Inferring mortality deceleration patterns from a gamma-Gompertz-Makeham framework (Extended Abstract)

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Abstract

We calculated life-table aging rates (LARs) for overall and cause-specific mortality by estimating a gamma-Gompertz-Makeham (ΓGM) model and taking advantage of LAR's parametric representation by Vaupel and Zhang (2010). For different HMD countries we study how the evolution of estimated LAR patterns (for all or specific causes of death) could explain observed 1) life expectancy dynamics, and 2) mortality improvement or deterioration at different ages. We compare our findings across countries (data from HMD) and major causes of death (data from WHO).

Preliminary results reveal that ΓGM model-based LAR fits well the observed LAR, capturing simultaneously, a shift in the age of mortality deceleration with time. Across the studied countries and between sexes, it's also identifiable different ages of mortality deceleration, what suggests a connection between the rate of life expectancy increase and the estimated LARs.

Summing up, we intend with this study answering the following questions: 1) can we identify a similar pattern across countries, sexes and CODs? 2) can ages of mortality deceleration be associated with the pace of life expectancy increase? 3) speaking statistically, does the LAR approximation of Vaupel and Zhang (2010) provides a good fit to the observed LAR?, and 4) can we find a more pronounced pattern according to different CODs?

Background and aim of the study

Horiuchi and Coale (1990) proposed a mortality measure, called later the life-table aging rate (LAR), defined as

$$\bar{b}(x) = \frac{1}{\mu_x}\frac{d\mu_x}{dx} = \frac{d\ln(\mu_x)}{dx} \qquad (1)$$

and measures the rate of aging for the population (pRoA) at age x (μ_x denotes the population's hazard). The individual rate of aging (iRoA), defined as the relative derivative of the baseline hazard of death in a given survival model, is a different characteristic. In many models for adult mortality, the iRoA is assumed to be constant.

Gampe (2010) acknowledge the existence of a possible plateau at ages 110–114, which speaks in favor of a mortality model with a Gompertz-Makeham baseline $\mu_x = ae^{bx} + c$ (Gompertz, 1825 & Makeham, 1860), where *a* measures the mortality level at the starting adult age *x*, *b* is the iRoA itself, and *c* captures the risk of dying that is not associated with the aging process. If in addition,

unobserved heterogeneity is modeled by a gamma distribution with a unit mean and γ variance (Vaupel, Manton & Stallard, 1979), the population's hazard μ_x equals

$$\mu_x = \frac{a\, e^{bx}}{1+\frac{\gamma a}{b}\left(e^{bx}-1\right)} + c. \quad (2)$$

The method itself is known as the gamma-Gompertz-Makeham (ΓGM) frailty model. If we estimate its parameters *a*, *b*, *c* and γ, we can take advantage of the LAR representation by Vaupel and Zhang (2010), applying:

$$\bar{b}(x) = b\left(1-\frac{c}{\mu_x}\right) - \gamma\left(1-\frac{c}{\mu_x}\right)(\mu_x - c). \quad (3)$$

Previous research (Horiuchi, Cheung and Robine, 2012) focused on reconstructing model-based LARs by fitting a Kannisto model for homogeneous populations. In this paper we focus on a ΓGM heterogeneous model to reflect the perception that populations consist of individuals that share the same baseline hazard, to which they are susceptible in a different (random) way.

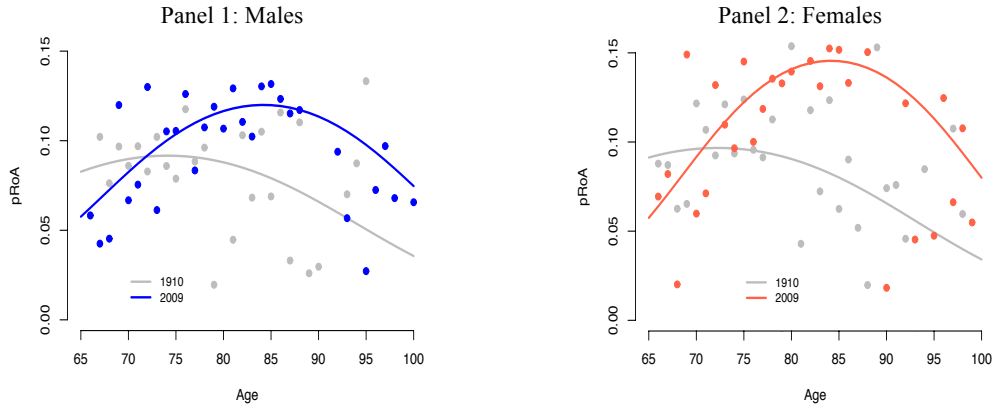
In step 1, we fit the ΓGM model to four countries: two from Southern Europe (Spain and Portugal), one from Western Europe (France), and one Nordic (Sweden). Our choice was made based on the qualitatively different patterns of life expectancy evolution over time: 1) France and Sweden, registering a high life expectancy at birth already in the 1950s, but the rate of increase dropped in the following decades; 2) Spain and Portugal experienced a low life expectancy at birth in the 1950s, but the rates of increase surpassed the life expectancy leaders at the time and the two countries caught up (the values of life expectancy in Spain even surpassed the ones registered lately for Sweden).

In step 2, we elaborate on the relationship between the estimated LARs and 1) the rate of life expectancy increase in the chosen countries; and 2) the age patterns of mortality deceleration — not only in the overall population, but also across causes of death (COD). At the same time we also test the goodness of fit of the LAR formula by Vaupel and Zhang (2010). One more point to verify would be the heterogeneity hypothesis by Horiuchi and Wilmoth (1998) that a) deceleration occurs for the most major CODs, being less pronounced for CODs with lower death rates; and b) mortality deceleration should occur at later ages due to selection effects. Depending on data availability, we focus on the last 100 years of mortality history, but always taking into account the limitations (availability and time range) associated with COD data. For the selected countries, and for each year in the available period for each country, for overall and COD data, we estimate the LAR by fitting a ΓGM model beyond age 65. To estimate the LAR for the overall and cause-specific mortality, the ΓGM frailty model will be fitted to our data by maximizing a Poisson log-likelihood for the death counts. We use data for the overall mortality from the *Human Mortality Database* (HMD: www.mortality.org) and cause of death data from the *World Health Organization* (WHO) mortality database.

Preliminary results

The preliminary results for France, presented in *figure 1* indicate that, as expected, the ΓGM model-based LAR is fitting well observed LAR (calculated from life-table mortality rates). At the same time, it captures the observed shift in the age of mortality deceleration between 1910 and 2009, for both sexes, and it seems to confirm point b) of the heterogeneity hypothesis: with increasing lifespans, mortality deceleration shifts and occurs always at older ages.

Figure 1: Empirical and model-based population rate of aging (pRoA) for France



Results presented in *figure 2* appear support what was stated before in the analysis of *figure 1* about the point b) of the heterogeneity hypothesis. Model-based LAR for Spain presents a shift in the age of mortality deceleration to older ages with time, and that is transversal to both sexes. From these results, it also seems that in the case of females, deceleration starts at older ages when compared with the male case, possibly being connected with the life expectancy gap registered between both sexes. Nevertheless, these are only preliminary results, and it is still soon to assure a precise elaboration about the connection between life expectancy and the age of mortality deceleration, and that the observed results will be repeated when we present the results for other countries and all observed years.

Figure 2: Model-based population rate of aging (pRoA) for Spain

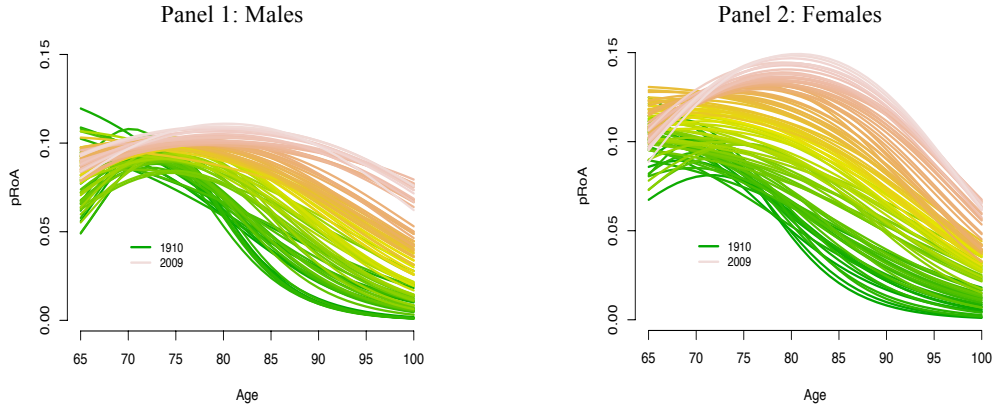
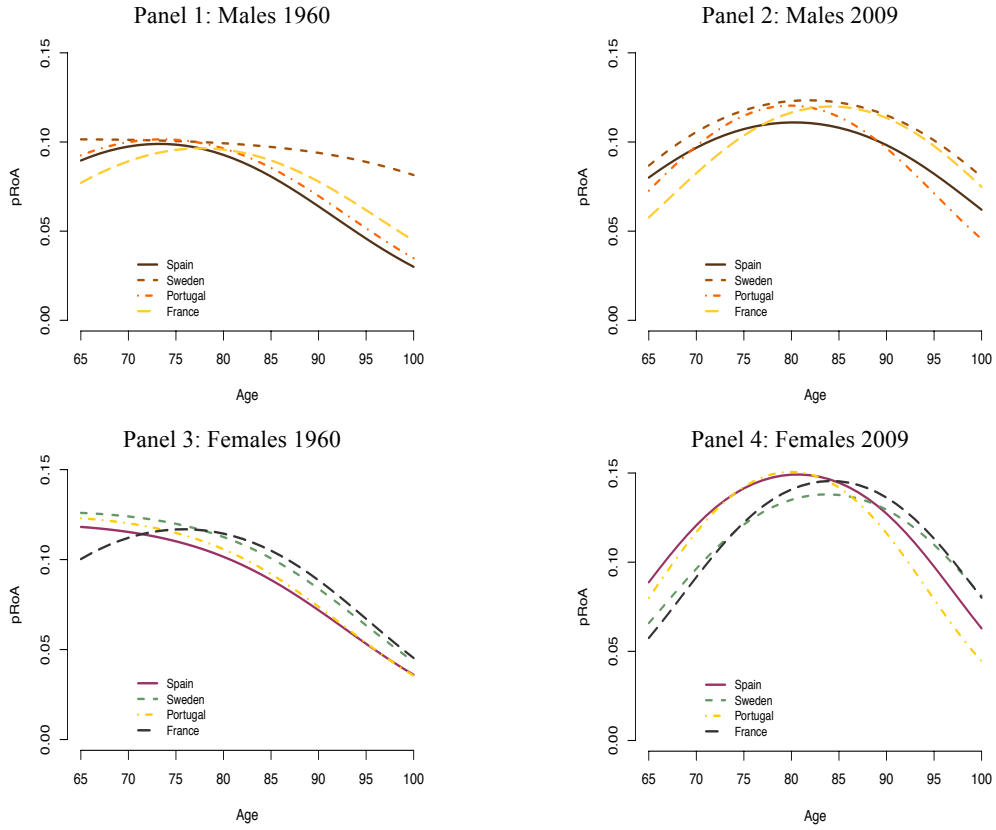


Figure 3, presents the preliminary results obtained for the four studied countries, and, at a first glance, it seems that the shift registered in mortality deceleration for France is transversal to all the other three countries, independently of the sex. However, these preliminary results also show that despite the presentation of a similar pattern in the evolution of LAR across countries and sexes, the age of mortality decrease appear to be different in each of them. This might mean that life expectancy can be related to the evolution of the age of mortality deceleration. Can we affirm that the pace of increase in life expectancy can be captured by the age of mortality deceleration? For the time being we don't have an answer, but this is one of the questions that we would like to approach in this piece of research.

Figure 3: Model-based population rate of aging (pRoA) by sex and country



Conclusion

Preliminary results show that ΓGM model-based LAR fits well observed LAR and at the same time that lifespan is increasing, mortality deceleration appears to occur always at later ages. However, presented results reflect overall population, for which we have a large number of observations in each age group. For COD data we might run into problems associated with low sample sizes, despite the fact that COD data are organized by five-year age groups, which might influence ΓGM parameter estimates. On the other hand, disaggregation of population data by causes of death, will aid understanding better the possible existence and strength of the relationship between LAR patterns and life expectancy dynamics across countries and by gender. Finally, in the next step, we intend to test point a) in the heterogeneity hypothesis.

Finalizing, this study aims to answering the following questions: 1) can we identify a similar pattern across countries, sexes and CODs? 2) can ages of mortality deceleration be associated with the pace of life expectancy increase? 3) speaking statistically, does the LAR approximation of Vaupel and Zhang (2010) provides a good fit to the observed LAR? ?, and 4) can we find a more pronounced pattern according to different CODs?

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