The Relative Effects of Shocks in Early- and Later-Life Conditions on Mortality

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DETERMINING THE relative importance of early-life conditions compared with conditions in later life as they relate to old-age mortality is crucial for understanding historical mortality decline and for forecasting future developments in mortality. For any given period, a cohort's mortality is affected by the period conditions. Earlier research as well as more recent analysis suggests that in addition to period conditions, early-life conditions may have a critical influence on cohorts' adult and old-age mortality. We do not know, however, the importance of early-life conditions when compared to period conditions.

Previous research has demonstrated that cohorts born closely together may experience different mortalities at specific ages or even over their life courses. These cohort differences, which are sometimes interpreted as signifying "cohort effects" on mortality, are well documented (for example, Wilmoth, Vallin, and Caselli 1990). But the sources of these differences remain uncertain. Early-life conditions are one potential source of differences in cohort mortality. Indeed, it has been shown that early-life conditions and adult health and mortality are linked at the both individual and cohort levels. However, the evidence regarding the importance of a cohort's early-life conditions on later mortality is mixed and debated (Bengtsson and Lindstrom 2000; Finch and Crimmins 2004; Barbi and Vaupel 2005; Catalano and Bruckner 2006; Bengtsson and Broström 2009; Bengtsson and Mineau 2009; Gagnon and Mazan 2009; van den Berg, Doblhammer, and Christensen 2009). In short, the absolute and relative importance of a cohort's early-life experiences for later mortality is uncertain, although it is well recognized that period conditions shape period mortality patterns (see, for example, Oeppen and Vaupel 2002).

This article examines the effects of cohort-level early-life conditions on later adult and old-age mortality and investigates how these lagged effects from early to later life compare with period effects in later life. I use historical time series for five European countries and mortality as a proxy for broad epidemiological and socioeconomic conditions including nutrition and disease load. The results show that shocks that increase cohorts' early mortality above trend have, on average, only weak lagged effects on adult and old-age mortality, and especially so when compared to the effects of shocks in period conditions. In addition, the effects of shocks in early-life conditions are most pronounced soon after the shock (at ages below 20) and are smaller at old ages. These findings are consistent with the emerging literature suggesting that cohorts' old-age mortality is not strongly linked to their early mortality (Bruckner and Catalano 2009; van den Berg, Doblhammer, and Christensen 2009), and they suggest that the majority of variation in adult and old-age mortality is attributable to changing period conditions.

Background

It is obvious almost by definition that period conditions at older ages affect old-age mortality. The other side of the coin, the idea that early-life conditions could also affect old-age mortality and produce cohort differences, dates back at least to 1934 when Kermack and colleagues compared age-specific mortality rates in England and Wales in 1855–1925 to baseline mortality in 1845 (Kermack, McKendrick, and McKinlay 1934; reprinted 2001). They found that cohorts seemed to carry with them the same relative mortality throughout life. Their conclusion that "the health of the man is determined by the physical constitution which the child has built up" is essentially a life-course interpretation of adult health. Preston and van de Walle (1978), who used similar methods to analyze urban French mortality in the nineteenth century, also found a pattern suggesting a cohort decline in adult mortality. Interest in the effects of early-life conditions on adult health and mortality has been revitalized by Barker and colleagues (Barker 1995; Barker et al. 2002; Eriksson et al. 1999), who stress the importance of early-life nutritional status on health in later life.

Few studies have attempted to compare the relative importance of early- versus later-life period conditions.[1] The number of studies that take an exclusive cohort perspective and do not compare the effects of early-life conditions to period effects is much larger. While many of these studies have found links between early-life conditions and later-life mortality, the overall evidence is mixed. Almond's (2006) analysis of US data found elevated disability rates for those in utero during the 1918 influenza epidemic. Mazumder et al. (2010), using the same epidemic as an exogenous shock, found that those exposed to disease in utero have higher prevalence of cardiovascular disease. Cohen,

Tillinghast, and Canudas-Romo (2010), however, analyzing data from 24 countries, detected no long-term mortality effects for prenatal or neonatal exposure to the 1918 influenza. The studies by Almond and Mazumder et al. identified the timing of exposure more accurately than Cohen et al., which may explain the difference in results. It is also possible that old-age health but not mortality is sensitive to early-life exposure to disease.

Studies of the long-term impact of early-life nutritional deprivation have also produced mixed results. Research on the 1866–68 famine in Finland (Kannisto, Christensen, and Vaupel 1997) and the Dutch famine of 1944–45 (Painter et al. 2005) found no association between nutritional deprivation early in life and later mortality. Van den Berg, Lindeboom, and Portrait (2007) analyzed the effects of the 1846–47 Dutch potato famine and found that men who were exposed to the famine in utero had increased mortality at ages above 50, but found no effects for women. In contrast to these results, Fogel (2004) and Costa and Lahey (2005) attributed much of the decline in old-age mortality in contemporary developed countries to improved nutrition.

The most consistent evidence regarding cohort-level links between early-life conditions and later-life mortality uses the state of the business cycle at birth as a proxy for early-life conditions. For example, van den Berg and colleagues (2006, 2008, 2009), studying nineteenth- and early-twentieth-century Danish and Dutch cohorts, observed that being born in a recession versus a boom is associated with increased old-age mortality, especially cardiovascular disease mortality. A study of the 1930s Depression in the United States, however, did not identify any long-lasting health effects for those born during this period (Cutler, Miller, and Norton 2007). This finding could be interpreted to indicate that the boom/bust effect on later-life health may have lessened over time.

Only a few studies have explicitly attempted to compare the effects of early-life and later period conditions. Using historical data for several European countries, Crimmins and Finch regressed cohorts' old-age mortality on early-life cohort mortality and period mortality (Finch and Crimmins 2004; Crimmins and Finch 2006a, 2006b). They found that a cohort's early-life conditions, proxied by early-life mortality, explain almost all (87–96 percent) of mortality variation at ages above 70, while period conditions (proxied by period mortality at ages 0–15) explain a far smaller proportion of variation in old-age mortality. Crimmins and Finch also found that early-life mortality conditions explain more of the mortality variation for women than for men, possibly indicating that men are more robust to exposure to infection in early life. Barbi and Vaupel (2005) analyze the same data and find that while early-life conditions are important, period conditions matter more for old-age mortality.

Some of the earlier results, however, may be biased since they may be influenced by unobserved factors driving the trends in mortality (Hendry 1980). One way to deal with the problem arising from trends in mortality

is to perform the analysis on de-trended variables. In demography, this approach was pioneered by Bengtsson and Lindström (2000, 2003), who analyzed the association between deviation from trend in mortality at the time of birth and deviation from trend in old-age mortality. Bengtsson and Lindström de-trended by using the Hodrick–Prescott filter (Hodrick and Prescott 1997), which has become a common tool in demography (for example, van den Berg, Doblhammer, and Christensen 2009; Gagnon and Mazan 2009). Bengtsson and colleagues have used data from eighteenth- and nineteenth-century Sweden to examine whether mortality conditions early in life, taken as a proxy for exposure to disease, predict mortality in adulthood (Bengtsson and Lindström 2000, 2003; Bengtsson and Broström 2009). Bengtsson and colleagues, using Cox proportional hazard models to estimate the effects of mortality deviations from trend early in life on adult mortality, determined that individuals born during times of high mortality manifest increased adult mortality. The magnitude of the effect is sizable; for example, in their 2009 study analyzing mortality above age 55 for the 1766–1839 birth cohorts, Bengtsson and Broström found that those born in years with very high infant mortality (deviation from trend in the highest decile) faced up to 43 percent higher old-age mortality than those born in times of lower infant mortality. Other researchers who have used similar designs, however, often failed to identify links between being born in times of high mortality and later mortality (van den Berg, Lindeboom, and Portrait 2006; van den Berg, Doblhammer, and Christensen 2009; Gagnon and Mazan 2009). It is not known what explains these inconsistent findings.

Catalano and Bruckner (2006) also employed a de-trending approach to study the association between cohorts' mortality in early life and in later years, using national-level historical mortality data for Denmark, England and Wales, and Sweden. Instead of the Hodrick–Prescott filter, Catalano and Bruckner de-trended the early and later-life mortality variables using ARIMA (Auto Regressive Integrated Moving Average) models and found that higher-than-expected mortality during the first five years of life may decrease life expectancy at age five by as much as 1.75 years, and that the effect is stronger for men than for women. The result suggests that at the cohort level, early-life conditions play an important role in determining later mortality. In follow-up research (Bruckner and Catalano 2009) the authors decomposed the effects of early-life conditions on later mortality by age, and determined that the majority of the effect is attributable to increased mortality at ages below 20 years.

This article studies the relative importance of cohort-level early-life conditions versus period conditions on cohorts' mortality. Using historical mortality time series for five European countries, I examine how variation in cohorts' mortality at a given age depends on cohorts' early-life conditions and on period conditions. To avoid identification problems often present in studies involving age, period, and cohort, I use mortality rates as proxies for period

and early-life conditions instead of using years to indicate cohorts and periods. To avoid spuriousness potentially arising from the fact that all mortality rates have a downward trend, I de-trend the variables and analyze deviations from trend, which I refer to below as "shocks." More specifically, as the dependent variable I use the relative deviation from trend in cohort age-specific mortality. The explanatory variables are based on a cohort's own mortality early in life and on period mortality. Early-life cohort mortality shocks are defined as relative deviations from trend in a cohort's mortality at ages 0 and 1–4 years. I measure shocks in period mortality as relative deviations from trend in period mortality at ages 0–4. Regressing the dependent variable, deviation from trend in cohort's age-specific mortality, on the cohort and period mortality shocks allows me to answer the question, "If, at a given age, a cohort had higher or lower mortality than would be expected on the basis of the surrounding cohorts, how well do the cohort's mortality experiences early in life explain this effect, and how well do period mortality conditions explain it?"

Potential mechanisms

Individual-level mechanisms

While this study uses aggregate data that do not allow separating different physiological and individual-level mechanisms that link early-life conditions to health in later life, it is important to specify what these links could be. Early-life factors influencing later health may be related to nutritional factors in utero (Barker 1995; Barker et al. 2002) and after birth (Eriksson et al. 1999; Gunnell et al. 1996), exposure to disease in utero (Almond 2006) and after birth (Case, Fertig, and Paxson 2005), or broader socioeconomic conditions and deprivation early in life (Arnesen and Forsdahl 1985; Notkola et al. 1985). Although the discussion below treats these factors separately, they tend to occur together.

The effect of early-life nutrition on adult health may operate through physiological and social factors. One of the body's critical systems affected by nutrition early in life is the immune system. Waaler (1984) showed that shorter individuals have excess adult mortality mostly resulting from cardiovascular disease, tuberculosis, and obstructive lung disease, all of which may depend on the functioning of the immune system. Alternative physiological hypotheses are based on the "programming hypothesis" of chronic diseases during gestation or early childhood (Barker 1995; Barker et al. 2002). According to this hypothesis, metabolic "programming" occurs at critical periods of early development and substantially determines health later in life. Eriksson et al. (1999, 2001) suggested that low birth weight (a proxy for in utero nutrition), followed by poor infant growth and rapid catch-up growth, are associated with coronary heart disease, hypertension, and type 2 diabetes. Low birth weight may also be associated with high blood pressure in adult-

hood (Davies et al. 2006), although the magnitude of the effect may be small (Kramer 2000). Nutrition in early life may also affect later health indirectly: through height and size, nutrition may be associated with productivity and socioeconomic status in adulthood.

Early exposure to disease has been linked to current and historical major causes of death— for example, respiratory tuberculosis, which can be latent in an individual for many years (Elo and Preston 1992); and cardiovascular disease, which may be attributable to chronically increased inflammation levels caused by early exposure to disease (Finch and Crimmins 2004; Mazumder et al. 2010). Early exposure to disease may influence later-life mortality indirectly through nutritional deprivation, which in turn may be related to adult health. Exposure to disease may also affect the ability to accumulate social and economic resources, leading to increased risk of death (Case, Fertig, and Paxson 2005). On the other hand, early exposure to disease may also improve later health. A certain amount of disease exposure may be beneficial for proper development of the immune system (Gurven et al. 2008), and some researchers hypothesize that modern epidemics of allergies and asthma result from underexposure of the immune system in early life (Folkerts, Walzl, and Openshaw 2000; Holt 1995).

In addition to disease and nutrition, childhood socioeconomic environment may affect adult health through opportunities to accumulate socioeconomic resources, inherited social behavior and attitudes, and the links that childhood socioeconomic environment may have to early-life nutrition and disease exposure (Kuh and Ben-Shlomo 2004). Socioeconomic environment, however, may not be very important for cohort-level mortality, as within-cohort socioeconomic status may be more important than cross-cohort differences.

Cohort-level mechanisms and anticipated effects

The population-level effects of shocks in the epidemiologic environment may differ from the individual-level effects. In the period perspective, there is no confusion. The direction of the effect is almost certainly positive, as any shock that increases mortality at one age is likely to increase mortality at other ages for the same period. The direction of the effects in the cohort perspective, however, depends on the relative contributions of selection, scarring, and immunity.[2]

First, a shock in a cohort's early-life conditions may have a selective effect, killing the weakest members. The influence on later cohort-level mortality is then negative, decreasing mortality. Second, the shock may have a scarring effect that increases the mortality of the surviving cohort. Third, the shock may induce immunity, lowering mortality for the surviving cohort. Thus, the effects of period shocks are anticipated to be positive (increase in period mortality at one age is associated with an increase in other ages), but

the direction and relative magnitude of the effects of shocks in a cohort's early epidemiologic environment are uncertain. In the cohort perspective, it is also unclear at what ages mortality would be altered by a shock in early life. The results of this study will shed light on which of the three within-cohort effects dominates—selection, scarring, or immunity—and which matters more for adult and old-age mortality—cohort or period conditions.

Data, variables, and methods

Data and variables

I use time series data on the numbers of deaths and years of exposure for Denmark (cohorts born in 1835–1915), England and Wales (1841–1915), Finland (1878–1915), the Netherlands (1850–1915), and Sweden (1751–1915). The data quality for these countries is comparatively good. The data source is the Human Mortality Database (University of California, Berkeley and the Max Planck Institute for Demographic Research 2008).

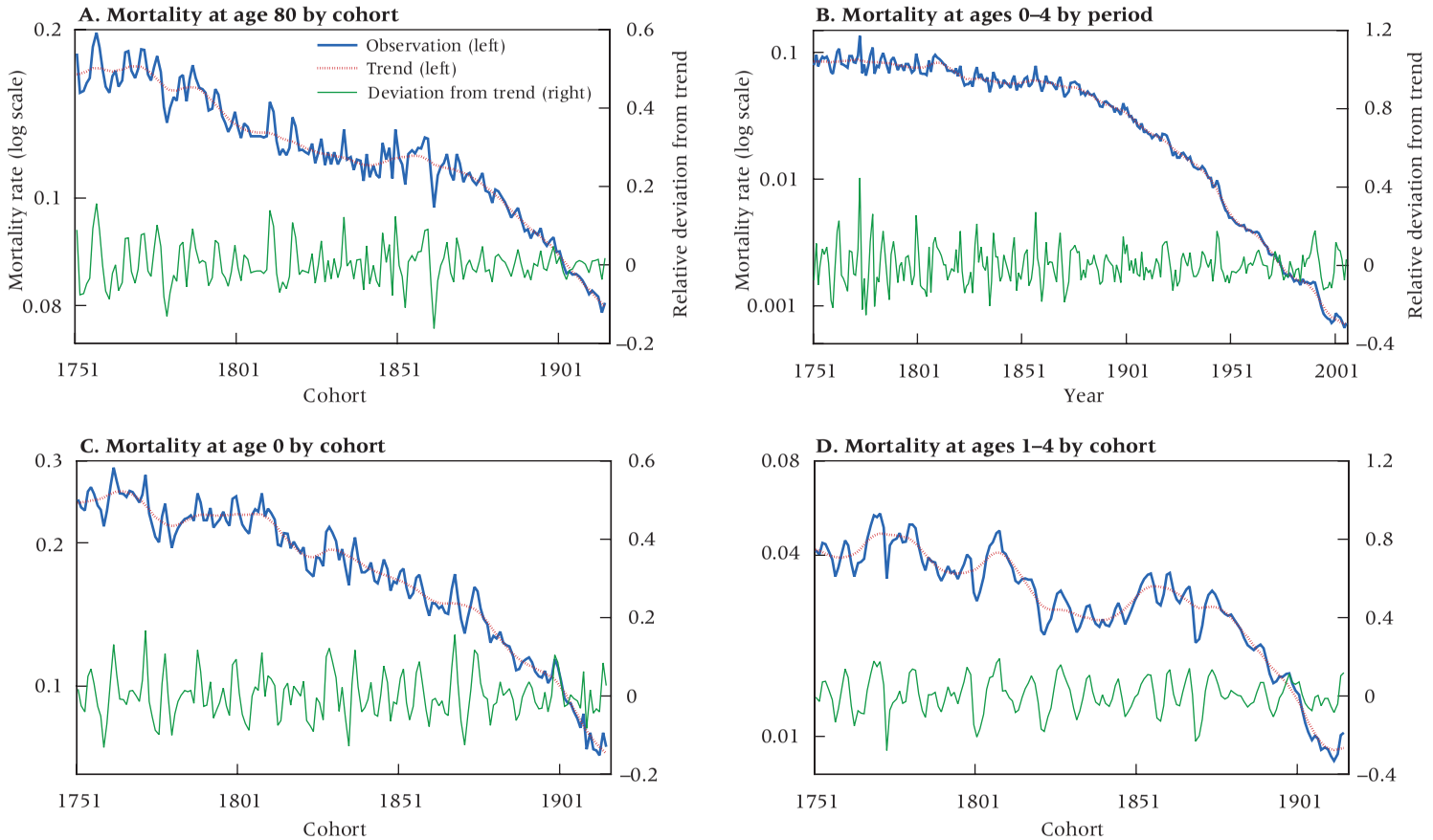
The dependent variable is derived from ${}_1m_x(c)$, which denotes the mortality rate at age x for cohort c. As the explanatory variables I use shocks in cohorts' early-life conditions and in period conditions. Shocks in a cohort's early-life conditions are derived from the cohort's own mortality rates at ages 0 and 1–4, denoted by ${}_1m_0(c)$ and ${}_4m_1(c)$. Shocks in period mortality conditions are derived from period mortality at ages 0–4.

Methods

To reduce heteroscedasticity, I transform the mortality rates to log scale. I translate all variables into deviations from trend to avoid the potential problem arising from unobserved factors driving the trends. In the decomposition to trend and deviation from trend, each series is de-trended over cohorts using the Hodrick–Prescott filter with smoothing parameter $\lambda = 100$, a standard choice for annual data (Maravall and del Río 2007). Without de-trending, the results would be mainly driven by the trends themselves, potentially leading to spurious results.

Figure 1 shows selected variables (original series, estimated trend, and relative deviation from trend) for Sweden, both sexes combined. Panel A shows the data for age 80. Relative deviation from trend in mortality at age 80 is one of the dependent variables; the analysis is performed for deviations from trend for all single-year age groups from 5 to 89. Panel B shows period mortality rates at ages 0–4. Relative deviation in period mortality at ages 0–4 is the period shock and is used to predict deviation from trend in mortality at higher ages. Panels C and D show cohort mortality rates at ages 0 and 1–4. Relative deviations from trend in these rates are the shocks in cohorts' early-life conditions and are used as the cohort predictors for mortality.

FIGURE 1 Sweden, selected mortality time series, both sexes combined



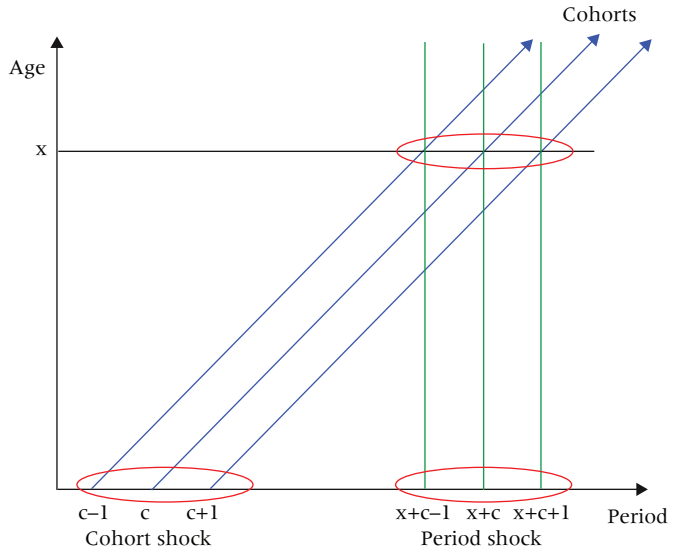
SOURCE: Observations, Human Mortality Database; trend and residuals, own calculations.

Deviations from trend are based on log rates and represent relative short-term increases or decreases in mortality. For Sweden, one standard deviation (SD) shock in period mortality at ages 0–4, measured as the relative deviation from trend, corresponds to an 11 percent increase in mortality. For cohorts, the corresponding increases in mortality for a one-SD shock are 5.6 percent for infant mortality and 7.8 percent for mortality at ages 1–4. For other countries, one-SD shocks in mortality rates are of similar magnitude to those of Sweden.[3]

Figure 2 illustrates how the cohort and period variables are matched with the dependent variable: relative deviation from trend in ${}_1m_x(c)$. It shows three cohorts progressing over time. The dependent variable is based on the cohort's mortality at age x, as shown in the figure (in the analysis x runs from ages 5 to 89). For illustrative purposes, consider calculating the difference in cohort mortality for cohort c at age x compared to the neighboring cohorts c–1 and c+1 at the same age x. These mortality rates are those prevailing in the top right ellipse in the figure. The obtained difference is the deviation from trend in mortality for cohort c at age x (in practice, the Hodrick–Prescott filter is used for de-trending, but the idea is the same).

Deviations from trend may be caused by the cohort's early-life conditions, period conditions, or something else such as chance. To determine how the deviation from trend depends on the cohort's early-life conditions and

FIGURE 2 Conceptual model of how shocks in a cohort's early-life conditions and shocks in period conditions are matched with deviations from trend in age-specific mortality rates

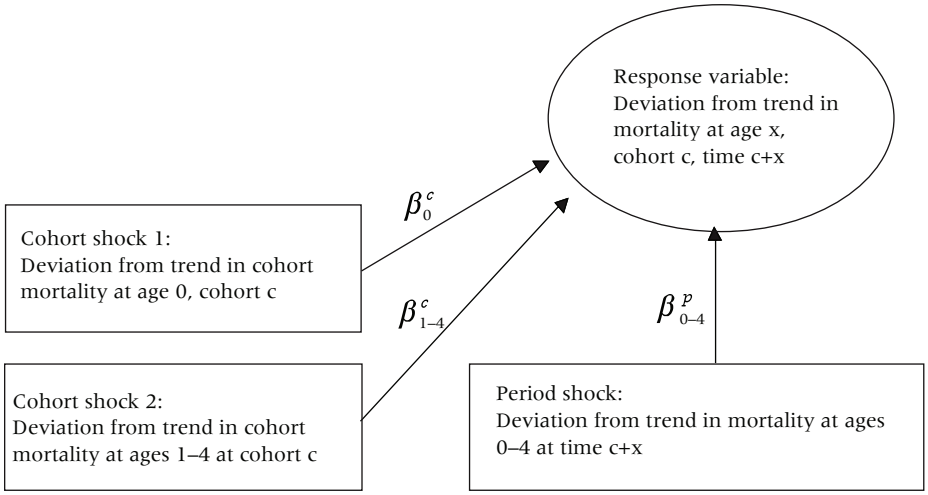


on the period conditions, I calculate the deviation from trend in the cohort's mortality at ages 0 and 1–4 (relevant mortality rates are those prevailing in the bottom left ellipse in the figure), and in period mortality at ages 0–4 (relevant mortality rates in the bottom right ellipse). Once these deviations from trend are obtained, the procedure is repeated for all cohorts. Finally, the deviation from trend in cohort mortality at age x is regressed on deviations in the same cohort's mortality at ages 0 and 1–4, and on deviations in period mortality at ages 0–4. The procedure is repeated for all ages from 5 to 89.

Figure 3 illustrates the regression model. The model is estimated separately for each age $x = 5,6\ldots,89$; thus there are 85 coefficients for both of the cohort variables (for each age from 5 to 89 coefficients β_0^c for cohort mortality at age 0 and β_{1-4}^c for cohort mortality at ages 1–4), and 85 coefficients for the period mortality variables (for each age from 5 to 89 coefficient β_{0-4}^p for period mortality at ages 0–4). If the cohort shocks are positively correlated with later mortality ($\beta_0^c, \beta_{1-4}^c > 0$) for all or most ages x, then the scarring effect dominates the selection and immunity effects. If the cohort shocks are negatively correlated with later mortality ($\beta_0^c, \beta_{1-4}^c < 0$) for all or most ages x, then selection and immunity effects dominate. Finally, if the magnitude of the cohort coefficients is larger than the magnitude of the period coefficients, early-life conditions may be more important than period conditions in determining adult and old-age mortality, and vice versa.

I estimate the cohort and period coefficients by regressing the dependent variable (deviation from trend in mortality) on the cohort and period variables. I control for the most exceptional period mortality shocks—World Wars

FIGURE 3 Illustration of the model used to estimate simultaneously the effects of a cohort's early-life conditions and period conditions on age-specific mortality



I and II (years 1914–18 and 1939–45) and the 1918 influenza epidemic—which might otherwise dominate the period effects. I summarize the estimates graphically, plotting exponentiated coefficients $\exp(\beta)$ against age. The coefficients are standardized to reflect a one standard deviation increase in the independent variables. An exponentiated coefficient for cohort shock at age 0 tells us how much, in proportional terms, the cohort's mortality at a given age increases or decreases for a one-SD shock in log mortality at age 0. The coefficients for cohort shocks at ages 1–4 and for period shocks at ages 0–4 are interpreted analogously. The Appendix provides further details on the model, including a discussion of whether the effects for infant and child mortality should be estimated jointly or separately. Here I present results for a model that estimates the effects simultaneously, as the results do not change if these effects are estimated separately. The finding that these effects can be estimated simultaneously is consistent with earlier research (Myrskylä 2010).

To clarify the differences in the magnitude between the effects for cohort and period shocks, I express the effects also in terms of life expectancy. I calculate the change in life expectancy at age 5 for standardized shocks in cohorts' mortality at ages 0 and 1–4 and for standardized shocks in period mortality at ages 0–4. I use country-specific 1900 cohort life tables as the basis for this calculation. The cohort life tables are used for both cohort and period shocks in order to keep the effects comparable. The effect of a standardized shock in cohorts' mortality at age 0 on life expectancy at age 5 is calculated by first multiplying the original age-specific mortality rates by the estimated coefficients β_0^c and then comparing the resulting life expectancy at age 5 to the original life expectancy at age 5. The effects for shocks in cohort mortality at ages 1–4 and in period mortality at ages 0–4 are calculated analogously, using the cohort coefficient β_{1-4}^c and the period coefficient β_{0-4}^p. I decompose the effect on life expectancy to juvenile and young adult ages (5–19), adult ages (20–59), and old ages (60–89), and average the effects over the five countries.

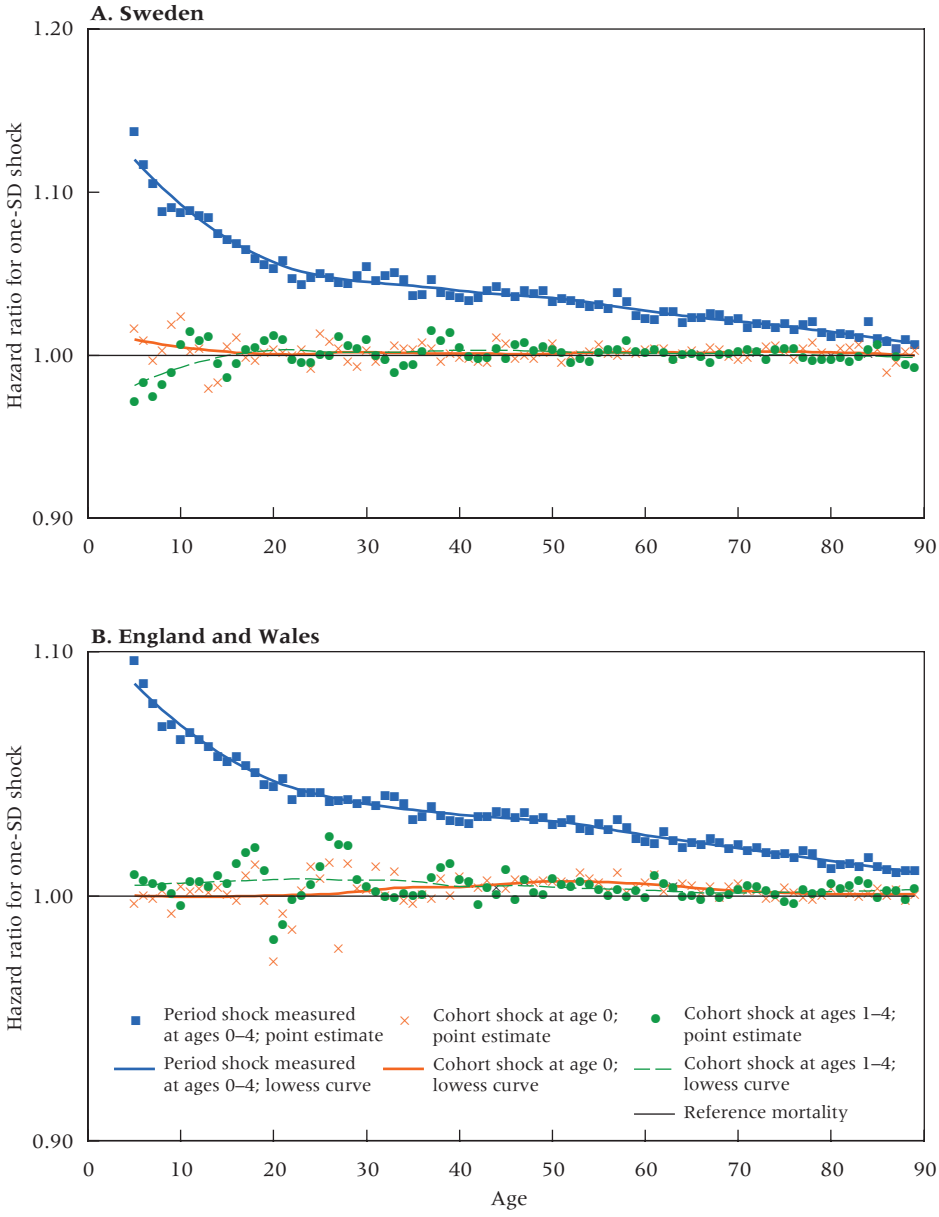
Results

The results are given in three sections. The first section focuses on Sweden and England and Wales. These countries are covered in more detail because the patterns for Denmark, Finland, and the Netherlands are very close to those of Sweden, and because the results for Sweden and England and Wales show informative contrasts. The second section covers all five countries. The third section discusses potential differences by sex.

Sweden and England and Wales

Figure 4 shows the effects of shocks in early-life cohort conditions and later-life period conditions, measured from cohort mortality at ages 0 and 1–4 and

FIGURE 4 Effects on mortality by age of one standard deviation (SD) shocks in period and early-life cohort mortality conditions measured from mortality at ages 0–4, Sweden and England and Wales



NOTE: Lowess curve = locally-weighted smoothed regression curve.
SOURCE: Author's calculations based on data from Human Mortality Database, cohorts 1751–1915 for Sweden and 1841–1915 for England and Wales.

from period mortality at ages 0–4 (Panel A: Sweden; Panel B: England and Wales). The point estimates plotted in Figure 4 are exponentiated coefficients $\exp(\beta)$ and are interpreted as hazard ratios for a one-standard deviation shock; the lines are locally weighted smoothed regression curves (lowess) fitted to $\exp(\beta)$. One can interpret these values as follows. An estimate of 1.02 at age 80 for a shock in the cohort's infant mortality ($1.02 = \exp(\beta_0^c)$ at age 80) means that a one-SD shock in the cohort's log mortality at age 0 is associated with a 2 percent increase in the same cohort's mortality at age 80. Similarly, for the period mortality shocks, an estimate of 1.05 for age 80 means that a one-SD shock in period mortality at ages 0–4 is associated with a 5 percent increase in mortality at age 80 for the same period.

Consider first the effects of the cohort shocks. For both Sweden and England and Wales, the hazard ratios for shocks measured at age 0 are close to 1 for all ages. When the coefficients for Sweden are applied to the Swedish 1900 cohort life table, a one-SD shock in infant mortality results in a 0.4-month reduction in life expectancy at age 5. For England and Wales, the effect of a shock in infant mortality is slightly above 1 between ages 25 and 75, converging to 1 at the oldest ages. When applied to the 1900 England and Wales cohort life table, this increased mortality attributable to a one-SD shock corresponds to a 0.5-month reduction in life expectancy at age 5. Based on the Swedish coefficients, a three-SD shock would correspond to a 1.2-month decrease in life expectancy in Sweden and a 1.5-month decrease in England and Wales. In general, these effects are not large.

The effects of shocks in mortality at ages 1–4 are also small. For Sweden, the hazard ratios are slightly below 1 at ages 5–20, but converge to 1 at older ages. Thus if a cohort had above-average mortality at ages 1–4, it will have below-average mortality at ages 5–20. This result is consistent with theories of selection. For all cohorts, mortality is very low at ages 5–20. Those few individuals who die at these ages may be very frail (accident mortality and wars may constitute exceptions). If mortality at ages 1–4 is above average, this mortality shock may selectively affect those who otherwise would have lived to age 5 but who died at earlier ages. An alternative explanation is acquired immunity, whereby those who survive the event that increases mortality at ages 1–4 acquire immunity for similar events later in life. In this study, however, it is not possible to distinguish between these theories. Whichever mechanism is responsible for the inverse link between mortality at ages 1–4 and 5–20, the effect is not particularly strong, corresponding to 0.3 months of increased life expectancy at ages 5–20 for a one-SD shock, and 0.8 months for a three-SD shock. Furthermore, while the effect is present in Denmark, Finland, and the Netherlands, it is not observed in England and Wales. In summary, while some individual hazard ratios for cohort shocks may differ markedly from 1, the lowess curves show

that, overall, shocks in cohorts' mortality at ages below 5 have little effect on later mortality.

The period coefficients for both Sweden and England and Wales are always above 1, as would be expected. If mortality increases at young ages, it is likely that mortality also does so at older ages. In relative terms, the effects of period mortality shocks dominate the effects of cohort shocks. In Sweden, a one-SD shock in period mortality at ages 0–4 is associated with an 8-month decrease in life expectancy at age 5, and a 6-month decrease in England and Wales. For a three-SD shock the corresponding decrease in life expectancy is 23 months Sweden and 19 months in England and Wales.

All countries

Figure 5 shows the effects for cohort and period shocks by country. The results are grouped so that Panel A shows the effects of cohort shocks at age 0, Panel B shows the effects of cohort shocks at ages 1–4, and Panel C shows the effects of period shocks at ages 0–4.[4]

Panels A and B confirm what Figure 4 suggested. In all countries, cohort shocks have very little influence on later mortality. Cohort shocks experienced in early childhood (ages 1–4; Panel B) decrease mortality at ages 5–20, but the effect is small. In Denmark, Finland, the Netherlands, and Sweden, mortality at ages 5–20 is reduced by approximately 1–2 percent for a one-SD shock at ages 1–4.

Panel C shows the effects of period shocks. In all countries high period mortality at ages 0–4 is correlated with high period mortality at older ages. In proportional terms the period association between mortality at ages 0–4 and older-age mortality becomes weaker over age. However, mortality increases over age at a faster rate than the coefficients shown in Panel C decline, so in terms of absolute mortality the period associations do not weaken over age. In fact, in absolute terms the period relationship between shocks in mortality at ages 0–4 and mortality at older ages resembles the almost universal pattern of human mortality, decreasing up to ages 10–15 and then increasing (these results not shown).

To appreciate the differences in the magnitude of the cohort and period effects, it is useful to look at the effects in terms of life expectancy. Figure 6 shows the effects of three-SD shocks in cohorts' infant and child mortality and in period mortality on life expectancy at ages 5–89. The estimates are based on the coefficients shown in Figure 5 and on country-specific cohort life tables for the year 1900. The effects are decomposed into effects at juvenile and young adult ages (5–19), adult ages (20–59), and old ages (60–89) and are averaged over the five countries. On average, the effect of a shock in cohort mortality at age 0 on life expectancy at ages 5–89 is negative. A three-SD shock results in 0.5 months' reduced life expectancy.[5] The majority of the

FIGURE 5 Effects of cohort and period shocks on the mortality hazard ratio by age and country

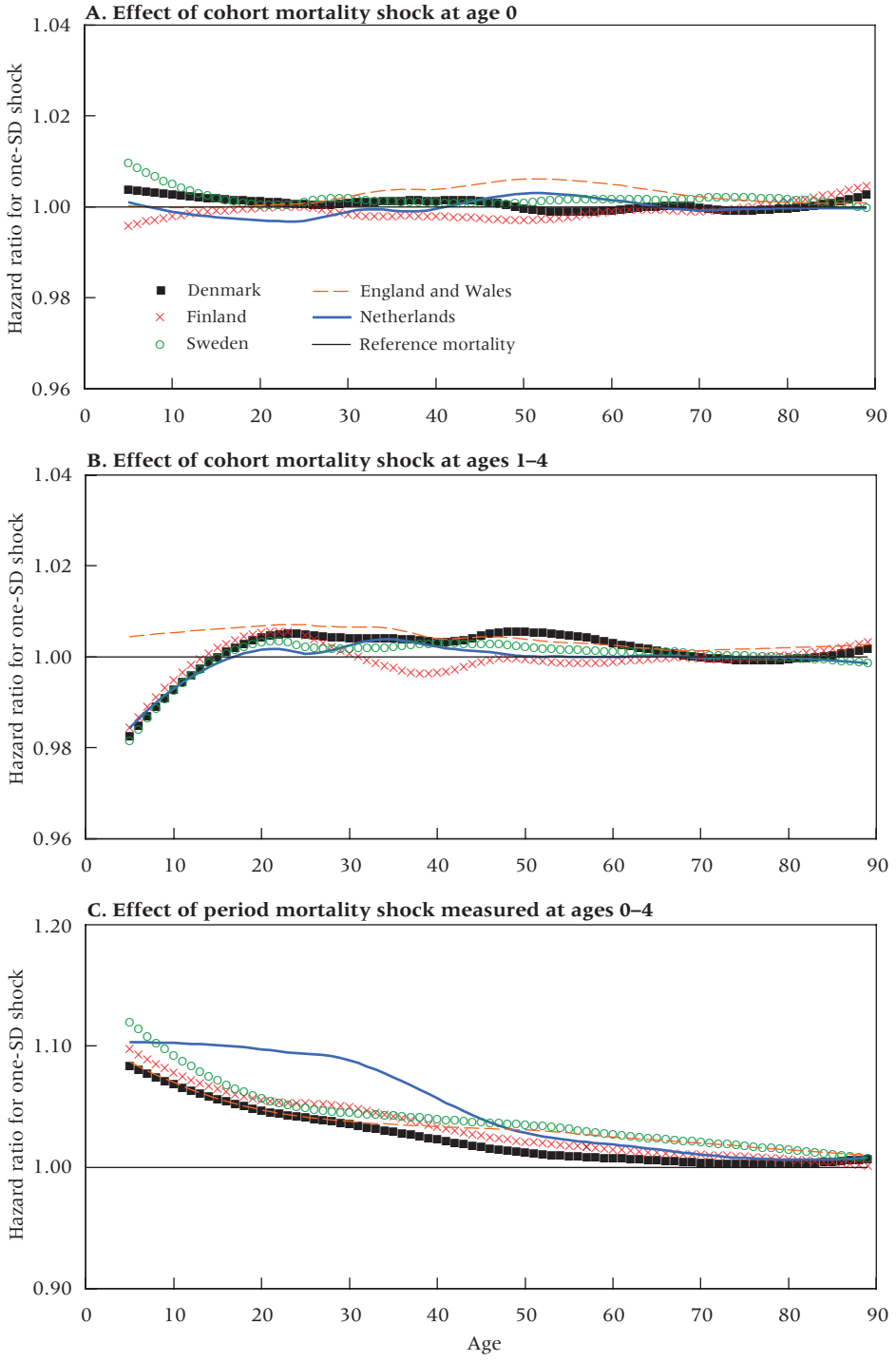
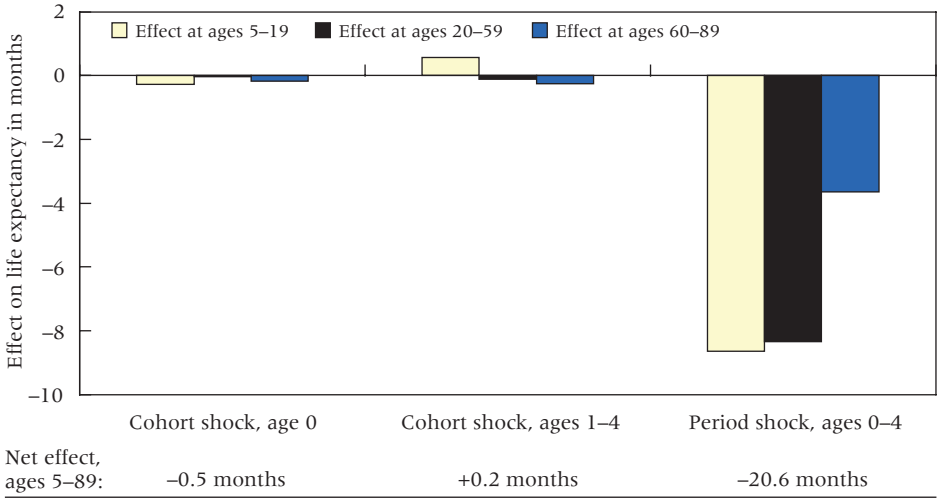


FIGURE 6 Effects of cohort and period shocks on conditional life expectancy (effects scaled to represent a three-standard deviation shock)



averaged effect (–0.3 months) is attributable to ages 5–19. At age 20–59 the effect is less than 0.1 months, and at ages 60–89 the effect is –0.2 months. For shocks in cohort mortality at ages 1–4 the net effect is positive, increasing life expectancy at ages 5–89 by 0.2 months. This positive effect is attributable to ages 5–19 (+0.6 months). At ages 20–59 the effect is negligible, and at old ages (60–89) it is –0.3 for a three-SD shock.

A three-SD shock in period mortality at ages 0–4, in turn, is associated with a decrease in life expectancy of about 8.7, 8.4, and 3.6 months at ages 5–19, 20–89, and 60–89, respectively. The net effect over the whole age range, 5–89, is –20.6 months. In comparison to period effects, the impact of shocks in a cohort's early-life conditions on later-life mortality seems modest.

Sex differences

Figure 7 shows that the effects of cohort shocks are similar for men and women. Panels A and B, which show the effects of shocks in infant mortality, reveal no differences between men and women. The same holds for cohort shocks measured at ages 1–4 (Panels C and D): for both sexes, above-average mortality at ages 1–4 is associated with below-average mortality at ages 5–19, with the exception of England and Wales, which is an outlier for both men and women. As seen in Figure 8, no significant differences by sex are observed for the effects of period shocks.

FIGURE 7 Effects of cohort shocks on the mortality hazard ratio by age, sex, and country

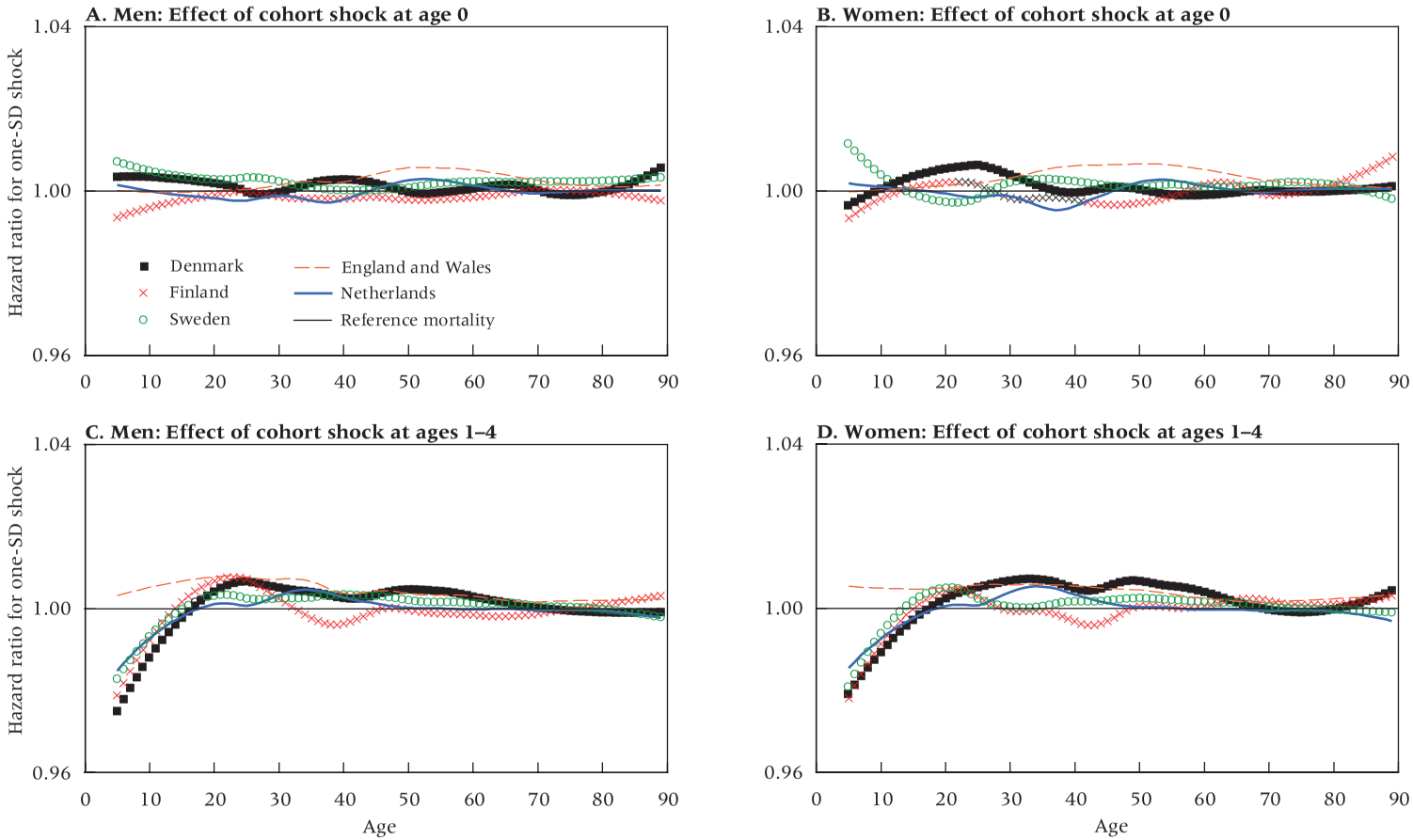
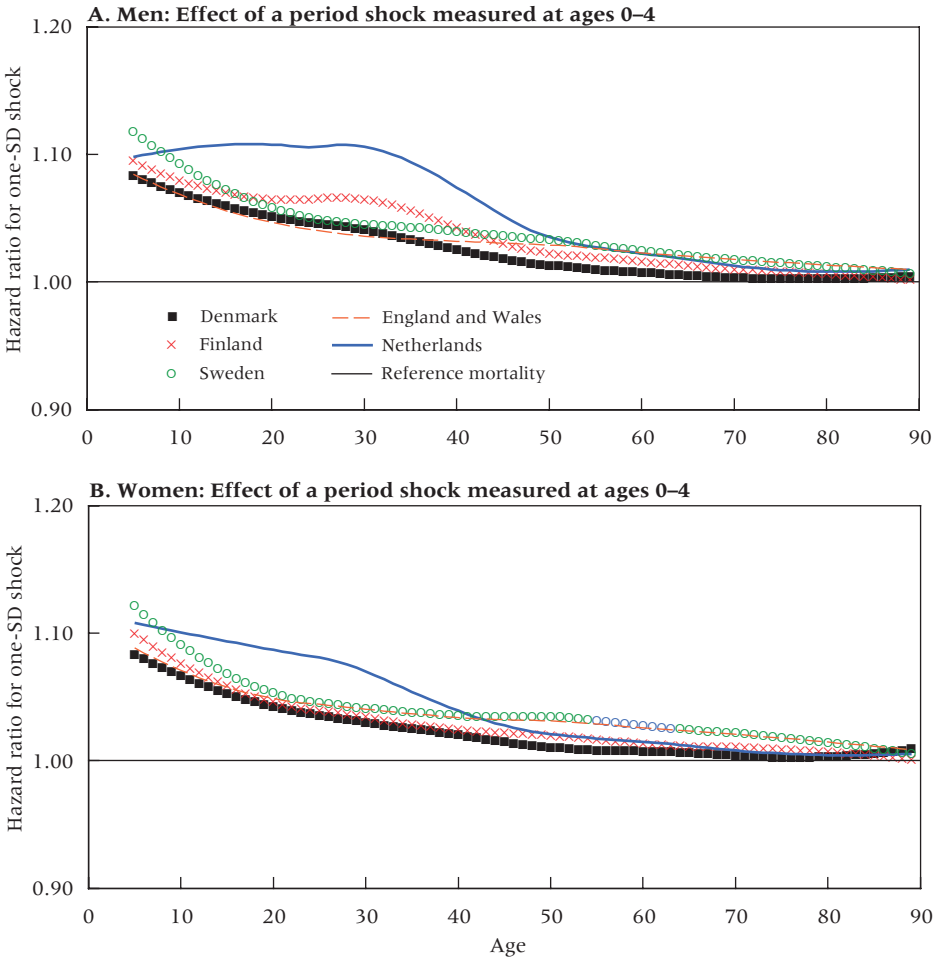


FIGURE 8 Effects of period shocks on the mortality hazard ratio by age, sex, and country



Sensitivity checks

I assessed the sensitivity of the results with respect to the de-trending technique, the regression models, and the time period for which the effects were estimated. This study used the Hodrick–Prescott filter with smoothing parameter λ =100. Alternative smoothing parameters, λ = 6.25 (suggested by Ravn and Uhlig 2002 for annual data) and λ = 1600 (often used for quarterly data), changed the magnitude of the deviations from trend but not the estimates for the model parameters. In addition, differencing instead of using the Hodrick–Prescott filter to de-trend the data did not change the results. Second, I studied the sensitivity of the results to the autocorrelation structure of the regression

model. The results shown above were obtained without autocorrelation in the models. Allowing autocorrelation with lags up to 3 did not change the results. Third, I estimated the models for the period preceding secular declines in mortality. Among the countries studied, only Sweden has observations for the period preceding mortality decline. Mortality was approximately stationary (and data were available) in years 1751–1810. The results for this period were consistent with the results obtained when using cohorts up to 1915. Fourth, I estimated the models for the period when life expectancy at birth was below 50 years (for Denmark, cohorts 1835–1870, England and Wales 1841–1879, Finland 1887–1905, Netherlands 1850–1886, and Sweden 1751–1877). The results for these cohorts were similar to those obtained using the full data. Fifth, I estimated the models using data only for the most extreme years by including only the observations for which the infant mortality shock was in the top or bottom quintile of the distribution. Again, the results did not change. Finally, I estimated the models using absolute instead of relative deviation from trend. The results did not change.

Discussion

This study has analyzed the relative effects of cohorts' early-life conditions versus period conditions on mortality by simultaneously estimating early-life cohort effects and period effects. Using historical mortality data for Denmark, England and Wales, Finland, the Netherlands, and Sweden, I found that shocks in early-life conditions, proxied by cohorts' infant mortality, are only weakly associated with cohorts' later mortality. In the cohort perspective, shocks in infancy (defined as relative deviations from trend in infant mortality) are associated with increased mortality at ages 5–20 and above 60. The magnitude of these effects, however, is small. Shocks at ages 1–4 are inversely, but not strongly, associated with mortality at ages 5–20; at older ages this association vanishes. Period mortality shocks (defined as deviations from trend in period mortality at ages 0–4) were positively and strongly correlated with mortality at all ages between 5 and 89 years, so that a shock that increases mortality at young ages also increases it at older ages. In comparison to the effects of shocks in cohorts' early-life conditions, shocks in period conditions had a much stronger influence on mortality.

The finding that shocks in cohorts' conditions during the first year of life are positively associated with old-age mortality (though only weakly) suggests that early-life events may have an impact on mortality decades later through scarring. The finding that high mortality at ages 1–4 may decrease mortality at ages up to 20 may be due to cohort selection or acquired immunity. For all cohorts, mortality is lowest at ages 5–20. Those who die at these ages are likely to be very frail. Excess mortality at ages 1–4 may act selectively on those who otherwise would have lived to age 5. Alternatively,

or in addition, acquired immunity may be responsible for the effect. In this study, however, it was not possible to separate these two mechanisms. Men and women responded similarly to period and cohort shocks, and differences by sex were small. Theories to explain why one sex would be more robust to harmful early-life conditions are plentiful. The empirical evidence is mixed (Catalano and Bruckner 2006; Crimmins and Finch 2006a; van den Berg, Lindeboom, and Portrait 2007), suggesting that the differences may be small.

In summary, the analyses point to three main results: a small increase in later mortality for cohorts that had above-trend mortality in infancy; slightly decreased mortality up to age 20 for cohorts with above-trend mortality in early childhood; and very strong period dependencies in mortality across all ages. These results apply most clearly to four of the countries analyzed: Denmark, Finland, the Netherlands, and Sweden. England and Wales appears to be an outlier, showing no evidence of decreased mortality at ages 5–20 for cohorts with unexpectedly high mortality at ages 1–4. There are several potential explanations why England and Wales looks different. First, the historical data for England and Wales may be of lower quality than for the other four countries (Wrigley and Schofield 1981). Second, World War I and the 1918 influenza outbreak increased mortality in England and Wales to the extent that life expectancy stalled or even declined among cohorts born between 1880 and 1895.[6] While other countries also suffered from the influenza outbreak, the toll of World War I was higher for England and Wales than for any other country analyzed here. The negative effects of the war on cohort mortality patterns have been well documented (Winter 1976). Derrick (1927; cited in Winter 1976) even claimed that in England and Wales, "the effects of losses during the European War were so great and indefinite as to obscure all normal changes."

Some previous research has suggested that early-life conditions may be more critical than period conditions for old-age mortality (Finch and Crimmins 2004) or that, even if period conditions are more important, early-life conditions proxied by a cohort's early mortality could still have a key role (Barbi and Vaupel 2005). This study adds to the accumulating evidence suggesting that at the cohort level, early-life mortality conditions may not be a critical predictor of adult and old-age mortality (Bruckner and Catalano 2009; Gagnon and Mazan 2009; van den Berg, Doblhammer, and Christensen 2009). The findings of this study are also consistent with Murphy (2010), who finds that period factors may be more important than cohort factors in determining mortality. In addition, the results are consistent with research finding no increased mortality among individuals who survived widespread famines as young children (Kannisto, Christensen, and Vaupel 1997; Painter et al. 2005) or were exposed to disease in utero or during the first year of life (Cohen, Tillinghast, and Canudas-Romo 2010).

Some of the inconsistencies in the literature may be caused by methodological differences. The major difference in study designs between the current analysis and those of Finch and Crimmins (2004) and Barbi and Vaupel (2005), who both find evidence that early-life conditions strongly predict later cohort mortality, is that the current study de-trended the mortality time series before analysis. Without de-trending, the observed correlations are potentially biased by unobserved factors driving the trends (Hendry 1980). De-trending, however, comes with a cost: while analysis of de-trended time series allows better identification of the regression parameters, the external validity of the estimated parameters may decrease. In other words, if the factors that increased or decreased mortality in the short term are different from those that influenced the secular trends in mortality decline, the parameters representing the effects for deviations from trend may not represent the effects for secular changes in early-life conditions. The majority of short-term variation in mortality in the eighteenth and nineteenth centuries was attributable to infectious diseases and epidemics. Declines in infectious diseases and epidemics were also important factors driving the secular decline in mortality in the eighteenth through the twentieth centuries. Therefore it seems reasonable to generalize the effects based on short-term variation to represent the effects of secular changes.

Nevertheless, studies showing negative effects on outcomes other than mortality (Almond 2006; Mazumder et al. 2010) and studies that find mortality effects for persons born during times of adverse early-life conditions (van den Berg, Lindeboom, and Portrait 2006; Bengtsson and Broström 2009) suggest that early-life conditions do influence later-life outcomes. It is possible that early-life conditions exert important influences on cohorts' later-life mortality, but that early-life *mortality* conditions generally do not capture the important aspects of early-life conditions. For example, van den Berg and colleagues (van den Berg, Lindeboom, and Portrait 2006; van den Berg, Doblhammer-Reiter, and Christensen 2008; van den Berg, Doblhammer, and Christensen 2009) found that early-life *macroeconomic* conditions predict cohorts' later mortality: being born in a recession, for example, increases later mortality. Cutler, Miller, and Norton (2007), however, did not identify any health effects in later life for persons born during the Depression of the 1930s. Portrait, Alessie, and Deeg (2010) analyzed the effects of macroeconomic conditions in early life on health in later life among Dutch cohorts born in 1909–1937 and also found that early-life macroeconomic conditions do not explain differences in health outcomes in later life, but later-life period conditions do. These findings mirror the results of the current analysis and suggest that while early-life macroeconomic conditions may have been an important predictor for later health in historical periods, the association may have weakened over time as nutrition has become more plentiful and exposure to disease less frequent and severe.

Despite the inconsistencies with some of the previous research, the findings of this study have implications for understanding mortality declines and old-age mortality. This study did not find important links between cohorts' early and old-age mortality, but did find strong period effects on mortality rates. This indicates that the majority of cohort differences in mortality are explained by factors other than early-life conditions, and that declines, or more generally changes, in cohort-level old-age mortality may be driven by changes in period conditions rather than changes in early-life conditions. While a full discussion of these period factors is beyond the scope of this study, I briefly discuss some of the potentially more influential factors. As discussed in Vallin and Meslé (2009), mortality started to decline in the late eighteenth to early nineteenth centuries. This change coincided with rapid developments in agricultural production and the adoption of Jenner's smallpox vaccine. In the nineteenth century, continued improvements in nutrition and sanitation, and the revolution in the understanding of the nature and diffusion of disease (the "germ theory" of disease), were crucial period-based factors driving mortality change. In the late nineteenth to early twentieth centuries the spread of clean water technologies decreased mortality in a largely period-based manner (Cutler and Miller 2005), and in the latter half of the twentieth century decreases in smoking (Preston and Wang 2006) and development of effective medical technologies, especially in prevention and treatment of cardiovascular diseases (Vallin and Meslé 2004), have further promoted mortality decline.

The results presented here should not be interpreted as refuting the several well-designed studies that have found evidence for cohort patterns in mortality or of long-lasting negative effects of adverse conditions early in life. For example, respiratory tuberculosis acquired in early years can be latent in an individual for many years, giving rise to lagged effects from early to late life (Des Prez and Goodwin 1985); differences in cohort smoking patterns produce cohort differences in mortality (Preston and Wang 2006); and persons exposed to the 1918 influenza pandemic in utero have excess heart disease prevalence when compared to the surrounding cohorts (Mazumder et al. 2010). In fact, the findings of the current study, showing small scarring effects for individuals born at times of high infant mortality, are consistent with the literature suggesting that old-age mortality is partially dependent on early-life conditions. This study, however, puts the magnitude of early-life effects into sharper focus and compares them to period effects. In this comparison, the importance of early-life conditions on later-life mortality seems modest. The variation in mortality rates is driven almost exclusively by period conditions.

Appendix: Model details

The model that I use to estimate the cohort coefficients β_0^c and β_{1-4}^c and the period coefficients β_{0-4}^p for each age $x = 5, 6, \ldots, 89$ is of the form

$$Y_x = \boldsymbol{\beta}_x' \mathbf{X}_x + \boldsymbol{\gamma}' \mathbf{Controls} + \varepsilon_x,$$

where Y_x is the dependent variable (relative deviation from trend in mortality at age x); $\mathbf{X}_x$ is the vector of cohort and period variables (cohort variables: relative deviations from trend in cohort mortality at ages 0 and 1–4; period variable: relative deviation from trend in period mortality at ages 0–4); **Controls** is a vector with dummies that capture the effects of the most exceptional period shocks of the observation period—World Wars I and II (1914–1918 and 1939–1945) and the 1918 influenza epidemic. I control for these exceptional period shocks as they might otherwise dominate the period effects. Without these controls, the period effects would be slightly larger. The model does not include intercepts as all de-trended variables have a zero mean. I estimate the model using ordinary least squares.

The model, when estimated for both sexes and by sex, produces 3,825 estimates, 85(age) x 4(2 cohort + 1 period coefficient) x 3(both sexes, men, women) x 5(countries). I summarize the estimates graphically, plotting exponentiated coefficients $\exp(\beta)$ against age. The coefficients are standardized to reflect a one standard deviation increase in the independent variables. An exponentiated coefficient for cohort shock at age 0 indicates how much, in proportional terms, the cohort's mortality at a given age increases or decreases for a one standard deviation shock in log mortality at age 0. The coefficients for cohort shocks at ages 1–4 and for period shocks at ages 0–4 are interpreted analogously.

To study the robustness of the results observed with the chosen model, I estimated several alternative model specifications. My model includes cohort shocks at ages 0 and 1–4 and period shocks at ages 0–4. With this specification the target parameter vector is $\beta_x = (\beta_0^c, \beta_{1-4}^c, \beta_{0-4}^p)$, where age subscripts x are not shown for the individual coefficients for readability. Shocks in infant and child mortality, however, may be so highly correlated that simultaneous estimation of the effects β_0^c and β_{1-4}^c is inaccurate. Therefore I estimated two complementary alternative models in which the two cohort coefficients were estimated separately, not simultaneously. The results were only marginally different from those obtained with the chosen model. In addition, I estimated models without control for period effects. Again, the results changed only marginally. Therefore, only results for the model specified above are shown.

To ensure that the results are robust to how the cohort and period shocks were defined, I estimated the effects using alternative definitions of cohort and period shocks. First, I replaced the cohort shocks by the deviation from trend in the Lee–Carter mortality index k (Lee and Carter 1992) at the year of birth. The results did not change in any significant manner. Second, I tried alternative specifications of the period effect. When estimating the period effects for cohort c at age x, I used the surrounding cohort's mortality at neighboring ages as the basis for the period shock. More specifically, I used two older and two younger cohorts as the surrounding cohorts. Thus for cohort c at age x, I based the period shock on the combined mortality rate of cohorts $c + x - 2$, $c + x - 1$, $c + x + 1$, and $c + x + 2$ at ages $x + 2$, $x + 1$, $x - 1$, and $x - 2$, respectively. The results for period effects were similar to what was observed

with my model. In addition, I used the Lee–Carter mortality index *k* as the basis for period shocks (matched with the year and cohort). Again, the results changed only marginally when compared to the chosen model.

Notes

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

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1 Research attempting to simultaneously estimate age, period, and cohort effects can be seen as an exception. However, I do not discuss the Age-Period-Cohort (APC) approach. APC models are well suited for describing mortality over the three dimensions of age, period, and cohort, but not for analyzing the particular aspects of factors associated with period or cohort that produce the effects.

2 The mechanisms presented here are closely related to the typology in Preston, Hill, and Drevenstedt (1998). They identified four mechanisms that relate the risk of death in childhood to the risk of death in adulthood; these are (1) positive and direct, (2) positive and indirect, (3) negative and direct, and (4) negative and indirect. In the typology of the current study, selection corresponds to (4), scarring to (1), and acquired immunity to (3). High risk of death early and late in life may also be indirectly related to (2) through "correlated environments," so that better access to education and health care in childhood results in higher adult socioeconomic status and lower adult mortality. This study uses data aggregated at the national level, so such an effect is intractable.

3 The magnitude of a one-SD shock depends on whether one looks at relative or absolute deviations from trend. In absolute terms, a one standard deviation increase in mortality would be largest for infant mortality. In relative terms, however, this is not the case as the changes are scaled to the mortality level.

4 Here only the lowess curves are shown, not single-age coefficients. Including the coefficients for every age would not change the results, but would make it very difficult to interpret the results.

5 For Sweden, the effect on life expectancy at ages 5–89 of a three-SD shock in infant mortality is –0.52 months; for England and Wales, –0.78 months.

6 The Human Mortality Database indicates that there was stalling or potentially even a decrease in life expectancy for these cohorts in England and Wales.

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