How Did Mortality Selection Change the Future of the Past? Consequences of Mortality Selection on Cohort Trends in Life-Course Mortality Patterns and Epidemiologic Transition

Hui Zheng, Ohio State University

How Did Mortality Selection Change the Future of the Past? Consequences of Mortality Selection on Cohort Trends in Life-Course Mortality Patterns and Epidemiologic Transition

This paper proposes a conceptual framework to understand how the effects of mortality selection on cohort trends in life-course mortality pattern and epidemiologic transition may be shaped by different historical mechanisms for the decline in pandemics. Among early-transition countries, socioeconomic development suppressed pandemics and triggered a cohort evolution mechanism, yielding a moderate reduction in mortality selection at early ages that did not override cohort evolution mechanisms. In contrast, among later-transition countries, medical advancements did not trigger cohort evolution mechanisms, but instead generated a substantial reduction in mortality selection in early life. This made cohorts born later, during the age of receding pandemics, have higher mortality rates at early and middle adulthood but lower mortality rates in late adulthood, as compared to preceding cohorts. This phenomenon should produce a crossover of mortality rates across age groups over successive time periods; and, past the age of receding pandemics, stagnation followed by an increase in life expectancy. In later-transition countries, further declines in early-life mortality will be driven by socioeconomic development and will begin to resemble the trend among early-transition countries. Using the Human Mortality Database, we compare three epidemiologic transition models represented by Sweden, Japan, Poland and Bulgaria, and find support for this conceptual framework.

The theory of epidemiologic transition has been the dominant framework for explaining increasing life expectancy and changing patterns of health and disease over the last three centuries (Omran 1971; Olshansky and Ault 1986). This theory attributes mortality decline to a changing mix of socioeconomic development, lifestyle changes, and medical innovations within each period. In contrast, cohort evolution theories, such as the cohort morbidity phenotype theory (Finch and Crimmins 2004) and the technophysio evolution theory (Fogel and Costa 1997), propose that age-specific mortality rates are positively correlated across birth cohorts due to the long-lasting protective consequences of a better health endowment and lower exposure to infection and inflammation in early life. These mechanisms link older-age mortality declines in the later stage of the epidemiologic transition to younger-age mortality declines in the earlier stage of this transition. Both epidemiologic transition and cohort evolution theories have found support in data on developed countries that had experienced a relatively early transition to increased life expectancies. Yet, studies on how well these theories fit countries experiencing the epidemiologic transition in later historical periods remain surprisingly scant.

Furthermore, both theories have not taken into account the consequences of mortality selection mechanism. The theory of population heterogeneity proposes that populations are composed of individuals or subpopulations with different physiological vulnerability to mortality, or *frailty* (Vaupel et al. 1979; Vaupel and Yashin 1987). Mortality tends to remove frailer individuals from the population at earlier ages, and leaves stronger individuals to survive to older ages. Later cohorts experience lower risks of infection and inflammation and have better nutrition and health capital during childhood, according to cohort evolution theories, so a relatively smaller proportion of frail individuals is selected out of the population at young ages.

later cohorts' overall mortality risk at older ages. In this case, old-age mortality rates may be potentially higher among later cohorts than among earlier cohorts, implying a high chronic disease burden and stagnation or decelerated increase in life expectancy in the periods when these cohorts reach old age. Zheng (2014), however, suggested that this mortality selection mechanism is not strong enough to override the cohort evolution mechanism among developed countries. But it is unknown if this finding also applies to the countries that have experienced the epidemiologic transition in more recent periods.

This study investigates the effects of mortality selection on cohort trends in life-course mortality patterns, and further consequences of mortality selection for period trends in life expectancy and the age-dependence of mortality. Data for these analyses are selected from four countries representing three different models of the epidemiologic transition. We propose a conceptual framework in which the effects of mortality selection are contingent on the historical mechanisms responsible for receding pandemics. In the early-transition countries, the primary reason for receding pandemics is socioeconomic development; whereas among the later-transition countries, the predominant reason for receding pandemics is medical technology. These different historical mechanisms imply different processes of mortality selection. In turn, different mortality selection processes generate different cohort trends in the pattern of mortality over the life-course, as well as different period trends in life expectancy and the age-dependence of mortality.

Epidemiologic Transition and Cohort Evolution

The theory of epidemiologic transition portrays four stages through which societies pass, starting with the age of pestilence and infectious diseases, continuing through the age of receding

pandemics, and advancing to the age of degenerative and man-made diseases and, at the transition's end, the age of delayed degenerative and man-made diseases. The timing, length, and mechanism of transition depend on the social and economic context of a given population. Most Western European and North American societies exemplify the classical model of the epidemiologic transition. In this model, societies begin the transition early and achieve the transition gradually over a period spanning the past two centuries. When these countries enter the early stages of the epidemiologic transition, mortality at young ages declines due to better sanitation and living standards; and in later stages, the elderly experience a substantial mortality decline due to improvements in medical technology. In the accelerated transition model, exemplified by Japan, a country does not experience the age of receding pandemics until around the early 20th century, but nevertheless completes the epidemiologic transition over just a few decades. Japan had begun a slow process of modernization prior to the drop in mortality in the 20th century, and, therefore, the early stage of its epidemiologic transition was shaped by both social development and medical advances (Omran 1971). Despite deviating in timing, speed, and transition mechanisms from the classical model, Japan ultimately followed an epidemiologic transition pattern similar to that experienced by Western countries. Going beyond the early- and accelerated-transition models, epidemiologic transition theory extends to the case of developing countries, which experience a later epidemiologic transition. Epidemiologic transition theory claims that, in such countries, imported medical technology and public health and disease control programs (made available through bilateral or international cooperation) initiate the age of receding pandemics without regard to the country's stage of socioeconomic development (Omran 1982). Although it would be naïve to reduce the complex mechanisms driving the epidemiologic transition in a given country to a singular cause, it does appear that, on the whole,

transitions in early-transition countries were predominantly socially determined, whereas transitions in later-transition countries were significantly more related to advances in medical technology (Omran 1971).

Whereas epidemiologic transition theory provides a period perspective to explain the historical mortality decline, other theories emphasize change across cohorts rather than across periods. These theories use life course and cohort replacement perspectives to attribute old-age mortality decline in later stages of the epidemiologic transition to mortality decline at younger ages for each cohort. One such theory describes the "cohort morbidity phenotype," proposing that cohorts that experience lower exposure to infection and inflammation during early childhood reap a lower mortality risk later in their lives (Finch and Crimmins 2004). Another theory, describing a trend of "technophysio evolution," argues that later cohorts are endowed with better health capital at birth, and enjoy lower rates of health capital depreciation over the life course due to increasing control over the environment, improved food and energy production, other technological innovation, and economic growth (Fogel and Costa 1997; Fogel 2004). Both theories suggest a positive correlation between early- and later-life mortality, although each emphasizes a different mechanism by which these are linked. Conversely, detrimental conditions in early life jeopardize survival in later life, a relationship that has been framed as "the physiological scarring effect" (Preston et al. 1998) or the "critical period" in epidemiology (Ben-Shlomo and Kuh 2002). Below, we refer to the theories of "cohort morbidity phenotype" and "technophysio evolution" collectively as the cohort evolution perspective, because both theories work at the cohort level rather than the individual level, and explicitly attribute historical declines in mortality to improvements in morbidity phenotypes or health capital endowment

across cohorts. The application of these theories to later-transition countries, however, has not yet been examined.

The Law of Mortality Selection

Epidemiologic transition theory recognizes that "mortality is a fundamental factor in population dynamics," as mortality leads to changes in fertility and subsequent changes in the age and sex structure of the population and changes in population growth (Omran, 1971). Yet, both the epidemiologic transition theory and the cohort evolution perspective neglect mortality selection, which may have important implications for differences in cohort life-course mortality pattern and the epidemiologic transition between early- and later-transition countries. The theory of population heterogeneity proposes that populations are composed of individuals or subpopulations with different physiological vulnerability to mortality, or *frailty* (Vaupel et al. 1979; Vaupel and Yashin 1987). Individual frailty is assumed to be fixed at birth. Mortality tends to survive to older ages. Therefore, the mortality selection mechanism implies that the frailty composition within a given birth cohort changes over the life course. Specifically, *the variance of the distribution of frailty declines over a cohort's lifetime*.

Cohort evolution theories attribute the positive correlation between young- and old-age *mortality rates* across birth cohorts to the positive correlation between early-life health and old-age health at *the individual level* (e.g., Bengtsson and Lindstrom 2003; Baker et al. 1991). By contrast, the mortality selection mechanism emphasizes the changing composition of a cohort as it ages. Therefore, the individual-level positive correlation between early-life health and old-age health may not necessarily be reflected in the cohort-level mortality selection process. For

example, cohort evolution theories suggest that later cohorts experience lower risks of infection and inflammation and have better nutrition and health capital during childhood. Therefore, a smaller proportion of frail individuals would be selected out of the population at young ages. In turn, this would cause a larger proportion of frail individuals to survive into old age, and would increase the cohort's overall mortality rate at older ages. In other words, a weakening of mortality selection in early life among later cohorts can amplify the rate of mortality acceleration. In this case, the old-age mortality rate may be potentially higher among later cohorts than among earlier cohorts, even though the individual-level positive correlation between early-life health and old-age health would persist within either cohort.

A useful illustration of the above argument takes advantage of Gompertz's (1825) classical law of mortality, which models the increase in mortality rates over adulthood within each birth cohort in an exponential pattern: $R_t = R_0 e^{\alpha t}$, where R_t is the mortality rate at age t, R_0 is the initial mortality rate, and α refers to the rate of increase in the mortality rate, alternately described as the rate of mortality acceleration. The cohort evolution mechanism suggests a positive correlation between young-age mortality *level* (or rate, $R_{young age}$) and old-age mortality *level* (or rate, $R_{old age}$) across cohorts, whereas the mortality selection mechanism emphasizes cohort change in the rate of mortality acceleration over the life course (i.e., the parameter α in the equation $R_t = R_0 e^{\alpha t}$, or the slope of the log mortality curve). Therefore, *the life-course mortality pattern of each successive cohort is determined by two mechanisms: cohort evolution and mortality selection.* Whether later cohorts have lower old-age mortality rates than earlier cohorts is determined not only by the later cohorts' lower young-age mortality rates (i.e., the cohort evolution mechanism) but also by a higher rate of mortality acceleration in later cohorts (i.e., the mortality selection mechanism).

The magnitude of α , or the slope of mortality acceleration over the life course, is not fixed across birth cohorts. This parameter is related to the variance of the distribution of frailty in the population within each birth cohort. "The slope of the mortality rate increases when the variance of heterogeneity distribution declines," (Yashin et al. 2002: 209; Vaupel 2010) because when a smaller proportion of frail individuals are selected out of the population at an early age, a larger proportion of frail individuals survive to older ages and the mortality curve at later ages becomes steeper. It is not well recognized in prior literature that an exacerbated acceleration of mortality in later life due to decreasing variance of the frailty distribution is consistent with Strehler and Mildvan (SM)'s (1960) general theory of mortality and aging. The latter theory posits that the initial mortality rate $\ln(R_0)^1$ and the slope α of the logarithm of the Gompertz mortality curve are negatively correlated. Therefore, both the theory of population heterogeneity and the SM model claim that a reduction in mortality selection in early life yields a steeper slope of mortality acceleration over life course. This claim means that cohort patterns of agedependence of mortality rates are affected not only by cohort evolution mechanisms, but also by the mortality selection mechanism. Below, we discuss how the relative strength of these two mechanisms may yield different cohort-specific patterns of mortality across the life-course between early- and later-transition countries.

Implications of Mortality Selection for Early-Transition Countries

In advanced societies, later cohorts born during the age of receding pandemics experience lower risks of infection and inflammation and have better nutrition and health capital during childhood. Therefore, according to cohort evolution theories, the variance of the frailty

¹ The initial mortality rate is denoted by $\ln(R_0)$, with subscript 0 indicating that this is the intercept of the logarithm of the Gompertz mortality curve.

distribution becomes smaller in successive cohorts, and a smaller proportion of frail individuals are selected out of the population at young ages. This, in turn, causes a larger proportion of frail individuals to survive into old age, amplifying mortality acceleration over the life course, and increasing the cohort's overall mortality rate at older ages. From the perspective of the SM model, a lower mortality rate at young ages, resulting from lower exposure to inflammation and improved health endowment, leads to a higher degree of mortality acceleration among later cohorts. In this case, the old-age mortality rate may be potentially higher for later cohorts than earlier cohorts. If so, young- and old-age mortality rates may not be positively correlated, contrary to the expectations of cohort evolution theories. Zheng (2014), however, suggested that this mortality selection mechanism is not strong enough to override the cohort evolution mechanism among advanced societies. In this case, even though later cohorts exhibit a steeper slope of mortality acceleration over the life course due to a reduction in mortality selection in early life, their old-age mortality rates remain lower than those of earlier cohorts, due to a better health endowment and lower inflammation exposure early in the life course (i.e., cohort evolution mechanisms). This pattern is summarized in Panel A of Figure 1, where a hypothetical later cohort 2 has lower old-age mortality rates than an earlier cohort 1 due to substantially lower mortality rates at younger ages, and despite having a steeper slope of mortality acceleration over the life course. This pattern was shown to be consistent with data from early-transition countries, and it may also apply to countries such as Japan, which had followed a similar but accelerated version of the classical model of the epidemiologic transition.

[Figure 1 about here]

It remains unclear why mortality selection does not override the cohort evolution mechanism in early-transition or advanced societies, such as those examined by Zheng (2014).

The relative strength of these two mechanisms may be determined by the reason for receding pandemics in each country. Among early-transition countries, the initial young-age mortality reduction during the age of receding pandemics is mainly due to socioeconomic development and consequent improvements in hygiene and nutrition, which are a byproduct of social change rather than a result of advancements in medical technology (Omran 1971). Socioeconomic development both improves the health endowment and also reduces exposure to inflammation among people born during this period. Therefore, socioeconomic development leads to lower mortality rates along the whole life course, according to cohort evolution theories. In other words, the specific mechanism of epidemiologic transition in early-transition countries provides the conditions for the cohort evolution mechanism to take effect. Furthermore, socioeconomic development reduces the variance of the frailty distribution at young ages among later cohorts. Therefore, diminished mortality selection in early life among people born during the age of receding pandemics in advanced societies is also a result of socioeconomic development, rather than medical advancement. This has two additional implications. First, socioeconomic development decreases the variance of the frailty distribution at young ages across cohorts gradually, rather than abruptly. Thus, the slope of the mortality curve will not be steep enough for older-age mortality rates among later cohorts to surpass those among earlier cohorts. Second, the variance of the distribution of frailty declines over a cohort's lifetime due to the mortality selection mechanism described above. Socioeconomic development can only save a moderate rather than a substantial proportion of frail individuals who otherwise would have died at a young age, meaning the variance of the frailty distribution will decline more slowly over the lifecourse of later cohorts, as compared to earlier cohorts. Also, due to a smaller variance in frailty at young ages (as a result of better health endowment and reduced exposure to inflammation),

later cohorts should have a smaller variance of the frailty distribution at any point in their lifetime, as compared to earlier cohorts. Therefore, the mortality curve over a cohort's lifetime will generally be steeper among later cohorts than among earlier cohorts. In sum, although socioeconomic development can reduce mortality selection in a way that yields a steeper mortality curve among later cohorts, this mortality curve would not be steep enough to cross over the mortality curve of earlier cohorts.

Implications of Mortality Selection for Later-Transition Countries

Among later-transition countries, the initial mortality reduction at young ages is mainly due to medical advancements rather than socioeconomic development, meaning that cohorts born in these countries during the age of receding pandemics might not have had significantly better nutrition or health endowment, or significantly lower exposure to inflammation, compared to earlier cohorts. Therefore, there is no solid foundation for mechanisms of cohort evolution. On the other hand, the mortality selection mechanism becomes a dominant determinant of cohort trends in the age-dependence of mortality rates. Vaccination and medical treatment allow an increasing share of frail individuals to survive into adulthood. In other words, medical advancements may depress mortality selection at early ages even further among later-transition countries, as compared to earlier-transition countries; and may cause the slope of mortality acceleration in later-transition countries to be especially steep. Although better nutrition and lower exposure to inflammation may still be linked with lower mortality risk at the *individual* level in any country, this mechanism may not produce the corresponding correlation at the cohort (or population) level in later-transition countries due to the extreme reduction of mortality selection at early ages achieved through medical advancement. This pattern is contrary to the cohort evolution perspective, which claims a positive correlation between young- and old-age

mortality rates. Instead, later cohorts may have higher mortality rates at old age than earlier cohorts born during the age of receding pandemics, due to the much steeper slope of mortality acceleration over the life course among the former. This pattern can be graphically presented in Panel B of Figure 1, where the hypothetical later cohort 2 has lower young-age mortality rates than the earlier cohort 1, but has higher old-age mortality rates due to the significantly steeper slope of mortality acceleration.

Panel B, however, may have oversimplified the dynamic mortality selection process among later-transition countries. It is important to emphasize that the initial young-age mortality reduction in these countries is mainly a result of medical advancement rather than socioeconomic development. Therefore, cohorts born during the age of receding pandemics might not necessarily have significantly better nutrition or health endowment, or significantly lower exposure to inflammation, as compared to earlier cohorts. This implies that the variance of the distribution of frailty at young ages might not be significantly smaller among later cohorts. Due to substantial reductions in mortality selection at early ages, a larger proportion of frail individuals survive to adulthood among later cohorts. This makes the variance of the frailty distribution decline at a significantly slower rate, possibly even exceeding the variance of the frailty distribution among earlier cohorts at some point during adulthood, such as age a, as shown in Panel C of Figure 1. As the slope of mortality acceleration is negatively associated with the variance of the frailty distribution (Yashin et al. 2002; Vaupel 2010), past age a the later cohort 2 will have a flatter slope of mortality acceleration than the earlier cohort 1. Conceptually, the rate of mortality acceleration declines after age a among cohort 2 because a larger proportion of frail individuals would die before reaching old age, resulting in a lower old-age mortality rate past age b among cohort 2. To clarify, the clear turning point at age a is a simplified example. In

reality, we should observe a smoother mortality curve than that portrayed in Panel C. Interestingly, in Panel C, young- and old-age mortality rates are still positively related, although this is a result of the mortality selection mechanism rather than the cohort evolution mechanism depicted in Panel A. Panel C does not apply to early-transition countries because, in these countries, the variance of the frailty distribution may decline at a slower rate among the later cohort 2, but it would not exceed the variance of the frailty distribution in cohort 1. The reason for the latter is that cohort 2 has a smaller initial variance of frailty at young ages, as a result of better health endowment and lower inflammation exposure, and exhibits a moderately slower decrease in the variance of the frailty distribution across the life course, as a result of socioeconomic development reducing mortality selection.

Panel D of Figure 1 describes how the age-dependence of mortality rates may change further in later cohorts 3 and 4. Due to continuing diffusion of medical treatment and public health programs, an even larger proportion of frail individuals might survive to adulthood in cohort 3, as compared to cohort 2. This generates an even steeper slope of mortality acceleration at younger ages, but, at the same time, reduces the rate at which the variance of the frailty distribution decreases. Therefore, the turning point in the mortality curve is now evident at an earlier age c, rather than age a, and here the variance of the frailty distribution in cohort 3 surpasses that of cohort 2. Consequently, the slope of mortality acceleration decreases after age c in cohort 3, and age-specific mortality rates become lower in cohort 3 than in cohort 2 past age d. The latter point arrives earlier in the life course than age b, when age-specific mortality rates become lower in cohort 2 than in cohort 1. Due to the mortality selection process, later cohorts may experience higher mortality rates in early or middle adulthood than earlier cohorts, but they will still have lower mortality rates at old ages compared to earlier cohorts. This produces a

positive correlation between young- and old-age mortality rates, but not as an outcome of cohort evolution mechanisms.

Further declines in the young-age mortality rate among later-transition countries are not mainly determined by medical advancements. In fact, declines in the mortality rate may become increasingly driven by socioeconomic development, if the economy and living conditions start improving in these countries. Such improvements would mean that later cohorts will start having better nutrition and health capital at young ages, and will face lower exposure to infection and inflammation during childhood, triggering the mechanism of cohort evolution. This process would gradually result in a smaller variance of the frailty distribution at young ages among the most recent cohorts, as illustrated by cohort 4 in Panel D. Due to weaker mortality selection operating on cohort 4, the variance of its frailty distribution declines at a slower rate throughout most of the life course, as compared to cohort 3, but it does not exceed the variance of the frailty distribution in cohort 3, which is significantly larger at young ages. Therefore, the slope of mortality acceleration is steeper in cohort 4 than in cohort 3 over most of the life course. Yet, old-age mortality rates in cohort 4 will not surpass those in cohort 3 due to a substantially better health endowment and lower exposure to inflammation in young age among this most recent cohort. In this case, even though mortality selection is at work, it would not override the cohort evolution mechanism. This scenario might characterize countries experiencing an early epidemiologic transition around the mid-19th century. The shift from "cohort 3" to "cohort 4" is definitely not as immediate as portrayed in Panel D. In fact, this shift may take two or three decades' worth of cohorts before the log-transformed mortality curves cease to cross over.

Changes in the age-dependence of mortality across birth cohorts, as portrayed in Panel D, have a very important implication for the epidemiologic transition among the later-transition

countries. Later cohorts in these countries, born during the age of receding pandemics, have a larger proportion of frail individuals survive to early and middle adulthood, although these individuals were exposed to unfavorable conditions in early life. Thus, we may observe a high prevalence rate of chronic diseases and an increase in the middle-age mortality rate among these cohorts. The increase in the middle-age mortality rate among later cohorts may counteract the effect of decreasing young-age mortality rates in recent periods, and cause life expectancy growth to slow or stagnate when the country enters the age of degenerative and man-made diseases. Therefore, even though later-transition countries may go through the age of receding pandemics at a relatively faster rate, compared to early-transition countries, the former may not be as fast in advancing from the age of degenerative and man-made diseases to the age of delayed degenerative and man-made diseases, because of the substantial reduction in mortality selection during the age of receding pandemics. When cohorts born during the age of receding pandemics reach old age, they will experience lower mortality rates than preceding cohorts, leading life expectancy to resume its increase after the period of stagnation. Further life expectancy increases will then occur as an outcome of cohort evolution mechanisms, as illustrated by cohort 4 in Panel D of Figure 1.

Data and methods

The mortality selection mechanism predicts different cohort trends in life-course mortality patterns between early- and later-transition countries because these countries differ from one another in the prominent cause of reduced mortality and mortality selection during the age of receding pandemics. In order to test this conceptual framework, we compare cohort trends in the age-dependence of mortality, period trends in the age-dependence of mortality, and period trends in life expectancy across three models of the epidemiologic transition: the classical model,

represented by Sweden; the accelerated transition model, represented by Japan; and the latertransition model, represented by Poland and Bulgaria. Mortality data on these countries are available from the Human Mortality Database (<u>http://www.mortality.org/</u>), which includes reliable life table, cohort, and period time series of mortality (population occurrence/exposure) rates. Japan, Poland and Bulgaria have limited data on young-age mortality rates in earlier cohorts and old-age mortality rates in later cohorts, but the available data are still sufficient for examining cohort trends in lifetime mortality rates. Due to problems of death under-registration and age uncertainty at very old ages, even in the best historical data (Crimmins and Finch 2006; Gavrilov and Gavrilova 2011), we restricted the upper end of age in our analysis to 94.

Ideally, we would have chosen an African or Asian developing country as another case representing the later-transition model, but cohort-level age-specific mortality rate data for these countries are rare, and most such countries did not enter the age of receding pandemics until around the 1960s or even later. Thus, most developing countries are still at this stage or at the next stage of the epidemiologic transition (the age of degenerative and man-made diseases). As few cohorts born after the 1960s have reached old age, this makes the study of old-age mortality rate in cohorts born during the age of receding pandemics currently infeasible for such countries. Poland and Bulgaria, however, entered the age of receding pandemics earlier than developing countries—around 1930-1940—but later than early-transition countries; and have completed the third stage of the epidemiologic transition. Their experience with the epidemiologic transition has important implications for other later-transition countries.

Nowadays, Poland and Bulgaria are classified as developed countries (United Nations 2012), yet these countries remain on a lower tier of the Human Development Index than Sweden and Japan (United Nations 2014). In a further contrast to today's most highly developed

countries, Poland and Bulgaria entered the age of receding pandemics around the 1930s in a way that mirrored the experience of developing countries around the 1960s and 1970s. The Polish and Bulgarian economic infrastructure and social environment were underdeveloped at that time: the economy was largely rural; technology, capital and access to markets were lacking (Watt 1998; Staniewicz 1999); and the Great Depression devastated every economic sector in both countries (Kaser and Radice 1987). Against this background of economic stagnation, medical treatment emerged as the main cause of initial declines in young-age mortality between the 1930s and 1960s, and included the diffusion of vaccinations, drugs, and medical technology. Further mortality declines after the 1960s, when these two countries started transitioning to the age of degenerative and man-made diseases (Vallin and Mesle 2004), became increasingly attributable to socioeconomic development, improved living standards, and improved nutrition. Therefore, if our conceptual framework is correct, cohorts born towards the end of the period in which mortality initially declined (1930s-1960s) should have lower mortality rates in young ages, higher mortality rates in early- and middle-adulthood, and lower mortality rates in late adulthood than cohorts born earlier during this period. Cohorts born after the 1960s, however, should start having lower mortality rates across the entire life course, as compared to earlier cohorts, due to the emergence of cohort evolution mechanisms. When members of the cohorts born after the 1930s reach adulthood around the 1960s-1970s, life expectancy may start stagnating. But, when these cohorts reach late adulthood around the 1990s, life expectancy should resume its increase.

In order to examine whether the trend of life expectancy has indeed been affected by cohort changes in lifetime mortality rates and cohort replacement mechanisms, we investigate the contribution of age-specific differences in mortality rates to the total difference in life expectancy over time. We use Arriaga's (1984) decomposition approach as specified below:

$${}_{n}\Delta_{x} = \frac{l_{x}^{1}}{l_{0}^{1}} \cdot \left(\frac{nL_{x}^{2}}{l_{x}^{2}} - \frac{nL_{x}^{1}}{l_{x}^{1}}\right) + \frac{T_{x+n}^{2}}{l_{0}^{1}} \cdot \left(\frac{l_{x}^{1}}{l_{x}^{2}} - \frac{l_{x+n}^{1}}{l_{x+n}^{2}}\right)$$

where l_x , ${}_nL_x$, and T_x are conventional functions of the life table, and refer to the number of people left alive at age *x*, person-years lived between ages *x* and *x* + *n*, and person-years lived above age *x*, respectively (Preston et al. 2001).

The term ${}_{n}\Delta_{x}$ refers to the total effect of a difference in mortality rates between ages *x* and x + n on the difference in life expectancy at birth between two populations, or between two time points within one population. Here, we investigate one population at two time points, denoted by the superscripts 1 and 2. Therefore, ${}_{n}\Delta_{x}$ can be interpreted as the difference in life expectancy at birth (in years) caused by a difference in the mortality rates between ages *x* and x + n.

The term $\frac{l_x^1}{l_0^1} \cdot \left(\frac{nL_x^2}{l_x^2} - \frac{nL_x^1}{l_x^1}\right)$ corresponds to the direct effect of a change in the number of

years lived between ages x and x + n on life expectancy at birth. The term $\frac{T_{x+n}^2}{l_0^1} \cdot \left(\frac{l_x^1}{l_x^2} - \frac{l_{x+n}^1}{l_{x+n}^2}\right)$ corresponds to the sum of indirect and interaction effects: the contribution of added person-years due to additional survivors at age x + n being exposed to new mortality conditions.

For the open-ended age interval, there will be only a direct effect:

$${}_{\infty}\Delta_{x} = \frac{l_{x}^{1}}{l_{0}^{1}} \cdot \left(\frac{T_{x}^{2}}{l_{x}^{2}} - \frac{T_{x}^{1}}{l_{x}^{1}}\right)$$

Therefore, for each age group between age *x* and age x + n, except for the open-ended age interval, a change in the mortality rate would have both direct and indirect effects on life expectancy at birth. The sum of these effects across all age groups, $\sum_{0}^{\infty} n\Delta x$, is equal to the difference in life expectancy between the two time points. Therefore, $\frac{100*n\Delta x}{\sum_{0}^{\infty} n\Delta x}$ represents the

percentage of total difference in life expectancy at birth attributable to the difference in mortality rates between age x and age x + n.

Results

Cohort trends in the age-dependence of mortality rates

Figure 2 shows age-specific mortality rates over the lifespan of several birth cohorts in four countries: Sweden, Japan, Bulgaria, and Poland. For this Figure, we chose cohorts that were born around the age of receding pandemics in each country. Unlike Sweden, which has complete life-course data for each birth cohort, the other three countries have incomplete life-course data, meaning that later cohorts only have data on younger-age mortality rates and earlier cohorts only have data on older-age mortality rates. Sweden entered the age of receding pandemics around the early 20th century, and moved to the age of degenerative and man-made diseases around the early 20th century. In Swedish cohorts born during this period, young-age and old-age mortality rates are generally positively correlated, meaning that later cohorts which had lower young-age mortality rates also had lower old-age mortality rates, as compared to earlier cohorts—despite a steeper slope of mortality acceleration among the former. A similar pattern is evident in Japan, although this country did not enter the age of receding pandemics until around the early 20th century, and went through this stage of the epidemiologic transition at a faster rate compared to Sweden.

[Figure 2 about here]

In Poland and Bulgaria, however, cohort trends in the age-specific mortality rates over the lifespan are very different than those in Sweden or Japan. Poland and Bulgaria entered the age of receding pandemics around the 1930s, even later than Japan did, and went through this stage of the transition at an even faster rate, due to medical advancements. Yet, older-age mortality rates exhibit substantial overlap across Polish and Bulgarian cohorts born at this stage of the epidemiologic transition. For example, in Bulgaria, the 1940 birth cohort had lower mortality rates than the 1920 birth cohort until age 40, at which point the mortality rates of the 1940 birth cohort surpassed those of the 1920 birth cohort. Yet, in the former cohort, the slope of the mortality curve became flatter between ages 40 and 60, eventually leading to lower mortality rates past age 60 as compared to the 1920 birth cohort. This pattern is consistent with Panel C of Figure 1.

Figure 3 focuses on the mortality curve between ages 20 and 74 across 10 year intervals of cohorts, and clearly shows the two-step crossover in mortality rates among birth cohorts born from 1930 until 1960 in Poland and Bulgaria. For example, in Poland, the 1940 birth cohort intersects with the 1930 birth cohort at ages 35 and 55. The intersection points between the 1940 and 1950 birth cohorts appear earlier in the life-course: at ages 25 and 45, respectively. Past the 1960 birth cohort, and especially past the 1970 birth cohort, mortality curves no longer cross over those of earlier cohorts. In Bulgaria, the 1940 birth cohort intersects with the 1930 birth cohort intersects with the 1950 birth cohort intersects with the 1940 birth cohort at ages 30 and 55; the 1960 birth cohort intersects with the 1950 birth cohort at ages 25 and 40; and the 1970 birth cohort intersects with the 1960 birth cohort at ages 15 (outside the range of ages shown in Figure 3) and 25. Past the 1980 birth cohort, the mortality curve no longer intersects with those of earlier cohorts. These patterns support Panel D of Figure 1.

[Figure 3 about here]

Period trends in the age-dependence of mortality rates and in life expectancy

Figure 4 shows period changes in age-specific mortality rates over ages 0-94 during and after the age of degenerative and man-made diseases in Sweden, Japan, Poland and Bulgaria. In Sweden and Japan, the positive correlation between young- and old-age mortality rates among cohorts born during the age of receding pandemics means that older-age mortality rates are generally lower in successive periods past that stage of the epidemiologic transition. This leads to a generally linear increase in life expectancy since the age of degenerative and man-made diseases, as shown in Figure 6.

[Figures 4, 6 about here]

Yet, the pattern of period trends is very different for Poland and Bulgaria. The substantial overlap in mortality rates among cohorts born during the age of receding pandemics means that older-age mortality rates also substantially overlap past that stage, as the cohorts born during this stage grow older. Figure 5 clearly portrays period changes in age-specific mortality rates over ages 20-74 in these two countries. Compared to 1958, Poland has higher middle-age mortality rates in 1980, when cohorts born during the age of receding pandemics begin to reach middle age. When these cohorts reach old age in 2000, old age mortality rates become lower than in 1980. Young- and middle-age mortality rates are also lower in 2000 than in 1980, consistent with emerging mechanisms of cohort evolution. As mentioned earlier, the mortality curves of later and earlier cohorts cease to cross over one another past the 1960 birth cohort. The same mechanisms can be used to explain lower mortality rates in 2009 relative to 2000 for ages 20-74.

[Figure 5 about here]

Bulgaria shows period changes in age-specific mortality rates that are similar to those observed in Poland. The difference between the two countries is that Bulgarian mortality rates at

middle ages are still higher in 2000 than in 1980. But, Bulgarian mortality rates over ages 20-74 all become smaller in 2010 than in 2000, consistent with a mechanism of mortality selection operating on earlier cohorts (older age groups in 2010) and cohort evolution mechanisms operating on cohorts born since the 1970s (younger age groups in 2010). Bulgaria's decade-long delay in the cessation of crossover between period mortality curves, relative to Poland, corresponds to a decade-long delay in the emergence of a strong cohort evolution mechanism in the Bulgarian data. Crossover in middle-age mortality rates between the late 1950s and 2000s implies that both Poland and Bulgaria may have experienced a period of stagnation in life expectancy at birth. As shown in Figure 6, despite life expectancy exceeding 50 in both countries during the 1950s, life expectancy has indeed stagnated around 70 years for several decades, and did not increase further in either country until the 1990s.

Tables 1 and 2 further demonstrate how differences in age-specific mortality rates contribute to differences in life expectancies at birth past the 1950s in Poland and Bulgaria, respectively. Three time periods are examined, based on the turning points in the life expectancy trends shown in Figure 6. The parameter ${}_{n}\Delta_{x}$ can be interpreted as the difference in life expectancy at birth (in years) between two time points caused by the difference in mortality rates between ages *x* and *x* + *n*. *Percent* refers to the percentage of the total difference in life expectancy at birth attributable to the difference in mortality rates between ages *x* and *x* + *n*. Here we use Poland as an example to illustrate the interpretation of these tables. Results for Bulgaria are similar to those for Poland.

[Tables 1 and 2 about here]

In Poland, life expectancy at birth increased by 4.89 years between 1958 and 1972, decreased by 0.45 years between 1972 and 1991, and increased again by 5.34 years between 1991 and 2009. During the first period, 1958-1972, the reduction in mortality rates before age 10 contributed about 3.48 years to the increase in life expectancy (71% of the total difference in life expectancy between 1958 and 1972). Survival improvement in other age groups contributed about 1 year (29% of the total difference) to the increase in life expectancy.

During the second period, 1972-1991, reduction in young-age mortality rates before age 10 contributed about 0.92 years to the change in life expectancy. This contribution accounted for a negative percentage (-205%) of the total difference because total life expectancy at birth decreased over this period. In other words, between 1972 and 1991, young-age mortality rates continued to decrease, but total life expectancy at birth did not improve. The positive effect of survival improvements at young ages was offset by increased mortality in early and middle adulthood (ages 30-64). Increased mortality in the latter age range accounted for a decline in life expectancy amounting to 1.33 years (298% of total difference). Early- and middle-adulthood mortality rates increased over this period due to the substantial reduction of mortality selection in cohorts born during the age of receding pandemics. Medical advances had enabled a larger proportion of frail individuals from these cohorts to survive into early and middle adulthood, but contributed to temporarily elevated mortality rates in the middle-age group between 1972 and 1991.

During the third period, 1991-2009, continuing reduction in mortality rates before age 10 contributed about 1.1 years (20% of the total difference) to the increase in life expectancy. Middle-age mortality rates also decreased, as augured by the reduction in young-age mortality rates in the second period (i.e., by the emergence of a cohort evolution mechanism). As shown in Figure 3, the cohort evolution mechanism emerged after the 1960s, and especially after the 1970s birth cohorts. Lower young-age mortality rates among these cohorts lead to lower middle-age mortality rates when these cohorts reach middle age during the third period. Therefore, a reduction in middle-age mortality rates positively contributes to the increase in life expectancy in the third period. Yet, a reduction in old-age mortality rates (at ages 60 and above) contributes even more—about 2.4 years (44% of total difference)—to the increase in life expectancy. This decline in old-age mortality rates is attributable to the higher mortality rates in early- and middle-adulthood among cohorts born during the age of receding pandemics. Only resilient individuals from these cohorts survive to late ages, and this reduces the old-age mortality rate in the third period.

Discussion and conclusion

This study investigates the effects of mortality selection on changes in the life-course mortality pattern across cohorts, and the further consequences of these changes for period trends in life expectancy and the age-dependence of mortality. Different consequences of mortality selection are contrasted across three models of the epidemiologic transition. The central premise of our conceptual framework is that the effect of mortality selection is contingent on the historical mechanisms that initiate the age of receding pandemics. Both early- and later-transition countries have experienced the age of receding pandemics, but for different reasons.

Among early-transition countries, the prominent factors driving receding pandemics are socioeconomic development, improved living standards, improved nutrition and improved hygiene. Socioeconomic development triggers cohort evolution mechanisms; at the same time, it also gradually reduces the variance of the frailty distribution at young ages across successive

birth cohorts and over the lifetime. This amplifies mortality acceleration among later cohorts; but these cohorts still experience an old-age mortality rate lower than preceding cohorts, due to cohort evolution mechanisms. Among later-transition countries, however, medical advancement is the prominent cause of receding pandemics, and it does not trigger the emergence of cohort evolution mechanisms. Instead, medical advancement exaggerates the process of mortality selection. This process first yields very steep mortality acceleration among later cohorts, resulting in higher mortality rates at early and middle adulthood. Yet, at a certain age, the variance of the frailty distribution among the later cohorts surpasses the corresponding variance among the preceding cohorts, and the slope of mortality acceleration becomes less steep among the later cohorts. This leads to lower mortality rates in late adulthood.

Using cohort data on age-specific mortality rates from the Human Mortality Database, we find very different cohort trends in life-course mortality patterns between early-transition countries, including Sweden (exemplifying the classical model) and Japan (exemplifying the accelerated transition model), and two countries that experienced a later transition, Poland and Bulgaria. In both Sweden and Japan, cohorts born towards the end of the age of receding pandemics have both lower mortality rates and a higher rate of mortality acceleration, as compared to cohorts born at the beginning of the age of receding pandemics. In Poland and Bulgaria, however, cohorts born towards the end of the age of receding pandemics have lower young-age mortality rates than cohorts born at the beginning of this period, but a much steeper slope of mortality acceleration, leading to high mortality rates in early and middle adulthood. Past middle adulthood, however, the later cohorts have a flatter slope of mortality acceleration, and exhibit lower mortality rates in old age, as compared to the earlier cohorts. Among cohorts born past the 1960s and 1970s in Poland and Bulgaria, cohort evolution mechanisms begin to

emerge due to socioeconomic development, and life-course mortality patterns begin to resemble those observed in Sweden and Japan.

Early- and later-transition countries have different life-course mortality patterns for cohorts born during the age of receding pandemics, and these differences are mirrored in different period trends in life expectancy and the age-dependence of mortality past the age of receding pandemics. In Sweden and Japan, mortality rates across age groups in a given period generally do not cross over mortality rates from earlier periods; in other words, the period which has the lower young-age mortality rate also tends to have the lower old-age mortality rate. This leads to a generally linear increase in life expectancy across periods.

The evolution of life expectancy differs in Poland and Bulgaria. When cohorts born during the age of receding pandemics reach early and middle adulthood, the corresponding periods have higher mortality rates at young and middle ages than preceding periods. Higher mortality rates in these age groups offset life expectancy gains from continually declining youngage mortality rates, and lead to stagnation in life expectancy. Thus, although life expectancy in Poland and Bulgaria surpassed 50 years by the 1950s, the next several decades witnessed stagnation in life expectancy around the 70-year mark. For example, between 1972 and 1991, an increasing mortality rate among young and middle-aged adults in Poland was estimated to elicit a 1.33 year decline in life expectancy at birth. Yet, when cohorts born during the age of receding pandemics reached late adulthood, the corresponding periods evinced lower mortality rates at old ages than preceding periods, and this contributed to an increase in life expectancy. For example, a reduction in Polish old-age mortality rates contributed about 2.4 years, or 44% of the total change, to the increase in life expectancy between 1991 and 2009. Thus, the mortality selection process affecting cohorts born during the age of receding pandemics in later-transition countries initially caused stagnation but later caused an increase in life expectancy past the age of receding pandemics.

The above arguments do not preclude the possible contribution of cohort evolution mechanisms to increases in life expectancy among later-transition countries. In fact, strong cohort evolution mechanisms appear beginning with the 1960s birth cohort in Poland and the 1970s birth cohort in Bulgaria, and these mechanisms contribute to lower middle-age mortality rates in the periods when these cohorts reach early and middle adulthood. In turn, this reduction in middle-age mortality contributes to an increase in life expectancy. For example, the reduction in Polish mortality rates between ages 20 and 49 contributes about 1.2 years (21% of the total difference) to the increase in life expectancy between 1991 and 2009.

In this paper, we mainly have used cohort perspectives, including mortality selection and cohort evolution, to explain period trends of life expectancy in later-transition countries. This is not to suggest that period effects are unimportant. On the contrary, the strong contribution of declining infant mortality rates to period trends in life expectancy in Poland and Bulgaria, as shown in Tables 1 and 2, implies a strong period effect. Moreover, socioeconomic development, medical advancements, and environmental changes may also have important consequences for changes in the mortality rates of other age groups. Particularly, in the context of Eastern Europe, economic stagnation before the collapse of the Soviet Union may have contributed to the stagnation in life expectancy after the 1970s. Yet, despite an upward trend in Poland's Gross Domestic Product per capita since 1980 (as shown in Appendix I), life expectancy in Poland continued to stagnate for another decade. It is beyond the scope of this paper to comprehensively investigate every determinant of the trend in life expectancy in Poland and Bulgaria; rather, these countries are presented as two examples of later-transition countries standing in contrast to early-

transition countries. There does not appear to be a persuasive period mechanism that could explain (1) why, in later-transition but not early-transition countries, there is an increase in mortality rates in young and middle adulthood and then a decrease in mortality rates in late adulthood for successive cohorts born during the age of receding pandemics; and (2) why, in later-transition but not early-transition countries, there is an increase in mortality rates among young and middle-age adults after the age of receding pandemics, and a subsequent decrease in mortality rates among older adults. In other words, the changing timing of the cross-over in mortality rates along the life course across successive cohorts and the changing timing of the cross-over in mortality rates across age groups over successive periods (as portrayed in Figures 2-5) cannot be explained by period mechanisms. Instead, cohort differences in life-course mortality patterns, arising from differences in mortality selection between later- and earlytransition countries, explain these phenomena in ways that show good agreement with the data.

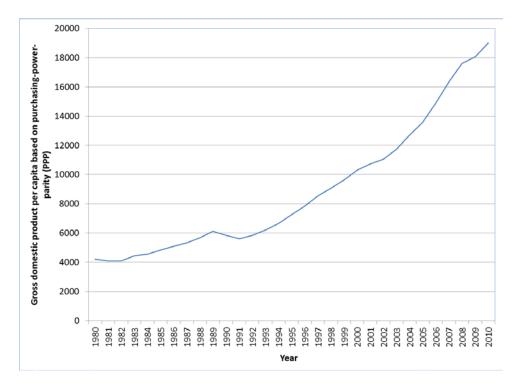
Mortality selection also has an important implication for the disease burdens in latertransition countries. Medical treatment allows a large proportion of frail individuals born during the age of receding pandemics to survive to early and middle adulthood. These individuals still have a poor health endowment and a history of high exposure to infection and inflammation in early life. Therefore, when they grow up, they may face a greater risk of developing and dying from chronic illnesses, such as cancer, stroke and heart disease. This would prolong the time later-transition countries spend in the age of degenerative and man-made diseases, even though they went through the age of receding pandemics within a much shorter time frame than earlytransition countries. Consistent with this prediction, studies have found that, in low- and middleincome countries, middle-aged adults are especially vulnerable to chronic diseases. People in these countries tend to develop chronic diseases at younger ages, suffer from more complications,

and die sooner than people in high-income countries (World Health Organization 2005). Studies also find that cardiovascular mortality rates increased in the age of degenerative and man-made diseases in some Eastern European countries (Vallin and Mesle 2004). These findings are consistent with our conceptual framework and results showing that in later-transition countries, blunted mortality selection due to medical advancement leads to increased mortality rates in middle age. Despite the likely origin of chronic disease burdens in patterns of mortality selection, current studies of chronic disease burdens in later-transition countries have primarily focused on period factors, including changes in air pollution, environmental degradation, economic stagnation, urbanization, nutrition transition, unhealthy diet, and a sedentary lifestyle (Caballero and Popkin 2002; Chen et al. 2013; Pimentel 2007; Smith 2000; Vallin and Mesle 2004; Yusuf 2001). These period factors certainly have important consequences for the incidence and prevalence of chronic diseases; but the interaction between such period factors and the composition of the population exposed to these factors may result in an especially large disease burden, because frail individuals are more susceptible to health risk factors (e.g., Gouveia and Fletcher 2000).

Several limitations should be noted. First, although the Human Mortality Database is considered to be of high quality, and has been widely used for cross-national and historical research (e.g., Minagawa 2013), we cannot completely dismiss the possibility of bias due to data misreporting. Second, only Poland and Bulgaria were chosen as examples of later-transition countries, because other countries that have experienced a late epidemiologic transition have very limited cohort data on age-specific mortality rates. Future research should replicate the analysis in this paper when comparable data become available for countries in the developing world.

To recapitulate, this study proposes a conceptual framework for understanding the effects of mortality selection on cross-cohort changes in mortality over the life-course; and further implications of mortality selection for period changes in life expectancy and the age-dependence of mortality. A key premise of this framework is that different historical mechanisms responsible for receding pandemics in each country have different implications for the subsequent development of mortality regimes. Among cohorts born during the age of receding pandemics, socioeconomic development triggers cohort evolution mechanisms and leads to moderate reductions in mortality selection at early life. This means mortality selection mechanisms will not override cohort evolution mechanisms. In this case, there is no crossover of age-specific mortality rates between earlier and later birth cohorts, or between earlier and later periods; and there is a linear increase in life expectancy past the age of receding pandemics. This pattern can be observed in early-transition countries, including Sweden and Japan. By contrast, advances in medical technology do not trigger cohort evolution mechanisms, but instead allow more frail individuals to survive to adulthood among cohorts born during the age of receding pandemics. This increases mortality rates in early and middle adulthood, relative to preceding cohorts. But, when the variance of the frailty distribution among later cohorts surpasses that among the preceding cohorts, the slope of mortality acceleration becomes flatter; and this leads later cohorts to exhibit lower mortality rates at old ages. Consequently, young- and old-age mortality rates are positively correlated across cohorts due to mortality selection, but not due to cohort evolution mechanisms. As later-transition countries pass the age of receding pandemics, crossover in agespecific mortality rates between early and late cohorts leads to crossover in age-specific mortality rates between early and late periods, and stagnation followed by an increase in life expectancy. This pattern can be observed in later-transition countries, including Poland and

Bulgaria. Further declines in early-life mortality are driven by socioeconomic development, as the economy and living conditions start improving in later-transition countries, and trigger cohort evolution mechanisms. The latter lead to cohort and period patterns of mortality, as well as period trends of life expectancy, that eventually come to resemble those experienced by earlytransition countries. Appendix I. Trend in Gross Domestic Product per capita based on Purchasing-Power-Parity (PPP) in Poland, 1980-2010



Source: International Monetary Fund World Economic Outlook, 2014

Reference

Arriaga, Eduardo. 1984. "Measuring and explaining the change in life expectancies," *Demography* 21(1): 83-96.

Barker, D.J.P., K.M. Godfrey, C. Fall, C. Osmond, P.D. Winter, and S.O. Shaheen. 1991. "Relation of birth weight and childhood respiratory infection to adult lung function and death from chronic obstructive airways disease," *British Medical Journal* 303: 671-675.

Bengtsson, Tommy and Martin Lindstrom. 2003. "Airborne infectious diseases during infancy and mortality in later life in southern Sweden, 1766-1894," *International Journal for Epidemiology* 32(2): 286-294.

Ben-Shlomo, Yoav and Diana Kuh. 2002. "A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives," *International Journal of Epidemiology* 31(2): 285-293.

Caballero, Benjamin, and Barry M. Popkin. 2002. *The Nutrition Transition: Diet and Disease in the Developing World*. London: Academic Press.

Chen, Yuyu, Avraham Ebenstein, Michael Greenstone, and Hongbin Li. 2013. "Evidence on the impact of sustained exposure to air pollution on life expectancy from China's Huai River policy," *Proceedings of the National Academy of Sciences* 110(32): 12936-12941.

Crimmins, Eileen M. and Caleb E. Finch. 2006. "Infection, inflammation, height, and longevity," *Proceedings of National Academy of Science* 103(2): 498-503.

Finch, Caleb E. and Eileen M. Crimmins. 2004. "Inflammatory exposure and historical changes in human life-spans," *Science* 305: 1736-1739.

Fogel, Robert W. 2004. "Changes in the process of aging during the twentieth century," *Population and Development Review* 30(Suppl.): 19-47.

Fogel, Robert W. and Dora L. Costa. 1997. "A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs," *Demography* 34: 49-66.

Gavrilov, Leonid A., and Natalia S. Gavrilova. 2011. "Mortality measurement at advanced ages: A study of the social security administration death master file," *North American Actuarial Journal* 15(3): 432-447.

Gompertz, Benjamin. 1825. "On the nature of the function expressive of the law of mortality," *Philosophical Transactions* 27: 513-585.

Gouveia, Nelson, and Tony Fletcher. 2000. "Time series analysis of air pollution and mortality: effects by cause, age and socioeconomic status," *Journal of Epidemiology and Community Health* 54: 750-755.

Kaser, M. C., and E. A. Radice. 1987. *The Economic History of Eastern Europe 1919-1975: Volume II: Interwar Policy, The War, and Reconstruction.* London: Oxford University Press.

Minagawa, Yuka. 2013. "Inequalities in healthy life expectancy in Eastern Europe," *Population and Development Review* 39(4): 649-671.

Olshansky, S. Jay and A. Brian Ault. 1986. "The fourth stage of the epidemiologic transition: The age of delayed degenerative diseases," *The Milbank Quarterly* 64(3): 355-391.

Omran, Abdel R. 1971. "The epidemiologic transition: A theory of the epidemiology of population change," *Milbank Memorial Fund Quarterly* 49(4): 509-538.

Orman, Abdel R. 1982. "Epidemiologic transition," Pp. 172-183 in *International Encyclopedia* of *Population*. New York: The Free Press.

Pimentel, D., S. Cooperstein, H. Randell, et al. 2007. "Ecology of increasing diseases: population growth and environmental degradation," *Human Ecology* 35 (6): 653-668.

Preston, Samuel H., Patrick Heuveline, and Michel Guillot. 2001. *Demography: Measuring and Modeling Population Processes*. Malden, MA: Blackwell Publishing.

Preston, Samuel H., Mark E. Hill, and Greg L. Drevenstedt. 1998. "Childhood conditions that predict survival to advanced ages among African-Americans," *Social Science & Medicine* 47: 1231-1246.

Smith, Kirk R. 2000. "National burden of disease in India from indoor air pollution," *Proceedings of the National Academy of Sciences* 97(24): 13286-13293.

Staniewicz, Witold. 1999. "The agrarian problem in Poland between the Two World Wars," *Slavonic & East European Review* 43(100): 23-33.

Strehler, Bernard L. and Albert S. Mildvan. 1960. "General theory of mortality and aging," *Science* 132: 14-21.

United Nations. 2012. World Economic Situation and Prospects 2012. New York: United Nations.

United Nations. 2014. *Human Development Report 2014*. New York: United Nations Development Programme.

Vallin, Jacques, and France Mesle. 2004. "Convergences and divergences in mortality. A new approach in health transition," *Demographic Research*, special collection 2, article 2, pages 11-44.

Vaupel, James W. 2010. "The theory of heterogeneity: A concise primer." Manuscript, Max Planck Institute for Demographic Research. Rostock, Germany.

Vaupel, James W., Kenneth G. Manton, and Eric Stallard. 1979. "The impact of heterogeneity in individual frailty on the dynamics of mortality," *Demography* 16(3): 439-454.

Vaupel, James W., and Anatoli I Yashin. 1987. "Repeated resuscitation: How lifesaving alters life tables," *Demography* 24(1): 123-135.

Watt, Richard. 1998. *Bitter Glory: Poland and Its Fate, 1918-1939*. New York: Hippocrene Books.

World Health Organization. 2005. Preventing Chronic Diseases: A Vital Investment: WHO Global Report. Geneva: World Health Organization.

Yashin, Anatoli I., Svetlana V. Ukraintseva, Serge I. Boiko, and Konstantin G. Arbeev. 2002. "Individual aging and mortality rate: how are they related?," *Social Biology* 49: 206-217.

Yusuf, Salim, Srinath Reddy, Stephanie Ounpuu, and Sonia Anand. 2001. "Global burden of cardiovascular diseases part I: general considerations, the epidemiologic transition, risk factors, and impact of urbanization," *Circulation* 104: 2746-2753.

Zheng, Hui. 2014. "Aging in the Context of Cohort Evolution and Mortality Selection," *Demography* 51(4):1295-1317.

	1958-1972		1972-1991		1991-2009	
age	$_{n}\Delta_{x}$	percent	$n\Delta_x$	percent	$_{n}\Delta_{x}$	percent
0	3.08	62.9%	0.75	-166.9%	0.95	17.9%
1	0.34	6.9%	0.11	-25.6%	0.10	1.8%
5	0.06	1.3%	0.06	-12.7%	0.04	0.8%
10	0.05	1.1%	0.02	-4.4%	0.04	0.7%
15	0.06	1.3%	0.00	-0.7%	0.06	1.2%
20	0.09	1.7%	0.01	-2.8%	0.10	1.9%
25	0.10	2.1%	0.00	0.3%	0.12	2.2%
30	0.08	1.7%	-0.03	6.5%	0.15	2.8%
35	0.04	0.9%	-0.09	21.0%	0.21	3.9%
40	0.03	0.7%	-0.16	36.5%	0.27	5.0%
45	0.04	0.8%	-0.23	51.3%	0.29	5.5%
50	0.07	1.3%	-0.28	62.5%	0.31	5.8%
55	0.14	2.8%	-0.30	66.6%	0.33	6.2%
60	0.17	3.6%	-0.24	53.7%	0.40	7.6%
65	0.15	3.1%	-0.11	24.2%	0.49	9.1%
70	0.15	3.0%	0.01	-2.6%	0.51	9.5%
75	0.12	2.4%	0.03	-6.6%	0.47	8.8%
80	0.10	1.9%	0.00	-0.5%	0.32	5.9%
85	0.02	0.3%	0.01	-1.9%	0.13	2.5%
90+	0.00	0.0%	-0.01	2.0%	0.05	0.9%
sum	4.89	100%	-0.45	100.0%	5.34	100.0%

Table 1. Age decomposition of differences in life expectancies at birth in Poland, 1958-2009

	1955-1965		1965-1997		1997-2010	
age	$n\Delta_x$	percent	$_{n}\Delta_{x}$	percent	$_{n}\Delta_{x}$	percent
0	3.96	60.9%	0.91	-90.4%	0.51	15.0%
1	1.25	19.2%	0.06	-5.7%	0.23	6.7%
5	0.09	1.4%	0.04	-3.7%	0.07	2.0%
10	0.09	1.3%	0.01	-1.0%	0.03	1.0%
15	0.08	1.2%	0.02	-2.0%	0.06	1.9%
20	0.13	1.9%	-0.01	1.3%	0.07	2.2%
25	0.13	2.0%	0.00	0.4%	0.05	1.3%
30	0.10	1.5%	-0.03	3.2%	0.08	2.3%
35	0.08	1.2%	-0.09	8.9%	0.12	3.6%
40	0.07	1.0%	-0.20	19.6%	0.13	3.9%
45	0.10	1.5%	-0.29	28.8%	0.17	4.9%
50	0.08	1.2%	-0.32	31.7%	0.19	5.7%
55	0.17	2.6%	-0.33	33.0%	0.19	5.4%
60	0.08	1.2%	-0.24	24.1%	0.26	7.6%
65	0.08	1.2%	-0.05	5.2%	0.30	8.7%
70	0.05	0.7%	-0.07	7.2%	0.38	11.2%
75	0.00	0.0%	-0.06	5.7%	0.30	8.7%
80	-0.01	-0.1%	-0.11	11.1%	0.19	5.6%
85	0.00	0.0%	-0.11	11.2%	0.07	2.1%
90+	0.01	0.2%	-0.11	11.3%	0.01	0.4%
sum	6.50	100%	-1.00	100.0%	3.42	100.0%

Table 2. Age decomposition of differences in life expectancies at birth in Bulgaria, 1955-2010

A: early transition countries **B: later transition countries** (simplified) Cohort 1 Cohort 1 Cohort 2 Cohort 2 ln(Rt) ln(Rt) Age (t) Age (t) C: later transition countries **D: later transition countries** (dynamic) (multiple cohorts) Cohort 1 Cohort 1 Cohort 2 Cohort 2 •• Cohort 3 Cohort 4 ln(Rt) In(Rt) b а b d a с Age (t) Age (t)

Figure 1. Hypothetical cohort changes in the age-dependence of mortality rates in early- and later-transition countries.

Note: $\ln(R_t)$ represents the logarithm transformation of age-specific mortality rate at age t.

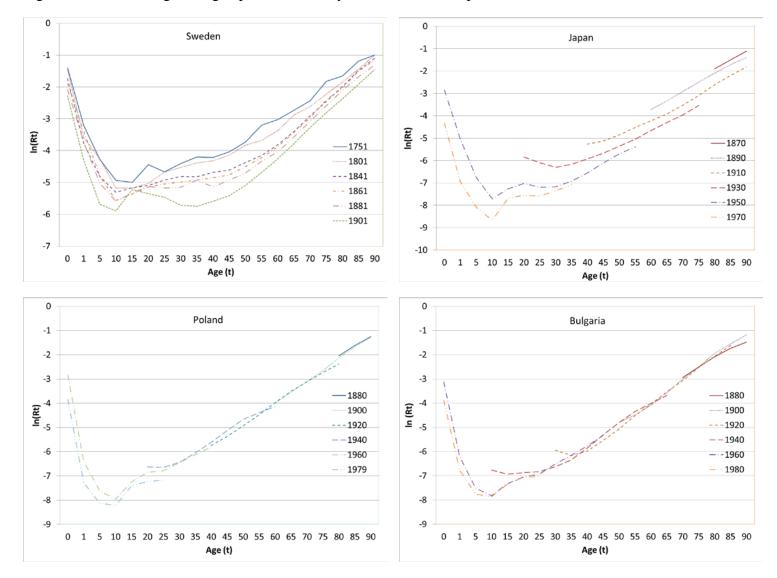


Figure 2. Cohort changes in age-specific mortality rates over the lifespan in various countries.

Note: $\ln(R_t)$ represents the logarithm transformation of age-specific mortality rate at age t.

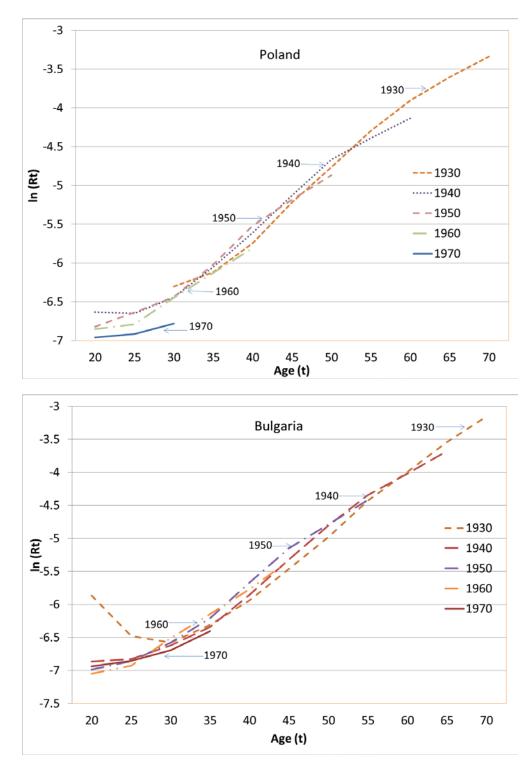


Figure 3. Cohort changes in age-specific mortality rates over age 20-74 in Poland and Bulgaria, 1930-1970.

Note: $\ln(R_t)$ represents the logarithm transformation of age-specific mortality rate at age t.

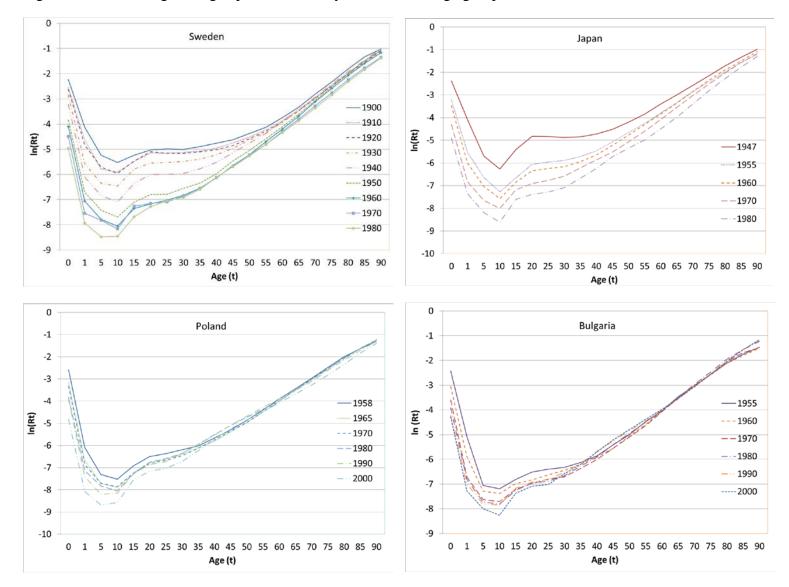


Figure 4. Period changes in age-specific mortality rates over the age groups in various countries.

Note: $\ln(R_t)$ represents logarithm transformation of age-specific mortality rate at age t.

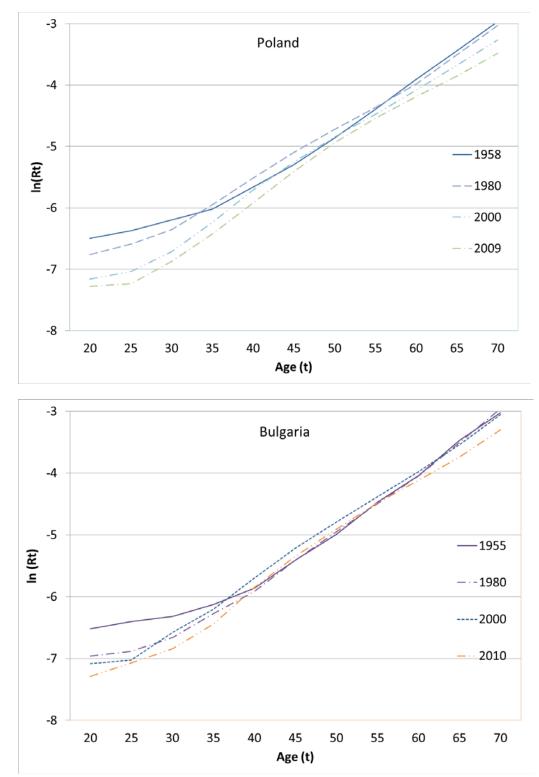


Figure 5. Period changes in age-specific mortality rates over ages 20-74 in Poland and Bulgaria, 1950s-2000s.

Note: $\ln(R_t)$ represents the logarithm transformation of age-specific mortality rate at age t.

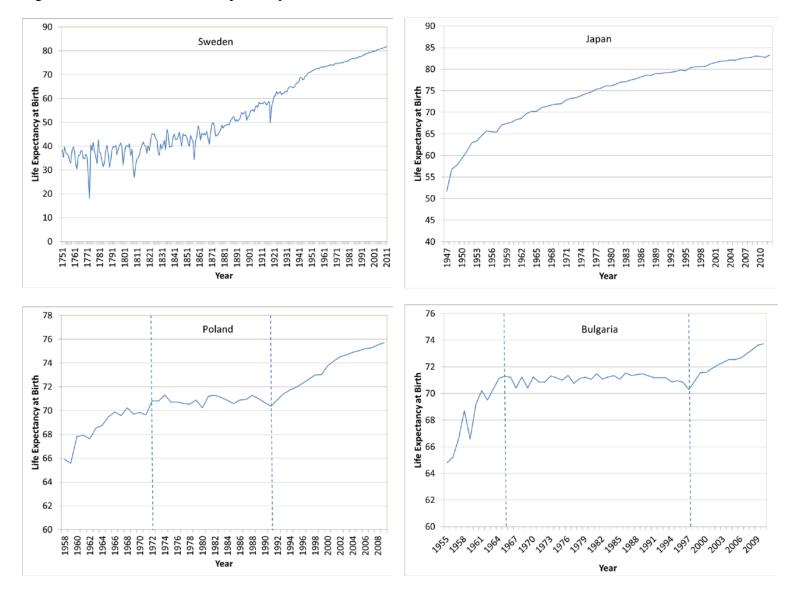


Figure 6. Period trends in life expectancy at birth in various countries.