

Interdisciplinary perspectives on human longevity

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Para mis padres

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Summary

For more than two centuries, deaths have been shifted from young to old ages, and more recently, from old to even older ages. Individuals now live much longer than they did previously, and the number of people surviving to the oldest ages continues to grow. The rise of longevity is one of the greatest achievements of humankind. However, our understanding about the longest lifespans is still limited. It is unknown, for example, if centenarians are living longer, or if it is possible for them to live longer. It is also unknown if these individuals share common traits or if their notable lifespans are random events. The prospect of extending longevity hinges on the survival trajectories of the individuals who live the longest.

The rise of longevity also poses unprecedented challenges to societies. An increasing number of elderly people jeopardizes the financial sustainability of pension schemes and puts pressure on healthcare systems. Moreover, not everyone survives to an advanced age, as extensions in longevity are not evenly distributed across populations. Females have a greater chance of becoming centenarians when compared to males, as do higher socio-economic groups when compared to lower socio-economic groups. The inherent inequality in longevity is translated into fundamental disadvantages for some individuals. The sources and implications of such disadvantages require to be scrutinized.

The present PhD dissertation comprises five interdisciplinary studies aimed at advancing our understanding about human longevity. The first and second studies examine regularities in lifespans of the longest lived individuals. In the first study, we examine an unprecedentedly large and reliable dataset comprising lifespans of individuals aged 105 and above in eight populations worldwide. We show that similar demographic patterns arise across all populations analysed. Evidence becomes more clear-cut in support of a levelling-off of the risk of dying as a regularity of longevous populations.

In the second study, health profiles and survival trajectories of Danish centenarians are examined. We address hidden heterogeneity in health profiles and show the existence of a super-select group of centenarians that outperform in different health dimensions and survive the longest. This group of centenarians is responsible for extending the frontier of human longevity.

The third and fourth studies focus on inequalities in longevity. The third study explores the financial and demographic implications of unequal lifespans after retirement. Specifically, we analyse a recently implemented pension policy in Denmark that links retirement ages to gains in life expectancy. We bring together actuarial and demographic methods and show that this type of policy has detrimental implications for males from lower socio-economic groups. The fourth study examines the sources of the sex gap in old-age survival. Using epidemiological models, we show that smoking-attributable deaths have contributed substantially to sex differences in old-age survival in high-income populations all around the world.

Finally, the fifth study examines the rise of longevity under a novel perspective. We propose a framework to study longevity in terms of *survivorship-ages*, rather than in chronological ages. We show that over more than 100 years, the relationship between the age variable and survival has changed, while the relationship between survival and the risk of dying is remarkably stable.

Resumé

I mere end to århundreder er dødsfald gået fra ung til gammel alder, og for nylig fra gammel til endnu ældre alder. Mennesker lever nu meget længere end de gjorde tidligere, og antallet af mennesker, der lever til de når de allerældste aldre fortsætter med at stige. Fremgangen af lang levetid er en af menneskehedens største bedrifter. Vores forståelse af de længste levetider er dog stadig begrænset. Det vides f.eks. ikke, om hundredårige lever længere end tidligere eller om det er muligt for dem at leve endnu længere. Det er også ukendt om disse personer har fællestræk, og om deres bemærkelsesværdige levetid skyldes tilfældige begivenheder. Udsigten til at forlænge levetiden afhænger af overlevelsesbanerne for de personer, der lever længst.

Fremgangen af lang levetid giver hidtil usete udfordringer for samfundet. Et stigende antal ældre bringer pensionernes økonomiske bæredygtighed i fare, og lægger dermed et pres på sundhedssystemerne. Desuden lever ikke alle til en fremskreden alder, da forlængelse af lang levetid ikke er jævnt fordelt på befolkningen. Kvinder har større chance for at blive hundredårige sammenlignet med mænd. Ligeledes har højere socioøkonomiske grupper større chance for at blive hundredårige sammenlignet med lavere socioøkonomiske grupper. Den iboende ulighed i lang levetid oversættes til grundlæggende ulemper for nogle individer. Årsager og konsekvenser af sådanne ulemper skal undersøges.

Denne ph.d.-afhandling består af fem tværfaglige artikler, der har til formål at fremme vores forståelse af menneskets levetid. De to første artikler i samlingen undersøger regelmæssigheder i levetider hos de længstlevende individer. I den første artikel undersøgte vi et hidtil uset, stort og pålideligt datasæt bestående af levetider for personer i alderen 105 år og derover i otte befolkninger over hele verden. Vi viser at lignende demografiske mønstre opstår i alle analyserede populationer og at dette mønster bliver tydeligere jo mere data, der anvendes.

I den anden artikel undersøges sundhedsprofiler og overlevelsесforløb for danske hundredårige. Vi adresserer skjult heterogenitet i sundhedsprofiler og påviser, at der eksisterer en særlig udvalgt gruppe af hundredårige, der overgår de forskellige sundhedsdimensioner og dermed lever længst. Denne gruppe af hundredårige er årsagen til at grænsen for menneskelig levetid udvides.

Den tredje og fjerde artikel fokuserer på uligheder i lang levetid. I den tredje artikel undersøges de økonomiske og demografiske konsekvenser af ulig levetid efter individerne er gået på pension. Helt konkret analyserer vi en nylig implementeret pensionspolitik i Danmark, der forbinder pensionsalder med gevinst i forventet levealder. Vi anvender aktuarmæssige og demografiske metoder og viser, at denne type politik har skadelige konsekvenser for mænd fra lavere socioøkonomiske grupper. Den fjerde artikel undersøger årsagerne til kønsforskelle i alderdomsoverlevelse. Ved hjælp af epidemiologiske modeller viser vi, at dødsfald, der kan tilskrives rygning, har bidraget væsentligt til kønsforskelle i alderdomsoverlevelse i højindkomstpopulationer overalt i verden.

Endelig undersøger den femte – og sidste – artikel stigningen i lang levetid under et nyt perspektiv. Vi foreslår en ramme til at studere levetid i form af *survivorship-ages* i stedet for kronologiske aldre. Vi viser, at forholdet mellem aldersvariabel og overlevelse har ændret sig i mere end 100 år, mens forholdet mellem overlevelse og risikoen for at dø er bemærkelsesværdig stabil.

List of manuscripts

This thesis is based on five manuscripts:

1. **Alvarez, J. A.**, Villavicencio, F., Strozza, C, & Camarda, C.G. (2021). *Regularities in human mortality after age 105*. Accepted for publication in PLOS One.
2. **Alvarez, J. A.**, Medford, A. Strozza, C. Thinggaard, M, & Christiensen, K (2021) *Stratification in health and survival among Danish centenarians*. BMC Geriatrics, 21:406.
3. **Alvarez, J. A.**, Kallestrup-Lamb, M., & Kjærgaard, S. (2021). *Linking retirement age to life expectancy does not lessen the demographic implications of unequal lifespans*. Insurance: Mathematics and Economics, 99, 363-375.
4. Wensink, M., **Alvarez, J. A.**, Rizzi, S., Janssen, F., & Lindahl-Jacobsen, R. (2020). *Progression of the smoking epidemic in high-income regions and its effects on male-female survival differences: a cohort-by-age analysis of 17 countries*. BMC Public Health, 20(1), 1-8.
5. **Alvarez, J. A.** & James W. Vaupel (2021). *Mortality as a function of survivorship*. In preparation for submission.

Other publications I co-authored during the realization of my PhD, which are not included in this dissertation:

- Baudisch, A., & **Alvarez, J. A.** (2021). *Born once, die once: Life table relationships for fertility*. Demographic Research, 44, 49-66.
- **Alvarez, J. A.**, Kallestrup-Lamb, M., Kjærgaard, S., & Vaupel J. W. (2020). *Trends in lifespan inequality after retirement*. In Living to 100 Monograph, Society of Actuaries.
- **Alvarez, J. A.**, Aburto, J. M., & Canudas-Romo, V. (2020). *Latin American convergence and divergence towards the mortality profiles of developed countries*. Population studies, 74(1), 75-92.
- Aburto, J. M., **Alvarez, J. A.**, Villavicencio, F., & Vaupel, J. W. (2019). *The threshold age of the lifetable entropy*. Demographic Research, 41, 83-102.
- Bergeron-Boucher, M. P., Pascariu, M. D., Aburto, J. M., **Alvarez, J. A.**, Basellini, U., Rizzi, S., & Vaupel, J. W. (2020). *Alternative Forecasts of Danish Life Expectancy*. In Developments in Demographic Forecasting (pp. 131-151). Springer, Cham.

Chapter 1

Introduction

Human longevity has risen. Deaths have been postponed from young to older ages. The probability of surviving has gone up at all ages but mostly at young ages where most mortality improvements have been achieved (Burger et al., 2012). Life expectancy has gone up (Medford, 2017; Oeppen and Vaupel, 2002) and the highest life expectancies are currently observed in Hong Kong and Japan¹ (Human Mortality Database, 2021). At the same time, the rise of life expectancy is accompanied by a decrease in the inequality in the length of life (Aburto et al., 2020; Colchero et al., 2016; Permanyer and Scholl, 2019; Vaupel et al., 2011), which indicates that people live longer lives and their lifespans become more similar.

The rise of longevity is attributed to several factors. Ongoing improvements in medical treatments help more and more people to survive to advanced ages (Christensen et al., 2009). Public health efforts that enhance living conditions and prevent diseases have been crucial in increasing longevity (Vaupel et al., 2021). In developed populations, diseases amenable to health care have gone down and child and infant mortality are at their lowest levels (Alvarez et al., 2020). The rise of longevity has been extensively documented (Oeppen and Vaupel, 2002; Vaupel, 2010; Vaupel et al., 2021). Still, it is unclear how much progress in reducing mortality at high advanced ages is being achieved and can be achieved. The prospect of extending longevity hinges on increases in lifespans of those individuals who live the longest. Increasing longevity has implications for societies and specifically for pension systems. In the context of recent pension reforms aiming at control for the effect of rising life expectancy (OECD, 2019b), a closer look at demographic trends contributes insights into the inherent risks in pension schemes arising from longevity dynamics.

The main objective of this PhD thesis is to enhance our knowledge about the increasing pattern of longevity. First, we examine mortality patterns after age 100 and some of the underlying mechanisms that drive these patterns. Next, we analyse some of the implications of socio-economic inequalities in longevity for pension systems and the sources of the sex gap in old age survival. Finally, we propose a new perspective to analyse mortality as a function of survivorship, which provides important insights into the core dynamics of mortality.

¹In 2017, life expectancy was 87.79 for females in Hong Kong and 87.31 for Japanese females (Human Mortality Database, 2021)

In this introduction I² first outline some background for the research included in this thesis. I present key concepts and empirical results that motivated this work. Next, I discuss salient issues that hinder the analysis of the longest lifespans. Finally, I explain how the studies included in this dissertation contribute insights into human longevity.

1.1 Mortality improvements

In this section I introduce some key indicators that are fundamental to the research presented in this PhD thesis. Let X be the random variable that denotes the age at death of the individual. The risk of dying at age x , $\mu(x)$, is defined as the instantaneous rate of death, conditioned upon survival to that age, such that:

$$\mu(x) = \lim_{\Delta x \rightarrow 0} \frac{\Pr(x \leq X < x + \Delta x | X \geq x)}{\Delta x}. \quad (1.1)$$

The risk of dying³ together with the survival function, $S(x)$, and density function, $f(x)$, are fundamental indicators that describe a mortality regime in a specific population (Dickson et al., 2013; Preston et al., 2000). Given that mortality is a process that changes over time t , we denote $\mu(x,t)$ as the the risk of dying at age x and time t . Changes over time in this indicator are captured by the rate of mortality improvement, $\rho(x,t)$:

$$-\frac{\frac{\partial \mu(x,t)}{\partial t}}{\mu(x,t)} = -\frac{\dot{\mu}(x,t)}{\mu(x,t)} = -\acute{\mu}(x,t) = \rho(x,t). \quad (1.2)$$

Positive values of $\rho(x,t)$ indicate mortality improvements and negative values imply deterioration. Throughout this thesis, we denote time derivatives by adding a dot on top of the indicator of interest and relative derivatives by adding a acute accent as in Equation (1.2).

²I write in first person whenever the text describes my motivations to perform research or expresses my own choices. I use *we* to refer to the joint research performed with co-authors. I also employ *we* whenever the discourse is explanatory, such as the description of equations or figures. In this context, *we* stands for *me and the reader*.

³It is also called the hazard function, force of mortality or mortality intensity. In this Chapter I choose to call it risk of dying because it is a more intuitive way of thinking about this indicator.

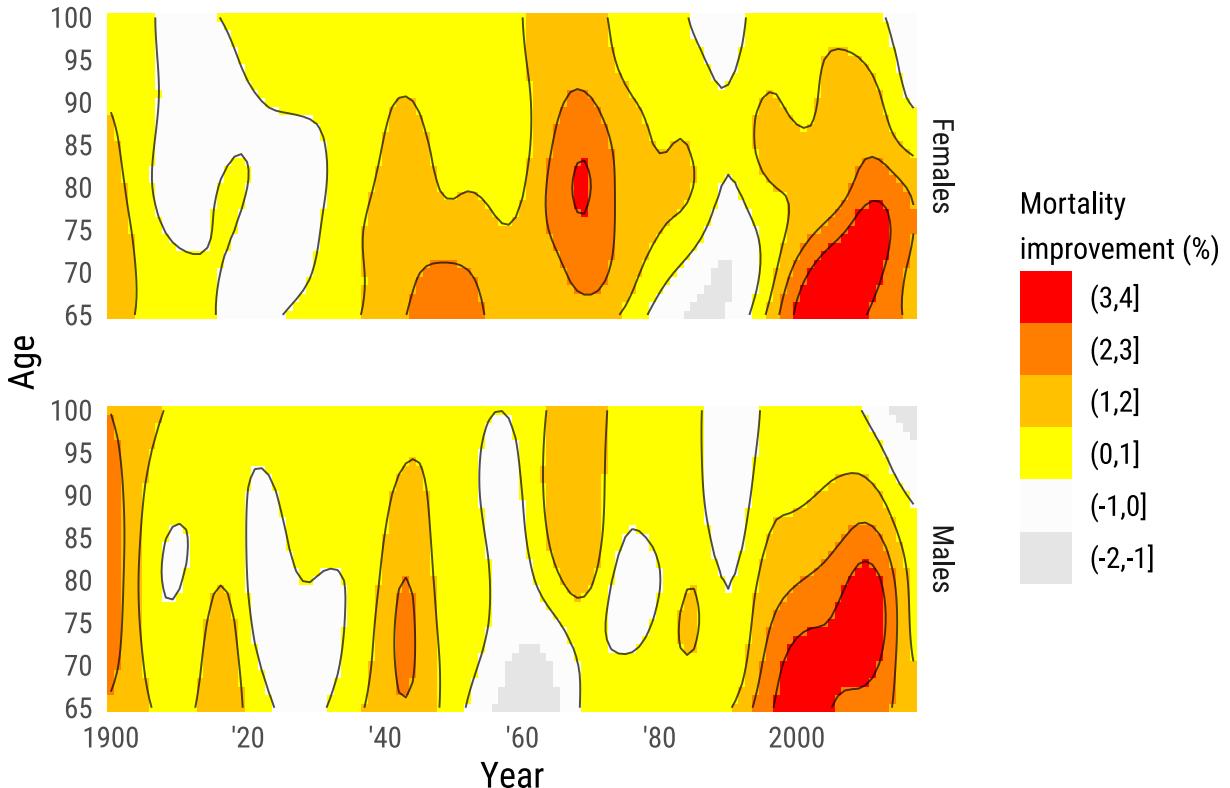


Figure 1.1: Rates of mortality improvement after age 65. Denmark, both sexes, 1900-2019.
Source: *Own elaboration based on the data obtained from the Human Mortality Database (2021)*.

Figure 1.1 shows rates of mortality improvement for Denmark between ages 65 and 100 during the period 1900-2019. We can observe that mortality has improved during most of the observation period. Since the year 2000, annual improvements are around 3% and 4% for ages 65 to 80. However, mortality deterioration is also observed during some periods. It is clear that for Danish females, mortality deteriorated during the decade of the '80s and at the beginning of the '90s. This is largely explained by smoking attributable deaths for those women born in the years between the two World Wars (Lindahl-Jacobsen et al., 2016a,b). The so-called *smoking epidemic* has taken a large toll of deaths in Denmark and in other high income countries (Janssen, 2020).

Mortality improvements, such as the ones shown in Figure 1.1, are important for analysis because they are the key drivers of increases in life expectancy. Indeed, Vaupel and Canudas-Romo (2003) showed that mortality improvements contribute to changes over time in life expectancy $e(x,t)$ as:

$$\dot{e}(x,t) = \frac{\partial e(x,t)}{\partial t} = \bar{\rho}(t)H(x,t), \quad (1.3)$$

where $\bar{\rho}(x,t)$ is the average rate of mortality improvement after age x such that

$$\bar{\rho}(x,t) = \frac{\int_x^\infty \rho(y,t)e(y,t)f(y,t)dy}{\int_x^\infty e(y,t)f(y,t)dy}, \quad (1.4)$$

and $H(x,t)$ is the lifetable entropy⁴:

$$H(x,t) = -\frac{\int_x^\infty \ln(s(y,t))s(x,t)dy}{\int_x^\infty s(y,t)dy}. \quad (1.5)$$

The entropy $H(x,t)$ can be interpreted as the elasticity of life expectancy to changes in the risk of dying at all ages above x (Aburto et al., 2019; Keyfitz, 1977; Leser, 1955). For example, if $H(x,t) = 0.4$, then a decrease of one percent in the risk of dying at all ages above age x will trigger an increase of 0.4 percent in $e(x,t)$ (Goldman et al., 1986; Keyfitz and Caswell, 2005). Thus, Equation (1.3) indicates that changes in life expectancy are driven by mortality improvements, which at the same time, are modulated by the elasticity of life expectancy.

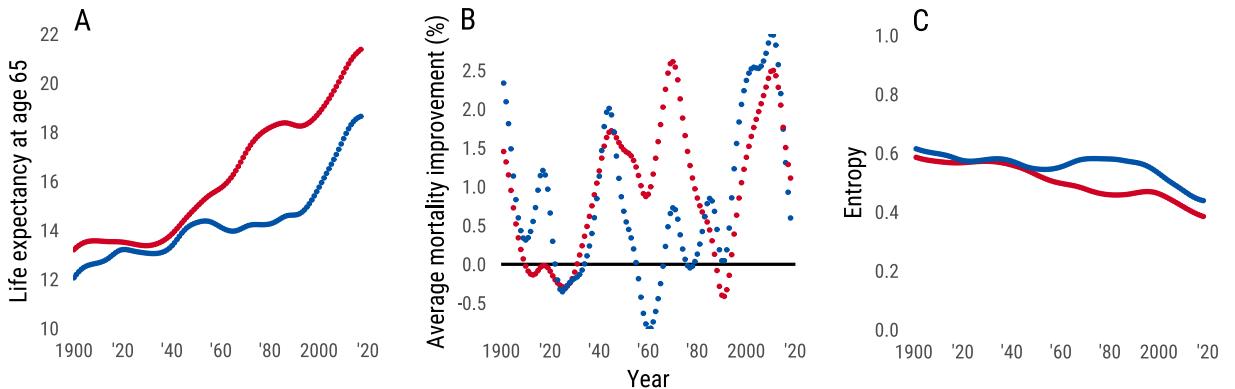


Figure 1.2: Changes over time in life expectancy at age 65, mortality improvements and entropy. Denmark, both sexes, 1900-2019. Red(blue) dots and lines indicate values for females(males). Source: Own elaboration based on the data obtained from the Human Mortality Database (2021).

Panel A of Figure 1.2 shows trends over time in Danish life expectancy calculated at age 65 for both sexes. We can observe that for both sexes, life expectancy has been going up. Panel B and C depict the components that drive changes in $e(x,t)$ as described in Equation 1.3. Panel B shows a general improvement in mortality after age 65. Panel C shows that the entropy has decreased over time, however, it is still much higher than the entropy calculated at birth (i.e. currently $H(0)$ is around 0.2 in modern populations, see Aburto et al. (2019, 2020)). Overall, Figure 1.2 illustrates that life expectancy at age 65 has increased as a consequence of the general mortality improvement that has taken place after this age, which is modulated by the entropy.

It is clear from Equation (1.3) and Figure 1.2 that the rise of life expectancy is driven by mortality improvements. Thus, the question arises as to how much more can life expectancy be

⁴The original concept of entropy comes from information theory (Shannon, 1948). In mortality research, this measure is often denoted as *Keyfitz' entropy* because Nathan Keyfitz popularized its use (Keyfitz, 1977). However, the application of the entropy to mortality dates back to an earlier article written in 1955 by the German econometrician Conrad Emanuel Victor Leser (Leser, 1955).

extended? Forecasts that life expectancy might increase beyond 100 years hinge on mortality improvements for centenarians (Vaupel et al., 2021). However, it is unclear how much progress in reducing mortality is being made after age 100. Indeed, mortality improvements after age 100 are not shown in Figure 1.1 because of the high uncertainty in mortality trends above this age. In the following section I describe this issue in detail.

1.2 Unknowns about the tail of longevity

Since the 1950s, the number of centenarians has risen as a result of reductions in the risk of dying at younger ages (Robine and Caselli, 2005). Still, it is less than clear-cut if centenarians are also living longer. Much of our knowledge about this issue comes from studies in Sweden and Denmark (Drefahl et al., 2012; Modig et al., 2017). The sparse available evidence from these countries, indicates little or no progress in the average lifespan in recent decades.

Figure 1.3 shows the probability of dying at age 100 for females in Sweden, Japan and France. This Figure shows that the probability of dying at age 100 has declined in France and Japan since the middle of the 20th century, stagnating recently in Japan but continuing to fall in France. Forecast trends using the method developed by Lee and Carter (1992) indicate that the probability of dying at age 100 will continue to fall in France and Japan. Male mortality at age 100 also appears to be declining but small numbers make estimation of trends problematic. Yet, it is unknown if there are some other populations that have experienced mortality improvements above age 100. There are three main important factors that constrain our knowledge about the tail of longevity: scarcity of reliable data, uncertainty about the trajectory of $\mu(x)$ above age 100 and the limited knowledge about the biological mechanisms behind extreme longevity.

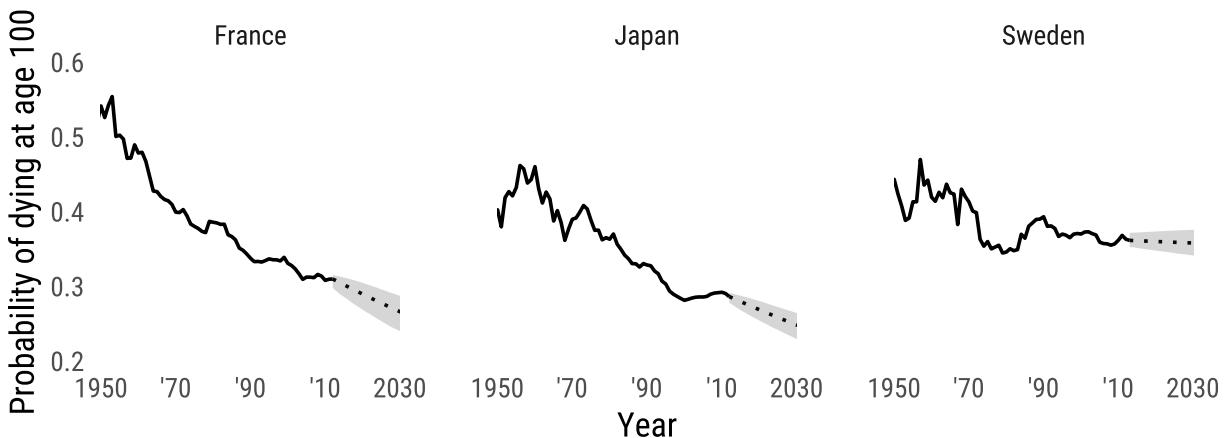


Figure 1.3: Probability of dying at age 100 for France, Japan and Sweden. Females, 1950-2030. *Dotted lines depict forecast values using Lee and Carter (1992) with the extension proposed by Brouhns et al. (2002). Shaded areas indicate 95% confidence intervals. Source: Own elaboration based on the data obtained from the Human Mortality Database (2021).*

1.2.1 Data sources

The assessment of mortality patterns above age 100 is complicated because of data constraints. The Human Mortality Database (2021) provides freely available and open source sex-specific data up to age 110 for 41 populations. This database has been regarded as the gold standard for high quality mortality data. Still, data above age 100 from this database is heavily modelled. Life tables are smoothed using a logistic curve. Therefore, estimations of mortality trends above age 100 from this database are entirely driven by the modelling algorithms to correct erratic data. Furthermore, data available in the Human Mortality Database (2021) have been grouped in whole ages (i.e. ages 100, 101, 102, ...) with an open interval of 110+. These aggregations reduce the level of detail in age patterns of mortality. The Human Mortality Database (2021) also provides raw data. However, the raw data reveals a large number of missing values which are a consequence of the scarcity of data at the oldest ages. Estimating mortality trends from such aggregated raw data implies high uncertainty and it is difficult to distinguish between noise and the signal without any statistical modelling (i.e. fitting curves, smoothing, etc).

Data sources that provide individual data on centenarians and supercentenarians (i.e. individuals aged 110 or older) are more suitable for the assessment of mortality patterns of the oldest old. For example, population registers comprise information on the date of birth, date of death or date of last observation for every individual in the population. Individual data from population registers are useful to study the tail of longevity. However, only a few countries have population registers (e.g. the Nordic countries, the Netherlands and Italy), thereby restricting the analysis to only certain populations.

Another important data source is the International Database on Longevity (IDL). In their first release (in 2010), this database comprised information on dates of birth and death of around 700 supercentenarians from a number of countries (Cournil et al., 2010). In 2020, this database suffered a major update. The current version provides data on individuals aged 105 years or more (i.e. semi-supercentenarians). This addition of information increased the sample size to more than 13,000 records (Maier et al., 2021). A very important feature of this database is that dates at birth and death have been verified by a number of experts (i.e. demographers, gerontologists, statistical offices).

1.2.2 Mortality plateau at extreme old ages

The age-pattern of the risk of dying at the oldest ages is strongly debated. On one hand, some researchers argue that the risk of dying continues to increase exponentially and follows a Gompertz curve (Gavrilov and Gavrilova, 2019a,b). On the other, there is increasing empirical evidence showing that the age trajectory of the risk of dying decelerates around ages 70-90⁵ (Bebbington et al., 2011; Feehan, 2018; Horiuchi and Wilmoth, 1998; Steinsaltz and Wachter, 2006; Wrigley-Field, 2014) and approaches a constant level (i.e. mortality plateau) at the extreme end.

⁵The British actuary Wilfred Perks was among the first to propose a parametric description of $\mu(x)$ that accounts for mortality deceleration (Perks, 1932). Later, Beard (1959) proposed a logistic curve that accounts for heterogeneity.

Evolutionary theories of ageing and theoretical models support the existence of a mortality plateau at the extreme end of life (Demetrius, 2001; Horvitz and Tuljapurkar, 2008; Pletcher and Curtsinger, 1998; Rose et al., 2002; Weitz and Fraser, 2001). Indeed, mortality plateaus have also been found in other species such as *Saccharomyces cerevisiae*, *Caenorhabditis elegans* and *Drosophila*, commonly known as fruitflies (Carey et al., 1998; Charlesworth and Partridge, 1997; Vaupel et al., 1998; Weitz and Fraser, 2001). Mathematical models suggest that if the plateau exists, the risk of dying might converge asymptotically to it (Finkelstein and Esaulova, 2006; Missov and Vaupel, 2015; Steinsaltz and Wachter, 2006). This means that $\mu(x)$ might never be completely flat, but very small increases in this indicator might still be perceptible.

As described in Chapter 2 of this thesis, recent empirical studies provide evidence in favour of the existence of a mortality plateau. Key among them are the studies by Gampe (2010, 2021) and Rootzén and Zholud (2017). By using high-quality data from the IDL (Cournil et al., 2010), they show that the risk of dying levels off after age 110 at values between 0.7 and 0.8. However, these findings have been questioned based on the scarcity of data (less than 1,000 individual records included in the analysis) (Gavrilov and Gavrilova, 2019a,b). Using a larger population size of around 4,000 individual records of semi-supercentenarians, Barbi et al. (2018) showed that the risk of dying also levels off at age 105 for Italy. This finding has been questioned by some researchers that argue that the statistical model used to assess the trajectory of $\mu(x)$ is not appropriate (Newman, 2018).

The assessment of the age pattern of mortality at extreme old ages is important because further longevity extensions depend on it. For example, if the mortality plateau exists and it is reached at level μ^P and at age c such that $x \leq c \leq y$. Then, $\mu(y, t) = \mu^P$ and $\rho(y, t) = 0, \forall y \geq c$. By Equation (1.3), μ^P also implies that $\dot{\epsilon}(y, t) = 0, \forall y \geq c$. Thus, in the case that the location of age c is unchangeable, mortality cannot be reduced after this age, lifespan extensions become unlikely and life expectancy cannot increase further. However, if c can be shifted to even older ages, further extensions in the tail of longevity can be achieved.

1.2.3 Who survives to the tail of longevity?

“How long can humans live?” is one of the most debated questions in science (Kennedy and Norman, 2005). Equally relevant, but less frequently asked questions are: can everyone extend their lifespan to advanced ages? Or is it that just a selected group of individuals make it to the tail of longevity? What are the characteristics of those individuals that survive the longest?

Understanding the characteristics of individuals surviving to the oldest ages is fundamental to determining if further longevity extensions can be achieved (Pignolo, 2019). In this regard, Medford et al. (2019) show that Danish centenarians surviving to the 95% percentile of the death distribution show mortality improvements in their individual lifespan and continue living longer against all expectations. Still, it is unclear if these individuals share similar characteristics or if their survival trajectories are random events as previously hypothesized (Hagberg and Samuelsson, 2008).

Recent evidence shows that the sustained survival at high advanced ages is the outcome of a complex interaction between many factors: lifestyles (Di Francesco et al., 2018; Mattson et al.,

2017; Stathakos et al., 2005; Voelcker-Rehage et al., 2011), genetics (Christensen and Murray, 2007; Murabito et al., 2012; Sebastiani et al., 2012), physiological makeup (Calabrese et al., 2015; Conte et al., 2019; Epel and Lithgow, 2014), etc.. Furthermore, there is a growing body of evidence highlighting the association between health outcomes and longevity (Christensen et al., 2009). Most of this evidence comes from studies on nonagenarians (i.e. individuals aged 90-99 years). For example, it has been shown that nonagenarians can be grouped according to their health profiles (Dato et al., 2012; Montesanto et al., 2010). Likewise, it has been shown that cognitive and physical health are good predictors of survival among Danish nonagenarians (Engberg et al., 2008; Nybo et al., 2003; Thinggaard et al., 2016). A recent study puts forward evidence in favour of reductions in mortality among Danish nonagenarians accompanied by improvements in health and functioning (Thinggaard et al., 2020).

In spite of all these findings it is still unclear if the associations between health and survival found in nonagenarians also hold at extreme old ages (i.e. above ages 100 or 105). The reason is that only around 10%-15% of those who reach age 90 survive to age 100 (Human Mortality Database, 2021), and the number of survivors above this age decreases rapidly (as shown in Chapter 2 of this thesis). Therefore, links between health and survival at extreme old ages cannot be directly made based on what takes place at younger ages (i.e. age 90). Determining the existence of common patterns among centenarians is fundamental to advancing knowledge about the mechanisms underlying extensions in the tail of longevity.

1.3 Longevity and pensions

In the previous sections, I discussed trends in mortality improvements and how they are linked to increasing trends in life expectancy. These dynamics have several implications for societies but specifically, they posit challenges for pension systems (Barr, 2006; OECD, 2017).

Ensuring income security to the elderly is the main aim of any pension system (OECD, 2018; Whitehouse et al., 2009). At the individual level, this is achieved through mechanisms of consumption smoothing and insurance (Barr and Diamond, 2006). Insurance is provided through financial instruments (e.g. life annuities) where an exchange of stream of payments is agreed between two parts (Møller and Steffensen, 2007). At the macro level, pension systems have additional objectives such as income redistribution (i.e. across generations, between socio-economic groups) and poverty relief (Ayuso et al., 2021; Sanchez-Romero et al., 2020). Pension systems should also be financially sustainable so they can fulfil their obligations (Alonso-García et al., 2018; Whitehouse et al., 2009). There may be some other additional objectives depending on the type of schemes (e.g. defined benefit, defined contribution with partially defined benefits, PAYG, etc.) that constitute the whole pension system.

Longevity dynamics play a crucial role in the fulfilment of pension objectives. In this sense, pension providers and life insurance companies deal with two sources of longevity risk. Unsystematic (or micro) longevity risk refers to the uncertainty in individual lifespans (Hari et al., 2008). This type of longevity risk is captured by indicators of lifespan variation⁶, such as the standard deviation (Alvarez et al., 2020), etc. Unsystematic risk is diversifiable because it can

⁶This is often called *lifespan inequality*.

be shared among individuals in a *sufficiently* large pool. Yet, the dispersion in the distribution of lifespans has important implications for policy holders, in particular for those exposed to high uncertainty in their age at death (Milevsky, 2020). Another type of risk is the so-called systematic (or macro) longevity risk. This type of risk refers to the the uncertainty in future improvements of mortality triggering average lifespan extensions (i.e. increases in life expectancy). Systematic longevity risk is not diversifiable and cannot be eliminated when the size of the portfolio is increased (Dahl et al., 2008).

To study the interaction between mortality improvements and longevity risk in closer detail, Haberman et al. (2011) extended the concept of the entropy (as shown in Equation (1.1)) to life annuities $\bar{a}(x)$. Following the same idea as Leser (1955) and Keyfitz (1977), the risk of dying $\mu(x)$ is perturbed by a constant factor ξ such that $\mu^*(x) = \mu(x)(1 + \xi)$. Then $\mu^*(x)$ is used to calculate the new value of the life annuity, $\bar{a}^*(x)$. Differentiating $\bar{a}^*(x)$ with respect to ξ in the neighborhood of $\xi = 0$ results in

$$\frac{\partial \bar{a}^*(x)}{\partial \xi} = -\bar{H}_x(\delta)\bar{a}(x), \quad (1.6)$$

where

$$H_x(\delta) = -\frac{\int_0^\infty s(x+t) \ln[s(x+t)] v(t) dt}{\int_0^\infty s(x+t) v(t) dt}, \quad (1.7)$$

such that $H_x(\delta)$ is the entropy of a life annuity and $v(t)$ is the discount factor with force of interest δ ⁷ for a cash-flow payable at maturity t . Haberman et al. (2011) show that at older ages, values of $H_x(\delta)$ are much higher, which indicate a high sensitivity of $\bar{a}(x)$ to changes in $\mu(x)$. This is in line with research showing that longevity risk plays an important role in the overall risk in pensions and life insurance, in particular, within low-interest environments (Hari et al., 2008; Karabey et al., 2014; Rabitti and Borgonovo, 2020). Moreover, an ongoing line of research uses the entropy and the convexity (i.e. second derivative of $\bar{a}^*(x)$ with respect to ξ) to construct hedging strategies against longevity risk (Li and Hardy, 2011; Tsai and Chung, 2013; Tsai and Jiang, 2011; Wang et al., 2010; Wong et al., 2014). These approaches are relevant in the context of current pension and life insurance regulations that aim at the assessment of risk-based solvency requirements for longevity (Dahl, 2004; Jarner and Møller, 2015).

1.3.1 Implications of socio-economic inequalities in lifespans in pension systems

Motivated by increasing trends in life expectancy and with the aim of ensuring financial sustainability in pensions schemes (OECD, 2019b; Whitehouse et al., 2009), many countries have decided to pass reforms to modify the operation of their pension systems. In Denmark, this is

⁷Haberman et al. (2011) assume a constant force of interest δ , but this can be extended to the forward interest rate at maturity, $\delta(t)$, representing a general form to express the term-structure of interest rates. Stochastic models of $\delta(t)$ can also be assumed and incorporated into the model in order to calculate values of the entropy that are consistent with the market (Møller and Steffensen, 2007). I describe these extensions at the end of this chapter.

done by linking retirement age to increases in life expectancy⁸ (Kallestrup-Lamb et al., 2020). While these reforms might reduce the exposure to longevity risk, they neglect the fact that some subgroups of the population experience lower life expectancies and higher lifespan variation than other subgroups. This gradient in longevity prevails across different socio-economic indicators: income (Chetty et al., 2016; Villegas and Haberman, 2014), education (Permanyer et al., 2018; Sasson, 2016), occupational class (van Raalte et al., 2018), etc..

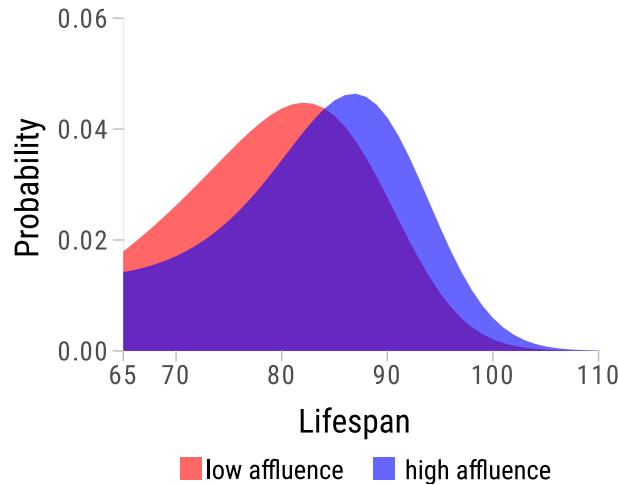


Figure 1.4: Distribution of lifespans after age 65 by affluence group. Denmark, females, 2016.
Source: Own elaboration based on data retrieved from Statistics Denmark. Affluence groups were calculated using the index developed by Cairns et. al (2019)

In Denmark, the longevity gradient has been documented in terms of an affluence index (Cairns et al., 2019). This index ranks individuals in terms of their income and wealth and makes use of all the potential of the highly detailed information available in population registers (Cairns et al., 2019). Figure 1.4 shows the distribution of lifespans after age 65 for Danish females in the highest and lowest affluence groups. There is approximately 86% overlap between both distributions. Still, both distributions are different; one is shifted more to the right and the other one is wider. These differences are translated into different values for life expectancy and lifespan variation, which at the same time, imply different demographic settings at retirement for these affluence groups.

Disparate mortality improvements (i.e. $\rho(x, t)$) are also observed across socio-economic groups. Figure 1.5 shows mortality improvements after age 65 for the highest and lowest affluence groups in Denmark for both sexes during the period 1985 to 2016. It is clear from Figure 1.5 that mortality improvements are not evenly shared across socio-economic groups. For example, mortality deteriorated for females in the highest affluence group during the period 1985 to 1995, whereas the lowest affluence group exhibited mortality improvements during the same period. Thereafter, both groups show similar mortality improvements. Yet, there are clear differences between sexes. As shown in Equation 1.3, mortality improvements and the entropy are the key drivers of changes over time of life expectancy, and therefore also for changes $\bar{a}(x)$ ⁹.

⁸Some pension schemes have addressed this issue by adjusting pay-outs (or guarantees) to future developments of life expectancy at retirement (Jarner and Preisel, 2017).

⁹In the context of low-interest rates, $\delta \rightarrow 0$, $\bar{a}(x) \rightarrow e(x)$, and $H_x(\delta) \rightarrow H(x)$. Currently, in most developed populations interest rates are at their lowest levels, highlighting the relevance of longevity (OECD, 2019a).

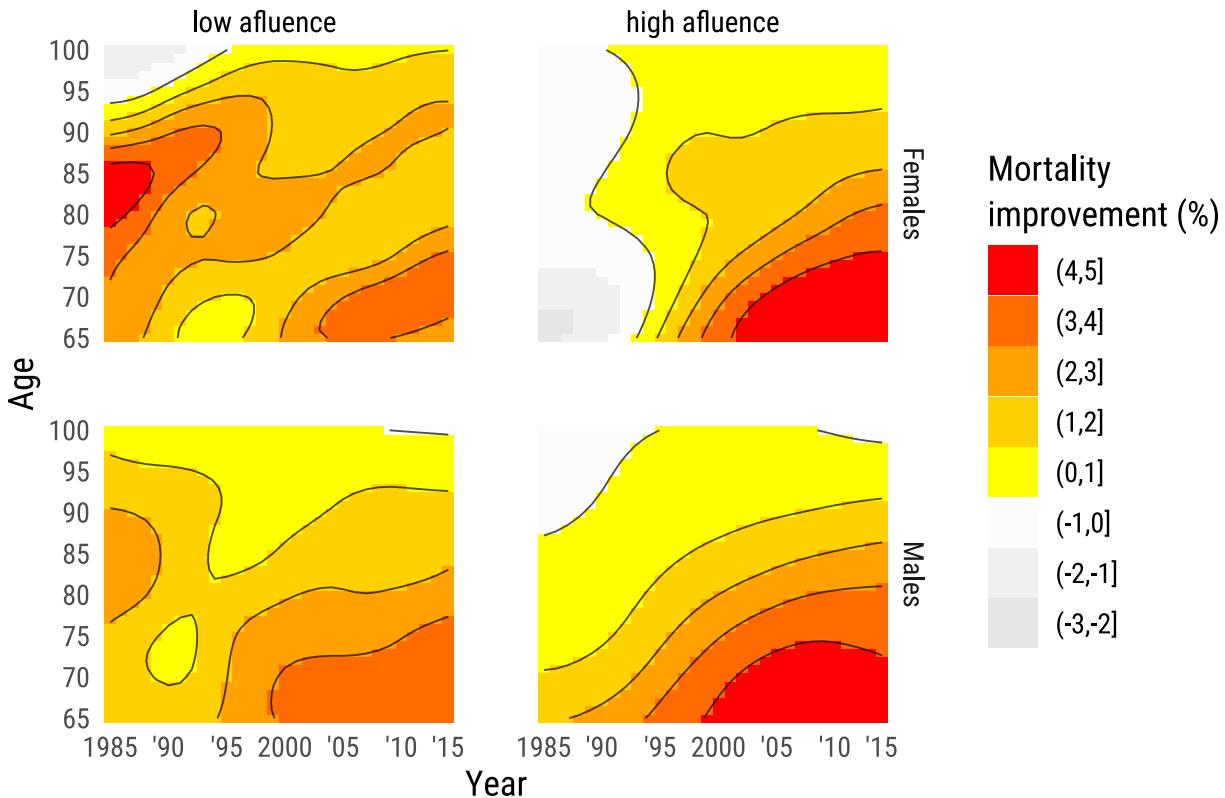


Figure 1.5: Rates of mortality improvement after age 65 by affluence group. Denmark, both sexes, 1950-2019. *Source: Own elaboration based on the data retrieved from Statistics Denmark. Affluence groups were calculated using the index developed by Cairns et. al (2019).*

Socio-economic differentials in longevity also affect macro pension objectives. In particular, these differentials affect the redistribution objective (Brown, 2003; Liebman, 2002) because they might induce transfers of income from low socio-economic groups with shorter lifespans to high socio-economic groups with longer lifespans (Villegas and Haberman, 2014; Whitehouse and Zaidi, 2008). A salient challenge for plan sponsors is to tailor longevity assumptions that reflect the characteristics of pensioners (Madrigal et al., 2011).

1.4 The postponement of mortality

Hitherto I have described the rise of longevity in terms of life expectancy and mortality improvements at different ages. There is a vast amount of evidence in favour of longevity increases (Vaupel et al., 2021). At the same time, there is a long standing disagreement about whether the rise of longevity is accompanied by compression or dispersion of deaths (Bergeron-Boucher et al., 2015; Kannisto, 2001; Myers and Manton, 1984; Nusselder and Mackenbach, 1996; Thatcher et al., 2010; Wilmoth and Horiuchi, 1999). Some scholars argue that deaths become more compressed as longevity increases. Some others argue that deaths will become more dispersed.

Zuo et al. (2018) put an end to the debate over compression/dispersion of deaths. They tackled this issue by examining percentiles in the distribution of deaths after age 65 in 20 developed

countries. Their analysis show that the distance between age 65 and the first quartile (i.e. the 25th percentile after age 65) has increased over time. Nonetheless, all percentiles after the first quartile are moving together toward older ages, while the distance between subsequent percentiles remains almost unchanged. These findings indicate that deaths are not compressing nor becoming more dispersed with ages. Instead, deaths after age 65 have been postponed to older ages at the same pace.

The study carried out by Zuo et al. (2018) is relevant because it puts forward evidence of the steady postponement of mortality (Vaupel, 2010). Still, the resulting dynamics of the risk of dying are unknown. Given that percentiles of death are constantly shifting to older ages, is it possible for the risk of dying to move at the same pace? It is also unknown if Zuo et. al's (2018) findings apply before age 65 where substantial mortality improvements have been reported (Burger et al., 2012).

1.5 Aims

The main objective of this thesis is to provide new insights into increasing patterns of human longevity. Given the background described in the previous section, five aims are pursued, one per manuscript:

1. To examine the age-trajectory of the risk of dying after age 105. In the first manuscript we provide insights into the discussion of how much further lifespans can be extended.
2. To explore patterns among health profiles and survival trajectories of centenarians. This analysis sheds light on the common traits of those individuals that survive the longest and are responsible for extending the tail of longevity toward even higher advanced ages.
3. To determine the implications of linking retirement age to life expectancy in terms of inequalities in longevity and their influence on pension outcomes across socio-economic groups.
4. To describe some of the sources in the sex gap of old-age survival. In particular, we determine the contribution of smoking attributable deaths to sex differences in life expectancy.
5. To examine the postponement of mortality (or increasing longevity) in terms of *survivorship ages*. This is a new framework developed to study mortality in terms of survivorship rather than in terms of chronological ages.

1.6 Data

The research contained in this thesis makes use of individual and aggregated data retrieved from different sources. Here I provide a general summary of these data sources. A more thorough description of the data, periods of observation and data checks can be found in each chapter of this thesis.

Individual data

International Database of Longevity

This database comprises individual data on dates of birth and death of more than 13,000 semi-supercentenarians in a number of countries all around the world. This dataset was used in Chapter 2. More information about the International Database on Longevity can be found in section 1.2.1 and in Maier et al. (2021).

Danish Population Registers

Denmark has a long tradition of collecting high quality and detailed data on their residents. By assigning a unique personal identification number (i.e. *CPR-nummer* in Danish) to every person residing in Denmark, it is possible to link many characteristics of the individual: demographic information (e.g. place of birth, date of birth and death and cause of death, etc..), education level, ethnicity, healthcare use (e.g. diseases, visits to the GP, hospitalizations), etc.. Statistics Denmark is the authority that is in charge of keeping and maintaining the datasets and ensuring that there is no misuse. See Thygesen et al. (2011) for a complete description of the information available in the Danish Population registers.

Individual data from different Danish population registers was used in Chapters 3 and 4. In Chapter 3, we used individual data on dates of birth and death for all Danish centenarians born in 1895, 1905 and 1915. Information on health characteristics for Danish centenarians was retrieved from the Birth Cohort Studies (see below).

For the study in Chapter 4 we used information on the ages at death or at last observation for individuals living in Denmark aged 50+ from 1985 to 2016. Furthermore, we used individual data on gross income and net wealth to construct the affluence index (Cairns et al., 2019) used in the allocation of individuals to a specific socio-economic group. Gross annual income comprises wage income, unemployment benefits, social assistance and pension income. Net wealth was calculated as the difference between total assets and liabilities. Assets include property, bank deposits, stocks, bonds and cash holdings. Liabilities consider all types of debt to private companies and the government. In the case of married couples, 50% of the total wealth was assigned to each spouse. More information about the composition of the affluence index can be found in Cairns et al. (2019)

Danish Birth Cohort Studies

The Danish Birth Cohort Studies were surveys conducted by the Epidemiology Unit of the University of Southern Denmark. The aim of these studies was to assess trends in health and functioning at old age. The assessment was performed on individuals aged 100 born in 1895, 1905, 1910 and 1915. The 1905 and 1915 cohorts followed nonagenarians (i.e. individuals aged 90-99) during various years through different waves of the surveys until they turned age 100. Specifically for the analysis of characteristics of centenarians in Chapter 3, we use information on physical health, cognitive impairment, disability and self rated health of Danish Centenarians born in 1895, 1905 and 1915. See Rasmussen et al. (2017) for more information about the Danish Birth Cohort Studies.

Aggregated data

Human Mortality Database

The general structure of the database was described in Section 1.2.1. The Human Mortality Database was used in the calculation of mortality improvements in this introduction and in the calculation of life expectancies at age 50 in Chapter 5. In Chapter 6, we used raw data for 23 sex-specific populations (46 in total) from 1910 to 2019 to examine the dynamics of mortality as a function of survivorship.

World Health Organization - Database on Causes of Death

Death counts from malignant neoplasms of trachea, bronchus and lung (i.e. lung cancer) were used to estimate smoking attributable mortality in Chapter 5. Such data were classified according to the International Statistical Classification of Diseases, Injuries and Causes of Death versions 7 through 10 (i.e. ICD-7: 162, 163; ICD-8 and ICD-9: 162; ICD-10: C33, C34). See Alvarez et al. (2020) for a more thorough description of the database.

1.7 Contributions

The contributions of each study are summarized as follow:

1. **Regularities in human mortality after age 105.** In this study we analyse a novel dataset on individual records of semi-supercentenarians to determine the trajectory of the risk of dying. We chose to examine mortality trajectories after age 105 in a completely non-parametric framework to avoid assumptions about the functional form of the risk of dying. We show that the levelling off of the risk of dying is a regularity of all the populations analysed here. This empirical finding has important implications for prospects of extensions in longevity and provides insights into the assumptions behind mortality modelling.
2. **Stratification in health and survival after age 100: evidence from Danish centenarians.** In this study we address unobserved heterogeneity in health and survival in centenarians. We show that there is a group of individuals that consistently outperform in health outcomes (i.e. physical and cognitive health) and survive the longest. These *super-select* centenarians are responsible for extensions in the tail of longevity to higher advanced ages. Furthermore, this study suggests that extreme survival is not a completely random event but rather the outcome of sustained health and functioning.
3. **Linking retirement age to life expectancy does not lessen the demographic implications of unequal lifespans.** Pension policies that increase retirement age in pace with increasing life expectancy might alleviate issues related to the sustainability of pension systems. In this study we show that this type of policy has detrimental implications for males in lower socio-economic groups because they spend shorter times in retirement, they experience higher uncertainty about their ages at death and pay higher pension annuity costs per year of expected benefits when annuities are valued with average death rates. This study serves to raise awareness of the impact of longevity inequalities in pension schemes.

4. **Progression of the smoking epidemic in high-income regions and its effects on male-female survival differences: a cohort-by-age analysis of 17 countries.** In this study we show that up to 50% of the sex gap in period life expectancies (between age 50 and 85) is explained by smoking attributable mortality in high income populations. Furthermore, we show that smoking attributable deaths have also contributed to modulate the sex gap across cohorts in high income populations. At present, smoking-attributable mortality remains high and a public health concern in high income countries.
5. **Mortality as a function of survival.** In this manuscript we take a different perspective to studying mortality and longevity. Instead of analysing mortality trajectories over *chronological ages*, we analyse them in terms of *survivorship ages* or *s-ages*. We develop a framework to study the dynamics of $\mu(s,t)$, where s corresponds to the proportion of individuals alive in the cohort. In this article we show that, over time, what has changed is the relationship between mortality and the age variable, but the relationship between mortality and survival has remained steady.

1.8 Discussion

The study of longevity is highly interdisciplinary; it can be tackled from different perspectives. This is embraced by this thesis as it incorporates perspectives and methods covering a wide variety of disciplines such as demography, public health, actuarial mathematics, epidemiology, gerontology and biology. The interdisciplinary approach prevailing in this thesis is the outcome of the continuous exposure to different ideas and ways of thinking during my appointment as PhD Research Fellow at the Interdisciplinary Centre on Population Dynamics. In the following paragraphs I briefly discuss how this thesis makes strides in advancing our knowledge of human longevity in different research strands, and the challenges ahead.

Regularities at the tail of longevity

Lifespans and health outcomes after age 100 are highly heterogeneous. Still, this thesis provides insights into the regularities of the most longevous lives. On one hand we show that the risk of dying levels off at age 105 (Chapter 2). On the other, we show that there is a *super-select* group of centenarians that are in better health and survive longer than other centenarians in their cohorts (Chapter 3). The super-select centenarians are capable to maintain relatively good health and are relatively functional even at high advanced ages. Therefore, centenarians reaching the mortality plateau are likely to be in relatively good health and functioning. In other words, it might be that mortality plateaus in humans are generated by the sustained health and functioning of the super-select centenarians that push the tail of longevity to even high advanced ages. These findings shed light on the biological mechanisms of extreme longevity (Demetrius, 2001; Pletcher and Curtsinger, 1998; Steinsaltz and Wachter, 2006; Wachter et al., 2013). Still, further research is necessary to determine if similar findings arise in different populations than the ones analysed here.

The findings of Chapters 2 and 3 also represent empirical evidence that might serve to refine old-age mortality models for $\mu(x,t)$. For example, a family of parametric models consistent with these findings are the ones that control for unobserved heterogeneity (i.e. Gamma-Gompertz,

Gamma-Gompertz-Makeham, etc.). These models are biologically meaningful and are flexible enough to capture the levelling off in the risk of dying (Missov and Vaupel, 2015). Along the same lines, findings from Chapters 2 and 3 can serve to calibrate forecasting models that account for biologically plausible scenarios in mortality improvements (Cairns et al., 2006; Jarner and Kryger, 2011; Lee and Carter, 1992). Actuaries and demographers might benefit from the incorporation of these empirical regularities into their models.

Unequal increases in longevity

As shown in the first part of this introduction as well as in Chapters 4 and 5, longevity has increased, but not for everyone at the same pace. Females live longer than males and the sex gap in life expectancy between age 50 and 80 has been largely shaped by smoking attributable mortality. These deaths are rooted in the lifestyle behaviour that has caused the most deaths in the last century. The sex gap in longevity is magnified when analysing trends across different socio-economic groups. In Chapter 4, we show that in Denmark, there is a difference of about eight years in life expectancies at retirement between the highest socio-economic group of females and the lowest socio-economic group for males. Furthermore, males from lower socio-economic groups are about 20% less likely to survive from age 50 to retirement ages and they live shorter lives once retired in comparison to the population average.

In Chapter 4, we show that inequalities in longevity (between sexes and across socio-economic groups) have important implications for pension objectives. Policies aiming at controlling for the effect of increasing life expectancy have (so far) neglected the effect of inequalities. To alleviate this issue some researchers have proposed differentiating retirement ages by socio-economic groups (Ayuso et al., 2017; Sanchez-Romero et al., 2020). However, this type of policy might be politically difficult to implement and might trigger other issues. Instead, the interplay between early and late retirement options that take into account working careers might be a plausible solution to diminish the effect of socio-economic inequalities in longevity. All in all, the findings shown in this dissertation contribute to the discussion about how the inequality in the length of life can be integrated into pension policy.

From the perspective of the pension provider, findings from Chapter 4 highlight the need for the valuation of pension products that reflect the characteristics of the annuitants. Models that take into account socio-economic differences in longevity are already applied to the market (Lu et al., 2014). Such approaches might prove useful for a better hedging of longevity risk.

A new perspective on longevity

As shown in Chapters 4 and 5, mortality trajectories over chronological ages x differ (i.e. by age, over time, across populations, between socio-economic groups). In Chapter 6, we build on Zuo et. al's (2018) findings to advance a new framework for studying mortality as a function of survivorship. The main contribution of Chapter 6 is in showing that populations experience similar mortality trajectories at specific levels of survival. This perspective allows us to dig into the core mechanisms of mortality dynamics and the rise of longevity. Specifically, our findings from Chapter 6 shed some light on Vaupel's (2010) hypothesis that all older humans share the same (or at least very similar) rate of ageing. Nonetheless, we make the distinction between the rate of ageing in terms of chronological ages, $b(x,t)$, and our rate $b(s,t)$. The rate of ageing $b(x,t)$ measures the degree of increase in $\mu(x,t)$ as age x increases. Our rate $b(s,t)$ captures the rate of change in $\mu(s,t)$ as survival s goes down from 1 to 0.

It is important to highlight that in Chapter 6 we chose to study mortality in terms of survival s , such that $x(s) = S^{-1}(x)$ because the interpretation of our results is appealing for demographers, actuaries and researchers interested in mortality (i.e. as the population dies out, mortality changes). However, the analysis shown in Chapter 6 could have been performed in terms of the cumulative density function, $F(x)$, and the results would have been the same because both functions are complementary; i.e. $F(x) = 1 - S(x)$. Indeed, the use of survivorship-ages is similar to the analysis of percentiles used in reliability theory and engineering to analyse the failure of a system (Gilchrist, 2000; Nair et al., 2013). In demography and actuarial mathematics, this framework opens the door to a new line of research where changes in mortality are conceived as function of the actual source of change: death and survival of the population members.

1.8.1 Future work

I foresee two main lines of the research in the light of the results of this thesis.

Interaction between longevity and financial risks in life contingent products

As pointed out by Dahl et al. (2008), one of the main challenges for pensions and life insurance is to assess (and control for) the combined financial and longevity/mortality risks inherent in life contingent contracts. Following this idea, and building upon the research presented in Chapter 4, together with my colleague Andrés Villegas from the University of New South Wales, we are in the process of developing a model to decompose the simultaneous contribution of financial and longevity risks to changes over time in life annuities. Specifically, this work is inspired by Equation (1.3) developed by Vaupel and Canudas-Romo (2003) and by the use of the entropy of a life annuity as a measure of risk (Haberman et al., 2011). Andrés and I have been working on this topic and we have shown that:

$$\tilde{a}_x(t) = \frac{\partial \bar{a}_x(t)}{\partial t} = \underbrace{\bar{\rho}_x(t) H_x(t)}_{\text{longevity component}} + \underbrace{\bar{\varphi}(t) D_x(t)}_{\text{financial component}}, \quad (1.8)$$

where $\bar{\rho}_x(t)$ and $\bar{\varphi}(t)$ are the weighted average paces of change in mortality and in the term structure of interest rates respectively. $H_x(t)$ is the entropy (Haberman et al., 2011), and $D_x(t)$ is the duration, commonly used in interest-rate immunization (Ho, 1992; Milevsky, 2013). Thus, Equation (1.8) implies that changes over time in $\bar{a}_x(t)$ are driven by $\bar{\rho}_x(t)$ and $\bar{\varphi}(t)$, which are modulated by the sensitivities to mortality and interest rates ($H_x(t)$ and $D_x(t)$ respectively). Equation (1.8) can be used to determine the sources of the overall change in life annuity factors and to construct better hedging strategies. Functions $\bar{\rho}_x(t)$ and $\varphi(t)$ can also be modelled as stochastic processes and can be used for the valuation of market-consistent life annuity products (Møller and Steffensen, 2007).

Furthermore, Equation (1.8) can be decomposed by age-specific and term-specific components with respective entropies and durations, allowing for hedging strategies that consider specific parts of the survival and interest curves. Another possible extension is the application of Equation (1.8) to stochastic multistate modelling (Hoem, 1969) in order to incorporate policyholder

behaviour (Buchardt and Møller, 2015). Under this framework, transition rates to different states, $\mu(x,t|J)$ and associated entropies can be calculated depending on the state J of the policyholder.

I foresee that the synergy between modern actuarial mathematics and some of the results shown in this thesis will enrich the toolkit to better assess risks inherent to life contingent products. At the same time, I must stress that these research ideas are still under development. Their realization will come one way or another.

Frailty models in terms of survivorship-ages

Another line of research that arises from the results of this PhD thesis is the application development of well-known mortality laws in terms of survivorship-ages. For example, in Chapter 6 we defined the Gompertz law of mortality in terms of s-ages, $x(s)$. Together with Prof. Trifon Missov and my PhD supervisor James Vaupel, we are working in the synergy between survivorship-ages and the theory of heterogenous populations (Vaupel et al., 1979; Vaupel and Missov, 2014). The main assumption of models that account for unobserved heterogeneity is that the population hazard $\bar{\mu}(x,t)$ can be expressed as:

$$\bar{\mu}(x,t) = \bar{z}(x,t)\mu(x,t), \quad (1.9)$$

where $\bar{z}(x,t)$ is the average frailty in the population and $\mu(x,t)$ is the risk of dying for the standard individual (Vaupel and Missov, 2014). Thus, a similar relationship to Equation (1.9) should also hold for the population risk of dying in terms of s ; i.e., $\bar{\mu}(s,t)$. The alliance between these two frameworks (i.e. mortality in terms of survivorhip ages and frailty models) might be useful to assess the hypothesis that “*after some advanced age, all individuals share the same rate of ageing*” (Vaupel, 2010). Future research endeavours will provide support for or against this hypothesis.

Bibliography

- Aburto, J. M., Alvarez, J.-A., Villavicencio, F., and Vaupel, J. W. (2019). The threshold age of the lifetable entropy. *Demographic Research*, 41:83–102.
- Aburto, J. M., Villavicencio, F., Basellini, U., Kjærgaard, S., and Vaupel, J. W. (2020). Dynamics of life expectancy and life span equality. *Proceedings of the National Academy of Sciences*, 117(10):5250–5259.
- Alonso-García, J., Boado-Penas, M. d. C., and Devolder, P. (2018). Adequacy, fairness and sustainability of pay-as-you-go-pension-systems: defined benefit versus defined contribution. *The European Journal of Finance*, 24(13):1100–1122.
- Alvarez, J.-A., Aburto, J. M., and Canudas-Romo, V. (2020). Latin american convergence and divergence towards the mortality profiles of developed countries. *Population Studies*, 74(1):75–92.
- Ayuso, M., Bravo, J. M., and Holzmann, R. (2017). Addressing longevity heterogeneity in pension scheme design. *Journal of Finance and Economics*, 6(1):1–21.
- Ayuso, M., Bravo, J. M., and Holzmann, R. (2021). Getting life expectancy estimates right for pension policy: period versus cohort approach. *Journal of Pension Economics & Finance*, 20(2):212–231.
- Barbi, E., Lagona, F., Marsili, M., Vaupel, J. W., and Wachter, K. W. (2018). The plateau of human mortality: Demography of longevity pioneers. *Science*, 360(6396):1459–1461.
- Barr, N. (2006). Pensions: Overview of the issues. *Oxford Review of Economic Policy*, 22(1):1–14.
- Barr, N. and Diamond, P. (2006). The economics of pensions. *Oxford Review of Economic Policy*, 22(1):15–39.
- Beard, R. E. (1959). Note on some mathematical mortality models. In *Ciba Foundation Symposium-The Lifespan of Animals (Colloquia on Ageing)*, volume 5, pages 302–311. Wiley Online Library.
- Bebbington, M., Lai, C.-D., and Zikitis, R. (2011). Modelling deceleration in senescent mortality. *Mathematical Population Studies*, 18(1):18–37.
- Bergeron-Boucher, M.-P., Ebeling, M., and Canudas-Romo, V. (2015). Decomposing changes in life expectancy: Compression versus shifting mortality. *Demographic Research*, 33:391–424.

- Brouhns, N., Denuit, M., and Vermunt, J. K. (2002). A Poisson log-bilinear regression approach to the construction of projected lifetables. *Insurance: Mathematics and Economics*, 31(3):373–393.
- Brown, J. R. (2003). Redistribution and insurance: Mandatory annuitization with mortality heterogeneity. *Journal of Risk and Insurance*, 70(1):17–41.
- Buchardt, K. and Møller, T. (2015). Life insurance cash flows with policyholder behavior. *Risks*, 3(3):290–317.
- Burger, O., Baudisch, A., and Vaupel, J. W. (2012). Human mortality improvement in evolutionary context. *Proceedings of the National Academy of Sciences*, 109(44):18210–18214.
- Cairns, A. J., Blake, D., and Dowd, K. (2006). A two-factor model for stochastic mortality with parameter uncertainty: theory and calibration. *Journal of Risk and Insurance*, 73(4):687–718.
- Cairns, A. J., Kallestrup-Lamb, M., Rosenskjold, C., Blake, D., and Dowd, K. (2019). Modelling socio-economic differences in mortality using a new affluence index. *ASTIN Bulletin: The Journal of the IAA*, pages 1–36.
- Calabrese, E. J., Dhawan, G., Kapoor, R., Iavicoli, I., and Calabrese, V. (2015). What is hormesis and its relevance to healthy aging and longevity? *Biogerontology*, 16(6):693–707.
- Carey, J. R., Liedo, P., Müller, H.-G., Wang, J.-L., and Vaupel, J. W. (1998). Dual modes of aging in mediterranean fruit fly females. *Science*, 281(5379):996–998.
- Charlesworth, B. and Partridge, L. (1997). Ageing: levelling of the grim reaper. *Current Biology*, 7(7):R440–R442.
- Chetty, R., Stepner, M., Abraham, S., Lin, S., Scuderi, B., Turner, N., Bergeron, A., and Cutler, D. (2016). The association between income and life expectancy in the United States, 2001–2014. *JAMA*, 315(16):1750–1766.
- Christensen, K., Doblhammer, G., Rau, R., and Vaupel, J. W. (2009). Ageing populations: the challenges ahead. *The Lancet*, 374(9696):1196–1208.
- Christensen, K. and Murray, J. C. (2007). What genome-wide association studies can do for medicine. *New England Journal of Medicine*, 356(11):1094–1097.
- Colchero, F., Rau, R., Jones, O. R., Barthold, J. A., Conde, D. A., Lenart, A., Nemeth, L., Scheuerlein, A., Schoeley, J., Torres, C., Zarulli, V., Altmann, J., Brockman, D. K., Bronikowski, A. M., Fedigan, L. M., Pusey, A. E., Stoinski, T. S., Strier, K. B., Baudisch, A., Alberts, S. C., and Vaupel, J. W. (2016). The emergence of longevous populations. *Proceedings of the National Academy of Sciences*, 113(48):E7681—E7690.
- Conte, M., Ostan, R., Fabbri, C., Santoro, A., Guidarelli, G., Vitale, G., Mari, D., Sevini, F., Capri, M., Sandri, M., et al. (2019). Human aging and longevity are characterized by high levels of mitokines. *The Journals of Gerontology: Series A*, 74(5):600–607.
- Cournil, A., Robine, J.-M., Maier, H., Gampe, J., and Vaupel, J. W. (2010). The international database on longevity: structure and contents. In *Supercentenarians*, pages 31–40. Springer.

- Dahl, M. (2004). Stochastic mortality in life insurance: market reserves and mortality-linked insurance contracts. *Insurance: Mathematics and Economics*, 35(1):113–136.
- Dahl, M., Melchior, M., and Møller, T. (2008). On systematic mortality risk and risk-minimization with survivor swaps. *Scandinavian Actuarial Journal*, 2008(2-3):114–146.
- Dato, S., Montesanto, A., Lagani, V., Jeune, B., Christensen, K., and Passarino, G. (2012). Frailty phenotypes in the elderly based on cluster analysis: a longitudinal study of two Danish cohorts. evidence for a genetic influence on frailty. *Age*, 34(3):571–582.
- Demetrius, L. (2001). Mortality plateaus and directionality theory. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 268(1480):2029–2037.
- Di Francesco, A., Di Germanio, C., Bernier, M., and de Cabo, R. (2018). A time to fast. *Science*, 362(6416):770–775.
- Dickson, D. C., Hardy, M., Hardy, M. R., and Waters, H. R. (2013). *Actuarial Mathematics for Life Contingent Risks*. Cambridge University Press.
- Drefahl, S., Lundström, H., Modig, K., and Ahlbom, A. (2012). The era of centenarians: mortality of the oldest old in Sweden. *Journal of Internal Medicine*, 272(1):100–102.
- Engberg, H., Christensen, K., Andersen-Ranberg, K., Vaupel, J. W., and Jeune, B. (2008). Improving activities of daily living in Danish centenarians—but only in women: a comparative study of two birth cohorts born in 1895 and 1905. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 63(11):1186–1192.
- Epel, E. S. and Lithgow, G. J. (2014). Stress biology and aging mechanisms: toward understanding the deep connection between adaptation to stress and longevity. *Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences*, 69(1):S10–S16.
- Feehan, D. M. (2018). Separating the signal from the noise: evidence for deceleration in old-age death rates. *Demography*, 55(6):2025–2044.
- Finkelstein, M. and Esaulova, V. (2006). Asymptotic behavior of a general class of mixture failure rates. *Advances in Applied Probability*, 38(1):244–262.
- Gampe, J. (2010). Human mortality beyond age 110. In *Supercentenarians*, pages 219–230.
- Gampe, J. (2021). Mortality of supercentenarians: Estimates from the updated idl. In *Exceptional Lifespans*, pages 29–35. Springer, Cham.
- Gavrilov, L. A. and Gavrilova, N. S. (2019a). Late-life mortality is underestimated because of data errors. *PLoS biology*, 17(2):e3000148.
- Gavrilov, L. A. and Gavrilova, N. S. (2019b). New trend in old-age mortality: Gompertzization of mortality trajectory. *Gerontology*, 65(5):451–457.
- Gilchrist, W. (2000). *Statistical modelling with quantile functions*. CRC Press.
- Goldman, N., Lord, G., and May, N. (1986). A New Look at Entropy and the Life Table. *Demography*, 23(2):275–282.

- Haberman, S., Khalaf-Allah, M., and Verrall, R. (2011). Entropy, longevity and the cost of annuities. *Insurance: Mathematics and Economics*, 48(2):197–204.
- Hagberg, B. and Samuelsson, G. (2008). Survival after 100 years of age: a multivariate model of exceptional survival in swedish centenarians. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 63(11):1219–1226.
- Hari, N., De Waegenaere, A., Melenberg, B., and Nijman, T. E. (2008). Longevity risk in portfolios of pension annuities. *Insurance: Mathematics and Economics*, 42(2):505–519.
- Ho, T. S. (1992). Key Rate Durations. *The Journal of Fixed Income*, 2(2):29–44.
- Hoem, J. M. (1969). Markov chain models in life insurance. *Blätter der DGVFM*, 9(2):91–107.
- Horiuchi, S. and Wilmoth, J. R. (1998). Deceleration in the age pattern of mortality at older ages. *Demography*, 35(4):391–412.
- Horvitz, C. C. and Tuljapurkar, S. (2008). Stage dynamics, period survival, and mortality plateaus. *The American Naturalist*, 172(2):203–215.
- Human Mortality Database (2021). University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). www.mortality.org.
- Janssen, F. (2020). Similarities and differences between sexes and countries in the mortality imprint of the smoking epidemic in 34 low-mortality countries, 1950–2014. *Nicotine and Tobacco Research*, 22(7):1210–1220.
- Jarner, S. F. and Kryger, E. M. (2011). Modelling adult mortality in small populations: The saint model. *ASTIN Bulletin: The Journal of the IAA*, 41(2):377–418.
- Jarner, S. F. and Møller, T. (2015). A partial internal model for longevity risk. *Scandinavian Actuarial Journal*, 2015(4):352–382.
- Jarner, S. F. and Preisel, M. (2017). Long guarantees with short duration: the rolling annuity. *Scandinavian Actuarial Journal*, 2017(6):471–494.
- Kallestrup-Lamb, M., Kjærgaard, S., and Rosenskjold, C. P. (2020). Insight into stagnating life expectancy: Analysing cause of death patterns across socio-economic groups. *Health Economics*.
- Kannisto, V. (2001). Mode and dispersion of the length of life. *Population: An English Selection*, 13(1):159–171.
- Karabey, U., Kleinow, T., and Cairns, A. J. (2014). Factor risk quantification in annuity models. *Insurance: Mathematics and Economics*, 58:34–45.
- Kennedy, D. and Norman, C. (2005). What don't we know? *Science*, 309(5731):75.
- Keyfitz, N. (1977). What difference would it make if cancer were eradicated? an examination of the Taeuber paradox. *Demography*, 14(4):411–418.
- Keyfitz, N. and Caswell, H. (2005). *Applied mathematical demography*, volume 47. Springer.

- Lee, R. D. and Carter, L. R. (1992). Modeling and forecasting US mortality. *Journal of the American Statistical Association*, 87(419):659–671.
- Leser, C. (1955). Variations in mortality and life expectation. *Population Studies*, 9(1):67–71.
- Li, J. S.-H. and Hardy, M. R. (2011). Measuring basis risk in longevity hedges. *North American Actuarial Journal*, 15(2):177–200.
- Liebman, J. B. (2002). Redistribution in the current US social security system. In *The distributional aspects of social security and social security reform*, pages 11–48. University of Chicago Press.
- Lindahl-Jacobsen, R., Oeppen, J., Rizzi, S., Möller, S., Zarulli, V., Christensen, K., and Vaupel, J. W. (2016a). Why did Danish womens life expectancy stagnate? the influence of interwar generations smoking behaviour. *European Journal of Epidemiology*, 31(12):1207–1211.
- Lindahl-Jacobsen, R., Rau, R., Jeune, B., Canudas-Romo, V., Lenart, A., Christensen, K., and Vaupel, J. W. (2016b). Rise, stagnation, and rise of Danish womens life expectancy. *Proceedings of the National Academy of Sciences*, 113(15):4015–4020.
- Lu, J., Wong, W., and Bajekal, M. (2014). Mortality improvement by socio-economic circumstances in England (1982 to 2006). *British Actuarial Journal*, pages 1–54.
- Madrigal, A. M., Matthews, F. E., Patel, D., Gaches, A., and Baxter, S. (2011). What longevity predictors should be allowed for when valuing pension scheme liabilities? *British Actuarial Journal*, pages 1–62.
- Maier, H., Jeune, B., and Vaupel, J. W. (2021). *Exceptional lifespans*. Springer Nature.
- Mattson, M. P., Longo, V. D., and Harvie, M. (2017). Impact of intermittent fasting on health and disease processes. *Ageing Research Reviews*, 39:46–58.
- Medford, A. (2017). Best-practice life expectancy: An extreme value approach. *Demographic Research*, 36:989–1014.
- Medford, A., Christensen, K., Skytthe, A., and Vaupel, J. W. (2019). A cohort comparison of lifespan after age 100 in Denmark and Sweden: are only the oldest getting older? *Demography*, 56(2):665–677.
- Milevsky, M. A. (2013). Life annuities: An optimal product for retirement income. *CFA Institute Research Foundation Monograph*.
- Milevsky, M. A. (2020). Swimming with wealthy sharks: longevity, volatility and the value of risk pooling. *Journal of Pension Economics and Finance*, 19(2):217246.
- Missov, T. I. and Vaupel, J. W. (2015). Mortality Implications of Mortality Plateaus. *SIAM Review*, 57(1):61–70.
- Modig, K., Andersson, T., Vaupel, J., Rau, R., and Ahlbom, A. (2017). How long do centenarians survive? Life expectancy and maximum lifespan. *Journal of Internal Medicine*, 282(2):156–163.

- Møller, T. and Steffensen, M. (2007). *Market-valuation methods in life and pension insurance*. Cambridge University Press.
- Montesanto, A., Lagani, V., Martino, C., Dato, S., De Rango, F., Berardelli, M., Corsonello, A., Mazzei, B., Mari, V., Lattanzio, F., et al. (2010). A novel, population-specific approach to define frailty. *Age*, 32(3):385–395.
- Murabito, J. M., Yuan, R., and Lunetta, K. L. (2012). The search for longevity and healthy aging genes: insights from epidemiological studies and samples of long-lived individuals. *Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences*, 67(5):470–479.
- Myers, G. C. and Manton, K. G. (1984). Compression of mortality: myth or reality? *The Gerontologist*, 24(4):346–353.
- Nair, N. U., Sankaran, P., and Balakrishnan, N. (2013). *Quantile-based reliability analysis*. Springer.
- Newman, S. J. (2018). Plane inclinations: A critique of hypothesis and model choice in Barbi et al. *PLOS Biology*, 16(12):e3000048.
- Nusselder, W. J. and Mackenbach, J. P. (1996). Rectangularization of the survival curve in the netherlands, 1950-1992. *The Gerontologist*, 36(6):773–782.
- Nybo, H., Petersen, H. C., Gaist, D., Jeune, B., Andersen, K., McGue, M., Vaupel, J. W., and Christensen, K. (2003). Predictors of mortality in 2,249 nonagenarians: the Danish 1905-cohort survey. *Journal of the American Geriatrics Society*, 51(10):1365–1373.
- OECD (2017). Pensions at a glance 2017. Technical report, OECD Publishing, Paris.
- OECD (2018). OECD Pensions Outlook 2018. Technical report, OECD Publishing, Paris.
- OECD (2019a). Main economic indicators, Volume 2019 Issue 7. Technical report, OECD Publishing, Paris.
- OECD (2019b). OECD Pensions at a Glance 2019. Technical report, OECD Publishing, Paris.
- Oeppen, J. and Vaupel, J. W. (2002). Broken limits to life expectancy. *Science*, 296(5570):1029–1031.
- Perks, W. (1932). On some experiments in the graduation of mortality statistics. *Journal of the Institute of Actuaries (1886-1994)*, 63(1):12–57.
- Permanyer, I. and Scholl, N. (2019). Global trends in lifespan inequality: 1950-2015. *PloS one*, 14(5):e0215742.
- Permanyer, I., Spijker, J., Blanes, A., and Renteria, E. (2018). Longevity and lifespan variation by educational attainment in Spain: 1960–2015. *Demography*, 55(6):2045–2070.
- Pignolo, R. J. (2019). Exceptional human longevity. In *Mayo Clinic Proceedings*, volume 94, pages 110–124. Elsevier.

- Pletcher, S. D. and Curtsinger, J. W. (1998). Mortality plateaus and the evolution of senescence: why are old-age mortality rates so low? *Evolution*, 52(2):454–464.
- Preston, S., Heuveline, P., and Guillot, M. (2000). Demography: measuring and modeling population processes. 2001. *Malden, MA: Blackwell Publishers*.
- Rabitti, G. and Borgonovo, E. (2020). Is mortality or interest rate the most important risk in annuity models? a comparison of sensitivity analysis methods. *Insurance: Mathematics and Economics*, 95:48–58.
- Rasmussen, S. H., Andersen-Ranberg, K., Thinggaard, M., Jeune, B., Skytthe, A., Christiansen, L., Vaupel, J. W., McGue, M., and Christensen, K. (2017). Cohort profile: the 1895, 1905, 1910 and 1915 Danish birth cohort studies—secular trends in the health and functioning of the very old. *International journal of epidemiology*, 46(6):1746–1746j.
- Robine, J.-M. and Caselli, G. (2005). An unprecedented increase in the number of centenarians. *Genus*, pages 57–82.
- Rootzén, H. and Zholud, D. (2017). Human life is unlimited—but short. *Extremes*, 20(4):713–728.
- Rose, M. R., Drapeau, M. D., Yazdi, P. G., Shah, K. H., Moise, D. B., Thakar, R. R., Rauser, C. L., and Mueller, L. D. (2002). Evolution of late-life mortality in drosophila melanogaster. *Evolution*, 56(10):1982–1991.
- Sanchez-Romero, M., Lee, R. D., and Prskawetz, A. (2020). Redistributive effects of different pension systems when longevity varies by socioeconomic status. *The Journal of the Economics of Ageing*, 17:100259.
- Sasson, I. (2016). Trends in life expectancy and lifespan variation by educational attainment: United States, 1990–2010. *Demography*, 53(2):269–293.
- Sebastiani, P., Solovieff, N., DeWan, A. T., Walsh, K. M., Puca, A., Hartley, S. W., Melista, E., Andersen, S., Dworkis, D. A., Wilk, J. B., et al. (2012). Genetic signatures of exceptional longevity in humans. *PloS one*, 7(1):e29848.
- Shannon, C. E. (1948). A mathematical theory of communication. *The Bell System Technical Journal*, 27(3):379–423.
- Stathakos, D., Pratsinis, H., Zachos, I., Vlahaki, I., Gianakopoulou, A., Zianni, D., and Kletsas, D. (2005). Greek centenarians: assessment of functional health status and life-style characteristics. *Experimental gerontology*, 40(6):512–518.
- Steinsaltz, D. R. and Wachter, K. W. (2006). Understanding mortality rate deceleration and heterogeneity. *Mathematical Population Studies*, 13(1):19–37.
- Thatcher, A. R., Cheung, S. L. K., Horiuchi, S., and Robine, J.-M. (2010). The compression of deaths above the mode. *Demographic Research*, 22:505.
- Thinggaard, M., Jeune, B., Osler, M., Vaupel, J. W., McGue, M., and Christensen, K. (2020). Are advances in survival among the oldest old seen across the spectrum of health and functioning? *The Journals of Gerontology: Series A*, 75(12):2354–2360.

- Thinggaard, M., McGuire, M., Jeune, B., Osler, M., Vaupel, J. W., and Christensen, K. (2016). Survival prognosis in very old adults. *Journal of the American Geriatrics Society*, 64(1):81–88.
- Thygesen, L. C., Daasnes, C., Thaulow, I., and Brønnum-Hansen, H. (2011). Introduction to Danish (nationwide) registers on health and social issues: structure, access, legislation, and archiving.
- Tsai, C. C. L. and Chung, S. L. (2013). Actuarial applications of the linear hazard transform in mortality immunization. *Insurance: Mathematics and Economics*, 53(1):48–63.
- Tsai, C. C.-L. and Jiang, L. (2011). Actuarial applications of the linear hazard transform in life contingencies. *Insurance: Mathematics and Economics*, 49(1):70–80.
- van Raalte, A. A., Sasson, I., and Martikainen, P. (2018). The case for monitoring life-span inequality. *Science*, 362(6418):1002–1004.
- Vaupel, J. W. (2010). Biodemography of human ageing. *Nature*, 464(7288):536–42.
- Vaupel, J. W. and Canudas-Romo, V. (2003). Decomposing change in life expectancy: a bouquet of formulas in honor of Nathan Keyfitz's 90th birthday. *Demography*, 40(2):201–16.
- Vaupel, J. W., Carey, J. R., Christensen, K., Johnson, T. E., Yashin, A. I., Holm, N. V., Iachine, I. A., Kannisto, V., Khazaeli, A. A., Liedo, P., et al. (1998). Biodemographic trajectories of longevity. *Science*, 280(5365):855–860.
- Vaupel, J. W., Manton, K. G., and Stallard, E. (1979). The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality. *Demography*, 16(3):439.
- Vaupel, J. W. and Missov, T. I. (2014). Unobserved population heterogeneity: A review of formal relationships. *Demographic Research*, 31(1):659–686.
- Vaupel, J. W., Villavicencio, F., and Bergeron-Boucher, M.-P. (2021). Demographic perspectives on the rise of longevity. *Proceedings of the National Academy of Sciences*, 118(9).
- Vaupel, J. W., Zhang, Z., and van Raalte, A. A. (2011). Life expectancy and disparity: an international comparison of life table data. *BMJ Open*, 1(1):e000128.
- Villegas, A. M. and Haberman, S. (2014). On the modeling and forecasting of socioeconomic mortality differentials: An application to deprivation and mortality in England. *North American Actuarial Journal*, 18(1):168–193.
- Voelcker-Rehage, C., Godde, B., and Staudinger, U. M. (2011). Cardiovascular and coordination training differentially improve cognitive performance and neural processing in older adults. *Frontiers in Human Neuroscience*, 5:26.
- Wachter, K. W., Evans, S. N., and Steinsaltz, D. (2013). The age-specific force of natural selection and biodemographic walls of death. *Proceedings of the National Academy of Sciences*, 110(25):10141–10146.

- Wang, J. L., Huang, H., Yang, S. S., and Tsai, J. T. (2010). An optimal product mix for hedging longevity risk in life insurance companies: The immunization theory approach. *Journal of Risk and Insurance*, 77(2):473–497.
- Weitz, J. S. and Fraser, H. B. (2001). Explaining mortality rate plateaus. *Proceedings of the National Academy of Sciences*, 98(26):15383–15386.
- Whitehouse, E., D’addio, A., Chomik, R., and Reilly, A. (2009). Two decades of pension reform: What has been achieved and what remains to be done? *The Geneva Papers on Risk and Insurance—Issues and Practice*, 34(4):515–535.
- Whitehouse, E. and Zaidi, A. (2008). Socio-economic differences in mortality: Implications for pensions policy. Technical report, OECD Publishing, Paris.
- Wilmoth, J. R. and Horiuchi, S. (1999). Rectangularization revisited: Variability of age at death within human populations. *Demography*, 36(4):475–495.
- Wong, T. W., Chiu, M. C., and Wong, H. Y. (2014). Time-consistent mean–variance hedging of longevity risk: Effect of cointegration. *Insurance: Mathematics and Economics*, 56:56–67.
- Wrigley-Field, E. (2014). Mortality deceleration and mortality selection: Three unexpected implications of a simple model. *Demography*, 51(1):51–71.
- Zuo, W., Jiang, S., Guo, Z., Feldman, M. W., and Tuljapurkar, S. (2018). Advancing front of old-age human survival. *Proceedings of the National Academy of Sciences*, 115(44):11209–11214.

Regularities in human mortality after age 105

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Chapter 2

Regularities in human mortality after age 105

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Abstract

Empirical research on human mortality and extreme longevity suggests that the risk of death among the oldest-old ceases to increase and levels off at age 110. The universality of this finding remains in dispute because of two main reasons: i) high uncertainty around statistical estimates generated from scarce data, and ii) the lack of country-specific comparisons. In this article, we estimate age patterns of mortality above age 105 using data from the International Database on Longevity, an exceptionally large and recently updated database comprising more than 13,000 validated records of long-lived individuals from eight populations. We show that, in all of them, similar mortality trajectories arise, suggesting that the risk of dying levels off after age 105. As more high-quality data become available, there is more evidence in support of a levelling-off of the risk of dying as a regularity of longevous populations.

Keywords: extreme longevity, International Database on Longevity, mortality plateau, oldest-old, semi-supercentenarians.

2.1 Introduction

Several regularities are associated with human mortality. It is broadly accepted, for instance, that the risk of death starts growing exponentially with age at early adulthood (Gompertz, 1825). However, at the frontier of human longevity, we are challenged by the limits of demographic knowledge. The trajectory of the risk of dying at extreme old ages is one of the demographic patterns that is most often questioned in evolutionary theories of aging (Charlesworth and Partridge, 1997; Demetrius, 2001; Mueller and Rose, 1996; Pletcher and Curtsinger, 1998; Wachter et al., 2014) mathematical models (Horvitz and Tuljapurkar, 2008; Missov and Vaupel, 2015;

Weitz and Fraser, 2001) and empirical studies (Gampe, 2010; Gavrilov and Gavrilova, 2019b; Gavrilova and Gavrilov, 2020; Horiuchi, 2003; Rootzén and Zholud, 2017). The identification of regularities in mortality patterns among the oldest-old has profound implications for societies and health sciences (Couzin, 2005), and it can radically reshape evolutionary thinking (Wachter et al., 2014). In this article, we examine an unprecedentedly large and reliable dataset of the longest-lived individuals to evaluate whether a universal pattern of human longevity emerges at extreme old ages, thereby, providing new insights into and a better understanding of the biological mechanisms of human aging.

Previous empirical research on extreme longevity has led to opposing standpoints. Some scholars have suggested that there is a mortality plateau after age 110, estimating a constant risk of death of 0.70 after that age, which corresponds to an annual probability of dying of about 0.5 (Gampe, 2010; Rootzén and Zholud, 2017). Those results, however, have been questioned, based on: i) high uncertainty around the estimates, given the small number of observations (usually not exceeding 1,000 records), and ii) on the aggregation of populations, thereby masking regional and country specificity (Gavrilov and Gavrilova, 2019b; Gavrilova and Gavrilov, 2020). Recently, a sample of 3,836 Italian semi-supercentenarians (individuals aged 105 years or older) has been used to show that the risk of dying reaches or closely approaches a plateau after age 105 (Barbi et al., 2018). This is an important result because it demonstrates a mortality plateau for a single country. Skepticism about the finding still prevails, however; mainly based on the statistical model chosen to evaluate the trajectory of the risk of dying (Beltrán-Sánchez et al., 2018; Newman, 2018; Wachter, 2018).

As people live longer, and more individuals reach exceptionally advanced ages, larger datasets on the oldest-old become available. Key among them is the International Database on Longevity (IDL), which is the result of an unprecedented collaboration between statistical offices from different countries, demographers, gerontologists and experts on longevity (Maier et al., 2019). The database, which was first launched in 2010, provided detailed information on 672 exhaustively validated cases of supercentenarians (individuals aged 110 or older) from a number of countries (Maier et al., 2010). In 2020, the IDL was updated and it now provides additional high-quality data on individuals who survived to age 105, increasing the sample size to more than 13,000 records (Maier et al., 2019).

In this article, we examine data from eight populations included in the IDL that report validated records of all individuals aged 105 and above: France, Germany, Belgium, the United States, Denmark, Quebec, Austria and Norway. Apart from Quebec, all of them are nationally representative. For the United States, we start the analysis at age 110 because available data before that age is not country-representative (Maier et al., 2019). We use a fully non-parametric framework to estimate the age trajectories of the risk of dying (see Materials and Methods for details). Although comparisons are more meaningful among countries with larger populations, the estimation of the risk of dying (i.e. mortality hazard) is still possible in the eight populations studied. Differences in population sizes are reflected in the size of the confidence intervals of the estimated hazards.

Our analyses represent three major contributions. First, the uncertainty around the estimates of the risk of dying is substantially reduced, by virtue of the large amount of high-quality data included in the analysis. Second, the exceptionally large dataset enables us to assess, for the first time, country-specific trajectories in the risk of dying above age 105. Third, our estimates

are purely data driven; no model structure is imposed on the age patterns of mortality. These are three compelling features of our study that were neither achieved in previous studies of supercentenarians (Gavrilov and Gavrilova, 2019a; Gavrilova and Gavrilov, 2020), nor applied to datasets where the scarcity of data was overcome by aggregating populations (Gampe, 2010; Rootzén and Zholud, 2017). According to the hypothesis that there is a mortality plateau at extreme ages (Barbi et al., 2018; Gampe, 2010), a leveling-off in the risk of dying is expected to arise as a regularity in all the populations analyzed.

2.2 Materials and methods

2.2.1 Mortality data

Exact dates of birth and death of all individuals who attained age 105 were obtained from the International Database on Longevity (IDL), which collects information from twelve European countries, Canada (Quebec only), Japan and the United States (see (Maier et al., 2010) for detailed description of the database). The IDL is an open access database and it can be retrieved from <https://www.supercentenarians.org/>. The data used in this study were retrieved on December 1st, 2020.

When the date of death is not available, the IDL provides information on the last date an individual was confirmed to be alive. In the case of the United States, the exact dates are unknown: for each individual, only the years of birth and death together with the age (in days) are given. Several sample frames are observed in the IDL, depending on the country (Gampe, 2010; Maier et al., 2010, 2019). The information is censored if we only know that the individual survived to a certain age (right-censored) or died between two ages (interval-censored, as in the United States). An observation is truncated if the individual was selected into the sample only because they survived to age 105 (left-truncated), or because they died before reaching a particular age (right-truncated).

2.2.2 Estimation of the risk of dying

Let X be a random variable that denotes the length of life after age 105. Given the exact dates on which individuals were last observed (dead or alive), we were able to partition the data set into J age intervals of length δ . By taking into account the observation frames described above, we computed the number of events δE_x and the exposures δN_x (number of subjects exposed to the risk of dying) within each age interval $[x, x + \delta)$ for $x \in I := \{105, 105 + \delta, \dots, 105 + J \cdot \delta\}$. Then we computed $\delta M_x = \frac{\delta E_x}{\delta N_x}$, i.e. the corresponding central death rate within each interval. By taking a small δ , δM_x provides a quasi-continuous estimate of the risk of dying $\mu(x)$.

2.2.3 Estimation of confidence intervals

Because of the small number of observations at the highest ages, standard methods that rely on the central limit theorem to compute confidence intervals are not valid. Hence, we assessed uncertainty surrounding our estimates by computing 95% empirical confidence intervals from data simulation. First, assuming the risk of death is constant within each age interval, for each population we transformed the estimated death rates into probabilities $\delta q_x = 1 - \exp[\sum_{i \in I, i \leq x} \delta \cdot \delta M_x]$. Then, we simulated n individuals who die according to the corresponding δq_x . The values of n match the number of records in the IDL for each country or region. We replicated this exercise 10,000 times, obtaining, for each population, 10,000 sets of death probabilities over age. Finally, we back transformed these sets of probabilities into death rates, and calculated the corresponding confidence intervals.

2.2.4 Random imputation of dates of birth in the United States

To use the same framework for the estimation of a quasi-continuous risk of dying in the United States, we carried out a random imputation of dates of birth. For all individuals, we first established lower and upper bounds of birth depending on the corresponding year of birth, year of death, and exact age at death (in days). For example:

- An individual born in 1900 and dying in 2010 at the exact age 109 years and 200 days could have been born on June 15, 1900 at the earliest, supposing they died on January 1, 2010 at that exact age. The latest date of birth is December 31, 1900, supposing they died on August 19, 2010 with 109 years and 200 days. This individual could not have died later, otherwise they would have turned out 110, since the year of birth is 1900.
- Besides, an individual born in 1900 and dying in 2010 at the exact age 110 years and 20 days could have been born on January 1, 1900 at the earliest, supposing they died on January 21, 2010 at that exact age. The latest date of birth is December 11, 1900 supposing they died on December 31, 2010 with exactly 110 years and 20 days.

Given these individual lower and upper bounds, we randomly imputed an exact date of birth to each US record. We then calculated the corresponding exact date of death given the exact age at death, which is known. We repeated the birthdate imputation $m = 10,000$ times, and in each step, we computed $k = 10,000$ sets of death probabilities over age, as for the other populations. We then aggregated the sets of all the imputation steps, obtaining $m \times k$ sets of death probabilities over age. We finally estimated the risk of dying over age from these $m \times k$ sets and corresponding empirical confidence intervals, as described above. These confidence intervals measure the uncertainty of the point estimates, also assessing the additional uncertainty due to the data imputation.

2.3 Results

Figure 2.1A shows the ages at last observation for all individuals included in the analysis. A rapid decrease in the number of individuals over age is observed: from 6,018 observations between ages 105-106 to 326 individuals above age 110, up to the longest documented lifespan achieved by Jeanne Calment, who died at the age of 122 years and 164 days (Robine et al., 2019). This pattern suggests an exponential decline in the number of survivors, which might thus call for an underlying constant risk of dying (i.e. mortality plateau). However, much heterogeneity is hidden in the main histogram of Figure 2.1A, because the number of observations is not evenly shared by countries and sexes. Females, for example, account for 90% of the data.

The insets of Figure 2.1B provide details on the distribution of observations by country. France accounts for the largest share in the IDL, with 9,853 observations (8,990 females and 863 males) from birth cohorts 1870-1912. Germany and Belgium show 970 and 859 observations, respectively, which represent about one-tenth of the population size in France. The United States also exhibits a large population size (504 individuals above age 110); however, all of its records are only partially observed, given that the exact dates of birth are not reported for confidentiality reasons (Maier et al., 2019). We performed non-parametric imputation of birthdates to produce mortality estimates for the United States (see Materials and Methods). Finally, Denmark, Quebec, Austria and Norway each account for 200-400 observations. Despite the fact that the populations analyzed are all of different sizes, the distributions of individuals by age are similar across countries (Figure 2.1B). This indicates that a rapid decrease in the number of individuals over age, shown in Figure 2.1A, is a regularity of longevous populations.

Figure 2.2 displays the risk of dying from ages 105 to 113 and 95% empirical confidence intervals (CI) for females. The risk of dying is expressed in age intervals of six months. In France, the risk of dying is 0.64 (95% CI 0.62-0.67) at age 105, and slowly increases toward values of about 0.8 at age 110. The large number of documented cases narrows confidence intervals between ages 105 and 110. The low uncertainty around the country-specific estimations of the risk of dying around these ages was never achieved in any previous study of extreme longevity (Barbi et al., 2018; Gampe, 2010; Rootzén and Zholud, 2017).

A similar pattern to that found in France is replicated for the other female populations: the risk of death ranges between 0.6 and 0.7 at age 105 and goes up slightly, approaching values of around 0.8. These regularities are more evident in Germany and Belgium, where the number of observations is considerably large. In the United States, the risk of dying also oscillates between 0.6 and 0.8 after 110. Estimates for Denmark, Quebec, Austria and Norway also fluctuate between 0.6 and 0.8 from ages 105-110. However, confidence intervals in these populations are wider, because the number of observations is much smaller than in the other countries (e.g., France or Germany). For males, the estimation of the risk of dying was only possible in France. As shown in Table S1 in the Supporting information (SI), mortality patterns between 105 and 110 in French males are somewhat similar to the ones found in female populations. Male survivors older than 105 years in the other populations are too few (less than 100) to produce reliable estimations. Similarly, in any population (females and males), the number of individuals aged 113 and above are too few to produce reliable estimates. The lack of observations hinders the assessment of the age pattern of mortality past this age (Villavicencio and Aburto, 2021).

Finally, we performed two sensitivity analysis to test the robustness of our results. First, we

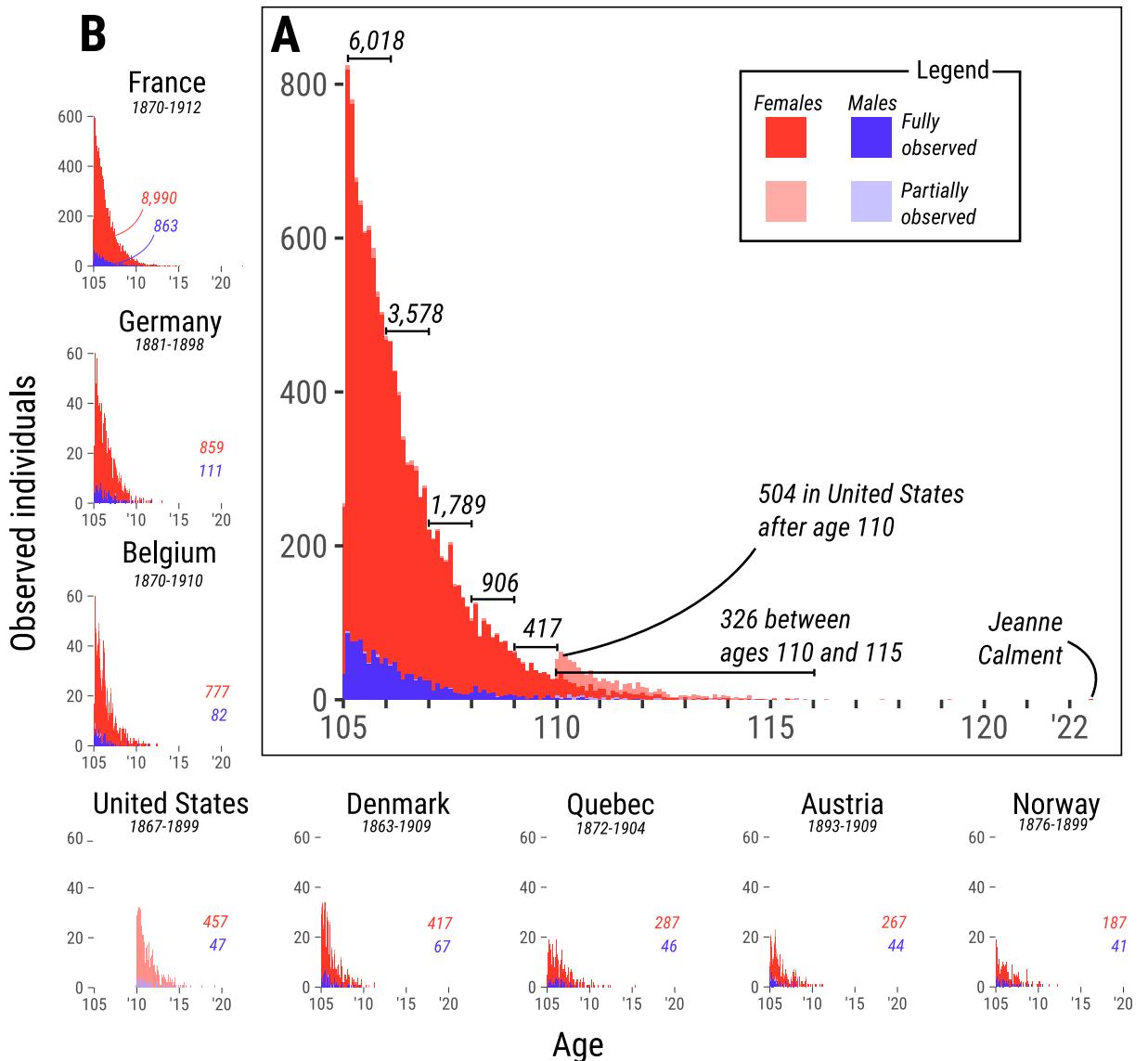


Figure 2.1: Histograms of individuals who reached age 105 reported in the International Database on Longevity, disaggregated by sex and observational scheme. (A) *Histogram depicting the distribution of ages at last observation for all countries combined.* (B) *Close up to the distribution of ages at last observation by country and region. Red bars represent females and blue bars represent males. When the exact age at death is known, we regard an individual as fully observed and it is represented by solidly colored bars. Conversely, partially observed individuals are those whose exact date of death is unknown (e.g., right-censored or interval-censored) and they are depicted with lighter-colored bars. Birth cohorts of individuals included in the analysis are indicated below each country name.*

reduced the age interval from six to three months (see Figure S1 in SI). The trajectories of the risk of dying are very much alike in both cases. This indicates that our results do not depend on the size of the age interval. Second, we calculated the risk of dying for the total population (i.e. adding up data for males and females, see Figure S2 in SI). The trajectory of the risk of dying for the total population is almost identical to the one depicted by females, which account for most of the observed cases. This analysis entails that the results shown in Figure 2.2 clearly

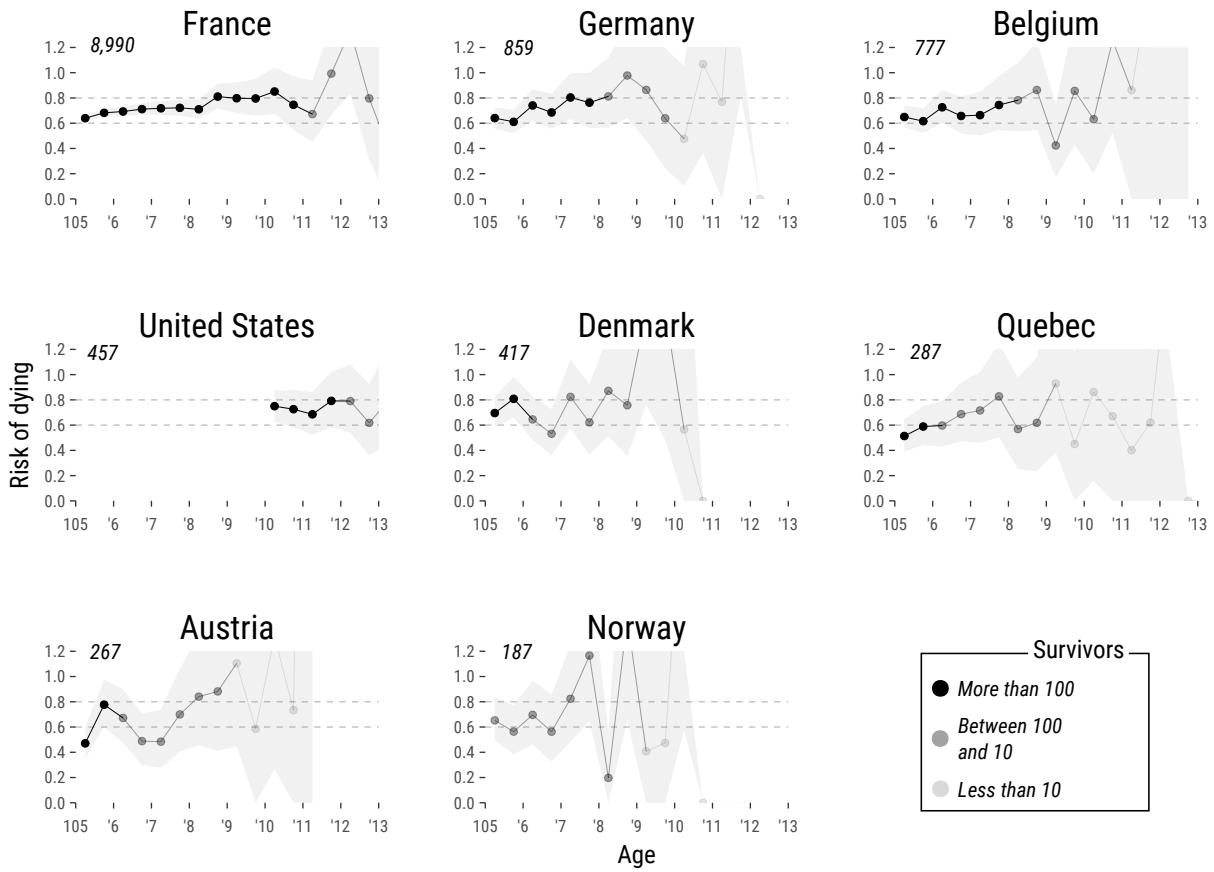


Figure 2.2: Risk of dying between ages 105 and 113 with 95% empirical confidence intervals for females. *The estimation of the risks of dying and empirical confidence intervals was performed using a fully non-parametric approach and by considering the observations schemes in each country. The risk of dying is expressed in age intervals of six months. Population size is indicated at the top-left corner of each panel. The darker the shade of the dot plot, the greater the number of survivors at every specific age (i.e. subjects exposed to the risk of dying). For example, at age 105, there were 8,990 French females exposed to the risk of dying. At age 113, only 18 survived.*

depict the trajectories of the risk of dying above age 105 for all the populations analyzed here. Our results are also coherent with the age-pattern of risk of dying occurring prior age 105 (see Figure S3 in SI).

2.4 Discussion

While it is true that population size and observation schemes play an important role in reducing the uncertainty around mortality estimates (Gampe, 2010), our findings provide evidence of regularities in the age pattern of mortality after age 105: none of the populations analyzed in this study indicate a rapid increase in the mortality hazard after age 105.

Our results are in stark contrast to previous studies that argue that the levelling-off in the risk of

death among the oldest-old is only observed when less accurate data are analyzed, and that more recent and reliable data depict a steady increase in the mortality risk (Gavrilov and Gavrilova, 2019a; Gavrilova and Gavrilov, 2020; Newman, 2018). Indeed, data from recent cohorts (e.g. after 1910) are not included in the analysis for most of the countries because they are not yet available through the IDL (Maier et al., 2010). We foresee that the inclusion of such data would have increased considerably the number of records, thereby, reducing the uncertainty in the estimates of the risk of dying in populations were confidence intervals are large.

A wide range of theoretical models from various mathematical perspectives (e.g., Markov chains, Weiner processes, frailty models, directionality theory and other stochastic matrix models) have been proposed to describe the mechanisms behind mortality plateaus that were previously found in several species (Charlesworth and Partridge, 1997; Demetrius, 2001; Mueller and Rose, 1996; Pletcher and Curtsinger, 1998; Vaupel and Yashin, 1985; Wachter et al., 2014). A common factor among these models is their support for the asymptotic convergence of the risk of dying toward a constant at the extreme end (i.e. mortality plateau). The asymptotic convergence implies that the observed risk of dying might never be completely flat, but it might be that minuscule increases indicate proximity to the asymptotic mortality plateau (Finkelstein and Esaulova, 2006; Horvitz and Tuljapurkar, 2008; Steinsaltz and Wachter, 2006; Vaupel and Yashin, 1985; Weitz and Fraser, 2001). The results of the present study are in line with the asymptotic behavior suggested by theoretical models, which contributes to bridging the demographic knowledge gap and help to enhance our understanding about mortality plateaus in human populations.

Another question that arises from our results is whether the levelling-off is immutable or flexible. This has key implications for human longevity prospects, because forecasts that propose that life expectancy might increase to more than 100 years hinge on reductions in the risk of death for centenarians and semi-supercentenarians (Vaupel et al., 2021). In the light of our findings, we foresee two contrasting scenarios. In the first of these, if the levelling-off shown in our findings is immutable over time, the risk of death cannot be reduced after age 105. Thus, there might be a limit to life expectancy that will be approached as more individuals survive to advanced ages. In the second scenario, if the onset age of the levelling-off is shifted toward ages beyond 105 (e.g. 115), old-age deaths can be postponed toward even older ages and life expectancy can continue to increase. This scenario is plausible as it has been shown that gains in life expectancy in the last two centuries were achieved by shifting deaths from early to late ages (Vaupel et al., 2021; Zuo et al., 2018). However, it remains to be seen whether medical advances and the biological plasticity of aging will continue their interplay, thus continuing to advance the frontier of human longevity.

Bibliography

- Barbi, E., Lagona, F., Marsili, M., Vaupel, J. W., and Wachter, K. W. (2018). The plateau of human mortality: Demography of longevity pioneers. *Science*, 360(6396):1459–1461.
- Beltrán-Sánchez, H., Austad, S., and Finch, C. (2018). Comment on the plateau of human mortality: Demography of longevity pioneers. *Science*, 361(6409).
- Charlesworth, B. and Partridge, L. (1997). Ageing: levelling of the grim reaper. *Current Biology*, 7(7):R440–R442.
- Couzin, J. (2005). How Much Can Human Life Span Be Extended? *Science*, 309(5731):83–83.
- Demetrius, L. (2001). Mortality plateaus and directionality theory. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 268(1480):2029–2037.
- Finkelstein, M. and Esaulova, V. (2006). Asymptotic behavior of a general class of mixture failure rates. *Advances in Applied Probability*, 38(1):244–262.
- Gampe, J. (2010). Human mortality beyond age 110. In *Supercentenarians*, pages 219–230.
- Gavrilov, L. A. and Gavrilova, N. S. (2019a). Late-life mortality is underestimated because of data errors. *PLoS biology*, 17(2):e3000148.
- Gavrilov, L. A. and Gavrilova, N. S. (2019b). New trend in old-age mortality: Gompertzialization of mortality trajectory. *Gerontology*, 65(5):451–457.
- Gavrilova, N. S. and Gavrilov, L. A. (2020). Are we approaching a biological limit to human longevity? *The Journals of Gerontology: Series A*, 75(6):1061–1067.
- Gompertz, B. (1825). Xxiv. on the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies. in a letter to francis baily, esq. frs &c. *Philosophical transactions of the Royal Society of London*, (115):513–583.
- Horiuchi, S. (2003). Interspecies differences in the life span distribution: Humans versus invertebrates. *Population and Development Review*, 29:127–151.
- Horvitz, C. C. and Tuljapurkar, S. (2008). Stage dynamics, period survival, and mortality plateaus. *The American Naturalist*, 172(2):203–215.
- Maier, H., Gampe, J., Jeune, B., Vaupel, J. W., and Robine, J.-M. (2010). *Supercentenarians*. Springer.

- Maier, H., Jeune, B., and Vaupel, J. W. (2019). Exceptional lifespans.
- Missov, T. I. and Vaupel, J. W. (2015). Mortality Implications of Mortality Plateaus. *SIAM Review*, 57(1):61–70.
- Mueller, L. D. and Rose, M. R. (1996). Evolutionary theory predicts late-life mortality plateaus. *Proceedings of the National Academy of Sciences*, 93(26):15249–15253.
- Newman, S. J. (2018). Plane inclinations: A critique of hypothesis and model choice in barbi et al. *PLoS biology*, 16(12):e3000048.
- Pletcher, S. D. and Curtsinger, J. W. (1998). Mortality plateaus and the evolution of senescence: why are old-age mortality rates so low? *Evolution*, 52(2):454–464.
- Robine, J.-M., Allard, M., Herrmann, F. R., and Jeune, B. (2019). The real facts supporting jeanne calment as the oldest ever human. *The Journals of Gerontology: Series A*, 74(Supplement_1):S13–S20.
- Rootzén, H. and Zholud, D. (2017). Human life is unlimited—but short. *Extremes*, 20(4):713–728.
- Steinsaltz, D. R. and Wachter, K. W. (2006). Understanding mortality rate deceleration and heterogeneity. *Mathematical Population Studies*, 13(1):19–37.
- Vaupel, J. W., Villavicencio, F., and Bergeron-Boucher, M.-P. (2021). Demographic perspectives on the rise of longevity. *Proceedings of the National Academy of Sciences*, 118(9).
- Vaupel, J. W. and Yashin, A. I. (1985). Heterogeneity's ruses: some surprising effects of selection on population dynamics. *The American Statistician*, 39(3):176–185.
- Villavicencio, F. and Aburto, J. M. (2021). Does the risk of death continue to rise among supercentenarians? In *Exceptional Lifespans*, pages 37–48. Springer, Cham.
- Wachter, K. W. (2018). Hypothetical errors and plateaus: A response to newman. *PLoS biology*, 16(12):e3000076.
- Wachter, K. W., Steinsaltz, D., and Evans, S. N. (2014). Evolutionary shaping of demographic schedules. *Proceedings of the National Academy of Sciences*, 111(Supplement 3):10846–10853.
- Weitz, J. S. and Fraser, H. B. (2001). Explaining mortality rate plateaus. *Proceedings of the National Academy of Sciences*, 98(26):15383–15386.
- Zuo, W., Jiang, S., Guo, Z., Feldman, M. W., and Tuljapurkar, S. (2018). Advancing front of old-age human survival. *Proceedings of the National Academy of Sciences*, 115(44):11209–11214.

Supporting information for:

Regularities in human mortality after age 105

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1. Additional tables and figures

Table S1. Estimates of the risk of dying and 95% confidence intervals for males in France

Age	France	
	$\mu(x)$	95% CI
105.0	0.76	(0.67,0.85)
105.5	0.75	(0.64,0.86)
106.0	0.80	(0.67,0.95)
106.5	0.87	(0.70,1.05)
107.0	0.76	(0.57,0.98)
107.5	0.47	(0.30,0.67)
108.0	1.35	(0.99,1.80)
108.5	0.86	(0.48,1.34)
109.0	0.96	(0.47,1.62)
109.5	1.53	(0.71,2.87)
110.0	1.31	(0.31,3.58)

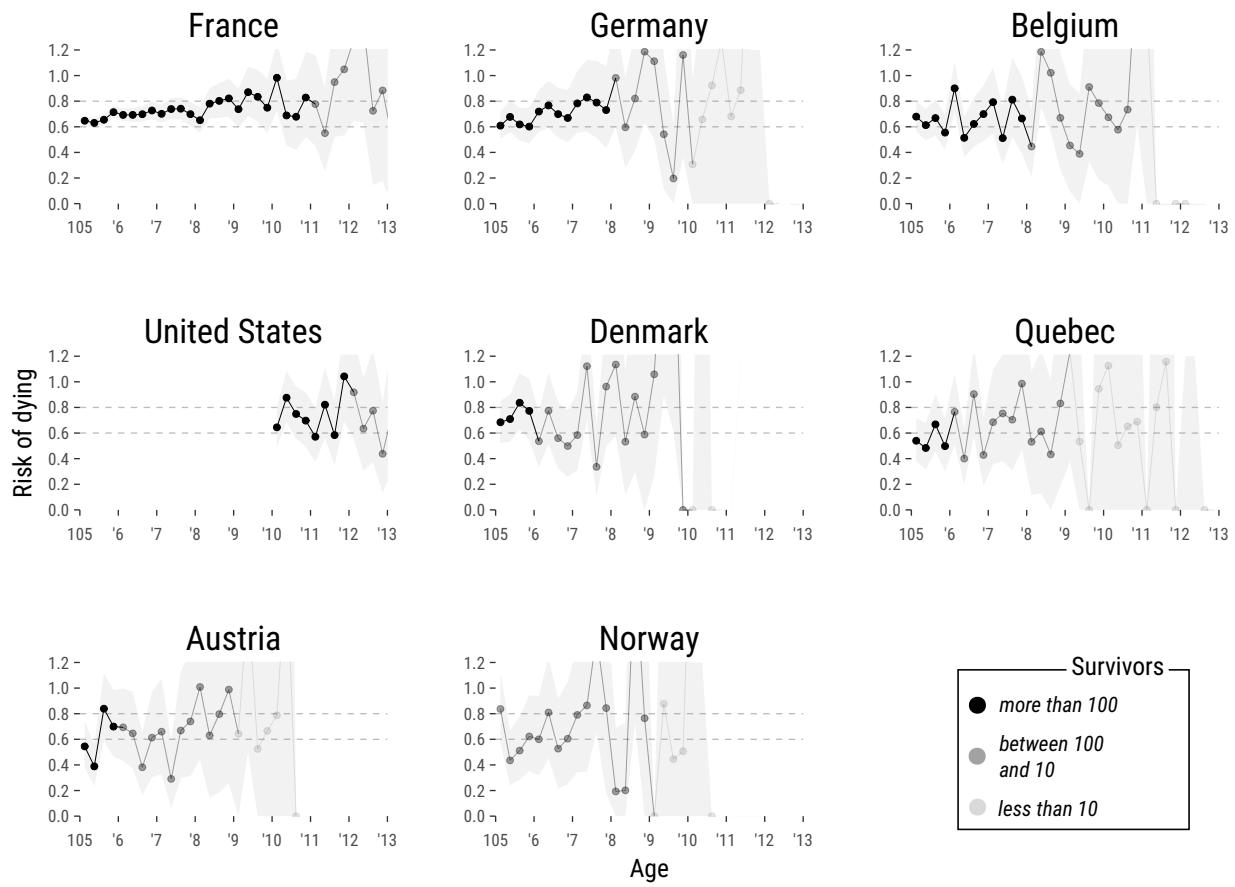


Fig. S1. Risk of dying between ages 105 and 113 with 95% confidence intervals for females. The risk of dying is expressed in age intervals of three months. The darker the shade of the dot plot, the greater the number of survivors during that specific age (i.e. subjects exposed to the risk of dying).

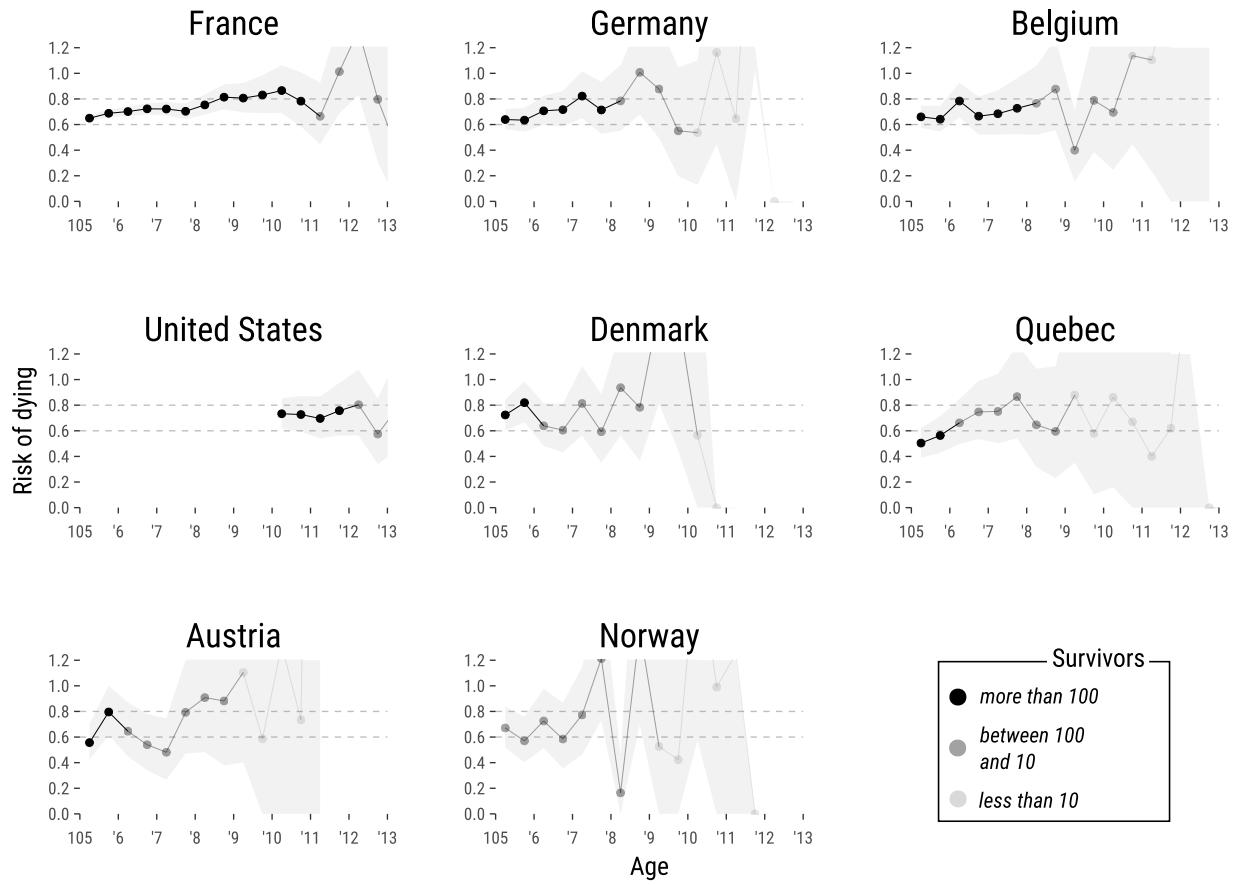


Fig. S2. Risk of dying between ages 105 and 113 with 95% confidence intervals for the total population. The risk of dying is expressed in age intervals of six months. The darker the shade of the dot plot, the greater the number of survivors during that specific age (i.e. subjects exposed to the risk of dying).

2. Coherent patterns between deceleration and levelling off in the risk of dying

In this section we perform additional analyses to examine the concordance of the trajectory of the risk of dying before age 105 with our estimates. Specifically, we calculated the risk of dying between ages 70 and 104 using cohort mortality data from the Human Mortality Database (2021). We retrieved age-specific death counts $D(x)$ and exposures $E(x)$ comprehending the same cohorts and populations included in our analysis in order to enable comparisons.

In Figure S3 we show the calculation of the risk of dying for females in France, Germany and Belgium from ages 70 to 113 using both datasets. Blue circles show estimations from the Human Mortality Database and black points are our non-parametric estimations of the risk of dying from the International Database of Longevity (i.e. same results as in Figure 2 in the manuscript). Note that the estimates in Figure S3 are in log scale.

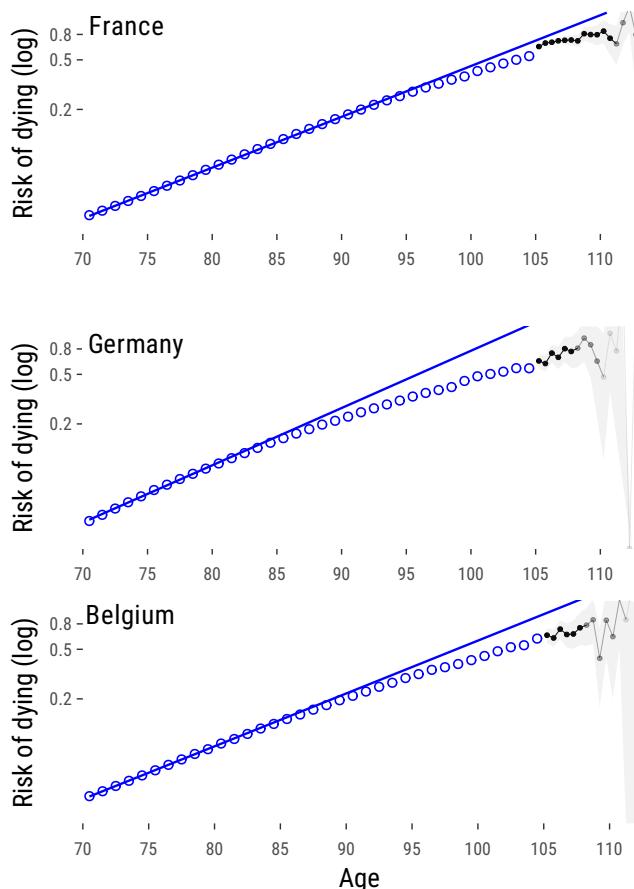


Figure S3. Risk of dying from ages 70 to 113 for females. Estimates from age 70 to age 105 were retrieved from the Human Mortality Database (2021)

Figure S3 shows concordant patterns between both datasets. Moreover, a deceleration in the increase of the risk of dying takes place before age 100. To formally test this pattern, we fitted a Gompertz model from ages 70 and 80 such that the hazard is given by $\mu(x) = ae^{bx}$, where the

parameter “a” indicates the initial value of the risk of dying and parameter “b” represents the rate of increase in $\mu(x)$. The estimation was performed by maximizing the Poisson log-likelihood:

$$L(x) = \sum \left(D(x) * \ln(\mu(x)) - E(x) * \mu(x) \right)$$

Blue straight lines in Figures S3 show the resulting Gompertz hazard. The parameters of the model are shown in Table S2. From Figure S3, we can observe that the risk of dying deviates from the Gompertz pattern before age 100. Depending on the population such deviations occur at ages between 80 and 90. The findings of this analysis are in line with previous investigations arguing for a deceleration in the risk of dying and a deviation from the Gompertz parametric model occurring prior age 100 (Perks (1932), Horiuchi and Wilmoth (1998)).

Table S2. Parameters and goodness of fit for Gompertz hazard model using cohort data from the Human Mortality Database, Females.

Gompertz hazard model fitted to HMD data to ages 70-80				
Country	Parameter	Estimate (95% CI)	Log-likelihood	AIC
France	a	0.03 (0.03, 0.03)	-1,246,821.463	2,493,646.926
	b	0.09 (0.09, 0.09)		
Germany	a	0.03 (0.03, 0.03)	-7,914,179.907	15,828,363.813
	b	0.11 (0.11, 0.11)		
Belgium	a	0.03 (0.03, 0.03)	-3,029,940.826	6,059,885.653
	b	0.1 (0.1, 0.1)		
Denmark	a	0.03 (0.03, 0.03)	-1,323,976.896	2,647,957.792
	b	0.09 (0.09, 0.09)		
Austria	a	0.03 (0.03, 0.03)	-1,246,821.463	2,493,646.926
	b	0.1 (0.1, 0.1)		
Norway	a	0.03 (0.03, 0.03)	-530,985.066	1,061,974.133
	b	0.11 (0.11, 0.11)		

References

- Human Mortality Database. University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at www.mortality.org or www.humanmortality.de (2021).
- D. R. Steinsaltz, K. W. Wachter, Understanding mortality rate deceleration and heterogeneity. *Mathematical Population Studies.* 13, 19–37 (2006).
- M. Finkelstein, V. Esaulova, Asymptotic behavior of a general class of mixture failure rates. *Advances in Applied Probability.* 38, 244–262 (2006).
- C. C. Horvitz, S. Tuljapurkar, Stage dynamics, period survival, and mortality plateaus. *The American Naturalist.* 172, 203–215 (2008).
- Perks, W. (1932). On some experiments in the graduation of mortality statistics. *Journal of the Institute of Actuaries (1886-1994)*, 63(1), 12-57.
- Horiuchi, S., & Wilmoth, J. R. (1998). Deceleration in the age pattern of mortality at olderages. *Demography*, 35(4), 391-412.

Stratification in health and survival after age 100: evidence from Danish centenarians

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RESEARCH

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Stratification in health and survival after age 100: evidence from Danish centenarians

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Abstract

Background: The existence of a super-select group of centenarians that demonstrates increased survivorship has been hypothesized. However, it is unknown if this super-select group possesses similar characteristics apart from extreme longevity.

Methods: In this study, we analyse high-quality health and survival data of Danish centenarians born in 1895, 1905 and 1910. We use Latent Class Analysis to identify unobserved health classes and to test whether these super-select lives share similar health characteristics.

Results: We find that, even after age 100, a clear and distinct gradient in health exists and that this gradient is remarkably similar across different birth cohorts of centenarians. Based on the level of health, we identify three clusters of centenarians - robust, frail and intermediate - and show that these groups have different survival prospects. The most distinctive characteristic of the robust centenarians is the outperformance in different health dimensions (physical, functional and cognitive). Finally, we show that our health class categorizations are good predictors of the survival prospects of centenarians.

Conclusions: There is a clear stratification in health and functioning among those over 100 years of age and these differences are associated with survival beyond age 100.

Keywords: Heterogeneity, Centenarian, Survival, Health, Latent class analysis

Background

Those who live to the oldest ages, particularly centenarians, are a select group [1]. Medford et al. [2] discuss the possibility of an additional layer of selection among centenarians – a so called “super-select” group – that consistently survives the longest beyond the age of 100 years. These individuals are the frontrunners of longevity, surviving as far as the 95th percentile of the distribution of lifespans above age 100 (i.e. beyond age 105) [2] and they exhibit greater improvements in their

individual lifespan than other centenarians [3, 4]. Though some may be robust from birth, resilience at younger ages does not necessarily translate into resilience during old age because an individual is exposed to the risk of sickness over their entire life course and may become infirm before reaching old age. It was previously believed that at extreme ages, survival chances were largely random and more driven by stochastic determinants than anything else [5]. However, Medford et al. [2] postulate that the super-select group of lives benefits most from improvements in medical technology and healthcare advances and are best positioned to take advantage of further increases in human lifespan. This hypothesis implies that (i) the super-select might share

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similar traits, (ii) such traits might be common across different birth cohorts and (iii) survival to extremely old ages may not be as random as some suggest. Therefore, a better understanding of the characteristics of exceptionally long-lived individuals may help to shed light on what is required for healthy aging.

Apart from extreme longevity, what traits distinguish the super-select? Centenarians have defeated death for at least 100 years, yet, no centenarian is exactly the same as another [1]. This uniqueness is due to different lifestyles [6–8], behaviour [9, 10], genetics [11], physiological make up [12–14], environmental determinants [15], exposure to prior and ongoing medical treatment [16–18] and many unobserved or unobservable factors [19] that ultimately lead to disparate lifespans. Most centenarians die within the first 2 years after reaching age 100 with relatively few surviving much longer¹ [20]. Heterogeneity in the context of individual lifespans and, in both observed and unobserved traits, is therefore natural and common among centenarians [21]. This inherent heterogeneity entails that some centenarians will make it to the frontier of survival [22] by chance and not necessarily because of any traits that they have in common with the super-select [5]. Similarly, some might be categorized as super-select but will die soon after their 100th birthday. Therefore, in order to correctly determine the traits of the super-select, it is paramount that the issue of heterogeneity is carefully addressed.

Previous studies on the health of nonagenarians (i.e. 93–95 years old) [23] provide valuable hints on the expected traits to be found in the super-select centenarians (i.e. 95th percentile of the distribution of lifespans above age 100, beyond age 105 [2]). By using cluster analysis to control for heterogeneity in health, some researchers [24, 25] have shown that nonagenarians can be categorized according to specific health classes, where one class has a consistent advantage in relation to the others. It has also been shown that factors which are usually good at differentiating and predicting survival at younger ages (e.g. smoking, obesity level, education, number of chronic diseases) do not explain survival differences among nonagenarians [26]. Instead, cognitive and physical abilities and to some extent, an optimistic personality, are regarded as strong predictors [26–28]. Further, survival among nonagenarians is improving across cohorts [29]. These improvements are accompanied by better health and functioning across the health spectrum [30–33].

It cannot be taken for granted that the associations between health and survival previously shown for nonagenarians will automatically apply for those aged 100 or

more. These associations [26, 30] cannot be blindly extrapolated to centenarians (or individuals surviving beyond age 100), because only 10–15% of nonagenarians make it to age 100 [20]. Furthermore, studies in Denmark and Sweden have shown that improvements in survival for centenarians are negligible when looking at the median and mean lifespan above age 100 [20]. Survival improvements for Denmark are observed for only a relatively small proportion, the super-select (i.e. the 95th percentile of the distribution of lifespans above age 100, above age 105) [2] and are not present for Sweden. Therefore, the assessment of health characteristics among centenarians is important to understand if survival above age 100 is a random process or if there are patterns that drive the survival improvements of the super-select. No commonalities among health characteristics might explain the lack of survival improvements observed in the mean lifespan of centenarians [20].

The aim of the study is to reveal the health characteristics that distinguish super-selected lives surviving more than 100 years. We hypothesize that the super-select are the most resilient centenarians in terms of health, by virtue of their capacity to enhance their survival chances and reach the frontier of human survival. Robustness is therefore linked with the plasticity of ageing at the individual level, in the sense that, the most robust individuals exhibit greater malleability in their lifespans. We identify robustness via the analysis of high-quality data from the 1895, 1905 and 1910 Danish Birth Cohort Studies [34] with a statistical technique known as Latent Class Analysis [35–40]. We test the predictive power of our findings by computing the Area Under the Curve statistic (AUC, see e.g. Robin et al. [41]). The key contribution of this study is in showing a clear stratification in health and functioning among those over 100 years of age and these differences are associated with survival beyond age 100.

Methods

Centenarian health data was retrieved from the 1895, 1905 and 1910 Danish Birth Cohort Studies. These are national population-based surveys with no exclusion criteria. All individuals born in 1895, 1905 and 1910 in Denmark were contacted to be interviewed and physically and cognitively tested during the year they would have turned 100 years. The 1895 cohort comprised of 207 out of 276 (75%) invited to participate and was examined by a geriatrician and a nurse. The assessments of the 1905 and 1910 cohorts were conducted by a specialized survey agency and comprised of 256 out of 439 (59%) and 273 out of 428 (63%) invited participants respectively. If someone was unable to participate because of their health status, a proxy respondent was invited to participate in the interview.

¹In Denmark in 2019 around 60% of female and 66% of male centenarians die by the age of 102 (Human Mortality Database, 2020).

We use four indicators to capture different health dimensions: physical ability, functional status, cognitive status, and self-rated health. The selection of the indicators was based on previous studies showing that these characteristics are related to the survival of nonagenarians [27]. The Chair Stand test was used to assess physical ability as it has been shown to be associated with lower body strength, disability, and survival at older ages in several studies [26, 27, 30, 42–44]. Individuals who can stand up from a chair without the use of arms are in better physical health than those who need to use hands or those who cannot [42]. Functional status was assessed by five questions out of eleven questions regarding the ability to perform activities of daily living (ADL): bathing, dressing, toileting, ability to walk and feeding. These five questions were used to calculate the Katz' disability score, where individuals were categorized into according to their answers [28, 45]. The cognitive status of centenarians was evaluated using the Mini-Mental State Examination (MMSE), which considers 19 questions. Such questions range from recalling dates and places (e.g. "What day of the week is it today?") to those where the individual is asked to perform arithmetic calculations (e.g. "Now I will ask you to deduct 7 from 100. Then you deduct 7 from the number you arrived at and continue to deduct 7 until you are asked to stop"). The higher the MMSE score, the better the cognitive status (0–30). We divided it into three categories: 24–30 indicates no cognitive impairment, 18–23 mild cognitive impairment and 0–17 severe cognitive impairment. This categorization is based on previous studies [26, 27, 30, 46]. It is important to note that five of the MMSE questions cannot be answered by individuals who are visually impaired. However, we used the results from the completed test to impute the missing values due to being visually impaired, hereby lowering this bias. Self-rated health answers were classified into three categories: "excellent or good", "acceptable" and "poor or very poor" [47].

It is worth noting that the questionnaire used in the assessment of health characteristics of centenarians for the 1895 cohort is slightly different from the one used for the 1905 and 1910 cohorts. First, the 1895 cohort survey does not include the Chair Stand test. Second, in the 1895 cohort, self-rated health was assessed with the question "Do you feel well considering your age?" The answers were (1) yes, (2) no and (3) reasonable. For the 1905 and 1910 cohorts, Self-Rated health was assessed with the question "All things considered, how do you consider the present status of your health?". The answers were (1) very good, (2) good, (3) acceptable, (4) bad and (5) very bad. The answers of the 1905 and 1910 questionnaires were grouped into three categories (1) very good/good, (2) acceptable and (3) bad/very bad to match

the three categories of the 1895 questionnaire. In addition, there were too few observations in the very bad and very good categories. The three-item categorization of self-rated health is also followed in previous studies of nonagenarians [26, 30, 48]. It is important to highlight that because of these differences in questionnaires, results from the 1895 are not directly comparable to the other two cohorts (1905 and 1910). Detailed information about the surveys is available in [34].

The four indicators of health considered in the analysis exhibited missing values (see Supplemental Material). To handle them without introducing bias into our results, we created a "not tested" category for Chair Stand, MMSE and Self-Rated health to classify individuals that have missing values because they could not be tested due to their very poor health. For the Chair Stand score, individuals with missing values who could not perform all eleven questions regarding activities of daily living (ADL) in the survey were included in the "not tested" category. For MMSE and Self-Rated health, we categorized those individuals that reported missing values, but with the answers provided by a proxy respondent, as "not tested". The rationale being that these tests cannot be performed by proxy respondents. For the Katz's disability score we did not create a "not tested" category. However, this score reported very few missing values (2 individuals in each cohort). The creation of the "not tested" category allowed us to considerably reduce the number of missing values for participants who were unable to respond due to ill health [36]. However, there were still some missing values in the dataset (see Table A4 in Supplemental Material). Thus, we remove individuals who have missing values in at least one of the variables in the analysis.²

The date of death of each centenarian in Denmark (participants and non-participants) was retrieved from the Danish Civil Registration System. Some survey participants died before turning age 100 (e.g. ages 99.7, 99.5, etc.). We excluded these individuals from the main analysis to avoid immortal time bias in the calculation of survival probabilities [49]. After removing individuals with missing values in at least one of the variables in the analysis and those that did not survive to age 100 (37 in the 1895 cohort, 36 in the 1905 cohort and 49 in the 1910 cohort, see Supplemental Material), we analyse 170 individuals in the 1895 Cohort; 195 individuals in the 1905 Cohort and 223 in the 1910 Cohort. Tables A5 and A9 of the Supplemental Material show the characteristics of individuals included in the analysis. To test if our

²The use of statistical imputation techniques like mean substitution or multiple imputation was avoided because these procedures might bias the results of the Latent Class Analysis and make comparisons among cohorts more uncertain. Therefore, we performed the analysis considering only the individuals that have complete values.

data is representative of the entire population, we use the log-rank test to compare survival trajectories of participants included in the analysis against those that did not participate in the survey. Survival trajectories of both groups (participants included in the analysis and non-participants) for the 1905 and 1910 cohorts are similar, which indicates that data used in our analysis is representative of national population of Danish centenarians for those cohorts. For the 1895 cohort, survival trajectories of individuals included in the analysis are statistically different from the survival trajectories of non-participants. This indicates a possible health selection in the 1895 cohort. We still analyse data of the cohort 1895 to determine if their health characteristics differ from the health characteristics of the 1905 and 1910 cohorts.

Statistical analysis

We perform a Latent Class Analysis (LCA) to shed light on the unobserved heterogeneity in health among Danish centenarians. LCA is a statistical method used to identify unobserved classes of individuals via observed categorical variables [36–40, 50]. By considering several individual characteristics, the LCA determines individual probabilities of belonging to the latent classes and probabilities of finding a person with a certain characteristic in each class. Reference [35] provides a thorough explanation about the LCA model and in the Supplemental Material we provide more details about the specific LCA setting used in this study. Individuals in each class share similar characteristics and at the same time, they are different from individuals in other classes. Our aim is to identify health classes to further contrast the survivorship of individuals belonging to each of them. We consider different dimensions of health in the LCA: physical health (Chair Stand test), functional status (Katz's Disability Index), cognitive impairment (MMSE) and Self-Rated Health. It is known that there are sex differences in health and survival among centenarians [51]. For this reason, we included sex as a covariate that allows us to place individuals into classes [35]. We could not stratify the analysis by sex because of the number of male centenarians that participated in the study is much smaller than the number of female centenarians in the study (see Table A5 in the Supplemental Material for details).

We performed LCA for each cohort. Since individuals in the 1895 cohort are not directly comparable to the ones in 1905 and 1910 due to differences in the questionnaire used and their survival trajectories differ from the non-participants (see details in Data section), we present the analysis of the 1895 cohort in the Supplemental Material and focus here on the 1905 and 1910 cohorts. For each cohort, various LCAs were performed by changing the number of classes in each iteration, from two to six. We

considered six health classes to be the maximum possible in each cohort. More than six classes would imply high heterogeneity in health patterns but also small and meaningless classes. The optimal number of classes was selected by looking at the Akaike and Bayesian Information Criteria (AIC and BIC respectively) but also considering the health patterns and size of each class. Once the optimal number of classes in each cohort was obtained, each centenarian was assigned to a single health class. Then, based on their ages at death, we computed survival curves and the associated 95% confidence intervals by health class and by cohort using the Kaplan-Meier estimator. We assess whether there are differences in survival among the different health classes by computing the log-rank test. This test compares the entire survival experience between groups and can be thought of as a test of whether the survival curves are identical (overlapping) or not [52].

Finally, we estimated the area under the curve (AUC) to test the ability of health classes to predict the chance of surviving to the frontier of survival. The AUC ranges from 0 to 1; a higher AUC implies a better prediction [41]. We define the frontier of survival [2, 53] as the 95th percentile of the centenarian age-at-death distribution. Note that such ages change across cohorts according to mortality improvements. In Table 1 we show such ages and values for the AUC calculated for different percentiles.

Results

Results from the Latent Class Analysis (LCA) indicate that the optimal number of health classes for the 1905 and 1910 cohorts is three (see Supplemental Material). For the 1895 cohort the optimal number of health classes is two, which indicates that there is less heterogeneity in health for this cohort possibly due to health selection. Indeed, as indicated in Section 2, survival trajectories of survey participants are statistically different to those that did not participate in the survey (see Table A1 in the Supplemental Material). Therefore, the results for the 1895 cohort are not nationally representative. In this section, we describe and compare the results of the 1905 and 1910 cohorts only (which are country representative). Results for the 1895 cohort can be found in the Supplemental Material.

Sex, included in the model as a covariate, is not statistically significant in either of the cohorts. This could be because most of centenarians are females (around 80% in each cohort). In the Supplemental Material we include a sensitivity analysis where only females are considered. The LCA health classes obtained from females-only analysis are practically the same as the ones obtained in the original analysis. This could be attributed to the fact that most of centenarians are women but also that health differences among sexes are already present in the health dimensions included in the LCA.

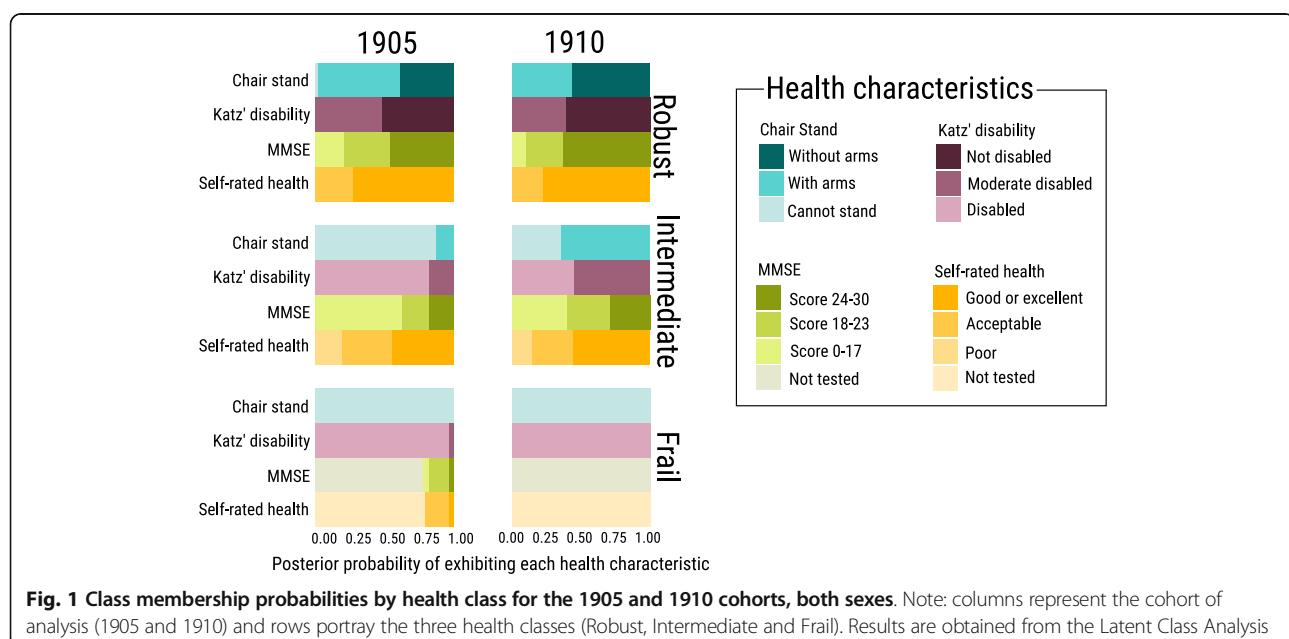
Every LCA class is composed of individuals who share similar health characteristics. Figure 1 shows the composition of each class for the 1905 and 1910 cohorts. Based on their characteristics, we denote the classes as robust, frail and intermediate. Each bar represents a health characteristic and the size of the coloured bar depicts the probability of depicting such characteristic. For example, robust centenarians have a 44% chance of being able to stand up from a chair with the use of hands (aqua green bar) and a 56% of being able to do so without using hands (dark green bar).

Robust centenarians comprise around 117 individuals (60%) of the 1905 and 90 individuals (40%) of the 1910 cohort population. They are likely to stand up from chairs by using their arms and have high probabilities of not being physically disabled at all or being only moderately disabled. It is likely that most of them do not show significant cognitive impairment. The majority perceive their health as good. Frail centenarians on the other hand, are likely to not be able to stand up from a chair and reporting physical disability. Due to their poor health, many of them could not be tested for their cognitive status and self-rated health. Frail centenarians comprise 16% and 17% of the 1905 and 1910 cohorts respectively (around 35 individuals in each cohort). Finally, the intermediate health class comprises 24% and 42% of the 1905 and 1910 cohorts respectively. This class includes centenarians who physically and cognitively perform worse than the robust centenarians. Most of them perceive their own health to be good or acceptable.

It has been shown that nonagenarians from younger cohorts perform better in health and functioning than those

from older cohorts [30]. Similar improvements in health and functioning are also portrayed in our analysis for centenarians. For example, in Fig. 1 we observe that the intermediate class of the 1910 is comprised by individuals that are more likely to be in better health in comparison to those in the intermediate class from 1905. Likewise, there is a larger share of individuals in the intermediate class in 1910 than in 1905. The robust health profile of the 1910 class is also slightly better than the 1905 class. Both health classes (robust and intermediate) comprise together around 82% of individuals in each cohort, while the frail class comprises around 18% of individuals. This indicates that improvements in health and functioning in health across cohorts are reflected in better health profiles for the robust and intermediate classes.

As mentioned above, improvements in health and functioning are reflected in higher health standards for the robust and intermediate classes in 1910. Still, the characteristics of the robust centenarians are very similar across the 1905 and 1910 cohorts (see Table A6 in Supplemental Material). Despite of not being directly comparable, the robust health class in the 1895 cohort resembles the robust health classes in the 1905 and 1910 cohorts. These commonalities in health classes across cohorts support our hypothesis about a group of centenarians outperforming in health outcomes. Thus, the question arises: are the robust centenarians also outperforming in survival? To answer this question, we computed survival curves and the associated 95% confidence intervals for the three health classes found in each cohort. Figure 2 shows the results for the 1905 and 1910 cohorts.



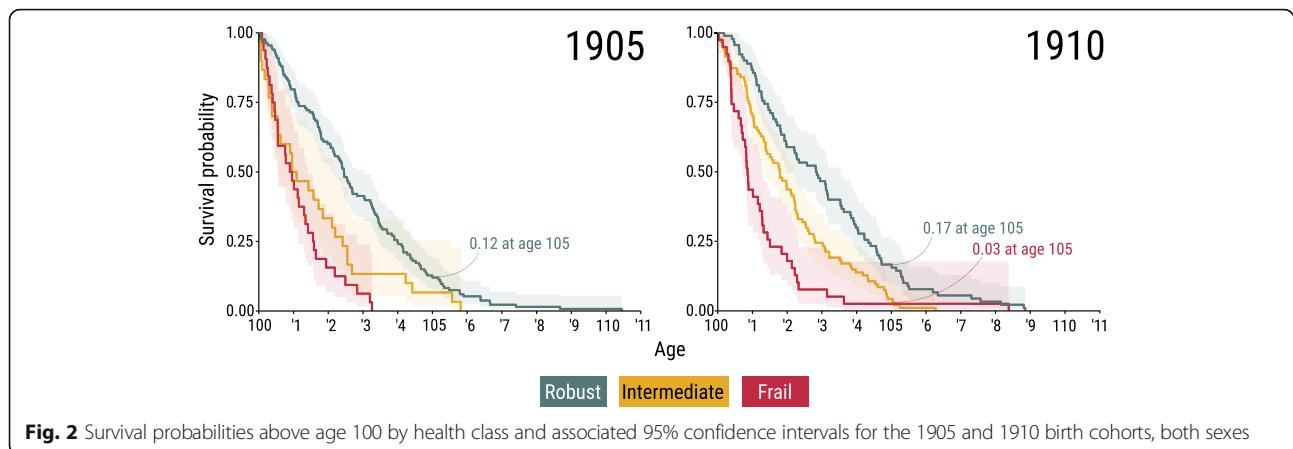


Fig. 2 Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1905 and 1910 birth cohorts, both sexes

Figure 2 shows clear differences in survival among health classes with generally non-overlapping confidence intervals. Note, however, that at the very highest ages, the confidence bands grow wider and tend to overlap due to the very small number of survivors at those ages. Nonetheless, the log-rank test confirms formally that the three survival curves are statistically distinct (see Supplemental Material). Robust centenarians live longer than those in the other two health classes. In the 1905 cohort, their probability of survival to 105 is 0.12. For the 1910 cohort, the equivalent survival probability is 0.17, which is almost six times that for those in the frail health class. A survival gap between the robust and frail classes is also present in the 1895 cohort (see Figure A2 in Supplemental Material).

Next, we tested the ability of health classes to predict survivorship to the frontier of survival, (defined by Medford et al (2019) as the 95th percentile of the centenarian age-at-death distribution) by computing the AUC (area under the curve). Depending on the percentile, AUC ranged between 0.65 and 0.68 for the 1905 cohort and 0.71 and 0.76 for the 1910 cohort (see Table 1). For the 1895 cohort, the area under the curve was estimated to be around 0.70. The AUC shows that the health class is a good predictor for reaching the frontier of survival. In particular, the AUC is consistently greater for younger cohorts, which indicates that the LCA health classes are slightly better at predicting survival of centenarians in the 1910 cohort than for centenarians in the 1905 cohort.

In a previous study, Thinggaard et al. [26] showed that the combination of Chair Stand and MMSE scores are good predictors of survival among nonagenarians so we compare the predictive ability of this approach with our LCA health classes.³ Both approaches (LCA health classes and Thinggaard et al. [26]) are useful in determining

the survival chances to extreme ages (see Supplemental Material). However, our LCA health classes provide a more thorough description of individual health, enabling us to identify similarities in the health of centenarians. The LCA health classification provides a framework to determine the traits involved in the optimal pathways of healthy ageing.

Sensitivity analysis

The focus of the present study is the relationship between health and survival trajectories of centenarians. For this reason, the LCA health classes only consider health dimensions (i.e. disability, functional health, cognitive status, and self-rated health). The selection of such health indicators is based on previous studies showing their association to survival at high advanced ages [26, 27, 30]. Nonetheless, we test how the class membership of the LCA health classes is affected when adding other factors. Specifically, we performed a sensitivity analysis of the LCA health classes by including information about smoking behaviour of the centenarians in addition to the four dimensions of health mentioned above. We show that the inclusion of smoking does not affect the identification of health classes (see

Table 1 Ability of health categorization to predict survivorship to the frontier of survival measured by the area under the curve (AUC) statistic. 1905 and 1910 cohorts

Percentile	1905 Cohort		1910 Cohort	
	Age	AUC	Age	AUC
95th	105.61	0.65	105.72	0.71
96th	105.95	0.68	106.08	0.71
97th	106.26	0.68	106.39	0.76
98th	106.95	0.68	107.09	0.76
99th	107.94	0.68	108.15	0.73

Note: The AUC ranges from 0 to 1; a higher AUC implies a better prediction. Medford et al. (2019) define the frontier of survival as the 95th percentile of the centenarian age-at-death distribution. We included upper percentiles as a robustness check

³Following Thinggaard et al (2016) approach, we categorise those that can stand up with and without hands from their chair and having a MMSE > 24 as robust. Under this approach, the AUC ranged between 0.60 and 0.72 for the cohort 1905 and between 0.61 and 0.66 for the cohort 1910. See Table A14 in the Supplemental Material.

Supplemental Material). This finding is in line with previous studies showing that smoking behaviour is not related to survival at high advanced ages [26, 27].

Next, we performed a sensitivity analysis where we only consider individuals with complete information (i.e. the “non tested” categories in Chair Stand, MMSE and Self Rated Health were not created, and all missing values were removed). Therefore, the sample size was reduced substantially as we only considered 170 individuals from the 1905 cohort and 182 participants from the 1910 cohort. Still, health classes remain identifiable (Figure A6 of Supplemental Material). For example, the robust class remains almost identical to the LCA analysis in Fig. 1 (i.e. when including the “no tested” category). The reason for this is that individuals with missing data are individuals in worse state of health, and they are allocated in the frail health classes. With this sensitivity analysis we confirm that there are no biases in the health classes introduced by the “not tested” category.

We performed two additional sensitivity analysis. In the first of them, we only considered females in the computation of LCA health classes. As described at the beginning of the Results section, this analysis is motivated by the fact that most centenarians in our data are females. Second, we performed a LCA by including all individuals that died before age 100. In both analyses we obtained similar results to the ones from the original LCA health classifications. Thus, we conclude that our analysis adequately captures the relationship between unobserved health categories and survival at extremely old ages. All the results from the sensitivity analyses can be found in the Supplemental Material.

Discussion

Those surviving to the oldest ages (i.e. beyond age 105) had better health at age 100 than other survivors from their cohort. The major contributions of this study are that (i) we show the existence of a clear stratification in health and functioning among those 100 years of age and (ii) we shed light on the characteristics of the super-select centenarians (i.e. those surviving to age 105 and above). To do so, we use a high quality dataset [34] and consider different dimensions of health: physical health (Chair Stand test), functional status (Katz’s disability Index), cognitive impairment (MMSE) and Self-Rated Health which when taken together provide a well-rounded view of centenarian health and functioning.

The majority of centenarians are females and the most distinctive characteristics of the robust cluster versus the other health clusters stem from their outperformance in physical, functional and cognitive health. Most of them perceive their own health to be good or excellent. This perhaps could explain the upward trend in lifespans previously observed within this group [2]. In contrast, the

intermediate and frail individuals show greater levels of physical and cognitive impairment and they have lower chances of surviving in comparison to those in the robust health class.

It was previously believed that at highest ages, the chances of survival were mostly random events [54, 55]. This school of thought suggests that survival is driven by stochastic determinants [5]. In reality, human survival is more idiosyncratic than this. We show that even at age 100 there are clear disparities in the survival prospects of centenarians based on their health profile. Furthermore, our study revealed that centenarians belonging to the robust health class are consistently in better health and survive the longer than the other centenarians. These super-select centenarians share similar health characteristics and were present in all the cohorts studied here: clearly identified in the 1905 and 1910 cohorts and slightly less clear cut in the 1895 cohort. However, we also show that there is selection in the 1895 cohort because the survival trajectories of the survey participants are statistically different than those that did not participated in the survey. Therefore, the results of the 1895 cohort should be taken with caution.

Limitations of the study

One clear limitation of this study is that health characteristics are recorded only at age 100 but decline is likely to be rapid after then. At very old ages, health deterioration is likely to appear from one year to another [48]. Still, the data used in this analysis measures a sufficiently wide range of functioning so that it reasonably depicts an individual’s general health status [30, 34]. Likewise, it is unknown if similar findings are observed among the centenarians of other countries. In Sweden, for example, Medford et al. [2] do not find a super-select group with increased plasticity of individual lifespans. It would be interesting to determine if a robust health-class is found in Sweden and to compare the results with our findings.

We also acknowledge that some heterogeneity in survival is still uncounted in our analysis and this could be attributed to some stochastic process. An analysis with more comprehensive measures (e.g. a comprehensive geriatric assessment) of the general health of centenarians could be useful to disclose such heterogeneity. However, at present, we do not count with such data, which is a limitation of the study.

Apart from health, other factors such as socio-economic factors (i.e. education, income, etc.), lifestyle (e.g. living arrangements, calorie intake), genetic endowments and demographic characteristics might be useful to depict a broader centenarian phenotype. However, adding too many indicators to the LCA analysis might become problematic due to our small sample sizes. This could lead to meaningless LCA classes (i.e. empty

classes). Instead, a similar approach as Goldman et al. [56] could be implemented to this aim.

Conclusion

We conclude that survival advances beyond age 100 are mainly driven by this super-select group of the healthiest individuals surviving for a longer time. This is not to say that those in poor health have not been living longer as well. They have been. However, the super-select lives have been living longer than any other group and any further pushing of the frontier of survival forward will most likely be by those in the most robust health and not those in poor health. Any improvements in the dimensions of health studied here could lead to a higher prevalence of robust centenarians and ultimately to a longer living population.

Abbreviations

LCA: Latent Class Analysis; BIC: Bayesian Information Criterion; AUC: Area Under the Curve; MMSE: Mini-Mental State Examination; ADL: Activities of Daily Living

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12877-021-02326-3>.

Additional file 1: Table A1. Survival probabilities above age 100 for participants and non-participants and associated 95% confidence intervals for the 1895 cohort.

Additional file 2: Table A2. Survival probabilities above age 100 for participants and non-participants and associated 95% confidence intervals for the 1905 cohort.

Additional file 3: Table A3. Survival probabilities above age 100 for participants and non-participants and associated 95% confidence intervals for the 1910 cohort.

Additional file 4: Table A4. Description of participants included in the analysis and missing values per health characteristic. Cohorts 1895, 1905 and 1910.

Additional file 5: Table A5. Summary statistics by gender for the cohorts 1905 and 1910.

Additional file 6: Table A6. Comparison of the class membership probabilities of the robust class for the 1905 and 1910 cohorts.

Additional file 7: Table A7. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1905 cohort.

Additional file 8: Table A8. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1910 cohort. Health classes were obtained from the Latent Class Analysis.

Additional file 9: Table A9. Summary statistics by gender for the cohort 1895.

Additional file 10: Figure A1. Class membership probabilities by health class for the 1895 cohort, both sexes.

Additional file 11: Figure A2. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the cohort 1895, both sexes.

Additional file 12: Table A10. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1895 cohort.

Additional file 13: Table A11. Goodness of fit for the Latent Class Analysis varying the number of clusters. Cohorts 1895, 1905, 1910.

Additional file 14: Figure A3. Class membership probabilities by health class for the 1905 and 1910 cohorts considering only females.

Additional file 15: Figure A4. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1905 and 1910 cohorts considering only females in the Latent Class Analysis.

Additional file 16: Table A12. Area under the curve by percentile for the 1905 and 1910 cohorts considering only females in the analysis.

Additional file 17: Figure A5. Class membership probabilities by health dimension for the 1905 and 1910 cohorts including Smoking in the Latent Class Analysis, both sexes.

Additional file 18: Figure A6. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1905 and 1910 cohorts including Smoking in the Latent Class Analysis, both sexes.

Additional file 19: Table A13. Area under the curve by percentile for the 1905 and 1910 cohorts including Smoking in the analysis.

Additional file 20: Figure A7. Class membership probabilities by health dimension for the 1905 and 1910 cohorts including only individuals with complete observations, without the creation of "no tested" category.

Additional file 21: Figure A8. Class membership probabilities by health dimension for the 1905 and 1910 cohorts including those individuals that died before age 100 in the Latent Class Analysis, both sexes.

Additional file 22: Table A14. Area under the curve by percentile for the 1905 and 1910 cohorts using Chair Stand (able to stand with and without hands) and MMSE (>24).

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Authors' contributions

JAA, AM designed the study. KC, MT collected the data. JAA, AM, CS performed the statistical analysis. All authors co-drafted the manuscript, critically revised the results and approved the final version. All authors agree to be accountable for all aspects of the work.

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Availability of data and materials

Data supporting the findings of this study were used under license of the Regional Committees on Health Research Ethics for Southern Denmark (<https://en.nvk.dk/>). Data are available from the authors upon request.

Declarations

Competing interest

The authors declare that they have no competing interests.

Ethics approval and consent to participate

The Danish Birth Cohort studies received approval from the Scientific Ethical Review Board (trial numbers, 1895: 95/93 and 95/93 MC; 1905: VF-20040240; 1910: S-20100011) and the Danish Data Protection Agency (1905: 2015-41-3834; 1910: 2016-41-4552).

The Danish Birth Cohort studies are prospective studies that collected information of the health of the participants when they turned 100 years. Informed consent was obtained from participants when they were tested. In the case where individuals participated through a proxy responder, the proxy responder provided informed consent. Date of death was obtained from the Danish Civil Registration System.

All methods were carried out in accordance with relevant guidelines and regulations.

Consent for publication

Not applicable.

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References

- Vacante M, D'Agata V, Motta M, Malaguarnera G, Biondi A, Basile F, et al. Centenarians and supercentenarians: a black swan. Emerging social, medical and surgical problems. *BMC Surg.* 2012;12(Suppl 1):S36.
- Medford A, Christensen K, Skytthe A, Vaupel JW. A cohort comparison of lifespan after age 100 in Denmark and Sweden: are only the oldest getting older? *Demography.* 2019;56(2):665–77.
- Vaupel JW, Lundstrom H, editors. The future of mortality at older ages in developed countries. In: Future population of the world: what can we assume today? Laxenburg, Austria: International Institute for Applied Systems Analysis [IIASA]; 1994. p. 295–315.
- Vaupel JW. Biodemography of human ageing. *Nature.* 2010;464(7288):536–42.
- Hagberg B, Samuelsson G. Survival after 100 years of age: a multivariate model of exceptional survival in Swedish centenarians. *J Gerontol Ser A.* 2008;63(11):1219–26.
- Di Francesco A, Di Germanio C, Bernier M, de Cabo R. A time to fast. *Science.* 2018;362(6416):770–5.
- Mattson MP, Longo VD, Harvie M. Impact of intermittent fasting on health and disease processes. *Ageing Res Rev.* 2017;39:46–58.
- Stathakos D, Pratsinis H, Zachos I, Vlahaki I, Gianakopoulou A, Zianni D, et al. Greek centenarians: assessment of functional health status and life-style characteristics. *Exp Gerontol.* 2005;40(6):512–8.
- Voelcker-Rehage C. Cardiovascular and coordination training differentially improve cognitive performance and neural processing in older adults. *Front Hum Neurosci.* 2011;5(26):12.
- Oltmanns J, Godde B, Winneke AH, Richter G, Niemann C, Voelcker-Rehage C, et al. Don't lose your brain at work – the role of recurrent novelty at work in cognitive and brain aging. *Front Psychol [Internet].* 2017;8:117 [cited 2020 Nov 17]. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5292433/>.
- Sebastiani P, Solovjeff N, DeWan AT, Walsh KM, Puca A, Hartley SW, et al. Genetic signatures of exceptional longevity in humans. Gibson G, editor. *PLoS One.* 2012;7(1):e29848.
- Kennedy BK, Berger SL, Brunet A, Campisi J, Cuervo AM, Epel ES, et al. Geroscience: linking aging to chronic disease. *Cell.* 2014;159(4):709–13.
- Epel ES, Lithgow GJ. Stress biology and aging mechanisms: toward understanding the deep connection between adaptation to stress and longevity. *J Gerontol A Biol Sci Med Sci.* 2014;69(Suppl 1):S10–6.
- Conte M, Ostan R, Fabbri C, Santoro A, Guidarelli G, Vitale G, et al. Human aging and longevity are characterized by high levels of mitokines. *J Gerontol Ser A.* 2019;74(5):600–7.
- Calabrese EJ, Dhawan G, Kapoor R, Iavicoli I, Calabrese V. What is hormesis and its relevance to healthy aging and longevity? *Biogerontology.* 2015; 16(6):693–707.
- Meinow B, Parker MG, Thorslund M. Complex health problems and mortality among the oldest old in Sweden: decreased risk for men between 1992 and 2002. *Eur J Ageing.* 2010;7(2):81–90.
- Hadler NM. Rethinking aging growing old and living well in an overtreated society [Internet]. University of North Carolina Press; 2011. [cited 2020 Nov 17]. Available from: <https://dementiawebkentandmedway.org.uk/book-id/rethinking-aging-growing-old-and-living-well-in-an-overtreated-society>
- Smith AK, Williams BA, Lo B. Discussing overall prognosis with the very elderly. *N Engl J Med.* 2011;365(23):2149–51.
- Vaupel JW, Manton KG, Stallard E. The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography.* 1979;16(3):439–54.
- Modig K, Andersson T, Vaupel J, Rau R, Ahlbom A. How long do centenarians survive? Life expectancy and maximum lifespan. *J Int Med.* 2017;282(2):156–63.
- Pignolo RJ. Exceptional human longevity. *Mayo Clin Proc.* 2019;94(1):110–24.
- Barbi E, Laguna F, Marsili M, Vaupel JW, Wachter KW. The plateau of human mortality: demography of longevity pioneers. *Science.* 2018;360(6396):1459–61.
- Nosrati L, Enroth L, Raitanen J, Hervonen A, Jylhä M. Do successful agers live longer? The vitality 90+ study. *J Aging Health.* 2015;27(1):35–53.
- Montesanto A, Lagani V, Martino C, Dato S, De Rango F, Berardelli M, et al. A novel, population-specific approach to define frailty. *Age.* 2010;32(3):385–95.
- Dato S, Montesanto A, Lagani V, Jeune B, Christensen K, Passarino G. Frailty phenotypes in the elderly based on cluster analysis: a longitudinal study of two Danish cohorts. Evidence for a genetic influence on frailty. *Age.* 2012; 34(3):571–82.
- Thinggaard M, McGue M, Jeune B, Osler M, Vaupel JW, Christensen K. Survival prognosis in very old adults. *J Am Geriatr Soc.* 2016;64(1):81–8.
- Nybo H, Petersen HC, Gaist D, Jeune B, Andersen K, McGue M, et al. Predictors of mortality in 2,249 nonagenarians—the Danish 1905-cohort survey. *J Am Geriatr Soc.* 2003;51(10):1365–73.
- Engberg H, Christensen K, Andersen-Ranberg K, Vaupel JW, Jeune B. Improving activities of daily living in Danish centenarians—but only in women: a comparative study of two birth cohorts born in 1895 and 1905. *J Gerontol A Biol Sci Med Sci.* 2008;63(11):1186–92.
- Christensen K, Dobhammer G, Rau R, Vaupel JW. Ageing populations: the challenges ahead. *Lancet.* 2009;374(9696):1196–208.
- Thinggaard M, Jeune B, Osler M, Vaupel JW, McGue M, Christensen K. Are advances in survival among the oldest old seen across the spectrum of health and functioning? Newman A, editor. *J Gerontol Ser A [Internet].* 2020. [cited 2020 Jun 13]; Available from: <https://academic.oup.com/biomedgerontology/advance-article/doi/10.1093/gerona/glaa009/5701592>
- Brailean A, Huisman M, Prince M, Prina AM, Deeg DJH, Comijs H. Cohort differences in cognitive aging in the longitudinal aging study Amsterdam. *J Gerontol B Psychol Sci Soc Sci.* 2018;73(7):1214–23.
- Koivunen K, Sillanpää E, Munukka M, Portegijs E, Rantanen T. Cohort differences in maximal physical performance: a comparison of 75- and 80-year-old men and women born 28 years apart. *J Gerontol Ser A [Internet].* [cited 2020 Nov 17]; Available from: <https://academic.oup.com/biomedgerontology/advance-article/doi/10.1093/gerona/glaa224/5901594>.
- Munukka M, Koivunen K, von Bonsdorff M, Sipilä S, Portegijs E, Ruoppila I, et al. Birth cohort differences in cognitive performance in 75- and 80-year-olds: a comparison of two cohorts over 28 years. *Aging Clin Exp Res [Internet].* 2020; [cited 2020 Nov 17]; Available from: <https://doi.org/10.1007/s40520-020-01702-0>.
- Rasmussen SH, Andersen-Ranberg K, Thinggaard M, Jeune B, Skytthe A, Christiansen L, et al. Cohort profile: the 1895, 1905, 1910 and 1915 Danish Birth Cohort Studies—secular trends in the health and functioning of the very old. *Int J Epidemiol.* 2017;46(6):1746–1746.
- Hagenaars JA, McCutcheon AL, editors. Applied latent class analysis. Cambridge/New York: Cambridge University Press; 2002. 454 p.
- Strozza C, Pasqualetti P, Egidi V, Loretì C, Vannetti F, Macchi C, et al. Health profiles and socioeconomic characteristics of nonagenarians residing in Mugello, a rural area in Tuscany (Italy). *BMC Geriatr [Internet].* 2020;20(1):289 [cited 2020 Aug 17]. Available from: <https://bmccgeriatr.biomedcentral.com/articles/10.1186/s12877-020-01689-3>.
- Lafontaine L, Béland F, Bergman H, Ankri J. Health status transitions in community-living elderly with complex care needs: a latent class approach. *BMC Geriatr [Internet].* 2009;9(1):6 [cited 2018 Apr 24]. Available from: <http://bmccgeriatr.biomedcentral.com/articles/10.1186/1471-2318-9-6>.
- Looman WM, Fabbriotti IN, Blom JW, Jansen APD, Lutomski JE, Metzelthin SF, et al. The frail older person does not exist: development of frailty profiles

- with latent class analysis. *BMC Geriatr* [Internet]. 2018;18(1):84 [cited 2020 Mar 11]. Available from: <https://bmceriatr.biomedcentral.com/articles/10.1186/s12877-018-0776-5>.
39. Kino S, Bernabé E, Sabbah W. Socioeconomic inequality in clusters of health-related behaviours in Europe: latent class analysis of a cross-sectional European survey. *BMC Public Health* [Internet]. 2017;17(1):497 [cited 2020 Mar 11]. Available from: <http://bmcpublichealth.biomedcentral.com/articles/10.1186/s12889-017-4440-3>.
 40. Zammit AR, Starr JM, Johnson W, Deary IJ. Profiles of physical, emotional and psychosocial wellbeing in the Lothian birth cohort 1936. *BMC Geriatr*. 2012;12:64.
 41. Robin X, Turck N, Hainard A, Tiberti N, Lisacek F, Sanchez J-C, et al. pROC: an open-source package for R and S+ to analyze and compare ROC curves. *BMC Bioinformatics*. 2011;12(1):77.
 42. Guralnik JM, Ferrucci L, Simonsick EM, Salive ME, Wallace RB. Lower-extremity function in persons over the age of 70 years as a predictor of subsequent disability. *N Engl J Med*. 1995;332(9):556–62.
 43. Jones CJ, Rikli RE, Beam WC. A 30-s chair-stand test as a measure of lower body strength in community-residing older adults. *Res Q Exerc Sport*. 1999; 70(2):113–9.
 44. Cesari M, Onder G, Zamboni V, Manini T, Shorr RI, Russo A, et al. Physical function and self-rated health status as predictors of mortality: results from longitudinal analysis in the iSIRENTE study. *BMC Geriatr*. 2008;8:34.
 45. Katz S, Downs TD, Cash HR, Grotz RC. Progress in development of the index of ADL. *Gerontologist*. 1970;10(1 Part 1):20–30.
 46. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state": a practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*. 1975;12(3):189–98.
 47. Ware JE, Kosinski M, Keller SD. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. *Med Care*. 1996;34(3):220–33.
 48. Strozza C, Zarulli V, Egidi V. Understanding health deterioration and the dynamic relationship between physical ability and cognition among a cohort of Danish nonagenarians. *J Aging Res*. 2020;2020:1–8.
 49. Lévesque LE, Hanley JA, Kezouh A, Suissa S. Problem of immortal time bias in cohort studies: example using statins for preventing progression of diabetes. *BMJ* [Internet]. 2010;340:b5087 [cited 2020 Nov 23]. Available from: <https://www.bmjjournals.org/content/340/bmjb5087>.
 50. Dayton CM, Macready GB. Concomitant-variable latent-class models. *J Am Stat Assoc*. 1988;83(401):173–8.
 51. Hazra NC, Dregan A, Jackson S, Gulliford MC. Differences in health at age 100 according to sex: population-based cohort study of centenarians using electronic health records. *J Am Geriatr Soc*. 2015;63(7):1331–7.
 52. Kleinbaum DG, Klein M. Kaplan-Meier survival curves and the log-rank test. In: Kleinbaum DG, Klein M, editors. *Survival analysis: a self-learning text* [Internet]. New York: Springer; 2012. p. 55–96. [cited 2021 Apr 6]. (*Statistics for Biology and Health*). Available from: https://doi.org/10.1007/978-1-4419-6646-9_2.
 53. Zuo W, Jiang S, Guo Z, Feldman MW, Tuljapurkar S. Advancing front of old-age human survival. *Proc Natl Acad Sci U S A*. 2018;115(44):11209–14.
 54. Parker MG, Schön P, Lagergren M, Thorslund M. Functional ability in the elderly Swedish population from 1980 to 2005. *Eur J Ageing*. 2008;5(4):299–309.
 55. Hadley J, Waidmann T, Zuckerman S, Berenson RA. Medical spending and the health of the elderly. *Health Serv Res*. 2011;46(5):1333–61.
 56. Goldman N, Glei DA, Weinstein M. What matters most for predicting survival? A multinational population-based cohort study. *PLoS One*. 2016; 11(7):e0159273.

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Supplemental Material for

Stratification in health and survival after age 100: Evidence from Danish centenarians

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A. Comparison of survival trajectories of participants of Danish Birth Cohort Studies vs non-participants for the 1895, 1905 and 1910 cohorts

Table A1. Survival probabilities above age 100 for participants and non-participants and associated 95% confidence intervals for the 1895 cohort.

Age	Participants		No Participants	
	Survival probability	CI (95%)	Survival probability	CI (95%)
100.0	1.00	(1,1)	1.00	(1,1)
100.5	0.83	(0.78,0.88)	0.69	(0.59,0.81)
101.0	0.64	(0.58,0.71)	0.47	(0.37,0.61)
101.5	0.48	(0.42,0.56)	0.30	(0.21,0.44)
102.0	0.35	(0.29,0.42)	0.24	(0.16,0.37)
102.5	0.29	(0.24,0.36)	0.16	(0.09,0.27)
103.0	0.20	(0.15,0.26)	0.13	(0.07,0.24)
103.5	0.15	(0.11,0.21)	0.07	(0.03,0.17)
104.0	0.11	(0.08,0.16)	0.05	(0.02,0.15)
104.5	0.09	(0.06,0.14)	0.04	(0.01,0.13)
105.0	0.08	(0.05,0.13)	0.01	(0,0.1)
105.5	0.06	(0.03,0.1)	0.01	(0,0.1)
106.0	0.04	(0.02,0.08)	0.01	(0,0.1)
106.5	0.04	(0.02,0.08)		
107.0	0.03	(0.01,0.06)		
107.5	0.02	(0.01,0.06)		
108.0	0.01	(0,0.04)		
108.5	0.00	(0,0.03)		
109.0				
109.5				
110.0				

Log-rank test p-value=0.007.

Table A2. Survival probabilities above age 100 for participants and non-participants and associated 95% confidence intervals for the 1905 cohort.

Age	Participants		No Participants	
	Survival probability	CI (95%)	Survival probability	CI (95%)
100.0	1.00	(1,1)	1.00	(1,1)
100.5	0.82	(0.77,0.87)	0.77	(0.7,0.84)
101.0	0.66	(0.61,0.73)	0.59	(0.52,0.67)
101.5	0.56	(0.5,0.63)	0.43	(0.36,0.52)
102.0	0.45	(0.39,0.52)	0.37	(0.29,0.45)
102.5	0.34	(0.28,0.4)	0.30	(0.23,0.38)
103.0	0.28	(0.23,0.35)	0.23	(0.17,0.31)
103.5	0.20	(0.16,0.26)	0.16	(0.11,0.23)
104.0	0.16	(0.13,0.22)	0.12	(0.07,0.18)
104.5	0.11	(0.08,0.17)	0.10	(0.06,0.17)
105.0	0.07	(0.05,0.12)	0.07	(0.03,0.13)
105.5	0.05	(0.03,0.09)	0.04	(0.01,0.09)
106.0	0.03	(0.01,0.06)	0.03	(0.01,0.07)
106.5	0.02	(0.01,5)	0.01	(0,0.06)
107.0	0.01	(0,0.04)	0.01	(0,0.05)
107.5	0.01	(0,0.03)	0.01	(0,0.05)
108.0	0.01	(0,0.03)	0.01	(0,0.05)
108.5	0.01	(0,0.03)		
109.0	0.01	(0,0.03)		
109.5	0.01	(0,0.03)		
110.0	0.01	(0,0.03)		

Log-rank test p-value =0.13

Table A3. Survival probabilities above age 100 for participants and non-participants and associated 95% confidence intervals for the 1910 cohort.

Age	Participants		No Participants	
	Survival probability	CI (95%)	Survival probability	CI (95%)
100.0	1.00	(1,1)	1.00	(1,1)
100.5	0.88	(0.08,0.92)	0.81	(0.75,0.88)
101.0	0.72	(0.66,0.77)	0.64	(0.56,0.72)
101.5	0.55	(0.49,0.62)	0.46	(0.38,0.55)
102.0	0.41	(0.35,0.47)	0.35	(0.28,0.44)
102.5	0.32	(0.27,0.38)	0.26	(0.19,0.34)
103.0	0.27	(0.22,0.33)	0.19	(0.13,0.26)
103.5	0.22	(0.18,0.27)	0.11	(0.07,0.18)
104.0	0.27	(0.12,0.21)	0.09	(0.06,0.16)
104.5	0.13	(0.09,0.17)	0.08	(0.05,0.14)
105.0	0.08	(0.05,0.12)	0.06	(0.03,0.12)
105.5	0.04	(0.02,0.07)	0.06	(0.03,0.11)
106.0	0.04	(0.02,0.07)	0.04	(0.02,0.09)
106.5	0.03	(0.01,0.05)	0.03	(0.02,0.08)
107.0	0.03	(0.01,0.05)	0.03	(0.01,0.07)
107.5	0.02	(0.01,0.05)	0.02	(0.01,0.06)
108.0	0.01	(0,0.04)	0.01	(0.01,0.06)
108.5	0.01	(0,0.03)	0.01	(0,0.05)
109.0	0.01	(0,0.03)		
109.5	0.01	(0,0.03)		
110.0	0.01	(0,0.03)		

Log-rank test p-value =0.08

According to the p-value of the log-rank tests, the survival trajectories of the participants of the 1905 and 1910 are not statistically different. However, for the 1895 the p-value of the log-rank test indicates that there are differences between the survival trajectories of the participants of the study and non-participants. This indicates that the data for the 1895 cohort is not country representative. The survival advantage for the participants could be attributed to health selection.

Table A4. Description of participants included in the analysis and missing values per health characteristic. Cohorts 1895, 1905 and 1910.

	Cohort 1895	Cohort 1905	Cohort 1910
Total participants	207	256	273
Participants that died before turning age 100	0	-25	-1
Participants in the study after removing those that did not turn age 100	207	231	272
<hr/>			
Missing values*			
MMSE (%)	64 (31)	56 (24)	84 (31)
Chair Stand (%)	NA	56 (24)	82 (30)
Self-Rated Health (%)	30 (14)	51 (22)	76 (28)
Katz's disability index (%)	0	2 (1)	2 (1)
<hr/>			
Proxy respondent	30	51	76
Individuals with missing values in at least one category after the creation of the "no tested" category	37	36	49
<hr/>			
Participants included in the analysis	170	195	223

* There are individuals that exhibit missing values in more than one category.

For example, for the 1905 cohort we excluded 36 individuals after creating the “no tested” category. 5 of those 36 had missing values in MMSE and 31 of the 36 had missing values in chair stand.

2 individuals with missing values in the disability index also have missing values in chair stand.

B. Summary statistics and survival probabilities for participants of the surveys of the 1905 and 1910 cohorts

Table A5. Summary statistics by gender for the cohorts 1905 and 1910.

	Males			Females			Sex differences	
	1905 cohort	1910 cohort	p-value*	1905 cohort	1910 cohort	p-value*	1905 cohort	1910 cohort
Participants included in the analysis, n								
	33	51		162	172			
Mean age of interview (SD)	99.8 (0.3)	100.3 (0.4)	<0.001	99.8 (0.28)	100.35 (0.3)	<0.001	0.410	0.532
Mean age at death (SD)	102.4 (1.8)	102.0 (1.6)	0.294	102.3 (1.9)	102.4 (1.9)	0.506	0.775	0.142
Proxy, n (%)			1.000			0.117	1.000	0.519
	Yes	4 (12.1)	7 (13.7)		20 (12.3)	32 (18.6)		
	No	29 (87.9)	44 (86.3)		142 (87.7)	140 (81.1)		
MMSE, n (%)			0.957			0.244	0.284	0.341
	Score 0-17	5 (15.2)	9 (17.6)		44 (27.2)	38 (22.1)		
	Score 18-23	8 (24.2)	10 (19.6)		47 (29.0)	43 (25.0)		
	Score 24-30	16 (48.5)	25 (49.0)		51 (31.5)	59 (34.3)		
	No tested	4 (12.1)	7 (13.7)		20 (12.3)	32 (18.6)		
Self-rated health, n (%)			0.647			0.177	0.683	0.536
	No tested	4 (12.1)	7 (13.7)		20 (12.3)	32 (18.6)		
	Poor	1 (3.0)	3 (5.9)		13 (8.0)	10 (15.8)		
	Acceptable	8 (24.2)	14 (27.5)		45 (27.8)	34 (19.8)		
	Good/Excellent	20 (60.6)	27 (51.9)		87 (51.9)	96 (55.8)		
Chair Stand, n (%)			0.705			0.531	0.664	0.641
	Cannot stand	10 (30.3)	14 (27.5)		50 (30.9)	55 (32.0)		
	With use of arms	16 (48.5)	22 (43.1)		67 (41.4)	78 (45.3)		
	Without use of arms	7 (21.2)	15 (29.4)		45 (27.8)	39 (22.7)		
Katz's disability index, n (%)			0.785			0.054	0.489	0.058
	Disabled	13 (39.4)	17 (33.3)		49 (30.2)	64 (37.2)		
	Moderately	10 (30.3)	15 (29.4)		61 (37.7)	72 (41.9)		
	Not disabled	10 (30.3)	19 (37.3)		52 (32.1)	36 (20.9)		

*Test to determine equal means between the populations analysed

MMSE: Mini-Mental State Examination

Note: We only included individuals that do not present missing values in any of the characteristics observed.

Table A6. Comparison of the class membership probabilities of the robust class for the 1905 and 1910 cohorts.

Dimension	Score	Cohort	
		1905	1910
Mini-mental state examination	24-30	0.46	0.63
	18-23	0.33	0.27
	0-17	0.21	0.1
	Not tested	0	0
Self-rated health	Good or excellent	0.73	0.78
	Acceptable	0.27	0.22
	Poor	0	0
	Not Tested	0	0
Chair stand test	Without arms	0.39	0.56
	With arms	0.59	0.44
	Cannot stand	0.02	0
Katz's disability score	Not disabled	0.52	0.61
	Moderate disabled	0.48	0.39
	Disabled	0	0

Table A7. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1905 cohort.

Age	Robust		Intermediate		Frail	
	Survival probability	CI (95%)	Survival probability	CI (95%)	Survival probability	CI (95%)
100.0	1.00	(1,1)	1.00	(1,1)	1.00	(1,1)
100.5	0.92	(0.87,0.97)	0.70	(0.55,0.89)	0.69	(0.54,0.87)
101.0	0.80	(0.73,0.87)	0.50	(0.35,0.72)	0.47	(0.32,0.68)
101.5	0.71	(0.64,0.8)	0.43	(0.29,0.65)	0.28	(0.16,0.49)
102.0	0.60	(0.52,0.69)	0.33	(0.2,0.55)	0.16	(0.07,0.35)
102.5	0.48	(0.4,0.57)	0.23	(0.12,0.45)	0.09	(0.03,0.28)
103.0	0.41	(0.34,0.51)	0.13	(0.05,0.33)	0.06	(0.02,0.24)
103.5	0.31	(0.24,0.4)	0.13	(0.05,0.33)		
104.0	0.25	(0.18,0.33)	0.13	(0.05,0.33)		
104.5	0.18	(0.13,0.26)	0.07	(0.02,0.25)		
105.0	0.12	(0.08,0.19)	0.07	(0.02,0.25)		
105.5	0.08	(0.04,0.14)	0.07	(0.02,0.25)		
106.0	0.05	(0.03,0.11)				
106.5	0.04	(0.02,0.09)				
107.0	0.02	(0.01,0.07)				
107.5	0.02	(0,0.06)				
108.0	0.02	(0,0.06)				
108.5	0.02	(0,0.06)				
109.0	0.01	(0,0.05)				
109.5	0.01	(0,0.05)				
110.0	0.01	(0,0.05)				

Log-rank test p-value<0.001

This p-value indicates that the null hypothesis should be rejected, thus the survival curves are statistically different from each other.

Table A8. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1910 cohort. Health classes were obtained from the Latent Class Analysis.

Age	Robust		Intermediate		Frail	
	Survival probability	CI (95%)	Survival probability	CI (95%)	Survival probability	CI (95%)
100.0	1.00	(1,1)	1.00	(1,1)	1.00	(1,1)
100.5	0.96	(0.91,1)	0.72	(0.59,0.87)	0.87	(0.81,0.94)
101.0	0.87	(0.8,0.94)	0.44	(0.31,0.62)	0.70	(0.62,0.8)
101.5	0.72	(0.64,0.82)	0.26	(0.15,0.44)	0.56	(0.47,0.67)
102.0	0.59	(0.5,0.7)	0.18	(0.09,0.35)	0.44	(0.35,0.55)
102.5	0.53	(0.44,0.65)	0.08	(0.03,0.23)	0.32	(0.24,0.43)
103.0	0.47	(0.37,0.58)	0.08	(0.03,0.23)	0.24	(0.17,0.35)
103.5	0.40	(0.31,0.52)	0.05	(0.01,0.2)	0.19	(0.13,0.29)
104.0	0.30	(0.22,0.41)	0.03	(0,0.18)	0.14	(0.08,0.23)
104.5	0.23	(0.16,0.34)	0.03	(0,0.18)	0.11	(0.06,0.19)
105.0	0.17	(0.11,0.26)	0.03	(0,0.18)	0.04	(0.02,0.11)
105.5	0.09	(0.05,0.17)	0.03	(0,0.18)	0.01	(0,0.07)
106.0	0.08	(0.04,0.16)	0.03	(0,0.18)	0.01	(0,0.07)
106.5	0.06	(0.02,0.13)	0.03	(0,0.18)		
107.0	0.06	(0.02,0.13)	0.03	(0,0.18)		
107.5	0.04	(0.02,0.12)	0.03	(0,0.18)		
108.0	0.03	(0.01,0.1)	0.03	(0,0.18)		
108.5	0.02	(0.01,0.09)				
109.0						
109.5						
110.0						

Log-rank test p-value <0.001

This p-value indicates that the null hypothesis should be rejected, thus the survival curves are statistically different from each other.

C. Analysis of the 1895 cohort

Table A9. Summary statistics by gender for the cohort 1895.

	1895 cohort		
	Males	Females	p-value*
Participants included in the analysis, n	40	130	
Mean age of interview (SD)	100.1 (0.04)	100.1 (0.06)	<0.001
Age of death	2.2 (1.58)	2.1 (1.87)	0.603
Proxy, n (%)			0.624
Yes	6 (15.0)	24 (18.5)	
No	34 (85.0)	106 (81.5)	
MMSE, n (%)			0.742
Score 0-17	9 (22.5)	36 (27.7)	
Score 18-23	14 (35.0)	38 (29.2)	
Score 24-30	11 (27.5)	32 (24.6)	
No tested	6 (15.0)	24 (18.5)	
Self-rated health , n (%)			0.183
No tested	3 (7.5)	20 (15.4)	
Poor	1 (2.5)	14 (10.8)	
Acceptable	6 (15.0)	15 (11.5)	
Good/Excellent	30 (15.0)	81 (61.3)	
Katz's disability index, n (%)			<0.001
Disabled	9 (22.5)	55 (42.3)	
Moderately	19 (47.5)	60 (46.2)	
Not disabled	12 (30.0)	15 (11.5)	

*Test to determine equal means between the populations analysed

MMSE: Mini-Mental State Examination

Note: We only included individuals that do not present missing values in any of the characteristics observed.

Figure A1. Class membership probabilities by health class for the 1895 cohort, both sexes.

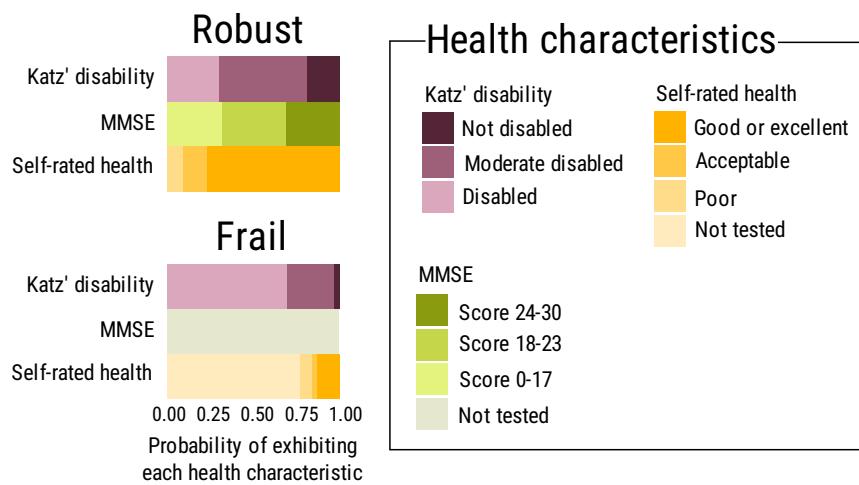


Figure A2. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the cohort 1895, both sexes.

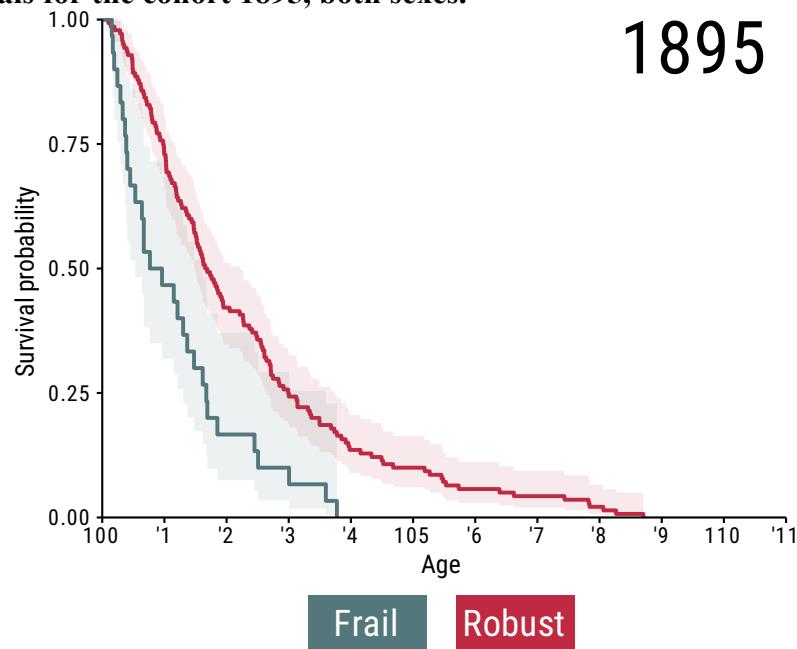


Table A10. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1895 cohort.

Age	Robust		Frail	
	Survival probability	CI (95%)	Survival probability	CI (95%)
100.0	1.00	(1,1)	1.00	(1,1)
100.5	0.89	(0.84,0.95)	0.67	(0.52,0.86)
101.0	0.73	(0.66,0.81)	0.47	(0.32,0.68)
101.5	0.57	(0.50,0.66)	0.30	(0.17,0.52)
102.0	0.42	(0.35,0.51)	0.17	(0.07,0.37)
102.5	0.36	(0.29,0.45)	0.13	(0.05,0.33)
103.0	0.24	(0.18,0.33)	0.10	(0.03,0.29)
103.5	0.19	(0.13,0.26)	0.07	(0.02,0.25)
104.0	0.14	(0.09,0.21)		
104.5	0.11	(0.07,0.18)		
105.0	0.10	(0.06,0.16)		
105.5	0.07	(0.04,0.13)		
106.0	0.06	(0.03,0.11)		
106.5	0.05	(0.02,0.1)		
107.0	0.04	(0.02,0.09)		
107.5	0.04	(0.02,0.08)		
108.0	0.02	(0.01,0.07)		
108.5	0.01	(0,0.05)		
109.0				
109.5				
110.0				

Log-rank test p-value<0.001

This p-value indicates that the null hypothesis should be rejected, which indicates that the survival curves are statistically different from each other.

D. Latent Class Analysis (LCA) model details

Suppose a latent class model with C classes to be estimated with M categorical variables and a covariate x . Let $Y_i = (Y_{i1}, \dots, Y_{iM})$ be the vector of individual's response to the M variables where $Y_{im} = 1, 2, \dots, r_m$ indicates the individual characteristic for a certain variable. Let $c_i = 1, 2, \dots, C$ be the latent class membership of the individual to the class; let $I(y_{im} = k)$ be the indicator function that is 1 if the response of individual i to the variable m^{th} variable y is equal to k and 0 otherwise; let λ_c be the probability of membership in each latent class and let $\rho_{mk|c}$ be the item-response probability (variable m , category k) conditional on the latent class membership (c). The latent class model is expressed as follows:

$$P(Y = y|x_i) = \sum_{c=1}^C \lambda_c(x_i) \prod_{m=1}^M \prod_{k=1}^{r_m} \rho_{mk|c} I(y_{im} = k)$$

where $\lambda_c(x_i) = P(C_i = c|x_i)$ is a standard baseline-category for the multinomial logistic model. In the case of one covariate, λ is expressed as follows:

$$\lambda_c(x_i) = P(C_i = c|x_i) = \frac{\exp\{\beta_{0c} + x_i \beta_{1c}\}}{1 + \sum_{j=1}^C \exp\{\beta_{0j} + x_i \beta_{1j}\}}$$

for $c = 1, \dots, C - 1$, where C is the reference class in the logistic regression. Finally, the log-odds of an individual falling into latent class c relative to the reference class C , giving x_i as the value for the covariate, is:

$$\log\left(\frac{\lambda_{c|c}(x_i)}{\lambda_{C|c}(x_i)}\right) = \beta_{0c|c} + \beta_{1c|c}x_i$$

Table A11. Goodness of fit for the Latent Class Analysis varying the number of clusters. Cohorts 1895, 1905, 1910.

Number of clusters	Akaike information criterion			Bayesian information criterion		
	1895 cohort	1905 cohort	1910 cohort	1895 cohort	1905 cohort	1910 cohort
2	1,072.38	1,561.93	1,680.75	1,128.83	1,652.72	1,755.71
3	1,176.21	1,541.43	1,601.32	1,264.01	1,633.94	1,717.17
4	1,068.78	1,640.55	1,655.70	1,187.94	1,791.11	1,812.43
5	1,163.47	1,899.22	2,003.25	1,313.99	2,089.05	2,200.87
6	1,183.47	1,942.05	1,761.15	1,365.35	2,171.16	1,999.65

E. Sensitivity analysis – considering only females in the LCA

Figure A3. Class membership probabilities by health class for the 1905 and 1910 cohorts considering only females.

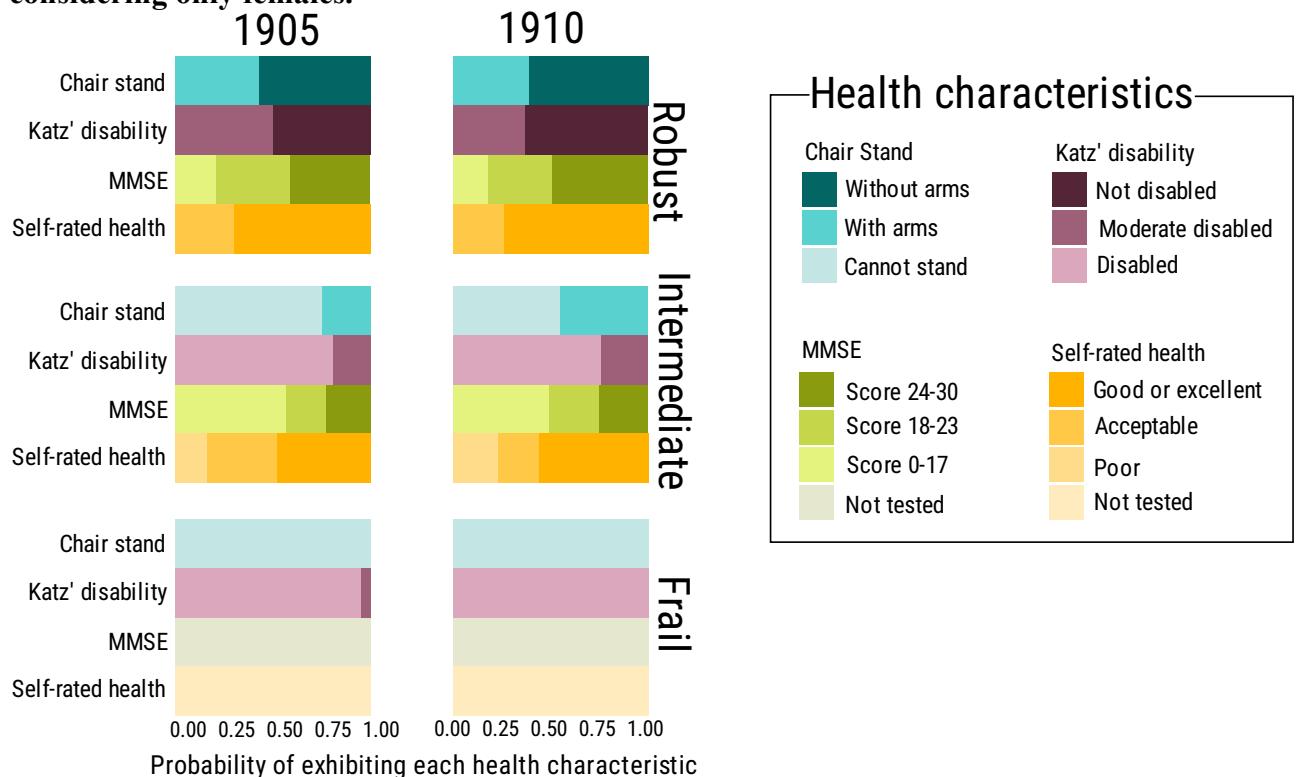


Figure A4. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1905 and 1910 cohorts considering only females in the Latent Class Analysis.

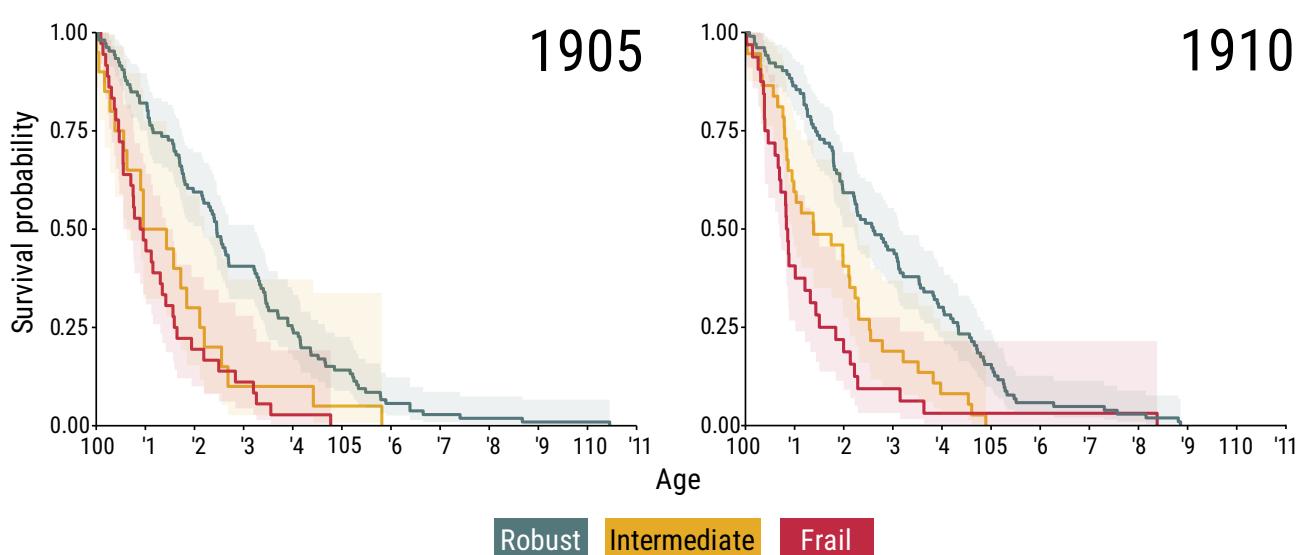


Table A12. Area under the curve by percentile for the 1905 and 1910 cohorts considering only females in the analysis.

Percentile	1905 Cohort		1910 Cohort	
	Age	AUC	Age	AUC
95th	105.61	0.64	105.72	0.65
96th	105.95	0.68	106.08	0.65
97th	106.26	0.68	106.39	0.64
98th	106.95	0.68	107.09	0.64
99th	107.94	0.68	108.15	0.62

Note: The AUC ranges from 0 to 1; a higher AUC implies a better prediction. Medford et al. define the frontier of survival as the 95th percentile of the centenarian age-at-death distribution. We included upper percentiles as a robustness check.

F. Sensitivity analysis – including smoking behaviour

Figure A5. Class membership probabilities by health dimension for the 1905 and 1910 cohorts including Smoking in the Latent Class Analysis, both sexes.

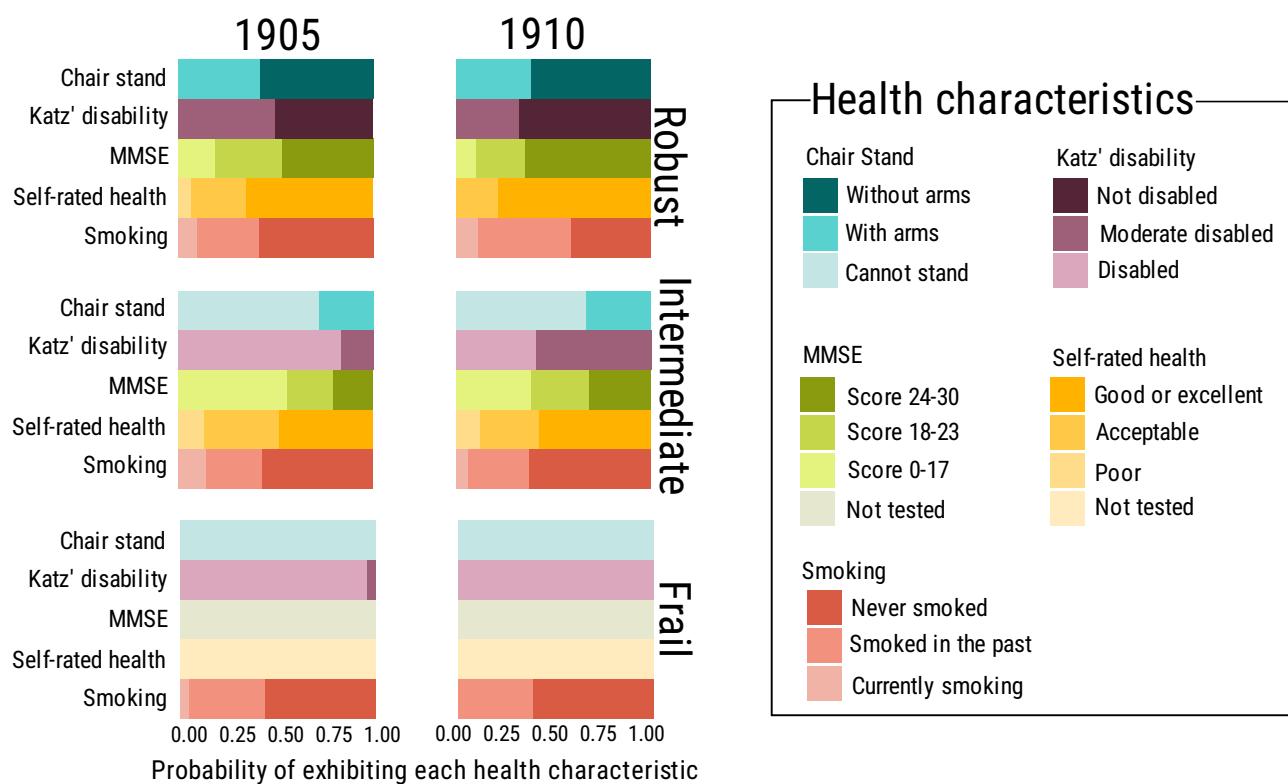


Figure A6. Survival probabilities above age 100 by health class and associated 95% confidence intervals for the 1905 and 1910 cohorts including Smoking in the Latent Class Analysis, both sexes.

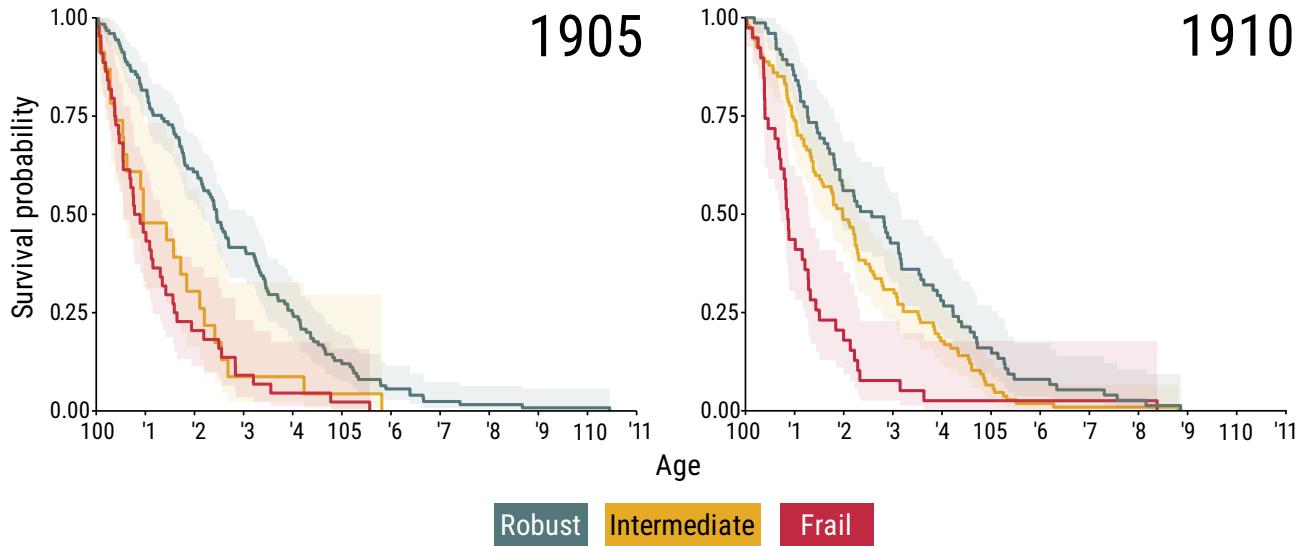


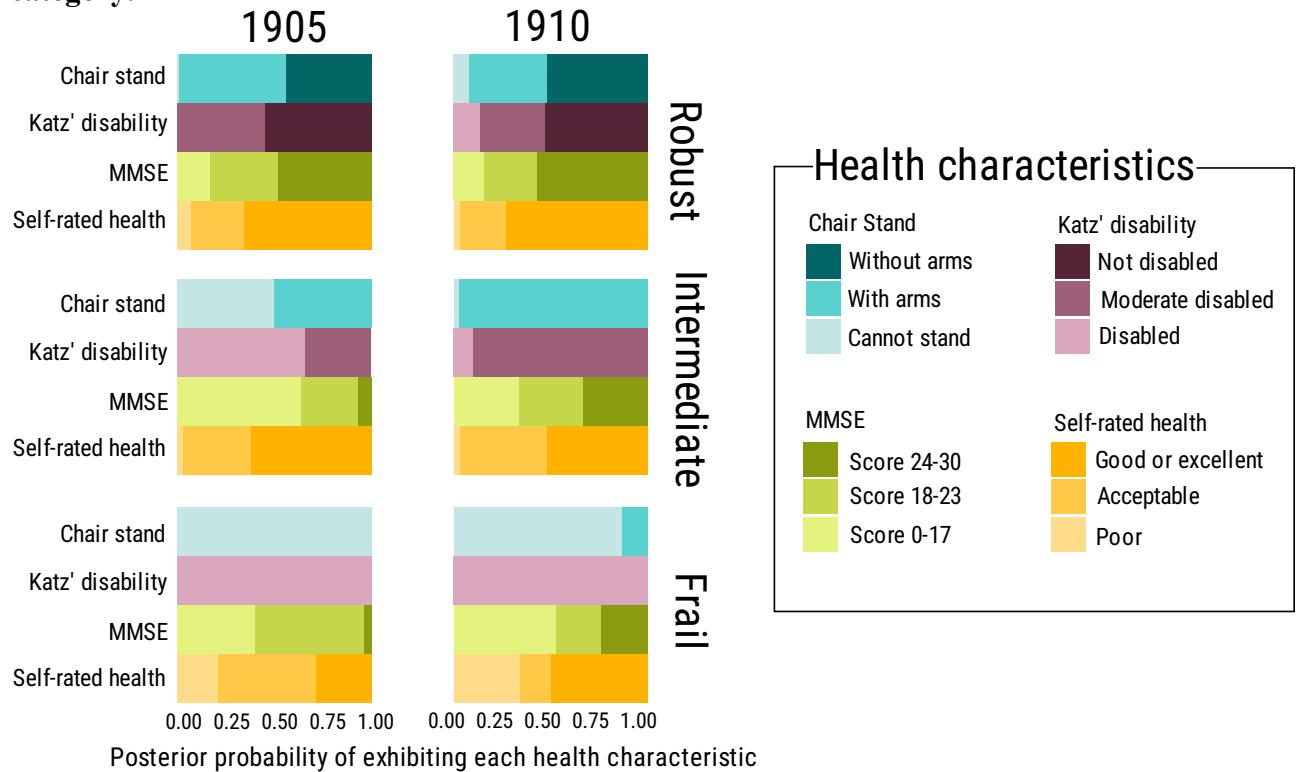
Table A13. Area under the curve by percentile for the 1905 and 1910 cohorts including Smoking in the analysis.

Percentile	1905 Cohort		1910 Cohort	
	Age*	AUC	Age*	AUC
95th	105.61	0.65	105.72	0.68
96th	105.95	0.68	106.08	0.68
97th	106.26	0.68	106.39	0.70
98th	106.95	0.68	107.09	0.70
99th	107.94	0.68	108.15	0.62

Note: The AUC ranges from 0 to 1; a higher AUC implies a better prediction. Medford et al. define the frontier of survival as the 95th percentile of the centenarian age-at-death distribution. We included upper percentiles as a robustness check.

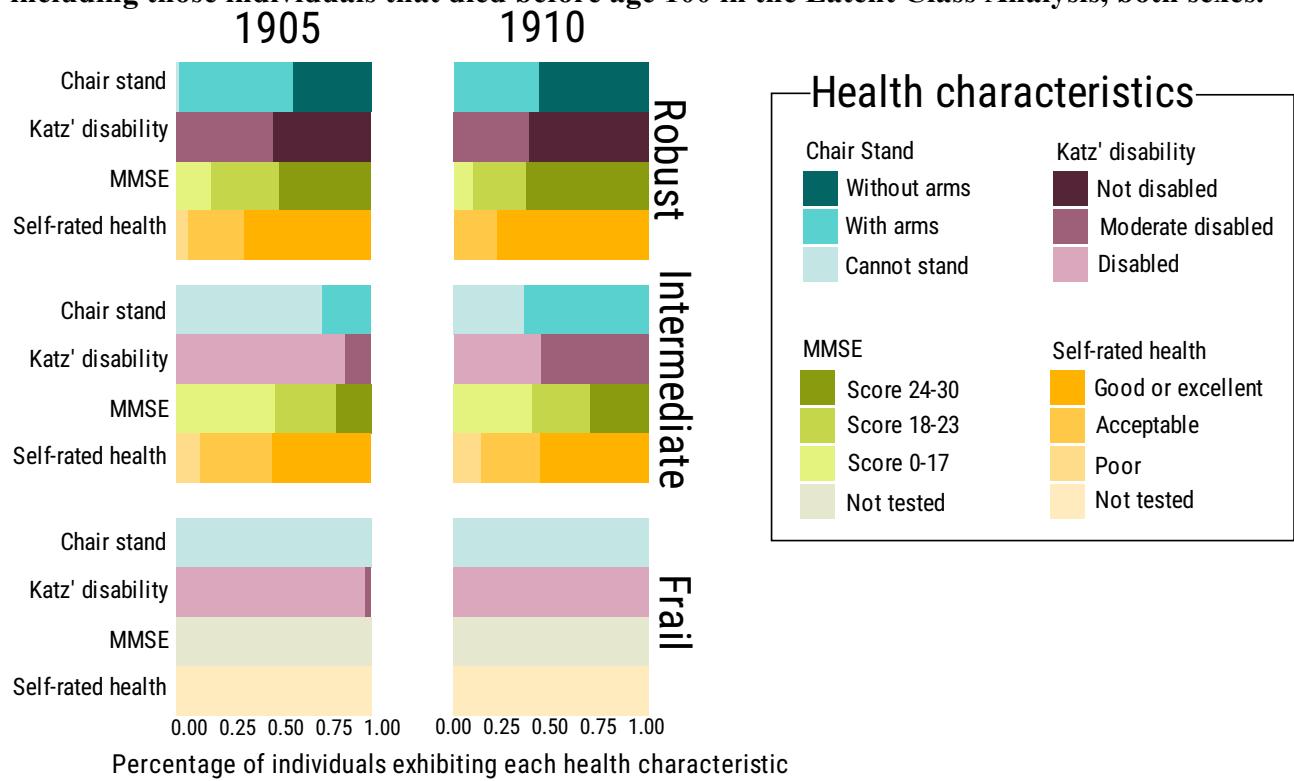
G. Sensitivity analysis – removing the missing data

Figure A7. Class membership probabilities by health dimension for the 1905 and 1910 cohorts including only individuals with complete observations, without the creation of “no tested” category.



H. Sensitivity analysis – including those that died before age 100

Figure A8. Class membership probabilities by health dimension for the 1905 and 1910 cohorts including those individuals that died before age 100 in the Latent Class Analysis, both sexes.



I. Calculation of AUC using only Chair Stand and MMSE as predictors of extreme survival as in Thinggaard et al (2016)

Thinggaard et al (2016) found that Chair Stand combined with MMSE are good predictors of survival among Danish nonagenarians in the 1905 cohort. Specifically, those exhibiting MMSE greater than 28 and being able to stand up from their chair without hands are at greater chance to survive to age 100. Here we tested the predictive power of these characteristics for centenarians in the 1905 and 1910 cohorts. However, we softened the “robust threshold” to those that are able to stand with and without hands and have MMSE>24 given that only few individuals appear to have values of MMSE>28 and are able to stand from chair without hands. By using the softened version of the robust threshold, we are able to obtain similar predictive power (depicted by the AUC) to the one depicted in our LCA categorization. Both approaches are useful to predict the length of life in centenarians.

Table A14. Area under the curve by percentile for the 1905 and 1910 cohorts using Chair Stand (able to stand with and without hands) and MMSE (>24)

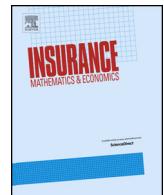
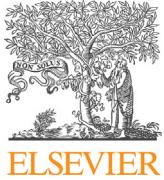
Percentile	1905 Cohort		1910 Cohort	
	Age	AUC	Age	AUC
95th	105.61	0.60	105.72	0.66
96th	105.95	0.73	106.08	0.66
97th	106.26	0.73	106.39	0.65
98th	106.95	0.71	107.09	0.65
99th	107.94	0.72	108.15	0.61

Note: The AUC ranges from 0 to 1; a higher AUC implies a better prediction. Medford et al. define the frontier of survival as the 95th percentile of the centenarian age-at-death distribution. We included upper percentiles as a robustness check. The AUC in this table was calculated using only Chair Stand and MMSE as in Thinggaard et al, (2016)

Linking retirement age to life expectancy does not lessen the demographic implications of unequal lifespans

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Linking retirement age to life expectancy does not lessen the demographic implications of unequal lifespans



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ABSTRACT

The fact that individuals are living longer and thus spending more time in retirement challenges the sustainability of pension systems. This has forced policy makers to rethink the design of pension plans to mitigate the burden of increased longevity. Countries such as the Netherlands, Estonia, Denmark and Finland have implemented reforms that link retirement age to changes in life expectancy. However, the demographic and financial implications of such linkages are not well understood. This study analyses the Danish case, using high-quality data from population registers during the period 1985–2016. We identify trends in demographic and actuarial measures after retirement by sex and socio-economic group. We also introduce a new decomposition method to disentangle the demographic sources of socio-economic disparities in pension costs per year of expected benefits. We reach two main results. First, linking retirement age to life expectancy increases uncertainty about length of life after retirement, with the financial cost becoming more sensitive to changes in mortality. Second, socio-economic disparities in lifespans persist regardless of the age at which individuals retire. Males from lower socio-economic groups are at a greater disadvantage, because they spend fewer years in retirement, pay higher pension costs per year of expected benefits and are exposed to higher longevity risk than the rest of the population. This disadvantageous setting is magnified when retirement age is linked to life expectancy.

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1. Introduction

Recently observed survival trajectories for individuals posit unprecedented challenges to existing pension systems. First of all, life expectancy at birth has increased over time (Medford, 2017; Oeppen and Vaupel, 2002; Pascariu et al., 2018). This measure has gone up chiefly because of a reduction in the risk of dying at young ages (Burger et al., 2012). As a consequence, a high proportion of individuals from recent cohorts survive to retirement ages. Secondly, the risk of dying during post-retirement ages has also trended downwards (Rau et al., 2008; Zuo et al., 2018), such that individuals from more recent cohorts spend a greater number of years in retirement in comparison to those from previous ones (Sanderson and Scherbov, 2010, 2017). As more people survive to retirement and live for longer once retired, there is an increased need to reform pension systems in many countries (Chomik and Whitehouse, 2010).

To ensure sustainability in their pension systems, many countries including Denmark,¹ Estonia, Finland, Italy, the Netherlands, Portugal and the Slovak Republic have passed reforms that modify the way pension systems operate (OECD, 2017). A common feature among these countries is that their pension systems will gradually take into account increases in life expectancy, either by modifying retirement age or by adjusting payouts (OECD, 2018b; Whitehouse, 2007). In Denmark, the statutory retirement age will gradually increase, targeting the age at which remaining life expectancy is 14.5 years² (hereafter referred to as *target retirement age*, see Danish Ministry of Economic Affairs and Interior (2017, 2018) and OECD (2015) for more details). Rather than focusing on the number of

¹ The outcome of other reforms at the beginning of the 1990s was the strengthening of actuarial fairness in the Danish pension system by shifting people into mandatory defined contribution schemes (Andersen, 2015).

² The way statutory retirement age will be determined in the coming years will be more complicated than merely matching it to the target retirement age (see Section 2 for further details). While it is possible that the statutory retirement age will not exactly match the target retirement age right away, the latter represents a long-term goal. The target retirement age serves as a reference of the future development of the statutory retirement age. Hence, its study is the starting point in understanding the implications of linking retirement ages to life expectancy.

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years already *lived* (fixed chronological age), the target retirement age is based on the number of years a person is *expected* to spend in retirement. This scheme implies that: (i) regardless of the year in which they retire, Danish retirees can expect to receive pension payments for an average period of 14.5 years and (ii) the number of years gained in life expectancy will be spent in work rather than in retirement.

In increasing retirement ages (in this case, by linking them to life expectancy), the demographic circumstances in which individuals can retire change. Pension payments are now conditioned upon surviving to an older age, at which the risk of dying is higher than at younger ages (Rau et al., 2008). Additionally, not all individuals experience the same age-specific survival probabilities at retirement; it has been shown that the probabilities differ considerably with respect to different socio-economic groups (Cairns et al., 2019; Sasson, 2016; Villegas and Haberman, 2014). Increasing retirement age might have unanticipated consequences for lower socio-economic groups, as they exhibit lower life expectancies and higher lifespan inequality³ (van Raalte et al., 2018). Individuals from lower socio-economic groups might be more affected by changes in retirement age than those with higher survival chances (Chetty et al., 2016). An analysis of the demographic circumstances in which individuals from different socio-economic groups retire is necessary to understand how increasing retirement age affects them, but also to determine the extent of socio-economic disparities.

From the perspective of the pension provider, large socio-economic differences in longevity also represent important implications in the financial sustainability of pension schemes (Villegas and Haberman, 2014). Given that pension providers and insurance companies price their annuities, calculate reserves and evaluate their exposure to longevity risks using mortality rates for national populations, neglecting socio-economic disparities in longevity could result in inadequate funding for pension liabilities (Coughlan et al., 2011). Further, socio-economic differentials in longevity could also distort redistribution properties of both defined benefit and defined contribution pension schemes (Brown, 2002, 2003; Liebman, 2002). Depending on the type of pension scheme and on the extent of such socio-economic disparities, undesirable transfers of wealth from lower socio-economic groups (with shorter lifespans) to higher socio-economic groups (with longer lifespans) could arise (Ayuso et al., 2017; Holzmann et al., 2019). Socio-economic differentials in longevity and their effect on pension schemes could be exacerbated when increasing retirement ages.

The aim of this study is to determine the implications of linking retirement age to life expectancy in terms of demographic inequalities and their influence on the financial cost of pensions across socio-economic groups. We use the Danish pension system to illustrate these issues. Specifically, we compare the previous demographic setting at retirement (in which individuals retire at a fixed chronological age) to the demographic setting, based on the Danish pension reform (Danish Ministry of Economic Affairs and Interior, 2018). A strategy to compare these two scenarios could be to forecast age-specific mortality rates into the future and determine the demographic differences between the target and current pension schemes (assuming that the current pension scheme will also be maintained into the future). However, this set-up implies

additional assumptions about the future path of age-specific mortality rates, and our results would hinge to a great extent on the model used to forecast. Instead, our comparisons are made retrospectively; we analyse the current and target pension schemes with historical data (during the period 1985–2016). The advantage of this strategy is that we are able to describe the current demographic scenario at retirement and compare it with the target setting by using observed data without making any additional assumptions about the future path of age-specific mortality rates. We inspect trends over time in demographic (life expectancy and lifespan inequality) and actuarial measures (the cost of life annuity at retirement and its entropy). Furthermore, we introduce a new decomposition method to determine the sources of the gap in the entropy of a life annuity between socio-economic groups.

In summary, we find that socio-economic disparities in longevity persist, regardless of the age at which individuals retire. Males from lower socio-economic groups are at a greater disadvantage because they have lower probability of surviving to retirement, spend fewer years in retirement, pay higher pension costs per year of expected benefits and are exposed to higher longevity risk. While linking retirement age to life expectancy might contribute to ensuring the sustainability of national pension systems, this policy introduces higher inequality and positions males from lower socio-economic groups in a more disadvantageous retirement setting. A key contribution of this paper is in bringing together a demographic and an actuarial perspective on socio-economic differences in pension outcomes, by using high-quality micro data. Our findings serve as a reference for the demographic and actuarial implications that might arise in other countries undergoing similar pension reforms to Denmark, in which retirement age will be linked to developments in life expectancy (OECD, 2015, 2018b).

We have organised the remainder of the paper as follows. First, we describe the components of the Danish pension system in Section 2. Then, in Section 3, we describe the data and summarise the methodology used in the paper. In Section 4, we show the results. Lastly, we conclude with a discussion of the implications of our results for the Danish pension system in Section 5.

2. The Danish pension system

The main objective of a pension system is to provide income security at old ages (OECD, 2017) while ensuring financial sustainability, actuarial fairness, limited inter/intra-generational redistribution, and adequate replacement rates (OECD, 2019b). To achieve these objectives, the Danish pension system follows a multi-pillar system that combines public and labour market pensions (World Bank, 1994). The first pillar consists of a basic public old age pension (OAP) financed on a Pay-As-You-Go (PAYG) basis combined with a public mandatory and contributory pension linked to earnings (the Labour Market Supplementary Pension Scheme, ATP, see Andersen (2015) for further details). Old age pension benefits consist of a universal basic amount (independent of the individual's labour market history) and a supplement, which is means-tested against all other pension- and labour income. Finally, pillar one contains a number of early retirement options; early retirement pay (in Danish: efterløn), disability benefits, and senior pension (OECD, 2018a).

The second pillar consists of defined contribution (DC) labour market pension schemes, which are fully funded. They cover more than 90% of wage earners between the ages 25 and 59 (Balter et al., 2020; OECD, 2015). Moreover, the second pillar contains tax-financed earnings-related civil servant pensions (constituted as defined benefit (DB) schemes) which are currently being phased out (Danish Ministry of Economic Affairs and Interior, 2018). The third pillar consists of individual, private pension savings schemes independent of employment conditions. Unlike pillar two, individ-

³ Lifespan inequality is an indicator of uncertainty about the number of years a person will spend in retirement. Haberman et al. (2011) showed that, in a context of low interest rates, high lifespan inequality indicates that the financial cost of a pension (actuarially represented by life annuity factors) is highly sensitive to fluctuations in mortality rates. Thus, linking retirement age to life expectancy could increase the exposure to longevity risk in particular for those socio-economic groups that exhibit a higher degree of lifespan inequality.

uals make their own choices about the size of contributions and composition of benefits in pillar three.⁴

The eligibility age for OAP has significantly changed over time. Until 2004, the OAP age was 67 and thereafter it was reduced to 65. The main rationale for doing so was to substitute old age retirement for early retirement among 65 and 66 year old's and thereby save on public finances, because the early retirement benefit entitlement exceeded the OAP benefit, (Bingley et al., 2020). Starting from 2019, the OAP age was increased again with 0.5 year steps and from 2025, the OAP (and early retirement) age will be raised each 5th year depending on increases in life expectancy.⁵ Moreover, since 1999, older workers have had the option to postpone OAP retirement by up to 10 years resulting in an actuarial adjustment (OECD, 2015).

The Danish pension system effectively prevents old age poverty and ensures reasonable replacement rates for most pensioners (Andersen, 2015). In pillar 1, redistribution and actuarial fairness objectives are achieved through floors and ceilings applied to contributions, benefits and accrual rates (Barr and Diamond, 2006). In pillars two and three, redistribution and inter/intra-generational fairness are unlikely to constitute an issue, as each individual is responsible for their contributions and benefits (OECD, 2018b).⁶ However, inequality in longevity between socio-economic groups potentially affects the pension objectives of the pension system. For instance, to the extent that the system is designed to be progressive, and intended to re-distribute income from rich to poor, longevity differences might reduce or even reverse the direction of redistribution (Sánchez-Romero et al., 2020). These issues are further discussed in Section 5.

3. Data and methods

3.1. Data

We use the newly developed affluence measure by Cairns et al. (2019), based on individuals' income and wealth, to divide the population into five socio-economic groups (quintiles) at each age and each point in time. This measure is an attractive choice, given that income and wealth are highly correlated with mortality rates and health outcomes (Duncan et al., 2002). The affluence index $A(j, x, y)$ for individual j aged x in year y is defined as

$$A(j, x, y) = W(j, x - 1, y - 1) + K \cdot I(j, x - 1, y - 1), \quad (1)$$

where $W(j, x - 1, y - 1)$ is the wealth for individual j at age $x - 1$ in the year $y - 1$, $I(j, x - 1, y - 1)$ is the income of individual j at age $x - 1$ in year $y - 1$, and K is the capitalisation factor (approximately a value of an annuity factor calculated at retirement age). Cairns et al. (2019) show that the allocation procedure is robust to different choices of K in the interval from 10 to 20 and argues that $K = 15$ is a reasonable choice. Individuals' incomes are measured as the gross annual income which includes wage income, unemployment benefits, social assistance and pension income. Net wealth is defined as total assets minus total liabilities. The assets include real property, bank deposits, stocks, bonds, and cash holdings. Liabilities include all types of debt to private companies and the government. For married couples, wealth is assigned by 50% to each spouse.

⁴ Individual pension schemes can be set up as DC plans with banks, insurance companies or pension funds (OECD, 2015).

⁵ The indexation will be done every 5th year with 15 years notice, conditional on the life-expectancy of 60 year-olds increasing by at least 0.6 years over the notice period (Danish Ministry of Economic Affairs and Interior, 2018).

⁶ These objectives might be distorted in DC schemes with pension guarantees, see Balter et al. (2020).

Individuals are ranked according to their affluence and thereafter the total population is divided into five equally sized socio-economic groups (quintiles). The five different groups are denoted group 1 (G1) to group 5 (G5), with G5 being the most affluent group with the highest combination of wealth and income. We refer to G1, G2,...,G5 as the low, low-middle, middle, middle-high, and high affluence group, respectively. A benefit of the affluence measure is that it is possible for individuals to have either high wealth or high income independently, or both. If, for example, only income is used, individuals with a low income but high wealth would wrongly be assigned to a low affluence group. Thus, by using both variables, it is possible to more effectively distinguish survival trajectories between different socio-economic groups. This is seen as the capacity of the affluence index to capture heterogeneity in longevity (Cairns et al., 2019).

As in Cairns et al. (2019), individuals can move between socio-economic groups but are locked into their assigned socio-economic group at age 67. This procedure avoids compositional effects between socio-economic groups and allows for the calculation of demographic and actuarial measures, such as life expectancies and annuity prices. Kallestrup-Lamb et al. (2020) performed a sensitivity analysis of the affluence index and the movement between socio-economic groups. They show that, every year since 1985, only 6.1% of females have moved up by one group and 5.7% have moved down by one socio-economic group. The percentage of individuals moving up or down by more than one group is negligible (less than 2%).

3.2. Methods

Our analysis is based on two different retirement ages: the current (c) and the target (t) retirement age. The current retirement age was operational during the whole study period. The target retirement age is in line with the Danish pension reform of 2007 (Danish Ministry of Economic Affairs and Interior, 2018) and it implies that remaining life expectancy after retirement should be constant at 14.5 years. Thus, t is the age that solves the equation $e(t) = 14.5$, where $e(t)$ denotes remaining life expectancy at age t . Both ages are calculated based on the demographic regime of the total Danish population. We calculated them under a period life table perspective: every year from 1985 to 2016.

To determine the exact age t , we smooth annual death rates over the age axis by sampling 100,000 individual lifetimes from an exponential distribution with piece-wise constant rate (Willekens, 2009). We performed this procedure for every single year from 1985 to 2016. The results obtained are equivalent to period life tables calculated directly from the observed death rates (Preston et al., 2000). However, with this procedure, we are able to obtain continuous estimates of the force of mortality. Another common approach to smoothing death rates is to use splines (Camarda et al., 2012), which serves as an interpolation procedure of lifetable outcomes. We decided to simulate individual lifespans (Willekens, 2009) instead of using splines given that the calculation of demographic and actuarial measures arises naturally. For example, the calculation of remaining life expectancy at a specific age (e.g., 71.57) could be easily done with our approach by simply taking the mean of all the lifespans above that age. Conversely, the calculation of the same remaining life expectancy with splines will result in an interpolation between life expectancy values at ages 71 and 72, retrieved from the lifetable.

3.2.1. Demographic measures

The demographic indicators used in this analysis are life expectancy and lifetable entropy. Life expectancy measures the average length of life after retirement (Preston et al., 2000) and it is defined as

$$e(x) \equiv \frac{\int_x^\infty l(y)dy}{l(x)}, \quad (2)$$

where $l(x)$ is the survivorship function at age x .

Lifetable entropy measures lifespan inequality after retirement (Aburto et al., 2019; Keyfitz and Caswell, 2005). It is defined as

$$H(x) \equiv \frac{\int_x^\infty e(y)d(y)dy}{e(x)} = \frac{e^\dagger(x)}{e(x)}, \quad (3)$$

where $e^\dagger(x) = \int_x^\infty e(y)d(y)dy$ denotes the number of years lost due to death (Vaupel and Canudas-Romo, 2003) and $d(x)$ is the distribution of lifespans after age x . Keyfitz and Caswell (2005) defined $H(x)$ as the elasticity of $e(x)$ due to changes in the force of mortality. For example, if $H(x) = 0.3$, then a uniform reduction of one percent in the force of mortality at all ages above x will yield an increase of 0.3 percent in $e(x)$. Goldman et al. (1986) and Vaupel (1986) reformulated $H(x)$ as a measure of relative lifespan inequality: if everyone dies at the same age (minimum inequality) then $H(x) = 0$ and in the case of high lifespan inequality, $H(x) = 1$. An important characteristic of $H(x)$ is that it does not depend on the level of mortality (Wrycza et al., 2015). This property is particularly important in our study because we compare the shape of the distribution of lifespans after retirement ages c and t , which changes over time. The use of measures of absolute inequality (e.g., standard deviation) could lead to different results depending on the onset age of the calculation, because these measures do not control for differences in the average remaining lifespan; in our case the average remaining lifespan also varies according to age c and t .

3.2.2. Actuarial measures

To evaluate the financial implications of linking retirement age to developments in life expectancy, we calculate the financial transaction of a pension in terms of life annuities. For this purpose, we calculate the present value of a series of monetary payments of \$1 made until the pensioner dies, also called the cost of a life annuity (Bowers et al., 1997; Dowd et al., 2011). We assume that there are no periods of guarantees to the life annuity and that pension payments of one monetary unit are paid continuously per year starting from age x . Further, pension payments carry interest, which is determined by the force of interest, δ . Thus, the cost of a life annuity is defined by:

$$\bar{a}(x) \equiv \frac{\int_x^\infty l(y)e^{-\delta y}dy}{l(x)e^{-\delta x}}, \quad (4)$$

If the force of interest is equal to zero, i.e. money carries no interest, the cost of a life annuity is only determined by the survival of the pensioners and equal to the expected number of years they have to live. This means that if $\delta = 0$ then $\bar{a}(x) = e(x)$.

We also calculate the entropy of the cost of a life annuity (Haberman et al., 2011). At a given value of δ , the entropy measures the sensitivity of $\bar{a}(x)$ to changes in death rates and it is defined as:

$$\bar{H}(x, \delta) \equiv \frac{\int_0^\infty \bar{a}(y)d(y)e^{-\delta y}dy}{\bar{a}(y)} \equiv \frac{\bar{a}^\dagger(y)}{\bar{a}(y)}, \quad (5)$$

where $\bar{a}^\dagger(x) = \int_x^\infty \bar{a}(y)d(y)e^{-\delta y}dy$ can be interpreted as the average number of life annuity payments lost due to death (see Appendix A, Section A.2 for more details on the derivation of Equation (5)). It is also straightforward to show that, in the case of $\delta = 0$, then $\bar{H}(x, \delta = 0) = H(x)$ and $\bar{a}^\dagger(x) = e^\dagger(x)$.

As the objective of this paper is to analyse the socio-economical differences in a pension system where the retirement age is linked

to life expectancy, we evaluate relative differences in $\bar{a}(x)$ and $\bar{H}(x, \delta)$ to the total:

$$\Delta\bar{a}_s(x) = \frac{\bar{a}_s(x) - \bar{a}_p(x)}{\bar{a}_p(x)}, \quad (6)$$

$$\Delta\bar{H}_s(x, \delta) = \frac{\bar{H}_s(x, \delta) - \bar{H}_p(x, \delta)}{\bar{H}_p(x, \delta)}, \quad (7)$$

where s and p denote values of $\bar{a}(x)$ and $\bar{H}(x, \delta)$ for a specific socio-economic group and for the total population, respectively.

For simplicity, we assume that all individuals in the population have accumulated the same amount of savings at retirement, that they all retire at the same retirement age (either c or t) and that they face the same interest rate δ . Thus, differences in $\bar{a}(x)$ and $\bar{H}(x, \delta)$ between socio-economic groups arise solely through differences in the distribution of lifespans for each group. Socio-economic groups with positive values of $\Delta\bar{a}(x)$ have a higher annuity cost and hence are expected to receive higher expected pension benefits compared to the population average. Socio-economic groups with positive values of $\Delta\bar{H}(x, \delta)$, exhibit more variable costs of the annuity and hence experience more uncertainty about their expected pension payments. The uncertainty is a consequence of more unequal lifespans and the expected pension payments thus vary because the times of death for these groups are more uncertain than for the population average. Hence, groups with a positive value are exposed to higher micro longevity risk (Hari et al., 2008) than the population average. The underlying demographic mechanisms $\bar{H}(x, \delta)$ are disentangled with a new decomposition method, introduced in Section A.5 of Appendix A.

4. Results

Fig. 1 summarises the two demographic scenarios at retirement that are analysed in this study. The first scenario portrays the pension scheme that was in force during the period 1985–2016. We denote this scenario as the *current* pension scheme (measures indicated in black). The second scenario assumes that the target retirement age was operational from 1985, such that remaining life expectancy is always 14.5 years at the retirement age ($e(t) = 14.5$). We denote this scenario as the *target* pension scheme, and it is indicated in Fig. 1 by the measures in green. All the calculations shown in Fig. 1 consider demographic information of the total population with no distinction between the sexes, because the reform does not make such distinction (Danish Ministry of Economic Affairs and Interior, 2018).

The current retirement age, c , was set at age 67 during the period 1985–2004. From 2004 onwards, c was fixed at age 65. In Panel B of Fig. 1, we observe that life expectancy after age c trended upwards. This indicates that, under the current pension scheme, individuals from more recent generations spend more time in retirement than those from previous generations. The increasing trajectory of $e(c)$ was the major reason for the Danish pension reform in 2007 (Danish Ministry of Economic Affairs and Interior, 2017, 2018). Panel A of Fig. 1 also shows the trajectory over time of the target retirement age, t . During the period 1985–1995, t remained constant around age 67. From 1995 onwards, age t went up, reaching values of 71 years in 2016. Given that t indicates the age at which $e(t) = 14.5$, the increasing trajectory of t implies that a person aged 71 years in 2016 enjoyed the same remaining life expectancy after retirement as a person aged 67 years in 1995 (14.5 years in this case). In Panel B of Fig. 1, we observe that the increasing pattern in $e(c)$ is offset by setting retirement age to t , since $e(t)$ remains constant over time. This implies that, under the target pension scheme, individuals are expected to spend the same average number of years in retirement

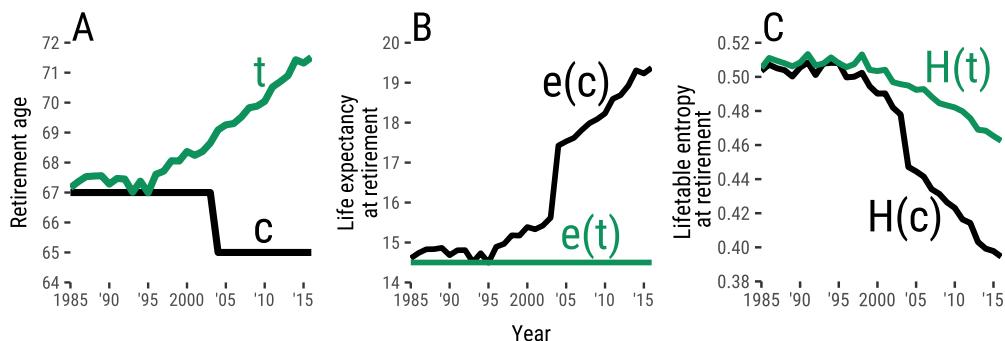


Fig. 1. Trends over time in statutory retirement ages (Panel A), life expectancy (Panel B) and lifespan inequality (Panel C) for the total population under the current and target pension schemes. 1985–2016. (For interpretation of the colours in the figure(s), the reader is referred to the web version of this article.)

regardless of the year in which they retire. This mechanism controls for unexpected changes in life expectancy, reducing exposure to macro-longevity risk (Blake et al., 2018; Hari et al., 2008).

Panel C of Fig. 1 shows trends in lifespan inequality measured by the lifetable entropy (Keyfitz and Caswell, 2005), calculated at the current and target retirement ages $H(c)$ and $H(t)$, respectively. We observe that $H(c)$ and $H(t)$ remained roughly constant during the period 1985–2000. Thereafter, both measures declined toward values of 0.39 for $H(c)$ and 0.46 for $H(t)$, respectively. The downward trend of $H(c)$ is more pronounced than the trend of $H(t)$ (the annual average decline of $H(c)$ was about 1.06% and 0.50% for $H(t)$). Furthermore, $H(c)$ is lower than $H(t)$, implying that lifespan inequality is lower after retirement age c than it is after age t . Lifespan inequality is also an indicator of uncertainty about the length of life at the individual level (Alvarez et al., 2019; van Raalte et al., 2018). Therefore, the results shown in Fig. 1 indicate that, at population level, pensioners are more uncertain about the number of years they spend in retirement when they retire at age t than when they retire at age c .

The patterns described in Fig. 1 are calculated for the total population. However, not all socio-economic groups have the same probability of surviving to retirement ages and not all of them exhibit the same $e(x)$ and $H(x)$. The levels and trends over time of these measures largely depend on the distribution of lifespans of each socio-economic group (see Section A.1 in Appendix A). In the following sections we present trends over time in the probability of surviving to retirement ages, together with trends in life expectancy and lifespan inequality according to specific demographic profile of each socio-economic group. We analyse differences between them and their impact on the cost of pensions.

4.1. Differences in survival at retirement between socio-economic groups

Fig. 2 shows that *i*) a clear socio-economic gradient in survival exists at retirement, and *ii*) such gradient varies depending on whether retirement age is set at age c or at age t . Panels A and B of Fig. 2 depict sex-specific probabilities of surviving from age 50 to retirement ages (to age c in solid lines and to age t in dashed lines) for the total population and by socio-economic groups. Females in the highest socio-economic group (G5) have similar probabilities of surviving to both retirement ages c and t (0.96 and 0.93 under retirement ages c and t in 2016). This is not the case for males in the lowest socio-economic group (G1), where probabilities of surviving to retirement ages c and t are lower than those for females and differ greatly (0.82 to retirement age c and 0.69 to retirement age t in 2016).

Panels C and D of Fig. 2 depict probabilities of surviving for 14.5 years after retirement ages c and t for females and males, respectively. Given that the pension reform (Danish Ministry of Economic

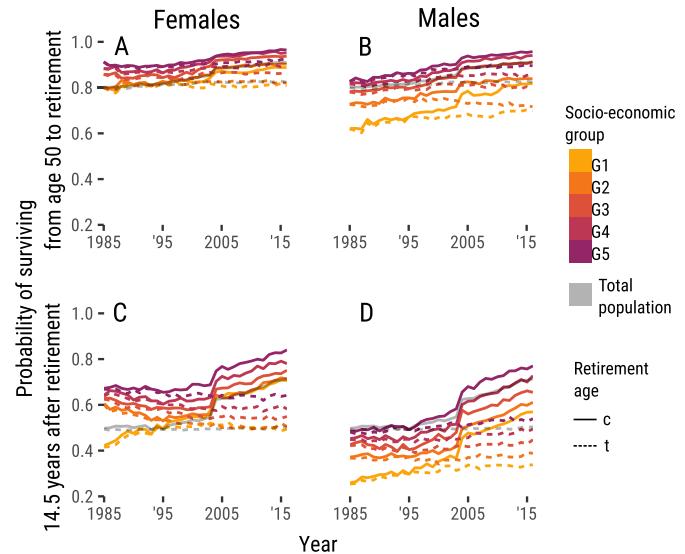


Fig. 2. Survival probabilities by socio-economic groups. Panels A and B indicate the probability of surviving from age 50 to retirement age (c and t). Panels C and D indicate the probability of surviving 14.5 years after retirement (c and t). Solid and dashed lines indicate the survival probability of retirement ages c and t , respectively. Grey lines indicate probabilities calculated for the total population. Both sexes, 1985–2016.

Affairs and Interior, 2017) implies that, on average, pensioners will receive pension payments during an expected period of 14.5 years, these probabilities are indicators of who is more likely to survive to the end of the expected time of pension payments. We also observe a socio-economic gradient in these probabilities; however, in this case, the magnitude of differences between socio-economic groups is much bigger than in Panels A and B. In 2016, the probabilities of surviving 14.5 years after retirement were 0.84 at age c and 0.64 at age t for females in the highest socio-economic group (G5). For males in the lowest socio-economic group (G1), these probabilities were 0.57 at age c and only 0.33 for males at age t . Fig. 2 entails that males in lower socio-economic groups have lower probability of surviving after retirement in comparison to females and that this disadvantageous setting is magnified when retirement age is linked to life expectancy (age t).

The socio-economic gradient in survival probabilities at retirement documented in Fig. 1 is reflected in summary measures such as life expectancy (Fig. 3) and lifespan inequality (Fig. 4). Figs. 3 and 4 show that higher socio-economic groups enjoy longer lives (higher life expectancy, $e(x)$) and experience less lifespan inequality (lower entropy, $H(x)$) than lower socio-economic groups. At the retirement age c (Panels A and B of Figs. 3 and 4), females in G5 exhibit $e(c)$ of 22.71 and $H(c)$ of 0.31 in 2016. Males in G1 exhibit a $e(c)$ of 16.04 and $H(c)$ of 0.47 during the same year.

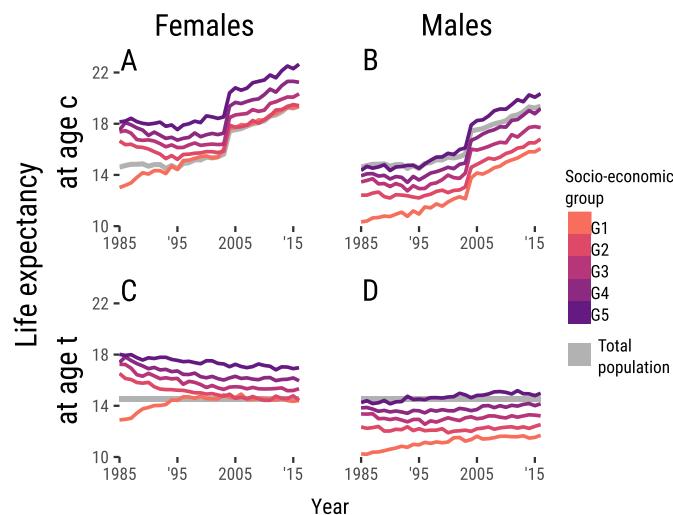


Fig. 3. Life expectancy at the current and target retirement ages by socio-economic groups. Both sexes, 1985–2016.

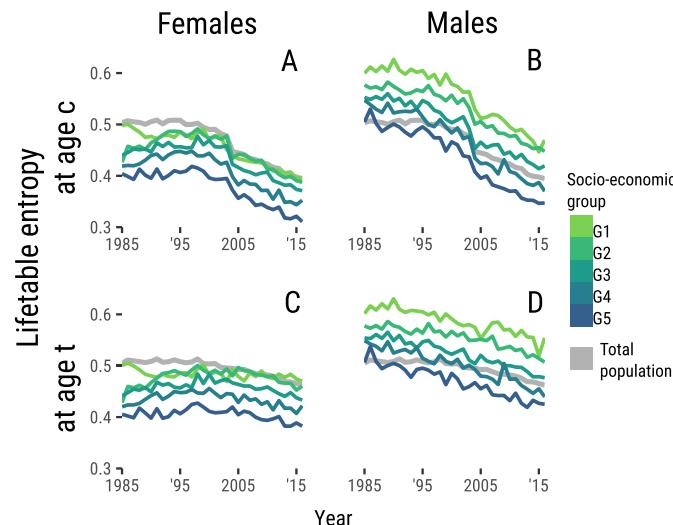


Fig. 4. Lifespan inequality at the current and target retirement ages by socio-economic group. Both sexes, 1985–2016.

These disparities between higher and lower socio-economic groups prevail throughout all the period. In particular, males from lower socio-economic groups under-perform in life expectancy and lifespan inequality.

The absolute disadvantages in $e(x)$ and $H(x)$ for lower socio-economic groups are magnified when these measures are calculated at the target retirement age t (Panels C and D of Figs. 3 and 4). For example, in 2016, males in G1 exhibit $e(t)$ of 11.69 and $H(t)$ of 0.55. Thus, by retiring at age t rather than at age c , males in G1 experience a reduction of 4.35 years in the average prospect of life after retirement (life expectancy) and an increase of 0.08 in its uncertainty (lifespan inequality) for the year 2016. This is a demographic implication of setting t at a higher age than c (t at age 71.51 and c at age 65 in 2016). From the analysis of Figs. 3 and 4, it is clear that linking retirement age to life expectancy sets individuals from lower socio-economic groups in a more disadvantaged demographic setting, compared to the current pension scheme, in which the statutory retirement age is constant.

Trends in $e(x)$ and $H(x)$ across socio-economic groups follow similar patterns over time to the ones observed for the total population (Panels B and C of Fig. 1 and grey lines in Figs. 3 and 4). The absolute level of these trends (in the total population and in socio-

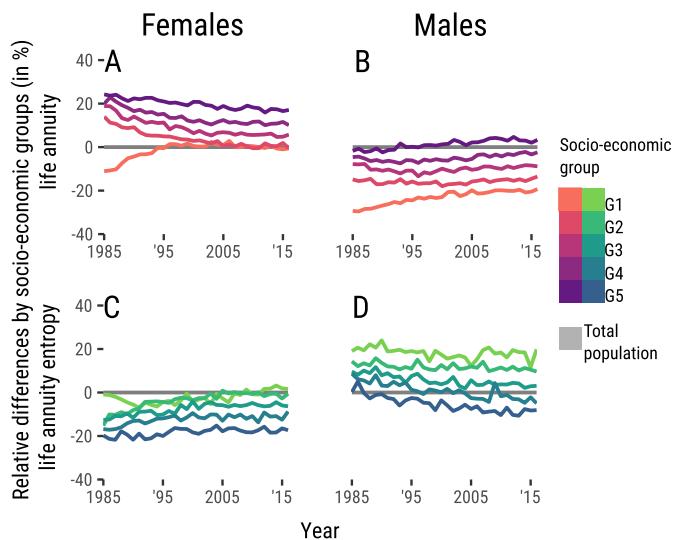


Fig. 5. Relative differences by socio-economic group in life annuities (Panels A and B) and in life annuity entropies (Panels C and D) by sex. Both sexes, 1985–2016.

economic groups) depends on how retirement ages are defined. For example, in 2004, c was reduced from age 67 to age 65 (Danish Ministry of Economic Affairs and Interior, 2017). This change in retirement age c implied an increase in life expectancies at retirement for the total population (Fig. 1) and for all socio-economic groups. However, this increase in life expectancy at retirement is an artefact of reducing age c from 67 to 65. Therefore, it is necessary to control for the absolute level in $e(x)$ and $H(x)$ of the total population to quantify the extent of the socio-economic gap.

4.2. Actuarial perspective on socio-economic inequalities

In this section we analyse relative differences in the financial cost of pensions between each socio-economic group and the total population. In Fig. 5 panels A and B, we show the relative difference in the cost of an annuity and its associated entropy for the socio-economic groups and for the total population, calculated using Equations (6) and (7). The cost of an annuity is higher for all female socio-economic groups after 1990, compared to the total population meaning that the present values of their pensions payments are higher. Hence, for the same annuity, females are expected to receive higher payments than the average of the population. On the contrary, only the top affluence group for males after 1995 has payments higher than the average for the population. The force of interest is assumed to be zero in Fig. 5, for simplicity but the results are robust to higher interest levels, see Fig. 8 in Appendix A. A low force of interest reflects the current financial situation in Denmark (OECD, 2019a).

Panels C and D show the associated entropy to the cost of an annuity. For all the female socio-economic groups, the entropy is lower than the total population meaning that the uncertainty related to pension payments for the females group is lower than the average. This is a result of a lower uncertainty about their average life span as shown in Section 4.1. The reverse is again true for the male group where only the highest group has an entropy value below the average. Hence, most of the male groups have lower expected pension payments for the same annuity with higher uncertainty related to the payments.

For both females and males there is a clear rank in Fig. 5 between the socio-economic groups meaning that most affluence groups for each sex have the highest expected payments with the lowest uncertainty from the same annuity. This shows that the socio-economic differences in life expectancies and lifespan in-

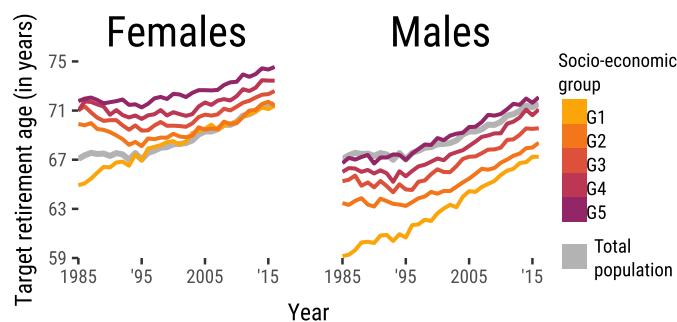


Fig. 6. Target retirement ages calculated according to the demographic profile of each socio-economic group. 1985–2016.

equality directly affect life annuities. This is especially true for a low interest regime, because the annuity payments with zero interest only are determined by an individual's length of life, which is the financial setting prevailing in Denmark and many other countries (OECD, 2019a). However the results are robust to higher to higher interest levels, as shown in Fig. 8 in Appendix A.

To understand the underlying mechanisms behind changes in $\bar{H}(x, \delta)$, in Appendix A.2 we have decomposed the differences in relative changes over time in $\bar{H}(x, \delta)$ between the highest and the lowest socio-economic groups. The main conclusion is that both, the level of life expectancy and absolute lifespan inequality play an important role in the uncertainty related to the cost of an annuity.

4.3. Differentiation in retirement ages by socio-economic groups

In this article, we quantify the socio-economic disparities at retirement that arise under retirement ages c and t , which are computed using demographic information of the total population. In this regard, previous studies (Ayuso et al., 2017; Sánchez-Romero et al., 2020) argue that socio-economic disparities at retirement can be reduced by differentiating retirement ages according to the demographic profile of each socio-economic group. In the Danish case, the differentiation could occur in terms of the target retirement age, where retirement ages are calculated such that each socio-economic group spends 14.5 years (in average) according to its own demographic profile. In Fig. 6, we show that this policy implies a difference of almost eight years between the highest socio-economic group for females and the lowest socio-economic group for males. We anticipate that a policy of this type could imply major legal issues and implementation challenges. It could also influence individuals to move between socio-economic groups (people can move to lower socio-economic groups to retire at an earlier age) and this could have a major impact in the sustainability of the pension system. Further, a differentiation of retirement ages by socio-economic group could be perceived as discriminatory, because, in practice, it might be difficult to allocate individuals to a specific socio-economic group (Arcanjo, 2019). Hence, while differentiating retirement ages by socio-economic group could reduce socio-economic disparities at retirement due to heterogeneity in longevity, other important issues might arise from this policy.

Then, the question arises as to how to set retirement ages. In this study we show that, while linking retirement age to life expectancy (of the total population) might contribute to ensuring the sustainability of national pension systems (Danish Ministry of Economic Affairs and Interior, 2018), this policy introduces higher inequality. Therefore, retirement ages should be seen as a trade-off between constant life expectancies and lower lifespan inequality. In addition, one possible policy to reduce socio-economic disparities is to link early retirement to the timing of individuals' first

full-time jobs.⁷ Early retirement could depend on the number of years since the time the individual enters to the labour market, by taking into account the years of unemployment. This type of policy tends to benefit individuals in lower socio-economic groups, as they tend to enter the labour market earlier than those in higher socio-economic groups (Bravo and Herce, 2019).

In this study, we explore the implications of unequal lifespans by looking at Danish pension reform. However, these issues are not restricted to Denmark. In a similar fashion, other countries, such as Estonia, Finland, Italy, the Netherlands, Portugal and the Slovak Republic will also modify retirement ages, by linking them to life expectancy (OECD, 2018b, 2019b) in a context of low interest rates (OECD, 2019b). Therefore, it is likely that demographic imbalances after retirement will also arise in those countries.

5. Discussion

In this study, we analyse the implications of linking retirement age to life expectancy, reflected in socio-economic disparities in longevity and their influence on the financial cost of pensions. We advance the following findings in terms of lifespan inequality, longevity risk and socio-economic differences in pension outcomes.

Lifespan inequality at retirement and longevity risk

Under the target pension scheme, the upward trend of life expectancy (calculated for the total population) is offset and the exposure of pensions to macro-longevity risk (Blake et al., 2018) is reduced, because it is expected that retirees spend similar average times in retirement. However, the linkage rule increases lifespan inequality after retirement and, in consequence, the sensitivity of life annuities to changes in mortality rates is also increased (micro-longevity risk, see Hari et al. (2008)). Allowing for life annuities, backed up by riskier financial products that yield higher interest rates, can reduce the exposition to micro-longevity risk that is embedded in the target pension scheme. Even in this case, individuals will deal with uncertain pension payments under the target pension scheme, either through increased micro-longevity risk or through higher financial risk.

Another implication of high lifespan inequality after retirement relates to ineffective financial planning. At an individual level, the high levels of lifespan inequality imposed by the target pension scheme can blur the perception about the future, such that individuals might take important financial decisions lightly (Aronsson and Blomquist, 2018). In particular, high lifespan inequality at retirement can discourage individuals from participating in schemes that require voluntary contributions to pension funds (pension schemes from pillar 2 and pillar 3). Consequently, individuals might incur shortages of pension wealth to face post-retirement years.

Socio-economic disparities in longevity and impact on pension schemes

In this study we also show that socio-economic disparities in longevity prevail, regardless of the age at which individuals retire. Males from lower socio-economic groups are at a clear disadvantage, since they spend less time in retirement, pay higher pensions costs per expected benefits and are exposed to higher longevity risk than upper socio-economic groups. These disparities are magnified when linking retirement age to developments in total life expectancy.

⁷ A policy of this type, which allows people to retire earlier was implemented in Denmark in 2020, see <https://bm.dk/media/15057/aftaletekst-tidlig-pension.pdf> in Danish.

The question thus arises: how do these socio-economic disparities in longevity affect pension objectives, such as redistribution⁸ of income in the Danish pension system? To understand this issue, it is necessary to distinguish between the different pension schemes operating in pillars 1 and 2. In the case of old age pensions of pillar 1 (PAYG scheme), individuals receive pension payments regardless of the time spent actively participating in the labour market (Andersen, 2015). In the case of non-participation and not having any income other than pension payments (i.e. lowest socio-economic groups), pensioners would receive the highest amount of old age pension. Furthermore, the amount of old age pension is reduced based on pension income from pillar 2. This means that old age pensions payments from pillar 1 are regulated by the other pillars. For example, individuals receiving higher pension income from labour market pensions (pillar 2), receive a small amount of old age pension (pillar 1). These progressive factors built into pillar 1 aim at reducing the financial inequality in pensions payments experienced by the lower socio-economic groups. In consequence, potential income redistribution might occur from higher to lower socio-economic groups via taxation, because people in high socio-economic groups pay higher taxes from higher earned income during their working life and receive less old age pension at retirement.

Pillar 2, on the other hand, is mostly constituted by mandatory defined contribution schemes (labour market pensions). It has been shown that pension objectives in defined contribution schemes are little affected by policies aiming at increasing retirement age (Alonso-García et al., 2018; Sánchez-Romero et al., 2020). However, redistribution issues could arise within highly heterogeneous defined contribution pension schemes that cover a large number of individuals from very different socio-economic groups (Brown, 2003; Holzmann et al., 2019; Milevsky, 2020).

Policies that allow for early and late retirement options also play an important role in either reversing or magnifying socio-economic inequalities in pension schemes. For old age pensions (pillar 1), individuals have the option to postpone retirement up to 10 years which results in a higher amount of pension (OECD, 2015). Thus, additional contributions to the old age pension translate into additional benefits, while annuity payments will run for a shorter period. For the case of labour market pensions (defined benefit schemes of pillar 2), individuals can postpone retirement, which results in higher contributions to their pension accounts. Early retirement on these schemes, however, is heavily penalised because pension payments are subject to high taxation rates⁹ (Arnberg and Barslund, 2014). This setting aims to promote retirement at older ages, but it also has detrimental implications for lower socio-economic groups. If they choose to retire early, they get penalised through high taxation rates, but if they decide to retire at the target retirement age, they would spend fewer expected years receiving pension (as shown in our results). Further research that would consider data on accumulation of pension wealth in Denmark is required, to properly quantify the interaction between early retirement, taxation and benefits (perhaps in a similar fashion to the analysis performed by (Sánchez-Romero et al., 2020)).

Robustness towards cohort measures

In this study we use period data to calculate retirement ages and to quantify socio-economic differences in demographic and actuarial outcomes. Alternatively, the same analysis could have been

⁸ In Appendix A we discuss how socio-economic disparities in longevity affect other pension objectives, such as actuarial fairness, sustainability and adequacy.

⁹ If individuals started saving in their defined contribution schemes before May 2007, they can retire some years before the official retirement age without being taxed (Arnberg and Barslund, 2014).

performed using a cohort life table approach (Ayuso et al., 2020). We replicate the same analysis by using cohort measures to quantify the differences between both approaches. The complete analysis can be found in Section A.4 of Appendix A. Our results show that the target retirement age is higher under the cohort perspective than under the period perspective (Fig. 10 in Appendix A). However, the magnitude of the gap between both measures is not overwhelmingly large, as also observed in other populations (e.g., the USA, see Goldstein and Wachter (2006)). In terms of lifespan inequality, entropies are remarkably similar from both the period and cohort perspectives. Further, we show that the socio-economic gap in life annuity factors is remarkably similar from perspectives (Fig. 11 in Appendix A). This shows that our calculations (using period data) correctly depict the implications of linking life expectancy to retirement age.

Declaration of competing interest

None declared.

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Appendix A

A.1. Distributions of lifespans by socio-economic groups

Fig. 7 shows the distributions of lifespans after age 65 for the lowest and highest socio-economic groups (G1 and G5) by sex, for selected years during the period 1985–2016. The survival trajectories of Danish females in the lowest socio-economic group have approached those in G5. This is reflected in the increasingly overlapping areas of the distributions of lifespans, which in 1985 comprised 73% and 84% in 2015. For males, the overlapping area between G1 and G5 groups remained similar over time. Despite the large shared area between distributions, we can observe noticeable differences between them.

The distribution of lifespans for the G5 group is, in all years, shifted to the right and its tail is much heavier, compared to the distribution of the lowest socio-economic group. This means that a greater number of individuals form the highest socio-economic group outlived those in the lowest socio-economic group. The shape of the distribution of lifespans for males also differs considerably in comparison to that of females. These discrepancies in the distribution of lifespans result in different longevity outcomes depending on the profile of each socio-economic group. Such inequalities are examined in Section 4.

A.2. Alternative expression of $\bar{H}(x, \delta)$

The present value of a life annuity at age x (Bowers et al., 1997) is defined as

$$\bar{a}(x) = \frac{\int_x^\infty l(y)e^{-\delta y} dy}{l(x)e^{-\delta x}}, \quad (8)$$

where $l(x)$ is the survivorship function at age x and δ represents the force of interest. Haberman et al. (2011) defined the entropy of a life annuity as

$$\bar{H}(x, \delta) = \frac{-\int_0^\infty \ln(l(y))l(y)e^{-\delta y} dy}{\bar{a}(x)}, \quad (9)$$

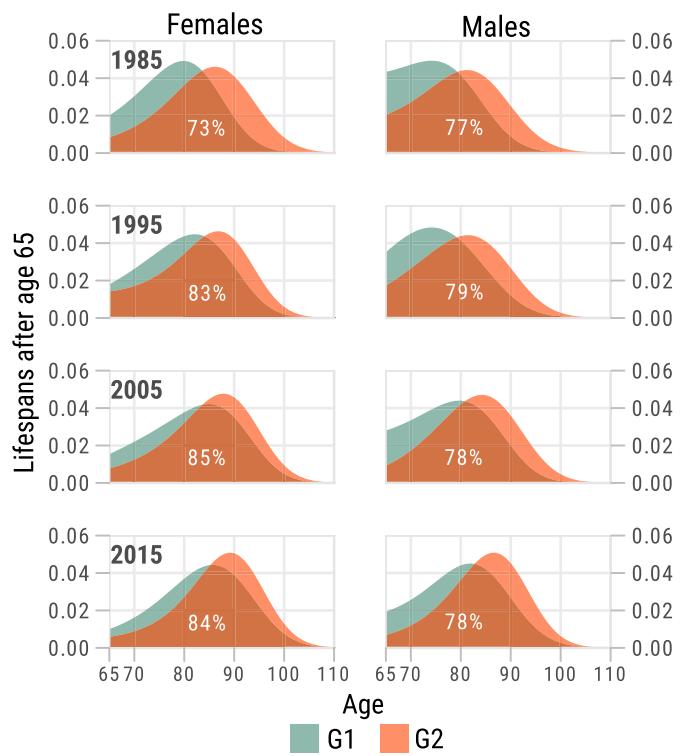


Fig. 7. Distribution of life table deaths conditioned on survival to age 65 for the highest (G5) and the lowest (G1) socio-economic groups in Denmark. Both sexes, selected years.

given that $\ln(l(y)) = -\int_0^y \mu(x)dx$, where $\mu(x)$ denotes the force of mortality and using reverse integration as in Vaupel (1986), we can re-express the numerator of Equation (9) as a measure of heterogeneity such that

$$\begin{aligned} - \int_0^\infty \ln(l(y))l(y)e^{-\delta y} dy &= \int_0^\infty l(y)e^{-\delta y} \int_0^y \mu(x)dx dy \\ &= \int_0^\infty \mu(x) \int_x^\infty l(y)e^{-\delta y} dy dx \\ &= \int_0^\infty \mu(x)l(x)e^{-\delta x}\bar{a}(x)dx \\ &= \int_0^\infty d(x)e^{-\delta x}\bar{a}(x)dx \end{aligned}$$

Thus,

$$\bar{H}(x, \delta) = \frac{\int_0^\infty d(x)e^{-\delta x}\bar{a}(x)dx}{\bar{a}(x)} = \frac{\dot{\bar{a}}(x)}{\bar{a}(x)}. \quad (10)$$

A.3. Decomposition of differences over time in $\bar{H}(\delta)$ between two populations

We denote the derivative with respect to time by placing a dot on top of the measure of interest. For example, the derivative of the life annuity factor is denoted as $\dot{\bar{a}}(x)$. As in Fernandez and Beltrán-Sánchez (2015), it is straightforward to show that the relative derivative of $\bar{H}(\delta)$ with respect to time is:

$$\dot{\bar{H}}(x, \delta) = \frac{\dot{\bar{a}}^\dagger(x)}{\bar{a}^\dagger(x)} - \frac{\dot{\bar{a}}(x)}{\bar{a}(x)} \quad (11)$$

Thus, Equation (11) implies that the relative derivative of $\bar{H}(x, \delta)$ with respect to time can be expressed as the difference between the relative derivative of $\bar{a}^\dagger(x)$ and $\bar{a}(x)$.

Assuming that we want to compare two populations A and B with entropies $\bar{H}_A(x, \delta)$ and $\bar{H}_B(x, \delta)$, by Equations (10) and (11) we can decompose the difference over time between $\bar{H}_A(x, \delta)$ and $\bar{H}_B(x, \delta)$ such that:

$$\frac{\dot{\bar{H}}_A(x, \delta)}{\bar{H}_A(x, \delta)} - \frac{\dot{\bar{H}}_B(x, \delta)}{\bar{H}_B(x, \delta)} = \underbrace{\frac{\dot{\bar{a}}_A^\dagger(x)}{\bar{a}_A^\dagger(x)} - \frac{\dot{\bar{a}}_B^\dagger(x)}{\bar{a}_B^\dagger(x)}}_{\text{dispersion}} + \underbrace{\frac{\dot{\bar{a}}_B(x)}{\bar{a}_B(x)} - \frac{\dot{\bar{a}}_A(x)}{\bar{a}_A(x)}}_{\text{translation}} \quad (12)$$

The *translation* component determines the difference due to changes in the mean value of the life annuity $\bar{a}(x)$ and the *dispersion* component denotes differences due to changes in absolute lifespan inequality denoted by $\bar{a}^\dagger(x)$. Positive values in any of these effects contribute to increasing the gap in $\bar{H}(x, \delta)$ between socio-economic groups, while negative values contribute to reducing it. We performed the decomposition at the current and the target retirement ages and the results are shown in Fig. 9. We selected the highest and lowest socio-economic groups as an illustration.

Fig. 9 shows that the dispersion component contributes positively to the socio-economic gap in $\bar{H}(x, \delta)$ for both sexes and at both retirement ages (orange bars in all Panels of Fig. 9). This means that the gap in $\bar{H}(x, \delta)$ between the highest and the lowest socio-economic groups is largely explained by differences in absolute lifespan inequality measured by $\bar{a}^\dagger(x)$. This component has remained similar over time, meaning that the gap in absolute lifespan inequality between the lowest and highest socio-economic groups has remained unchanged during the period of observation.

However, we also observe that the translation component (green bars in all Panels of Fig. 9) plays an important role in the reduction of the gap for both sexes, but particularly for females (Panels A.1, A.3, B.1 and B.3 of Fig. 9). The translation component is related to the cost of life annuities (or life expectancy in Panels A.1 and B.1, since $\bar{a}(x) = e(x)$ at $\delta = 0\%$). Thus, this finding entails that cost of life annuities for females in the lowest socio-economic group has decreased over time in comparison to the life annuity cost for the highest socio-economic groups. For males (Panels A.2 and B.2), the translation component has a smaller effect in reducing the gap between socio-economic groups than the one observed in females.

For an interest rate level of 5%, the dispersion and translation components reduce as the difference in survival matters less for the gap in the cost of the annuity. For males, the dispersion component remains the same when $\delta = 5\%$, since the absolute lifespan inequality weighs more in $\bar{H}(x, \delta)$ than the financial factor $e^{-\delta}$ (see Equation (9)). A δ higher than 5% would be necessary to reduce the dispersion component in males.

In conclusion, Fig. 9 depicts the demographic processes behind the socio-economic gap in $\bar{H}(x, \delta)$ and how these processes react to different interest rates. Fig. 9 shows that the translation effect played an important role in reducing the socio-economic gap in $\bar{H}(x, \delta)$ for females. Hence, improving survival for low socio-economic females not only reduced the gap in life expectancy and in the cost of life annuities, but also reduced the uncertainty related to the cost of the annuity, making it easier to hedge their micro-longevity risk. We also show that the socio-economic gap in $\bar{H}(x, \delta)$ is similar under both the current and the target retirement schemes.

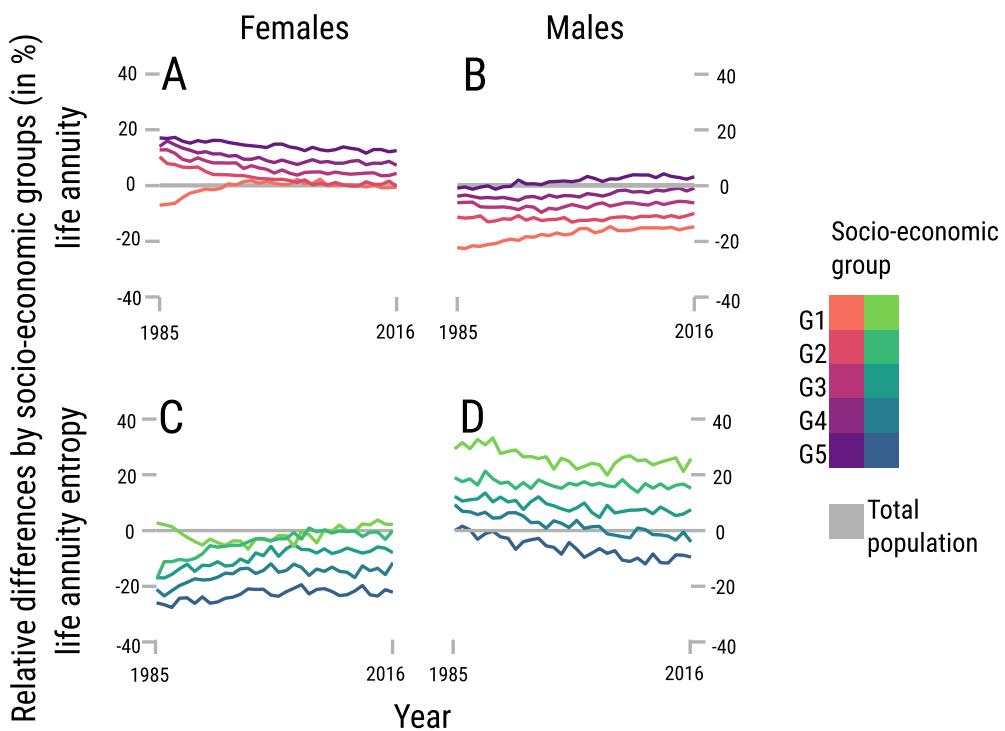


Fig. 8. Relative differences by socio-economic groups in life annuities (Panels A and B) and in life annuity entropies (Panels C and D) by sex assuming constant force of mortality of 5%. Both sexes, 1985–2016.

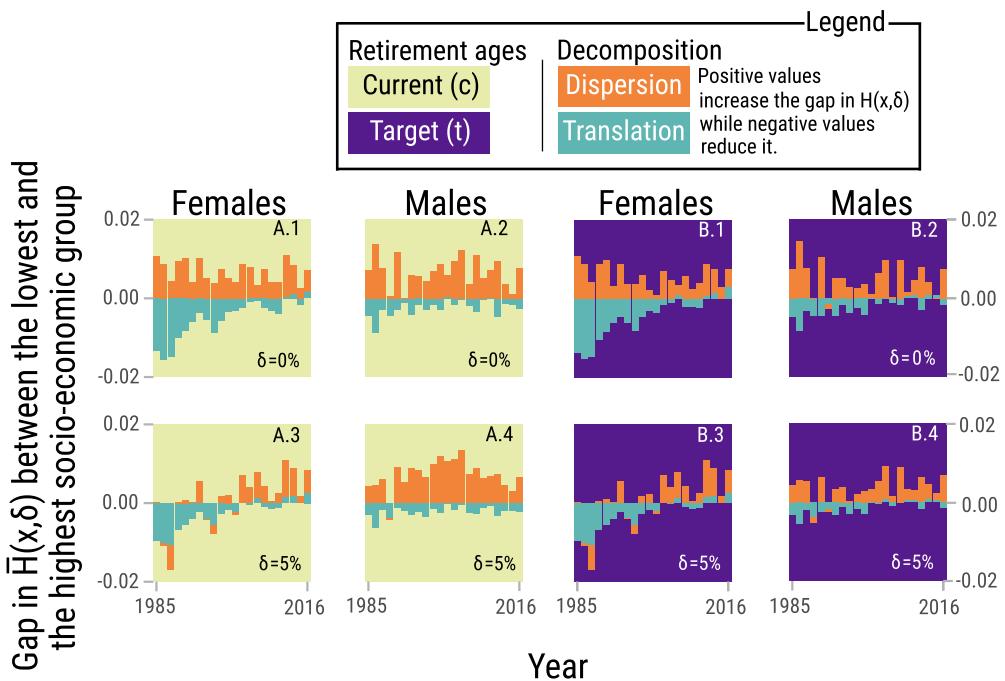


Fig. 9. Decomposition of differences in the relative change over time in the entropy of a life annuity by socio-economic group. Both sexes, 1985–2016.

A.4. Target retirement age from a cohort perspective

To quantify the impact of using cohort measures instead of period ones, we first forecasted period mortality rates by socio-economic groups using the method developed by Li and Lee (2005). Then, we reorganised such rates by cohort and computed target retirement age and life annuity factors by socio-economic groups. The rule to calculate the target retirement age in a cohort perspective is the same as the one used in this study with period data (i.e. the value of the variable " t_c " that solves the equation

$e(t_c) = 14.5$, where t_c is the target retirement age by cohort). To compare the cohort trend (t_c) against the target retirement age in period basis, we calculate the corresponding year at which each cohort will be eligible to retire such that Year = Cohort + t_c . Note that by calculating cohort trends from forecasted data we introduce uncertainty regarding the validity of assumptions used to project future demographic trends (Ayuso et al., 2020).

In panel A of Fig. 10, we observe that the target retirement age in the cohort basis (t_c , in red) is higher than the one calculated

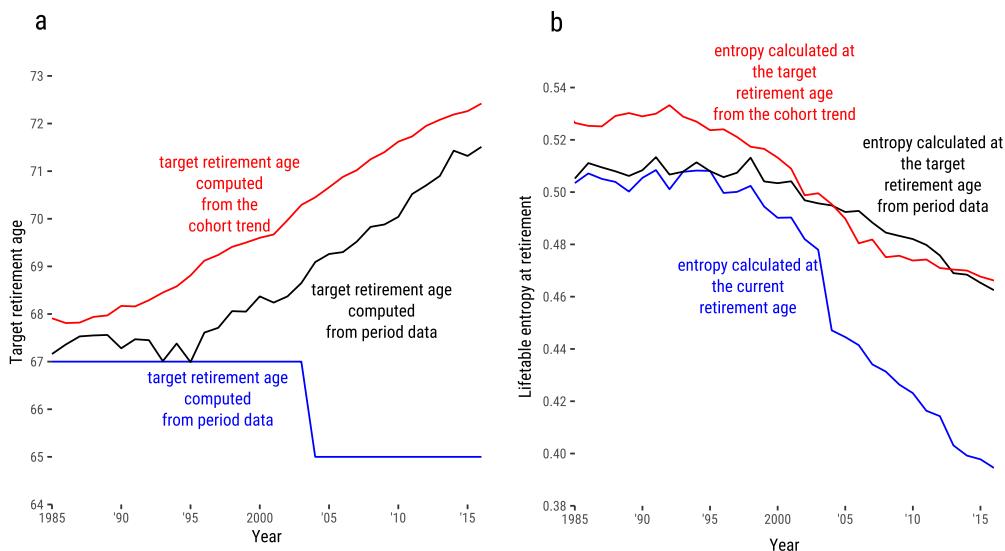


Fig. 10. Comparison of retirement ages and entropies using cohort data. 1985–2016.

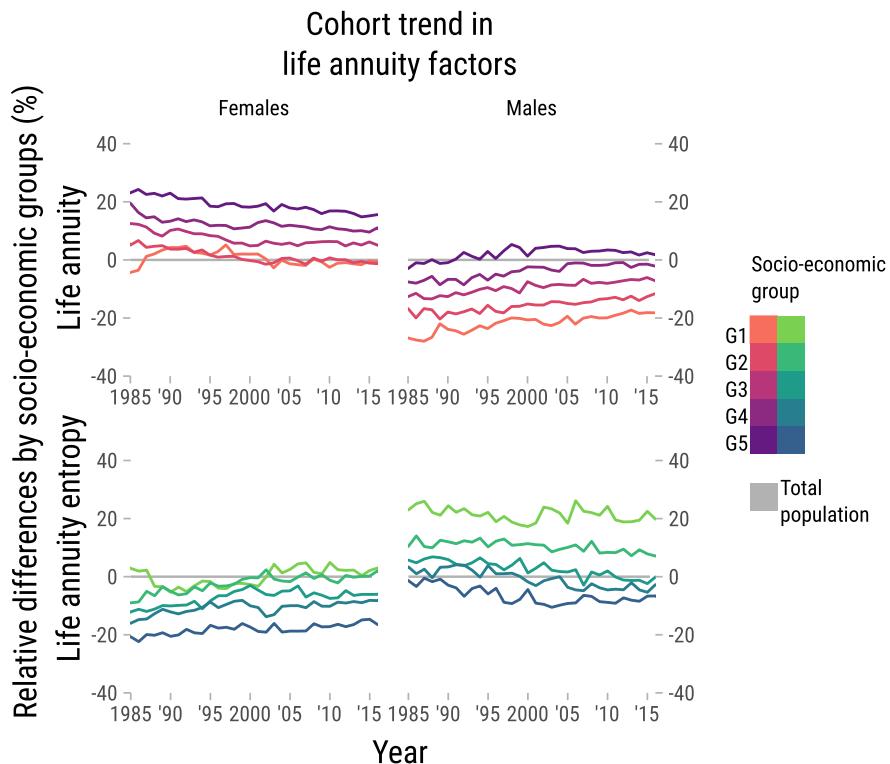


Fig. 11. Relative differences in the financial cost of pensions by socio-economic group with respect to the total population. Calculations performed using a cohort trend. 1985–2016.

from period data (in black). From 1985 to 1990, the gap was less than one year. After 1990, both retirement ages increased at the same pace and the gap remained constant around one year. While it is true that there are discrepancies between both target retirement ages, the magnitude of the gap is not overwhelmingly large, as also observed in other populations (e.g. the USA, see (Goldstein and Wachter, 2006)). Next, we computed life table entropy at the cohort retirement age (panel B of Fig. 10) and compared it with the entropy calculated with period data. We found that both entropies (period and cohort) are remarkable similar. This shows that, in terms of lifespan inequality after retirement, our results are practically the same under period or cohort perspectives.

In Fig. 11, we show the relative difference in the financial cost of pensions (life annuities) across socio-economic groups, by assuming interest rates of zero percent (which equals life expectancy) and five percent (as in Fig. 5 in the manuscript). We observe that the socio-economic gap under the cohort perspective is almost identical to the gap in the period perspective. Similarly, when increasing interest rates, the socio-economic gap reduces and converges towards the total population. Fig. 11 shows that the socio-economic gap in life annuities remains the same, regardless of using period or cohort data. Hence, this shows that our results capture well the existing socio-economic gap in life annuities. Such gap remains similar when looking at either the cohort and period perspective.

A.5. Socio-economic disparities in longevity and interaction adequacy of pensions

Replacement rates (including the old age pension and mandatory defined contribution schemes) are high in Denmark. The OECD (2019b) reports an average gross replacement ratio of 74.4% from public and private pension schemes, which is among the highest in the OECD countries (the OECD average is about 49%). When looking at socio-economic differences, the OECD (2019b) reports a gross replacement rate of 113% for the lowest income group and 64% for the highest income group. We assert that linking retirement age to life expectancy could affect these replacement rates and pension adequacy across the different socio-economic groups. Under the new retirement setting, individuals with higher life expectancies (i.e. higher socio-economic groups) will work for longer periods and accumulate more pension wealth. Conversely, those in lower socio-economic groups, while they might also save for a longer time, will not benefit from it, because they still die earlier. Thus, replacement rates reported by the OECD (2019b) are likely to be affected by the retirement policy.

References

- Aburto, J.M., Alvarez, J.-A., Villavicencio, F., Vaupel, J.W., 2019. The threshold age of the lifetable entropy. *Demographic Research* 41, 83–102.
- Alonso-García, J., Boado-Penas, M.d. C., Devolder, P., 2018. Adequacy, fairness and sustainability of pay-as-you-go-pension-systems: defined benefit versus defined contribution. *The European Journal of Finance* 24 (13), 1100–1122.
- Alvarez, J.-A., Aburto, J.M., Canudas-Romo, V., 2019. Latin American convergence and divergence towards the mortality profiles of developed countries. *Population Studies*, 1–18.
- Andersen, T.M., 2015. Robustness of the Danish pension system. CESifo DICE Report, 13 (2), 25–30.
- Arcanjo, M., 2019. Retirement pension reforms in six European social insurance schemes between 2000 and 2017: more financial sustainability and more gender inequality? *Social Policy and Society* 18 (4), 501–515.
- Arnberg, S., Barslund, M., 2014. The crowding-out effect of mandatory labour market pension schemes on private savings: evidence from renters in Denmark. CEPs Working Document (390).
- Aronsson, T., Blomquist, S., 2018. Uncertain Length of Life, Retirement Age, and Optimal Pension Design. CESifo Working Paper Series.
- Ayuso, M., Bravo, J.M., Holzmann, R., 2017. Addressing longevity heterogeneity in pension scheme design. *Journal of Finance and Economics* 6 (1), 1–21.
- Ayuso, M., Bravo, J.M., Holzmann, R., 2020. Getting life expectancy estimates right for pension policy: period versus cohort approach. *Journal of Pension Economics & Finance* 1 (20).
- Balter, A.G., Kallestrup-Lamb, M., Rangvid, J., 2020. Variability in pension products: a comparison study between the Netherlands and Denmark. *Annals of Actuarial Science* 14 (2), 338–357.
- Barr, N., Diamond, P., 2006. The economics of pensions. *Oxford Review of Economic Policy* 22 (1), 15–39.
- Bingley, P., Datta Gupta, N., Kallestrup-Lamb, M., Pedersen, P., 2020. The role of social security reforms in explaining changing retirement behavior in Denmark 1980–2016. In: ESocial Security and Retirement Around the World. In: National Bureau of Economic Research, vol. 10.
- Blake, D., Cairns, A.J.G., Dowd, K., Kessler, A.R., 2018. Still living with mortality: the longevity risk transfer market after one decade. *British Actuarial Journal* 24.
- Bowers, N.L., Gerber, H., Hickman, J., Jones, D., Nesbitt, C., 1997. *Actuarial Mathematics*. The Society of Actuaries, United States of America.
- Bravo, J.M., Herce, J.A., 2019. Career breaks, broken pensions? Long-run effects of early and late-career unemployment spells on pension entitlements. *Journal of Pension Economics & Finance*, 1–27.
- Brown, J., 2002. Differential mortality and the value of individual account retirement annuities. In: *The Distributional Aspects of Social Security and Social Security Reform*. University of Chicago Press, pp. 401–446.
- Brown, J.R., 2003. Redistribution and insurance: mandatory annuitization with mortality heterogeneity. *The Journal of Risk and Insurance* 70 (1), 17–41.
- Burger, O., Baudisch, A., Vaupel, J.W., 2012. Human mortality improvement in evolutionary context. *Proceedings of the National Academy of Sciences* 109 (44), 18210–18214.
- Cairns, A.J., Kallestrup-Lamb, M., Rosenskjold, C., Blake, D., Dowd, K., 2019. Modelling socio-economic differences in mortality using a new affluence index. *ASTIN Bulletin: The Journal of the IAA*, 1–36.
- Camarda, C.G., et al., 2012. Mortalitysmooth: an R package for smoothing Poisson counts with p-splines. *Journal of Statistical Software* 50 (1), 1–24.
- Chetty, R., Stepner, M., Abraham, S., Lin, S., Scuderi, B., Turner, N., Bergeron, A., Cutler, D., 2016. The association between income and life expectancy in the United States, 2001–2014. *JAMA* 315 (16), 1750–1766.
- Chomik, R., Whitehouse, E.R., 2010. Trends in pension eligibility ages and life expectancy, 1950–2050. Technical report, OECD Social, Employment and Migration Working Papers, No. 105. OECD Publishing, Paris.
- Coughlan, G.D., Khalaf-Allah, M., Ye, Y., Kumar, S., Cairns, A.J., Blake, D., Dowd, K., 2011. Longevity hedging 101: a framework for longevity basis risk analysis and hedge effectiveness. *North American Actuarial Journal* 15 (2), 150–176.
- Danish Ministry of Economic Affairs and Interior, 2017. Denmark's Convergence Programme 2017. Technical report, Danish Government, Copenhagen.
- Danish Ministry of Economic Affairs and Interior, 2018. Denmark's Convergence Programme 2018. Technical report, Danish Government, Copenhagen.
- Dowd, K., Cairns, A.J., Blake, D., Coughlan, G.D., Khalaf-Allah, M., 2011. A gravity model of mortality rates for two related populations. *North American Actuarial Journal* 15 (2), 334–356.
- Duncan, G.J., Daly, M.C., McDonough, P., Williams, D.R., 2002. Optimal indicators of socioeconomic status for health research. *American Journal of Public Health* 92 (7), 1151–1157.
- Fernandez, O.E., Beltrán-Sánchez, H., 2015. The entropy of the life table: a reappraisal. *Theoretical Population Biology* 104, 26–45.
- Goldman, N., Lord, G., May, N., 1986. A new look at entropy and the life table. *Demography* 23 (2), 275–282.
- Goldstein, J.R., Wachter, K.W., 2006. Relationships between period and cohort life expectancy: gaps and lags. *Population Studies* 60 (3), 257–269.
- Haberman, S., Khalaf-Allah, M., Verrall, R., 2011. Entropy, longevity and the cost of annuities. *Insurance. Mathematics & Economics* 48 (2), 197–204.
- Hari, N., De Waegenaere, A., Melenberg, B., Nijman, T.E., 2008. Longevity risk in portfolios of pension annuities. *Insurance. Mathematics & Economics* 42 (2), 505–519.
- Holzmann, R., Alonso-García, J., Labit-Hardy, H., Villegas, A.M., 2019. NDC schemes and heterogeneity in longevity: proposals for redesign. World Bank.
- Kallestrup-Lamb, M., Kjærgaard, S., Rosenskjold, C.P., 2020. Insight into stagnating life expectancy: analysing cause of death patterns across socio-economic groups. *Health Economics*.
- Keyfitz, N., Caswell, H., 2005. *Applied Mathematical Demography*, vol. 47. Springer.
- Li, N., Lee, R., 2005. Coherent mortality forecasts for a group of populations: an extension of the Lee-Carter method. *Demography* 42 (3), 575–594.
- Liebman, J.B., 2002. Redistribution in the current us social security system. In: *The Distributional Aspects of Social Security and Social Security Reform*. University of Chicago Press, pp. 11–48.
- Medford, A., 2017. Best-practice life expectancy: an extreme value approach. *Demographic Research* 36, 989–1014.
- Milevsky, M.A., 2020. Swimming with wealthy sharks: longevity, volatility and the value of risk pooling. *Journal of Pension Economics & Finance* 19 (2), 217–246.
- OECD, 2015. Ageing and Employment Policies: Denmark 2015. Technical report, OECD Publishing, Paris.
- OECD, 2017. Pensions at a glance 2017. Technical report, OECD Publishing, Paris.
- OECD, 2018a. Key policies to promote longer working lives: Country note 2007 to 2017. Technical report, OECD Publishing, Paris.
- OECD, 2018b. *OECD Pensions Outlook 2018*. Technical report, OECD Publishing, Paris.
- OECD, 2019a. Main economic indicators, Volume 2019 Issue 7. Technical report, OECD Publishing, Paris.
- OECD, 2019b. *OECD Pensions at a Glance 2019*. Technical report, OECD Publishing, Paris.
- Oppen, J., Vaupel, J.W., 2002. Broken limits to life expectancy. *Science* 296 (5570), 1029–1031.
- Pascariu, M.D., Canudas-Romo, V., Vaupel, J.W., 2018. The double-gap life expectancy forecasting model. *Insurance. Mathematics & Economics* 78, 339–350.
- Preston, S., Heuveline, P., Guillot, M., 2000. *Demography: Measuring and Modeling Population Processes*. 2001. Blackwell Publishers, Malden, MA.
- Rau, R., Soroko, E., Jasilionis, D., Vaupel, J.W., 2008. Continued reductions in mortality at advanced ages. *Population and Development Review* 34 (4), 747–768.
- Sánchez-Romero, M., Lee, R.D., Prskawetz, A., 2020. Redistributive effects of different pension systems when longevity varies by socioeconomic status. *The Journal of the Economics of Ageing*, 100259.
- Sanderson, W.C., Scherböv, S., 2010. Remeasuring aging. *Science* 329 (5997), 1287–1288.
- Sanderson, W.C., Scherböv, S., 2017. An easily understood and intergenerationally equitable normal pension age. In: *The Future of Welfare in a Global Europe*. Rutledge, pp. 193–220.
- Sasson, I., 2016. Trends in life expectancy and lifespan variation by educational attainment: United States, 1990–2010. *Demography* 53 (2), 269–293.
- van Raalte, A.A., Sasson, I., Martikainen, P., 2018. The case for monitoring life-span inequality. *Science* 362 (6418), 1002–1004.
- Vaupel, J.W., 1986. How change in age-specific mortality affects life expectancy. *Population Studies* 40, 147–157.
- Vaupel, J.W., Canudas-Romo, V., 2003. Decomposing change in life expectancy: a bouquet of formulas in honor of Nathan Keyfitz's 90th birthday. *Demography* 40 (2), 201–216.

- Villegas, A.M., Haberman, S., 2014. On the modeling and forecasting of socioeconomic mortality differentials: an application to deprivation and mortality in England. *North American Actuarial Journal* 18 (1), 168–193.
- Whitehouse, E., 2007. Life expectancy risk and pensions.
- Willekens, F., 2009. Continuous-time microsimulation in longitudinal analysis. *New Frontiers in Microsimulation Modelling*, 353–376.
- World Bank, 1994. *Adverting the Old Age Crisis: Policies to Protect the Old and Promote Growth*. Oxford University Press.
- Wrycza, T.F., Missov, T.I., Baudisch, A., 2015. Quantifying the shape of aging. *PLoS ONE* 10 (3).
- Zuo, W., Jiang, S., Guo, Z., Feldman, M.W., Tuljapurkar, S., 2018. Advancing front of old-age human survival. *Proceedings of the National Academy of Sciences* 115 (44), 11209–11214.

Progression of the smoking epidemic in high-income regions and its effects on male-female survival differences: a cohort-by-age analysis of 17 countries

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RESEARCH ARTICLE

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Progression of the smoking epidemic in high-income regions and its effects on male-female survival differences: a cohort-by-age analysis of 17 countries

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Abstract

Background: Of all lifestyle behaviours, smoking caused the most deaths in the last century. Because of the time lag between the act of smoking and dying from smoking, and because males generally take up smoking before females do, male and female smoking epidemiology often follows a typical double wave pattern dubbed the ‘smoking epidemic’. How are male and female deaths from this epidemic differentially progressing in high-income regions on a cohort-by-age basis? How have they affected male-female survival differences?

Methods: We used data for the period 1950–2015 from the WHO Mortality Database and the Human Mortality Database on three geographic regions that have progressed most into the smoking epidemic: high-income North America, high-income Europe and high-income Oceania. We examined changes in smoking-attributable mortality fractions as estimated by the Preston-Glei-Wilmoth method by age (ages 50–85) across birth cohorts 1870–1965. We used these to trace sex differences with and without smoking-attributable mortality in period life expectancy between ages 50 and 85.

Results: In all three high-income regions, smoking explained up to 50% of sex differences in period life expectancy between ages 50 and 85 over the study period. These sex differences have declined since at least 1980, driven by smoking-attributable mortality, which tended to decline in males and increase in females overall. Thus, there was a convergence between sexes across recent cohorts. While smoking-attributable mortality was still increasing for older female cohorts, it was declining for females in the more recent cohorts in the US and Europe, as well as for males in all three regions.

Conclusions: The smoking epidemic contributed substantially to the male-female survival gap and to the recent narrowing of that gap in high-income North America, high-income Europe and high-income Oceania. The precipitous decline in smoking-attributable mortality in recent cohorts bodes somewhat hopeful. Yet, smoking-attributable mortality remains high, and therefore cause for concern.

Keywords: Sex differences, Life expectancy, Smoking epidemic, Mortality, Health inequality

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Background

According to the Global Burden of Disease Study, in 2015 worldwide, one out of ten deaths was due to smoking [1]. The same data suggest that smoking is the single most important killer in the world with nearly twice as many victims as the 5% deaths from AIDS, malaria and tuberculosis combined [2]. As such, the smoking epidemic is having a huge impact on the world population and on the individual risk of transitioning to the worst possible health condition, i.e. to die [3].

The enormous increase and subsequent decline in smoking prevalence and later smoking-attributable mortality, and sex differences therein, has been described in detail and termed the ‘smoking epidemic’ [4, 5]. The smoking epidemic model describes that men in high-income countries (particularly the Anglo-Saxon countries) were the first to take up smoking and that smoking-attributable mortality rose some three decades after the rise in smoking prevalence. Women began to smoke later in time than males. Attention for the negative health effects of smoking and associated prevention campaigns led the proportion of males that smoke to decline and the peak in the smoking prevalence among women to be considerably lower than for men. Because of the various time lags, there is a stage where the proportion of males dying from smoking begins to decline, but the proportion of females continues to rise (Fig. 1) [4, 5].

In the 1950s, over 50% of males in the United States (US) were smokers. By 2015, this had changed to less than 20%. For females, these numbers were 24% in the 1950s, versus 12–15% currently [6–11]. For Europe this pattern was similar [7, 8]. Because the percentages of

smokers for males versus females are first divergent, then convergent, in line with the theoretical model outlined above and in Fig. 1, and because high-income countries have progressed furthest into the smoking epidemic, we aimed to chart the progression of deaths from smoking in high-income North-America, high-income Europe and high-income Oceania, as well as the way these deaths have influenced male-female survival differences.

The cohort perspective has been helpful in understanding the unfolding of the smoking epidemic [12]. Also, smoking has been shown to be a significant driver of sex differences in survival [13–18]. Hence, we performed a cohort-by-age analysis of smoking-attributable mortality and investigated its effect on male-female life expectancy differences. We hypothesized that the smoking epidemic may have been the main contributor to the widening and subsequent narrowing of the male-female survival gap in high-income regions in the second half of the twentieth century. We also hypothesized that the cohort perspective could reveal important aspects of the smoking epidemic.

Methods

All-cause death rates and person-years at risk by age, sex, year and country were retrieved from the Human Mortality Database (HMD) [19], which collects these data from national registries worldwide and, after quality control, publishes these in a uniform format [20]. To estimate smoking-attributable mortality (see below), we used lung cancer deaths, defined as malignant neoplasms of trachea, bronchus and lung classified according to the International Statistical Classification of Diseases,

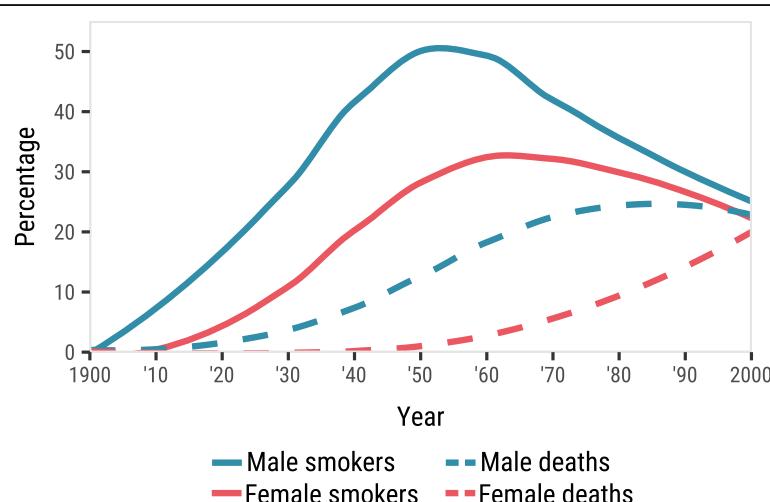


Fig. 1 Schematic diagram of smoking epidemic, after reference 5. Males (in blue) take up smoking (solid line) at a steady pace until smoking-attributable mortality surges (dashed line) and the proportion smoking starts to decline. Females (in red) take up smoking later than males and reach a lower maximum proportion smoking. Smoking-attributable mortality in females is the last to increase to significant proportions. An essential feature of the model is the large time gap between the act of smoking and dying from it

Injuries and Causes of Death versions 7 through 10 (ICD-7: 162, 163; ICD-8 and ICD-9: 162; ICD-10: C33, C34). Death counts from lung cancer were retrieved from the World Health Organization (WHO) Mortality Database [21]. Their cause-of-death statistics come from national vital registration systems. The information is compiled by the national authority and submitted to WHO every year. WHO verifies that the data submitted are coded with the official ICD codes [21].

Mortality data by age (50–85 years) and sex for the populations of 17 high-income countries during the period 1950–2015 were used. The focus on age 50 and over stems from the technique that we used to indirectly estimate smoking-attributable mortality (see below). This method relies on deaths from lung cancer, which are generally rare below age 50 [22]. Furthermore, previous research has shown that most of male-female mortality differences are concentrated between ages 50 and 70 [13].

The method that we used to estimate smoking-attributable mortality, the Preston-Glei-Wilmoth (PGW) method [23] (see below), was based on 20 high-income countries from around the world. We used much the same data set, but we added recent data and excluded three countries. We excluded Iceland because of its geographical location between Europe and North America. We excluded countries that spent much of the 1950–2015 period behind the Iron Curtain, because the countries missed out on the cardiovascular revolution for decades, a phenomenon sometimes called the state socialist syndrome [24]. This led us to exclude Hungary. Japan was excluded because of its atypical smoking history [25]. For Portugal, cause-specific mortality data were not available for the period 2004–2006. We imputed death rates for males and females for Portugal during the period 2004–2006 (3 observations per sex in total, details in Additional file 1) by taking into account all the remaining information about death rates since 1950. We use a non-parametric method based on random forest algorithms to perform the imputation (“missForest” R package, version 1.4 [26]). The 17 remaining countries were grouped into three geographical categories: high-income North America (US and Canada), high-income Europe (Austria, Belgium, Denmark, Finland, France, Italy, the Netherlands, Norway, Portugal, Spain, Sweden, Switzerland and the UK), and high-income Oceania (Australia and New Zealand).

The Preston-Glei-Wilmoth (PGW) method [23] was used to appraise the proportion of deaths attributable to smoking. This method assumes that “after adjusting for sex and age, smoking is the only source of variation in lung cancer death rates in the populations under consideration” [23]. It estimates smoking attributable deaths indirectly by assuming that lung cancer rates of smokers

and never-smokers match those observed among individuals in the Cancer Prevention Study II in the US. The model then uses negative binomial regression to model smoking attributable mortality from causes other than lung cancer as a function of lung cancer mortality [23]. An analysis of deviance to assess the model fit is included in the Additional file 1.

We then smoothed smoking-attributable mortality estimates with the penalized composite link model [27], giving year-by-age estimates of the proportions of death due to smoking for all 17 countries for males and females over age 50. We used such year-by-age estimates to construct birth-cohorts’ mortality profiles between 1870 and 1965 for high-income North America, high-income Europe and high-income Oceania [28]. Thus we obtained the proportion of smoking-attributable mortality by sex, cohort and age (cohorts 1870–1965).

To shed light on how smoking-attributable mortality affected period life expectancy, the way in which the sex gap in survival is often analyzed [13], we calculated period life expectancy between ages 50 and 85 ($e_{50 \mid 85}$, the average number of years lived between ages 50 and 85) over the period 1950–2015 following standard demographic procedures [29]. This measure is defined as follows:

$$e_{50|85} = \frac{\sum_{50}^{85} l(x)}{l(50)},$$

where $l(x)$ denotes number of survivors at age x . We applied the life table calculations to all-cause mortality rates (including smoking-attributable mortality) and to non-smoking-attributable mortality rates (excluding smoking-attributable mortality) for the three regions and the different years. From those results we calculated male-female $e_{50 \mid 85}$ differences for all regions and periods.

All analyses were run on R version 3.6.

Results

Between 1950 and 2015, the smoking epidemic caused a total of 39 million deaths at ages 50–85 in the three high-income geographical locations. Of these, 29 million deaths were men and 10 million were women. The largest numbers of deaths attributable to smoking were in high-income North America with 13 million men and 7 million women, followed by high-income Europe with 15 million and 3 million, respectively. In high-income Oceania these numbers were 0.7 million and 0.2 million.

For males in high-income Europe, North America and Oceania, we found a steep increase in the proportion of smoking-attributable mortality from the cohorts born

1870 up to about 1900–1910, when smoking-attributable mortality was the highest (Fig. 2). For high-income North America and Oceania, after reaching a peak for the 1910–1930 cohorts, there was a large drop in the proportion of smoking-attributable mortality in more recent cohorts. For Europe, the upsurge was followed by a stagnation period up to the most recent cohorts, where a steep drop was seen.

For females in high-income North America, the upsurge in smoking-attributable mortality was delayed by about 30 years relative to males (Fig. 2). At the highest ages, which are necessarily older cohorts, the peak in the proportion of smoking-attributable mortality does not seem to have been reached as yet, although some indication exists. For the younger ages, the peak was reached for the 1930 cohorts, with a steep decline after, interrupted only by the 1950 cohorts. For European females, smoking-attributable mortality increased less steeply and peaked later and lower than for North-American females. Any decline in smoking-attributable mortality in European females is seen in the most recent cohorts only, necessarily at younger ages. At higher ages, the proportion of smoking-attributable mortality is still on the increase. For Oceania, the pattern was similar to that of the Europe, but without any significant drop to date.

In terms of absolute levels of smoking-attributable mortality (rather than trends), smoking-attributable

mortality was higher in males than for females in Europe for all age groups and all cohorts, even though for recent cohorts the absolute differences were small (Fig. 2). For the US and Oceania, smoking-attributable mortality was higher for males than for females for most cohorts and age groups, in particular those that drove recent changes. However, for recent cohorts at relatively young ages, smoking-attributable mortality was similar between sexes, even slightly higher for females than for males.

We produced an alternative version of Fig. 2 with age on the horizontal axis and each birth cohort represented by a line (Additional file 1: Figure S3), providing a complementary perspective of the smoking epidemic in the high-income regions.

The effect of removing smoking-attributable mortality on $e_{50 \mid 85}$ was similar for the three regions. For males, it increased gradually for the years ~ 1950–1970, to up to ~ 2 years of partial life expectancy (Fig. 3, top panels). This was particularly pronounced for Belgium, The Netherlands and the UK, while the increase was smallest for Sweden (Additional file 1: Figure S4). In the following years the effect of removing smoking-attributable mortality on $e_{50 \mid 85}$ remained more or less constant for some decades until approximately 1990. Afterward it declined to ~ 1 year for recent years. For females, the effect of removing smoking-attributable mortality on $e_{50 \mid 85}$ was negligible for the years 1950–1970. Afterwards it

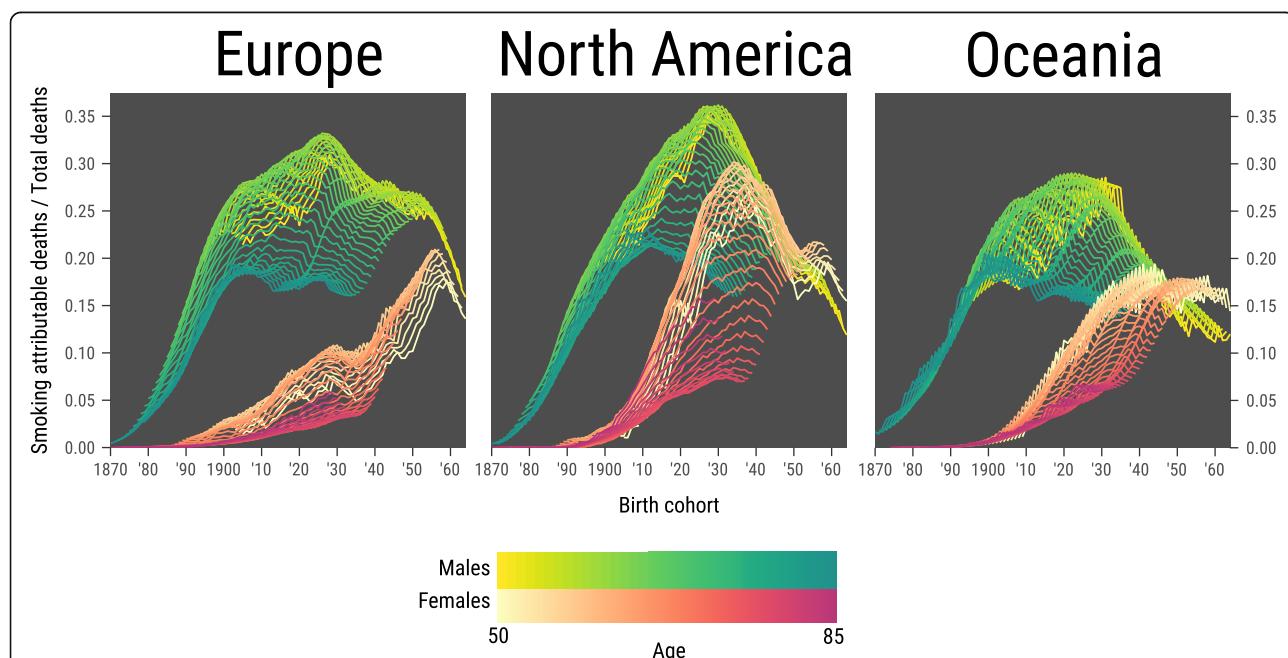


Fig. 2 Cohort-by-age analysis of the proportion of overall mortality that is attributed to smoking. Each birth cohort is on a single vertical line. For males, ages are shaded from yellow (age 50) to turquoise (age 85). For females, ages are shaded from beige (age 50) to fuchsia (age 85). The more recent a cohort, the smaller the number of age groups for which data are available (recent cohorts have not yet reached the higher ages). Results given for high-income Europe (13 countries), high-income North America (2 countries) and high-income Oceania (2 countries)

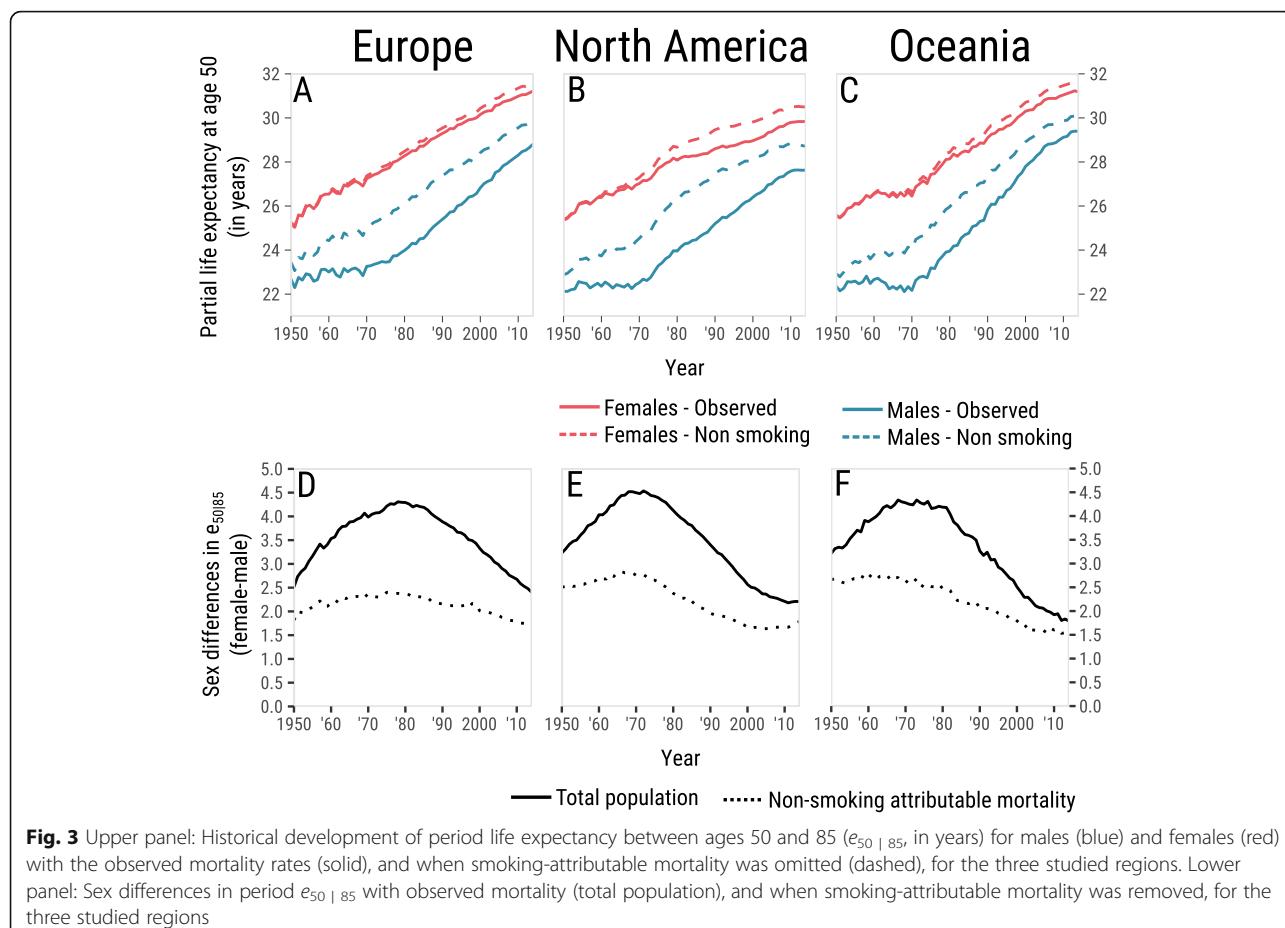


Fig. 3 Upper panel: Historical development of period life expectancy between ages 50 and 85 ($e_{50|85}$, in years) for males (blue) and females (red) with the observed mortality rates (solid), and when smoking-attributable mortality was omitted (dashed), for the three studied regions. Lower panel: Sex differences in period $e_{50|85}$ with observed mortality (total population), and when smoking-attributable mortality was removed, for the three studied regions

grew slowly but steadily to ~0.5 year. This was particularly pronounced in the US, but less so in Europe and Oceania.

Globally, over all three regions, the sex gap in $e_{50|85}$ was approximately 3 years in 1950, then increased to some 4.5 years around 1975, and afterwards decreased towards 2 years (Fig. 3, bottom panels). Omitting smoking-attributable deaths, the male-female difference in $e_{50|85}$ would have been approximately 2 years lower at its peak and much more constant over time than with smoking included (Fig. 3, bottom panels).

The contribution of smoking to male-female differences in life expectancy is now on the decline (Figs. 2 and 3). In some countries (Italy, New Zealand, Finland, Spain), this is due to declining smoking attributable mortality in males. In other countries (Sweden, Norway, Iceland), this is mainly due to increasing smoking-attributable mortality in females. Finally, there are countries (Canada, Austria, UK, US, Netherlands, Australia) where the decline is caused by an approximately equal contribution to each side of the $e_{50|85}$ gap (Additional file 1: Figure S5).

Discussion

Our study advanced three main results. First, in all three high-income regions, smoking explained up to 50% of sex differences in period life expectancy between age 50 and 85 over the study period 1950–2015. Second, the decline in these sex differences since approximately 1980 is largely driven by smoking-attributable mortality. Third, whereas smoking-attributable mortality is still increasing for many older female cohorts, it is declining for females in the more recent cohorts in the US and Europe, as well as for males in all three regions.

The massive impact of smoking on mortality is in line with previous studies addressing smoking effects on mortality at the population level; it has been found for the United States [14], in European countries [12, 15, 30–35], and worldwide [36]. Smoking affects various causes of death, such as various forms of cancer, cardiovascular disease and multifarious diseases of the respiratory tract [37, 38]. Smoking also explains important differences in life expectancy between countries [39]. Finally, the historical trajectories of divergence between life expectancy with and without smoking-attributable

mortality that we found are broadly similar to those previously found for specific countries [40].

Insights into the smoking epidemic across cohorts

Our cohort-by-age analysis of high-income regions confirmed the mortality element of the smoking epidemic model [4, 5]: the increase in smoking-attributable mortality started later among females than for males, and resulted in a later peak at a lower level. Without constructing cohort profiles, we would not have been able to trace two additional important regularities of the smoking epidemic. First, while smoking-attributable mortality in older cohorts still increased, a precipitous decline in smoking-attributable mortality took place in recent cohorts at younger ages. Second, smoking-attributable mortality for males versus females converged across cohorts.

The continuous increase in smoking-attributable mortality in older female cohorts remains cause for concern. It is an essential feature of the smoking epidemic, though, that these deaths result from the high smoking prevalence of women decades ago. More encouraging is the decline in smoking-attributable mortality in recent cohorts at younger ages. Also, smoking prevalence has generally come down over the last decades in the studied regions. For example, in Australia smoking prevalence in females aged 15+ came down from 22.0% in 2000 to 12.4% in 2015 (for males, these numbers were 27 and 14.3%, respectively) [41].

In the same vein, current sex-specific smoking prevalence gives some indication of future sex-specific smoking attributable mortality. In 2015 smoking prevalence generally remained higher in males than in females. For example, in 2015 32% of French men smoked, versus 22% of females; in Germany, 25% of males smoked versus 17% of females; while in the US 14% of males smoked versus 12% of females [7]. Consequently, male smoking-attributable mortality is likely to remain (or become again) higher than female smoking-attributable mortality.

The smoking epidemic and the sex gap in life expectancy

In 1950 the sex gap in period $e_{50 \mid 85}$ was 2.0–3.5 years. It subsequently grew to 4.3–4.5 years at the maximum around 1970–1980, and then decreased to 1.8–2.5 years in 2015 for the three regions. The rise, stagnation and decline in the sex differences in survival has been described in detail elsewhere [13, 16–18]. Smoking behaviour has been found to explain international differences in the life expectancy sex gap [42–44]. We here show that across high-income regions, almost no increase in the sex gap would have occurred without smoking-attributable mortality. Smoking-attributable mortality

caused almost all the increase and most of the decrease in the sex gap over the study period.

Although the major part of the decline in the sex gap hitherto is caused by the steep drop in smoking-attributable mortality in males, more and more so this is also due to the increases in smoking-attributable mortality in females overall. For all-causes mortality, in contrast, it has been found that a reduction in the male-female life expectancy gap is, for most countries, due to men dying at lower rates, rather than women at higher rates [18, 45].

We suggest that we may not have seen the end of the narrowing in $e_{50 \mid 85}$ sex differences in these regions yet. To date, male smoking-attributable mortality generally still exceeds that of females. Meanwhile, trends are downward for males generally, while for females they are upwards for older cohorts. This suggests scope for further narrowing sex differences in $e_{50 \mid 85}$ in these regions. However, the extent to which this may happen seems limited because male smoking prevalence generally remains higher nowadays than female smoking prevalence (see above). Of course, smoking-attributable mortality is not the only factor that affects the sex gap, and there is evidence that mortality from some causes other than smoking may currently be widening the gap [16]. Still, smoking-attributable mortality could overwhelm the effect of mortality from other causes on the sex gap. This could happen especially in countries with a high proportion of women taking up smoking some decades ago, where smoking-attributable mortality for men and women could potentially cross over (e.g. U.K., Denmark and the Netherlands) [8], as we have found for the most recent cohorts in the US and Oceania.

Limitations

One clear limitation of our study is the indirect calculation of the smoking-attributable deaths. Such a limitation is unavoidable: comparisons between different methods to estimate smoking-attributable mortality did not reveal a best-practice method [46, 47], and even if good estimates of smoking prevalence are available to potentially directly estimate smoking-attributable mortality, other factors like smoking intensity are often harder to measure and to take into account in direct estimates.

Since the PGW method [23] extrapolates the lung cancer rates of non-smokers from a US study to other countries, there may be a bias in our estimates for those other countries. Also, the PGW is based on study participants that are more likely than the US overall population to be Caucasian and middle class, and to have achieved a relatively high level of education [47]. However, previous analyses have shown that the indirect estimation by PGW resulted in roughly similar

outcomes compared to other indirect estimation techniques [30, 31, 48], so we are confident that our results are broadly reliable. Results obtained making a modification to the PGW method proposed by Rostron [49], discussed in [50], are included in the Additional file 1. Making this modification would not have affected our main conclusion.

As a final limitation, our estimates of smoking-attributable mortality were smoothed over ages, which may lead to minor distortions. We do not expect that to be the case here due to relatively regular source data.

Conclusion

While a sharp decrease in recent cohorts is a strong positive development, smoking-attributable mortality for both sexes has converged across cohorts to a level that remains high. Males and females dying of smoking at the same rate is not an end to the smoking epidemic. In high-income regions, smoking remains a major killer.

Supplementary information

Supplementary information accompanies this paper at <https://doi.org/10.1186/s12889-020-8148-4>.

Additional file 1. Appendix.

Abbreviations

HMD: Human Mortality Database; ICD: International Statistical Classification of Diseases, Injuries and Causes of Death; PGW: Preston, Glei, Wilmoth; WHO: World Health Organization

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Authors' contributions

MW drafted the paper with help from JA, FJ and RL. JA and SR performed data analysis. RL conceived of the study. All authors critically interpreted and discussed the results. All authors read and approved the manuscript.

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Availability of data and materials

All data are publicly available from the Human Mortality Database (www.mortality.org) and the WHO Mortality Database (http://www.who.int/healthinfo/mortality_data/en/).

Ethics approval and consent to participate

All data are publicly available on an aggregate basis. Consequently, no ethics approval was necessary.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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References

1. GBD 2015 Tobacco Collaborators MB, Fullman N, Ng M, et al. Smoking prevalence and attributable disease burden in 195 countries and territories, 1990–2015: a systematic analysis from the Global Burden of Disease Study 2015. *Lancet*. 2017;389:1885–906.
2. Wang H, Naghavi M, Allen C, et al. Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980–2015: a systematic analysis for the global burden of disease study 2015. *Lancet*. 2016;388:1459–544.
3. World Health Organization. WHO report on the global tobacco epidemic, 2017: monitoring tobacco use and prevention policies. https://www.who.int/tobacco/global_report/2017/en/. Accessed 26 Jan 2019.
4. Lopez AD, Collishaw NE, Piha T. A descriptive model of the cigarette epidemic in developed countries. *Tob Control*. 1994;3:242–7.
5. Thun M, Peto R, Boreham J, Lopez AD. Stages of the cigarette epidemic on entering its second century. *Tob Control*. 2012;21:96–101.
6. Brandt AM. The cigarette century: the rise, fall, and deadly persistence of the product that defined America. New York: Basic Books; 2007.
7. tobaccoatlas.org, respective country information sheets, Accessed 1 Nov 2019.
8. Graham H. Smoking prevalence among women in the European Community 1950–1990. *Soc Sci Med*. 1996;43:243–54.
9. Centers for Disease Control and Prevention (CDC). Tobacco Use — United States, 1900–1999. *Morb Mortal Wkly Rep*. 1999;48:986–93.
10. United States Public Health Service. Office of the Surgeon General, Office on Smoking and Health. The health consequences of smoking: a report of the Surgeon General. 2004.
11. World Bank Open Data. 2017. <https://data.worldbank.org/indicator/SH.PRV.SMOK.FE>. Accessed 15 Mar 2018.
12. Lindahl-Jacobsen R, Oeppen J, Rizzi S, Möller S, Zarulli V, Christensen K, Vaupel JW. Why did Danish women's life expectancy stagnate? The influence of interwar generations' smoking behaviour. *Eur J Epidemiol*. 2016; 31:1207–11.
13. Beltrán-Sánchez H, Finch CE, Crimmins EM. Twentieth century surge of excess adult male mortality. *Proc Natl Acad Sci*. 2015;112:8993–8.
14. Preston HP, Wang H. Sex mortality differences in the United States: the role of cohort smoking patterns. *Demography*. 2006;43:631–46.
15. Lew EA, Garfinkel L. Differences in mortality and longevity by sex, smoking habits and health status. *Transac Soc Actuaries*. 1987;39:107–30.
16. Pampel FC. Cigarette use and the narrowing sex differential in mortality. *Popul Dev Rev*. 2002;28:77–104.
17. Thorslund M, Wastesson JW, Agahi N, Lagergren M, Parker MG. The rise and fall of women's advantage: a comparison of national trends in life expectancy at age 65 years. *Eur J Ageing*. 2013;10:271–7.
18. Meslé F. Life expectancy: a female advantage under threat? *Popul Soc*. 2004; 40:2:1–4.
19. Human Mortality Database. <https://www.mortality.org/>. Accessed 20 Sept 2018.
20. Barbieri M, Wilmoth JR, Shkolnikov VM, Glei D, Jasillionis D, Jdanov D, Boe C, Riffe T, Grigoriev P, Winant C. Data resource profile: the human mortality database (HMD). *Int J Epidemiol*. 2015;44:1549–56.
21. World Health Organization. Health statistics and information systems 2018 http://www.who.int/healthinfo/mortality_data/en/. Accessed 20 Sept 2018.
22. National Cancer Institute. SEER: Cancer statistics and facts: long and bronchus cancer. <https://seer.cancer.gov/statfacts/html/lungb.html>. Last accessed 25 Mar 2019.
23. Preston SH, Glei DA, Wilmoth JR. A new method for estimating smoking-attributable mortality in high-income countries. *Int J Epidemiol*. 2010;39: 430–8.

24. Józán P. Crisis and renewal in epidemiological development after world war II in Hungary. Budapest: MTA Társadalomkutató Központ; 2008.
25. Honjo K, Kawachi I. Effects of market liberalisation on smoking in Japan. *Tob Control*. 2000;9:193–200.
26. Stekhoven DJ, Bühlmann P. MissForest—non-parametric missing value imputation for mixed-type data. *Bioinformatics*. 2011;28:112–8.
27. Rizzi S, Gampe J, Eilers P. Efficient estimation of smooth distributions from coarsely grouped data. *Am J Epidemiol*. 2015;182:138–47.
28. Carstensen B. Age-period-cohort models for the Lexis diagram. *Stat Med*. 2007;26:3018–45.
29. Preston SH, Heuveline P, Guillot M. Demography. Measuring and modeling population processes. Oxford: Blackwell Publishers; 2001.
30. Stoeldraijer L, Bonneux L, van Duijn C, van Wissen LJG, Janssen F. The future of smoking-attributable mortality: the case of England & Wales, Denmark and the Netherlands. *Addiction*. 2015;110:336–45.
31. Janssen F, van Wissen LJG, Kunst AE. Including the smoking epidemic in internationally coherent mortality projections. *Demography*. 2013;50:1341–62.
32. Lindahl-Jacobsen R, Rau R, Jeune B, et al. Rise, stagnation, and rise of Danish women's life expectancy. *Proc Natl Acad Sci*. 2016;113:4015–20.
33. Jacobsen R, Keiding N, Lynge E. Long term mortality trends behind low life expectancy of Danish women. *J Epidemiol Community Health*. 2002;56:205–8.
34. Jacobsen R, Von Euler M, Osler M, Lynge E, Keiding N. Women's death in Scandinavia - what makes Denmark different? *Eur J Epidemiol*. 2004;19:117–21.
35. Jacobsen R, Keiding N, Lynge E. Causes of death behind low life expectancy of Danish women. *Scand J Public Health*. 2006;34:432–6.
36. Rentería E, Jha P, Forman D, Soerjomataram I. The impact of cigarette smoking on life expectancy between 1980 and 2010: a global perspective. *Tob Control*. 2016;25:551–7.
37. WHO, Cancer IAfRo. Personal Habits and Indoor Combustions Volume 100e A Review Of Human Carcinogens. IARC Monogr Eval Carcinog Risks Hum. 2012;100:1–441.
38. Aburto JM, Wensink M, van Raalte A, Lindahl-Jacobsen R. Potential gains in life expectancy by reducing inequality of lifespans in Denmark: An international comparison and cause-of-death analysis. *BMC Public Health*. 2018;18:831.
39. Janssen F. (provisionally accepted) The role of smoking in country differences in life expectancy across Europe, 1985–2014. *Nicotine Tob Res*.
40. Janssen F, Rousson V, Paccaud F. The role of smoking in changes in the survival curve: an empirical study in 10 European countries. *Ann Epidemiol*. 2015;25:243–9.
41. WHO (2015) Global report on trends in tobacco smoking 2000–2025 – First edition.
42. Bobak M. Relative and absolute gender gap in all-cause mortality in Europe and the contribution of smoking. *Eur J Epidemiol*. 2003;18:15–8.
43. Janssen F. (forthcoming) Changing contribution of smoking to the sex differences in life expectancy in Europe. *Eur J Epidemiol*.
44. Janssen F. Similarities and Differences Between Sexes and Countries in the Mortality Imprint of the Smoking Epidemic in 34 Low-Mortality Countries, 1950–2014. *Nicotine Tob Res*. 2019;ntz154. <https://doi.org/10.1093/ntr/ntz154>.
45. Meinow B, Parker M, Thorslund M. Complex health problems and mortality among the oldest old in Sweden: decreased risk for men between 1992 and 2002. *Eur J Ageing*. 2010;7:81–90.
46. Pérez-Ríos M, Montes A. Methodologies used to estimate tobacco-attributable mortality: a review. *BMC Public Health*. 2008;8:22.
47. Tachfouti N, Raherison C, Obtel M, Nejjari C. Mortality attributable to tobacco: review of different methods. *Arch Public Health*. 2014;72:22.
48. Martikainen P, Mäkelä P, Peltonen R, Myrskylä M. Income differences in life expectancy: the changing contribution of harmful consumption of alcohol and smoking. *Epidemiology*. 2014;25:182–90.
49. Rostron B. A modified new method for estimating smoking-attributable mortality in high-income countries. *Demogr Res*. 2010;23:399–420.
50. Fenelon A, Preston SH. Estimating smoking-attributable mortality in the United States. *Demography*. 2012;49:797–818.

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Supplemental Material

Missing data

We report (our handling of) missing data according to the guidelines set out by Sterne J et al. Multiple Imputation for Missing Data in Epidemiological and Clinical Research: Potential and Pitfalls. BMJ, 338, b2393 2009 Jun 29. PMID: 19564179.

Missing item: *cause-specific death rates, 3 years for each sex, Portugal*. The reason for missingness was that Portugal used a special classification system during that period, which cannot be reliably translated to ICD-10. Given this known reason, missingness was unlikely to be related to the number of lung cancer deaths in these years. Overall death rates (which are known) did not deviate from the trend in the years in which cause-of-death information was missing. Variables used in imputation where the cause-specific death rates for Portugal in the available years. Thus, we assumed that the time trend in the years 2004-2006 was a smooth continuation of the time trend before and after 2004-2006, and so that the cause-specific death rates for those years were missing completely at random. Population size of Portugal is small compared with the rest of high-income Europe and we imputed for three years only, so that this imputation in no way affects our overall conclusions (which take a large perspective). The missForest algorithm took 10 iterations until convergence.

Model fit

To assess model fit, we performed an analysis of deviance and visually inspected the residuals. For females, the PGW model had a null deviance of 1,768,593 on 11,103 degrees of freedom versus a residual deviance of 11,095 on 10,980 degrees of freedom. This gives a p-value for the model of $p < 10^{-20}$ and a Chi-squared goodness-of-fit test p-value of 0.22. All parameters were significant ($p < 10^{-7}$). For males, the PGW model had a null deviance of 1,587,178 on 11,103 degrees of freedom versus a residual deviance of 11,218 on 10,980 degrees of freedom. This gives a p-value for the model of $p < 10^{-20}$ and a Chi-squared goodness-of-fit test p-value of 0.055. All parameters were significant ($p < 10^{-7}$). The residuals were symmetric (Figures S1 and S2).

Histogram of residuals of the PGW model for females

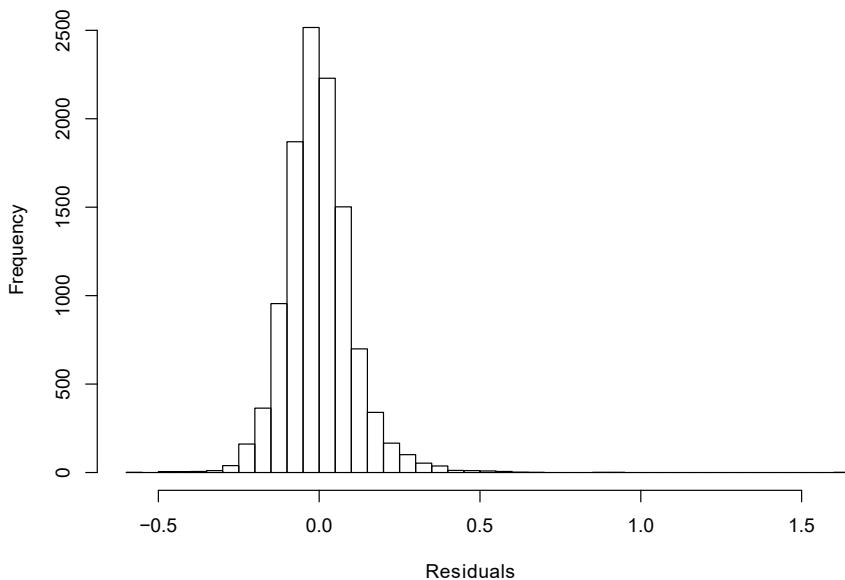


Figure S1. Histogram of residuals of the Preston-Glei-Wilmoth model. Females.

Histogram of residuals of the PGW model for Males

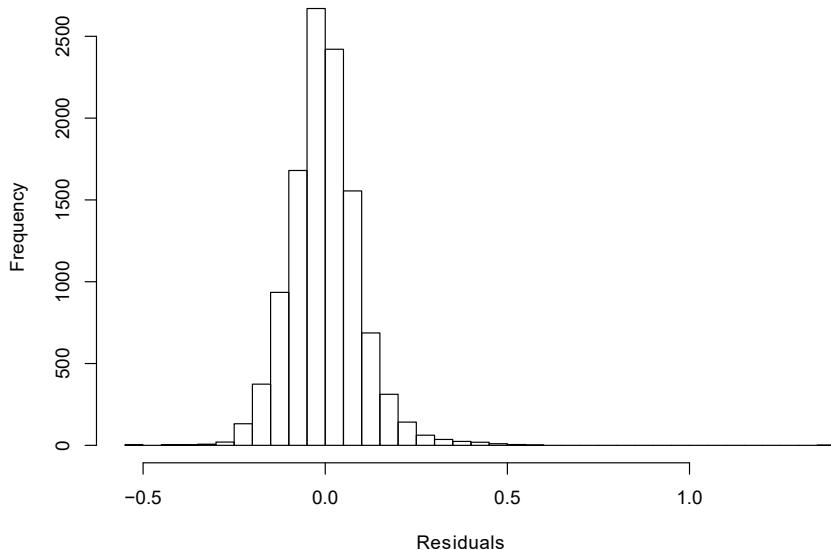


Figure S2. Histogram of residuals of the Preston-Glei-Wilmoth model. Males.

Alternative Figure 2

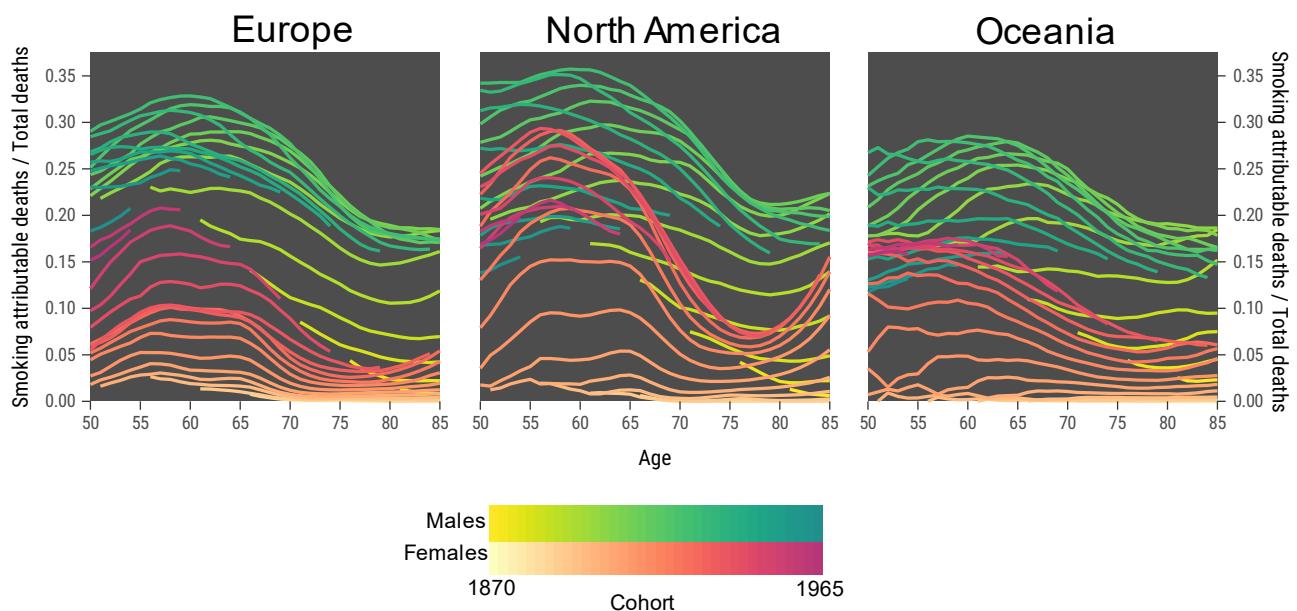


Figure S3. Proportion of overall mortality that is attributed to smoking. This is an alternative representation of the results shown in Figure 2. Each birth cohort is on a single vertical line. For males, cohorts are shaded from yellow (earlier cohorts) to turquoise (younger cohorts). For females, cohorts are shaded from beige (earlier cohorts) to fuchsia (younger cohorts). Results given for high-income Europe (13 countries), high-income North America (2 countries) and high-income Oceania (2 countries).

Per-country trends

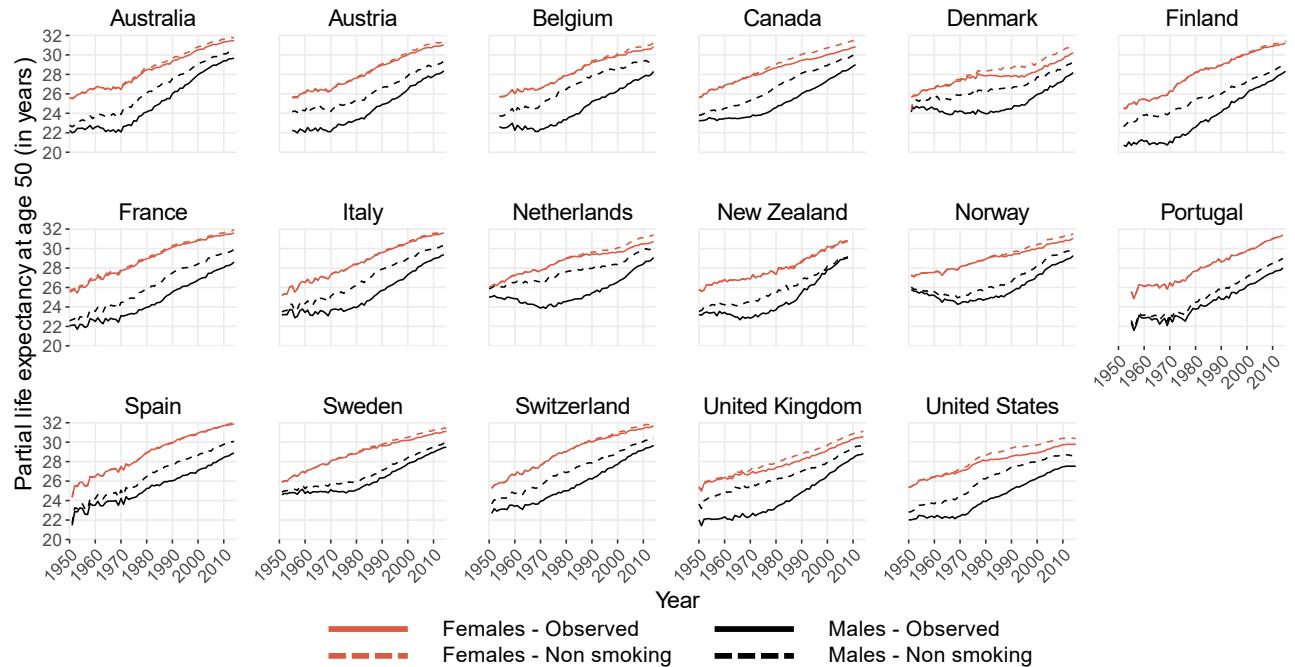


Figure S4. Historical development of period partial life expectancy between ages 50 and 85 ($e_{50|85}$, in years) for males (in black) and females (in red) with the observed mortality rates (solid), and when smoking-attributable mortality was omitted (dashed). Results shown for individual countries

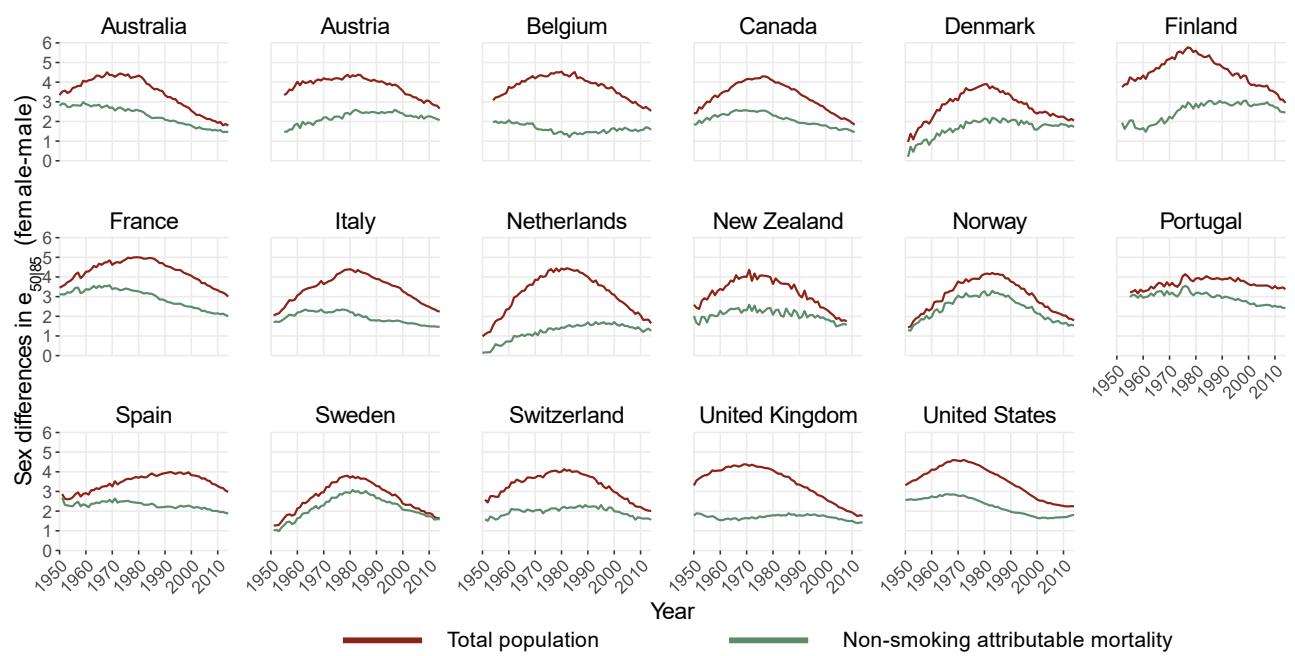


Figure S5. Sex differences in period $e_{50|85}$ with observed mortality (total population), and when smoking-attributable mortality was removed. Results shown for individual countries.

Age-period interaction

The PGW method has been criticized for producing results that are different from earlier results produced by the Peto-Lopez method [49 of main text], especially for older females. The modification of the PGW method by Rostron produced lower smoking-attributable mortality for ages 80 and over, which were more in line with earlier results [49 of main text]. On the other hand, the reason for introducing the PGW method was that the authors felt that the Peto-Lopez method unduly relied on relative risk estimates [23, 50 of main text]. Differing from earlier results is therefore not necessarily a weakness of the method. Both methods produce estimates, not truth, but our estimates certainly seem reasonable. Because the same method has been used over time, the time trends are most likely less affected than the levels themselves. In our analysis, the modification proposed by Rostron led to lower results for higher ages for both sexes (Figure S6 below). This would not have changed the main conclusions of our study.

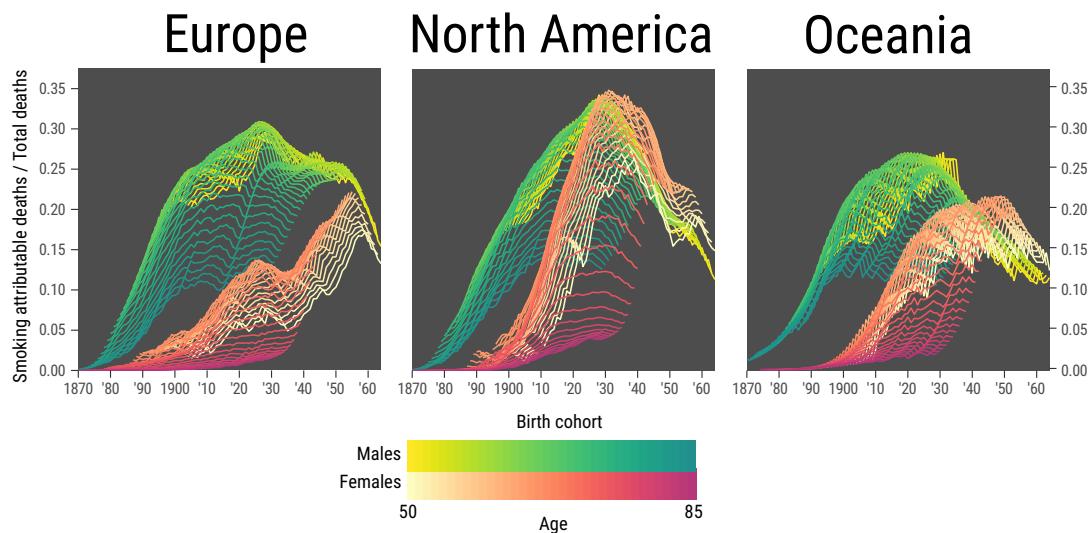


Figure S6. Similar to Figure 2 in the main text, but with a modification proposed by Rostron (ref 49 of the main text). An interaction term is added between age groups and time to the regression equation used by PGW (ref 23 of the main text).

Mortality as a function of survivorship

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Chapter 6

Mortality as a function of survivorship

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Abstract

Everyone has a chronological age. Because survivorship declines relentlessly in populations with age-specific death rates greater than zero, everyone also has a survivorship age (s-age), the age at which a proportion s of the population is still alive. S-ages can be estimated both for periods and cohorts. While trajectories of mortality over chronological ages (e.g., across populations, over time, by sex or by any subpopulation), differ, mortality trajectories over s-ages are remarkably similar, a sign that populations experience similar mortality dynamics at specific levels of survivorship. We show that this important demographic regularity holds for 23 sex-specific populations analyzed during a period comprising more than 100 years.

Keywords: survivorship age, chronological age, risk of dying, postponement.

6.1 Introduction

Empirical research supports the view that human mortality is being postponed to later ages (Bongaarts and Feeney, 2002; Canudas-Romo, 2008; Kannisto, 2001; Vaupel, 2010; Vaupel and Gowan, 1986; Wilmoth and Horiuchi, 1999). Popular sayings like 60s are the new 50s reflect the change in the relationship between mortality and the age variable¹ (Burger et al., 2012). Mortality postponement can be observed through changes in demographic indicators such as the mortality hazard, $\mu(x)$, survival function, $S(x)$, and density function, $f(x)$, occurring at any given chronological age x . Each of these indicators describes a specific characteristic of the mortality regime in a population. These indicators are interrelated, and one can be expressed in terms of the other one². However, it is not clear how the postponement of mortality to older ages affects the relationship between these demographic indicators. For example, according to the

¹We refer to chronological age simply as ages. Later in the manuscript, we introduce the concept of survivorship ages and we refer to them as s-ages.

²for example, $\mu(x) = \frac{f(x)}{S(x)}$, $S(x) = e^{\int_0^x \mu(a)da}$, and $f(x) = -\frac{dS(x)}{dx}$.

1910 life table for Swedish females (Human Mortality Database, 2021), the survival function was 0.90 at age 5 (i.e. $S^{1910}(5) = 0.90$). In 2019, the survival function was 0.90 at age 70 (i.e. $S^{2019}(70) = 0.90$). Given that the values for $S^{1910}(5)$ and $S^{2019}(70)$ are identical, can we expect that $\mu^{1910}(5)$ and $\mu^{2019}(70)$ are also similar?

Two contrasting scenarios might arise if one controls the effect of postponement in mortality towards older ages. In the first scenario, a steady relationship between the survival function and the mortality hazard might arise. In this case, specific values of $S(x)$, will always result in the same values for $\mu(x)$, regardless of the age x at which these two indicators are calculated. In other words, this scenario indicates that a certain proportion of individuals would experience the same risk of dying regardless of the age at which those individuals are still alive. Under this scenario, we can frame Vaupel (2010) hypothesis that all older humans have a similar rate of ageing³.

In the second scenario, specific values of $S(x)$ would yield to different values $\mu(x)$ depending on the age x at which these indicators are calculated. This irregular relationship between the survival function and the mortality hazard would indicate that mortality is an unstable and non-deterministic process. In the case of finding empirical evidence to support this second scenario, a redefinition of theories of senescence (Baudisch and Vaupel, 2012; Colchero et al., 2021, 2016; Le Bourg, 2001; Omholt and Kirkwood, 2021; Vaupel, 2010; Wachter et al., 2014) and a reshape of demographic thinking would be necessary. Hence, it is essential to examine changes in the relationship between survival and the risk of dying in order to unravel the demographic mechanisms behind the dynamics of human mortality. Thus, the question arises as to how this relationship can be examined without the influence of mortality postponement to older ages?

The ground-breaking article of Zuo et al. (2018) sheds light on this issue. By analyzing percentiles⁴ in the distribution of deaths after age 65 they show that the old-age deaths follow an advancing front, like a traveling wave. They show that: i) deaths occurring after the first quartile have been shifting towards older ages at a similar pace since the year 1950, and ii) the distance between age 65 and the first quartile has increased, whereas the distance between upper percentiles has remained constant over time. Zuo et al. (2018) findings are highly relevant because they overrule the long-lasting debate about deaths being compressed or dispersed with age (Bergeron-Boucher et al., 2015; Kannisto, 2001; Myers and Manton, 1984; Nusselder and Mackenbach, 1996; Thatcher et al., 2010; Wilmoth and Horiuchi, 1999). Indeed, conclusions about compression or dispersion of deaths are heavily driven by the use of chronological ages as the time variable⁵.

Nonetheless, the choice of Zuo et al. (2018) to start their analysis at age 65 is debatable. The selection of the onset age neglects what takes place at younger ages. It is likely that steady shifts in death percentiles start well before age 65. Indeed, it has been shown that major reductions in death rates have taken place at younger ages (Beltrán-Sánchez and Subramanian, 2019; Bergeron-Boucher et al., 2015). Young-ages and old-ages are both part of the same continu-

³This is sometimes called the *b-hypothesis*, in reference to the *b* parameter of the Gompertz distribution. The *b*-hypothesis refers to a constant rate of ageing at individual level. However, *b* can only be estimated by parametric models that account for unobserved heterogeneity (Vaupel et al., 1979). The estimation of the *b* parameter for the standard individual is beyond the scope of this paper.

⁴ages by which p% of individuals would die.

⁵this has been pointed out by Wilmoth and Horiuchi (1999), Canudas-Romo (2008), Beltrán-Sánchez and Subramanian (2019) and many others. However, Zuo et al. (2018), provide compelling evidence about this issue.

ous process of ageing. Therefore, truncating at chronological ages could distort the signal and trigger misleading dynamics of demographic indicators.

In this article, we analyze the dynamics of the risk of dying after controlling for mortality postponement towards older ages. To do so, we develop a framework to study the risk of dying in terms of survivorship ages (or s-ages) and examine sex-specific mortality dynamics in 23 populations from 1900 to 2018. We start our analysis at birth to portray the entire and continuous process of human ageing. Extensive literature has shown that, when mortality trajectories over chronological ages are compared (e.g., across populations, over time, by sex or by any subpopulation), the curves differ. In this article, we show that, when such mortality trajectories over s-ages are compared, they are similar. The key contribution of this article is in showing that, over time, what has changed is the relationship between the age variable and human survival, but the relationship between survival and the risk of dying is regular.

6.2 Survivorship ages

Survival is customarily seen as a function of age x . The survivorship function $S(x)$ gives the proportion of a cohort still alive at age x . If the force of mortality (i.e. hazard of death or risk of dying) is positive at all ages, then $S(x)$ is a monotonically decreasing function of x and at every age there is a unique value of s . Consequently, chronological age x can be seen as a function of survival s . Then $x(s)$ is the survivorship age or *s-age* such that $x(s) := S^{-1}(x)$. Note that from this perspective $x(s)$ is a function and not a scalar value, and s is a scalar value and not a function. Instead of taking chronological age as what varies over a lifetime, survival age is what varies, and chronological age is a function of it⁶.

Let us denote the negative derivative of $x(s)$ with respect to s by:

$$\psi(s) = -\frac{dx(s)}{ds}. \quad (6.1)$$

By definition, $\psi(s)$ is the quantile density function of s (Gilchrist, 2000; Nair and Sankaran, 2009; Nair et al., 2013). Then, it can be shown that the mortality hazard, $\mu(s)$, calculated at survival level s can be expressed as:

$$\mu(s) = \frac{1}{s \psi(s)}. \quad (6.2)$$

Equation (6.2) is crucial in our study because it links the risk of dying and the survival function without the influence of the chronological age as a time-scale.

⁶The link between chronological ages and the survival function could have also been done in terms of the cumulative distribution function, $F(x)$ and the results would have been identical because $S(x) = 1 - F(x)$. Indeed, the use of $F(x)$ leads to percentiles of the distribution of deaths used in Beltrán-Sánchez and Subramanian (2019); Wilmoth and Horiuchi (1999); Zuo et al. (2018). In this study we chose to use $S(x)$ instead of $F(x)$ because it is more intuitive to think about changes in the mortality as the population extinguishes (i.e. s goes from 1 to 0) rather than deaths accumulating.

Dynamics of the risk of dying in terms of survivorship

Let us now assume that all the quantities defined in the previous section vary with respect to the time variable y , such that $\psi(s,y)$ and $\mu(s,y)$ are, respectively, the density function and the mortality hazard of s at time y . Thus, changes over time in $\psi(s,y)$ are denoted by adding a dot on top of the quantity:

$$\dot{\psi}(s,y) = \frac{\partial \psi(s,y)}{\partial y}. \quad (6.3)$$

Changes over time in $\mu(x,y)$ are captured by the rate of mortality improvement, $\rho(s,y)$ which is commonly used in the demographic literature (Rau et al., 2008). However, in this context, $\rho(s,y)$ measures how the risk of dying calculated at specific survival level s has changed over time. This indicator is defined as:

$$\rho(s,y) = -\frac{\frac{\partial \mu(s,y)}{\partial y}}{\mu(s,y)} = -\frac{\partial \ln(\mu(s,y))}{\partial y}. \quad (6.4)$$

Finally, we introduce function $b(s,y)$, which measures the relative change in the risk of dying with respect to s :

$$b(s,y) = \frac{\frac{\partial \mu(s,y)}{\partial s}}{\mu(s,y)} = \frac{\partial \ln(\mu(s,y))}{\partial s}. \quad (6.5)$$

Given that survival is a monotonically decreasing function, $b(s,y)$ indicates how the risk of dying changes as survival s decreases.

6.2.1 Assuming a Gompertz function

Consider the trajectory of the force of mortality μ defined by the Gompertz⁷ function $\mu(x) = ae^{bx} = be^{b(x-M)}$, where a is the initial level of the force of mortality at age zero, b is the rate of aging, and M is the mode of the distribution of deaths, the most common age at death (Missov et al., 2015). In this case, the survival function is given by:

$$S(x) = e^{-e^{-bM}(e^{bx}-1)}. \quad (6.6)$$

Solving for x yields

$$x(s) = \ln(1 - e^{bM} \ln(s))/b. \quad (6.7)$$

⁷In this section we illustrate our framework by assuming a Gompertz parametric function. This could have been done with other parametric function (i.e. Gamma-Gompertz, Gamma-Gompertz-Makeham, etc.). It is important to highlight that this section is solely for illustration purposes because the fit of parametric modelling is out of the scope of this paper. We are in the progress of writing a follow up paper together with Prof. Trifon Missov that specifically account for parametric modelling.

This expression can be used, for example, to calculate the median age at death, when s is 50%. If b is 0.14 and M is 90, then the median age at death is 87.4. Survival to age x is given by $e^{-H(x)}$, with cumulative hazard $H(x) = \int_0^x \mu(y)dy$. Hence, $H(s) = -\ln(s)$ and Equation (6.7) can be written as $x(s) = \frac{\ln(1 + H(s)e^{bM})}{b}$. Furthermore, in the Gompertz case we have that the density and the hazard functions of s result in:

$$\psi(s) = b(e^{-bM} - \ln(s))s, \quad (6.8)$$

and

$$\mu(s) = b(e^{-bM} - \ln(s)). \quad (6.9)$$

In many applications, e^{-bM} will be very small. For instance, $e^{-(.14)(90)} = 0.000003$. In such cases, $\mu(s) = -b\ln(s) = bH$ a result that implies that if two populations share the same value of b , then the trajectory of mortality over s-age will be the same even if the two populations have different modal ages at death and different initial values of mortality at age zero. The force of mortality will rise linearly with H , i.e., with $-\ln s$.

In the Gompertz case, (6.5) reduces to

$$b(s) = \frac{1}{s \ln(s)} \quad (6.10)$$

Hence, for Gompertz mortality, the rate of aging in a survivorship framework does not depend on the Gompertz parameters a (or M) or b : it is only a function of s . Now suppose the force of mortality follows Gompertz' law, $\mu(x, y) = a(y)e^{bx}$, with the initial level of mortality varying over time but with b invariant. The rate of mortality improvement, $\rho(s, y)$ reduces to:

$$\rho(s, y) = -\frac{da(y)/dy}{a(y) - b \ln s} \quad (6.11)$$

If $a(y)$ is small, then the numerator will be small and $\rho(s, y)$ will be close to zero: the force of mortality at an s-age will change little over time.

6.3 Data

In this article, we use raw death counts and exposures by sex and calendar year comprising the period 1900-2018 for 23 populations, from the highest data quality available, in the (Human Mortality Database, 2021): Australia, Austria, Belgium, Canada, Czech Republic, Denmark, Finland, France, Germany, Great Britain, Hong Kong, Italy, Israel, Japan, Korea, Luxemburg, Netherlands, New Zealand, Norway, Portugal, Sweden, Switzerland and United States.

We required a continuous surface of mortality to calculate the location of s-ages. For this reason, we smoothed raw death rates over time and over age using 2D P-Splines (Camarda et al., 2012).

Given the non-parametric properties of 2D P-Splines, we ensured that the smoothing procedure did not distort estimations of the risk of dying or any of the quantities analyzed here. Having continuous estimates of mortality over age and time allows for the calculation of s-age and associated functions with high precision. This statistical procedure has been used in previous investigations (Colchero et al., 2016; Jones et al., 2014).

We performed two sensitivity analyses to test whether our results were driven by the choice of the smoothing algorithm (see Supplementary Material). In the first analysis, we used a generic spline model (De Boor, 2001) to smooth death rates by age. In the second sensitivity analysis, we did not apply any smoothing algorithm. Instead, we used linear interpolation to calculate s-age and associated functions. In both analyses, our results are almost identical to the ones produced with 2D P-Splines (Camarda et al., 2012). This indicates that our results are robust and that they do not depend on the smoothing algorithm employed.

6.4 Results

Figure 6.1 illustrates the calculation of s-ages (panel A) and the corresponding risk of dying (panel B). Function $x(s)$ indicates the age at which survivorship is s , and $\mu(s)$ is the risk of dying at that specific survival level. We start the calculation of s-ages at birth, so that $x(1)$ represents 100% of the population and it is always located at birth. We end the analysis at $x(.01)$ because the noise in the last s-age is high and it is not possible to accurately estimate the location of $x(0)$ from aggregated data.

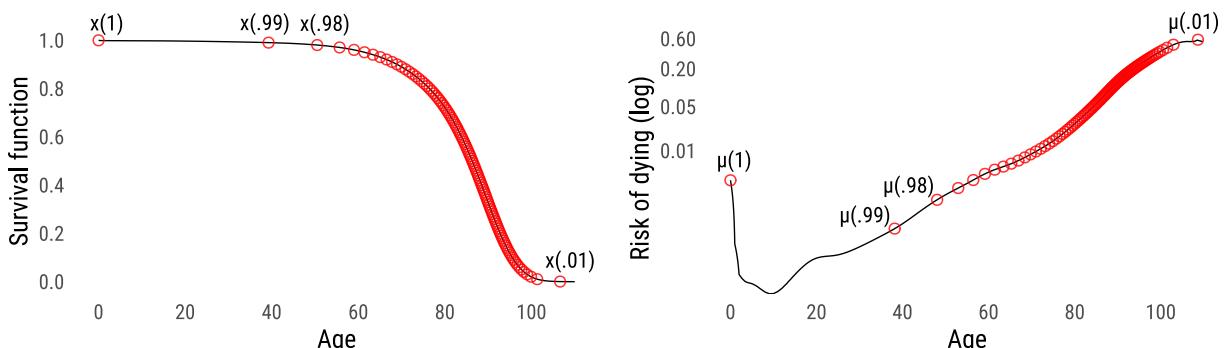


Figure 6.1: Survival function and associated risk of dying for Swedish females, 2018. Red circles indicate the location of s-ages.

From Figure 6.1, we observe that $x(s)$ are not equidistant. For example, there is almost 40 years between $x(1)$ and $x(.99)$, whereas $x(.99)$ and $x(.98)$ are located less than 15 years apart. Subsequent s-ages are closer to each other, indicating a greater concentration of deaths at those s-ages. Changes over time in the location of s-ages reflect changes over time in survivorship. The risk of dying associated to each s-age also varies accordingly. In the following sections, we examine changes over time in s-ages and how they trigger changes in the risk of dying.

The steady postponement of survival

Figure 6.2 depicts trends in s-ages, $x(s)$, from 1900 to 2010 for females in France, Italy and Sweden. We illustrate our results with these countries because they exhibit high-quality data dating back to 1900. Nonetheless, similar results hold across all the 23 populations analyzed in this study (see Supplementary Material for further details).

In Figure 6.2, we observe that major shifts in survival occurred in the top ten s-ages $x(.99)$ to $x(.90)$. At the beginning of the twentieth century, 90% of the population (i.e. $x(.90)$) survived to age 2 in France and Italy and to age 5 in Sweden. Thereafter, deaths unfolded into a much wider age-interval. For example, in 2018, 90% of the population in each of these countries survived to age 70. Even more impressive is that, in 2018, 99% of the population (i.e. $x(0.99)$) survived to age 35. Figure 6.2 also shows that major shifts of s-ages from $x(.99)$ to $x(.90)$ produced a relocation in all subsequent $x(s)$. For example, in France, $x(.10)$ (i.e. the age where only 10% of the population is still alive) shifted from age 87.61 in 1950 to age 97.04 in 2017. A similar pattern is observed from $x(.89)$ to $x(.01)$.

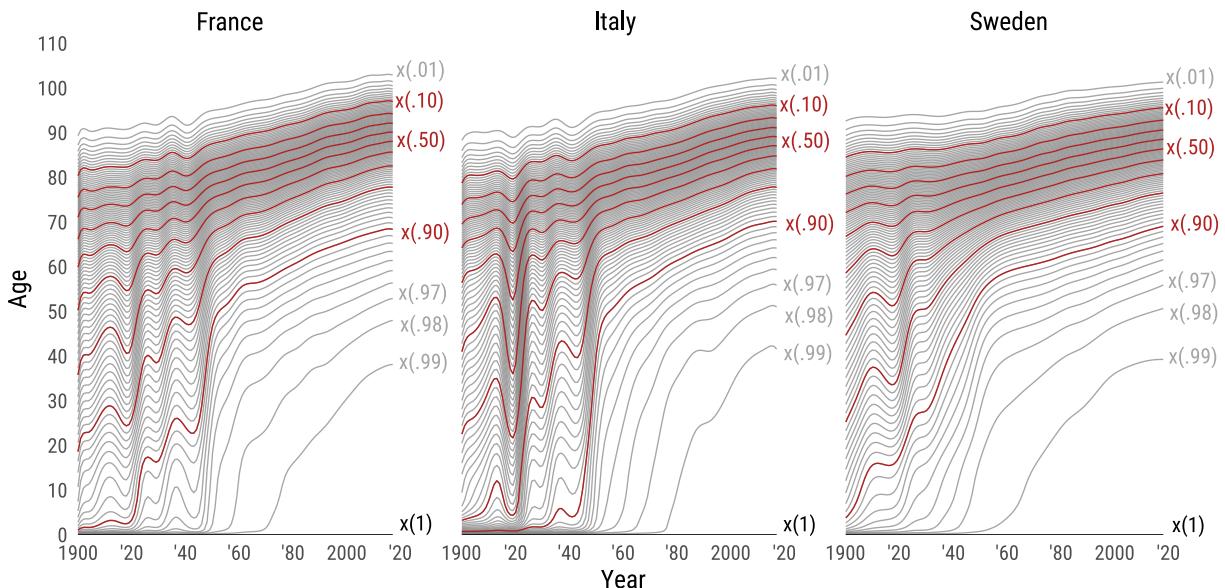


Figure 6.2: Survivorship ages for France, Italy and Sweden. Females, 1900-2018. Red lines indicate deciles.

From Figure 6.2, we observe that distances between consecutive s-ages from $x(.90)$ to $x(.01)$ have remained constant over time. In Figure 6.3, we explore this pattern in detail by looking at the time derivative of the density function of s , $\psi(s,y)$. From 1900 to 1950, there are distortions in the distances between s-ages as $\psi(s,y)$ fluctuates from positive to negative values. However, after 1950, $\psi(s,y)$ is close to zero for all s from 0.90 to 0.01. This indicates that: i) distances between s-ages for $s \geq 0.90$ have remained constant over time, and ii) s-ages relocate all together at the same pace. Wilmoth and Horiuchi (1999) provided some evidence of parallel shifts in the concentration of deaths, and Zuo et al. (2018) analyzed this issue in depth after age 65. In the present study, we show that steady shifts in the concentration of deaths also hold for survival probabilities. However, we also show that those steady shifts already begin at the age where 90% of individuals are still alive (i.e. s-age $x(.90)$), which is located well before age 65. Our results show that Zuo et al. (2018) finding regarding an increasing distance between age 65 and

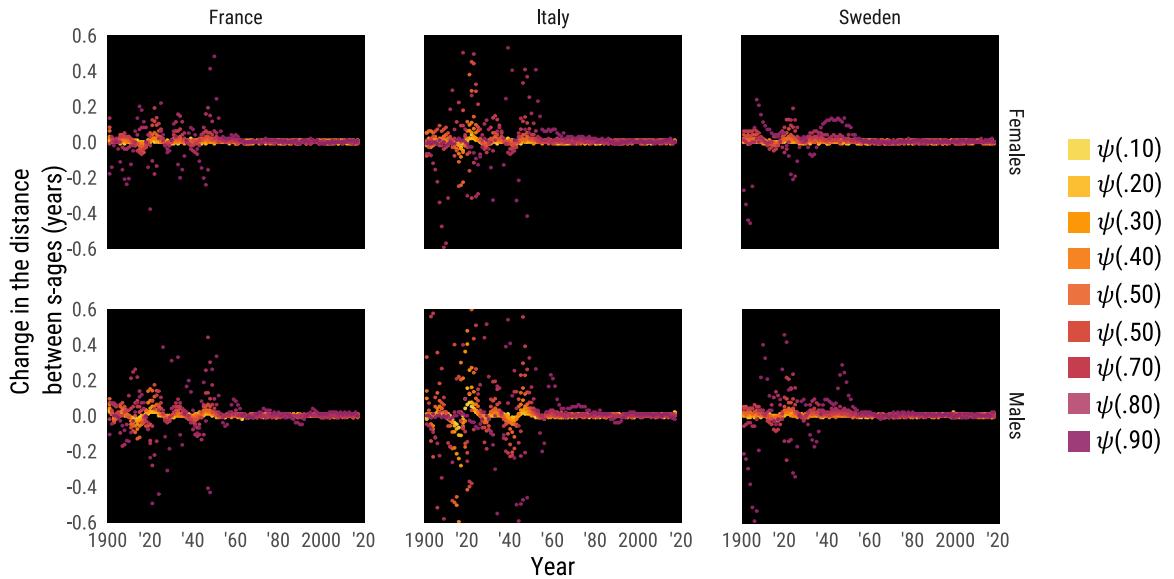


Figure 6.3: Change in the distance between subsequent s-ages captured by $\psi(s,y)$. France, Italy and Sweden, both sexes, 1900-2018. For illustration purposes, only deciles are shown.

the 25th percentile is the consequence of truncating the distribution of deaths at age 65. To test this, we performed a sensitivity analysis (see Supplementary Material), where we started calculations at various chronological ages (e.g., $x(1)$ is set at, respectively, ages 35, 50 and 65). Indeed, we show that truncating at any chronological age distorts the distances between s-ages because they are compressed (Kannisto, 2001; Thatcher et al., 2010). Therefore, truncating at chronological ages triggers misleading results about demographic patterns of survival, the risk of dying and associated functions of these indicators (i.e. life expectancy and lifespan inequality indicators).

The constant dynamics of the risk of dying

Figure 6.4, Panel A shows the risk of dying over chronological age x for six different sex-specific populations during different years. Figure 6.4, Panel B also shows the risk of dying for the same populations, but in this case, the risk of dying is expressed in terms of s . Figure 6.4 shows an important finding: When trajectories of mortality over age x are compared, the curves differ, but when such trajectories over s are compared, they are similar. In this section, we examine this finding in detail.

Figure 6.5 depicts trajectories over time in the risk of dying in terms survivorship, $\mu(s,y)$. We observe two important patterns in this figure. First, major shifts in top s-ages (i.e. $x(.99)$, $x(.98), \dots, x(.90)$) reported in Figure 2 coincide with pronounced declines in $\mu(s,y)$. In particular, $\mu(.99,y)$ plummeted and it is around $x(.99)$ where the minimum risk of dying has been located for almost four decades (Ebeling, 2018). Figure 6.5 shows that the relationship between the risk of dying and survival suffered distortions at the beginning of the survival curve (i.e. $s = 1, 0.99, \dots, 0.90$). However, after 1980, declines in $\mu(s,y)$ ceased, with exception of the risk of dying at birth, $\mu(1,y)$, which continues trending downwards. Second, in Figure 6.5 we

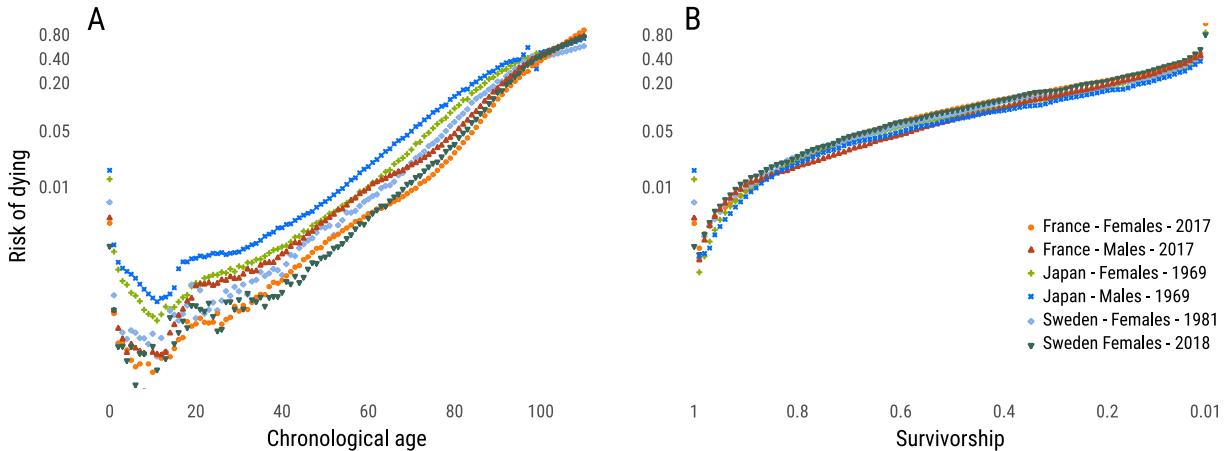


Figure 6.4: Mortality trajectories for six different sex-specific populations during different years. *Panel A shows the risk of dying over chronological ages. Panel B depicts the risk of dying over survivorship. In both panels, there the risk of dying was calculated from raw data, without smoothing, to show the differences and similarities between mortality trajectories.*

also observe steady trends over time in $\mu(s, y)$ for $s = 0.90, \dots, 0.01$. Since 1950, the risk of dying for survivorship levels between 0.90 and 0.50 has remained almost constant over time and there have been minuscule increases in $\mu(s, y)$ for survivorship between 0.50 to 0.01. These patterns indicate a regular association between the risk of dying and survival. This is an important finding entailing that, over time, what has changed is the relationship between the age variable and survival (as shown in Figure 6.2), while the demographic relationship between survival and the risk of dying has remained stable.

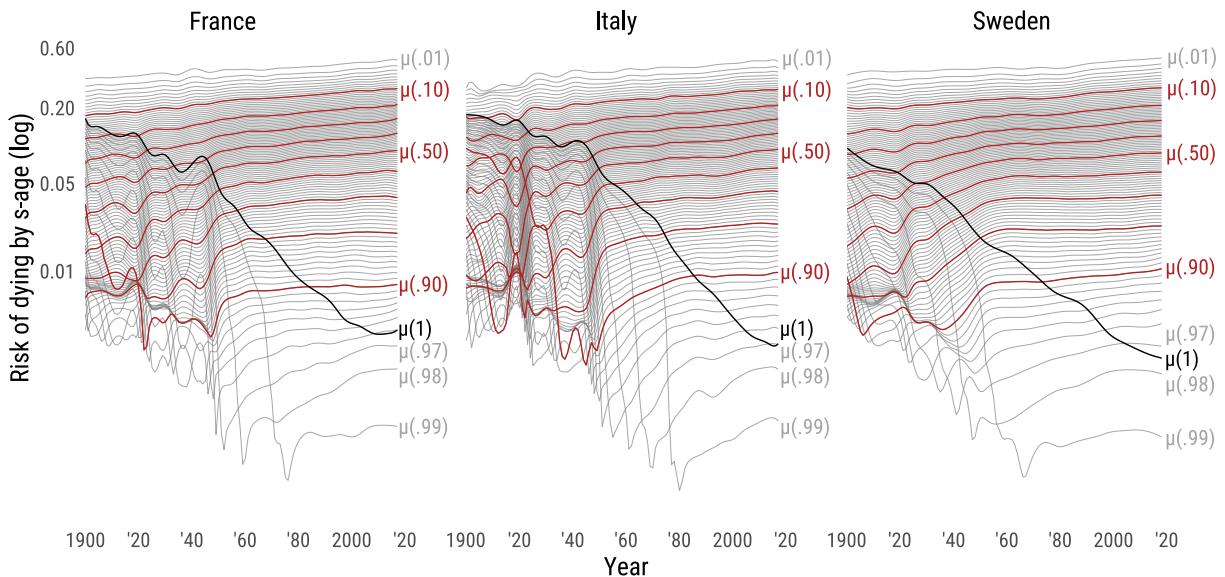


Figure 6.5: Trends over time in risk of dying by s for France, Italy and Sweden. Females, 1900-2018.

We next examine trends over time in the rate of mortality improvement, $\rho(s, y)$. Figure 6.6 depicts values of $\rho(s, y)$ for $s = 0.90, 0.80, \dots, 0.10$ for the three populations analyzed here⁸.

⁸For illustrative purposes, in Figure 6.6, we only show values of $\rho(s, y)$ from $s = 0.9, 0.8, \dots, 0.1$, but our

As expected from the results shown in Figure 6.6, fluctuations in $\rho(s,y)$ are noticeable prior to 1950. Such fluctuations could be attributed to deaths occurring during the two world wars and the Spanish flu epidemic (Johnson and Mueller, 2002). Thereafter, $\rho(s,y)$ takes values close to zero at all survivorship s . At first glance this finding could be surprising, given that previous research has shown mortality improvements at different chronological ages (Rau et al., 2017, 2008). However, it is important to highlight that $\rho(s,y)$ does not measure mortality improvement by chronological age. Instead, $\rho(s,y)$ is an indicator that captures changes over time in the mortality at different levels of survival s . Thus, null values of $\rho(s,y)$ entail that the risk of dying has remained constant⁹ over time at any level of survivorship s below 0.90.

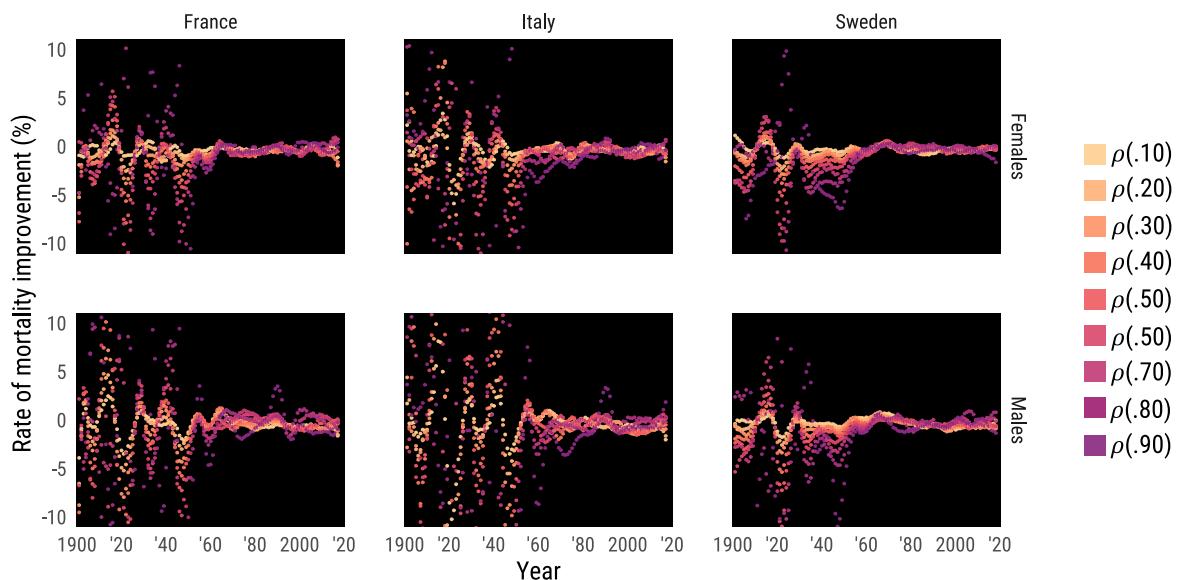


Figure 6.6: Rates of mortality improvement by s . France, Italy and Sweden, both sexes, 1900-2018. *For illustration purposes, only deciles are shown.*

Finally, we analyze the rate of change in the risk of dying with respect to decreases in survival s , denoted by the indicator $b(s,y)$. In Figure 6.7, we show trends over time in $b(s,y)$. Similar to the results shown in Figure 6 for $\rho(s,y)$, we observe fluctuations in $b(s,y)$ prior to 1950. Thereafter, $b(s,y)$ remains constant over time. For example, $b(.10,y)$ for French females took values of 0.038 in 1950, 0.038 in 1970, 0.036 in 1990 and 0.037 in 2017. The stability over time of $b(s,y)$ after 1950 is notorious below $s = 0.70$. However, we also observe that $b(s,y)$ varies depending on s . As mentioned in Section 2.3, $b(s,y)$ does not measure how the risk of dying changes with increases in chronological ages x . Instead, $b(s,y)$ indicates how the risk of dying changes as survival s goes down, from 1 to 0. In this sense, high values of $b(s,y)$ indicate a fast increase in the risk of dying from one level of survival to the next one, where there are fewer individuals alive.

Our findings regarding $b(s,y)$ shed light on contemporary demographic riddles. For example, Vaupel (2010) postulated that, after a certain advanced age, increases in the individual risk of dying with age might be constant and it would be the same for all populations. Our results

findings apply to any level of survivorship between 0.9 and 0.01.

⁹In relative terms, as $\rho(s,y)$ is defined as the relative derivative of the risk of dying with respect to time, see Equation (6.4).

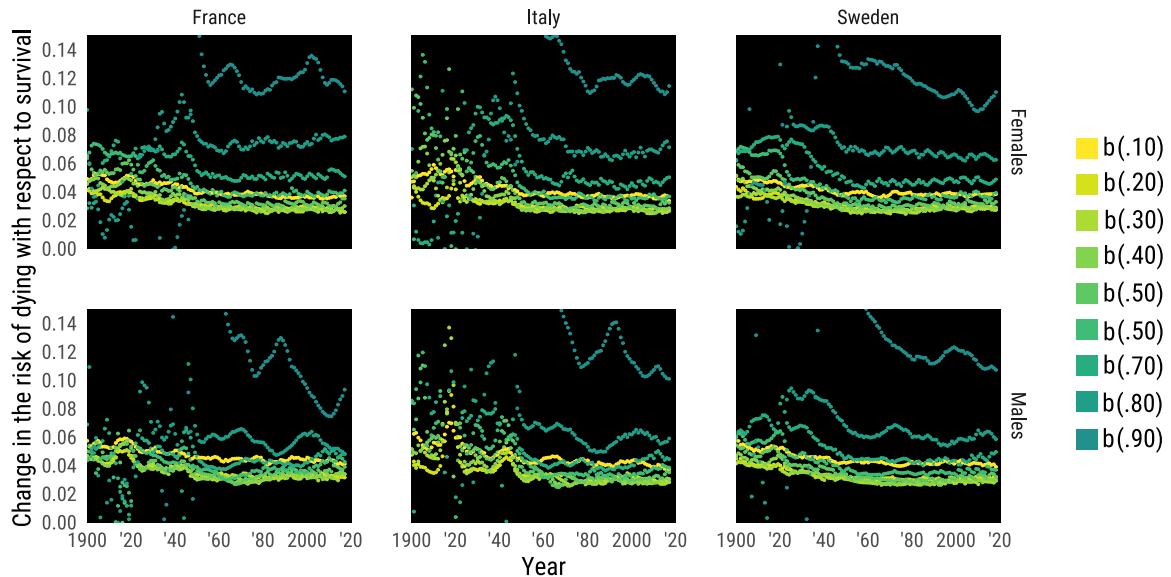


Figure 6.7: Rate of change in the risk of dying with respect to change in survival s . France, Italy and Sweden, both sexes, 1900-2018. *For illustration purposes, only deciles are shown.*

indicate that a variant of Vaupel (2010) hypothesis is correct, given that the rate of change in the risk of dying with respect to survival is constant over time. However, we also show that such advanced age has shifted over time, and it is defined in terms of $x(s)$.

6.5 Discussion

Everyone has a chronological age x and, depending on the population they are in, a value of s . When trajectories of mortality over age x are compared (e.g., across populations, over time, or females vs. males), the curves differ. When mortality trajectories over s are compared, they are similar. This is an important demographic regularity that holds for all the 23 populations analyzed here.

In this study we show that, over more than 100 years, major shifts in survival occurred at ages where the first 10% of the population die. It is also around those ages where most of the changes in the shape of the risk of dying were observed. However, for the remaining 90% of the population, the relationship between survival and the risk of dying has remained steady over time. This finding indicates that, over time, what has changed is the relationship between mortality and chronological ages but the relationship between survival and the risk of dying has remained remarkably regular. In other words, populations experience similar dynamics in the risk of dying at levels of survivorship below 0.90.

The relationship between survival and the risk of dying is regular but it is not immutable. In our results for France, Sweden and Italy, we show that this steady relationship was distorted during some periods before 1950. Such distortions might be attributed to the massive number of deaths that occurred during the two world wars and the Spanish flu pandemic. Nonetheless, after such events ceased, the relationship between demographic indicators became regular again. In the

light of the excess deaths attributed to the ongoing Covid-19 pandemic (Kashnitsky and Aburto, 2020), we foresee that a new disruption will be observed during the following years. However, based on our results, we also anticipate that a regular relationship will emerge once again.

Chronological ages and survivorship ages

Chronological ages have a long history of being used as a measure of maturity (Field and Syrett, 2020) and they are widely used in social sciences as the main time variable to describe demographic events (Thane, 2020). However, it has also been shown that they influence results about the dynamics of mortality (Beltrán-Sánchez and Subramanian, 2019; Wilmoth and Horiuchi, 1999; Zuo et al., 2018).

A key contribution of this article is the definition of survivorship ages, or s-ages. Contrary to chronological ages, survivorship ages relate the lifespan of individual to the actual source of demographic change, i.e. death and survival. This perspective is specifically developed for demographic studies. Here, we show that the use of s-ages provides important insights about the dynamics of human mortality. Further research endeavours aimed at the development of summary demographic measures (e.g., life expectancy or lifespan inequality indicators) in terms of s-ages will enrich the demographic toolkit.

While we highlight the advantages of using survivorship ages in demographic studies, it is unlikely that they will replace chronological ages as the main time dimension. Chronological ages are widely used because of their simplicity and interpretability. We acknowledge the advantages of using chronological ages in demographic research. Nonetheless, we also highlight their shortcomings. Specifically, our results raise awareness about how truncating to chronological ages might result in misleading conclusions about the dynamics of the risk of dying and related indicators. An example of this issue is the increasing distance between the 25th percentile and age 65, reported in Zuo et al. (2018), which we show is a consequence of starting the analysis at age 65, where the postponement of human mortality is not fully captured. Researchers should therefore be cautious when truncating at chronological ages and aware that the steady postponement of mortality will most likely affect their results.

Unsolved demographic questions

Our results shed light on the demographic mechanisms of senescence. We show that the rate of change in the risk of dying with respect to survival is constant over time. This indicates that populations experience similar dynamics in the risk of dying at specific levels of survivorship. This finding shows that a variant of Vaupel (2010) hypothesis (regarding a constant rate of increase in the risk of dying with age) is correct. However, one should be careful in the interpretation of this result as changes in the risk of dying are analyzed in the current study with respect to the decrease in survival (from 1 to 0), and not with respect to increases with age, as originally stated by Vaupel (2010).

Moreover, there are important aspects of Vaupel (2010) hypothesis that require further examination. Vaupel (2010) makes a distinction between the risk of dying observed at the population level and the inherent risk of dying that each individual has. The former is calculated using

aggregated life table data, whereas the latter can only be inferred from statistical models controlling for unobserved heterogeneity (Vaupel et al., 1979). In the current study, we focus the analysis on demographic indicators at population level. Therefore, it remains unknown whether the individual risk of dying is constant or not. To solve this demographic riddle, models that account for unobserved heterogeneity and the shifts of survival should be developed, applied, and tested with empirical data. In our study, we have put forward a framework to study such issues in terms of s-ages. Thus, a synergy between the framework developed here and the mathematics behind the theory of heterogeneous populations (Vaupel and Missov, 2014) will contribute to the advancement of knowledge regarding regularities in the individual risk of dying.

Finally, there are two important questions that are still unsolved: Why are shifts in survival so regular across populations? Why is the risk of dying so steady at specific levels of survival? The mechanisms behind these demographic regularities are yet unknown. Theories of senescence might hint at answers to these questions (Baudisch and Vaupel, 2012; Colchero et al., 2021, 2016; Le Bourg, 2001; Omholt and Kirkwood, 2021; Vaupel, 2010; Wachter et al., 2014). We envisage that our results will serve to enrich such theories.

Bibliography

- Baudisch, A. and Vaupel, J. W. (2012). Getting to the root of aging. *Science*, 338(6107):618–619.
- Beltrán-Sánchez, H. and Subramanian, S. (2019). Period and cohort-specific trends in life expectancy at different ages: Analysis of survival in high-income countries. *SSM-population health*, 8:100422.
- Bergeron-Boucher, M.-P., Ebeling, M., and Canudas-Romo, V. (2015). Decomposing changes in life expectancy: Compression versus shifting mortality. *Demographic Research*, 33:391–424.
- Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.
- Burger, O., Baudisch, A., and Vaupel, J. W. (2012). Human mortality improvement in evolutionary context. *Proceedings of the National Academy of Sciences*, 109(44):18210–18214.
- Camarda, C. G. et al. (2012). Mortalitysmooth: An R package for smoothing poisson counts with p-splines. *Journal of Statistical Software*, 50(1):1–24.
- Canudas-Romo, V. (2008). The modal age at death and the shifting mortality hypothesis. *Demographic Research*, 19:1179–1204.
- Colchero, F., Aburto, J. M., Archie, E. A., Boesch, C., Breuer, T., Campos, F. A., Collins, A., Conde, D. A., Cords, M., Crockford, C., et al. (2021). The long lives of primates and the invariant rate of ageing hypothesis. *Nature communications*, 12(1):1–10.
- Colchero, F., Rau, R., Jones, O. R., Barthold, J. A., Conde, D. A., Lenart, A., Nemeth, L., Scheuerlein, A., Schooley, J., Torres, C., Zarulli, V., Altmann, J., Brockman, D. K., Bronikowski, A. M., Fedigan, L. M., Pusey, A. E., Stoinski, T. S., Strier, K. B., Baudisch, A., Alberts, S. C., and Vaupel, J. W. (2016). The emergence of longevous populations. *Proceedings of the National Academy of Sciences*, 113(48):E7681—E7690.
- De Boor, C. (2001). Calculation of the smoothing spline with weighted roughness measure. *Mathematical Models and Methods in Applied Sciences*, 11(01):33–41.
- Ebeling, M. (2018). How has the lower boundary of human mortality evolved, and has it already stopped decreasing? *Demography*, 55(5):1887–1903.
- Field, C. T. and Syrett, N. L. (2020). *Chronological Age: A Useful Category of Historical Analysis Introduction*. Oxford University Press.

- Gilchrist, W. (2000). *Statistical modelling with quantile functions*. CRC Press.
- Human Mortality Database (2021). University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). www.mortality.org.
- Johnson, N. P. and Mueller, J. (2002). Updating the accounts: global mortality of the 1918–1920 “spanish” influenza pandemic. *Bulletin of the History of Medicine*, pages 105–115.
- Jones, O. R., Scheuerlein, A., Salguero-Gómez, R., Camarda, C. G., Schaible, R., Casper, B. B., Dahlgren, J. P., Ehrlén, J., García, M. B., Menges, E. S., Quintana-Ascencio, P. F., Caswell, H., Baudisch, A., and Vaupel, J. W. (2014). Diversity of ageing across the tree of life. *Nature*, 505(7482):169–73.
- Kannisto, V. (2001). Mode and dispersion of the length of life. *Population: An English Selection*, 13(1):159–171.
- Kashnitsky, I. and Aburto, J. M. (2020). Covid-19 in unequally ageing european regions. *World Development*, 136:105170.
- Le Bourg, É. (2001). A mini-review of the evolutionary theories of aging. is it the time to accept them? *Demographic Research*, 4:1–28.
- Missov, T. I., Lenart, A., Nemeth, L., Canudas-Romo, V., and Vaupel, J. W. (2015). The gompertz force of mortality in terms of the modal age at death. *Demographic Research*, 32:1031–1048.
- Myers, G. C. and Manton, K. G. (1984). Compression of mortality: myth or reality? *The Gerontologist*, 24(4):346–353.
- Nair, N. U. and Sankaran, P. (2009). Quantile-based reliability analysis. *Communications in Statistics Theory and Methods*, 38(2):222–232.
- Nair, N. U., Sankaran, P., and Balakrishnan, N. (2013). *Quantile-based reliability analysis*. Springer.
- Nusselder, W. J. and Mackenbach, J. P. (1996). Rectangularization of the survival curve in the netherlands, 1950-1992. *The Gerontologist*, 36(6):773–782.
- Omholt, S. W. and Kirkwood, T. B. (2021). Aging as a consequence of selection to reduce the environmental risk of dying. *Proceedings of the National Academy of Sciences*, 118(22).
- Rau, R., Bohk-Ewald, C., Muszyńska, M. M., and Vaupel, J. W. (2017). Visualizing mortality dynamics in the lexis diagram.
- Rau, R., Soroko, E., Jasilionis, D., and Vaupel, J. W. (2008). Continued reductions in mortality at advanced ages. *Population and Development Review*, 34(4):747–768.
- Thane, P. (2020). Old age in european cultures: A significant presence from antiquity to the present. *The American Historical Review*, 125(2):385–395.
- Thatcher, A. R., Cheung, S. L. K., Horiuchi, S., and Robine, J.-M. (2010). The compression of deaths above the mode. *Demographic Research*, 22:505.

- Vaupel, J. W. (2010). Biodemography of human ageing. *Nature*, 464(7288):536–42.
- Vaupel, J. W. and Gowan, A. E. (1986). Passage to methuselah: some demographic consequences of continued progress against mortality. *American Journal of Public Health*, 76(4):430–433.
- Vaupel, J. W., Manton, K. G., and Stallard, E. (1979). The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality. *Demography*, 16(3):439.
- Vaupel, J. W. and Missov, T. I. (2014). Unobserved population heterogeneity: A review of formal relationships. *Demographic Research*, 31(1):659–686.
- Wachter, K. W., Steinsaltz, D., and Evans, S. N. (2014). Evolutionary shaping of demographic schedules. *Proceedings of the National Academy of Sciences*, 111(Supplement 3):10846–10853.
- Wilmoth, J. R. and Horiuchi, S. (1999). Rectangularization revisited: Variability of age at death within human populations. *Demography*, 36(4):475–495.
- Zuo, W., Jiang, S., Guo, Z., Feldman, M. W., and Tuljapurkar, S. (2018). Advancing front of old-age human survival. *Proceedings of the National Academy of Sciences*, 115(44):11209–11214.

Acknowledgements

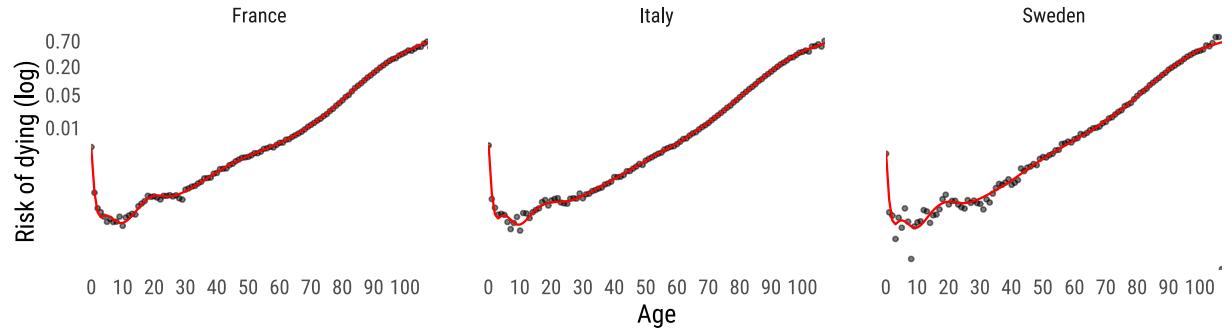
We thank Trifon Missov and José Manuel Aburto for comments on the manuscript and for checking the validity of Equations developed in Section 6.2. Jesús-Adrián Alvarez was supported by the AXA Research Fund, through the funding for the AXA Chair in Longevity Research.

Supplementary Material

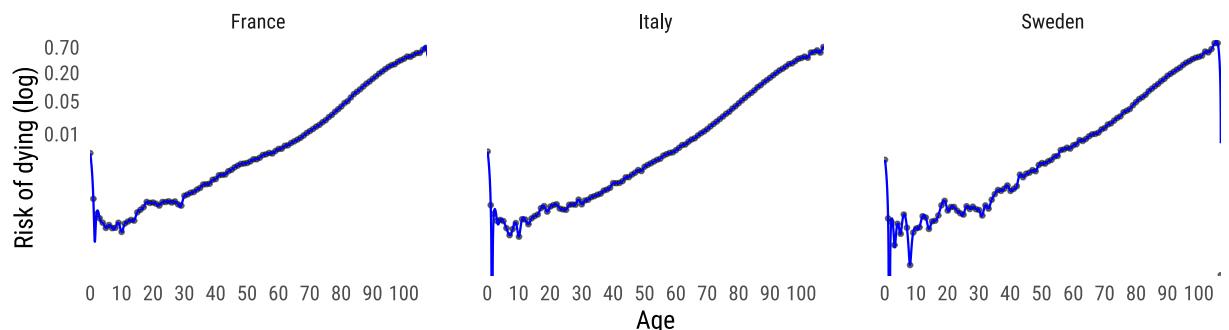
Sensitivity analyses using different smoothing algorithms

We performed two sensitivity analyses to test whether our results were driven by the smoothing algorithm. In the first analysis, we smoothed raw death rates using a generic algorithm of B-Splines (De Boor, 2001). This algorithm is widely used to smooth all kinds of data (not only mortality data) and it is implemented in R in the package *splines*. In the second analysis, we did not smooth the data. Instead, we calculated s-ages and associated functions by using linear interpolation on raw data. In Figure 8, we show the estimation of the age trajectory of the risk of dying using 2D P-Splines, a generic spline and linear interpolation for females in France, Italy and Sweden in 2017. We observe that P-Splines capture better the age trajectory of the risk of dying, as they are specifically developed for mortality data (Camarda et al., 2012). The generic spline show fluctuations at younger ages. The estimates using linear interpolation go through all the raw death rates. In Figures 9 and 9, we show trajectories over time in s-ages calculated using a generic spline and linear interpolation. Both figures are very similar to Figure 2 (using P-Splines). Likewise, in Figures 11 and 12, we show trends over time in the risk of dying by s ; i.e. $\mu(s)$. Results are very similar to Figure 5. This indicates that our findings are robust and not driven by the smoothing algorithm used in this study.

2D P-Splines



Generic B-Splines



Linear interpolation

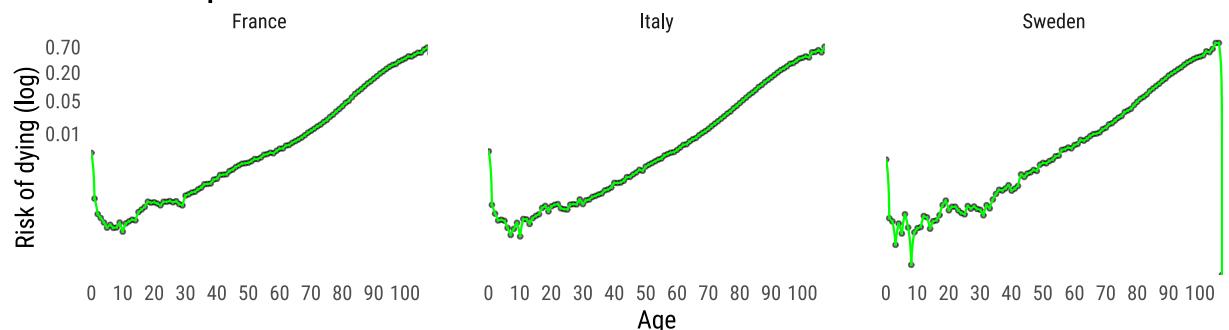


Figure 8: Comparison of smoothing procedures of risk of dying for females in France, Italy and Sweden, 2017

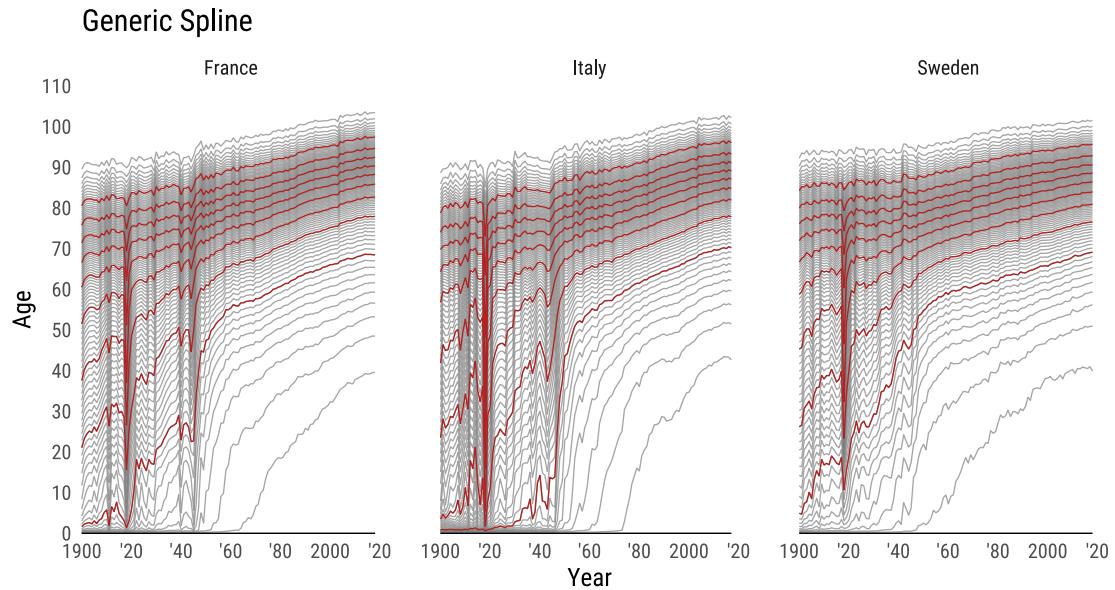


Figure 9: Trends over time in s-ages using a generic spline to smooth mortality data. Females in France, Italy and Sweden, 1900-2018

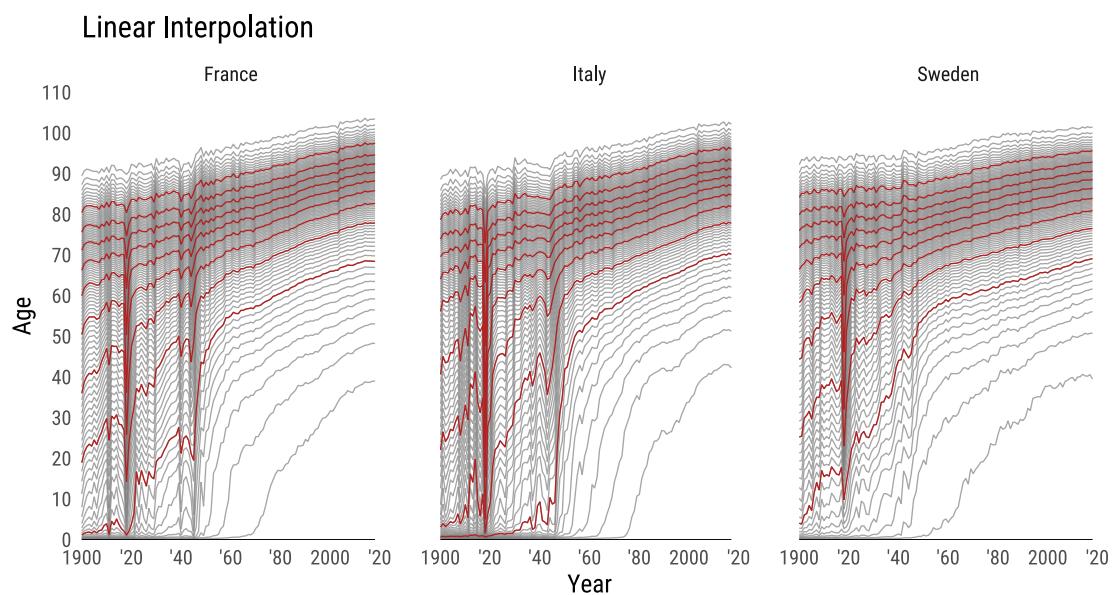


Figure 10: Trends over time in s-ages calculated using linear interpolation on raw data. Females in France, Italy and Sweden, 1900-2018

Generic Spline

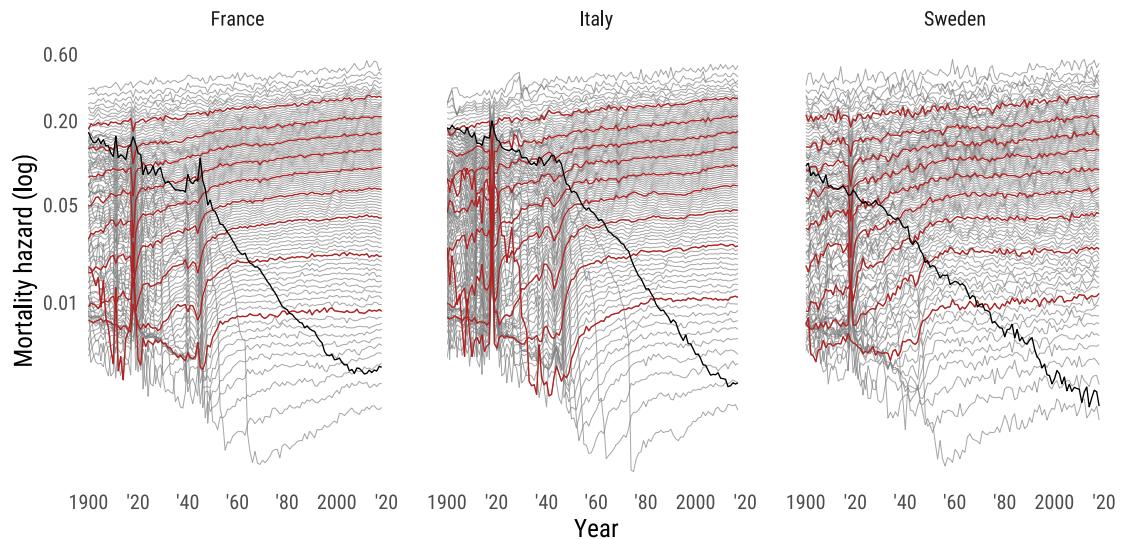


Figure 11: Trends over time in the risk of dying by s-ages calculated using a generic spline to smooth mortality data. Females in France, Italy and Sweden, 1900-2018

Linear Interpolation

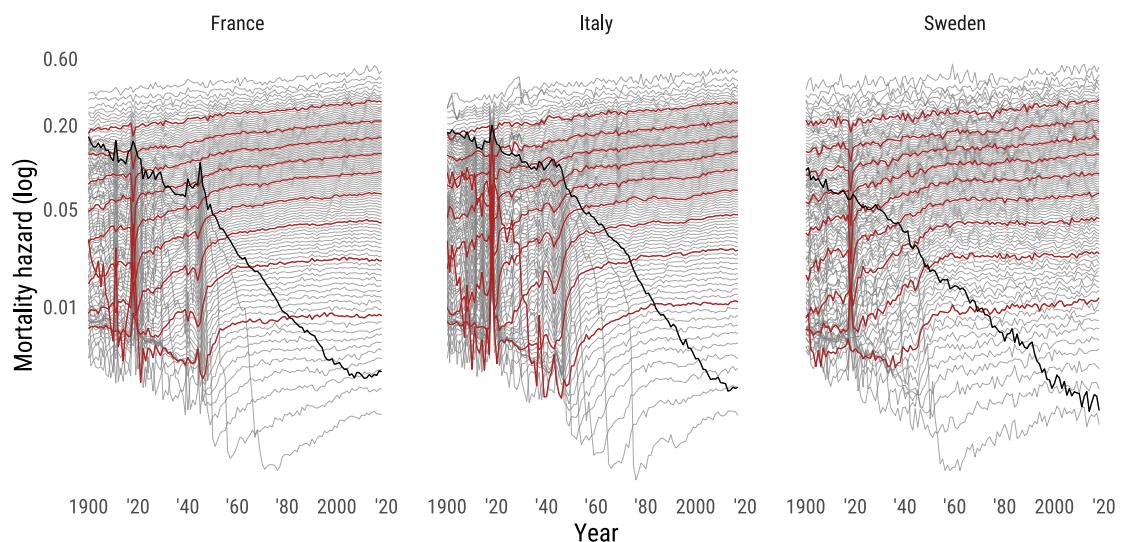


Figure 12: Trends over time in the risk of dying by s-ages calculated using linear interpolation on raw data. Females in France, Italy and Sweden, 1900-2018

Calculation of s-ages by truncating at different chronological ages

In this section, we perform a sensitivity analysis by calculating s-ages at different chronological ages. Specifically, we truncate at ages 35, 50 and 65, respectively. In Figures 13, 14 and 15 we can observe that truncation affects the distances between s-ages as they are reduced, producing an artificial compression of deaths (Kannisto, 2001). Truncating at arbitrary chronological ages (as we do here), has an impact on the survival function, risk of dying and summary measures of mortality that depend on them (life expectancy and lifespan inequality indicators). It is therefore recommended that the calculation of demographic indicator start at birth, where the total population is alive. In the case where that is not possible (due to data constraints, specific research question, etc.), researchers should be aware that the steady postponement of mortality will most likely have an impact on their results.

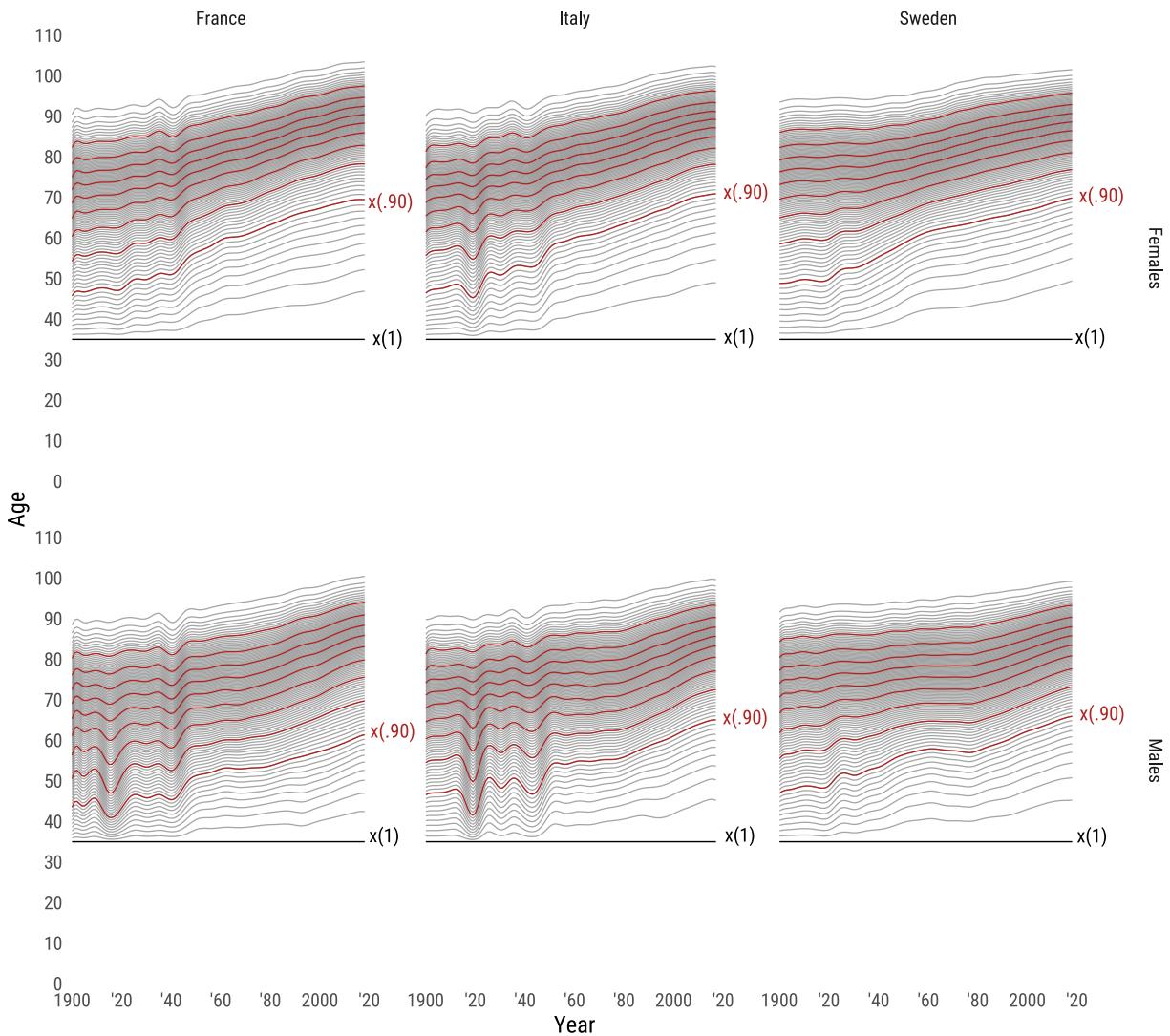


Figure 13: Survivorship ages for France, Italy and Sweden. Females, 1900-2018. Calculations start at age 35, i.e., $x(1) = 35$

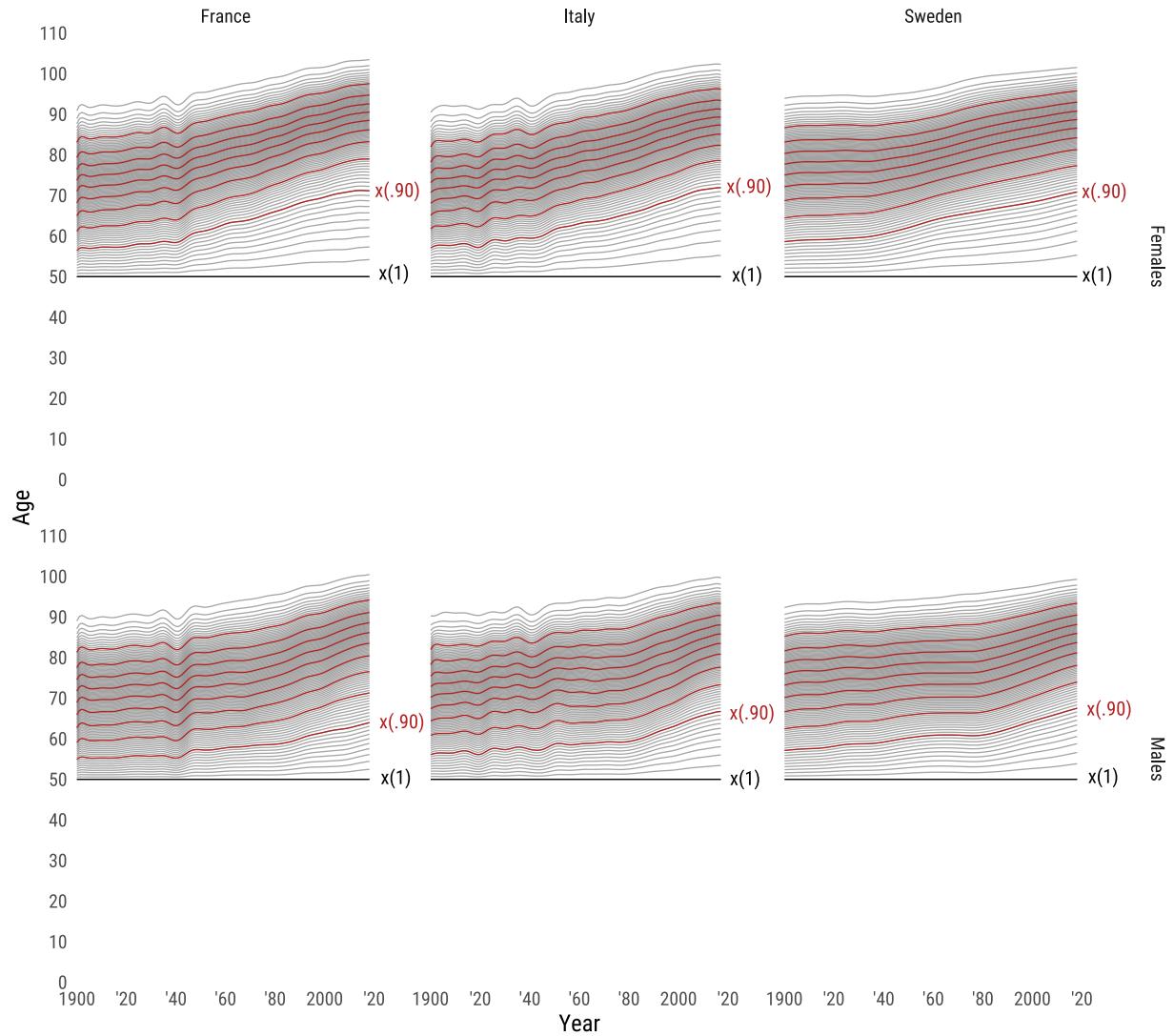


Figure 14: Survivorship ages for France, Italy and Sweden. Females, 1900-2018. Calculations start at age 50, i.e., $x(1) = 50$

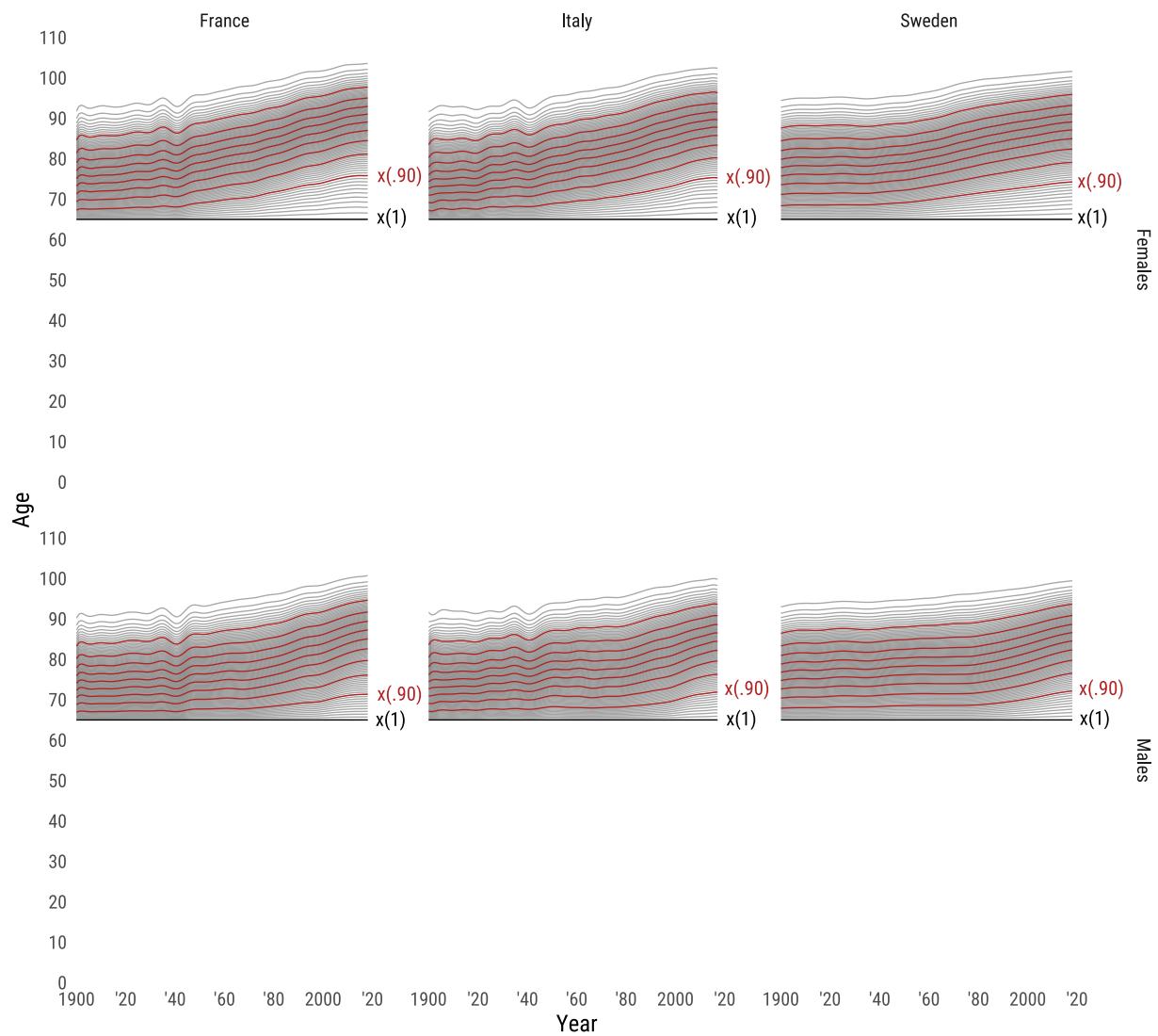


Figure 15: Survivorship ages for France, Italy and Sweden. Females, 1900-2018. Calculations start at age 65, i.e., $x(1) = 65$

Results for other populations included in the study

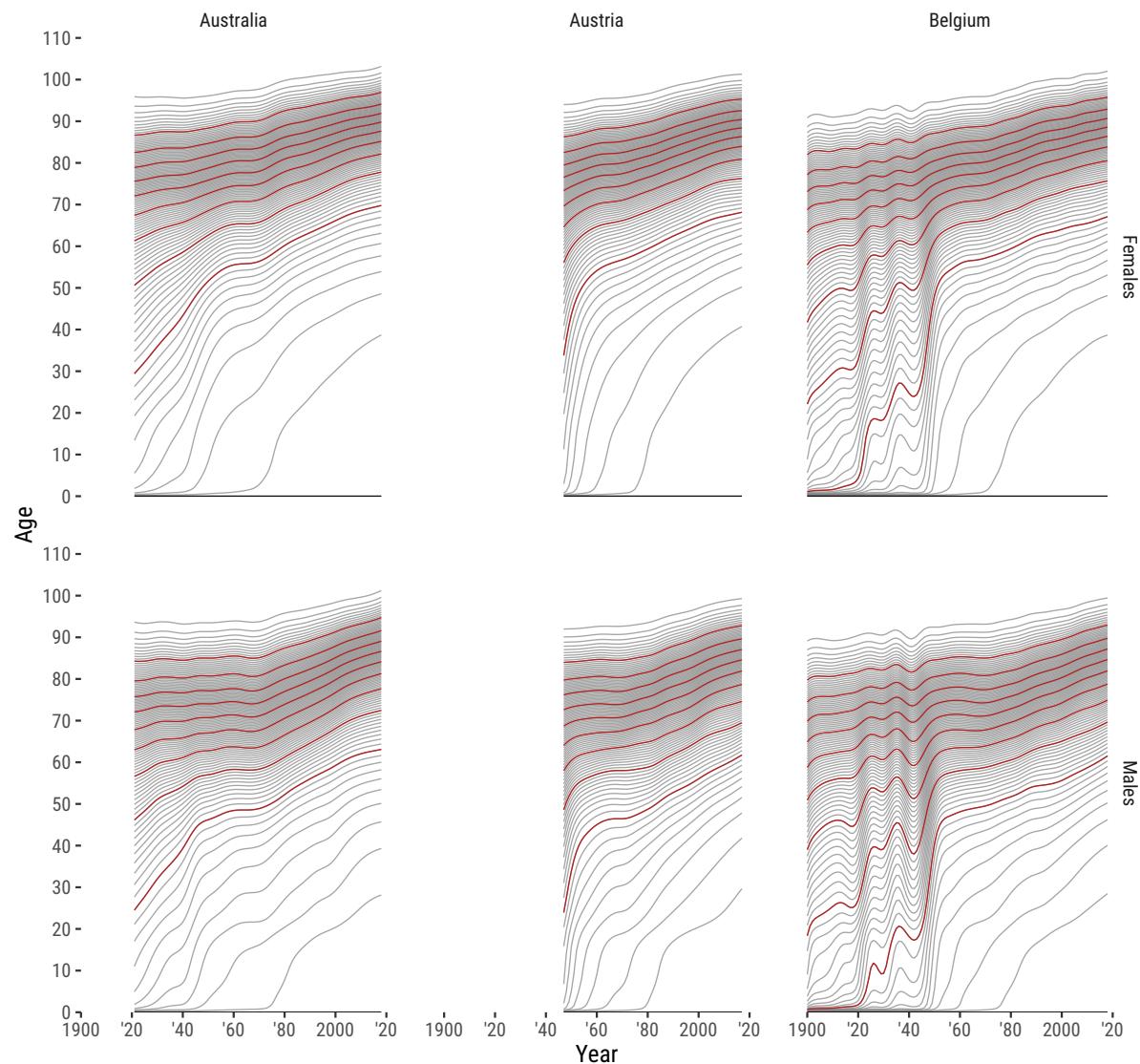


Figure 16: Survivorship ages for Australia, Austria and Belgium, 1900-2018.

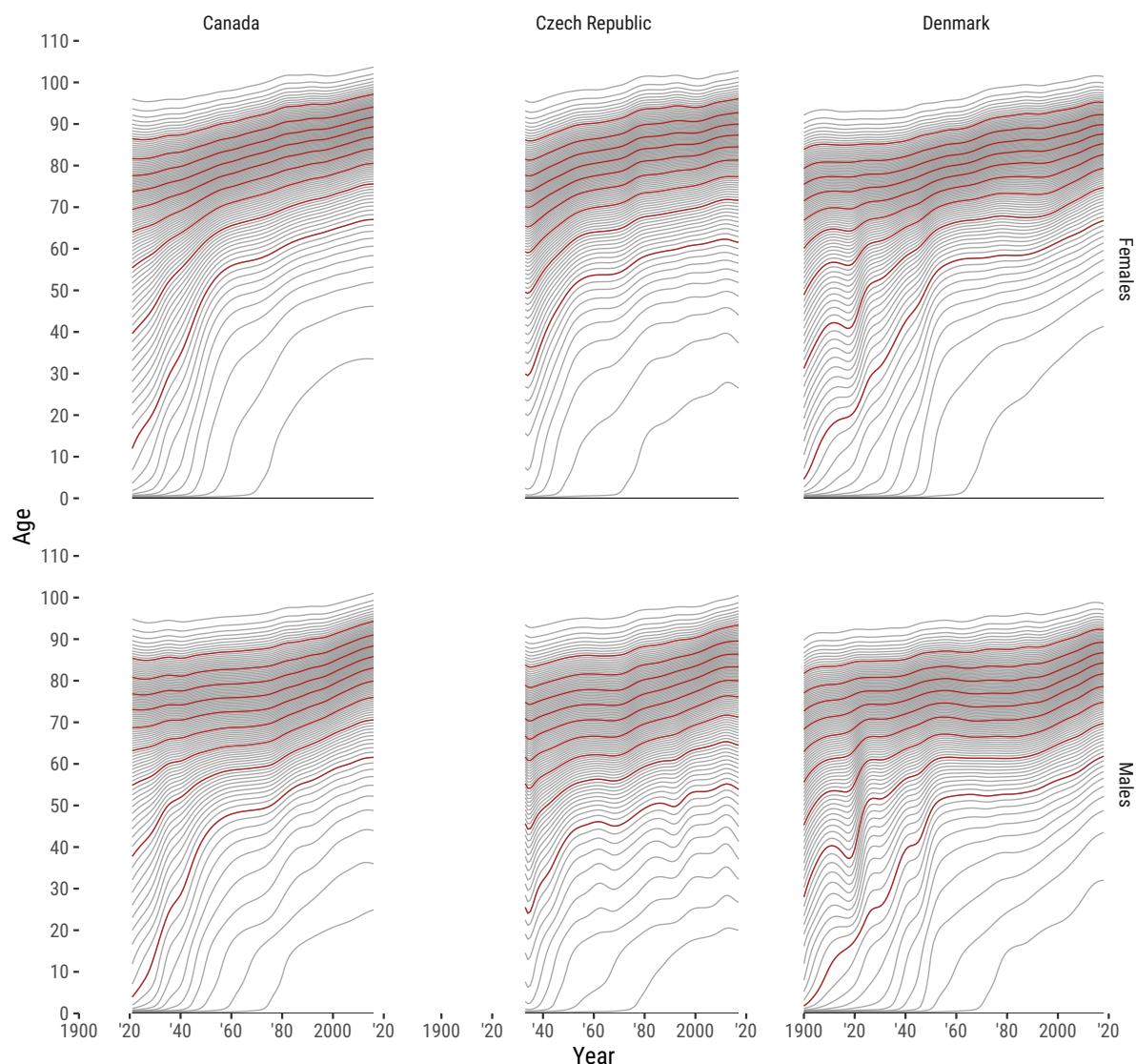


Figure 17: Survivorship ages for Canada, Czech Republic and Denmark, 1900-2018.

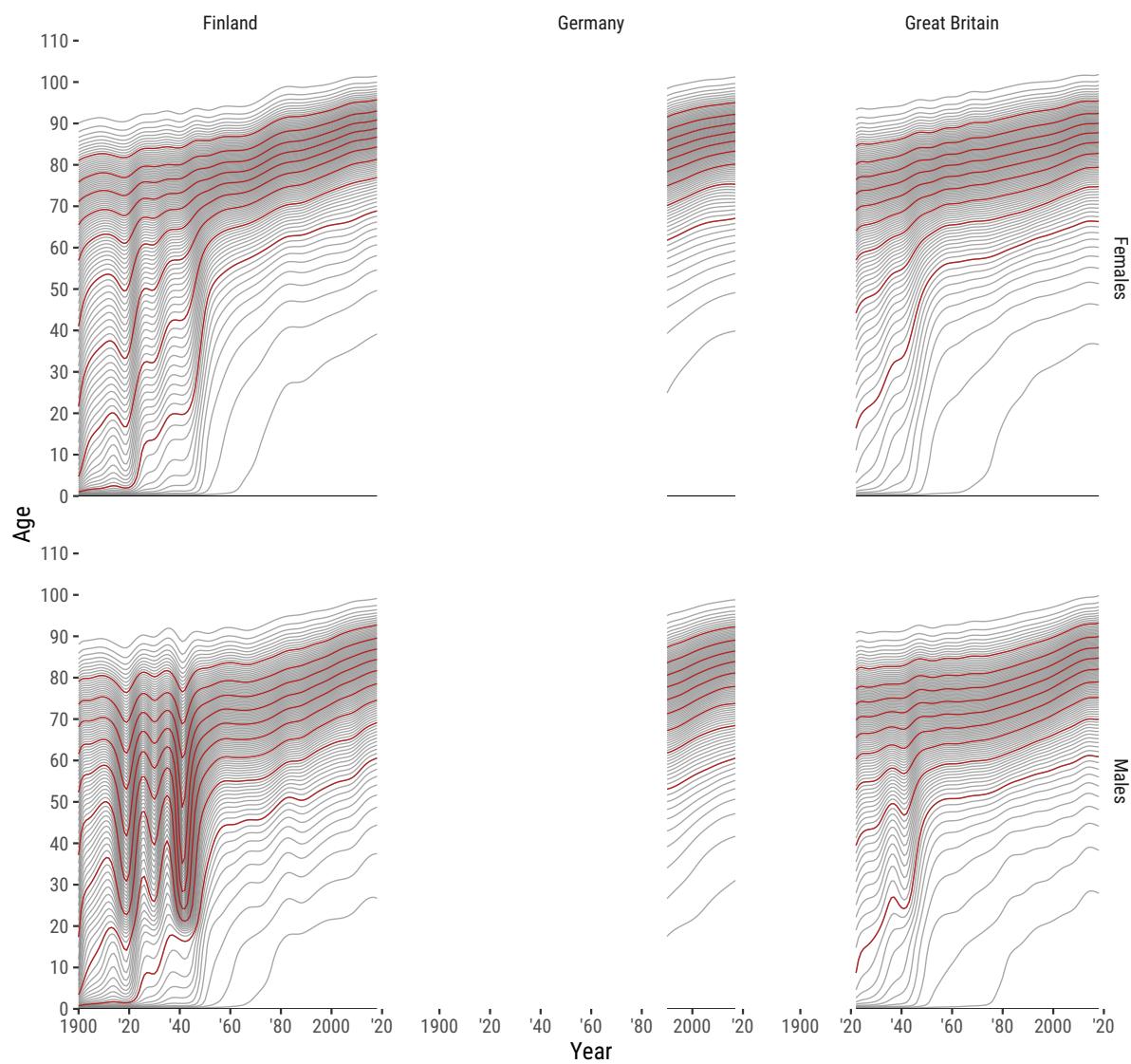


Figure 18: Survivorship ages for Finland, Germany and Great Britain, 1900-2018.

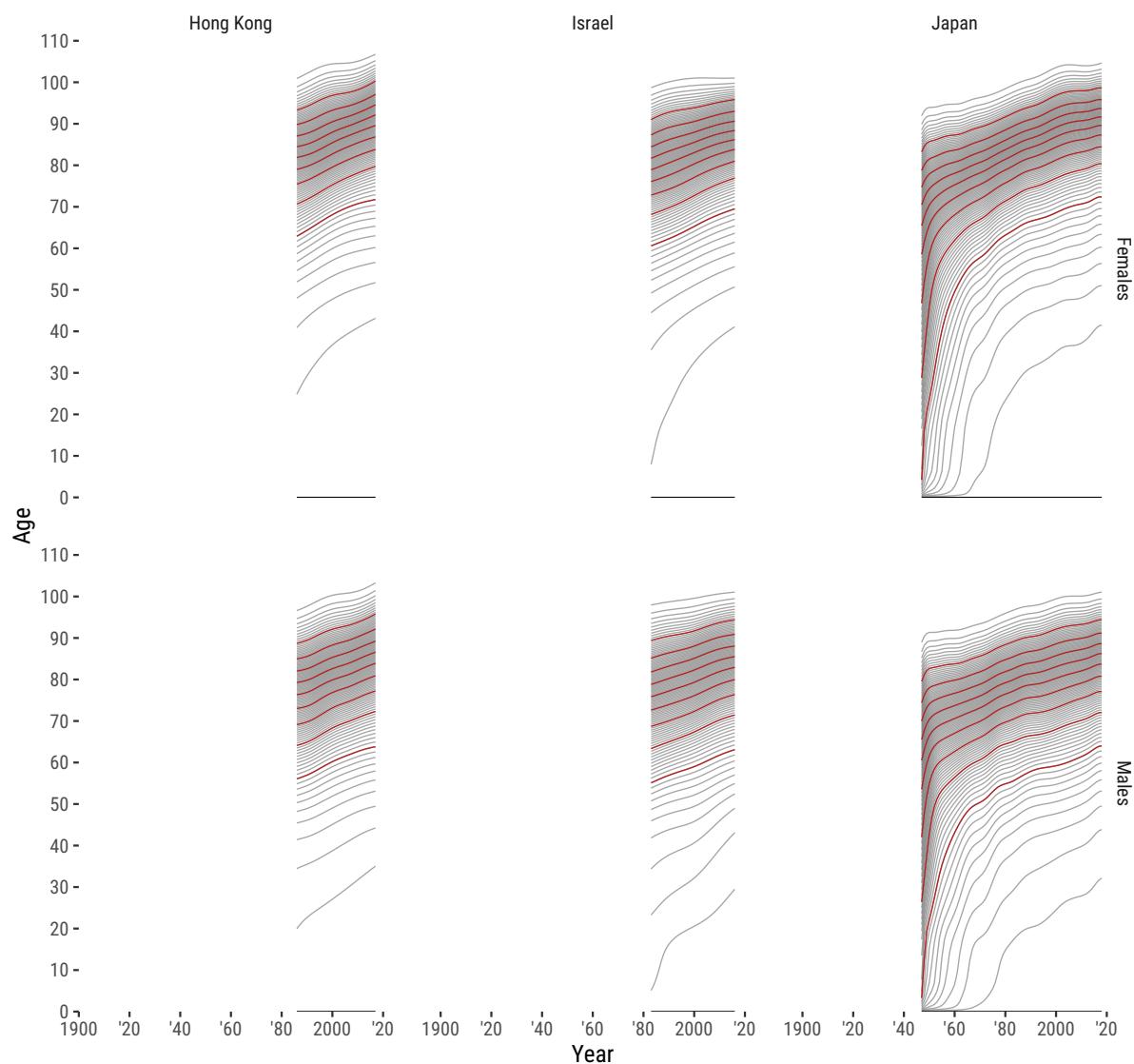


Figure 19: Survivorship ages for Hong Kong, Israel and Japan, 1900-2018.

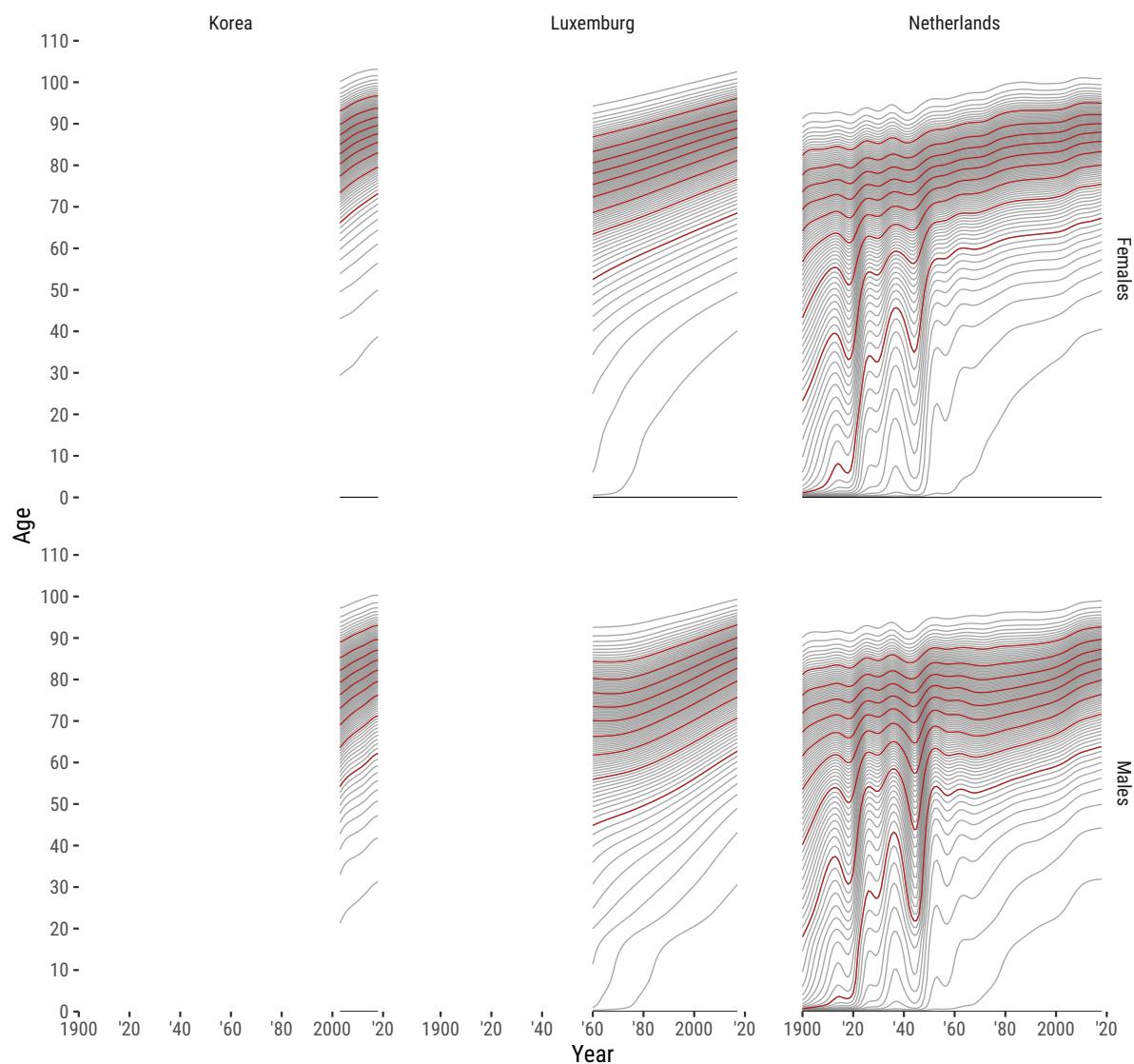


Figure 20: Survivorship ages for Korea, Luxembourg and Netherlands, 1900-2018.

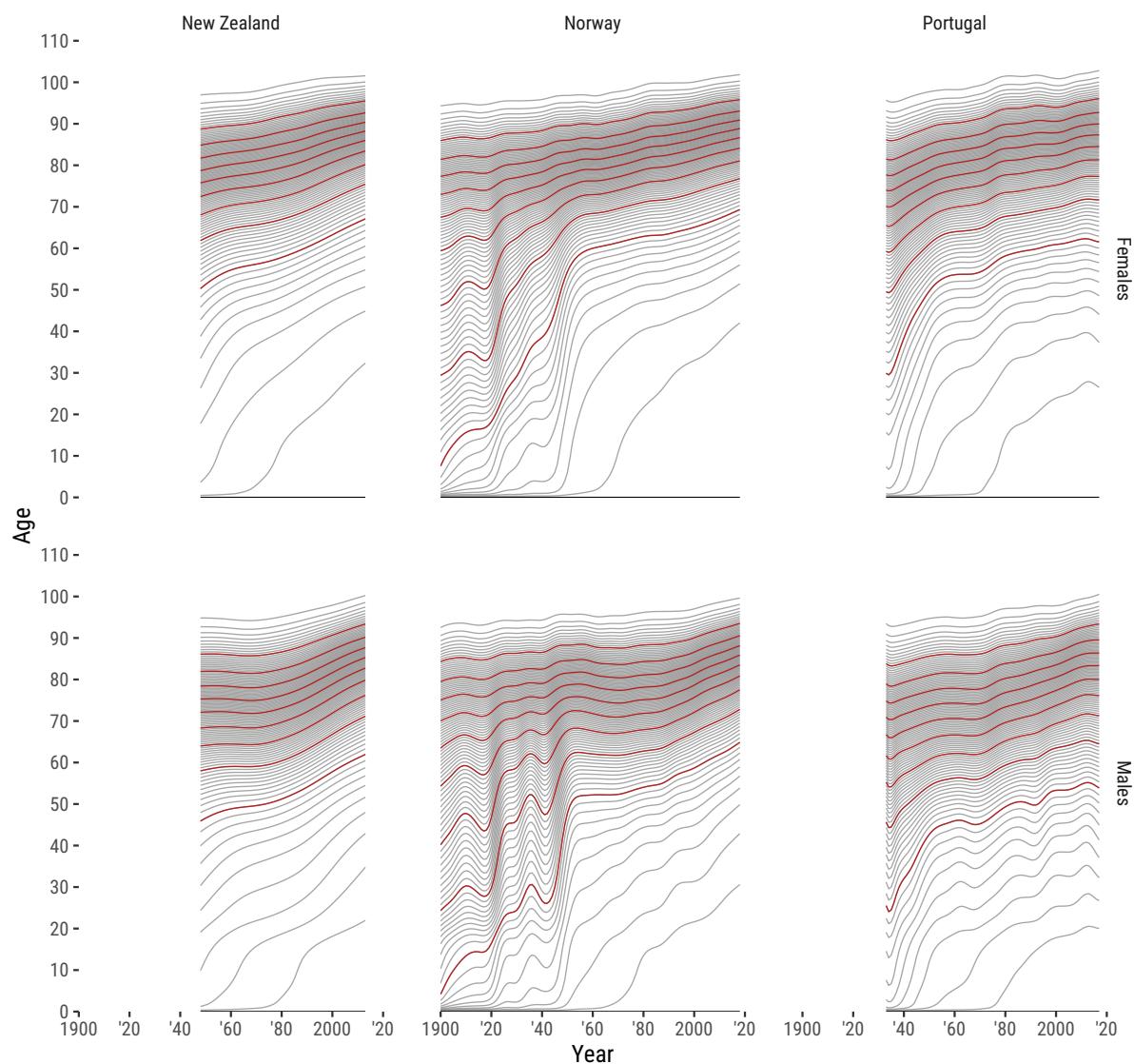


Figure 21: Survivorship ages for New Zealand, Norway and Portugal,, 1900-2018.

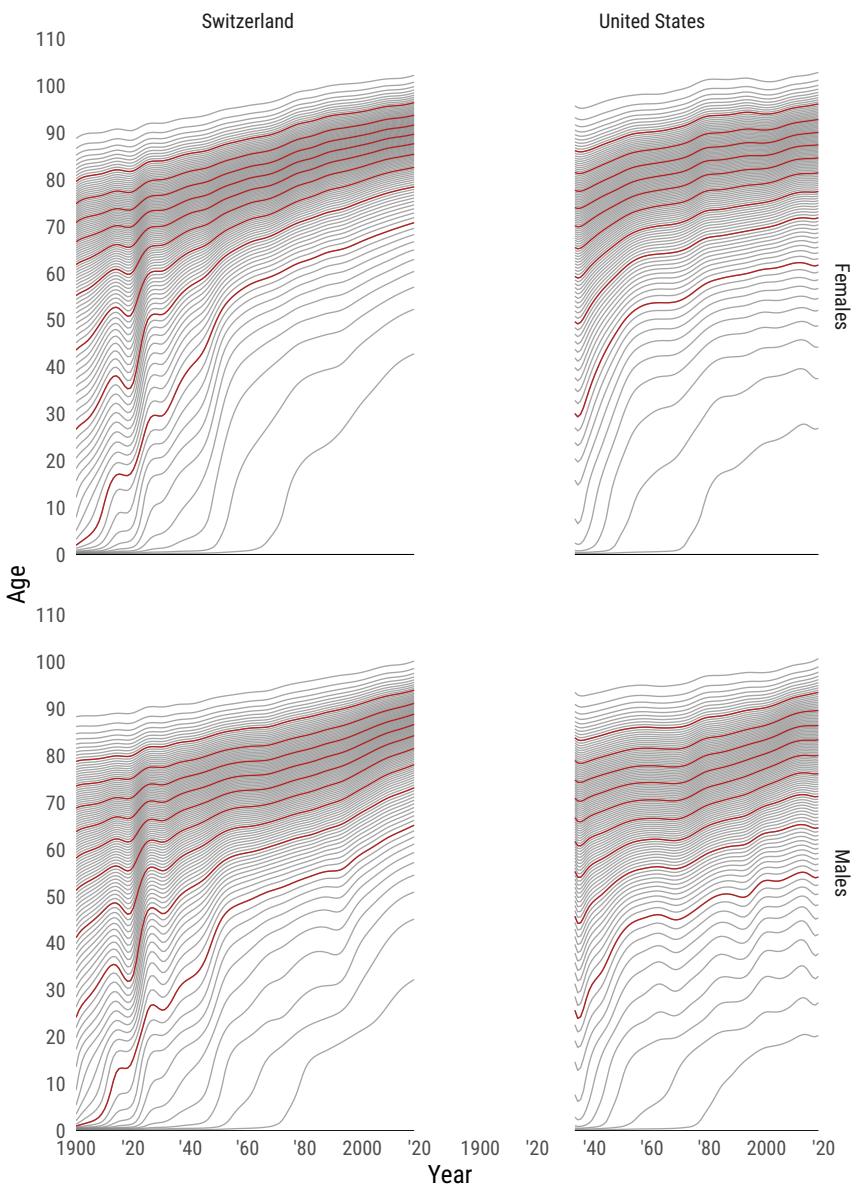


Figure 22: Survivorship ages for Switzerland and United States, 1900-2018.

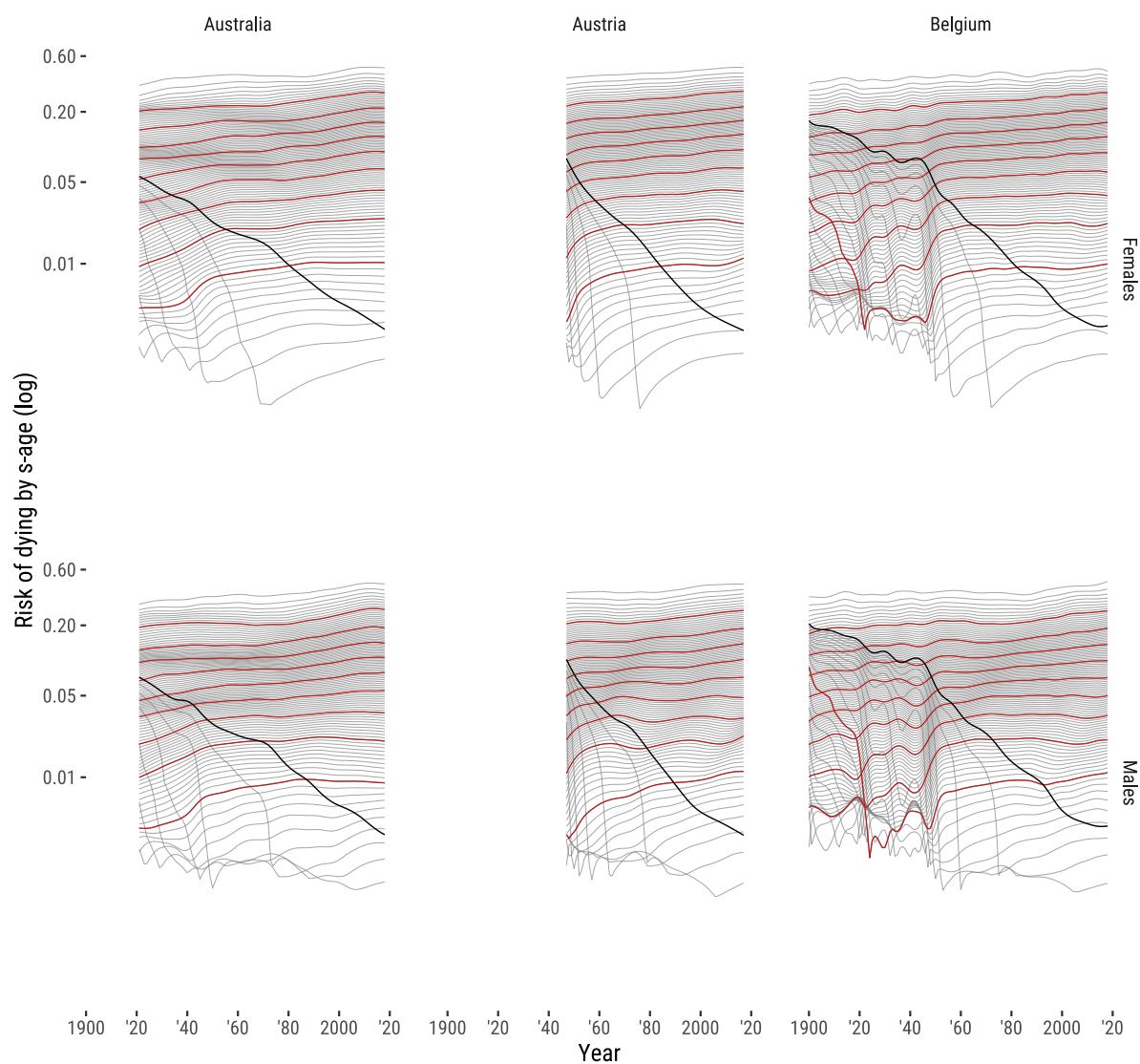


Figure 23: Risk of dying by s for Australia, Austria and Belgium, 1900-2018

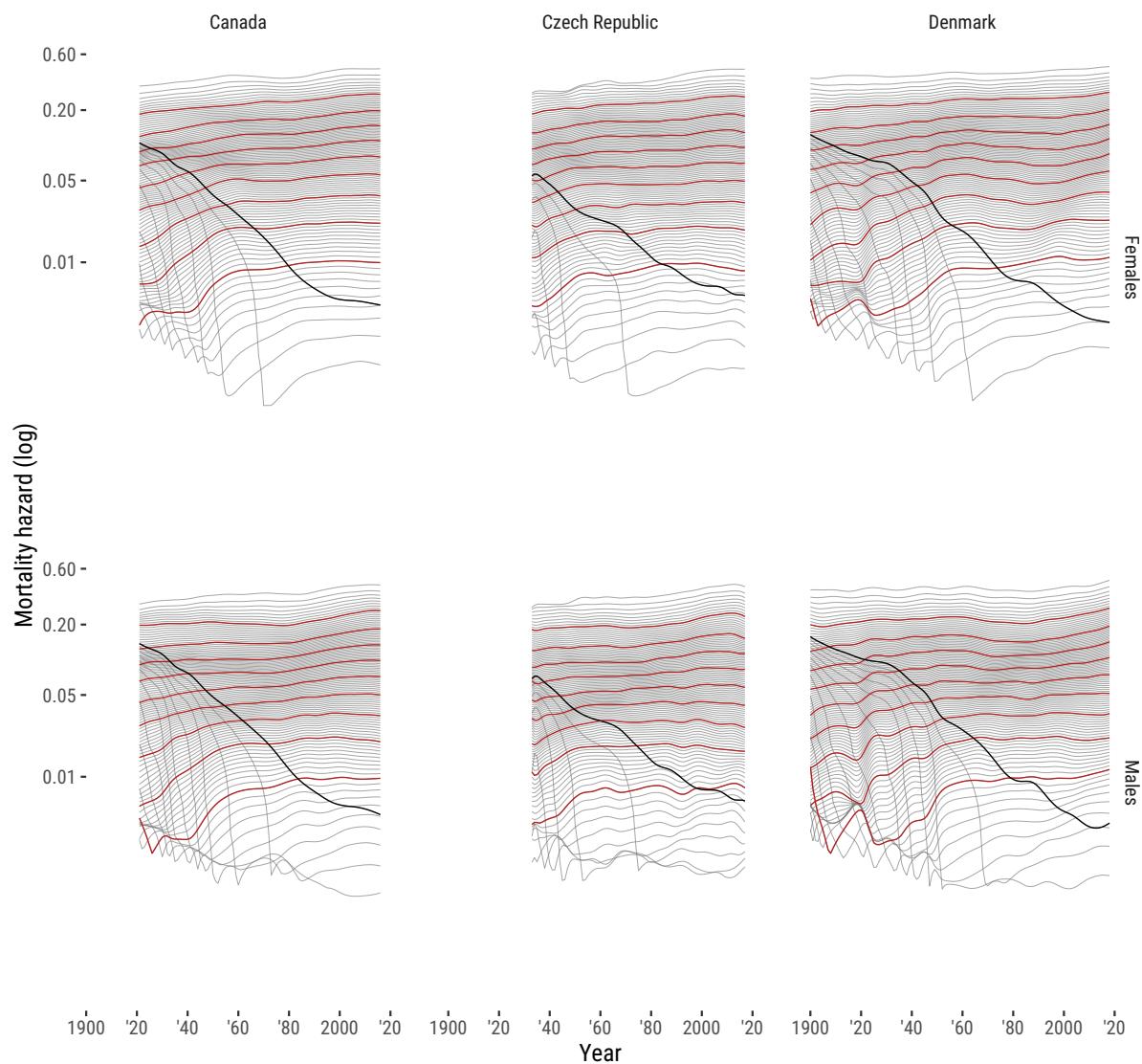


Figure 24: Risk of dying by s for Canada, Czech Republic and Denmark, 1900-2018

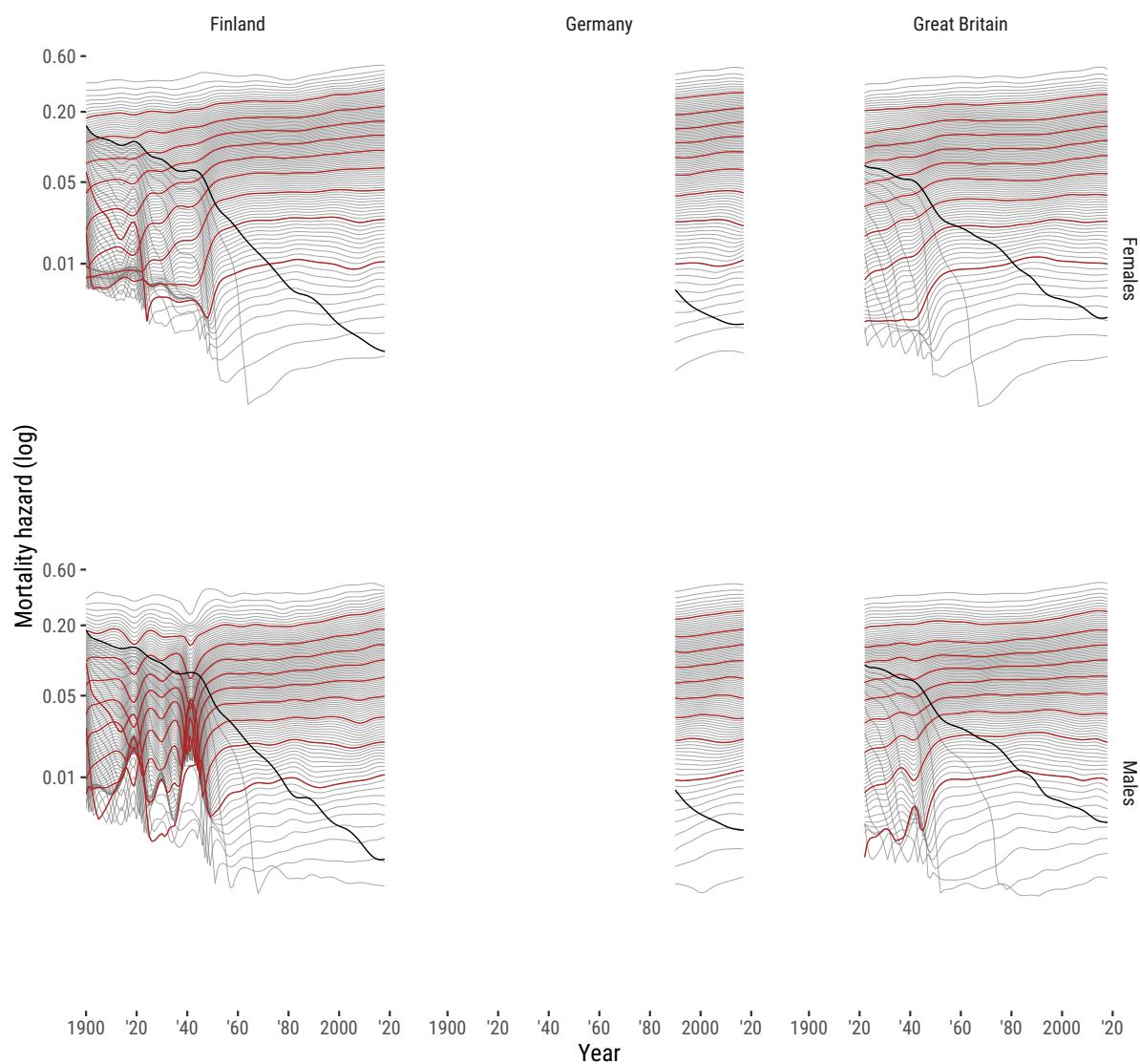


Figure 25: Risk of dying by s for Finland, Germany and Great Britain, 1900-2018

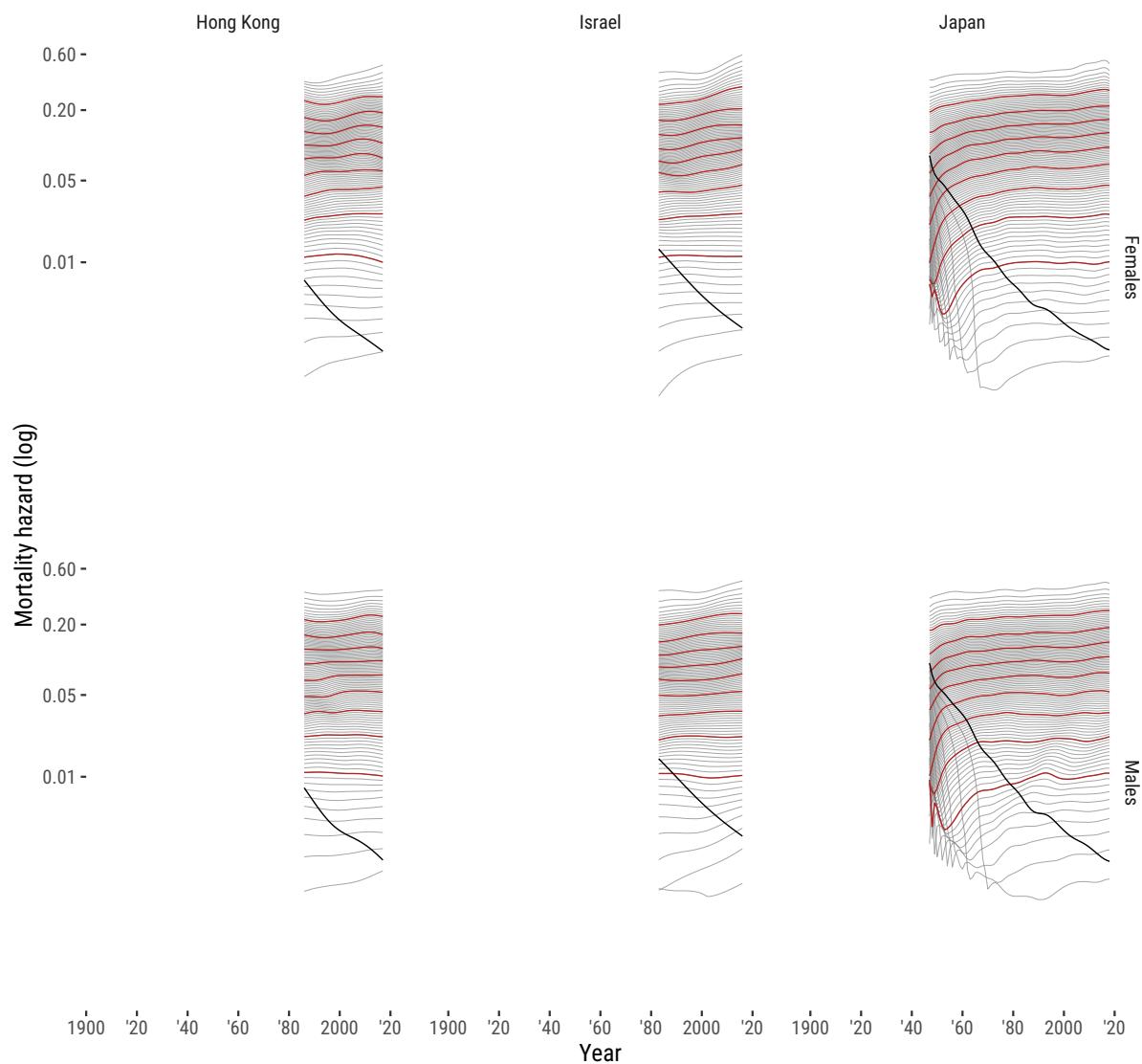


Figure 26: Risk of dying by s for Hong Kong, Israel and Japan, 1900-2018

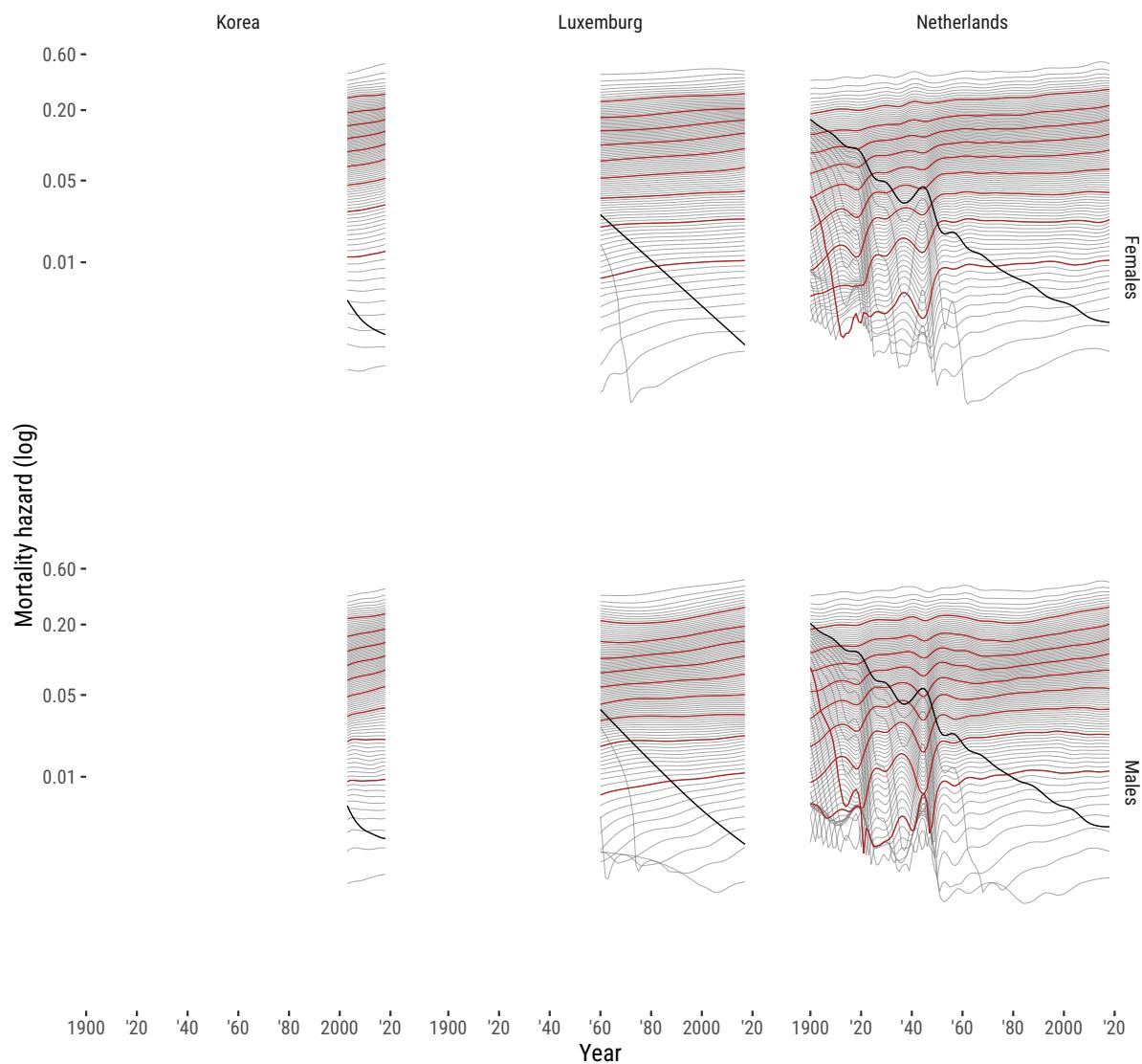


Figure 27: Risk of dying by s for Korea, Luxembourg and Netherlands, 1900-2018

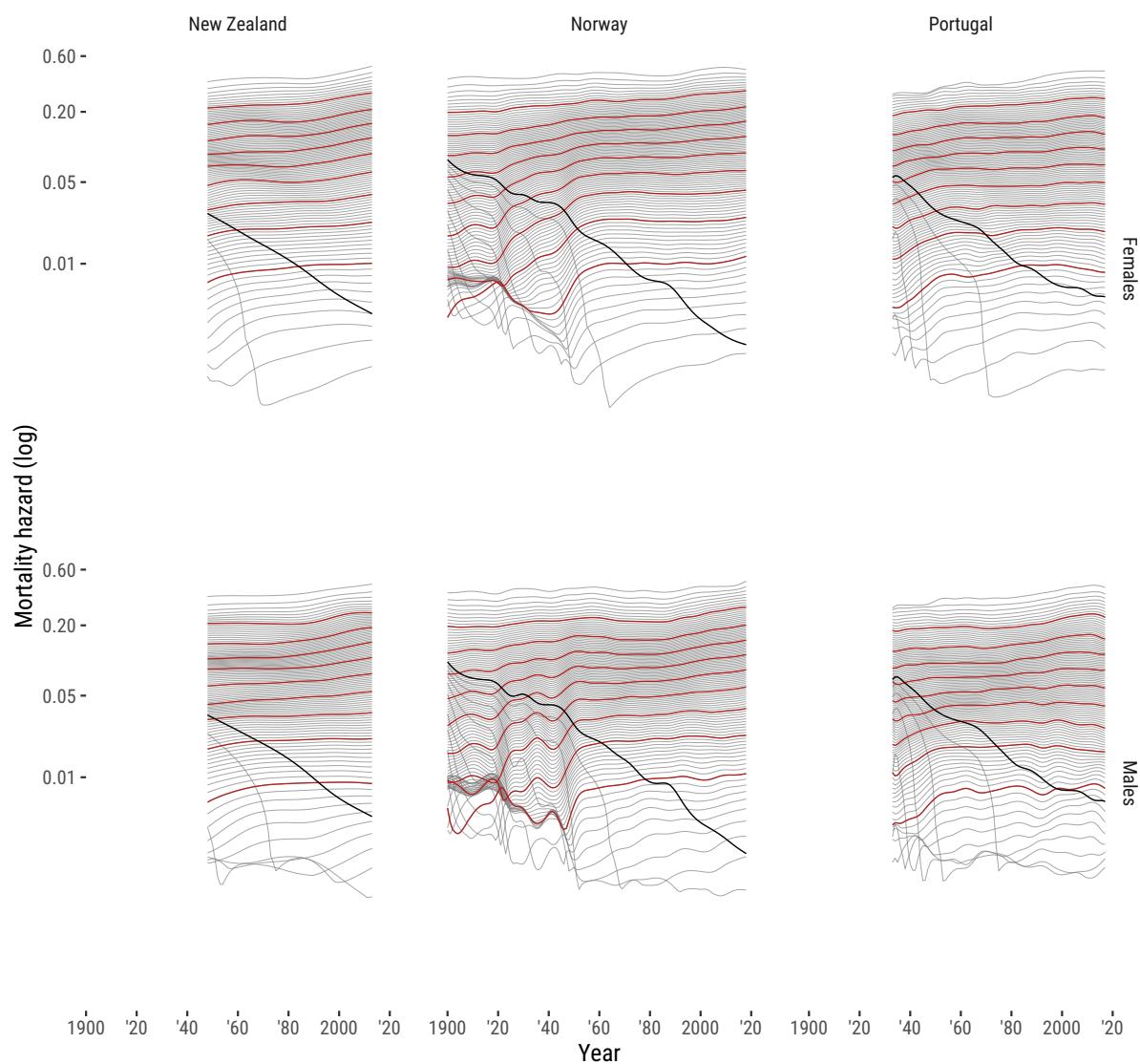


Figure 28: Risk of dying by s for New Zealand, Norway and Portugal, 1900-2018

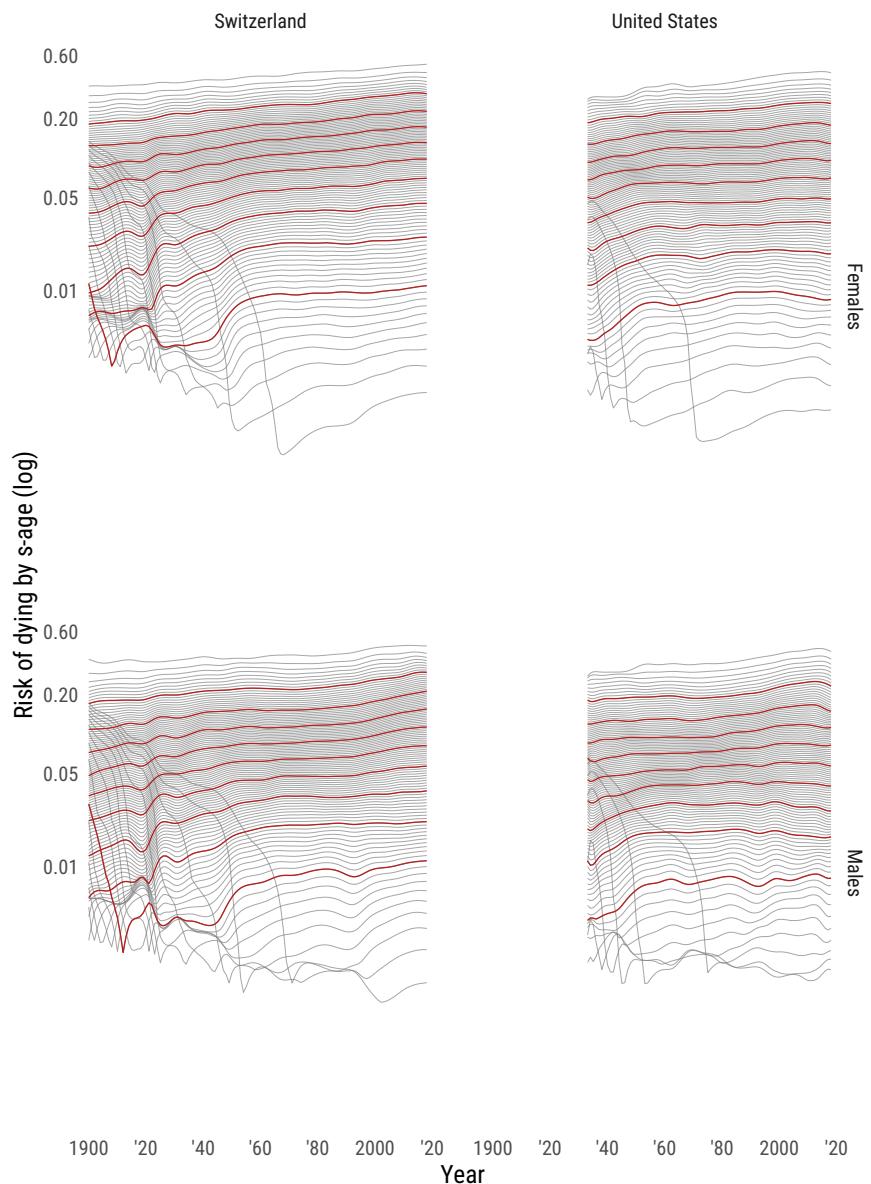


Figure 29: Risk of dying by s for Switzerland and United States, 1900-2018