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# A simulation study of the role of cohort forces in mortality patterns

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**ABSTRACT**

This study uses the micro-simulation method to investigate the role of cohort forces in age-dependent mortality pattern. We test the micro mechanisms for cohort evolution and mortality selection, and how these two biological and demographic forces may interact with epidemiologic transition to shape the cohort age-dependence of mortality pattern in both early- and later-transition countries. We show that cohort evolution is due to the declining rate of mortality acceleration at the individual level, which is associated with lower initial mortality rates but not smaller variance of frailty distribution in later birth cohorts. The steeper slope of mortality acceleration at the population level among later birth cohorts is due to mortality selection mechanism associated with smaller variance of frailty distribution but not lower initial mortality rates. These two forces jointly shape the non-crossover cohort age-dependence of mortality pattern regardless of the differential mechanisms of epidemiologic transition in early- and later-transition countries.

The forces shaping the cohort age-dependent mortality pattern is one of fundamental questions in formal demography. The technophysio evolution theory (Fogel and Costa 1997) and the cohort morbidity phenotype theory (Finch and Crimmins 2004) propose that due to the long-lasting protective consequences of a better health endowment and lower exposure to infection and inflammation in early life, subsequent birth cohorts experience lower age-specific mortality rates throughout the life course. These theories are collectively referred to as the cohort evolution theories because they explain a positive correlation between young-age mortality *level* and old-age mortality *level* across cohorts (Zheng 2014).

Meanwhile, the mortality selection mechanism, a process through which the fitter survive while the frailer die over a cohort's life course, may cause later cohorts which have a smaller proportion of frail individuals in early life to have a more pronounced acceleration of mortality in late life than earlier cohorts, in which a larger proportion were frail. In other words, mortality selection mechanism suggests a negative correlation between young-age mortality *level* and the *rate* of mortality acceleration across birth cohorts. Therefore, cohort differences in life-course mortality pattern are determined by two mechanisms: cohort evolution and mortality selection. Zheng (2014) finds that among developed countries the mortality selection mechanism is not strong enough to override

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the cohort evolution mechanism; therefore old-age mortality rates are still lower among later cohorts than among earlier cohorts.

But several important questions remain. First, what is the micro mechanism for cohort evolution? Cohort evolution theories aggregate the positive correlation between early-life health and old-age health at the individual level to cohort (group) level. Yet to the contrary, the mortality selection mechanism implies that the cohort pattern may deviate from individual pattern. Understanding how these two competing forces shape mortality patterns requires not only a close examination of the micro processes over a cohort's life cycle but also an cross-cohort comparison that takes advantage of varying conditions for different cohorts of the population. Second, what is the micro mechanism for mortality selection? Several elements of the cohort life course process may determine the mechanism of mortality selection. Is the steeper slope of mortality acceleration at later cohort due to the observed lower mortality rate (Strehler and Mildvan 1960) or typically unobserved variance of frailty distribution (Vaupel 2010; Yashin et al. 2002)?

Third, is the non-crossover cohort mortality pattern jointly shaped by cohort evolution and mortality selection in countries that have experienced the epidemiologic transition in more recent periods? The main difference between early- and later-transition countries is the mechanism contributing to the mortality decline during receding pandemics. May the different historical mechanisms imply different processes of cohort evolution and mortality selection were at work in these countries? How epidemiologic transition, cohort evolution and mortality selection mechanisms interact and shape the cohort age-dependence of mortality pattern and affect historical mortality declines is still an unsolved question in demography.

Motivated by these questions, this study uses microsimulation to examine the role of cohort forces (cohort evolution and mortality selection) in age-dependent mortality pattern. More specifically, we aim to investigate the micro mechanisms for these two biological and demographic forces, and examine how they may interact with epidemiologic transition to shape the cohort age-dependence of mortality pattern in both early- and later-transition countries.

## Cohort Evolution and Mortality Selection

Cohort morbidity phenotype theory proposes that when cohorts experience lower exposure to infection and inflammation during early childhood, they reap a lower mortality risk later in life (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 2004a; Fogel and Costa 1997). Both theories suggest a positive correlation between early- and later-life mortality. Zheng (2014) further proposes that due to lower young-age mortality rates, the rate of biological aging at the individual level decelerates among later birth cohorts and contributes to the positive correlation between young- and old-age mortality rates across cohorts. These studies inspired our conjecture about cohort evolution:

*Conjecture 1: According to cohort evolution theories, cohorts with lower young age mortality rates have lower rate of mortality acceleration at the individual level, which leads to lower mortality rates over the life course at the cohort level.*

While 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., Barker et al. 1991; Bengtsson and Lindstrom 2003), the mortality selection mechanism may imply the opposite pattern on the *cohort level*. The theory of population heterogeneity proposes that cohort is composed of individuals with different physiological vulnerability to mortality, or *frailty* (Vaupel, Manton, and Stallard 1979; Vaupel and Yashin 1987). Mortality tends to remove frailer individuals from the population at earlier ages, and leave stronger individuals 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, which may lead to a discrepancy in the individual-level and cohort-level mortality patterns.

For example, cohort evolution theories suggest that later cohorts experience lower risk of infection and inflammation and have better nutrition and health capital during childhood. Therefore, the variance of frailty in later cohorts is reduced, resulting in a smaller proportion of frail individuals being selected out of the population at young ages and a larger proportion of frail individuals to survive into old age. As a result, later cohorts may exhibit higher cohort-level mortality rate at older ages, not because mortality increases for individuals, but only because of the changed composition of the population that survive into older ages. In this case, the old-age mortality rate may be potentially higher among later cohorts than among earlier cohorts, even though the individual-level mortality risk is lower in later cohorts at all ages.

A useful illustration of this 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: $\bar{u}(x) = ae^{\beta x}$ as illustrated in Figure 1, where $\bar{u}(x)$ is the cohort mortality rate at age $x$, $a$ is the initial mortality rate, $\ln(a)$ is the intercept of log cohort mortality curve $\ln(\bar{u}(x)) = \ln(a) + \beta x$, and $\beta$ refers to the rate of increase in the

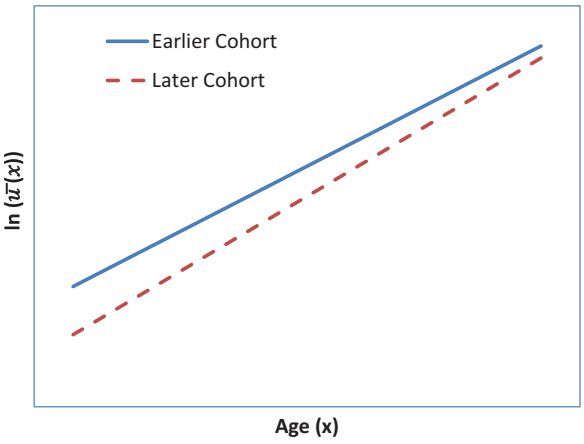



**Figure 1.** An illustration of log Gompertz mortality curve across two hypothetical cohorts.
Note: $\ln(\bar{u}(x))$ represents the logarithm transformation of age-specific mortality rate at age $x$.

cohort mortality rate, alternately described as the rate of mortality acceleration (or the slope of log mortality curve). 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 as captured by $\ln(a)$ (i.e., the cohort evolution mechanism), but also by a higher rate of mortality acceleration in later cohorts as captured by $\beta$ (i.e., the mortality selection mechanism).

The theory of population heterogeneity emphasizes that the magnitude of $\beta$ can depend on 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 is selected out of the population at an early age, a larger proportion of frail individuals survives to older ages and the mortality curve at later ages becomes steeper:

> *Conjecture 2: According to the theory of population heterogeneity, cohorts with a smaller variance of frailty at young age should have a steeper slope of mortality acceleration.*

While the population heterogeneity theory emphasizes the cross-cohort changes in the variance of frailty, Strehler and Mildvan's (1960) general theory of mortality and aging proposes a different rule governing the slope of mortality acceleration. This theory posits that the initial mortality rate $\ln(a)$ and the slope $\beta$ of the logarithm of the Gompertz mortality curve are negatively correlated. It remains unclear whether the level of initial mortality rate or the heterogeneity in frailty matters more for the cohort differences in the slope of mortality curve. We address this question by using microsimulation to examine the impact of the lowered young-age mortality on the slope of mortality and compare the result with that from Conjecture 2:

> *Conjecture 3: According to the Strehler and Mildvan's general theory of mortality and aging, cohorts with lower young-age mortality rates should have a steeper slope of cohort mortality curve.*

## The Role of Cohort Evolution and Mortality Selection in Early-Transition Countries

Among early-transition countries, the initial young-age mortality reduction during the age of receding pandemics is mainly due to socioeconomic development and consequential 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 reduces exposure to inflammation among individuals born during this period. This specific mechanism of epidemiologic transition 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 and amplifies mortality acceleration over the life course. Therefore, diminished mortality selection in early life among individuals born during the age of receding pandemics is also a result of socioeconomic development, and this socioeconomic development decreases the variance of the frailty distribution at young ages across cohorts gradually rather than abruptly. Thus, as Zheng (2014) suggested, the slope of the mortality curve will not be steep enough for older-age mortality rates among

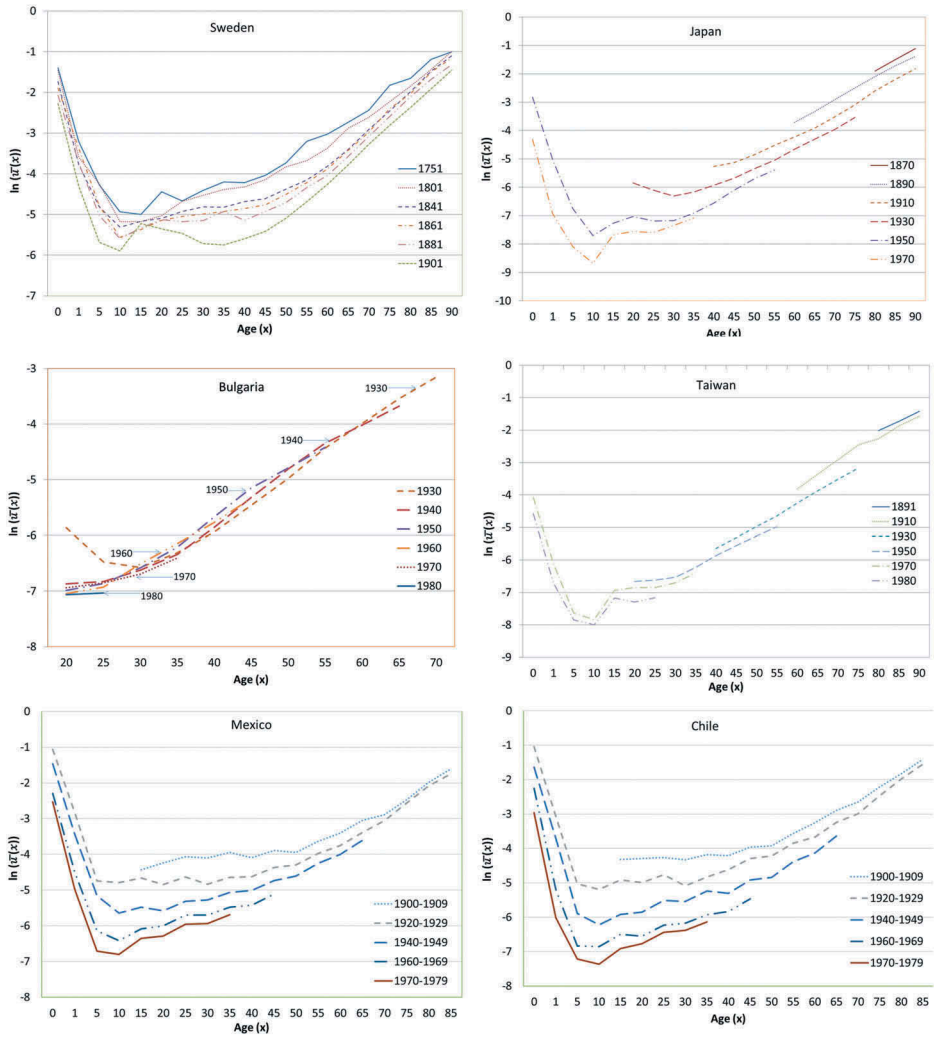



**Figure 2.** Cohort changes in age-specific mortality rates over the life course in various countries.

Note: Data for Sweden, Japan, Bulgaria and Taiwan are from the Human Mortality Database (HMD). Data for Mexico and Chile are from Latin American Mortality Database (LAMBdA) (Palloni et al. 2014). Cohort age-specific mortality rates for Mexico and Chile are constructed from the period data provided by LAMBdA so they may not be completely accurate. $\ln(\bar{u}(x))$ represents the logarithm transformation of age-specific mortality rate at age x.

later cohorts to surpass those among earlier cohorts. This pattern is represented by the Sweden and Japan Panels of Figure 2.

These arguments lead to a fourth conjecture:

*Conjecture 4: Due to cohort evolution, among early-transition countries later cohorts with lower young age mortality rates should have lower mortality rates throughout the life course than preceding cohorts despite the function of mortality selection.*

## The Role of Cohort Evolution and Mortality Selection in Later-Transition Countries

In contrast, 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 might be no or weak effect of the cohort evolution mechanism. On the other hand, the mortality selection mechanism might have become a dominant determinant of cohort trends in the age-dependence of mortality rates. Vaccination, diffusion of drugs and medical technologies allow an increasing share of frail individuals to survive into adulthood who were nonetheless exposed to infection and inflammation in early life, the negative effect of which will affect them over the life course and may potentially increase mortality rates at old ages compared to earlier birth cohorts. Palloni and Souza (2013) posited that weakened mortality selection in the stage of receding pandemics in Latin American and the Caribbean countries may cause life expectancy at older ages to stall or even decline in the future.

A more complicated pattern has been observed in some Eastern European countries such as Bulgaria, where older-age mortality rates exhibit substantial two-step crossover across cohorts born at the age of receding pandemics (1930–1960) (see Figure 2). The 1940 birth cohort intersects with the 1930 birth cohort at ages 40 and 60. The intersection points between the 1940 and 1950 birth cohorts appear earlier in the life-course: at ages 30 and 50, respectively. The intersection points between the 1950 and 1960 birth cohorts appear even earlier in the life-course. Past the 1970 birth cohort, and especially past the 1980 birth cohort, mortality curves no longer cross over those of earlier cohorts. Bulgaria entered the age of receding pandemics in the 1930s, even later than Japan did, and went through this stage of transition at an even faster rate due to medical advancements. Its economies were largely rural; technology, capital and access to markets were lacking; and the Great Depression devastated every economic sector (Kaser and Radice 1987). Against this background of economic stagnation, medical treatment emerged as the main cause of initial declines in young-age mortality from the 1930s through the 1960s, and included the diffusion of vaccinations, drugs, and medical technology. Further mortality declines after the 1970s, when Bulgaria started transitioning to the age of degenerative and man-made diseases (Vallin and France 2004), became increasingly attributable to socioeconomic development, improved living standards, and improved nutrition.

But a different pattern is found in another later-transition country, Taiwan (Figure 2). There is no crossover in any birth cohorts from 1891 to 1980, which resembles the pattern in Sweden and Japan. Palloni and Souza (2013) presumed mortality crossover in old ages across cohorts in Latin American countries, but based on our reconstruction of period mortality data to cohort age-specific mortality rates in Mexico and Chile, no mortality crossover has been found either. These counterexamples question the cohort frailty arguments about mortality crossover at old age in later transition countries. The competing argument is there may be some spill-over effects from reduction in the intensity and duration of illnesses induced by medical technology. There is a synergistic relationship between nutrition and infection (Scrimshaw and SanGiovanni 1997). Improved nutrition can increase resistance to infection and improve recovery from illness (Fogel 2004b;

Scrimshaw 1992, 1997), while lower contraction rate of infectious diseases (due to vaccination) and reduced duration and severity of infection and better recovery from diseases (due to medical treatment) can be translated to better nutrition status even in the absence of improvement in nutrition intake (Palloni and Souza 2013). Thus, even though living standards and nutrition intakes might not significantly improve at the stage of receding pandemics in the later-transition countries, people born during that time might be relatively better off than preceding cohorts due to a significant reduction in nutrition loss caused by medical technologies. In this case, cohort evolution mechanism may still function in that stage. Following this argument, the age-specific mortality curve should not have crossed over across birth cohorts if only cohort mechanisms are working. In other words, the two-step cross-over in some Eastern European countries should have been caused by period shocks.

These arguments lead to a fifth and sixth conjectures:

*Conjecture 5: Medical improvement-induced receding pandemics among later-transition countries saved an increasing share of frail individuals who had been exposed to adverse early conditions and allowed them to survive to adulthood, which led to significantly larger share of frail individuals at older ages and caused the mortality crossover across birth cohorts.*

*Conjecture 6: Medical improvement-induced receding pandemics among later-transition countries reduced exposure, duration and severity of infection and improved recovery from diseases, which translated to better nutrition status and enabled cohort evolution mechanism. Therefore, the mortality curve should not cross over across birth cohorts. The observed or expected crossover was driven by period trends.*

## Research Design and Model Setup

In order to test the above six conjectures, we use the microsimulation method to simulate the observed cohort age-dependent mortality pattern from age 30 to 90 on the individual hazard curve only allowing the cohort mechanisms to function in early-transition countries represented by Sweden and later-transition countries represented by Bulgaria. We start with age 30 because cohort mortality curve follows a Gompertz law beginning at that age. We restrict the upper end to age 90 due to uncertainty of hazard function at very old ages (Crimmins and Finch 2006). We only present simulations on male mortality pattern for the sake of space. Empirical mortality data on these countries are available from the Human Mortality Database (http://www.mortality.org/).

### *Basic Mathematical Formulation*

Following Vaupel, Manton, and Stallard (1979), suppose that individuals in a cohort differ from each other in the value of frailty (denoted as $z$) characterizing their susceptibility to death, such that their force of mortality conditional on $z$ is as follows:

$$u_i(x) = z_i u_0(x) \tag{1}$$

where $u_i(x)$ is the force of mortality for individual $i$ at instantaneous age $x$, $z_i$ is frailty for individual $i$ at initial age, $u_0(x)$ is the unobserved baseline hazard function with frailty of 1. An individual with a frailty of 1 can be called a "standard" individual. An individual with

frailty of 1.5 is one and half times more likely to die at any particular age than the standard individual. Frailty $z_i$ follows a gamma distribution at initial age, with *p.d.f.*:

$$f_0(z) = \lambda^k z^{k-1} e^{-\lambda z}/\Gamma(k) \tag{2}$$

where $\lambda$ and $k$ are the parameters of the distribution. The mean and variance of a gamma variate are given by

$$\bar{z} = k/\lambda \tag{3a}$$

$$\sigma^2 = k/\lambda^2 \tag{3b}$$

We follow earlier work to set mean frailty $\bar{z}$ as 1 (which is also the value of frailty for a "standard" individual). Thus the shape parameter $k$ equals $\lambda$, and the variance of frailty distribution $\sigma^2$ equals the inverse of $k$.

The mortality selection mechanism yields a cohort-level force of mortality $\bar{u}(x)$ as follows:

$$\bar{u}(x) = \frac{u_0(x)}{1 + \sigma^2 H(x)}, \tag{4}$$

where the cumulative hazard function from initial age to age $x$ is $H(x) = \int_0^x u_0(t)dt$ (Vaupel, Manton, and Stallard 1979). From formula (4), it is obvious that cohort mortality function $\bar{u}(x)$ deviates from individual hazard function $u_0(x)$. The higher value of the variance of frailty distribution $\sigma^2$, the more the slope of $\bar{u}(x)$ deviates from that of $u_0(x)$; the deviation also increases with age as $H(x)$ is an increasing function of $x$ (Yashin et al. 2002).

Even though cohort mortality function $\bar{u}(x)$ in discrete time is observed and is often parameterized as a Gompertz function, there are limited empirical data to support any conjecture about individual hazard curve. The theory of population heterogeneity posits that death selectively removes the frailest members of a cohort so that mortality rate at cohort level becomes increasingly dominated by robust members over the life course (Vaupel, Manton, and Stallard 1979; Vaupel and Yashin 1985a, 1985b). This means that individual hazard curve should be steeper than cohort mortality curve, or individuals "age" faster than heterogeneous cohorts (Vaupel and Yashin 1985a). This conclusion is also inferred from formula (4). Yashin and Iachine (1997) infer the underlying individual hazard function from the semiparametric shared-frailty model using Danish twins data, and findings support the assumption that individuals age faster than cohorts.

Following Vaupel and Yashin (1985a) with some modification, we assume individual baseline hazard function as follows:

$$u_0(x) = ae^{bx} e^{\frac{a\left(e^{bx}-1\right)}{b}}. \tag{5}$$

In this case, the force of mortality for individual $i$ at instantaneous age $x$ is follows:

$$u_i(x) = z_i a e^{(bx)} e^{\frac{a\left(e^{bx}-1\right)}{b}}, \tag{6a}$$

or

$$\ln(u_i(x)) = \ln(a) + bx + \frac{a\left(e^{bx}-1\right)}{b} + \ln(z_i). \tag{6b}$$

The rate of increase in mortality rate at age $x$ is the derivative of $\ln(u_i(x))$ at age $x$, i.e., $\frac{d\ln(u_i(x))}{dx} = b + ae^{bx}$. In other words, the rate of individual mortality acceleration itself is a Gompertz function of age $x$; it increases as age increases. By assuming individual hazard function as formula (6a) or (6b), the simulated cohort mortality curve will follow a Gompertz function $\bar{u}(x) = ae^{\beta x}$. For a detailed description of simulation procedure, please refer to Appendix A and B.

In reality, the distribution of frailty may be more complicated than what was assumed in the main models (Wrigley-Field 2014). It is beyond the scope of this paper to conduct a comprehensive comparison of these various specifications, but it is helpful to explore some potential implications and point to possible directions for future work. Several auxiliary simulations are presented in this spirit. In Appendix C, we experiment with two sets of models, in which (1) the frailty distribution is allowed to vary by subgroups in the population, and (2) the severity of frailty is allowed to vary across the life cycle, both linearly and non-linearly. The results are overall consistent with our simplified model, and we note that further development of the model to incorporate variations across subgroups is needed in future research.

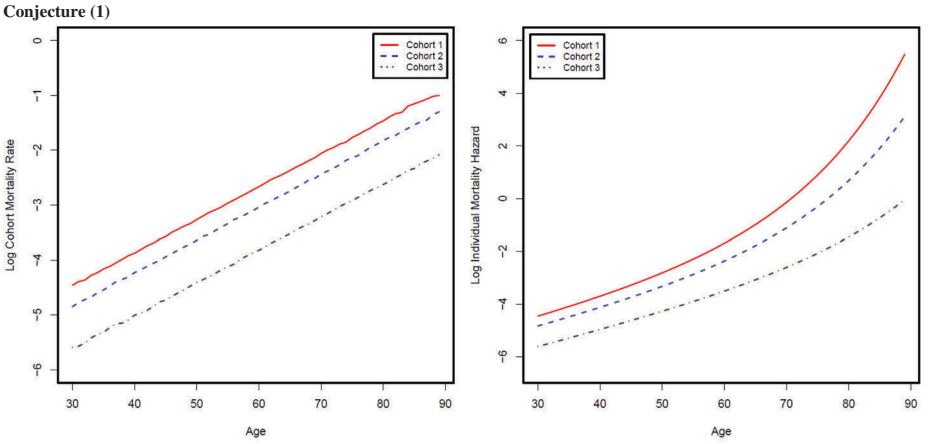
## Results

### Micro Mechanisms for Cohort Evolution

In order to examine the micro mechanisms for cohort evolution (Conjecture 1), we do one scenario of simulation as presented in Figure 3. We set variance of frailty distribution $\sigma^2$ and parameter $b$ fixed at three cohorts and only allow $\ln(a)$ decreases. The simulation result on the left panel supports the cohort evolution theories that cohorts with lower young-age mortality rates have lower mortality rates over the life course. The right panel portrays the mortality curves of the standard individual (frailty level $z_i = 1$) among these three cohorts. The standard individuals among later cohorts with lower mortality rates at age 30 have smaller rates of mortality acceleration that cause them to have lower mortality rates over the life course. According to the formula for rate of mortality acceleration at the individual level, $b + ae^{bx}$, it is obvious that it is affected by initial mortality rate $a$, but not affected by the variance of frailty distribution $\sigma^2$. This provides a micro explanation for the cohort evolution mechanism portrayed in the left panel. Overall these simulations support Conjecture 1.

### Micro Mechanisms for Mortality Selection

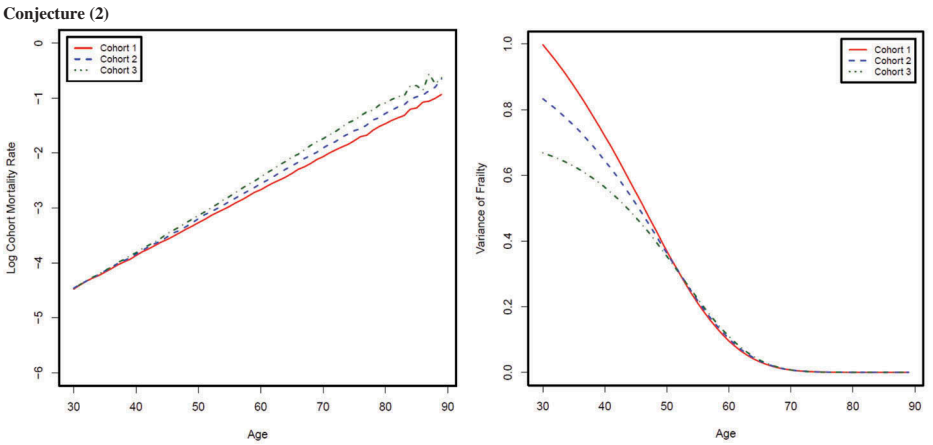
The left panel of Figure 3 also tests Conjecture 3. It finds that cohorts with lower young-age mortality rates do not have a steeper but paralleling slope of mortality acceleration $\beta$ at




| Cohort | $\ln(a)$ | $\sigma^2$ | $b$ |
|---|---|---|---|
| Cohort 1 | -4.529 | 1.00 | 0.06 |
| Cohort 2 | -4.901 | 1.00 | 0.06 |
| Cohort 3 | -5.678 | 1.00 | 0.06 |

| Individual | $\ln(a)$ | $z_i$ | $b$ |
|---|---|---|---|
| Cohort 1 | -4.529 | 1.00 | 0.06 |
| Cohort 2 | -4.901 | 1.00 | 0.06 |
| Cohort 3 | -5.678 | 1.00 | 0.06 |

**Figure 3.** Testing micro mechanisms for cohort evolution.




| Cohort | $\ln(a)$ | $\sigma^2$ | $b$ |
|---|---|---|---|
| Cohort 1 | -4.529 | 1.00 | 0.06 |
| Cohort 2 | -4.529 | 0.83 | 0.06 |
| Cohort 3 | -4.529 | 0.67 | 0.06 |

**Figure 4.** Testing micro mechanisms for mortality selection.

the cohort level. This refutes Conjecture 3. In order to test Conjecture 2, we set $\ln(a)$ and $b$ fixed at three cohorts and only allow variance of frailty distribution $\sigma^2$ decreases as presented in Figure 4. The simulation result on the left panel supports the proposition from the theory of population heterogeneity that cohorts with a smaller variance of frailty at young age do have a steeper slope of mortality acceleration. Therefore, opposite to the forces driving the rate of mortality acceleration at the individual level, the rate of mortality acceleration at the cohort level $\beta$ is affected by the variance of frailty distribution $\sigma^2$, but not by initial mortality rate $a$. The right panel further portrays the micro forces driving the

steeper slope among cohorts with smaller $\sigma^2$ on the left panel: variance of frailty declines over the life course and it declines at a lower rate among cohorts with a smaller variance of frailty at initial age. Thus, a smaller proportion of frail individuals are selected out of the population at initial age, leaving a relatively larger proportion of frail individuals to survive to older ages and making the mortality curve steeper. These simulations suggest the negative correlation between $\ln(a)$ and $\beta$ observed in Strehler and Mildvan (1960) is due to the negative correlation between $\sigma^2$ and $\beta$ and the general positive correlation between $\ln(a)$ and $\sigma^2$, meaning that the cohorts with lower initial mortality rates tend to have smaller variance of frailty.

### Simulating Swedish Male Mortality Pattern

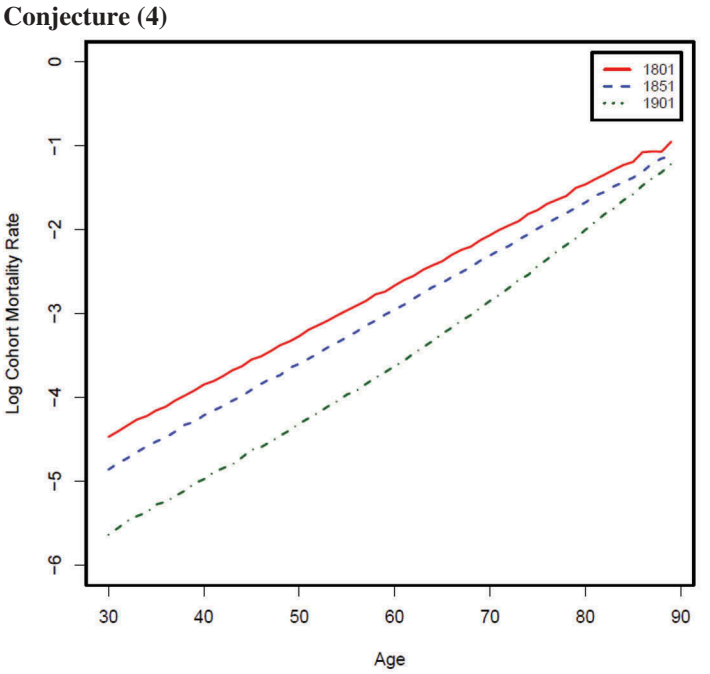
In order to test Conjecture 4, we simulate the male mortality pattern in the cohorts of 1801, 1851, and 1901 in Sweden. We set $\ln(a)$ at the levels of log mortality rates at age 30 among these three cohorts to facilitate cohort evolution mechanism, and let $b$ equal 0.06.[1] We let $\sigma^2$ decrease across these three cohorts to facilitate the mortality selection mechanism suggested by Conjecture 2. The left panel of Figure 5 portrays a cohort pattern from this simulation, which replicates the observed pattern. This supports Conjecture 4 that the Swedish cohort mortality pattern is a joint outcome of cohort evolution and mortality selection mechanisms.

### Simulating Bulgarian Male Mortality Pattern

In order to test Conjectures 5 and 6, we try to simulate the male mortality pattern in the 1930 and 1940 cohorts in Bulgaria. Conjecture 5 implies that the variance of frailty distribution at early adulthood among later cohorts could have been the same or even larger than that of proceeding cohorts. So we simulate five scenarios where we set $\ln(a)$ at the levels of log mortality rates at age 30 among these two birth cohorts as shown in Figure 6. We let $\sigma^2$ be constant ($\sigma^2 = 2$) or increase ($\sigma^2 = 4$, $\sigma^2 = 8$) across these two cohorts. But contrary to the predictions in Conjecture 5, the simulated 1940 cohort either has a paralleling or flatter slope than the 1930 cohort. In any case, there is no mortality crossover. These three scenarios reject Conjecture 5.

In fact, if we only allow cohort forces in effect, we have to substantially *decrease* instead of increase the variance of frailty distribution among the 1940 cohort in order to create the first mortality crossover at age 40 as shown in scenarios ($\sigma^2 = 0.5$, $\sigma^2 = 0.25$). Because the variance of frailty is unobserved, there is no way to use empirical data to either support or dispute this decrease in variance within 10 years of birth cohorts. But compared to the decrease in variance within every 50 years of cohorts in the simulations for Sweden, we note that this amount of decrease to be unrealistic for Bulgaria. These simulations demonstrate that there is no way to produce the empirical cohort pattern in Bulgaria by only allowing cohort mechanisms in effect. The mortality crossover across birth cohorts must have been caused by period shocks.

Figure 7 shows the trends in age-specific mortality rates ages 20–74 across time periods from 1947 through 2010 in Bulgaria. Mortality rates within the age groups 30–59 increased from the mid-1960s until the mid-1990s, which caused later cohorts to have higher mortality




| Cohort | $\ln(a)$ | $\sigma^2$ | $b$ |
|---|---|---|---|
| Cohort 1801 | -4.529 | 1.00 | 0.06 |
| Cohort 1851 | -4.901 | 0.83 | 0.06 |
| Cohort 1901 | -5.678 | 0.33 | 0.06 |

**Figure 5.** Simulate Swedish male mortality pattern with cohort evolution and mortality selection mechanisms.

rates than earlier cohorts at each age within this age range. Mortality rates before age 30 and after age 60, however, did not significantly increase from the mid-1960s through the mid-1990s. This may have generated the two-step crossover in mortality rates across cohorts.

## Discussion and Conclusion

This study uses microsimulation method based on mathematical models to investigate the role of cohort forces (cohort evolution and mortality selection) in age-dependent mortality pattern. We show that cohort evolution is due to the declining rate of mortality acceleration at the individual level, which is associated with lower initial mortality rates but not smaller variance of frailty distribution in later birth cohorts. The steeper slope of mortality acceleration at the population level among later cohorts is due to the mortality selection mechanism associated with a smaller variance of frailty distribution but not lower initial mortality rates among later cohorts. Our simulations further imply that even though the prominent mechanisms for receding pandemics are different between early- and later-transition countries, their cohort patterns in age-dependence of mortality rates still resemble each other. That is because vaccination and medical technology can still trigger the emergence of cohort evolution mechanisms due to the gained nutrition status from the reduction of exposure, duration and

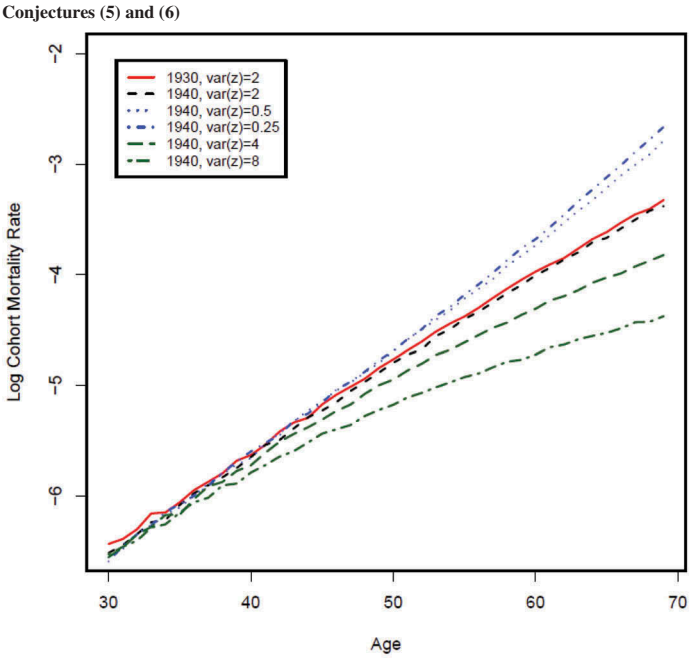



**Figure 6.** Simulate Bulgarian male mortality patterns.

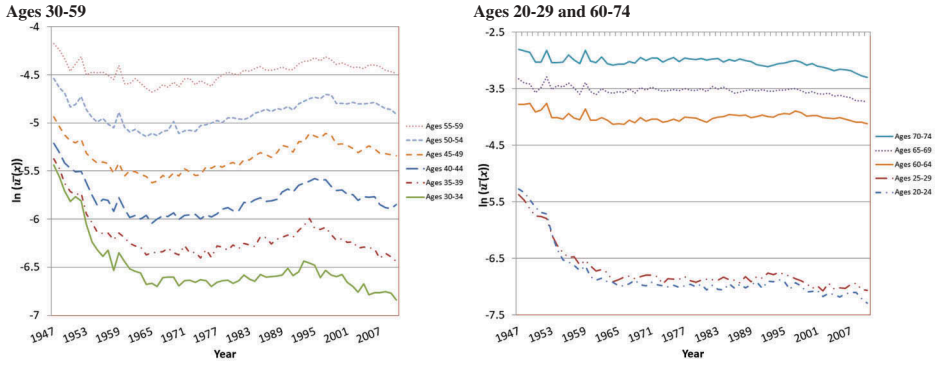



**Figure 7.** Period changes in age-specific mortality rates ages 20–74 in Bulgaria, 1947–2010.

Note: Data are from the Human Mortality Database. $\ln(\bar{u}(x))$ represents the logarithm transformation of age-specific mortality rate at age x.

severity of infection caused by them (Palloni and Souza 2013; Scrimshaw 1992). Frailty arguments rely on the long lasting negative consequence of poor nutrition and exposure to infection and inflammation in early life on adult health compared to those with good nutrition and no or less exposure to infection and inflammation. But the reference group for historical cohort comparison is earlier cohorts with similar or even worse nutrition and exposure to infection and inflammation. Compared to these earlier cohorts, later cohorts are still better off because lower contraction rate of infectious diseases (due to vaccination) and reduced duration

and severity of infection and better recovery from diseases (due to medical treatment) can be translated to better nutrition status even in the absence of improvement in nutrition intake.

The mortality crossover in some later transition countries should not have been caused by the mortality selection mechanism, but instead should have been caused by period shocks. Particularly, in the context of Central and Eastern Europe, economic stagnation during cold war have contributed to the increasing middle-age (age 30–59) mortality rates (e.g., suicide rate) after the 1970s. Economic difficulties might also have held back the dissemination of expensive new technologies (Vallin and France 2004). Moreover, health system failed to respond to the epidemiological transition of the population (Dimova et al. 2012). The highly centralized social system, which succeeded in fighting infectious diseases before the 1970s, worked to their disadvantage in getting individuals to take responsibility for their own health through behavioral and lifestyle changes (Vallin and France 2004). Bulgaria had experienced a dramatic increase in the consumption of ethanol and animal fat, tobacco and alcohol since the early 1970s, that exacerbated the upward trend in mortality rates from cancer, stroke, cardiovascular disease (CVD) and non-CVD among people in early and middle adulthood, especially in men (Caselli, Meslé, and Vallin 2002; Georgieva et al. 2002; Kubik et al. 1995). This has caused the two-step mortality crossover within this age group across successive birth cohorts. Mortality rates, especially the cardiovascular death rate, has declined sharply in Bulgaria since the mid-1990s (Karanikolos, Adany, and Martin 2017; Meslé 2002), so mortality rates stop crossing over among the recent birth cohorts.

This study implies that observed or expected rising mortality in middle or late adulthood in the later period among later-transition countries might not be due to reduced mortality selection in early life in early periods. Studies have found that, in low- and middle-income countries, middle-aged adults are especially vulnerable to chronic diseases. Individuals in these countries tend to develop chronic diseases at younger ages, suffer from more complications, and die sooner than individuals in high-income countries (Vallin and France 2004; World Health Organization 2005). Our simulation, however, implies that rising mortality conditions in middle and old age in later periods should not be caused by blunted mortality selection in early periods because medicine-induced infection alleviation can be translated into better nutrition status. Instead, period factors – including changes in air pollution, environmental degradation, economic stagnation, urbanization, nutrition transition, unhealthy diet, and a sedentary lifestyle – might have caused increased chronic disease burdens in later-transition countries (Caballero and Popkin 2002; Chen et al. 2013; Pimentel et al. 2007; Smith 2000; Vallin and France 2004; Yusuf et al. 2001).

## Notes

1. Following Vaupel and Yashin (1985a), we set $b$ fixed at certain values. We tried different values of $b$; 0.06 best approximates the observed mortality pattern.
2. We also experimented with holding the natural logarithm of $z$ constant across cohorts instead of holding the value of $z$ constant. Results show no substantial change with the alternative specification.

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## Appendix A. Simulation procedure

We start our simulation from age 30 because cohort mortality curve follows a Gompertz curve beginning around that age (Gompertz 1825). We let $a$ in (6b) equal the observed cohort mortality rate at age 30, which represents the mean of mortality hazards across individuals within this cohort at initial age. So it equals the mortality hazard for a "standard" individual with frailty of 1.[2]

Our simulation proceeds in every 1 year of age among a hypothetical population of 1,000,000 individuals. On the individual level, the force of mortality $u_i(x)$ should change continuously within each tiny age interval. But we assume it is constant within each 1 year of age, denoted as $u_i^*$ (so-called piecewise-constant force of mortality assumption). Piecewise-constant force of mortality is a reasonable assumption for a small age interval like 1 year. It also has computational advantages: it can easily convert hazard rate to probability and save enormous computation time. Under this assumption, the central mortality rate with each year of age for every individual $i$, $m_x^i$, equals $u_i^*$. Then the probability of surviving between age $x$ and $x + 1$ for every individual $i$, denoted as $p_x^i$, equals $e^{-m_x^i}$ (Preston, Heuveline, and Guillot 2001). The simulation process proceeds from age to age. At each age, we calculate the probability of dying for each surviving individual at age $x$ as $q_x^i = 1 - p_x^i$, and then perform a random draw following a binomial distribution where the probability of getting a value of 1 equals $q_x^i$. Individuals who receive a value of 1 will die between age $x$ and $x+ 1$. We stop the simulation at age 90 for the Swedish population as mortality patterns past age 90 do not typically follow the Gompertz curve (Vaupel 1997). We stop the simulation at age 70 for the Bulgarian population to more closely examine the pattern within this age range.

After generating the simulated cohort survival data, we next calculate the cohort age-dependent mortality rate. An important step here is to calculate the person-years. We hold the same piecewise-constant force of mortality assumption like the one in the simulation procedure. Under this assumption, person-years within each 1-year age interval equals $\frac{(l_{x+1}-l_x)}{\ln\left(\frac{l_{x+1}}{l_x}\right)}$, where $l_x$ is the number of people left alive at age $x$ (Preston, Heuveline, and Guillot 2001). The cohort age-dependent mortality rates are calculated as the number of dead divided by this measure of person-years within each 1-year age interval.

In different rounds of simulations, we alter the values of initial mortality rate (ln(a)) and the variance of frailty parameter ($z_i$) (i.e., $\sigma^2$) in specifying the individual-level mortality hazard to experiment with different cohort conditions. We then compare the resulting cohort mortality rate to decide what micro-level changes in mortality patterns are likely to occur in order to produce the observed cohort-level mortality patterns.

## Appendix B. The relationship between individual and cohort mortality functions

Before testing the six conjectures, we test assumptions about the unobserved individual- hazard function specified in formulas (6b). Some studies have assumed individual hazard curve as a Gompertz function in human populations (Service 2000; Wrigley-Field 2014). The model can be specified as follows:

$$u_0(x) = ae^{bx}, \tag{7}$$

where $a$ is the hazard at initial age and $b$ is the rate of mortality acceleration. Replacing $u_0(x)$ in formula (1) with formula (7), we get the force of mortality for individual $i$ at instantaneous age $x$

$$u_i(x) = z_i ae^{bx}, \tag{8a}$$

or

$$\ln(u_i(x)) = \ln(a) + bx + \ln(z_i). \tag{8b}$$

The rate of increase in mortality rate at age $x$ is the derivative of $\ln(u_i(x))$ at age $x$, i.e., $\frac{d\ln(u_i(x))}{dx} = b$. In other words, individual log mortality curve is a linear function of $x$ with fixed slope $b$. However, by assuming individual hazard function as formula (8a) or (8b), the simulated cohort mortality curve will "age" even slower than Gompertz, which does not accurately characterize the empirical cohort mortality pattern.

The left panel of Figure B1 portrays the simulated cohort mortality pattern from age 30 to 90 based on these two different hazard function assumptions (i.e., 6b and 8b). Cohort 1 is based on formula (6b) and cohort 2 is based on formula (8b); we set the same values of log mortality rate at age 30 (= −4.529) and variance of frailty distribution $\sigma^2$ (= 1) and $b$ (= 0.06). Cohort 1 observes a linear slope of log mortality rate at cohort level, which is consistent with the empirical pattern (i.e., Gompertz mortality curve). In contrast, cohort 2 observes a declining rate of mortality increase during this age range, which is inconsistent with the empirical pattern. In other words, Gompertz assumption of individual hazard function will generate a cohort which ages slower than Gompertz. The difference in these simulated cohort patterns is not contingent on different values of $\ln(a)$, $\sigma^2$ and $b$. This leads to our decision to use formula (6b) as a basis for further simulation.

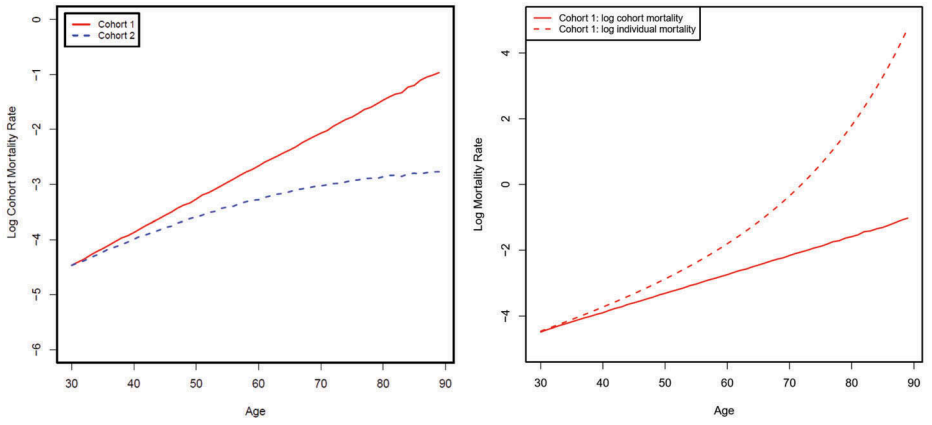
The right panel of Figure B1 further portrays the relationship between individual hazard curve (based on formula 6b) and the corresponding cohort mortality curve. For any given birth cohort, there are numerous individual hazard curves with different frailties $z_i$ but the same shape of mortality acceleration ($b + ae^{bx}$). We present the curve for the "standard" individual with frailty level $z_i = 1$, which puts the initial mortality rate at age 30 in line with cohort mortality rate at age 30 and makes the comparison more straightforward. As demonstrated in this figure, cohort log mortality curve follows a linear function. More importantly, cohort mortality rate increases at a slower rate than the individual mortality rate, which is due to the mortality selection mechanism that removes frailest individuals at earlier ages and causes the cohort mortality pattern to be increasingly dominated by robust individuals over the life course.

## Appendix C. Auxiliary Simulations Demonstrating Mortality Patterns under More Complex Assumptions about Frailty Distribution in the Population

In our main simulations, we have assumed that the frailty for each cohort is distributed as a Gamma distribution. In keeping with the main aim of our paper, which is to examine the impact of the mortality selection mechanism on changes in cohort mortality patterns, we have focused on the changes in the *variance* of the frailty distribution across cohorts. However, as Wrigley-Field (2014) suggested, in reality, the distribution of frailty may be more complicated than what was assumed in the main models. It is beyond the scope of this paper to conduct a comprehensive comparison of these various (and arguably more realistic) specifications, but we think it is helpful to explore some of the potential implications and point to possible directions for future work. The following auxiliary simulations are presented in this spirit. Here, we experiment with two sets of models, in which (1) the frailty distribution is allowed to vary by subgroups in the population, and (2) the severity of frailty is allowed to vary across the life cycle, both linearly and non-linearly.

*Auxiliary Simulations 1: Different Frailty Distributions across Subgroups of the Population*

In the first set of auxiliary simulations, we explore the potential subgroup variations in the distribution of frailty. To do this, in Figure C1, we compare the simulation results based on our original specification in the main analysis with those based on the assumption that the population is composed of two subgroups, with the percentage of the majority group being 90% (the left plot) and 70% (the right plot), respectively. In each plot, we further explored three possible frailty




| Cohort | Assumptions for individual hazard function | $\ln(a)$ | $\sigma^2$ | $b$ |
|---|---|---|---|---|
| Cohort 1 | $\ln(u_i(x)) = \ln(a) + bx + \frac{a(e^{bx}-1)}{b} + \ln(z_i)$ | -4.529 | 1.00 | 0.06 |
| Cohort 2 | $\ln(u_i(x)) = \ln(a) + bx + \ln(z_i)$ | -4.529 | 1.00 | 0.06 |

**Figure B1.** Simulated cohort age-dependence of mortality pattern based on different assumptions of unobserved individual hazard function.

distributions of the added subgroup: (1) adding a subgroup with a higher frailty variance than the majority group; (2) adding a subgroup with a higher baseline mortality rate; (3) adding a subgroup with both a higher frailty variance and higher baseline mortality rate.

Figure C1 presents these results. Consistent with our main results, adding a higher variance group leads to a flatter cohort mortality curve over age (e.g., the red dashed line is flatter than the black solid line in the original specification). As can be expected, the addition of a subgroup with higher baseline mortality rate will shift the mortality curve upwards, because the "average individual" in this cohort becomes frailer. In addition, the larger the minority subgroup (30% vs. 10%), the more pronounced the difference is between the new and original specifications. One interesting observation from these results is that, the effect of adding a high variance subgroup is stronger when the added subgroup's baseline mortality rate is the same as the majority subgroup, and becomes much milder when the added subgroup's baseline mortality rate is relatively higher (e.g., the difference between the green and blue lines is smaller than that between the red and black lines). This suggest that, when the population is composed of multiple subgroups, the impact of frailty variance and mortality selection may also depend on the baseline frailty of these subgroups, a direction worth exploring in future research. But overall, the inverse relationship between variance of frailty and slope of mortality acceleration at the population level are supported in these simulations.

*Auxiliary Simulations 2: Age-varying Severity of Frailty*

In the second set of auxiliary simulations, we explore the implications of allowing the severity of frailty to vary linearly and non-linearly with age. This may be a reasonable assumption, because the risks of frailty may evolve in complex ways not just across subgroups, but also within a cohorts' life cycle. We present the results from our auxiliary simulations in Figure C2. In the left plot, we allow frailty to increase linearly with age, and compare the resulting cohort mortality curves under relatively low and high rates of the linear increase with the curve from our original specification, which assumes a constant severity of frailty over the life cycle. As the results demonstrate, increasing severity of frailty increases the steepness of the cohort mortality curve, implying that

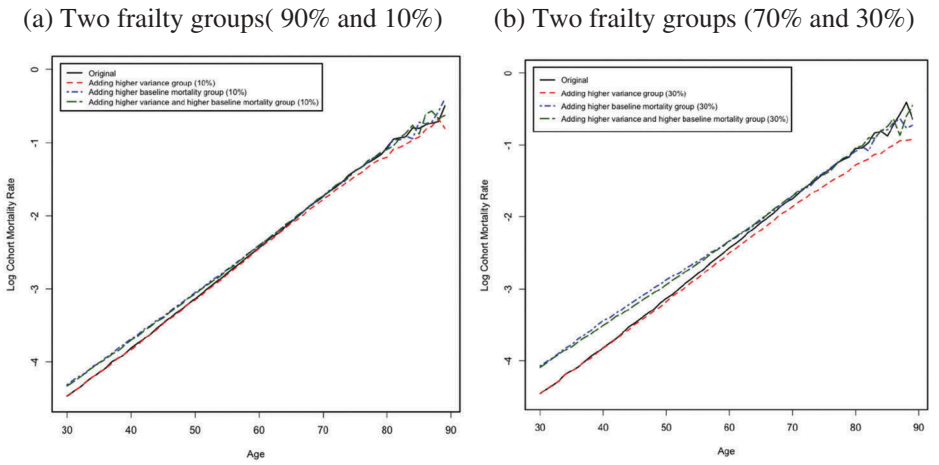



**Figure C1.** Simulation Results with Two Frailty Groups.

this increase will lead to a faster mortality process. In addition, the plot also shows that this effect is stronger before age 60, and becomes milder at older ages. In other words, the results suggest that the increasing severity of frailty on the individual level affects the mortality rate on the cohort level the most during middle life.

The linear increase in the severity of frailty may be unrealistic, in that the age-varying severity of frailty may reach its peak at a certain point in the life course, and the severity of frailty for different cohorts/individuals may also peak at different ages. We therefore include an additional round of simulations, in which we specify the individual-level severity of frailty as a *quadratic* function of age, with the coefficient on the squared term of age being negative. This non-linear specification allows the severity of frailty to increase at a faster rate during younger ages, but the increase will slow down at older ages. The right plot of Figure C2 shows the results from two such simulations, one with a relatively earlier peaking (red dashed line) and the other with later peaking (blue dash-dotted line). As may be expected, the cohort mortality curve in the earlier-peaking case goes up faster than that in the later-peaking case, because the severity of frailty took off at a faster rate in the former. Yet, at around age 70, the two curves cross over each other, with the mortality curve for the later-peaking case taking a higher position. This is driven by the fact that severity of frailty in the earlier-peaking case has slowed down at later ages, while the frailty in the later-peaking case kept climbing after this age.

In this study, we focus on the demographic frailty and the variance of frailty as the key factor affecting cohort mortality patterns, rather than biological frailty. Although demographic frailty is fixed at birth, since individual mortality risk is a product between its demographic frailty as well as standard individual (frailty of 1) mortality risk, our simple model does imply biological frailty varies over life course. Our model also produces the linear mortality curve at cohort level at the log scale, consistent with empirical pattern. As these auxiliary simulations demonstrate, it may not be straightforward to replicate the observed linear log mortality curve at the population level under the assumption of age-varying severity of frailty, and thus additional assumptions or alternative specifications may be introduced to better replicate the empirical pattern. While future work can more carefully explore the more complex, dynamic patterns of individual biological frailty, it should be mindful about the implication of these patterns on the empirical cohort mortality pattern.

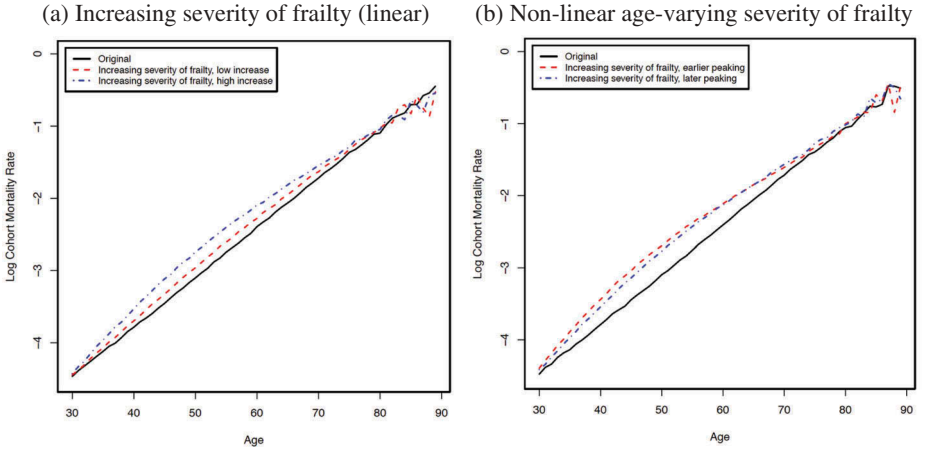



**Figure C2.** Simulation Results with Linear and Non-linear Age-varying Severity of Frailty.