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Variation and Vulnerability in Aging Populations

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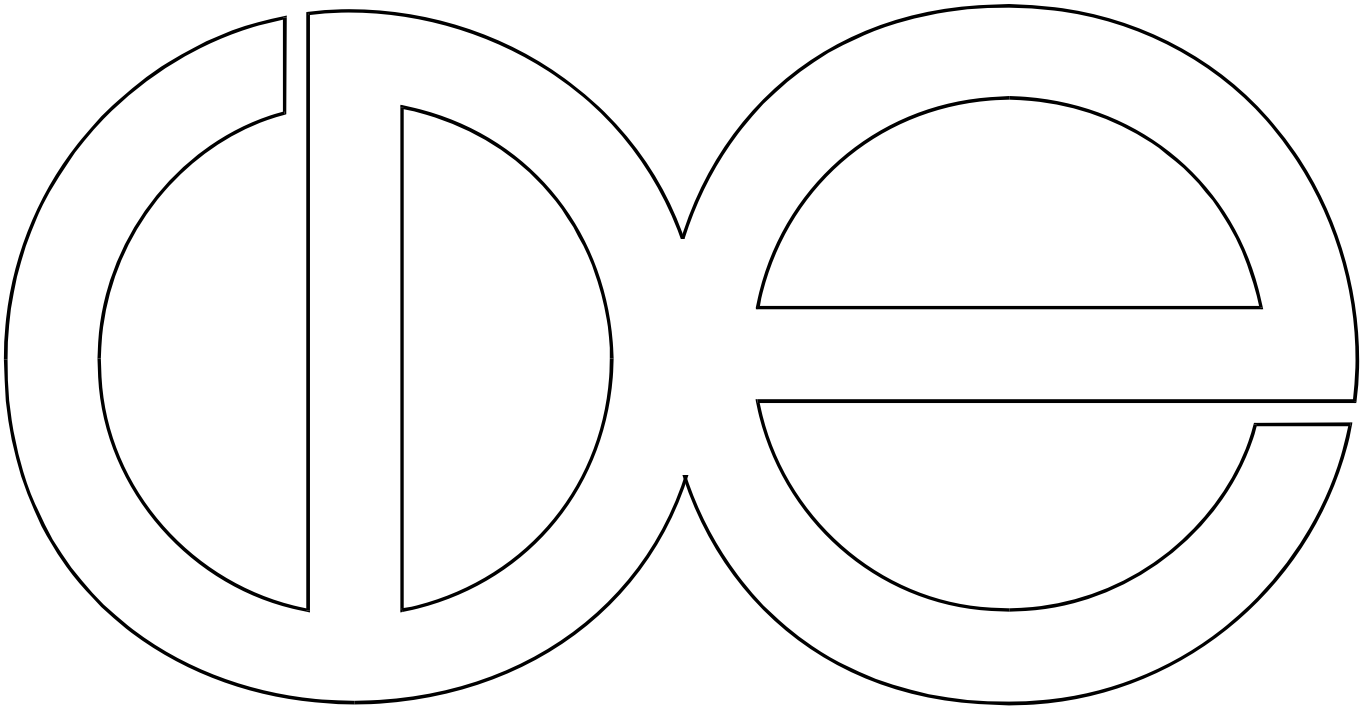

Frailty in Transition: Variation and Vulnerability in Aging Populations

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Abstract

This paper asks how the demographic and epidemiologic transitions of the past century and a half have influenced patterns of survival and health in aging populations. We hypothesize that survival improvements at younger ages may yield an older population with a more heterogeneous health profile relative to previous cohorts with higher early-life mortality. To test this hypothesis, we develop a theoretical and statistical rationale for a frailty model designed to examine changes in the composition of successive aging cohorts. The model incorporates a cohort-specific frailty indicator into an equation describing the mortality hazard trajectory across the full lifespan, and its parameters are estimated using life table data for cohorts born 1885-1919. The findings indicate that more recent cohorts are more homogeneous with respect to the timing of mortality than earlier ones – but also less selected, as death culls fewer individuals in early life. We argue that due to ongoing survival improvements, delayed mortality selection has shifted disparities into later life, where they manifest in increasing mortality variability and growing heterogeneity in health.

Introduction: A Variation-Centered Perspective on the Demography of Aging

Due to celebrated declines in mortality and fertility, aging, once considered a notable population issue mainly in high-income nations, has become a global phenomenon. This paper asks how the demographic and epidemiologic transitions of the past century and a half have influenced patterns of health and survival in aging populations. The shifting experience and meaning of aging in global contexts has received attention from demographers (e.g. Treas and Logue 1986; Hermalin 2002) and other social scientists (e.g. Simmons 1945; Cowgill and Holmes 1972; Livingston 2003). One issue that still invites explicit consideration has to do with the relationships between broad demographic, epidemiological, and socioeconomic changes and the health profiles of the cohorts that age through them.

The life table indicator e_0, representing the average length of life or life expectancy at birth, has been a widespread measure of population health since the 1920s (Dublin 1923). Variability in the distribution of ages at death is likewise a key measure of uncertainty and inequality in population health. As life expectancy proceeded along a trajectory of dramatic increase, variability in the complete mortality distribution declined systematically (Wilmoth and Horiuchi 1999). Observing the growing rectangularization of the survival curve, Fries (1980) optimistically predicted that the compression of mortality into increasingly later ages would be followed by a compression in morbidity, affording most people lives that were not only long but also healthy.

Since Fries' landmark article, relatively few studies have simultaneously considered both questions of inequalities in longevity and historic mortality transitions. The literature on social inequalities in health tends to describe the differences in average levels of mortality between national populations or subpopulations defined by race and other socioeconomic attributes (e.g. Schroeder 2007), paying relatively less attention to within-group differences in longevity or to the differing experiences of cohorts aging during times

of marked demographic and socioeconomic change. A close examination of variability in mortality and health among aging populations provides one avenue for engaging with these issues. Recent empirical analysis of age-specific trends in 23 national populations has shown that variability in longevity has followed distinctive patterns for the young and old: while overall mortality variation has decreased as life expectancy at birth rose, survivors to older ages have become increasingly heterogeneous in their mortality risks (Engelman et al. 2010). What might account for these diverging trends?

All populations comprise individuals who vary greatly in their vulnerability to mortality of all causes. This heterogeneity in what demographers call "frailty" is often unmeasured, but in the past three decades a diverse group of statistical and population scientists have developed theories and methods for characterizing the impact of this hidden heterogeneity on observed hazard trajectories (Vaupel et al. 1979; Manton et al. 1981; Heckman and Singer 1984; Manton et al. 1986; Gage 1989; Hougaard 1995; Bandeen-Roche and Liang 1996; Yashin and Iachine 1997; Butt and Haberman 2004; Vaupel and Yashin 2006; Yashin et al. 2008; Li and Anderson 2009). Frailty models provide a powerful way for comparing heterogeneous populations by explicitly recognizing that any given population is composed of different sub-populations with varied risk profiles based on myriad social, economic, and demographic factors. Here, we consider changes in the frailty distribution across successive cohorts in the context of the rising variability of mortality among survivors to older ages.

Two assumptions about the sometimes slippery concept of frailty[1] have been central to the exposition of the simplest-case model: first, that individual frailty is fixed at birth, and second, that the profile of frailty within a cohort follows an analytically tractable gamma distribution with an initial mean of 1. Those with frailty < 1 are considered less

[1] In the study of aging, two distinct notions of frailty coexist: *demographic* frailty is a statistical concept referring to unobserved heterogeneity marking differential vulnerability to mortality within populations; *geriatric* frailty, on the other hand, is used to assess individual health status in clinical settings. This paper focuses on the former, demographic definition of frailty.

susceptible to mortality than a standard individual, while those with frailty > 1 are more vulnerable. All else being equal, as members of a given cohort age, the frailest individuals are removed from the population via selective mortality at a higher rate than their more robust contemporaries, leaving behind a smaller population whose distribution of frailty is shifted over towards the more robust end of the spectrum. Thus, both the mean and variance of frailty decrease with age, rendering the surviving members of the cohort more homogeneous with respect to the timing of death.

Several developments across age and time are likely to complicate this basic scenario. For one, while a static frailty model may be conceptually and statistically consistent with intrinsic susceptibility to mortality that increases with age, experiences and behaviors may nonetheless alter individual frailties across the lifespan in ways that might be informatively modeled. Whether due to programmed biology or accumulated exposure to environmental factors that wear down the human body (see Arking 2006 for a review of biological theories of aging), individuals do grow increasingly susceptible to mortality as they age. Thus, in formulating his *Law of Mortality*, Benjamin Gompertz (1825) noted that "it is possible that death may be the consequence of two generally co-existing causes; the one, chance, without previous disposition to death or deterioration; the other, a deterioration, or an increased inability to withstand destruction." This "increased liability to death," in Gompertz' words, suggests that individual frailty may change with age to reflect increased vulnerability, rather than remaining static. Lifetime experiences (e.g. illness, recovery, exposure to deprivation or abundance) may affect both trajectories of individual resilience and the dynamics of selective mortality in populations (Vaupel et al. 1988), and may be more effectively captured using more complex dynamic models (Yashin et al. 1994; 2008). While the subsequent analysis uses the fixed definition of frailty prevalent in the literature, the more dynamic clinical notion of geriatric frailty (discussed later) may help researchers understand how individual endowments of vulner-

ability may change with age or over time.

Another extension to the classical frailty framework has to do with the question of how the demographic and epidemiological transitions may have changed the distribution of frailty across successive cohorts, and what implications such changes may have for population health trends. Cohorts born during periods of demographic transition experience varied combinations of age-specific mortality changes. When mortality at younger ages is high, the challenges of reaching older ages fashion a highly selected group of survivors. As life expectancy rises, later cohorts experience less mortality selection at every age relative to earlier cohorts. Depending on the age-pattern of improvements to survival, such mortality changes may result in a larger proportion of frail individuals reaching older ages even as old-age mortality risks decline.[2] If the rates of survival improvement are slower at older ages than in early life, increased frailty on a population level may manifest itself in mortality (and possibly also health) patterns at later ages.

Below, we use successive cohort life tables and a frailty model to examine changes in population composition at older ages. Our findings indicate that more recent cohorts are more homogeneous with respect to the timing of mortality than earlier ones – but also less selected, as death culls fewer individuals in early life. We argue that the arrival of a less selected group of survivors to older ages may be shaping an older population whose mortality profile is more diverse relative to earlier populations at the same age, reflecting an ongoing shift of health disparities into later life. Because population patterns of health and mortality are closely intertwined, a more thorough understanding of the consequences of mortality transitions may be essential for predicting and planning for subsequent patterns in population health.

[2]Mortality improvements at young ages will increase old-age heterogeneity if survival improvements apply relatively uniformly to individuals at all frailty levels. Such a scenario may be unlikely in highly stratified societies (where health improvements result from technological or behavioral changes that differentially benefit the most socioeconomically advantaged), but is more plausible during the historical "public health revolution" period covered by this analysis, when improvements in sanitation and standards of living led to rising life expectancy and broad improvements in population health.

Methods and Materials

Modeling Frailty

Heterogeneity is a particularly salient concern in mortality analysis because the age pattern of mortality for a given cohort reflects both an underlying age trajectory of individual mortality risk as well as the effects of compositional changes within the population. This compositional change – the evolution of the cohort's heterogeneity as frail individuals are culled – is a mechanism that, while distinct from the biological age-dependent processes that influence senescence and mortality, operates in conjunction with them.

One key approach for capturing such compositional effects has been to treat heterogeneous populations as comprising homogeneous subgroups that differ in their susceptibility to mortality (Vaupel and Yashin 1985). Vaupel et al. (1979) proposed a proportional-hazard (relative risk) mixture model that includes an unobserved random variable – frailty – which explicitly represents all individual (or within-population) differences in the endowment for longevity. The model depicts the age-specific mortality hazard of any individual at age x with frailty level z relative to the hazard of a standard individual of the same age ($\mu(x)$) via:

$$\mu(x, z) = z\mu(x). \tag{1}$$

Thus, the nonnegative variable z acts as a multiplier, reflecting the proportionally increased or decreased vulnerability of a person with a specific frailty relative to a "standard" individual with frailty $z = 1$.

Since z is not observed for individuals and must be estimated from aggregate life table data, the identifiability of the heterogeneity distribution is a (long-recognized) problem. One solution (e.g. Vaupel et al. 1979) is to assume that frailty at any age follows a gamma

distribution with the probability density function:

$$f_z(z) = \frac{\lambda^\kappa z^{\kappa-1} e^{-\lambda z}}{\Gamma(\kappa)}, \tag{2}$$

with mean $\bar{z} = \frac{\kappa}{\lambda}$, and variance $\sigma^2 = \frac{\kappa}{\lambda^2}$. The κ parameter determines the shape of the distribution, while the λ parameter sets its scale. The biological basis of this assumption has been questioned, and nonparametric alternatives which correct for heterogeneity without identifying its distribution have been proposed (Heckman and Singer 1984). However, for those interested specifically in characterizing the heterogeneity in populations, gamma-based frailty models have the advantage of being analytically tractable, providing a good fit to the data, and being consistent with mortality's asymptotic properties (Manton et al. 1986; Yashin et al. 1994; Butt and Haberman 2004; Missov and Finkelstein 2011). Furthermore, empirical estimates have been shown (Manton et al. 1986) to be less sensitive to the specification of frailty distributions than to the choice of underlying conditional failure model.

Under the gamma assumption, the mean frailty for any cohort at birth is set at $\bar{z}(0) = 1$, so that $\kappa = \lambda$. One useful mathematical result (see Vaupel et al. 1979) of assuming that frailty at birth is gamma-distributed for a cohort, is that the distribution of frailty among survivors to any age x can then be shown to also be gamma-distributed, with the same value of the shape parameter κ. The value of the scale parameter λ does change as the cohort ages, taking the value

$$\lambda(x) = \lambda + H(x), \tag{3}$$

where $H(x)$ is the cumulative hazard of mortality up to age x, $H(x) = \int_0^x \mu(t)\, dt$.

Using this relationship, we can obtain the mean frailty for survivors to any subsequent

age x via:

$$\bar{z}(x) = \left(1 + \sigma_0^2 H(x)\right)^{-1}, \tag{4}$$

where σ_0^2 is the variance of the cohort's frailty distribution at birth. The observed population hazard trajectory can be shown to be a function of mean frailty in the population:

$$\bar{\mu}(x) = \bar{z}(x)\mu(x). \tag{5}$$

Combining equations (4) and (5) shows that "injecting" frailty into a basic hazard model gives rise to a logistic-type function, which more precisely accounts for the deceleration in the force of mortality at older ages than models that assume a continued exponential increase throughout adulthood (Vaupel et al. 1979; Thatcher et al. 1998; Vaupel and Yashin 2006).

Frailty models address variance more explicitly than models focusing solely on the age trajectories of mortality hazards and thus offer a promising avenue for investigating mortality variation trends. Using the definition of variance in a gamma distribution and the changing value of λ described in equation (3), the variance of the frailty distribution at any age x can be calculated as a function of the variability of frailty in the cohort at birth (σ_0^2) as well as the cumulative hazard of mortality up to age x.

$$\sigma^2(x) = \frac{\kappa}{(\lambda + H(x))^2} = \left(\frac{1}{\sigma_0^2} + 2H(x) + \sigma_0^2 \left(H(x)\right)^2\right)^{-1}. \tag{6}$$

The gamma assumption thus allows the characterization of both the mean and variance of the frailty distribution at any age. Under this framework, changes in the value of σ_0^2 represent changes in overall variation within successive populations over time, while the pattern of change in $\sigma^2(x)$ reflects the effect of mortality selection with age. We followed the methodology described by Manton et al. (1981) to visualize the changing distribution of frailty within and across cohorts, plotting the gamma distributions of

frailty for selected cohorts at birth, and for survivors to ages 45, 65, and 85. In each case, the plot is normalized to a probability mass equal to each cohort's survivorship proportion at age x, $l_c(x)$. This normalization procedure standardizes the frailty distribution across scales, thereby facilitating comparisons across time and age.

Tuljapurkar and Edwards (2009) demonstrated that adding gamma-distributed frailty to the traditional Gompertz hazard model amplifies the variance of the associated mortality distribution, capturing the observed trend in mortality variability more accurately than the standard model or its logistic extension. Their mathematical analysis showed that changes in mortality variance over time are due either to changes in the Gompertz slope parameter β or to changes in the variance of frailty (σ_0^2) over time. Finding no clear interpretation for the latter result, they noted that "temporal change in frailty has not been a feature of mortality models."

Changes in the distribution of frailty during the course of mortality transitions are, however, broadly accommodated within the theory of heterogeneity (Vaupel et al. 1979). As mentioned above, the theory predicts that within cohorts, both the mean and variance of frailty will decrease with age, rendering the surviving members of the cohort more homogeneous with respect to the timing of death. Progress in reducing mortality at younger ages would allow a greater proportion of individuals (including frail ones) to survive to older ages. The seemingly counterintuitive result – an apparent plateau or increase in mortality hazards due to changing population composition – is one of heterogeneity's classic ruses (Vaupel and Yashin 1985, Fig. 11).

Extending this line of thought, we hypothesize that cohorts aging through periods when mortality is declining may be expected to be more heterogeneous (that is, have a larger distribution of frailty at a given age) than a comparable cohort which did not experience lower mortality rates. Such an expansion of frailty should become especially apparent at older ages, when mortality rates and survival changes are substantial enough

to alter the population composition.[3]

An altered frailty distribution may, in turn, contribute to the observed pattern of increased variation around the mean of old age mortality (Engelman et al. 2010). A steady but slow pace of survival improvement at older ages (Finch and Crimmins 2004; Robine et al. 2006) may have fostered a dynamic with gradually declining mortality rates at older ages and, at the same time, variability trends that hint at the changing population composition. Below, we describe a frailty model that builds on a hazard trajectory covering the full age spectrum, and then use life table data to estimate its parameters and obtain insights into the dynamics of unobserved heterogeneity over time.

The Siler Mortality Trajectory

To illustrate the way demographic heterogeneity may affect the pattern of mortality variability across the full age spectrum, we examine a model of population change that assumes the hazard of mortality on the individual level follows a Siler (1979) trajectory. We chose this model, rather than the more-commonly used Gompertz (1825), because the latter includes information only on adult mortality risks whereas the Siler model follows the entire life course, including three terms that describe (1) an exponentially declining hazard describing mortality in childhood, (2) an exponentially increasing (Gompertz-like) hazard characterizing mortality in adulthood, and (3) a constant component reflecting a certain "background" mortality level (see figure 1 for an illustration).

[Figure 1 about here]

The Siler model fits as well or better than most other models to human mortality data (Gage and Dyke 1986; Gage and Mode 1993), and its three components have been shown to be biologically coherent (Gage 1991). It has been used to describe changes

[3]This is particularly the case if progress in reducing mortality at older ages is not sufficiently fast or large to counterbalance the effect of improved survival at younger ages. Then, rising levels of frailty would mask the full scale of survival improvement, causing mortality reductions to be underestimated.

in mortality during the demographic transition (Canudas-Romo 2008), and a simulation study demonstrated that the Siler model is better able to capture the divergence in the age-specific variability trends over the course of the demographic transition (results available upon request). Representing the full age range is key for our purpose, because we are interested in how reductions in mortality in early and mid-life influence the distribution of frailty and subsequent variation in later-life mortality.

The basic Siler model is given by:

$$\mu(x) = e^{\alpha_1 - \beta_1 x} + e^{\alpha_2 + \beta_2 x} + e^{\alpha_3}, \tag{7}$$

where the α constants describe the base hazard levels and the β parameters represent fixed rates of mortality decline or increase over age. Under this model, the cumulative hazard function $H(x)$ is given by:

$$H(x) = \frac{-1}{\beta_1}(e^{\alpha_1 - \beta_1 x} - e^{\alpha_1}) + \frac{1}{\beta_2}(e^{\alpha_2 + \beta_2 x} - e^{\alpha_2}) + x e^{\alpha_3}. \tag{8}$$

Combining the Siler model in equation (7) with the gamma-distributed age-specific frailty, the observed population trajectory of mortality described in equation (5) may thus be represented via the logistic-like Siler-gamma model:

$$\bar{\mu}(x) = \frac{e^{\alpha_1 - \beta_1 x} + e^{\alpha_2 + \beta_2 x} + e^{\alpha_3}}{1 + \sigma_0^2(\frac{-1}{\beta_1}(e^{\alpha_1 - \beta_1 x} - e^{\alpha_1}) + \frac{1}{\beta_2}(e^{\alpha_2 + \beta_2 x} - e^{\alpha_2}) + x e^{\alpha_3})}. \tag{9}$$

The theoretical properties of the combined Siler-gamma model have been described in the literature (Gage 1989), but to our knowledge, the model has not been previously applied to analyses of empirical life tables. Here, we use newly-developed optimization techniques and data on successive cohorts of Swedish females to explicitly estimate the six parameters of the Siler-gamma model in order to explore trends in child, adult, and

background mortality and, most notably, population heterogeneity.

Data

The Human Mortality Database (HMD 2012) contains detailed time series of mortality data and life tables for populations with reliable vital registration and census data. To examine the changing distribution of ages at death for cohorts born during mortality transitions, we use data on females from Sweden – the nation with the longest and most reliable time-series of vital statistics. The HMD presents cohort life tables for all extinct cohorts – those whose members are assumed to have all died by the end of the observation period – and some almost-extinct cohorts, where death rates for ages not yet observed are based on the average experience of previous cohorts. For complete life tables beginning at age 0, the HMD requires that the total projected exposure (in person-years lived) for unobserved ages be no more than one percent of the total lifetime exposure for the cohort. Thus, the most recent life table available for Sweden is for the cohort born in 1919.

While more recent phases of the mortality transition are less observable in the available cohort life tables than in their period counterparts, a cohort analysis depicts changes in real cohorts whose mortality experiences underlies the synthetic patterns observed in the period data. Since the influence of mortality reductions on cohorts becomes noticeable in the latter part of the 19th century (Engelman et al. 2010), this analysis focuses on those cohorts born between 1885-1919.

Parameter Estimation

For a given life table cohort, the hazard of dying at age x, $\mu(x)$, was defined according to the Siler-gamma hazard in equation (9). We used nonlinear least squares minimization to estimate the six Siler-Gamma parameters based on log-transformed mortality hazards calculated from Swedish life tables for successive cohorts of females. In the nonlinear least

squares approach, we directly fit the Siler-Gamma hazard to life table data and obtained parameter estimates by minimizing the sum of the squared deviations of model-predicted and observed log survival probabilities. We compared several nonlinear and maximum-likelihood optimization methods (gradient-type and Newton-type algorithms) available through the **optimx** package in R (Nash and Varadhan 2011) to ensure that the parameter estimates are reliable. The **optimx** results allow the comparison of the performance of different algorithms in terms of objective function values, computational effort, and the quality of solution.The parameter estimates from the nonlinear least squares procedure were superior to maximum likelihood and other model-fitting procedures, yielding smaller standard errors for individual parameter estimates and a curve that more closely approximated the empirical hazard trajectory (results available upon request). We obtained robust standard errors for the parameter estimates using a sandwich variance estimator (White 1980; Zeileis 2006). These robust estimators provide a consistent estimate of the standard errors for parameters even when the underlying distribution of the errors is not Gaussian. We ran the optimization procedure independently for each of the cohort life tables, obtaining six simultaneous parameter estimates for each life table and examining the resulting trends.

Results: Frailty in Transition?

The frailty model and the methods detailed above allow an examination of the age-specific distributions of frailty over time and an investigation of the extent to which they have changed during the course of the mortality transition. Table 1 lists the parameter estimates and their standard errors for each birth cohort. There are clear declines in the three α parameters, reflecting mortality reductions at all ages. The trend in the negative β_1 parameter, representing the age-trajectory of mortality in childhood, increases over time (with the exception of declines in the first decade of the 20th century that are

overturned in the second decade), reflecting the known improvements in child survival. The β_2 trend, representing the age trajectory of adult mortality appears to increase in the latter part of the 19th century and then decline after the turn of the 20th century, perhaps balancing out a similar inflection in the α_2 parameter that co-determines the pattern of adult mortality. Finally, σ_0^2 initially shows no particular trend for cohorts born in the 19th century, but then displays a steady decline in value for subsequent cohorts, consistent with an overall decrease in mortality variability over time. (Figure A1 in the appendix presents these trends graphically.)

[Table 1 about here]

Next, figure 2 presents trends in the mean $\bar{z}(x)$ and variance $\sigma_2(x)$ of the frailty distribution across both time and age. Due to their reliance on the cumulative hazard of mortality in the cohort, both trajectories decline monotonically with age, resembling the well-known survival curve. These trajectories reflect the reduction in the mean and variance of frailty within each cohort as frail individuals drop out of the cohort. In the top panel, we can observe a rectangularization in the mean frailty trajectory over time. For the 1885 cohort, mean frailty declined relatively fast during the first years of life, due to the selection imposed by high rates of infant mortality. It then continued to decline at a slow but steady pace until members of the cohort reached age 70, after which the pace of decline was much accelerated. By the time the last members of the cohort died, mean frailty had declined to a level below 0.2. from its 1.0 start point. The reduced force of selective mortality over time is indicated by the fact that subsequent cohorts' trajectories are both higher (reflecting increased mean frailty at every age) and shorter (reflecting reduced selection up to and at every age) than their earlier (older) counterparts. Because frailty is calibrated to start with a mean of 1 for every cohort, the later cohorts – which experienced less mortality across the lifespan – were slightly more frail on average at each age than their earlier counterparts.

[Figure 2 about here]

The variance trajectories, in the lower panel of figure 2 also show a pattern of decline with age (as predicted by the theory of heterogeneity), but unlike the trajectories of means, they decline, rather than increase, over time. All cohorts after 1885 (once σ_0^2 began its decline) show an age trajectory whose initial values decrease, mirroring the reduction in variation for the complete mortality distribution. The age trajectory of decline – defined and almost rectangular for the earlier cohorts – becomes increasingly flat over time, with a less noticeable difference in the variability of the frailty distribution between young and older members of a given cohort. The crossing of the cohort variance trajectories at older ages suggests that the variation in the frailty distribution is not declining at the same pace across cohorts. Furthermore, at older ages, the slower pace of change may even create an apparent increase in the frailty variance over time. The crossover in the variance trajectories is also consistent with the idea that at the oldest ages, earlier cohorts were more selected for robustness than more recent cohorts.

For a fuller visualization of frailty changes during mortality transitions, figure 3 displays the frailty distribution within four cohorts – those born in 1885, 1900, 1910, and 1919 – from age 0, through ages 45 and 65, and up to 85. Note that each frailty distribution curve is normalized (Manton et al. 1981) to a probability mass equal to each cohort's survivorship proportion at age x, $l_c(x)$. Within individual cohorts, the effect of mortality selection is seen in the contraction of the frailty distribution with age, as selective mortality removes the frailest from the population and leaves a more homogeneous group of robust survivors. Notably, the shape of the distribution changes across cohorts, becoming more concentrated for later cohorts - in line with the idea that the full cohorts are becoming more homogeneous in their decreasing mortality risks over time. Furthermore, the age pattern of contraction has changed across cohort – while the 1885 cohort displays a relatively big gap between the distribution at age 0 and at age 45 (since the

frailest members of the cohort were removed at younger ages and only those persons with very low frailty survived), this gap, as well as those between successive ages, is smaller for later cohorts. (Figure A2 in the appendix shows the implications of cohort changes in frailty for age-specific trends across periods, and particularly for the pattern of increasing mortality variation at older ages.)

[Figure 3 about here]

Results from this exploratory analysis support the idea that the distributions of frailty – characterizing unobserved heterogeneity in the population – have changed in systematic ways during the course of the mortality transition, at least for Swedish females. Lower rates of overall mortality are resulting in cohorts that are more homogeneous with respect to the timing of mortality, as reflected by the striking decline in σ_0^2 values. However, as these more homogeneous cohorts age, mortality removes relatively fewer individuals at younger ages, leading to an increasingly less-selected older population. This reduced selection potentially manifests in the observed increase in mortality variability at older ages.

Discussion

Mortality and Frailty in Aging Populations

Combining observations about historical mortality transitions with concepts of selection and heterogeneity gives rise to the expectation that, in cohorts experiencing improved survival at younger ages, delayed mortality selection will produce a frailty distribution with a growing proportion of relatively vulnerable individuals. The arrival of a less selected group of survivors to older ages may be shaping an older population whose health profile is more diverse relative to earlier populations at the same age. This growing diversity may, in turn, give rise to the the observed trends of growing mortality variability

in later life as mortality disparities that were previously manifested in early and mid life are pushed into increasingly older ages. While this hypothesis cannot be tested directly with the present data, the findings above do suggest that as mortality declines transform the structure of populations, they also alter the composition of survivors to older ages. Trends in the Siler-Gamma parameter values reflect reductions in overall mortality over time, as well as specific improvements in early-childhood as well as adult survival. The decline in the model's frailty parameter further suggests that the level of unobserved heterogeneity in the population as a whole declined for cohorts born in the early 20th century, consistent with improvements to survival that lowered mortality rates and raised life expectancy for these cohorts over the course of the demographic and epidemiologic transitions.

As reaching advanced ages became increasingly common, the demographic frailty distribution reflected increasing heterogeneity in the older population, with successive cohorts being characterized by higher mean frailty at all ages and a slower pattern of contraction in the frailty distribution, even as overall variation in frailty declines. The crossover in the variance trajectories at the oldest ages suggest that due to higher early-life mortality, earlier cohorts were more selected for robustness than more recent cohorts. This suggests that population trends in mortality rates reflect countervailing forces of survival improvement at every age and co-occurring reductions in the force of selective mortality. The changing composition of the older population may thus create a dynamic whereby the observed trends at older ages in fact underestimate the amount of progress in survival over time. Indeed, improvements in early-life survival appear to lead to increasing heterogeneity at older ages. Because of the relatively short time frame covered by the available cohort life tables, the changes associated with the demographic transition are seen at their incipient stages, but the growing variation in mortality for survivors to older ages likewise hints at this dynamic.

Frailty models that account for changes in population composition over time capture both the temporal trend and age-pattern of mortality variability, and call attention to the future population health implications of historical demographic changes. The interpretation of the results are, however, complicated by some of the same assumptions that facilitate the estimation of the heterogeneity parameters. We have assumed parametric forms for both the individual hazard function (represented by the Siler model) and the allotment of frailty in the population (the gamma distribution). Previous work (Vaupel 1979; Heckman and Singer 1984) had proposed models that allow one of these two components to remain non-parametric, but it has not proved possible for both the hazard and heterogeneity distributions to be specified in a nonparametric form. The Siler model was appealing here because its additive form covered the hazard of mortality across the full age spectrum and its components are correlated with changing epidemiological profiles (Gage 1991). The gamma distribution's mathematical properties made the frailty model analytically tractable, though the strong assumptions built into the gamma may have unduly influenced the results we obtained. While the small standard errors inspire some confidence in the parameter estimates, the interpretation of individual parameter trends should be approached with caution as there is considerable interdependence among the parameter pairs describing childhood and adult mortality.

Cohorts which live through demographic and epidemiological transitions experience less mortality selection – but they are also likely to exhibit improved health – complicating the interpretation of the observed change in frailty. A related limitation is that the gamma distribution's ability to take on several shapes does not however, include an option that allows for an increase in the left (more robust) tail, as might be expected in populations experiencing reduced mortality and improved health. However, this is only a serious concern in populations with a substantial proportion of individuals in the zero frailty (or complete robustness) range. Furthermore, because mortality has been

declining during the period of analysis, the assumption that all cohorts are born with a frailty distribution centered around an average of 1 produces a situation where the same level of frailty implies different mortality risks across time. This imposed recalibration causes frailty to become a relative (rather than absolute) measure of vulnerability relative to a changing population average over time, and thus the meaning of a given frailty level also changes as overall mortality declines. A number of less restrictive alternatives to the gamma distribution have been suggested (see Hougaard 1995 for a review and discussion of stable, inverse Gaussian, and power variance exponential distributions), but it is not clear which offers the most sound option for modeling frailty and capturing the relevant sources of heterogeneity.

Conclusion: Frailty in Transition

Notably, the goal of this analysis was not to establish the superior appropriateness or fit of a particular frailty model, but rather to use this model as a descriptive tool for exploring a hypothesis about changes in unobserved heterogeneity in populations. Statistical distributions and mathematical functions are designed to provide an empirically fitting – rather than biologically coherent or etiologically meaningful – model of the population process of interest. And yet, as has been repeatedly shown, models like the Gompertz and Siler and their counterparts describe the age pattern associated with many individual causes of death as well as all-cause mortality. Wood et al. (1992) have noted that this apparent commonality in hazard trajectories despite differences in the physiological mechanisms that cause them may suggest that all age-related pathologies are, in some way, multi-stage or multi-hit processes. This idea is closely related to the notion of multisystem physiological dysregulation that is thought to be a cause of *geriatric* frailty, a clinical measure of individuals' changing vulnerability to stressors and adverse health events in later life (Fried et al. 2009). While the preceding analysis focused on aggregate

mortality and demographic frailty, the geriatric notion of frailty is helpful in considering the potential implications of our results for trends in population health. In particular, as improved survival in early life increases the proportion of *demographically* frail persons who reach older ages, these compositional differences may manifest in differential patterns of later-life health, including trajectories of morbidity, disability, or geriatric frailty.

Aggregate patterns of mortality arise both from population dynamics and from characteristics of the individual life course. While a cursory consideration of health transition may yield an expectation for concurrent improvements in population survival and health, connecting trends in mortality to changes in morbidity and disability is not straightforward. As people live longer, there is more time for chronic diseases and other health problems to manifest, and there is growing recognition that health in later life is a function of factors operating throughout the lifespan starting as far back as the in-utero environment (Barker 1995). The susceptibilities that underlie aggregate mortality trends are thus linked to fundamental processes of growth, development, and senescence, while individual health trajectories are also shaped by aggregate conditions.

Debates about whether adults today are reaching old age with more or less morbidity and disability than in the past are ongoing (Parker and Thorslund 2007; Martin et al. 2010), and analyses of the trajectories of clinical frailty across cohorts and populations will provide much-needed insight into both physiological and socioeconomic determinants of vulnerability. With fewer deaths taking place in early and mid-life, variation in later-life mortality may increasingly reflect differences in people's underlying (or static) endowment, in their accumulated lifetime experiences, and/or in their later-life health dynamics. Combining the two complementary concepts of demographic and geriatric frailty may eventually yield an analytic framework that fosters both accurate statistical estimation and a realistic modeling of health trajectories and mortality processes across diverse sub-populations. While demographic frailty accounts for improved survival and

changing population composition across time, further research into clinical frailty could offer insights into how differential vulnerabilities may be manifesting in later life health, rather than earlier mortality. While clinical frailty is often positioned as a predictor of mortality outcomes in the geriatric literature (e.g. Mitnitski et al. 2002, Bergman et al. 2007), our analysis reversed this intuition and explored the relationship between increased survival and the manifestations of demographic frailty in population mortality patterns. Additional research on the dynamic trajectories of clinical frailty may provide further clarification about the link between vulnerability to mortality and differential patterns of health and functional decline at the end of life.

Demographers traditionally worked with aggregate population data, and the classic statistical frailty models were developed to indirectly characterize the presence of hidden or unmeasured heterogeneity in the aggregate, offering no specific way to associate these unobserved differences with the frailty of individuals. And yet, the historical changes that have curbed the force of selective mortality and reshaped the parameters of population health also have implications for individual trajectories of health. As longevity increases and the number of older people rises, the health of survivors both on average and in terms of its variability may differ from the health of those who survived to old age in the past. This analysis suggests that the growing population of survivors to older ages may increasingly encompass both robust individuals and those at various degrees of frailty. Since heterogeneity in mortality among the old has increased concomitantly with survival improvements throughout the lifespan, it is possible that delayed mortality selection has pushed back health disparities from early life to increasingly older ages. Mortality analysis has shown that this delayed selection may be manifested in the growing variation in longevity. The implications of these historical changes for the health and well-being of contemporary older adults and future aging populations await further elucidation.

References

[1] Arking, R. (2006). *The Biology of Aging.* Third edition. New York: Oxford University Press.

[2] Bandeen-Roche, K., & Liang, K.Y. (1996). Modeling failure-time associations in data with multiple levels of clustering. *Biometrika* 83(1):29-39.

[3] Barker, D.J.P. (1995). Fetal origins of coronary heart disease. *BMJ* 311:171-174.

[4] Bergman, H., Ferrucci, L., Guralnik, J., Hogan, D. B., Hummel, S., Karunananthan, S., & Wolfson, C. (2007). Frailty: An emerging research and clinical paradigm – Issues and controversies. *Journals of Gerontology: Series A, Biological Sciences & Medical Sciences* 62(7): 731737.

[5] Butt, Z. & Haberman, S. (2004). Application of frailty-based mortality models using generalized linear models. *Astin Bulletin* 34(1):175-197.

[6] Canudas-Romo, V. (2008). The modal age at death and the shifting mortality hypothesis. *Demographic Research* 19(30): 1179-1204.

[7] Cowgill, D.O. & Holmes, L.D., Eds. (1972). *Aging and Modernization.* New York: Appleton-Century-Crofts.

[8] Dublin, L.I. (1923). The possibility of extending human life. *Metron* 3(2): 175-197.

[9] Edwards, R., & Tuljapurkar, S. (2005). Inequality in life spans and a new perspective on mortality convergence across industrialized countries. *Population and Development Review* 31(4):645-675.

[10] Engelman, M., Canudas-Romo, V., & Agree, E.M. (2010). The influence of increased survivorship on mortality variation among aging populations. *Population and Development Review* 36(3): 511-539.

[11] Finch, C.E., & Crimmins, E.M. (2004). Inflammatory Exposure and Historical Changes in Human Life-Spans. *Science* 305(5691): 1736-1739.

[12] Fried, L.P., Xue, Q.L., Cappola, A.R., Ferrucci, L., Chaves, P., Varadhan, R., Guralnik, J.M., Leng, S.X., Semba, R.D., Walston, J.D., Blaum, C.S., Bandeen-Roche, K. 2009. Nonlinear multisystem physiological dysregulation associated with frailty in older women: implications for etiology and treatment. *Journals of Gerontology: Medical Sciences* 64(10):1049-57.

[13] Fries J.F. (1980). Aging, natural death, and the compression of morbidity. *NEJM* 303: 130-135.

[14] Gage, T.B. (1989). Bio-mathematical approaches to the study of human variation in mortality. *Yearbook of Physical Anthropology* 32:185-214.

[15] Gage, T.B. (1991). Causes of death and the components of mortality: Testing the biological interpretation of a competing hazards model. *American Journal of Human Biology* 3:289-300.

[16] Gage, T.B., & Dyke, B. (1986). Parameterizing abridged mortality tables: The Siler three-component hazard model. *Human Biology* 58:275-291.

[17] Gage, T.B., & Mode, C.J. (1993). Some laws of mortality: How well do they fit? *Human Biology* 65:445-461.

[18] Goldstein, J.R., & Wachter, K.W. (2006). Relationships between period and cohort life expectancy: Gaps and lags. *Population Studies* 60(3): 257-269.

[19] Gompertz, B. (1825). On the nature of the function expressive of the law of human mortality and on a new mode of determining the value of life contingencies. *Philosophical Transactions of the Royal Society of London B Biological Sciences* 115: 513-585.

[20] Heckman, J. & Singer, B. (1984). The identifiability of the proportional hazards model. *Review of Economic Studies* 51:231-241.

[21] Hermalin, A.I. Ed. (2002). *The Well-Being of the Elderly in Asia: A Four-Country Comparative Study.* Ann Arbor, MI: University of Michigan Press.

[22] Hougaard, P. (1995). Frailty models for survival data. *Lifetime Data Analysis* 1: 255-273.

[23] Human Mortality Database. (2012). University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at www.mortality.org or www.humanmortality.de (data downloaded on 1/4/2012).

[24] Li, T. & Anderson, J.J. (2009). The vitality model: A way to understand population survival and demographic heterogeneity. *Theoretical Population Biology* 76:118-131.

[25] Livingston, J. (2003). Reconfiguring old age: Elderly women and concerns over care in southeastern Botswana. *Medical Anthropology* 22(3):205-231.

[26] Manton, K.G., Stallard, E. & Vaupel J.W. (1981). Methods for comparing the mortality experience of heterogeneous populations. *Demography* 18(3):389-410.

[27] Manton, K.G., Stallard E. & Vaupel, J.W. (1986). Alternative models for the heterogeneity of mortality risks among the aged. *Journal of the American Statistical Association* 81(395):635-644.

[28] Martin, L.G., Freedman, V.A., Schoeni, R.F., & Andreski, P. (2010). Trends in disability and related chronic conditions among people ages fifty to sixty-four. *Health Affairs* 29(4):725-31.

[29] Missov, T.I. & Finkelstein, M. (2011). Admissible mixing distributions for a general class of mixture survival models with known asymptotics. *Theoretical Population Biology* 80:6470.

[30] Mitnitski, A. B., Mogilner, A. J., MacKnight, C., & Rockwood, K. (2002). The mortality rate as a function of accumulated deficits in a frailty index. *Mechanisms of Ageing and Development* 123(11): 1457-1460.

[31] Nash, J.C., & Varadhan, R. (2011). Unifying Optimization Algorithms to Aid Software System Users: optimx for R. *Journal of Statistical Software* 43(9), 1-14.

[32] Parker, M.G. and M. Thorslund. 2007. Health trends in the elderly population: Getting better and getting worse. *The Gerontologist* 47(2): 150-158.

[33] Robine, J.M., Crimmins, E., Horiuchi, S., & Zeng, Y. (Eds). (2006). *Human Longevity, Individual Life Duration, and the Growth of the Oldest-Old Population Hu- man Longevity, Individual Life Duration, and the Growth of the Oldest-Old Population.* Springer.

[34] Schroeder, S.A. (2007). Shattuck Lecture. We can do better – improving the health of the American people. *New England Journal of Medicine* 357(12):1221-8.

[35] Siler, W. (1979). A competing risk model for animal mortality. *Ecology* 60(4):750-757.

[36] Simmons, L.W. (1945). *The role of the aged in primitive society.* London: Oxford University Press.

[37] Thatcher, A.R., Kannisto, V., & Vaupel, J.W. (1998). *The Force of Mortality at Ages 80 to 120.* In Monographs on Population Aging No. 5. Odense, Denmark: Odense University Press. (Available online at: http://www.demogr.mpg.de/Papers/Books/Monograph5/start.htm)

[38] Treas, J., & Logue, B. (1986). Economic development and the older population. *Population and Development Review* 12(4):645-673.

[39] Tuljapurkar, S., & Edwards, R. (2009). Variance in death and its implications for modeling and forecasting mortality. NBER Working Paper No. 15288.

[40] Vaupel, J.W. (1997). Trajectories of mortality at advanced ages. In K.W. Wachter and C.E. Finch, Eds. *Between Zeus and the Salmon: The Biodemography of Longevity.* Washington, DC: National Academy Press, 17-37.

[41] Vaupel, J.W., Manton, K.G., & Stallard, E. (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography* 16(3): 439-454.

[42] Vaupel, J.W., Yashin, A.I., & Manton, K.G. (1988). Debilitations aftermath: stochastic process models of mortality. *Mathematical Population Studies* 1(1): 21-48.

[43] Vaupel, J.W. & Yashin, A. I. (1985). Heterogeneitys ruses: Some surprising effects of selection on population dynamics. *The American Statistician* 39(3); 176-185.

[44] Vaupel, J.W., & Yashin, A.I. (2006). Unobserved population heterogeneity. In: Caselli G, Vallin J and Wunsch G, eds. *Demography: Analysis and Synthesis. A Treatise in Population Studies* Volume 1. Elsevier, p. 271-278.

[45] White, H. (1980). A heteroskedasticity-consistent covariance matrix estimator and a direct test for heteroskedasticity. *Econometrica* 48: 817830.

[46] Wilmoth, J.R., & Horiuchi, S. (1999). Rectangularization Revisited: Variability of age at death within human populations. *Demography* 36(4):475-495.

[47] Wilmoth, J.R., Deegan, L. J., Lundstrm, H.,& Horiuchi, S. (2001). Increase in maximum life span in Sweden, 1861-1999. *Science* 289:2366-2368.

[48] Wood, J.W., Holman, D.J., Weiss, K.M., Buchanan, A.V., & LeFor, B. (1992). Hazard models for human population biology. *Yearbook of Physical Anthropology* 35:43-87.

[49] Yashin, A.I. & Iachine, I.A. (1997). How frailty models can be used for evaluating longevity limits: Taking advantage of an interdisciplinary approach. *Demography* 34(1):31-48.

[50] Yashin, A.I., Vaupel, J.W., & Iachine, I.A. (1994). A duality in aging: the equivalence of mortality models based on radically different concepts. *Mechanisms of Ageing and Development* 74:1-14.

[51] Yashin, A.I., Arbeev, K.G., Akushevich, I., Kulminski, A., Akushevich, L., & Ukraintseva, S.V. (2008). Model of hidden heterogeneity in longitudinal data. *Theoretical Population Biology* 73:1-10.

[52] Zeleis, A. (2006). Object-oriented computation of sandwich estimators. *Journal of Statistical Software* 16(9): 1-16.

Table 1. Parameter estimates and standard errors for the Siler-gamma model. Based on cohort life tables for Swedish females born 1885-1916.

Cohort	α_1	(se)	β_1	(se)	α_2	(se)	β_2	(se)	α_3	(se)	σ^2	(se)
1885	-2.62	(0.24)	0.66	(0.09)	-12.01	(0.34)	0.12	(5.62E-03)	-5.26	(0.05)	0.18	(0.06)
1886	-2.71	(0.26)	0.61	(0.08)	-11.64	(0.26)	0.11	(4.08E-03)	-5.31	(0.04)	0.10	(0.04)
1887	-2.75	(0.25)	0.60	(0.07)	-11.53	(0.25)	0.11	(3.75E-03)	-5.33	(0.04)	0.08	(0.03)
1888	-2.67	(0.21)	0.64	(0.07)	-12.37	(0.32)	0.13	(5.26E-03)	-5.30	(0.05)	0.22	(0.06)
1889	-2.60	(0.19)	0.70	(0.07)	-11.89	(0.24)	0.12	(3.85E-03)	-5.34	(0.04)	0.10	(0.04)
1890	-2.60	(0.19)	0.72	(0.07)	-11.60	(0.24)	0.11	(3.67E-03)	-5.40	(0.05)	0.05	(0.03)
1891	-2.62	(0.21)	0.75	(0.08)	-12.44	(0.31)	0.13	(4.95E-03)	-5.35	(0.05)	0.17	(0.05)
1892	-2.65	(0.24)	0.80	(0.11)	-12.16	(0.29)	0.12	(4.42E-03)	-5.40	(0.05)	0.08	(0.04)
1893	-2.71	(0.27)	0.80	(0.14)	-12.25	(0.32)	0.12	(4.93E-03)	-5.43	(0.05)	0.11	(0.05)
1894	-2.77	(0.28)	0.75	(0.13)	-12.10	(0.26)	0.12	(3.76E-03)	-5.46	(0.05)	0.06	(0.03)
1895	-2.74	(0.24)	0.75	(0.12)	-12.52	(0.33)	0.13	(5.19E-03)	-5.46	(0.06)	0.17	(0.06)
1896	-2.73	(0.26)	0.75	(0.11)	-12.16	(0.28)	0.12	(4.25E-03)	-5.52	(0.05)	0.06	(0.04)
1897	-2.71	(0.20)	0.75	(0.08)	-12.03	(0.26)	0.12	(3.86E-03)	-5.57	(0.06)	0.05	(0.03)
1898	-2.59	(0.14)	0.86	(0.07)	-12.72	(0.30)	0.13	(4.44E-03)	-5.53	(0.05)	0.15	(0.04)
1899	-2.56	(0.19)	0.90	(0.09)	-12.97	(0.31)	0.13	(4.67E-03)	-5.56	(0.05)	0.16	(0.05)
1900	-2.61	(0.21)	0.93	(0.14)	-12.86	(0.28)	0.13	(4.24E-03)	-5.60	(0.05)	0.16	(0.04)
1901	-2.64	(0.17)	0.95	(0.11)	-12.82	(0.27)	0.13	(3.95E-03)	-5.64	(0.05)	0.12	(0.04)
1902	-2.68	(0.14)	1.02	(0.08)	-12.94	(0.26)	0.13	(3.78E-03)	-5.68	(0.05)	0.14	(0.04)
1903	-2.69	(0.13)	1.10	(0.08)	-12.76	(0.24)	0.13	(3.58E-03)	-5.72	(0.04)	0.12	(0.04)
1904	-2.72	(0.18)	1.12	(0.16)	-12.97	(0.23)	0.13	(3.35E-03)	-5.75	(0.04)	0.12	(0.04)
1905	-2.80	(0.20)	1.09	(0.14)	-12.99	(0.23)	0.13	(3.41E-03)	-5.77	(0.04)	0.13	(0.04)
1906	-2.84	(0.20)	1.03	(0.13)	-13.04	(0.22)	0.13	(3.31E-03)	-5.81	(0.04)	0.13	(0.04)
1907	-2.87	(0.20)	1.06	(0.18)	-13.02	(0.22)	0.13	(3.25E-03)	-5.85	(0.04)	0.12	(0.04)
1908	-2.85	(0.27)	1.09	(0.20)	-13.08	(0.22)	0.13	(3.31E-03)	-5.88	(0.04)	0.13	(0.04)

Cohort	α_1	(se)	β_1	(se)	α_2	(se)	β_2	(se)	α_3	(se)	σ^2	(se)
1909	-2.94	(0.16)	1.03	(0.11)	-12.94	(0.22)	0.13	(3.26E-03)	-5.92	(0.04)	0.12	(0.03)
1910	-3.05	(0.32)	0.90	(0.20)	-13.06	(0.23)	0.13	(3.38E-03)	-5.97	(0.05)	0.12	(0.03)
1911	-3.16	(0.39)	0.86	(0.28)	-13.09	(0.23)	0.13	(3.40E-03)	-5.99	(0.05)	0.12	(0.04)
1912	-3.32	(0.46)	0.70	(0.25)	-12.88	(0.24)	0.13	(3.35E-03)	-6.08	(0.06)	0.10	(0.03)
1913	-3.36	(0.38)	0.63	(0.16)	-12.83	(0.23)	0.12	(3.25E-03)	-6.15	(0.06)	0.10	(0.03)
1914	-3.22	(0.33)	0.65	(0.12)	-12.73	(0.20)	0.12	(2.93E-03)	-6.23	(0.05)	0.10	(0.03)
1915	-3.12	(0.26)	0.72	(0.09)	-12.67	(0.20)	0.12	(2.88E-03)	-6.28	(0.05)	0.09	(0.03)
1916	-3.11	(0.29)	0.80	(0.08)	-12.67	(0.20)	0.12	(2.84E-03)	-6.34	(0.04)	0.09	(0.03)
1917	-2.98	(0.05)	0.92	(0.05)	-12.56	(0.18)	0.12	(2.64E-03)	-6.40	(0.04)	0.08	(0.03)
1918	-2.98	(0.21)	1.11	(0.12)	-12.49	(0.18)	0.12	(2.57E-03)	-6.44	(0.04)	0.08	(0.03)
1919	-3.06	(0.21)	1.22	(0.15)	-12.52	(0.19)	0.12	(2.68E-03)	-6.47	(0.05)	0.07	(0.03)

Siler-Gamma parameters were estimated from life tables using nonlinear least square minimization. Robust standard errors were calculated using a sandwich variance estimator.

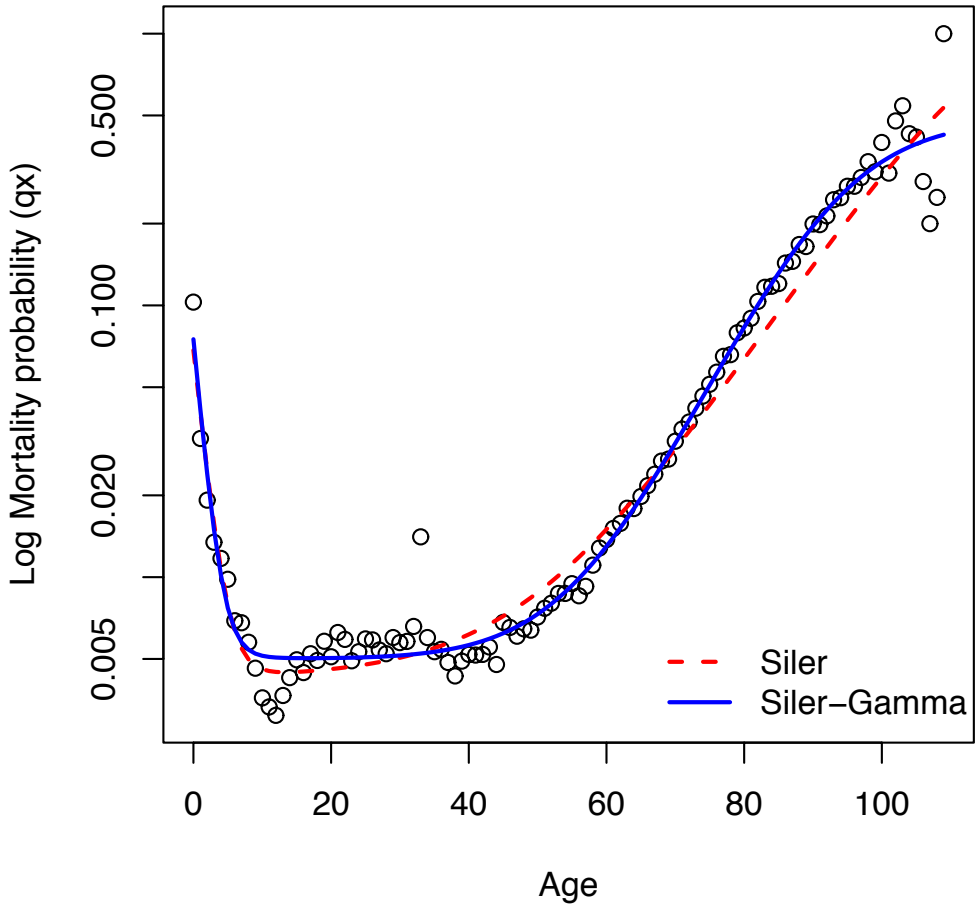

Figure 1: **Mortality trajectories for Swedish females: Empirical data for the cohort born in 1885 and trajectories based on the Siler and Siler-gamma models.** Siler model trajectory created with parameter values: $\alpha_1 = -2.71$, $\beta_1 = 0.58$, $\alpha_2 = -9.73$, $\beta_2 = 0.09$, and $\alpha_3 = -5.46$. Siler-gamma model trajectory created with parameter values: $\alpha_1 = -2.62$, $\beta_1 = 0.66$, $\alpha_2 = -12.01$, $\beta_2 = 0.12$, $\alpha_3 = -5.26$, and $\sigma_0^2 = 0.18$.

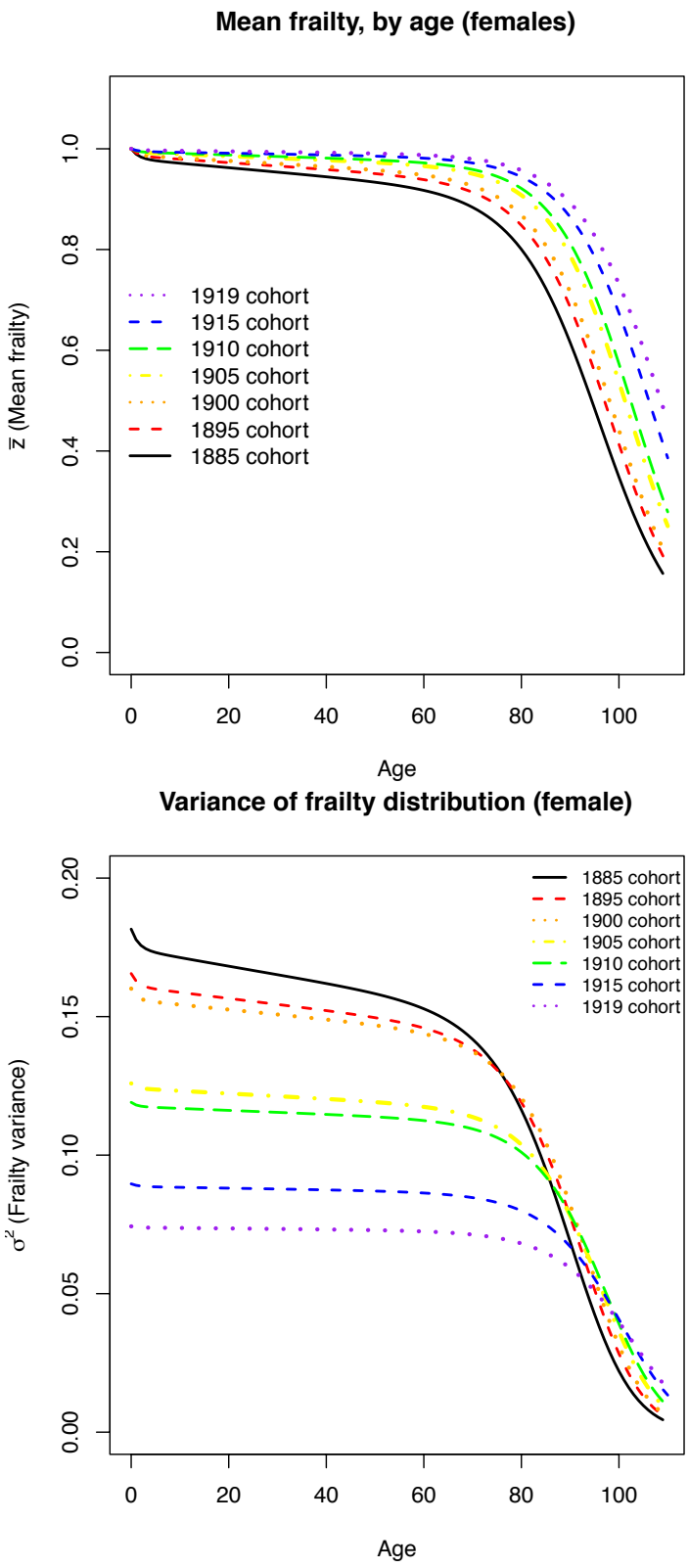

Figure 2: **Change across cohorts in the age-trajectory of the frailty distribution's mean (top) and variance (bottom).** Calculated from cohort life tables for Swedish females born 1885-1919.

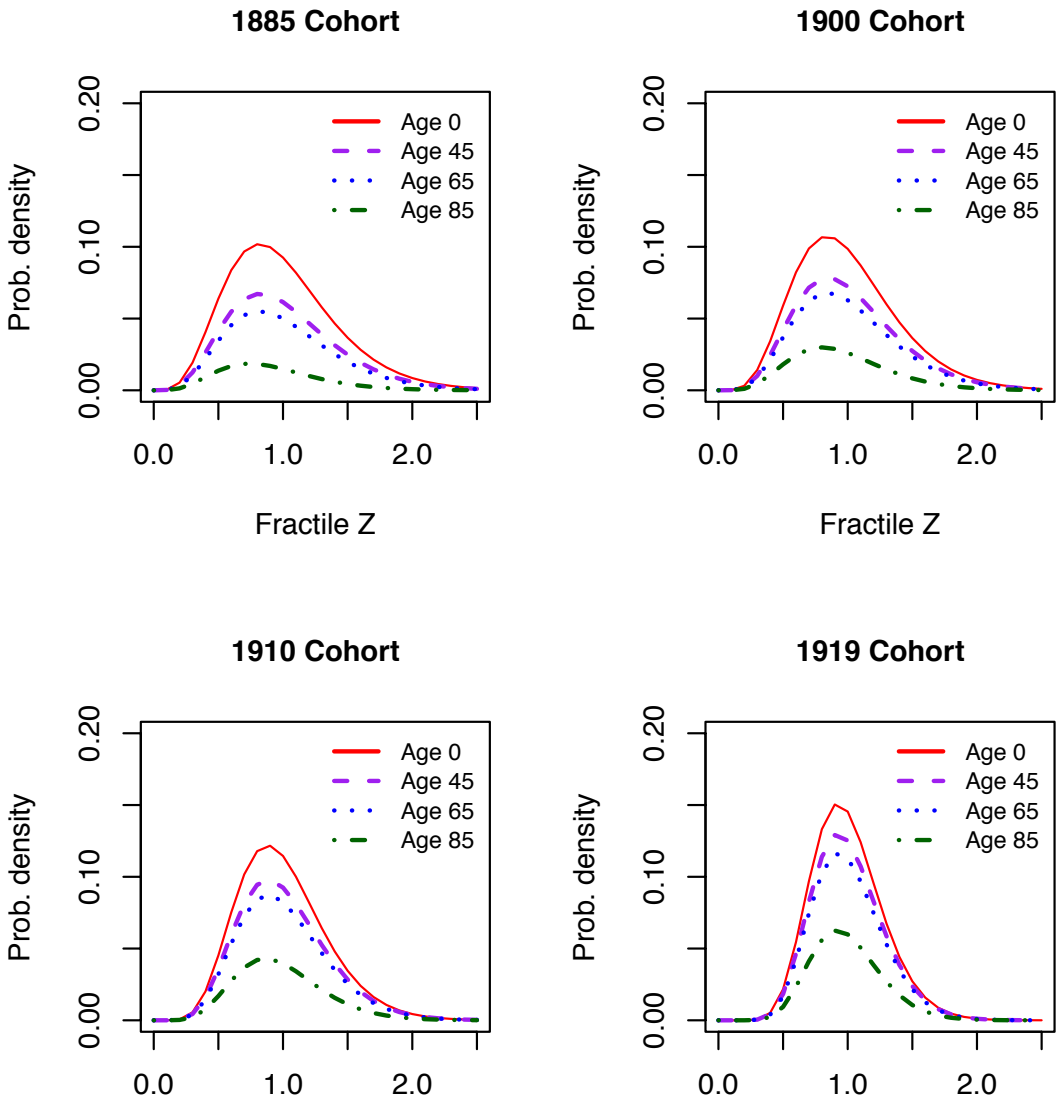

Figure 3: **The age-pattern of frailty contraction within cohorts: Swedish females born 1885-1919.** Each frailty distribution curve is normalized to a probability mass equal to the cohort's survivorship proportion at age x, $l_c(x)$.

Appendix A

The two figures in the appendix elaborate on the trends in the Siler-gamma model parameter values and their implications for age-specific distributions of frailty in successive cohorts.

Figure A1 presents trends in the values of the six Siler-gamma model parameters, estimated from life tables for cohorts of Swedish females born between 1885-1919. Each parameter is shown in its own scale to facilitate an analysis of the trends over time.

Figure A2 highlights the implications of cohort changes in frailty for age-specific trends across periods. While mean frailty is constrained to remain at $\bar{z}(0) = 1$, the plot for the four birth cohorts (age 0) nonetheless displays a modal shift upwards and to the right, reflecting the decline in frailty variance (σ_0^2) as the cohort overall becomes more robust. The plots for survivors to ages 45, 60, and 85 likewise indicate that the mode of the frailty distribution has shifted to the right over time, a result linked to both the changing variance and slightly higher mean frailty at every age. While the distributions are more concentrated around this mode for later cohorts than earlier ones, the countervailing effect of increased survival by individuals with slightly higher frailty values creates an impression of slight expansion in the distribution at older ages.

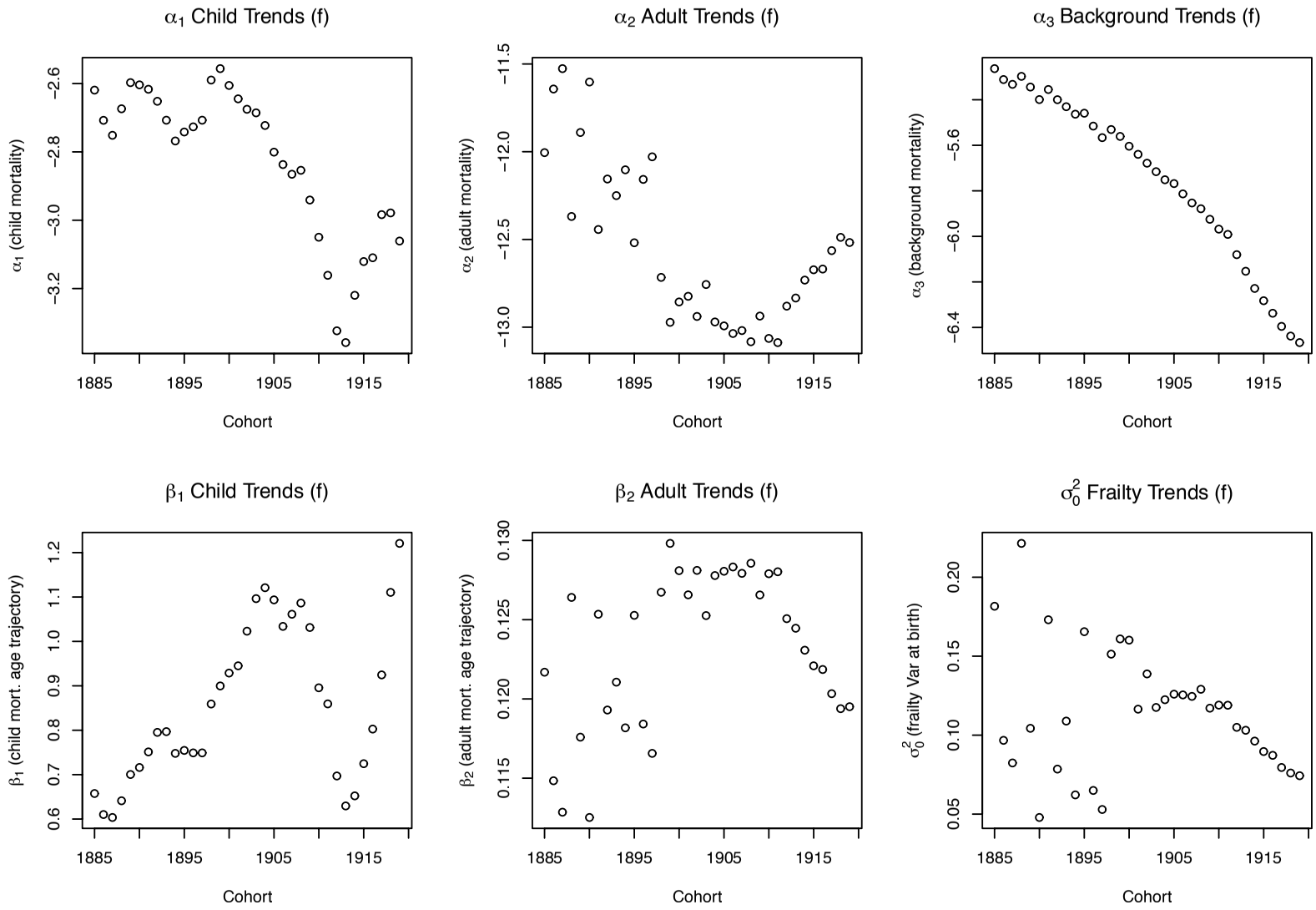

Figure A1. Cohort trends in parameter estimates for the Siler-Gamma model. Note that the child mortality parameter β_1 is negative in the Siler-gamma equation, while all other parameters are positive. Based on cohort life tables for Swedish females born 1885-1919.

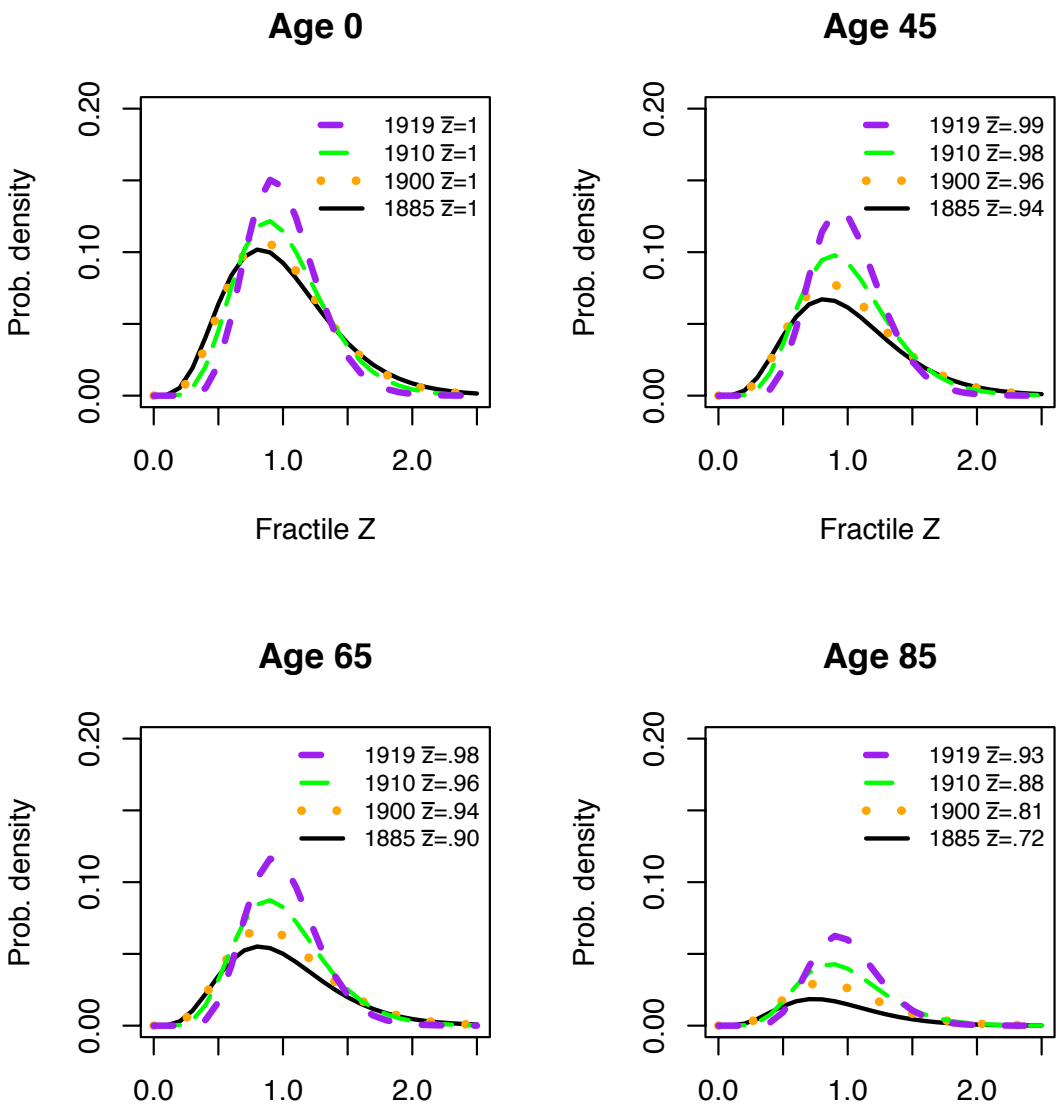

Figure A2. Changing distribution of age-specific frailty across cohorts: Swedish females born 1885-1919 at four ages. Each frailty distribution curve is normalized to a probability mass equal to the cohort's survivorship proportion at age x, $l_c(x)$.

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