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Reexamining the Dominance of Birth Cohort Effects on Mortality

MICHAEL MURPHY

COHORT PATTERNS—often referred to as cohort "effects"—are assumed to exert an important, even predominant, influence on mortality. The analysis of cohort experience has been of long-standing interest, especially since the 1920s.[1] It has recently received renewed attention in a number of fields: in biology, with ways in which early-life circumstances may affect later health outcomes;[2] in epidemiology, with life-course approaches;[3] and in actuarial studies, for forecasting mortality.[4] Cohort effects on mortality remain a continuing interest among demographers.[5]

Three sets of birth cohorts have been identified as having particularly clear-cut cohort patterns: those of Britain in the late nineteenth and early twentieth century (Kermack, McKendrick, and McKinlay 1934a); those of Japan in the early twentieth century (Willets 2004); and cohorts born in Britain in the 1930s, often referred to as the "golden generations" because they have shown high relative rates of mortality improvement for many years (Richards 2008). The three key articles just cited discussing these cases were published in medical, actuarial, and statistical journals, respectively, with conclusions based on a variety of methods: descriptive, graphical, and formal statistical model-fitting. This article gives particular attention to the identification of cohort effects and considers competing explanations for the patterns identified. The article ends with recommendations for further work.

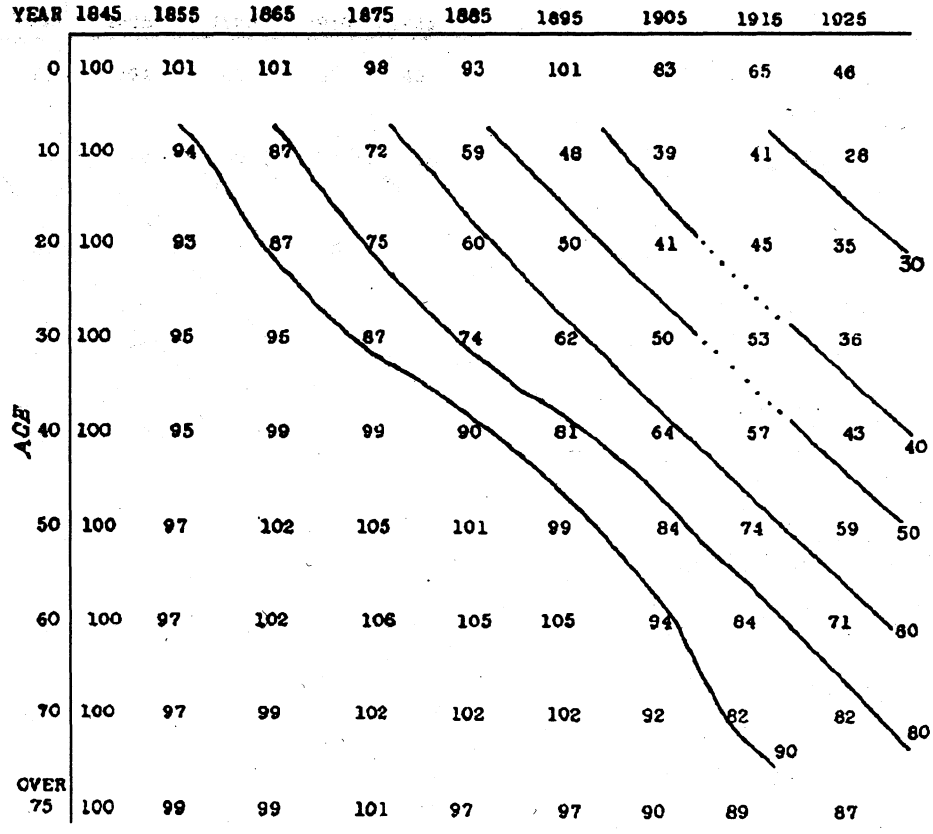
The analysis by Kermack, McKendrick, and McKinlay

The debate about the relative importance of period and cohort effects on mortality has been long-standing. Kermack, McKendrick, and McKinlay (1934a;

referred to as KMM hereafter)[6] presented a table with hand-drawn contours of age-specific mortality rates for England and Wales indexed on the average values for the period 1841–50.[7] They concluded (p. 698): "It will be seen that these contours show a remarkable tendency to follow the diagonals...the figures along a diagonal represent the rates experienced by a particular group (or generation) of individuals all born in a particular year-period.... The general conclusion...would seem to be that...relative mortality is approximately constant for each generation at all periods of life." This last sentence overstates their position since it does not apply to persons at young and old ages, as the authors acknowledge elsewhere in their article. In fact in the second half of the nineteenth century, there were about as many deaths under age 5 as

TABLE 1 Table 2 from Kermack, McKendrick, and McKinlay (1934a)

England and Wales: relative mortalities
(The figures in the zero row refer to deaths under one year per 1000 births.)



SOURCE: Reprinted from W. O. Kermack, A. G. McKendrick, and P. L. McKinlay, "Death-rates in Great Britain and Sweden: Some general regularities and their significance," *The Lancet*, Vol. 223, pp. 698–703, ©1934, with permission from Elsevier.

there were between ages 10 and 65 (based on data from the Human Mortality Database). KMM also presented data for Scotland and Sweden but most discussion has centered on the findings for England and Wales.

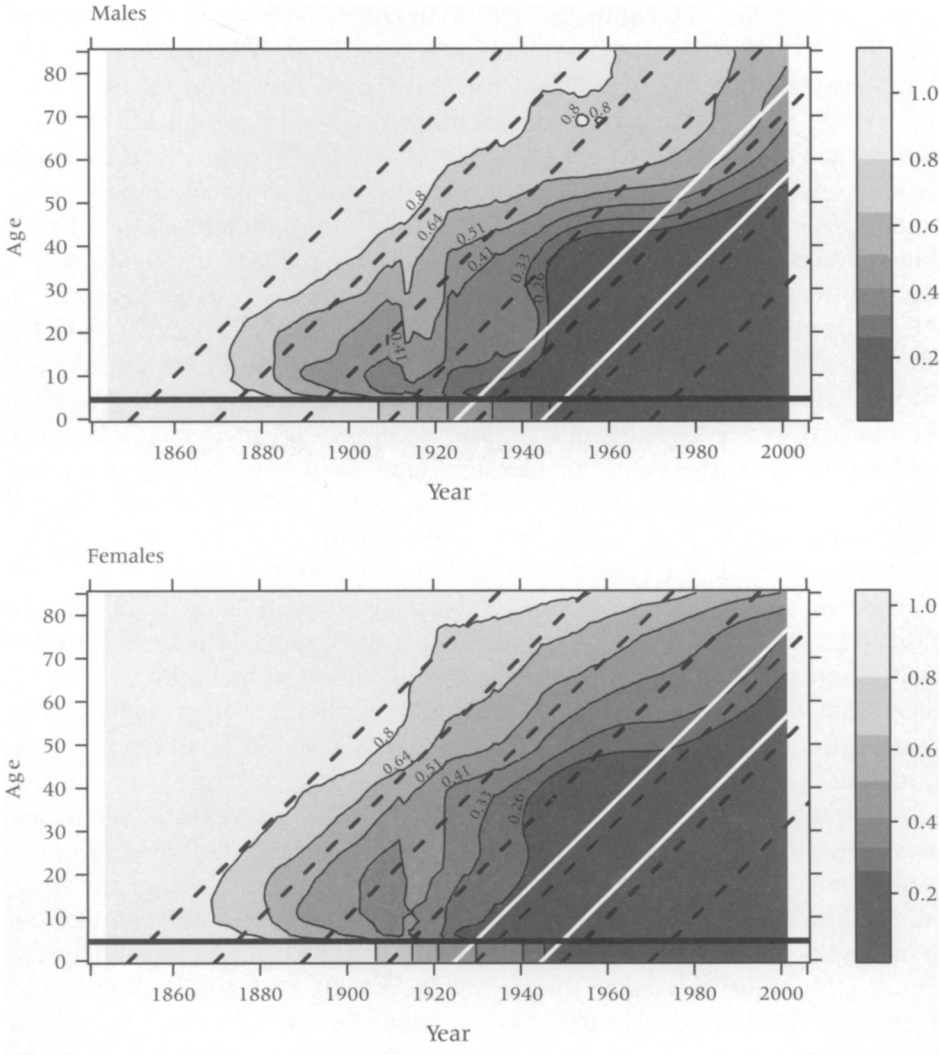
The widely cited results of KMM are a useful starting point for discussion since they show a straightforward and easily interpreted cohort effect that alters the relative risk of mortality by the same proportion at most ages. The authors present virtually the whole set of data available to them to avoid the criticism of showing only that subset of the data consistent with their standpoint. Moreover, they emphasize that their conclusions should be applicable beyond the period within which they were formulated, permitting validation with out-of-sample data from 1930. KMM was preceded by a study by Derrick (1927), who presented age-specific mortality rates for England and Wales in the period 1841–1925 by year of birth and, like KMM, 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" (p. 144). In the accompanying discussion some of the commentators were less convinced, including Major Greenwood, the first Professor of Epidemiology and Vital Statistics at the London School of Hygiene and Tropical Medicine and later president of the Royal Statistical Society, who stated that "almost any hypothesis could be made to cover these [results]" (see the discussion in Derrick 1927, pp. 144 and 154). The debate remained lively: in the discussion of the work of the Statistics Committee of the Royal Commission on Population in the late 1940s, the Secretary, J. G. Kyd, wrote subsequently: "We had on our Committee Mr Derrick, the great exponent of [the generational] theory, and we had on the other side Professor Kuczynski [Reader in Demography at the London School of Economics], who was the exponent of the year of observation theory, and the arguments were so furious at some of these meetings that I sometimes had considerable doubt whether the rate of mortality among the Committee might not be unduly high!" (Kyd 1953, cited in Davey Smith and Kuh 2001, p. 701).

Although the analyses of Derrick and of Kermack and his colleagues are now extensively cited by those who view cohort effects as dominant, Derrick and others who held similar views actually lost the argument at the time, and for the next half-century attention was concentrated almost entirely on period effects. Hobcraft, Menken, and Preston (1982, p.12) concluded 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." Kuh and Davey Smith (1993) noted that even in the 1930s as their analyses were being published, the predictions of Derrick and Kermack et al. went unconfirmed, and cohort approaches fell out of favor. With the resurgence of interest in cohort patterns noted by Davey Smith and Lynch (2004, p. 691), however, interest in this topic has been renewed, not

only for interpreting current mortality but also as a tool for forecasting future mortality.

Figure 1 shows KMM's approach for England and Wales by sex, updated to 2006[8] using unpublished estimates of mortality rates by the Government

FIGURE 1 Contour map showing age-specific mortality rates by age and sex indexed on values for 1841–50, England and Wales, 1845–2006



NOTES: Values are 10-year averages by age and year similar to Kermack, McKendrick, and McKinlay 1934a. Values for ages 1–4 years replaced by infant mortality values. Cohorts shown as dashed lines; limits for 1925 to 1945 "golden generations" shown as solid lines. Cohorts born during the period 1925–45 (and centered around the early 1930s) have exhibited higher rates of mortality improvement than those born in surrounding earlier or later decades. They are therefore often referred to as the "golden generations" or "golden cohorts" (e.g., Office for National Statistics 2008, p. 79), especially in actuarial discussions. For further discussion, see Murphy (2010a).
SOURCE: Government Actuary's Department, unpublished estimates.

Actuary's Department for single calendar years and single years of age. The upward diagonal lines represent birth cohorts, and the contours (similar to those drawn in Figure 1 but using more detailed data) are lines of equal age-specific mortality rates relative to the age-specific rates in the 1840s. Thus the contour maps provide an overview of the way in which mortality changes, especially for facilitating comparisons of the patterns of mortality decline for different age groups in order to identify possible modes of change (Vaupel et al. 1997).[9] Patterns below age 20 are clearer than those in Table 1. Values are also available for each single year/single age combination apart from values adjacent to the borders of the figure such as ages 0 to 4 years, where there are insufficient observations to calculate the centered average. Figure 1 shows the value of infant mortality below age 5 because of its importance for overall mortality. The data for cohorts born in the period 1841–90 show some tendency for isoquants of both adult men and women to be at an angle of 45 degrees but only until 1930 and between ages 20 and 60. After 1930 the diagonal pattern seems to hold only for women over age 40—illustrating why cohort approaches fell out of favor.

KMM has been better received in recent decades, especially since the authors also anticipated later life-course approaches by suggesting that prenatal and infant experiences were likely to be the cause of their assumed cohort effects. For example, Hobcraft, Menken, and Preston (1982, p. 15) said their work provided a "convincing demonstration of the power of a cohort approach to data." Preston and Wang (2006, p. 638) stated, "Cohort influences on mortality have been recognized since the pioneering work of Kermack, McKendrick, and McKinlay [1934a]. Most of the successful studies, like theirs, used graphical methods to demonstrate that age patterns of mortality by cohort were very different from those arranged by period and to argue that the cohort patterns reflected genuine and persistent influences embedded in cohorts." Finch and Crimmins (2005, p. 1734) characterized KMM as among studies that "clearly documented the existence of cohort effects." A series of articles in the *International Journal of Epidemiology* (see Davey Smith and Kuh 2001) devoted to the work of Kermack was overwhelmingly positive about KMM's contribution. A dissenter was Szreter (1988, 2004; see also Szreter and Woolcock 2004), who argued that public health and housing measures were important for nineteenth-century mortality change. Szreter pointed out (2004, p. 705) that mortality improvement starting at different ages at different time periods could produce the observed patterns and that the lines in KMM's Table 2 (shown as Table 1 in this article) were "wavy."

Definitive statements such as those above suggest that the existence of nineteenth- and early-twentieth-century cohort mortality patterns has been established with few reservations. However, there are six potential difficulties in accepting KMM's conclusion concerning the preeminence of cohort patterns. The first is that accepting KMM's conclusion requires accepting that the form of presentation in Table 1 shows clear-cut linear diagonal patterns

for cohorts from 1850. Davey Smith and Ebrahim (2001, p. 4) concluded that "for 10 year olds mortality started to decline in 1855, for 20 year olds in 1865, for 30 year olds in 1875, and so on." Both Table 1 and the more detailed data in Figure 1 show values below age 20 that do not exhibit such clear-cut patterns. (Figure 5 below presents an alternative way of identifying these trends.) Table 1 contains seven sets of observations (excluding the values centered around the year 1845 for which cohort and period are not separable) that include values for the two age groups centered around ages 10 and 20 years for both the same time period and the same cohort. The values for the same *period* are much closer to each other than values for the same *cohort* in every case; that is, the contour lines should appear vertical rather than diagonal at these ages in Table 1. Above age 60, only one inconclusive observation for the post-1845 cohorts is shown, so there is no evidence for cohort patterns outside the age range 20–60. Among those born in the last two decades of the nineteenth century, only 20 percent of total deaths occurred between ages 20 and 60, compared with just under 30 percent below age 20 and about 50 percent at ages 60 and over. Given that the diagonal pattern is confined to half of the observations in Table 1, the conclusion that cohort patterns exist is based on a sub-section that contains only 17 percent of all deaths included in the table (based on data from the Human Mortality Database).

The second difficulty is that there is an inconsistency in KMM's approach, which Hobcraft, Menken, and Preston (1982, p. 16 and footnote 4) note but which appears to have been ignored subsequently. The baseline data used to index mortality in the period 1841–50 necessarily refers to very different cohorts; for example, those aged around 70 in 1841–50 were born around 1775. The pattern identified by Derrick (1927) and by KMM started to emerge around the mid-nineteenth century, about the time vital registration was introduced in 1839. The "remarkable" result of constant *cohort* ratios for cohorts born in the period 1850–90 disappears, *unless* it was the case that mortality was essentially unchanged in the period before 1841. If so, the contour lines in Table 1 would be similar to what would have been obtained if they were relative to values for cohorts born around the 1770s, the earliest cohort used to construct the reference population in Table 1. In fact, age-specific mortality rates for the earlier period were not available in the 1930s. Later analyses suggest that mortality gradually improved, largely monotonically, from the middle of the eighteenth century through most of the nineteenth, before improving more rapidly at the end of that century (Wrigley and Schofield 1979, Table A3.1 and Figure 7.6; Wrigley et al. 1997, Table A9.1; Woods 2000; Charlton and Murphy 1997).[10]

The third difficulty is that the most widely used graphical approach for identifying cohort patterns in contemporary mortality schedules fails to show a predominance of cohort patterns for the period before 1930. Conversely, the presentation used by KMM fails to show clear cohort patterns for those

born after 1930, where the "golden generations" born around 1925–45 are centered (Figure 1). Thus conclusions about the existence of cohort patterns depend substantially on the particular form of presentation chosen. In addition, there is no agreed way of producing or interpreting graphical results, some of which may give undue visual prominence to events at some ages—for example, deaths at working ages as in Table 1 and Figure 1.

The fourth problem relates to the interpretation of diagonal isoquants like those shown in Table 1. Such isoquants cannot identify unambiguous cohort mortality patterns in situations of essentially monotonic improvements in mortality. Preston and Wang (2006, p. 638) write: "Less successful have been statistical efforts to disentangle age, period, and cohort effects.... [W]hen nonlinear terms for cohort and period are introduced along with a common linear drift term, the typical result across countries is that the linear drift term explains the great majority of variation in all-cause mortality" (see also Janssen and Kunst 2005). To illustrate this point, consider the case of a general improvement in mortality, that is, constant linear drift. In this case, the model can be written as

$$\mu_{x,c,t} = \exp(\lambda c)\, \mu^*_x, \tag{1}$$

where $\mu_{x,c,t}$ is the mortality rate at age x for cohort c at time t, and μ^*_x is the set of age-specific mortality rates for the reference cohort. Hence

$$\mu_{x,c,t} / \mu^*_x = \exp(\lambda c),$$

showing that the mortality ratio changes uniformly with cohort value; that is, isoquants would be at 45 degrees as in Table 1 and would be equidistantly spaced for equal changes in mortality ratios (i.e., equal changes of the logarithm of the ratios).

However, since $t = c + x$, model (1) can also be written as

$$\mu_{x,c,t} = \exp(\lambda (t - x))\, \mu^*_x,$$

that is,

$$\mu_{x,c,t} = \exp(\lambda t)\, \{\mu^*_x \exp(-\lambda x)\},$$

where $\mu^*_x \exp(\lambda (t - x))$ is the period age-specific distribution of mortality rates at time $t = 0$, then

$$\mu_{x,c,t} / \{\mu^*_x \exp(-\lambda x)\} = \exp(\lambda t),$$

showing that the mortality ratio also changes uniformly with period value and would also be equidistantly spaced for equal changes in mortality ratios, but in this case the isoquants would be vertical in the Table 1 presentation.

Thus a diagonal isoquant pattern observed in such presentations as Table 1 and Figure 1 is not necessarily evidence of the existence of cohort patterns for a population with largely uniform improvement in mortality.

The angle is determined solely by the choice of reference mortality schedule: if a period standard had been chosen, the contour lines would be vertical rather than diagonal. The distinguishing feature of model (1) is that the contour lines are equidistant for equal changes of the ratios of mortality rates at a given age, but almost any angle of the isoquants, including vertical, forward or backward sloping, may be obtained by the choice of an appropriate initial reference schedule. Non-equidistant straight parallel lines, which would indicate differential rates of mortality improvement associated with a given cohort or time period, would be potentially informative, but no claims for the existence of cohort patterns have been made based on non-equidistance.[11] Thus the assertion that diagonal lines such as those in part of Table 1 by themselves provide compelling evidence for cohort patterns may be challenged.

The fifth difficulty in asserting the predominance of cohort patterns is that the cohort pattern was identified clearly only for Britain (Scotland showed a broadly similar but less clear-cut diagonal isoquant pattern compared to that for England and Wales) but not for Sweden, a country with more accurate data over a longer time period from 1751. Although KMM acknowledge that the Swedish results were less convincing, little subsequent attention has been given to the Swedish findings (but see Fridlizius 1989). (Updated values for Scotland and Sweden are shown in Murphy 2010a.) Nineteenth-century Britain was heterogeneous, with very different mortality regimes in urban and rural areas to the extent that Britons may be more usefully considered as a set of sub-populations with their own mortality regimes, rather than as a single group undergoing simultaneous changes (e.g., Woods 1985, 2000; Szreter and Mooney 1998). The unusual pattern of cause-specific mortality decline over this period in England and Wales, possibly associated with the rapid rise of urbanization, was noted by Preston (1976) and Woods et al. (1997).

Two main questions regarding mortality trends for England and Wales in the second half of the nineteenth century remain. First, why did adult mortality start to improve initially among younger adults before progressively starting to improve at older ages along a line on the mortality surface that is broadly similar to that of cohorts born around 1850? Second, why was improvement in child mortality delayed and infant mortality even more so? These questions will now be considered.

Epidemiological transition theory and period patterns of mortality change

The sixth reservation about the conclusion of KMM is that hypotheses other than cohort effects should be assessed. Even though their cohort model poorly described mortality trends in Sweden, the only country other than Britain investigated, most attention has been given to the results for England

and Wales. Derrick (1927) and KMM interpreted these results as cohort effects based on a process of elimination, since they were unable to identify any other plausible explanation that produced what they regarded as clear-cut parallel diagonal lines.[12] An alternative explanation for the relatively faster rates of mortality improvement at young, but not at the youngest ages (Woods, 2000; Harris, 2001) is offered by epidemiological transition theory (Omran 1971, 1998), whereby mortality initially starts to improve as communicable diseases decline and only later do chronic diseases fall substantially. Because communicable diseases led to a higher proportion of deaths among young people (Preston, Keyfitz, and Schoen 1972; Preston 1976), rates of overall mortality improvement are initially greater for younger than for older persons. That mortality tends to decline more rapidly at younger than at older ages as overall mortality improves was observed empirically in the set of cross-sectional life tables for societies with different levels of mortality[13] that underpinned the first set of model life tables produced by the United Nations (1955). These life tables were used later by Omran (1971) in developing his theory. This process was also formalized by William Brass in his "logit" life table system (Brass 1971), whereby mortality "pivots" around values at the oldest ages. The Brass model is still widely used (Vassin 1994) and remains the standard description of how mortality improves through time. An elaborated form of the logit model underpins the set of model life tables for 191 countries produced recently by the World Health Organization (Murray et al. 2003).

The reasons for the nineteenth-century mortality decline remain a matter of debate. The main arguments center around the relative contribution of such factors as improving nutrition (McKeown 1976, 1988) and public health measures (e.g., Szreter 1988); however, both are considered mainly as period processes.[14] Considerable evidence documents the association of such period factors with the epidemiological transition in both now-developed and contemporary developing countries (e.g., Omran 1971, 1998; Gribble and Preston 1993), although the theory is not without its critics (e.g., Riley 2001; Mackenbach 1994).

Cohort patterns in Britain emerged at the same time as communicable diseases were declining during the transition between the second ("age of receding pandemics") and the third ("age of degenerative and man-made diseases") stages of Omran's epidemiological transition, but such patterns were much less prominent at both earlier and later times. Changing causes of death could explain parsimoniously not only why mortality improvement among adults started at younger ages, but also why it was confined to a particular sub-section of the mortality contour map coinciding with the principal period of shift from communicable to chronic diseases.[15]

Thus the firmness of KMM's conclusions may result from a failure to consider other explanations.[16] While the interpretation of the hand-drawn lines in Table 1 is subjective, the general patterns for the relatively small

fraction of deaths that occur to working-age adults are consistent both with cohort effects acting throughout adult working life and with period changes in cause of death during the epidemiological transition. The transition after about 1870 from an approximately constant level of mortality to a generally declining trend cannot be attributed unambiguously to underlying period or cohort mechanisms without additional information.

The role of infant and child mortality

Neither epidemiological transition theory nor the approach of KMM explains why infant mortality in England and Wales did not improve significantly until the twentieth century (Harris 2001; Woods 2000), about 30 years after child mortality started to fall (Figure 1).[17] Early-age experiences hold a special place in life-course theories that emphasize the key role of prenatal, infant, and early childhood factors via early nutrition and disease environments; KMM's proposed explanation looks particularly insightful in retrospect:

> The anomalous behaviour of this first age group [i.e. infants] is, however, consistent with the previous postulate of improved child environment. It has been observed above that the improvement in the infantile death rate became apparent only after 1901. If we remember that before birth and during its first year of life, the child is dependent for its welfare to a very large degree upon the general health and vitality of the mother, then it would be expected that a substantial improvement in the health of the latter would show itself in a reflected improvement in the infantile death rate. (1934a, p. 701)

The lack of empirical justification for this explanation suggests that it is a *post hoc* hypothesis. Szreter (2004, p. 706) refers to the explanation as "devious" and "diversionary," although Hobcraft, Menken, and Preston (1982, p. 16) argue that it is amply supported by later empirical findings such as the study of Preston and van de Walle (1978). On the other hand, KMM's own data for Sweden fail to support this hypothesis since the relative timing of the onset of substantial mortality improvement for infants and young adults from the second half of the nineteenth century was reversed. The mortality rate at ages 15–25 years was only 4 percent lower in 1901–10 than in 1841–50, whereas infant mortality fell by 44 percent over the same period (KMM, Table 4).

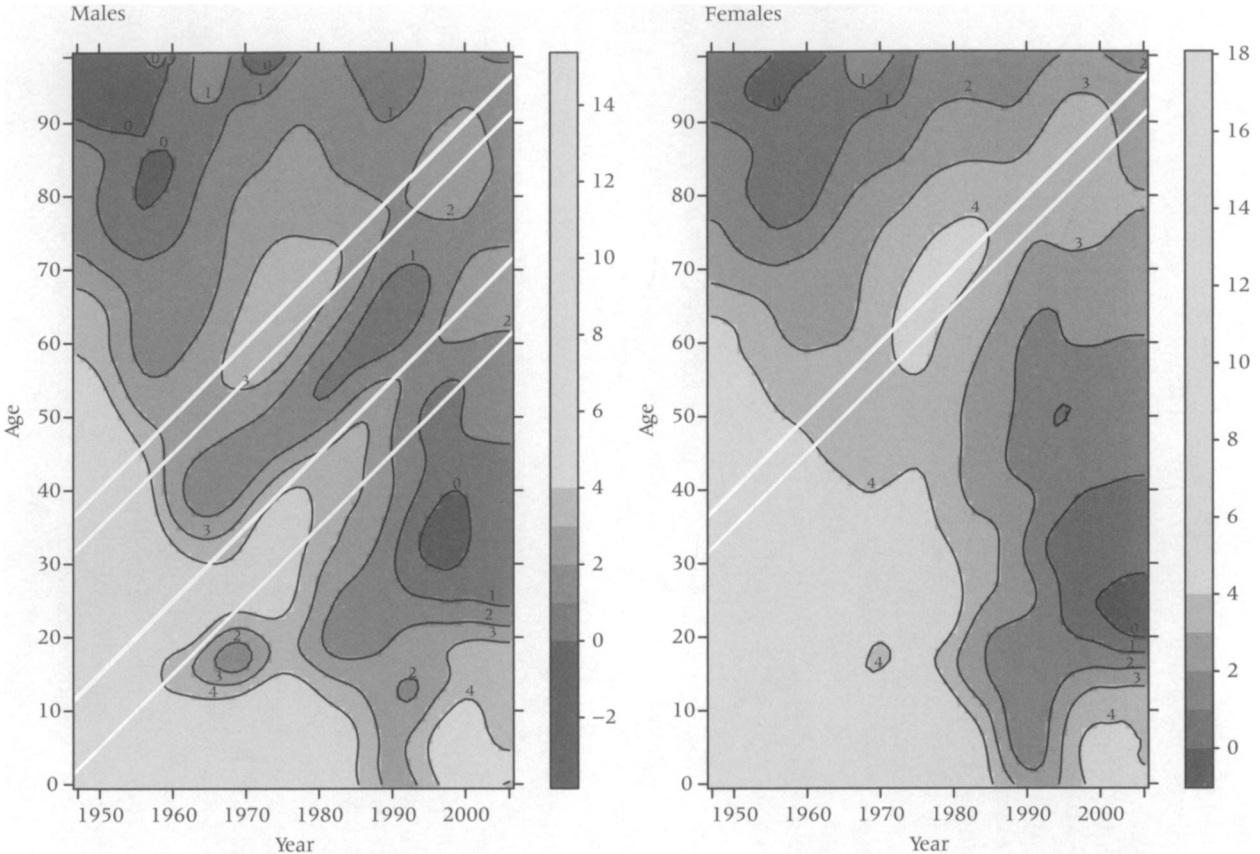
In *Reading History Sideways* (2005) Thornton compares life tables for more recent populations with different mortality regimes and does not find a pattern of delayed improvement of infant mortality (i.e., infant mortality does not remain relatively constant while mortality improves at older ages). Thus the phenomenon of delayed infant mortality decline appears to be limited to the nineteenth- and early-twentieth-century mortality transition in countries such as England and Wales. It is not replicated in Sweden at that time or in developing countries today.

Mortality decline in Japan

Derrick (1927) and KMM are concerned with broad movements and major transformations in mortality regimes, and they concentrate on mortality at young and middle ages. For more recent cohorts, mortality at older ages is increasingly important, but this limits studies only to cohorts born up to the early decades of the twentieth century since later cohorts will not have reached the highest ages. Figure 1 provides little evidence for the existence of persistent cohort effects for persons born in the early decades of the twentieth century in Britain. Japan, however, has been identified as having particularly clear patterns, with an advantaged (or "select") generation of both men and women born just after 1910, who are now nearly centenarians (Willets 2004).[18] Therefore these groups will be considered so as to assess whether such advantages continue up to the highest ages.

Recent graphical approaches for identifying cohort effects have mostly presented rates of change of age-specific mortality rates that do not require selection of an arbitrary reference population (e.g., Office of Population Censuses and Surveys 1995; Richards et al. 2007; MacMinn and Weber 2009). However, Willets (2004, Tables 8–10) introduced a different way of identifying select cohorts for Japan. He estimated age-specific rates of mortality change using a nine-point centered moving average, and he presented graphically only values within 95 or 90 percent of the maximum value between ages 45–95 in each calendar year from 1954 to 1995.[19] The relative advantage for males born just after 1910 appears to have ended by about 1982, when they reached about age 70, since there is no subsequent year when the rate of mortality improvement for these select cohorts reached 90 percent of the highest level. This analysis excludes males below age 45. Because deaths in the age range 45–70 account for only about one-sixth of total deaths for these male cohorts, the overall lifetime impact of events occurring at these ages is relatively small. For female cohorts, however, the advantage appears to continue to higher ages up to 1995, the last year for which data are available, when members of these cohorts were in their early 80s. Willets (2004, p. 875) argues that these results support the view that certain cohorts such as those of Britain's "golden generations" may continue to maintain high rates of mortality improvement up to the highest ages, although given that this pattern fails to hold for Japanese men, the evidence is by no means overwhelming.[20] One limitation of this form of graphical presentation is that it removes all period influences since it compares values within each time period; thus it is impossible to draw conclusions about the relative contribution of period and cohort factors. To address this issue, Figure 2 shows estimated percentage rates of change.[21] The use of model-based derivative estimates and the availability of data up to 2007 provide an additional 12 years of mortality change compared with Willets's 2004 analysis. The figure also presents estimates for all ages and for all available years, and therefore provides a more comprehensive basis for

FIGURE 2 Contour map showing estimated rate of mortality improvement (percent per annum), by age and sex, Japan, 1947–2007



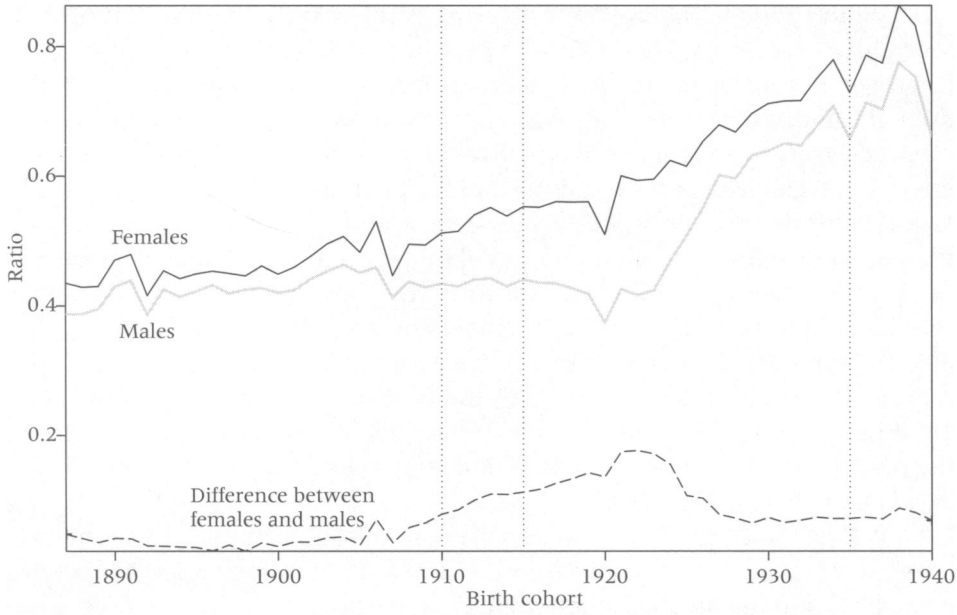
NOTES: Based on derivative of logarithm of spline-smoothed age-specific mortality rates (m_x). Limits for 1910 to 1915 select cohorts (and for 1935 to 1945 select cohorts for males only) shown as diagonal solid lines.
SOURCE: Human Mortality Database.

comparison (data obtained from the Human Mortality Database are available only from 1947).

Willets (2004, Tables 8–10) presents data for only the top half of the mortality contour map, from age 45 years, but Figure 2 shows that mortality improvement in Japan was particularly substantial among infants and children. If comparisons were made with the maximum value of the complete age range, the conclusions above about advantaged cohorts would be very different. Figure 2 suggests two additional points. First, the dominant post–World War II patterns are mainly period ones—the isoquants are more often vertical than at 45 degrees. Second, high rates of improvement for female cohorts of the years centered on 1910 have not continued beyond 1995, the end point of Willets's (2004) analysis when members of this cohort were in their early 80s. The attenuation of this effect will have a substantial influence on overall mortality of these cohorts, since with the period mortality levels of the late 1990s, two-thirds of Japanese women would still be alive at age 82. Therefore the persistence of high rates of improvement in select cohorts up to the oldest ages remains unsubstantiated.

Willets (2004, pp. 873–874) also drew attention to another apparent cohort effect in Japan: male cohorts born in 1935–45 have high relative rates of mortality improvement, although this advantage becomes attenuated for ages about 70 years and older, at which values for different cohorts become more similar as was the case for men of the 1910 cohort. (There is little sign of such a cohort effect for women born in 1935–45.) For Japan, the high rates of mortality improvement for men born around 1925 may be more usefully interpreted in light of the effect on these cohorts of Japan's participation in wars in China in the late 1930s and in World War II. Figure 3 shows the ratio of numbers of men and women at age 60 relative to the corresponding birth cohort size.[22] The deficit of surviving males born around 1925 is clear. Combatants, who were drawn from the fitter members of their cohorts, were particularly likely to be killed in action. About 2 million Japanese men died in combat in the period 1937–45, mostly from disease and starvation rather than trauma (Dower 1986). Japanese men in the armed forces who survived may have suffered from injuries and other privations; therefore, the relatively poor mortality experience of this group later in life is plausible. Discussions of the long-term effects of early cohort disadvantage often concern the opposing consequences of selection (frailer members of the cohort are more likely to die earlier, leaving a group with above-average fitness) and scarring (survivors retain long-term damage due to adverse experiences); however, among those involved in combat, it is likely that both forces will act in tandem to produce unambiguously worse outcomes for the cohort.[23] Thus the apparent advantage of surrounding cohorts in part reflects their experience compared with that of a uniquely disadvantaged group—suggesting that concentrating on "advantaged" while ignoring "less advantaged" groups may hinder full analysis, even though most recent work on birth cohorts has been concerned with advantaged groups.[24]

FIGURE 3 Ratio of number of persons aged 60 to cohort number of births, by sex, Japan, birth cohorts 1887–1940



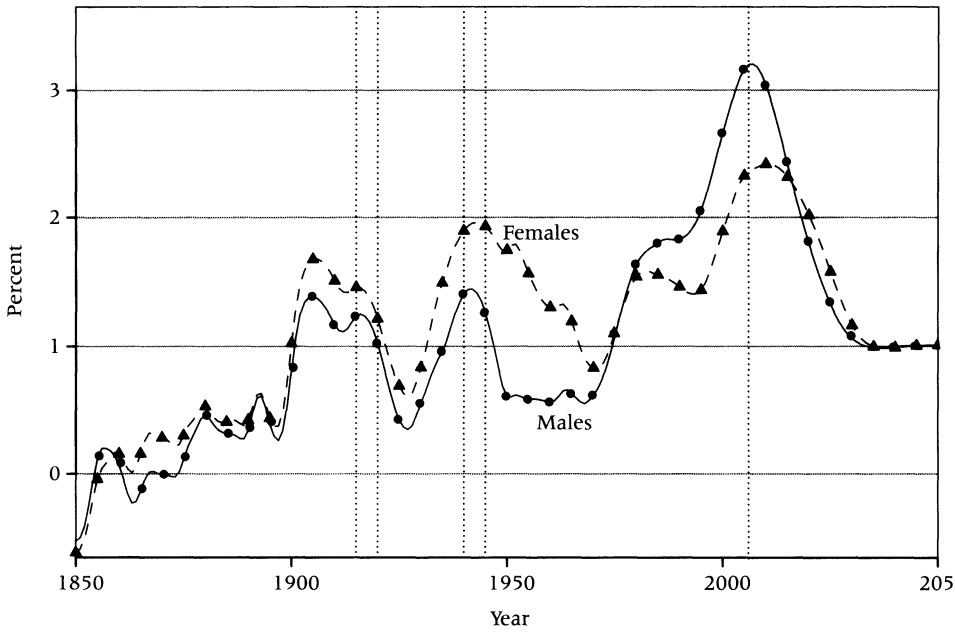
NOTE: Limits for the 1910 to 1915 cohorts and the location of the 1935 cohort are marked by dotted lines.
SOURCE: Human Mortality Database.

Britain's "golden generations": Recent patterns and methods

Britons born in the period 1925 to 1945 and centered around 1930 are the third population for which cohort effects have been identified. These golden generations have been identified as experiencing especially high rates of improvement in mortality (Dunnell 2008; Office of Population Censuses and Surveys 1995). In the early 2000s, overall age-standardized mortality rates (both sexes combined) have been improving at a historically unprecedented rate exceeding 2.5 percent per annum (Figure 4),[25] but this value is heavily influenced by trends in deaths at ages where they are concentrated. In 2005, just over 50 percent of deaths in England and Wales occurred between ages 65 and 85, 56 percent for men and 45 percent for women, members of the golden generations. Current British official mortality projections assume that these cohorts will continue to enjoy advantages up to the highest ages. As these cohorts are replaced in the main mortality age groups by less-favored cohorts, rates of mortality improvement are expected to decline to a much lower level (Office for National Statistics 2008).

Information on Britain's golden generations is available at present only up to about age 75. This falls to about age 60 or less for the postwar generations, whose experiences are crucially important for the interpretation of

FIGURE 4 Annual rate of improvement in standarized death rate by sex, England and Wales, estimates and projections, 1851–2050

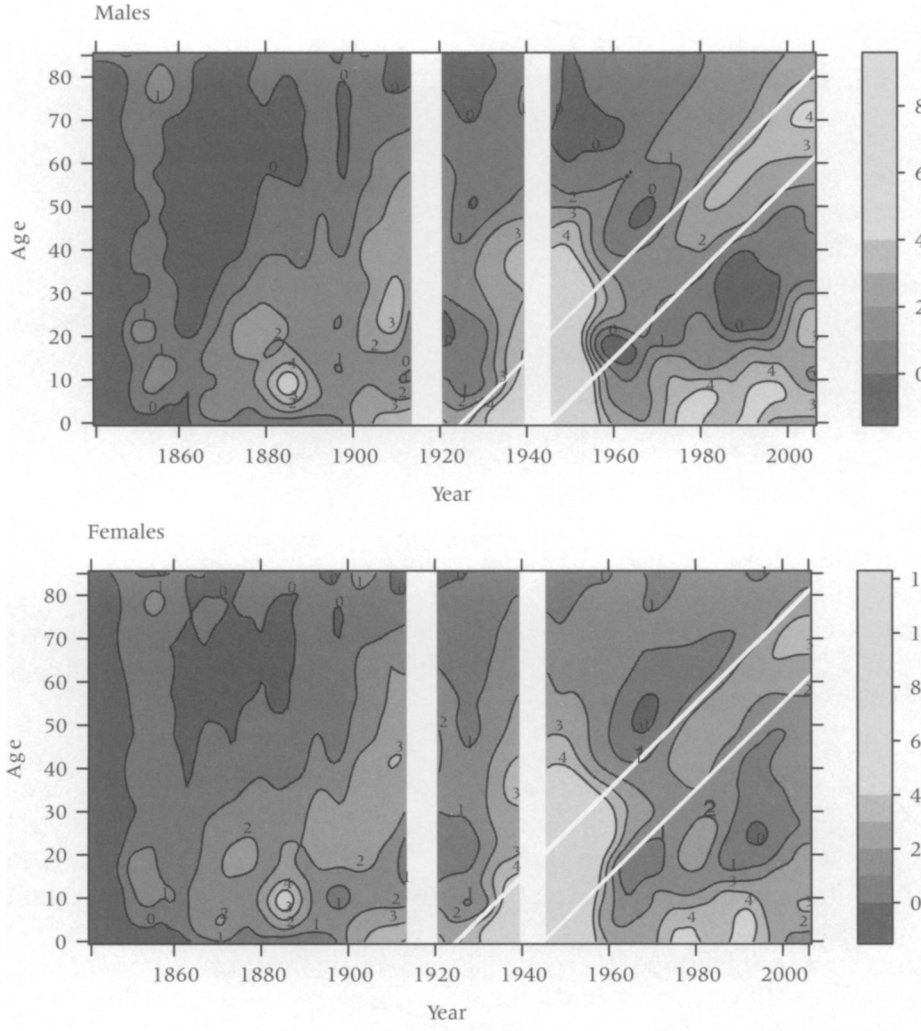


NOTES: Based on WHO European standard population (World Health Organization 2001). Smoothed values based on smoothing spline fitted to logarithm of standardized death rate data. Fit excludes years 1914–20 and 1940–45.
SOURCE: Government Actuary's Department, unpublished estimates for period 1851–2006 and 2006-based ONS official projections for the period 2007–2050.

mortality trends and mortality forecasts, but who have not yet started to suffer high mortality rates (at age 60, mortality rates for men and women in Britain in 2005 were about one-sixth of the values in their first year of life, that is, in 1945). The presentation in Figure 1 is relatively uninformative because cohort patterns are identified as deviations from equidistance of the contour lines rather than by their slope. Therefore Figure 5, using the form of presentation in Figure 2, shows estimates of annual age-specific rates of mortality change but excluding years 1914–20 and 1940–45 from the calculations.[26] While the main patterns are period ones over the whole surface (and would be even more distinctively so if the years for World Wars I and II were included), the golden generations show higher rates of mortality improvement along the 45-degree diagonal line for ages above 40 than surrounding cohorts for both men and women. However, the method used by KMM to identify later nineteenth-century cohort patterns shown in Figure 1 gives little indication of cohort patterns in the period 1930 to 1960.[27]

Other ways in which cohort patterns have been identified include fitting statistical models that incorporate some or all of age, period, and cohort variables (see, e.g., Richards's 2008 analysis of the golden generations). As noted earlier by Preston and Wang (2006, p. 638), such analyses usu-

FIGURE 5 Contour map showing estimated rate of mortality improvement (percent per annum), by age and sex, England and Wales, 1841–2006



NOTES: Based on derivative of logarithm of spline-smoothed age-specific mortality rates (m_x). Fit excludes years 1914–20 and 1940–45. Limits for 1925 to 1945 "golden generations" shown as diagonal solid lines.
SOURCE: Government Actuary's Department, unpublished estimates.

ally cannot discriminate between period and cohort effects in situations of largely monotonic mortality change, which is the general pattern for overall mortality change in Western countries over at least the last century. While it may be possible to identify clear period or cohort influences in particular cases (such as cohort influences on lung cancer mortality), most such analyses are unable to establish the predominance of cohort or period effects. Formal statistical models may produce misleading conclusions if the common linear

drift is definitively attributed to either the period or the cohort dimension, especially if the models fail to allow for interactions between age and period components (as pointed out, e.g., by Wilmoth 1990).[28]

Conclusions

Mortality rates in Britain in the early 2000s have been improving at over 2.5 percent per year and are expected to continue to do so at a similar rate in the short term since no predictable circumstance is expected to alter this trend. (Unforeseen events might do so, but it is impossible to know whether or when a deleterious period effect such as a lethal epidemic may occur.) However, for forecasting mortality when later generations come to dominate mortality trends, extrapolation of current values becomes inadequate. It is important to know what factors are driving mortality change, but little attention has been given to the cohorts that will dominate mortality trends beginning about 2020. The recent literature relevant to forecasting mortality in Britain is concerned almost entirely with members of the golden generations, who are usually compared with earlier cohorts who have reached high ages, especially persons born around World War I who matured in the interwar depression years. Therefore the fact that later cohorts appear to do well compared to such earlier ones is unsurprising. Dunnell's (2008, p. 19) recent overview concluded that there is no clear explanation for these findings, but only a series of hypotheses that include:

—changing smoking patterns between generations;
—better diet and environmental conditions during and after World War II;
—less competition for resources as persons born in periods of low fertility age; and
—benefits from the introduction in the late 1940s of the welfare state.

Among other possible explanations, Britain's golden generations may have benefited more than other cohorts from the introduction of new medical treatments and pharmaceutical products. And, based on US experience, improvements in food preparation and packaging in the 1920s and 1930s may have had a beneficial influence on later mortality (Singer and Manton 1998).[29]

However, when mortality has been generally improving for a century and a half, more insight may be obtained by investigating cohorts who appear to have particularly *poor* rather than *good* patterns. Those born in the 1950s in Britain were the first beneficiaries of the welfare state, including the National Health Service, and were raised in an economic boom period with unprecedented family life stability; nevertheless, these postwar cohorts have shown only low mortality improvement or even mortality increases in some cases (Figure 5). A key question is whether these cohorts are likely to retain this relative disadvantage (Murphy 2010a).

A related question is what magnitude of effects might be expected from the hypothesized explanations for cohort mortality change in contemporary developed countries such as those for Britain's golden generations. The Japanese male cohorts born around 1925 suffered very large numbers of excess deaths disproportionately among their fitter members, and poor conditions were experienced by many of the survivors; yet their subsequent excess mortality is relatively small compared with what might have been expected. Thus, the plausibility of various hypothesized factors with apparently much smaller potential for influencing later mortality should be assessed.[30] Other examples of adverse conditions at a different stage of the life course, in infancy or prenatally, that might be expected to lead to poor mortality outcomes have been investigated. The severe 1869 famine in Finland was found later to have no discernible effect for those born around that time (Kannisto, Christensen, and Vaupel 1997). The extreme hardships suffered by those born around the time of the 1941–44 siege of Leningrad in World War II (estimated average daily rations of around 300 calories, containing virtually no protein) and the Dutch "Hunger Winter" of 1944 have been extensively studied for subsequent excess health risks (Lumey et al. 2007; Stanner et al. 1997; Bell 2004; Croft 2004). Lumey and van Poppel (1994, p. 245) note that "the long-term effects are not easily detected" and appear to be relatively small. For example, an increased risk of high blood pressure among children aged about 6–15 years at the time of the Leningrad siege was identified, but girls who were exposed were actually taller than those who were not (Sparén et al. 2004; Koupil et al. 2007). Babies conceived or born during the 1959–61 Great Leap Forward famine in China do not show higher mortality than pre- or post-famine cohorts (Song 2009). Some disadvantages have been identified for persons born around the time of the 1918–19 influenza pandemic, such as over 20 percent higher prevalence of heart disease at ages 60–82 in the United States (Almond 2006; Mazumder et al. 2009). Compared with the extreme differences experienced by the above cohorts relative to the immediately surrounding ones, the variability in health and nutrition risk factors among cohorts born in countries such as Britain at different segments of the twentieth century is much smaller, and the magnitude of effects on later mortality would be expected to be correspondingly smaller.

The analysis presented here is not designed to question the range of convincing and innovative studies that have established the existence of early-life effects on later mortality and health outcomes at the individual level (e.g., Barker 1994; Kuh and Ben-Schlomo 1997; Davey Smith et al. 1997; Jefferis, Power, and Hertzman 2002; Hayward and Gorman 2004; Bengtsson and Broström 2009; Bengtsson and Lindström 2000, 2003), and the existence of cohort effects associated with earlier events such as patterns of smoking (e.g., Doll et al. 1994, 2004). But it argues that these well-grounded studies have sometimes been used inappropriately to contend that cohort effects are the dominant driver of contemporary national-level mortality

trends without establishing any underlying mechanism—just as period effects were often simply assumed to have underpinned mortality change in the postwar period. Three studies cited at the outset that have made strong and unambiguous claims for the existence of cohort effects have been examined using a variety of approaches. The evidence has been shown to be much less convincing than often asserted. Care is needed to ensure that such findings are not applied inappropriately, such as assuming current levels of mortality improvement are transient on the grounds that they are—and will continue to be—primarily driven by cohorts' experiences of unidentified events that occurred many decades ago.

Many analyses designed to identify cohort effects have been based solely on mortality data cross-tabulated by two of the dimensions of age, period, and cohort. Assertion of the existence of clear-cut cohort effects using such data is incompatible with the long-recognized impossibility of unambiguously identifying such patterns (e.g., Glenn 1976, 2006). Birth cohort, like time period, is particularly uninformative as a classificatory variable since it simply acts as a proxy for the underlying causal processes, which may include characteristics fixed at birth or some later age including duration since previous birth and parents' social class or nutritional status at time of birth. As Hobcraft, Menken, and Preston (1982, p. 5) state, age, periods, and cohorts do not directly determine demographic outcomes; rather they are markers for underlying biological, epidemiological, social, and economic factors. At present, considerable attention is given to identifying the existence and role of cohort effects. There does not appear to be a comparable number of committed supporters of period effects in recent decades, even though that was a dominant paradigm for much of the twentieth century. More attention clearly needs to be devoted to the potential macro-level determinants of mortality change.

Notes

Figures in this article are available in color in the electronic edition of the journal.

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1 See, e.g., Derrick 1927; Kermack, McKendrick, and McKinlay 1934a, b.

2 Finch and Crimmins 2004; Crimmins and Finch 2006a, b.

3 Barker, Osmond, and Law 1989; Barker 1994; Davey Smith and Kuh 2001; Kuh and Davey Smith 1993; Kuh and Ben Shlomo 1997; Janssen et al. 2005.

4 Office of Population Censuses and Surveys 1995; Willets 2004.

5 See, e.g., Hobcraft, Menken, and Preston 1982; Caselli et al. 1985; Wilmoth 1990; Preston and Wang 2006; Wang and Preston 2009. A distinction may be drawn between birth cohort studies concerned principally with identifying birth cohorts with notable characteristics and those concerned with the impact of earlier experiences, including prenatal influences. Some of the latter studies

may coincidentally be concerned with birth cohorts, as in the case where a well-defined temporal event such as a famine leads to a large proportion of malnourished babies in a particular time period, but the time period itself will not be of interest.

6 There is a much longer tradition that has emphasized the importance of period and cohort factors. In 1829 René Villermé stated: "Human height becomes greater and growth takes place more rapidly, other things being equal, in proportion as the country is richer, comfort more general, houses, clothes and nourishment better and labour, fatigue and privation during infancy and youth less" (as quoted by Tanner 1981, p. 162). Bengtsson and Broström (2009, p. 1583) note a similar point made by Järta in 1824.

7 In Table 1, the values at ages 10, 20, and so forth are averages for the ten year-of-age intervals centered around that age, while age 0 is infant mortality; all values are ten-year calendar averages so, e.g., 1845 refers to the period 1841–50.

8 Others who have updated these tables include Harris (2001) and Davey Smith and Lynch (2004), but both have used banded data. Vaupel et al. (1997, Figures 22 and 24) show contour map data for England and Wales and Sweden using both 1870 cohort and period reference schedules. Vaupel et al. also give a useful summary of the development of such graphical approaches.

9 These values, like those of KMM, are ten-year calendar averages of mortality rates indexed to the values at the same age for the period 1841–50. Values are located on Figure 1 at the middle calendar year and age. The values are therefore essentially identical to KMM's, except that they are unweighted averages of ten adjacent single-year age groups, whereas KMM used five- or ten-year rates. The values are shown for comparability with the earlier results, although this may not be the most efficient way to present such data (Murphy 2010a).

10 However, one should acknowledge "the relative paucity and inaccessibility of reliable, relevant demographic evidence for this particular period, the 'dark age' of Britain's modern historical demography, 1780–1850" (Szreter and Mooney 1998, p. 84), and alternative interpretations of mortality trends exist (e.g., Razzell 1993). More recently, information on adult height has also been used to draw inferences about earlier mortality patterns (e.g., Floud, Wachter, and Gregory 1990). Szreter and Woolcock (2004, p. 658) argue that an "urban mortality crisis" in the second quarter of the nineteenth century had the effect of stabilizing overall mortality levels rather than increasing them. The fact that vital registration coverage was incomplete until around 1870 does not appear to have invalidated mortality rates computed for the period from 1840 (Lee and Lam 1983), although the magnitudes of changes being considered here are relatively small.

11 Figure 1 shows a series of isoquants each 20 percent lower than the preceding one rather than with equal absolute differences, both because this is more informative about the nature of the underlying process and because absolute differences relative to an arbitrary standard have no useful interpretation. However, the visual differences in these alternative methods of presentation are minor.

12 KMM considered the possibility of changing causes of death, but rejected it on the grounds that it seemed to be an unlikely explanation: "It may also be pointed out that some of the virulent infective diseases, such as smallpox and enteric fever, which have both practically disappeared [by 1930], exerted a heavy toll on the children, and the removal of these diseases naturally exerted the greatest effect on that age-group...[A]lthough the summation of causes such as the above might produce an effect most marked in children, and a smaller effect in the adult population, nevertheless it would be somewhat surprising if the quantitative regularity just pointed out should emerge" (p. 700).

13 Because these tables relate to different populations, direct cohort influences could not exist.

14 For summaries of this controversy, see Woods (2000) and Grundy (2005).

15 Moreover, the emergence of mortality improvement starting with young adults from about 1870 is also consistent with the time scale of major improvements in public health measures (Szreter 1988, 2004).

16 The possibly confounding effect on overall mortality of rapid urbanization with

substantial area-level mortality differentials may lead to problems in identifying underlying mortality trends.

17 Woods (2000) tentatively concludes that this fact is likely due to changing pathogen virulence. For alternative explanations for the lag in improvement in infant and child mortality, see Finch and Crimmins (2004), who cite the role of inflammation in infants and children associated with later mortality, and Hall and Drake (2007), who emphasize the role of infant diarrhea.

18 Wilmoth (1990) identified a mortality disadvantage for both male and female cohorts born in Japan around 1900.

19 Willets (2004, Table 11) also shows values of mortality improvement above the mean in a given year.

20 Kannisto (1994) showed that above age 80, age-specific mortality changes were more strongly correlated with values at other ages in the same time period than with values for the same cohort, and concluded that period factors were the most important at advanced ages. However, given the vulnerability of older persons to short-term factors such as temperature extremes and infections, year-to-year changes would be expected to be greater than cohort-to-cohort annual changes (Kalkstein and Valimont 1987; Ekamper et al. 2009). Such essentially random period factors could potentially mask underlying long-term period or cohort factors.

21 In this form of presentation, the angle of isoquants provides useful information. The values shown are analytical first derivatives of a smoothed spline model fitted to the logarithm of mortality rates for each year of age (Murphy 2010a). Unlike a centered moving average fitted to $2n+1$ values, in which the first and last n values are missing, spline-based estimates are available for all time points.

22 Japan, which has very low rates of immigration and emigration, is one of the few countries where such comparisons can be used to show mortality trends.

23 Cohort effects may not act similarly over the whole age range. Events around the time of birth (both pre- and postnatal) can lead to different chances of cardiovascular disease starting in late middle age (Barker 1994; Barker, Osmond, and Law 1989). Inflammation or other insults in early childhood may influence mortality at even higher ages (Finch and Crimmins 2004; Catalano and Bruckner 2006), although other studies find no effect (Gagnon and Mazan 2009). Other causal factors occurring later in life, such as the effects of smoking on mortality, especially from lung cancer, are largely manifested as a cohort phenomenon at older ages (Charlton and Murphy 1997). It is possible that the competing influences of selection and scarring could lead to cohort effects that are reversed beginning at some age. For example, early adverse circumstances may lead to proportionately more deaths at young ages among the frailer members of the cohort, while the surviving and consequently, on average, fitter members of the cohort may experience lower mortality at older ages (e.g., Vaupel, Manton, and Stallard 1979; Vaupel and Yashin 1985). The existence of such "crossover" effects has been debated for many years, especially in the context of racial differences in mortality in the United States. While it is often difficult to separate the effects of data errors from real processes (Coale and Kisker 1986; Elo and Preston 1997; Preston and Elo 2006), evidence is accumulating that the crossover is real, even if possibly confined to high or very high ages (e.g., Dupre, Franzese, and Parrado 2006).

24 While a number of studies have investigated the outcomes of specific earlier experiences such as poor fetal environment, these have different objectives than systematic studies to identify particularly advantaged groups such as that of MacMinn and Weber (2009), who identified 129 male (104 clearly and 25 tentatively) and 128 female (102 clearly and 26 tentatively) advantaged cohorts in the Human Mortality Database. There are no corresponding studies to identify disadvantaged cohorts.

25 These are based on the author's calculations using the WHO European standard (Doll and Cook 1967), with data from the Human Mortality Database updated with published deaths (Office for National Statistics 2009).

26 The very sharp fluctuations in the "outlier" War years 1914–20 (which also include the 1918–19 influenza pandemic) and 1940–45 are removed since the reasons for their particular patterns are well-defined. It

can be argued that these should be included, since to exclude the major period factors simply because the underlying cause is well-recognized would tend to minimize the influence of period factors; our main interest, however, is in years for which no obvious explanation exists.

27 Moreover, the form of presentation here makes the identification of a clear-cut dating of the onset of mortality improvement problematic compared with the presentation in Figure 1. However, the results are sensitive to the choice of reference population. In Figure 1 if the reference population had been taken as either a period or cohort schedule located in the middle of the period covered, thereby making it more typical of the overall pattern, there would be even less sign of diagonal isoquants (available on request from m.murphy@lse.ac.uk).

28 The interpretation of statistical models including age, period, and cohort factors is a long-standing area of interest. For contrasting views of some of the issues in fitting and interpreting such models, see Yang et al. (2004), Carstensen (2006), and Murphy (2010b).

29 An alternative mechanism for cohort influences—childhood morbidity, especially inflammation (later extended to include height; Crimmins and Finch 2006a)—was suggested by Finch and Crimmins (2004), although they argued that the suggested mechanism is relevant only to cohorts born in the nineteenth century and earlier.

30 This Japanese cohort had higher mortality than earlier cohorts only between the ages of 50 and 70 and involving a relatively small number of deaths; at other ages this cohort exhibited lower mortality (see Figure 2).

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