

## 2 Methodological developments

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

### 2.1 Social value choices

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

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

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

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

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

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

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

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

### **Box 2.1: Interpreting a disability weight**

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

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

## **2.2 Causal attribution**

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

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

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

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

## Categorising deaths

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

### **Box 2.2: Death registration in Australia**

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

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

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

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

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

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

## **Redistributing non-specific causes of death**

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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





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

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

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

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

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

## Alternative categories

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

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

## 2.3 Comorbidity and health

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

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

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

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

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

### **Box 2.3: Combining disability weights**

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

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

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

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

*where  $\prod$  denotes the product operator.*

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

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

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

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

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

## 2.4 Risks to health

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

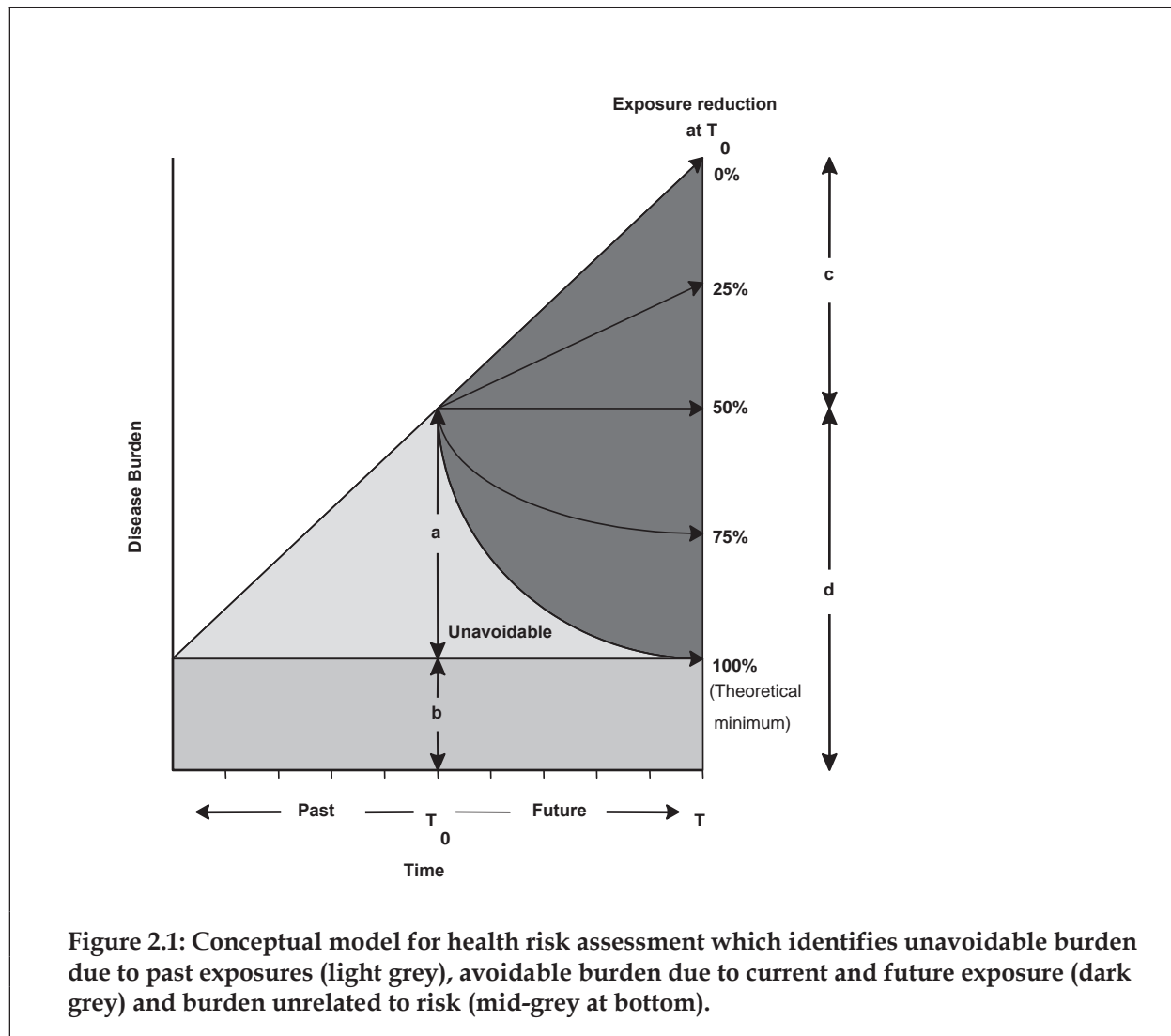
Techniques for attributing outcomes to health risks have advanced considerably in recent times, particularly through the contribution of the Comparative Risk Assessment (CRA) project. This was a large-scale effort by international panels of experts under the direction of the World Health Organization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

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

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

## Explicit 'counterfactuals'

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



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

## Joint risk attribution

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

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

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

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

where  $\text{PAF}_i$  is the PAF of individual risk factors.

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

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

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

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

## 2.5 Past, present and future burden

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

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

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

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

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

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

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

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

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

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

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

## **Mortality trends and projections**

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

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

## **Incidence and case-fatality**

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

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

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

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

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

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

## **Non-fatal conditions**

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

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

## 2.6 Differentials in burden

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

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

### Categorising geographic areas

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

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

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

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

## **Estimating burden for subpopulations**

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

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

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

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

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

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

## **Subpopulation comparisons in this report**

This report is limited to the following subpopulation comparisons:

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

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