Mortality Crossovers: A Simple Model to Disentangle "Real" Effects from Compositional Effects

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Abstract

Empirical studies often find that differences in mortality by socioeconomic status tend to converge and even cross-over with increasing age. Does this imply that risks are also converging on the individual level? Not necessarily. The decreasing gap can be also explained by compositional changes: people who are frailer than others tend to die younger, resulting in a selected healthier sub-population with an observed leveling-off in the observed mortality at higher ages.

We present here a simple model that allows to disentangle direct effects from compositional effects. The model assumes that individuals in two groups are subject to a Gompertz mortality schedule and that the hazards of the two groups are proportional to each other. Individuals within the two groups differ in their frailty, which is assumed to be Gamma-distributed.

We show that a simple mathematical expression allows us to estimate at what age the two hazards would converge *if only compositional effects were present*. If the observed crossover age is earlier, we can postulate that hazards do actually converge on the individual level. If the two trajectories have not yet converged at the estimated age, we hypothesize that individuals hazards are diverging with increasing age.

Our model is not restricted to analyze only converging socioeconomic mortality differentials. It can be employed whenever two groups exist whose observed mortality hazards converge or crossover (e.g. smokers and non-smokers).

Introduction. Few relationships in the social sciences are as well established as the one between socioeconomic status (SES) and mortality. Regardless of its measurement, people from lower socioeconomic groups typically experience higher mortality [4]. With regard to the development of these differences over age, two contrasting theories are typically brought forward [5]: *Differences decrease with age* because social factors lose and biological factors gain importance; the promotion of welfare states policies such as Medicare to reduce inequalities in access to health care or the disappearance of job-related hazards at higher ages. Alternatively, it can be argued that *differences increase with age*. For instance, there is a considerable time-lag between detrimental behavior such as smoking, which is more common among people from lower socioeconomic groups, and its impact on health and mortality. Furthermore, it can be argued that advantages early in life may accumulate over the life course and vice versa. As summarized by Willson et al. [15, p. 1891]: "[C]umulative advantage as a mechanism of inequality suggests that early advantage continues to grow over time, conceptualized in terms of increasing returns to resources such as education."

Empirical studies of SES and mortality typically exhibit patterns where the differences in the excess risk of dying become smaller with age, eventually leading to a complete convergence or even a cross-over.

Does this imply that the theory of cumulative advantage/disadvantage is wrong? Not necessarily. If we make the reasonable assumption that some individuals *within* a given socioeconomic group are frailer than others, we can expect them to die on average at younger ages than their more robust peers. Consequently, the observed mortality is lower than expected in a homogeneous population [12, 13, 14]. Hence, the trend towards convergence

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may potentially be the result of compositional effects *despite* diverging mortality hazards on the individual level.

This extended abstract outlines a simple method which allows to disentangle compositional effects from "real" effects. Differentiating between those two effects is not new in demography. Vaupel and Canudas Romo [11], for instance, provide general methods to decompose the two effects. Our model is less universal. It employs a different approach and is specifically tailored to analyze mortality convergence and crossovers.

Mathematical Derivation. Let's assume that two groups, A and B, exist in a population. The individual age-specific hazards $\mu^0(x)$ are proportional to each other by a factor c. This is the underlying assumption in many survival models, most notably the Cox regression model.

$$c\mu_A^0(x) = \mu_B^0(x) \tag{1}$$

The age-specific hazards themselves follow a Gompertz distribution with the same slope parameter β but a different α-component to ensure proportionality.

$$\mu_A^0(x) = \alpha_A\, e^{\beta x}; \quad \mu_B^0(x) = \alpha_B\, e^{\beta x} = c\alpha_A\, e^{\beta x} \tag{2}$$

Let's assume further that the respective members of groups A and B are not homogeneous but that there is unobserved heterogeneity within the two groups. Some individuals are frailer and some others are more robust. This distribution of frailty is assumed to follow a Gamma distribution with an initial unit mean and variance σ^2. While Beard [2] was the first to model heterogeneous populations with the Gamma distribution, it became only widely used after 1979 when Vaupel et al. [13] employed the Gamma distribution for such "frailty models". Recent mathematical papers confirmed that assuming a Gamma distribution for frailty is reasonable for human mortality [7, 8].

Since frailer individuals tend to die earlier on average, the observed force of mortality is not increasing as predicted by the individual Gompertz hazards but levels off at higher ages. Based on [13] and assuming the aforementioned Gamma distribution, Vaupel [10] showed that the observed mortality hazard at age x, $\bar{\mu}(x)$, can be expressed as

$$\bar{\mu}(x) = \mu^0(x)\, \bar{s}_c(x)^{\sigma^2} \tag{3}$$

where $\mu^0(x)$ is the individual force of mortality at age x, $\bar{s}_c(x)$ denotes cohort survivorship until that age and σ^2 is the variance of the Gamma distribution.

To obtain the age when both observed mortality hazards converge, we can write:

$$\bar{\mu}(x)_A = \bar{\mu}(x)_B \tag{4}$$

Under the given assumptions of a Gamma-Gompertz model and proportional hazards, this expands to:

$$\alpha_A e^{\beta x}\left(e^{\frac{\alpha_A}{\beta}\left(1-e^{\beta x}\right)}\right)^{\sigma^2} = \alpha_B e^{\beta x}\left(e^{\frac{\alpha_B}{\beta}\left(1-e^{\beta x}\right)}\right)^{\sigma^2} = c\alpha_A e^{\beta x}\left(e^{\frac{c\alpha_A}{\beta}\left(1-e^{\beta x}\right)}\right)^{\sigma^2} \tag{5}$$

Solving for age x, we obtain

$$x = \frac{\ln\left(1 - \frac{\ln(c)}{\left(\frac{\alpha_A\, \sigma^2(1-c)}{\beta}\right)}\right)}{\beta}. \tag{6}$$

Application. Doblhammer et al. [3] report 60,444 men with basic education in Austria in 1991 in age-group 35–39 years. 143 of them died during the following year resulting in a probability of dying of $\frac{143}{60444} = 0.0023658$. This can be translated into a hazard of $\alpha_A = 0.00236303$.[2] For people with high education, mortality is even lower with a probability of dying of $\frac{27}{27745} = 0.0009731$, corresponding to a hazard of $\alpha_A = 0.0009736221$. Hence excess mortality c equals 2.432801.

At what age are the two mortality hazards expected to converge? According to the equation above, we need two more quantities: the slope of the individual hazard (β) and the variance of the gamma distribution (σ^2). A typical finding for the observed slope of the Gompertz distribution is a value of about 0.10. Since the observed force of mortality overestimates the mortality hazard on the individual level,[3] a value of 0.10 represents a lower boundary for the slope of the individual hazard. Different values have been estimated in the literature for the variance of the gamma distribution. Barbi [1], for instance, obtains values for σ^2 of 0.14663 for women and 0.09710 for men. Manton et al. [6] estimate values between 0.2544 and 0.3584. An overview of potential σ^2 values is given in [5]. Assuming that the average person in the age-group 35–39 is 37.5 years old, the expected ages of the convergence of the two mortality hazards are given in Table 1 for the range of potential β- and σ^2-values. With the exception of a rather homogeneous population ($\sigma^2 = 0.10$) combined with a slow age-specific increase of mortality on the individual level ($\beta = 0.1$), one can expect that the mortality hazards of the two groups should converge before the age of 90. According to the data published in [3], though, mortality of people with basic education was still almost 40% higher at ages 85–89, the highest observed ages, than of people with tertiary education. How can we interpret this? A "pure" compositional effect would have led to a convergence before age 90. If the observed convergence had occured before that expected age of convergence, we could have postulated that hazards are converging also on the individual level. Since we have not observed any convergence yet, one can deduce that hazards are not converging but rather diverging on the individual level.

Table 1: Expected age of convergence of mortality hazards for Austrian men with low and high education depending on assumptions for slope β and σ^2

		β						
		0.10	**0.11**	**0.12**	**0.13**	**0.14**	**0.15**	**0.16**
σ^2	**0.10**	93.22	89.02	85.45	82.37	79.70	77.34	75.25
	0.15	89.18	85.35	82.08	79.27	76.81	74.65	72.73
	0.20	86.33	82.75	79.70	77.06	74.76	72.74	70.94
	0.25	84.11	80.74	77.85	75.36	73.18	71.26	69.55
	0.30	82.31	79.09	76.35	73.97	71.89	70.05	68.42
	0.35	80.79	77.71	75.07	72.79	70.80	69.03	67.46

Summary & Discussion. Our simple model allows to specify at what age mortality trajectories are expected to converge for two sub-populations if only compositional changes due to mortality selection were present. The assumptions for our model are standard for mortality research: We expect a) that mortality hazards are proportional to each other, b) mortality harzards increase on the individual level according to Gompertz' law, and c) individuals within a group are heterogeneous following a Gamma distribution. If the variances of the Gamma distribution in the two groups are not equal but differ by a factor f, the model's equation changes only slightly to:

[2] Using the approximation of $\mu(x + 0.5) \approx -ln(1 - q(x))$ [9].

[3] The only exception is when $\bar{s}_c(x)^{\sigma^2} = 1$, see Equation 3.

$$x = \frac{\ln\left(1 - \frac{\ln(c)}{\left(\frac{\alpha_A \sigma^2 (1-cf)}{\beta}\right)}\right)}{\beta}.$$

The model itself can easily be adapted for other research questions such as: "For a given age x, what is the expected excess mortality of one sub-population?" Although we have chosen socioeconomic mortality differentials, the model can be also employed for other applications such as black/white mortality differentials, the mortality crossover of smokers and non-smokers, ...
Finally, it should be pointed that the example we have shown here has an obvious problem: We assume that selection works in the same way in a period perspective as it does for cohorts. However, this is not the fault of the model but of the example.

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