupings or applying person: separation hospitalisation ratios from linked data to non-linked, however the modelling is minimal and primarily specific to the population condition-specific and is evidence based.This may also be a combination of a 5 and 3 star rating.

3 stars	Relevant, high quality data however for the condition required it has either medium specificity, derived from a single smaller-scale Australian study or is from a generalisable review or meta-analyses. This may also be a combination of a 4 and 2 star rating.	Assumptions to be made as there is no information to model trends, or modelling was required using methods which were not specific to the population or were from various sources with differing definitions for the condition. This may also be a combination of a 4 and 2 star rating.
2 stars	A small good-quality Australian/ International study/ Review or meta-analyses generalisable to the Australian population that may not be recent or has low specificity for that condition. This may also be a combination of a 3 and 1 star rating.	Indirect modelling methods based on evidence which was; less than 5 years from the reference year, non-specific to the the condition or population or inferences were made from related data with medium specificity. This may also be a combination of a 3 and 1 star rating.
1 star	A small Australian study more than 5 years old from the reference year with questionable applicability/ an international study with questionable generalisability to the Australian population or is indirect and from a secondary data source.	Indirect modelling methods based on evidence which was either; more than 5 years old to the reference year, non-specific to the condition or population or inferences were made from slightly related data.

This interactive data visualisation reports on the quality information regarding the risk factor exposure data estimates for the national population and for the Aboriginal and Torres Strait Islander population. The specific risk factor can be selected by the user. There are 2 sections – the first section displays the quality information of the risk factor estimates for the national Australian population, the second section displays the quality information of the risk factor estimates for the Aboriginal and Torres Strait Islander population. For each risk factor, there are two scores – one for data and one for methods. Each score is a whole number out of 5. There is also a description of the data and methods used to obtain risk factor exposure data.

Visualisation not available for printing

Guide to quality scoring Risk factor exposure data source or method used in ABDS 2018

Rating	Data score	Method score
5 stars	Recent, relevant, fully enumerated data of high quality with either diagnostically confirmed exposure; or established high correlation between self-report and clinical diagnosis of exposure specific to the Australian population.	Minimal or no extra modelling; estimate was derived directly from source data
4 stars	Relevant, high quality data however data is either not fully enumerated, not diagnostically confirmed, is non-specific to the population, has high variability, is not derived from the reference year. This may also be a combination of a 5 and 3 star rating.	Modelling such as disaggregating broad age groups into finer age groupings or to project estimates to the reference year, however the modelling is minimal and primarily specific to the population exposure-specific and is evidence based. This may also be a combination of a 5 and 3 star rating.
3 stars	Relevant, high quality data however for the exposure required it has either medium specificity to exposure, derived from a single smaller-scale Australian study or is from a generalisable review or meta-analyses. This may also be a combination of a 4 and 2 star rating.	Assumptions to be made as there is no information to model trends, or modelling was required using methods which were not specific to the population. This may also be a combination of a 4 and 2 star rating.
2 stars	A small good-quality Australian/ International study/ Review or meta-analyses generalisable to the Australian population that may not be recent or has low specificity for that exposure. This may also be a combination of a 3 and 1 star rating.	Indirect modelling methods based on evidence which was; less than 5 years from the reference year, non-specific to the exposure or population or inferences were made from related data with medium specificity. This may also be a combination of a 3 and 1 star rating.
1 star	A small Australian study more than 5 years old from the reference year with questionable applicability/ an international study with questionable generalisability to the Australian population or is indirect and from a secondary data source.	Indirect modelling methods based on evidence which was either; more than 5 years old to the reference year, non-specific to the exposure or population or inferences were made from slightly related data.

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 fairly 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.

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Technical notes

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Disease group methods development and analysis were undertaken in consultation with disease experts by:

- blood & metabolic disorders—Vergil Dolar (national), Kalijah Madsen-Guarini (Indigenous)
- cancer & other neoplasms—Chenkun Zhao (national), Nick Mann (Indigenous and national)
- cardiovascular diseases—Julianne Garcia (national), Tracy Dixon (Indigenous)
- endocrine disorders—Julianne Garcia (national), Tracy Dixon (Indigenous)
- gastrointestinal disorders—Ruihua Guo and Charles Chak (national), Kalijah Madsen-Guarini (Indigenous)
- hearing & vision disorders—Ruihua Guo (national), Ilona Brockway and Tracy Dixon (Indigenous)
- infant & congenital conditions—Wendy Ho (national), Eleanor Bateman (Indigenous)
- infectious diseases—Lucas Mills (national and Indigenous)
- injuries—Nick Mann (national and Indigenous)
- kidney & urinary disorders—Anna Reynolds (national), Tracy Dixon (Indigenous)
- mental health conditions & substance use disorders—Julianne Garcia (national), Kalijah Madsen-Guarini (Indigenous)
- musculoskeletal conditions—Anna Reynolds (national), Ilona Brockway (Indigenous)
- neurological conditions—Yolanda Lovie-Toon (national), Ilona Brockway (Indigenous)
- oral disorders—Charles Chak and Wendy Ho (national), Kalijah Madsen-Guarini (Indigenous)
- reproductive & maternal conditions—Eleanor Bateman (national and Indigenous), Paula Laws (national)
- respiratory conditions—Anna Reynolds (national), Ilona Brockway (Indigenous)
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Technical notes

Abbreviations and symbols

Abbreviations

AATSIHS	Australian Aboriginal and Torres Strait Islander Health Survey
ABDS	Australian Burden of Disease Study
ABS	Australian Bureau of Statistics
ACD	Australian Cancer Database
ACFDR	Australian Cystic Fibrosis Data Registry
ACHI	Australian Classification of Health Interventions
ACOD	associated cause of death
ACT	Australian Capital Territory
AHS	Australian Health Survey
AIHW	Australian Institute of Health and Welfare
ALL	acute lymphoblastic leukaemia
ALSWH	Australian Longitudinal Study on Women's Health
AML	acute myeloid leukaemia
ANU	Australian National University
ANZDATA	Australian and New Zealand Dialysis and Transplant Registry
APC	admitted patient care
APEG	Australasian Paediatric
ASGS	Australian Statistical Geography Standard
AusDiab	Australian Diabetes, Obesity and Lifestyle Study
BEACH	Bettering the Evaluation and Care of Health survey
BMI	Body Mass Index
BOLD	Burden of Obstructive Lung Disease
CAREX	Carcinogen Exposure Research Project
CDE	Census Data Enhancement
CI	confidence interval
CIMHA	Consumer Integrated Mental Health Application
CLL	chronic lymphocytic leukaemia
CML	chronic myeloid leukaemia
COPD	chronic obstructive pulmonary disease
DALY	disability-adjusted life years
DCIS	ductal carcinoma *in situ*
DisMod II	Disease Modelling II
DLQI	Dermatology Life Quality Index

DMFT	decayed, missing and filled teeth
DT	decayed teeth
ED	emergency department
EMD	Enhanced Mortality Database
FASD	fetal alcohol spectrum disorders
FGID	functional gastrointestinal disorders
FT	filled teeth
g	gram
GBD	Global Burden of Disease
GBS	Guillain-Barré Syndrome
GMFCS	Gross Motor Function Classification System
GORD	gastro-oesophageal reflux disease
GP	general practitioner
HALE	health-adjusted life expectancy
Hib	*Haemophilus influenzae* type b
HIV	human immunodeficiency virus
HIV/AIDS	human immunodeficiency virus/acquired immune deficiency syndrome
IBD	inflammatory bowel disease
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
ICPC-2+	International Classification of Primary Care Version 2+
IDEA	Intellectual Disability Exploring Answers
IRSD	Index of Relative Socioeconomic Disadvantage
kg/m2	kilogram per square metre
LT	long-term
MBS	Medicare Benefits Schedule
MCOD	multiple causes of death
MET	metabolic equivalent of tasks
µg/m3	micrograms per cubic metre
mmHg	millimetre of mercury
mmol/L	millimole per litre
MND	motor neurone disease
MT	missing teeth
NATSIHS	National Aboriginal and Torres Strait Islander Health Survey
NATSISS	National Aboriginal and Torres Strait Islander Social Survey
NDI	National Death Index
NDR	National Diabetes Registry

NDSHS	National Drug Strategy Household Survey
NDSS	National Diabetes Services Scheme
NHANES-III	Third National Health and Nutrition Examination Survey
NHMD	National Hospital Morbidity Database
NHMP	National Homicide Monitoring Program
NHS	National Health Survey
NIHSI AA v0.5	National Integrated Health Services Information Analysis Asset version 0.5
NMD	National Mortality Database
NMSC	non-melanoma skin cancer
NNAPEDCD	National Non-admitted Patient Emergency Department Care Database
NNDSS	National Notifiable Diseases Surveillance System
NNPAS	National Nutrition and Physical Activity Survey
NPDC	National Perinatal Data Collection
NPMDC	National Perinatal Mortality Data Collection
NSAOH	National Survey of Adult Oral Health
NSW	New South Wales
NT	Northern Territory
NZBDS	New Zealand Burden of Disease Study
PAF	population attributable fraction
PBS	Pharmaceutical Benefits Scheme
PM	particulate matter
PPV	positive predictive value
PSS	Personal Safety Survey
RACS	Residential Aged Care Services
RPBS	Repatriation Pharmaceutical Benefits Scheme
RTI	road traffic injury
RR	relative risks
RSE	relative standard error
SA	South Australia
SA2	Statistical Area Level 2
SDAC	Survey of Disability, Ageing and Carers
SEIFA	Socio-Economic Indexes for Areas
ST	short-term
TBI	traumatic brain injury
TMRED	theoretical minimum risk exposure distribution
TMRLT	theoretical minimum risk life table
UCOD	underlying cause of death
WA	Western Australia
WARDA	Western Australian Registry of Developmental Anomalies

WHO	World Health Organization
YLD	years lived with disability
YLL	years of life lost

Symbols

>	greater than
≥	greater than and/or equal to
<	less than
—	nil or rounded to zero
. .	not applicable
n.a.	not available
Π	product of
Σ	sum of

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Technical notes

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.
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).
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.
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).
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.
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).

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'.

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.

rate: A rate is one number (the numerator) divided by another number (the denominator). The numerator is commonly the number of events in a specified time. The denominator is the population at risk of the event. Rates (crude, age-specific and age-standardised) are generally multiplied by a number such as 100,000 to create whole numbers.

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

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

relative risk: The risk of an event relative to exposure, calculated as the ratio of the probability of the even occurring in the exposed group to the probability of it occurring in the non-exposed group.

risk exposure distribution: The measure of the spread or distribution of exposure to the risk factor in the population that have encountered, experienced, or have the risk factor.

risk factor: Any factor that causes or increases the likelihood of a health disorder or other unwanted condition or event.

risk-outcome pair: Associates a condition in the disease list with a known risk factor for that condition.

sequelae: Health consequences of diseases and injuries, such as heart failure due to coronary heart disease. Each sequela may be mapped to one or more health states.

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

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

years of life lost (YLL): Measures years of life lost due to premature death, defined as dying before the global ideal life span at the age of death. YLL represent fatal burden.

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Data

Data tables: Australian Burden of Disease Study: Methods and supplementary material 2018

Resource

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