Death registration and mortality trends in Australia 1856–1906

By Michael Willem de Looper

A thesis submitted for the degree of

Doctor of Philosophy of The Australian National University

May 2014

Declaration

This thesis is the original work of the author carried out during Ph.D. candidature in the Demography and Social Research Program of the Australian Demographic and Social Research Institute at the Australian National University.

Michael de Looper

28 May 2014

Acknowledgements

First and foremost, I would like to express my grateful thanks to Heather Booth, Rebecca Kippen, Len Smith and Ching Choi for their guidance throughout the course of this thesis. I appreciate your knowledge and wisdom.

I am deeply indebted to the staff of the following libraries for their assistance in locating sources: the National Library of Australia, the Menzies Library at the Australian National University, the State Library of New South Wales, the Fisher Library at the University of Sydney, the Royal Australian College of General Practitioners library, the Australian Bureau of Statistics library in Canberra and the Bibliothèque nationale de France in Paris.

Thanks to Rasmus Hoffmann and Caspar Looman from Erasmus University, Rotterdam, for their advice on mortality turning points, and for sharing their R program. I also thank Romola Davenport from the Cambridge Group for the History of Population and Social Structure for her insights on the decline of tuberculosis mortality.

Christy, to you goes my love and appreciation for your forbearance and support over these eight years.

S. D. G.

Abstract

Analyses of national mortality data in Australia generally do not examine the period before the formation of the Commonwealth Bureau of Census and Statistics in 1905. Yet detailed information on death and its causes is available from the beginning of civil registration in the colonies in the mid-nineteenth century, and in the case of Tasmania, as far back as 1838. By 1856, all colonies had enacted legislation for the compulsory registration of births, deaths and marriages.

Between 1856 and 1906, more than 1.75 million deaths were registered in Australia. Annual summaries or 'abstracts' of mortality were published by colonial Registrars and Statists, containing information on age, sex and causes of death. Although not without methodological problems, these abstracts can be compiled to allow for the examination of the course of all-cause and cause-specific mortality in Australia, a period of great epidemiological change.

The age-standardised rate of all-cause mortality peaked at around 2,000 per 100,000 population in 1860 — a year of fearsome epidemics. An important turning point occurred in 1885, after which mortality declined steadily and with less annual variation. The death rate fell from 1,600 in 1885 to under 1,000 in 1906, a fall of one-third over two decades. Life expectancy at birth rose from 43 years for males and 46 for females in the 1850s, to 47 and 51 in the 1880s, and 54 and 58 in the 1900s. More than half of the improvement was contributed by the reduction of under-5 mortality. Two key components were declines in infant

deaths from gastrointestinal infections, and in tuberculosis mortality among young adults.

Cause-of-death data allow for the measurement of epidemiological transition—the replacement of infectious diseases by chronic diseases over time as mortality declined. Although there were changes in the contribution of specific causes of death such as gastrointestinal infections and tuberculosis, the relative contribution to total mortality of communicable diseases, non-communicable diseases and injuries remained largely unchanged. To that end, there was little evidence of epidemiological transition during the period.

With scientific medicine largely absent, social and environmental factors—the conditions in which people live, grow, work and age—emerge as important determinants of mortality during this period. Deaths graded by occupation and by a socioeconomic index of area reveal a social gradient. Degradation of the physical environment through urbanisation and public health improvements in the form of clean water and sanitation were key determinants of mortality.

Contents

Chapter 1

Introduction

Historical demography has described many of the striking changes in mortality which began in the latter part of the nineteenth century in the countries of western Europe and in their 'offshoots' in other regions of the world. In countries such as England, France, Germany, Sweden and the United States, the diminishing of the 'crisis mortality' of the eighteenth century—when epidemic diseases caused widespread loss of life with great annual variability in death rates—was followed by a period of stabilisation in the nineteenth. Much of the nineteenth century was characterised by deaths from endemic infectious diseases, and a slowing of gains in life expectancy (Schofield & Reher, 1991). In the last decades of the century however, mortality rates again declined sharply.

In England, the unfolding of the Industrial Revolution during 1830–75 was accompanied by a stagnant death rate before a further decline commenced around 1875 (Szreter, 2004). Life expectancy at birth for males rose from 41.4 years in 1871–80 to 48.5 in 1910–10, and for females from 44.6 to 52.4 (Woods & Hinde, 1987). In France, there was little change in the forty years following 1845, the death rate only resuming its decline towards the end of the 1880s (Vallin, 1991). The crude death rate in Germany declined from 28 per thousand in 1871–75 to 18 in 1911–15, while the expectation of life at birth rose from 37.0 to 49.0 years (Haines & Kintner, 2000). In Sweden, all sexes and age groups

benefited from 'a substantial mortality decline during the second half of the nineteenth century parallel to the rapid industrialisation of the country' (Sundlin & Willner, 2009). In the United States city of Philadelphia life expectancy at birth rose from 39.6 in 1870 to 45.8 in 1900, with the age-standardised death rate over the same period declining from 21 per thousand to 17, a phenomenon repeated in other major urban centres including New York, Chicago and Baltimore (Condran & Cheney, 1982).

Stolnitz (1955) observed that the modern rise in western life chances first became marked late in the century. National increases in life expectancy at birth in the half century since the 1890s had almost doubled the gains of the preceding half century. The initiation of substantial gains in life expectancy before the 1890s occurred among the countries of western Europe and in Canada, the United States, Australia and New Zealand (Riley, 2005).

The course of death rates in these countries suggested that the end of the nineteenth century, and more particularly the 1880s 'appear to mark the beginning of a new mortality regime' where the variability of epidemic disease and crisis mortality was reduced, so that these 'chance factors no longer play as large a part as they did previously' (Vallin, 1991).

This period between 1850 and 1900 has attracted widespread attention from demographers, epidemiologists, economists, historians and other students of social change. Not only did mortality decline to unprecedented low levels in many western countries in the last decades of the Victorian era, but fertility and family size also fell, with improved life chances for young and old, male and female. Leading causes of illness and diseases causing death changed in their frequency and severity, and this change occurred largely in the absence of scientific medicine offering efficacious treatment or cure (Kunitz, 1991).

The transition from high birth and death rates to low birth and death rates as countries developed from pre-industrial to industrialised economic systems has been termed the 'Demographic Transition'. The new science of statistics made possible the measurement and description of these health transitions, with the

underlying belief by social reformers and public health officials that enquiry into these phenomena would lead to a better life and health for all.

Much of the description and theorising about trends in mortality flowed from observations undertaken in Britain and other European countries such as Sweden, France and Germany. The early adoption of civil registration allowed these countries to report on the administrative data resulting from the registration process. England had passed legislation to introduce registration of births, marriages and deaths in 1836, and was well-placed to report on trends in mortality. Statistician and epidemiologist William Farr, who became the first compiler of scientific abstracts in the General Register Office, established a system to record causes of death and used the results as evidence as he sought to improve public health (Eyler, 1979). Farr believed that social change played an important role in determining leading causes of death.

DESCRIBING THE MORTALITY DECLINE: OMRAN'S THEORY OF EPIDEMIOLOGICAL TRANSITION

The rapid decline in death rates in these countries was accompanied by increases in population growth rates and in life expectancy during the second half on the nineteenth century. This was an important phase in a longer-term decline in mortality from high to low levels. The long-term decline in mortality, with a shift in major causes of death away from infectious (communicable) diseases, to chronic and degenerative (non-communicable) diseases, has been termed the 'epidemiological transition'. The epidemiological transition has been described as 'the mortality component of the demographic transition' (Mackenbach, 1994).

A theory of epidemiological transition was first proposed by Abdel R. Omran in 1971 (Omran, 1971; Weisz & Olszynko-Gryn, 2010). Omran argued that changes in mortality affect a broader transition, with important demographic, economic and sociological consequences. He summarised the major precepts of his theory in five propositions:

Proposition 1: *Mortality is a fundamental factor in population dynamics.*

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Proposition 2: *During the transition, a long-term shift occurs in mortality and disease patterns whereby pandemics of infection are gradually displaced by degenerative and man-made diseases as the chief form of morbidity and primary cause of death.* Typically, mortality patterns distinguish three successive stages of the epidemiological transition, (i) The Age of Pestilence and Famine, where high and fluctuating mortality leads to little population growth and low life expectancies of 20 to 40 years, (ii) The Age of Receding Pandemics, where mortality begins to decline as epidemic peaks also decline, leading to an increase in average life expectancy of 30 to 50 years, and (iii) The Age of Degenerative and Man-Made Diseases, where mortality continues to decline and then stabilise at relatively low levels, non-communicable diseases predominate and average life expectancy at birth rises gradually to exceed 50 years.

Proposition 3: *During the epidemiological transition, the most profound changes in health and disease patterns obtain among children and young women.*

Proposition 4: *The shifts in health and disease patterns that characterise the epidemiological transition are closely associated with the demographic and socioeconomic transitions that constitute the modernisation complex.*

Proposition 5: *Peculiar variations in the pattern, the pace, the determinants and the consequences of population change differentiate three basic models of the epidemiological transition: the classical or western model, the accelerated model and the contemporary or delayed model.*

Omran used pre-1971 aggregate mortality data from a number of countries — England and Wales, Chile, Ceylon (now Sri Lanka), Japan and Sweden — to demonstrate that as infections accounted for less of total deaths, degenerative diseases such as cardiovascular disease and cancer increased proportionally.

Several models differentiated the distinctive patterns of the epidemiological transition. The mortality experience of England and Wales and of Sweden illustrated a 'classical' or 'western' model, where the decline in mortality began relatively earlier and the transition took approximately 100 years to complete. In other countries such as Japan, the transition began later but occurred more

rapidly (the 'accelerated' model). In most developing countries, the transition began even later, and by 1971 had not yet been completed (the 'contemporary' or 'delayed' model) (Omran, 1971).

For western model countries, socioeconomic, political and cultural determinants led to rising living standards, better hygiene and improved nutrition, and were important contributing factors in the transition from communicable to non-communicable diseases. These factors are contrasted with medical and public health measures, including improved sanitation, immunisation and therapies for disease, which had a relatively earlier influence in the accelerated and contemporary transitions (Omran, 1971).

Evaluations and critiques of Omran's theory

The adaptability of Omran's theory to encompass mortality change in different countries with varying disease determinants, over broad time periods and under a variety of cultural, sociological and behavioural conditions has been widely debated (e.g. Mackenbach, 1994; Caldwell, 2001; Caselli, Meslé & Vallin, 2002).

A number of commentators took issue with Omran's terminology. The description 'degenerative and man-made disease', used by Omran to name his third stage of transition, is not found in modern medicine, and has been labelled as 'vaguely moralistic' (Mackenbach, 1994). 'Degenerative disease' implies an irreversible and age-related pathology which is not well-suited to current views on the causation of chronic diseases such as cancer and coronary heart disease. The term 'degenerative disease' is more commonly used to refer to musculoskeletal conditions such as osteoarthritis. 'Man-made diseases' went unexplained in Omran's first paper (Caldwell, 2001), although in 1982 he clarified that these included 'radiation injury, mental illness, drug dependency, traffic accidents, occupational hazards' (Omran, 1982). Typifying cardiovascular disease, cancer, diabetes and other non-communicable diseases as man-made implies that infectious diseases—which are often facilitated by poor water quality, lack of sanitation, overcrowding and malnutrition—are not man-made.

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However, communicable diseases can be facilitated by human agency. In this work the term 'non-communicable disease' is preferred.

Omran's 1971 paper theorised that both life expectancy and the proportion of mortality due to 'degenerative and man-made diseases' would continue to increase, but would eventually stabilise. By focussing on total populations and aggregate national data, Omran did not comment on the considerable rises in age-specific mortality from non-communicable diseases such as cardiovascular disease and cancer in developed countries after World War I, including in the United States, the United Kingdom, Sweden, Australia and New Zealand. These rises affected life expectancy to such an extent that it plateaued in many of these countries from the end of World War II to the 1970s (Mirzaei *et al.*, 2009).

At the time of writing, Omran could not have foreseen the large declines in cardiovascular mortality in developed countries which began in the 1970s. These declines resulted largely from changes in population risk factors, and led to renewed rapid increases in life expectancy. Neither could he have foreseen increases in communicable diseases such as HIV-AIDS and the resurgence of tuberculosis and other infections.

Omran relied extensively on aggregate statistics from Sweden, England and Wales to develop his theory. Further analyses of Swedish data indicate that mortality showed considerable variation throughout the country, with a variety of transitions occurring at regional levels (Rogers & Nelson, 1997). Similarly, in Victorian England and Wales, 'where one lived had an especially important bearing not only on when one died, but also on how one died' (Woods, 2000, p.310). In Germany, evidence by region for the period 1860–1935 also indicated wide spatial variations in mortality and differential transitions (Haines & Kintner, 2000).

In many nations, including the United States, a substantial mortality 'penalty' was imposed through living in urban areas in the late nineteenth and early twentieth century (Haines, 2001; Ferrie & Troesken, 2008). The urban mortality transition often differed in its timing and in its rate from non-urban areas, with

rapid urban growth, degradation of water and sanitation and an inadequate understanding of disease processes contributing to a mortality crisis in mid-nineteenth century American cities. Understanding the origin and nature of the mortality decline in different regional areas requires a fuller understanding of small-area socioeconomic and demographic variables. To fully appreciate the mechanisms behind the disappearance of epidemic disease, and the transition from communicable to non-communicable disease, more than aggregate national data are required, since these often 'hide more than they reveal' (Rogers & Nelson, 1997).

Mackenbach (1994) found Omran's concept of epidemiological transition to be ill-defined, and had difficulty in locating both its beginning and its end in time. As a consequence, the theory cannot be put into operation without ambiguity. Insofar as mortality since prehistoric times has been high and fluctuating, Mackenbach suggested locating the beginning of the transition at some point between Omran's first and second stages. For many researchers, the second stage in western model countries began 'about the middle of the 19th century'.

Omran defined the beginning of the transition on the basis of trends in all-cause mortality, but for many countries data to assess trends during the mid-nineteenth century are lacking. Recent research has raised questions about this dating, since in a number of countries all-cause mortality trends indicate that the beginning of the transition should be located much earlier in time (Mackenbach, 1994).

If the timing of the transition is based on changes in cause-of-death patterns, the same problems arise. Data to assess these changes are lacking, since few countries have cause-specific data before the middle of the nineteenth century, and for those that do, the quality of the assigned causes is questionable. This work seeks to extend back in time the trend of all-cause and cause-specific mortality in Australia. Registration of deaths in some Australian colonies predates the 1850s and although reliable cause-specific data are lacking for these early years, insights can be gained into when and why mortality rates began to decline.

The third proposition by Omran—that the epidemiological transition would especially benefit women—was not born out in Swedish statistics, since female mortality was lower than for males from the pre-transition period onward (Rogers and Nelson, 1997). It is not clear why Omran believed that females benefitted 'especially', although in many countries female mortality did fall in reproductive ages as fertility declined (Caldwell, 2001). Men also damaged their health to a greater extent than women through smoking and other risk factor behaviours.

Omran's fifth proposition specified three models of epidemiological transition (Omran, 1971). By 1998, a number of sub-models had been proposed—no less than six in the Americas alone—in an attempt to accommodate the wide variety of country transitions, since 'co-existence of more than one model may be found in some multiracial or multicultural societies' (Omran, 1998). This multiplicity of models served to 'underestimate the flow around the world of ideas, behavioural models, education systems, public health approaches and medical technologies' (Caldwell, 2001). Since the 1960s, numerous countries defied the general trend of increasing life expectancy predicted by Omran, and for various reasons were prevented from completing certain stages of the transition. They did not experience rapid declines in cardiovascular mortality, and were subject to new epidemics such as AIDS or the re-emergence of older infectious diseases. These countries were epidemiological transition theory exceptions (Caselli, Meslé & Vallin, 2002).

Difficulties arise in matching the mortality data of many countries throughout the course of the twentieth-century to Omran's Age of Degenerative and Man-Made Diseases. These difficulties prompted Olshansky & Ault (1986) to propose a fourth stage of the epidemiological transition, based on the mortality experience of the United States between 1900 and 1980 and on projections for that country to the year 2020. This fourth stage—the 'Age of Delayed Degenerative Diseases'—was characterised by rapid mortality declines in older ages which are caused by a postponement of the ages at which 'degenerative diseases' lead to death. This mortality decline would lead to further increases in life expectancy 'into and perhaps beyond eight decades'.

Olshansky & Ault attributed their fourth stage to a combination of factors including a shift in population age structures toward older ages, advances in medical technologies, expanded health care programs for the elderly and reductions in risk factors at the population level. Olshansky and colleagues (1997) also suggested that the resurgence of infectious diseases combined with the emergence of new communicable diseases and antibiotic resistance could again lead to rising mortality—evidence for a fifth stage.

Critical appraisal led Omran (1983, 1998) and others to embark on refined applications and summaries, a preliminary update, and a revisited formulation of the theory based on wider geographic, temporal and conceptual inputs. Omran acknowledged the addition of further stages to his theory. He supported a fourth stage characterised by a rise in life-expectancy to 80–85 years, followed by a period of stabilisation and a decrease of cardiovascular mortality, the emergence of new diseases (HIV, hepatitis B and C, Ebola, Lyme disease) and the resurgence of former diseases (cholera, malaria, dengue, tuberculosis, Chagas disease). Omran also added a fifth stage—the Age of Aspired Quality of Life—with 'paradoxical longevity and persistent inequities'.

The modern utility of Omran's theory

Omran's theory of epidemiological transition, now in its fifth decade, remains a 'potentially powerful framework' within which to study population disease and mortality, and their historical and international variations (Mackenbach, 1994). Despite its shortcomings in encompassing twentieth- and twenty-first century mortality change in all countries, the theory itself has not been called into question (Caselli, Meslé & Vallin, 2002). Its value remains in stimulating inquiry into the 'mortality side of demographic transition' (Caldwell, 2001). Its broad and eclectic nature extends beyond a narrow description of 'changing disease progressing everywhere in a uniform and unilinear manner' (Weisz & Olszynko-Gryn, 2010).

Calls have been made, however, for revision of the theory, to replace the concept of 'epidemiological transition' with a wider concept of 'health transition'. This would place epidemiological characteristics within a wider

health context which would include explanations of how societies respond to changing health situations as a result of cultural, social and behavioural determinants (Frenk *et al.*, 1991; Caselli, Meslé & Vallin, 2002).

A thorough investigation of historical changes in mortality in a broader context is also an aim of this work. Epidemiological transition theory will be used to describe patterns of all-cause and cause-specific mortality in Australia throughout the second half of the nineteenth century. In western model countries, this period is generally understood as occurring during the mid-phase of the transition—the 'Age of Receding Pandemics'—characterised by declines in mortality particularly among children, and life expectancy reaching in excess of 50 years. Changes in mortality and its causes in Australia will be examined across age groups and geographic areas. The social determinants of these changes will be interpreted in their historical context.

UNDERSTANDING THE MORTALITY DECLINE: THE SEARCH FOR DETERMINANTS

The reasons for the late nineteenth-century decline in mortality rates and the changing epidemiology of causes of death form part of a broader inquiry which seeks to understand the causal processes affecting the health of populations. These causal processes can be broadly categorised into two groups: (i) explanations emphasising economic growth and rising standards of living, and (ii) explanations emphasising improvements in personal and public health services and in social development.

The McKeown interpretation

Medical historian Thomas McKeown, in his highly influential publication *The Modern Rise of Population* (1976), argued that population growth in England and Wales was not due to an increase in the birth rate brought about by reduced restraints on fertility, as some had thought, but was instead due to a decline in mortality.

McKeown used William Farr's mortality abstracts to calculate age-standardised death rates for the mid-1850's and for 1901. He noted that the decline in the

death rate between the two periods was largely the result of fewer deaths from airborne or water- and food-borne infectious diseases, such as tuberculosis, typhus, typhoid, scarlet fever, cholera and chickenpox. Especially notable was the apparent decline in tuberculosis mortality. McKeown dismissed a number of possible explanations for the decline in deaths from infectious diseases, such as changes in disease virulence, and effective immunisation or medical therapy. For McKeown, the role of clinical medicine and institutional health care in the decline was minimal during this period (McKeown & Record, 1962).

This left only two possibilities. Firstly, that the mortality decline beginning in the late eighteenth century was due to rising standards of living, with an improvement in nutrition resulting from growth in average income, better agricultural production and a greater food supply, which, he argued, served to improve diet and increase resistance to disease (McKeown & Brown, 1955). Secondly, rising living standards were reinforced by improvements in the environment in the second half of the nineteenth century following the introduction of hygienic measures—water purification, sewage disposal and food hygiene—which led to a substantial reduction in intestinal infections (McKeown, 1976, pp.152–153). Regarding the lesser contribution of non-infectious diseases to the decline, McKeown thought that medical measures, but also contraception and improvements in nutrition had most effect (p.154).

The study of average population height has been used to support McKeown's rising living standards and nutrition interpretation, on the assumption that eating more leads to growing taller and therefore living longer. Modernisation in Australia during the public health movement has also been linked to a modest rise in stature (Steckel, 2008; Jackson & Thomas, 1995; Whitwell *et al.* 1997; Whitwell & Nicholas, 2001). This view has since been tempered by its proponents, leading to the modified understanding that although nutrition is crucial for determining growth in children before the age of five, it is largely irrelevant in explaining adult height outside of the disease environment in which individual growth takes place (Johansson, 1994).

Nutrition proponents have acknowledged that diet and food availability do not offer a monocausal explanation for mortality change, but that a 'synergistic' relationship exists between infection and nutrition. They grant that a wide range of additional factors, including urbanisation, sanitary reform and cohort-specific susceptibilities to disease impacted the pattern of mortality decline in the second half of the nineteenth century (Harris *et al.*, 2010).

McKeown's conclusions about the role of living standards and improved nutrition, along with his methods and reasoning, fuelled widespread debate and attracted criticism. Johansson (1994) charged McKeown with using persuasive rhetoric to subvert the 'surer science' of germ theory and with marginalising the role of the state in mortality decline through public health improvement. The causal relationship between nutrition, disease and premature death was at best tenuous and declines in cause-of-death data, with all their accompanying classification problems, could in no way be used to measure the contribution of better nutrition.

Mackenbach (1996) suggested that the direct contribution of medical care to historical mortality decline was not as negligible as McKeown had believed, and that there was also an indirect contribution, through the transfer of medical knowledge from the medical profession to sanitary reformers and to the public.

Woods (2000) proposed that much more attention be paid to age-specific and place-specific variations in mortality, since these reveal important patterns and trends in the complex disease environment of Victorian England and Wales which McKeown had overlooked. Much of the decline occurred among infants and young children, and McKeown's focus on adult mortality 'diverts interest from the potentially even more critical issue of how infants and young children were cared for' (p.250). For Woods, urban/rural mortality differentials were central to an understanding of the modern mortality transition.

Public health reform

Szreter (1988), in responding to McKeown's interpretation, required that much more evidence be forthcoming to support a population-wide increase in nutrition levels. Szreter also questioned the magnitude of McKeown's stated

decline in respiratory tuberculosis—a key part of the nutrition argument—with much of the apparent decline coinciding with a rise in respiratory diseases such as bronchitis and pneumonia. Szreter believed that the decline in tuberculosis mortality was an artefact, resulting from many deaths which previously would have been classified as due to TB being transferred to other respiratory causes.

Szreter argued that medicine's role in the decline in mortality levels should be reinstated. He championed the public health movement—and the use of epidemiological knowledge by government health officers to introduce locally administered preventive health measures—in combating the increase in disease brought about by rapid economic growth in Victorian Britain (Szreter, 1988, 1997). For Szreter,

> '...[the] preventive and public health branch of medicine deserves to inherit much of the praise for bringing about the modern mortality decline...only the increasing vigilance of public health and local government officials, (over such matters as the quality of foodstuffs and the standard of amenity in working class housing), ensured that increased wages could buy something that was worth having from a health point of view' (Szreter, 1992).

Health services and social factors

Economistic explanations rely on the observation that countries which have undergone, or are undergoing, economic development generally have lower death rates than those countries which have not.

American demographer and sociologist Samuel H. Preston described the relationship between life expectancy and real per capita income for the 1900s, 1930s and 1960s using aggregate cross-sectional country data (Preston, 1975). Persons born in richer countries lived longer on average than those born in poorer countries, but only to a point. At higher levels of GDP the curve illustrating the relationship flattens, with increasing income associated with little change in life expectancy.

Significantly, Preston's curve has shifted upwards during the twentieth century, so that life expectancy has increased in many countries independent of changes in income. Preston claimed that 'income has been a trivial factor in recent

mortality trends', with factors exogenous to a country's current income accounting for 75–90% of the growth in life expectancy, and income growth only 10–25% (Preston, 1975). These exogenous factors, Preston argued, include improved health services and social development—better technology, vaccination, and medical and public health, with improved nutrition and education playing additional, but small roles.

The importance of these exogenous factors may have been underestimated for earlier time periods including the nineteenth century, according to Preston. Reductions in smallpox mortality through vaccination, empirical validation of germ theory, improved antiseptic practices, quarantine, cleaner food and water, better personal sanitation and improved infant feeding were all exogenous factors which diffused throughout European and other nations at very different levels of economic development, and these led to substantial increases in life expectancy (Preston, 1975).

Historical evidence shows that economic growth and industrialisation in nineteenth-century Britain, in other countries of western Europe and in the United States and Australia posed critical challenges and threats to population health and welfare. The negative consequences of rapid economic growth were conceptualised by Szreter (1997, 1999) as a sequential model: the 'four Ds' of disruption, deprivation, disease and death. The emergence of the Industrial Revolution in Victorian Britain was Szreter's case in point. It entailed serious environmental, ideological and political disruption, leading to social inequality and deprivation, especially in urban centres. These in turn led to increased rates of disease and death, and a halting of the upward trend in life expectancy. Rather than ushering in a decline, economic development during this period led to an increase in death rates.

Social determinants of health

More recently, Richard Wilkinson, Michael Marmot and others have hypothesised that social and economic inequalities—determined by factors which occur largely outside of medical and health care systems—explain

differences in population health, and that redressing these social determinants will lead to health improvements among disadvantaged population groups.

The World Health Organization Commission on Social Determinants of Health, chaired by Marmot, concluded that despite significant gains over the past century, large differences in health still exist within populations, and that these differences are closely linked to degrees of social disadvantage. Even in affluent countries, disadvantaged people have shorter life spans and considerably more illness and disability than the well-off (WHO, 2008; Wilkinson & Marmot, 2003).

These differences arise because of the conditions in which people are born, live, grow, work and age, and because of the variety of arrangements in place to deal with illness. The poor health of the poor and the social gradient in health are determined by the unequal distribution of power, income, goods, and services—access to health care, schools, and education, conditions of work and leisure—in homes, communities, towns, or cities. This unequal distribution of health-damaging experiences is the result of 'a toxic combination of poor social policies and programmes, unfair economic arrangements, and bad politics' (WHO, 2008).

Together, the structural determinants and conditions of daily life constitute the social determinants of health and are responsible for a major part of health inequities between and within countries. Societal development in both rich and poor countries can be judged by the quality of population health—how fairly health is distributed across the social spectrum and whether people are protected from disadvantage if ill.

In concluding that illness and death are socially determined, modern thinking on the origins of health has readopted elements of a mid-nineteenth-century perspective. For the public health and social reformers of that time, the only determinants which really mattered were the conditions in which people lived, grew, worked, and—if they did well—aged.

How did the lack of public health services and poor social circumstances affect health 150 years ago? In developing this theme, this work will explore the

relationship between social determinants and mortality in nineteenth-century Australia.

STUDYING NINETEENTH CENTURY AUSTRALIAN MORTALITY

By the mid-1850s, all Australian colonies had established civil registration of births, deaths and marriages. Data from the registration process can be used to explore whether the same processes of change affecting death rates in the Old World were operative in this country. From the start, the abstracts of mortality published by Colonial Registrars and Statists were indeed used by interested parties—medical practitioners, sanitary reformers, government officers of health and others—to investigate patterns of mortality. These investigations generally focused on particular disease outbreaks for local areas or single colonies, for short periods of time, and with contemporary medical interpretations.

More recent studies have examined selected aspects of mortality in nineteenth-century Australia. A brief survey would include:

- Lancaster's articles in the *Medical Journal of Australia* throughout the 1950s and 1960s, which surveyed important causes of death in the last decades of the nineteenth century (Lancaster, 1960; Seneta & Eagleson, 2004)
- Curson (1985a), who considered the impact and effects of six epidemics of infectious disease between 1789 and 1900 on the population of Sydney
- Vamplew (1987), who oversaw the production of a valuable compendium of demographic information, including some important time series of nineteenth-century Australian mortality
- Cumpston's (1989) overview of infectious disease, ill-health and mortality in Australia from European settlement in 1788 to 1928
- Taylor, Lewis & Powles (1998a), who described the decline in all-cause mortality, relying on crude rates for 1788–1859 and indirectly standardised rates for 1860–1900. They noted that annual age-specific death rates were unavailable before 1900, with tabulations calculated by

Lancaster for the period 1889–1900 apparently 'disposed of in the early 1980s' (p.28). A second article (1998b) describes changes in major attributed causes of death for the period after 1907

- Kippen (2002a), who examined cause-specific mortality in Tasmania for the period 1838–1899, using a unique database of individual-level death registrations. This work remains one of the most thorough statistical treatments of the topic

- Lewis's (2003) description of the history of public health in Australia from settlement to the aftermath of the Second World War. The American and British experiences place the Australian effort within a global context

- Smith's (2011) use of individual case histories and archival research to study the impact of illness on colonial Australians, and their efforts to cope with these.

THE NEED FOR A FULLER ASSESSMENT

Despite these and other recent achievements in historical demography in Australia (Day, Kippen & Lucas, 2006), large knowledge gaps remain. Many surveys of nineteenth century Australian mortality are qualitative and do not utilise data as evidence, despite there being data in abundance.

No standardised time series of death rates are available, and since cause-specific death rates are also lacking, little can be said about epidemiological transition in Australia during this period. No attempt has been made to compile colonial mortality data into a single national database, despite these being official statistics and embodying a rich source of information. They have the potential to make a significant contribution to the further development of demographic theory and to our understanding of population change in both historic and contemporary Australia (Zhao, 2010).

The reasons for these omissions are easily understood. Such a compilation requires access to a complete set of colonial reports on vital statistics spanning over half a century. More than 1.75 million deaths were registered in the fifty

years from 1856 to 1906. Data management requires annual summary information on these deaths, with digitisation of deaths in each age group, sex and cause of death, and for each of the six Australian colonies.

This thesis seeks to fill this knowledge gap. It will compile these data and chart all-cause and cause-specific death rates in Australia during the last half of the nineteenth century. Although it will largely be concerned with national rates, this work will build on finer level data from each colony. Trends in mortality rates in each colony, and in Australia, will be interpreted using contemporary and more recent descriptive studies.

The underlying data in this work stem from the administrative reporting of the colonial Registrars-General and Statists, who tabulated deaths registered in their jurisdictions, and published annual 'abstracts of mortality' in Parliamentary Papers or in Government Gazettes. Their analysis and commentary give comprehensive and often minutely-detailed reviews of deaths and causes of death during the period, in the best Victorian fashion.

Several Registrars and Statists — W. H. Archer and H. H. Hayter in Victoria, Chris Rolleston and T.A. Coghlan in New South Wales — made pioneering demographic efforts. Other figures, such as medical practitioner and health officer James Jamieson, and first Director-General of the Australian Department of Health J. H. L. Cumpston, added important findings on infant mortality, infectious diseases and other causes of death.

Small-area data — at a metropolitan and suburban level — provide added insights into trends in death rates. Deaths in the wards, towns, cities and boroughs of Greater Melbourne for instance, were reported on a monthly, quarterly and annual basis. Mortality in these smaller geographic regions is highly illustrative. It was not uncommon for change to first take place — and to take place in the greatest measure — in urban regions. Small-area changes reinforce our understanding that transitions in mortality are not homogeneous, and that national mortality transition is composed of numerous regional and sub-population transitions (Woods, 2000).

The scope of this study is the six jurisdictions of New South Wales, Victoria, Queensland, Western Australia, South Australia and Tasmania, which collectively formed Australia during this period. For convenience, these jurisdictions are here referred to as 'colonies', although they became states of the Commonwealth of Australia following Federation on 1 January 1901.

The commencement of civil registration suggests that the starting point for this study should be the year in which civil registration of death was first operative in all colonies. New South Wales was the final colony to introduce civil registration, and so the first year for which national data can be compiled is 1856. The end-point is 1906. From 1907, the newly-formed Bureau of Census and Statistics began to report on mortality for the entire Commonwealth of Australia, and these data are readily available (CBCS, 1908; AIHW, 2014).

An extra half-century of data will, for the first time, allow for a continuous time-series of national all-cause and cause-specific mortality to span 150 years. Much of the unfolding of the epidemiological transition took place during this period. Few countries have such a lengthy record.

AIMS OF THIS WORK

The aims of this work are to document how civil registration was established in the colonies of Australia, and by using abstracts of death registrations to construct, analyse and report on a national dataset of mortality, classified by 5-year age groups, sex and cause of death, covering the period from 1856 to 1906.

These aims lead to the following three research questions:

(i)   How were deaths registered in nineteenth-century Australia?
(ii)   What mortality patterns and trends were evident in Australia between 1856 and 1906?
(iii)   What factors may have had a bearing on mortality patterns and trends in Australia between 1856 and 1906?

To answer these three questions, chapters of this study will explore:

- The commencement of civil registration of death in the colonies, and the circumstances which led to registration. Who established these systems, and what information did they collect (Chapter 2).

- The completeness and accuracy of registration. How death registrations were reported. What information was, and was not reported. Can this information be used to construct demographic indicators? (Chapter 3).

- The trend in all-cause mortality in the colonies and in Australia. Life expectancy (Chapter 4).

- How causes of death were recorded and classified. The shortcomings of nineteenth-century categorisations of death (Chapter 5).

- The trends in major causes of death in the colonies and in Australia (Chapter 6).

- How mortality compared at the start and end of the time period. Changes in infant and under-five mortality, in cause-specific mortality, and in leading causes of death, including tuberculosis. How these findings inform our understanding of the epidemiological transition in Australia (Chapter 7).

- Whether certain social and environmental determinants of health—urbanisation, water and sanitation, illegitimacy, occupation and socioeconomic position—had an effect on nineteenth-century mortality in Australia (Chapter 8).

Chapter 2

Registering deaths

The statistical recording of death and its causes is a relatively recent European phenomenon. The first Australians, the Aboriginal and Torres Strait Islander people, kept no written records although their art forms—rock and bark painting, engraving, carving and weaving—give descriptive information about their social activities, myths and religion. Instead, Aboriginal oral histories transmitted information about ancestral births, unions and deaths.

The deaths of Aboriginal people in the millennia before the arrival of European settlers are by definition countless, but they almost certainly outnumber the total recorded deaths in Australia (Smith *et al.*, 2008). Aboriginal births, deaths and marriages were increasingly recorded in the same fashion as for the European population throughout the nineteenth century—particularly if families were in regular contact with church or government officials. Nonetheless, oral histories remain an important source of information about the lives and deaths of Aboriginal peoples during the colonial era.

The first non-Aboriginal deaths recorded in Australia occurred among passengers and crew of Dutch East India Company vessels and other early voyages of discovery. In March 1606, several of Willem Janz's crew were killed by Aborigines at Batavia River on Cape York. In 1622, the *Tryall* foundered on the west coast at what became known as Tryall Rocks—the first known European wreck on the Australian coastline. Although 46 people including

English captain John Brookes managed to flee in two small vessels, a further 93 perished at the wreck site. The following year, 1623, Claes Hermanszoon, landed the *Leijden* south of Dirck Hartog Island while charting a section of the Western Australian coast, and Australia's first European birth occurred (Pearson, 2005, p.35).

The vessel *Batavia* was wrecked on Morning Reef in the Houtman Abrolhos archipelago in 1629. Over 200 people survived the initial wreck, but their numbers were more than halved as a result of a mutiny by members of the crew. We know the names of many of these unfortunates, and also their cause of death—those who did not drown died from assault. For almost a century-and-a-half, the *Batavia* graves were the only evidence of European deaths on Australian soil.

During the charting of the continent's east coast in 1770, one of Lieutenant James Cook's seamen died of tuberculosis. Cook wrote in his private log,

> 'Tuesday, May 1st. (1770)...Last night departed this life Forby Sutherland, Seaman, who died of a consumption and in the a.m. his body was entard ashore at the watering place. This circumstance occasioned my calling the south point of this bay Sutherland's Point' (Cleland, 1968).

Another early death was Pere Louis Receveur, a priest-chaplain on La Pérouse's expedition of discovery, map-making and scientific investigation. Receveur had sustained an injury in the Samoan Islands which in the log was described as a 'violent contusion of the eye'. He did not recover from his wound, and was buried at Botany Bay on 17 February 1788, close to the grave of Forby Sutherland.

This event coincided with the arrival of the First Fleet, the eleven ships which sailed from Great Britain to establish a penal settlement in New South Wales. Systematic recording of vital statistics for the new colony began soon after. Reporting the state of the colony to the Colonial Office in England was an annual task for administrations prior to self-government. Before his departure, Arthur Phillip, the first Governor, had received his *Instructions* (composed by Lord Sydney) from King George III, 'with the advice of his Privy Council'. The

original document is now lost, but a manuscript draft in the National Archives in London includes the following passage:

> 'And you are to take especial care that a Table of Marriages as established by the Canons of the Church of England be hung up in every Orthodox Church or Chapel and be duly observed, and also to transmit…an account of such Deaths, Marriages, and Births as may take place within your said Government, by every opportunity which offers to Our Secretary of state for Plantation Affairs' (C.O. 201/1 ff 29–45v).

This passage is struck through in the manuscript draft. It forms part of an amendment which deletes two pages referring to the implementation, in Phillip's administration, of tenets and practices of the Church of England.

Although it is uncertain whether the passage was included in the instructions as received by Phillip, returns of deaths were duly compiled and sent to the Home Office in London. In Phillip's despatch of 16 November 1788 to Under Secretary Nepean, he includes an enclosure compiled by Surgeon John White, which lists 27 male convicts, 13 female convicts, 9 children of convicts, 4 marines and 2 children of marines as having died since landing at Port Jackson on 26 January 1788 (HRA Series I, Vol. 1, pp.103–4).

Phillip's successor, Lieutenant-Governor Grose, was informed in November 1793 that 'It is highly necessary that a yearly return should be made and signed by the Governor of the settlement, or the person administering the government thereof, of all births and deaths within the settlement' (HRA Series I, Vol. 1, p.456). This was also requested of Lieutenant-Governor Collins in February 1803, upon the establishment of the new settlement at Port Phillip (HRA Series I, Vol. 4, p.13).

In practice the returns that were made recorded only some of the births and deaths in the colony. In 1801, William Balmain, the surgeon responsible for the returns commented that

> 'The state of births and deaths in this report is accurate as far as comes within our knowledge, but people die and children are born without our being made acquainted therewith' (HRA Series I, Vol. 3, p.55).

Occasionally, additional detail on the cause of death was added to the simple monthly tallies of deaths. The return of deaths for the year 1803, compiled at the General Hospital by Principal Surgeon Thomas Jamison, noted that

> 'This return stated the names of the mothers and the dates of birth of twenty-seven male and eighteen female children. It also gave the particulars of forty-eight deaths from causes as follow: Consumption, two males, two females; Dysentery, 25 males, two females; Debility, one male, one female; Scurvy, 3 males; Dropsy, 2 males; Hydrothorax, one male; Venereal, 1 male; Gangrene, 1 male; Accident, 2 males, 2 children; Not diagnosed, 1 male, 2 females' (HRA Series I, Vol. 4, p.517).

Early in his Governorship, Lachlan Macquarie directed that chaplains Samuel Marsden, William Cowper and Robert Cartwright be responsible for keeping regular accounts of births and deaths in all districts and parishes throughout the colony. If no chaplains were available, magistrates or commandants would undertake the task. Quarterly returns were to be transmitted to the Principal Chaplain, Marsden, who was to compile a general return to be laid before the Governor (*SG*, 22 Sep. 1810).

In the records for St Philip's, Australia's first parish, entries on burials after 1811 include the first and surname of the deceased, the date of burial, age of deceased, often the date of death (1814–1826 and 1830–1848), quality or occupation, status (convict, free, soldier, sailor) and residence. Sometimes, manner of death was also recorded, especially if violent or unusual, for example, 'speared by natives', 'struck by lightning', 'drowned' (Curson, 1978).

The chaplains duly applied to Macquarie for the right to charge fees for conducting marriages, christenings and funerals, and Macquarie agreed to this (SG, 22 Dec. 1810). Although the returns to the Home Secretary remained a civil responsibility, ecclesiastical records were now to be used as the information source. This would create its own difficulties, not least of which being that these were not records of births and deaths, but of baptisms and burials.

Macquarie wrote to Lord Liverpool in November 1812,

> 'The Deaths Can only be Collected from the Reports of the Chaplains, and until After My Arrival Many of the Interments took place Without the Attendance of the Chaplains, or even the Decency of Carrying the Bodies to the regular prescribed

Burial Grounds Which were at the time of My Arrival, only three in Number, vizt. Sydney, Parramatta and Windsor; the Consequence is that No Registration of these Events Could possibly take place, and even now the Returns made by the Chaplains are not Sufficient to Account for All the Deaths, Neither Can their Reports be looked upon as Correct in the Cases of Convicts, as they Can only Acquire that Information on Hear-Say from some of the persons attending the Funerals' (HRA Series I, Vol. 7, pp.616).

The arrangement still only allowed for partial coverage, and problems grew as the colony expanded. In 1821 Macquarie directed that Sydney be divided into two parishes, with the hope that each clergyman would be better able to attend to their duties, including 'a more correct Register of Baptisms and Funerals, than is at present the case' (*SG*, 18 Aug. 1821).

Adherents to denominations or religions other than the Church of England were poorly served. Although the presence of Catholicism in Australia began with the arrival of the First Fleet, priests were not allowed to travel to the Colony until 1820. The absence of clergy in the colony before this date meant that Roman Catholics baptisms and funerals were often omitted from returns. A large proportion of vital events of non-conformists, however, were included in Anglican registers prior to 1820 (Curson, 1978). From 1821, Catholic priests, as well as Wesleyan Missionaries were required to send quarterly returns of baptisms, marriages and burials in their parishes or districts to the Colonial Secretary.

Around this time, annual statistical returns from the Australian colonies to the Colonial Office in London began to be known as the *Blue Books*. So called because of the colour of the report cover, these compilations became the medium firstly for the transferral of financial and expenditure information, and later for other detail, including educational, economic, agricultural and demographic statistics. New South Wales and Van Diemen's Land produced their first *Blue Books* in 1822, Western Australia in 1834 and South Australia in 1840. Following separation from New South Wales, Victoria began reporting in 1851. Although some of the colonies kept the title, the *Blue Books* were to be

transformed into what became the *Statistical Registers* in the second half on the nineteenth century (Forster & Hazlehurst, 1988).

Throughout the 1820s and 1830s, legislation was passed to increase the scope and better regulate the recording of vital events. Under Governor Thomas Brisbane, an 1825 Act (6 George IV No. 21) requested every clergyman in New South Wales and Van Diemen's Land to keep a register in which each baptism, marriage and burial was to be entered, according to a schedule (*SG*, 3 Nov. 1825). Entries for burials were to include the date of the event, the name and age of the deceased, ship's name if relevant, abode, quality, trade or profession, and by whom the ceremony was performed. Exact copies of each entry for the preceding year were to be sent annually to the Registrar of the Archdeacon's Court. These parish registers were also to be made available for searches by the public.

In New South Wales, an 1834 Marriage Act (5 William IV No. 2) recognised all marriages performed before the passing of the Act that were solemnised by ordained ministers of the Church of Scotland and priests of the Roman Catholic Church. Further Acts in 1839 and 1840 (3 Vic No. 7, 3 Vic No. 23 and 4 Vic No. 14) recognised rites conducted by the Wesleyan Methodist Society, and the Congregational or Independent and Baptist denominations. Certificates of baptisms and burials within these denominations were henceforth to be transmitted to the Registrar of the Supreme Court, but clergymen of the Church of England were to send copies of their register books to the new Bishop of Australia's registrar (*NSWGG*, 10 Oct. 1840).

As in England, systems of recording births, deaths and marriages in the Australian colonies were undergoing refinement, but they were still deficient. The exposure of the shortcomings of the parochial method would be the means for ushering in a new method of registering births, deaths and marriages—civil registration.

CIVIL REGISTRATION IN ENGLAND

Parish registers had been the source for records of ceremonies within English churches from the time of Henry VIII. They were of vital importance for tracing family history, and served to answer questions relating to descent, alliance or birth. The registers also provided a legal recourse for disputes regarding inheritances, or for placing children in professions or trades.

The registers had important secondary uses. The information they contained could be utilised for statistical purposes, such as calculating rates of birth and death. An early pioneer was Thomas Short, a Scottish doctor based in Sheffield, and author of *New Observations…on City, Town and Country Bills of Mortality* (1750) and *A Comparative History of the Increase and Decrease of Mankind* (1767). More influential was Thomas Malthus, who wrote his *Essay on the Principle of Population* (1798) at a time when it was evident that baptisms were far exceeding burials in parishes throughout the land.

In 1812, 'Sir George Rose's Act' (52 Geo. III, c. 146) sought to better regulate the use of parish registers. It enacted that registers of baptisms, marriages and burials according to the rites of the Established Church be kept by officiating ministers, and that annual copies of these be made for transferral to diocesan registrars (Hammick, 1875).

The new science of statistical inquiry, which burgeoned in Britain from the 1830s, made extensive use of the records. This was largely for utilitarian purposes such as actuarial calculations of the value of insurance premiums, but also for the more general 'arts of civil life', and for enquiries into the nature of English society (Glass, 1973). Studies of the living conditions of the urban poor in England were a popular topic in the early nineteenth century (Eyler, 1979). The increased use of records of baptisms and burials for these purposes also drew attention to their shortcomings.

The ecclesiastical nature of the record-keeping had serious drawbacks. They recorded baptisms and burials, thus providing no direct legal proof of either birth or death. Dissenters from the Church of England and other nonconformists were often excluded, since their conviction was that only

persons attaining the age of 16, or professing faith in Christ could be baptised. Indeed, two-thirds of the entire population of Wales, and 4,000,000 people throughout Britain (one-third of the entire population) were Dissenters (*Han.* vol.16, 1833, cc.1218). Although many Dissenting churches kept their own registers, these had no legal standing in the English judicial system, and so despite their convictions, some Dissenters baptised their infants in order to obtain the benefits of registration for their children. Many of the poor were also excluded as fees were payable on registering a baptism, with parochial registration being used by the Church as a source of revenue.

Parish record keeping was sometimes casual, and not always carried out with due diligence. No central repository for the registers existed, and parish registration remained a local affair. The registers themselves were sometimes lost, stolen or damaged (Eyler, 1979, p.37–38).

The inadequacies of the system called for investigation and remedy. In March 1833, a Select Committee of the House of Commons recommended that a national civil registration be established. Bills brought in by Lord Nugent and William Brougham proposed that parish registrars or tax collectors undertake the task, but both were deemed unsuitable. The Act of 4 & 5 Will. IV amended laws relating to the poor, created a set of officers, and divided the country into registration districts, thus providing the means of carrying a new and complete system of civil registration into effect (Hammick, 1875).

During the administration of Lord Melbourne, a bill was introduced by Lord John Russell, the Secretary of State for the Home Department for 'registering births, deaths and marriages in England'. Russell, on introducing the bill stated it to be

> 'a most important subject - important for the security of property - important to ascertain the state and condition of individuals under various circumstances - important to enable the Government to acquire a general knowledge of the state of the population of the country – that there should be a general registration of births, marriages and deaths' (*Han.* vol.31, 1836, cc.367–85).

The bill proceeded with little hindrance and Parliament passed *An Act for Registering Births, Deaths and Marriages in England* (6 Will. IV No. 56) into law on 17 August 1836.

The Registration Act created a General Register Office at Somerset House in London, and a Registrar-General. Every district had a superintendant registrar as well as a registry office; districts were divided into sub-districts, each of which had a registrar of births and deaths. Thomas Henry Lister, novelist and brother-in-law of Lord John Russell, was appointed as the first Registrar-General. The new system took effect on 1 July 1837, and initially covered England and Wales. It was extended to Scotland in 1854 and Ireland in 1863.

CIVIL REGISTRATION IN THE AUSTRALIAN COLONIES

Before self-government, the Australian colonies were closely beholden to England in matters legal and judicial. The legislation leading to civil registration was no different. Within twenty years of the passing of Britain's 1836 Registration Bill, each of the colonies had introduced similar legislation for the registration births, deaths and marriages within their jurisdictions (Table 2.1).

Table 2.1: Legislation for the civil registration of births, deaths and marriages in the Australian colonies, 1838–1855

	Tas	WA	SA	Vic	NSW	Qld
Founding year of colony	1825	1829	1836	1851	1788	1859
Name of Act	2 Vic 8	4 & 5 Vic 9	5 Vic 13	16 Vic 26	19 Vic 34	As NSW
Date passed	7 Aug 1838	27 May 1841	22 Mar 1842	11 Jan 1853	23 Nov 1855	As NSW
Date commenced	1 Dec 1838	1 Sep 1841	1 Jun 1842	31 Mar 1853	1 Mar 1856	As NSW
First year of responsible government	1855	1890	1856	1855	1856	1859

Tasmania

It was not New South Wales, the first colony, which led the way but Tasmania, which was then still known as Van Diemen's Land. Founded as a penal colony in 1803 dependent on New South Wales, it became independent in 1825 and gained its own Legislative Council. Council members were appointed and not elected, and played only a limited role in advising the ruling Lieutenant-Governor. In 1856 the colony was granted responsible self-government with its own representative parliament, and the name of the island and colony was changed to Tasmania.

The population of Van Diemen's Land had grown to around 44,000 persons by the end of the 1830s, 40 per cent of whom were convicts under sentence, with the remaining 60 per cent enjoying freedom as either ex-convicts, military or free settlers (Newman, 2005).

Lieutenant-Governor Sir John Franklin, who arrived in Hobart in January 1837, had observed the passing of the English Registration Act with interest (Kippen, 2002b). At his first session presiding over the Legislative Council in July 1837, he informed members that 'I have, also, directed to be prepared…in imitation of the recent English law on that subject, An act for the Registration of Births, Marriages, and Deaths' (*HTC*, 14 Jul. 1837). The bill was tabled on 30 June 1838, with Franklin reiterating the need for a civil system to register persons of all religious denominations, provide security of property and title, and to use the results for statistical analyses,

> 'The Registration Bill…is in itself one of extreme importance; and I am sure it will be gratifying to you to introduce, at this early period in the history of the Colony, an Act, the advantages of which will be participated in by all denominations of Christians; and which is calculated, in so important a degree, to confirm to the right heirs in future generations, those estates which now, through the industry and enterprise of their present possessors, are daily increasing in fertility and value; and also to prevent the occurrence of those disputes, and that litigation, which have so often terminated in the disappointment of apparently well founded hopes, and the destruction of many a fair inheritance. Neither are advantages such as these, important as they are, the only beneficial consequences which may be expected from the Act; some idea of its probable value to posterity, in other

respects, may be formed, when it is remembered how much interesting information has been lost, from the formerly imperfect registration prevailing in Great Britain' (*HTG*, 3 Jul. 1838).

The bill was referred to committee several times for revision, and went through three readings, but *An Act for Registering Births, Deaths and Marriages in the Island of Van Diemen's Land and its Dependencies* (2 Vic. No. 8) passed by nine votes to one early in August 1838 (TasLC, 7 Aug. 1838).

In his dispatch of 27 August 1838 to Lord Glenelg, Secretary of State for the Colonies, Franklin noted that the Act was

> '…founded upon the Act which was recently passed in England for the same purpose, and differs from it only in some points of detail introduced for the purpose of accommodating its provisions to the limited extent of the population, and to the Ecclesiastical constitution of the Colony' (C.O. 280/96, AJCP 477).

Tasmania thus became the first British colony to introduce civil registration of births, deaths and marriages. Steps were immediately taken to put a workable system in place. A registrar, Charles Bethel Lyons, was appointed (see Appendix 2), and the colony was divided into registration districts, each with a Deputy Registrar. The first births, deaths and marriages were registered in November 1838.

An 1842 amendment (6 Vic. No. 12) introduced fees for each entry of births and deaths made in the registers. These fees were payable to Deputy Registrars as an inducement towards more 'zealous performance of their duties' (Kippen, 2002b). The effect was to increase the numbers of births and deaths registered in 1843, assisting with the establishment of the fledgling system.

In 1895, *An Act to consolidate and amend the law relating to the registration of births and deaths in Tasmania* (59 Vic. No. 9) introduced new clauses and amendments, most notably the requirement that deaths be medically certified from 1 January 1896, the practice previously being carried out on a voluntary basis.

Western Australia

Western Australia closely followed Van Diemen's Land in adopting civil registration. The colony at Swan River had been founded in 1829 by Captain James Stirling. A decade or so later, in 1841, its population had reached a bare 3,000 people, ruled by Governor and Commander-In-Chief John Hutt, who had succeeded Stirling at the beginning of 1839. Hutt presided over a Legislative Council consisting of self-appointed officials and non-officials.
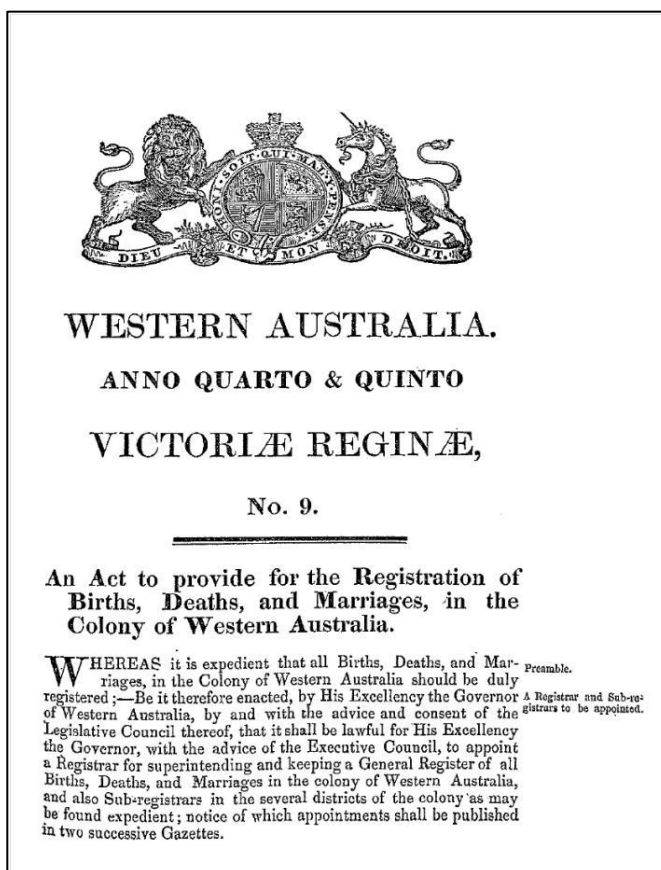

Figure 2.1: Title page of the 1841 Act to register births, deaths and marriages in Western Australia

On 1 April 1841, Governor Hutt moved for the first reading of a *Bill for the Registration of Births, Deaths and Marriages*. After consideration in committee, the Bill was read a third time, and *An Act to provide for the Registration of Births, Deaths and Marriages, in the Colony of Western Australia* (4 & 5 Vic. No. 9) was passed by the Legislative Council's eight members (WALC, 27 May 1841). The Act became operative on 1 September, Governor Hutt noting,

'The business of the Session being concluded, I have now only to thank you for the care and attention which you have bestowed upon the many and important subjects that have been brought before your notice…In saying this, I might name particularly the Act for the Registration of Births Deaths and Marriages, which, whilst it is of a nature to afford valuable statistical details to the Government, provides at the same time an incontestable record, which will be found hereafter of vital consequence as regards the security of property to each Individual in the community' (WALC, 30 Sep. 1841).

An 1847 amendment added further information to be collected following a death. Another amendment in 1856 consolidated the Act's functioning by revising fees and introducing new schedules. Medical certification of death became compulsory with the 1879 *Registration Ordinance Amendment Act* (43 Vic. No. 15).

The widespread development of the colony which followed the discovery of gold in the 1890s necessitated the fixing of registry districts and the appointing of District Registrars. The *Registration of Births, Deaths and Marriages Act* of 1894 (58 Vic. No. 16) established 40 districts, with District Registrars located in each of the major towns.

South Australia

Although Tasmania was the first colony to enact civil registration of deaths, it was not the first to propose legislation—that fell to the founders of the colony of South Australia, before the first colonists had arrived.

London lawyer James Hurtle Fisher had been drawn into the colonising movement, being appointed to the office of Resident Commissioner for the free colony of South Australia in July 1836. The Colonizing Commission gave Fisher the power to dispose of public lands in the new province, with the monies from land sales to be used to promote and finance immigration. The position was second in power only to the Governor (Pike, 1967).

Fisher was asked by the Chair of Commissioners, Colonel Robert Torrens, to 'prepare drafts of Colonial Acts for establishing a Registration of Births, Deaths

and Marriages, and for facilitating the transfer of real property' (Pike, 1966). In 1836, the year of the colony's foundation, the commissioners published *A Sketch of Three Colonial Acts, Suggested for Adoption in the New Province of South Australia.*

Fisher's draft Act was similar in form to the British Act which would pass in that same year, 1836. It had an identical schedule of information to be collected about the deceased person. The sentiments of the English Act were also closely echoed,

> 'The Registry of Births, Deaths and Marriages will afford a faithful and secure record of those events among all classes and sects, and will obviate many objections and difficulties which would otherwise prevail, and by furnishing identity of the parties named will tend to secure that evidence of pedigree which is so essential to support the title to property, the absence of which evidence leads to so much litigation and expense which will thus be avoided' (Fisher, 1836).

Fisher and his family departed England in July 1836, along with Governor John Hindmarsh's party, and arrived in South Australia in late December. Despite the plan being announced as 'already in part approved of by the governor' (SAR, 18 Jun. 1836), Fisher's proposed Acts were not passed. He had argued bitterly with Hindmarsh, and Chief Justice John Jeffcott was also opposed to the adoption of Fisher's Acts. Fisher had sought appointment for life as registrar at an exorbitant salary, and it was feared that he would gain too intimate a knowledge of settlers' private affairs (SAR, 22 Sep. 1838). Although Fisher ceased to operate as resident commissioner with the arrival of Governor George Gawler in October 1838, he was later to become first mayor of Adelaide (Pike, 1966).

Political and religious factionalism and legislative neglect in the colonies' early years led to financial hardship and irregularities in government spending (Pike, 1967). The press agitated for the passing of registration laws, to avoid 'the evils that have now occurred' in records of land sales and of births, deaths and marriages (SAR, 14 Sep. 1839, 19 Jun. 1841).

New Governor George Grey arrived in May 1841. In a July despatch on expenses to Secretary of State for the Colonies Lord John Russell, Grey advised

that no Registration Act existed in the colony, but it was his intention 'as speedily as possible, to carry a Registration Bill through the Legislative Council' (C.O. 13/20, AJCP 584). In September, Grey gave notice that he would introduce a Bill to provide for the Registration of Births, Marriages and Deaths (SAGG, 23 Sep. 1841). Discussions had not concluded before the parliamentary session ended in December, but following a third reading *An Act for Registering Births, Deaths, and Marriages in the Province of South Australia* (5 Vic. No. 13) was passed on 22 March 1842 (*SAR*, 26 Mar. 1842).

Grey's dispatch of 16 April 1842 to new Secretary of State Lord Stanley reassured Her Majesty's Government that

> 'This Act contains provisions similar to those which occur in other Colonial Acts of the like character, and I have no intention of incurring any expenditure for carrying this Act into operation, greater than the amount of Fees which will be levied under the authority of the Act itself' (C.O. 13/25, AJCP 588).

Explorer and soldier Charles Sturt—who much to Grey's chagrin had offered himself for vice-regal office after Governor Gawler's recall—was given the inferior post of Registrar-General, becoming the first to administer civil registration in the colony.

The Act was repealed in 1856 by 19 Vic. No. 3, *An Act to amend the law relating to Registering of Births, Deaths, and Marriages in the Province of South Australia*, which required attending physicians to supply a medical certificate of death. This provision for medical certification predated Britain (Holton, 1983). Annual tables of vital statistics for South Australia were first published by Registrar-General Robert Richard Torrens in this year. Further amendments to the Act took place in 1874 (37 & 38 Vic. No. 10) and in 1900 (63 & 64 Vic. No 744).

During this time, South Australia included the area which was later to become the Northern Territory. The Northern Territory was not separated from South Australia until 1 January 1911.

Victoria

In 1851, the British Government separated the region of Port Phillip from New South Wales, proclaiming the new Colony of Victoria. Gold was discovered near Ballarat in the same year, and then also at Bendigo and many other sites, triggering a gold rush and a massive population influx. In the ten years from 1851 to 1861, the population of Victoria increased sevenfold from 76,000 to 540,000. Victoria operated under a limited form of representative self-government through a Legislative Council consisting of thirty Members, with twenty elected and the remainder appointed by the Lieutenant-Governor.

With the increases in population and in wealth, the new colony soon recognised the need for civil registration and 'the early adoption of a more general and effective system' (*Argus*, 17 Jan. 1851). In June 1852, Lieutenant-Governor Charles La Trobe addressed his Legislative Council, proposing to submit an Act for the Registration of Births, Deaths and Marriages and a General Marriage Act (*Argus*, 23 Jun. 1852).

Bills were introduced late in October, and steps were also taken to 'establish a General Registry for the Registration of Births, Deaths, and Marriages, and of Deeds, and for the compilation of general Statistics' (*Argus*, 3 Nov. 1852). On moving the second reading of the Registration Bill, Attorney-General William Stawell

> '…briefly explained its provisions, stating that it has been founded on the English Registration Act…The necessity for this measure was great, as was evidenced by the fact that some ministers of religion had found it advisable to keep private registers, owing to the absence of any public provision for that purpose' (*Argus*, 11 Nov. 1852).

Some time was spent considering the Bill in committee, but *An Act for Registering Births, Deaths, and Marriages in the Colony of Victoria* (16 Vic. No. 26) was passed following its third reading on 11 January 1853. It received Royal assent a week later, and was to commence on 31 March.

An editorial in the *Argus*, with one eye on eternal destiny and the other on earthly riches, opined that

'…it is of very considerable importance to the community at large, touching, as it does, every human being that may hereafter see the light of heaven for the first or last time, or shall take for better or worse a partner for life in this our golden land of Victoria…there is perhaps no other country in the world where it is so imperative; to have a complete registry system, seeing that a very large proportion of our population consists of immigrants who have accumulated property…and with whom questions of identity must frequently arise, when they shall have gone to their last home' (*Argus*, 14 May 1853).

Figure 2.2: A specimen entry from a Victorian deaths register

Figure 2.3: Extract from the 1866 Victorian abstracts of mortality

A Registrar-General's Department was established under the provisions of the Act, and a Chief Registrar appointed who reported to the Colonial Secretary, and who had responsibility for Deputy-Registrars. The first registrar was William Henry Archer; appointed on a temporary basis, he was quickly succeeded by E. S. Norman Campbell in 1854. Archer—along with Government Statistician Henry Heylyn Hayter—would soon loom large in demographic statistics and record-keeping in nineteenth-century Australia, and he returned to serve as Registrar-General in Victoria from 1859 to 1874.

A medical certificate was required to be produced at the time of registration from 1865 (28 Vic. No. 246, *Registration of Births, Deaths and Marriages Statute*) (*VicGG*, 12 May 1865). Prior Acts were repealed and new legislation with new schedules was introduced in 1890 (54 Vic. 1137, *Registration of Births, Deaths and Marriages Act*).

New South Wales

New South Wales was comparatively late in introducing civil registration, but not through want of trying. The legislation of the late 1830s had extended ecclesiastical registration beyond the Church of England to a number of other denominations, although coverage was still far from complete.

An office of Registrar-General was created by the *Deed Registration Act* of 1843, with the officer responsible for the registration of wills and deeds, the conveyance of property, and registration 'of certain marriages, births, baptisms and burials'. This office was abolished in December 1849, only to be revived under the provisions of the 1855 Registration Act.

Christopher Rolleston, who was the first Registrar-General appointed following the passing of that Act, summarised the state of ecclesiastical registration,

> 'Perhaps not less than one-fourth of the births and deaths which have occurred in the colony are unrecorded, and in the absence of civil and compulsory registration, would continue to be so, owing to the universal apathy which prevails on the subject, and to the absence of these facilities which the late enactment is designed to supply' (Rolleston, 1858b).

As early as 1844 Charles Cowper, member for the County of Cumberland and later five times Premier of New South Wales, introduced a Registration Bill, 'in conformity with the practice pursued in the Mother Country and Van Diemen's Land' (*SMH*, 8 Aug. 1844)—to which he might also have added Western Australia and South Australia. Foreseeing the administrative challenges that any Registration Act would bring, Cowper moved that the Bill be referred to a Select Committee. In bringing up its report, the Committee agreed that

> '…those Bills cannot, either in their present shape, or with any amendments which they could suggest, be passed into laws likely to act beneficially or to give satisfaction. The Acts passed in Van Diemen's Land, from which these Bills have been prepared, require a machinery which would necessarily create a large expenditure, without any corresponding advantage' (*SMH*, 7 Oct. 1844).

The scene was set for more than a decade of legislative failures to introduce civil registration.

The *Sydney Morning Herald* reviewed the situation in the colony in 1845, noting that the registrations then being carried out by the Registrar-General 'apply to little more than two-fifths of the population; and even of those two-fifths his records are confessedly most incomplete'. The editorialist's solution was simplistic in that it demanded better performance from the existing ecclesiastical registration system (*SMH*, 17 & 20 May 1845). In response, 'Ex Officio' argued that

> 'the desideratum at present is 'good registration,' not of *baptisms, burials* and marriages, but of *births, deaths* and marriages…the benefits to be secured by registration are essentially civil, and not ecclesiastical…the registration of baptisms and burials is not all that is required, because (to recapitulate) many are born who are never baptised, and some die who are never buried by human hands' (*SMH*, 22 May 1845).

There were two camps. A June 1848 letter from the Registrar-General to the Colonial Secretary again argued for improved performance from the current system (*SMH*, 24 Feb. 1849) and a bill to that effect was introduced in 1849, only to be later withdrawn by the Colonial Secretary (*SMH*, 4 Oct. 1849).

Another attempt late in 1851 lapsed in the House, with Cowper suggesting to the Government that perhaps it might be time to re-establish the office of Registrar-General, whose department had been abolished two years before (*SMH*, 5 & 13 Dec. 1851). What was essentially the same Bill was reintroduced in August 1853, with a number of clauses being passed before the Bill was discharged from the agenda paper in mid-December.

The Bill returned in September 1854, with Attorney-General John Plunkett adding a note of urgency since 'it was his intention as soon as it was passed, to introduce a Marriage Bill' (*SMH*, 21 Oct. 1854). Plunkett noted that the Bill had been before the House many times, and that legislation to the same effect had already been passed in all other colonies. It was only right that a 'debtor and creditor account of the mortality of the colony' be kept, and 'It was necessary that these statistics should be kept for many reasons, as they laid the basis of all social science, and by the information offered by them social progress could alone be made' (*SMH*, 27 Oct. 1854).

Plunkett thought that a sum of £5,000 might be needed to bring the Bill into operation, but that the fees raised would soon make the measure self-supporting. Drawing on the experience of Victoria, which had passed an Act the previous year, Plunkett intended to adopt the same type of printed forms. Charles Cowper, now Member for Durham, who had first introduced measures for improved registration more than ten years before, stressed the importance of appointing the correct officers to undertake registration and that these officers not be members of the clergy. The clauses were then considered in detail, following the House resolving itself into a Committee of the whole.

James Martin, Member for Cook and Westmoreland and later also to be Premier and Chief Justice of the Supreme Court, objected to the not inconsiderable amount that was required to establish the Bill's measures, when improvements in the colony were being neglected, and 'the roads and bridges throughout the colony were in a scandalous condition' (*SMH*, 2 Nov. 1854). Since the House was about to rise for that year, a movement was passed for further consideration of the Bill in six months time.

The Bill did not return until the following October when Governor General Sir William Denison transmitted a draft titled *A Bill for Registering Births, Marriages, and Deaths* to the Legislative Council (NSWLC, 11 Oct. 1855). Attorney-General Plunkett moved for a first reading on 17 October 1855, explaining that the Bill was a necessary accompaniment to the just-passed Marriage Act, and proposing a one-year trial so that it could be amended as necessary (*SMH*, 18 & 19 Oct. 1855). James Martin again rose to voice objections both moral and financial, but the Bill went to a Committee of the whole.

Each clause was considered in turn. The seventh clause, fees for the performance of duties, was spoken against by Charles Cowper and Solicitor-General William Montagu Manning, and was postponed pending an estimate for the office. A number of other clauses were also postponed, and one was expunged (*SMH*, 25 Oct. 1855).

Further consideration of the Bill was postponed a number of times and it did not come before the House again until mid-November. The House went into committee, altered the troublesome seventh clause, introduced several new clauses, and altered others to produce an amended Bill. The report from the Committee of the whole Council was adopted, and following a third reading, the Bill was passed on 23 November 1855 (NSWLC, 23 Nov. 1855). *An Act for Registering Births, Deaths, and Marriages* (19 Vic. 34) received Royal Assent on 3 December 1855 and became operative on 1 March 1856. Registration of births, deaths and marriages across all the colonies of Australia dates from this commencement.

Christopher Rolleston, formerly a commissioner of crown lands and private secretary to Governor-General Denison, was appointed Registrar-General. He divided the Colony into fixed districts, secured Registrars for each, and devised schedules and instructions for the recording of information.

Quarterly returns of registration were to be made by District Registrars and supplied to the Registrar-General, who was to keep these returns in the General Registry. Annual compilations of registered deaths, in table form with commentary, were to be presented to Parliament. These compilations continued

to be functions of the Registrar-General until the appointment of a Government Statistician and the creation of the New South Wales Bureau of Statistics in 1887.

In his Second Annual Report for the year 1857, Rolleston commented on

'... the growing appreciation of the advantages, social, political, legal, statistical, and sanitary, which have been found to attend the collection of vital statistics in every civilised country. As a necessary branch of vital statistics, the importance of a Civil Registry of Births, Deaths, and Marriages, is every day becoming more apparent...errors as to facts are thus daily exploded, and more just data are supplied for the judgment of the legislator, and for the right comprehension of the principles which should guide the proceedings of governments and societies, to the promotion of the physical and moral improvement of the people; and, besides this, the importance of having legal records easily accessible, to give security to the principles of inheritance, and to the legal succession to property, is very generally felt and acknowledged' (*NSWRG*, 1858).

In 1878, the *Clergy Returns Transfer Act* provided for copies of the pre-civil registration Church of England records of baptisms, marriages and burials, which were held by the Supreme Court, to be transferred to the Registrar-General. Other denominational church records were also subsequently transferred.

The *Registration of Births, Deaths and Marriages Act, 1899* repealed the 1855 Registration Act, but this was simply a consolidating Act and made no changes to the law and practice of registration.

Medical certification in the registration of deaths did not become compulsory in New South Wales until the passing of the *Registration of Births, Deaths and Marriages (Amendment) Act 1934* (Cumpston, 1989).

Queensland

Queensland was separated from New South Wales on 6 June 1859, when Queen Victoria signed the Letters Patent to proclaim the new colony. Queensland was

the only colony which commenced with its own parliament, rather than first operating as a Crown Colony.

Registration of death in the area which became Queensland had been required under the New South Wales legislation of 1855. Ten New South Wales Registry Districts formed the Northern Division and these would eventually become the 'new northern Colony' of Queensland (New South Wales Registrar-General, 1858). Summary tables of deaths in Brisbane are available from 1856 and in Queensland from 1860.

Following the separation of Queensland as an independent colony, the 1855 New South Wales Act remained in force. A Register-General's office was established on 6 January 1860 with the Reverend Robert Creyke, District Registrar for Moreton Bay, acting in the position until September when Frederick Orme Darvall was appointed Registrar-General.

The *Registration of Births Deaths and Marriages Amended Registration Act, 1867* (31 Vic 7) repealed some clauses of the 1855 Act, and altered the permissible time for registration of births. Regulations under Section 6 of the Act, dated 18 December 1885, made a medical certificate compulsory for specifying the cause of death (Cumpston, 1989). From 1904, the Registrar-General in Queensland also held the position of Government Statistician.

ESTABLISHING A SYSTEM FOR REGISTERING DEATHS—W. H. ARCHER IN VICTORIA

Following the passing of their Act, each colony was faced with implementing a workable system to carry out the requirements of registration. The burden fell on those public servants who were first appointed as Registrars General—Charles Bethel Lyons in Tasmania, George F. Stone in Western Australia, Charles Sturt in South Australia, W. H. Archer in Victoria, Christopher Rolleston in New South Wales, and Robert Creyke and F. O. Darvall in Queensland (see Appendix 2).

William Henry Archer (1825–1909) emerged to become a distinguished pioneer in the establishment of official statistics, firstly in Victoria, but then more widely

as the other colonies looked to him for assistance, innovation and leadership. For this reason, Victoria provides an informative example of how colonial systems of civil registration were first established.

Archer was suited to the role. He had trained and had worked as an actuary and statistician in London, prior to his emigration to Port Phillip in 1852 (Hopper, 1986). This was a time of extensive development for the new science of statistics, following the establishment of the Statistical Department of the Board of Trade in 1832, the foundation of the Statistical Society of London (later the Royal Statistical Society) in 1834, and the General Register Office in 1837 following the passing of the Registration Act (Forster & Hazlehurst, 1988).

Archer benefitted from training and working within this milieu. He was greatly influenced by Dr. William Farr (1807–1883), the renowned British epidemiologist and vital statistician who played such an important role in the reporting and analysis of causes of death at the General Register Office. Both Farr and Archer shared an interest in medical statistics, and the two corresponded throughout their lives.

Eighteen fifty three was a busy year for Archer. He had arrived in Melbourne in November 1852, and with the passing of the Act in January, the colonial administration in Victoria needed someone to administer it. Archer sought employment in the statistical branch of the Colonial Secretary's Department. In February 'the Colonial Secretary…placed in my hand the Act…requesting me to draw up a general plan for the guidance of the Registrar-General, and rules in detail for the Deputy Registrars' (Archer, 1854). Lieutenant-Governor La Trobe appointed Archer as clerk to read the new Act.

Archer requested identical boundaries for both registration and census collection through two replies dated 10 March and 22 March, and suggested that schedules should record similar information to that recommended by the Registration Committee of the Statistical Society of London. His proposals indicate that he saw his role as acting more broadly so as to establish the profession of statistics in the colonies (Forster & Hazlehurst, 1988). Having a particular interest in infant mortality, he emphasised the collection of social

statistics on health and education. La Trobe was quick to approve Archer's proposals.

The Act provided that registration in Victoria commence on 31 March 1853, but in practice it did not come into operation until 1 July (Campbell, 1855a). Notices and handbills advertising the new requirements and locations where registration might take place were circulated from late June, and instructions were regularly published in the press (see, for example, *Argus*, 30 Jun. 1853 and 6 Sep. 1854). The Act prescribed that the particulars to be furnished in a case of death should include:

- Name
- Rank or Profession
- Sex and Age
- Date and Place of death
- Cause of death, Person certifying, Date last saw deceased
- Burial place
- Place of birth, Years lived in Colony
- Parent's Names and Rank or Profession
- Marital status and Issue

Occupants of houses where a death had occurred were required to give notice to the Deputy Registrar of their district within 15 days of the event. Persons present at the death or attending the last illness also had to provide information within 60 days. Coroners were to inform the Registrar-General of the particulars of deaths in those cases where an Inquest or Magisterial Enquiry was held.

Once a registration had been completed, Deputy Registrars gave a certificate of registry of death to an undertaker or other person having charge of the funeral, so that burial might take place. Informants were also entitled to receive a certificate of death free of charge.

Every quarter, Deputy Registrars were to send in a duplicate sheet of each registration to the Registrar-General's office. These were checked and amended

if needed, indexed and then examined by 'a medical gentleman' for the purposes of classifying diseases and tabulating mortality. From these returns, abstracts summarising deaths in the colony were compiled and published.

La Trobe had appointed Archer as Acting Registrar-General in April 1853 at an annual salary of £600. Archer had hoped to receive permanent status but he was informed in August that the position would go to the Governor's private secretary, Major E. S. Norman Campbell, who commenced in the role in January 1854. Campbell, however, proved to be an amiable superior and Archer was left free from administrative responsibility to travel throughout the colony in 1853–54, recruiting and advising Deputy Registrars.

The system of registration took several years to fully establish. Doctors had to be trained in the use of William Farr's cause-of-death classification, or nosology, and clergy in the completion of marriage forms. Assistant Registrars had to be found to serve the irregularly distributed population within the colony.

By April 1855, 127 registration officers had been employed, 'settlers, medical men, clerks of the peace and petty sessions, schoolmasters, postmasters, chemists and druggists, and sometimes storekeepers', as well as 133 ministers of religion to register marriages. Numbers of unpaid Assistant Deputy Registrars, usually settlers in remote country areas, were co-opted to aid in the task.

As in Tasmania, the government had decided to remunerate Deputy Registrars on the basis of work actually done in the form of completed registrations, rather than pay them fixed salaries. This provided a 'wholesome stimulus' to the system, so that Registrar-General Norman Campbell could say 'registration has been so constantly increasing in efficiency that, I believe, in the greater portion of the Colony, comparatively speaking, not many of the births or deaths escape notice' (Campbell, 1855a). This view is tempered by other statements to the effect that 'many deaths necessarily remain unregistered' among the largely mobile goldfields population (Campbell, 1855b).
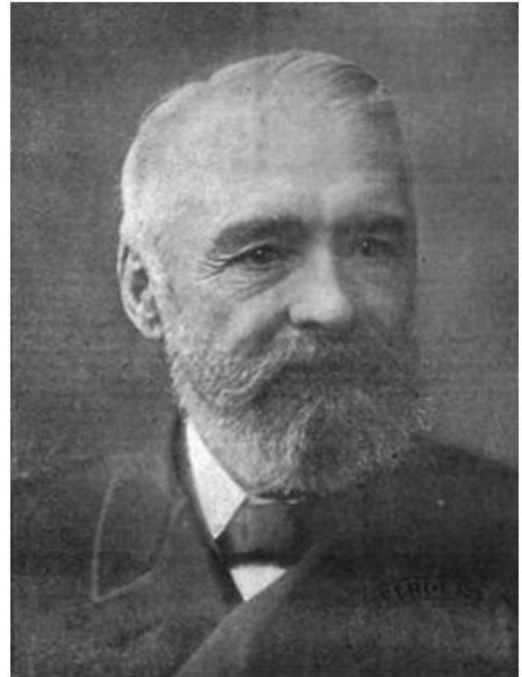
Figure 2.4: William Henry Archer (1825–1909), and Henry Heylyn Hayter (1821–1895)

Figure 2.5: Sir Timothy Augustine Coghlan (1855–1926), and John Howard Lidgett Cumpston (1880–1954)

Along with the recommendations for registration, Archer had also been asked to prepare the *Blue Book*, and in assembling a collection of general statistics for that publication, he began an action that would ultimately result in the Registrar-General also becoming the Government Statistician in Victoria. The Registrar-General's Department took over functions previously carried out by the Statistics Branch of the Colonial Secretary's Department, as well as other related responsibilities such as census taking, preparation of statistics for Parliament, and administering Friendly Society annual returns (Public Record Office Victoria, 2011).

Under the title *Statistics of the Colony of Victoria* the annual publication incorporated the newly collected information on deaths, and it was later to expand to become the *Statistical Register of the Colony of Victoria*. Archer's first months also produced *The Statistical Register of Victoria, From the Foundation of the Colony: With an Astronomical Calender for 1855* (Archer, 1854), which compiled a large array of information including historical deaths data, a record of much of his work to date, and an abridged version of Farr's nosology. Archer also circulated Farr's nosology for the benefit of the Deputy Registrars in a separate publication (Archer, 1856), despite this version first been used in England and Wales for the Registrar-General's Fourth Annual Report as long ago as 1840–41.

Following E. S. Norman Campbell's death in January 1859, Archer was made Registrar-General, holding this position until May 1874 when he was promoted to Secretary of Lands and Survey. Archer worked for uniform statistical procedures throughout the colonies during this time, joining with New South Wales in persuading South Australia and Queensland to conduct a census on the same day as the English census of 1861, as well as moving towards similar reporting in the *Blue Books*.

On the suggestion of South Australia, a meeting of colonial statisticians was held in October-November 1861, attended by the Registrars-General of New South Wales, Victoria and Queensland (Rolleston, Archer and Darvall) and the South Australian Government Statistician (Boothby), with Archer presiding as

the dominant figure (Forster & Hazlehurst, 1988). The conference worked to achieve uniformity in the content and classification of the annual statistical publications.

Besides the annual publication of mortality abstracts in *Statistics of the Colony of Victoria*, Archer also compiled abstracts of mortality in Melbourne and suburbs. These appeared weekly in the *Government Gazette* from July 1856, with monthly and quarterly summaries being added from January 1860.

A newer version of Farr's nosology was published by Archer in 1863, incorporating ten years of modifications since the earlier version had been circulated, with a third edition appearing in 1868 (Archer, 1863, 1868). Archer also continued to popularise statistics among the wider public, publishing works such as *The Progress of Victoria: A Statistical Essay* which included a summary of deaths occurring during the decennial period 1856–1865 (Archer, 1867).

Archer was succeeded in the position of Registrar-General by Richard Gibbs in 1874, leaving behind a well-functioning government department which reported comprehensively on mortality in the colony,

> 'The statistical records of Australia are not excelled either in fullness or in accuracy by those of any other country; and as the statistical system initiated in Melbourne in 1853 is gradually being followed by statisticians in surrounding states, there is every reason to hope that, at no distant date, thorough unity will exist both of purpose and of action in relation to all the leading lines of statistical work throughout Australasia' (Archer, 1873).

At this time a separate Statistical Branch was created, with the position of Government Statistician filled by Henry Heylyn Hayter from 1874 to 1895. Hayter took over the statistical content of the annual reports, the publication of mortality abstracts and the revision of the nosologies, soon becoming the most eminent statistician in Australia.

SUMMARY

From the commencement of European colonisation, statistics on deaths were collected and returned to the Colonial Office in London. After 1810, the administrative powers took advantage of ecclesiastical record-keeping of baptisms, marriages and burials, including additional religious denominations as legislation allowed, and these ecclesiastical records served as the vital statistics of the colonies. This system afforded only partial coverage of the population, and the records provided no legal surety or proof of either birth or death.

Within two decades of the passing of legislation for the civil registration of deaths in Britain in 1836, each Australian colony also had an Act in place. Systems administered by public servants were established to register vital events, with a Registrar-General heading a team of assistant District Registrars. William Henry Archer was instrumental in establishing Victoria's system, and in facilitating better registration in the other colonies.

Chapter 3

Colonial mortality data and its quality

The compilation and publication of summaries of records of deaths by the colonial Registrars and their statistical bureaus allows for the study of trends in mortality in nineteenth and early twentieth-century Australia. Although they are a rich data source, the use of these administrative data for demographic purposes first requires a careful assessment of their strengths and weaknesses.

CONTENT OF THE STATISTICAL ABSTRACTS OF MORTALITY

In this work the primary sources of information are the 'abstracts of mortality' which were included in the annual reports produced by the colonial Registrars and Government Statisticians. These abstracts date from the commencement of civil registration in colonies. They take the form of tables of yearly numbers of registered deaths classified by sex, age group, and cause of death.

The abstracts were included in either a specific report on births, deaths and marriages, or in a broader volume of vital statistics. These reports were usually published as Parliamentary Papers, or in Government Gazettes. Extracts or whole reports were often reproduced in contemporary newspapers or in medical journals such as the *Australasian Medical Gazette*.

The upper bound in this thesis is the year 1906. From 1907, abstracts of mortality for the entire Commonwealth of Australia appear in the *Population*

and Vital Statistics Bulletin, a publication issued by the newly-established Commonwealth Bureau of Census and Statistics, headed by G. H. Knibbs (CBCS, 1908).

The level of detail included in the statistical abstracts of mortality grew with time. Beginning with simple tabulations of annually registered deaths by sex, the reports variously added classification by age group, district, time period, cause of death, occupation and other variables. These details were not collected or reported uniformly in all colonies at all times, and so to compare colonies a degree of harmonisation or adjustment of sources must take place. This is required, first and foremost, for the classification of age and of cause of death.

Table 3.1 indicates whether sex, age or cause-of-death was reported in the statistical abstracts, and in which publication these abstracts appeared. An entry appearing in brackets in the table indicates that, although reported, it lacks the comparability needed for calculating age-standardised rates. Deaths in the earliest South Australian abstracts, for instance, were reported for age groups 0–1, 2–4, 5–9, 10–29, 30–49 and 50+, rather than the 5-year age structure employed here for age standardisation (0, 1–4, 5–9…70–74, 75+).

Problems also arise with classifications of cause-of-death. Non-standard nosologies and cause-of-death terminologies were sometimes used. More commonly, deaths classified to a particular cause might be combined with other causes and published as groups or sub-groups, so that it is impossible to learn how many deaths occurred from an individual cause (Cumpston, 1989). Cause-of-death reporting will be discussed more fully in Chapter 5.

Ideally, deaths would be classified in a standard fashion across all colonies for all three of the variables of interest (i.e. by Sex x Age x Cause). In some cases this does not occur. The Tasmanian abstracts, for example, lack standard age classifications before 1868, and cause-of-death reporting is also absent. Although South Australia introduced cause-of-death reporting in their abstract of 1856, there was a hiatus from 1868 to 1872 when no causes were given. New South Wales, Victoria and Queensland grouped causes of deaths together in

their abstracts for certain time periods. In the case of New South Wales, this occurred for almost two decades between 1856 and 1874.

Western Australia is especially problematic. Reporting began well, with Registrar George Frederick Stone compiling comprehensive abstracts covering the years 1841–42 to 1847–48 for the small annual numbers of deaths in the colony. However, reporting ceased on the orders of the government, and Stone resigned in 1854 following 'some disagreement' with Governor Charles Fitzgerald (*PG*, 14 Apr. 1854).

A hand-written annotation in the *Western Australian Blue Book* of 1855 laments

> 'Owing to the defective nature of the Registration Act and the inability to enforce the Registration of Deaths this Return must be not taken as the actual number of deaths which have occurred in the period quoted but only as the number of Registrations effected. Probably an addition of 50 per cent would be nearer the true number of deaths' (WABB, 1855)'.

Only limited reporting on deaths appeared in the *Western Australian Blue Books* and census reports for the next fifteen years. Detailed reporting recommenced in 1870 although five-year age groups were not employed before 1886, and individual causes of death were grouped together from 1886 to 1896.

In Western Australia, the desired Sex x Age x Cause format does not appear until 1897, coinciding with the commencement of a period of rapid development in the colony; the discovery of gold, a dramatic rise in population and the appointment of the respected Registrar-General and Government Statistician Malcolm A. C. Fraser all occurred at this time.

In this work, an alternative data source is used for Western Australia alongside the statistical abstracts of mortality, in order to provide a standard classification of Sex x Age. Historic indexes of deaths from 1841 to 1906 are available through the Internet and these give information on name, year of death and age at death (Government of Western Australia, 2014).

Table 3.1: Reporting of deaths tabulated by sex, age and cause in statistical abstracts of mortality, 1839–1906

Colony/Years	Variables reported	Publication
Tasmania		
1839–1846	Sex	TasSR
1847	Sex x (Age)	TasSR
1848–1849	Sex x Age	TasSR
1850–1867	Sex x (Age)	TasSR
1868–1906	Sex x Age x Cause	TasSR
Western Australia		
1841–1847	Sex x (Age) x (Cause)	WAGG, WABB
1848–1854	Sex x (Age), (Cause)	1854 Census, WABB
1855–1858	Sex	1861 Census, WABB
1859	Sex x (Age) x (Cause)	1859 Census, WABB
1860–1869	Sex x (Age), Sex x Cause	1870 Census, WABB
1870–1885	Sex x Cause	WASR
1886–1896	Sex x Age, Sex x (Age) x (Cause)	WASR
1897–1906	Sex x Age x Cause	WASR
South Australia		
1842–1855	Sex x (Age)	SASR
1856–1861	Sex x Age x Cause	SAGG
1862–1867	Sex x Age x Cause	SASR
1868–1872	Sex x (Age), Sex x Cause	SASR
1873–1906	Sex x Age x Cause	SASR
Victoria		
1853–1863	Sex x Age, Sex x Cause	VicSR
1864–1906	Sex x Age x Cause	VicSR
New South Wales		
1856–1862	Sex x Age, Sex x Cause	NSWRG
1863–1874	Sex x Age x (Cause)	NSWRG
1875–1906	Sex x Age x Cause	NSWRG
Queensland		
1859–1869	Sex x Age x (Cause)	QldRG
1860–1863	Cause	QldRG
1870–1906	Sex x Age x Cause	QldRG

Note: See Bibliography for publication abbreviations.

These indexes do not explicitly state the sex of the deceased, and this must be inferred from their names. Consequently there is a level of uncertainty regarding the sex of a number of individuals. Additionally, this source supplies the year of occurrence of death, and not the year of registration. Since the colonial abstracts report on year of registration of death, there will be some anomaly in numbers, although this is not considered critical. Lastly, the Western Australian historic indexes provide no information on cause of death.

Individual records of deaths in electronic format are currently only available for two colonies. For Western Australia, limited information is available through the afore-mentioned online indexes from 1841 onwards. For Tasmania, Gunn and Kippen have assembled *Tasdeaths*, a database of information on approximately 93,000 deaths appearing in the Tasmanian death registries for the period 1838–1899. This source has been used in the past to report on cause-specific deaths in Tasmania (Kippen, 2002a).

The original colonial registers of deaths are still extant, and copies are widely available on microfiche, microform, CD-ROM and other formats. These are commonly used for genealogical research, but they do not contain electronic abstracts of the type needed for demographic analysis. Some of the information missing from the statistical abstracts of mortality might be reconstructed from these sources, but this enterprise is not attempted here.

In 1989, the Australian Bureau of Statistics made available on microfiche most of the publications and compilations produced by Australia's six colonial statistical bureaus and their precursors (ABS, 1989). This collection includes the colonial Registrar-General and vital statistics reports.

NUMBERS OF REGISTERED DEATHS

The numbers of deaths registered in the colonies are presented in Table 3.2. In 1856, the year in which registration was first carried out in all the colonies of Australia, 11,812 deaths (7,307 males and 4,505 females) were recorded. By 1906, registrations had risen to 44,281 (25,322 males and 18,959 females) (Figure 3.1).

Table 3.2: Registered deaths in statistical abstracts of mortality, by colony, 1839–1906

Year	NSW[1]	Vic[2]	Qld[3]	WA[4]	SA[5]	Tas[6]
1839						485
1840						501
1841				13		484
1842				29	143	577
1843				41	158	1,140
1844				53	140	786
1845				63	238	697
1846				31	360	736
1847				55	495	953
1848				68	509	773
1849				29	834	1,013
1850				54	986	1,070
1851				37	973	1,107
1852				65	1,098	1,449
1853		2,158		92	1,275	2,017
1854		6,261		58	1,346	1,928
1855		6,603		58	1,663	1,692
1856	3,506	5,728		96	1,147	1,335
1857	4,846	7,449		164	1,304	1,418
1858	5,883	9,015		164	1,859	1,562
1859	5,642	9,469	331	209	1,923	1,410
1860	6,562	12,061	478	217	2,336	1,749
1861	5,343	10,522	499	257	1,962	1,477
1862	6,524	10,080	797	229	1,918	1,362
1863	6,652	9,502	1,275	251	2,221	1,418
1864	6,445	8,887	1,446	329	2,565	1,433
1865	6,596	10,461	1,733	333	2,174	1,263
1866	7,361	12,286	2,362	327	2,753	1,327
1867	8,631	11,733	1,745	360	2,939	1,418
1868	7,225	10,067	1,799	354	2,516	1,464
1869	6,691	10,630	1,761	334	2,211	1,335
1870	6,558	10,420	1,645	378	2,544	1,404
1871	6,407	9,918	1,785	332	2,378	1,355
1872	7,468	10,831	1,936	358	2,896	1,411
1873	7,611	11,501	2,250	418	2,631	1,503
1874	8,652	12,222	2,794	487	3,434	1,689
1875	10,771	15,287	4,104	473	4,136	2,078
1876	11,193	13,561	3,467	383	3,550	1,730
1877	9,869	12,776	3,373	433	3,235	2,038
1878	10,763	12,702	4,220	394	3,749	1,700
1879	10,200	12,120	3,207	411	3,580	1,688

(continued)

Table 3.2: Registered deaths in statistical abstracts of mortality, by colony, 1839–1906 (continued)

Year	NSW	Vic	Qld	WA	SA	Tas
1880	11,231	11,652	3,017	382	3,912	1,832
1881	11,536	12,302	3,320	424	4,012	1,733
1882	12,816	13,634	4,274	430	4,393	1,906
1883	12,249	13,006	5,041	560	4,435	2,122
1884	14,220	13,505	6,861	707	4,789	1,990
1885	15,282	14,364	6,235	600	3,987	2,036
1886	14,587	14,952	5,575	806	4,234	1,976
1887	13,448	16,005	5,166	702	3,944	2,161
1888	14,408	16,287	5,529	673	3,759	2,036
1889	14,796	19,392	6,132	611	3,501	2,098
1890	14,218	18,012	5,638	540	3,923	2,118
1891	16,286	18,631	5,170	869	4,211	2,234
1892	14,410	15,851	5,266	931	3,711	2,069
1893	16,022	16,508	5,695	945	4,520	2,071
1894	15,170	15,430	5,298	1,081	4,001	1,938
1895	14,914	15,636	5,152	1,604	3,921	1,811
1896	15,839	15,714	5,645	2,020	4,038	1,901
1897	14,264	15,126	5,423	2,643	3,973	1,947
1898	16,661	18,695	6,243	2,716	4,675	2,359
1899	15,901	16,578	6,144	2,324	4,406	2,204
1900	15,118	15,215	5,747	2,240	3,774	1,903
1901	16,021	15,904	6,007	2,519	3,974	1,814
1902	16,646	16,177	6,204	2,823	4,236	1,914
1903	16,497	15,595	6,346	2,788	3,873	2,116
1904	15,360	14,393	5,250	2,817	3,719	1,974
1905	14,978	14,676	5,503	2,709	3,761	1,844
1906	14,975	15,237	5,095	3,084	3,872	2,018

Notes:

1. 1856 is 1 March to 31 December.

2. 1853 is 1 July to 31 December.

3. Queensland was part of New South Wales before 1859.

4. 1841–1848 are years ending 31 August.

5. 1843 is 1842–43. Deaths in the Northern Territory from 1891 are excluded.

6. Registered deaths of free persons only.

Over this half-century, registrations increased almost four-fold, at an average of 2.5 per cent per year for males and 2.9 per cent for females.

The rise in registrations reflects the increasing size of the colonial population. Annual fluctuations largely reflect the severity of disease outbreaks, particularly of infectious diseases.

Figure 3.1: Annual number of deaths registered in Australia, 1856 to 1906
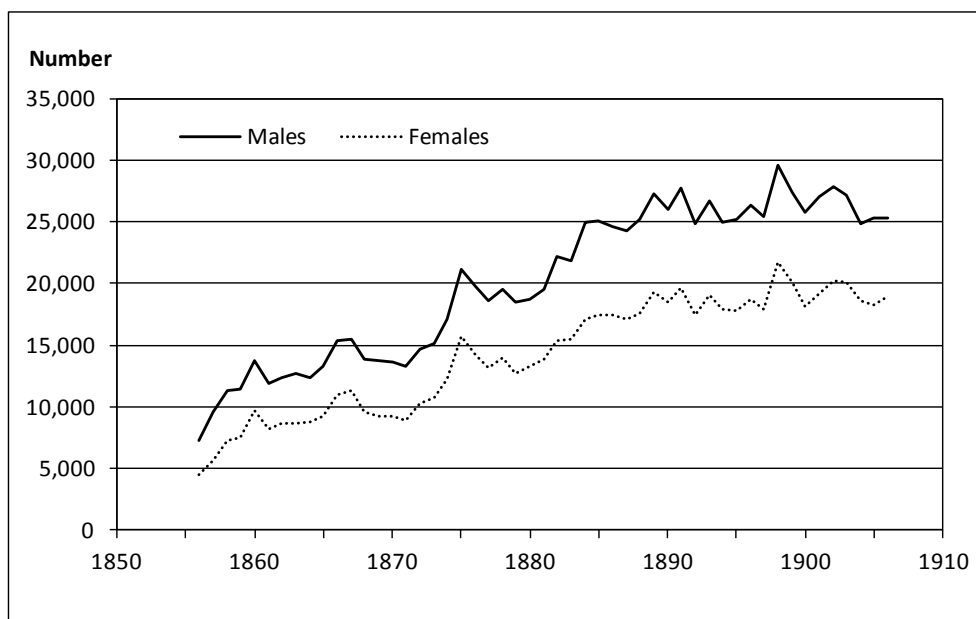
In each year, more male deaths were registered, reflecting in part the sex imbalance in the population. During the 1850s, in excess of 150 male deaths were registered for every 100 female deaths (Figure 3.2), although for most of the fifty-year period to 1906 the ratio was around 140 male deaths for every 100 female deaths. By way of comparison, in 2009 there were 106 male deaths for every 100 female deaths (ABS, 2010).

Figure 3.2: Sex ratio of deaths registered in Australia, 1856 to 1906
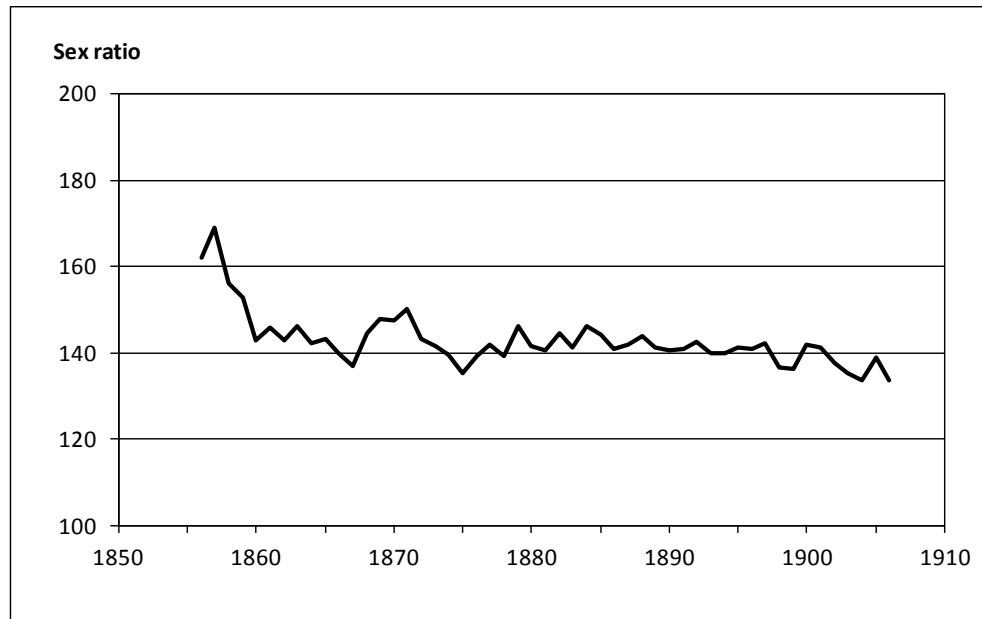
THE COMPLETENESS AND ACCURACY OF COLONIAL MORTALITY DATA

Since it is the primary source for demographers, epidemiologists and historians who wish to study death and disease in colonial Australia, the data in the abstracts of mortality warrants careful examination. It is valid to ask questions about the completeness of registration—especially in the early years following the establishment of a system of civil registration—and whether details of the deceased were correctly recorded, particularly their age and their cause of death.

The possible deficiencies of the abstracts of mortality are here considered under the following headings (Shryock & Siegel, 1976),

- Completeness of registration
- Completeness of recording of sex, age and cause of death
- Accuracy of the definition of death
- Accuracy of recording of sex, age and cause of death
- Accuracy of recording of time and place of deaths.

COMPLETENESS OF REGISTRATION

Carefully derived estimates of the completeness of registration in the Australian colonies are difficult to obtain. A suite of demographic methods, commonly known as 'death distribution methods', attempt to estimate the proportion of deaths which are collected and counted by civil registration systems (Murray *et al.*, 2010).

One of the most widely used of these, the Generalised Growth Balance method, relies on a mathematical relationship to balance birth rates, growth rates and death rates. It requires as inputs the age distribution of population from two censuses and the deaths registered between the two censuses by age. The attempt to balance these involves the estimation of the completion of registration, and results in a correction factor which is used to obtain adjusted mortality levels.

A recent variation of the Generalised Growth Balance method also adjusts for populations that are affected by migration (Bhat, 2002). Migration levels in nineteenth-century Australia were high, and so the Bhat method could assist in assessing the completeness of registration in these populations. It is, however, very sensitive to the accuracy of the population counts in the two censuses, and to errors in the age distribution of deaths (ABS, 2008b; Barnes *et al.*, 2008). Since this work relies on populations derived from problematic census counts with fluctuating migration levels (Hayter, 1893), the method was judged unsuitable. Instead, more direct observations provide the evidence for assessing the completeness of registration.

By all accounts, the Registrars General believed that registration was quickly established throughout their jurisdictions with substantial, although not complete coverage. The early years produced the sort of problems that necessarily accompany the introduction of new rules and regulations. People were unaware or unfamiliar with the new requirements, and needed time to acquaint themselves with systems requesting that they divulge personal and intimate details.

Some wondered what 'the public good' or the statistical value of the information might be, and under whose authority details on the domestic history of their family were being collected. The print media carried public debates on the new regulations. The more strident views insisted that it was just 'not correct' to ask these questions of the recently bereaved; they were 'inquisitorial', 'offensive' and 'puerile', and an 'insult to the whole community' (*SMH*, 30 Oct 1856, p.3; 3 Nov. 1856, p.8; 30 Sep. 1857, p.5).

Some refused outright to register births and deaths, although these cases were rare; '…few are believed wilfully to neglect the Registration of Birth or Death, although some, particularly among foreigners, do so from ignorance' (QldRG, 1861).

In certain regions, a lack of zealousness not on the part of those being registered, but of the District Registrars called into question the completeness of records, as New South Wales Registrar-General Christopher Rolleston pointed out,

> 'The Districts of Tamworth, Carcoar, and Wagga Wagga, are the only districts in which I have had reason to be dissatisfied with the progress of the system. The Clerks of Petty Sessions have had charge of these districts; and I have reason to believe that the law has been allowed to remain almost inoperative in their hands' (NSWRG, 1857).

Generally, however, the early reports of the Registrars give the impression that there was little active resistance to the new arrangements, and that most of the problems surrounding the completeness of registration related to two areas: under-registration of deaths in remote areas, and the under-registration of infant deaths.

Under-registration in remote areas

In remote areas, people had trouble accessing Registrars. F. O. Darvall, the Queensland Registrar-General, reported the observations of one of his assistants,

> 'When it is considered that this is a Pastoral District, that many of the persons at sheep stations have no communication with the towns for months, or with the

head stations for weeks, that fully one-third of the labouring class are German or Chinese, and that in most instances the information is necessarily transmitted through the superintendent or proprietor, it will appear quite possible that many Births and Deaths occur which I never hear of' (QldRG, 1861).

A similar situation existed in South Australia, where

'It must be premised that these returns do not exhibit the exact and accurate numbers under each respective head of enumeration, but simply the number registered. In some of the outlying districts the difficulty connected with registration is considerable, and we fear that many omissions obtain in consequence' (SAA, 18 Apr. 1859, p.4).

Around 800 apparently unregistered deaths occurring in South Australia between 1842 and 1906 have been identified, the details of which have been made available for genealogical research (Jaunay, 2004). Over the same period, approximately 183,000 deaths were registered in South Australia, so these 800 deaths found to date represent less than one half of one percent of the total.

From 1891, G. H. Ayliffe, the Registrar-General in South Australia, began reporting on deaths in the area that would become the Northern Territory. The population in the Territory at this time numbered slightly more than 5,000 people, 'about 1,550 were Europeans by birth or parentage. The remainder were Asiatics, very nearly all of them being Chinese' (SARG, 1891). Exclusive of Aboriginal people, registered deaths in the Northern Territory between 1891 and 1906 never exceeded 100 per annum. These deaths are excluded from this work.

E. S. Norman Campbell, the Victorian Registrar, commented on the chaotic situation that existed on the goldfields,

'These difficulties arose…partly from the peculiar circumstances in which the Colony was placed, with its population either congregated in large masses on the Gold Fields and in a few large towns, or thinly scattered over a vast surface, almost devoid of roads or of the machinery for diffusing the system into the rural and more remote districts…It is my impression, however, that these latter figures represent but imperfectly the mortality that has taken place, especially on or about the gold fields, as it is difficult to overtake the instances of interments which have occurred in private burial places' (Campbell, 1855a).

The establishment of public cemeteries throughout the colony, and increased numbers of Deputy Registrars helped to deal with the problem in Victoria, so that Campbell could say in his next report that they 'have greatly diminished the probability that cases of mortality can, to any great extent, have escaped being registered' (VicRG, 1856).

By 1853, registration had been operating in Tasmania for more than a decade. Registrar John Abbott was confident enough to state that he believed registration to be complete, due to the 'liability of the Clergy to a fine in the event of their burying without a Certificate' (Kippen, 2002a). The requirement that a certificate be supplied before burial could take place, along with the penalties imposed on households for non-compliance helped undergird Abbott's confidence.

Victorian statistician H. H. Hayter also believed that 'The deaths which escape registration must necessarily be few, as in all colonies it is illegal to bury a corpse until the death has been registered, and there is every reason to believe that this law is not evaded' (Hayter, 1893).

Under-registration of infant deaths

Infant deaths are the second major area believed to be characterised by under-registration, especially in the years immediately following the establishment of civil registration. Here, techniques exist to quantify the level of deficiency.

Variation in infant mortality rates—the number of deaths of children less than one year of age per 1,000 live births—and the quality of registration data can be assessed using the biometric techniques developed by French demographer Jean Bourgeois-Pichat (1951) (see Appendix 1).

Kippen (2002a) used the Bourgeois-Pichat transformation to demonstrate that infant deaths in colonial Tasmania were under-registered relative to the number of births. Although under-registration existed in Tasmania between 1840 and 1899, the estimates of endogenous mortality, which fluctuated between 21 and 25 deaths per 1,000 live births, was not excessive (Wrigley, 1977; Galley & Woods, 1999).

Figure 3.3: Bourgeois-Pichat biometric analysis of infant mortality, Western Australia, 1870–79
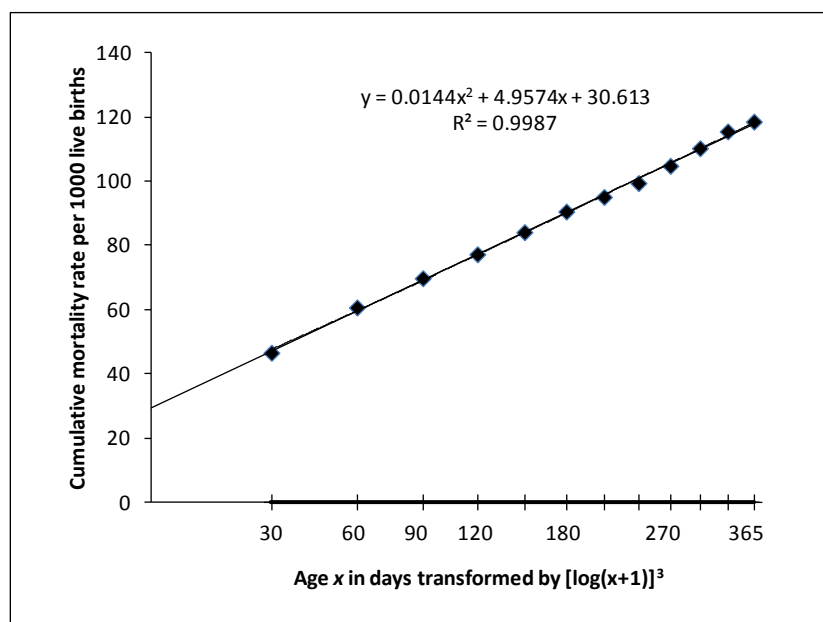
The Western Australian historical indexes also have the information required for calculating the Bourgeois-Pichat transformation; annual births and infant deaths classified by age in days. Infant mortality for the period 1870–79 was 118 deaths per 1,000 live births, and the estimated endogenous mortality was 31 deaths per 1,000 live births (Figure 3.3). This indicates a level of under-registration of deaths relative to births greater than in Tasmania, however the fitted curve is relatively straight, and the figure of 31 is not overly excessive.

There is also enough detailed data on age at death in the Victorian abstracts of mortality to calculate Bourgeois-Pichat transformations. Registrar-General Archer began publishing numbers of infant deaths in the abstracts of mortality for ages 'Under 1 month, 1 to 3 months, 3 to 6 months and 6 to 12 months' from 1867 onwards.

Endogenous infant mortality in Victoria fell from 26 deaths per 1,000 live births in 1870–79 to 21 in 1900–06 (Figure 3.4). The fitted curves display some concavity but again suggest that the data are not seriously misleading. Wrigley's value of 21 deaths per 1,000 live births for England and Wales in 1905 is similar to that in Victoria, and also 'suggest that endogenous mortality was

low at a time when there was no longer reason to doubt the completeness of registration' (Wrigley, 1977, p.304).

Figure 3.4: Bourgeois-Pichat biometric analysis of infant mortality, Victoria, 1870–79 to 1900–06
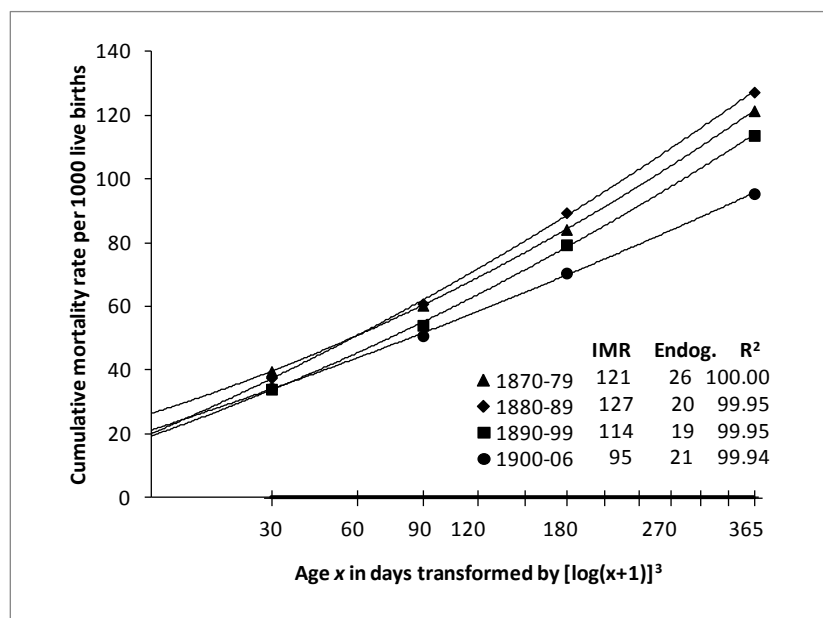
Under-registration of Aborigines and other peoples

In introducing civil registration of deaths, the colonies intended that it have universal coverage in their jurisdictions. Article XXI of the 1855 New South Wales Registration Act, for example, stipulated that '…in each case of the death of any person, the tenant of the house or place shall within thirty days…inform the District Register of such…death and of all the particulars concerning the same'.

Although the provision existed, the completeness of registration of Aboriginal deaths varied throughout the colonies. Over the course of the 19th century, more Aboriginal deaths were recorded, particularly if families were in regular contact with church or government officials. The deaths of Aboriginal people who were living a traditional lifestyle in rural and remote areas remained almost totally unregistered.

In Victoria, the remnant Aboriginal population was under state surveillance on reserves from the early 1870s, and the colony's well-developed registration system captured many of their births, deaths and marriages (Smith *et al.*, 2008).

In Western Australia, the first recognisably Aboriginal names in the historical indexes appear in 1874, when the deaths of 'Johnny Aborigine' aged 24, 'Johnny Nurnurgub' aged 21, and 'Naringa' age unknown were registered. More Aboriginal persons were registered in the following decades; they were generally males with Anglicised given names and the surname 'Aborigine', although men such as Yandamarra who died in 1877 at an unknown age, and Wandigill in 1879 aged 22 also appeared. Few Aboriginal women or children were registered.

The majority of Aborigines whose deaths were registered in Western Australia appear to have come from the penal institution on Rottnest Island (Durey, 1980). Colonial Surgeon Alfred Waylen reported on an influenza epidemic that led to the deaths of 70 native prisoners on the island in 1883, so that 'The registration of the deaths of these aboriginals had a very appreciable bearing on the rate of mortality affecting the whole Colony' (WAPH, 1884).

In Western Australia, the registration of deaths of Chinese, Indonesian, Indian and Malay men also increased in frequency. Japanese deaths begin to appear, their numbers coinciding with the cyclones that struck the pearling industry vessels that were operating along the north-west coast.

Chinese and Pacific Islander immigration increased, in part to meet the labour shortages that followed the cessation of convict transportation in 1840. At the height of the gold rush, the total number of Chinese immigrants reached more than 40,000 according to one estimate, with most settling in New South Wales and Victoria (Cumpston, 1989). During the years 1853–1861, 2,183 Chinese deaths were registered in Victoria (Archer, 1867). By 1886, approximately 10,000 Pacific Islanders were living in Queensland, working on cotton and cane plantations. The completeness of registration for these two population groups may also be questioned.

Tasmania was unique among the colonies in that the deaths of convicts were kept in a separate register from the remainder of the population and were not included in the abstracts of mortality. Article XXII of the 1838 Tasmanian Registration Act stipulated that '…nothing contained in this Act shall extend or apply to the registration of the death of any prisoner of the Crown serving under an unexpired sentence of transportation in this Island or its Dependencies whether the same shall have been partially remitted or not'. Only deaths among the free population appeared in the abstracts of mortality, although occasionally convict deaths were recorded in the civil registers, particularly if they had held tickets of leave, and those reporting the death were not aware that they were under sentence (Kippen, 2002b). In 1860, 13 prisoner deaths were registered, and by 1865 there were only three (TasSR 1866).

COMPLETENESS OF RECORDING OF SEX, AGE AND CAUSE OF DEATH

Over the period 1856–1906, 1.75 million deaths registered in Australia appeared in the statistical abstracts of mortality. Only a handful of deaths of persons of unknown sex were reported in the abstracts—one occurred, for example, in New South Wales in 1863 and another in Tasmania in 1865, although several more have been identified in the *Tasdeaths* data file (Kippen, 2002a).

In the abstracts, 10,341 deaths (or 0.6 per cent of the total) lack information on age at death. Males with unknown ages were more common than females (0.9 per cent vs. 0.2 per cent). Reporting of age at death improved over time; whereas in 1856–59, 1.5 per cent of deaths had no recorded age, by 1900–1906 this had fallen to 0.2 per cent.

In terms of the proportions of total annual deaths, Queensland and Western Australia were the colonies most affected by omission of age. Queensland had more than 100 annual deaths with no recorded age in 1868, in 1871 and in each year from 1873 to 1880 (Figure 3.5). Western Australia had less than 500 registered deaths per annum in the years to 1883, but also had 7 deaths with no recorded age in 1847, 6 in 1855, and 31 in 1881. A numbers of these were

coincident with catastrophic events, such as natural disasters or epidemics of disease.

In this work, deaths with unknown ages have been proportionately distributed over all age groups (see Appendix 1).

Figure 3.5: Proportion of deaths with no recorded age in the Queensland and Western Australia abstracts of mortality, 1841–1906
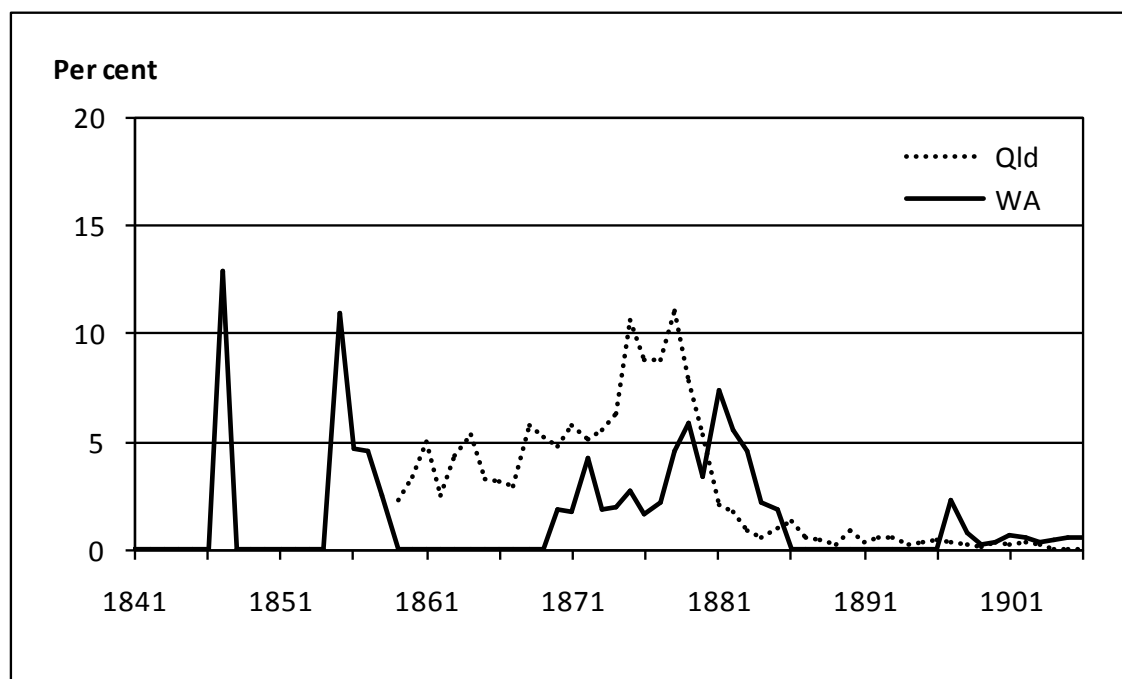

Deaths for which no cause, or unascertained causes were recorded are more common, with at least 13,000 (or 0.8 per cent of the total) occurring from 1853 to 1906. The abstracts of mortality report these under a number of headings, using the classifications that were available in the nosologies of the time. The headings include 'Causes not specified', 'Sudden deaths (cause unascertained)' and 'Other ill-defined causes' (see Appendix 4).

There are several reasons why a death might appear under one of these headings. No cause might appear on the certificate of death or the registry entry, these having been left completely blank. The Colonial Surgeon, Alfred Waylen, urged for improvement in the Western Australian system of registration, finding that 7 per cent of deaths in 1881 had no cause specified at

all (WAPH, 1882). Alternatively, an entry might appear on the certificate of death, but because the attending doctor or coroner was unable to come to a decision as to what brought about death, or specified a vague or nonsensical cause, the Deputy Registrar may only be able to attribute the case of death as 'unascertained' or 'ill-defined'. Deaths in these categories are discussed more fully in Chapter 5.

Figure 3.6 shows the proportion of deaths in the abstracts of mortality that have an unspecified or unascertained cause. In New South Wales and Victoria, it took a decade or so following the introduction of civil registration before the proportion of deaths for which a cause could not be attributed with satisfaction fell below 1 per cent. In Queensland this took longer, with high proportions of ill-defined or unspecified causes from 1859 to 1869, so that the proportion with cause unknown did not fall below 1 per cent until 1880.

Figure 3.6: Annual percentage of deaths with an unspecified or unascertained cause, Australian colonies, 1853–1906
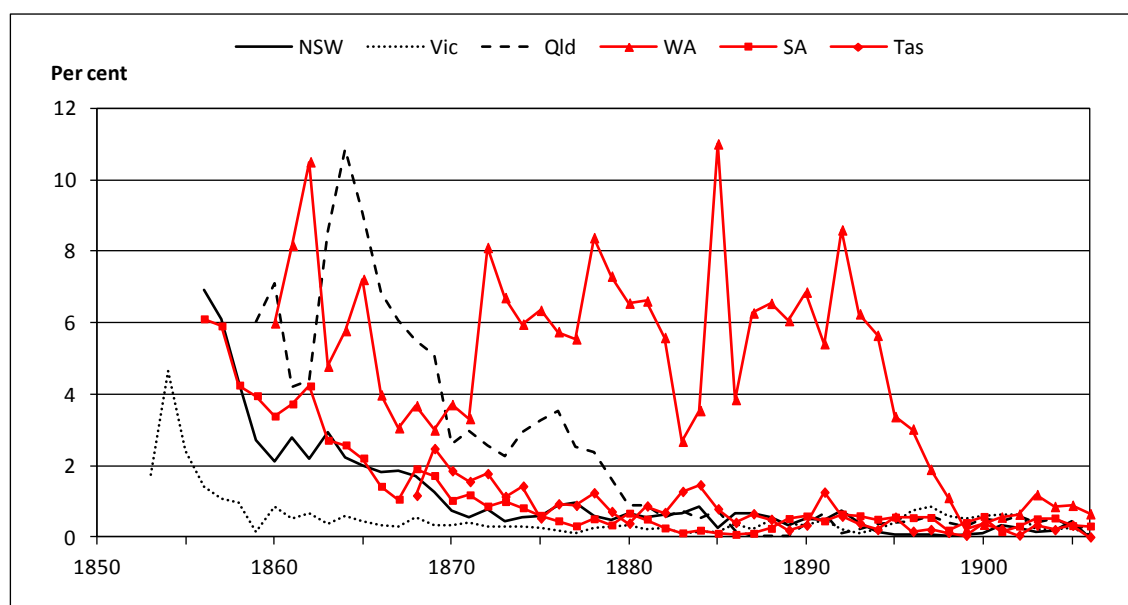
Western Australia had large proportions of unspecified or ill-defined deaths for most of the period from 1860 until 1897. In South Australia, the proportion fell below one per cent in 1872, and in Tasmania in 1875, although occasionally (1878, 1883, 1884 and 1891) it rose slightly above this figure.

ACCURACY OF THE DEFINITION OF DEATH

The requirement that life and death be given formal definition so that vital events can be correctly recorded might seem obvious, but this was not at all clear in nineteenth-century Australia.

No definition of death, whether expressed in legal, cultural or clinical terms is to be found in the registration Acts or in other contemporary legislation. Nineteenth-century medical opinion was equivocal, placing most reliance on a number of symptoms to ascertain death, such as cessation of heartbeat and respiration,

> 'It is not always easy to determine when the spark of life has been finally extinguished…The conditions most resembling actual death are syncope, asphyxia, and trance, particularly the last. We cannot, however, say that any infallible criterion applicable by the vulgar has been discovered, and we do not rely exclusively on any one sign, but combine several' (Ferrier, 1885, p.327).

Regarding the registration of a death, much of the ambiguity bore on how to count live births, and stillbirths (or fetal deaths). Currently, a live birth is the birth of a child who, after delivery, breathes or shows any other evidence of life such as a heartbeat. Death is the permanent disappearance of all evidence of life after birth has taken place, and this definition excludes all deaths prior to live birth (ABS, 2010). Although the term 'stillbirth' was used freely in colonial times, less precision was attached to its meaning.

The uncertainty was typified by the concern that surrounded the intentional misreporting of infant deaths as stillbirths by midwives and other birth attendants. A Select Committee appointed by the New South Wales Legislative Council in 1886 found that the absence of registration of stillbirths made provision for the possibility of 'crimes against the lives of young children'. Some cemetery authorities required certificates from doctors or midwives that the body of the child to be buried was stillborn, but this was uncommon and the certificates were deemed to be of little value 'as a precaution against the concealment of intentional foul play' (*AMG*, Dec. 1886, p.72–73; Dec. 1887, p.73).

Some 2,282 stillbirths (or around two per cent of all births) appear in the Western Australian historic indexes between 1841 and 1906, with most of these registered after the late 1890s. Stillbirths in Western Australia were, in all likelihood, under-registered in the 1870s and 1880s, and over-registered in the 1890s, due to a changed interpretation of what a 'stillbirth' and a 'premature birth' was (Durey, 1980, 1982). In this work, all entries in the historic indexes termed 'stillbirths' have been removed from analysis. In Tasmania, the other colony with available information, only 179 stillbirths were registered between 1838 and 1899, and these were also removed before analysis (Kippen, 2002a).

A number of deaths which were termed stillbirths may in fact be infant deaths. This will have resulted in a very small underestimate of infant mortality in the abstracts of mortality. In 1907, Western Australia became the first State to introduce compulsory registration, with stillbirths registered as both births and deaths (7 Edw. VII No. 19).

ACCURACY OF RECORDING OF SEX, AGE AND CAUSE OF DEATH

Having discussed their completeness, what can be said about the accuracy of these data? Is there reason to believe that the details of sex, age and cause of death were reported incorrectly, and how can this be known?

Regarding the sex of individuals, problems can occur with simple recording or transcription errors or with deliberate misreporting. Doctors or Assistant Registrars may enter the wrong sex. Confusion might also arise over non-English or unfamiliar names. Because the abstracts of mortality are summaries of individual records, there is no provision to check for these types of errors, but there is little reason to believe that misreporting of sex, whether accidental or deliberate, is anything but negligible.

The misreporting of age at death gives cause for greater scrutiny. Measurement techniques have been developed for age data, prompted largely by the need for actuaries to identify errors and correct reported data for the construction of life tables.

Digit preference or 'age heaping' is the tendency to report certain ages over others, with a preference for ages ending in '0' or '5' being most common. It is the principal type of error in single-year-of-age data, but since the abstracts of mortality do not report single years of age, there is no way of verifying whether they were subject to digit preference. The Western Australian historic indexes, however, do report single years of age at death.

Deaths in Western Australia occurring between 1841 and 1905, and reported for each year of age between 20 and 80, are presented in Figure 3.7. Digit preference for years ending in 0 or 5 is apparent for males, and to a lesser extent for females, from ages 30 to 70. Ages at death of 30, 35, 40, 45, 50 or 60 were most commonly reported.

Figure 3.7: Age pyramid of deaths in Western Australia, 1841–1905
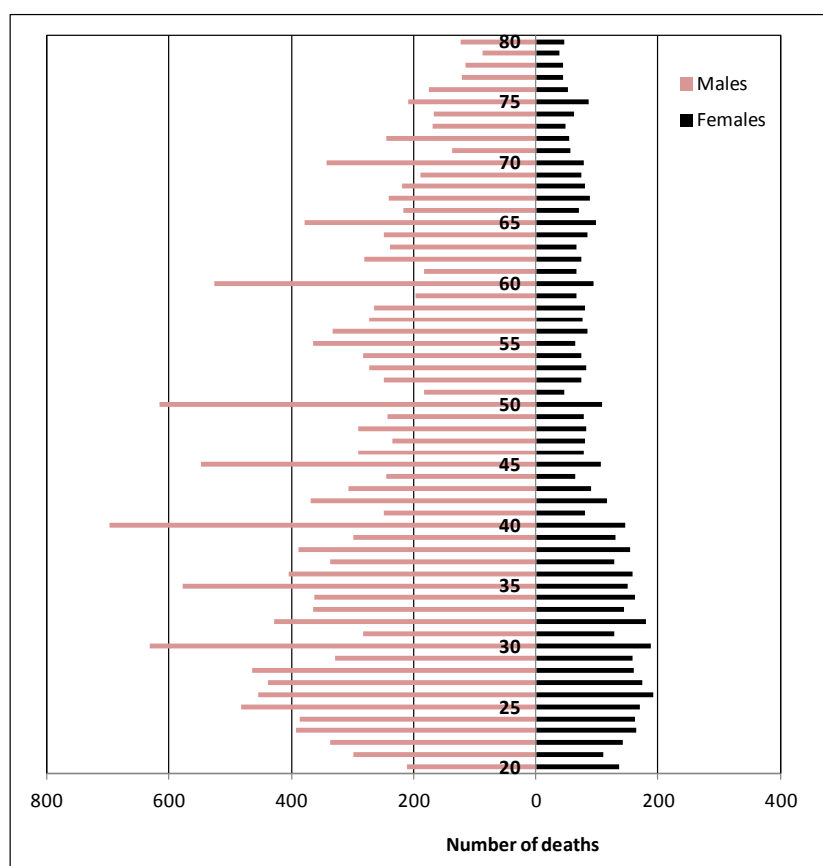
Why should age at death be so poorly reported in Western Australia? The ages of the deceased were usually given to Deputy Registrars or doctors by the next-of-kin, who may be ill-informed, or may simply guess. Some males may have

been single, with no next-of-kin. Most of the persons dying during this time period would not have had birth certificates, and even if these did exist, it would not necessarily mean that age at death would be remembered or recorded precisely (Woods, 2000). Shryock & Siegel (1976) noted that digit preference is more common among populations with lower educational status. The consolation is that these Western Australian data are from a smaller colony with a governmental registration and statistical system that was still undergoing development.

Using the *Tasdeaths* database, Kippen (2002b) showed that Tasmania also exhibited digit preference for years ending in 5, and even more so for those ending in 0. Ages implying a birth year of 1800 were especially preferred. Digit preference in Tasmania diminished over the second half of the nineteenth century.

The ignorance of household informants was perhaps greatest when reporting the deaths of those at an advanced age of, say, 70 years and over. The tendency was to overstate older ages, either from the desire to attach some form of prestige through adding to the longevity of the deceased, or simply through lack of knowledge (Shryock & Siegel, 1976).

The proportion of persons attaining age 70 was still relatively small in the mid-nineteenth century. Mid-century, a cause of death of 'old age' was routinely returned for persons aged 60–64. Only 1.8 per cent of all registered male deaths in 1861 were at ages 70 and over; but by 1901 this had increased to 16.9 per cent.

Mindful of the problems raised by digit preference in registrations of death, English Registrar William Farr was wary of reporting data on enumerated populations by single years of age, preferring to use five- or ten-year age groups (Woods, 2000). The abstracts of mortality in the Australian colonies also report age at death in this fashion. Although grouping age data serves to reduce the error introduced by digit preference, some residual error may remain.

Reclassifying age groups

In some of the abstracts of mortality for Western Australia, South Australia and Tasmania, ages at death were tabulated using irregular age groups (Table 3.3). These are irregular in the sense that they depart from the five-year age groups adopted in this work for the purposes of age-standardisation and other analysis, namely ages 0, 1–4, 5–9…70–74, 75+.

Table 3.3: Irregular age groups in statistical abstracts of mortality

Colony/Years	Age groups
Western Australia	
1841	0, 1–4, 5–29, 30+
1842–1844	0–1, 2–4, 5–9, 10–19…50–59, 60+
1845–1847	0, 1–2, 3–9, 10–19…40–49, 50+
1848–1854	0–2, 3–9, 10–19, 20–29, 30–59, 60+
1860–1869	0, 1–2, 3–4, 5–14, 15–19, 20–29…50–59, 60+
1886–1896	0, 1–4, 5–9, 10–14, 15–19, 20–29, 30–39, 40–59, 60+
South Australia	
1850–1855	0–1, 2–4, 5–9, 10–29, 30–49, 50+
1868–1872	0–1, 2–4, 5–9, 10–29, 30–49, 50+
Tasmania	
1847	0–9, 10–19…60–69, 70+
1850–1854	0–9, 10–19…70–79, 80+
1855–1867	0, 1–4, 5–9, 10–19…70–79, 80+

Note: Regular age groups are 0, 1–4, 5–9...70–74, 75+

Deaths in these irregular age groups were redistributed into five-year age groups. Exponential functions were fitted to time series of existing five-year age groups to provide estimates for each age group for the years in question. Irregular ten- or twenty-year age segments provide the boundaries for the numbers of deaths to be redistributed.

In some cases, deaths occurring at ages within the age range 0–4 needed redistribution into age groups 0 and 1–4 (Table 3.3). Karup-King coefficients

were used to split age groups into single years of age, which were then recombined to provide age groups 0 and 1–4 (see Appendix 1).

ACCURACY OF RECORDING OF TIME AND PLACE OF DEATH

Besides the sex, age and cause-of-death of the deceased, the abstracts of mortality often include information regarding the time of occurrence of death, and the place of death. These are also useful for mortality analysis.

Time of death

Deaths presented in the abstracts of mortality are those that were *registered* during the year in question, and not necessarily those that *occurred* during that year. Statistics based on year of registration may be lower or higher than those based on year of occurrence. Deaths occurring in a year may not be registered until the following year or later, and deaths registered during a year may include those occurring in earlier years.

There is usually a lag between the occurrence and registration of a death. This can be caused by a delay either in submitting a completed form to the registry, or a delay by the registry in processing the death. Coroner's inquests may delay the registration of a death, or a death in the outback may not be discovered until some years have passed. A full count of deaths by year of occurrence can take many years to finalise.

Some insight into these differences can be gained by comparing the counts in the Western Australian abstracts of mortality, based on year of registration, with the online historic indexes which are based on year of occurrence (Figure 3.8). The ratio varies around one, which would equate the number of deaths occurring in a given year to the number of deaths registered in that year. In Western Australia, annual differences were usually within the range of plus or minus five per cent, a figure not dissimilar from current practice (ABS, 2010).

Figure 3.8: Ratio of deaths classified by year of occurrence to year of registration, Western Australia, 1855–1905
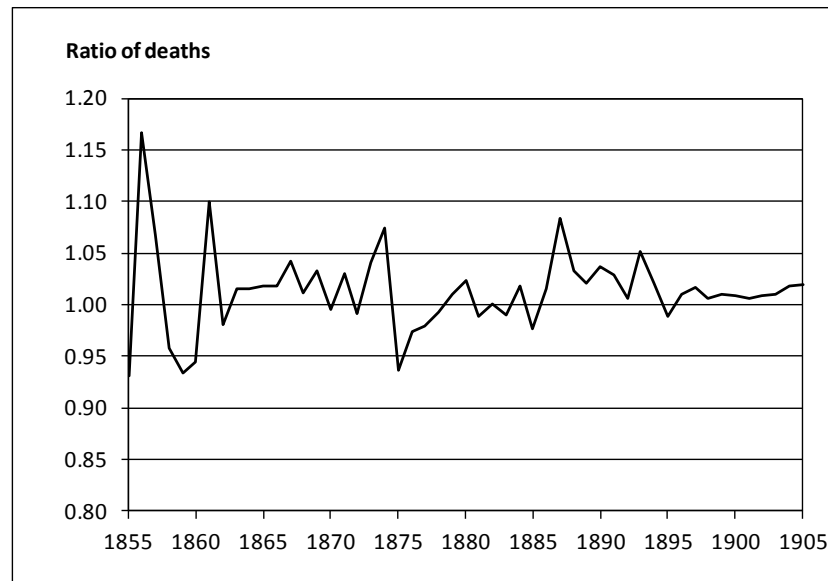
In some of the earliest abstracts of mortality, deaths are presented for the balance of the year in which the Registration Act commenced. The first annual report for New South Wales covers the period 1 March–31 December 1856. In such cases, estimates are made for the entire year, with an allowance if needed for seasonality of death, based on the following year.

Place of death

Geographic location of death is also of interest. Spatial disparities in deaths give insight into the epidemiological processes which occur in different places, and these constitute an important determinant of mortality. Overcrowding of populations in large cities, for example, allows certain epidemic diseases to spread with ease whereas the sparse population of outback areas can act to halt the same epidemic. Geographic allocation of deaths by city and suburb, urban and rural region, or across colonies enables an examination of spatial disparities in mortality.

Both the place of residence and the place at which death occurred were usually recorded on death certificates. Difficulties arise when the place of occurrence

differs from the usual place of residence, because the context in which people live, work, grow ill and die is misrepresented.

As the provision of health care facilities increased over the latter part of the nineteenth century, fewer deaths occurred at home. By 1880, one-in-five (21 per cent) of all deaths registered in Melbourne and suburbs took place in public institutions such as hospitals and charitable homes (Hayter, 1881a). Urban residents, but also people from suburban and country areas sought care in metropolitan hospitals, resulting in an excessive allocation of deaths to these large cities. Deaths by accident and deaths at sea might also occur away from place of usual residence.

Methods are available for redistributing deaths that occur in institutions according to the usual place of residence (Mooney *et al.*, 1999). These require detailed patient or inmate information which is not found in the abstracts of mortality, and this type of redistribution is not attempted here. Hence, a proportion of deaths presented by place in the abstracts of mortality may not reflect the place of usual residence.

CONSTRUCTING POPULATION ESTIMATES

To calculate age-specific mortality rates, annual estimates of the population at risk are required by sex for each age group 0, 1–4, 5–9…70–74, 75+, and for each year to 1906 in which deaths were registered. For most of the years between 1850 and 1906, age-specific population estimates do not exist, and so must be calculated, guided by available population information.

Annual population estimates

Official annual population estimates by sex for each colony and State are available, for 31 December for 1788 onward, and for 30 June from 1901 (ABS, 2008a). These estimates are based on the colonial censuses which were undertaken periodically throughout the second half of the nineteenth century, and into the twentieth century (Table 3.4). Mid-year population estimates from 1850 to 1900 were derived from the ABS 31 December estimates.

Table 3.4: Colonial censuses 1844 to 1891, and Commonwealth censuses, 1901 to 1911

New South Wales	Victoria	Queensland	Western Australia	South Australia	Tasmania
				26 Feb 1844	
				26 Feb 1846	
					31 Dec 1847
			10 Oct 1848		
				1 Jan 1851	
1 Mar 1851					1 Mar 1851
	26 Apr 1854				
				31 Mar 1855	
1 Mar 1856		1 Mar 1856[a]			
	29 Mar 1857				31 Mar 1857
			31 Dec 1859		
				1 Apr 1860	
7 Apr 1861	7 Apr 1861	7 Apr 1861	7 Apr 1861	7 Apr 1861	7 Apr 1861
		1 Jan 1864			
				26 Mar 1866	
		2 Mar 1868			
					7 Feb 1870
			31 Mar 1870		
2 Apr 1871	2 Apr 1871	2 Apr 1871		2 Apr 1871	
				26 Mar 1876	
		1 May 1876			
3 Apr 1881	3 Apr 1881	3 Apr 1881	3 Apr 1881	3 Apr 1881	3 Apr 1881
		1 May 1886			
5 Apr 1891	5 Apr 1891	5 Apr 1891	5 Apr 1891	5 Apr 1891	5 Apr 1891
31 Mar 1901	31 Mar 1901	31 Mar 1901	31 Mar 1901	31 Mar 1901	31 Mar 1901
3 Apr 1911	3 Apr 1911	3 Apr 1911	3 Apr 1911	3 Apr 1911	3 Apr 1911

(a) Part of New South Wales.

Age distributions

The ABS total population estimates have no information on the age distribution of the colonial populations. The colonial censuses do, and provide age for the years in which they were conducted (Caldwell, in Vamplew, 1987; ABS, 2008a; HCCDA, 2014). These provide the information needed for calculations of population age distributions across the entire time period 1850–1906.

The age groups included in published census results usually conform to the required 5-year structure, although non-standard groupings were used in some of the earlier censuses (Table 3.5).

Table 3.5: Age groups published in colonial and Commonwealth census results, 1844 to 1911

Colony /State	Census year	Age groups
NSW	1851	0–1, 2–6, 7–13, 14–20, 21–44, 45–59, 60+
	1856	0–1, 2–3, 4–6, 7–13, 14–20, 21–44, 45–59, 60+
Vic	1854, 1857	0–4, 5–9...75–79, 80+
Qld	1856	0–1, 2–3, 4–6, 7–13, 14–20, 21–44, 45–59, 60+
	1864	0, 1–4, 5–9...75–79, 80+
	1868	0–4, 5–9...75–79, 80+
	1876, 1886	0–4, 5–9…55–59, 60–69, 70–79, 80–89, 90–99, 100+
WA	1848	0–2, 3–13, 14–59, 60+
	1859	0, 1–4, 5–14, 15–20, 21–29, 30–39...70–79, 80+
SA	1844, 1846, 1851, 1855, 1860	0–1, 2–6, 7–13, 14–20, 21–44, 45–59, 60+
	1866, 1876	0, 1, 2...14, 15–19, 20, 21–24...75–79, 80+
Tas	1847, 1851, 1857	0–1, 2–6, 7–13, 14–20, 21–44, 45–59, 60+
All colonies	1861, 1871, 1881, 1891	0–4, 5–9...75–79, 80+
	1901, 1911	0–4, 5–9...75–79, 80–84, 85+

A number of adjustments were made to the age distributions obtained from published census results, as follows:

- Unspecified ages were redistributed, according to the proportions of known age.

- Some census results were published by single years of age—1891 and 1901 for New South Wales, Western Australia and Tasmania; 1876, 1881, 1891 and 1901 for South Australia—and these show evidence of digit preference. Corrections were made by smoothing, using the Karup-King Newton method for each sex across ages 10–69 years (see Appendix 1). Populations published in the Victorian census reports from 1857 onwards had already undergone smoothing (see for example, Hayter, 1881b, p.35).

- Regression curves were fitted to those censuses with standard age groups, and these were used to derive estimates for each 5-year age group for censuses with non-standard age groups.

- The census estimates by 5-year age groups were linearly interpolated across intercensal years.

- Estimated populations for each 5-year age group were then calculated by pro-rating the ABS total population estimates by the calculated age-specific proportions.

Excluded populations

Information about Aborigines was not collected consistently in colonial censuses. There was considerable variation among colonies in their efforts to count the native population, but also inconsistencies within each colony from one census to the next (Camm, 1988).

Generally, Aboriginal people frequenting the districts settled by Europeans, or employed by colonists, or superintended on reserves were enumerated, but they were included in only some of the published population tabulations. They were often included in New South Wales and Victorian census tables, in some of the Western Australian tables, but were excluded from South Australian tables. In Queensland, they were not counted at all.

Occasionally, estimates of the total number of Aboriginals, including the so-called 'roving Aborigines' were published, usually with the qualification that the counts should be considered highly imperfect (Hayter, 1881b, p.16). Again, these estimates were excluded from census tabulations. Considered as a whole, neither Aboriginal deaths nor populations were counted consistently. All that can be said in consequence of the inconsistency in counting Aborigines is that in each colony there will be a small numerator-denominator discrepancy.

The censuses took better account of Chinese persons and other 'aliens', with special efforts made to count these populations in Victoria and Queensland, through employing Chinese translators and sub-enumerators. The Registrar-General's Report for the Queensland census of 1871 specified that

> 'Chinamen, Malays, Polynesians and other foreigners, of whatever nation or color, are to be counted; but the native blacks (aboriginals of Australia) are not to be included' (Scott, 1872).

The Tasmanian total population estimates, based on census results, include counts of both convict and free populations (ABS, 1997). Since only deaths of free persons were included in the abstracts of mortality, with convict deaths registered separately, that colony's population must be adjusted accordingly to exclude convicts. Newman (2005) provides estimates of the convict population as a proportion of the total Tasmanian population, ranging from 34.4 per cent in 1847 to 3.7 per cent in 1857, and these estimates were used to adjust the Tasmanian population aged 20 years and over.

It is known that nineteenth-century censuses were subject to under-counting (Lee & Lam, 1983; Camm, 1988). Shortfalls occurred in population estimates based on the census, relative to those calculated by Registrar-General's departments, largely due to the inability to monitor overland migration between the colonies (Hayter, 1893). Although these census undercounts should not affect the ABS total population estimates, they may affect the age distributions of the population if the undercount was not uniform across all age groups.

CALCULATING MORTALITY RATES

Annual deaths and mid-year population estimates by five-year age groups allow for the calculation of age-specific mortality rates by sex and for the total population. Rates were calculated for each colony and for Australia through aggregating each colony's deaths and population estimates.

To compare these mortality rates over time or from area to area, the effects of differences in age structures of populations were eliminated through direct age-standardisation (Shryock & Siegel, 1976) (see Appendix 1).

SUMMARY

Commencing in Van Diemen's Land in 1839, summary information on registered deaths were compiled by Registrars-General and Government Statists and were tabulated in abstracts of mortality. These abstracts, published in annual reports of statistics and in other government publications, make

possible the calculation of rates of mortality and of causes of death in each colony from the mid-nineteenth century onwards. Analysis covering the whole of Australia dates from 1856 which was the year in which New South Wales, the final colony to introduce civil registration, began to publish mortality statistics.

Although it took some years for each colony to establish fully functioning systems of civil registration, the Registrars-General were of the opinion that registration of death soon reached near-complete levels, largely because of the legal prescription that a burial required a completed death certificate. The greatest doubts as to coverage surrounded deaths occurring in remote areas, among infants, and among Aboriginal and other non-European peoples.

The usefulness of information obtained from administrative death registrations depends on its completeness and accuracy. A proportion of records lacked sufficient information on sex, age or cause of death, either through omission or through errors in recording of detail, such as digit preference. Published information from Western Australia was most problematic, with the lack of detail in the abstracts of mortality for earlier years requiring that an alternative data source be used for this study.

Some of the data published on age of death required reclassification so that mortality rates could be calculated in a standard fashion across all colonies. Population estimates by sex and age group were then constructed for each year from 1850 to 1906. The resultant death and population estimates enable the calculation of age-specific and age-standardised mortality rates and other vital statistics.

Chapter 4

All-cause mortality, 1856–1906

Examining total mortality—deaths from all causes combined—is the first step in surveying the epidemiological history of nineteenth-century Australia. Calculating age-specific and age-standardised death rates for each of the six colonies will allow, for the first time, the course of all-cause mortality in Australia to be followed for the period from 1856 to 1906. Adding life table analysis will reveal the extent to which life expectancy increased or decreased over the period as a result of changes in mortality. These changes, however, were not uniform. Differential mortality between colonies, but also within urban and rural areas and by season, can provide an indication of the variable impact that environmental and other determinants had on health.

By first examining all-cause mortality without reference to specific causes of death, the problem of disease substitution is avoided, as certain disease-specific trends may appear quite different from those for all-cause mortality (Taylor, Lewis & Powles, 1998a). A considered explanation of trends in all-cause mortality, however, will need to weigh the relative contribution of individual causes of death, and this follows in Chapters 5 and 6.

The all-cause mortality rate in the second half of the nineteenth century was five times higher than today. With no recourse to antibiotics, and with few effective medical therapies, epidemics of infectious disease were feared. One-in-ten infants died before their first birthday, and the expectation of life at birth

was less than 60 years of age. Colonial life was shaped by the greater nearness of death.

ALL-CAUSE MORTALITY IN THE COLONIES

Age-standardised mortality rates for males and females in each colony during the period are shown in Figure 4.1. Although rates differ, certain broad similarities are apparent. All colonies exhibit large annual fluctuations in mortality among both males and females which typify a period dominated by recurrent epidemics of infectious disease. With the exception of Western Australia, a downward trend in mortality rates began in the 1880s. By 1906 rates in each colony had reached around 1,000 deaths per 100,000 population.

In New South Wales, death rates fluctuated between 1,500 and 2,000 deaths per 100,000 population from 1856 to the mid-1880s, with a notable fall and then a rise during the period 1867–1875. From the mid-1880s rates declined steadily although with some annual fluctuation, so that by 1906, the male rate was 978 and female 826 deaths per 100,000 population.

During the gold rush years of the 1850s, Victoria's death rates were high at over 2,000 deaths per 100,000 population, and they varied between 1,500 and 2,000 for most of the 1860s and 1870s. Unlike New South Wales, male and female death rates in Victoria increased during the 1880s, and declined from late in the decade, so that by 1906 rates stood at around 1,000 deaths per 100,000 population.

Death rates were higher in Queensland than in any other Australian colony between 1860 and 1885, although considerable fluctuations occurred. Male rates were in excess of 2,500 deaths per 100,000 population in 1863, 1866 and 1884, and in 1875 reached close to 3,000. As in New South Wales, a trough is apparent from the mid-1860s to the mid-1870s. From the mid-1880s rates began to decline, and by 1906 were at levels similar to those in New South Wales and Victoria. Among all the colonies, male-female disparities in mortality rates were greatest in Queensland.

Figure 4.1: Age-standardised mortality rates, by colony and sex, 1850–1906
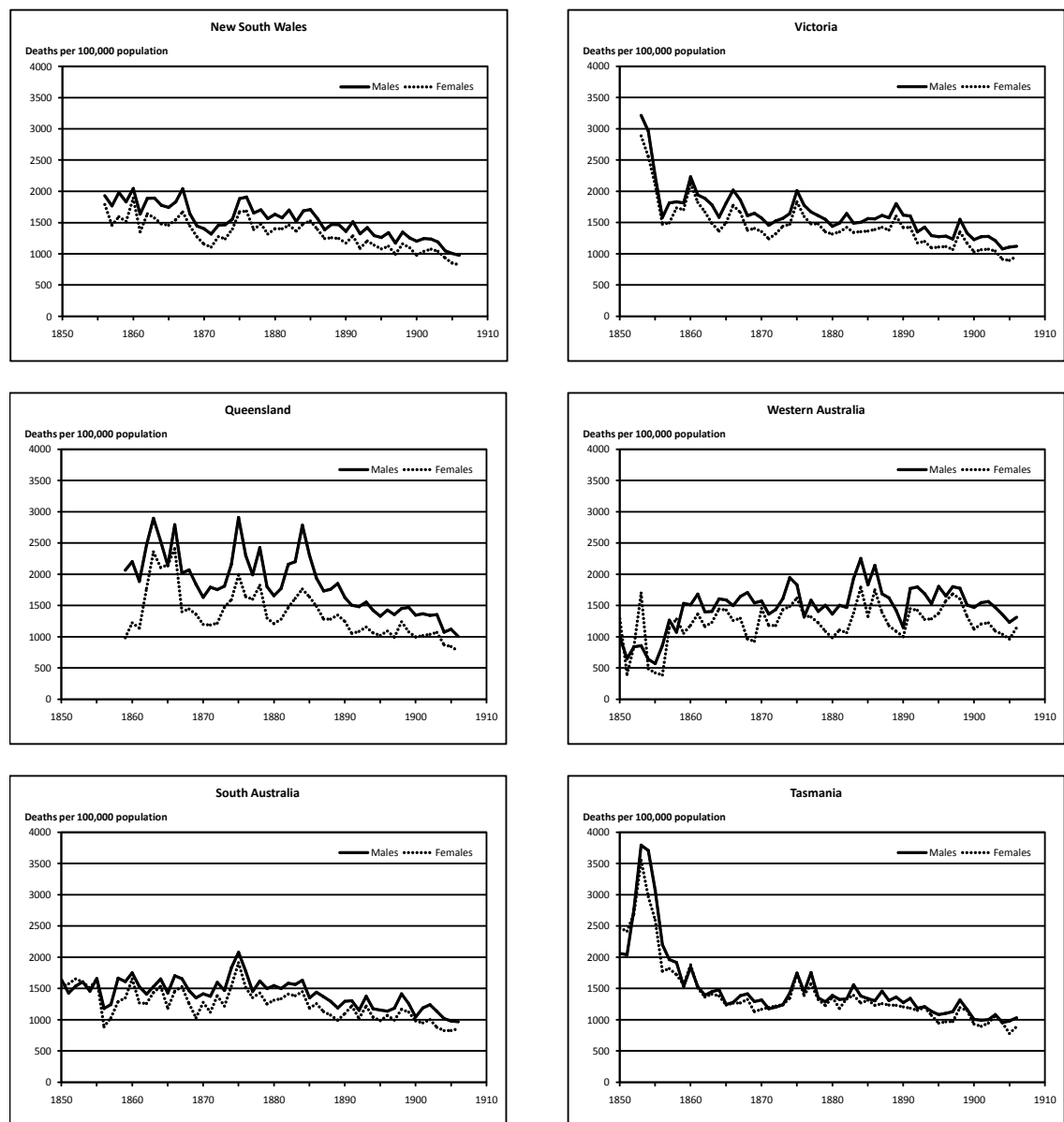
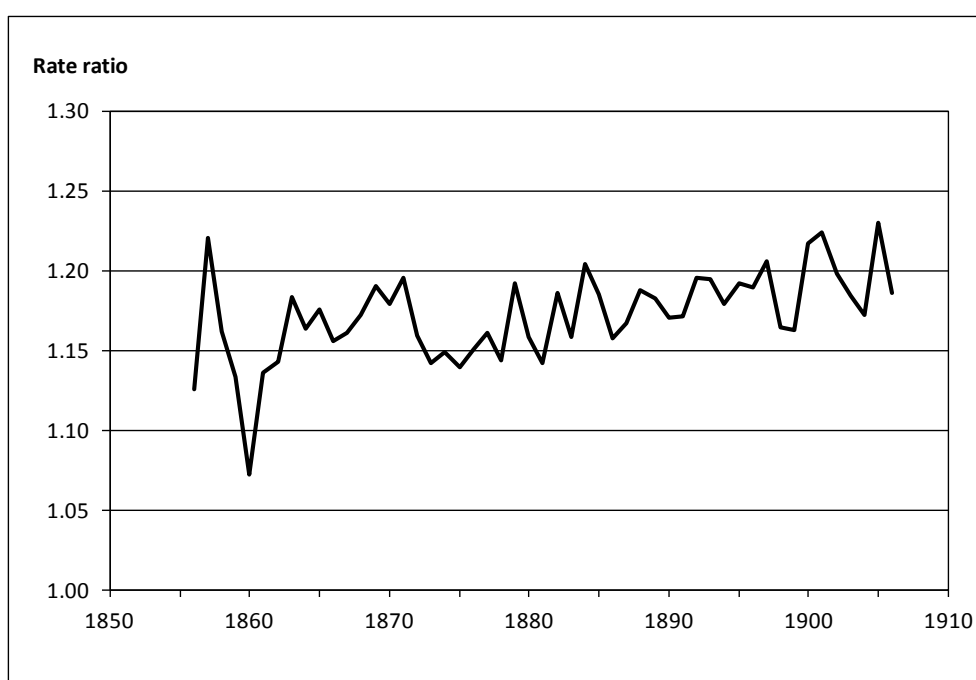*Note:* Mortality rates are age-standardised to the 1881 total Australian census population.

Western Australia was atypical, with a rise in mortality rates from 1850 to 1860, and then with little net change until the late 1890s, although there were annual fluctuations. Mortality remained close to 1,700 deaths per 100,000 population for males, and between 1,000 and 1,500 for females. After 1897, both male and female rates declined.

Death rates in South Australia showed little net change between 1850 and the mid-1880s, at around 1,500 per 100,000 population for males and slightly less

for females. The year 1875 was an exception, with a sharp rise in both male and female mortality. Rates declined after 1884 so that by 1906 they stood at 1,000 deaths per 100,000 population for males and 900 for females.

Mortality in Tasmania was high during the 1850s at over 3,500 deaths per 100,000 population. Rates declined until the early 1860s, and then remained steady to the late 1880s at around 1,300, apart from two spikes in 1875 and 1877. The decline resumed, and by 1906 mortality had fallen to 1,000 deaths per 100,000 population, a rate similar to other colonies.

ALL-CAUSE MORTALITY IN AUSTRALIA

Reflecting the similarity of fluctuations in the individual colonies, the annual death rate in Australia also varied considerably between 1856 and the early 1880s (Figure 4.2).

Figure 4.2: Age-standardised mortality rates by sex, Australia, 1856–1906
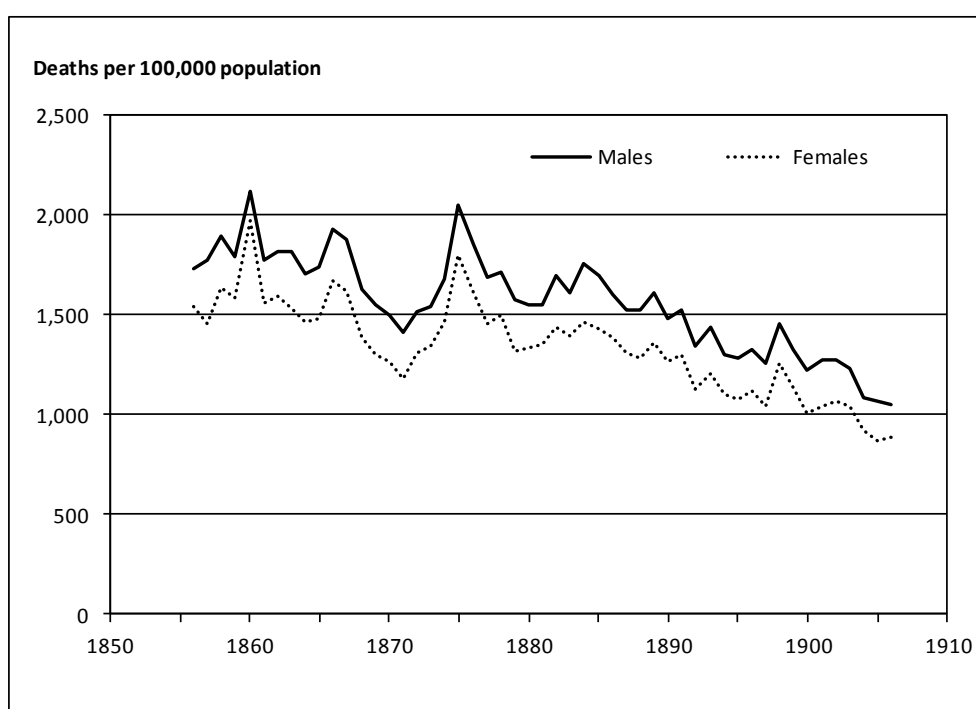
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

For the total population, age-standardised mortality stood at 1,657 deaths per 100,000 population in 1856, reaching a peak of 2,059 in 1860. Although

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exhibiting annual rises and falls, the rate showed a net decline during the decade of the 1860s, and by 1871 mortality had fallen to 1,313. Over the next four years, mortality rose rapidly to reach 1,945 in 1875, falling again to settle at 1,450 by 1880, and 1,580 deaths per 100,000 population in 1885.

From the mid-1880s, mortality declined steadily for both sexes, with less annual variation than in previous decades, and by 1906 rates for males and females had fallen to around 1,000 deaths per 100,000 population.

Male mortality was higher than female at all times. Generally, male rates were 15–20 per cent higher, although in 1860, an epidemic year, the disparity was reduced to only 7 per cent (Figure 4.3).

Figure 4.3: Mortality sex ratio, Australia, 1856–1906

Note: Mortality rates are age-standardised to the 1881 total Australian census population.

From around 1875 the male:female mortality ratio began to increase, and by the early 1900s male rates were 20 per cent higher than female rates. This disparity would persist and grow over the course of the twentieth century to reach 70 per cent by the 1980s, after which the ratio fell to reach its current level of 50 per cent (AIHW, 2014).

These calculations for the years 1856–1906 can now be added to existing information from 1907 onward, to provide an uninterrupted Australian mortality series from the mid-nineteenth century to the present day (Figure 4.4).

Figure 4.4: Age-standardised mortality rate, Australia, 1856–2010
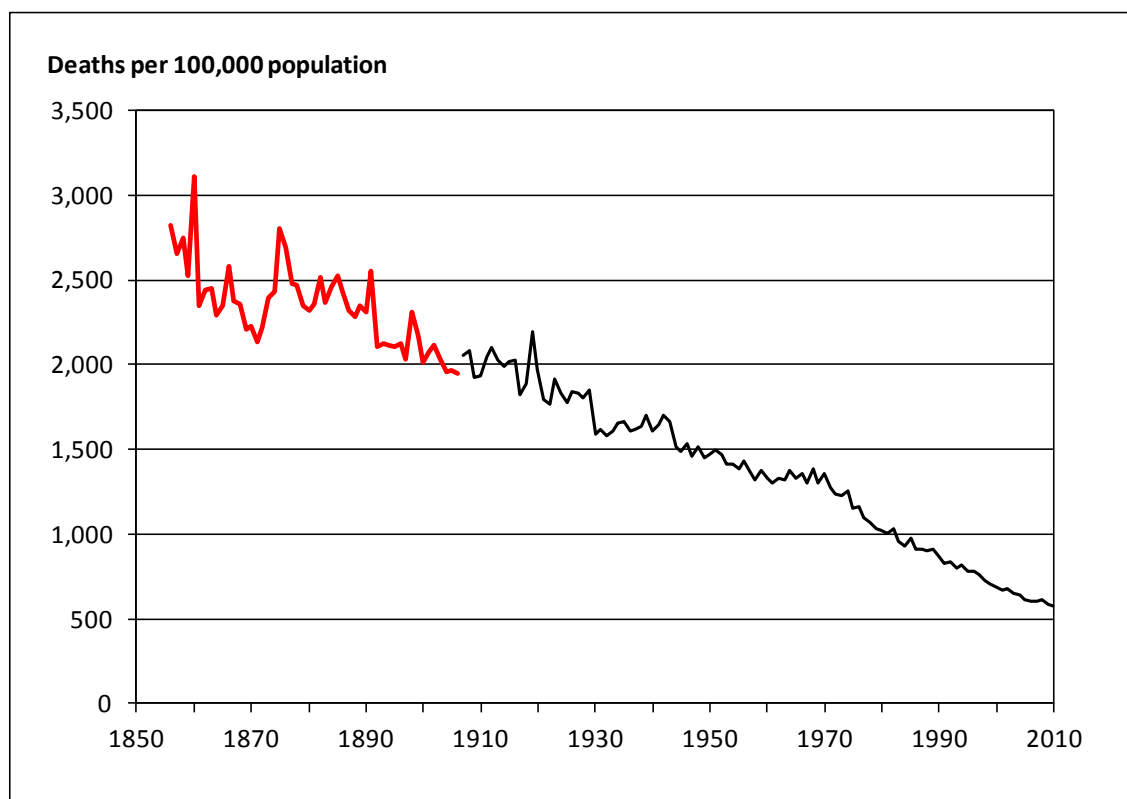
Note: Mortality rates are age-standardised to the 2001 Australian census population.

Source: AIHW (2014), from 1907 onward

Between 1856 and the early 1900s, mortality was high, and showed significant annual variation. From the mid-1880s, the rate began to decline, with the pace of change slowing after 1910, although mortality spiked for one last time during the great influenza pandemic of 1919. After 1930 mortality plateaued, and again from the 1950s, remaining largely unchanged until 1970, when a steep decline commenced that continues to the present day (Taylor, Lewis & Powles, 1998a).

The reduction in mortality that occurred over the two decades from the mid-1880s to mid-1900s was unique. There were prior falls, such as those from 1866–1871 and again from 1875–1880, but these were temporary, and were soon reversed by recurring epidemics of disease.

But the rate of decline that began in the mid-1880s, although occasionally tempered by a year in which mortality rose, was consistent. In little more than two decades—from 1885 to 1906—mortality declined by almost one-quarter (22.9 per cent), falling from 2,522 to 1,945 deaths per 100,000 population. Such reductions would not be seen again in Australia until the 1970s.

AGE-SPECIFIC MORTALITY RATES

Three years—1860, 1880 and 1900—are selected to illustrate the patterns of age-specific mortality in Australia throughout the second half of the nineteenth century (Figure 4.5).

During the period, rates of death in infancy were high at over 10,000 per 100,000 population—more than one-in-ten infants died before their first birthday. Mortality rates declined sharply throughout childhood years, and were lowest at age 10–14, at below 500 deaths per 100,000 population.

Figure 4.5: Age-specific mortality rates, Australia, 1860, 1880 and 1900
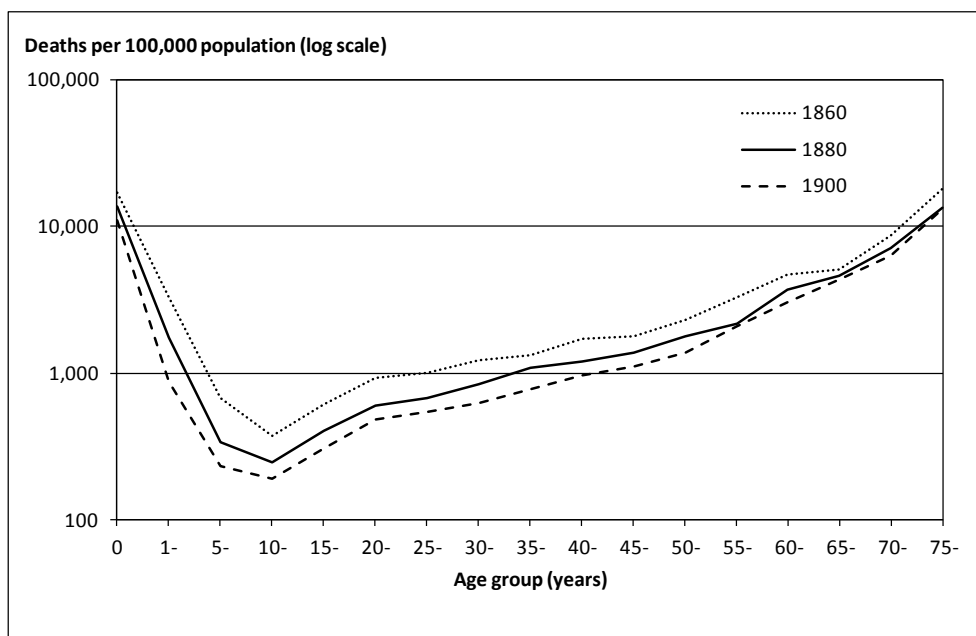
From age 10–14 years, mortality rates increased; rapidly until age 20–24 years, and then through to middle-age of 40–44 years, reaching around 1,000 deaths per 100,000 population. After age 45 years, age-specific rates again increased

rapidly until old-age at 75 years and over, where they reached similar values to those in infancy, at over 10,000 deaths per 100,000 population.

Age-specific mortality rates for each age group exhibit net declines from 1860 to 1880, and again from 1880 to 1900 (Figure 4.5).

Figure 4.6: Selected age-specific mortality rates, Australia, 1856–1906
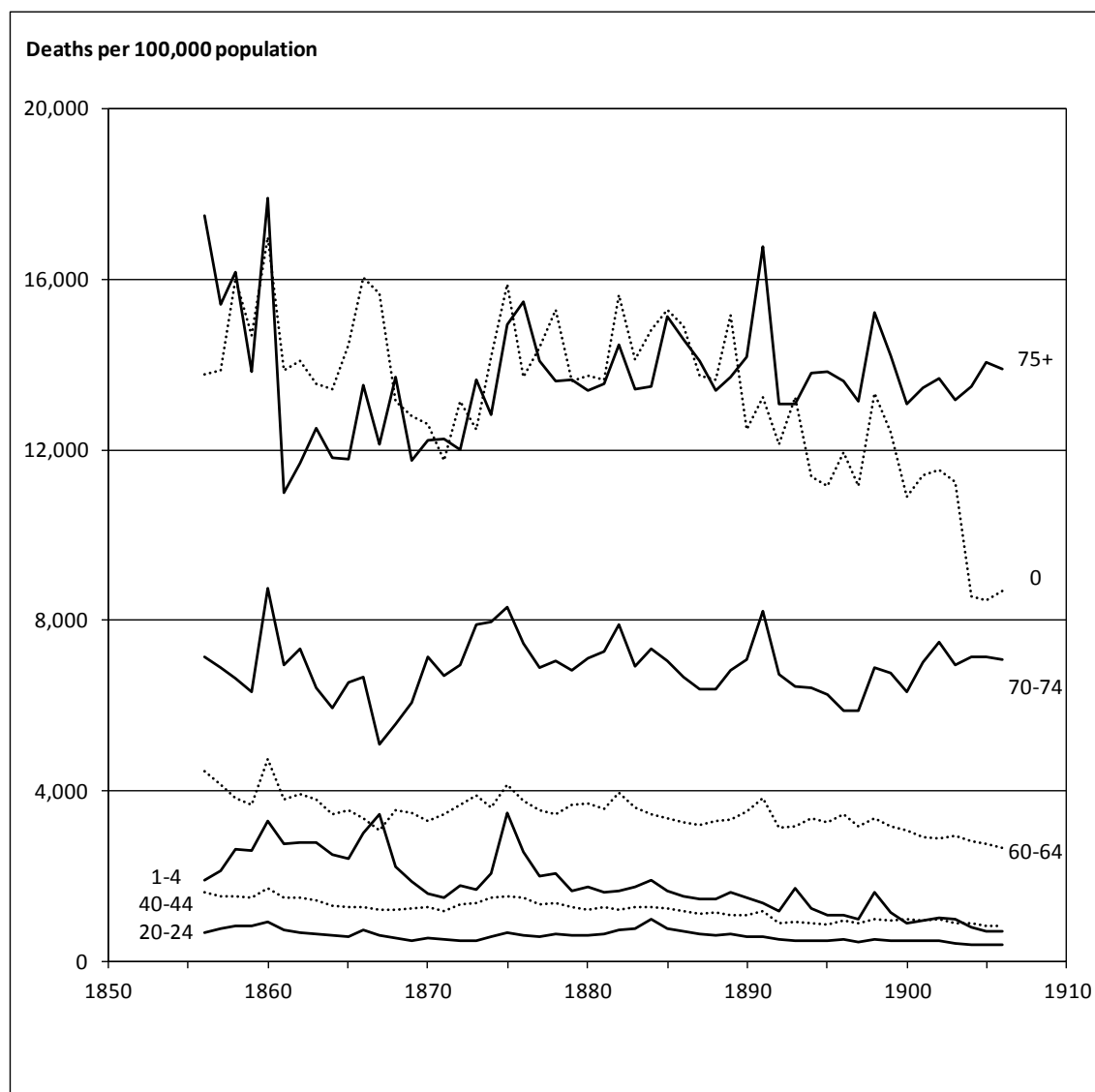
Trends in age-specific rates are shown in Figure 4.6. The largest declines during the fifty years from 1856 to 1906 were among younger age groups, with a prominent fall among infants from the mid-1880s. Between 1885 and 1905, mortality at age 0 fell by 45 per cent from 15,289 to 8,472 deaths per 100,000

population. A particularly large fall took place between 1903 and 1904, from 11,253 to 8,573 deaths.

Both these age groups—and to a lesser extent the older age groups of 70–74 years and 75 years and over—exhibit a trough in mortality that occurred from 1867–1875. The youngest and oldest age groups, age 0 and age 75 years and over, have similar mortality rates between 1855 and 1890.

Ages 20–24 years, 40–44 years and 60–64 years also exhibited declines in mortality after the mid-1880s. The older ages of 70–74 years and 75 years and over underwent great annual fluctuations in earlier decades, but with little net change between 1856 and 1906. Both these age groups underwent a peak in mortality in 1891.

Over the course of the second half of the nineteenth century, mortality rates improved most among children, with a decline of two-thirds (66%) among ages 1–4 years and a halving (49%) among ages 5–9 years (Figure 4.7).

Figure 4.7: Improvement rate of age-specific mortality, Australia, 1856–60 to 1901–06
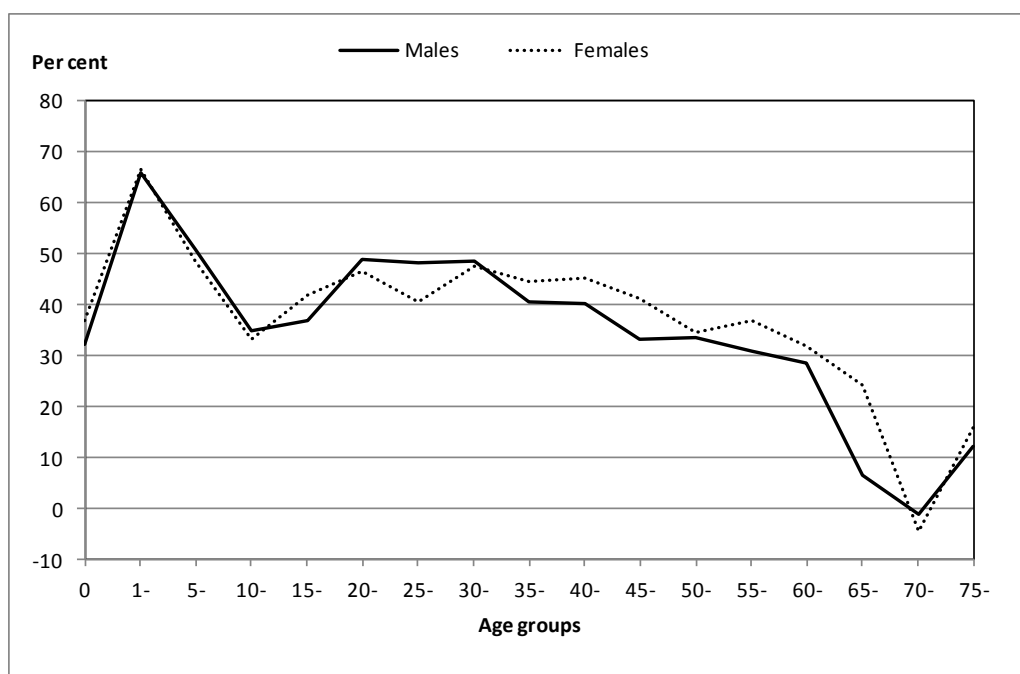
Although the improvement rate in mortality at age 0 was similar to that among young persons and adult age groups (declines of around 30–50%), the number of lives saved at age 0 far outweighed those in other age groups. Less

improvement was seen in mortality among older age groups, and for the age group 70–74 years, mortality rates in the early 1900's remained much the same as in the 1850's.

LIFE EXPECTANCY AT BIRTH

Another method of summarising the mortality experience of the Australian population is through a life table, a mathematical model which combines the mortality rates of a population at different ages to derive expectations of life at different ages (see Appendix 1).

The primary purpose in calculating life tables in this work is to measure the expectation of life at birth, since this is a widely recognised indicator of population health. Although life expectancy at birth reflects mortality at all ages, the high level of infant mortality throughout the period examined here bears heavily on the results. A low life expectancy is indicative of a high level of mortality, especially among infants.

The age-specific death rates derived from the abstracts of mortality allow for the construction of annual life tables for each colony, and thus also for Australia. Since age-specific rates are only available for five-year age groups, the life tables are abridged. As nineteenth-century mortality fluctuated considerably from year-to-year, abridged life tables have also been calculated for 10-year time periods to give more stable estimates.

Life expectancy in the colonies and in Australia

Life expectancies at birth by sex for decennial periods beginning in 1851, as derived from the calculated life tables, are presented in Table 4.1. For both New South Wales and Australia the commencing period is 1856–60, and for Victoria 1853–60. For each jurisdiction the final period is for the years 1901–06.

Table 4.1: Life expectancy at birth, colonies and Australia, by sex, 1851–60 to 1901–06

Sex / Colony	1851–60[a]	1861–70	1871–80	1881–90	1891–00	1901–06
Males						
New South Wales	43.07	45.17	46.74	48.01	51.62	55.15
Victoria	40.81	44.40	46.54	47.14	50.81	53.95
Queensland	42.03	39.74	40.69	41.52	49.54	53.42
Western Australia	n.a.	48.26	48.03	46.07	46.00	50.15
South Australia	49.55	49.53	46.93	49.80	53.25	55.63
Tasmania	38.58	50.45	50.14	50.74	54.11	57.17
Australia	**43.08**	**45.05**	**46.09**	**46.97**	**50.98**	**54.26**
Females						
New South Wales	46.27	49.16	49.97	51.01	55.20	58.22
Victoria	42.99	48.23	49.15	49.90	54.04	57.49
Queensland	n.a.	46.27	48.64	49.67	55.96	58.92
Western Australia	n.a.	53.78	52.02	51.86	49.95	55.65
South Australia	50.97	53.70	50.48	53.23	55.88	59.64
Tasmania	39.80	51.66	50.73	52.35	55.72	58.94
Australia	**46.00**	**48.91**	**49.50**	**50.70**	**54.78**	**58.02**

(a) 1856–60 for New South Wales and Australia, and 1853–60 for Victoria.

The probability of dying ($_nq_x$) for males and females in Australia are provided in Table 4.2, along with life expectancies at ages 0, 1, 5, 10 and 75.

Life expectancy at birth for males in Australia rose from 43.08 years in 1856–60 to 54.26 years in 1901–06, an addition of eleven years of life over five decades. For females, the increase was slightly higher, rising from 46.00 years in 1856–60 to 58.02 in 1901–06. For both males and females, most of the increase in years of life occurred from 1881–90 onward.

Table 4.2: Probability of dying ($_nq_x$) by sex, Australia, 1856–60 to 1901–06

| Age group	1856–60	1861–70	1871–80	1881–90	1891–00	1901–06
Males						
0	0.1440	0.1372	0.1359	0.1390	0.1201	0.1010
1	0.0989	0.0987	0.0809	0.0634	0.0488	0.0347
5	0.0235	0.0298	0.0259	0.0192	0.0154	0.0116
10	0.0156	0.0170	0.0161	0.0130	0.0112	0.0102
15	0.0245	0.0217	0.0228	0.0256	0.0178	0.0155
20	0.0429	0.0307	0.0295	0.0380	0.0263	0.0222
25	0.0484	0.0397	0.0342	0.0418	0.0325	0.0253
30	0.0563	0.0479	0.0430	0.0434	0.0360	0.0293
35	0.0629	0.0574	0.0534	0.0490	0.0433	0.0378
40	0.0783	0.0663	0.0693	0.0605	0.0499	0.0474
45	0.0872	0.0825	0.0818	0.0773	0.0624	0.0592
50	0.1127	0.1019	0.1020	0.1031	0.0840	0.0763
55	0.1422	0.1291	0.1262	0.1255	0.1142	0.1004
60	0.2014	0.1759	0.1840	0.1754	0.1699	0.1484
65	0.2306	0.2235	0.2340	0.2304	0.2237	0.2173
70	0.3247	0.2806	0.3296	0.3142	0.3028	0.3278
75	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
e_0	43.08	45.05	46.09	46.97	50.98	54.26
e_1	49.27	51.16	52.30	53.51	56.89	59.32
e_5	50.46	52.54	52.73	52.99	55.71	57.38
e_{10}	46.62	49.08	49.06	48.98	51.54	53.02
e_{75}	6.01	7.71	6.95	6.89	6.80	6.86
Females						
0	0.1294	0.1166	0.1158	0.1206	0.1024	0.0846
1	0.0957	0.0921	0.0762	0.0605	0.0466	0.0329
5	0.0204	0.0273	0.0243	0.0176	0.0146	0.0106
10	0.0136	0.0153	0.0148	0.0118	0.0097	0.0091
15	0.0245	0.0198	0.0207	0.0201	0.0154	0.0143
20	0.0354	0.0297	0.0275	0.0298	0.0223	0.0191
25	0.0397	0.0358	0.0360	0.0368	0.0287	0.0238
30	0.0536	0.0452	0.0428	0.0395	0.0345	0.0284
35	0.0628	0.0519	0.0528	0.0482	0.0410	0.0354
40	0.0703	0.0569	0.0595	0.0530	0.0409	0.0392
45	0.0738	0.0654	0.0624	0.0620	0.0482	0.0440
50	0.0804	0.0712	0.0738	0.0765	0.0621	0.0532
55	0.1149	0.0950	0.0976	0.0942	0.0853	0.0740
60	0.1597	0.1314	0.1435	0.1294	0.1266	0.1118
65	0.2184	0.1881	0.1872	0.1887	0.1677	0.1703
70	0.2593	0.2563	0.2728	0.2666	0.2494	0.2689
75	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
e_0	46.00	48.91	49.50	50.70	54.78	58.02
e_1	51.80	54.32	54.95	56.62	59.99	62.36
e_5	53.06	55.63	55.31	56.14	58.82	60.42
e_{10}	49.12	52.12	51.63	52.09	54.65	56.04
e_{75}	6.71	9.10	7.97	7.55	7.75	7.99

Life expectancy at birth for females always exceeded that for males, at an average of 3.6 years over the entire period 1856–1906. The sex gap was narrowest in the period 1856–60 (2.9 years), and widest in 1861–70 (3.9 years).

Among the colonies, life expectancy in 1851–60 was lowest in Tasmania, it being affected by very high mortality rates during those years (see Figure 4.1). However, Tasmania's position improved rapidly, so that by 1901–06 it enjoyed the highest life expectancy among all colonies for males, and second highest for females, behind South Australia.

Life expectancy at birth in Western Australia improved little over the half-century. It began to rise only from the 1890s, at a time when significant economic development accompanied the discovery of gold.

The annual fluctuations in life expectancy at birth for males and females in nineteenth-century Australia are apparent in Figure 4.8. Mirroring mortality, life expectancy rose, and then fell during the years 1867–1875. Sustained increases in life expectancy did not begin until the mid-1880s.

Figure 4.8: Life expectancy at birth, by sex, Australia, 1856–1906
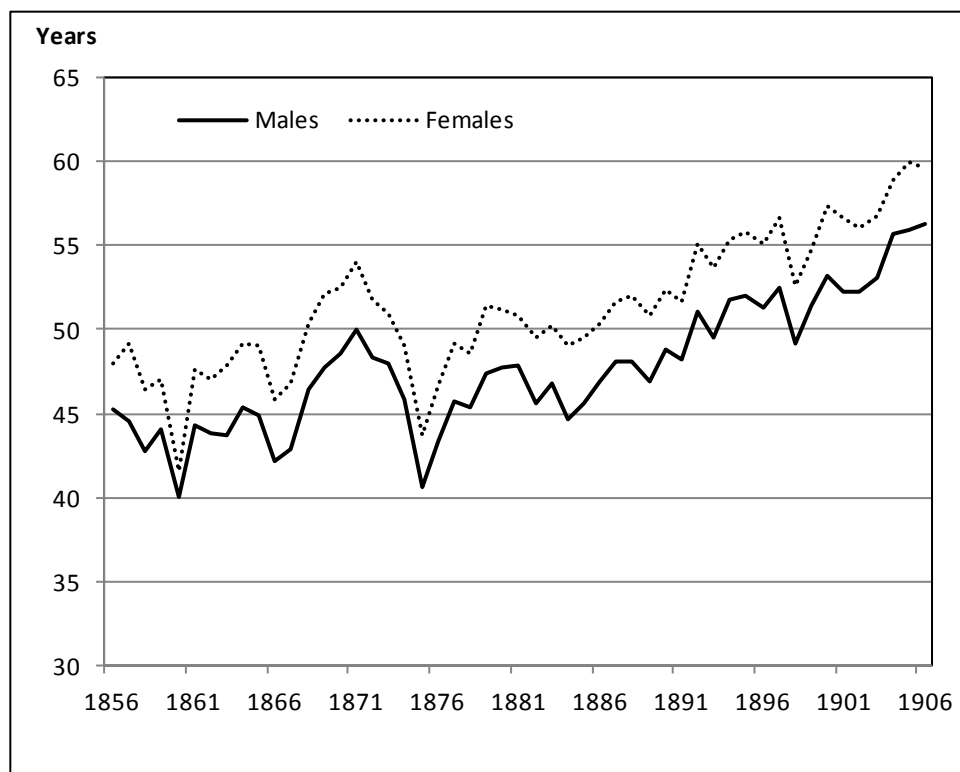
Comparison with previous estimates

The life expectancies calculated in this work are compared with previous estimates in Table 4.3. Professor M. B. Pell of Sydney University was the first to use the new statistical abstracts of mortality to estimate a life expectancy at birth of 45.58 years for the total New South Wales population for the years 1856–1866 (Lancaster, 1960; Seneta, 1988).

Table 4.3: Previous estimates of life expectancy at birth for nineteenth-century Australia and colonies

Colony	Period	Males	Females	Source
NSW	1856-66	45.58		Pell, 1867
NSW	1860-75	47.08	49.71	Pell, 1879
NSW, Vic & Qld	1870-81	46.47	49.64	Burridge, 1884
Vic	1871	49.16	52.35	Burridge, 1882
NSW & Vic	1881-91	47.79	50.71	Dovey, 1893
NSW & Vic	1881-91	49.37		Duckworth, 1894
Australia	1881-90	47.20	50.84	Wickens, 1930
NSW	1891-92	49.60	52.90	Coghlan, 1892
Australia	1891-1900	51.06	54.76	Wickens, 1930
Australia	1901-10	55.20	58.84	Wickens, 1930

Life expectancies were increasingly used in actuarial and life insurance calculations from the 1880s onward, with estimates made for the more populous colonies by Burridge (1882, 1884), Dovey (1893) and Duckworth (1894) who also added life expectancies for Sydney and Melbourne. Other studies during this time calculated life expectancies for certain population groups, such as for assured persons or Friendly Society members (Wickens, 1930).

Burridge (1884) calculated life expectancies at birth of 46.47 years for males and 49.64 years for females in New South Wales, Victoria and Queensland combined for the years 1870–1881. These compare closely with a total Australian life expectancy here estimated to be 46.09 years for males and 49.50 years for females for 1871–1880.

More recently, Kippen (2002a) calculated abridged life tables for the Tasmanian population for each year from 1860 to 1899, noting that life expectancy in the colony was among the highest world-wide for much of the period. Leppard (2002) also calculated abridged life tables for South Australia for selected years between 1841 and 1996.

C. H. Wickens, who would succeed Sir George Knibbs as Commonwealth Statistician, constructed colonial and Australian life tables for the decennia 1881–90, 1891–1900 and 1901–10 and published these in the census report of 1911. Wickens was the first to make comparisons between all the States on a uniform basis, finding that life expectancy had increased since 1881–90, and that female rates of mortality were generally lower than those of males.

The calculations made here concur with Wickens' findings. In addition, they date the rise in life expectancy at birth in Australia from the mid-1880s.

Comparison with other countries

Life expectancies at birth in Australia during this period are compared with twelve European countries (Figure 4.9). Australia had comparatively high life expectancies during the second half of the nineteenth century, and from 1890 these were among the highest, a position that continues to the present day (ABS, 2010).

Norway and Sweden had the highest life expectancies for much of the period, with Norway leading from 1856–1886, and then sharing the position with Sweden from 1887. For certain years—1868–69, 1871, 1892, 1894, 1897, 1899–1900 and 1904–06—Australia led the way.

The lowest life expectancies from 1856 to 1871 alternated between the Netherlands and Iceland, and for most years from 1872, Italy. The years 1860 and 1882 were disastrous for Iceland, with life expectancy at birth falling below 20 years as a consequence of twin epidemics of diphtheria and measles. These took an especially heavy toll among infants (Guttormsson & Garðarsdóttir, 2002).

Figure 4.9: Life expectancy at birth in Australia, England and Wales, and selected European countries, 1856–1906
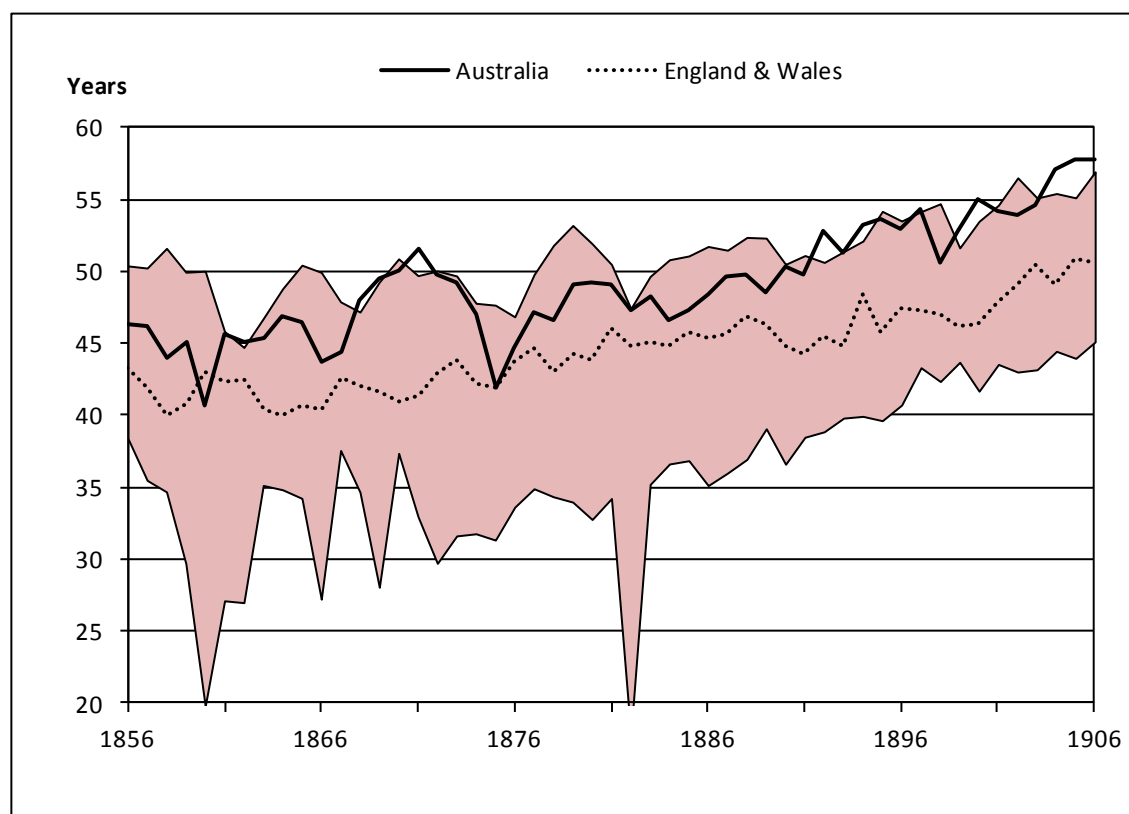
Note: The shaded region represents the range of life expectancies at birth across twelve European countries: Belgium, Denmark, England and Wales, Finland, France, Iceland, Italy, the Netherlands, Norway, Scotland, Sweden and Switzerland.

Source: Human Mortality Database (2011) for countries other than Australia.

Although the calculations of life expectancies in Australia were used mainly for actuarial purposes, they were also used to promote the salubrity and desirability of life in Australia. Comparisons were most often made with the Mother Country, England. Pell noted that

> 'I have undertaken the task of examining the reports of the Registrar-General, from the year 1856 to the present time, and the Census returns for the years 1856 to 1861, for the purpose of ascertaining and exhibiting, as exactly as the data will permit, the rates of mortality at various ages; so that a comparison may be made between this colony and England, so far as regards the effect of the climate and condition of the people upon the duration of life' (Pell, 1867).

Life expectancy at birth in Australia exceeded that of England for most of the period, by the considerable margin of four-to-five years. The years 1860 and

1875 were exceptions (Figure 4.9), with both being years of high mortality in Australia. From 1875 to 1889, the Australian advantage over England was at its lowest, averaging less than three years of additional life.

The comparisons that were increasingly being made between life expectancy in Australia and in other countries were an advance over previous comparisons of health status, which relied on crude rates of mortality, on infant mortality or on other age-specific rates.

Age decomposition of differences in life expectancy

The improvement of life expectancy in Australia over the course of the nineteenth century was the result of progress in survival among both children and adults. The changes in the levels of life expectancy at birth between 1856–60 and 1901–06 can be decomposed according to the contributions of mortality decline in various age groups (see Appendix 1).

The relative contribution of each age group is illustrated in Tables 4.4 and 4.5.

Table 4.4: Age decomposition of change in life expectancy at birth, males, Australia, 1856–60 to 1901–06

Age x	lx (1856-60)	nLx (1856-60)	Tx (1856-60)	lx (1901-06)	nLx (1901-06)	Tx (1901-06)	Change	Per cent
0	100000	89917	4307615	100000	92930	5425659	2.584	23.1
1	85595	325451	4217698	89901	353364	5332729	3.263	29.2
5	77130	381130	3892247	86781	431388	4979365	0.507	4.5
10	75321	373671	3511117	85774	426691	4547977	0.209	1.9
15	74147	366198	3137446	84902	421210	4121286	0.309	2.8
20	72332	353896	2771249	83582	413267	3700076	0.640	5.7
25	69226	337748	2417353	81725	403458	3286809	0.620	5.5
30	65873	320101	2079605	79658	392447	2883351	0.615	5.5
35	62167	301057	1759504	77320	379296	2490905	0.482	4.3
40	58256	279871	1458446	74398	363170	2111609	0.489	4.4
45	53693	256761	1178575	70870	343865	1748439	0.354	3.2
50	49011	231244	921814	66676	320660	1404574	0.359	3.2
55	43486	201974	690570	61588	292476	1083914	0.305	2.7
60	37304	167738	488596	55403	256458	791438	0.273	2.4
65	29792	131781	320857	47181	210269	534980	0.045	0.4
70	22921	95999	189077	36927	154373	324711	-0.007	-0.1
75+	15479	93077	93077	24822	170338	170338	0.131	1.2
Sum							11.180	100.0

Table 4.5: Age decomposition of differences in life expectancies at birth, females, Australia, 1856–60 to 1901–06

Age x	lx (1856-60)	nLx (1856-60)	Tx (1856-60)	lx (1901-06)	nLx (1901-06)	Tx (1901-06)	Change	Per cent
0	100000	90942	4600202	100000	94075	5802203	2.823	23.5
1	87059	331583	4509260	91536	360112	5708128	3.407	28.3
5	78732	389636	4177677	88520	440258	5348016	0.455	3.8
10	77122	382980	3788042	87583	435913	4907758	0.187	1.6
15	76070	375685	3405061	86782	430811	4471845	0.388	3.2
20	74204	364446	3029377	85542	423632	4041034	0.553	4.6
25	71575	350762	2664931	83910	414562	3617402	0.475	4.0
30	68730	334446	2314168	81915	403762	3202840	0.652	5.4
35	65048	315027	1979722	79590	390910	2799077	0.604	5.0
40	60963	294104	1664695	76774	376350	2408168	0.570	4.7
45	56679	272940	1370591	73766	360718	2031818	0.442	3.7
50	52497	251940	1097651	70521	343217	1671100	0.319	2.7
55	48279	227526	845711	66766	321479	1327882	0.371	3.1
60	42731	196596	618185	61825	291846	1006403	0.318	2.6
65	35907	159933	421589	54913	251193	714557	0.219	1.8
70	28066	122137	261656	45564	197187	463364	-0.028	-0.2
75+	20789	139519	139519	33311	266177	266177	0.266	2.2
Sum							12.020	100.0

The columns 'Change' indicates the additional number of years lived between ages *x* and *x+n* having an effect on life expectancy at birth. Thus, for females, 0.6 years out of an increase in life expectancy of 12.0 years between 1856–60 and 1901–06 occurred because of improvements in mortality in the age group 35–39 years.

For both males and females, greater than half of the total change in life expectancy was achieved through reductions in under-five mortality. For males, 23% of the increase was contributed by the change in mortality at age 0 years, and 29% by ages 1–4 years. For females, the figures were 24% and 28%. The contribution by ages 1–4 years is notable, and outweighs the improvement in infant mortality.

Around 20% of the gain in life expectancy was contributed by ages 20–39 years, reflecting the decline in causes of mortality among this age group. Very little improvement occurred among older ages of 60 years and over; less than half a year of the total 11.2 year gain in male life expectancy over the period 1856–60 and 1901–06 was attributable to mortality decline above age 60, and less than one year out of 12.0 years for females.

Improvements in life expectancy occurred through reductions in mortality at all ages, except perhaps age 70–74. However, reductions in death rates among children aged under five dominated survival improvements.

TURNING POINTS IN MORTALITY

This work previously asserted that a notable change in the death rate in Australia took place in the mid-1880s. The trend began a steady decline unlike any that had occurred before, and exhibited less annual variation. The observation relied on a visual examination of graphical trends. To determine with greater certainty when turning points in mortality occurred—among each sex and age group, along with rates of decline before and after turning points, and whether these changes occurred simultaneously in all colonies—a formal statistical method is adopted using joinpoint analysis (see Appendix 1).

Figure 4.10: Turning point analysis showing all-cause mortality decline in Australia, 1856–1906
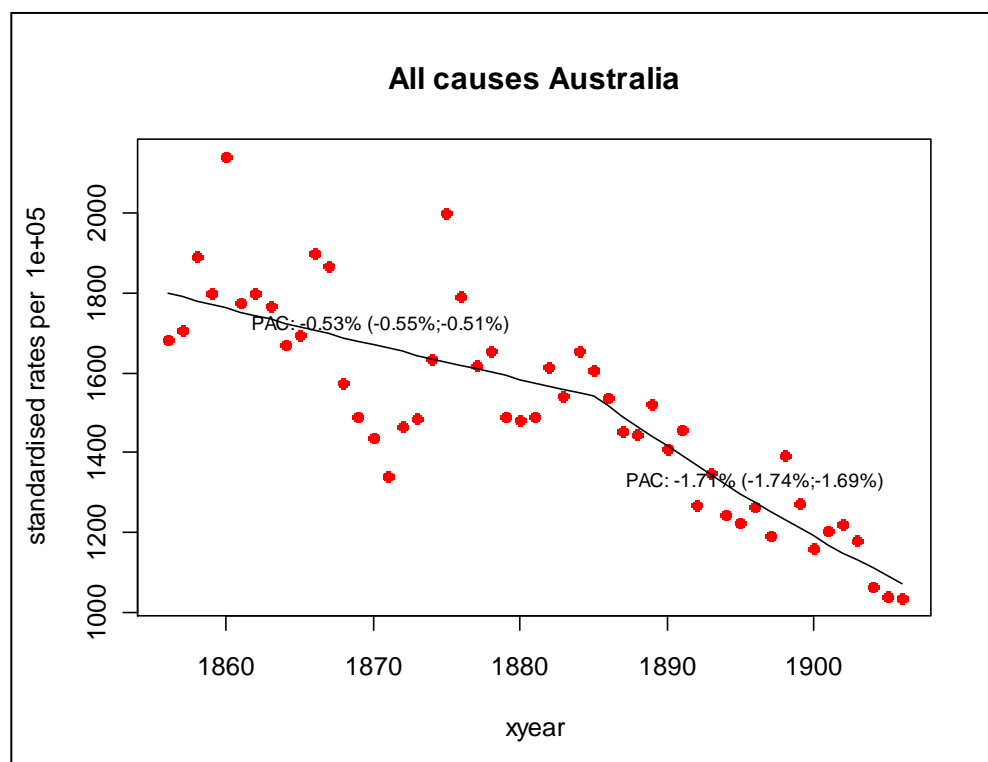
A preliminary analysis of the entire time series of mortality from 1856 to 2010 (Figure 4.4) finds a single turning point in 1969, interpreted as being largely due to coronary heart disease mortality among males reaching a peak (Christie, 1974; Dobson *et al.*, 1981). If two turning points are specified, the joinpoint model returns a second turning point in the mid-1880s. It is this second turning point that is here the focus of further analysis.

Table 4.6: Turning points and percent annual change (PAC) for age-standardised and age-specific mortality rates in Australia, and in each colony, 1856–1906

Sex / Age group / Colony	Turning point	Percent annual change (PAC) before and after turning point
All persons	1885	-0.53, -1.71
Males	1885	-0.50, -1.60
Females	1885	-0.61, -1.80
Age 0	1887	0.04, -2.18
Age 1-4	1860	12.00, -2.64
Age 5-9	1861	22.00, -2.43
Age 10-14	1875	0.04, -1.81
Age 15-19	1884	0.28, -2.40
Age 20-24	1885	0.08, -2.61
Age 25-29	1887	-0.18, -2.56
Age 30-34	1898	-0.96, -3.98
Age 35-39	1882	-0.56, -1.69
Age 40-44	1877	-0.44, -1.58
Age 45-49	1884	-0.41, -1.82
Age 50-54	1886	-0.12, -2.04
Age 55-59	1889	-0.32, -1.58
Age 60-64	1882	-0.12, -1.01
Age 65-69	1878	0.29, -0.51
Age 70-74	1874	0.61, -0.13
Age 75+	1886	0.32, -0.19
New South Wales	1885	-0.49, -1.84
Victoria	1889	-0.80, -1.65
Queensland	1884	-0.27, -2.85
Western Australia	1897	0.49, -4.13
South Australia	1875	0.42, -1.46
Tasmania	1889	-0.68, -1.65

For the period 1856–1906, the turning points for age-standardised and age-specific all-cause mortality rates in Australia, along with the age-standardised rates for each colony, are presented in Table 4.6.

For total mortality in Australia, the optimal turning point was identified as occurring in 1885. Figure 4.10 is a scatter plot of age-standardised all-cause death rates for all persons in Australia from 1856 to 1906. A joinpoint model is also drawn on this graph, being the two line segments joined at the turning point of 1885. The model indicates that mortality in Australia declined at an annual average rate of 0.53 per cent between 1856 and 1885. The decline was more rapid between 1885 and 1906, at an annual rate of 1.71 per cent. The year 1885 was also identified as the turning point for male and female all-cause mortality. The rate of decline in the female rate—both before and after 1885— was slightly greater than for males.

Age-specific rates were also analysed. A turning point around the year 1885 was found for a number of age groups: 1882 for age 35–39 and age 60–64, 1884 for age 15–19 and age 45–49, 1885 for age 20–24, 1886 for age 50–54 and age 75+, 1887 for age 0 and age 25–29, and 1889 for age 55–59 years. The rates of change after the turning point were highest for young persons aged 20–24, 25–29 and 15–19 years, although each of these are age groups with low overall rates of mortality (Figure 4.6). The decline for age 0 which began in 1887 was considerable, falling at an average annual rate of 2.18 per cent to 1906.

The decline began well before 1885 for several age groups. For age 1–4 it began in 1860, for age 5–9 in 1861, and for age 10–14 in 1875, with a decline after the turning point of around 2 per cent per annum. Note that the all-cause death rate among ages 5–9 and 10–14 years is lowest among all age groups. Mortality for age groups 40–44, 65–69 and 70–74 years also began to decline in the mid-to-late 1870s, although for the latter two age groups the decline was small in magnitude.

For age 30–34 years the turning point in mortality did not take place until well after the mid-1880s. Commencing in 1898, the fall to 1906 was steep at an average annual rate of almost 4 per cent.

Turning points varied across colonies. The joinpoint model identified a turning point for South Australia as early as 1875, attributed to a mortality peak resulting from twin epidemics of measles and scarlet fever. If the mortality rate in South Australia during 1874–1876 had remained at pre-epidemic levels (i.e. 1,500 deaths per 100,000 population) then the year 1882 would have been the turning point.

In New South Wales, Victoria, Queensland and Tasmania, the mid-to-late 1880s were turning point years. Queensland had the largest post-turning point fall among these four colonies, with a 2.85 percent average annual decline in mortality from 1884 to 1906. Victoria had the largest pre-turning point decline; mortality was already falling before 1889, albeit at a low annual rate of 0.80 per cent between 1856 and 1889.

In Western Australia, mortality rose at an annual rate of 0.49 per cent from 1856 to 1897, and then fell by more than 4 per cent annually to 1906. As mentioned (and see also Figure 4.1), the fall was coincident with an influx of population and socioeconomic development that accompanied the 1890s gold rush.

MORTALITY BY GEOGRAPHIC REGION

For much of the period, the Registrars and Government Statisticians published crude mortality rates for capital cities and suburbs as well as for non-metropolitan regions, and occasionally for other large cities in their jurisdiction.

These are instructive since differentials in mortality rates by geographic regions often point to differences in underlying mortality processes. Many factors varied across regions—exposures to diseases and risk factors, the diffusion of preventative or treatment measures, inequalities in access to health care facilities and the availability of health practitioners.

Mortality rates in metropolitan areas—in this case in Sydney and Melbourne—were higher than in other regions, although with time the differential narrowed (Figure 4.11). The Sydney mortality rate in the 1870s and early 1880s was around 60% higher than in country New South Wales. As Sydney's suburbs grew in size and in population density, their mortality rate also rose, so that by

the mid-1880s, it had reached a par with inner Sydney. After the mid-1880s, the suburban mortality rate began to decline rapidly, and by the mid 1890s it had achieved parity with country New South Wales, where the rate had shown little or no change. The mortality rate in Sydney City showed little decline during the 1890s. In the suburbs of Sydney, mortality had almost halved in the ten years from 1885 to 1895, falling from 23.7 to 12.1 deaths per 1,000 population.

Figure 4.11: Crude mortality rates in metropolitan and country areas of New South Wales and Victoria, 1870–1906
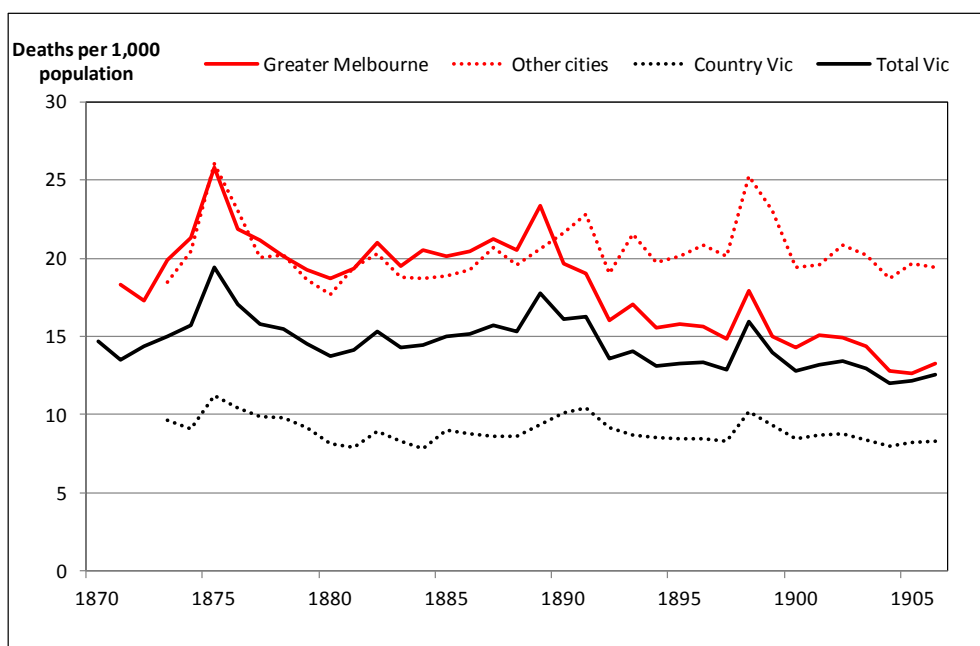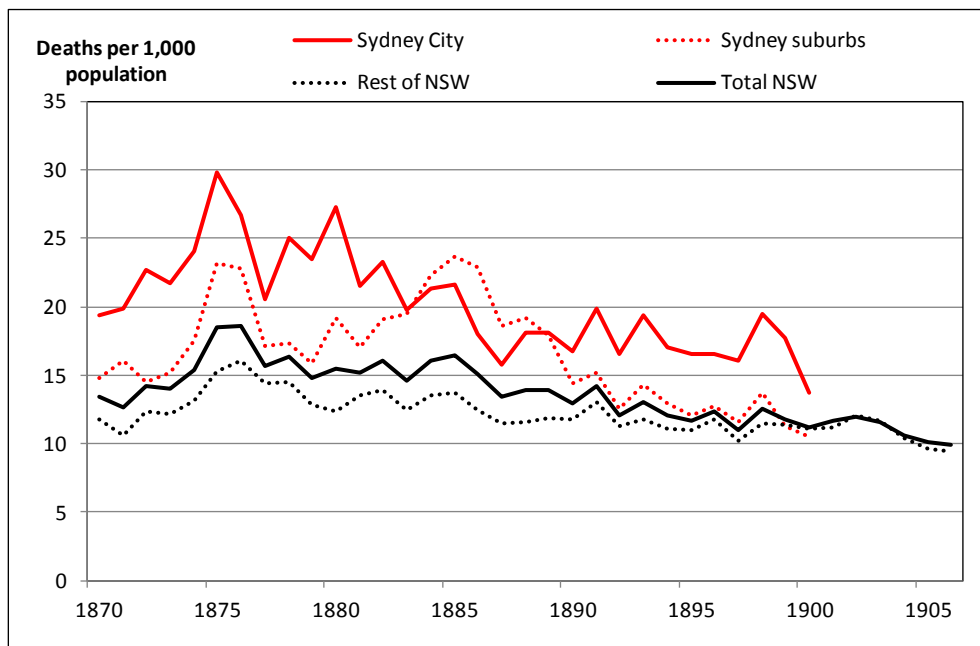
The same phenomenon occurred in Victoria, although here it began slightly later than in New South Wales. From 1870 to 1890, mortality in Greater Melbourne was twice that in country Victoria. After 1890, the rate began to decline, although unlike Sydney it did not reach the same level as in country regions. It is also noteworthy that the mortality rate in extra-metropolitan cities such as Ballarat and Bendigo remained unchanged.

The factor, or factors, responsible for the fall in mortality in New South Wales and Victoria were most apparent in metropolitan regions. W. H. Hall, the New South Wales Statistician, attributed the decline to government-initiated public health measures—'The fall began in the metropolis after 1889, the year when the improved sewerage system was installed, and about the same time that the Dairies Supervision Act came into operation' (Hall, 1906, p.665). The Victorian Government Statist favoured better country living conditions, combined with the rural ill seeking urban health care,

> 'In every country the death rate is higher in towns than it is in the country districts. This circumstance, although no doubt partly attributable to the superior healthfulness and immunity from contagion prevailing in the latter, is also to a great extent due to the fact that hospitals and charitable institutions, which are frequented by patients from the country as well as by town residents, are generally situated in the towns; and further, that outside of charitable institutions many persons die who have come from the country on the approach of a serious illness for the sake of the superior nursing and medical attendance to be obtained in towns' (Drake, 1908, pp.337–338).

These explanations bear further scrutiny and a fuller discussion of urban-rural mortality follows in Chapter 8.

SEASONALITY OF MORTALITY

Beyond yearly reporting, the abstracts of mortality published additional detail on when deaths occurred. For most colonies, deaths were also tabulated by month of registration. In addition, weekly and monthly reports were compiled and published in colonial *Government Gazettes* for some jurisdictions such as Sydney and Melbourne and their suburbs.

During much of the nineteenth century, the search for disease aetiology focussed on the relationship between temperature and illness. Climate was seen to be a contributing factor—especially to high rates of infant mortality—and so many of the abstracts were accompanied by detailed information on maximum and minimum daily and monthly temperatures.

Monthly tabulations of deaths make possible a more detailed examination of trends and patterns in mortality than that provided by annual compilations alone. Deaths attributable to a number of causes, most notably infectious diseases but also cardiovascular disease and others, follow a cycle that peaks in certain seasons and occurs less frequently in others (AIHW: de Looper, 2002; Dewdney, 1960).

However, deaths were tabulated by month of registration, rather than by the month of their occurrence, and it is occurrence which is preferred for epidemiological purposes. Some deaths were registered many months after the event, due to factors such as delayed notification, requirements for a coronial inquest, end-of-year delays or even coder shortages among Registration Office and Statistical Bureau staff (Taylor, Lewis & Powles, 1998a). These were the exception, however. In 1905, the registration of an infant death in Sydney typically took place two days after the event (Armstrong, 1905, p.517). Any lags between occurrence and registration will have some effect on monthly tabulations, less effect on seasonal (3-monthly) tabulations, and less effect still on yearly tabulations.

The Registration Acts specified the time period during which deaths were to be notified to Deputy Registrars or to those responsible for registration. This requirement varied between colonies; as specified in the original Acts it was within eight days following a death in South Australia, 10 days in Tasmania, 15 days in Victoria, and one month or thirty days in Western Australia, New South Wales and Queensland. As Acts were altered or repealed, the time periods often changed – in 1856, the period for notifying a death in Western Australia was revised to 14 days by the new Registration of Births, Deaths and Marriages Act (19 Vic. 12).

Longer time periods for notification led to greater opportunities for lags between the occurrence and registration of death. Queensland Registrar-General Henry Jordan noted that

> 'Considerable variations will be observed in the number of registrations in the monthly returns. These cannot be made to exhibit the exact number of deaths actually occurring in each month of the year, because some delay in registration is allowed by law, for the convenience especially of persons resident in country places; but compared, as these monthly returns are, with the corresponding months of the previous year, the record is on the whole a safeguard, and has its special value in keeping up public attention to a subject of vital importance; for, as it has been well said, "It is not with these events a mere question of money, or of fluctuations of prosperity that come and go, but of life itself."' (QldRG, 1876).

In the abstracts, deaths in New South Wales and Victoria combined—two colonies with readily available data—numbered approximately 126,000 during 1880–1884, at an average of 2,100 for every month, or 69 for every day of the year over these five years. Deaths occurred (or to be more precise, were registered) more frequently in some months than in others (Figure 4.12). There was an average of 77 deaths per day in January and December, whereas during each day in September there were 61. Deaths tended to occur more often in the warmer months (December to March) and less often in colder months (June to September).

Longer time periods shows the same cyclical pattern (Figure 4.13). When the monthly average of deaths is compared with the average over the entire year, deaths exhibit a peak in summer and a trough in winter, varying by 30 per cent or more between low and high months. In some months, such as January 1875, the peaks are higher, indicating an excess due to a seasonal epidemic.

The pattern of summer peaks in mortality—largely because of the increased incidence of diarrhoea, dysentery and other enteric diseases—was a reversal of the experience in England, where peaks of deaths from influenza, pneumonia, tuberculosis and bronchitis were the norm during the wet and cold of winter (Stevenson, 1981).

Figure 4.12: Average daily number of deaths, by month, New South Wales and Victoria, 1880–1884

Number of deaths per day
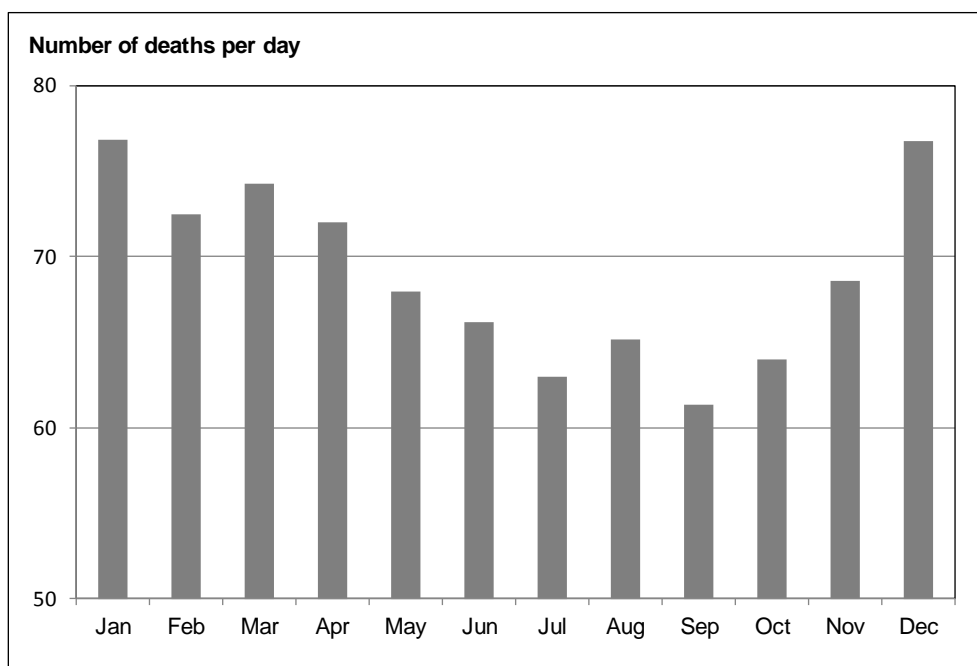
Figure 4.13: Seasonality of deaths, New South Wales and Victoria, January 1871 to December 1890

Per cent of average monthly number of deaths
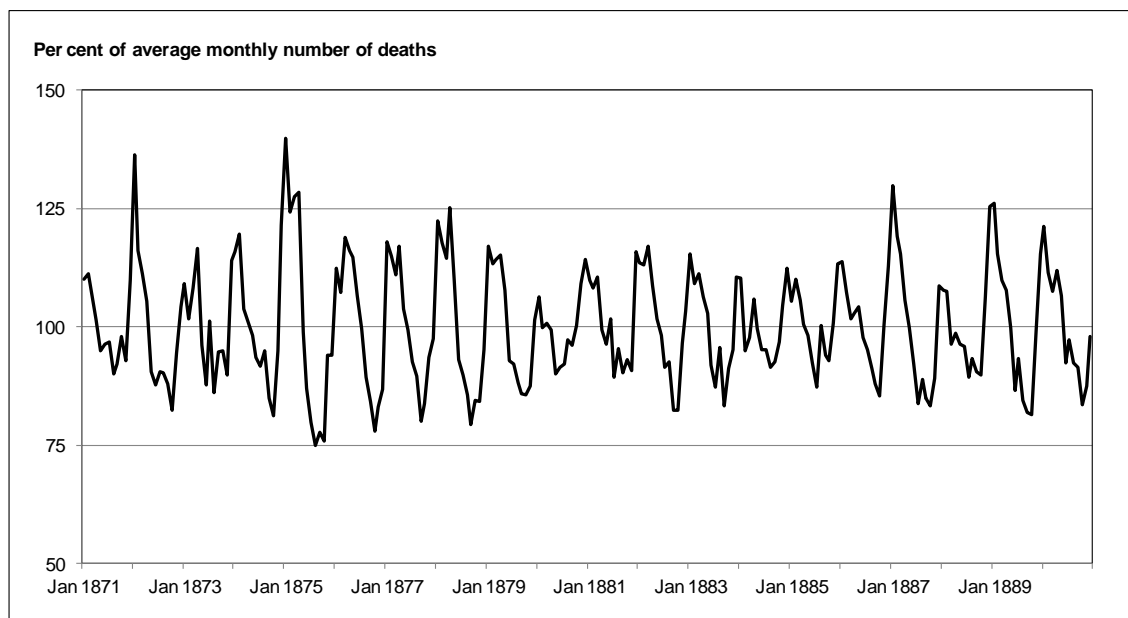
The summer peak in Australian mortality underwent change throughout the nineteenth century. In the mid-1850s, deaths during summer months averaged

20–30 per cent higher than for the entire year. This excess declined progressively until the first decade of the twentieth century, when excess winter deaths began to occur (Figure 4.14). In this way, the seasonal pattern of deaths in Australia began to resemble that of England. Excess summer deaths continued to decline throughout the twentieth century until the 1960s, when the decline levelled. Excess winter deaths peaked in the 1960s, and have declined somewhat since then (AIHW: de Looper, 2002).

Dewdney (1960) commented on the disappearance of excess summer deaths and the emergence of a new peak in winter. He noted a decline in mortality from certain diseases which had previously been most severe in the summer months, such as dysentery, gastroenteritis and other infectious and parasitic diseases. Accompanying this was an increase in diseases associated with a high mid-year mortality rate, such as diseases of the circulatory system and respiratory diseases such as pneumonia and influenza. Certain other causes also changed in their seasonal incidence.

Figure 4.14: Seasonal excess mortality, New South Wales and Victoria, 1856–1906
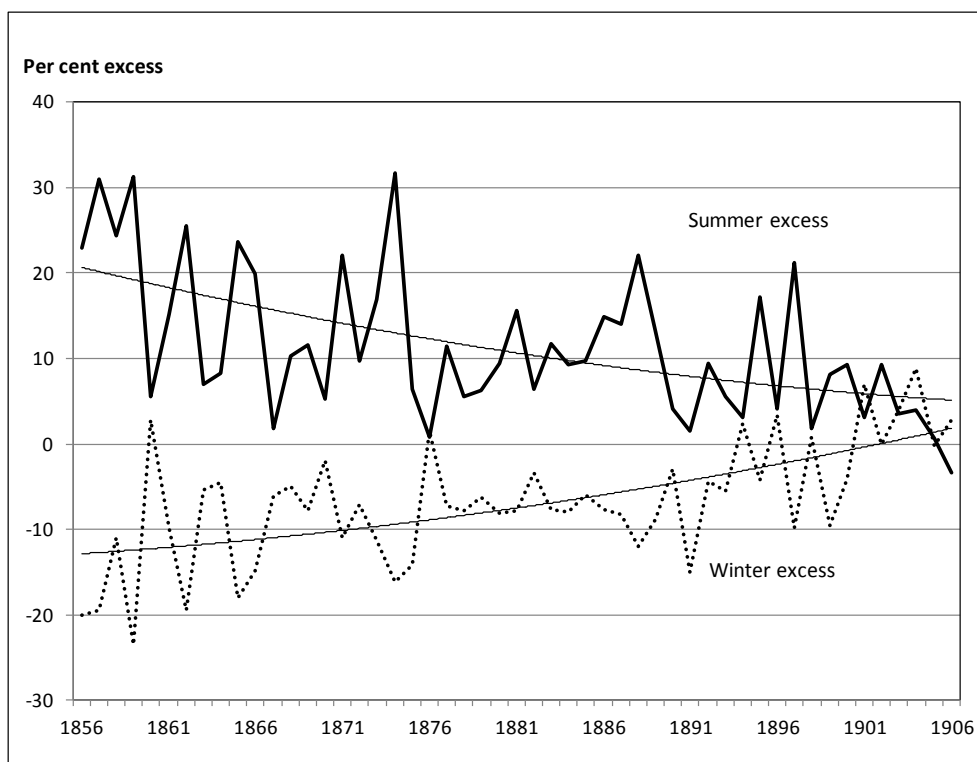
SUMMARY

All-cause mortality in Australia fluctuated considerably from 1856, at between 1,300 and 2,000 deaths per 100,000 population. Mortality rates were highest among the youngest and oldest age groups, age 0 and 75 years and over.

The year 1885 was identified as a turning point in mortality in Australia, after which death rates began to exhibit a steady decline, and with less annual fluctuation. From 1,580 deaths per 100,000 population in 1885, all-cause mortality fell to below 1,000 deaths per 100,000 population by 1906, a reduction of more than one-third over two decades. The largest declines were among the younger age groups of 0 and 1–4 years, with the decline among infants becoming more rapid after the mid-1880s.

Life expectancy in Australia increased over the period. For the years 1856–60, life expectancy at birth for males was 43.08 years, and for females 46.00 years. By 1901–06, this had increased to 54.26 and 58.02 years, with most of the increase in years of life occurring between 1881–90 and 1901–06. Over half of the gain in life expectancy between 1856–60 and 1901–06 can be attributed to reductions in under-five mortality. Life expectancy in Australia was high between 1890 and 1906, compared with other European countries.

Mortality declines in the twenty years after the mid-1880s were most apparent in urban areas, with rural death rates remaining largely unchanged. Mortality in Australia was seasonal, initially with a summer peak, although this disappeared over the course of the second half of the nineteenth century, to be replaced by a winter excess.

Chapter 5

Registering and classifying cause of death

Much of the explanation as to why mortality rose and fell in nineteenth-century Australia is bound up with investigating specific causes of death among the population. Rises and falls in levels of mortality largely reflected the ebbs and flows of infectious and epidemic diseases. Their increasing amelioration and control during the last two decades of the century led to a lasting mortality decline in Australia. The waning of communicable disease also heralded a new foe, as the proportion of deaths attributed to non-communicable diseases began to rise (Omran, 1971).

Official cause-of-death statistics in Australia were derived from the information collected in compliance with the registration laws. In one sense, they were products of a legal process, the registration of death (Moriyama *et al.*, 2011). However, their usefulness far outweighs their status as an administrative by-product. Since data on sickness and morbidity were largely absent, cause-of-death formed the evidence base for nineteenth-century public health improvement. Today, health indicators based on high quality mortality statistics remain an essential part of public health monitoring systems.

To better understand the historical statistics on cause of death in Australia, it helps to know how these were registered. The manner in which diseases and accidents leading to death were identified, codified and reported during the nineteenth century assists in explaining the data and its restrictions.

CAUSE-OF-DEATH: FROM REGISTRATION TO ABSTRACT

The collection method for cause-of-death in Victoria is illustrative: as prescribed in the Registration Act, the occupier of the house in which a person had died, or the person(s) present at death, or the doctor in attendance during the final illness had 60 days in which to give the particulars of death to the Deputy Registrar as part of the process of registering a death. These particulars included what was understood to be the cause of death. Should an inquest or magisterial inquiry be required, it was the task of the jury or magistrate to inform the Registrar-General or Deputy Registrar of these particulars.

The Deputy Registrars were required to send copies of their registers to the Registrar-General four times per year. If the details therein, including information on cause of death, were incomplete or inadequate, the Registrar-General's office could return the register to the Deputy Registrar for amendment. The registers were then examined by a doctor — generally a clerk in the office — and causes of death were tabulated and classified. These returns were revised if needed by the Registrar-General and were then published.

The process of collecting information on cause-of-death varied somewhat across the Australian colonies. Although we are indebted to nineteenth-century colonial administrations that these data were collected at all, this non-standardisation of collection methods led to statistics that varied in accuracy and quality. The requirement for medical certification of cause of death was perhaps the most important determinant of the quality of cause-of-death statistics, and the colonies introduced certification into their legislation at different stages (Cumpston, 1989).

Medical certification

A medical certificate of cause of death, filled by a doctor with appropriate training, provided greater precision as to cause-of-death, although the information from certificates was subject to the limitations of nineteenth-century medical knowledge and practice.

The original Registration Acts made no provision for medical certification of death. Commenting on Victoria's 1853 Act, W. H. Archer noted that

> 'On examining the Schedules…appended to the Act, very many important omissions were discernable. Among others…a certificate signed by the medical attendant should be required, stating the cause of death, duration of last sickness, and when the party was last seen by him. All very important and omitted' (Archer, 1854, p.113).

In 1857, a number of Geelong's medical practitioners lamented the need for greater certification. Of the 331 deaths registered during the first six months of the year, only 158 were medically certified, and most of these from the coroner and the house surgeon at the hospital. Many of the remaining causes of death were, in the opinion of the doctors, wrongly assigned (*AMJ*, Oct. 1857, p.285).

In Western Australia the Colonial Surgeon Alfred Waylen reported

> 'In conclusion, I may be allowed to refer to the extremely unsatisfactory manner in which deaths are registered. At present the Register is not a reliable source from whence to frame statistics. This arises from the fact that any one may register a death, without producing a certificate of the cause from a qualified practitioner. This may at times be difficult to procure in some of the outlying districts, but certainly in towns and centres of population there does not seem to be any reason why it should not be made compulsory to produce a Medical Certificate of the cause of death' (WAPH, 1877).

Before the passing of legislation requiring certification at the time of registration of death, Registrars relied largely on the largesse of the medical profession to provide assistance with recording cause of death. In the progress report on the establishment of registration in New South Wales, Registrar-General Chris Rolleston requested

> 'Moreover, with the view of obtaining the co-operation of the medical profession, and collecting data for the formation of Tables of Sickness and Mortality, I prepared a Circular, which has been printed and distributed to the members of the profession throughout the Colony, pointing out the advantages which would accrue to medical science, if they would give me their valuable assistance in recording the causes of death, and the duration of last illness' (Rolleston, 1856).

Each colony struggled with the issue. Some Registrars needed to win over the support of the medical profession before doctors would use the new cause-of-death classifications. Registrar-General Darvall asked

'I hope that the medical profession in Queensland will...cordially assist "even in opposition to their individual ideas of classification," in perfecting tables which will hereafter supply such valuable information to their body, although perhaps not exactly given in the form each thinks best. That there is plenty of room for improvement is evidenced by the fact that one-seventh of all deaths in the country are unspecified. So much importance is "in the Colony of South Australia" attached to these certificates, that they have been made the subject of legal enactment, and a heavy penalty imposed on their omission' (QldRG, 1860).

Medical certification of death at the time of registration became compulsory through legislation, firstly in South Australia in 1856, then Victoria in 1865, Western Australia in 1879, Queensland in 1885, Tasmania in 1896 and belatedly, New South Wales in 1934.

As per the 1856 South Australian Act (19 Vic. No. 3), medical practitioners who had attended or examined a deceased person were required to deliver a certificate to the person responsible for notifying the death to the District Registrar. The certificate was to set forth "to the best of his knowledge and belief the cause of death". Deaths certificates had provision for several causes to be entered; these were entered 'in the order of their appearance, and not in the presumed order of their importance' (Archer, 1854, p.136).

By 1903, 85 per cent of the 2,788 deaths registered in Western Australia in that year were medically certified. Another 254 (10 per cent) were the subject of coroner's inquests, 64 (4 per cent) had a judicial order for burial without an inquest, and 39 (2 per cent) were not certified or not stated (Fraser, 1906, p.383).

CLASSIFYING CAUSES OF DEATH: THE USE OF NOSOLOGIES

The production of comparable cause-of-death statistics relies strongly on a system of disease classification, or 'nosology', so that information might be grouped and displayed in a similar fashion in different places and times (Moriyama *et al.*, 2011).

Diseases may be classified in a number of fashions; by their cause, the mechanism by which the disease is caused, by the disease symptoms, or by the organ system involved, although many diseases affect more than one organ. Nineteenth-century nosologies were constructed according to prevailing medical knowledge, and often exhibit uncertainty in definitions and classifications. Disease aetiology was in many cases unknown, resulting in diagnoses that reflected one or more symptoms rather than a cause.

In 1839, William Farr became the first compiler of statistical abstracts in the General Register Office for England and Wales. His responsibility included the collection and organisation of official medical statistics in England and Wales, which were published in the *First Annual Report of the Registrar-General of Births, Deaths and Marriages in England* (Registrar-General of England, 1839; Eyler, 1979).

One of Farr's first priorities at the G.R.O. was to introduce a nosology, this being "…as important in this department of inquiry, as weights and measures in the physical sciences" (Registrar-General of England, 1839, p.71). Although Farr was acquainted with existing nosologies, such as those of William Cullen (1769) and John Mason Good (1817), these were found to be unsuited for his purposes and so he began to draw up his own system.

His nosology was based on how disease affected the population,

> "…whether they are generated and prevail only in particular localities (endemics), extend like cholera over nations (epidemics), or are propagated by contagion; whether they arise in an isolated manner (sporadically) from ordinary causes, and sources existing in the organization itself; or whether they are caused by violent means" (Registrar-General of England, 1839, p.67).

Farr divided causes into three classes, the first class consisting of epidemic, endemic and contagious diseases. Farr termed these 'zymotic diseases', and he believed that they were caused by similar morbid processes. He regarded this class of diseases as an index of public health and well-being (Eyler, 1979, p.55). The second class consisted of diseases held to arise sporadically, arranged under eight organ systems (nervous system, respiratory organs, etc.), plus

subdivisions for those of uncertain location such as tumours, malformations, debility, sudden death, and old age. The third class was for deaths by violence. Farr's nosology was used for more than twenty years by the G.R.O. to classify causes of death.

Just as the Australian colonies closely followed England in introducing registration of births, deaths and marriages, they also followed suit in adopting Farr's cause-of-death classification. Victoria was again the pioneer in introducing the systematic classification of diseases and death into Australia.

Archer circulates Farr's nosology

Reflecting on his appointment in 1853 to carry out the provisions of the Registration Act in Victoria, W. H. Archer recalled that,

> 'I made it part of my duty to assimilate the nosological nomenclature for use in this Colony to that promulgated by Dr. Farr for use in England. My object was primarily to promote, as far as possible, scientific accuracy in the returns of "Cause of Death", and secondarily to secure uniformity of statistical practice both here and in England' (Archer, 1863).

E. S. Norman Campbell, the Registrar-General, and Archer in his role as Assistant Registrar-General arranged for the Chief Medical Officer to circulate copies of Farr's nosology among Deputy Registrars and medical practitioners in Victoria (Archer 1854, 1856). This table, with explanatory notes by Farr, was somewhat dated even in 1853, being the version first recommended by the Presidents of the Royal College of Physicians and Surgeons in 1837, and published in the English Registrar-General's Fourth Annual Report for 1840–41 (Campbell, 1855a, 1856). Regardless, the system was quickly adopted by three other colonies, South Australia, New South Wales and Queensland, and was used for reporting their abstracts of mortality (Table 5.1).

Table 5.1: Nosologies in use for colonial abstracts of mortality, 1853–1906

Colony	Archer, 1853	Archer, 1863	Hayter, 1886	RCP, 1896	CBCS, 1907
New South Wales	1856-1862	1863-1885	1886-1904	1905	1906
Victoria	1853-1858	1864-1885	1886-1906		
Queensland	1860-1863	1864-1884	1885-1902	1903-1906	
Western Australia		1860-1891	1892-1906		
South Australia	1856-1860	1861-1883	1884-1906		
Tasmania		1868-1886	1887-1902	1903-1906	

Note: Details of nosologies can be found in Appendix 4.

In 1856, South Australia passed a new Registration Act (19 Vic. No. 3). Incoming Registrar John Fullerton Cleland began to use Farr's classification forthwith although his abstracts of mortality still retained a number of unfamiliar terms. The new nosology caused some local consternation,

> '…the table of diseases and deaths, is given in the most incomprehensible terms to be found in the technical glossaries of medicine and pathology. There is no attempt to *classify* diseases; no division into sections, showing which are preventible diseases, and which are not; which are contagious, and which are not; or showing any other distinction whatever. We have enumerated between 90 and 100 of the ills that flesh is heir to, although some of them are described in terms that the ear will never become accustomed to. What an army of evil demons stand in typographical array! Rascola and Varicella, Cynanche Tonsillaris and Stephalitis, with other designations before which the unlearned must stand perfectly appalled, never having heard before that such, ghouls and vampires were let loose upon society!' (SAA, 17 Mar. 1860, p.3).

In his Second Annual Report, New South Wales Registrar-General Chris Rolleston underlined his use of the Farr system,

> 'The nosology, or classification of diseases adopted in the Mortality Tables, appended to this Report, is identical with that employed in the Mother Country, and in the Colony of Victoria' (New South Wales Registrar-General, 1858).

Archer's 1863 revision

Ten years later Archer, who was now Registrar-General in Victoria, felt that a revised nosology was required. Over the previous decade Farr had made a number of changes to his system, some of which had been adopted by Archer,

but now he thought it 'advisable to issue for more extended use a complete Nosological Index in accordance with the latest statistical improvements' (Archer, 1863). Archer's 1863 nosology most closely resembled the version employed by Farr for the *Twenty-First Annual Report of the Registrar-General in England*, for the year 1858 (Registrar-General of England, 1860).

Archer was assisted by his deputy Henry Heylyn Hayter, head of the statistical branch, in making some local modifications to Farr's list. These modifications were highlighted in the publication by the use of the suffix 'a' following a disease number. Thus, death from Hydatid Disease (coded I-4-2a) was added to the other parasitic diseases such as Thrush (I-4-1) and Worms, etc. (I-4-2). Other local additions involved Chicken Pox, Diphtheria, Tumour, Polypus, Haemoptysis, Congestion of the Lungs and Pulmonary Apoplexy, Ostitis and Periostitis, Sunstroke and Bite of Snake or Insect (see Appendix 4). The index to the new nosology also included a complete list of terms which were used in the 1853 version, and this allows a mapping of causes of death that were classified under the old version to the new.

Queensland followed Victoria and New South Wales in adopting the new system, with Registrar-General F. O. Darvall acknowledging that

> 'The colonies of Victoria and of New South Wales, have remodelled their Nosological Tables, so as to correspond with the modifications introduced into the English tables by Dr Farr, and I have thought it right to follow their example, as, by so doing, comparison is facilitated, and the value of the tables increased' (QldRG, 1865, p.vi).

Western Australia, South Australia and Tasmania followed the lead of the larger colonies, in some cases backdating the publication of causes of death in their abstracts of mortality so that they conformed to the new system. The new nosology was not introduced in Tasmania until 1867, when the newly appointed Government Statistician E. C. Nowell

> '…received valuable assistance in my attempt to introduce the system here from the Nosological Index prepared by the Registrar-General of Victoria, several copies of which he was kind enough to forward to me; and I have supplied one to each of the Institutions included in the Return. It is very desirable that the deaths for the

whole Colony should be classified according to the same system, which might
easily be done by supplying to each of the Deputy-Registrars a copy of the Index
referred to, with proper directions as to the use of it' (TasSR, 1866, p.xxvii).

Archer's revision remained in use in Australia for the next twenty-five years.
From time-to-time slight modifications were made, and new editions were
published. Most notably, the third edition revised the cause of death I-1-7
Typhus (and Infantile Fever) to I-1-7 Typhus and Infantile Fever, Typhoid, etc
(Archer, 1868). The fourth edition revised I-1-7 Typhus and Infantile Fever,
Typhoid, etc. to I-1-7 Typhoid Fever etc and I.1.7a Typhus, the first time that the
term 'typhoid fever' was used as a separate cause of death (Archer, 1878;
Cumpston, 1989).

Hayter's 1886 revision

Farr's successor as Superintendent of Statistics in the General Register Office in
London was Dr William Ogle (1827–1912). Ogle made the decision to change
the classification that was being used for the English Registrar-General's
reports. Until 1880, most of the tables were classified according to Farr's 1853
list, with only slight changes. In addition, a single table of total deaths was
published using a classification devised by the Royal College of Physicians.

It was the College classification which Ogle favoured, and he modified their
arrangement to meet his needs for the Registrar-General's reports (Robb-Smith,
1971). From 1881, the English Registrar-General's reports introduced a
classification which was in essence the same as the College's first revision of
their Nomenclature (Registrar-General of England, 1883; Royal College of
Physicians, 1885).

The new classification system, again with some local modifications, was
adopted in Victoria in 1886 by Government Statistician H. H. Hayter. The
publication again included an Index, showing the correspondence between the
old and new systems (Hayter, 1887a, 1887b). The system was adopted by the
registration departments in all Australasian colonies (including New Zealand)
(Table 5.1), which received copies as needed from the Victorian Government.

The Registrar-General of England and Bertillon classifications

The Royal College of Physicians prepared a third edition of its Nomenclature, which it published in 1896. While this revision was under way, French statistician and demographer Dr Jacques Bertillon (1851–1922) presented a classified nomenclature of causes of death to the 1893 Chicago meeting of the International Statistical Institute, and this was recommended for international use. The Bertillon classification took into account a number of historical classifications, including Farr's 1842 nosology, the College's 1869 arrangement, and Bertillon's own *Nomenclature des cause de décès* of 1886 (Robb-Smith, 1971).

By the time of the 1899 meeting of the International Statistical Institute, a number of countries, including Canada, France, Mexico and most states of the United States had adopted the Bertillon classification. Great Britain and Australia were notable exceptions. In the following year, 1900, the first decennial revision of the Bertillon classification took place, with twenty-six countries taking part. This was the genesis of the International Classification of Diseases, still in use in Australia today.

The Conference of Statisticians held in Hobart in January 1902 resolved that the classification of causes of death to be used by each Australian State be the same as that adopted since 1901 by the Registrar-General of England, as approved by the Royal College of Physicians (Parliament of Tasmania, 1902). Although the Bertillon system was considered, it was felt that the initiation of a new system should first occur in England before introduction throughout the Commonwealth (Fraser, 1902, p.46).

New South Wales used the new system to classify deaths for the year 1905, and Queensland and Tasmania for 1903. Despite the Conference resolution, Victoria, South Australia and Western Australia continued to use the Farr-Ogle system, thus complicating comparisons of cause of death.

Another Conference of Statisticians was held in Melbourne in November-December 1906. This time, it resolved that the latest modification of the Bertillon system be adopted as soon as practicable, pending the publication of a

complete nosological index by the Commonwealth Statistician (Conference of Statisticians, 1906).

George Knibbs, the newly appointed director of the Commonwealth Bureau of Census and Statistics, duly published the Bertillon classification in 1907 (CBCS, 1907). New South Wales was first to adopt the system for the year 1906, meaning that three different nosologies were operative across the States in this year. From 1907, statistics of deaths were published by the Commonwealth Statistician for the whole of Australia using the Bertillon classification (CBCS, 1908).

SHORTCOMINGS OF NINETEENTH-CENTURY CAUSES OF DEATH

The series of cause-of-death statistics collected by the colonial Registrars before 1907 are the primary source for the study of diseases and deaths during the period. But their value and historical importance should not overshadow their shortcomings, which are considerable. From the beginning, the Registrars recognised that standard nomenclatures and classifications for cause-of-death statistics could not be guaranteed,

> 'The 'Abstracts of Death'…convey probably a close approximation to the truth, though in some particulars they must not be taken to be specifically exact. A certain degree of inaccuracy will necessarily exist, in consequence of the frequent absence of explicit information as to the cause of death, especially in cases wherein some of the Coroners have signed the returns as informants. In many instances the causes of death are very indefinitely stated; in others, several causes are given conjointly. Thus we find assigned as causes of death, "Natural causes," "Visitation of God," "Accidental death," &c. On the other hand we have "Measles, scarletina, and dysentery;" "Phthisis, pneumonia, diarrhoea;" "Teething, measles, and dysentery."' (Campbell, 1855a).

Conformity was elusive in nineteenth-century Australia, and more so as the epidemiological transition began. Cause-of-death classifications were adopted at different times by the colonies. Over time, revisions were published requiring translations between old and new nosologies, and these new nosologies were also adopted at different times.

As Cumpston (1989, p.78) saw it, the reliability of cause-of-death information was subject to three factors:

    i.   The state of knowledge of pathology;

    ii.   The skill of the certifying practitioner;

    iii.   The extent to which it was considered desirable to reveal the true cause of death to the relatives.

The state of knowledge

With the improvement of medical knowledge came improved nosologies and better diagnosis of cause-of-death. Sir George Knibbs, the first Commonwealth Statistician, and Member of the International Institute of Statistics commented on the development of disease classifications, that

> '…any classification is influenced by accessions of knowledge, by new aspects of the subject matter arising therefrom and by the elimination of perceived defects. And it may be added that even changes in terminology will necessitate revision. One may say then that the development of a classification is or should be collateral with the development of the scientific knowledge affecting it' (Knibbs, 1929).

The prevailing ideas about disease causation changed markedly during this time. The predominant disease theory mid-century was the *miasmatic theory* which held that poisonous vapours or mists filled with particles from decomposed matter (miasmata) were the cause of illnesses. Diseases were the product of environmental factors such as contaminated water, foul air, and poor hygienic conditions. Infection was identifiable by its foul smell, and would affect individuals who resided within the locale that gave rise to such vapors. Farr, through his own *zymotic theory*, was an important early supporter of miasma theory (Eyler, 1979, pp.97 ff.).

Opposing miasmatic theory were contagionists, who believed that disease was passed through physical contact, and that *germ cells* or *animalculars* were the transmitting agents. John Snow had demonstrated that cholera was transmitted by water, following an epidemic in Soho, London in 1854. Between 1860 and 1864, Louis Pasteur demonstrated the relationship between germs and disease.

By 1866, medical opinion had changed to support the germ theory of cholera and its waterborne transmission.

As Snow's data and other supporting evidence emerged, William Farr's views also changed and he acknowledged that water, and not miasmata was the most important means of cholera transmission (Registrar-General of England, 1868). His conversion to germ theory took place during the 1870s (Eyler, 1979, p.107).

In the 1870s Joseph Lister developed practical applications of the germ theory of disease to surgical techniques. The following decade saw Robert Koch devise a series of tests to assess the germ theory of disease. Koch's Postulates were published in 1890, and were derived from his demonstrations that anthrax was caused by the bacterium *Bacillus anthracis*. These postulates helped determine whether newly discovered diseases were caused by a microorganism. By the end of the century, germ theory had gained widespread acceptance in Europe and more broadly.

In Australia, the burgeoning sanitary movement initially drew ideological strength from miasmatic theory, driven by the urgency of the country's high rate of infant deaths and rates of infectious disease. Set against this were a small number of independent thinkers, including Melbourne doctor and epidemiologist William Thomson. A disciple of William Farr, Thomson advocated the contagionist doctrine through his many publications between 1870 and 1883, although his outspoken views aroused widespread opposition (e.g., *Argus*, 27 Feb. 1872, p.6; and see *AMJ*, Jul. 15, 1883, p. 315–321; Gandevia, 1976). Eminent medical practitioner and university teacher James Jamieson, later to be Health Officer of Melbourne and editor of the *Australian Medical Journal*, also promoted germ theory, better hygiene and the use of disinfectants, and helped to swing professional opinion (Jamieson, 1876, 1883; Dunstan, 2005).

Regardless of local controversy, the Registrars routinely adopted the new English nosologies, which reflected revisionist thinking on disease causation. It remained for the Deputy Registrars and local doctors to acquaint themselves with the new thinking on cause-of-death that these nosologies embodied.

Prior to germ theory, diseases—infectious and otherwise—were often diagnosed by their symptoms alone. This had great potential for confusion and misdiagnosis, since different diseases can have similar symptoms, and one disease may have many symptoms. Put simply, symptoms were often understood as being the cause of death.

From the outset, the nosologies included examples in the 'Instructions to Deputy Registrars' which '…serve to illustrate the manner in which certain causes of death, now too often vaguely expressed, should be recorded' (Archer, 1863). Despite the plea for accuracy, symptoms embodying vague and unsatisfactory terms such as 'dropsy' and 'paralysis' were often given as causes of death. For infants, 'convulsions', 'teething' and 'atrophy and debility' were leading causes of death, and Victorian statist Hayter acknowledged that

> 'It is probable, however, that the causes of death amongst children are sometimes loosely given, and that sufficient pains are not taken to ascertain the primary disease' (Hayter, 1880, p.135).

Diagnostic problems were most severe for infant and elderly deaths, these being the age groups for which the proportion of uncertified deaths was highest (Williams, 1996). As the state of medical knowledge evolved, diagnoses based on symptoms alone grew less, and fewer unsatisfactory terms appeared in the nosologies (Table 5.2).

Changing ideas about medical causality affected certification for certain diseases over time. As the nineteenth century progressed, deaths previously coded to 'Old Age', for example, were increasingly allocated to other causes such as pneumonia or circulatory disease, thereby affecting longitudinal series (Hardy, 1994). Early-twentieth century trends in heart disease and cancer are still unclear, with apparent rises perhaps simply reflecting better diagnosis of deaths that were previously classified as ill-defined or unknown (Hall, 1906; Alter & Carmichael, 1996). Some idea of the effect might be gained by comparing the pattern of a particular disease with other associated diseases; an apparent decline in respiratory tuberculosis, for example, may be offset by increases in certification to other respiratory diseases.

Table 5.2: Some unsatisfactory terms in colonial nosologies, and an indication as to the numbers of deaths coded to these terms

Archer, 1863		Hayter, 1886		RCP, 1896	
Dropsy	large	Dropsy (oedema, where?)	trivial	Dropsy, Ascites, Ansarca	trivial
Paralysis	large	Paralysis Agitans	trivial		
Convulsions	large	Convulsions	large		
Teething	large	Dentition	large		
Old Age	large	Old Age	large	Old Age	large
Atrophy & Debility	large	Debility, Atrophy, Inanition (wasting, debilitas, starvation, want of breast milk (infants))	large	Atrophy, Debility	trivial
Tumor	trivial	Tumor (where?)	trivial	Tumor	trivial
Polyps	trivial	Abscess (where?)	trivial	Abscess	trivial
Mortification	trivial	Mortification (death of a part)	trivial		
Sudden deaths (cause unascertained)	trivial	Sudden (cause unascertained)	trivial	Sudden (cause unascertained)	trivial
Diseases or deaths not specified or ill-defined	trivial			Other ill-defined causes, Causes not specified	trivial
		Haemorrhage (where?)	trivial	Haemorrhage	trivial

By the turn of the century, Registrars and Statisticians had recognised that increases in knowledge and skill were changing the practice of cause-of-death registration. With respect to diarrhoea, a major killer of infants, New South Wales Chief Statistician T. A. Coghlan noted that

> 'The decline in the number of deaths from diarrhoea may be in part due to the fact that of late years more skilful diagnosis in some cases makes possible the ascription of death to ailments of which diarrhoea may be only a symptom' (Coghlan, 1904, p.198).

The Victorian Government Statist E. T. Drake added

> 'Many deaths in earlier years which were attributed to diarrhoeal diseases, would, under modern diagnosis, be classified as enteritis or gastro-enteritis' (Drake, 1908, p.367).

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A similar situation applied to the increases in cancer and circulatory system deaths, and declines in deaths attributed to old age, and atrophy and debility (Fenton, 1901, pp.738, 759; Coghlan, 1904, pp.202, 204).

The skill of the doctor

Closely related to the state of knowledge of medicine, the skill of the doctor who certified a death bore heavily on the accuracy of cause of-death statistics.

Although cause-of-death based on medical certification of death provided the opportunity for more accurate statistics, non-certification did not necessarily exclude a considered medical opinion regarding a cause-of-death. Deputy Registrars who were responsible for recording the details of deaths routinely required attendant doctors to provide a cause-of-death. Doctors were also employed in the Registrar-General's offices to tabulate cause-of-death from the sometimes obtuse and unsatisfactory details supplied by the Deputy Registrars and Coroners' juries. Ultimately, though, the accuracy of the stated cause-of-death depended on the skill of the doctor supplying the cause.

Registration of qualified medical practitioners commenced in New South Wales as early as 1838, followed by legislation in 1855 to ensure minimal standards of education, as well as acceptable qualifications for foreign-trained doctors (Cummins, 1979, p.158). Although the skill of medical practitioners could not readily be regulated or standardised, Cumpston (1989, p.78) considered the standard of medical education in Australia to be high, largely due to the establishment of medical schools in Melbourne and Sydney early in the second half of the nineteenth century.

Nineteenth-century Australian medical practitioners were, however, in an unenviable position. Often they had not attended or observed a death, meaning that they relied on the family or acquaintances of the deceased to obtain information about the course of the final illness needed to identify the cause of death (Williams, 1996). Presuming that they had personal knowledge of the death, diagnosis of cause-of-death was based largely on symptoms. Germ theory had yet to make the transition from European laboratories to local practices. Once the diffusion of new thought began, many doctors were

required to put aside accepted theories of disease causality, and learn new terminologies as well as the use of the new nosologies.

Difficulty in assigning death to a single cause was a challenge, both for physicians and Registrars, since one cause had to be adopted for the purposes of tabulation (Knibbs, 1929). Numbers of contributing diseases or symptoms might be present at death. The dilemma was certainly not solved by the end of the nineteenth century. Bertillon proposed rules for use when two or more causes of death were reported jointly, but consensus on the term 'cause of death', and the development of an appropriate form of medical certificate for the purpose of determining an underlying cause of death took many decades to resolve (Gaminiratne, 2001; Moriyama *et al.*, 2011).

Some medical practitioners were wont to develop habits in certifying particular causes of death. In cases of death from respiratory disease, for example, one practitioner tended to assign 'bronchial pneumonia', whereas another assigned 'bronchitis' (Knibbs, 1907). Doctors were required to stay abreast of current nosological practice. Changing disease labels, seen by some commentators as representing culturally constructed fads or artefacts, were not uncommon in nineteenth-century medical diagnosis, especially if a cause of death went beyond a well-known, but narrow set of infectious diseases (Alter & Carmichael, 1999).

Registration office tabulators generally did not attempt to vary the accuracy of the certificates given to them by physicians (Knibbs, 1929). However, one method used to obtain greater accuracy was the referral system which operated between Registrar's offices and certifying medical practitioners, should a cause-of-death need greater elaboration (Hardy, 1994).

Farr believed that the process of certification itself encouraged greater care in reporting cause of death (Eyler, 1979, p.53). Vague and unsatisfactory causes, however, continued to be employed into the 1880s and beyond (Table 5.2).

Table 5.3: Selected nineteenth-century causes-of-death which were often misdiagnosed

Cause of death	Often misdiagnosed as:	References
Childbirth-associated deaths (Puerperal sepsis, Toxaemia, Haemorrhage, Accidents)	Peritonitis, Pyaemia, Blood poisoning, Haemorrhage, Metritis, Uterus disease, Heart disease, Dysentery	Smith, 1979; Hardy, 1994; Kippen, 2005
Circulatory disease	Old age	Hall, 1906; Hardy, 1994
Diabetes	Tuberculosis	Hardy, 1994
Diphtheria	Sore throat, Quinsy, Angina, Croup, Inflammation of throat, Laryngitis, Tonsillitis, Scarlet fever, Thrush	Gandevia, 1978; Hardy, 1994; Smith, 1999; Hooker & Bashford, 2002; Condran, 2008
Diarrhoeal diseases (Dysentery, Diarrhoea, Cholera, Gastritis, Enteritis, Gastroenteritis)	Typhoid, Typhus, Debility & Atrophy, Failure to thrive, Marasmus, Convulsions, Teething, Dentition, Want of breast milk	Jamieson, 1882c; Gandevia, 1978; Smith, 1979; Lewis, 1979; Hardy, 1994; Smith, 2002; Ferrie & Troesken, 2008
Influenza	Bronchitis, Pneumonia, Other Diseases of the Respiratory System	Fenton, 1901
Joint diseases	Tuberculosis	Gandevia, 1978
Peritonitis	Puerperal fever	Hardy, 1994
Plague	Typhus	Long, 1898
Pneumonia	Old age	Hardy, 1994
Puerperal fever	Accidents of childbirth, Convulsions, Haemorrhage, Rupture of uterus	Jamieson, 1882c
Quinsy	Tonsillitis	Noymer & Jarosz, 2008
Respiratory tuberculosis	Bronchitis, Typhoid, Hydrocephalus, Scrofula, Tabes mesenterica, Lung cancer	Smith, 1979; Walker, 1983; Hardy, 1994
Rheumatism	Rheumatic fever, Rheumatism of the heart	Hardy, 1994; Noymer & Jarosz, 2008
Scarlet fever	Sore throat, Quinsy, Angina, Diphtheria	Gandevia, 1978
Stroke	Paralysis, Hemiplegia, Softening of the brain, Brain paralysis, Paraplegia	
Syphilis	General paralysis of the insane, Locomotor ataxy	Hardy, 1994
Urinary organs	Dropsy	Hardy, 1994

There may well have been an element of wryness when Knibbs (1907) commented

> 'The logical structure of the mind that furnishes 'teething' as a cause of death is not
> profoundly different from one that would enter 'life' as a cause of death. In cases

but 'teething' or 'dentition' remained a listed cause-of-death in all nosologies used in Australia, through to the adoption of the Royal College of Physicians classification by Queensland and Tasmania in 1903.

For some doctors who were trained decades before germ theory, old habits were hard to break. Others rushed to embrace the new theory, often ignoring previously vital factors in diseases causation, such as environment, climate and the patient's constitution (Hardy, 1987). Misdiagnosis was not unusual. Some of the more common mistakes are listed in Table 5.3.

Non-medical factors

The reliability of cause-of-death statistics were subject to a number of non-medical factors, including the influence of close family or influential persons. Deaths from socially sensitive diseases such as tuberculosis, syphilis, suicide and alcoholism were on occasion registered as other causes (Hardy, 1994). Because of the stigma attached to these causes, relatives were often unwilling or unable to accept true diagnoses to be entered on death registrations.

This meant that the real level and increase or decrease of these diseases was, to some extent, an open question. In the case of tuberculosis, what was already a major cause of death may well take on even greater importance.

On statistics of deaths from alcoholism, Victorian Government Statist J. J. Fenton was suspicious,

'…returns of the mortality from intemperance are of doubtful value, as comparatively few deaths are set down to alcoholism pure and simple, although a large number of complaints are no doubt brought on or aggravated, and many lives are doubtless shortened, from that cause, which, however, is not mentioned in the returns' (Fenton, 1901, p.728–729).

And on suicide,

'It should, however, be stated that the death records frequently do not upon the surface show that the death has been suicidal, and close examination, with sometimes further inquiry, is therefore necessary to determine that fact. It is hence

likely that the full extent to which suicide prevails in some of the colonies is not ascertained' (Fenton, 1901, p.750).

AVAILABILITY OF CAUSE-OF-DEATH DETAIL WITHIN COLONIES

Differences in the reporting of cause-of-death statistics across the colonies present challenges for comparisons. The colonies began their reporting at different times, with different levels of detail, and using different nosologies.

Western Australian Registrar George Frederick Stone was the first to routinely publish cause-of-death statistics in his annual reports, commencing with the year 1841–42. Farr's system was not used, however,

> 'The nomenclature of diseases, aptly said to be of as much importance as weights and measures in the physical sciences, is also attended with difficulty at present; and it is next to impossible, without the assistance of the medical gentlemen in the several districts, to enter the causes of death uniformly in the same names—local terms of equivocal meaning having been frequently sent to this office; much must therefore be left to future improvement, to attain anything like perfection in this and other respects' (*PG*, 7 Oct. 1843, p.3).

Following Stone's resignation in 1854, reporting in Western Australia fell into abeyance. In 1859, Registrar Alfred Durlacher published in his census report a table which compiled deaths registered from 1854–1859, distributed by sex, age group and cause (Durlacher, 1859, p.31). The table did not include information for individual years, and neither was Farr's classification employed. The 1870 census report repeated this exercise, and included a table on deaths by cause and sex, this time for the individual years 1860 to 1869, and now classified in accordance with Farr's system (Knight, 1870, p.69).

From 1870 to 1885, tables on principal causes of death classified by sex were published in the *Western Australian Blue Book*. From 1886 to 1896, two tables were published annually; one reported deaths using Farr's broad classes and age groups, and another reported on detailed cause for each quarter of the year. There was no reporting by sex. Only in 1897 did Registrar-General Malcolm A. C. Fraser begin to report deaths by detailed cause, age group and sex in the *Statistical Register of Western Australia*.

South Australia began classifying deaths by cause almost from the commencement of registration, publishing the returns in their Legislative Council papers (Holton, 1983; and see also S. A. Almanack, 1849, p.35; Duncan, 1850, p.29–30; SAR, 19 Jul. 1850, p.4). However, as Handasyde Duncan, the Health Officer for Port Adelaide observed,

> 'For the first three months of that year [1848] there was some uncertainty about the names of the diseases, as no one in the Registrar's office was acquainted with medicine, but during the subsequent months, a gentleman who had previously studied medicine has superintended the returns, and to his intelligence and care we are indebted for the minute information contained in the return' (Duncan, 1850, p.28).

Indeed, the information is minute, and useful, even though Farr's classification was not used. Abstracts for the years 1856 to 1861 using Farr's system, and classified by sex, age group and cause of death, appeared in the *South Australian Government Gazette*. From 1862 these abstracts were published in *Statistics of South Australia*, later the *Statistical Register of South Australia*. For the years 1868–1872, age group is not specified, meaning that only crude rates of death can be calculated.

Victoria was a leader among the colonies in many areas of statistical reporting, and it used Farr's classification from the outset. However, for the first ten years following the establishment of registration, detailed statistics on causes of death were not published. For the years 1853–54 to 1857–58, the abstracts aggregated causes of death into broader classes. A more detailed return for the thirteen years 1853–1865, by sex and individual cause of death (but not age group) was published in the 1865 edition of *Statistics of Victoria*.

Registrar-General Archer's monthly reports on Melbourne and suburbs published numbers of deaths by detailed cause in the *Victorian Government Gazette* from 1860 onwards. Only from 1864 onward did the abstracts of mortality in the *Statistics of Victoria* contain detailed cause of death information by sex and age group.

In the early years following the introduction of registration in New South Wales, the Registrar-General followed Victoria's example. Returns for the years

1856 to 1862 did not include individual causes of death, but instead aggregated deaths into classes. Returns from 1863 to 1874 also aggregated deaths, but not into classes – instead, they were combined into groups and sub-groups. Diseases such as smallpox, typhus and cholera were grouped as 'Miasmatic diseases', with only the total for the group being shown. Historical epidemiologist and first Director-General of the Australian Department of Health J. H. L. Cumpston (1880–1954) noted with irritation that 'As a result, it is now impossible to learn how many deaths occurred from any individual cause for the years concerned' (1989, p.79). Only from 1875 were detailed returns by sex, age group and cause of death published for New South Wales.

In Queensland, the early abstracts of mortality also aggregated deaths into Farr's classes. From 1864 to 1869, the statistics suffered the same disadvantage as in New South Wales, being shown only in groups, and not individually. Individual causes by sex and age group first appear for the year 1870.

In Tasmania, causes of death were not included in the statistical abstracts of mortality before 1868. However, from the early 1850s, local doctor Edward Swarbreck Hall (1804–1881) compiled and published monthly summaries of meteorological and health conditions for Hobart, and for some years, the whole of Tasmania (Curson, 1985b). These summaries included classifications of deaths by cause, using Farr's system (see, for example, Hall, 1863, 1864b; Abbott, 1872). Hall also corresponded with the Joint Committee of the Royal College of Physicians in London prior to the publication of their 1869 Nomenclature of Diseases (Cumpston, 1989).

In 1867, the new Tasmanian Statistician E. C. Nowell compiled diseases and deaths occurring in charitable institutions using Farr's classification, and published the return in *Statistics of Tasmania*. From 1868, this was extended to cover all deaths in Tasmania, and showed sex, age group and cause of death.

It is evident that the colonies, in reporting cause-of-death, varied in their level of detail. Age-standardised rates for causes of death classified using Farr's system can only be calculated when the level of detail includes individual cause of death, standard age groups and sex, *viz*., for New South Wales from 1875,

Victoria from 1864, Queensland from 1870, Western Australia from 1897, South Australia from 1856 (but with a hiatus from 1868 to 1872) and Tasmania from 1868.

For earlier years of less detail, there is enough information in the abstracts of mortality to calculate the crude death rate for individual causes of death classified according to Farr's system. Crude death rates for males and females can be calculated for New South Wales for the years 1856–1862, Victoria 1853–1863, Queensland 1860–1863 (but only for all persons), Western Australia 1860–1885 (and for all persons 1886–1896), and South Australia 1868–1872. Because these are crude rates only, their comparability between colonies and over time is compromised.

A cause-of-death measure for 'Australia'

Calculating age-standardised rates for individual causes of death for the whole of Australia from 1856 to 1906 is problematic, due to varied availability of cause-of-death information within each colony. Strictly speaking, this is not possible before 1897, the year in which detailed data for Western Australia became available.

In this work, for the years 1864–1896, a proxy measure is adopted to represent Australia. It is comprised of available data for as many colonies as possible, ensuring that at least one major colony (New South Wales or Victoria) is included. Thus, the age-standardised mortality rate for 'Australia' is composed of:

- From 1864 to 1867: Victoria and South Australia
- From 1868 to 1869: Victoria and Tasmania
- From 1870 to 1872: Victoria, Queensland and Tasmania
- From 1873 to 1874: Victoria, Queensland, South Australia and Tasmania
- From 1875 to 1896: New South Wales, Victoria, Queensland, South Australia and Tasmania.

From 1897 to 1906, data from all colonies (New South Wales, Victoria, Queensland, Western Australia, South Australia and Tasmania) are available.

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GROUPING CAUSES OF DEATH

Changes in cause-of-death classifications lead to difficulties in determining real changes in patterns of death over time. During the half-century from 1856 to 1906, five different nosologies were employed in the Australian colonies, sometimes simultaneously (Table 5.1). Revisions of these nosologies, along with changes in death certification practices, pose problems for the interpretation of mortality time series.

A number of methods have been developed to deal with the issues which arise from changes in the production of mortality data. Three main kinds can be distinguished: bridge-coding (where a large number of death certificates are coded twice, applying the nosology prevailing before and after any revision), time series 'jump' analysis (a statistical detection and correction of abrupt changes in cause-specific mortality trends), and the use of concordance tables and cause recombination (Rey *et al.*, 2011).

Because of the lack of information in nineteenth-century mortality statistics needed for bridge-coding or jump analysis, the latter approach is adopted in this work. Recombining causes to develop concordance tables consists of determining consistent cause categories — guided by medical knowledge — for successive nosology revisions. These consistent categories aim to unite the different classifications (e.g. Preston, 1976; Kintner, 1986; Wolleswinkel-van den Bosch, 1996; Naghavi *et al.*, 2010; Kippen, 2011). Although there is much commonality, the underlying classifications of death differ in detail, and so each analysis requires a new schema to group causes of death over the time period under study. This thesis also requires a new schema, since there has been no previous attempt to develop a concordance table which unites the nosologies that were in use in the colonies between 1856 and 1906.

The requirements of a schema are such that:

1. causes of death should be distinct and mutually exclusive on epidemiological grounds,
2. causes of death should be nosologically continuous for the period under study,

3. as many deaths as possible should be placed in categories of known causes, and as few as possible in residual categories,

4. diseases with similar processes or symptoms, which might easily be mistaken for each other, should be grouped together,

5. categories should be broad enough to be constructed from available data, and should be relatively insensitive to temporal differences in diagnosis and coding,

6. categories should reflect causes which are important in the context of the epidemiological transition, and hence should include communicable diseases, non-communicable diseases and external causes of death.

To maintain nosological continuity, some schemas partition single causes of death into two or more categories when it is believed that they contain a mixture of underlying causes. This is not attempted here, since it requires either a bridge coding study, where deaths in one year are coded to both an old nosology and its revision, or expert nosological knowledge (e.g. Vallin & Meslé, 1990; Taylor, 1992; Meslé & Vallin, 1996). Bridge coding studies are available for some countries for more recent ICD revisions, but not for nineteenth-century nosologies. It is more than likely that the lack of precision in nineteenth-century cause-of-death reporting would render moot any attempts at bridge coding.

Valuable information is provided by the indexes of each nosology. These indexes generally include terms that were used in prior nosologies, allowing correspondences to be built between the current and prior cause-of-death terminologies. In addition, some of the English Registrar-General's Reports contained reference tables of diseases which were grouped differently in old and new lists of causes of death (Registrar-General of England, 1883, 1903).

In attempting to unite nosologies, due regard should be given to the warnings of social historians who caution against using modern-day criteria to retrospectively diagnose the diseases of yesteryear (Risse, 1997). Although the historical significance of the statistics is paramount, for the reasons discussed above they should be approached with 'a high degree of caution and scepticism' (Luckin, 1980; Hardy, 1994).

With these caveats in mind, a cause-of-death schema has been developed which arranges the detailed causes of the five different nosologies used by the colonies over the period 1856 to 1906 into 28 broad categories (Table 5.4).

Table 5.4: Arrangement of 19th and early 20th century Australian nosologies into a broad cause-of-death schema

Cause-of-death	Archer, 1853	Archer, 1863	Hayter, 1886	Royal College of Physicians, 1901	Bertillon, 1907
1. All infectious diseases	1-7, 9-12, 15-17, 19-27, 33, 38, 42, 44, 46, 53-54, 73-74, 79-80, 100, 120, 129, 131	I-1-1 to 3a, 6-8, 12-18 I-2-1 to 5 I-4-1 to 3 II-2-1 to 5 III-1-7 III-4-1, 2 IV-1-5, 6 IV-4-1	I-1-1 to 7, 9 to 15 I-2-1 to 3 I-3-1 to 3 I-4-1 to 4 I-5-1 to 2 I-6-2, 3 II-1 to 4 IV-2, 6 to 9, 14 VI-1-6, 9, 11 VI-5-2, 6, 8 VI-11-3 VIII-2	I-1-1 to 9, 11 to 17 I-2-1 to 5 I-3-1 to 4 I-4-1 to 2 I-5-7 to 9, 12 I-6-1 to 9 I-7-1 to 4 I-12-6 II-1-3, 7, 9 II-6-4, 6, 7 IV-1	1-9a, 11-24, 26-38, 62, 67, 71-72, 104, 105-107, 111-111a, 151, 179a,b
2. Typhoid fever	17, 19	I-1-7	I-1-14	I-1-16	1
3. Diphtheria	5	I-1-3a	I-1-11	I-1-13	9-9a
4. Measles	3	I-1-2	I-1-3	I-1-4	6
5. Respiratory tuberculosis	73	II-2-3, 3a	IV-8	I-6-1, 2	26-27
6. Other tuberculosis	33, 46, 129	II-2-1, 2, 4, 5	IV-6, 7, 9 VI-11-3	I-6-3 to 9	28-35
7. Scarlet fever	4	I-1-3	I-1-5	I-1-6	7
8. Whooping cough	7	I-1-6	I-1-9	I-1-11	8
9. Diarrhoea, dysentery, cholera, enteritis	10-12, 79-80	I-1-12, 13, 14 III-4-1, 2	I-2-1, 1a, 2, 3 VI-5-6, 8	I-2-1, 2, 3, 4 II-6-4, 6, 7	12-14a, 104, 105-106
10. Convulsions, teething	54, 74	III-1-7 IV-1-5, 6	VI-1-9 VI-5-2	I-12-6 II-1-7	71, 179b
11. Atrophy, debility	38, 42	IV-4-1	VIII-2	IV-1	151, 179a
12. Neoplasms	35-37	II-1-3, 3a	IV-5 VIII-4	I-10-1, 2, 3 II-9-1, 3 IV-4	39-46, 53, 129, 131
13. Endocrine, nutritional, metabolic diseases	14, 96, 136	I-3-1, 2, 3, 5 III-5-4	III-1, 1a, 2 IV-4, 12 VI-6-3, 4	I-8-1, 2 I-11-1, 5 I-12-5 II-7-3, 4	25, 49-52, 89, 153
14. Nervous system	45, 50-52, 55-57	III-1-1, 4, 5, 6, 8	VI-1-1, 4, 5, 7, 8, 12, 13	II-1-1, 5, 6, 10, 11, 12, 13	60-61a, 63, 66, 69-70, 73-74
15. Circulatory	47-48, 58-66	III-1-2, 3 III-2-1, 2, 3	IV-1 VI-1-2, 3 VI-2-2 VI-3-1 to 11 VI-6-1	I-5-5 I-9-1, 2 II-1-2 II-2-2 II-3-1 to 7 II-4-1 to 8 II-7-2	64-65, 77-86

(continued)

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Table 5.4: Arrangement of 19th and early 20th century Australian nosologies into a broad cause-of-death schema (*continued*)

16. All respiratory	8, 13, 67-72, 76	I-1-4, 5, 11 III-3-1 to 6	I-1-8 VI-1-10 VI-4-1 to 8 VI-5-3	I-1-10 I-5-6 II-1-8 II-5-1 to 12 II-6-1, 2	10, 87-88, 90-99a
17. Bronchitis, emphysema, asthma	69, 72	III-3-2, 5	VI-4-4, 5	II-5-5, 9	90-91, 96-98
18. Influenza, pneumonia	13	I-1-11 III-3-4	I-1-8 VI-4-6	I-1-10 I-5-6 II-5-6, 7, 8	10, 92-93
19. Digestive	75, 77-78, 81-91	II-1-4 III-4-3 to 15	VI-5-1, 4, 5, 7, 9 to 20	II-6-3, 5, 8 to 15	100-103, 104a, 108-110, 112-114, 116-118
20. Genitourinary	92-95, 97-99, 101-103	III-5-1 to 3, 5 to 7 III-6-1, 2 IV-2-1	VI-7-1 to 8 VI-8-1 to 6	II-8-1 to 5 II-9-2, 4, 5, 6	119-128, 130, 132-133
21. Pregnancy, childbirth	18, 104	I-1-9 IV-2-2	I-6-4 VI-9-1 to 6	I-5-1 to 4 II-10-1 to 5	134-141
22. Perinatal, congenital	43	IV-1-1 to 4	V-1 to 7	I-12-1 to 4	150, 150a, 151a, 152
23. Injury, poisoning, external causes	40, 137-145	V-1-1 to 7 V-2-1 to 3 V-3-1 V-4-1 to 5 V-5-1 Violence n.c.	VII-1-1 to 8 VII-2-1 to 2 VII-3-1 to 6 VII-4 VII-5-1 to 2	I-8-5 to 7 V-1-1 to 20 V-2-1 to 3 V-3-1 to 9 V-4-1	57-59, 155-176c
24. Accidents	n.a.	V-1-1 to 7	VII-1-1 to 8	I-8-5 to 7 V-1-1 to 20	57-59, 164-176
25. Intentional self-harm	n.a.	V-4-1 to 5	VII-3-1 to 6	V-3-1 to 9	155-163
26. All ill-defined, unspecified causes	28, 30, 39, 41	II-1-2, 5, 6 IV-2-3 IV-3-1 Sudden deaths N.S or ill-def.	IV-14 V-8 VIII-1, 3, 6, 7, 8	IV-2, 3, 6 to 9	55, 142, 154, 177-179
27. Old age	41	IV-3-1	V-8	IV-2	154
28. All other causes	29, 31-32, 34, 49, 87*, 105-119, 121-128, 130, 132-135	I-1-10 I-3-4a,b II-1-1 III-4-16 III-7-1, 1a, 2 III-8-1 to 3	I-6-1 III-3a,b IV-3, 10, 11 VI-2-1, 3 VI-6-2 VI-10-1 to 3 VI-11-1, 2, 4 to 7 VIII-5	I-8-3, 4 I-9-3 to 5 I-11-2 to 4 II-1-4 II-2-1, 3 II-7-1 II-11-1 to 3 II-12-1 to 4 IV-5	47-48, 54, 56, 68, 75-76, 115, 143-149

Note: Details of nosologies can be found in Appendix 4.

The grouping of certain diseases in the above schema was based on the following criteria:

Tuberculosis

For tuberculosis, a major nineteenth-century cause of death, a distinction is made between Respiratory Tuberculosis (including such terms as 'Tuberculosis' and 'Phthisis') and Other Tuberculosis (including 'Tabes mesenterica', 'Scrofula' and 'Tubercular meningitis'). Archer's 1853 nosology placed phthisis in the group with pneumonia, bronchitis and other diseases of the respiratory system, and 'many doubtful cases of lung disease were classified as phthisis' (Cumpston, 1989, p.277). Archer's 1863 nosology reclassified tuberculosis as a constitutional disease. Apart from changes in disease definition, tuberculosis was also often misdiagnosed (Table 5.3). Both Respiratory Tuberculosis and Other Tuberculosis are here grouped among infectious diseases. Deaths from tuberculosis are discussed more fully in Chapters 6 and 7.

Diarrhoea, dysentery, cholera, enteritis

These gastrointestinal infections are here grouped together, but they were divided among zymotic and digestive diseases in nineteenth-century nosologies. Again, often misdiagnosed (Table 5.3), they were a major cause of death among infants. Infant mortality is discussed more fully in Chapter 7.

Convulsions, teething

These ill-defined and unsatisfactory terms often appear together, and were leading causes of death among infants. They represent a collection of symptoms which were predominant in summer and highly seasonal in nature.

In the absence of more accurate diagnosis and certification, deaths from gastrointestinal infections were regularly attributed to convulsions or teething (Armstrong, 1905; Lewis, 1979). Deaths from gut infections were often coincident with teething in infants, and convulsions often manifested in the final stages of illness. Convulsions in infancy

> "...are excited by mere peripheral irritations, as during the process of teething...Diarrhoea, worms, etc, also take their place as more or less frequent

excitants of convulsive paroxysms in infants...In children the exalted irritability of the nervous system during dentition predisposes to diarrhoea from slight determining causes...Diarrhoea, which is a very common complication of dentition, is especially frequent during the warmer months...A catarrhal condition of the bowels should be cured as quickly as possible, especially during dentition...the danger of severe choleraic diarrhoea being set up is therefore correspondingly threatening" (Quain, 1885, p.299–304, 341–342, 357–361).

Convulsions were originally classed as a disease of the nervous system. Both convulsions and teething are here grouped among infectious diseases.

Atrophy, debility

These terms are also ill-defined and symptomatic. Again, deaths ascribed to these causes occur most commonly among infants. Atrophy and debility often described dehydration or emaciation, being a 'want of nourishment...a wasting of the whole body...a gradual interference of nutrition' and in children these were 'due, in the majority of cases, to chronic functional derangements which interfere with the digestion and elaboration of food' (Quain, 1885, p.95). These derangements included unsuitable food, and chronic vomiting or diarrhoea. As with convulsions and teething, atrophy and debility were closely linked to gastrointestinal infections by early paediatricians (Armstrong, 1905; Litchfield, 1905; Lewis, 1979; Mein Smith, 1997, pp.41–45).

Some recent commentary has suggested that these causes might not correspond solely with infant diarrhoea or other gastrointestinal infections. 'Weanling diarrhoea' seemed most often to occur between 6 and 18 months, often being associated with the transition from breast feeding, and linked to protein-calorie malnutrition (Kunitz, 1983). Mein Smith (1997, p.43) suggests that half of all deaths from convulsions should appear elsewhere, and that a fraction of atrophy deaths should also be excluded to allow for weak babies who faded away from improper food. McCalman *et al.* (2011) develop this theme, arguing that among impoverished babies born in the Melbourne Lying-In Hospital, causes such as 'debility', 'marasmus' and 'inanation' comprised a separate entity which was different from gastrointestinal infection, and which showed little seasonal variation. Instead, infants who 'failed to thrive' died from a

syndrome exacerbated by feeding difficulties, improper food and emotionally detached or ill-prepared mothers. But to which cause of death, then, do these determinants contribute?

Regardless of the pattern of infant mortality in the Lying-In Hospital, deaths from atrophy and debility—including those occurring in Melbourne and suburbs—clearly display seasonality (Figure 5.1). This suggests that infectious diseases were indeed responsible for a large proportion of these deaths.

Figure 5.1: Monthly deaths from atrophy and debility, Sydney and Melbourne, 1880–1889
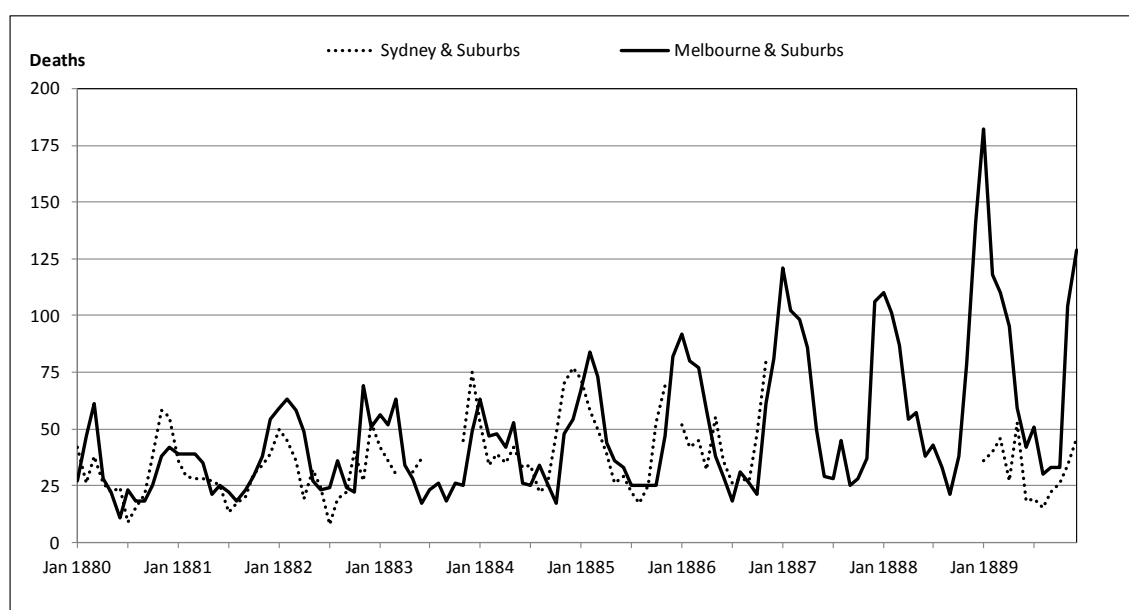
These views can be somewhat reconciled, since 'failure to thrive' and gastrointestinal infections might conceivably be co-morbid. Babies who were weak and emaciated through feeding problems were easily carried off by waves of gastrointestinal infections that swept through the colonies each summer.

The underlying cause of death of infants dying from atrophy or debility remains unclear. Opinion to date has favoured gastrointestinal infections, but if 'failure to thrive' initiated the train of events leading to death, then a proportion of these deaths should perhaps appear under the rubric 'Endocrine, nutritional and metabolic diseases'. In any event, 'underlying cause' was a concept which was not employed in the nineteenth-century abstracts of mortality.

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With these qualifications in mind, atrophy and debility are here provisionally grouped among infectious diseases.

Old age

Although neither symptom nor cause, old age is a risk factor for a number of chronic diseases. A wholly unsatisfactory term, it was commonly attributed as a cause of death in nineteenth-century Australia.

From the outset, both Farr and Archer had reservations regarding its use,

> 'There is reason to believe that many of the diseases of the aged are not detected; and that the terms, "Old Age," and "Natural Decay," are often incorrectly assigned as causes of death. Pneumonia is a very frequent cause of death in the aged; and can be detected by the physical signs, though its other symptoms may be latent' (Archer, 1856).

No attempt is made to reassign 'old age' deaths in the above schema.

All other causes

This residual category includes causes with small numbers of deaths that do not appear elsewhere in the schema. Were they to be assigned to contemporary criteria, they would appear among Diseases of the Blood and Blood-Forming Organs, Mental and Behavioural Disorders, Diseases of the Eye, Diseases of the Ear, Diseases of the Skin and Subcutaneous Tissue, or Diseases of the Musculoskeletal System. Causes of death from Diseases of the Blood and Blood-Forming Organs and Mental and Behavioural Disorders were poorly developed in nineteenth-century nosologies.

This schema should be considered indicative only. Since there are no rules of translations from historic to contemporary nosologies, demographers are required to have a close understanding of the times and places they are studying, and the ways in which causes of death were understood and recorded (Kunitz, 1999). More detailed work, uniting both demographic and medical history, would result in an improved and more informed arrangement (Alter & Carmichael, 1996; Risse, 1997; Woods, 2007).

SUMMARY

Cause of death was a requirement of the registration laws. Attendant doctors, occupants of houses where persons had died, or other persons present at death were required to give cause-of-death particulars to Deputy Registrars as part of the process of registering a death. Cause-of-death was also ascertained through coronial or judicial inquests. Medical certification of death, introduced gradually throughout the colonies, provided greater cause-of-death accuracy, although within the limitations of nineteenth-century medical knowledge.

Systems of disease classification, or nosologies, allow for the production of comparable cause-of-death statistics. The Australian colonies closely followed England in their adoption; W. H. Archer had circulated Farr's nosology in Victoria as early as 1853, with other colonies following suit in its adoption. Archer and his deputy H. H. Hayter published a revised nosology in 1863, and in 1886 Hayter adopted the new Royal College of Physicians classification. Some colonies chose to adopt a further revision of the College nosology during the first decade of the twentieth century. New South Wales was first to use the Bertillon classification for the statistics of 1906.

Although they are the primary source for the study of disease and death, nineteenth-century cause-of-death statistics have numerous shortcomings. Theories of disease causation underwent considerable change during this time; the emergence of germ theory revolutionised medical knowledge, ultimately reflected in changing classifications and cause-of-death abstracts. The quality of the statistics largely reflects the skills of the doctors who determined cause of death, and misdiagnosis was not uncommon. Non-medical factors such as the deliberate miscoding of socially sensitive diseases add further compromise.

In addition to questionable data, comparisons of cause-of-death statistics are complicated by the differential adoption of nosologies by the colonies, and the varying amounts of detail published in their abstracts of mortality. Compiling longitudinal cause-of-death data for the whole of Australia requires uniting different classifications and harmonising colonial reporting. To this end, a schema has been developed for use in this thesis.

Chapter 6

Cause-specific mortality, 1856–1906

Most nineteenth-century deaths in Australia can be classified to a relatively small number of nosological categories which corresponded to the leading causes of death. Close inspection of rises and falls in deaths from these causes, and an appreciation of the role that each cause played in the overall decline in mortality provides a basis for understanding the late nineteenth-century phase of the mortality transition.

Thirteen of the twenty-eight causes which form part of the cause-of-death schema developed in Table 5.4 are examined in detail in this chapter. These causes represent some of the most prevalent health problems in nineteenth-century Australia. In order, they include infectious diseases—airborne epidemics most commonly affecting children (diphtheria, whooping cough, measles and scarlet fever) followed by tuberculosis, water-borne gastrointestinal infections and typhoid. Chronic non-communicable diseases—cancer, circulatory, respiratory and digestive diseases—are discussed, and then maternal mortality and external causes of death.

A number of indicators are used. Trends in age-standardised death rates and in crude rates where age-standardised rates are not available show how causes varied over time, by age group and by sex, along with their proportional

contribution to all-cause mortality. Commentary on the historical context in which these deaths occurred helps in understanding their role in a rapidly changing epidemiological milieu.

DIPHTHERIA

Diphtheria arrived in Victoria and Tasmania late in 1858 as a 'new, brutal mystery', probably being introduced via migrants who had left England in the middle of the same year (Smith, 1999). The first Australian death ascribed to 'diphtheria' occurred in Melbourne on 20 October 1858, and deaths were also registered in Tasmania early in the following year. The disease soon spread to other colonies; South Australia in 1859, and Queensland and Western Australia in 1860. Records in New South Wales are unclear, but Sydney's Health Officer Henry Graham, reported that the disease was present (*SMH*, 27 Sep. 1859, p.3). At first, diagnosis was often confused with croup, scarlatina, quinsy or even measles.

The initial outbreak in Victoria was virulent, with the crude death rate rising from 55 per 100,000 population in 1859 to 120 in 1860. This was the highest rate recorded in Australia, apart from a late 1860s outbreak among the small Western Australian population. Diphtheria was epidemic in Victoria during the years 1859–70 and again in 1889–90, as well as in Queensland in 1873, Western Australia in 1864–69, 1874 and 1883, South Australia in 1859–72 and 1888–91, and Tasmania in 1875–80 (Figure 6.1).

Diphtheria assumed an endemic form following its explosive introduction. The total death rate for Australia fluctuated, but declined overall from 1860 to 1886 (Figure 6.2). Between 1887 and 1890, all colonies experienced serious epidemics, but after 1890 rates declined rapidly.

In most years, female mortality rates were higher than male. Diphtheria averaged around two per cent of total Australian mortality over the entire period, although in epidemic years it approached five per cent. Children under five were most affected, with diminishing incidence to age 15 (Figure 6.3). Case notifications assume a different profile, with 20-30 per cent under five years of

age, 40-50 per cent between five and 15, and the remainder among older age groups (Cumpston, 1989, p.296). In the 1860s and 1870s, the case-fatality rate was around one-in-three or -four, although in epidemic years, Victorian case-fatality reached as high as 50-60 per cent (Smith, 1999). Deaths usually peaked in early winter.
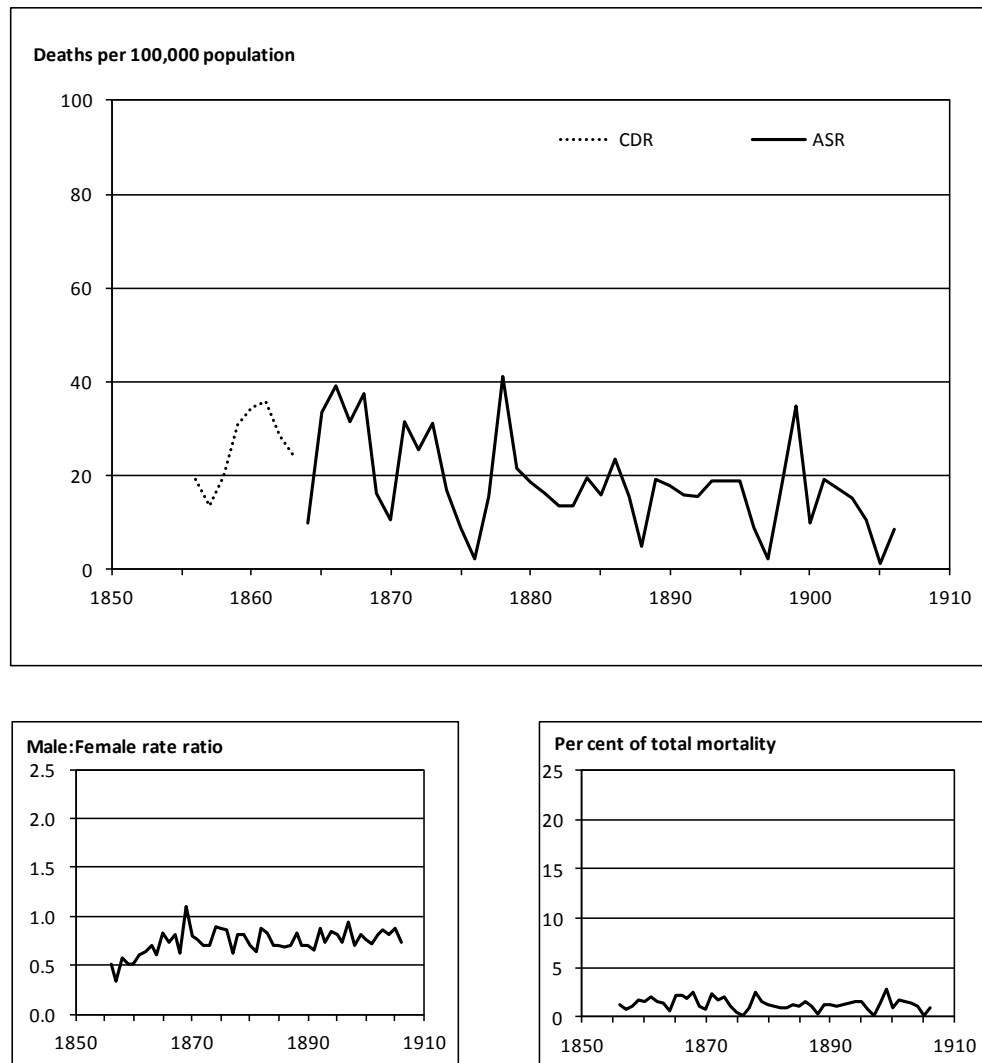
Figure 6.1: Diphtheria mortality rates, by colony, 1853–1906

Note: Mortality rates are age-standardised to the 1881 total Australian census population.

Dr. J. A. Moore was first to report the disease in Australian medical literature, with his account of the January 1859 New Norfolk, Tasmania outbreak (*AMJ*, Jul. 1859, p.166–169). Victorian Registrar-General Archer's weekly abstracts also documented the unfolding epidemic in Melbourne,

> 'Catarrhal afflictions have been greatly on the increase both in and around the metropolis. At Brunswick several cases of quinsy of a severe form occurred this week…A child under 3 at Collingwood died of "diphtheria and exhaustion". In the Flemington district malignant sore throat has attacked young and old…At Richmond two cases of death occurred in one family within a few minutes of each other; the sufferers being children of the respective ages of 2 and 4 years old, and the disease in both instances, ulcerated sore throat' (*VicGG*, 11 Mar., 19 May, 23 Sep. 1859).

Figure 6.2: Diphtheria mortality rate, Australia, 1856–1906
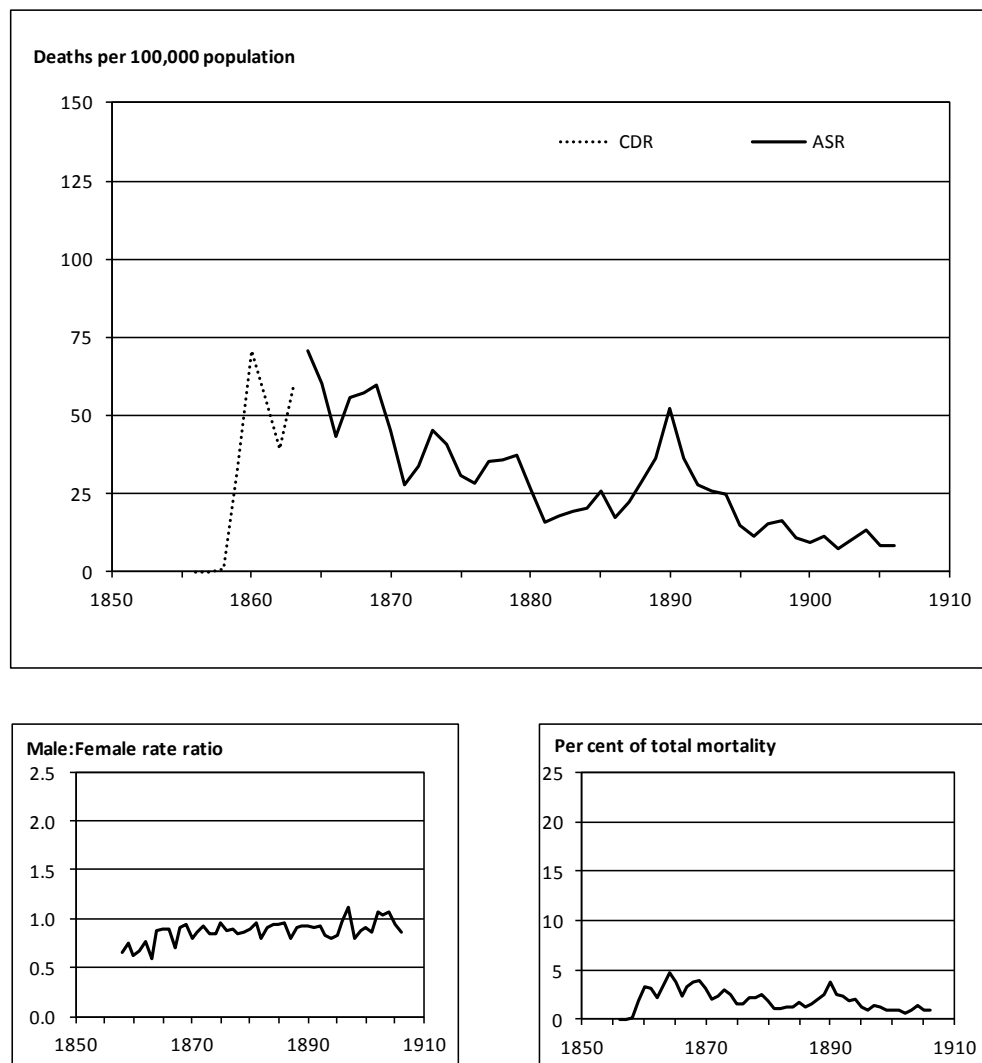
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

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A Royal Commission into diphtheria was launched in Victoria in 1871. The findings shed much light on early thinking about the disease — it concluded (erroneously) that the disease had spread to Victoria from Tasmania; that unsanitary conditions did not cause the disease (and hence that it was not miasmatic) but that filth could make attacks more severe; that it was a distinct disease, although often co-morbid with scarlet fever, rubella or measles; and that it was contagious (Smith, 1999). Dr William Thomson used the opportunity to downplay the effect of climate and to promote germ theory, arguing for house quarantine and fumigation (*AMJ*, Jul. 1872, pp.193–211).

Figure 6.3: Age-specific mortality rates for diphtheria, Australia, 1875–79 and 1900–04
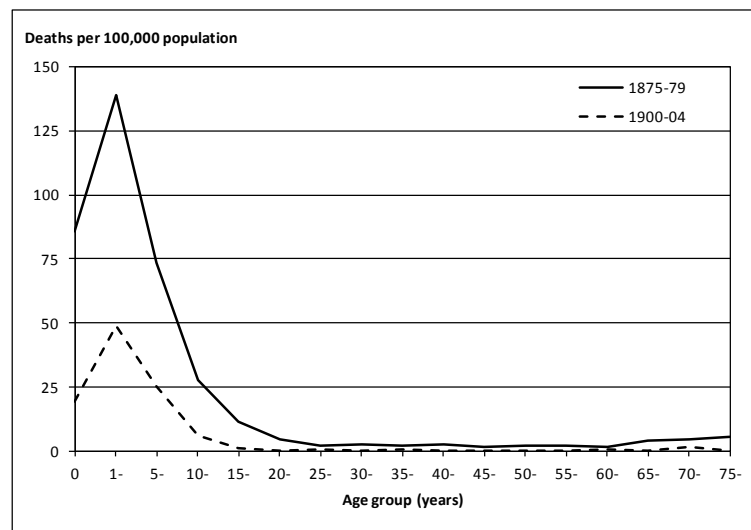
In 1877, Blair published a resume of papers on diphtheria and included a table of deaths in Victoria by age and sex for the years 1858–1876, supplied by Victorian statist H. H. Hayter (*AMJ*, Oct. 1877, pp.298–301).

The Klebs-Loeffler bacillus (*Corynebacterium diphtheria*) was isolated in Germany in 1884 and identified as the cause of the disease. In the 1890s, Emil von Behring developed an antitoxin that did not kill the bacterium, but neutralized its toxic poisons. Antitoxin was first used in Australia by Springthorpe in 1895, and by 1906 most cases were treated by this method. The death rate fell to less than 10 per 100,000 population, although it would rise again during the early decades of the twentieth century (Turner, 1899a, b; Cumpston, 1989, p.293).

Diphtheria was made notifiable in New South Wales, South Australia and Western Australia in 1898 and in the other colonies shortly thereafter. It was one of the first diseases to become closely identified with the new science of bacteriology and laboratory-based public health (Hooker & Bashford, 2002).

Figure 6.4: Injecting the serum (*Australian Town & Country Journal*, 19 Jan. 1895)

Figure 6.5: Death sits on the unclean tank (*The Boomerang*, 16 Mar. 1889)

WHOOPING COUGH

Whooping cough was known in all the Australian colonies by the middle of the nineteenth century, and was endemic, with regular epidemics occurring at three-to-four yearly intervals. Mortality rates were high in New South Wales in 1878 and 1899; in Victoria in 1865–66, 1871, 1873, 1878 and 1899; in Queensland in 1870, 1873 and 1878–79; in Western Australia in 1864–65, 1869, 1873–74, 1881 and 1886; in South Australia in 1867–68, 1872, 1883, 1890, 1893 and 1898–99; and in Tasmania in 1868, 1872, 1874, 1880, 1899 and 1903 (Figure 6.6).

Figure 6.6: Whooping cough mortality rates, by colony, 1853–1906
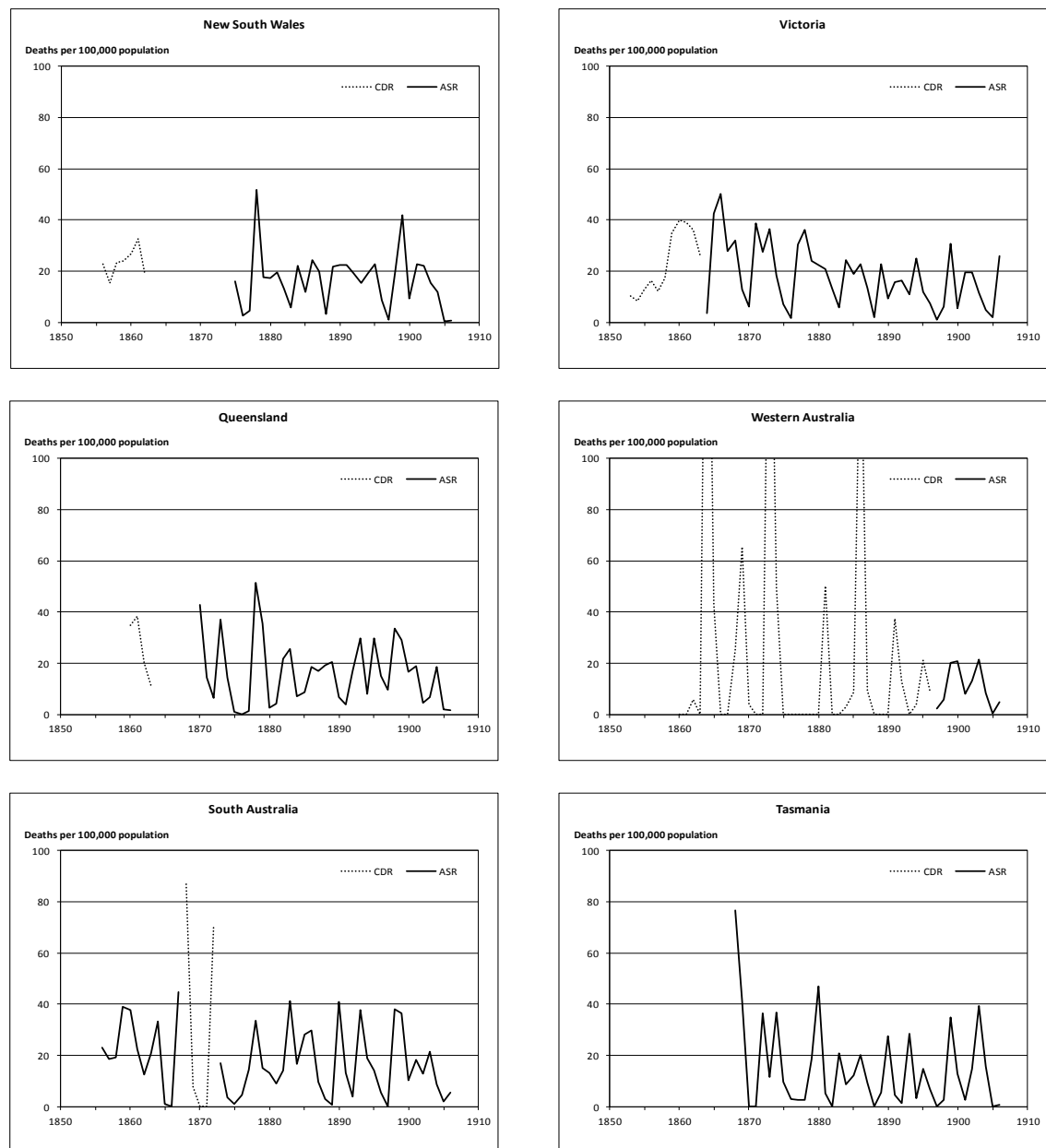
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

Cumpston (1989) identified three phases of whooping cough, each having its own general level of mortality. Before 1880 epidemic death rates were high, from 1880 to 1900 epidemic mortality was lower, and from 1900 mortality was lower still, with less-pronounced fluctuations and higher inter-epidemic mortality than was previously the case (Figure 6.7).

Figure 6.7: Whooping cough mortality rate, Australia, 1856–1906

Note: Mortality rates are age-standardised to the 1881 total Australian census population.

Female mortality was generally higher, of the order of 25 per cent. Whooping cough averaged between one and two per cent of total mortality over the period. Over 95 per cent of all deaths occurred under the age of five years;

infants were most affected, with a diminishing incidence to age five years (Figure 6.8).

Figure 6.8: Age-specific mortality rates for whooping cough, Australia, 1875–79 and 1900–04
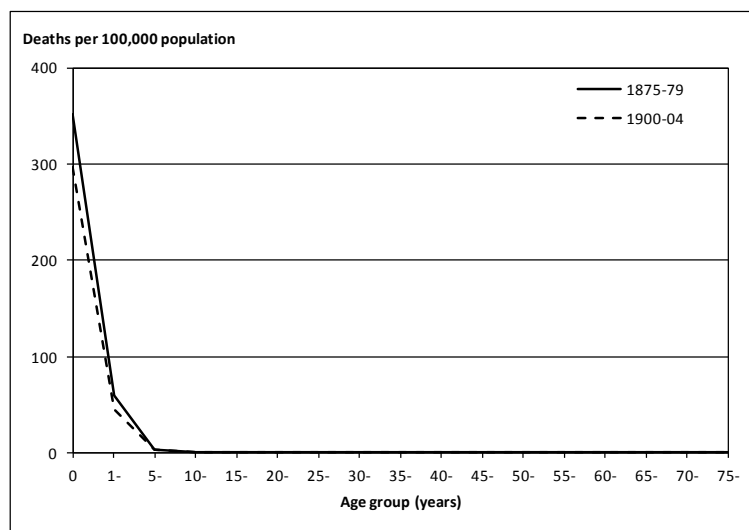
Although other infectious diseases received more medical and public attention, whooping cough was a major hazard to children. An 1867 epidemic, together with measles, caused high mortality in Sydney, with 77 deaths in the Destitute Children's Asylum alone, this being around 10 per cent of all the children in care (Gandevia, 1978). The epidemic 'presented itself in its severest form, affecting chiefly the youngest children, and prevailing to an alarming extent' (*Empire*, 22 May 1867, p.5). The first cases appeared on 10 March, and by 20 March over 400 were affected. The deceased were buried in the Asylum's burial ground.

Victorian figures suggest that around half of all city children had had whooping cough by the age of five years, and 63 per cent by age 15. Among country children, only one-third had the disease by age five, but 83 per cent by age 14 years (Gandevia, 1978, p.83). Case fatality rates of one-to-three percent were usual in late nineteenth and early twentieth century Australia, occasionally rising to six per cent. Vaccines were not available until 1920 and mass immunisation began in the 1940s (Cumpston, 1989, p.312).

MEASLES

Although earlier outbreaks are suspected, measles made a definite appearance in Victoria in July 1850, through its introduction by the ship *Persian*. It assumed epidemic form in 1853–54 during the Victorian gold rush, spreading also to New South Wales in 1853, Tasmania in 1854, Queensland in 1857, South Australia in 1859 and Western Australia in 1860.

Figure 6.9: Measles mortality rates, by colony, 1853–1906
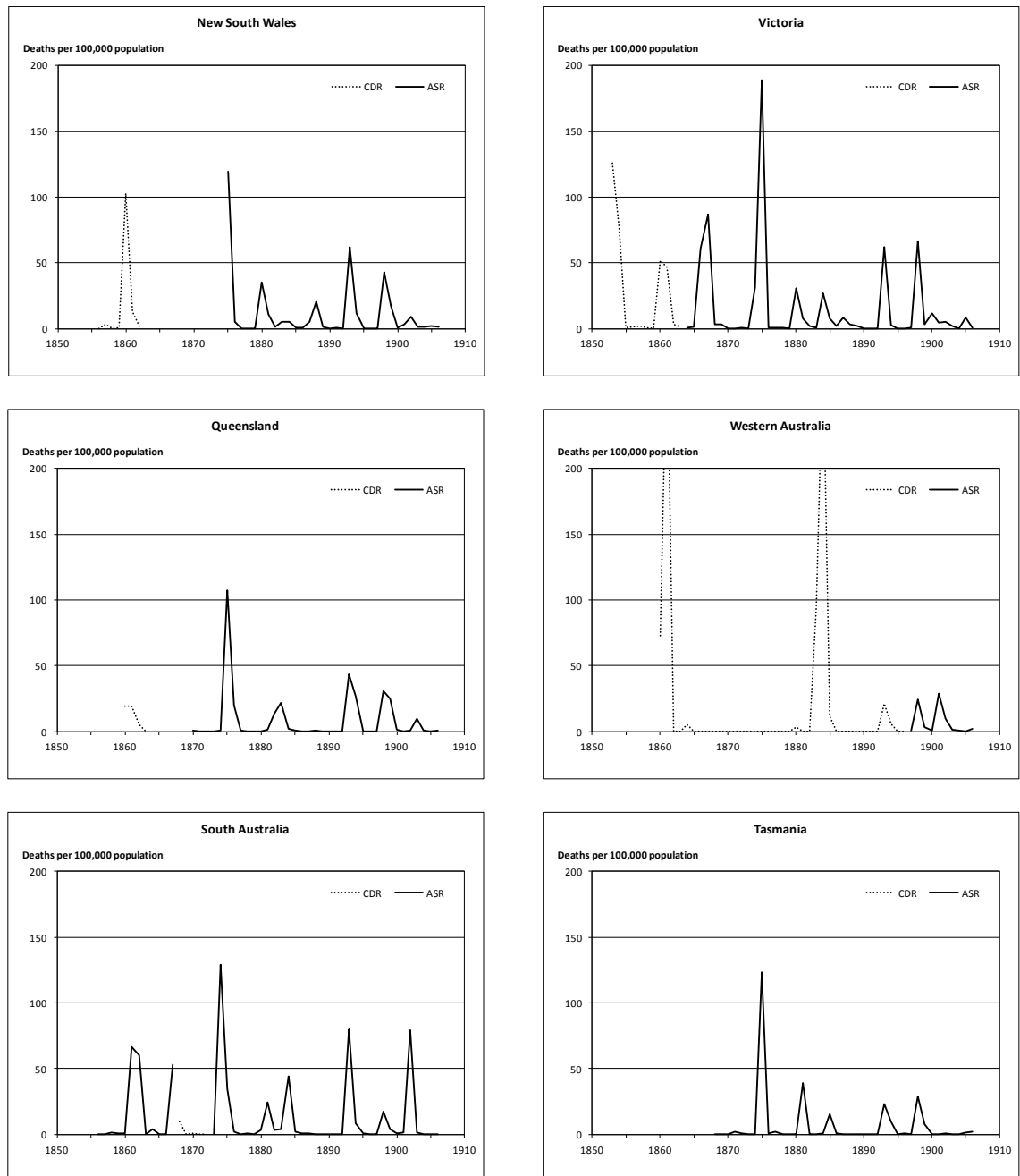
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

The disease exhibited distinct epidemic features, with mortality rising to a high rate and falling to previous levels within a short time. Measles epidemics occurred simultaneously in many of the colonies at intervals of five-to-six years (Table 6.1). Since a single bout produces life-long immunity, a new cohort of susceptible children was needed before the disease could reassert itself. The 1860, 1866–67 and 1874–75 outbreaks were especially severe (Figure 6.9) with the latter two possibly interconnected with English manifestations, since 1866 and 1874 were epidemic years there (Curson, 1985a).
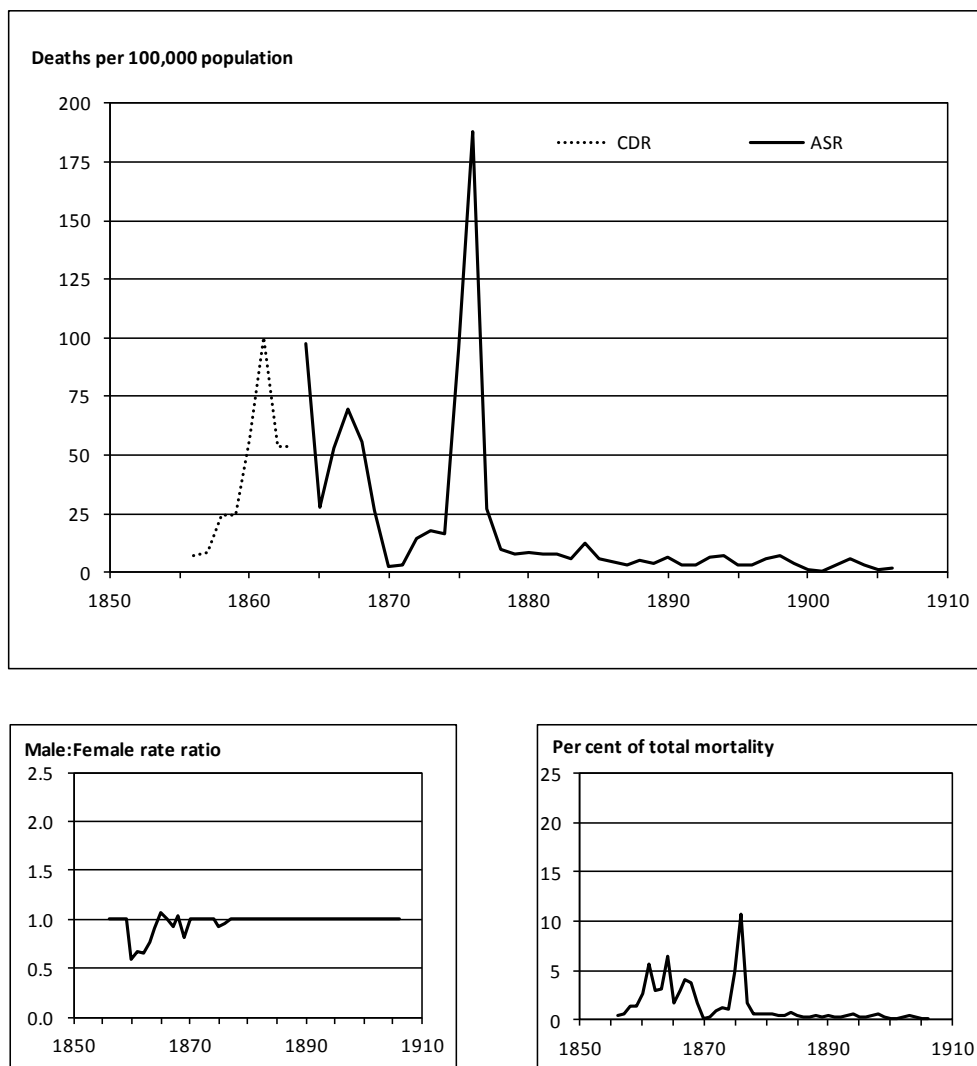
Figure 6.10: Measles mortality rate, Australia, 1856–1906
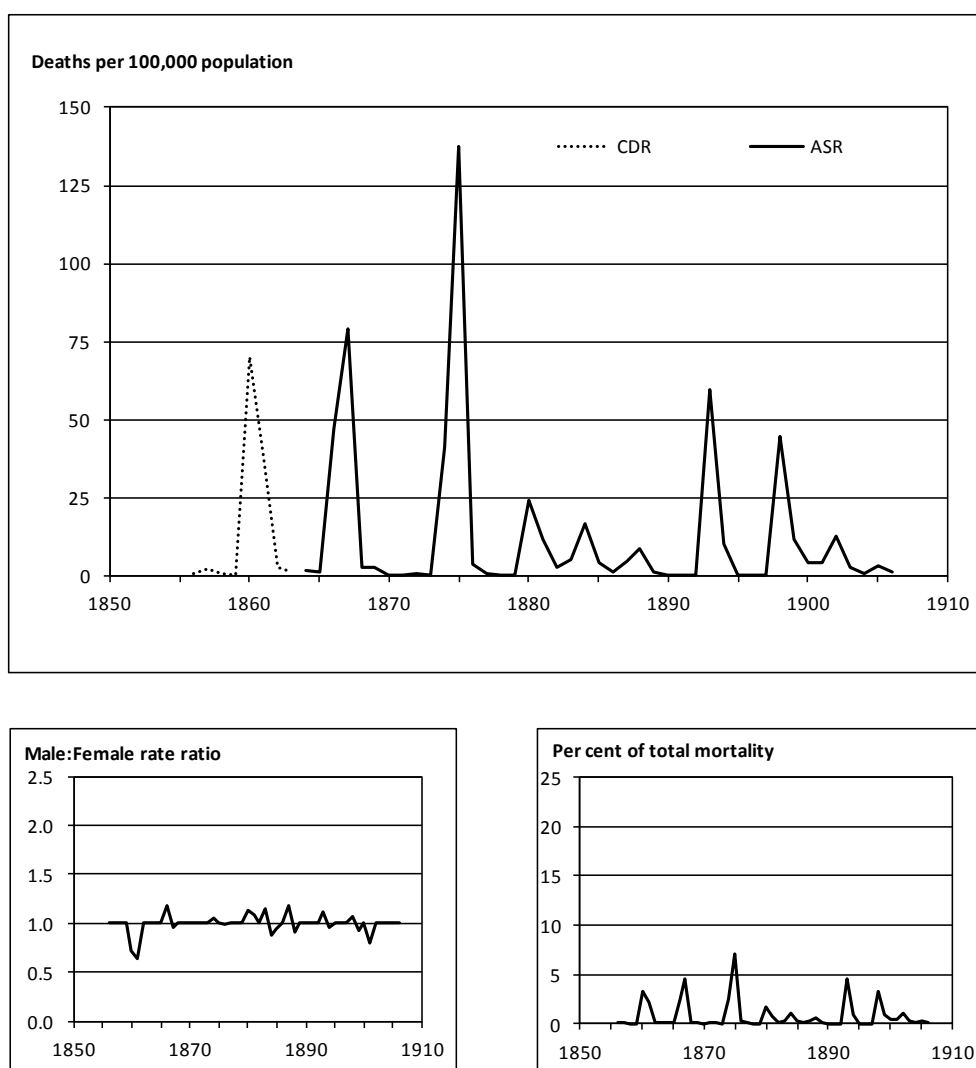
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

Male and female mortality rates were similar in years of significant measles occurrence. Measles accounted for five per cent or more of total mortality in

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epidemic years (Figure 6.10). Most deaths occurred among young children with a rapidly diminishing incidence after age five years. Infants under one year were largely exempt because of the *in utero* transfer of antibodies from the mother (Figure 6.11).

Figure 6.11: Age-specific mortality rates for measles, Australia, 1875–79 and 1900–04
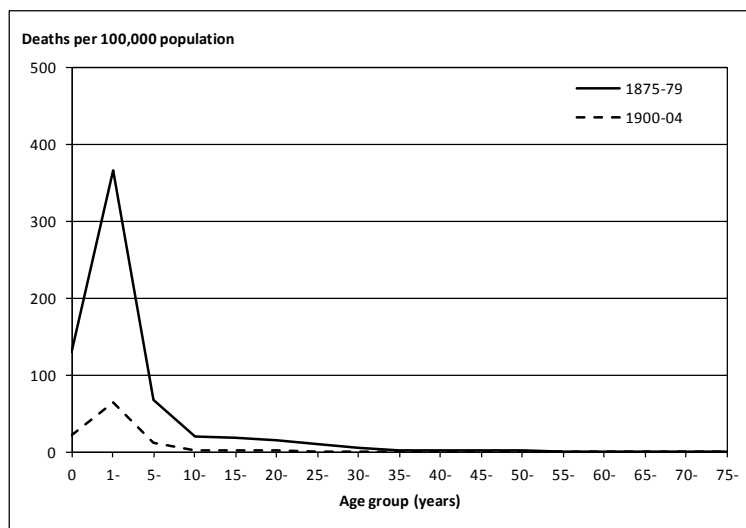
Although later accepted as commonplace, measles was feared in the nineteenth and early twentieth century, especially among the urban poor. A bout of measles usually led to weakness and weight loss and if not causing death outright, often triggered other gastrointestinal or respiratory diseases.

Table 6.1: Measles epidemics in Australia, 1860–1906

New South Wales	Victoria	Queensland	Western Australia	South Australia	Tasmania
1860	1860	1860	1860	1861-62	1861
1867	1866-67	n.a.	—	1868	1867
1874-75	1874-75	1875	—	1874-75	1875
1880-81	1880	—	—	1881	1881
—	—	1882-83	—	—	—
1883-84	1884	—	1883-84	1884	1885
1888	—	—	—	—	—
1893	1893	1893	1893	1893	1893-94
1898	1898	1898	1898	1898	1898-99
1902	1905	1903	1901	1902	1905-06

Curson (1985a) nominates the 1867 epidemic as 'possibly the greatest childhood disaster of the nineteenth century' in Australia. The monthly reports of the Registrars General summarise the unfolding mortality (Figure 6.12).

Figure 6.12: Measles mortality in Sydney and Melbourne during 1866–67
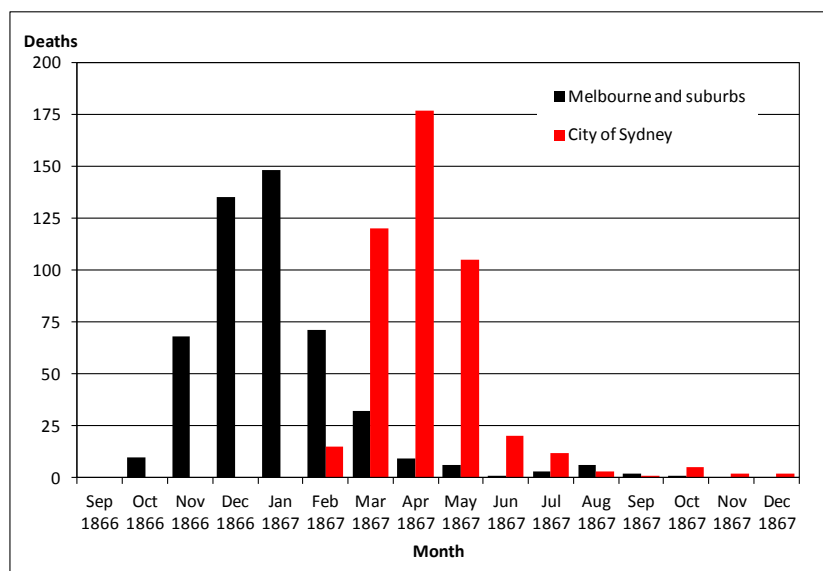
The epidemic in Melbourne preceded that in Sydney, with December 1866 and January 1867 the worst months. Measles, along with scarlet fever, killed over 1,200 persons in Victoria in 1867. In his weekly report of 3 January, Registrar-General Archer reported that,

> 'Twenty-one out of thirty deaths registered in Collingwood were of children under three years of age. Sixteen out of the thirty-five deaths registered in South Melbourne occurred in public institutions…Measles still very prevalent and fatal in most districts. The general report shows a very unsatisfactory condition of public health' (*VicGG*, 11 Jan. 1867, p.82).

In the Sydney Metropolitan Area, perhaps 13,000 young children (or 70 per cent of the total) caught the disease, with over 700 deaths. Almost half of under-five deaths in 1867 were caused by the epidemic. The poor suffered most, especially in the dense working-class neighbourhoods extending from the lower Rocks to the Haymarket. Multiple deaths within a household were not uncommon, and a significant proportion occurred within institutions, including the Benevolent Asylum and Destitute Children's Asylum.

SCARLET FEVER

Scarlet fever first appeared in Australia in 1833, becoming more virulent from the 1840s, and establishing itself in every colony by 1860, with the possible exception of Western Australia. The disease was widely confused with diphtheria until the mid-1860s. Scarlet fever was severe in Victoria during 1853, 1860–62 and 1866–68, in South Australia during 1863–64 and in Tasmania during 1843 and 1853 (Kippen, 2002a). The great scarlet fever epidemic of 1875–76 occurred in every colony, except Western Australia (Figure 6.13).

Figure 6.13: Scarlet fever mortality rates, by colony, 1853–1906
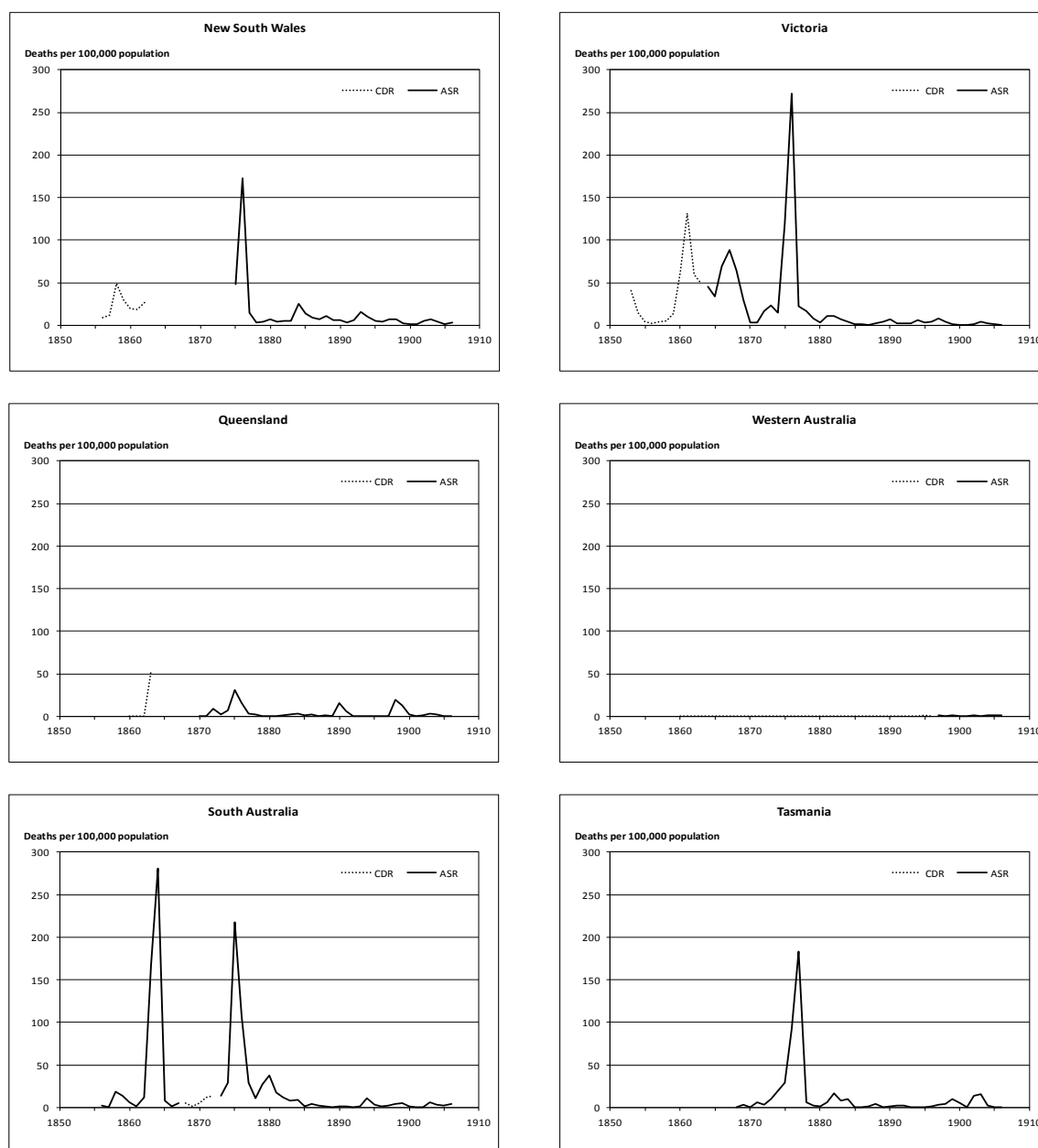
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

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Between 1865 and 1880, epidemics occurred regularly, and from then on mortality declined rapidly (Figure 6.14). In the years of significant scarlet fever occurrence before 1865, female mortality rates were higher than male. Scarlet fever accounted for five per cent or more of total Australian mortality in epidemic years, but in 1876 it exceeded ten per cent.

Figure 6.14: Scarlet fever mortality rate, Australia, 1856–1906

Note: Mortality rates are age-standardised to the 1881 total Australian census population.

Although most commonly attacking the 5–14 year age group, scarlet fever was more often fatal among young children aged 0–4 years, with a diminishing incidence from age five years (Figure 6.15). By the turn of the twentieth century, death rates were low among all ages, including among children.

Figure 6.15: Age-specific mortality rates for scarlet fever, Australia, 1875–79 and 1900–04
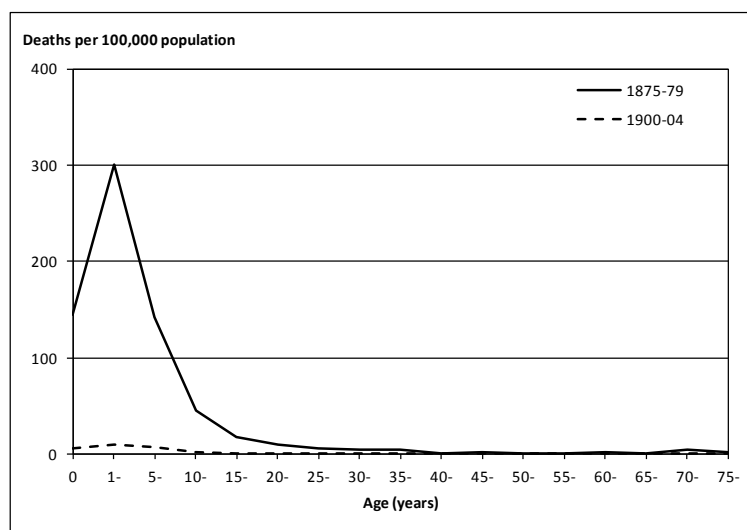
The 1875–76 epidemic, which extended into 1877 in Tasmania, claimed over 6,000 lives, and led to the deaths of many more through associated illnesses. It was a 'dramatic event in the lives of the people' (Cumpston, 1989). In Sydney, the epidemic was especially virulent from the second week of November 1875 until June of the following year, with the proportion of monthly deaths consistently in excess of 20 per cent of all deaths. There was estimated to be 8,000–10,000 cases in the city alone (Curson, 1985a).

The disease spread rapidly in country Victoria early in 1876,

> 'The spread of scarlet fever in the Castlemaine district is becoming very serious. Many fatal cases are reported, and children going to bed well are dead in the morning...The *Courier* states that such is the terror caused by the rapid spread of this disease, that the attendance of the children at the State and private schools in Ballarat has fallen off about one-third during the last few days' (*SMH*, 23 Mar. 1876, p.3).

The epidemic produced widespread public and official reaction with a renewed concern for health and sanitation, since it was widely perceived to be a miasmatic or 'filth' disease. Scarlet fever became notifiable in New South Wales, Western Australia and South Australia in 1898, Queensland in 1900, Tasmania in 1903 and Victoria in 1916.

TUBERCULOSIS

In late nineteenth-century Australia, tuberculosis was recognised as 'the most deadly disease that the world knows in the present generation' (Mullins, 1898). Between 1856 and 1906, some 150,000 deaths caused by pulmonary and other forms of tuberculosis (such as scrofula, *tabes mesenterica* and tubercular meningitis) were registered in Australia.

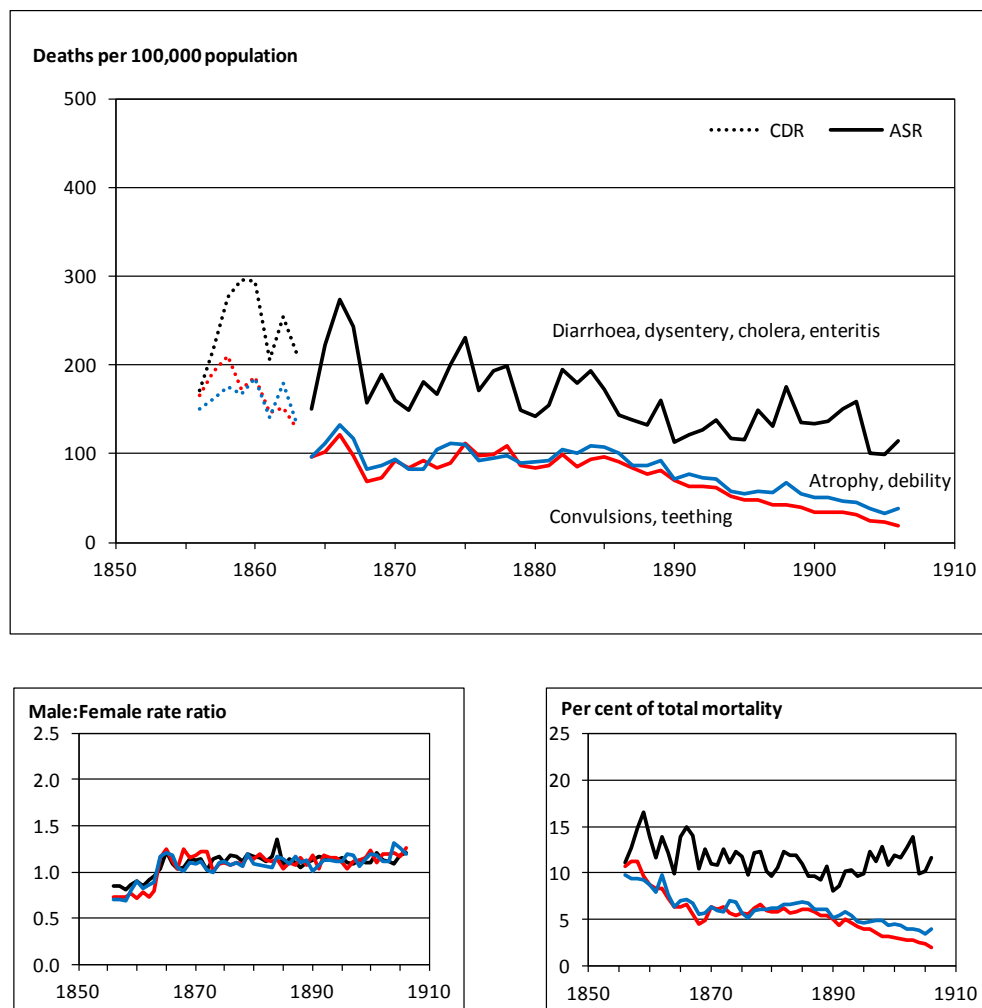**Figure 6.16: Tuberculosis mortality rates, by colony, 1853–1906**
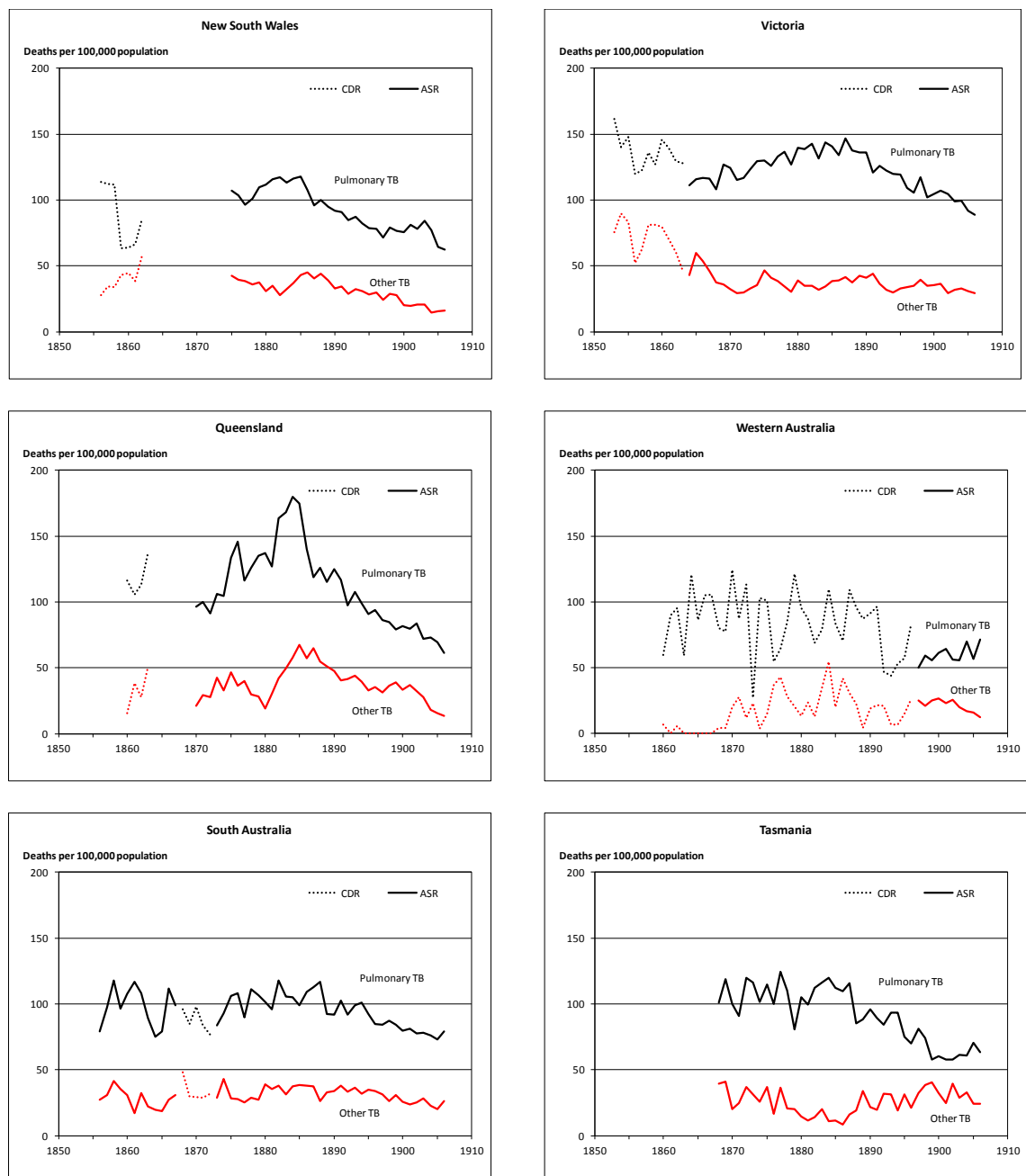
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

In New South Wales during 1885–96, approximately the same number of deaths were registered from *phthisis* (here understood as referring to pulmonary tuberculosis) as from the six chief zymotic diseases — smallpox, measles, scarlet fever, whooping cough, diphtheria and typhoid fever — combined.

Early data before the 1860s are questionable since phthisis was recorded as a respiratory disease (see Appendix 4), leading H. H. Hayter, the Victorian Government Statist, to conclude that many cases of lung disease were classified as phthisis.

Figure 6.17: Tuberculosis mortality rate, Australia, 1856–1906
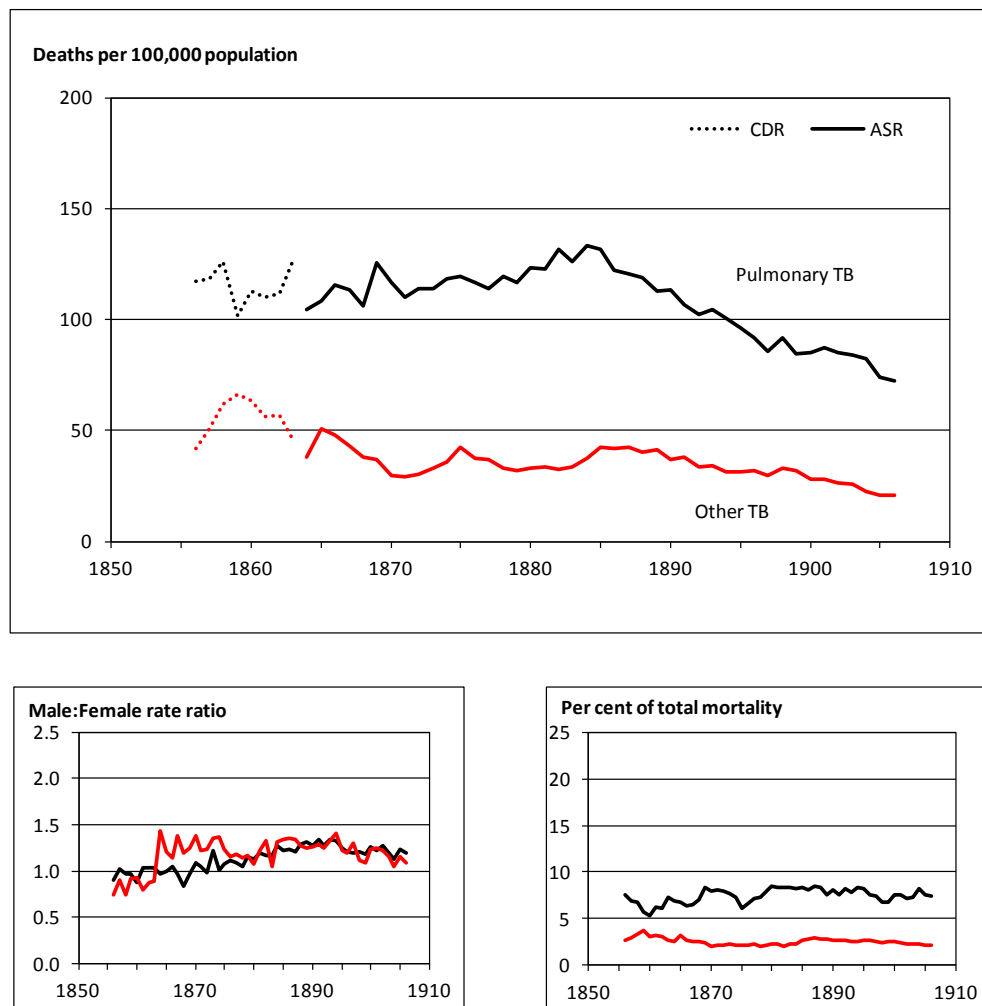
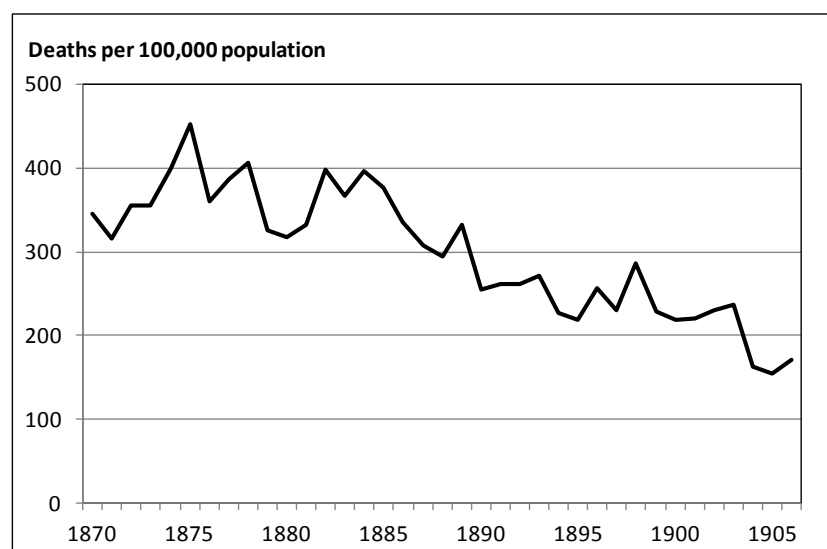*Note:* Mortality rates are age-standardised to the 1881 total Australian census population.

From 1862, two distinct phases are apparent in the major colonies; death rates from pulmonary tuberculosis rose steadily until the mid-1880s and fell rapidly

thereafter (Figures 6.16 and 6.17). In Western Australia and Tasmania the course was different, although Tasmania also experienced a decline from the mid-1880s.

For most of the period, male mortality rates were higher than female rates. Tuberculosis was consistently responsible for ten per cent of total Australian mortality from year-to-year.

Tuberculosis was most fatal in the prime of life. Age-specific death rates were lowest at 5–9 years of age and rose quickly until middle age (Figure 6.18). Rates then vary; in the late 1870s they plateaued and fell rapidly after age 55, whereas in the early 1900s they declined until age 50, rose again until age 65 and then declined. Caution has been advised in comparing changes in age patterns of period-based TB mortality rates, because of so-called cohort effects.

Figure 6.18: Age-specific mortality rates for tuberculosis, Australia, 1875–79 and 1900–04
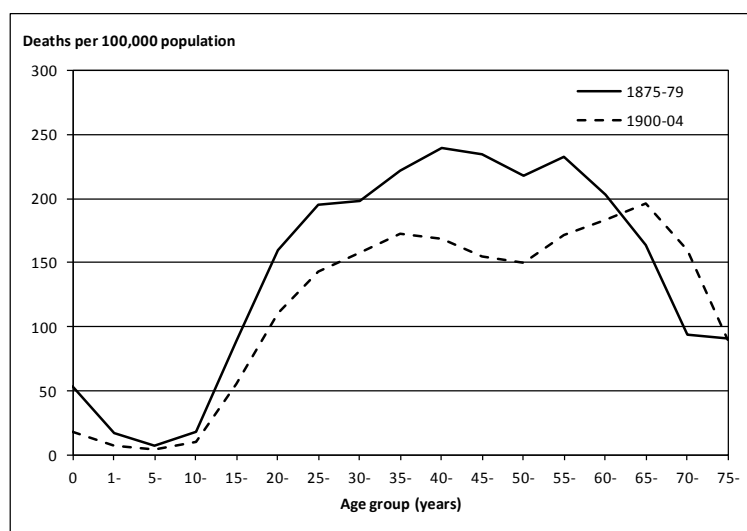
Although adverse social conditions such as poverty, overcrowding and malnutrition were often linked to tuberculosis, it affected all social classes in nineteenth-century Australia. The disease's heavy death toll and its major role in the mortality transition require that it be considered in further detail, and this follows in Chapter 7.

GASTROINTESTINAL INFECTIONS

Diarrhoea, dysentery, cholera and enteritis—the diseases here included under the heading 'gastrointestinal infections'—were collectively the largest cause of mortality in nineteenth-century Australia. Their toll greatly exceeded that of tuberculosis, which was commonly held to be the major killer of the day (Litchfield, 1905). The lack of regard for their deadly harvest was perhaps due to the fact that it was mostly infants, and not adults, who were affected.

Figure 6.19: Gastrointestinal infections mortality rates, by colony, 1853–1906
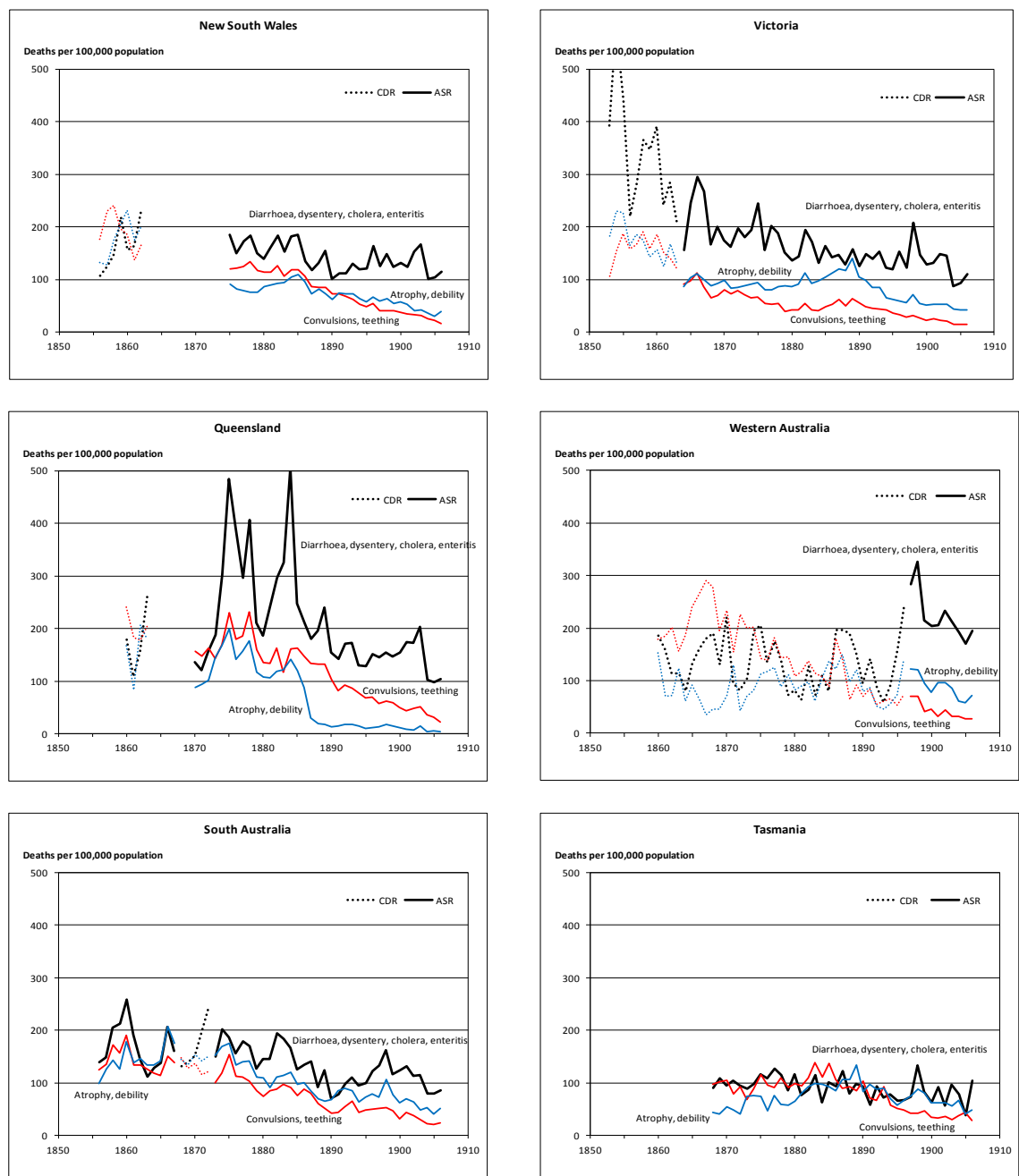
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

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As high as they were, deaths recorded under these causes hid much additional mortality. Previously it was argued that many deaths from 'convulsions', 'teething', 'atrophy' and 'debility' should also be added to gastrointestinal infections. To add further uncertainty, there was misclassification between diarrhoeal diseases and typhoid (Cumpston, 1989, p.226). What is not in doubt, however, is that mortality from these causes was large, summing in any year to between 20 and 30 per cent of total mortality (Figure 6.20).

Figure 6.20: Gastrointestinal infections mortality rate, Australia, 1856–1906

Note: Mortality rates are age-standardised to the 1881 total Australian census population.

W. H. Archer, commenting on the mortality of children in Victoria as compared with the United Kingdom, observed,

'But, on the other hand, dysentery, of a character hardly known at home, and diarrhoea and convulsions are, at certain seasons, such successful weapons in the hands of death, in Victoria, as to demand all the wisdom of the wisest, and the best skill of the skilful, in hygiene and medicine, to stay their frequent preventable ravages' (Archer, 1859).

Extremely high mortality rates from diarrhoea, dysentery, cholera and enteritis—above 300 deaths per 100,000 population—were recorded for goldfields Victoria to 1860, and in Queensland for most years between 1875 and 1885. For Australia as a whole, deaths stood at around 200 per 100,000 population until the mid-1880s, a rate which eased only somewhat thereafter (Figures 6.19 and 6.20).

Much larger falls occurred among deaths ascribed to convulsions, teething, atrophy and debility. However, greater accuracy in diagnosis and certification meant that deaths which were previously allocated to these causes were being reassigned to other recognised causes such as diarrhoea or enteritis, downplaying any real decline in the latter (Armstrong, 1905). Taken as a whole, the decline in mortality from all gastrointestinal infections after the mid-1880s was considerable (Figure 6.21).

Figure 6.21: Mortality rate for all gastrointestinal infections combined, Australia, 1870–1906

Note: Includes deaths from diarrhoea, dysentery, cholera, enteritis, atrophy, debility, convulsions and teething. Mortality rates are age-standardised to the 1881 total Australian census population.

Gastrointestinal infections were seasonal in nature, and 'summer diarrhoea' came to be feared, especially among the urban working class (Stawell, 1899). Their activity began as summer commenced, and terminated with the conclusion of autumn. Initially, the 'diarrhoea and enteritis' group was urban rather than rural in its fatal incidence (Cumpston, 1989, p.227). Whereas urban mortality declined after the mid-1880s, rural rates remained unchanged; by 1900 city and country death rates were similar (Gandevia, 1978, p.89). The diseases cut a swath through infants, and to a lesser extent, older persons (Figure 6.22).

Figure 6.22: Age-specific mortality rates for diarrhoea, dysentery, cholera and enteritis combined, Australia, 1875–79 and 1900–04
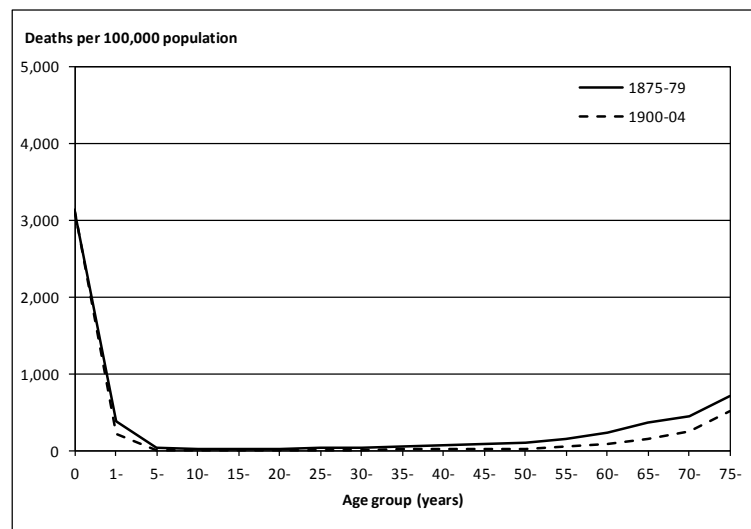
Considerable attention was paid by Australian investigators to determine the identity of 'diarrhoea'. It was variously seen as being caused by unhygienic conditions, by dietetic causes such as lack of breastfeeding, unsuitable food or poor milk supplies, or by climatic influences (Mitchell, 1893; Stawell, 1895; Armstrong, 1905; Lewis, 1982). Only much later were the roles of viruses ascertained, particularly rotavirus, and bacteria including *E. coli* and *Campylobacter*, transmitted via contaminated water, food or poor hygiene. The foundation of disease management, long sought for but not grasped by Victorian-era medicine, was simple rehydration with an electrolyte solution.

TYPHOID

Typhoid fever, also known as 'colonial', 'continued', 'low', 'bilious', 'remittent', 'gastric' or 'enteric' fever, was introduced into Australia with European colonisation. Sporadic cases were recorded during the first half of the nineteenth century, but from the 1850s typhoid became endemic (Cumpston, 1989; Smith, 2002). Typhoid was often confused with diarrhoea or dysentery, especially in earlier years, and among young children (Barrett, 1883).

Figure 6.23: Typhoid mortality rates, by colony, 1853–1906
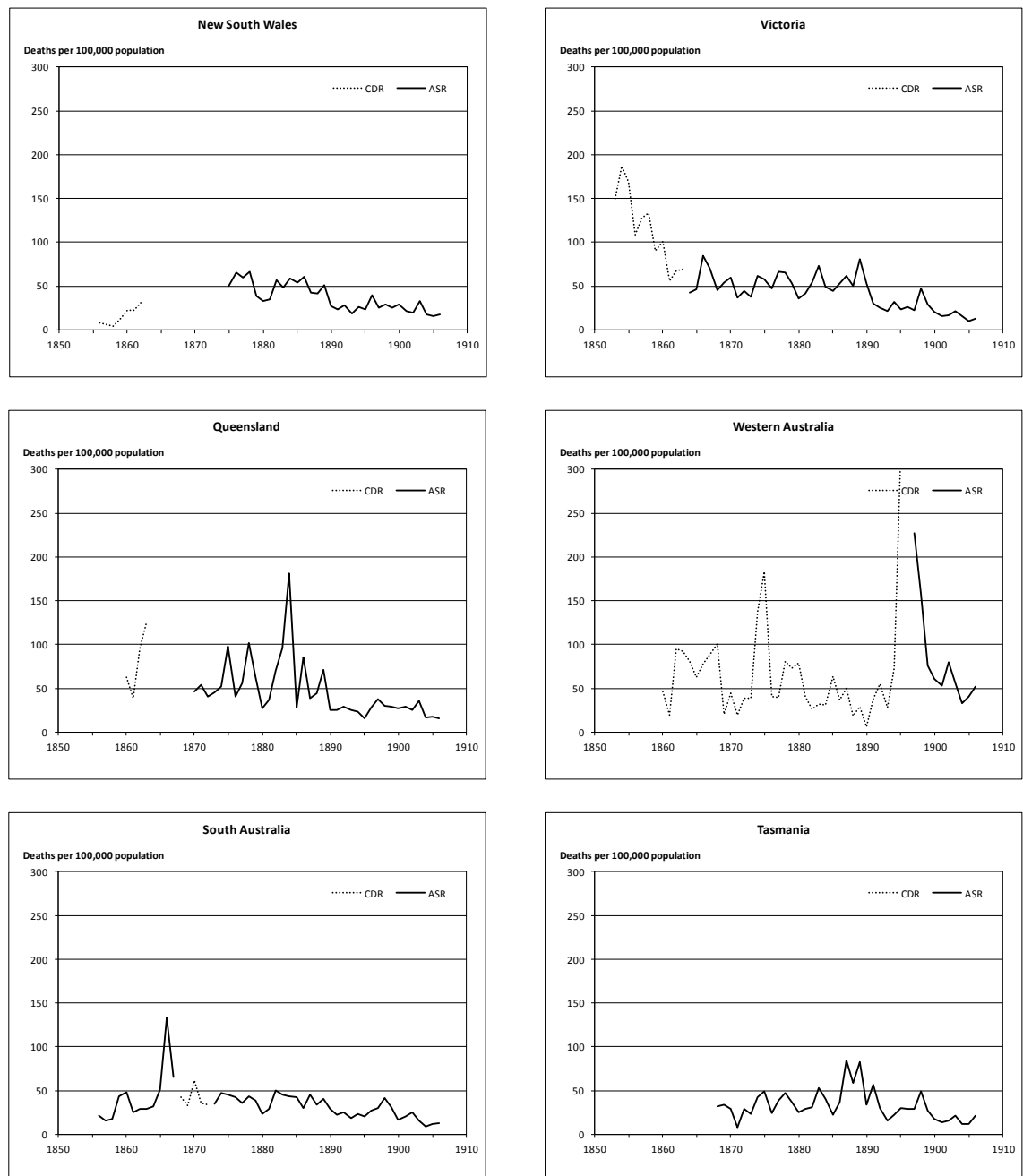
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

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Typhoid is typically transmitted through water or food contaminated with the urine or faeces of a person infected with the bacterium *Salmonella typhi*. In colonial Australia it was seasonal in nature, the incidence rising with the onset of summer and peaking in autumn.

In Western Australia, 1895 to 1898 was a period of extremely high typhoid mortality. Other periods of heightened prevalence occurred in Victoria between 1850 and 1855, Queensland 1883–1889, South Australia 1865–1867 and Tasmania between 1887 and 1889. In New South Wales, mortality fluctuated little (Figure 6.23). In both Victoria and Western Australia, typhoid fever was characterised as a 'goldfields disease', being linked to the high population influx and sanitary chaos that accompanied the discovery of gold.

Figure 6.24: Typhoid mortality rate, Australia, 1856–1906
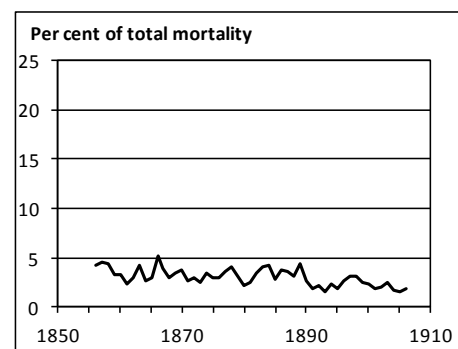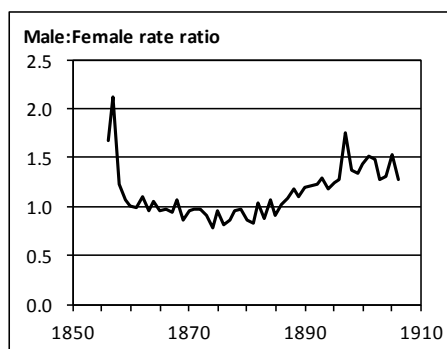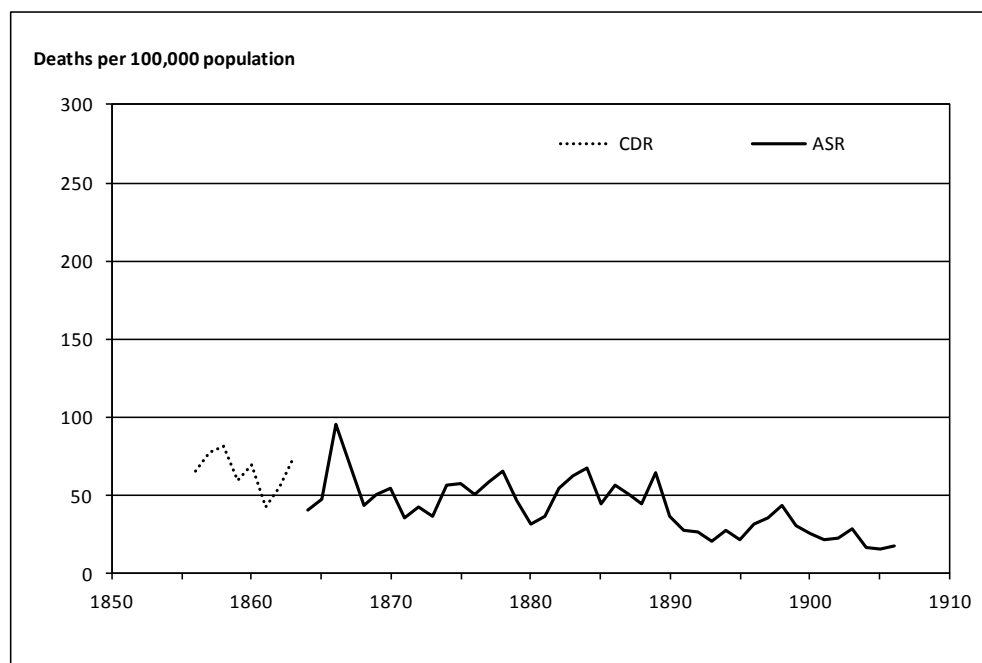
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

The year 1890 marks a division between two phases of typhoid mortality in Australia; before then, mortality was high, averaging around 50 deaths per 100,000 population, and after 1890 mortality was halved to be around 25 (Figure 6.24). The division holds for all colonies, except Western Australia. Death rates among males and females were similar, except for the years before 1860 and after 1890, which coincide with the gold rush years in Victoria and Western Australia. The disease was responsible for 3–5 per cent of total Australian mortality before 1890, falling to 1–3 per cent in the years 1890–1906.

Typhoid fever affected all age groups, but it was seen as a disease of adolescence and early adult life, and was especially fatal for the age group 15–44 years (Figure 6.25). The disease was probably under-diagnosed among the elderly (Barrett, 1883).

Figure 6.25: Age-specific mortality rates for typhoid, Australia, 1875–79 and 1900–04
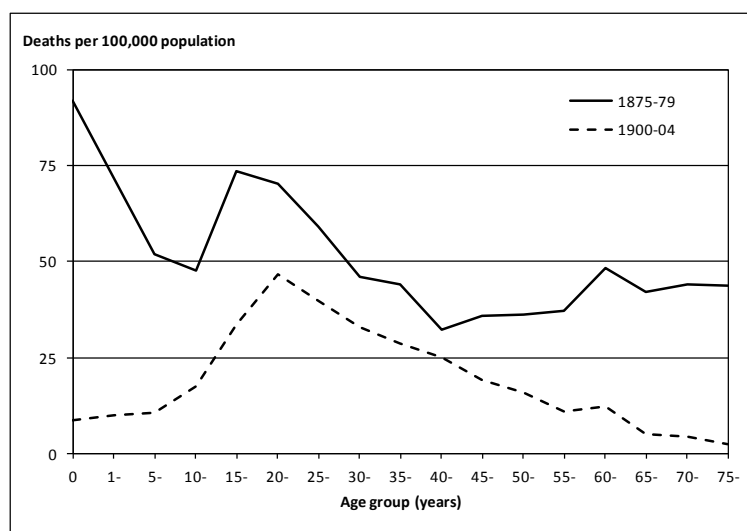
Before germ theory, typhoid was characterised as a miasmatic or 'filth' disease, with overcrowding, poor sanitation and polluted water the culprits. Kellaway (1989) found that each of these factors contributed to the change from endemic to epidemic typhoid in Hobart in the late 1880s. A sequence of hot, dry summers which affected water quantity and quality was accompanied by changes in the system for the disposal of excreta and overcrowding following a notable population increase during this time.

In Victoria, the ascerbic William Thomson was most vocal in promoting contagionism and germ theory and he did much to investigate the nature of typhoid. His 'creative observations and speculations' included noting that years of high diarrhoea prevalence in summer were often followed by bad typhoid in autumn. His 1874 book received scathing reviews from the medical establishment (*AMJ*, June 1875, pp.219–228). Although the typhoid bacterium was identified by Eberth and Gaffky in the early 1880s, it took until the end of the decade for germ theory as the cause of typhoid to be widely accepted in Australia (Robertson, 1889; Carstairs, 1889).

Typhoid became a notifiable disease in Tasmania in 1886, Victoria in 1890, New South Wales and South Australia in 1898, Queensland in 1900 and Western Australia in 1902. In New South Wales during the years 1898–1904, the annual typhoid notification rate was 2.3 per 1,000 population, but there were significant city-country disparities, with incidence in the Sydney metropolis 1.7 and in the country 4.5 (Millard, 1906).

Epidemics in country towns caused great hardship and dislocation. An 1889 outbreak in Balranald, New South Wales saw 120 cases among a population of 670, with 15 deaths. In February-March 1903, a typhoid epidemic linked to contaminated milk swept through Coonamble, with more than 200 cases and a dozen deaths in a population of 1,600 (*AMG*, 20 Mar. 1903, p.132). Board of Health President Ashburton-Thompson dispatched special teams of nurses and marquee tents to lend support to the overwhelmed local hospital. The panic saw one-fourth of the population leave the town, many by special trains organised from Sydney (*SMH*, 19 Feb. 1903, p.8; 4 Mar. 1903, p.6).

Case fatality for typhoid varied between three and 15 per cent over the nineteenth century, with an average of around 10 per cent (Cumpston, 1989). Immunisation did not become widely available in Australia until the 1920s, and outbreaks continued well into the 1940s (Smith, 2002).

CANCER

Neoplasms are abnormal tissue growth causing lumps or tumours which may be either benign or malignant (cancer). In colonial Australia, cancer was not numbered among the leading causes of death, although it assumed increasing importance over the course of the century. The nosologies of the time returned cancer deaths under a variety of headings including 'cancer', 'tumour', 'malignant disease', 'carcinoma', 'sarcoma' and 'leucaemia' (Appendix 4).
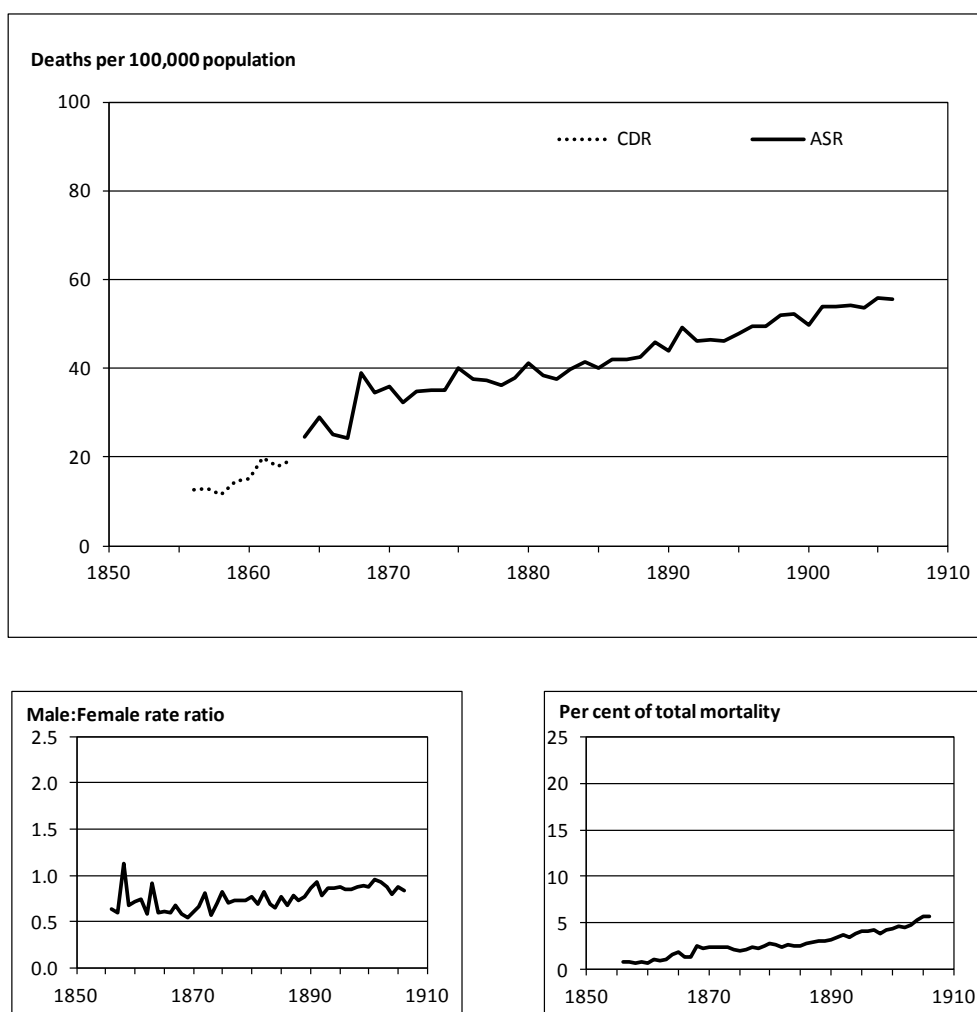
Figure 6.26: Cancer mortality rates, by colony, 1853–1906
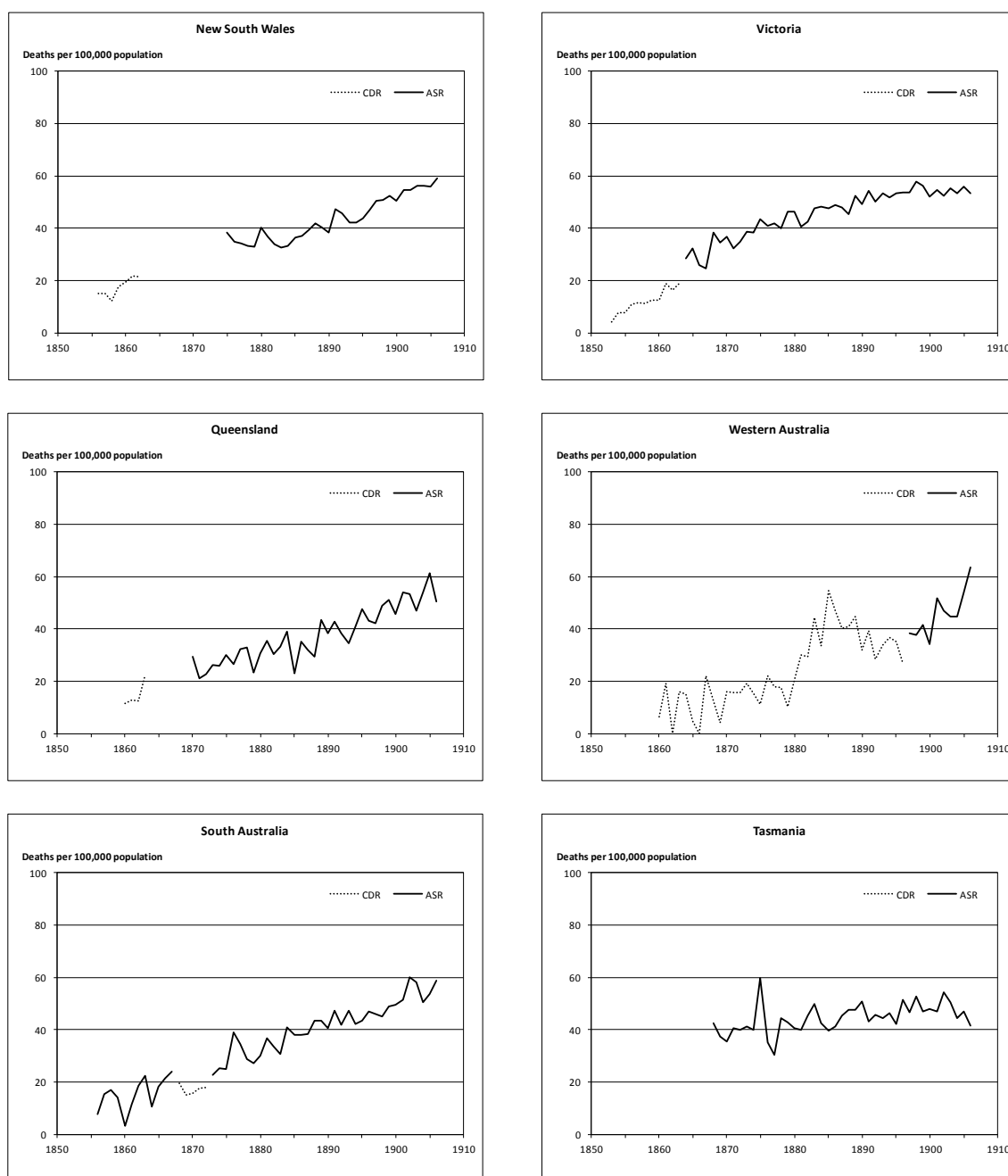
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

For most of the period, the statistical abstracts of mortality did not include tables on the part of the body affected by cancer. New South Wales was first to do so for the year 1895. The 1902 Conference of Statisticians resolved that the States routinely include special tables in their vital statistics 'showing, in respect to Cancer, the ages of persons who died from this complaint, and the portion of the body affected thereby' (Parliament of Tasmania, 1902).

Figure 6.27: Cancer mortality rate, Australia, 1856–1906

Note: Mortality rates are age-standardised to the 1881 total Australian census population.

Deaths attributed to cancer rose markedly in each colony, except in Tasmania (Figure 6.26). Across Australia, mortality rose more than four-fold, from 13 deaths per 100,000 population in 1856 to 56 in 1906 (Figure 6.27). Cancer as a proportion of total deaths increased accordingly, from around one per cent in

the 1860s to more than five per cent by 1906. Cancer deaths were more common among women, due to the contribution of breast and 'generative' cancers, although the disparity narrowed over time.

Much of the contemporary discussion on cancer statistics bore on whether there was an underlying rise in cancer incidence, or whether the increase was simply due to improvements in diagnosis and certification of death. Victorian Government Statist H. H. Hayter published a report on cancer deaths occurring between 1861 and 1884 in the *Victorian Year Book* for 1884–5. Haviland (1887) and Mullins (1896) were also early contributors in recognising the rise of 'this dreadest, and unfortunately increasing disease'.

The 1902 Intercolonial Medical Congress devoted a session to reviewing evidence on the rise of cancer mortality. New South Wales Government Statistician T. A. Coghlan presented revised statistics by body site dating from the commencement of registration (Table 6.2), and concluded that the rise was real, even allowing for an increase in the number of people at older ages and improved diagnosis. He also linked the rise to a coincident decline in tuberculosis, suggesting that the two diseases were complementary in some fashion (Coghlan, 1903; and see Knibbs, 1912, p.230–234).

Table 6.2: Cancer deaths in New South Wales, by seat of disease, 1861–1900

	1861-70	1871-80	1881-90	1891-00
Males	**511**	**1,136**	**1,874**	**3,564**
Stomach	105	256	433	937
Mouth, throat, tongue	111	193	390	717
Other digestive	28	73	206	495
Liver	22	70	200	459
Respiratory	6	25	37	122
Females	**424**	**825**	**1,522**	**2,912**
Generative	132	248	396	770
Stomach	51	117	235	517
Liver	12	48	151	363
Breast	71	105	166	359

Source: Coghlan, 1903.

Allen (1903), a pathologist, along with Verco (1903) from South Australia, argued that the apparent increase occurred among older age groups only, whereas a real rise in incidence would require corresponding increases across other age groups. This, combined with improvements in medical examination, diagnosis and registration was, for them, enough to explain the escalating mortality. Lancaster (1950a, 1958) agreed that there was no real increase in underlying incidence, adding that it continued into the 1920s, but he did allow that there may have been a real rise in certain cancers.

Figure 6.28: Age-specific mortality rates for cancer, Australia, 1875–79 and 1900–04
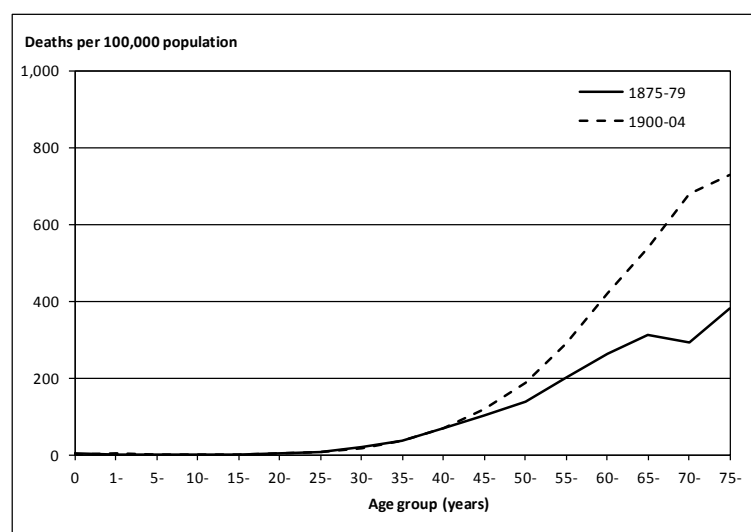
The statistics support the rise among older age groups only, say over 45 years of age (Figure 6.28), but they do not identify which other cause(s) cancer deaths were being attributed to in earlier years. Lancaster (1958) had dismissed the tuberculosis hypothesis of Coghlan and others, although it is likely that at least some lung cancer deaths were recorded as pulmonary tuberculosis, in lieu of their conspicuous absence in the statistics (Table 6.2), and the similarity of symptoms between the two diseases. Lung cancer deaths were also conceivably being recorded as cancers of the mouth, throat or tongue.

DISEASES OF THE CIRCULATORY SYSTEM

Diseases of the circulatory system are currently the leading cause of mortality among Australians, being responsible for almost one-third of all deaths in 2010 (ABS, 2012). As with cancer, circulatory system diseases seemed to have considerably less impact on colonial Australians, although they claimed a growing proportion of deaths as the nineteenth century drew to a close.

Circulatory system deaths were returned under a number of nosological headings, including 'endocarditis', 'pericarditis', 'aneurism', 'heart disease', 'angina pectoris', 'syncope', 'embolism' and 'thrombosis'. Current nomenclatures such as 'heart attack', 'infarction', 'ischaemic heart disease' and 'cerebrovascular disease' were absent.

Deaths from 'apoplexy', 'paralysis', 'congestion and haemorrhage of the brain' and 'softening of the brain'—terms which are analogous to stroke—were generally included under Diseases of the Nervous System. These, plus other selected Constitutional and Local Diseases are here included among diseases of the circulatory system (see Table 5.4).

'Valvular disease' was returned as a Disease of the Circulatory System. 'Rheumatic fever', 'rheumatism of the heart' and 'infective endocarditis' were inconsistently coded, appearing variously under the headings Zymotic Diseases, Constitutional Diseases or General Diseases (see Appendix 4). These conditions might be expected to make an important contribution to circulatory disease deaths although misclassification compromised accurate attribution.

Much circulatory disease was misdiagnosed or went undiagnosed. Its pathology was not well understood until the 1880s and effective diagnostic procedures were largely absent, with the poor being especially affected (Smith, 1979, p.325; Morley *et al.*, 2006). By 1906, New South Wales Government Statistician W. H. Hall felt that at least some of the apparent increase in circulatory disease deaths

> '…is due to a better acquaintance with the action of the heart, and to the fact that many deaths which were formerly attributed to old age are now referred to some form of heart disease' (Hall, 1906, p.683).

Diagnosis also improved, so that

> '…a more definite description by medical practitioners of these causes of death is noticeable in recent years, proportionately fewer were certified as syncope and "heart disease," and a larger number as endocarditis and valvular diseases' (Drake, 1908, p.364).

Figure 6.29: Mortality rates for diseases of circulatory system, by colony, 1853–1906
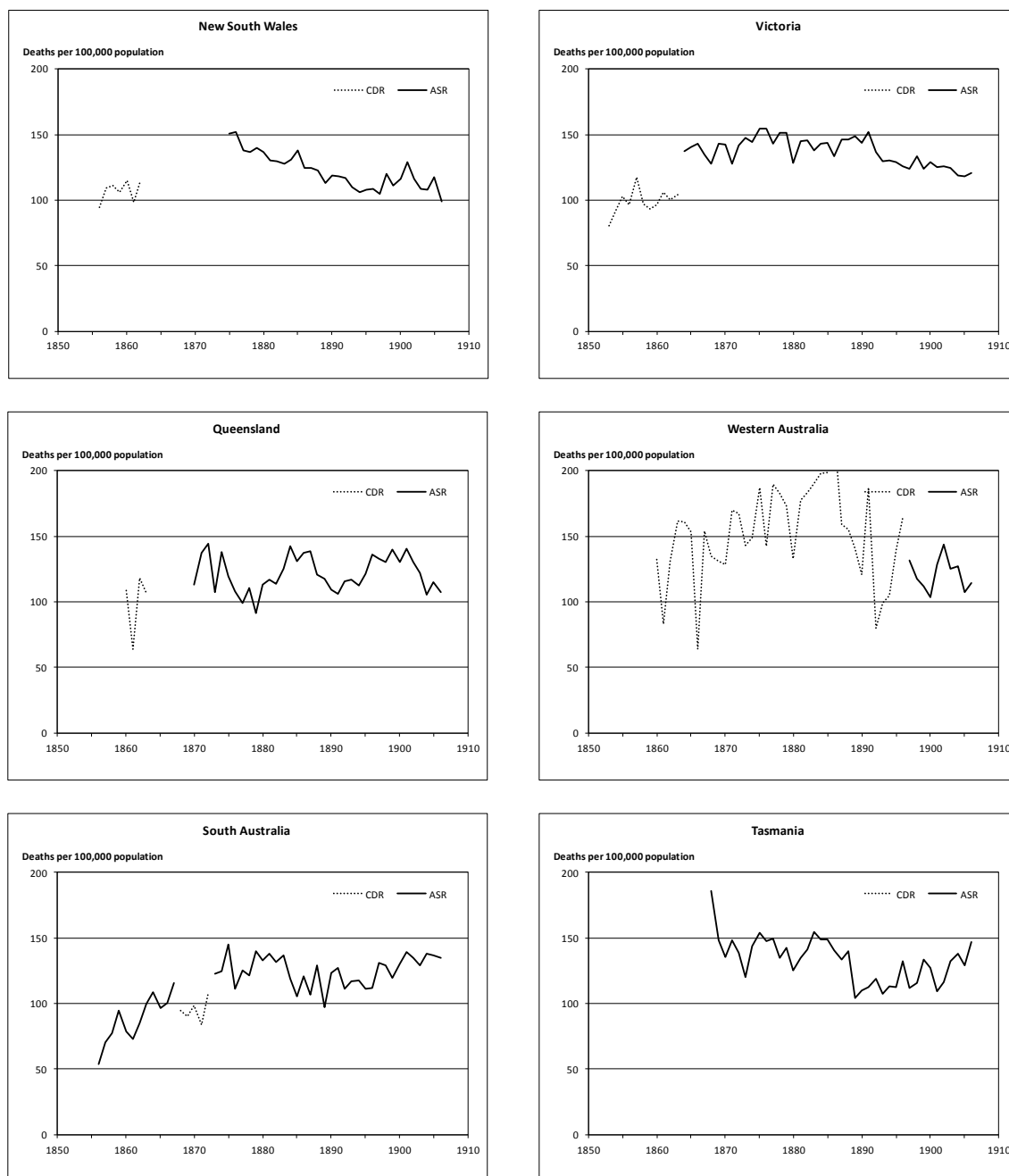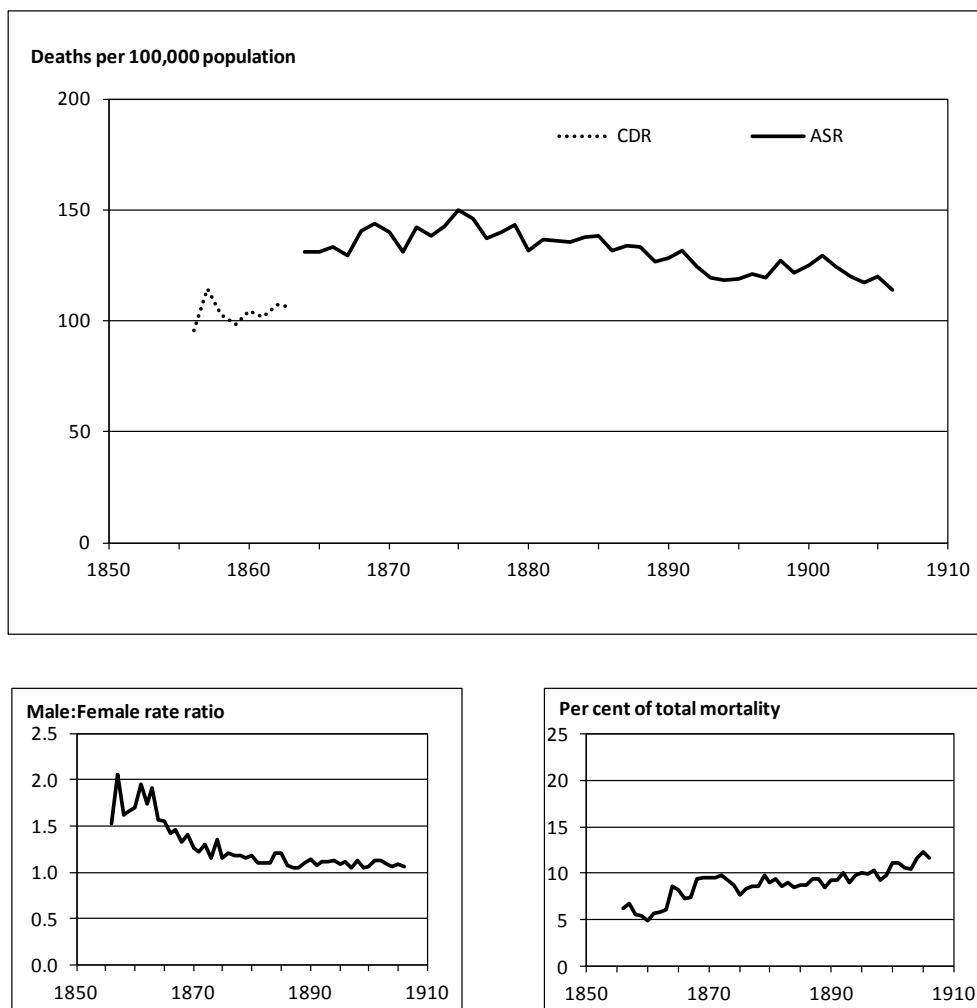

Note: Mortality rates are age-standardised to the 1881 total Australian census population.

The statistics of the colonies show differing patterns—circulatory disease mortality declined somewhat after 1870 in New South Wales, Victoria and perhaps Western Australia, whereas it showed little net change in Queensland, South Australia and Tasmania (Figure 6.29). New South Wales Statistician Coghlan thought that higher rates in Victoria and South Australia in 1901–03 existed because they 'contain the largest number of persons of middle and old age who are most prone to this order of disease' (Coghlan, 1904).

Taken as a whole, Australian mortality from diseases of the circulatory system fell from around 140 deaths per 100,000 in the mid-1870s to 120 in the mid-1900s. Rates plateaued somewhat between 1890 and 1900 (Figure 6.30).

Figure 6.30: Mortality rate for diseases of the circulatory system, Australia, 1856–1906


Note: Mortality rates are age-standardised to the 1881 total Australian census population.

Male rates were higher, especially in the years to 1870. Referring obliquely to the greater prevalence of circulatory disease risk factors among males, the New South Wales Statistician felt that the elevated rates were 'probably due to the greater risks and shocks to which males are exposed' (Hall, 1906, p.683).

Circulatory disease deaths comprised a growing proportion of total mortality from around five per cent in the mid-1860s to over ten per cent by 1906, by which time it had assumed the status of a leading cause of death.

Mortality increased rapidly after age 40 (Figure 6.31). Deaths among the elderly were increasingly returned as circulatory disease; 13 per cent of all deaths among persons aged 75 and over in 1875–79, rising to 21 per cent in 1900–04.

Figure 6.31: Age-specific mortality rates for diseases of the circulatory system, Australia, 1875–79 and 1900–04
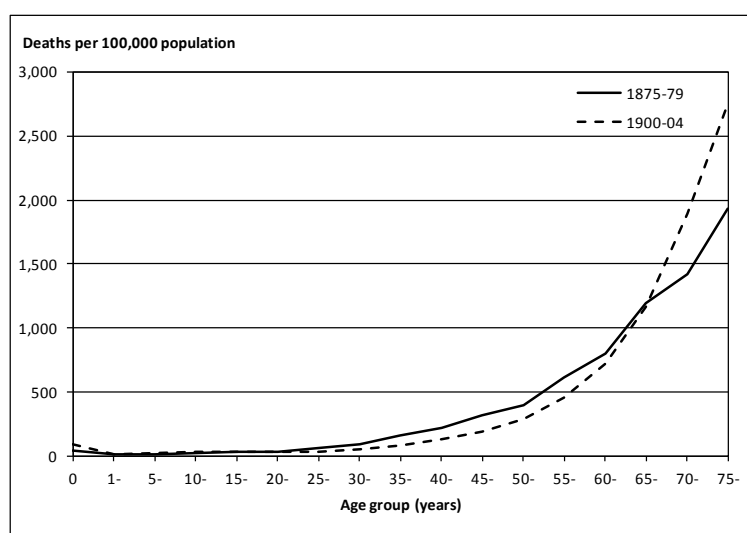
In reviewing the status of circulatory disease at the end of the nineteenth century, one commentator anticipated better days ahead;

> 'May we not hope that wiser and quieter conditions of life will yet arrest the modern tendency to heart disease...Who will venture to say that, in view of these many cases of needless dying, the moral and intellectual progress of the race will not yet achieve an enormous saving of human life? Perhaps the coming century will multiply by ten the proportion of mankind that reaches the full span of years.'
> (*Brisbane Courier*, 19 Sep. 1898, p.4)

They could not possibly foresee the impact that the disease would have during the course of the twentieth century.

DISEASES OF THE RESPIRATORY SYSTEM

The diseases of the respiratory system have here been further divided to focus on two major groupings, 'Influenza and pneumonia', and the chronic lower respiratory diseases 'Bronchitis, emphysema and asthma'.

Again, misdiagnosis of these diseases was not unknown among doctors certifying deaths, affecting the accuracy of mortality returns. In earlier years there was confusion as to the relationship between croup and diphtheria. Influenza was confused with bronchitis, pneumonia, or other diseases of the respiratory system. Respiratory tuberculosis was confused with other respiratory diseases, and bronchial pneumonia with bronchitis (Table 5.3).

Death rates may also be overstated. Bronchitis or pneumonia were often complications of other underlying conditions, especially among the elderly, but were commonly recorded as the cause of death.

Influenza was first recorded in the colonies early in the nineteenth century, with regular outbreaks occurring between 1820 and the early 1850s (Carstairs, 1894). Springthorpe (1890) makes special mention of the effects of the 1847–48 pandemic, which reached Australia. Influenza returned each autumn and winter, but in the second half of the nineteenth century three occurrences were especially noteworthy (Figure 6.32). A general epidemic occurred throughout Australia in 1860, although it was not well documented (Cumpston, 1989, p.313–314). E. S. Hall recorded its effects in Hobart, and estimated that 23,000 persons out of 25,000 in the registration district were attacked, with the elderly being terribly affected (Hall, 1860; *Hobart Mercury*, 17 Jul. 1860, p.3).

The 1885–86 epidemic was known as 'fog fever' because it occurred during an unusually wet and foggy winter (Springthorpe, 1885). Originating in Melbourne, it quickly spread to the other colonies; 'the cases were very numerous, some very severe in nature—whole families being affected'. Adelaide doctor Melville Jay reported presciently, 'From its highly infectious nature, I think there can be little doubt that the disease is imported by some contagious virus, and the means by which it spreads from one another, is the breath' (Jay, 1886).

Figure 6.32: Mortality rates for diseases of respiratory system, by colony, 1853–1906
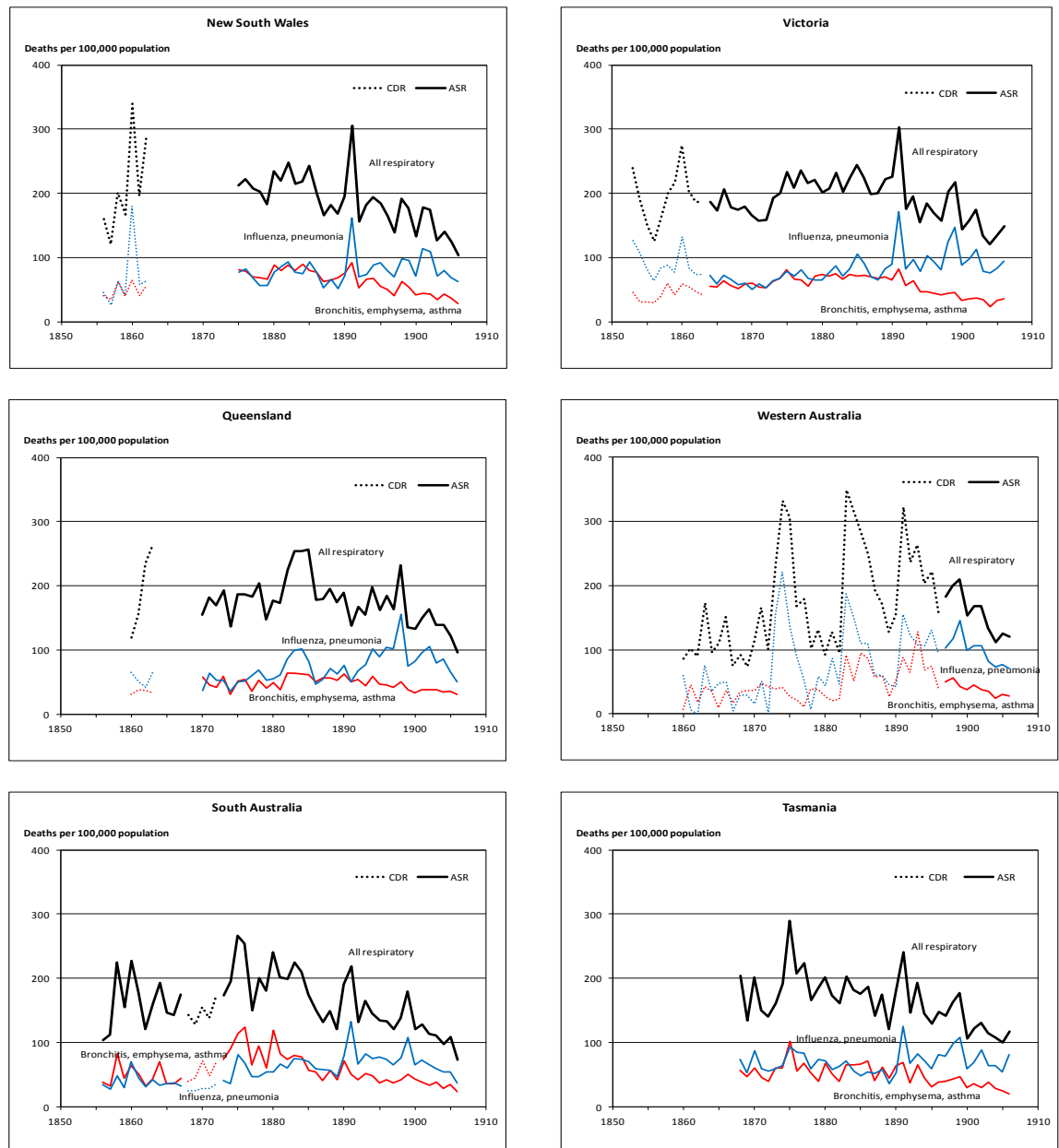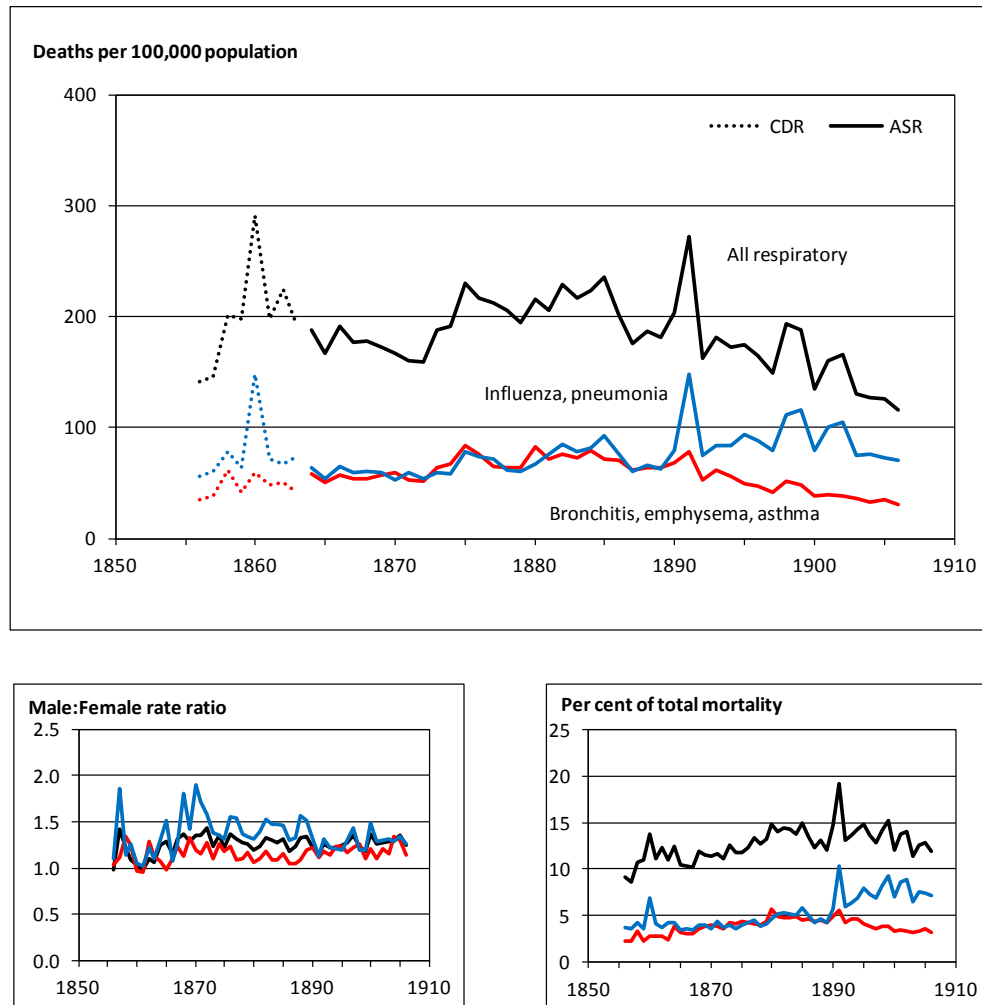

Note: Mortality rates are age-standardised to the 1881 total Australian census population.

The 1890–91 epidemic gained most attention in Australia, since it formed part of the great 'Russian influenza' pandemic which was believed to have affected up to 40 per cent of the population worldwide (Cumpston, 1989, p.320). Reaching Hobart in March 1890, by the end of May it had spread to Perth (Carstairs, 1892), ultimately causing almost 2,500 deaths across all colonies. Mortality was most severe in New South Wales and Victoria, with Queensland largely spared (Figure 6.32). As in England, the epidemic had considerable social and cultural

impact, largely through its reporting in mass-circulation newspapers (Honigsbaum, 2010, and see, for example, *Argus*, 26 Mar. 1890, p.6).

Influenza would achieve even greater notoriety early in the new century. The 1918–19 'Spanish influenza' pandemic claimed over 12,000 victims in Australia, and was to be the single most devastating disease event in Australian history.

Figure 6.33: Mortality rate for diseases of the respiratory system, Australia, 1856–1906


Note: Mortality rates are age-standardised to the 1881 total Australian census population.

Pneumonia was the most fatal of the respiratory diseases and like influenza was highly seasonal. By the early twentieth century, pneumonia had become a leading cause of death (Hall, 1906, p.677) although it often appeared as a complication of other underlying diseases. In years of high influenza mortality pneumonia deaths also rose. The Queensland Registrar-General, W. T.

Blakeney, made special note of deaths from pneumonia among Polynesian labourers, 'these people being particularly susceptible to diseases of that nature' (QldRG, 1886).

The mortality rate for bronchitis, emphysema and asthma was closely related to that of influenza and pneumonia until 1891, the year of the Russian flu epidemic (Figure 6.33). Thereafter, bronchitis, emphysema and asthma mortality declined by more than half, from 79 deaths per 100,000 population in 1891 to 31 in 1906.

Figure 6.34: Age-specific mortality rates for diseases of the respiratory system, Australia, 1875–79 and 1900–04
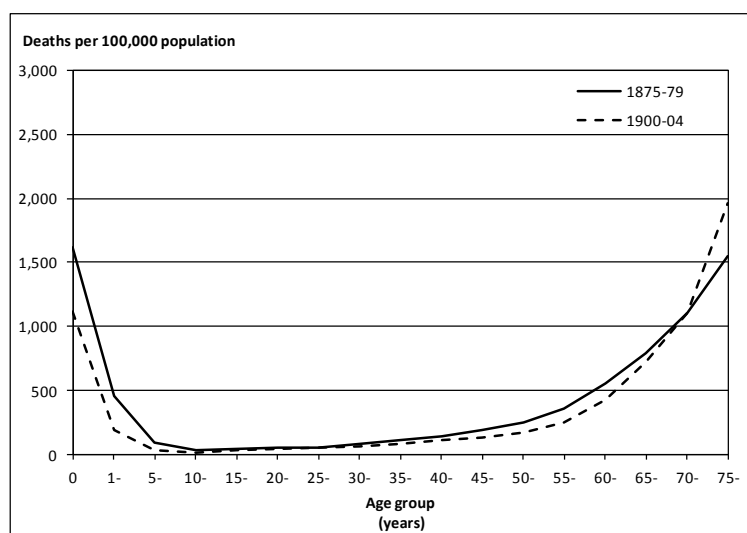

Influenza epidemics notwithstanding, respiratory disease mortality began to decline in Australia from the mid-1880s. Fatality was higher among males, and for influenza and pneumonia the ratio was especially unequal between 1870 and 1890, at three male deaths for every two female deaths. Despite the decline in rates, the diseases comprised a growing proportion of total mortality, from around 10 per cent in 1870 to 15 per cent in 1900.

Respiratory diseases affected all ages, but mortality was highest among the young (aged 0–4 years) and the old, with rates increasing rapidly after the age of 50 years (Figure 6.34).

DISEASES OF THE DIGESTIVE SYSTEM

A number of causes of death are included in this broad grouping, including hernia, peritonitis, cirrhosis and other liver diseases, ileus or obstruction of the intestine, ulceration of the intestine, and stomach diseases. The nosologies commonly included enteritis and dentition as diseases of the digestive system, but in this work these have been reclassified as gastrointestinal infections.
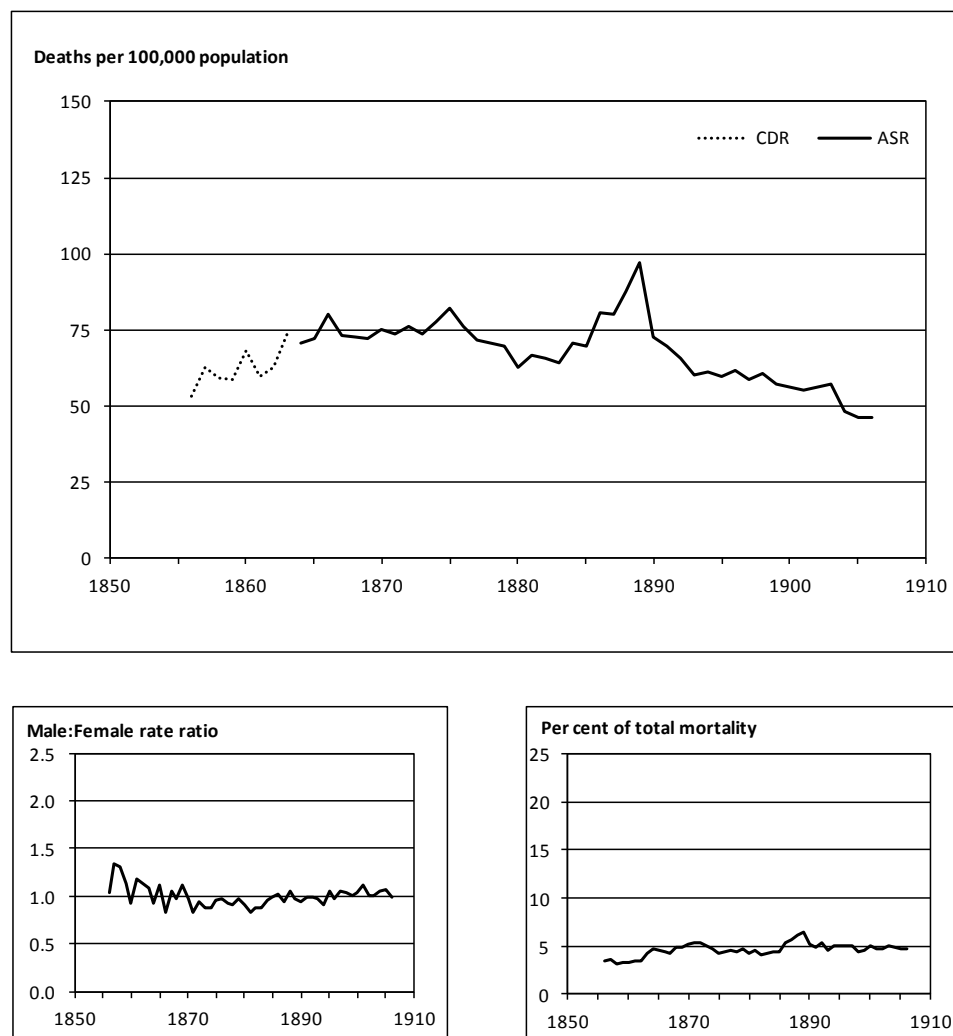
Figure 6.35: Mortality rates for diseases of the digestive system, by colony, 1853–1906
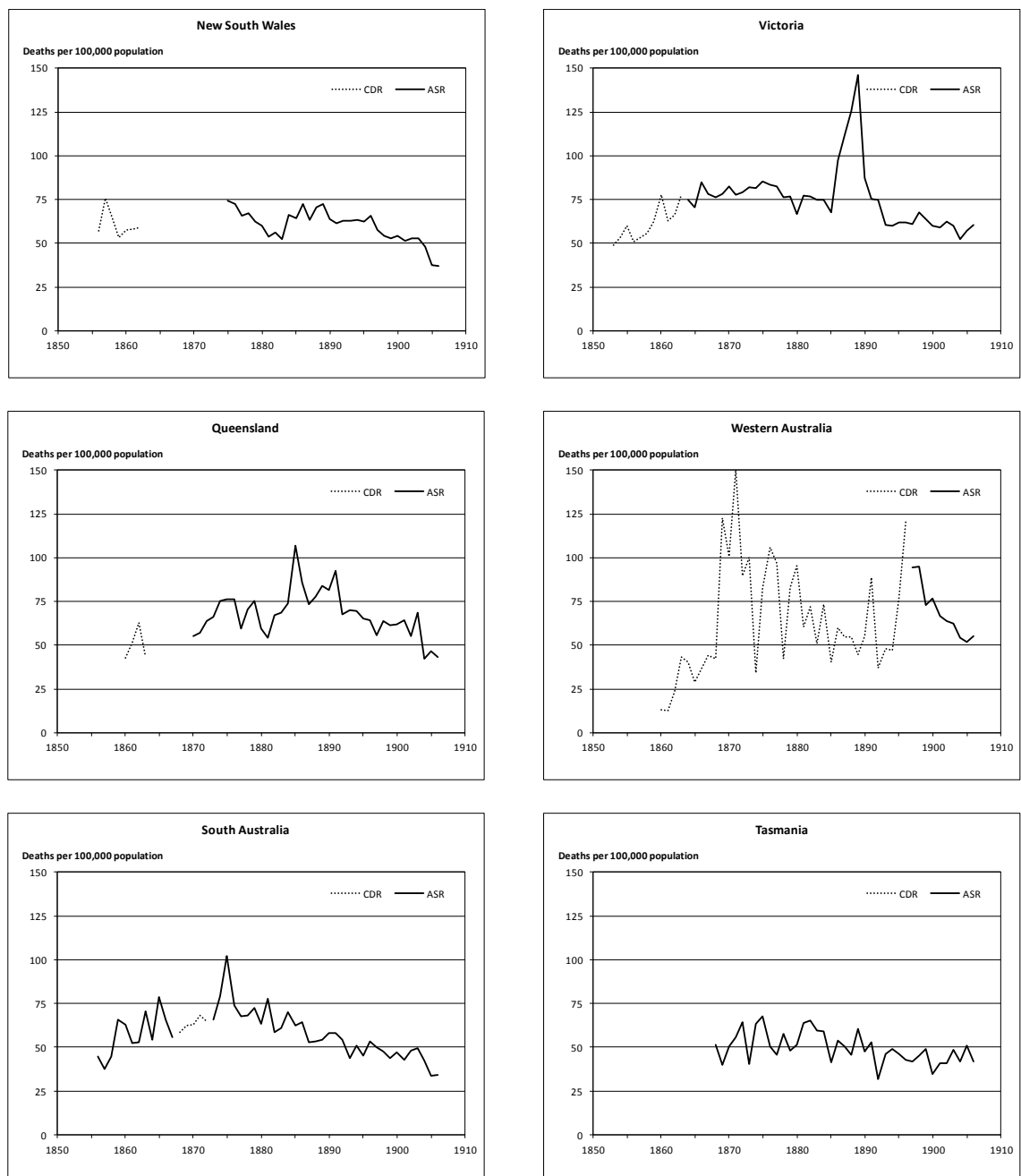
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

The sudden rise in mortality from diseases of the digestive system in Victoria during 1887–1889, which also affects the Australian rate, remains unexplained (Figures 6.33 and 6.34). Almost all excess deaths occurred among infants and were attributed to diseases of the stomach. Sudden peaks in diseases of the digestive system also appeared in South Australia in 1875 and in Queensland in 1885 and 1891.

Figure 6.36: Mortality rate for diseases of the digestive system, Australia, 1856–1906

*Note:* Mortality rates are age-standardised to the 1881 total Australian census population.

Although Government Statistician Hayter had recently introduced a new nosological system which was first used for the Victorian statistics of 1886, no rise was apparent in that year. It would appear that the explanation for the rise does not lie in unfamiliarity with a new classification.

In commenting on mortality in Melbourne and suburbs for the year 1889, Hayter observed that 286 more deaths from diseases of the digestive system had occurred than in the previous year, and that it had been a year of exceptionally high mortality all round, especially from diphtheria and typhoid fever (*VicGG*, 3 Apr. 1890; *Argus*, 24 Apr. 1890, p.9). Deaths from atrophy and debility had also been high. There was perhaps some misclassification of deaths among infants from these causes to diseases of the stomach.

Across Australia, mortality from diseases of the digestive system stood at 75 deaths per 100,000 population in 1875 (Figure 6.36). A steady decline took place over the next thirty years, interrupted only by the unexplained peak in Victoria in the late 1880s. Mortality fell below 50 by 1905.

Death rates from diseases of the digestive system were similar among males and females. The diseases were responsible for about 5 per cent of total mortality.

Figure 6.37: Age-specific mortality rates for diseases of the digestive system, Australia, 1875–79 and 1900–04
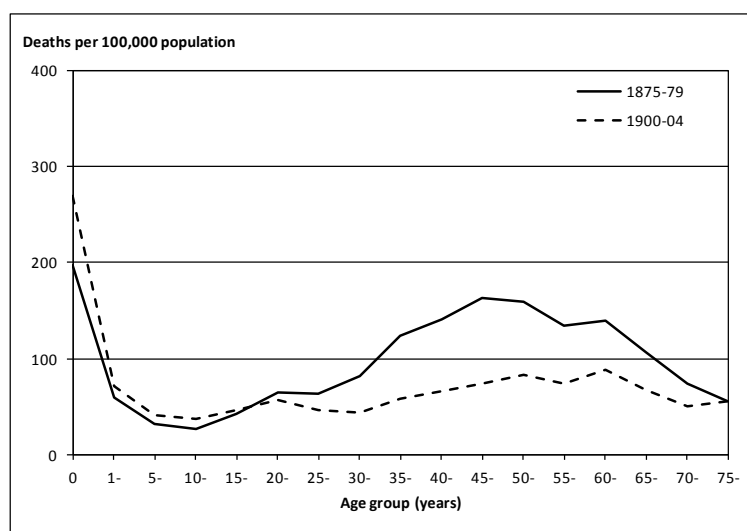
Deaths rates were highest among infants (Figure 6.37). From early childhood to old age rates were consistent, although in earlier years middle-aged persons were at greater risk.

MATERNAL MORTALITY

Pregnancy and the puerperal period—childbirth and the weeks following—presented much pain, suffering and danger to nineteenth-century Australian women. Death claimed many through eclampsia and hypertensive disorders of pregnancy, haemorrhage, embolism, complications of unsafe abortion, and that great fear of pre-antibiotic times, puerperal sepsis. In the 1890s and early 1900s, around 600 deaths a year Australia-wide were attributed to maternal causes.

Quantifying the incidence of maternal mortality, however, presents problems. The nosological indexes provided neither a definition for, nor a grouping under which to count maternal deaths. Deaths resulting from complications of pregnancy and childbirth were returned under a number of causes (Table 6.3), and these were classified variously as Miasmatic diseases, Diseases of the organs of generation, Developmental diseases of adults, Septic diseases, Diseases of parturition or Puerperal conditions.

Table 6.3: Causes of maternal deaths in nineteenth-century Australian nosologies

Archer, 1853	
18 Puerperal Fever	104 Childbirth, Abortion

Archer, 1863	
I-1-9-Metria	IV-2-2-Childbirth

Hayter, 1886	
I-6-4-Puerperal Fever	VI-9-4-Placenta Praevia, Flooding
VI-9-1-Abortion, Miscarriage	VI-9-5-Phlegmasia Dolens
VI-9-2-Puerperal Mania	VI-9-6-Other Accidents of Childbirth
VI-9-3-Puerperal Convulsions	

Royal College of Physicians, 1896	
I-5-1-Puerperal Septicaemia, Saproemia	II-10-1-Abortion, Miscarriage
I-5-2-Puerperal Pyaemia	II-10-2-Puerperal Mania
I-5-3-Puerperal Phlegmasia Dolens	II-10-3-Puerperal Convulsions
I-5-4-Puerperal Fever	II-10-4-Placenta Praevia, Flooding
	II-10-5-Pregnancy, Childbirth, Other Accidents

Bertillon, 1906	
134 Accidents of Pregnancy	138 Puerperal Albuminuria and Eclampsia
134a Illegal Operations	139 Puerperal Phlegmasia alba dolens
135 Puerperal Haemorrhage	140 Other Puerperal Accidents, Sudden Death
136 Other Accidents of Childbirth	141 Puerperal Diseases of the Breast
137 Puerperal Septicaemia	

Misclassification, whether unintentional or deliberate, was significant. Deaths which should have been classified as metria or as puerperal fever were often

returned as convulsions, haemorrhage, rupture of the uterus or some other cause (Jamieson, 1882b). A proportion of maternal deaths were wrongly attributed to associated symptoms or non-maternal conditions such as debility, exhaustion, peritonitis, pyaemia, septicaemia, uterus disease, cardiovascular disease, or other fevers such as typhoid (Hayter, 1889, p.143). Doctors, midwives and those managing labour were reluctant to admit that the deaths of women under their care were due to maternal causes (Jamieson, 1882b, 1887). Deaths from induced abortions were also known to be registered to other causes, such as miscarriage (Smith, 2011, pp.68–73, 190–196).

Figure 6.38: Maternal mortality ratio, by colony, 1853–1906
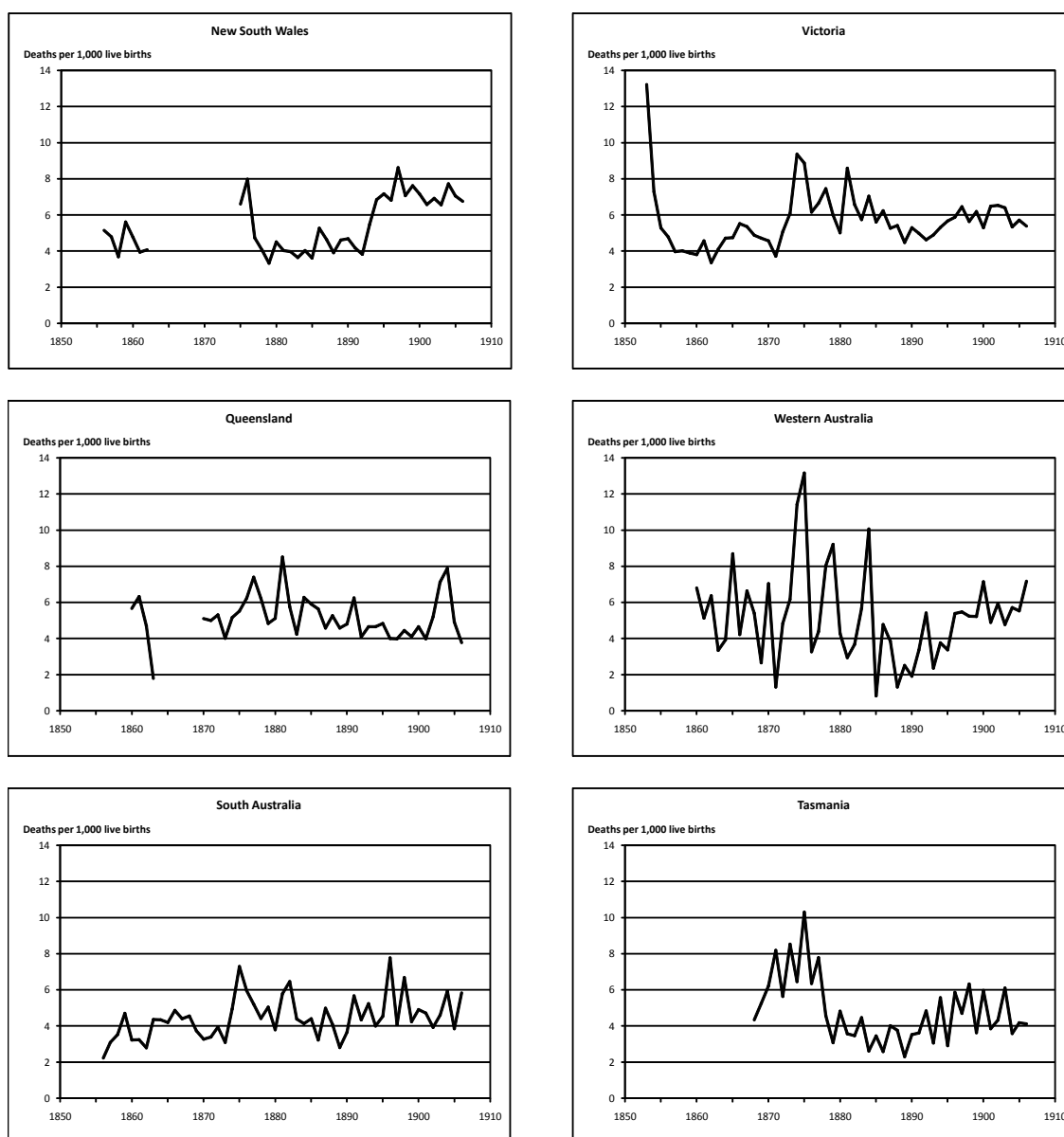
The official statistics under-represented maternal mortality and for much of the period perhaps grossly so (Smith, 1979, p.13ff.). Kippen (2005) a number of different methods to count maternal deaths in 1880s Tasmania, finding that the *Statistics of Tasmania* may only have reported half of the actual number.

Nonetheless, the official statistics have here been used to calculate the maternal mortality ratio (MMR), this being the number of maternal deaths per 1,000 live births. It should also be noted that the determination of annual numbers of live births presents its own set of problems, since these were subject to under-registration, and even more so than deaths. Registering births and deaths was a legal obligation, but whereas burial could not proceed until a death had been registered, there was no equivalent sanction for birth. For whatever reason, many births were simply not registered. 'Concealment of birth', or misreporting of infant deaths as stillbirths by midwives and other birth attendants also occurred.

The MMR varied markedly across the colonies (Figure 6.38). It exceeded 10 deaths per 1,000 live births in Victoria in the early 1850s, in Western Australia in 1874–75 and 1884 and in Tasmania in 1875. Rates in mid-1870s Victoria remained high with Hayter's justification being that his returns were more exact than those of other colonies (Jamieson, 1882b). Rises in 1866–67, 1874–75, 1881–82 and 1884 were concurrent with epidemics of measles and scarletina (Hayter, 1889) with one explanation linking these rises to changes in the prevalence of *streptococcus* in the population (Loudon, 1987).

The 'great outbreak' of 1874–75 occurred simultaneously in Australia, England and other countries. Farr felt that this 'deep, dark and continuous stream of mortality' was attributable to a want of skill on the part of midwives and physicians (Hayter, 1880, p.141). Jamieson (1882b, 1884) commented on the high prevalence of puerperal fever and other infectious diseases during these years, and proposed that puerperal fever should be closely linked if not analogous to puerperal pyaemia or septicaemia, erysipelas, pyaemia and septicaemia.

Most births occurred in the home and were attended by midwives, or increasingly by medical practitioners. Although chloroform and antisepsis were

to revolutionise obstetrics, these were confined to hospitals and had little effect on home deliveries (Woolcock *et al.*, 1997; Loudon, 1997). Mortality was high in lying-in hospitals — charitable maternity hospitals largely utilised by the poor — although the introduction of antiseptic midwifery in 1887 led to a fall in rates at the Melbourne Lying-In Hospital (Anderson, 1888; Balls-Headley, 1888; Hayter, 1889; McCalman & Morley, 2003).

Figure 6.39: Maternal mortality ratio, Australia, 1856–1906
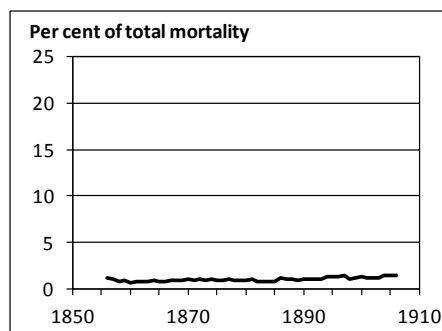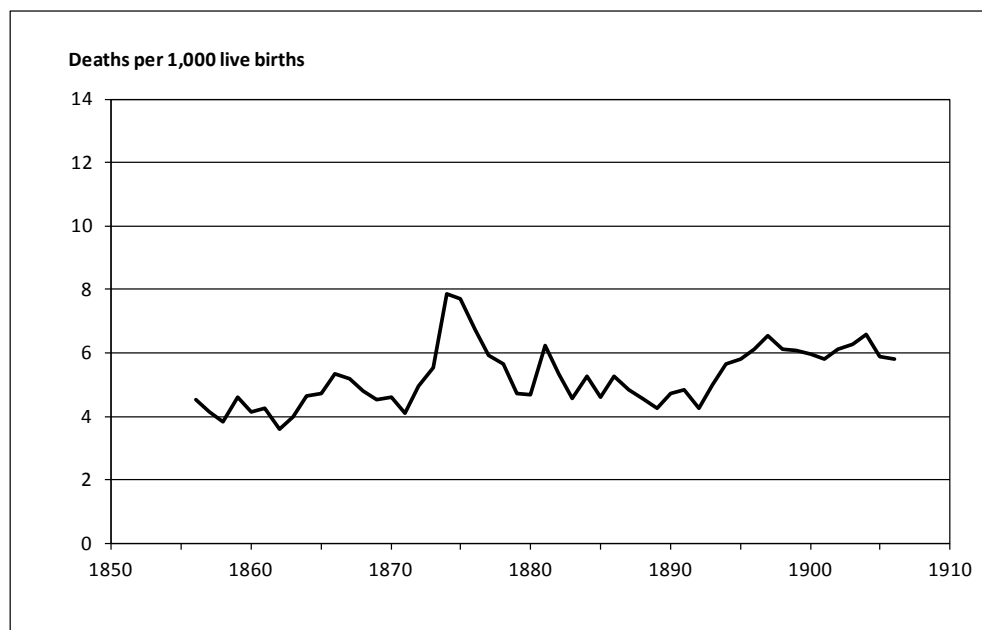

Unlike so many other causes of death, maternal mortality rates refused to fall in the latter decades of the nineteenth century. A number of colonies (New South Wales, Victoria, Western Australia, Tasmania) saw rises in MMR in the 1890s, although these can be partly ascribed to improvements in registration of causes of death. Coghlan (1900) believed parochially that 'the New South Wales

returns since 1892 have been compiled with great care, and are perhaps nearest to the truth, while the Victorian returns are also fairly reliable'.

Deaths from maternal causes comprised around one per cent of total mortality in nineteenth-century Australia (Figure 6.39). Risks were greater for first confinements, and for unmarried women for whom many confinements took place in lying-in hospitals (Jamieson, 1884; Coghlan, 1898). Women aged in their thirties were most at risk of maternal death (Figure 6.40). In 1875–79, among women aged 35–39 years, 159 in every 100,000 died from maternal causes. By 1900–04, the rate had fallen somewhat to 111.

Figure 6.40: Age-specific maternal mortality rate, Australia, 1875–79 and 1900–04
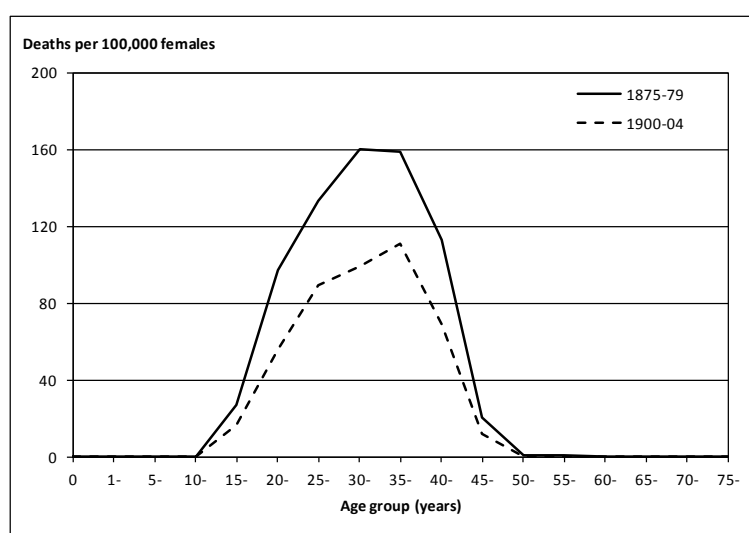
In 1906, MMR in Australia stood at 5.80 deaths per 1,000 live births. It remained steady for the next three decades, not falling below five. This trend in maternal mortality was replicated in almost all western countries with little change in rates before the mid-1930s (Loudon, 1991).

Although a new image of responsible motherhood in Australia began to emerge around the turn of the century, fears about low levels of population growth supported efforts to increase the welfare of infants, and concern for mothers lagged (Lewis, 1980). Significant improvements were not seen before changes in maternal policies and systems of care and the advent of antibacterial sulphonamide drugs in the 1930s (De Costa, 2002; Loudon, 1992).

EXTERNAL CAUSES

Deaths classified to external causes can also be referred to as deaths due to violence (Lancaster, 1964). Following the nomenclature first adopted for Archer's 1863 nosology, these deaths can be further grouped into three major categories: (i) accidents, including injury, drowning, suffocation and accidental poisoning, (ii) suicide, and (iii) homicide and judicial executions.

From the earliest days, external causes were a leading cause of death. In his first report for 1841–42, G. F. Stone, the Registrar-General in Western Australia commented that 'out of the twenty-nine registered deaths during this past year, ten died from accidental causes' (*PG*, 15 Oct. 1842, p.3). There were hazards in pioneering new settlements, and in living and working in dangerous rural, urban and unregulated industrial settings. During the entire period 1856–1906, around eight per cent (or one in 13) deaths in Australia were due to external causes.

The means of death were many and varied—in 1880s Queensland for example, the range of accidents included falls from horses, kicks by cows, falling trees, blasts and falls of earth, run over by drays, trains or other vehicles, falling down mine shafts, ship's hatchways or from other heights, and killed by alligators (*sic*) (QldRG, 1882). The most common methods to end one's life were by hanging, gunshot wound (males), drowning, cutting or stabbing. Females usually took poison.

Legislation required that unexplained, non-natural or accidental deaths were to be subject to an inquest with the presiding coroner or magistrate to inform the registrar of his or a jury's finding (Archer, 1854). On occasion, the cause of death was unknown or imperfectly known. The intent of suicide could not always be determined and in all likelihood these, along with cases of deliberate concealment, led the official suicide statistics to be underestimates.

The distribution of external causes in Table 6.4 refer to the early 1880s abstracts of mortality, and show the predominance of fractures and contusions, i.e. injury, especially among men. One-in-five deaths from external causes were through drowning, this large number including deaths from shipping and

maritime disasters along with boating accidents and drowning in lakes, rivers or seas. Suicide was more common among males with the highest rates in South Australia and Victoria. One quarter of all female deaths from external causes were caused by burns and scalds, these occurring most commonly among the very young and old and often resulting from domestic accidents involving cooking fires, kettles of boiling water or kitchen coppers (Smith, 2011, p. 198). Suffocation was most prevalent among infants and was usually caused by being overlain in bed. Females were more subject to domestic violence, and had higher rates of death due to homicide.

Table 6.4: Deaths from external causes, Australian colonies, 1880–1884 (per cent)

	NSW	Vic	Qld	WA	SA	Tas	Aus
Males							
Fractures, contusions, etc.	43.7	44.1	38.0	24.9	42.5	27.0	41.7
Drowning	22.9	22.4	29.3	28.0	21.7	27.0	24.0
Suicide	8.0	12.6	8.9	9.3	13.0	6.3	10.1
Burns and scalds	5.4	5.4	3.5	2.1	4.6	11.2	5.2
Suffocation	3.2	5.5	1.6	2.6	5.6	6.3	4.0
Sunstroke, lightning	3.1	1.5	7.4	2.6	0.0	0.9	2.9
Poison	3.3	1.6	3.1	1.6	2.3	2.1	2.6
Homicide	1.9	1.6	4.7	7.8	2.0	2.1	2.4
Other violence	8.4	5.2	3.5	21.2	8.4	17.2	7.2
Total	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**
Females							
Fractures, contusions, etc.	22.6	17.8	22.3	13.2	19.7	21.2	20.4
Drowning	18.3	20.7	22.0	15.8	18.1	25.0	20.1
Suicide	5.5	8.3	5.4	5.3	11.3	5.2	6.9
Burns and scalds	27.3	25.7	22.0	21.1	31.9	13.7	25.3
Suffocation	5.9	11.5	6.0	0.0	8.0	6.6	8.0
Sunstroke, lightning	4.4	3.0	10.5	10.5	0.0	1.9	4.2
Poison	6.4	3.9	3.6	10.5	3.8	1.9	4.7
Homicide	4.4	5.1	4.2	10.5	2.9	2.8	4.4
Other violence	5.3	3.9	3.9	13.2	4.2	21.7	6.0
Total	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**

Rates of death from external causes declined in the colonies between 1856 and 1906, although the changes in South Australia and Western Australia were less pronounced (Figure 6.41). Queensland in the early 1860s was frontier country, and deaths from external causes were high at over 300 per 100,000 population.

South Australia and Tasmania were comparatively safe colonies to live in. By early in the new century, rates in all colonies had fallen to 100 deaths per 100,000 population, or below.

Figure 6.41: Mortality rates for external causes, by colony, 1853–1906
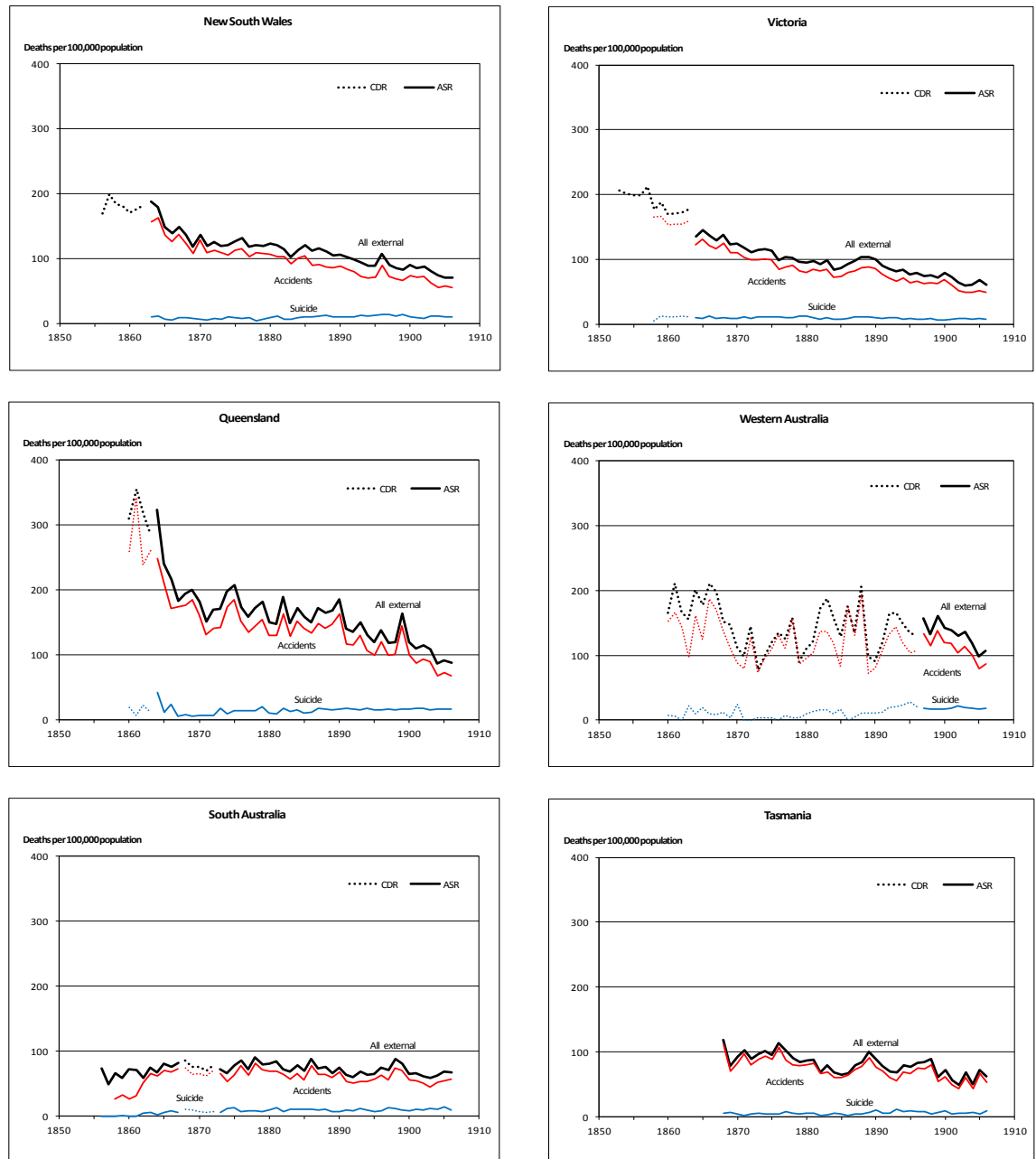
Deaths attributed to suicide remained reasonably constant, not exceeding 20 per 100,000 population, except during Queensland's earliest years. In New South Wales, male rates reached a peak in the mid-1890s (21 per 100,000 population),

coinciding with a period of recession and drought, with deaths among men aged 60 years or over most common (Morrell, Page & Taylor, 2002). But whereas suicide rates among the elderly have since declined, those among young people have fluctuated, and they were to increase greatly over the second half of the twentieth century.

Figure 6.42: Mortality rate for external causes, Australia, 1856–1906
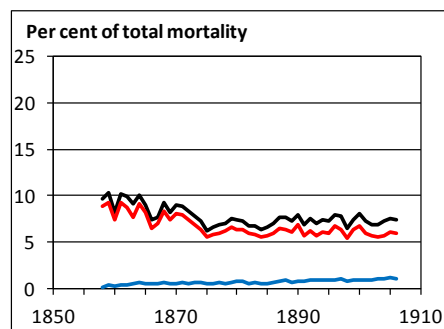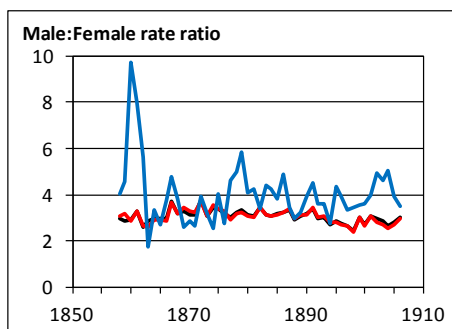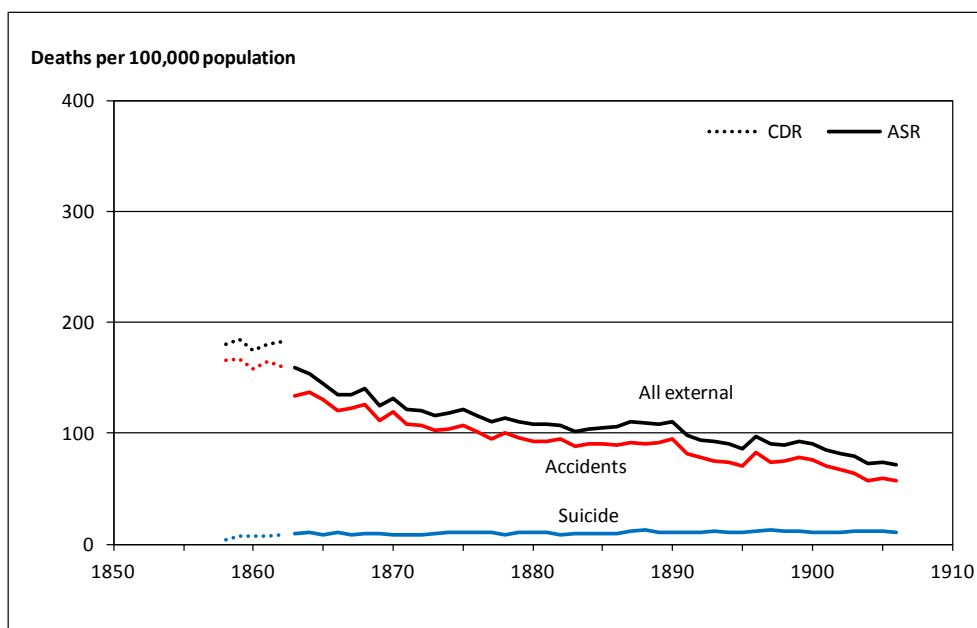
*Note:* Mortality rates are age-standardised to the 1881 total Australian census population.

Mortality from external causes fell in Australia by 40% between 1865–69 and 1900–04 (Figure 6.42). All the major causes except suicide declined, and so most of the improvement was due to lower death rates from fractures and contusions and from drowning. Registrars often commented on reductions in deaths from

mining and rural accidents (e.g. Hayter, 1880, p.136) as well as from drowning (Drake, 1908, p.368).

Deaths from accidents were more prevalent among males, especially between the ages of 15 and 54 (Lancaster, 1964). For all external causes, the ratio was three male deaths to every female death, and for suicide, slightly more at four-to-one. External causes accounted for ten per cent of total mortality in 1860, but fell to around seven per cent from 1875. Suicide averaged less than one per cent of total mortality, although the proportion increased slightly from 1860–69 (0.5%) to 1900–1906 (1.0%).

Figure 6.43: Age-specific mortality rates for external causes, Australia, 1875–79 and 1900–04
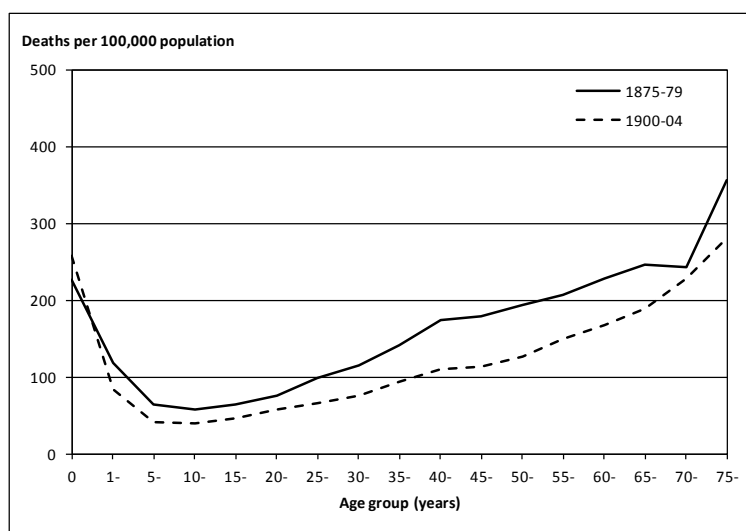
After the hazards of infancy, mortality from external causes rose steadily with age (Figure 6.43). The nature of the accidents and violence involved changed with age (Lancaster, 1964). Homicide, along with burns, scalds and suffocation were most dangerous for the youngest (aged 0–4) and oldest (aged 75 and over). Suicide rates and rates of death from fractures and contusions increased steadily from age 15. Hip fracture contributed to old age mortality, especially among females, although it did not always appear as a principal cause of death. Rates of drowning were similar among all age groups, except for the oldest aged 75 and over, who had a higher rate.

Fig. 6.44: In the casualty ward of the hospital, visiting day (*Illustrated Australian News*, 15 Sep. 1888)

SUMMARY

Eighteen common causes of death in Australia were profiled for the period 1856 to 1906, using information summarised in the abstracts of mortality. These causes include diphtheria, whooping cough, measles, scarlet fever, tuberculosis, gastrointestinal infections, typhoid fever, cancer, circulatory diseases, respiratory diseases, digestive diseases, maternal mortality and external causes of death.

Time series of age-standardised death rates illustrate trends in mortality from these diseases in each colony and in Australia. The ratio of male-to-female death rates, along with the contribution of each disease to all-cause mortality further illustrates the burden which these diseases imposed. Age patterns and changes in age patterns over time describe how this burden varied among infants, young people, the middle-aged and elderly.

Although each cause of death is examined singly, these analyses allow for the quantification of the important phase of the mortality transition that began in Australia in the mid-1880s. The following chapter will determine the relative contribution of each cause of death to the decline in mortality. The declines in rates of infectious diseases, and the pivotal roles of infant gastrointestinal infection and of adult tuberculosis in the transition are already apparent.

Chapter 7

Mortality in transition

The mortality regime in Australia underwent substantial transformation during the late nineteenth and early twentieth century. In a relatively short period of time dating from the mid-1880s, death rates fell sharply and leading causes of death underwent rearrangement. Falling death rates combined with declines in fertility and falling birth rates to lead to improvements in the chances of infant survival and in the expectation of life.

Examinations of patterns of deaths in their socioeconomic and demographic context have given rise to theories which attempt to order trends in both births and deaths into a systematic whole. The historical mortality data in this work is evidence to test these theories.

Epidemiological transition describes the reduction in the proportion of deaths from communicable diseases and rise in the proportion of deaths from chronic non-communicable diseases. It is also a key component of the demographic transition, the long-term movement from high to low mortality and fertility. By using the mortality profiles developed in previous chapters, the progress of the epidemiological transition in Australia can be described in detail, along with its role in demographic transition.

Falls in mortality rates were driven largely by reductions in deaths among certain age groups and for certain causes of death. Declines in under-five mortality and in deaths from tuberculosis both played important roles, to the extent that they have elsewhere been described as together 'constituting the transition' in Australia (Smith, 1997, p.32). These warrant close examination.

Gastrointestinal infection was the predominant cause of death for both infants and children aged 1–4 years. Among youth and adults, the predominant cause was death from respiratory and other forms of tuberculosis — TB was commonly considered to be the leading cause of death of the era. Under-five deaths and tuberculosis together accounted for over 70% of all deaths in the early 1860s, and around 40% in the early 1900s.

This section begins with comparing deaths and public health issues in 1860 and in 1900. These years provide a context for understanding the mortality transformation which Australian society underwent during this time.

DEATHS IN 1860

By 1860, civil registration in the smaller colonies of Tasmania, Western Australia and South Australia had been underway for several decades. New South Wales, Victoria and Queensland, however, had only recently commenced registering deaths.

The year marks an early phase in the sanitary movement in Australia, the approach to public health which first developed in England out of increasing urbanisation and industrialisation, and which emphasised the removal of filth and waste to combat infectious diseases.

In 1860, Queen Victoria had ruled the United Kingdom of Great Britain and Ireland for 22 years. The colonisation of Australia had been underway for some 70 years, and native-born currency lads and lasses who were children of the first convicts and settlers were now bearing children of their own. The total Australian population was around one million, with 17% aged under five. Sydney's population of 56,000 was less than half of Melbourne's 123,000, which had been swelled by the recent discovery of gold in the region. The life

expectancy of a boy born in 1860 was 40 years, and for a girl 42 years. Queensland celebrated its first anniversary as a separate colony in 1860, and South Australia changed its border from 132 degrees East to 129 degrees East. Sir Joseph Cook, later to become sixth Prime Minister of Australia, was born on 7 December 1860.

The 1860s were dominated by the struggles of selectors and goldminers to gain control of monopolised Crown land from squatters. Numerous expeditions searched for farming pastures in the interior of the country, symptomatic of the demand for land. In April 1860, John McDouall Stuart reached the centre of the continent, and in August Burke and Wills set off from Melbourne to cross Australia from south to north. In December, riots broke out on the Lambing Flat goldfields in New South Wales. Migration continued to increase, attracting free settlers, Chinese fortune seekers and indentured labourers from the South Pacific to a land of opportunity.

Death rates in Australia in 1860 were high. The age-standardised mortality rate for all causes reached 2,059 deaths per 100,000 population, which remains the highest value recorded since the commencement of reliable national record keeping in 1856 (Figure 4.2). The New South Wales mortality rate of 1,983 also remains the highest recorded in the colony. The Victorian rate of 2,196 in 1860 was exceeded only by the rate during the gold rush years of 1853-54. In South Australia, the 1860 rate (1,703 deaths per 100,000 population) would be exceeded only once, in 1875. Tasmanian rates throughout 1847–1858, however, had been higher than in 1860. Only in Western Australia did death rates continue to increase after 1860 (Figure 4.1).

The year was a bad one for epidemics, as the disease profiles in Chapter 6 illustrate. Influenza struck New South Wales, Victoria and Tasmania. Measles were prevalent in New South Wales and Western Australia, diphtheria in Victoria and South Australia, and scarlet fever in Victoria. Childhood gastrointestinal infections were ever-present.

The incumbent Registrars-General in 1860 included Christopher Rolleston in New South Wales, William Henry Archer (who had just taken office) in Victoria, and the long-serving J. F. Cleland in South Australia.

Rolleston lamented that the death rate in Sydney during the second quarter of 1860 was 'fearfully' above the average of the previous three years, being

> 'in the enormous annual ratio of 37 in every thousand of the population, or one death in every 27 persons!...We can find nothing like a parallel to this maximum of Sydney mortality in any of the towns of England...' (*SMH*, 24 Jul. 1860, p.4).

An influenza epidemic raged with great virulence during June in both Sydney city and suburbs, while deaths from measles began in June, increased in July and continued into August (NSWRG, 1860).

The very young and the elderly, along with the poor, were most affected. For Sydney Health Officer Henry Graham, the causes were clear,

> '...the late epidemic here has been more fatal among the poor and working classes, where there has been poverty of diet, want of cleanliness, medical attendance, and other comforts which are always connected with want of employment in the labouring classes...I would now beg to point out four points of a sanitary character to which I respectfully direct attention...1st. Sewerage and Drainage. 2nd Reclaiming the Mud Flats...3rd Closing the City Burial Grounds. 4th Extension of Public and Medical Institutions within the city for the benefit of the sick poor' (*SMH*, 11 Sep. 1860, p.8).

British political economist and logician W. S. Jevons worked as an assayer at the Sydney Mint in the late 1850s. He recorded his social observations as he travelled, considering the inner-city working-class housing to be the worst he had ever seen, lacking even rudimentary drainage and sanitation and exceeding London, Liverpool or Paris in squalor. Jevons castigated the sanitary state of the city,

> '...nowhere have I seen such a retreat for filth and vice as the Rocks of Sydney...nowhere are the country and the beauty of nature so painfully contrasted with the misery and deformity which lie to the charge of man' (*SMH*, 7 Oct. 1858, p.2; 23 Nov. 1929, p.13).

He asked pointedly why Registrar-General Rolleston's mortality figures did not seem to overly concern the City Officer of Health, or the Council, the City Engineer, or the Aldermen of Gipps Ward.

In Victoria, the Central Board of Health also faced criticism for its perceived inaction in the face of increasing deaths, especially from scarlatina and measles,

> 'It has been the pleasing duty of this board for many previous years to report to your Excellency the continued immunity enjoyed by the inhabitants of this colony from the attacks of serious epidemic or endemic disease, but we regret to have now to report that the indications of the past year have shown that this immunity no longer exists, and that disease in an epidemic form, and producing considerable mortality, has been present to a very serious extent…the co-operation of the local board has been called for and given, to the extent of the means at its disposal, in attempting to reduce the mortality in so far as attributable to imperfect cleansing, by enforcing the regulations for the cleansing of lanes and removal of filth from private premises' (*Argus*, 23 Apr. 1861, p.5; 26 Jul. 1861, p.4).

Queensland Registrar F. O. Darvall reported monthly on deaths in Brisbane, and offered a diagnosis,

> 'There has been a great deal of sickness this month, and many deaths; cases of bilious diarrhoea and cholic have been so common as almost to seem epidemic.…Out of a large total of cases [of scarlet fever], the cause of many has probably been vegetable miasm, the result of great moisture, with heat…More than the average sickness has occurred this month. Influenza has been epidemic, and colds very common, also several cases of measles and continued fever have been noticed' (*Moreton Bay Courier*, 3 Mar. 1860, p.2; 5 Apr. 1860, p.2; 7 Jul. 1860, p.2).

Darvall transmitted Queensland's first annual report on births, deaths and marriages to Parliament. Following his remarks on the vicissitudes of establishing the new system of registration, he compares mortality in Brisbane and Sydney,

> 'In the large mortality in Sydney we clearly see the evil effects, even in these salubrious climates, of a condensed population unprovided with the essential safeguards to health, viz. effective sewerage and abundance of pure water. It is earnestly to be hoped that early steps may be taken to guard this town from a like lamentable expenditure of human life' (QldRG, 1860).

South Australia also struggled to come to terms with new epidemics of disease,

'Some little alarm having been excited by the recent prevalence of measles and hooping-cough…measles, a disease unknown to many here, has carried off no less than 70 victims during the quarter, mostly children…Hooping cough numbers only 10 victims during the quarter, and diphtheria 20…We do not think that it necessarily follows from this that Adelaide is an unhealthy city, but wherever large numbers are congregated together, contagious diseases being much more readily propagated, would naturally tend to an increased ratio of mortality…although we are happy to say we have never been visited with any alarming epidemic which has carried off large numbers of our population' (*SAA*, 26 Oct. 1861, p.4).

The small numbers of settlers populating Western Australia were devastated by disease in 1860–61. In March 1860, some influential colonists, who were hoping to establish a sanatorium for retired military personnel, declared that Western Australia had 'no epidemics, no cholera, no measles, no smallpox' (*PG*, 23 Mar. 1860, p.2). By early November, measles has 'at last' appeared in Perth, and by the end of the year there was more than enough reason for the festive season to be dampened,

'This cannot be said to be a Merry Christmas to many of our readers. There are few households in this town where sickness has not appeared, and we have either affliction in our own homes, or we have to deplore sad visitations in those of our neighbours and friends. The measles, which at first most of us imagined would visit the colony but lightly, has tried many families, and in some instances death has entered our dwellings, if not from the direct attack of this disease, yet subject to and consequent upon it' (*Inquirer & Commercial News*, 26 Dec. 1860, p.2).

The Western Australian measles epidemic would grow worse in the New Year, and would strike hardest among the Aboriginal population, whose deaths mostly remained unrecorded and unregistered. A decade later, Registrar-General William Knight reported that

'This Colony, in its history of forty-one years' experience, has been singularly exempt from contagious and destructive diseases, the only interruption of which has been…Measles in 1860…Among the European Population…reckoning altogether 59 cases of death, being 11 in 1860, and 48 in 1861…With the Aborigines the disease caused a widespread desolation, extending from tribe to tribe with most fatal consequences' (Knight, 1870, pp.23–24).

The *Australian Medical Journal* was the only active medical journal in Australia in 1860, then being in its fifth year (Due, 1994). Its content for the year concerned itself with *de rigeur* Victorian-era medical and surgical matters; articles on dislocations, uterine diseases, snakebite and syphilis, the merits of ether versus chloroform, and a new splint for the treatment of leg fractures. Vital statistics were championed as holding great promise for the advance of medical research. The burgeoning field of public health saw discussions on the comparative effects of climate on certain diseases, the link between water from Melbourne's Yan Yean reservoir and lead poisoning, and some observations on an outbreak of diphtheria in the colony of Victoria in 1859.

In the October issue, Tasmanian medical practitioner and pioneering epidemiologist Edward Swarbreck Hall published a lengthy article on the influenza epidemic which had attacked Hobart earlier in the year. For several years Hall had been extracting and classifying monthly records of death from the Registrar-General's collection. In accordance with the prevailing miasmatic theory of disease causation, he 'weighed the presumed influence of each meteorological phenomenon in its effects on the diseases and deaths of the month' (Hall, 1860, p.252).

Of the nearly 25,000 persons in the Hobart registration district, 23,000 were attacked by the epidemic, which directly or indirectly caused 100 out of a total of 113 deaths in July. Hall could not remember

> '…any invasion of epidemic influenza ever before so general or so fatal…Little sanitary knowledge is required to discover the "fount of evil" in a city where the water supply is altogether short of its requirements; drainage as defective as possible; cesspools everywhere; efficient ventilation nowhere; and quantities of decomposed food constantly sold and consumed' (p.252, 263).

Hall made a detailed examination of the meteorological conditions of the city at the time of the epidemic. He noticed the high barometric pressure which produced 'congestion of the internal organs and ruptures of diseased blood vessels', land winds which were 'least favourable to atmospheric purity and health', daily variations and extremes in temperature 'that act so injuriously on the human organism', and rain which caused streets to exhibit black mud and

green slime 'so characteristic of stagnant foul moisture, and so polluting to the atmosphere'.

If this were not enough, large amounts of ozone were also present, which when combined with 'high, cold, dry winds, might irritate and mucous membrane of the air passages, nose, larynx, bronchial tubes, &c., and so produce one of the prominent symptoms of epidemic influenza, as well as ordinary catarrh'. For Hall, public health was seldom favourable 'when the sky is grey, the air moist, the temperature low, the daylight diminished, and the electricity negative' (p. 269).

Eighteen-sixty was the year in which epidemic disease broke out in the Australian population in a fashion not previously experienced. The mortality burden added fuel to the fire of sanitary reform, and lent weight to growing calls for public health improvement. Sydney Health Officer Henry Graham sounded a clarion call,

> 'While public attention is directed to the necessity of erecting defences for the protection of the city against the invasion of an enemy, in the event of war taking place at a future period between England and other European powers, equal necessity exists to guard against the invasion of disease, which might decimate the population, and to guard the community against those influences which are calculated to undermine the health of the citizens' (*SMH*, 19 Mar. 1860, p.5).

Battle was joined. The enemy, however, was unseen, and still largely unknown.

DEATHS IN 1900

By 1900, germ theory had captured the high ground of disease causation. The formulations, laboratory discoveries and field trials undertaken by Henle, Pasteur, Koch and others had convinced the Australian medical establishment to shift its thinking, although a minority of physicians and public health officials still clung to miasma and filth theories. The proof and further elaboration of germ theory would lead to an assault on all epidemic and infectious diseases.

Queen Victoria was in her sixty-third and final year as reigning monarch over the British Empire. The Australian colonies would Federate and form one Commonwealth of Australia on 1 January 1901. The States, as they were henceforth to be known, would keep their own systems of government but would now also have a federal government. In years to come, public health would increasingly become a federal responsibility.

The total population of Australia in 1900 was 3.7 million, more than three times that of 1860. Around 12% of the population were aged under five years, a fall from the 17% of 1860. Life expectancies had increased by one-third; a baby boy born in 1900 could expect to live 53 years, and a girl 57 years. The populations of Sydney and Melbourne had both grown rapidly, and Sydney had now reached Melbourne's level of around 480,000 persons. Following the gold-driven migration of the 1850s, another great period of migration had begun in 1876 which continued until 1890. The 1870s and 1880s were years of urban boom, economic expansion and widespread prosperity in Australia. The 1890s, in contrast, were characterised by social unrest, strikes and depression (Fitzgerald, 1987, p.223).

During 1900, electric lighting was installed on Adelaide streets. The *Glenelg* and the *Sierra Nevada* were both wrecked off the Victorian coast, with a combined loss of over fifty lives. Australian military contingents were involved in the Boer War in South Africa and in the suppression of the Boxer Rebellion in China. Politician John McEwan and cricketer Bill Ponsford were both born in 1900, and three-time Premier of Queensland Thomas McIlwraith died.

Reflecting the optimism surrounding the imminent Federation, all colonies were encouraged by their favourable health situations in 1900, as the mortality abstracts and health authority annual reports make clear. The only sour notes were stubbornly high infant mortality and rises in proportions of deaths from chronic diseases such heart disease and the increased detection and diagnosis of cancer. Timothy Coghlan, the New South Wales Statistician, summarised the situation in the colonies,

> 'The death-rate of Australia is much below that of any of the European states, and is steadily declining. Every year sees an advance in the sanitary condition of the people in the large centres of population, and to this cause may be ascribed the greater part of the improvement in the death-rate…In spite of all the sanitary improvements that have been effected in recent years, the [infant mortality] rate, as judged from the last quinquennial period does not seem to have decreased very appreciably in any of the states except South Australia' (Coghlan, 1902b, pp.503, 509).

The all-cause mortality rate for Australia had fallen by almost half since 1860 to be 1,123 per 100,000 population in 1900. Rates in each colony were much the same, with the sole exception of Western Australia, which now had the highest rate at 1,337, a value almost exactly the same as in 1860.

Mortality reporting had gained in sophistication, with much added detail. In his 1900–01 state report, Coghlan took pains to point out differences in rates of death between metropolitan and country districts. Although rates had declined, infection still claimed many lives, with notification of cases of infectious disease increasingly becoming compulsory. By 1900, most colonies had introduced notification for typhoid fever, scarlet fever, diphtheria, leprosy, relapsing fever, variola, yellow fever, and for the newly arrived bubonic plague. Typhoid fever outbreaks featured in Newcastle Medical Officer of Health Robert Dick's annual report, along with the oft-repeated plea for cleaner water and more efficient sewage and waste disposal (*AMG*, 20 Sep. 1901, pp.403–406).

Infant mortality in areas of New South Wales remained unacceptably high, with the blunt observation that 'So far as Sydney and its suburbs are concerned, there is no cause for congratulation; on the contrary, the rate at which children of tender years drop into the grave forms a pathetic commentary on the civilisation of the state' (Coghlan, 1902a, p.1004). Despite this, the metropolitan mortality rate was one of the lowest yet recorded. Pulmonary tuberculosis remained a formidable foe, although chronic diseases were increasingly being registered,

> '…the number of deaths from phthisis in the state during 1900 was greater than from any other disease. The mortality rate per 10,000 living from this cause was, however, lower for the five years ending 1900 than for any previous five-year

period since 1871…the diseases which deserve special mention…are cancer, phthisis and heart disease…Accidental deaths are very numerous, especially in the country' (Coghlan, 1902a, p.1014-15, 1018).

Following the presentation of James Jamieson's annual report on Melbourne's health, authorities felt that there was at least some cause for celebration.

'…the information it contained was the subject matter for hearty congratulation, inasmuch as a record had been established as far as the health of the city was concerned. It was especially gratifying to note the small death rate among infants and the all-round reduction of mortality. As it had been particularly noticed that the community were now enjoying the sanitary advantages which followed the completion of sewerage connections, the health committee were very desirous of seeing that important work properly finished' (*Argus*, 30 May 1901, p. 8)

Victorian government statistician James J. Fenton concurred on the matter of lower death rates, but also took note of the increase in deaths from chronic diseases, as in New South Wales,

'The rate of mortality in 1900 was also the lowest in the last twenty years. Ever since 1892–with one exception, 1898–the mortality has been exceptionally low. …The mortality in 1900 from all causes…except measles, cancer, and heart diseases, were considerably below the average of the last ten years' (*VicGG*, 24 May 1901, pp.1692, 1695).
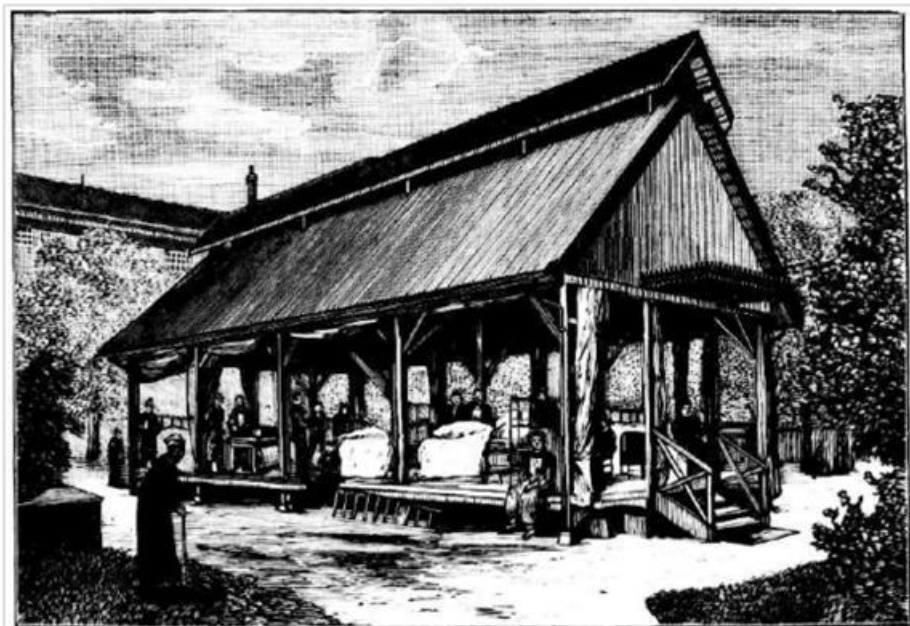

Figure 7.1: Isolating pavilions for contagious diseases (*Illustrated Sydney News*, 12 Apr. 1884)

In Queensland, the total death rate had also fallen, notwithstanding a rate among the ten thousand or so Pacific Islanders which was three times that of Europeans (31 vs. 12 per 1,000 population). Infant mortality in mining centres such as Gympie and Westwood was especially bad, comprising nearly half of the total deaths of the district (*QldRG*, 1900, p. xxv). Phthisis remained the leading cause of death in Queensland, again being over-represented among the Pacific Island population, since they were 'peculiarly liable to the ravages of these complaints', and 'taking less care of themselves, they contract these kinds of diseases here with a greater readiness'. Cancer was 'much more prevalent than it was a few years ago', and for heart disease, 'a greater proportion of the deaths is now ascribed to this as a cause' (*Ibid*, p. xxxii, xxxiii).

Lower mortality in Adelaide in 1900 was seen as evidence of the cities' claim as a 'sanitary city', with the years' work in improving general sanitation, especially in the poorer parts of town, ascribed to the 'systematic and effective work of the inspectors and trained nurse' (*AMG*, 20 Jan. 1902, p.48). Across the whole State, 'considering the diminution in number, in rate, in deaths from zymotic diseases, especially from enteric fever, and in infantile mortality, it may be doubted whether South Australia has ever known a better year' (*SARG*, 1900, p.8).

Hobart, in Tasmania, enjoyed a 'remarkably low' death rate during the year, much lower than the other capital cities (*Mercury*, 27 May 1901, p.2). In Launceston, the health of the citizens had been 'very good, as the death rate was very low, probably the lowest on record'. The Government Statistician noted the lowness of the general death rate over the whole of Tasmania (*Launceston Examiner*, 26 Feb 1901, p.3).

Even Western Australia, which by now had the highest death rate of all the colonies, felt there was some cause for celebration. Mortality in 1900 was at a ten-year low, with abatement in the prevalence of typhoid fever on the goldfields, and infant mortality had also declined somewhat following the extremely high rates of the mid-1890s. Dr. Hope, the Medical Officer for Fremantle, was indeed hopeful,

'It might be taken that the…figures indicated a continuous improvement in the sanitary and hygienic conditions of the municipality, and gave reasonable expectation that typhoid would, before long, cease to exist in this locality' (*The West Australian*, 2 Mar. 1901, p.11).

Fifty-two cancer deaths were registered in Western Australia in 1900, and this would climb to 83 in 1901. Phthisis deaths continued to increase, however, reaching over six per cent of total deaths in 1900. Deaths due to accidents were still common, because of the 'increased employment of labour during recent years in the mining, timber, and other industries of the State, and also on railways and other public works' (Fraser, 1902, p.54).

As in other parts of the country, the risk of death in the metropolitan district was greater, although Western Australian Registrar-General Malcolm A. C. Fraser cautioned that the whole of this higher death rate should not be set down to inferior sanitary conditions (*Ibid*, p.46). He held that the age distribution of the population and the movement of the sick and aged to urban health care facilities co-operated to swell the metropolitan death-rate.

The self-congratulation over improved mortality had an element of bravado. Most states were still unhappy with the high number of childhood deaths, and cancer and cardiovascular disease increasingly drew attention as leading causes of death (Coghlan, 1902b, pp.522, 525).

Overshadowing this, the year will be forever remembered for the outbreak of bubonic plague, part of the last great pandemic which had originated in China, and which had reached most parts of the world by 1900 (Curson, 1985a, p.137). This visitation was a first for Australia, and although the epidemic would ultimately involve only a relatively small number of cases and deaths, widespread hysteria and panic ensued (Curson & McCracken, 1989). Most of the 458 cases which appeared in 1900 occurred in Sydney (303 cases), with Brisbane, Rockhampton and Townsville also featuring. There were some 154 resultant deaths nation-wide, representing a case fatality of one-in-three.

Medical men were divided as to the cause of this new disease. Some still took refuge in miasmatic theory, thinking that the disease arose from filth, bad air or

insanitary living conditions. Some people burnt barrels of tar in the streets in the hope that this would purify the air. Others, perhaps over-enthused with the germ theory revolution, thought that a bacteria was responsible, which was gastrointestinal in origin and which spread through personal contact. Still others accepted the recently proposed rat-flea theory (Curson, 1985a, p.139).

Sydney Medical Officer of Health W. G. Armstrong played a central role in dealing with the outbreak. In addition to his actions, his research—along with that of other medical officers such as Ashburton-Thompson, Tidswell and Burnett Ham—added to the understanding of how the *Yersinia pestis* bacteria spreads to humans by fleas from infected rats.

Armstrong understood that big public health issues such as plague needed big government solutions, although these were often painfully slow in coming,

> 'The year will always be a marked one in the sanitary history of the metropolis for several reasons. The fatal and deplorable invasion of Sydney by the bubonic plague, the resumption by the State Government of certain slum areas, and the amendment of the Corporation Act of the City of Sydney in some most important directions, stamp the year as one to be remembered' (*AMG*, 20 Aug. 1901, p.349).

Plague spanned the continent, reaching Fremantle in Western Australia during the year. Although it was felt that an outbreak could not be avoided, authorities believed that the effects could at least be contained,

> 'We can scarcely escape another visitation of plague, as the communication with those countries where plague exists is so regular. The risk to human life, and the amount of fear which is produced by this knowledge, will depend upon the perfection to which the municipal authorities bring the cleanliness and healthiness of towns, and the confidence the public have in their sanitary surroundings' (*The West Australian*, 2 Mar. 1901, p.11).

In all states, but especially in New South Wales, the plague outbreak galvanised public health efforts and sanitary reform. Gangs of workmen exterminated rats, premises were inspected, fumigated, disinfected and cleaned. Waste, rubbish and filth were removed, and sewers and drains were flushed. Some buildings in bad repair were demolished. In Sydney, the epidemic led to the resumption and remodelling of the wharves and the area adjacent to Darling Harbour, where

the outbreak was at its worst. Health administration was also modernised. Inspectors were appointed, isolation wards in hospitals were established, quarantine facilities were improved, and new medical laboratories opened (Lewis, 2003, pp.125–134).

The plague put a brake on the optimism of the civil and medical authorities, lest they think that their lower mortality rates meant the winning of the public health war. The appearance of this most fearsome disease again highlighted the shortcomings of urban public health and living conditions. Socially, the disease proved to be highly selective, spreading because of the substandard and insanitary living and working conditions of the city's poor (Lewis, in Cumpston, 1989, p.197; Curson, 1985a, pp.144–146). But that was not news; Jevons had said as much half a century before.

THE ROLE OF INFANT AND UNDER-FIVE MORTALITY

In public health, infants and young children are among the most vulnerable. This was starkly demonstrated in nineteenth-century Australia by the sheer numbers of infant and under-five deaths; the mortality abstracts documenting over 50,000 registrations in Australia during 1860–64 alone. More than half of the total deaths in this five-year period were children under five. These deaths were comprised in a ratio of 60:40 between infants under one year of age and children aged 1–4 years (Table 7.1).

Table 7.1: Deaths under five years of age, Australia, 1860–64 to 1900–04

	1860–64	1870–74	1880–84	1890–94	1900–04
	Number				
Age 0	31,621	36,990	51,293	59,690	51,406
Age 1–4	21,946	18,373	22,651	24,308	16,249
Age under 5	53,567	55,363	73,944	83,998	67,655
Deaths among all ages	*106,793*	*125,216*	*182,291*	*222,767*	*228,964*
	Per cent of total deaths				
Age 0	29.6	29.5	28.1	26.8	22.5
Age 1–4	20.5	14.7	12.4	10.9	7.1
Age under 5	50.2	44.2	40.6	37.7	29.5

Two decades later, in 1880–84, the proportion of under-five deaths had eased slightly to two-fifths of the total number, at a ratio of 70:30, and by the turn of the century deaths under five comprised 30% of all deaths, divided 75:25 among infants and ages 1–4 years.

The sad legacy is that in the fifty years between 1856 and 1906, there were 687,000 registered deaths under five years of age—472,000 infants and 216,000 children aged 1–4 years—summing to almost 40% of all deaths.

Yet as has been shown, declines in death rates among children dominated survival improvements in Australia between 1856 and 1906. More than half of the total change in life expectancy during this time was achieved through reductions in mortality among children under five years of age (Tables 4.4 and 4.5) with improvements in mortality among ages 1–4 years outweighing the contribution from reduced infant mortality.


Figure 7.2: Convalescent. A sketch at the Children's Hospital (*Illustrated Australian News***, 6 Apr. 1881)**

The relative contribution of each age group to improvements in life expectancy serves as a useful indicator of the stage of demographic transition. In populations with low life expectancy at birth, and at an early stage in the transition, the proportion of improvement in life expectancy that is due to improvements in survival among children outweighs that contributed by adults. As life expectancy at birth improves, the contribution of older ages increases (United Nations, 2012). The improved survival among infants and young children at this time indicates that the late nineteenth century was still an early phase of the transition in Australia.

Infant mortality rates varied greatly among the colonies from 1856 to 1906. In the 1860s and 1870s, rates were highest in Victoria, Queensland and South Australia, on occasion exceeding 150 deaths per 1,000 live births, but were relatively low in Tasmania (Figure 7.3) (Hayter, 1878–79). Rates in New South Wales fluctuated between 90 and 130 deaths per 1,000 live births until 1903. Infant mortality began to fall in Queensland and South Australia from the 1870s, in Tasmania from the mid-1880s, Victoria from the early 1890s and then in New South Wales from the early 1900s. Infant mortality fluctuated greatly in Western Australia, with an overall increase until the late-1890s, after which the rate began to fall.

Figure 7.4 shows mortality rates for the whole of Australia and for three different age groups; infant mortality (per 1,000 live births), age 1–4 years, and under-five (both per 1,000 population). Infant mortality remained high from the mid-1850s to the mid-1880s, fluctuating around 125 deaths per 1,000 live births, after which it began to fall, albeit slowly at first. A turning-point analysis (introduced in Chapter 4) indicates two important turning points, with a moderate decline in infant mortality rate for Australia commencing in 1885, and accelerating after 1903.

Figure 7.3: Infant mortality rates by colony, 1856–1906
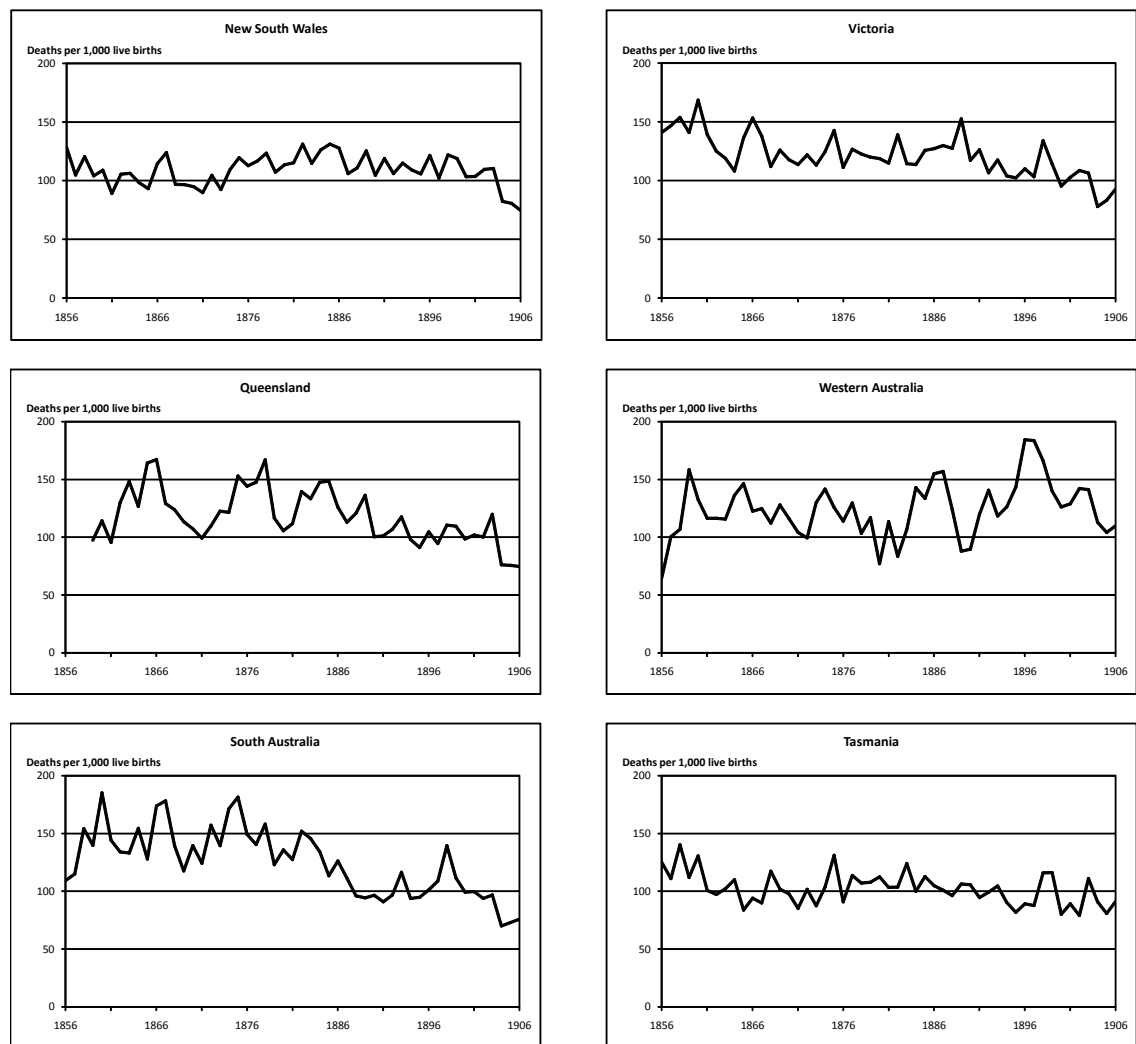
In 1885, the infant mortality rate in Australia stood at 128 deaths per 1,000 live births, and by 1903 it had fallen somewhat to 111. This was the break-through year, with the rate falling sharply to 82 in 1904, a fall of over one-quarter in a single year. Never again would infant mortality in Australia rise above 100 deaths per 1,000 live births, although rates in some parts of the country (Western Australia and later, the Northern Territory) were to remain high for some years yet.

Figure 7.4: Infant, age 1–4 and under-five mortality in Australia, 1856–1906
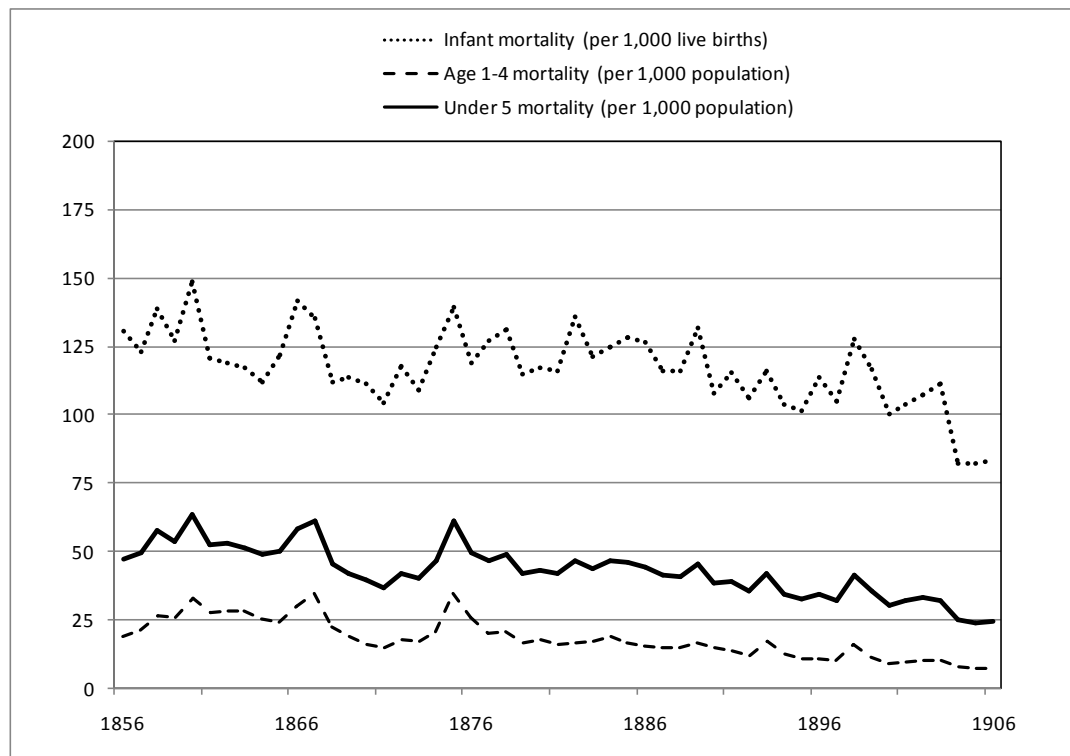
Mortality among the age group 1–4 years unfolded differently from infants, with a slow, steady decline dating from as long ago as 1860 being punctuated by high epidemic death rates in 1867, 1875, 1893 and 1898. In 1860, the death rate among children aged 1–4 years was 33 per 1,000 population but by 1906 this had declined to 7, a fall of almost 80%.

The under-five mortality curve reflects trends in both infant and in age 1–4 mortality, with a turning point in 1889 (Figure 7.4).

The profound nature of both these declines is more apparent when examining longer-run falls (Figure 7.5). Infant mortality continued to decline rapidly following the plunge of 1903–04, falling to below 75 deaths per 1,000 live births in the 1910s, then 50 in the 1930s, and 25 in the 1950s. Age 1–4 mortality also continued its steady decline, falling by another 80% (from 7 to 1.6) between 1906 and 1954.

Figure 7.5: Infant, age 1–4 and under-five mortality in Australia, 1856–2006
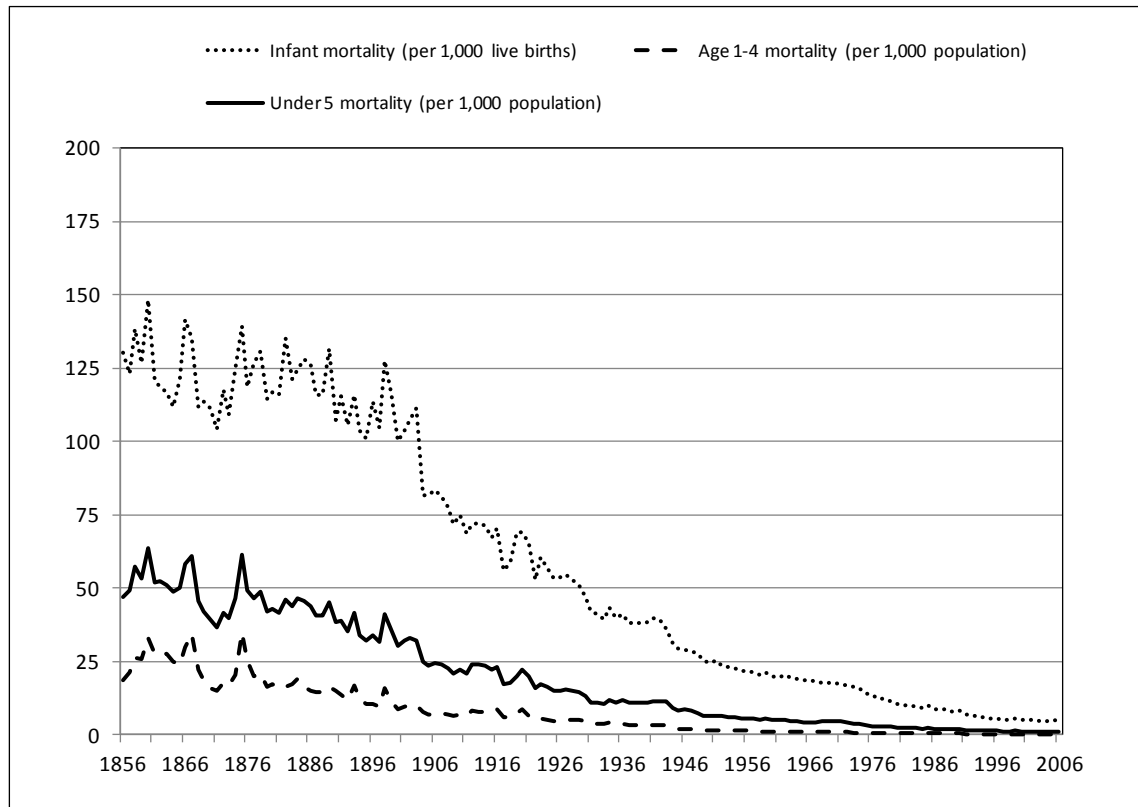
Source: ABS (2008a) and AIHW (2014) for 1907 onward.

The information in the mortality abstracts allows for the further dissection of infant and under-five mortality rates by age at death. The abstracts for Victoria contain counts of infant deaths for ages less than 1 month, 1–2 months, 3–5 months and 6–12 months, as well as for one, two, three and four years of age for the years 1867 onward.

In each selected five-year period between 1870–74 and 1900–04, there were two age groups in which deaths peaked in Victoria—during the first four weeks of life, and again between six and 12 months of age (Figure 7.6).

Among all children born in Victoria during the years 1880–84, 17% died during the first five years of life, with 12% dying in the first year, and 4% during the first month of life. Mortality during the first five years of life declined quickly after the first year.

The classification of deaths into these age groups makes possible the construction of neonatal and post-neonatal mortality rates, with neonatal mortality being deaths of a live-born infant from 0 to 27 days of life, and post-neonatal mortality being deaths from 28 through to 364 days of life.

Figure 7.6: Age-specific deaths for under-five mortality, Victoria, 1870–74 to 1900–04
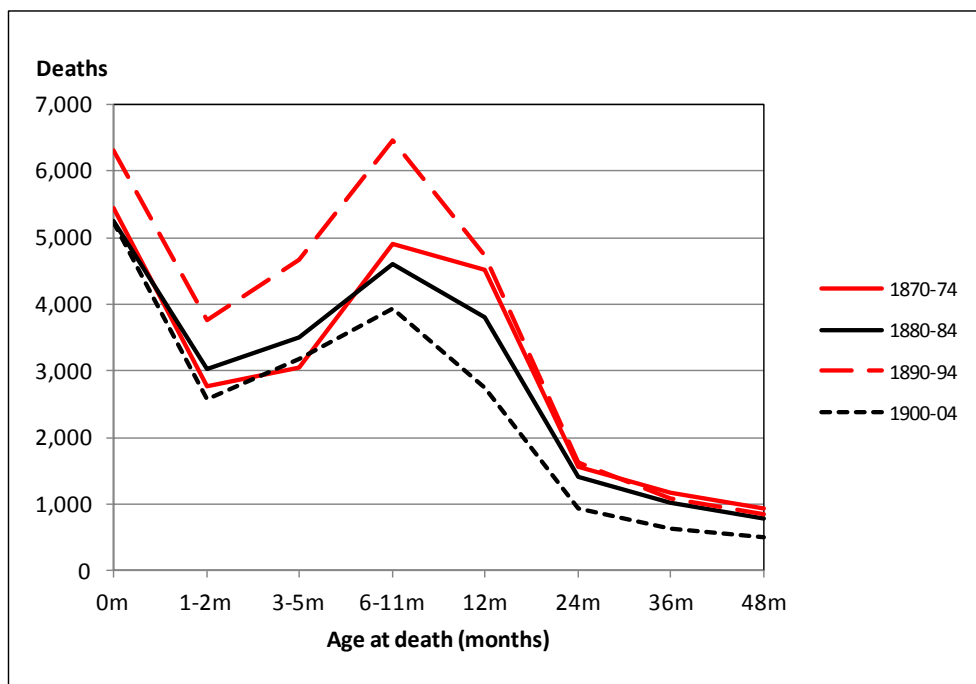
These rates give greater insight into the processes leading to death. William Farr first recognised that factors affecting the death rate vary at different stages of infancy, with the need to further subdivide the first year of life into months or even days. Most deaths during the neonatal period are associated with events surrounding the prenatal period and delivery, whereas post-neonatal deaths are more likely to be associated with conditions or events that arise after delivery, and which therefore reflect environmental factors.

Figure 7.7 makes clear that the factors responsible for the declining death rate among infants in Victoria were largely environmental in nature. Whereas neonatal mortality remained almost constant between 1867 and 1906, falling only slightly from 42 to 34 deaths per 1,000 live births, post-neonatal mortality was halved in the two decades from the mid-1880s to the mid-1900s.

Figure 7.7: Neonatal, post-neonatal and infant mortality, Victoria, 1867–1906
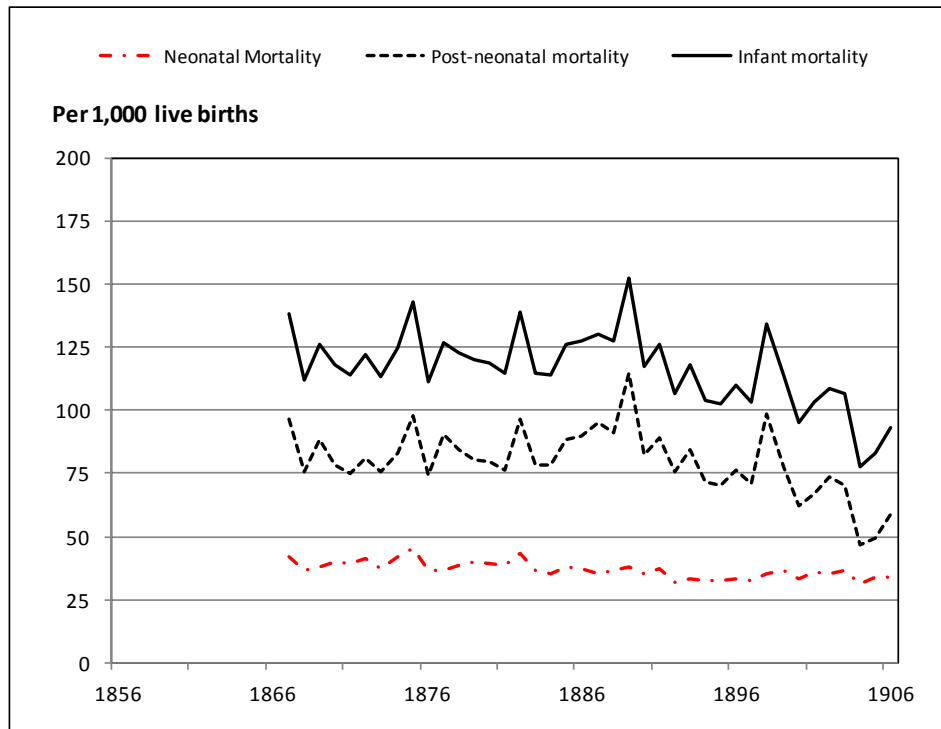
Mortality was always higher in urban regions. Between 1870 and the mid-1880s, infant mortality in both the Sydney metropolitan region, and in Greater Melbourne averaged around 75% higher than in the rest of New South Wales and Victoria (Figure 7.8). In the 15-year period from 1885 to 1900, the differential narrowed substantially, following improvements in urban infant mortality rates. Infant mortality outside the urban regions showed little change. After 1900 there was greater parity between urban and non-urban rates, although urban rates remained higher.

Figure 7.8: Rate ratio of urban: non-urban infant mortality rates in New South Wales, Victoria and Western Australia
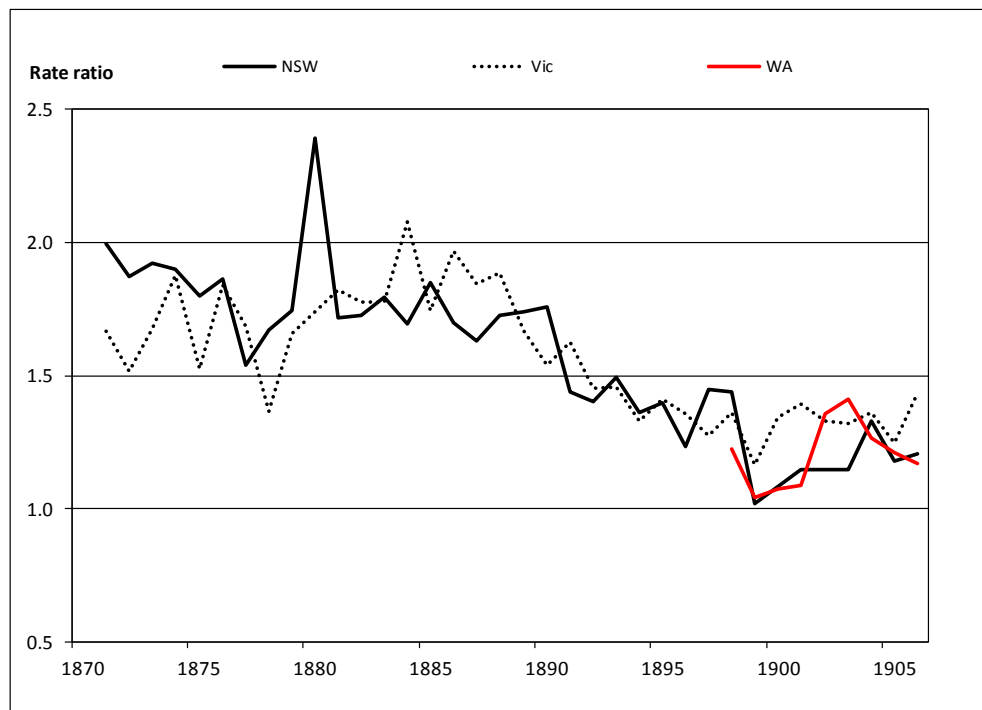
Under-five deaths exhibited a seasonal pattern. Deaths in Melbourne and suburbs between 1880 and 1903, for example, show peaks characterising a greater number of deaths during the summer months of December, January and February (Figure 7.9). The summers of 1886 to 1892 were especially severe, with over 400 and up to 700 deaths registered in any one month. Those commentators who linked the increased mortality to climatic conditions noted that the period was characterised by very heavy rainfall, which preceded the so-called 'Federation' drought commencing in 1895.

Increased summer mortality was more pronounced among infants than among children aged 1–4. Infants were more prone to causes of death which were prevalent during the season, these typically being the food- and water-borne infectious diseases (Fisman, 2007). As Archer had written,

> 'Medical men, from Hippocrates to Farr have noted the existence of a connection between high temperature and an increased prevalence of dysentery and diarrhoea among persons of all ages; but by limiting the comparison…to infants under

twelve months old, the result of climatic influence is brought out in far bolder relief' (Archer, 1859).

Mortality among ages 1–4 also showed some evidence of seasonality.

Figure 7.9: Seasonality of under-five mortality, Melbourne and suburbs, 1880–1903
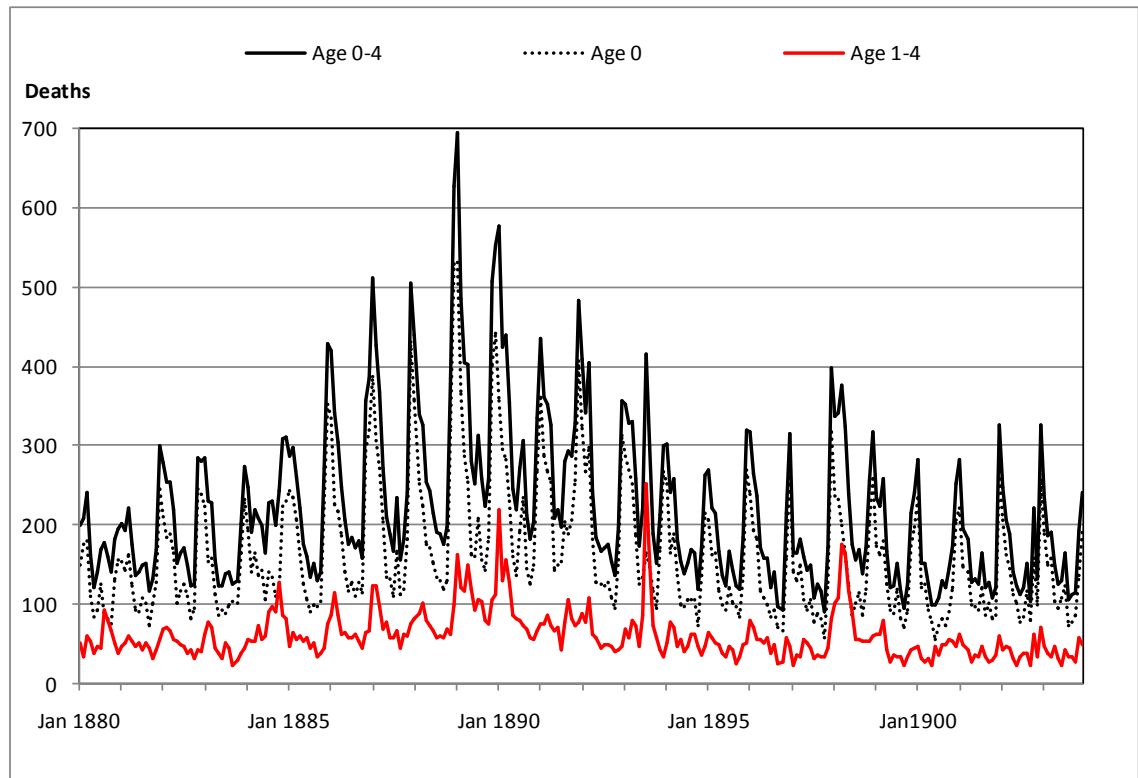
The profiles of mortality which were developed in Chapter 6 introduced some of the causes responsible for such high rates of under-five mortality. The gastrointestinal infections, returned in the abstracts under 'diarrhoea', 'dysentery', 'cholera' and 'enteritis', as well 'convulsions', 'teething', 'atrophy' and 'debility' were especially disastrous for infants.

The five main causes of infant mortality presented in Figure 7.10 show the pre-eminent role of diarrhoea (and see also Anon., 1908). Over the entire period 1864–1906, deaths from gastrointestinal infections in Australia (categories 1–3 in Figure 7.10) averaged over half (54%) of all infant deaths.

Figure 7.10: Cause-specific rates of infant mortality and their proportional contribution, Australia, 1864-1906
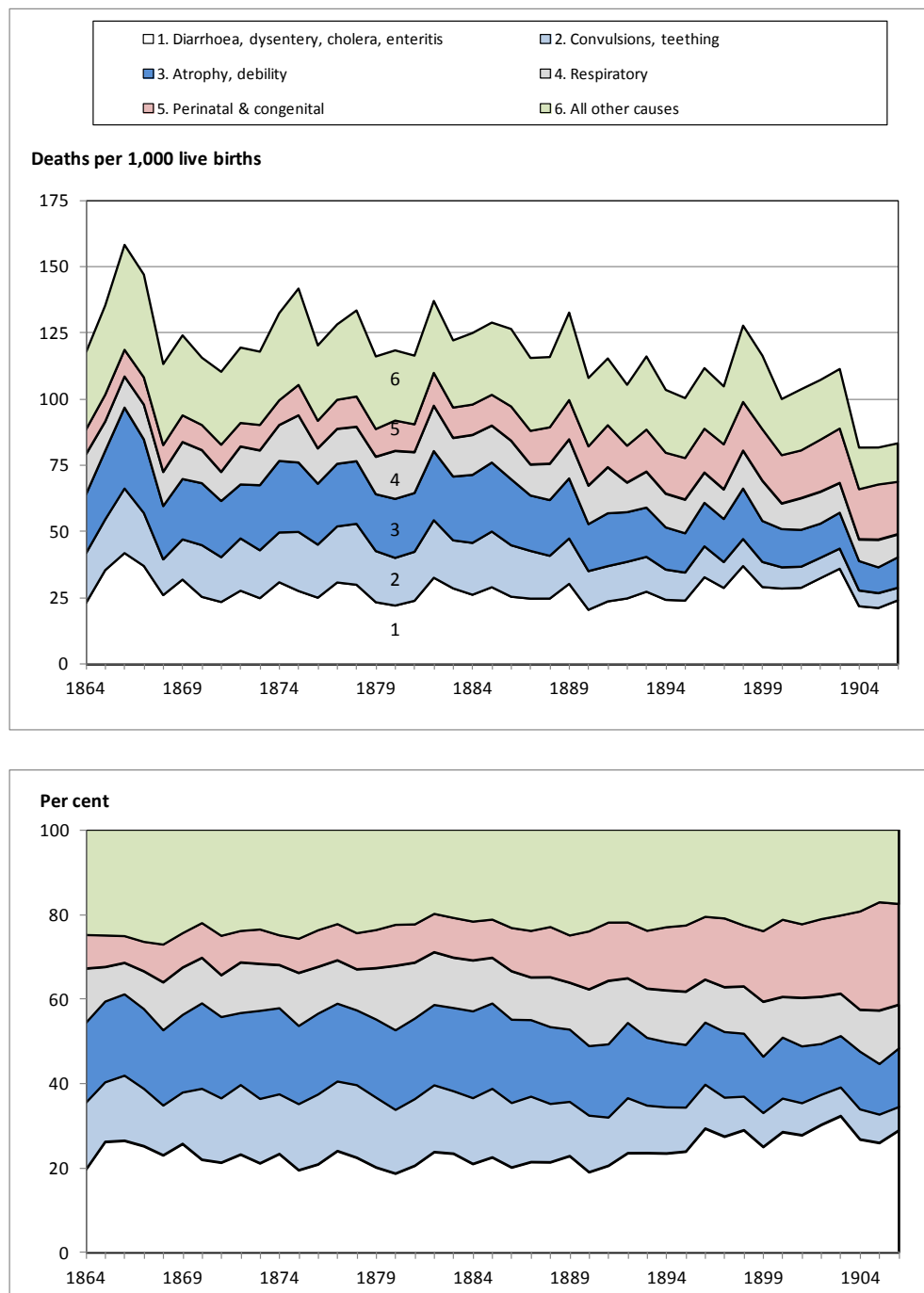
Mein Smith (1997, p.43–44) also emphasised the debilitating effects of these diseases on infants, although for this author the makeup of causes differed slightly. Some additional causes were added such as 'want of breast milk' and 'malnutrition' (which have here been classified under 'Endocrine, nutritional and metabolic disorders') along with 'diseases of the stomach'. Mein Smith also

discounted to 75% the proportion of 'debility and atrophy' deaths which were thought to be caused by diarrhoea, and 'convulsions' was also discounted to 50%. The broad conclusion that half of all infant deaths were due to gastrointestinal infections remains, as the early paediatricians Litchfield (1905) and Armstrong (1905) had maintained.

The relative contribution of each cause to total deaths among infants changed somewhat over time. Diarrhoea, dysentery, cholera and enteritis were responsible for 21% of all infant deaths in 1880–84, rising to 29% in 1902–06 (Figure 7.10, lower panel). At least part of this rise was due to the reassigning of deaths from other outmoded categories such as convulsions and teething (15% in 1880–84, down to 7% in 1902–06), and atrophy and debility (19 in 1880–84, down to 13% in 1902–06) (Armstrong, 1905).

Taken together, all gastrointestinal infections (diarrhoea, dysentery, cholera, enteritis, convulsions, teething, atrophy and debility) caused 56% of all infant deaths in 1880–84, falling only slightly to 48% in 1902–06. Although the infant mortality rate began to fall after 1885, and more rapidly after 1903, the contribution of gastrointestinal infections as a proportion of all infant deaths remained largely unchanged.

One group that did rise substantially was deaths due to congenital and perinatal causes. The neonatal mortality rate remained constant over the period (Figure 7.7), but congenital and perinatal deaths comprised an increasing proportion of infant deaths over the period, rising from 9% in 1880–84 to 22% in 1902–06. It is difficult to maintain that this represents a real change in underlying causes of infant mortality, and instead it is more likely to be due to greater precision in determining and coding cause-of-death.

Respiratory deaths, and the residual 'All other causes' category, the other two major groups in Figure 7.10, remained largely unchanged over the period, contributing around 12% and 20% of infant deaths respectively.

Causes of death among the 1–4 age group differed to those for infants (Figure 7.11). Gastrointestinal diseases were still formidable, consistently claiming one-third of all children in the age group, even as rates fell. However, convulsions

and teething were responsible for a much lower proportion of deaths among ages 1–4, and atrophy and debility have disappeared as major causes. Other infectious diseases assume greater importance, with diphtheria, for example, responsible for an average of 8% of deaths.

Figure 7.11: Cause-specific rates of age 1–4 mortality and their proportional contribution, Australia, 1864-1906
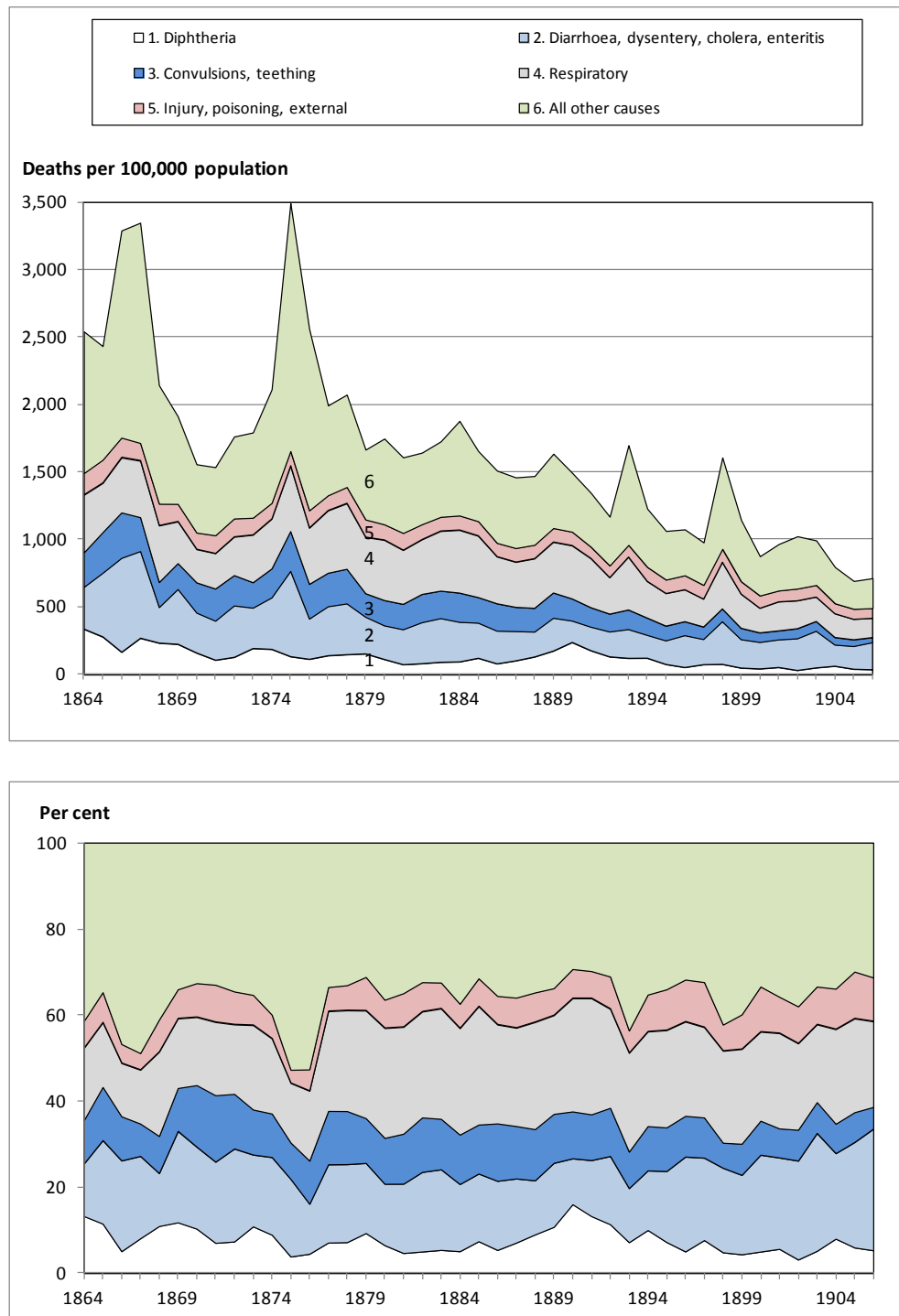
Mortality peaks in 1866-67, 1875, 1893 and 1898 reflect epidemics of measles, whooping cough and scarlet fever, alone or in combination. Respiratory diseases averaged 20% of mortality, and external causes another 7%. Various forms of tuberculosis add to the disease burden for young children.

Again, although mortality rates for children aged 1–4 declined over the years 1864–1906, the relative contribution of each cause of death remained reasonably constant (Figure 7.11, lower panel). There was little epidemiological transition to speak of among age 1–4 deaths, in that gastrointestinal infections were still responsible for most deaths at the end of the period, just as they were at the beginning. However, the ferocity of epidemics did ease over the period.

Indeed, why should there be any marked transition in either infant or age 1–4 causes of death? Although germ theory had revolutionised public health and had led to the discovery of many of the pathogens responsible for infectious diseases, the years 1856 to 1906 saw little progress in developing vaccines or cures for infant and childhood diseases, the diphtheria antitoxin notwithstanding. The infant welfare movement was yet to make its mark. Melbourne Medical Officer A. Jeffreys Wood reflected in a lecture to the Australian Health Society that,

> 'I have dwelt on…the saving of infant life on account of the absolute feeling of helplessness that besets my colleagues and myself at the Children's Hospital during the summer months, when we have 1,000 babies a week coming up for treatment for diarrhoea. Medicines and advice are useless when the baby has to be sent back to the sweltering dusty lanes in the city, to be re-poisoned by dirty milk and city germ-laden air' (Jeffreys Wood, 1908).

A number of risk factors—both proximal and distal—have been linked to the decline in the rate of fatal intestinal infections, and thus to the fall in infant mortality. These range from effects of the economic depression of the 1890s, to sanitary reforms such as the cleanup campaigns in response to the outbreak of plague in 1900, to low rainfall during the period, and to the greater prevalence of breast feeding in the decade prior to 1910. Each has been deemed to have 'played some part' in infant mortality decline, although the quantification of how much has proven elusive (Cumpston, 1989, p.109-110).

It is, perhaps, enough to say that reduced infant and age 1–4 mortality at the start of the new century largely reflected the amelioration of a number of environmental risk factors such as unclean water and waste, along with the improvement of public health infrastructure, and the betterment of some of the conditions in which children were born, lived and grew.

The decline in infant and childhood mortality in the fifteen years after 1890 should not be understated. Public health measures had helped to contain and to some extent prevent infectious disease, but although medical science in early twentieth century Australia had advanced, its contribution was yet to reach fruition. Better education of women also played a role, so that mothers reaching the age of fertility at the turn of the century were the first cohort of Australian women to enjoy universal primary education (Lewis, 2003, p. 50).

Real change, through addressing the underlying causes of infant and child mortality and preventing or curing the diseases responsible was still some way off. Three things were necessary: a fuller understanding of the epidemiology of infant diseases and how they behaved and spread, an understanding of the pathology of diseases and what they did within the body, and an understanding of the nature of the pathogen, and which organisms caused disease (Barry, 2004, p.256).

Beyond mortality, other broader demographic processes played important roles leading to the decline in the death rate. Reduced fertility and family transition occurred amid economic prosperity, and a greater prevalence of contraception and spacing of births led to smaller completed families and better nurturing (Smith, 1997, p.42–43). The cost of rearing children, the empowerment of women, and the influence of education on both women and children motivated parents to limit family size (Mein Smith, 1997, p.25ff). Parenthetically, gut infections affected families large and small, rich and poor, educated and uneducated, although infants and young children in smaller families might conceivably have had improved survival chances.

Traditional demographic transition theory holds that the declines in mortality and fertility were both key factors, but the precise sequence of causation—

whether under-five mortality fell as a result of a fall in fertility, or vice versa—continues to be debated (Dyson, 2010; van Poppel *et al.*, 2012).

THE ROLE OF TUBERCULOSIS

Tuberculosis is a common and often lethal infectious disease caused by mycobacteria, usually *Mycobacterium tuberculosis*. It typically attacks the lungs, but can also affect other parts of the body, and is spread when people who have an active infection cough, sneeze or otherwise transmit respiratory fluids.

Respiratory tuberculosis, or tuberculosis of the lungs, was commonly known in the nineteenth century as *consumption*, since it generally manifested itself through wasting of the body. Another archaic term for the disease was *phthisis*, which had its origins in the Greek *phthinein*, also meaning to waste away, this being akin to the Sanskrit *kṣiṇoti*, 'he destroys'.

The onset of respiratory tuberculosis is gradual but persistent, with a lack of energy, weight loss, and a cough leading to a deterioration of general health. As the cough increases, chest pains result from pleurisy, and fever leads to drenching night sweats. The TB bacteria spread slowly throughout lungs, causing the formation of hard nodules (tubercles) or other masses that break down the respiratory tissues and form cavities within the lungs. Blood vessels can also be damaged, causing the infected person to cough blood (*hemoptysis*). Tubercular lesions can spread extensively in the lung, causing large areas of destruction, cavities, and scarring. The amount of lung tissue available for the exchange of gases in respiration decreases and if untreated the sufferer dies from respiratory failure, toxaemia or exhaustion.

Non-respiratory forms of tuberculosis can variously affect the lymph nodes, especially of the neck (this was known as *scrofula*), the intestines or the lymph nodes of the abdomen (*Tabes mesenterica, tubercular enteritis*), the membranes of the brain and spinal cord (*tubercular meningitis,* sometimes registered as *hydrocephalus*), the skin (*lupus*) or the kidneys (*tubercular nephritis*) (see Appendix 4).

No disease was more feared in nineteenth-century Australia than tuberculosis. Leaving aside gastrointestinal infection—that grim reaper which quietly decimated the voiceless young—tuberculosis was widely perceived as

> '...the most deadly disease that the world knows in the present generation. In most countries of the habitable globe and in all the Australasian Colonies it stands at the head of the list of the causes of death' (Mullins, 1898).

In late nineteenth-century Victoria, phthisis stood at the head of the cause of death list during non-epidemic years (*Victorian Year-Book, 1895-8*, p.731). Tasmanian health officer and public health reformer Gregory Sprott, who was himself a sufferer (Smith, 2011, p.124), concluded that

> 'Tuberculosis is one of the most widespread diseases we have to deal with, and causes more suffering to humanity than any other known disease at the present time. Its ravages are only equalled by those of smallpox in the last century' (Sprott, 1896).

Tuberculosis began its pre-eminence by claiming the first European to be buried on the Australian mainland, Seaman Forby Sutherland, who crewed Cook's ship *Endeavour*. It was present throughout the first decades of the new colony, and first drew official notice in the early 1820s, followed by more detailed descriptions from ex-naval surgeon and author Peter Cunningham (1827), and physician and naturalist George Bennett (1834). Deputy inspector of military hospitals Donald McLeod had noted in 1829 that,

> 'Consumption of the Lungs (Phthisis Pulmonalis) is much more frequent than from the mildness of the Climate might be expected, and more in advanced Life suffer from this disease than in England. It is remarked that in people who arrive in this Colony labouring under this Complaint, it runs a much more rapid course than it is observed to do in Colder Climates' (HRA Series I, Vol. 15, p.375).

Other commentators (Thomas, Reeves, Hall) also drew attention to the high prevalence of respiratory tuberculosis during early colonial history (Cumpston, 1989, p.276).

Useful statistics on tuberculosis deaths in Australia, derived from the Registrars' abstracts of mortality, date from the 1860s. Earlier data are

unreliable because of the nosological confusion that existed between tuberculosis and other respiratory diseases. Hayter wrote in 1877,

> 'It is questionable whether the returns for 1861, or any year prior to it, are reliable as regards the mortality from phthisis, for, until 1862, phthisis was placed in the group with pneumonia, bronchitis, and other diseases of the respiratory system, and there is reason to believe that many doubtful cases of lung disease were classified as phthisis. From 1862 phthisis has been classed as a constitutional disease, and great care has been exercised to tabulate as such no cases except those actually returned as of 'Phthisis' or some term equivalent to it' (*Victorian Year-Book, 1876–7*, in Cumpston, 1989, p.277).

From 1862, the course of respiratory tuberculosis mortality in the larger colonies (New South Wales, Victoria, Queensland), and in Australia as a whole exhibited two distinct phases; rates rose steadily until the mid-1880s and fell rapidly thereafter (Figures 6.16 and 6.17). New South Wales rates peaked in 1885, Victoria in 1887, and Queensland in 1884. The South Australian rise dates somewhat later, from 1872, and continues until 1888. In Western Australia and Tasmania the course of the disease was different, although Tasmania also experienced a decline from the mid-1880s. The mortality rate from respiratory tuberculosis peaked in Australia in 1884 at 133 deaths per 100,000 population, falling thereafter so that by 1906 it had almost halved to be 72.

Non-respiratory tuberculosis followed a similar course, although the rise before the mid-1880s was not as pronounced in some colonies as for respiratory TB. The similarity in patterns, however, argues that the rise in respiratory tuberculosis was genuine, and was not a transfer of deaths from other respiratory causes.

Who died from tuberculosis in Australia? Many people did, and often in the prime of their lives. From 1880, male death rates from respiratory tuberculosis were consistently higher than female rates, averaging almost one quarter higher (23%) in the years through to 1906 (Figure 6.17). Mortality rates from other forms of tuberculosis were also higher among males.

Deaths from respiratory tuberculosis averaged around seven per cent of the total mortality burden in nineteenth-century Australia, rising to eight per cent

throughout the 1880s. Other forms of tuberculosis added another three per cent. Tuberculosis in all its forms was consistently responsible for ten per cent of total Australian mortality from year-to-year, and exceeded 11 per cent in 1887–1888.

Age groups

Deaths from tuberculosis varied substantially across age groups. It was most fatal in young adulthood and took a heavy toll at ages 15 to 39 years (Table 7.2). The disease was responsible for one-in-five deaths of young people aged 15–19 years, rising to almost one-in-three at age 25–29. Thereafter its severity fell away, although consumption still claimed one-in-ten deaths at age 50–54. Fewer than one-in-100 persons aged 75 and over died as a result of respiratory TB.

Table 7.2: Respiratory tuberculosis deaths as a proportion of total deaths, by age group, 1870–74 to 1900–04 (per cent)

Age group	1870–74	1880–84	1890–94	1900–04
0	0.2	0.5	0.2	0.2
1-4	0.6	1.0	0.6	0.8
5-9	1.5	2.6	1.7	1.9
10-14	4.9	7.1	5.5	5.2
15-19	21.6	21.3	19.2	17.9
20-24	29.1	28.1	30.1	24.6
25-29	29.5	29.3	31.3	26.8
30-34	24.2	25.8	27.3	25.4
35-39	20.4	21.0	22.6	22.1
40-44	16.9	17.8	18.4	17.9
45-49	14.0	15.0	15.0	14.1
50-54	10.0	12.4	11.6	10.8
55-59	8.8	9.4	9.4	8.7
60-64	5.8	5.0	7.1	6.3
65-69	3.7	3.1	4.2	4.4
70-74	1.9	1.5	1.8	2.3
75 and over	0.5	0.5	0.5	0.7
All ages	**7.7**	**8.3**	**8.1**	**7.7**

Age-specific death rates were lowest at five years of age and rose quickly until age 35–39 (Figure 6.18). In the late 1870s rates typically fell rapidly after age 40, whereas in the early 1900s these declined until age 50, rising again until age 65, and then declining in old age.

Birth cohorts

An examination of age-specific tuberculosis mortality data over time reveals so-called generational, or cohort effects. The peak in pulmonary tuberculosis mortality, which occurred in the 25–44 age group in Australia in 1865–74, moved to older ages in subsequent years, so that in 1895–04 the peak occurred in the 55–74 age group (Table 7.3). Tuberculosis mortality appears to age when examining cross-sectional (or 'period') data.

Table 7.3: Age-specific death rates from respiratory tuberculosis, Australia, 1865–74 to 1895–04 (per 100,000 population)

Age group	1865-74	1875-84	1885-94	1895-04
0	45	61	35	18
1-4	13	17	9	8
5-9	9	8	7	5
10-14	17	18	13	12
15-19	82	95	79	70
20-24	166	183	171	135
25-29	209	216	220	176
30-34	212	211	216	193
35-39	221	224	200	204
40-44	216	230	196	191
45-49	211	229	204	184
50-54	195	229	216	180
55-59	207	225	216	212
60-64	164	193	219	230
65-69	191	155	193	224
70-74	152	100	110	181
75 and over	74	81	76	105
All ages	**116**	**123**	**118**	**110**

Andvord (1930) and Frost (1939) were the first to surmise that changes in age-specific tuberculosis mortality demonstrated 'physiological changes in resistance with age'. The inference they drew from cohort patterns of TB mortality was that early life conditions can have long-lasting impacts and can predict mortality in later life. Improved survival among successive birth cohorts was interpreted as evidence of progressive improvements in early life conditions.

Lancaster used Australian data after 1907 to come to a similar conclusion,

'The generation method reveals that the so-called postponement of death to the later years is an artefact, and that high rates in later life represent the residual effect of high infection rate in the past as evidenced by a high mortality rate of the same cohorts at the earlier ages' (Lancaster, 1950b).

Figure 7.12: Age-specific tuberculosis mortality rates by period, Australia
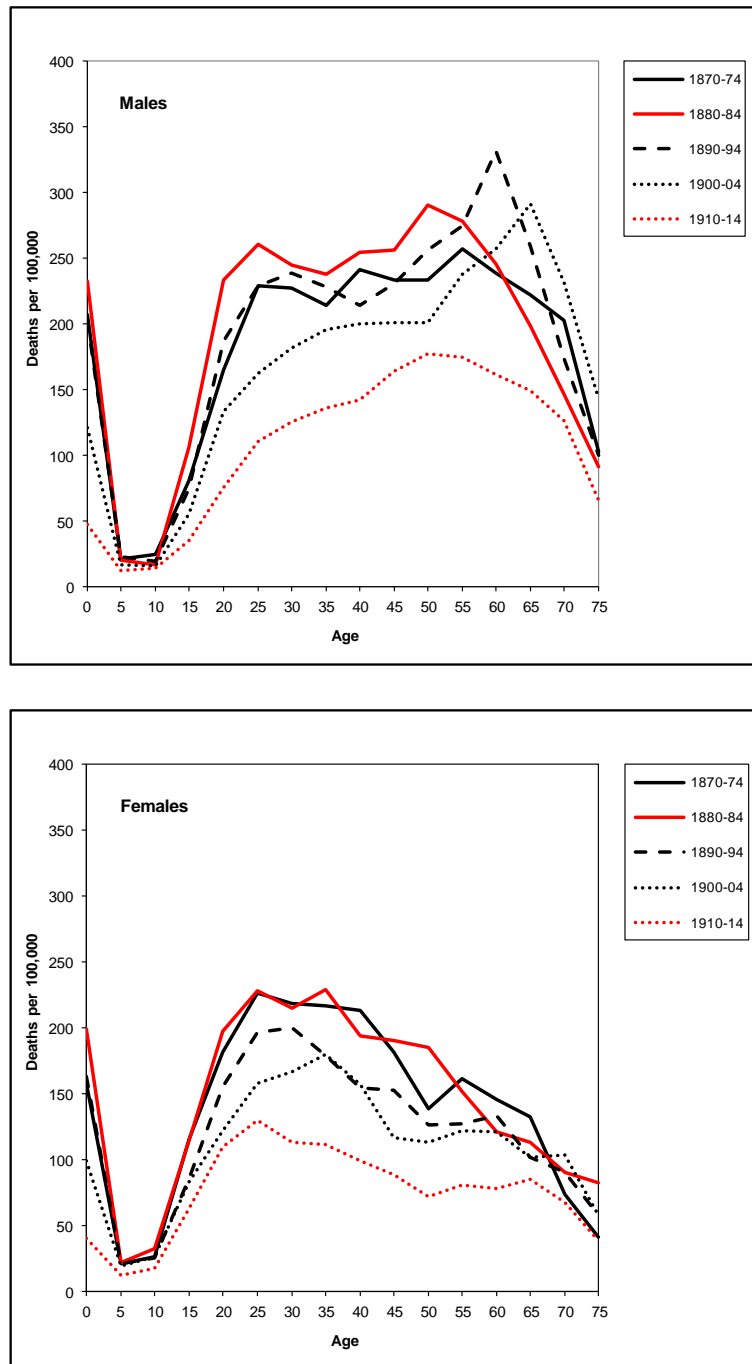
The availability of earlier data provides additional evidence on the nature of the decline in tuberculosis mortality. Figure 7.12 shows the age distribution of deaths from all forms of tuberculosis separately for males and females. In males, an initially high death rate among infants fell away during childhood, only to rise rapidly from ages 10–14 to early adulthood. From age 25-29, mortality continued to increase, although more slowly. Mortality peaked in late adulthood, before falling away among older age groups.

Figure 7.13: Age-specific tuberculosis mortality rates by birth cohort, Australia
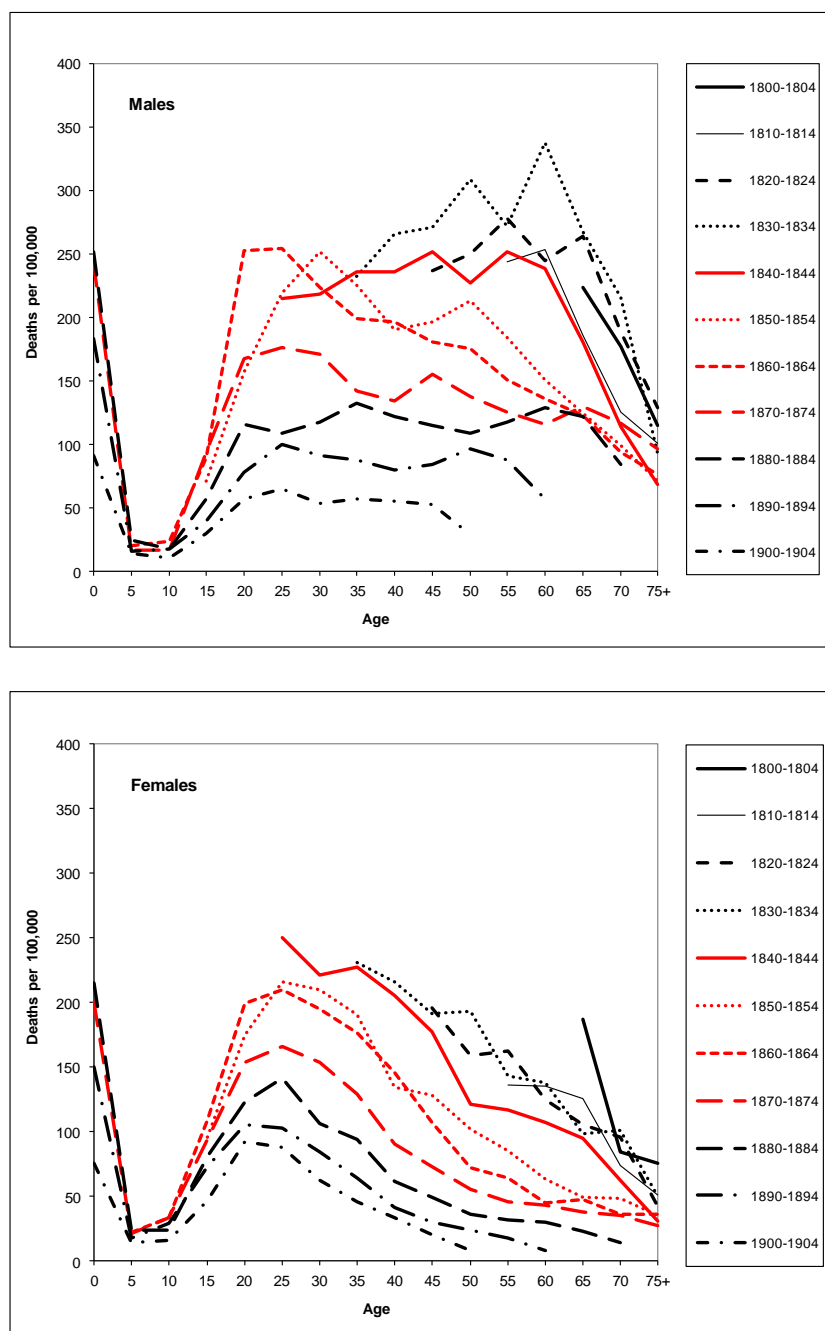
The 1880–84, 1890–94 and 1900–04 curves exhibit successive mortality declines in most age groups, except for older males. Here, there is some indication of a shift in the age distribution to older ages for males over the late-nineteenth and early-twentieth century.

The course for females is different, with mortality peaking much earlier — in the 25–34 years age-group — and then declining steadily to old age. There is no evidence of a shift in mortality to later ages.

Successive birth cohorts are shown in Figure 7.13. Tuberculosis mortality tended to be higher among earlier birth cohorts, suggesting that they may have been at greater risk of contracting the disease. Mortality tends to peak in early adulthood, although some male birth cohorts show rates that plateau in middle age.

Davenport (2013) has reanalysed historical tuberculosis data from a number of countries, concluding that the onset of declines in mortality occurred simultaneously in many age groups, and not in the youngest age groups first, as would be required by an early-life conditions hypothesis. The pattern of decline more closely resembled period, and not cohort-dependent effects.

A closer examination of Australian age-specific trends — aided by a turning point analysis — supports Davenport's findings. A marked decline in respiratory tuberculosis began concurrently in most age groups, and dates from the mid-1880s (Figure 7.14).

The Australian data also confirm differences in patterns of mortality between males and females, thus requiring that the underlying causal mechanisms be sex-specific. This also argues against an early-life conditions hypothesis. The factors that drove reductions in mortality did so at all ages, although they affected males and females differently. Davenport suggests that additional influences — such as smoking — impeded the decline in tuberculosis in older adult males, producing sex-specific shifts in age distributions of mortality that were previously interpreted as cohort-dependent declines.

Figure 7.14: Age-specific respiratory tuberculosis mortality rates, 1870–1906, Australia, with turning-point analysis
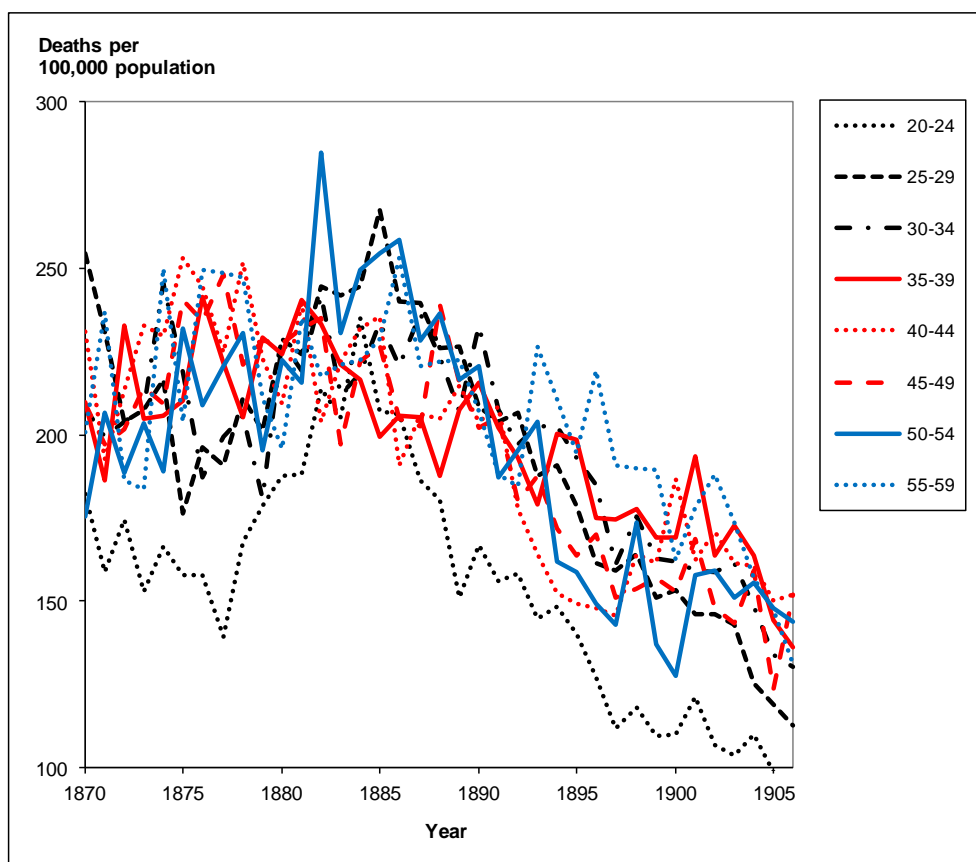
Age group	Phase 1	Annual per cent change	Phase 2	Annual per cent change
15-19	1870-1882	1.24	1882-1906	-2.86*
20-24	1870-1884	2.10*	1884-1906	-3.50*
25-29	1870-1887	0.92*	1887-1906	-3.63*
30-34	1870-1888	0.67*	1888-1906	-2.79*
35-39	1870-1881	1.10	1881-1906	-1.58*
40-44	1870-1875	3.40	1875-1906	-1.64*
45-49	1870-1880	1.68*	1880-1906	-2.11*
50-54	1870-1884	1.97*	1884-1906	-2.86*
55-59	1870-1886	0.69	1886-1906	-2.07*

* The annual per cent change is significantly different from zero at alpha = 0.05.

Urban and rural mortality

Since people who had poor general health and who lived close to TB sufferers for long periods were at greater risk of contracting the disease, TB mortality rates were higher in urban areas. Consumptives seeking treatment also gravitated to urban areas, where health and welfare services were more widely

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available. Victorian statistician Hayter calculated rates for metropolitan and non-metropolitan regions, showing the disparity (*AMG*, Jan. 1899, p.7).

Phthisis—and other tubercular—mortality fell rapidly in Melbourne and suburbs after 1885, remaining relatively unchanged in extra-metropolitan districts (Figure 7.15). Preventative measures undertaken in Sydney City were also associated with falling death rates, whereas suburban rates remained unchanged (*AMG*, 20 Aug 1904, p.429).

Figure 7.15: Crude phthisis mortality rates for Victorian metropolitan and non-metropolitan regions
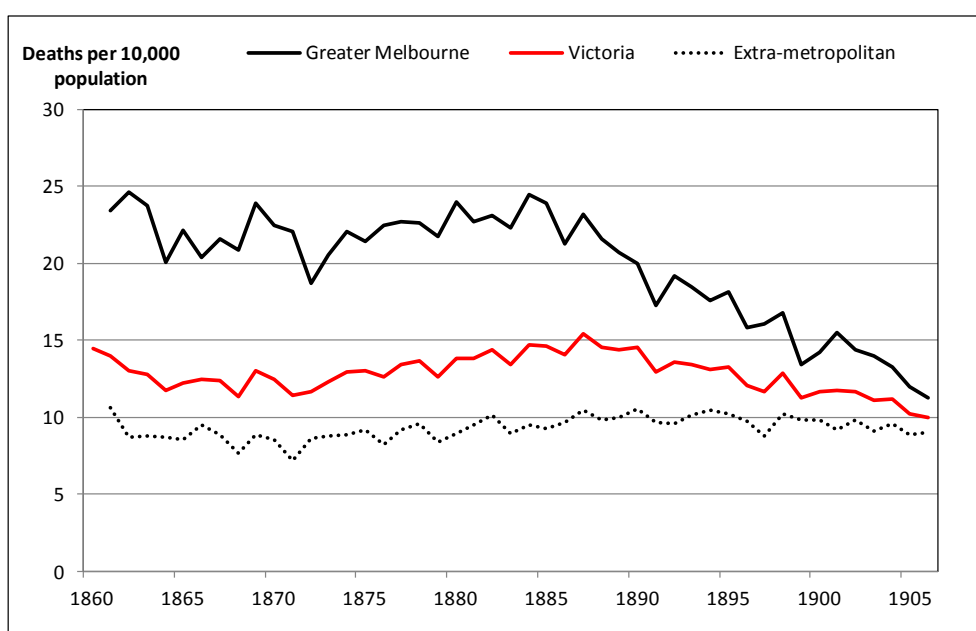
Population groups

Respiratory tuberculosis mortality was demonstrably higher among certain population groups, such as South Sea Islanders living in Queensland. Kanaka labourers suffered TB mortality rates in excess of twenty times those of other persons in the 1870s and 1880s, having previously been free from infection. Nearly half of all phthisis deaths in Queensland in 1884 and 1885 were among 'Polynesians'. It was W. T. Blakeney, the Queensland registrar's view that

> 'It is not apparent…that they are as a rule in any degree affected at the time of their
> arrival, but they would appear, from their habits and constitution, to be peculiarly

liable to contract all forms of tubercular disease, and there is no doubt that when once attacked they offer but feeble resistance to the disease, and very quickly succumb to it' (*QldRG*, 1896, p.xxxvi).

Cumpston links the increase to co-morbidity with other respiratory infections which, in the years prior to 1876, had been high (1989, p.289).

In the ten years to 1890, 16 per cent of deaths of Chinese persons in Victoria were attributed to phthisis. he Victorian statistician, also commented that 'It is stated on good authority that phthisis is the chief cause of the death of the Australian Aborigines, who, when once affected, very seldom recover' (*Victorian Year-Book, 1895-8*, p.734).

Natural immunity

The question of natural immunity to tuberculosis among native-born Australians preoccupied medical men throughout the nineteenth-century. The benefits of a long sea voyage, residence in a warm, dry climate and wholesome food were together promoted as a curative for British consumptives (Kilgour, 1855; Smith, 2011, p.119–121). Studies were made as to the 'elevation at which immunity from phthisis may be secured in every degree of latitude', since it was observed that deaths

> 'from chronic diseases, and especially consumption, are rare…the impression that a larger proportion of those who go to the colonies with chest disease do well than of those who remain, is strong in my mind as in that of most authorities on the subject' (Symes Thompson, 1873).

But medical men also began to suspect that consumptive emigrants were inflating local mortality rates (Singleton, 1871; Girdlestone *et al.*, 1877). The redoubtable William Thomson published widely on the subject, examining Melbourne Hospital patient records and the registrar's mortality records to conclude that there was no real difference, and that the Australian-born were just as vulnerable to death from tuberculosis. His conclusions attracted only vilification from the medical establishment (e.g. *AMJ*, Nov. 1870, pp.344–349).

Thomson found that of the 1,088 persons who died from phthisis in 1877, the vast majority (96%) had contracted the disease in Australasia, these persons

having been resident for more than one year, with the remainder having become tuberculous elsewhere (Thomson, 1879, p.41). The rise in mortality from TB was not prompted by the influx of consumptives from abroad, and neither did Australia provide any natural immunity (Cumpston, 1989, p.280).

Sydney Officer of Health W.G. Armstrong also conducted investigations into the circumstances surrounding deaths from phthisis. He stated that

> 'The information gathered under the head of locality in which the disease was contracted indicated that an immense preponderance of the fatal cases of consumption in Sydney contract their illness here, and that the cases of imported phthisis bear a very small proportion of the total' (*AMG*, 20 Aug. 1904, p.428).

As late as 1903, however, the effect of the arrival of consumptives from overseas was still being discussed (Gresswell, 1904, p.233).

Germ theory and public health

In 1882, German physician and scientist Robert Koch presented his discovery that *Mycobacterium tuberculosis* was the bacterium that caused tuberculosis, laying the foundation for the triumph of germ theory over miasma and climate theories. In the years that followed, science progressed knowledge on the cause, effects and treatment of TB (e.g. Crivelli, 1891; Coxwell, 1893; Mullins, 1898), leading to a better informed medical community and public as to the infective nature of the disease. Statisticians contributed by refining methods to describe the epidemiology of the disease (Knibbs, 1908; 1912, pp.230–233). The association between the increase in knowledge about the disease, active sanitary reform and public health measures, and the decline in tuberculosis mortality beginning in 1884 is noteworthy.

Following the demonstration of the infective nature of the disease, the improvement of hygiene was perhaps the first effect of medical intervention on the disease (Walker, 1983). The disease was now thought to be preventable, and perhaps even curable (Jamieson, 1898; Gresswell, 1899). In Victoria, Dr D. A. Gresswell's *Tuberculosis and its Prevention* went through numerous editions between 1893 and 1902, playing a major role in education on the nature of the disease and the precautions needed to stave off infection. Heightened public

awareness also led to a plethora of new remedies, many based on quackery, but some based on science including Koch's unsuccessful *tuberculin* (Smith, 2011, p.125–130).

Public health legislation was key; compulsory notification of cases began in South Australia in 1898 (Turner, 1904), along with prohibitions against spitting and other forms of expectoration, compulsory examination and isolation, disinfection of premises, the establishment of dispensaries and sanatoria, welfare payments for sufferers and their families, and the establishment of state tuberculosis authorities (*AMG*, Nov. 1901–Jul 1902).

Progress in Sydney in 1901 was reported by the Medical Officer, W. G. Armstong,

> "The local authority of the city of Sydney has made the disinfection of dwellings in which deaths from phthisis have occurred a matter of routine. A by-law passed by the same authority, prohibiting expectoration on the footways, and firmly enforced, has been another step in the right direction. The provisions of a similar by-law should also be applied to trams and other public vehicles plying for hire. Balmain and some other suburban districts have imitated the action of the city in prohibiting spitting on the footpaths" (*AMG*, 20 Sep. 1902, p.484).

LEADING CAUSES OF DEATH

Changes in leading causes of death throughout the second half of the nineteenth century illustrate how mortality changed in its nature in Australia. In 1865 and again two decades later in 1885, the broad grouping of gastrointestinal infections—diarrhoea, dysentery, cholera and enteritis—was the leading cause of death among both males and females, although it was generally only under-fives who were affected (Table 7.4). It was the sheer number of deaths among infants and young children that secured the place of gastrointestinal infections at the head of the list. In 1865, around 14% of all deaths were due to gastrointestinal infections, falling to 11% in 1885, and 8% in 1905. Were deaths from atrophy and debility, convulsions and teething to be included, the proportions would be even greater.

Table 7.4: Leading cause of death among age groups and total population, by sex, 1865, 1885 and 1905

	1865	1885	1905
Males			
0	Gastrointestinal infection	Gastrointestinal infection	Gastrointestinal infection
1-4	Gastrointestinal infection	Gastrointestinal infection	Gastrointestinal infection
5-9	Accidents	Accidents	Accidents
10-14	Accidents	Accidents	Accidents
15-19	Accidents	Respiratory TB	Accidents
20-24	Respiratory TB	Respiratory TB	Respiratory TB
25-29	Accidents	Respiratory TB	Respiratory TB
30-34	Accidents	Respiratory TB	Respiratory TB
35-39	Accidents	Respiratory TB	Respiratory TB
40-44	Accidents	Respiratory TB	Respiratory TB
45-49	Respiratory TB	Circulatory	Circulatory
50-54	Circulatory	Circulatory	Circulatory
55-59	Circulatory	Circulatory	Circulatory
60-64	Circulatory	Circulatory	Circulatory
65-69	Circulatory	Circulatory	Circulatory
70-74	Circulatory	Old age	Circulatory
75+	Old age	Old age	Old age
Total leading cause	**Gastrointestinal infection**	**Gastrointestinal infection**	**Circulatory**
Second leading cause	**Accidents**	**Circulatory**	**Gastrointestinal infection**
Third leading cause	**Circulatory**	**Respiratory TB**	**Respiratory TB**
Females			
0	Gastrointestinal infection	Gastrointestinal infection	Gastrointestinal infection
1-4	Gastrointestinal infection	Gastrointestinal infection	Gastrointestinal infection
5-9	Diphtheria	Diphtheria	Accidents
10-14	Diphtheria	Typhoid	Circulatory
15-19	Respiratory TB	Respiratory TB	Respiratory TB
20-24	Respiratory TB	Respiratory TB	Respiratory TB
25-29	Respiratory TB	Respiratory TB	Respiratory TB
30-34	Respiratory TB	Respiratory TB	Respiratory TB
35-39	Respiratory TB	Respiratory TB	Respiratory TB
40-44	Respiratory TB	Respiratory TB	Respiratory TB
45-49	Circulatory	Circulatory	Circulatory
50-54	Circulatory	Circulatory	Neoplasms
55-59	Circulatory	Circulatory	Circulatory
60-64	Circulatory	Circulatory	Circulatory
65-69	Old age	Circulatory	Circulatory
70-74	Old age	Old age	Circulatory
75+	Old age	Old age	Old age
Total leading cause	**Gastrointestinal infection**	**Gastrointestinal infection**	**Circulatory**
Second leading cause	**Atrophy, debility**	**Respiratory TB**	**Gastrointestinal infection**
Third leading cause	**Convulsions, teething**	**Circulatory**	**Old age**

Among males in 1865, accidents were the second leading cause and circulatory diseases third. By 1885, circulatory disease had risen to be the number two cause of death, with 8.8% of the total population—mostly in middle-aged and older adults—dying as a result. But in 1905, circulatory disease—a chronic disease—had displaced gastrointestinal infection—a communicable disease—as the leading cause of death in Australia, being responsible in that year for 12.3% of all deaths. Gastrointestinal infections were still formidable, however, claiming 10.1% of all deaths, predominantly in young children.

Leading causes of death also changed within certain age groups over time. In children under five they did not, with gastrointestinal infections predominating during the entire forty year period. Among children aged five to 14, an age group with numerically few deaths, accidents predominated over the entire period among boys, with diphtheria and typhoid fever giving way to accidents and circulatory disease among girls.

Between ages 15 to 44, respiratory tuberculosis almost reigned supreme, the only qualifier being that accidents killed many males in earlier years. At ages 45 to 64, circulatory disease was the leading cause of death among both males and females, the exception being neoplasms among females aged 50–54 in 1905. Among older persons aged 65 years and over, the unsatisfactory cause 'old age' was displaced by circulatory disease as the leading cause.

MORTALITY DECLINE AND THE MCKEOWN INTERPRETATION

Thomas McKeown's theory of population growth argued that improvements in living standards served to increase population resistance to infectious disease and death. These improvements largely occurred through agrarian reform, and better nutrition which resulted from a growth in average income and an increased food supply. A central tenet of the theory was that the decline in mortality was largely due to fewer deaths from airborne or water- and food-borne infectious diseases (McKeown, 1976).

McKeown's method of enquiry took the form of a comparison of cause-specific death rates in England and Wales for 1851–60 and 1891–1900. English

demographer Robert Woods has re-examined McKeown's data. Woods disaggregated and expanded the original analysis to include 19 cause-of-death categories, and used this revised list to indicate the contribution of causes of death to the mortality decline between the 1860s and 1890s (Woods, 2000, pp.344–359).

The same approach can be used to examine change in Australia, and to compare its magnitude with England and Wales (Table 7.5). The 28 causes of death which are included in the schema of Table 5.4 have here been set alongside Woods' 19 causes. Although the adaptation has shortcomings, it improves on McKeown's categories with their 'reductionist and *ad hoc* assumptions'. It also disaggregates McKeown's large 'Other causes' residual category, which contained almost 20% of all his 1890s deaths.

There are obvious differences between deaths in England and Wales, and in Australia. Smallpox was largely unknown in Australia, whereas declines in measles and diphtheria had a much greater impact. Rates for deaths attributed to stomach or digestive diseases rose in England and Wales, but improved somewhat in Australia.

Great disparity surrounds the role of diarrhoea and associated intestinal infections in infant mortality. In Australia, declines in these causes may have been responsible for more than half of the reduction, depending on the contribution of the vague causes *atrophy*, *debility*, *convulsions* and *teething*. In England and Wales, diarrhoea, if linked with typhus, accounts for around 28% of the improvement, although a still substantial 'Other causes' category has the potential to add to this.

The largest differences between the two countries are found in contribution of tuberculosis. Declines in *phthisis* (respiratory TB) mortality contributed 35% to the improvement in England and Wales. Rises in rates of other forms of TB (*scrofula*), however, offset this somewhat. In Australia, the net fall in TB mortality between 1861–70 and 1891–1900 masks the substantial rise in rates that occurred to the mid-1880s, and the equally substantial fall in the following decade (Figure 6.17). Although TB rates had begun to decline by the 1890s,

much of the improvement was yet to come, and so the combined contribution to improvement between 1861–70 and 1891–1900 appears understated at less than 7%.

Table 7.5: Changes in cause-specific mortality, England & Wales and Australia, 1861–70 to 1891–1900

England and Wales				Australia			
	1861-70	1891-1900	Contribu -tion to change		1861-1870	1891-1900	Contribu -tion to change
	Standard deaths	Deaths	Per cent		Deaths per 100k	Deaths per 100k	Per cent
Smallpox	46,713	4,058	4.27				
Measles	119,471	126,841	-0.74	4. Measles	29	13	5.50
Scarlet Fever	272,437	48,290	22.46	7. Scarlet fever	51	4	16.15
Diphtheria	52,319	80,671	-2.84	3. Diphtheria	52	19	11.34
Whooping cough	140,748	115,670	2.51	8. Whooping cough	23	16	2.41
Diarrhoea	294,643	226,143	6.86	9. Diarrhoea, dysentery, cholera, enteritis	185	134	17.53
Typhus	268,467	55,996	21.29	2. Typhoid fever	49	29	6.87
Diarrhoea & Typhus	563,110	282,139	28.16				
Cancer	119,413	232,178	-11.30	12. Neoplasms	27	49	-7.56
Scrofula	121,864	189,782	-6.81	6. Other tuberculosis	36	32	1.37
Phthisis	777,350	426,224	35.19	5. Respiratory tuberculosis	111	95	5.50
Brain	795,075	665,301	13.01	14. Nervous system	69	40	9.97
Heart	414,686	507,730	-9.32	15. Circulatory	121	123	-0.69
Lung	971,696	1,044,719	-7.32	16. All respiratory	170	180	-3.44
Stomach	296,120	365,484	-6.95	19. Digestive	69	61	2.75
Kidneys	91,828	141,202	-4.95	20. Genitourinary	21	42	-7.22
Generative	18,635	14,094	0.46				
Childbirth	52,833	46,591	0.63	21. Pregnancy, childbirth	16	15	0.34
Violence	283,484	202,363	8.13	23. Injury, poisoning, external causes	125	92	11.34
Other Causes	1,435,513	1,082,098	35.42	11. Atrophy, debility	114	62	17.87
				10. Convulsions, teething	104	49	18.90
				13. Endocrine, nutritional, metabolic diseases	13	14	-0.34
				22. Perinatal, congenital	27	55	-9.62
				26. All ill-defined, unspecified causes	96	56	13.75
				28. All other causes	31	68	-12.71
Total	**6,573,295**	**5,575,435**	**100.00**		**1,539**	**1,248**	**100.00**

Source: Woods, 2000, pp.350–351 for England and Wales.

McKeown's nutrition interpretation is unsatisfactory in explaining the improvement of mortality in Australia. Food supplies may have been monotonous, but they were seen to be adequate or even abundant, especially in the supply of meat, tea and sugar. Vegetables may have been an exception. Depending on the location, these were cheap, although often in short supply, and overall consumption was thought to be low (McCalman, 1988, p.57; Smith, 2011, pp.230–234).

Although poverty was rife in inner-city working class suburbs (Mayne, 1982; Fitzgerald, 1987; McCalman, 1984), widespread malnutrition was not. Intense effort might have been needed to feed working class families, but 'the vast majority of families managed to fill children's tummies somehow' (McCalman, 1984, p.57).

Broadly, food was also affordable. Globally, Australians experienced high per capita incomes during the late nineteenth century (Butlin, 1964), and by 1890–91 the annual average cost of food in relation to wages in New South Wales was perhaps the second lowest in the world after the United States, according to statistician Coghlan.

LATE NINETEENTH-CENTURY EPIDEMIOLOGICAL TRANSITION

How might epidemiological transition in Australia be described? With few exceptions, analyses to date have been qualitative, and have generalised the English transition to explain the Australian occurrence, assuming that the transition in this country also adhered to a classical or western model. No attempts have been made to quantify the contribution of individual diseases to the decline in mortality. Quantification is necessary, if only to identify the commencement of Omran's third stage, when the proportion of deaths from infectious diseases began to decline and that of chronic diseases rise. The disease profiles developed in Chapter 6 enable a quantitative description.

Some caution is needed, however. The scope of the population described should be recalled—persons whose deaths were registered—along with the varied quality of data. The transitions among white Europeans in Australia can

be described reasonably well, but much less can be said about Indigenous people (McCalman *et al.*, 2009). Neither should epidemiological transition be understood as a uniform process. The Australian transition was the product of six disparate colonies, with varied disease histories, ethnicity, social conditions and age structures, and with a mix of climates, health services, and urban-rural settings.

To assist in examining the epidemiological transition, this study uses a broad cause classification scheme, similar that introduced in the 1990 Global Burden of Disease (GBD) study (Murray & Lopez, 1996). This scheme is based on aetiology and health transition theory, with the main purpose of distinguishing between communicable and non-communicable diseases.

The GBD study classifies disease and injury, causes of death and burden of disease into three broad cause groups: Group I – Communicable, maternal, perinatal and nutritional conditions, Group II – Non-communicable diseases, and Group III – Injuries. Group I diseases include (but are not limited to) 'pandemic' diseases, as understood by Omran (Mackenbach, 1994). Similarly, Group II diseases include (but are not limited to) Omran's 'degenerative and man-made diseases'.

The twenty-eight causes of death outlined in Table 5.4 have been reassigned to these three broad cause groups (Table 7.6). Influenza and pneumonia do not appear as respiratory diseases, since most if not all deaths ascribed to these causes in the Victorian period were infectious (Mooney, 2007). The correspondence between the GBD classification and the cause-of-death schema developed for this study is not exact, and qualifications have been noted in the table. It is rigorous enough, however, to assess the extent of epidemiological transition.

Table 7.6: Twenty-eight causes of death assigned to the GBD broad cause classification

Group I. Communicable, maternal, perinatal and nutritional conditions	
1. All infectious diseases	(includes causes 2-11)
13. Endocrine, nutritional & metabolic	(although diabetes should be in Group II)
18. Influenza & pneumonia	
21. Pregnancy & childbirth	
22. Perinatal & Congenital anomalies	(although congenital anomalies should be in Group II)
26. All ill-defined & unspecified	(includes cause 27, and is distributed proportionately between Groups I and II)
28. All other causes	(distributed proportionately b/w Groups I and II)
Group II. Non-communicable diseases	
12. Neoplasms	
14. Nervous	
15. Circulatory	
16. All respiratory	(includes cause 17, minus cause 18)
19. Digestive	
20. Genitourinary	
26. All ill-defined & unspecified	(includes cause 27, and is distributed proportionately between Groups I and II)
28. All other causes	(distributed proportionately b/w Groups I and II)
Group III. Injuries	
23. Injury, poisoning, external	(includes causes 24-25)

Sources: Table 5.4, and Murray & Lopez, 1996.

Group I: Communicable, maternal, perinatal and nutritional conditions

The death rate from Group I causes—comprised of communicable, maternal, perinatal and nutritional conditions—was high and variable in Australia until the mid-1880s (Figure 7.16). Between 1864 and 1884, annual mortality averaged 949 deaths per 100,000 population. A turning point occurred in 1885, the same year as for all-cause mortality, after which the Group I mortality rate began to decline steadily. By 1906, the rate had declined to 528 deaths per 100,000 population, falling by almost half in two decades.

Figure 7.16: Death rates from Group I causes, and their proportional contribution, Australia, 1864-1906
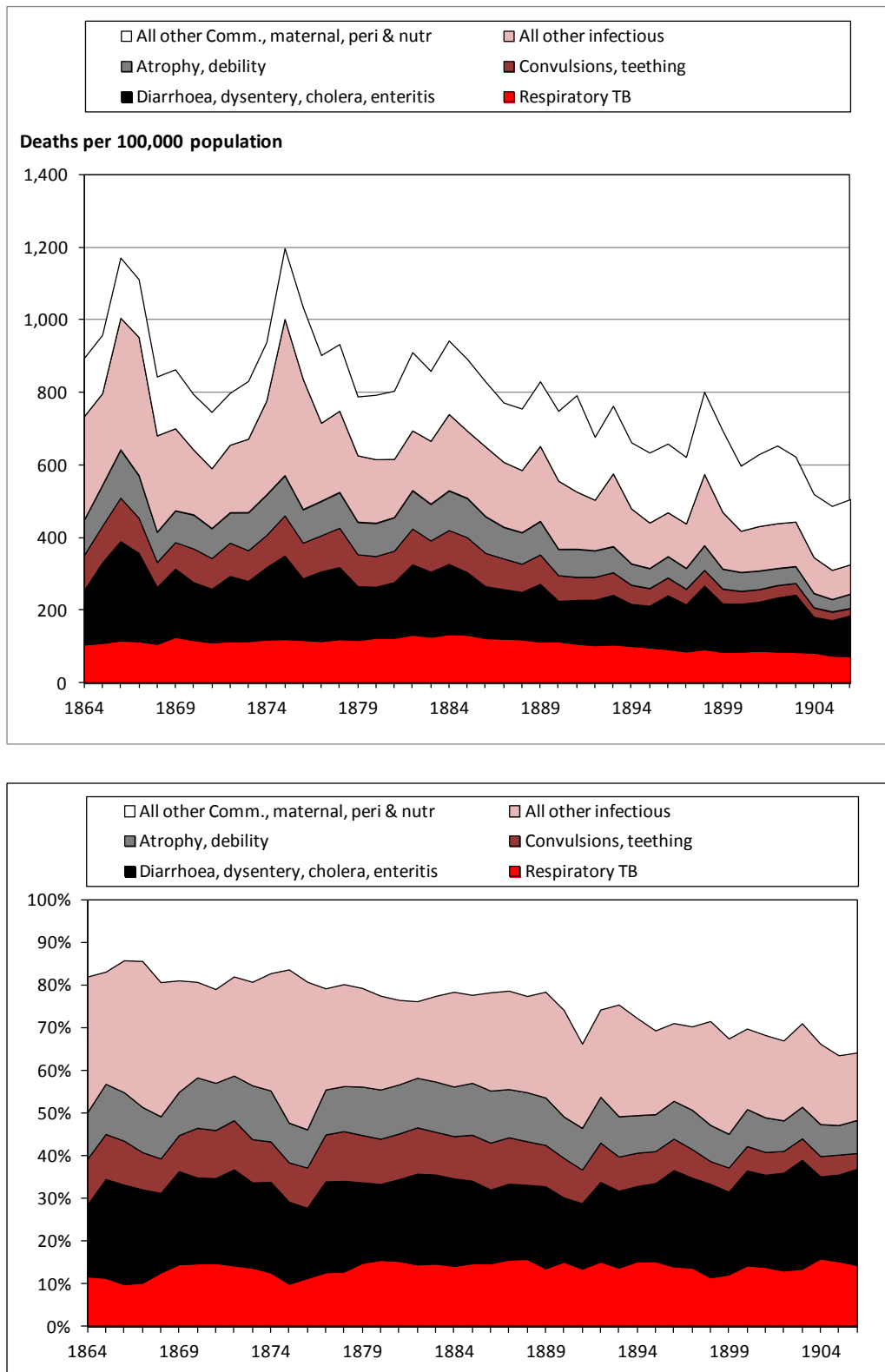
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

Figure 7.16, bottom panel, illustrates the proportional contribution of individual causes of death to Group I mortality. Respiratory tuberculosis remained constant over the period 1864 to 1906 at around 13% of the total Group I mortality burden. Diarrhoea contributed 19% and this also did not change greatly. To diarrhoea should be added most of 'convulsions and teething', which declined from an average annual contribution of 10% before 1885 to 7% after, resulting from greater precision in coding of deaths. 'Atrophy and debility', a related cause, also fell, but mostly after 1900.

The category 'All other infections', comprised largely of typhoid fever, non-respiratory forms of tuberculosis and whooping cough, also declined in its contribution, from an average of 25% in the years to 1885 to 20% in the years after.

The residual category 'All other Communicable, maternal, perinatal and nutritional conditions' grew. Here, the major contributors are influenza and pneumonia along with perinatal and congenital conditions. Influenza epidemics in 1890–91, 1898–99 and 1901–02 swelled mortality in the last decade of the nineteenth century and the first of the twentieth. Congenital and perinatal deaths comprised an increasing proportion of infant deaths over the period (Figure 7.7) but this should almost certainly be attributed to greater precision in determining and coding cause-of-death.

Group II: Non-communicable diseases

The death rate from Group II causes—non-communicable, mostly chronic diseases—increased only slightly between the mid-1860s and mid-1880s, and then commenced a steady decline in a similar fashion to Group I causes (Figure 7.17). Between 1864 and 1884, annual mortality averaged 511 deaths per 100,000 population, slightly more than half the rate of Group I causes. By 1906, mortality had fallen to 375, a fall of one-quarter.

Although it might be expected that death rates from Group II causes would increase, substituting for the substantial declines in Group I rates, this did not occur. Group II mortality also declined, reflecting the improvements in Group I rates. A reasonable conclusion which follows from this is that the same factors

which underpinned improvements in infectious disease rates were also responsible for improvements in chronic disease rates.

However, Figure 7.17 illustrates that not all chronic disease death rates declined. Mortality from neoplasms rose from 6% of the Group II burden in the 1860s to 14% in the 1900s, although this apparent rise reflects better diagnosis and certification. In contrast, mortality from diseases of the nervous system, an ill-defined category, fell away.

Circulatory disease mortality declined slightly, from 138 deaths per 100,000 population before 1885, to 125 after. It is again difficult to separate real changes in underlying mortality from coding and certification improvements which came with a better understanding of the cardiovascular system and its diseases. Despite much circulatory disease remaining undiagnosed, this cause remained the largest contributor to Group II, averaging 28% of the total over the entire period.

Respiratory diseases also declined after 1885, falling from 143 to 45 deaths per 100,000 population in 1906, a fall of two-thirds. Bronchitis, emphysema and asthma were the largest contributors to this disease group, since influenza and pneumonia are included as Group I infectious diseases. Bronchitis, emphysema and asthma mortality rates fell rapidly in Australia after the 1891 Russian flu pandemic (Figure 6.33). Whereas respiratory diseases comprised 27% of Group II deaths in the early 1880s, by 1906 this had fallen to 12%.

Rates of digestive disease fell over the period, from 73 deaths per 100,000 population in the 1860s, to 52 in the early 1900s. Despite falling rates, their contribution to Group II remained much the same, at 14%. Rates of genitourinary disease, however, increased from 22 in the 1860s to 48 in the 1900s. Accordingly, their contribution to Group II rose, from 5% to 12%. The residual category of 'Other non-communicable diseases' averaged 8% in its contribution to Group II.

Figure 7.17: Death rates from Group II causes, and their proportional contribution, Australia, 1864-1906
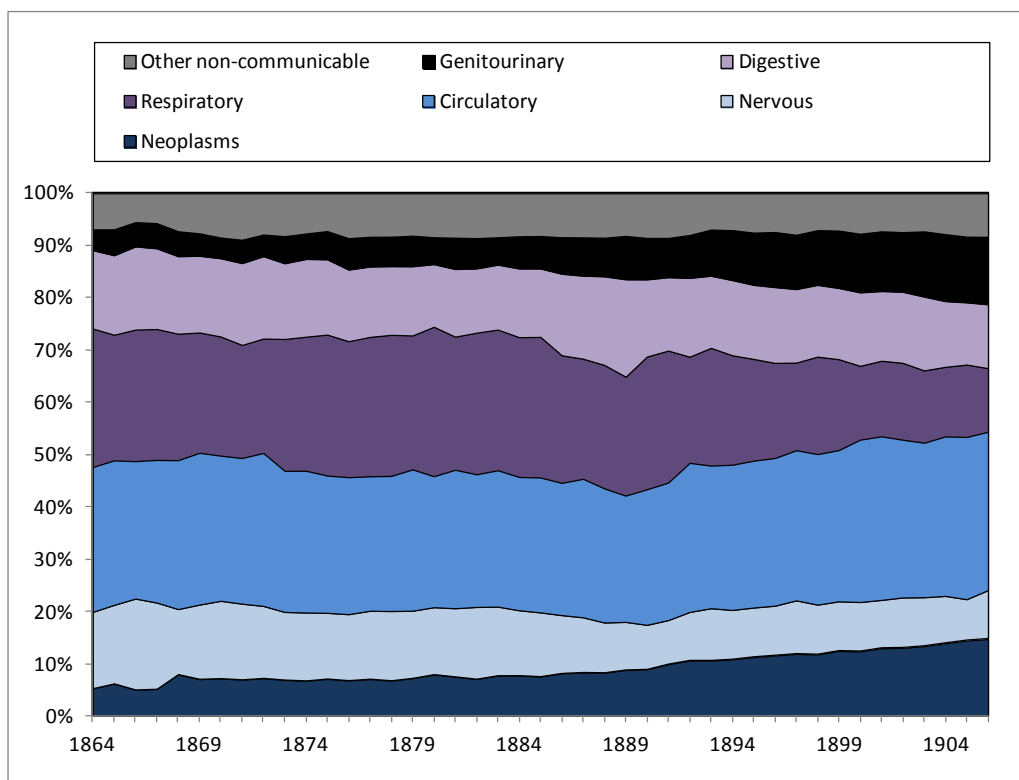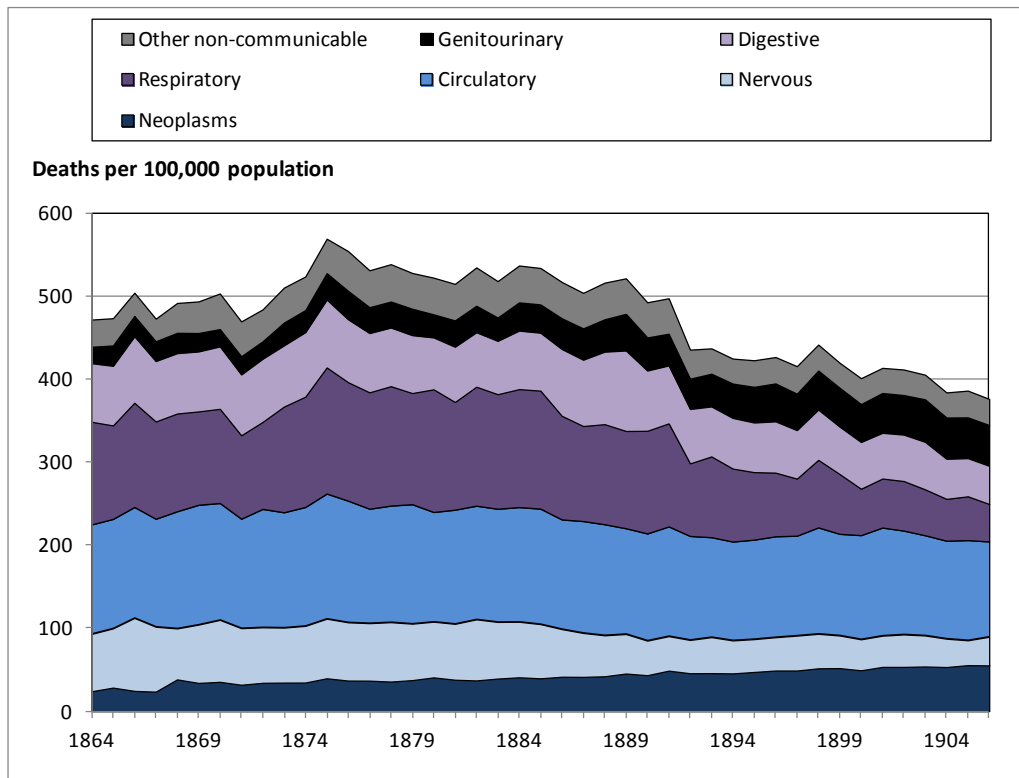
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

The burden of disease

The contribution of each broad cause of death group—Group I: Communicable, maternal, perinatal and nutritional conditions; Group II: Non-communicable diseases and Group III: Injuries—to nineteenth-century Australian mortality is shown in Figure 7.18. Together, the changes in proportions of deaths in each of these groups illustrate to what extent epidemiological transition occurred in Australia during the period.

White settlement in Australia did not undergo Omran's first Age of Pestilence and Famine. Since colonisation did not commence until late in the eighteenth century, Australia largely avoided Europe's Malthusian 'positive checks' of epidemics, famines and wars. Although there may have been high and fluctuating mortality throughout the first half of the nineteenth century (Taylor, Lewis & Powles, 1998a), widespread hunger was absent—except, perhaps, for a short time immediately before and after the arrival of the Second Fleet in 1789. War in Australia was unknown. As far as can be ascertained, life expectancy at birth in Australia never fell below 40 years, this value being Omran's threshold between the first and second stages of the epidemiological transition.

The all-cause death rate declined slowly between the mid-1850s and mid-1880s, at an average annual rate of 0.5% (Figure 4.10). In the 1860s, mortality in Australia was at a level which limited life expectancy at birth to 45 years for men and 49 for women (Table 4.1). These are values which typify the Age of Receding Pandemics. Little gain took place over the following two decades, and by the mid-1880s, male life expectancy at birth had stalled at 47 years, and females at 51.

As Group I mortality, previously high and variable, began to fall, this changed. Between 1885 and 1906, the all-cause death rate declined at an average annual rate of 1.7%. Life expectancy at birth increased by four years in a decade for both men and women, to reach 51 and 55 years respectively in the mid-1890s, and another three years in the following decade, to reach 54 and 58 years in the mid-1900s.

Figure 7.18: Epidemiological transition in Australia, 1864-1906
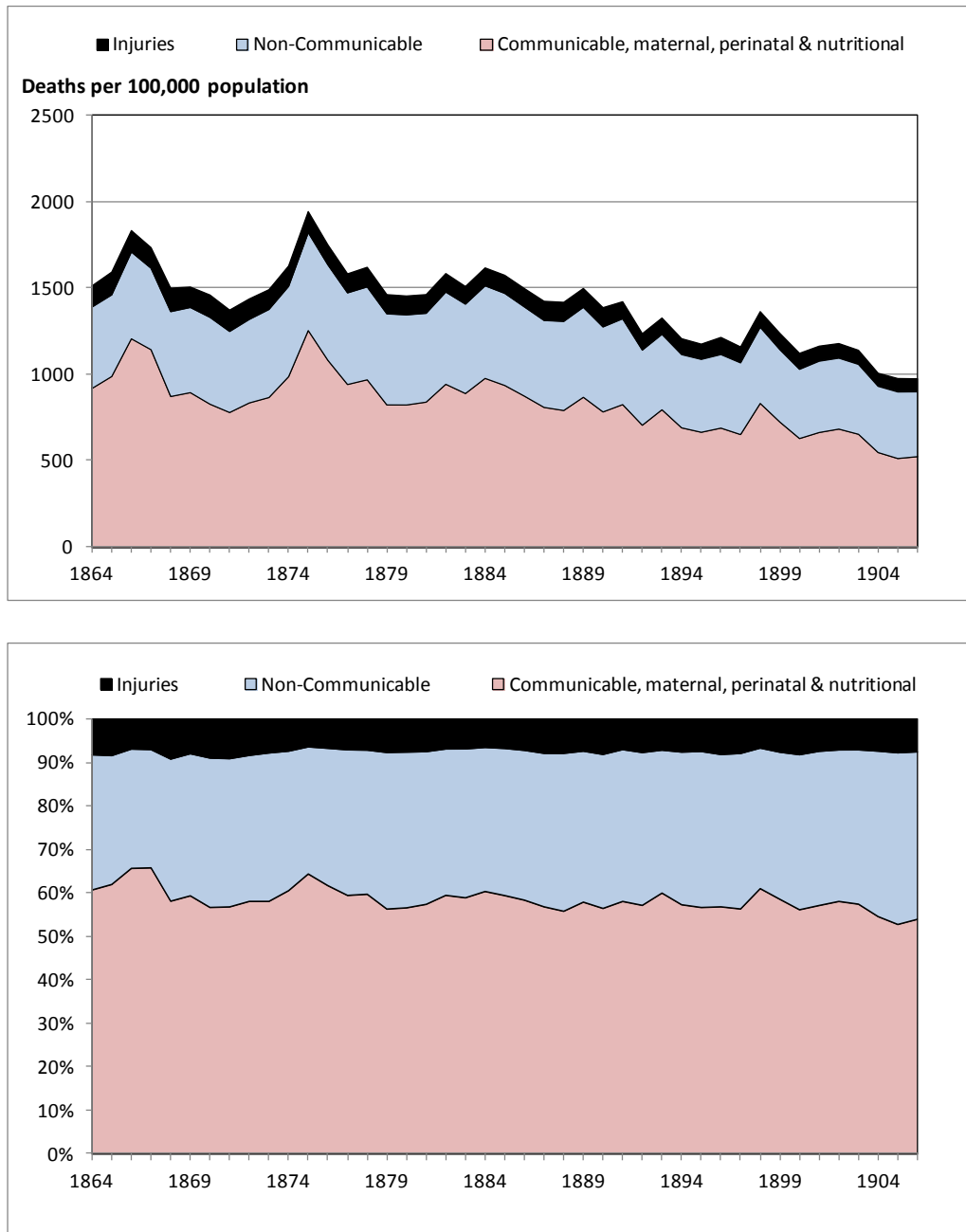
Note: Mortality rates are age-standardised to the 1881 total Australian census population.

Group II and III mortality rates also declined after 1885, in a similar fashion to Group I. Falls in the mortality rates of each Group meant that the proportions of total deaths claimed by each Group underwent very little change over the years 1864–1906, with little departure from a ratio of 60:34:6 between Groups 1, 2 and 3 (Figure 7.18, bottom panel).

For two decades after the mid-1880s turning point, the death rate for the chronic disease group declined alongside that of infectious disease. Only during the last few years of the period of interest—1903 to 1906—does the contribution of Group I diseases decline, and that of Group II increase. This coincided with large falls in infant mortality at this time.

Figure 7.19: Epidemiological transition in Australia, 1864-2007
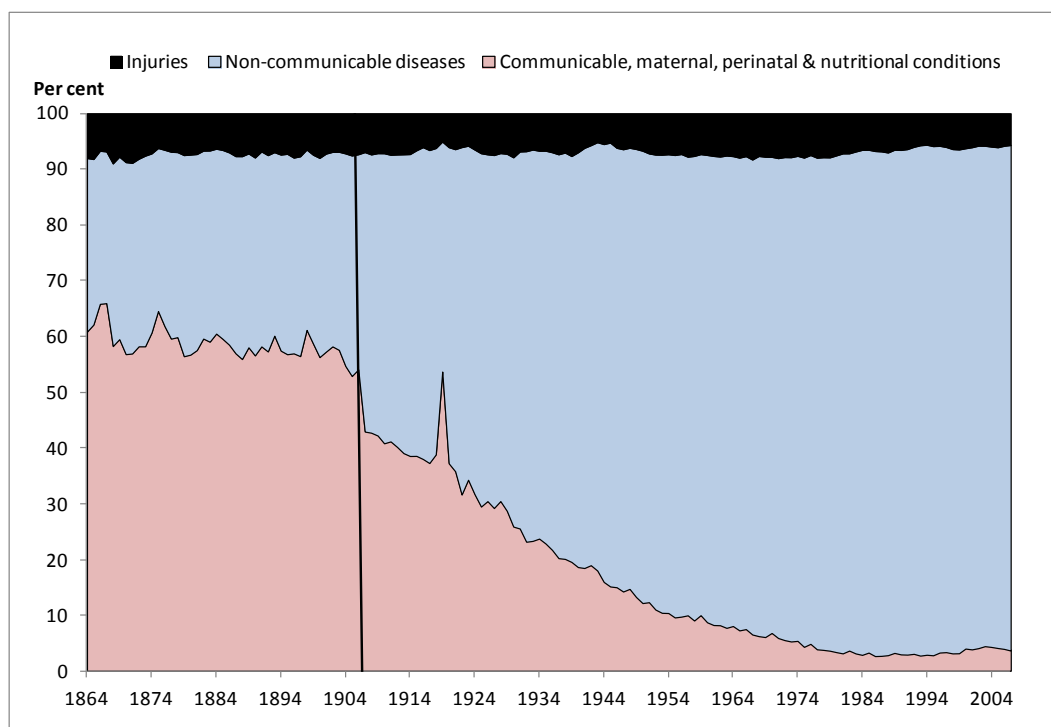
A broader time period shows the fuller impact of the transition. Although hindered by difficulties in uniformly assigning deaths after 1906 to the broad cause schema, Figure 7.19 illustrates that the transition gathered pace only in the first decades of the twentieth century, as infectious diseases receded, and death rates from chronic diseases rose. Mortality rates continued to fall, and life expectancy rose steadily.

Omran had proposed that the epidemiological transition would especially benefit children and young women. The improvements in mortality rates

among under-five children in the latter half of nineteenth-century Australia are striking (see Tables 4.4 and 4.5) and have been discussed at length. Improvements among young women are less obvious.

The female risk of dying is less than for males in post-reproductive years. At lower life expectancy, however, females have a higher probability of death during adolescent and reproductive years (Omran, 1971). In Australia, differences in life expectancy at ages 15–19 to 35–39 did not widen appreciably to favour females over the period (Figure 7.20). Omran, using Coale and Demeny regional life tables, indicated that much of the improvement for young women occurred when life expectancy at birth is still low, below 40 years of age (Figure 7 in Omran, 1971). By the middle of the nineteenth century, female life expectancy at birth in Australia was close to 50 years (Table 4.1).

Figure 7.20: Probability of death (1,000q$_x$) by age and sex at two levels of life expectancy at birth, Australia
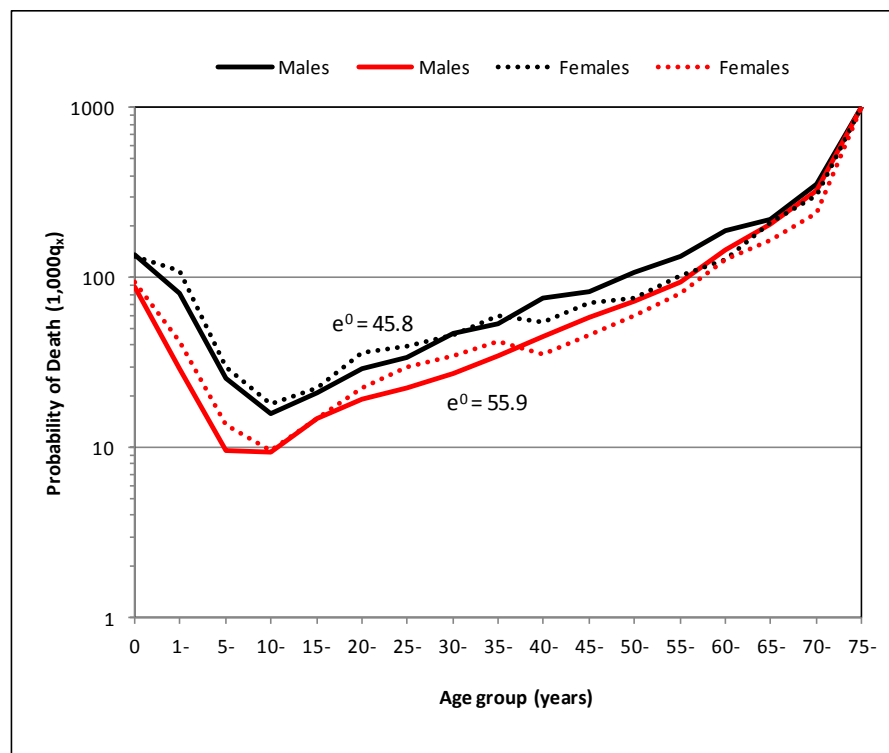
The pattern of the epidemiological transition in Australia adheres well to Omran's classical (or western) model. Despite the absence of a first stage of

Pestilence and Famine, "...a slow and unsteady rate of mortality decline gradually gave way to more precipitous declines around the turn of the twentieth century..." (Omran, 1971). Here, the 'precipitous decline' occurred rather more suddenly, and it also dates somewhat earlier from the mid-1880s. The broad trends in causes of death illustrated in Figure 7.19 are not dissimilar to those in England and Wales (Figure 4 in Omran, 1971), and in the United States (Figure 3 in Omran, 1998). Both of these countries also underwent transitions which are best described as classical.

THE ROLE OF MORTALITY IN DEMOGRAPHIC TRANSITION

Beyond epidemiological transition, trends and patterns in mortality are a central component of *demographic transition*, the historical shift from high to low mortality and fertility which occurs as a country develops from a pre-industrial to an industrialised economic system. A cogent theory of demographic transition was first proposed in 1945 by American demographer Frank Notestein. His theory has also enjoyed longevity, and as for epidemiological transition theory, its force remains (Kirk, 1996).

A recent presentation of demographic transition theory asserts that as societies undergo the transition, they experience a number of roughly sequential demographic processes which are causally related to each other (Dyson, 2010). The transition begins with mortality decline, which initiates, in turn, population growth, fertility decline, urbanisation and population ageing. Mortality transition is itself the cause of subsequent declines in fertility and rises in urbanisation, each of these having effects on economic, social and political development (Dyson, 2010; Canning, 2011).

The order in which the sequences of the demographic transition unfold has been contended. Most versions of the theory hold that mortality decline is the initiating process for the demographic transition. Once mortality begins to decline then fertility declines follow, with an extended period where birth rates are higher than death rates; the result being a period where natural increase –

an excess of births over deaths—occurs (Dyson, 2010). For Omran, mortality decline was also the initiating process,

> "In modern times, it was predominantly the declining mortality more than increasing fertility that caused the West's growth phase in western Europe in the 18th and 19th centuries" (Omran, 1998).

Although demographic transition in Australia adheres broadly to this classic outline, some important qualifications apply. In 1860, Australia experienced both high fertility rates and death rates (Figure 7.21). Married women could expect to bear around seven live births during their lifetime (Quiggin, 1988; McDonald *et al.*, in Vamplew, 1987) and life expectancy at birth had already reached the mid-forties.

The nature of Australia's demographic transition was different from long-established and settled European populations. Australia's young population was augmented throughout the nineteenth century by waves of young immigrants, and often by whole families. As a 'country of immigration', Australia enjoyed favourable economic conditions, including an abundant availability of land, secure supplies of food and generally low population densities, each of which nurtured the young population and assisted child survival.

As shown, the all-cause death rate in Australia was high and variable until the mid-1880s, after which it began a steady decline with consequent increases in life expectancy. Mortality change cannot be seen as a major determinant of demographic transition in the years prior to 1885. The high fertility rate was already in decline from at least the 1860s (Figure 7.21), although it plateaued in the 1880s. The decline in the fertility rate between 1860 and the mid-1880s does not appear to have been triggered by any appreciable decline in mortality, and can therefore be seen as occurring outside the context of the classic model of demographic transition. After the mid-1880s, however, the decline in mortality in Australia was accompanied by a further decline in fertility, and by a fall in natural increase (Figure 7.21).

Figure 7.21: Demographic transition in Australia, 1860 to 2010
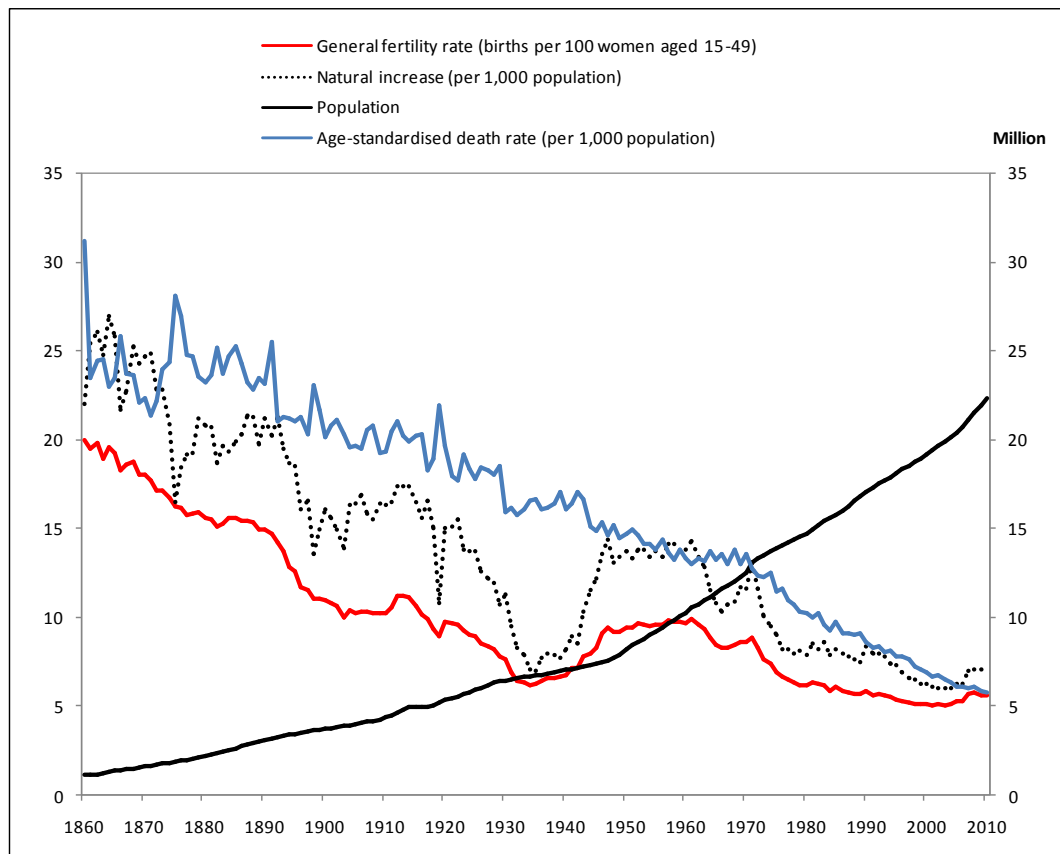
Source: Births: ABS, 2008a.

Family size changed markedly during this period. The cohort of women born in the early 1870s could on average expect to bear four children, which was a large fall from the family sizes of earlier cohorts (Ruzicka & Caldwell, 1977, p. 153). As was the case for mortality decline, the fall in fertility first occurred in urban areas, typically in the upper-middle and middle socioeconomic groups, and among women who were born in the United Kingdom (Ruzicka & Caldwell, 1977, Chapter 3; Borrie, 1994, Chapter 7). Declining fertility during the last decade of the nineteenth century occurred during a time of economic recession, and was perhaps further augmented by the cultural diffusion of family planning ideas and practices.

Dyson (2010, pp.93–95) interprets the fall in fertility which followed the mid-1880s decline in mortality as occurring within the context of the classic model of demographic transition. This pattern was termed by French demographer

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Chesnais a *demi-transition* (1992, p.281), or 'half-transition', since the trends in mortality and fertility before the mid-1880s were considered to be unrelated. Australia's demographic transition in the nineteenth century occurred in a similar fashion to other 'offshoot populations' from north-western Europe, such as Canada, the United States and New Zealand, all of which were young societies greatly affected by large-scale immigration.

Other theorists look elsewhere for the determinants of fertility decline, including in economic and cultural theory, in the role of government and in diffusion of methods of fertility control (Kirk, 1996).

An important alternative to mortality as the prime agent in demographic transition has been proposed by Australian demographer J. C. Caldwell (2006). For Caldwell, the fundamental factor in transition is human capital formation—changes in the direction of intergenerational transfers of wealth within the family, whether in the form of money, goods or resources. The economic role of children therefore becomes paramount—their cost or benefit to families, and the security they can provide to elderly parents.

Caldwell held that fertility decisions in all societies are economically rational responses to these familial wealth flows. Cultural transmission of new family values, of education and increases in the status of women are important in predicating declines in fertility and in determining demographic transition.

SUMMARY

In the 40 years from 1860 to 1900, the all-cause death rate in Australia halved, declining from 2,059 per 100,000 population to 1,123. Falling death rates among infants and young children, along with fewer young-adult deaths from tuberculosis contributed largely to the fall.

The infant mortality rate of 128 deaths per 1,000 live births in 1885 declined to 111 in 1903, and in a single year, fell to 82 in 1904. Much of the decline resulted from falling post-neonatal death rates, reflecting factors which were exogenous to the birth process and which were environmental in origin. Mortality among young children aged 1–4 years also fell substantially, by 80% between 1860 and 1906. In the early 1860s, under-five deaths comprised half of all deaths — by the early 1900s this had fallen to 30 per cent, a still-substantial proportion. Under-five mortality rates were always higher in urban regions, and in the summer months, when food- and water-borne infectious diseases took their greatest toll.

Tuberculosis caused around one-tenth of total deaths in Australia, with young adults in urban areas most affected. The TB death rate exhibited two distinct phases, rising steadily until the mid-1880s and falling rapidly thereafter. Although TB mortality in Australia ostensibly demonstrated cohort effects, the onset of the decline occurred simultaneously in many age groups, which indicates a period effect. Early epidemiologists debunked the commonly-held myth of natural immunity to TB among native-born Australians.

In 1865 and again in 1885, gastrointestinal infection was the most common cause of death, with infants most affected. By 1905, circulatory disease, a chronic cause, headed the list of causes of death. Leading causes varied among age groups, with gastrointestinal infection most common among infants, tuberculosis among young adults, circulatory disease among older adults, and the vague cause 'old age' among the elderly.

In this work, the decline in mortality in Australia throughout the second half of the nineteenth century has been interpreted with the aid of a number of theories of demographic change. Thomas McKeown's interpretation of the reasons for population growth largely dismisses changes in the virulence of organisms,

immunisation and the role of clinical medicine in treating illness, and focuses on economic development and rising living standards through improved nutrition, accompanied by sanitary improvements in the latter half of the nineteenth century. An apparent decline in respiratory tuberculosis was central to McKeown's analysis of falling English mortality rates. Tuberculosis was also a major cause of death in Australia, although much of the course of its rise and fall took place in the interim period between McKeown's two points of measurement. In Australia, falling under-five death rates from gastrointestinal diseases lay claim to a large share of the mortality decline. As far as can be ascertained, improved nutrition did not play a major role in mortality decline.

Epidemiological transition theory has as its focus the long-term change in mortality as the proportion of total deaths from communicable diseases declines and the proportion from non-communicable diseases increases. Australian data indicate that death rates from both communicable and non-communicable diseases declined during 1885–1906 and that the proportional contribution of each remained constant, indicating that there is no real evidence of epidemiological transition during the period. Only in the last few years—after 1903—did the proportion of non-communicable disease begin to rise appreciably and communicable disease fall, a pattern which would continue throughout most of the twentieth century.

Classical demographic transition theory holds that mortality decline is the major determinant of falls in fertility. Fertility in Australia appears to have been declining since at least the 1860s, and mortality may only have been an important determinant of fertility decline after the 1885 turning point in death rates. Other agents apart from a decline in the mortality rate also have a claim to causality.

Chapter 8

Social and environmental determinants of mortality

The sanitary revolution which underpinned the public health movement in Australia began in the 1850s, and ensured that the social and environmental determinants of health—the conditions in which people are born, grow, live, work and age—were at the forefront of Victorian-era health reform (Lewis, 2003, pp.52–93). The state had little involvement; health matters in the mid-nineteenth century were still largely an individual responsibility, and health inequities went unacknowledged and unchallenged. Poorer living and working conditions led to more sickness and shorter lives.

Responding to the harmful consequences of the rapidly deteriorating physical environment in most Australian capital cities, professional and political struggle began to agitate for health reform (Lewis, 2003). Locally administered preventive health measures sought to offset the deleterious impacts of bad water, poor sewage disposal, no sanitation and other urban fallouts of growth and industrialisation (Szreter, 1988). The need to deal with poor health across the entire social gradient became apparent if public health improvements were to usher in population health gains where they were needed most, which was among the urban poor and disadvantaged.

The examination of social and environmental determinants of health can provide useful insights into why death rates declined rapidly in Australia after the mid-1880s. This chapter develops this theme. It suggests that in nineteenth-century Australia it was where one lived, and the conditions in which one lived that were important and proximate determinants of mortality.

A modern framework for understanding the action of health determinants, which includes not only social, but also biological, behavioural and other determinants, is presented in Figure 8.1. This framework also illustrates pathways of health in colonial Australia. Some of these determinants—health literacy and knowledge, access to health services, vaccination—were less relevant 150 years ago. Others—such as housing, the built environment, food security, occupational health and safety, and birth weight—assume greater importance as determinants of health in the late nineteenth-century.

Figure 8.1: A modern framework for the determinants of health
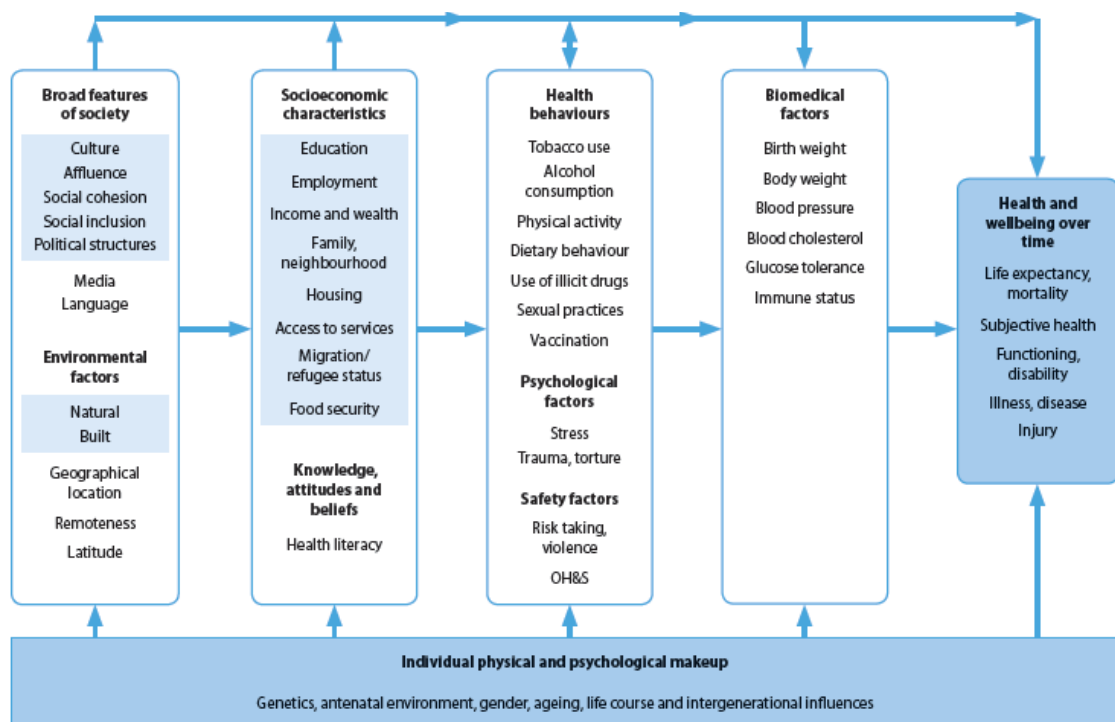
Note: Blue shading highlights selected social determinants of health.

Source: AIHW, 2012.

The framework illustrates that the broad features of society along with environmental factors such as geographic location act to determine socioeconomic characteristics including level of education, employment, income and housing (AIHW, 2012). These 'upstream' factors act in their own right to promote the health of individuals and also influence more immediate health behaviours and biomedical factors, leading ultimately to their effects on illness and mortality.

Deriving measures of social determinants and their effects during this era is problematic. Without population surveys, there are difficulties in measuring socioeconomic position and in linking these measures to any health outcome at all, let alone mortality. Administrative data, in the form of death certificates, offers information on usual occupation and place of residence, and these are on occasion summarised in the Registrar's abstracts of mortality. These are useful. Another method infers relationships through correlations of unlinked socioeconomic and mortality data. Both methods are used in this chapter to seek to establish whether a relationship between social and environmental determinants and mortality existed during this period.

URBANISATION AND THE BEGINNINGS OF PUBLIC HEALTH

In the second half of the nineteenth century, Australia became the English-speaking world's most urbanised population, and its first nation of suburbanites (Butlin, 1964, p.181). The 1891 census revealed that almost two-thirds of the Australian population were now living in cities and towns. Sydney's population had grown from 96,000 persons in 1861 to 496,000 in 1901, with its share in the total NSW population increasing from 27 to 37 per cent. Melbourne grew from 125,000 persons to 478,000 or from 23 to 40 per cent of the Victorian population. Adelaide expanded from 35,000 people (28% of the South Australian population) to 141,000 (39%). Other capitals also grew rapidly although extra-urban expansion kept pace in these cities.

The three decades from the 1860s were years of unparalleled economic growth and prosperity. Gauged by aggregates, and without reference to distributional

inequities, Australians seemingly lived in a 'workingman's paradise' and could claim to be among the wealthiest peoples in the world (Lewis, 2003, p.52). New South Wales Government Statistician Coghlan was of the opinion that 'the contrast between rich and poor, which seems so peculiar a phase of modern civilisation, finds no parallel in these southern lands' (1887, p.491).

Immigration had largely been channelled through capital cities; these were the centres of government and trade, and development from an early date focussed on commercial and industrial specialisations which were unique to urban societies.

Although rapid economic growth might be expected to deliver enhanced population health (Sinclair, 1975), in practice the reverse occurred, and the 'Four D's of disruption, deprivation, disease and death' characterised Australia along with other rapidly developing nations (Szreter, 2004).

Inherent in increasing urbanisation was the problem of public health. The same processes which created the booming economy, the factory and the modern urban environment brought into being health problems that necessitated new means of disease prevention and health protection (Rosen, 1958, p.177). Increasingly, social and environmental determinants combined to create 'disease environments', so that where one lived had great bearing on when and how one died (Woods & Shelton, 2000). The shared goal of many nineteenth-century professionals concerned about housing, city planning and public health was to reduce the transmission of infectious disease and to lower mortality, especially in urban areas (Lawrence, 2009).

In England, Edwin Chadwick, Secretary of the Poor Law Board, had established that there was a connection between housing conditions and population health. People living in sanitary conditions as a rule lived longer than those in slums, and rural dwellers could expect to live longer than their counterparts in the same socioeconomic class in urban areas. The sanitary reform movement founded in the 1840s had identified links between environmental conditions such as water supply, sewage disposal, and damp and mould in housing.

Overcrowding in urban areas, gauged by the number of persons per habitable room, was also considered unhygienic and unsanitary.

In Australia as in England, a relatively small public health movement bore the brunt of the challenge and strove against the competing interests of city corporations, landowners and ratepayers (Bashford, 2009; Szreter, 1988). This loose collection of concerned persons was comprised of government health officers, sanitary inspectors and certain other officials, enlightened medical men, health societies and philanthropic groups, working class improvement societies and sections of the print media. Elements of the public health movement had also spread through a number of professions, including medicine, science and engineering, so that social wellbeing was commonly linked to environmental factors (Wong, 1999).

Urban planning had rarely kept pace with the unrestricted growth of the major cities in the colonies. Despite the need, settlement did not follow the provision of utilities. Much substandard housing was built, occupied mostly by the working-classes, and gross overcrowding combined with deplorable sanitary provisions to spread infection. This, along with working-class poverty — which existed despite Coghlan's enthusiasm — led to death rates which were not so different to those of much older cities in Europe and the United States (Lewis, 2003, p.53).

By the 1850s, Sydney's population numbered about 39,000, and the city had already developed major slums (Lewis & MacLeod, 1987). In February 1851, the *Sydney Morning Herald* began a series of ten articles on the sanitary state of the city, aiming,

> '...by calm enquiry, to amass a vast quantity of useful information, in order to show the masses, as well as those in power, that the [sanitary] evils are deep-rooted and highly destructive to the common weal, and that it is the duty, as it is the interest, of all classes to combine, in order to eradicate them' (*SMH*, 1 Feb. 1851, p.2).

Two years on, an editorial in the *Empire* questioned whether Sydney's hurried urban growth warranted the social costs,

'The health of the city is now becoming a very serious question. The population is rapidly augmenting, the means of accommodation are wholly inadequate even for our present numbers, and our sanitary arrangements are just on an equality with those of an English village or market town of the humbler class. Our population is now decimated by sickness, and though it may be contended that influenza and sewerage have no necessary connection, the heavy scourge of a general epidemic occurring twice in an interval of little more than six months, and that in a climate hitherto looked upon as unexampled in point of salubrity, is at least ominous' (*Empire*, 9 May 1853, p.2).

In 1857, the Sydney Corporation established the position of Chief Health Officer with Dr Isaac Aaron, a former member of Birmingham's local health board and editor of the *Australian Medical Journal*, the first appointee (see Appendix 3). Aaron pursued sanitary improvement with enthusiasm, but soon clashed with the City Council (Lewis, 2003, p.76). In his inaugural quarterly report, Aaron promoted the new hygienic science 'as applicable to the congregation of large numbers of beings within a comparatively limited space' (*Empire*, 29 Sep. 1857, p.6). He cautioned that given the state of the city, it could not expect to remain free from the devastating scourges which appeared in other cities around the globe, and 'The extent of the evil will necessarily render the work of sanitary reform laborious, expensive and slow'.

Chris Rolleston, NSW Registrar General, likewise warned in his first annual report that

'The Registration of the last year has shown that the rate of mortality in Sydney exceeded that of London in 1854—a year of cholera—and that the infantile mortality is far beyond what it ought to be. These facts are incontrovertible, and call for remedy' (*NSWRG*, 1857, p.4).

In a paper on the sanitary condition of Sydney, given to the NSW Philosophical Society in the following year, Rolleston made use of his new registration data, asserting,

'...that no city in the world possesses within itself such elements of health that Sydney does, but as the density of our population increases, we must not neglect those sanitary precautions—such as free circulation of air, efficient sewerage, abundance of pure water, and general cleanliness—which have been found, in all countries, necessary to the maintenance and security of the public health:—blessed

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are we by nature, we have no right to expect immunity from disease, if we disregard the filth of our streets and alleys, and the improper ventilation of the dwellings of the labouring classes' (Rolleston, 1858a).

These messages were to be repeated by Registrars, Statisticians and city health officers across Australia for decades to come (Mayne, 1982, p.55ff.).

As a public health consciousness grew, so did the need for evidence to drive reform. The Registrar's mortality abstracts were one of the few available statistical resources, and these were often used by city health officers,

> "These tables are of extreme value, although perhaps not generally studied. Still, it is upon such calculations as these that sanitary reforms must to a great extent be based" (*SAA*, 4 Apr. 1861, p.2).

Figure 8.2: Overcrowding and mortality, Melbourne sub-districts, 1871–1901
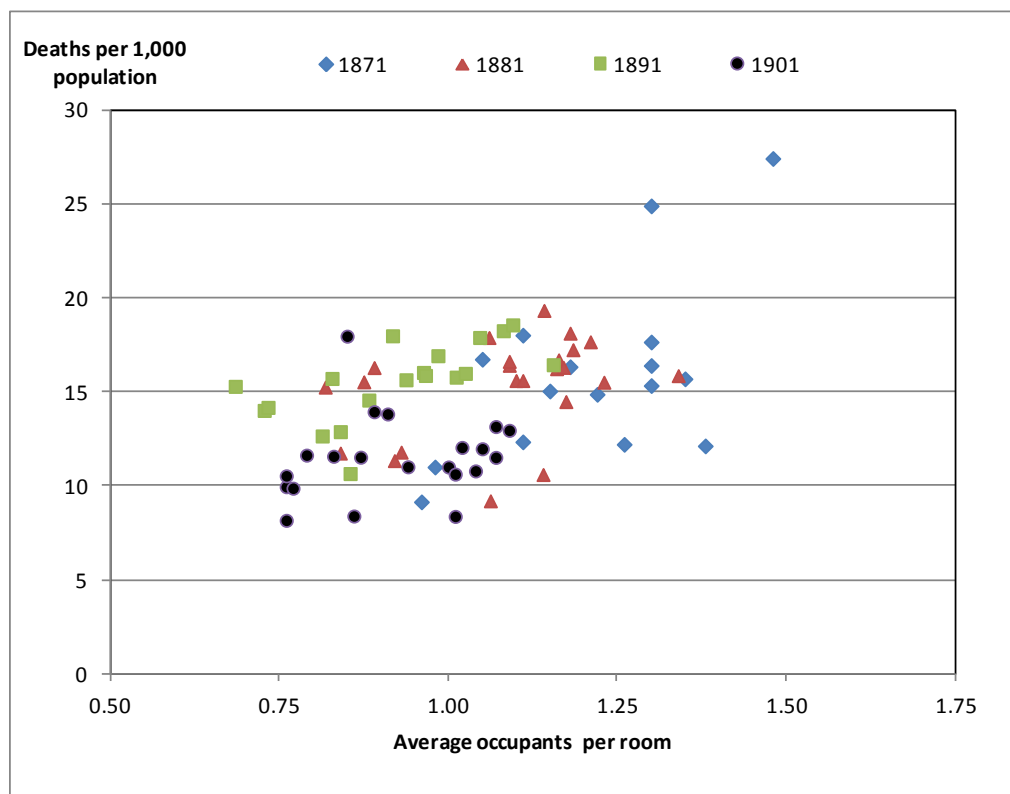
Urbanisation and mortality increased together. The changing role of the city is best illustrated by considering not the size of the population, but its density. Census data and Registrar's abstracts can be combined to illustrate the positive relationship between population density (here measured by occupants per

room) and mortality rates in Greater Melbourne (Figure 8.2). Areas with greater population density generally had higher mortality rates, the relationship being most robust for the year 1891 (r^2=0.41). The plush multi-roomed houses of Brighton, St Kilda, Prahan and Hawthorn stood in stark contrast to the mean dwellings and close confines of working-class North Melbourne, Footscray, Port Melbourne and Collingwood (Davison, 2004).

Outside the city residents fared somewhat better. Substantial mortality differentials existed between urban and rural regions within colonies from at least the 1870s (Figs. 4.11, 7.6 and 7.13). Infant deaths provided a sensitive indicator of quality of life and of the geographic differential in overall mortality (Fig. 8.3). Given the pattern of cause-specific mortality, with much due to gastrointestinal infection, it is reasonable to argue that poor living conditions were decisive for infant mortality (Lewis & MacLeod, 1987). Infants benefited from improved life chances according to the region in which they were born (Mein Smith, 1997, p.19ff.). Babies born in rapidly growing cities and their outer suburbs were at greater risk of gastrointestinal infection.

Figure 8.3: Infant mortality in urban/rural New South Wales, 1871 to 1906
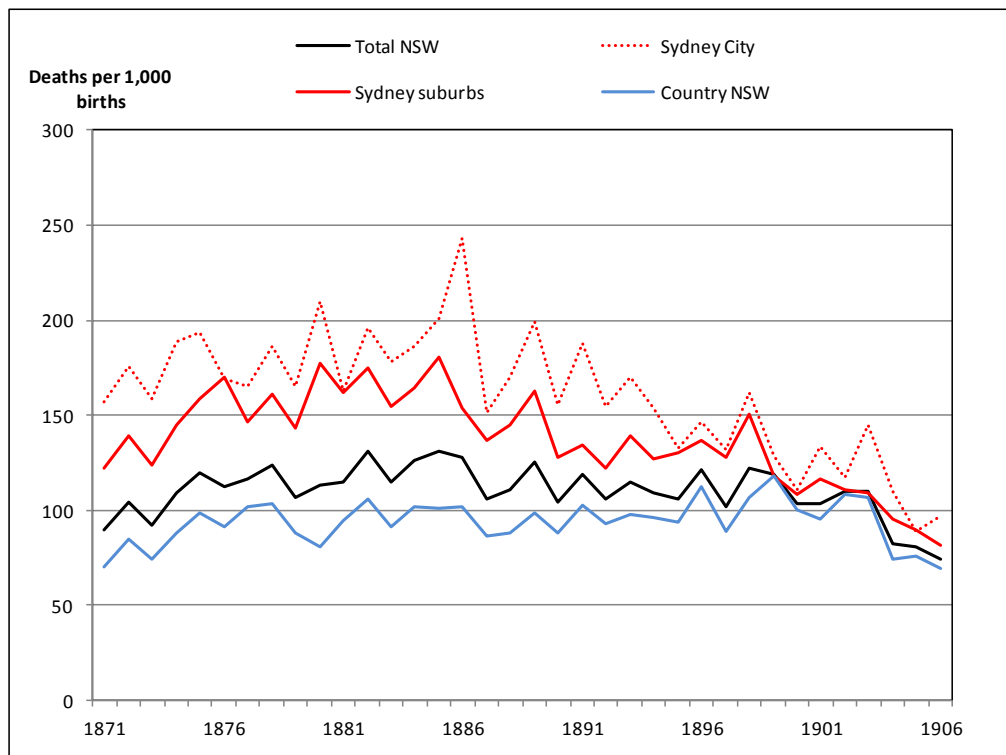
In 1875, Sydney experienced one of its highest mortality rates to date. In March, the *Sydney Morning Herald* commented that 'there has scarcely been a locality or a street in which disease, in some form, has not made its appearance' (*SMH*, 4 Mar. 1875, p.4), and throughout the remainder of the year the newspaper regularly castigated its readership for their perceived apathy concerning the health state of the city (Mayne, 1982, p.28).

In Sydney, London and many other world cities, the late 1870s and early 1880s saw increased scrutiny of the social, economic and moral problems arising out of inner city poverty and urban slums (Ibid, p.86; Kelly, 1978). A Sewage and Health Board was appointed in 1876 to inquire urgently into overcrowding, poor housing and other social determinants of Sydney's worsening ill-health. Public meetings launched Health Societies in both Sydney and Melbourne (Dunstan, 2003), and the Royal Society of NSW formed a sanitary science section.

Legislation followed. Victoria had been the first colony to pass a public health act in 1854, creating a Central Board of Health. The spur of smallpox epidemics in the early 1880s, along with the example of the English Public Health Act of 1875 led to a host of legislation, culminating in each colony passing a public health act, largely based on British law (Whittell, 1892).

> 'By the close of the nineteenth century, public health in Australia involved state-imposed prevention of disease through environmental sanitary regulation — reinforced by publicly run reticulated water and underground sewerage systems, at least in the larger colonial cities — and controls over infectious diseases through the practice of notification, inspection, disinfection and isolation' (Lewis, 2003, p.72).

In New South Wales, the *City of Sydney Improvement Act of 1879* provided new regulations to police housing unfit for human habitation, and gave impetus to the large-scale slum clearances which would take place throughout the following decade (Mayne, 1982, p.170). The *Infectious Diseases Supervision Act, 1881* formed a Central Board of Health, and required householders to notify cases of smallpox. Incessant typhoid epidemics led to the *Dairies Supervision Act, 1886*, this being the first legislation influenced by germ theory, with human

and bovine infectious disease cases to be reported, and standards of sanitation established for dairies (Lewis, 2003, p.81). A comprehensive *Public Health Act* was finally passed in New South Wales in 1896. With hindsight however, the prevailing opinion on the actions of most colonial legislatures was that they were slow-moving, if not inept (Fitzgerald, 1987, p.100).
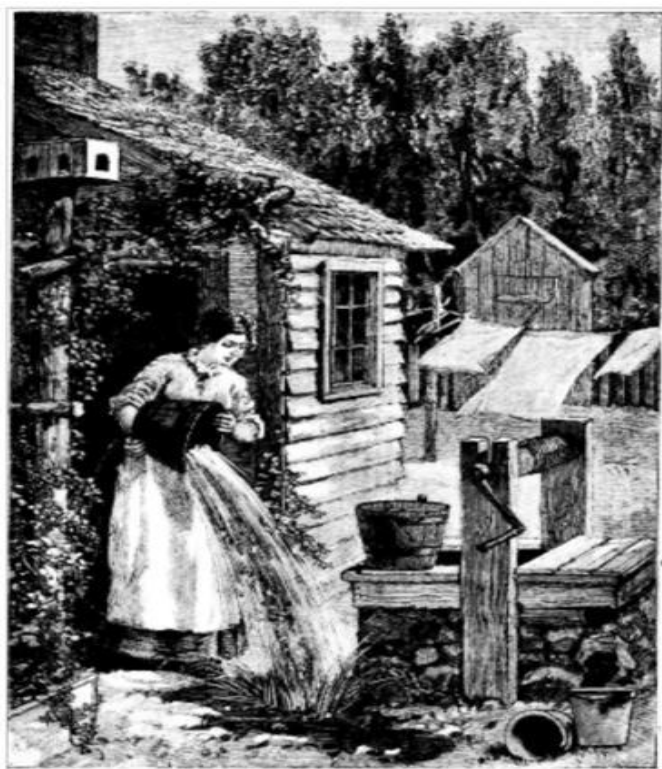
Figure 8.4: Preparing the way for fever (*Illustrated Sydney News*, 19 Mar. 1881)

In the suburbs, urbanisation was also having effect. By 1881, the suburban population of Sydney had exceeded that of the inner city (Fitzgerald, 1987, p.18). The 1880s was known as a decade of smallpox scares, but was also the 'decade of the suburbs'. In Sydney and a number of other capital cities, suburban mortality rates climbed. Crowding was no longer confined to the inner city.

> 'Turning to the suburbs, which most persons have been accustomed to look upon as healthy retreats from the confinement of the city, the case is still more alarming...the hope of the city lies in the fact that its salubrity has steadily improved, and may continue to do so, while, in regard to the suburbs, the death-rate has been steadily growing' (Coghlan, 1888, p.359).

Figure 8.5: A New Year's vision - a new broom much wanted in Sydney (*Illustrated Sydney News*, 24 Jan. 1880)

In some capitals suburban mortality outstripped that of the inner city due largely to untrammelled growth, a lack of infrastructure and little foresight in planning leading to a deteriorating environment,

> '...the suburban portions of a country are generally the most inimical to health, and are loaded with the largest mortality; for whilst such areas possess a population sufficiently large to create an insanitary condition, yet the provisions for sanitation are not, of necessity, equal to securing as complete a system of sewage and scavengering as are possible with the more populous and wealthy urban centres' (QldRG, 1896, p.xxvii).

For much of the period, the injurious effects of miasma associated with city filth, in addition to more orthodox social determinants, was thought to be the reason for the higher mortality, especially for infants,

> 'There can be no doubt whatever that a large percentage of deaths of children...is traceable to the neglect of parents, want of proper nourishment, and to the ill-constructed, badly drained and ventilated dwellings of the poorer classes in towns...even in Brisbane, with the advantage of a large supply of pure water, the chances are greatly against the life of a child inhabiting some of the low-lying

streets of that city...Who will venture to say that the poisonous miasma from the stagnant pools and open sewers in these localities does not convey the cause of death to many a child living in such precincts?' (QldRG, 1870, p.ix).

Germ theory was yet to have its full impact in some jurisdictions,

'...it would appear that the absence of the insulating septum of pure air around town dwellings renders the inmates of every house more liable to the attack of all infectious diseases which may be floating in any part of the atmosphere within the town' (TasSR, 1887, p.xxxii).

For the New South Wales Statistician, urban mortality held no secrets,

'The comparatively high death rates of Sydney certainly do not arise from natural causes...What nature with lavish hand had bestowed, was, however, until recently, in danger of being destroyed or polluted; for looking through the causes of death the conclusion is inevitable that no small part of the mortality of Sydney and its suburbs arose, primarily or indirectly, from diseases which sanitary precautions might have averted' (Coghlan, 1902a, p.1008).

Figure 8.6: A back lane in Sydney (*Illustrated Sydney News*, 19 Jul. 1890)

Urban growth would eventually require heavy new investment in public utilities such as underground sewerage systems, larger water supplies, gas,

roads, street lighting and public transport (McCarty, 1970). Government authorities were having to play catch-up following years of neglect,

> 'In Edwardian Victoria local government bore responsibilities for social welfare, sanitation and drainage, food and drink adulteration, roads and quarries, town planning, building regulation and slum control, infant welfare and pasteurized milk, health and cultural enlightenment—a burden far in excess of its financial and human resources' (McCalman, 1984, p.36).

The differential eventually narrowed. The turning point in urban-rural mortality among infants and children as well as adults was similar to that for the total mortality; Australian cities became safer places to live late in the century, with improvement dating from 1885 in Sydney, and 1890 in Melbourne (Figs. 4.11, 7.6).

By 1906, urban and rural mortality rates in New South Wales were much the same. The reasons are complex, but were held at the time to be due to the newly constructed sewerage system, and also reduced tuberculosis mortality attributed to better quality milk.

> 'In both metropolis and country the rate has steadily improved, but very much more in the metropolis, so that there the rate is now very little higher than in the country districts, whereas twenty years ago it was 50 per cent higher. The fall began in the metropolis after 1889, the year when the improved sewerage system was installed; and about the same time that the Dairies Supervision Act came into operation' (Trivett, 1909, p.63).

For infants, the urban-rural difference continued to narrow, to the extent that by the late 1920s in Sydney and the late 1940s in Melbourne, urban centres became the safest place in which to live (Mein Smith, 1997, p.21).

WATER AND SANITATION

The greatest health gains in nineteenth-century Australia were delivered by improved water and sanitation, those two most basic foundations of public health (Bartram & Cairncross, 2010). Clean water and the effective disposal of sewage and other refuse from streets and houses prevent the spread of communicable diseases, and especially gastrointestinal infections in the form of

diarrhoea, dysentery, cholera, typhoid or enteritis. These were the diseases which were largely responsible for the harvest of infant and under-five deaths which comprised half of all mortality.

Water quality remained a problem throughout much of Australian colonial history. City supplies were more-or-less polluted by the time they reached the consumer, yet it was only when supply problems reached crisis proportions that general community disinterest and inertia were challenged, prompting calls for change.

In Sydney, urban growth overwhelmed the capacity for water supply or adequate sewage disposal. Construction often preceded drainage, and large numbers of houses were poorly constructed. The worst areas were those where human errors compounded natural inadequacies of the site, and where geographical separation encouraged social divisions between the poor and better-off (Fitzgerald, 1987, pp.226–227).

Sanitary conditions were already bad by 1850, when an Act 'for the better sewerage, cleansing and draining of the City of Sydney' was passed (Cumpston, 1989, p.383). Water supply was little better, with only 2,300 houses out of approximately 8,500 connected to mains supplies from Busby's Bore, the city's water source (Aird, 1961). The disadvantaged were inordinately affected, and working-class communities often had to pay water carriers for their weekly supply,

> 'Half the time of some of the poor is taken up in obtaining water from pumps, sometimes more than a quarter of a mile distant. At all hours of the day and night these pumps are at work; two and three o'clock in the morning, different parts of the neighbourhood are the scenes of water brawls, when the pumps are regularly besieged in the struggles of the people for WATER' (*SMH*, 1 Feb. 1851, p.2).

W. S. Jevon's survey of late-1850s Sydney offers much detail on the sanitary state of the city, and the social effects of the inadequate water supply (Clark, 1978, p.60). He described Sydney's Rocks area, with its

> '...utter absence of all means of drainage or of removing filthy matter...that in many cases the foul drainage of one cottage trickles down the hill till it encounters...the back or front wall of the house next below; here it accumulates, soaking down into

the foundations, or sometimes actually running in at the door' (*SMH*, 7 Oct. 1858, p.2).

An 1860 Select Committee enquired into the condition of Sydney's working classes, focussing on sanitary conditions. Their poor personal hygiene was blamed on the difficulty in securing water for their homes, and compounded by authorities doing away with street fountains in favour of mains connections, which many urban poor could ill-afford. Inner city sewage facilities were inadequate; of 1,446 houses inspected, only 356 had water closets (Lewis, 1979).

Between 1859 and 1886, the city's water was supplied from the Botany Swamps. The area was 'liable to pollution from many sources, and the district of Botany itself has been for many years a hot bed for diarrhoea and typhoid fever' (Armstrong, in Lewis, 1979). Despite the clamour for improvement, the council refused to consider alternative schemes, swayed by vested interests and hampered by a lack of technical ability and of funds (Clark, 1978, p.55; Fitzgerald, 1987, p.81). Even though the Botany swamp water was bad, areas without a mains supply suffered even more, with the absence of a sewerage system leading to a higher incidence of water-borne infectious diseases.

The issue was water quality. From the 1870s, increasingly shrill commentary pointed out the high mortality rate in the metropolis, with the Chief Health Officer of Sydney, Dr Dansey linking this in part to poor water and lack of provision for sewerage. The ominous warnings were borne out in 1875, the *annus horribilis* of disease epidemics.

The Sewerage and Health Board was created in that same year of 1875. It reported that less than 50 per cent of all households were connected to sewers, and 4,700 of Sydney's 5,400 water closets were connected directly to water mains with faulty valves, so that the supply was contaminated with excreta, and repeatedly trickled 'gross...faecal matter' (Ibid, p.83; Smith, 2011, p.235). Much of the rest of the city relied on pans, with cesspits common in the suburbs. Water supplies here were generally provided by ground wells, which were often little more than open sewers. Dust, street sweepings and animal manure were further sources of contamination.

Figure 8.7: The water famine in the Sydney suburbs — moonlight scene in Woollahra (*Illustrated Sydney News*, 4 Sep. 1880)

By the 1880s, city water supplies were often partial and intermittent. The increased surge of interest in public health in the mid-1880s led at last to the completion of Nepean water scheme in 1887. An underground sewerage scheme completed in 1889 served some areas, although it took another two decades to fully connect the city (Aird, 1961). Even in 1906, Sydney water was still unfiltered (Smith, 2011, p.235).

Sydney's problems were common to other cities and towns (Smith, 2011, ch. 10). Although disease epidemiology was still largely based on miasma theory, the health officers continually linked high mortality, especially of infants, to the parlous state of the water supply and inadequate sewage provision. Henry Jordan, Queensland Registrar-General, reported that

"Sewage and drainage must be on an intelligent and comprehensive scale, almost regardless of cost, if the death rate in our large towns is to be materially lessened; or, in other words, if we wish to preserve our children alive, and to live ourselves till, in the natural order of things, we go to our graves. As it is, we are poisoned by

the impure air we breathe, and by the zymads which live in the water we drink"
(QldRG, 1876, p.xxxiv).

Newcastle, New South Wales' second city, grappled with sanitation questions from the mid-1870s, with debates over nightsoil disposal, drainage and water supply becoming increasingly interwoven. Problems were most acute in the overcrowded inner city residential area,

> 'It is beyond the power of any pen to describe the misery that pervades those localities where there is a scanty supply of water, or, in many instances, no supply at all. The unwashed floors, the fetid smells, the pallid faces, the general aspect of dirt and wretchedness at once reveal the plague inviting spots where water is denied' (*Newcastle Chronicle*, 25 Apr. 1874, p.2).

The city medical officer, Dr Robert Dick, thought that the mortality rate was 'three times that which nature dictated as inevitable', with typhoid or 'dirt fever' attributed to bad smells, bad drainage, bad water, and filthy water closets (Lloyd, Troy & Schreiner, 1992, pp.6, 35).

In Western Australia, the colonial secretary attracted attention by informing the citizens of Perth that they were living on a dunghill. Having been spared many of the diseases of the eastern colonies, in 1883 Western Australia was visited by a series of epidemics, with the first outbreak of measles since 1860 and a cold and rainy winter favouring an upsurge of typhoid and diphtheria among working class inhabitants of Perth (Hunt & Bolton, 1978).

Colonial Surgeon Dr Alfred Waylen reported that 'Fever and diphtheria may be looked on as endemic diseases in Perth and Fremantle fostered by sewage contamination of air and water'. He added wearily

> 'I have for so many years past animadverted on the absence of some system of sewage disposal and the consequent contamination of drinking water, not only in Perth and Fremantle, but in other towns of the Colony, that I only now refer to the subject in the hope that, ere long, municipal councils will become alive to a sense of their duty' (*WAPH*, 1883, p.4).

With unprecedented growth resulting from the 1890s gold rush and railway building, impossible strains were placed on the water supply. The years 1895 to

1898 were a period of extremely high typhoid mortality, mostly among poorer classes (Nyulasy, 1898; Hunt & Bolton, 1978).

With few exceptions, progress was slow. The date of commencement for the installation of sewerage systems in major cities is shown in Table 8.1.

Table 8.1: Installation of sewerage systems in cities

City	Date of commencement of installation
Launceston, Tasmania	1856
Adelaide, South Australia	1879
Sydney, New South Wales	1885—completed 1889
Melbourne, Victoria	1895—first house connection in 1897
Hobart, Tasmania	1902
Burnie, Tasmania	1907
Newcastle, New South Wales	1907
Geelong, Victoria	1910
Perth, Western Australia	1911
Brisbane, Queensland	1914—first house connection in 1922

Sources: Cumpston, 1989; Lloyd, Troy & Schreiner, 1992.

In Melbourne, city officials examined the mortality statistics hoping for some evidence that their labours and costs were bearing fruit (Jamieson, 1905; Thwaites, 1906). The annual reports of the Melbourne and Metropolitan Board of Works routinely included vital statistics of the metropolis alongside tables of the ever-growing number of sewerage house connections (Figure 8.8).

Figure 8.8: Sewerage connections, infant mortality and typhoid death rates, Greater Melbourne, 1893–94 to 1909–10
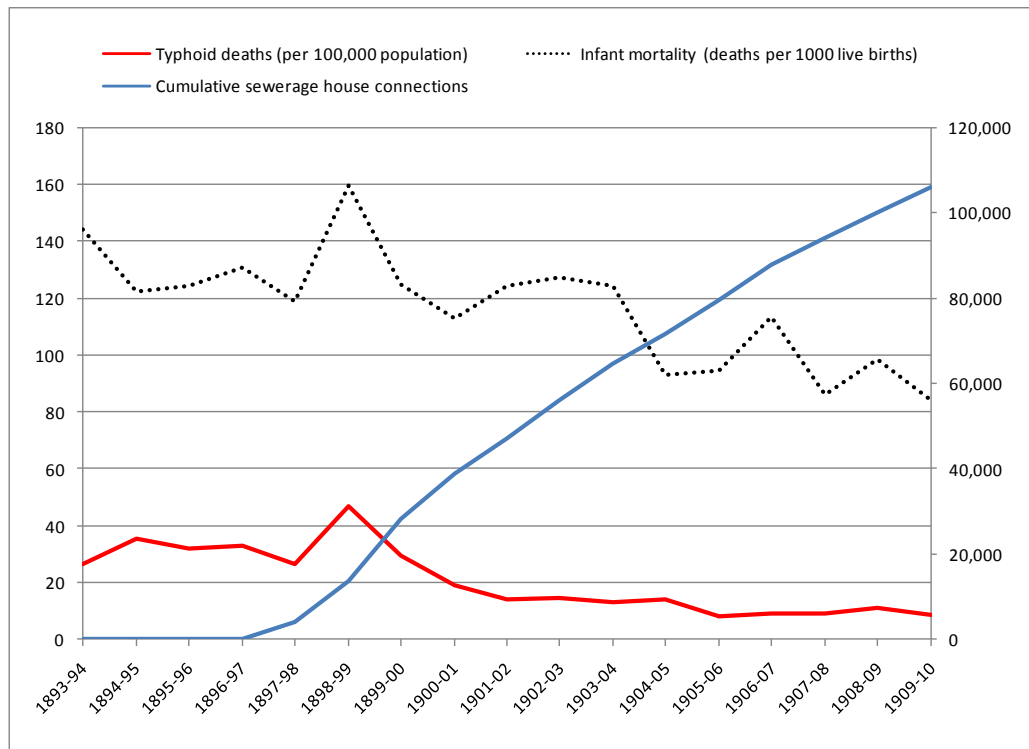
Source: Dingle & Rasmussen, 1991, p.102 for sewerage connections.

Death rates from typhoid, diphtheria and diarrhoeal diseases vied with the vestiges of miasmatic theory in the form of records of rainfall, humidity and temperature. Chief Engineer William Thwaites had a firm belief in the ability of the engineer to bring about social improvement,

> 'As the Melbourne Sewerage System advances, the immense saving of human life is becoming more and more manifest, independently of the vastly improved comforts of the homes not only of the richer, but also of the poorer classes; for the fittings provided in the latter are not only as sanitary as those of their more highly favoured brethren, but the workman's cottage is on the average much better provided for than in any city in the world' (Melbourne and Metropolitan Board of Works, 1906–07, p.689).

However, death rates from gastrointestinal infection in a number of capital cities showed little change in the period following the installation of sewerage systems (Figure 8.9):

Figure 8.9: Enteric fever death rate in capital cities, 1875–1925
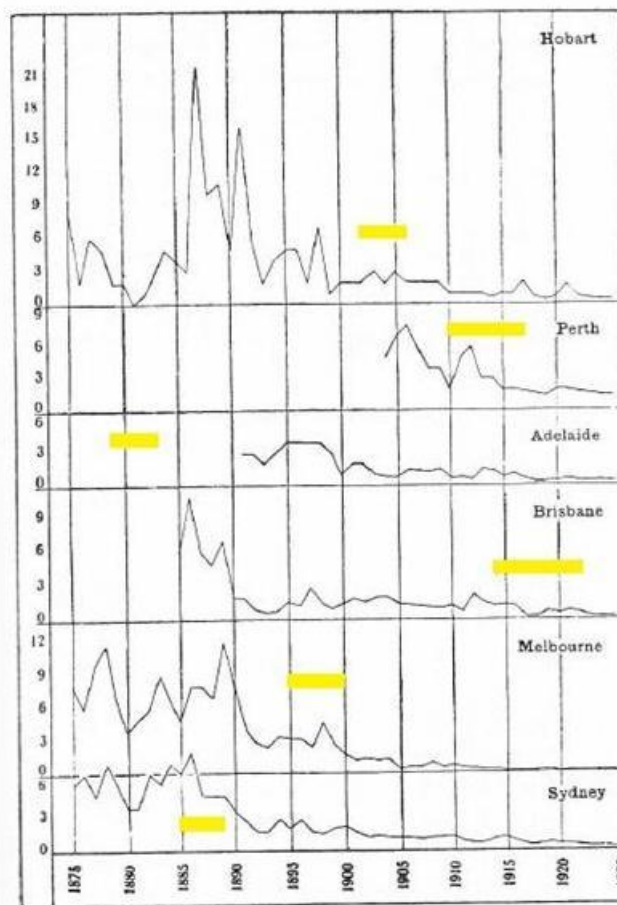
Note: Yellow enclosures indicate years during which sewerage systems were installed.

Source: Cumpston, 1989, p.235.

The clinical equation as we now know was simple. Modern research shows that a huge disease burden is associated with poor hygiene, sanitation and water supply, and that this can largely be prevented with proven interventions that will ultimately provide much greater benefit than health benefits alone (Bartram & Cairncross, 2010). Mortality reductions result when the level of pathogen ingestion in water- and sewerage-connected households improves in comparison with non-connected households. And for every death avoided from waterborne causes, there are three or more deaths from other causes not usually considered waterborne that are also prevented (Ferrie & Troesken, 2008). Quality drinking water, sanitation and personal hygiene each influence disease load (van Poppel & van de Heijden, 1997).
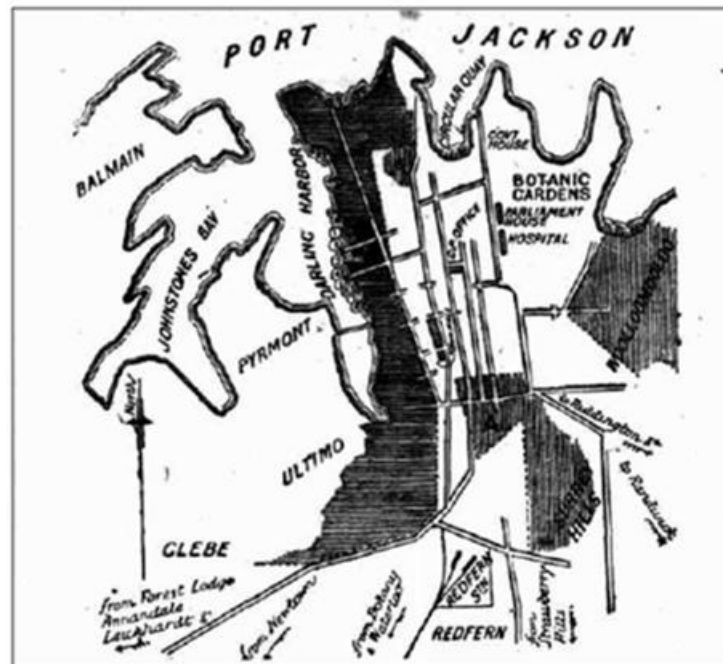
Figure 8.10: A map of insanitary Sydney (*Albury Banner*, 6 Apr. 1900)

Yet the effects of improved water or sewerage on public health were not self-evident. Cumpston realised that falls in gastrointestinal infection death rates could not be attributed solely to the introduction of sewerage systems into capital cities. Typhoid death rates began to decline more than seven years before houses were first connected to sewers in Melbourne, and the Victorian decline from 1890 also occurred in unsewered regional cities such as Ballarat and Bendigo (Lewis, 2003, p.75). Only Sydney and Perth saw the rate of death fall immediately after the introduction of sewerage (Figure 8.9), and even in these cities 'the possibility of coincidence rather than association cannot...be overlooked' (Cumpston, 1989, p.235). Sewerage was not the sole cause of mortality decline, but it did serve to reinforce existing downward trends in mortality.

For Cumpston, it was the cumulative effect of numerous public health activities, and executive action in many directions dating from at least the 1850s that made the difference (Ibid, p.236). Legislation, including Public Health Acts, had some effect by regulating milk production and the operation of noxious trades. The wholesale adoption of English legislation, however, was deemed

'mechanical and unenlightened'. 'Notwithstanding the spasmodic activities after 1850 it was not until after 1880 that organized public health activities became at all general' (Cumpston, 1928). Cumpston dated modern sanitary improvement from the turn of the century,

> '…it was only with the commencement of the twentieth century that Australia as a whole developed an informed and sensitive sanitary conscience. Consequently, the history of the intestinal infection has two phases — the nineteenth century of increasing incidence, of explosive manifestations, of general fluctuations and of spasmodic but inefficient control; the twentieth century of progressive and orderly control to a point far below that of any period of the nineteenth century' (Cumpston, in Lewis, 1979).

Cumpston's model of mortality decline might have benefitted from the addition of other social determinants of health, along with increases in medical knowledge and care and changes in economic well-being (van Poppel & van de Heijden, 1997). Although improvements in water quality and sewerage provision can claim an upstream position, so to speak, in mortality improvement, unidimensional explanations simplify the circumstances which lead to improved life-chances. Rather, clean water and reliable sewerage disposal co-exist in an inter-relationship with other social and environmental factors in influencing health and mortality.

ILLEGITIMACY

Perhaps no social factor was more disadvantageous to successful life chances than illegitimacy. Despite (or possibly because of) Australia's convict past, being born out of wedlock in the last quarter of the nineteenth century carried an utter public stigma. Illegitimate children were more often than not born to poor and unsupported mothers, in locations such as Sydney's Benevolent Asylum or in Melbourne's Lying-In Hospital.

Around 4.4% of births in New South Wales in 1880 were held to be illegitimate, the figure rising steadily over the next two decades to be 7.0% in 1900 (NSWRG, 1881, 1901). Rates were twice as high in metropolitan Sydney as in country

districts, being partly influenced by expectant women from all areas of the state travelling to the city to give birth in anonymity.

The average birthweight of babies born to single mothers was lower than for married women. In Melbourne's Lying-In Hospital, the decline in birthweight between 1860 and the mid-1870s was attributed to the effects of social determinants such as increasing poverty, alcohol abuse and criminal activity, as well as to disease and mental illness. Birthweights began to increase after 1880, following the replacement of old housing stock in the surrounding area, and with improved state and medical interventions for poor single mothers (Morley, McCalman & Carlin, 2003).

Having survived birth, greater hazards awaited the newborn infant. Since legal registration of still-births was unnecessary, the spectres of infanticide or 'concealed births' along with spurious baby-farming (or paid fostering) imperilled young lives (Fitzgerald, 1987, pp.99, 192–194). The Medical Officer was pessimistic regarding the future of illegitimate infants housed in Melbourne's Children's Hospital,

> 'The massing together of infants leads to high death rates...[neither] has any provision been made for isolating either new cases or cases of illness, the consequence is that at any time...dread disease may be suddenly launched into the centre of 100 or more susceptible little beings, all with lowered vitality, and the boasted small death rate immediately becomes an appalling one...Every infant is entitled to one pair of mother's arms; infants massed in an institution are deprived of this right' (Jeffreys Wood, 1908).

Infant mortality in Australia was already high in 1900, with one-in-ten dying before their first birthday (100 deaths per 1,000 live births) (see Chapter 7). But more than one-in-five babies born out of wedlock died before their first birthday. The mortality of illegitimate infants in New South Wales was nearly three times that of legitimate infants (278 vs. 100 deaths per 1,000 live births for the years 1895–1900) (Figure 8.11). Victoria had similar rates (232 vs. 82 per 1,000 live births for 1903–06). The differential holds up across each cause of death examined including gastrointestinal infections, respiratory diseases and

violence (accidental suffocation and murder), as well as for deaths attributed to 'artificial feeding' (Drake, 1908, p.348).

Figure 8.11: Legitimate and illegitimate infant mortality, New South Wales, 1895–1902
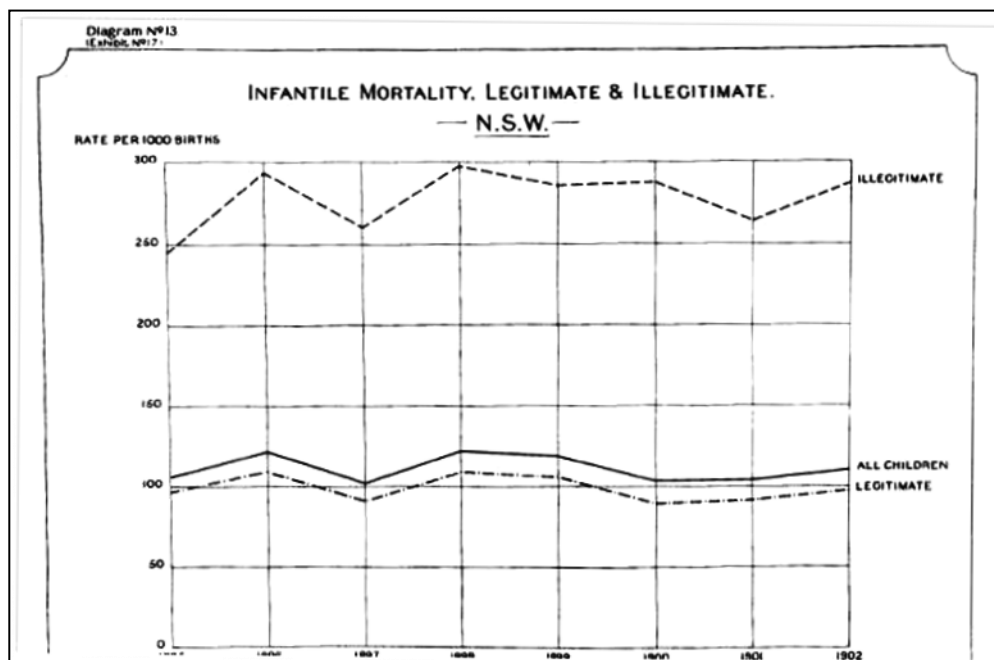
Source: New South Wales Government, 1904.

For some, the chances of life were even more perilous. Among babies born in the Melbourne Lying-In Hospital before the mid-1880s, infant mortality reached the exceptionally high figure of one-in-two (McCalman & Morley, 2008). The greatest risk surrounded 'unsupported women'; mothers who lacked a male breadwinner and adequate shelter, food and security to support their babies.

Insights as to why mortality was thought to be higher among ex-nuptial births come from the *Royal Commission on the Decline of the Birth-Rate* held in New South Wales in 1903. Five causes which led to higher death rates among illegitimate infants were:

'Maternal indifference and the social and economic disabilities of the mothers; The defective management of Foundling Homes, Infant Homes, and other places and institutions where illegitimate infants are received; The secret adoption of infants for gain; The separation of infants from their mothers; Infanticide, and foeticide of viable infants, undetected owing in some measure to existing facilities for

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interment in cemeteries and elsewhere of newborn dead infants as still-born infants' (New South Wales Government, 1904, p.39).

Despite their paternalistic attitudes of blame, the Commissioners recognised that the inequalities engendered by the 'social and economic disabilities of mothers' played a role in determining that most final of health outcomes, mortality.

The huge disparity between nuptial and ex-nuptial rates closed in the decades following the 1880s, and more so after the 1900s. This suggested to Mein Smith that more than economic variables were at work, with a greater contribution from family and social support for mothers (1997, p.24).

OCCUPATIONAL STATUS

There are a number of different theoretical approaches which conceptualise socioeconomic position and social class. These approaches have produced measures of socioeconomic position which are based on prestige, using attributes such as income, educational attainment or occupational status (Duncan *et al.*, 2002).

One influential measure of social class was developed by Goldthorpe, for use in research on social stratification, occupational mobility and educational attainment (Erikson & Goldthorpe, 1992). The measure includes class groupings of managers and professionals, white collar workers, self-employed and small business people, and divisions of manual workers based on levels of skill and supervision. Studies of social stratification emphasising occupational status typically ask respondents to rank occupations in terms of prestige, these being closely correlated with income and wealth (Broom, 1977; Jones, 1989).

Occupational status is the only socioeconomic attribute included in nineteenth century Australian death registrations that can be examined directly for its relationship with mortality. The occupation of males at time of death was routinely entered onto death certificates, and classified tables reporting on the occupations of decedents often formed part of the Registrar's abstracts of

mortality. Victorian statist Hayter commonly included these tables in his annual reports and year books.

Larson (1994) has developed an occupational classification derived from late nineteenth-century Victorian census data and municipal rate books. Occupations of males were apportioned to six classes which are ordered by social prestige — Professionals, White-Collar, Businessmen, Skilled Manual, Semi-skilled and Labourers.

Male deaths in occurring in Victoria during 1880–1882 were classified according to Larson's scheme. A mortality gradient is apparent across a number of occupational classes (Figure 8.12).

Figure 8.12: Mortality and occupational class among males aged 20 years and over, Victoria, 1880–1882
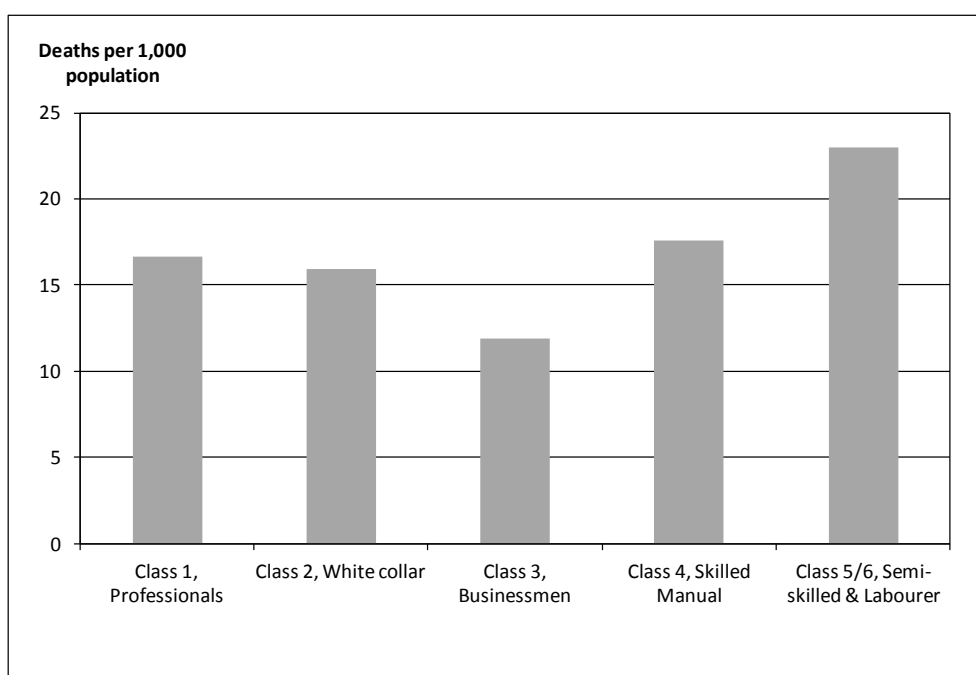
Sources: Calculations based on Victorian Year-Book 1883–4, pp.288–290; Larson, 1994, pp.193–204.

Businessmen (Class 3) had a crude mortality rate of 12 per 1,000 population. The rate for skilled manual workers (Class 4) was half as much again (18 per 1,000) and for semi-skilled workers and labourers (Classes 5 and 6) it was double (23 per 1,000).

The rate of 16 deaths per 1,000 population for professionals and white collar workers (Classes 1 and 2) was higher than for businessmen, but lower than for skilled manual workers. Professionals were the most economically advantaged group at this time, if reckoned by the mean annual value of the house in which they lived although their advantage over businessmen was only slight (Larson, 1994, p.197). White collar workers ranked third, and with a considerable economic gap. Despite professionals and white collar workers having higher occupational status than businessmen, they had higher mortality rates. A social gradient in mortality is apparent only for Classes 3 to 6.

This particular example illustrates that economic inequality was not invariably associated with unequal death rates, and that other aspects of culture and behaviour can also have an effect, even when infectious diseases are widely prevalent (Kunitz, 2007).

Colonial life was widely held to offer good opportunities for social mobility and well-being for the hard-working and industrious. Yet Fitzgerald, examining occupational mobility in late nineteenth-century Sydney, found no evidence that workers could rapidly climb the occupational ladder. The economy of the time, with its high demand for unskilled labourers and limited number of skilled and white-collar jobs, may well have resulted in a downgrading in status (Fitzgerald, 1987, p.227).

Occupation is not only an indicator of socioeconomic position, but can itself be a determinant of health. Working conditions often exposed those in manufacturing and rural occupations to dangers, illness and injury. Certain trades, such as woolwashing, fellmongering, boiling down and tanning and leather, were characterised as 'noxious'. Poor ventilation, respiratory hazards, too much noise and accidents from unprotected cog wheels, presses, rollers and construction scaffolding were common industrial perils (Fitzgerald, 1987, p.142).

Miners were especially at risk, with certain forms associated with higher mortality from accidents and pulmonary diseases. In Victoria during the twenty five years to 1898, around fifty gold miners were killed and one hundred

injured annually (Fenton, 1901, p.757). While phthisis mortality had declined in Victoria during 1875–1906, mortality among Bendigo miners had increased almost threefold, and deaths from chronic bronchitis fourfold (Cumpston, 1989, pp.162–169).

Late in the century, Factories and Shops Acts began to make greater provisions for cleanliness, ventilation, sanitation and safety. Legislation was most urgently needed in Victoria by virtue of the colony's large manufacturing sector, and this was passed in 1885, followed by South Australia in 1894, New South Wales and Queensland in 1896, Western Australia in 1897 and Tasmania in 1910 (Ibid, pp.148–149).

SOCIOECONOMIC POSITION

Mortality is closely associated with socioeconomic position. The evidence is now considered to be unequivocal, that as a rule people in lower socioeconomic positions fare worse health-wise and have higher mortality rates for most major causes of death (Wilkinson & Marmot, 2003).

From the mid-1870s, the Chief Health Officer of Sydney, Dr Dansey warned about worsening ill-health among the working-class residential district of the City, fearing that poverty-bred epidemics would spread across the entire community (Mayne, 1982, p.89). Inspection tours of infected working-class districts by sanitary officials during Sydney's smallpox epidemic of 1881 pointed to the connection between poverty, dirt and disease and conditions of 'pestilence and death' (Ibid, p.98).

Was mortality really higher in poorer residential areas? Using a Socioeconomic Index for Areas derived from census information (see Appendix 5), it can be shown that a relationship existed between socioeconomically disadvantaged areas and mortality in 1880s Victoria (Figure 8.13). The mortality rate for the year 1881 in suburbs of Melbourne is higher in areas with lower socioeconomic standing.

A socioeconomic gradient is apparent across Melbourne suburbs, from working-class areas such as Hotham, bordering the railway yards, 'low-lying

and imperfectly drained' Collingwood and Sandridge, to the fashionable, superior middle-class suburbs of Hawthorn and St Kilda.

Figure 8.13: Crude death rate and socioeconomic disadvantage, Melbourne suburbs, 1881
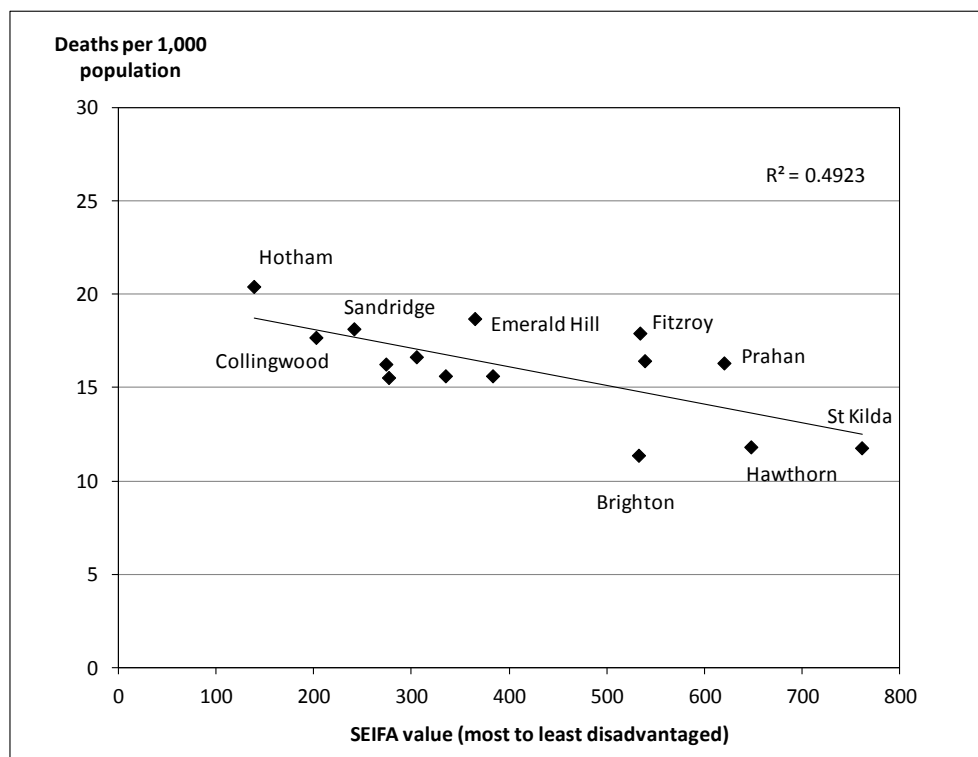
Note: Deaths occurring in three large long-term care institutions have been attributed to the suburbs in which the facilities are located, i.e. Immigrants Home (Emerald Hill), Metropolitan Lunatic Asylum (Kew), Benevolent Asylum (Hotham).

Brighton does better than expected—appearing below the regression line—and with a lower mortality rate than Hawthorn and St Kilda, suburbs with greater socioeconomic advantage. Emerald Hill, Fitzroy and Prahan, on the other hand, fare somewhat worse.

Fricker (in Larson, 1994, pp.210–211) compared the occupational status of Hotham and Hawthorn, two Melbourne suburbs at each end of the social gradient. In 1881, a large proportion of the male labour force in Hotham (North Melbourne) worked as unskilled labourers (21%), compared with only 6% in Hawthorn. Similarly, whereas 15% of the Hawthorn labour force worked in a professional occupation, only 2% of the Hotham labour force did so.

Mortality in advantaged suburbs was consistently less than in disadvantaged suburbs (Figure 8.14). Although mortality declined for all social groups, the differential narrowed from the late 1880s, however it cannot be ruled out that the changing social composition of suburbs after 1881 (this being the census year on which the socioeconomic index is based) may have an effect.

Figure 8.14: Crude death rate in high and low SES Melbourne suburbs, 1880–1906
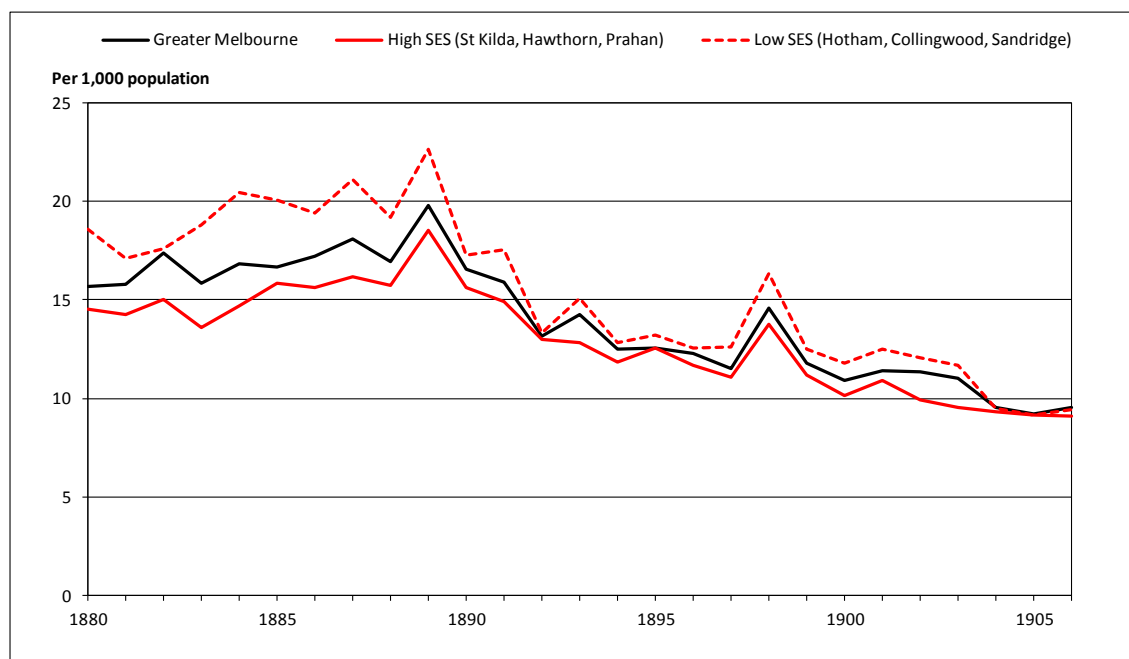
Mein Smith notes that infant mortality in working-class Fitzroy was three times that of suburban Kew, and twice as high in Adelaide's city centre compared with genteel North Adelaide. This disparity appeared in most capital cities, and the operating factor was thought to be families' socioeconomic class and circumstances within houses (Mein Smith, 1997, p.23).

Plague in 1900 Sydney was also class selective, with most cases among males living in the centre of the city, where substandard and insanitary living and working conditions prevailed (Curson, 1985a, pp.144–146).

Research has found a phased transition in English mortality decline, with the pace and timing varying in different occupations and social classes. Infants of professionals and middle classes made the most gains, with lower infant mortality in bigger houses (Mein Smith, p.23). Social class (if English social

classes are synonymous with SES groups) does not appear to have influenced the timing of the decline in Melbourne, with both advantaged and disadvantaged socioeconomic groups exhibiting declines from the late 1880s (Fig 8.13).

Advantaged socioeconomic groups in modern times have more power, resources and opportunities to make advantageous health decisions (WHO, 2008). This was not so in 1880's Melbourne. Money could not buy better health care, since little efficacious clinical care was available. Neither could knowledge lead to better health opportunities; miasma theory still clouded epidemiological thought and medical knowledge among the general populace was minimal. The sanitation movement, however, did correct certain public health practices, although germ theory was some way off.

Although it was known that poor living and working conditions led to poor health and shorter lives, the pathways through which these determinants act are complex and today are still the subject of enquiry.

SUMMARY

Differences in population health are closely linked to degrees of social disadvantage, with greater disadvantage associated with poorer health and a shorter life expectancy. These differences arise because of the conditions in which people are born, live, grow, work and age. This work proposes that social and environmental determinants of health also had a bearing on mortality in nineteenth-century Australia, and acted as causes of the causes of illness and death. Five of these determinants are investigated for their possible effects on mortality.

Increasing urbanisation in Australia and in other developing countries led to public health crises that obligated new methods of disease prevention and health protection. The growth of populations in large urban centres overtaxed public utilities and led to a deteriorating physical environment. Overcrowding facilitated the spread of infectious disease in the absence of adequate urban services and housing, and mortality rose. The disadvantaged were most at risk.

Two corollaries of increasing urbanisation were degradation of the water supply and absent or inadequate sanitation. These were perhaps the weightiest determinants of mortality in nineteenth-century Australia. Their effects were most apparent among infants and children under five years through the devastation caused by water-borne infection and transmission of fecal-oral diseases. The improvement of water and sewerage facilitated better public health in the last decades of the century, and these acted with other determinants to add force to the downward trend in mortality.

Illegitimacy imperilled the life chances of infants. The death rate among infants born outside of marriage at this time was almost three times that of infants born within marriage. Illegitimate infants were at greater risk of death from infectious disease and unsupported mothers also lacked the income, food, shelter and security needed to support their vulnerable babies.

Occupation is also widely considered to be a social determinant of health. In this study the findings on the relationship between occupation and mortality are unclear. Ordering deaths in early-1880s Victoria according to an occupational hierarchy reveals a social gradient in mortality for several classes only; males in the highest occupational classes were an exception, having higher death rates than males in lower occupational classes. Some semi-skilled and manual types of work which are lower in occupational class carried greater health risks.

A social gradient in mortality was evident in 1880s Victoria which is consistent with socioeconomic position. Utilising a socioeconomic index constructed from census data, death rates in Melbourne suburbs were found to be higher in areas of socioeconomic disadvantage.

The findings lend weight to the suggestion that in nineteenth-century Australia, where one lived and worked, and the conditions in which one lived and worked had a role in determining mortality.

Chapter 9

Conclusion

The decline of mortality in Australia in the latter part of the nineteenth century was one of the most striking demographic changes the country has undergone. The fall in the death rate after 1885 was sharp, with reductions in child and infant mortality responsible for much of the decline, although all ages benefited from the gains in life expectancy. Much of the improvement in mortality can be attributed to lower death rates in urban areas from diseases such as gastrointestinal infection and tuberculosis.

This phenomenon and the reasons underlying it, however, are not well understood. The uncertainty stems largely from the absence of evidence; a lack of data on the occurrence of death and its causes during this time. There are also broader uncertainties surrounding the methodological and theoretical approaches to the study of the decline.

This thesis has sought to redress these uncertainties. It has amassed the evidence needed to study the decline through documenting the history of civil registration of death, and through constructing and reporting on a dataset of deaths registered in Australia between 1856 and 1906. It considers the repercussions of this evidence for a number of theories which have sought to explain the reasons for the secular decline in mortality.

The findings answer the three research questions that were posed in the introduction to this thesis.

How were deaths registered in nineteenth-century Australia?

From the European colonisation of Australia in 1788 onward, statistics on deaths were collected by authorities and were returned to the Colonial Office in London. After 1810, ecclesiastical record-keeping served as documentation of births, deaths and marriages. At first Church of England adherents were recorded, and although these records progressively included additional religious denominations as legislation allowed, coverage was partial, and the records provided no legal surety or proof of either birth or death.

Legislation in the Australian colonies to enact civil registration of births, deaths and marriages was first passed in Tasmania in 1838. By 1856, all colonies had Acts in place. Registrars-General were appointed to administer these Acts and, working in conjunction with teams of district Assistant Registrars, were responsible for the registration of deaths which occurred in their jurisdictions. The Registrars-General—and later the Government Statists—routinely published tables of summary information on registered deaths. These abstracts of mortality included information on the sex, age and cause-of-death of persons. It is these abstracts which make possible the analysis of mortality trends.

The Registrars-General believed that death registration had reached reasonable levels of completion within a decade or so of legislation, their confidence aided by the legal prescription that burial required a completed death certificate. Coverage of deaths in remote areas, among infants, and among Aboriginals and other non-European people was thought to be less complete.

The veracity of these abstracts as data sources for demographic analysis relies on their completeness and accuracy. Sufficient information on age or on cause-of-death was sometimes missing, either through omission or through recording errors. Western Australia was especially problematic. The complete absence or poor quality of abstracts for the colony's earlier years meant that an alternative data source was needed for this study.

Some of the details on age published in the abstracts of mortality required reclassification. Summary data on the 1.75 million deaths registered between 1856 and 1906 were then transcribed into an electronic database. Population estimates by sex and age group were constructed, and the resultant estimates of deaths and populations enable the calculation of age-specific and age-standardised mortality rates and other vital statistics.

Recording a cause of death on a death certificate was a requirement of the registration laws. Examining statistics derived from cause-of-death frequencies is the basis for understanding mortality transition in Australia. Certification of cause-of-death by medical practitioners was progressively adopted throughout the colonies and provided greater accuracy for these statistics, although within the limitations of nineteenth-century medical knowledge.

The effective comparison of cause-of-death statistics requires the use of a standard system of disease classification, known as a nosology. The Australian colonies closely followed England in their adoption with Assistant Registrar W. H. Archer circulating Farr's English nosology in Victoria in 1853, and other colonies following suit.

Nineteenth-century cause-of-death statistics, however, have numerous shortcomings. Their quality largely reflected the skill of the doctor who determined a cause of death, and misdiagnosis was not uncommon. Non-medical factors, including the deliberate miscoding of socially sensitive diseases added further compromise. The rapid advance of medical knowledge and emergence of germ theory led to revised classifications and as a consequence, underlying cause-of-death headings and descriptions changed.

Comparison is further complicated by the differential adoption of nosologies by colonies and the variations in details of deaths published by the Registrars in their abstracts of mortality. Compiling longitudinal cause-of-death data for the whole of Australia requires uniting these different classifications and harmonising colonial reporting. To this end, a cause-of-death schema was developed for this work.

What mortality patterns and trends were evident in Australia between 1856 and 1906?

In common with many western European and offshoot countries, the death rate in Australia declined rapidly during the last decades of the nineteenth-century. All-cause mortality had fluctuated considerably from 1856 to 1885, with high but variable age-standardised rates of between 1,300 and 2,000 deaths per 100,000 population. In 1860, all-cause mortality reached a high-point of 2,059 per 100,000 population. Mortality rates were highest among the youngest and oldest age groups—ages 0 and 75 years and over.

A turning point analysis identified that the death rate in Australia exhibited a steady decline after 1885, and with less annual fluctuation. By 1906, all-cause mortality had fallen to below 1,000 deaths per 100,000 population, a decline of one-third over two decades.

Mortality declines after the mid-1880s were most apparent in urban areas, with rural death rates remaining largely unchanged. There was high seasonal variation in death rates, with summer peaks although these gradually disappeared over the course of the second half of the nineteenth century to be replaced by a winter excess.

Large declines in death rates occurred among infants and children under five. In the early 1860s, under-five deaths comprised half of all deaths, but by the early 1900s had fallen to 30 per cent. The infant mortality rate declined after 1885, slowly at first and rapidly after 1903, this being attributed to lower post-neonatal death rates and reflecting the operation of environmental factors. Mortality among children aged 1–4 years fell by a remarkable 80% between 1860 and 1906. Under-five mortality was higher in urban regions and in the summer months when food- and water-borne infectious diseases took their toll.

The decline in mortality rates in all age groups translated into increasing life expectancy—in 1856–60, life expectancy at birth for males was 43.1 years and for females 46.0 years, rising to 54.3 and 58.0 by 1901–06. Most of the increase occurred from 1881–90, and over half of the gains can be attributed to reductions in under-five mortality.

Causes of deaths in the colonies were classified using different nosologies at different times. Having harmonised these through the development of one cause-of-death schema, this work then examined trends and patterns in a number of leading causes of death. These causes of death include diphtheria, whooping cough, measles, scarlet fever, tuberculosis, gastrointestinal infections, typhoid fever, cancer, circulatory diseases, respiratory diseases, digestive diseases, maternal mortality and external causes of death.

Changes in leading causes of death illustrate the extent of the mortality transition in Australia. In 1865 and again in 1885, gastrointestinal infection affecting infants was the most common cause of death, but by 1905 circulatory disease, a chronic cause, headed the list of causes. Deaths from tuberculosis affected mostly young adults in urban areas, and comprised one-tenth of total deaths during the period. TB death rates rose steadily until the mid-1880s, and fell rapidly thereafter, with the onset of the decline in Australia occurring simultaneously in many age groups.

The cause-of-death data allow for the measurement of epidemiological transition—the replacement of infectious diseases by chronic diseases over time as mortality declined. To judge whether such a shift occurred in Australia between 1856 and 1906, cause-of-death summary statistics were redistributed into a broad classification comprised of three categories: communicable diseases, non-communicable diseases and injuries. Death rates from each of these broad causes, communicable, non-communicable and injury, declined in Australia after the mid-1880s.

Although there were changes in the contribution of specific causes of death such as gastrointestinal infections and tuberculosis, the relative contribution to total mortality of each of the three categories—communicable diseases, non-communicable diseases and injuries—remained largely unchanged. To that end, there was little evidence of epidemiological transition during the period 1856–1906.

What factors may have had a bearing on mortality patterns and trends in Australia between 1856 and 1906?

The causes underlying nineteenth-century declines in mortality continue to be debated among historical demographers, historians and economists. The repercussions of Australian mortality data for a number of influential demographic perspectives which seek to explain the reasons for the secular decline in mortality were assessed in this work.

Economic factors which improved living standards through better nutrition and a cleaner environment were the focus of Thomas McKeown's interpretation of the reasons for nineteenth-century population growth. The interpretation has attracted criticism, particularly with respect to the methods used to measure both the decline in mortality and improved nutritional status, and indeed of the assertion that improved nutrition was an underlying cause of the decline. Neither does Australian data support the McKeown interpretation. Poor nutrition was not prevalent in Australia, and cannot explain under-five mortality from gastrointestinal infection. Other salient factors better explain the decline in mortality rates and the increase in life expectancy in Australia.

Mortality decline is held to be the major determinant of lower fertility in classical demographic transition theory. The stagnant death rate between 1860 and 1885, and the decline in fertility apparent since at least the 1860s and would seem to exclude mortality as a determinant of fertility decline in Australia before 1885. However, a declining death rate and a further fall in fertility following a period of little change indicate that mortality played a role in fertility decline after 1885.

A theory of the social determinants of health holds that differences in population health are closely linked to degrees of social disadvantage arising from the conditions in which people are born, live, grow, work and age. This work proposes that social and environmental factors also operated to determine mortality in nineteenth-century Australia. Examining a number of indicators of socioeconomic inequality finds that:

- As urbanisation increased in the absence of adequate urban services and housing, so did mortality. The disadvantaged were most at risk

- Poor quality water and absent or inadequate sanitation were determinants of mortality. Their improvement added force to the downward trend in mortality

- Illegitimacy imperilled the life chances of infants

- Employment provided material resources and gave social status and influence. Ordering deaths according to an occupational hierarchy reveals a social gradient in mortality, although not for the highest classes. Some types of work carried greater health risks

- A social gradient in mortality operated which is consistent with socioeconomic position. Utilising a socioeconomic index constructed from census data, death rates in Melbourne suburbs were found to be higher in areas of socioeconomic disadvantage.

The findings suggest that in nineteenth-century Australia it was where one lived, and the conditions in which one lived that affected life chances.

What this thesis adds

This is an original work in the field of historical demography, describing for the first time a national picture of mortality and causes of death in nineteenth-century Australia. It adds to previous studies which relate to single cities, or to a colony.

This work has presented a history of death registration in all Australian colonies, the process of death coding, and the publication of statistics on deaths by colonial Registrars and statistical agencies.

The colonial Registrars' abstracts of mortality from 1856 to 1906 have been transcribed into an electronic database, and a full assessment of the availability, validity and completeness of colonial mortality data has been undertaken. Data were reconstructed and recoded where necessary. This was particularly necessary in order to compare cause-of-death data over time and across colonies.

This thesis has assessed and explained the extent of epidemiological transition in Australia between 1856 and 1906. It proposed that social and environmental factors were important determinants of mortality during this period, and assessed this proposal through examining indicators of inequality in death rates.

Suggested work for the future

Historical demography in Australia remains a fertile field of enquiry. Although this work begins to fill the knowledge gap on mortality and the early years of the epidemiological transition, little is known about the pre-registration era. For this period, new data sources are needed. Family reconstitution and ecclesiastical records have been successfully used for this purpose in England (Wrigley & Schofield, 1989; Wrigley *et al.*, 2005). Several Australian studies have employed similar methods for some jurisdictions (Curson, 1978; Bradley *et al.*, 2010). A cohort study of early convict and settler arrivals might also provide a demographic history of the decades immediately following colonisation in 1788.

Much insight will be gained through the study of smaller geographic areas and specific population groups. Microanalysis will further reveal the multi-faceted nature of the transition, since disease environments often differed markedly between colonies and within large cities (Woods, 2000). The rural-urban mortality differential also bears further examination. The regular reporting of vital statistics of large cities by the Registrars offers much promise, data-wise.

The transition for some populations such as Indigenous Australians has unfolded very differently to that of white Europeans. Again, family reconstitution can be used to further understand the mortality experience of Australia's original inhabitants (McCalman *et al.*, 2009, 2011).

The final chapter in this work moved beyond describing patterns and trends and focussed on measuring mortality differentials. Further descriptions of these inequalities would illuminate the social, economic and demographic processes which determined the mortality decline in Australia.

Appendix 1: Methods and conventions

AGE-STANDARDISATION

Age-standardisation is a method of minimising the influence of age when comparing populations with different age structures — either different populations at one time or the same population at different times (Shryock & Siegel, 1976).

Two different methods of age-standardisation can be used: direct and indirect. Direct age-standardisation is generally used when the populations under study are large and the age-specific rates are reliable:

$$m_1 = \frac{\sum m_a P_a}{P} \times 1{,}000$$

where $m_a = \frac{d_a}{p_a}$ = age-specific death rate,

P_a represents the standard population at each age,

and P represents the total standard population.

Unless otherwise stated, all rates presented throughout this work are age-standardised.

The 1881 total Australian census population has been used as the standard population in this work, since it represents a population from the middle of the time period 1856–1906 and has an age distribution similar to the populations under study (Shryock & Siegel, 1976, p.242, and see Table A1.1). The same

population has been used for standardising males and females to allow valid comparison of age-standardised rates between the sexes.

Table A1.1: Age composition of populations used for age-standardisation

Age group	1881 total Australian census population	2001 total Australian estimated resident population
0	67,023	253,031
1–4	251,733	1,029,326
5–9	287,601	1,351,664
10–14	268,622	1,353,177
15–19	240,220	1,352,745
20–24	226,159	1,302,412
25–29	170,855	1,407,081
30–34	138,768	1,466,615
35–39	131,428	1,492,204
40–44	121,687	1,479,257
45–49	105,972	1,358,594
50–54	87,735	1,300,777
55–59	54,311	1,008,799
60–64	45,193	822,024
65–69	25,379	682,513
70–74	16,555	638,380
75+	13,376	1,114,641
Total	**2,252,617**	**19,413,240**

Source: Vamplew, 1987; ABS, 2003.

Selected figures in this work use the notation 'ASR' to indicate that rates are age-standardised.

BIRTH RATE

The number of live births occurring among a population during a given period per 1,000 mid-year total population during the same period.

BOURGEOIS-PICHAT MEASURE OF INFANT MORTALITY

The biometric measure of infant mortality developed by French demographer M. Bourgeois-Pichat (1951) can be used to determine whether infant deaths are under-registered relative to births.

Bourgeois-Pichat distinguished between endogenous infant deaths (those associated with congenital mortality and the trauma of birth) and exogenous deaths (those attributed to the post-natal environment, especially infections).

The model relies on several features of infant mortality: that almost all endogenous mortality occurs within the first 30 days of life, and that cumulative exogenous mortality is proportional to [log $(x + 1)$]^3, where x is the age in days. The model relates cumulative mortality rate at ages under 365 days (y) to age in days (x).

Bourgeois-Pichat noted that a straight line regression equation can be fitted to the relationship between y and x for deaths at age over 30 days (i.e. postneonatal mortality), once x had been transformed (Woods 2000; Woods, 2009),

$$y = b[\log(x + 1)]^3 + a$$

The constant a or the intercept ($x=0$) of the regression line indicates the level of mortality at birth, and hence provides an estimate of the level of endogenous mortality.

Since the infant mortality rate is composed of either endogenous or exogenous mortality, the latter can then be found by subtraction. Bourgeois-Pichat concluded that if the curve fitted to observations is not linear, or if a is unreasonably low based on levels found in good quality data, there are grounds for believing that the registration system may be defective.

DEATH RATE OR MORTALITY RATE

An estimate of the proportion of a population that dies during a specified period.

In this work, a number of death rates are used. A full discussion of the deaths and population data used in this work to calculate death rates can be found in Chapter 3.

Age-specific death rate

The number of deaths in a specified age group during a year per 100,000 mid-year population in that age group.

Age-standardised death rate

See 'Age-standardisation'.

Crude death rate

The number of deaths during a year per 100,000 mid-year total population.

Selected figures in this work use the notation 'CDR' to indicate that rates are crude rates.

Neonatal mortality rate

The number of deaths of infants under 28 days of age in a given year per 1,000 live births in that year.

Post-neonatal mortality rate

The annual number of deaths of infants aged 28 days to 1 year per 1,000 live births in a given year.

Infant mortality rate

The number of deaths of infants under age 1 per 1,000 live births in a given year.

Under-5 mortality rate

The number of deaths of children aged under 5 years per 1,000 live births in a given period.

Maternal mortality ratio (MMR)

The number of women who die as a result of pregnancy and childbirth complications per 100,000 live births in a given year.

Maternal mortality rate

The number of women who die as a result of pregnancy and childbirth complications per 100,000 women of reproductive age in a given year.

GENERAL FERTILITY RATE (GFR)

A more refined measure of fertility than the crude birth rate (Last, 1988). It is the number of live births in a year per 1,000 women aged 15–44.

JOINPOINT ANALYSIS OF MORTALITY TRENDS

Joinpoint regression analysis can be used to test whether a multi-segmented line is a significantly better fit to a set of data than a straight or less-segmented line (McNeill *et al.*, 1977; Wolleswinkel-van den Bosch *et al.* 1998). Joinpoint analysis involves fitting a series of joined straight lines on a log scale to trends in data. Line segments are joined at points called joinpoints. Each joinpoint denotes a statistically significant change in trend.

Once line segments are established, the estimated percent annual change (PAC) is used to describe and test the statistical significance of the trends in the model. Testing the hypothesis (two-sided p value = 0.05) that the PAC is equal to zero is equivalent to testing the hypothesis that the trend in rates is neither increasing nor decreasing.

In this work, joinpoints represent the years in which a significant change in a trend of mortality took place. The model requires age-specific deaths and person-years for each year of interest—here, for the years 1856 to 1906—as well as a standardising population. The 1881 total Australian census population was retained for this purpose.

The joinpoint software used in this work is an unpublished R program available from the author. It was written by Rasmus Hoffmann and Caspar Looman from Erasmus University, the Netherlands, for the AMIEHS (Avoidable Mortality in the European Union) Project (London School of Hygiene and Tropical Medicine, 2009). It is similar in its function to the National Cancer Institute's Joinpoint Regression Program (NCI, 2014; Kim, Fay, Feuer & Midthune, 2000).

LIFE EXPECTANCY

Life tables are perhaps the most widely used method in demographic analysis. They conveniently summarise aspects of the variation of mortality with age (Hinde, 1998).

Period life tables show the mortality and survival experience of people in a specific time period. Period life tables were constructed for this work, with selected results presented to describe the pattern of mortality and survival in Australian colonial populations. Since deaths and population data are available only for aggregated age groups (0, 1–4, 5–9…70–74, 75 and over), abridged life tables were calculated.

This work utilises life table software developed by the United Kingdom Office for National Statistics (ONS, 2003, 2010) (Table A1.2). The software is based Chiang's long-established and widely used method of constructing abridged life tables (Chiang 1978, 1984).

Table A1.2: Calculation of abridged life table, by Chiang method

	x	n	a_x	pop	death	M_x	q_x	p_x	l_x	d_x	L_x	T_x	e_x
<1	0	1	0.3	291,382	26216	0.089971	0.0846407	0.915359	100000	8464	94075	5802203	58.02
1-4	1	4	0.5	1,057,620	8857	0.008375	0.0329473	0.967053	91536	3016	360112	5708128	62.36
5-9	5	5	0.5	1,339,647	2851	0.002128	0.0105836	0.989416	88520	937	440258	5348016	60.42
10-14	10	5	0.5	1,269,545	2334	0.001838	0.0091487	0.990851	87583	801	435913	4907758	56.04
15-19	15	5	0.5	1,180,346	3396	0.002877	0.0142823	0.985718	86782	1239	430811	4471845	51.53
20-24	20	5	0.5	1,106,979	4265	0.003853	0.0190809	0.980919	85542	1632	423632	4041034	47.24
25-29	25	5	0.5	1,004,239	4834	0.004814	0.0237831	0.976217	83910	1996	414562	3617402	43.11
30-34	30	5	0.5	860,278	4952	0.005757	0.0283747	0.971625	81915	2324	403762	3202840	39.10
35-39	35	5	0.5	741,155	5340	0.007205	0.0353900	0.964610	79590	2817	390910	2799077	35.17
40-44	40	5	0.5	607,520	4854	0.007990	0.0391692	0.960831	76774	3007	376350	2408168	31.37
45-49	45	5	0.5	471,435	4242	0.008998	0.0440013	0.955999	73766	3246	360718	2031818	27.54
50-54	50	5	0.5	359,921	3937	0.010939	0.0532368	0.946763	70521	3754	343217	1671100	23.70
55-59	55	5	0.5	284,983	4380	0.015370	0.0740054	0.925995	66766	4941	321479	1327882	19.89
60-64	60	5	0.5	233,596	5532	0.023684	0.1117989	0.888201	61825	6912	291846	1006403	16.28
65-69	65	5	0.5	188,340	7010	0.037219	0.1702534	0.829747	54913	9349	251193	714557	13.01
70-74	70	5	0.5	121,368	7542	0.062141	0.2689265	0.731074	45564	12253	197187	463364	10.17
75+	75	16	0.5	117,389	14691	0.125145	1.0000000	0.000000	33311	33311	266177	266177	7.99

Source: ONS, 2010.

The following functions are used:

Age specific death rate:

$$_nM_x = \frac{_nD_x}{_nP_x}$$

where $_nD_x$ is the annual number of deaths of people aged between x and $x+n$ and $_nP_x$ is the mid-year population aged between x and $x+n$.

Probability of dying between age x and age $x+n$:

$$_nq_x = \frac{_nM_x}{1 + (1 - a_x)_nM_x}$$

where a_x is the fraction of the last age interval of life,

with a_0 (separation factor for age 0) = 0.3,

$_{\omega}q_{75} = 1$, where ω is the life span limit

Probability of survival at age x:

$$_np_x = 1 - {_nq_x}$$

Population surviving to age x:

$$l_0 = 100{,}000$$

$$l_{x+n} = l_x (1 - {_nq_x})$$

Number of survivors to age x:

$$l_{x+1} = l_x \cdot (1 - {_nq_x}) = l_x \cdot {_np_x}$$

$$\frac{l_{x+1}}{l_x} = {_np_x}$$

Number of deaths at age x:

$$_nd_x = l_x - l_{x+1} = l_x \cdot (1 - {_np_x}) = l_x \cdot {_nq_x}$$

Number of person-years lived at age x:

$$_nL_x = n(l_{x+1} + (a_x \cdot {_nd_x}))$$

$$_{\omega}L_{75} = \frac{l_{75}}{_{\omega}M_{75}}, \text{ where } \omega \text{ is the life span limit}$$

Number of person-years lived after age x:

$$T_x = \sum_{i=x}^{\infty} {}_nL_i$$

where i takes the values x, $x+n$, $x+2n$, and so on

Life expectancy at age x:

$$e_x = \frac{T_x}{l_x}$$

Stochastic variation and statistical significance

Death rates and life expectancies calculated in this work are subject to stochastic or random variation. To make inferences about statistically significant differences in life expectancies, variances and standard errors can be calculated to produce 95% confidence intervals (Chiang, 1984; ONS, 2010).

Variance of life expectancy:

$$S^2{}_{e_i} = \frac{1}{l^2{}_i} \sum_{j=i}^{w=1} l^2{}_j \left[(1 - a_j)n_j + e_{j+1}\right] S^2{}_{p_j}$$

where the variance of quantity p is:

$$S^2{}_{p_j} = \frac{q^2{}_j p_j}{D_j}$$

95% confidence interval:

$$e_i \pm 1.96\, S_{e_i}$$

where S_{e_i} is the standard error of e_i given by $\sqrt{S^2{}_{e_i}}$

Often, deaths in large populations produce narrow confidence intervals, and stochastic variation can be ignored. To determine the size of confidence intervals for the populations in this work, a sensitivity analysis was undertaken (Table A1.3).

Table A1.3: Reference results for life expectancy at birth, with 95% confidence interval, by population size

Population	Population size	Lower confidence limit	Upper confidence limit	Width of confidence interval
Western Australia, Females, 1861–70	77,401	52.00	55.56	3.56
Western Australia, Females, 1871–80	109,439	50.84	53.21	2.37
Western Australia, Females, 1891–00	384,924	49.32	50.58	1.26
Western Australia, Females, 1901–06	543,910	55.09	56.21	1.12
Victoria, Males, 1851–60	1,961,785	40.51	41.10	0.59
Victoria, Males, 1871–80	4,247,571	46.36	46.72	0.36
Australia, Females, 1871–80	8,772,323	49.37	49.63	0.26
Australia, Females, 1891–00	16,325,737	54.68	54.87	0.19

As population size increases, the width of the confidence interval decreases. For life expectancies calculated from populations of less than 70,000, the width of the confidence interval can be expected to be in excess of 4 years. It is unlikely that life expectancies with confidence intervals of this size would allow for the inference of significant difference.

In this work, life expectancies for populations under 70,000 were not calculated. Confidence intervals were calculated for all other life expectancies at birth. All inferences of difference in life expectancies are statistically significant unless otherwise noted.

Age decomposition of differences in life expectancy

Changes in life expectancy between two time periods can be decomposed according to the contributions of mortality decline in various age groups (Arriaga, 1984; Preston, Heuveline & Guillot, 2001, pp.64–65).

The total effect of a difference in mortality rates between exact ages x and $x+n$ on life expectancy at birth is given by:

$$\text{change } (x,\, x+n) = \frac{l_x^1}{l_0^1} \cdot \left(\frac{{}_nL_x^2}{l_x^2} - \frac{{}_nL_x^1}{l_x^1} \right) + \frac{T_{x+n}^2}{l_0^1} \cdot \left(\frac{l_x^1}{l_x^2} - \frac{l_{x+n}^1}{l_{x+n}^2} \right)$$

where l_x, ${}_nL_x$ and T_x are life table functions, and where the superscripts 1 and 2 refer to time 1 and 2.

For the open-ended age interval in the life table, the following equation is applied:

$$\text{change } (x,\ x{+}n) = \frac{l_x^1}{l_0^1} \cdot \left(\frac{T_x^2}{l_x^2} - \frac{T_x^1}{l_x^1} \right).$$

POPULATION ESTIMATES

To calculate death rates using the annual numbers of deaths tabulated in the abstracts of mortality, mid-year population estimates are required. A discussion of the construction of the population estimates used in this work can be found in Chapter 3.

In addition, the following statistical techniques were employed:

Redistribution of unknown ages

Populations or deaths with unknown ages have been proportionately distributed over all age groups, such that

$$\frac{\sum_0^x P_a + P_u}{\sum_0^x P_a} \times P_a$$

where P_a represents the number reported at each age and P_u the number whose age was not reported. The arithmetic distribution of unknown ages is here assumed to be the same as those of known age.

Karup-King coefficients

Interpolation infers intermediate values in a given series of data through the use of a mathematical formula or graphic procedure (Shryock & Siegel, 1976). Formulas for interpolation can be expressed in terms of coefficients or multipliers which can be applied to given data.

The Karup-King Third-Difference formula provides a set of multipliers for the sub-division of grouped data. In this work, these multipliers have been used to subdivide population estimates given in an age group 0–4 years into single years of age, and then to recombine these to provide population estimates for ages 0 and 1–4.

Given data for three 5-year age groups G_1, G_2 and G_3, the following multipliers were used to subdivide the groups into fifths:

Interpolated subgroup	Coefficients to be applied to:		
	G_1	G_2	G_3
First panel			
First fifth of G_1	+0.344	-0.208	+0.064
Second fifth of G_1	+0.248	-0.056	+0.008
Third fifth of G_1	+0.176	+0.048	-0.024
Fourth fifth of G_1	+0.128	+0.104	-0.032
Last fifth of G_1	+0.104	+0.112	-0.016
Middle panel			
First fifth of G_2	+0.064	+0.152	-0.016
Second fifth of G_2	+0.008	+0.224	-0.032
Third fifth of G_2	-0.024	+0.248	-0.024
Fourth fifth of G_2	-0.032	+0.224	+0.008
Last fifth of G_2	-0.016	+0.152	+0.064
Last panel			
First fifth of G_3	-0.016	+0.112	+0.104
Second fifth of G_3	-0.032	+0.104	+0.128
Third fifth of G_3	-0.024	+0.048	+0.176
Fourth fifth of G_3	+0.008	-0.056	+0.248
Last fifth of G_3	+0.064	-0.208	+0.344

Source: Shryock & Siegel, 1976.

Karup-King-Newton quadratic interpolation

'Smoothing' is type of interpolation designed to obtain a smooth series of values from an irregular series of observed values. The smoothed series is taken to represent some underlying law governing the behaviour of the observed data. Smoothing is commonly used to adjust series on the age distribution of population and deaths.

Census data on age distribution of population may have errors due to inaccurate reporting of ages, such as digit preference, or under-enumeration.

In this work, some census results published by single years of age—1891 and 1901 for New South Wales, Western Australia and Tasmania; 1876, 1881, 1891 and 1901 for South Australia—show evidence of digit preference.

The software program AGESMTH utilising the Karup-King-Newton quadratic interpolation (US Census Bureau, 2014), has been used to smooth irregular census data.

RATE RATIO

The ratio of two rates, comparing the incidence rates of events occurring at any given point in time.

Appendix 2: Colonial Registrars-General and Government Statisticians

NEW SOUTH WALES

Registrar-General

1856–1864	Christopher Rolleston
1864–1870	Theodore James Jaques
1870–1890	Edward Grant Ward
1890–1896	Charles Hart Townley Pinhey
1896–1898	Alfred Parry Long
1898–1927	William Gordon Hayes-Williams

Government Statistician

1886–1905	Timothy Augustine Coghlan
1905–1906	William Henry Hall (acting)
1906–1908	Henry Charles Lennox Anderson

VICTORIA

Registrar-General

1853	William Henry Archer (acting)
1854–1859	Envidale Savage Norman Campbell
1859–1874	William Henry Archer
1874–1887	Richard Gibbs
1887–1892	Henry Krone

| 1892–1896 | Henry Colden Antill Harrison |
| 1896–1909 | Thomas Byrne |

Government Statistician

1874–1895	Henry Heylyn Hayter
1895	Evans Frederick Owen (acting)
1896–1903	James Jemison Fenton
1903–1905	William McLean
1906–1908	Edwin Tiptree Drake

QUEENSLAND

Registrar-General

1860	Robert Creyke (acting)
1860–1867	Frederick Orme Darvall
1867–1870	Frederick Rawlins
1870–1875	Henry Scott
1875–1883	Henry Jordan
1883–1898	William Theophilus Blakeney
1898–1903	Joseph Hughes
1904	Richard Baron Howard

Registrar-General and Government Statistician

| 1904–1914 | Thornhill Weedon |

WESTERN AUSTRALIA

Registrar-General

1841–1854	George Frederick Stone
1854–1856	Charles Scholl
1857–1861	Alfred Durlacher
1863–1870	William H. Knight
1871–1872	Edward Locke Courthope
1876–1881	Laurence Stirling Eliot
1881–1886	Charles Hippuff Clifton
1886–1890	Godfrey Charles Knight

1889–1890   Walter Augustus Gale (acting)

Registrar-General and Government Statistician

1890–1918   Malcolm Alexander Clement Fraser

SOUTH AUSTRALIA

Registrar-General

1842–1847   Charles Sturt

1847–1852   Boyle Travers Finniss

1852–1856   Robert Richard Torrens

1856–1885   John Fullerton Cleland

1885–1891   Horatio Thomas Whittell

1891–1906   George Hamilton Ayliffe

1906–1908   John Ambrose Plunkett

Government Statistician

1860–1879   Josiah Boothby

1879–1882   George Spiller Wright (acting)

1882–1890   Henry James Andrews

1890–1916   Lionel Henry Sholl

TASMANIA

Registrar-General

1838–1840   Charles Bethel Lyons

1840–1846   John Abbott

1846–1847   Chester Eardley-Wilmot

1847–1856   John Abbott

1857–1860   William M. Sorell

1860–1866   John Aston Watkins

1866–1881   Henry John Buckland

Government Statistician

1867–1882   Edwin Craddock Nowell

Registrar-General and Government Statistician

1882–1918   Robert Mackenzie Johnston

Appendix 3: Colonial Health and Medical Administrators

NEW SOUTH WALES

Government Medical Advisor

(and from 1882, President of the Board of Health; and from 1896, Chief Medical Officer)

1856–1866	Richard Greenup
1867–1875	Edward Samuel Pickard Bedford
1876–1882	Haynes Gibbes Alleyne
1882–1885	Charles Kinnaird Mackellar
1885–1889	Henry Normand MacLaurin
1889–1892	Frederic Norton Manning
1893–1896	Thomas Peter Anderson Stuart
1896–1913	John Ashburton Thompson

Chief Health Officer, City of Sydney

1857–1859	Isaac Aaron
1859–1870	Henry Graham
1870–1888	George Frederick Dansey
1888–1890	William Rudolph Clay
1890–1900	Devereaux Gwynne-Hughes
1900–1901	Cyril Ernest Corlette

Medical Officer, Metropolitan Health District

1898–1913	William George Armstrong

VICTORIA

President, Central Board of Health

1855–1879   William McCrea

1879–1884   Richard Youl

1884–1890   Arthur Purssell Akehurst

Chairman, Board of Public Health

1890–1894   Charles Alfred Topp

1894–1904   Dan Astley Gresswell

1904–1909   William Perrin Norris

Health Officer, City of Melbourne

1868–1885   Tharp Mountain Girdlestone

1885–1912   James Jamieson

QUEENSLAND

President, Central Board of Health

From 1865 this position was held by either the Premier or the Colonial Secretary

Health Officer, City of Brisbane

1853–1888   William Hobbs

1887–1891   Joseph Bancroft

1894–1901   Peter Bancroft

1901–1902   Arthur Charles Frederick Halford

Commissioner of Public Health

1901–1909   Nathaniel Burnett Ham

SOUTH AUSTRALIA

Colonial Surgeon

1835–1839   Thomas Young Cotter

1839–1855   James George Nash

1855–1858   William Gosse

1858–1869   Robert Waters Moore

1870–1896   Alexander Stewart Paterson

1896–1915	William Lennox Cleland

President, Central Board of Health

1874–1881	William Christie Gosse
1883–1899	Horatio Thomas Whittell
1899–1929	William Ramsay Smith

Health Officer, Port Adelaide

1849–1878	Handasyde Duncan
1878–1883	Robert Gething
1883–1899	John Tressilian Toll

Health Officer, City of Adelaide

1877–1882	Edward Willis Way
1882–1886	Robert Robertson
1886–1900	John Sprod
1900–1924	Thomas Borthwick

WESTERN AUSTRALIA

Colonial Surgeon

1829–1831	Charles Simmons
1833–1835	Alexander Collie
1835–1847	Joseph Harris
1847–1872	John Ferguson
1872–1895	Alfred Robert Waylen

President, Central Board of Health

1886–1895	Alfred Robert Waylen
1895–1899	Thomas Henry Lovegrove
1900–1906	Ernest Black

Health Officer, Perth

1877–1879	Alfred Robert Waylen
1888–1893	Adam Jameson
1893	Edward Scott
1893–1907	Michael O'Connor

TASMANIA

President, Central Board of Health

1886–1888	Henry Alleyne Perkins
1888–1899	Philip Oakley Fysh
1899–1900	William Moore
1900–1903	George Thomas Collins
1903	John McCall

Health Officer, City of Hobart

1841–1850	Robert Officer
1852–1856	Edward Samuel Pickard Bedford
1856–1860	William Benson
1860–1863	Edward Samuel Pickard Bedford
1863–1874	William Benson
1874–1875	George Washington Turnley
1875–1881	Edward Swarbreck Hall
1881–1895	Edward Owen Giblin
1896–1903	Gregory Sprott

Chief Health Officer

1903–1910	John Simeon Colebrook Elkington

Appendix 4: Nosologies used in colonial Australia

ARCHER'S 1853 NOSOLOGY

Published by Archer (1854, 1856).

I-ZYMOTIC DISEASES

1-Small-Pox, Chicken-Pox
2-Miliaria
3-Measles
4-Scarlatina, Putrid Sore Throat
5-Diphtheria
6-Mumps
7-Hooping-Cough
8-Croup
9-Thrush
10-Diarrhoea
11-Dysentery
12-Cholera
13-Influenza
14-Scurvy, Purpura
15-Ague
16-Remittent Fever, Yellow Fever
17-Typhus
18-Puerperal Fever
19-Pestis
20-Rheumatic Fever
21-Syphilis (Primary), Syphilis (Secondary), Gonorrhoea

22-Erysipelas, Traumatic Erysipelas, Erythema
23-Necusia, Malignant Pustule, Hospital Gangrene, Tetanus, Mildew Mortification
24-Glanders
25-Hydrophobia
26-Porrigo
27-Scabies

2-SPORADIC DISEASES OF UNCERTAIN OR VARIABLE SEAT

28-Haemorrhage
29-Anemia, Chlorosis
30-Dropsy
31-Abscess, Purulent deposit
32-Ulcer, Caries, Fistula
33-Tuberculous Disease
34-Gout
35-Cancer
36-Melanosis
37-Hypertrophy, Tumour, Polypus
38-Atrophy, Emaciation

39-Mortification
40-Catacausis
41-Old Age
42-Debility (from Birth), Premature Birth
43-Malformation
44-Worms

3-SPORADIC DISEASES OF SPECIAL SYSTEMS AND ORGANS

NERVOUS SYSTEM

45-Meningitis, Encephalitis, Cephalitis, Myelitis
46-Hydrocephalus (Acute)
47-Apoplexy
48-Paralysis, Shaking Palsy
49-Delirium Tremens
50-Chorea
51-Mania, Monomania, Dementia, Idiocy
52-Epilepsy, Hysteria
53-Tetanus, Traumatic Tetanus, Cramp
54-Convulsions

55-Neuralgia, Tic Douloureux

56-Ophthalmia or Ophthalmitis

57-Otitis

ORGANS OF CIRCULATION

58-Carditis, Pericarditis,
Endocarditis

59-Disease of the Cardiac Valves

60-Hypertrophy of the Heart,
Atrophy of the Heart

61-Aneurism of the Heart

62-Angina Pectoris

63-Fainting

64-Arteritis, Ossification of
Arteries

65-Phlebitis

66-Varicose Veins

RESPIRATORY ORGANS

67-Laryngitis

68-Laryngismus Stridulus

69-Bronchitis, Bronchorrhoea

70-Pleurisy or Pleuritis,
Empyema, Pneumothorax

71-Congestion of the Lungs,
Pneumonitis or Pneumonia,
Pleuripneumonia

72-Asthma, Emphysema.

73-Phthisis

DIGESTIVE ORGANS

74-Teething

75-Glossitis, Stomatitis, Parotitis

76-Quinsy

77-Pharyngitis

78-Oesophagitis

79-Gastritis

80-Enteritis

81-Peritonitis, Tubercular
Peritonitis

82-Ileus, Intussusception, Lead
Colic

83-Hernia

84-Stricture of

85-Ulceration of, Perforation of

86-Dyspepsia, Pyrosis,
Gastralgia

87-Pancreas Disease, *Spleen
Disease

88-Hepatitis

89-Jaundice, Gall Stones

90-Cirrhosis

91-Fatty Liver

URINARY ORGANS

92-Nephritis

93-Ischuria

94-Diuresis

95-Granular Disease of the
Kidneys or Nephria

96-Diabetes

97-Calculus, Gravel

98-Cystitis, Cystirrhoea

99-Disease of the Prostate Gland

100-Stricture of the Urethra,
Infiltratic Urinae

ORGANS OF GENERATION

101-Orchitis

102-Hysteritis

103-Paramenia

104-Childbirth, Abortion

ORGANS OF LOCOMOTION

105-Arthritis (knee)

106-Chronic Rheumatism

107-Ostitis

108-Exostosis

109-Brittleness of the Bones

110-Softening of the Bone,
Curvature of the Spine

111-Caries

112-Necrosis

INTEGUMENTARY SYSTEM

113-Roseola

114-Urticaria

115-Eczema

116-Herpes

117-Pemphigus

118-Rupia

119-Ecthyma

120-Impetigo

121-Acne

122-Mentagra

123-Lichen

124-Prurigo

125-Psoriasis

126-Pytiriasis

127-Ichthyosis

128-Greek Elephantiasis

129-Lupus

130-Molluscum

131-Yaws

132-Carbuncle, Boil

133-Phlegmon

134-Whitlow

**4-EXTERNAL CAUSES:
POISONING, ASPHYXIA,
INJURIES**

135-Intemperance

136-Starvation

137-Poisoning

138-Scald, Burn

139-Explosion

140-Lightning

141-Hanging

142-Immersion in Nitrogen (or in
any irrespirable gas)

143-Drowning

144-Contusion, Concussion,
Fracture, Dislocation

145-Wound

ARCHER'S 1863 NOSOLOGY

Published by Archer (1863), with revised editions in 1868 and 1878.

I-ZYMOTIC DISEASES

I-1-Miasmatic Diseases

I-1-1-Smallpox

I-1-1a-Chicken pox, etc.

I-1-2-Measles

I-1-3-Scarlatina

I-1-3a-Diphtheria

I-1-4-Quinsy

I-1-5-Croup

I-1-6-Whooping Cough

I-1-7-Typhus (and infantile
   fever)

I-1-8-Erysipelas

I-1-9-Metria

I-1-10-Carbuncle, Boil, etc.

I-1-11-Influenza, Coryza,
   Catarrh, etc.

I-1-12-Dysentery

I-1-13-Diarrhoea

I-1-14-Cholera

I-1-15-Ague

I-1-16-Remittent Fever

I-1-17-Rheumatism

I-1-18-Other Miasmatic Diseases

I-2-Enthetic Diseases

I-2-1-Syphilis

I-2-2-Gonorrhoea, Stricture of
   the Urethra

I-2-3-Hydrophobia

I-2-4-Glanders

I-2-5-Other Enthetic Diseases

I-3-Dietic Diseases

I-3-1-Privation

I-3-2-Want of breast milk

I-3-3-Purpura and Scurvy

I-3-4-Alcoholism: a. Delerium
   Tremens, b. Intemperance

I-3-5-Other Dietic Diseases

I-4-Parasitic Diseases

I-4-1-Thrush

I-4-2-Worms, etc.

I-4-2a-Hydatid

I-4-3-Other Parasitic Diseases

**II-CONSTITUTIONAL
   DISEASES**

II-1-Diathetic Diseases

II-1-1-Gout

II-1-2-Dropsy

II-1-3-Cancer

II-1-3a-Tumour

II-1-4-Noma

II-1-5-Mortification

II-1-6-Other Diathetic Diseases

II-2-Tubercular Diseases

II-2-1-Scrofula

II-2-2-Tabes mesenterica

II-2-3-Phthisis

II-2-3a-Haemoptysis

II-2-4-Hydrocephalus

II-2-5-Other Tubercular Diseases

III-LOCAL DISEASES

**III-1-Diseases of the Nervous
   System**

III-1-1-Cephalitis

III-1-2-Apoplexy

III-1-3-Paralysis

III-1-4-Insanity

III-1-5-Chorea

III-1-6-Epilepsy

III-1-7-Convulsions

III-1-8-Brain disease, etc.

**III-2-Diseases of the Organs of
   Circulation**

III-2-1-Pericarditis

III-2-2-Aneurism

III-2-3-Heart disease, etc.

**III-3-Diseases of the
   Respiratory System**

III-3-1-Laryngitis

III-3-2-Bronchitis

III-3-3-Pleurisy

III-3-4-Pneumonia

III-3-4a-Congestion of the Lungs,
   Pulmonary Apoplexy

III-3-5-Asthma

III-3-6-Lung disease, etc.

**III-4-Diseases of the Digestive
   Organs**

III-4-1-Gastritis

III-4-2-Enteritis

III-4-3-Peritonitis

III-4-4-Ascites

III-4-5-Ulceration of Intestines

III-4-6-Hernia

III-4-7-Ileus

III-4-8-Intussusception

III-4-9-Stricture of Intestines

III-4-10-Fistula

III-4-11-Stomach disease, etc.

III-4-12-Pancreas disease, etc.

III-4-13-Hepatitis

III-4-14-Jaundice

III-4-15-Liver disease, etc.

III-4-16-Spleen disease, etc.

**III-5-Diseases of the Urinary
   Organs**

III-5-1-Nephritis

III-5-2-Ischuria

III-5-3-Nephria

III-5-4-Diabetes

III-5-5-Stone

III-5-6-Cystitis

III-5-7-Kidney disease, etc.

**III-6-Diseases of the Organs of
   Generation**

III-6-1-Ovarian Dropsy

III-6-2-Uterus disease, etc.

III-7-Diseases of the Joints

III-7-1-Arthritis

III-7-1a-Ostitis Periostitis, etc.

III-7-2-Joint disease, etc.

III-8-Diseases of the

Integumentary System

III-8-1-Phlegmon

III-8-2-Ulcer

III-8-3-Skin disease, etc.

IV-DEVELOPMENTAL

DISEASES

IV-1-Developmental Diseases

of Children

IV-1-1-Premature Birth

IV-1-2-Cyanosis

IV-1-3-Spina Bifida

IV-1-4-Other Malformations

IV-1-5-Teething

IV-1-6-Other Developmental

Diseases of Children

IV-2-Developmental Diseases

of Adults

IV-2-1-Paramenia

IV-2-2-Childbirth

IV-2-3-Other Developmental

Diseases of Adults

IV-3-Developmental Diseases

of Old People

IV-3-1-Old Age

IV-4-Developmental Diseases

of Nutrition

IV-4-1-Atrophy and Debility

V-VIOLENCE

V-1-Accident or Negligence

V-1-1-Fractures, Contusions, etc.

V-1-2-Wounds, a. Gunshot, b.

Cuts, etc.

V-1-3-Burns and Scalds

V-1-3a-Sunstroke

V-1-4-Poison

V-1-4a-Bite of Snake or Insect

V-1-5-Drowning

V-1-6-Suffocation

V-1-7-Other Accidents

V-2-Wounds in Battle

V-2-1-Gunshot Wounds in Battle

V-2-2-Sword or Bayonet Wounds

in Battle

V-2-3-Other Wounds in Battle

V-3-Homicide

V-3-1-Murder or Manslaughter

V-4-Suicide

V-4-1-Suicide by Wounds, a.

Gunshot, b. Cut, Stab, etc.

V-4-2-Suicide by Poison

V-4-3-Suicide by Drowning

V-4-4-Suicide by Hanging

V-4-5-Other Suicide

V-5-Execution

V-5-1-Execution by Hanging

Violence (not classed)

Sudden Deaths (cause

unascertained)

Diseases or Deaths not

specified or ill-defined

HAYTER'S 1886 NOSOLOGY

Published by Hayter (1887a, 1887b), with revisions in 1892, and by Long (1898).

**I-SPECIFIC FEBRILE OR
    ZYMOTIC DISEASES**

I-1-Miasmatic Diseases

I-1-1-Smallpox, a. Vaccinated, b.
    Unvaccinated, c. Not stated

I-1-2-Chicken pox

I-1-3-Measles

I-1-4-Epidemic rose rash

I-1-5-Scarlet fever

I-1-6-Typhus

I-1-7-Relapsing fever

I-1-8-Influenza

I-1-9-Whooping Cough

I-1-10-Mumps

I-1-11-Diphtheria

I-1-12-Cerebro-Spinal Fever

I-1-13-Simple Continued Fever

I-1-14-Typhoid, Enteric Fever

I-1-15-Other Miasmatic Diseases

I-2-Diarrhoeal Diseases

I-2-1-Cholera, Sporadic (Simple)

I-2-1a-Cholera, Epidemic
    (Asiatic)

I-2-2-Diarrhoea

I-2-3-Dysentery

I-3-Malarial Diseases

I-3-1-Remittent Fever

I-3-2-Intermittent Fever (Ague)

I-3-3-Other Malarial Diseases

I-4-Zoogenous Diseases

I-4-1-Hydrophobia

I-4-2-Glanders

I-4-3-Splenic Fever

I-4-4-Cow-pox and other effects
    of Vaccination

I-5-Venereal Diseases

I-5-1-Syphilis

I-5-2-Gonorrhoea, Stricture of
    the Urethra

I-6-Septic Diseases

I-6-1-Phagedaena

I-6-2-Erysipelas

I-6-3-Pyaemia, Septicaemia

I-6-4-Puerperal Fever

II-PARASITIC DISEASES

II-1-Thrush

II-2-Other Diseases from
    Vegetable Parasites

II-3-Hydatids

II-4-Other Diseases from Animal
    Parasites

III-DIETIC DISEASES

III-1-Starvation. Want of breast
    milk

III-2-Scurvy

III-3-Intemperance, a. Chronic
    Alcoholism, b. Delerium
    Tremens

**IV-CONSTITUTIONAL
    DISEASES**

IV-1-Rheumatic fever,
    Rheumatism of Heart

IV-2-Rheumatism

IV-3-Gout

IV-4-Rickets

IV-5-Cancer, Malignant Disease

IV-6-Tabes Mesenterica

IV-7-Tubercular Meningitis
    (Acute Hydrocephalus)

IV-8-Phthisis

IV-9-Other forms of
    Tuberculosis, Scrofula, etc.

IV-10-Purpura, Haemorrhagic
    Diathesis

IV-11-Anaemia, Chlorosis,
    Leucocythaemia

IV-12-Diabetes Mellitus

IV-13-Other Constitutional
    Diseases

IV-14-Leprosy

**V-DEVELOPMENTAL
    DISEASES**

V-1-Premature Birth

V-2-Atelectasis

V-3-Cyanosis

V-4-Spina Bifida

V-5-Imperforate Anus

V-6-Cleft Palate, Harelip

V-7-Other Congenital Defects

V-8-Old Age

VI-LOCAL DISEASES

**VI-1-Diseases of the Nervous
    System**

VI-1-1-Inflammation of Brain or
    its Membranes

VI-1-2-Apoplexy

VI-1-3-Softening of Brain

VI-1-4-Hemiplegia, Brain
    Paralysis

VI-1-5-Paralysis (undefined)

VI-1-6-Insanity (General
    Paralysis of Insane)

VI-1-7-Chorea

VI-1-8-Epilepsy

VI-1-9-Convulsions

VI-1-10-Laryngismus Stridulus

VI-1-11-Idiopathic Tetanus

VI-1-12-Paraplegia, Diseases of
    Spinal Cord

VI-1-13-Other Diseases of the
    Nervous System

VI-2-Diseases of the Organs of Special Sense

VI-2-1-Otitis, Otorrhoea

VI-2-2-Epistaxis and Diseases of Nose

VI-2-3-Ophthalmia and Diseases of Eye

VI-3-Diseases of the Circulatory System

VI-3-1-Endocarditis, Valvular Disease

VI-3-2-Pericarditis

VI-3-3-Hypertrophy of Heart

VI-3-4-Angina Pectoris

VI-3-5-Syncope

VI-3-6-Aneurism

VI-3-7-Senile Gangrene

VI-3-8-Embolism, Thrombosis

VI-3-9-Phlebitis

VI-3-10-Varicose Veins

VI-3-11-Other Diseases of the Circulatory System

VI-4-Diseases of the Respiratory System

VI-4-1-Laryngitis

VI-4-2-Croup

VI-4-3-Other Diseases of Larynx and Trachea

VI-4-4-Asthma, Emphysema

VI-4-5-Bronchitis

VI-4-6-Pneumonia

VI-4-6a-Congestion of Lungs

VI-4-7-Pleurisy

VI-4-8-Other Diseases of the Respiratory System

VI-5-Diseases of the Digestive System

VI-5-1-Stomatitis

VI-5-2-Dentition

VI-5-3-Sore throat, Quinsy

VI-5-4-Dyspepsia

VI-5-5-Haematemesis

VI-5-6-Melaena

VI-5-7-Diseases of Stomach

VI-5-8-Enteritis

VI-5-9-Ulceration of Intestine

VI-5-10-Ileus, Obstruction of Intestine

VI-5-11-Stricture or Strangulation of Intestine

VI-5-12-Intussusception of Intestine

VI-5-13-Hernia

VI-5-14-Fistula

VI-5-15-Peritonitis

VI-5-16-Ascites

VI-5-17-Gall-stones

VI-5-18-Cirrhosis of Liver

VI-5-19-Other Diseases of Liver

VI-5-20-Other Diseases of the Digestive System

VI-6-Diseases of the Lymphatic System and Ductless Glands

VI-6-1-Diseases of Lymphatic System

VI-6-2-Diseases of Spleen

VI-6-3-Bronchocele

VI-6-4-Addison's disease

VI-7-Diseases of the Urinary System

VI-7-1-Nephritis

VI-7-2-Bright's Disease (Nephria)

VI-7-3-Uraemia

VI-7-4-Supression of Urine

VI-7-5-Calculus

VI-7-6-Haematuria

VI-7-7-Diseases of Bladder and of Prostate

VI-7-8-Other Diseases of the Urinary System

VI-8-Diseases of the Organs of Generation

VI-8-1-Ovarian Disease

VI-8-2-Diseases of Uterus and Vagina

VI-8-3-Disorders of Menstruation

VI-8-4-Pelvic Abscess

VI-8-5-Perineal Abscess

VI-8-6-Diseases of Testes, Penis, Scrotum, etc.

VI-9-Diseases of Parturition

VI-9-1-Abortion, Miscarriage

VI-9-2-Puerperal Mania

VI-9-3-Puerperal Convulsions

VI-9-4-Placenta Praevia, Flooding

VI-9-5-Phlegmasia Dolens

VI-9-6-Other Accidents of Childbirth

VI-10-Diseases of the Organs of Locomotion

VI-10-1-Caries, Necrosis

VI-10-2-Arthritis, Ostitis, Periostitis

VI-10-3-Other Diseases of the Organs of Locomotion

VI-11-Diseases of the Integumentary System

VI-11-1-Carbuncle

VI-11-2-Phlegmon, Cellulitis

VI-11-3-Lupus

VI-11-4-Ulcer, Bedsore

VI-11-5-Eczema

VI-11-6-Pemphigus

VI-11-7-Other Diseases of the Integumentary System

VII-VIOLENCE

VII-1-Accident or Negligence

VII-1-1-Fracture, Contusions

VII-1-2-Gunshot wounds

VII-1-3-Cuts, etc.

VII-1-4-Burns and Scalds

VII-1-4a-Sunstroke

VII-1-4b-Lightning

VII-1-5-Poison

VII-1-5a-Bite of Snake or Insect

VII-1-6-Drowning

VII-1-7-Suffocation

VII-1-8-Other Accident or Negligence

VII-2-Homicide

VII-2-1-Murder and Manslaughter

VII-2-2-Justifiable Homicide

VII-3-Suicide

VII-3-1-Suicide by Gunshot
Wounds

VII-3-2-Suicide by Cut, Stab

VII-3-3-Suicide by Poison

VII-3-4-Suicide by Drowning

VII-3-5-Suicide by Hanging

VII-3-6-Other Suicide

VII-4-Execution

VII-4-Execution by Hanging

VII-5-Violent Deaths Not
Classed (Open Verdict)

VII-5-1-Found drowned

VII-5-2-Other violent death not
classified

**VIII-ILL-DEFINED AND NOT
SPECIFIED CAUSES**

VIII-1-Dropsy

VIII-2-Debility, Atrophy,
Inanition

VIII-3-Mortification

VIII-4-Tumour

VIII-5-Abscess

VIII-6-Haemorrhage

VIII-7-Sudden Deaths (cause
unascertained)

VIII-8-Not specified or ill-
defined

ROYAL COLLEGE OF PHYSICIANS 1896 NOSOLOGY

Published by the Royal College of Physicians (1896) and Qld RG (1903).

CLASS I-GENERAL DISEASES

Order 1
I-1-1-Small-pox
I-1-2-Cow pox & effect of
 vaccination
I-1-3-Chicken-pox
I-1-4-Measles (Morbilli)
I-1-5-Epidemic Rose Rash,
 German Measles
I-1-6-Scarlet Fever
I-1-7-Typhus
I-1-8-Plague
I-1-9-Relapsing Fever
I-1-10-Influenza
I-1-11-Whooping Cough
I-1-12-Mumps
I-1-13-Diphtheria, Membranous
 Croup
I-1-14-Cerebro Spinal Fever
I-1-15-Simple Continued Fever
I-1-16-Enteric Fever
I-1-17-Beri Beri

Order 2
I-2-1-Asiatic Cholera
I-2-2-Epid. Diarrhoea, Epid. Or
 Zim. Enteritis
I-2-3-Diarrhoea, Choloraic
 Diarrhoea
I-2-4-Dysentery
I-2-5-Malarial Fever

Order 3
I-3-1-Hydrophobia
I-3-2-Glanders, Farcy
I-3-3-Anthrax, Splenic Fever
I-3-4-Tetanus

Order 4
I-4-1-Syphilis
I-4-2-Gonorrhoea, Stricture of
 Urethra

Order 5
I-5-1-Puerperal Septicaemia,
 Saproemia
I-5-2-Puerperal Pyaemia
I-5-3-Puerperal Phlegmasia
 Dolens
I-5-4-Puerperal Fever
I-5-5-Infective Endocarditis
I-5-6-Epidemic Pneumonia,
 Pneumonic Fever
I-5-7-Erysipelas
I-5-8-Septicaemia (not Puerperal)
I-5-9-Pyaemia (not Puerperal)
I-5-10-Phlegmon, Carbuncle (not
 Anthrax)
I-5-11-Phagadaena
I-5-12-Other Septic Diseases

Order 6
I-6-1-Tuberculous Phthisis,
 Pulm. Tuberculosis
I-6-2-Phthisis
I-6-3-Tuberculous Meningitis
I-6-4-Tuberculous Peritonitis
I-6-5-Tabes Mesenterica
I-6-6-Lupus
I-6-7-Tubercle of Other Organs
I-6-8-General Tuberculosis
I-6-9-Scrofula

Order 7
I-7-1-Hydatids
I-7-2-Ankylostoma
I-7-3-Other Animal Parasites
I-7-4-Other Parasites

Order 8
I-8-1-Privation (Starvation, etc.)
I-8-2-Scurvy
I-8-3-Alcoholism, Delerium
 Tremens
I-8-4-Opium, Morphia Habit
I-8-5-Ptomaine Poisoning
I-8-6-Chronic Poisoning (Not
 intentional)

I-8-7-Industrial Poisoning

Order 9
I-9-1-Rheumatic Fever, Acute
 Rheumatism
I-9-2-Rheumatism of the Heart
I-9-3-Chronic Rheumatism
I-9-4-Rheumatic Arthritis,
 Rheumatic Gout
I-9-5-Gout

Order 10
I-10-1-Carcinoma
I-10-2-Sarcoma
I-10-3-Malignant Disease, Cancer

Order 11
I-11-1-Rickets
I-11-2-Purpura
I-11-3-Haemophilia,
 Haemorrhagic Diathesis
I-11-4-Anaemia, Leucocythaemia
I-11-5-Diabetes Mellitus

Order 12
I-12-1-Premature Birth
I-12-2-Congenital Defects
I-12-3-Injury at Birth
I-12-4-Atelectasis
I-12-5-Want of Breast Milk
I-12-6-Teething

CLASS II-LOCAL DISEASES

**II-1-Disease of the Nervous
 System**
II-1-1-Meningitis, Inflammation
 of Brain
II-1-2-Softening of Brain
II-1-3-General Paralysis of Insane
II-1-4-Insanity (not Puerperal)
II-1-5-Chorea
II-1-6-Epilepsy
II-1-7-Convulsions

II-1-8-Laryngismus Stridulus

II-1-9-Locomotor Ataxy

II-1-10-Paraplegia and Diseases of Cord

II-1-11-Neuritis, Periph. And Poly. Neuritis

II-1-12-Brain Tumor (not Specific)

II-1-13-Nervous System, Other Diseases

II-2-Disease of Organs of Special Sense

II-2-1-Otitis, Mastoid Disease

II-2-2-Epistaxis, Nose Disease

II-2-3-Ophthalmia, Eye Disease

II-3-Disease of Heart

II-3-1-Valve Dis., Endocarditis (not Infective)

II-3-2-Pericarditis

II-3-3-Hypertrophy of Heart

II-3-4-Angina Pectoris

II-3-5-Dilation of Heart

II-3-6-Fatty Degeneration of Heart

II-3-7-Syncope, Heart Disease (not Specified)

II-4-Diseases of Blood Vessels

II-4-1-Cerebral Haemorrhage, Emb. Throm.

II-4-2-Apoplexy, Hemiplegia

II-4-3-Aneurism

II-4-4-Senile Gangrene

II-4-5-Embolism, Thrombosis (not Cerebral)

II-4-6-Phlebitis

II-4-7-Varicose Veins

II-4-8-Blood Vessels

II-5-Diseases of the Respiratory Organs

II-5-1-Laryngitis

II-5-2-Membranous Laryngitis (not Diphtheritic)

II-5-3-Croup (not Spasmodic or Membranous)

II-5-4-Larynx, Other Diseases (not Specified)

II-5-5-Bronchitis

II-5-6-Lobar, Croupous-Pneumonia

II-5-7-Broncho, Catarrhal, Lobular, Pneumonia

II-5-8-Pneumonia

II-5-9-Emphysema, Asthma

II-5-10-Pleurisy

II-5-11-Fibroid Disease of Lung

II-5-12-Respiratory Diseases, Other

II-6-Diseases of the Digestive System

II-6-1-Tonsilitis, Quinsy

II-6-2-Mouth, Pharynx, Disease (not Specified)

II-6-3-Gastric Ulcer

II-6-4-Gastric Catarrh

II-6-5-Stomach, Other Diseases (not Malignant)

II-6-6-Enteritis (not Epidemic)

II-6-7-Gastro Enteritis

II-6-8-Appendicitis, Perityphlitis

II-6-9-Hernia

II-6-10-Intestinal Obstruction

II-6-11-Other Diseases of Intestines

II-6-12-Peritonitis (not Puerperal)

II-6-13-Cirrhosis of Liver

II-6-14-Liver and Gall Bladder, Other Diseases

II-6-15-Digestive System, Other Diseases

II-7-Lymphatic Diseases

II-7-1-Spleen Disease

II-7-2-Lymph System, Other Diseases

II-7-3-Thyroid Body Disease

II-7-4-Supra Renal Capsules Disease

II-8-Diseases of Urinary System

II-8-1-Nephritis, Acute

II-8-2-Chronic Bright's Disease, Albuminuria

II-8-3-Calculus (not Biliary)

II-8-4-Bladder and Prostate Disease

II-8-5-Urinary System, Other Diseases

II-9-Diseases of Generative System

II-9-1-Ovarian Tumor (not Malignant)

II-9-2-Other Diseases of Ovary

II-9-3-Uterine Tumor (not Malignant)

II-9-4-Other Diseases of Uterus and Vagina

II-9-5-Disorders of Menstruation

II-9-6-Gener. And Mam. Orgs., Other Diseases

II-10-Accidents of Childbirth

II-10-1-Abortion, Miscarriage

II-10-2-Puerperal Mania

II-10-3-Puerperal Convulsions

II-10-4-Placenta Praevia, Flooding

II-10-5-Pregnancy, Childbirth, Other Accidents

II-11-Diseases of Locomotive System

II-11-1-Caries, Necrosis

II-11-2-Arthritis, Periostitis

II-11-3-Locomotor System, Other Diseases

II-12-Skin Disease

II-12-1-Ulcer, Bedsore

II-12-2-Eczema

II-12-3-Pemphigus

II-12-4-Skin Diseases, Other

CLASS III-OTHER SPECIFIED DISEASES

CLASS IV-ILL-DEFINED AND NOT SPECIFIED DISEASES

IV-1-Atrophy, Debility

IV-2-Old Age

IV-3-Dropsy, Ascites, Anasarca

IV-4-Tumor

IV-5-Abscess

IV-6-Haemorrhage

IV-7-Sudden (Cause
   unascertained)

IV-8-Other Ill-defined Causes

IV-9-Causes not Specified

CLASS V-VIOLENT DEATHS

V-1-Accident or Negligence

V-1-1-In Mines and Quarries

V-1-2-Railways

V-1-3-Tramways

V-1-4-Other vehicles

V-1-5-Horses, etc.

V-1-6-Ships, Boats, and Docks
   (not Drowning)

V-1-7-Building Operations

V-1-8-Machinery

V-1-9-Weapons and Implements

V-1-10-Burns and Scalds

V-1-11-Poisons, Poisonous
   Vapours

V-1-12-Drowning

V-1-13-Suffocation

V-1-14-Falls (Otherwise)

V-1-15-Weather-Sunstroke and
   Heat Apoplexy

V-1-16-Weather-Lightning

V-1-17-Weather-Other

V-1-18-Snake Bites

V-1-19-Insect Bites

V-1-20-Otherwise, or not stated

V-2-Homicide

V-2-1-Murder

V-2-2-Manslaughter

V-2-3-Justifiable Homicide

V-3-Suicide

V-3-1-Suicide, by Poison

V-3-2-Suicide, by Asphyxia

V-3-3-Suicide, by Hanging or
   Strangulation

V-3-4-Suicide, by Drowning

V-3-5-Suicide, by Firearms

V-3-6-Suicide, by Cutting
   Instruments

V-3-7-Suicide, by Jumping from
   High Places

V-3-8-Suicide, by Crushing

V-3-9-Suicide, by Other Means

V-4-Execution

V-4-1-Execution

BERTILLON 1906 NOSOLOGY

Published by the Commonwealth Bureau of Census and Statistics (1907).

I-GENERAL DISEASES

1 Typhoid Fever
2 Typhus
3 Recurrent Fever
4 Intermittent Fever and Malarial Cachexia
5 Small-Pox
6 Measles
7 Scarlet Fever
8 Whooping Cough
9 Diphtheria and Croup
9a Diphtheria
10 Influenza
11 Military Fever
12 Asiatic Cholera
13 Cholera Nostras
14 Dysentery
14a Epidemic Dysentery
15 Plague
16 Yellow Fever
17 Leprosy
18 Erysipelas
19 Other Epidemic Diseases
20 Purulent Infection and Septicaemia
21 Glanders and Farcy
22 Malignant Pustule and 'Charbon'
23 Rabies
24 Trichinosis, etc.
25 Pellagra
26 Tuberculosis of Larynx
27 Tuberculosis of Lungs
28 Tuberculosis of the Meninges
29 Tuberculosis of the Peritonaeum
30 Pott's Disease
31 Cold Abscess; Symptomatic Abscess
32 White Swellings
33 Tuberculosis of other Organs
34 General Tuberculosis
35 Scrofula
36 Syphilis
37 Blennorrhagia of Adults
38 Gonococcic Diseases of Children under five years of age
39 Cancer, etc., of Mouth
40 Cancer, etc., of the Stomach and Liver
41 Cancer, etc., of the Peritonaeum, the Intestines, and the Rectum
42 Cancer, etc., of the Female Genital Organs
43 Cancer, etc., of the Breast
44 Cancer, etc., of the Skin
45 Cancer, etc., of other Organs
46 Other Tumors (Tumors of the Female Genital Organs excepted)
47 Acute articular Rheumatism
48 Chronic Rheumatism and Gout
49 Scurvy
50 Diabetes
51 Exophthalmic Goitre
52 Addison's Disease
53 Leucaemia
54 Anaemia, Chlorosis
55 Other General Diseases
56 Acute and chronic Alcoholism
57 Lead Poisoning
58 Other chronic Poisonings due to occupations
59 Other chronic Poisonings

II-DISEASES OF THE NERVOUS SYSTEM AND OF THE ORGANS OF SPECIAL SENSE

60 Encephalitis
61 Simple Meningitis
61a Epidemic Cerebro-Spinal Meningitis
62 Progressive Locomotor Ataxia
63 Other Diseases of the Spinal Cord
64 Congestion and Haemorrhage of the Brain
65 Softening of the Brain
66 Paralysis without indicated cause
67 General Paralysis
68 Other forms of Mental Alienation
69 Epilepsy
70 Eclampsia (non-puerperal)
71 Convulsions of children under five years of age
72 Tetanus
73 Chorea
74 Other Diseases of the Nervous System
75 Diseases of the Eyes and their appendages
76 Diseases of the Ear

III-DISEASES OF THE CIRCULATORY SYSTEM

77 Pericarditis
78 Acute Endocarditis
79 Organic Diseases of the Heart
80 Angina Pectoris
81 Diseases of the Arteries, Atheroma, Aneurism, etc.
81a Aneurism
82 Embolism and Thrombosis
83 Diseases of the Veins (Varices, Varicose Ulcer, Haemorrhoids)
84 Diseases of the Lymphatic System
85 Haemorrhage
86 Other Diseases of the Circulatory System

IV-DISEASES OF THE RESPIRATORY SYSTEM

87 Diseases of the Nasal Fossae
88 Diseases of the Larynx
89 Diseases of the Thyroid Body
90 Acute Bronchitis
91 Chronic Bronchitis

92 Broncho-pneumonia

93 Pneumonia

94 Pleurisy

95 Congestion of the Lungs and Pulmonary Apoplexy

96 Gangrene of the Lungs

97 Asthma

98 Pulmonary Emphysema

99 Pulmonary Haemorrhage

99a Other Diseases of the Respiratory System (Consumption excepted)

V-DISEASES OF THE DIGESTIVE SYSTEM

100 Diseases of the Mouth and its associated organs

101 Diseases of the Pharynx

102 Diseases of the Oesophagus

103 Ulcer of the Stomach

104 Gastritis

104a Other Diseases of the Stomach (Cancer excluded)

105 Diarrhoea and Enteritis (children under two years of age only)

105a Diarrhoea and Enteritis, chronic (children under two years of age only)

106 Diarrhoea and Enteritis (children over two years and Adults)

107 Intestinal Parasites

108 Hernia, intestinal Obstruction

109 Other Diseases of the Intestines

110 Icterus gravis

111 Hydatid Tumors of the Liver

111a Hydatids, undefined

112 Cirrhosis of the Liver

113 Biliary Calculi

114 Other Diseases of the Liver

115 Diseases of the Spleen

116 Simple Peritonitis (non-puerperal)

117 Other Diseases of the Digestive System (Cancer and Tuberculosis excepted)

118 Appendicitis and Abscess of the Iliac Fossa

VI-DISEASES OF THE GENITO-URINARY SYSTEM AND ADNEXA

119 Acute Nephritis

120 Bright's Disease

121 Other Diseases of the Kidneys and their Adnexa

122 Calculi of Urinary System

123 Diseases of the Bladder

124 Other Diseases of the Urethra, Urinary Abscess, etc.

125 Diseases of the Prostate

126 Non-veneral Diseases of the Male Genital Organs

127 Metritis

128 Uterine Haemorrhage (non-puerperal)

129 Uterine Tumor (non-cancerous)

130 Other Diseases of the Uterus

131 Cysts and other Ovarian Tumors

132 Other Diseases of the Female genital Organs

133 Non-puerperal Diseases of the Breast (Cancer excepted)

VII-PUERPERAL CONDITION

134 Accidents of Pregnancy

134a Illegal Operations

135 Puerperal Haemorrhage

136 Other Accidents of Childbirth

137 Puerperal Septicaemia

138 Puerperal Albuminuria and Eclampsia

139 Puerperal Phlegmasia alba dolens

140 Other Puerperal Accidents, Sudden Death

141 Puerperal Diseases of the Breast

VIII-DISEASES OF THE SKIN AND OF THE CELLULAR TISSUE

142 Gangrene

143 Carbuncle

144 Phlegmon, Acute Abscess

145 Other Diseases of the Skin and Adnexa

IX-DISEASES OF THE ORGANS OF LOCOMOTION

146 Non-tuberculous Diseases of the Bones

147 Arthritis and other Diseases of the Joints (Tuberculosis and Rheumatism excepted)

148 Amputation

149 Other Diseases of the Organs of Locomotion

X-MALFORMATIONS

150 Congenital Malformations (exclusive of Still-born)

XI-INFANCY

150a Malformations, Hydrocephalus

151 Congenital Debility, Icterus, and Scleroma, of children under three months of age

151a Premature Birth

152 Other Diseases Peculiar to Infancy

153 Want of Care (Infants)

XII-OLD AGE

154 Senile Debility

XIII-VIOLENCE

155 Suicide by Poison

156 Suicide by Asphyxia

157 Suicide by Hanging or Strangulation

158 Suicide by Drowning

159 Suicide by Firearms

160 Suicide by Cutting Instruments

161 Suicide by Precipitation from a Height

162 Suicide by Crushing

163 Other Suicides

Appendix 5: Constructing a Socioeconomic Index for colonial Melbourne

Historic analyses of socioeconomic inequalities in Australia are hampered by a lack of data on individuals and their characteristics. One method to circumvent this is to construct a Socioeconomic Index for Areas (SEIFA), using relevant information collected in censuses and other administrative data.

A SEIFA can be used to rank geographic areas according to the socioeconomic characteristics shared by their residents. The assumption that follows is that all people living in these areas, regardless of their circumstances, will be assigned an average SEIFA value. SEIFAs have been used recently to classify populations living in different geographic areas (ABS, 2008c). There is no intrinsic reason why a SEIFA cannot be constructed and used for historical analysis, if suitable data can be sourced.

No single census collected information on all Australians prior to Federation in 1901. Colonial censuses, however, were conducted regularly and often concurrently (Table 3.4). In Victoria they were undertaken decennially from 1861. The information collected in a census provides the basis for the construction of a SEIFA, however, since social conditions changed rapidly, the SEIFA can only be used for a small number of years surrounding the reference year.

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Small-area mortality data are readily available for Melbourne and suburbs. These were first published in the 1860 *Victorian Government Gazette*, and are now available on-line (gazette.slv.vic.gov.au). Since the 1881 Victorian census represents a convenient point in the study period, this will provide the underlying socioeconomic data (HCCDA, 2014).

Table A5.1: Indicators for constructing a SEIFA for Melbourne and suburbs, 1881

	Property value per ratepayer (£)	Persons per Room	Houses w/ more than 6 rooms (%)	Ratio Brick/ Stone to Wood etc.	Ratio English to Irish-born	Ratio Church of England to Catholic	Depend ency ratio (0-14 & 60+/15-59)	Read & write to Read only & Cannot read (Age 15+)	Ratio Married to Unmarr ied & widow (Age 21+)	Sick- ness and Infirm (per 1000 pop.)
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)	(i)	(j)
Brighton	465.78	0.92	36.3	0.79	2.21	3.64	0.43	20.80	1.73	19.56
Brunswick	186.60	1.16	11.9	0.94	2.32	3.08	0.47	11.82	3.95	13.18
Collingwood	176.49	1.21	6.7	0.68	2.29	1.91	0.44	14.67	3.14	25.22
Emerald Hill	365.42	1.17	13.5	0.62	3.42	1.82	0.41	21.47	2.34	30.42
Essendon & Flemington	421.30	1.10	19.9	0.49	1.83	2.68	0.43	22.82	2.26	16.60
Fitzroy	314.99	1.06	16.1	3.77	2.60	2.16	0.38	30.41	2.08	16.78
Footscray	160.45	1.23	11.3	0.36	2.52	2.50	0.46	19.87	3.86	11.85
Hawthorn	445.66	0.93	39.6	1.31	2.92	3.29	0.41	29.52	1.84	11.96
Hotham	247.47	1.34	8.1	0.91	1.16	1.02	0.42	14.44	2.47	49.44
Kew	436.38	1.14	42.5	0.98	1.70	2.52	0.36	7.07	1.16	190.07
Melbourne	624.05	1.09	21.5	3.51	1.42	1.31	0.33	16.41	1.37	27.51
Bourke Ward		1.17	20.5	6.21						
Gipps Ward		1.18	22.0	9.55						
La Trobe Ward		0.82	41.3	9.98						
Lonsdale Ward		1.06	32.0	5.76						
Victoria Ward		1.16	8.8	3.14						
Albert Ward		0.87	53.4	5.36						
Smith Ward		1.14	16.8	1.42						
Prahan	481.19	0.89	23.9	0.85	3.06	3.62	0.39	36.05	2.07	16.77
Richmond	285.09	1.09	11.8	0.61	2.09	1.81	0.42	19.26	2.56	18.16
Sandridge	234.23	1.18	7.3	0.32	2.05	1.89	0.42	16.76	3.14	17.33
St Kilda	507.53	0.84	41.0	1.05	1.75	4.94	0.37	37.29	1.39	11.58
Williamstown	278.17	1.11	16.0	0.18	2.79	2.32	0.43	20.60	3.12	17.05

Sources:

(a) Statistical Register of the Colony of Victoria, 1881-Population, pp.21-23

(b) Census of Victoria, 1881. General Report. Table XXI, p.207.

(c) Census of Victoria, 1881. Part I - Inhabitants and Houses. Table XVI, pp.53-54.

(d) Census of Victoria, 1881. Part I - Inhabitants and Houses. Table XVI, pp.48-49.

(e) Census of Victoria, 1881. Part II - Birthplaces of the People. Table V, pp.24-32.

(f) Census of Victoria, 1881. Part III - Religions of the People. Table V, pp.26-34.

(g) Census of Victoria, 1881. Part IV - Ages of the People. Table VI, pp.25-33.

(h) Census of Victoria, 1881. Part V - Education of the People. Table V, pp.54-74.

(i) Census of Victoria, 1881. Part VI - Conjugal Condition of the People. Table III, pp.20-37.

(j) Census of Victoria, 1881. Part VIII - Sickness and Infirmity. Table III, p.7.

Income, education and occupation are generally considered to be optimal indicators of socioeconomic position for health research, and have been shown to affect health outcomes, including mortality (Duncan *et al.*, 2002).

The 1881 Victorian census offers small-area information on education, along with housing construction, population density, birthplace, religion, marital status, sickness and infirmity, all which bear on socioeconomic position (Table A5.1). In addition, estimates of property values for Melbourne and suburbs were obtained from the *Statistical Register of Victoria*.

There may be correlation between some of these indicators, and although their selection might have benefitted from a principal component analysis, this was not undertaken. Each indicator was assumed to have an equal weighting.

The location of health and welfare facilities in some of these suburbs is also problematic. The presence of the Metropolitan Lunatic Asylum in Kew, for example, may explain that suburb's low level of literacy and of marriage, and the disproportionate sickness and infirmity (Indicators h to j in Table A5.1). By all accounts, the suburb was genteel (Davison, 2004), and it may also be affected by large numbers of retirees and older persons. Hotham's SEIFA value might also be affected by hosting the Benevolent Asylum, which was located on its boundary with Melbourne City.

Although techniques exist to redistribute deaths which occur in institutions according to usual place of residence (Mooney *et al.*, 1999), this was not undertaken. These require detailed patient information not found in the abstracts of mortality. Although unfortunate, individuals live, work and die in these institutions, and since they serve as a place of abode, it could be argued that redistribution should not occur.

Values were ordered from low-to-high, i.e. from most- to least-socioeconomically disadvantaged. Values for each suburb were scaled from 0 (most disadvantaged) to 1 (least disadvantaged), summed and multiplied by a factor of 100. Since not enough data are available for the Wards within Melbourne City, these were excluded from further analysis.

The following serves as a rudimentary SEIFA for early 1880s Melbourne, with a low value representing the most disadvantaged suburb, and a high value the least disadvantaged:

Brighton	**533**
Brunswick	**274**
Collingwood	**202**
Emerald Hill	**365**
Essendon & Flemington	**383**
Fitzroy	**534**
Footscray	**277**
Hawthorn	**648**
Hotham	**139**
Kew	**445**
Melbourne	**539**
Prahan	**620**
Richmond	**305**
Sandridge	**241**
St Kilda	**761**
Williamstown	**335**

In 1881 Melbourne, St Kilda was the suburb with the greatest socioeconomic advantage, and working-class Hotham the least.

Davison (2004) offers qualitative evidence on whether these values accurately reflect the social character of colonial Melbourne (Table A5.2). The SEIFA values correspond reasonably well, with Fitzroy perhaps being the only suburb which belies its description.

Other studies offer additional descriptions of individual suburbs—e.g. McCalman (1984), who has provided a valuable social history of life in working-class Richmond from the turn of the twentieth century.

Table A5.2: Social descriptions of late nineteenth-century Melbourne suburbs, along with SEIFA values

Suburb	Description	SEIFA value
St Kilda	Plush, fashionable, wealthy geriatrics, seaside, middle class	761
Hawthorn	New commuter suburb, healthy, superior middle-class, outer, well-to-do, bourgeois, comparatively comfortable	648
Prahan	New commuter suburb, high population	620
Melbourne		539
Fitzroy	Inner, predominantly working class, low rent, social instability, comfortable and respectable, plebeian	534
Brighton	Seaside, fashionable, middle class, boom suburb, comparatively comfortable	533
Kew	Fashionable, prestigious, middle class	445
Essendon & Flemington	Pleasant, boom suburb, comparatively comfortable	383
Emerald Hill (South Melbourne)	Inner, working class industrial, heavy engineering, crowded	365
Williamstown	Heavy engineering, working class, comparatively comfortable	335
Richmond	Inner, working class industrial, crowded	305
Footscray	Working class, modest suburbanism, industrial, foul	277
Brunswick	Working class, industrial, heavily populated	274
Sandridge (Port Melbourne)	Heavy engineering, working class	241
Collingwood	Inner, working class industrial, low rent, pauperism, blighted inner, back street slums, poor, dear land	202
Hotham (North Melbourne)	Working class, high population	139
East Melbourne	Fashionable, elegant	
West Melbourne	Heavy engineering, poorer, working	

Source: Davison, 2004.

Bibliography

ABBREVIATIONS

Parliamentary papers and reports, Government Gazettes, journals and newspaper titles are abbreviated as follows:

AJCP   Australian Joint Copying Project

AMG   *Australasian Medical Gazette*

AMJ   *Australian Medical Journal*

CO   Colonial Office, The National Archives, London

Han   *Hansard's Parliamentary Debates*

HRA   *Historical Records of Australia*

HTC   *Hobart Town Courier*

HTG   *Hobart Town Gazette*

NSWGG   *New South Wales Government Gazette*

NSWLC   New South Wales. *Votes and Proceedings of the Legislative Council*

NSWRG   New South Wales. *Annual report from the Registrar-General on Vital Statistics*, continued by *Annual Report from the Government Statistician on Vital Statistics*, continued by *Vital Statistics*

PG   *Perth Gazette, and Western Australian Journal (…and Independent Journal of Politics and News)*

QldRG   Queensland. *Annual Report by the Registrar-General* (title varies)

SAA   *South Australian Advertiser*

SAGG   *South Australian Government Gazette*

SAR	*South Australian (Gazette and Colonial) Register*
SARG	South Australia. *Registrar-General of Births, Deaths and Marriages Annual Report*
SASR	*Statistics of South Australia*, continued by *Statistical Register of South Australia*
SG	*Sydney Gazette and New South Wales Advertiser*
SMH	*Sydney Morning Herald*
TasLC	*Votes and Proceedings of the Legislative Council of Van Diemen's Land*
TasSR	*Statistical Returns of Tasmania*, continued by *Statistics of Tasmania*
VicGG	*Victoria Government Gazette*
VicRG	Victoria. *Annual Report of the Registrar-General* (title varies)
VicSR	*Statistics of the Colony of Victoria*, continued by *Statistical Register of the Colony of Victoria*
WABB	Western Australia. *Blue Book*
WAGG	*Western Australian Government Gazette*
WALC	Western Australia. *Minutes of the Proceedings of the Legislative Council*
WAPH	Western Australia. *Report by the Colonial Surgeon on the Public Health of the Colony, for the Year…*
WARG	Western Australia. *Report of the Registrar-General on the Vital Statistics*
WASR	*Statistical Register of Western Australia*

LEGISLATION

New South Wales

19 Vic. 34. An Act for Registering Births, Deaths, and Marriages, 1855

Act No. 17, 1899. Registration of Births, Deaths and Marriages Act, 1899

Victoria

16 Vic. 26. An Act for Registering Births, Deaths and Marriages in the Colony of Victoria, 1853

28 Vic. No. 246. Registration of Births, Deaths and Marriages Statute, 1865

54 Vic. 1137. Registration of Births Deaths and Marriages Act, 1890

Queensland

Regulations dated 18 Dec 1855, under Section 6 of 19 Vic 34

31 Vic. 7. Registration of Births Deaths and Marriages Amended Registration Act, 1867

Western Australia

4 & 5 Vic. No. 9. An Act to provide for the Registration of Births, Deaths and Marriages, in the Colony of Western Australia, 1841

10 Vic. No. 17. Births, deaths and marriages registration amendment Act, 1847

12 Vic. No. 13. Births, deaths and marriages registration amendment Act, 1849

19 Vic. No. 12. Registration of Births, Deaths and Marriages Act, 1856

43 Vic. No.15. Registration Ordinance Amendment Act, 1879

58 Vic. No. 16. Registration of Births, Deaths and Marriages Act, 1894

64 Vic. No. 31. Registration of Births, Deaths, and Marriages Amendment Act, 1900

7 Edw. VII No. 19. An Act to amend the Registration of Births, Deaths, and Marriages Act, 1894, 1907

South Australia

5 Vic. No. 13. An Act for Registering Births, Deaths, and Marriages in the Province of South Australia, 1842

19 Vic. No. 3. An Act to amend the law relating to Registering of Births, Deaths, and Marriages in the Province of South Australia, 1855–56

37 & 38 Vic. No. 10. An Act to amend the Law relating to the Registration of Births and Deaths in the Province of South Australia, 1874

63 & 64 Vic. No 744. An Act to amend The Registration of Births and Deaths Act 1874, 1900

Tasmania

2 Vic. No. 8. An Act for registering Births, Deaths and Marriages in the Island of Van Diemen's Land and its Dependencies, 1838

6 Vic. No. 12. An Act to amend an Act passed in the second year of the reign of Her present Majesty intituled An Act for registering births deaths and marriages in the island of Van Diemen's Land and its dependencies, 1842

59 Vic. No. 9. An Act to consolidate and amend the law relating to the registration of births and deaths in Tasmania, 1895

CONTEMPORARY WORKS

Abbott, F. (1872), *Results of Five Years' Meteorological Observations for Hobart Town*, Government Printer, Hobart Town.

Allen, H. B. (1903), 'Cancer. Introductory Address', *Intercolonial Medical Congress of Australasia, Transactions of the Sixth Session*, 1–55.

Anderson, E. (1888), 'The Midwifery Practice of the Women's Hospital, With Analysis of, and Reports on, Five Hundred Consecutive Cases', *Australian Medical Journal*, 10: 211–218.

Anon. (1908), 'Infantile Mortality and Summer Diarrhoea: A Retrospect', *Intercolonial Medical Journal*, 13: 249–252.

Archer, W. H. (ed.) (1854), *The Statistical Register of Victoria, From the Foundation of the Colony: With an Astronomical Calender for 1855*, Government Printer, Melbourne.

Archer, W. H. (1856), *A Statistical Nosology, For the Use of Those Who Return the Causes of Death. Circular to Coroners, and a Classification of the Causes of Violent Deaths*, Government Printer, Melbourne.

Archer, W. H. (1859), 'On Mortality of Children in Victoria', *Australian Medical Journal*, 4: 139–145.

Archer, W. H. (1863), *Nosological Index, or Guide to the Classification and Tabulation of the Various Causes of Death*, second edition, revised and corrected, Government Printer, Melbourne.

Archer, W. H. (1867), *The Progress of Victoria: A Statistical Essay*, Blundell & Co., Melbourne.

Archer, W. H. (1868), *Nosological Index, or Guide to the Classification and Tabulation of the Various Causes of Death, third edition, revised and corrected*, Government Printer, Melbourne.

Archer, W. H. (1873), *The Progress of Victoria: A Statistical Essay*, Government Printer, Melbourne.

Archer, W. H. (1878), *Nosological Index, or Guide to the Classification and Tabulation of the Various Causes of Death, fourth edition, revised and corrected by the Government Statist*, Government Printer, Melbourne.

Armstrong, W. G. (1905), 'Some Lessons From the Statistics of Infantile Mortality in Sydney', *Australasian Medical Gazette*, 24: 512–520.

Balls-Headley, W. (1888), 'Antiseptic Midwifery', *Australian Medical Journal*, 10: 314–323.

Barrett, J. W. (1883), *Typhoid Fever in Victoria*, George Robertson, Melbourne.

Bennett, G. (1834), *Wanderings in New South Wales, Batavia, Pedir Coast, Singapore and China: Being the Journal of a Naturalist in Those Countries, During 1832, 1833 and 1834* (Vol. 1), Richard Bentley, London.

Burridge, A. (1882), 'On the Rates of Mortality in Victoria', *Journal of the Institute of Actuaries*, 23: 309–335.

Burridge, A. (1884), 'On the Rates of Mortality in Australia', *Journal of the Institute of Actuaries*, 24: 333–358.

Campbell, N. (1855a), *Births, Deaths and Marriages in the Colony of Victoria, During the Year Ending 30th June, 1854. First Annual Report of the Registrar-General*, Government Printer, Melbourne.

Campbell, N. (1855b), *Births, Deaths, and Marriages in the Colony of Victoria. Second Annual Report of the Registrar-General*, Government Printer, Melbourne.

Campbell, N. (1856), *Births, Deaths, and Marriages in the Colony of Victoria. Third Annual Report*, Government Printer, Melbourne.

Carstairs, J. G. (1889), 'The Etiology of Typhoid', *Intercolonial Medical Congress of Australasia. Transactions of Second Session*: 155–159.

Carstairs, J. G. (1892), 'An Inquiry into the Cause of Influenza and the Course of the Pandemic Wave in 1890', *Intercolonial Medical Congress of Australasia, Transactions of Third Session*: 612–617.

Carstairs, J. G. (1894), 'An Enquiry into the Change of Type in the Specific Fevers: Its Nature and Causation', *Australian Medical Journal*, 16(10): 480–495.

CBCS (Commonwealth Bureau of Census and Statistics) (1907), *The Nomenclature of Diseases and of Cause of Death, as Revised and Adopted in 1900 by the International Commission and Published by Dr. Jacques Bertillon*, Government Printer, Melbourne.

CBCS (1908), *Population and Vital Statistics. Bulletin No. 8. Vital Statistics of the Commonwealth for the Year 1908*, Government Printer, Melbourne.

Coghlan, T. A. (1887), *The Wealth and Progress of New South Wales 1886–87.* Government Printer, Sydney.

Coghlan, T. A. (1888), *The Wealth and Progress of New South Wales 1887–88.* Government Printer, Sydney.

Coghlan, T. A. (1892), 'A New South Wales Life Table', *Intercolonial Medical Congress. Transactions*, 3rd Session: 677–681.

Coghlan, T. A. (1898), 'Deaths in Child-birth in New South Wales', *Journal of the Royal Statistical Society*, 61: 518–533.

Coghlan, T. A. (1900), *A Statistical Account of the Seven Colonies of Australasia, 1899–1900*, Government Printer, Sydney.

Coghlan, T. A. (1902a), *The Wealth and Progress of New South Wales 1900–01.* Government Printer, Sydney.

Coghlan, T. A. (1902b), *A Statistical Account of the Seven Colonies of Australasia, 1901–1902*, Government Printer, Sydney.

Coghlan, T. A. (1903), 'The Causes of the Increase or Alleged Increase in Cancer, Examined in the Light of the Statistics of New South Wales, Since 1856', *Intercolonial Medical Congress of Australasia, Transactions of the Sixth Session*, 63–78.

Coghlan, T. A. (1904), *A Statistical Account of Australia and New Zealand 1903–4*, Government Printer, Sydney.

Conference of Statisticians (1906), *Conference of Statisticians of the Commonwealth and States of Australia and Colony of New Zealand, Melbourne, November and December, 1906*, Government Printer, Melbourne.

Coxwell, C. F. (1893), 'A Study of Phthisis Mortality in Victoria', *Australian Medical Journal*, 15: 185–206.

Crivelli, M. (1891), 'On the Bacillus of Koch and Tuberculosis', *Australian Medical Journal*, 13: 59–66, 68–77, 99–100.

Cullen, W. (1816), *Synopsis of Nosology* (translated from Latin by John Thompson), Thomas Dobson, Philadelphia.

Cunningham, P. (1827), *Two Years in New South Wales; A Series of Letters, Comprising Sketches of the Actual State of Society in That Colony*, Henry Colburn, London.

Dovey, W. (1893), 'The Rates of Mortality in New South Wales and Victoria', *Australasian Insurance and Banking Record*, 17: 832–836.

Drake, E. T. (1908), *Victorian Year-Book, 1906–7*, Government Printer, Melbourne.

Duckworth, A. (1894), 'A Comparison of Populations and Rates of Mortality in New South Wales and Victoria, in Sydney and Melbourne', *Australasian Insurance and Banking Record,* 18: 656–661.

Duncan, H. (1850), *The Colony of South Australia*, T. & W. Boone, London.

Durlacher, A. (1859), *Western Australia. Report on the General Statistics of the Colony, for the Year 1859*, Government Printer, Perth.

Fenton, J. J. (1901), *Victorian Year-Book, 1895–8*, Government Printer, Melbourne.

Ferrier, D. (1885), 'Signs of Death', In: Quain, R. (ed.), *A Dictionary of Medicine, Eighth Edition*, D. Appleton & Company, New York.

Fisher, J. H. (1836), *A Sketch of Three Colonial Acts, Suggested for Adoption in the New Province of South Australia, With a View to Ensure the Most Perfect Security of Title to Property, to Simplify and Facilitate the Mode and Moderate the Expense of its Transfer*, W. Clowes & Sons, London.

Fraser, M. A. C. (1902), *Western Australian Year-Book for 1900–01, Volume 2*, Government Printer, Perth.

Fraser, M. A. C. (1906), *Western Australian Year-Book for 1902–04,* Government Printer, Perth.

Girdlestone, T. M., J. Singleton, J. Williams & J. Jamieson (1877), 'Report on the Prevalence of Phthisis in Victoria', *Australian Medical Journal*, 22: 358–366.

Good, J. M. (1817), *A Physiological System of Nosology, With a Corrected and Simplified Nomenclature*, Cox and Sons, London.

Gresswell, D. A. (1899), 'Tuberculosis and its Prevention', *Intercolonial Medical Journal of Australasia*, 4: 389–407.

Gresswell, D. A. (1904), 'Prevalence of Tuberculosis in Australia', in: *Victorian Year-Book, 1903*, Government Printer, Melbourne, pp.232–236.

Hall. E. S. (1860), 'On the Influenza Epidemic in Hobarton (Tasmania), in July, 1860', *Australian Medical Journal*, 5: 252–269.

Hall, E. S. (1863), 'Vital Statistics of Tasmania in 1861', *Transactions of the Epidemiological Society of London*, 1: 363–4

Hall, E. S. (1864a), 'On the Epidemic Diseases of Tasmania', *Transactions of the Epidemiological Society of London*, 2: 69–87.

Hall, E. S. (1864b), 'Analysis of the Observatory Records for 1864, in Conjunction With Those of Births, Deaths, etc.', *Monthly Notices of Papers and Proceedings of the Royal Society of Tasmania,* The Mercury, Hobart.

Hall, W. H. (1906), *The Official Year Book of New South Wales, 1904–5*, Government Printer, Sydney.

Hammick, J. T. (1875), *The Acts Relating to the Registration of Births, Deaths and Marriages*, Shaw & Sons, London.

Haviland, A. (1887), 'The Statistics of Cancer in England and Victoria Compared', *Australian Medical Journal*, 9(3): 97–101.

Hayter, H. H. (1878–79), 'Infantile Mortality in South Australia', *Trans Proc Phil Soc Adelaide*, 2: 65–74.

Hayter, H. H. (1880), *Victorian Year-Book for 1878–9*, Government Printer, Melbourne.

Hayter, H. H. (1881a), 'Government Statist's Report on the Vital Statistics of Melbourne and Suburbs–Year 1880', *Victorian Government Gazette*, February 18, 1881, p.528–529.

Hayter, H. H. (1881b), *Census of Victoria, 1881. General Report*, Government Printer, Melbourne.

Hayter, H. H. (1887a), *Victorian Year-Book for 1886–7*, Government Printer, Melbourne.

Hayter, H. H. (1887b), *Nosological Index, or Guide to the Classification and Tabulation of the Various Causes of Death*, Government Printer, Melbourne.

Hayter, H. H. (1889), *Victorian Year-Book for 1888–9*, Government Printer, Melbourne.

Hayter, H. H. (1892), *Nosological Index, or Guide to the Classification and Tabulation of the Various Causes of Death, Sixth Edition, Revised and Corrected*, Government Printer, Melbourne.

Hayter, H. H. (1893), 'Disturbance of the Population Estimates in Australia by Defective Records', *Journal of the Royal Statistical Society*, 56(2): 327–330.

Jamieson, J. (1876), 'On the Parasitic Theory of Disease', *Australian Medical Journal*, 21: 256–262, 287–300, 315–324.

Jamieson, J. (1882a), 'Contributions of the Vital Statistics of Australia, I. On the Frequency of Abortion in Victoria', *Australasian Medical Gazette*, 1: 103.

Jamieson, J. (1882b), 'Contributions of the Vital Statistics of Australia, II. Puerperal Fever, its Prevalence and Pathological Affinities', *Australasian Medical Gazette*, 1: 129–132.

Jamieson, J. (1882c), 'Contributions of the Vital Statistics of Australia, III. Infant Mortality', *Australasian Medical Gazette*, 1: 162–165.

Jamieson, J. (1883), 'On the Exact Role of the Pathogenic Bacteria', *Australian Medical Journal*, 5: 1–8.

Jamieson, J. (1884), 'The Present State of the Puerperal Fever Question', *Australian Medical Journal*, 6: 244–252.

Jamieson, J. (1887), 'Childbirth Mortality in the Australian Colonies', *Australian Medical Journal*, 9: 442–448.

Jamieson, J. (1898), 'Tuberculosis and its Prevention', *Intercolonial Medical Journal of Australasia*, 3: 637–643.

Jamieson, J. (1905), 'Twenty Years of Sanitary Progress in Melbourne', *Intercolonial Medical Journal of Australasia*, 10: 441–449.

Jay, M. (1886), 'On the Recent Epidemic of Influenza In and About Adelaide', *Australasian Medical Gazette*, 5: 164–165.

Jeffreys Wood, A. (1908), 'Preservation of Infant Life', *Intercolonial Medical Journal*, 13: 128–141.

Kilgour, J. (1855), *Effect of the Climate of Australia Upon the European Constitution in Health and Disease*, William Vale, Geelong.

Knibbs, G. H. (1907), 'The Classification of Disease and Causes of Death, From the Standpoint of the Statistician', *Intercolonial Medical Journal*, 12: 332–349.

Knibbs, G.H. (1908), 'The Tuberculosis Frequency Duration Curves and the Number of Existing Cases Ultimately Fatal', *Australasian Medical Congress*, 2, 196–202.

Knibbs, G. H. (1912), *Official Yearbook of the Commonwealth of Australia*, Commonwealth Bureau of Census and Statistics, Melbourne.

Knight, W. (1870), *Census of the Colony of Western Australia, Taken at the 31st March, 1870, as Also the General Statistics of the Colony, for the Ten Years Ending 31st December 1869*, Registrar-General's Office, Perth.

Litchfield, W. F. (1905), 'Summer Diarrhoea in Infants: From the Public Health Point of View', *Transactions of the Australasian Medical Congress*, 421–425.

Long, A. P. (1898), *New South Wales. Nosological Index, or Guide to the Classification and Tabulation of the Various Causes of Death, With Instructions to District Registrars*, Government Printer, Melbourne.

Melbourne and Metropolitan Board of Works (1906–07), *Notice Papers, Minutes, Returns, Reports and Memoranda, Vol. 16*, MMBW, Melbourne.

Millard, R. J. (1906), 'Typhoid Fever in New South Wales, 1898–1904', *Australasian Medical Gazette*, 25: 125–134.

Mitchell, J. T. (1893), 'Summer Diarrhoea of Infants', *Australasian Medical Gazette*, 12: 234–237.

Mullins, G. L. (1896), 'Cancer in New South Wales', *The Australasian Medical Gazette*, 15: 1–8.

Mullins, G. L. (1898), 'Tuberculosis and the Public Health', *Australasian Medical Gazette*, 17: 9–16.

New South Wales Government (1904), *Royal Commission on the Decline of the Birth-Rate and of the Mortality of Infants in New South Wales, Volume 1, Report*, Government Printer, Sydney.

New South Wales Registrar-General (1857), *First Annual Report from the Registrar-General*, Government Printer, Sydney.

New South Wales Registrar-General (1858), *Second Annual Report from the Registrar-General*, Government Printer, Sydney.

Nyulasy, A. J. (1898), 'Typhoid Fever & The Perth Water Supply', *Intercolonial Medical Journal of Australasia*, 3: 506–510.

Parliament of Tasmania (1902), *Conference of Statisticians at Hobart, January, 1902*, Parliamentary Paper No. 25, Government Printer, Hobart.

Pell, M. B. (1867), 'On the Rates of Mortality and the Expectation of Life in New South Wales as Compared With England and Other Countries', *Transactions of the Royal Society of New South Wales*, 1: 66–76.

Pell, M. B. (1879), 'On the Rates of Mortality in New South Wales, and on the Construction of Mortality Tables from Census Returns', *Journal of the Institute of Actuaries*, 21: 257–288.

Quain, R. (ed.) (1885), *A Dictionary of Medicine, Eighth Edition*, D. Appleton & Company, New York.

Registrar-General of England (1839), *First Annual Report of the Registrar-General of Births, Deaths, and Marriages, in England,* W. Clowes and Sons, London.

Registrar-General of England (1860), *Twenty-First Annual Report of the Registrar-General of Births, Deaths and Marriages in England*, George E. Eyre and William Spottiswoode, London.

Registrar-General of England (1868), *Twenty-Ninth Annual Report of the Registrar-General of Births, Deaths and Marriages in England*, George E. Eyre and William Spottiswoode, London.

Registrar-General of England (1883), *Forty-Fourth Annual Report of the Registrar-General of Births, Deaths and Marriages in England*, George E. B. Eyre and William Spottiswoode, London.

Registrar-General of England (1903), *Sixty-Fourth Annual Report of the Registrar-General of Births, Deaths and Marriages in England and Wales*, His Majesty's Stationery Office, London.

Robertson, J. (1889), 'History of Typhoid Fever in Victoria, and its Etiology', *Intercolonial Medical Congress of Australasia. Transactions of Second Session*: 149–154.

Rolleston, C. (1856), *Registration. (Report From the Registrar General.)*, Government Printer, Sydney.

Rolleston, C. (1858a), 'The Sanitary Condition of Sydney', *The Sydney Magazine of Science and Art*, 1: 37–41.

Rolleston, C. (1858b), 'The Science of Statistics', *The Sydney Magazine of Science and Art*, 2: 254–258.

Royal College of Physicians (1885), *The Nomenclature of Diseases, Second Edition Being the First Revision*, Harrison and Sons, London.

Royal College of Physicians (1896), *The Nomenclature of Diseases, Third Edition Being the Second Revision*, The Royal College of Physicians, London.

S. A. Almanack (1849), *The Royal South Australian Almanack and General Directory for 1849*, John Stephens, Adelaide.

Scott, H. (1872), *Queensland. Census of 1871, Registrar-General's Report*, Government Printer, Brisbane.

Singleton, J. (1871), 'On Phthisis in Victoria', *Australian Medical Journal*, 16: 206–209.

Springthorpe, J. W. (1885), 'Some Points of Interest in the Late Epidemic', *Australian Medical Journal*, 8: 457–468.

Springthorpe, J. W. (1890), 'Introductory Paper on Influenza', *Australian Medical Journal*, 12: 402–415.

Sprott, G. (1896), 'Tuberculosis', *Papers and Proceedings of the Royal Society of Tasmania*, 45–68.

Stawell, R. R. (1895), 'Some Notes on the Aetiology and Pathological Anatomy of Summer Diarrhoea in Infants', *Australian Medical Journal*, 17: 67–77.

Stawell, R. R. (1899), 'Summer Diarrhoea: A Review of Facts and a Study in Statistics', *Intercolonial Medical Journal of Australasia*, 4: 146–159.

Symes Thompson, E. (1873), 'On the Elevated Health Resorts of the Southern Hemisphere', *Medico-Chirurgical Transactions*, 56: 285–324.

Thomson, W. (1879), *On Phthisis, and the Supposed Influence of Climate*, Stillwell and Co., Melbourne.

Thwaites, W. (1906), 'Sewage System of Melbourne', *Intercolonial Medical Journal*, 10: 24–38.

Trivett, J. B. (1909), *The Official Year Book of New South Wales, 1907–08*, Government Printer, Sydney.

Turner, A. J. (1899a), 'The Diphtheria Mortality of the Three Principal Australian Colonies for the Past Fifteen Years', *British Medical Journal*, Nov. 18 1899, 1409–1412.

Turner, A. J. (1899b), 'The Treatment of Diphtheria', *Intercolonial Medical Journal of Australasia*, 4: 585–599.

Turner, A. Jefferis (1904), 'The Notification of Consumption', *Intercolonial Medical Journal of Australasia*, 9: 357–367.

Verco, J. C. (1903), 'The South Australian Statistics of Cancer', *Intercolonial Medical Congress of Australasia, Transactions of the Sixth Session*, 56–62.

Whittell, H. T. (1892), 'The Health Laws of Australia', *Intercolonial Medical Congress*, 14: 539–545.

GENERAL WORKS

ABS (Australian Bureau of Statistics) (1989), *Catalogue of Australian Statistical Publications, 1804–1901*, ABS catalogue no. 1115.0, ABS, Canberra.

ABS (1997), 'Census History in Tasmania', *Tasmanian Year Book, 1998*, ABS, Hobart.

ABS (2003), *Australian demographic statistics, September quarter 2002*, ABS catalogue number 3101.0, ABS, Canberra.

ABS (2008a), *Australian Historical Population Statistics, 2008. Section 1, Population Size and Growth*, ABS catalogue no. 3105.0.65.001, ABS, Canberra. Available online: www.abs.gov.au

ABS (2008b), *Discussion Paper: Assessment of Methods for Developing Life Tables for Aboriginal and Torres Strait Islander Australians*, ABS catalogue number 3302.0.55.002, ABS, Canberra.

ABS (2008c), *Information Paper: An Introduction to Socio-Economic Indexes for Areas (SEIFA), 2006*, ABS catalogue no. 2039.0, ABS, Canberra.

ABS (2010), *Deaths, Australia, 2009*, ABS catalogue no. 3302.0, ABS, Canberra.

ABS (2012), *Causes of Death, Australia, 2010*, ABS catalogue no. 3303.0. ABS, Canberra.

AIHW (Australian Institute of Health and Welfare) (2005), *Mortality Over The Twentieth Century in Australia: Trends And Patterns in Major Causes of Death*, AIHW cat. no. PHE 73, AIHW, Canberra.

AIHW (2014), *GRIM (General Record of Incidence of Mortality) Books*, AIHW, Canberra. Available online:
aihw.gov.au/mortality/data/grim_books_national.cfm.

AIHW (2012), *Australia's Health 2012*, AIHW cat. no. AUS 156, AIHW, Canberra.

AIHW: de Looper, M. (2002), *Seasonality of Death*, Bulletin No. 3, AIHW cat. no. AUS 29, AIHW, Canberra.

Aird, W. V. (1961), *The Water Supply, Sewerage and Drainage of Sydney*, Halstead Press, Sydney.

Alter, G. & A. Carmichael (1996), 'Studying Causes of Death in the Past', *Historical Methods,* 29: 44–48.

Alter, G. & A. Carmichael (1999), 'Classifying the Dead: Toward a History of the Registration of Causes of Death', *Journal of the History of Medicine*, 54: 114–132.

Andvord, K. F. (1939, 2008), 'Continued Studies of Tuberculosis Considered as a Generation Illness', reprinted in *International Journal of Epidemiology*, 37: 917–922.

Arriaga, E. E. (1984), 'Measuring and Explaining the Change in Life Expectancies', *Demography*, 21: 83–96.

Barnes, T., L. Smith, Y. Zhao & S. Guthridge (2008), *A Comparative Analysis of Indirect Methodologies for Estimating Indigenous Life Expectancy*, Charles Darwin University, Darwin.

Barry, J. M. (2004), *The Great Influenza*, Viking, New York.

Bashford, A. (2009), *Australasia and Oceania*, In: Cook, H. J., S. Bhattacharya & A. Hardy (*eds.*), *History of the Social Determinants of Health: Global Histories, Contemporary Debates*, Orient BlackSwan, Hyderabad.

Bartram, J. & S. Cairncross (2010), 'Hygiene, Sanitation, and Water: Forgotten Foundations of Health', *PLOS Medicine*, 7(11): 1-9.

Bhat, P. N. Mari (2002), 'General Growth Balance Method: A Reformulation For Populations Open To Migration', *Population Studies*, 56: 23–34.

Borrie, W. D. (1994), *The European Peopling of Australasia, A Demographic History, 1788–1988*, Demography Program, Research School of Social Sciences, Australian National University, Canberra.

Bourgeois-Pichat, J. (1951), 'La mesure de la mortalite infantile. I. Principes et methods', *Population*, 6(2): 233–248.

Bradley, J., R. Kippen, H. Maxwell-Stewart, J. McCalman & S. Silcot (2010), 'Research Note: the Founders and Survivors project', *History of the Family*, 15: 467–477.

Broom, L. D. (1977), *Investigating Social Mobility*, Department of Sociology, Research School of the Social Sciences, Australian National University, Canberra.

Butlin, N. G. (1964), *Investment in Australian Economic Development*, Cambridge University Press.

Caldwell, J. C. (2001), 'Population Health in Transition', *Bulletin of the World Health Organization*, 79: 159–160.

Caldwell, J. C. (2006), *Demographic Transition Theory*, Dordrecht, Springer.

Camm, J. (1988), *The Early Nineteenth Century Colonial Censuses of Australia*, Historical Statistics Monograph No. 8, Australian Reference Publications, Bundoora, Victoria.

Canning, D. (2011), 'The Causes and Consequences of Demographic Transition', *Population Studies*, 65: 353–361.

Casselli, G., F. Meslé & J. Vallin (2002), 'Epidemiologic Transition Theory Exceptions', *Genus: Journal of Population Sciences*, 9: 9–51.

Chesnais, J.-C. (1992), *The Demographic Transition – Stages, Patterns and Economic Implications*, Clarendon Press, Oxford.

Chiang, C. L. (1978), *Life Table and Mortality Analysis*, World Health Organization, Geneva.

Chiang CL (1984), *The Life Table and its Applications*, Malabar, FL: R.E. Krieger Publ. Co.

Christie, D. (1974), 'Mortality from Cardiovascular Disease', *Medical Journal of Australia*, 1: 390–393.

Clark, D. (1978), 'Worse Than Physic: Sydney's Water Supply 1788–1888', In: Kelly, M. (ed.), *Nineteenth-Century Sydney. Essays in Urban History*, Sydney University Press, Sydney.

Cleland, J. B. (1938), 'Some Early References to Tuberculosis in Australia', *Medical Journal of Australia*, 1: 256–258.

Condran, G. A. (2008), 'The Elusive Role of Scientific Medicine in Mortality Decline: Diphtheria in Nineteenth- and Early Twentieth-Century Philadelphia', *Journal of the History of Medicine and Allied Sciences*, 63: 484–522.

Condran, G. A. & R. A. Cheney (1982), 'Mortality Trends in Philadelphia: Age- and Cause-Specific Death Rates 1870–1930', *Demography*, 19: 97–123.

Cook, H. J., S. Bhattacharya & A. Hardy (*eds.*), *History of the Social Determinants of Health: Global Histories, Contemporary Debates*, Orient BlackSwan, Hyderabad.

Cummins, C. J. (1979), *A History of Medical Administration in New South Wales 1788–1973*, Health Commission of NSW, Sydney.

Cumpston, J. H. L. (1928), 'The Development of Public Health in Australia', *Medical Journal of Australia*, 1: 332–336.

Cumpston, J. H. L. (1989), *Health and Disease in Australia: A History*, Australian Government Publishing Service, Canberra.

Curson, P. (1978), 'The 'St Philip's Project': The Historical Demography of Inner Sydney 1788–1888', In: Kelly. M. (ed), *Nineteenth-Century Sydney–Essays in Urban History*, Sydney University Press, Sydney.

Curson, P. (1985a), *Times of Crisis, Epidemics in Sydney 1788–1900*, Sydney University Press, Sydney.

Curson, P. (1985b), 'Edward Swarbreck Hall (1804–81): Pioneer Australian Demographer', *Journal of the Australian Population Association*, 2: 104–113.

Curson, P. & K. McCracken (1989), *Plague in Sydney: the Anatomy of an Epidemic*, UNSW Press, Kensington.

Davenport, R. (2013), 'Year of Birth Effects in the Historical Decline of Tuberculosis Mortality: A Reconsideration', PLoS ONE, 8(12): e81797.

Davison, G. (2004), *The Rise and Fall of Marvellous Melbourne*, Melbourne University Press, Melbourne.

Day, C., R. Kippen & D. Lucas (2006), 'Historical Demography', in: D. Lucas & P. Meyer (ed.), *Beginning Population Studies (3rd ed.)*, Australian National University, Canberra, pp. 1–10.

De Costa, C. (2002), 'The Contagiousness of Childbed Fever: A Short History of Puerpal Sepsis and its Treatment', *Medical Journal of Australia*, 177: 668–671.

Dewdney, J. C. H. (1960), 'A Note on Seasonal Swings in Mortality in Australia, 1861–1955', *Australian Journal of Statistics*, 2: 84–90.

Dingle, T. & C. Rasmussen (1991), *Vital Connections: Melbourne and its Board of Works 1891–1991*, McPhee Gribble, Ringwood, Victoria.

Dobson, A. J., R. W. Gibberd, D. J. Wheeler & S. R. Leeder (1981), 'Age-specific Trends in Mortality from Ischaemic Heart Disease and Cerebrovascular Disease in Australia', *American Journal of Epidemiology*, 113: 404–412.

Due, S. (1994), 'Early Medical Journals of Australia', *Medical Journal of Australia*, 161: 340–341.

Duncan, G. J., M. C. Daly, P. McDonough & D. R. Williams (2002), 'Optimal Indicators of Socioeconomic Status for Health Research', *American Journal of Public Health*, 92: 1151–1157.

Dunstan, D. (2003), 'Rules of Simple Cleanliness: The Australian Health Society 1875-1900', *Victorian Historical Journal*, 74: 67–79.

Dunstan, D. (2005), 'Jamieson, James (1840–1916)', in *Australian Dictionary of Biography, Supplementary Volume*, Melbourne University Press, Melbourne.

Durey, M. (1980), 'The Death Registers of the Registrar-General as Sources for Social and Demographic History', *Bulletin (Australia 1888)*, 6: 88–93.

Durey, M. (1982), 'Infant Mortality in Perth, Western Australia, 1870–1914: A Preliminary Analysis', *Studies in Western Australian History*, 5: 62–71.

Dyson, T. (2010), *Population and Development: The Demographic Transition*, Zed Books, London.

Erikson, R. & J. Goldthorpe (1992), *The Constant Flux. A Study in Class Mobility in Industrial Nations*, Clarendon Press, Oxford.

Eyler, J. M. (1979), *Victorian Social Medicine, the Ideas and Methods of William Farr*, Johns Hopkins University Press, Baltimore, Maryland.

Ferrie, J. P. & W. Troesken (2008), 'Water and Chicago's Mortality Transition, 1850–1925', *Exploration in Economic History*, 45: 1–16.

Fisman, D. (2007), 'Seasonality of Infectious Diseases', *Annual Review of Public Health*, 28: 127–143.

Fitzgerald, S. (1987), *Rising Damp, Sydney 1870–90*, Oxford University Press, Melbourne.

Forster, C. & C. Hazlehurst (1988), 'Australian Statisticians and the Development of Official Statistics', in *Year Book Australia 1988*, ABS Cat. No. 1301.0, Australian Bureau of Statistics, Canberra.

Frenk, J., J. L. Bobadilla, C. Stern, T. Frejka & R. Lozano (1991), 'Elements For A Theory Of The Health Transition', *Health Transition Review*, 1: 21–38.

Frost, W. H. (1939, 1995), 'The Age Selection of Mortality From Tuberculosis in Successive Decades', reprint in *American Journal of Epidemiology*, 141: 4–9.

Galley, C. & R. Woods (1999), 'On the Distribution of Deaths During the First Year of Life', *Population: An English Selection*, 11: 35–59.

Gaminiratne, W. K. (2001), 'Recent Developments in Causes of Death Statistics in Australia: Automation and Multiple Coding', *Genus*, 57: 123–141.

Gandevia, B. (1976), 'Thomson, William (1819–1883)', In: *Australian Dictionary of Biography*, Volume 6, Melbourne University Press, Melbourne, 270–272.

Gandevia, B. (1978), *Tears Often Shed: Child Health and Welfare in Australia From 1788*, Pergamon Press, Rushcutters Bay.

Glass, D. V. (1973), *Numbering the People: The Eighteenth-Century Population Controversy and the Development of Census and Vital Statistics in Britain*, Saxon House, Farnborough, Hants, England.

Government of Western Australia (2014), *Searching Western Australian Online Indexes*, www.bdm.dotag.wa.gov.au/_apps/pioneersindex/default.aspx, Department of the Attorney General, Registry of Births, Deaths and Marriages.

Guttormsson, L. & O. Garðarsdóttir (2002), 'The Development of Infant Mortality in Iceland, 1800–1920', *Hygiea Internationalis*, 3(1): 151–176.

Haines, M. R. (2001), *The Urban Mortality Transition in the United States, 1800–1940*, NBER Historical Paper 134, National Bureau of Economic Research, Cambridge, MA.

Haines, M.R. & H. J. Kintner (2000), 'The Mortality Transition in Germany, 1860–1935', *Historical Methods*, 33: 83–103.

Hardy, A. (1994), 'Death is the Cure of All Diseases: Using the General Register Office Cause of Death Statistics for 1837–1920', *Social History of Medicine*, 7: 472–492.

Hardy, S. (1987), 'Ferments, Zymes and the West Wind: Adapting Disease Theories and Therapies in New South Wales, 1860–1880', in: Attwood, H. & Kenny, G. (ed.), *Reflections on Medical History and Health in Australia. Third National Conference on Medical History and Health in Australia 1986*, Melbourne, Medical History Unit, University of Melbourne.

Harris, B., R. Floud, R. W. Fogel & S. C. Hong (2010), *Diet, Health and Work Intensity in England and Wales, 1700-1914*, NBER Working Paper 15875, National Bureau of Economic Research, Cambridge, MA.

HCCDA (Historical Census and Colonial Data Archive) (2014), *Colonial Census Publications and Reports*, ANU, Canberra, http://hccda.ada.edu.au/

Hinde, A. (1998), *Demographic Methods*, Arnold, London.

Holton, S. (1983), 'Social Medicine in Nineteenth Century South Australia', *Community Health Studies*, 7: 121-135.

Honigsbaum, M. (2010), 'The Great Dread: Cultural and Psychological Impacts and Responses to the 'Russian' Influenza in the United Kingdom, 1889–1893', *Social History of Medicine*, 23(2): 299–319.

Hooker, C. & A. Bashford (2002), 'Diphtheria and Australian Public Health: Bacteriology and its Complex Applications, c.1890–1930', *Medical History*, 46: 41–64.

Hopper, J. L. (1986), 'The Contribution of W. H. Archer to Vital Statistics in the Colony of Victoria', *Australian Journal of Statistics*, 28(1): 124–137.

Human Mortality Database (2011), University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany), Available at www.mortality.org, (data downloaded on 4 August 2011).

Hunt, S.-J. & G. Bolton (1978), 'Cleansing the Dunghill: Water Supply and Sanitation in Perth 1878-1912', *Studies in Western Australian History*, 2: 1-17.

Jackson, R. V. & M. Thomas (1995), 'Height, Weight and Wellbeing: Sydney Schoolchildren in the Early Twentieth Century', *Australian Economic History Review*, 35: 39–65.

Jaunay, G. (2004), *Unregistered Deaths in South Australia From July 1842 (CD-ROM)*, Adelaide Proformat, Adelaide, www.jaunay.com/unreg.html.

Johansson, S. Ryan (1994), 'Food For Thought: Rhetoric and Reality in Modern Mortality History', *Historical Methods*, 27: 101–125.

Jones, F. L. (1989), 'Occupational Prestige in Australia. A New Scale', *Australian and New Zealand Journal of Sociology*, 25(2): 187–199.

Kellaway, R. G. (1989), 'The Hobart Typhoid Epidemic of 1887–88', *Social Science and Medicine*, 29(8): 953–958.

Kelly, M. (1978), 'Picturesque and Pestilential: The Sydney Slum Observed', In: Kelly, M. (ed.), *Nineteenth-Century Sydney: Essays in Urban History*, Sydney University Press, Sydney.

Kim, H.J., M. P. Fay, E. J. Feuer & D. N. Midthune (2000), 'Permutation Tests for Joinpoint Regression With Applications to Cancer Rates', *Statistics in Medicine*, 19: 335–351 (correction: 2001;20:655).

Kintner, H. (1986), 'Classifying Causes of Death During the Late Nineteenth and Early Twentieth Centuries: The Case of German Infant Mortality', *Historical Methods*, 19: 45–54.

Kippen, R. (2002a), *Death in Tasmania: Using Civil Registers to Measure Nineteenth-Century Cause-Specific Mortality*, PhD Thesis, The Australian National University, Canberra.

Kippen, R. (2002b), 'An Indispensable Duty of Government: Civil Registration in Nineteenth-Century Tasmania', *Tasmanian Historical Studies*, 8(1): 42–58.

Kippen, R. (2005), 'Counting Nineteenth-Century Maternal Deaths: The Case of Tasmania', *Historical Methods*, 38: 14–25.

Kippen, R. (2011), 'Incorrect, Loose and Coarse Terms: Classifying Nineteenth-Century English-Language Causes of Death for Modern Use. An Example Using Tasmania Data', *Journal of Population Research*, 28: 267–291.

Kirk, D. (1996), 'Demographic Transition Theory', *Population Studies*, 50: 361–387.

Knibbs, G. H. (1929), 'The International Classification of Disease and Causes of Death and its Revision', *Medical Journal of Australia*, 1: 2–12.

Kunitz, S. J. (1983), 'Speculations on the European Mortality Decline', *Economic History Review*, 36: 349–364.

Kunitz, S. J. (1991), 'The Personal Physician and the Decline of Mortality in the Late Nineteenth and Early Twentieth Centuries', In: R. Schofield, D. Reher & A. Bideau *eds.*, *The Decline of Mortality in Europe*, Clarendon Press: Oxford.

Kunitz, S. J. (1999), 'Premises, Premises: Comments on the Comparability of Classifications', *Journal of the History of Medicine*, 54: 226–240.

Kunitz, S. J. (2007), *The Health of Populations, General Theories and Particular Realities*, Oxford University Press: New York.

Lancaster, H. O. (1950a), 'Cancer Mortality in Australia', *Medical Journal of Australia*, 2: 501–506.

Lancaster, H. O. (1950b), 'Tuberculosis Mortality in Australia, 1908 to 1945', *Medical Journal of Australia*, 2: 655–662.

Lancaster, H. O. (1958), 'Cancer Statistics in Australia. Part 1', *Medical Journal of Australia*, 2: 350–356.

Lancaster, H. O. (1960), 'Australian Mortality in the Late Nineteenth Century', *Medical Journal of Australia*, 2: 84–87.

Lancaster, H. O. (1964), 'The Mortality From Violence in Australia, 1863 to 1960', *Medical Journal of Australia*, 1: 388–393.

Larson, A. (1994), *Growing Up in Melbourne: Family Life in the Late Nineteenth Century*, Australian Family Formation Project Monograph No. 12, Demography Program, Australian National University, Canberra.

Last, J. M. (1988), *A Dictionary of Epidemiology, second edition*, Oxford University Press, New York.

Lawrence, R. J. (2009), *Health Determinants in Urban Areas*, In: Cook, H. J., S. Bhattacharya & A. Hardy (*eds.*), *History of the Social Determinants of Health: Global Histories, Contemporary Debates*, Orient BlackSwan, Hyderabad.

Lee, R. & D. Lam (1983), 'Age Distribution Adjustments for English Census, 1821 to 1931', *Population Studies*, 37(3): 445–464.

Leppard, P. I. (2002), *An Analysis of Population Lifetime Data of South Australia 1841–1996*, M.Sc. Thesis, School of Applied Mathematics, The University of Adelaide, Adelaide.

Lewis, M. (1979), 'Sanitation, Intestinal Infections, and Infant Mortality in Late Victorian Sydney', *Medical History*, 23: 325–338.

Lewis, M. (1980), 'The Problem of Maternal Mortality and Population Growth in Australia, 1880–1939: Some Ideological Considerations', *Community Health Studies*, 4: 104–110.

Lewis, M. (1982), 'Some Infant Health Problems in Sydney, 1880–1939', *Journal of the Royal Australian Historical Society*, 68: 67–73.

Lewis, M. (2003), *The People's Health: Public Health in Australia, 1788–1950*, Praeger Publishers, Westport, CT.

Lewis, M. & R. MacLeod (1987), 'A Workingman's Paradise? Reflections on Urban Mortality in Colonial Australia 1860–1900', *Medical History*, 31: 387–402.

Lloyd, C., P. Troy & S. Schreiner (1992), *For the Public Health: The Hunter District Water Board 1892–1992*, Longman Cheshire, Melbourne.

London School of Hygiene and Tropical Medicine (2009), 'Avoidable Mortality in the European Union: Towards Better Indicators for the Effectiveness of Health Systems', amiehs.lshtm.ac.uk/, Accessed 30 April 2014.

Loudon, I. (1987), 'Puerperal Fever, the Streptococcus and the Sulfonamides, 1911–1945', *British Medical Journal*, 295 (No. 6596): 485–490.

Loudon I. (1991), 'On Maternal and Infant Mortality 1900–1960', *Social History of Medicine*, 4: 29-73.

Loudon, I. (1992), 'The Transformation of Maternal Mortality', *British Medical Journal*, 305 (No. 6868): 1557–1560.

Loudon, I. (1997), 'Childbirth', In: *Western Medicine, An Illustrated History*, Oxford University Press, Oxford.

Luckin, B. (1980), 'Death and Survival in the City: Approaches to the History of Disease', *Urban History*, 7: 53–62.

Mackenbach, J. P. (1994), 'The Epidemiologic Transition Theory', *Journal of Epidemiology and Community Health*, 48: 329–331.

Mackenbach, J. P. (1996), 'The Contribution of Medical Care to Mortality Decline: McKeown Revisited', *Journal of Clinical Epidemiology*, 49: 1207–1213.

Mayne, A. J. C. (1982), *Fever, Squalor and Vice: Sanitation and Social Policy in Victorian Sydney*, University of Queensland Press, St Lucia.

McCalman, J. (1984), *Struggletown, Public and Private Life in Richmond, 1900–1965*, Melbourne University Press, Carlton, Victoria.

McCalman, J. & R. Morley (2003), 'Mother's Health and Babies' Weights: the Biology of Poverty at the Melbourne Lying-In Hospital, 1857–83', *Social History of Medicine*, 16: 39–56.

McCalman, J. & R. Morley (2008), 'Inequalities of Gender and Health 1857-1985: A Long-run Perspective from the Melbourne Lying-In Hospital Birth Cohort', *Australian Journal of Social Issues*, 43: 29-44.

McCalman, J., L. Smith, I. Anderson, R. Morley & G. Mishra (2009), 'Colonialism and the Health Transition: Aboriginal Australians and Poor Whites Compared, Victoria, 1850-1985', *History of the Family*, 14: 253–265.

McCalman, J., R. Morley, L. Smith & I. Anderson (2011), 'Colonial Health Transitions: Aboriginal and 'Poor White' Infant Mortality Compared, Victoria 1850–1910', *History of the Family*, 16: 62–77.

McCarty, J. W. (1970), 'Australian Capital Cities in the Nineteenth Century', *Australian Economic History Review*, 10: 107-137.

McCracken, K. & D. R. Phillips (2012), *Global Health: An Introduction to Current and Future Trends*, Routledge, London & New York.

McKeown, T. (1976), *The Modern Rise of Population*, Edward Arnold, London.

McKeown, T. & R. Brown (1955), 'Medical Evidence Related to English Population Changes in the Eighteenth Century', *Population Studies*, 9: 119–141.

McKeown, T. & R. Record (1962), 'Reasons For The Decline of Mortality in England and Wales During the Nineteenth Century', *Population Studies*, 16: 94–122.

McNeil, D., T. J. Trussell & J. C. Turner (1977), 'Spline Interpolation of Demographic Data', *Demography*, 14: 245–252.

Mein Smith, P. (1997), *Mother and King Baby: Infant Survival and Welfare in an Imperial World: Australia 1880–1950*, MacMillan Press Ltd, London.

Meslé, F. & J. Vallin (1996), 'Reconstructing Long-Term Series of Causes of Death', *Historical Methods*, 29: 72–86.

Mirzaei, M., A. S. Truswell, R. Taylor & S. R. Leeder (2009), 'Coronary Heart Disease Epidemics: Not All The Same', *Heart*, 95: 740–746.

Mooney, G. (2007), 'Infectious Diseases and Epidemiologic Transition in Victorian Britain? Definitely', *Social History of Medicine*, 20: 595–606.

Mooney, G., B. Luckin & A. Tanner (1999), 'Patient Pathways: Solving the Problem of Institutional Mortality in London During the Later Nineteenth Century', *Social History of Medicine*, 12(2): 227–269.

Moriyama, I. M., R. M. Loy & A. H. T. Robb-Smith (2011), *History of the Statistical Classification of Diseases and Causes of Death*, National Center for Health Statistics, Hyattsville, M.D.

Morley, R., J. McCalman & J. Carlin (2003), 'Trends in Birthweight Between 1857 and 1883, in Melbourne, Australia', *Paediatric and Perinatal Epidemiology*, 17: 236–243.

Morley, R., J. McCalman & J. B. Carlin (2006), 'Birthweight and Coronary Heart Disease in a Cohort Born 1857–1900 in Melbourne, Australia', *International Journal of Epidemiology*, 35: 880–885.

Morrell, S., A. Page & R. Taylor (2002), 'Birth Cohort Effects in New South Wales Suicide, 1865–1998', *Acta Psychiatrica Scandinavica*, 106: 365–372.

Murray, C. J. L. & A. D. Lopez *eds.* (1996), *The Global Burden of Disease*, The Harvard School of Public Health on behalf of the World Health Organization, Boston, Mass.

Murray, C. J. L., J. K. Rajaratnam, J. Marcus, T. Laakso & A. D. Lopez (2010), 'What Can We Conclude From Death Registration? Improved Methods For Evaluating Completeness', *PLOS Medicine*, 7: e1000262.

Naghavi, M., S. Makela, K. Foreman, J. O'Brien, F. Pourmalek & R. Lozano (2010), 'Algorithms For Enhancing Public Utility of National Causes-of-Death Data', *Population Health Metrics*, 8:9.

NCI (National Cancer Institute) (2014). Joinpoint Regression Program, Version 4.1.0, surveillance.cancer.gov/joinpoint/, Accessed 30 April 2014.

Newman, T. (2005), *Becoming Tasmania: Renaming Van Diemen's Land*, Parliament of Tasmania, Hobart.

Noymer, A. & B. Jarosz (2008), 'Causes of Death in Nineteenth-Century New England: The Dominance of Infectious Disease', *Social History of Medicine*, 21: 573–578.

Olshansky, S. J. & A. B. Ault (1986), 'The Fourth Stage of the Epidemiologic Transition: The Age of Delayed Degenerative Diseases', *The Milbank Quarterly*, 64: 355–391.

Olshansky, S. J., B. Carnes, R. G. Rogers & L. Smith (1997), 'Infectious Diseases—New and Ancient Threats to World Health', *Population Bulletin*, 52: 1–52.

Omran, A. R. (1971), 'The Epidemiologic Transition: A Theory of the Epidemiology of Population Change', *Milbank Memorial Fund Quarterly*, 49: 509–538.

Omran, A. R. (1983), 'The Epidemiological Transition Theory. A Preliminary Update', *Journal of Tropical Pediatrics,* 29: 305–316.

Omran, A. R. (1998), 'The Epidemiologic Transition Theory Revisited Thirty Years Later', *World Health Statistics Quarterly*, 51: 99–119.

ONS (Office for National Statistics) (2003), *Life Expectancy at Birth: Methodological Options for Small Populations*, National Statistics Methodological Series No.33, Norwich: HMSO.

ONS (2010), *Life Table Templates*, <www.ons.gov.uk/ons/search/index.html?pageSize=50&newquery=ONS+Template+Life+table>, Accessed 18 September 2010.

Pearson, M. (2005), *Great Southern Land: the Maritime Exploration of Terra Australis*, Department of the Environment and Heritage, Canberra.

Pike, D. (ed.) (1966), 'Fisher, Sir James Hurtle (1790–1875)', in *Australian Dictionary of Biography, Volume 1*, Melbourne University Press, Melbourne, pp.379–381.

Pike, D. (1967), *Paradise of Dissent, South Australia 1829–1857*, Melbourne University Press, Melbourne.

Preston, S. H. (1975), 'The Changing Relation Between Mortality and Level of Economic Development', *Population Studies*, 29: 231–248.

Preston, S. H. (1976), *Mortality Patterns in National Populations: With Special Reference to Recorded Causes of Death*, Academic Press, New York.

Preston, S. H., P. Heuveline & M. Guillot (2001), *Demography: Measuring and Modelling Population Processes*, Blackwell, Oxford.

Public Record Office Victoria (2011), Public Record Office Victoria Online Catalogue, www.access.prov.vic.gov.au, Accessed 11 January 2011.

Quiggin, P. (1988), *No Rising Generation: Women & Fertility in Late Nineteenth Century Australia*, Department of Demography, Australian National University, Canberra.

Rey, G., A. Aouba, G. Pavillon, R. Hoffmann, I. Plug, R. Westerling, E. Jougla & J. Mackenbach (2011), 'Cause-specific Mortality Time Series Analysis: A General Method to Detect and Correct for Abrupt Data Production Changes', *Population Health Metrics*, 9: 52.

Riley, J. C. (2005), 'The Timing and Pace of Health Transitions Around the World', *Population and Development Review*, 31: 741–764.

Risse, G. (1997), 'Cause of Death as a Historical Problem', *Continuity and Change*, 12: 175–188.

Robb-Smith, A. H. T. (1971), 'A History of the College's Nomenclature of Diseases: Its Reception', *Journal of the Royal College of Physicians*, 4: 5–26.

Rogers, J. & M. Nelson (1997), 'The Epidemiologic Transition Revisited: Or What Happens If We Look Beneath the Surface?', *Health Transition Review*, 7: 240–254.

Rosen, G. (1958, 1993), *A History of Public Health, Expanded Edition*, Johns Hopkins University Press, Baltimore.

Ruzicka, L. & J. C. Caldwell (1977), *The End of Demographic Transition in Australia*, Australian Family Formation Project Monograph No. 5, Department of Demography, Australian National University, Canberra.

Saltelli, A., M. Ratto, T. Andres, F. Campolongo, J. Cariboni, D. Gatelli, M. Saisana & S. Tarantola (2008), *Global Sensitivity Analysis. The Primer*, John Wiley & Sons Ltd.: Chichester.

Schofield, R. & D. Reher (1991), 'The Decline of Mortality in Europe', In: Schofield, R., D. Reher & A. Bideau *eds.*, *The Decline of Mortality in Europe*, Clarendon Press: Oxford.

Seneta, E. (1988), 'Silhouettes in Early Australian Statistics', *Australian Journal of Statistics*, 30(B): 2–22.

Seneta, E. & G. K. Eagleson (2004), 'Henry Oliver Lancaster 1913-2001', *Historical Records of Australian Science*, 15: 223-250.

Shryock, H. S. & J. S. Siegel (1976), *The Methods and Materials of Demography, condensed edition*, Academic Press, San Diego.

Sinclair, W. (1975), 'Economic Growth and Well-being: Melbourne 1870–1914', *The Economic Record*, 51: 153–173.

Smith, F. B. (1979), *The People's Health 1830–1910*, Holmes & Meier Publishers, Inc., New York.

Smith, F. B. (1997), 'The First Health Transition in Australia, 1880–1910', in: Jones, G. W. *et al.* (*eds.*), *The Continuing Demographic Tradition*, Clarendon Press, Oxford.

Smith, F. B. (1999), 'Comprehending Diphtheria', *Health and History*, 1: 139–161.

Smith, F. B. (2002), 'Disputes About Typhoid Fever in Victoria in the 1870s', *Health and History*, 4: 1–18.

Smith, F. B. (2011), *Illness in Colonial Australia*, Australian Scholarly Publishing, North Melbourne.

Smith, L., R. J. Hyndman & S. J. Wood (2004), 'Spline Interpolation For Demographic Variables: The Monotonicity Problem', *Journal of Population Research*, 21: 95–98.

Smith, L., *et al.* (2008), 'Fractional Identities: The Political Arithmetic of Aboriginal Victorians', *Journal of Interdisciplinary History*, 38: 533–551.

SRS1 Software (2010), *Cubic Spline for Excel 1.02*, SRS1 Software LLC, www.srs1software.com, Accessed 18 September 2010.

Steckel, R. H. (2008), *Heights and Human Welfare: Recent Developments and New Directions*, NBER Working Paper 14536, National Bureau of Economic Research, Cambridge, MA.

Stevenson, T. (1981), 'Miasmas, Morbidity and Milieu: Mortality in Victorian South Australia', *Proceedings of the Royal Geographical Society of Australasia (S.A. Branch)*, 81:40–58.

Stolnitz, G. J. (1955), 'A Century of International Mortality Trends: I', *Population Studies*, 9: 24–55.

Sundlin, J. & S. Willner (2009), 'The History of Social Determinants and Health in Europe: A Swedish Example', In: Cook, H. J., S. Bhattacharya & A. Hardy (*eds.*), *History of the Social Determinants of Health: Global Histories, Contemporary Debates*, Orient BlackSwan, Hyderabad.

Szreter, S. (1988), 'The Importance of Social Intervention in Britain's Mortality Decline c.1850–1914: A Re-interpretation of the Role of Public Health', *Social History of Medicine*, 1: 1–37.

Szreter, S. (1992), 'Mortality and public health, 1815–1914', *ReFresh, Recent Findings of Research in Economic and Social History*, 14: 1–4.

Szreter, S. (1997), 'Economic Growth, Disruption, Deprivation, Disease, and Death: On the Importance of the Politics of Public Health for Development', *Population and Development Review*, 23: 693–728.

Szreter, S. (1999), 'Rapid Economic Growth and the 'Four Ds' of Disruption, Deprivation, Disease and Death, Public Health Lessons From Nineteenth-Century Britain for Twenty-First-Century China?', *Tropical Medicine and International Health*, 4: 146–152.

Szreter, S. (2004), 'Industrialization and Health', *British Medical Bulletin*, 69: 75–86.

Taylor, F. (1992), *Guide to Use of International Classifications of Diseases in Australia*, AGPS, Canberra.

Taylor, R., M. Lewis & J. Powles (1998a), 'The Australian Mortality Decline: All-Cause Mortality 1788–1990', *Australian and New Zealand Journal of Public Health*, 22: 27–36.

Taylor, R., M. Lewis, & J. Powles (1998b), 'The Australian Mortality Decline: Cause-Specific Mortality 1907–1990', *Australian and New Zealand Journal of Public Health*, 22: 37–44.

United Nations (1955), *Methods of Appraisal of Quality of Basic Data for Population Estimates*, Manual II, Population Studies No. 23, United Nations, New York.

United Nations, Department of Economic and Social Affairs, Population Division (2012), *Changing Levels and Trends in Mortality: The Role of Patterns of Death by Cause*, United Nations, New York.

U.S. Census Bureau (2014), *International Programs, Population Analysis Spreadsheets (PAS)*, www.census.gov/ipc/www/pas.html

Vallin, J. (1991), 'Mortality in Europe From 1720 to 1914. Long-Term Trends and Changes in patterns by Age and Sex', In: Schofield, R., D. Reher & A. Bideau *eds.*, *The Decline of Mortality in Europe*, Clarendon Press: Oxford.

Vallin, J. & F. Meslé (1990), 'The Causes of Death in France, 1925–1978: Reclassification According to the Eighth Revision of the International Classification of Diseases', in J. Vallin, S. D'Souza & A. Palloni (*eds.*), *Measurement and Analysis of Mortality: New Approaches*, Clarendon Press, Oxford.

Vamplew, W. (ed.) (1987), *Australians: Historical Statistics*, Fairfax, Syme & Weldon Associates, Broadway, New South Wales.

van Poppel, F. & C. van der Heijden (1997), 'The Effects of Water Supply on Infant and Childhood Mortality: A Review of Historical Evidence', *Health Transition Review*, 7: 113–148.

van Poppel, F. *et al.* (2012), 'Mortality Decline and Reproductive Change During the Dutch Demographic Transition: Revisiting a Traditional Debate With New Data', *Demographic Research*, 27/11: 299–338.

Walker, R. (1983), 'The Struggle Against Pulmonary Tuberculosis in Australia, 1788–1950', *Historical Studies*, 20(80): 439–461.

Weisz, G. & J. Olszynko-Gryn, 2010, 'The Theory of Epidemiologic Transition: The Origins of a Citation Classic', *Journal of the History of Medicine*, 65:287–326.

Whitwell, G., C. de Souza & S. Nicholas (1997), 'Height, Health, and Economic Growth in Australia, 1860–1940', In: R. H. Steckel & R. Floud (eds), *Health and Welfare During Industrialization*, University of Chicago Press, Chicago, pp. 379–422.

Whitwell, G. & S. Nicholas (2001), 'Weight and Welfare of Australians, 1890-1940', *Australian Economic History Review*, 41: 159–175.

WHO (World Health Organization) (2008), *Closing the Gap in a Generation. Commission on Social Determinants of Health Final Report*, WHO, Geneva.

WHO (2012), *Trends in Maternal Mortality: 1990 to 2010*, WHO, Geneva.

Wickens, C. (1930), 'Australian Mortality', *Journal of the Institute of Actuaries*, 61: 165–213.

Wilkinson, R. & M. Marmot (*eds.*) (2003), *Social Determinants of Health: The Solid Facts, 2nd edition*, WHO, Copenhagen.

Williams, N. (1996), 'The Reporting and Classification of Causes of Death in mid-Nineteenth Century England: The Example of Sheffield', *Historical Methods*, 29: 58–72.

Wolleswinkel-van den Bosch, J., F. van Poppel & J. P. Mackenbach (1996), 'Reclassifying Causes of Death to Study the Epidemiological Transition in the Netherlands, 1875–1992', *European Journal of Population*, 12: 327–361.

Wolleswinkel-van den Bosch, J., F. van Poppel, E. Tabeau & J. P. Mackenbach (1998), 'Mortality Decline in the Netherlands in the Period 1850–1992: A Turning Point Analysis', *Social Science and Medicine*, 47: 429–443.

Wong, A. (1999), 'Colonial Sanitation, Urban Planning and Social Reform in Sydney, New South Wales 1788–1857', *Australian Historical Archaeology,* 17: 58–69.

Woods, R. (2000), *The Demography of Victorian England and Wales*, Cambridge University Press, Cambridge.

Woods, R. (2007), 'Medical and Demographic History: Inseperable?', *Social History of Medicine*, 20: 483–503.

Woods, R. (2009), *Death Before Birth: Fetal Health and Mortality in Historical Perspective*, Oxford University Press, Oxford.

Woods, R. & P. R. Andrew Hine (1987), 'Mortality in Victorian England: Models and Patterns', *Journal of Interdisciplinary History*, 18: 27–54.

Woods, R. & N. Shelton (2000), 'Disease Environments in Victorian England and Wales', *Historical Methods*, 33: 73–81.

Woolcock, H. R., M. J. Thearle & K. Saunders (1997), 'My Beloved Chloroform. Attitudes to Childbearing in Colonial Queensland: A Case Study', *Social History of Medicine,* 10: 437–457.

Wrigley, E. A. (1977), 'Births and Baptisms: The Use of Anglican Baptism Registers as a Source of Information about the Numbers of Births in

England before the Beginning of Civil Registration', *Population Studies,* 31(2): 281–312.

Wrigley, E. A. & R. S. Schofield (1989), *The Population History of England 1541– 1871*, Cambridge University Press, Cambridge.

Wrigley, E. A., R. S. Davies, J. E. Oeppen & R. S. Schofield (2005), *English Population History from Family Reconstitution 1580-1837*, Cambridge University Press, Cambridge.

Zhao, Z. (2010), 'Historical demography (English Version)', in: Y. Zeng (ed.) *Demography, in Encyclopedia of Life Support Systems (EOLSS)*, Developed under the Auspices of the UNESCO, EOLSS Publishers, Oxford, UK. <http://www.eolss.net/>.