Can Frailty Models Explain Mortality Differentials by Socioeconomic Status?

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Abstract

In this paper we investigate whether a selection hypothesis provides a consistent and plausible explanation for the observed convergence and cross-over of mortality trajectories by sex and socioeconomic status (education) at old ages. In this context, the analyses compare a parametric with a non-parametric approach and are based on a newly proposed modified DeMoivre hazard function and a covariate-identified frailty model. Both approaches allow us to model and investigate the effect of heterogeneity in a broad age range comprising adult working up to old and oldest-old ages. The analyses are based on the British Longitudinal Study that covers the period 1971-1996, and follows up the mortality of cohorts that are 50–70 year old in 1971. Our analyses indicate that the patterns of mortality convergence between socioeconomic groups may be primarily attributed to changes in the frailty composition of these socioeconomic groups, instead of fundamental differences in the process of aging itself or variation in the effect of socioeconomic risks factors over the individuals' life course. Our analyses suggest also that the individual risk of death may arise faster than exponential.

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1 Introduction

Variations in mortality by age, sex, race/ethnicity and socioeconomic groups have been
documented systematically in all countries on the basis of individual- or aggregate-level
data (House et al. 1990; Feinstein 1993; Preston and Taubman 1994; Mackenbach and
Kunst 1995; Hummer et al. 1998; Liu et al. 1998; Leon 2001; Kohler 2001). In particu-
lar, two patterns of mortality differentials are of a considerable research interest: *different
levels* of mortality experienced by males and females, or by socioeconomic groups, and *dif-
ferential increases* of the mortality hazard with age observed by sex and various population
groups. While differences in the level of mortality by age group, sex and socioeconomic
group are very well documented for many countries (see for example Valkonen and Martelin
1988; Valkonen 1987; Valkonen and Martikainen 1995; Doblhammer 1996; Hummer et al.
1996; Drever et al. 1996; Williams 1990), very few studies address explicitly the question
of differences in the increase of the mortality hazard with age (exceptions include Hori-
uchi and Coale 1990; Gavrilov and Gavrilova 1991; Carey and Liedo 1995; Horiuchi and
Wilmoth 1997; Kohler and Kohler 2000).

The prevailing focus in the epidemiological and demographic literature on differences
in the level of mortality is surprising since the two characteristics of mortality differentials–
level and increase by age–are closely associated with each other. For instance, it is well
documented that male mortality exceeds female mortality in many populations, and that
this female advantage in survival often diminishes with age. Male and female mortality
thus tend to converge at older age. Several epidemiological and demographic studies have
observed similar patterns of mortality change with age also between race/ethnicity, or
socioeconomic groups that are subject to quite different mortality levels at adult working

ages but not necessarily at older ages (Antonovsky 1967; Kitigawa and Hauser 1973; Pappas et al. 1993; Preston and Taubman 1994; Doblhammer 1996; Martelin et al. 1998; Liao et al. 1999; Liu et al. 1998; Kohler 2001). This observation, however, implies a different pattern of mortality increase with age and consequently a convergence or even a crossover of the mortality trajectories by socioeconomic group at old and oldest-old ages.

In summary, the existing studies convincingly suggest potential interactions between the level and increase of mortality across age, sex and socioeconomic groups. However, neither the nature nor the mechanisms of this relationship are fully understood. In particular, despite the ample evidence on existing sex and socioeconomic mortality differentials by age, most of the existing studies do not address the question of compositional changes that occur over time when different socioeconomic groups age. For instance, when a cohort of individuals declines due to mortality, this results in a change of its frailty composition. Cohorts with high initial mortality levels undergo a faster selection towards less frail individuals, and the survivors would tend to have low mortality levels. The observed age-specific mortality trajectories therefore reflect both, compositional changes that occur due to selection forces as well as individual-level changes that accompany the aging process of men and women, or different socioeconomic groups. If the empirical analyses do not distinguish between these two types of demographic change–individual-level versus population changes–then this may result in misleading conclusions about the effect and interplay of some individual-level characteristics with age.

In this paper we investigate the role of selective forces in the convergence of the mortality trajectories by sex and socioeconomic groups observed at old and oldest-old ages. The analyses are based on cohort data obtained from the British Longitudinal Study (LS) and cover the period 1971-1997. We examine with these data whether and how the effect of fixed socioeconomic characteristics, such as education, changes over the life span of individuals. In particular, a central concern in our paper is whether the observed convergence of socioeconomic differentials and possible crossover of the mortality trajectories by education at old and oldest-old ages may be explained primarily by a differential speed of selection operating among different educational groups. For example, educational groups experience substantially different levels of mortality at younger ages ranging from low

mortality for people with high education to high mortality for people with low educational attainment, and thus, are subject of differential selection forces over the life course of individuals.

Alternatively, the differential increase in the force of mortality per year of age may be explained by a differential aging process between males and females or socioeconomic groups rather than by a stronger selection process of the high mortality groups. In this context, we define the rate of aging by the life table aging rate (LAR) (Horiuchi and Coale 1990; Horiuchi and Wilmoth 1997). The LAR reflects the extent to which each additional year of age is detrimental for an individual through a variety of mechanisms that combined lead to an increase of mortality. This increase in the force of mortality per additional year of age can differ across sex and socioeconomic groups due to the instantaneous and cumulative effect of factors such as biological and genetic differences, differential knowledge and resources available for health care, persistent differences in health relevant behavior such as smoking or alcohol consumption, differential exposure to environmental hazards, and other determinants of health that vary systematically across sex and socioeconomic groups.

To understand the dynamics that underlie the convergence and possible crossovers of the mortality trajectories by sex and/or socioeconomic groups at older ages, the empirical analyses need to consider and incorporate the effect of heterogeneity already at adult working ages. This relatively early onset of a selection of the population towards low-frailty individuals is supported by recent evidence from twin studies, which suggest that unobserved heterogeneity and selection are important for the estimation of the mortality patterns already at younger ages, and not only at oldest-old ages (Caselli et al. 2000; Iachine et al. 1998). In addition, these and also some other studies suggest that human mortality does not increase with age at a constant rate through adult ages until very old ages, but the relative increase of the mortality hazard at younger ages is faster than exponential (Manton et al. 1995; Horiuchi and Wilmoth 1997; Kohler and Kohler 2000). However, in the context of frailty models this is problematic, because the selection process occurs at lower pace at younger ages. Thus, the application of relative frailty models to adult ages is hampered by the fact that the observed mortality patterns at working ages

lack the typical flattening of the mortality curve, which usually is visible when the level of mortality becomes moderately high and a strong selection process towards low-frailty individuals operates in the observed population.

In this paper, we implement new statistical methods that overcome the estimation problem of frailty models at younger (working) ages. In particular, the analyses compare a non-parametric (the covariate-identified frailty model) with a parametric approach (the modified DeMoivre hazard function proposed by Kohler and Kohler 2000). Both models allow the baseline hazard to increase faster than exponentially at adult ages. However, while the DeMoivre hazard function imposes a specific parametric form on the baseline hazard of mortality, the covariate-identified frailty model is estimated nonparametrically using a piecewise-constant proportional hazard estimation of the baseline mortality pattern.

The remainder of this paper is structured as follows. Section 2 presents and discusses the covariate-identified frailty model and the DeMoivre hazard function. In Section 3 we describe the British Longitudinal Study, on which our analyses are based. The following Section 4 discusses the selection hypothesis versus a differential aging process as an explanation of the convergence of mortality trajectories at older ages by education or socioeconomic status. The next Section 5 presents the results and compares in particular the estimates obtained from the DeMoivre versus piecewise-constant estimation of variance in frailty. Section 6 discusses the implication of frailty on the estimates of life expectancy, and focuses on the difference between individual versus population life expectancy. The last section 7 summarizes and discusses the results.

2 Methods: Parametric versus Nonparametric Approach for Frailty Modelling at Adult Ages

In the selection hypothesis, the relative effect of a fixed socioeconomic characteristic, such as education, on the mortality hazard is assumed to be constant over the life span and does not change with age (as indicated by many demographic and epidemiological studies). This hypothesis implies that mortality of individuals with the same relative

frailty is more or less proportional in different socioeconomic groups, while the observed mortality trajectories exhibit a pattern of convergence or crossovers that occurs primarily due to differential selection forces operating among these socioeconomic groups.

In this section we examine two mathematical models in order to better understand the impact of selection on basic relations between socioeconomic factors and mortality during the life span of the individuals. Because our focus is on hidden/unobserved heterogeneity, we restrict our attention to the simple case where the covariates are fixed (rather then changing over time).

In the first model, the covariate identified frailty model (CIFM), we make no specific assumption about the age-pattern of the mortality and we estimate a piecewise-constant baseline hazard. In the second model, we assume a specific parametric model of this baseline mortality hazard, the modified DeMoivre hazard, that allows the application of relative frailty models to mortality at adult and old ages.

The nonparametric model has the advantage that it does not restrict the age-pattern of the baseline hazard. Such an estimation of the baseline hazard can be viewed as being superior or advantageous to the parametric specification especially in large datasets that compensate for the loss of precision that is inevitable associated with nonparametric methods. However, the model is very time-consuming in the estimation and it does not yield a possibility to summarize the age-pattern of mortality with only a few key and easily interpreted parameters.

Our second estimation based on the modified DeMoivre hazard function overcomes this limitation; in particular, the age-pattern of mortality is summarized–quite similar as with the Gompertz model–in terms of a level parameter a and a slope parameter b. This hazard increases faster than exponential, and when combined with unobserved heterogeneity it can capture an observed population hazard that is approximated by a Gompertz or Logistic mortality function. Moreover, since a few parameters can be used to describe the individual- and population-level mortality hazard, the estimates from the model can easily be used to calculate standard life-table functions such as the life-expectancy.

Before we proceed with our empirical estimations, we briefly review the covariate identified frailty model and the modified DeMoivre hazard function in the following sections.

2.1   The Covariate-Identified Frailty Model (CIFM)

The basic assumption in the covariate-identified frailty model is that the mortality hazards by socioeconomic groups, conditional on frailty z, differ only by a factor of proportionality. In this case, it is possible to combine a Gamma-distributed relative frailty with a piecewise-constant hazard function $\mu^{PW}(x)$ that does not impose parametric restrictions on the shape of the baseline mortality pattern.

The covariate-identified proportional hazard model with unobserved relative frailty is specified as follows. Consider the age-intervals $(c_0, c_1], \ldots, (c_{j-1}, c_j], \ldots, (c_{K-1}, c_K]$ that separate the observed age range into K disjoint intervals. Then assume that the mortality hazard, conditional on a frailty $z = 1$ and the observed covariates y_a, is constant within each of these age intervals and equals $a(y_a)\mu_j$ for $x \in (c_{j-1}, c_j]$. In this specification μ_j is the mortality hazard prevailing in the age interval $(c_{j-1}, c_j]$, $j = 1, \ldots, K$, and $a(y_a)$ is the factor of proportionality for individuals with characteristics y_a. Denote as $s^{PW}(x)$ the corresponding survival function at age x. The observed hazard at age x in a heterogeneous population with a Gamma-distributed relative frailty then equals

$$\bar{\mu}^{PW}(x) = \frac{a(y_a)\mu_j}{(1 - a(y_a)\sigma^2 \log s^{PW}(x))} \qquad \text{for } x \in (c_{j-1}, c_j]. \tag{1}$$

Because of the numerical difficulties in estimating this piecewise-constant hazard function via maximum likelihood in the presence of many age-intervals, we implement a slight approximation to the hazard function in Equation (1). In particular, the difficulties in the estimation arise because the hazard $\bar{\mu}^{PW}(x)$ is not constant within age intervals. This results from the fact that the value of the survival function $s^{PW}(x)$ in the denominator declines with age x. For sufficiently small age-intervals, however, the effect of this changing value of the survival function on the observed hazard $\bar{\mu}^{PW}(x)$ within an age interval is small. The piecewise-constant hazard function with relative frailty can therefore be approximated by replacing the value of the survival function $s^{PW}(x)$ in Equation (1) with the value of the survival function at the mid-point of each age-interval. With this approximation, the observed hazard $\bar{\mu}^{PW}(x)$ is constant within age intervals and the MLE estimation is substantially simplified. The covariate-identified relative frailty model is

estimated with a constant mortality risk within two-year age intervals.

2.2 The DeMoivre Hazard Function and Its Application to Frailty Models

Kohler and Kohler (2000) have recently proposed a modified DeMoivre hazard function that implies a faster increase of the baseline hazard than exponential and allows to model heterogeneity at adult and young old ages, which are usually well fitted by the Gompertz model. Moreover, the DeMoivre hazard function is suitable to investigate the hypothesis whether the convergence of mortality by sex and socioeconomic groups is merely a result of a differential selection process and not to differential aging process or changing (diminishing) effects with age.

In particular, the modified DeMoivre hazard function, denoted μ^{MD}, has the following form:

$$\mu^{MD}(x) = a \left(1 - \frac{x}{\omega}\right)^{-b\omega}, \tag{2}$$

where a is the level-parameter of mortality, b is the slope-parameter and shows how fast mortality increases with age, and ω corresponds to a maximum attainable age at death, which in our estimation is set to 122.45 (Madame's Jeanne Calment age at death)[1]. The existence of this maximum attainable age implies that the mortality hazard increases faster than exponential, especially when x approaches ω. The corresponding survival curve s^{MD} is given by

$$s^{MD}(x) = \exp\left[-\frac{a\omega}{b\omega - 1}\left((1 - \frac{x}{\omega})^{-(b\omega-1)} - 1\right)\right]. \tag{3}$$

The slope-parameter b in Equations (2) and (3) needs to satisfy $b > \frac{1}{\omega}$ in order for the hazard and survival curves to be meaningful. (For an extensive discussion of the properties

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[1] Madame Jeanne Calment's age at death – 122 years and 5 months, or 122.45 years) is currently the highest verified age at death.

The parameter ω, which in our estimations is set to 122.45 years, can be also estimated directly in addition to the remaining parameters a and b. However, Kohler and Kohler (2000) have shown that the estimate of ω depends strongly on the age at which the data are censored, and this can yield an implausible estimate of ω.

of the modified DeMoivre hazard function see Kohler and Kohler 2000).

If we assume a Gamma-distributed relative frailty (following the notation from Vaupel et al. 1979) the observed hazard rate and survival curve implied by the modified DeMoivre hazard function, denoted by $\bar{\mu}^{MD}(x)$ and $\bar{s}^{MD}(x)$, have the form:

$$\bar{\mu}^{MD}(x) = \frac{\mu^{MD}(x)}{1 - \sigma^2 \log s^{MD}(x)} \tag{4}$$

and

$$\bar{s}^{MD}(x) = (1 - \sigma^2 \log s^{MD}(x))^{-1/\sigma^2}, \tag{5}$$

where $\mu^{MD}(x)$ and $s^{MD}(x)$ are the hazard function and survival curve for individuals with a constant frailty $z = 1$.

The modified DeMoivre hazard function has several properties that make it a plausible choice for estimating frailty models in mortality in the age range below age 100. The hazard, conditional on a constant frailty z, increases faster than exponential and agrees highly with nonparametric estimates of the age pattern of mortality (Yashin et al. 1995; Caselli et al. 2000; Kohler and Kohler 2000). The observed hazard implied by the modified DeMoivre hazard function, on the other hand, can capture a broad range of mortality patterns that are commonly encountered in the analysis of mortality at the age range 40–90.

3 The British Longitudinal Study

The analyses in this paper are based on the British Longitudinal study (LS).[2] The Longitudinal Study is a dataset compiled from a linkage between the census and vital event information for one percent of the population of England and Wales. The demographic and socioeconomic characteristics of the sample are collected since 1971, and our data cover a period of 26 years cohort follow-up until December 1997.

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[2] Detailed information about the Longitudinal Study (LS) is available at http://www.statistics.gov.uk/themes/compendia_reference/articles/longitudinal.asp.

Table 1: Summary statistics of the sample population by sex and type of exit from the Longitudinal Study (LS) in 1997.

Type of exit from the Longitudinal Study for England and Wales		
	Males	*Females*
Dead before end of study	44,789 (77.37%)	41,472 (63.44%)
Emigrated before end of study	485 (0.84%)	566 (0.87%)
Lost to follow-up before end of study	2,327 (4.02%)	3,346 (5.12%)
Alive at the end of study	10,288 (17.77%)	19,988 (30.58%)
Total	*57,889*	*65,372*

Our analyses are based on cohorts of men and women who were at least 50 years old and did not exceed the age of 70 years in April 1971. The total number of men in this selected sample at the beginning of the observation period is 57,889 (46.96%), and the total number of women is 65,372 (53.04%). During the 26 years of follow-up of these cohorts, 44,789 (77.37%) men and 41,472 (63.44%) women die before the end of the observation period in 1997. 10,288 (17.77%) men and 19,988 (30.58%) women of the initial sample population are still alive at the end of the observation period. Table 1 summarizes our sample by sex and type of exit from the study. We exclude from the analyses those individuals who are lost to follow-up before the end of the study or have emigrated during the period of observation.

The covariate education that is considered in our analyses, is measured at the beginning of the Longitudinal Study in 1971. Education corresponds to the number of years of education a person has had in 1971. As our analyses consider only elderly men and women in the age range 50 to 70 years, education is a time-invariant covariate.

Table 2: Relative risks by sex and education estimated from a piecewise-constant proportional hazard model.

	Age Groups			
	50–70 yrs	70–90 yrs	50–70 yrs	70–90 yrs
	Males		*Females*	
High education	1	1	1	1
Medium education	1.38**	1.23**	1.08	1.00
Low education	1.53**	1.39**	1.26**	1.19**
Unknown education	2.03**	1.55**	1.63**	1.35**

Notes: Standard errors are not reported. *p-values:* $^{+}$ $p < 0.05$; * $p <$ 0.01; ** $p < 0.001$.

4   The Effect of Education over the Life Span − Selection or Differential Aging Process

Consider Table 2 that reflects the relative risks of mortality by education experienced by members of the cohorts born 1901–1921 during two different age ranges, 50–70 years and 70–90 years. These relative risks are obtained from a piecewise-constant proportional hazard model that is estimated separately for men and women in these two age ranges. The baseline hazard (not reported in the table) is held constant within 2-year age intervals, and the model allows the baseline hazard to vary across these intervals.

The estimates conform to the well established gradient in mortality known from extensive research on socioeconomic mortality differentials: individuals with high education have the lowest mortality risk, while less educated men and women are exposed to a considerably higher risk of death during their life. For example, 50 to 70 years old men whose education is unknown, have about two times higher risk of death as compared to highly educated men in England and Wales. This gradient in mortality pertains also to the female cohorts, although the relative differentials in the risk of death are smaller for females as the ones observed for the male population. For example, the difference in the relative risk of death between women with medium and high education is not statistically significant at ages 50–70 years and even diminishes completely at older ages. Similar convergence in the relative risks of death is also estimated for the male and female cohorts in other educational groups, when they are 70–90 years old. When the cohorts are 70–90 years old, the

difference in the risk of death between men with unknown and high education diminishes to 1.55 (as compared to 2.03 at younger ages), and men with low education have a 39% higher risk of death as compared to highly educated men, whose risk of death at younger ages is about 53% lower. Similarly, the relative differences in mortality diminish also for the female cohorts so that low educated women at ages 70–90 years have 19% higher risk of death, in contrast to younger ages, when their risk of death is 1.23 times higher as compared to females with high education.

The cohort-based estimates in Table 2 suggest that the relative effect of education declines when the cohorts age, and the mortality trajectories by education may even converge at oldest-old ages. However, inferences about the changes in the effect and pattern of individual characteristics such as education over the life span of individuals are potentially biased if we do not consider that the observed mortality patterns for the respective populations may substantially deviate from the mortality dynamics on individual level (see also Vaupel and Yashin 1985). In this context, it is important to ask whether the observed convergence of the mortality trajectories by education at older ages that is more or less universal across place, occurs due to changes in the initial composition of the respective educational groups. This implies that the surviving population by education at old ages differs from the initial population observed at younger ages, and thus, the mortality trajectories converge due to the effect of differential pace of selection across groups of people with different degrees of education.

The selection hypothesis implies that the effect of individual-level characteristics such as education that are fixed relatively early in the life span of individuals remains stable over the life course, and the observed decline of educational differentials at older ages is in fact a 'heterogeneity ruse' (Vaupel and Yashin 1985) due to dynamics of population selection. If we assume that the effect of education or socioeconomic status is fixed over the life span of individuals, this would suggest that mortality of individuals with the same relative frailty is more or less proportional in different socioeconomic groups, while the observed mortality trajectories exhibit a pattern of convergence or crossovers that occurs primarily due to differential selection forces operating among these socioeconomic groups.

An alternative explanation of the observed convergence and even a cross-over of mor-

tality differentials by education is that people belonging to different socioeconomic groups are exposed to different environments and risk factors over their life, and the interaction of nature (genetic/biological influences on development) and nurture (exposure to different environmental risk factors) may result and probably cause a differential aging process. For example, aging as a process can be basically determined by three main domains – biological, psychological and social aging (Bergeman 1997). The biologically determined aging process refers to the anatomical and physiological changes of the organism that occur over time.[3] Psychological aging refers to age-related changes in behavior and personality, cognitive abilities and changes in the individual's ability to cope and adjust to new situations in life. The social aging is associated with age-related changes that are to a large extent influenced by the individual's social environment or socially imposed constraints. Thus, the social aging is to a large extent determined by the individual's social roles and status, access to various resources, access to health care facilities, differences in the quality of living arrangements, etc. It is obvious that these three domains are clearly related to each other, and the individual aging process itself is determined by a variety of factors that operate simultaneously.

Moreover, people belonging to different socioeconomic groups differ more or less in all three domains that determine the aging process. For example, a person with high education usually enjoys a higher social status in the society, a better access to resources, which allows for a more successful adjustment to new situations and coping with stressful life events. In part, the marginal impact of aging on mortality can be also off-set by differential investments in health or differential access to medical care. Similarly, knowledge about health prevention or healthy life styles that differs across social strata, can be an important factor influencing the extent to which aging raises mortality.

Thus, the differential increase of mortality observed by socioeconomic groups may in fact reflect a differential aging process that is determined by an interplay of various environmental risk factors. In this contest, the effect of individual characteristics (e.g.,

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[3]Extreme examples of biologically determined aging process are the Werner's Syndrome and the Hutchinson-Gilford Syndrome. Both are associated with a rare genetic disorder that causes an accelerated aging process. However, the symptoms of these diseases appear at different ages. While the Hutchinson-Gilford Syndrome appears early in life, the onset of the Werner's Syndrome is around age thirty.

education) may change during the life span so that at old and oldest-old ages the inter-action of the organism with other environmental factors may become more important for survival, and the effect of factors such as education or social status diminishes.

In order to investigate these patterns, we proceed further with the application of frailty models that allow for the estimation of the effect of compositional changes on the observed mortality patterns and test whether a selection hypothesis may plausibly explain the convergence of mortality differentials at older ages.

5 Results

Table 3 shows the results obtained for the male cohorts born 1901–1921 from the esti-mation of the modified DeMoivre hazard model and the covariate-identified frailty model (CIFM). Both functions assume a Gamma-distributed frailty. In particular, we specify two different types of model estimation. Model 1 and Model 3 assume that the different educational groups share an identical variation in individual-level frailty, and thus the variance distribution for σ^2 is set equal for the four educational groups. Model 2 and Model 4 provide an extension to the first two models and incorporate different variance of unobserved heterogeneity among the educational groups. This second specification is based on the observation that different educational groups are characterized by a great variation in life styles, socioeconomic and other environmental conditions, and thus, may differ in their frailty distribution.

Model 1, which is based on the modified DeMoivre hazard function, estimates that the variance of frailty in the male population equals $\sigma^2 = \exp(-0.43348) = 0.65$. According to this model, about 4% of the male population has a frailty of $z \leq .1$, about 31% have a frailty of $z \leq .5$, whereas approximately 11% have a frailty of $z \geq .2$.

The covariate-identified frailty model however, estimates a lower variance of frailty among males in England and Wales. According to the estimates in Model 3, the variance distribution of $\sigma^2 = \exp(-0.91851) = 0.40$. This implies that .78% of the males have a frailty of $z \leq .1$, 22% have a frailty of $z \leq .5$, while 7% of the males have a frailty of $z \geq 2$.

Model 2 and Model 4 represent an extension of the above two models and allow for

Table 3: Estimates of the modified DeMoivre hazard model and the covariate-identified model with a Gamma-distributed frailty for male cohorts born 1901-1921. (The baseline hazard of the CIFM-model is not reported; Moreover, the model does not contain a constant since it is included in the baseline hazard.)

Method	DeMoivre		Non-parametric	
	Model 1	*Model 2*	*Model 3*	*Model 4*
Specification for parameter α				
α_0 (constant)	-4.97650	-4.96968	–	–
	(0.04857)**	(0.06371)**		
α_1 (High	-0.35666	-0.31679	-0.31148	-0.30981
education)	(0.05124)**	(0.07379)**	(0.04910)**	(0.07421)**
α_2 (Low	0.17301	0.15568	0.15196	0.15333
Education)	(0.04235)**	(0.06104)$^+$	(0.03841)**	(0.06070)$^+$
α_3(Unknown	0.44068	0.42325	0.37575	0.42337
education)	(0.05608)**	(0.07990)**	(0.05588)**	(0.07990)**
Specification for parameter β				
β_0 (constant)	0.08698	0.08751	–	–
	(0.00162)**	(0.00167)**		
Specification for parameter γ				
γ_0 (constant)	-0.43348	-0.38050	-0.91851	-0.33829
	(0.05238)**	(0.11893)*	(0.27585)**	(0.30769)
γ_1 (High		0.10912		0.12866
education)		(0.12688)		(0.12018)
γ_2 (Low		-0.04335		-0.04760
education)		(0.10542)		(0.10340)
γ_3 (Unknown		-0.04416		-0.05106
education)		(0.12107)		(0.12548)
Specification for parameter ω				
ω	set to 122.45	set to 122.45	–	–

Notes: Standard errors in parentheses. *p-values:* $^+$ $p < 0.05$; * $p < 0.01$; ** $p < 0.001$.

differences in the variance of frailty among educational groups. The DeMoivre hazard model estimates that males with high education represent the most heterogeneous group of the male population. For example, the variance of frailty among highly educated males yields an estimate of $\sigma^2 = \exp(-.3805 + .1091) = 0.76$. Males with medium education have a variance of frailty of $\sigma^2 = 0.68$. According to this DeMoivre hazard specification there is almost no difference in the frailty distribution of men with low and unknown education, and both frailty distributions do not diverge substantially from the frailty distribution of males with medium education. Low and unknown educational groups represent the least heterogenous male population and have a variance in frailty of $\sigma^2 = 0.65$.

The results obtained from the covariate-identified frailty model (Model 4) yield a higher variance of frailty distribution across educational groups as compared to the estimates based on the DeMoivre hazard function in Model 2. However, the estimated pattern of variance in frailty across different educational groups is identical in the parametric as well as in the non-parametric approach. According to the non-parametric approach (Model 4), the most heterogenous group is represented by males with high education, who have a variance of frailty $\sigma^2 = 0.81$. Males with medium education represent a less heterogenous group ($\sigma^2 = 0.71$). Similarly to the modified DeMoivre model, the covariate-identified frailty model estimates also that there is no difference in the frailty distribution of men with low and without education. The model estimates a variance of frailty in both educational groups of $\sigma^2 = 0.68$.

Figure 1 shows the observed, population and individual hazard of mortality obtained from Model 2 and Model 4 in Table 3, which assume different frailty distribution between the educational groups. The upper panel in Figure 1 shows the results based on the modified DeMoivre hazard function, while the lower panel shows the fit of the covariate-identified frailty model. As Figure 1 shows, the parametric as well as the non-parametric approach yield a very good fit of the observer population hazard for males. In both approaches, the observed mortality hazards converge across socioeconomic groups, and this convergence is closely traced by the estimates of the population hazard. Since the population hazard is partly determined by changes in the frailty composition of the population, it does not reflect the age pattern of the individual mortality risks. The estimates of the
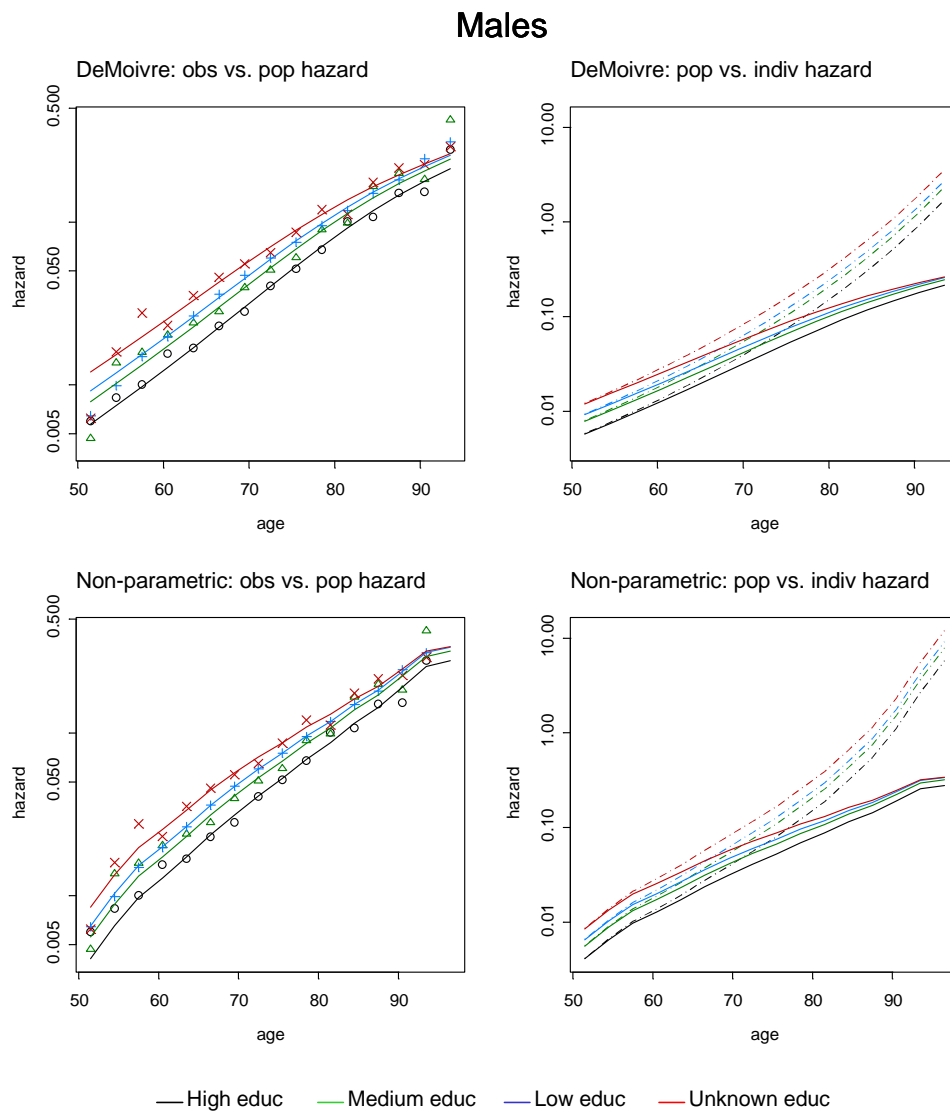
Figure 1: Observed, population and individual hazard for males based on the assumption of differences in the variance of frailty between educational groups. Upper graphs are based on Model 2 (modified DeMoivre hazard function), lower graphs are based on the estimates from Model 4 (Covariate-identified frailty model).

individual mortality hazard in the right graphs of Figure 1 however, show that on the individual level the mortality trajectories by education do not converge, but increase in a parallel fashion. This also implies that the educational differentials in mortality in fact do not diminish with age, but remain constant over the life span of individuals.

The results shown in Figure 1 also convincingly suggest that the individual hazard of mortality increases faster then exponentially, and this pattern of mortality increase is similar in both approaches – the modified DeMoivre and the CIFM. However, the figure shows also that the increase in the individual mortality hazard based on the modified DeMoivre approach is slower as compared to the increase in the individual risk of death with age estimated from the non-parametric approach. This pattern of differential increase in the individual mortality hazard is consistent with the estimates of frailty. The CIFM estimates a higher variance of frailty across socioeconomic groups, which results in a steeper increase of the individual mortality hazard.

Table 4 shows the results for the female cohorts born between 1901–1921. Model 5 is based on the modified DeMoivre hazard function and assumes an identical frailty distribution among females. The model estimates a variance of $\sigma^2 = \exp(-.7438) = 0.48$. According to this estimate, 1% of the female population has a frailty of $z \leq .1$, about 25% of the females have a frailty of $z \leq .5$, whereas approximately 9% have a frailty of $z \geq .5$.

The covariate-identified frailty Model 7, which is also based on the assumption that the educational groups have an identical frailty distribution, estimates a substantially higher variance in frailty across females ($\sigma^2 = 0.80$) as compared to the modified DeMoivre approach. Model 7 estimates that about 6% of females in the sample have a frailty of $z \leq .1$, around 35% have a frailty of $z \leq .5$, and 12% of the females have a frailty of $z \geq 2$.

In analog to the analyses for males, Model 6 and Model 8 allow for differences in the frailty composition across female educational groups. In this context, the DeMoivre approach (Model 6) estimates that there is no a substantial difference in the frailty composition of females with high, low and unknown education. For example, the estimates of σ^2 for low educated women and women with unknown education yields a values of $\sigma^2 = .47$, while the variance distribution of frailty among highly educated females equals 0.45. The least heterogenous group among females consists of women with medium edu-

Table 4: Estimates of the modified DeMoivre hazard model and the covariate-identified model with a Gamma-distributed frailty for female cohorts born 1901-1921. (The baseline hazard of the CIFM-model is not reported; Moreover, the model does not contain a constant since it is included in the baseline hazard.)

Female Mortality in England and Wales, Estimates for Cohorts born 1901–1921				
Method	**DeMoivre**		**Non-parametric**	
	Model 5	*Model 6*	*Model 7*	*Model 8*
Specification for parameter α				
α_0 (constant)	-5.58137	-5.65623	–	–
	(0.05424)**	(0.06959)**		
α_1 (High education)	-0.03603	0.03046	-0.03850	0.02003
	(0.05729)	(0.07905)	(0.06442)	(0.08049)
α_2 (Low education)	0.20583	0.28495	0.23778	0.27701
	(0.04869)**	(0.06652)**	(0.06078)**	(0.06986)**
α_3 (Unknown education)	0.42309	0.50335	0.49772	0.50264
	(0.05517)**	(0.07589)**	(0.08705)**	(0.08527)**
Specification for parameter β				
β_0 (constant)	0.07544	0.07529	–	–
	(0.00115)**	(0.00117)**		
Specification for parameter γ				
γ_0 (constant)	-0.74383	-1.22524	-0.22661	-0.86306
	(0.06723)**	(0.36978)**	(0.35525)	(0.96853)
γ_1 (High education)		0.41962		0.27346
		(0.38195)		(0.33368)
γ_2 (Low education)		0.47840		0.29371
		(0.35564)		(0.38946)
γ_3 (Unknown education)		0.48117		0.27961
		(0.36446)		(0.43852)
Specification for parameter ω				
ω	set to 122.45	set to 122.45	–	–

Notes: Standard errors in parentheses. *p-values:* $^{+}$ $p < 0.05$; * $p < 0.01$; ** $p < 0.001$.

cation ($\sigma^2 = 0.29$).

The CIFM estimates a higher variance of frailty among females as compared to the parametric approach. In particular the model estimates for females with medium education $\sigma^2 = 0.42$, which is considerably higher as compared to the parametric estimate. However, the non-parametric approach confirms the general pattern of frailty distribution among females obtained from the DeMoivre model. The CIFM estimates also that there is no substantial difference in the frailty composition of females with high, low and unknown education (σ^2 for females with high education equals 0.55, for females with low education $\sigma^2 = 0.57$, and for females with unknown education $\sigma^2 = 0.56$).

These differences in the estimated variance of frailty between the DeMoivre hazard model and the CIFM are also reflected in the differential increase of the individual baseline hazard shown in the right graphs of Figure 2. The analysis in Figure 2 is based on the assumption that the educational groups differ by their frailty distribution. Similarly to the pattern estimated for the male population, Figure 2 shows that the individual mortality hazard that is estimated nonparametrically, increase steeper as compared to the estimated baseline hazard from the DeMoivre approach (upper right graph in Figure 2). Nevertheless, both approaches suggest that the mortality rates for different socioeconomic groups with a constant relative frailty $z = 1$ increase in a parallel fashion. This common result suggests that the convergence of the mortality trajectories by socioeconomic groups may be traced back to differences in the selection process operating among different social strata rather then by differences in the aging process itself.

6   Selection or Differential Aging – Why Does It Matter?

The proceeding analyses provide a convincing evidence that a substantial part of the differential increase of mortality by sex and educational groups, which results in a convergence of the mortality trajectories at older ages, can be explained primarily by the differential pace of the selection process that operates among social strata rather then by a differential aging process. In this section, we pursue these analyses further and investigate the implication of unobserved heterogeneity on the estimation of various life table functions.
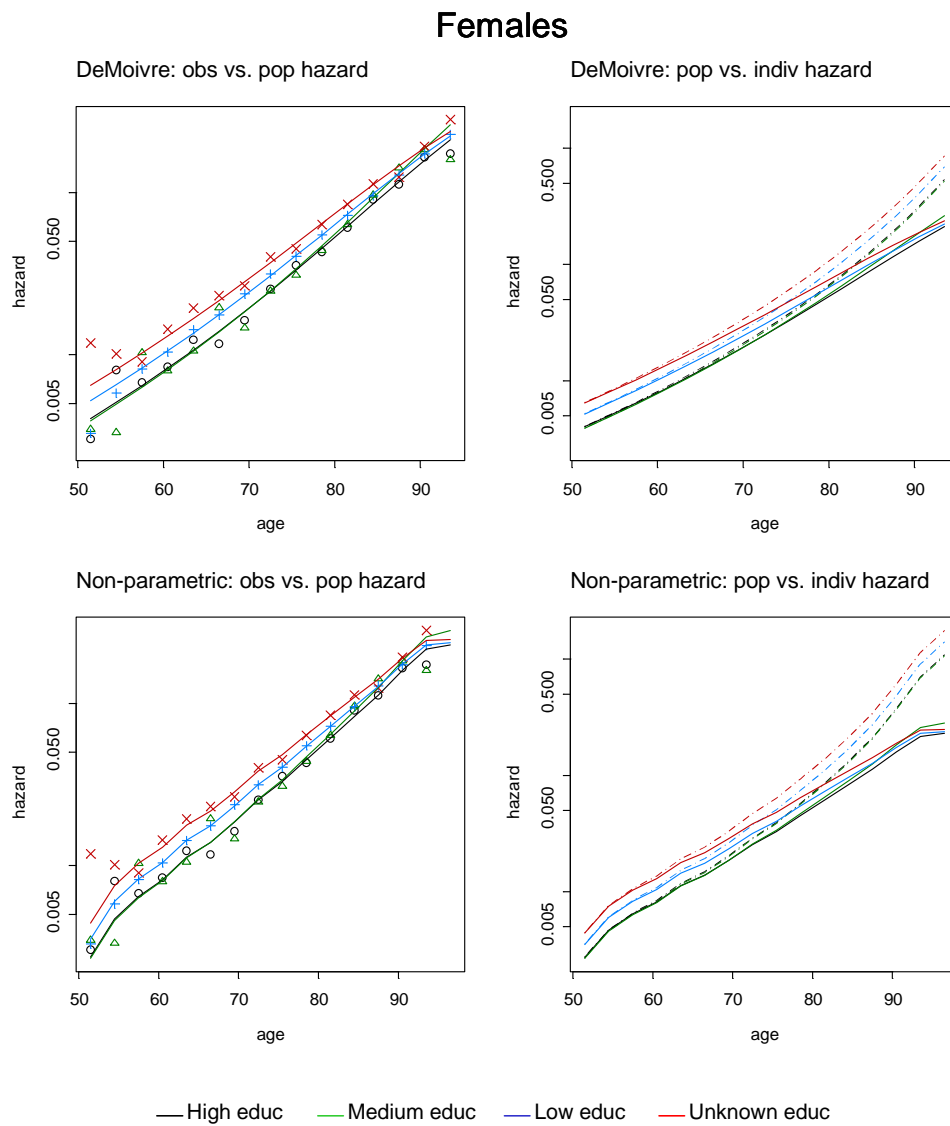
Figure 2: Observed, population and individual hazard for females based on the assumption of differences in the variance of frailty between educational groups. Upper graphs are based on Model 6 (modified DeMoivre hazard function), lower graphs are based on the estimates from Model 8 (Covariate-identified frailty model).

In particular, we discuss the difference in population (cohort) survivorship and individual survivorship based on the estimates from the modified DeMoivre hazard model with unobserved heterogeneity.

The upper graph of Figure 3 shows the population (cohort) versus individual hazard of mortality for males with medium and high education. The estimates are based on the modified DeMoivre hazard function that assumes different frailty by education (see Model 2 in Table 3). While on the population level we observe a flattening of the mortality trajectory at ages above 80, the mortality hazard of an individual with a frailty $z = 1$ increases faster then exponentially. Moreover, once we control for compositional changes in the population that occur the older the cohorts become, the mortality trajectories for high and medium education do not converge anymore. Quite in reverse, the educational differences in mortality for individuals with high and medium education remain stable over the life span of individuals.

These differences in the increase of the population versus individual hazard of mortality are also reflected in the survival curves shown in the lower graph of Figure 3. As this graph indicates, the cohort survivorship above age 50 is greater as compared to the survival of the 'average individual' among the population of 50-year old, who has a frailty of $z = 1$. The survival curve of this individual is shifted to the left, which implies shorter life expectancy as compared to the cohort life expectancy. In particular, this difference between individual and cohort survivorship becomes visible at older ages above 70 years.

Figure 4 shows the individual and cohort life expectancy at age 50 for males with medium and high education as a function of the frailty z (*Note*: life expectancy is plotted on a logarithmic scale). The two parallel full lines in the upper graph of Figure 4 reflect the level of life expectancy for high and medium education, which we would estimate at age 50 on the basis of the population (cohort) mortality hazard without consideration of the frailty composition of these cohorts. On the population level, males at age 50 with high education have a remaining life expectancy of 27.28 years, while men with medium education have 2.2 years shorter life expectancy and have a remaining life expectancy of 25.08 years.

The two dotted lines show the individual life expectancy for men in these two so-
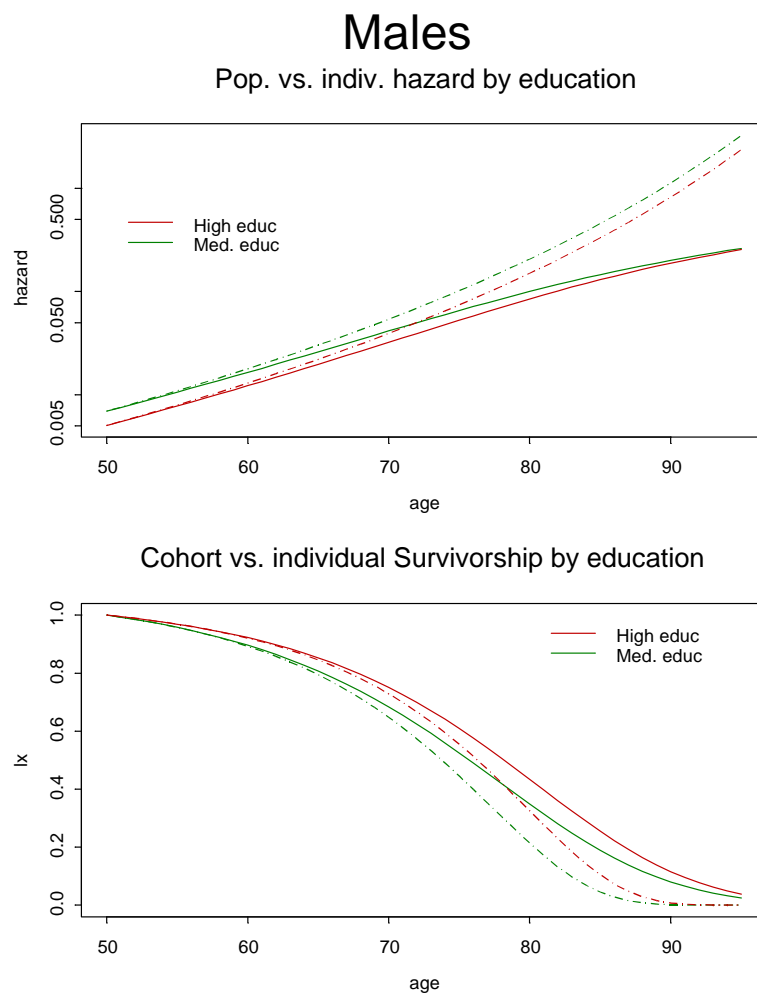
Figure 3: *Upper graph*: cohort versus individual mortality hazard for males with high and medium education. *Lower graph:* cohort versus individual survivorship for males with high and medium education. Estimates are based on the modified DeMoivre hazard model, Model 2 in Table 3.
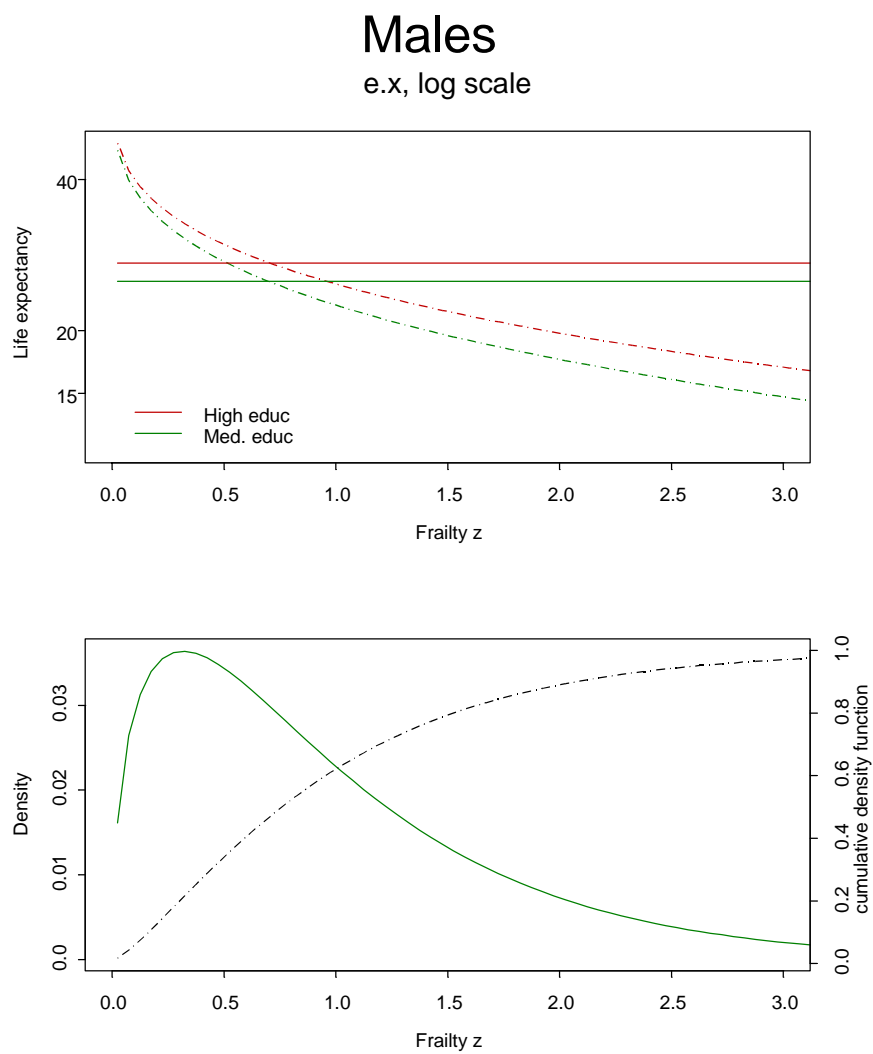
Figure 4: *Upper graph:* Cohort and individual male life expectancy for high and medium education at age 50 as a function of frailty z. *Lower graph:* Density and cumulative density functions of frailty z.

cioeconomic groups as a function of the frailty z. The difference between the estimated population and individual life expectancy indicates that if we do not consider the frailty composition and frailty distribution in the population, we tend to overestimate the survival of the average individuals with a frailty of $z = 1$. This is even more pronounced for individuals with a high frailty (e.g., $z \geq 2$). The density and cumulative density functions of frailty plotted in the lower graph of Figure 4 indicate that about 54% of individuals with an average frailty $z \approx 0.7$ have in fact individual life expectancy that is sometimes substantially lower then the estimated life expectancy on a cohort level.

In summary, the most important insight from these analyses is that life expectancy of the average individual with a frailty of $z = 1$ is below the average life expectancy of the population, and if we do no consider the impact of heterogeneity in the analyses, we tend to overestimate individual survival.

In addition, the upper graph of Figure 4 reveals a very interesting aspect of how the effect of socioeconomic factors depends on frailty. If we consider individuals with low frailty ($z \leq 0.5$), then we do no observe large relative differences in individual life expectancy between men with high and medium education. However, if we move to the right tail of the frailty distribution, that is, we consider individuals with a high frailty $z \geq 1.5$, then as indicated by the upper graph of Figure 4, the observed relative socioeconomic differentials in individual life expectancy become substantially bigger as compared to the pattern estimated for low-frailty individuals. This result indicates that socioeconomic risk factors are indeed a very important determinant of individual survival. Moreover, the impact of socioeconomic factors on survival is not equal for all individuals, but the effect of having a high education is greater for those individuals who are 'placed' more to the right side of the frailty distribution, e.g. high-frailty individuals.

Furthermore, on individual level the effect of socioeconomic factors varies also quite substantially by age. According to our estimates, if we do not consider frailty, we would estimate that males with high education have about 9% higher life expectancy at age 50 as compared to those wit medium education. On individual level, i.e. for individuals with a frailty of $z = 1$, the difference in life expectancy between men in these two socioeconomic groups at age 50 is 'only' 10% in favor of highly educated men. If we consider old men at

age 80, then our estimates show that on the cohort level we would estimate that old men with high education have 8% higher life expectancy as compared to those with medium education. However, on the individual level ($z = 1$), highly educated men have 24% higher life expectancy as compared to those men with a medium education and a frailty of $z = 1$. Thus, our results suggest that while on the population level we will estimate that socioeconomic differentials between different groups diminish with age, in fact, on the individual level the differences in survival between educational groups become even large the older the population is.

7   Conclusions

A pervasive finding in the literature on mortality differentials by sex, ethnicity, and/or socioeconomic status is the convergence and the crossover of the mortality trajectories at old and oldest-old ages. For instance, it is well documented that the observed socioeconomic mortality differentials are largest at adult working ages and diminish at older ages, which suggests that the effect of various socioeconomic characteristics declines gradually with age. However, if the empirical analyses do not consider the compositional changes that occur due to selection forces operating among the population at risk, our conclusions about the effect and interplay of some individual-level characteristics with age may be biased. In this paper, therefore, we investigate whether a selection hypothesis can provide a plausible and consistent explanation for the observed convergence of the mortality trajectories by sex and socioeconomic status (education) at old and oldest-old ages.

To understand the dynamics that underlie the convergence of the mortality trajectories by sex and socioeconomic status with increasing age, we need to incorporate in the analyses unobserved heterogeneity. However, this consideration of selection is often hampered by the fact that the effect of differential selection becomes primarily visible when the level of mortality is moderately high (i.e. at old ages), and a strong selection process operates in the observed population. Furthermore, in our analyses we are interested in whether and how the effect of certain fixed individual-level characteristics such as education changes over the life span of individuals, and therefore we need to incorporate the effect of differ-

ential selection already at younger ages. In order to overcome this estimation problem, we implement new statistical methods that allow for modelling unobserved heterogeneity already at adult working ages. In particular, we compare a parametric (the modified DeMoivre hazard function) with a non-parametric (the covariate-identified frailty model) approach.

Both approaches yield consistent and very similar results. Our analyses suggest that the individual risk of death arises faster than exponential. However, the increase of the baseline mortality hazard estimated from the covariate-identified frailty model (CIFM) is steeper and faster as compared to the parametrically estimated baseline hazard.

Furthermore, the analyses convincingly suggest that the convergence of the mortality trajectories by sex and socioeconomic groups may be primarily attributed to the effect of differential selection forces operating among these social strata, instead to fundamental differences in the aging process or variation in the effect of fixed socioeconomic characteristics over the individual's life-span. The estimates based on the parametric and non-parametric approach show that, when we control for compositional changes, the individual baseline hazard by education increases in a parallel fashion, while the observed population trajectories of mortality converge at older ages. This suggests that the mortality differentials by socioeconomic groups remain stable over the life span of individuals with the same relative frailty and the effect of fixed socioeconomic characteristics such as education does not change over the individual's life span.

If we assume that socioeconomic groups differ by their frailty distribution, then both approaches (the modified DeMoivre hazard model and the CIFM) estimate similar pattern of variance in frailty across educational groups. However, the non-parametric approach estimates in general higher variance of frailty for both sexes as compared to the parametric estimates. Both approaches estimate that the most heterogenous group among men is represented by highly educated men, while there is no a substantial difference in the variance of frailty estimated between men with medium, low and no education. Among women, this pattern is quite different: the least heterogenous group is represented by women with medium education, while there is almost no difference in the variance of frailty among women with high, low and no education.

One of the most important insights from our analyses is that there is a substantial difference between the cohort and individual survivorship. Our estimates show that life expectancy of the 'average individual' with a frailty of $z = 1$ is substantially below the average life expectancy that we estimate on a population level. Moreover, our analyses show also that the effect of socioeconomic factors on life expectancy is not equal for all individuals, but the effect of having higher education is greater the more frail are the individuals. In addition, on individual level we estimate that the relative differences in life expectancy by socioeconomic group even increase, which is in contrast to the estimates on population level.

In summary, our analyses suggest an important role of frailty and selection considerations in the assessment of how socioeconomic factors influence mortality. In particular, our results question the widespread belief that the mortality hazards across socioeconomic groups are converging at higher ages, and they suggest that analyses on population level may substantially overestimate the life expectancy of the 'average individuals'. In future research, explicit measures of persistent frailty differentials among individuals, that are included in analyses of socioeconomic mortality differentials, can provide more direct evidence for the above hypotheses. Our analyses, however, suggests that such research is quite promising for measuring mortality differentials by socioeconomic status.

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