

Swedish Mortality Does Not Decelerate in Recent Cohorts

Extended Abstract of Preliminary Results

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Abstract

Background: Mortality at the highest ages tends to level off, a phenomenon typically described as mortality deceleration. The standard explanation is mortality selection. There are claims in the literature, however, that the observed mortality deceleration is an artifact.

Objective: By using data of the highest quality, which do not suffer from any of the problems claimed, we wanted to show that mortality does, indeed, decelerate.

Data & Method: We used Swedish cohort data for birth years 1890 through 1917. All required data were available by exact date to be able to estimate exact person-years of exposure. We plotted the resulting death rates and fitted Gompertz- and Gamma-Gompertz-models to the observed data.

Results: While we found mortality deceleration for older birth cohorts, none of the younger birth cohorts showed any signs of mortality deceleration. This finding contradicts our expectations completely and requires further investigation.

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Introduction

Background For most of adult life the rate of change of mortality with age is fairly constant: Mortality increases by slightly more than 10% from one age to another. This log-linear increase is often parameterized by the slope b in Gompertz' mortality law

$$\mu(x) = ae^{b(x)} \quad (1)$$

where $\mu(x)$ denotes the mortality hazard at age x . It has often been observed that mortality at the oldest ages falls below the predicted straight line in a Gompertz setting, typically labeled "deceleration" (e.g., Horiuchi and Coale, 1990; Horiuchi and Wilmoth, 1998). Age misreporting could obviously be one of the reasons. As stated by Preston et al. (1999, p. 165): "For each of the age misreporting patterns and each of the methods of mortality estimation, we find that age misstatement biases mortality estimates downwards at the oldest ages." Alternatively, mortality might actually increase at a slower pace at older ages due to a lower metabolic rate (Ukrainitseva and Yashin, 2001). The most common explanation refers to a compositional change in the population: Individuals may actually age at the same pace throughout their whole life, translating into a Gompertzian mortality increase. Individuals are not the same, however. Some are frailer than others. They die, on average, at younger ages. As a consequence, the *observed* population-level mortality levels off with increasing age (Vaupel et al., 1979; Vaupel and Yashin, 1985). The age-specific hazard of individual i is therefore modeled as $\mu_i(x) = z(i)\mu_0(x)$, where $z(i)$ is the frailty distribution among individuals and $\mu_0(x)$ is the baseline mortality hazard. The baseline hazard is still modeled by a Gompertz distribution and the frailty distribution is often assumed to follow a Gamma Γ distribution with a mean of 1. The population level hazard at age x can then be expressed as (Vaupel and Missov, 2014):

$$\bar{\mu}(x) = \frac{ae^{bx}}{1 + \frac{a\gamma}{b}(e^{bx} - 1)} \quad (2)$$

where γ denotes the variance of the Γ -distribution.

While the majority of researchers agrees with the phenomenon of mortality deceleration and might only argue about the actual cause, the best possible model, . . . , there is a minority of researchers who deny the existence of mortality deceleration. Their perspective is probably best described by the summarizing sentences in the abstract of Gavrilov and Gavrilova (2011, p. 432):

“Earlier reports of mortality deceleration at ages below 100 appear to be artifacts of mixing together several birth cohorts with different mortality levels and using cross-sectional instead of cohort data. Age exaggeration and crude assumptions applied to mortality estimates at advanced ages may also contribute to mortality underestimation at very advanced ages.”

Objective Since some of the methods of Gavrilov and Gavrilova (2011) can be criticized (e.g., assigning of sex by first name, the onset of mortality deceleration is probably earlier than age 88 when their estimates start, non-availability of data to replicate their findings), we wanted to show with cohort data of the highest quality that mortality does indeed decelerate.

Data and Methods

We obtained data for all individuals from birth cohorts 1890 through 1917 who were alive in Sweden on 01 January 1960. Out of these 2,376,076 individuals. We only included those individuals with the highest data quality and who were born in Sweden (2,086,179 individuals). 2,015,574 died before the end of the observation window (31 Dec 2017), 1536 emigrated and 1891 were still alive.

All data are available by exact dates (day, month, year), which allows us to estimate

the number of person-years lived at each age for each cohort more precisely than done in other studies on mortality deceleration.

Our main method are plots of death rates by sex and birth cohort. Deaths rates, $m(x)$, are simply the numbers of deaths at a given age, sex, and birth cohort, $D(x)$ (suppressing the subscripts for sex and cohort), divided by the corresponding number of person-years lived, $N(x)$ (“exposures”).

The visual display of the death rates as discrete approximations to the instantaneous hazard (the force of mortality) is emphasized by two parametric fits, which were estimated in the usual Poisson likelihood framework: 1) A Gompertz model; 2) A Gamma-Gompertz model.¹

Results

Figures 1–3 (pages 7–9) show the observed death rates for each cohort from 1890 through 1917, separately for women and men, as black + symbols. The fitted Gompertz- and Gamma-Gompertz-lines are illustrated by translucent red and blue lines, respectively. For the oldest cohorts included in this study (1890–1895), we can see some deceleration of mortality at the highest observed ages. This leveling off is gradually disappearing when we look at younger birth cohorts. No deceleration can be detected at least since birth cohort 1912, also supported by the congruent parametric fits.

We also find support for our finding when plotting the γ term, i.e. the variance of the frailty distribution in the Gamma-Gompertz model as shown in Figure 4 (page 10): The γ component of older birth cohorts gradually disappears. With $\gamma \rightarrow 0$, the denominator of Equation 2 becomes 1 and we end up with the Gompertz model.

¹We did a constrained optimization where we forced the parameters to be positive.

Outlook

Our goal was to show that mortality deceleration does, indeed, exist. For this purpose we obtained data of the highest reliability and granularity: Swedish cohort data with information available by single days. This should invalidate most claims by Gavrilov and Gavrilova (2011) towards studies that exhibited mortality deceleration.

While we found mortality deceleration for older birth cohorts, we could not detect any leveling off at all for younger birth cohorts.

Due to the data design, we could not cover the same ages for all birth cohorts. But even the youngest birth cohort is included until age 100. One possible explanation is that mortality deceleration due to selection might appear at later ages. This contradicts, however, previous findings: Mortality typically starts decelerating at younger ages.

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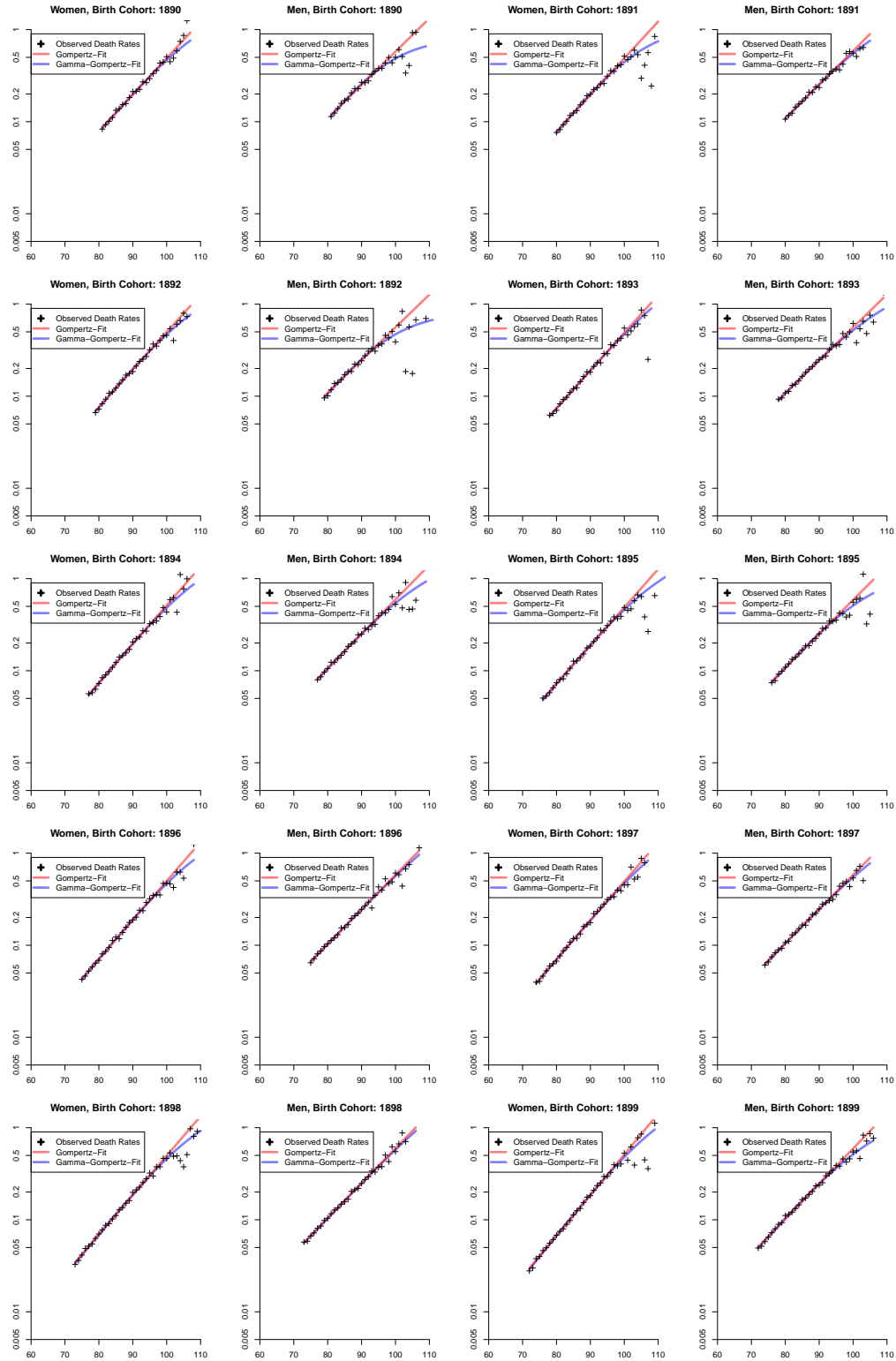


Figure 1: Observed Death Rates (+) and Parametric Fits (Gompertz, Gamma-Gompertz) by Sex for Swedish Birth Cohorts 1890–1899

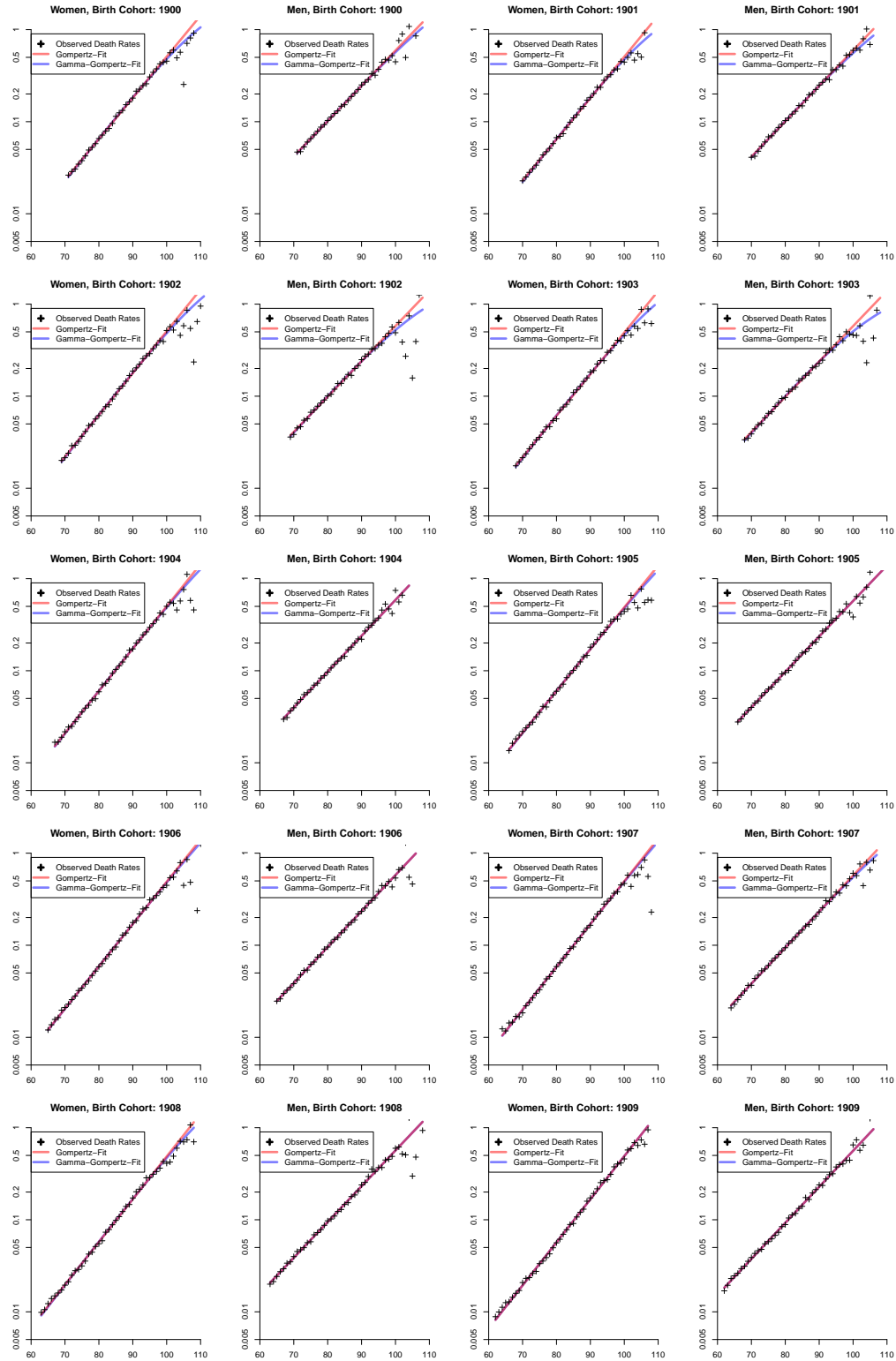


Figure 2: Observed Death Rates (+) and Parametric Fits (Gompertz, Gamma-Gompertz) by Sex for Swedish Birth Cohorts 1900–1909

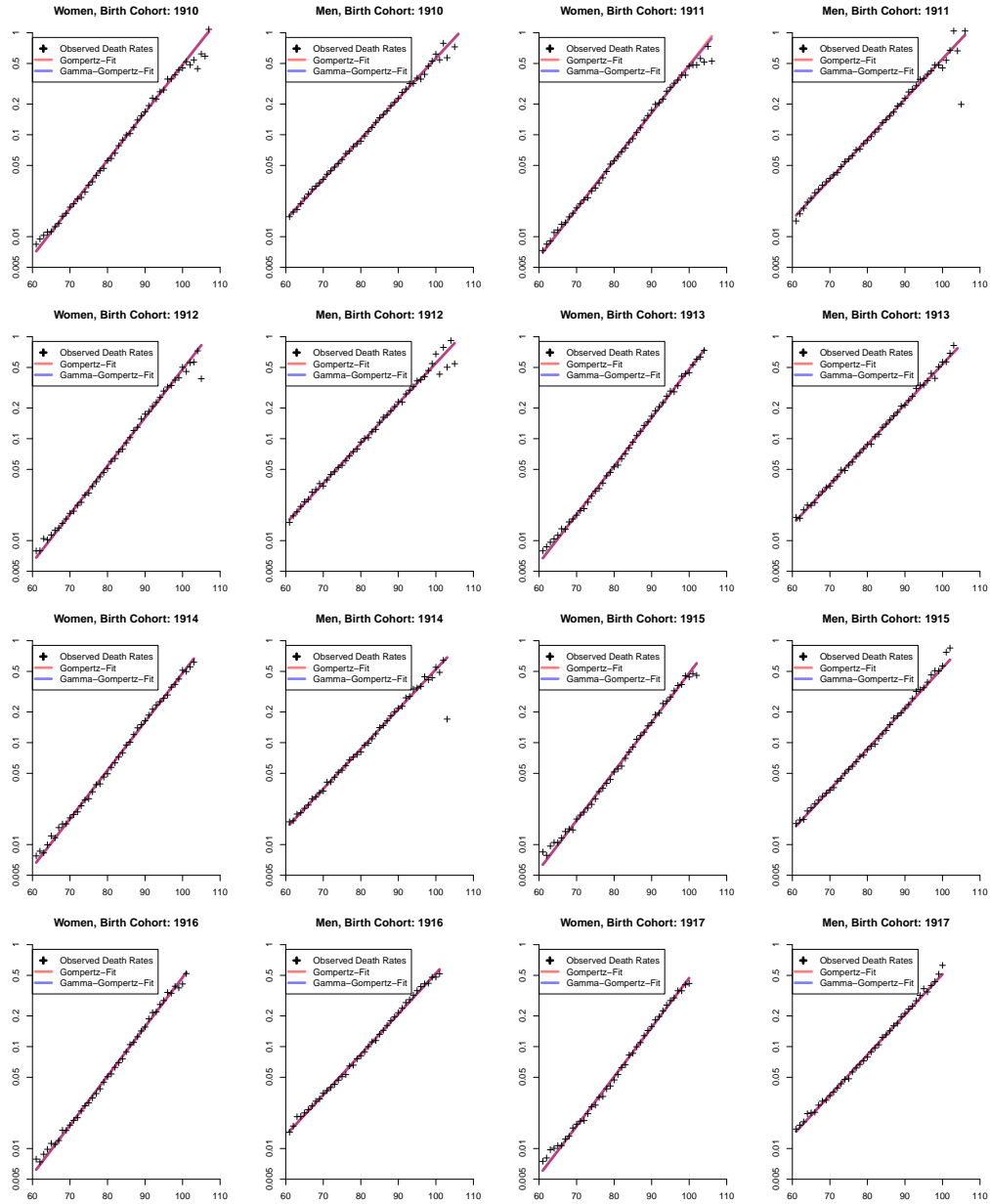


Figure 3: Observed Death Rates (+) and Parametric Fits (Gompertz, Gamma-Gompertz) by Sex for Swedish Birth Cohorts 1910-1917

Estimated variance, γ , in the Gamma-Gompertz Models

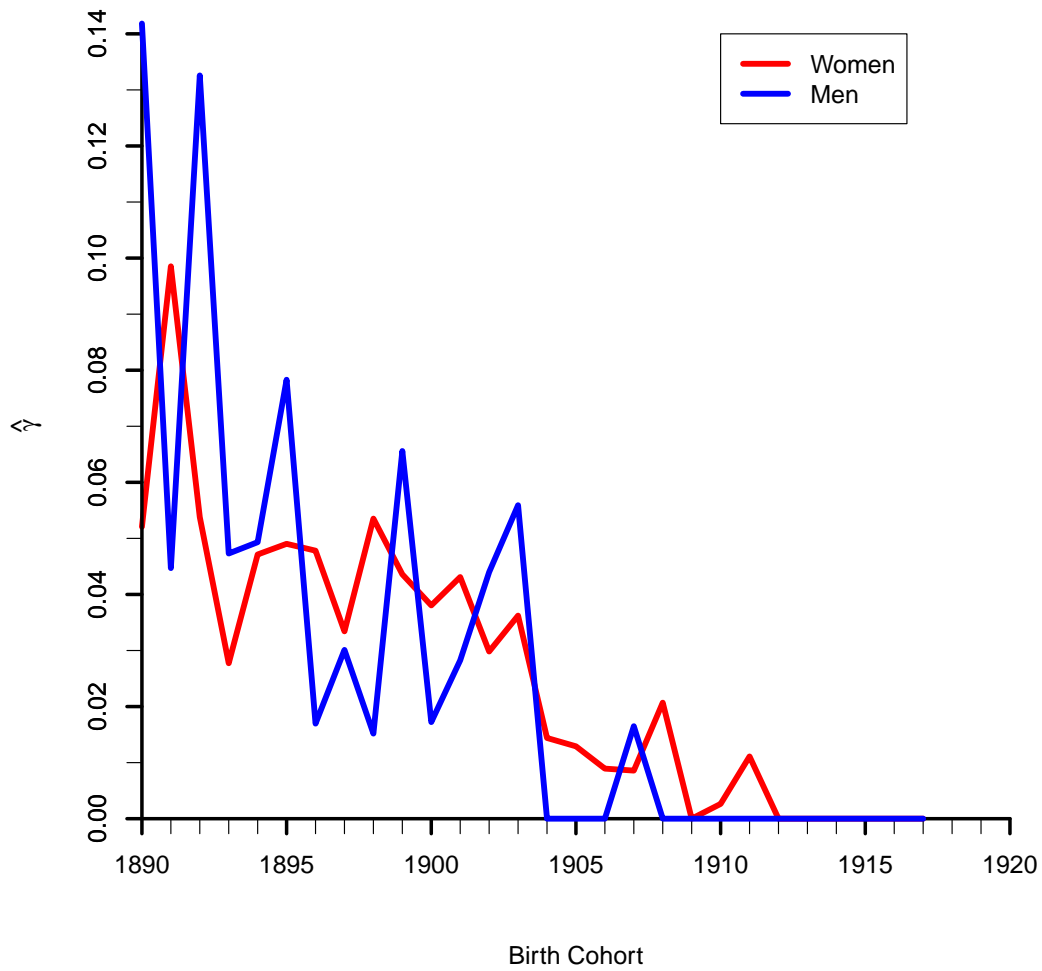


Figure 4: Estimated variance, γ in the Gamma-Gompertz Models for Women and Men of Birth Cohorts 1890–1917