

Epidemiologic Transition in Australia: The last hundred years

Heather Booth¹

Leonie Tickle

Jiaying Zhao

Abstract

Mortality change in Australia since 1907 is analysed in the light of Epidemiologic Transition theory. Australia began the twentieth century in the second age of the Epidemiologic Transition, the Age of Receding Pandemics. Australia probably moved to the third, the Age of Degenerative and Man-Made Diseases before 1946, which is slightly in advance of most Western countries. Transition to the fourth, the Age of Delayed Degenerative Diseases, is clearly marked by a downturn, in about 1970, in circulatory disease mortality, concurrent with other Western countries.

Keywords: mortality, trends, decomposition, life expectancy, differentials. Australia.

Résumé

La théorie de la transition épidémiologique sert de base pour une analyse des changements de mortalité en Australie depuis 1907. Au début du XXe siècle, l'Australie était dans la deuxième phase de la transition épidémiologique, celle du recul des pandémies. Néanmoins, l'Australie entrait probablement avant 1946 dans la troisième phase, celle des maladies dégénératives, ce qui est légèrement en avance sur la plupart des pays occidentaux. La transition vers la quatrième phase, celle des maladies dégénératives retardées, est clairement marquée par un ralentissement depuis environ 1970 dans la mortalité par maladies circulatoires, en même temps que chez d'autres pays occidentaux.

Mots-clés : mortalité, tendances, décomposition, espérance de vie, écarts, Australie.

Introduction

Australia enjoys a life expectancy that is among the highest in the world. In 2011–13, life expectancy at birth among females was 84.3 years, and 80.1 years among males (ABS 2014a). The recently-released United Nations *World Population Prospects 2015* (UNPD 2015) shows that for life expectancy at birth in 2010–15, Australian males rank eighth internationally and Australian females rank tenth. The top five ranked countries for males are Hong Kong, Iceland, Switzerland, Italy, and Israel, and for females they are Hong Kong, Japan, Singapore, Italy, and Spain.

1. Corresponding author: Prof. Heather Booth, School of Demography, The Australian National University, Canberra ACT 2601, Australia, e-mail: heather.booth@anu.edu.au; Prof. Leonie Tickle, Faculty of Business and Economics, Macquarie University, Sydney; and Dr. Jiaying Zhao, School of Demography, The Australian National University.

This paper examines changing mortality in Australia since 1907, six years after the Commonwealth of Australia was created. The focus is at the national level, with some discussion of differentials. The theoretical framework of the analysis is the Epidemiologic Transition (Omran 1971; Olshansky and Ault 1986). As noted by de Looper (2015), the more recent Epidemiologic Transition in Australia has not been addressed as such, though studies of twentieth-century mortality decline there do exist (e.g., Taylor and Lewis 1998; Taylor et al. 1998; Booth 2003). This paper remedies the omission.

The paper is organized as follows. After a discussion of Epidemiologic Transition theory and a description of the data and methods employed, the paper examines trends in life expectancy at birth and at selected ages by sex. Cause of death, in conjunction with age, is then explored through a series of decompositions of temporal change in life expectancy over the course of the lengthy period considered. The following section addresses age patterns of change and focuses on infant mortality, the adolescent and young adult mortality hump, and old age mortality. The penultimate section presents geographic, indigeneity and socio-economic mortality differentials, and the paper concludes with a discussion of the findings in relation to Epidemiologic Transition theory.

Epidemiologic Transition theory

The theory of Epidemiologic Transition (Omran 1971, 1983) describes health changes during the process of modernisation as a series of three successive stages of transition or 'Ages'. The first is the 'Age of Pestilence and Famine', characterised by low and fluctuating life expectancy in the range 20–40 years. The second is the 'Age of Receding Pandemics' when life expectancy increases steadily from an average of about 30 years to 50 (Omran 1971) or 55 (Omran 1983) years, largely as a result of less frequent epidemics and the decline of infectious diseases; the underlying causes were primarily socio-economic, 'augmented by the sanitary revolution in the late nineteenth century and by medical and public health progress in the twentieth century' (Omran 1971, reprint p.753). The third 'Age of Degenerative and Man-Made Diseases' is characterised by a slow increase in life expectancy due to the balancing effects of the disappearance of infectious diseases and the rise of 'degenerative and man-made' or non-communicable diseases such as heart disease, stroke, cancers, and external causes. At the time of publication of the theory, the general consensus was that there was a limit to life expectancy which would soon be reached (see Meslé and Vallin 2011); for example, United Nations (1975) took this limit to be 75 years.

In response to renewed mortality decline from the 1970s, Olshansky and Ault (1986) proposed a fourth 'Age of Delayed Degenerative Diseases' characterised by the decline of cardiovascular and other non-communicable diseases at increasingly older ages, due to advances in medical technology and improved health programs. Rogers and Hackenberg (1987) also proposed a fourth 'hubristic' (or 'hybristic') stage giving prominence to the decline of social pathologies arising from individual behaviour and lifestyle, which are driven by 'hubris' or notions of excessive self-confidence and invincibility. These two proposed extensions of Omran's Epidemiologic Transition theory address different aspects of the same stage.² The Epidemiologic Transition theory has been criticised by Robine (2001) and by Meslé and Vallin (2006), particularly in regard to the distinction between the third and fourth Ages.

Omran (1971) defined three models of Epidemiologic Transition, in recognition of the differing dates of onset and speeds of transition among countries. The Classical or Western model applies to the populations of Europe and North America. Compared with this, the Accelerated model involves

2. Proposals of fifth and sixth stages exist; these are not considered.

a more rapid transition such as occurred in Japan. The Contemporary or Delayed model applies to the populations of developing countries.

While Epidemiologic Transition theory was developed to explain global patterns, it can be used in the study of mortality decline in individual countries (e.g., Caselli, Meslé and Vallin 2002; Lussier, Bourbeau and Choinière 2008). Several exceptions to the overall theory have been identified (Caselli, Meslé and Vallin 2002). A limitation is that Omran did not provide clear guidelines to determine when successive Ages begin and end (Mackenbach 1994). It has been argued that the approach is overly broad, and that there is a need to take greater account of how population subgroups experience epidemiologic transitions differently (Gaylin and Kates 1997).

The early years of the Epidemiologic Transition in the settler³ population of Australia have been comprehensively documented by de Looper (2015) who notes that the Age of Pestilence and Famine was absent in Australia.⁴ Though there was no shortage of epidemics in the second half of the nineteenth century, famine was almost entirely absent, and life expectancy was always above the defining threshold of 40 years for transition to the second stage (Omran 1971). Thus, the second Age of Receding Pandemics characterises the start of the ‘truncated’ Epidemiologic Transition in Australia, confirmed by life expectancies in the 1860s of 45 years for males and 49 years for females. Further, de Looper (2015) concluded that, although there was rapid mortality decline in the period 1885 to 1903, there was no evidence of transition to the third Age of Degenerative and Man-Made Diseases because the major causes of death (infectious diseases, non-communicable diseases, and external causes) declined proportionately. Commencing in 1907, this analysis therefore begins in the second Age of Receding Pandemics.

Data and methods

Data for international trends and comparisons of life expectancies are from the Human Mortality Database (HMD 2015). The analyses use five-year averages from 1920–24 to the present. For Australia, HMD covers 1921 to 2011, so that the first period is 1921–24. The countries for comparison are Canada, England and Wales, France, Japan, and the United States, selected on the basis of high income and either historical links and cultural similarities to Australia (Canada, England and Wales, United States) or recent leading-edge mortality experience (France, Japan). HMD data are also used for the examination of trends at specific ages.

Australian cause of death data are from the Australian Institute of Health and Welfare (AIHW) General Record of Incidence of Mortality (GRIM) books (AIHW 2015a), which contain mortality rates by five-year age groups for ages 0 to 84 and for the age 85+, from 1907 to 2012. Over this period, the International Classification of Diseases underwent numerous revisions (WHO 1992), leading to inconsistencies in cause of death classification and discontinuities in time series of data. These potential problems have been largely mitigated in this analysis by considering only the major cause of death categories. The six major causes of death employed are *infectious diseases*, *neoplasms*, *circulatory diseases*, *respiratory diseases*, *external causes*, and *‘all other’* causes. Note that in the early part of the century,

3. The first British settlers arrived in Australia in 1788. Comprehensive mortality data have been compiled from 1856, when registration began, by de Looper (2015).

4. Smith (1980) and Gray (1985) suggest that the historic Indigenous population was stationary prior to settlement. Therefore, this population would not have been subject to the fluctuation defining the Age of Pestilence and Famine. Thus, neither the Indigenous nor settler population appears to have experienced Omran’s first Age.

many ‘all other’ causes of death were indicated as ‘ill-defined’, but this classification was reduced to near zero by 1960 (Lancaster 1990).

The cause of death analysis uses standardized mortality rates and life expectancy decomposition. Standardized mortality rates are computed by sex and the six major causes of death, using the 1981 total Australian population (both sexes) by five-year age groups as the standard. Life expectancy decomposition uses the Arriaga (1984) method to attribute differences in life expectancy at birth to mortality change by age and major cause simultaneously. To facilitate discussion of the Epidemiologic Transition, decomposition analyses were conducted for four periods:⁵ 1922–46, 1946–70, 1970–94, and 1994–2011. The periods were identified on the basis of internal consistency of patterns; that they are of roughly equal length assists in their comparison.

Trends in life expectancy

Australia in international context

Figure 1 compares historic male and female Australian life expectancies at birth with those of Canada, England and Wales, France, Japan, and the United States. The upward trends confirm the experience of Epidemiologic Transition. Deaths of Australian military personnel during World War II were excluded from national mortality statistics (Taylor et al. 1998), accounting for the absence of a downward spike in male life expectancy observed for some other countries.

In 1921–24, life expectancy in Australia was the highest among the six selected countries. Figure 1 shows that for both males and females, Australian life expectancy exceeded that of the second-highest of this group by as much as four years. Over the next two to three decades, this advantage diminished, and in the 1950s and 1960s Australia fell behind other countries. In the 1960s, for males, only US life expectancy was less than Australian life expectancy, while for females Australian life expectancy was as low as any other at this time. Australian life expectancy has since recovered, ranking first among the selected group for males, and third for females, in the most recent period.

It may be observed in Figure 1 that the life expectancies of the selected countries largely converged in the 1960s. Convergence among the five Western countries persisted for about two decades, and coincided with the period when Japan overtook the Western countries. In recent decades, all six countries have tended to diverge, with differences of as much as five years occurring in the most recent decade. This pattern of convergence and divergence is consistent with wider trends (Meslé and Vallin 2011). Cardiovascular diseases, which are of key importance in the transition from the Age of Degenerative and Man-Made Diseases to the Age of Delayed Degenerative Diseases, played a dominant role in both the convergence and divergence of countries over the entire period (Meslé and Vallin 2011).

Japanese life expectancy increased rapidly in the post–World War II years, in keeping with its characterization as undergoing Accelerated Epidemiologic Transition (Omran 1971; Zhao et al. 2014). Japan has been a leader in life expectancy since the 1970s, particularly for females. In 2011, Japanese females had a 1.6 year advantage over Australian females, though Japanese males were at a slight disadvantage compared with Australian males (HMD 2015). In contrast, the United States has generally ranked last—since the mid-1960s for males, and since the early 1990s for females (Figure 1); this has been attributed to higher prevalences of smoking, obesity, and violence, as well as restricted access to health care (Caselli et al. 2014: 231).

5. 1921 was omitted owing to a large increase in that year.

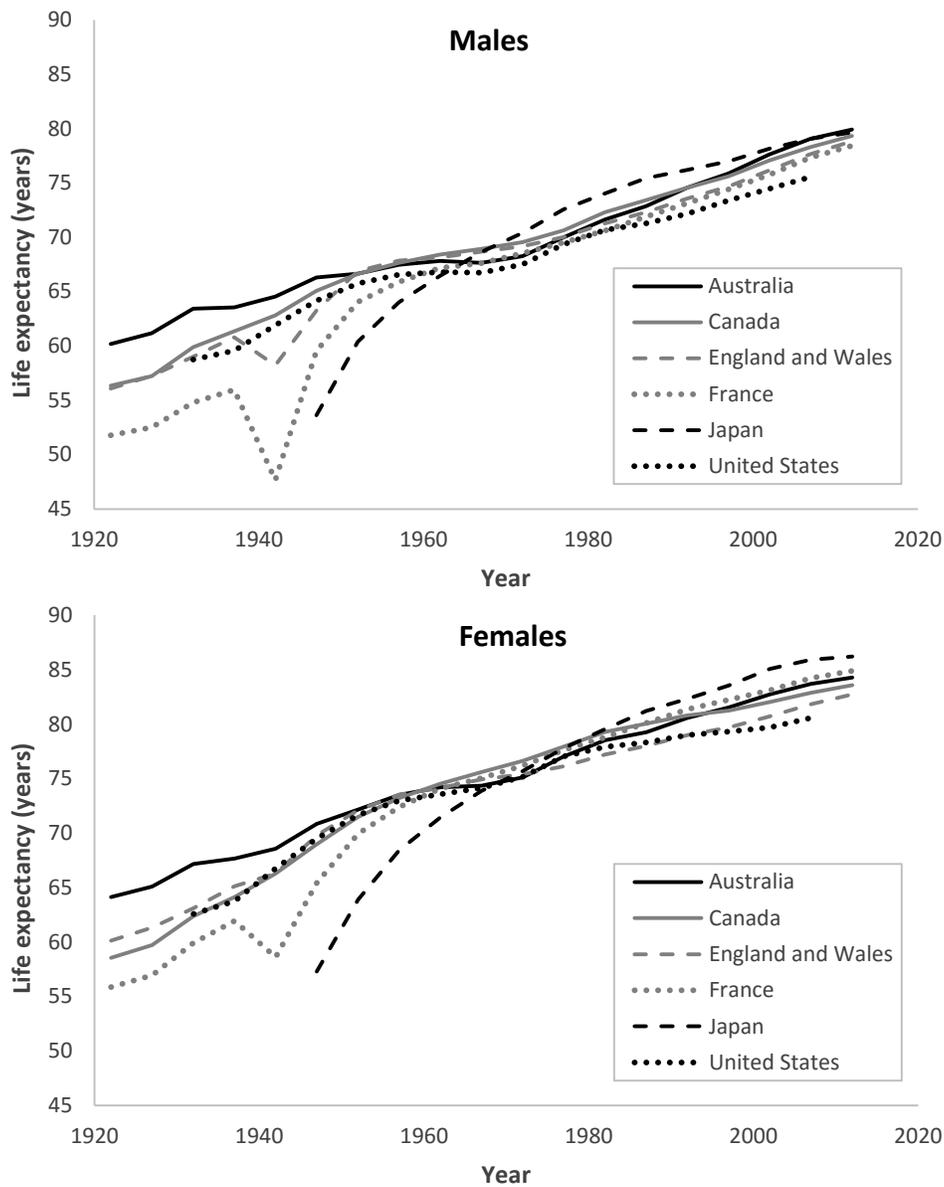


Figure 1. Life expectancy at birth for males and females, selected countries, 1920–24 to present.

Source: Human Mortality Database (HMD 2015) Life tables, five-year.

The sex difference in life expectancy at birth for the selected countries is shown in Figure 2. Ignoring war-related deviations, all countries experienced a general increase in the sex difference, followed by a downturn, as male improvements began to exceed female improvements. The turning point differs among countries, occurring first for England and Wales followed by the United States, Canada, and Australia, France, and finally (only around a decade ago) Japan. This same pattern has been found for high-income countries more generally, and has been attributed in most countries primarily to sex differences in the age pattern of mortality, rather than declining sex ratios in mortality (Glei and Horiuchi 2007). A decomposition analysis of the G7 countries over the three decades to 2000 found that the main causes of death contributing to narrowing of the sex difference were circulatory diseases and accidents, violence, and suicide (Trovato and Heyen 2006).

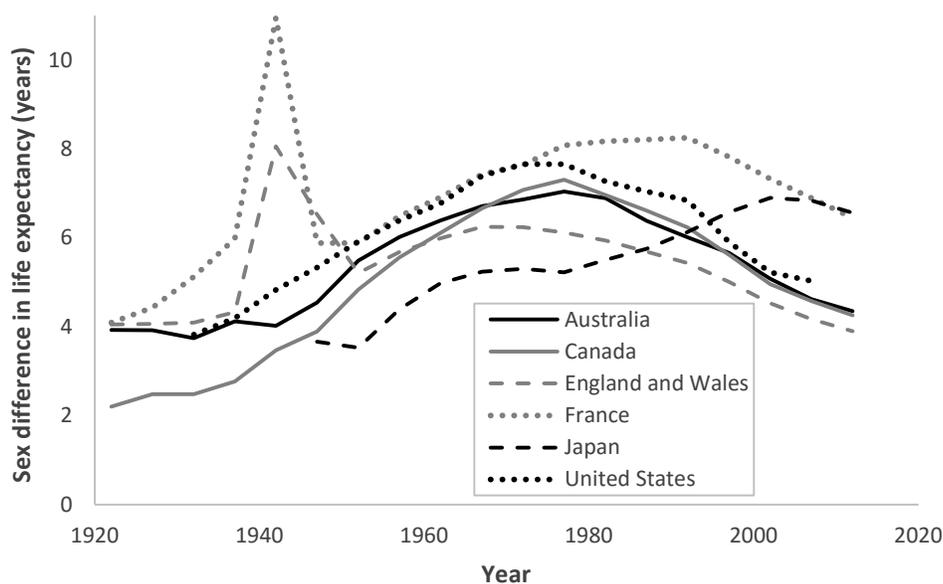


Figure 2. Sex difference in life expectancy at birth in years, selected countries, 1920–24 to present.

Source: Human Mortality Database (HMD 2015) Life tables, five-year.

Australia currently has a sex difference in life expectancy of 4.3 years; this is broadly similar to Canada, the United States, and England and Wales, and lower than France and Japan, where the turning points occurred later. In Australia, the sex difference increased from 3.9 years in 1921–24 to a maximum of 7.0 years in 1975–79, and has since declined linearly. More detailed decomposition analyses appear in Pollard (1996), Trovato and Lalu (1997), Booth (2003), and Tickle (2016).

The Australian experience

Over the period from 1921–24 to 2010–11, life expectancy at birth in Australia increased from 60.2 to 79.9 for males, an average of 2.2 years per decade, and from 64.1 to 84.3 for females, an average of 2.3 years per decade. However, as already indicated, improvements in life expectancy were not uniform over this period. After increases from 1921 to 1960 averaging 1.9 years per decade for males and 2.5 years per decade for females, mortality levels stagnated during the 1960s. The earlier, more rapid increases in life expectancy are characteristic of the second Age of Receding Pandemics, while the slow increases are characteristic of the third Age of Degenerative and Man-Made Diseases as described by Omran (1971). Mortality decline resumed in the early 1970s, with subsequent average improvement rates per decade of 3.0 years for males and 2.3 years for females. This post-1970 experience accords with the fourth Age of Delayed Degenerative Diseases, as described by Olshansky and Ault (1986).

Life expectancies for Australian males and females at ages 0, 50, 65, and 85 from 1921 to 2011 are shown in Figure 3 and Table 1. It is evident that virtually all of the improvement in male life expectancy at birth between 1921 and 1970 was due to mortality decline at ages less than 50: life expectancy at age 50 remained roughly constant over this period and actually declined during the 1930s and 1960s. In contrast, female life expectancy at age 50 improved before 1970, although the 1930s and 1960s were periods of stagnation. Since 1970, gains at the older ages have been rapid, particularly for males. This pattern is consistent with the Epidemiologic Transition, in that it describes mortality improvement as first occurring among children and young women, followed later by reductions in chronic and non-communicable diseases among older people. The age and cause-of-death groups

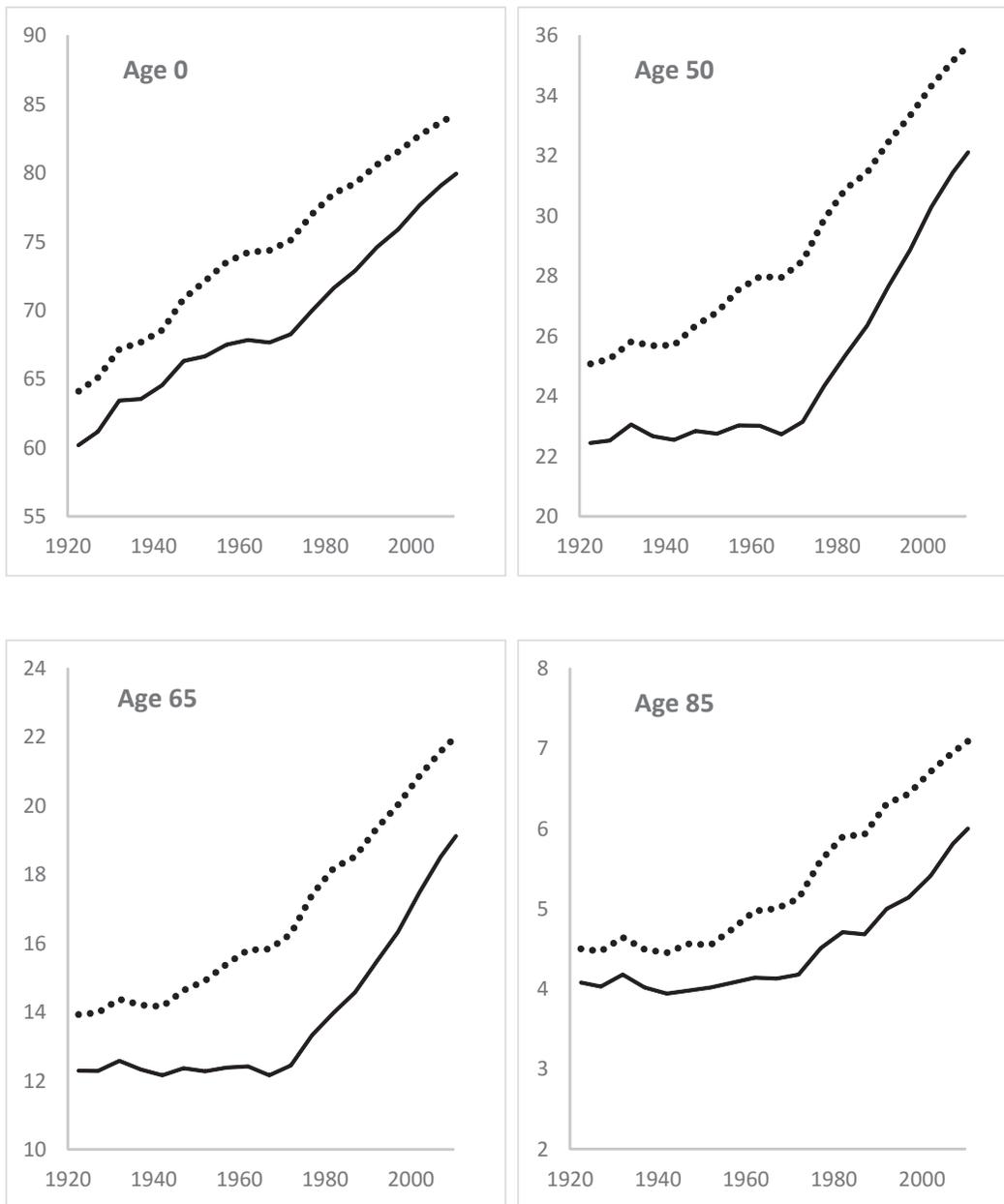


Figure 3. Life expectancy at ages 0, 50, 65 and 85 for males (solid line) and females (dotted line), Australia, 1921 to 2011.

Source: Human Mortality Database (HMD 2015) Life tables, five-year.

contributing to changing life expectancy in Australia are discussed in Section 5; for a more detailed analysis for the period since 1979, see Tickle (2016).

The widening and then narrowing pattern in the sex difference in Australian life expectancy at birth generally also applies at the older ages, as Figure 3 and Table 1 show. The sex differences in life expectancy at ages 0, 50, and 65 all increased to maxima—7.1, 5.7, and 4.2 years, respectively—at around 1980. In contrast, at the oldest ages increases in female advantage persisted longer; the sex difference in life expectancy at age 85 widened to a maximum of 1.4 years in the mid-1990s, before narrowing began.

Table 1. Life expectancy at ages 0, 50, 65 and 85 for males and females and the sex difference, Australia, selected years

Year	Life expectancy at											
	age 0			age 50			age 65			age 85		
	Male	Female	Diff.	Male	Female	Diff.	Male	Female	Diff.	Male	Female	Diff.
1922	60.9	65.1	4.2	22.6	25.3	2.7	12.4	14.0	1.6	4.1	4.5	0.4
1946	65.9	70.2	4.3	22.7	26.1	3.4	12.2	14.5	2.3	3.9	4.5	0.6
1970	67.4	74.2	6.8	22.6	27.8	5.2	12.0	15.7	3.7	4.1	5.1	1.0
1994	74.9	80.8	5.9	27.9	32.6	4.7	15.6	19.4	3.8	4.9	6.3	1.4
2011	80.0	84.3	4.3	32.1	35.7	3.6	19.2	22.1	2.9	6.0	7.1	1.1

Source: Human Mortality Database (HMD 2015) Life tables, single years of age.

Causes of death

Long-term changes in major causes of death

Australia experienced substantial changes in cause-specific mortality over the period 1907 to 2012 (AIHW 2006). Figure 4 shows that mortality from infectious diseases decreased substantially during the first half of the twentieth century: in 1907, infectious diseases accounted for 16 per cent of the total standardised mortality rate for males and 23 per cent for females, but by 1946 accounted for less than 6 per cent for both sexes, and decreased to insignificant levels by 1960. The initial decline of infectious diseases as a cause of death is characteristic of the Age of Receding Pandemics, while their ongoing decline and virtual disappearance is a characteristic of the Age of Degenerative and Man-Made Diseases. The decreasing prevalence of tuberculosis contributed significantly to the decline of infectious diseases as a cause of death in Australia.

The most recent epidemic significantly affecting Australia was Spanish Influenza, a respiratory disease. Though it began in Spain in 1918, this epidemic did not reach Australia until 1919. The occurrence of this epidemic would indicate that Australia experienced the Age of Receding Pandemics until at least 1920. Apart from this epidemic, the mortality rate from respiratory diseases generally decreased over the whole period, and in relative terms it declined from 14 per cent of the total standardised mortality rate in 1921 to 10 per cent or less since 1946. Respiratory diseases contributed substantially to excess male over female mortality, especially in the second half of the 20th century, partly as a result of smoking-related diseases such as COPD, which increased in relative terms.

The pattern of change in circulatory disease mortality involved a substantial increase, beginning in about 1920, before decline commenced, as seen in Figure 4. In 1907, circulatory diseases accounted for 19 per cent of male and 20 per cent of female mortality. For males, mortality from circulatory diseases increased to the late 1960s, reaching 54 per cent of the total standardized mortality rate in 1970. The increase for females was much less pronounced, and rates stagnated in the 1950s and 1960s, but circulatory disease still accounted for 58 per cent of the total standardized mortality rate in 1970. This pattern would suggest that the transition to the Age of Degenerative and Man-Made Diseases began in 1920 or soon thereafter. For both sexes, circulatory disease mortality declined rapidly from about 1970, marking the beginning of the transition to the fourth Age of Delayed Degenerative Diseases. Circulatory diseases remain both a leading cause of death and a leading cause of excess male mortality.

The increase in circulatory disease mortality has been attributed to high blood pressure, smoking, elevated blood cholesterol, and dietary factors (particularly the consumption of saturated fat and salt; AIHW 2000). Other risk factors include socio-economic status, obesity and physical inactivity, and the harmful use of alcohol (AIHW 2014). The decrease in circulatory diseases from 1970 has been substantially attributed to medical advances and health service improvement, as there has been little change

in physical activity levels and a significant increase in the prevalence of overweight (AIHW 2000, 2014). Thus, the Australian experience of circulatory disease is consistent with Olshansky and Ault (1986) in that the main agent for delayed non-communicable morbidity and mortality has been advances in medical technology (Weisfeldt and Zieman 2007), health care programs for the older population, and reduction of risk factors in communities. The Australian experience also supports the hubristic hypothesis of Rogers and Hackenberg (1987) in that lifestyle factors serve to limit and delimit mortality decline.

Deaths from neoplasms accounted for 8 per cent of male and 9 per cent of female mortality in 1907.⁶ Though rates remained fairly constant during the first half of the century, declines in overall mortality were such that by the 1950s, neoplasms ranked second among leading causes of death. Mortality from neoplasms subsequently increased, especially among males; this has been attributed to changes in smoking behaviour, diet, and environmental factors (AIHW 2000). Despite decreases from about 1990, neoplasms have ranked first among the leading causes of death since 2005. Deaths from neoplasms currently account for around one-third of total age-standardized mortality rates. The causes of cancer are not yet fully understood, but it has been estimated that in high-income countries, smoking, alcohol use, and overweight and obesity were the most important causes at the turn of the century (Danaei et al. 2005). In terms of the Epidemiologic Transition, the Australian experience of neoplasms since 1990 is characteristic of the Age of Delayed Degenerative Diseases.

Mortality rates from external causes were fairly constant over much of the century, although changes occurred for specific external causes such as motor vehicle accidents and suicides. Rates were consistently higher for males than for females, while for both sexes decreases occurred in recent decades. The relative contribution of external causes of death to overall mortality increased slightly, especially for males. Deaths due to external causes are discussed in greater detail below.

The contribution of ‘all other’ causes of death to overall mortality declined substantially over time, particularly before 1950, due largely to improvements in the classification of specific conditions and an associated reduction in the ‘ill-defined’ causes (Lancaster 1990). It is clearly the case that if these deaths had been otherwise classified, the early pattern of mortality decline by cause of death could have looked somewhat different. This observation extends to the causes of excess male over female mortality, most of which is attributed to all other causes in the early part of the period (Figure 4).

These trends in 20th-century Australian mortality rates by major cause of death are broadly similar to those in other Western countries (Meslé and Vallin 2011; Zhao et al. 2014; Bourbeau and Ouellette 2016; this volume). The decline in infectious disease mortality and the timing of Spanish Influenza were contemporaneous across countries. As noted, the transition to the Age of Degenerative and Man-Made Diseases began in Australia in 1920 at the earliest. The increase in circulatory disease mortality also began in about 1920, though it is unknown to what degree improved classification influenced the early trend. It is clear that mortality levels stagnated during the 1960s, due to the combined effect of declining infectious disease mortality and, until around 1970, increasing circulatory disease mortality (Taylor et al. 1998). This pattern of counterbalancing causes of death is characteristic of the third Age of Degenerative and Man-Made Diseases, as described by Omran (1971). The ensuing rapid decline in circulatory disease mortality, combined with declines in respiratory disease mortality from the 1970s, and with declines in deaths from neoplasms from the 1990s, marked the transition to the Age of Delayed Degenerative Diseases, when life expectancy resumed its increase. This decline in circulatory disease mortality was more pronounced in Australia than in other Western countries, including Canada and the US (Barbieri and Ouellette 2012) and England and Wales (Griffiths and Brock 2003).

6. These are probably underestimates as in 1907 the diagnosis and certifying of cause of death for cancer was problematic.

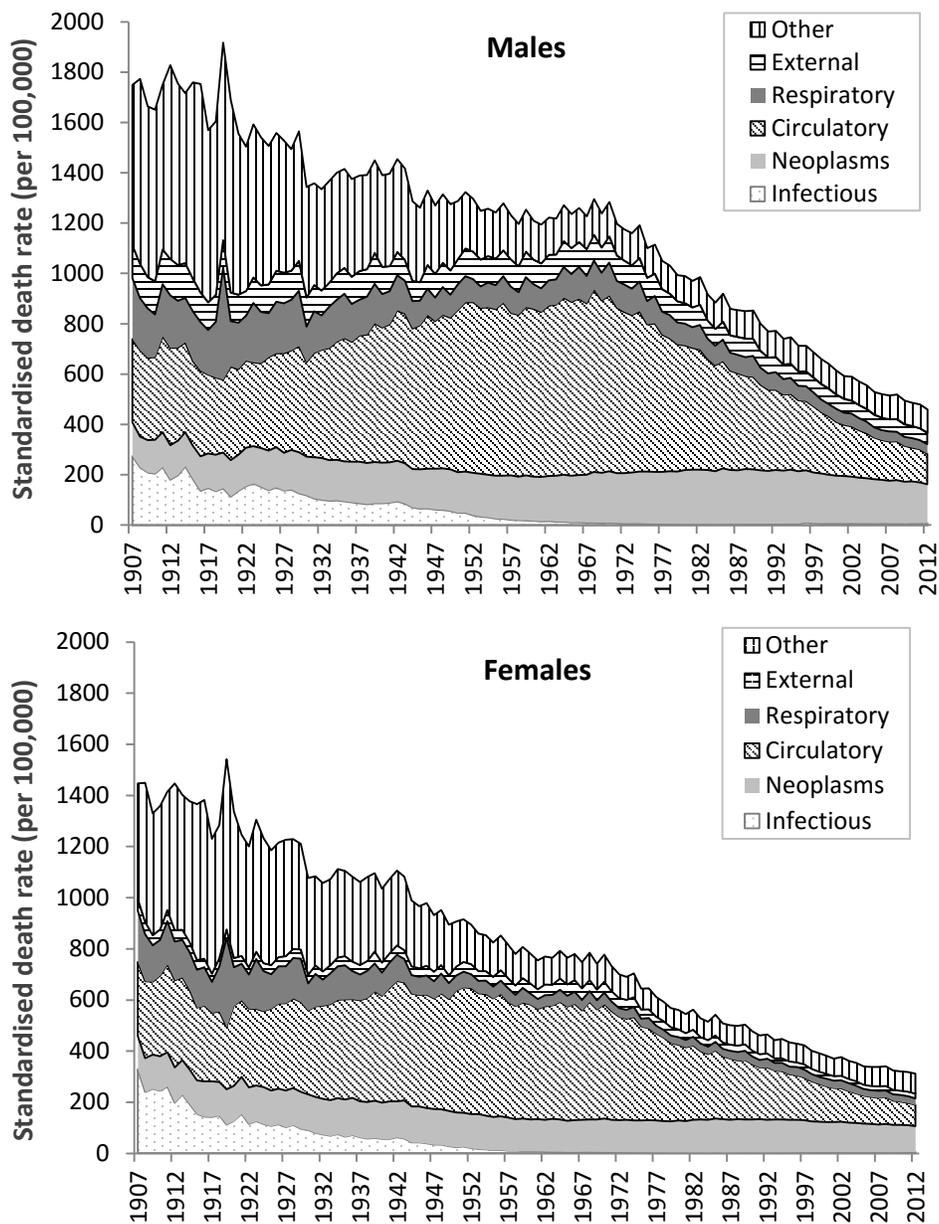


Figure 4. Standardized mortality rate (per 100,000) for males and females by major cause of death, Australia, 1907 to 2012.

Source: Authors' calculations based on data from AIHW (2015a).

Contribution of changes in age-cause-specific mortality to changes in life expectancy at birth

To further identify the roles of the six major causes of death by age group during the Epidemiologic Transition, decomposition analyses of changes in life expectancy were conducted for the four selected periods (see the section on 'Data and methods'). Life expectancies for relevant years are shown in Table 1. The decompositions by age and cause of death are shown in Figure 5 for males and Figure 6 for females.

From 1922 to 1946, life expectancy increased by 5.0 years for both sexes. Mortality reductions at ages 0 to 4 were responsible for a life expectancy gain of 2.6 years for males and 1.9 years for females, half of which was attributable to infectious diseases. Reductions in infectious and respiratory disease mortality at ages 5 and older also contributed to the life expectancy gains, but at older adult ages were counterbalanced by increases in circulatory disease mortality (see Figures 5 and 6). Thus, ages 65 and older for males and 85 and older for females contributed negatively to the life expectancy gains.

In terms of the contribution of deaths from different causes to the increases in life expectancy, infectious diseases contributed 2.3 years for males and 2.1 years for females, while respiratory diseases contributed 1.1 years for males and 0.9 years for females. In contrast, rising circulatory disease mortality at ages 45 and older produced negative contributions of 1.5 years for males and 1.0 year for females. It is noted that reduced deaths from all other causes (which were partly due to better classification) made substantial positive contributions in this period, particularly for females; this may account for some of the negative contribution of circulatory disease mortality (Lancaster 1990). In terms of the Epidemiologic Transition, this analysis would indicate that 1922–46 was the period of transition from the Age of Receding Pandemics to the Age of Degenerative and Man-Made Diseases. The substantial increase in life expectancy over this period would, however, indicate that the transition occurred rather late in the period.

The changes in life expectancy from 1946 to 1970 are smaller than those occurring in the previous period, and differ substantially by sex: life expectancy for females increased by 4.0 years, while that for males increased by only 1.6 years (Table 1). Again, the mortality decline at ages 0 to 4 contributed significantly to the overall increase in life expectancy—by 1.2 years for both sexes. The large sex difference in the net gain in life expectancy is accounted for by a much larger decrease in female than male mortality at ages 15 and older. For males, an increase in circulatory disease mortality (occurring at ages 35 and older) resulted in an overall 0.6-year loss of life expectancy, while an increase in deaths from neoplasms accounted for a 0.4-year loss. In contrast, for females, reduced deaths from circulatory diseases (except at ages 75 and older) and neoplasms contributed to overall gains of 0.3 and 0.2 years, respectively. Further, the life expectancy gains due to respiratory disease mortality were larger for females than for males. Deaths from external causes contributed negatively to the change in life expectancy for both sexes, particularly among males and those aged 15 to 29, attributable to the emergence of the accident hump. Again, reduced deaths from all other causes contributed substantially and positively to life expectancy. During this period, Australia exhibited characteristics of the Age of Degenerative and Man-Made Diseases, especially among males for whom circulatory, respiratory, and external causes—associated with lifestyle and man-made factors such as smoking and motor vehicles—were key. While female mortality was less affected by such factors and continued to decline, albeit at a slower pace than previously, male mortality stabilized and at some ages increased. For both sexes, as shown in Figure 4, non-communicable diseases became dominant, with circulatory diseases becoming the primary killer.

The period 1970–94 was one of renewed acceleration in mortality decline, with life expectancy increasing by 7.5 years for males and 6.6 years for females. The decline in mortality at ages 0 to 4 contributed 1.3 years for males and 1.0 year for females, but these were no longer dominant. Declines at ages 45 to 84 made much larger positive contributions to life expectancy, primarily due to reduced circulatory disease mortality, but also due to reductions in respiratory disease mortality. Mortality from external causes also declined, notably among males aged 15 to 24, contributing to higher life expectancy. In contrast, mortality from neoplasms at ages 65 and older made a small negative contribution to life expectancy, 0.1 years for both sexes. This pattern of change is consistent with the

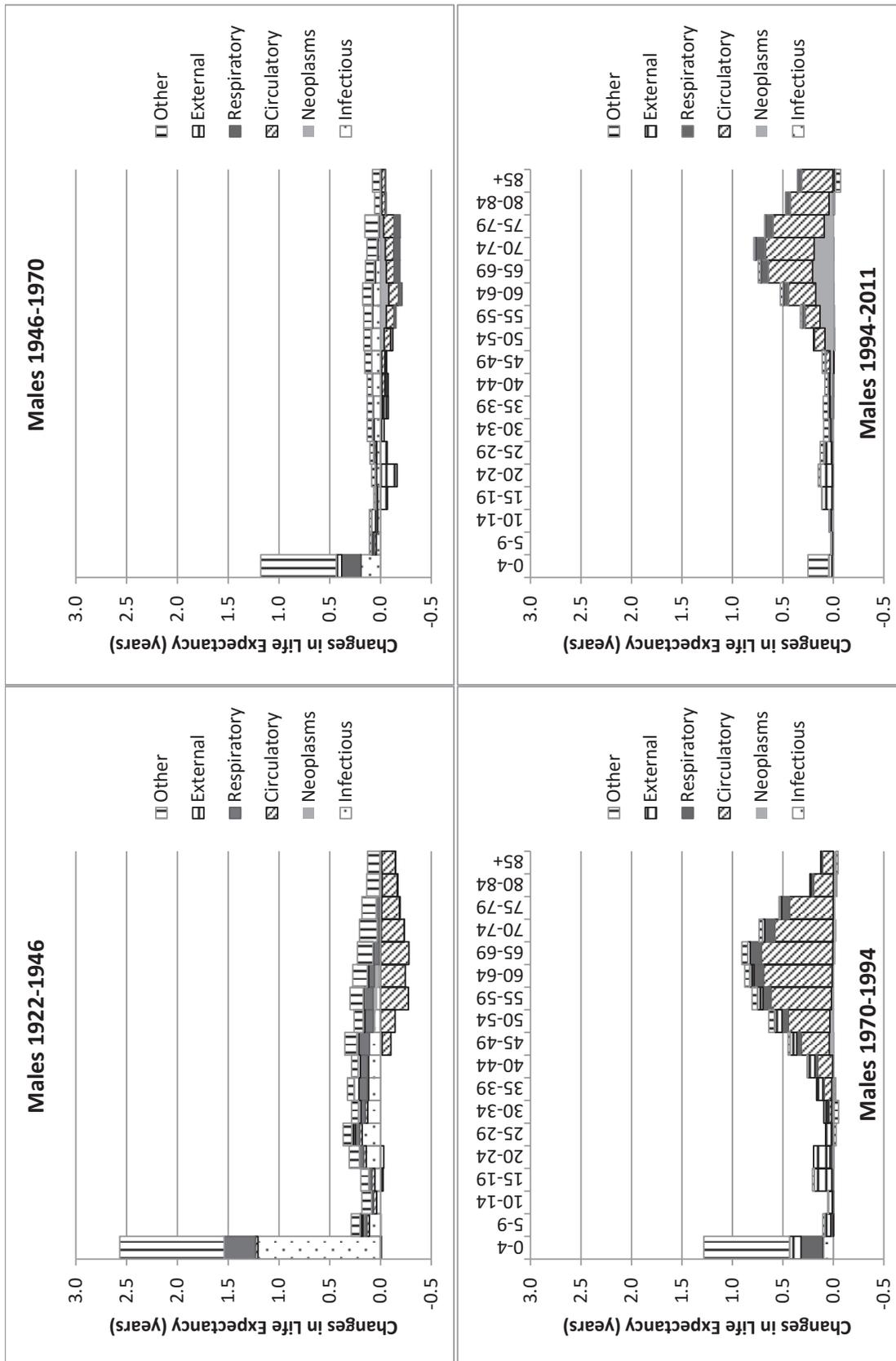


Figure 5. Decomposition of change in life expectancy by period, age and cause of death, males, Australia, 1922 to 2011

Source: Authors' calculations based on data from AIHW (2015a).

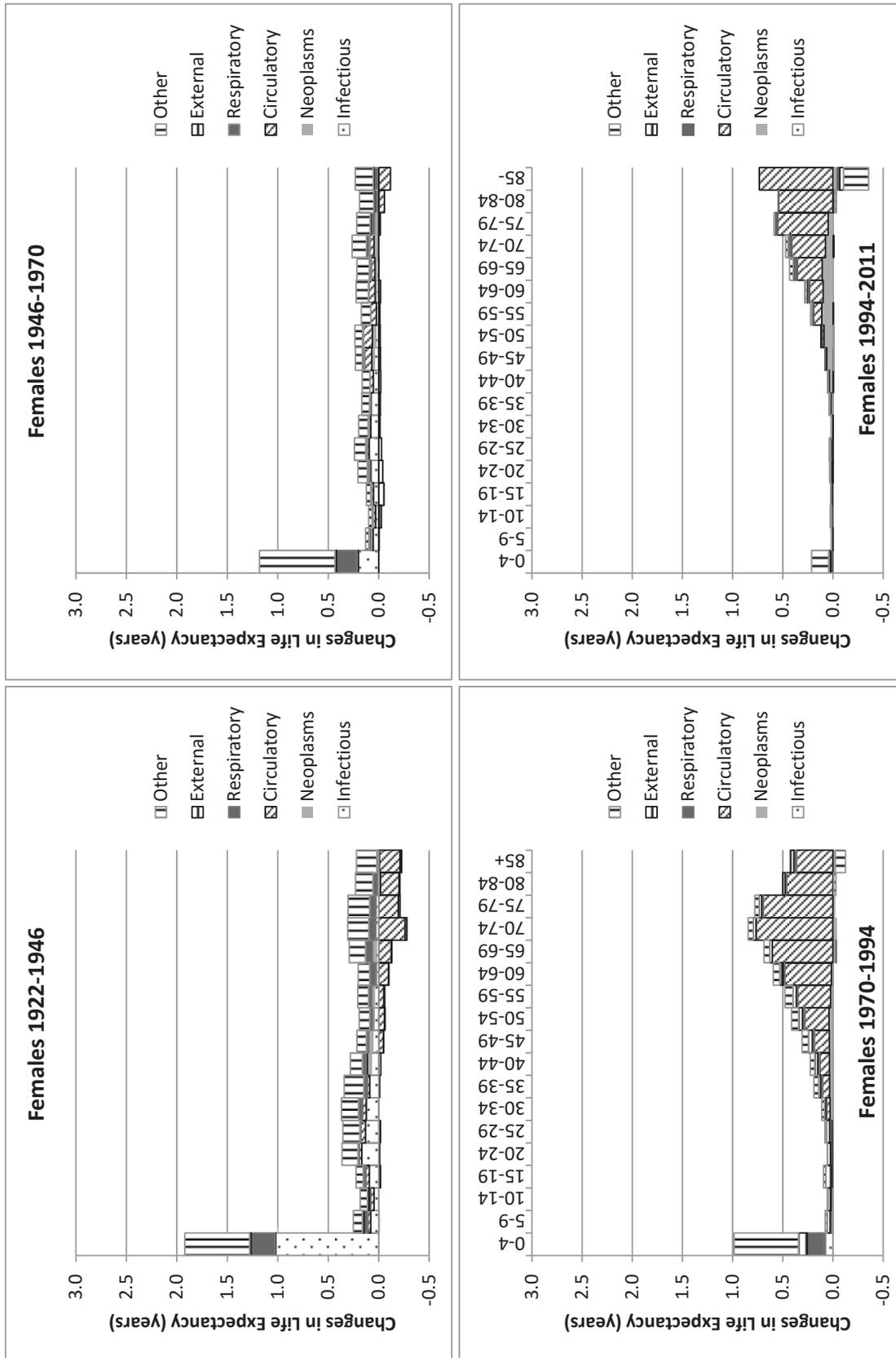


Figure 6. Decomposition of change in life expectancy by period, age and cause of death, females, Australia, 1922 to 2011.

Source: Authors' calculations based on data from AIHW (2015a).

Age of Delayed Degenerative Diseases, which is characterised by a significant decline in circulatory disease mortality (Olshansky and Ault 1986). In Australia, fully 4.3 years of the net gain in both male and female life expectancy was due to reduced deaths from circulatory diseases.

During the final period considered, 1994–2011, life expectancy increased by 5.1 years for males and 3.5 years for females. These increases were mainly attributable to declines in mortality at advanced ages. Indeed, life expectancy at age 65 increased by 3.0 years for males and 2.4 years for females. In terms of causes of death, circulatory diseases were still the primary contributor, accounting for 2.7 years for males and 2.6 years for females. In contrast to the previous period, reduced mortality from neoplasms—mainly at ages 50 to 79 years for males and 45 to 74 for females—contributed positively to changes in life expectancy, though rates continued to increase at older ages. This pattern is consistent with the notion of delayed non-communicable diseases. For males, reduced mortality from respiratory diseases, mainly at older ages and from external causes at young ages, each produced a gain of 0.4 years in life expectancy, while for females, smaller gains of 0.2 and less than 0.1 years, respectively, were produced. The emergence of a loss in life expectancy due to ‘all other’ causes at age 85 and older, particularly for females, is due to the increased incidence and better reporting of neurological diseases such as dementia.⁷ Generally, over this period, the patterns of change in the major causes of death are characteristic of the Age of Delayed Degenerative Diseases, in that the rapid decline in death rates is concentrated mostly at advanced ages (Olshansky and Ault 1986).

Comparison of the four decompositions throws further light on the evolution of the Epidemiologic Transition in Australia. Being chosen on the basis of internal consistency of age-by-cause patterns, these time periods help to identify the processes taking place in the transition. Comparing 1922–46 and 1946–70, it is clear that circulatory disease mortality was more important in the earlier period in limiting life expectancy gains. During this period, gains due to infectious disease mortality, which was concentrated at ages 0 to 4, were counterbalanced by losses due to circulatory disease mortality at older ages. Thus the transition moved into the Age of Degenerative and Man-Made Diseases during this period. For male mortality, the decomposition for 1946–70 shows not only a continuation of this pattern but also life expectancy losses due to increased mortality from external cause (see below subsection on the adolescent and young adult mortality hump), and from neoplasms and respiratory diseases, both of which are associated with smoking. The near-absence of these losses in female life expectancy during this period is largely attributable to the later and more-restricted uptake of smoking among females (AIHW 2000).

It is clear from comparison of the decompositions for 1946–70 and 1970–94 that 1970 marked a turning-point in cause-of-death patterns in Australia. Pre-1970 life expectancy losses, due principally to circulatory disease mortality, became large positive post-1970 gains. Thus, 1970 can be regarded as a watershed between the Age of Degenerative and Man-Made Diseases and the Age of Delayed Degenerative Diseases. Comparison of the decompositions for 1970–94 and 1994–2011 demonstrates how gains in life expectancy due to circulatory disease mortality have moved to older ages, which is a characteristic of the Age of Delayed Degenerative Diseases. Comparison of males with females in 1970–94 and 1994–2011 shows that female gains occur at older ages than male; given higher female life expectancy, this is consistent with the Age of Delayed Degenerative Diseases, in which deaths are progressively delayed. In general terms, it can be said that the Epidemiologic Transition is more advanced for females than for males.

7. Alzheimer’s disease was introduced into the ICD in 1979.

Age patterns of change

The broad theoretical approach of the Epidemiologic Transition is complemented in this section using two approaches. First, age patterns of mortality change are examined over time. Second, we focus on three ages that are important in determining the shape of the mortality schedule and the evolution of life expectancy: these are *infancy*, *adolescence and young adulthood*, and *older age* (or *senescence*).

The age patterns of mortality change themselves change over time. This is seen in Figure 7 which shows the average annual percentage decline in age-specific mortality rates for the four selected time periods. These curves are similar to the overall patterns shown in Figures 5 and 6, but enable direct comparison by age and period in the speed of decline.

In 1922–46, mortality decline was most rapid at childhood (but not infant) ages and at about age 30. In contrast, there was very little change at older adult ages, where in fact some increases occurred, especially for males. This echoes the counterbalancing trends in infectious and circulatory diseases, and provides supporting evidence that the transition to the Age of Degenerative and Man-Made Diseases occurred during this period.

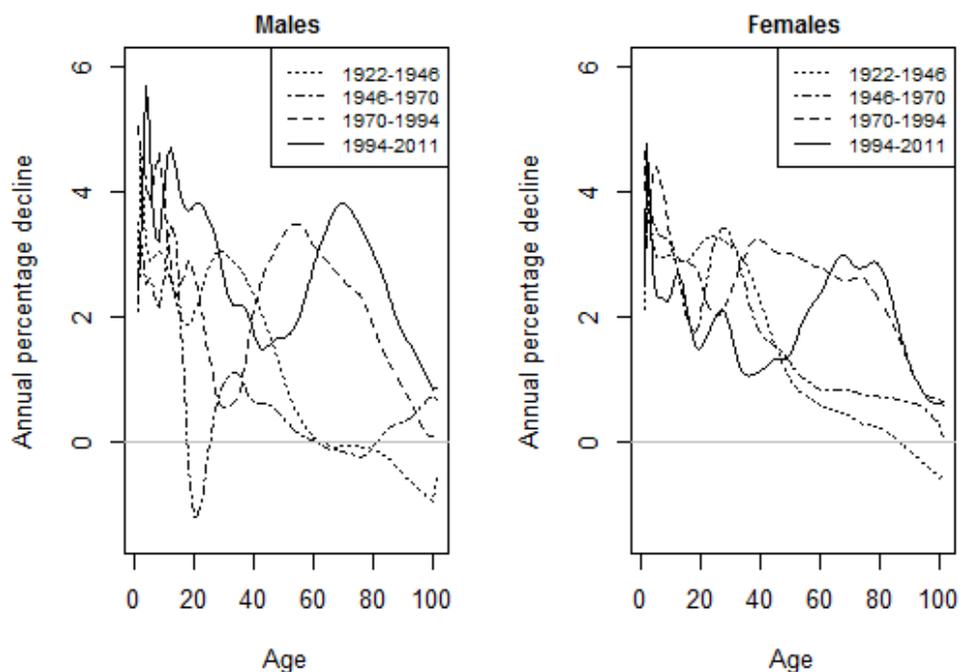


Figure 7. Average annual percentage decline in age-specific mortality rates by sex by period, Australia, 1922 to 2011.

Source: Authors' calculations based on data from HMD (2015).

The second period, 1946–70, is notable for the substantial sex difference in the patterns of mortality decline at adult ages. Though for both sexes the rate of decline was relatively low at age 20, for males the rate was negative around this age, indicating increased mortality that, in combination with modest increases at older ages, resulted in the mortality stagnation of the last decade or so of this period. At older ages, negative rates of decline for males reflect the increasing mortality from circulatory and respiratory diseases and neoplasms that characterizes the Age of Degenerative and Man-Made Diseases. Infant and early childhood mortality declined relatively slowly during this period, again consistent with the Age of Degenerative and Man-Made Diseases.

In 1970–94, infant and childhood mortality resumed a more rapid decline. At post-childhood ages, the most rapid declines occurred at ages 40 to 80 among females and at 50 to 60 among males. This was the period when circulatory disease mortality declined rapidly and life expectancy increases resumed, indicative of transition to the Age of Delayed Degenerative Diseases, as described by Olshansky and Ault (1986). The fact that little change occurred at ages 20–30, especially among males, supports the hubristic hypothesis of Rogers and Hackenberg (1987).

Finally, in 1994–2011, the most rapid declines in adult mortality were at ages 50 to 90 years. The rapid shift of the modal age of mortality decline is a characteristic of the Age of Delayed Degenerative Diseases. While high rates of decline still occurred in infancy and early childhood, they apply to very low rates, reducing their overall impact.

Infant mortality

In common with high-income countries generally, over the last century rapidly declining infant mortality accounted for a very significant component of the increase in Australian life expectancy at birth. This was particularly so during the earlier part of this period, when Australia experienced a rapid decline in infectious disease mortality. de Looper (2015, Chapter 7) noted that deaths in infancy occurred at a rate of about 1 in 10 in 1900, and that they declined sharply after 1903, thanks largely to social and environmental factors.

Figure 8 shows the infant mortality rate, defined as annual deaths of infants under one year of age per 1,000 live births in the same year, over the period 1921–2011. While the decline has been fairly continuous, more rapid declines occurred in the early 1940s—following the introduction of sulfa drugs in the 1930s to combat infection, notably in childbirth—and again in the mid-to-late 1970s—coincident with both the introduction of the Medibank (now Medicare) universal health insurance scheme (Taylor and Lewis 1998) and the operation of new neonatal intensive care units (Taylor and Lewis 1998). By the mid-1970s, infant mortality rates had declined to one-quarter of 1921 levels, and by 2011 to only 6 per cent of 1921 levels. Infant mortality rates in Australia are now such that over 99.5 per cent of infants survive to their first birthday. Although further dramatic reductions are therefore not possible, scope for significant gains is indicated by even lower rates in numerous countries—including Singapore and Hong Kong, where current infant mortality rates are less than half of those in Australia (UNPD 2015). It is noted, however, that many among these countries do not have remote or inaccessible areas such as those in Australia, and are therefore in a better position to deliver universal health services and risk factor reduction programs.

Adolescent and young adult mortality hump

A notable feature of changing Australian mortality over the last century was the appearance and subsequent diminution of the so-called *accident hump* at late teenage and early adult ages. Figure 9 illustrates this phenomenon, showing the emergence of a pronounced hump for males in the 1960s and 1970s⁸ and its transformation into a plateau or ‘shoulder’ by the early 1990s, and the later emergence of a much smaller accident hump for females (Pollard 1996). Since the early 1990s, mortality rates have declined further at all ages, and the plateau shape has been retained. It is questionable whether this feature will persist or eventually disappear. It is noted that historic mortality patterns do not display this feature, and that for Swedish females it has all but disappeared (Booth and Tickle 2008).

8. The main increase (for males) or stagnation (for females) in the probability of death occurred in the 1960s.

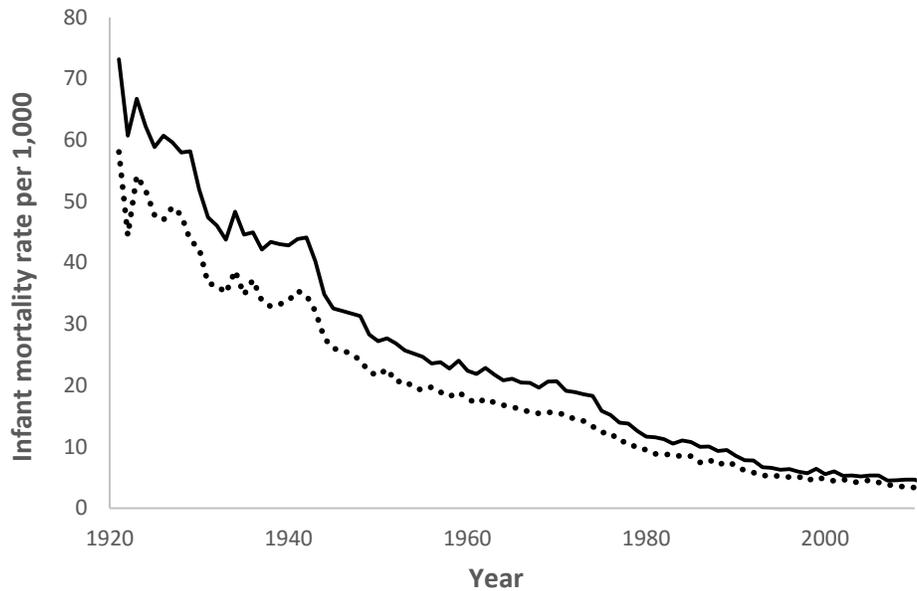


Figure 8. Infant mortality rate per 1,000 live births for males (solid line) and females (dotted line), Australia, 1921 to 2011.

Source: Human Mortality Database (HMD 2015), Births and Deaths, single year.

Pollard (1996) found that the factors behind the change in the Australian male accident hump included a decline in motor vehicle accident mortality in late teenage and early twenties, due to public health measures—including seat belt and random breath testing legislation—as well as improvements in road systems and in vehicle design. Thus, the accident hump is a feature of the Age of Degenerative and Man-Made Diseases, and its diminution may be viewed as characteristic of the Age of Delayed Degenerative Diseases.

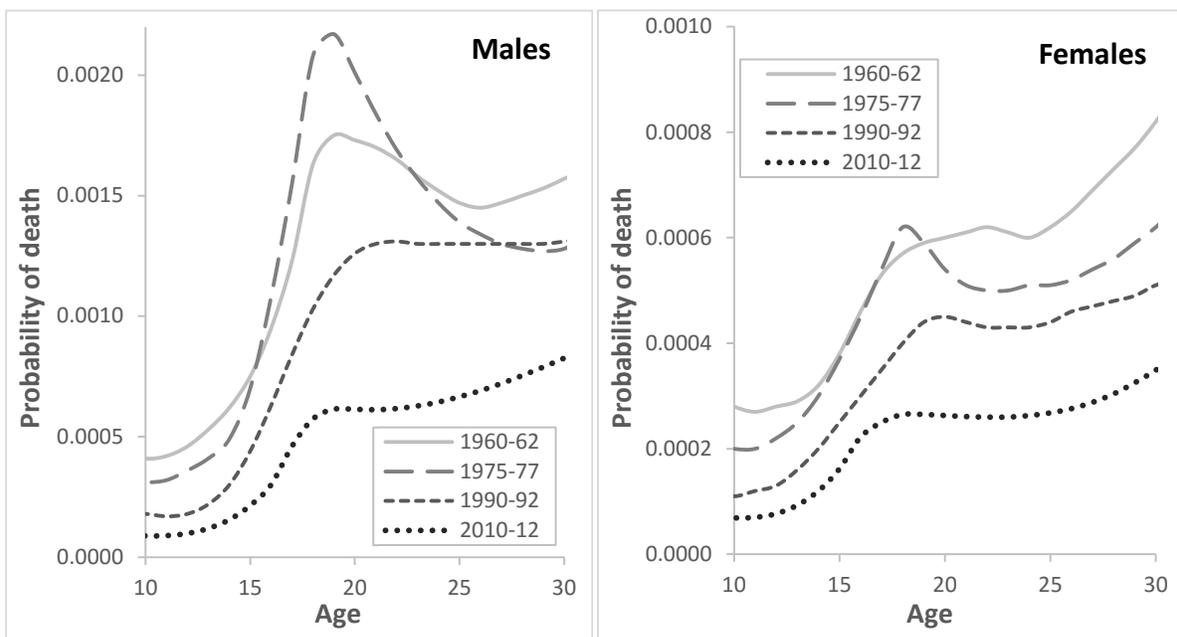


Figure 9. Probability of death at ages 10 to 30 for selected periods for males and females, Australia.
Source: Australian Government Actuary Australian Life Tables (various dates).

Mortality at advanced ages

One of the remarkable features of the Australian mortality transition is the speed at which mortality at advanced ages has declined in recent decades. It has already been demonstrated in Figure 7 that the most rapid rates of decline moved to older ages over time, and that rates of decline at older ages increased substantially. This transition was underway in 1970–94, and by 1994–2011, mortality at ages 50 to 90 was declining rapidly. Such a pattern of change is characteristic of the Age of Delayed Degenerative Diseases (Olshansky and Ault 1986). Figure 7 also shows that in the two most recent periods, the speed of decline at advanced ages (80 years and older) is very similar for males and females, which is also characteristic of this Age. Additionally, Figures 5 and 6 have demonstrated that deaths due to non-communicable diseases—circulatory diseases and neoplasms—have shifted to progressively older ages, which is also characteristic. This is further evidence that the Australian mortality experience from about 1970 is consistent with the Age of Delayed Degenerative Diseases.

Differentials in mortality

Though the Epidemiologic Transition is concerned with broad developments, usually addressed at the national level, the Age of Degenerative and Man-Made Diseases and the Age of Delayed Degenerative Diseases may be experienced at different speeds or times by different subpopulations within a nation. As noted by Caselli, Meslé, and Vallin (2002), the later stages of the transition depend progressively on personal responsibility for one's own health. Many public health messages, initiatives, and services are effective only to the degree of personal compliance. Education, income, occupation, residential environment, and cultural factors all play important roles in health and health behaviour, influencing diet, physical activity, smoking, alcohol consumption, and risky behaviour (AIHW 2014). Government also plays a role through legislation, taxation, and the creation of equitable health-promoting environments. In Australia, substantial differentials in mortality can be found by geographic area and by socio-economic characterization of area, but the largest differential is by indigeneity. This section examines three sources of heterogeneity in mortality based on life expectancy at birth: states and territories, indigeneity, and socio-economic factors.

Mortality for states and territories

Australia consists of six states—New South Wales (NSW), Victoria, Queensland, Western Australia, South Australia, and the island state of Tasmania—and two territories, the Northern Territory and the Australian Capital Territory (ACT).⁹ Life expectancy at birth by state and territory since 1971 is shown in Figure 10.

For males, ACT residents clearly have the highest life expectancy at birth, with a consistent advantage over the next-ranked state or territory, averaging just less than one year since the early 1980s. NSW, Victoria, Queensland, South Australia, and Western Australia are ranked next and have similar levels of male life expectancy, differing by at most one year since the early 1990s. Tasmania currently lags behind the lowest of this group by about one year, and there is then a gap of four years to the Northern Territory. For females, similar patterns apply, but the ACT has a smaller lead, Tasmania has a larger lag, and the Northern Territory is currently three years below Tasmania. These patterns of mortality decline indicate that the Epidemiologic Transition in Australia is led by the ACT, with the Northern Territory being a significant laggard.

9. The ACT is a small territory enclaved within NSW and containing the capital city, Canberra.

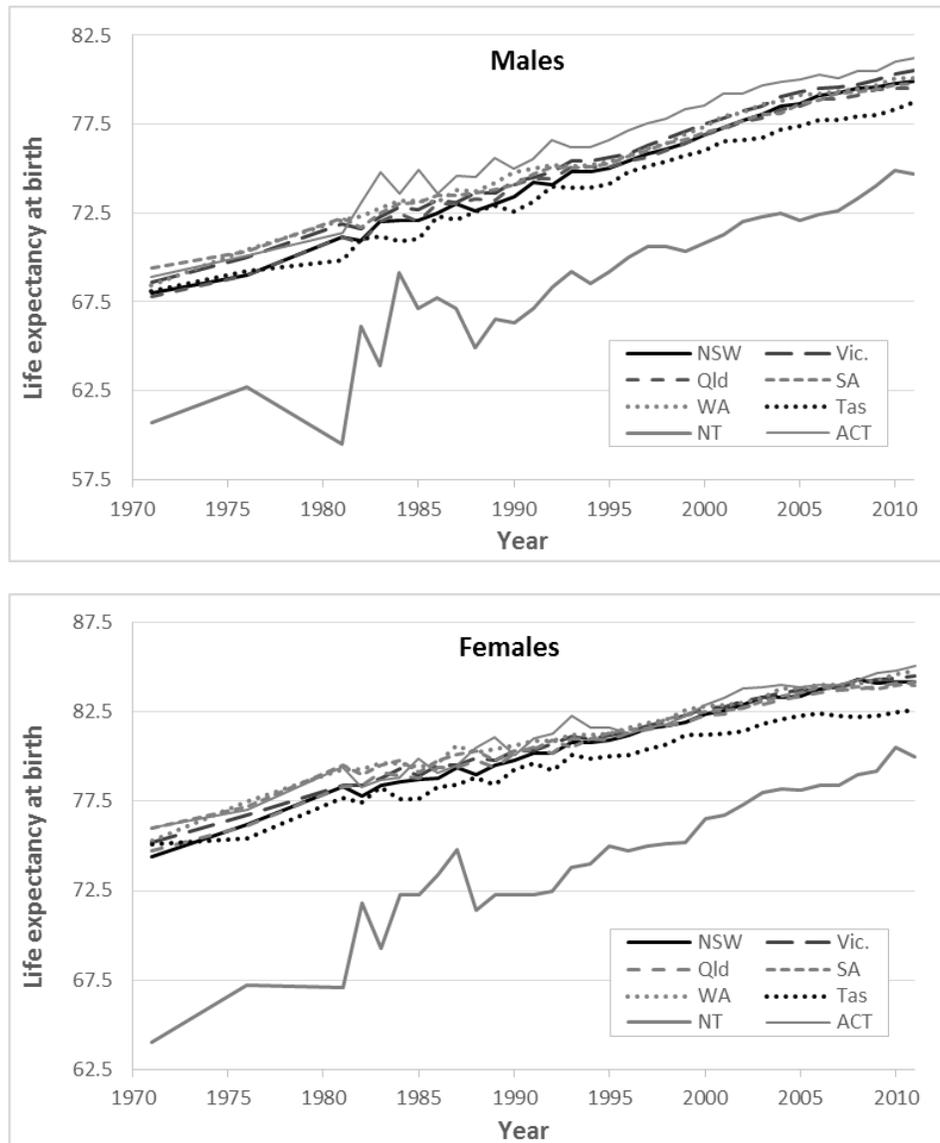


Figure 10. Life expectancy at birth (years) for males and females by state/territory, Australia, 1971 to 2012

Source: Australian Bureau of Statistics (2014b). Figures for 1971 to 1993 are for single years; figures for 1994–2012 are three-year averages.

It is also seen in Figure 10 that the gap in life expectancy between the Northern Territory and other states and territories has narrowed considerably for females. Compared with the Australian average, Northern Territory life expectancy for females was about 10 years lower in the 1970s and is currently about four years lower. For males, there is much less narrowing in evidence, and the gap between the Northern Territory and the Australian average has remained at roughly six years since the 1990s. For both sexes, Northern Territory mortality in 2011 was equivalent to that experienced in 1990–95 in other parts of Australia.

A number of factors account for the state and territory differences. Low life expectancy in the Northern Territory reflects at least in part a relatively high proportion of Indigenous (Aboriginal and Torres Strait Islander) peoples, and a significant proportion of the Australian population living in

remote areas. Indigenous mortality is higher than non-Indigenous mortality (see subsection on ‘Indigenous mortality’), while mortality in remote areas generally exceeds that in regional areas, which in turn generally exceeds that in major cities (AIHW 2003). Indigenous mortality is a major contributor to higher mortality in remote areas; and people living in regional and remote areas tend to have lower levels of access to health services (AIHW 2007a). Socio-economic factors are also relevant to state and territory differences: for example, that average weekly adult full-time earnings are highest in the ACT and lowest in Tasmania (ABS 2015) accords with observed mortality differentials.

Indigenous mortality

There is a very large gap between Indigenous and non-Indigenous life expectancy. Indeed, in 2009, the Council of Australian Governments (COAG) adopted, among a number of targets to address Indigenous disadvantage, the intention ‘to close the life expectancy gap within a generation’ (COAG 2009).

Assessment of the gap in life expectancy between Indigenous and non-Indigenous peoples is problematic because of unreliable estimates of Indigenous life expectancy. Both deaths and population data suffer from problems in the reporting of indigeneity: Indigenous deaths may not be identified as Indigenous by the family, health worker, or funeral director, while Indigenous population counts vary according to changes in the propensity to identify as Indigenous (AIHW 2015b). The Australian Bureau of Statistics makes allowances for this, based on an Indigenous deaths and census records linkage study (ABS 2014c); others have linked death records and hospital and other data to obtain different estimates (Neville et al. 2011; AIHW 2015b; Madden et al. 2012). The Australian Institute of Health and Welfare recently estimated the indigeneity gap as 10.6 years for males and 9.5 years for females (AIHW 2015b).

This ten-year difference between Indigenous and non-Indigenous life expectancy places the Indigenous population at mortality levels experienced by the non-Indigenous population some 30 to 40 years ago (see Figure 3, noting that the Indigenous population comprises less than 3 per cent of the total population of Australia). In other words, Indigenous life expectancy is equivalent to that experienced by the non-Indigenous population in the 1970s. Two-thirds of the gap in life expectancy is estimated to be due to deaths from circulatory diseases, endocrine, metabolic and nutritional disorders (including diabetes), cancer, and respiratory diseases (AIHW 2015b). This suggests that the Indigenous population may still be experiencing the third stage of the Epidemiologic Transition—the Age of Degenerative and Man-Made Diseases—and may be best described as undergoing Delayed Epidemiologic Transition (Omran 1971).

Socio-economic and geographic differentials

The collection of data on the characteristics of deceased persons at time of registration of death is not comprehensive in Australia, limiting the availability of data on socio-economic and other differentials in mortality. The method adopted by official agencies for measuring socio-economic differentials relies on area-based socio-economic indices (SEIFA) constructed from census data (ABS 2013). These indices are used to classify geographic areas (generally postcodes) into the quintiles of the socio-economic distribution. As both deaths and population estimates are available by geographic area, the SEIFA scores enable the estimation of mortality differentials. This method involves inaccuracies, in that the SEIFA score is an average for the area and cannot reflect the range of personal socio-economic characteristics in the area.

Table 2. Life expectancy by sex by socio-economic quintile and remoteness, Australia, 2003

	Males	Females	Persons	Sex difference
Socio-economic quintile				
High	80.9	84.5	82.7	3.6
Moderately high	79.0	83.5	81.2	4.5
Average	77.7	82.7	80.2	5.0
Moderately low	77.4	82.8	80.0	5.4
Low	76.9	82.3	79.6	5.4
Remoteness				
Major cities	78.8	83.5	81.2	4.7
Regional	77.5	82.7	80.0	5.2
Remote	75.4	81.5	78.1	6.1
Australia	78.3	83.2	80.7	4.9

Source: AIHW (2007b) Chapter 5.

Table 2 shows life expectancy by socio-economic quintile for 2003¹⁰ (AIHW 2007b). The gradient in mortality by socio-economic quintile demonstrates that the Epidemiologic Transition is led by higher socio-economic areas. The difference in life expectancy between the low and high socio-economic quintiles is greater for males (4.0 years) than for females (2.2 years). Further, the sex difference in life expectancy at the low socio-economic level (5.4 years) is greater than at the high socio-economic level (3.6 years). The Epidemiologic Transition is least advanced among low socio-economic males.

Table 2 also shows life expectancy differences by geographic area: major cities, regional areas, and remote areas. The major city–remote area differences are 3.4 years for males and 2.0 years for females; these are smaller than the socio-economic differences. Sex differences in remote and regional areas are larger than in major cities. These differences demonstrate that the Epidemiologic Transition is led by major cities, and that males in remote areas lag considerably.

Discussion

This analysis has found that the Australian mortality experience over the last hundred years is broadly consistent with the second and third Ages of the Classical or Western model of the Epidemiologic Transition defined by Omran (1971, 1983) and with the fourth Age of Delayed Degenerative Diseases, as described by Olshansky and Ault (1986) and Rogers and Hackenberg (1987). However, the relatively high life expectancy in the early decades of the twentieth century is somewhat anomalous in the Epidemiologic Transition framework.

As noted, the first Age of the Epidemiologic Transition was essentially absent in Australia. From first settlement in 1788, health and mortality conditions were best described as characteristic of the second Age of Receding Pandemics. After a slow decline between the mid-1850s and mid-1880s, mortality declined more rapidly. Based on cause of death analysis, de Looer (2015) concluded that there was no evidence of epidemiologic transition before 1906.

By the early 1920s, Australia enjoyed a life expectancy close to the highest in the world. In 1921–24, life expectancy exceeded the threshold for transition to the Age of Degenerative and Man-Made Diseases by as much as ten years, suggesting a more advanced transition than in other Western populations, where the Age of Receding Pandemics is generally viewed as continuing until mid-century

10. These area-based analyses of mortality are not routinely available.

(e.g., Robine 2001; Lussier et al. 2008). Further, what was to be Australia's last epidemic—Spanish Influenza in 1919—could be seen as having been precipitated by the particular circumstances of the First World War and its aftermath, and the widespread movement of people in 1918–19 (Oxford et al. 2005), such that the Age of Receding Pandemics was exceptionally prolonged.¹¹

The analyses in this paper provide partial support for the proposition of a relatively advanced Epidemiologic Transition in Australia, but not to the extent implied by life expectancy levels. During 1922–46, significant decreases in infectious and respiratory disease mortality occurred, as well as substantial increases in circulatory disease mortality. While Figure 4 shows increasing circulatory disease mortality from about 1920, it is possible that this trend is influenced by the improved classification of cause of death (Lancaster 1990), such that the increase is exaggerated. However, the disappearance of ill-defined causes (Lancaster 1990) would imply that by 1946, circulatory disease mortality was much more reliably reported, such that some increase can be confirmed. It can therefore be concluded that Australian mortality was transitioning to the third Age of Degenerative and Man-Made Diseases during this period, but it is not possible to be more precise about timing. Thus, the Australian Epidemiologic Transition may indeed have been slightly advanced in comparison with other industrialized countries at this time.

Whenever its beginnings, the Age of Degenerative and Man-Made Diseases can be said to have endured until about 1970, in keeping with other industrialized countries. From about 1950, increasing circulatory disease mortality began to slow among males, and rates declined among females, a result of prior changes in risk factor behaviours. Over the period 1946–70, circulatory disease mortality made a small positive contribution to the change in female life expectancy, but a sizeable negative contribution in male life expectancy. Thus, for males especially, overall mortality stagnated in the 1960s. This pattern of slow decline and the predominance of circulatory disease mortality is characteristic of the Age of Degenerative and Man-Made Diseases. Life expectancy in 1970 was about 71 years, close to the limit of 70–75 years that was accepted wisdom at the time (Olshansky and Ault 1986).

The beginnings of the Age of Delayed Degenerative Diseases seem clear. Life expectancy increased with renewed vigour from about 1970, driven largely by rapid declines in circulatory disease mortality, in line with the 'cardiovascular revolution' (Meslé and Vallin 2011). Ongoing declines occurred in mortality from circulatory and respiratory diseases from the 1970s and in neoplasms from the 1980s, such that mortality decline is greatest at increasingly older ages, roughly equally for the sexes. This fourth stage continues in the 21st century.

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11. While Spanish Influenza affected many populations, their lower life expectancies were more in keeping with the Age of Receding Pandemics.

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