Centenarian Survival: Stagnating of Improving?

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Abstract

Centenarians are the forerunners of longevity. They have emerged as a consequence of reductions in the risk of dying at young ages. Still, it is not clear-cut if these individuals are living longer and how their outstanding survival trajectories are mirrored on aggregate measures such as life expectancy at age 100 ($\bar{e}_{100}$). Here we present the case of four female populations in which mortality above age 100 is improving across cohorts. At population level, mortality hazards decline as part of the general postponement of deaths occurring in modern populations. As a result, $\bar{e}_{100}$ and measures of lifespan variability are trending upwards, indicating that lifespans among centenarians are highly malleable and very heterogeneous. However, we also show that deaths are not being postponed at the same pace for all the members of the population since, when controlling for unobserved heterogeneity, individual mortality improves more rapidly than what is observed in the aggregate level. Therefore, unobserved heterogeneity prevents populations from further mortality declines. Our results support the view that the plasticity in the risk of dying above age 100 is a complex interaction between biological endowments and social behaviours. Such interactions are moderated differently among sexes and across countries by external factors.

Keywords: longevity | centenarians | population heterogeneity

The malleability in the risk of dying encountered in human populations (Burger et al., 2012) has had several implications in the shaping of longevity outcomes over time. There is general agreement that newborns of modern populations doing the best have gradually benefited from the downward trend of death rates at young ages (Colchero et al., 2016). These individuals can now enjoy a greater expectation of life (Oeppen and Vaupel, 2002; Rau et al., 2008) and less uncertainty about their age at death (Colchero et al., 2016; Vaupel et al., 2011; Zhang and Vaupel, 2009) than previous generations. Mortality improvements at young ages have also contributed to the increased number centenarians (individuals surviving to age 100) observed since the second half of the twentieth century (Lenart and Vaupel, 2017). Likewise, there is new evidence (Zuo et al., 2018) showing that old-age mortality is been postponed (Vaupel, 2010) at a speed of about 3 years per generation (e.g. mortality today at age 68 is equivalent to age 65 a generation ago). In spite of all these findings, there is at present limited understanding about *(i)* how malleable mortality above age 100 is and *(ii)* whether life expectancy at age 100 ($\bar{e}_{100}$) is increasing or not.

Much of our knowledge about this issue comes from studies on Sweden and Denmark (Drefahl et al., 2012; Modig et al., 2017), where mortality among centenarians has not improved over time where

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and $\bar{e}_{100}$ has remained fairly constant. Recently, Medford et al. (2019) demonstrated that just the oldest old in Denmark (centenarian lifespans in the 90th percentile) have been getting older, while there has been no evidence of any increase in lifespan for Swedes. As yet, it is unknown if there are some other populations that have suffered from mortality improvements above age 100. Further, the dynamics of life expectancy at age 100 and the variability of lifespans after such age are still unexplored.

Centenarians are individuals that live their lives on the frontier of survival. However, it is not clear how their outstanding survival trajectories are mirrored on the trends that we observe at a population level. Indeed, it has been proven that in humans (Christensen et al., 2008) and in other species (Rebke et al., 2010), old-age mortality trajectories for individuals radically differ from what it is observed at the population level. These differences arise as a consequence of unobserved heterogeneity (Vaupel and Yashin, 1985), which implies that populations are comprised by several subgroups of individuals with different mortality risks Vaupel et al. (1979b). In this sense, the composition of the cohort changes as individuals age leading to a deceleration in the population death rates (Horiuchi and Wilmoth, 1998; Steinsaltz and Wachter, 2006; Wrigley-Field, 2014) or ultimately to level-off at extreme old ages (Barbi et al., 2018; Gampe, 2010; Rootzén and Zholud, 2017). These phenomena reflect a balance between the increasing mortality of each sub-population and the changing mix of them. These issues have to be carefully addressed in the study of mortality trajectories among centenarians. Then, it is of paramount importance to make use of adequate mathematical models that allow us to control for unobserved heterogeneity and disentangle between population and individual mortality trajectories at high advanced ages.

It is well known that females outlive males even in the worst conditions (Zarulli et al., 2018). Therefore, it is not surprising to observe a greater number of female centenarians in comparison to males (Perls, 2017). This triggers to high uncertainty in our calculations of longevity outcomes for males. For this reason we centre our study solely on female centenarians. Here we cogently document cohort mortality trajectories for female centenarians in 4 developed countries that exhibit high-quality data. We examine trends across cohorts in life expectancy at age 100 and determine how variable lifespans become after this age. We also make use of frailty models to unravel the discrepancies between mortality trajectories for individuals and the observed ones for the population. We use this approach to *(i)* test out whether mortality above age 100 is improving or not and *(ii)* to measure the impact of heterogeneity in slowing down the rate of mortality progress at high advanced ages. We hypothesize that, in some of these populations, mortality among centenarians has improved across cohorts. In consequence, such improvements have triggered to extensions in the tail of longevity.

Results

The length and inequality of life after age 100

France, Italy and Japan exhibit clear upward trends in $\bar{e}_{100}$ (see Figure 1). In particular, the sharp rise of $\bar{e}_{100}$ depicted in Japan is notable. Japanese women born during 1870-79 displayed $\bar{e}_{100}$ values of about 1.8 years. Thirty years later, $\bar{e}_{100}$ in this country was 35% higher. In France and Italy, $\bar{e}_{100}$ increased 21% and 22% in France and Italy respectively from the cohort 1860-69 to 1880-89. Thereafter, declines are depicted in both countries. Great Britain, on the other hand, portray a fairly constant trend across cohorts since $\bar{e}_{100}$ took values of around 2.1 years during the whole period analysed.

Figure 1 about here

Figure 1 also reveals lifespan variability values (portrayed by life disparity, $\bar{e}^{\dagger}_{100}$) very much alike to the ones depicted by life expectancy. As a result, extensions of lifespans above age 100 are accompanied with increases in life disparity. This entails that the distribution of lifespans after age 100 becomes more variable as $\bar{e}_{100}$ increases. This finding is noteworthy for many reasons. First of all, increases in lifespan variability at high advanced ages are at odds of previous analyses arguing for a looming limit in human lifespan (Dong et al., 2016). As pointed out by Zuo et al. (2018): if lifespans are approaching a limit, death should gradually become compressed towards the most advanced ages near the limit and in consequence lifespan variability would decrease. Instead, we observe that the average length of life is extending but also the distribution of lifespans has become increasingly dispersed over time, which demonstrates that compression in not achieved in the populations studied here.

Second, the similarity between $\bar{e}_{100}$ and $e^{\dagger}_{100}$ yields to outcomes of Keyfitz's entropy ($\bar{H}_{100}$) close to unity. As mentioned in the *Materials and Methods* section, $\bar{H}_{100}$ is defined as the elasticity of life expectancy with respect to a proportional change in mortality (Keyfitz and Caswell, 2005). Hence, changes in death rates at all ages after age 100 translate into similar changes in $\bar{e}_{100}$. This suggests that increases in $\bar{e}_{100}$ are driven by improvements in mortality after age 100. Third, values of $\bar{H}_{100}$ close to unity also imply an exponential decline in the survival curve. Certainly, half of the centenarians die before their $\bar{e}_{100}$ and half of them survive to even older ages (see Figure 6 in SI Appendix). This shows the outstanding ability of centenarians to prolong their length of life and survive to extreme ages. Fourth, the exponential decline in the survival curve corresponds to a constant risk of dying at some age after 100. This regularity is in line with previous studies, which have encountered a mortality plateau in human populations (Barbi et al., 2018; Gampe, 2010; Rootzén and Zholud, 2017).

Relationship between individual trends and the entire population

The dynamics of mortality that individuals experience were estimated by controlling for unobserved heterogeneity across cohorts (Vaupel et al., 1979a). See *Materials and Methods* for a complete description of the estimation procedure. The results of the statistical modelling are shown in the *SI Appendix*.

We found that life expectancy is shorter for individuals (e_{100}) than what is observed at the population level. However, both longevity measures follow a similar trajectory across cohorts (Figure 7 in *SI Appendix*). In Figure 2, we compare relative changes in $\bar{e}_{100}$ and e_{100}. At the population level, we observe that in Japan, the annual rate of change in $\bar{e}_{100}$ (denoted to as $\dot{\bar{e}}_{100}$) increased from 1% to 1.8%. For Italy, $\dot{\bar{e}}_{100}$ takes positive values of around 1.0% but it turns to negatives during the most recent cohorts. Similarly, French e_{100} increased at a speed of 0.5% per decade and in Great Britain, changes in $\bar{e}_{100}$ are almost null. At individual level, e_{100} has trended upwards more rapidly than $\bar{e}_{100}$ in all countries and all the analysed cohorts (Figure 2). In Japan, for example, values of $\dot{e}_{100}$ are twofold $\dot{\bar{e}}_{100}$. It is also notable that gains in e_{100} are achieved even when $\dot{\bar{e}}_{100}$ depict deterioration (see France 1900-04).

Figure 2 about here

Figure 2 also depicts the rate of increase in $\bar{e}_{80}$ and $\bar{e}_{90}$. It is striking that similar progress in $\bar{e}_x$ is achieved at ages 80, 90 and 100. This implies that $\bar{e}_{80}$ and $\bar{e}_{90}$ are increasing partly due to mortality improvements above age 100. In Japan $\dot{e}_{100}$ is even higher than $\dot{\bar{e}}_{80}$ and $\dot{\bar{e}}_{90}$, which entails that the average length of life among individuals is extending more rapidly than what is observed at the population level. Altogether, Figures 1 and 2 provide cogent evidence that the average length of life above age 100 is increasing across cohorts.

Figure 3 displays mortality hazards at age 100 ($\bar{\mu}(100,y)$), the estimated hazards for individuals ($\mu(100,y)$) and their respective 95% confidence intervals. It is clear that mortality hazards in the four populations decrease across cohorts. Nonetheless, the decline is more pronounced for individuals than for the population as whole. For instance, $\bar{\mu}(100,y)$ for Japanese women reduced by 0.16 (from 0.50 in 1860-69 to 0.34 in 1900-04), whereas the estimated $\mu(100,y)$ halved during the same period of time. Moreover, the gap between individuals and population hazards narrows down across cohorts implying that individuals' mortality gradually converges to population trajectories.

From Figure 3 it is clear that mortality at age 100 has improved over time. We additionally test out whether similar progress in reducing mortality has been achieved at other ages above 100. In this sense, Figure 4 illustrates annual rates of mortality improvement ($\bar{\rho}(x,y)$) for the entire population for ages between 100 and 105. We limited our analysis to these ages since confidence intervals are too wide after age 105, making difficult to draw reliable conclusions. Rates of mortality improvement were annualized under the assumption that such rates prevail during each 10-year cohort. This allows a straightforward interpretation of $\bar{\rho}(x,y)$.

Mortality has reduced above age 100. This regularity prevails in France (cohorts 1860-1900), Italy (1870-1900) and Japan (1870-1900), where $\bar{\rho}(x,y)$ has taken values of around 1% and 2%. In particular, mortality hazards above age 100 have consistently decreased in Japan across cohorts and there are no signs of mortality deterioration in this country. Such improvements have propelled $\bar{e}_{100}$ across cohorts, putting forward evidence that reductions in the risk of dying are necessary for sizeable extensions in $\bar{e}_{100}$. Conversely, $\bar{\rho}(x,y)$ in France, Great Britain and Italy has slowed down during some cohorts and diminished towards negative values at older ages. This pattern explains the stagnation of British $\bar{e}_{100}$ and the small extensions of French and Italian $\bar{e}_{100}$ in comparison with Japan.

A closer inspection to the trajectories over time of $\bar{\mu}(x,y)$ reveals that deaths above age 80 are being postponed. In Japan, for example, deaths have being delayed such that mortality hazards for the cohort 1900-04 are at the same level as those of people 5 years younger in 1880-89 (see Figure 8 in *SI Appendix*). This occurs even at ages above age 100, where $\bar{\mu}(100,1900-04)$ is at the same levels of $\bar{\mu}(95,1880-1889)$ and $\bar{\mu}(105,1900-1904)$ is equivalent to $\bar{\mu}(100,1880-89)$. Similar patterns occur in France and Italy are postponed to a rate of 5 years every three generations. Thus, this suggests that mortality improvements are largely attributed to the postponement of deaths taking place in modern populations (Vaupel and Lundstrom, 1994; Vaupel, 2010; Zuo et al., 2018). Here we demonstrate that such phenomenon also occurs across cohorts and at high advanced ages.

Finally, we estimated rates of mortality improvement for individuals ($\rho(x,y)$, see Figure 5). Similar to our analysis of $\bar{\mu}(100,y)$ and $\mu(100,y)$, we found that mortality above age 100 is improving more rapidly for individuals than what is observed for the population. For some French and Japanese cohorts, improvements for individuals doubled the mortality achievements at population level ($\rho(x,y)$ depict values around 2% annually). This finding clearly demonstrates that heterogeneity prevents the population from further mortality improvements at high advanced ages as well as from extensions in $\bar{e}_{100}$. Moreover, the fact that mortality is reducing for both, individuals and for the entire population puts forward evidence about the high malleability of lifespans above age 100.

1 Discussion

In this study we shed light on the dynamics of mortality among female centenarians of four low-mortality populations across the cohorts 1860-1904. We emphasize three findings. First, centenarians are increasingly living longer over time. Increases in $\bar{e}_{100}$ are comparable to those depicted by $\bar{e}_{80}$ and $\bar{e}_{90}$. Likewise, the variability in the age at death has gone up, which implies high heterogeneity among centenarians' lifespans. The upward trends in $\bar{e}_{100}$ and $\bar{e}_{100}^{\dagger}$ put forward evidence against mortality compression (Dong et al., 2016).

Second, contrary to previous studies on Denmark and Sweden (Drefahl et al., 2012; Modig et al., 2017), we show that mortality hazards above age 100 are falling down in France, Great Britain, Italy and Japan, entailing a high malleability in the risk of dying at high advanced ages. We also show that such mortality declines are consequence of the general postponement of deaths occurring in modern populations (Vaupel and Lundstrom, 1994; Vaupel, 2010; Zuo et al., 2018). For instance, we present evidence that in Japan, death rates above age 100 today are similar to those portrayed by individuals 5 years younger 2 decades ago. Third, we found that mortality improvements are greater for individuals than those observed for the entire population. This confirms that unobserved heterogeneity (Vaupel et al., 1979a; Vaupel and Missov, 2014; Yashin et al., 1985) prevents centenarians from further improvements in mortality and consequently, from extensions in the average length of life.

Altogether, these findings prove that the tail of longevity is extending in France, Italy and Japan and to a lower extend in Great Britain. However, a number of questions arise: what are the mechanisms behind such mortality declines after age 100? what explains the high heterogeneity in lifespans after age 100? why mortality improvements occur solely in some female populations? what characterizes those individuals surviving to ages beyond $\bar{e}_{100}$?

It has been pointed out that the plasticity of human ageing is driven by external factors (Burger et al., 2012). In this context, we argue that attained reductions in the risk of dying as well as the high variability of lifespans above age 100 found in this research are the outcome of a complex interaction between biological endowments, genetics and social behaviours. Such determinants are moderated differently among sexes and across countries by external factors such as standards of living, education, medical interventions and public health efforts aiming at prolonging the length of life of individuals (Riley, 2001, 2005; Schofield and Reher, 1991). There is increasing evidence that supports this reasoning.

Centenarians are in poor health (Andersen-Ranberg et al., 2001; Hazra et al., 2015), which translates into a high risk of dying. However, there is a growing body of evidence showing that these individuals are increasingly been beneficed from external factors such as medical interventions. Proof of this can be seen in the upward trend of hospitalization rates among centenarians in a number of developed countries (Brandão et al., 2017; Engberg et al., 2009). Thus, the exposure to efficient health care systems allows them to delay their physiological failure (Seals et al., 2016). In addition to poor health, we found that mortality among centenarians is also being postponed (see Figure 8 in *SI Appendix*). Therefore, centenarians living further more years (half of them survive beyond $\bar{e}_{100}$, see Figure 6 in *SI Appendix*) prolong their debility for longer periods and postpone their deaths on repeated occasions through timely medical interventions, which are a consequence of robust health systems in countries doing the best. This mechanism leads to mortality declines observed across cohorts (see Figures 3, 4 and 5).

There is no doubt that health systems are central on the postponement of ageing, nonetheless, other factors rooted in social behaviours play an important role in this phenomena. For instance, recent studies suggest that individuals aged 70+ who experience frequent physical activity, cardiovascular training (Voelcker-Rehage et al., 2011) as well as novelty tasks during their work time (Oltmanns et al., 2017) depict notable improvements about their cognitive functioning and most importantly, they are capable to

delay the effect of brain ageing. Recently, fasting and reduction of calorie intake have also been found to be determinants in the postponement of diseases and ageing among the elderly (Di Francesco et al., 2018). These behaviours are pointed out to be rooted in evolution since they are natural phenomena to which humans and other organisms (such as unicellular yeast, nematode worms, fruitflies, mice, and primates) were exposed regularly (Mattson et al., 2017; Speakman and Mitchell, 2011).

Contrary to reductions in the calorie intake, some other social behaviours entrenched in modern populations' lifestyles have proven to be detrimental factors in the survival of individuals. This is the case of smoking, which has become an epidemic in high income countries (Glei and Horiuchi, 2007). Smoking is responsible for the death of thousands of individuals in their middle ages and it has become a major public health issue. This behaviour might be responsible of the female survival advantage in comparison to males affecting longevity outcomes since it impedes a great number of males to survive to age 100 or beyond. Likewise, it is likely that the burden of smoking-attributable deaths is reflected on the stagnation of British $\bar{e}_{100}$ and the Nordic countries (Drefahl et al., 2012; Modig et al., 2017).

Biology is at the heart of individuals' ability to react to external factors and postpone their deaths. In this sense, hormesis has been pointed out to be the biological mechanism behind such responses, which are central components of normal development, growth, maintenance, repair and ageing activities (Calabrese et al., 2015; Vaiserman, 2010). This mechanism suggests that, when subjected to low levels of stress, organisms regulate and constrain the allocation of biological resources leading to optimal uses of them. Thus, the biological plasticity of ageing may be described by the quantitative features of hormetic responses. Exposures to short-term stress can strengthen cellular responses promoting longevity. Recent studies have put forward evidence in favour of the association between extreme longevity and an increased number of stress response molecules as an attempt of cell to cope with stress (Calabrese et al., 2015; Conte et al., 2018). Hormesis can, therefore, be seen as the fundamental component of biological adaptability, neutralizing many exogenous challenges (Epel and Lithgow, 2014). External sources of hormetic responses in longevous individuals cover stress from physical exercise, reductions in the calorie uptake, glucose restriction as well as low-dose viral and bacterial pathogens affecting individuals (Manton and Yashin, 2000).

The malleability in the risk of dying of centenarians entails that these individuals are highly responsive to external factors. However, such factors are modulated differently across individuals by social behaviours and by country-specific determinants such as public health systems, standards of living, housing and sanitation conditions among others (Riley, 2005). This affects in different manners the physiology of individuals deriving into a wide spectrum of vitality trajectories (Li and Anderson, 2009; Wachter et al., 2014). At the population level, mortality declines are the outcome of postponing deaths of individuals with different vitality trajectories. Thus, extensions in $\bar{e}_{100}$ result in higher lifespan variability. Likewise, mortality improvements achieved at individual level are masked by population heterogeneity. Certainly, when we control for unobserved heterogeneity on frailty, our results indicate that the progress in reducing mortality is more pronounced for individuals than the observed progress at the population level. This indicates that heterogeneity is preventing populations from further declines in mortality.

This study provides evidence about the outstanding ability of longevous individuals to postpone their deaths to even high advanced ages. Further, our findings about the attained reductions in the risk of dying above age 100 provide evidence that not only the tail of longevity has extended over time but also that these populations exhibit the necessary conditions to achieve further extensions. Such conditions are framed by individual's biological endowments, social behaviours and their interaction with external factors. Therefore, it is utterly important to develop interdisciplinary approaches to enhance our knowledge about the mechanisms behind the plasticity of human longevity.

Materials and methods

Data

Cohort data from the HMD (Human Mortality Database, 2017) were used in this study. This database includes high-quality information on death rates and life tables' functions (e.g., life expectancy) for more than 40 populations. The HMD supplies underlying raw data used in the calculation of mortality estimates (i.e., births, ages at death, census counts, official population estimates and extinct-cohort estimates of age-specific population sizes). In this investigation, we use raw data that comprehends complete birth cohorts between 1860 and 1904. We analysed sex-specific data grouped into 10-year time intervals. For Japan, data are available for birth cohorts between 1870 and 1904, which shortens the period of study in this country.

The quality of mortality data at advanced ages is a serious concern (Black et al., 2017). Several studies have evaluated the data quality above age 80 (Coale and Kisker, 1986; Kannisto, 1994, 1999; Wang et al., 1998; Wilmoth and Lundström, 1996). In particular, Jdanov et al. (Jdanov et al., 2008) scrutinized the quality of old age mortality data available at the HMD. They looked at several age-overstatement and age-heaping indicators to identify potential data quality issues for each country. In this study, we solely considered those countries that exhibit high data quality according to the expert of opinion of Jdanov et al. (2008) and a total population size over 50 million. We excluded Germany since they went through a process of reunification between East and West Germany. Thus, the analysed countries are France, Great Britain (England and Wales excluding Scotland), Italy and Japan. This set of countries exhibit reliable estimates of death counts and almost no deviations between the official population estimates. We performed the analysis by considering sex specific 10-year cohorts. Nonetheless, due to the scarcity of male individuals surviving to age 100, our estimates for male populations depict wide confidence intervals. Therefore, we are unable to draw precise conclusions about mortality of male centenarians. We centre our analysis on females trends.

Methods

In the study of mortality schedules at high advanced ages it is utterly important to control for unobserved heterogeneity and distinguish between mortality trajectories for the individuals and for entire population. We discern between both types of measures by adding a bar on top of the population ones. For instance, $\bar{\mu}(x,y)$ represents the mortality hazard at age x for the cohort y for the entire population, whereas $\mu(x,y)$ is the homologous measure for individuals. Similarly, $\bar{e}(x,y)$ and $e(x,y)$ depict life expectancy for the total population and for individuals.

We use a dot over a variable to denote the derivative with respect to time of the mortality hazards. Hence, the time derivative of the hazard for individuals is expressed as:

$$\dot{\bar{\mu}}(x,y) \equiv \frac{\partial \bar{\mu}(x,y)}{\partial y}.$$

An acute accent is used over a variable to represent the relative derivative with respect to time such that:

$$\acute{\bar{\mu}}(x,y) \equiv \frac{\frac{\partial \bar{\mu}(x,y)}{\partial y}}{\bar{\mu}(x,y)}.$$

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Therefore, the rate of mortality improvement at age x for cohort y is defined as minus the relative derivative of $\bar{\mu}(x,y)$ with respect to y:

$$\bar{\rho}(x,y) = -\frac{\dot{\bar{\mu}}(x,y)}{\bar{\mu}(x,y)} = -\acute{\bar{\mu}}(x,y). \tag{1}$$

Population measures of longevity: life expectancy and lifespan variability at age 100

At the population level, life expectancy at age 100 is denoted to as:

$$\bar{e}_{100} \equiv \bar{e}(100,y) \equiv \frac{\int_{100}^{\omega} l(x,y)dx}{l(100,y)}, \tag{2}$$

where $l(100,y)$ is proportion surviving at age x and cohort y.

We also estimated the change across cohorts in $\bar{e}_{100}$ by computing its derivative with respect to the variable of time depicted by y:

$$\dot{e}_{100} \equiv \dot{e}(100,y) \equiv \frac{\partial e(100,y)}{\partial y}.$$

The shape of lifespans after age 100 was measured by computing life disparity and Keyfitz's entropy. Life disparity (denoted to as $\bar{e}^{\dagger}_{100}$) is defined as the average remaining life expectancy at the age when death occurs, or alternatively the average years of life lost in a population attributable to death Vaupel (1986); Vaupel and Canudas-Romo (2003). At age 100, this measure is defined as:

$$\bar{e}^{\dagger}_{100} \equiv \bar{e}^{\dagger}(100,y) \equiv \frac{\int_{100}^{\omega} d(x,y)\bar{e}(x,y)dx}{l(100,y)}, \tag{3}$$

where $d(x,y)$ is the distribution of lifespans at age x and time y and $e(x,y)$ denotes the distribution of remaining life expectancies.

Keyfitz's entropy ($\bar{H}$) is defined as the elasticity of life expectancy with respect to mortality change (Keyfitz and Caswell, 2005). At age 100 it is calculated as:

$$\bar{H} \equiv \bar{H}(100,y) \equiv \frac{\bar{e}^{\dagger}(100,y)}{\bar{e}(100,y)} \tag{4}$$

Estimating the links between individuals and the entire population

The model for individuals depends on the random latent variable Z called frailty Vaupel et al. (1979b). The general relationship between $\bar{\mu}(x,y)$ and $\mu(x,y)$ is given by $\bar{\mu}(x,y) = \bar{z}(x)\mu(x,y)$ where $\bar{z}(x)$ is the average frailty among survivors to age x. If Z is assumed to follow a Gamma distribution and the Gompertz law of mortality to be the baseline hazard, then the population hazard follows a Gamma-Gompertz (ΓG) distribution:

$$\bar{\mu}(x,y) = \frac{\alpha e^x}{1 + (\frac{\alpha\gamma}{\beta})(e^{\beta x} - 1)}. \tag{5}$$

The ΓG has been pointed out several times to be the most adequate and meaningful demographic model describing mortality at high advanced ages Feehan (2018); Missov and Finkelstein (2011); Missov et al. (2016); Missov and Vaupel (2015); Steinsaltz and Wachter (2006). Here we fitted the ΓG model to 10-year cohort death rates in a standard Poisson maximum-likelihood framework by maximizing the equation:

$$\ln L(y) = \sum_{x \geq 80} [D(x,y) \ln \bar{\mu}(x,y) - E(x,y)\bar{\mu}(x,y)], \tag{6}$$

where $D(x,y)$ and $E(x,y)$ are observed death counts and exposures at age x and cohort y respectively and $\bar{\mu}(x,y)$ is the corresponding hazard. In order to be consistent with the HMD Human Mortality Database (2017) procedures, we fitted the model starting at age $x = 80$. We calculated 95% confidence intervals by using a delta-method Efron and Tibshirani (1986).

Our estimates of the parameters α, β and γ vary among countries and across cohorts. Therefore, we test the stability of our estimates through rolling-window analyses by fitting the ΓG model to windows of 10 and 20 consecutive cohorts for every country. The iteration was performed by moving forward the rolling-window one year starting in 1860 until the last observed cohort. The resulting estimates of the ΓG parameters can be found in Table 1 in *SI Appendix*. Likewise, we used rolling-window estimates to compute the trajectory of $\bar{e}_{100}$ over time and the associated $\dot{e}_{100}$ (Figures 10 and 11 in *SI Appendix*)

In a ΓG setting, the link between population and individual hazards Vaupel (2002); Vaupel and Missov (2014) is given by

$$\bar{\mu}(x,y) = \mu(x,y)[\bar{s}_c(x,y)]^{\gamma}, \tag{7}$$

where $\bar{s}_c(x,y)$ is the cohort survivorship for the population at age x and time y, and γ is the coefficient of variation of the random variable frailty Z at any age x obtained from Equation 5. Likewise, the relationship between $\bar{\rho}(x,y)$ and $\rho(x,y)$ is given by the following equation Vaupel and Missov (2014):

$$\bar{\rho}(x,y) = \rho(x,y)[\bar{s}(x,y)]^{\gamma}. \tag{8}$$

Since we fit the ΓG model at age 80, we assume that at that age the initial value of frailty is $Z = 1$. In consequence, $\bar{\rho}(80,y) = \rho(80,y)$, which implies that $\bar{\rho}(x,y)$ and $\rho(x,y)$ for $x > 80$ are conditional to values of $\bar{\rho}(80,y)$ such that:

$$\frac{\bar{\rho}(x-80,y)}{\bar{\rho}(80,y)} = \frac{\rho(x-80,y)\bar{s}_c(x-80,y)^{\gamma}}{\rho(80,y)}, \tag{9}$$

we use Equation 9 to estimate rates of mortality improvements for individuals. Values for γ were obtained from the rolling-window analysis (see Table 1 in *SI Appendix*)

Figures
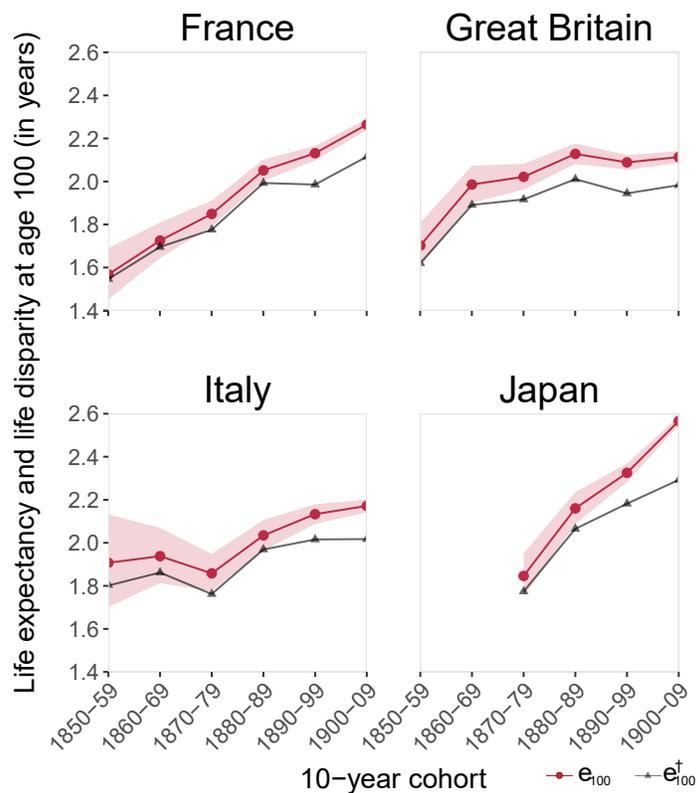
Figure 1: Cohort life expectancy and life disparity at age 100 for 10-year cohorts of females born between 1850 and 1904. Red lines represent the trajectories of life expectancy at age 100 for the population and its 95% confidence intervals. Gray lines depict the associated values for lifespan disparity.
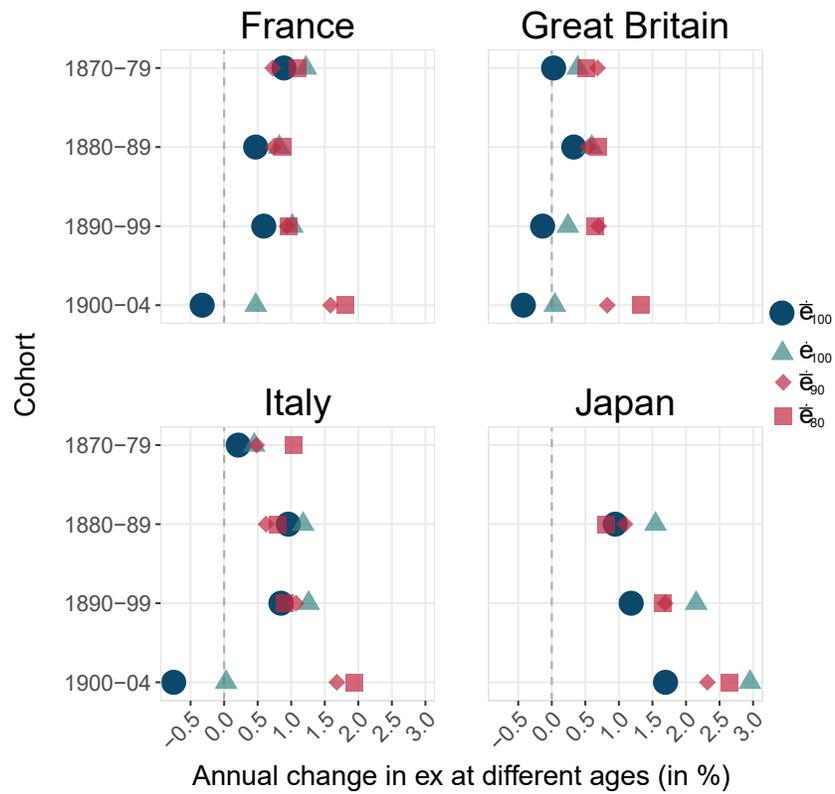
Figure 2: Speed of change in female $\bar{e}_x$ for different ages. Blue dots represent $\ddot{e}_{100}$, whereas green triangles, red diamonds and red squares depict $\dot{e}_{100}$, $\dot{e}_{90}$ and $\dot{e}_{80}$ respectively. Values to the right (left) of the vertical dashed line portray increases (decreases) in $\bar{e}_x$.
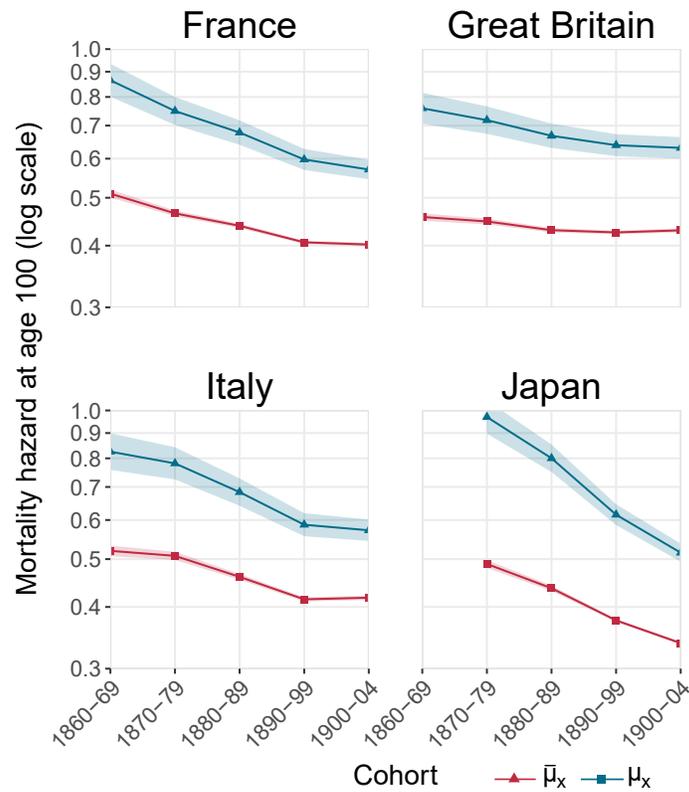
Figure 3: Mortality hazards at age 100 across cohorts of females born between 1860 and 1904. Blue lines represent the trajectories of mortality hazards for the entire population and red lines depict the estimated trajectories for individuals.
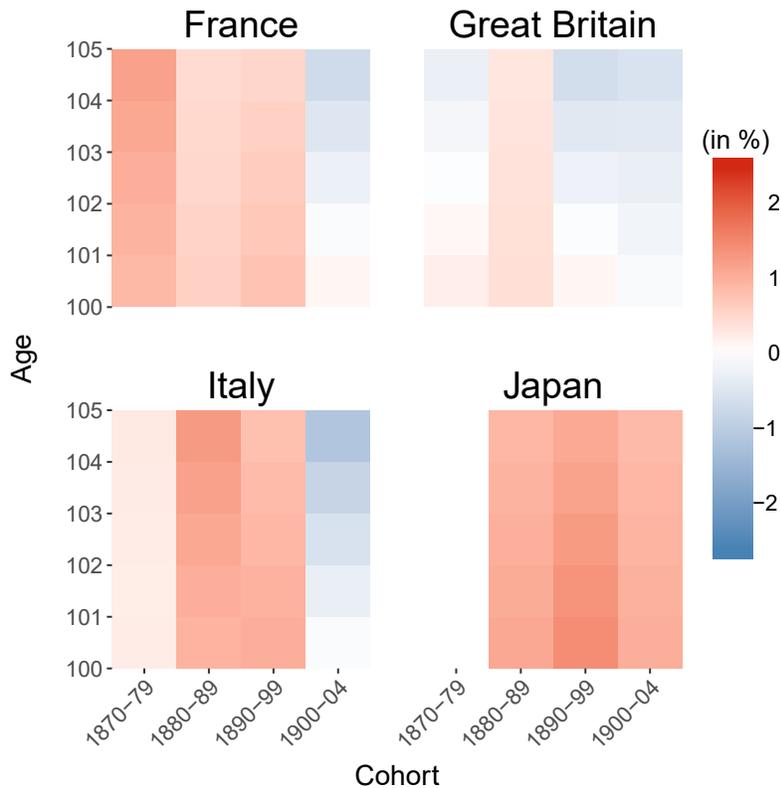
Figure 4: Heatmap of annual rates of mortality improvement for the entire population.
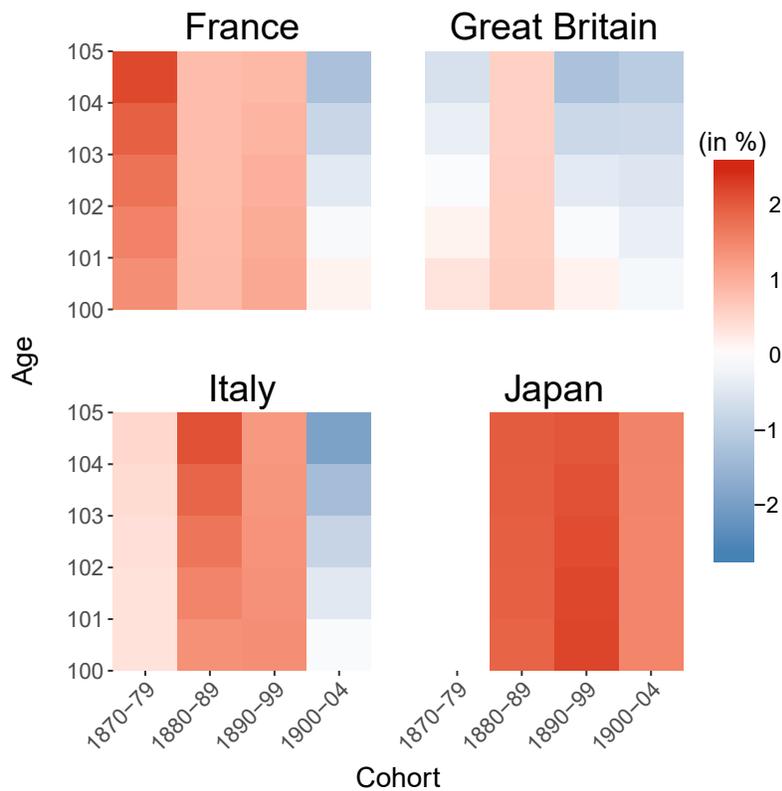
Figure 5: Heatmap of estimated annual rates of mortality improvement for individuals. Estimates are performed based on the estimated mortality levels for individuals at age 80. See *Materials and methods* for a detailed description of the estimation procedure.

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Supporting information
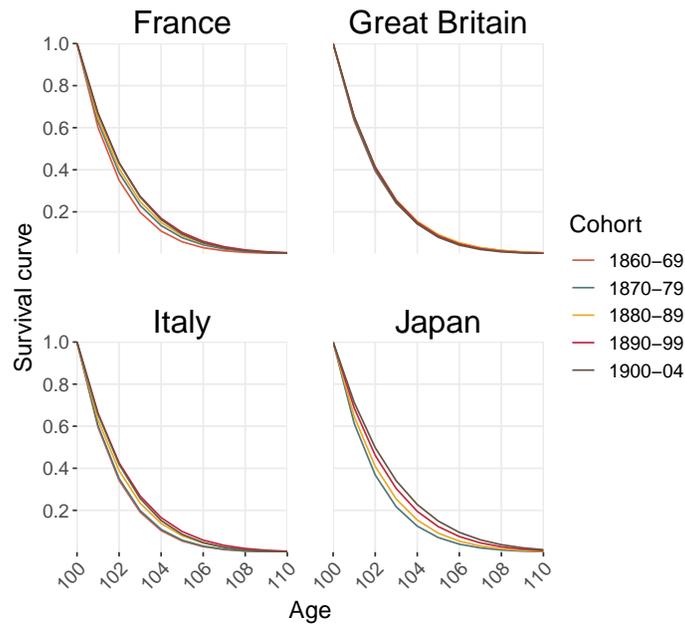
Figure 6: Cohort survivorship above age 100. Females, 1860-1904.
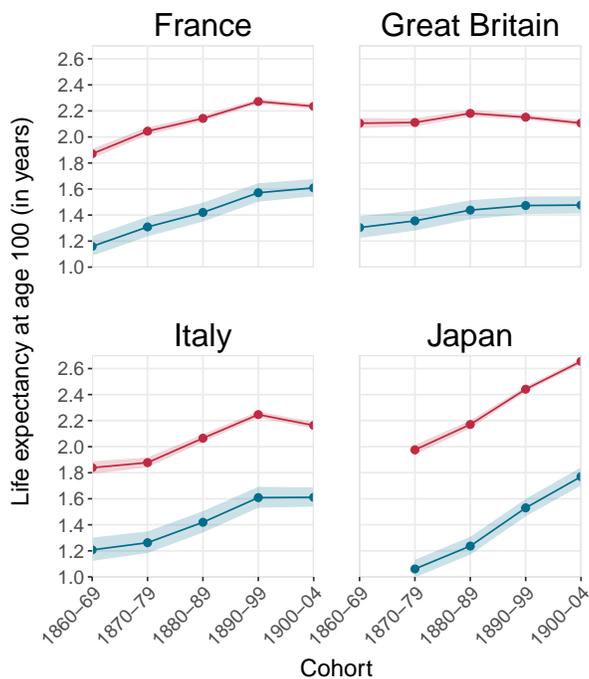
Figure 7: Cohort life expectancy at age 100 for individuals and for the total population. Red lines represent the trajectories of $\bar{e}_{100}$ for the population and its 95% confidence intervals. Blue lines depict the associated values for individuals.
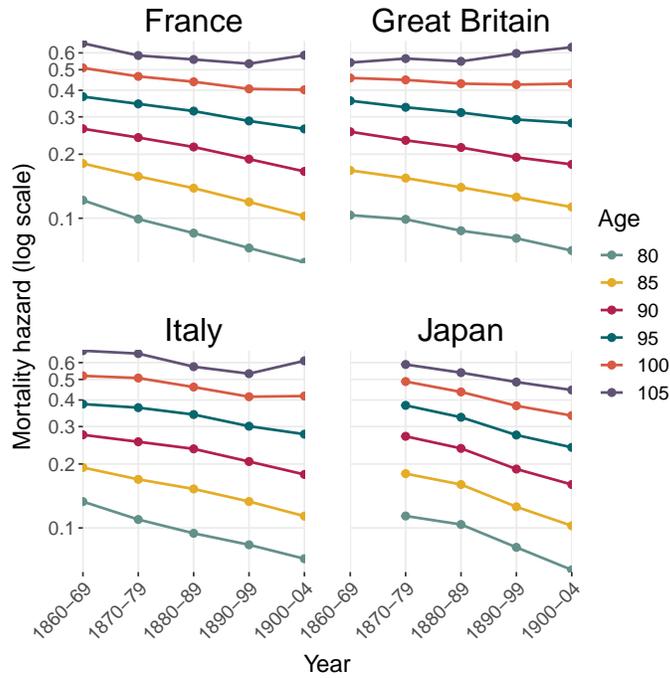
Figure 8: Mortality hazards above age 80. Females, 1860-1904
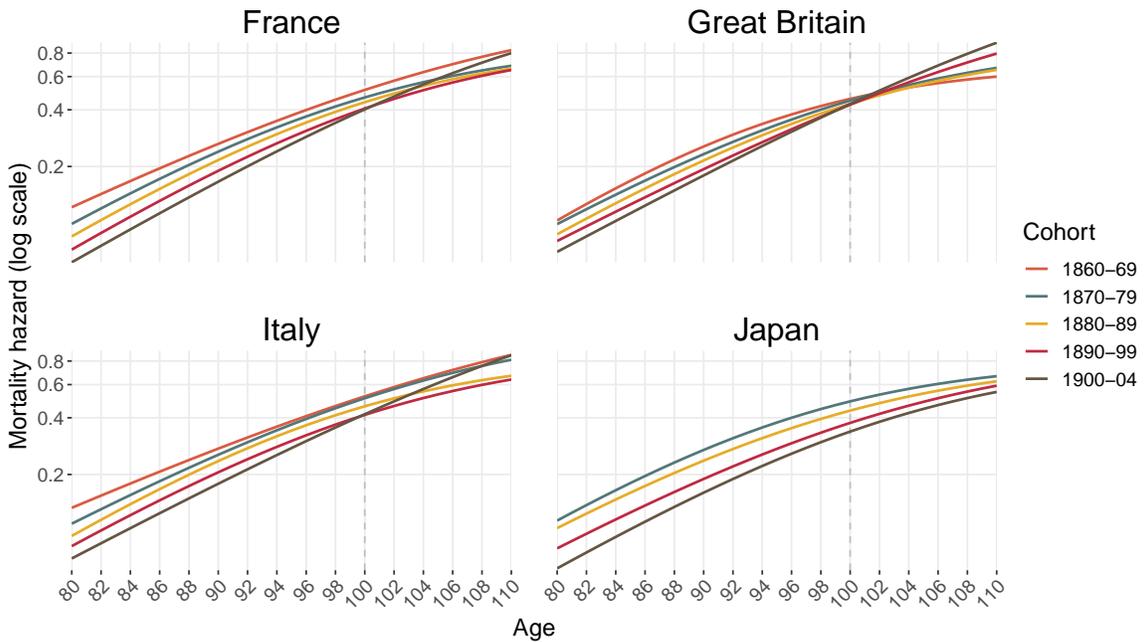
Figure 9: Fitted mortality hazards. Females, 1860-1904
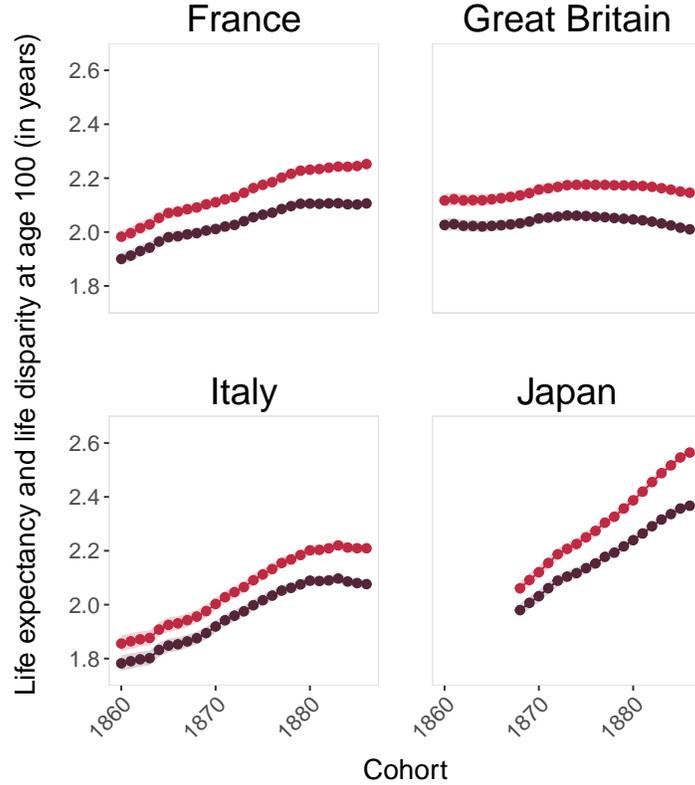
Figure 10: Cohort life expectancy and life disparity at age 100 calculated from the estimates of the 20-year rolling-window analysis. Red dots represent the trajectories of life expectancy and grey dots depict the associated values for lifespan disparity.

	Non-overlapping cohorts			20-year cohort rolling window		
Country	$\hat{\alpha}$	$\hat{\beta}$	$\hat{\gamma}$	$\hat{\alpha}$	$\hat{\beta}$	$\hat{\gamma}$
France	0.084 (0.020)	0.105 (0.008)	0.097 (0.030)	0.088 (0.014)	0.106 (0.006)	0.112 (0.016)
Great Britain	0.084 (0.011)	0.105 (0.009)	0.099 (0.050)	0.088 (0.008)	0.104 (0.004)	0.108 (0.025)
Italy	0.094 (0.021)	0.099 (0.011)	0.082 (0.038)	0.098 (0.014)	0.101 (0.008)	0.100 (0.026)
Japan	0.086 (0.019)	0.105 (0.005)	0.126 (0.010)	0.091 (0.010)	0.102 (0.004)	0.127 (0.009)

Table 1: Mean and standard deviations of the ΓG parameters across cohorts. The parameters were estimated for non-overlapping 10-year cohorts (shown in the main results of this article) and for 20-year rolling-window analyses. Mean values were computed across cohorts for each country. Standard deviations are shown in parenthesis.
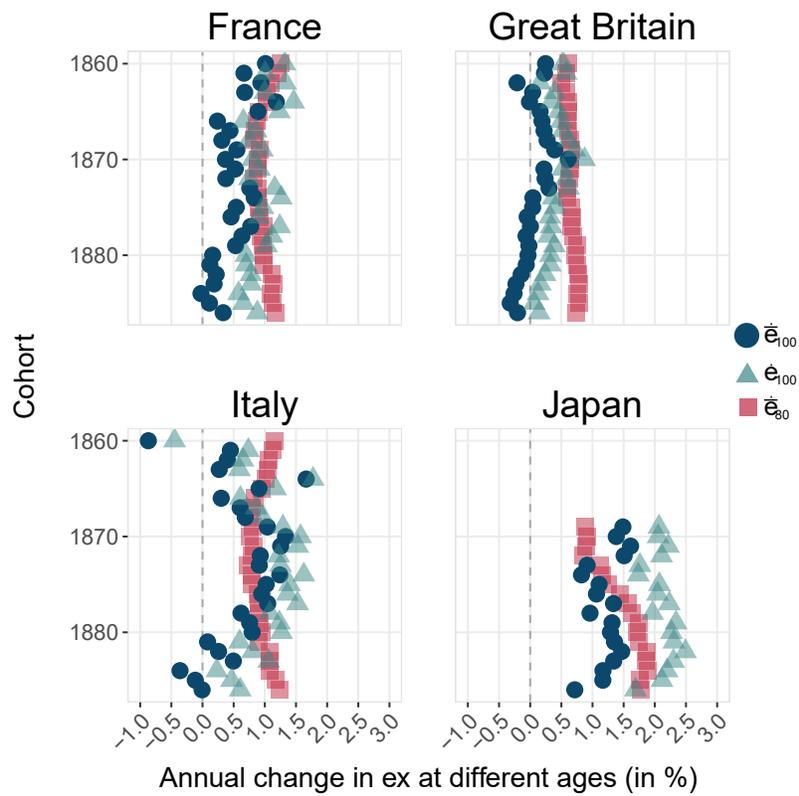
Figure 11: Speed of change in $\bar{e}_x$ calculated from the estimates of the 20-year rolling-window analysis. Blue dots represent $\ddot{\bar{e}}_{100}$, whereas green triangles and red squares depict $\dot{e}_{100}$ and $\dot{e}_{80}$ respectively. Values to the right (left) of the vertical dashed line portray an increase (decrease) of $\bar{e}_x$.