Heterogeneity's Ruses: The Impact of Mortality Selection on Dynamics of Health Disparity and Life Expectancy

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Unobserved individual frailty is prevalent and consequential in the population pattern of health and mortality. This study investigates how unobserved frailty may complicate the cohort trend in health disparity and life expectancy. We use the micro-simulation method based on a mathematical model of individual mortality hazard and group frailty distribution to generate an aggregate pattern of age-dependent mortality rates for less-educated (high school or less) and better-educated (any college) groups. We alter the values of the frailty variance to generate hypothetical mortality patterns and life expectancy to test the effect of unobserved frailty. Our exercises reveal that mortality selection can cause convergence, parallels, and divergence in age-dependent mortality at the group level, even though cumulative advantage mechanism works at the individual level. In the absence of a change in the individual mortality curve, life expectancy can increase or decrease across birth cohorts due to the increase or decrease in variance of frailty distribution. A life expectancy gap between the more and less educated groups can either widen or narrow across cohorts as a result of the change in frailty variance. Using the number of diseases before age 17 from Panel Studies of Income Dynamics and National Health and Nutrition Examination Survey data as a proxy for frailty and cohort changes in the age-dependent mortality pattern from National Health Interview Survey data, we find mortality selection has contributed up to 24% of the widening life expectancy gap between these two education groups from the 1950s to 1960s birth cohorts in the United States.

Key words: Unobserved frailty, mortality selection, cumulative advantage, birth cohorts, life expectancy, simulation

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Introduction

Unobserved individual frailty is prevalent and consequential in the population patterns of health and mortality. The theory of population heterogeneity proposes that populations are composed of individuals or subpopulations that vary in 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 to leave stronger individuals to survive to older ages. Therefore, age-specific mortality rate within any given birth cohort becomes increasingly dominated by robust individuals as the cohort ages. This *mortality selection mechanism* leads to the deviation of a population-level mortality pattern from the individual-level mortality pattern, and it may produce a population mortality pattern surprisingly different from the individual pattern (Vaupel et al. 1979). For example, it can produce cohort mortality deceleration at older ages at the population level even if no individuals experience mortality deceleration (Vaupel and Yashin 1985). It may also cause mortality convergence or crossover between groups (e.g., black and white, lower and higher socioeconomic statuses) in old age even if survival advantage widens over the life course at the individual level (Dupre et al. 2006; Lynch and Brown 2003).

Most of the extant research focuses on the contribution of mortality selection to the life course health and mortality patterns within a single birth cohort. But due to the macro-level "technophysio evolution" (Fogel and Costa 1997), improving "cohort morbidity phenotype" (Finch and Crimmins 2004), and other social and epidemiological changes (Omran 1971), the variance of frailty distribution may change across birth cohorts (Zheng 2014). As a result of the

mortality selection mechanism operating in the context of changing frailty variance, the life course health and mortality patterns and life expectancy may change across cohorts. If frailty variance changes between groups (e.g., lower educated vs. higher educated) across birth cohorts, mortality selection mechanism may further complicate the cohort trend in health disparities and the life expectancy gap between groups. Broadly speaking, the impact of mortality selection on cohort trends belongs to a sizable body of literature in demography that considers how changes in population composition determine changes in observed, aggregate-level trends. This study builds on that literature and employs microsimulations to illustrate that mortality selection may generate seemingly contradictory trends in health disparities and life expectancy across birth cohorts at the group and individual level.

Substantively, this paper will contribute to the active academic and public discourse in the widening socioeconomic inequality in health and life expectancy in the U.S. by illustrating the impact of mortality selection on educational health disparities. A growing body of literature has reported that educational disparities have a profound impact on health, mortality, and life expectancy (Meara et al. 2008; Miech et al. 2011; Olshansky et al. 2012). Recent studies caution that the apparent increase in educational health disparities may be caused by lagged selection bias or compositional changes (Dowd and Hamoudi 2014; Hendi 2015, 2017). Researchers argue that educational expansion in the United States makes higher education more accessible and equitable, so individuals without higher education will increasingly come from vulnerable family backgrounds, those with lower socioeconomic status (SES), and this will be pronounced in recent cohorts rather than earlier cohorts. This changing selection-into-education process may have led to the enlarged health disparities between lower- and higher-educated groups across cohorts. This line of thought focuses on population composition in terms of average frailty

instead of the heterogeneity in frailty. Researchers have not yet considered how mortality selection based on frailty variance further complicates the analysis.

This study proposes a straightforward counterfactual simulation to estimate the contribution of mortality selection to the trends in the life expectancy gap between two educational groups (high school or less and any college) across recent birth cohorts in the United States. With empirical data from Panel Studies of Income Dynamics (PSID), National Health and Nutrition Examination Survey (NHANES), and National Health Interview Survey (NHIS), we found that mortality selection has contributed from 21% to 24% of the widening life expectancy gap between these two educational groups from the 1950s to the 1960s cohorts. The impact of group composition and unobserved heterogeneity in studying health disparities and life expectancy across cohorts should not be underestimated.

Background

The law governing the slope of mortality acceleration over the life course

The theory of population heterogeneity posits that death selectively removes the frailest members of a cohort so that mortality rate at the cohort level becomes increasingly dominated by robust members over the life course (Vaupel et al. 1979; Vaupel and Yashin 1985). This means that the individual hazard curve should be steeper than the cohort mortality curve, or individuals "age" faster than heterogeneous cohorts (Vaupel and Yashin 1983). Yashin and Iachine (1997) infer that the underlying individual hazard function from the semiparametric shared-frailty model using Danish twins' data supports the assumption that individuals age faster than cohorts.

The theory of population heterogeneity further suggests that the slope of mortality acceleration at the population level is negatively related to the variance of the distribution of frailty in the population (Yashin et al. 2002; Vaupel 2010). For example, the slope of the age-dependent mortality curve becomes steeper when the variance of frailty distribution declines because when a smaller proportion of frail individuals is selected out of the population at earlier ages, a relatively larger proportion of frail individuals survive to old age. But to our knowledge, no previous work has applied this idea to understand the consequence of changing frailty distribution on health disparities and life expectancy across birth cohorts.

Health disparities over the life course and the impact of mortality selection

Social and health scientists share a long-standing interest in the life course pattern of health disparities. Cumulative advantage mechanism, which is popular in the medical sociology and social epidemiology literature, posits that disparities in health, physical functioning, well-being, disease incidence, and mortality between more and less advantaged groups increase over the life course due to the cumulative health benefits of advantaged resources (Ross and Wu 1996; Lauderdale 2001; Dannefer 2003; Dupre 2007; House et al. 1994; Dannefer 2003). This argument may appear to be in conflict with the findings from most empirical studies that report convergence, instead of divergence, in health status over the life course. But this contradiction will be resolved if we consider the law of mortality selection. Mortality selection mechanism predicts that if a smaller proportion of frail individuals among more advantaged groups was selected out of the population at younger ages, this in turn would cause a larger proportion of frail individuals to survive into old age and could cause their overall mortality rate to converge with less advantaged groups (Lynch 2003; Eberstein et al. 2008; Zajacova et al. 2009).

Therefore, while cumulative advantage mechanism predicts a divergence in health status between more and less advantaged groups at the individual level, health convergence may still happen at the group level due to mortality selection mechanism. Thus, without purging the mortality selection effect, empirical findings on health convergence at the group level may be misleading with regard to patterns on the individual level. While the effect of mortality selection on the group-level, age-dependent mortality pattern within a single birth cohort is well recognized, no studies have investigated how mortality selection may influence the trends in life course health and mortality patterns and health disparities across birth cohorts.

Health disparities across birth cohorts and the impact of mortality selection

A wealth of studies has found an increase in socioeconomic disparities in health, disability, and life expectancy in the United States in recent decades (e.g., Feldman et al. 1989; Pappas et al. 1993; Preston and Elo 1995; Hummer et al. 1998; Meara et al. 2008; Jemal et al. 2008; Schoeni et al. 2005; Crimmins and Saito 2001; Goesling 2007; Liu and Hummer 2008; Montez et al. 2013; Miech et al. 2011; Sasson 2016). Some studies suggest that mortality rates may have increased in some of the poorer U.S. counties (e.g., Kindig et al. 2013) or among the least educated non-Hispanic whites (Olshansky et al. 2012; Case and Deaton 2015). There is also evidence for a widening gap in self-rated health and mortality by education levels across birth cohorts (Chen et al. 2010; Lynch 2003; Masters et al. 2012).

These findings are striking and attract substantial attention from academicians, health practitioners, and the media. At the same time, they raise significant methodological concerns regarding the extent of model misspecification and misinterpretation of patterns. Dowd and Hamoudi (2014) put forward the concept of lagged selection bias—the theory that changing

selection mechanisms into exposure groups may result in misleading conclusions. Lagged selection bias is prevalent in trend studies, but public health research has largely ignored this important issue. For example, studies of the differences in health outcomes for individuals with bachelor's degrees and those without have paid little attention to the empirical issue that the mechanism of selection into college has substantially changed in the United States. Due to the expansion of college education, individuals are gaining more equitable access to college. Individuals without a college degree may increasingly come from families in the lower and lowest socioeconomic status groups over time. This changing population composition could increase the health gap between individuals with and without a college degree even if the true causal effect of college on health is constant. Using simulations, Dowd and Hamoudi (2014) demonstrated that educational expansion may cause individuals who did not complete high school to be increasingly composed of individuals from vulnerable family backgrounds, and their mortality risk as a group may appear to increase over time even if they have not experienced a rising mortality risk at the individual level. Hendi (2015, 2017) suggests that this compositional change explains about 53% of the decline in life expectancy for the least-educated white women, and 87% and 26% of the widening educational life expectancy gaps between 1991 and 2005 for white men and women, respectively.

While the work by Dowd, Hamoudi, and Hendi emphasizes that exposure groups may be non-comparable due to cohort changes in the level of vulnerability with regard to family background, we argue that these changing selection mechanisms may further change the variance of unobserved frailty or vulnerability to mortality among these groups. Importantly, mortality selection mechanisms that hinge on this frailty variance may widen or narrow health disparities among these groups across birth cohorts, depending on the changes in frailty variance.

As Dowd and Hamoudi (2014) contended, individuals in more recent cohorts who were vulnerable to a shorter life span due to a disadvantaged family background had increasing access to higher education, while those exposed to similar conditions in earlier birth cohorts had no such opportunity. Therefore, the more highly educated group might become increasingly heterogeneous with regard to family background across birth cohorts. Dowd and Hamoudi further implied that the less educated group might have become increasingly homogeneous with regard to family background because individuals who did not have access to higher education were increasingly vulnerable individuals coming from disadvantaged family backgrounds. If the individuals coming from disadvantaged families were more vulnerable to mortality or had higher levels of frailty, we might then expect the variance of unobserved frailty distribution to increase among the highly educated group and decrease among the less educated group across birth cohorts.

But the trends in the level and variance of frailty across birth cohorts are not just affected by family background but also by macro "technophysio evolution" (Fogel and Costa 1997) and improving "cohort morbidity phenotype" (Finch and Crimmins 2004) or "cohort evolution" process (Zheng 2014). Increasing health capital across birth cohorts may generally reduce the variance of frailty (Zheng et al. 2016). Changing family-background-based selection mechanisms into different education groups (Dowd and Hamoudi 2014) may interact with the macro process of improving health capital and cause the variance of frailty distribution to decrease at a faster rate among the lower educated group than the higher educated group. But this statement needs to be examined with empirical evidence.

What are the implications of changing frailty variance across cohorts on the trends of health disparity and life expectancy? According to the law of mortality selection, if the variance

of frailty distribution among the lower educated group decreases by a larger extent compared to that among the higher educated, the slope of the lower education group's morality curve would increase more than that of the higher education group. This, in turn, would widen disparities in health and increase the gap in life expectancy between the lower and higher educated groups across birth cohorts. In this case, without purging the mortality selection effect, we would overestimate the growth in health disparities between groups with less and more education. Conversely, if the variance of frailty distribution among the lower educated group increases by a larger extent compared to that of the higher educated, their mortality curve might become even flatter compared to the higher educated group. In this case, without purging the mortality selection effect, we would underestimate the degree of enlarging health disparities. In our analysis, we first use microsimulations to demonstrate the effect of mortality selection on group-level, age-dependent mortality pattern, health disparities over the life course, and the life expectancy gap across birth cohorts. After these simulation experiments, we will use empirical data from PSID, NHANES, and NHIS to create a measurement of variance of frailty and analyze how changing frailty variance may have affected the trends in life expectancy gap between high school or less and any college from the 1950s to later cohorts. The percentage of college graduates in the United States has increased substantially since the 1915 birth cohort, stalled around the 1950s birth cohort, and resumed its upward trend with the 1960s birth cohort (Torche 2011). So, comparing the 1950s to later birth cohorts is an ideal case for testing the selection effect induced by the expansion of higher education.

Methods

Microsimulation method

Basic mathematical formulation

We use the microsimulation method to conduct both the simulation experiments and empirical evaluations. We start our simulation by setting up a model for individual hazard function. Following Vaupel et al. (1979), we let individuals in a cohort differ from each other in the value of frailty (denoted as z) characterizing their susceptibility to death, such that the force of mortality conditional on z is

$$\mu_i(x) = z_i \mu_0(x)$$

where $\mu_i(x)$ is the force of mortality for individual, i at instantaneous age x, z_i is frailty for individual i at the initial age, and $\mu_0(x)$ is the unobserved baseline hazard function with a frailty of 1. An individual with a frailty of 1 can be called a "standard" individual. An individual with a frailty of 1.5 is one and half times more likely to die at any particular age than the standard individual. An individual with a frailty of 0.5 is only half as likely to die. We specify the distribution of frailty z_i as a Gamma distribution at the initial age.

Following Vaupel and Yashin (1983), we further assume the individual baseline hazard function as

$$\mu_0(x)=ae^{bx}e^{\frac{a(e^{bx}-1)}{b}},$$

where the force of mortality for individual i at instantaneous age x is

$$\mu_i(x) = z_i a e^{(bx)} e^{\frac{a(e^{bx}-1)}{b}},$$

or

$$\ln\big(\mu_i(x)\big) = \ln(a) + bx + \frac{a(e^{bx}-1)}{b} + \ln(z_i).$$

It can be shown that the simulated cohort mortality curve will follow a Gompertz function $\bar{\mu}(x) = ae^{\beta x}$ or $\ln\big(\bar{\mu}(x)\big) = \ln(a) + \beta x$, which is consistent with empirical pattern. For a detailed explanation of this mathematical formation and alternative model specifications, please refer to Appendices 1-4.

Simulation procedure

In the simulation experiments, we estimate parameter combinations so that the generated aggregate age-dependent mortality patterns approximate those of the 1990 synthetic birth cohort from NHIS 1986-2009 surveys with linked mortality data through 2011 (Blewett et al. 2008).[1] We let *a* be the observed mortality rates at initial age 30 for two groups (high school or less, and any college) in the 1990 synthetic birth cohort. Thus, *a* equals the mean of mortality hazards across individuals within each group at the initial age. We conduct simulations for every year beginning at age 30 because the cohort mortality curve follows a Gompertz law starting from that age (Gompertz 1825). We use calibration methods to determine the optimal values for b and the variance of frailty parameter (z_i) (i.e., σ^2) that best fit the observed mortality curve of 1990 synthetic birth cohort. Specifically, we vary b and σ^2 to create a large set of possible combinations of their values and simulate the cohort mortality curve based on each combination.

[1] For details about NHIS linked mortality data, please refer to the following "Data for empirical evaluation" section. The 1990 synthetic birth cohort includes 253,367 individuals who experience 2,599 deaths in 1990.

We compare this simulation result with the empirical mortality pattern for the 1990 synthetic birth cohort from age 30 to 90 to narrow down to the combination of b and σ^2 that generates the closest fit in terms of mean squared error, that is, the average of the squares of the difference between the observed and simulated mortality rates.

Simulations proceed in every one year of age for a hypothetical population of 1 million individuals. At the individual level, we rely on the piecewise-constant force of mortality assumption to specify a constant (μ_i^*) force of mortality within each year of age. Under this assumption, the central mortality rate with each year of age for every individual *i,* m_x^i, equals μ_i^*. Then the probability of surviving between age *x* and *x* + 1 for every individual *i* is denoted as p_x^i, equals $e^{-m_x^i}$ (Preston et al. 2001). We stop the simulation at age 90 because mortality patterns past age 90 do not typically follow the Gompertz curve (Vaupel 1997). 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.

After generating the simulated group survival data, the next step is to calculate the group age-dependent mortality rate. We hold the same piecewise-constant force of mortality assumption that we use in the simulation procedure. Under this assumption, person-years within each one-year age interval equal $\frac{(l_{x+1}-l_x)}{\ln(\frac{l_{x+1}}{l_x})}$, where l_x is the number of individuals left alive at age *x* (Preston et al. 2001). The group age-dependent mortality rates are calculated as the number of deaths divided by this measure of person-years within each one-year interval. Life expectancy at age 30 is constructed under the same piecewise-constant force of mortality assumption.

After using calibration and simulation methods, we alter the values of the variance-of-frailty parameter (z_i) (i.e., σ^2) in specifying the individual-level mortality hazard to experiment with different frailty conditions. These simulations demonstrate the effect of mortality selection on group-level age-dependent mortality pattern, health disparities over the life course, and life expectancy gaps across birth cohorts, when operating in the context of changing frailty variance.

Data for empirical evaluation

In order to further illustrate the impact of mortality selection on health disparities across birth cohorts in the United States, we use empirical data from PSID, NHANES, and NHIS. We use PSID and NHANES to calculate frailty variance across birth cohorts for both the high school or less and any college groups, and we use NHIS to produce observed age-specific mortality rates across birth cohorts.

PSID survey began in 1968 with a nationally representative sample of families in the United States. The survey was administered annually until 1997, then biennially thereafter. We use the *Family Files 1968-2013.* Children from the original 1968 families have the PSID 'gene' and are also interviewed in the *Family Files* after they become the head of a household or spouse. Since 2007, PSID collected self-reported childhood diseases information before age 17. Our sample consists of all individuals born in 1950-1989 who provided information on early life disease measures before age 17. These early life disease measures consist of the sum of any of the 12 health problems a respondent reported he or she had before age 17, and scores for this index range from 0 to 12. These health problems are: asthma, diabetes, respiratory disease, allergies, heart trouble, epilepsy, severe headaches/migraines, stomach problems, high blood pressure, depression, drug/alcohol problem, and emotional/psychiatric problem. The original

sample size is 26,783. After dropping respondents with missing data for any of the 12 diseases (n = 15,389), the final sample size is 11,394.

In order to test the robustness of disease index from PSID data, we further create a similar index from NHANES data. The NHANES collected information about health and diet from a nationally representative sample of the noninstitutionalized civilian U.S. population. We utilize data from 1999 to 2012. We select health problems consistently measured throughout the waves that first occurred before the individual reached age 17. These health problems are asthma, arthritis, heart failure, coronary heart disease, angina, heart attack, stroke, emphysema, thyroid, chronic bronchitis, liver condition, and diabetes. We construct a health index based on these 12 diseases. The original sample size for those born from 1950 to 1989 is 31,492. After dropping respondents with missing data for education (n = 31) or any of the 12 diseases (n = 9,280), the final sample size is 22,181. For both PSID and NHANES data, the variance of the summary health index is used as a proxy for the unobserved frailty variance. This variance measure is calculated for the high school or less and any college and for the 1950, 1960, 1970, and 1980 cohorts.

We use IPUMS NHIS 1986-2009 surveys linked to mortality data through the year 2011 to generate age-dependent mortality patterns across birth cohorts (https://ihis.ipums.org/ihis/) (Blewett et al., 2018). The NHIS is a multistage probability sample survey of the non-institutionalized civilian U.S. population conducted by the National Center for Health Statistics. NHIS collects health information for each member of a family or household sampled, as reported by one primary respondent. Respondents are linked to death records in the National Death Index (NDI) through probabilistic record-matching methods based on 12 criteria to ascertain the vital

status of each respondent. To date, death records from the NHIS 1986-2009 surveys are available to the public. At the time of our study, mortality information at quarter-year intervals was available through December 31, 2011. Because the 1970s and later cohorts are not old enough to produce reliable mortality patterns, we focus on the birth cohorts of the 1950s and 1960s. The original sample size for these two 10-year birth cohorts with eligible mortality status is 579,183. After dropping respondents with missing data for education (n = 3,478), the final sample size is 575,705 experiencing 23,665 deaths. We reshape the data to person-year format left truncated at age at survey and right-censored at the age of death or age at December 31, 2011. This sample contributes 8,509,452 person-years of exposure.

Counterfactual simulation procedure for empirical evaluation

Cross-cohort changes in health disparities can be affected by a number of mechanisms besides the mortality selection effect. These include changes in the true causal effect at the individual level, changes in individual-level hazard pattern, and external period effects. We propose a straightforward counterfactual simulation procedure to remove these confounding factors and to evaluate the extent to which the widening educational mortality disparities from the 1950s to 1960s birth cohorts are due to mortality selection. We proceed in the following steps: (1) construct a person-year file from NHIS data and compute age-specific mortality rates by education and cohort for every five-year age category. The large sample size contained in the NHIS facilitates stable estimates within five-year age categories. We then use log-Gompertz to smooth and extrapolate log mortality rates to age 90. We then construct life tables, generate life expectancies, and calculate the degree of widening gap in life expectancies between two educational groups across birth cohorts; (2) calibrate the frailty variance for the two education groups in the 1950s cohort so that the predicted age-dependent mortality rates can replicate those

observed in the NHIS data;[2] (3) obtain frailty variances for the later birth cohorts based on their relative percentages as calculated from PSID or NHANES data; (4) create mortality patterns for the later birth cohorts using observed mortality rates at age 30 and their corresponding frailty variances obtained in step 3, construct life tables, generate life expectancies for two education groups across birth cohorts, and calculate the growth in educational gap across birth cohorts; and (5) create mortality patterns for the later birth cohorts using observed mortality rates at age 30 and fixing the frailty variance at the 1950s level, construct life tables, generate life expectancies for two education groups across birth cohorts, and calculate the degree of widening gap across birth cohorts. This gives the counterfactual mortality patterns for the later cohorts assuming mortality selection is absent but individual-level hazard pattern and all other confounding factors are fixed in this simulated world; (6) calculate the net contribution of mortality selection on the widening gap in life expectancy between lower and higher educated groups across birth cohorts by taking the difference in the degree of widening gap in life expectancy obtained in steps 4 and 5 as a percentage of the widening gap obtained in step 4; (7) predict the size of mortality selection's effect in observed mortality data by multiplying the percentage obtained in step 6 with the observed widening gap obtained in step 1.

[2] We use calibration methods to determine the optimal values for b and the variance of frailty parameter (z_i) (i.e., σ^2) that best fit the observed mortality curve. Specifically, we vary b and σ^2 to create a number of combinations of their values and simulate the cohort mortality curve based on each combination. We compare this simulation result with the empirical mortality pattern for the 1950s cohort from ages 30 to age 90 and find the combination of b and σ^2 that generate the closest fit in terms of mean squared error, that is, the average of the squares of the difference between the observed and simulated mortality rates.

Results

Simulation Experiments

Mortality patterns over the life course

We first conduct a thought experiment from simulated data where the generated age-specific mortality-rate pattern approximates that of the 1990 synthetic birth cohort from NHIS data. Appendix 5 shows that when $b = 0.075$, $\sigma^2 = 0.77$ and 0.34 for the lower educated and higher educated, respectively, the simulated mortality pattern is very close to the observed mortality pattern. Therefore, we choose to use this combination of parameters to generate the simulated data. Figure 1 shows the mortality differentials between lower and higher educated groups over the life course at the group level (left panel) and for a "standard" individual with frailty of 1 (right panel) from the simulated data. Within each education group, individual mortality hazard increases at a faster rate over the life course than does group mortality rate due to the mortality selection mechanism that selectively removes the frailest members of a group. These patterns are consistent with Yashin and Iachine (1997), which infers the underlying individual hazard function from semiparametric shared-frailty model using Danish twins' data. Further, we find that mortality differential diverges over the life course at the individual level due to the cumulative advantage mechanism, and it converges at group level due to mortality selection. We apply standard Cox model to these simulated data and find that without purging mortality selection mechanism, survival benefits associated with higher education diminish over the life course, as indicated by the 1.03 hazard ratios for the interaction between five-year age groups (i.e., 30-34, 35-39, …, 85-90) and the higher education group (Table 1).

[Figure 1, Table 1 here]

Mortality patterns across birth cohorts with increasing frailty variance among higher-educated groups

We then examine how mortality selection operates in the context of changing frailty variance while the individual-level hazard pattern is fixed (i.e., the true effect of education on mortality is fixed at the individual level). Figure 2 shows the mortality differentials between lower-educated and higher-educated groups over the life course across four birth cohorts assuming a fixed-mortality pattern at the individual level. We further let frailty variance be fixed at 0.77 across four hypothetical birth cohorts for lower-educated group, while it increases from 0.34 to 1.50 for the higher-educated group. The mortality curve of the higher-educated group becomes flatter at the group level, while the individual hazard curve remains unchanged. This is because when frailty variance increases, frail individuals are selected out of the population at a faster rate (Figure 3). This causes the group-level mortality curve to be quickly dominated by relatively robust members and consequently become flatter. As a result, mortality differentials between the lower- and higher-educated groups widen at the group level across four cohorts but remain fixed at the individual level. In other words, even though the true effect of education on mortality remains unchanged at the individual level, the educational mortality gap at the group level can still increase across cohorts as a result of the changing frailty variance. We further apply the standard Cox model to these simulated data and find that without purging mortality selection mechanism, the yielded changing associations between education and mortality risk over the life course reflect the group-level rather than individual-level pattern (Table 1).

[Figures 2 and 3 here]

Due to the flatter mortality curve at the group level, the corresponding life expectancy at age 30 increases from 50.35 years to 52.50 years across these four hypothetical cohorts with increasing frailty variance among higher-educated groups. This, in turn, leads to a widening gap in life expectancy between lower- and higher-educated groups across cohorts as shown in Figure 4, even though the extent of survival advantage associated with higher education remains unchanged at the individual level.

[Figures 4 here]

Mortality patterns across birth cohorts with decreasing frailty variance among lower-educated groups

Next, we conduct a simulation experiment in which the frailty variance decreases among the lower-educated group (Appendix 6). We let the individual-level hazard pattern be fixed or the true effect of education on mortality be fixed at the individual level. We further fix the frailty variance at 0.34 across three hypothetical birth cohorts for the higher-educated group and let it decrease from 0.77 to 0.25 for the lower-educated groups. When variance of frailty distribution decreases across birth cohorts for lower-educated groups, the mortality curve becomes steeper at the group level while individual hazard remains unchanged. But the demographic mechanism contributing to these changes is the same as the one causing the flatter slope among the higher-educated group when frailty variance decreases; the slope of the mortality curve at group level is negatively associated with the variance of frailty distribution. When frailty variance decreases, it becomes harder to select frail individuals out of the population. So, a relatively larger proportion of frail individuals survive to older age, and the mortality curve at group level steepens (Appendix 7). As a result, mortality differentials between lower- and higher-educated groups

widen across these three hypothetical cohorts while the gap in individual mortality hazard does not change.

Appendix 8 shows that the corresponding life expectancy at age 30 decreases from 46.95 years to 45.19 years across these three hypothetical cohorts with decreasing frailty variance among lower-educated groups. This, in turn, leads to a growing gap in life expectancy between lower- and higher-educated groups across cohorts, even though the extent of survival disadvantage associated with lower education remains unchanged at the individual level.

Empirical evaluation

How does mortality selection work in the real world? A major challenge to the empirical investigation of cohort changes and mortality selection is that the true distribution of frailty is unobserved in the population. However, since our main focus is the change between cohorts and education differences in the relative magnitude of frailty variation (rather than the variances of the absolute scores of unobserved frailty), it is possible to construct a proxy indicator of relative frailty variances through longitudinal survey data. We rely on data from the PSID, a nationally representative sample, to construct this frailty indicator for individuals and sub-populations before they reach age 17, after which some of them will be in college. We calculate a health problem summary index by adding up the number of health problems from a 12-item list before the person reaches age 17. The variance of this index is then used as a proxy for the unobserved frailty variance.[3]

[3] To examine the distributional properties of our constructed frailty measure, we fit a Gamma distribution to the empirical distribution of our PSID-based frailty measure and compare them in Appendix 9. The theoretical Gamma density is fitted using maximum likelihood estimation. The figure suggests that the empirical distribution of our constructed frailty measure can be reasonably approximated by the Gamma distribution.

We calculate the variance measure for high school or less and any college and for the 1950, 1960, 1970, and 1980 cohorts respectively. We also adjust the individual-level index for the differences in the mean of this index across cohort-education subgroups, so that the cohort changes in frailty variance is purged of any changes in their means. We then express the calculated frailty variance, for the college and non-college groups as a percentage of the frailty variance in their respective 1950 cohorts. This gives us the relative sizes of frailty variance. For example, if the frailty variance in cohort 1950 and cohort 1960 are σ^2_{1950} and σ^2_{1960} respectively, then the relative percentage for cohort 1960 is calculated as: $\frac{\sigma^2_{1960}}{\sigma^2_{1950}}$.

Figure 5 reports the calculated absolute frailty variance and relative frailty variance using this method. The left panel shows the absolute frailty variance. Different from the simulations for the 1990 synthetic birth cohort, the any college has a larger frailty variance than non-college across all four birth cohorts, which is partially due to lower means among this group. This implies that the age-dependent mortality pattern between these two groups will be similar to that of the higher-educated cohort 4 vs. lower-educated in Figure 2. That is, the any college group will have a flatter slope than non-college group. The right panel shows relative frailty variance declines across cohorts for both education groups, but the decline is more dramatic in relative terms for the lower-educated group. This pattern is consistent with our prediction that the changing family background composition in different education groups may interact with the macro process of improving health capital and cause the variance of frailty distribution to decrease at a faster rate among the lower-educated group than the higher-educated group. This implies that mortality curves will become steeper for both education groups across four birth

cohorts but the change is greater for those without a college education, which may then widen the mortality gap between these two education groups across birth cohorts. For sensitivity analysis, we also created a similar health index from NHANES 1999-2012. Appendix 10 shows the cross-cohort pattern is similar to the one in Figure 5.

[Figure 5 here]

We use the counterfactual simulation procedure to estimate the contribution of mortality selection to the widening educational health disparities from the 1950s to 1960s cohorts.

Step (1): We generate logged age-dependent mortality rates for the two education groups in the 1950s and 1960s birth cohorts from NHIS data. Following the Gompertz function of age-dependent mortality pattern, we use a linear function of log mortality rate to extrapolate mortality rates up to age 90 as shown in Figure 6. As predicted from the frailty variance in Figure 5, any college has a flatter slope than high school or less for both birth cohorts. However, the 1960s birth cohorts have flatter slopes than those from the 1950s despite a smaller variance of frailty, which is probably due to period-related medical advancement in older ages. Based on these age-specific mortality rates, we construct life tables and generate life expectancies at age 30 as shown in the top panel of Table 2. Life expectancies for high school or less and any college increase by 1.50 and 2.56 years across the two birth cohorts, respectively. Therefore, the gap between the two education groups increases from 5.70 to 6.76 years, which indicates a difference of 1.06 years between the two birth cohorts.

[Figure 6, Table 2 here]

Step (2): We calibrate the frailty variance for the two education groups in the 1950s cohort so that the predicted age-dependent mortality rates can replicate those observed in the NHIS. By following the calibration method, we are able to generate simulated 1950s mortality patterns very similar to those created from the NHIS data as shown in Figure 7 when $b = 0.075$, $\sigma^2 = 0.63$ and 0.87 for high school or less and any college, respectively. Therefore, we decide to use these numbers as the variance of frailty for these two education groups in the 1950s cohort.

[Figure 7 here]

Step (3): We obtain frailty variances for the later birth cohorts based on their relative percentages as calculated from PSID data. We use the calibrated variances from Step 2 as the 1950 cohorts' baseline in deriving the 1960 cohorts' frailty variance, which is 0.54 for high school or less and 0.83 for any college. Note that the larger frailty variance in the any college group is inconsequential for our analysis because only the relative percentage to the 1950 cohorts within each education group is used for the simulation.

Step (4): We create mortality patterns for the two education groups in the 1960s birth cohorts using observed mortality rates at age 30 and frailty variances obtained in Step 3. We then construct life tables, generate life expectancies for the two education groups across birth cohorts and calculate the degree of widening gap across birth cohorts as shown in the middle panel of Table 2. Life expectancies for the high school or less and any college groups increase by 0.03 and 0.99 years across the two birth cohorts, respectively. Therefore, the gap between the two education groups increases from 5.73 to 6.69 years, which indicates a 0.96-year increase in the educational life expectancy gap across the two birth cohorts.

Step (5): We create mortality patterns for the two education groups in the 1960 birth cohorts using observed mortality rates at age 30 and keeping the frailty variance fixed at the 1950s' level. We then construct life tables, generate life expectancies by education and birth cohort and calculate the increase in the education gap across birth cohorts as shown in the bottom panel of Table 2. Life expectancies for the high school or less and any college groups increase by 0.33 and 1.05 years across the two birth cohorts, respectively. Therefore, the gap between the two education groups increases from 5.73 to 6.46 years, which indicates a 0.72-year increase in the educational life expectancy gap across the two birth cohorts. This is the counterfactual change in the life expectancy gap assuming that mortality selection is absent and all other confounding factors are fixed.

Step (6): We calculate the relative contribution of mortality selection to the increase in the gap in life expectancy between lower- and higher-educated groups across birth cohorts by taking the difference in the increase in the educational life expectancy gap obtained from Step 4 and Step 5 as a percentage of widening gap obtained from Step 4. That is, (0.96-0.72)/0.96=24%. This means mortality selection contributes to 24% of the increase in the educational life expectancy gap across these two birth cohorts.

Step (7): We predict the size of mortality selection's effect in observed mortality data by multiplying the percentage obtained in Step 6 with the observed widening gap obtained from Step 1. That is, 1.06*24%=0.26 years. This means mortality selection adds 0.26 years to the observed widening gap in life expectancies between lower- and higher-educated groups from the 1950s to the 1960s birth cohorts. After adjusting for the effect of mortality selection, the cross-cohort increase in the educational life expectancy gap still exists but is now 0.8 year.

We replicate this counterfactual analysis using relative frailty variance information obtained from NHANES data and find that mortality selection contributes to 21% of the increase in the educational life expectancy gap from the 1950s to the 1960s cohorts, which translates to 0.22 years (Appendix 11). In other words, the gap would have increased by 0.84 years (1.06-0.22=0.84) if mortality selection were not in effect; this estimate is very similar to the PSID results.

Discussions and Conclusions

In demographic research, cohort changes in age-specific mortality rates and life expectancy are important indicators of the cohort changes in health conditions and between-group disparities. However, the presence of unobserved frailty and mortality selection mechanisms may lead to cohort changes in these population-level indicators even when there are no actual cohort changes in the individual mortality patterns. Therefore, empirical estimates of cohort changes in health disparities and life expectancy may be biased if we do not adjust for the mortality selection mechanism. We examined the impact of mortality selection in simulated as well as real data and reached the following conclusions:

1. Mortality selection can lead to convergence, parallels, and divergence in mortality rates over ages at the group level even though the cumulative advantage mechanism works at the individual level. Therefore, the age-dependent mortality differential pattern at group level does not necessarily reflect the individual life-course pattern (Figures 1, 2, Appendix 6).

2. Trends in life expectancy across birth cohorts can be altered by the variance of frailty distribution in the absence of change in the individual mortality curve. Increasing variance of frailty across cohorts can lead to flatter age-dependent mortality curves at the group level and an increase in life expectancy even if the level and shape of the individual mortality curve remain unchanged (Figures 2 and 4). Decreasing variance of frailty across cohorts can lead to steeper age-dependent mortality curves at the group level and a decrease in life expectancy even if the level and shape of the individual mortality curve remain unchanged (Appendices 6 and 8). Without considering the changes in the variance of frailty distribution, we may either overestimate or underestimate life expectancy.
3. Trends in disparities in health and life expectancy between more- and less-educated groups across birth cohorts may be distorted by the changes in the variance of frailty distribution in the absence of change in the health (mortality) difference at the individual level. If variance of frailty distribution increases among the more-educated group (Figure 2) or decreases among the less-educated group (Appendix 6), disparities in health and life expectancy widen across birth cohorts (Figure 4 and Appendix 8). If variance of frailty distribution decreases among the more-educated group or increases among the less-educated group, disparities in health and life expectancy narrow across birth cohorts.

These simulation experiments demonstrate the complexity of the ways in which changing unobserved frailty distributions may contribute to the observed patterns in health disparities between lower- and higher-educated groups across cohorts. We then demonstrate the relevance of the mortality selection mechanism to empirical research using the counterfactual simulation method. Using the number of diseases before age 17 from PSID and NHANES data as proxies

for frailty, we find that those with any college have a larger frailty variance than non-college across all four birth cohorts (1950s, 1960s, 1970s, and 1980s). This is consistent with extracted age-dependent mortality patterns from NHIS data. We further find that frailty variance declines across cohorts for both education groups, but the decline is larger in proportion for those without a college education. The declining frailty variance across birth cohorts is consistent with implications from the "technophysio evolution" theory (Fogel and Costa 1997) and "cohort morbidity phenotype" theory (Finch and Crimmins 2004). The larger decline among the lower-educated group is consistent with predictions that increasing homogeneity of family backgrounds among this group is induced by increasing equitable access to higher education (Dowd and Hamoudi 2014).

The differential declines in frailty variance imply that the cohort mortality curve will become steeper for both education groups across four birth cohorts, but the change is greater for the non-college group due to the mortality selection mechanism. This then can wide mortality differentials between the two educational groups across birth cohorts. The estimated mortality patterns from NHIS data suggest that the mortality slope becomes flatter for both education groups in the 1960s cohort and that the change is greater for the any college group, which leads to a widening of the mortality gap between the two education groups across the two birth cohorts. These empirical patterns do not necessarily dispute the function of mortality selection for the 1960s cohort but imply period-based medical advancement may further pull down the mortality curves among the later birth cohorts.

In order to uncover the net contribution of mortality selection to these widening mortality differentials, we create two scenarios—one with mortality selection in effect, the other a counterfactual scenario without mortality selection. By comparing the different amounts of

growth in the life expectancy gap in these two scenarios, we find that mortality selection contributes to 21-24% of the widening life expectancy gap between the high school or less and any college groups from the 1950s to 1960s birth cohorts. Since the observed gap grows by 1.06 years, mortality selection adds 0.22-0.26 years to the widening gap. In other words, if mortality selection were not in effect, the life expectancy gap between these education groups would have grown by 0.80-0.84 years from the 1950s to 1960s birth cohorts. We further looked at a different categorization of education (college degree or more vs. without college degree) and the results suggest that mortality selection contributes to about 20% to widening gap in life expectancy between these two education groups from the 1950s to 1960s birth cohorts. The number is comparable to the contribution of mortality selection to the widening life expectancy gap between high school or less and any college groups. Mortality selection accounts for a sizeable amount of contribution but does not eliminate the overall widening life expectancy gap by education across these two birth cohorts.

A major challenge to the empirical evaluation of the mortality selection effect is that true frailty distribution is not observed. We use the summary disease index before age 17 from PSID and NHANES as a proxy for frailty. We are not particularly concerned about the higher frailty variance among the higher-educated group for three reasons. First, the frailty variance here is the coefficient of relative variation, which is the variance divided by mean, to account for changing means across birth cohorts. The reason why frailty variance is higher among the higher-educated group is partially because their means are lower. Second, the information we used in the counterfactual simulation is not the raw frailty variance but the cohort changes in relative magnitude of frailty variation. That is the percentage of the frailty variance in later birth cohorts to their respective 1950s cohort within each educational group. This percentage is not affected by

the between-educational group difference in frailty variance. Third, the empirical age-dependent mortality patterns by education for the 1950s birth cohort are consistent with the larger frailty variance among the higher-educated group, which has a flatter slope of mortality acceleration compared to the lower-educated group. Better frailty measures should be collected in the future in order to more robustly and accurately estimate the contribution of mortality selection to the trends in health disparities.

We caution that population health scientists need to carefully consider the impact of composition changes on the trends in health disparities and life expectancy across cohorts. Other work has pointed out that it is not easy to completely and accurately solve the mortality selection bias even if we know the underlying individual-level hazard function and group-level frailty distribution function (Heckman and Singer 1982; Keiding et al. 1997; and Hougaard 1994). Recent methodological developments suggest using an appropriately weighted survival curve (Cole and Hernan 2004; Hernan 2010) or accelerated-failure-time model (Bradburn et al. 2003) to mitigate mortality selection bias. These strategies for addressing changing mortality selection across birth cohorts is worth further examination. The counterfactual simulation procedure we propose in this article can be utilized as an alternative to these methods to mitigate or estimate the mortality selection effect.

Unobserved individual frailty is prevalent and consequential in the population patterns of health and mortality. Attention to unobserved frailty is important when we compare across heterogeneous populations. For example, survival advantage among diverse older-age Americans compared to the smaller and more homogeneous elderly population in European countries despite disadvantage in younger age groups may be partially driven by the mortality selection effect (Ho and Preston 2010). Hence, without ruling out the mortality selection effect, we may

underestimate the gap in life expectancy between residents of the United States and other countries. Due to a reduction in prenatal and postnatal exposure to infection and inflammation and an improvement in living standards and nutrition, later birth cohorts tend to have better health endowment and smaller variance of frailty distribution compared to earlier birth cohorts. This can lead to a steeper slope of mortality curve at the cohort level (Zheng 2014). Without purging the mortality selection effect, the progress of life expectancy across birth cohorts may be understated.

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Table 1. Results from standard Cox model applied to simulated data

	Figure 1		Figure 2	
	HR	95% CI	HR	95% CI
Higher educated, cohort 1	0.55	[0.53, 0.57]	0.55	[0.53, 0.57]
(Higher educated, cohort 1) * age groups	1.03	[1.03, 1.04]	1.03	[1.03, 1.04]
Higher educated, cohort 2			0.59	[0.57, 0.62]
(Higher educated, cohort 2) * age groups			1.02	[1.01, 1.02]
Higher educated, cohort 3			0.71	[0.68, 0.73]
(Higher educated, cohort 3) * age groups			0.98	[0.98, 0.99]
Higher educated, cohort 4			0.82	[0.78, 0.85]
(Higher educated, cohort 4) * age groups			0.95	[0.95, 0.96]

Notes:

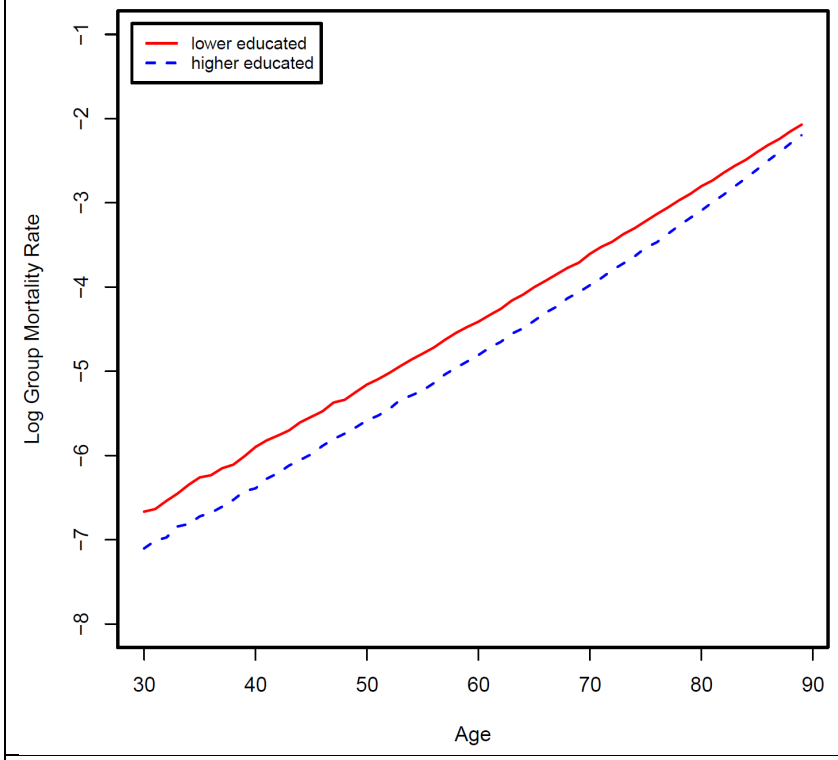
1. HR: Hazard Ratio; CI: Confidence Interval
2. 100,000 cases are randomly drawn from 1 million cases in each simulated data. They are split into long format by 12 five-year age groups: 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, and 85-90. These age groups are coded from 1 to 12.
3. Time metric in continuous Cox model is attained age (age of death or being censored). Therefore, the main effects of age groups are not controlled in the model. Inclusion does not alter the results.
4. Advantageous over other parametric models, Cox model does not need to make any parametric assumption of the underlying hazard function. Piecewise exponential model (piecewise constant hazard) and discrete time non-parametric baseline model (estimated with pgmhaz command) are also used to test the robustness of results. These two models provide almost identical results to those from Cox model. But these models do not adjust for gamma distribution of frailty.

Table 2. Life expectancies at age 30 in high school or less and any college groups in the 1950s and 1960s birth cohorts from empirical and simulated data using PSID frailty measure

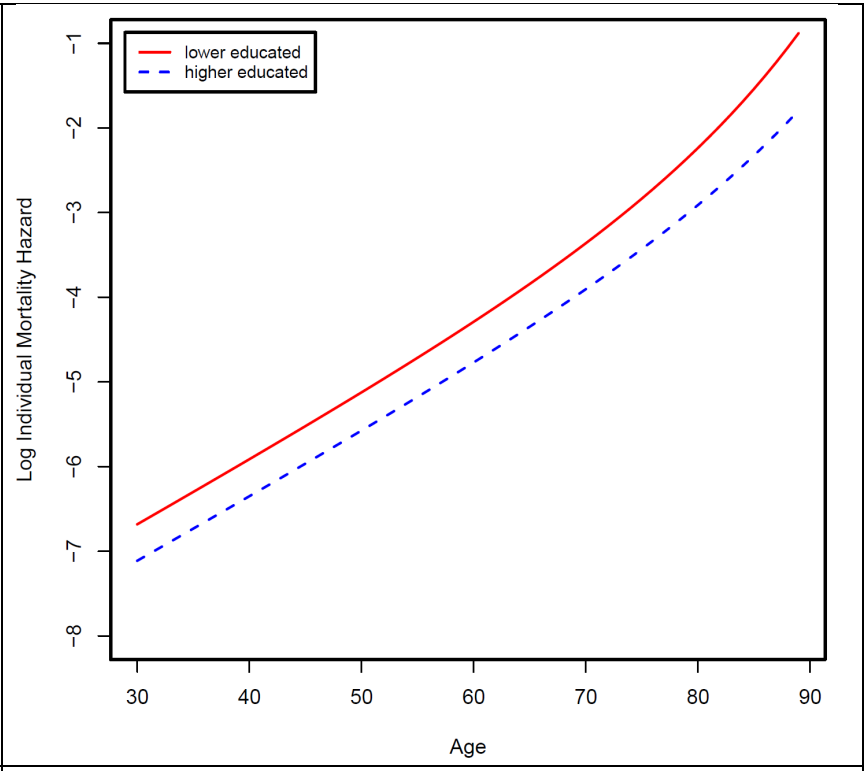
	High school or less	Any college	
	Empirical		*C-H*
Cohort 1950s	47.02	52.72	5.70
Cohort 1960s	48.52	55.28	6.76
1960s-1950s	1.50	2.56	**1.06**
	Simulated (mortality selection present)		*C-H*
Cohort 1950s	46.65	52.38	5.73
Cohort 1960s	46.68	53.37	6.69
1960s-1950s	0.03	0.99	**0.96**
	Simulated (mortality selection absent)		*C-H*
Cohort 1950s	46.65	52.38	5.73
Cohort 1960s	46.98	53.43	6.46
1960s-1950s	0.33	1.05	**0.72**

Notes: Empirical data are from NHIS 1986-2009 surveys linked to mortality data through 2011. The sample size for the 1950s and 1960s birth cohorts is 575,705 experiencing 23,665 deaths and 8,509,452 person-years of exposure. Following the Gompertz function of age-dependent mortality pattern, we use a linear function of log mortality rate to extrapolate mortality rates up to age 90. Based on these observed and extrapolated mortality rates, we construct life tables and calculate the life expectancies at age 30.

Figure 1. Mortality differentials between lower-educated and higher-educated groups over the life course at group and individual levels

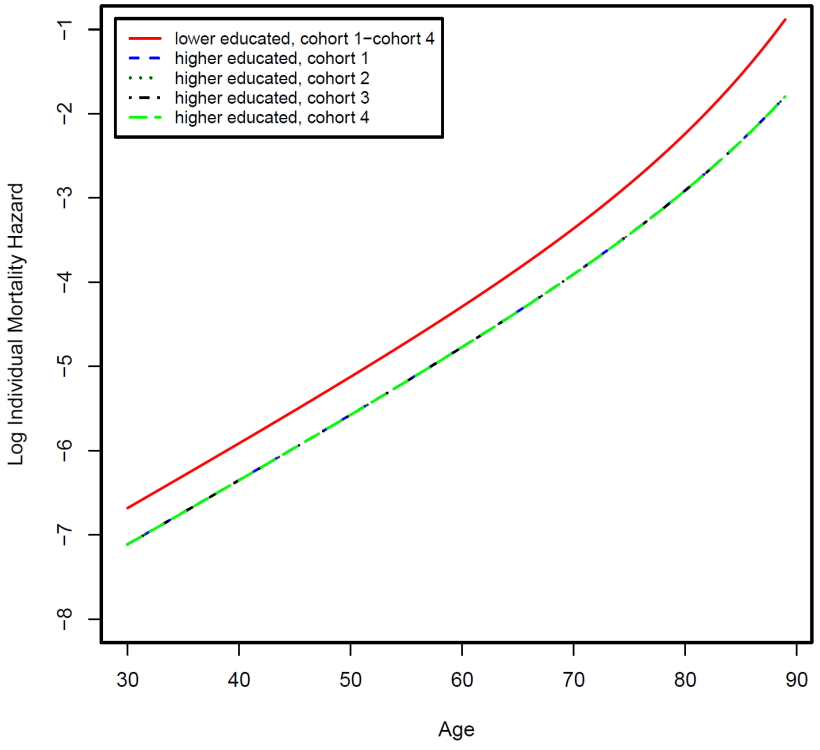


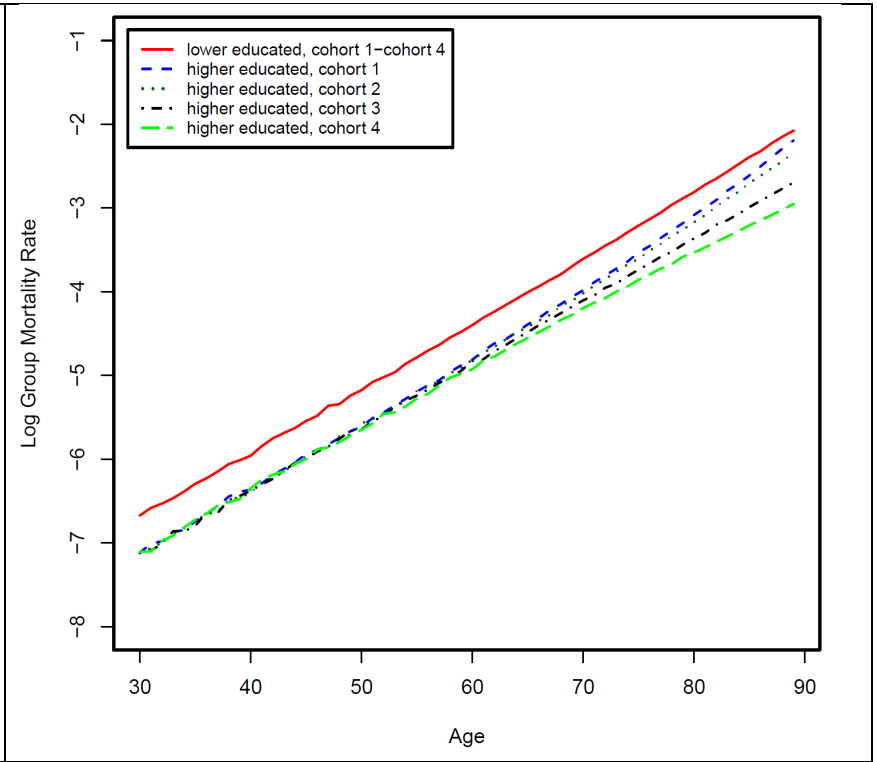
Group	$\ln(a)$	σ^2	b
Lower educated	-6.76	0.77	0.075
Higher educated	-7.19	0.34	0.075



Individual	$\ln(a)$	z_i	b
Lower educated	-6.76	1.00	0.075
Higher educated	-7.19	1.00	0.075

Figure 2. Mortality differentials between lower-educated and higher-educated groups over the life course across multiple birth cohorts assuming increasing frailty variance among the higher-educated group.





Individual	$\ln(a)$	z_i	b
Lower educated, cohort1-cohort4	-6.76	1.00	0.075
Higher educated, cohort1	-7.19	1.00	0.075
Higher educated, cohort2	-7.19	1.00	0.075
Higher educated, cohort3	-7.19	1.00	0.075
Higher educated, cohort4	-7.19	1.00	0.075

Group	$\ln(a)$	σ^2	b
Lower educated, cohort1-cohort4	-6.76	0.77	0.075
Higher educated, cohort1	-7.19	0.34	0.075
Higher educated, cohort2	-7.19	0.50	0.075
Higher educated, cohort3	-7.19	1.00	0.075
Higher educated, cohort4	-7.19	1.50	0.075

Figure 3. Variance of frailty over the life course among four hypothetical cohorts of higher-educated groups

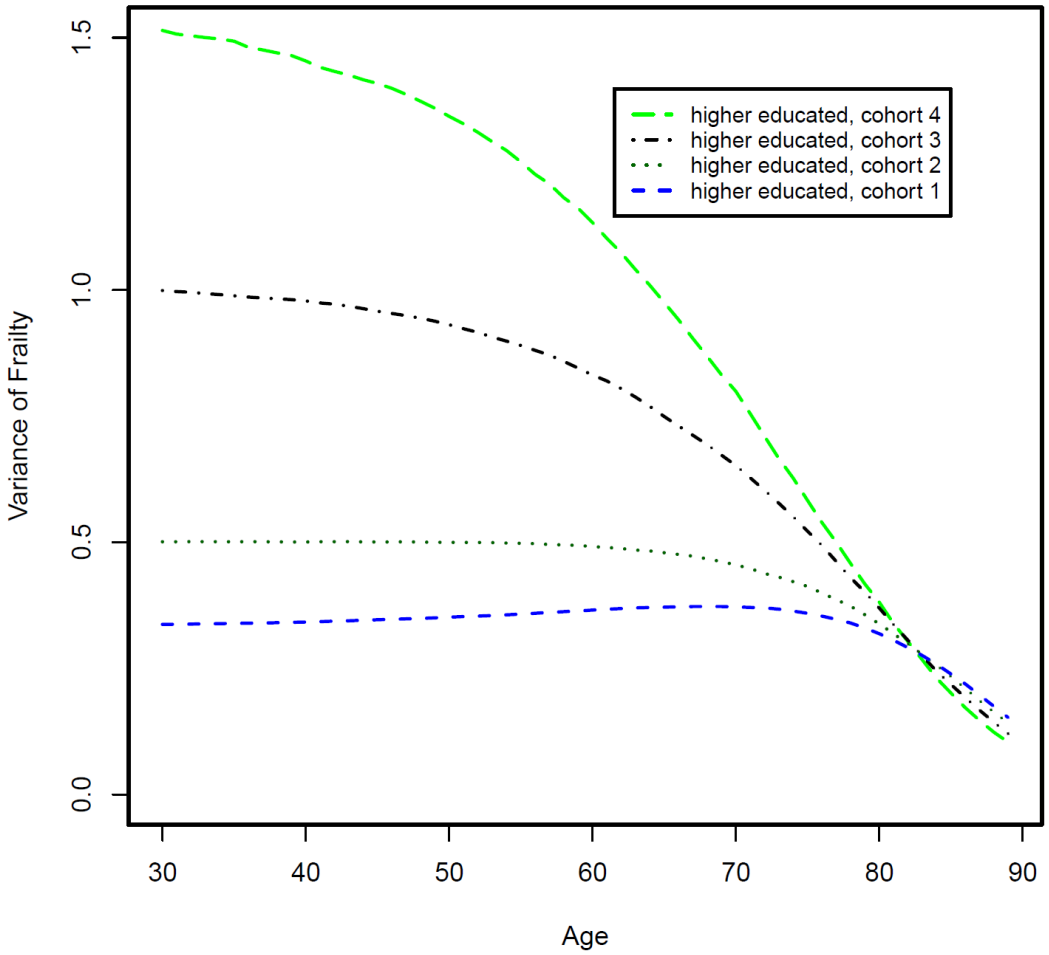


Figure 4. Gaps in life expectancy at age 30 between lower-educated and higher-educated groups across four hypothetical cohorts assuming increasing frailty variance among the higher-educated group

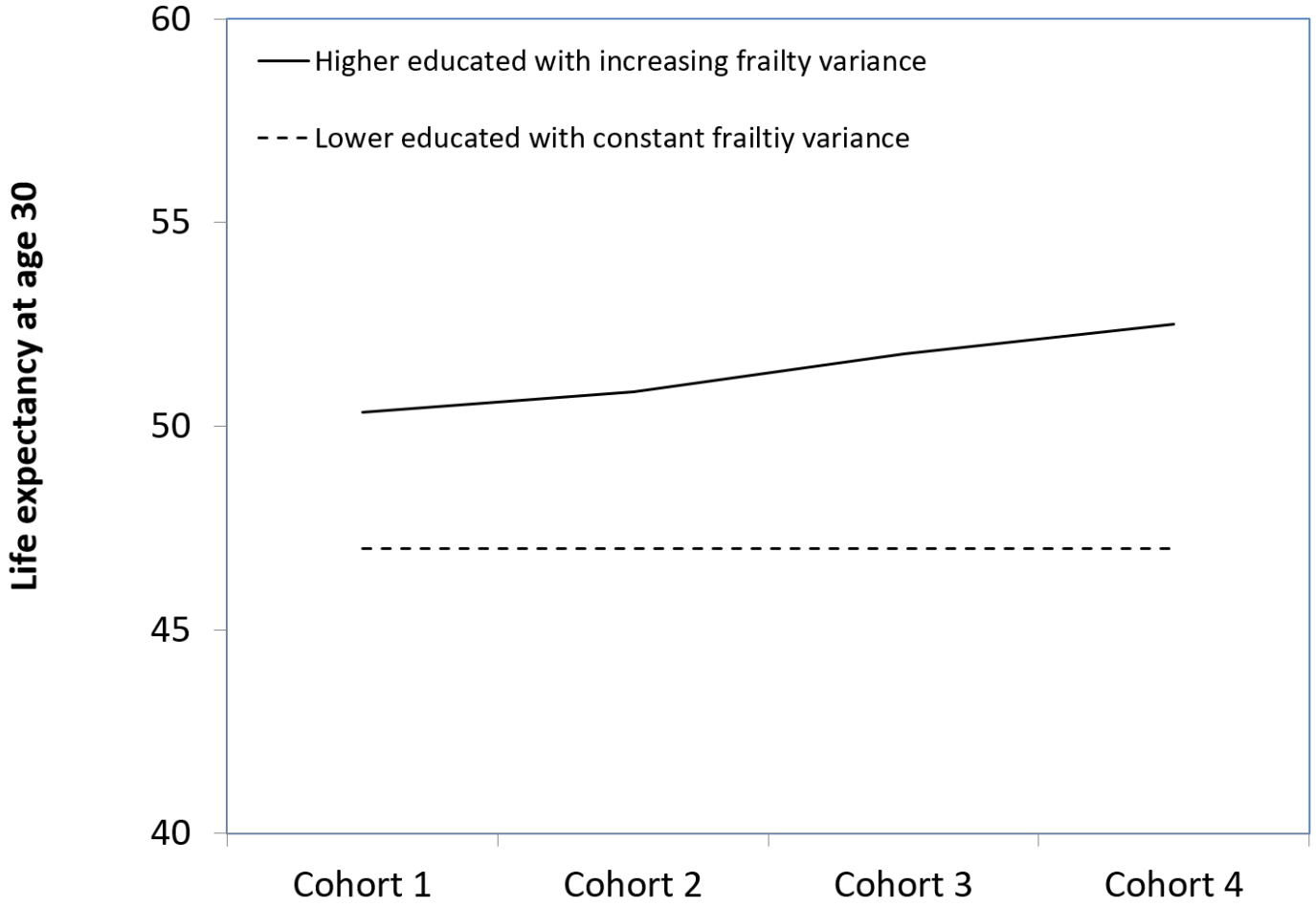
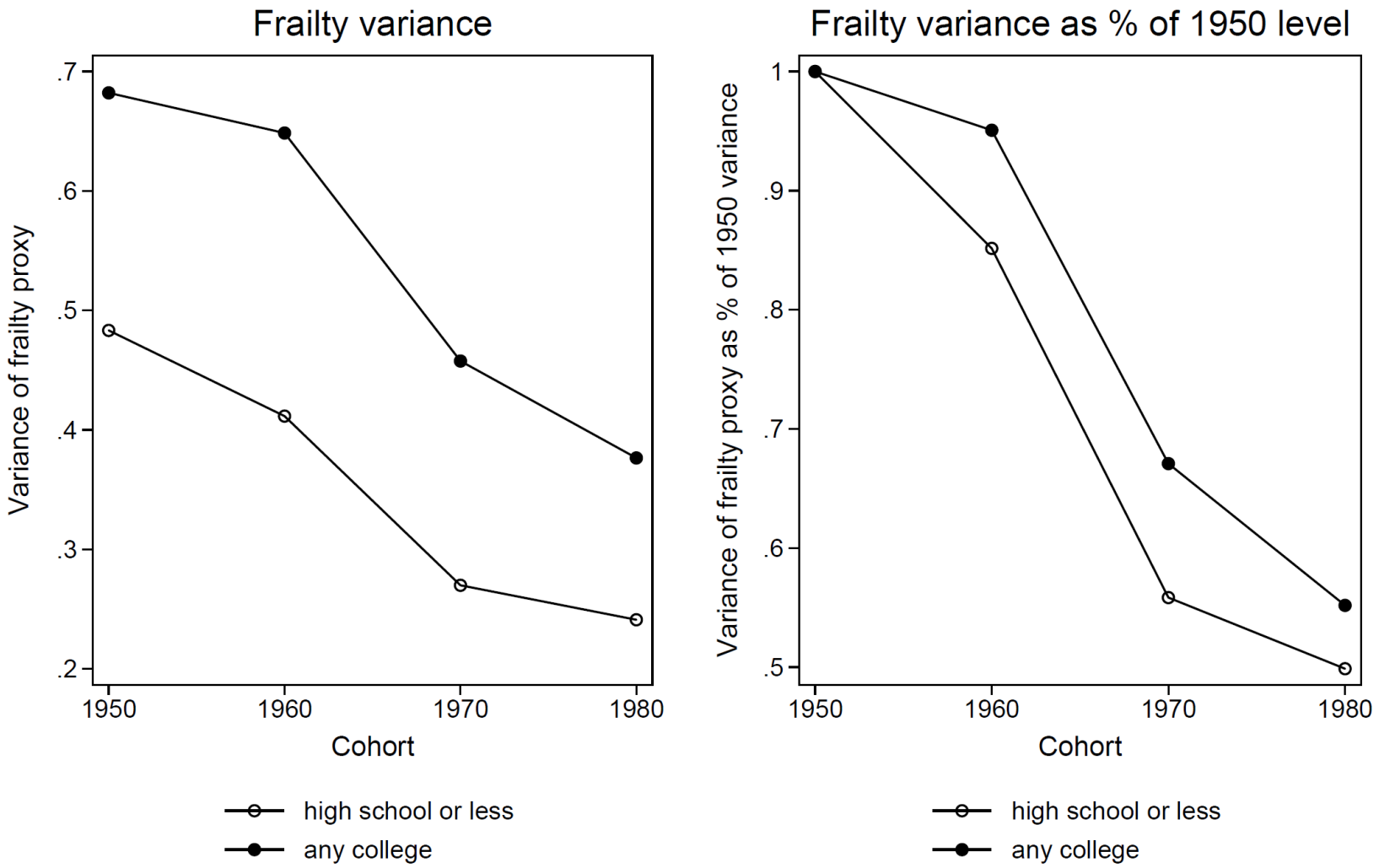
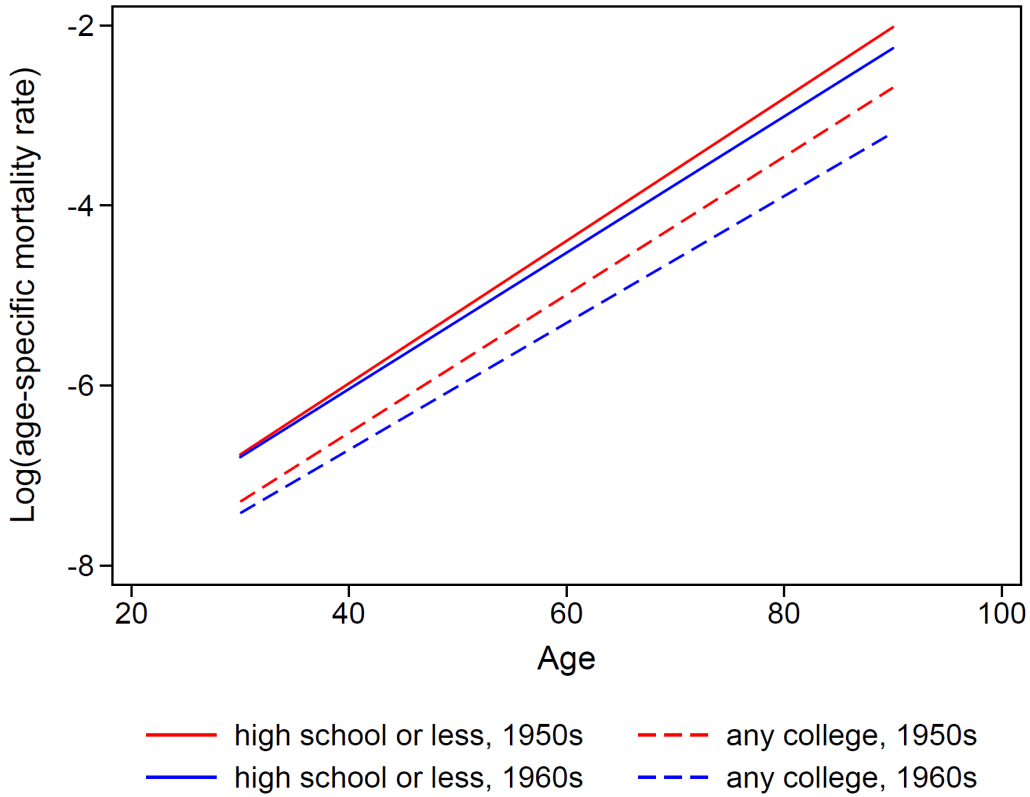


Figure 5. Absolute frailty variance and relative frailty variance as a percentage of the 1950 level for high school or less and any college groups from PSID 1968-2013



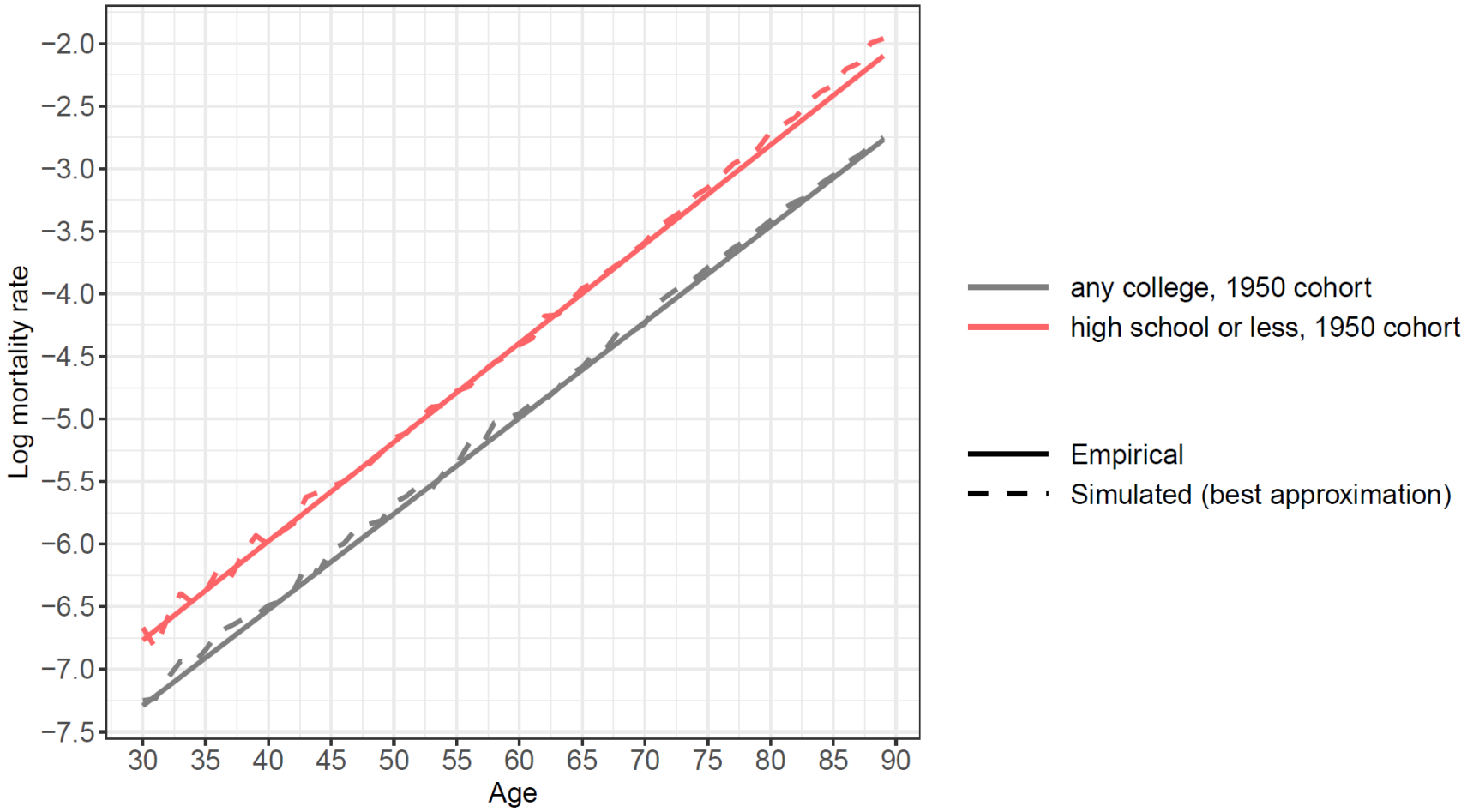
Notes: Data are from PSID 1968-2013. Sample consists of all individuals born from 1950-1989 who had information on early life disease measures before age 17. Sample size for these four cohorts is 11,394.

Figure 6. Observed and extrapolated mortality patterns for two education groups in the 1950s and 1960s birth cohorts from NHIS 1986-2009 with linked mortality through 2011



Notes: Data are from NHIS 1986-2009 surveys linked to mortality data through 2011. Sample size for the 1950s and 1960s birth cohorts is 575,705 experiencing 23,665 deaths and 8,509,452 person-years of exposure. Following the Gompertz function of age-dependent mortality pattern, we use a linear function of log mortality rate to extrapolate mortality rates up to age 90.

Figure 7. A comparison between a simulated mortality pattern using variance of frailty from calibration and an observed mortality pattern from NHIS data, 1950s birth cohort



Notes: Empirical data are from NHIS 1986-2009 surveys linked to mortality data through 2011. Sample size for the 1950s birth cohort is 320,173 experiencing 17,728 deaths and 5,093,549 person-years of exposure. Following the Gompertz function of age-dependent mortality pattern, we use a linear function of log mortality rate to extrapolate mortality rates up to age 90.

Appendix 1. Basic mathematical formulation for simulation

We start our simulation by setting up a model for individual hazard function. Following Vaupel et al. (1979), we let individuals in a cohort differ from each other in the value of frailty (denoted as z) characterizing their susceptibility to death, such that the force of mortality conditional on z is

$$\mu_i(x) = z_i \mu_0(x) \quad (1)$$

where $\mu_i(x)$ is the force of mortality for individual i at instantaneous age x, z_i is frailty for individual i at the initial age, and $\mu_0(x)$ 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. An individual with a frailty of 0.5 is only half as likely to die. Frailty z_i follows a Gamma distribution at the initial age, with *p.d.f.*:

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

where λ and k are the parameters of the distribution. The mean and variance of a Gamma variable are given by:

$$\bar{z} = k/\lambda \quad (3a)$$

$$\sigma^2 = k/\lambda^2 \quad (3b)$$

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 λ, and the variance of frailty distribution σ^2 equals the inverse of k.

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

$$\bar{\mu}(x) = \frac{\mu_0(x)}{1+\sigma^2 H(x)}, \quad (4)$$

where the cumulative hazard function from initial age to age *x* is $H(x) = \int_0^x \mu_0(t)dt$ (Vaupel et al. 1979). From comparing formula (2) and (4), we see that cohort mortality function $\bar{\mu}(x)$ deviates from individual hazard function $\mu_0(x)$. The higher value of the variance of frailty distribution σ^2, the more the slope of $\bar{\mu}(x)$ deviates from that of $\mu_0(x)$; the deviation also increases with age as $H(x)$ is an increasing function of x (Yashin et al. 2002).

The theory of population heterogeneity posits that death selectively removes the frailest members of a cohort so that the mortality rate at cohort level becomes increasingly dominated by robust members over the life course (Vaupel et al. 1979; Vaupel and Yashin 1985). This means that the individual hazard curve should be steeper than the cohort mortality curve, or individuals "age" faster than heterogeneous cohorts (Vaupel and Yashin 1983). 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.

Even though cohort mortality function $\bar{\mu}(x)$ 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. 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:

$$\mu_0(x)=ae^{bx}, \quad (5)$$

where a is the hazard at initial age and b is the rate of mortality acceleration. Replacing $\mu_0(x)$ in formula (1) with formula (5), we get the force of mortality for individual i at instantaneous age x

$$\mu_i(x) = z_i a e^{bx}, \tag{6a}$$

or the logarithm form:

$$\ln\big(\mu_i(x)\big) = \ln(a) + bx + \ln(z_i). \tag{6b}$$

The rate of increase in mortality rate at age x is the derivative of $\ln\big(\mu_i(x)\big)$ at age x, i.e., $\frac{d\ln(\mu_i(x))}{dx} = b$. 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 (6a) or (6b), the simulated cohort mortality curve will "age" even slower than Gompertz, which does not accurately characterize the empirical cohort mortality pattern. Our simulations based on this Gompertz specification, presented in Appendices 2-4 as Model specification A, are also consistent with this view.

Instead, Vaupel and Yashin (1983) showed that the individual mortality curve can be specified in a different way so that the population-level mortality curve closely approximates the empirical pattern. Following their formulation, we assume individual baseline hazard function as

$$\mu_0(x)=a e^{bx} e^{\frac{a(e^{bx}-1)}{b}}, \tag{7}$$

where the force of mortality for individual i at instantaneous age x is

$$\mu_i(x) = z_i a e^{(bx)} e^{\frac{a(e^{bx}-1)}{b}}, \tag{8a}$$

or

$$\ln\big(\mu_i(x)\big) = \ln(a) + bx + \frac{a(e^{bx}-1)}{b} + \ln(z_i). \quad (8b)$$

The rate of increase in mortality rate at age *x* is the derivative of $\ln\big(\mu_i(x)\big)$ at age *x*, i.e., $\frac{d\ln(\mu_i(x))}{dx} = b + ae^{bx}$. In other words, the rate of individual mortality acceleration is a Gompertz function of age *x*; it increases as age increases. More importantly, by assuming individual hazard function as formula (8a) or (8b), the simulated cohort mortality curve will follow a Gompertz function $\bar{\mu}(x) = ae^{\beta x}$ or $\ln\big(\bar{\mu}(x)\big) = \ln(a) + \beta x$, which is consistent with empirical pattern. The group-level rate of mortality acceleration is β. Our simulations based on this specification, presented in Appendices 2-4 as Model specification B, are also consistent with this view.

We follow Vaupel and Yashin (1983) in specifying the frailty distribution as a Gamma distribution, but alternative distributions, namely Weibull and Lognormal distributions, are used in robustness checks and presented in Appendices 2-4. Specifying the frailty distribution as Gamma and Weibull distribution both provide reasonably good approximation of cohort mortality curve. We keep with previous literature such as work by Vaupel and Yashin in using the Gamma distribution, and the results are similar under a Weibull distribution (see Appendix 4).

Appendix 2. Robustness checks for alternative specifications of mortality

In this appendix, we examine the robustness of our results with respect to alternative specifications of (1) the functional form of the individual-level force of mortality and (2) the distribution of frailty in the population. The exact parametric forms, in themselves, are unobserved and therefore cannot be directly tested. Therefore, previous literature (e.g., Vaupel and Yashin 1983, 1985) has typically relied on the implied cohort-level age-specific mortality rates as a benchmark for selecting the proper form of these individual-level parameters. This literature suggests that to fit the observed empirical pattern of cohort mortality rate, which follows a Gompertz curve, the individual mortality hazard is best specified as the form of : $\mu_i(x) = z_i a e^{(bx)} e^{\frac{a(e^{bx}-1)}{b}}$, and the frailty distribution in the population is best fitted as a gamma distribution (see Vaupel and Yashin 1983).

In our main simulations, we follow this literature closely in selecting our preferred model specifications for individual mortality hazard and frailty distribution. In addition, alternative specifications are examined as robustness checks. These alternative specifications are presented in Appendix 3 and resulting logged cohort mortality curves are given in Appendix 4. The first row of Appendix 4 presents simulation results for logged cohort mortality in which the functional form of individual mortality hazard is changed to a Gompertz curve: $\mu_i(x) = z_i a e^{(bx)}$, and we vary the functional form and variances of the frailty distribution. We fix the mean frailty at one unit in all these simulations. In the second row of this figure, we keep the individual mortality hazard as specified in our main simulations, and the frailty distribution is altered in the same way as it is in the first row. Vaupel and Yashin (1983) also listed examples of other functional forms of individual mortality hazard, but as their mathematical derivation

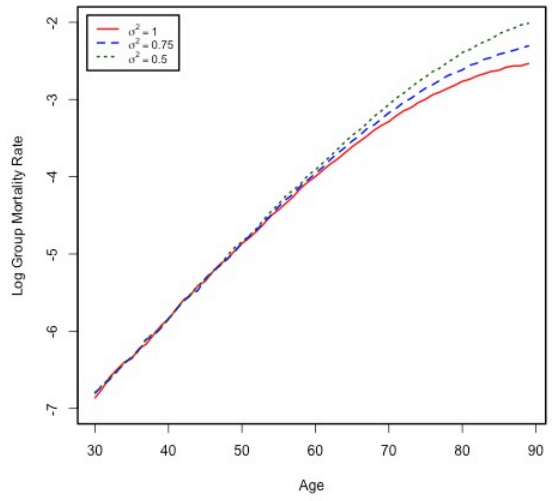
shows, other functional forms do not generate an increasing cohort mortality rate over age and are therefore omitted in our analysis.

Our additional simulation results yield four conclusions that all support our choice of model specifications in our main simulations. First, although a natural choice for the functional form of mortality pattern is to choose a Gompertz mortality curve at the individual level, we found that, if the individual-level mortality hazard is specified as Gompertz, the implied cohort mortality pattern will not follow the observed Gompertz curve. Second, in our specification in the main simulation analysis, as well as in Vaupel and Yashin's original specifications, the individual-level mortality hazard curve that takes the form of $\mu_i(x) = z_i a e^{(bx)} e^{\frac{a(e^{bx}-1)}{b}}$ generates a Gompertz pattern on the cohort level, which closely approximates the empirical pattern. Third, specifying the frailty distribution as Gamma and Weibull distribution both provide reasonably good approximation of cohort mortality curve. We keep with previous literature such as work by Vaupel and Yashin in using the Gamma distribution, and the results are similar under a Weibull distribution. Fourth, even though alternative specifications generate different shapes of the cohort mortality curve, it is reassuring to see, from comparing the curves within each sub-plot of Appendix 4, that our main conclusion—that the cohort mortality increases at a faster rate over age when the variance of frailty is smaller—holds throughout all specifications.

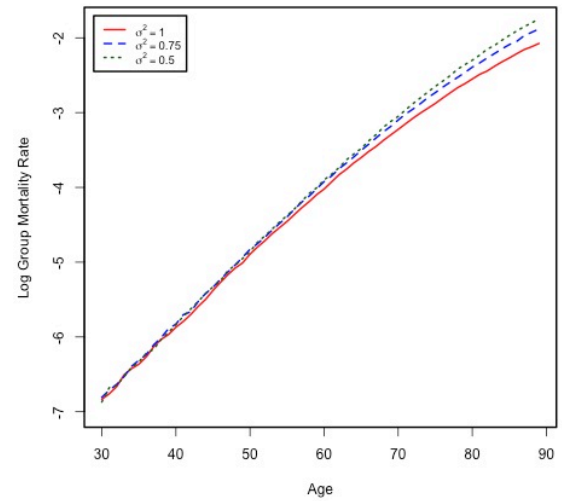
Appendix 3. Alternative specifications with different frailty distributions and different functional forms of the individual-level force of mortality

	Frailty Distribution		
	Gamma	**Weibull**	**Lognormal**
Model Specification A: individual-level force of mortality $\mu_i(x) = z_i a e^{(bx)}$			
	$\sigma^2 = 1$	$\sigma^2 = 1$	$\sigma^2 = 1$
Frailty Variance	$\sigma^2 = 0.75$	$\sigma^2 = 0.75$	$\sigma^2 = 0.75$
	$\sigma^2 = 0.5$	$\sigma^2 = 0.5$	$\sigma^2 = 0.5$
Model Specification B: individual-level force of mortality $\mu_i(x) = z_i a e^{(bx)} e^{\frac{a(e^{bx}-1)}{b}}$			
	$\sigma^2 = 1$	$\sigma^2 = 1$	$\sigma^2 = 1$
Frailty Variance	$\sigma^2 = 0.75$	$\sigma^2 = 0.75$	$\sigma^2 = 0.75$
	$\sigma^2 = 0.5$	$\sigma^2 = 0.5$	$\sigma^2 = 0.5$

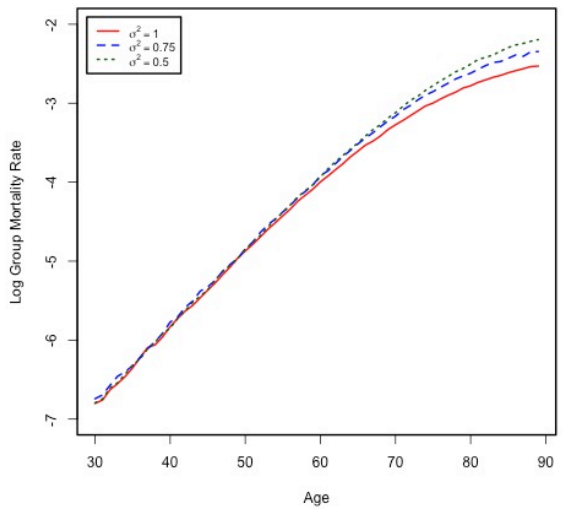
Appendix 4. Simulation results for alternative specifications with different frailty distributions and different functional forms of the individual-level force of mortality



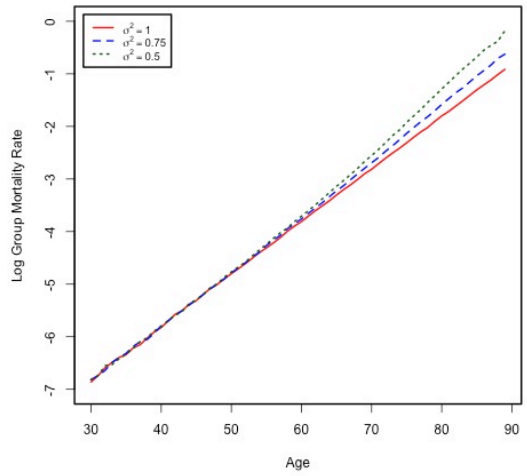
(a) Model specification A, Gamma frailty



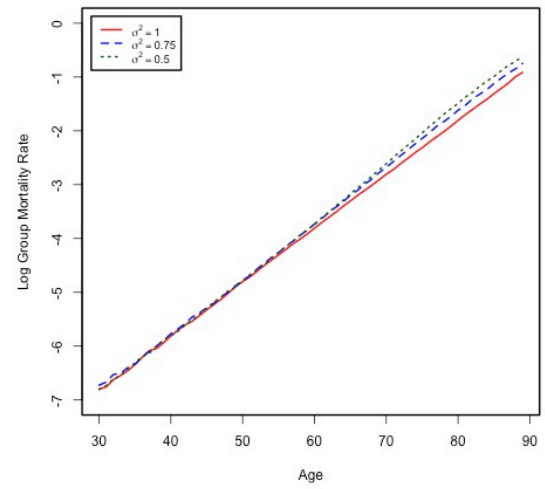
(b) Model specification A, Weibull frailty



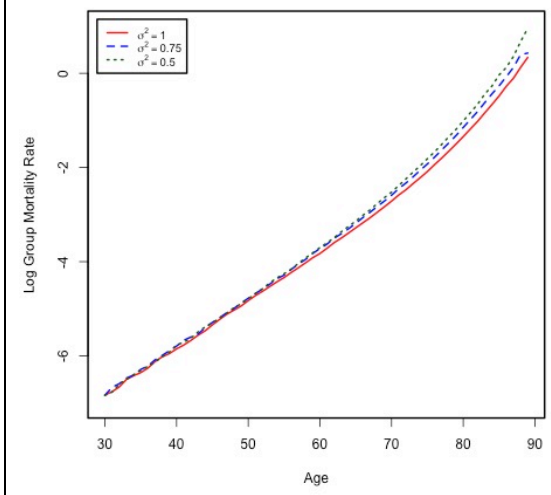
(c) Model specification A, Lognormal frailty



(d) Model specification B, Gamma frailty

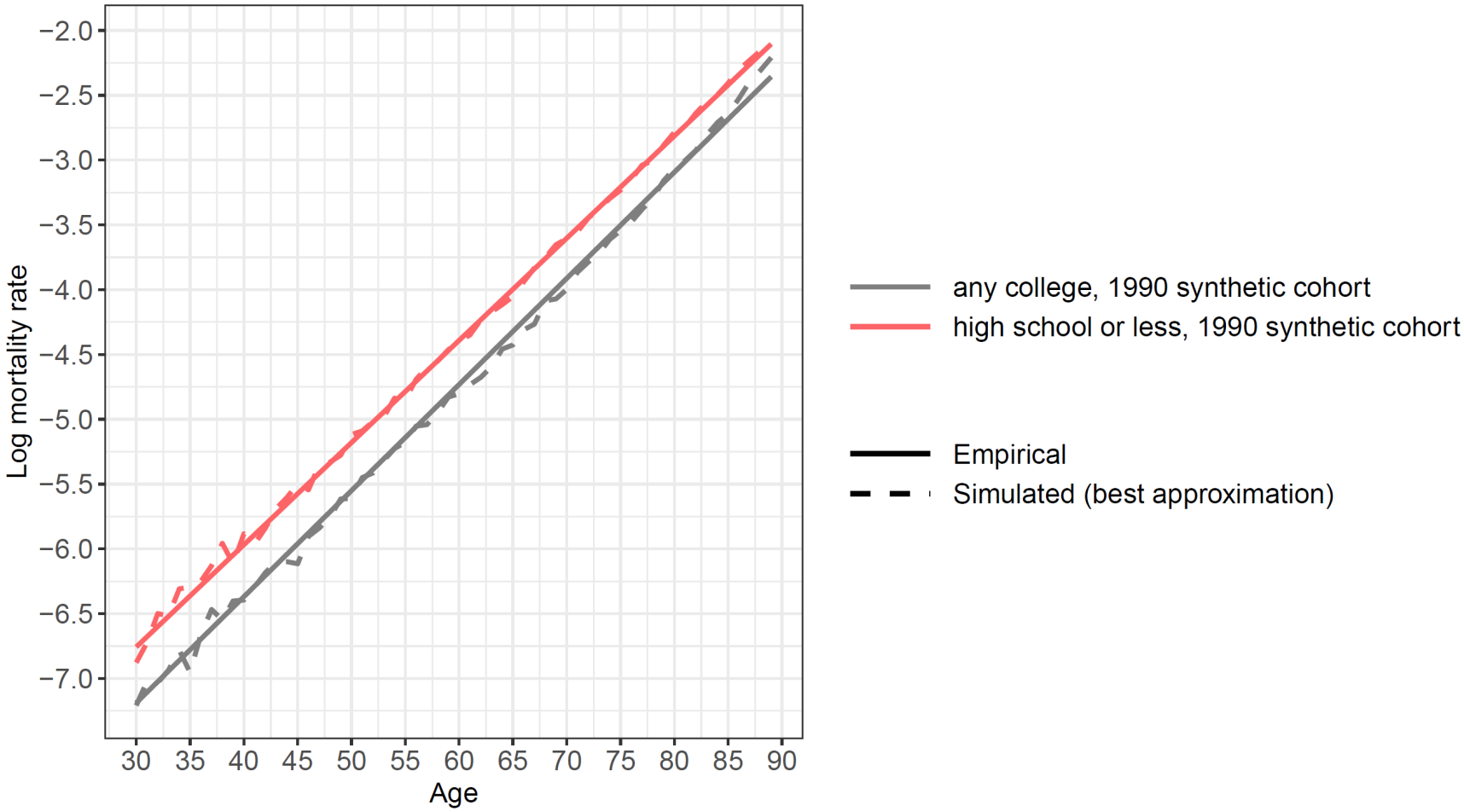


(e) Model specification B, Weibull frailty



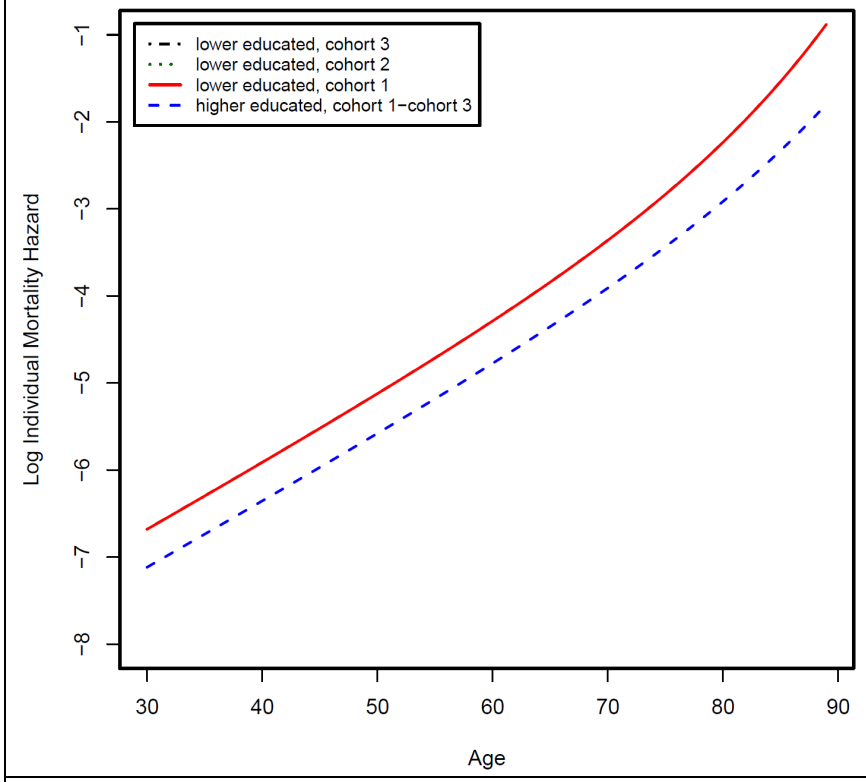
(f) Model specification B, Lognormal frailty

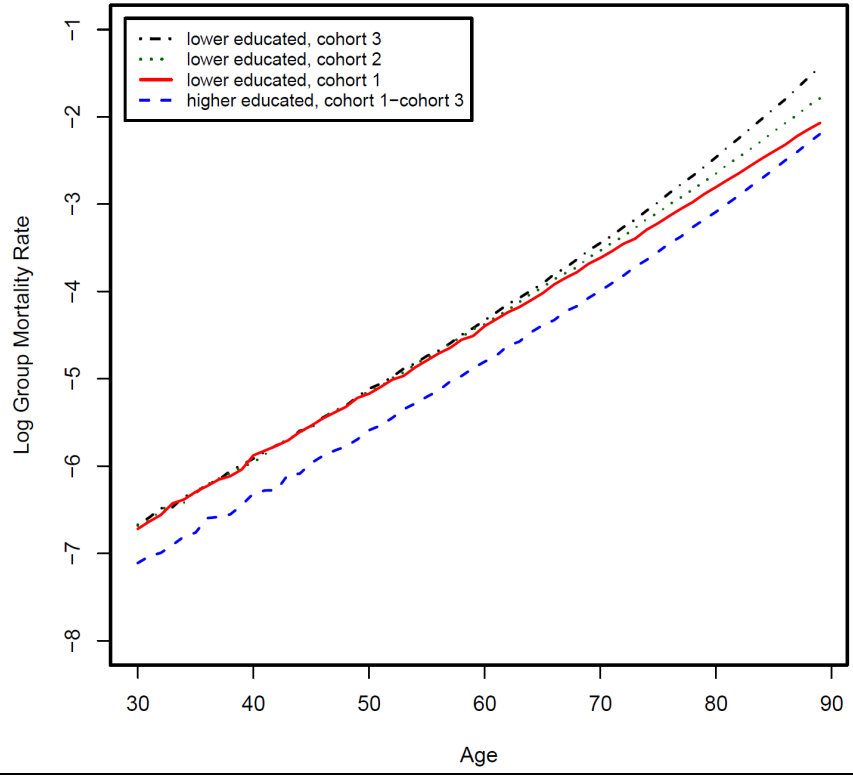
Appendix 5. A comparison between a simulated mortality pattern using variance of frailty from calibration and an observed mortality pattern from NHIS data, 1990 synthetic birth cohort



Notes: Empirical data are from NHIS 1986-2009 surveys linked to mortality data through 2011. Sample size for the 1990 synthetic birth cohort is 253,367 experiencing 2,599 deaths in 1990. Following the Gompertz function of age-dependent mortality pattern, we fit a linear function of log mortality rate to smooth the trend.

Appendix 6. Mortality differentials between lower-educated and higher-educated groups over the life course across multiple birth cohorts assuming decreasing frailty variance among the lower-educated group

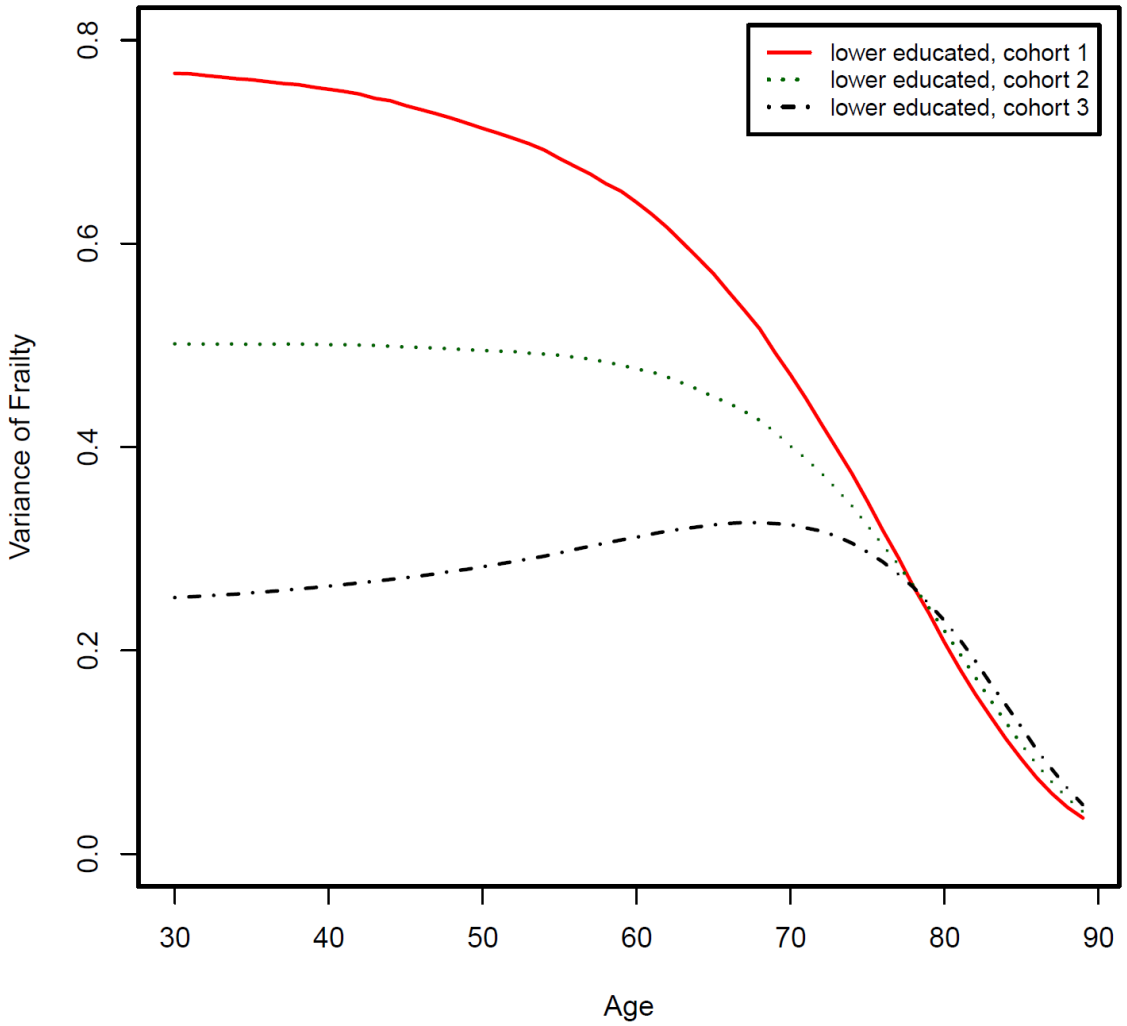




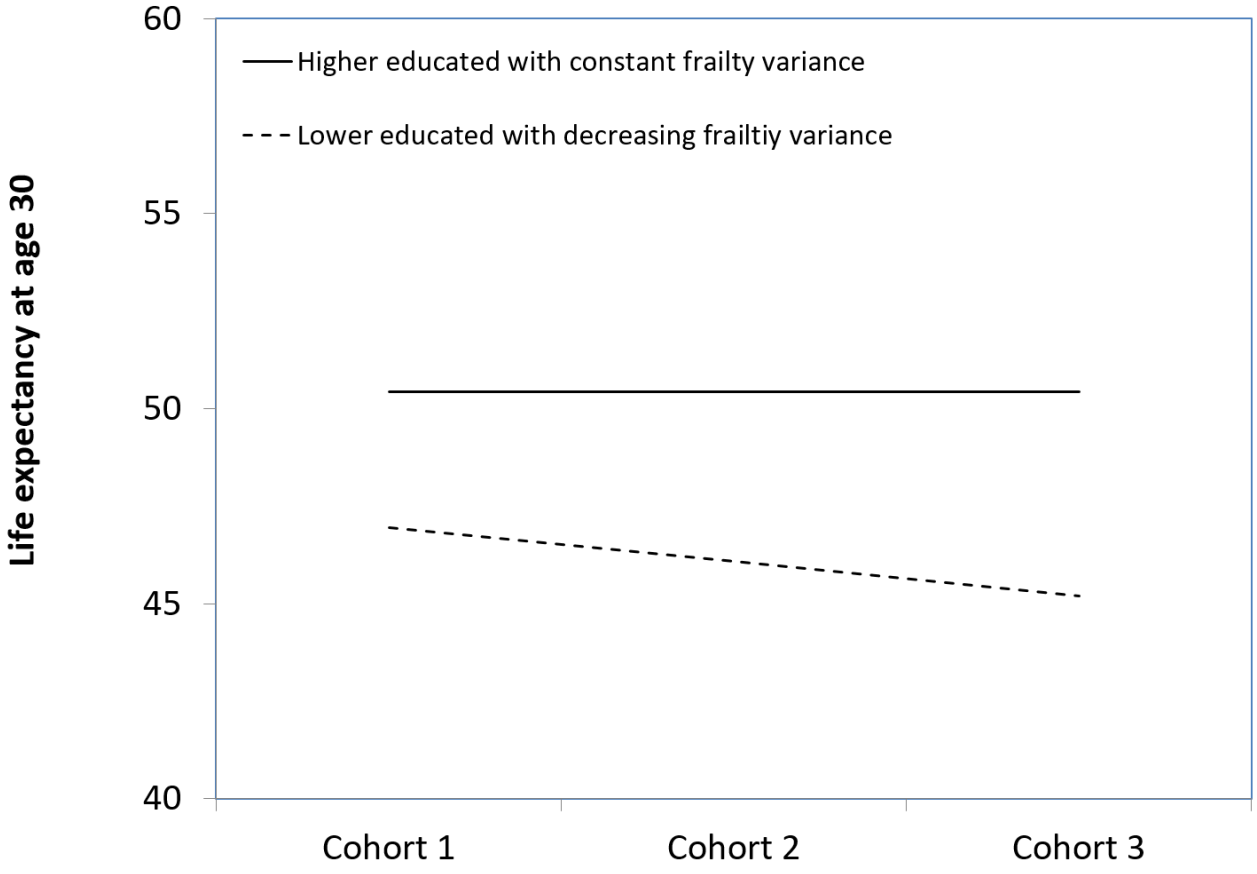
Individual	$\ln(a)$	z_i	b
Lower educated, cohort1	-6.76	1.00	0.075
Lower educated, cohort2	-6.76	1.00	0.075
Lower educated, cohort3	-6.76	1.00	0.075
Higher educated, cohort1-cohort3	-7.19	1.00	0.075

Group	$\ln(a)$	σ^2	b
Lower educated, cohort1	-6.76	0.77	0.075
Lower educated, cohort2	-6.76	0.50	0.075
Lower educated, cohort3	-6.76	0.25	0.075
Higher educated, cohort1-cohort3	-7.19	0.34	0.075

Appendix 7. Variance of frailty over the life course among three hypothetical cohorts of the lower-educated group

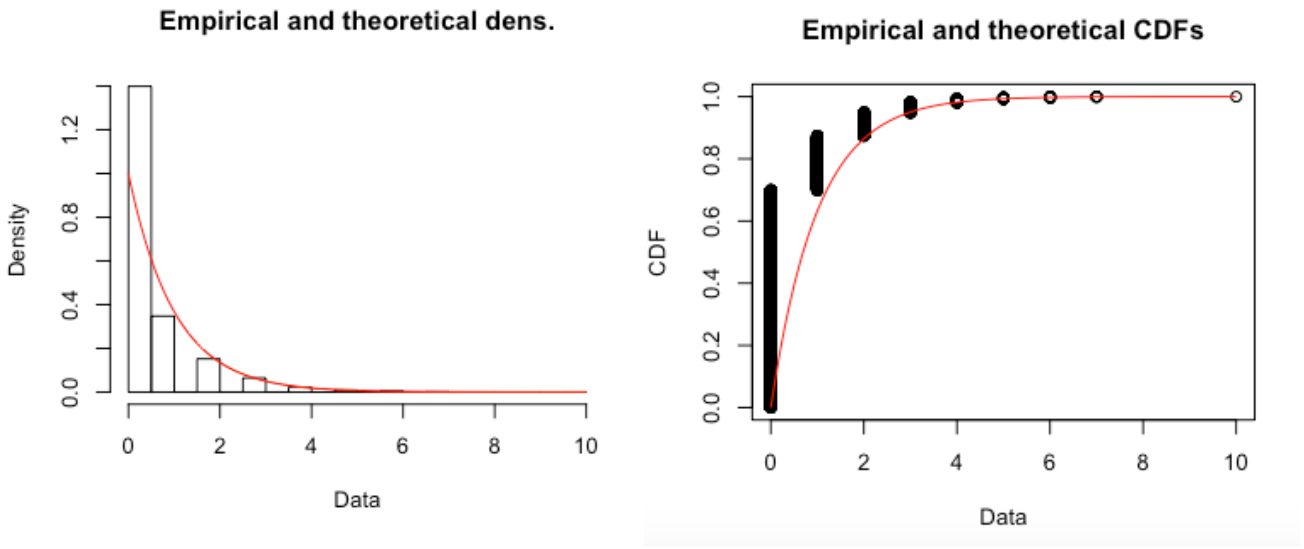


Appendix 8. Gaps in life expectancy at age 30 between lower-educated and higher-educated groups across three hypothetical cohorts assuming decreasing frailty variance among the lower-educated group

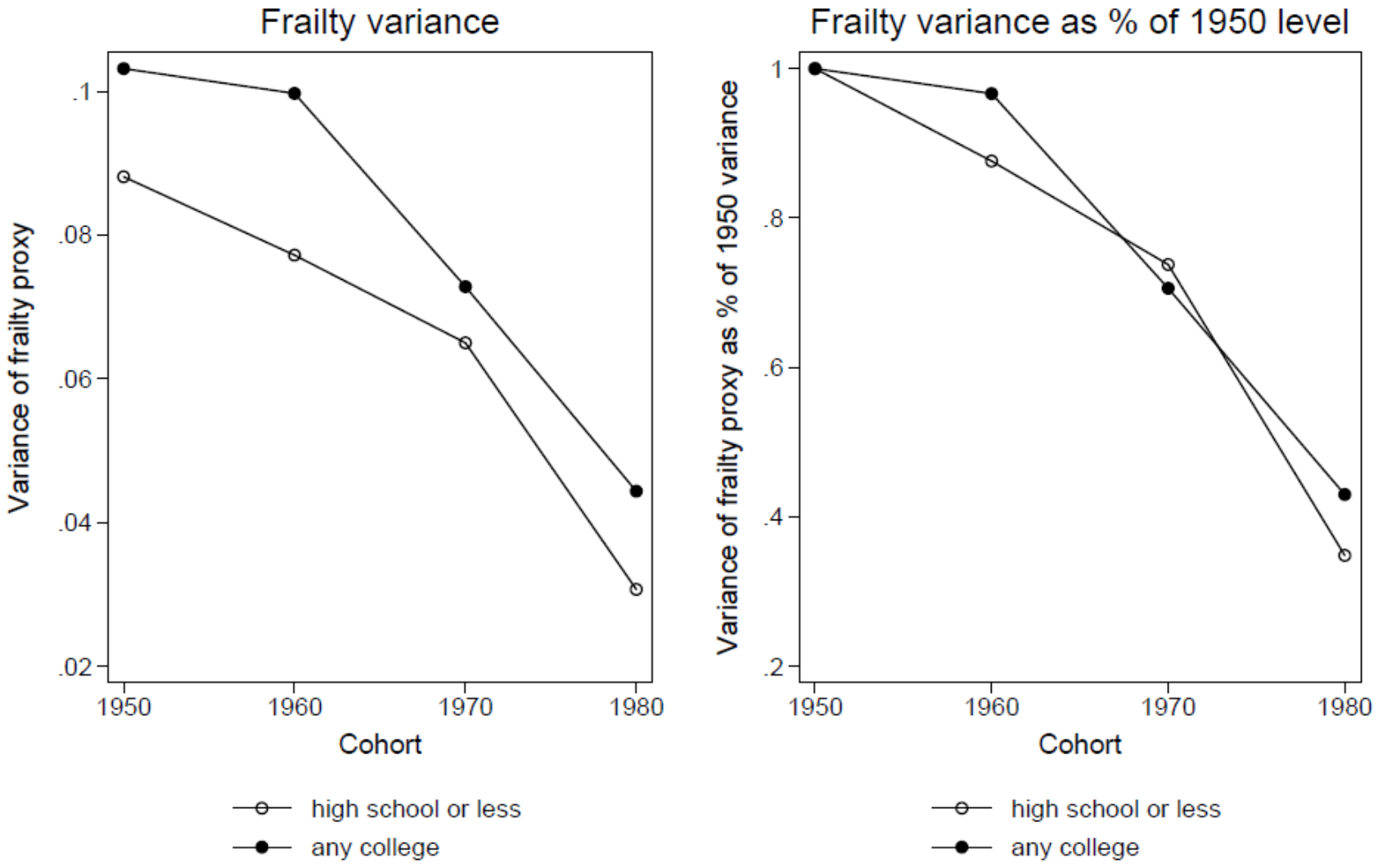


Appendix 9. Empirical (histogram) and Theoretical (Gamma Distribution, red smooth line) Distributions of the Frailty Measure Constructed from PSID Data

To examine the distributional properties of our constructed frailty measure, we fit a Gamma distribution to the empirical distribution of our PSID-based frailty measure and compare them. The theoretical Gamma density is fitted using maximum likelihood estimation. The left panel of the figure compares the empirical and theoretical density functions, and the right panel of the figure compares the two cumulative distribution functions (CDF). The two panels suggest that the empirical distribution of our constructed frailty measure can be reasonably approximated by the Gamma distribution. In sum, while there is no perfect measure for the frailty distribution in the population, the distributional properties of our frailty measure seem to be generally consistent with our assumption of the Gamma distribution in the simulation models.



Appendix 10. Absolute frailty variance and relative frailty variance as a percentage of the 1950 level for high school or less and any college groups from NHANES 1999-2012



Notes: Data are from NHANES 1999-2012. Sample consists of all individuals born from 1950-1989 who had information on early life disease measures before age 17. Sample size for these four cohorts is 22,181.

Appendix 11. Life expectancies at age 30 in high school or less and any college groups in the 1950s and 1960s birth cohorts from empirical and simulated data using NHANES frailty measure

	High school or less	Any college	
	Empirical		*C-H*
Cohort 1950s	47.02	52.72	5.70
Cohort 1960s	48.52	55.28	6.76
1960s-1950s	1.50	2.56	**1.06**
	Simulated (mortality selection present)		*C-H*
Cohort 1950s	46.65	52.38	5.73
Cohort 1960s	46.74	53.39	6.65
1960s-1950s	0.09	1.00	**0.92**
	Simulated (mortality selection absent)		*C-H*
Cohort 1950s	46.65	52.38	5.73
Cohort 1960s	46.98	53.43	6.46
1960s-1950s	0.33	1.05	**0.72**

Notes: Empirical data are from NHIS 1986-2009 surveys linked to mortality data through 2011. Sample size for the 1950s and 1960s birth cohorts is 575,705 experiencing 23,665 deaths and 8,509,452 person-years of exposure. Following the Gompertz function of age-dependent mortality pattern, we use a linear function of log mortality rate to extrapolate mortality rates up to age 90. Based on these observed and extrapolated mortality rates, we construct life tables and calculate life expectancies at age 30.