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The principles and practice of assessing population health on a routine and comprehensive basis: a case study

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Abstract

The ‘burden of disease’ methodology introduced by Murray and Lopez in the early 1990s represents a useful platform from which to develop insights into contemporary issues confronting health systems. However, it is often regarded as having limited value beyond what is generally accepted as the domain of public health. The central argument developed here is that while such an impression with respect to the underlying conceptual framework is largely unfounded, a substantial reinterpretation of Murray and Lopez’s original contributions may be required before this possibility achieves more general acceptance. Underlying this thesis is the proposition that with a new model of implementation, a number of practical impediments—which in the past have prevented the methods from being employed for other than a limited range of applications—can be overcome.

The setting in which to test this hypothesis presented itself in the form of a project undertaken by the University of Queensland in collaboration with the Australian Institute of Health and Welfare (AIHW). Commissioned by the Australian Government Department of Health and Ageing (DOHA) in early 2003 this project had as one of its objectives facilitating the routine use of the framework by government agencies such as AIHW and state and territory health departments. As such, simple program logic principles were adopted to assess the characteristics of an alternative implementation model with respect to both its range of outputs compared to what might be expected and its intended outcomes more broadly with respect to using the proposed model on a routine basis.

The main findings are presented in four parts. Part I comprises a critical evaluation of existing tools and methods, or inputs, before proposing alternative tools and workflows that make the underlying conceptual framework more internally consistent, efficient and flexible in practice. Parts II and III comprise a portfolio of four papers, or outputs, that serve as important links between the proposed model and the intended outcome by seeking to address several areas of contemporary policy concern. The final part is a commentary on the success or otherwise of the case study with respect to government use of the proposed model on a routine basis.

The key conclusion to draw from this thesis is that, with respect to outputs, the proposed model allowed for a much greater range of analyses than was attempted in the first ‘burden of disease study’ in Australia. The specific areas where these benefits were observed included

the ability disaggregate the primary results to a flexible range of geographic areas, the ability to fore- and back-cast these results while preserving internal consistency between parameters, the ability to generate a complete set of alternative results for various groupings of Australia's Indigenous populations, and the ability to account for dependent and independent comorbidity in disability calculations. In addition, the model extended the range of secondary analyses regarded as feasible to areas not previously associated with 'burden of disease', such as health expenditure projections and causal decomposition analyses of health-adjusted life expectancy.

With respect to outcomes however, the conclusions are more circumspect. Certainly, the analysis demonstrated that three state governments successfully implemented the model on at least one occasion each to update the original case study outputs with more recent data. A delay in the release of critical data was the primary factor preventing more extensive use in these settings. Nevertheless, a more coordinated adoption of the model at a national level was not achieved in the five years since its development, despite efforts by Australia's peak population health information advisory committee to facilitate this outcome. While there is emerging evidence of renewed interest in national forums to make further use of the Murray and Lopez framework in Australia, it is too early to predict what role design considerations will play in these developments.

Declaration by author

This thesis is composed of my original work, and contains no material previously published or written by another person except where due reference has been made in the text. I have clearly stated the contribution by others to jointly-authored works that I have included in my thesis.

I have clearly stated the contribution of others to my thesis as a whole, including statistical assistance, survey design, data analysis, significant technical procedures, professional editorial advice, and any other original research work used or reported in my thesis. The content of my thesis is the result of work I have carried out since the commencement of my research higher degree candidature and does not include a substantial part of work that has been submitted to qualify for the award of any other degree or diploma in any university or other tertiary institution. I have clearly stated which parts of my thesis, if any, have been submitted to qualify for another award.

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Additional statements

Publications during candidature

Books and book sections

Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *Burden of disease and injury in Australia, 2003*, Australian Institute of Health and Welfare, Canberra, 2007.

Mathers, C, Salomon, J, Ezzati, M, Begg, S, Vander Hoorn, S and Lopez, A, "Sensitivity and uncertainty analyses for burden of disease and risk factor estimates", in Lopez, A, Mathers, C, Ezzati, M, Jamison, D and Murray, C, eds, *Global burden of disease and risk factors*, The World Bank and Oxford University Press, Washington, 2006.

Lopez, A, Begg, S and Bos, E, "Demographic and epidemiological characteristics of major regions, 1990-2001", in Lopez, A, Mathers, C, Ezzati, M, Jamison, D and Murray, C, eds, *Global burden of disease and risk factors*, The World Bank and Oxford University Press, Washington, 2006.

Refereed journal articles

Zhao, Y, Wright, J, Begg, S and Guthridge, S, "Decomposing Indigenous Life Expectancy Gap by Risk Factors: A Life Table Analysis", *Population Health Metrics*, in press.

McCormack, GR, Shiell, A, Giles-Corti, B, Begg, S, Veerman, L, Geelhoed, E, Amarasinghe, A, Emery, JCH, "The association between sidewalk length and walking for different purposes in established neighborhoods", *International Journal of Behavioral Nutrition and Physical Activity*, 2012, vol. 9(1), pp. 92.

Askew, D, Jackson, C, Ware, RS, Begg, S, Crossland, L, Cranston, P, and Mitchell, P, "Diabetic retinopathy screening and monitoring of early stage disease in general practice: Design and methods", *Contemporary Clinic Trials*, 2012, vol. 33(5), pp. 969-75.

Katzenellenbogen, JM, Vos, T, Somerford, P, Begg, S, Semmens, JB and Codde, JP, "Burden of stroke in indigenous Western Australians: a study using data linkage", *Stroke,* 2011, vol. 42(6), pp. 1515-21.

Katzenellenbogen, JM, Vos, T, Somerford, P, Begg, S, Semmens, JB and Codde, JP, "Excess mortality rates for estimating the non-fatal burden of stroke in Western Australia: a data linkage study", *Cerebrovasc Dis,* 2010, vol. 30(1), pp. 57-64.

Katzenellenbogen, JM, Begg, S, Somerford, P, Anderson, CS, Semmens, JB, Codde, JP and Vos, T, "Disability burden due to stroke in Western Australia: new insights from linked data sources", *Int J Stroke,* 2010, vol. 5(4), pp. 269-77.

Magliano, D, Peeters, A, Vos, T, Sicree, R, Shaw, J, Sindall, C, Haby, M, Begg, S and Zimmet, P, "Projecting the burden of diabetes in Australia - what is the size of the matter?", *Australian and New Zealand Journal of Public Health,* 2009, vol. 33(6), pp. 540-3.

Vos, T, Barker, B, Begg, S, Stanley, L and Lopez, AD, "Burden of disease and injury in Aboriginal and Torres Strait Islander Peoples: the Indigenous health gap", *Int J Epidemiol*, 2009, vol. 38(2), pp. 470-7.

Begg, SJ, Vos, T, Barker, B, Stanley, L and Lopez, AD, "Burden of disease and injury in Australia in the new millennium: measuring health loss from diseases, injuries and risk factors", *Med J Aust*, 2008, vol. 188(1), pp. 36-40.

Begg, S, Vos, T, Goss, J and Mann, N, "An alternative approach to projecting health expenditure in Australia", *Aust Health Rev*, 2008, vol. 32(1), pp. 148-55.

Page, A, Begg, S, Taylor, R and Lopez, A, "Global comparative assessments of life expectancy: the impact of migration with reference to Australia", *Bulletin of the World Health Organization,* 2007, vol. 85, pp. 474-81.

Michaud, CM, McKenna, MT, Begg, S, Tomijima, N, Majmudar, M, Bulzacchelli, MT, Ebrahim, S, Ezzati, M, Salomon, JA, Gaber Kreiser, J, Hogan, M and Murray, CJ, "The burden of disease and injury in the United States 1996", *Popul Health Metr,* 2006, vol. 4(1), p. 11.

Mathers, CD, Iburg, KM and Begg, S, "Adjusting for dependent comorbidity in the calculation of healthy life expectancy", *Popul Health Metr,* 2006, vol. 4(1), p. 4.

Rao, C, Lopez, AD, Yang, G, Begg, S and Ma, J, "Evaluating national cause-of-death statistics: principles and application to the case of China", *Bull World Health Organ,* 2005, vol. 83(8), pp. 618-25.

Begg, S, Rao, C and Lopez, AD, "Design options for sample-based mortality surveillance", *Int J Epidemiol,* 2005, vol. 34(5), pp. 1080-7.

Non-refereed electronic publications

Begg, S, Bright, M and Harper, C, *Burden of disease and injury, life expectancy and health-adjusted life expectancy in Queensland Health Service Districts, 2006,* Queensland Health, Brisbane, 2009, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c6_districts_final.pdf

Begg, S, Bright, M and Harper, C, *Differentials in burden of disease and injury in Queensland, 2006,* Queensland Health, Brisbane, 2009, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c4_series2.pdf

Bright, M, Begg, S and Harper, C, *Age group differences in burden of disease and injury in Queensland, 2006,* Queensland Health, Brisbane, 2009, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c3_series2.pdf

Bright, M, Begg, S and Harper, C, *Leading causes of burden of disease and injury in Queensland, 2006,* Queensland Health, Brisbane, 2009, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c2_series2.pdf

Bright, M, Begg, S and Harper, C, *Overview of the burden of disease and injury in Queensland, 2006,* Queensland Health, Brisbane, 2009, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c1_series2.pdf

Begg, S, Khor, S, Bright, M, Stanley, L, O'Brien, J and Harper, C, *Burden of disease and injury in Queensland Health Service Districts, 2003,* Queensland Health, Brisbane, 2008, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c6_districts_final.pdf

Begg, S, Khor, S, Bright, M, Stanley, L, O'Brien, J and Harper, C, *Differentials in the burden of disease and injury in Queensland, 2003,* Queensland Health, Brisbane, 2008, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c5_differential_final.pdf

Begg, S, Khor, S, Bright, M, Stanley, L, O'Brien, J and Harper, C, *Risk factor impact on the burden of disease and injury in Queensland, 2003,* Queensland Health, Brisbane, 2008, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c4_risk_factors_final.pdf

Begg, S, Khor, S, Bright, M, Stanley, L, O'Brien, J and Harper, C, *Age group differences in burden of disease and injury in Queensland, 2003,* Queensland Health, Brisbane, 2008, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c3_age_final.pdf

Begg, S, Khor, S, Bright, M, Stanley, L, O'Brien, J and Harper, C, *Leading causes of burden of disease and injury in Queensland, 2003,* Queensland Health, Brisbane, 2008, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c2_causes_final.pdf

Begg, S, Khor, S, Bright, M, Stanley, L, O'Brien, J and Harper, C, *Overview of the burden of disease and injury in Queensland, 2003,* Queensland Health, Brisbane, 2008, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/c1_overview_final.pdf

Begg, S, Khor, S, Bright, M, Stanley, L and Harper, C, *Burden of disease and health-adjusted life expectancy in Health Service Districts of Queensland Health, 2003,* Queensland Health, Brisbane, Queensland Health, Brisbane, 2008, viewed 16 August 2011, http://www.health.qld.gov.au/ph/documents/pdu/hale_for_hsd_final.pdf

Reports

Vos, T, Goss, J, Begg, S and Mann, N, "Projection of health care expenditure by disease: a case study from Australia: A background paper", *World Economic and Social Survey 2007: Development in an Ageing World - 60th Anniversary Edition, 1948-2007*, United Nations, New York, 2007.

Vos, T and Begg, S, *The burden of cardiovascular disease in Australia for the year 2003*, National Heart Foundation of Australia, Canberra, 2007.

Vos, T, Goss, J, Begg, S and Mann, N, *Projected Health Care Costs Report: A technical report for the Australian Government Department of Health & Ageing*, School of Population Health, University of Queensland and Australian Institute of Health and Welfare, Brisbane, 2005.

Vos, T, Barendregt, J and Begg, S, *Estimating the contribution of lifestyle risk factors to the burden of disease in Australia in 2003*, School of Population Health, University of Queensland, Brisbane, 2005.

Yusoff, A, Mustafa, A, Kaur, G, Omar, M, Vos, T, Rao, V and Begg, S, *Malaysian Burden of Disease and Injury Study*, Division of Burden of Disease, Institute of Public Health, Ministry of Health, Kuala Lumpur, 2004.

Doran, C, Begg, S, Vos, T and Lopez, A, *Fiji Burden of Disease from Tobacco: A report prepared on behalf of WHO Fiji Regional Office, Secretariat of the Pacific Communities and the Fiji National Health Research Committee*, School of Population Health, University of Queensland, Brisbane, 2004.

Vos, T, Goss, J, Begg, S and Mann, N, *Projected Health Care Costs: A Report Prepared for the Department of Health and Ageing*, School of Population Health, University of Queensland & Australian Institute of Health and Welfare, Brisbane & Canberra, 2004.

Publications included in this thesis

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Vos, T	Conception and design (25%) Analysis and interpretation of data (25%) Drafting and writing (10%)
Goss, J and Mann, N (collectively)	Conception and design (50%) Analysis and interpretation of data (50%) Drafting and writing (10%)

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Begg, S	Conception and design (40%) Analysis and interpretation of data (60%) Drafting and writing (80%)
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Lopez, AD	Conception and design (5%) Analysis and interpretation of data (5%) Drafting and writing (5%)

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Begg, S	Conception and design (40%) Analysis and interpretation of data (60%) Drafting and writing (80%)
Vos, T	Conception and design (40%) Analysis and interpretation of data (20%) Drafting and writing (10%)
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Contributions by others to the thesis

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Keywords and classifications

Keywords

descriptive epidemiology, burden of disease, population health, health economics

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Fields of Research (FoR) Classification

FoR code: 1117, Public Health and Health Services, 75%

FoR code: 1402, Applied Economics, 25%

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Abbreviations

ABS	Australian Bureau of Statistics
AF	Attributable fraction
AHMAC	Australian Health Ministers' Advisory Council
AHMC	Australian Health Ministers' Conference
AIHW	Australian Institute of Health and Welfare
ALHE	Absolute lost health expectancy
ANPHA	Australian National Preventive Health Agency
APHDPC	Australian Population Health Development Principal Committee
ARC	Australian Research Council
ARIA+	Accessibility/Remoteness Index of Australia
AusDIAB	Australian Diabetes, Obesity and Lifestyle study
BEACH	Bettering the Evaluation And Care of Health
BMI	Body mass index
CEOs	Chief Executive Officers
COAG	Council of Australian Governments
COPD	Chronic obstructive pulmonary disease
CVD	Cardiovascular disease
DALYs	Disability life adjusted years
DE_0	Disability expectancy at birth
DFLE	Disability free life expectancy
DOHA	Australian Government Department of Health and Ageing
DW	Disability weight
GBD 1990	Global burden of disease and injury study 1990
GBD 2000	Global burden of disease and injury study 2000
GDP	Gross domestic product
GGB	Generalise growth balance
GST	Goods and services tax
HALE	Health adjusted life expectancy
HIV/AIDS	Human Immunosuppressive Virus/Acquire Immune Deficiency Syndrome
HPPPC	Health Policy Priorities Principal Committee
ICD	International Classification of Diseases
IPM	Incidence-prevalence model

LE	Life expectancy
MHS	National Mental Health Survey 1997
NBoDCN	National Burden of Disease Collaborative Network
NEHIPC	National E-Health and Information Principal Committee
NHIMPC Plan	National Health Information Management Principal Committee Strategic Work Plan 2007-08 to 2012-13
NHHRC	National Health and Hospitals Reform Commission
NHIA	National Health Information Agreement
NHMRC	National Health and Medicare Research Council
NHP Framework	National Health Performance Framework
NHPA	National Health Performance Authority
NMCBoD	National Monitoring Centre for Burden of Disease
NNDSS	National Notifiable Diseases Surveillance System
NPHIWG	National Public Health Information Working Group
NSW	New South Wales
NT	Northern Territory
OECD	Organisation for Economic Co-operation and Development
PHIDG	Public Health Information Development Group
PIF	Population impact fraction
PYLD	Prevalent years lived with to disability
RLHE	Relative lost health expectancy
RR	Relative risk
SDAC	Survey of Disability, Ageing and Carers 2003
SLA	Statistical local area
STDs	Sexually transmitted diseases
T1DM	Type 1 diabetes mellitus
T2DM	Type 2 diabetes mellitus
USA	United States of America
WA	Western Australia
WHO	World Health Organization
YLD	Years lived with disability
YLL	Years of life lost

Part I: Introduction and methods

Chapter 1: Introduction

Several broad social trends in Australia are likely to be of universal interest. On the one hand, there has been unprecedented growth in levels of health over the last century and, notwithstanding examples of persistent disadvantage, we are, by most measures, healthier than ever. On the other, the economic cost of accommodating this growth has increased much faster than would seem sustainable but for the very high value we place on health.[1] For example, over the last half century alone, the expected life span of the average Australian has increased by around 15%, to become amongst the longest in the world at a projected 82 years in 2011.[2] In contrast, total expenditure on health compared to the rest of the economy has grown over the same period by almost one and a half times to an estimated 9.5% of GDP.[3] These seemingly inexorable but related trajectories have occurred in the context of a strong public healthcare system that remains committed to the principle of universal access, notwithstanding having undergone recent reform.

It is reassuring, therefore, to learn that our overall return on investment in the health sector is still comparatively good value internationally.[4] However, satisfying public expectations in a landscape that is both epidemiologically and technologically vastly different to the one in which free hospital care regardless of means became entrenched public policy is becoming increasingly difficult. For example, the Queensland Government currently spends around a quarter of its total budget on the health portfolio and this expenditure is growing at close to 10% per annum in nominal terms just to keep pace with existing expectations. Clarity around the underlying drivers of this growth seems fundamental to mature debate about how we might place publicly funded healthcare on a more sustainable foundation.

The 'burden of disease' methodology introduced by Professors Christopher Murray and Alan Lopez in the early 1990s represents a useful platform from which to develop these and other

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1 From an economic perspective, total spending on health is sustainable so long as the value it produces exceeds the opportunity cost. In a growing economy, health spending can grow at a faster rate than other sectors of the economy without necessarily compromising economic sustainability.

2 Estimate based on data to 2009 from Australian Social Trends, Mar 2011 (Cat. no. 4102.0), Australian Bureau of Statistics, Canberra, 2011, viewed 21 August 2011, http://www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/4102.0Main+Features10Mar+2011.

3 Estimate based on data to 2008-09 from Health expenditure data cubes, Australian Insititute of Health and Welfare, Canberra, 2011, viewed 21 August 2011, http://www.aihw.gov.au/expenditure-data/.

4 Banks, G, *Health costs and policy in an ageing Australia*, in Health Policy Oration, Menzies Centre for Health Policy, John Curtin School of Medical Research, Australian National University, Canberra, 26 June 2008.

insights. However, it is often portrayed in terms of its theoretical contributions to the field of summary measures of population health,[5] or as part of an agenda for setting priorities on behalf of others.[6] While these characterisations may well have been effective at establishing the methods as an internationally recognised set of techniques for quantifying health loss in particular ways, arguably they have also created the impression 20 years on that, in general, the approach has limited value beyond what is commonly considered the domain of public health.

The central thesis I intend developing here is that such an impression with respect to the underlying conceptual framework is unfounded; however, for this possibility to achieve more general acceptance, a substantial reinterpretation of Murray and Lopez's original contributions may be required. In this chapter I attempt to lay the groundwork for such a proposition by outlining what I believe have been basic impediments to employing the methods for other than a limited range of applications.[7] In the remaining chapters I seek to illustrate how these apparent constraints can, in fact, be overcome. While some of the examples I will be presenting may appear inconsistent with the existing literature on 'burden of disease', each has as its basis the core concepts first proposed by Murray and Lopez.

Conceptual framework

Superficially at least, the conceptual framework that provides the foundation for the following chapters reflects the underlying structure of the International Classification of Diseases (ICD),[8] a hierarchical nomenclature for attributing all causes of illness in a population on a categorical as opposed to a counterfactual basis. In other words, each cause of health loss has a place in a mutually exclusive and exhaustive list of all possible causes. Counterfactual attribution, on the other hand, is more complex, at least in terms of presentation, in that outcomes are not necessarily mutually exclusive. In epidemiology, counterfactual attribution is where the impact of a causal agent (e.g. diabetes) on an outcome of interest (e.g. mortality) is assessed by removing its influence from the analysis. This

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5 Murray, C, Salomon, J, Mathers, C and Lopez, A, eds, *Summary measures of population health: concepts, ethics, measurement and applications*, World Health Organization, Geneva, 2002.

6 Lopez, AD, Mathers, CD, Ezzati, M, Jamison, DT and Murray, CJL, eds, *Global burden of disease and risk factors*, Oxford University Press and the World Bank, New York, 2006.

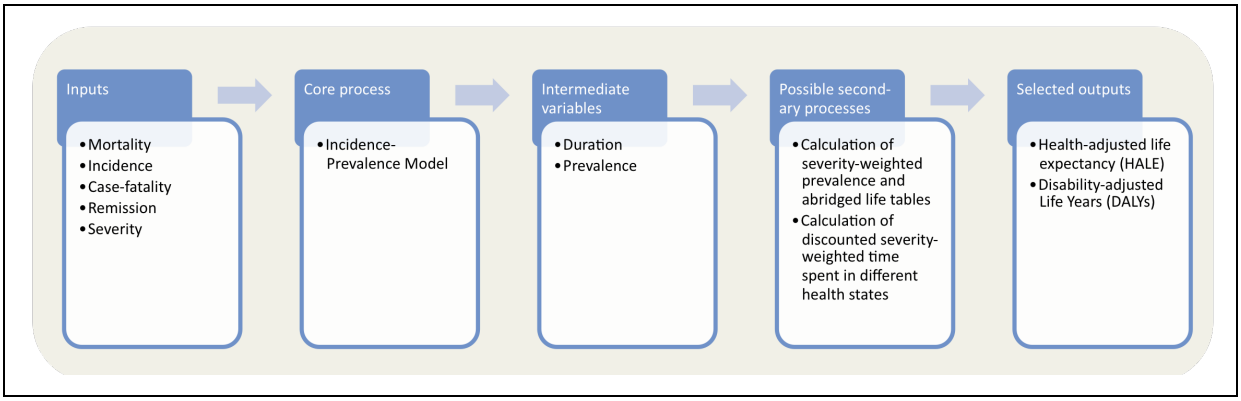
7 I make this observation having worked on a number of 'burden of disease' projects, details of which are provided in Appendix A.

8 International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, World Health Organization, Geneva, 1990, viewed 16 August 2011, http://www.who.int/classifications/icd/en/.

approach plays an important role in several processing aspects of the framework, one of which is explored in Chapter 3. For the moment though, the implications of counterfactual attribution can be put to one side.

More central to the general thesis being developed here is the proposition that the main elements of the framework are best understood as a series of transformative processes that ultimately depends on only a handful of parameters: mortality (as a population rate); incidence, case-fatality and remission (all as hazards); and severity (as a composite weight ranging from 0 to 1). From these five inputs, all but one of which is epidemiological, every other framework parameter that might be of interest can be derived. In this sense, the core of the framework is essentially a descriptive epidemiological exercise in the tradition of the seventeenth century pioneer of population health statistics, John Graunt.[9] The conceptual model underpinning this understanding is illustrated in terms of the essential information flows and processes depicted in Figure 1.

Figure 1: Information flows and processes within the Murray and Lopez framework



One process in this figure is labelled a core process due to its fundamental transformative function. First introduced in the context of 'burden of disease' as the 'Harvard Incidence-Prevalence Model' (hereafter, the IPM),[10] this process formalises the mathematical relationship between the hazard parameters identified above (the *input* parameters) and prevalence and average duration, parameters that, depending on the purpose of the analysis, can be regarded as either inputs for further processes (that is, *intermediate* parameters) or as outputs in their own right. Murray and Lopez's call for the rigorous application of the IPM to

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[9] I am indebted to Dr Chris Bain for pointing out this link to me.

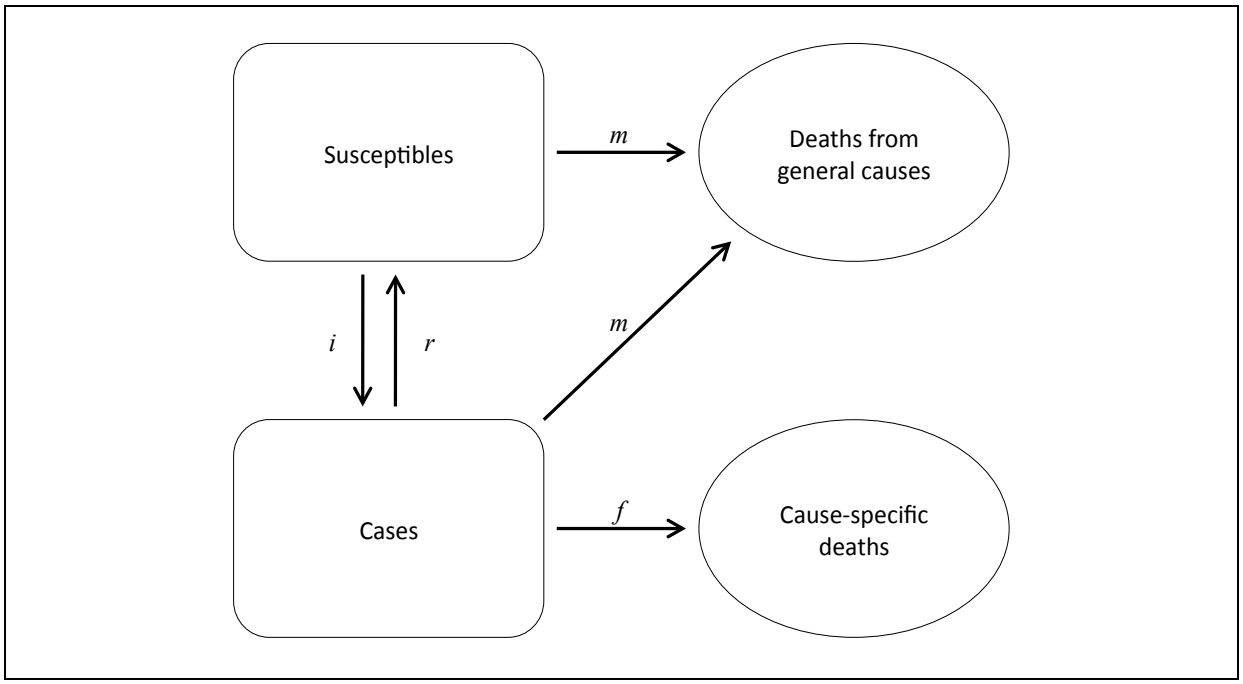
[10] Murray, CJ and Lopez, AD, "Quantifying disability: data, methods and results", *Bull World Health Organ*, 1994, vol. 72(3), pp. 481-94.

expose inconsistencies in epidemiological data is arguably one of their more provocative challenges to those with an interest in population health statistics.

Determining the precise origins of the IPM is difficult because reference to possible intellectual antecedents[11] is absent from the relevant literature. Nevertheless, a reasonably well-developed discussion of its conceptual basis appears in a series of books on the Global Burden of Disease 1990 study (GBD 1990) edited by Murray and Lopez for the World Bank and the World Health Organization.[12] Important to this discussion is the diagrammatic representation in Figure 2, which the authors explain as follows:

> Susceptibles in a population are assumed to be at risk of incurring a condition at rate *i* and can die at a general mortality rate *m*. Prevalent cases of the condition can remit at rate *r*, die from general causes at the same rate as the susceptibles *m*, or die from cause-specific mortality from the condition at rate *f*.[13]

Figure 2: The Incidence-Prevalence Model (IPM), as introduced by Murray and Lopez in 1996[14]



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11 e.g. Freeman, J and Hutchison, GB, "Prevalence, incidence and duration", *Am J Epidemiol*, 1980, vol. 112(5), pp. 707-23; Leske, MC, Ederer, F and Podgor, M, "Estimating incidence from age-specific prevalence in glaucoma", *Am J Epidemiol*, 1981, vol. 113(5), pp. 606-13; O'Neill, T, Tallis, G and Leppard, P, "The epidemiology of a disease using hazard functions", *Australian Journal of Statistics*, 1985, vol. 27(3), pp. 283-97; Preston, SH, "Relations among standard epidemiologic measures in a population", *Am J Epidemiol*, 1987, vol. 126(2), pp. 336-45.

12 Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I.*, Harvard School of Public Health on behalf of WHO and the World Bank, Cambridge, 1996.

13 ibid.

14 ibid.

In practice, the number of numerical values that must be analysed on the basis of this model can be large. Typical applications of the framework are stratified at least by cause, age and sex, but other dimensions have also been considered, such as time, socioeconomic status, geography and ethnicity. While the addition of extra dimensions undoubtedly makes the outputs more appealing to policy-makers, it also increases the number of values required as inputs and, ultimately, the number of values that must be processed and stored as intermediate parameters and outputs. However, since many of the calculations used to derive these values are identical across dimensions, the addition of extra dimensions can increase processing time or analytical load but should not necessarily imply an increase in underlying complexity.

History

From a purely practical perspective the history of the framework can usefully be divided into several distinct but possibly overlapping phases. The first phase undoubtedly begins with the GBD 1990 and is characterised by its focus on those parameters most closely related to the outputs of immediate interest. In the case of disability-adjusted life years (DALYs), arguably perceived by many as the *raison d'être* of the framework, these are mortality (absolute number of deaths using categorical attribution), incidence (cases), duration (average time incident cases spend as prevalent cases) and severity. In the case of health-adjusted life expectancy (HALE) when calculated using the Sullivan method,[15] they are mortality (population rate), prevalence (population proportion) and severity.[16] In both cases, the two inputs less proximal to the outputs (i.e. case-fatality and remission) are not useful in themselves, and tend not to be retained consistently, if at all, in an accessible manner. Other parameters not yet discussed (e.g. relative risk and attributable mortality) also tend not to be readily accessible. Typical information flows and processes characteristic of this phase are depicted in Figure 3.

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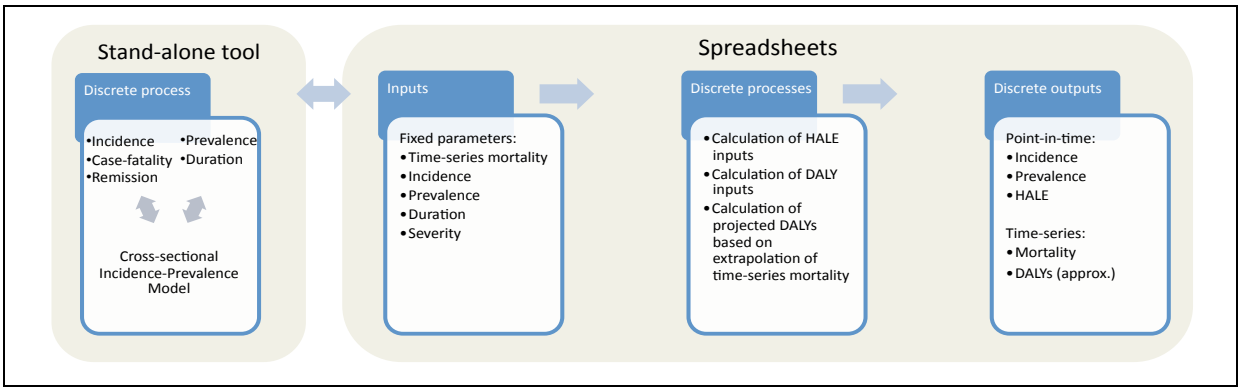
[15] Sullivan, DF, "A single index of mortality and morbidity", *HSMHA Health Rep*, 1971, vol. 86(4), pp. 347-54.

[16] According to Ass. Prof Jan Barendregt, HALE can also be calculated in a multi-state life table by using incidence and mortality as inputs.

Figure 3: Information flows and processes characteristic of phase one



A key limitation of this approach is reflected in the difficulties Dr Colin Mathers and others (including myself) experienced while attempting to replicate some of calculations underlying GBD 1990, despite having access to much of the original material.[17] In part, these difficulties were due to incomplete documentation, with only three of the planned ten volumes in the GBD 1990 series reaching publication.[18] However, they were exacerbated by Murray and Lopez's extensive reliance on a software implementation of the IPM called Dismod,[19] a critical shortcoming of which was the absence of a mechanism for copying or exporting values for the input and intermediate variables into other software environments. Instead, the only way of preserving these parameters outside of the tool was to transcribe the values from the screen or print and store them on paper.

A further limitation relates to the number of dimensions for which the application of the IPM is perceived as being feasible. Again, the GBD 1990 is useful for illustrating this point. For each of the eight world regions considered in their analysis, Murray and Lopez used Dismod to estimate values for the intermediate variables identified above, which they combined with

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17 For example, Begg, S and Tomijima, N, *Global burden of injury in the year 2000: an overview of methods*, World Health Organization, Geneva, 2003; Truelsen, T, Begg, S and Mathers, C, *The global burden of cerebrovascular disease*, World Health Organization, Geneva, 2003.

18 Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I*; Murray, CJL and Lopez, AD, eds, *Global Health Statistics: a compendium of incidence, prevalence and mortality estimates for over 200 conditions. Vol II.*, Harvard School of Public Health on behalf of WHO and the World Bank, Cambridge, 1996; Murray, CJL and Lopez, AD, *Health dimensions of sex and reproduction : the global burden of sexually transmitted diseases, HIV, maternal conditions, perinatal disorders, and congenital anomalies*, Harvard School of Public Health on behalf of WHO and the World Bank, Cambridge, 1998. This series provides the most detailed account of the framework these authors used to describe global health in 1990 across eight regions of the world for the GBD 1990 study.

19 Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I.*

input variables in spreadsheets to calculate various outputs of interest.[20] They also estimated deaths and DALYs for future years, although in this case not by using Dismod.[21] Instead they projected trends in mortality, from which they inferred trends in DALYs. Thus they report on alternative future scenarios for deaths and DALYs but not on trends in other potentially useful framework parameters, such as incidence, prevalence, prevalent disability or HALE. Professor Theo Vos and I replicated this approach in one of the first applications of the framework in Australia,[22] which we undertook in collaboration with Mathers, who at the time was employed by the Australian Institute of Health and Welfare (AIHW).[23]

Yet another limitation relates to the use of spreadsheets as the primary analytical tool of choice. In the absence of careful planning, this can make managing information with more than several dimensions unwieldy and inefficient, thus increasing perceived complexity and the likelihood of errors due to incorrect or out of date cell references. This point is illustrated by an analysis for the state of Victoria in which Vos and Ms Anne Magnus used relativities between broad population groupings to disaggregate state-level estimates of incidence, prevalence and duration to seventy-eight subpopulations.[24] From these disaggregations they calculated subpopulation-level estimates of DALYs and HALE in spreadsheets. However, an examination of the published results shows that the sum of the subpopulation-level DALYs does not add to the previously published estimates of total DALYs for the state.[25] At the time the project material revealed that this discrepancy was due to systematic spreadsheet errors.

In this history the first phase has no distinct end. However, a second phase arguably begins with the first critical evaluation of how best to apply the underlying concepts of the

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20 Murray, CJ and Lopez, AD, "Quantifying disability: data, methods and results"; World Bank, *World Development Report: Investing in Health*, Oxford University Press, New York, 1993.

21 Murray, CJ and Lopez, AD, "Alternative projections of mortality and disability by cause 1990-2020: Global Burden of Disease Study", *Lancet*, 1997, vol. 349(9064), pp. 1498-504; Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I.*

22 See Vos, T and Begg, S, *Victorian Burden of Disease Study: Morbidity*, Public Health Division, Victorian Department of Human Services, Melbourne, 1999; Vos, T and Begg, S, *Victorian Burden of Disease Study: Mortality*, Public Health Division, Victorian Department of Human Services, Melbourne, 1999.

23 See Mathers, C, Vos, T and Stevenson, C, *The burden of disease and injury in Australia*, (Cat No. PHE 17), Australian Institute of Health and Welfare, Canberra, 1999; Mathers, CD, Vos, ET, Stevenson, CE and Begg, SJ, "The Australian Burden of Disease Study: measuring the loss of health from diseases, injuries and risk factors", *Med J Aust*, 2000, vol. 172(12), pp. 592-6; Mathers, CD, Vos, ET, Stevenson, CE and Begg, SJ, "The burden of disease and injury in Australia", *Bull World Health Organ*, 2001, vol. 79(11), pp. 1076-84.

24 Burden of Disease (BoD) - LGAs & regions 1996, Department of Health Victoria, Melbourne, 2011, viewed 7 August 2011, http://www.health.vic.gov.au/healthstatus/composite/bod/bod96-index.htm.

25 *cf.* Vos, T and Begg, S, *Victorian Burden of Disease Study: Morbidity*. These results received substantial media coverage at the time, e.g. Toy, M, "Our sickest suburbs", *The Age,* 17 January 2001 2001, p. p1.

framework in the context of a large-scale project. Led by Mathers at WHO, the purpose of this evaluation was to update estimates from GBD 1990 as part of an ongoing research agenda known at the time as GBD 2000.[26] To facilitate this update WHO commissioned a second implementation of the IPM called Dismod II, which Mathers' team used extensively to derive values for the intermediate variables from sets of inputs relating to seventeen world regions. What makes this approach distinct from phase one is that Mathers transferred values for selected parameters from spreadsheets to a structured processing environment containing country-specific information on mortality. In so doing, Mathers was then able to interpolate values for 191 member states of the United Nations and extrapolate these values forward in time with more up-to-date mortality information as it became available.[27] This approach provided the basis for a time-series of country-level HALE estimates, which appear as appendix tables in the *World Health Report* series from 2001 to 2004.[28]

Figure 4 attempts to represent the essential elements of this phase in the development of the framework. The main advantages were that it allowed for time-series outputs within an efficient and flexible environment, and maintained a degree of consistency between the intermediate variables and the output variables, at least in the short term. However, because not all of the inputs indentified in Figure 1 were transferred from spreadsheets to the structured possessing environment, Mathers could not use this series of processes to maintain consistency between the inputs and intermediate parameters. This represents a key limitation over the medium term.[29]

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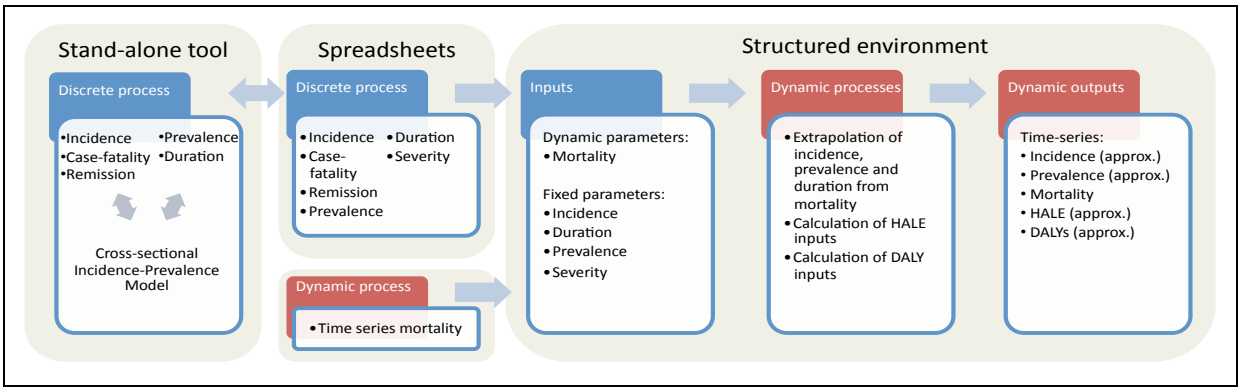
[26] Mathers, C, Salomon, J, Ezzati, M, Begg, S, Vander Hoorn, S and Lopez, A, "Sensitivity and uncertainty analyses for burden of disease and risk factor estimates", in Lopez, A, Mathers, C, Ezzati, M, Jamison, D and Murray, C, eds, *Global burden of disease and risk factors*, The World Bank and Oxford University Press, Washington, 2006.

[27] The routines for achieving these calculations were collectively known as the "DALYNATOR", the code for which was written by Mr Niels Tomijima.

[28] WHO, *World Health Report 2001- Mental Health: New understanding, New Hope.*, World Health Organization, Geneva, 2001; WHO, *World Health Report 2002- Reducing Risks, Promoting Healthy Life*, World Health Organization, Geneva, 2002; WHO, *World Health Report 2003 - Shaping the future*, World Health Organization, Geneva, 2003; WHO, *World Health Report 2004 - Changing history*, World Health Organization, Geneva, 2004.

[29] It appears these implications had not been anticipated when designing the spreadsheets since the primary objective was to disaggregate to individual countries the immediate outputs of interest (in this case DALYs and HALE). While the distortions introduced by these methods were probably insignificant over the period reported on by WHO, the effects over the medium term would have been non-trivial.

Figure 4: Information flows and processes characteristic of phase two



It is worth briefly making a few observations about the version of the IPM Mathers had to work with. Within the context of this discussion, the key advantage of Dismod II over its predecessor was the ability to store the inputs and calculated intermediate parameters in an accessible database format, and the inclusion of a function that allowed this information to be exported into various other formats, including spreadsheets. This increased the chance of preserving key input parameters over time. It also incorporated a goal-seeking algorithm for working with a larger combination of inputs than just incidence, case-fatality and remission (i, f and r using the notation in Figure 2 — the only inputs accepted by its predecessor) and the ability to transfer inputs to different populations for further processing, both functions that increased its efficiency. In addition, the tool included various enhancements not relevant to this discussion, including the ability to manipulate inputs prior to processing.

However, none of Dismod II's functions, including the core IPM transformation, could be accessed via internal scripts, nor could they be initiated externally via batch processing techniques. Moreover, calculated values for more than one time period could not be accessed, even though temporal trends in the inputs could be specified. In practice, therefore, the second version was identical to its predecessor in the sense that both tools operated as a stand-alone means for manually applying the IPM model one dimension at a time. Thus neither tool was particularly suited to maintaining consistency between inputs and intermediate parameters across multiple dimensions, as might occur in large-scale implementations of the framework, especially if inputs were to be updated on a regular basis.

My reason for considering these issues arose after joining Mathers' team in mid 2002. One discussion at the time focussed on whether it would be possible to maintain consistency between framework parameters by developing an interface between the inputs identified in Figure 1 and an automation of the equations underlying the IPM as described by Barendregt

and colleagues.[30] Exploring the feasibility of such an approach required access to a set of inputs across a reasonably large number of dimensions, which Mathers was attempting to collect from the many researchers who had contributed to GBD 2000. Translating the equations into a set of workable routines was relatively straightforward. However, we were unable to develop the idea beyond the proof-of-concept stage, in part because of difficulties obtaining a complete set of inputs, but also because the problem of divergence inherent in the existing system was unlikely to be material, at least in the short term.

Thus the opportunity to explore the practical implications of this approach did not arise until late 2003 when Lopez himself offered me the opportunity to coordinate an update of the national analysis Mathers had undertaken for Australia. The overall analysis had multiple aims, the reasons for which are not relevant to the present discussion. In terms of the history being portrayed here, however, one particular challenge was conceptually identical to meeting the information needs of the World Health Report series. This presented itself in the third of five funding agreements for the project, which required us to,

> Make reasonable efforts to automate the calculation process of small area estimates in order to facilitate regular updates of estimates rather than special studies to be conducted every 5 years. As such, it should become part of routine surveillance task e.g. State Health Departments and AIHW.[31]

Aims and objectives

To set the scene for the following chapters I have recast the above task in terms of a case study, the aim of which was to make comprehensive health outcomes assessment a routine function of government by increasing the feasibility of implementing the Murray and Lopez framework on a regular basis. Implicit here is the assumption that there exists an appetite (at least among the project funders) for up-to-date information on health outcomes to inform government activity. Figure 5 is an attempt to encapsulate the essential elements of this vision.

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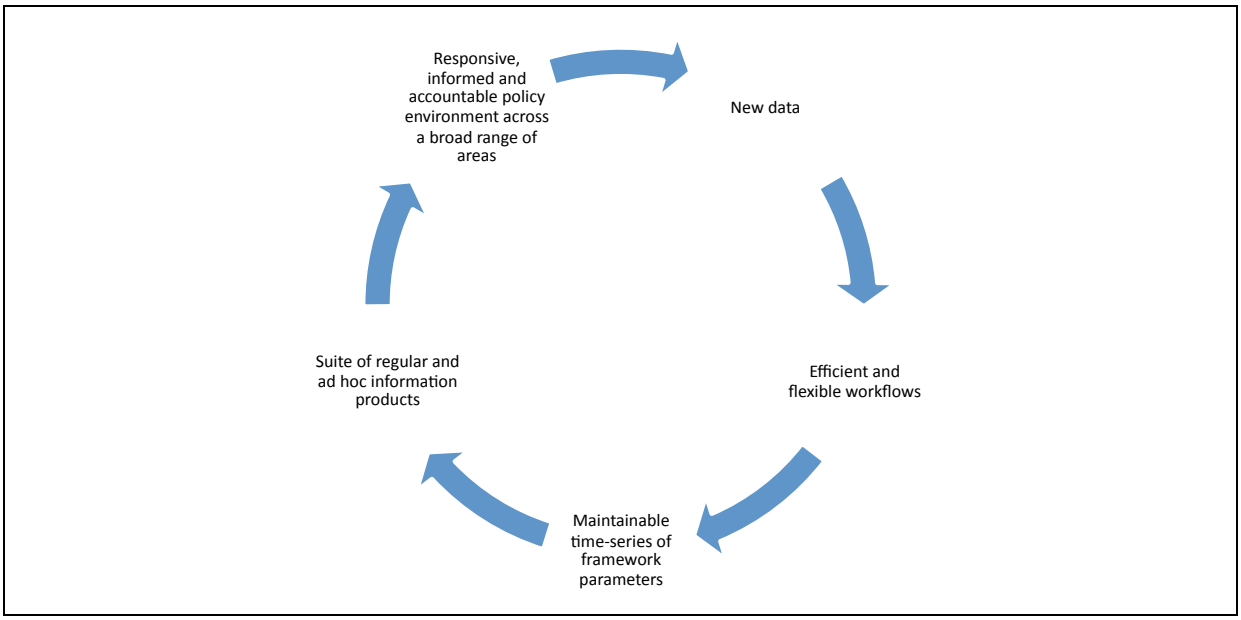
[30] Barendregt, JJ, Van Oortmarssen, GJ, Vos, T and Murray, CJ, "A generic model for the assessment of disease epidemiology: the computational basis of DisMod II", *Popul Health Metr*, 2003, vol. 1(1), pp. 4.

[31] Vos, ET, *Updating the Australian Burden of Disease: small areas estimates component study*, funded by Rural Health, Palliative Care and Health Strategies Branch, DOHA.

Figure 5: Selected aims and objectives of the case study



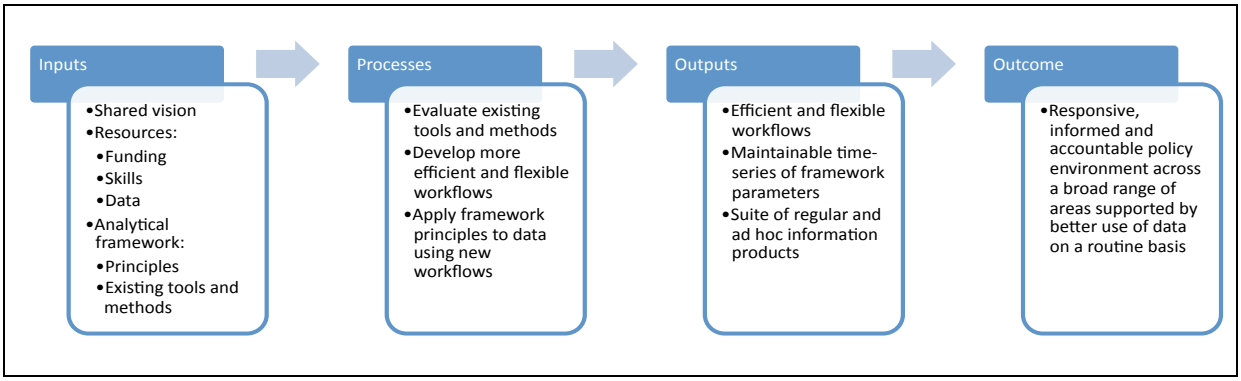
It is worth noting here that I have formulated Figure 5 on the basis of the terms of the funding agreement quoted above. As such, it would be incorrect to interpret it as a normative statement about the actual translation of knowledge into policy, which I acknowledge is an inherently political or 'non-linear' process.[32] Rather, my objective here is to characterise the case study as an attempt to optimise the potential for health outcomes assessment activities to contribute to this process.

As an analytical framework for the thesis as a whole I have attempted to assess the success or otherwise of the case study in facilitating the change implied in Figure 5 by applying simple program logic principles. Program logic encourages a focus on the 'inputs', 'processes' and 'outputs' required to achieve an intended 'outcome'. Consistent with my opening remarks, the desired outcome in this case can be defined in terms of a responsive, informed and accountable policy *environment* (as opposed to policy itself) supported by a routine population health monitoring capacity. Returning to Figure 5, the key outputs that might be expected to enable this transformation include efficient and flexible workflows, a contemporary time-series of framework parameters, and a suite of regular and ad hoc information products. Various inputs and processes are necessary for delivering these outputs, including a shared vision, the framework itself and sufficient resources to develop

[32] I am indebted to one of my examiners, Professor Martin McKee CBE, for pointing out the extensive literature on these aspects of policy formulation.

and apply enhanced workflows to available data. Figure 6 presents a more complete articulation of these relationships within a program logic framework.

Figure 6: Analysis of the case study within a program logic framework



Structure

The thesis itself is loosely structured around this framework and is divided into four parts. Part I comprises these introductory remarks and two additional chapters, the first of which is largely a technical discussion in which I critically evaluate existing tools and methods, or *inputs*, before proposing an alternative set of tools and workflows that I believe make the underlying conceptual framework more internally consistent, efficient and flexible in practice. The development and refinement of these alternatives represents a core activity or *process* of the case study, aspects of which my colleagues and I have already described in a report published by the AIHW in 2007.[33] That report therefore serves as an important stand-alone appendix both to the chapter and the thesis more generally. Chapter 3 presents a more in-depth examination of the IPM and illustrates some of its limitations in practice.

Parts II and III are structurally related and comprise a portfolio of information products or *outputs* that serve as links between the underlying processes and the project *outcome*. The particular outputs I have selected represent four papers—three of which have been published—that seek to address several areas of current policy concern: the dynamics of the chronic disease epidemic; a decomposition of the drivers of growth in health expenditure; and the magnitude and distribution of health problems, including the Indigenous health gap. Each paper has been allocated on the basis of whether it relied primarily on those methods outlined

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[33] Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*, (Cat. no.PHE 82), Australian Institute of Health and Welfare, Canberra, 2007. According to AIHW documentation, this report was the second most downloaded publication from the AIHW website, after Australia's Health 2006, including 48,000 times in the first few days after its release. By October 2009, the entire publication had been downloaded more than 322,000 times.

in Chapter 2 relating to projections, in which case it is presented in Part II, or those relating to subpopulation disaggregations, in which case it appears in Part III. While the published papers were prepared in collaboration with others, I have taken the liberty of adding comments pointing to specific processes where these were understated or overlooked altogether in the original text.

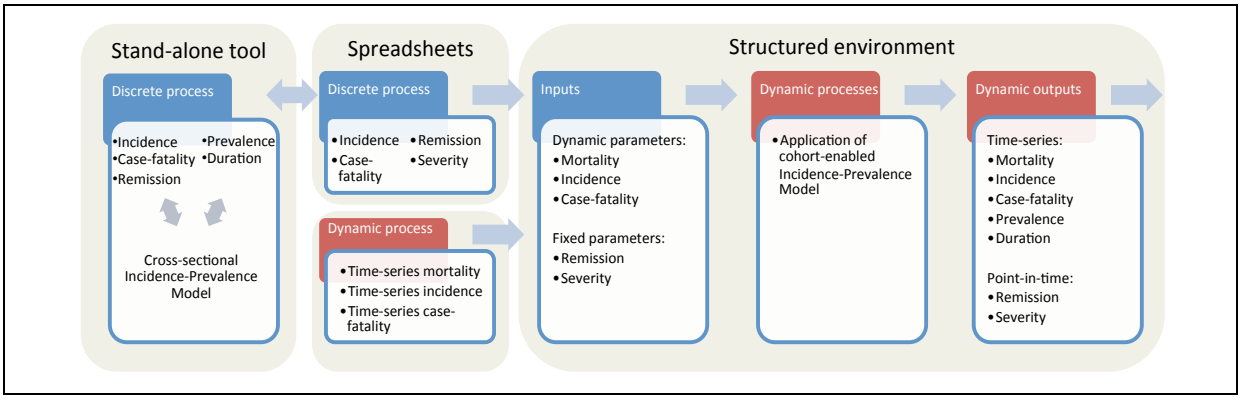
The final part is a commentary on the success or otherwise of the case study with respect to its intended outcome. In other words, to what extent did an alternative implementation model as represented by the set of tools and workflows presented in Chapter 2 facilitate government uptake of the Murray and Lopez framework in Australia? I approach this question from two angles: first in Chapter 8, from the perspective of various state-based implementations of the model, and then in the final chapter, from a national perspective.

Chapter 2: Critical evaluation of typical workflows and proposed alternatives

As mentioned in the opening chapter, the most critical limitation of both Dismod II and its predecessor in the context of this thesis is the inability to automate the core IPM transformation function across multiple dimensions. In the past, the main alternative was to adopt methods that either compromise the mathematical integrity or consistency between each of the framework parameters, or omit certain parameters altogether. Such outcomes are clearly undesirable for certain purposes and, arguably, limit the appeal of the conceptual framework proposed by Murray and Lopez for other than a restricted range of applications.

My objectives in this chapter, therefore, were twofold: first, to show how it is possible to incorporate the IPM into a set of workflows that not only maintains consistency across a complete set of framework parameters but, more importantly, achieves this across several dimensions fundamental to population health statistics (i.e. time, place and person); and second, to introduce the secondary problem of comorbidity and to illustrate how this too can be resolved within the context of the proposed workflows. The underlying conceptual model is illustrated in terms of the information flows and processes depicted in Figure 7, which have their origins in discussions with Mathers and colleagues at WHO. The unique and defining characteristic is its focus on the processes identified in Figure 1 of the previous chapter as core, rather than on possible secondary processes, such as the calculation of DALYs or HALE.

Figure 7: Information flows and processes adopted in the case study



To implement this approach one requires a set of working rules that does not inhibit existing work practices (e.g. creating cross-sectional epidemiological models using spreadsheets and

Dismod II) where there is no better alternative, but that ensures the information generated through such activities is accurately passed to a structured environment for further processing.

In the context of the case study, the following protocol was both workable and not overly restrictive:

1. Model each health problem such that each component sequela can be explicitly identified;
2. Estimate a complete set of transition hazards (i.e. incidence, remission and case-fatality) for each sequela, either in Dismod II or by other means; and
3. Record transition hazards and a relevant severity weight for each sequela in a predetermined format within the relevant spreadsheet.

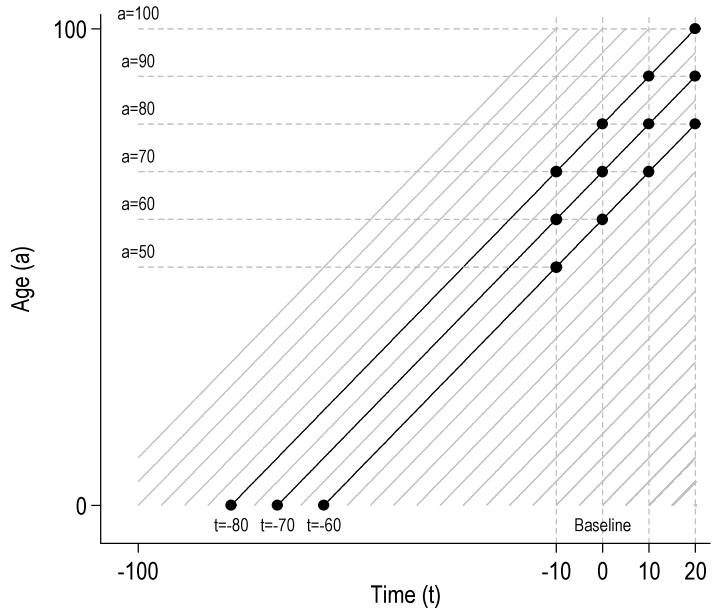
These workflows enabled me to construct a database of cross-sectional parameters that served as inputs for the methods described in the remainder of the chapter. An important aspect of the case study not revisited here are the sources of data and modelling assumptions for each of the 180 individual disease and injury models developed as part of the above activities. These details are available elsewhere.[34]

Cohort-enabled IPM

Figure 8 provides a conceptual model for an alternative implementation of the IPM that overcomes Dismod II's limitations with respect to the time dimension (i.e. the inability to access calculated values for more than one time period and the inability to automate the core IPM transformation function across multiple dimensions). In this model, values for those parameters that might be of interest for secondary processes are derived by determining complete epidemiological representations of causes on a cohort as opposed to a cross-sectional basis. Using the notation in Figure 2 of Chapter 1, such a model requires for each cause and associated sequela the complete specification of the parameters m and i, f and r at each preceding age a and time t relevant to the cross-section t of interest. For this reason I have called it the Cohort-enabled Incidence-Prevalence Model.

[34] See ibid.

Figure 8: Conceptual model for the cohort-enabled Incidence-Prevalence Model (IPM)



I have implemented the calculations underlying this model in Mata using the equations set out by Barendregt and colleagues.[35] Mata is a matrix-programming environment built into the Stata statistics and data management software package.[36] The model consists of the following Mata subroutines: `mata_cohort-IPM`, which executes the underlying calculations for each age cohort as it ages over time; `mata_IPM_calc`, which performs most of the actual calculations, except duration; and `mata_dur_calc`, which performs the duration calculations. These routines are called by a Stata ado program `cohort-IPM`, which, if installed correctly, can be accessed from the command line like any other Stata command. The last two subroutines are also used elsewhere, as discussed shortly.

`cohort-IPM` is fast and 'byable', meaning the user can specify, in theory, any number of stratifying variables within which the calculations are to be repeated. However, in practice the actual number of dimensions is limited by memory considerations, although the minimum requirement is a variable that stratifies the data by cause and associated sequela. For convenience, hereafter I use the right-hand subscript k to denote a cause within a complete set of K causes.

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[35] Barendregt, JJ, Van Oortmarssen, GJ, Vos, T and Murray, CJ, "A generic model for the assessment of disease epidemiology: the computational basis of DisMod II".

[36] StataCorp, *Stata Statistical Software: Release 11*, StataCorp LP, College Station, TX, 2009.

By choosing the `by` option, `cohort-IPM` automatically checks whether the variables specified in the `by` statement uniquely identify the data across the following required input variables: *age* (0, 1, 5, 10…100, although other age groupings could be implemented), *year* (1993, 1994…2023, although other calculation periods could be implemented), *sex* (1=males). In addition, `cohort-IPM` requires *i*, *f* and *r*, all expressed as a hazard per unit of population, and a valid filename and path to a data file containing all-cause mortality (essentially a hazard, which I denoted as m_K) by age, sex and year using the same categorisations as the inputs. The code that enables `mata_IPM_calc` and `mata_dur_calc` is at Appendix B, while the code that enables `cohort-IPM` is at Appendix C.

In practice, various assumptions can simplify specifying the inputs required by `cohort-IPM`. For most chronic diseases, *i* and *f* are the primary drivers of changes in observed cause-specific mortality (which I denoted as m_k), while *r* plays little if any role. This is because the 'hazard' of recovering from a chronic disease is, by definition, small. It follows that a complete epidemiological model in this instance can be back-cast from cross-sectional estimates of *i* and *f* by making plausible assumptions about the relative contribution of these hazards to observed changes in mortality. This reasoning applies equally to projections if one is willing to make predictions about mortality into the future.

The above general approach depends on complete sex-specific vectors of mortality for each cause and birth cohort over the calculation period. Fortunately for the case study, Australia has a high quality vital registration system by international standards and, with several notable exceptions, observable trends in mortality provide a reasonably consistent (albeit indirect) source of information on changes in the incidence of diseases and injuries over the last century, notwithstanding frequent revisions to the ICD over this period. Nevertheless, these revisions make it difficult to analyse cause-specific trends in mortality over more than a decade or so, except at very broad levels of aggregation. As such, a reasonable compromise between specificity and the length of the data series is to focus on the period from 1979,

when the ninth version of the ICD was introduced for the coding of mortality data in Australia.[37]

In the case study, I derived plausible sex-specific vectors of mortality for each cause and birth cohort over $[t \mid -24 \leq t \leq 20]$ where $t = 0$ represents the year 2003 (the most recent year of available data at the time) as follows. For each age and sex group, observed m_K over $[t \mid -24 \leq t \leq 0]$ were extrapolated to $[t \mid 1 \leq t \leq 20]$ in a Poisson regression model that used t as a predictor and population as an offset variable. Observed vectors of m_k over $[t \mid -24 \leq t \leq 0]$ were then aggregated into 51 clinically meaningful groups (denoted by $k+$), and multinomial logistic regression was used to predict changes in each m_{k+} proportional to m_K as a consequence of changes in $ln(m_K)$. This model was used to extrapolate each m_{k+} for $[t \mid 1 \leq t \leq 20]$ using projected $ln(m_K)$ as a predictor. Average distributions of m_k within each m_{k+} over $[t \mid -2 \leq t \leq 0]$ were then applied to projected m_{k+} to derive projected vectors of m_k over $[t \mid 1 \leq t \leq 20]$. In the absence of more detailed historic trends, values of m_k over $[t \mid -110 \leq t \leq -25]$ were assumed to be equivalent to values for $t = -24$.

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

This approach also requires complete vectors of i, f and r for each cause and birth cohort over the calculation period. In the case study, I derived these from the time-series vectors of m over $[t \mid -110 \leq t \leq 20]$ described above and the cross-sectional vectors for i, f and r from the database described above. I combined these using the simplifying set of assumptions set out in Table 1, which stem from a number of observations:

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[37] It should be noted that due to changes in coding rules and practices associated with the ICD, even mortality trends from 1979 are unreliable indicators of trends in incidence for certain diseases (most notably pneumonia and diabetes).

1. For a number of causes (e.g. cancers, COPD, alcohol-related conditions, road traffic accidents, falls, suicide and homicide), past trends in observed mortality are most likely due to changes in incidence.
2. For cardiovascular diseases (CVD), there is evidence that past trends in observed mortality are the result of changes in both incidence and case-fatality.[38]
3. For Type 2 diabetes (T2DM), trends in observed mortality are likely to be unreliable due to changes in diagnostic criteria and coding practices over time. A feasible alternative in this instance is to translate historical trends in measured body mass index (BMI), the main risk factor for T2DM, into changes in incidence using established risk attribution methods[39] and published regression models of the distribution of BMI on age, birth cohort and sex.[40] In the absence of direct measures of case-fatality, it seems reasonable to assume that half the mortality in diabetics is most likely due to vascular causes. The methods arising from these assumptions are set out in greater details in Chapter 8.
4. For other causes, a reasonable default assumption is no trend in any of the input hazards.

Table 1: Assumptions in the case study for extrapolating baseline input hazards

Cause	$\%\Delta i_k$	$\%\Delta f_k$	$\%\Delta r_k$
Cancers, COPD, alcohol-related conditions, road traffic accidents, falls, suicide and homicide	$\%\Delta m_k$	No trend	No trend
Cardiovascular diseases (CVD)	$\%\Delta m_{CVD} \times 58\%$	$\%\Delta m_{CVD} \times 42\%$	No trend
Type 2 diabetes	$\%\Delta i_{BMI}$	$\%\Delta m_{CVD} \times 42\% \times 50\%$	No trend
All other causes	No trend	No trend	No trend

From these vectors of m_K, i, f and r I estimated vectors of average duration d and prevalence p for each cause, sex, and birth cohort over $[t \mid -110 \leq t \leq 20]$ and $[a \mid 0 \leq a \leq 100]$ using `cohort-IPM`. This approach is analogous to dynamic population modelling[41] and has several advantages over the projection methods mentioned in the previous chapter. First, it provides an efficient method for deriving a complete set of framework parameters by age and

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38 Unal, B, Critchley, JA and Capewell, S, "Explaining the decline in coronary heart disease mortality in England and Wales between 1981 and 2000", *Circulation*, 2004, vol. 109(9), pp. 1101-7.

39 Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 1*, World Health Organization, Geneva, 2004.

40 Haby, MM, Markwick, A, Peeters, A, Shaw, J and Vos, T, "Future predictions of body mass index and overweight prevalence in Australia, 2005-2025", *Health Promot Int*, 2011.

41 Frauenthal, JC, "A dynamic model for human population growth", *Theor Popul Biol*, 1975, vol. 8(1), pp. 64-73.

sex for a large number of causes and time periods. Unlike other approaches, however, internally consistency between parameters is maintained. Second, it allows for incremental revisions of these parameters by updating selected inputs with new data as it becomes available. Finally, the underlying principles can be adapted to maintain internal consistency for dimensions other than time, an advantage I expand on below.

Subpopulation-enabled IPM

In addition to `cohort-IPM`, I developed an alternative implementation of the IPM to assist with maintaining internal consistency when estimating framework parameters for multiple subpopulations. The general approach is conceptually more straightforward than the one described in the previous section since it is based on a cross-sectional model; the underlying calculations are therefore the same as in `cohort-IPM` when no trends in hazard are specified. However, each of the hazards must nevertheless be fully specified for every subpopulation of interest.

A generic version of the model consists of the following Mata subroutines: `mata_subpop-IPM`, which executes the underlying calculations for each subpopulation; `mata_IPM_calc`, which performs most of the actual calculations, except duration; and `mata_dur_calc`, which performs the duration calculations. These routines are called by a Stata ado program `subpop-IPM`, which requires the same inputs as the cohort version upon which it is based, except that it requires a subpopulation variable as an input (instead of year) and a valid filename and path to a Stata data file containing m_K by age, sex and subpopulation (using the same categorisations as the inputs).

In the case study I implemented `subpop-IPM` in two versions: the first was designed to estimate framework parameters for a 15-cell matrix of subpopulations comprising three remoteness categories (major cities, regional areas and remote areas) by five socioeconomic quintiles. The second was designed to estimate framework parameters for two Indigenous populations (major cities and regional areas combined, and remote areas). The code that enables each version is at Appendix D and E, respectively.

While these versions are technically similar, their application within the context of the case study was conceptually quite different. In the first application a complete set of framework parameters for Australia in the year 2003 had already been established by the processes described above; the objective of `subpop-IPM`, therefore, was to maintain internal

consistency when disaggregating each of the parameters within the constraints imposed by this envelope.

Table 2: Data used to disaggregate *i* across jurisdictions, and 15 subpopulations within each jurisdiction

Cause	Jurisdictions	Subpopulations
Tuberculosis, other sexually transmitted diseases, HIV/AIDS, diarrhoeal diseases, septicaemia, hepatitis b (including hepatitis b related liver cancer and cirrhosis), trachoma, other infectious and parasitic diseases, lower respiratory tract infections, upper respiratory tract infections, otitis media, maternal haemorrhage, maternal sepsis, hypertensive disorders of pregnancy, obstructed labour, abortion, other maternal conditions, birth trauma and asphyxia, low birth weight, neonatal infections, protein-energy malnutrition, deficiency anaemia, other nutritional deficiencies, uterine myomas, benign neoplasms of meninges and brain, other benign neoplasms, type 1 diabetes, type 2 diabetes, haemolytic anaemia, other non-deficiency anaemia, cystic fibrosis, haemophilia, other endocrine and metabolic disorders, alcohol dependence and harmful use (including alcoholic cirrhosis), heroin or polydrug dependence and harmful use, benzodiazepine dependence and harmful use, cannabis dependence and harmful use, other drug dependence and harmful use, schizophrenia, anorexia nervosa, Alzheimer and other dementias, epilepsy, Parkinson's disease, multiple sclerosis, glaucoma-related blindness, cataract-related blindness, other nervous system and sense organ disorders, rheumatic heart disease, ischaemic heart disease, stroke, inflammatory heart disease, non-rheumatic valvular disease, aortic aneurysm, peripheral vascular disease, other cardiovascular disease, chronic obstructive pulmonary disease, other chronic respiratory diseases, cirrhosis of the liver (excluding alcoholic and hepatic cirrhosis), appendicitis, intestinal obstruction, diverticulitis, gallbladder and bile duct disease, pancreatitis, inflammatory bowel disease, vascular insufficiency bowel, other digestive system diseases, benign prostatic hypertrophy, other genitourinary diseases, ulcers, other skin diseases, slipped disc, other musculoskeletal diseases, congenital heart disease, cleft lip and/or palate, other digestive system malformations, other urogenital tract malformations (including polycystic renal failure), road traffic accidents, other transport accidents, poisoning, falls, fires, burns and scalds, drowning, sports injuries, natural and environmental factors, machinery accidents, suffocation and foreign bodies, surgical and medical misadventure, adverse effects of drugs in therapeutic use, cutting and piercing accidents, striking and crushing accidents, other unintentional injuries, suicide and self-inflicted injuries, homicide and violence, legal intervention and war	Hospital data	Hospital data
Syphilis, chlamydia, gonorrhoea, poliomyelitis, meningitis, Ross River virus, dengue, other arbovirus infection, hepatitis A, hepatitis C, malaria	NNDSS data	Hospital data
Anxiety and depression, personality disorders (isolated)	None	MHS
Bipolar disorder	Hospital data	MHS
Stomach cancer, colorectal cancer, pancreas cancer, lung cancer, melanoma, breast cancer, cervix cancer, corpus uteri cancer, ovary cancer, prostate cancer, testicular cancer, bladder cancer, kidney cancer, brain cancer, lymphoma, leukaemia	Cancer registry data	Mortality data
Other conditions arising in the perinatal period	Hospital data	Mortality data
Haemophilus influenzae type b (Hib),	NNDSS data	Mortality data
Mouth and oropharynx cancers, oesophagus cancer, liver cancer, gallbladder cancer, bone and connective tissue cancer, non-melanoma skin cancers, thyroid cancer, multiple myeloma, larynx cancer, eye cancer, other malignant neoplasms, motor-neuron disease, Huntington's chorea, muscular dystrophy, nephritis and nephrosis, renal agenesis	Mortality data	Mortality data
Autism spectrum disorders, migraine	Hospital data	NHS
Hypertensive heart disease, peptic ulcer disease	Mortality data	NHS
Attention-deficit hyperactivity disorder, macular degeneration, refractive errors, other vision loss, urinary incontinence, rheumatoid arthritis, osteoarthritis	None	NHS
Adult-onset hearing loss, asthma, back pain	SDAC	NHS
Other hepatitis	Hospital data	None
Diphtheria, whooping cough, tetanus, measles, rubella, Barmah Forest virus	NNDSS data	None
Bulimia nervosa, other eating disorders, other mental disorders, infertility, eczema, acne, psoriasis, occupational overuse syndrome, systemic lupus erythematosus, gout, anencephaly, spina bifida, anorectal atresia, oesophageal atresia, abdominal wall defect, Down syndrome, other chromosomal disorders, other congenital anomalies, dental caries, periodontal disease, edentulism, pulpitis, other oral conditions, sudden infant death syndrome, chronic fatigue syndrome	None	None

Notes: NHS denotes the National Health Survey 2001; SDAC denotes the Survey of Disability, Ageing and Carers 2003; MHS denotes the National Mental Health Survey 1997; NNDSS data denotes National Notifiable Diseases Surveillance System; Hospital data denotes the national hospital morbidity dataset for 2003; Mortality data denotes the national mortality dataset for 2003.

Disaggregation was achieved for each cause by first apportioning *m* and *i* across states and territories, and then across up to 15 subpopulations within each jurisdiction. In the case of *m*, mortality data was used to determine this apportionment. For *i*, preference was given to the data source underpinning the baseline model, as outlined in Table 2.[42] These subpopulation-specific vectors of *i* were combined with total population vectors of *f* and *r*, from which complete vectors of *d* and *p* for each subpopulation were estimated using `subpop-IPM`. Minor rescaling was performed if any total across subpopulations was not consistent with the corresponding estimate for the total population.

In the second application, the objective was to generate a complete set of framework parameters for the two Indigenous populations. Various strategies were used to derive m_K, *i*, *f* and *r* for each population, although, since there was no overall envelope of disability to work within, none involved disaggregation. Instead, the results were subtracted from the corresponding total population estimate.

In the case of m_K, an indirect demographic technique known as the Generalised Growth Balance (GGB) method determined the amount to be subtracted.[43] The GGB corrects for under-reporting, which is necessary when using mortality data relating to Indigenous people.

For the other hazards, in a relatively small number of cases individual models were developed to derive Indigenous-specific estimates of *i*, *f* and *r*. For the majority, however, *f* and *r* were assumed to be the same as the total population, while Indigenous to non-Indigenous relativities from a variety of datasets were applied to total population estimates of *i* to derive Indigenous-specific vectors of this parameter. Table 3 identifies for each cause the data source used to determine *i* either directly or via relativities, and also those causes for which assumptions with respect to additional parameters were also made.

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[42] These sources were only used to apportion the total number of incident cases between subpopulations for a particular cause, not the number itself (which should capture multiple episodes of illness).

[43] Hill, K, Barker, B and Vos, T, "Excess Indigenous mortality: are Indigenous Australians more severely disadvantaged than other Indigenous populations?", *Int J Epidemiol*, 2007, vol. 36(3), pp. 580-9.

Table 3: Data used to determine *i* for Indigenous populations

Cause	Source[b,c]
Chlamydia, gonorrhoea, whooping cough, Haemophilus influenzae type b, dengue, other arbovirus infection, hepatitis A, malaria, Ross River virus, Barmah Forest virus, hepatitis B[(a)], tuberculosis, measles	NNDSS data
HIV/AIDS[(a)]	HIV/AIDS data
Nephritis & nephrosis	Transplant Registry
Malignant neoplasms, nephritis & nephrosis	Mortality data
Low birth weight	Perinatal Data
Syphilis, other STDs, diarrhoeal diseases, meningitis, septicaemia, maternal haemorrhage, maternal sepsis[(a)], hypertensive disorders of pregnancy, obstructed labour, abortion[(a)], other maternal conditions, deficiency anaemia, cataract-related blindness[(a)], ischaemic heart disease[(a)], peripheral vascular disease[(a)], heart failure[(a)], chronic obstructive pulmonary disease[(a)], pancreatitis, tuberculosis, lower respiratory tract infections, upper respiratory tract infections, birth trauma & asphyxia[(a)], low birth weight[(a)], neonatal infections[(a)], uterine myomas, benign neoplasms of meninges and brain, type 2 diabetes[(a)], cannabis, other drug dependence, schizophrenia, haemolytic anaemia, other non-deficiency anaemia, cystic fibrosis, haemophilia, heroin or polydrug dependence, bipolar disorder, anorexia nervosa, bulimia nervosa, other eating disorders, multiple sclerosis, glaucoma-related blindness[(a)], refractive errors, stroke[(a)], hypertensive heart disease, aortic aneurysm, peptic ulcer disease, cirrhosis of the liver, appendicitis, intestinal obstruction, diverticulitis, gallbladder and bile duct disease, vascular insufficiency bowel, benign prostatic hypertrophy, other genitourinary diseases, psoriasis, ulcers, osteoarthritis, slipped disc, systemic lupus erythematosus, gout, other musculoskeletal diseases, anencephaly, spina bifida, congenital heart disease, cleft lip and/or palate, anorectal atresia, oesophageal atresia, other digestive system malformations, renal agenesis, other urogenital tract malformations, abdominal wall defect, pulpitis, chronic fatigue syndrome, unintentional injuries, intentional injuries	Hospital data
Otitis media[(a)]	BEACH
Stroke[(a)]	WA data
Trachoma[(a)], otitis media[(a)]	Eye data
Asthma[(a)], alcohol dependence[(a)], anxiety & depression[(a)], otitis media[(a)]	NHS
Anxiety & depression[(a)]	NSW survey & WA survey
Dental caries[(a)], periodontal disease, edentulism	Oral health survey
Dental caries[(a)]	SA dental survey
Measles, tetanus, trachoma[(a)], hepatitis B[(a)], type 2 diabetes[(a)], intellectual disability, epilepsy[(a)], autism and Asperger's	Epidemiological studies

Notes: (a) Denotes the model also included alternative assumptions for case-fatality, remission or severity. (b) Some causes incorporated data from multiple sources. (c) NNDSS data denotes the National Notifiable Diseases Surveillance System; HIV/AIDS data denotes the National HIV Database and National AIDS Registry; Transplant Registry denotes the Australia and New Zealand Dialysis and Transplant Registry; Mortality data denotes the national mortality dataset; Perinatal Data denotes the National Perinatal Data Collection; Hospital data denotes the national hospital morbidity dataset; BEACH denotes the Bettering the Evaluation And Care of Health collection; WA data denotes the Western Australian Data Linkage System; Eye data denotes the National Trachoma Eye Health Program 1976–78; NHS denotes the National Health Survey 2001 & 2004–05; NSW survey denotes the NSW Health Survey 1997–98; WA survey denotes the Western Australian Aboriginal Child Health Survey 2001–02; Oral health survey denote the 2004–06 National Oral Health Survey; SA dental survey denotes the South Australian Child and Adult Dental Health Survey 1999–01.

Table 4 identifies those causes for which *i*, *f* and *r* were assumed to be the same as the total population. In both cases, complete vectors of *d* and *p* for each subpopulation were estimated using `subpop-IPM`. A number of causes were assumed to be absent in the Indigenous population such as trachoma (for the non-remote population only), diphtheria, poliomyelitis,

rubella, other conditions arising in the perinatal period, and occupational overuse syndrome. Further details regarding these methods are provided in a separate report.[44]

Table 4: Causes for which *i, f and r* were assumed to be the same as the total population

Cause	Source
Other hepatitis, type 1 diabetes, benzodiazepine dependence, personality disorders (isolated), attention-deficit hyperactivity disorder, Alzheimer and other dementias, Parkinson's disease, motor-neuron disease, Huntington's chorea, muscular dystrophy, macular degeneration, adult-onset hearing loss, other vision loss, migraine, inflammatory bowel disease, urinary incontinence, infertility, eczema, acne, rheumatoid arthritis, back pain (acute and chronic)	As per the total Australian population

Comorbidity

My second objective in this chapter was to introduce the problems created by comorbidity and to illustrate how these can be resolved as a secondary process within the context of the workflows depicted in Figure 7. Comorbidity is when two or more health problems occur in a person simultaneously, either by chance or because the conditions are related to each other in some way. Independent comorbidity is where the probability of having multiple conditions at the same time equals the product of the probabilities for each condition. Dependent comorbidity, on the other hand, is where the probability of having multiple conditions is greater than the product of the probabilities for each condition, and occurs because of common causal pathways (for example common risk factors causing both diabetes and heart disease) or because one health problem may increase the risk of another.

Both types of comorbidity can be problematic for the conceptual framework proposed by Murray and Lopez,[45] particularly when the set of available severity weights is comprised of evaluations for each health state as it occurs independently from others. In the case study, the severity weights were mostly derived from two sources,[46] with extrapolations based on alternative methods in some cases. Nearly all of these weights were based on independent evaluations.

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[44] Vos, T, Barker, B, Stanley, L and Lopez, A, *The Burden of Disease and Injury in Aboriginal and Torres Strait Islander Peoples 2003*, School of Population Health, The University of Queensland, Brisbane, 2007.

[45] van Baal, PH, Hoeymans, N, Hoogenveen, RT, de Wit, GA and Westert, GP, "Disability weights for comorbidity and their influence on health-adjusted life expectancy", *Popul Health Metr*, 2006, vol. 4, pp. 1.

[46] Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I*; Stouthard, ME, Essink-Bot, M, Bonsel, GJ, Barendregt, JJ, Kramers, PGN, van de Water, HPA, Gunning-Schepers, LJ and van der Maas, PJ, *Disability weights for diseases in The Netherlands*, Department of Health, Erasmus University Rotterdam, Rotterdam, 1997.

The severity of a health state associated with two or more conditions in combination may not necessarily be the sum of the severity weights for each condition. In most cases, it is likely to be less than the sum. In others, there may be exacerbating effects on overall health of having the combination 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). However, the experience of the latter with profound deafness may be equal to or even more disabling than the summation approach indicates.

In an early response to this problem, Mathers proposed an adjustment that assumed health state valuations (that is, 1 minus the severity weight) are multiplicative, so that a combined weight for two conditions is more severe than the weight for either condition on its own but less than if the weights were simply added together. In this approach, the combined severity weight for causes $k = 1$ and $k = 2$ is given by,

$$comDW_{[1,2]} = 1 - (1 - DW_1) \times (1 - DW_2).$$

Equation 1

This can be generalised to n conditions thus,

$$comDW_{[1,n]} = 1 - \prod_{j=1}^{n} (1 - DW_j),$$

Equation 2

where $\prod$ denotes the product operator. Equation 2 has been extensively used to derive combined weights for comorbid conditions in a number of applications of the framework. Subsequent work indicates that, in the absence of anything else, the multiplicative approach to deriving composite weights is reasonably robust.[47]

Mathers' initial implementation derived individual weights consistent with these composite weights by leaving the weight for the most severe condition unchanged but adjusting the weight for the milder condition such that it equalled the composite weight minus the weight

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[47] Flanagan, W, McIntosh, CN, Le Petit, C and Berthelot, JM, "Deriving utility scores for co-morbid conditions: a test of the multiplicative model for combining individual condition scores", *Popul Health Metr*, 2006, vol. 4, pp. 13.

for the more severe condition. For example, for a person with symptomatic grade 2 osteoarthritis of the hip or knee ($DW_1 = 0.14$) and severe vision loss ($DW_2 = 0.43$), the combined weight for both conditions ($comDW_{[1,2]}$) is 0.51 and the adjusted weight for the osteoarthritis ($adjDW_2$) is 0.08. However, the 'adjusted' weight for severe vision loss remains unchanged (i.e. $adjDW_1 = DW_1$). These adjustments were calculated in a spreadsheet and manually fed back into multiple spreadsheets relating to individual disease and injury models.

Implicit in this approach is an assumption that the prevalence of a set of comorbid conditions is equal to the product of the individual prevalences of these conditions; in other words, that health problems occur independently of each other. Subsequent work by Mathers, Iburg and me demonstrated that correcting for dependence between groups of conditions has a non-trivial impact on comorbidity-adjusted severity weights and ultimately summary measures such as HALE and DALYs.[48]

In the context of the case study, the research team decided in consultation the project's steering committee that severity weights based on independent evaluations should be combined multiplicatively and in a way that corrected for dependency. The following discussion sets out an approach that is consistent with both principles.

Using basic set theory, I define the universe of every cause k of interest as the set A. The power set of A, denoted here as $\wp(A)$, is the set of all possible subsets of A, including the empty set and A itself; because A is finite with $|A| = n$ elements, $\wp(A)$ contains 2^n elements. In addition, I define B as a set in which each element corresponds to the largest element of A for which estimates of prevalence can be derived by age and sex from an element of set C. To construct B, I define C to be the set of available empirical data sources in which each corresponding element of B intersects with at least one other element from that set. The intersection between elements of B is important for capturing information on dependency between causes across empirical data sources.

From these definitions I construct, for each age and sex, a set based on the union of power sets for each element of B (which, for convenience, I denote as D) such that each element in the new set represents an estimate of prevalence from C expressed as a population probability

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[48] Mathers, CD, Iburg, KM and Begg, S, "Adjusting for dependent comorbidity in the calculation of healthy life expectancy", *Popul Health Metr*, 2006, vol. 4(1), pp. 4.

(i.e. 0 to 1). It follows that the super set that contains these sets represents the subset of $\wp(A)$ for which empirical probabilities are available by age and sex. This super set can be used in a series of Monte Carlo simulations to expose an artificial cohort reflecting the age and sex profile of the population of interest to conditional probabilities across the relevant subset of $\wp(A)$. Such a cohort can then be aggregated so as to derive a set of simulated age- and sex-specific estimates of prevalence p' for every element of D.

Estimating comorbidity-adjusted severity weights on the basis of these principles involves several steps. First, a partially adjusted severity weight for a particular cause, age and sex for any given element of D can be derived from Equation 2 using,

$$part\_adjDW_k = \frac{DW_k}{\sum_{j}^{|d|} DW_j} \times \left(1 - \prod_{j}^{|d|}\left(1 - DW_j\right)\right)$$

Equation 3

where d is an element of D. This approach differs somewhat from the one proposed by Mathers in that the attenuating effect of the adjustment is applied to each cause in proportion to its contribution to an additively derived composite weight, rather than to all but the most severe cause. While neither approach is empirically based, this algorithm is arguably less arbitrary.

To correctly combine the differential attenuations that exist across every combination of causes in which k occurs, $part\_adjDW_k$ is summed across elements of D in proportion to the corresponding p' for each element using,

$$adjDW_k = \frac{\sum_{j}^{|D|} part\_adjDW_{k,j} \times p'_j}{\sum_{j}^{|D|} p'_j}.$$

Equation 4

With these methods, each $adjDW_k$ accounts for dependent and independent comorbidity to the extent that this is reflected in the simulated cohort. Simulated estimates of prevalence could obviously be substituted with empirical estimates subjected to availability.

Table 5: Attenuating effect (%) on severity weights of comorbidity adjustments applied in the case study

	Comorbidity attenuation (%)							
Cause	**0–14**	**15–29**	**30–44**	**45–59**	**60–69**	**70–79**	**80+**	**All ages**
Infectious and parasitic diseases	-2.5	-3.9	-5.5	-7.9	-10.7	-13.5	-16.6	-7.3
Acute respiratory infections	-2.2	-1.9	-2.6	-3.5	-3.8	-3.1	-5.2	-2.6
Maternal conditions	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Neonatal causes	-9.1	-10.4	-11.4	-12.6	-13.7	-15.0	-16.7	-11.4
Nutritional deficiencies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malignant neoplasms	-6.5	-7.6	-8.8	-11.1	-13.3	-14.9	-16.5	-13.1
Other neoplasms	-3.9	-6.8	-10.3	-13.7	-18.6	-23.2	-29.3	-14.4
Diabetes mellitus	-3.4	-4.6	-6.5	-8.4	-10.4	-12.4	-15.3	-10.8
Endocrine and metabolic disorders	-3.6	-4.9	-6.8	-9.4	-12.1	-14.8	-18.0	-8.3
Mental disorders	-4.8	-6.8	-8.4	-10.8	-12.7	-14.1	-15.5	-9.3
Nervous system & sense organ disorders	-7.9	-5.8	-6.3	-8.6	-11.9	-16.1	-20.6	-15.1
Cardiovascular disease	-1.4	-2.3	-4.7	-8.5	-10.7	-13.1	-15.7	-11.9
Chronic respiratory disease	-2.4	-3.4	-6.2	-9.8	-12.9	-15.1	-17.6	-9.9
Diseases of the digestive system	-5.5	-6.8	-8.5	-11.0	-14.0	-17.0	-20.5	-12.6
Genitourinary diseases	-7.5	-7.7	-8.8	-11.3	-13.5	-15.6	-18.7	-11.0
Skin diseases	-2.6	-4.1	-5.7	-8.2	-12.2	-15.8	-20.9	-8.9
Musculoskeletal diseases	-6.3	-6.1	-7.1	-9.7	-12.3	-14.6	-17.0	-12.0
Congenital anomalies	-11.9	-12.9	-13.8	-14.7	-15.5	-16.3	-17.5	-13.7
Oral conditions	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ill-defined conditions	n.a	-6.6	-9.4	-12.4	-15.9	-19.5	-23.8	-11.6
Unintentional injuries	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Intentional injuries	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
All causes	-5.1	-6.1	-7.3	-9.3	-11.4	-14.1	-17.7	-10.6

Notes: Based on simulations of comorbidity as measured by Ausdiab 1999, the National Health Survey 2001, the Survey of Disability, Ageing and Carers 2003, and the National Mental Health Survey 1997. Attenuation calculated as ((prevalence multiplied by adjusted severity weight) divided by (prevalence multiplied by unadjusted severity weight) minus one) multiplied by 100. n.a denotes not applicable.

For the case study I implemented the above approach in Stata to derive a set of adjusted severity weights for each age and sex across the group of 180 disease and injuries, and their individual sequela. The attenuating effect of these adjustments is summarised at an aggregated level in Table 5, while the adjusted weights themselves are summarised at the same level of detail in Table 6. The routines that performed the underlying calculations are at Appendix F. Time constraints prevented the estimation of separate sets of adjustments factors severity weights for different subpopulations and time periods.

Table 6: Comorbidity-adjusted severity weights applied in the case study

	Severity weight							
Cause	**0–14**	**15–29**	**30–44**	**45–59**	**60–69**	**70–79**	**80+**	**All ages**
Infectious and parasitic diseases	0.045	0.049	0.087	0.091	0.089	0.083	0.090	0.077
Acute respiratory infections	0.060	0.054	0.055	0.066	0.075	0.086	0.105	0.061
Maternal conditions	0.112	0.113	0.140	0.294	0.388	0.388	0.388	0.135
Neonatal causes	0.216	0.200	0.198	0.194	0.189	0.183	0.173	0.199
Nutritional deficiencies	0.006	0.006	0.006	0.007	0.006	0.006	0.006	0.006
Malignant neoplasms	0.391	0.165	0.087	0.072	0.083	0.081	0.075	0.080
Other neoplasms	0.238	0.186	0.146	0.139	0.156	0.168	0.185	0.148
Diabetes mellitus	0.068	0.069	0.069	0.069	0.070	0.068	0.066	0.069
Endocrine and metabolic disorders	0.198	0.170	0.149	0.152	0.183	0.206	0.207	0.174
Mental disorders	0.114	0.082	0.089	0.135	0.132	0.103	0.077	0.102
Nervous system & sense organ disorders	0.065	0.036	0.033	0.037	0.045	0.070	0.117	0.060
Cardiovascular disease	0.240	0.232	0.188	0.163	0.159	0.156	0.149	0.159
Chronic respiratory disease	0.053	0.053	0.060	0.074	0.093	0.104	0.104	0.072
Diseases of the digestive system	0.247	0.132	0.090	0.077	0.093	0.114	0.131	0.094
Genitourinary diseases	0.010	0.017	0.036	0.036	0.060	0.070	0.069	0.032
Skin diseases	0.026	0.034	0.029	0.030	0.042	0.063	0.080	0.036
Musculoskeletal diseases	0.057	0.043	0.042	0.044	0.047	0.051	0.055	0.047
Congenital anomalies	0.203	0.188	0.177	0.166	0.155	0.142	0.118	0.176
Oral conditions	0.023	0.016	0.011	0.007	0.005	0.005	0.005	0.008
Ill-defined conditions	0.000	0.316	0.308	0.298	0.286	0.274	0.260	0.300
Unintentional injuries	0.064	0.095	0.115	0.116	0.117	0.117	0.137	0.111
Intentional injuries	0.166	0.198	0.187	0.175	0.175	0.145	0.099	0.174
All causes	0.066	0.055	0.062	0.067	0.063	0.068	0.083	0.065

Notes: Severity weights measured on a scale from 0 (equivalent to perfect health) to 1 (equivalent to death).

A practical consideration arises when deriving DALYs using adjusted severity weights such as the ones implied by Table 6.[49] In their original discussion, Murray and Lopez introduce the general equation for the non-fatal component of the DALY as,

$$YLD = D\left(\frac{KCe^{ra}}{(r+\beta)^2}\left(e^{-(r+\beta)(L+a)}\left(-(r+\beta)(L+a)-1\right)-e^{-(r+\beta)a}\left(-(r+\beta)a-1\right)+\frac{1\_K}{r}\left(1-e^{-rL}\right)\right)\right),$$

Equation 5

where YLD is years lived with disability, a is the age of onset of the disability, L is the duration of disability, r is the discount rate, β is the age-weighting parameter, K is the age-

[49] I am indebted to Professor Theo Vos for drawing my attention to this detail.

weighting modulation factor, C is the adjustment constant necessary because of unequal age-weights and D is the severity weight.[50]

Obscured in Equation 5 by the discounting and age-weighting functions—the latter being a normative assumption ignored in many applications of the framework—is the assumption that weighting for severity is constant across duration and therefore, by implication, across age. Thus a simplified expression that is consistent with the notation already established in this chapter, is given by,

$$unadjYLD_k^a = DW_k^a \times f(a,d,\beta,\delta),$$

Equation 6

where a is the age of onset of cause k, d is the duration of k, δ is the discount rate, β is the age-weighting parameter, and DW is the severity weight. In this equation, I have added the right-hand superscript a to both the severity weight and the result to make explicit the age-constancy assumption. I have also added the *unadj* prefix to indicate that *YLD* calculated in this way are unadjusted for age effects arising from factors such as comorbidity.

A modified version of Equation 6 that accommodates age-varying severity weights is given by,

$$adjYLD_k^a = \begin{cases} adjDW_k^a \times f(a,d,\beta,\delta), & \text{if } d \le 1 \\ adjDW_k^a \times f(a,d,\beta,\delta) + & \\ \sum_{j=1}^{\lfloor d \rfloor - 1} \left(adjDW_k^{a+j} \times f(a+j,j+1,\beta,\delta) - adjDW_k^{a+j} \times f(a+j,j,\beta,\delta) \right) + & \\ \left(adjDW_k^{a+\lfloor d \rfloor} \times f(a+\lfloor d \rfloor,d,\beta,\delta) - adjDW_k^{a+\lfloor d \rfloor} \times f(a+\lfloor d \rfloor,\lfloor d \rfloor,\beta,\delta) \right), & \text{elsewhere} \end{cases}$$

Equation 7

where $\lfloor x \rfloor$ indicates the floor function.

For the case study I implemented Equation 7 as a discrete process using Stata as set out at Appendix F. The code is run as a secondary process using as inputs i and d from the set of

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[50] Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I.*

workflows depicted in Figure 7, and the corresponding *adjDWs* implied by Table 6. Table 7 summarises the percentage difference between Equation 5 and Equation 7 when using severity weights that varying by age.

Table 7: Difference (%) between Equation 5 and Equation 7 to calculate YLD using age-varying severity weights as applied in the case study

	Difference (%)							
Cause	**0–14**	**15–29**	**30–44**	**45–59**	**60–69**	**70–79**	**80+**	**All ages**
Infectious and parasitic diseases	1.3	1.6	1.8	1.2	0.9	0.3	-0.1	1.4
Acute respiratory infections	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Maternal conditions	0.0	0.0	0.0	0.0	n.a	n.a	n.a	0.0
Neonatal causes	1.6	n.a	n.a	n.a	n.a	n.a	n.a	1.6
Nutritional deficiencies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malignant neoplasms	0.3	0.9	2.1	3.0	2.1	0.6	-0.9	1.7
Other neoplasms	0.0	0.5	0.2	0.0	0.0	0.0	0.0	0.1
Diabetes mellitus	2.3	2.6	2.5	2.1	1.6	1.2	0.7	1.9
Endocrine and metabolic disorders	1.6	1.1	1.1	1.0	0.5	0.3	0.1	1.2
Mental disorders	0.6	-1.2	-8.1	3.0	17.5	21.1	0.1	-1.6
Nervous system & sense organ disorders	0.7	1.2	1.8	2.3	1.9	-1.8	0.6	0.5
Cardiovascular disease	0.2	0.3	0.5	-1.2	-1.2	0.3	0.3	-0.3
Chronic respiratory disease	0.6	1.6	2.3	1.9	1.5	1.0	0.6	1.3
Diseases of the digestive system	2.6	2.7	2.6	1.9	1.4	0.9	0.3	2.1
Genitourinary diseases	-3.1	0.7	0.3	1.3	-4.4	1.2	0.7	0.0
Skin diseases	0.2	0.2	0.2	0.2	0.1	0.0	0.0	0.1
Musculoskeletal diseases	0.9	0.9	0.9	0.9	0.9	0.8	0.6	0.9
Congenital anomalies	0.6	2.2	1.7	1.2	1.0	0.6	0.3	0.6
Oral conditions	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ill-defined conditions	n.a	0.5	0.7	0.9	1.2	n.a	n.a	0.8
Unintentional injuries	-0.2	-0.3	-0.4	-0.9	0.0	0.0	0.0	-0.3
Intentional injuries	-0.3	-0.6	-1.3	-2.5	0.0	0.0	0.0	-1.0
All causes	0.6	-0.4	-2.4	1.7	1.7	-0.1	0.4	0.2

Notes: Difference calculated as ((YLD using Equation 5 divided by YLD using Equation 7) minus one) multiplied by 100. Age-varying severity weights were used in both cases. n.a denotes not applicable.

The overall effect of this correction is marginal (not greater than ±2.4% across all age and cause groups). However, it is relatively large for mental disorders between the ages of 30 and 79 years due to the combined influence of long duration and declining severity with age, at least from 45 years.

Chapter 3: Cause-specific mortality and the IPM

Introduction

By their own account, Murray and Lopez can be credited with having the IPM implemented as the software package Dismod, in which a hypothetical cohort is exposed to a set of user-specified values with respect to *i*, *f* and *r (*using the notation in Chapter 1 Figure 2) and 'general mortality' to derive values for those parameters that might be of interest for secondary processes.[51] As an alternative to *f*, users could enter a 'relative risk minus one coefficient' as a mortality input, although the correct use of this particular function is not elaborated on in their initial explanations of the model. As a result, I will denote the relative risk component of this parameter as *RR´* to indicate that it is for moment undefined.

A key requirement of Dismod was that *i*, *r*, and *f* (or *RR´*-1) must be entered as inputs. However, some or all of these parameters are often not known, in which case users of the tool had to guess the correct values using an iterative process such that the modelled outputs were consistent with observed data or assumptions. As noted already in Chapter 1, to overcome this and other limitations WHO commissioned a second implementation of the IPM in the late 1990s to facilitate work on the GBD 2000. Most noticeably from the user's perspective, Dismod II incorporated a goal-seeking algorithm to find values for the hazards such that a consistent model could be developed from most combinations of at least three of the model's parameters.[52] This implementation of the IPM was used extensively in the GBD 2000 study[53] and other work that followed.[54]

In their introduction to Dismod II, Associate Professor Jan Barendregt and others, including Murray and Vos, refer to a conceptual model that is identical to the one depicted in Chapter 1

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51 ibid.

52 Less noticeably, an exact solution to the underlying differential equations was implemented rather than the finite difference method used in the original software.

53 Mathers, C, Salomon, J, Ezzati, M, Begg, S, Vander Hoorn, S and Lopez, A, "*Sensitivity and uncertainty analyses for burden of disease and risk factor estimates*".

54 e.g. Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Begg, S, Vos, T, Goss, J and Mann, N, "An alternative approach to projecting health expenditure in Australia", *Aust Health Rev*, 2008, vol. 32(1), pp. 148-55; Bundhamcharoen, K, Teerawatananon, Y, Vos, T and Begg, S, *The Burden of Disease and Injuries in Thailand*, Bureau of Health Policy and Planning, Ministry of Public Health, Bangkok, 2002; Vos, T, Barker, B, Begg, S, Stanley, L and Lopez, AD, "Burden of disease and injury in Aboriginal and Torres Strait Islander Peoples: the Indigenous health gap", *Int J Epidemiol*, 2009, vol. 38(2), pp. 470-7; Yusoff, A, Mustafa, A, Kaur, G, Omar, M, Vos, T, Rao, V and Begg, S, *Malaysian Burden of Disease and Injury Study*, Division of Burden of Disease, Institute of Public Health, Ministry of Health, Kuala Lumpur, 2004.

except that *m* is used to denote mortality from 'all other causes'[55] rather than the more inclusive meaning provided in the original explanation. Whereas Murray and Lopez do not discuss the relationship of this parameter to others in the model, Barendregt and colleagues refer specifically to *m* by arguing that because it does not influence *i*, *r*, and *f* when it is truly independent of a cause (i.e. it is experienced by healthy and diseased people equally) it can be omitted from the equations without affecting all but one of the calculated parameters. The exception is average duration (*d* using the notation in Chapter 2), which can only be calculated once *m* is determined.

Barendregt and colleagues define another parameter *b* to represent 'all excess mortality caused by the disease', a definition which, in their view, is consistent with a broader but otherwise undefined aim of the framework. In explaining this parameter, they note that *b* is not necessarily consistent with cause-specific mortality reported by national statistics agencies (or m_k using the notation in Chapter 2). In other words, the actual deaths coded to a cause may not reflect all mortality that might be attributable to that particular cause. My objectives in this chapter therefore are twofold: to position this distinction within a more developed conceptual framework than provided for in the literature; and to explore its implications for the IPM, particularly with respect to the relative risk function in two implementations of the model.[56]

Cause-specific mortality

In Chapter 1, I noted two separate traditions for attributing causes to health outcomes: categorical and counterfactual attribution. This distinction has its origins in a discussion by Mathers, Murray, Lopez and others with respect to comparative risk assessment.[57] The initial treatment of mortality within the Murray and Lopez framework largely reflects categorical attribution. However, Barendregt and colleagues point to situations in which this approach is an inadequate basis for determining cause-specific mortality in the IPM.

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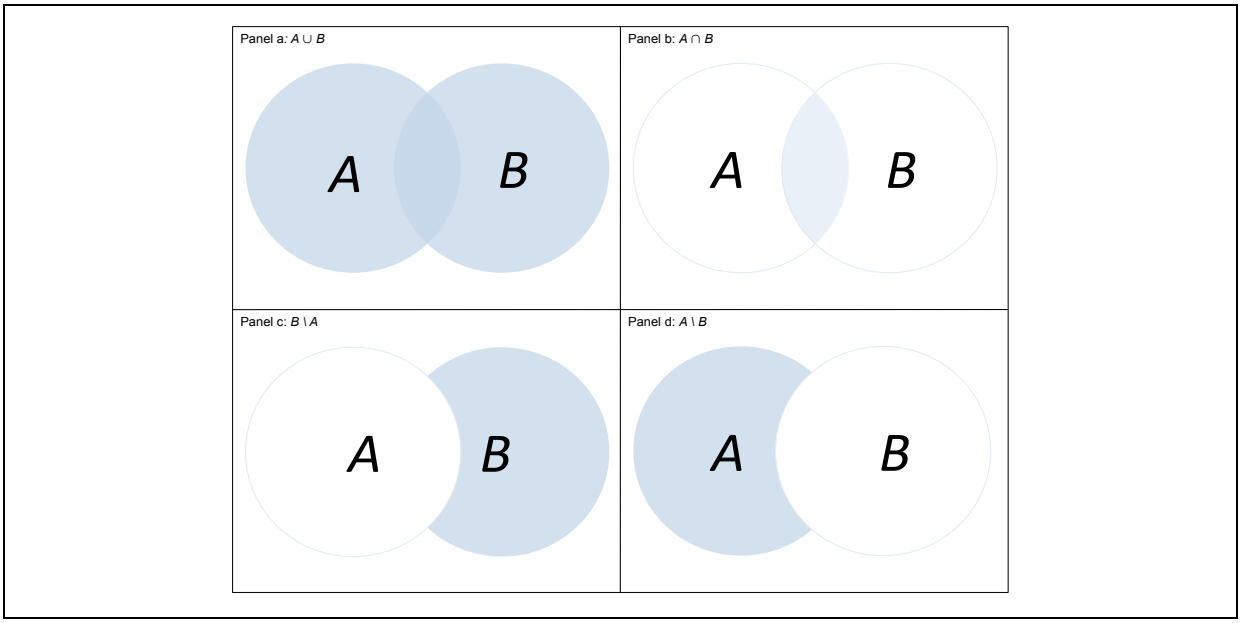
[55] Barendregt, JJ, Van Oortmarssen, GJ, Vos, T and Murray, CJ, "A generic model for the assessment of disease epidemiology: the computational basis of DisMod II".

[56] I am indebted to Dr Colin Mathers for inspiring me to undertake this investigation.

[57] Mathers, CD, Ezzati, M, Lopez, AD, Murray, CJL and Rodgers, AD, "Causal decomposition of summary measures of population health", in Murray, CJL, Salomon, J, Mathers, CD and Lopez, AD, eds, *Summary measures of population health: concepts, ethics, measurement and applications*, World Health Organization, Geneva, 2002.

A useful example is perhaps diabetes, which confers an underlying vascular risk that predisposes diabetics to die from other related causes in addition to diabetes, such as cardiovascular disease and stroke; thus the actual mortality coded to diabetes does not reflect the true mortality associated with this disease.[58] This phenomenon can be illustrated using basic set theory. In Figure 9, I denote A as the set of deaths not coded to diabetes and B the set of deaths attributable to diabetes. It follows that the union between these sets is the universe of all deaths (panel a) and the intersection is the subset of deaths attributable to diabetes but not coded to this disease (panel b). Similarly, the relative complement of A in B (or $B \setminus A$) is the subset of deaths coded to diabetes (panel c).

Figure 9: Understanding *m* in the IPM using basic set theory



With respect to the equations underlying Dismod II, the logic proposed by Barendregt and colleagues depends on b being independent of m to derive externally valid results from the IPM. In theory, this condition is met when m is understood as the subset of A that excludes B (panel d); in practice, as Kruijshaar, Barendregt and others note in an earlier discussion, satisfying this condition is difficult, making it a problematic aspect of IPMs more generally.[59] While Barendregt and colleagues suggest in their introduction to Dismod II that the problem relates to the unobservable nature of b, the intersection between A and B is at the centre of

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[58] A proportion of diabetics will also be obese, which confers additional mortality risk from non-vascular diseases such as certain cancers. To the extent that the proportion of obesity is greater in diabetics than in non-diabetics, this would confer additional mortality risk over and above the risk associated with simply having diabetes.

[59] Kruijshaar, ME, Barendregt, JJ and Hoeymans, N, "The use of models in the estimation of disease epidemiology", *Bull World Health Organ*, 2002, vol. 80(8), pp. 622-8.

these difficulties, not the subset of *B* that excludes *A,* which is, by definition, observable. In any case, the *RR´* function in both versions of Dismod provides what appears to be a solution by allowing *b* to be specified via counterfactual analysis principles instead of as a categorical parameter.

Relative risk of mortality

Relative risk is an established concept in the epidemiological literature and typically expresses the ratio of total mortality or other health outcome in a group exposed to a particular risk over that experienced by a group in which the risk is absent. This ratio can be used to estimate the proportion of a health outcome due to exposure to a particular risk.[60] In its simplest form, the formula for this calculation is,

$$AF = \frac{p(RR-1)}{p(RR-1)+1}$$

Equation 8

where *AF* is the attributable fraction of the health outcome in a population that is due to exposure to the risk, *p* is the prevalence of exposure in the population, and *RR* is the relative risk as defined above.

While Murray and Lopez first introduce relative risk in the context of Dismod as a means for determining the 'number of deaths associated with a disease',[61] arguably this application has only limited value given they largely adopt categorical attribution for the mortality component of their framework.[62] Nevertheless, as a description of the intended relationship between *RR´* and *b* it is possibly a more useful explanation than the one provided by Barendregt and colleagues. In the Dismod II documentation, Barendregt and colleagues

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60 Ezzati, M, Hoorn, SV, Rodgers, A, Lopez, AD, Mathers, CD and Murray, CJ, "Estimates of global and regional potential health gains from reducing multiple major risk factors", *Lancet*, 2003, vol. 362(9380), pp. 271-80; Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 1*; Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 2*, World Health Organization, Geneva, 2004; Mathers, CD, Ezzati, M, Lopez, AD, Murray, CJL and Rodgers, AD, "*Causal decomposition of summary measures of population health*"; Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I.*

61 Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I.*

62 Murray and Lopez do present an alternative set of results for a group of 10 diseases based on counterfactual attribution. However, is not clear whether the relative risk function in Dismod was used in these calculations.

define *RR′* as the mortality hazard in prevalent cases over the mortality hazard in prevalent and non-prevalent cases combined. I will denote these hazards as m_p and m_t, respectively, and use RR_d to denote the documented definition of *RR′*. In comparison, a conventionally defined relative risk, which I denote as RR_c, has the mortality hazard in non-prevalent cases as the denominator. I will use m_n to denote this hazard.

It is worth briefly noting the relationships between RR_c and previously defined measures of mortality. For example, an alternative expression of the hazard *f* is the difference between m_p and m_n, which is consistent with a measure referred to elsewhere as *excess risk*.[63] It can also be expressed as *b* over *p,* where these parameters are as defined above. Similarly, *f* can be derived from RR_c and m_n using,

$$f = m_n\left(RR_c - 1\right),$$

Equation 9

a relationship that presumably underpins Barendregt and colleagues' observation, 'case fatality and relative risk for total mortality contain the same information, given total mortality'.[64]

Given these relationships, it seems reasonable to assume that Murray and Lopez conceived of the *RR′* function as a means of deriving *f* from relative risk in situations where the former could not be observed directly and where an estimate of the latter was available. Certainly, in Barendregt and colleagues' implementation the derivation of the former from the latter must occur before proceeding with the main calculations because *f* is one of the three inputs to the underlying set of formulae, not relative risk. However, the formula implemented by Barendregt and colleagues to derive *f*, which can be determined from a comparison of inputs and outputs, is given by,

$$f' = m_t\left(RR' - 1\right).$$

Equation 10

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63 Gail, MH and Bénichou, J, *Encyclopedia of epidemiologic methods*, Wiley, Chichester, 2000.

64 Barendregt, JJ, Van Oortmarssen, GJ, Vos, T and Murray, CJ, "A generic model for the assessment of disease epidemiology: the computational basis of DisMod II".

In this formula I use the prime to denote it represents an approximate rather than an exact derivation because of the use of m_t in place of m_n.[65]

Thus it is possible to identify two potential issues with the *RR´* function as implemented in Dismod II. First, the relative risk measure referred to in the documentation relating to how to input *RR´* appears at odds with convention.[66] Second, m_t is used to derive an approximation of *f* when an exact derivation might be possible. Common to both issues is the use of m_t as a proxy for m_n; the latter, by definition, does not intersect with *b* and is therefore experienced by prevalent and non-prevalent cases equally. The former, on the other hand, represents a superset of *b* and is therefore not independent of the disease. The greater *b* is in proportion to the total number of deaths, the greater the independence assumption would appear to be compromised by the *RR´* function. This observation seems true whichever relative measure of mortality is used as an input.

Implications

It is possible to compare RR_d and RR_c as separate inputs to *RR´* and assess whether the substitution of one for the other has material implications for the derivation of *f* and ultimately *b.* In the case of RR_d, my expectation was that Dismod II would underestimate *f* because, by definition, the relative measure of mortality used in this instance does not represent the full excess mortality risk between prevalent and non-prevalent cases. Nevertheless, it is understandable that users might input RR_d since it is the documented definition of *RR´*.[67]

In the case of RR_c, users might reasonably expect this relative measure of mortality to result in an exact derivation of *f* since RR_c expresses the full excess mortality risk between prevalent

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65 Associate Professor Jan Barendregt confirms that the derivation occurs in this way. He also acknowledges that this leads to an acceptable approximation when disease prevalence is low (personal communication, 20 August 2009 and 18 August 2011), although such a caveat is not mentioned in the formal literature or in Dismod II's documentation.

66 Professor Theo Vos counters by noting that Dismod II's definition follows that of the Standardised Mortality Ratio (SMR) and is therefore not without precedent in the epidemiological literature. The SMR is typically used to compare the mortality risk between two populations with dissimilar age distributions—particularly when the size of one population is too small to use other standardisation techniques—and is interpreted as the number of deaths in one population (usually the smaller one) over the number of deaths in that population had it experienced the age-specific death rates of the other. This seems conceptually distinct from the way in which relative risk might be entered into Dismod II.

67 It is also consistent with a view promoted by Professor Theo Vos that Dismod II somehow transforms RR_d as an input into RR_c as an output (personal communication, 7 August 2009).

and non-prevalent cases. However, the internal derivation still uses m_t rather than m_i, thus f is in fact overestimated by a factor equivalent to,

$$e_c = p\left(RR_c - 1\right),$$

Equation 11

where e_c denotes the RR_c derivation error and p denotes prevalence. These predictions can be explored using data from an existing Dismod II model for diabetes in Australian males[68], which I have reproduced in columns *a* through *d* of Table 8. The remaining columns represent four expressions of mortality derived from these parameters without using Dismod II.

Table 8: Selected epidemiological parameters for diabetes in Australian males

	Population structure[a]	p[b]		Mortality per 1,000				f[h]
Age group	(%)	(%)	RR_c[c]	m_t[d]	m_n[e]	m_p[f]	RR_d[g]	(%)
<1	1.3	0.0	2.43	5.3	5.3	13.0	2.43	0.8
1-4	5.3	0.0	2.43	0.3	0.3	0.7	2.43	0.0
5-9	7.0	0.0	2.43	0.1	0.1	0.3	2.43	0.0
10-14	7.2	0.0	2.43	0.1	0.1	0.3	2.43	0.0
15-19	7.1	0.0	2.43	0.6	0.6	1.5	2.43	0.1
20-24	7.1	0.0	2.43	0.9	0.9	2.2	2.43	0.1
25-29	6.9	0.1	2.43	1.0	1.0	2.4	2.43	0.1
30-34	7.6	0.5	2.43	1.1	1.1	2.6	2.42	0.2
35-39	7.3	1.6	2.43	1.3	1.3	3.2	2.38	0.2
40-44	7.7	3.5	2.43	1.8	1.7	4.1	2.32	0.2
45-49	7.0	6.9	2.37	2.5	2.3	5.5	2.16	0.3
50-54	6.6	9.7	2.27	3.5	3.1	7.1	2.03	0.4
55-59	5.9	12.6	2.16	5.9	5.1	11.0	1.88	0.6
60-64	4.4	16.1	2.02	9.7	8.3	16.9	1.73	0.9
65-69	3.6	19.6	1.87	16.2	13.8	25.8	1.60	1.2
70-74	3.1	23.4	1.71	27.6	23.7	40.6	1.47	1.7
75-79	2.4	26.4	1.57	46.1	40.1	62.8	1.36	2.3
80-84	1.5	30.5	1.44	78.0	68.8	98.9	1.27	3.0
85-89	0.7	34.8	1.34	135.6	121.2	162.7	1.20	4.1
90-94	0.2	39.5	1.28	206.6	186.0	238.1	1.15	5.2
95-99	0.0	43.1	1.22	293.2	267.8	326.7	1.11	5.9
100+	0.0	45.1	1.18	359.7	332.7	392.5	1.09	6.0
All ages	100.0	5.9	1.93	7.0	6.2	10.6	1.71	1.4

Notes: (a) Estimated resident population for 2000 (n=9,871,642; Source: ABS); (b) Prevalence of diabetes in per cent (Source: AusDIAB); (c) Relative Risk defined as total mortality rate in diabetics over total mortality rate in non-diabetics (Source: Asia Pacific Cohort Studies Collaboration); (d) Total mortality rate in general population per 1,000 (Source: ABS); (e) Total mortality rate in non-diabetics per 1,000, calculated as $m_t/(p(RR_c-1)+1)$; (f) Total mortality rate in diabetics per 1,000, calculated as m_n*RR_c; (g) Relative Risk defined as total mortality rate in diabetics over total mortality rate in general population, calculated as m_p/m_t; (h) Case-fatality hazard calculated as $m_n(RR_c-1)$.

[68] Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Begg, SJ, Vos, T, Barker, B, Stanley, L and Lopez, AD, "Burden of disease and injury in Australia in the new millennium: measuring health loss from diseases, injuries and risk factors", *Med J Aust*, 2008, vol. 188(1), pp. 36-40.

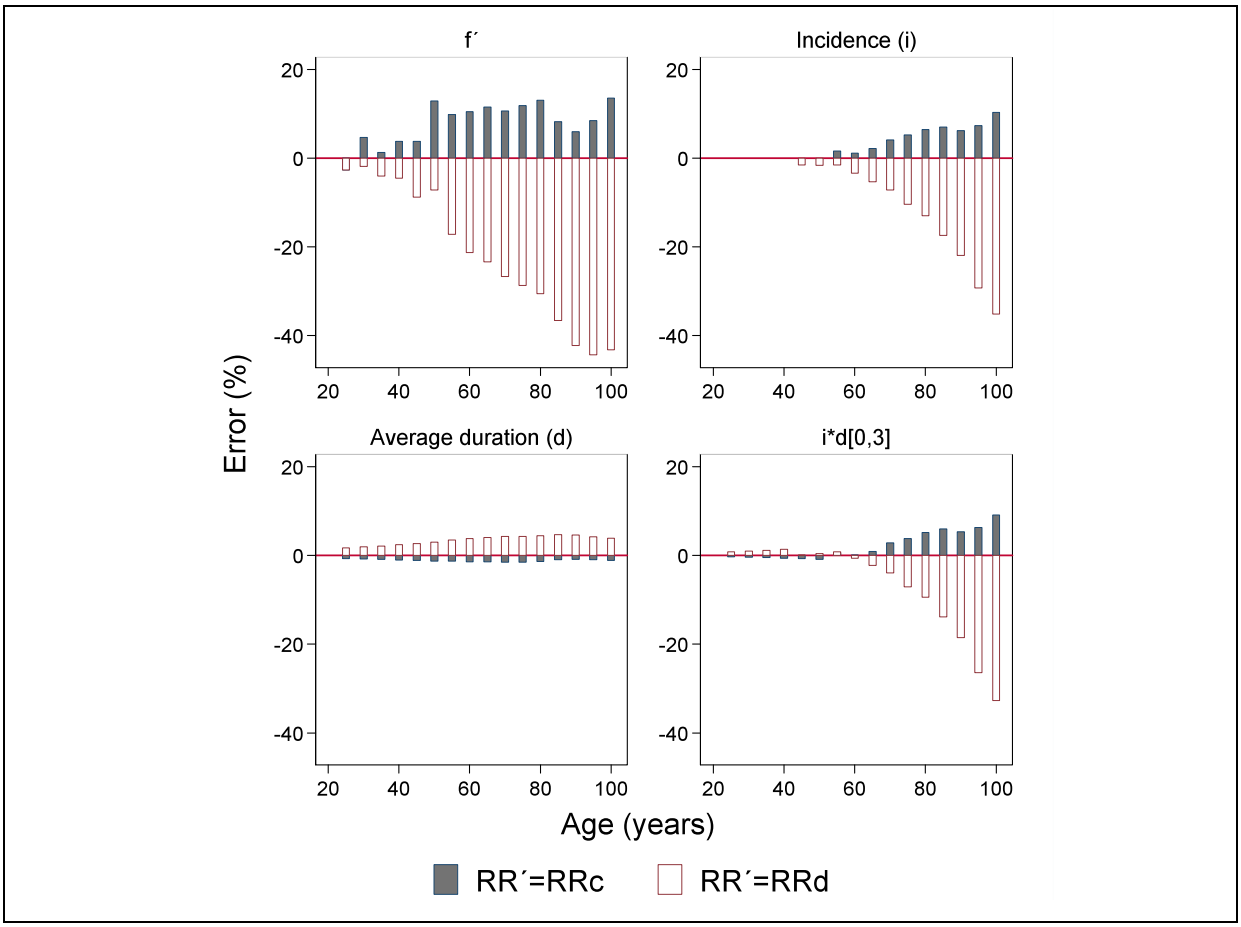
In Table 9, I present the 'case-fatality' output (f') from Dismod II for two models: Model 1, in which I used RR_c as the mortality input, and Model 2, in which I substituted RR_c for RR_d. Both models were calculated with the default settings using p as the prevalence input and zero for the remission input. Population structure and total mortality were the same in both instances. As predicted, f' is greater than or equal to the true case-fatality (f in Table 9) at each age in the first model. Conversely, f' is less than or equal to the true case-fatality in second model.

Table 9: 'Case-fatality' output from *Dismod II*

	Actual	f´(%)	
Age group	**f (%)**	**Model 1**	**Model 2**
25-29	0.1	0.1	0.1
30-34	0.2	0.2	0.2
35-39	0.2	0.2	0.2
40-44	0.2	0.3	0.2
45-49	0.3	0.3	0.3
50-54	0.4	0.5	0.4
55-59	0.6	0.7	0.5
60-64	0.9	0.9	0.7
65-69	1.2	1.3	0.9
70-74	1.7	1.9	1.2
75-79	2.3	2.5	1.6
80-84	3.0	3.4	2.1
85-89	4.1	4.5	2.6
90-94	5.2	5.5	3.0
95-99	5.9	6.4	3.3
100+	6.0	6.8	3.4
All ages	1.4	1.50	0.98

In Figure 10, I present the percentage difference from the true values, or bias, under both models for three Dismod II outputs: *f,* incidence (i) and average duration (d). I also present the product of i and d with a 3% discounting function (denoted as $i*d[0,3]$), which represents the epidemiological component of the YLD calculation, as specified in Equation 7 of the previous chapter.

Figure 10: Percentage difference from the true values under models 1 and 2 for *f*, *i*, *d*, and *i*d*[0,3]



These results indicate that under typical usage, the relative risk approach to estimating cause-specific mortality in Dismod II results in epidemiological models that are systematically inconsistent with intended assumptions or what can be implied from observational data. Such inconsistencies appear to be material in some cases and are likely to be greater with a relative risk defined as total mortality in diseased people over total mortality in the population as a whole—as the limited literature on this aspect of the tool implies should be used—than with a conventional relative risk.

The results also indicate that incorrectly specifying cause-specific mortality in the IPM can have a non-trivial effect, especially in the elderly. As will be discussed in the following chapter, for countries entering a possible new phase in human development, accurate information on the old and very old is becoming increasingly important as greater proportions of the population survive to these ages.[69] In the case study, for example, at least 25% of total

[69] Caldwell, JC, *What we know about health transition : the cultural, social and behavioural determinants of health : the proceedings of an international workshop, Canberra, May 1989*, Health Transition Centre, Australian National

DALYs was experienced by Australians aged 75 years and above, and approximately 30% of total DALYs for all ages was based on epidemiological models that relied on relative risk as an input.[70] As such, it seems likely that the results reported in the remaining chapters are not entirely consistent with the intended mortality assumptions. Further work would be required to determine whether the same is true for Murray and Lopez's original GBD 1990 study and subsequent analyses that make extensive use of the IPM.[71]

Options

Based on the above, I propose several options for accommodating the biases inherent within Dismod II if prevalence is known. First, they can be corrected using,

$$RR_e = \frac{RR_c - 1}{p(RR_c - 1)} + 1,$$

Equation 12

where RR_e is an error-corrected relative risk, which can be used as an input instead of its conventional equivalent. Alternatively, they can be avoided altogether by deriving f outside Dismod II using,

$$f = \frac{m_t\left(RR_c - 1\right)}{p\left(RR_c - 1\right) + 1}.$$

Equation 13

One advantage of Equation 13 is that Dismod II has the ability to output values for f that correspond exactly to the input values, whereby allowing users to develop epidemiological models that are entirely consistent with observational data or assumptions. The equivalent functionality is not available in Dismod II if choosing to use Equation 12.

These considerations notwithstanding, it seems advisable to balance judgements about the plausibility of models estimated by Dismod II—including the effect of any potential biases, corrected or otherwise—against the extent to which the available observational data can be

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University, Canberra, 1990; Frenk, J, Bobadilla, JL, Stern, C, Frejka, T and Lozano, R, "Elements for a theory of the health transition", *Health Transit Rev*, 1991, vol. 1(1), pp. 21-38.

[70] Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*.

[71] Such considerations may also apply to a third version of Dismod being used for the most recent Global Burden of Disease Study, although this was not assessed in the present analysis.

considered an accurate reflection of the true epidemiology of the cause being modelled. For example, information on relative risk is often only available for very coarse age groupings, or for all ages combined. In these situations, the systematic biases identified above might be a secondary consideration to deriving a plausible age pattern for *RR´*. Similarly, it is not uncommon for information on case-fatality or relative risk to be reported by age of incidence rather than age of death. Dr Judy Katzenellenbogen and colleagues show how this further complicates the way in which such information can be used to derive total attributable mortality in the IPM.[72]

This concludes Part I of this thesis. In the following two parts I present a portfolio of papers or *outputs* that seek to address several areas of contemporary policy concern. Those in Part II, 'Policy applications of proposed projection methods', rely predominantly on an alternative set of projection methods, as proposed in Chapter 2, while those in Part III, 'Policy applications of proposed subpopulation methods', depend on the subpopulation disaggregation methods also outlined in that chapter.

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[72] Katzenellenbogen, JM, Vos, T, Somerford, P, Begg, S, Semmens, JB and Codde, JP, "Excess mortality rates for estimating the non-fatal burden of stroke in Western Australia: a data linkage study", *Cerebrovasc Dis*, 2010, vol. 30(1), pp. 57-64.

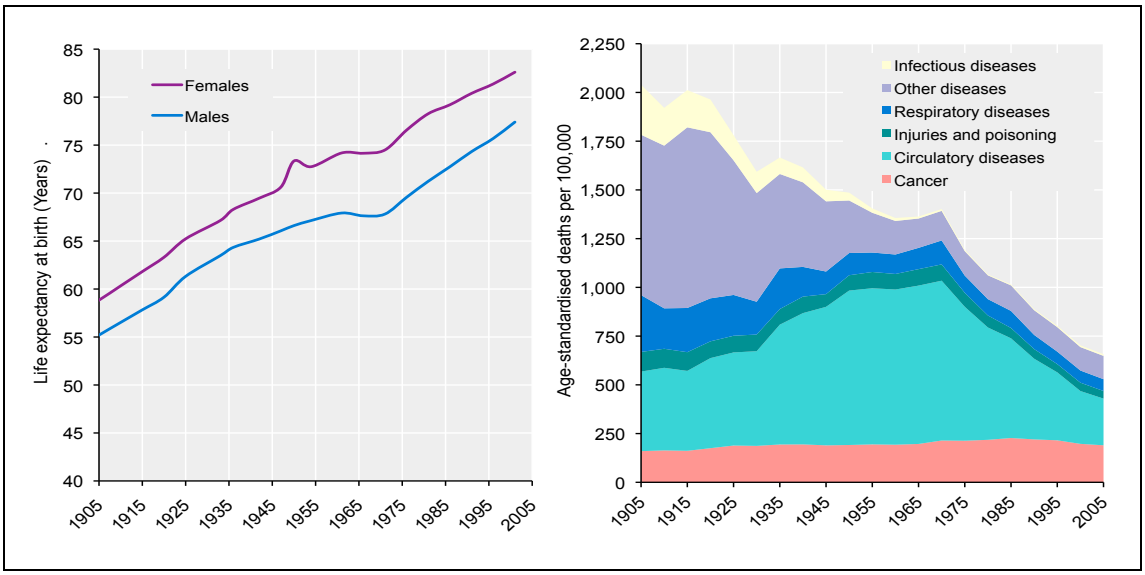
Part II: Policy applications of proposed projection methods

Chapter 4: Health in a post-transition population – adding years to life or life to years?

Introduction

The emerging field of population health dynamics is informed by a number of overlapping theories about change. From demography comes the theory of the demographic transition. Characterised as the central preoccupation of modern demography,[73] this is a generalisation from observed trends that development leads to low birth and death rates where once both were high. The transition is the period separating the two situations and is typically characterised by rapid population growth and more gradual population ageing.[74] The discipline of epidemiology provides the complimentary theory of the epidemiologic transition,[75] a generalisation, again from observed trends, that a decreasing proportion of deaths caused by infectious diseases accompanies declining mortality. Figure 11 provides a graphic illustration of these trends in Australia over the twentieth century.

Figure 11: A century of health improvement in Australia[76]



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73 Caldwell, JC, "Demography and social science", *Popul Stud (Camb)*, 1996, vol. 50(3), pp. 305-33.

74 As the survival curve becomes increasingly 'rectangular'; see Rowland, D, *Demographic methods and concepts*, Oxford University Press, New york, 2003.

75 Omran, AR, "The epidemiologic transition. A theory of the epidemiology of population change", *Milbank Mem Fund Q*, 1971, vol. 49(4), pp. 509-38.

76 Adapted from AIHW, *Mortality over the twentieth century in Australia: Trends and patterns in major causes of death*. Mortality Surveillance Series no. 4, Australian Institute of Health and Welfare, Canberra, 2005.

More recently, researchers have become interested in describing what happens to levels of morbidity as mortality declines. Termed the health transition,[77] this dynamic has proven more difficult to characterise due to differing interpretations of the term morbidity and the paucity of reliable and consistently recorded time-series information on non-fatal health outcomes at a population level. Nevertheless, several competing theories have emerged. The compression of morbidity hypothesis, first proposed by Fries,[78] posits an increase in both the absolute expectation of life towards a biological limit and the proportion of the life span free of illness. A variation of this idea, termed the relative compression of morbidity scenario, is that only the latter increases. The expansion of morbidity hypothesis, on the other hand, predicts an increase in the proportion of the life span with disability[79] because of improvements in the survival of those with disabling conditions and a shift in the distribution of causes of disability from fatal to non-fatal diseases of ageing.[80]

The theory of dynamic equilibrium, first described by Manton,[81] combines elements of both the compression and expansion hypotheses by proposing that mortality reductions are associated with a redistribution of disease and disability from more to less severe states. Under this scenario, the proportion of the life span with serious illness stabilises or decreases with decreasing mortality, whereas the proportion with less severe illness increases.

As mentioned in Chapter 1, understanding the impact of future trajectories of morbidity and mortality has important practical and strategic implications, particularly in developed countries where increasingly attention is turning to the future sustainability of public finances as life expectancies increase to unprecedented levels

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77 Caldwell, JC, *What we know about health transition : the cultural, social and behavioural determinants of health : the proceedings of an international workshop, Canberra, May 1989*; Frenk, J, Bobadilla, JL, Stern, C, Frejka, T and Lozano, R, "Elements for a theory of the health transition".

78 Fries, JF, "Aging, natural death, and the compression of morbidity", *N Engl J Med*, 1980, vol. 303(3), pp. 130-5.

79 Gruenberg, EM, "The failures of success", *Milbank Mem Fund Q Health Soc*, 1977, vol. 55(1), pp. 3-24.

80 Olshansky, SJ, Rudberg, MA, Carnes, BA, Cassel, CK and Brody, JA, "Trading Off Longer Life for Worsening Health: The Expansion of Morbidity Hypothesis", *J Aging Health*, 1991, vol. 3(2), pp. 194-216.

81 Manton, KG, "Changing concepts of morbidity and mortality in the elderly population", *Milbank Mem Fund Q Health Soc*, 1982, vol. 60(2), pp. 183-244.

and populations grow older. A useful class of analytic tools for exploring this dynamic is health expectancy.[82] First developed in a practical sense by Sullivan,[83] health expectancy extends the concept of life expectancy by reducing the estimated duration a person can expect to live at various ages given current risks of mortality by the amount of time spent at each age in states of less than perfect health.

Like life expectancy, measures of health expectancy are expressed in years and are independent of the age structure of the population being measured. Unlike life expectancy, however, health expectancy takes account of both mortality and health in the surviving population thereby providing information on both the length and healthfulness of life.

Measures of health expectancy have been used to show that the health transition in different contexts can add both years to life and 'life to years'.[84] Until quite recently, however, no decomposition or partitioning method was available for assessing the respective contribution of particular causes of death and disability to these findings. Instead, researchers looked at what happened when a specific disease was fully eliminated compared to the status quo.[85] Although useful for identifying major causes of health loss, the disease elimination approach is less appropriate for assessing the contribution of specific causes to differences in health expectancies over time.

Thus it is timely for Nusselder and Looman to have developed a method for partitioning differences in health expectancy into the additive contributions of death and disability from individual causes.[86] Using a modification of Arriaga's method for decomposing differences in life expectancy,[87] they illustrate the utility of their approach by decomposing male-female differences in disability-free life expectancy (DFLE) for the Netherlands. DFLE uses age-specific proportions of the population that meet a threshold criterion for disability (available in many countries from

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82 Robine, J-M, *Determining health expectancies*, J. Wiley, Chichester, 2003.
83 Sullivan, DF, "A single index of mortality and morbidity".
84 Robine, J-M, *Determining health expectancies*.
85 Mathers, CD, "Gains in health expectancy from the elimination of diseases among older people", *Disabil Rehabil*, 1999, vol. 21(5-6), pp. 211-21.
86 Nusselder, WJ and Looman, CW, "Decomposition of differences in health expectancy by cause", *Demography*, 2004, vol. 41(2), pp. 315-34.
87 Arriaga, EE, "Measuring and explaining the change in life expectancies", *Demography*, 1984, vol. 21(1), pp. 83-96.

surveys) to dichotomise the number of years lived at each age into those with and without disability.

DFLE has the advantage of being relatively straightforward to calculate. However, it ignores variations in severity below and above what is essentially an arbitrary threshold and is therefore a fairly crude measure for assessing theories about the health transition such as the one proposed by Manton.[88] Health-adjusted life expectancy (HALE), on the other hand, is more suited to this purpose because it involves summing across all major causes of health loss the product of the prevalence proportion for each cause and a corresponding numerical weight for severity ranging from 0 (equivalent to full health) to 1 (equivalent to death). Although HALE has greater information requirements than DFLE, it can still be calculated using the Sullivan method and can therefore be decomposed using the method proposed by Nusselder and Looman.

In this chapter I use HALE to explore the dynamics of the health transition as they are manifest at the beginning of the twenty-first century in Australia, a country that experienced changes over the preceding century characteristic of the demographic and epidemiologic transitions and which, as a consequence, now experiences very low levels of mortality. My aim is to determine whether existing theories about the health transition adequately describe the likely impact of future trajectories of morbidity and mortality in a population as it moves into a post epidemiologic transition phase of development. Australia is a convenient setting within which to pursue this investigation due to our recent comprehensive assessment of the health status of its population.[89]

Methods

My primary outcome of interest is the difference between life expectancy (LE) and health-adjusted life expectancy, which I will refer to as *absolute lost health expectancy* (ALHE) and define as,

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88 Manton, KG, "Changing concepts of morbidity and mortality in the elderly population".

89 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Begg, SJ, Vos, T, Barker, B, Stanley, L and Lopez, AD, "Burden of disease and injury in Australia in the new millennium: measuring health loss from diseases, injuries and risk factors".

$$ALHE_a^t = LE_a^t - HALE_a^t,$$

Equation 14

using the standard demographic right-hand superscript and subscript to denote a point in time *t* and an exact age *a*, respectively. I estimate LE and HALE using the Sullivan method for abridged life tables[90] from the estimates of mortality and prevalent disability described below and presented in Table 10. I measure absolute change in morbidity from *t* to *t*+*n* in years using,

$$\Delta ALHE_a^{(t,t+n)} = ALHE_a^t - ALHE_a^{t+n},$$

Equation 15

where absolute compression over time is indicated by a negative value and absolute expansion by a positive value. A related outcome of interest is lost health expectancy expressed as a proportion of life expectancy, which I call here *relative lost health expectancy* (RLHE) and define as,

$$RLHE_a^t = \frac{ALHE_a^t}{LE_a^t}.$$

Equation 16

I assess change in RLHE from *t* to *t*+*n* using,

$$\Delta RLHE_a^{(t,t+n)} = RLHE_a^t - RLHE_a^{t+n},$$

Equation 17

where relative compression in morbidity over time is indicated by a negative value and relative expansion by a positive value. The other outcome of interest is evidence of a shift towards less severe causes of health loss with decreasing mortality, which I explore by partitioning changes in ALHE over time into additive contributions from changes in levels of cause-specific mortality and disability.

The decomposition analysis followed the equations described by Nusselder and Looman[91], which I implemented in the Mata matrix programming environment.[92] The

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90 Sullivan, DF, "A single index of mortality and morbidity".

91 Nusselder, WJ and Looman, CW, "Decomposition of differences in health expectancy by cause".

model consists of the following Mata subroutines: `mata_cause_decomp`, `mata_decomp` and `mata_lt_rate`. These routines are 'wrapped' in the Stata ado program `hale_decomp`, which, if installed correctly, can be accessed from the command line like any other Stata command.

`hale_decomp` operates on a uniformly structured dataset and requires the following variables as inputs: *age* (0, 1, 5, 10…100, although other age groupings could be implemented), *ax* (the average midpoint for each age category in the population), *deaths* (the number of cause-specific deaths in each age category in the population), *pyld* (the number of cause-specific prevalent YLD in each age category in the population). It also requires the following variables (numeric or string) as inputs: *cause* (a variable that stratifies the data by cause), *strata* (a variable that stratifies the data by another dimension such as time or subpopulation group) and *base* (the value in *strata* that identifies the baseline stratum against which other strata are to be compared). The code for enabling `hale_decomp` is at Appendix G.

Mortality inputs were obtained from the previously mentioned analysis of actual and projected mortality by age and sex for a complete set of 180 diseases and injuries of public health importance in Australia over the period 1993 to 2023.[93] The same analysis was used for estimates of prevalent disability, which I refer to here as prevalent years lived with disability (PYLD) to be consistent with Murray and Lopez's original terminology. A prevalent year lived with disability is defined as,

$$_kPYLD = {}_kP \times {}_kDW,$$

Equation 18

where P denotes the prevalence proportion of cause k in the population and DW denotes the corresponding comorbidity-adjusted severity weight (range: 0 for full health to 1 for complete loss of health). Table 10 summarises the basic characteristics of the dataset used in this analysis.[94]

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92 StataCorp.

93 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Begg, SJ, Vos, T, Barker, B, Stanley, L and Lopez, AD, "Burden of disease and injury in Australia in the new millennium: measuring health loss from diseases, injuries and risk factors".

94 See Chapter 2 for full details.

Table 10: Mortality and prevalent disability by selected stratifications, Australia, 1993 and 2023

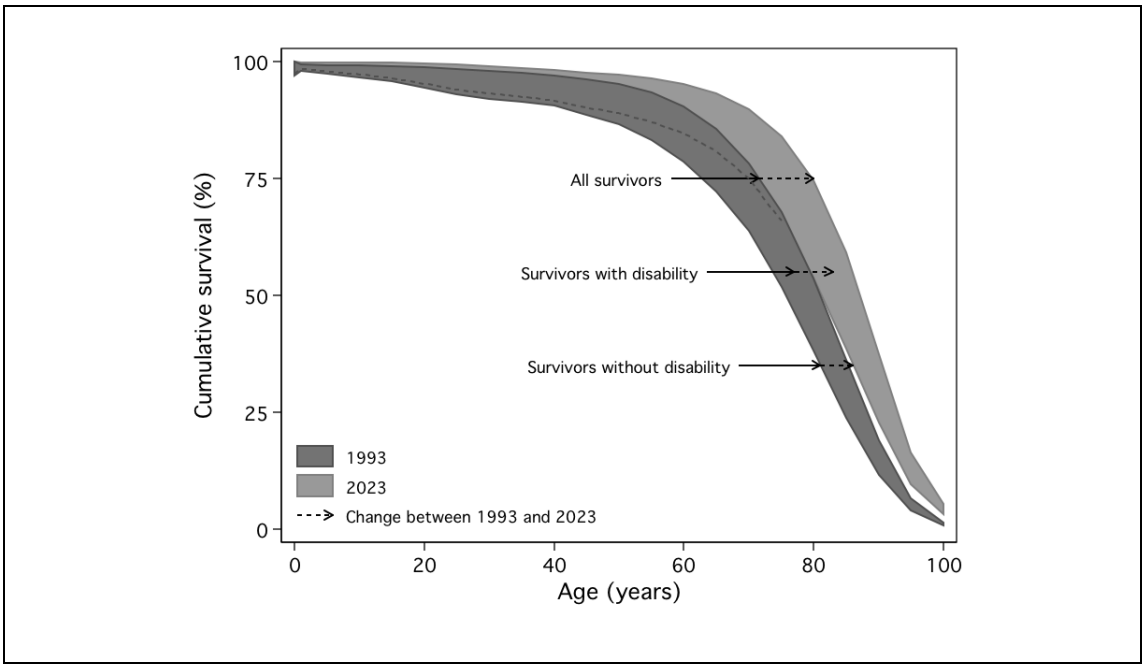
	Mortality[a]				PYLD[a]			
	1993		2023		1993		2023	
	n (%)	Rate	n (%)	Rate	n (%)	Rate	n (%)	Rate
Total[b]	122 (100.0)	68.8	187 (100.0)	37.9	1,311 (100.0)	742.2	2,185 (100.0)	698.6
Age group								
0 to <50	13 (10.7)	9.8	9 (4.9)	6.1	619 (47.2)	467.7	706 (32.3)	473.6
50 to <60	8 (6.9)	50.9	6 (3.3)	19.2	163 (12.4)	986.5	289 (13.2)	909.3
60 to <70	20 (16.4)	142.6	17 (9.0)	58.2	199 (15.2)	1421.4	354 (16.2)	1225.1
70 to <80	34 (28.3)	363.5	39 (21.0)	181.1	196 (14.9)	2068.2	406 (18.6)	1872.8
80 to <90	35 (28.4)	931.3	63 (33.5)	627.4	114 (8.7)	3056.1	305 (14.0)	3050.9
90 to <100	11 (8.8)	2,077.3	48 (25.8)	1,698.7	20 (1.5)	3883.3	114 (5.2)	3995.7
100+	1 (0.4)	4,759.3	5 (2.7)	1,866.4	0 (0.0)	4365.9	11 (0.5)	4019.8
Cause group[b,c]								
Infectious	2 (1.4)	1.0	4 (2.0)	0.9	18 (1.4)	10.4	28 (1.3)	10.5
Acute respiratory	1 (0.8)	0.6	9 (4.8)	1.4	11 (0.8)	6.3	14 (0.6)	6.3
Maternal	0 (0.0)	0.0	0 (0.0)	0.0	2 (0.2)	1.1	2 (0.1)	1.1
Neonatal	1 (0.7)	0.5	0 (0.1)	0.2	34 (2.6)	19.5	46 (2.1)	19.5
Nutritional	0 (0.1)	0.1	0 (0.1)	0.0	5 (0.4)	2.8	7 (0.3)	2.8
Cancer	33 (26.8)	18.5	54 (29.0)	12.6	79 (6.0)	44.8	103 (4.7)	27.9
Other neoplasms	1 (0.5)	0.3	1 (0.8)	0.3	3 (0.2)	1.4	4 (0.2)	1.3
Diabetes	3 (2.3)	1.6	6 (3.1)	1.2	64 (4.9)	36.2	204 (9.3)	55.5
Endocrine	1 (1.0)	0.7	2 (1.3)	0.5	16 (1.2)	9.1	31 (1.4)	10.0
Mental	1 (1.1)	0.7	2 (0.8)	0.5	345 (26.3)	195.2	485 (22.2)	194.9
Neurological	5 (3.9)	2.7	15 (7.9)	2.6	199 (15.2)	112.9	458 (21.0)	115.4
Cardiovascular	53 (43.6)	30.0	56 (29.6)	9.3	111 (8.4)	62.6	146 (6.7)	36.3
Chronic respiratory	7 (6.1)	4.2	15 (7.9)	3.0	129 (9.8)	73.0	188 (8.6)	60.5
Digestive	3 (2.2)	1.5	4 (2.3)	0.8	28 (2.1)	15.7	51 (2.3)	15.4
Genitourinary	2 (1.8)	1.3	8 (4.0)	1.3	38 (2.9)	21.8	60 (2.7)	22.1
Skin	0 (0.2)	0.1	1 (0.4)	0.1	16 (1.2)	9.2	25 (1.1)	9.1
Musculoskeletal	1 (0.5)	0.4	1 (0.7)	0.3	75 (5.7)	42.4	150 (6.8)	42.7
Congenital	1 (0.7)	0.5	1 (0.3)	0.2	35 (2.6)	19.6	44 (2.0)	19.0
Oral	0 (0.0)	0.0	0 (0.0)	0.0	20 (1.5)	11.2	32 (1.5)	11.2
Other syndromes	0 (0.2)	0.2	0 (0.1)	0.0	7 (0.5)	4.1	11 (0.5)	4.1
Injuries	7 (6.0)	4.1	9 (4.8)	2.8	76 (5.8)	42.8	97 (4.4)	32.5
Severity group[b,d]								
0 to <0.1	13 (10.6)	7.3	28 (14.9)	6.0	430 (32.8)	243.7	828 (37.9)	264.6
0.1 to <0.2	59 (48.5)	33.4	81 (43.2)	15.4	430 (32.8)	243.4	647 (29.6)	211.0
0.2 to <0.3	27 (22.6)	15.6	44 (23.3)	9.6	206 (15.7)	116.3	307 (14.1)	103.9
0.3 to <0.4	17 (13.7)	9.4	23 (12.2)	4.5	139 (10.6)	78.6	205 (9.4)	62.8
0.4 to 1	6 (4.6)	3.2	12 (6.4)	2.5	106 (8.1)	60.2	196 (9.0)	55.9

Notes: (a) numbers expressed in units of 1,000, rates expressed per 10,000 population; (b) rates standardised to the age structure of Australian population in 1993; (c) approximately equivalent to chapter headings of the International Classification of Diseases version 10; (d) determined by classifying each cause-, age and sex-specific PYLD estimate to its corresponding severity weight; totals may not add due to rounding; PYLD = prevalent years lived with disability.

Results

The total area under the survival curve for all survivors (including those with and without disability) and survivors without disability represents LE_0^t and $HALE_0^t$, respectively (Figure 12). The projected trajectories for cause-, age- and sex-specific mortality and prevalent disability presented in Table 10 differentially shift these survival curves to the right over time, as demonstrated by a 0.28% per annum increase in $LE_0^t$to 84.6 years in 2023 compared with a 0.26% increase per annum in $HALE_0^t$ to 76.2 years in the same year.

Figure 12: Cumulative survival with and without disability, Australia, 1993 and 2023



Cumulative survival with disability (the dark and light grey areas in Figure 12) is presented on its own axis in Figure 13. The area under the lines represents $AHLE_0^t$, which is projected to increase from 7.3 years in 1993 to 8.5 years in 2023. The difference between the areas under the lines for 2023 and 1993 is the absolute change in life with disability between the two periods (represented by the total area in grey in Figure 2). A net value of greater than 0 across the life span represents absolute expansion of morbidity over time; a net value less than 0 represents absolute compression. Between 1993 and 2023 absolute morbidity is projected to expand by 1.22 years. $RLHE_0^t$ is projected to increase from 9.3% in 1993 to 10.0% in 2023,

indicating a relative expansion of morbidity of 0.7% of the total life span over this period.

Decomposition of the projected absolute expansion in morbidity between 1993 and 2023 (the grey area in Figure 13) by age, cause and severity is presented in Table 11. Overall, declining trends in mortality across all ages, but particularly between ages 75 and 94, contributed to an expansion in morbidity of 1.70 years over the study period. This was offset by declining trends in disability below the age of 85, which contributed to morbidity compression of 0.49 years. Increasing trends in morbidity between ages 85 and 99 contributed to a slight expansion in morbidity of about 0.01 year. The combined effect of trends in both mortality and disability below age 70 was a compression of morbidity of 0.14 years. From age 70, the effect was a net expansion of morbidity of 1.36 years.

Figure 13: Cumulative survival with disability, Australia, 1993 and 2023

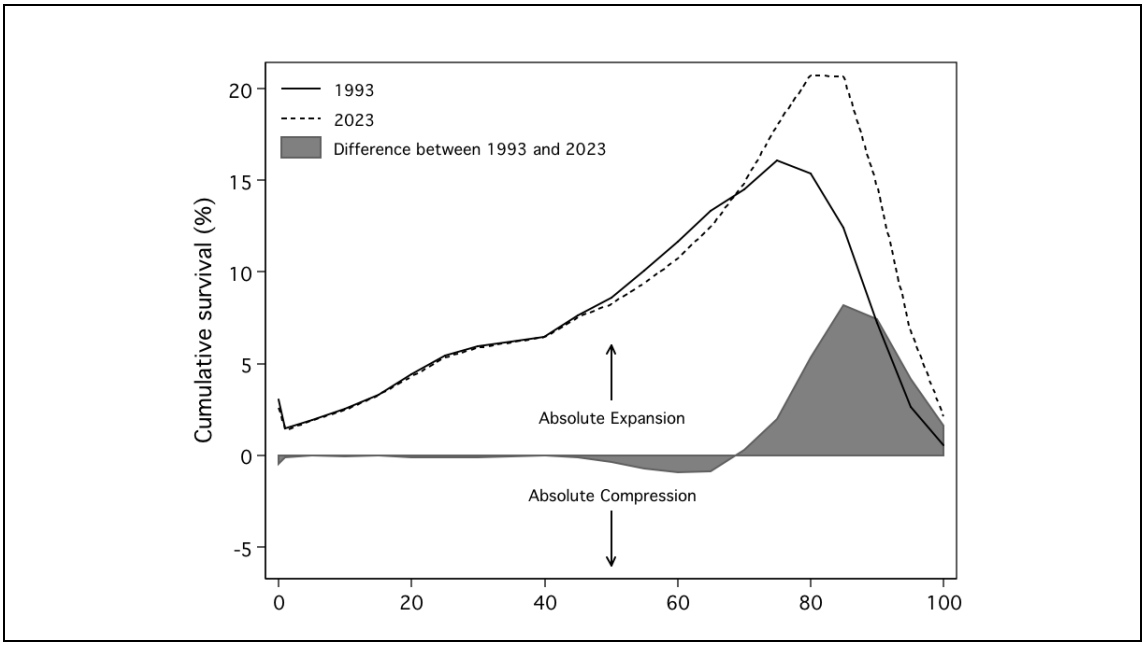


Table 11: Decomposition of projected absolute expansion of morbidity by age, cause and severity, Australia, 1993 to 2023

	Mortality		Disability		Net
	Rate	$\Delta ALHE_0$	Rate	$\Delta ALHE_0$	$\Delta ALHE_0$
	%Δpa	Years (%)	%Δpa	Years (%)	Years (%)
All causes	-2.0	1.70 (100.0)	-0.2	-0.48 (100.0)	1.22 (100.0)
Decomposition by age group					
0 to <50	-1.6	0.02 (1.4)	0.0	-0.06 (12.6)	-0.04 (-2.9)
50 to <60	-3.2	0.03 (1.9)	-0.3	-0.08 (16.2)	-0.05 (-3.7)
60 to <70	-2.9	0.11 (6.4)	-0.5	-0.17 (35.8)	-0.06 (-5.2)
70 to <80	-2.3	0.33 (19.5)	-0.3	-0.16 (33.2)	0.17 (14.1)
80 to <90	-1.3	0.67 (39.7)	0.0	-0.02 (3.5)	0.66 (53.8)
90 to <100	-0.7	0.42 (24.7)	0.1	0.01 (-2.5)	0.43 (35.4)
100+	-3.1	0.11 (6.4)	-0.3	-0.01 (1.1)	0.10 (8.5)
Decomposition by cause group					
Infectious	-0.1	0.00 (0.2)	0.0	0.00 (0.2)	0.00 (0.2)
Acute respiratory	3.1	-0.05 (-2.7)	0.0	0.00 (-0.1)	-0.05 (-3.8)
Maternal	-1.5	0.00 (0.0)	0.0	0.00 (0.0)	0.00 (0.0)
Neonatal	-3.8	0.02 (1.1)	0.0	0.00 (-0.1)	0.02 (1.6)
Nutritional	-2.9	0.00 (0.1)	0.0	0.00 (0.0)	0.00 (0.2)
Cancer	-1.3	0.32 (19.1)	-1.6	-0.19 (39.4)	0.14 (11.2)
Other neoplasms	-0.7	0.00 (0.2)	-0.3	0.00 (0.3)	0.00 (0.2)
Diabetes	-0.8	0.02 (1.1)	1.4	0.23 (-48.7)	0.25 (20.6)
Endocrine	-1.2	0.01 (0.6)	0.3	0.03 (-6.0)	0.04 (3.2)
Mental	-1.3	0.01 (0.8)	0.0	0.00 (0.1)	0.01 (1.1)
Neurological	-0.1	0.00 (0.1)	0.1	0.03 (-6.5)	0.03 (2.8)
Cardiovascular	-3.8	1.13 (66.6)	-1.8	-0.37 (78.1)	0.76 (62.1)
Chronic respiratory	-1.2	0.07 (4.1)	-0.6	-0.12 (25.1)	-0.05 (-4.1)
Digestive	-2.3	0.04 (2.5)	-0.1	0.00 (-0.1)	0.04 (3.5)
Genitourinary	0.0	0.00 (0.0)	0.0	0.01 (-1.6)	0.01 (0.7)
Skin	-0.1	0.00 (0.0)	0.0	0.00 (0.4)	0.00 (-0.1)
Musculoskeletal	-1.2	0.01 (0.3)	0.0	0.00 (-0.3)	0.01 (0.6)
Congenital	-2.6	0.02 (0.9)	-0.1	0.00 (0.8)	0.01 (1.0)
Oral	0.0	0.00 (0.0)	0.0	0.00 (0.1)	0.00 (0.0)
Ill defined	-7.2	0.01 (0.5)	0.0	0.00 (0.0)	0.01 (0.7)
Injuries	-1.3	0.07 (4.3)	-0.9	-0.09 (18.8)	-0.02 (-1.4)
Decomposition by severity group					
0 to <0.1	-0.7	0.07 (4.4)	0.3	0.28 (-58.8)	0.35 (29.1)
0.1 to <0.2	-2.6	0.99 (58.2)	-0.5	-0.42 (88.3)	0.57 (46.4)
0.2 to <0.3	-1.6	0.33 (19.3)	-0.4	-0.12 (24.7)	0.21 (17.2)
0.3 to <0.4	-2.4	0.27 (16.0)	-0.7	-0.18 (38.6)	0.09 (7.2)
0.4 to 1	-0.8	0.04 (2.2)	-0.2	-0.03 (7.2)	0.00 (0.2)

Note: column totals may not add due to rounding; $\Delta ALHE_0$ = change in absolute lost health expectancy at birth between 1993 and 2023.

Cardiovascular disease, diabetes and cancer together contributed 1.14 years (93.8%) to the projected 1.22-year expansion of morbidity between 1993 and 2023. A decline in cardiovascular disease mortality accounted for 1.13 years of this expansion but was offset by more modest declines in cardiovascular disease disability, resulting in a net contribution from this cause of 0.76 years (62.1%). The 0.25-year (20.6%) net contribution from diabetes, on the other hand, was almost entirely due to increasing disability from this cause, with modest declines in diabetes mortality expected to make only a 0.02-year contribution to total morbidity expansion. Cancer is expected to make a net contribution of 0.14 years (11.2%), with the 0.32-year contribution from declines in cancer mortality being partially offset by declines in disability from this cause.

Discussion

This analysis indicates that the most likely consequence of a continuation of recent trajectories in levels of mortality and morbidity in Australia will be a modest expansion of morbidity over the life course. This expansion will be manifest in both an absolute and a relative sense; in other words, time spent with disability will most likely increase both in terms of numbers of years lived and as a proportion of the average life span. Underlying this dynamic is the expectation that overall levels of mortality will continue decreasing at a faster rate than overall levels of disability (-2.0% and -0.2% per annum, respectively), resulting in gains in total life expectancy that exceed gains in health-adjusted life expectancy (0.28% and 0.26% per annum, respectively).

The analysis also indicates that the vast majority of projected changes to levels of morbidity over the life course will most likely hinge on a small group of chronic diseases: cardiovascular disease, diabetes and cancer (particularly lung cancer). In two out of three of these diseases, the prevailing trends in both mortality and disability are favourable; these can rightly be regarded as twentieth century public health success stories. In fact, the only unfavourable trend of any note is the increase in disability from diabetes, which is based on the expectation that recent failures to curb rising levels of obesity will continue. Mortality from diabetes, on the other hand, is expected to decline in line with declining cardiovascular disease mortality since most mortality from diabetes has vascular origins.

The strength of this analysis is that it quantifies for a comprehensive range of diseases and injuries of public health importance the impact of trends in both fatal and non-fatal health outcomes using a standardise analytical framework and uniform methods. In addition, it represents one of the first attempts to analyse temporal dynamics in HALE in terms of age, cause and impact (fatal and non-fatal) within an additive decomposition framework, as opposed to the single cause deletion approach. The availability of a structured database in which each of these dimensions was represented greatly facilitated the analytical processes.

The limitation of the analysis, of course, is that the underlying data set relied on a range of simplifying assumptions that cannot possibly reflect the full range of population health dynamics over the projection period. Notwithstanding this important caveat, however, the analysis is illustrative in several respects.

First, the results undermine support for a popular interpretation of Fries' hypothesis that populations, even in advanced stages of the epidemiologic transition, experience a compression of morbidity. This refutation is, of course, qualified since the underlying data set did not include assumptions about changes in levels of severity for a particular cause arising from, for example, technological innovations; rather, these assumptions were limited to expected changes in the underlying epidemiological parameters. Nevertheless, given expected declines in mortality outpaced expected declines in morbidity by a factor of 10, the impact of technology would have to very large for gains in health-adjusted life expectancy to catch up with life expectancy. Varying the severity weights within plausible bounds would shed further light on this dimension of the results.

Second, the results provide qualified support for Manton's theory that the proportion of the life span with serious illness stabilises or decreases with decreasing mortality, whereas the proportion with less severe illness increases. This is evident in the results from the decomposition analysis by severity group, which indicated that over three-quarters of the absolute expansion in morbidity over the projection period was accounted for by low severity causes (i.e. those causes with severity weight of less than 0.2). By far the greatest contributor was the 0.1 to <0.2 severity group, which together accounted for 46.4% of morbidity expansion and was driven by much faster declines in rates of mortality than disability over this period (-2.6% and -0.5% per

annum, respectively). The second largest was the zero to <0.1 severity group (29.1%), although the dominant driver in this case was a modest increase in rates of morbidity of 0.3% per annum, with the -0.7% per annum decline in mortality having only a minor effect. Again, a sensitivity analysis of the severity weight assumptions would shed further light on these dynamics.

The results also have implications for how epidemiology should be used to explain health system demand pressures. Correctly understood, the role of cardiovascular disease and cancer—both diseases typically portrayed as central to these pressures[95]—has been to increase demand, but not because the underlying risks associated with these causes is increasing. In fact, the risk of disability and mortality from both is *decreasing*. Rather it is because these declines have already had such a profound impact on life expectancy that people are now living to ages where they experience the effects of degenerative diseases of old age (such as sensory and musculoskeletal disorders, dementias and Parkinson's disease) for which risk has remained relatively stable but for which prevalence is growing as a result of the increasingly greater proportion of the population living at older ages. These are, in broad terms, the dominant epidemiological drivers of health system demand in a post transition population.

Of course there are other non-epidemiological drivers as well, such as trends in fertility, technological innovation and increasing wealth. As such, the tendency in policy discourse to portray prevention as having untapped potential to curb demand pressures arising from the chronic disease epidemic that is engulfing the health system, while rhetorically appealing, is nevertheless inconsistent with the evidence and thus not very helpful. In fact, such efforts will, if the past is any guide, most likely result in a further expansion of morbidity hence demand simply by continuing the tendency for the health system as a whole to generate faster declines in mortality than in disability. The dynamic at play here is that past successes have for the most part been confined to causes with a predominantly fatal impact (e.g. cardiovascular disease, cancer) whereas health service demand is driven by the prevalence of disability more broadly.

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[95] National Health Priority Action Council, *National Chronic Disease Strategy*, Australian Government Department of Health and Ageing, Canberra, 2006.

The most notable exception to these general trends is diabetes, which is expected to account for around one-fifth of the total expansion in morbidity over the projection period. This is driven almost entirely by the expected 1.8% per annum increase in risk of disability, with the -0.7% per annum decline in risk of mortality having a much smaller but nevertheless still expansionary impact. Thus diabetes is a key area where prevention as a demand mitigation strategy might have potential. However, efforts to prevent further growth in levels of obesity, the key risk factor for diabetes, have largely been unsuccessful and it seems likely that the overall expansionary impact of this disease will continue in the absence of a much greater commitment to developing the evidence base on how to implement effective obesity control.

This analysis also has more fundamental philosophical implications. It should be of general concern, for example, that one of the returns on our increasing investments in health relative to both total government expenditure and the rest of the economy appears to be a modest expansion of morbidity over the life course. This observation is only partially tempered by the related finding that we are, on average, getting healthier in absolute terms (i.e. the length of the life course is increasing). Perhaps the greatest policy challenge over the coming decades, therefore, will be to articulate the benefits of an increasingly costly health system adept at adding years to life but not necessarily life to years, particularly amongst those with degenerative diseases of old age.

Chapter 5: An alternative approach to projecting health expenditure in Australia[96]

Introduction

Improvements in health, particularly among the elderly, have been an important consequence of economic development. 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. Interest is increasing across the developed world in the long-term sustainability of public finances in the context of these widespread demographic trends.

In most countries with time series data, expenditure on health has increased substantially across all components of the health system beyond what can be explained by changing age structure and size of the population alone. The main non-demographic factors influencing these trends are: new technologies, such as diagnostics, drugs or procedures; changing medical practice and policy; the organisation and financing of the health care system; the intensity or coverage of health services; the greater rate of increase in health prices compared with general prices (excess health inflation); and changes in population health status.

The influence of non-demographic factors is unlikely to be uniform across the health care system. Furthermore, the impact of these factors is likely to vary over time depending on the type of health service and the particular health problem it addresses. Taking into account such detail when projecting health expenditure would be impractical. Instead, analysts have tended to extrapolate from observed trends in expenditure growth for aggregate categories of expenditure.

A common approach has been to apply growth factors for the combined effect of the non-demographic growth in health expenditure over time without necessarily making explicit the identified non-demographic growth factors.[97] A few of these studies have considered overall

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[96] This chapter was first published as Begg, S, Vos, T, Goss, J and Mann, N, "An alternative approach to projecting health expenditure in Australia". See Declarations and Statements for the respective contributions of each author.

[97] ABS, *Population Projections Australia 2002 to 2101*, (Cat. No. 3222.0), Australian Bureau of Statistics, Canberra, 2003; de Hollander, AEM, Hoeymans, N, Melse, JM, J.A.M., vO and Polder, JJ, *Zorg voor gezondheid: Volksgezondheid Toekomst Verkenning 2006*, Rijksinstituut voor Volksgezondheid en Milieu, Bilthoven, the Netherlands, 2006; Economic Policy Committee and the European Commission (DG ECFIN), *The impact of ageing on public expenditure: projections for the EU-25 Member States on pensions, healthcare, long-term care,*

changes in population health status by making assumptions about the increase in healthy years lived as life expectancy increases.[98] Some have also included an adjustment to account for the fact that many health resources are used in the last year of life[99]; improvements in life expectancy shift period to older ages.[100]

Two health expenditure projection models have explicitly accounted for changes in population health status in greater detail.[101] The first model projected public health expenditure in The Netherlands based on historical expenditure by disease, age and sex, and epidemiological projections of incidence and prevalence for 52 disease groups. This study did not quantify the contribution of disease trends to expected changes in health expenditure. The second model projected health expenditure in Australia for nine disease groups in a pilot study covering less than half of all health expenditure. This study did not quantify the contribution of changes in population health status to expected changes in total health expenditure.

The aim of this chapter is to introduce a body of work undertaken by researchers at the University of Queensland's School of Population Health and the Australian Institute of Health and Welfare that applied the methods developed for the pilot study to all components of health expenditure.[102] This work provides an alternative analysis of future health expenditure in Australia by incorporating likely changes in both the epidemiology of disease and injury, and the volume of health service delivery for a comprehensive set of health outcomes over the period 2003–2033.

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education and unemployment transfers (2004–50). 2006; OECD, *Projecting OECD health and long-term care expenditures: What are the main drivers?,* Organisation for Economic Co-operation and Development, Paris, 2006, viewed 16 August 2011, http://www.oecd.org/dataoecd/57/7/36085940.pdf; United Nations, *World Population Ageing 1950-2050*, 2002; Vos, T, Goss, J, Begg, S and Mann, N, *Australian Burden of Disease and Injury Study: Projected health care costs report*, University of Queensland, Brisbane, Australia, 2005.

98 Economic Policy Committee and the European Commission (DG ECFIN), *The impact of ageing on public expenditure: projections for the EU-25 Member States on pensions, healthcare, long-term care, education and unemployment transfers (2004–50)*; OECD, *Projecting OECD health and long-term care expenditures: What are the main drivers?*

99 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Economic Policy Committee and the European Commission (DG ECFIN), *The impact of ageing on public expenditure: projections for the EU-25 Member States on pensions, healthcare, long-term care, education and unemployment transfers (2004–50)*; United Nations, *World Population Ageing 1950-2050*.

100 AIHW, *Health system expenditure on disease and injury in Australia, 2000-01*, (Cat. No. HWE 28), Australian Institute of Health and Welfare, Canberra, 2005; Mathers, C, Penm, D, Carter, C and Stevenson, C, *Health system costs of diseases and injury in Australia 1993-94*, (Health and Welfare Expenditure Series No. 2), Australian Institute of Health and Welfare, Canberra, 1998.

101 de Hollander, AEM, Hoeymans, N, Melse, JM, J.A.M., vO and Polder, JJ, *Zorg voor gezondheid: Volksgezondheid Toekomst Verkenning 2006*; Vos, T, Goss, J, Begg, S and Mann, N, *Australian Burden of Disease and Injury Study: Projected health care costs report*.

102 United Nations, *World Population Ageing 1950-2050*.

Methods

Separate projections were calculated for each health condition (or group of conditions in some cases) and type of expenditure (hospital care, medical services, pharmaceuticals, aged care homes and other health services). Analyses accounted for expected changes in the number of affected cases (*epidemiology*), the proportion of cases treated (*treatment proportion*), the volume of health services per treated case (*treatment volume*), and excess health price inflation (*price*). Numbers of cases were calculated to be a function of changes in population size and age structure, as well as trends in epidemiology. A brief overview of the data sources and methods used to derive each of these components follows. A more detailed account is available as a background paper for the United Nations World Economic and Social Survey 2007.[103]

Population size and age structure

Population projections were obtained from the Australian Bureau of Statistics (ABS) *Series 8* population projections.[104] This series is based on the 2001 census and assumes high net overseas migration (125,000 annually), constant improvements in life expectancy (low mortality assumption), and total fertility declining to a rate of 1.6 by 2011 and then remaining constant.

Incidence and prevalence[105]

Estimates of incidence and prevalence were obtained from the Australian Burden of Disease and Injury 2003 study. Methods and assumptions for these estimates are described in detail elsewhere.[106] The key analytical steps were:

1. Baseline models specifying the complete epidemiology for over 180 diseases and injuries in Australia for the year 2003 were developed using a large range of data sources, methods and assumptions.

2. Trends in observed cause-specific mortality over the period 1979–2003 were analysed and projected into the future using a combination of regression techniques.

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103 ibid.
104 ABS, *Population Projections Australia 2002 to 2101*.
105 See Chapter 2 for further details.
106 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*.

3. Hazards for fatal conditions were extrapolated backwards and forwards from baseline using assumptions about the relative contribution of incidence and case-fatality to changes in cause-specific mortality (both observed and projected). For non-fatal conditions, incidence was the only hazard for which extrapolations were made.

4. The epidemiology of each condition was estimated in a temporal model that accounted for changes in all-cause mortality as well as changes in incidence and case-fatality (where appropriate) at all points throughout the projection period.

5. Absolute numbers of incident and prevalent cases were derived by applying the population rates from the above analyses to projected population estimates.

Treatment proportion and treatment volume

Comparable per-unit health care costs by health condition and type of expenditure (hospital care, medical services, pharmaceuticals, aged care homes and other health services) were available for two time periods (1993–94 and 2000–01) from previous work.[107] These estimates were divided by epidemiological estimates for the same years to derive estimates of treatment proportion and treatment volume for each health condition, type of expenditure and time period. Expected future changes in these parameters were extrapolated from the observed changes between 1993–94 and 2000–01.

For most conditions, prevalent cases were used to derive these parameters because total expenditure for a condition was assumed to be primarily influenced by the number of people with the condition at a point in time. For cancer, incident cases were used because most expenditure for cancer occurs in the first year after diagnosis. For ischaemic heart disease and stroke, expenditure on admitted patients was derived from incident cases, while medical and pharmaceutical expenditure was derived from prevalent cases. For some conditions the data for the period 1993–94 to 2000–01 were deficient so that valid trends in treatment proportion and volume could not be estimated. In this case a standard growth in treatment volume of 2.5% per 5 years was assumed.

Judgement was used to adjust observed trends in treatment proportion and treatment volume that appeared unusual and could not be explained, or were considered unsustainable into the

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[107] AIHW, *Health system expenditure on disease and injury in Australia, 2000-01*; Mathers, C, Penm, D, Carter, C and Stevenson, C, *Health system costs of diseases and injury in Australia 1993-94*.

future.[108] Lipid lowering drugs for the prevention of cardiovascular disease, for example, experienced a large increase in expenditure over the period 1993–94 and 2000–01. When these drugs go off patent it is likely that per unit pharmaceutical costs for this disease will decrease.

Price

The *price* factor is the amount by which health prices are expected to exceed general inflation in the economy. This is often called excess health price inflation. In the period 1993–94 to 2000–01 excess health price inflation averaged 0.73% per year, with small variations across areas of expenditure. In this model, excess health price inflation was assumed to increase into the future at the average rate of 0.73% across all areas of expenditure except dental services where a higher rate of 2.0% per was assumed. It would be desirable in future models to vary this assumption for more areas of expenditure. For example, an important source of uncertainty in this analysis is the impact on pharmaceutical prices of patent expiry and the growing use of cost control measures.

Decomposition of factors

Decomposition of the respective contribution of each of the factors in the projection model to changes in total health expenditure should account for expected interaction between factors. A simplified model was adopted in which interaction effects were allocated to each of the six factors in the analysis in proportion to the ratio of the sixth root of a particular factor to the sixth root of all factors combined.

Comparisons with other studies

Comparisons with other studies that use different projection periods was achieved by annualising projected growth in health expenditure and expressing this as percentage points above projected growth in gross domestic product (GDP).

Results

Total health expenditure in Australia is expected to increase from $71.4 billion in 2002–03 to $162.3 billion in 2032–33, an increase of 127.4% or $90.9 billion (Table 12). On the basis of

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[108] The term judgement in this context refers to the process whereby commonsense was applied when determining model parameters.

Australian Treasury estimates, GDP will increase by 96.9% over the same period, meaning that health expenditure is expected to increase from 9.4% of GDP in 2002–03 to 10.8% of GDP in 2032–33. This represents an increase of 14.9% in the *health to GDP* proportion, or an annual growth of 0.5% greater than growth in the economy more generally.

Table 12: Projected total health expenditure (2002–03 dollars), Australia, 2002–03 to 2032–33

	Year				Change
	2002–03	2012–13	2022–23	2032–33	2003–2033 (%)
Total health expenditure ($ billion)	71.4	91.7	122.2	162.3	127.4
Gross domestic product[a] (GDP; $ billion)	762.0	995.0	1,230.0	1,500.0	96.9
Total health expenditure as a proportion of GDP (%)	9.4	9.2	9.9	10.8	14.9

Notes: (a) Sourced from the Australian Treasury.

Neurological and sense disorders—mostly dementia and Parkinson's disease—are expected to experience the greatest absolute growth in expenditure over the projection period, followed by cardiovascular disease and dental services (Table 13). The expected growth for cardiovascular disease is due to a $5.0 billion increase in expenditure on treatment services and a $3.3 billion increase in expenditure on prevention efforts (mainly blood pressure lowering drugs and lipid lowering drugs). Diabetes is expected to experience the greatest proportional increase in expenditure over the projection period, followed by neurological disorders, musculoskeletal conditions and dental services.

Table 13: Projected total health expenditure (2002–03 dollars) by cause, Australia, 2002–03 to 2032–33

	Expenditure by year ($ billion)		Change 2003 to 2033	
Cause	**2002–03**	**2032–33**	**$ billion**	**%**
Cardiovascular	7.9	16.2	8.3	104.7
Treatment	4.5	9.4	5.0	111.1
Prevention	3.4	6.8	3.3	96.2
Respiratory	5.9	12.6	6.7	113.0
Chronic obstructive pulmonary disease	0.6	0.8	0.2	35.3
Other respiratory	5.3	11.8	6.5	121.8
Injuries	5.6	9.4	3.8	67.4
Dental	5.1	12.4	7.3	144.0
Mental	4.3	8.5	4.2	97.1
Digestive	4.0	9.7	5.6	139.0
Neurological	4.0	15.1	11.1	279.9
Dementia and Parkinson's	3.5	13.9	10.4	294.3
Other neurological	0.5	1.2	0.8	167.8
Musculoskeletal	3.7	9.9	6.1	163.5
Genitourinary	3.1	6.8	3.7	122.2
Cancer	2.8	5.2	2.4	84.0
Sense disorders	2.3	5.1	2.8	124.4
Endocrine, nutritional and metabolic	2.2	4.1	2.0	91.2
Skin	2.0	4.5	2.5	127.4
Maternal	1.8	2.5	0.7	40.9
Infectious	1.5	2.7	1.2	74.6
Diabetes	1.4	7.0	5.6	400.8
Neonatal	0.5	0.7	0.2	41.7
Congenital	0.3	0.4	0.1	55.3
Other	13.0	29.6	16.6	127.4
Total health expenditure	71.4	162.3	90.9	127.4

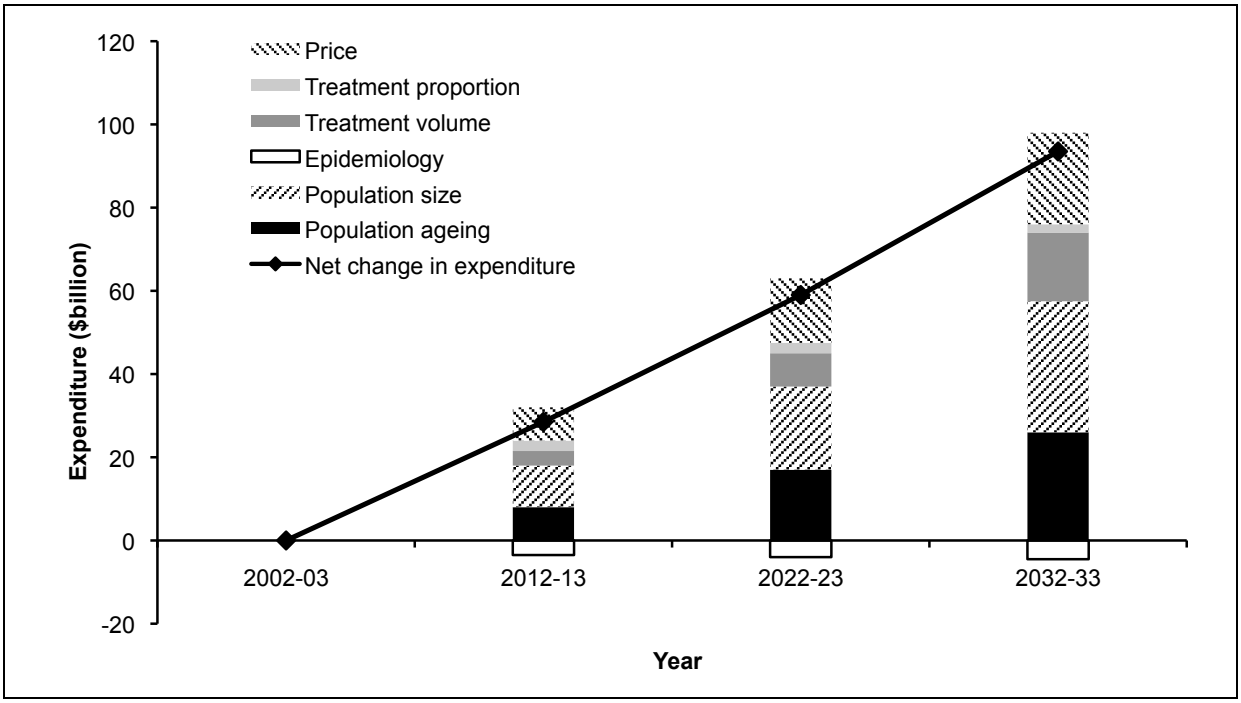
Admitted patient services are expected to experience the greatest absolute growth in expenditure over the projection period, followed by other health services and pharmaceutical scripts (Table 14). Residential aged care expenditure is likely to show the greatest proportional increase in expenditure, followed by pharmaceutical expenditure. Expenditure on admitted patients in hospitals is expected to show a similar growth to health expenditure as a whole, while medical services expenditure will experience a somewhat lower growth.

Table 14: Projected total health expenditure (2002–03 dollars) by area of expenditure, Australia, 2002–03 to 2032–33

	Expenditure by year ($ billion)		Change 2003 to 2033	
Expenditure area	**2002–03**	**2032–33**	**$ billion**	**%**
Admitted patient services	23.4	51.7	28.3	121.0
Medical services	14.6	28.7	14.1	96.5
Pharmaceutical prescriptions	10.0	24.5	14.5	145.2
Residential aged care (high care)	4.9	16.8	11.9	241.9
Other health services	18.5	40.6	22.1	120.0
Total health expenditure	71.4	162.3	90.9	127.4

Decomposition analysis shows that population ageing and increases in population size are likely to account for two-thirds of the expected $90.9 billion increase in total health expenditure over the projection period ($29.4 billion and $28.4 billion, respectively) (Figure 14). Excess health price inflation ($19.1 billion), changes in treatment volume (number of health services provided) per case ($14.0 billion) and, to a lesser extent, treatment proportion ($1.3 billion) also contribute to this increase.

Figure 14: Decomposition of factors leading to projected change in total health expenditure, Australia, 2002–03 to 2032–33



Favourable trends in the epidemiology of cardiovascular disease, chronic obstructive pulmonary disease (COPD), cancers and injuries are expected to decrease overall expenditure by $5.0 billion. This reduction will be offset by large increases in diabetes and other diseases,

which are expected to result in a $3.7 billion increase in treatment expenditure. The net effect of epidemiological trends is expected to be a $1.3 billion reduction in total health expenditure over the projection period.

Annualised projected growth in health expenditure in this study is comparable to reported estimates for the European Union and New Zealand. Higher estimates are reported for Hong Kong, the United States and Australia in previous studies, with those for OECD (Organisation for Economic Co-operation and Development) countries lying in the middle of this range (Table 15).

Table 15: Health expenditure growth as percentage points above projected gross domestic product (GDP) growth for selected countries

Country	Annual growth in health expenditure (% above growth in GDP)
Australia	
Intergenerational Report	1.7
Productivity Commission	1.6
OECD	
Cost pressure scenario	0.9
Cost containment scenario	1.2
This study	0.5
Other countries	
USA	2.3
Hong Kong	1.9
OECD average	
Cost pressure scenario	1.4
Cost containment scenario	0.9
New Zealand	0.5
European Union countries	
Average	0.5
High (Spain)	0.6
Low (Portugal)	0.2

OECD = Organisation for Economic co-operation and Development

Discussion

This analysis suggests that total health expenditure in Australia will grow by 0.5% greater than growth in the economy, to 10.8% of GDP in 2032–33. Population ageing will account for 32.3% of this growth; and non-demographic factors (excess price inflation, treatment proportion and volume per case) a further 36.5%. The remaining 31.2% will be due to

increases in population size, a particular feature of a high immigration country such as Australia.

An annual growth of 0.5% greater than growth in GDP is comparable to estimates for the European Union and New Zealand but is lower than estimates for Hong Kong and US. Other estimates for Australia are not directly comparable as they relate to different projection periods or do not quantify expected changes in total health expenditure. The Australian Treasury, for example, estimated that federal government spending on health (including aged care) would grow by 1.7% greater than growth in the economy to 7.9% of GDP in 2032–33.[109] The Australian Government's Productivity Commission estimated that all government spending (federal, state and territory) would grow by 1.6% greater than growth in the economy to 9.4% of GDP in 2034–35.11 The OECD estimated that all government health expenditure in Australia would grow by 0.9% greater than growth in the economy to 8.5% of GDP in 2050 in a cost-containment scenario.[110] In a cost-pressure scenario, this growth was estimated to be 1.2%.

Few studies have explicitly commented on the relative contribution of demographic and non-demographic factors to growth in health expenditure. The OECD study estimated that the effect of population ageing would be about half the estimated effect of non-ageing residual factors in a cost-pressure scenario, but that these factors would contribute in equal proportions in a cost-containment scenario.[111] Similarly, the New Zealand study estimated that for the period 2020–2040—which is when the post-WWII generation will move into the very old ages—ageing would have a similar impact as non-demographic growth.[112] With the exception of the OECD cost-pressure scenario, these estimates are largely consistent with the findings reported here.

Variation in estimates of growth in health expenditure between different models is likely to reflect differences in underlying assumptions. Certain assumptions have only a small impact on projection estimates. The OECD and Productivity Commission models allowed for

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109 Australian Treasury, *Intergenerational Report 2002-03*, Australian Government, Canberra, 2002.
110 OECD, *Projecting OECD health and long-term care expenditures: What are the main drivers?*
111 ibid.
112 Johnston, G and Teasdale, A, *Population ageing and health spending: 50-year projections*, Ministry of Health, Wellington, 1999.

proximity to death costs but this had only a minor downward impact on projections.[113] The OECD model also assumed that years gained from improvements in life expectancy were equivalent to years in full health, an assumption that lowered estimates of growth by a small amount. Likewise, epidemiological trends, as this paper has shown, have only a marginal downward effect when the net impact across all conditions is considered.

Assumptions regarding non-demographic growth factors have a much greater impact. The OECD, Treasury and Productivity Commission all used estimates of non-demographic expenditure growth of around 2.6% per year compared with an average of around 1.2% per year in our study. The latter was calculated separately for each condition to explicitly account for condition-specific assumptions regarding excess health price inflation, volume per case and treatment proportion. Since excess health price inflation was set to be constant across conditions, much of the variability in expenditure estimates is due to differences in volume per case assumptions.

Changes in volume per case over time are largely influenced by the introduction of new technologies and changes in treatment practices. Volume per case assumptions in this paper were based on information from two time points 7 years apart. By quantifying changes in volume per case for each condition over this period, it was possible to ensure that trends in volume per case remained within plausible limits. There is uncertainty about whether trends observed over such a short period are likely to continue to influence expenditure in future years. There is likely to be greater uncertainty, however, around a single non-demographic growth estimate for all conditions, as has been assumed, either explicitly or implicitly, in other expenditure projection models.

An important by-product of this work is the quantification, for the first time, of a comprehensive description of likely future health expenditure in Australia by area of expenditure, health condition, age and sex. Researchers wanting to model the cost-effectiveness of treating health conditions under various intervention scenarios will find this a useful resource. Health planners concerned with the changing health service needs of Australia's ageing population may also find it of interest. The findings presented here, for example, show that there is likely be twice the growth in demand for residential age care

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[113] OECD, *Projecting OECD health and long-term care expenditures: What are the main drivers*; Productivity Commission, *Economic implications of an Ageing Australia*, Productivity Commission, Canberra, 2005.

services than there will be for admitted patient services over the next 30 years. Similarly, growth in demand for services from particular specialty areas such as diabetes, neurology and geriatrics is expected to outstrip growth in demand for other specialty areas such as paediatrics and gynaecology.

Australian's preparedness for the economic and social consequences of population ageing will be greatly enhanced by forward planning around the infrastructure and workforce needs that are likely to emerge over the coming decades. The analyses introduced here have the potential to make a valuable contribution to such debate.

Afterword

Following the publication of this paper by the *Australian Health Review*, one of my co-authors, Mr John Goss, revisited some of the parameter assumptions in the underlying expenditure model to derive an updated set of projections. This reanalysis was commissioned by the National Health and Hospitals Reform Commission (NHHRC) and was subsequently published by the AIHW as two separate reports,[114] both of which were used to inform the Commission's final recommendations.[115]

The key difference between the revised analysis and its predecessor is the higher rate of growth in total expenditure over the projection period (3.6% per annum compared to 2.8%), resulting in a 52.9% increase in projected change in expenditure from $105.3 billion to $161.0 billion. This was largely driven by a change in assumptions relating to volume per case treated, which resulted in a 444.1% increase in projected change in expenditure from $14.9 billion to $81.3 billion. However, offsetting some of this increase was a change in assumptions relating to per unit price, which resulted in a 30.3% decrease in projected change in expenditure from $21.0 billion to $8.8 billion.

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[114] AIHW, *Estimating the impact of selected National Health and Hospitals Reform Commission (NHHRC) reforms on health care expenditure, 2003 to 2033*, (Cat. no. HWE 45), Australian Institute of Health and Welfare, Canberra, 2009; Goss, J, *Projection of Australian health care expenditure by disease, 2003 to 2033*, (Cat no. HWE 43), Australian Institute of Health and Welfare, Canberra, 2008.

[115] NHHRC, *A healthier future for all Australians : final report : June 2009*, National Health and Hospitals Reform Commission, Canberra, 2009.

Table 16: Comparison between results published by the Australian Health Review and those published by AIHW

	Original ($ billion)	Revised ($ billion)	Difference (%)
Total expenditure (2006-07 dollars)			
2002-03	82.7	85.1	2.9
2032-33	188.0	246.1	30.9
Expenditure change	105.3	161.0	52.9
Decomposition of change			
Ageing	33.7	37.8	12.2
Population	32.7	34.4	5.1
Disease rate	-1.8	-2.3	25.3
Volume per case	14.9	81.3	444.1
Treatment	1.5	1.0	-30.3
Price	21.0	8.8	-58.0

This concludes Part II of this thesis. In Part III, I present two additional papers, this time to illustrate the potential policy relevance of the subpopulation disaggregation methods proposed in Chapter 2.

Part III: Policy applications of proposed subpopulation methods

Chapter 6: Burden of disease and injury in Australia in the new millennium: measuring health loss from diseases, injuries and risk factors[116]

Introduction

Information on the magnitude and distribution of health problems in a population is important for health policy decision-making. Popular epidemiological measures such as mortality, incidence and prevalence are available for many health problems, but cannot be compared across causes as comprehensive indicators of population-level health. Summary measures of population health, on the other hand, extend the utility of descriptive epidemiology by combining information on mortality and non-fatal health problems into a common measure that can be used to provide a comprehensive picture of the health status of a population.[117]

We present here a reanalysis of a large body of work that used summary measures to describe the health of Australians in the new millennium.[118] The research on which it is based follows a comparable study for the year 1996 reported in the *Medical Journal of Australia* in 2000.[119] Both studies use a particular summary measure—the *disability-adjusted life year* (DALY)—to quantify health loss from a comprehensive set of diseases, injuries and health risks of public health importance in Australia. The DALY, in turn, has its origins in an assessment of global health for the World Bank.[120] One undiscounted DALY is equivalent to one lost year of healthy life and represents the gap between current health status and an ideal situation of

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116 This chapter was first published as Begg, SJ, Vos, T, Barker, B, Stanley, L and Lopez, AD, "Burden of disease and injury in Australia in the new millennium: measuring health loss from diseases, injuries and risk factors". See Declarations and Statements for the respective contributions of each author.

117 Murray, C, Salomon, J, Mathers, C and Lopez, A, eds, *Summary measures of population health: concepts, ethics, measurement and applications*.

118 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*.

119 Mathers, CD, Vos, ET, Stevenson, CE and Begg, SJ, "The Australian Burden of Disease Study: measuring the loss of health from diseases, injuries and risk factors".

120 Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I*; Murray, CJL and Lopez, AD, eds, *Global Health Statistics: a compendium of incidence, prevalence and mortality estimates for over 200 conditions. Vol II.*

the whole population living into old age in full health.[121] This gap is referred to here as *health loss*, rather than the less accurate but more commonly used term 'burden of disease'.

The DALY combines the descriptive epidemiology of each health condition of interest with a multidimensional numerical weighting for the severity of that condition. As the weighting given to each dimension implies a judgement about its relative importance to the total measure, the DALY has obvious normative characteristics that make it not necessarily compatible with other classifications of health (e.g., WHO's International classification of functioning, disability and health[122]). For this reason, others have highlighted the importance of limiting interpretation of the DALY to the specific purposes for which it is being used[123]—which, in this case, is as a comparative measure of health loss.

Our article provides an assessment of the magnitude and distribution of health problems in Australia in order to identify key opportunities for health gain. Our specific objectives were to calculate:

1. DALYs by cause, age and sex for the year 2003;
2. DALYs attributable to past and current exposure to major modifiable health risks;
3. Differentials in DALY rates between subpopulations (e.g. between state and territory jurisdictions, socioeconomic groups, and remoteness categories[124]); and
4. DALYs projected 10 and 20 years beyond 2003.

Methods

Health loss was estimated for a comprehensive set of diseases and injuries of public health importance in Australia, using DALYs as the outcome measure. Diseases and injuries were the smallest reported unit of disaggregation, and are referred to here as *specific causes* or *conditions*. Each is mutually exclusive and belongs to one of 22 *broad cause groups*, most of

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121 The published version of this sentence used the more common explanation, "one DALY is equivalent to one lost year of healthy life ". This explanation is misleading when used in reference to discounted DALYs.

122 International Classification of Functioning, Disability and Health (ICF), World Health Organization, Geneva, 2011, viewed 16 August 2011, http://www.who.int/classifications/icf/en/.

123 AIHW, *Disability and its relationship to health conditions and other factors*, (AIHW Cat. No. DIS 37), Australian Institute of Health and Welfare, Canberra, 2004.

124 Indigeneity was another key subpopulation grouping, although the relevant analyses were not complete at the time of publication.

which correspond to chapter-level headings of the International classification of diseases.[125] Each broad cause group, in turn, belongs to one of three broad clusters: communicable, maternal, neonatal and nutritional conditions; non-communicable diseases; and injuries. A brief overview of methods and assumptions is provided below.

Baseline models

Baseline models describing the epidemiology of each specific cause for Australia in 2003 were developed using a range of data sources, methods and assumptions.[126] Typical inputs included prevalence (from surveys), incidence (from disease registers), case fatality (from cohort studies), remission (from cohort and intervention studies), clinical judgement, and information about changes over time in any of these variables. Complete and internally consistent cross-sectional epidemiological models were derived from three of these inputs using modelling software.

Epidemiological trends[127]

Trends in observed cause-specific mortality over the period 1979–2003 were analysed and projected to 2023 using a combination of regression techniques. Transition hazards for conditions that cause mortality were extrapolated from baseline using assumptions about the relative contribution of incidence and case fatality to changes in cause-specific mortality. For non-fatal conditions, incidence was the only transition hazard for which extrapolations were made. Estimates for each specific cause through time were calculated in a model that accounted for changes in all-cause mortality as well as changes in incidence and case fatality (where appropriate) at all points throughout the study period. Absolute numbers of incident and prevalent cases were derived by applying the population rates from these analyses to Australia Bureau of Statistics population projections.[128]

DALY estimates

DALYs were calculated by applying severity weights (range, 0–1) to the estimated number of incident cases and average duration for each condition. Weights were derived from two

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125 International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, World Health Organization.

126 See Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*.

127 See Chapter 2 for further details.

128 ABS, *Population Projections Australia 2002 to 2101*.

sources[129], with extrapolations based on alternative methods in some cases. Adjustments were made to account for the possibility of two or more conditions occurring simultaneously in the same person, either by chance or because the conditions are related. These corrections were achieved by determining numbers of people for every combination of causes of ill health as measured by various surveys and hospital admission data.[130]

Health risk assessment[131]

Past and current exposure to 14 selected risk factors (listed in Table 19) were analysed for their contribution to health loss in 2003. Analyses were based on the theoretical framework developed for the WHO-initiated Comparative Risk Assessment project.[132] This approach incorporates a 'hypothetical minimum' as the alternative exposure distribution against which health loss is calculated, and uses continuous rather than categorical measures of exposure where appropriate. Results were also calculated for the combined effect of health risks.

Health differentials

Health differentials were assessed by comparing subpopulation-specific age-standardised DALY rates derived from disaggregated national DALY estimates. Disaggregation was achieved in two stages, whereby condition-specific estimates of incidence and mortality were first apportioned to states and territories and then to a 15-cell matrix of subpopulations. The matrix was composed of three remoteness categories (major cities, regional areas and remote areas) by five socioeconomic quintiles within each jurisdiction.

To disaggregate conditions with a predominantly fatal impact, preference was given to mortality data. For the remaining conditions, preference was given to the data source on which the baseline model was based (e.g., hospital data, health survey data). Condition-

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129 Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I*; Stouthard, ME, Essink-Bot, M, Bonsel, GJ, Barendregt, JJ, Kramers, PGN, van de Water, HPA, Gunning-Schepers, LJ and van der Maas, PJ, *Disability weights for diseases in The Netherlands*.

130 See Chapter 2 for further details.

131 For the purposes of this thesis, comparative risk assessment is regarded as a secondary analysis for applying established models to a complete set of framework outputs. While the comparative risk assessment in the case study was facilitated by the workflows outlined in Chapter 2, the underlying analytical modelling was largely derivative of other work. For further details, see Appendix 2 of Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*.

132 Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 1*; Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 2*.

specific estimates of prevalence and duration for each subpopulation were derived from subpopulation-specific incidence and all-cause mortality rates, as well as national assumptions regarding remission and case fatality. Subpopulation-specific DALYs were calculated using comorbidity-corrected national severity weights.

Results

Key findings are presented here at two levels of aggregation: *broad cause groups* and *specific conditions*. Both levels are referred to as *causes* and are ranked in terms of *leading* causes compared with others at the same level of aggregation.

Leading broad cause groups

Total health loss in Australia in 2003 was 2.63 million DALYs or 132 DALYs lost per 1000 people. Fifty-one per cent of the loss was from non-fatal causes. Over 75% was accounted for by the six leading broad cause groups: cancer, cardiovascular disease, mental disorders, neurological and sense organ disorders, chronic respiratory diseases, and injuries (Table 17).

Table 17: Health loss, by broad cause group, Australia, 2003

	Rate/1000 people (%)		
Broad cause group	**Non-fatal health loss[a]**	**Fatal health loss[b]**	**Total health loss[c]**
Cancers	4.4 (6.5)	20.7 (32.2)	25.1 (19.0)
Cardiovascular disease	5.3 (7.7)	18.6 (28.9)	23.8 (18.0)
Mental disorders	16.5 (24.2)	1.2 (1.8)	17.6 (13.3)
Neurological and sense organ disorders	13.0 (19.1)	2.7 (4.2)	15.7 (11.9)
Chronic respiratory diseases	5.8 (8.5)	3.6 (5.6)	9.4 (7.1)
Injuries	2.2 (3.2)	7.1 (11.0)	9.3 (7.0)
Diabetes mellitus	5.6 (8.2)	1.6 (2.5)	7.2 (5.5)
Musculoskeletal diseases	5.0 (7.3)	0.4 (0.5)	5.3 (4.0)
Genitourinary diseases	2.1 (3.0)	1.2 (1.9)	3.3 (2.5)
Other	8.3 (12.3)	7.3 (11.4)	15.6 (11.7)
Total	68.1 (100.0)	64.3 (100.0)	132.4 (100.0)

Notes: (a) Calculated as incidence × severity weight (range, 0–1) × average duration in years (discounted at 3%) and referred to as years lost due to disability (YLD); (b) Calculated as deaths × global standard expectation of life at age of death in years (discounted at 3%) and referred to as years of life lost (YLL); (c) Calculated as YLD + YLL and referred to as disability-adjusted life years (DALYs).

Broad cause groups, by age

DALY rates increased steeply with age, apart from small but significant peaks in infancy and early adulthood. Injuries (particularly in males) and mental disorders accounted for the majority of DALYs in early adulthood, after which cancer, cardiovascular disease, and

neurological and sense organ disorders were more prominent. The contribution from cancer peaked at age 70 years then declined, leaving cardiovascular disease as the major cause of DALYs in the very old.

Leading specific conditions, by sex

Ischaemic heart disease was the leading specific cause of health loss in males, followed by type 2 diabetes, anxiety and depression, lung cancer and stroke. For females, anxiety and depression was the leading specific cause of health loss, followed by ischaemic heart disease, stroke, type 2 diabetes and dementia (Table 18).

Table 18: Ten leading specific causes of health loss[(a)], by sex, Australia, 2003

	Males		Females	
Rank	**Specific cause**	**Rate/1000 people (%)**	**Specific cause**	**Rate/1000 people (%)**
1	Ischaemic heart disease	1.5 (11.1)	Anxiety and depression	1.3 (10.0)
2	Type 2 diabetes	0.7 (5.2)	Ischaemic heart disease	1.1 (8.9)
3	Anxiety and depression	0.7 (4.8)	Stroke	0.7 (5.1)
4	Lung cancer	0.6 (4.0)	Type 2 diabetes	0.6 (4.9)
5	Stroke	0.5 (3.9)	Dementia	0.6 (4.8)
6	Chronic obstructive pulmonary disease	0.5 (3.6)	Breast cancer	0.6 (4.8)
7	Adult-onset hearing loss	0.4 (3.1)	Chronic obstructive pulmonary disease	0.4 (3.0)
8	Suicide and self-inflicted injuries	0.4 (2.8)	Lung cancer	0.3 (2.7)
9	Prostate cancer	0.4 (2.7)	Asthma	0.3 (2.7)
10	Colorectal cancer	0.4 (2.5)	Colorectal cancer	0.3 (2.3)

Notes: (a) Expressed as disability-adjusted life years lost per 1000 people.

Risks to health, by broad cause group

The 14 selected risk factors together explained almost a third of health loss (expressed as total DALYs). Ten risk factors explained 32.9% of cancer-related health loss, tobacco use being the most important. Twelve risk factors explained 69.3% of health loss from cardiovascular disease, with high blood pressure and high blood cholesterol levels being the largest contributors. Four risk factors explained 26.9% of health loss from mental disorders, with alcohol and illicit drug use contributing in roughly equal proportions. Seven risk factors explained 31.7% of injury-related health loss, alcohol consumption being the dominant risk. Two risk factors explained 60.1% of health loss from type 2 diabetes, high body mass being the largest contributor (Table 19).

Table 19: Health loss[(a)] attributable[(b)] to 14 selected risk factors, by selected broad cause groups, Australia, 2003

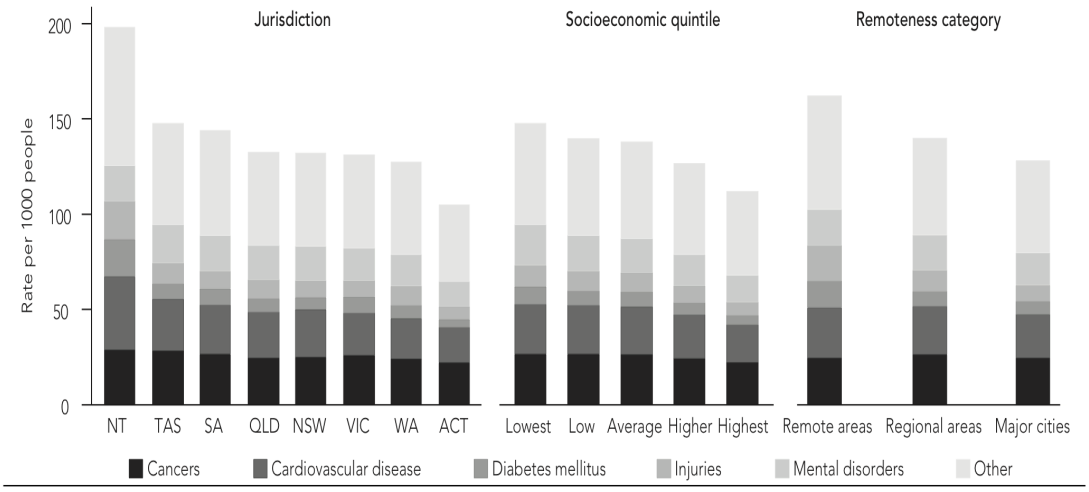
	Broad cause group					
	Cancers	**CVD**	**Mental disorders**	**Injuries**	**Diabetes mellitus**	**All causes**
Total health loss (DALYs lost/1000 people)	25.1	23.8	17.6	9.3	7.2	132.4
Attributable[(b)] health loss—individual (%)[(c)]						
Tobacco use	20.1	9.7	na	0.5	na	7.8
High blood pressure	na	42.1	na	na	na	7.6
High body mass	3.9	19.5	na	na	54.7	7.5
Physical inactivity	5.6	23.7	na	na	23.7	6.6
High blood cholesterol levels	na	34.5	na	na	na	6.2
Alcohol consumption	3.1	- 4.7	9.7	18.1	na	2.3
Low consumption of fruit and vegetables	2.0	9.6	na	na	na	2.1
Illicit drug use	na	< 0.1	8.0	3.6	na	2.0
Occupational exposures and hazards	3.1	0.4	na	4.7	na	2.0
Intimate partner violence	0.5	0.3	5.5	2.5	na	1.1
Child sexual abuse	< 0.1	< 0.1	5.8	1.4	na	0.9
Urban air pollution	0.8	2.7	na	na	na	0.7
Unsafe sex	1.0	na	na	na	na	0.6
Osteoporosis	na	na	na	2.4	na	0.2
Attributable(b) health loss—combined (%)[(d)]	32.9	69.3	26.9	31.7	60.1	32.2

Notes: CVD = cardiovascular disease; DALY = disability-adjusted life year; (a) Expressed as DALYs lost per 1000 people; (b) *Attributable* health loss is health loss that is explained by past exposure and that might be averted through future changes in exposure to a health risk; (c) Attributable health loss within each column is expressed as a percentage of total DALY rates for that column; (d) Figures for combined effects are not necessarily column totals because risk factors can share common causal pathways.

Health differentials

Age-standardised DALY rates were 31.7% higher in the lowest socioeconomic quintile than in the highest, and 26.5% higher in remote areas than in major cities. Age-standardised DALY rates in the Northern Territory were 88.7% higher than in the Australian Capital Territory, these jurisdictions having the highest and lowest rates, respectively (Figure 15).

Figure 15: Age-standardised[133] DALY rates per 1000 people, by jurisdiction, socioeconomic quintile and remoteness category, for selected broad cause groups, Australia, 2003



ACT = Australian Capital Territory. DALY = disability-adjusted life year. NSW = New South Wales. NT = Northern Territory. QLD = Queensland. SA = South Australia. TAS = Tasmania. VIC = Victoria. WA = Western Australia.

Past, present and future health loss

Age-standardised DALY rates declined from 151.0/1000 people to 132.4/1000 people over the period 1993–2003, and are projected to decline by 0.8% per year to 111.4/1000 people by the year 2023. Over the period 2003–2023, age-standardised DALY rates associated with cardiovascular disease are expected to experience the greatest annual rate of decline (−2.5%), followed by cancer (−1.4%), injuries (−1.1%) and chronic respiratory conditions (−0.9%). On the other hand, age-standardised DALY rates associated with diabetes are projected to grow by 1.8% a year over the period 2003–2023. Age-standardised DALY rates for other broad cause groups are likely to experience much smaller changes over this period (Table 20).

[133] Standardised to the age structure of the Australian population (males and females combined).

Table 20: Past and projected future changes in health loss[(a)], by selected broad cause groups and sex, Australia, 1993–2023

	Proportion of total (%)				Standardised rate/1000 people[(b)]			
Broad cause group	**1993**	**2003**	**2013**	**2023**	**1993**	**2003**	**2013**	**2023**
Cancers	18.8	19.0	18.9	18.2	29.5	25.1	21.8	18.2
Cardiovascular disease	22.3	18.0	15.4	13.1	36.5	23.8	17.1	12.0
Mental disorders	13.2	13.3	12.9	11.9	17.8	17.6	17.8	17.7
Neurological and sense disorders	9.6	11.9	13.9	16.4	15.1	15.7	16.1	16.3
Chronic respiratory diseases	7.1	7.1	6.8	6.9	10.6	9.4	8.4	7.7
Injuries	7.7	7.0	6.3	5.4	10.6	9.3	8.4	7.3
Diabetes mellitus	4.1	5.5	7.0	8.7	6.3	7.2	8.4	9.8
Musculoskeletal diseases	3.4	4.0	4.5	4.9	5.2	5.3	5.4	5.5
Other	13.8	14.2	14.1	14.4	19.5	18.9	17.7	16.8
Total	100.0	100.0	100.0	100.0	151.0	132.4	121.1	111.4

Notes: (a) Expressed as disability-adjusted life years; (b) Standardised to the age structure of the Australian population (males and females combined).

Discussion

Our findings emphasise that despite steady improvements in Australia's health over the past decade, significant opportunities for further progress remain at the beginning of the twenty-first century.

The strength of our analysis is that it is based on an internally consistent assessment of the incidence, prevalence, duration and mortality for a mutually exclusive and comprehensive set of diseases and injuries of importance in Australia. Health loss from these causes was quantified for different periods, subpopulations and risks to health using methods that incorporate fatal and non-fatal health outcomes and include adjustments to account for individuals who simultaneously experience multiple conditions. Health loss is likely to be over-estimated without such corrections, as the severity weights used to derive DALYs were originally determined for health states in isolation, without reference to coexisting conditions.[134]

A potential limitation is that the severity weights used in our analysis were derived from international sources[135] and applied without evidence of their validity in Australia. However,

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134 Mathers, CD, Iburg, KM and Begg, S, "Adjusting for dependent comorbidity in the calculation of healthy life expectancy".

135 Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I*; Stouthard, ME, Essink-Bot,

studies conducted elsewhere suggest that there are only minor variations across populations in the values people ascribe to different health states.[136]

We have not quantified uncertainty in our analysis, although a qualitative assessment suggests it is unlikely to be excessive.[137] Overall, about half of the total estimated health loss is due to mortality, for which estimates are fairly robust. Of the remainder, half is due to non-fatal outcomes from conditions for which reasonably good data are available (including cardiovascular disease, cancers, diabetes, common mental disorders and injuries), leaving a quarter with varying and probably higher levels of uncertainty. Precision varies between causes, with estimates for hearing loss, neurological conditions, osteoarthritis and cirrhosis being the most inaccurate.

Our results are not directly comparable with previous DALY estimates for Australia[138], owing to the different methods used. First, a number of the epidemiological models in our analysis benefit from more accurate inputs, particularly the cardiovascular disease models, which incorporated linked data from Western Australia. Second, unlike in the previous analysis, the comorbidity adjustments here capture the dependent nature of certain health states (e.g., diabetes increases the risk of heart disease). Third, the current risk attribution methods incorporate a number of methodological advances absent from previous health risk analyses.[139] Because of this lack of comparability, we back-calculated estimates for 1993 based on methods that were consistent with estimates for 2003.[140]

Several implications for policy are worth emphasising. For example, all of the health risks examined here are amenable to modification through intervention, and together explain a large proportion of health loss in Australia. In addition, the large health differentials between

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M, Bonsel, GJ, Barendregt, JJ, Kramers, PGN, van de Water, HPA, Gunning-Schepers, LJ and van der Maas, PJ, *Disability weights for diseases in The Netherlands*.

136 Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I.*

137 Professor Theo Vos now considers this assessment optimistic on the basis that our selection of epidemiological data was largely arbitrary and we did not consider uncertainty around key parameters such as mortality risk, remission or causal attribution in mortality data.

138 Mathers, CD, Vos, ET, Stevenson, CE and Begg, SJ, "The Australian Burden of Disease Study: measuring the loss of health from diseases, injuries and risk factors".

139 English, DR, Holman, CDJ, Milne, E, Winter, MG, Hulse, GK, Codde, JP, Bower, CI, Corti, B, de Klerk, N, Knuiman, MW, Kurinczuk, JJ, Lewin, GF and Ryan, GA, *The quantification of drug caused morbidity and mortality in Australia*, Commonwealth Department of Human Services and Health, Canberra, 1995; Mathers, CD, Vos, ET, Stevenson, CE and Begg, SJ, "The Australian Burden of Disease Study: measuring the loss of health from diseases, injuries and risk factors"; Ridolfo, B and Stevenson, C, *The quantification of drug-caused mortality and morbidity in Australia, 1998*, (7), Australian Institute of Health and Welfare, Canberra, 2001.

140 See Chapter 2 for details.

subpopulations are due, in part, to differential exposure to these risks. Significant health gains are likely to be achieved through realistic changes to future levels of exposure to health risks, given that even small changes in distribution of exposures can lead to substantial reductions in population-level risk.[141]

The predicted strong growth in DALY rates associated with diabetes is notable in that it is mostly due to increasing body mass. Given that current strategies have failed to mitigate this risk, new approaches are critical. The impact of increasing diabetes incidence will be magnified by reductions in case fatality from cardiovascular disease through successful strategies to reduce smoking and lower cholesterol levels and blood pressure.[142] Increased survival will result in a greater number of people with diabetes developing other health conditions such as renal failure, retinopathy, neuropathy and peripheral vascular disease. Notwithstanding the apparent intractability of diabetes, further reductions in cardiovascular disease could be achieved given that most of the health loss from this condition continues to be explained by exposure to known health risks.

The much higher DALY rates in the NT compared with other jurisdictions is largely explained by a higher concentration of Indigenous people in the NT. Health loss in this particular population is considered elsewhere.[143]

Several areas for further research flow from this work. First, health loss and expenditure under a business as usual approach to health risk management have been projected into the future[144] and could usefully be extended to include various 'what if?' risk-reduction scenarios.[145] Second, simulation methods have been used elsewhere to quantify uncertainty in DALY estimates[146], and would enhance interpretability if applied to these findings. Third,

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141 Rodgers, A, Ezzati, M, Vander Hoorn, S, Lopez, AD, Lin, RB and Murray, CJ, "Distribution of major health risks: findings from the Global Burden of Disease study", *PLoS Med*, 2004, vol. 1(1), pp. e27.

142 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Taylor, R, Dobson, A and Mirzaei, M, "Contribution of changes in risk factors to the decline of coronary heart disease mortality in Australia over three decades", *Eur J Cardiovasc Prev Rehabil*, 2006, vol. 13(5), pp. 760-8.

143 See Chapter 7 for further details.

144 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Begg, S, Vos, T, Goss, J and Mann, N, "An alternative approach to projecting health expenditure in Australia".

145 See Chapter 8 for an example.

146 Mathers, C, Salomon, J, Ezzati, M, Begg, S, Vander Hoorn, S and Lopez, A, "*Sensitivity and uncertainty analyses for burden of disease and risk factor estimates*".

developments in health state valuation methods could, if applied in Australia, increase confidence in the use of the DALY as a valid comparative measure of health loss.

Finally, our analysis is undermined, to some degree, by significant gaps in Australia's health information infrastructure. In particular, there is limited information on mental disorders, neurological conditions, hearing loss, chronic respiratory diseases and musculoskeletal disorders. Even more importantly, Australia, unlike other countries, has no mechanism for regularly collecting measurement data on biomedical indicators such as body mass, blood pressure, and blood glucose and cholesterol levels. Better and more frequent monitoring in each of these areas would strengthen future comparative assessments of health in Australia, thus enhancing their value for policy and program development.

Chapter 7: Burden of disease and injury in Aboriginal and Torres Strait Islander Peoples—the Indigenous health gap[147]

Introduction

The colonisation of Australia involved a litany of unjust and misguided policies against Aboriginal and Torres Strait Islander peoples[148] that manifested in the 'dispossession, physical ill-treatment, social disruption, population decline, economic exploitation, codified discrimination, and cultural devastation' of the first inhabitants of this land.[149] The fact that Indigenous Australians continue to experience substantially worse health, social and economic outcomes compared with other Australians is undisputed.[150]

Despite the plethora of reports documenting the health disadvantage of Indigenous Australians, to date it has been difficult to set priorities for Indigenous health development. In part, this is because many of the traditional population health indicators (life expectancy at birth, disease specific mortality and hospital rates, health survey findings, small epidemiological study results and notifiable disease registry data)[151] are based on fragmentary data, and do not capture the complete spectrum of diseases, injuries and risk factors. Further, routine data collection systems systematically underestimate true disease occurrence due to inadequate identification of Indigenous status. Thus, for health policy decision-making, these disparate measures inadequately indicate where the opportunities for health improvement lie.

More than a decade ago, a summary measure of population health, the Disability-Adjusted Life Year, or DALY, was developed and has been widely applied since to measure disease

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147 This chapter was first published as Vos, T, Barker, B, Begg, S, Stanley, L and Lopez, AD, "Burden of disease and injury in Aboriginal and Torres Strait Islander Peoples: the Indigenous health gap". See Declarations and Statements for the respective contributions of each author.

148 Steering Committee for the Review of Government Service Provision, *Overcoming Indigenous Disadvantage: Key Indicators 2005*, Productivity Commission, Canberra, 2005.

149 Gardiner-Garden, J, *From dispossession to reconciliation. Research Paper 27,* Commonwealth of Australia, Canberra, 1998-99, viewed 14 August 2011, http://www.aph.gov.au/library/pubs/rp/1998-99/99rp27.htm.

150 ABS and AIHW, *The Health and Welfare of Australia's Aboriginal and Torres Strait Islander Peoples 2005*, Australian Bureau of Statistics and Australian Institute of Health and Welfare, Canberra, 2005.

151 ABS and AIHW, *The Health and Welfare of Australia's Aboriginal and Torres Strait Islander Peoples 2003*, (ABS cat. no. 4704.0, AIHW cat. no. IHW11), Australian Bureau of Statistics and Australian Institute of Health and Welfare, Canberra, 2003; ABS and AIHW, *The Health and Welfare of Australia's Aboriginal and Torres Strait Islander Peoples 2005*.

burden in numerous populations across the globe, including Australia.[152] Apart from a study in the Northern Territory,[153] the burden of disease framework has not been applied to measure disease burden in Indigenous populations, although the need for such evidence to guide policies and programs is clear.

This chapter reports on the first burden of disease and injury study for the Aboriginal and Torres Strait Islander population of Australia. The study assessed the comparative importance of over 170 diseases and injuries for the health of Indigenous Australians in 2003, including the contribution of 11 risk factors.[154] Similar estimates for the total Australian population have been reported elsewhere.[155] This paper focuses on those diseases and risk factors that are most responsible for the gap in health status between Indigenous Australians and the overall Australian population.

Methods

For this analysis, we have used the same methods as were applied in the Burden of Disease and Injury in Australia 2003 study.[156] The main features of this method are the use of the DALY as a common integrating metric for fatal and non-fatal health loss; the use of existing demographic and epidemiological data sources; critical examination and correction of bias in these data sources; the intention to measure loss of health comprehensively even if scarce or poor quality data are available; and full transparency of all assumptions and data. Applying these common methods to the Indigenous population of Australia posed methodological challenges due to the under-identification of Indigenous people in routine health statistics

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152 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Begg, SJ, Vos, T, Barker, B, Stanley, L and Lopez, AD, "Burden of disease and injury in Australia in the new millennium: measuring health loss from diseases, injuries and risk factors"; Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I*; Victorian Department of Human Services, *The Victorian Burden of Disease Study: Mortality and Morbidity in 2001*, Victorian Department of Human Services, Melbourne, 2005.

153 Zhao, Y, Guthridge, S, Magnus, A and Vos, T, "Burden of disease and injury in Aboriginal and non-Aboriginal populations in the Northern Territory", *Med J Aust*, 2004, vol. 180(10), pp. 498-502. This work is misleading referred to as a *pilot* study in the original text.

154 Vos, T, Barker, B, Stanley, L and Lopez, A, *The Burden of Disease and Injury in Aboriginal and Torres Strait Islander Peoples 2003*.

155 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Begg, SJ, Vos, T, Barker, B, Stanley, L and Lopez, AD, "Burden of disease and injury in Australia in the new millennium: measuring health loss from diseases, injuries and risk factors"; Vos, T, Barker, B, Stanley, L and Lopez, A, *The Burden of Disease and Injury in Aboriginal and Torres Strait Islander Peoples 2003*.

156 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Begg, SJ, Vos, T, Barker, B, Stanley, L and Lopez, AD, "Burden of disease and injury in Australia in the new millennium: measuring health loss from diseases, injuries and risk factors"; Vos, T, Barker, B, Stanley, L and Lopez, A, *The Burden of Disease and Injury in Aboriginal and Torres Strait Islander Peoples 2003*.

collections. Under-identification affects numerator data (i.e. counts of health events) and denominator data (i.e. population estimates) but not to the same extent as the former relies on health staff, funeral directors or relatives to identify while the latter is determined by the likelihood of being enumerated and, if so, self-identification. A further complication is that Indigenous population counts have increased from census to census by a greater proportion than expected from births, deaths and migration. We chose to anchor our results to the Indigenous population as identified in the 2001 census.

Population data were accessed publicly and via customized data requests from the Australian Bureau of Statistics (ABS). Australian mortality data were provided by the Australian Institute of Health and Welfare. Although almost all deaths in Australia are considered to be registered, not all deaths have an identified Indigenous status and there are doubts about the validity of Indigenous identification if stated. As a result the number of deaths registered as Indigenous is an underestimate. We applied the generalized growth balance (GGB) method, an indirect demographic technique to correct for under-registration of deaths. The basic premise of indirect demographic methods is to compare population counts from two successive censuses and relate these to the number and age distribution of deaths recorded in the intercensal period. In a population unaffected by migration the number of people enumerated in a particular 5-year age group should equal the number of people enumerated in the previous age group minus the deaths that occurred during the 5-year intercensal period. The difference between recorded and expected deaths in the intercensal period is a measure of under-registration of deaths.[157] We applied the GGB method separately to population and mortality data for remote and non-remote areas using the ARIA+ classification, a geographic approach to remoteness based on road distance to service centres and the population size of those centres as a surrogate for availability of services.[158] We corrected for migration from remote to non-remote areas with data supplied by ABS on change in reported usual area of residence between the 1996 and 2001 censuses. Migration of Indigenous Australians in and out of the country is considered negligible.

Indirect demographic methods cannot estimate correction factors for childhood mortality rates in an intercensal period as they were not yet born at time of the first census. We

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157 Hill, K, Barker, B and Vos, T, "Excess Indigenous mortality: are Indigenous Australians more severely disadvantaged than other Indigenous populations?".

158 AIHW, *Rural, Regional and Remote Health: A Guide to Remoteness Classifications*, (AIHW cat. no. PHE 53), Australian Institute of Health and Welfare, Canberra, 2004.

observed that infant mortality estimates in Western Australia (WA) using linked birth and death data[159] were similar to recorded Indigenous mortality rates. We therefore assumed the WA infant mortality rates by remoteness applied across the whole country. We adjusted the observed mortality in 1- to 4-year-old Indigenous children in WA for the increased likelihood of mortality by remoteness.[160]

This gave us a set of corrected age- and sex-specific mortality rates reflecting the average mortality experience over the period 1996–2001 for those identified in the 2001 census as Indigenous Australians. We then assumed that these mortality rates had remained unchanged to 2003, the baseline year of study. We applied the cause of death structure by age and sex from recorded Indigenous deaths between 2001 and 2003 onto these corrected mortality estimates to derive counts of death by age, sex and cause in 2003.

We undertook a comprehensive search to identify data sources for calculating Years Lived with Disability (YLD) for Indigenous Australians including routine data collections (such as Australian perinatal collection data, notification data, mortality data and hospital data; accounting for 53% of YLD estimates); self-report and measured population health surveys (28% of YLD) and epidemiological studies that identified Indigenous Australians (3% of YLD). For a number of conditions, we assumed the same disease occurrence as the national study because of the lack of data to suggest otherwise (10% of YLD). In keeping with the national study, we applied a ratio of Years of Life Lost to YLD to estimate disability for rest categories such as 'other cancers' (6% of YLD). We used the same disease case definitions (stages and sequelae) and disability weights as the national study.[161]

Eleven common health risk factors were analysed for their contribution to disease burden for Indigenous Australians in 2003. Analyses were undertaken using standard methods.[162] Briefly, for each risk factor we define a theoretical minimum population distribution of risk

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159 Freemantle, CJ, Read, AW, de Klerk, NH, McAullay, D, Anderson, IP and Stanley, FJ, "Patterns, trends, and increasing disparities in mortality for Aboriginal and non-Aboriginal infants born in Western Australia, 1980-2001: population database study", *Lancet*, 2006, vol. 367(9524), pp. 1758-66.

160 Freemantle, C, *Indicators of Infant and Childhood Mortality for Indigenous and Non-Indigenous Infants and Children Born in Western Australia from 1980 to 1997 Inclusive*, University of Western Australia, Perth, 2003.

161 Chapter 2 presents a more complete discussion of these methods.

162 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*; Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 1*; Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 2*.

and estimate population attributable fractions from the prevalence of exposure in the population of interest and relative risks of disease outcomes causally related to the exposure from systematic reviews of the international literature. Data on the prevalence of exposure to these risk factors were largely derived from national survey data[163] and a few epidemiological studies with measurement data.[164]

Results

If Indigenous Australians in 2003 had experienced the same rate of disease burden as the total Australian population, a total of 56,455 DALYs would have been avoided, equivalent to 59% of the total burden of disease (95,976 DALYs) estimated for Indigenous Australians (Table 21). Non-communicable diseases explained 70% of this health gap, with cardiovascular disease the leading cause group followed by diabetes, mental disorders and chronic respiratory disease (Figure 16).

Ischaemic heart disease (14%), Type 2 diabetes (12%) and substance use disorders (6%) were the main non-communicable diseases that contributed to the health gap. Infectious diseases and neonatal conditions explained almost the entire gap from Group I causes. Suicide (4%), road traffic accidents (3%) and homicide and violence (3%) were the major injuries contributing to the gap (Table 21).

The largest contribution to the Indigenous health gap occurred in the 35- to 54-year-old age group (35%) followed by the 15- to 34-year-old age group (25%), the 55-year and older age group (23%) and then children under 15 years (17%). Cardiovascular disease, particularly ischaemic heart disease and diabetes, were the main contributors to the health gap at ages 35 years and over. Injuries and mental disorders were the main contributors to the health gap in young adults aged 15–34 years; even at these young ages, cardiovascular disease and diabetes were responsible for one-fifth of the health gap. Suicide explained almost half of the health

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163 ABS, *National Aboriginal and Torres Strait Islander Health Survey 2004-05*, (ABS cat. no. 4715.0), Australian Bureau of Statistics, Canberra, 2006; AIHW, *Child Protection Australia 2002-03*, Australian Institute of Health and Welfare, Canberra, 2004; AIHW, *Female SAAP Clients and Children Escaping Domestic and Family Violence 2003-04*, Australian Institute of Health and Welfare, Canberra, 2005; Department of Human Services and Health, *National Drug Strategy Household Survey: Urban Aboriginal and Torres Strait Islander Supplement, 1994*, Department of Human Services and Health, Canberra, 1995.

164 Cunningham, J, O'Dea, K, Dunbar, T, Weeramanthri, T, Zimmet, P and Shaw, J, "Study protocol--diabetes and related conditions in urban indigenous people in the Darwin, Australia region: aims, methods and participation in the DRUID Study", *BMC Public Health*, 2006, vol. 6, pp. 8; Wang, Z and Hoy, WE, "Hypertension, dyslipidemia, body mass index, diabetes and smoking status in Aboriginal Australians in a remote community", *Ethn Dis*, 2003, vol. 13(3), pp. 324-30.

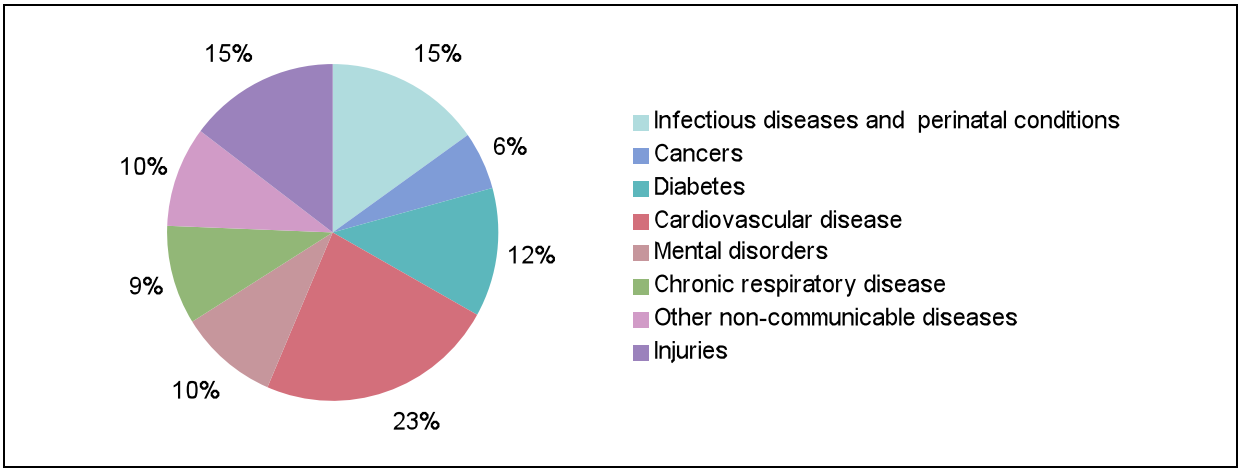
gap from injuries in young males. In young females, injuries contributed a lesser proportion to the gap but there was still considerable excess health loss from road traffic accidents, suicide and violence. Substance use disorders explained most of the gap from mental disorders in young adults (Table 21).

Table 21: Indigenous health gap (DALYs) due to selected causes, expressed as a proportion of total Indigenous health gap by sex and as a proportion of total Indigenous burden, 2003

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

Notes: (a) Communicable diseases, maternal and neonatal conditions.

Figure 16: Indigenous health gap (DALYs), proportional contribution by broad cause groups, 2003



The Indigenous health gap was equally distributed within males and females (Table 22). Two-thirds was due to premature mortality compared with 54% of the total Indigenous burden of disease, reflecting higher case fatality rates: i.e. when sick, Indigenous Australians are more likely to die.

Table 22: Indigenous health gap (DALYs) due to selected causes by sex and remoteness, expressed as a proportion of total Indigenous health gap, 2003

Cause	Males (%)	Females (%)	Non-remote (%)	Remote (%)
All causes	100	100	100	100
Group I[(a)]	14	16	12	20
Infectious and parasitic diseases	9	10	7	14
Neonatal causes	5	4	4	4
Non-communicable diseases	67	73	76	62
Cancers	4	7	6	5
Diabetes	10	14	12	12
Cardiovascular disease	24	23	24	23
Ischaemic heart disease	16	13	16	13
Stroke	3	3	3	3
Mental disorders	10	9	14	4
Substance use disorders	7	5	7	4
Other mental disorders	4	5	7	0
Chronic respiratory disease	9	10	10	8
Injuries	18	11	12	18
Road traffic accidents	4	3	2	5
Suicide	6	2	4	5
Homicide & violence	3	3	2	4

Notes: (a) Communicable diseases, maternal and neonatal conditions.

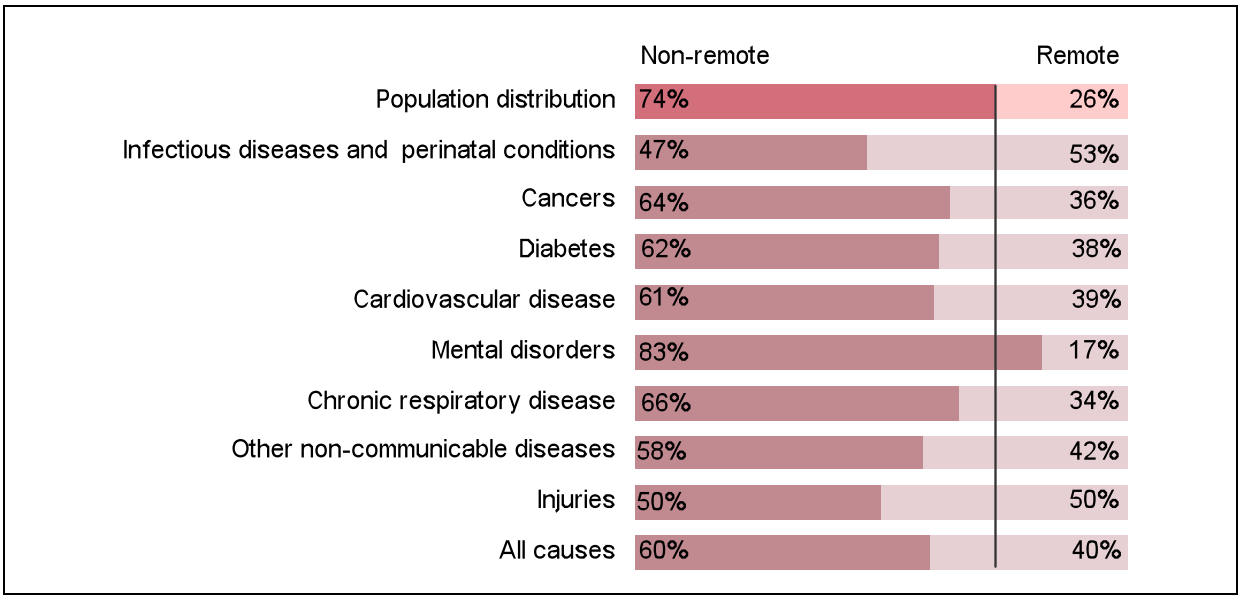
Tobacco (17%), high body mass (16%), physical inactivity (12%), high blood cholesterol (7%) and alcohol (4%) were the main risk factors contributing to the health gap (Table 23).

Table 23: Indigenous burden of disease and health gap (DALYs) due to 11 risk factors, 2003

	Disease burden		Health gap	
	DALYs	% of total	DALYs	% of total
Total Indigenous burden of disease	95,976	100	56,455	100
Attributable to:				
Tobacco	11,633	12	9,816	17
High body mass	10,919	11	8,953	16
Physical inactivity	8,032	8	6,554	12
High blood cholesterol	5,262	5	3,994	7
Alcohol	5,171	5	2,362	4
High blood pressure	4,417	5	3,215	6
Low fruit and vegetable intake	3,344	3	2,873	5
Illicit drugs	3,264	3	2,150	4
Intimate partner violence	2,469	3	1,836	3
Child sexual abuse	1,390	1	869	2
Unsafe sex	1,174	1	926	2
11 risk factors combined	35,908	37	27,383	49

The majority of the health gap occurred in the non-remote Indigenous Australian population (60%). However, the 26% of Indigenous Australians residing in remote areas experienced a disproportionate amount of the health gap (40%) compared with those living in non-remote areas. This was true across all major disease groups (Table 22 and Figure 17).

Figure 17: Indigenous health gap (DALYs) by broad cause groups expressed as proportions by remoteness, 2003



Discussion

This analysis is the first comprehensive assessment of the health of Indigenous Australians and indeed, of Indigenous populations anywhere. It confirms previous findings of substantial excess mortality and greater disease occurrence in Indigenous Australians. The particular value of this analysis is that it provides a quantification and comparative assessment of those diseases and risk factors that contribute most to the overall Indigenous burden of disease and, even more of interest, that contribute most to the Indigenous health gap. The latter is most indicative of the potential for health gain.

Precision of estimates

Because of inaccurate or incomplete identification of Indigenous status in health and population data, ascertainment of the true death rate requires adjustments by demographic methods, which inherently introduce an additional degree of uncertainty. We have shown elsewhere that our corrected estimates of Indigenous mortality rates are relatively insensitive to the assumption of a constant age pattern of mortality and the assumption of a constant age pattern of the unexplained population growth in the 1996–2001 intercensal period.[165]

In this analysis, we assumed that the level of mortality estimated for the 1996–2001 period applied to our baseline year of 2003. However, recent information has indicated an improvement in life expectancy in Aboriginal people in the Northern Territory.[166] If we apply the Northern Territory trend to adjust our mortality estimate downwards, the total amount of DALYs in Indigenous Australians would have been lower by 4%.

Estimates of disability for the more than 170 disease and injury categories included in this study depended on a combination of methods. When available, we included data on directly observed Indigenous health events in routine health statistics databases, health surveys or epidemiological studies.[167] For many diseases, such information did not exist. Instead, ratios of the differences between Indigenous and total population rates for hospital admissions or mortality were sought as proxy measures of disease occurrence. The consequence is that there are varying degrees of confidence about the accuracy of these estimates. Only six of the top

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[165] Hill, K, Barker, B and Vos, T, "Excess Indigenous mortality: are Indigenous Australians more severely disadvantaged than other Indigenous populations?".

[166] Wilson, T, Condon, JR and Barnes, T, "Northern Territory indigenous life expectancy improvements, 1967-2004", *Aust N Z J Public Health*, 2007, vol. 31(2), pp. 184-8.

[167] See supplementary Webtable 1 at http://ije.oxfordjournals.org/content/38/2/470/suppl/DC2

20 disabling conditions—ischaemic heart disease, stroke, homicide and violence, low birth weight, birth trauma and asphyxia and caries—were deemed to have good-quality sources of incidence data.[168] We judged the accuracy of estimates for alcohol dependence, migraine and personality disorders as particularly poor. The accuracy of estimates for the remaining top 20 conditions was judged as fair. This indicates that there is a large gap in knowledge to accurately estimate the disease occurrence of many major diseases contributing to the disability component of the burden of disease; including mental disorders, Type 2 diabetes, asthma and COPD. We resorted to finding differentials between Indigenous Australians and the total Australian population in proxy data sources, or were limited to a few isolated epidemiological studies to generalize to the whole population.

A key gap in available data to estimate the burden attributable to major risk factors was the lack of representative health measurement data on risk factors, such as blood pressure, cholesterol and body mass.[169] We recommend that a representative Indigenous health measurement survey be undertaken to measure the population distribution of exposure to these risk factors.

Policy implications

A detailed description of the burden of disease and injury in a population is alone not sufficient for setting priorities in public health. It is, however, an important foundation on which to base debates and economic evaluations that underpin health policies. This study contributes most obviously by identifying the excess amount of disease burden—the Indigenous health gap—and by quantifying the contribution of major modifiable risks to this gap, indicating where the greatest potential for health gain lies. The fact that much lower rates of disease burden are experienced by the majority of Australians means that with existing knowledge and technologies, a reduction of most of the Indigenous health gap is theoretically achievable. However, reducing this health gap will require comprehensive actions given that the health disadvantage of Indigenous Australians is apparent for almost all diseases and risk factors, at all ages, in men and women, and in remote and less remote areas.

Collectively, cardiovascular disease, diabetes and other tobacco-related conditions, such as lung cancer and chronic respiratory disease accounted for half the Indigenous health gap.

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[168] See supplementary Webtable 2 at http://ije.oxfordjournals.org/content/38/2/470/suppl/DC2
[169] See supplementary Webtable 3 at http://ije.oxfordjournals.org/content/38/2/470/suppl/DC2

These diseases share additional lifestyle risk factors, including high body mass, physical inactivity, raised blood pressure and cholesterol. These health problems largely affect middle-aged and older Indigenous Australians; however, they start at young ages, and there already is a sizeable burden in the 15- to 34-year-old age group. Prevention efforts should therefore target Indigenous adolescents and young adults.

However, efforts to reduce the health gap should not focus on prevention alone. For example, two-thirds of the health gap was due to mortality, which in part reflects the much higher case fatality for most diseases in Indigenous Australians than in their non-Indigenous counterparts. This is most likely due to a combination of factors such as late presentation for treatment, shortcomings in acute surgical and medical management, and inadequate follow-up during the course of disease, all of which have important implications for the way in which health services are delivered to Indigenous Australians.

It is also important to note that while the rate of disease burden may be higher in remote areas, the bulk of the health gap for Indigenous people arises in non-remote areas since the vast majority of Indigenous Australians reside in non-remote areas. The implication is that the focus of health service action to reduce inequalities in Indigenous health needs to include culturally appropriate and uniquely targeted approaches for non-remote and remote areas. Our findings will guide policy makers as to the emphasis to be given to health problems by broad remoteness area.

Addressing the multitude of health problems facing Indigenous Australians is complex and will require a wide range of initiatives to increase preventive and curative efforts and particularly to strengthen Indigenous health services. However, responses from within the health sector alone are not sufficient. There is an urgent need to address the social and economic disadvantages that contribute to the poor health status of Indigenous Australians. This is in keeping with the broader Indigenous concept of health which acknowledges that improving Indigenous health is about improving the physical wellbeing of an individual within a context of improving the social, emotional and cultural well-being of the whole community.[170]

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[170] NACCHO, *Submission to the Commonwealth Parliamentary inquiry into the needs of urban dwelling Aboriginal and Torres Strait Islander peoples,* National Aboriginal Community Controlled Health Organisation, Canberra, 2001 viewed 14 August 2011, http://www.naccho.org.au/Files/Documents/Urbaninquirysubmission.pdf

While this may be so, these requirements should not lead to inaction by the health sector arguing that the social and economic problems should be tackled first. Much can be done by appropriately resourced health services to reduce a sizeable proportion of the Indigenous health gap.

The results of this analysis are of immediate policy relevance to Australia yet also demonstrated how disparities in health experienced by Indigenous people in other countries, or indeed any other disadvantaged population group, can be comprehensively documented. Detailed information on the diseases and risk factors that contribute most to a disadvantage in health is a vital element of the evidence base for prioritizing interventions that can reduce such gaps.

Afterword

The above analysis was structured in response to the Council of Australian Governments (COAG) policy framework for Indigenous health matters, which features as a headline indicator the gap between Indigenous and non-Indigenous life expectancy. Its relevance in this respect, however, is debatable.

A key issue is whether a gap analysis based on the DALY—an incidence-based measure, which incorporates a normative assumption about time preference—is numerically related to the causal drivers of a gap in life expectancy. In their original discussion on time preferences Murray and Lopez note that a discount rate of 3%—the rate applied to the DALY calculations above—has an important effect on not only the proportion of total DALYs in a population that is due to non-fatal causes, but also the age-distribution of DALYs and the distribution of DALYs by broad cause groups.[171] Life expectancy, on the other hand, is unaffected by these considerations, although such technical distinctions are not mentioned in the published version of the above analysis.

The potential for this omission to mislead is demonstrated by the National Health and Hospitals Reform Commission's (NHHRC) use of the paper as evidence for the claim that

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[171] Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I.*

70% of the Indigenous life expectancy gap is due to chronic illness.[172] Presumably, the Commission derived this figure from Table 21.

Arguably a more relevant metric for the Commission's purposes because of its technical relatedness to life expectancy would have been HALE. However, this metric has traditionally not been regarded as a useful alternative to the DALY when information on causal drivers is required, mainly because of the perception that it cannot be decomposed by cause. Thanks to Nusselder and Looman such a perception no longer has any basis, as demonstrated not only by the results presented in Chapter 4 but also by those presented in a recent Queensland Government publication titled *Making Tracks towards closing the gap in health outcomes for Indigenous Queenslanders by 2033 – policy and accountability framework.*[173]

Making Tracks represents Queensland's response to the COAG target relating to closing the life expectancy gap and draws on a decomposition analysis of the difference HALE between Indigenous and non-Indigenous Queenslanders, the methods for which are described in Chapter 8. The focus here, however, is on how the results compare with the gap analysis based on the DALY, as presented in the preceding pages.

Figure 18 is a reproduction of a chart presented in *Making Tracks* and shows, in order of importance, the major drivers of the health expectancy gap between Indigenous and non-Indigenous Queenslanders, expressed as numbers of years. Cardiovascular conditions account for more than 4 years of this gap, followed by diabetes (3 years), injuries (more than two years), chronic respiratory conditions (1.5 years), cancers (more than 1 year) and infectious and parasitic conditions (about a year). The analysis has an intuitive interpretation by showing for any particular cause by how many years the health expectancy gap would be reduced if rates of mortality and disability in Indigenous Queenslanders were reduced to levels experienced by the rest of the population.

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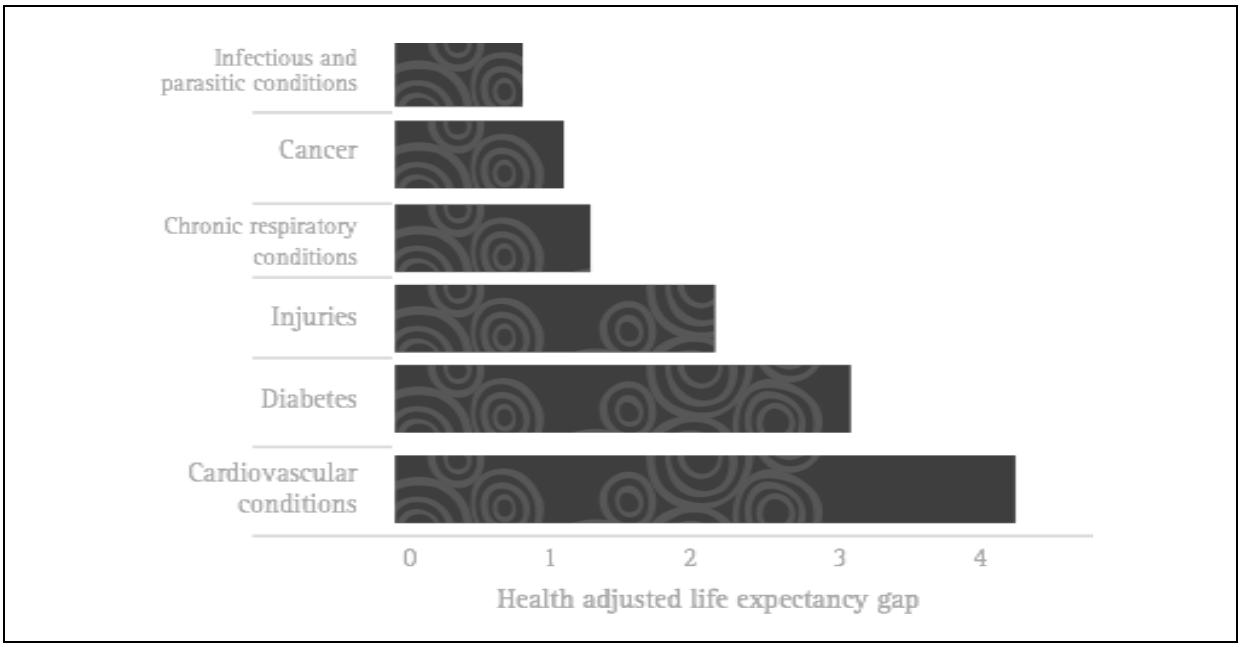
[172] NHHRC, *A healthier future for all Australians : Interim report : December 2008*, National Health and Hospitals Reform Commission, Canberra, 2008.

[173] Queensland Health: Serghi, M, Begg, S and others, *Making tracks : toward closing the gap in health outcomes for Indigenous Queenslanders by 2033 : policy and accountability framework*, Queensland Health, Brisbane, Qld., 2010.

Figure 18: What causes the Indigenous health gap in remote areas of Queensland?[174]



To illustrate the magnitude of the Commission's error in equating DALYs with health expectancies it is useful to compare the results presented in Table 21 with a HALE decomposition analysis based on the same underlying data set (Table 24). This comparison indicates that a gap analysis based on DALYs underestimates the contribution of chronic illness (called non-communicable diseases in Table 24) to the health expectancy gap by more than 10 percentage points (70% and 81%, respectively). This is because the DALY as typically calculated attributes the non-fatal impact of a cause back to the age at which it becomes incident whereas for HALE it is more commonly attributed on the basis of prevalence (i.e. the age at which it is experienced).[175] Life expectancy is less useful in this context since, while it can be decomposed by cause, it is calculated exclusively from mortality rates and thus ignores the impact of predominantly non-fatal but nevertheless chronic conditions such as diabetes and many mental disorders.

[174] ibid.

[175] At one state there were indications that the main results from the latest Global Burden of Disease Study may be presented using a new version of the DALY calculated on the basis of prevalence.

Table 24: Comparison between alternative measures of the Indigenous health gap

Cause	DALY gap[(a)] (%)	HALE gap[(b)] (%)	Difference (%)
All causes	100	100	0
Group I	15	10	-34
Infectious and parasitic diseases	9	4	-59
Neonatal causes	4	3	-29
Non-communicable diseases	70	81	16
Cancers	6	8	29
Diabetes	12	15	25
Cardiovascular disease	23	31	35
Ischaemic heart disease	14	19	35
Stroke	3	6	115
Mental disorders	10	7	-29
Substance use disorders	6	4	-27
Other mental disorders	4	3	-32
Chronic respiratory disease	9	12	30
Injuries	15	7	-53
Road traffic accidents	3	2	-31
Suicide	4	3	-37
Homicide & violence	3	2	-36

Notes: (a) Reproduced from Table 21; (b) Calculated using `hale_decomp` from the same data set used to estimate the DALY gap.

Calculated in these ways, the DALY gives greater weight than HALE to causes experienced earlier in life, such as infectious and parasitic diseases (9% compared to 4%), mental disorders (10% compared to 7%) and injuries (15% compared to 7%), notwithstanding the attenuating effect of discounting. The opposite is true for causes experienced later in life, such as cancers (6% compared to 8%), diabetes (12% compared to 15%) cardiovascular diseases (23% compared to 31%) and chronic respiratory disease (9% compared to 12%).

This concludes Part III of this thesis. In Part IV, the final part, I consider the success or otherwise of the methods proposed in Chapter 2 with respect to facilitating the uptake of the Murray and Lopez framework by Australian governments.

Part IV: Implementation and conclusions

Chapter 8: Health outcomes in Queensland

In this chapter I explore the practical dimensions of implementing the model represented by the set of workflows presented in Chapter 2 from outside the context of the case study. I use as an example a work program at Queensland Health, which relied on the process `subpop-IPM` to derive a complete set of framework parameters for Queensland in 2003, stratified by cause, sex, age, socioeconomic quintile and remoteness category. This data set generated state-level totals consistent with the results for Queensland presented in Chapter 6 and provided the starting point for a number of analyses summarised here under three high-level objectives.

Original baseline

My initial objective was to integrate estimates from the Indigenous component of the case study, which, as described in chapter 2, had been derived using `subpop-IPM` mainly from the initial database of parameters but reported separately due to timing considerations. Given the much lower overall health status of Indigenous people compared to the rest of the Australian population, Indigeneity is a critical dimension when describing the health status of geographically defined populations that have a large Indigenous proportion, which is the case in some administrative areas of Queensland. These are currently referred to as Health Service Districts but will become Hospital and Health Services as a result of recent health reforms agreed to by the Council of Australian Governments.

To integrate the Indigenous and non-Indigenous dimensions of the case study, I derived a set of selected parameters (incidence, prevalence, deaths, PYLD, YLD and YLL) for Indigenous people by combining the Indigenous proportion of the population in each statistical local area (SLA)—the smallest geographical unit of analysis in health data for practical purposes — with population rates of the selected parameters for Indigenous Australians, stratified by cause, sex, age and remoteness category. Estimates for non-Indigenous people were derived by combining the non-Indigenous proportion of the population in each SLA with population rates of the selected parameters for Queensland, stratified by cause, sex, age, socioeconomic status and remoteness. I assigned a socioeconomic and remoteness classification to each SLA to allocate the correct population rate in each case. An unavoidable consequence of these methods was that for some causes, state-level totals were marginally different from the results presented in Chapter 6 relating to Queensland.

I then derived a set of outputs for each Health Service District by aggregating the selected parameters from the SLA level while preserving certain within-district stratifications (i.e. cause, sex, age, Indigeneity, socio-economic status and remoteness). I assumed these parameters were consistent with the SLA-level estimates so did not reprocess them using `subpop-IPM`. However, I did use the `hale_decomp` process described in Chapter 4 to decompose the drivers of differences in HALE for each Health Service District from the state average. In addition, a colleague undertook a state-level comparative risk assessment by combining risk models from the case study with estimates of risk prevalence for Queensland in 2003. As mentioned already, a discussion of these models is provided in the report that accompanies this thesis, although the details are not relevant to the present discussion.

The results from these analyses provided the basis for the various information products listed in Table 25 with the identifier *2003* as the baseline year. The exception to this general flow of information was the 2006 edition of Queensland Health's flagship series *The Health of Queenslanders*, which went to press prior to commencing the above work program and was therefore based on a set of estimates that excluded an explicit Indigenous dimension.

Dynamic baseline

My second objective was to update the set of framework parameters for Queensland with more contemporary data. The first step involved creating a new dataset with two supplementary parameters, stratified by year, cause, sex and age. The first of these recorded for Queensland the annual percentage change in incidence between 2003 and a new baseline year, which my colleagues derived from various proxy sources such as hospitalisation data, surveys, registry data and mortality data. The second recorded mortality data for Queensland between 2003 and the new baseline year. We combined both with state-level input parameters for 2003 and trend assumptions for the period before 2003 to derive a complete time-series of input parameters for the period 1979 to the new baseline, stratified by cause age, sex and year.

Table 25: Inventory of publications that incorporate information from the Queensland work program

Publication	Baseline year
Chief Health Officer Reports[c]	
The Health of Queenslanders 2006: First report of the Chief Health Officer Queensland	2003[a]
The Health of Queenslanders 2008: Second report of the Chief Health Officer Queensland	2006
The Health of Queenslanders 2010: Third report of the Chief Health Officer Queensland	2007
The Health of Queenslanders 2012: Fourth report of the Chief Health Officer Queensland[b]	2007
Health Service Districts Reports[d]	
Burden of disease and health adjusted life expectancy in Health Service Districts of Queensland Health, 2003	2003
Burden of disease and health adjusted life expectancy in Health Service Districts of Queensland Health, 2006	2006
Queensland Burden of Disease and Injury Circulars[d]	
First Series	
Circular 1 - Overview of the burden of disease and injury in Queensland, 2003	2003
Circular 2 - Leading causes of burden of disease and injury in Queensland, 2003	2003
Circular 3 - Age group differences in burden of disease and injury in Queensland, 2003	2003
Circular 4 - Risk factor impact on the burden of disease and injury in Queensland, 2003	2003
Circular 5 - Differentials in the burden of disease and injury in Queensland, 2003	2003
Circular 6 - Burden of disease and injury, life expectancy and health adjusted life expectancy in Queensland Health Service Districts, 2003	2003
Second Series	
Circular 1 - Overview of the burden of disease and injury in Queensland, 2006	2006
Circular 2 - Leading causes of burden of disease and injury in Queensland, 2006	2006
Circular 3 - Age group differences in burden of disease and injury in Queensland, 2006	2006
Circular 4 - Differentials in the burden of disease and injury in Queensland, 2006	2006
Circular 5 - Burden of disease and injury, life expectancy and health adjusted life expectancy in Queensland Health Service Districts, 2006	2006
Circular 6 - Risk factor impact on the burden of disease and injury in Queensland, 2007	2007
Circular 7 - Projected burden of disease and injury in Queensland, 2007 to 2016	2007

Notes: (a) These estimates do not include a specific Indigenous dimension; (b) In preparation at the time of writing; (c) These publications can be accessed online at http://www.health.qld.gov.au/cho_report/default.asp; (d) These publications can be accessed online at http://www.health.qld.gov.au/epidemiology/publications/burden-of-disease.asp

This new data set was then processed with `cohort-IPM` in a number of ways. In the first instance, I derived a complete set of framework parameters for 2006, this being the latest year for which mortality data was available at the time. In the second, a colleague derived a set for the period 2007 to 2016.[176] For the projection component of the latter analysis, we processed state-level mortality using the regression methods described in chapter 2, except we used national multinomial models in place of those based on Queensland data because the

[176] We had hoped that 2008 might be the new baseline in this instance but the required mortality data from the ABS was unavailable (at the time of writing, this data was still unavailable due to a change in dissemination policies being implemented by the ABS).

observed trends for some causes were unstable in the latter. In the third, I combined elements of the set of parameters for 2007 with new inputs for key diseases to derive a complete set of parameters for Indigenous Queenslanders, stratified by three remoteness categories in the year 2007.

In each case, we processed parameters for the new baselines using Equation 7 of Chapter 2 to account for the comorbidity considerations discussed in that chapter. In addition, we used the processes described above but modified to incorporate a new set of boundaries to disaggregate the parameters for 2006 to Health Service Districts. My colleagues also updated the state-level comparative risk assessments for both Indigenous and non-Indigenous Queenslanders to the year 2007.

On the basis of these analyses we released a second series of information circulars, as listed in Table 25 with the baseline identifier *2006* or *2007*, respectively, and, at the time of writing, we were in the process of preparing a second report in response to the *Making Tracks* document discussed in the afterword to Chapter 7. In addition, the South Australian and Western Australian health departments approached me to update the original set of parameters for these jurisdictions to 2006, and then disaggregate the new sets of parameters to relevant subpopulation groupings. Results from those consultancies are listed elsewhere.[177]

A common feature in each of these analyses is the incorporation of new data on mortality, incidence and case-fatality. As such, they provide evidence for the successful implementation of the model illustrated in Figure 8 of Chapter 2. However, several points regarding this general observation are worth noting. First, not all jurisdictions had the resources to adopt the proposed model, hence there was no way to aggregate the individual analyses into a nationally cohesive picture such as the one presented in Parts II and III of this thesis. Second, no jurisdiction was in a position to create a more up-to-date national picture since access to a number of key national datasets was restricted. Finally, there was only limited capacity to review and update the parameter assumptions underlying the model, hence the need for additional resources to undertake such a body of work over the medium to longer term.

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[177] See http://www.sahealth.sa.gov.au/wps/wcm/connect/public+content/sa+health+internet/about+us/our+performance/health+statistics/other+statistics/burden+of+disease+in+sa and http://www.health.wa.gov.au/publications/BOD_Technical.cfm

In the remainder of this chapter I illustrate the potential scope of these work programs by describing an analysis that sought to quantify the projected health and expenditure outcomes of obesity reduction targets of the former Queensland Government.

Scenario modelling

Introduction

Obesity is most commonly assessed using the body mass index (BMI), which is calculated as weight in kilograms over the square of height in metres. Typically, a healthy body weight is defined as a BMI of 18.5 to less than 25, overweight as a BMI of 25 to less than 30 and obese as a BMI of 30 or above. As indicated in Chapter 6, high body mass accounted for more than one half of the total health loss from diabetes in Australia in 2003. Moreover, this risk is increasing, with a growth of almost 12% in recorded prevalence of overweight and obese Australian adults between 2001 and 2007–08. These attributes alone would appear to justify the setting of rigorous targets by government as a policy framework for mitigating further growth in levels of obesity.

In this analysis my Queensland Health colleagues and I attempt to quantify the potential health and financial benefits of one target in particular as it relates to diabetes in the Queensland population.

Methods

Our methods are identical to those established for diabetes in Chapter 2, but are described here in greater detail. In this approach the established relationship between BMI and the most common form of diabetes —Type 2 diabetes mellitus (or T2DM)—is modelled via the *population impact fraction* (PIF) [178], which can be used to assess the proportional change in incidence of disease after a change in exposure to a related risk. The incidence after this change is,

$$I' = I(1 - PIF),$$

Equation 19

[178] Morgenstern, H and Bursic, ES, "A method for using epidemiologic data to estimate the potential impact of an intervention on the health status of a target population", *J Community Health*, 1982, vol. 7(4), pp. 292-309.

where I is the original incidence. We treat BMI as a log-normally distributed continuous risk because this is the distribution that most closely resembles the population-level data. The PIF for a continuous risk is given by,

$$PIF = \frac{\int_{x=1}^{h} RR(x)P(x) - \int_{x=1}^{h} RR(x)P'(x)}{\int_{x=1}^{h} RR(x)P(x)},$$

Equation 20

where $RR(x)$ is the relative risk at exposure level x, $P(x)$ the population distribution of exposure, $P'(x)$ the counterfactual distribution of exposure, and l and h are the integration boundaries.[179] Unlike dichotomous or categorical risk attribution models, Equation 20 assumes the risk conferred on T2DM increases exponentially with every unit increase in BMI, and quantifies this risk at all points in the population distribution minus a counterfactual. The counterfactual in this case is a theoretical minimum risk distribution for BMI, which—following Ezzati and colleagues—we assume has a mean of 21 and a standard deviation of 1.

Together with some additional data and assumptions, these equations allow us to model via the `cohort-IPM` function both the likely prevalence of T2DM if current trends in obesity were to continue, and what this prevalence might be under alternative obesity trajectories. Prevalence of T2DM is a key parameter of interest in this analysis since, while obesity is a risk for becoming an incident case of T2DM, healthcare costs accrue from treating and managing prevalent cases of this disease. This general framework operationalises a set of ideas described by Murray and others in terms of the generic model illustrated in Figure 19. However, it differs from the model used by Goss to assess the impact of health reform proposals for the NHHRC[180], which incorrectly assumed changes in obesity operate directly on prevalence rather than via incidence.[181]

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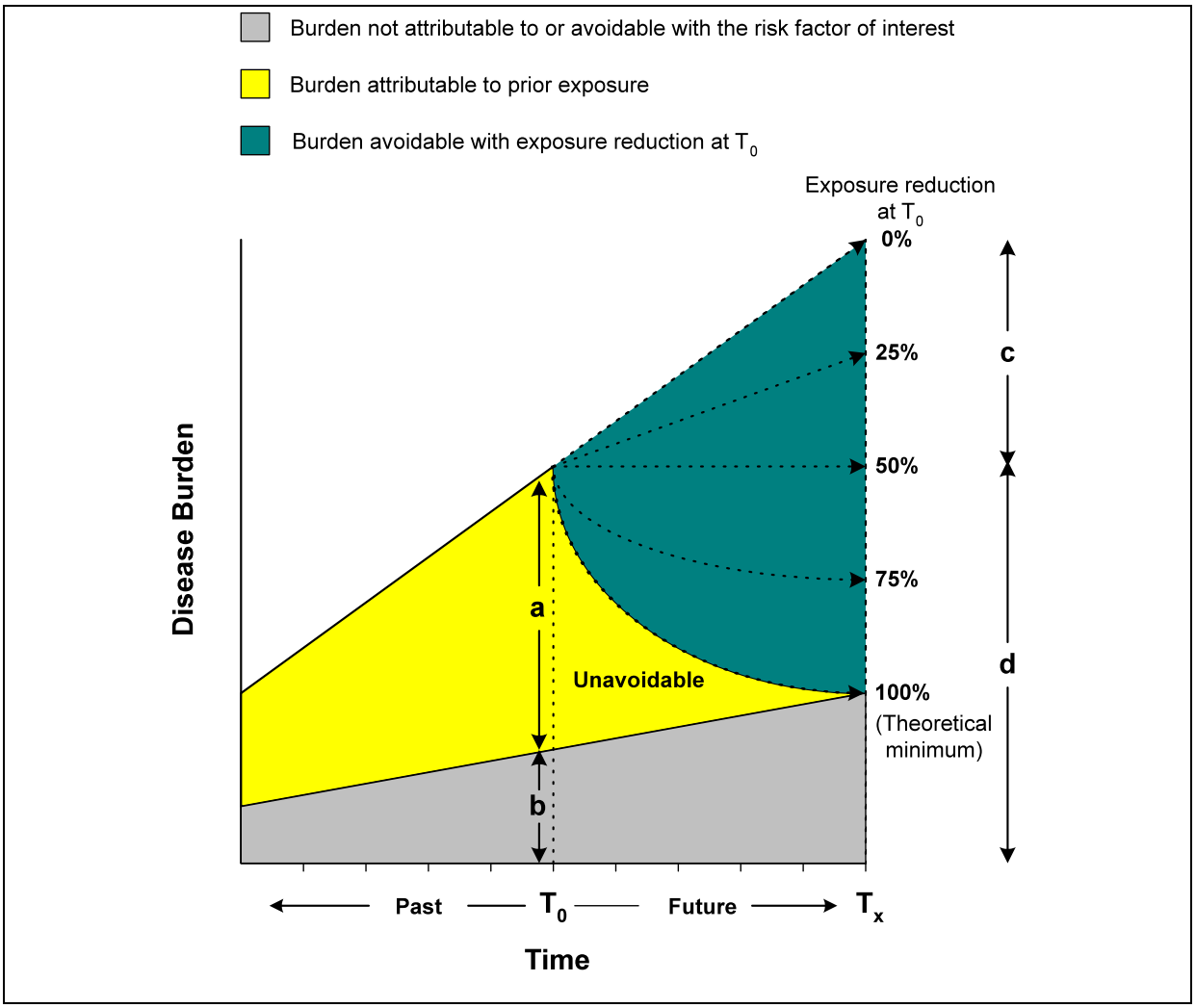
179 Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 1*.

180 See Afterword to Chapter 5.

181 AIHW, *Estimating the impact of selected National Health and Hospitals Reform Commission (NHHRC) reforms on health care expenditure, 2003 to 2033*.

Figure 19: A generic model for health risk assessment[182]



We derived the input parameters for the analysis from various sources, but primarily from the set of parameters for Queensland in 2003 described at the beginning of this chapter. This in turn was derived from the underlying epidemiological models referred to above but hitherto not described in any detail except in the accompanying case study report. As set out in Appendix 1 of that report, we derived the incidence of type 1 diabetes mellitus (or T1DM) in 2000 from the National Diabetes Register,[183] from which we estimated prevalence in Dismod II, assuming no remission and age-specific relative risk of dying for all diabetes from the Asia Pacific Cohort Studies Collaboration.[184] For under 25-year-olds, we derived the

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182 Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 1*.

183 AIHW, *National diabetes register: statistical profile, December 2001*, Australian Institute of Health and Welfare, Canberra, 2003.

184 Woodward, M, Zhang, X, Barzi, F, Pan, W, Ueshima, H, Rodgers, A and MacMahon, S, "The effects of diabetes on the risks of major cardiovascular diseases and death in the Asia-Pacific region", *Diabetes Care*, 2003, vol. 26(2), pp. 360-6.

incidence of T2DM also from the National Diabetes Register. For older ages, we subtracted the prevalence of T1DM from the total prevalence of measured diabetes in 2000 from the Ausdiab study—still the most reliable estimates available at the time of writing—before deriving incidence in Dismod II assuming an annual growth in incidence of 2.5% for males and 1.5% for females over the period 1980–1999.[185]

In the absence of a direct measure of trends in incidence or prevalence of T2DM, we assumed the historically flat trend in mortality from this disease reflects the net effect of an increase in incidence and a decrease in case-fatality. The latter was assumed to be equivalent to half the trend in cardiovascular disease case-fatality since the main causes of death in people with diabetes have a vascular origin. On this basis we incorporated an annual decline in case-fatality of 2% for males and 1% for females over the same period for which we modelled increasing trends in incidence. We extrapolated incidence and case-fatality by continuing the above trends and then used Dismod II, again with the same trend assumptions, to derive a complete epidemiological model for T2DM in Australia for the year 2003.

As briefly outlined in Chapter 2, we disaggregated the incidence parameter in this model to states and territories using jurisdictional proportions of hospitalisation data. On the basis that case-fatality and remission are likely to be relatively uniform across jurisdictions, this provided a set of incidence, case-fatality and remission parameters for Queensland up until the year 2003, which we projected to 2007 to coincide with the most recent reliable source of BMI in Queenslanders, measured height and weight from the National Health Survey 2007-2008. We modelled trends in incidence between 2003 and 2007 to follow trends in the PIF for BMI, which we calculated from various inputs: a regression model of observed mean BMI on age, birth cohort and sex[186]; the relationship between changes in the mean of BMI and its standard deviation based on measured distributions from Ausdiab, National Heart Foundation and Busselton studies; and the Asia Pacific Cohort relative risk of mortality estimates mentioned above.

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[185] Dunstan, DW, Zimmet, PZ, Welborn, TA, De Courten, MP, Cameron, AJ, Sicree, RA, Dwyer, T, Colagiuri, S, Jolley, D, Knuiman, M, Atkins, R and Shaw, JE, "The rising prevalence of diabetes and impaired glucose tolerance: the Australian Diabetes, Obesity and Lifestyle Study", *Diabetes Care*, 2002, vol. 25(5), pp. 829-34.

[186] Haby, MM, Vos, T, Carter, R, Moodie, M, Markwick, A, Magnus, A, Tay-Teo, KS and Swinburn, B, "A new approach to assessing the health benefit from obesity interventions in children and adolescents: the assessing cost-effectiveness in obesity project", *Int J Obes (Lond)*, 2006, vol. 30(10), pp. 1463-75.

As stated already, we assumed half the trends in case-fatality for cardiovascular disease would also apply to diabetes. Following Unal, we modelled trends in cardiovascular disease case-fatality as being 48% of the overall trends in observed cardiovascular disease mortality, the remainder being accounted for by trends in incidence.[187] Projections of cardiovascular disease mortality to 2033 were achieved by applying the combination of Poisson and multinomial regression techniques set out in Chapter 2.

For 2007 and beyond we modelled four incidence trajectories for T2DM via the PIF. The first was a continuation of past trends in changes to the distribution of BMI, which we call the *business as usual* scenario. To achieve this we used the regression models discussed above, which we recalibrated to fit observed means and standard deviations for Queensland from the National Health Survey 2007-2008. The remaining scenarios were attenuations of the above as follows:

1. *One-third reduction* scenario—based on the former Queensland Government's target to cut the proportion of overweight or obese Queenslanders by one-third[188], which we interpreted as a shift in the distribution of BMI such that the proportion of overweight or obese people in 2020 is approximately two-thirds the proportion observed in 2007;
2. *Immediate elimination* scenario—based on the assumption that the proportion of overweight or obese people in 2007 is reduced to zero in 2008 and thereafter; and
3. *No change* scenario—based on the assumption that the age-specific proportions of overweight or obese people in 2007 remain unchanged for the foreseeable future.

Figure 20 illustrates the implications for the distribution of BMI in adults with respect to the first three of these scenarios (by definition, the fourth has no such implications). It also presents historic trends in measured BMI from various sources. Indicated in light grey is the proportion of overweight or obese people for 2007 with the historic distributions, and for 2020 with the three scenarios.

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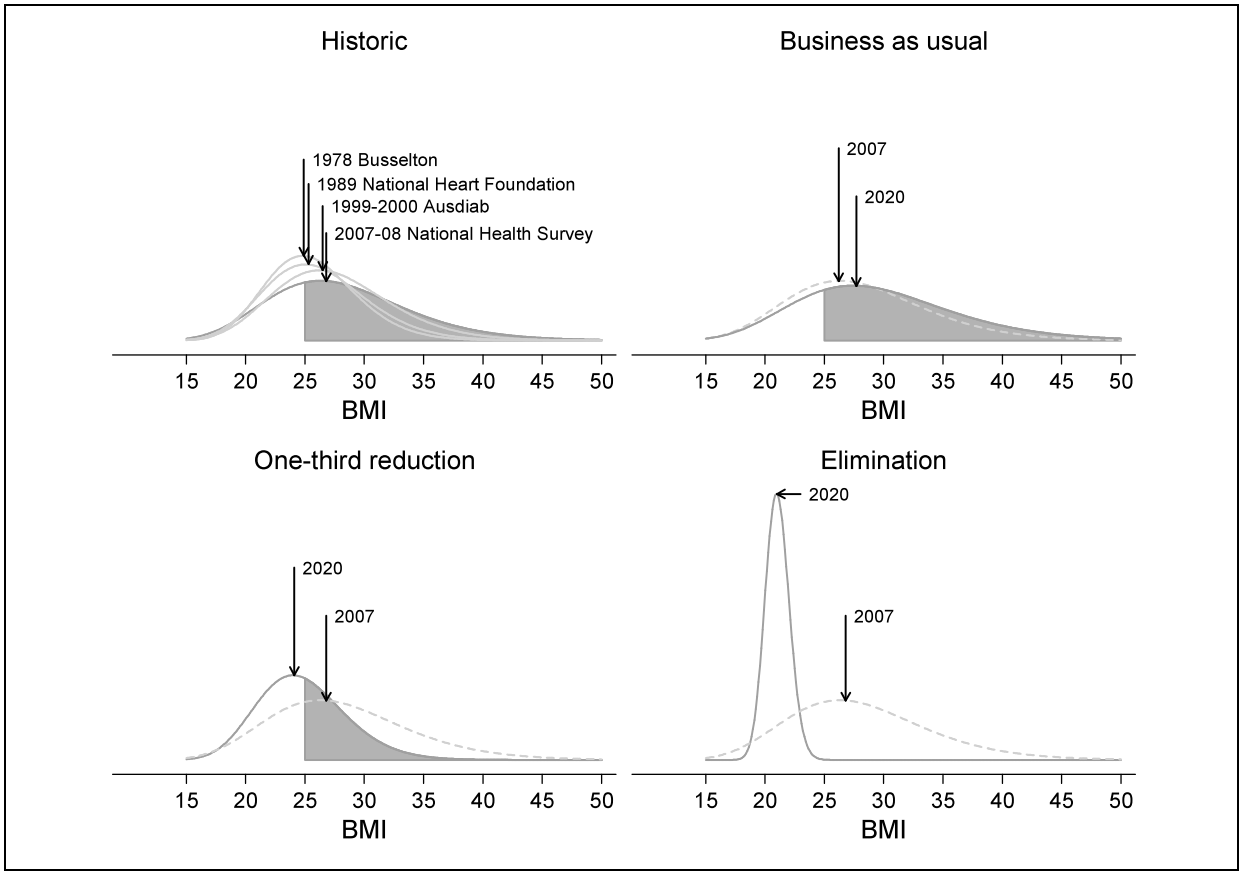
[187] Unal, B, Critchley, JA and Capewell, S, "Explaining the decline in coronary heart disease mortality in England and Wales between 1981 and 2000".

[188] The State of Queensland (Department of the Premier and Cabinet), *Toward Q2: Tomorrow's Queensland,* Queensland Government, Brisbane, 2008, viewed 19 August 2011, http://www.towardq2.qld.gov.au/tomorrow/library/pdf/Towards_Q2_Tomorrows_Queensland.pdf.

Figure 20: Distribution of BMI in adults, historic trends from 1978 to 2007, under three scenarios, 2007 and 2020



To quantify the potential change in treatment-related expenditure under each scenario we applied an annual cost per prevalent case assumption, which we estimated on the basis of the calculations by Goss as presented in the afterword to Chapter 5. This was equivalent to $1,211 in 2003 (in 2006–07 constant dollars), and growing in real terms by 2.2% per year thereafter.[189] As discussed already, both sources use the same methodology but differ with respect to certain parameter assumptions. This led to a slower annual cost growth of 1.5% in the earlier calculations. However, in both cases the cost parameter should be regarded as conservative because it only relates to direct expenditure on type 2 diabetes and its complications, thus it ignores expenditure on conditions for which diabetes confers an elevated risk, such as cardiovascular disease.

[189] AIHW, *Estimating the impact of selected National Health and Hospitals Reform Commission (NHHRC) reforms on health care expenditure, 2003 to 2033*; Goss, J, *Projection of Australian health care expenditure by disease, 2003 to 2033*.

Results

Table 26 summarises the key results of this analysis and shows that under the business as usual scenario, we expect 63.2% of the Queensland population to be overweight or obese by 2020—an annual average growth of 0.9% from 2007 levels—and 7.5% to have T2DM. At these levels we expect direct expenditure on T2DM to be around $714 million (in 2006-07 dollars), representing an average growth in real terms of 6.8% per year.

Should the former Queensland Government's target be reached instead, the proportion of overweight or obese Queenslanders in 2020 will be 37.8% (by definition), which represents an annual decline in risk of 3% over the projection period. Such a trajectory would most likely result in a stabilisation of T2DM at around 6.4% of the population by 2020 but a 18.4% increase from 2007, reflecting the lag between risk reduction and accumulated disease in the population. At this level of T2DM, direct annual expenditure would be around $617 million in 2020 (in 2006-07 dollars), which is equivalent to a potential saving of around $100 million compared to the business as usual scenario in that year, or $436 million in cumulative savings since 2007. However, growth would be only 1.2 percentage points lower than the business as usual scenario at 5.6% per year.

Table 26: Overweight/obese, type 2 diabetes and direct expenditure on type 2 diabetes under various obesity scenarios, all ages, Queensland, 2007 and 2020

	2007		2020				
	Total	Un-related	Business as usual	No change	One-third reduction	Elimin-ation	Un-related
Overweight/obese							
Crude prevalence (%)	56.5	n.a.	63.2	57.5	37.8	0.0	n.a.
Change pa. (%)	n.a.	n.a.	0.9	0.1	-3.0	n.a.	n.a.
Type 2 diabetes							
Crude prevalence (%)	5.4	2.1	7.5	7.3	6.4	4.2	2.3
Change pa. (%)	n.a.	n.a.	2.4	2.3	1.3	-1.9	0.5
Direct expenditure							
Annual ($ millions)	304	97	714	700	617	407	219
Change pa. (%)	n.a.	n.a.	6.8	6.6	5.6	2.3	4.9
Since 2007 ($ millions)	n.a.	n.a.	6,838	6,783	6,402	4,985	2,270

In the event that age-specific levels of overweight or obese Queenslanders were to remain constant over the period 2007–2020 (the no change scenario), we would still expect a one percentage point increase in overweight and obesity across all ages because of the combined

effect of population ageing and the fact that older people tend to be fatter. At this level of risk we would expect real growth in direct expenditure on T2DM to be 6.6% per year. On the other hand, the immediate removal of obesity as a risk (if this were possible), would result in growth of 2.3% per year, until some point well beyond the projection period when the higher growth associated with non-obesity T2DM takes over as the dominant driver of expenditure for this disease. These trends are depicted graphically in Figure 21 and Figure 22.

Figure 21: Prevalence (%) of type 2 diabetes under various obesity scenarios, all ages, Queensland, 2000 to 2020

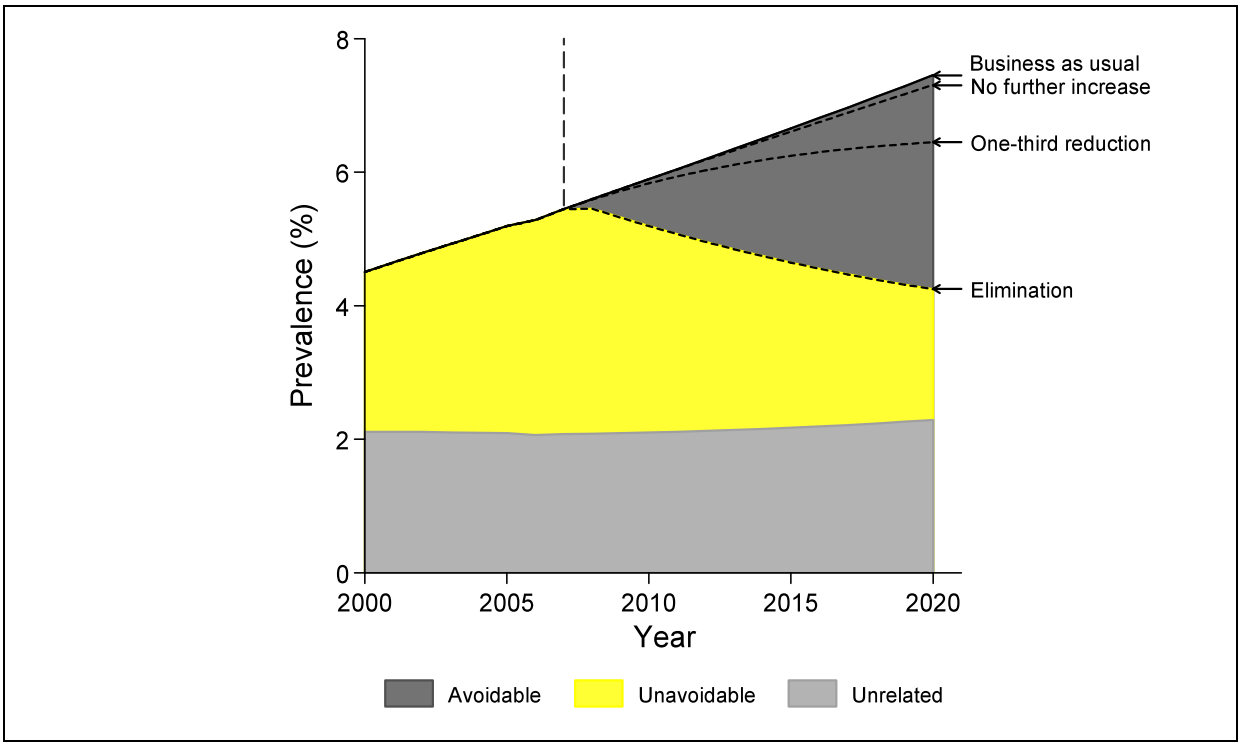
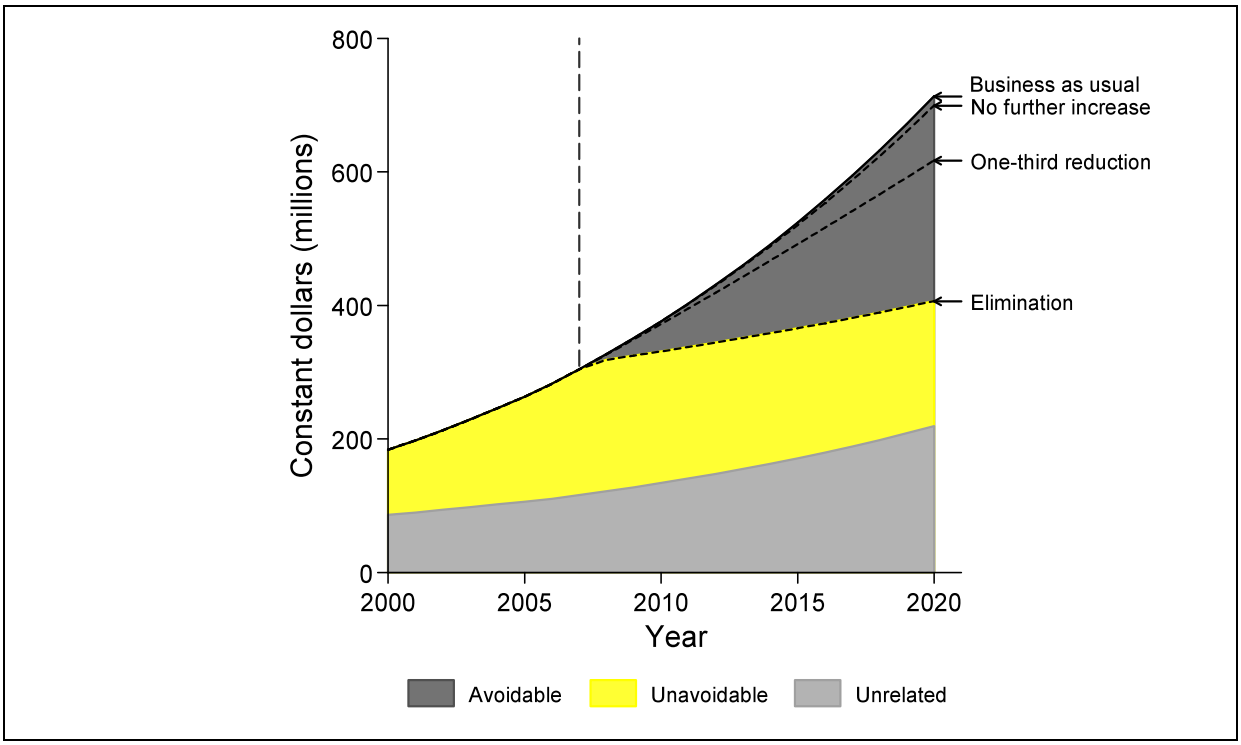


Figure 22: Direct health expenditure in 2006-07 constant dollars (millions) on type 2 diabetes under various obesity scenarios, all ages, Queensland, 2000 to 2020



Discussion

This analysis demonstrates that an attenuation of recent trajectories in the distribution of body mass is likely to reduce the prevalence of T2DM, at least from what it would have otherwise been had these trends continued. However, it also shows that even if we were successful in achieving a one-third reduction in overweight and obesity by 2020, the prevalence of T2DM would continue to rise, albeit at a decreasing rate of growth, and would only start to stabilise towards the end of this period. Of the four scenarios examined, prevalence would only reduce from 2007 levels under the elimination scenario, illustrating the long lead-time between risk mitigation and absolute reduction in the prevalence of this disease.

Whether these findings have economic implications is less clear. While the analysis shows there are likely to be direct expenditure savings under each scenario compared to business as usual, a more complete analysis would consider other health outcomes in addition to T2DM as well as the level of investment required to achieve the implied reductions in obesity. Changes in levels of health, productivity and utility might also be considered, depending on both the available evidence and the particular perspective of the analysis.

For example, a recent analysis by Vos and colleagues—which incorporated the epidemiological models developed as part of the case study—indicates that it may be cost-effective from the perspective of the *health system* to focus on a limited set of interventions such as a 10% tax on unhealthy food, laparoscopic gastric banding for those who are severely obese, and diet and exercise interventions targeting overweight and obese people in primary care settings.[190] However, this analysis does not account for the potential loss of individual utility resulting from, for example, increasing the cost of unhealthy food even for people of healthy weight.[191] More importantly, its scope is severely restricted by the paucity of credible evidence on effective obesity control, suggesting that more evaluation research is needed before such analyses become more widely accepted as useful inputs into policy development in this area.

Perhaps the most important finding from the analysis therefore is that direct expenditure on T2DM will continue to grow in real terms irrespective of our efforts to curb obesity. This conclusion is consistent with the findings presented in Chapter 5, which illustrate that factors other than underlying population risk are important drivers of growth in health expenditure. That this is likely to be the case even if there were unlimited resources for tackling obesity suggests that a balanced policy response to T2DM as an emergent risk to health system sustainability would focus on both the epidemiological *and* technological drivers of growth in expenditure on this disease.

Afterword

While undertaking this analysis I discovered an error in the original PIF calculation that underpinned the diabetes trends in the case study, which arose from my misinterpretation of advice from Barendregt regarding the use of a spreadsheet-based integration function he had developed.[192] This error had a marginal downward effect on all results relating to diabetes projections from that work, including those discussed in Parts II and III of this thesis and the case study report. It also has implications for a paper titled 'Projecting the burden of diabetes

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[190] Vos, T, Carter, R, Barendregt, J, Mihalopoulos, C, Veerman, J, Magnus, A, Cobiac, L, Bertram, M, Wallace, A and ACE–Prevention Team, *Assessing Cost-Effectiveness in Prevention (ACE–Prevention): Final Report.* , University of Queensland and Deakin University, Brisbane & Melbourne, 2010.

[191] I am indebted to Mr Paul McGuire for highlighting this particular limitation.

[192] Risk factor Integral, Version 2.0, Epigear, Brisbane, 2011, viewed 12 August 2011, http://www.epigear.com/index_files/risk_factor.html.

in Australia - what is the size of the matter?'[193] in which we discuss the importance of transparency in epidemiological modelling, particularly in relation to projecting the prevalence of diabetes given the policy significance of this disease. Table 27 reproduces the original Table 1 of that paper but with updated prevalence estimates based on a correct implementation of Equation 20.

Table 27: Summary of the different published projections of diabetes prevalence in Australia

	Year	Age-group (years)	Number of people with diabetes (millions)	Predicted prevalence of diabetes (%)
Sicree et al.[194]	2025	20–79	1.3	7.7
Magliano et al.[195]				
Static incidence and mortality rates[(a)]	2025	≥25	2.0	11.4
Dynamic incidence and mortality rates[(b)]	2025	≥25	3.0	17.0[(c)]
Begg et al.[196(d)]	2023	All ages	2.5	10.4
Corrected Begg et al.	2023	All ages	2.5	10.1

Notes: (a) Static diabetes incidence and mortality rates for 2005 applied until 2025; (b) Incidence of diabetes rises by 4% every year and mortality (for those with and without diabetes) falls by 2.2% each year; (c) The prevalence of diabetes across all ages would be approximately 12.%; (d) Increasing but incorrectly calculated incidence trends are applied until the year 2023. The miscalculation was based on my erroneous assumption that the change in the first integral in the numerator of Equation 20 would be equivalent to the entire equation because Barendregt's integral function included the ability to specify a reference point on the exposure scale.

193 Magliano, D, Peeters, A, Vos, T, Sicree, R, Shaw, J, Sindall, C, Haby, M, Begg, S and Zimmet, P, "Projecting the burden of diabetes in Australia - what is the size of the matter?", *Australian and New Zealand Journal of Public Health*, 2009, vol. 33(6), pp. 540-3.

194 Sicree, R, Shaw, J and Zimmet, P, "Diabetes and impaired glucose tolerance", in Gan, D, ed., *Diabetes Atlas*, 3rd ed, International Diabetes Federation, Brussels, 2006.

195 Magliano, DJ, Shaw, JE, Shortreed, SM, Nusselder, WJ, Liew, D, Barr, EL, Zimmet, PZ and Peeters, A, "Lifetime risk and projected population prevalence of diabetes", *Diabetologia*, 2008, vol. 51(12), pp. 2179-86.

196 Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*.

Chapter 9: Conclusions

I began this thesis with the proposition that the 'burden of disease' methodology introduced by Murray and Lopez in the early 1990s represents a useful platform from which to develop insights into several broad trends in Australia, unprecedented growth in health over the last century and even faster growth in the cost of accommodating this social phenomenon. This argument was positioned in the context of growing concerns about the sustainability of publicly funded healthcare, both here and overseas. Strictly speaking, it is government spending on health where these concerns are most acute since there is little evidence that total spending on health (i.e. both public and private) is close to exceeding the opportunity cost of using these resources elsewhere. Such a distinction is important when considering sustainability from the perspective of health system reform, for example, but for the present purposes can be put to one side.

I then asserted that the corollary of this proposition—the generally held perception that Murray and Lopez's contributions have little to offer such debates—is unfounded. The portrayal of their contributions in the literature was offered as one explanation for such a perception, although the point was not developed except to note that these discussions tend to be dominated by theoretical rather than policy considerations. Of course other explanations also exist, such as the language of 'burden of disease', a term commonly attached to the word 'study' to imply its application is a discrete exercise, and the tendency to conflate the framework with one particular metric, the disability adjusted life year (DALY). Uncritical acceptance of these and other characterisations are unlikely to have broadened the appeal of the underlying concepts, although for the present purposes this point too can be put to one side.

More central to my overall argument was the view that in order for propositions one and two to achieve more general acceptance a thorough overhaul of 'burden of disease' practice might be in order. Underlying this idea was the proposition that the main elements of the framework are best understood as a series of transformative processes that ultimately depends on only a handful of parameters, all but one of which is epidemiological; from these few parameters all other parameters of interest can be derived. Another way of expressing this is to regard the framework as a collection of principles about the practice of comprehensive and internally consistent descriptive epidemiology, and the transformation of information so derived into a

DALY or a comparative risk assessment as a second order process. I illustrated the consequences of not implementing the framework in this way by drawing attention to practical considerations that have constrained the usefulness of a key process to the range of applications typically associated with 'burden of disease' studies.

The most obvious and arguably most important application beyond this range is monitoring trends in population health on a routine and comprehensive basis. Given health accounts for over one-fifth of total public expenditure in Australia each year (or $84.8 billion in 2009-10), the argument for this being an accepted function of government on financial accountability grounds alone seems strong. Nevertheless, Australia's current framework for monitoring population health (the National Health Performance Framework[197]) reflects an overwhelming preference for partial indicators of success and, more revealingly, lacks an accepted process for synthesising the large volumes of information routinely collected each year into answers to simple questions such as how healthy are Australians, is it the same for everyone, or where is the most opportunity for improvement?

I then introduced a setting in which I could test these propositions, which presented itself in the form of a project undertaken by the University and Queensland in collaboration with the Australian Institute of Health and Welfare (AIHW). This project was commissioned by the Australian Government Department of Health and Ageing (DOHA) in early 2003 and had as one of its objectives facilitating the uptake of the framework on a routine basis by government agencies, such as AIHW and state and territory health departments. As such, I adopted simple program logic principles to assess an alternative implementation model with respect to both its range of outputs compared to what might be expected and its intended outcomes more broadly with respect to facilitating the use of the framework on a routine basis.

The key conclusion to draw from the material presented in the preceding chapters is that, with respect to outputs, the implementation model developed for the case study, which owed its

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[197] AIHW, *National Health Performance Framework 2009,* Canberra, 2009, viewed 12 August 2011, http://meteor.aihw.gov.au/content/index.phtml/itemId/392569. The Australian Health Ministers' Conference (AHMC) noted a revised version of this framework in September 2009 and COAG agreed to around 100 indicators in response, the majority of which (72) relate to the health system performance domain; the remainder are split between the determinants of health and the health status domains (18 and 10, respectively). The intent of the latter is guided by several high-level questions such as how healthy are Australians and is it the same for everyone, although the indicators agreed to by COAG in this group have a much more narrow focus and comprise of several very basic measures of mortality and the prevalence of specific health conditions.

origins to a model developed at WHO for the GBD 2000 project, allowed for a much greater range of analyses than was attempted in a previous implementation by AIHW. The specific areas where these benefits were observed included the ability disaggregate the primary results to potentially any geographic boundary of interest in Australia, the ability to fore- and back-cast these results while preserving internal consistency between parameters, the ability to generate a complete set of alternative results for various groupings of Australia's Indigenous population, and the ability to account for dependent and independent comorbidity in disability calculations.

In addition, the analysis demonstrated that the approach expanded the range of secondary analyses regarded as feasible to areas not previously associated with 'burden of disease', such as health expenditure projections and causal decomposition analyses of health-adjusted life expectancy. The former analysis was subsequently used by the National Health and Hospital Reform Commission in its consideration of reform options for Australia's health system, demonstrating that the framework has practical applications beyond those first articulated by Murray and Lopez.

When it comes to outcomes, however, the conclusions are more circumspect. Certainly, the material presented in the preceding chapter demonstrates that several state governments successfully implemented the model on at least one occasion each to update the original case study outputs relating to these jurisdictions to the year 2006. It also demonstrates that, at the time of writing, the model was still being used on a semi-routine basis by one state government to update outputs relating to this jurisdiction for the year 2007, and to generate new outputs for three groupings of Indigenous populations. Its use beyond these applications was only really limited by the absence of dedicated resources to undertake additional analyses and by the lack of a critical input, coded mortality data for years later than 2007.

Yet even this more restricted outcome suggests the model has potential as the basis of a country-level population health monitoring system, both Australia and elsewhere. The cost of such a system has not been worked out in any detail here but is likely to be marginal compared to the amount already spent on a routine basis on collecting many of the primary data inputs. Moreover, the return on this type of investment in terms of more informed policy discourse is likely to be substantial considering there is no other comparable system for synthesising the range of existing partial indicators of health across its various domains. Parts II and III of this thesis illustrate just a few of the contemporary policy debates that could

potentially benefit from such a system, particularly if a range of outputs were to be made available on a regular basis. It is worth exploring in these final pages, therefore, the extent to which there exists an appetite within government for the type of monitoring system embodied by the objective outlined in the opening chapter.

To answer this question I present in the discussion that follows a brief analysis of relevant events, based mainly on documentary evidence. While a much wider range of methods—such as structured key informant interviews, thematic analysis and discourse analysis—could also have been employed, a more rigorous qualitative approach was considered beyond the scope of these concluding remarks.

With respect to overarching policy direction, there appears to be strong alignment between the strategic priorities of the Australian Health Ministers' Advisory Council (AHMAC)—the peak forum for governments to discuss health matters in Australia—and comprehensive monitoring of the population health becoming a routine function of government. This is evident in an AHMAC document titled the *National Health Information Management Principal Committee Strategic Work Plan 2007–08 to 2012–13* (hereafter, the NHIMPC Plan), which, as its name implies, is the current strategic blueprint for NHIMPC, one of a number of principal committees of AHMAC.[198]

The NHIMPC Plan outlines four priorities areas for better health outcomes in Australia, one of which is to increase the visibility of emerging risks through targeted investment in health information.[199] In broad terms, the intent appears to have been to identify strategies for enhancing the scope and coverage of our health information infrastructure through (amongst other things) better health outcomes monitoring. To this end, Objective 4.5 is to improve the coverage, quality, utilisation and coordination of public health information. Two pre-existing AHMAC documents are referred to as evidence of ongoing progress in this respect, one of which is titled the *National Public Health Information Plan 2005*.

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[198] Formerly called the National Health Information Management Principal Committee, the National E-Health and Information Principal Committee (NEHIPC) is one of several principal committees that report to AHMAC, which in turn provides support to the Standing Council on Health (SCoH), the peak decision-making body on health matters in this country.

[199] National E-Health and Information Principal Committee, *National Health Information Management Principal Committee Strategic Work Plan 2007-08 to 2012-13,* Australian Health Ministers' Advisory Council, Canberra, 2007, viewed 12 August 2011, http://www.ahmac.gov.au/NHIMPC_Strategic_Work_Plan.pdf.

The National Public Health Information Plan 2005 is noteworthy in the context of the present discussion because it lists as a key strategy for using public health information in Australia to its fullest the case study.[200] However, this is not as clear a statement of support for the objective stated in the opening chapter as it first appears. The plan itself, for instance, is presented as a joint effort of the Australian Institute of Health and Welfare (AIHW) and the National Public Health Information Working Group (NPHIWG), and is referred to hereafter as the AIHW/NPHIWG Plan in recognition of the stakeholders responsible for its development. The previously mentioned NHIMPC Plan attributes responsibility for its implementation to the Public Health Information Development Group (PHIDG) not NPHIWG, presumably to accommodate a restructuring of AHMAC in the period between the two documents.

PHIDG is made up of essentially the same group of stakeholders that constituted NPHIWG (that is, representatives of the Commonwealth, state and territory governments, and of AIHW). However, it has no formal reporting relationship to NEHIPC (unlike NPHIWG had) nor does its website acknowledge responsibility for the AIHW/NPHIWG Plan.[201] Instead, PHIDG reports to the Australian Population Health Development Principal Committee (APHDPC)—a separate principal committee of AHMAC—and aligns itself with the principles and practices of the National Health Information Agreement (hereafter, the NHIA). The NHIA, in turn, refers (confusingly) to NPHIWG not PHIDG, but makes no reference to the AIHW/NPHIWG Plan.[202] More confusingly, of the five terms of reference listed on APHDPC's website, only one relates to information and this is in the context of strengthening health development infrastructure rather than health information infrastructure.[203]

Thus it seems that NPHIWG's vision with respect to the case study was overtaken by events following the introduction of new lines of reporting within AHMAC. This conclusion is consistent with there being no more recent plan than NPHIWG's for strengthening Australia's health information infrastructure, just as it is with PHIDG's confirmation that it has no

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200 See p. 22 of AIHW and NPHIWG, *National Public Health Information Plan 2005* Australian Institute of Health and Welfare, Canberra, 2005, viewed 12 August 2011, http://www.aihw.gov.au/publication-detail/?id=6442467796.

201 Population Health Information Development Group (PHIDG), AIHW, Canberra, 2011, viewed 11 August 2011, http://www.aihw.gov.au/phidg/.

202 COAG, *National Health Information Agreement 2004-10,* AIHW, Canberra, 2004, viewed 12 August 2011, http://meteor.aihw.gov.au/content/index.phtml/itemId/182135.

203 Australian Population Health Development Principal Committee, AHMAC, Canberra, 2011, viewed 12 August 2011, http://www.ahmac.gov.au/site/membership.aspx.

intention of building on the roadmap established by its predecessor, despite NPHIWG's expectation that the AIHW/NPHIWG Plan would be reviewed after three years.[204]

In addition to legitimate questions about the relevance of these two documents in the context of current AHMAC structures, it is also doubtful whether they should be called strategic since even the first post-dated negotiations leading to the case study by several years. Similarly, there is nothing in the project documentation to suggest that the provision of funding by DOHA to the University was in any way related to an AHMAC decision. As such, it is reasonable to characterise the objective discussed in the opening chapter as the product of a bilateral agreement between the respective agencies to undertake work on behalf of other agencies, rather than as the outcome of a clear policy intent to develop a national health monitoring capacity, as determined through established intergovernmental governance arrnagements.

Notwithstanding the obvious success of this less formal approach at attracting funding for the case study, it does not appear to have been sufficient to affect lasting change at the national level. While it is inappropriate to go into the details here, there are grounds for believing that the effect of not having a clearly articulate benefits realisation strategy was compounded, at least initially, by a public dispute between the University and the Australian Bureau of Statistics.[205] Certainly, soon after this dispute DOHA rejected a University proposal to develop a dissemination platform for more disaggregated results than were available in the case study report. Similarly, AIHW was subsequently unsuccessful in securing ongoing funding from DOHA's base appropriation for related purposes.

Less clear, however, is the role of either of these factors over the longer term. For example, in a paper to PHIDG dated 7 April 2008 South Australia expressed the need for more disaggregated and regular information from the case study than was available publicly at the time, and revealed a preference for nationalising this function on efficiency grounds. While the letter AHIW wrote to state and territory health department CEOs in response was ultimately successful in securing resources to address both the short-term information needs of this level of government, and those of AIHW in relation to its flagship publication

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[204] Queensland's representative to PHIDG (personal communication July 2011).

[205] See, for example, Hare, J, "Whistleblower stonewalled as uni ignores orders to respond", *The Australian*, 30 May 2012, p. 5; O'Keefe, B, "Hackers pick up UQ cash prize", *The Australian,* 21 March 2007, p. 33; O'Keefe, B, "Stats all, folks, for hackers", *The Australian,* 7 Feb 2007, p. 30.

Australia's Health,[206] it also created the opportunity for AIHW to observe to jurisdictional health CEOs, 'the question of ongoing funding for burden of disease work remains'.

A more strategic response to the problems articulated by South Australia presented itself in the form of a paper to PHIDG on 7 October 2009 by AHIW titled 'Proposal for a National Monitoring Centre for Burden of Disease'. Based broadly on the views expressed at a public forum of state, territory and AIHW officials on 2 October 2009, this paper set out the business case and funding options for establishing a comprehensive national health monitoring capacity such as the one embodied in the objective discussed in the opening chapter. The minutes of PHIDG's consideration of this agenda item indicate in-principle support for the idea and note its recommendation that AIHW redraft the proposal for consideration by PHIDG's parent committee, APHDPC.

The available information indicates that the final submission was for $2.25 million over four years funded on an established cost sharing arrangement involving a 50% contribution from the Commonwealth (around $280,000 per year) and a contribution to the remainder from each state and territory on the basis of population share (from around $2,900 per year for NT to $95,000 per year for NSW). The proposal notes:

> This bid is unlikely to be agreed from the AHMAC cost-shared budget as it requires an on-going funding source. [However,] it would be feasible for all jurisdictions to agree to contribute their share outside the cost-shared budget… PHIDG jurisdictional members have expressed strong support for coordinated, ongoing work in BoD measures.

It is surprising given this level of support for the establishment of a comprehensive national health monitoring capacity that not only was APHDPC unable to provide in-principle support for the idea, even subject to further discussions around funding, but between rejecting it in late 2009 and PHIDG's next meeting in mid 2010 APHDPC asserted its own view of the role of 'burden of disease' in Australia by providing PHIDG with the following advice:

> APHDPC would like PHIDG to continue and meet as and when required, noting the main body of work being Burden of Disease (BoD). The APHDPC advised this will remain the case until the

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[206] See Section 2.7 of AIHW, *Australia's health 2010 : the twelfth biennial health report of the Australian Institute of Health and Welfare*, Australian Institute of Health and Welfare, Canberra, 2010.

outcome of the National Preventive Health Agency (NPHA) is known, at which time it would be reviewed.[207]

These views reveal much about the balance of power within AHMAC and attribute to PHIDG a more active involvement in the implementation aspects of the framework in Australia than, with the exception of comparative risk assessment—a secondary process with clear links to prevention—the evidence allows. For example, some state and territory PHIDG representatives had experience with comparative risk assessment or were involved in calculating population attributable fractions at the time. However, none was considering a review of the parameters underlying the model described in Chapter 2 or, more importantly, had the resources to do so even if they wanted to.

At its next meeting PHIDG considered another paper by AIHW titled 'Burden of disease business case development', which reiterated the various funding options for the proposed national monitoring centre. However, the minutes of this meeting simply note that funding was no longer available from either the Commonwealth or AHMAC for such purposes. Furthermore, PHIDG's Queensland representative is able to confirm that no alterative options—such as contributions from interested parties outside of AHMAC arrangements—were discussed.

In a further example of its longer-term aspirations, AIHW's next move was to offer to PHIDG the services of one of its senior officers for a limited period to:

1. Form a network of State/Territory/Commonwealth BoD personnel
2. Develop a workplan for further development of the national core work, including classification of the way BoD can respond to key policy issues requiring measurement under the National Healthcare Agreement
3. Prepare a bid for National Health and Medical Research Council (NHMRC) funding for the future work. [208]

This three-part strategy approach resulted in PHIDG establishing a working group comprising representatives from each state and territory health department, as well as from

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[207] Minutes from 21-22 July 2010 meeting of PHIDG.
[208] Ibid.

DOHA and AIHW.[209] Referred to as the National Burden of Disease Collaborative Network (NBoDCN), this group—of which I was a member as one of Queensland's self-appointed representatives—was chaired by AIHW and met several times during the course of 2010 primarily to assist AIHW in developing a plan for implementing the Murray and Lopez framework at a national level.

The work plan that emerged from this process set out desirable outputs from the perspective of NBoDCN should further national work occur, and how these might inform contemporary policy debate. More importantly, it attempted to address a key issue for network members, how to obtain these outputs on a regular and sustainable basis. Given this outcome, the plan shared many characteristics with the implementation model outlined in Chapter 2. AIHW provided a final draft of the plan for consideration by the chair of PHIDG in early 2011.

At the time of writing these conclusions in May 2012, PHIDG's chair had not yet distributed to the rest of PHIDG the AIHW plan. Several reasons have been provided in response to my queries about this situation, illness soon after the work was completed being the most likely. It seems equally plausible, however, that, on executing its obligations to PHIDG, AHIW judged it unproductive to further pursue the matter through AHMAC forums on the basis of existing advice from DOHA that, while representing a useful statement of aspirations, the plan could nevertheless be construed as an attempt to re-prosecute APHDPC's decision not to support ongoing, nationally coordinated work.[210]

In any event, a retreat from trying to engage DOHA on these issues appears ultimately vindicated by advice from PHIDG in March 2012 that, as foreshadowed by APHDPC, a newly established Commonwealth body, the Australian National Preventive Health Agency (ANPHA), had begun discussing 'the future of burden of disease' in national forums. This was followed by advice from AIHW in April 2012 that the Commonwealth had commissioned two papers on 'possible national burden of disease work'. AIHW intends preparing these papers—which are to focus on technical issues associated with secondary aspects of the framework such as population attributable fractions and disability weights—with input from researchers involved in Murray and Lopez's most recent global analysis. In

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[209] In accordance with its undertaking to PHIDG AIHW also approached NHMRC. However, on the basis of advice that NHMRC submissions have, on average, around a 20% chance of success AIHW decided not to pursue this route.

[210] DOHA's representative to NBoDCN (personal communication, January 2011)

separate advice, it appears that APHDPC has all but dissolved as a principal committee of AHMAC.

It is too early at this stage to predict what priorities might emerge from these latest developments. Nevertheless, after two publicly funded national 'burden of disease studies' in Australia since Murray and Lopez coined the term in the early 1990s it is useful to reflect on how far we have come with respect to such activities in this country. Equally, it is important to be aware of the choices now available to us. If, as seems likely, we ultimately embark on a third 'study', we will be guilty of no worse than confirming the wisdom of past practices. If, on the other hand, we were to start using the Murray and Lopez framework as the basis for a comprehensive program of population health monitoring nationally, there is no reason why we could not realise practical opportunities from beyond this paradigm. As this thesis has attempted to illustrate, such an alternative is not only feasible but is also likely to have tangible benefits for health policy analysis and debate more broadly.

References

ABS, *Population Projections Australia 2002 to 2101*, (Cat. No. 3222.0), Australian Bureau of Statistics, Canberra, 2003.

ABS, *National Aboriginal and Torres Strait Islander Health Survey 2004-05*, (ABS cat. no. 4715.0), Australian Bureau of Statistics, Canberra, 2006.

ABS and AIHW, *The Health and Welfare of Australia's Aboriginal and Torres Strait Islander Peoples 2003*, (ABS cat. no. 4704.0, AIHW cat. no. IHW11), Australian Bureau of Statistics and Australian Institute of Health and Welfare, Canberra, 2003.

ABS and AIHW, *The Health and Welfare of Australia's Aboriginal and Torres Strait Islander Peoples 2005*, Australian Bureau of Statistics and Australian Institute of Health and Welfare, Canberra, 2005.

AIHW, *National diabetes register: statistical profile, December 2001*, Australian Institute of Health and Welfare, Canberra, 2003.

AIHW, *Child Protection Australia 2002-03*, Australian Institute of Health and Welfare, Canberra, 2004.

AIHW, *Disability and its relationship to health conditions and other factors*, (AIHW Cat. No. DIS 37), Australian Institute of Health and Welfare, Canberra, 2004.

AIHW, *Rural, Regional and Remote Health: A Guide to Remoteness Classifications*, (AIHW cat. no. PHE 53), Australian Institute of Health and Welfare, Canberra, 2004.

AIHW, *Female SAAP Clients and Children Escaping Domestic and Family Violence 2003-04*, Australian Institute of Health and Welfare, Canberra, 2005.

AIHW, *Health system expenditure on disease and injury in Australia, 2000-01*, (Cat. No. HWE 28), Australian Institute of Health and Welfare, Canberra, 2005.

AIHW, *Estimating the impact of selected National Health and Hospitals Reform Commission (NHHRC) reforms on health care expenditure, 2003 to 2033*, (Cat. no. HWE 45), Australian Institute of Health and Welfare, Canberra, 2009.

AIHW, *National Health Performance Framework 2009*, Canberra, 2009, viewed 12 August 2011, http://meteor.aihw.gov.au/content/index.phtml/itemId/392569.

AIHW, *Australia's health 2010 : the twelfth biennial health report of the Australian Institute of Health and Welfare*, Australian Institute of Health and Welfare, Canberra, 2010.

AIHW and NPHIWG, *National Public Health Information Plan 2005* Australian Institute of Health and Welfare, Canberra, 2005, viewed 12 August 2011, http://www.aihw.gov.au/publication-detail/?id=6442467796.

Arriaga, EE, "Measuring and explaining the change in life expectancies", *Demography*, 1984, vol. 21(1), pp. 83-96.

Australian Population Health Development Principal Committee, AHMAC, Canberra, viewed 12 August 2011, http://www.ahmac.gov.au/site/membership.aspx.

Australian Social Trends, Mar 2011 (Cat. no. 4102.0), Australian Bureau of Statistics, Canberra, viewed 21 August 2011, http://www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/4102.0Main+Features10Mar+2011.

Australian Treasury, *Intergenerational Report 2002-03*, Australian Government, Canberra, 2002.

Banks, G, *Health costs and policy in an ageing Australia*, in Health Policy Oration, Menzies Centre for Health Policy, John Curtin School of Medical Research, Australian National University, Canberra, 26 June 2008.

Barendregt, JJ, Van Oortmarssen, GJ, Vos, T and Murray, CJ, "A generic model for the assessment of disease epidemiology: the computational basis of DisMod II", *Popul Health Metr*, 2003, vol. 1(1), pp. 4.

Begg, S and Tomijima, N, *Global burden of injury in the year 2000: an overview of methods*, World Health Organization, Geneva, 2003.

Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A, *The burden of disease and injury in Australia, 2003*, (Cat. no.PHE 82), Australian Institute of Health and Welfare, Canberra, 2007.

Begg, S, Vos, T, Goss, J and Mann, N, "An alternative approach to projecting health expenditure in Australia", *Aust Health Rev*, 2008, vol. 32(1), pp. 148-55.

Begg, SJ, Vos, T, Barker, B, Stanley, L and Lopez, AD, "Burden of disease and injury in Australia in the new millennium: measuring health loss from diseases, injuries and risk factors", *Med J Aust*, 2008, vol. 188(1), pp. 36-40.

Bundhamcharoen, K, Teerawatananon, Y, Vos, T and Begg, S, *The Burden of Disease and Injuries in Thailand*, Bureau of Health Policy and Planning, Ministry of Public Health, Bangkok, 2002.

Burden of Disease (BoD) - LGAs & regions 1996, Department of Health Victoria, Melbourne, viewed 7 August 2011, http://www.health.vic.gov.au/healthstatus/composite/bod/bod96-index.htm.

Caldwell, JC, *What we know about health transition : the cultural, social and behavioural determinants of health : the proceedings of an international workshop, Canberra, May 1989*, Health Transition Centre, Australian National University, Canberra, 1990.

Caldwell, JC, "Demography and social science", *Popul Stud (Camb)*, 1996, vol. 50(3), pp. 305-33.

COAG, *National Health Information Agreement 2004-10*, AIHW, Canberra, 2004, viewed 12 August 2011, http://meteor.aihw.gov.au/content/index.phtml/itemId/182135.

Cunningham, J, O'Dea, K, Dunbar, T, Weeramanthri, T, Zimmet, P and Shaw, J, "Study protocol--diabetes and related conditions in urban indigenous people in the Darwin, Australia region: aims, methods and participation in the DRUID Study", *BMC Public Health*, 2006, vol. 6, pp. 8.

de Hollander, AEM, Hoeymans, N, Melse, JM, J.A.M., vO and Polder, JJ, *Zorg voor gezondheid: Volksgezondheid Toekomst Verkenning 2006*, Rijksinstituut voor Volksgezondheid en Milieu, Bilthoven, the Netherlands, 2006.

Department of Human Services and Health, *National Drug Strategy Household Survey: Urban Aboriginal and Torres Strait Islander Supplement, 1994*, Department of Human Services and Health, Canberra, 1995.

Dunstan, DW, Zimmet, PZ, Welborn, TA, De Courten, MP, Cameron, AJ, Sicree, RA, Dwyer, T, Colagiuri, S, Jolley, D, Knuiman, M, Atkins, R and Shaw, JE, "The rising prevalence of diabetes and impaired glucose tolerance: the Australian Diabetes, Obesity and Lifestyle Study", *Diabetes Care*, 2002, vol. 25(5), pp. 829-34.

Economic Policy Committee and the European Commission (DG ECFIN), *The impact of ageing on public expenditure: projections for the EU-25 Member States on pensions, healthcare, long-term care, education and unemployment transfers (2004–50)*. 2006.

English, DR, Holman, CDJ, Milne, E, Winter, MG, Hulse, GK, Codde, JP, Bower, CI, Corti, B, de Klerk, N, Knuiman, MW, Kurinczuk, JJ, Lewin, GF and Ryan, GA, *The quantification of drug caused morbidity and mortality in Australia*, Commonwealth Department of Human Services and Health, Canberra, 1995.

Ezzati, M, Hoorn, SV, Rodgers, A, Lopez, AD, Mathers, CD and Murray, CJ, "Estimates of global and regional potential health gains from reducing multiple major risk factors", *Lancet*, 2003, vol. 362(9380), pp. 271-80.

Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 1*, World Health Organization, Geneva, 2004.

Ezzati, M, Lopez, AD, Rodgers, A and Murray, CJL, eds, *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors - Volume 2*, World Health Organization, Geneva, 2004.

Flanagan, W, McIntosh, CN, Le Petit, C and Berthelot, JM, "Deriving utility scores for co-morbid conditions: a test of the multiplicative model for combining individual condition scores", *Popul Health Metr*, 2006, vol. 4, pp. 13.

Frauenthal, JC, "A dynamic model for human population growth", *Theor Popul Biol*, 1975, vol. 8(1), pp. 64-73.

Freeman, J and Hutchison, GB, "Prevalence, incidence and duration", *Am J Epidemiol*, 1980, vol. 112(5), pp. 707-23.

Freemantle, C, *Indicators of Infant and Childhood Mortality for Indigenous and Non-Indigenous Infants and Children Born in Western Australia from 1980 to 1997 Inclusive*, University of Western Australia, Perth, 2003.

Freemantle, CJ, Read, AW, de Klerk, NH, McAullay, D, Anderson, IP and Stanley, FJ, "Patterns, trends, and increasing disparities in mortality for Aboriginal and non-Aboriginal infants born in Western Australia, 1980-2001: population database study", *Lancet*, 2006, vol. 367(9524), pp. 1758-66.

Frenk, J, Bobadilla, JL, Stern, C, Frejka, T and Lozano, R, "Elements for a theory of the health transition", *Health Transit Rev*, 1991, vol. 1(1), pp. 21-38.

Fries, JF, "Aging, natural death, and the compression of morbidity", *N Engl J Med*, 1980, vol. 303(3), pp. 130-5.

Gail, MH and Bénichou, J, *Encyclopedia of epidemiologic methods*, Wiley, Chichester, 2000.

Gardiner-Garden, J, *From dispossession to reconciliation. Research Paper 27*, Commonwealth of Australia, Canberra, 1998-99, viewed 14 August 2011, http://www.aph.gov.au/library/pubs/rp/1998-99/99rp27.htm.

Goss, J, *Projection of Australian health care expenditure by disease, 2003 to 2033*, (Cat no. HWE 43), Australian Institute of Health and Welfare, Canberra, 2008.

Gruenberg, EM, "The failures of success", *Milbank Mem Fund Q Health Soc*, 1977, vol. 55(1), pp. 3-24.

Haby, MM, Markwick, A, Peeters, A, Shaw, J and Vos, T, "Future predictions of body mass index and overweight prevalence in Australia, 2005-2025", *Health Promot Int*, 2011.

Haby, MM, Vos, T, Carter, R, Moodie, M, Markwick, A, Magnus, A, Tay-Teo, KS and Swinburn, B, "A new approach to assessing the health benefit from obesity interventions in children and adolescents: the assessing cost-effectiveness in obesity project", *Int J Obes (Lond)*, 2006, vol. 30(10), pp. 1463-75.

Hare, J, "Whistleblower stonewalled as uni ignores orders to respond", *The Australian*, 30 May 2012, p. 5.

Health expenditure data cubes, Australian Insititute of Health and Welfare, Canberra, viewed 21 August 2011, http://www.aihw.gov.au/expenditure-data/.

Hill, K, Barker, B and Vos, T, "Excess Indigenous mortality: are Indigenous Australians more severely disadvantaged than other Indigenous populations?", *Int J Epidemiol*, 2007, vol. 36(3), pp. 580-9.

International Classification of Functioning, Disability and Health (ICF), World Health Organization, Geneva, viewed 16 August 2011, http://www.who.int/classifications/icf/en/.

International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, World Health Organization, Geneva, viewed 16 August 2011, http://www.who.int/classifications/icd/en/.

Johnston, G and Teasdale, A, *Population ageing and health spending: 50-year projections*, Ministry of Health, Wellington, 1999.

Katzenellenbogen, JM, Vos, T, Somerford, P, Begg, S, Semmens, JB and Codde, JP, "Excess mortality rates for estimating the non-fatal burden of stroke in Western Australia: a data linkage study", *Cerebrovasc Dis*, 2010, vol. 30(1), pp. 57-64.

Kruijshaar, ME, Barendregt, JJ and Hoeymans, N, "The use of models in the estimation of disease epidemiology", *Bull World Health Organ*, 2002, vol. 80(8), pp. 622-8.

Leske, MC, Ederer, F and Podgor, M, "Estimating incidence from age-specific prevalence in glaucoma", *Am J Epidemiol*, 1981, vol. 113(5), pp. 606-13.

Lopez, AD, Mathers, CD, Ezzati, M, Jamison, DT and Murray, CJL, eds, *Global burden of disease and risk factors*, Oxford University Press and the World Bank, New York, 2006.

Magliano, D, Peeters, A, Vos, T, Sicree, R, Shaw, J, Sindall, C, Haby, M, Begg, S and Zimmet, P, "Projecting the burden of diabetes in Australia - what is the size of the matter?", *Australian and New Zealand Journal of Public Health*, 2009, vol. 33(6), pp. 540-3.

Magliano, DJ, Shaw, JE, Shortreed, SM, Nusselder, WJ, Liew, D, Barr, EL, Zimmet, PZ and Peeters, A, "Lifetime risk and projected population prevalence of diabetes", *Diabetologia*, 2008, vol. 51(12), pp. 2179-86.

Manton, KG, "Changing concepts of morbidity and mortality in the elderly population", *Milbank Mem Fund Q Health Soc*, 1982, vol. 60(2), pp. 183-244.

Mathers, C, Penm, D, Carter, C and Stevenson, C, *Health system costs of diseases and injury in Australia 1993-94*, (Health and Welfare Expenditure Series No. 2), Australian Institute of Health and Welfare, Canberra, 1998.

Mathers, C, Salomon, J, Ezzati, M, Begg, S, Vander Hoorn, S and Lopez, A, "Sensitivity and uncertainty analyses for burden of disease and risk factor estimates", in Lopez, A, Mathers, C, Ezzati, M, Jamison, D and Murray, C, eds, *Global burden of disease and risk factors*, The World Bank and Oxford University Press, Washington, 2006.

Mathers, C, Vos, T and Stevenson, C, *The burden of disease and injury in Australia*, (Cat No. PHE 17), Australian Institute of Health and Welfare, Canberra, 1999.

Mathers, CD, "Gains in health expectancy from the elimination of diseases among older people", *Disabil Rehabil*, 1999, vol. 21(5-6), pp. 211-21.

Mathers, CD, Ezzati, M, Lopez, AD, Murray, CJL and Rodgers, AD, "Causal decomposition of summary measures of population health", in Murray, CJL, Salomon, J, Mathers, CD and Lopez, AD, eds, *Summary measures of population health: concepts, ethics, measurement and applications*, World Health Organization, Geneva, 2002.

Mathers, CD, Iburg, KM and Begg, S, "Adjusting for dependent comorbidity in the calculation of healthy life expectancy", *Popul Health Metr*, 2006, vol. 4(1), pp. 4.

Mathers, CD, Vos, ET, Stevenson, CE and Begg, SJ, "The Australian Burden of Disease Study: measuring the loss of health from diseases, injuries and risk factors", *Med J Aust*, 2000, vol. 172(12), pp. 592-6.

Mathers, CD, Vos, ET, Stevenson, CE and Begg, SJ, "The burden of disease and injury in Australia", *Bull World Health Organ*, 2001, vol. 79(11), pp. 1076-84.

Morgenstern, H and Bursic, ES, "A method for using epidemiologic data to estimate the potential impact of an intervention on the health status of a target population", *J Community Health*, 1982, vol. 7(4), pp. 292-309.

Murray, C, Salomon, J, Mathers, C and Lopez, A, eds, *Summary measures of population health: concepts, ethics, measurement and applications*, World Health Organization, Geneva, 2002.

Murray, CJ and Lopez, AD, "Quantifying disability: data, methods and results", *Bull World Health Organ*, 1994, vol. 72(3), pp. 481-94.

Murray, CJ and Lopez, AD, "Alternative projections of mortality and disability by cause 1990-2020: Global Burden of Disease Study", *Lancet*, 1997, vol. 349(9064), pp. 1498-504.

Murray, CJL and Lopez, AD, eds, *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Vol I.*, Harvard School of Public Health on behalf of WHO and the World Bank, Cambridge, 1996.

Murray, CJL and Lopez, AD, eds, *Global Health Statistics: a compendium of incidence, prevalence and mortality estimates for over 200 conditions. Vol II.*, Harvard School of Public Health on behalf of WHO and the World Bank, Cambridge, 1996.

Murray, CJL and Lopez, AD, *Health dimensions of sex and reproduction : the global burden of sexually transmitted diseases, HIV, maternal conditions, perinatal disorders, and congenital anomalies*, Harvard School of Public Health on behalf of WHO and the World Bank, Cambridge, 1998.

NACCHO, *Submission to the Commonwealth Parliamentary inquiry into the needs of urban dwelling Aboriginal and Torres Strait Islander peoples*, National Aboriginal Community Controlled Health Organisation, Canberra, 2001 viewed 14 August 2011, http://www.naccho.org.au/Files/Documents/Urbaninquirysubmission.pdf

National E-Health and Information Principal Committee, *National Health Information Management Principal Committee Strategic Work Plan 2007-08 to 2012-13*, Australian Health Ministers' Advisory Council, Canberra, 2007, viewed 12 August 2011, http://www.ahmac.gov.au/NHIMPC_Strategic_Work_Plan.pdf.

National Health Priority Action Council, *National Chronic Disease Strategy*, Australian Government Department of Health and Ageing, Canberra, 2006.

NHHRC, *A healthier future for all Australians : Interim report : December 2008*, National Health and Hospitals Reform Commission, Canberra, 2008.

NHHRC, *A healthier future for all Australians : final report : June 2009*, National Health and Hospitals Reform Commission, Canberra, 2009.

Nusselder, WJ and Looman, CW, "Decomposition of differences in health expectancy by cause", *Demography*, 2004, vol. 41(2), pp. 315-34.

O'Keefe, B, "Hackers pick up UQ cash prize", *The Australian*, 21 March 2007, p. 33.

O'Keefe, B, "Stats all, folks, for hackers", *The Australian*, 7 Feb 2007, p. 30.

O'Neill, T, Tallis, G and Leppard, P, "The epidemiology of a disease using hazard functions", *Australian Journal of Statistics*, 1985, vol. 27(3), pp. 283-97.

OECD, *Projecting OECD health and long-term care expenditures: What are the main drivers?*, Organisation for Economic Co-operation and Development, Paris, 2006, viewed 16 August 2011, http://www.oecd.org/dataoecd/57/7/36085940.pdf.

Olshansky, SJ, Rudberg, MA, Carnes, BA, Cassel, CK and Brody, JA, "Trading Off Longer Life for Worsening Health: The Expansion of Morbidity Hypothesis", *J Aging Health*, 1991, vol. 3(2), pp. 194-216.

Omran, AR, "The epidemiologic transition. A theory of the epidemiology of population change", *Milbank Mem Fund Q*, 1971, vol. 49(4), pp. 509-38.

Population Health Information Development Group (PHIDG), AIHW, Canberra, viewed 11 August 2011, http://www.aihw.gov.au/phidg/.

Preston, SH, "Relations among standard epidemiologic measures in a population", *Am J Epidemiol*, 1987, vol. 126(2), pp. 336-45.

Productivity Commission, *Economic implications of an Ageing Australia*, Productivity Commission, Canberra, 2005.

Ridolfo, B and Stevenson, C, *The quantification of drug-caused mortality and morbidity in Australia, 1998*, (7), Australian Institute of Health and Welfare, Canberra, 2001.

Risk factor Integral, Version 2.0, Epigear, Brisbane, viewed 12 August 2011, http://www.epigear.com/index_files/risk_factor.html.

Robine, J-M, *Determining health expectancies*, J. Wiley, Chichester, 2003.

Rodgers, A, Ezzati, M, Vander Hoorn, S, Lopez, AD, Lin, RB and Murray, CJ, "Distribution of major health risks: findings from the Global Burden of Disease study", *PLoS Med*, 2004, vol. 1(1), pp. e27.

Rowland, D, *Demongraphic methods and concepts*, Oxford University Press, New york, 2003.

Serghi, M, Begg, S and others, *Making tracks : toward closing the gap in health outcomes for Indigenous Queenslanders by 2033 : policy and accountability framework*, Queensland Health, Brisbane, Qld., 2010.

Sicree, R, Shaw, J and Zimmet, P, "Diabetes and impaired glucose tolerance", in Gan, D, ed., *Diabetes Atlas*, 3rd ed, International Diabetes Federation, Brussels, 2006.

StataCorp, *Stata Statistical Software: Release 11*, StataCorp LP, College Station, TX, 2009.

Steering Committee for the Review of Government Service Provision, *Overcoming Indigenous Disadvantage: Key Indicators 2005*, Productivity Commission, Canberra, 2005.

Stouthard, ME, Essink-Bot, M, Bonsel, GJ, Barendregt, JJ, Kramers, PGN, van de Water, HPA, Gunning-Schepers, LJ and van der Maas, PJ, *Disability weights for diseases in The Netherlands*, Department of Health, Erasmus University Rotterdam, Rotterdam, 1997.

Sullivan, DF, "A single index of mortality and morbidity", *HSMHA Health Rep*, 1971, vol. 86(4), pp. 347-54.

Taylor, R, Dobson, A and Mirzaei, M, "Contribution of changes in risk factors to the decline of coronary heart disease mortality in Australia over three decades", *Eur J Cardiovasc Prev Rehabil*, 2006, vol. 13(5), pp. 760-8.

The State of Queensland (Department of the Premier and Cabinet), *Toward Q2: Tomorrow's Queensland*, Queensland Government, Brisbane, 2008, viewed 19 August 2011, http://www.towardq2.qld.gov.au/tomorrow/library/pdf/Towards_Q2_Tomorrows_Queensland.pdf.

Toy, M, "Our sickest suburbs", *The Age*, 17 January 2001 2001, p. p1.

Truelsen, T, Begg, S and Mathers, C, *The global burden of cerebrovascular disease*, World Health Organization, Geneva, 2003.

Unal, B, Critchley, JA and Capewell, S, "Explaining the decline in coronary heart disease mortality in England and Wales between 1981 and 2000", *Circulation*, 2004, vol. 109(9), pp. 1101-7.

United Nations, *World Population Ageing 1950-2050*, 2002.

van Baal, PH, Hoeymans, N, Hoogenveen, RT, de Wit, GA and Westert, GP, "Disability weights for comorbidity and their influence on health-adjusted life expectancy", *Popul Health Metr*, 2006, vol. 4, pp. 1.

Victorian Department of Human Services, *The Victorian Burden of Disease Study: Mortality and Morbidity in 2001*, Victorian Department of Human Services, Melbourne, 2005.

Vos, ET, *Updating the Australian Burden of Disease: small areas estimates component study*, funded by Rural Health, Palliative Care and Health Strategies Branch, DOHA.

Vos, T, Barker, B, Begg, S, Stanley, L and Lopez, AD, "Burden of disease and injury in Aboriginal and Torres Strait Islander Peoples: the Indigenous health gap", *Int J Epidemiol*, 2009, vol. 38(2), pp. 470-7.

Vos, T, Barker, B, Stanley, L and Lopez, A, *The Burden of Disease and Injury in Aboriginal and Torres Strait Islander Peoples 2003*, School of Population Health, The University of Queensland, Brisbane, 2007.

Vos, T and Begg, S, *Victorian Burden of Disease Study: Morbidity*, Public Health Division, Victorian Department of Human Services, Melbourne, 1999.

Vos, T and Begg, S, *Victorian Burden of Disease Study: Mortality*, Public Health Division, Victorian Department of Human Services, Melbourne, 1999.

Vos, T, Carter, R, Barendregt, J, Mihalopoulos, C, Veerman, J, Magnus, A, Cobiac, L, Bertram, M, Wallace, A and ACE–Prevention Team, *Assessing Cost-Effectiveness in Prevention (ACE–Prevention): Final Report.* , University of Queensland and Deakin University, Brisbane & Melbourne, 2010.

Vos, T, Goss, J, Begg, S and Mann, N, *Australian Burden of Disease and Injury Study: Projected health care costs report*, University of Queensland, Brisbane, Australia, 2005.

Wang, Z and Hoy, WE, "Hypertension, dyslipidemia, body mass index, diabetes and smoking status in Aboriginal Australians in a remote community", *Ethn Dis*, 2003, vol. 13(3), pp. 324-30.

WHO, *World Health Report 2001- Mental Health: New understanding, New Hope.*, World Health Organization, Geneva, 2001.

WHO, *World Health Report 2002- Reducing Risks, Promoting Healthy Life*, World Health Organization, Geneva, 2002.

WHO, *World Health Report 2003 - Shaping the future*, World Health Organization, Geneva, 2003.

WHO, *World Health Report 2004 - Changing history*, World Health Organization, Geneva, 2004.

Wilson, T, Condon, JR and Barnes, T, "Northern Territory indigenous life expectancy improvements, 1967-2004", *Aust N Z J Public Health*, 2007, vol. 31(2), pp. 184-8.

Woodward, M, Zhang, X, Barzi, F, Pan, W, Ueshima, H, Rodgers, A and MacMahon, S, "The effects of diabetes on the risks of major cardiovascular diseases and death in the Asia-Pacific region", *Diabetes Care*, 2003, vol. 26(2), pp. 360-6.

World Bank, *World Development Report: Investing in Health*, Oxford University Press, New York, 1993.

Yusoff, A, Mustafa, A, Kaur, G, Omar, M, Vos, T, Rao, V and Begg, S, *Malaysian Burden of Disease and Injury Study*, Division of Burden of Disease, Institute of Public Health, Ministry of Health, Kuala Lumpur, 2004.

Zhao, Y, Guthridge, S, Magnus, A and Vos, T, "Burden of disease and injury in Aboriginal and non-Aboriginal populations in the Northern Territory", *Med J Aust*, 2004, vol. 180(10), pp. 498-502.

Part V: Appendixes

Appendix A: Summary of practical experience with the Murray and Lopez framework

Population	Baseline	Project sponsor	Terms of engagement
Australia			
All states	1996	Australian Institute of Health & Welfare	Epidemiologist, Victorian Department of Human Services
All states	2003	Commonwealth Department of Health & Ageing	Lecturer, University of Queensland
All states	2003	Commonwealth Treasury	Consultant
All states	2003	UN	Consultant
All states (Indigenous)	2003	National Health & Hospital Reform Commission	In kind contribution
All states (risk factors)	2003	National Heart Foundation	Consultant
Victoria	1996	Victorian Department of Human Services	Epidemiologist, Victorian Department of Human Services
Victoria	2001	Victorian Department of Human Services	In kind contribution
WA	2006	WA Department of Health	Consultant
SA	2006	SA Department of Health	Consultant
NT	2003	NT Department of Health	Consultant
Queensland	2003	Queensland Health	Lecturer, University of Queensland
Queensland	2006	Queensland Health	Consultant
Queensland (Indigenous)	2006	Queensland Aboriginal & Islander Health Council	In kind contribution
Queensland	2008	Queensland Health	Director, Queensland Health
NSW	2003	NSW Department of Health	In kind contribution
Thailand	1999	Ministry of Health	Consultant
USA	1996	Centers for Disease Control & Prevention	Research Fellow, Harvard University
Turkey	2000	Baskent University	Consultant
Malaysia	2005	Ministry of Health	Consultant
Singapore	2004	Ministry of Health	Consultant
World (injuries)	2000	World Health Organization	Consultant
World (stroke)	2000	World Health Organization	Scientist, World Health Organization
World	n.a.	World Health Organization	Consultant

Appendix B: IPM subroutines

The code below consists of the following Mata subroutines: `mata_IPM_calc`, which performs most of the calculations set out by Barendregt and colleagues,[211] except duration; and `mata_dur_calc`, which performs the duration calculations. These routines are used by the Stata ado programs `cohort-IPM`, `subpop-IPM` and `Indig-IPM`, as indicated in Appendices C, D and E, respectively.

```
real matrix mata_IPM_calc(real matrix i, real matrix r, real matrix f)
{
	S=J(101,1,.)
	C=J(101,1,.)
	D=J(101,1,.)
	PY=J(101,1,.)
	c=J(101,1,.)
	b=J(101,1,.)
	l = i :+ f :+ r
	q = sqrt(i :^2 :+ 2 :* i :* r :- 2 :* i :* f :+ r :^2 :+ 2 :* f :* r :+ f
:^2)
	w = exp(-1 :* (l :+ q) :/ 2)
	v = exp(-1 :* (l :- q) :/ 2)
	for (a=1;a<=101;a++) {
		subS=(a==1 ? 1000 : S[a-1])
		subC=(a==1 ? 0 : C[a-1])
		subD=(a==1 ? 0 : D[a-1])
		S[a] =max((0,(q[a]==0 ? subS : (2 * (v[a] - w[a]) * (subS * (f[a] +
r[a]) + subC * r[a]) + subS * (v[a] * (q[a] - l[a]) + w[a] * (q[a] + l[a])))) / (2 *
q[a]))))
		C[a] =max((0,(q[a]==0 ? subC : -1 * ((v[a] - w[a]) * (2 * ((f[a] +
r[a]) * (subS + subC) - l[a] * subS) - subC * l[a]) - subC * q[a] * (v[a] + w[a]))
/ (2 * q[a]))))
		D[a] =max((0,(q[a]==0 ? subD : ((v[a] - w[a]) * (2 * f[a] * subC -
l[a] * (subS + subC)) - q[a] * (subS + subC) * (v[a] + w[a]) + 2 * q[a] * (subS +
subC + subD)) / (2 * q[a]))))
		PY[a] = 0.5 * (subS + subC + S[a] + C[a])
		c[a] = 0.5 * ((subC + C[a]) / PY[a])
		b[a] = (D[a] - subD) / PY[a]
	}
	return(c,b)
}

real matrix mata_dur_calc(real matrix B, real matrix i)
{
	Dur=J(101,1,0)
	y=J(101,1,.)
	x=J(101,1,.)
	for (a=1;a <=101;a++) {
		if (i[a]>0) {
			if (exp(-i[a])~=1) {
				if (B[a]==0) {
					y[a]=1
					x[a]=.5
				}
				else if (B[a]==i[a]) {
					y[a]=i[a]*(exp(-i[a])/(1-exp(-i[a])))
```

[211] Barendregt, JJ, Van Oortmarssen, GJ, Vos, T and Murray, CJ, "A generic model for the assessment of disease epidemiology: the computational basis of DisMod II".

```
                                x[a]=1/B[a]-1/(exp(B[a])-1)
                        }
                        else {
                                temp=(exp(-B[a])-exp(-i[a]))/(1-exp(-i[a]))
                                y[a]=(i[a]/(i[a]-B[a]))*temp
                                x[a]=1/B[a]-(i[a]/((i[a]-B[a])*B[a]))*temp
                        }
                        for (k=a+1;k <=100;k++) {
                                if (B[k]==0) {
                                        y[k]=y[k-1]
                                        x[k]=x[k-1]
                                }
                                else {
                                        y[k]=y[k-1]*exp(-B[k])
                                        x[k]=(y[k-1]/B[k])*(1-exp(-B[k]))
                                }
                        }
                        if (B[a]==0) {
                                if (a==101) x[101]=1000
                                else x[101]=y[100]*1000
                        }
                        else {
                                if (a==101) x[101]=1/B[101]
                                else x[101]=y[100]/B[101]
                        }
                        for (k=a;k <=101;k++) {
                                Dur[a]=Dur[a]+x[k]
                        }
                }
        }
}
return(Dur)
}
```

Appendix C: `Cohort-IPM` routine

The Stata ado program `cohort-IPM` is a 'wrapping' routine that calls the Mata subroutine `mata_cohort-IPM`, which, in turn, executes `mata_IPM_calc` and `mata_dur_calc` for each age cohort as it ages over time. Instructions on how to use `cohort-IPM` are provided in Chapter 2. The code for `mata_IPM_calc` and `mata_dur_calc` is included at Appendix B.

```
program define cohort-IPM, byable(recall)
      version 9.1
      syntax  varname(numeric) using/ [if],  Sex(varname numeric ) Inc(varname
numeric) Cf(varname numeric) Rem(varname numeric) [Lag(varname numeric)]
[Year(varname numeric)]
      marksample touse
      local index=_byindex()
      quietly if `index'==1 {
            local allvars "`_byvars'  `sex' `varlist' `year'"
            global allvars : list uniq allvars
            global reshapevars : list allvars - year
            global hazvars "`inc' `cf' `rem'"
            cap confirm var `varlist'
            cap bysort `_byvars' `varlist' `sex' `year': assert _N==1
            if _rc~=0 {
                  di in red "the variables $allvars do not uniquely identify the
data"
                  error 1
            }
            assert inlist(sex,1,2)
            cap gen _prev=.
            cap gen _dur=.
            mata: mata matuse "`using'", replace
            mata: st_matrix("pop_m",pop_m)
            mata: st_matrix("pop_f",pop_f)
            mata: st_matrix("mort_m",mort_m)
            mata: st_matrix("mort_f",mort_f)
      }
      quietly sum `sex' if `touse'
      local sexval=r(max)
      if `sexval'==1{
            local mort "mort_m"
            local pop "pop_m"
      }
      else {
            local mort "mort_f"
            local pop "pop_f"
      }
      if "`lag'"~="" {
            quietly sum `lag' if `touse'
            local lagflag=r(max)>0
      }
      else local lagflag=0
      mata: mata_cohort-IPM("`1'","`inc'","`rem'","`cf'","`lag'",
`lagflag',"`year'")
end

version 9.1
mata:
mata clear
void mata_cohort-IPM(string scalar agecat,string scalar inc,string scalar rem,
string scalar cf,string scalar lag,numeric scalar lagflag, string scalar year)
```

```
{
	tM=st_matrix(st_local("mort"))
	pop=st_matrix(st_local("pop"))
	st_view(data_in,.,(agecat,year,inc,cf,rem),st_local("touse"))
	agecat=uniqrows(data_in[.,1])
	agerows=rows(agecat)
	i=rowshape(data_in[.,3],agerows)
	f=rowshape(data_in[.,4],agerows)
	r=rowshape(data_in[.,5],agerows)
	years=cols(i)
	for (k=1;k<=agerows;k++) {
		thisage=agecat[k]
		nextage=(k==agerows ? 101 : agecat[k+1])
		mult=J(nextage-thisage,years,1)
		trend_i=(k==1 ? mult :* i[k,.] : trend_i\mult :* i[k,.])
		trend_f=(k==1 ? mult :* f[k,.] : trend_f\mult :* f[k,.])
		trend_r=(k==1 ? mult :* r[k,.] : trend_r\mult :* r[k,.])
	}
	for (k=1;k<=years+100;k++) {
		row1=max((102-k,1))
		col1=max((1,k-100))
		col2=min((k,years))
		row2=row1+(col2-col1)
		i=diagonal(trend_i[|row1,col1\row2,col2|])
		f=diagonal(trend_f[|row1,col1\row2,col2|])
		r=diagonal(trend_r[|row1,col1\row2,col2|])
		if (row1-1>=1) {
			i=trend_i[|1,1\(row1-1),1|]\i
			f=trend_f[|1,1\(row1-1),1|]\f
			r=trend_r[|1,1\(row1-1),1|]\r
		}
		if (row2+1<=101) {
			i=i\trend_i[|(row2+1),years\.,years|]
			f=f\trend_f[|(row2+1),years\.,years|]
			r=r\trend_r[|(row2+1),years\.,years|]
		}

		IPM_calc=mata_IPM_calc(i,r,f)

		trend_c=(k==1 ? IPM_calc[.,1] : trend_c,IPM_calc[.,1])
		trend_b=(k==1 ? IPM_calc[.,2] : trend_b,IPM_calc[.,2])
		trend_f1=(k==1 ? f : trend_f1,f)
		trend_r1=(k==1 ? r : trend_r1,r)
		trend_i1=(k==1 ? i : trend_i1,i)
	}
	trend_c=trend_c[(101::1),.]
	trend_b=trend_b[(101::1),.]
	trend_f1=trend_f1[(101::1),.]
	trend_r1=trend_r1[(101::1),.]
	trend_i1=trend_i1[(101::1),.]
	for (k=1;k<=years;k++) {
		c1=diagonal(trend_c[|1,k\.,.|])[(101::1),.]
		b1=diagonal(trend_b[|1,k\.,.|])[(101::1),.]
		f1=diagonal(trend_f1[|1,k\.,.|])[(101::1),.]
		r1=diagonal(trend_r1[|1,k\.,.|])[(101::1),.]
		i1=diagonal(trend_i1[|1,k\.,.|])[(101::1),.]

		B = r1 :+ f1 :+ (tM[.,k] :- b1)
		d1=mata_dur_calc(B,i1)
		i_num=i1:*pop[.,k]
		for (a=1;a<=agerows;a++) {
			thisage=agecat[a]
			nextage=(a==agerows ? 101 : agecat[a+1])
			temp=sum(c1[|thisage+1\nextage|] :*
pop[|thisage+1,k\nextage,k|]) / sum(pop[|thisage+1,k\nextage,k|] )
			c2=(a==1 ? (a,k,temp) : c2\(a,k,temp))
			temp=(sum(i_num[|thisage+1\nextage|] )==0 ? 0 :
sum(d1[|thisage+1\nextage|] :* i_num[|thisage+1\nextage|]) /
```

```
sum(i_num[|thisage+1\nextage|] ))
                    d2=(a==1 ? (a,k,temp) : d2\(a,k,temp))
             }
             c3=(k==1 ? c2: c3\c2)
             d3=(k==1 ? d2: d3\d2)
      }
      _sort(c3,(1,2))
      _sort(d3,(1,2))
      st_store(.,"_prev",st_local("touse"),c3[.,3])
      st_store(.,"_dur",st_local("touse"),d3[.,3])
}
end
```

Appendix D: `Subpop-IPM` routine

The Stata ado program `subpop-IPM` is a 'wrapping' routine that calls the Mata subroutine `mata_subpop-IPM`, which, in turn, executes `mata_IPM_calc` and `mata_dur_calc` for 15 subpopulations stratified by three remoteness categories (major cities, regional areas and remote areas) and five socioeconomic quintiles. Instructions on how to use `subpop-IPM` are provided in Chapter 2. The code for `mata_IPM_calc` and `mata_dur_calc` is included at Appendix B.

```
program define subpop-IPM, byable(recall, noheader)
      version 9.1
      syntax  varname(numeric) using/ [if],  Sex(varname) Inc(varname numeric)
Cf(varname numeric) Rem(varname numeric) Subpopulation(varlist numeric)
      tokenize `varlist'
      local age `1'
      marksample touse
      local index=_byindex()
      local firstcase=_byn1()
      quietly if `index'==1 {
            cap gen _prev=.
            cap gen _dur=.
            mata: mata matuse "`using'", replace
            mata: st_matrix("pop_m_1_1",pop_m_1_1)
            mata: st_matrix("pop_m_2_1",pop_m_2_1)
            mata: st_matrix("pop_m_3_1",pop_m_3_1)
            mata: st_matrix("pop_m_4_1",pop_m_4_1)
            mata: st_matrix("pop_m_5_1",pop_m_5_1)
            mata: st_matrix("pop_m_1_2",pop_m_1_2)
            mata: st_matrix("pop_m_2_2",pop_m_2_2)
            mata: st_matrix("pop_m_3_2",pop_m_3_2)
            mata: st_matrix("pop_m_4_2",pop_m_4_2)
            mata: st_matrix("pop_m_5_2",pop_m_5_2)
            mata: st_matrix("pop_m_1_3",pop_m_1_3)
            mata: st_matrix("pop_m_2_3",pop_m_2_3)
            mata: st_matrix("pop_m_3_3",pop_m_3_3)
            mata: st_matrix("pop_m_4_3",pop_m_4_3)
            mata: st_matrix("pop_m_5_3",pop_m_5_3)
            mata: st_matrix("pop_f_1_1",pop_f_1_1)
            mata: st_matrix("pop_f_2_1",pop_f_2_1)
            mata: st_matrix("pop_f_3_1",pop_f_3_1)
            mata: st_matrix("pop_f_4_1",pop_f_4_1)
            mata: st_matrix("pop_f_5_1",pop_f_5_1)
            mata: st_matrix("pop_f_1_2",pop_f_1_2)
            mata: st_matrix("pop_f_2_2",pop_f_2_2)
            mata: st_matrix("pop_f_3_2",pop_f_3_2)
            mata: st_matrix("pop_f_4_2",pop_f_4_2)
            mata: st_matrix("pop_f_5_2",pop_f_5_2)
            mata: st_matrix("pop_f_1_3",pop_f_1_3)
            mata: st_matrix("pop_f_2_3",pop_f_2_3)
            mata: st_matrix("pop_f_3_3",pop_f_3_3)
            mata: st_matrix("pop_f_4_3",pop_f_4_3)
            mata: st_matrix("pop_f_5_3",pop_f_5_3)
            mata: st_matrix("mort_m_1_1",mort_m_1_1)
            mata: st_matrix("mort_m_2_1",mort_m_2_1)
            mata: st_matrix("mort_m_3_1",mort_m_3_1)
            mata: st_matrix("mort_m_4_1",mort_m_4_1)
            mata: st_matrix("mort_m_5_1",mort_m_5_1)
            mata: st_matrix("mort_m_1_2",mort_m_1_2)
            mata: st_matrix("mort_m_2_2",mort_m_2_2)
```

```
		mata: st_matrix("mort_m_3_2",mort_m_3_2)
		mata: st_matrix("mort_m_4_2",mort_m_4_2)
		mata: st_matrix("mort_m_5_2",mort_m_5_2)
		mata: st_matrix("mort_m_1_3",mort_m_1_3)
		mata: st_matrix("mort_m_2_3",mort_m_2_3)
		mata: st_matrix("mort_m_3_3",mort_m_3_3)
		mata: st_matrix("mort_m_4_3",mort_m_4_3)
		mata: st_matrix("mort_m_5_3",mort_m_5_3)
		mata: st_matrix("mort_f_1_1",mort_f_1_1)
		mata: st_matrix("mort_f_2_1",mort_f_2_1)
		mata: st_matrix("mort_f_3_1",mort_f_3_1)
		mata: st_matrix("mort_f_4_1",mort_f_4_1)
		mata: st_matrix("mort_f_5_1",mort_f_5_1)
		mata: st_matrix("mort_f_1_2",mort_f_1_2)
		mata: st_matrix("mort_f_2_2",mort_f_2_2)
		mata: st_matrix("mort_f_3_2",mort_f_3_2)
		mata: st_matrix("mort_f_4_2",mort_f_4_2)
		mata: st_matrix("mort_f_5_2",mort_f_5_2)
		mata: st_matrix("mort_f_1_3",mort_f_1_3)
		mata: st_matrix("mort_f_2_3",mort_f_2_3)
		mata: st_matrix("mort_f_3_3",mort_f_3_3)
		mata: st_matrix("mort_f_4_3",mort_f_4_3)
		mata: st_matrix("mort_f_5_3",mort_f_5_3)
	}
	tokenize `subpopulation'
	local seifaval=`1'[`firstcase']
	local remoteval=`2'[`firstcase']
	local sexval=`sex'[`firstcase']
	if `sexval'==1{
		local mort "mort_m_`seifaval'_`remoteval'"
		local pop "pop_m_`seifaval'_`remoteval'"
	}
	else {
		local mort "mort_f_`seifaval'_`remoteval'"
		local pop "pop_f_`seifaval'_`remoteval'"
	}
	mata: mata_subpop-IPM("`age'","`inc'","`rem'","`cf'")
end

version 9.1
mata:
mata clear
void mata_subpop-IPM(string scalar agecat,string scalar inc,string scalar rem,
string scalar cf)
{
	tM=st_matrix(st_local("mort"))
	pop=st_matrix(st_local("pop"))
	st_view(age_in,.,agecat,st_local("touse"))
	st_view(i_in,.,inc,st_local("touse"))
	st_view(f_in,.,cf,st_local("touse"))
	st_view(r_in,.,rem,st_local("touse"))
	agerows=rows(age_in)
	age=(0::100)
	i=J(101,1,0)
	f=J(101,1,0)
	r=J(101,1,0)
	for (a=age_in[agerows]+1;a<=101;a++) {
		i[a]=i_in[agerows]
		f[a]=f_in[agerows]
		r[a]=r_in[agerows]
	}
	for (k=1;k<=agerows-1;k++) {
		for (a=age_in[k]+1;a<=age_in[k+1];a++) {
			i[a]=i_in[k]
			f[a]=f_in[k]
			r[a]=r_in[k]
		}
	}
```

```
	IPM_calc=mata_IPM_calc(i,r,f)
	c= IPM_calc[.,1]
	B = r :+ f :+ (tM :- IPM_calc[.,2])
	Dur=mata_dur_calc(B,i)
	i_num=i:*pop
	for (a=1;a<=agerows;a++) {
		temp=sum(c[|age_in[a]+1\(a==agerows ? 101 : age_in[a+1])|] :*
pop[|age_in[a]+1\(a==agerows ? 101 : age_in[a+1])|]) /
sum(pop[|age_in[a]+1\(a==agerows ? 101 : age_in[a+1])|] )
		c_out=(a==1 ? temp : c_out\temp)
		temp=(sum(i_num[|age_in[a]+1\(a==agerows ? 101 : age_in[a+1])|] )==0 ?
0 : sum(Dur[|age_in[a]+1\(a==agerows ? 101 : age_in[a+1])|] :*
i_num[|age_in[a]+1\(a==agerows ? 101 : age_in[a+1])|]) /
sum(i_num[|age_in[a]+1\(a==agerows ? 101 : age_in[a+1])|] ))
		Dur_out=(a==1 ? temp : Dur_out\temp)
	}
	st_store(.,"_prev",st_local("touse"),c_out)
	st_store(.,"_dur",st_local("touse"),Dur_out)
}
end
```

Appendix E: `Indig-IPM` routine

The Stata ado program `Indig-IPM` is a 'wrapping' routine that calls the Mata subroutine `mata_Indig-IPM`, which, in turn, executes `mata_IPM_calc` and `mata_dur_calc` for each of two Indigenous populations (major cities and regional areas combined, and remote areas). Instructions on how to use `Indig-IPM` are provided in Chapter 2. The code for `mata_IPM_calc` and `mata_dur_calc` is included at Appendix B.

```
program define Indig-IPM, byable(recall, noheader)
	version 9.1
	syntax  varname(numeric) using/ [if],  Sex(varname) Inc(varname numeric)
Cf(varname numeric) Rem(varname numeric) Remote(varlist numeric)
	tokenize `varlist'
	local age `1'
	marksample touse
	local index=_byindex()
	local firstcase=_byn1()
	quietly if `index'==1 {
		cap gen _prev=.
		cap gen _dur=.
		mata: mata matuse "`using'", replace
		mata: st_matrix("pop_m_1",pop_m_1)
		mata: st_matrix("pop_m_2",pop_m_2)
		mata: st_matrix("pop_f_1",pop_f_1)
		mata: st_matrix("pop_f_2",pop_f_2)
		mata: st_matrix("mort_m_1",mort_m_1)
		mata: st_matrix("mort_m_2",mort_m_2)
		mata: st_matrix("mort_f_1",mort_f_1)
		mata: st_matrix("mort_f_2",mort_f_2)
	}
	local remoteval=`remote'[`firstcase']
	local sexval=`sex'[`firstcase']
	if `sexval'==1{
		local mort "mort_m_`remoteval'"
		local pop "pop_m_`remoteval'"
	}
	else {
		local mort "mort_f_`remoteval'"
		local pop "pop_f_`remoteval'"
	}
	mata: mata_Indig-IPM("`age'","`inc'","`rem'","`cf'")
end

version 9.1
mata:
mata clear
void mata_Indig-IPM(string scalar agecat,string scalar inc,string scalar rem,
string scalar cf)
{
	tM=st_matrix(st_local("mort"))
	pop=st_matrix(st_local("pop"))
	st_view(age_in,.,agecat,st_local("touse"))
	st_view(i_in,.,inc,st_local("touse"))
	st_view(f_in,.,cf,st_local("touse"))
	st_view(r_in,.,rem,st_local("touse"))
	agerows=rows(age_in)
	age=(0::85)
	i=J(86,1,0)
```

```
        f=J(86,1,0)
        r=J(86,1,0)
        for (a=age_in[agerows]+1;a<=86;a++) {
                i[a]=i_in[agerows]
                f[a]=f_in[agerows]
                r[a]=r_in[agerows]
        }
        for (k=1;k<=agerows-1;k++) {
               for (a=age_in[k]+1;a<=age_in[k+1];a++) {
                      i[a]=i_in[k]
                      f[a]=f_in[k]
                      r[a]=r_in[k]
               }
        }
        dismod_calc=mata_dismod_calc(i,r,f)
        c= dismod_calc[.,1]
        B = r :+ f :+ (tM :- dismod_calc[.,2])
        Dur=mata_dur_calc(B,i)
        i_num=i:*pop
        for (a=1;a<=agerows;a++) {
               temp=sum(c[|age_in[a]+1\(a==agerows ? 86 : age_in[a+1])|] :*
pop[|age_in[a]+1\(a==agerows ? 86 : age_in[a+1])|]) /
sum(pop[|age_in[a]+1\(a==agerows ? 86 : age_in[a+1])|] )
               c_out=(a==1 ? temp : c_out\temp)
               temp=(sum(i_num[|age_in[a]+1\(a==agerows ? 86 : age_in[a+1])|] )==0 ?
0 : sum(Dur[|age_in[a]+1\(a==agerows ? 86 : age_in[a+1])|] :*
i_num[|age_in[a]+1\(a==agerows ? 86 : age_in[a+1])|]) /
sum(i_num[|age_in[a]+1\(a==agerows ? 86 : age_in[a+1])|] ))
               Dur_out=(a==1 ? temp : Dur_out\temp)
        }
        st_store(.,"_prev",st_local("touse"),c_out)
        st_store(.,"_dur",st_local("touse"),Dur_out)
}
```

Appendix F: Comorbidity routines

The set of routines below implement the comorbidity correction principles outlined in Chapter 2.

Prepare data

```
local group1="A B C D E"
local group2="F G H I J K L M N O P Q R S Z"
local group3="T U"
local files=`""NHS2001 Comorbidity" "SDAC2003 COMORBIDITY" "MHS1997 Comorbidity"
"AusDiab Comorbidity""'
quietly foreach file of local files  {
       odbc load, table("`file'") clear
       if "`file'"=="NHS2001 Comorbidity" drop J* H??
       if "`file'"=="SDAC2003 COMORBIDITY" drop J02-J04 ZILL INJURIES-XN33_DIS
       rename AGE agecat
       rename SEX sex
       foreach var of varlist * {
              if inlist("`var'","sex","agecat","TOTAL") continue
              local firstchar=substr("`var'",1,1)
              forvalues x=1/3{
                     local groupflag : list firstchar in group`x'
                     if `groupflag'==1 local group=`x'
              }
              gen v`var'=`var'==1
       }
       recode agecat 75/100=75
       tokenize "`file'"
       aorder
       collapse (sum) total`1'=TOTAL if inrange(agecat,25,75), by(sex agecat v*)
       save "`1'", replace
       noisily des
}
tempfile data
local files="NHS2001 SDAC2003 MHS1997 AusDiab"
quietly foreach file of local files  {
       use `file', clear
       gen source="`file'"
       cap append using `data'
       save `data', replace
}
egen total=rsum(total*)
save "Combined comorbidity data", replace
```

Run simulations

```
tempfile sexage sourcedata sequence combined
use "Combined comorbidity data", clear
save `combined'
collapse (sum) total, by(sex age)
egen double prob=pc(total), prop
replace prob=prob+prob[_n-1] if _n~=1
gen count=_n
save `sexage'
mkmat  sex agecat prob, matrix(sexage)

use "Combined comorbidity data", clear
collapse (count) v*, by(age sex source)
save `sourcedata'
collapse (count) v*, by(source)
```

```
reshape long v, i(source) j(cause) string
levelsof cause, local(allvars) clean

file open hndl using c:\temp\simulations.txt, write text replace
file write hndl "sex" _tab "age" _tab
foreach var of local allvars {
	file write hndl "`var'"
	if "`ferest()'"~="" file write hndl _tab
}
local starttime= c(current_time)
file write hndl _newline
quietly forvalues z=1/1000000 {
	noisily di `z'
	use `sexage', clear
	local rnd=uniform()
	sum count if prob>`rnd'
	local row=r(min)
	local sex=sex[`row']
	local age=age[`row']
	file write hndl "`sex'" _tab "`age'" _tab
	use `sourcedata' if sex==`sex' & age==`age', clear
	gen sourceorder=uniform()
	sort sourceorder
	replace sourceorder=_n
	reshape long v, i(age sex source) j(cause) string
	reshape wide v sourceorder, i(age sex cause) j(source) string
	gen order=uniform()
	sort order
	replace order=_n
	local maxorder=_N
	reshape long
	keep if v~=0
	sort age sex source order
	by age sex source: gen count=_n
	sort age sex order count sourceorder
	by age sex order : gen max= _n==_N
	save `sequence', replace
	macro drop outcome*
	forvalues x=1/`maxorder'  {
		use `sequence', clear
		levelsof source if order==`x' & max==1, local(source) clean
		levelsof order if order==`x' & max==1, local(order) clean
		levelsof cause if order==`x' & max==1, local(targetvar) clean
		local conditionalvars=""
		cap levelsof cause if source=="`source'" & order<=`order' &
cause~="`targetvar'", local(conditionalvars) clean
		use `combined' if sex==`sex' & age==`age' & source=="`source'", clear
		renpfix v
		foreach conditionalvar of local conditionalvars {
			count if `conditionalvar'==`outcome`conditionalvar''
			if r(N)~=0 {
				keep if `conditionalvar'==`outcome`conditionalvar''
			}
		}
		collapse (sum) total,by(`targetvar')
		egen prob=pc(total), prop
		replace prob=prob+prob[_n-1] if _n~=1
		local rnd=uniform()
		sum `targetvar' if prob>`rnd'
		local outcome`targetvar'= r(min)
	}
	foreach var of local allvars {
		file write hndl "`outcome`var''"
		if "`ferest()'"~="" file write hndl _tab
	}
	file write hndl _newline
}
local endtime= c(current_time)
```

```
file close hndl
noisily di "Start time: `starttime' End time: `endtime'"
}
insheet using "simulations.txt", clear
foreach var of varlist a-z02 {
	local varname=upper("`var'")
	rename `var' flag`varname'
}
rename age agecat
contract sex age flag*, freq(num)
egen prev=pc(num), by(sex age) prop
save "simulations", replace
```

Create adjustment factors

```
use "s:\bod\aus trends\Epi by bodicode, seq, sex, age 1979 to 2033" if year==2003,
clear
keep bodicode seq sex agecat dw _prev3 pop comorbid_code inc
tempfile dw
save `dw'
replace _prev3=_prev3*pop
drop if comorb=="XXXX"
rename bodicode code
bysort code seq sex (agecat): replace dw=dw[_n-1] if _n>1 & dw[_n]==0 & dw[_n-1]>0
& dw[_n-1]<=1
gsort code seq sex -agecat
by code seq sex : replace dw=dw[_n-1] if _n>1 & dw[_n]==0 & dw[_n-1]>0 & dw[_n-
1]<=1
assert dw>0 if inc>0
collapse (mean) dw [iw=_prev3] if ~missing(comorbid), by(comorbid_code sex agecat)
reshape wide dw , i(sex age) j(comorbid_code ) string
tempfile data
save `data'

use "simulations", clear
merge sex age using `data', nokeep
drop _*
foreach var of varlist flag* {
	local cause=subinstr("`var'","flag","",1)
	cap sum dw`cause'
	if _rc~=0 {
		di in red "`cause'"
		drop `var'
	}
}

gen total_unadj_dw=0
gen combined_dw=1
foreach var of varlist flag* {
	local cause=subinstr("`var'","flag","",1)
	replace dw`cause'=cond(`var'==0,.,dw`cause')
	replace combined_dw=combined_dw*(1-cond(missing(dw`cause'),0,dw`cause'))
	replace total_unadj_dw=total_unadj_dw+cond(missing(dw`cause'),0,dw`cause')
}
replace combined_dw=1-combined_dw

foreach var of varlist flag* {
	local cause=subinstr("`var'","flag","dw",1)
	gen orig_`cause'=`cause'
	replace `cause'=(`cause'/total_unadj_dw)*combined_dw
}
collapse (mean) dw* (max) orig* [iw=prev], by(sex age) fast
reshape long dw orig_dw, i(sex age) j(cause) string
gen AdjFact=dw/orig
sort cause sex age
tempfile factors
```

```
save `factors'

contract cause
levelsof cause, local(causes)
local N=_N+1
set obs `N'
replace cause="XXXX" if missing(cause)
drop _f
tempfile data1 data2
save `data1'
foreach sex of numlist 1 2 {
       foreach age of numlist 0 1 5(5)100{
              use `data1', clear
              gen sex=`sex'
              gen agecat=`age'
              cap append using `data2'
              save `data2', replace
       }
}
gen nage=age
replace age=75 if age>75
sort cause sex age
merge cause sex age using `factors'
replace age=nage
tab _m
drop _m

xi i.sex
gen PredAdjFact=1
gen lnAdj=logit(Adj) if ~(cause=="K01" & agecat<50)
bysort cause sex (agecat): gen
age_at_onset=cond(_n==_N,102.5,(agecat[_n]+agecat[_n+1])/2)

quietly foreach cause of local causes {
noisily di in green "`cause'"
       cap reg lnAdj age_at _Isex_2 if cause=="`cause'"
       if _rc==0{
              tempvar temp
              predict `temp'
              replace PredAdjFact=invlogit(`temp') if cause=="`cause'"
       }
       else di in red "`cause'"
}
replace AdjFact =. if agecat>75
rename cause comorbid_code
keep comorbid_code sex agecat PredAdjFact
joinby comorbid_code sex agecat using `dw'
gen adjdw= PredAdjFact*dw
keep  bodicode seq sex agecat adjdw
reshape wide adjdw, i(bodicode seq sex) j(agecat)
save "Comorbidity adjustment factors", replace
```

Apply adjustment factors to epidemiological data

```
quietly {
use year bodicode seq sex agecat _inc _prev3 _dur3 _dur1 seqlagflag pop lag
time_symptomatic duration dw comorbid_code /*
       */ using "Epi by bodicode, seq, sex, age 1979 to 2033" if
inlist(year,1993,2003,2013,2023), clear
sort bodicode seq sex
merge bodicode seq sex using "Comorbidity adjustment factors", nokeep
assert _m==3
drop _m
rename bodicode code

rename lag _lag
rename duration _duration
```

```
rename dw _dw

gen lag=cond(seqlagflag==1,_dur1,0)
rename _prev3 prev
rename _dur3 duration
rename _inc inc
bysort year code seq sex (agecat): gen
age_at_onset=cond(_n==_N,102.5,(agecat[_n]+agecat[_n+1])/2)
local DisRate=.03
cap drop low hi dis_dur_* check* yld*
gen low=age_at_onset+lag
gen hi=age_at_onset+duration+lag
gen yld=0
gen lagcur_age=0

foreach cur_agecat of numlist 0 1 5(5)100 135{
       if "`ferest()'"~=""{
              tokenize "`ferest()'"
              gen dur_`cur_agecat'=cond(`cur_agecat'<=hi & `1'>=low,min(`1',hi)-
max(`cur_agecat',low),0)
              replace lagcur_age=cond(`cur_agecat'<=hi &
`1'>=low,max(`cur_agecat',low)-age_at_onset,0)
              replace yld=yld+adjdw`cur_agecat'*time_symptomatic* /*
                     */ (1/`DisRate'*(exp(-`DisRate'*lagcur_age)-exp(-
`DisRate'*(lagcur_age+ dur_`cur_agecat'))))
       }
}
drop dur_*
foreach cur_agecat of numlist 0 1 5(5)100{
       gen pyld_`cur_agecat'=adjdw`cur_agecat'*time_symptomatic*prev*pop if
agecat==`cur_agecat'
       gen prev_`cur_agecat'=pop*prev if agecat==`cur_agecat'
       gen yld_`cur_agecat'=inc*pop*yld if agecat==`cur_agecat'
       gen inc_`cur_agecat'=inc*pop if agecat==`cur_agecat'
       gen pop_`cur_agecat'=pop if agecat==`cur_agecat'
       gen dur_`cur_agecat'=duration if agecat==`cur_agecat'
       gen _dur_`cur_agecat'=_duration if agecat==`cur_agecat'
       gen _yld_`cur_agecat'=inc*pop*_dw*time_sym*(1/`DisRate'*(exp(-
`DisRate'*_lag)-exp(-`DisRate'*(_lag+_duration)))) if agecat==`cur_agecat'
}
collapse (sum) pop_* inc_*  yld_* _yld_* prev_* pyld_* dur_* _dur__*, by(year code
seq sex) fast
reshape long pop_ inc_ yld_ _yld_ prev_ pyld_ dur_ _dur_, i(year code seq sex)
j(agecat)
}
save "Corrected results", replace
```

Appendix G: `hale_decomp` routine

The Stata ado program `hale_decomp` is a 'wrapping' routine that calls a number of Mata subroutines which, in turn, implement the equations set out by Nusselder and Looman.[212] Instructions on how to use `cohort-IPM` are provided in Chapter 4.

```
program define hale_decomp
	version 9.1
	syntax  varlist(min=3 max=3),  Deaths(varname numeric) PYLD(varname numeric)
POP(varname numeric) Strata(varname) base(string)
	confirm new var _Mx _PIx _ex _lx _Lx _PIex _iRyk _iPIxk _iTOTxk _iLWD_MORxk
_iLWD_DISxk _iLWD_TOTxk _iDFLE_TOTxk _iDFLE_DISxk _iDFLE_MORxk
	tokenize `varlist'
	local cause `1'
	local age `2'
	local ax `3'
quietly {
	sort `strata'  `age' `cause'
	local allvars "`strata'  `age' `cause'"
	global allvars : list uniq allvars
	cap by $allvars : assert _N==1
	if _rc~=0 {
		noisily di in red "the variables $allvars do not uniquely identify the
data"
		error 1
	}
	levelsof `strata', local(stratum)
	local baseok : list base in stratum
	if `baseok'~=1 {
		noisily di in red "the strata variable `strata' does not contain the
base value `base'"
		error 1
	}
	tempvar stratavar causevar flag_n0 flag_n1
	cap egen `stratavar'=group(`strata') if `strata'~=`base'
	cap egen `stratavar'=group(`strata') if `strata'~="`base'"
	replace `stratavar'=0 if missing(`stratavar')
	gen `flag_n0'=`stratavar'==0
	gen `flag_n1' = 0
	egen `causevar'=group(`cause')
	preserve
	collapse (sum) `deaths' `pyld' (max) `pop' `ax', by( `strata' `age'
`stratavar' `flag_n0' `flag_n1' ) fast
	gen _Mx=`deaths'/`pop'
	gen _PIx=`pyld'/`pop'
	by `strata' : _lifetable `age' `ax', rate(_Mx) pyld(_PIx)
	quietly sum `stratavar'
	local stratavarmax=r(max)
	forvalues t=1/`stratavarmax'{
		replace `flag_n1' = `stratavar' == `t'
		mata: mata_decomp("iTOTxy_`t'")
	}
	tempfile data
	save `data'
	restore
	merge `strata' `age' using `data'
	egen _iRyk=pc(`deaths'), prop by( `strata' `age')
	gen _iPIxk=`pyld'/`pop'
```

[212] Nusselder, WJ and Looman, CW, "Decomposition of differences in health expectancy by cause".

```
	drop _m
	sort `strata'  `cause' `age'
	gen _iTOTxk=.
	gen _iLWD_MORxk=.
	gen _iLWD_DISxk=.
	quietly sum `causevar'
	local causevarmax=r(max)
	forvalues t=1/`stratavarmax'{
		forvalues k=1/`causevarmax'{
			replace `flag_n1' = `stratavar' == `t' & `causevar'==`k'
			replace `flag_n0' = `stratavar' == 0 & `causevar'==`k'
			mata: mata_cause_decomp("iTOTxy_`t'")

		}
	}
	gen _iLWD_TOTxk=_iLWD_MORxk+_iLWD_DISxk
	gen _iDFLE_TOTxk=_iTOTxk-_iLWD_TOTxk
	gen _iDFLE_DISxk=-_iLWD_DISxk
	gen _iDFLE_MORxk=_iDFLE_TOTxk-_iDFLE_DISxk
	}
end
program define _lifetable, byable(recall, noheader) sortpreserve
	version 9.1
	syntax  varlist(min=1 max=2 numeric) [if],  [Rate(varname numeric)]
[PYLD(varname numeric)]
	tokenize `varlist'
	local age `1'
	local ax `2'
	marksample touse
	cap gen _ex=.
	cap gen _lx=.
	cap gen _Lx=.
	cap gen _PIex=.
	mata: mata_lt_rate()
end
version 9.1
mata:
mata clear
void mata_cause_decomp(string scalar matname)
{
	st_view(_iRyk_n0,.,"_iRyk",st_local("flag_n0"))
	st_view(_iRyk_n1,.,"_iRyk",st_local("flag_n1"))
	st_view(_Mx_n0,.,"_Mx",st_local("flag_n0"))
	st_view(_Mx_n1,.,"_Mx",st_local("flag_n1"))
	st_view(_PIx_n0,.,"_PIx",st_local("flag_n0"))
	st_view(_PIx_n1,.,"_PIx",st_local("flag_n1"))
	st_view(_iPIxk_n0,.,"_iPIxk",st_local("flag_n0"))
	st_view(_iPIxk_n1,.,"_iPIxk",st_local("flag_n1"))
	st_view(_Lx_n0,.,"_Lx",st_local("flag_n0"))
	st_view(_Lx_n1,.,"_Lx",st_local("flag_n1"))
	iCyk=((_iRyk_n1 :* _Mx_n1) :- (_iRyk_n0 :* _Mx_n0)) :/ (_Mx_n1 :- _Mx_n0)
	iTOTxk=colsum(iCyk :* st_matrix(matname))'
	iMORxk=((_PIx_n1 :+ _PIx_n0) :/ 2) :* iTOTxk
	del_iPIxk=_iPIxk_n1 :- _iPIxk_n0
	iDISxk=((_Lx_n1 :+ _Lx_n0) :/ 2) :* del_iPIxk
	st_store(.,"_iTOTxk",st_local("flag_n1"),iTOTxk)
	st_store(.,"_iLWD_MORxk",st_local("flag_n1"),iMORxk)
	st_store(.,"_iLWD_DISxk",st_local("flag_n1"),iDISxk)
}

void mata_decomp(string scalar matname)
{
	st_view(_Lx_n0,.,"_Lx",st_local("flag_n0"))
	st_view(_Lx_n1,.,"_Lx",st_local("flag_n1"))
	st_view(_lx_n0,.,"_lx",st_local("flag_n0"))
	st_view(_lx_n1,.,"_lx",st_local("flag_n1"))
	st_view(_PIx_n0,.,"_PIx",st_local("flag_n0"))
	st_view(_PIx_n1,.,"_PIx",st_local("flag_n1"))
```

```
	rownum=rows(_Lx_n0)

	_del_iLx=_Lx_n1 :- _Lx_n0
	_del_iPIx=_PIx_n1 :- _PIx_n0
	_iMORx=((_PIx_n0 :+ _PIx_n1) :/ 2) :* _del_iLx
	_iDISx=((_Lx_n0 :+ _Lx_n1) :/ 2) :* _del_iPIx

	iIExy=J(rownum,rownum,0)
	iOExy=J(rownum,rownum,0)
	iDExy=diag((_lx_n0 :/ _lx_n0[1]) :* ((_Lx_n1 :/_lx_n1) :- (_Lx_n0
:/_lx_n0)))
	for (y=1;y<rownum;y++) {
		for (x=y+1;x<=rownum;x++) {
			iIExy[y,x]=(_Lx_n0[x] :/_lx_n0[1]) :*((_lx_n0[y] :*
_lx_n1[y+1]) :/(_lx_n0[y+1] :* _lx_n1[y]) :-1)
			iOExy[y,x]=(_Lx_n1[x] :/_lx_n0[1]) :*((_lx_n0[y] :/ _lx_n1[y])
:- (_lx_n0[y+1] :/ _lx_n1[y+1]))
		}
	}
	iIxy=iOExy :- iIExy
	iTOTxy=iDExy :+ iIExy :+ iIxy
	st_matrix(matname,iTOTxy)
}

void mata_lt_rate()
{
	st_view(age,.,st_local("age"),st_local("touse"))
	st_view(M,.,st_local("rate"),st_local("touse"))
	st_view(ax,.,st_local("ax"),st_local("touse"))
	st_view(pyld,.,st_local("pyld"),st_local("touse"))
	n=J(rows(age),1,.)
	T=J(rows(age),1,.)
	l=J(rows(age),1,1)
	for (i=1;i<=rows(age)-1;i++) {
		n[i]=(age[i+1]-age[i])
	}
	q=(n :* M):/(1 :+ (n :* M :*(1 :- ax))) ; q[rows(q)]=1
	d=l :* q
	for (i=2;i<=rows(age);i++) {
		l[i]=l[i-1]-d[i-1]
		d[i]=l[i]*q[i]
	}
	L=((l :- d) :* n) :+ (d :* ax :* n)
	L[rows(L)]=d[rows(d)]/M[rows(M)]
	for (i=1;i<=rows(age);i++) {
		T[i]=sum(L[|i,1\.,1|])
	}
	ex=T :/ l
	st_store(.,"_ex",st_local("touse"),ex)
	st_store(.,"_lx",st_local("touse"),l)
	st_store(.,"_Lx",st_local("touse"),L)
	L=(1 :- pyld) :* L
	for (i=1;i<=rows(age);i++) {
		T[i]=sum(L[|i,1\.,1|])
	}
	ex=T :/ l
	st_store(.,"_PIex",st_local("touse"),ex)
}
end
```