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Trends in the Quantiles of the Life Table Survivorship Function

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Abstract: We offer a new approach for modeling past trends in the quantiles of the life table survivorship function. Trends in the quantiles are estimated, and the extent to which the observed patterns fit the unit root hypothesis or, alternatively, an innovative outlier model, are conducted. Then a factor model is applied to the de-trended data and it is used to construct quantile cycles. We enrich the on-going discussion about human longevity extension by calculating specific improvements in the distribution of the survivorship function, across its full range, and not only at the central-age ranges. To illustrate our proposal, we use data for the United Kingdom from 1922 to 2013. We find that there is no sign in the data of any reduction in the pace of longevity extension during the last decades.

Keywords Survivorship function quantiles, longevity extension, structural breaks, population aging, longevity risk.

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1 Introduction

The study of human longevity has traditionally focused on the estimation of central tendencies in the data describing mortality. For instance, Riley (2001) documents a significant increment in the life expectancy at birth around the world, from 1800 to 2000, by over 30 years. Wilmoth (1998), Oeppen and Vaupel (2002) and White (2002) also use central tendencies to argue in favor of an unstoppable pace of longevity extension, during the last century.

Nevertheless, although the recorded significant increments in human life expectancy in recent decades are not under discussion, many authors have cast doubts on the ability of life expectancy to accurately capture all the relevant information on the tendencies of lifespan distributions and, therefore, have focused on analyzing other distribution features. For example, Bongaarts and Feeney (2002, 2003), Bongaarts (2005a) show that traditional period life expectancy is subject to biases when mortality rates are changing during the sample period, and therefore, it constitutes a non-fully satisfactory measure of longevity. Edwards and Tuljapurkar (2005) point to inequality in adult lifespan as the main driver of divergence in mortality around the world. Such lifespan inequality may become a feature of future demographic change, if mortality continues to stagnate at young ages while declining steadily at old ages, as suggested by Guillespie et al. (2014). Nevertheless, Vaupel et al. (2011) show that reducing life disparity and increasing life expectancy, are not incompatible objectives and, indeed, the countries that have been the most successful in reducing life disparities have consistently been the life expectancy leaders[1].

Other examples in the literature, which have turned to explore and analyze different measures of human longevity, on top of the traditional life expectancy, include the study of the modal and the median ages at death as suggested by Canudas-Romo (2008, 2010) or Horiuchi et al. (2013); or measures that explicitly focus on characterizing the variability in the distributions of lifespans as in Engelman et al. (2014).

We propose an alternative methodological framework, useful to understand the trend of the shape of the life table survivorship function, on a wider basis than those allowed by the existing literature. Our model is by construction sensible enough as to distinguish the potential asymmetries that may exist, in the temporal reduction of specific mortality rates at different ages. We argue that in order to obtain a fuller understanding of the changes in the distribution of human lifespan over the last century, and of those that it seems likely to undergo in forthcoming decades, we need to turn to more general procedures that extend the analysis beyond that of the central tendencies and the dispersion around them.

In this paper, we propose the means for constructing these measures, based on quantiles, and undertake a rigorous study of the features of these statistical constructs. This analysis allows us to shed light on the mechanisms of the dynamics of human longevity. In this way, we also present a new contribution to the controversy concerning whether the rate of improvement in human longevity[2] can be considered constant as stated by Oeppen and Vaupel (2002) and White (2002) or decreasing, and probably bounded, as suggested by Vallin and Meslé (2010), King and Soneji (2011), Mayhew and Smith (2015) and very recently by Dong, et al. (2016) and Olshansky (2016).

Our methodology is able to encapsulate in a single framework many of the features of the mortality shifting dynamics, without resorting to indirect analyses on the interaction between different statistics of human longevity, as is commonly done (see for example Canudas-Romo (2008, 2010) or Edwards and Tuljapukar (2005)); or comparisons between the tempo-adjusted measures of human longevity and the non-adjusted measures (Bongaarts, 2005a).

The study of the asymmetries in the dynamics of the survivorship functions is important, because they affect the full range of the lifespan distribution. For instance, Wilmoth et al. (2000), Rau et al. (2008) and Engelman et al. (2014) provide a detailed quantification of the impact of changing demographic parameters on the pattern of lifespan trends and variability. The former authors highlight the importance of the reduction in death rates at old ages in the increments in life expectancy witnessed during the last century, especially above age 80. Engelman et al. (2014) document the importance of declining childhood mortality on the reduction of the general lifespan variability, and the impact of improved survival in adulthood on the rising variability of lifespans at older ages. In the same strand of the literature, aiming to understand the hidden imbalances in the temporal improvements of mortality at different ages, van-Raalte and Caswell (2013) provide estimates of the sensitivity of several indices of lifespan variability to changes in underlying

[1] This is arguably also true within populations. For instance, by education is possible to increase life expectancy while reducing the variance of life expectancy between groups.

[2] That is, the growth rate at which life expectancy and related statistics increase, following reductions in specific mortality rates.

mortality. Other authors have explored alternative avenues to document hidden asymmetries in life expectancy. For example, based on a decomposition of specific mortality rates into natural and accidental rates, Guillén and Vidiella-i-Anguera (2005) show that constructing counterfactual scenarios, eliminating one mortality cause at a time, leads to a better understating of the influence of external causes of mortality in the forecast of life expectancy. Finally Basellini and Camarda (2016) use age at death distributions to model the age-specific pattern of mortality and to inform mortality forecasts. This approach allows them to capture the compression and shifting dynamics of mortality.

The model we propose is based on the quantiles of the distribution of the life table survivorship function. A quantile indicates an age at which only a fraction of the population survives; for instance, if the 95th quantile[3] is equal to age x, then only 5% of the population lives more than x years. A quantile is defined for a given level, here 95%. The analysis of quantiles allows us to focus directly on the asymmetries recorded at different age-ranges of the distribution, without resorting to comparisons among central tendencies, or among central tendencies and variability measures. It provides an alternative path to traditional analyses in which the trend of a central tendency is calculated, and then decomposed by age (or age and cause) to attribute the change to specific rates. In the demographic literature is common to see trend lines of age-specific rates, or full-on Lexis surfaces of rates, or full-on Lexis surfaces of changes in rates, among others. These calculations allow the exercise of intuitive and practical relationships between the preferred central tendency measures and age-specific patterns, which are intuitive to the general public. Our methodology aims at complementing this traditional approach by directly analyzing quantiles of life tables[4].

In addition to constructing the empirical quantiles, our methodology identifies trends in the series of quantiles, and the specification of alternative hypotheses, which imply the estimation of endogenous structural breaks affecting the temporal trends of the series. We complement the deterministic model of the lifespan distribution, by adding uncertainty to the otherwise deterministic projections by means of a factor model, which is fundamental in demography.

Our methods and results have a practical application to the analysis of the viability of pension schemes, but the tools we propose should be of general interest for describing and understanding human longevity extension, and the challenges faced by an aging population. This challenge is an important one and consequently has received considerable attention in the fields of economics and demography during the last years. For example, Waite (2004) discusses the consequences of an aging world, in terms of the societal transformations that must be pursued if we want to be prepared for a world in which one out of five people will be above 65 years, and a high percentage of those, will be above 85 years. She states that these oldest-old men and women are much more likely than the young-old to live in nursing houses, to have substantial disabilities, and to have lack of financial resources. Moreover, she argues that the number of older adults to be expected in the future gives us valuable information about how many hospital beds, geriatricians, home health aides, and nursing home beds will be required. Our model enables us to advance one step forward in the comprehension of the dynamics of longevity extension by providing, by the first time, novel measures based on quantiles of this phenomenon.

The analysis addressed in this article is especially relevant for studying longevity risk. Risk managers of pension funds and financial institutions offering life annuities are interested in projecting and analyzing the trends for fractions of the cohorts that survive more than the expected.

In our empirical section we study the time-series properties of each quantile recorded over a nine-decade period for the United Kingdom (men and woman, from 1922 to 2013). We find that most survivorship quantiles are better described by deterministic trends subject to random breaks in specific periods than by stochastic trends with drifts. Because of the dramatic fall in infant mortality rates at the beginning of our sample (1922-1940), we found greater improvements in that period for the lowest quantiles than we did during the last seven decades of the sample. This finding is in line with the outcomes reported by Vallin and Meslé (2010). However, we find no sign of a reduction in the rate at which population aging and longevity extension are occurring; indeed, from our model, we can expect further improvements at the different quantiles of the duration of life. We can also expect the continuing aging of the population, mainly as a consequence of a greater number of people reaching more advanced ages (above 65 or 85 years), but also due

[3] If we split the population into 100 quantiles, the quantile equals the percentile.

[4] Notice that our work also shares a certain relationship with the studies by Sanderson and Scherbov (2005, 2007) and Lutz et al. (2008). They relate age and life expectancy at different ages. That is, they use the full age pattern of life expectancy, a conditional life expectancy, which is more informative than using just a single instance of central tendency. The amount of information returned by their procedure or ours is roughly the same, as it allows to analyze longevity extension in a more comprehensive fashion than central tendencies do.

to increments in the duration of life at the highest quantiles (at which most of the population would expect to be dead).

The rest of this paper is organized as follows. The second section presents the data and methodology, and includes a description of the construction of the quantiles, an explanation of the time-series tools needed to distinguish between stochastic and deterministic trends in the data. The third section presents the results; the fourth section discusses these findings and section five draws conclusions and limitations of our study.

2 Data and Methods

The data for the empirical section in this piece of research were taken from the Human Mortality Database. We used the annual mortality rates up to 110 years, for males and females, from the United Kingdom (m_x). Our full sample spans the period from 1922 to 2013, that is, a total of 92 calendar years.

Our methodology involves the following two steps: first, we transform traditional mortality surfaces, composed of period mortality rates discriminated by sex and age, into the survivorship function, and we determine its quantiles. Second, we assess whether the constructs are best described by stochastic or deterministic trends, and we explore the possible presence of structural breaks in these trends. Third, we conduct structural change test on the estimated linear trends of the quartiles. Below we provide further details for each step.

2.1 Measures of the Duration of Life

Traditional analyses of human lifespan have been based primarily on the estimation of central tendencies in the data. Statistics such as the median, the mean and the mode of the lifespan distribution have dominated practices since the first demographic and actuarial research agendas were drawn up. Today, however, the importance of incorporating measures that can describe the dispersion of the data around a given central point is widely recognized, and the sensitivity of such measures to the underlying mortality scheme has been explored. And, yet, focusing solely on the measurement of central tendencies and dispersion leaves critical aspects of mortality and longevity patterns unexplored. This can be corrected by decomposing the original trends into cause and age-specific mortality rates or by analyzing the full spectrum of the life table quantiles as we propose here.

We construct period life tables on the basis of central death rates. Thus m_{xt} are mortality rates at age x and for period t. Let d_{xt} denote the number of deaths at age x in period t, and l_{xt} be the number of individuals alive at age x in period t (i.e. $l_{0t} = P$). Traditional central tendencies of the data can be constructed in this way, including period life expectancy (e_{0t}), modal age at death ($m_t = \{x|\max[d_{xt}] \text{ for } x > 10\}$) and median age at death ($\mathcal{M}_t$). The latter is the age at which half the population has died; that is, when the survival function is equal to one half. It also represents a specific case for the quantiles of the distribution of human longevity, which can be defined as:

$$Q_t^{\alpha} = \{x|\ell_x = 1 - \alpha\}, \;\; \alpha \in (0,1). \tag{1}$$

Thus, for a given level α, Q_t^{α} is the age at which a fraction $(1 - \alpha)$ of the initial population is still alive. Conversely, a proportion equal to α is dead at age Q_t^{α}. Another way to think of the quantiles is as the cumulative deaths function, or complement of the life table survivorship function.

2.2 Deterministic or Stochastic Trends: Allowing for Endogenous Structural Breaks

Demographic phenomena inherit uncertainty and therefore statistical demography should always account for the stochasticity process behind them. Although that is clearly correct, we stress out here that there are different ways to add stochasticity into the modeling process. For example, one can assume that mortality rates (or survivorship quantiles, as in our case) present unit roots (i.e. stochastic trends) and therefore any shock affecting them persists in time without vanishing. In this case, quantiles (or rates) should be understood as the aggregation of an infinite number of random shocks (perhaps white noise shocks).

On the other hand, stochasticity can be added as a complement to a deterministic trend and, in this case, what we face is a statistical process composed by three parts: a line, a stationary component that is clearly stochastic, and random noise. Both processes (those with stochastic or deterministic trends) reflect the stochastic nature of demographic phenomena, but they do not imply the same for the analysis of mortality and

longevity. In the latter case most of the shocks always vanish after some periods, while other shocks are able to produce permanent shifts in the trajectory of the survivorship quantiles (what we call structural changes). We content that one should not assume that trends are either stochastic or deterministic, and permanent breaks are either present or absent, but this needs to be tested in an appropriate fashion.

In seeking to acknowledge these alternatives and in order to correct the tests so as to provide reliable inferences about the presence or absence of unit roots, various proposals have been made in the time-series literature. In this regard Kim and Perron's (2009) proposal is particularly appealing, because it allows for a single break in the series under both the null and the alternative hypotheses. In the demographic literature, a single break seems sufficient for the description of mortality series in several countries (Ouellette et al. 2014)[5]

Below, we present the models used to test for the presence or absence of unit roots in survivorship quantiles and for breaks in the linear trends of these quantiles. Equations 2 and 3 correspond to what is called an innovative outlier (IO) model. Equation 2 is the null hypothesis of the test and equation 3 is the alternative hypothesis. IO models are well suited to deal with processes that have experienced gradual breaks, that is, for which the full effects are only evident after a certain amount of time has elapsed. Notice that in case of a null rejection the series would be better described by a deterministic trend plus random breaks, while under the null it would be better to fit a stochastic trend to the data.

Under the null hypothesis, the data generating processes are given by:

$$Q_t^{\alpha} = \mu^{\alpha} + q_{t-1}^{\alpha} + \psi^{\alpha *}(L)\left[I_{1,t}^{\alpha}\mu_b^{\alpha} + I_{2,t}^{\alpha}\beta_b^{\alpha} + \varepsilon_t^{\alpha}\right], \quad \alpha \in (0,1), \tag{2}$$

where $I_{1,t}^{\alpha} = 1$ if $t = T_1 + 1$ and 0 otherwise; $I_{2,t}^{\alpha} = 0$ if $t \leq T_1$ and 1 if $t > T_1$. $\psi^{\alpha *}(L)$ are lag polynomials in L of order p and they satisfy regular conditions of stationarity and invertibility (see Kim and Perron (2009) for further details). Sub-index b indicates the change in the parameter due to the structural break.

Under the alternative hypothesis, we have that:

$$Q_t^{\alpha} = \mu^{\alpha} + \beta^{\alpha} t + \psi^{\alpha}(L)\left[I_{1,t}^{\alpha}\mu_b^{\alpha} + I_{2,t}^{\alpha}\beta_b^{\alpha} + \varepsilon_t^{\alpha}\right], \quad \alpha \in (0,1). \tag{3}$$

in our empirical exercise we set $\alpha \in \{0.1, 0.15 \ldots 0.9, 0.95\}$.

An additional consideration here concerns the fact that the break date T_1 can be estimated using different procedures. We follow Perron and Zhu (2005), who propose estimating the unknown dates in a model composed of a deterministic trend with breaks by minimizing the sum of squared residuals in simple recursive regressions. The breaks in the trend may be due either to a change in the slope or to a shift in the level of the model. Our approach is very much in line with that taken by Ouellette et al. (2014), who use a criterion of maximizing the R^2 of the model in each regression to estimate the dates of the breaks.[6]

2.3 Factor Models

Factor models constitute an attractive approach for modeling and projecting demographic data. They allow reducing the dimensionality of the original set and constructing accurate projections. The general strategy involves making the time series dependent on just a few unobserved stochastic factors, extracting these factors using proper techniques (such as principal components), and using the estimated factors in subsequent steps. In so doing, the number of estimated parameters in the model is significantly reduced and optimal forecasts are feasible. Examples of mortality data modeled using factor models are abundant in the actuarial and demographic fields, most notably with the model constructed by Lee and Carter (1992) and the multiple extensions and applications of it, by Brouhns and Denuit (2002), Guillén and Vidiella-i-Anguera (2005), Delwarde et al. (2006), Shang et al. (2011), Stoeldraijer et al. (2013), Lemoine (2014), among many others.

Here, we fit the factor model to the stationary component of the quantiles; that is, we model each quantile using models composed of deterministic linear trends with breaks, plus an intercept (as in equation 3). The

5 Pre-testing for unit roots and cointegration is necessary to avoid under- or over-differentiation of the series. Indeed, the time-series literature in the demographic and actuarial fields includes recent studies of unit root pretesting, cointegration tests and structural breaks (D'Amato et al. 2014; Gaille and Sherris 2011; Torri 2011; Niu and Melenberg 2014; Ouellette et al. 2014). As for the specific task of projecting mortality surfaces, various studies explore the differences between differentiating and using the series in levels with key implications for the forecasting accuracy achieved by the models (Mitchell et al. 2013; Chuliá et al. 2016).

6 An alternative approach would be considering joint distributions of the quantiles series, which would require substantial modifications to the tests employed here to assess time series properties of these constructs. We leave this for future research.

break dates are estimated using the approach described in section 2.2. Afterwards, we extract the residuals of each model (which already includes the breaks) and label it c_t^{α}. We may think of c_t^{α}as the cyclical component of the quantile series. This cyclical component describes the stochastic dynamics of the quantile series around a deterministic trend plus breaks. In this sense, it represents shocks to the system that lack a permanent impact on the quantile series. See Appendix (section A.1.) for more details about the factor model used in this step.

3 Results

3.1 Quantiles of the Survivorship Function

Figure 1 shows the smoothed distribution of human lifespan in the UK for ages ranging from 0 to 110 for different years. We used B-splines, as recommended by Ouellette and Bourbeau (2011). Alternatively, a parametric distribution can be fitted to the data, such as that proposed by Robertson and Allison (2012). These authors show that a Gaussian distribution, whose scale parameter decreases linearly with attained age (that is, a compressed Normal distribution) is a good proxy for more than 74 life tables from 35 countries. Similarly, Beer (2012) discusses different alternatives to the one used here.

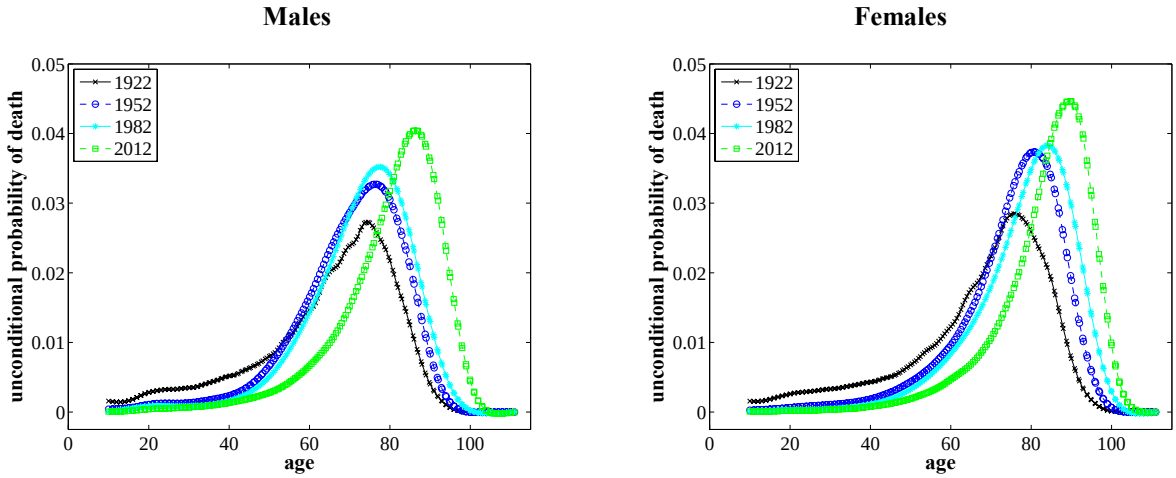

Fig. 1 Life table deaths distribution of the United Kingdom (1922-2013) for males and females smoothed using B-splines. We construct the lines of ages at death for the life tables corresponding to 1922, 1952, 1982 and 2012. We use the mortality rates from the United Kingdom for males (left) and females (right). It can be seen that the distributions have shifted to the right with time, albeit to a greater extent in the left tail than in the right tail. Data source: Human Mortality Database.

Fig. 1 shows that in the last century the lifespan distribution has shifted to the right. This has been documented in the literature for many countries, especially economically developed ones (Riley 2001). The effects of this shift in the distribution on the traditional measurement of the central tendencies of life span, including life expectancy, median duration of life, and modal age at death, have also been explored. Thus, several studies have examined how improvements in specific age ranges of the distribution have impacted human longevity, for instance, Wilmoth (2000), Wilmoth et al. (2000), Canudas-Romo (2010) and Rau et al. (2008). These studies have provided some indications regarding the fact that the left tail of the distribution has shifted further to the right than the right tail.

In order to explore the phenomenon of the asymmetric improvements in longevity in greater depth, we set α at different levels between 10 and 95%; that is, we study quantiles of longevity $\{0.1, 0.15 \ldots 0.9, 0.95\}$ for a total of 18 quantiles. We excluded $\alpha = 0.05$, because before 1946, more than 5% of the population had died before the age of one year. We also calculate period life expectancy, the median of lifespan, and the modal age at death.

In Fig. 2 and 3 we present the central tendencies of the data and the estimated quantiles, respectively.

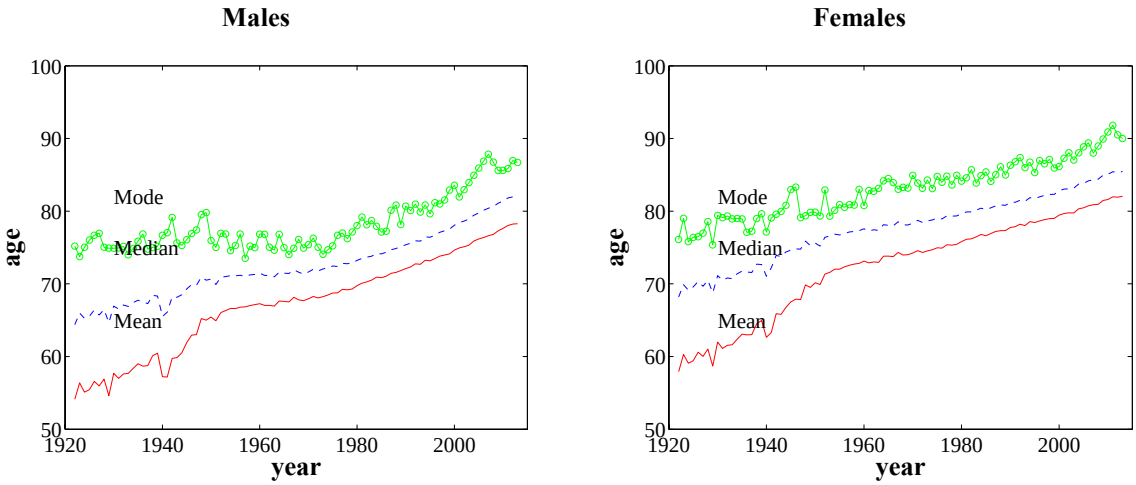

Fig. 2 Evolution of median age at death, modal age at death and life expectancy in the United Kingdom (1922-2013). The calculations are based on the mortality rates in the United Kingdom for males (left) and females (right) (1922- 2013).

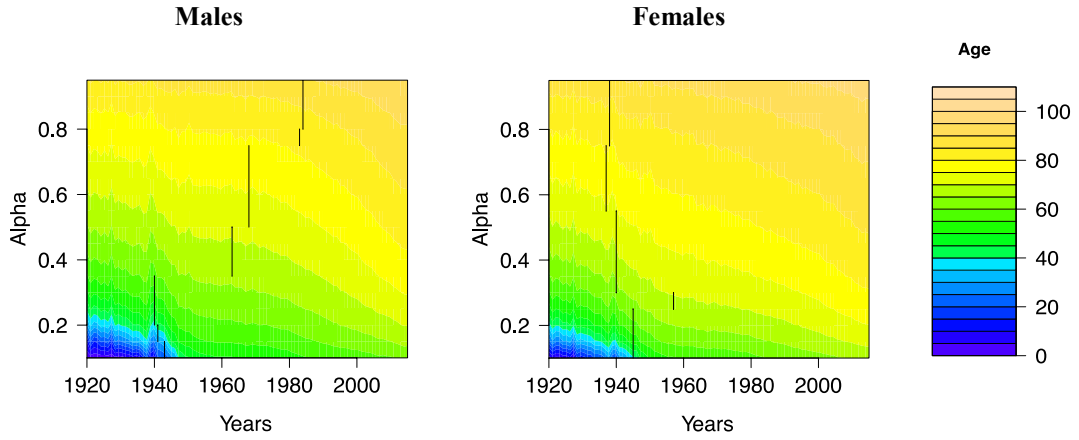

Fig. 3 Evolution of the age-quantiles (1922-2013). Note: We use the UK's mortality rates for males and females. We estimated the quantiles as $Q_t^{\alpha} = \{x|\ell(l_{xt}/l_{0t}) = \alpha\}$, $\alpha \in \{0.1, \ldots, 0.9, 0.95\}$. The black lines show the dates of the estimated structural breaks. These dates are presented in Table A.3 of the appendix.

A given quantile of the survivorship function will increase as mortality improvements are recorded at a lower age. For this reason, we see parallel increments in the quantile surfaces (each quantile becomes clearer in time). Nevertheless, by construction, lower quantiles (below a certain age at which a mortality reduction is observed) are insensitive to improvements at higher ages and, even if the quantiles measures are clearly dependent, they still are able to reflect mortality idiosyncratic improvements at different ages in the mortality surface. And as such, they offer richer information about the distribution of human lifespan than traditional statistics such as the mean or the mode alone.

In line with the findings in previous studies, for developed countries, for instance Canudas-Romo (2008, 2010) the three central tendencies have experienced significant increments over the last century both for males and for females. The median life duration for females has increased from 68.20 years in 1922 to 85.45 years in 2013 (that is, 17.25 years over the nine decades in our sample). In terms of life expectancy, the increment for females has been even more pronounced, rising from 57.93 to 82.03 years (a difference of 24.10 years). Finally, the modal age at death for females has also increased, but in a significantly smaller magnitude, rising from 76.11 to 89.99 years (a difference of 13.88 years). The general trend for males is similar. In this case, the median has increased from 64.39 to 82.03 (17.64 years), life expectancy from 54.12 to 78.27 (24.15 years), and the modal age at death from 75.19 to 86.71 (11.52).

Figure 3 shows the evolution of the survivorship quantiles at different levels. The evolution of the first quantile in the female population sample (10th percentile of human longevity) shows that in 1922 while 10% of the population could expect to die by the age of 2.61 years, 92 years later the same percentage of the population would not, on average, be dead until 66.60 years. This dramatic change, slightly lower than 65 years, stresses the impact of improvements in infant mortality. The evolution of the last quantile (95th percentile) in our sample shows that in 1922 while 95% of the female population could expect to have died by the age of 86.99 years, nine decades later this threshold had increased to 97.35 years. Although significant, this increment of 10.36 years in the 95th percentile is not comparable to that recorded by the 10th percentile. However, the improvement recorded in the 95th percentile is not very different from those observed in the 70th and 90th percentiles (from 76.00 to 90.01 years, and from 84.05 to 95.21 years, respectively, that is 14.11 and 11.16 years of improvements). A similar trend is recorded for the male population.

It is also evident that the increment in the modal age at death is not attributable solely to improvements in the most advanced age categories but also to small improvements in all age categories. Thus, not only do the eldest of the old live longer, but also more individuals now die at these advanced ages, as highlighted by Wilmoth et al. (2000) and Rau et al. (2008).

3.2 Stochastic Trends against Deterministic Trends

The analysis can be further refined by studying the stochastic properties of the processes describing each quantile over time. In the Appendix (section A.2.) we present the results of the unit root tests for each quantile of the survivorship function. We test two alternative hypotheses: intercepts and intercepts and linear trends. The series of quantiles in almost all the cases are better described by a linear model with a one-time change in slope, than by a unit root process (stochastic trend). What is means is that, indeed, there are signs of acceleration or deceleration (depending on the sign of the rotation) in the rate of improvements for these quantiles, but such changes are one-time breaks during the whole sample. Once these *big shocks* have impacted mortality rates, subsequent shocks lack a permanent or cumulative effect on the quantile-trends of the lifespan distribution.

We estimate the break dates (T_1^α) and construct an indicator variable, $I_{3,t}^\alpha$, such that: $I_{3,t}^\alpha = 1$ if $t \leq T_1^\alpha$ and 0 if $t > T_1^\alpha$, as in the following equation:

$$Q_t^\alpha = \mu^\alpha + \beta^\alpha t + \mu_b^\alpha I_{3,t}^\alpha + \beta_b^\alpha I_{3,t}^\alpha \, t + c_t^{\alpha,TS}, \tag{4}$$

in this way, we are able to estimate directly the change in the sensitivity (i.e., the change in the slope of the regression β_b^α) due to the break (labeled with sub index b). In Fig. 4 we present the slopes of the coefficients of the temporal trends at the beginning (1922) and at the end of the sample (2013). There is one slope for each quantile before ($\beta^\alpha + \beta_b^\alpha$) and after the structural breaks (β^α). The slopes of the coefficient can be interpreted as the sensitivity of each quantile to the temporal trend, that is, as the annual growth rate of improvements in human longevity at the specific α associated to a certain quantile.

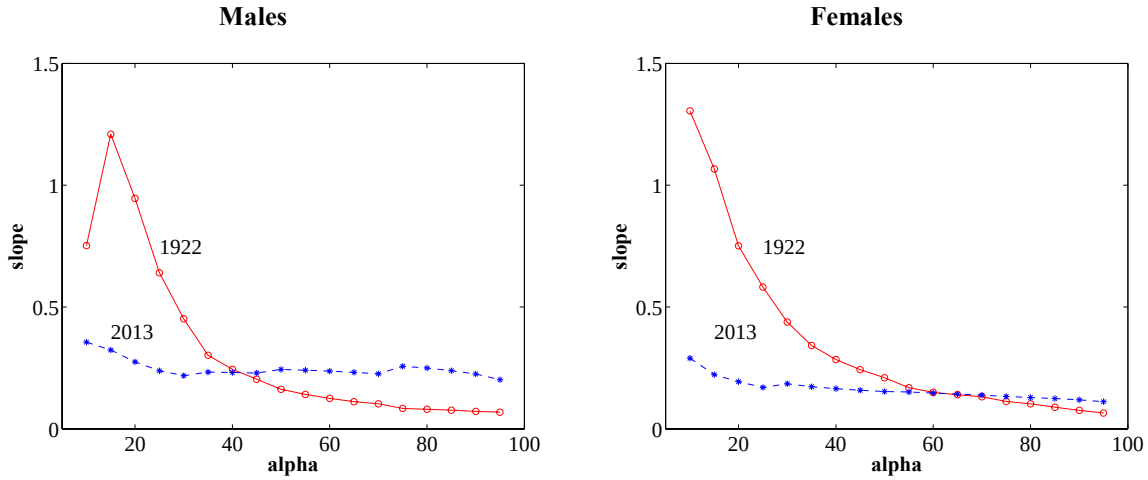

Fig. 4 Slopes of the temporal trends of lifespan quantiles against alpha from the 10th to the 95th percentile in 1922 (before any break) and in 2013 (after the breaks). Note: We estimated an equation including the temporal trends and dummy variables to determine directly the sensitivity of each quantile to the sustained upward trend. As expected, the rate of improvement is considerably faster at the lowest quantiles and slower at the highest. The behavior is more homogeneous at the end of the sample, after the majority of structural breaks had occurred in the 1940s and 1950s.

Fig. 4 contains valuable information about the estimation of the sensitivity of the different quantiles to the generalized upward trend. For instance, the improvements in longevity are much more significant for the lower quantiles, where only a small fraction of the total population can expect to die, than they are for the higher quantiles. This difference, moreover, was considerably more pronounced at the beginning of the sample than it was at the end, before some structural breaks occurred.

It is not, of course, necessary to go back to 1922 to obtain the shape of the solid line (period before the breaks) in Fig. 4, given that this was the process describing the data for women practically up to 1940, when the more significant structural breaks occurred (see Fig. 3). From 1940 onwards, the dotted line describes the situation better. Thus, additional increments in life expectancy in the future will come from further improvements along the whole curve and not solely from improvements in the lower quantiles (as was the case before the 1950s). Nevertheless, today when the current rates of improvement constitute the more homogeneous group, it is still true that the greatest improvements occur at the lower quantiles.

3.3 Stochastic cycles

To complement our linear trend model we breaks we included one stochastic cycle from a factor model, in an attempt at explaining more than 50% of the variability within the system, with the minimum number of unobservable or underlying factors. The estimated subjacent factor is presented in Fig. 5. The factor has a cyclical behavior that captures the main dynamics of the series under study, for both men and women. These factors can be interpreted as *survivorship cycles* in the evolution of the lifespan distributions[7]. That is, the estimated factors described cyclical patterns of increasing and decreasing periods, in the survivorship quantiles, which explain temporary distancing from the deterministic trends that best describe each quantile. For example, in the case of males, while an increasing in the deterministic trend characterized the evolution of the quantiles under study, during the whole sample period, we also observed a positive cycle, from 1970-2000. This means that during such period the empirical quantiles were higher than those predicted by the linear model (even taking into account possible breaks in the pace of improvement, as discussed before). During the same period there was also a positive cycle in the case of females.

On the contrary, both men and woman, experienced temporary lower quantiles, than those predicted by the linear models, from 1940 to 1960. This means that traditional trends were still recovering after the WWI and WWII and this process required more or less 20 years to be completed.

[7] This mortality cycles should not be confused with sine or cosine functions describing symmetric and deterministic oscillations about a certain trend. Instead, we are talking about stochastic cycles that describe asymmetric and random departures from the trend.

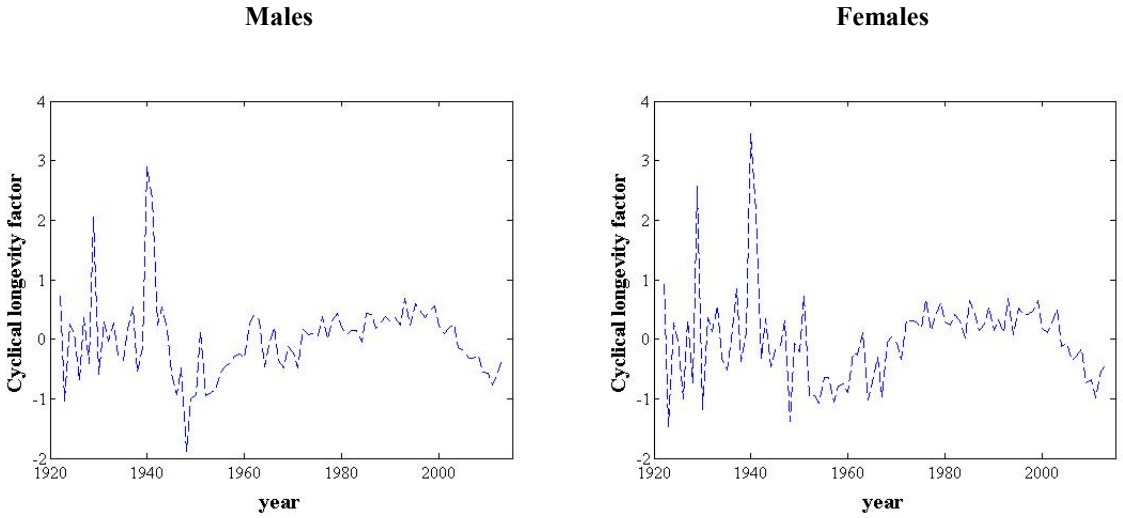

Fig. 6 Stochastic factor subjacent to all the lifespan quantiles. We estimate the factor in each case using principal components. The factor captures the common cyclical behavior of the stationary component in the quantiles. The long run component is modeled using broken trend models.

Quantiles series provide a different perspective on the analysis of human lifespan, compared to what can be currently found in the demographic or actuarial literature. We could have used instead mortality rates as our fundamental units, as is done in regular exercises. In this case, we would have tested for unit roots in mortality rates and perhaps fitted deterministic trends with breaks to characterize mortality series, instead of fitting them to the survivorship quantile series. Working with rates as building blocks seems convenient and intuitive in the context of mortality risk and risk heterogeneity analyses. Nevertheless, quantiles offer a complementary view of the same phenomenon, and they are natural candidates to analyze the whole distribution of human lifespan in a novel and intuitive way. Being denominated in ages, it is easy to explain to the general public what a certain quantile means, and what an increment or a reduction in its trajectory implies for human lifespan. From this angle, for example, issues related to longevity risk seem to be much more naturally addressed using survivorship quantiles than using mortality rates.

From our results we do not discard future reductions in infant, young or adult mortality, as potential drivers of future increments in our quantile series. We look at changes in the pace at which these survivorship improvements have been occurring during the last century. Our conclusion is that, using time series data-driven techniques, as the ones proposed here, not limits to lifespan can be recorded and not changes in the dynamics of the deterministic trends, that characterize quantiles of the lifespan distribution, are documented (for the UK, both for men and women), or at least not yet. This contrasts with what has been recently proposed by Dong et al. (2016). They interpret a reduction in the support of the life span distribution (which for example is evident from figures 3 and 4 here), as a sign of a reduction in the pace of lifespan improvement. We look directly to the rate of improvement in each fragment of the lifespan distribution, and we found that consistently with them not all the survivorship quantiles improve at the same rate (see figure 4), but contrary to them, we also document that there has been not reduction in the pace at which any quantile has been improving during the last 50 years in our sample. That is, even if the variance among the quantiles series is reducing, the trend in human lifespan has not changed..

4 Discussion

Many of our findings concerning the long run behavior of the central tendencies of human longevity are related to previous works in the literature. For example, Oeppen and Vaupel (2002), Cheung and Robine (2007), White (2002) and Canudas-Romo (2010), have documented an upward trend in the central measures of life duration using data sets for various developed countries over the last century. They conclude, moreover, that this upward trend in life expectancy, median duration of life, and modal age at death is a clear

reflection of improvements in public health, medicine, economic development, nutrition, education, and household conditions.

However, several studies also present evidence pointing to the fact the pace of these trends is dependent on the specific ages at which the improvements occur, for instance: Wilmoth and Horiuchi (1999), Wilmoth (2000), Wilmoth et al. (2000), Felipe et al. (2001), Fledelius et al. (2004), Wrycza and Baudisch (2012); among others, particularly Canudas-Romo (2010), who stresses that life expectancy and the median duration of life are highly sensitive to improvements in infant and child mortality, while the modal age at death is not.

The upward trend in the modal age at death has changed over time and it is widely known that the increase in life expectancy used to be mainly due to the reduction in infant, child and young-adult mortality, but in recent decades, it was mainly due to reduction in old-age mortality, as documented by Horiuchi et al. (2008) and Wilmoth and Lundström (1996). Thus, the apparent discontinuities in the slopes of the trends of the three measures are explained by the contributions made by different ages to total mortality. In the early years of the sample, the dramatic improvements in infant and child mortality led to marked increments in life expectancy and the median duration of life, but they had only a small impact on the mode. Once infant and child mortality rates stabilized around 1940-1945, the rate of increases in life expectancy slowed down, and subsequent improvements in expectancy can be attributed in the main to the increase in the survivorship of individuals at older ages. Survivorship at these late ages has had a smaller impact on life expectancy and no effect on the median duration; in contrast, the impact on the modal age at death has been marked. Thus, all in all, the longevity dynamics present broken trends with different slopes for the three central tendencies of the lifespan.

However, as stressed in the introduction, there is a dearth of studies focusing on the evolution recorded at the quantile level of the distribution of deaths in the fields of demography and actuarial sciences. It is our contention, that the study of these quantiles is much more informative than the analysis of the central tendencies alone. The analysis of quantiles permits quantifying the dynamics affecting different parts of the life table survivorship function distribution.

There are important substantive issues regarding the trends in life tables and the number of deaths reported at different ages, and therefore at different quantiles of the survivorship distribution. Regarding the shifting of the deaths distribution (Figure 2) toward older ages, and with respect to changes in the shapes of these curves, one may wonder, how fast did the curve move to older ages? Was the move accelerating, decelerating, or on a constant pace? Was the shape of old-age death distributions compressed, expanded or did it remain unchanged? In what way are those changes in location and changes in shape related? Do females and males differ in those aspects (periods of compression against periods of constant shape)? Are there noticeable international variations in those respects? Those and other related questions, as the shifting mortality hypothesis proposed by Bongaart (2005), could be addressed in direct and more comprehensive fashion, using the proposed methodology.

Focusing on the quantiles of the survivorship function is also novel because, as mentioned before, it switches the assumptions about what the fundamental atoms in the study of longevity and mortality are. This approach is certainly related to a strand in the demographic literature that uses directly estimations based on the life table function of the distribution of deaths (Zanotto et al., 2016) or on the age at death distribution (Basellini and Camarda, 2016), as an alternative to modeling the logarithm of the central death rate for each age ($log(m_{xt})$)as is traditionally done in the field. Doing so allows getting linear improvements in life expectancy, whereas working with the logs of mortality rates, you force e_0 to taper. Specifically, in our case, this explains why you observe linear and constant improvements in the trends of the quantiles of the survivorship function (Fig. 4) without tapering even at the highest ages. Particularly, it explains the path followed by the median of the lifespan distribution ($Q_t^{\alpha}, \alpha = 0.5$), which is constant and linear during the last 50 years in the sample period.

5 Conclusions and limitations

From our exercise it is clear that the rate of improvement at different age-categories is not the same, and indeed it is more pronounced still today for young ages than for old ones. Nevertheless, we also know that the proportion of people above certain ages is increasing at a constant rate. Structural breaks did occur, but only at the beginning of the sample. These results are consistent with well-established findings in the literature on demographic and epidemiologic transitions (that is, that mortality improvements at younger ages happened earlier and were of a bigger magnitude than improvements at older ages).

Moreover, our methods may enable us to project the trajectories of the quantile series in the future. Such exercise would take into account, not only the linear trends of the data, but also the stochastic cycles around

these, which are generally overlooked in the literature. One possible limitation of our methods in this regard is that they should not be used to forecast the quantiles long time ahead (hundreds of years), when possible crossovers among the quantile lines, although very unlikely, still might occur. One alternative is to assume that all the quantiles shared a stochastic trend (i.e. they are cointegrated), in the same spirit of Lee-Carter. But given that we rejected the null of unit roots and therefore the quantiles cannot be cointegrated. This is an interesting issue that certainly needs to be addressed in future research.

Longevity extension owes more to the fact that more people are getting to older ages, than to the fact more people are reaching extremely high ages in the tail of the distribution. This latter situation remains true, but to a lesser extent, compared to the former.

This behavior has important implications for pension funds and welfare states. An aging population represents a challenge, not solely because individuals live longer, but also because more elderly people die at these advanced ages. This concentration of the age at death is changing the traditional point of view regarding the evolution of longevity and has led to a reassessment of the viability of defined-benefit pension schemes, which frequently recommend increasing the age of retirement to ease the pressure posed by an aging population. The future funding of pension schemes will not only have to take into account the fact that people contributing to the system live longer (and so will enjoy benefits for longer periods), but to the fact that more people will have to be subsidized in the near future. That is all the quantile series in our exercise are increasing at constant rates during the last decades in our sample.

Our study fails to detect a reduction in the pace of longevity extension over the last 50 years in our sample of UK males and females. A reduction in the rates of improvement did occur prior to the 1950s, but no further signals of deceleration are to be expected in the near future.

With our methodology, we model the trends of longevity extension by looking at the evolution of age-limits reached by only a fraction of the population. Future comparisons among different countries, and related dependency-ratios, may be conducted using our methodology, and they will enhance our understanding of longevity extension and will contribute to improve measurement of the increase of human life duration. Our methods facilitate comparisons of longevity dynamics across countries or regions (as done in Rees et al. 2012). That is, by focusing on the quantiles of the survivorship function it is possible to analyze relative dynamics, which otherwise would be hidden in traditional analysis of the central tendencies of the mortality statistics.

In theory it is possible to go from mortality rates to quantiles and vice-versa. In practice, coming back from projected quantiles to implied mortality surfaces and to constructs based on them, such as life expectancy, is unfeasible. Or at least it is, when we use a relatively small number of quantiles as we did (we used 18 quantiles). One needs a much more finer grid of quantiles to be able of getting back representations in terms of mortality rates. This would take us very far from our main objective of analyzing fundamental time series characteristics of the survivorship function, and the pace of human longevity expansion, so we leave it for future research.

Compliance with Ethical Standards:
Conflict of Interest: The authors declare that they have no conflict of interest.

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Appendix

A.1. Factor Models

In this context, the factor model on the quantile cycles can be presented as:

$$\begin{aligned} c_t^{\alpha} &= \lambda^{\alpha} \boldsymbol{F}_t + error_t^{\alpha} \\ c_t^{\alpha} &= \boldsymbol{\Gamma}_t^{\alpha} + error_t^{\alpha}, \end{aligned} \tag{A.1}$$

where $error_t^{\alpha}$ is referred to as the idiosyncratic error of the model, λ_{α} is a 'factor load' coefficient that measures how much the cycle reacts to general shocks, which affect all the quantiles of the distribution. $\boldsymbol{\Gamma}_t^{\alpha} = \lambda^{\alpha} \boldsymbol{F}_t$ is the common component of the model, that is shocks that impact all the quantiles. If we define $\boldsymbol{C}_t = \left(c_t^{\alpha_1}, c_t^{\alpha_2}, \dots, c_t^{\alpha_m}\right)'$ and $\boldsymbol{\Lambda} = (\lambda^{\alpha_1}, \dots, \lambda^{\alpha_m})'$, where $\alpha_j, j = 1, \dots m$ represents specific quantile indexed by j (and therefore $0 < \alpha_j < 1$), we have, in matrix form:

$$\underset{(m \times 1)}{\boldsymbol{C}_t} = \underset{(m \times r)}{\boldsymbol{\Lambda}} \underset{(r \times 1)}{\boldsymbol{F}_t} + \underset{(m \times 1)}{\boldsymbol{error}_t}, \tag{A.2}$$

where $\boldsymbol{error}_t = \left(error_t^{\alpha_1}, error_t^{\alpha_2}, \dots, error_t^{\alpha_m}\right)'$. Note that, without loss of generality, even when the model specifies a static relationship between c_t^{α} and $\boldsymbol{F}_t$, $\boldsymbol{F}_t$ can be considered a dynamic vector process. If $\boldsymbol{F}_t$ and $\boldsymbol{X}_t$ are jointly stationary, then $\boldsymbol{F}_t$ evolves according to a vector autoregression (VAR) process, as follows:

$$\boldsymbol{A}(L)\boldsymbol{F}_t = \boldsymbol{u}_t, \tag{A.3}$$

where $\boldsymbol{A}(L)$ is a polynomial of the lag operator. This model is referred to in the literature as a dynamic factor model if $\boldsymbol{F}_t$ includes primitive factors and their lags or as a static factor model if it accounts only for the primitive factors (Bai and Ng 2008).

The factors in Eq. 5 can be estimated using principal components (PC) or singular value decompositions (SVD), both methods allowing the estimation of the factors and the factor loads.

Identification issues arise owing to the fact that $\boldsymbol{F}$ and $\boldsymbol{\Lambda}$ are clearly not separately identifiable. For any arbitrary $(r \times r)$ invertible matrix $\boldsymbol{H}$ we have that:

$$\boldsymbol{F}\boldsymbol{\Lambda}' = \boldsymbol{F}\boldsymbol{H}\boldsymbol{H}^{-1}\boldsymbol{\Lambda}' = \boldsymbol{F}^*\boldsymbol{\Lambda}'^*, \tag{A.4}$$

where $\boldsymbol{F}^* = \boldsymbol{F}\boldsymbol{H}$ and $\boldsymbol{\Lambda}^* = \boldsymbol{\Lambda}\boldsymbol{H}^{-1}$. In this case, the factor model is observationally equivalent to $\boldsymbol{C} = \boldsymbol{F}^*\boldsymbol{\Lambda}'^* + \boldsymbol{error}$. Therefore, r^2 restrictions are required to uniquely fix $\boldsymbol{F}$ and $\boldsymbol{\Lambda}$ (Bai and Wang 2012). Notice that the estimation of the factors when using either PC or SVD imposes the normalization that $\frac{\boldsymbol{\Lambda}'\boldsymbol{\Lambda}}{\boldsymbol{M}} = \boldsymbol{I}_r$ and $\boldsymbol{F}'\boldsymbol{F}$ be diagonal, which are sufficient to guarantee identification (up to a column sign change). We follow this approach here.

A.2. Further considerations when dealing with stochastic versus deterministic trends

When working with time series, such as mortality rates or, as in this case, temporal quantiles of the lifetable survivorship function, the trends and cycles in the data must be modeled accurately in order to determine whether the time dynamics of the system respond to stochastic or deterministic trend components (or, alternatively, whether the processes are stationary in levels and, thus, do not house any trend at all).

The way of approaching each case differs considerably. For example, when the system is stationary in levels (that is, there is not a deterministic, nor a stochastic trend in the data), it is possible to project future patterns using traditional time-series ARMA-models (Enders 2010) or traditional principal components analysis (Stock and Watson 2002). In this case, the shocks to the system, for example, mortality reductions due to specific improvements in health treatments, vaccines, medical facilities, etc.; would lack a permanent effect on mortality rates. Indeed, the effects of such shocks disappear after several periods, perhaps with some level of persistency, but in the long run, they do not modify the level of mortality rates.

However, if the series are trend-stationary, they have to be detrended before the estimation of causal or forecasting models. In this case, a simple deterministic model would suffice to describe the trend and thus, the projection of future patterns should focus on forecasting the cycles of the series, around the deterministic trend.

Finally, if the series are difference-stationary, traditional analysis is only valid after checking for cointegration (Engle and Granger 1987; Johansen 1988) or after differentiating each series, as many times as is required, to achieve stationarity. Cointegration refers to a situation in which two or more time series (mortality rates or survivorship quantile series) shared the same stochastic trend. That is, the effects of shocks that affect the dynamics of the series do not disappear and, moreover, they are the same for all the series.

In pragmatic terms, the first step to take is to check for unit roots in the data. Traditional and augmented unit root tests proposed by Dickey and Fuller (1979) and Said and Dickey (1984), respectively, are employed in this study, to determine whether the series present evidence of unit roots.

It is especially important to compare with the case in which the series are well described by a temporal trend that faces unexpected random breaks. In this case, traditional unit root tests suffer a very inconvenient lack of power and might easily conclude in favor of a unit root, even when the process is better described by a linear trend with one or several breaks. This is especially important in the present context, because, on the one hand, under the unit root hypothesis, any shock affecting the quantiles of the survivorship distribution would have a permanent effect on the trajectory of such quantiles. On the other hand, if the unit root-hypothesis is rejected, the effects of a shock would disappear, around the deterministic trend, creating what we could label as *mortality cycles*. That is, cyclical patterns of increasing and reduction in mortality quantiles, around the deterministic trend. Under a deterministic trend model with a break, we could identify which shocks to the system produce a permanent effect, in terms of mortality reduction, and which of them disappear in few years. The former will be associated to the *break-dates*, while the latter will describe the stationary cycles around the quantile-trend.

A.3. Testing Procedure.

Table A.1. Traditional unit root tests (t) for lifespan quantiles and critical values (cv). We used an Augmented Dickey-Fuller test with different lags (from 0 to 2) and with alternative hypotheses: intercept and intercept and trend. In the cases when the absolute value of the calculated t-statistic is higher than the absolute value of the critical value (cv), the null of a unit root is rejected. We report the critical values at 95% in every case.

Males

Alternative	Intercept						Intercept and Trend					
Lags	0		1		2		0		1		2	
Percentile	t	cv	t	cv	t	cv	t	cv	t	cv	t	cv
10	-1.9	-2.9	-1.8	-2.9	-2.3	-2.9	-1.0	-3.5	-1.0	-3.5	-0.7	-3.5
15	-2.7	-2.9	-2.0	-2.9	-2.6	-2.9	-2.6	-3.5	-1.5	-3.5	-1.9	-3.5
20	-2.4	-2.9	-1.8	-2.9	-2.4	-2.9	-3.1	-3.5	-2.0	-3.5	-2.3	-3.5
25	-2.0	-2.9	-1.4	-2.9	-1.9	-2.9	-3.4	-3.5	-2.1	-3.5	-2.3	-3.5
30	-1.5	-2.9	-0.9	-2.9	-1.2	-2.9	-3.8	-3.5	-2.3	-3.5	-2.3	-3.5
35	-1.0	-2.9	-0.3	-2.9	-0.4	-2.9	-4.0	-3.5	-2.4	-3.5	-2.1	-3.5
40	-0.7	-2.9	0.1	-2.9	0.2	-2.9	-3.7	-3.5	-2.2	-3.5	-1.8	-3.5
45	-0.3	-2.9	0.6	-2.9	0.7	-2.9	-3.1	-3.5	-1.8	-3.5	-1.2	-3.5
50	-0.1	-2.9	0.8	-2.9	1.1	-2.9	-2.6	-3.5	-1.5	-3.5	-0.9	-3.5
55	0.1	-2.9	1.1	-2.9	1.5	-2.9	-2.3	-3.5	-1.2	-3.5	-0.6	-3.5
60	0.2	-2.9	1.4	-2.9	1.8	-2.9	-2.0	-3.5	-1.0	-3.5	-0.3	-3.5

65	0.3	-2.9	1.6	-2.9	2.0	-2.9	-1.9	-3.5	-0.8	-3.5	-0.2	-3.5
70	0.4	-2.9	1.7	-2.9	2.2	-2.9	-1.8	-3.5	-0.8	-3.5	-0.1	-3.5
75	0.4	-2.9	1.7	-2.9	2.2	-2.9	-1.8	-3.5	-0.8	-3.5	-0.1	-3.5
80	0.3	-2.9	1.7	-2.9	2.2	-2.9	-1.9	-3.5	-0.8	-3.5	-0.2	-3.5
85	0.2	-2.9	1.6	-2.9	2.1	-2.9	-2.1	-3.5	-1.0	-3.5	-0.4	-3.5
90	0.1	-2.9	1.5	-2.9	2.0	-2.9	-2.4	-3.5	-1.2	-3.5	-0.5	-3.5
95	-0.1	-2.9	1.3	-2.9	1.8	-2.9	-3.0	-3.5	-1.6	-3.5	-0.9	-3.5

Females

Alternative	Intercept						Intercept and Trend					
Lags	0		1		2		0		1		2	
Percentile	t	cv	t	cv	t	cv	t	cv	t	cv	t	cv
10	-2.6	-2.9	-2.9	-2.9	-3.6	-2.9	-1.4	-3.5	-1.0	-3.5	-1.2	-3.5
15	-2.8	-2.9	-2.8	-2.9	-3.9	-2.9	-2.4	-3.5	-1.5	-3.5	-1.9	-3.5
20	-2.8	-2.9	-2.8	-2.9	-3.8	-2.9	-2.9	-3.5	-1.7	-3.5	-2.1	-3.5
25	-2.5	-2.9	-2.4	-2.9	-3.2	-2.9	-2.9	-3.5	-1.7	-3.5	-2.0	-3.5
30	-2.1	-2.9	-2.0	-2.9	-2.6	-2.9	-3.0	-3.5	-1.7	-3.5	-1.9	-3.5
35	-1.8	-2.9	-1.6	-2.9	-2.1	-2.9	-3.1	-3.5	-1.8	-3.5	-1.8	-3.5
40	-1.5	-2.9	-1.2	-2.9	-1.6	-2.9	-3.3	-3.5	-1.9	-3.5	-1.8	-3.5
45	-1.4	-2.9	-1.0	-2.9	-1.3	-2.9	-3.8	-3.5	-2.0	-3.5	-1.9	-3.5
50	-1.2	-2.9	-0.7	-2.9	-0.9	-2.9	-4.2	-3.5	-2.3	-3.5	-1.9	-3.5
55	-1.1	-2.9	-0.5	-2.9	-0.7	-2.9	-4.8	-3.5	-2.6	-3.5	-2.1	-3.5
60	-0.9	-2.9	-0.4	-2.9	-0.5	-2.9	-5.2	-3.5	-2.8	-3.5	-2.2	-3.5
65	-0.9	-2.9	-0.3	-2.9	-0.4	-2.9	-5.8	-3.5	-3.2	-3.5	-2.5	-3.5
70	-0.9	-2.9	-0.3	-2.9	-0.3	-2.9	-6.5	-3.5	-3.5	-3.5	-2.8	-3.5
75	-0.9	-2.9	-0.2	-2.9	-0.2	-2.9	-7.1	-3.5	-3.9	-3.5	-3.1	-3.5
80	-0.9	-2.9	-0.2	-2.9	-0.1	-2.9	-8.1	-3.5	-4.7	-3.5	-3.7	-3.5
85	-0.9	-2.9	-0.1	-2.9	0.0	-2.9	-9.1	-3.5	-5.5	-3.5	-4.5	-3.5
90	-0.9	-2.9	-0.1	-2.9	0.0	-2.9	-9.4	-3.5	-6.0	-3.5	-5.0	-3.5
95	-1.1	-2.9	-0.2	-2.9	-0.1	-2.9	-9.8	-3.5	-6.3	-3.5	-5.4	-3.5

We first perform the test against the alternative hypothesis of an intercept. That is, assuming in the alternative hypothesis that the quantiles fluctuate around a constant mean, which would be the case of underlying constant mortality rates across age categories. As expected, the null of a unit root cannot be rejected in this case (that is, we reject the constant mortality hypothesis) regardless of the quantile under consideration. This result is not surprising, because of the clearly increasing dynamics during the sample of every series, which is evident in Fig. 3.

At this point, we had to construct the test with an alternative that included a time-trend. In this case, we found that for some of the quantiles, that is, those above the 45th percentile for females and those between the 30th and 40th percentiles for males, the null of a unit root must be rejected. This means that the processes within these ranges are trend-stationary and that they should not be modeled as if they contained a stochastic

trend. This is an important finding. It means that the best model describing the data, within the aforementioned categories, is a simple deterministic linear model. In this case, given the constant slope of the straight line, no signs of increasing or decreasing pace in the process of longevity extension is found. It does not mean that mortality is constant at these categories, but rather that the rate of the mortality shift.

For quantiles lying outside the aforementioned range, it is unclear as to whether they are described by a unit root process or by a deterministic trend with a break. Table A.2 reports the values of the corrected statistics and the corrected critical values, considering a break in both the null and the alternative, for the IO model described in section 2.2. We only report the results of the tests for those quantiles for which we failed to reject the null of a unit root (see Table A.1), thus, the blanks in this table correspond to the cases for which a simple linear model suffices and, therefore, we do not need to test for the IO model. In the other cases we do need to consider the presence of structural breaks in the trend. A consideration of this possibility shows that the null of a unit root is rejected at every quantile for females (with the sole exception of the 25th percentile).

In the case of men, the null hypothesis of a unit root is rejected at the 10th and 15th percentiles, at a confidence of 95%, and the 20th, 25th, and 95th percentiles, at a confidence of 90%. Percentiles lying between the 45th and 90th levels for males could, alternatively, be modeled via a stochastic trend. However, in this case, additional specification tests are needed before we might conclude in favor of the unit root hypothesis (i.e., tests allowing for a greater number of breaks, and different functional forms of the deterministic trends). Therefore, we prefer to model all the quantiles using the broken trend model, because it proves to be more appropriate in most cases.

Once again we are facing an interesting finding. The series of quantiles in almost all the cases are better described by a linear model with a one-time change in slope, than by a unit root process (stochastic trend). What is means is that, indeed, there are signs of acceleration or deceleration (depending on the sign of the rotation) in the rate of improvements for these quantiles, but such changes are one-time breaks during the whole sample. Once these *big shocks* have impacted mortality rates, subsequent shocks lack a permanent or cumulative effect on the quantile-trends of the lifespan distribution.

Table A.2. Unit root tests (t), critical values (cv) and fraction of the sample where the structural break is presented (θ) for the innovative outlier model. We used the statistic proposed by Kim and Perron (2009), which allows for a single break under both the null and the alternative hypotheses. Critical values at a confidence of 95% are shown. θ is the fraction of the sample in which the structural break is detected. Blanks correspond to the quantiles better described by a linear model, for which there is not necessity of considering neither the IO model or the unit-root hypothesis.

	Males			**Females**		
Percentile	**t**	**cv**	**θ**	**t**	**cv**	**θ**
10	-8.7	-3.99	0.2	-6.6	-4.17	0.3
15	-6.1	-3.99	0.2	-5.7	-4.17	0.3
20	-3.6	-3.99	0.2	-4.9	-4.17	0.3
25	-3.9	-3.99	0.2	-2.2	-4.17	0.3
30	-	-	-	-4.4	-3.99	0.2
35	-	-	-	-4.5	-3.99	0.2
40	-	-	-	-4.3	-3.99	0.2
45	-2.4	-4.24	0.4	-4.3	-3.99	0.2
50	-2.8	-4.24	0.5	-	-	-
55	-2.8	-4.24	0.5	-	-	-
60	-2.7	-4.24	0.5	-	-	-
65	-2.8	-4.24	0.5	-	-	-
70	-2.8	-4.24	0.5	-	-	-

75	-2.9	-4.24	0.7	-	-	-
80	-3.2	-4.18	0.7	-	-	-
85	-3.3	-4.18	0.7	-	-	-
90	-3.7	-4.18	0.7	-	-	-
95	-4.2	-4.18	0.7	-	-	-

At this point, we need to identify the periods in which the breaks in the linear trends occurred and estimate the sign of such rotations. Given the results above, the break dates are determined endogenously and they are reported in Table 3. In this table we calculated the break date for all the quantiles, even for which they are not statistically significant, in the sake of completeness.

Table A.3. Unit root tests for the innovative outlier model. We used the statistic proposed by Zhou and Perron (2005) to estimate endogenously the breaks in the trends.

	Males				**Females**		
Percentile	break year	**Percentile**	break year	**Percentile**	break year	**Percentile**	break year
10	1943	**55**	1968	**10**	1945	**55**	1937
15	1941	**60**	1968	**15**	1945	**60**	1937
20	1940	**65**	1968	**20**	1945	**65**	1937
25	1940	**70**	1968	**25**	1957	**70**	1937
30	1940	**75**	1983	**30**	1940	**75**	1938
35	1963	**80**	1984	**35**	1940	**80**	1938
40	1963	**85**	1984	**40**	1940	**85**	1938
45	1963	**90**	1984	**45**	1940	**90**	1938
50	1968	**95**	1984	**50**	1940	**95**	1938

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