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Forecasting disability: application of a frailty model

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Random changes in individual frailty occur due to the stochastic processes of physical deterioration or environmental influences. This paper implements a stochastic ageing model using maximum likelihood methods and calibrates the model to more than 30 years of historical Australian mortality data in order to examine cohort and gender differences in health-state distributions among older adults. We find that frailty levels have declined over time for both male and female cohorts. Nonetheless, patterns of frailty are different between genders. Older females experience a faster pace of health deterioration than their male counterparts causing them to move quicker into worse states of health. Health states are also more heterogeneous among women than men. Population-level estimates suggest that the number of elderly Australians requiring aged care services will exceed that projected under governmental assumptions by 2050.

Keywords: population ageing; morbidity; disability risk; health-state distributions

1. Introduction

People aged 65 or over constitute one of the fastest growing age group within the population in Australia. Census data from the Australian Bureau of Statistics (ABS) reveal that the Australian population has been steadily ageing since the early 1970s: for example, the proportion of people aged 65 years and over increased from just 8.3% in 1971 to almost 14.7% in 2014 (ABS 2014). Whether increasing longevity is accompanied by higher levels of frailty and morbidity is an issue of concern since these could translate into higher levels of disability and dependency, which has implications for healthcare and long-term care costs.

This paper implements a stochastic ageing model to study cohort and gender differences in health-state distributions among older adults in Australia. In particular, we examine whether recent cohorts of older Australians are on average more or less frail than their predecessors, allowing for observed increases in life expectancies. To map frailty levels to different health states, we use recent data from cross-sectional disability surveys in Australia. Finally, time-series methods are employed to project trends in disability prevalence for older

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individuals up to 2050. Projection estimates are illustrated for 75-year-old individuals, and separately, for those age 85.

Frailty models have been extensively employed to quantify heterogeneity in population mortality based on widely available population-level data (e.g. Vaupel *et al.* 1979; Manton *et al*. 1986; Congdon 1994). The concept of frailty refers to an individual's relative susceptibility to death compared to a standard. Traditional frailty models generally assume that frailty is fixed throughout a person's lifetime and does not vary with age; fixed frailty models do not allow for improving time trends in mortality rates (Su & Sherris 2012). In contrast to fixed frailty models, the stochastic ageing model draws on the concept of acquired susceptibility to death due to physiological changes and environmental influences (Yashin *et al.* 1994; Lin & Liu 2007). By allowing for randomly changing frailty, the stochastic ageing model incorporates the random nature in an individual's biological ageing process. In particular, the ageing process is viewed as an internal stochastic process of deterioration in terms of human body's physiological capacity and the status of an individual's physiological capacity is called 'physiological age'. A higher physiological age implies increased susceptibility to various hazards, including death.

The stochastic ageing model was originally introduced by Le Bras (1976). The Le Bras model assumes a potentially infinite ageing process and is shown to provide a good fit to mortality at older ages. Yashin *et al.* (1994) further validated the model using Swedish data for male and female cohorts born in the period 1850–1879. In addition, Yashin *et al.* (1994) demonstrate that the representation of the average force of mortality in the Le Bras stochastic ageing model is equivalent to that in the fixed frailty model, resulting in the same parametric form of observed age-specific death rates. Based on the Le Bras model, Lin & Liu (2007) develop a deterministic survival rate model based on a Markov ageing process. Each state in their model represents a 'physiological age' similar to the stochastic ageing model. Unlike the Le Bras model, however, Lin & Liu (2007) assume a maximum physiological age of $n = 200$ which the authors deem an appropriate approximation to the potentially infinite ageing process. What became known as the Markov ageing model is essentially an extension of the stochastic ageing model, and has been subsequently used to assess population heterogeneity for life annuity portfolios (Su & Sherris 2012) as well as to model disability rates for calculating disability insurance premiums (Zadeh *et al.* 2014).

The stochastic ageing model is suitable for our analysis of cohort and gender differences in health-state distributions among older adults in several ways. First, it relies solely on readily available population-level mortality data. The model relies on the concept that the human ageing process can be modelled in terms of physiological ages. Each physiological age represents a different level of frailty, and an increase in physiological age represents a decline in human bodily function. Consequently, mortality data alone is sufficient to derive the implied frailty distribution for a given cohort of individuals. Second, Markov ageing models have been shown to provide reasonably a good fit to cohort mortality data from a number of countries including US, Australia and Sweden (see, e.g. Su & Sherris 2012). Third, frailty levels can be associated with different health states through a mapping process. In fact, some studies use the terms 'physiological age' and 'health index' interchangeably (Lin & Liu 2007). This allows us to explicitly identify frailty levels that may be associated

with being in an old-age disabled state and then use the model to the project levels of disability prevalence among the aged population going forward.

The remainder of the paper is structured as follows. Section 2 describes the stochastic ageing model and maximum likelihood methods. Section 3 outlines the mortality data for Australian cohorts and presents the model estimation results. This is followed by Section 4 which summarizes disability prevalence among the elderly in Australia, and uses the information to demarcate frailty levels that are associated with able versus disabled health states. Section 5 illustrates our population projections of the proportion of elderly disabled for up to 2050. Section 6 concludes.

2. Stochastic ageing model

The stochastic ageing model used is that developed and estimated in Yashin *et al.* (1994). We follow the notation, structure and assumptions of their model, with some modifications. The model is based on the concept of physiological capacity or physiological age. Specifically, the ageing process is viewed as an internal process of deterioration in terms of human body's physiological capacity; a higher physiological age reflects a decline in human body function or greater frailty. In essence, the model focuses on the relationship between mortality and physiological age, rather than a relationship between mortality and chronological age.

We posit a continuous-time discrete-state multi-state model with states defined by physiological ages and death. At any given age x, an individual can be in one of the states 0, …, n corresponding to different levels of frailty. All newborns in a cohort are assumed to start from the state 0.[1] At state i (where $i < n$), an individual can either move to the next state $i + 1$, where the chances of survival are lower, or move to the death state (absorbing state). This follows from the implicit assumption that physiological ageing is irreversible, as is human ageing. Consequently, transitions between physiological ages are always to the next higher physiological age. Herein, we will use the terms physiological age and frailty state interchangeably.

Formally, let λ_0 and μ_0 be the transition intensities from state 0 to state 1, and from state 0 to death, respectively. Mortality and morbidity rates are assumed to be an increasing function of the number of the state i, that is: at state i, the force of mortality is given by $\mu_0 + i\mu$ and the force of transiting to a worse health state is $\lambda_0 + i\lambda$. This implies that each state has unique rates of transition. The probability that an individual age x is found in the initial state 0 (P_x^0) and in the state i (P_x^i), respectively, are expressed as follows:

$$P_x^0 = P_0^0 \cdot e^{\{-(\lambda_0+\mu_0)x\}}. \tag{1}$$

[1]There may be cases that deviate from this general assumption, for example, when a newborn is born impaired with an initial damage load.

$$P_x^i = \frac{P_x^0}{i!}\left[\frac{\lambda - \lambda e^{\{-(\lambda+\mu)x\}}}{\lambda+\mu}\right]^i \prod_{k=1}^{i}\left(\frac{\lambda_0}{\lambda} + (k-1)\right), \qquad i > 0. \tag{2}$$

Under the assumption of a potentially infinite ageing process (i.e. infinite number of states), the survival function tends to the limit S_x

$$S_x = e^{\{-(\lambda_0+\mu_0)x\}}\left\{\frac{\lambda+\mu}{\mu+\lambda e^{\{-(\lambda+\mu)x\}}}\right\}^{\frac{\lambda_0}{\lambda}}, \tag{3}$$

whereby the logarithmic derivative of S_x yields the formula for the true mortality rate (μ_x)

$$\bar{\mu}_x = \mu_0 + \frac{\mu\lambda_0\left[1 - e^{\{-(\lambda+\mu)x\}}\right]}{\mu+\lambda e^{\{-(\lambda+\mu)x\}}}. \tag{4}$$

Model parameters (μ_0, μ, λ_0 and λ) are estimated using the maximum likelihood method. Applying a constant force of mortality assumption, the likelihood function (L_x) and log-likelihood function (l_x) can be expressed as follows:

$$L_x = e^{\left\{-\bar{\mu}_x E_x^c\right\}}\bar{\mu}_x^{\{D_x\}}, \tag{5}$$

and

$$l_x = \sum_x -\bar{\mu}_x E_x^c + D_x \log(\bar{\mu}_x), \tag{6}$$

where $\bar{\mu}_x$ is the mortality rate for lives aged x, E_x^c is the number of years of exposure and D_x is the observed number of deaths.[2] Note that $D_x = \hat{\mu}_x E_x^c$, where $\hat{\mu}_x$ is the observed mortality rate. We derive the maximum likelihood estimator by minimizing l_x with respect to the four parameters. The standard errors are given by the trace of the inverse of the negative Hessian (second derivative) matrix. Estimations were performed using *STATA* version 14.0.

Izsak & Gavrilov (1995) demonstrate that Equations (3) and (4) can also be derived using a cascade process model for mortality increase. Importantly, the formula for the mortality rate in Equation (4) captures not only the mortality selection developing on the population level, but also stochastic ageing developing on the individual level. Mortality selection refers to the process whereby the more frail individuals tend to die earlier, leaving survivors on average to be those with lower frailty and lower mortality. Stochastic ageing refers to the decline in human body function modelled in terms of a physiological age. The joint influence of these two processes generates the distribution of surviving individuals by physiological age, or the so-called implied frailty distribution for a given birth cohort.

Assuming the stochastic ageing model is a good fit, the proportion of survivors at any given age x is expressed as follows:

[2]More formally, the likelihood function in Equation (5) is proportional to the expression given, rather than equal to it.

$$\pi_x^i = \frac{P_x^i}{\sum_{i=0}^{n} P_x^i}, \qquad i = 0, 1, 2, \ldots, n, \tag{7}$$

where it is to be recalled that the numerator P_x^i is the probability that an individual aged x is found in the frailty state i. The denominator is the sum of the probabilities across all possible states (ranging from zero to n). Accordingly, we have $\sum_{i=0}^{n} \pi_x^i = 1$. Plotting the graph of π_x^i by physiological age yields the implied frailty distribution.

3. Data and model estimation

We use mortality data from the Human Mortality Database (HMD), which is compiled from various sources including the Australian Bureau of Statistics, Australian Centre for Population Research and Australian Institute of Health and Welfare. Specifically, cohort data for the 1865–1895 birth cohorts is employed so as to cover a broad cross-section of older Australians who are retired or near retirement in the mid-twentieth century.

Since the stochastic ageing model is primarily suitable for adult and old-age mortality, we focus on the age range of 45 onwards. Although HMD contains death rates for older adults up to age 109, we set the maximum observed age at 105 due to the limited risk exposure data at extreme advanced ages. Note that the use of cohort data reduces the significance of mortality improvement that must be allowed directly when using period life tables. The cohort force of mortality for birth cohorts 1880 and 1890 for both males and females is shown in Figure 1. On the log scale, these are close to linear, and they support the use of the Gompertz–Makeham model of mortality for the age range considered (see Appendix 1 for details).

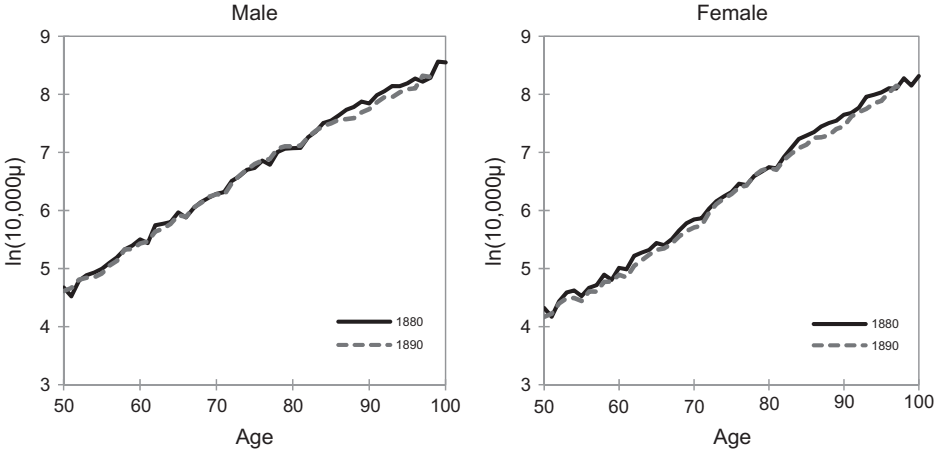

Figure 1. Observed cohort death rates by chronological age: log transform.

3.1. Model estimation

The maximum likelihood parameter estimates for the four mortality and morbidity parameters are shown in Table 1 for selected cohorts. We note that the standard errors are generally small and appear reasonable when compared to the parameter values. In particular, very low standard errors for the λ parameters are indicative that the values of λ are spread over a very narrow range of values and plausibly constant across cohorts. Figure 2 plots the fitted parameters for the entire range of birth cohorts analysed (1865–1895 cohorts). We see some cohort-to-cohort variation in the estimated parameters.

Figure 3 displays a matrix of scatter plots showing bivariate correlations among pairs of variables. Some correlations are weak, for example, the correlation coefficient for λ_0 and λ is only -0.04 for males. The correlation coefficient between λ_0 and μ_0 for males is 0.5 which is moderate. The highest correlation is between the variables μ and μ_0 for males (coefficient of -0.9) although the corresponding value for females is somewhat lower at -0.6. This negative correlation between the variable pair was also noted in Yashin *et al.* (1994) using Swedish population data. Like Yashin *et al.* (1994), we noted a moderately negative correlation between μ and λ which ranged from -0.5 to -0.6. The relationship between μ and λ can be rationalized as follows: in any given frailty state i, a higher force of mortality (μ) means that individuals are more likely to die thus resulting in a lower rate of transition to the next frailty state (λ).

3.2. Frailty distribution and trend across cohorts

In the stochastic ageing model, the heterogeneity of population mortality is measured by the distribution of physiological ages through time. Specifically, this heterogeneity arises from the different health conditions of individuals at the same age. Of interest are the gender and cohort differences in health-state distributions among older Australians at advanced ages.

To examine gender differences, we plot the implied frailty distributions for elderly males and females for two illustrative birth cohorts (the cohorts of 1880 and 1890). Figure 4 shows the empirical density functions for the distribution of physiological age at age 65 (or π^i_{65}). For each cohort, we see that the female distribution is to the right of the male distribution, with a fatter tail in frailty states corresponding to high physiological ages. This is representative of the rest of the birth cohorts, and implies that surviving age 65 females are biologically older (less healthy) than their same-aged male counterparts. These findings suggest that the ageing process develops at a faster pace for women than for men in their 60s and 70s. Note that physiological ages do not correspond one-to-one with chronological ages.

Table 1. Estimated parameters by gender for selected cohorts. Note: Standard errors are shown in parentheses.

	1880 male	1890 male	1880 female	1890 female
μ_0 ($\times 10^{-3}$)	2.39 (0.48)	2.11 (0.41)	4.02 (0.27)	3.18 (0.23)
μ ($\times 10^{-6}$)	6.98 (0.64)	9.47 (0.77)	2.05 (0.16)	2.61 (0.18)
λ_0	1.075 (0.191)	0.547 (0.043)	0.808 (0.087)	0.677 (0.059)
λ	0.093 (0.002)	0.100 (0.002)	0.109 (0.002)	0.107 (0.002)

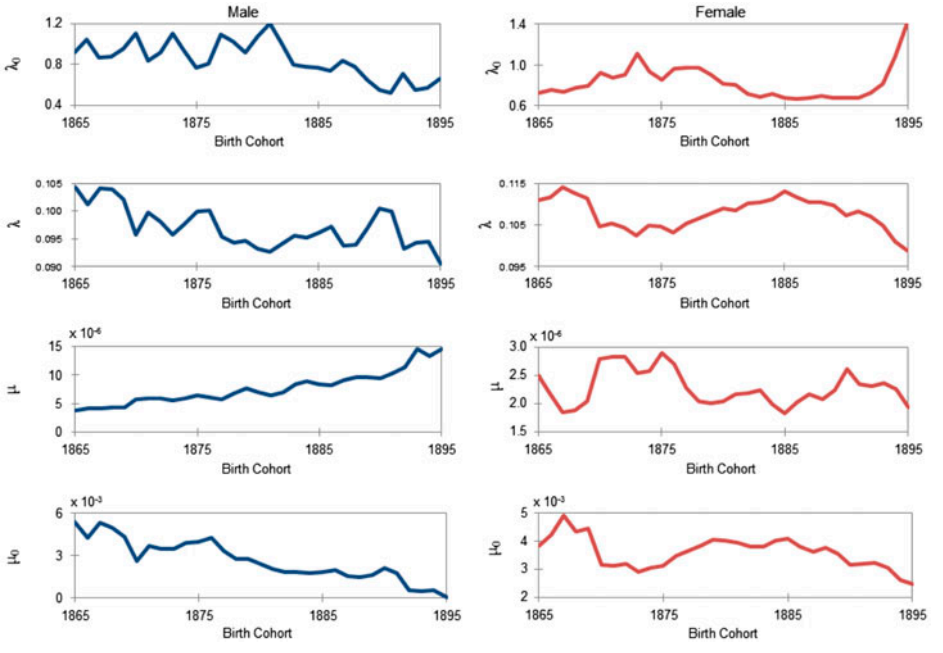

Figure 2. Values of the model parameters across birth cohorts.

Figure 5 highlights how the frailty distributions differ by chronological age. Importantly, the level of heterogeneity of the cohort increases with age. At younger ages, say age 55, the distribution is relatively narrow and concentrated. At more advanced ages, for example age 95, the density curve flattens and the distribution is wider. This suggests that the cohort is more heterogeneous at more advanced ages, a finding consistent with Su & Sherris (2012). Additionally, the female distribution is consistently positioned to the right of the male distribution regardless of the chronological age evaluated.

Figure 6 illustrates the limiting distribution of π by frailty state for the two illustrative birth cohorts. The limiting distribution is the implied frailty distribution for cohort individuals at very advanced ages (i.e. $x \longrightarrow \infty$).[3] Not surprisingly, we find that the limiting distribution for females lies to the right of that for males all the birth cohorts examined. This has important implications. The fact that surviving females at very old ages are, on average, in worse health states than males from the same cohort implies that Australian females who die at advanced ages die at an older biological age (i.e. in frailer states) than Australian males. We conclude from these findings that the ageing process develops at a faster pace for women than for men across all post-retirement chronological ages, causing the former to move quicker into worse states of health.

[3]Theoretically, the model does not impose an upper limit to the maximum age which might be attained. For the cohorts examined in this paper, we find that the limiting age is approximately 219 on average. See also Appendix 1 for a mathematical derivation of the limiting average physiological age ($\bar{i}_\infty$). $\bar{i}_\infty$ represents the average level of frailty among survivors at very advanced ages within a given cohort.

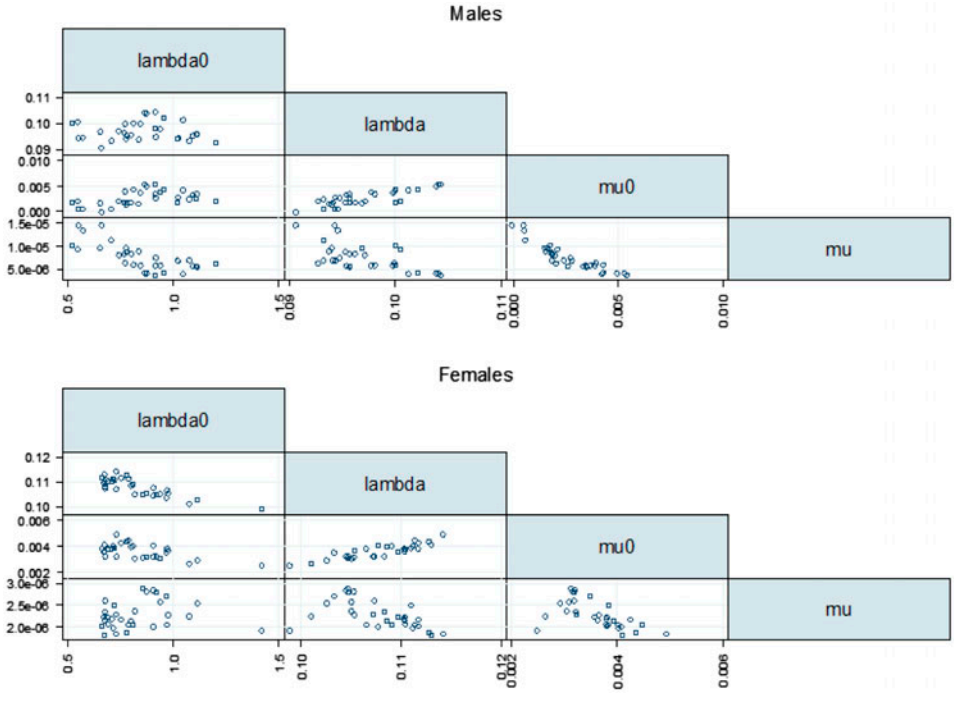

Figure 3. Correlations between fitted model parameters.

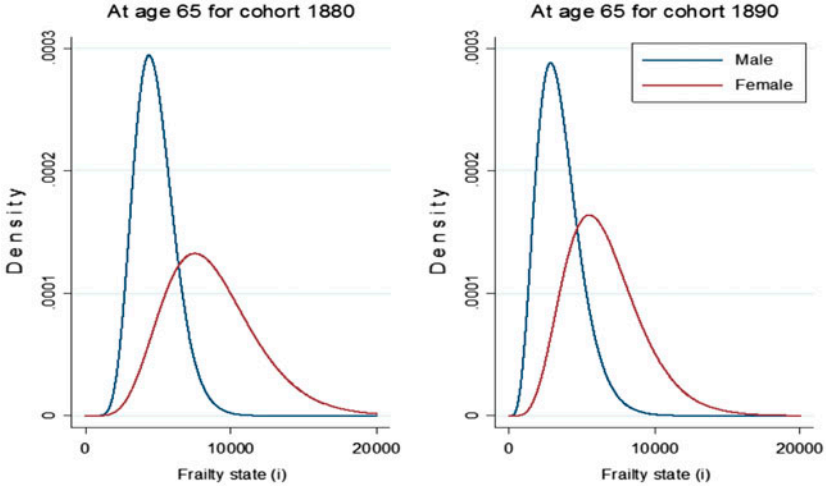

Figure 4. Distribution of physiological age at age 65 for different birth cohorts.

Further analysis reveals that this male–female divergence in health deterioration, which is already evident at age 65, grows considerably as individuals advance in age (see Figure 7). Of note is the unabated pace of rapid health deterioration observed for older females over a

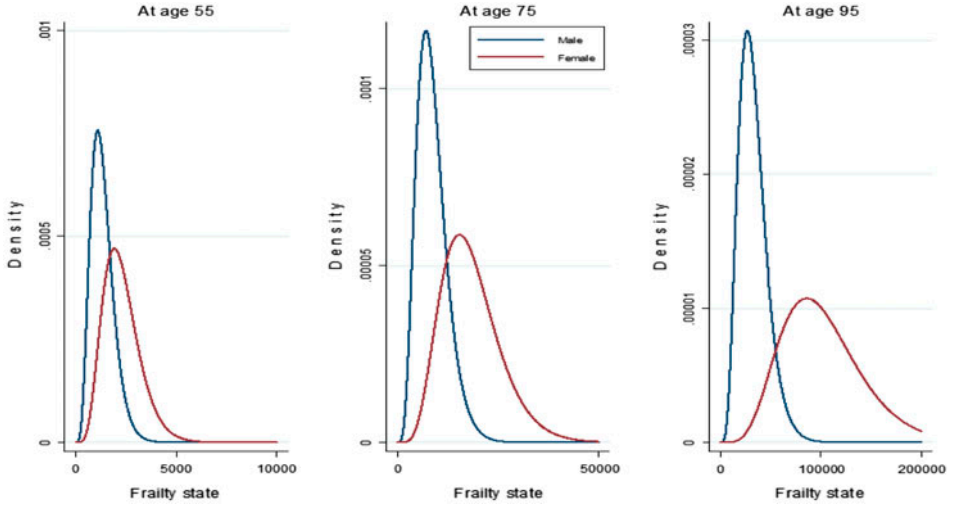

Figure 5. Distribution of physiological age at different ages (1890 birth cohort).

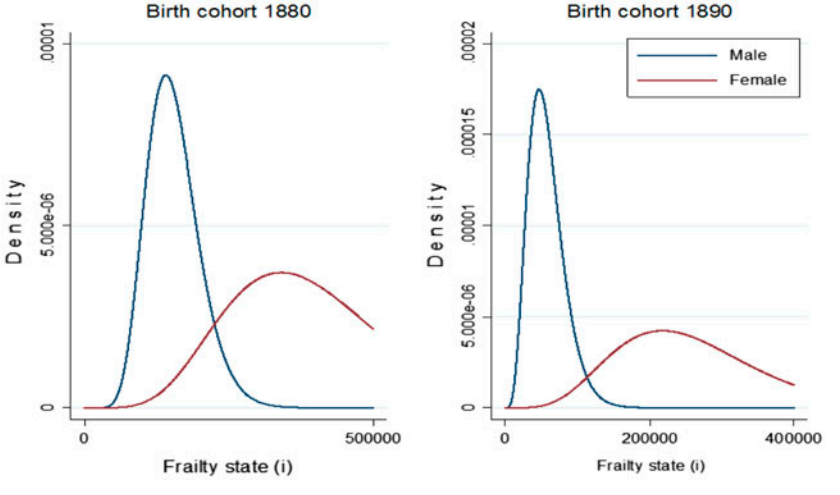

Figure 6. Limiting frailty distribution for Australian males and females born in 1880 and 1890.

relatively protracted age range, being age 90 up till about age 115. The one-to-one mapping of physiological ages to chronological ages in Figure 7 is, especially useful in helping one relate the frailty states to individual chronological ages. Recall that a newborn starts from frailty state 0. At a retirement age of 65, a typical Australian male (female) would have deteriorated to frailty state 5085 (8731). Not surprisingly, those who survive to age 80 are associated with frailty states in the upward range of 17,700–37,800. The frailty distribution approaches the limiting distribution at around ages 200–240.

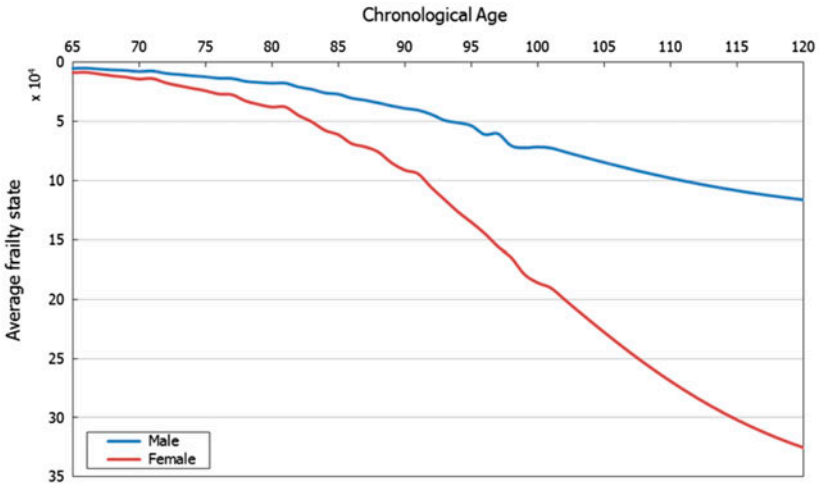

Figure 7. Average i by chronological age across all birth cohorts.
Note: The vertical axis is inversed in the Figure to portray *deteriorating* health (frailty) states.

To examine cohort differences, we graph the average physiological age for individuals at age 65 ($\bar{i}_{65}$), and separately, for individuals at age 85 ($\bar{i}_{85}$) across all the birth cohorts analysed in Figure 8. Interestingly, the results using Australian data show that the average frailty

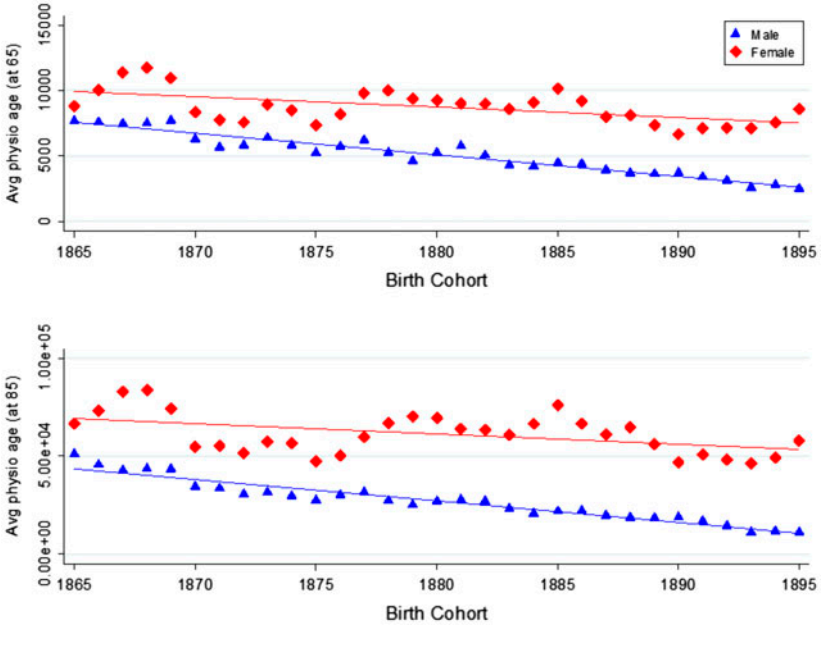

Figure 8. Average physiological age across cohorts evaluated at age 65 (top) and age 85 (bottom).

level for both males and females has trended downwards across time. This suggests that recent cohorts of survivors are on average less frail and generally experience better health than their predecessors. Various factors could have accounted for these changes in frailty level including differences in disease prevalence, differences in lifestyle and behaviour as well as changes in environmental risk factors over time. Another contributing factor might be the government policy: considerable attention had been devoted to men's and women's health at the national level in Australia over the past years (Commonwealth of Australia 2013).

3.3. *Some first conclusions*

Our empirical results thus far are consistent with Mathers *et al.* (2001) and Mathers (1996) who find that elderly females in Australia tend to experience greater disability severity than males, resulting in more years lived in disability. Clinical studies have also shown that women are generally more predisposed to developing frailty than men because of hormonal factors, lower baseline levels of muscle mass and other reasons (see, e.g. Walston & Fried 1999). Older women are more vulnerable to falls, functional decline and morbidity because they are structurally weaker. Hence, while females enjoy longer life expectancies than males (achieve higher chronological age), they are also likely to be in worse states of health at old age and death (higher physiological age).

We observe greater heterogeneity in health states among older women as compared to older men in Australia. The frailty distributions for females are markedly more dispersed than those for males across the cohorts examined. This may be partly rationalized by women's susceptibility to very different life experiences, e.g. childbirth, causing them to differ considerably in terms of their physiological capacity and thus their level of frailty.

Our finding that average frailty levels have fallen over time for both male and female birth cohorts offers a fresh perspective to the current debate on whether morbidity has expanded or compressed among the aged population in Australia. Recent analyses by the Australian Institute of Health and Welfare (AIHW) found no evidence of absolute expansion or absolute compression of morbidity over the period 1998–2009; there was also no indication of a dynamic equilibrium scenario (AIHW 2012). This stands in vivid contrast to the experience in other developed countries, such as the US, Denmark, Italy, the Netherlands and Finland, where declines in functional disability have been documented among elderly persons aged 65+ (Lafortune *et al.* 2007).

4. Disability prevalence and mapping to health states

In this section, we provide an overview of the current levels of disability prevalence among the elderly in Australia and then describe how this information can be utilized to map frailty states to health states in the model.

4.1. Disability prevalence among the elderly in Australia

Since the 1980s, the Australian government has collected information on people with disability. Conducted by the Australian Bureau of Statistics, the periodic disability surveys in Australia follow the conceptual framework and major concepts of the International Classification of Functioning, Disability and Health. The most recent survey is the 2012 Survey of Disability, Ageing and Carers (SDAC) administered throughout Australia from August 2012 to March 2013.

The SDAC survey measures the prevalence of disability in Australia and aims to provide a demographic and socio-economic profile of people with disability across a wide spectrum of age groups and their carers. Consequently, SDAC's definition of 'disability' is rather broad: 'disability' is defined as any limitation, restriction or impairment, which restricts everyday activities and has lasted or is likely to last for at least six months (ABS 2013a). Examples range from loss of sight that is not corrected by glasses, to arthritis which causes difficulty dressing, to advanced dementia that requires constant help and supervision. In this present paper, we focus only on adults who have profound or severe levels of disability. This aligns with the Intergenerational Report 2010 prepared by the Commonwealth Government, which notes that older individuals who seek long-term aged care are likely to be those who suffer from high-severity core activity limitations (Commonwealth of Australia 2010).[4]

Table 2 summarizes the cross-sectional prevalence levels of disability reported by the ABS for Australians aged 60 and above in recent survey waves. We observe that gender-specific disability levels are relatively stable from year to year. This observation is an important one because it rationalizes why the Australian Governmental projections on aged care spending assume disability rates to remain at current levels for the intermediate term up till 2050 (Commonwealth of Australia 2010). Note also that higher proportions of elderly women in the population suffer from profound or severe core activity limitations as compared to elderly men. These gender differences are, especially striking for ages past 80.

4.2. Mapping physiological ages to health states

The ageing process is an internal process of deterioration in terms of the human body's physiological capacity. An increase in the physiological age i thus reflects worse health. To demarcate physiological ages (frailty states) that likely correspond to being in an old-age disabled state, we use the ABS data on the proportion disabled in the Australian population

[4]The Intergenerational Report is commissioned by the Commonwealth Government every 5 years and is prepared by The Treasury Department. It assesses the fiscal and economic challenges of an ageing population, implications of demographic change for economic growth and financial implications of continuing current policies and trends over the next four decades. Core activities comprise self-care tasks such as bathing or toileting, mobility tasks such as moving around at home or using public transport as well as communication with family and friends. The SDAC surveys specify four levels of severity (profound, severe, moderate and mild) based on the person's ability to perform tasks relevant to these areas and on the amount of help required. Specifically, individuals who are classified as having 'profound' or 'severe' core activity limitations require personal help or supervision constantly or sometimes, and are unable to perform tasks without help or supervision.

Table 2. Proportions with profound or severe core activity limitation by age and gender (in percentages).

	Males					Females				
Age	2012	2009	2003	1998	Avg	2012	2009	2003	1998	Avg
60–64	8.0	8.2	7.6	8.3	8.0	9.5	8.8	9.8	9.3	9.4
65–69	9.7	8.4	9.5	7.8	8.9	9.1	9.4	10.3	9.2	9.5
70–74	12.0	12.8	11.6	11.8	12.1	12.8	15.1	17.4	15.0	15.1
75–79	15.2	16.2	18.7	19.0	17.3	21.1	19.1	21.5	24.9	21.7
80–84	26.6	23.5	27.3	24.3	25.4	32.2	31.4	40.5	35.6	34.9
85–89	38.1	41.1	38.9	53.0	42.8	51.0	49.9	57.3	60.8	54.8
90+	62.0	58.0	59.4	64.9	61.1	69.1	75.0	79.1	83.7	76.7

Source: ABS (2004, 2013a).

in 1998, 2003, 2009 and 2012.[5] The mapping process aims to identify the cut-off physiological age (denoted i^*) such that individuals of age x in a given cohort who are found in state i^* and beyond are classified as being functionally disabled. We begin by obtaining the abridged frailty distributions for each age group corresponding to the ABS survey data from our fitted model. For instance, the proportion of female survivors found in state i for age group 80–84 (π^i_{80-84}) is derived using the average π^i_{80}, π^i_{81}, π^i_{82}, π^i_{83} and π^i_{84} across all female birth cohorts examined. This process is repeated for each frailty state (up to $n \approx 200{,}000$) and for each age group given in the ABS data.

Figure 9 (left panel) displays the abridged frailty distribution for females age group 80–84 built from our estimates of π^1_{80-84}, π^2_{80-84}, π^3_{80-84} and so on. To calculate the cut-off physiological age (i^*) for this group of individuals, we derive the cumulative density function and simultaneously extract relevant information from the SDAC surveys. The right panel of Figure 9 illustrates this procedure. On the cumulative density function for age 80–84 females, we graph a horizontal line indicating the average proportion of disabled females per SDAC surveys (being 34.9%; see Table 2). This intersect translates to a cut-off physiological age (i^*) of 50,413 on the horizontal axis. For our purposes, persons who are found in state i^* or beyond will thus be classified as old-age disabled (i.e. have profound or severe core activity limitations which likely require long-term care services).

Age-specific disability cut-off ages (i^*_x) are obtained by fitting simple linear functions to the data points for the aggregated age groups (see Figure 10). The R^2 values are 99.2% for males and 99.4% for females suggesting reasonable fit. Not surprisingly, we see from the scatterplot that i^* increases monotonically with chronological age for the range of ages examined. This is consistent with the relationship observed between SDAC actual disability prevalence rates and age.

With the cut-off physiological ages in hand, we can compute disability prevalence rates by birth cohort and gender as follows. First, we apply the set of age-specific cut-off age to all birth cohorts examined. Next, we generate the frailty distribution at each exact age for every cohort examined and calculate the proportion of survivors who exceed the specified

[5]Older SDAC survey waves in 1981, 1988 and 1993 are not used in our analysis because the final age group for those earlier waves terminated at age 75+ instead of age 90+.

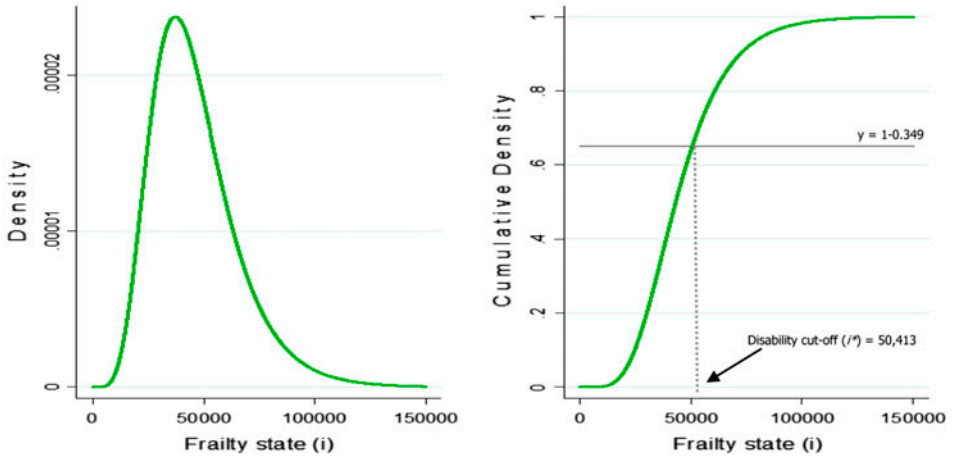

Figure 9. Abridged frailty distribution (*left*) and cumulative distribution function (*right*) for females in age group 80–84 across all birth cohorts.

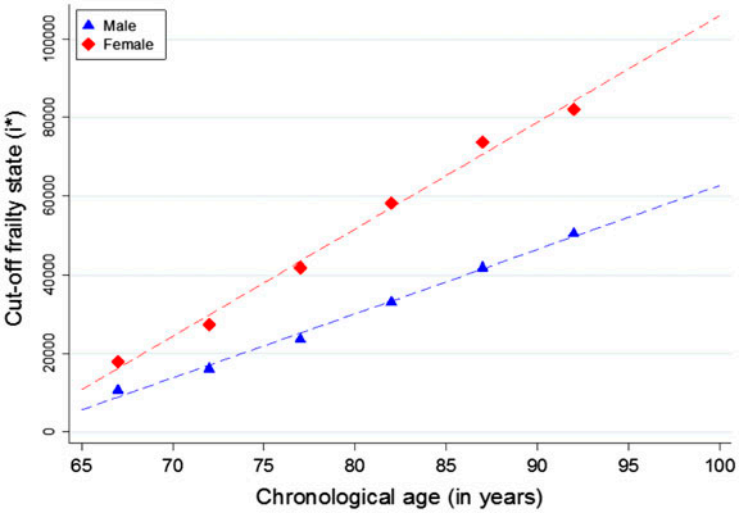

Figure 10. Age- and gender-specific cut-off physiological ages.

cut-off age. As an example, let us focus on females of exact age 85, whereby $i^*_{85} = 65,196$ per Figure 10. Assume that this value applies to all cohorts. We subsequently observe from the frailty distribution that 49.7% of age 85 female survivors from the 1870 cohort have a physiological age of 65,196 or higher (i.e. old-age disabled). The corresponding proportions for the 1880 and 1890 cohorts are 53.0 and 35.3%, respectively. Repeating this calculation for each cohort and each chronological age, we derive the cohort disability prevalence rates. The output is shown in Figure 11 for several illustrative birth cohorts.

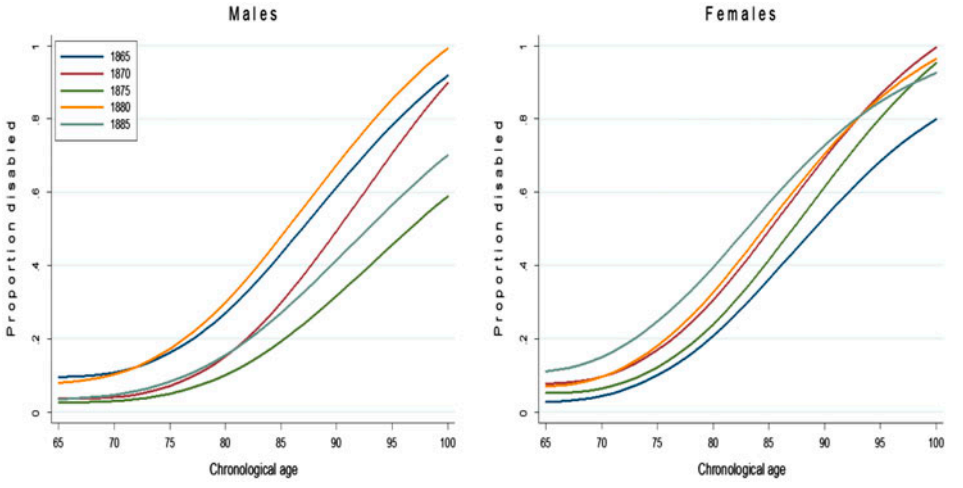

Figure 11. Estimates of proportion disabled by age for male and female cohorts.

5. Forecasting disability prevalence with time-series models

A goal of this paper is to use the empirical model to develop forecasts of disability prevalence levels, and also mortality, among the Australian elderly up to 2050. Time-series methods are useful in this regard because they allow for a flexible modelling of trend, and have been successfully applied in numerous demography and actuarial science studies to forecast aggregate mortality, fertility and total population (Carter & Lee 1986; McNown & Rogers 1989; Russolillo *et al.* 2011). For example, McNown & Rogers (1989) demonstrated that overall forecast accuracy is reasonably high when univariate time-series methods are applied to forecast the US mortality over a multi-period horizon.

5.1. *Mortality forecasts*

To generate forecasts of the gender-specific mortality rates (the $\bar{\mu}_x$), we apply univariate time-series models to the model parameters. The four parameters in the stochastic ageing model presented earlier are fitted using cohort mortality data from 1865 to 1895. Since a 65-year-old person in 2050 is born in 1985, we need to project the modelled parameters up to birth cohort 1985. We begin by logarithmically transforming the parameters and testing the residuals using the Augmented Dickey–Fuller test.[6] First, differencing on each parameter is sufficient to achieve stationarity. Several autoregressive integrated moving average models, including white noise, AR(1) and AR(2), are then identified and estimated for each series. For each parameter, we pick the model which yields the smallest Bayesian information criterion value (see Table 3).

[6]This transformation stabilizes the variances and sets a lower bound of zero on parameter forecasts.

Table 3. Bayesian information criterion values from fitting alternative stationary time-series models.

	μ_0	μ	λ_0	λ
Males				
White noise	**11.10**	**−50.45**	−18.37	**−123.23**
AR(1)	14.47	−47.10	−15.51	−120.20
AR(2)	14.96	−49.71	**−21.82**	−121.89
		Females		
White noise	**−51.67**	**−43.73**	**−43.68**	**−147.88**
AR(1)	−48.30	−42.20	−42.91	−145.22
AR(2)	−45.25	−41.88	−39.56	−142.07

Note: For each gender subgroup, the smallest BIC value obtained for each parameter is bolded for emphasis.

Figure 12 illustrates our forecasts of the Australian mortality by gender for birth cohorts 1896–1985 at exact ages 75 and 85. We see that these long-range forecasts are sensible when compared against the hazard values in the data region. Also, female mortality is forecasted to persistently decline across successive cohorts for all ages examined. This is reasonable considering that humans are likely to experience mortality improvements over time, which could stem from improvements in health technology or other factors. Mortality forecasts for males also exhibit an overall declining trend for the majority of ages examined,

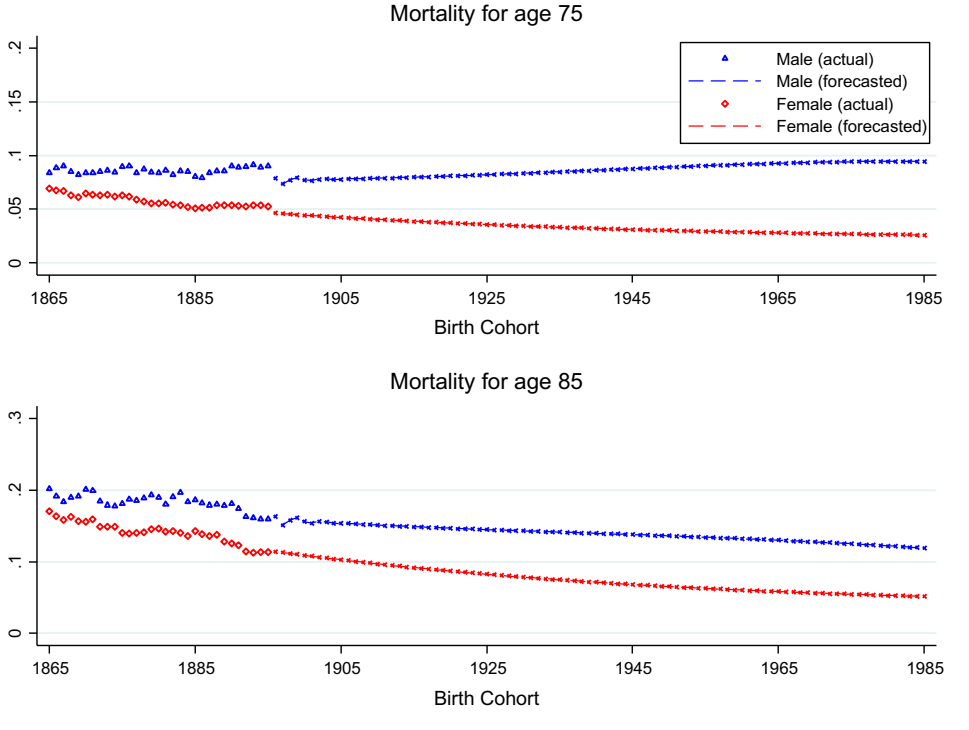

Figure 12. Forecasted and actual mortality rates at selected ages.

except for some ages below 80 where the trend is level or mildly increasing (see plot for $\bar{\mu}_{75}$). This could reflect historical patterns in mortality among the younger old males.

5.2. *Disability prevalence forecasts*

Individual parameter forecasts are used to derive the frailty distributions for male and female cohorts in the forecast region (i.e. cohort 1896 and beyond). Comparing forecasted distributions to the earlier fitted distributions, we observe that forecasted distributions tend to be narrower. This suggests that, in any given cohort, there are smaller proportions of individuals in very high physiological ages, a finding that is consistent with the decline in historical average frailty levels observed in Figure 8. Age-specific disability cut-offs (i_x^*) are then applied to each individual-forecasted distribution to obtain the proportion disabled by cohort and age. For instance, the proportion disabled at age 75 evaluated for cohort 1945 will correspond to calendar year 2020, cohort 1955 will correspond to calendar year 2030 and so on.

Figure 13 shows the projected disability prevalence levels for ages 75 and 85. Three observations are worth highlighting. First, in any given year, a larger proportion of the oldest old suffer from profound or severe levels of disability as compared to the younger old. For example in 2025, it is estimated that 12.2 and 38.0% of age 75 and age 85 males will be disabled, respectively. This is logical since individuals who are in advanced ages are more likely to experience core activity limitations. Second, old-age disability remains more prevalent among females than males over the forecast horizon. Third, the forecast trends differ by gender. For males, the proportion disabled is forecasted to fall monotonically over time. This resonates with the narrowing of the frailty distributions noted above. Interestingly, however, the proportion of disabled females is forecasted to dip slightly and then increase at the ages evaluated. This is because the effect of greater longevity starts to outweigh the effect of

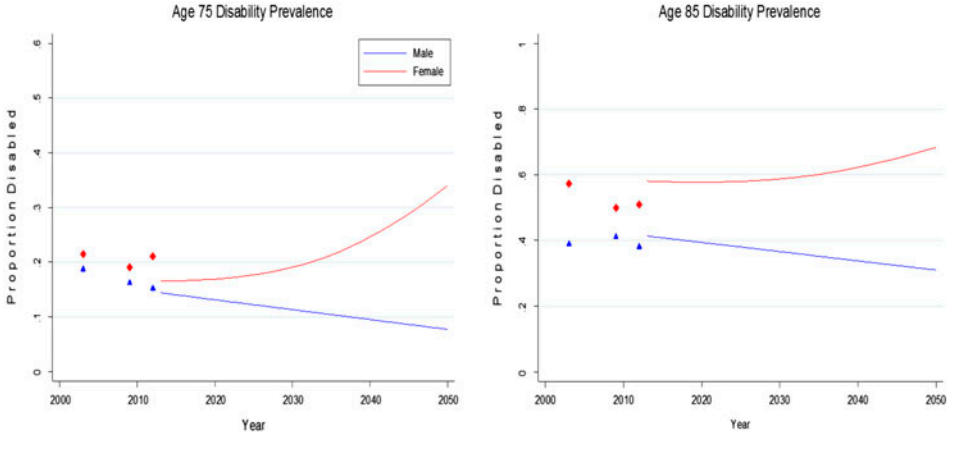

Figure 13. Projected disability prevalence levels for individuals of age 75 and age 85 Note: The symbols in the graphs indicate actual abridged disability prevalence levels as reported in the 2003, 2009 and 2012 SDAC surveys. The lines indicate our model projections.

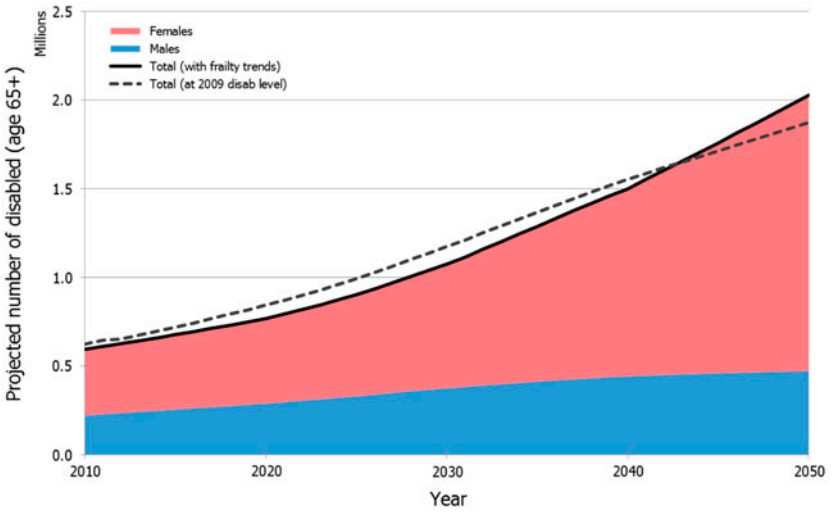

Figure 14. Projected number of disabled elderly under two different scenarios.

declining frailty levels as we go further out into the forecast horizon. The persistent fall in female mortality rates (recall Figure 12) implies that more females are living to very old ages, whether healthy or unhealthy. Consequently, the survivors aged 75 or 85 in these future cohorts are not as select (in terms of hardiness) as those in previous cohorts. A comparison of the females' forecasted distributions over time confirms that the mode has shifted right, indicating that a greater majority of the surviving females in later cohorts are indeed less healthy than their predecessors.

To estimate the number of elderly Australians with disability up to 2050, we combine the above disability prevalence rate projections with whole-of-population projections.[7] Figure 14 shows the number of disabled older Australians age 65+ projected under two different scenarios. In the first scenario, we employ the disability prevalence rates estimated from our stochastic ageing model. Results reveal that the number of persons age 65+ with disability is projected to rise from 767,753 to 2027,363 (or 164%) between 2020 and 2050 (see solid line). For comparison, we turn to consider an alternative scenario in which disability levels remain constant at 2009 levels over the projection period. This mirrors the current assumption used in the Australian Government's estimates for future aged care spending (Commonwealth of Australia 2010).[8] Under this scenario, the number of persons aged 65 or over with disability is expected to rise from 842,663 to 1874,182 (or 122%) between 2020 and 2050 (see dotted line). The crossover point occurs at 2043.

One can conclude from these results that even if current levels of disability prevalence within the aged population were to remain stable over time, the absolute number of older

[7]Appendix 2 provides details about the population data used.

[8]Page 145 of the Intergenerational Report 2010 states: 'The Australian Bureau of Statistics surveys and Australian Institute of Health and Welfare analyses continue to suggest a relatively stable prevalence rate of severe disability among older Australians. Accordingly, the base projections presented here do not assume any change in severe disability rates.'

adults with disability will increase sizably going forward because of population ageing and the burgeoning share of the elderly in the Australian population. In fact, the Commonwealth Government projects that spending on aged care in Australia will rise from 0.8 per cent of GDP in 2010 to 1.8% of GDP by 2050 (Commonwealth of Australia 2010). By factoring historical trends in frailty into the computations and allowing for old-age disability prevalence rates to vary, a fuller picture emerges. Although Australia will benefit from declines in disability prevalence in the initial years, the effect of rising disability prevalence among older females in the later years will eventually outweigh the effect of falling disability prevalence among older males. Consequently, at the population level, the number of elderly Australians requiring aged care services by 2050 is estimated to exceed that projected under the constant prevalence rate scenario.

6. Conclusion

In this paper, we used the available population mortality data along with the Le Bras stochastic ageing model to examine gender and cohort differences in health-state distributions among older Australians aged 45 and above born in 1865–1895. Results are fairly encouraging. We find that, on average, survivors today are healthier and hardier than their predecessors. Average frailty levels have fallen over time for both male and female birth cohorts. A more detailed comparison between genders reveal that older females tend to experience a faster pace of health deterioration than their male counterparts causing them to move quicker into worse states of health. Health states are also more heterogeneous among older women than older men.

Individuals associated with very high physiological ages and who are very frail may suffer from high-severity core activity limitations. Our whole-of-population estimates which take into account these patterns in frailty and projected population growth indicate that the number of persons aged 65 and above with disability will increase by 164% between 2020 and 2050. This exceeds that projected under governmental assumptions and thus bears important implications for future government aged care spending. One limitation of our study pertains to the use of univariate time-series methods in forecasting cohort mortality which implicitly assumes independence between the model parameters. Future research can explore using multivariate methods to examine possible linkages among the various parameters in the Le Bras ageing model and related statistical issues.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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Appendix 1

This Appendix contains the derivations for several useful formulas with relation to the stochastic ageing model.

First, we show that under certain assumptions, the stochastic ageing model can be approximated by the Gompertz–Makeham mortality model. From Equation (4), we have:

$$\bar{\mu}_x = \mu_0 + \frac{\mu\lambda_0\left[1 - e^{\{-(\lambda+\mu)x\}}\right]}{\mu + \lambda e^{\{-(\lambda+\mu)x\}}}$$

Rearranging,

$$\bar{\mu}_x = \mu_0 + \frac{\mu\lambda_0\left[1 - e^{\{-(\lambda+\mu)x\}}\right]}{\left[\frac{\mu}{\lambda} + e^{\{-(\lambda+\mu)x\}}\right]\lambda}.$$

Following from Izsak & Gavrilov (1995), in the case that $\mu \ll \lambda$, then $\frac{\mu}{\lambda} \to 0$ and $\bar{\mu}_x$ may be approximated by:

$$\begin{aligned}\bar{\mu}_x &= \mu_0 + \frac{\mu\lambda_0\left[1-e^{\{-(\lambda+\mu)x\}}\right]}{\left[e^{\{-(\lambda+\mu)x\}}\right]\lambda} \\ &= \mu_0 + \frac{\mu\lambda_0}{\lambda}e^{(\lambda+\mu)x} - \frac{\mu\lambda_0}{\lambda} \\ &= \left(\mu_0 - \frac{\mu\lambda_0}{\lambda}\right) + \frac{\mu\lambda_0}{\lambda}e^{(\lambda+\mu)x}.\end{aligned}$$

□

Note that the stochastic ageing model (or cascade process model) may also be separately approximated by the Makeham-Beard model of mortality (see details in Richards 2008).

Second, we show that the limiting distribution of π may be derived from the empirical density function for the distribution of physiological age at age x. From Equation (7), we have:

$$\pi_x^i = \frac{P_x^i}{\sum_{i=0}^{n} P_x^i},$$

where the frailty state $i = 0, 1, 2, \ldots, n$.

Substituting Equations (2) and (3) into the above formula:

$$\pi_x^i = \frac{1}{i!}(V_x)^i(1 - V_x)^{\frac{\lambda_0}{\lambda}}\prod_{k=1}^{i}\left(\frac{\lambda_0}{\lambda} + (k-1)\right)$$

where

$$V_x = \frac{\lambda - \lambda e^{\{-(\lambda+\mu)x\}}}{\lambda + \mu}.$$

As $x \to \infty$, $V_x \to \frac{\lambda}{\lambda+\mu}$ and $1 - V_x \to \frac{\mu}{\lambda+\mu}$.

Thus, the limiting distribution of π is:

$$\pi_{\infty}^{i} = \frac{1}{i!}\left(\frac{\lambda}{\lambda+\mu}\right)^{i}\left(\frac{\mu}{\lambda+\mu}\right)^{\frac{\lambda_0}{\lambda}}\prod_{k=1}^{i}\left(\frac{\lambda_0}{\lambda}+(k-1)\right).$$

A useful summary measure of frailty or health which can be derived from the stochastic ageing model is the so-called 'limiting average physiological age' (denoted $\bar{i}_{\infty}$). This is the average level of frailty among survivors at advanced ages within a cohort. Given that the mortality rate at age x is the number of individuals who die divided by the number of individuals who are alive as at age x, we have:

$$\bar{\mu}_x = \frac{\sum_{i=0}^{n} P_x^i(\mu_0 + i\mu)}{\sum_{i=0}^{n} P_x^i}.$$

Rearranging,

$$\begin{aligned}\bar{\mu}_x &= \mu_0 \frac{\sum_{i=0}^{n} P_x^i}{\sum_{i=0}^{n} P_x^i} + \mu \frac{\sum_{i=0}^{n} P_x^i \cdot i}{\sum_{i=0}^{n} P_x^i} \\ &= \mu_0 + \mu \cdot \bar{i}_x.\end{aligned}$$

Consequently, the average physiological age at age x can be expressed as follows:

$$\bar{i}_x = \frac{\bar{\mu}_x - \mu_0}{\mu}.$$

As $x \longrightarrow \infty$, then $\bar{\mu}_t \rightarrow \mu_0 + \lambda_0$, which yields the mathematical formula for the limiting average physiological age as follows:

$$\bar{i}_{\infty} = \frac{\lambda_0}{\mu}.$$

Appendix 2

We use the population statistics for abridged age groups sourced from the ABS. The projected numbers of males and females are based on ABS' Series B assumptions of births, deaths and migration in the population; see ABS (2013b) for details. Table A1 shows an extract of the projected number of total persons aged 65 and above for selected years up to Year 2050. Notably, the number of elderly persons is expected to increase almost twofold from 4.2 million in 2020 to 7.9 million in Year 2050. These figures are broadly in line with the population projections separately published by the Treasury Department in the Intergenerational Report 2010 (Commonwealth of Australia 2010).

Table A1. Number of persons aged 65 and above in the population (in millions).

	Our paper	Intergenerational Report 2010
2010	3.0	3.0
2020	4.2	4.2
2030	5.6	5.6
2040	6.8	6.9
2050	7.9	8.1

Source: ABS (2013b) and Commonwealth of Australia (2010).