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THE 'GOLDEN GENERATIONS' IN HISTORICAL CONTEXT

By Michael Murphy

ABSTRACT

Assumptions about future mortality are more important than those for factors such as fertility, migration, disability trends or real interest rates for cost projections of the U.S. Old Age, Survivors, Disability and Health Insurance scheme. Recently, one factor has been assumed to be the key driver of future mortality in both official British population projections and actuarial ones: a 'cohort effect' associated with a group who were born in a period centred on the early 1930s who have been identified as having experienced particularly rapid improvements in mortality rates and are often referred to as the 'golden generations' or 'golden cohorts'. The concept of 'cohort effects' is discussed; limitations of national-level cohort data considered; and methods for identifying such effects are reviewed. Particular attention is given to the analysis of populations which have been identified as having clear cut cohort effects; those of Britain and Sweden in the later part of the nineteenth century and early twentieth century, as well as the contemporary British population. The likely magnitude of such effects is discussed using a stylised model to assess the extent to which members of the 'golden generations' are especially privileged.

KEYWORDS

Mortality; Cohort Patterns; Historical Demography; Golden Generations

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1. Introduction

Pensions liabilities in the OECD countries are estimated as 20 trillion dollars (or 13 trillion pounds); countries such as the United Kingdom, U.S.A. and Switzerland had, in 2005, pensions liabilities larger than their annual GDP (OECD, 2007; SwissRe, 2007, 2008). Sensitivity analysis of costs undertaken for the 2008 OASDI Trustees Report of the U.S. Old Age, Survivors, Disability and Health Insurance scheme found that assumptions about future mortality were more important than those for factors such as fertility, migration, disability trends or real interest rates (real earnings over

the 50-year period was the only factor that had a larger impact[1]) (Social Security Administration, 2008; see also Antolin, 2007). Both future levels of mortality and current forecasts of these will have profound implications for planning of both public and private institutions and are potential sources of financial instability.

Official projected increases in life expectancy in Britain between 2005 and 2030 of over five years in the 2008-based projections (Office for National Statistics, 2009a) are rather higher than values of three to four years by Eurostat for Western European EU countries and just over three years by the Bureau of the Census for the U.S.[2] for the same base year. In the past, projected mortality improvement has usually been assumed to cease or to be substantially reduced progressively ahead of the period when the projections were made (Murphy, 1995). Recently, one factor has been assumed to be the key driver of future mortality in both official British population projections (e.g. Office for National Statistics, 2008b) and actuarial ones (e.g. Continuous Mortality Investigation, 2007): a 'cohort effect' associated with a group who were born in a period centred on the early 1930s who have been identified as having experienced particularly rapid improvements in mortality rates and are often referred to as the 'golden generations' or 'golden cohorts' including actuarial discussions, the academic literature (e.g. Cairns *et al.*, 2006), official reports (e.g. The Pensions Regulator, 2008; Office for National Statistics, 2008b), the financial press (e.g. Euromoney, 2008), and the informed media.[3]

At present, overall age standardised mortality rates (both sexes combined) are improving at about 2.5% per annum in England and Wales with higher rates of improvement at older than at younger ages (Figure 1),[4] but current overall trends are heavily influenced by patterns at ages where deaths are concentrated (see Appendix A). In 2005, just over 50% of deaths in England and Wales occurred to people born in the period 1925 to 1945, the birth cohorts of the 'golden generations' (Office for National Statistics, 2008c). Current British official mortality projections are based on the assumption that this group will continue to have higher than average rates of mortality improvement in the future so that the differentials in Figure 1 reflect persistent cohort differences (Office of Population Censuses and Surveys,

[1] This may change, but the issue of population ageing will remain important in the long term even though the credit crunch is exacerbating the problems of pensions funding. In 2007 defined benefit (final salary) schemes in Britain had 2.7 million members in the private sector and 5.2 million in the public sector (Office for National Statistics, 2008a), see also Steventon (2008).

[2] 2008-based Eurostat projections available at http://epp.eurostat.ec.europa.eu/portal/page/portal/statistics/search_database and U.S. ones at http://www.census.gov/population/www/projections/2008projections.html

[3] See, for example, http://news.bbc.co.uk/1/hi/business/4295362.stm

[4] These are based on author's calculations using the WHO European standard (Doll & Cook, 1967) with unpublished Government Actuary's Department data.

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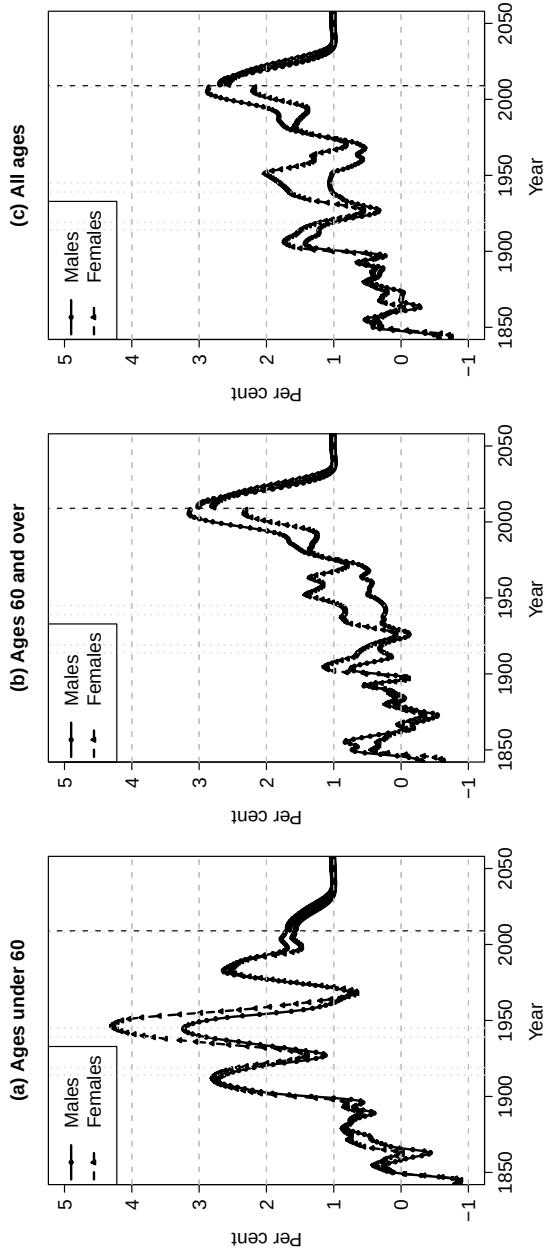

Figure 1. Annual period rates of mortality improvement by age and sex, England and Wales, 1841-2006

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1995; Office for National Statistics, 2008b; Willets, 1999, 2004), similar assumptions have come to form the basis of much British actuarial mortality forecasting as well, but their 'golden cohorts' are located slightly earlier reflecting the generally higher financial and social-economic status of those who are of particular interest to the financial sector[5] (Continuous Mortality Investigation, 2007). Thus the high levels of mortality improvement observed in recent years are assumed to be a transient phenomenon largely determined by those born around the period 1925-45 and that as these cohorts are replaced in the main mortality age groups by less favoured cohorts, rates of mortality improvement will fall in future. The British official projections assume that mortality improvement will decline by more than 60% to a value of 1% per annum in about 25 years' time after the 'golden generations' effect has worked itself out of the system. The half-century of accelerating mortality rate improvement for males is projected to be reversed at a rate not experienced since records started in the 1840s.

The analysis of cohort effects on mortality has been a long-standing area of interest from the 1920s (Derrick, 1927; Kermack *et al*., 1934), which has recently become renewed largely independently in a number of fields, such as demography (Preston & Wang, 2006), biology (Finch & Crimmins, 2004), actuarial studies (Willets *et al*., 2004) and epidemiology (Kuh & Ben-Schlomo, 1997), although developments in other disciplines may be unrecognised: for example, Willets (Willets *et al*., 2004, p879) points out that life course analysis, a major area of epidemiological research, is largely unfamiliar to actuaries.

The paper is organised as follows. The concept of 'cohort effects' is discussed; limitations of national-level cohort data considered; and methods for identifying such effects are reviewed. Particular attention is given to the analysis of populations with identified cohort effects; those of Britain and Sweden in the later part of the nineteenth century and early twentieth century (Kermack *et al*., 1934) as well as the contemporary British population. The likely magnitude of such effects and potential drivers is discussed using a stylised model to assess the extent to which members of the 'golden generations' are especially privileged. Possible determinants are considered and the role of the 'post-golden generations' for future mortality trends is considered.

2. Defining Cohort Effects

The term 'cohort effect' is widely used but rarely defined. The *Handbook of the Life Course* (Alwyn & McCammon, 2006, p26) and Susser (2001) do

[5] Within the financial sector, groups with advantaged survivorship are sometimes referred to somewhat less positively as the 'toxic tail' (Blake & Pickles, 2008; Blake *et al*., 2007).

give similar definitions from sociological and epidemiological backgrounds, respectively, in which a cohort effect is defined as a causal factor acting early in life that leads to later identifiable consequences (a more extended discussion is in Murphy, 2010a). Many recent actuarial publications are based on the influential work of Willets (1999, p5), who defined a 'cohort effect' as a "wave of rapid improvements, rippling upwards through mortality rates in the United Kingdom" and also as "a descriptive term for the observed trend [of the 'golden generations'], and does not have a specific statistical meaning", or indeed a causal one, since as Hobcraft *et al.* (1982, p5) observe: "ages, periods, and cohorts do not have either direct or indirect effects on demographic or social phenomena".

In the first definition above, cohort effects exist only if causal mechanisms that drive the observed regularities can be established, whereas in the second case, cohort effects are usually inferred by examination of tables of age-specific mortality trend data. The latter approach was adopted by the first study identifying the 'golden generations', the Official Report on the British 1992-based population projections: "a ... higher than average rate of improvement is a special feature of generations born between 1925 and 1945 (which more detailed charts show to be centred on the generation born in 1931). It is not yet understood precisely why the members of the generation born about 1931 have been enjoying so much lower death rates throughout adult life than the preceding generation ..." (Office of Population Censuses and Surveys, 1995, p 10). This statement has stood the test of time well and remains unchanged in official publications until the present day.

A term such as 'cohort effect' may be unhelpful and potentially misleading, if it is assumed that it is a causal effect that may be identifiable from analysis of a table of mortality rates cross classified by any two of age, year of occurrence, or year of birth. This may be illustrated by an artificial example. One society introduces vaccination of infants, which leads to a reduction in subsequent mortality for all those vaccinated from that age forward. The second introduces an income support scheme that reduces the mortality of recipients in the given year, which was originally targeted on young children, but, eligibility was extended over time up the age range by one year in each successive calendar year. The observed mortality patterns are identical (and would often be interpreted as a cohort effect), but the future implications are very different: the vaccinated population will continue to benefit, whereas the income policy population's prospects depend on the way in which the policy is implemented in future periods. Examination of the mortality surface would give no indication as to whether the driving process was a cohort or a period one. The most parsimonious explanation might suggest one relating to early life conditions, but just analysing the mortality surface, however apparently sophisticated the approach, cannot establish the mechanisms involved, but only indicate areas where further investigation might take place. As Newman (2001, p216) states: "in order to separate out age, period,

and cohort effects ... is necessary to incorporate additional ... substantive knowledge, and therefore the allocation to age, period or cohort effects is not a statistical issue''. In practice, of course, such clear-cut alternatives as the cases above are rare, but situations in which mortality improves differentially by age, starting first at young ages and moving progressively to older ages over time, would appear to look like a cohort phenomenon, whereas the driver may be a period factor.

Although apparently very different, neither of the artificial examples above requires that cohort effects act similarly over the whole age range. There are plausible causal mechanisms and empirical evidence that suggest that events around the time of birth (both pre- and post-natal) lead to different chances of cardiovascular disease starting at late middle ages (Barker, 1994; Barker *et al*., 1989), and that inflammation in early childhood may influence mortality at even higher ages (Finch & Crimmins, 2004). Other causal factors occurring later in life, such as the well-established effects of smoking on mortality, especially lung cancer, are largely manifested at older ages (Charlton & Murphy, 1997).

It is possible to have cohort effects that reverse from some ages; for example, early adverse circumstances and high mortality at young ages might lead to proportionately more deaths among the more frail members of the cohort but the surviving and consequently, on average, fitter members of the cohort experience lower mortality at older ages (assuming that frailty is constant for an individual over the lifetime, e.g. Vaupel *et al*., 1979; Vaupel & Yashin, 1985). Some theories of ageing argue that some alleles might prioritise health at younger ages in order to maximise reproductive success at the expense of long-term survival ('antagonistic pleiotropy', Williams, 1957) that could lead to crossovers of different genotypes such as, for example, by diverting resources from immune function to nutritional status. The existence of such 'crossover' effects has been debated for many years especially in the context of racial differences in mortality in the U.S. where reported mortality rates for Blacks fall below those for Whites at older ages. While it had been argued that this was due to data errors (Coale & Kisker, 1986), the evidence is accumulating the crossover is real. In the 2005 U.S. life tables, mortality rates of Blacks are at least 25% higher than those of Whites at all ages below 68, but lower than those of Whites beyond age 86 (Arias *et al*., 2010).

3. Identifying Cohorts

A cohort usually refers to a group with a common initial characteristic (such as those born in Britain in the same year) followed through time. A problem is that the group of people dying in a country such as England and Wales at a given age and year of birth is not a well-defined cohort, since it includes people who were not born in England and Wales but migrated later,

and excludes native-born people who emigrate and subsequently die elsewhere. The magnitude of migration in Britain has been substantial in the twentieth century. In 2008, there were an estimated 6.7 million overseas born people in the United Kingdom (Office for National Statistics, 2009b) and over the twentieth century, Britain was a net exporter rather than an importer of people so that the number of British-born people living outside the country is also substantial (Coleman & Salt, 1992; Murphy 2009) but with a very different age structure to that of immigrants to Britain. Unless the mortality rates of both immigrants and emigrants are similar to the resident population, the use of such data as an indicator of cohort mortality change, which will often be concerned with the difference of a few percentage points, is potentially misleading. For example, the immigrant population in the U.S. grew from 9.6 million in 1970 to 32.5 million in 2002 and immigrants have very different mortality patterns from their native-born counterparts. Black immigrant men had 9.4 years longer life expectancy than black U.S.-born men and the corresponding figure was 4.3 years for Hispanics in the period 1986-94 (Singh and Miller, 2004), with the latter figure having a substantial impact on overall cohort Hispanic mortality given the large numbers of non-native born Hispanics.

This observation is particularly pertinent to the most recent period when migration has become much more important. In Britain, for example, considerable attention has been drawn to the relatively poor mortality performance of young adults of cohorts born in the 1950s and 1960s following a substantial deterioration in the rate of mortality improvement for young adults in the 1980s, which has been used to highlight the apparently privileged position of the 'golden generations'. A major reason for this was increases in mortality from HIV/AIDS, external causes and substance abuse (Aylin *et al*., 1999). In 1994-6 there were 35,324 deaths among those aged 20-39 in England and Wales, but if the 1986-8 rates had applied in 1994-6, there would have been 83 fewer deaths than were actually observed, indicating a slight deterioration in mortality over the period. However, this was largely due to an increase of 925 infectious disease deaths, to which HIV infection was the major contributor (Aylin *et al*., 1999, p38). Inclusion of recent immigrants with high rates of mortality from such causes may lead to misinterpretation of cohort patterns. Information to calculate values for U.K.-born cohorts in this period is not published, but despite comprising less than 1% of the total U.K. population, the number of Black Africans (not all of whom are overseas-born) currently diagnosed with HIV is similar to that of the white population, who comprise more than 90% of the population.[6] The absence of fitter-than-average emigrants from the resident British population may also be relevant: Australian age standardised cardiovascular

[6] See http://www.avert.org/uk-race-age-gender.htm

disease mortality was 23% lower than that of the U.K. in 2002,[7] but even so British Isles' migrants to Australia aged 45 to 64 in 1998-2002 had circulatory disease and diabetes mortality 30% lower than native-born Australians (Gray *et al.*, 2007), suggesting a substantially lower rate than those remaining in the U.K. Thus the distinction between real and pseudo-cohorts should not be ignored when interpreting mortality trends and differentials.

4. Identifying Cohort Effects

The validity of hypothesised mechanisms may be tested empirically in a number of 'natural experiments'. Experiences of very adverse conditions at a different stage of the life course, such as infancy or pre-natally, that might be expected to lead to later poor mortality outcomes have been investigated. Case studies include the severe 1869 famine in Finland which was found later to have no discernable effect for those born around that time (Kannisto *et al.*, 1997). The effects of extreme hardship suffered by those born around the time of the 1941-44 siege of Leningrad (where estimated average daily rations were around 300 calories, containing virtually no protein) and the Dutch Hunger Winter of 1944 have been extensively studied for subsequent excess health risks (Van der Zee, 1998; Lumey *et al.*, 2007; Stein *et al.*, 1975) but Lumey & Van Poppel (1994, p245) conclude that even for such extreme experiences "the long-term effects are not easily detected". Some disadvantages have been identified for those born around the time of the 1918-19 influenza pandemic in the U.S. (Almond, 2006; Mazumder *et al.*, 2009). While inconsistent results have been found, substantial nutritional deficits, pre-natally, post-natally, in childhood and in adolescence, appear to be associated with no excess or only relatively small additional mortality in later life, which would appear to put a limit on the expected magnitude of such effects in cases where nutritional changes were much smaller over time such as in Britain over the twentieth century.

A second way in which cohort patterns have been identified is by fitting statistical models that incorporate some or all of age, period, and cohort variables. The appropriate way to model such processes remains an active research area (Carstensen, 2007; Yang *et al.*, 2004) with a substantial statistical, sociological, demographic and epidemiological literature on this topic. The model is usually specified as

$$\ln(\mu_{a,c,t}) = \alpha_a + \beta_c + \gamma_t + \varepsilon_{act} \quad (1)$$

[7] World Health Organisation WHOSIS database http://apps.who.int/whosis/data/Search.jsp accessed 16th November 2009.

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where

$\mu_{a,c,t}$ is the mortality rate for age a in year t for cohort c;
α_a, β_c and γ_t are coefficients, and,
ε_{act} is a residual term.

or, alternatively,

$$\ln(\mu_{a,c,t}) = f(a) + g(c) + h(t) + \varepsilon_{act} \tag{2}$$

where

$f(a)$, $g(c)$ and $h(t)$ are functions of age, cohort and period, respectively.

The problem with such models is the identification problem since

$$t = c + a \tag{3}$$

which means that there is no unique solution to equation (1) without some further assumption and/or restriction. In particular, if there is a common linear change ('drift') in mortality, this cannot logically be uniquely attributed to either period or cohort axes as Preston & Wang (2006, p638) note (see also Murphy, 2010a).[8] In practice, it may be difficult to establish if the data are free of errors and conform to the model assumptions, such as a fixed age pattern. For example, Richards *et al.* (2007, p498) commented on their model results for England and Wales: "One point of particular note is the result for males in England and Wales [where period effects dominate], as this flatly contradicts the result in Richards *et al.* (2006) ... [which] concluded that cohort effects dominated period effects". Possible explanations for such different interpretations are the use of slightly different data sources and/or upper age limits, but such findings emphasise the frequently found difficulties in drawing definitive conclusions from such approaches.

A third way for identifying cohort patterns is use of graphical methods as undertaken by Kermack *et al.* (1934) (reproduced in Davey Smith & Kuh, 2001 and Murphy, 2010b), Office of Population Censuses and Surveys (1995), Willets (1999, 2004), Richards *et al.* (2006), etc. These range from straightforward hand-drawn lines on a mortality table to contour or heat maps of changing mortality rates using either simple comparisons or various model-based smoothing approaches (see Appendix B). As Preston & Wang (2006, p638) note, graphical methods appear to be considerably more

[8] Another general issue is that model-fitting usually uses data in the form of a rectangular mortality surface of n age groups by m time periods; in such cases the number of cohorts included is $n + m - 1$, but two of the cohorts include only a single observation. It would seem more appropriate to have symmetric data structures for the two dimensions of period and cohort if judgements are to be made about their relative contribution to explanation, but this rarely if ever is done.

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successful in identifying cohort influences than statistical models. Both approaches have advantages and disadvantages — statistical models can produce goodness of fit indicators but tend to be inflexible so are not necessarily particularly useful for exploratory analysis. There is the problem of indeterminacy in attributing change unambiguously to the period or cohort dimension. The majority of studies concerned with identifying cohort mortality patterns are confined to certain age groups only, usually older ages since many of these studies involve various forms of cancer. Such models usually assume that the effect is manifested as a fixed relative risk on mortality at each age in the age range selected and therefore, for example, would not be able to identity 'crossover' effects. Some of the graphical methods used make few assumptions about the nature of the underlying process, whereas others may attempt to remove other sources of variability such as period changes in order to maximise the likelihood that cohort patterns will become visible. However, given the lack of an agreed way of presenting such data, the fact that the analyst can both select the method of presentation and the section of the mortality surface to be highlighted, the scope for subjectivity in interpretation of results is substantial (see Appendix C).

5. Historical Discussion

The debate about the relative importance of period and cohort effects on mortality has been a longstanding one. Derrick (1927, p144) presented age-specific mortality rates in the period 1841 to 1925 by year of birth and concluded that "the parallelism is remarkable" and that "nearly the whole of the temporal change is due to an entirely independent generation influence, each generation being endowed with a vitality peculiarly its own". Others were less convinced (see the discussion in Derrick, 1927) including some members of the Statistics Committee of the Royal Commission on Population, of which Victor Derrick was a member (Kyd, 1953). Both Derrick (1927) and Kermack *et al.* (1934) argued that cohort approaches would provide superior forecasts of future mortality than alternative approaches. However, such predictions turned out to be poor (Kuh & Davey Smith, 1993) and cohort approaches fell out of favour. For the next half-century, attention was concentrated almost entirely on period ones, to the extent that Hobcraft *et al.* (1982, p.12) could conclude that: "most population specialists appear to believe that cohort mortality and effects are sufficiently minor that they need not be incorporated into models of mortality relations".

Kermack *et al.* (1934) presented data on Scotland and Sweden as well as for England and Wales, although attention has been concentrated mainly on the results for England and Wales, and the findings for the other countries

have been largely ignored. Figures 2 to 4 present values using Kermack *et al.*'s approach with mortality indexed to that of period 1841-50 (1855-64 for Scotland since data are only available from 1855) for the three countries by sex updated to 2006 or 2007 with data obtained from the Human Mortality Database (2009). Others who have updated these tables include Harris (2001) and Davey Smith & Lynch (2004) but both have used banded data. The data presented here use the penalised spline method discussed in Appendix B. While by no means perfect, the data for both men and women up to about age 75 for cohorts born in both England and Wales and in Scotland in the period 1841 to 1910 show a tendency for isoquants to be at a 45 degree angle until 1930 for ages 20 to 60 years for males, and somewhat later for women. Patterns at other ages and periods are less convincing, although interpretation is inevitably subjective. However, Kermack *et al.* (1934) acknowledge that their cohort model was not particularly successful in explaining mortality trends for Sweden, the only non-British country they investigated up to 1926, and the continuing lack of any cohort-like patterns is clear in Figure 4.

There are six potential reservations about Kermack *et al.*'s (1934) method of presentation and the extent to which definitive conclusions may be drawn concerning the pre-eminence of cohort patterns, which are discussed in more detail in Murphy (2010b). In particular, the results of Table 1 from Kermack *et al.* (1934), which are among the most widely-cited examples of a cohort effect, can arise from a non-cohort mechanism as in the earlier artificial example, as they acknowledge.[9] The reasons for the nineteenth century mortality decline remain a matter of debate, with the main arguments centring about the relative contribution of factors such as improving nutrition (McKeown, 1976, 1988) and public health measures (e.g. Szreter, 1988), but both are considered mainly as period processes. Preston (1996) argues that acceptance of the germ theory of disease had a substantial impact on infant mortality improvements in the late nineteenth century. An alternative explanation for the later nineteenth century and early twentieth century mortality patterns of relatively faster rates of improvement in a given year at young, but not at the youngest ages (Woods, 2000; Harris, 2001) is Epidemiological Transition Theory (Omran, 1971, 1998), whereby mortality initially starts to improve by reducing communicable diseases and only later chronic diseases fall substantially. Since communicable diseases formed a

[9] Kermack *et al.* (1934, p700) considered if "the consecutive improvements which have taken place in succeeding age-groups are the result of a series of independent sets of conditions, or legislative acts, and that the apparent regularity is largely fortuitous. It might for instance be suggested that early industrial legislation was directed towards the welfare of children, and that at a later date general industrial and social conditions improved, and that older people were last in being affected by industrial and housing changes ...", but concluded that "it would be somewhat surprising if the quantitative regularity just pointed out should emerge.".

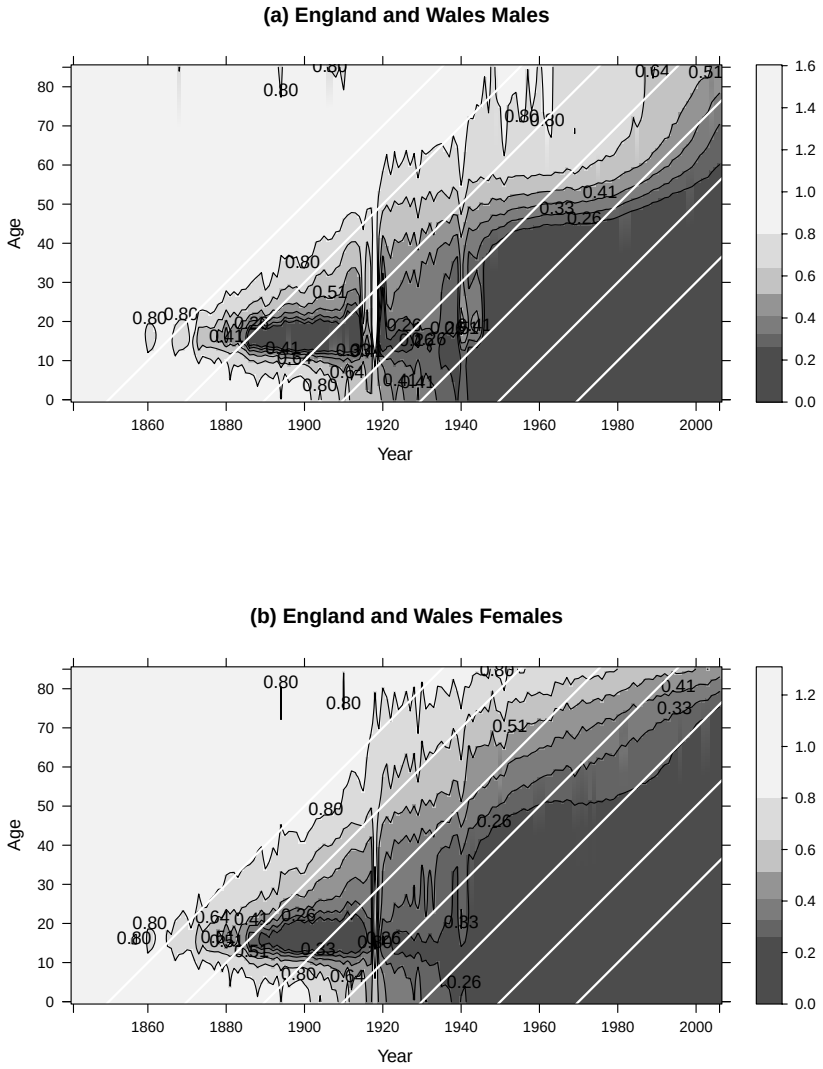

Figure 2. Annual period rates of mortality indexed to mid nineteenth century values for England and Wales, by sex 1841-2006

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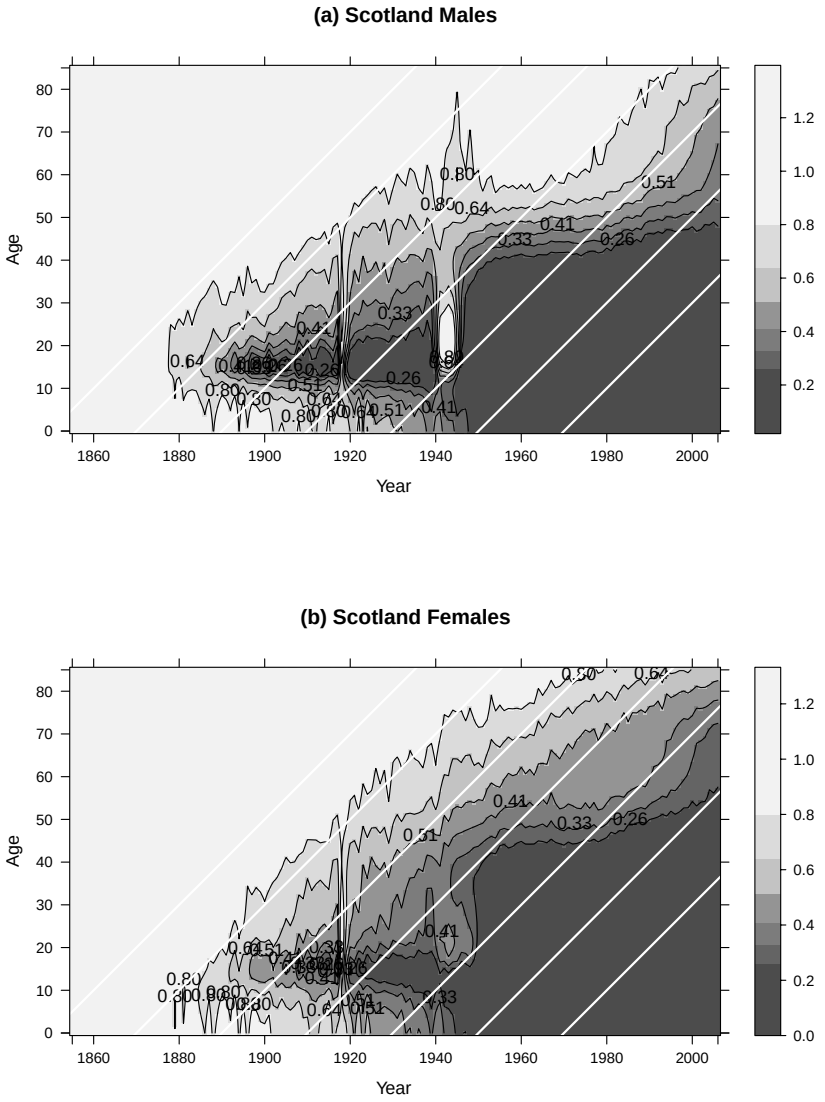

Figure 3. Annual period rates of mortality indexed to mid nineteenth century values for Scotland, by sex 1855-2006

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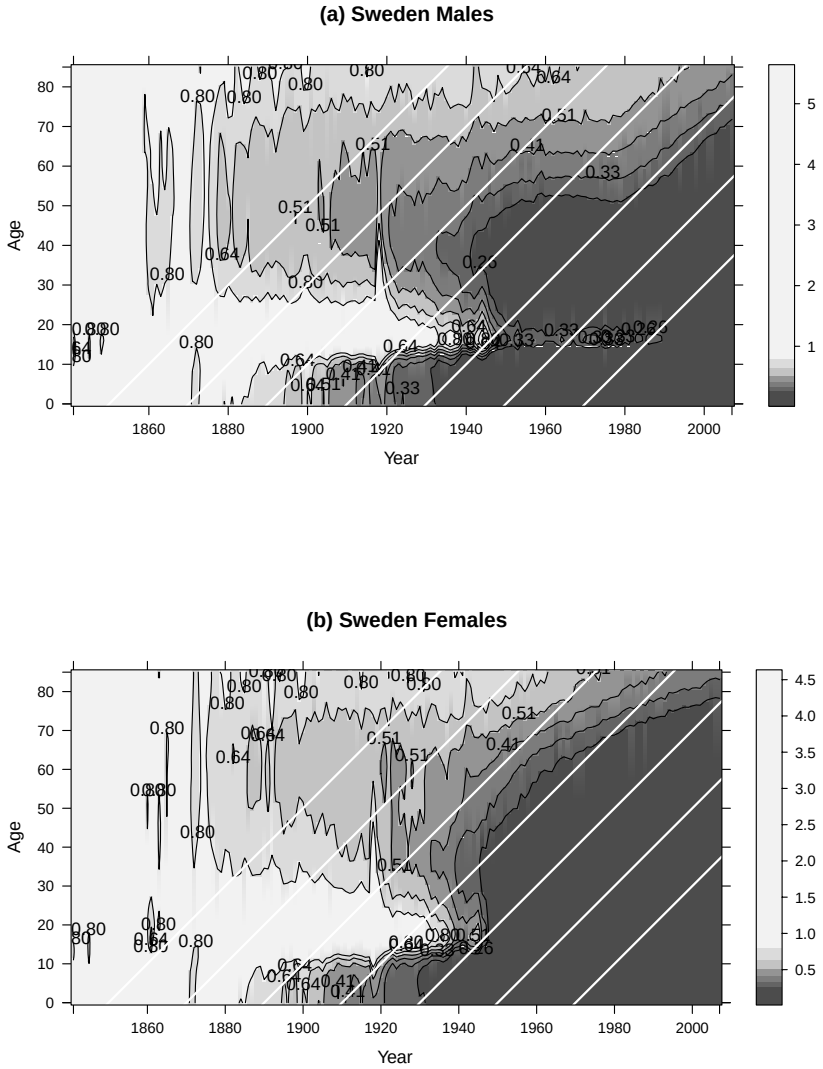

Figure 4. Annual period rates of mortality indexed to mid nineteenth century values for Sweden, by sex 1841-2007

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higher proportion of deaths among young people (Preston *et al.*, 1972; Preston, 1976), rates of overall mortality improvement are initially greater for younger than for older people.

6. The Role of Infant and Child Mortality

The fact that infant mortality shows very little improvement over the nineteenth century in England and Wales, and Scotland (Figures 2 and 3), whereas toddler mortality starts improving many decades earlier requires specific explanations, perhaps related to patterns of urbanisation in Britain and changing disease virulence (e.g. Woods, 2000). Nevertheless, the approach of Kermack *et al.* (1934) has been generally endorsed in recent times, in part because they anticipate much later work concerned with life course effects, suggesting that pre-natal and infant experiences are likely to have consequences later in life. They argued that infant mortality improved only from the start of the twentieth century after their mothers' health had already improved. They identified cohorts born about 1870 as the first with substantially improved mortality (Kermack *et al.*, 1934, p701). However, the lack of empirical justification for the crucial role of early childhood conditions suggests that it is a *post hoc* hypothesis. Moreover, this hypothesis fails to explain why Swedish data show a completely reversed pattern: both infant and toddler mortality start to improve from the 1860s and show sustained improvement from the 1880s, but mortality of those in their mid-20s shows little improvement until after 1920.

7. Recent Patterns and Methods

The visual presentation of Figures 2 to 4 has limitations (Murphy, 2010b). Recent graphical approaches for identifying cohort effects in contemporary Britain have tended to use estimated rates of age-specific change in mortality, especially for identifying the 'golden generations', including Office of Population Censuses and Surveys (1995), Willets (2004), Willets *et al.* (2004) and Richards *et al.* (2006). Figures 5 to 7 show the full set of values for England and Wales, Scotland and Sweden for all ages and time periods since the mid-nineteenth century.[10] These figures generally

[10] The two most substantial period mortality effects, the very sharp fluctuations in the 'outlier' years 1914-20 (which included the 1919 influenza pandemic), and 1939-45 are removed since the reasons for their influence are well-defined in Britain. In Sweden only years 1918-20 are excluded as they were not a combatant nation. See Appendix B for a discussion of the approach used and its rationale. It can be argued that these years should be included, since to exclude the major period factors simply because the underlying cause is well-recognised would tend to downplay the influence of period factors.

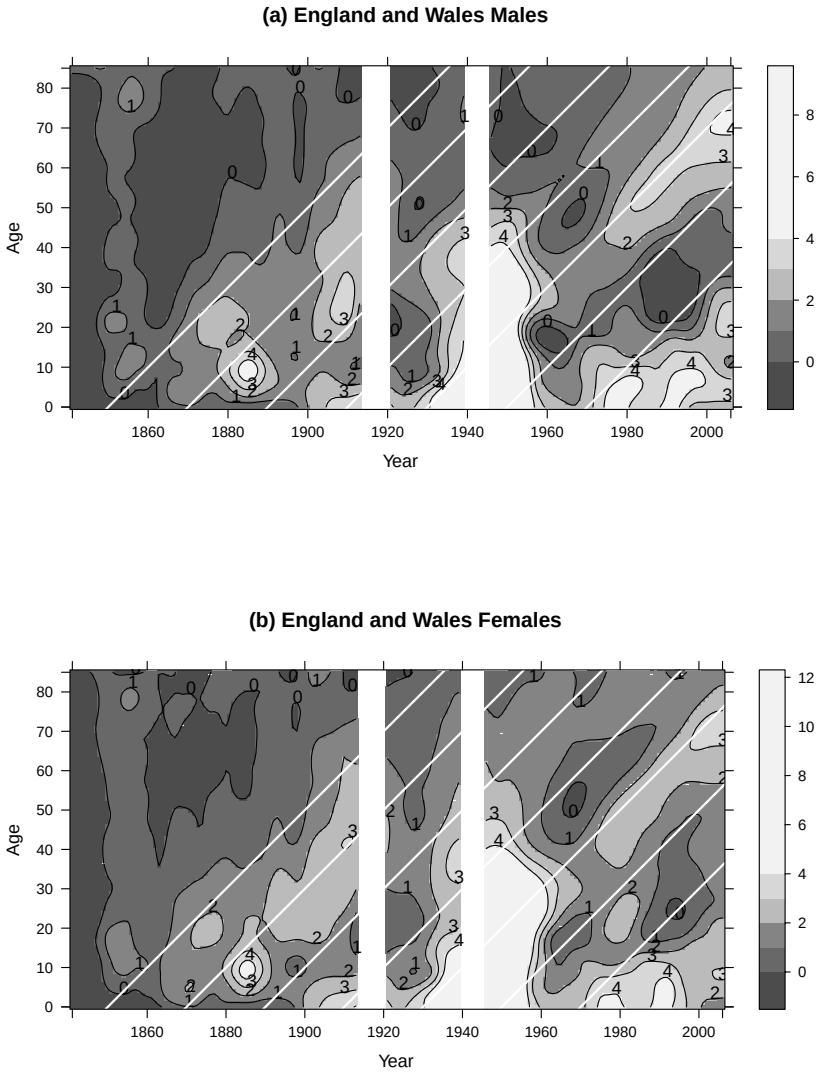

Figure 5. Annual period rates of mortality improvement for England and Wales, by sex 1841-2006

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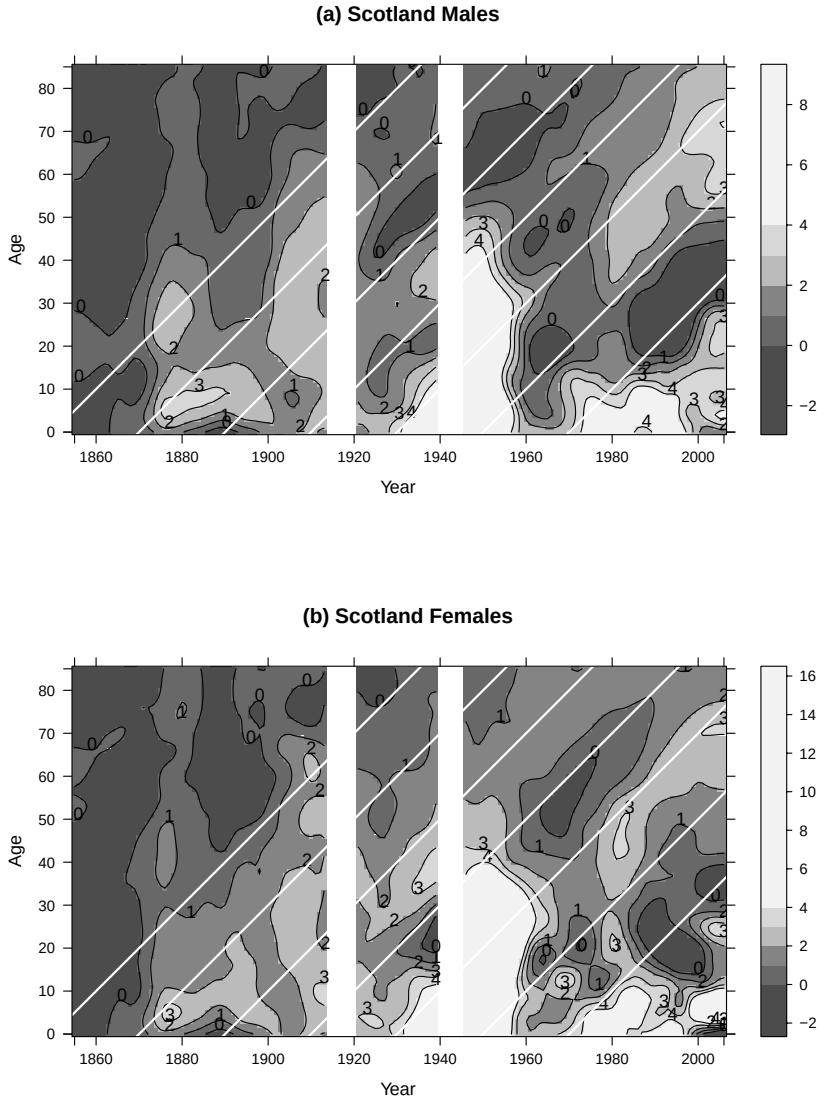

Figure 6. Annual period rates of mortality improvement for Scotland, by sex 1855-2006

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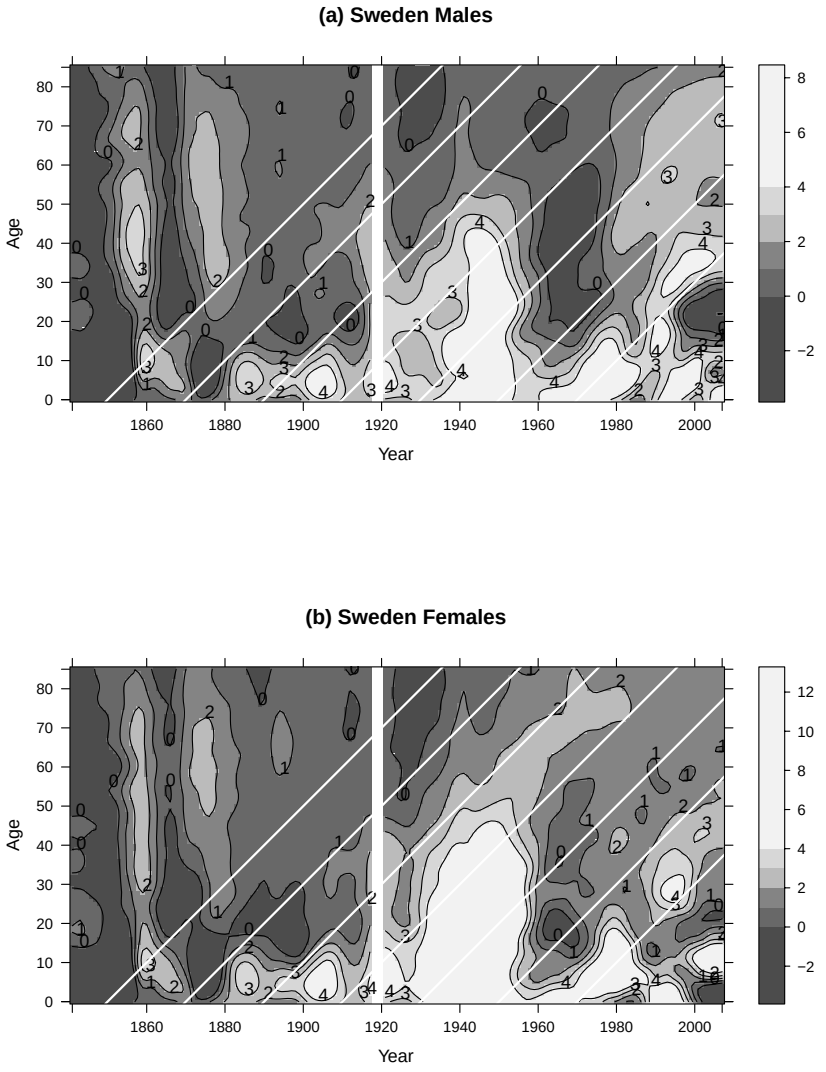

Figure 7. Annual period rates of mortality improvement for Sweden, by sex 1841-2006

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confirm the findings of Figures 2 to 4 such as that mortality first shows signs of improvement from about age 20 years for both sexes from about 1860 in both England and Wales, but that improvements became sustained and widespread only from about 1880 in both England and Wales and Scotland. In contrast, Sweden shows a completely different pattern with mortality improvement occurring largely contemporaneously at all ages, apart from the very marked delay in improvement among young adults noted earlier.

The British 'golden generations' are visible as those with higher rates of mortality improvement than surrounding cohorts for both men and women, with isoquants lying along the 45° diagonal line for those born around 1930 for ages above about 40 years, but this pattern is less clear cut for Scotland and non-existent for Sweden, where, for example, women born about 1960 appear to have relatively high rates of improvement, the reverse of the British pattern. Little of the patterns identified for earlier cohort in Britain in Figures 2 and 3 are visible in Figures 5 and 6, suggesting that conclusions about the existence of cohort patterns arise in part from a particular form of presentation. However, the lower quality of data estimates in the nineteenth century needs to be acknowledged.

8. A Stylised Model for the 'Golden Generations'

Two simplified versions of a 'golden cohorts' model shown in Figure 8 are designed to indicate the orders of magnitude of effects that can be obtained. The first (Model 1) is based on changing trends in a risk factor, which, for concreteness, might be characterised as 'smoking'. In this example, 'smoking' moves from a low level to a high-point to and back to a low level over a period of 40 years; the mortality of both 'non-smokers' and 'smokers' improves by 1% p.a., but smokers have double the mortality of non-smokers at any age (Figures 8(1a) and 8(1b)) — further details are given in Appendix C. In order for the maximum rate of mortality improvement to reach 2% p.a. under this model, the proportion of the cohort who are smokers would have to reach a maximum of 14% (Figure 8(1c)). With this model, the emergence and subsequent disappearance of a risk factor will lead initially to a lower rate of overall mortality improvement but later to a higher rate than the baseline level value that would have been experienced if the risk had not occurred. In particular, the 'golden generations', the group for whom mortality improvement is a maximum, experience higher overall risks than if the mortality had remained at the baseline level.

The alternative scenarios (Model 2) arises from an innovation such as the 'introduction of the Welfare State' (or of vaccination), during which cohort mortality improves, but as above the rates of improvement in the pre- and post-transitional phases are 1% p.a. (Figure 8(2a)). In this case, mortality

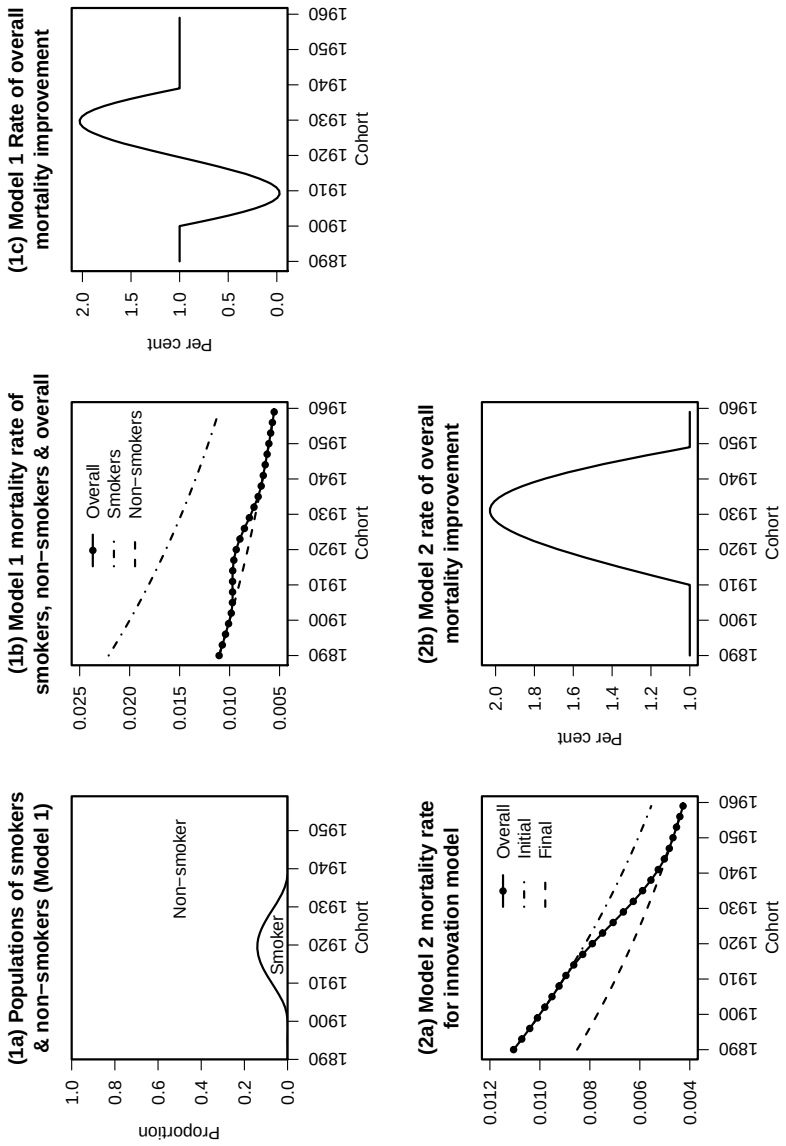

Figure 8. Stylised alternative models for 'golden generations'

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improvement is never below 1% p.a. With the assumptions of Appendix C, a maximum rate of improvement for the 1930 cohort of 2% (1% above baseline) would imply a 23% lower level of mortality in the post-transitional phase than would have been the case if the innovation had not occurred (Figure 8(2b)).

Temporary high rates of mortality improvement based on transient patterns due to factors such as reductions in smoking imply the existence of groups with corresponding levels of below-baseline mortality improvement (Appendix C) and the underlying change will be over-stated, if comparisons are made with these groups. (Note that these changes need not be symmetric and different assumptions about the time over which they occur will produce different results, but the values shown are designed to indicate plausible bounds.) A second point is that for both simplified models, the 'golden generations' are located at the point of inflexion of the time trend of prevalence of the risk factor ('smoking') or the point at which the proportions benefitting is increasing maximally ("welfare state"). In both cases, this is where the values of the second derivative of the aggregate mortality rate is zero and the third derivative is positive. The 'golden generations' have intermediate mortality levels compared with surrounding cohorts and intermediate exposure to 'smoking' or access to welfare state provision (compared with surrounding cohorts). Reference to the 'point of inflexion generations' might be a more accurate but less punchy description of the phenomenon, but it might be optimistic to hope that it displaces the now so widely used phrase 'golden generations'. Of course, there is no change in the trend in risk over time at the individual level in the 'smoking' case for either smokers or non-smokers, as both groups improve at a constant rate of 1% p.a. Therefore neither a smoker nor a non-smoker gains any advantage from being a member of the 'golden generations' — the changing overall mortality rate is simply due to the changing proportions of smokers and non-smokers in the population. Even in this case, the 'golden generations' can only be considered advantaged in that they are a group in the process of recovering from the below-average performance of earlier cohorts. The widely-used adjective 'golden' may not be the most appropriate in this context.

9. Explanations for Cohort Effects

As exploratory techniques, graphical and statistical modelling methods may suggest the existence of cohort patterns (or indeed period ones, although a review of the literature suggests that this is rarely if ever done, since the overwhelming interest is in establishing cohort effects), but they do not elucidate the underlying causes. In an authoritative consensus view of this area, the British National Statistician (Dunnell, 2008, p19) accepts the existence

of the 'golden generations', but concludes that there are still only a series of explanatory hypotheses that include:

1. Changing smoking patterns between generations
2. Better diet and environmental conditions during and after the Second World War
3. Those born in periods of low fertility facing less competition for resources as they age
4. Benefits from the introduction in the late 1940s of the Welfare State
5. Benefits from medical advances.

Singer & Manton (1998) suggest an additional explanation for these specific cohorts: that improvement in food preparation and packaging in the 1920s and 1930s may have had an influence on later mortality. Therefore, nearly two decades after the phenomenon of the 'golden generations' was identified, no clear-cut causal mechanisms have been established. The initial and most commonly cited reason for the existence of the 'golden generations' is changing smoking patterns (Office of Population Censuses and Surveys, 1995; Willets, 2004). It is therefore surprising how little attention has been given to establishing the validity or otherwise of this hypothesised explanation. At present, there is no definitive evidence for the primacy of the smoking explanation, although work has been undertaken to examine how far smoking can account for observed patterns of sex differentials in mortality in United States by Preston & Wang (2006). In Britain, mortality trends for males and females are very similar (Figures 5 and 6), although levels and trends of smoking and the associated variable of lung cancer are very different for men and women (Willets, 2004; Di Cesare & Murphy, 2009). Alternative cohort influences have been suggested: foetal environment and early life experiences on later cardiovascular mortality (Barker, 1994), and childhood morbidity, especially inflammation, on old age mortality (Finch & Crimmins, 2004, although they argue that environmental factors and medical advances mean that such childhood effects would not be expected to be observed for twentieth century cohorts that are the focus of this paper). Other studies emphasise the role of early patterns of nutrition, as reflected in childhood height (e.g. Floud *et al.*, 1990; Crimmins & Finch, 2006). Since the causal mechanisms involved are unknown, the assumption built into mortality projections that they should continue to act in the same way in years to come is speculative.

However, understanding the experiences of the 'golden generations' may not be the area of highest priority or interest. More attention should be given to those born after the 'golden cohorts' in the period 1945 to 1965 in Britain[11]

[11] The group born in the period 1945-64 is often but incorrectly referred to as 'baby-boomers', which is taken from U.S. fertility patterns. In fact in Britain, there were more births in the quinquennium 1971-75 than in the central period of the nominal British 'baby boom' period.

who will dominate mortality trends in the future, since they appear to have worse mortality than might be expected with little or no mortality improvement when compared with their immediate predecessors to date. This is particularly anomalous since they were the first products of the Welfare State, including the National Health Service, and brought up in a boom economic period with unprecedented family life stability. These post-World War II generations, who start to reach age 65 years only from 2010 onwards, have not yet started to experience especially high mortality rates: indeed, at age 65 years, men and women in Britain today experience far lower mortality rates than they did in their first year of life.

10. Conclusions

The analysis presented here suggests that the evidence for the existence of cohort effects based on an analysis of simple mortality tables using increasingly sophisticated computational techniques has sometimes been over-interpreted, and that there has been a lack of attention to the underlying mechanisms. If the changes are driven by some as yet unidentified series of events that occurred many decades ago, it is unclear whether such effects will continue until the highest ages (e.g., HIV/AIDS-related mortality was a major factor for the apparently poor mortality in the early 1990s for those born around 1960, but deaths from this cause are now negligible for this cohort). In fact, birth cohort has no particular advantage as a classificatory variable, but is simply another variable that provides the option of investigating differences between groups; other and possibly more informative examples of characteristics fixed at birth include sex, ethnic group, duration since previous birth and parental social class at birth.

The recent literature on future British mortality prospects is concerned almost entirely with the 'golden generations'. The fact that they appear to do well compared with preceding ones — people born around the time of the First World War, who were brought up in the inter-war depression years — is unsurprising given that rates of mortality improvement have been generally accelerating for more than a century (Figure 1). From a forecasting viewpoint, at present mortality rates are improving at about 2.5% p.a. and similar rates would be expected to continue in the short term. What does matter in forecasting is what happens when the following generations, the 'post-golden generations', come to dominate mortality trends. It is at this stage that it becomes important for mortality analysts to know what factors are driving mortality change.

In 1955, John Hajnal, who together with Bryan Hopkin, was responsible for producing the first detailed set of modern population projections as part of the work of the Royal Commission on Population and might be regarded as the father of British population forecasting, reflected on this area of work.

He argued that population projections will continue to be in high demand in the future as in the past but will often be fairly wide of the mark. Nevertheless the process of making projections can give insight into the underlying processes, but only if it is approached in the correct way:

> "as little forecasting as possible should be done, and ... if a forecast ... is undertaken, it should involve less computation and more cogitation than has generally been applied. Forecasts should flow from analysis of the past. Anyone who has not bothered with analysis should not forecast. The labor spent in doing elaborate projections on a variety of assumptions by a ready-made technique would often be much better-employed in a study of the past." (Hajnal, 1955, p321)

Such comments appear to be pertinent today, including the issue of the likely future experiences of the 'golden generations' in the light of earlier studies concerned with establishing cohort patterns.

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APPENDIX A

The standardised death rate in year t, SDR_t, is given by the formula

$$SDR_t = \sum_x w_x D_{xt}/P_{xt}$$

or

$$SDR_t = \sum_x w_x m_{xt}$$

where x is age, and D_{xt}, P_{xt} and m_{xt} are the number of deaths, population size and mortality rate, respectively, at age x in year t, and w_x are a set of fixed weights with the age distribution of the standard population.

The rates of change of individual age–specific mortality rates, m_{xt} (equal to M_{xt}/P_{xt}) are given by r_{xt}, and the overall rate of change is

$$\begin{aligned} d(\ln(SDR_t))/dt &= \left(d\left(\sum_x w_x D_{xt}/P_{xt}\right)/dt\right)/SDR_t \\ &= \left(\sum_x w_x r_{xt} D_{xt}/P_{xt}\right)/SDR_t \\ &= \left(\sum_x w_x r_{xt} D_{xt}/p_{xt}\right)/(P_t SDR_t) \end{aligned}$$

where $P_t = \sum_x P_{xt}$ and $p_{xt} = \sum_x P_{xt}/P_t$

$$d(\ln(SDR_t))/dt = \left(\sum_x (1+\lambda_{xt}) r_{xt} D_{xt}\right)/(P_t SDR_t)$$

where $w_x/p_{xt} = (1+\lambda_{xt})$ and $abs(\lambda_{xt}) \ll 1$ since in order for the results to be useful, the weights w_x are chosen to have a fixed distribution broadly similar to that of the populations being analysed. Therefore the overall rate of change is a weighted average in age-specific rates of change, with weights proportional to number of deaths times a factor close to 1, i.e. largely determined by rates of change at ages at which deaths are most common.

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APPENDIX B

A number of methods have been used to identify cohort patterns in mortality data surfaces. Kermack *et al.* (1934) showed ratios of age-specific mortality rates to a reference mortality schedule that they took as the average of the first years available (10 years for England and Wales, and Sweden, but three years for Scotland). The data were time period and age group averages so that, for example, the tabulated 1875 figure for England and Wales relates to the period 1871-80, and the age 40 relates to ages 35-44; in particular age zero refers to infants, so toddler mortality (ages 1-4) is not included. The values are presented as ratios to average mortality rates in the same age-group in the reference period such as 1841-50 for England and Wales. Therefore the results depend on an arbitrary choice determined by data availability, and there are other limitations of this approach (Murphy, 2010b); although this method is rarely used today, the conclusions based on the 1934 approach are widely accepted.

An alternative method adopted by Willets (1999, 2004) to identify cohort patterns does not depend on a reference group. Rates of change were estimated as the slope of a nine-point centred linear regression and he presents values within 95% or 90% of the maximum value in any calendar year between 1954 and 1995 for Japan, and within 70% for England and Wales males between ages 30 and 84 years for calendar years 1965 to 1997. This form of presentation led Willets (2004, p874) to conclude that: "cohort effect for males in England and Wales is not 'wearing off' with time or increased age.'' However, there are a number of limitations of this approach:

1. The method attempts to remove all period variation since comparisons are made only within time periods. This may potentially remove most of the information about mortality trends, especially in the case of countries like Japan where period mortality improvement is substantial in the post-war period, with period life expectancy at birth, e_0, increasing from 50.1 years in 1947 to 79.2 in 2008 for males and from 54.0 to 86.1 for females (Ministry of Health, Labour and Welfare, 2009).
2. The reason for a particular choice of age limits is unclear (and it also varies between applications). It would appear more natural when comparing cohorts to make use of the full age range, but if so, the patterns found may be completely different, such as in the case of Japan, where mortality improvement was highest at the excluded young ages. Thus the results can be determined by the choice of limits.
3. The reason for the choice of substantially different cut-off values between 95% and 70% of the maximum is not discussed. In particular, if variability in a given year is low, a relatively small deviation will be identified as a high improvement value, whereas in another year a larger absolute value may not be.

More recently, the most common way of identifying patterns in mortality surfaces is by showing contour maps of estimated rates of age-specific mortality change. This method does not suffer from the need to choose an arbitrary reference index population and it treats period and cohort largely symmetrically. The precise method of estimating rates of change varies: the Office of Population Censuses and Surveys (1995) shows the ratio of 5-year age-specific rates five years apart, whereas Willets (2004) presented the slope of a nine point regression of the logarithm of mortality rates to identify ages and periods/cohorts with particularly high relative rates of improvement in England and Wales, and MacMinn & Weber (2009) use 5- and 3-point regression slopes.

The reasons for such choices are not explicit, but no optimality criteria are applied, see e.g. Kenny & Durbin (1982). Since the main purpose is to identify the rate of change (i.e. the first derivative of the logarithm of the time series of the age-specific series), it is more appropriate to use a method that estimates the first derivative directly. There are a number of alternative methods for estimating trends that will also estimate values at all points of the time series and will track non-linear trends more accurately, such a locally fitted regressions and Kalman filters, which tend to provide better estimates than linear filters (Bianchi *et al.*, 1999). This paper uses analytic first derivatives obtained from fitting a smoothing spline, $s(x)$ to each single year of age data, x_i, where i denotes year. The formula for such a model is

$$\sum_i (y_i - s(x_i))^2 + \lambda \int_x dz \left(\frac{d^2 s}{dz^2}\right)^2$$

where λ is the smoothing parameter (in the R function smooth.spline, the spar smoothing parameter is a monotonic transformation of λ, R Development Core Team, 2009). Such a method allows the user to estimate the derivative by using some optimality criterion, such as generalised cross-validation where the choice of smoothing is explicitly decided in terms of a well established method. Unlike linear filters, such methods track non-linear curves and provide estimates for all points of the time series (if fitted to $2n + 1$ values, estimates are not available for the first and last n points with a centred filter and a missing value would mean that values in the interval from n years before to n years later would be missing). While results from linears filters and spline-based models are generally similar, the greater coverage, flexibility and optimality properties of model-based methods make them preferable. Another advantage of such a method is that it can include gaps in the data series, such as missing years around World War I, including the 1918-19 influenza pandemic and the Second World War which have been excluded as extreme values from the fitting process (the smoothed spline model estimates values for missing sections of

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the time series). If these years were not included on the grounds that they would distort the underlying trend, with a 9-point moving average, information would be available only for years 1925 to 1934 in the whole period between 1909 and 1950.

APPENDIX C

The population is assumed to comprise two groups. One is at low risk ('lifetime non-smokers') and the other is at high risk ('lifetime smokers'). In the case shown here, mortality for both groups declines exponentially at 1% p.a., and so the annual rates of mortality change are constant for both groups. However, the level is twice as large for smokers as non-smokers in each year. This is obviously over-simplified since it does not take account of cessation of smoking (but it could be reformulated as equivalent numbers of lifetime smokers), but it is made to illustrate the care needed in applying conclusions from aggregate-level data to individual cases.

The model is as follows:

$$p(s, x) = k[1 - \cos((x - 1900)\pi/20)]/2 \quad 1900 \leq x \leq 1940 = 0 \text{ otherwise}$$

where

$p(s, x)$ is the proportion of smokers for cohort born in year x, i.e. it rises from zero to a maximum of k and then returns to zero.

The proportion of non-smokers in cohort x is

$$p(n, x) = 1 - p(s, x).$$

The mortality rates for non-smokers and smokers, $\mu(n, x)$ and $\mu(s, x)$, respectively, are given by the formulae

$$\mu(n, x) = 0.01 \exp(-0.01(x - 1900))$$
$$\mu(s, x) = 0.02 \exp(-0.01(x - 1900))$$

so the overall mortality rate is

$$\mu(o, x) = \mu(n, x)(1 + p(s, x))$$

and the rate of overall mortality improvement is

$$r(o, x) = 0.01 - (k\pi/40) \sin((x - 1900)\pi/20)/(1 + p(s, x)) \quad 1900 \leq x \leq 1940$$
$$= 0.01 \text{ otherwise.}$$

The maximum rate of improvement occurs around cohort 1930, with approximate value

$$0.01 + (k\pi/40)/(1 + k/2).$$

If this is set at 1% over the baseline value, the value of k is 0.14, i.e. the

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maximum number of smokers would be about 14% for the 1920 cohort, and the 'golden generations' would be centred about a prevalence level of 7%.

More generally, consider a twice differentiable function $f(t)$ with $f(t) = 0$ for $t \leq t_1$ and $t \geq t_2$; and $f(t) \geq 0$ for $t_1 \leq t \leq t_2$, but otherwise unspecified. If the absolute maximum value in the interval $t_1 \leq t \leq t_2$ occurs at $t_{\max}$ then the sum of values of the first derivative between t_1 and $t_{\max}$ is $f(t_{\max})$, and between $t_{\max}$ and t_2 is $-f(t_{\max})$. The mean of the first derivative between t_1 and $t_{\max}$ is $f(t_{\max})/(t_{\max} - t_1)$ and between $t_{\max}$ and t_2 is $-f(t_{\max})/(t_2 - t_{\max})$. The minimum rate of change occurs at the absolute minimum of $f'(t)$, when $f''(t) = 0$ and $f'''(t) > 0$; if $f(t)$ has a single maximum, the minimum of $f'(t)$ must occur between $t_{\max}$ and t_2.

With the alternative model ("introduction of welfare state around 1950"), mortality declines exponentially at 1% p.a., before and after the event, but in the transitional period it improves faster as it moves towards the new level for the transitional cohorts born between 1910 and 1950 (the assumption is that the oldest cohorts benefit only at older ages so their overall lifetime benefit is small, but young cohorts increasingly obtain cumulative benefits so that 1950 and later cohorts fully benefit).

The model assumes that mortality changes smoothly over a 40-year period as shown in Figure 8(2b) where the final mortality level is lower than it would have been without the innovation as follows:

$$\nu(x) = 0.01 \exp(-0.01(x - 1900)) \quad x < 1910$$

$$\nu(x) = 0.01(1 - \kappa) \exp(-0.01(x - 1900)) \quad x > 1950$$

$$\nu(x) = 0.01 \exp(-0.01(x - 1900))\{1 - (\kappa/2)[1 - \cos((x - 1910)\pi/40)]\} \quad 1910 \leq x \leq 1950$$

where

$\nu(x)$ is the force of mortality for cohort x, and the rate of overall mortality improvement is

$$r(x) = 0.01 + (\kappa\pi/80) \sin((x - 1900)\pi/40)/\{1 - (\kappa/2)[1 - \cos((x - 1910)\pi/40)]\} \quad 1910 \leq x \leq 1950$$

$$= 0.01 \text{ otherwise.}$$

The maximum rate of improvement over the baseline value occurs for cohorts born around 1930, with approximate value $0.01 - (\kappa\pi/80)/(1 - (\kappa/2))$.

As before, if this is set at 2%, the value of κ is 0.23, i.e. a final mortality level is about 23% lower than it would have been, leads to a maximum rate of mortality improvement of 2% p.a. with the model assumptions above.

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