The effects of cohort and period mortality shocks on mortality at different ages

Mikko Myrskylä[1]

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Abstract

I study how mortality at a given age depends on cohort and period mortality shocks. I define cohort mortality shocks as deviations from trend in cohort's own infant and early childhood mortality. Period shocks are defined as deviations from trend in early childhood mortality during the period under consideration. Using annual cohort data for five European countries I find that cohort mortality shocks are essentially unrelated to later mortality. Period shocks are strongly correlated with mortality at all ages, but at older ages the association grows weaker. The results are similar for men and women, and suggest that cohort's early life experiences may have only little influence on cohort's later mortality and that period conditions dominate variation in mortality.

[1] Population Studies Center, University of Pennsylvania, 3718 Locust Walk, Philadelphia, PA 19104, USA. Email myrskylm@pop.upenn.edu. I thank Samuel H. Preston, Hiram Beltrán Sánchez, Janice Madden, Neil K. Mehta, Douglas Ewbank, James Oeppen, Beth Soldo, James W. Vaupel, John R. Wilmoth and participants of the demographic research seminar at the University of Pennsylvania for their comments.

1 Introduction

Understanding the relative importance of cohort's early life experiences and period conditions to mortality is crucial for understanding the historical mortality decline and for predicting future developments in mortality. Prior research suggests that early life conditions may be linked to adult mortality (Elo and Preston 1998), but separating these effects from the effects of period conditions is challenging. Consequently, there is an ongoing debate on whether mortality at older ages is mainly determined by cohorts' early life experiences or period conditions (Barker 1995, Finch and Crimmins 2004; Barbi and Vaupel 2005; Costa and Lahey 2005; Catalano and Bruckner 2006).

Cohorts born within a short period of time may have quite different mortality rates at a given age. The important question is what explains these cohort differences. The theory of fetal and infant origins of adult disease (Barker 1992, 1994; Eriksson et al. 1999; Barker et al. 2002) predicts that the differences may, at least partly, be explained by cohorts' early life experiences, such as quality of nutrition (Fogel 2004; Sparén 2003) or exposure to disease in infancy and early childhood (Bengtsson and Lindström 2000, 2003; Almond 2006). Some recent research, however, suggests that period conditions may play a larger role than cohort experiences in determining mortality (Barbi and Vaupel 2005).

In this paper I study how variation in mortality at a given age depends on cohorts' early life conditions and on period conditions using historical mortality time series for Denmark, England and Wales, Finland, Netherlands and Sweden. I use cohort mortality shocks, defined as deviations from trend in cohort's mortality at ages 0 and 1-4, as proxies for early life conditions. I use period mortality shocks, defined as deviations from trend period mortality at ages 0-4 and in surrounding cohorts' period mortality, as the measure for period conditions. As the dependent variable I use the deviation from trend in mortality over cohorts for a given age.

In previous research three approaches have been used to estimate what are called cohort and period effects: i) the age-period-cohort (APC) methodology; ii) an approach to evaluating the effect of one single shock such as famine or epidemic; iii) an approach in which mortality rates are compared across and within cohorts over long periods of time, with no consideration of specific shocks. These three approaches answer essentially different questions.

The APC approach, pioneered by Mason et al. (1973) and further developed by Glenn (1978), Fienberg and Mason (1978, 1985), Caselli and Capocaccia (1989) and Wilmoth (1990), simultaneously estimates age, period and cohort effects. This approach, however, cannot analyze the particular aspects of factors associated with period or cohort which *produce* the effects. This is because the focus is on the effects, not on the causes. Thus with the APC approach one can describe the mortality surface over the three dimensions of age, period and cohort, but it is more difficult to use this approach to study how mortality at a certain age is related to early life experiences.[2]

Another approach explicitly identifies a cohort mortality shock and compares the mortality of a cohort subject to the shock to the mortality of surrounding cohorts not marked by this event. Such shocks may be famines or disease epidemics. Studies on 1866-68 Finnish famine (Kannisto et al. 1997) and 1944-45 Dutch famine (Painter et al. 2005) have found no association between old-age mortality and nutrition early in life. The survivors of the 1941-42 Leningrad siege may have had elevated cardiovascular disease mortality (Sparén 2003), but the results are potentially confounded by differentials in surviving probabilities (Croft 2004; Bell 2004). Results on the effects of exposure to disease early in life are more robust: Bengtsson and Lindström (2000, 2003) have found strong evidence that disease load during the first year of life is associated with higher mortality later in life, and Almond (2006) finds elevated

[2] The APC approach also suffers from an identification problem: age, period, and cohort are linearly dependent, so the three effects can not be identified in a regression model. One must pose identifying restrictions on the parameters, but the results are very sensitive to these restrictions (e.g. Yang Yang 2004, Smith 2004). Recently there have been attempts to find less sensitive APC estimators, most notably the intrinsic estimator (Fu 2000; Knight and Fu 2000; Fu, Hall and Rohan 2004; for applications see Yang 2004, 2008). But also in this approach the focus is in the effects, not in the causes.

disability rates for those *in utero* during the 1918 influenza. These studies provide valuable information on the effects of single shocks and may indicate that diseases matter more than nutrition (Fogel (2004), however, attributes much of the declines in old age mortality to improved nutrition). The analysis of rare or unique events in history, however, is not well suited to comparing the influence of period and cohort, and can not tell us whether cohorts who experience high mortality early in life are, on average, suffering higher or lower mortality later in life.

The third approach, which is used in this study, compares mortality rates across and within cohorts over long periods of time. The approach has a long history, dating back to at least Kermack et al. (1934). Finch and Crimmins (2004) applied this methodology to compare Swedish cohorts born between 1751 and 1927, and concluded that mortality declines among the old and young begin in the same cohort, and attributed declines in old-age mortality largely to reduced exposure to inflammation in early childhood. The conclusions were not based on a statistical analysis of the data, however, but on a visual inspection of graphs with highly aggregated data. Barbi and Vaupel (2005) compared mortality rate correlations both within and across cohorts for the same data, and found that both correlations are positive, large, and highly significant, and that within-cohort correlations (cohort effects) are smaller. Moreover, the within-cohort correlation between infant and old age mortality was lowest when infant mortality was high, which is contradictory with the hypothesis that high infant mortality would lead to higher late life mortality. These correlations, however, may be spurious since both the dependent and independent variables are subject to trend.[3]

In more recent papers Finch and Crimmins (Finch and Crimmins 2005; Crimmins and Finch 2006a) study the absolute and relative effects of cohort and period mortality by regressing mortality at ages 70-74 on each cohort's earlier mortality up to age 15 and on period mortality. Using data for four European countries, they find that a cohort's earlier mortality explains 87 to 96 % of mortality variation at

[3] Regressing a variable with trend on another variable with trend may yield statistically significant results, irrespective of the nature of the variables. This is because the assumption of homoscedastic, zero-mean uncorrelated residuals is violated. For a discussion on this topic see Hendry (1980).

ages 70-74, and contemporary mortality at ages 0-15 explains only 7 to 68 % of the variation. Crimmins and Finch also find that the cohort variables explain more of the mortality variation for women than for men, indicating that men are more robust to exposure to infection in early life. These studies suffer from two statistical problems. First, all the mortality variables have a downward trend, so the results may be spurious.[4] Second, even without the spuriosity problem arising from trends, the estimates for childhood mortality variables (measured at ages 0, 1-4, 5-9 and 10-14) could be misleading due to the high collinearity between these variables.

To avoid spurious results, it is important to de-trend the variables before analysis. De-trending also significantly reduces the multicollinearity problem. Catalano and Bruckner (2006) focused on cohort effects and used ARIMA models (Box and Jenkins 1970) in a study which compared the *unexpected* components in the odds of dying before age 5 and in later mortality, as summarized by life expectancy at age 5. Catalano and Bruckner found that higher than expected childhood mortality is correlated with lower than expected life expectancy at age 5, and that the association is stronger for men than for women. The effects, however, are small: the highest and lowest recorded childhood mortality rates would result in 1.75 years of difference in life expectancy for men and 1.56 for women (the authors do not report the effects for one or two standard deviation shocks).

Catalano and Bruckner do not separate infant and early childhood effects, although exposure to adverse conditions at ages 0 or 1-4 could have different effects. As they measure life expectancy at 5, we do not know whether the observed correlations mean that high childhood mortality increases mortality over the whole life course, late in life, or at ages close to 5. Other problems in the study are that first, life expectancies are used for cohorts born as late as 1913. This means that for some countries up to 30% of the dependent variable is based on forecasts, and the uncertainty and potential bias arising from forecasts is not taken into account. Second, before modeling the relationship between early life and late life mortality the dependent variable is differenced and pre-whitened with ARMA models with autoregressive

[4] See the footnote 3 above.

lags up to 10 and moving average lags up to 6. Extensive pre-whitening with complicated ARMA models makes it challenging to understand what exactly are the residuals that enter the regression analysis. Given the AR lags up to 10, it is also difficult to know what do the signs in residuals mean. The authors also use ARMA models in the final stage of their analysis, where the pre-whitened residuals in life expectancy are regressed on the pre-whitened residuals from the odds of dying before age 5 and autocorrelation is allowed up to lag 10, moving average lag up to 8. At this stage unclear whether negative signs in the coefficients indicate positive or negative correlation with high childhood mortality and later life expectancy.

To summarize, robust results about the relative importance of cohort and period mortality shocks on later mortality are rare. The goal of this paper is to study

1) what are the absolute and relative contributions of cohort and period mortality shocks on later mortality at different ages,
2) whether cohort shocks at infancy and early childhood have different effects, and
3) whether men and women respond differently to cohort and period shocks.

I use a decomposition, breaking observed series into two unobserved components, trend over cohorts and residual. The residual in mortality at a given age is the dependent variable. Residuals in period mortality at ages 0-4 and in surrounding cohorts' mortality are the period shocks, and residuals in cohort's early life mortality are the cohort shocks. The design removes all trends and allows me to answer the question "If a cohort had higher/lower mortality than we would expect based on the surrounding cohorts, how well does cohort's mortality early in life explain this, and how well does period mortality explain this?".

2 Anticipated influence of cohort and period shocks

I aim to estimate both the direction and magnitude of the effects of cohort and period mortality shocks. The direction of the effect of a period mortality shock is likely to be positive: a shock that increases mortality at one age is bound to increase mortality also at other ages at the same period. The influence of cohort shocks, however, can be both positive and negative. This is because mortality shocks in early life may affect later mortality by selection, scarring or induced immunity.[5] This section makes explicit the predictions of these three pathways.

First, a cohort mortality shock may act selectively, killing the weakest (I consider cohort mortality shocks at ages below 5, so it is unlikely that any shock would kill selectively the more robust ones). An example of such a shock could be famine, disease epidemic, or war. The influence of a selective mortality shock on later mortality would be positive, lowering mortality. Second, a cohort mortality shock may damage the surviving population, resulting in a scarring effect which increases the mortality of the surviving cohort. Third, a cohort mortality shock may induce immunity, lowering the mortality for the surviving cohort.

To summarize, conceptually cohort mortality shocks may either increase or decrease mortality later in life. Moreover, it is not clear at which ages mortality would be altered by an early life shock, or whether the cohort shocks are more important than period shocks. Only empirical evidence, then, can tell us the relative importance of cohort and period shocks, direction of the effects, and whether selection, scarring or immunity dominates the cohort effect.

[5] Preston et al. (1998) have a typology presenting four mechanisms that relate the risk of death in childhood and risk of death in adulthood; these are (1) positive and direct, (2) positive and indirect, (3) negative and direct, and (4) negative and indirect developed. In the typology of Table 1, selection/culling corresponds to (4), to Scarring to (1) and acquired immunity to (3). High risks of death early and late in life may be also indirectly related (2), through "correlated environments" so that better access to education and health care in childhood results in higher adult SES and lower adult mortality. This study uses data aggregated on national level, so such an effect is intractable.

3 Data, variables and methods

3.1 Data and variables

I use time series data on 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 (1750-1915). The data quality for these countries is comparatively good. The data source is the Human Mortality Database (University of California, Berkeley, and Max Planck Institute for Demographic Research 2007).

The dependent variable is derived from $m(x,t-x)$, mortality rate at age *x* at time *t* for cohort *t-x*. Cohort mortality shocks are derived from cohort's own mortality rates at ages 0 and 1-4, denoted by $m(0,t-x)$ and $m(1-4,t-x)$. Two period mortality shocks are considered; the first is derived from period mortality at ages 0-4; the second is derived from mortality rates of cohorts surrounding the cohort *t-x*. I use two older and two younger cohorts as the surrounding cohorts. Thus for cohort *t-x* at age *x* I use the combined mortality rate of cohorts $t-x-2$, $t-x-1$, $t-x+1$, and $t-x+2$ at ages $x+2$, $x+1$, $x-1$, and $x-2$, respectively.

3.2 Methods

All the time series considered in this study are subject to downward trend: mortality is and has been decreasing at all age groups in the countries that are studied. When studying variables subject to trend the variables should be de-trended before conducting a time series analysis. The reason for de-trending is that regressing one variable with a trend on another variable with a trend may yield statistically significant results, irrespective of the true nature of connections between the variables. This is because the fundamental assumptions of regression analysis (uncorrelated zero mean residuals with constant variance) may not hold. As a consequence, meaningless regressions with unrelated variables may deliver spurious results, and meaningful regressions are difficult to distinguish from meaningless ones (Hendry 1980).

Moreover, the problem of multicollinearity is markedly reduced if trends are removed. Therefore I de-trend the series before analysis.

Before de-trending, I transform all the time series to log scale. I decompose the log of the mortality time series into trend and deviation from trend, and model the deviations from trend. I de-trend each of the series *over cohorts* using the Hodrick-Prescott filter (Hodrick and Prescott 1997) with smoothing parameter $\lambda = 100$, a standard choice for annual data (Maravall and del Río 2007).[6] Figure 1 shows the variables (original series, estimated trend and residuals) for Sweden for selected ages.

FIGURE 1 ABOUT HERE

The outcome variables, shown in Panels 1-2 of Figure 1, are deviations from trend in log mortality at ages 20 and 80; in the analyses I use all ages from 5 to 89. The Panels 3 and 4 show shocks in cohorts' mortality rates at ages 0 and 1-4, and Panels 5 and 6 show the period mortality shocks (Panel 6 is for the surrounding cohort mortality for age group 20). .

Before regressing the outcome variable on the predictors, I standardize the cohort and period mortality shocks. Standardization is done because the magnitude of different types of shocks varies significantly. Figure 2 illustrates the estimated regression model.

FIGURE 2 ABOUT HERE

The model depicted in Figure 2 is estimated separately for each age $x = 5, 6, ..., 89$. That means that I will estimate 85 coefficients for infant mortality shocks ($\beta^{c}_{0,x}$), early childhood mortality shocks ($\beta^{c}_{1-4,x}$), period mortality shocks measured at ages 0-4 ($\beta^{p}_{0-4,x}$) and period mortality shocks measured from the surrounding cohorts ($\beta^{p}_{S,x}$). If cohort mortality shocks were positively correlated with later mortality

[6] Two other smoothing parameters were also analyzed: $\lambda = 6.25$, suggested by Ravn and Uhlig (2002) and $\lambda = 1600$, often used for quarterly data. The larger the λ less there is smoothing, so the estimated residuals did change when λ was changed, but the estimates for the model parameters were essentially unchanged.

($\beta^c_{0,x}, \beta^c_{1-4,x} > 0$) for all or most ages *x*, then the scarring effect must dominate the selection and immunity effects. If mortality shocks were negatively correlated with later life expectancy ($\beta^c_{0,x}, \beta^c_{1-4,x} < 0$) for all or most ages *x*, then those who survive would be on average less frail than those who die as a result of the mortality shocks. Finally, if the magnitude of the cohort coefficients is larger than the magnitude of the period coefficients, early life experiences may be more important in determining mortality at a given age than period conditions.

I estimate the coefficients $\beta^c_{0,x}, \beta^c_{1-4,x}, \beta^p_{0-4,x}, \beta^p_{S,x}$ using four different models, all of which are of the general form

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

where Y_x is the dependent variable (deviation from trend in log mortality at age *x*) and $\mathbf{X}_x$ is the vector of predictors. The first model includes cohort mortality shocks at ages 0 and 1-4 and period mortality shocks at ages 0-4, so $\boldsymbol{\beta}_x = (\beta^c_{0,x}, \beta^c_{1-4,x}, \beta^p_{0-4,x})$. The second model replaces the period mortality shock at ages 0-4 by period mortality shock in the cohorts surrounding age *x* at time *t*, so $\boldsymbol{\beta}_x = (\beta^c_{0,x}, \beta^c_{1-4,x}, \beta^p_{S,x})$. The third and fourth model estimate separately the cohort coefficients while controlling for the period coefficient. For the third model $\boldsymbol{\beta}_x = (\beta^c_{0,x}, \beta^p_{0-4,x})$, and for the fourth model $\boldsymbol{\beta}_x = (\beta^c_{1-4,x}, \beta^p_{0-4,x})$. Models 3 and 4 were considered because infant and early childhood mortality shocks may be so correlated that joint estimation of the effects $\beta^c_{0,x}, \beta^c_{1-4,x}$ could be inaccurate. The effects, however, changed from the effects obtained in Model 1 only marginally. Therefore results from Models 3 and 4 are not reported. The cohort coefficients $\beta^c_{0,x}, \beta^c_{1-4,x}$ in Models 1 and 2 were also essentially the same. Therefore I will report the full results for Model 1, and period coefficient results for Model 2.

The models are estimated separately for both genders and for men and women. Autocorrelation in the residual is allowed up to lag 3 (that is $\varepsilon_x \sim AR(p)$ with $p \leq 3$). The optimal lag was chosen using the generalized Durbin-Watson test.

The models produce 5100 estimates, 85(age) x 4(2 cohort + 2 period coefficients) x 3(both genders, men, women) x 5(countries). I summarize the estimates graphically, plotting exponentiated coefficients $\exp(\beta)$ against age. An exponentiated coefficient for, say, cohort mortality shock at age 0 tells us how much, on average, mortality at a given age increases/decreases for a one standard deviation shock in log mortality at age 0. The coefficients for cohort mortality shocks at ages 1-4 and period mortality shocks at ages 0-4 and at surrounding cohorts are interpreted analogously.

4 Results

The results are in three sections. Section 4.1 shows the overall results for Model 1 (cohort mortality shocks at ages 0 and 1-4 and period mortality shock at ages 0-4) by country. Section 4.2 shows the results for Model 1 and Model 2 by mortality shock. Section 4.3 focuses on gender differences.

4.1 The effects of cohort and period mortality shocks by country; both genders

Figure 3 shows the effects of cohort mortality shocks at ages 0 and 1-4 and period mortality shocks at ages 0-4 on mortality. The dots in the graph show exponentiated coefficients from the Model 1; the smoothed lines are locally weighted smoothed regression curves. The interpretation for the dots and lines is the following. An estimate, say, 1.03 for age 20 and cohort mortality shock at age 0 ($1.03 = \exp(\beta^{c}_{0,20})$) means that one standard deviation shock in cohort's log mortality at age 0 is associated with a 3% increase in the same cohort's mortality at age 20. Coefficients for cohort mortality shocks at ages 1-4 are interpreted analogously. For the period mortality shocks an estimate, say, 1.05 for age 20 ($1.05 = \exp(\beta^{p}_{0-4,20})$) means that on average, one standard deviation period mortality shock at ages 0-4 is associated with a 5% increase in period mortality at age 20.

FIGURE 3 ABOUT HERE

Let us first consider the period shock coefficients (black dots/lines). These are generally above 1, which is expected: if mortality increases at young ages, it is likely that it increases also at other ages. The graphs also show that i) the link between period mortality at ages 0-4 and mortality at older ages is strongest at younger ages; and ii) all the countries show a similar pattern, but for England and Wales the link between period mortality at ages 0-4 and mortality at older ages is weaker than in other countries.

The period mortality shock coefficients are large compared to the cohort shock coefficients. Some individual cohort shock coefficients may be as high as 1.03-1.04, but the smoothed regression curve shows that overall, both cohort mortality shocks have only little if any effect on later mortality. In all countries, the effect of infant mortality shock (red dots/line) are close to 1 at all ages. The effect of early childhood mortality shock (blue dots/line) also has very little effect on later mortality at all ages, except perhaps at ages 5-15. At these ages, the effect seems to be below 1, indicating that if cohort had a higher than expected mortality at ages 1-4, it will have lower than expected mortality at ages 5-15. This result is surprising but not inconceivable, and can be due to selection or acquired immunity: For all cohorts, mortality is generally very low at ages 5-15. Thus those few who die at these ages are likely to be very frail (wartimes, and accident mortality, may constitute exceptions). If mortality at ages 1-4 happens to be higher than on average, then it is plausible that this mortality shock affects especially those who otherwise would have lived to age 5 but still died at relatively young ages. If this was true, the selection and culling effect would explain the observed association between mortality at ages 1-4 and 5-15. It should be noted, however, that the finding is weak, and does not hold in England and Wales.

4.2 The effects of cohort and period mortality shocks by type of shock; both genders

Figure 4 shows the smoothed effects of cohort and period shocks on mortality by type of shock. The results are grouped so that panel 1 of Figure 4 shows the effects of infant mortality cohort shocks, panel 2 shows the effects of early childhood mortality cohort shocks, panel 3 is for period mortality shocks at ages 0-4 and panel 4 is for period shocks in the surrounding cohorts.

FIGURE 4 ABOUT HERE

Panels 1 and 2 confirm what was observed in Figure 3. Cohort mortality shocks at age 0 have no influence on later mortality. Cohort mortality shocks at ages 1-4 may decrease mortality at ages 5-15, but the effect is small (for one standard deviation increase in log mortality at ages 1-4, mortality at ages 5-15 may be approximately 1% lower). Moreover, England and Wales is an outlier in this respect, showing no

decreased mortality at ages 5-15 but possibly slightly increased mortality for cohort mortality shocks at ages 1-4.

Panels 3 and 4 show the effects of period mortality shocks. All countries show a similar patter: High period mortality at ages 0-4 is correlated with high period mortality at older ages, but the older the age group, the weaker the association (panel 3). For England and Wales the association is the weakest. Panel 4 shows results from a model where period mortality shocks were derived from the mortality of surrounding cohorts. For these period shocks, the general picture is the same: at older ages the association between mortality at a given age and period mortality shock decreases, but overall the association is strong and positive.

4.3 Do men and women respond differently to mortality shocks?

Figure 5 shows the same curves as Figure 4 but broken down by gender. Overall, the results for men and women are very similar. Panels 1 and 2, which show the effect of cohort mortality shocks at ages 0 and 1-4, do not reveal any differences between men and women. The same holds for panel 3, which shows the effect of period mortality shocks at ages 0-4: in each country, the estimated curves are close to each other for men and women.

FIGURE 5 ABOUT HERE

Panel 4, which shows the effects of period shocks derived from the mortality rates of surrounding cohorts, there seems to be some differences between men and women: the coefficients for men seem to be stronger especially at ages 15-35. Keeping in mind how the period shocks were constructed in panel 4, this might reflect wartime mortality: during war, mortality is especially high for men aged 15-35, and the unexpectedly high mortality generally applies to all ages in this range.

5 Discussion

Cohorts born within a short time period may have quite different mortality rates at a given age. These differences are cohort differences, and may potentionally be explained by differentials in early life conditions, or by period fluctuations. This study analyzes how mortality at a given age depends on cohorts' early life experiences and on period mortality conditions. Using historical data on mortality for Denmark, England and Wales, Finland, Netherlands and Sweden I find that shocks in infant mortality are weakly, if at all, associated with later mortality. Mortality shocks at ages 1-4 are inversely associated with cohort's mortality at ages 5-15, but the effect is not particularly strong..

Period mortality shocks have much stronger influence on mortality at any given age. I considered two alternative ways to define period mortality shocks; the first was based on period mortality at ages 0-4 and the second on period mortality at surrounding cohorts. The results were qualitatively the same for both formulations: period mortality shocks are positively and strongly correlated with mortality at any age between 5 and 89 years, but the association is weaker for older ages. This suggests that at older ages the proportion of purely random variation (variation not explained by cohort or period conditions) increases.

The effects of cohort mortality shocks were similar for men and women. The period mortality shocks derived from mortality at ages 0-4 also had similar effects for men and women, but the period shocks derived from the mortality of surrounding cohorts had larger effect on men aged 15-35 than on women of similar age. This may be explained by wartime mortality, when mortality is especially high for men aged 15-35, and the higher than normal mortality applies to all ages in this range.

These results of this study apply most clearly to four of the countries analyzed: Denmark, Finland, Netherlands and Sweden. England and Wales, however, is clearly an outlier, having much weaker link between period and mortality rates and showing no evidence of decreased mortality at ages 5-15 for cohorts which had 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 it is for the other countries, and the methodology used in Human Mortality Database to convert aggregate data to Lexis surfaces may construct these high-frequency cohort dependencies not observed for other countries. Second, the first world war and 1918 Spanish influenza increased mortality in England and Wales so that for cohorts born in 1880-1895 there was stalling and even decline in life expectancy. These events caused disturbances in the mortality dynamics and may have affected the results of this study. In previous research these disturbances have been well documented (e.g. Winter 1976). Derrick (1921; in Winter 1976) even claimed that "the effects of losses during the European War were so great and indefinite as to obscure all normal changes", including changes which might have resulted from early life cohort mortality shocks. Of course, the mortality dynamics may be inherently different in England and Wales than they are in other countries, but this seems unlikely.

The finding that high mortality at ages 1-4 may decrease mortality at ages 5-15 may be due to selection or acquired immunity during the mortality shock. For all cohorts, mortality is very low at ages 5-15. Thus those few who die at these ages are likely to be very frail (wartimes, and accident mortality, may constitute exceptions). If mortality at ages 1-4 happens to be higher than on average, then it is plausible that this mortality shock affects especially those who otherwise would have lived to age 5 but still died at relatively young ages. If this was true, the selection and culling effect would explain the observed association between mortality at ages 1-4 and 5-15. The other explanation, acquired immunity during the shock, is also possible, but in this study it was not possible to separate these two explanations.

This study found no significant differences in how men and women respond to early life mortality shocks. In previous research Catalano and Bruckner (2006) find some indication that women are more robust than men, but Crimmins and Finch (2006b) get the opposite result. The theories which would explain why one of the sexes would be more robust to harmful early life conditions are plentiful (Crimmins and Finch 2006b), but they are not consistent with each other. As the empirical evidence is equally inconsistent, the tentative conclusion is that there are no strong differences between the two sexes.

The results of this paper have important implications on how we should think about mortality declines and old-age mortality. Previous research has suggested that early life conditions may be more important than period conditions (Finch and Crimmins 2004), or that even if period conditions were more important, early life conditions could still play an important role in determining old-age mortality (Barbi and Vaupel 2005). This study did not find important links between old-age and early life mortality, but did find strong period dependencies in mortality rates. This indicates that declines, or more generally changes, in old-age mortality are driven by period conditions. It may be that the inconsistencies between these and earlier results are due to methodological differences, most importantly the differences in the way trends are handled. This study de-trended all the variables, thus losing information about the broad patterns of mortality declines but allowing statistically sound estimation of the effects of interest.

One earlier study which applied similar de-trending techniques but did not consider period influences (Catalano and Bruckner 2006) found that early life mortality shocks decrease later life expectancy. It is difficult to say what causes the differences between the results of this study and the study by Catalano and Bruckner (2006). Potential explanations could be that the high amount of imputed data in Catalano and Bruckner biases the results, or that the complexity of ARIMA models (the authors used AR-lags up to 10) distort the estimation. It is also worth noting the Catalano and Bruckner get their strongest results in England and Wales, which is exceptional in terms of its mortality dynamics (Winter 1976).

The results of this study – no increased old-age mortality for cohorts who had higher than expected infant and childhood mortality, and strong period dependencies in mortality rates – are consistent with the studies finding no increased mortality among those who survived great famines as young children (Kannisto et al. 1997, Painter et al. 2005). Other studies, however, have found that disease load *in utero* or during the first year of life is associated with higher disability and mortality later in life (Almond 2006; Bengtsson and Lindström 2000, 2003). These studies differ from the current one in one important way: both focus on epidemics and identify the timing of the epidemics very accurately, whereas in this study all mortality shocks were considered together and identified on an annual basis. In future studies about

cohort influences on old-age mortality it would be important to focus on monthly or even weekly cohorts, data permitting. Incorporating information about the quality of nutrition (or proxies for this, such as grain prices), disease patterns, and weather conditions might also be useful. Moreover, it would be important to consider other outcomes in addition to mortality, such as educational and occupational outcomes, marriage market outcomes, and fertility. Almond's (2006) research indicates that if early life or *in utero* conditions influence cohort mortality, these may be some of the pathways through which the effect is channeled.

References

Catalano R, Bruckner T 2006. Child mortality and cohort life span: a test of diminished entelechy. Int J Epidemioly;35:1264–69.

Barbi E, Vaupel J (2005). Comment on "Inflammatory Exposure and Historical Changes in Human Life-Spans(17 June 2005) *Science* 308 (5729), 1743a. [DOI: 10.1126/science.1108707]

Barker DJP, ed. Fetal and infant origins of adult disease. London: BMJ Books, 1992.

Barker D. J. P. (1994). Mothers, Babies, and Disease in Later Life. British Medical Journal Publishing Group, London.

Barker, D., Eriksson, J., Forsen, T, Osmond, C (2002). Fetal origins of adult disease: strength of effects and biological basis. Int J Epidemiol 31: 1235-1239 [Abstract] [Full text]

Bell C 2004. Long term mortality after starvation during the Leningrad siege: No evidence that starvation around puberty causes later cardiovascular disease BMJ 2004 328: 346. [Extract] [Full Text]

Bengtsson T and Martin Lindstrom (2000) Childhood Misery and Disease in Later Life: The Effects on Mortality in Old Age of Hazards Experienced in Early Life, Southern Sweden, 1760-1894. Population Studies Vol. 54, No. 3 (Nov., 2000), pp. 263-277

Bengtsson T. and M. Lindström (2003) "Airborne Infectious Diseases during Infancy, and Mortality in Later Life, Southern Sweden 1766-1894." *International Journal of Epidemiology* 32:2, 286-294

Box, George and Jenkins, Gwilym (1970) *Time series analysis: Forecasting and control*, San Francisco: Holden-Day

Caselli G, Capocaccia R. Age, period, cohort and early mortality: an analysis of adult mortality in Italy. Popul Stud. 1989;43:133–153.

Crimmins EM, Finch CE (2006a). Infection, inflammation, height, and longevity. Proc Natl Acad Sci USA;103:498–503.

Crimmins EM, C. E Finch (2006b) Commentary: Do older men and women gain equally from improving childhood conditions?. Int. J. Epidemiol. 35, 1270-1271

Croft MD 2004. Long term mortality after starvation during the Leningrad siege: Crucial confounding factor was overlooked. BMJ 2004 328: 346. [Extract] [Full Text]

Costa, DL and Lahey, JN (2005). Predicting older age mortality trends. Journal of the European Economic Association April–May 2005 3(2–3):487–493

Derrick VPA. Observations on (1) Errors of Age in the Population Statistics of England and Wales, and (2) the Changes in Mortality Indicated by the national records. Journal of the Institute Actuaries 1927;58:117–59.

Elo IT, Preston SH. Effects of early-life conditions on adult mortality: a review. Popul Index 1992;58:186–212.

Eriksson JG, Forsen T, Tuomilehto J, Winter PD, Osmond C, Barker DJ. Catch-up growth in childhood and death from coronary heart disease: longitudinal study. Bmj 1999;318:427-31

Fienberg, Stephan E., and William M. Mason. 1978. Identification and Estimation of Age-Period-Cohort Models in the Analysis of Discrete Archival Data. Pp. 1–67 in Sociological Methodology, Vol. 8.

Fienberg, Stephan E., and William M. Mason. 1985. Specification and Implementation of Age, Period, and Cohort Models. Pp. 45–88 in Cohort Analysis in Social Research, edited by William M. Mason and Stephan E. Fienberg. New York: Springer-Verlag.

Finch C, Crimmins E (2004) Inflammatory Exposure and Historical Changes in Human Life-Spans. (17 September 2004) *Science* 305 (5691), 1736. [DOI: 10.1126/science.1092556]

Finch CE, Crimmins EM (2005) Response to Comment on "Inflammatory Exposure and Historical Changes in Human Life-Spans". Science 308 (5729), 1743b. [DOI: 10.1126/science.1109017]

Fogel RW. The Escape from Hunger and Premature Death, 1700–2100: Europe, America, and the Third World. New York: Cambridge University Press, 2004.

Fu, Wenjiang J. 2000. Ridge Estimator in Singular Design with Application to Age-Period-Cohort Analysis of Disease Rates. Communications in Statistics—Theory and Method 29:263–78.

Fu WJ, Hall P y Rohan TE 2004. Age-Period-Cohort Analysis: Structure of estimators, estimability, sensitivity, and asymptotics In: http://www.stat.tamu.edu/~wfu/apc/apctrend1.pdf.

Glenn, Norval D. 1976. Cohort Analysts' Futile Quest: Statistical Attempts to Separate Age, Period, and Cohort Effects. American Sociological Review 41:900–904.

Granados JAT. Increasing mortality during the expansions of the US economy, 1900–1966. Int J Epidemiol 2005;34:1194–1202.[Abstract/Free Full Text]

Hendry D 1980. Econometrics-Alchemy or Science? Economica, New Series, Vol. 47, No. 188, pp. 387-406.

Hobcraft J, Menken J, Preston S 1982. Age, Period, and Cohort Effects in Demography: A Review. Population Index, Vol. 48, No. 1, pp. 4-43.

Hodrick R, Prescott E 1997. Postwar us business cycles: An empirical investigation. Journal of Money, Credit and Banking, 29, 1–16.

Human Mortality Database. University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at www.mortality.org or www.humanmortality.de (data downloaded on Dec 10, 2007).

Kannisto V, K. Christensen, J. W. Vaupel, Am. J. Epidemiol. 145, 987 (1997).[Abstract/Free Full Text]

Knight, Keith, and Wenjiang J. Fu. 2000. Asymptotics for Lasso-Type Estimations. Annals of Statistics 28:1356–78.

Maravall A and Ana del Río (2007) Temporal aggregation, systematic sampling, and the Hodrick-Prescott filter. Documents de Trabajo No. 0728, Bank of Spain.

Mason, Karen Oppenheim, William M. Mason, H. H. Winsborough, W. Kenneth Poole 1973. Some Methodological Issues in Cohort Analysis of Archival Data. *American Sociological Review*, 38:242-258.

Preston SH, Mark E. Hill and Greg L. Drevenstedt (1998) Childhood conditions that predict survival to advanced ages among African–Americans Social Science & Medicine Volume 47, Issue 9, November 1998, Pages 1231-1246

Ravn, M.O., Uhlig, H., 2002. On adjusting the Hodrick–Prescott filter for the frequency of observations. Review of Economics and Statistics 84, 371–376.

Sparén P, Vågerö D, Shestov DB, Plavinskaja S, Parfenova N, Hoptiar V, et al. Long term mortality after severe starvation during the siege of Leningrad: prospective cohort study. BMJ 2004;328: 11. (3 January.)[Abstract/Free Full Text]

Yang, Yang, Wenjiang J. Fu, and Kenneth C. Land. 2004. A Methodological Comparison of Age-Period-Cohort Models: Intrinsic Estimator and Conventional Generalized Linear Models. *Sociological Methodology* 34: 75-110.

Wilmoth, John R. 1990. Variation in Vital Rates by Age, Period, and Cohort. Pp. 295–335 Sociological Methodology Vol. 20.

Winter JM (1979) Some Aspects of the Demographic Consequences of the First World War in Britain. Population Studies > Vol. 30, No. 3 (Nov., 1976), pp. 539-552

Figure 1. Mortality at ages 20 and 80 (dependent variables), mortality at ages 0 and 1-4 (cohort mortality shocks) and period mortality at ages 0-4 and at cohorts surrounding age 20 (period mortality shocks). Source: Human Mortality Database

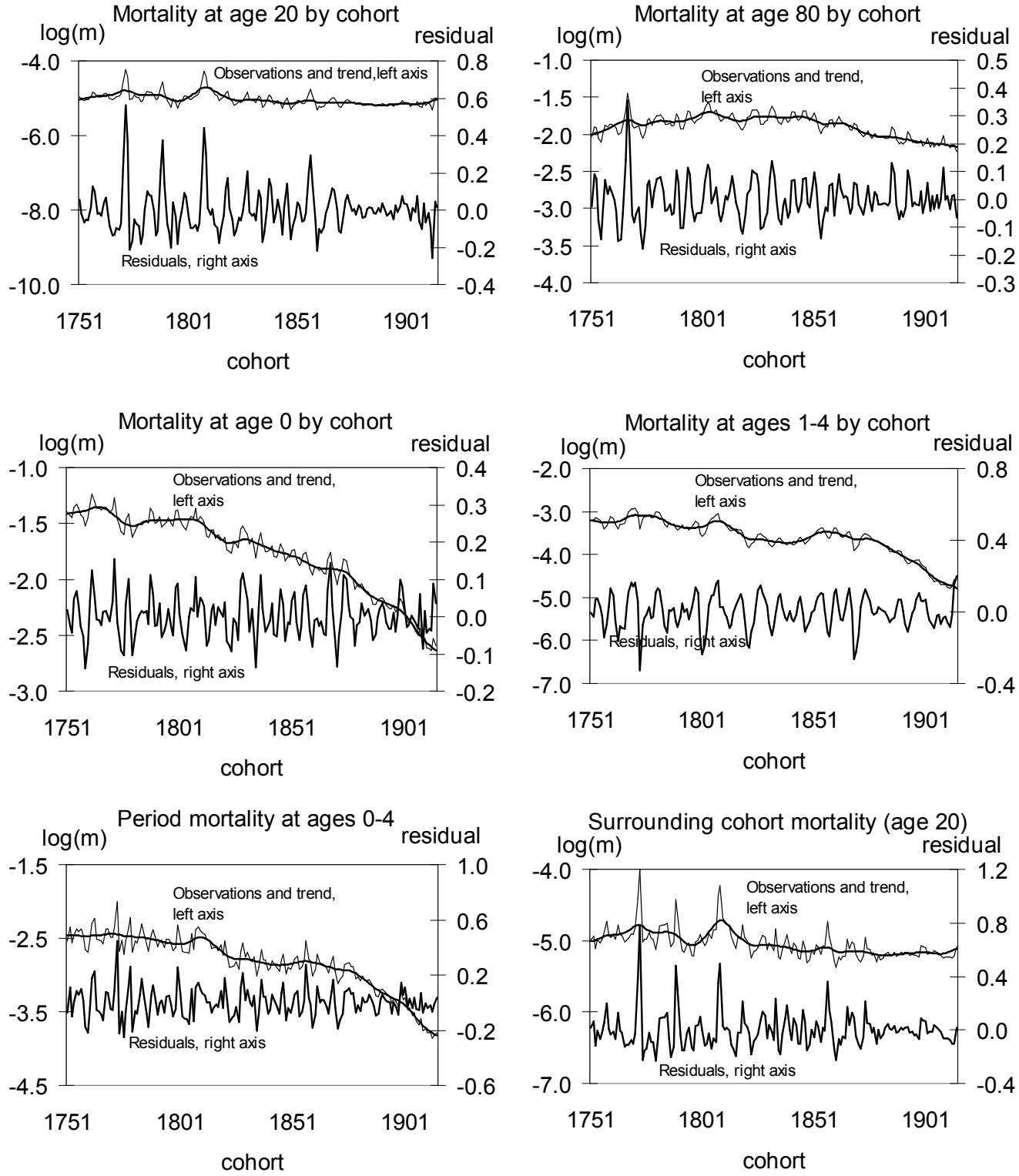

Figure 2. Illustration of the model.

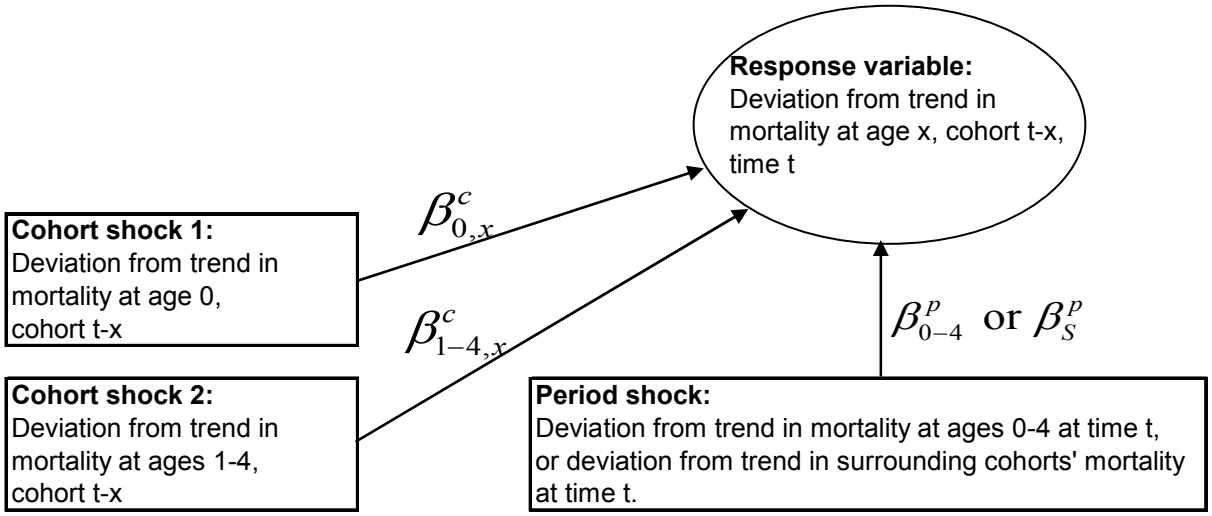

Figure 3. The effects of cohort and period shocks on mortality at ages 5-89 by country. Age on horizontal axis, effect (exponentiated coefficient) on horizontal axis.

Black line, black dots: The effect of a period mortality shock at ages 0-4
Red line, red dots: The effect of a cohort mortality shock at age 0
Blue line, blue dots: The effect of a cohort mortality shock at ages 1-4

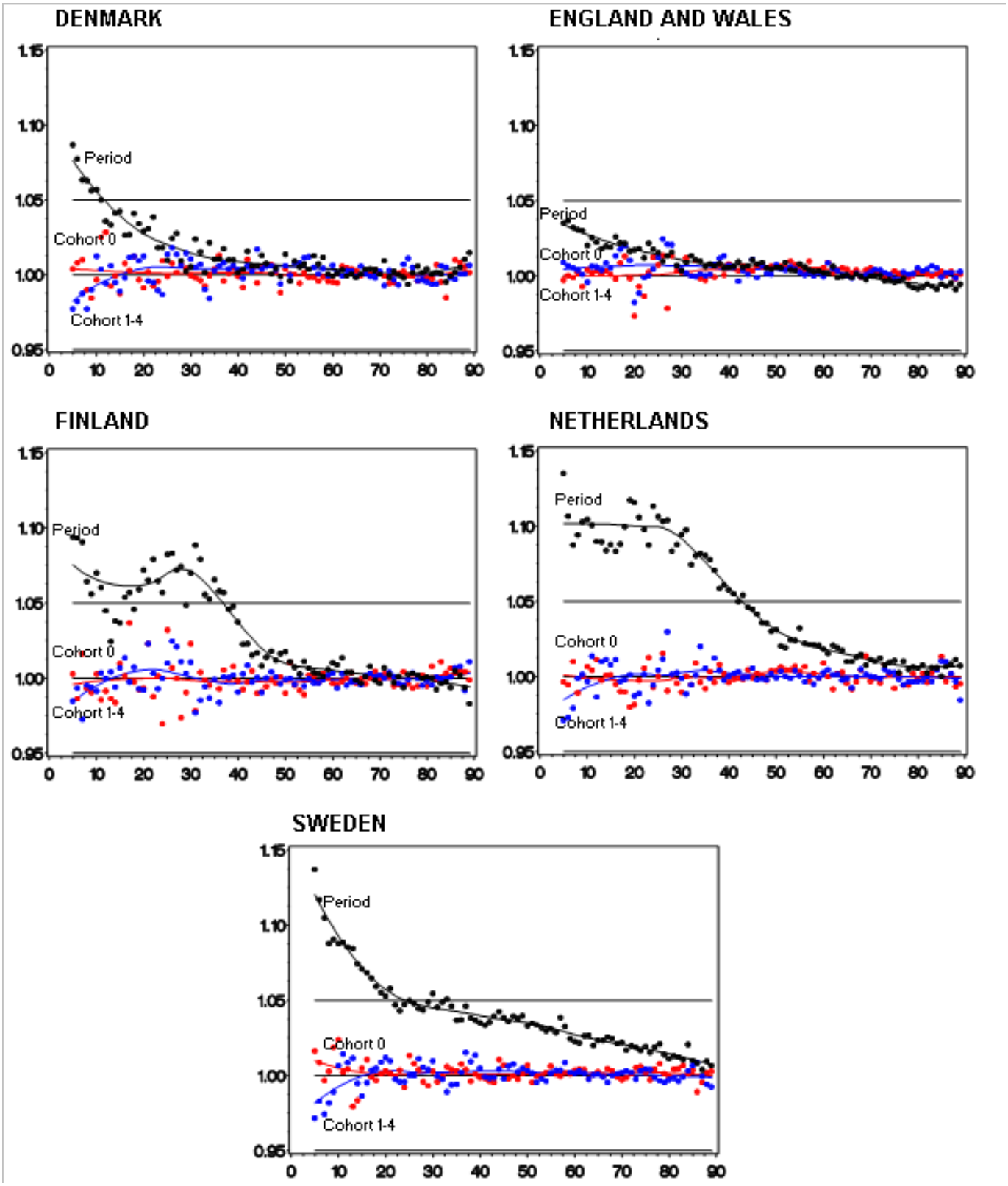

Figure 4. The effects of cohort and period shocks on mortality at ages 5-89 by type of shock. Age on horizontal axis, effect (exponentiated coefficient) on horizontal axis.

Red=Denmark Blue=England and Wales Black=Finland Brown=Netherlands Green=Sweden

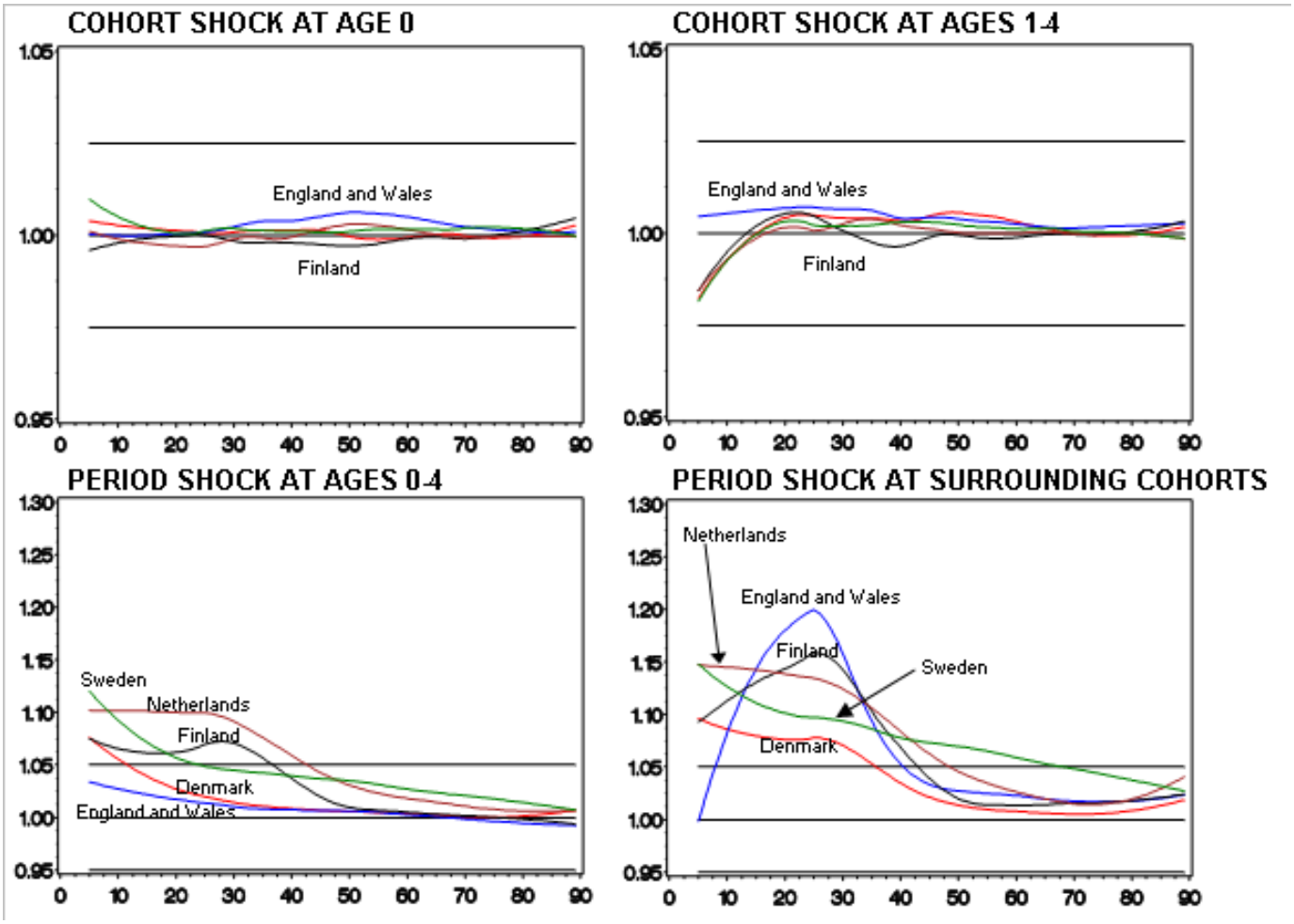

Figure 5. The effects of cohort and period shocks on mortality at ages 5-89 by type of shock and gender. Age on horizontal axis, effect (exponentiated coefficient) on horizontal axis.

Red=Women Blue=Men