Measuring mortality trends and dynamics in an era of continuous mortality decline

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Curriculum Vitæ

Education

October 2018: Ph.D. in Demography, University of Rostock, Doctoral Thesis: "Measuring mortality trends and dynamics in an era of continuous mortality decline"

July 2013: European Master in Demography, European Doctoral School of Demography, Universitat Autónoma de Barcelona and Center d'Estudis Demográfics, Thesis: "Why do humans suffer exceptional high levels of senescence – A cause of death analysis based on the pace and shape of aging"

September 2012: Master of Science in Demographie, University of Rostock, Master Thesis: "Rectangularization of the survival function"

September 2010: Bachelor of Arts (Majors: Demography and Economics), University of Rostock, Bachelor Thesis: "Säuglingssterblichkeit in den USA"

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List of Original Publications

The core of this thesis are five research articles. Additionally, two articles are appended to the thesis, which evolved while working one the main articles.

- MARCUS EBELING, FREDERIK PETERS, ROLAND RAU (under review): The concept of equivalent time as a simple indicator for the assessment of survival progress. Under review in **Population Health Metrics**

- MARCUS EBELING (2018): How has the lower boundary of human mortality evolved and has it already stopped decreasing?. Forthcoming in **Demography**

- MARCUS EBELING, ROLAND RAU, ANNETTE BAUDISCH (2018): Rectangularization of the survival curve reconsidered: The maximum inner rectangle approach. **Population Studies**, Online first (DOI: 10.1080/00324728.2017.1414299)

- MARCUS EBELING, KARIN MODIG, ANDERS AHLBOM, ROLAND RAU (2018): The effects of increasing longevity and changing incidence on lifetime risk differentials: a decomposition approach. **PLoS ONE**, Vol. 13(4): e0195307. (DOI: 10.1371/journal.pone.0195307)

- CHRISTINA BOHK-EWALD, MARCUS EBELING, ROLAND RAU (2017): Lifespan Disparity as an Additional Indicator for Evaluating Mortality Forecasts. **Demography**, Vol. 54: pp. 1559–1577 (DOI:10.1007/s13524-017-0584-0)

Appended articles:

- ROLAND RAU, MARCUS EBELING, FREDERIK PETERS, CHRISTINA BOHK-EWALD,

TRIFON I. MISSOV (2017): Where is the level of the mortality plateau. **2017 Living to 100 Monograph**, Society of Actuaries, pp. 1–22

- MARIE-PIER BERGERON BOUCHER, MARCUS EBELING, VLADIMIR CANUDAS-ROMO (2015): Decomposing changes in life expectancy: Compression versus shifting mortality. **Demographic Research**, Vol. 33: pp. 391–424 (DOI:10.4054/DemRes.2015.33.14)

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Executive summary

Background: Three key observations have been made about the mortality changes of the last century and a half. First, mortality has been improving continuously. Second, the age pattern of mortality improvements has been changing. Third, there are additional opportunities for longevity extension at the highest ages. We face ongoing challenges and questions when seeking to measure and understand the pattern and consequences of the mortality dynamics that underlie these findings. The individual articles of this thesis address these specific challenges and questions.

Objectives: Based on these three key findings, four thematic clusters have been derived. The first cluster addresses the issue of how survival progress is measured, and proposes an alternative measure for this purpose. The second cluster refers to the mortality dynamics at the upper and the lower boundary of age-specific mortality, and thus aims to shed light on different aspects of these dynamics, such as on the levels and locations of these boundaries. The third cluster poses methodological questions about how best to measure and explain life expectancy changes and lifespan variability, as well as about the extent to which increasing longevity affects the lifetime risk of contracting a disease in the context of changing age patterns of mortality improvement. Recognizing the unused potential for longevity extension at the highest ages as well as the different patterns of lifespan variability since the 1950s, the fourth cluster aims to show the benefits of using lifespan variability to evaluate mortality forecasting approaches.

Outcomes: The individual articles present novel measures as well as empirical applications.

Cluster one introduces a new measure, which translates differences in the level of survival improvements between a population and a reference into a lag, expressed in calendar time. The cluster also includes a proposal for a classification of different settings for the comparison of populations, and discusses their respective shortcomings. *Cluster two* illustrates the tremendous decline in the lower boundary of age-specific mortality, as well as its shift to younger and younger ages over time. Recent trends also suggest that these developments are likely to continue, at least in the near future. Moreover, the analysis of the maximum levels of age-specific mortality produces slightly higher levels than those estimated in previous publications. *Cluster three* introduces a refined analysis framework for the rectangularization of the survival curve, as well as two different decomposition methods for the contributions of mortality dynamics to life expectancy change, and for the contributions of longevity increase and disease incidence to lifetime risk differentials. The evaluation of mortality forecasting approaches in *cluster four* shows that the tested approaches perform fairly well in capturing life expectancy levels, but that they have difficulties capturing the related lifespan variability pattern, especially in situations in which life expectancy is changing as lifespan variability is stagnating or increasing.

Conclusion: Mortality changes over the last one and a half centuries are associated not just with an increase in the length of the average human lifespan, but with an expansion in the range of age-specific mortality and shifts in the dynamics of these trends. The enormous life expectancy gains that have been made, and particularly the development of the lower boundary of age-specific mortality, suggest that there is still enormous room for improving mortality at all ages. However, the extent of the changes at the lower boundary should also motivate us to consider potential "failures of success" as consequences of the advancement in the past. When assessing survival progress, it is clear that in many instances, the patterns and the speed of improvement are often more informative than the actual levels of advancement, especially when analyzing key characteristics of mortality, such as life expectancy or infant mortality. The proposed approaches for measuring the dynamics driving mortality changes highlight the multiple ways in which lifespan variability and life expectancy have been related since the on-

set of sustained mortality decline at older ages. Given the still large opportunities for longevity extension, this topic will remain a key issue for mortality research in the future. The evaluation of mortality forecasting is of prime importance in this context, because it highlights how problematic deviations from the usual patterns can be. In addition to providing methodological insights, the outcomes of this thesis shed light on related topics, such as the role distributive justice plays in further reducing mortality in a population, and the shape of progress in populations with lagging levels of advancement. The methods proposed in this thesis should prove useful in addressing such questions.

1. Background

1.1. The continuity of mortality improvement

The average number of years that humans can expect to live has been increasing for more than one and a half centuries. Prior to the sustained increase in life expectancy, the global average lifespan fluctuated at around 30 to 35 years (Riley, 2005). Currently, Japanese females are living longest, with an average lifespan of almost 87 years. The rise in life expectancy has been occurring with a remarkable degree of regularity. Based on the highest annual life expectancy observed, Oeppen and Vaupel (2002) have suggested that the increase is best illustrated by a straight line starting around 1840 that increases by approximately a quarter year per year. Other authors have proposed depicting the increase of life expectancy using a segmented trend line with three distinct development periods (Vallin and Meslé, 2009). Regardless of whether life expectancy improvement has been occurring at a single or at different speeds, there is extensive evidence of the striking regularity of the historic increase in longevity (among others White, 2002; Riley, 2001; Colchero et al., 2016).

Figure 1.1 visualizes the continuity of mortality improvements. The graph depicts period estimates for life expectancy at birth for each country with at least one year of data in the Human Mortality Database (2017a) between 1850 and 2015. To approximate some general trends, the annual average across all countries with data in the respective year is highlighted with a thick solid line. Estimates for males are in blue and estimates for females are in red.

Figure 1.1 illustrates the tremendous increase in life expectancy at birth. Based on the av-

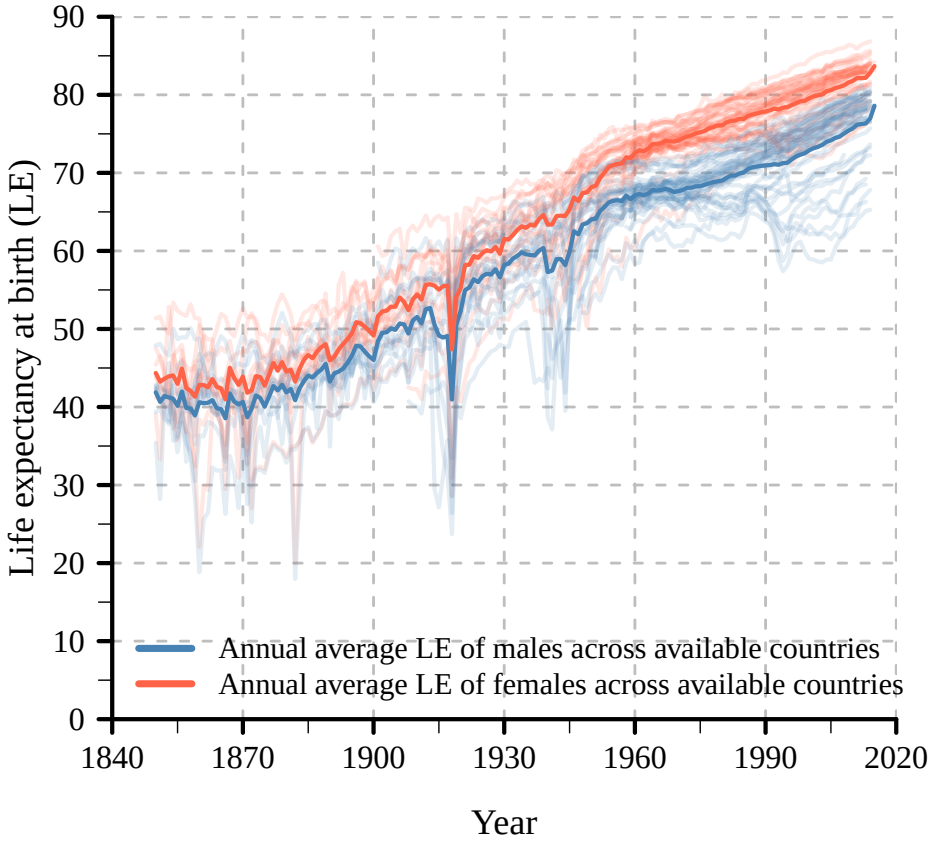

Figure 1.1.: Life expectancy at birth, males and females, various countries, 1850-2015. *The graph includes estimates for all countries included in the Human Mortality Database (2017a) with at least one year of data between 1850-2015. The annual average is calculated across all countries, providing a life expectancy estimate in the respective year. Hence, the number of countries included in the average varies by year. Life expectancy estimates are based on period life tables, using age-specific death counts and exposures provided by the Human Mortality Database (2017a). The abbreviation "LE" stands for life expectancy.*

erage value across countries, the average lifespan almost doubled between 1850 and the most recent years. Despite short-term fluctuations and the influence of strong period effects, such as the Spanish flu (1918–1920) and the World Wars (1914–1918, 1939–1945); it is clear that

survival has been continuously improving since at least 1870. However, if we look at the annual cross-country average, we see that the pace of improvement started slowing around 1950. This trend is likely attributable to the onset of sustained old-age mortality declines (Kannisto, 1994).

Several authors have emphasized the commonalities in the patterns of survival improvement across countries. Tuljapurkar et al. (2000), for instance, have provided evidence of a roughly constant rate of exponential mortality decline across the G7 countries. Taking a broader perspective, Leon (2011) pointed out that the pace of the life expectancy increase has been similar across western Europe, Japan, and the USA; but also that some deviating trends can be observed in regions such as eastern Europe. Using a joint perspective, White (2002) showed that the variability in the level of life expectancy across countries has been decreasing over time. Additionally, he argued that the earliest industrialized countries have shared a stable rate of change and a stable degree of variability of life expectancy levels at least since around 1980. He concluded from these findings that the countries included in his analysis increasingly appear to have a common mortality trend. While acknowledging the occurrence of short-term fluctuations, Wilmoth (1998) also emphasized the remarkable stability and regularity of improvements. The expectation that past trends will continue in the future prompted him and other scholars to simply extrapolate past trends to forecast future levels of life expectancy (Wilmoth, 1998; Torri and Vaupel, 2012).

Over a long run, the general trend in life expectancy and the life expectancy trends of almost all of the countries in Figure 1.1 are remarkably regular. Nonetheless, there is still a considerable degree of inequality in life expectancy levels. For instance, Ribeiro et al. (2016) provided evidence for spatial inequalities in old-age survival across European regions. They observed a north-south pattern with higher life expectancy in the southern regions. Moreover, some scholars have described a divergence of old-age mortality declines across low-mortality countries (Meslé and Vallin, 2006; Rau et al., 2008). According to Meslé and Vallin (2006), the trends in the USA and the Netherlands and those in France and Japan can be seen as ex-

emplary groups with differing trends. By analyzing the time point at which countries started to diverge from some general trend, Ouellette et al. (2014) and Li et al. (2011) found that most trend breaks occurred between 1970 and the end of the 1980s. Peters et al. (2016) argued that most of these trends breaks can be explained by the smoking epidemic. Moreover, Thun et al. (2012) found that the four-stage model of the smoking epidemic, which was originally published by Lopez et al. (1994), provides a useful description of the mortality trends in many developed countries. It is therefore possible to speculate that since 1950, tobacco smoking has been the main driver of the variations in mortality trends across countries. However, in some eastern European countries in particular, alcohol consumption and other factors have also had considerable effects on the changes in life expectancy (McKee and Shkolnikov, 2001).

Innovations and changes in nearly every branch of life are responsible for the marked increase in the average lifespan. According to Cutler et al. (2006) the factors that have contributed to rising longevity include better nutrition, improvements in public health, urbanization (albeit with some negative effects), vaccination, better medical treatments, and the long-term effects of improvements in early-life conditions. Advances in education, welfare, and infrastructure are other potential determinants of the increase in the average lifespan (Riley, 2001). Researchers have also found correlations between life expectancy and a number of other development indicators, such as gross domestic product (Preston, 1975; Mackenbach and Looman, 2013). It is, however, unclear, which of these determinants have been and currently are the most important. Scholars have observed a complex interplay of all of these factors with variations over age, period, and cohort (Oeppen and Vaupel, 2002; Riley, 2001). An attempt to create a theoretical framework for these fundamental changes was made by Fogel and Costa (1997). They proposed the theory of technophysio evolution. This concept relates the alteration of human physiology to the steady increase in environmental control that has occurred over the last 10 generations, and which has been particularly pronounced in the last three to four generations. Fogel and Costa (1997) characterized this evolution as a long term and still ongoing process.

In addition to the determinants responsible for progress across populations, factors that influence mortality within populations have become increasingly important. Health care systems, medical care, resource allocation, differences in health-related behaviors, social structure, and stress seem to have driven recent and current mortality developments within and across countries (Cutler et al., 2006; Marmot, 2005). The emergence of modern epidemics, such as the smoking epidemic, have slowed or even reversed mortality improvements (Thun et al., 2012; Lopez et al., 1994). Although being overweight is probably not as deleterious to health as smoking tobacco, the obesity epidemic appears to be threatening further health and mortality improvements in developed countries (Prentice, 2005; Caballero, 2007).

The enormous number of years added to the average lifespan also raises questions about the health status of people during their additional years of life. Are these years spent in poor or good health? Three competing hypotheses that attempt to answer this question have been proposed. The compression of morbidity hypothesis (Fries, 1980) argues that these additional years are healthy years: The dynamic equilibrium hypothesis (Manton, 1982) assumes that the proportion of the lifespan spent in poor health remains the same. Finally, the expansion of morbidity hypothesis (Gruenberg, 1977; Kramer, 1980) argues that the additional years or life are spent in poor health. Although many studies have favored the compression of morbidity theory, the research outcomes on this issue diverse (Chatterji et al., 2015; Vaupel, 2010). Thus, it has recently been asserted that the question of whether the additional years are spent in good or in poor health is the next million-dollar question in demography [1]. There are also different hypotheses about the influence of improved survival on the general health status of the population. For example, the failure of success hypothesis claims that medicine has saved the lives of frail individuals in the population in particular, and that this has in turn resulted in an increasingly unhealthy population (Gruenberg, 1977; Rosen and Haglund, 2005). The counterpart to this idea could be called the success of success hypothesis, which asserts that the general health status of the population has been improving. However, given the contro-

[1] http://demogr.mpg.de/en/news_press/news/news/the_next_million_dollar_question_in_demography_4802.htm

versies surrounding health status during the additional years of life, the questions of mortality decline and population health are linked is similarly debatable.

In light of the expectation of further advances in the various forces of improvement and the recent mortality trends in developed countries, it is unlikely that the rise in life expectancy will halt in the near future (Vaupel, 2010; Mathers et al., 2015). Nevertheless, what the levels and the limits of the average and the maximum lifespan will turn out to be are questions that continue to be debated. Although Oeppen and Vaupel (2002) have shown that many suggested limits have already been exceeded in the projected year, opinions about the future of human life expectancy still range from limited to limitless. For instance, Olshansky et al. (1990) discussed the evidence in favor of the assumption that life expectancy is limited. They argued that unless major breakthroughs occur, life expectancy is unlikely to exceed 85 years. However, in more recent publications, the same authors have revised their view on the limits of life expectancy (Olshansky et al., 2005). By contrast, De Grey (2006) offered a highly optimistic, almost limitless view on the future of longevity. Based on extrapolations of scientific progress and political responses, he argued that the cohorts born in the 21st century are likely to achieve a life expectancy of more than 1000 years. A much more pessimistic perspective was presented in a recent article by Dong et al. (2016), who argued that even the maximum lifespan is unlikely to exceed 115 years. However, the results of other recent studies have convincingly demonstrated that these findings are flawed (Hughes and Hekimi, 2017; Lenart and Vaupel, 2017; Rozing et al., 2017; de Beer et al., 2017).

1.2. The changing age pattern of mortality improvement

As populations evolved from having low to high life expectancy, the major drivers of increases in life expectancy shifted from young to old ages. Accordingly, the forces that drove the progress made at the beginning of the life expectancy revolution differ from the factors that

influence life expectancy in the current context (Christensen et al., 2009; Riley, 2001; White, 2002). At the beginning of the revolution, declines in infant and child mortality as well as in mortality among adolescents and young adults made the largest contributions to the increase in the average lifespan. Since the 1950s, however, mortality improvements at older ages are the primary drivers of rising life expectancy levels (Christensen et al., 2009; Kannisto, 1996; Rau et al., 2008). This trends is also a consequence of the continuous mortality decline. As mortality levels across almost all ages have been dropping from one historical minima to the next, the opportunities for further reductions in mortality at younger ages are narrowing. By contrast, there are still enormous opportunities for further improvements in mortality at the highest ages.

Figure 1.2 illustrates the changing effects of mortality reductions at younger and older ages on increases in life expectancy at birth. The graph depicts the relative contributions of mortality declines to increases in life expectancy at birth between 1900 and 1950 (left panel) and between 1950 and 2000 (right panel) for several countries, separated into the age ranges of below and above 30. In each panel, the bars on the left depict the contribution of ages below 30. The bars on the right indicate the contribution of ages above 30. Estimates for males and females are depicted in blue and red, respectively.

On average, around 75% of the increase in life expectancy between 1900 and 1950 was generated by mortality decline at ages below 30. Accordingly, only around 25% of the increase was due to mortality declines above age 30. The results of the decomposition between the years 1950 and 2000 reveals a different pattern: during this period, mortality declines at ages below 30 generated only around 25% to 50% of the increase in life expectancy, whereas mortality declines at ages above 30 contributed around 50 % to 75% to the increase in life expectancy. The period during which mortality improvements at older ages were the main drivers of life expectancy increase has been described by Kannisto (1994) as the “era of sustained old-age mortality decline.” For several countries, Kannisto (1994) also provided time points of the onset of this development. For instance, his analysis showed that this trend started

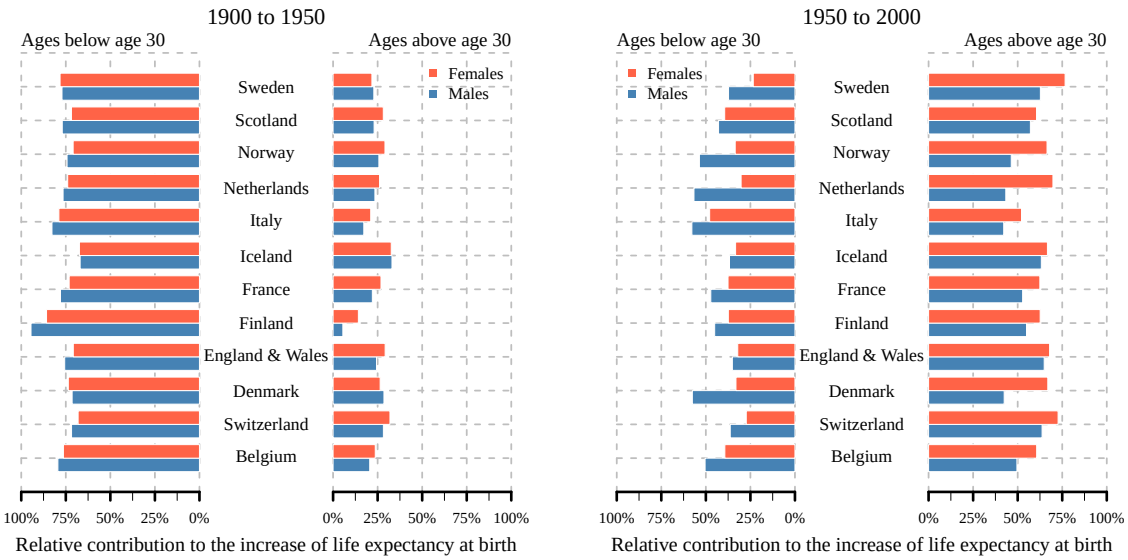

Figure 1.2.: Relative contribution to the increase in life expectancy at birth, males and females, various countries, 1900-1950 and 1950-2000. *The graph depicts all countries that provide data for the years 1900, 1950, and 2000. The decomposition is based on the methodology presented in Arriaga (1984), which is concisely summarized in Preston et al. (2001). The unequal age intervals are justified by the higher sensitivity of life expectancy to mortality changes at younger ages. All of the included countries showed an increase in life expectancy at birth over time. The estimates are based on based on period life tables, using age-specific death counts and exposures provided by the Human Mortality Database (2017a).*

first among females in France (1955 or earlier), Switzerland, Sweden (all 1956), and Denmark (1962); and much later among women in Spain, New Zealand (both 1974), the Czech Republic (1975), Ireland (1979), and Estonia (1981). Looking at males, the analysis found that this trend started first in France (1955 or earlier), Japan (1966), Switzerland (1967), Finland, and Portugal (both 1970); and later in Denmark, Iceland, East Germany (all 1984), Hungary, the Czech Republic (1985), and Estonia (1989). On average across all of the countries analyzed, females experienced the onset in 1968 and males experienced the onset in 1976 (Kannisto, 1994). Vaupel (2010) has noted that as a consequence of improvements in survival at older

ages, the number of centenarians has been increasing exponentially in Sweden and Japan since 1950.

The shift in the age pattern of mortality improvement is attributable to the success of the fight against communicable diseases. During the transition, communicable diseases, such as cholera and other infectious diseases, were replaced as major causes of death by non-communicable diseases, such as cardiovascular diseases and cancer (Riley, 2001). Although many regions have experienced this transition, the timing of the onset and the level of progress achieved differ greatly across the globe. Riley (2005) has dated the start of the earliest health transitions by populations to 1770 in Europe; to the mid 19th century in America, Asia, and Oceania; to the late 19th century in the former Soviet Republics; and to the 1920s in Africa (Riley, 2005). Irrespective of these geographic differences, non-communicable diseases dominate the current global ranking of major causes of death (Feigin et al., 2016). Hence, the fight against non-communicable diseases, such as cardiovascular and other degenerative diseases, is the major driver of current survival progress (Robine, 2001; Vallin and Meslé, 2004). However, despite intensive efforts to reduce mortality from non-communicable diseases, the general process of physical deterioration with age (aging, sometimes also called senescence) remains untouched. Thus, these efforts have simply resulted in the gaining of extra time prior to contracting these diseases through the postponement of senescence to higher and higher ages Vaupel (2010).

Several efforts have been made to conceptualize the shift in the disease and cause-of-death spectrum of humans. The most prominent examples are arguably the epidemiological transition theory by Omran (1971) and, based on this work, the more general theory of the health transition (see, for instance, Riley, 2001; Fogel, 2004)). In the epidemiological transition theory, Omran (1971) described three distinct periods of change during the development from low to high life expectancy. In the first period, mortality is high and fluctuating due to, for instance, pandemics such as plague (Walter and Schofield, 1989). The second period marks the onset and the continuation of mortality improvements and increasing life expectancy, which

are generated by success in fighting infectious diseases. In the third period, life expectancy levels exceed age 70 and non-communicable diseases are the major killers. The epidemiological transition is an appropriate description of the mortality change up until the 1960s/70s. However, an unprecedented reduction in cardiovascular mortality, which is commonly referred to as the "cardiovascular revolution" accelerated this progress (Vallin and Meslé, 2004; Frenk et al., 1991). Together with the delaying of the onset of other degenerative diseases, this development marks a fourth stage of a general health transition: namely "the age of delayed degenerative diseases" (Olshansky and Ault, 1986; Robine, 2001).

The changes in the age pattern of mortality improvement also led to shifts in the lifespan distribution. Taking a long-term perspective, we can see that increasing life expectancy has been accommodated by decreasing lifespan variability (Colchero et al., 2016). As a consequence of mortality improvements that have been occurring mainly at older ages, new and divers patterns of changes in lifespan variability have been emerging that could result in a decoupling of this long-term relationship (Vaupel et al., 2011; Smits and Monden, 2009). Deaths are being postponed to older ages, while mortality at younger ages is declining. This pattern results in decreasing lifespan variability and increasing life expectancy because the old-age mortality hump in the lifespan distribution becomes more and more compressed. In his canonical work, Fries (1980) used the term "compression of mortality" to describe these dynamics. However, with the onset of the decline in old-age mortality, other dynamics became prevalent. A "shifting of mortality" has been observed, whereby deaths are postponed to higher ages, but with an approximately constant shape of the lifespan distribution (Kannisto, 1996; Bongaarts, 2005; Canudas-Romo, 2008). When a shifting of mortality occurs, life expectancy increases alongside constant lifespan variability. Moreover, it has been suggested that an expansion of mortality at higher ages is possible (Rothenberg et al., 1991; Cheung et al., 2005; Engelman et al., 2010). In this situation, life expectancy would increase alongside an increase in lifespan variability due to a shift in the right tail of the lifespan distribution to higher and higher ages, which is in turn caused by a dispersal of the old-age mortality hump.

1.3. The potential for further increases in longevity

Due to the continuity of mortality improvements as well as the changing age pattern of these improvements, opportunities for the further extension of life expectancy and longevity have become increasingly concentrated at the highest ages. Figure 1.3 illustrates the current situation in various countries. The figure depicts the average number of person-years lived in the age intervals 0–69 and 70–109 (partial life expectancy) relative to the respective number of maximum possible life years in both age intervals; 70 and 40 years, respectively. For each country, the last year with available data is used, which ranges from 2010 to 2015. Estimates for females and males are shown in red and blue, respectively. Looking at the figure, we can see that the populations of almost all of these countries are living close to 100% of the maximum possible years between ages zero and 69. Accordingly, only marginal additional gains in the number of life years could be achieved among people in this age group. But we can also see that the populations of the analyzed countries are using only 25% to 50% of the maximum possible years between ages 70 and 109. Accordingly, it is clear that any increases in life expectancy, and in maximum lifespan will be achieved primarily by reducing the mortality among people aged 70 and older.

In addition to making further advances in the "classical" drivers of survival improvement, scholars of various disciplines have proposed a number of groundbreaking ways of prolonging life at the higher ages, such as slowing human aging or eradicating major diseases through innovative forms of treatment and prevention. Citing findings from animal studies in which aging patterns have been modified through, for instance, dietary restrictions or the manipulation of genes, some scholars have are proposed that such modifications could be used to slow human aging and fundamentally change the mortality trajectory (Longo et al., 2015; Sierra et al., 2009). Moreover, Sierra et al. (2009) presented findings from human observational studies, clinical trials, and other research settings that could point to strategies for extending the human lifespan. However, while strategies for altering several "hallmarks of aging" have been suggested, an effective approach for halting senescence in humans has yet to be developed (López-Otín et al., 2013).

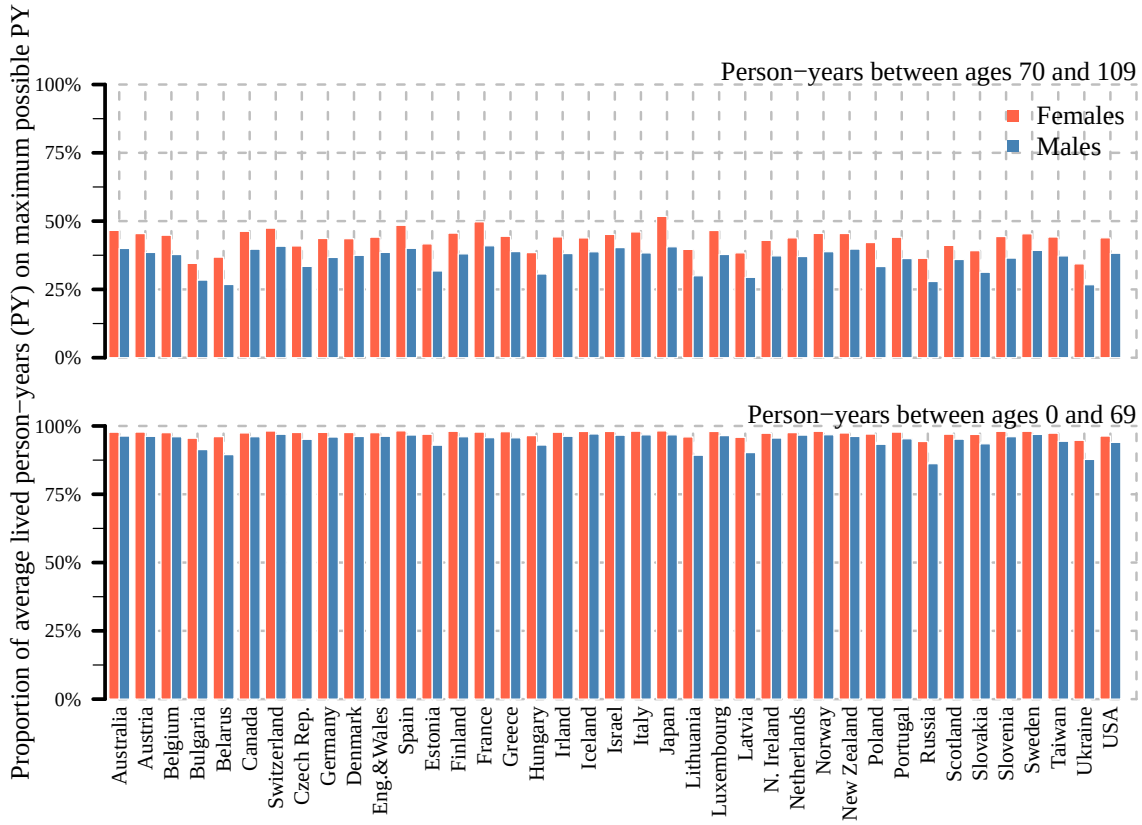

Figure 1.3.: Partial life expectancy between ages 0–69 and 70–109, males and females, various countries, 2010–2015 (estimates for the last year available by country). *The graph depicts partial life expectancy in the last year for which data are available. Accordingly, the years range from 2010 to 2015. Partial life expectancy is calculated by* $\frac{T_{x_2}-T_{x_1}}{l_{x_1}}$ *with* $x_2 > x_1$*, with* T_x *expressing the person-years lived above age* x *and* l_x *being the survivors at age* x*. It expresses the average number of person-years lived in specific age-interval (e.g.;* x_1 *to* x_2*). For this example, age intervals zero to 69 (left) and 70 to 109 are used. Accordingly, in the first interval, the maximum number of person-years is 70; and in the second interval, the maximum number of person-years is 40. The estimated partial life expectancy is divided by the respective maximum number of person-years to calculate the proportion of the living potential used in the two distinct age intervals. The estimates are based on period life tables, using age-specific death counts and exposures provided by the Human Mortality Database (2017a).*

Irrespective of the specific strategy proposed, opinions vary about the feasibility and the consequences of interventions aimed at slowing or stopping the aging process. From ancient times to the present, there have been those who favor having an extremely long and youthful life, and those who have warned of the dangers and the negative consequences of altering the human aging pattern and pursuing extreme longevity (Jeune, 2002). Regardless of whether any of these interventions succeed, Goldstein and Cassidy (2012) have shown that there are several ways of reducing mortality that would lead to considerable gains in life expectancy, and thus to the utilization of the potential for survival at the highest ages. Interestingly, the slowing of the aging process in humans is the intervention that appears to offer the greatest promise (Goldstein and Cassidy, 2012).

1.4. Aim of the thesis

The three main findings discussed above – namely, that mortality is continuously improving, that the age pattern of mortality improvement is changing, and that there are further opportunities for increasing longevity – are the major cornerstones of this thesis. The individual articles – the core of this thesis – stand on the shoulders of these well-established demographic facts. The persistence and the dynamics of mortality improvement, as well as the potential for future improvements, lead us to question whether existing demographic approaches and measures are sufficient for understanding these developments going forward. To organize the specific issues and aims of the articles, four thematic clusters are derived, which are related in different ways to the three key findings (see Figure 1.4). The clusters differ in terms of their thematic perspectives and their scientific tasks/approaches. In addition to providing some thematic orientation, the creation of these clusters allow us to map the distinct steps of the scientific process: namely, the *assessment* of a specific phenomenon, the *exploration* of the phenomenon and the formulation of future/alternative patterns (*outlook*).

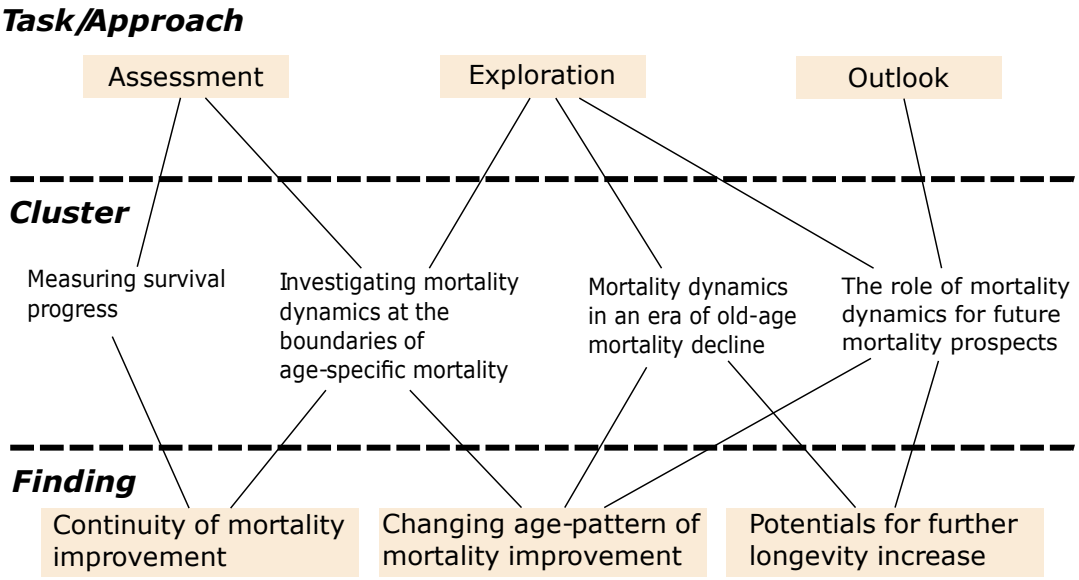

Figure 1.4.: Thematic outline and structure of the doctoral thesis. *The graph depicts the key findings, the thematic clusters, and the different approaches. The black solid lines express the relation of the different elements with each other. The figure is an own illustration.*

The sole focus of the first cluster, *measuring survival progress*, is the continuity of mortality improvement. Although declining mortality can be observed in almost all parts of the world, development levels are not equally distributed. Some populations have not yet completed the health transition, whereas others are already looking for ways to modify the human aging pattern in order to further extend the lifespan (Riley, 2005; Sierra et al., 2009). Therefore, monitoring survival progress is of great societal and political importance. Moreover, analysis of variations across populations and of deviant cases can provide valuable opportunities for studying the dynamics of specific survival determinants. Given the continuity of mortality improvement, this approach might be particularly useful for identifying the determinants of mortality dynamics and their respective changes. Hence, this cluster aims to provide methodological alternatives for *assessing* survival progress in order to simplify the respective evaluations and to stimulate a critical examination of reference trends.

The second cluster, *investigating mortality dynamics at the boundaries of age-specific mor-*

tality, refers to mortality development at the lower and the upper boundary of the age-specific mortality trajectory. Continuous improvements have pushed mortality rates to lower and lower levels. Moreover, in response to the changes in the age patterns of mortality improvement in developed and developing countries, efforts to prolong lifer are increasingly concentrated on generating further declines in mortality rates at advanced ages. Obviously, this is because the opportunities for reducing mortality are greatest at those ages. However, whether these efforts are having any effect on the lower boundary of age-specific mortality is unknown. Generally, relatively little is known about the evolution of the lower boundary of age-specific mortality. While more is known about the development of the maximum level of age-specific mortality, only a few analyses have examined the specific characteristics of this development, such as the level of maximum mortality. Additionally, the boundaries of age-specific mortality can be regarded as extreme cases. Hence, examining the dynamics and the changes in mortality patterns can help us better understand the plasticity of age-specific mortality, the potential frontiers of improvement, and the opportunities for overcoming previous limits in order to make further progress at the edges of the mortality trajectory. In addition to being useful for the assessment and the exploration of empirical trends, investigating mortality dynamics at the boundaries can have important implications for developing or refining methods such as mortality forecasting approaches. The main aim of this cluster is to *assess* trends and to *explore* the consequences and implications of the developments at the boundaries of age-specific mortality, with a special emphasis on the lower boundary.

The third cluster, *mortality dynamics in an era of old-age mortality decline*, deals with the methodological challenges that arise as a result of the sustained decline in old-age mortality. The shift in mortality improvements from younger to older ages has altered mortality dynamics. Moreover, old-age mortality improvements are likely to generate the greatest additional gains in life expectancy and longevity in the future. However, these emerging patterns call into question existing analysis frameworks and specific measurement techniques. Hence, this cluster addresses different measurement problems in this area, and aims to provide new approaches for *exploring* the emerging patterns.

The fourth cluster, *the role of mortality dynamics for future mortality prospects*, refers to the link between mortality forecasting approaches and mortality dynamics. Future life expectancy increases will be solely based on the exploitation of the potential for prolonging life at the highest ages. Moreover, as the age patterns of improvement have changed, mortality dynamics have already been altered at least once. Hence, further improvements might lead to additional changes in these dynamics. However, regardless of whether this happens or current trends continue, the methods used to measure these dynamics should be able to capture and anticipate different kinds of mortality dynamics at the highest ages. It is particularly important that mortality forecasting approaches are able to perform these functions because they are used to estimate future mortality trends. Hence, this cluster aims to *explore* the capacity of forecasting approaches to capture different kinds of mortality dynamics, and to provide an overview (*outlook*) of the methodological challenges involved in developing forecasting approaches.

The combined aim of the four clusters rests on the extension of opportunities to capture, analyze, and evaluate recent and future mortality dynamics; and to assess the likelihood that potential scenarios will develop in the future. Taken together, the clusters are expected to provide coherent outcomes that should result in advances in the assessment and exploration of mortality change in an era of continuous mortality decline, as well as in the projection of future scenarios.

1.5. Structure of the summary

Chapter one of this summary separately introduces the three demographic key findings, which provide the basis for the research presented. These findings are related to the continuity of mortality improvement, the changing age pattern of mortality improvement, and the prospects for further longevity increases at the highest ages. Based on these cornerstones, the aim of this

thesis is presented in chapter one.

The articles of this thesis are classified into four different thematic clusters. The outcomes of each cluster, as well as of the respective papers in the cluster, are presented in individual chapters. The results of the first cluster, measuring survival progress, are described in chapter two. The outcomes of the second cluster, investigating mortality dynamics at the boundaries of age-specific mortality, are presented in chapter three. The results of the third cluster, mortality dynamics in an era of old-age mortality decline, are outlined in chapter four. The outcomes of the fourth and final cluster, the role of mortality dynamics in future mortality developments, are presented in chapter five.

Each of the cluster-specific chapters follows the same outline. The chapter opens by introducing the cluster objectives. Due to the thematic range, each of the articles in cluster three (chapter four) also has an individual objectives section. The outcomes of the individual papers are then presented separately. The structure of these sections depends on the content of the respective article. Each subsection includes both a presentation of an outcome as well as a discussion and conclusion of the respective finding. The article-specific summaries focus on the most important outcomes, and are thus shortened and adjusted versions of the original text. Moreover, although the aim of these summaries is to provide a comprehensive overview, they do not offer the level of detail of the original article. To ensure that the focus remains on the respective outcomes, the technical details are reduced to a minimum. However, for the interested reader, each article summary contains a materials and data section that provides details on the data and methods used. After the outcomes of the individual articles are discussed, each cluster-specific chapter closes with a summary of the outcomes of the whole cluster.

Chapter six contains the overall conclusions of the thesis. The sections in this chapter are structured to cover the outcomes for the three specific tasks – namely, assessment, exploration, and outlook – that underlie the different thematic clusters. The last section of chapter six presents the conclusions for all of the clusters combined.

Chapter seven contains the original versions of the five main articles, and, if available, the respective supplemental information.

The appendix contains an overview of the authors' contributions to the preparation of the manuscripts, the original versions of the two appended articles, and the affidavit.

2. Cluster I – Measuring survival progress

2.1. Cluster objectives

Sklair (1970, p. XIV) defines progress as: "*...the end point, temporary or permanent, of any social action that leads from a less to a more satisfactory solution of the problems of man in society.*". The prolongation of the lifespan could be seen as the most important outcome of human actions intended to create a more satisfactory state. A series of advancements ranging from improvements in sanitation to nutrition to medical care have been instrumental to this achievement. These continuous and intrinsically motivated efforts to improve living conditions are responsible not only for the steady progress in survival, but for the shift in the age pattern of mortality improvement. For instance, the "cardiovascular revolution" (Frenk et al., 1991), which is an outcome of major advances primarily in medicine, might be seen as one of the more recent breakthroughs in improving quality of life, and as an example of how such advancements translate into changes in mortality.

In a comparative perspective, survival progress is also an important indicator for tracking the development of populations. Life expectancy is often used as a central indicator in this context. Among other advantages, the main benefit of using life expectancy is that it correlates with various economic, political and social indicators (Preston, 1975; Bergh and Nilsson, 2010). For instance, the level of life expectancy is one of three dimensions of the Human Development Index (United Nations, 2016); and although mortality has declined almost ev-

erywhere around the globe (Feigin et al., 2016), the level of progress achieved differs greatly across regions and across social strata (Lim et al., 2016; Marmot, 2005). Therefore, survival progress as defined in, for example, the Sustainable Development Goals remains instrumental to the United Nations' agenda of advancing the development of populations (Maurice, 2015).

Theory building and modeling also benefit from the assessment of progress (Keyfitz, 1975; Burch, 2018). For instance, the three periods of Omran's (1971) epidemiological transition theory are distinguished by the respective pace of survival progress. Because of variations in levels of achieved progress and speeds of progression, Vallin and Meslé (2004) have even proposed rethinking Omran's framework and replacing it with a more broadly applicable health transition theory. The mortality index in the canonical forecasting approach of Lee and Carter (1992) might be seen as a technical example whereby survival progress – in this case, the pace of survival progress across ages – is used for modeling and developing methods. In addition to their relevance in these areas, progress differentials and cases of exceptionally strong or weak improvements can be used to understand the effects and the dynamics of specific mortality determinants. For instance, cases in which life expectancy trends deviate from the general trajectory by, for example, stagnating or declining are often used to investigate the effects on overall progress of specific determinants, such as lifestyle factors or health care (among others Drefahl et al., 2014; Nusselder and Mackenbach, 2000; Tarkiainen et al., 2011).

From a technical perspective, there are two different ways to measure survival progress. The most common way is to look at the extent to which a mortality summary measure such as life expectancy differs between a comparison and a reference population at a specific point in time (gap). In some approaches, this gap is translated into an index to make it comparable across countries; such as in the Human Development Index or in the Global Burden of Diseases studies (Global Burden of Disease Group, 2017; United Nations, 2016). An alternative to measuring this gap is to compare the pace of improvement over time. For example, annual increases in life expectancy may be compared between a comparison and a reference population. However, both the gap and the slope include only one specific piece of information

about the progression of survival improvement. The gap could be understood as representing a static perspective, whereas the slope could be seen as reflecting a dynamic perspective. As both perspectives are needed to get a full picture of the advancement of survival progress, it would be beneficial to have an alternative measure that combines the gap and the slope in a single quantity.

The objective of this cluster is to (re)-introduce an alternative concept for the assessment of survival progress. The measure combines both the gap and the slope into a single value and translates the delay in development into calendar years; the most common unit used to assess progress. Moreover, the advantages and the drawbacks of different kinds of reference trends are discussed.

Paper in this cluster:
Ebeling, Peters, Rau (2018). The concept of equivalent time as a simple indicator for the assessment of survival progress. Under review in *Population Health Metrics*.

2.2. The concept of equivalent time

To introduce the concept of equivalent time, survival progress is linked operationally to the level of life expectancy at birth over time. Thus, countries with higher levels of life expectancy experience greater advances in survival progress than countries with lower levels of life expectancy. Likewise, increases and decreases in life expectancy over time reflect advances and declines in survival progress, respectively. While the former development is expressed by the difference in the life expectancy levels of two countries at the same point in time (gap), the latter development is measured by computing the absolute change over a given period of time within a country or a group of countries (slope; see also Figure 2.1). Hence, the gap might be understood as a static aspect of development, and the slope as a dynamic aspect of development.

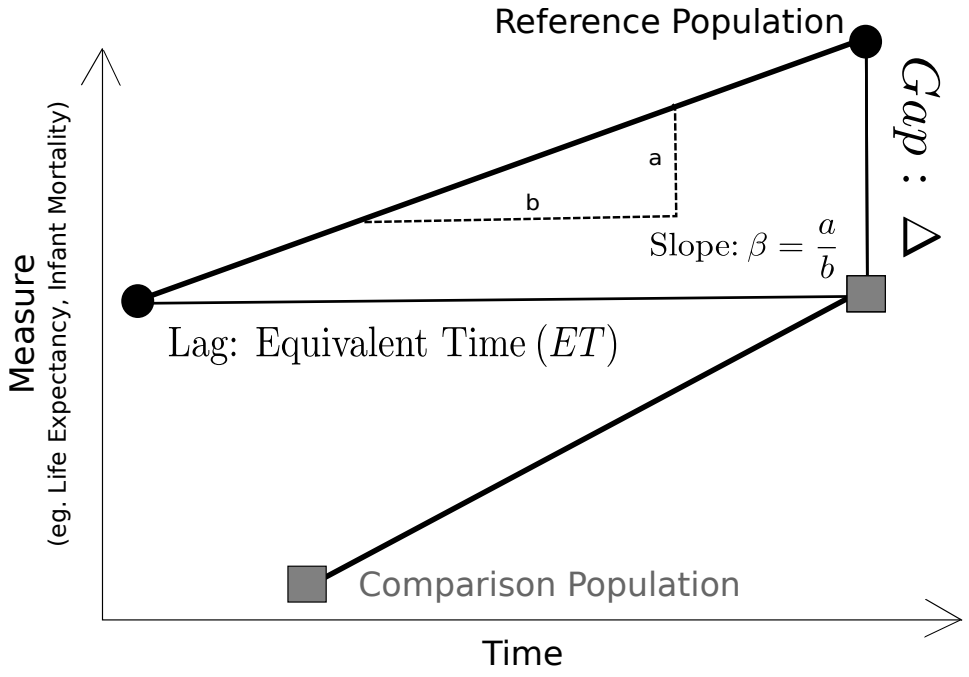

Figure 2.1.: Schematic representation of measuring survival progress.

There is a third, previously neglected dimension for tracking the development of survival progress that expresses how far back in time we need to go to find the equivalent level of life expectancy in a country relative to that of a reference group. This indicator is defined as the length of time the advancement of a country's life expectancy lags behind that of a reference population. We label this quantity *"equivalent time"* (ET). Figure 2.1 schematically illustrates ET and its relationship to the gap and the slope. In cases in which the reference trend is linearly increasing, ET could be simply computed by dividing the gap by the slope. Thus, ET combines the static and the dynamic aspects of development in a single quantity, expressed as the delay in development.

To identify a unique ET, the reference trend must fulfill certain requirements. To exclude the possibility that there is more than one equivalent value, the reference time series must change monotonically over time. This could, however, be viewed as a minor issue, because we see fairly steady improvements in most measures of survival progress, such as life expectancy and mortality measures like age-standardized death rates. Moreover, to provide a

straightforward benchmark for progress, the cases chosen as references usually have a steady pace of development. For the slope, it is not necessary to assume a constant change over time. It is also possible to directly compare empirical values without calculating a general slope.

The idea of using lags to assess survival progress was already suggested by Stolnitz more than 60 years ago (Stolnitz, 1955). He applied this approach in studying the delays in mortality decline in non-western countries relative to those in western countries. Surprisingly, we are not aware of any other study that has applied this approach. More recently, Goldstein and Wachter used a comparable concept to study the relationship between cohort and period life expectancy within countries (Goldstein and Wachter, 2006). They defined the lag as "how far back in time from the current period we have to go to find a cohort with equivalent life expectancy" (Goldstein and Wachter, 2006, pp. 259). The general idea of time lags has also been used to evaluate the decline in age-specific mortality by expressing the improvement in ages with equivalent mortality levels, which are usually called "equivalent ages" (Burger et al., 2012; Rau and Vaupel, 2014). While these earlier studies focused on detailed aspects of mortality and survival, our definition of ET is more general, and refers to cross-country comparisons of overall survival progress.

2.3. Types of reference trends

To demonstrate the added value of ET, we compare the trends in life expectancy among US males to those of three reference groups based on the gap, the slope, and the ET between 1960 and 2015. The goal in selecting the reference groups was to ensure that we were covering the spectrum of reference types typically used in comparative studies. We label the three main types intra-group, inter-group, and supra-group. In the *intra-group comparison*, a counterfactual of the trends in the country of interest is used as the reference: namely, cancer-free life expectancy of US males. In the *inter-group comparison*, a second external group is used as the reference: namely, Japan, the world leader in LE. Finally, in the *supra-group compari-*

son, a more general trend is used as the reference: namely, the average LE of the G7 countries.

Table 2.1.: Classification of different kinds of references for the evaluation of survival progress. *The listed examples are using the reference perspective in at least some part of their empirical analysis.*

Type	Example	Specific Features	Exemplary Study
Intra-group	$e_{0,\text{Country A}} - e^{-i}_{0,\text{Country A w/o health burden i}}$	peculiarity or commonality?	Aburto et al. (Aburto et al., 2016)
Inter-group	$e_{0,\text{Country A}} - e_{0,\text{Country B}}$	prime example or exception?	Meslé and Vallin (Meslé and Vallin, 2006)
Supra-group	$e_{0,\text{Country A}} - e_{0,\text{Cross-country average}}$	common trend?	Bongaarts (Bongaarts, 2006)
e_0 –Life Expectancy at Birth			

Obviously, which references are chosen depends on the research objective. Even if a specific reference fits the research purpose, it is important to take into account the specific features of the respective reference type. Table 2.1 provides an overview of specific reference types and their characteristics. The results stemming from an intra-group comparison are not sufficient to determine whether the measured lags are a peculiarity of the respective population, or are occurring across several populations. Moreover, the outcomes of an inter-group comparison do not tell us whether the reference population is a prime example of the specific gains being evaluated, or is an outlier. Finally, the results of the supra-comparison do not indicate whether the more general reference trend is reflective of a common trend across populations.

2.4. Materials and data

The estimates of life expectancy at birth for US males — the comparison group — are based on death counts and exposures from the Human Mortality Database (Human Mortality Database, 2017b). In the intra-group comparison, cancer-eliminated life expectancy is used as the reference (see, for instance, (Preston et al., 2001) for more details on cause-elimination life tables). Cause-specific death counts were obtained from the National Center for Health Statistics (NCHS) (2016). The total time period analyzed encompasses the years 1960 to 2015. Cancer

deaths were reconstructed using the International Classification of Diseases (ICD) codes 140–239 (ICD 7–9) and C00–D48 (ICD 10). The calculations of Japanese life expectancy in the inter-group comparison and of the life expectancies for the G7 countries in the supra-group comparison were also based on death count and exposure data from the Human Mortality Database (2017b). To ensure that the supra-group also covers the years 1960-2015 for countries that were missed in the most recent life expectancy estimates, values were obtained from the World Bank (2017). The equivalent time in all three examples was calculated using linear interpolation between the time series of the comparison population and the reference population. The whole analysis was conducted in the language R (R Core Team, 2017).

2.5. Illustrative example: assessing the survival progress of US males

Figure 2.2 shows the results for all three comparisons. The rows depict the results for the intra-, inter-, and supra-group comparisons, respectively. The rows display the results for the intra-, inter-, and supra-group comparisons; while the columns show the life expectancy, the gaps, the slopes, and the equivalent times. The slopes are calculated as annual increases over five year periods, assuming a linear change between the respective time points.

The life expectancy level of US males increased continuously between 1960 and 2015 (panel a). Although an increase can also be observed for all three reference groups, the level of life expectancy differs across the groups, and compared to the US pattern (panels a–c). The life expectancy gap between US males and the inter-group reference category remained fairly stable at a level of about three years, while the life expectancy gap between US males and the intra-group reference category declined from one year higher in 1960 to four years lower in 2015 (panels d–e). The life expectancy gap between US males and the supra-group reference category increased from very small in 1960 to about three years in 2015 (panel f).

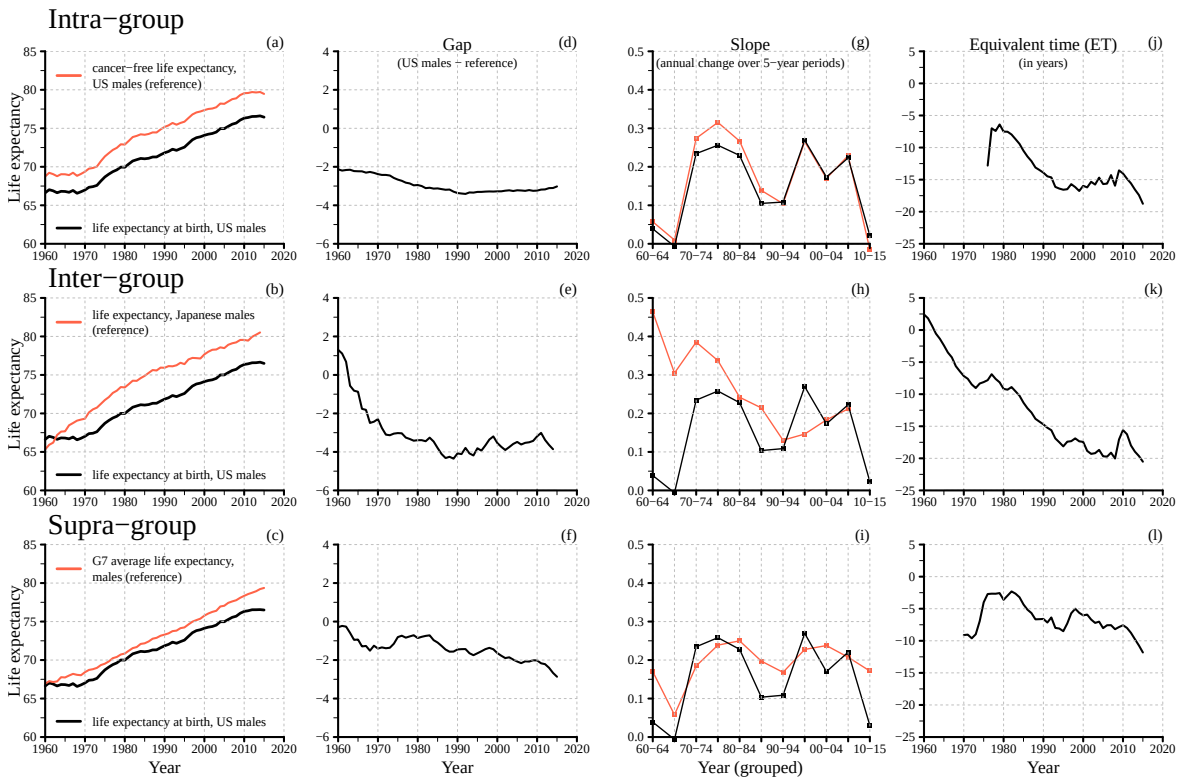

Figure 2.2.: Life expectancy, gap, slope (annual change over five year periods), and equivalent time of US life expectancy compared to three references, males, 1960-2015. *All of the estimates are based on period life expectancy. The slopes indicate the annual change between the two years, comprising five-year intervals. The last group 2010-15 comprises six years. All calculations of the slope assume a linear change between the two time points. Equivalent time is calculated using linear interpolation. The various example rest on data from the National Center for Health Statistics (NCHS) (2016), the Human Mortality Database (2017b) and the World Bank (2017).*

An assessment based on the pace of improvement alone provides limited insights. Both cancer-free and average G7 life expectancy had almost the same slopes as that of US male life expectancy across the different year groups. By contrast, Japanese life expectancy increased more rapidly until 1985-89, but changed at almost the same pace thereafter (panels g–i). Generally, however, the slopes varied greatly over time, which makes it difficult to derive solid

conclusions about the advancement of survival progress.

Compared to the gap and the slope, the ET shows larger differentials in the development levels of the reference groups and larger differences between the groups in the advancement of survival progress over time. In the intra-group comparison, US male life expectancy in 1975 is the first value, which can be located on the reference trend line. In this year, the survival progress of US males lagged around 13 calendar years behind the level that hypothetically could have been achieved if cancer had been eradicated. This lag grew consistently over time to reach a magnitude of almost 19 calendar years in 2015 (panel j). In the inter-group comparison, we even see that the survival progress of US males was two calendar years ahead of that of Japanese males in 1960. But by 2015, this advantage had turned into a lag of almost 21 calendar years (panel k). In the supra-group comparison, US male life expectancy in 1970 is the first value, which can be located on the G7 average trend line. In this year, the survival progress of US males lagged around 10 years behind the average level of progress of the G7 countries. After a period of improvement, the lag among US males increased consistently to reach a level of 12 calendar years by 2015 (panel l).

2.6. Cluster conclusion

In this cluster, a measure for assessing survival progress has been introduced. It is labeled equivalent time (ET). The measure complements the current practice of comparing development at a single point in time and of comparing changes in development over time. It offers a simple and intuitive way to express in calendar years the delay in the development of a population in comparison to that of a reference population. ET arguably has the potential to become a standard approach in the toolbox that is used for the assessment of survival progress in both large-scale cross-country comparisons and case studies, and for the evaluation of single countries. To illustrate ET, period life expectancy has been used, but any other continuous variable that is monotonically increasing or decreasing over time could be used as well.

The simplicity of ET is, of course, also a shortcoming. Since it is built on a single variable, critics might see ET as an oversimplified measure that does not capture the multiple dimensions of development. This criticism is certainly justified. Thus, in cases in which reliable data on other dimensions are available, the use of more complex measures might indeed be more appropriate. However, for many countries, and particularly for longer historical periods, such data are often not available.

The illustrative example depicts a potential application of ET that is complementary to the classical approaches. The results indicate that the survival progress of US males has been increasingly falling behind that of other groups, as the large increases in ET found among US males over the study period demonstrate. For instance, the application of ET showed that relative to their Japanese peers, US males experienced a delay in LE development of two decades. This is a remarkable insight given that US life expectancy increased virtually throughout the whole study period at a pace that was almost comparable to that of the reference categories. At the same time, the gap between US life expectancy and that of the reference groups remained fairly stable, at least compared to that of the intra and supra-group reference categories. The results are even more intriguing given that the US has the highest health care expenditures in the world (Wang et al., 2013). Moreover, the results show that ET is a more sensitive measure of advances in survival than the comparison of gaps or slopes.

In addition to introducing another tool for measuring progress and development, we were able to show that the assessment of the performance of a specific country is not straightforward. Rather, we illustrated that such assessments are sensitive to the selected indicator (e.g., life expectancy), perspective (gap, slope, lag), and reference (e.g., G7 countries). It is therefore important to use care in interpreting results that are based on a single measure, perspective, or reference. If possible, multiple factors should be taken into account for each of these dimensions.

3. Cluster II – Investigating mortality dynamics at the boundaries of age-specific mortality

3.1. Cluster objectives

Mortality has been declining for more than 160 years (Oeppen and Vaupel, 2002; Riley, 2001). Mortality rates at almost all ages have regularly reached unprecedented minima over this period. These developments are attributable to continuous advances in nearly every area of life, which also altered some fundamental aspects of human mortality, such as the cause of the spectrum. While each of these successive improvements was unprecedented, the mortality trajectory has retained certain key features (see Figure 3.1). For example, levels of infant mortality have been consistently similar to mortality levels between ages 60 to 80. Moreover, adult mortality has shown a log-linear increase over age, and mortality levels between infancy and early adulthood have followed a U-shaped pattern.

Moreover, as these advances were occurring, major improvements in survival shifted from younger to older ages (Christensen et al., 2009). The changing age pattern of mortality improvement might also be interpreted as a sign that all of the major opportunities for reducing mortality at younger ages have been exploited. Today, the contributions of the youngest ages to the increase in the average age at death are small to negligible. Nevertheless, in almost all countries of the world, mortality at younger ages has been decreasing continuously (Armour-

Marshall et al., 2012; Verguet et al., 2014). This trend has resulted not only in an increasingly pronounced U-shaped pattern of mortality between infancy and early adulthood (see Figure 3.1), but in a widening of the range of age-specific death rates.

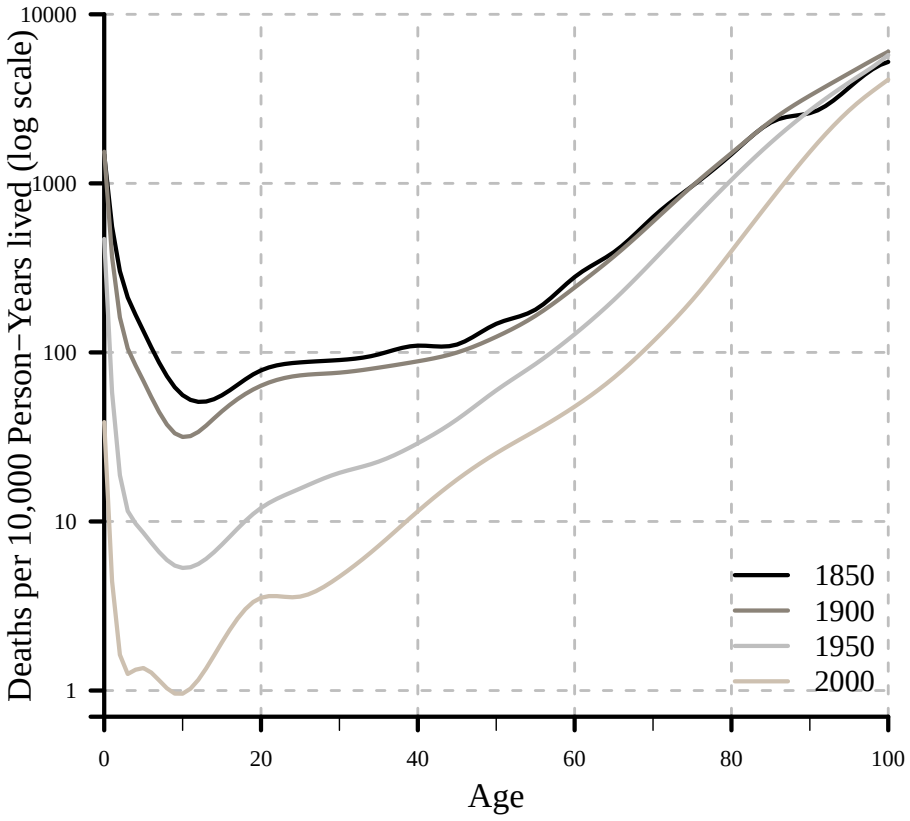

Figure 3.1.: Age-specific death rates, France, females, years 1850,1900, 1950, and 2000. *Death rates are smoothed. The rates are plotted on a logarithmic scale. The estimates are based on age-specific death counts and exposures provided by the Human Mortality Database (2017a).*

The lower boundary of age-specific mortality is located at the inflection point of the U-shaped mortality pattern between infancy and early adulthood. The upper boundary is reached at the late-life mortality plateau Gampe (2010). Both the upper and the lower boundary are

valuable indicators of the dynamics at the edges of human mortality. However, both boundaries have unique characteristics that reveal their fundamental differences.

The plasticity of the lower boundary compared to the stability of the upper boundary is perhaps the most important difference. Gampe (2010) estimated a mortality rate of around 7,000 deaths per 10,000 person-years lived at the upper boundary of age-specific mortality. But when we look at Figure 3.1, large declines in the lower boundary are immediately evident. Explanations for the stable upper boundary are diverse, and range from a more homogeneous population composition at those ages to physiological and genetic aging processes (Wachter, 1999; Pletcher and Curtsinger, 1998; Missov and Vaupel, 2015; Vaupel et al., 1998). Minimum mortality, by contrast, appears to (still) be alterable. Most importantly, improved living standards (e.g., better sanitation and nutrition) and medical breakthroughs, such as antibiotics and vaccinations, are the main reasons for the survival gains at childhood and juvenile ages (Cutler et al., 2006; Blum, 2009; Gore et al., 2011).

After the degree of alterability, the location is the second major difference between minimum and maximum mortality. Maximum mortality is located at the end of the lifespan, which seems obvious due to the log-linear increase in mortality with age. Minimum mortality is however reached within a tiny age range at the end of the first decade of life. It marks a specific point over the life course at which physical and social development levels are most favorable for avoiding death. The explanations for why mortality levels are lowest in this exact age range are mainly drawn from evolutionary mechanisms, such as high selection pressure prior to the beginning of the reproductive period; i.e. the proximity of these ages to the onset of sexual maturity (Burger et al., 2012; Chu et al., 2008; Levitis, 2011). However, for both males and females, scholars have observed over the past century temporal changes in related processes, such as faster body growth or the earlier onset of puberty (Frisch, 1978; Tanner, 1973; Goldstein, 2011; Schönbeck et al., 2012). The location of minimum mortality might be similarly affected by these shifts.

Male-female differences are the third key feature that could reveal further disparities between maximum and minimum mortality. Gampe (2010) did not find any relevant differences in maximum mortality between males and females, even though most of the data used in her analysis were for females. Unfortunately, until recently there have been relatively few studies that examined the level of the plateau and potential sex-specific differences. Among children and adolescents, mortality is higher for males, even though the absolute gap becomes smaller with decreasing levels of mortality (Gissler et al., 2009). Surprisingly, external causes of death as an indicator for sex-specific risk-adverse behavior explain only a minor part of the male-female differences (Gissler et al., 2009). For example, Gissler et al. (2009) measured a higher rate of non-external causes for boys than for girls. It therefore appears that additional factors must be responsible for these differences.

Examining these key features help us better understand about the frontiers of human mortality improvement because minimum and maximum mortality (the lower and the upper boundary) indicate specific points along the mortality trajectory at which the condition of individuals as well as the composition of the population are most extreme in terms of mortality severity. Hence, it could be hypothesized that potential frontiers and fundamental breakthroughs that might affect all ages in the long run are primarily detectable at the boundaries of age-specific mortality.

Understanding the development of the mortality boundaries is also important for mortality modeling. For instance, mortality forecasting approaches usually assume that death rates have no lower limit. Instead, the logarithm of death rates is used, which allows rates to decline infinitely, while staying between zero and one (see, for instance, Hyndman and Ullah, 2007; Lee and Carter, 1992). Accordingly, examining the evolution of the lower boundary of age-specific mortality can provide further insights that would help us better understand the implications of recognizing lower boundaries in mortality forecasting approaches. Moreover, attempts were made in previous parametric mortality models, such as the Siler model (Siler, 1983) or the Heligman-Pollard model (Heligman and Pollard, 1980), to include mortality at

all ages. For instance, the Siler models incorporate the lower boundary by adding a constant to the two terms, which captures the exponentially decreasing and increasing parts of the force of mortality. In the Heligman and Pollard (1980) model, no specific parameter is added to model the lower boundary, but the parametrization generates such a pattern. Neither parametric models take the pattern of the upper boundary of mortality into account. Accordingly, the mortality pattern of both models would generate further increases at the highest ages, although the increase in death rates would decelerate (Horiuchi and Wilmoth, 1998) prior to the leveling-off at the late-life mortality plateau (note that the Heligman and Pollard model shows some deceleration). However, evidence for a leveling-off and clear empirical proof of the mortality plateau has been provided by studies published long after the publication of the works of Siler (1983) and Heligman and Pollard (1980), following the development of other parametric models (see among others Thatcher et al., 1998; Thatcher, 1999; Gampe, 2010). The examples of mortality forecasting and parametric mortality models demonstrate the added value of exploring the boundaries of age-specific mortality in order to refine existing approaches or even develop new models.

Based on these considerations, the objectives of this cluster are to investigate the evolution of the lower and the upper boundary of mortality, with a special emphasis on the lower boundary. Accordingly, three essential questions should be asked and answered when examining minimum mortality. These questions are:

- How pronounced are the sex-differences at the lower boundary of age-specific mortality?
- At which ages is minimum mortality reached, and how have these ages changed over time?
- How has the level of minimum mortality evolved, and is minimum mortality still decreasing?

Given the unprecedented mortality improvements of the past, it is not far-fetched to hypothesize that minimum mortality will be the first death rate to finally hit a lower (natural) limit;

and that minimum mortality indicates the absolute frontier of human mortality improvement.

For the upper boundary, the question to be answered is simply:

- Where is the level of the late-life mortality?

Thus, the aim is to investigate whether it is possible to find further support for a maximum mortality level of 70 deaths per 100 person-years lived using an estimation procedure that represents an alternative to the procedure used by Gampe (2010).

Papers in this cluster:
Ebeling (2018). How has the lower boundary of human mortality evolved and has it already stopped decreasing? Forthcoming in *Demography*.

Appended paper: Rau, Ebeling, Peters, Bohk-Ewald, Missov (2017). Where is the level of the plateau? Published in *Living to 100 Monograph*.

3.2. Outcomes for the lower boundary of age-specific mortality

3.2.1. Material and data

Minimum mortality is estimated using cohort and age-specific data because only a cohort follow-up provides the life course perspective that is needed to clearly identify the lowest mortality rate over age. Moreover, many forces shape minimum mortality, such as selection in a cohort. The cohort data used come from the Human Mortality Database (2017c). All of the estimates are based on data for individuals aged one to 20. The earliest cohort considered was born in 1900. For some countries, the data start later. The most recent cohort considered also varies by country. Countries with data covering fewer than 20 cohorts are excluded from

the analysis.

Although data from the Human Mortality Database cover national populations, and can thus be considered complete enumerations, the data are still subject to stochastic variation (Udry et al., 1979; Kirkby and Currie, 2010; Klotz, 2016). In addition, it is usually assumed that the underlying mortality process is smooth (see also Kirkby and Currie, 2010). Minimum mortality could be considered to be especially vulnerable to population size and stochastic variation because of its low intensity. Hence, around the age of minimum mortality, mortality could be quite noisy. Minimum mortality contains another, rather technical and theoretical problem: namely, that mortality rates are bound at zero at the lower end. In low-mortality countries with a small population size, mortality at some ages below 20 is already at such low levels that the number of age intervals without any deaths is consistently increasing over time. To deal with zero death rates and stochastic variation, we complemented the analysis of observed trends with estimates based on a two-dimensional smoothing approach by Camarda (2012), and applied it over age and cohort (see the original paper for a detailed discussion of model selection and descriptions of the different models). The method is readily available via the package "MortalitySmooth" for the statistical programing language R (2017).

The occurrence of period shocks, such as the Spanish flu or a war, can lead to problematic estimates when two-dimensional smoothing approaches are applied, because in such situations the respective mortality rates are subject to more than just stochastic variation (Kirkby and Currie, 2010; Palloni, 1990). When the two dimensions are cohort and age, the situation becomes especially difficult because the period shocks are located on a backward 45-degree line in the age-cohort surface. Due to the data structure, the 1915–1919 and 1938–1947 periods, which cover the Spanish flu and World Wars I and II, are excluded from the analysis. The approach we used interpolates the emerging gaps, which are generated when the mortality of the respective ages is ignored.

To avoid potential distortions, the range of ages searched for the minimum mortality level

was restricted to ages five to 15. This is theoretically reasonable given the range of observed ages.

3.2.2. The level of minimum mortality

Figure 3.2 shows the minimum mortality levels for males and females in France (see the original paper for results for all of the other countries). The graph depicts the level of minimum mortality per 100,000 person-years lived. The levels are shown on a log scale, using the logarithm of two to emphasize level halving. Accordingly, the horizontal contour lines mark the consecutive halving of the mortality level. The solid lines are minimum mortality estimates based on the age-cohort smoothing, and the colored area around this line indicates the 95% confidence interval. The plus signs indicate observed minimum mortality. The squares mark the observed minimum mortality for cohorts who spent at least one year in the omitted periods (1915–1919, 1938–1947). Females are depicted in red and males in blue.

In Figure 3.2, we can see that minimum mortality declined from almost 250 deaths per 100,000 person-years lived to around 10 deaths per 100,000 person-years lived over the cohorts born between 1900 and 1993. Over the course of this decline, the trajectories for French males and females show four periods with distinct developments (see tags in Figure 3.2). Minimum mortality improves only slowly in the first period (I), which encompasses the cohorts born between 1900 and 1920. The second period (II) is characterized by rapid improvements. This period spans the cohorts born between 1920 and 1950. In the third period (III), minimum mortality improvements decelerated, and were close to stagnation at certain points. This trend lasts up to the cohorts born in the early 1960s. The fourth period (IV), which covers the most recent cohorts, again shows steady improvements. The trajectory of France is exemplary of the trajectories observed in the majority of countries analyzed. The only variation that could be found were small temporal differences and differences in the manifestation of the patterns in the four periods described. The minimum mortality levels of a few countries only, such as those of Russia or Belarus, deviate from the general trend. In these countries, the improve-

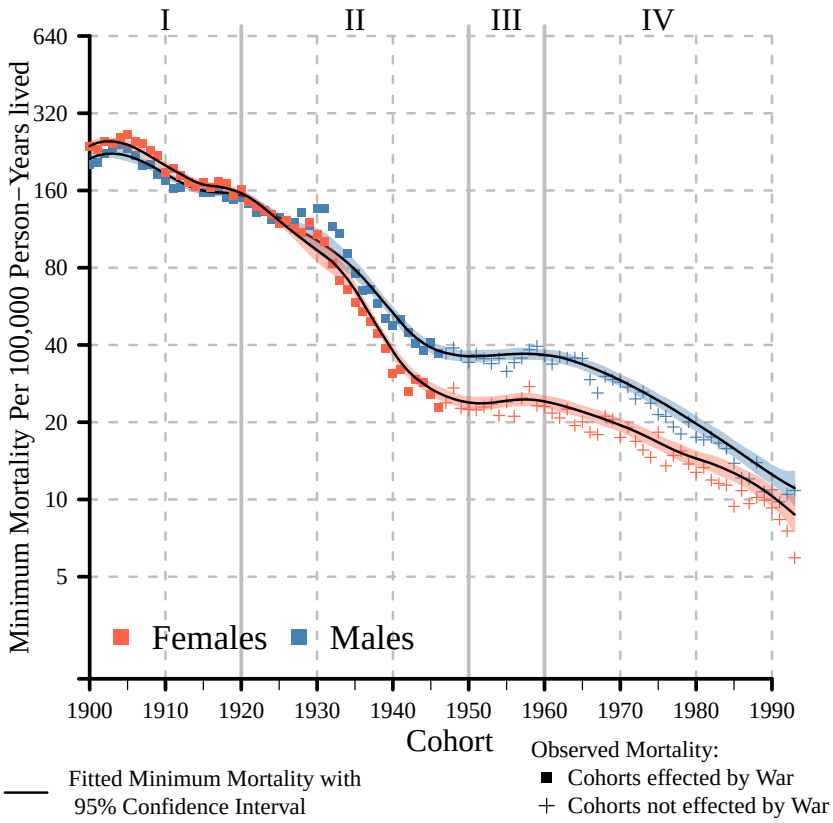

Figure 3.2.: Minimum mortality, France, females and males, birth cohorts 1900–1993. *The graph depicts the observed (squares and crosses) as well as the smoothed (solid line) minimum mortality levels. The observed rates marked with a square indicate the cohorts who spent at least one year in the omitted periods (World Wars I and II). The colored area around the smoothed minimum mortality estimates depict the 95% confidence interval, as calculated by the approach of Camarda (2012). Minimum mortality rates are illustrated using the logarithm with basis two. The solid gray grid lines and the Roman numerals mark the different periods of development. The age- and cohort-specific death counts and exposure data were obtained from the Human Mortality Database (2017c).*

ments in minimum mortality over the analyzed cohorts are marginal to non-existent.

Based on the broad picture across all of the countries analyzed, it could be concluded that

the level of minimum mortality decreased continuously over the observed time period, and that the pace of improvement among recent cohorts has been relatively steady. Therefore, the recent trends do not suggest an imminent end to this decline. Among the best practice countries, the current levels are about eight deaths per 100,000 person-years for females and about 10 deaths per 100,000 person-years for males (see the original paper for a comparison of the recent minimum mortality levels across all of the countries analyzed). However, the lower boundary of mortality has not been decreasing constantly. Especially among the post-war cohorts, improvements have temporarily slowed, with some countries even experiencing stagnating or slightly increasing levels. The reasons why minimum mortality has stagnated are not entirely clear. Among the potential explanations for this development are the introduction of mass immunization for several diseases in the 1960s (Riley, 2001); changes in fertility-related behavior (Billari and Kohler, 2004); and changes in child care (Vandell et al., 2010).

Given that this decline has been steady, it is intriguing that the lower boundary of mortality has still not reached a lowest limit after almost 170 years of continuous mortality improvements. The question of whether minimum mortality has a lower limit remains open and continues to present challenges. If such a limit does exist, mortality might follow a constant force of mortality, whereby simply good or bad luck are the essential mortality determinants. However, recent developments in childhood health call into question such an optimistic assumption. For instance, Brüne and Hochberg (2013) have found that since the late 1980s in particular, the prevalence of chronic diseases in childhood, such as obesity, diabetes, and autoimmune diseases has been increasing. They argued that changes in the environment favor this development; speculating that evolutionary and medical factors — such as thrifty genes, hygiene, fetal programming, or the extensive intake of cow's milk — might explain this trend. Although the rising prevalence of chronic diseases in childhood might not have an immediate effect on death, it might increase the vulnerability of children, and could thus have an indirect effect on the level of minimum mortality. However, based on these results, we cannot confirm that these developments have had any negative impact on minimum mortality levels among the most recent cohorts.

3.2.3. The location of minimum mortality

The results for the minimum mortality ages can be seen in Figure 3.3. The observed mortality rates as well as the smoothed mortality rates are given by age. Hence, the ages of minimum mortality are also measured in integers. Due to stochastic fluctuations and the impact of the world wars, minimum mortality ages based on the smoothed mortality estimates are considered as the basis. Furthermore, the trends across countries are very homogeneous, and show only small variations. Therefore, we pooled the ages of minimum mortality across countries and summarized the results for 10 consecutive cohort groups. Due to the varying lengths of the data available, the number of ages in each of these 10 groups differs.

The age of minimum mortality decreased over the cohorts studied. This trend is also visible if we look at the ages based on the observed mortality estimates. For the cohorts up to those born in 1920–1929, the modal value jumps between ages 11 and 12. The distribution of males and females for the 1930–1939 and 1940–1949 cohorts are right–skewed, which means that ages higher than the mode are observed more frequently than younger ages. This is, however, likely an effect of World War II. Although minimum mortality among the cohorts born in 1950–1959 or later has been relatively consistently located at age 11, the distribution is shifting toward younger ages. The shift is indicated quite well by the growing frequency of age 10 as the age of minimum mortality over the respective groups. Accordingly, for the last two cohort groups, the modal value is already located at age 10, and ages above and below the mode are almost evenly observed. However, the growing frequency of age nine over the last two cohort groups could suggest that the shift toward younger ages might be continuing.

The location of the lowest mortality level is closely associated with the onset of sexual maturity. Hence, the change in the location of minimum mortality might be generated by changes in related mechanisms. Evolutionary theories of aging argue that evolutionary fitness, defined as the intrinsic rate of natural increase, is most sensitive to mortality changes around the age of

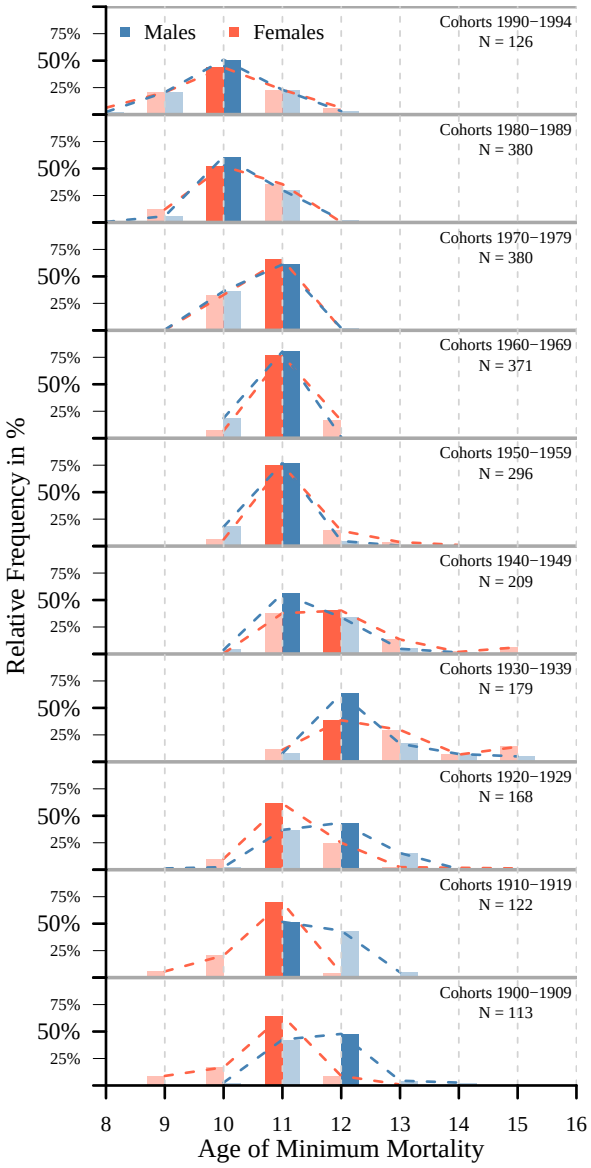

Figure 3.3.: Distribution of minimum mortality ages, all countries together, females and males, grouped birth cohorts 1900-1994. *The bars show the relative frequency of the ages in the respective cohort groups; pooled over all of the available countries. The age of minimum mortality is measured in integers. The number of countries varies over time in each cohort group, and is indicated by N. The highlighted bar represents the modal age in the respective year. The bars correspond to full ages. The age- and cohort-specific death counts and exposures data were obtained from the Human Mortality Database (2017c).*

sexual maturity (Hamilton, 1966). Accordingly, selection pressure on age-specific mortality should be highest around the onset of the reproductive period, when the respective mortality rates are pushed to their lowest possible levels. Similarly, the age of maturity itself should be under strong selection pressure as a key age that defines the onset of reproduction. Other authors have also argued that intergenerational transfers, such as parental care, shape selection pressure, and are thus an important determinant of the shape of human life history, and, in turn, mortality (Gurven et al., 2012). The investments of older generations in younger generations therefore lead to a concave shape of selection pressure, which may push mortality down even further at the onset of the reproductive period, when such investments start to pay off (Chu et al., 2008; Lee, 2003; Bogin, 1997). As a result of the close relationship between the location of minimum mortality and sexual maturity, it could be hypothesized that the earlier occurrence of minimum mortality might be related to temporal changes in different aspects of childhood development such as body growth, and thus to the earlier occurrence of puberty and sexual maturity that has been reported by a number of authors (Goldstein, 2011; Tanner, 1973; Frisch, 1978; Schönbeck et al., 2012). However, the similar locations of minimum mortality of males and females could be seen as a sign that this is not the case, because different studies provide evidence for a slightly earlier onset of puberty of females, which also holds for the transition through the different developmental stages of sexual maturity (Susman et al., 2010; Lee, 1980). In addition to these factors, Levitis and Martínez (2013) have offered further hypotheses for why the juvenile mortality pattern is U-shaped, and for why we therefore see an inflection point between the decreasing and the increasing parts of mortality over age. However, the plasticity of minimum mortality challenges all of these concepts. Given the enormous gains that have been made over a short period of time, it is possible to speculate about whether human progress has decoupled minimum mortality from evolutionary mechanisms — or has, at least, weakened the relationship between them.

3.2.4. Sex–specific differences of minimum mortality

Figure 3.4 shows the absolute male–female differences in the levels of minimum mortality for all of the countries analyzed. The trajectories of Japan, Russia, France and Norway are highlighted. A negative difference expresses higher mortality for females and vice versa.

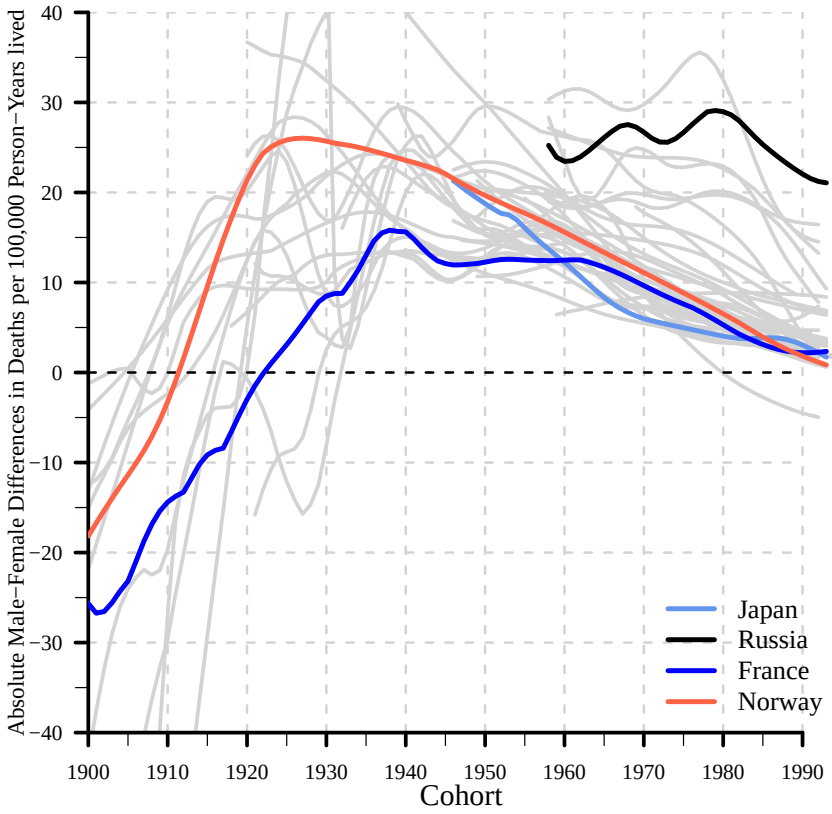

Figure 3.4.: Absolute male–female minimum mortality differences, Japan, Russia, France and Norway, birth cohorts 1900-1994. *The gray lines depict all of the other countries included in the analysis. The sex differences are calculated based on the smoothed minimum mortality estimates. The age- and cohort-specific death counts and exposures data were obtained from the Human Mortality Database (2017c).*

The development of absolute *sex-specific differences* at the lower boundary of mortality shows two distinct patterns: female mortality is higher with a consistently growing male dis-

advantage, followed by a trend toward convergence. Russia is an exception to this overall pattern. Among the most recent Russian cohorts analyzed, men have a minimum level that is still around 20 deaths per 100,000 person-years higher than that of females; whereas in, for example, Japan, France, and Norway, the absolute male-female differences are almost negligible.

The female excess mortality found in the oldest cohort analyzed is an intriguing pattern. Tuberculosis could be one explanation for this trend. Several authors have documented higher tuberculosis death rates among females in the respective age range (10-14) for similar cohorts and for the calendar years in which the respective cohorts reached their minimum mortality level (Frost, 1995; Springett, 1952a,b). Other potential explanations for this finding such as discrimination related to sex or birth order (Modin, 2002) are conceivable, but remain vague speculations.

The female disadvantage turned relatively rapidly into a male disadvantage, which reached the maximum level at some point among the interwar cohorts. Since then, a continuous trend toward convergence can be observed. The male disadvantage is the result of slower improvements in the minimum mortality levels of males than of females. These pace differentials are striking because the usual determinants of sex-specific differences, such as lifestyle and behavioral factors, should be less relevant at these ages. We can speculate that excess mortality caused by environmental conditions is decreasing more quickly among females than among males. It is also possible that after this type of excess mortality is no longer relevant, the gender gap is still primarily driven by biological factors. Currently, it appears that communicable diseases can be excluded as a potential driver, and that external causes of death explain only a small part of the gender gap (Gissler et al., 2009). Accordingly, non-behavioral causes and non-communicable diseases could be emerging as the potential drivers. Studies that used cancer — a leading cause of death in childhood in the developed countries — to illustrate this development found that boys are more likely than girls to develop a childhood cancer (Kaatsch, 2010; Dorak and Karpuzoglu, 2012).

3.3. Outcomes for the upper boundary of age-specific mortality

3.3.1. Material and data

The level of the late-life mortality plateau is the upper boundary of age-specific mortality. Accordingly, after the log-linear increase in mortality at adult ages, the force of mortality tends to decelerate between around the ages 80 to 85. This phenomenon is commonly referred to as "mortality deceleration" (Horiuchi and Wilmoth, 1998). This deceleration ultimately results in the late-life mortality plateau, at which mortality remains constant (see, for instance, Thatcher et al. (1998); Missov and Vaupel (2015)). The explanation for this pattern is most often drawn from the selective mortality hypothesis, which rests on a simple idea. Populations are heterogeneous, which means that some individuals in the population are frailer than others. On average, these frailer individuals tend to die earlier than their more robust peers. Accordingly, the force of mortality observed on the population level ($\overline{\mu}_x$) could be interpreted as average across these different subpopulations. As these frailer individuals die off, the force of mortality tends to level off. The general relationship between the population-level hazard and the risks faced by subpopulations is described in more detail by Vaupel and Yashin (1985) and Vaupel (2010).

The formalization of this idea originates from the canonical work of Vaupel et al. (1979), in which the authors introduced frailty models. Assuming that frailty is fixed over the lifetime for each individual, and that frailty affects the baseline mortality proportionally, the force of mortality of an individual with frailty z – the simplest frailty model – is expressed by

$$\mu_{x,z} = z\mu_{0,x}, \tag{3.1}$$

where $\mu_{0,x}$ depicts the baseline force of mortality at age x. Accordingly, the force of mortality at the population level can be expressed by

$$\overline{\mu}_x = \int_0^\infty z\, \mu_x\, f_{x,z}\, dz, \tag{3.2}$$

where $f_{x,z}$ is the probability density function of frailty among the survivors at age x. Hence, the force of mortality of the population at age x is the weighted average across individual force of mortality, where the weights stem from the probability density function of frailty among the survivors at age x. Vaupel et al. (1979) already assumed that frailty is gamma-distributed with a mean of one, which was later discussed in more detail by, for instance, Vaupel and Missov (2014). By combining this assumption with the frequently investigated idea that the force of mortality at the individual level follows the Gompertz mortality model (Steinsaltz and Wachter, 2006; Finkelstein and Esaulova, 2006), the force of mortality at the population level can be expressed by the gamma-Gompertz model, which is described by Vaupel and Missov (2014) as

$$\overline{\mu}_x = \frac{be^{b(x-M)}}{1 + \gamma e^{-bM}(e^{bx} - 1)}, \tag{3.3}$$

where b is the slope of the force of mortality over age, M is the modal age at death, and γ expresses the variance of the gamma distribution. The mortality plateau for this model can then be expressed by

$$\lim_{x \to \infty} \overline{\mu}_x = \frac{b}{\gamma}. \tag{3.4}$$

Our investigation of the mortality plateau rests on this model. Based on the minimum AIC (Akaike, 1974), the gamma-Gompertz came out as the best model in a comparison of several parametric mortality models (see the original paper for further details).

All of the estimates are based on death counts and exposures from the Human Mortality Database (2016). The estimation of the gamma-Gompertz model and of the models used in the comparison rests on the maximum-likelihood framework, which assumes that age-specific death counts are Poisson-distributed (Brillinger, 1986). The data cover the age range 80 to 109 and the years 1960 to 2010.

3.3.2. Level of maximum mortality

Figure 3.5 depicts the distribution of the level of the mortality plateau across all of the countries and the periods analyzed. The histogram includes estimates for females and males. Additionally, the estimated density curves for females (red) and males (blue) are shown. The boxes at the top of the graph depict the interquartile range of the estimated mortality plateaus with the median (solid line) for both sexes combined (gray), and for females and males, respectively.

The median value of the mortality plateau for both sexes combined is slightly below one, which translates into a probability of dying of ≈63%. The median value for females is slightly above a level of 0.8 (≈55%). For males the median value is slightly below a level of 1.2 (≈70%). However, as the estimates for males in particular have large variations, the median value provides only limited insights. To investigate the role of population size as a potential source of variation, we employed a simulation: we fixed b at a level of 0.14 and γ at a level of 0.2, both of which resulted in a mortality plateau of 0.7, and varied the population size (1,000, 1,0000 ,and 100,000 individuals). The results of this simulation clearly show that variation decreases with increasing population size. For instance, for a population of 1,000 individuals, the interquartile range is between the levels of 0.58 and 0.92. However, this finding only partially explains the variation that is evident in Figure 3.5 ,because even in 1960, more than 10,000 males and females were alive at age 80 in Sweden, one of the smallest countries analyzed. A separate analysis by period and country also does not explain the variation. Therefore, the analysis was further restricted to countries with the best grade of data quality (Jdanov et al., 2008), high life expectancy, and a combined population size of at least 10 million individuals at the respective ages for the years 2005–2010. Using these criteria, the sample of countries consists of Belgium, France, Germany, West Germany, Italy, and Japan. The results of this more restricted analysis show a median value of 0.799 for females (IQR: 0.775–0.829; probability of dying: 0.55%), and no clear pattern for males. Hence, the median value is 1.246 (1.1324–1.5487; 71%). The considerable difference for males is primarily driven by the variation in the variance of the gamma-distribution (γ).

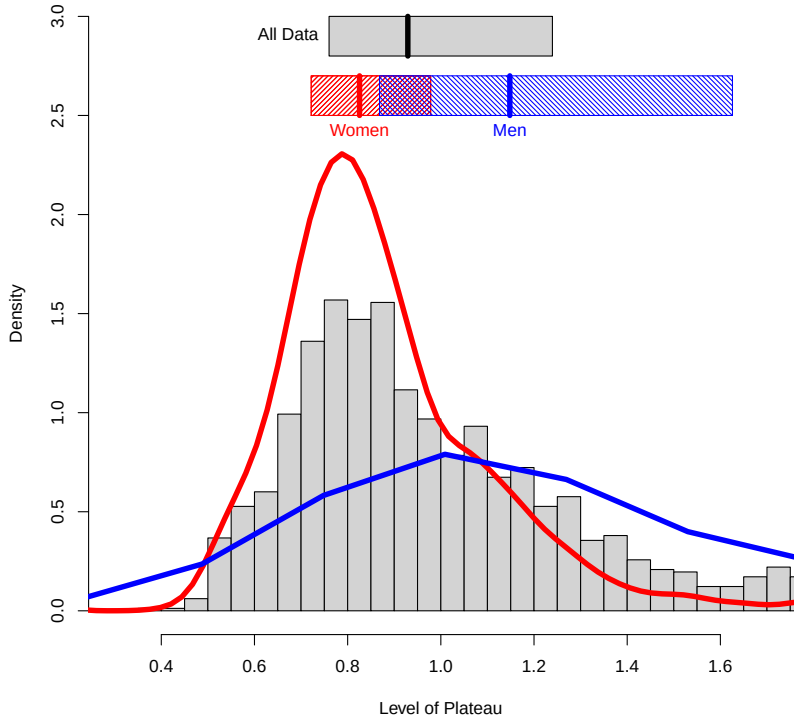

Figure 3.5.: Empirical Density Function of Mortality Plateaus for Women and Men, 1960–2010. *Data for Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany (West), Italy, Japan, Norway, Spain, Sweden, Switzerland, the United Kingdom, and the United States. The red curve is estimated density for women, and the blue curve is estimated density for men for the same countries and years. The horizontal rectangles are interquartile ranges of the mortality plateaus for both sexes combined (gray), women (red), and men (blue). The calculations are based on death counts and exposures from the Human Mortality Database (2016).*

Gampe (2010) estimated a level of 0.7 for the late-life mortality plateau, which is also the upper boundary of age-specific mortality. Of the 637 individuals included in Gampe's analysis, 573 were females. Thus, her estimates were mainly driven by female mortality trajectories. However, our results for females are not in line with Gampe's estimates, as we found a level of

0.8 for females. For males, we estimated an even higher level of 1.2. Given that Gampe's estimates are based mainly on females, the sex-specific differences are less surprising. However, the strong variation across the 16 countries and years analyzed is much more worrisome. All of our efforts to restrict the analysis failed to considerably reduce these variations. Therefore, we can carefully claim that our analysis provides evidence of a mortality plateau for females at a level of 0.8. For males, however, the results are much less clear.

3.4. Cluster conclusion

The age-specific boundaries of mortality can be regarded as extreme cases of the mortality trajectory. In both cases, humans have characteristics that make them more or less likely to die. but for the lower boundary, it is not clear whether and, if so, how the continuity of mortality improvements has affected the development. For the upper boundary, the changing age pattern of mortality improvement might act as an additional force of change. Based on this starting point, this cluster aimed to assess and explore the development at the boundaries of age-specific mortality, with a special focus on the lower boundary.

Three key characteristics of the lower boundary — level, location, and sex-specific difference — have been investigated for this purpose. Given the continuity of mortality improvements, the lower level might be of special interest. Our results show that minimum mortality has declined continuously across almost all of the countries analyzed, and particularly among the most recent cohorts. These findings support the expectation that the minimum mortality levels for both males and females are also likely to decrease in the near future. Apart from some rare exceptions such as Russia, all of the countries analyzed exhibit strong declines in the lower boundary over the study period. Moreover, in the majority of these countries, a stable pattern of improvement could be observed in the cohorts born since the 1970s. Hence, even after more than 160 years of continuous mortality improvements, opportunities for further improvements in the lower boundary of age-specific mortality remain. The results also

showed higher levels for males in the most recent periods. However, this has not always been the case. Among the oldest cohorts analyzed, females exhibited higher levels. More detailed investigations will be needed to explain this development and the excess male mortality in the most recent cohorts.

The location of minimum mortality decreased over time. Changes in related mechanisms, such as pubertal timing, are promising candidates for explaining this pattern. The location is also of general interest for mortality change, because at the minimum mortality age, the physiological and social constitutions of humans are most capable of withstanding death. Following Belsky et al. (2015), we could argue that aging research should focus on the age groups that are still in the very early stages of the aging process. In their study, they focused on individuals aged 26–38. Minimum mortality as such marks the inflection point between decreasing and increasing mortality, and could thus be interpreted as the point at which aging begins. Hence, investigating the aging process starting from the age of minimum mortality might be even more intriguing. We could then ask the following questions: Are there some lessons we can learn from the findings on minimum mortality about how aging and mortality might be postponed to or altered at later ages? Are we able to extend minimum mortality levels to the point of developing a second mortality plateau at younger ages? Or does minimum mortality reflect the baseline level of mortality if humans did not age? These are all non-trivial questions for which there may be no clear answers. However, they invite further investigation of the lower boundary of human mortality.

For the upper boundary, only the level has been investigated. It turned out that conducting this analysis alone was an ambitious undertaking. The results for males do not allow us to make any reliable claim about the level of the late-life mortality plateau. Although our results support the existence of such a plateau for males, further analysis is required. More importantly, we will have to wait until the number of male centenarians and semi-supercentenarians is higher. The results for females suggest a level of 80 deaths per 100 person-years lived. This is slightly higher then the level of 70 deaths per 100 person-years lived calculated by Gampe

(2010) using an alternative estimation procedure. The currently available data and our analysis setting do not permit to make any claims about time trends and changes in the location of the plateau.

When we look at the lower and the upper boundary together, it becomes clear that the span of age-specific mortality has increased, and is likely to increase further. This trend is attributable to a decrease in the lower boundary. This is an important finding for the development of mortality forecasting approaches in particular. These methods are designed to capture and to map the complete pattern and range of age-specific mortality. Hence, these approaches are needed to capture even bigger ranges of age-specific mortality, and so far do not require any assumptions about the minimum values until death rates are allowed to decline. The insights we gained are also of interest for those studying the frontiers of human mortality improvement. It seems that we are still far from reaching the frontiers at the lower boundary. For the upper boundary, however, it is difficult to interpret the results in light of potential frontiers. Using sophisticated approaches and data that cover time trends and include an investigation of the location of the mortality plateau could help to uncover clues about the upper frontiers.

4. Cluster III – Mortality dynamics in an era of old-age mortality decline

4.1. Cluster objectives

The onset of sustainable declines in old-age mortality had a wide range of implications for disease and mortality dynamics. In particular, lifespan disparity as an indicator of mortality dynamics has been affected by the change in the age pattern of mortality improvement. In a long-term perspective, life expectancy and lifespan are closely related: life expectancy increased, while lifespan variability decreased (Colchero et al., 2016). This dynamic is commonly labeled "compression of mortality"; a concept that was introduced in the seminal article by Fries (1980). However, the onset of the old-age mortality decline led to the emergence of new patterns that have been characterized as "expansion of mortality" and "shifting of mortality" (Rothenberg et al., 1991; Engelman et al., 2010; Canudas-Romo, 2008; Kannisto, 1996; Bongaarts, 2005). Each of these concepts describes different relationships between mean lifespan and lifespan dispersion. In particular, the shifting of mortality – a shift of the lifespan distribution to higher ages with an approximately constant shape – has been viewed as the successor to the compression of mortality pattern(Canudas-Romo, 2008; Kannisto, 1996; Bongaarts, 2005). In this scenario, life expectancy increases alongside stagnating lifespan variability.

The shifting of mortality is also an appropriate scenario for mortality change, under which a pattern referred to as the "postponement of senescence" could proceed. The postponement

of senescence describes a delay in the physical aging process of humans to higher ages, as distinct from a mere slowing of this aging process. Vaupel (2010) argued that this concept describes the pattern of changes in the occurrence of diseases and deaths at the population level, at least since the onset of the sustainable declines in old-age mortality. However, he also argued that the pattern of health improvements is much less clear than the dynamics of mortality improvement (Vaupel, 2010). Therefore, the postponement of senescence might be only one possible scenario that describes how mortality dynamics are altered through changes in health dynamics. From this example, however, it becomes clear that mortality changes must be accompanied by changes in health patterns. Moreover, Christensen et al. (2009), for instance, have reported improvements in several health measures over time.

Considering the complexity of mortality and health dynamics and its effects on several other aspects of mortality and health, which are generated with the onset of the sustainable declines in old-age mortality, measuring and quantifying these patterns poses challenges for existing models and measures. Moreover, the future trajectories of mortality and health patterns are highly dependent on the extent to which the opportunities for further lifespan extension at the highest ages are exploited. Therefore, the methods and models applied in this context should also be able to capture any new dynamics that may arise. However, given the enormous thematic range in this field, the work in this cluster focuses on three specific phenomena:

- the rectangularization of the survival curve;
- the effects of increasing longevity and changing disease incidence on the lifetime risk of getting a specific disease; and
- the contributions of different mortality dynamics to the increase in life expectancy.

With a focus on these three phenomena, the objective of this cluster is to provide solutions for the problems and the incidences of theoretical misspecification that are generated by the sustainable decline in old-age mortality. Our aim is to develop novel or refined tools and measures that provide deeper insights into recent changes, and that allow us to evaluate and gain a

better understanding of potential developments.

Papers in this cluster:

Ebeling, Rau , Baudisch (2018). Rectangularization reconsidered: the maximum inner rectangle approach. Published in *Population Studies*.

Ebeling, Modig, Ahlbom, Rau (2018). The effects of increasing longevity and changing incidence on lifetime risk differentials: a decomposition approach. Published in *PLoS ONE*.

Appended paper: Bergeron-Boucher, Ebeling, Canudas-Romo (2015). Decomposing changes in life expectancy: compression versus shifting mortality. Published in *Demographic Research*.

4.2. Outcomes for rectangularization of the survival curve

4.2.1. Objectives

The rectangularization of the survival curve describes the increasingly rectangular shape of the survival curve due to the postponement of deaths to higher and higher ages. This concept has become instrumental to the description and the analysis of the compression of mortality. Although this phenomenon has been described in earlier work (Pearl and Miner, 1935; Comfort, 1956), extensive research has been conducted since its recognition in the seminal article by Fries (1980). Most of this research has been focused on proving or disproving Fries' ideas (see among others Nagnur, 1986; Nusselder and Mackenbach, 1996; Cheung et al., 2005; Myers and Manton, 1984).

However, given the current state of knowledge, it can clearly be stated that several of the

assumptions Fries made in formulating his initial concept have become obsolete. Most importantly, reductions in oldest-old mortality were not anticipated in the original framework. The emergence of this trend in turn invalidated several (partly implicit) assumptions of Fries' theory. For example, the idea that life expectancy has "looming limits" has been rejected (Oeppen and Vaupel, 2002; Vallin and Meslé, 2009); the contributions of premature mortality improvements to increases in life expectancy have become negligible (Christensen et al., 2009); and Fries' assessment (p. 130) that "[...] there has been no detectable change in the number of people living longer than 100 years [...]" has been disproven (e.g., Vaupel and Jeune, 1995; Vaupel, 2010). Although we have not witnessed an increase in the maximum observed lifespan since the death of Jeanne Calment at age 122 in 1997 (Robine, 1998), Fries' prediction (p. 133) that "human life span may not be fixed but may be slowly increasing, perhaps a month or so each century" had already been exceeded by more than an order of magnitude with the increases that occurred between 1980 and 1997 (Wilmoth et al., 2000).

Furthermore, life expectancy gains can only be generated in Fries' framework by a decrease in lifespan variability (see, for instance, also Nagnur, 1986; Nusselder and Mackenbach, 1996). Compression would be completed when "under ideal conditions" (Fries, 1980, p. 132) lifespans were scattered in a normal distribution around a mean of 85.6 years, with a standard deviation of about four years (in 1989, Fries assumed wider intervals). Moreover, a newly emerging pattern such as the shifting of mortality does not lead to further rectangularization, but to increasing life expectancy (Kannisto, 1996; Bongaarts, 2005; Canudas-Romo, 2008). It is also clear that the almost constant difference between the mean and the modal age at death (Canudas-Romo, 2010) cannot be combined with normally distributed deaths across age, even with historically low levels of premature mortality.

Despite all of these drawbacks and flaws, the general underlying idea of rectangularization remains simple and intuitive, which makes the concept appealing. Furthermore, rectangularization is one of the few theoretical frameworks that incorporates the relationship between lifespan disparity and life expectancy. For these reasons, and in light of its frequent use in

mortality and health research, we believe that this concept should be refined and extended to allow for an incorporation of recent and potential mortality changes.

4.2.2. Maximum inner rectangle approach (MIRA)

When we attempt to extend the concept of rectangularization beyond its classical application and seek to incorporate recent and potential mortality changes into it, several issues arise. First, the rectangularization framework needs to be detached from its static perspective. Instead, the framework should dynamically capture mortality changes at all ages, and not just in the premature age range. Second, it would be beneficial if the concept included a measurement approach capable of differentiating between and quantifying premature/early and late changes in age-specific mortality. Third, it would be desirable if an extension of Fries' framework still allowed us to determine whether there are any impending limits to lifespan.

The framework proposed here, which is called the "maximum inner rectangle approach" (MIRA), is designed to address those issues. The approach uses two dimensions of rectangularization. We call the classical perspective "outer rectangularization" because it relates the survival curve, and, accordingly, life expectancy, to the maximum living potential. Hence, it compares the current experience with a theoretical maximum. There is, however, another perspective, which we call "inner rectangularization," that has so far largely been neglected. Defined as the rectangle with the largest area under the survival curve, it relates current lifespan equality to current life expectancy. The basic idea is illustrated in Figure 4.1. The bold solid line denotes a hypothetical survival curve starting from a radix of one and reaching zero at the highest attainable age ω. The thin solid line denotes the classic reference used to estimate the advancement of rectangularization: namely, the outer rectangle, which also expresses the maximum living potential. The dashed line depicts the newly proposed concept: namely, the maximum inner rectangle.

Outer rectangularization is the standard perspective of rectangularization, and captures

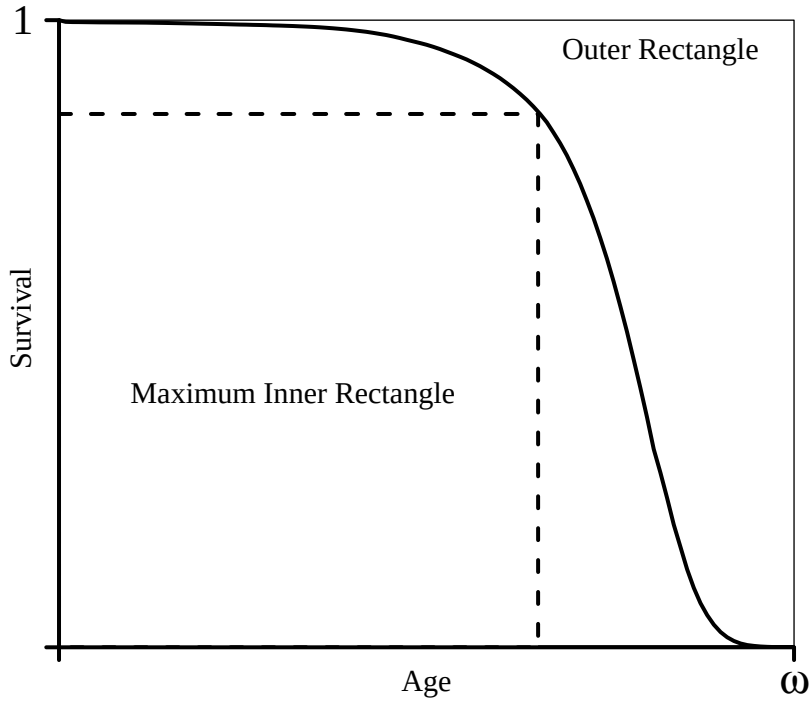

Figure 4.1.: Survival curve and its maximum inner rectangle and outer rectangle. *Own illustration.*

progress in mean lifespan,e_0, relative to progress in maximum lifespan, ω. The latter can be interpreted as the "the maximum living potential," which also defines the outer frame of the survival curve or the outer rectangle. It counts the hypothetical number of person-years that could be lived in a population if everyone survived to the maximum age and then died. In comparison, the actual number of person-years lived in a population corresponds to the area under the survival curve, and determines mean lifespan. Mean and maximum lifespan serve to capture the degree of outer rectangularization of the survival curve. We define the outer rectangle ratio (ORR) as

$$ORR = \frac{\int_0^{\omega} l_a \, da}{\omega} = \frac{e_0}{\omega}, \tag{4.1}$$

with l_a being the survival function at age a. The maximum age should be able to move forward or backward depending on the underlying mortality development. Therefore, we link ω to a specific survival proportion (k) – such as 1% of the population still alive – so that $l_\omega = k$.

By definition, $0 \leq ORR \leq 1$. The ratio relates the observed to the maximum possible number of person-years lived in a population. For example, if $ORR = 0.8$, then current living conditions allow the population to exploit 80% of current maximum lifespan potential.

Inner rectangularization adds a new perspective. In contrast to the outer rectangle, we seek the largest rectangle under the survival curve. Any inner rectangle (IR) under the survival curve is horizontally defined by age, x; and is vertically defined by survival to that age, l_x. Consequently, the corresponding area is $IR_x = x \cdot \ l_x$. The first age derivative of IR_x then identifies the age x^* that corresponds to the maximum inner rectangle (MIR) with an area of

$$MIR = x^* \cdot l_{x^*} \tag{4.2}$$

as the solution to

$$\frac{d\, IR_x}{d\, x} = 0. \tag{4.3}$$

Although there is no closed form solution, x^* can be found numerically given that

$$\mu_x > 0 \quad \forall\, x \in [0, \omega], \tag{4.4}$$

where μ_x denotes the force of mortality (see original paper for further details).

MIR counts "maximum uniformly shared person-years." It is determined by the maximum shared lifespan (x^*) and the survival proportion up to this lifespan (l_{x^*}). At ages $x < x^*$, the share of the population receiving an equal number of person-years would be larger than l_{x^*}, but the number to be received per individual would be smaller than x^*. Likewise, at ages $x > x^*$, the number to be received per individual would be larger than x^*, but the share of the population receiving an equal number of person-years would be smaller than l_{x^*}. In both cases, the total number of uniformly shared person-years would be reduced.

Using this definition of MIR allows us to add an inner perspective to the process of rectangularization. In analogy to the maximum living potential (ω), we can interpret life expectancy (e_0) as a population's current theoretical maximum number of life years that could be shared uniformly. Accordingly, with perfectly uniform lifespans, 100% of individuals in a population would share a lifespan of length e_0. When, however, actual lifespan equality is measured by MIR, a maximum survival fraction of $l_{x^*} < 100\%$ shares a uniform lifetime of at most x^* years. Thus, by relating MIR to e_0, we define inner rectangularization as the process of a population approaching its current lifespan equality potential. It is measured by the inner rectangle ratio (IRR), which is given by

$$IRR = \frac{MIR}{e_0}. \tag{4.5}$$

The inner rectangle ratio captures a trend that differs from that of the outer rectangle ratio, because changes in the MIR do not require a change in the mean or the maximum lifespan. Indeed, the trend could be characterized by a constant mean and a falling maximum lifespan, or by an increasing mean but a constant maximum lifespan, or even by a falling mean and a falling maximum lifespan. Though closely related, the inner rectangle ratio differs from the outer rectangle ratio because it is essentially an index of lifespan equality, while the outer rectangle ratio is an index of exploiting maximum living potential. Accordingly, if $IRR = 0.8$, then current living conditions allow the population to exploit 80% of its current lifetime equality potential.

Table 4.1 summarizes the quantities presented here, as well as additional areas and specific ages included in the MIRA. The table provides the name and the acronym of each measure, its mathematical expression, and a short interpretation of it.

As far as we know, there are no demographic predecessors to our concept of inner rectangularization. Thus, the age maximizing the inner rectangle provides a novel point of reference indicating maximum shared lifespan. This point represents the optimal trade-off between past lifetime and a sufficient number of survivors in terms of lived person-years. Hence, the principle of inner rectangularization rests on identifying the optimal combination of two (inversely

Table 4.1.: Quantities of the maximized inner rectangle approach (MIRA).

Name	Acronym	Expression	Interpretation
inner rectangle	IR_x	xl_x	Age-specific uniformly shared person years (PY)
maximum shared lifespan	x^*	$max[xl_x]$	Maximum number of uniformly shared life years by largest number of survivors
maximum proportion	l_{x^*}	l_{x^*}	Largest proportion alive at the maximum shared lifespan
maximum inner rectangle	MIR	$x^* l_{x^*}$	Population's current maximum amount of uniformly shared PY
life expectancy	e_0	$\int_0^\omega l_a \, da$	Population's current amount of PY, i.e. mean lifespan
outer rectangle	ω	ωl_0	Maximum possible PY
inner rectangle ratio	IRR	$\frac{MIR}{e_0}$	Proportion of uniformly shared PY from all PY lived
outer rectangle ratio	ORR	$\frac{e_0}{\omega}$	Proportion of PY lived from maximum possible PY
total rectangle ratio	TRR	$\frac{MIR}{\omega}$	Proportion of uniformly shared PY lived on maximum possible PY

related) inputs, age and survival, which unify the biggest area under a curve representing their respective relationships. Such measures have previously been applied elsewhere. For instance, the Hirsch index, or the h-index, measures the productivity and the citation impact of scientists (Hirsch, 2005). It depicts "x publications of a scientist have been cited at least x times." The geometric equivalent is a list of all publications by a scientist (y-axis) sorted by the number of citations (x-axis). This approach is similar to our application, where the survival curves could be interpreted as a sorted list of life lengths (x-axis) of the population (y-axis).

4.2.3. Materials and data

MIRA quantities are computed using period life tables, which we estimated from death counts and corresponding exposures from the Human Mortality Database (2015a). We have chosen to highlight the trajectories of females in Sweden, Denmark, and Italy because these three countries provide three exemplary mortality developments. Furthermore, all three countries have sufficient data coverage over time. In estimating x^* and l_{x^*}, a key challenge we faced was that the data are only available in discrete integer units, but x and l_x need to be continuous. We estimated x^* in two steps. First, we smoothed the product of x and l_x with cubic splines using R's `splinefun()` function (R Core Team, 2017), which allows us to evaluate the function value at arbitrary precision. Second, we used R's general purpose univariate optimization function `optimize()` to find the maximum. A similar two-step approach with splines has been used in previous mortality research to estimate the modal age at death (Ouellette and Bourbeau, 2011). Other age estimates, such as ω or the threshold ages discussed in the next section, have been calculated using the same procedure.

In several empirical studies on rectangularization, the maximum age ω was not set at the actual age at which there were no survivors left in the life table population. Wilmoth and Horiuchi (1999), for instance, set the cutoff age at the point at which 0.1% of the population are still alive. Rossi et al. (2013) used the 10% threshold; and, most recently, Schalkwijk et al. (2016) used the 0.1%, 1%, and 10% thresholds. In our study, we opted for a threshold of 1%. Sensitivity analysis revealed that the actual choice for l_ω had only minor effects on the results. As the maximum age changes with varying survival fractions, the estimates of TRR and ORR change quantitatively. However, the patterns of the ratios remain stable over time.

4.2.4. Illustrative examples of the inner, the outer, and the total rectangle ratio

Each panel in Figure 4.2 depicts the inner (IRR, black line), the outer (ORR, light gray), and the total rectangle ratio (TRR, dark gray) for females in Italy (upper left), Denmark (upper right), and Sweden (lower left). Figure 4.2 illustrates the following key points:

Two important empirical findings can be derived from the results presented in Figure 4.2. First, we found that outer rectangularization has shown continuous gains over time. This is a consequence of the straight linear increase in life expectancy (Oeppen and Vaupel, 2002), which has been faster than the increase in the longest lifespans, as measured by ω. However, we also detected a considerably slower pace of outer rectangularization since the middle of the 20th century. Additionally, we saw no convergence of the TRR to the ORR. Both have been developing almost in parallel for about 160 years. If Fries' ideas were correct, we would expect to observe that the TRR undergoes a period of "catching-up" to the ORR until Fries' "ideal conditions" of a life expectancy of about 85 years are reached. So far, none of the selected countries has reached this level of life expectancy. Consequently, we should see a continued narrowing of the gap. It is therefore clear that Fries' concept of rectangularization needs to be revised.

Second, we found that inner rectangularization also increased rather uniformly until around 1950; and that the patterns thereafter cannot be summarized with a general trend, because they are rather country-specific: i.e., there was a steady increase in Italy, a slow increase in Sweden, and a slight dip in Denmark for two decades starting in about 1970. The country-specific patterns suggest that the forces behind this development vary. For instance, the steady increase in Italy suggests that the rise in life expectancy was accompanied by more growth of the maximum inner rectangle. The almost stagnating IRR in Sweden between 1960 and 1990 suggests that life expectancy increased in line with the maximum inner rectangle. Denmark's deviant dynamics suggest that the maximum inner rectangle was declining while life expectancy stagnated. The differences across countries appear to be attributable to the variation in the onset of

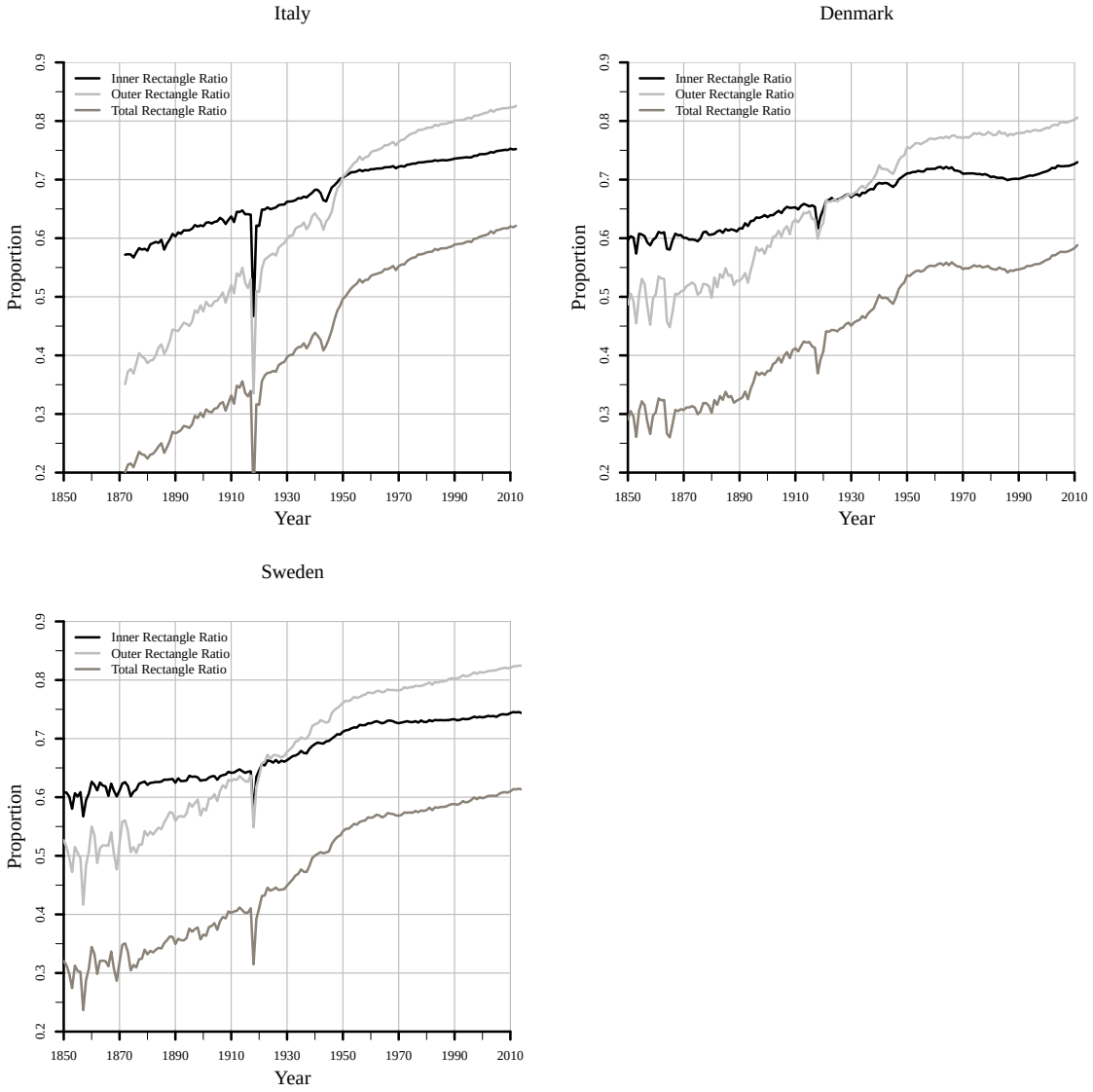

Figure 4.2.: Inner rectangle ratio, outer rectangle ratio and total rectangle ratio, Italy (1872-2012), Denmark (1850-2011), and Sweden (1850-2014), females, 1850-2013. *All calculations are based on period life tables in the respective year. The calculation is based on death counts and exposures from the Human Mortality Database (2015a).*

sustained mortality declines among the oldest-old (Kannisto, 1994), as well as to other factors, such as smoking among Danish women (e.g., Juel et al., 2000; Jacobsen et al., 2002; Lindahl-

Jacobsen et al., 2016), and reforms of the health care system (Mackenbach et al., 2011; Peters et al., 2015).

In this context, the inner rectangle ratio highlights how evenly the magnitudes and the trends (decrease or increase) of age-specific mortality changes are spread over age. We believe that these outcomes provide us with a new perspective on lifespan variability. Our intuition is strengthened by our finding of a correlation between the IRR and other summary measures of lifespan variability (see the original article for further details). This pattern is especially pronounced for the time period in which gains in premature survival were instrumental for the increase in life expectancy.

4.2.5. Applying the MIRA to separate premature from old-age mortality

Premature and old-age mortality are terms that are frequently used in mortality research, but they are often loosely defined, which might be sufficient for many applications. However, in analyzing rectangularization, how these terms are defined is a crucial issue. In Fries' description, premature mortality plays a central role. He argued that premature mortality decline drives the process of rectangularization, and implicitly assumed that premature mortality improvements alone are generating life expectancy increase. While how Fries defines premature ages is unclear, his descriptions suggest that he views life expectancy as a threshold. We argue that x^* can be interpreted as an age that allows us to separate premature from old-age mortality. Accordingly, the threshold in MIRA is based on the longest lifespan that is shared by the largest fraction of the population.

Figure 4.3 illustrates the relationship between x^* (horizontal axis) and l_{x^*} (vertical axis); i.e., the coordinates for measuring the number of maximum uniformly shared person-years, again for females in Italy, Denmark, and Sweden. The number of life-years lived in the maximum inner rectangle is depicted in the gray contour lines. The two time periods 1850–1950

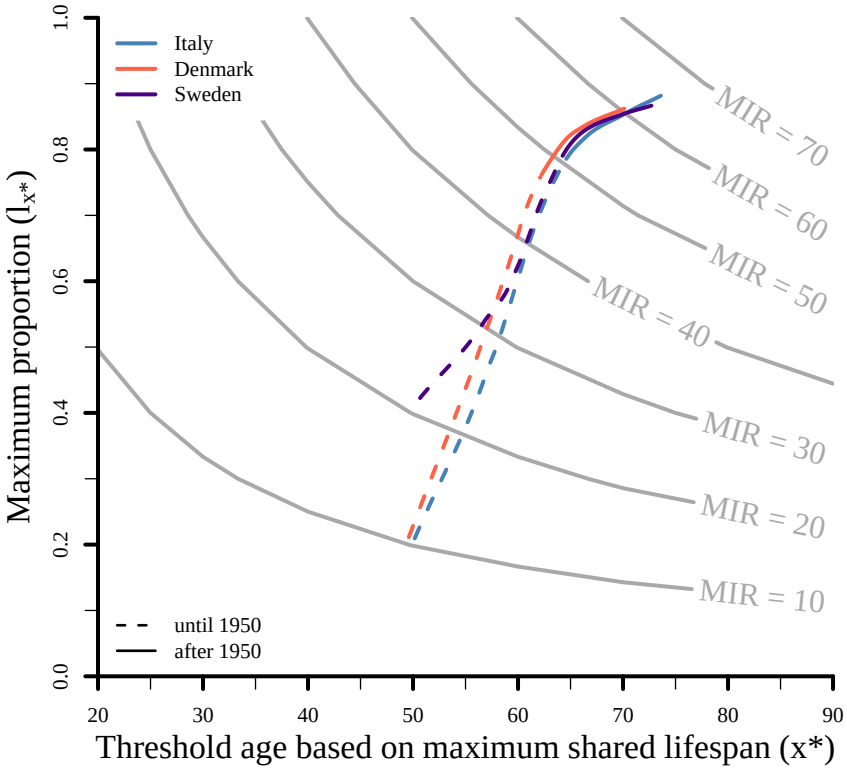

Figure 4.3.: Scatterplot between x^* and l_{x^*}, females, Italy (1872-2012), Denmark (1850-2011), and Sweden (1850-2014). *All calculations are based on period life tables in the respective year. Lines are based on a locally weighted smoothing to highlight the patterns only. Additionally, contour lines visualize the corresponding number of person-years lived in equality (MIR), since this is determined by the product of both. The calculation is based on death counts and exposures from the Human Mortality Database (2015a)*

and 1951–2014 are illustrated by dashed and solid lines, respectively.

The share of the life table population dying at old ages is denoted by l_{x^*}. Consequently, $1 - l_{x^*}$ equals the proportion dying prematurely. Generally, two trends can be distinguished: a "vertical" development (until 1950) and a "horizontal" development (after 1950). Premature

mortality improvements drove progress until 1950, which is illustrated by the increasing share of survivors. With improving old-age mortality, x^* shows an accelerated movement toward higher ages; whereas the corresponding survival fraction at x^* (l_{x^*}) shows only small gains. To compare the trajectories resulting from the MIRA, we also analyzed the relationship between the threshold ages and the corresponding surviving proportions (see the original article) for the alternative approaches by Zhang and Vaupel (2009) and Gillespie et al. (2014), which are based on the variance (Gillespie et al., 2014) and the number of life years lost (Zhang and Vaupel, 2009). These estimates show similar trends. However, within the 150 years of life expectancy development analyzed, the proportion dying at old age had changed relatively little under the threshold ages suggested by Zhang and Vaupel (2009) and Tuljapurkar et al. (2000). In both cases, the change amounted to less than 15 percentage points; a shift we consider to be rather small. In contrast, our measure shows a shift of about 65 percentage points, from 20% dying at old age in 1850 to about 85% dying at old age in the most recent years. These numbers seem to be more in line with the findings of, for example, Christensen et al. (2009), who estimated that almost 80% of recent gains in life expectancy for Japanese women were caused by survival improvements among the elderly.

The patterns of all three approaches suggest that there was a switch at around 1950 from avoiding premature deaths to extending the premature age range. This dynamic also points to a potential minimum proportion of individuals dying prematurely. Depending on the underlying definition of threshold ages, the share dying prematurely varies between 10%–15% (MIRA), 15%–20% (Gillespie et al., 2014), and 30%–35% (Zhang and Vaupel, 2009) under current mortality conditions. Hence, Fries' prediction that premature mortality would be almost completely eradicated seems rather unlikely. We can, however, see that the definition and the measurement of premature mortality are issues that have been unresolved at least since Lexis (1877).

4.3. Outcomes for estimating the effect of increasing longevity and changing disease incidence on the lifetime risk of getting a specific disease

4.3.1. Objectives

Summary measures of population health combine information on mortality and health outcomes to indicate population health within a single index (Murray et al., 2000). Moreover, these summary measures are useful in several situations, such as when analyzing the benefits of health interventions, identifying and quantifying health inequalities within the population, or monitoring changes in the health of a population and comparing levels of health across populations (Murray et al., 2000). Especially for the last two purposes, understanding differentials of summary measures between different time points or populations is essential. However, these differentials are not always easy to interpret because of confounding factors, such as the age structure of the population. To address these specific problems, decomposition techniques are applied (Canudas-Romo, 2003). These methods allow us to quantify the contributions of specific components to these differentials.

To examine the nature of such changes, Vaupel and Canudas-Romo (2002) provided a general procedure for the decomposition of population averages, which include a wide range of population summary measures. In addition to providing a formal derivation, they proposed a framework consisting of three different kinds of explanations that can be adapted to almost any evaluation of population summary measure differentials. They defined a level-0 explanation, which includes data or methodological problems. Their level-1 explanation refers to direct changes in the quantity of interest, such as improving health. The level-2 explanation captures possible confounding effects, such as changes in the population age structure. Given a specific population summary, decomposition methods allow us to quantify the contributions of factors belonging to level-1 or level-2 explanations, respectively. For many summary measures, such decomposition techniques have already been developed (see, for instance, Nusselder and

Looman, 2004).

The lifetime risk of getting a disease is another specific example of a summary measure. It expresses the probability of developing a certain disease throughout one's lifetime or from a certain age onward (remaining lifetime risk). It is a useful indicator for monitoring the burden of a disease in a population, and is frequently used in cancer research (Sasieni et al., 2011; Wun et al., 1998; Feuer et al., 1993; Goldberg et al., 1956). In many studies, lifetime risk is estimated based on longitudinal data for cohorts, such as the Rotterdam study, the Framingham Heart Study, or population register data (Heeringa et al., 2006; Vasan et al., 2002). In these approaches, lifetime risk refers to the observed lifetimes of individuals. However, lifetime risk has also been calculated using cross-sectional data (Sasieni et al., 2011; Goldberg et al., 1956; Karampampa et al., 2015). In such an analysis, lifetime risk rests on observed disease and death patterns at a specific point in time. For at least two reasons, using cross-sectional lifetime risks is a valuable alternative to relying on estimates based on longitudinal data. First, the data requirements are much lower when using lifetime risk, since having data with a sufficiently long follow-up time is not necessary. Second, a cross-sectional lifetime risk summarizes the current burden in a population, whereas a lifetime risk based on longitudinal data primarily summarizes past trends. Hence, a cross-sectional lifetime risk is a useful indicator for the above-mentioned situations in which summary measures are applied.

When calculating lifetime risk, the death (mortality rates) and disease (incidence rates) patterns at a specific point in time are assumed to apply as if a real cohort were passing through time. Accordingly, the disease of interest and death as the competing risk are the two possibilities for exiting from the population. The resulting lifetime risk estimate is determined by age-specific incidence and survival. Hence, differences in lifetime risk over time or between populations can arise from differences in incidence or in survival, but changes in just one of the two are unlikely in reality. Indeed, different authors have acknowledged that comparisons of cross-sectional lifetime risks suffer from increasing longevity, and thus from the interplay of mortality and health changes (Sasieni et al., 2011; Karampampa et al., 2015). For example,

a study on hip fractures documented an increasing lifetime risk, despite declining age-specific incidence rates (Karampampa et al., 2015).

Given that old-age mortality declines appear to be sustainable, a pivotal task we face is to better understand the influence of mortality dynamics on population summary measures of health. Using lifetime risk as a specific example, our research question may be formulated as follows:

- How much of the change in lifetime risk can be attributed to (I) changing survival; and (II) how much can be attributed to changing disease incidence?

Hence, the objective of this study is to provide a methodological solution for the outlined problem that allows us to answer this question.

4.3.2. Decomposition approach for lifetime risk differentials

Assuming that incidence rates depict the first occurrence of the disease of interest, the rate of either dying or getting diagnosed at age x, μ_x, can be written as

$$\mu_x = m_x + I_x \tag{4.6}$$

where m_x is the death rate at age x and I_x is the incidence rate of getting a disease at age x (Sasieni et al., 2011; Ahmad et al., 2015). The probability of staying alive and healthy within one age interval x can be expressed by $exp[-\mu_x]$[1]. Hence, the fraction alive and healthy at age x_i can be calculated by $exp[-\sum_{x \leq y < x_i} \mu_y]$[2]. The lifetime risk of contracting a disease from age

[1] To allow readers to comprehend the omitted steps and further details, the notation in this part of the thesis is similar to that in the original paper. This notation differs from that used in the rest of the thesis.

[2] Please note that the equations in this part differ from those published in the original article. In the original article, the equations, which express survival, contain an incorrect summation index. Please see the correction attached to the original article for further details. At the time of the submission of this thesis, an official correction was processed by the journal.

x onward, lr_x, can then be calculated by

$$lr_x = \sum_{x \leq x_i \leq \omega} I_{x_i}\, exp[- \sum_{x \leq y < x_i} \mu_y] = \sum_{x \leq x_i \leq \omega} I_{x_i}\, exp[- \sum_{x \leq y < x_i} I_y]\, exp[- \sum_{x \leq y < x_i} m_y], \quad (4.7)$$

where ω denotes the highest age attained.

We are interested in decomposing the change in the lifetime risk of contracting a given disease; denoted with Δ, between two time points A and B, or, more generally, the difference between two populations:

$$\Delta = lr_{x,A} - lr_{x,B}. \quad (4.8)$$

In the further derivation, we write ϕ_{x_i} for $I_{x_i}\, exp[- \sum_{x \leq y < x_i} I_y]$ and l_{x_i} for $exp[- \sum_{x \leq y < x_i} m_y]$. Given the general definition of lifetime risk of Equation 4.7, we can then rewrite Equation 4.8 to

$$\Delta = \underbrace{\sum_{x \leq x_i \leq \omega} [l_{x_i,A} - l_{x_i,B}]\, \frac{\phi_{x_i,A} + \phi_{x_i,B}}{2}}_{\text{Contribution of Changing Survival Conditions}} + \underbrace{\sum_{x \leq x_i \leq \omega} [\phi_{x_i,A} - \phi_{x_i,B}]\, \frac{l_{x_i,A} + l_{x_i,B}}{2}}_{\text{Contribution of Changes in Incidence}}. \quad (4.9)$$

Equation 4.9 provides two distinct interpretable terms (see the original paper for further details on the derivation). The left term expresses the contribution of changing survival conditions to the difference in the lifetime risk between populations A and B. The right term expresses the contributions of changes in disease incidence to the difference in the lifetime risk between populations A and B. Note that the methodological outline is based on previous studies that provided general results for mathematical problems of this kind (Kitagawa, 1955; Gupta, 1991).

Table 4.2 presents three hypothetical examples to that illustrate the decomposition as presented in Equation 4.9. In the first example (I), survival improves as reflected in the l_x–columns, while the incidence proportions are unchanged. The lifetime risk rose by 12 percentage points from $lr_A = 0.24$ to $lr_B = 0.36$. Because we subtracted B from A, we obtained a negative value. The contribution to the increase in the lifetime risk from changes in the incidence proportions (last column) is obviously zero, since $i_{A,x}$ and $i_{B,x}$ do not differ at any age.

Table 4.2.: Illustration of the decomposition method with hypothetical examples. (I) Changes in age-specific survival. (II) Changes in age-specific incidence. (III) Changes in age-specific survival and incidence. *In all three examples, we decomposed the change from A to B ($A - B$).*

Scenario	Age x	$l_{A,x}$	$l_{B,x}$	$\phi_{A,x}$	$\phi_{B,x}$	Contribution of Change in Age-Specific Survival†	Contribution of Change in Age-Specific Incidence‡
	1	1.0	1.0	0.0	0.0	0.00	0
(I) Change in	2	0.7	0.8	0.2	0.2	-0.02	0
Survival	3	0.2	0.4	0.3	0.3	-0.06	0
	4	0.1	0.2	0.4	0.4	-0.04	0
$lr_A = 0.24$ $lr_B = 0.36$	$\Delta = -0.12$				$\sum$	-0.12	0
	1	1.0	1.0	0.0	0.0	0	0.00
(II) Change in	2	0.7	0.7	0.2	0.1	0	0.07
Incidence	3	0.2	0.2	0.3	0.4	0	-0.02
	4	0.1	0.1	0.4	0.2	0	0.02
$lr_A = 0.24$ $lr_B = 0.17$	$\Delta = 0.07$				$\sum$	0	0.07
	1	1.0	1.0	0.1	0.2	0.000	-0.100
(III) Changes in	2	0.5	0.8	0.2	0.1	-0.045	0.065
Survival **and** Incidence	3	0.3	0.4	0.3	0.4	-0.035	-0.035
	4	0.1	0.2	0.4	0.2	-0.030	0.030
$lr_A = 0.33$ $lr_B = 0.48$	$\Delta = -0.15$				$\sum$	-0.11	-0.04

† Estimated by first part of Equation 4.9.

‡ Estimated by second part of Equation 4.9.

As expected, the difference in the lifetime risk can be completely attributed to improvements in survival.

A complementary picture is provided by the second example (II). Age-specific survival

does not differ between time points A and B. Instead, the age-specific incidence proportions changed over time. The overall reduction in lifetime risk of 0.07 is therefore equivalent to the sum of the contributions from the different age categories.

Although all of the examples are hypothetical, the third example (III) is probably closest to reality because the contributions to lifetime risk differences originate from changes in age-specific survival, as well as from changes in age-specific incidence proportions. The lifetime risk increased by 15 percentage points, from 0.33 to 0.48. Our decomposition method allows us to disentangle the overall effect into contributions due to varying mortality conditions and varying age-specific incidence proportions. It turns out that the increase in lifetime risk is due to a combination of higher incidence proportions and higher survival. In addition to offering this qualitative assessment, we can state that improved survival contributed almost three times more (0.11) to the increase in lifetime risk than the actual incidence risk (0.04).

4.3.3. Materials and data

We also applied the method to the remaining lifetime risk of being diagnosed with myocardial infarction, colorectal cancer, or hip fracture among Swedish males at age 60. Note that the decomposition can be applied to lifetime risk starting at any age. For the different diseases, we compared the lifetime risk between two different time points. These time points are 1987 and 1994 for colorectal cancer, 1994 and 2014 for hip fractures, and 1994 and 2004 for myocardial infarction.

The incidence estimates for the three disease outcomes were obtained from Swedish registry data maintained by Statistics Sweden and the National Board of Health and Welfare (Modig et al., 2013). For myocardial infarction and hip fracture, the first event occurring after age 60 after a seven-year disease-free period was identified using data from the National Patient Register. For colorectal cancer, information on the date of cancer diagnosis was collected from the Swedish Cancer Register. The incidence counts have been smoothed across age and time

to reduce random fluctuations using the MortalitySmooth package in R (R Core Team, 2017; Camarda, 2012). In our example, death rates are based on death counts and exposures of the total Swedish male population provided by the Human Mortality Database (2015b) (see the original article for a discussion on the impact of using death rates of the total population on the estimation of lifetime risk).

4.3.4. Decomposition of the lifetime risk of myocardial infarction, hip fracture and colorectal cancer for Swedish males

The left panel of Figure 4.4 depicts the time trends of lifetime risk for the respective diseases[3]. The dots mark the time points that are selected for the decomposition. The selection is based on the pattern of the respective lifetime risks. Accordingly, for myocardial infarction, we have chosen the end points of a period with almost stagnating levels of lifetime risk. For hip fracture, the time points mark the boundaries of a period of slightly increasing lifetime risk. For colorectal cancer, the time points mark the boundaries of a period of increasing lifetime risk. The right panel illustrates the results of the decomposition. The bars show the contributions of changing survival and of changing incidence, as well as the total change in the lifetime risk.

In the case of myocardial infarction, the lifetime risks are similar at the two time points, which could lead us to conclude that there have been no improvements in the incidence above age 60. However, when applying the decomposition, it becomes clear that the declining incidence should have generated a decrease in the lifetime risk of more than three percentage points, but that increasing longevity prevented such a decrease from occurring. If mortality had changed between the two time points, the mortality improvements — and, hence, the higher number of individuals surviving to older ages — would have resulted in an increase in

[3] After the adjustment for the incorrect summation index, the empirical results changed slightly. Therefore, the estimates in Figure 4.4 differ slightly from those in the graph of the original article. Please see the correction attached to the original article for further details. At the time of the submission of this thesis, an official correction was processed by the journal.

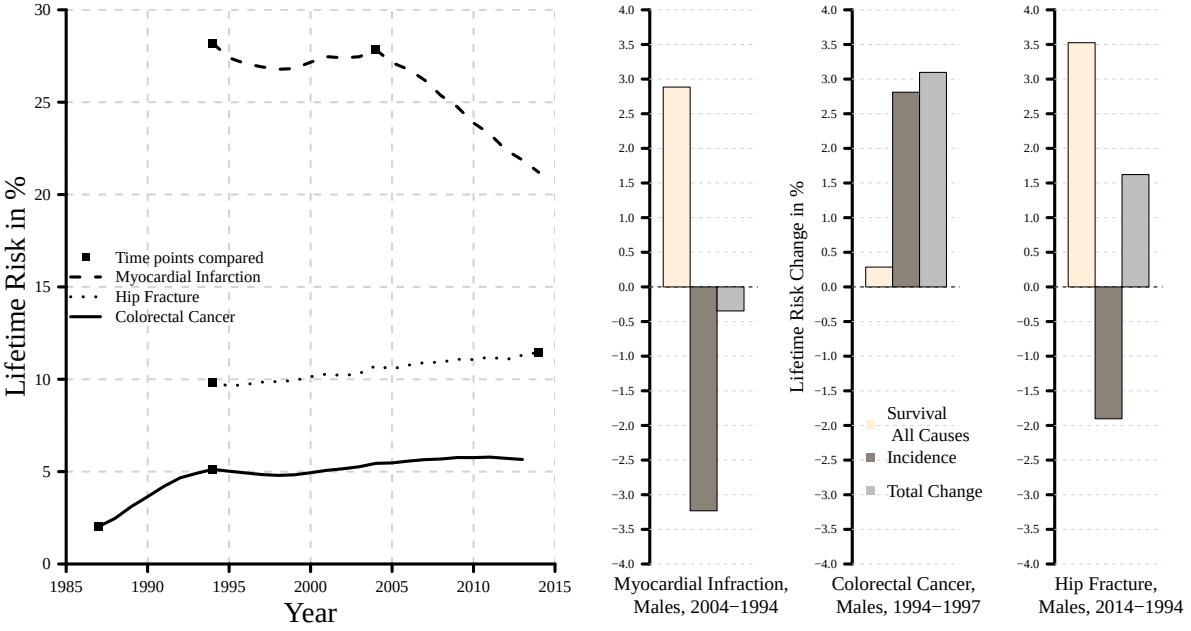

Figure 4.4.: Remaining Lifetime Risk at Age 60 and Lifetime Risk Decomposition for Myocardial Infarction, Hip Fracture, and Colorectal Cancer, Sweden, Males. *Lifetime risk estimates are based on combing death counts, exposures and disease incidence of a certain period in a multiple-decrement life table. Death counts and exposures are provided by the Human Mortality Database (2015b). Information on disease incidence are obtained from Swedish registry data maintained by Statistics Sweden and the National Board of Health and Welfare (Modig et al., 2013).*

the lifetime risk of more than 2.5 percentage points. In sum, the counteracting factors resulted in an overall change in the lifetime risk of less than a half percentage point.

For hip fracture, we observe a slight but steady increase over time in the remaining lifetime risk above age 60. This rise is, however, entirely driven by increasing longevity. Given the same mortality at both time points, the declining incidence should have contributed to a decrease in the lifetime risk of more than 1.5 percentage points; whereas given the same incidence at both time points, the survival improvements should have generated an increase of

more than three percentage points. Accordingly, the total change sums up to an increase of more than 1.5 percentage points.

Colorectal cancer is an example of a disease for which the lifetime risk increased relatively steeply over a short period of time. Unlike in the other two examples, the incidence contributed to a rise in the remaining lifetime risk at age 60. Consequently, the incidence between 1987 and 1994 increased. Rising incidence alone should have generated an increase in the lifetime risk of more than 2.5 percentage points. Moreover, the effects of improving survival on changes in the lifetime risk are different from those in the other two examples. Because, on average, colorectal cancer occurred earlier, its impact on longevity advancement was smaller. Hence, given the same incidence at both time points, the contribution of increasing survival should have resulted in an increase of slightly more than 0.25 percentage points. Thus, the total change sums up to an increase in the lifetime risk of more than three percentage points.

As we have seen from these empirical examples, whether increasing longevity influences the development of lifetime risk, depends on the timing of the respective disease. In recent decades, mortality improvements have been especially large at higher ages (Christensen et al., 2009). Accordingly, the lifetime risks of diseases that tend to occur at higher ages are influenced to a greater extent by increasing survival. This is because improved survival causes a higher number of people to survive to the ages at which disease incidence is highest. Thus, even when the incidence of a disease is going down, the declines at those ages are offset by the higher number of people at risk. Conversely, for diseases that tend to occur at ages at which survival improvements have been marginal, incidence declines of the same magnitude should have a stronger effect on lifetime risk. That is because the numbers of survivors at those ages differ only marginally between the two populations.

The decomposition method presented above allows us to quantify these conflicting dynamics. The method can also be extended beyond the application presented here. In addition to being useful for quantifying the contributions of incidence and survival to changes in life-

time risk, the general procedure can be adjusted to incorporate the contributions of changes in disease-related mortality and case fatality.

4.4. Outcomes for estimating the contributions of different mortality dynamics to the increase in life expectancy

4.4.1. Objectives

Our main objective has been to gain a better understanding of the influence of mortality dynamics on changes in summary measures of mortality, particularly in changes in life expectancy. Several techniques have been developed for decomposing changes in life expectancy using different components, such as cause of death and age, for discrete (Andreev et al., 2002; Pollard, 1982; Arriaga, 1984; Pressat, 1985) as well as continuous changes (Vaupel and Romo, 2003; Beltrán-Sánchez et al., 2008; Vaupel, 1986; Keyfitz, 1977; Horiuchi et al., 2008). These methods have been instrumental to efforts quantify and understand the changing age pattern of mortality improvement.

With the emergence of alternative patterns of mortality change, there has been an increasing interest in understanding the contribution of such dynamics to changes in life expectancy. For instance, Goldstein and Cassidy (2012) investigated how different kinds of mortality change translate into life expectancy changes. They found that slowing the relative increase of mortality with age would have the biggest impact on the level of life expectancy, given current levels of mortality. However, alternative dynamics such as a shifting of mortality – i.e., a pure postponement of deaths to higher ages without changing the relative increase – would also generate considerable gains in life expectancy.

So far, the compression of mortality and the shifting of mortality have arguably been the

dominant dynamics that have shaped mortality change since the onset of the sustained decline in old-age mortality (see among others Fries, 1980; Nusselder and Mackenbach, 1996; Bongaarts, 2005; Canudas-Romo, 2008). Hence, from that point onward, these dynamics have also driving changes in life expectancy. However, the specific forces generating changes in life expectancy differ for each of these patterns. The compression of mortality produces gains in life expectancy through declines in lifespan variability; while the shifting of mortality generates increases in life expectancy due to changes in the timing of mortality, without having an impact on lifespan variability. Although the approach by Goldstein and Cassidy (2012) allows us to quantify the impact of a particular dynamic on the level of life expectancy, it is solely based on a perturbation analysis. Hence, their method does not allow us to decompose a life expectancy differential into the respective contributions of the compression and the shifting of mortality. In general, methods that are designed to quantify the contributions of these dynamics to changes in life expectancy have, until recently, been lacking [4]. Hence, the objective of this study is to provide a methodological approach as well as some empirical results to answer the following questions:

- What is the impact of the compression and the shifting of mortality on the increase in life expectancy over time?
- How did one process replace the other, and to what extent did they do so?

4.4.2. Decomposing the contributions of different mortality dynamics to differentials in life expectancy

The following outline of the decomposition methodology uses the Gompertz mortality model as an example. However, the general idea can also be applied to other parametric mortality models (see the original article for results for other models).

[4] A recent publication by de Beer and Janssen (2016), which provides such an approach, might be seen as a follow-up to the approach suggested in the article.

Figure 4.5 shows the distribution of deaths for Gompertz parameters under two scenarios. It illustrates how changes in mortality can be decomposed into effects due to the compression and the shifting of mortality. Assuming a general change in mortality between the two distributions (arrow denoted as A), the shifting effect is the hypothetical change that results only if the modal age at death (M) changes between those two distributions (arrow B). The variability effect (compression) is the hypothetical change produced if only the slope of the hazard function (β) changes from one distribution to another (arrow C). The latter transformation C, of changing the slope of the hazard distribution, also changes the shape of the density distribution, and thus its variability (Wilmoth, 1997).

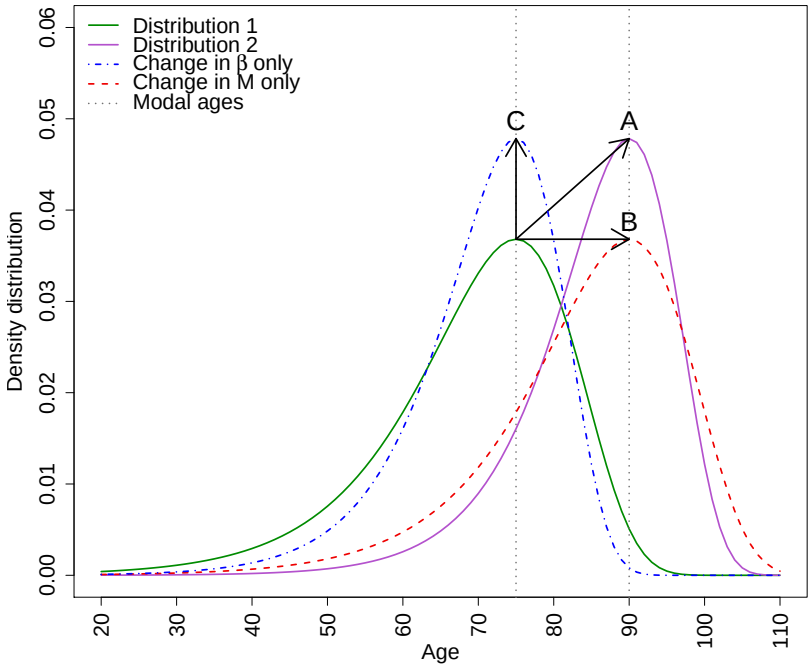

Figure 4.5.: Illustration of the shifting and the variability effects in the density function of the distribution of deaths for simulated data from a Gompertz model. *The different distributions shown rest on the combination of shape parameters* $\beta_1 = 0.10$ *and* $\beta_2 = 0.13$ *and modal ages at death* $M_1 = 75$ *and* $M_2 = 90$.

Following the illustration in Figure 4.5, the change in life expectancy at birth over time, $\dot{e}_{0,t}$,

can be expressed by

$$\dot{e}_{0,t} = \Delta\beta + \Delta M, \tag{4.10}$$

where $\Delta\beta$ and ΔM are the gains in life expectancy resulting from changes in the shape parameter and modal age at death, respectively. Following Vaupel and Romo (2003), a dot on top of a variable denotes its derivative with respect to time.

Following the work of Horiuchi et al. (2013) and Missov et al. (2015), the Gompertz mortality model can be calculated using the modal age at death. Accordingly, the force of mortality can be expressed by

$$\mu_{x,t} = \beta_t e^{\beta_t(x-M_t)}, \tag{4.11}$$

where β_t is the shape parameter at time t of the Gompertz hazard function $\mu_{x,t}$, and M_t is the modal age at death. The change over time in the force of mortality ($\dot{\mu}_{x,t}$) can be decomposed into the respective components of change for the shape ($\dot{\beta}_t$) and the mode ($\dot{M}_t$), which can be expressed by

$$\dot{\mu}_{x,t} = \dot{\beta}_t \left[\mu_{x,t}(\frac{1}{\beta_t} + x - M_t)\right] - \dot{M}_t \ [\beta_t \mu_{x,t}]. \tag{4.12}$$

The components of change for the shape ($\dot{\beta}_t$) and the modal age at death ($\dot{M}_t$) are each multiplied by a weighting function. For simplicity, these weighting functions are summarized by $f_\beta(\mu_{x,t})$ for the shape and $f_M(\mu_{x,t})$ for the mode. Equation 4.12 then changes to

$$\dot{\mu}_{x,t} = \dot{\beta}_t \ f_\beta(\mu_{x,t}) \ - \dot{M}_t \ f_M(\mu_{x,t}). \tag{4.13}$$

The change in life expectancy at birth over time can be expressed by

$$\dot{e}_{0,t} = \int_0^\omega \dot{l}_{a,t} \, da = -\int_0^\omega l_{a,t} \int_0^a \dot{\mu}_{x,t} \, d_x \, d_a, \tag{4.14}$$

where $\dot{l}_{a,t}$ is the time derivative of the survival function $l_{a,t}$. Equation 4.13 can now be substituted in Equation 4.14, which results in

$$\dot{e}_{0,t} = \underbrace{-\dot{\beta}_t \int_0^\omega l_{a,t} \int_0^a f_\beta(\mu_{x,t}) \, d_x \, d_a}_{\Delta\beta} + \underbrace{\dot{M}_t \int_0^\omega l_{a,t} \int_0^a f_M(\mu_{x,t}) \, d_x \, d_a}_{\Delta M}. \tag{4.15}$$

The first term in Equation 4.15 expresses the gain in life expectancy due to a change in the shape, ($\Delta\beta$), capturing the compression of mortality; while the second term expresses the gain in life expectancy due to a shift in the modal age at death (ΔM), capturing the shifting of mortality.

4.4.3. Materials and data

The Siler mortality model is used for the illustration of the decomposition method (Siler, 1983). Using the parameterization of the Gompertz mortality model with the modal age at death, the force of mortality of can be expressed by

$$\mu_{x,t} = \alpha_t \, e^{-b_t x} + c_t + \beta_t e^{\beta_t (x - M_t)}, \tag{4.16}$$

where α and c are capturing the initial level of infant and background mortality, the parameters b and β are the constant rates of mortality change with age for infant and adult mortality, and M is the modal age at death. By including the infant and background parameters, the Siler model provides a more detailed estimation of the compression and the shifting effects because it captures mortality at all ages. However, it must be noted that the Siler model appears to have some shortcomings in capturing the U-shaped mortality trajectory at infant, childhood and adolescent ages. It has also problems in capturing the deceleration of mortality at old ages. Nevertheless, it is useful for illustrating the method and for analyzing the impact of both mortality dynamics. The effect sizes calculated should, however, be interpreted with care (see the original paper for a further discussion on the accuracy of the method).

The decomposition is applied to the mortality of Swedish females and to the average female mortality in selected countries of the Human Mortality Database (HMD 2015c) (see the original paper for further details). The parameters of the mortality models are estimated for each country independently, and then averaged across all countries (with equal weights) to obtain the HMD average. The Siler models are fitted to the observed mortality trends using the maximum-likelihood framework and assuming that age-specific death counts are Poisson-

distributed (Brillinger, 1986).

4.4.4. Decomposition results using the Siler mortality model

To illustrate the accuracy of the method, the decomposition has been applied to the differences in life expectancy at birth between the years 1900–1905, 1950–1955 and 2000–2005. Table 4.3 shows the results for each setting, as well as for for each parameter in the Siler model. Although the method requires some adjustments to allow us to apply the continuous functions to discrete data and to capture the change over time (e.g., linear, exponential; see the original paper for further details) of the respective quantities, the results suggest that the proposed decomposition method provides fairly accurate results, which can be seen by the similar values for the life expectancy differentials and the sum of the contributions of the individual parameters.

To tackle the two research questions, Figure 4.6 depicts the results of the Siler decomposition for five-year periods between 1900–2010 for females in Sweden and the average across the countries of the Human Mortality Database. Each bar in the graph corresponds to the decomposition of the life expectancy differential between the start and the end year of the respective five-year period. The colors highlight the contributions of the individual parameters, with blue referring to the compression of mortality and orange referring to the shifting of mortality. Apart from showing some fluctuations in years affected by war or larger epidemics, Figure 4.6 clearly illustrates that the compression of mortality was the dominant force in increasing life expectancy until the 1950s. Thereafter, the shifting of mortality, measured by the change in the modal at at death, became instrumental to increasing life expectancy. Given the patterns shown in Figure 4.6, the transition from the compression to the shifting of mortality as the major driver proceeded relatively rapid. However, when we look at the relative rather than the absolute contributions (as shown in Figure 4.6), we see a more gradual shift from one to the other with an onset in the 1940s becoming visible.

Table 4.3.: Female life expectancy at age 0 ($e_{0,t}$) and its decomposition due to changes in the Siler parameters, Sweden and the HMD average, 1900, 1950, and 2000. *By rounding the numbers to the second decimal point in the table, the sum of the contributions ($\sum \Delta_i$) might differ slightly from the difference in life expectancy ($\dot{e}_{0,t}$). The estimation is based on age-specific death counts and exposures from the Human Mortality Database (2015c).*

	1900	1950	2000
$e_{0,t}$	56.06	72.78	81.68
$e_{0,t+5}$	58.00	74.44	82.34
$\dot{e}_{0,t}$	1.94	1.66	0.66
$\Delta\alpha$	0.52	0.06	0.01
Δb	0.54	0.02	-0.04
Δc	0.69	0.44	0.02
$\Delta\beta$	-0.17	0.03	0.05
ΔM	0.34	1.11	0.62
$\Delta\alpha$+Δb+ Δc+$\Delta\beta$+ΔM	1.94	1.66	0.66

The proposed method allows us to quantify the contributions of the compression and the shifting of mortality to the increase in life expectancy. However, because the procedure rests on the parametric mortality models, the results are sensitive to the selected model, and to whether the model used includes mortality at young and middle ages. Moreover, the method provides accurate results for decomposition over short time horizons, such as five or 10 years. But with longer time horizons, the sum of the individual parameter contributions start to deviate slightly from the initial difference in life expectancy. This is due to the approximation of the equations to discrete data, as well as the assumption of how a respective quantity changes between two time points.

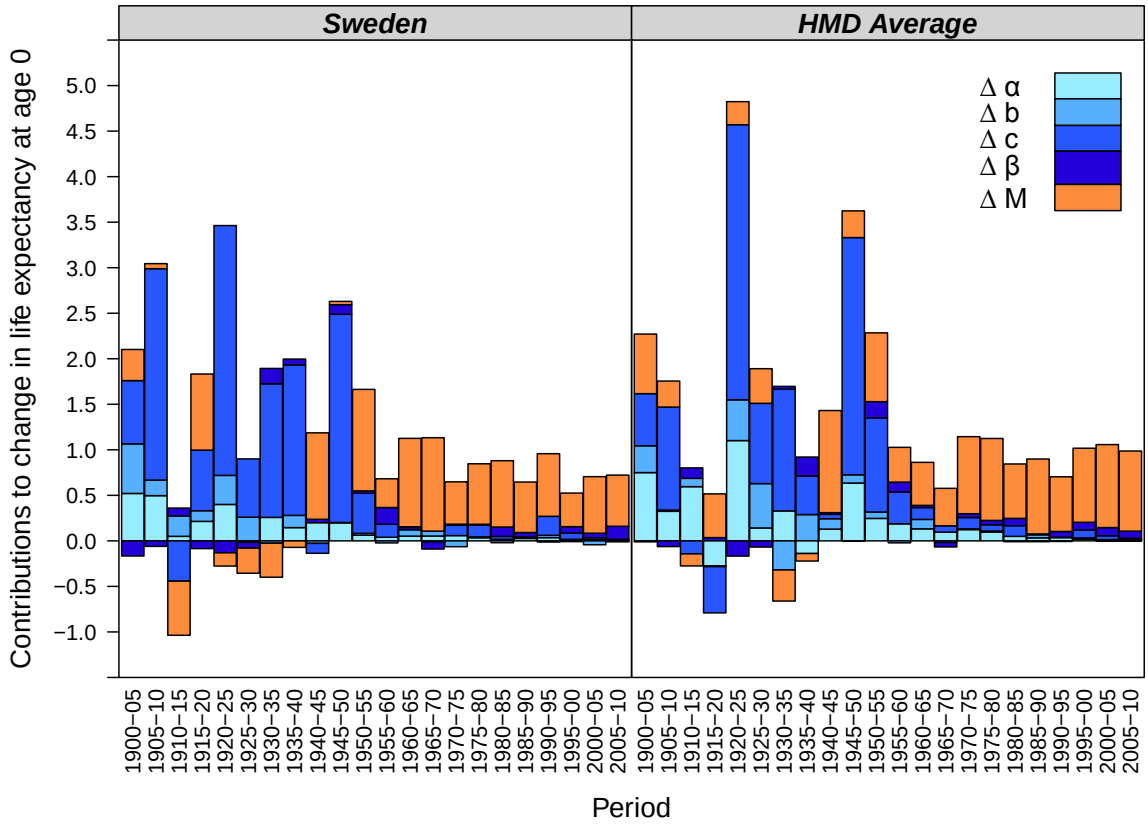

Figure 4.6.: Trends over time of the Siler parameters' contribution to changes in female life expectancy at age 0 ($\dot{e}_{0,t}$), Sweden and HMD average, 1900-2010. *The Siler models have been fitted using the Poisson-likelihood. The estimation is based on age-specific death counts and exposures from the Human Mortality Database (2015c).*

4.5. Cluster conclusion

The individual articles in this cluster aim to provide methodological solutions for problems, which are generated by sustained declines in old-age mortality. The maximum inner rectangle approach is the only approach that also contains a theoretical component. The two other approaches are decomposition methods for different types of differentials. However, in all three cases, the approaches aim to capture the influence of recent mortality changes, and they can also be used to anticipate and capture the patterns that may arise from further expected

improvements in old-age survival such as an expansion of mortality.

The three approaches tackle different problems that arise following the onset of sustained declines in old-age mortality. The classical idea of the rectangularization of the survival curve did not include the possibility that life expectancy would increase due to the decline in mortality at advanced ages. Therefore, a theoretical refinement was necessary. The maximum inner rectangle extends the whole concept by introducing a second type of rectangularization, which captures the degree of equality across the total person-years lived. The decomposition method for lifetime risk differentials captures the problem that arises incidentally from declining old-age mortality: namely, population aging. Migration and fertility are also important factors in this process. However, for summary measures of health and mortality, which rest on an observed or derived (from a model) population age structure, the increase in longevity must be taken into account when time trends or differentials are compared. Lifetime risk is a concrete example of such a measure, but the proposed procedure can also be applied to other summary measures that rest on the population age structure. The onset of the sustained decline in old-age mortality was also the time point at which dynamics other than the compression of mortality became instrumental for the change in life expectancy. The proposed decomposition method represents a first attempt to quantify the importance of these dynamics for the overall increase in the average lifespan; and to analyze whether, and, if so, how these dynamics (the shifting and the compression of mortality) have replaced each other.

The presented approaches can also be used to analyze and explain the dynamics of future mortality prospects. The progressive exploitation of opportunities for extending life at the highest ages — which is likely to continue occur, at least in the near future — will affect both the pattern of lifespan variability as well as the increase in longevity. The maximum inner rectangle approach and the life expectancy decomposition method are designed to capture potential changes in lifespan variability. In this context, the maximum inner rectangle approach offers further extension possibilities beyond the measures presented. Mostly importantly, the possibility of decomposing the complete maximum living potential into the different kinds of

person-years lived and lost represents a promising opportunity to gain further insights into the relationship between the compression and the shifting of mortality, as well as into the potential expansion of mortality as increasing life expectancy is accompanied by increasing lifespan variability. In addition to the decomposition method, lifetime risk as such is an interesting measure for anticipating changes in the life expectancy because it expresses the likelihood that an average individual will contract a disease in the future; a crucial information for anticipating future changes in life expectancy. It may therefore makes sense to combine information on survival and diseases incidence in a single index. Lifetime risk provides a unique opportunity in this context. Further extending this method, which allows us to decompose lifetime risk differentials into the components of disease-related mortality and case fatality, can help us better understand the dynamics of two potential drivers of increasing longevity.

5. Cluster IV – The role of mortality dynamics for future mortality prospects

5.1. Cluster objectives

Mortality forecasting approaches aim to predict future levels of life expectancy. Given the changes in the age patterns of mortality improvement — and perhaps more importantly, the location of improvements potentials at the highest ages — the accuracy of the approaches used to predict increases in life expectancy depends to a large extent dependent on their ability to capture and forecast mortality dynamics at the highest ages. However, these trends have been divers in the past. Figure 5.1 illustrates this diversity. The figure illustrates the correlation between life expectancy at birth (x-axis) and average life years lost at birth, $e_0^\dagger$ (y-axis) for females in Denmark, Italy, and Japan. Average life years lost at birth ($e_0^\dagger$) – a measure of lifespan variability – can be interpreted as an indicator of the underlying mortality dynamics, because the respective pattern of lifespan variability is determined by the distribution and intensity of age-specific mortality improvements.

Until the three populations reach a life expectancy level of 75 years, there are only marginal differences in the corresponding levels of average life years lost. However, as soon as the countries exceeded this level of life expectancy, the corresponding levels of average life years lost show diverse patterns, with a continuation of the regular decline (the compression of mor-

tality) for Italian females; an increase and a decrease for Danish females; and a leveling-off (the shifting of mortality) for Japanese women. These findings illustrate that different underlying mortality trajectories can lead to similar levels of life expectancy, but to varying levels of lifespan variability. Different authors have already discussed this phenomenon in more detail (Wilmoth and Horiuchi, 1999; Smits and Monden, 2009; Vaupel et al., 2011).

The diversity of mortality dynamics is a challenge for mortality forecasting approaches, because the future development of life expectancy depends entirely on whether and, if so, how improvement potentials at the highest ages are exploited. In Figure 5.1, we can see that these improvements can be realized through multiple dynamics, such as a further compression in the case of Italy or a shifting of mortality in the case of Japan. To provide robust forecasts of life expectancy, mortality forecasting approaches should be able to capture and process a variety of mortality dynamics. In this instance, lifespan variability could serve as an indicator of mortality dynamics, which could be used to evaluate the performance of mortality forecasting approaches in capturing and forecasting these mortality dynamics.

Basic life table functions, such as life expectancy at birth (a measure of central tendency) and age-specific mortality (a measure of mortality intensity) are usually applied to evaluate the performance of forecasting approaches. Accordingly, the more closely a forecast fits the observed values, the greater the forecasting performance is. Based on these assumptions, the goodness-of-fit test and other validation procedures are applied to quantify the predictive ability of mortality forecasting approaches. For the evaluation of the differences between the predicted and the observed mortality, ex-post quantitative aspects are generally used (Armstrong and Collopy, 1992; Cairns et al., 2011; Keilman, 1997; Shang, 2015). The quantified forecast errors can than be expressed in absolute or relative terms, and can be averaged over different dimensions, such as ages, time periods, or populations (Booth et al., 2006; Keilman and Pham, 2004; Koissi et al., 2006; Shang et al., 2011; Smith et al., 2001). Depending on the respective error statistic, different dimensions of predictive ability are evaluated, such as accuracy in the case of absolute errors and bias in the case of positive and negative errors. However, errors

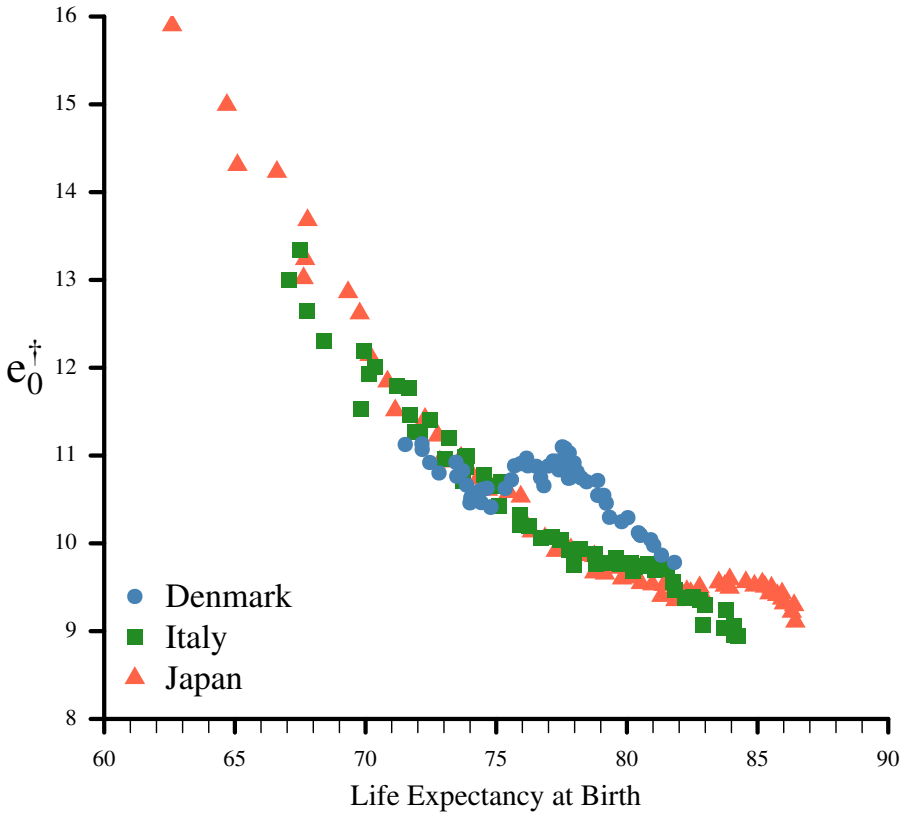

Figure 5.1.: Scatterplot of life expectancy at birth and average life years lost at birth due to death, females, Denmark, Italy and Japan, 1950-2012. *Life expectancy at birth and average life years lost at birth due to death are calculated using the approach presented in Vaupel et al. (2011). Accordingly, the average life years lost at birth due to death ($e_0^\dagger$) are calculated by applying the equation $e_0^\dagger = \frac{1}{l_0}\int_0^\omega e_x d_x \, dx$, with e_x being life expectancy at age x, d_x being the life table deaths at age x, and l_0 being the life table radix, to discrete data. Both life expectancy and average life years lost estimates are based on period life tables, using age-specific data on death counts and exposures provided by the Human Mortality Database (2017a).*

and test statistics of basic life table functions are useful for specifying precisely how mortality has been forecast. But small errors in the forecasting of, for instance, average lifespan do not necessarily indicate that the underlying mortality dynamics are plausible.

The objective of this study is to explore and highlight the question of whether mortality forecasting approaches can capture and forecast different trends in life expectancy at birth and lifespan variability. Hence, the aim is to emphasize the benefits of incorporating lifespan variability as an additional indicator in the toolkit that is used to evaluate the performance of mortality forecasts.

Paper in this cluster:
Bohk-Ewald, Ebeling, Rau (2017). Lifespan disparity as an additional indicator for evaluating mortality forecasts. Published in *Demography*.

5.2. Approaches used to forecast mortality

Three approaches are selected to forecast mortality. They differ in their ability to capture dynamic age shifts in survival improvement. These models are the Lee-Carter model (Lee and Carter, 1992), its rotating variant (Li et al., 2013), and the model developed by Bohk and Rau (Bohk-Ewald and Rau, 2017). As their levels of modeling flexibility differ, each of these approaches model the various trends in lifespan variability differently. Many other approaches are equally appropriate for meeting our research objective. However, the aim of this analysis is to show the additional information that can be gained when evaluating the forecasted spread of mortality in the presence of different trends for life expectancy at birth and lifespan variability. Hence, an extensive evaluation of the performance of different forecasting approaches is beyond the scope of this study.

The canonical model by Lee and Carter (1992) is undeniably a standard approach in the field of mortality forecasting. The *Lee-Carter model* forecasts mortality by age and calendar year

on the logarithmic scale, while assuming that the relative changes in mortality are constant across the ages over time. Hence, if the survival improvements were relatively large at young ages and were small at old ages in the reference years, it was assumed that this proportion would be unchanged in the forecast years. The absence of a dynamic shift in survival improvements to progressively higher ages may induce (1) an underestimation of life expectancy at birth, as well as (2) a strong compression of deaths, which may in turn be accompanied by a strong decline in lifespan variability.

Many scholars have refined the Lee-Carter model to address the problem of the inflexibility in the age profile of mortality change (Booth et al., 2006; Shang et al., 2011; Soneji and King, 2011). An important step in this direction was taken by Li et al. (2013), hereafter called *Lee-Carter-rotated*, who implemented a time-variant age schedule of mortality change that rotates from a present level to an ultimate level. The timing and the pace of the rotation depend on the average lifespan, which has been forecasted in a previous step using the original Lee-Carter model. As soon as life expectancy at birth exceeds a certain level, the rotation starts and proceeds until life expectancy at birth reaches an ultimate level. The rotating age pattern basically induces a postponement of relatively large survival improvements from younger to older ages. Given that the average lifespan is forecasted using the original Lee-Carter model, it is important to note that the rotation only affects the underlying mortality dynamics, not the average level of mortality. Assuming a regular decline in mortality, we expect to find that like the original model, the rotated model may (1) underestimate the additional years of life; but that unlike the original model, it may (2) be able to forecast a mortality compression that is less strong because of its greater modeling flexibility.

The *model of Bohk and Rau* (Bohk-Ewald and Rau, 2017) provides an alternative strategy for forecasting that relatively large rates of mortality improvement proceed from younger to older ages. The model predicts survival improvements instead of death rates. Moreover, the Bohk-Rau model has a linear and an exponential core model for forecasting time series of age-specific mortality change; it uses simulation-based Bayesian inference to run those mod-

els and to estimate coherent changes in mortality among adjacent ages. Although both the Lee-Carter-rotated and the Bohk-Rau model allow the age profile of the rates of mortality improvement to change, the latter model appears to be more flexible, since it does not assume an approximation of an ultimate schedule. We expect to find that the Bohk-Rau model will perform similarly to the Lee-Carter-rotated model in forecasting average mortality and lifespan variability for populations with regular mortality declines. But we hypothesize that the Bohk-Rau model will perform even better in generating forecasts for populations with irregular mortality developments, because it is more adaptable to different forecasting situations.

5.3. Materials and data

In the evaluation, we examine whether each of the three models is able to generate precise forecasts of average lifespan and lifespan variability. The examples are designed to indicate whether the approaches can capture (1) regular and irregular trends of average lifespan, as well as (2) different dynamics in the age shifts of survival improvements. We have chosen to compare the forecasts for women in Italy (regular e_0 and $e_0^\dagger$), Japan (regular e_0 and irregular $e_0^\dagger$), and Denmark (irregular e_0 and $e_0^\dagger$) because in recent decades these groups have differed substantially in their levels of life expectancy and lifespan dispersion (see Figure 5.1).

We use death counts and exposures by single years of age from age zero to ages $110+$, and by calendar years from 1950 to 2009, from the Human Mortality Database (2015d). To enable the forecasting approaches to shift deaths to ages beyond $110+$, we have extended the age range to $130+$ with the Kannisto model (Thatcher et al., 1998) (see the original paper for further details).

To generate the mortality forecasts, we implemented all models in the statistical software R (R Core Team, 2017). We implemented the Bohk-Rau model and the Lee-Carter model in their original versions, but we made a few adjustments to the original version of the Lee-

Carter-rotated model. Most importantly, we set the onset of the rotation in the age pattern to a life expectancy level of 75 because differences in lifespan disparity started to develop for women in Italy, Japan, and Denmark as the average lifespan exceeded this value (see Figure 5.1). To avoid jump-off bias for the Lee-Carter model and the Lee-Carter-rotated model, we use the last observed death rates to forecast mortality.

To measure lifespan variability, we take the average number of life years lost at birth (Vaupel and Romo, 2003; Zhang and Vaupel, 2009), $e_0^\dagger$, estimated by

$$e_0^\dagger = \frac{\int_0^\omega e_a d_a \, da}{l_0}, \tag{5.1}$$

with e_a being the remaining life expectancy at age a and d_a being the life table deaths at age a, with both integrated from age zero to ω, the highest age at death. l_0 is the radix of the life table. A major reason why we have chosen $e_0^\dagger$ is that it is demographically interpretable as the average life years lost. Since $e_0^\dagger$ refers to the lost living potential, it also provides information about the capacity for further increases in life expectancy. We argue that these key features mean that $e_0^\dagger$ in particular is suitable to be used to evaluate the plausibility of mortality forecasts.

5.4. Evaluation results of the mortality forecasts

Our evaluation is based on four different evaluation settings, which vary in terms of their reference periods and forecast years. In these settings, mortality is always forecasted up to 2009, but using different reference periods (1965–1990 1960–1985, 1955–1980, and 1950—1975). Thus, the range of forecast years varies across these settings (the results for the last three settings can be seen in the original article). Figure 5.2 presents the results of the central validation setting. The mortality forecasts are based on the reference period 1965–1990. Mortality is then forecasted from 1991 to 2009. Based on these forecasted death rates, life expectancy at birth (e_0) and lifespan equality ($e_0^\dagger$) are calculated. Based on this setting, we are able to

compare the estimations with the actually observed values. The upper, middle, and bottom panels depict the results for life expectancy (left) and lifespan variability (right) for women in Italy, Japan, and Denmark. The observed trajectories are in black. The blue, green, and red solid lines refer to the Bohk-Rau model, the Lee-Carter-rotated model and the Lee-Carter model, respectively. Moreover, the forecasted years, from 1991 to 2009, are highlighted in gray; and the reference period, from 1965 to 1990, is highlighted in beige.

Besides employing visualization techniques for the evaluation, we calculated forecast errors to evaluate the forecasting performance of each method. To quantify forecast accuracy in terms of the mean and the spread of mortality, we use the Absolute Percentage Error (APE). This is a relative error that relates the absolute difference between forecasted and observed values to the size of the actual values. Since the APE can deal with measures of different scales, we use it to compare the forecasting performance (across time and by country) of the methods using e_0 and $e_0^\dagger$. Table 5.1 lists those MAPEs (Mean Absolute Percentage Errors) averaged over all four validation settings.

The fits of life expectancy at birth and lifespan variability basically appear to depend on the regularity of mortality trends and the ability of the approaches to capture them appropriately. Since the mortality developments among Japanese and Danish women were irregular, making precise forecasts for these groups is particularly challenging. Thus, the predictive ability of the approaches declines as the magnitude of the MAPEs increases. This effect appears to be greater for the Lee-Carter model than for the other two models, and it appears to be more pronounced in forecasts of lifespan variability than of average lifespan. For example, the greatest overall MAPE, 10.7%, is for $e_0^\dagger$ in Japan from the Lee-Carter model; whereas the smallest overall MAPE, 0.4%, is for e_0 in Italy from the Bohk-Rau model (see Table 5.1). The greater forecast error found for the Lee-Carter model is probably due to the extrapolation of average trends of the reference period. Hence, if the overall trend in lifespan variability is decreasing in the reference period, the Lee-Carter model may be expected to predict a decreasing pattern as well, and vice versa (see Figure 5.2). The structural breaks observed in Danish and Japanese

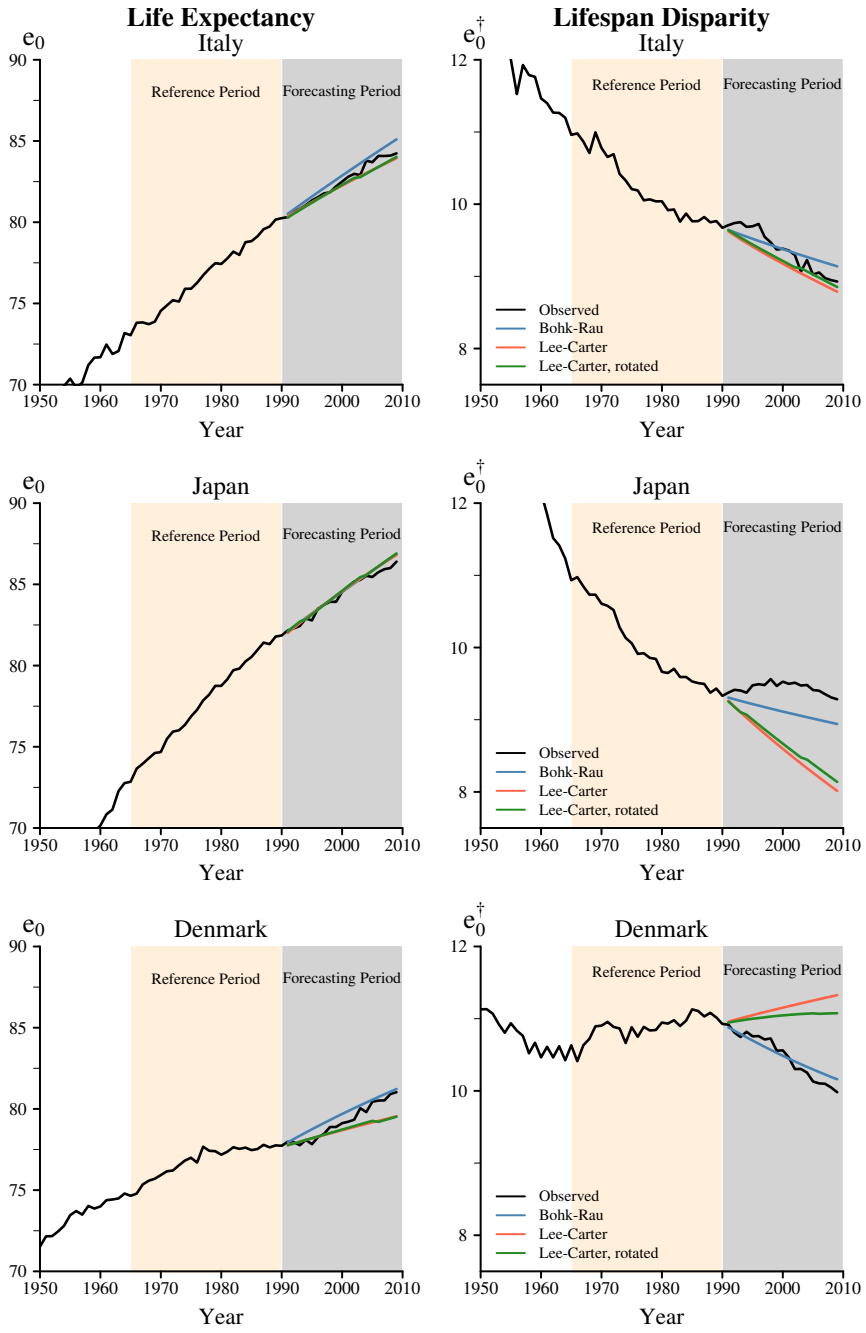

Figure 5.2.: Life expectancy at birth (left panels) and life years lost at birth (right panels) for women in Italy (top), Japan (center), and Denmark (bottom). *Observed data are in black, while forecasted data are in red (Lee-Carter model), green (Lee-Carter-rotated), and blue (Bohk-Rau model). The forecast years are 1991 to 2009, based on the reference period 1965 to 1990. All estimates are based on age-specific death counts and exposures provided by the Human Mortality Database (2015d).*

Table 5.1.: Mean of the Absolute Percentage Errors (MAPE) for e_0 and $e_0^\dagger$ over all validation settings by country and method. *The MAPE is calculated over the APE of all four validation settings. The reference periods (1965–1990 1960–1985, 1955–1980, and 1950—1975) and the forecast years of the settings differ. All of the forecasts range from the year after the end of the reference period until 2009. The calculations in all of the settings are based on age-specific death counts and exposures provided by the Human Mortality Database (2015d).*

Country	Measure	LC	LC, rotated	Bohk-Rau
Average across all validation settings				
Italy	e_0	0.011	0.011	0.004
	$e_0^\dagger$	0.027	0.019	0.024
Japan	e_0	0.008	0.008	0.005
	$e_0^\dagger$	0.107	0.086	0.025
Denmark	e_0	0.009	0.008	0.022
	$e_0^\dagger$	0.057	0.045	0.024

lifespan variability are unexpected, and are difficult for all of the models to capture since they are not designed for this specific task.

By focusing exclusively on lifespan variability, we are able to discern large differences between the approaches, particularly between the two Lee-Carter models in the forecasts of Japanese female mortality. The rotating variant appears to capture the flattening decline in lifespan disparity in the forecast years much better than the original model, and thus substantially improves forecasting performance with an overall MAPE for $e_0^\dagger$ in Japan, which is substantially lower for the Lee-Carter-rotated (8.6%, Lee-Carter model: 10.7%). This finding demonstrates the need for time-variant survival improvements in order to capture dynamic trends in the variability of the age at death. It should be noted that we do not expect forecasting errors to be equal to zero, since they show more signs of overfitting than of displaying high forecasting performance.

5.5. Cluster conclusion

The comparative analysis revealed that, irrespective of the reference period, forecasting performance basically depends on the regularity (or continuation) of mortality trends and the ability of the approaches to capture them appropriately. While the forecasts of life expectancy at birth generated by the Lee-Carter models are rather conservative, the forecasts generated by the Bohk-Rau model often have small forecast errors, but also a few upward outliers across all validation settings. Moreover, the Japanese forecasts were found to be precise when we looked at average lifespan only, but they turned out to be rather inaccurate when we took lifespan disparity into account as well. Hence, the models were not able to capture the flattening decline of Japanese lifespan disparity in the forecast years, although the rotating model and the Bohk-Rau model fared better than the Lee-Carter model due to time-variant survival improvements.

However, the remaining deviations from the observed values indicate that the refinement or the development of forecasting approaches should focus not only on average mortality, but on lifespan disparity. This may be particularly important given the concentration of mortality improvement potentials at the highest ages. Improving mortality at those ages would likely mean that people will be able to live beyond the current maximum ages. Hence, it is crucial that forecasting approaches are able to capture multiple trends in the (right) tail of the lifespan distribution (stagnation or expansion). The approaches should therefore be able to forecast further reductions in mortality not only up to the current maximum ages, but to higher ages and beyond. Reaching this goal will require a high degree of modeling flexibility, which has been missing in existing approaches.

To summarize, our analysis illustrates that the joint evaluation of the average lifespan, e_0; and the life years lost, $e_0^\dagger$; provide new insights that we believe are needed for a comprehensive evaluation of the predictive performance of mortality forecasts. We also suggest that these new insights are used when improving or developing new methods for forecasting mortality. Until now, these approaches were exclusively designed to capture the almost linear increase in life

expectancy at birth. Hence, it is not surprising that forecasts of the average lifespan turn out to be more accurate and yield smaller forecast errors. The incorporation of lifespan disparity as a quality criterion or even central outcome may substantially improve the methodology.

6. Conclusion

6.1. Assessing the consequences and the implications of the continuous mortality improvements

The primary aim of both the first cluster, measuring survival progress, and the second cluster, investigating mortality dynamics at the boundaries of age-specific mortality, was the assessment of mortality change. The initial motivation for undertaking such an assessment was the persistent continuity of mortality improvements (White, 2002; Oeppen and Vaupel, 2002; Vallin and Meslé, 2009). The two clusters addressed this issue from different angles. The first cluster focused on how the advancement of the continuous change is measured, while the second cluster looked at the implications of the continuous change for the boundaries of age-specific mortality.

The formulation of the concept of "equivalent time" (ET) and the classification of reference types are the two key outcomes of the first cluster. ET combines two common perspectives on assessing survival progress, gap and slope, into a single index. This index expresses differentials in the advancement of survival improvement in calendar years, which is a common unit for tracking the development of populations. In the study, we showed the benefits of ET and its advantages when it comes to assessing past progress and to gauging future prospects.

The proposal of the classification of reference trends is preceded by a question: What is

desirable progress? Is it sufficient to keep up with a reference trend, or should a population overtake the levels of a reference trend? Obviously, answers to this question can be given on a case-by-case basis only, and in light of the specific issues to be addressed. However, by restricting ourselves to overall survival progress, we can use the change in life expectancy as a basis of evaluation. Thus, the previous question could be reformulated as follows: Is keeping up with the increase in record life expectancy sufficient progress for a population with much lower levels of life expectancy? Or should this population be improving more quickly in order to catch up with the record values? Moreover, is record life expectancy an appropriate point of reference in this context? The proposed breakdown of reference types and comparison settings into intra-, inter-, and supra-groups can help us critically review and select the appropriate progress benchmark for addressing these questions.

The consequences and implications of the continuous mortality improvements for the lower boundary of age-specific mortality (minimum mortality) received special attention in the second cluster. Past developments of three key characteristics – the level, the gap, and the sex-specific differences – have been analyzed in order to gain a better understanding of the consequences and implications of more than a century and a half of continuous survival improvements. To my knowledge, this is the first investigation of this kind. The results represent trends at the population level, and are intended to stimulate follow-up questions for further investigations. The general findings can be summarized as follows: minimum mortality has decreased tremendously, and is likely to decline further; the location of minimum mortality has shifted to younger ages; and the sex-specific differences in minimum mortality changed from temporary female excess mortality to persistent male excess mortality. Hence, for minimum mortality, the main consequence of the continuous mortality improvement could be the decline to recent levels of eight females deaths and 10 male deaths per 100,000 person-years lived among the best practice countries.

What are the implications of the finding for minimum mortality? After one and a half centuries of continuous survival improvements, there is still room for further gains, even in the

historically lowest death rate across age. This finding alone might not be surprising. However, evidence of the continuity of the decline in minimum mortality among the most recent cohorts and of the likelihood of future improvements at the lower boundary are impressive signs of the ongoing diffusion of advances in various areas of life, and of how these advances continue to generate further survival improvements, especially in situations in which opportunities for improvement are arguably very small. This implies not only that the prospects for reducing mortality at the lower edge are good, it also shows that the opportunities for further mortality improvements at all ages are enormous; an important implication in particular by recognizing that it is sometimes assumed that mortality cannot be further reduced, despite the occurrence of major breakthroughs (Olshansky et al., 1990).

Understanding the implications of the shift in the location of minimum mortality is not straightforward, as the reasons for this change are unclear. Karapanou and Papadimitriou (2010) have reported that improvements in socioeconomic conditions in the 20th century resulted in an earlier onset of puberty, which can be seen in the decline in the average age at first menarche. Given the close association between the location of minimum mortality and the onset of sexual maturity, the shift may be attributed to similar advances, which are in turn the drivers of the overall mortality improvements. Thus, on the one hand, we could emphasize the positive effects of these advances; but one the other, we could focus on the negative consequences of earlier menarche, such as higher rates of cancer mortality, especially of breast cancer (Karapanou and Papadimitriou, 2010). The shift in the location to younger ages could therefore also be regarded as a "failure of success" (Gruenberg, 1977). In conclusion, the location of minimum mortality is a good example of the ambivalent implications of these general advances.

The investigation of maximum mortality, or of the level of the late-life mortality plateau, provided only limited insights. For females, a maximum level of eight deaths per 10 person-years lived was found, whereas the results for males did not allow us to derive a concrete empirical value. However, this project shed light on the problems that can arise when study-

ing mortality in this age range, particularly the lack of appropriate data. Hence, although mortality has been improving over a long period of time, it seems that we will have to wait even longer to collect the data we need to gain insights into the upper boundary. Once these data are available, examining the time trends in the level of the plateau and the ages at which it is reached could provide important insights into the dynamics of mortality change and the question of whether the continuous mortality improvements are large enough to alter essential features of the human mortality trajectory, such as the late-life mortality plateau.

Combining the findings of the two clusters allows us to draw one general conclusion: the *pattern* of advancement is often more informative than the actual *level* of advancement for understanding the consequences and implications of the continuous mortality improvements. ET, the different reference types, and the findings for the boundaries of age-specific mortality all indicate that studying delays in, speeds of, and patterns of improvement are more informative when accompanied by an exploration of the consequences and implications of continuous survival improvement.

6.2. Exploring the consequences and implications of the changing age-pattern of mortality improvement

The third cluster, mortality dynamics in an era of old-age mortality improvements, aimed to explore the consequence and implications of the changing age pattern of mortality improvement. The different articles focused on three specific aspects of this field: the rectangularization of the survival curve, the effects of longevity increases on lifetime risk differentials, and the contributions of different mortality dynamics to the increases in life expectancy. The objectives of all three studies centered on the sustained decline in old-age mortality, which generated various kinds of methodological challenges.

One type of method has been instrumental to providing answers to the different questions: the decomposition approach. Generally, decomposition methods are used to quantify the contributions of various components to the differences between two or more populations in a specific measure. The lifetime risk of getting a disease and life expectancy are the two central measures used in the presented approaches. To my knowledge, the method for the decomposition of lifetime risk is the first approach of its kind, whereas there are several existing approaches for the decomposition of life expectancy differences (see, for instance, Andreev et al., 2002; Pollard, 1982; Vaupel, 1986; Keyfitz, 1977). However, the decomposition method presented here employs a new perspective. It decomposes life expectancy differentials into the contributions of different mortality dynamics; more specifically, into the contributions of a general shift of deaths to higher ages (the shifting of mortality) and of a further compression of deaths toward the end of the lifespan (the compression of mortality).

Although the use of the maximum inner rectangle approach (MIRA) as a decomposition method has not been presented, the method can easily be applied in this way. However, the initial objective of the MIRA was to refine the framework of the rectangularization of the survival curve, which was established in a seminal article by Fries (1980). In addition to offering some modifications of the original framework, we proposed a novel type of rectangularization, inner rectangularization, which complements the usual perspective on this process. Commonly, rectangularization has been understood as the process of a population approaching its maximum living potential, which is given by the current maximum lifespan (see, for instance, Wilmoth and Horiuchi, 1999; Cheung et al., 2005). However, using the newly developed maximum shared lifespan as a reference point, inner rectangularization can be defined as the process of a population approaching its current maximum lifespan equality, which is given by the current level of life expectancy. Moreover, the inner rectangle ratio, the measure of inner rectangularization, extends the landscape of alternative lifespan variability measures by measuring the dispersion among the number of person-years lived, rather than effect or the size of person-years lost due to death.

In the MIRA as well as in the life expectancy decomposition, mortality dynamics are captured through the pattern of lifespan variability. In addition to representing methodological advances, both approaches provided new insights into the consequences of the old-age mortality decline for mortality dynamics. The application of the life expectancy decomposition to Swedish females showed that the shifting of mortality has gradually replaced the compression of mortality as the leading force of life expectancy increase; a process that has been underway at least since the 1940s. The application of the MIRA to the same population also confirmed the importance of this dynamic for the overall mortality change. However, the results of the MIRA also suggest that in recent years, the compression of mortality has been strengthening among Swedish females. Moreover, the results across all of the countries analyzed with the MIRA reveal the existence of various patterns of mortality dynamics in recent decades. For instance, an increase in lifespan variability was found among Danish females, which is believed to be a consequence of tobacco smoking (Lindahl-Jacobsen et al., 2016); while a continuous process of compression was observed among Italian females, even after the onset of old-age mortality declines. These findings highlight the country-dependent advancement of old-age mortality declines. The results also suggest that although life expectancy increases have been accompanied by a compression pattern over the long term (Colchero et al., 2016), new dynamics have been emerging since the onset of the sustained decline in old-age mortality. It thus appears that the age pattern of mortality improvement has been changing, as the evidence that new lifespan variability patterns are accompanying life expectancy increases indicates. The question of how future mortality change will alter the relationship between life expectancy and lifespan variability therefore remains open. The MIRA can be a valuable tool for answering this question.

With regards to the potential for lifespan extension at the highest ages, another closely related question arises: What are the future prospects for mortality dynamics? It is clear that old-age mortality improvements will continue to be needed, and that life expectancy will rise even further in the future. But how will these dynamics be shaped? The answer to this ques-

tion depends entirely on the plasticity of mortality at the highest ages. Several scenarios are conceivable, such as a further shifting of mortality in the coming decades. Alternatively, if mortality at the highest ages is difficult to change, a new era of compression with a further rectangularization of the survival curve may emerge. We can only speculate about the likelihood of either of these scenarios. An important determinant of future trends will be the development of health and its relationship to mortality. Looking at the current evidence, we see that even though we have already experienced an era with major innovations (Fogel and Costa, 1997), these general advances have resulted only in a delay of the aging process, which Vaupel (2010) called the "postponement of senescence." Thus, while we have succeeded in generating extra life years, it so far appears that we have not altered the process of deterioration with age. This development aligns almost perfectly with the shifting of mortality, which, given the results of the presented studies, seems to have been the predominant dynamic in many low-mortality countries since the 1950s. Moreover, the decomposition of lifetime risk as a measure while combining information on mortality and health has shown that increasing longevity and improving incidence levels can almost offset each other. Nevertheless, analyses of other populations are needed before we can reach any clear conclusions on this topic. Although only the lifetime risk decomposition refers to the direct intersection of health and mortality development, all of the presented methods allow us to investigate these processes and their consequences. The MIRA and the lifetime risk decomposition are also flexible enough to capture and analyze patterns beyond the shifting or the compression of mortality.

While the presented methods do not directly indicate how further advances in different fields, such as medicine or health care, might translate into mortality change, the outcomes arguably provide a sufficient basis to investigate their relationship. In particular, the MIRA represents a valuable approach in this context because future mortality dynamics will be determined by the distribution of these advances in the population. Accordingly, from a philosophical and from a future-oriented perspective, the MIRA can be interpreted as a measure of distributive justice in a population with respect to mortality. While individual lifestyle choices affect mortality, the provision, the quality, and the advancement of health care in a popula-

tion have important effects on lifespan variability. In that context, Marchand et al. (1998) discussed four alternative views that are applicable to equality in person-years lived: first, targeting a maximization of the total sum of health in a population would generate no further equality increases; second, reducing class inequalities in health would result in more equality; third, improving health for the lowest socioeconomic group would also increase equality; and, fourth, priority should be given to the sickest individuals, irrespective of their social class, which would potentially also increase equality. These third and fourth views are related to Rawls' "difference principle," as outlined in his work of political philosophy, "A Theory of Justice" (Rawls, 1971). Clearly, the evaluation, assessment, and determination of such strategies are difficult and complex tasks, but the consequences of population aging and increasing longevity might lead us to focus more on addressing these issues in the future. The presented methods can provide valuable insights from different angles that can prove helpful in this context. In addition to using the MIRA as a measure for lifespan equality, we could, for example, apply the lifetime risk decomposition in order to highlight how advancement translates into the improvement of different aspects, such as case fatality or disease-related mortality.

So far, the main focus of mortality research has been on the consequences of the old-age mortality decline. Yet the changes observed in the age pattern of mortality improvement show the decreasing contribution of mortality at younger ages to overall survival improvement. Using the MIRA to quantify young/premature and old-age mortality has provided intriguing insights into the importance of premature mortality. Since 1850, survival improvements have largely resulted in a continuously declining share of premature deaths. Obviously, continued improvements in old-age mortality are conditional on this development. However, as the age pattern of improvement changes, the share of the population who die prematurely tends to level-off. This lead us to formulate the hypothesis that in low-mortality countries, the proportion of premature deaths is going to reach a lower limit in the near future. The MIRA suggests that this limit could be at around 10%-15% of all deaths. However, the results of the second cluster challenge this hypothesis, because the past and the future declines in minimum mortality could be interpreted as signs of further declines in the share of premature deaths.

Although this is only one specific death rate, it is a death rate that is particularly interesting for investigating the frontiers of mortality decline, since it is the lowest rate across all ages. Hence, the falsification of this hypothesis remains a task for future research.

The shift away from saving premature deaths toward extending the premature age range is also associated with the changing meaning of chronological age: i.e., of who is considered young and who is considered old. The increase in life expectancy and the related evolution of new or extended lifespan stages, such as time spent in retirement, have fueled this debate, and have led to the proposal of some alternatives to the chronological age, such as subjective age (Kotter-Grühn et al., 2016). Nevertheless, this issue should receive further attention especially among demographers, since age is central variable in our discipline. Moreover, for the presented approaches, age remains an instrumental variable. However, the MIRA and the life expectancy decomposition have the potential to make contributions to this debate.

6.3. The prospects for prolonging life at the highest ages

Although our results do not provide any specific quantitative outcomes of future mortality development, the initial objective of the fourth cluster, understand how mortality dynamics affect future mortality trends, is an investigation of the prospects of prolonging life at the highest ages. The outcomes of the cluster are an evaluation of the capacity of our tools to provide us with the best possible information on future mortality prospects, or, in other words, with the best possible approaches for projecting mortality trends. Accordingly, the cluster illustrates the benefits of using lifespan variability as an indicator for mortality dynamics to evaluate the performance of mortality forecasting approaches.

The results can briefly be summarized as follows: as long as life expectancy increases were accompanied by a reduction in lifespan variability, the compared approaches performed well;

but when life expectancy increases were accompanied by other lifespan variability patterns, the compared approaches started to show deficiencies. It may be assumed that many mortality forecasting approaches anticipate the compression of mortality as the predominant force for further life expectancy change. But this assumption appears to be incorrect, because most approaches capture past trends that largely support the predominance of compression, and extrapolate them into the future. Accordingly, if we anticipate that this relationship will no longer hold in the coming decades, the analysis suggests some ideas that could be used to improve mortality forecasting approaches. Incorporating a flexible modeling of the dynamic age shifts of mortality improvements is of particular importance in this context. Lifespan variability can provide two focal contributions for this task. First, as was already mentioned, it can serve as a criterion for the evaluation of approaches. Second, and perhaps more important, lifespan variability could become a quantitative input for mortality forecasting approaches. For instance, parameters of lifespan variability and their likelihood could serve as priors for Bayesian mortality forecasting approaches. This is clearly a vague proposal. In fact, it should be understood as an illustration of the claim that while lifespan variability and, more generally, the range of mortality dynamics have so far been neglected component by forecasters, they could be used to increase the precision of mortality forecasts.

The findings of the fourth cluster bring us back to the question we raised previously: What are the future prospects for mortality dynamics? In the previous chapter, different aspects of this question were outlined. But for mortality forecasting, one more claim could be made. Expert opinions have been used as inputs for mortality forecasts (Booth and Tickle, 2008). Experts from different fields have, for example, been asked for their opinions on the future of a specific cause of death or on future levels of life expectancy, which have then been used as target values (Booth and Tickle, 2008). The variety of mortality dynamics since the onset of the sustained decline in old-age mortality, together with the prospects for lifespan extension (Sierra et al., 2009) are illustrative of the opportunities for altering human mortality that emanate from advancements in various fields. Thus, in order to broaden our perspectives, demographers should ask scholars in other fields about their expectations for future mortality

trends, and about how these expectations affect the approaches we use and the questions we ask in researching mortality.

6.4. Measuring trends and dynamics in an era of continuous mortality decline

The main focus of this thesis is on the consequences and implications of the continuous mortality decline for mortality dynamics. Three key findings – the remarkable continuity of mortality improvement, the changing age pattern of mortality improvement, and the location of longevity extension potentials at the highest ages – are the basis for the individual articles of this thesis. The articles explore in particular the various methodological challenges that are caused by the interplay of the above mentioned developments. Five out of the seven articles included in the thesis propose new or refined approaches. Three of these approaches use lifespan disparity as an indicator for mortality dynamics, while the two other approaches address the interplay of disease dynamics and longevity extension, and the measurement of survival progress in an era of continuous mortality declines. Although the two remaining articles propose no new methods, they apply existing approaches in a novel way to investigate the effects of the continuous mortality decline on the boundaries of age-specific mortality.

The application of these novel approaches, as well as the insights offered on mortality change at the boundaries, shed light on some of the challenges both demographers and the general public face currently, and will face in the future. In the past, survival progress has been remarkably stable and regular, especially among the leading western countries. Since the onset of the change in the age pattern, mortality improvements have continued to proceed relatively continuously, but appear to have slowed in a number of countries. Moreover, the level as well as the pace of advancement vary greatly across populations. Hence, the challenges we face are two-fold. First, what factors will drive future longevity increases in the leading countries, and how will these advancements shape mortality change? Second, what level of

progress is sufficient for the lagging populations to catch up with the leading populations? This thesis does not provide concrete answers to these questions, but it offers methodological opportunities to tackle these issues. Moreover, the development of the lower boundary of age-specific mortality has not only illustrated the opportunities for further advancements and for additional reductions in mortality; it points to the value of analyzing the potential failures of this success.

With the changing age pattern of mortality improvement, different kinds of mortality dynamics have emerged. This development and the prospects for further improvements at the highest ages suggest that the current variety of mortality dynamics will persist into the future, and may even expand as new patterns emerge. In particular, the evaluation of mortality forecasting approaches and the proposed lifespan variability measurements highlight the methodological challenges associated with this development. A central question in this context is how life expectancy and lifespan variability will be related in the future. Again, the proposed approaches and measure do not provide an instant answer to this question, but the evaluation of mortality forecasting approaches has demonstrated the need to seek out a range of answers to the question of how such approaches should be developed and refined. The proposed lifespan variability tools, as well as the lifetime risk decomposition method could be used to help answer this question. The MIRA refers to a further related question in this context: Namely, how are future mortality improvements altering lifespan equality? The availability as well as the distribution of drivers for further survival improvements, such as innovations in medicine or health care across and within populations, will be important determinants in either reducing or increasing lifespan equality. Given that resources are limited, this issue might be of particular interest for future research.

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7. Main research articles

7.1. Paper I: The concept of equivalent time as a simple indicator for the assessment of survival progress

RESEARCH

The concept of equivalent time as a simple indicator for the assessment of survival progress

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Abstract

Background: Mortality decline is a central aspect of the advancement in development. Differences in development are usually assessed based on the gap between two countries at a single point in time or by comparing rates of improvement over time. Here, we propose a third approach that combines these two indicators in a single quantity, which we will call *"equivalent time"* (ET).

Methods: ET translates the gap between a country's population and a reference population into a time lag. It expresses how many years we need to go back in time to find the value for the reference group that is equivalent to the value for the country's population. The translation from the gap to ET can be performed with any indicator, given that the values of the reference are changing monotonically over time. We illustrate the application of ET to US male life expectancy (LE) using three reference categories that cover different types of development benchmarks: cancer-free LE of US males (intra-group), LE of Japanese males (inter-group), and average LE of males in the G7 countries (supra-group).

Results: Between 1960 and 2015, LE increased almost continuously among the US male population and the three reference groups. The LE gap between US males and the inter-group reference category remained fairly stable, at a level of about three years; while the LE gap between US males and the intra-group reference category declined from one year higher in 1960 to four years lower in 2015. The LE gap between US males and the supra-group reference category increased from a small difference in 1960 to about three years in 2015. Thus, ET suggests that over the study period, US males continuously lost ground relative to these reference group: by 2015, the life expectancy of US males was lagging almost 20 calendar years behind that of the intra- and inter-group reference populations, and almost 15 calendar years behind that of the supra-group reference population.

Conclusion: ET represents an intuitive indicator for assessing differentials in survival advances that can be also applied in a broader context of development. Our illustrative examples demonstrate that ET might be a more sensitive measure than commonly applied approaches. ET thus has the potential to become a standard procedure for assessing survival progress and general development, complementing classical approaches.

Keywords: advances; gap; slope; references; mortality; progress; equivalent time

Background

Humankind has made substantial progress along important dimensions of development, including longevity, since the early 19th century, and especially since the end of World War II [1–3]. Spearheaded by a group of Western countries, progress

in living conditions diffused through most countries, albeit at varying paces [4]. Monitoring the state of development is crucial for evaluating the success or failure of past national policies, but also for identifying opportunities for further advancement. This type of monitoring is particularly important in cases in which a country is failing to keep up with the general trend, or has periods in which its development is stagnating, or even reversing.

One of the most important dimensions of development is the decline in mortality rates, which has been occurring almost everywhere in the world [5]. Thus, measuring survival improvements is among the main approaches used for tracking the general development of populations. For instance, the level of life expectancy is one of the three dimensions measured in the Human Development Index [6]. Furthermore, global goals and targets based on progress in mortality and health, such as the Millennium Development Goals and the more recently established Sustainable Development Goals [7], are instrumental to the development agenda of the United Nations. While mortality decline is observed in most parts of the world, the pace of decline varies greatly across regions and across social strata [8–10]. In addition to having societal relevance, monitoring and analyzing trends in survival progress are crucial to theory-building and modelling [11].

One of the most popular approaches used to assess and compare the decline in mortality in different countries is to compute the difference in life expectancy at a certain point in time, which we refer to here as the gap perspective. Alternatively, researchers can compare country-specific annual changes in life expectancy, which represent the pace of survival improvement. The goal of our paper is to (re-)introduce a concept that combines the gaps in and the pace of life expectancy changes in a more intuitive indicator, which we call *"equivalent time"* (ET).

Methods

The concept of equivalent time

In our analysis, survival progress is linked operationally to the level of life expectancy at birth (LE) over time. Thus, countries with higher levels of LE experience greater advances in survival progress than countries with lower levels of LE. Likewise, increases and decreases in LE over time reflect advances and declines in survival progress, respectively. While the former is expressed by the difference in LE between two countries at the same point in time (gap), the latter is measured by computing the absolute change over a given period of time within a country or a group of countries (slope). Hence, the gap might be understood as a static aspect of development, and the slope as a dynamic aspect of development.

We propose a third, previously neglected dimension for tracking development that expresses how far back in time we need to go to find the equivalent level of LE in a country relative to that of a reference group. This indicator is defined as the length of time the advancement of a country's LE lags behind that of a reference population (see Fig. 1). We label this quantity *"equivalent time"* (ET). An example: If

we observe that the life expectancy of the comparison population is five years lower than that of the reference population ($\Delta = -5$), and that LE in the reference population is increasing at an annual rate of 0.1 years ($\beta = 0.1$), then $ET = \Delta/\beta$, and we observe an equivalent time of -50 years. This value expresses that the progress of the comparison population lags 50 years behind that of the reference population. Or, in other words, the comparison population would need 50 calendar years at the given pace to reach the life expectancy level of the reference population at the point in time of the comparison. Hence, in cases in which the reference trend is linearly increasing, ET could be simply computed by dividing the gap by the slope. Thus, ET combines the static and the dynamic aspects of development in a single quantity, expressed as the delay in development.

To identify a unique ET, the reference trend must fulfill certain requirements. To exclude the possibility that there is more than one equivalent value, the reference time series must change monotonically over time. This could, however, be viewed as a minor issue, because we see fairly steady improvements in most measures of survival progress, such as life expectancy and mortality measures like, age-standardized death rates. Moreover, to provide a straightforward benchmark for progress, the cases chosen as reference usually have a steady pace of development. For the slope, it is not necessary to assume a constant change over time. It is also possible to directly compare empirical values without calculating a general slope.

Illustrative examples

Materials

To demonstrate the added value of ET, we compare the trends in life expectancy among US males to those of three reference groups based on the gap, the slope, and the ET between 1960 and 2015 (see Fig. 2). Our goal in selecting the reference groups was to ensure that we were covering the spectrum of reference types typically used in comparative studies. We label the three main types intra-group, inter-group, and supra-group. In the *intra-group comparison*, a counterfactual of the trends in the country of interest is used as the reference: namely, cancer-free life expectancy of US males. In the *inter-group comparison*, a second external group is used as the reference: namely, Japan the world leader in LE. Finally, in the *supra-group comparison*, a more general trend is used as the reference: namely, the average LE of the G7 countries. Obviously, which references are chosen depends on the research objective. Even if a specific reference fits the research purpose, it is important to take into account the specific features of the respective reference type (see Table 1). The results stemming from an intra-group comparison are not sufficient to determine whether the measured lags are a peculiarity of the respective population, or are occurring across several populations. Moreover, the outcomes of an inter-group comparison do not tell us whether the reference population is a prime example of the specific gains being evaluated, or is an outlier. Finally, the results of the supra-comparison do not indicate whether the more general reference trend is reflective of a common trend across populations.

Data

The estimates of life expectancy at birth for US males — the comparison group — are based on death counts and exposures from the Human Mortality Database [12]. In the intra-group comparison, cancer-eliminated life expectancy is used as the reference (see, for instance, Preston et al [13] for more details on cause-elimination life tables). Cause-specific death counts were obtained from the National Center for Health Statistics [14]. The total time period analyzed encompasses the years 1960 to 2015. Cancer deaths were reconstructed using the International Classification of Diseases (ICD) codes 140–239 (ICD 7–9) and C00–D48 (ICD 10). The calculation of Japanese life expectancy in the inter-group comparison and of the life expectancies for the G7 countries in the supra-group comparison were also based on death count and exposure data from the Human Mortality Database [12]. To ensure that the supra-group also covers the years 1960-2015 for countries that were missed in the most recent life expectancy estimates, values were obtained from the World Bank [15]. The equivalent time in all three examples was calculated using linear interpolation between the time series of the comparison population and the reference population. The whole analysis was conducted in the language R [16].

Results

Figure 2 shows the results for all three comparisons. The rows display the results for the intra-, inter-, and supra-group comparisons; while the columns show the life expectancy, the gaps, the slopes and the equivalent times. The slopes are calculated as annual increases over five-year periods, assuming a linear change between the respective time points.

The life expectancy level of US males increased continuously between 1960 and 2015 (panel a). Although an increase can also be observed for all three reference groups, the level of life expectancy differs across the groups, and compared to the US pattern (panels a–c). The LE gap between US males and the inter-group reference-category remained fairly stable at a level of about three years, while the LE gap between US males and the intra-group reference-category declined from one year higher in 1960 to four years lower in 2015 (panels d–e). The LE gap between US males and the supra-group reference-category increased from very small in 1960 to about three years in 2015 (panel f).

An assessment based on the pace of improvement alone provides limited insights. Both cancer-free and average G7 life expectancy had almost the same slopes as that of US males life expectancy across the different year groups; whereas Japanese life expectancy increased more rapidly until 1985-89, but changed at almost the same pace thereafter (panels g–i). However, in general, the slopes varied greatly over time, which makes it difficult to derive solid conclusions about the advancement of survival progress.

Compared to the gap and the slope, the ET shows larger differentials in the development levels of the reference groups and larger differences between the groups

in the advancement of survival progress over time. In the intra-group comparison, US male life expectancy in 1975 is the first value, which can be located on the reference trend line. In this year, the survival progress of US males lagged around 13 calendar years behind the level that hypothetically could have been achieved if cancer had been eradicated. This lag grew consistently over time to reach a magnitude of almost 19 calendar years in 2015 (panel j). In the inter-group comparison, we even see that the survival progress of US males was two calendar years ahead of that of Japanese males in 1960. But by 2015, this advantage had turned into a lag of almost 21 calendar years (panel k). In the supra-group comparison, US male life expectancy in 1970 is the first value, which can be located on the G7 average trend line. In this year, the survival progress of US males lagged around 10 years behind the average level of progress of the G7 countries. After a period of improvement, the lag among US males increased consistently to reach a level of 12 calendar years by 2015 (panel l).

In sum, the results indicate that the survival progress of US males has been increasingly falling behind that of other groups, as reflected in the large increases in ET among US males over the study period. This is a remarkable insight given that US life expectancy increased virtually throughout the whole study time at a pace that was almost comparable to that of the reference categories. At the same time, the gap between US life expectancy and that of the reference groups remained fairly stable, at least compared to that of the intra- and supra-group reference categories. This means that ET is a more sensitive measure of advances in survival than the comparison of gaps or slopes.

Discussion

In this paper, we have introduced a measure used to assess survival progress, which we labeled *"equivalent time"* (ET). The measure complements the current practice of comparing development at a single point in time and of comparing changes in development over time. It offers a simple, generic, and intuitive way to express in calendar years the delay in the development of a population in comparison to that of a reference population. We argue that ET has the potential to become a standard approach in the toolbox that is used for the assessment of survival progress in both large-scale cross-country comparisons and case studies, and for the evaluation of single countries. We used period life expectancy as an example to illustrate ET. Any other continuous variable that is monotonically increasing or decreasing over time could be used as well.

The simplicity of ET is, of course, also a shortcoming. Since it is build on a single variable, critics might see ET as an oversimplified measure that does not capture the multiple dimensions of development. This criticism is certainly justified. Thus, in cases in which reliable data on other dimensions are available, the use of more complex measures might indeed be more appropriate. However, for many countries, and particularly for longer historical periods, such data are often not available.

The idea of using lags to assess survival progress was already suggested by Stolnitz more than 60 years ago [17]. He applied this approach in studying the delays in mortality decline in non-western countries relative to those in western countries. Surprisingly, we are not aware of any other study that has applied this approach. More recently, Goldstein and Wachter used a comparable concept to study the relationship between cohort and period life expectancy within countries [18]. They defined the lag as "how far back in time from the current period we have to go to find a cohort with equivalent life expectancy" [18, pp. 259]. The general idea of time lags has also been used to evaluate the decline in age-specific mortality by expressing the improvement in ages with equivalent mortality levels, which are usually called "equivalent ages" [19, 20]. While these earlier studies focused on detailed aspects of mortality and survival, our definition of *ET* is more general and refers to cross-country comparisons of overall survival progress.

ET adds a dynamic component to research on differentials in survival advances; or, more generally, of advances in population development. The previous literature on this topic is dominated by studies examining the gap between specific measures; i.e., differences between countries at a certain point in time. For example, Drefahl et al. [21] used the declining ranking of Sweden across countries with the highest life expectancy as the initial impetus for investigating why Swedish life expectancy is "losing ground". By comparing the best- and the worst-performing US counties, Wang et al. [22] investigated the question of why US life expectancy lags behind that of other high-income countries, even though health care expenditures in the US are the highest in the world. Jasilionis and Shkolnikov [23] used life expectancy differentials between different educational groups to study the relationship between education and the longevity increase. They suggested that the trajectory of the highly educated group shows the way to higher levels of life expectancy for other population groups. The Human Development Index includes a measure based on the gap between an observed life expectancy and a hypothetical minimum life expectancy divided by the range between the hypothetical minimum life expectancy and the highest observed life expectancy in a specific calendar year [6]. The Global Burden of Disease research program [10] uses a similar approach to assess progress on a large collection of health and mortality indicators in 188 countries. Similar to the Human Development Index, the program's progress benchmark is a scale bounded by the best- and the worst-performing population for each indicator at a certain point in time. Since all of these studies target a dynamic research question, a delay-based measure such as *ET* could add to their analyses.

Using an illustrative example, we proposed a potential application of *ET* that complements classical approaches. The application of *ET* to the case of US males life expectancy between 1960 and 2015 revealed that LE in this group was increasingly falling behind, even though it increased almost continuously over the whole study period; and that the LE gap between US males and the reference groups was more or less constant over most of the study period. For instance, the application of *ET* showed that relative to their Japanese peers, US males experienced a delay in LE development of two decades. This is an intriguing result, especially given that

the US has the highest health care expenditures in the world [22]. In this context, earlier studies have demonstrated that the quality of health care — in the form of both treatment and the use of more aggressive measures to discourage poor health behaviors like smoking — plays a crucial role in further lowering death rates [24–26].

In future research, the usefulness of *ET* as a measure of differentials in survival progress and in advances in general development should be examined. Among the promising research settings for measuring these differentials are the countries of western and eastern Europe before and after the fall of the Iron Curtain in 1990, as well as eastern and western Germany before and after reunification [27, 28]. Similar exercises might be carried out by examining the *ET* of countries during the implementation of large policy reforms or economic crises. Moreover, *ET* might be useful for studying the burden of certain diseases in a population. Since *ET* allows us to quantify the delay in development due to a specific disease, it may, for example, be used to investigate the consequences of eradicating cancer [29].

Conclusion

In addition to introducing another tool for measuring progress and development, we were able to show in this paper that the assessment of the performance of a specific country is not straightforward. Rather, we illustrated that such assessments are sensitive to the selected indicator (e.g., life expectancy), perspective (gap, slope, lag), and reference (e.g., G7 countries). It is therefore important to use care in interpreting results that are based on a single measure, perspective, or reference. If possible, multiple factors should be taken into account for each of these dimensions. The concept of *equivalent time* represents a new (or rejuvenated) approach for studying advances in development. Using a single number, the measure reflects both the differences in development at a certain point in time and the pace of change over time. *ET* therefore offers a simple and intuitive way of expressing the delay in the development of a given population relative to the development of a reference population.

List of abbreviations
ET: equivalent time, LE: life expectancy, ICD: International Classification of Diseases

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Availability of data and materials
The data that support the findings of this study are available from the Human Mortality Database [www.mortality.org], National Center for Health Statistics [http://www.cdc.gov/nchs/data_access/vitalstatsonline.htm] and the World Bank [https://datacatalog.worldbank.org/dataset/world-development-indicators].

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Author's contributions
ME and FP conceived the study. ME conducted the analysis. ME, FP and RR prepared the manuscript. All authors contributed to drafting and critical revision.

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Figures

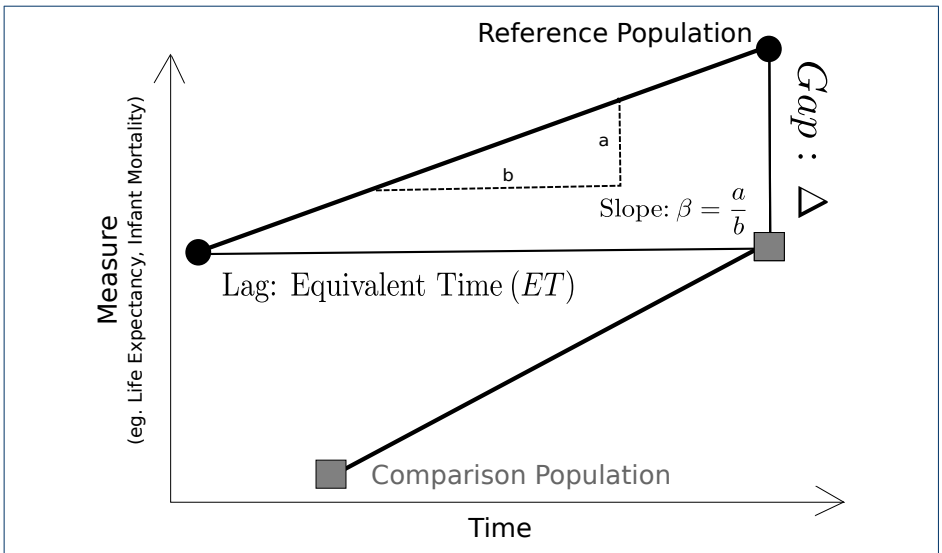

Figure 1 Schematic representation of measuring advancements in the decline of mortality.

Tables

Table 1 Classification of different kinds of references for the evaluation of survival progress. The listed examples are using the reference perspective in at least some part their empirical analysis.

Type	Example	Pitfall	Exemplary Study
Intra-group	$e_{0,\text{Country A}} - e^{-i}_{0,\text{Country A w/o health burden i}}$	peculiarity or commonality?	Aburto et al. [30]
Inter-group	$e_{0,\text{Country A}} - e_{0,\text{Country B}}$	prime example or exception?	Meslé and Vallin [31]
Supra-group	$e_{0,\text{Country A}} - e_{0,\text{Cross-country average}}$	common trend?	Bongaarts [32]

e_0 –Life Expectancy at Birth

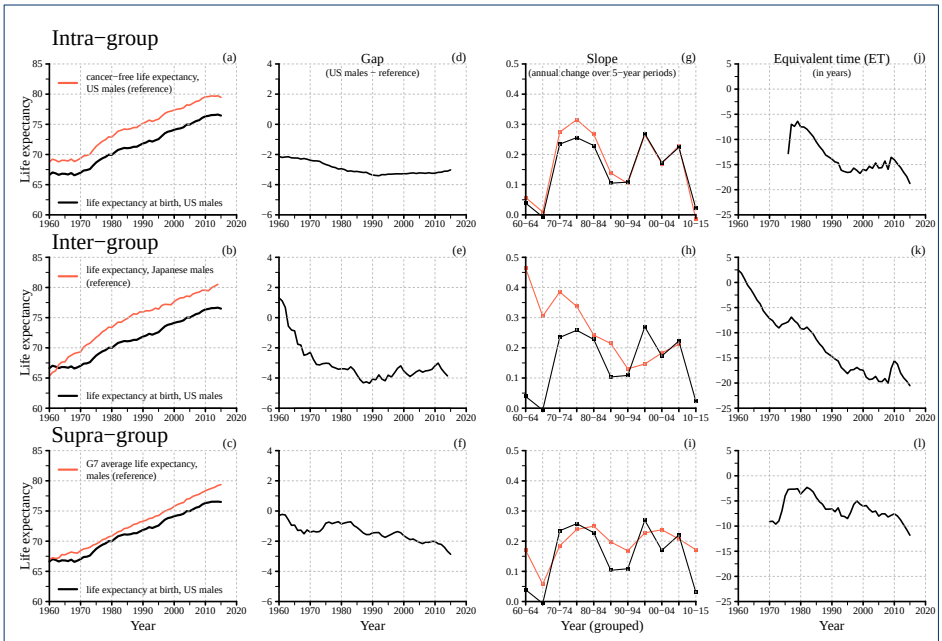

Figure 2 Life expectancy, gap, slope (annual change over five-year periods), and equivalent time of US life expectancy compared to that of three references, males, 1960-2015. All estimates are based on period life tables. The slopes indicate the annual change between the two years, comprising five-year intervals. The last group, 2010-15, comprises six years. The slope calculation is based on the assumption of a linear change between the two time points. Equivalent time is calculated using linear interpolation.

7.2. Paper II: How has the lower boundary of human mortality evolved and has it already stopped decreasing?

How has the lower boundary of human mortality evolved and has it already stopped decreasing?

Marcus Ebeling[1,2]

Abstract

In contrast to the upper boundary of mortality, the lower boundary has so far largely been neglected. Based on the three key features location (1), sex-specific difference (2) and level (3), this paper analyzes past and present trends in the lower boundary of human mortality. The analysis is based on cohort mortality data for 38 countries, covering all the cohorts born between 1900 and 1993. Minimum mortality is analyzed using observed as well as smoothed estimates. The results show that the ages at which minimum mortality is reached have shifted to lower ages. Although the differences have become almost negligible over time, males are showing higher levels of minimum mortality than females. The level of minimum mortality was halved more than five times over the analyzed time horizon. The results also suggest that even after more than one and a half centuries of mortality improvements, minimum mortality has not yet reached a lowest limit, and is likely to decrease further in the near future. Trends in the three key features also raise questions about the importance of evolutionary, social, and biological determinants for the recent and future development of minimum mortality.

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Introduction

Whereas the upper boundary of mortality (maximum mortality) is a topic that has previously attracted the attention of scholars (e.g., Gampe, 2010; Rau et al., 2017), the topic of the lower boundary (minimum mortality) has so far largely been neglected. In this paper, we aim to analyze past and current trends in the ages and levels of minimum mortality, as well as the sex-specific differences in minimum mortality.

The steady decline in mortality is one of the greatest success stories in human history (Oeppen and Vaupel, 2002; Riley, 2001). Mortality rates across almost all ages have been consistently declining to lower and lower levels for more than one and a half centuries.While the successive improvements have been unprecedented, the mortality trajectory has retained its key features (see Figure 1): For example, levels of infant mortality have been consistently similar to mortality levels between ages 60 to 80. Moreover, adult mortality has shown a log linear increase over age, and mortality levels between infancy and early adulthood have followed a U-shaped pattern.

As mortality declined in developed countries, the main improvements in life expectancy shifted from younger to higher ages (Christensen et al., 2009). Today, the contributions of the youngest ages to the increase of the average age at death are almost negligible. Nevertheless, in almost all countries of the world, mortality at younger ages has been decreasing continuously (Armour-Marshall et al., 2012; Verguet et al., 2014). Consequently, the U-shaped mortality trajectory between infancy and juvenile ages has become more and more pronounced over time (see Figure 1). Minimum mortality represents the inflection point of this pattern; i.e. it marks the lowest mortality level across ages, and the threshold between mortality decrease and increase over age.

[Figure 1 about here]

Together with the maximum level, minimum mortality embraces the range of age-

specific mortality. The maximum mortality level has been estimated at a level of around 7,000 deaths per 10,000 person-years lived, indicating the magnitude of the late-life mortality plateau (Gampe, 2010). Minimum mortality cannot be assessed to a specific level. Minimum mortality declined from around 30 per 10,000 to a level of less than one per 10,000 over the time horizon of 150 years (see Figure 1). The plasticity of the lower boundary compared to the stability of the upper boundary points to the most important difference between the two. Explanations for the stable upper boundary are diverse, and range from a more homogeneous population composition at those ages to physiological and genetic aging processes (Wachter, 1999; Pletcher and Curtsinger, 1998; Missov and Vaupel, 2015; Vaupel et al., 1998). Minimum mortality, by contrast, appears to (still) be alterable. Most importantly, improved living standards (e.g., better sanitation and nutrition) and medical breakthroughs, such as antibiotics and vaccinations, are the main reasons for the survival gains at childhood and juvenile ages (Cutler et al., 2006; Blum, 2009; Gore et al., 2011). The major causes of death at those ages have shifted away from infectious diseases and toward behavioral causes (Blum, 2009). Today, injuries and non-communicable diseases are the leading causes of death for people aged 5 to 14 in low-mortality countries (Patton et al., 2009).

After the degree of alterability, the location is the second major difference between minimum and maximum mortality. Maximum mortality is located at the end of the lifespan, whereas minimum mortality is reached within a tiny age range at the end of the first decade of life. It marks a specific point over the life course at which physical and social development levels are most favorable for withstanding the risk of death. The explanations of why mortality levels are lowest in this exact age range are mainly drawn from evolutionary mechanisms, such as high selection pressure prior to the beginning of the reproductive period; i.e. the proximity of these ages to the onset of sexual maturity (Burger et al., 2012; Chu et al., 2008; Levitis, 2011). However, for both males and females, scholars have observed over the past century temporal changes in related processes, such as faster body growth or the earlier onset of puberty (Frisch,

1978; Tanner, 1973; Goldstein, 2011; Schönbeck et al., 2012). The location of minimum mortality might be similarly affected by these shifts.

Male-female differences are the third key feature that could reveal further disparities between maximum and minimum mortality. The question of whether there are sex-specific differences in maximum mortality cannot be answered clearly. Gampe (2010) found no relevant differences between the male and female levels, whereas Rau et al. (2017) documented higher levels for males. Among children and adolescents, mortality is higher for males, despite the fact that the absolute gap becomes smaller with decreasing levels of mortality (Gissler et al., 2009). Surprisingly, external causes of death as an indicator for sex-specific risk-adverse behavior explain only a minor part of the male-female differences (Gissler et al., 2009). For example, Gissler et al. (2009) measured a higher rate of non-external causes for boys than for girls. It therefore appears that additional factors must be responsible for these differences.

An assessment of minimum mortality could have also implications for the development of methods and models in mortality research. For instance, mortality forecasting approaches usually assume that death rates have no lower limit. Instead, the logarithm of death rates is used, which allows rates to decline infinitely, while staying between zero and one (see, for instance, Hyndman and Ullah, 2007; Lee and Carter, 1992). A proof seems impossible, as the question of whether mortality rates can decline infinitely has yet to be answered. Examining trends in minimum mortality can help us better understand the dynamics at the lower edge of human mortality. Moreover, attempts were made in previous parametric mortality models, such as the Siler-model (Siler, 1983) or the Heligman-Pollard model (Heligman and Pollard, 1980), to include mortality at all ages; and thus the decreasing and increasing parts of age-specific mortality, which are connected by minimum mortality. Mortality modeling could benefit from an investigation of minimum mortality in, for instance, the refinement of existing models or the development of new models.

Based on these considerations, three essential key questions should be asked when seeking to identify the characteristics of the lower boundary of mortality. First, how pronounced are the sex differences in the lower boundary of age-specific mortality? Second, at which ages is minimum mortality located, and how have these ages changed over time? Third, how has the level of minimum mortality evolved, and is minimum mortality still decreasing? Given the unprecedented mortality improvements of the past, it is not far-fetched to hypothesize that minimum mortality might be the first mortality rate that finally hits a lower (natural) limit; and that therefore indicates the absolute frontiers of human mortality improvement.

Data

We have chosen a cohort perspective to estimate minimum mortality. Only a cohort follow-up ensures a life course perspective, and thus the clear identification of the lowest mortality rate over age. Because the mortality rates of several cohorts are combined in period perspective, period-based estimates could be distorted by peculiarities across cohorts, such as fluctuating birth cohort sizes or unexpected external shocks and changes, like political crises or medical breakthroughs. In addition, many forces shaping minimum mortality such as selection work in cohort direction. Hence, a cohort perspective allows to evaluate and discuss the trends in the light of these potential explanations and determinants. This is not readily possible in a period based comparison. Furthermore, a cohort perspective still covers a sufficient observation time and provides current values, since minimum mortality is located at an early stage of life.

We use cohort data from the Human Mortality Database (2017). Death counts are given by Lexis–triangles. Accordingly, the full death counts for one age are the sum of two consecutive triangles in cohort direction. The two triangles are spread over

two periods, and thus the end-year/start-year population between them is used as the exposures-to-risk (see the supplemental information for an illustration). For most countries, the majority of raw data for death counts in the Human Mortality Database are given by Lexis–triangles. Especially at the beginning of the 20th century, this number was growing. For other countries and periods with raw data of a different structure, a regression approach is applied to split the death counts within a Lexis–square into counts by a Lexis–triangle (see Wilmoth et al. (2007) for further details). All estimates are based on data for ages one to 20. The earliest cohort considered includes those born in 1900. For some countries, the data start later in time. The last cohort available also varies by country. Countries with data covering fewer than 20 cohorts are excluded from the analysis. (see the supplemental information for further details).

Methods

Although data from the Human Mortality Database cover national populations and can thus be considered complete enumerations, the data are still subject to stochastic variation (Udry et al., 1979; Kirkby and Currie, 2010; Klotz, 2016). For instance, Udry et al. (1979) have demonstrated that the smaller a population is, the more unstable the respective mortality rates are. In addition, it is usually assumed that the underlying mortality process is smooth (see also Kirkby and Currie, 2010). Minimum mortality could be considered to be especially vulnerable to population size and stochastic variation because of its low intensity. Hence, around the age of minimum mortality, mortality could be quite noisy. Minimum mortality contains another, rather technical and theoretical problem: namely, that mortality rates are bound at zero at the lower end. In low-mortality countries with a small population size, mortality at some ages below 20 is already at such low levels that the number of age intervals without any deaths is consistently increasing over time (see the supplemental information for further details). To deal with zero death rates and stochastic variation, we complement the analysis of observed trends with estimates based on a two-dimensional smooth-

ing approach. Although using other approaches is also conceivable, we have chosen a smoothing approach for count data developed by Camarda (2012), and applied it over age and cohort. The main reason for this choice is that the approach was developed to capture the data-generating process behind mortality, which is assumed to be Poisson (Brillinger, 1986). Therefore, the approach can also process intervals with zero death counts. Furthermore, the approach provides a sufficient fit to both mortality and the age of minimum mortality (see the supplemental information for further details on method evaluation and selection). However, model testing and evaluation were necessary to determine the best calibration for the problem at hand.

The approach by Camarda (2012) uses P-splines within a generalized linear array model. This or a similar approach is widely used in practice to smooth observed mortality rates (see, for instance, Colchero et al., 2016; Bohk-Ewald and Rau, 2017; Currie et al., 2004). The method relies on the assumption that death counts are Poisson-distributed over age and time. The Poisson assumption implies that the mean and the variance are equal. However, for most countries in the dataset, variance is considerably higher (or lower) than the mean, and the Poisson assumption is therefore violated (see the supplemental information for further details). This phenomenon is known as extra Poisson variation, or (under-) overdispersion (Breslow, 1984; Djeundje and Currie, 2011). Data may be overdispersed for a number of reasons. In many cases, population heterogeneity and unexpected events, such as period shocks, are responsible for the overdispersion. Since variance is higher than the mean, incorporating overdispersion is especially important when seeking to generate robust standard errors. The approach by Camarda (2012) can integrate this specific feature by allowing the variance to change proportional to the mean. Furthermore, because the Poisson assumption allows for the incorporation of age intervals without any deaths, the approach is able to utilize this information. The method is readily available via the package "MortalitySmooth" for the statistical programing language R (2016).

The occurrence of period shocks, such as the Spanish flu or a war, can lead to problematic estimates when two-dimensional smoothing approaches are applied, because in such situations the respective mortality rates are subject to more than just stochastic variation (Kirkby and Currie, 2010; Palloni, 1990). When the two dimensions are cohort and age, the situation becomes especially difficult because the period shocks are located on a backward 45-degree line in the age-cohort surface. Hence, in the model calibration, we investigated the influence of such ruptures by fitting them with and without information about ages, which are affected by period shocks. Due to the data structure, we excluded the 1915–1919 and 1938–1947 periods, which cover the Spanish flu and World Wars I and II. The P-spline approach we used interpolates the emerging gaps, which are generated when the mortality of the respective ages is ignored.

Based on the evaluation and model testing, the following settings were used to obtain smooth minimum mortality estimates (see the supplemental information for further details). Irrespective of the impact in the specific country, we excluded war years to ensure a consistent analysis framework. To avoid potential distortions, we restricted the range of ages to be searched for the minimum mortality to the ages five to 15; which is theoretically reasonable given the range of observed ages. We also decided to control for overdispersion across all countries to provide consistency. Even after these restrictions were applied, overdispersion or underdispersion was found in almost all of the countries (see the supplemental information for further details).

Results

Level of minimum mortality: Figure 2 shows the level of minimum mortality for males and females in France. The supplemental information contain the minimum mortality trajectories for all other countries. The graph depicts the level of minimum mortality per 100,000 person-years lived. Levels are shown on a log-scale, using the

logarithm of two to emphasize level halving. Accordingly, the horizontal contour lines mark the consecutive halving of the mortality level. The solid lines are minimum mortality estimates based on the age-cohort smoothing and the colored area around this line indicates the 95% confidence interval. The plus signs indicate observed minimum mortality. The squares mark the observed minimum mortality for cohorts who spent at least one year in the omitted periods (1915–1919, 1938–1947). Females are colored in red and males in blue.

[Figure 2 about here]

In Figure 2, we can see that minimum mortality declined from almost 250 deaths per 100,000 person-years lived to around 10 deaths per 100,000 person-years lived over the cohorts born between 1900 and 1993. Over the course of this decline, the trajectories for French males and females show four periods with distinct developments (see tags in Figure 2). Minimum mortality improves only slowly in the first period (I), which encompasses the cohorts born between 1900 and 1920. The second period (II) is characterized by rapid improvements. This period spans the cohorts born between 1920 and 1950. In the third period (III), minimum mortality improvements decelerated, and were close to stagnation at certain points. This trend lasts up to the cohorts born in the early 1960s. The fourth period (IV), which runs until the most recent cohorts, again shows steady improvements. The trajectory of France is exemplary of the trajectories observed in the majority of countries analyzed. Variation can only be found in small temporal differences and in the manifestation of patterns in the four periods described. Only the minimum mortality levels for a few countries, such as those of Russia or Belarus, deviate from the general trend. In these countries, the improvements in minimum mortality over the analyzed cohorts are marginal to non-existent.

Figure 3 shows the minimum mortality levels for three recent cohorts (1970, 1980, and 1990) for all of the countries observed. The countries are ordered based on the

minimum mortality level of the 1990 cohort, with the lowest to the highest values being displayed from left to right. To identify the significance of differences in the most recent estimates (1990), minimum mortality for the 1990 cohort is depicted with the corresponding 95% confidence interval, which is illustrated by the bar around the respective median estimate. Note that the axes for males and females have different ranges.

[Figure 3 about here]

Japan currently has the lowest minimum mortality levels at around eight deaths for females and 10 deaths for males per 100,000 person–years lived. For the females of the 1990 birth cohort, the three countries with the lowest minimum mortality levels are Japan, Sweden, and Northern Ireland; while for the males of 1990 birth cohort, the three countries with the lowest minimum mortality levels are Japan, Austria, and Sweden. Note that the estimates for Luxembourg and Iceland rest on data with many zero death rates, and should therefore be interpreted with care. The levels among the best-practice countries are very close to each other, which is reflected in the black solid trend and the overlapping confidence intervals. This overlapping, which applies to almost half of the countries, suggests that the differences are marginal, and are not statistically significant. Therefore, efforts to rank or select clear leaders would be inappropriate. However, among the countries with the highest minimum mortality levels, clear distinctions can be made. Russian males and females have by far the highest levels. Males and females in other Eastern European and Baltic countries, as well as in Portugal, also have relatively high minimum mortality levels. Among the G7 or the countries with the greatest economic power, no uniform pattern can be observed. These countries are spread over the remaining rankings above the group of the worst performing countries.

The finding that mortality has declined continuously across almost all countries, and especially among the most recent cohorts, supports the assumption that minimum

mortality for both males and females is likely to decrease in the near future. All of the countries studied showed at least a marginal decline in mortality over time. Moreover, in the majority of these countries, a stable pattern of improvement could be observed in the cohorts born since the 1970s; and this trend may be expected to continue.

Age of minimum mortality: The results for the minimum mortality ages can be seen in Figure 4. The observed mortality rates as well as the smoothed mortality rates are given by age. Hence, the ages of minimum mortality are also measured in integers. Due to stochastic fluctuations and the impact of the world wars, minimum mortality ages based on the smoothed mortality estimates are considered as the basis. Furthermore, the trends across countries are very homogeneous, and show only small variations. Therefore, we pooled the ages of minimum mortality across countries and summarized the results for 10 consecutive cohort groups. Due to the varying length of the data, the number of ages in each of these 10 groups differs.

[Figure 4 about here]

The age of minimum mortality decreased over the cohorts studied. This trend is also visible if we look at the ages based on the observed mortality estimates. For the cohorts up to the cohorts born in 1920–1929, the modal value jumps between ages 11 and 12. The distribution of males and females for the 1930–1939 and 1940–1949 cohorts are right–skewed, which means that ages higher than the mode are observed more frequently than younger ages. This is, however, likely an effect of World War II. Although minimum mortality among the cohorts born in 1950–1959 or later has been relatively consistently located at age 11, the distribution is shifting toward younger ages. The shift is indicated quite well by the growing frequency of age 10 as the age of minimum mortality over the respective groups. Accordingly, for the last two cohort groups, the modal value is already located at age 10, and ages above and below the mode are almost evenly observed. However, the growing frequency of age nine over the last two

cohort groups could suggest that the shift toward younger ages might be continuing.

In addition to the location of minimum mortality, we also analyzed the form of the U-shape pattern, using the ratio of death rates at ages adjacent to the age of minimum mortality (see the supplemental information for more details). However, there is no clear trend across cohorts, countries or sexes. For some countries, the U-shape remained almost constant, whereas in other countries considerable difference are visible. Therefore, the location of minimum mortality is much more clear in some countries than in others.

Sex–specific differences: The sex-specific differences in the ages of minimum mortality are declining over the cohort groups. For the first three cohort groups, the frequency of the modal value is more pronounced for females than for males. However, especially for the post-war cohorts, the distributions for both males and females are almost similar.

[Figure 5 about here]

Figure 5 shows the absolute male–female differences in the levels of minimum mortality for all of the countries analyzed. The trajectories of Japan, Russia, France and Norway are highlighted. A negative difference expresses higher mortality for females and vice versa.

The development of sex–specific differences over time shows two distinct patterns: higher female mortality with a consistently growing male disadvantage followed by a trend toward convergence. In all of the countries observed from the 1900 birth cohort onward, the female minimum mortality levels were higher in the first 10 to 20 cohorts; albeit with vastly differing magnitudes. For instance, in the 1900 birth cohort, the minimum mortality level of females was around 25 deaths per 100,000 person-years

higher than that of males in France, whereas the female minimum mortality level was only around seven deaths higher than that of males in Norway. However, the starting level is also the point at which the female disadvantage was the greatest. The gender gap decreased steadily, with males and females reaching similar levels of minimum mortality somewhere between the 1905 and the 1920 birth cohorts. Thereafter, these varying developments continued and the sex-specific differences widened once again. The gender gap in minimum mortality rates peaked among the 1940 birth cohort. For example, French males of the cohorts born between 1935 and 1940 had minimum mortality levels that were around 15 deaths per 100,000 person-years higher than the minimum mortality levels of their female peers. The growing male disadvantage was mainly driven by the rapid improvements in minimum mortality among females in the cohorts born between 1920 and 1940. After the male disadvantage reached its maximum level, the levels converged steadily in almost all of the countries. Russia has, however, been an exception to this overall pattern. Among the most recent Russian cohorts analyzed, men have a minimum level that is still around 20 deaths per 100,000 person-years higher than that of females; whereas in, for example, Japan, France, and Norway, the absolute male-female differences are almost negligible.

Discussion and Conclusion

Whereas the upper boundary of mortality is a topic that has previously attracted the attention of scholars, the issues surrounding the development and characteristics of the lower boundary have so far been neglected. The lower boundary, hereafter also called minimum mortality, is the lowest mortality rate across ages. It is located at the bottom of the U-shaped pattern, which describes the trajectory of childhood and early adolescent mortality. This paper addresses the past and the present developments of three key features of minimum mortality: level, location, and sex-specific differences.

Our analysis rests on cohort mortality data. The lengths of the available time series vary by country, with a maximum range covering the cohorts born between 1900 and 1993. In addition to using observed trends, we complemented the analysis with smoothed mortality estimates using a two-dimensional P-spline approach over age and cohort (Camarda, 2012). This approach was selected after it was compared with a locally weighted regression, tested, and assessed under different model specifications (see the supplemental information for more details). To prevent the distortion of the minimum mortality estimates by period shocks, we excluded the data for the ages that were affected by World Wars I and II.

Although we removed the war years, a remaining effect of the two world wars cannot be ruled out completely. The temporary acceleration in the decline of minimum mortality for the 1920-1950 cohorts in the smoothed as well as in the observed estimates is visible in almost all of the countries, but the pattern is especially pronounced in countries such as Italy, France or England and Wales, which experienced war on their territories. Those estimates probably do not reflect the real minimum mortality, and are additionally influenced by post-war improvements. Thus, the estimates might not reflect the minimum mortality levels that would have been observed in the absence of war. A further problem for small populations with decreasing mortality is the emergence of age intervals without any deaths. Although the P-spline approach is able to handle this phenomenon, the estimates for Iceland and Luxembourg in particular are not reliable, and should be interpreted with care. The number of such intervals within one cohort increases constantly over time, and reaches problematic levels. For example, the Icelandic cohorts born between 1975 and 1992 contain an average of 11 such intervals for males and 12 such intervals for females between ages one and 20. Given their sudden appearance, the zeroes in Iceland might even be subject to data problems.

The *level of minimum mortality* decreased continuously over the observed time period. Furthermore, the pace of improvement among recent cohorts has been rela-

tively steady. Therefore, recent trends do not suggest an imminent end to this decline. Among the best-practice countries, the current levels are about eight deaths per 100,000 person-years for females and about 10 deaths per 100,000 person-years for males. However, the lower boundary of mortality has not been decreasing constantly. Especially among the post-war cohorts, improvements slowed temporarily, with some countries even experiencing stagnating or slightly increasing levels. The reasons of why minimum mortality stagnated and declined again are open to speculation. The introduction of mass immunization for several diseases in the 1960s could hint at potential explanations (Riley, 2001). Other factors such as the changing fertility-related behavior at that time (Billari and Kohler, 2004) or changes in the child care (Vandell et al., 2010) might also serve as potential explanations. Given that the current levels are unprecedented, it is even more intriguing that the lower boundary of mortality has still not reached a lowest limit after almost 170 years of continuous mortality improvements. The question of whether minimum mortality has a lower limit remains open and continues to present challenges. If such a limit does exist, mortality might follow a constant force of mortality, whereby simply good or bad luck are the essential mortality determinants. However, recent developments in childhood health challenge such optimistic and futuristic thoughts. For instance, Brüne and Hochberg (2013) found that chronic diseases in childhood such as obesity, diabetes or autoimmune diseases have been increasing, especially since the late 1980s. They argued that changes in the environment favor this development; speculating that evolutionary and medical factors — such as thrifty genes, hygiene, fetal programming, or the extensive intake of cow's milk — might explain this trend. Although rising prevalence of chronic diseases in childhood might not have an immediate effect on death, they could potentially increase the vulnerability, and thus, have an indirect effect on the level of minimum mortality. However, based on our results, we cannot confirm any negative influence of such a development on the improvement of minimum mortality up to the most recent cohorts.

The *minimum mortality age* shifted toward lower ages. For the oldest birth cohorts

analyzed, minimum mortality was mainly located at age 12. Over time, however, the modal value shifted toward lower ages, and the distribution around this modal value moved to lower ages. In the most recent cohorts, the lower boundary of age-specific mortality is located at age 10. The location of the lowest mortality across ages is closely associated with the onset of sexual maturity. Evolutionary theories of aging argue that evolutionary fitness, defined as the intrinsic rate of natural increase, is most sensitive to mortality changes around the age of sexual maturity (Hamilton, 1966). Accordingly, selection pressure on age-specific mortality should be highest around the onset of the reproductive period, when the respective mortality rates are pushed to their lowest possible levels. Similarly, the age of maturity itself should be under strong selection pressure as a key age that defines the onset of reproduction. Other authors have also argued that intergenerational transfers, such as parental care, shape selection pressure and are an important determinant of the shape of human life history, and thus of, mortality (Gurven et al., 2012). Consequently, the investments of older generations in younger generations lead to a concave shape of selection pressure, which may push mortality down even further at the onset of the reproductive period, when such investments start to pay off (Chu et al., 2008; Lee, 2003; Bogin, 1997). As a result of the close relationship between the location of minimum mortality and sexual maturity, it could be hypothesized that the earlier occurrence of minimum mortality might be related to temporal changes in different aspects of childhood development such body growth, and perhaps as a consequence, the earlier occurrence of puberty and sexual maturity, which has been reported by a number of authors (Goldstein, 2011; Tanner, 1973; Frisch, 1978; Schönbeck et al., 2012). However, the similar locations of minimum mortality of males and females could be seen as a sign against this idea because different studies provide evidence for a slightly earlier onset of the puberty of females, which also holds for the transition through the different development stages of sexual maturity (Susman et al., 2010; Lee, 1980). In addition to these factors, Levitis and Martínez (2013) have offered further hypothesis for why juvenile mortality is U-shaped, and for why we therefore see an inflection point between the decreasing and the increasing

parts of mortality over age. However, the plasticity of minimum mortality challenges all of these concepts. Given the enormous gains that have been made over a short period of time, it is possible to speculate about whether human progress has decoupled minimum mortality from evolutionary mechanisms — or has, at least, weakened the relationship between them.

The development of absolute *sex-specific differences* in the lower boundary of mortality shows two distinct patterns: higher female mortality with a consistently growing male disadvantage, followed by a trend toward convergence. In the oldest cohort analyzed, females had higher minimum mortality levels. Tuberculosis could be one potential explanation for this female excess mortality. Different authors document higher tuberculosis death rates of females in the respective age range (10-14) for similar cohorts as well as for calendar years, in which the respective cohorts reached their minimum mortality (Frost, 1995; Springett, 1952b,a). Further reason such as discrimination related to sex or birth order (Modin, 2002) are conceivable but remain as vague speculations. The female disadvantage turned relatively rapidly into a male disadvantage, which reached its maximum level at some point among the interwar cohorts. Since then, a continuous trend toward convergence can be observed. The male disadvantage is the result of slower improvements in the minimum mortality levels of males than of females. These pace differentials are striking because the usual determinants of sex-specific differences, such as lifestyle and behavioral factors, should be less relevant at these ages. We can speculate that excess mortality, caused by environmental conditions is decreasing more quickly among females than among males. Furthermore, it is possible that after this type of excess mortality is no longer relevant the gender gap will be primarily driven by biological factors. Currently, communicable diseases can be excluded as a potential driver and external causes of death explain only a small part of the gender gap (Gissler et al., 2009). Accordingly, non-behavioral causes and non-communicable diseases could be emerging as the potential drivers. Studies that used cancer — a leading cause of death in childhood in the developed countries —

to illustrate this development found that boys are more likely than girls to develop a childhood cancer (Kaatsch, 2010; Dorak and Karpuzoglu, 2012).

It is clear that at the minimum mortality age, the physiological and social constitutions of humans are most capable of withstanding death. Following Belsky et al. (2015), we argue that aging research should focus on the age groups that are still in the very early stages of the aging process. In their study, they focused on individuals aged 26–38. Minimum mortality as such marks the inflection point between decreasing and increasing mortality, and could thus be interpreted as the point at which aging begins. Hence, investigating the aging process starting from the age of minimum mortality might prove to be even more intriguing. We could then ask the following questions: Are there some lessons we can learn from the findings on minimum mortality about how aging and mortality might be postponed to or altered at later ages? Are we able to extend minimum mortality levels to the point of developing a second mortality plateau at younger ages? Or does minimum mortality reflect the baseline level of mortality if humans did not age? These are all non-trivial questions for which there may be no clear answers. However, they all invite further investigation into the lower boundary of human mortality.

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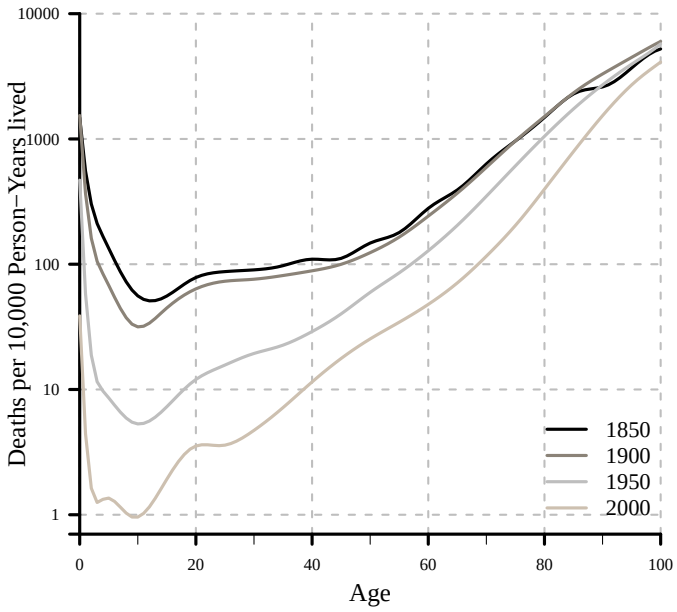

Figure 1. Age-specific mortality, France, females, years 1850,1900, 1950, and 2000. *Mortality rates are smoothed. Source: Own illustration using age-specific death counts and exposures-to-risk from the Human Mortality Database (2017).*

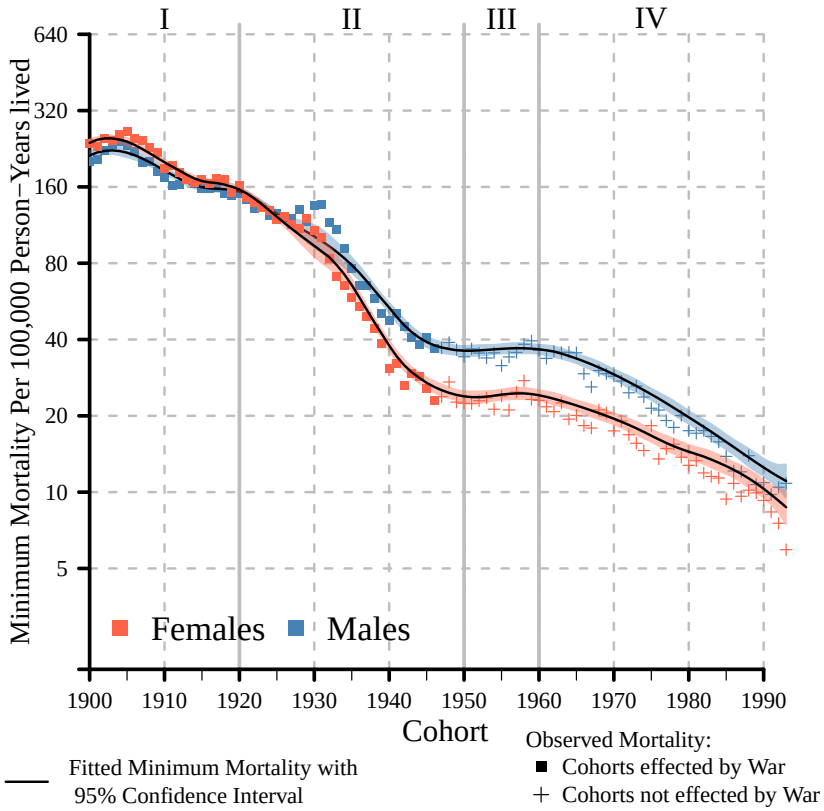

Figure 2. Minimum mortality, France, females and males, birth cohorts 1900–1993. *The graph depicts the observed (squares and crosses) as well as the smoothed (solid line) minimum mortality. The observed rates marked with a square indicate the cohorts who spent at least one year in the omitted periods (World Wars I and II). The colored area around the smoothed minimum mortality estimates depict the 95% confidence interval as calculated by the approach of Camarda (2012). Minimum mortality rates are illustrated using the logarithm with basis two. The solid gray grid lines and the Roman numerals mark the different periods of development. Source: Own illustration using age- and cohort-specific death counts and exposure-to-risk from the Human Mortality Database (2017).*

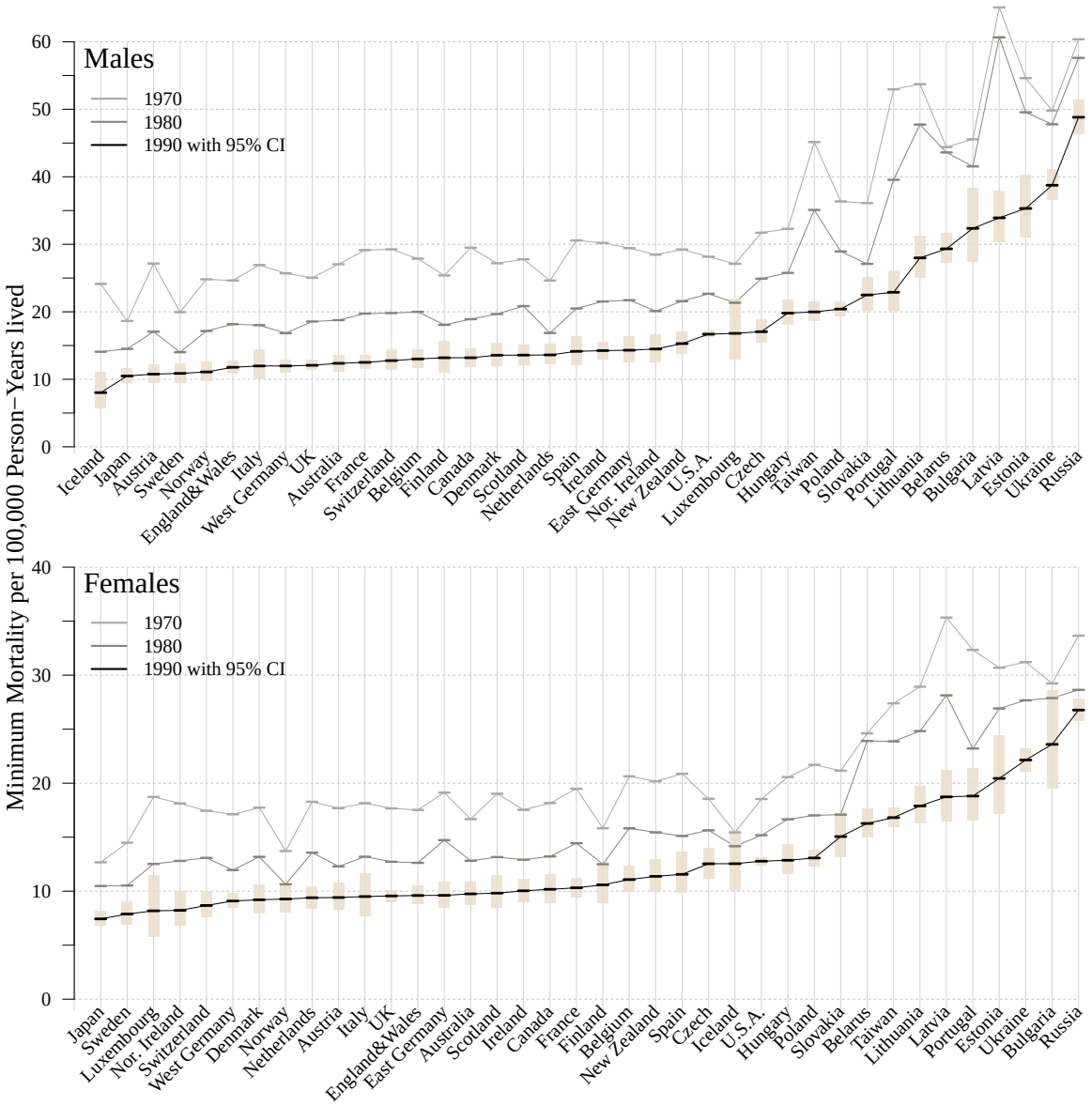

Figure 3. Minimum mortality, females and males, birth cohorts 1970, 1980 and 1990. *The graph depicts the levels of minimum mortality for the 1970, 1980 and 1990 birth cohorts for all countries analyzed. Note that for Bulgaria, the last available cohort was born in 1989. Countries are ordered according to the minimum mortality level for the cohort born in 1990, with the lowest to the highest levels displayed from left to right. The bars around the median estimate of the 1990 birth cohort depict the 95% confidence interval, as calculated by the approach of Camarda (2012). Note that the vertical axis has different ranges for males and females. Source: Own illustration using age- and cohort-specific death counts and exposure-to-risk from the Human Mortality Database (2017).*

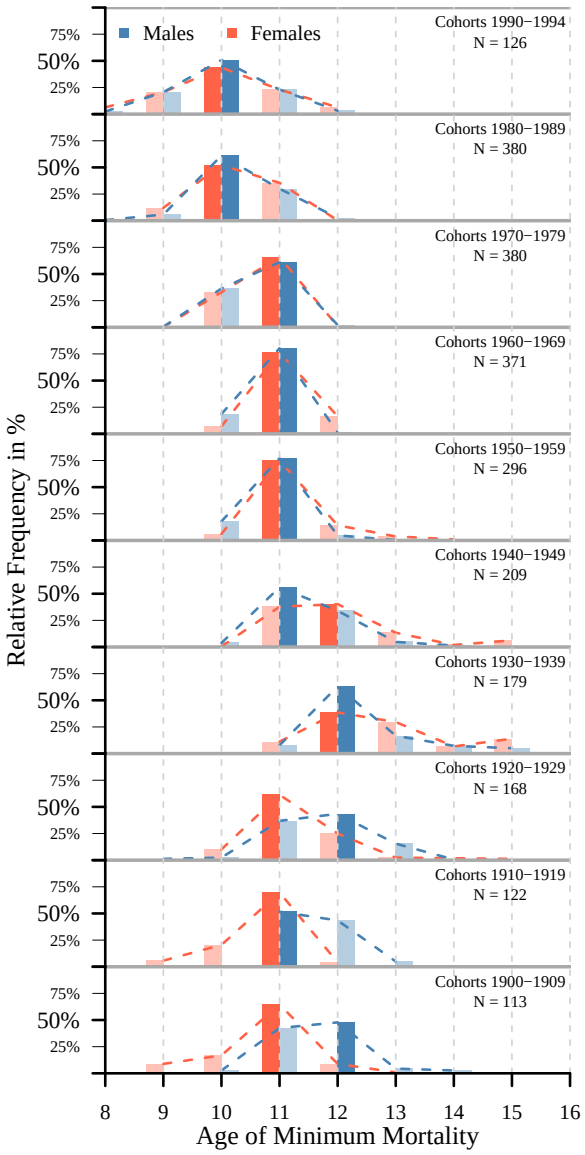

Figure 4. Distribution of minimum mortality ages, all countries together, females and males, grouped birth cohorts 1900-1994. *The bars show the relative frequency of the ages in the respective cohort groups; pooled over all of the available countries. The age of minimum mortality is measured in integers. The number of countries varies over time in each cohort group and is indicated by N. The highlighted bar represents the modal age in the respective year. The bars correspond to full ages. Source: Own illustration using age- and cohort-specific death counts and exposure-to-risk from the Human Mortality Database (2017).*

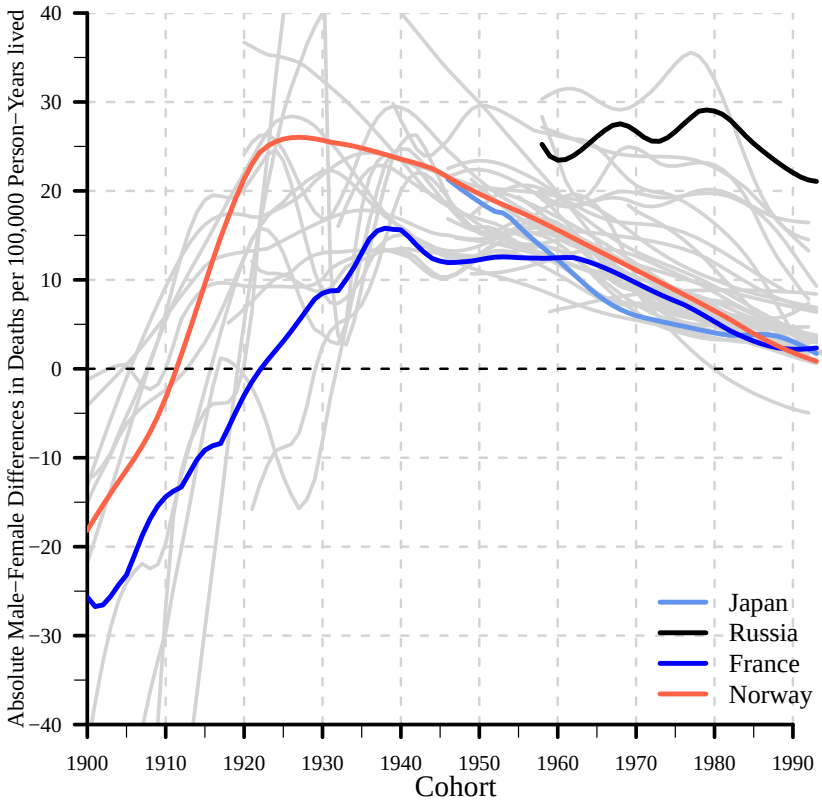

Figure 5. Absolute male–female minimum mortality differences, Japan, Russia, France and Norway, birth cohorts 1900-1994. *The gray lines depict all of the other countries included in the analysis. The sex–differences are calculated based on the smoothed minimum mortality estimates. Source: Own illustration using age- and cohort-specific death counts and exposure-to-risk from the Human Mortality Database (2017).*

Supplemental Information:

How has the lower boundary of human mortality evolved and has it already stopped decreasing?

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1 Country-specific details

Table 1. Overview of the country-specific details. *An overdispersion parameter close to one indicates that there is no overdispersion. A parameter (smaller) bigger than one indicates (under-) overdispersion. Zero death depicts the number of age intervals without any death over the entire cohort range. Data: Human Mortality Database (2017)*

		Males			**Females**		
		Overdispersion Parameter			Overdispersion Parameter		
Country	Cohort Range	w Shocks	w/o Shocks	zero Death	w Shocks	w/o Shocks	zero Death
Australia	1920–1990	1.1096	0.9590	0	1.0598	0.8942	2
Austria	1946–1993	1.0959	1.0959	0	0.9977	0.9946	1
Belgium	1918–1994	2.2458	1.0370	0	1.6513	1.1506	1
Bulgaria	1946–1989	1.7444	1.7356	0	1.7718	1.7626	0
Belarus	1958–1993	1.0168	1.0168	0	0.7194	0.7194	0
Canada	1920–1990	1.5618	1.2300	0	1.4573	1.1523	0
Switzerland	1900–1993	1.6633	1.0885	0	1.7624	1.0983	4
Czech	1949–1993	1.1055	1.1055	0	1.0871	1.0871	0
East Germany	1955–1992	1.4714	1.4714	0	1.2217	1.2217	0
West Germany	1955–1992	1.8816	1.8816	0	1.3631	1.3631	0
Denmark	1900–1993	1.4443	0.9926	1	1.3514	0.9759	2
Spain	1907–1993	10.8917	4.8185	0	9.1480	3.8431	0
Estonia	1958–1992	0.8162	0.8162	4	0.8716	0.8716	19
Finland	1900–1994	5.1371	1.3763	3	2.9506	1.2812	3
France	1900–1993	18.8175	2.0831	0	11.8500	1.7096	0
Northern Ireland	1921–1992	0.5730	0.5031	1	0.5735	0.5108	5
United Kingdom	1921–1992	2.5081	1.3913	0	2.1009	1.0925	0
Scotland	1900–1992	1.1885	0.6947	0	1.3090	0.6730	0
England and Wales	1900–1992	13.8741	2.1381	0	7.5534	1.9456	0
Hungary	1949–1993	1.2149	1.2149	0	1.1390	1.1390	0
Ireland	1949–1993	0.5220	0.5220	0	0.6297	0.6297	0
Iceland	1900–1992	0.5678	0.5226	232	0.4639	0.4214	299
Italy	1900–1991	41.6297	4.7319	0	37.7099	4.4166	0
Japan	1946–1993	4.9041	3.9621	0	3.1759	2.1095	0
Lithuania	1958–1992	0.7205	0.7205	0	0.5350	0.5350	0
Luxembourg	1959–1993	0.6319	0.6319	113	0.6211	0.6211	208
Latvia	1958–1992	0.6645	0.6645	0	0.6046	0.6046	0
Netherlands	1900–1991	6.0515	1.0800	0	5.2064	0.9995	0
Norway	1900–1993	1.1325	0.7643	2	1.0105	0.6839	6
New Zealand	1947–1992	0.8661	0.8661	0	0.9191	0.9191	2
Poland	1957–1993	1.3319	1.3319	0	1.0967	1.0967	0
Portugal	1939–1991	1.3432	1.2131	0	1.2641	1.0713	0
Russia	1958–1993	6.2416	6.2416	0	1.8984	1.8984	0
Slovakia	1949–1993	1.0929	1.0929	0	1.1056	1.1056	1
Sweden	1900–1993	2.5450	1.2409	0	2.5207	1.1638	1
Taiwan	1969–1993	1.5302	1.5302	0	0.7381	0.7381	0
Ukraine	1958–1992	2.3891	2.3891	0	1.0001	1.0001	0
U.S.A.	1932–1993	2.7886	2.3596	0	2.0869	1.7153	0

2 Illustration of death rate construction

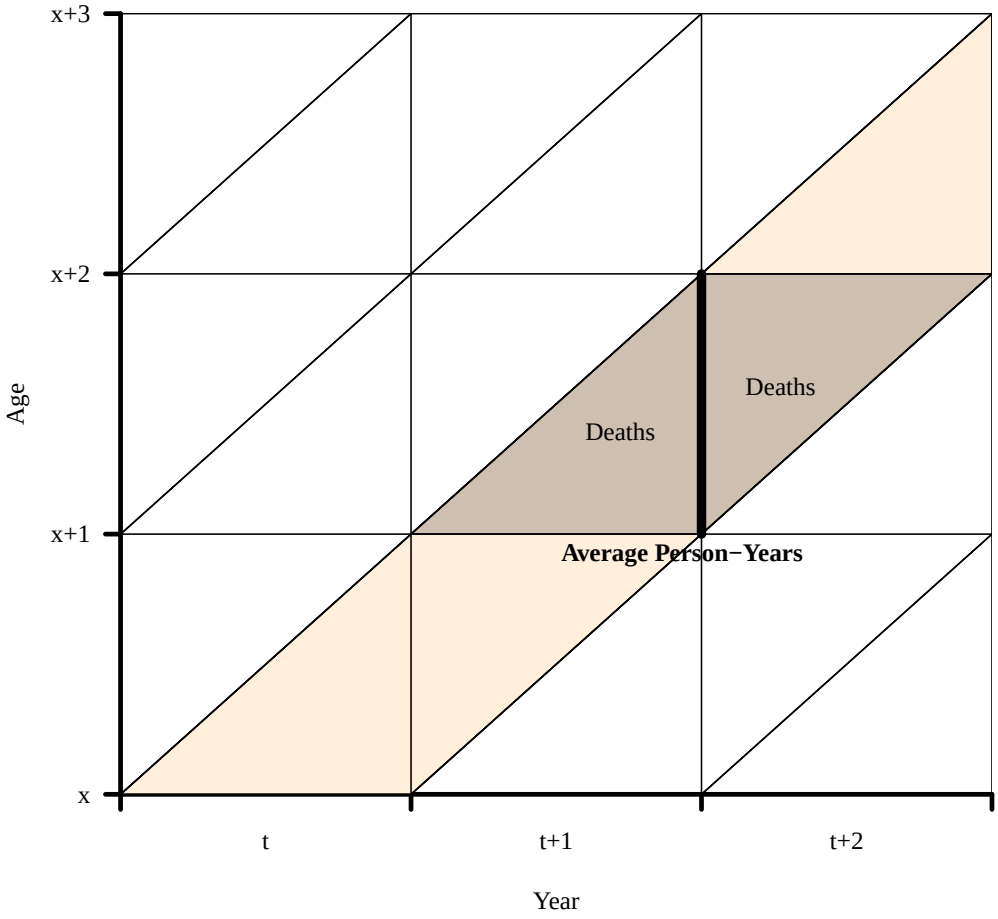

Figure 1. Illustration of the death rates used for the analysis.

3 Method evaluation

To identify an appropriate method, we compared a smoothing approach for count data developed by Camarda (2012) and a local regression model (LOESS) (Cleveland and Devlin, 1988; Chambers and Hastie, 1991), both applied over age and cohort. The use of other approaches, such as GAMs with an appropriate specification of the smoothing function and the distribution of the response (see Wood (2006)), is, of course, also conceivable (Kafadar, 1994; Currie et al., 2004). However, it is beyond the scope of the paper to either develop an appropriate approach or to provide an exhaustive method comparison of several approaches. Thus, availability, practicability, and accuracy are paramount considerations.

Note that the approach by Camarda (2012) has been described in more detail in the main text.

In the LOESS, logarithmic mortality rates over age and cohort are required as an input. Cohort and age are then used as the predictors in the model. To obtain the fit of a specific point, a weighted least squares regression model is fitted by minimizing for a set of observations within a pre-specified span around the specific observation. Weights are distributed so that observations closer to the central point have a higher weight than observation with a larger distance. Accordingly, a small span results in a high degree of adaptation to the data and a big span in a rough fit (for more details, see Cleveland and Devlin (1988) or Chambers and Hastie (1991)). To get the fit of a new point, a new regression model for a new set of respective observations and weights must be fitted. We determined the optimal span by using the value that minimizes the Akaike information criterion (AIC).

Although this approach has advantages over other two-dimensional smoothers (Kafadar, 1994), the LOESS rests on linear regression. It can also be applied using non-linear regression with additional quadratic terms for the two predictors. But consider-

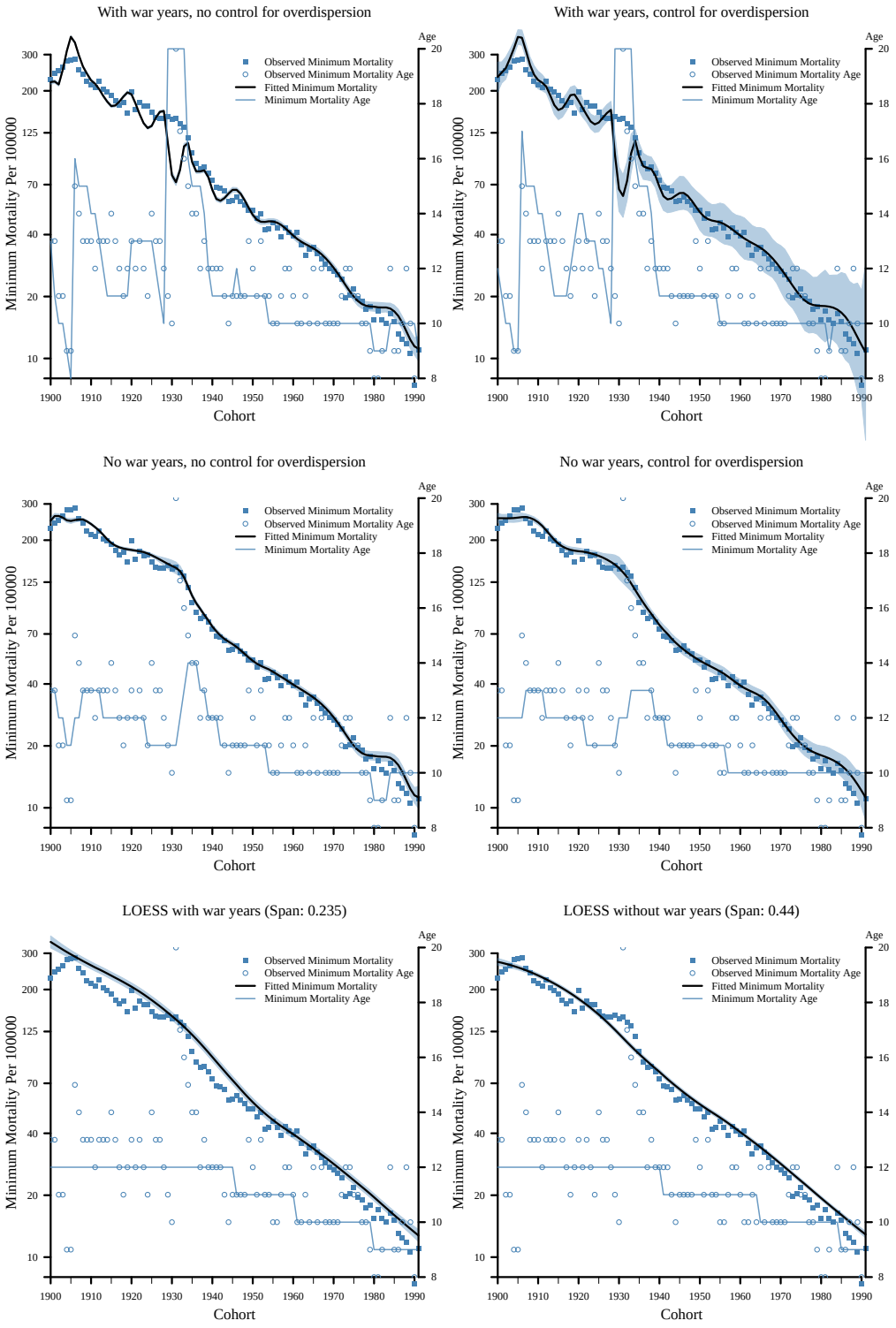

Figure 2. Minimum mortality and minimum Mortality Age estimation under different model specifications, Italy, males, cohorts 1900–1991. Data: Human Mortality Database (2017)

ing the results of Kafadar (1994), the linear LOESS seems to outperform the quadratic version. However, the use of linear regression to fit age-specific mortality has been criticized for several reasons (Pletcher, 1999; Promislow et al., 1999). First, the technique does not capture the data-generating process, and is thus theoretically wrong. Accordingly, in the LOESS, errors are assumed to be normally distributed with a constant variance (Cleveland and Devlin, 1988), which is a problematic assumption for age-specific mortality rates. An additional drawback resulting from the application of linear regression is that the logarithm of death rates is required as a response to keep the rates within the range of zero and one. For this reason, the LOESS –in opposite to the approach by Camarda (2012)– is not able to include zero mortality rates as information in the model fit. The respective death rates have to be considered as missing. These rates are then interpolated by borrowing information from neighboring cohorts and ages. All of these disadvantages are reasons to reject the LOESS as a potential method, but we still chose to test it as an alternative to the P-spline approach because of its great practicability.

As was mentioned in the main text, we excluded the 1915–1919 and 1938–1947 periods, which cover the Spanish flu and World Wars I and II. Both the LOESS and the P-spline approach interpolate the emerging gaps, which are generated when the mortality levels of the respective ages are ignored.

We chose Italian males as a comparison basis because even after omitting the war years, they were affected by the two World Wars and had a large amount of overdispersion, as indicated by their overdispersion parameter (see Table 1 in the main article). We complemented this assessment by evaluating Swedish males, a small population among whom the influence of period shocks was weak. The results for Swedish males can be found in Figure 3.

Figure 2 presents the results of the different models and the respective model spec-

Table 2. Model accurracy, residual standard error (RSE), males, Italy, ages 1–20, cohorts 1900–1991. The RSE is based on the data used to fit the model. RSE in brackets for models with war years is based on comparing only non-war years. Data: Human Mortality Database (2017)

Model	War Year	Control Overdispersion	RSE
P-spline	Yes	No	0.1434 (0.1172)
P-spline	Yes	Yes	0.1462 (0.1197)
P-spline	No	No	0.0866
P-spline	No	Yes	0.0900
LOESS	Yes	–	0.2589 (0.1651)
LOESS	No	–	0.1287

ifications for Italian males. The two upper panels show the results of the P-spline approach, including the war years, with (left) and without (right) accounting for overdispersion. The two middle panels depict the outcomes of the P-splines approach excluding the wars years, with (left) and without (right) accounting for overdispersion. The two lower panels illustrate the outcomes of the LOESS with (left) and without (right) the war years. In addition to the observed minimum rates (squares), the estimated minimum rate (black solid line) with the 95% confidence intervals (colored area) and the estimated ages of minimum mortality are shown (solid blue line). Observed ages are depicted with circles. The left axis depicts mortality per 100,000 person-years lived on a log scale. The right axis depicts the minimum mortality age. In addition to the visual inspection, we calculated the residual standard error (RSE) for all ages and cohorts considered in the model fit. Table 2 shows the values for all six models. The RSE in brackets is based on comparing only fitted and observed values of non-war years for approaches that include the war years in the model fit.

The RSE across the considered ages and cohorts is lower when the war years are

omitted. Hence, for both the LOESS and the P-spline approach, the fit of the models is increased. In comparison, however, the P-spline approach shows a higher degree of accuracy, which is expressed by a lower RSE. The minimum mortality trajectories of both approaches are affected differently by the war years. The LOESS consistently overestimates the level of minimum mortality up to the first post-war cohorts, whereas the P-spline approach even reveals implausible spikes up to that point. In general, the minimum mortality estimates of the LOESS are rougher than those of the P-spline approach. For Swedish males and for both approaches, the smoothed minimum mortality estimates are not as close to the observed rates as they are for Italian males; and they are generally above the observed minimum. These more conservative estimates are acceptable for countries with a small population size because, due to higher fluctuations, the observed minimum mortality rates could represent more outliers than in countries with a bigger population size.

The exclusion of the war years provides a more stable trajectory for the minimum mortality age. In general, the observed ages fluctuate considerably, but they change even more for the cohorts affected by war. The impact of war is especially visible for the 1931 cohort, who had a minimum mortality age of 20; which is six years after the end of WW II (see also Figure 4). Given the age trajectories of the adjacent cohorts, it is highly unlikely that age 20 would have been the minimum mortality age for this cohort in the absence of war. Only the LOESS and the spline approach without the war years are able to provide such stable minimum mortality age patterns over time.

Allowing for overdispersion in the P-spline approach has only a small effect on the accuracy of the model, but a big impact on the standard errors. Regarding the median estimates, Camarda (2012) stated that by controlling for overdispersion, the trajectories are smoother. This is visible in the slightly higher RSE for both models with overdispersion. However, the standard errors of minimum mortality for the most recent cohorts in particular increase when the relationship between the mean and the

variance is relaxed. The confidence intervals in the LOESS approaches are generally narrower. For both approaches, it should be noted that in the figures, the confidence bounds seem smaller because of the logarithmic axis for mortality rates.

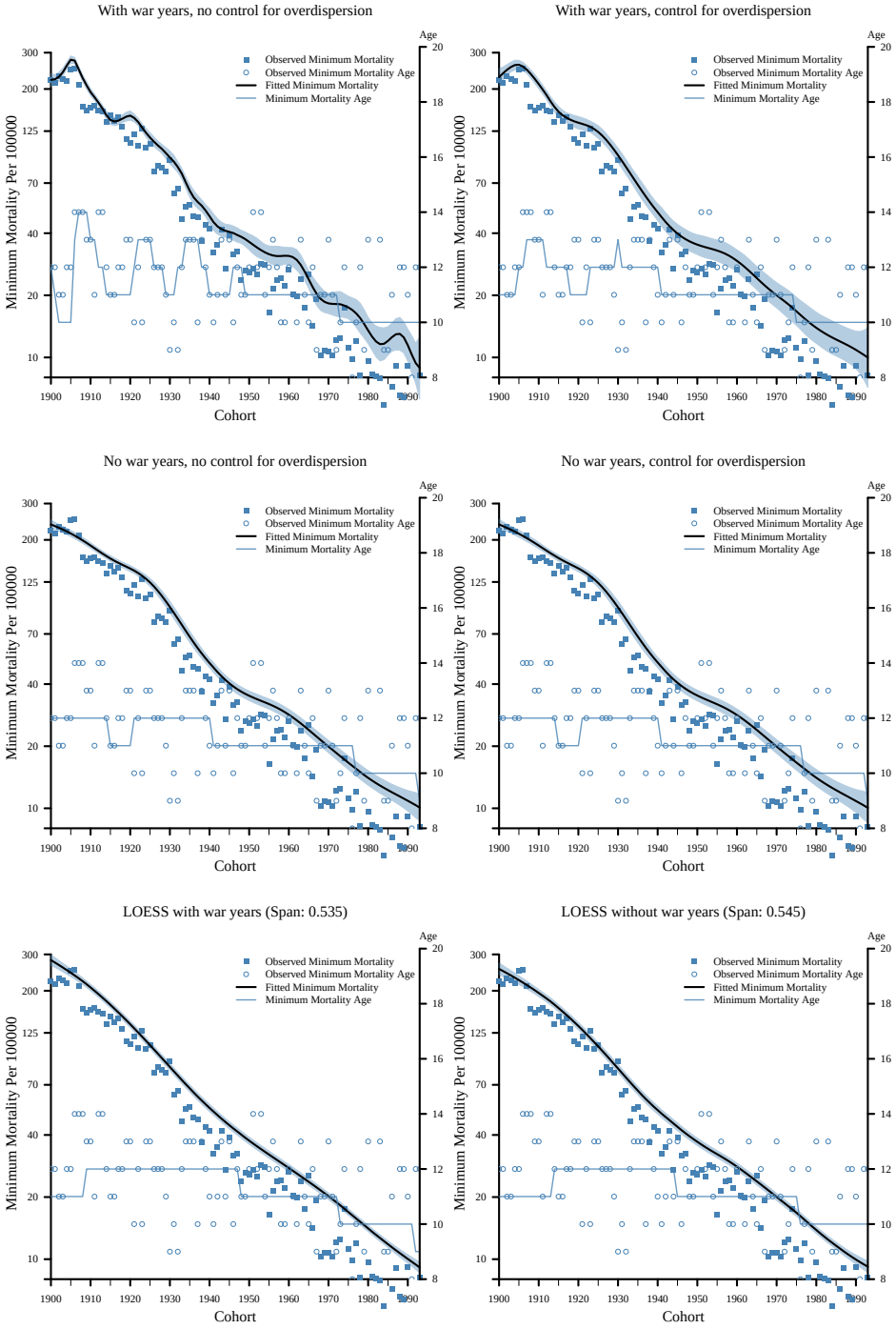

Figure 3. Minimum Mortality and Minimum Mortality Age Estimation under different Model Specifications, Sweden, Males, Cohorts 1900–1993. Data: Human Mortality Database (2017)

4 Influence of war on age-specific mortality pattern

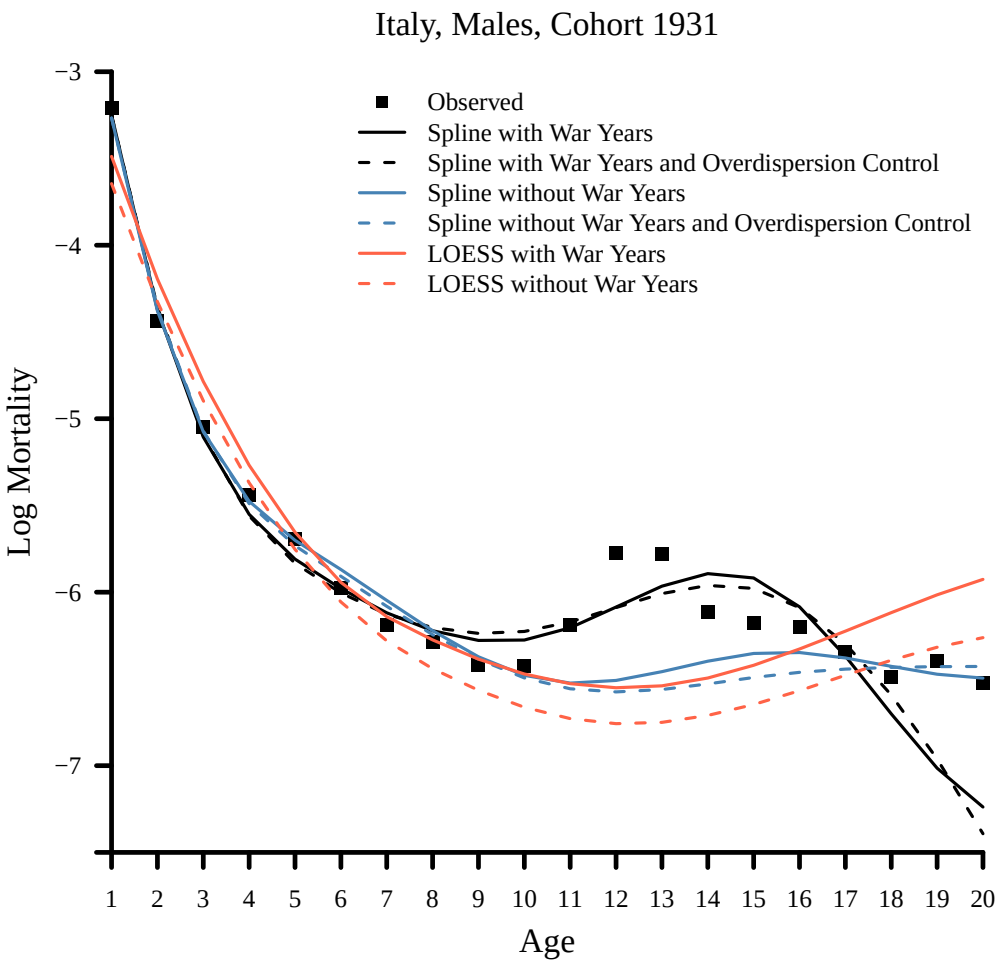

Figure 4. Observed and fitted age-specific mortality trajectory, Italy, males, cohort 1931. Data: Human Mortality Database (2017)

5 Minimum Mortality All Other Countries

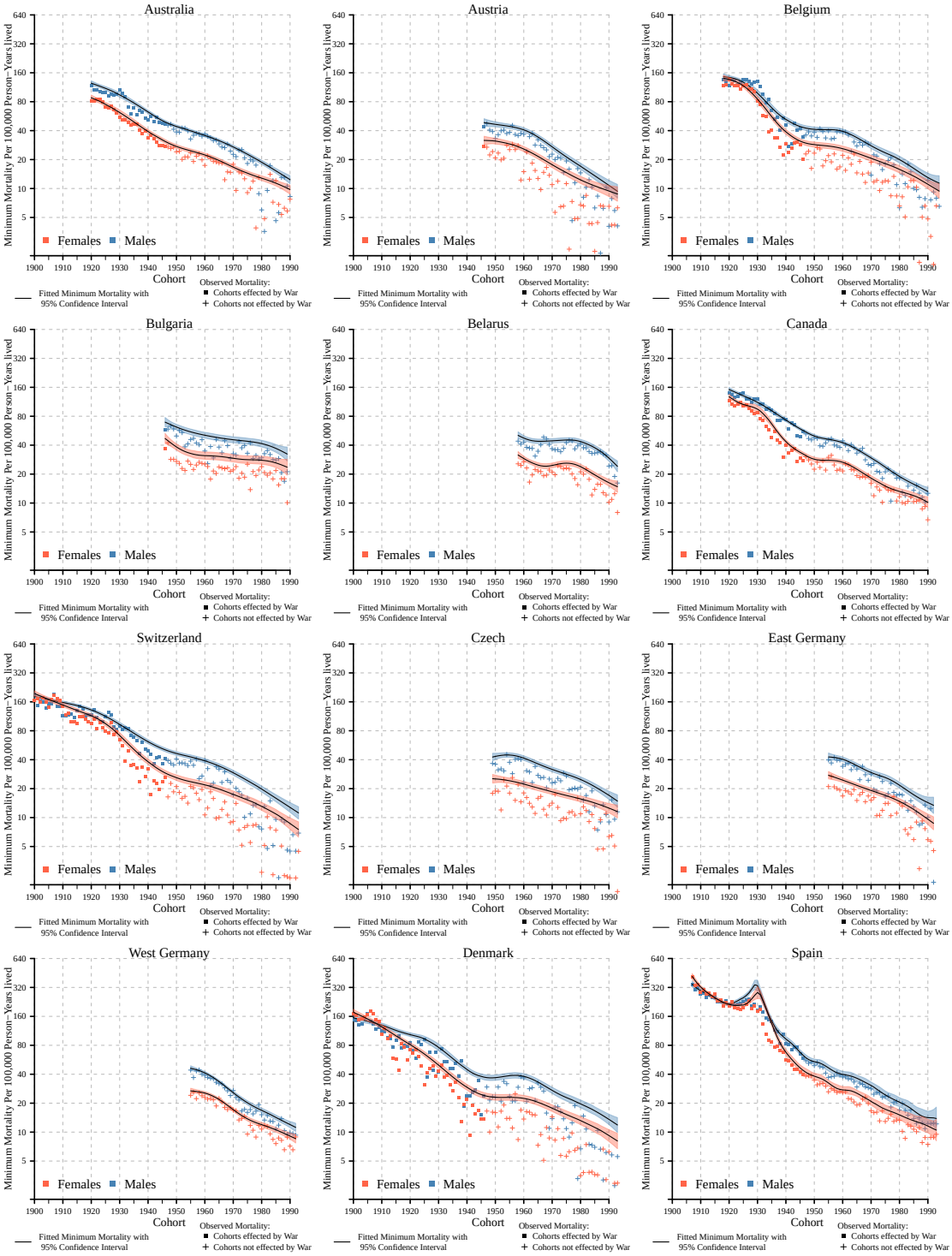

Figure 5. Minimum mortality, all other countries, females and males. Data: Human Mortality Database (2017)

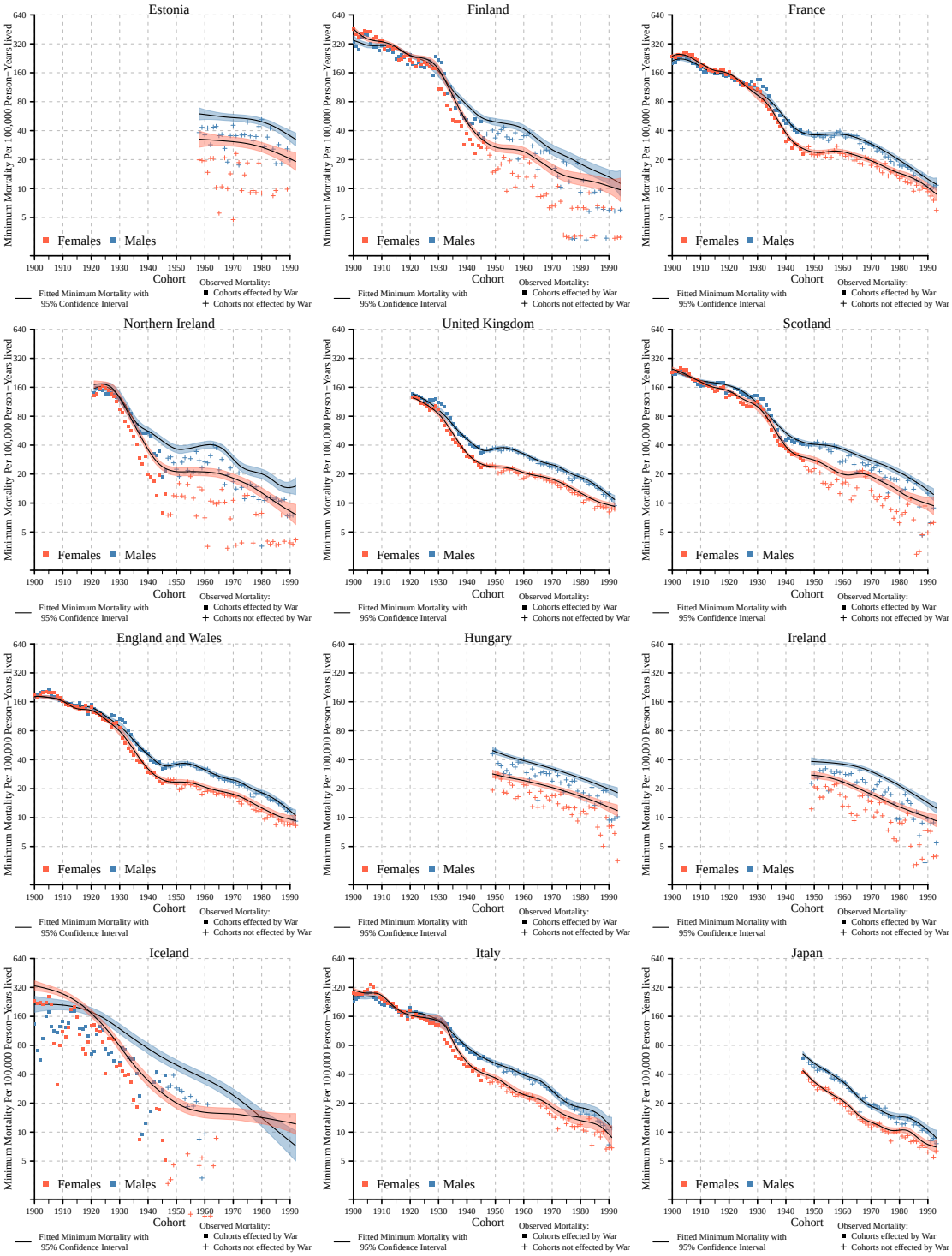

Figure 6. Minimum mortality, all other countries, females and males. Data: Human Mortality Database (2017)

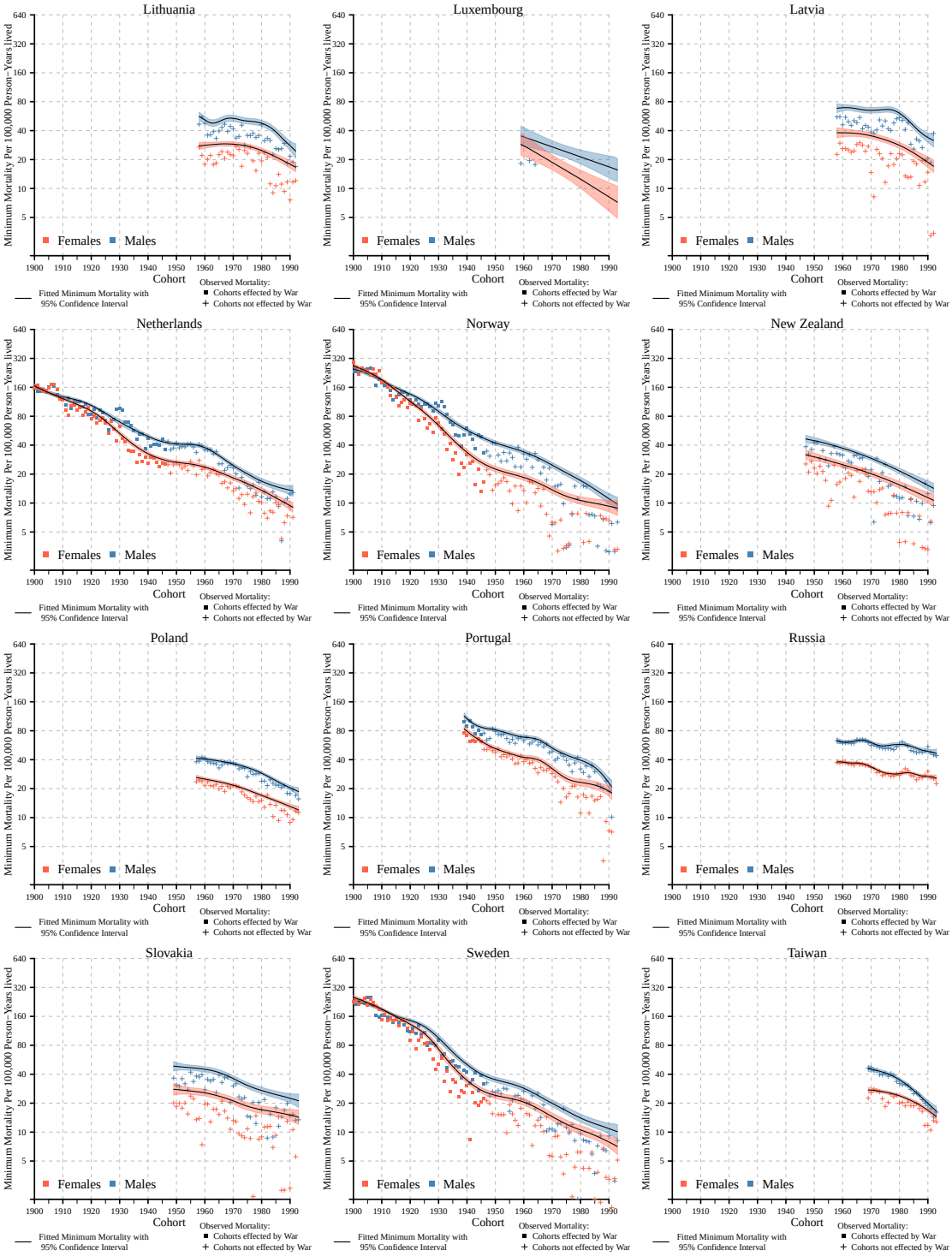

Figure 7. Minimum mortality, all other countries, females and males. Data: Human Mortality Database (2017)

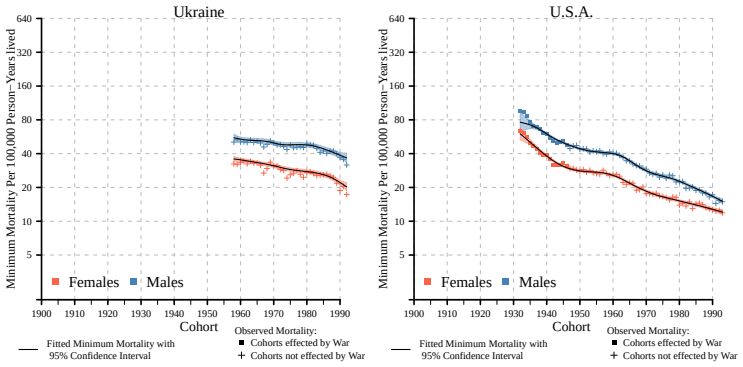

Figure 8. Minimum mortality, all other countries, females and males. Data: Human Mortality Database (2017)

6 Development of the U-shaped mortality pattern

Figure 9 shows the ratio of age-specific death rates adjacent to the minimum mortality and minimum mortality for various countries and four different cohorts. The trajectories do not reveal a shared trend for both perspective across cohorts and across countries. For instance, with the exception of the birth cohort 1900, Italian females show an approximately stable U-shape across cohorts with marginal convergence towards minimum mortality for younger ages and marginal divergence at ages higher than the minimum mortality age. An almost similar pattern can be seen for Danish females. However, French females show a different trend. For this population, the U-shaped pattern became flatter over time, which is especially visible for ages younger than the minimum mortality age. For French males, in opposite, it seems that the U-shaped pattern became more pronounced over time. Generally, the difference of minimum mortality and adjacent death rates varies also across countries.

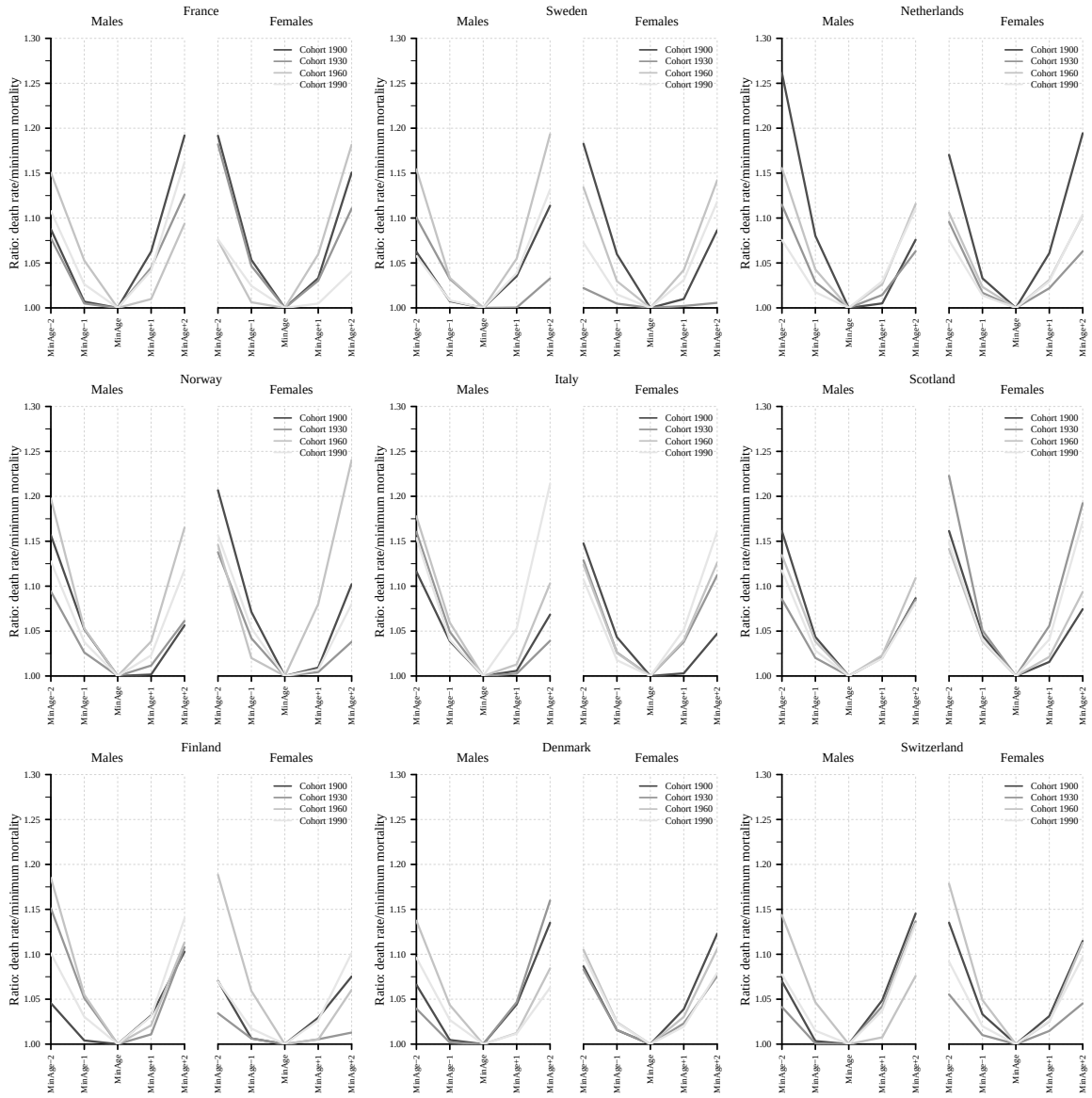

Figure 9. Ratio of death rates at adjacent ages to the location at minimum mortality and minimum mortality, cohorts 1900, 1930, 1960, 1990, males and females, various countries. *Standardized age-specific death rates are shown for the age range: minimum mortality age ± two years of age. The shown estimates are based on the smoothed age-specific death rates. Source: Own illustration using age- and cohort-specific death counts and exposure-to-risk from the Human Mortality Database (2017).*

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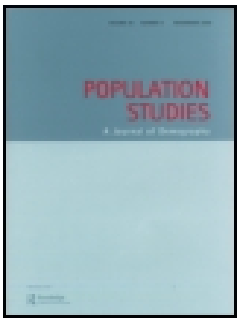
7.3. Paper III: Rectangularization of the survival curve reconsidered: The maximum inner rectangle approach



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Rectangularization of the survival curve reconsidered: The maximum inner rectangle approach

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Rectangularization of the survival curve reconsidered: The maximum inner rectangle approach

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Rectangularization of the survival curve—a key analytical framework in mortality research—relies on assumptions that have become partially obsolete in high-income countries due to mortality reductions among the oldest old. We propose refining the concept to adjust for recent and potential future mortality changes. Our framework, the 'maximum inner rectangle approach' (MIRA) considers two types of rectangularization. Outer rectangularization captures progress in mean lifespan relative to progress in maximum lifespan. Inner rectangularization captures progress in lifespan equality relative to progress in mean lifespan. Empirical applications show that both processes have generally increased since 1850. However, inner rectangularization has displayed country-specific patterns since the onset of sustained old-age mortality declines. Results from separating premature and old-age mortality, using the MIRA, suggest there has been a switch from reducing premature deaths to extending the premature age range; a shift potentially signalling a looming limit to the share of premature deaths.

Supplemental material for this article is available at: http://dx.doi.org/10.1080/00324728.2017.1414299

Keywords: mortality; lifespan variability; rectangularization; compression; survival curve

[Submitted June 2016; Final version accepted July 2017]

Introduction

Rectangularization of the survival curve is one of the key analytical frameworks in mortality research. We argue that the canonical understanding of rectangularization is outdated. Instead of abolishing the concept completely, we suggest extending the framework to accommodate recent developments in mortality.

While the process of rectangularization had been recognized earlier (e.g., Pearl and Miner 1935; Comfort 1956), Fries' (1980) interpretation of the concept has arguably become the commonly accepted view. Mortality developments in high-income countries over recent decades have, however, rendered several of Fries' assumptions obsolete. Most importantly, potential reductions in mortality among the oldest old were not anticipated in the classical rectangularization framework. Mortality for people aged 80 and above has been declining in a number of countries since the 1960s (see, e.g., Kannisto 1994; Kannisto et al. 1994; Vaupel 1997; Rau et al. 2008). This trend in turn has invalidated several (partly implicit) assumptions of Fries' theory: the idea that life expectancy has 'looming limits' has been rejected (Oeppen and Vaupel 2002; Vallin and Meslé 2009), the contributions of improvements in premature mortality to increases in life expectancy have become negligible (Christensen et al. 2009), and Fries' (1980, p. 130) assessment that '[…] there has been no detectable change in the number of people living longer than 100 years […]' has been disproven (e.g., Vaupel and Jeune 1995; Vaupel 2010). Although we have not witnessed any increase in maximum observed lifespan since the death of Jeanne Calment at age 122 in 1997 (Robine and Allard 1998), Fries' (1980, p. 133) prediction that 'human life span may not be fixed but may be slowly increasing, perhaps a month or so each century' had already been exceeded by more than an order of magnitude with the increases that occurred between 1980 and 1997 (Wilmoth et al. 2000).

Furthermore, in Fries' framework, life expectancy gains can only be generated by a decrease in lifespan variability (see, e.g., Nagnur 1986; Nusselder and

Mackenbach 1996). This compression would be completed when, 'under ideal conditions' (Fries 1980, p. 132), lifespans were scattered in a normal distribution around a mean of 85.6 years, with a standard deviation of about four years. Later, Fries (1989) assumed wider intervals. But since the second half of the twentieth century, both stagnating variability and increasing life expectancy have been observed simultaneously (Kannisto 1996; Bongaarts 2005; Canudas-Romo 2008; Bergeron-Boucher et al. 2015). This phenomenon is commonly labelled the 'shifting of mortality'. Some authors go one step further and discuss the possibility of life expectancy gains in the presence of increasing variability in lifespans; this is called the 'expansion of mortality' (Myers and Manton 1984; Rothenberg et al. 1991; Cheung et al. 2005; Engelman et al. 2010, 2014). It is, however, clear that the almost constant difference between the mean and the modal age at death (Canudas-Romo 2010) are not compatible with normally distributed deaths across age, even with historically low levels of premature mortality.

It may come as a surprise that we do not suggest entirely discarding the concept of rectangularization. Indeed, we find the simplicity and intuitiveness of the concept appealing, and recognize that rectangularization is one of the few theoretical frameworks that incorporates the relationship between (average) length of life and lifespan variability. Thus, rather than rejecting the concept, we wish to extend it to incorporate recent mortality changes, as well as future developments.

The first step in extending rectangularization is to detach the framework from its static perspective. It needs to capture mortality changes dynamically at all ages, and not just in the premature age range. It would also be beneficial if a measurement approach could differentiate between and quantify changes in premature and old-age mortality. Furthermore, it would be desirable if an extension of Fries' framework still allowed us to assess the potentially impending limits to lifespan.

The framework we propose, which we call the 'maximum inner rectangle approach' (MIRA), is designed to address these issues. Our approach uses two dimensions of rectangularization. We call the classical perspective 'outer rectangularization' because it relates the survival curve, and, accordingly, life expectancy, to the 'maximum living potential'. Hence, it compares the current experience with the current theoretical maximum, if everyone survived to and then died at the actual maximum lifespan. There is, however, another perspective that has so far largely been neglected, which we call 'inner rectangularization'. Defined as the rectangle under the survival curve with the largest possible area, this perspective relates the current inequality in lifespan to current life expectancy. The basic idea is illustrated in Figure 1. The bold solid line denotes a hypothetical survival curve starting from a radix of one and reaching zero at the highest attainable age, ω. The dotted line denotes the classic reference used to estimate the advancement of rectangularization—the outer rectangle—which also expresses the maximum living potential. The dashed line depicts our newly proposed concept: the maximum inner rectangle.

Maximum inner rectangle approach (MIRA)

The MIRA is based on different areas and specific ages that will be introduced in the following section. Table 1 provides an overview of all MIRA quantities.

In the MIRA, we distinguish between inner and outer rectangularization. *Outer rectangularization* is the standard perspective of rectangularization, and captures progress in mean lifespan relative to progress in maximum lifespan. Hence, the outer frame of the survival curve serves as a reference point. We denote the area of the outer rectangle as ω, because it is determined by the maximum attainable age (ω) and the radix of the survival function (l_0), which we set to one. The maximum age should be able to move forwards or backwards depending on the underlying mortality development. In the empirical application we link ω to a specific survival proportion, k—such as the age at which 1 per cent of the population is still alive—so that $l_\omega = k$.

In a population, ω can be interpreted as the maximum living potential. It counts the hypothetical number of person-years that could be lived in a population if everyone survived to the maximum age and then died. In comparison, the actual number of person-years lived in a population corresponds to the area under the survival curve, and determines mean lifespan. The ratio of mean to maximum lifespan serves to capture the degree of outer rectangularization of the survival curve. Thus, we define the outer rectangle ratio (ORR) as

$$\mathrm{ORR} = \frac{\int_0^\omega l_a \mathrm{d}a}{\omega} = \frac{e_0}{\omega}, \tag{1}$$

with e_0 denoting life expectancy (or mean lifespan) and ω maximum age. By definition, $0 \leq \mathrm{ORR} \leq 1$. The ratio relates the observed number of person-years lived in a population to the maximum person-

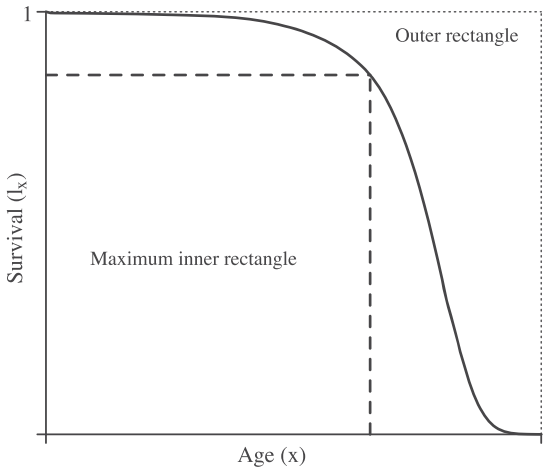

Figure 1 The survival curve and its maximum inner rectangle and outer rectangle

years possible. For example, if ORR = 0.8, then current living conditions are allowing the population to exploit 80 per cent of its current maximum living potential.

Inner rectangularization adds a new perspective. In contrast to the outer rectangle, we seek the largest rectangle under the survival curve. Any inner rectangle (IR) under the survival curve is defined horizontally by age, x, and defined vertically by survival to that age, l_x. Consequently, the corresponding area is $\mathrm{IR}_x = x \times l_x$. The first age derivative of IR_x then identifies the age, x^*, that corresponds to the maximum inner rectangle (MIR) with an area of

$$\mathrm{MIR} = x^* \times l_{x^*} \tag{2}$$

as the solution to

$$\frac{\mathrm{d}\ \mathrm{IR}_x}{\mathrm{d}\ x} = 0 \tag{3}$$

which simplifies to

$$x^* = \frac{1}{\mu_{x^*}}\,. \tag{4}$$

Although there is no closed form solution, x^* can be found numerically, given that

$$\mu_x > 0 \quad \forall\, x \in [0, \omega], \tag{5}$$

where μ_x denotes the force of mortality. A proof for a unique maximum in the case of increasing mortality with age is included in the supplementary material (section A).

MIR counts the 'maximum uniformly shared person-years'. It is determined by the maximum shared lifespan (x^*) and the survival proportion up to this lifespan (l_{x^*}). At ages below x^*, the share of the population living for x years (l_x) would be larger than l_{x^*}, but the number of years lived per individual would be smaller than x^*. Likewise, at ages above x^*, the number of years lived per individual would be larger than x^*, but the share of the population living for x years (l_x) would be smaller than l_{x^*}. In either case, the total number of uniformly shared person-years, as indicated by MIR, would be reduced.

Using this definition of MIR allows us to add an inner perspective to the process of rectangularization. In an analogy to the maximum living potential (ω), we can interpret life expectancy (e_0) as a population's current theoretical maximum number of life years that could be shared uniformly. Accordingly, with perfectly uniform lifespans, 100 per cent of individuals in a population would share a lifespan of length e_0. With actual lifespan inequality as measured by MIR, however, a maximum survival fraction of $l_{x^*} < 100$ per cent shares a uniform lifetime of at most x^* years. Thus, by relating MIR to e_0, we define inner rectangularization as the process of a population approaching its current lifespan equality potential. It is measured by the inner rectangle ratio (IRR), which is given by

$$\mathrm{IRR} = \frac{\mathrm{MIR}}{e_0}. \tag{6}$$

The IRR captures a trend that differs from that of the ORR, because changes in the MIR do not require a change in the mean or the maximum lifespan. Indeed, the trend could be characterized by a constant mean and a falling maximum lifespan, or by an increasing mean but a constant maximum lifespan, or even by a falling mean and a falling maximum lifespan. Though closely related, the IRR differs from

Table 1 Quantities of the maximum inner rectangle approach (MIRA)

Name	Acronym	Expression	Interpretation
Inner rectangle	IR_x	$x \times l_x$	Age-specific uniformly shared person-years (PY)
Maximum shared lifespan	x^*	$\max[x \times l_x]$	Maximum number of uniformly shared life years by largest number of survivors
Maximum proportion	l_{x^*}	l_{x^*}	Largest proportion alive at the maximum shared lifespan
Maximum inner rectangle	MIR	$x^* \times l_{x^*}$	Population's current maximum number of uniformly shared PY
Life expectancy	e_0	$\int_0^\omega l_a da$	Population's current number of PY, i.e., mean lifespan
Outer rectangle	ω	$\omega \times l_0$	Maximum possible PY
Outer rectangle ratio	ORR	e_0/ω	Proportion of PY lived from maximum possible PY
Inner rectangle ratio	IRR	MIR/e_0	Proportion of uniformly shared PY from all PY lived
Total rectangle ratio	TRR	MIR/ω	Proportion of uniformly shared PY lived of maximum possible PY

the ORR because it is essentially an index of lifespan equality, while the ORR is an index of exploiting maximum living potential. Accordingly, if IRR = 0.8, then current living conditions are allowing the population to exploit 80 per cent of its current lifetime equality potential.

The two indices can be combined into a single index to measure total rectangularization. We define the total rectangle ratio (TRR) as

$$\text{TRR} = \text{IRR} \times \text{ORR} = \frac{\text{MIR}}{\omega}. \qquad (7)$$

The TRR measures achieved lifespan equality in relation to maximum possible equality. Accordingly, if TRR = 0.8, then current living conditions are allowing the population to achieve 80 per cent of its maximum possible lifespan equality at present.

Data and estimation procedure

We computed MIRA quantities using period life tables, which we estimated from death counts and corresponding exposures from the Human Mortality Database (2015). In this paper, we choose to highlight the trajectories of Swedish, Danish, and Italian females because these countries provide three exemplary mortality developments. Furthermore, all three countries have sufficient data coverage over time. In estimating x^* and l_{x^*}, a key challenge we faced was that the data are only available in discrete integer units, but x and l_x need to be continuous. Therefore, we estimated x^* in two steps. First, we smoothed the product of x and l_x with cubic splines using *R*'s *splinefun()* function (R Core Team 2015), which allowed us to evaluate the function value with arbitrary precision. Second, we used *R*'s general-purpose univariate optimization function *optimize()* to find the maximum. A similar two-step approach with splines has been used previously in mortality research to estimate the modal age at death (Ouellette and Bourbeau 2011). We calculated other age estimates, such as ω and the threshold ages discussed in the next section, using the same procedure.

In several empirical studies on rectangularization, the maximum age (ω) is not set at the actual age at which there are no survivors left in the life table population. Wilmoth and Horiuchi (1999), for instance, set the cut-off age at the point at which 0.1 per cent of the population were still alive. Rossi et al. (2013) used the 10 per cent threshold and, most recently, Schalkwijk et al. (2016) used the 0.1, 1, and 10 per cent thresholds. In our study, we opted for a threshold of 1 per cent. Sensitivity analysis revealed that the actual choice of value for l_ω had only minor effects on the results. As the maximum age changes with varying survival fractions, the estimates of TRR and ORR change quantitatively. However, the patterns of the ratios remain stable over time.

Illustration of the inner, the outer, and the total rectangle ratio

Figure 2 shows the three ratios for females in Italy (upper left panel), Denmark (upper right), and Sweden (lower). It depicts the IRR (black line), the ORR (light grey), and the TRR (dark grey). Figure 2 illustrates the following key points:

(1) The TRR and the ORR have been developing almost in parallel for about 160 years. This suggests that Fries' concept of rectangularization needs to be revised. If Fries' ideas were correct, we would have expected to witness a 'catching-up period' of the TRR to the ORR until his 'ideal conditions' with

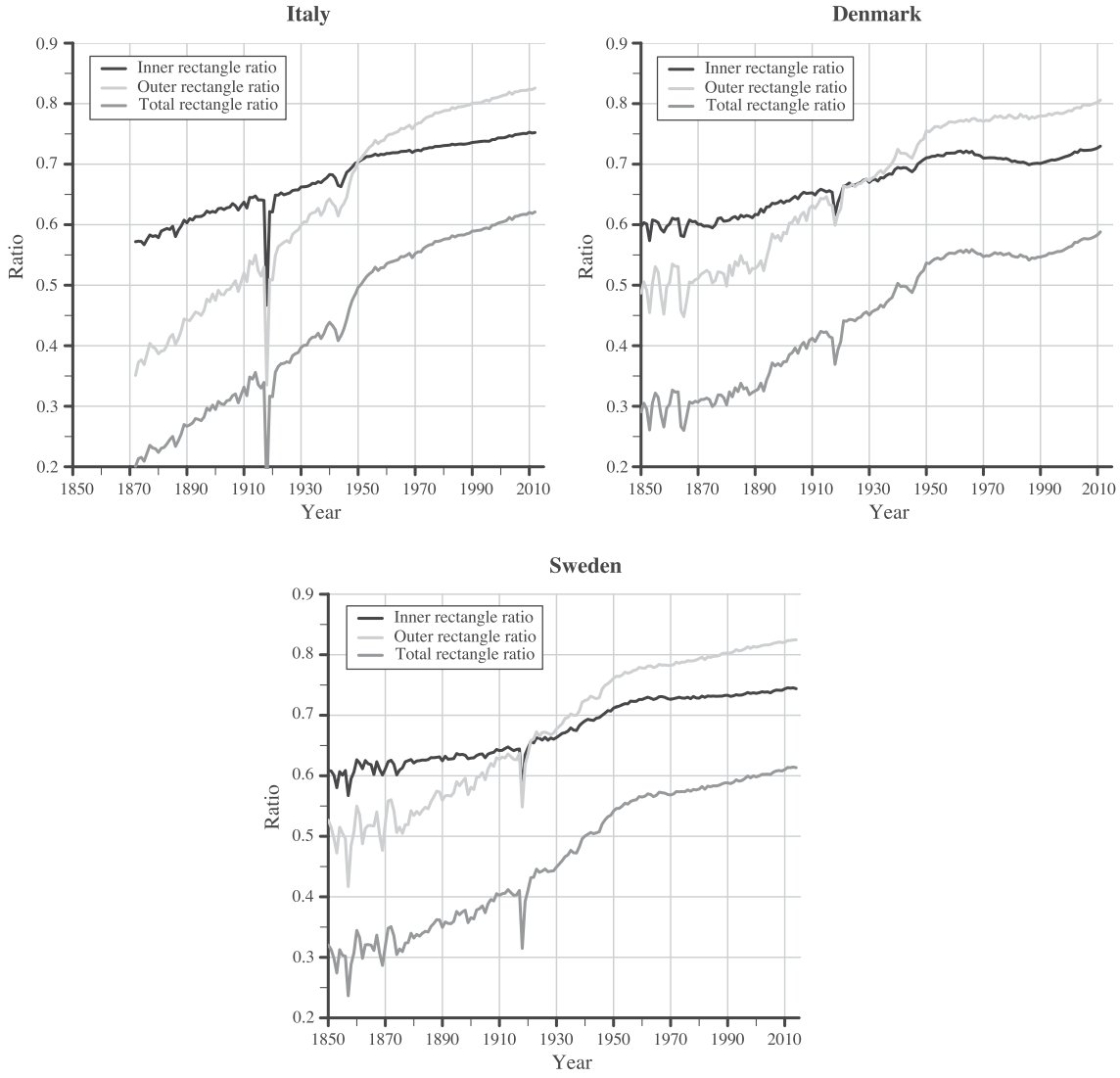

Figure 2 Inner rectangle ratio, outer rectangle ratio, and total rectangle ratio for females, Italy (1872–2012), Denmark (1850–2011), and Sweden (1850–2014)
Note: All calculations are based on period life tables in the respective year.
Source: Human Mortality Database (2015).

life expectancy of about 85 years were reached. None of the selected countries has reached this level of life expectancy yet. Consequently, we should see a continued narrowing of the gap, but this is not the case.

(2) Inner rectangularization describes a different dimension of mortality progress. Its trajectory is decoupled from those of the ORR and the TRR, mainly because its frame of reference is not maximum lifespan, but life expectancy. In each of the examples we can see an increase over time, with a trend change occurring sometime during the 1950s when the slope becomes shallower. This break can likely be attributed to the shift in survival improvements from younger to older ages (e.g., Christensen et al. 2009).

(3) We can see that the IRR in each of these three countries has followed a different trajectory over the last half century, with a steady increase in Italy, a slow increase in Sweden, and a slight dip in Denmark during the 1970s and 1980s. Even though the IRR has generally increased over time, the country-specific patterns suggest that the

forces behind this development vary. For instance, the steady increase in Italy suggests that a rise in life expectancy accompanies a faster growth of the MIR. The almost stagnating IRR in Sweden between 1960 and 1990 suggests that life expectancy has increased similarly to the MIR. Denmark's unusual dynamics suggest a declining MIR while life expectancy stagnated.

(4) The IRR highlights how evenly the magnitude of and trends in age-specific mortality changes are spread over age. We claim that this provides a new perspective on lifespan variability. Our suggestion is strengthened by the correlation between the IRR and other summary measures of lifespan variability. Table 1 in the supplementary material (section B) shows that the IRR is less correlated with common measures of lifespan variability (shown in Table 2 in the supplementary material) than those measures are correlated with each other. This pattern is especially pronounced for the time period when gains in premature survival were instrumental for the increase in life expectancy (the period 1850–1950 in our analysis).

Applying the MIRA to separate premature from old-age mortality

Premature and old-age mortality are terms that are frequently used in mortality research, but they are loosely defined, which may be sufficient for many applications. However, in analysing rectangularization, these definitions are a crucial issue. In Fries' description, premature mortality plays a central role. He argues that declines in premature mortality drive the process of rectangularization, and implicitly assumes that only these improvements are generating life expectancy increases. Although, Fries remains unclear in his definition of premature ages, his descriptions most evidently suggest that he is referring to life expectancy as a threshold. We argue that x^* can be interpreted as an age that allows us to separate premature from old-age mortality. With x^*, we provide an approach that is embedded within our rectangularization framework, and which quantifies the threshold. Accordingly, the threshold in MIRA is based on the longest lifespan that is shared by the largest fraction of the population.

Even though existing approaches, such as those by Zhang and Vaupel (2009) and Gillespie et al. (2014), rest on different lifespan variability measures, their respective threshold ages result from a proportional perturbation of age-specific mortality and, hence, they rely on the same perturbation/definition. In both approaches, the threshold refers to a specific age, such that proportional mortality reductions before this age would result in a decrease in lifespan variability, whereas reductions at higher ages would lead to an increase in lifespan variability (see section F of the supplementary material for more details). Hence, x^* could serve as an alternative definition of a threshold age separating premature and old-age mortality, based on the maximum shared lifespan.

Figure 3(a) illustrates the relationship between x^* (horizontal axis) and l_{x^*} (vertical axis); that is, the coordinates to measure the number of maximum uniformly shared person-years; again, this is shown for females in Italy, Denmark, and Sweden. The grey contour lines depict the number of life years lived in the MIR. The two time periods 1850–1950 and 1951–2014 are illustrated by dashed and solid lines, respectively. Generally, two trends can be distinguished in Figure 3(a): a 'vertical' development (until 1950) and a 'horizontal' development (after 1950).

The share of the life table population dying at older ages is denoted by l_{x^*}. Consequently, $1 - l_{x^*}$ equals the proportion dying prematurely. Premature mortality improvements drove progress until 1950, as illustrated by the increasing share of survivors (l_{x^*}). With improving old-age mortality, x^* shows an accelerated movement towards higher ages; whereas the corresponding survival fraction at x^* (l_{x^*}) shows only small gains. This pattern is similar to that of the modal age at death, which is also robust to mortality changes at lower ages, but sensitive to changes at higher ages (Canudas-Romo 2010).

To compare the trajectories resulting from our approach with alternative approaches, we also plot the relationship between the threshold ages proposed by Gillespie et al. (2014; Figure 3(b)) and Zhang and Vaupel (2009; Figure 3(c)), which are based on the variance and the number of life years lost, respectively, and the corresponding survival proportions. These estimates show similar shifts in the trend. The slopes of the curves in each of the three parts of Figure 3 have become shallower in recent decades. This development could point to the existence of a limit to premature mortality that cannot be lowered any further. Fries (1980) argued that premature mortality would be almost eliminated,

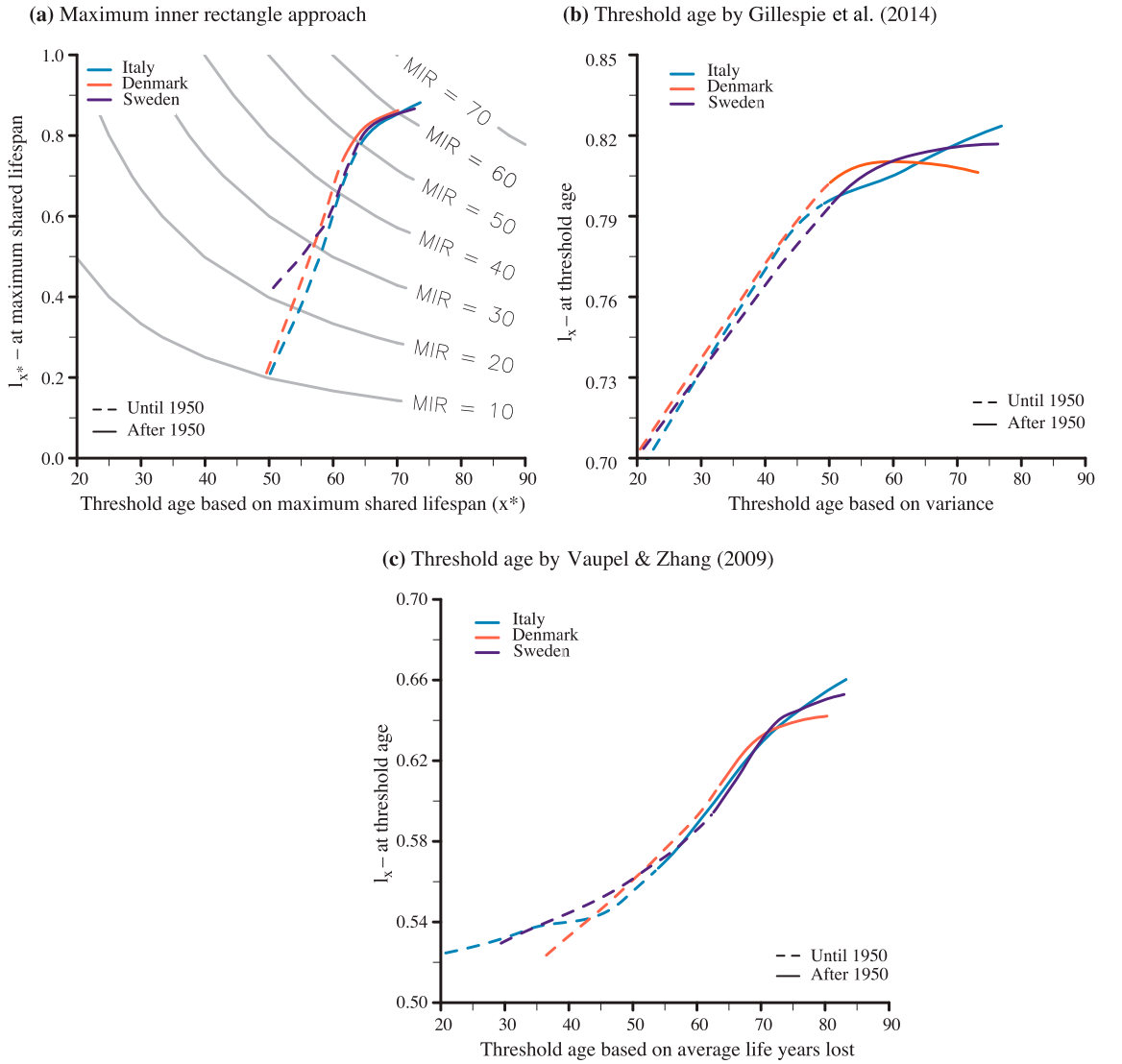

Figure 3 Scatterplot of the threshold age and survival proportion at the threshold age according to: (a) MIRA; (b) Gillespie et al. (2014); and (c) Zhang and Vaupel (2009), women, Italy (1872–2012), Denmark (1850–2011), and Sweden (1850–2014)

Notes: The trend lines are based on a locally weighted smoothing to highlight the patterns only. Additionally, contour lines in panel (a) visualize the corresponding number of person-years lived in equality (MIR), since this is determined by the product of both. The approaches of Zhang and Vaupel (2009) and Gillespie et al. (2014) and their calculation are explained in more detail in the supplementary material.

Source: As for Figure 2.

declining to a maximum share of 2–5 per cent of all deaths. The three measures presented here suggest that there is a limit of at least 10–15 per cent (the minimum is from the MIRA measure).

It should, however, be noted that we use different vertical scales within Figure 3. If we instead used the scale from our measure in Figure 3(a) for the other two measures, we would obtain almost horizontal lines for those measures. Within the 160 years of life expectancy development contained in the figure, the proportion dying at old age has changed relatively little under the threshold ages suggested by Zhang and Vaupel (2009) and Gillespie et al. (2014). In both cases, the change amounts to less than 15 percentage points; a shift we consider to be rather small. In contrast, our measure shows a shift of about 65 percentage points, from 20 per cent dying at old age in 1850 to about 85 per cent dying

at old age in the most recent years. These numbers seem to be more in line with the findings of, for example, Christensen et al. (2009), who estimated that almost 80 per cent of recent gains in life expectancy for Japanese women were caused by survival improvements among older people.

Discussion and conclusion

Rectangularization is one of the established analytical frameworks in mortality research. We propose refining the classical concept to adjust for recent changes in survival improvements, and to allow for the incorporation of anticipated mortality trajectories in the near future. This new framework, which we call the maximum inner rectangle approach (MIRA), rests on two theoretically distinct types of rectangularization: inner rectangularization and outer rectangularization.

Outer rectangularization relates the number of life years that are currently lived (i.e., life expectancy) to a theoretical maximum where everyone dies at the same (maximum) age. We extend this standard definition of rectangularization by introducing the concept of *inner rectangularization*. This novel concept corresponds to the largest rectangle *under* the survival curve. This rectangle captures the largest number of life years lived by the largest proportion of the population. Thus, it measures the proportion of lifespan equality at the current level of life expectancy. By contrast, outer rectangularization measures the degree of living potential exploited, using maximum lifespan as a reference point.

The measurement of both constituent parts of the MIRA rests on simple ratios. To measure outer rectangularization, we use the well-known concept of the moving rectangle (Wilmoth and Horiuchi 1999; Rossi et al. 2013; Schalkwijk et al. 2016). As far as we know, there are no demographic predecessors to our concept of inner rectangularization. Thus, the age that maximizes the IR provides a novel point of reference, indicating maximum shared lifespan. This point represents the optimal trade-off between past lifetime and number of survivors in terms of lived person-years. Hence, the principle of inner rectangularization rests on identifying the optimal combination of two (inversely related) inputs—age and survival—which unify the biggest area under a curve representing their respective relationship. Such measures have previously been applied elsewhere. For instance, the Hirsch index (or h-index) measures the productivity and citation impact of scientists (Hirsch 2005). It depicts that x publications of a scientist have been cited at least x times. The geometric equivalent is a list of all publications by a scientist (y-axis) sorted by the number of citations (x-axis). This approach is similar to our application, where the survival curves could be interpreted as a sorted list of life lengths (x-axis) of the population (y-axis). Another example of such a maximum rectangle can be found in physics: the 'maximum power point' indicates the maximum power of a photovoltaic module, with a given current–voltage curve (Wasynezuk 1983). Our approach is also related to Cohen's (2015) decomposition of life expectancy model, which derives Markov's inequality and Chebyshev's inequality for tail probabilities in a novel way. In this approach, Cohen decomposes life expectancy into three parts, one of these parts being a non-maximized version of the IR.

Our most important empirical findings are as follows. First, we found that outer rectangularization has shown continuous gains over time (see also Figures 1 and 2 in sections C and D of the supplementary material). This is a consequence of the straight linear increases in life expectancy (Oeppen and Vaupel 2002), which have been faster than the increase in the longest lifespans, as measured by ω. However, we also detected a considerably slower pace of outer rectangularization since the middle of the twentieth century. Second, we found that inner rectangularization also increased rather uniformly until around 1950; and that the patterns thereafter could not be summarized with a general trend because they are rather country-specific (see also Figures 1 and 2 in sections C and D of the supplementary material). These country-specific patterns appear to be attributable to differences in the onset of sustained mortality declines among the oldest old (Kannisto 1994), as well as by other factors, such as smoking among Danish women (e.g., Juel et al. 2000; Jacobsen et al. 2002; Lindahl-Jacobsen et al. 2016), and postponed reforms of the healthcare system in the Netherlands (Mackenbach et al. 2011; Peters et al. 2015).

If we interpret x^*, the age maximizing the IR, as a threshold age separating premature from old-age mortality, then the rises in x^* (see also Figure 3 in section E of the supplementary material) and in corresponding survival, l_{x^*}, switched in around 1950, from a reduction in premature deaths to an extension of the premature age range. This dynamic also points to a potential minimum proportion of individuals dying prematurely. Depending on the underlying definition of threshold age, the share dying prematurely varies between 10–15 per cent (MIRA), 15–20 per cent (Gillespie et al. 2014), and 30–35 per

cent (Zhang and Vaupel 2009) under current mortality conditions. Hence, Fries' prediction that premature mortality would be almost completely eradicated seems rather unlikely. We can, however, see that the definition and measurement of premature mortality are issues that have been unresolved at least since Lexis (1877).

Using x^* as a reference point also would enable us to extend the MIRA beyond the areas and ratios presented here. These areas above and below the survival curve would allow us to decompose life expectancy and maximum living potential because they capture all person-years apart from those included in the MIR. An application of the decomposition could provide a basis for a more detailed analysis of past and potential future developments of rectangularization. For instance, the non-uniform number of person-years lived of life expectancy (e_0 − MIR) could be subdivided into the numbers for those dying prematurely and for those living longer than x^*. Such an analysis can show to what extent changes in life expectancy, and thus rectangularization, are determined by lifespan equality increases, premature mortality reductions, and longevity extensions. Generally, we would expect to see continuous gains in life expectancy if large shares of the population benefit from mortality improvements. This would result in rising lifespan equality. Indeed, the absolute number of uniformly shared person-years (MIR) has increased in almost all countries with continuously rising life expectancy (see Figure 3(a), for example). But relative to life expectancy, the country-specific patterns of the IRR after 1950 question this relationship (as shown in Figure 2). The rise in uniformity of person-years lived seems to be more detached from overall gains in life expectancy in some countries than in others. In the selected countries, for instance, Italian females were shown to be closest to the described scenario; whereas Danish females, with their convex IRR trend, were found to have a stronger degree of detachment. The extension opportunities offered by the MIRA should help us to gain deeper insights into these dynamics.

Notes and acknowledgements

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Rectangularization of the survival curve reconsidered: The maximum inner rectangle approach (Online Supplemental Material)

A Second Derivative of MIRA Equation

In general, the relationship between hazard ($\mu(x)$), density ($f(x)$) and survival function ($l(x)$) is given by

$$f(x) = l(x)\mu(x) \tag{1}$$

Hence, the derivative with respect to x of equation 1 is

$$\frac{d\, f(x)}{d\, x} = \frac{d\, l(x)}{d\, x}\mu(x) + \frac{d\, \mu(x)}{d\, x} l(x) \tag{2}$$

Dividing equation 2 by $f(x)$ results in

$$\frac{\frac{d\, f(x)}{d\, x}}{f(x)} = -\mu(x) + \frac{\frac{d\, \mu(x)}{d\, x}}{\mu(x)}. \tag{3}$$

The general equation for calculating x^* is given by

$$A(x) = xl(x). \tag{4}$$

Deriving equation 4 with respect to x results in

$$\frac{d\, A(x)}{d\, x} = A' = l(x) - xf(x). \tag{5}$$

For an extremum x^*, it is required that

$$A'(x^*) = l(x^*) - x^* f(x^*) = 0. \tag{6}$$

Rewriting equation 6 gives

$$x^* = \frac{1}{\mu(x^*)}. \tag{7}$$

The second derivative of equation 4 is equal to

$$\frac{d^2\, A(x)}{d^2\, x} = A''(x) = -f(x) - f(x) - x \cdot \frac{d\, f(x)}{d\, x} = -2f(x) - x \cdot \frac{d\, f(x)}{d\, x}. \tag{8}$$

For a maximum, it has to hold that

$$-2f(x) - x \cdot \frac{d\, f(x)}{d\, x} < 0 \qquad \forall\, x = x^*. \tag{9}$$

This can also be expressed by

$$\frac{-2}{x} < \frac{\frac{d\, f(x)}{d\, x}}{f(x)} \qquad \forall\, x = x^*. \tag{10}$$

Inserting equations 7 and 3 in equation 10 results in

$$-2\mu(x) < \frac{\frac{d\,\mu(x)}{d\,x}}{\mu(x)} - \mu(x) \qquad \forall\, x = x^*. \tag{11}$$

Simplifying equation 11 reduces the problem to

$$-\mu(x)^2 < \frac{d\,\mu(x)}{d\,x} \qquad \forall\, x = x^*, \tag{12}$$

which is always true for an increasing force of mortality over age, representing the adult human pattern.

B Correlation Analysis of Common Lifespan Variability Measures and MIRA Ratios

Table 1: Kendall's correlation coefficient τ for measures of variability and MIRA indices for period data available in the Human Mortality Database, both sexes combined. The lower left triangle refers to the years 1850–1950, the upper right triangle refers to the years 1951–2014. We selected Kendall's τ because it does not rely on a linear relationship like Pearson's r, or a monotonic relationship like Spearman's ρ. For New Zealand, data for the entire population are included. For Germany, separate data for the eastern and the western regions are included. England and Wales, Scotland, and Northern Ireland are included separately. Measures are calculated based on period life tables in the respective years. The measures in continuous notation and examples of their usage can be found in Table 2. (G - Gini-coefficient, CV - coefficient of variation, H - Keyfitz' entropy, IQR - inter quartile range, SD - standard deviation, VAR - variance, S_{10} - standard deviation above age 10, $e^\dagger$ - average life years lost)

	IRR	ORR	TRR	IQR	$e^\dagger$	H	G	CV	SD	VAR	S_{10}
IRR		0.73	0.84	-0.88	-0.81	-0.79	-0.69	-0.60	-0.53	-0.53	-0.78
ORR	0.78		0.89	-0.77	-0.85	-0.92	-0.88	-0.82	-0.73	-0.73	-0.67
TRR	0.83	0.94		-0.85	-0.89	-0.93	-0.83	-0.75	-0.66	-0.66	-0.74
IQR	-0.64	-0.80	-0.77		0.86	0.82	0.73	0.65	0.59	0.59	0.80
$e^\dagger$	-0.69	-0.74	-0.75	0.76		0.90	0.81	0.74	0.68	0.68	0.81
H	-0.78	-0.92	-0.91	0.81	0.81		0.88	0.80	0.71	0.71	0.73
G	-0.74	-0.94	-0.90	0.84	0.75	0.91		0.91	0.82	0.82	0.65
CV	-0.72	-0.93	-0.88	0.84	0.74	0.90	0.98		0.89	0.89	0.58
SD	-0.50	-0.68	-0.64	0.81	0.71	0.69	0.73	0.74		1.00	0.54
VAR	-0.50	-0.68	-0.64	0.81	0.71	0.69	0.73	0.74	1.00		0.54
S_{10}	-0.60	-0.54	-0.57	0.54	0.71	0.60	0.54	0.53	0.52	0.52	

Table 2: Overview of common measures of variability. All measures are used in the correlation analysis. Not all mentioned examples apply the measure according to the mentioned notation.

Measure	Expression	Example
Gini-Coefficient	$G = 1 - \frac{1}{e_0} \int_0^\omega l_a^2 \, da$	Shkolnikov et al. (2003); Smits and Monden (2009)
Average Life Years Lost	$e^\dagger = \int_0^\omega e_a d_a \, da$	Vaupel et al. (2011)
Keyfitz' Entropy	$H = \frac{\int_0^\omega e_a d_a \, da}{e_0}$	Nagnur (1986); Nusselder and Mackenbach (1996)
Standard Deviation	$SD = \sqrt{\int_0^\omega (a - e_0)^2 d_a \, da}$	Myers and Manton (1984); Edwards and Tuljapurkar (2005); Thatcher et al. (2010); Kannisto (2000)
Variance	$VAR = \int_0^\omega (a - e_0)^2 d_a \, da$	Edwards and Tuljapurkar (2005); Gillespie et al. (2014)
Coefficient of Variation	$CV = \frac{\sqrt{\int_0^\omega (a-e_0)^2 d_a \, da}}{e_0}$	Anand et al. (2001)
Standard Deviation above Age 10	$S_{10} = \frac{\sqrt{\int_{10}^\omega ([a-10]-e_{10})^2 d_a \, da}}{l_{10}}$	Edwards and Tuljapurkar (2005); Gillespie et al. (2014)
Inter Quartile Range	$IQR = X_{l_{0.75}} - X_{l_{0.25}}$	Wilmoth and Horiuchi (1999)

C MIRA Ratios for all HMD Countries: Females

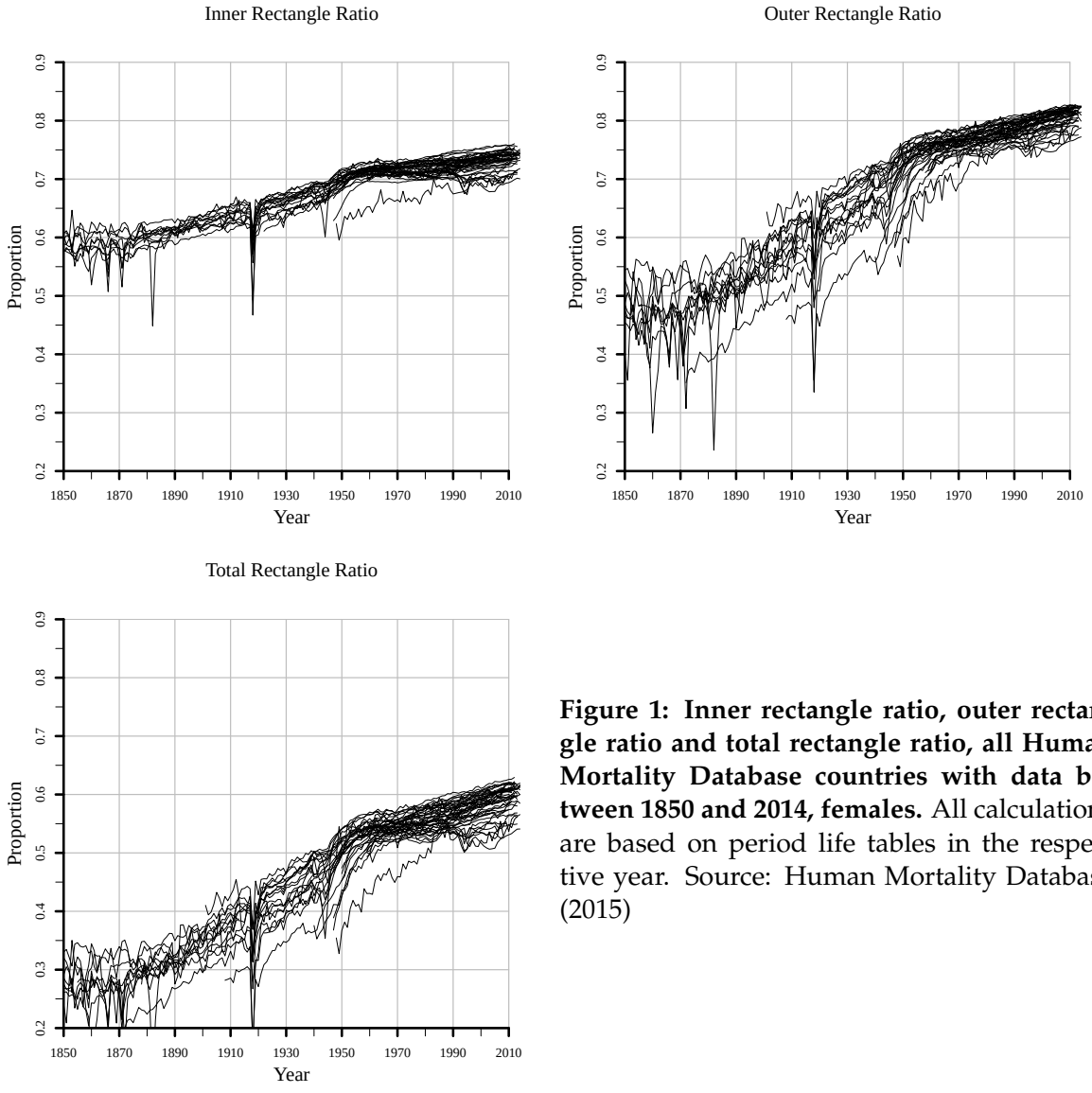

Figure 1: Inner rectangle ratio, outer rectangle ratio and total rectangle ratio, all Human Mortality Database countries with data between 1850 and 2014, females. All calculations are based on period life tables in the respective year. Source: Human Mortality Database (2015)

D MIRA Ratios for all HMD Countries: Males

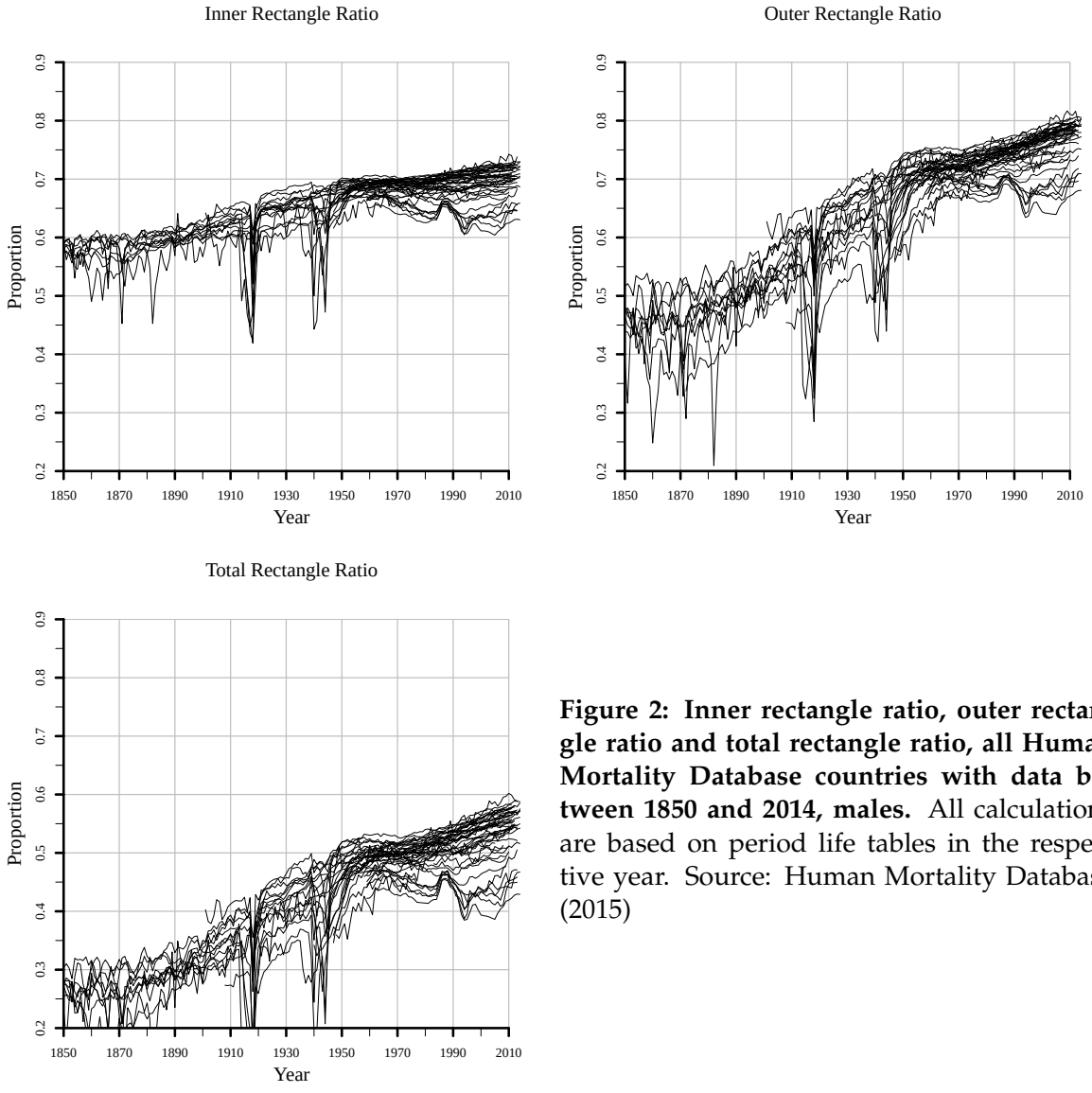

Figure 2: Inner rectangle ratio, outer rectangle ratio and total rectangle ratio, all Human Mortality Database countries with data between 1850 and 2014, males. All calculations are based on period life tables in the respective year. Source: Human Mortality Database (2015)

E Maximum shared lifespan x^* for all HMD Countries

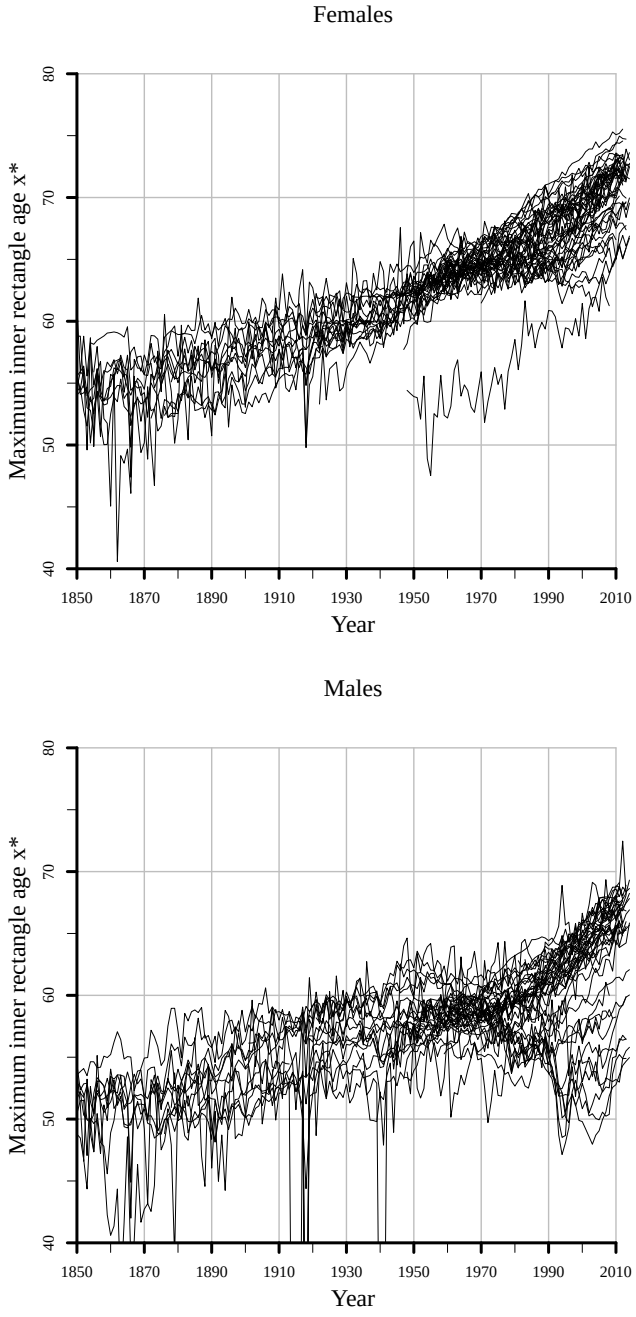

Figure 3: Maximum shared lifespan x^*, all Human Mortality Database countries with data between 1850 and 2014, males and females. All calculations are based on period life tables in the respective year. Source: Human Mortality Database (2015)

F The threshold ages used to separate early and late mortality

The term "threshold age" is commonly used to describe a specific age, which represents the cut-off age between premature and old-age mortality. Existing approaches commonly define this border such that mortality reductions before this age would result in a decrease of lifespan variability, whereas mortality reduction at higher ages would lead to an increase of lifespan variability. Zhang and Vaupel (2009) and Gillespie et al. (2014) derive threshold ages using average life years lost ($e^{\dagger}$) and variance of the age at death (VAR), respectively. Van Raalte and Caswell (2013) provide results for a range of lifespan variability measures using matrix calculus.

The approaches by Zhang and Vaupel (2009) rests on a proportional change in age-specific mortality, expressed by the derivative of the respective measures with respect to the logarithm of the force of mortality (μ_x). Accordingly, the age separating early and late mortality in the approach by Zhang and Vaupel (2009) is given by the root of equation 13,

$$k_x = e_x^{\dagger} + e_x(H_x - 1) \tag{13}$$

where $e_x^{\dagger}$ denotes average person years lost above age x, e_x is the remaining life expectancy at age x, and H_x denotes the cumulative hazard at age x.

In their applications, Gillespie et al. (2014) use different starting ages for the calculation of their threshold age. For comparison reasons, we choose the variant which starts directly at birth. Similar to the application by Zhang and Vaupel (2009), Gillespie et al. (2014) base their threshold age also on a proportional change in age-specific mortality. Accordingly, equation 14 results from deriving the variance of the age at death with respect to $\ln \mu_x$.

$$k_x = -2\mu_x \int_x^{\omega} l_a(a - e_0)\, da \tag{14}$$

In equation 14, l_a denotes the survival fraction at age a and e_0 is life expectancy at birth. The threshold age is then given by the root.

We calculated these ages by estimating the respective function empirically and searching for the root numerically. The procedure is similar to those applied for calculating MIRA quantities and the endpoint of the survival curve.

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7.4. Paper IV: The effects of increasing longevity and changing incidence on lifetime risk differentials: A decomposition approach

PLOS ONE

RESEARCH ARTICLE

The effects of increasing longevity and changing incidence on lifetime risk differentials: A decomposition approach

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Data Availability Statement: The authors confirm that, for approved reasons, some access restrictions apply to the disease-specific data underlying the findings. The incidence rates are based on national registers in Sweden and the datasets contain sensitive information. The restrictions of these data are imposed by the data owners, the National Board of Health and Welfare in Sweden (http://www.socialstyrelsen.se/english) and Statistics Sweden (http://www.scb.se/en_/). Therefore only the unidentifiable dataset that was used for the analyses presented in this paper is

Abstract

Increasing longevity can distort time trends in summary measures of health and mortality, such as the lifetime risk of getting diseased. If not observing a cohort, this lifetime risk is calculated with cross-sectional data on age-specific incidence and survival. In those instances, incidence and survival may work in opposite directions resulting in lifetime risk estimates where, reductions in incidence might be offset by a simultaneous longevity increase. The proposed method decomposes the difference between two lifetime risks into contributions of changing incidence and changing survival. The approach can be extended to measure the contributions of changes in disease related mortality and even case fatality. We illustrate the method with hypothetical examples as well as remaining lifetime risk at age 60 of experiencing a myocardial infarction, colorectal cancer and hip fractures for Swedish males. The empirical examples show that the influence of increasing longevity on the development of lifetime risk depends on the respective age profile of occurrence. In the cases of myocardial infarction and hip fracture, longevity increases of the general population counterbalanced or even exceeded the substantial gains in disease incidence, while for colorectal cancer, the lifetime risk was almost unaffected by the longevity improvement. This was because colorectal cancer has an on average earlier onset than myocardial infarction and hip fracture.

Introduction

Lifetime risk expresses the probability to develop a certain disease throughout lifetime or from a certain age onward (remaining lifetime risk). This makes it a useful indicator for monitoring the burden of a disease in a population. It is, for instance, widely applied in cancer research [1–4].

There is no common procedure for the calculation of lifetime risk. In many studies, it rests on longitudinal data for cohorts, such as the Rotterdam study, the Framingham Heart Study or population register data [5, 6]. Lifetime risk in these approaches refers to observed lifetimes

located in a secure server at the institution the authors are affiliated with. This dataset that the authors used is available upon request from the corresponding author, given that the person that is interested to use it can obtain approval from the data owners, the National Board of Health and Welfare in Sweden and Statistics Sweden. Interested researchers may also contact the data owners directly for access to the dataset. The used mortality data for the entire Swedish population can be downloaded from the Human Mortality Database(www.mortality.org).

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of individuals. However, lifetime risk has also been calculated based on cross-sectional data [1, 4, 7]. This approach is sometimes called "current probability method" [8]. In such a context, lifetime risk is based on observed disease and death patterns at a certain point in time. For at least two practical reasons, the use of cross-sectional lifetime risks are a valuable alternative to estimates based on longitudinal data. First, the data requirements are much lower for such a lifetime risk since no data with a sufficiently long follow-up time are necessary. Second, a cross-sectional lifetime risk summarizes the current burden in the population, whereas a lifetime risk based on longitudinal data summarizes past trends.

For the calculation of lifetime risk based on cross-sectional data, death and incidence rates for the respective period and population are required. Essentially by using standard life table techniques, these death and incidence rates are assumed to apply as if a real cohort would pass through time. The necessary assumption of time-invariant mortality and incidence are obviously false but since we are aiming to depict the current situation, this is, nevertheless, acceptable. Let us assume we have age-specific death and incidence rates of myocardial infarction for males aged 60 and older in a certain period. For this example, an interpretation of a lifetime risk would be: a 60 year-old male at the specific point in time has on average a remaining lifetime risk of, for instance, 30% to experience a myocardial infarction, if mortality and incidence stay constant over time.

The underlying risk population for a cross-sectional lifetime risk is usually drawn from a life table [1, 4, 8]. Accordingly, the two exit possibilities are the disease of interest and death as competing risk. The resulting lifetime risk estimate is based on the population age-structure of the life table, which can be regarded as a closed and stationary population, and which, solely depends on age-specific incidence and survival. Hence, differences in lifetime risk over time or between populations can arise from differences in incidence or in general survival. However, changes in just one of the two are unlikely in reality. In this instance, the interplay of survival and disease incidence can distort lifetime risk estimates and complicates meaningful comparisons of cross-sectional lifetime risks over time or between populations [1]. For example, a recent study has shown that the lifetime risk of hip fracture increased over time, despite declining age-specific incidence rates [7]. But how much of the change in lifetime risk can be attributed to each of the two factors?

In this paper, we aim to present a simple method to disentangle the two factors, allowing us to quantify how much of the change in lifetime risk is due to 1) changes in age-specific incidence and 2) changes in age-specific survival in the general population. We illustrate the method with an example based on artificial data and an application to empirical estimates of lifetime risk of myocardial infarction, hip fracture and colorectal cancer, using incidence and mortality of Swedish males.

Methods

Decomposing lifetime risk

Assuming that our incidence rates depict the first occurrence of the disease of interest, the rate of either dying or getting diagnosed at age x, μ_x, can be written as

$$\mu_x = m_x + I_x \tag{1}$$

where m_x is the death rate at age x and I_x is the incidence rate of getting diseased at age [1, 9]. For these estimation, death rates ideally refer to the disease-free population because they are the exit rate for those who die before getting diseased, whereas incidence covers the exit rate for those who get diseased before they die. In empirical application, however, these data requirements can not always be fulfilled and alternative death rates must be used (see data and

materials for further discussion). Note that death and incidence rates are referring to age intervals, which can also be bigger than one year of age. However, the length of the age-groups does not change the basic derivation of the decomposition method. To improve readability, we therefore excluded the notation of intervals throughout the manuscript. Moreover, the whole approach assumes constant rates within age intervals and also independence of age-specific death and incidence rates.

The probability of staying alive and healthy within one age interval x can be expressed by $exp[-\mu_x]$. Hence, the fraction alive and healthy at age x can be calculated by $exp[-\sum_{0\leq x_i<x} \mu_{x_i}]$, where x_i denotes the running index of the sum, which is age. The lifetime risk of becoming diseased from age x onward, lr_x, can then be calculated by

$$lr_x = \sum_{x\leq x_i\leq\omega} I_{x_i}\, exp[-\sum_{0\leq x_i<x} \mu_{x_i}], \tag{2}$$

where ω denotes the highest age attained. Eq 2 can be rewritten as

$$lr_x = \sum_{x\leq x_i\leq\omega} I_{x_i}\, exp[-\sum_{0\leq x_i<x} I_{x_i}]\, exp[-\sum_{0\leq x_i<x} m_{x_i}]. \tag{3}$$

For simplicity, we will write ϕ_{x_i} for $I_{x_i}\, exp[-\sum_{0\leq x_i<x} I_{x_i}]$ and l_{x_i} for $exp[-\sum_{0\leq x_i<x} m_{x_i}]$. Hence, Eq 3 changes to

$$lr_x = \sum_{x\leq x_i\leq\omega} \phi_{x_i} l_{x_i}. \tag{4}$$

We are interested in decomposing the change in lifetime risk to experience a given disease, denoted with Δ, between two time points A and B or, more generally, the difference between two populations:

$$\Delta = lr_{x,A} - lr_{x,B}. \tag{5}$$

The following methodological outline and derivation of the decomposition is based on previous studies, which provide general results for mathematical problems of such kind [10, 11]. Given the general definition of lifetime risk of Eq 4, we can rewrite Eq 5 to

$$\Delta = \sum_{x\leq x_i\leq\omega} \phi_{x_i,A} l_{x_i,A} - \sum_{x\leq x_i\leq\omega} \phi_{x_i,B} l_{x_i,B}. \tag{6}$$

Rearranging Eq 6 leads to

$$\Delta = \underbrace{\sum_{x\leq x_i\leq\omega} \left[l_{x_i,A} - l_{x_i,B}\right] \frac{\phi_{x_i,A} + \phi_{x_i,B}}{2}}_{\text{Contribution of Changing Survival Conditions}} + \underbrace{\sum_{x\leq x_i\leq\omega} \left[\phi_{x_i,A} - \phi_{x_i,B}\right] \frac{l_{x_i,A} + l_{x_i,B}}{2}}_{\text{Contribution of Changes in Incidence}}. \tag{7}$$

The omitted steps for deriving the expression in Eq 7 are listed in the S1 Appendix. Eq 7 provides now two distinct interpretable terms. The left term expresses the contribution of changing survival conditions to the difference in the lifetime risk between populations A and B. The right term expresses the contributions of changes in disease incidence to the difference in the lifetime risk between populations A and B. For both left and right term, age-specific differences in incidence and survival are standardized by average age-specific survival and incidence between both populations, respectively. This could be interpreted as if mortality or,

respectively, incidence would have been the same for both populations. Note in the S2 Appendix you find an extension of this decomposition, which additionally includes the mortality of the specific disease.

Hypothetical example

Table 1 presents three hypothetical examples to illustrate the decomposition as presented in Eq 7. In the first example (I), survival improves as reflected in the l_x–columns, while the incidence proportions are unchanged. The lifetime risk rose by 12 percentage points from $lr_A = 0.24$ to $lr_B = 0.36$. Because we subtracted B from A we obtained a negative value. The contribution to the increase in the lifetime risk from changes in incidence proportions (last column) is obviously zero since $i_{A,x}$ and $i_{B,x}$ do not differ at any age. As expected, the difference in lifetime risk can be completely attributed to improvements in survival.

The complementary picture is provided by the second example (II). Age-specific survival does not differ between time points A and B. Instead, the age-specific incidence proportions changed over time. The overall reduction in lifetime risk of 0.07 is therefore equivalent to the sum of the contributions from the different age categories.

Although all examples are hypothetical, the third example (III) is probably closest to reality because contributions to lifetime risk differences originate from changes in age-specific survival as well as from changes in age-specific incidence proportions. The lifetime risk increased by 15 percentage points from 0.33 to 0.48. Our decomposition method allows us to disentangle the overall effect into contributions due to varying mortality conditions and varying age-specific incidence. It turns out that the increase in lifetime risk is due to a combination of higher incidence proportions and higher survival. In addition to this qualitative assessment, we can also state that improved survival contributed almost three times more (0.11) to the increase in lifetime risk than the actual incidence risk (0.04).

Table 1. Illustration of the decomposition method with hypothetical examples. (I) Changes in age-specific survival. (II) Changes in age-specific incidence. (III) Changes in age-specific survival and incidence. In all three examples, we decomposed the change from A to B ($A - B$).

						Contribution of Change in Age-Specific	
Scenario	**Age x**	$l_{A,x}$	$l_{B,x}$	$\phi_{A,x}$	$\phi_{B,x}$	**Survival**[†]	**Incidence**[‡]
(I) Change in Survival	1	1.0	1.0	0.0	0.0	0.00	0
	2	0.7	0.8	0.2	0.2	-0.02	0
	3	0.2	0.4	0.3	0.3	-0.06	0
	4	0.1	0.2	0.4	0.4	-0.04	0
$lr_A = 0.24$ $lr_B = 0.36$ $\Delta = -0.12$					Σ	-0.12	0
(II) Change in Incidence	1	1.0	1.0	0.0	0.0	0	0.00
	2	0.7	0.7	0.2	0.1	0	0.07
	3	0.2	0.2	0.3	0.4	0	-0.02
	4	0.1	0.1	0.4	0.2	0	0.02
$lr_A = 0.24$ $lr_B = 0.17$ $\Delta = 0.07$					Σ	0	0.07
(III) Changes in Survival **and** Incidence	1	1.0	1.0	0.1	0.2	0.000	-0.100
	2	0.5	0.8	0.2	0.1	-0.045	0.065
	3	0.3	0.4	0.3	0.4	-0.035	-0.035
	4	0.1	0.2	0.4	0.2	-0.030	0.030
$lr_A = 0.33$ $lr_B = 0.48$ $\Delta = -0.15$					Σ	-0.11	-0.04

[†] Estimated by first part of Eq 7.

[‡] Estimated by second part of Eq 7.

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Empirical examples

Material and data

We applied the method to remaining lifetime risk of getting diagnosed with myocardial infarction, colorectal cancer and hip fracture for Swedish males at age 60. Note, however, that the decomposition can be applied to lifetime risk starting at any age. For the different diseases, we compared the lifetime risk between two different time points. These are 1987 and 1994 for colorectal cancer, 1994 and 2014 for hip fractures and 1994 and 2004 for myocardial infarction.

The incidence estimates for the three disease outcomes were obtained from Swedish registry data maintained by Statistics Sweden and the National Board of Health and Welfare [12]. For myocardial infarction and hip fracture, the first event occurring after age 60 after a seven year disease free period was identified from the National Patient Register. For colorectal cancer, the date of the diagnosis of the cancer was collected from the Swedish Cancer Register. The incidence counts have been smoothed across age and time to reduce random fluctuations, using the MortalitySmooth package in R [13, 14].

Death—the competing risk of getting diseased— should ideally be measured by the death rates of the disease-free population. This requires sufficiently detailed data sources, which allow to follow individuals over time, in order to determine whether they can be considered disease-free or not. In most cases, however, such detailed data are not available, and therefore, alternative data sources must be used. In our example, death rates are based on death counts and population data of the total Swedish male population, provided by the Human Mortality Database [15]. For two reasons, these death rates are very likely overestimating mortality in a cross-sectional setting. First, persons who get diseased and die in the same year are considered in the incidence as well as in the death rate and are therefore counted as two separate exits. Second, in our quasi-cohort perspective, death rates at consecutive ages are also potentially too high because total population death rates still contain the diseased population. It is very likely that this population suffers from higher mortality than the disease-free population. For practical reasons, however, the use of such death rates is acceptable because this misclassification likely overestimates mortality, and thus, leads to upper bound estimates of lifetime risk and the contribution of increased survival. Note that the bias of using such death rates depends on the respective relationship between mortality and the disease of interest.

The left panel of Fig 1 depicts the time trends of lifetime risk for the respective diseases. The dots mark the time points, which are selected for the decomposition. The selection is based on the pattern of the respective lifetime risks. Accordingly, for myocardial infarction, we choose the end points of a period with almost stagnating levels of lifetime risk. For hip fracture, both time points comprise a period of slight increases and for colorectal cancer, the time points are the boundaries of a period of increasing lifetime risk. The right panel illustrates the results of the decomposition. The bars show the contribution of changing survival and of changing incidence as well as the total change in lifetime risk.

Results

In the case of myocardial infarction, the lifetime risks are almost similar at the two time points, which could lead to the conclusion that there have been no improvements in incidence above age 60. By applying the decomposition, however, it becomes clear that declining incidence would have generated a decrease in lifetime risk by more than three percentage points but increasing longevity prevented this decline. If only mortality would have changed between the two time points, the mortality improvements and, hence, the higher number of survivors to

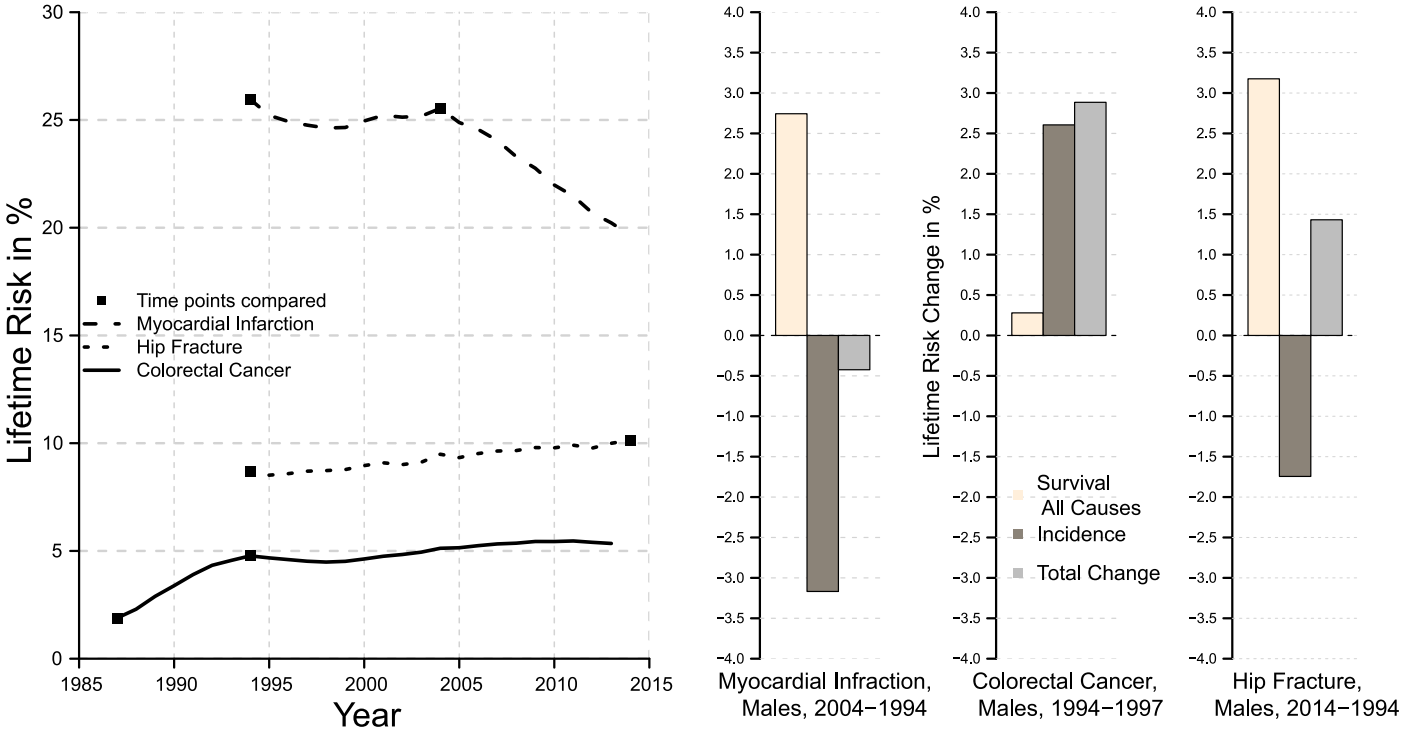

Fig 1. Remaining lifetime risk at age 60 and lifetime risk decomposition for myocardial infarction, hip fracture and colorectal cancer, Sweden, males.

https://doi.org/10.1371/journal.pone.0195307.g001

older ages would have resulted in an increase of lifetime risk by more than 2.5 percentage points. In sum, the counteracting factors resulted in an overall change of lifetime risk by less than a half percentage point.

For hip fracture, we observe a slight but steady increase in remaining lifetime risk above age 60 over time. This rise, however, is entirely driven by increasing longevity. The declining incidence, given the same mortality at both time points, would have contributed to a decrease of lifetime risk by more than 1.5 percentage points, whereas the survival improvements, given the same incidence at both time points, would have generated an increase by more than 3 percentage points. Accordingly, the total change sums up to an increase by almost 1.5 percentage points.

Colorectal cancer is an example, where lifetime risk increases relatively steeply in a short period of time. In contrast to the other two examples, incidence contributes to a rise in the remaining lifetime risk at age 60. Consequently, the incidence between 1987 and 1994 has increased. Rising incidence alone would have generated an increase of lifetime risk by more than 2.5 percentage points. Also the role of improving survival for changes in the lifetime risk are different compared to the other two examples. The on average earlier occurrence of colorectal cancer lowered the impact of the longevity advancement. Hence, the contribution of increasing survival, given the same incidence at both time points, would have resulted in an increase by slightly more than 0.25 percentage points. The total change, thus, sums up to a rise of lifetime risk by almost 3 percentage points.

Discussion and conclusion

In this paper, we presented a decomposition method, which allows to separate the contribution of changing incidence and changing survival on the difference between two lifetime risks. The

method can be applied to compare lifetime risks over time or between different populations. We illustrated the method and the interpretation of its estimates using hypothetical as well as empirical data for three different diseases, which differ in their development of lifetime risk over time.

In the analysis of time trends of the incidence of a certain disease, lifetime risk may be a suitable summary measure across age categories or lifespan. In such an application, it is conceivable that the incidence decreases with time in parallel with a reduction in death risks, which in turn determines survival. This is the situation in which the change in incidence forces lifetime risk to go down, while the change in survival forces lifetime risk to go up. Thus, the two forces work in opposite directions and a positive development of the risk of getting a disease may be hidden but can be excavated by a decomposition method. Of course, other approaches such as age-standardization are conceivable to investigate the change in incidence but such methods would ignore the quantification of the influence of improved survival. In this context, lifetime risk provides a valuable, perhaps unique, opportunity to combine information on survival and incidence within one single index. The presented decomposition method allows to investigate incidence change and additionally survival change at the same time by comparing directly the observed age-specific incidence and death rates, thereby omitting the use of some arbitrary standard population.

As we have seen from the empirical examples, whether increasing longevity influences the development of lifetime risk or not, depends on the timing of the respective disease. In recent decades, mortality improvements have been especially prevalent at higher ages [16]. Accordingly, especially lifetime risks of diseases, which tend to occur at higher ages, are influenced by increasing survival. This is because improved survival causes a higher number of survivors to the ages where disease incidence is highest and, hence, even when incidence is going down, the declines at those ages are at least compensated by the higher number of persons under risk. In turn, for disease which tend to occur at ages with only marginal survival improvements, incidence declines of the same magnitude would have a stronger effect on lifetime risk. That is because of the number of survivors at that ages, which differs only marginally between the two populations.

Furthermore, it is worth noting that for fatal diseases there is a certain degree of dependency between the disease risks and the death risks. However, as we show in the appendix the method could easily be extended to capture this dependency. In this case, it is a three factor decomposition, which separates, besides the contributions of survival and incidence, the contribution of changes in disease related mortality to incorporate the interrelation of declining incidence as a potential driver of increasing longevity over time. In fact, the approach could even be extended to a four factor decomposition, which could additionally provide the contribution of changing case fatality.

Many common summary measures of population health and mortality are distorted by the increase in longevity, or more generally by the change of the age-structure of the underlying population. The general procedure presented here can add to our understanding of the influences of these factors on such measures. Practically, these decompositions could also be important in forecasting and planning health care resources in the future.

Supporting information

S1 Appendix. Mathematical derivation of the method: Omitted steps.
(PDF)

S2 Appendix. Extending the decomposition by the disease related mortality rate: Equations and illustrative example.
(PDF)

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Visualization: Marcus Ebeling.

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The effects of increasing longevity and changing incidence on lifetime risk differentials: A decomposition approach (Appendix 1)

Marcus Ebeling[*], Karin Modig[†], Anders Ahlbom[‡] and Roland Rau[§]

Mathematical derivation of the method: omitted steps

In the main text, we have not shown the full derivation of the decomposition method. The steps to derive the final decomposition formula are shown below. Starting from the equation

$$\Delta = \sum_{x \le x_i \le \omega} \phi_{x_i,A} l_{x_i,A} - \sum_{x \le x_i \le \omega} \phi_{x_i,B} l_{x_i,B}. \tag{1}$$

we split the two components in the first line of Eq 2 in halves, and add and subtract identical terms in the second line of Eq 2. We do this in order to end up with easily interpretable terms.

$$\begin{aligned}\Delta = &\frac{\sum_{x \le x_i \le \omega} l_{x_i,A}\phi_{x_i,A}}{2} + \frac{\sum_{x \le x_i \le \omega} l_{x_i,A}\phi_{x_i,A}}{2} - \frac{\sum_{x \le x_i \le \omega} l_{x_i,B}\phi_{x_i,B}}{2} - \frac{\sum_{x \le x_i \le \omega} l_{x_i,B}\phi_{x_i,B}}{2} \\ &+ \frac{\sum_{x \le x_i \le \omega} l_{x_i,A}\phi_{x_i,B}}{2} - \frac{\sum_{x \le x_i \le \omega} l_{x_i,A}\phi_{x_i,B}}{2} + \frac{\sum_{x \le x_i \le \omega} l_{x_i,B}\phi_{x_i,A}}{2} - \frac{\sum_{x \le x_i \le \omega} l_{x_i,B}\phi_{x_i,A}}{2}.\end{aligned} \tag{2}$$

By grouping, we can reduce Eq 2 to four terms:

$$\begin{aligned}\Delta = &\sum_{x \le x_i \le \omega} l_{x_i,A} \frac{\phi_{x_i,A} + \phi_{x_i,B}}{2} + \sum_{x \le x_i \le \omega} \phi_{x_i,A} \frac{l_{x_i,A} + l_{x_i,B}}{2} \\ &- \sum_{x \le x_i \le \omega} l_{x_i,B} \frac{\phi_{x_i,A} + \phi_{x_i,B}}{2} - \sum_{x \le x_i \le \omega} \phi_{x_i,B} \frac{l_{x_i,A} + l_{x_i,B}}{2}.\end{aligned} \tag{3}$$

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Eq 3 can now be rewritten to get the decomposition formula

$$\Delta = \underbrace{\sum_{x \le x_i \le \omega} \left[l_{x_i,A} - l_{x_i,B} \right] \frac{\phi_{x_i,A} + \phi_{x_i,B}}{2}}_{\text{Contribution of Changing Survival Conditions}} + \underbrace{\sum_{x \le x_i \le \omega} \left[\phi_{x_i,A} - \phi_{x_i,B} \right] \frac{l_{x_i,A} + l_{x_i,B}}{2}}_{\text{Contribution of Changes in Incidence}}. \tag{4}$$

The effects of increasing longevity and changing incidence on lifetime risk differentials: A decomposition approach (Appendix 2)

Marcus Ebeling[*], Karin Modig[†], Anders Ahlbom[‡] and Roland Rau[§]

Extending the decomposition by the disease related mortality rate: equations and illustrative example

To investigate and illustrate the relationship between declining incidence of the respective disease and its role as a potential driver of increasing longevity over time, we incorporate disease-related mortality as a third factor to the decomposition. If we are including the death rate of the respective disease at age x, d_x, the rate of either dying or getting diagnosed at age x changes to

$$\mu_x = m_x + I_x + d_x, \tag{1}$$

where, for interpretation reasons, m_x now depicts the death rate for all other causes than the disease in question. By applying this modification, the calculation of lifetime risk changes to

$$lr_x = \sum_{x \leq x_i \leq \omega} I_{x_i}\, exp[- \sum_{x \leq y < x_i} I_y]\, exp[- \sum_{x \leq y < x_i} m_y]\, exp[- \sum_{x \leq y < x_i} d_y]. \tag{2}$$

For simplicity, we will write again ϕ_{x_i} for $I_{x_i}\, exp[- \sum_{x \leq y < x_i} I_y]$ and l_{x_i} for $exp[- \sum_{x \leq y < x_i} m_y]$ and, furthermore, $\lambda_{x,i}$ for $exp[- \sum_{x \leq y < x_i} d_y]$. Given these changes, the original decomposition formula is extended by a third term, which indicates the contribution of changes in disease-specific

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mortality between both populations. Accordingly, the formula writes as follows

$$\begin{aligned}
&\sum_{x\le x_i\le\omega}\phi_{x_i,A}l_{x_i,A}\lambda_{x_i,A}-\sum_{x\le x_i\le\omega}\phi_{x_i,B}l_{x_i,B}\lambda_{x_i,B}= \\
&\underbrace{\sum_{x\le x_i\le\omega}(\phi_{x_i,A}-\phi_{x_i,B})\left[\frac{l_{x_i,A}\lambda_{x_i,A}+l_{x_i,B}\lambda_{x_i,B}}{3}+\frac{l_{x_i,A}\lambda_{x_i,B}+l_{x_i,B}\lambda_{x_i,A}}{6}\right]}_{\text{Contribution of Changing Incidence Risks}} \\
&+\underbrace{\sum_{x\le x_i\le\omega}(l_{x_i,A}-l_{x_i,B})\left[\frac{\phi_{x_i,A}\lambda_{x_i,A}+\phi_{x_i,B}\lambda_{x_i,B}}{3}+\frac{\phi_{x_i,A}\lambda_{x_i,B}+\phi_{x_i,B}\lambda_{x_i,A}}{6}\right]}_{\text{Contribution of Changing Survival Conditions (except Disease)}} \\
&+\underbrace{\sum_{x\le x_i\le\omega}(\lambda_{x_i,A}-\lambda_{x_i,B})\left[\frac{\phi_{x_i,A}l_{x_i,A}+\phi_{x_i,B}l_{x_i,B}}{3}+\frac{\phi_{x_i,A}l_{x_i,B}+\phi_{x_i,B}l_{x_i,A}}{6}\right]}_{\text{Contribution of Changing Disease Related Survival}}
\end{aligned} \tag{3}$$

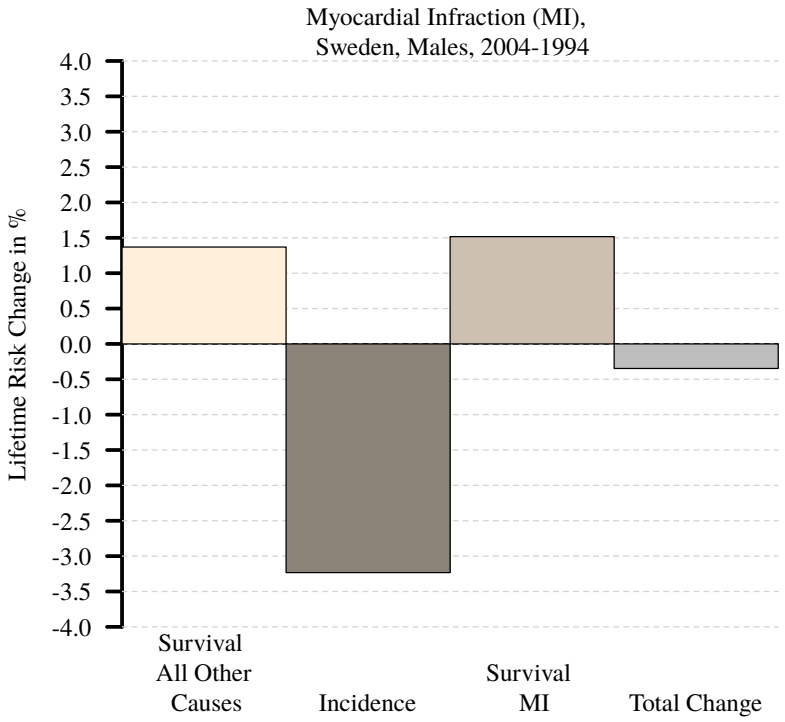

Figure 1: **Decomposition of Lifetime Risk at Age 60 for Myocardial Infarction, Sweden, Males, 1994-2004.**

Fig. 1 shows an application of the three factor decomposition to the example of lifetime risk for myocardial infarction for Swedish males, comparing the years 1994 and 2004. During

this period, lifetime risk almost stagnated, which could lead to the presumption that there has been no improvement in the incidence of getting a myocardial infarction. By applying the three factor decomposition, however, we see that the opposite is true. Not only incidence declined, also the mortality of myocardial infarction improved. Given the same mortality for myocardial infarction and all other causes, the contribution of changing incidence between both time points would have resulted in a decrease of lifetime risk by more than 3 percentage points. However, the improved survival for both myocardial infarction and all other causes prevented this decline. Hence, given the same incidences and mortality of myocardial infarction at both time points, the contribution of increased survival for all other causes would have resulted in a rise of lifetime risk by more than 1.25 percentage points. Additionally, improved mortality for myocardial infarction would have resulted in an increase by almost 1.5 percentage points. In sum, the three factors add up to a total change of less than -0.5 percentage points.

The effects of increasing longevity and changing incidence on lifetime risk differentials: A decomposition approach

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Correction

We noticed an incorrect summation index that appears in some equations. This error has no effect on the content of the article. By recalculating our empirical examples with the revised formulas, we noticed only minor changes in the empirical outcomes of our decomposition method. The magnitude of the changes does not require any adjustments of the text. In fact, they even strengthen the message of our paper. In conclusion, the incorrect summation index requires the following corrections:

1. revise the respective formulas in the original paper
2. upload a revised S2 Appendix
3. exchange Figure 1 with a revised figure

1. revise the respective formulas in the original paper

Original text (page 3 in the original manuscript):

> Hence, the fraction alive and healthy at age x can be calculated by $exp[-\sum_{0\leq x_i<x} \mu_{x_i}]$, where x_i denotes the running index of the sum, which is age. The lifetime risk of becoming diseased from age x onward, lr_x, can then be calculated by
>
> $$lr_x = \sum_{x\leq x_i\leq\omega} I_{x_i}\, exp[-\sum_{0\leq x_i<x} \mu_{x_i}], \tag{2}$$
>
> where ω denotes the highest age attained. Eq 2 can be rewritten as
>
> $$lr_x = \sum_{x\leq x_i\leq\omega} I_{x_i}\, exp[-\sum_{0\leq x_i<x} I_{x_i}]\, exp[-\sum_{0\leq x_i<x} m_{x_i}]. \tag{3}$$
>
> For simplicity, we will write ϕ_{x_i} for $I_{x_i}\, exp[-\sum_{0\leq x_i<x} I_{x_i}]$ and l_{x_i} for $exp[-\sum_{0\leq x_i<x} m_{x_i}]$.

Corrections/track changes of the respective part (changes are highlighted in blue):

Hence, the fraction alive and healthy at age x_i can be calculated by $exp[-\sum_{x \le y < x_i} \mu_y]$; ~~where x_i denotes the running index of the sum, which is age~~. The lifetime risk of becoming diseased from age x onward, lr_x, can then be calculated by

$$lr_x = \sum_{x \le x_i \le \omega} I_{x_i} \, exp[-\sum_{x \le y < x_i} \mu_y], \tag{2}$$

where ω denotes the highest age attained. Eq 2 can be rewritten as

$$lr_x = \sum_{x \le x_i \le \omega} I_{x_i} \, exp[-\sum_{x \le y < x_i} I_y] \, exp[-\sum_{x \le y < x_i} m_y]. \tag{3}$$

For simplicity, we will write ϕ_{x_i} for $I_{x_i} \, exp[-\sum_{x \le y < x_i} I_y]$ and l_{x_i} for $exp[-\sum_{x \le y < x_i} m_y]$.

2. upload a revised S2 Appendix

The incorrect summation index appears also in the S2 appendix. Therefore, this document requires also minor revisions. Please see the attached document.

3. exchange Figure 1 with a revised figure

Figure 1 shows the graph as printed in the published article. Figure 2 depicts the revised version of the graph after adjusting for the incorrect summation index.

The following changes occurred after the adjustment for the incorrect summation index:

- myocardial infarction
  - slight increase in the lifetime risk estimates
- colorectal cancer
  - slight increase in the lifetime risk estimates, the total change between the two lifetime risks compared, and the contributions of changes in disease incidence
- hip fracture
  - slight increase in the lifetime risk estimates, the total change in the two lifetime risks compared, and the contribution of changes in survival
  - slight decline in the contribution of changes in disease incidence

The outlined changes do not influence the outcomes of the article and do not require any editing of the text in the manuscript.

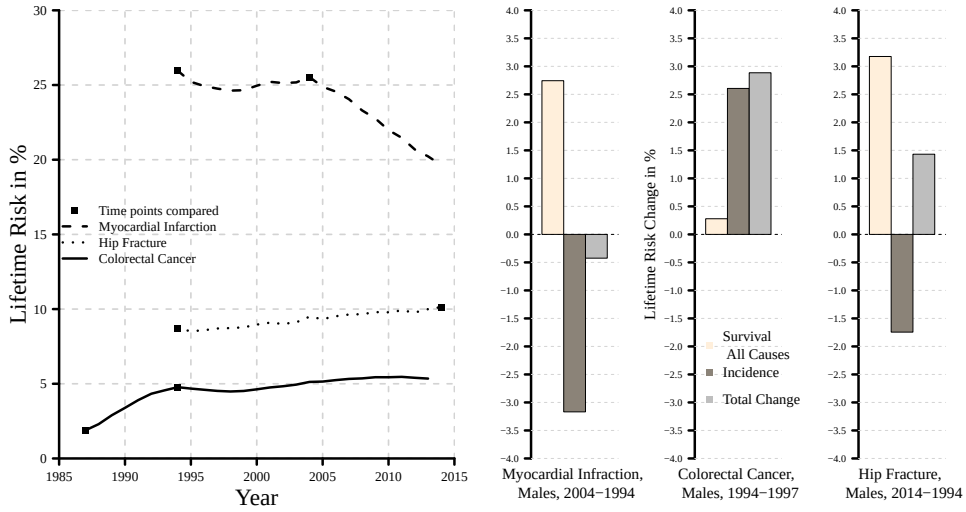

Figure 1: Figure 1 OLD

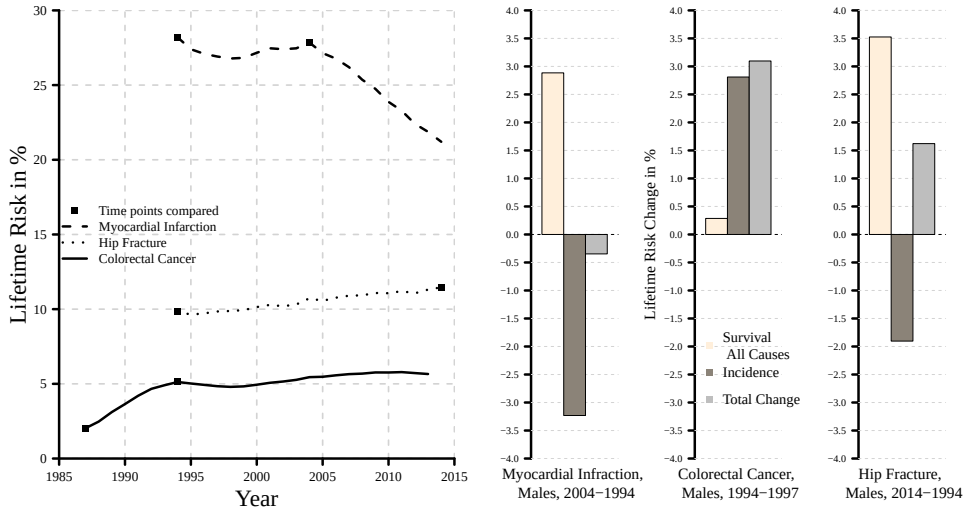

Figure 2: Figure 1 NEW/REVISED

7.5. Paper V: Lifespan disparity as an additional indicator for evaluating mortality forecasts

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Lifespan Disparity as an Additional Indicator for Evaluating Mortality Forecasts

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Abstract Evaluating the predictive ability of mortality forecasts is important yet difficult. Death rates and mean lifespan are basic life table functions typically used to analyze to what extent the forecasts deviate from their realized values. Although these parameters are useful for specifying precisely how mortality has been forecasted, they cannot be used to assess whether the underlying mortality developments are plausible. We therefore propose that in addition to looking at average lifespan, we should examine whether the forecasted variability of the age at death is a plausible continuation of past trends. The validation of mortality forecasts for Italy, Japan, and Denmark demonstrates that their predictive performance can be evaluated more comprehensively by analyzing both the average lifespan and lifespan disparity—that is, by jointly analyzing the mean and the dispersion of mortality. Approaches that account for dynamic age shifts in survival improvements appear to perform better than others that enforce relatively invariant patterns. However, because forecasting approaches are designed to capture trends in average mortality, we argue that studying lifespan disparity may also help to improve the methodology and thus the predictive ability of mortality forecasts.

Keywords Evaluation · Forecasting performance · Lifespan disparity · Average lifespan

Electronic supplementary material The online version of this article (doi:10.1007/s13524-017-0584-0) contains supplementary material, which is available to authorized users.

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Introduction

The aim of most mortality forecasts is to predict how many additional years of life people will gain in the future. Basic life table functions—such as life expectancy at birth (a measure of central tendency) and age-specific death rates (measures of mortality intensity) are usually applied to evaluate the precision of such forecasts. The closer a forecast is to the observed development, the greater is its forecasting performance—or, interchangeably, its predictive ability. Goodness-of-fit tests as well as validation procedures are typically used to evaluate the predictive ability of mortality forecasts. Placing particular emphasis on *ex post* quantitative aspects (Armstrong and Collopy 1992; Cairns et al. 2011b; Keilman 1997; Shang 2015), conventional evaluation measures quantify the difference between predicted and observed mortality. It is commonly considered that the greater such forecast errors, the poorer is the forecasting performance. However, although deviations are supposed to be small, zero deviations would indicate overfitting rather than a good forecasting performance. Forecast errors can be expressed in absolute or relative terms, and they can be averaged over dimensions such as age, time, and population (Booth et al. 2006; Keilman and Pham 2004; Koissi et al. 2006; Shang et al. 2011; Smith et al. 2001). The meaning of these errors changes in each case. For example, means of absolute errors measure accuracy, whereas means of positive and negative errors measure bias—that is, systematic over- or underestimation. Relative errors deal with scale dependency and therefore allow comparison of errors across measures and methods. Dowd et al. (2010); Koissi et al. (2006), and Lee and Miller (2001) analyzed how errors or (standardized) residuals are distributed. In addition to employing visualization techniques, these authors used statistical tests such as chi-squared, Levene's test, the variance ratio test, or the Jarque-Bera normality test. Moreover, Shang (2015) recently proposed using test statistics to reveal significant differences in the forecast accuracy of point and interval estimates as well as differences between the forecasts of multiple approaches.

Errors and test statistics of basic life table functions are useful for specifying precisely how mortality has been forecasted. However, small errors in the forecasts of average lifespan do not necessarily indicate that the forecasted underlying mortality developments are plausible. Figure 1 illustrates this issue in more detail with a scatterplot that displays the negative correlation between life expectancy at birth and lifespan disparity measured by average life years lost at birth, $e_0^\dagger$ (e.g., Vaupel and Canudas-Romo 2003), for women in Italy, Denmark, and Japan from 1950 to 2012. In contrast to basic life table measures, $e_0^\dagger$ provides information about the underlying mortality developments. Although life expectancy at birth has increased in recent decades because of reductions in mortality at progressively higher ages, $e_0^\dagger$ has decreased mainly as a result of survival improvements at premature ages, which shifted deaths toward the end of the lifespan. Figure 1 shows a striking pattern: the average lifespan of Italian, Danish, and Japanese women has been similar in recent decades, whereas the decline in the variability of the age at death differed considerably among these groups of women as soon as their average lifespan exceeded 75 years. Specifically, lifespan dispersion (1) declined regularly for Italian women, (2) leveled off for Japanese women, and (3) increased and decreased for Danish women.

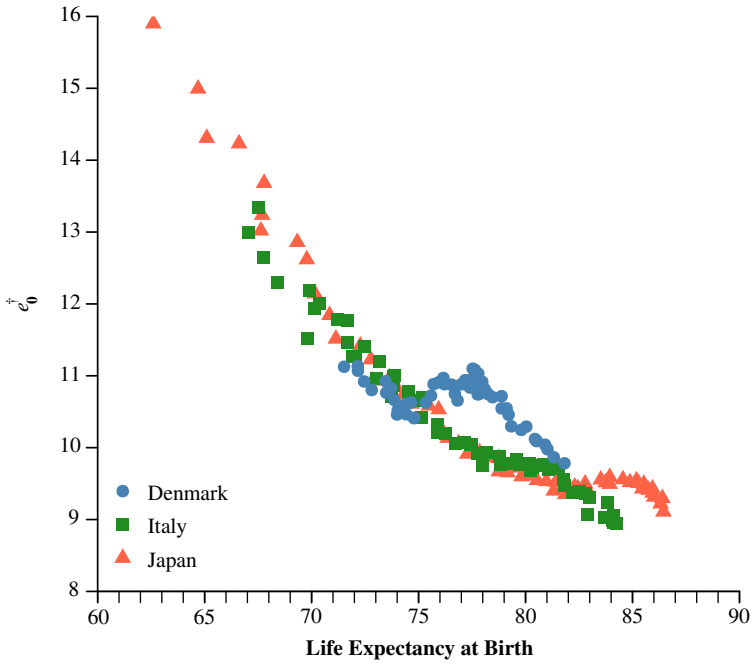

Fig. 1 Scatterplot of life expectancy at birth and average life years lost at birth due to death for women in Denmark, Italy, and Japan from 1950 to 2012

These findings illustrate that different underlying mortality trajectories can lead to similar average lifespans and lifespan disparities. Other researchers have discussed this relationship in detail (e.g., Smits and Monden 2009; Vaupel et al. 2011; Wilmoth and Horiuchi 1999). For example, Wilmoth and Horiuchi (1999) showed that different levels of life expectancy at birth can come across with different levels of lifespan dispersion. Goldstein and Cassidy (2012) and Bergeron-Boucher et al. (2015) analyzed the impact of changing slopes in the mortality age profile on life expectancy at birth and lifespan dispersion. For instance, Goldstein and Cassidy concluded that changes in the slope have a relatively larger effect on life expectancy at birth than changes in the level of mortality. To ensure that we take these underlying trajectories into account, we propose expanding the toolkit of conventional evaluation procedures. Basic life table functions should be complemented by measures of lifespan dispersion to improve the assessment of *ex post* quantitative aspects and to evaluate the plausibility of underlying mortality trends. To the best of our knowledge, only Cairns et al. (2006, 2011b) have taken a similar approach. They added an *ex ante* evaluation with qualitative criteria to *ex post* measures; that is, they examined the forecasting performance using qualitative criteria, such as the biological validity of the age schedule of mortality, and investigated the consistency of the forecasts using historical data. However, as far as we know, no existing studies have used lifespan disparity as an evaluation measure for the plausibility of mortality forecasts. The objective of this article is to highlight the necessity to assess whether mortality forecasting methods can capture and forecast different trends of life expectancy at birth and lifespan disparity—that is, expose the benefits of incorporating lifespan disparity as an additional indicator in the toolkit that is used to evaluate the performance of mortality forecasts.

Lifespan Disparity: Measures and Concepts

Lifespan disparity describes the variation in the lifespan distribution—that is, the differences in the length of life across members of a population. A wide range of approaches can be used to measure lifespan disparity, including (1) classic statistical variability measures, such as the standard deviation or the interquartile range; (2) equality measures, such as the Gini coefficient; or (3) geometric approaches, such as the Prolate index (Cheung et al. 2005; Eakin and Witten 1995; Kannisto 2000; Wilmoth and Horiuchi 1999). However, because all these measures are highly correlated (Vaupel et al. 2011; Wilmoth and Horiuchi 1999), we can expect that their impact on the results would be minor. Although those measures are highly correlated, however, their trends may differ. For example, if equality were rising, measures of variability would decrease, whereas measures of rectangularity would increase (Wilmoth and Horiuchi 1999).

To measure lifespan dispersion,[1] we take the average number of life years lost at birth (Vaupel and Canudas-Romo 2003; Zhang and Vaupel 2009), $e_0^\dagger$, estimated by

$$e_0^\dagger = \frac{\int_0^\omega e_a d_a da}{l_0}, \tag{1}$$

with e_a being remaining life expectancy at age a, and d_a being life table deaths at age a, with both integrated from age 0 to ω, the highest age at death. l_0 is the radix of the life table. A major reason why we chose $e_0^\dagger$ is that it is demographically interpretable as the average life years lost. Because $e_0^\dagger$ refers to the lost living potential, it also provides information about the capacity for further increases in life expectancy. We argue that these key features enable $e_0^\dagger$ in particular to be used to evaluate the plausibility of mortality forecasts.

Measuring lifespan disparity may reveal one of three general patterns: the compression, shifting, or expansion of mortality. Although these patterns are not mutually exclusive in the real world, they are useful for explaining trends in lifespan disparity. Fries (1980) established the concept of mortality compression, originally describing a postponement of mortality to some fixed upper lifespan limit, which in turn induces a reduction in lifespan disparity. Although the expected levels of lifespan disparity have not been reached and the proposed levels of average lifespan have been exceeded, the concept of mortality compression is typically used to describe the massive reductions in lifespan variability since the mid-nineteenth century (Kannisto 2000; Nagnur 1986; Nusselder and Mackenbach 1996). The concept of shifting mortality describes a postponement of the old-age death bulk toward higher ages with an approximately constant level of lifespan variability. Empirical studies have provided evidence that shifting mortality may occur following mortality compression (e.g., Bongaarts 2005; Canudas-Romo 2008; Kannisto 1996). The concept of mortality expansion refers to progressive improvements in survival to very old ages that have not previously been

[1] We use the terms "lifespan disparity," "lifespan dispersion," and "lifespan variability" interchangeably. Some scholars also call $e_0^\dagger$ "life disparity" (Vaupel et al. 2011). However, we use the term "lifespan disparity" to describe the general concept of lifespan variability that we measure using average life years lost.

reached by many people. Mortality expansion to very old ages induces temporarily increasing lifespan variability, although its impact on total variability of the age at death has not been evident until recently. However, increasing lifespan dispersion has been observed in multiple populations at approximately age 60 (Engelman et al. 2010, 2014; Rothenberg et al. 1991).

A (positive or negative) change in life expectancy at birth, along with a compression, a shifting, and/or an expansion of mortality, are possible developments that should be captured in a mortality forecast. Because these developments are closely related and occur at different times in different populations, mortality forecasting approaches may need to be adjusted to ensure that they are captured appropriately.

Mortality Forecasting Approaches That Tackle Variability of the Age at Death

Concise Overview

Many approaches, like the canonical Lee-Carter model (1992), extrapolate past trends while assuming that the relative progress in mortality made among people of different ages has been time-invariant. This assumption is, however, implausible, given that survival improvements differ considerably by age over time. In the first half of the twentieth century, large reductions in mortality occurred among infants and young children in many highly developed countries. More recently, most of the survival improvements have been among adults and the elderly. In the coming decades, mortality is expected to decline mainly among the very old. Hence, the assumption of time-invariant changes in mortality by age may induce forecasts that are prone to errors. Recently developed approaches respond to this problem in different ways. For example, Janssen and de Beer (2016) accounted for the distribution of the age at death; Li et al. (2013) rotated the age pattern of mortality change with time; and Haberman and Renshaw (2012), Mitchell et al. (2013), and Bohk-Ewald and Rau (2017) used rates of mortality improvement instead of death rates to forecast dynamic age shifts in mortality decline. Moreover, Li and Lee (2005), Cairns et al. (2011a), Hyndman et al. (2013), and others used coherent approaches to jointly forecast mortality among multiple populations, allowing populations to adapt their below- or above-average increases in life expectancy to a shared trend among multiple populations. Capturing ruptures in long-term trends that emerge from irregular patterns of mortality change is also challenging. For example, Coelho and Nunes (2011) dealt with long-term trend changes in mortality forecasts, Janssen et al. (2013) included exogenous variables such as tobacco smoking, and Renshaw and Haberman (2006) considered cohort mortality to account for this issue.

We select three of these approaches, which differ in their ability to capture dynamic age shifts in survival improvement, to forecast mortality exemplarily for women in Italy, Japan, and Denmark up to 2009 (see the next section). These models are the Lee-Carter model, its rotating variant developed by Li et al., and the model developed by Bohk-Ewald and Rau. Given that their levels of modeling flexibility differ, each approach models the various trends in lifespan disparity in the three populations differently (see Fig. 1). All the approaches mentioned in the concise overview are equally qualified to be

selected for the case studies to show the advantages when evaluating the forecasting performance using the mean—and, as an extra criterion, the spread of mortality. Hence, this analysis is designed to show the additional information that can be gained when evaluating the forecasted spread of mortality in the presence of different trends for life expectancy at birth and lifespan dispersion. Although the case studies provide some results for comparing the forecasting performance of the three approaches, this should be considered preliminary and rather a byproduct than an incentive to conduct this analysis; a valid model comparison would instead require a systematic evaluation of the forecasting performance using extensive mortality data of multiple countries and periods, which is beyond the scope of this work. Hence, for the case studies, we select three forecasting models that cover the range of available approaches and modeling strategies quite well. In addition to describing the method-based assumptions of the selected approaches for capturing dynamic age shifts in survival improvement, as well as some details on implementation, we offer hypotheses regarding the effect that each approach might have on the forecasted mean lifespan and lifespan disparity.

Impact of Model-Based Assumptions on Lifespan Disparity

Lee-Carter Model

Although the Lee-Carter model has been used and revised extensively since it was first developed in 1992 (Booth and Tickle 2008; Booth et al. 2006; Butt and Haberman 2010; Shang 2012; Shang et al. 2011), we use its original version as a benchmark in our case studies. The Lee-Carter model forecasts mortality by age and calendar year on the logarithmic scale while assuming that the relative changes in mortality were constant between the ages over time. Hence, if the survival improvements were relatively large at young ages and small at old ages in the reference years, this proportion would assumedly be unchanged in the forecast years. Yet, given the shifts by age in survival improvements over time, we hypothesize that the inflexibility in the age profile of mortality change would have affected the Lee-Carter forecasts up to 2009. The extrapolation of declining mortality at infant, child, adult, and old ages based solely on the mortality trends observed in the reference period may result in a reliable forecast for the near future given that the prevalence of mortality reductions at very old ages will still be low. However, if mortality continues to decline at progressively higher ages in the coming decades, the Lee-Carter model may produce forecasts that fail to capture correctly both the average lifespan and lifespan disparity. The absence of a dynamic shift in survival improvements to progressively higher ages may then induce (1) an underestimation of life expectancy at birth as well as (2) a strong compression of deaths, which may in turn be accompanied by a strong decline in the lifespan dispersion. To generate the mortality forecasts with the Lee-Carter model, we implemented the model in the statistical software R (R Core Team 2015).

Li et al. Model

Many scholars have refined the Lee-Carter model to address the problem of the inflexibility in the age profile of mortality change (Booth et al. 2006; Shang et al. 2011; Soneji and King 2011). Li et al. (2013) took an important step in this direction by

implementing a time-variant age schedule of mortality change that rotates from a present level to an ultimate level. The timing and the pace of the rotation depend on the average lifespan, which has been forecasted in a previous step with the original Lee-Carter model. As soon as life expectancy at birth exceeds a value of 80 years, the rotation starts; it then proceeds until life expectancy at birth reaches an ultimate level of 102 years. The greater the number of forecasted additional years of life is, the faster the ultimate schedule is achieved, and the rotation stops. The ultimate schedule of mortality change is constant for ages 0 to 64, and it gradually declines thereafter. The rotation basically induces a postponement of relatively large survival improvements from younger to older ages. Given that the average lifespan is forecasted using the original Lee-Carter model, the rotation affects only the underlying mortality dynamics—not the average level of mortality. Assuming a regular decline in mortality, we expect to find that (1) like the original model, the rotated model may underestimate additional years of life; but (2) unlike the original model, it may be able to forecast a mortality compression that is less strong because of its greater modeling flexibility. To take these dynamic mortality changes into account, Ševčíková et al. (2016) implemented the rotation in Raftery et al.'s (2013) model, which has been used in the UN World Population Prospects (2014, 2015). To derive the age profiles of mortality using the rotated Lee-Carter model, we implement this model in R with a few adjustments. Because we allow approaches to shift deaths beyond the maximum age of the data (see the upcoming section, "Estimation and Evaluation Procedure"), we set the ultimate schedule of mortality change constant until age 80, and as gradually declining thereafter. Moreover, for the rotation, we change the recommended bounds of the forecasted lifespan, which are 80 years and 102 years. We set the lower bound at 75 because differences in lifespan disparity started to develop for women in Italy, Japan, and Denmark as the average lifespan exceeded this value (see Fig. 1). Finally, to avoid jump-off bias, we use the last observed death rates to forecast mortality.

Bohk and Rau Model

The model of Bohk-Ewald and Rau (2017) provides an alternative strategy for forecasting that relatively large rates of mortality improvement proceed from younger to older ages. This model predicts survival improvements instead of death rates, and it optionally combines the mortality trends of multiple populations to account for (anticipated) trend changes in the forecast years. Although this model allows us to assume mortality convergence between a country of interest and reference countries, we do not use this feature in the case studies in order to enable a fair comparison with the model of Li et al. (2013). Moreover, the Bohk and Rau model has a linear and an exponential core model to forecast time series of age-specific mortality change, using simulation-based Bayesian inference to run those models and to estimate coherent changes of mortality among adjacent ages. The model has been applied to forecast mortality for some European countries (Bohk and Rau 2014; Bohk-Ewald and Rau 2017) as well as for the United States (Bohk and Rau 2016). Although both the rotating Lee-Carter model and the Bohk and Rau model allow the age profile of the rates of mortality improvement to change, the latter model appears to be more flexible because it does not assume an approximation of an ultimate schedule. If mortality declines regularly, we expect that the Bohk and Rau model will perform as well as the rotating

Lee-Carter model in forecasting average mortality and lifespan disparity and that it will perform even better in generating forecasts for populations with irregular mortality developments because it is more adaptable to different forecasting situations. To generate the forecasts with the Bohk and Rau model, we use its implementation in R, which is described in detail in Bohk-Ewald and Rau (2017).

Illustrative Examples

In this section, we validate the forecasting performance of the Lee-Carter model, its rotating variant, and the model of Bohk-Ewald and Rau. Using illustrative examples, we examine whether each model is able to generate precise forecasts of average mortality and lifespan disparity. These illustrative examples are designed to indicate whether the approaches can capture (1) regular and irregular trends of average lifespan and (2) dynamic age shifts in survival improvements.

Estimation and Evaluation Procedure

The mortality forecasts up to 2009 rely on four reference periods (1965–1990, 1960–1985, 1955–1980, and 1950–1975). We compare the estimations with the observed values. Besides e_0, which is a common indicator in evaluations, we also compare the forecasted $e_0^\dagger$ values with the observed values to assess the ability of the forecasting approaches to predict average mortality and lifespan disparity. We focus our main analysis on e_0 and $e_0^\dagger$, but we also provide results for e_{65} and $e_{65}^\dagger$ in Online Resource 1 in order to show how sensitive (or robust) our findings are.

We employ visualization techniques as well as forecast errors to evaluate the forecasting performance of each method. To quantify forecast accuracy in terms of the mean and spread of mortality, we use the absolute percentage error (APE) because it is a relative error that relates the absolute difference between forecasted and observed values to the size of the actual values. Because the APE can, therefore, deal with measures of different scales, we use it to compare the forecasting performance (across time and by country) between the methods using e_0 and $e_0^\dagger$. Given that the chosen approaches provide probabilistic mortality forecasts, we focus not only on the evaluation of median point estimates but also on the calibration of prediction intervals. We use empirical frequencies to evaluate the uncertainty estimates of probabilistic forecasts; empirical frequencies give the proportion of observed values that actually fall within the prediction intervals. For instance, a 95 % prediction interval should capture 95 % of all observations. If it captures more or fewer observations, it is too wide or too narrow, respectively (e.g., Raftery et al. 2013; Schmertmann et al. 2014).

We generate forecasts of mortality for women in Italy (regular e_0 and $e_0^\dagger$), Japan (regular e_0 and irregular $e_0^\dagger$), and Denmark (irregular e_0 and $e_0^\dagger$) because these groups have differed substantially in recent decades in their levels of life expectancy and lifespan dispersion (see Fig. 1). As input data, we use deaths and exposures by single age from 0 to 110+, and by calendar year from 1950 to 2009, from the Human Mortality Database (n.d.). To enable the forecasting approaches to shift deaths to ages beyond 110+, we extend the age range to 130+ with the Kannisto model (Thatcher et al. 1998), the details of which we explain in Online Resource 1 (section A). This approach

is similar to Ševčíková et al.'s (2016) revised UN approach. The estimation of e_0 and $e_0^\dagger$ (and of e_{65} and $e_{65}^\dagger$) is based on life tables produced from the forecasted and observed age-specific death rates.

Results

Visualize Forecast Performance Figure 2 displays the average lifespan, e_0, and the average number of life years lost, $e_0^\dagger$, for women in Italy, Japan, and Denmark. The observed data are in black, and the forecasted data are in red (Lee-Carter model), green (rotating variant proposed by Li et al. 2013), and blue (Bohk and Rau model). Moreover, the forecasted years (1991–2009) are highlighted in gray, and the reference period (1965–1990), is highlighted in beige. Given the technical construction of the Lee-Carter models, it is not surprising that the forecasts of average lifespan are almost identical: both models use the forecasted life expectancy at birth of the original model. The rotated variant deviates no more than +/– 0.1 years, which we used as a tolerance level when adjusting the age profile of mortality change with the rotation to fit the average lifespan of the original Lee-Carter model. By contrast, the forecasts of the Lee-Carter models differ in terms of lifespan disparity. The effect shown here is greater than it would have been with Li et al.'s (2013) original implementation because we let the rotation start when the average lifespan exceeded the value of 75 years, not of 80 years.

Quantify Forecast Performance Table 1 lists the mean of the APEs for e_0 and $e_0^\dagger$ over the forecast years by country and forecasting method for each validation setting, and Table 2 lists those mean absolute percentage errors (MAPEs) averaged over all four validation settings. Strikingly, the MAPEs appear to be greater for $e_0^\dagger$ than for e_0 for almost any country, validation setting, and method. The overall mean of all MAPEs is approximately 0.01 for e_0 and 0.046 for $e_0^\dagger$; that is, the forecasts deviate on average by 1 % from life expectancy at birth and by 4.6 % from lifespan dispersion. As a consequence, the forecasting performance is depreciated for all methods when we consider $e_0^\dagger$ in addition to e_0. Furthermore, the MAPEs for e_{65} and $e_{65}^\dagger$ are listed in Tables S1 and S2 in Online Resource 1. In contrast with mortality over the entire lifespan, errors often appear to be smaller for $e_{65}^\dagger$ than for e_{65}. An exception is Japan; in the validation settings 1 and 2, the errors appear to be larger for $e_{65}^\dagger$ than for e_{65}.

The empirical frequencies for e_0 and $e_0^\dagger$ in Table S3 and for e_{65} and $e_{65}^\dagger$ in Table S4 (Online Resource 1) confirm our findings for the median forecasts and show even more clearly that current approaches struggle to forecast lifespan disparity. The 95 % prediction intervals capture, on average, a fairly large number of observations for life expectancy at birth and, albeit slightly fewer, for remaining life expectancy at age 65. By contrast, many fewer observations are captured by the 95 % prediction intervals for lifespan disparity; empirical frequencies range from 0 % to 96.5 %, with the average being only approximately 26 %.

Case of Italy: Regular Trends for Mean Lifespan and Lifespan Disparity

If mortality develops regularly without any trend changes in the forecast years, the predictions of all three approaches appear to be close to the observed values. In Italy, we detect a regular increase in the average lifespan as well as a regular decline in

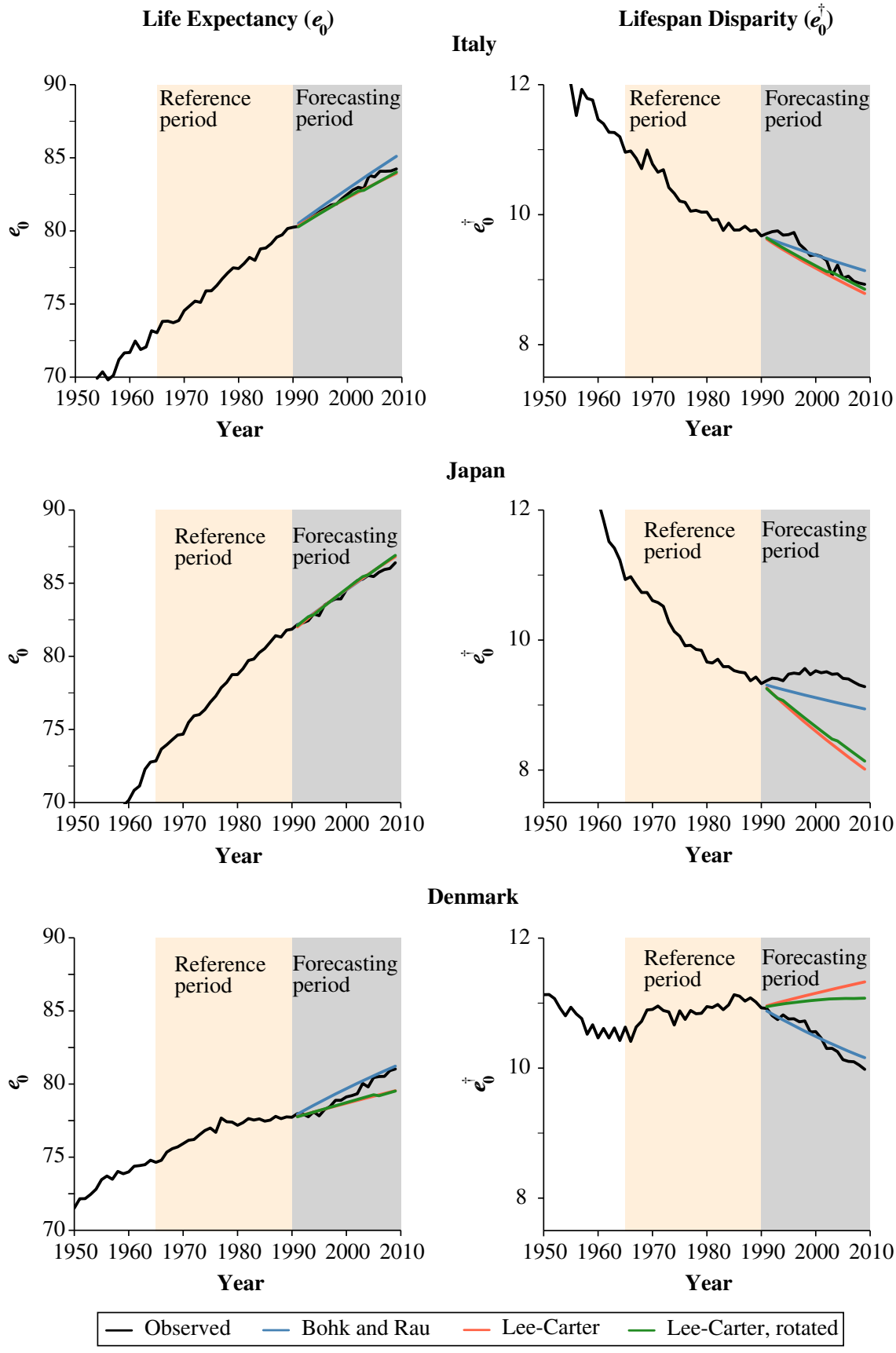

Fig. 2 Life expectancy at birth (left panels) and life years lost at birth (right panels) for women in Italy, Japan, and Denmark: Observed data, forecasted data using the Lee-Carter model, the Lee-Carter rotating variant (proposed by Li et al. 2013), and the Bohk and Rau model. Forecast years are 1991–2009. Reference period is 1965–1990

Table 1 Mean of the absolute percentage errors (MAPE) for e_0 and $e_0^\dagger$ over the forecast years by country and method

Country and Measure	Method		
	Lee-Carter	Lee-Carter, Rotated (Li et al.)	Bohk and Rau
Validation 1 (ref. years: 1965–1990; forecast years: 1991–2009)			
Italy			
e_0	0.003	0.003	0.005
$e_0^\dagger$	0.019	0.015	0.014
Japan			
e_0	0.002	0.003	0.002
$e_0^\dagger$	0.087	0.080	0.034
Denmark			
e_0	0.008	0.007	0.006
$e_0^\dagger$	0.065	0.054	0.009
Validation 2 (ref. years: 1960–1985; forecast years: 1986–2009)			
Italy			
e_0	0.010	0.010	0.002
$e_0^\dagger$	0.029	0.021	0.018
Japan			
e_0	0.002	0.002	0.004
$e_0^\dagger$	0.092	0.076	0.014
Denmark			
e_0	0.005	0.004	0.017
$e_0^\dagger$	0.094	0.077	0.021
Validation 3 (ref. years: 1955–1980; forecast years: 1981–2009)			
Italy			
e_0	0.014	0.014	0.002
$e_0^\dagger$	0.027	0.019	0.012
Japan			
e_0	0.009	0.009	0.003
$e_0^\dagger$	0.118	0.092	0.022
Denmark			
e_0	0.007	0.008	0.029
$e_0^\dagger$	0.048	0.033	0.023
Validation 4 (ref. years: 1950–1975; forecast years: 1976–2009)			
Italy			
e_0	0.018	0.018	0.008
$e_0^\dagger$	0.032	0.023	0.053
Japan			
e_0	0.018	0.018	0.012
$e_0^\dagger$	0.131	0.094	0.032
Denmark			
e_0	0.015	0.014	0.035
$e_0^\dagger$	0.020	0.018	0.043

Note: MAPEs are shown for four validating settings that all forecast mortality until 2009, but they use different historical periods.

Table 2 Mean of the absolute percentage errors (MAPE) for e_0 and $e_0^\dagger$ over all validation settings by country and method

Country and Measure	Method		
	Lee-Carter	Lee-Carter, Rotated (Li et al.)	Bohk and Rau
Italy			
e_0	0.011	0.011	0.004
$e_0^\dagger$	0.027	0.019	0.024
Japan			
e_0	0.008	0.008	0.005
$e_0^\dagger$	0.107	0.086	0.025
Denmark			
e_0	0.009	0.008	0.022
$e_0^\dagger$	0.057	0.045	0.024

lifespan disparity in the entire 1965–2009 period. Hence, Italian women experienced no trend changes, and their additional years of life were probably due to a compression of mortality that lasted (without any interruptions) in the reference and forecast years. Given these regular trends, the forecasts of all the approaches capture mean lifespan and its disparity with only negligible deviations. However, the MAPEs are smaller for e_0 (0.3 % to 0.5 %) than for $e_0^\dagger$ (1.9 % for the Lee-Carter model and 1.5 % for the other two models).

Case of Japan: Regular Trend for Mean Lifespan and Irregular Trend for Lifespan Disparity

If the average lifespan trend is regular but the lifespan disparity trend is not, differences in the predictive ability of the three approaches are present but become visible only if we complement the evaluation with a measure of dispersion. In Japan, we observe a strong increase in the average lifespan from 1965 to 2009 as well as a decline in lifespan disparity that levels off in the forecast years. Hence, among Japanese women there was a trend change in the forecast years, and their additional years of life were probably due to a compression of mortality in the reference period and a shifting of mortality in the forecast years. Given the partial instability of mortality trends among Japanese women, the forecasts of the three models are close to the observed mean lifespan. The MAPEs for e_0 range between 0.2 % and 0.3 %, suggesting that the forecasts were precise. However, the analysis of lifespan disparity shows that all the approaches overestimate the observed decline in the variability of the age at death. The deviations are greater for the Lee-Carter models (MAPEs for $e_0^\dagger$ are 8.7 % for the original model and 8.0 % for its rotated variant) than for the Bohk and Rau model (MAPE for $e_0^\dagger$ is 3.4 %). As a consequence, all three approaches predict a continuation of mortality compression while assuming that the concentration of deaths at higher ages will be greater than it actually was.

Case of Denmark: Irregular Trends for Mean Lifespan and Lifespan Disparity

If the trends of the mean lifespan and lifespan disparity are irregular, both evaluation measures indicate forecast errors. In Denmark, we observe an increase in the average lifespan in the forecast years after a period of stagnation in the 1980s and the early 1990s. We also observe a slight increase in lifespan disparity in the reference years that turns into a sharp decline in the forecast years. Hence, Danish women experienced trend changes in the forecast years. Their additional years of life were probably due to a mixture of a shifting and a worsening of mortality at different ages in the reference years as well as mortality compression in the forecast years. This result indicates that since the early 1990s, the mortality trends of Danish women have been catching up to those of vanguard populations, such as women in Italy and in Japan. Given these unstable mortality trends among Danish women, the forecasts of the three models capture the increasing trend of the average lifespan quite well. The MAPEs for e_0, 0.6 % to 0.8 %, are only slightly higher than for Italy and Japan. However, the situation is different for lifespan disparity: the Lee-Carter models (more so the original model than the rotating variant) predict an increase in the forecast years despite an actual decline. This outcome not only deviates substantially from the observed values yielding MAPEs for $e_0^\dagger$ of 6.5 % and 5.4 %, but it also appears to be rather implausible given the general negative correlation between rising life expectancy at birth and declining lifespan disparity (see Fig. 1). In contrast, the Bohk and Rau model appears to capture the changing trend in lifespan disparity in the forecast years quite well, resulting in a more plausible forecast with only small deviations from the realized values (a MAPE for $e_0^\dagger$ of only 0.9 %).

Model Comparison

The illustrative examples suggest that the Lee-Carter model is less flexible than the other two models. This shortcoming is particularly noticeable when we look at the changing mortality trends in the forecast years, especially among women in Japan and Denmark. By contrast, the rotating variant and the Bohk and Rau model appear to be more capable of adapting to changing trends because unlike the original Lee-Carter model, which assumes that the relative changes are time-invariant across ages, these models assume that survival improvements will change over time. Analyzing the forecast errors, the rotated Lee-Carter model appears to perform on average better than the other two models because its MAPEs for e_0 and $e_0^\dagger$ are relatively small for all countries and validation settings. By contrast, the original Lee-Carter model often has the largest MAPEs; and although the Bohk and Rau model often has the smallest MAPEs for life expectancy at birth and lifespan dispersion, it also has a few upward outliers that reduce its overall forecasting performance.

Sensitivity of the Results to the Reference Period

To examine the sensitivity of the above results to the choice of the reference period, we look at mortality forecasts up to 2009 that rely on different reference periods: 1960–1985, 1955–1980, and 1950–1975. The analyses are shown in Figs. S2–S4 in Online Resource 1, and have the same color scheme as in Fig. 2. Although the key message of

the results presented is not affected by changing the reference period, a comparative analysis helps to identify method-based differences.

The fits of life expectancy at birth and lifespan disparity basically appear to depend on the regularity of mortality trends and the ability of the approaches to capture them appropriately. Because Japanese and Danish women experienced irregular mortality developments, making precise forecasts for them is particularly challenging. Thus, the predictive ability of the approaches declines as the magnitude of the MAPEs increases. This effect appears to be greater for the Lee-Carter model than for the other two models, and it appears to be more pronounced in forecasts of lifespan disparity than of average lifespan. For example, the greatest overall MAPE (10.7 %) is for $e_0^\dagger$ in Japan from the Lee-Carter model, whereas the smallest overall MAPE (0.4 %) is for e_0 in Italy from the Bohk and Rau model (Table 2). The greater forecast error for the Lee-Carter model is probably due to the extrapolation of average trends of the reference period. Hence, if the overall trend of lifespan disparity is decreasing in the reference period, the Lee-Carter model tends to predict a decreasing pattern as well, and vice versa. However, the structural breaks in Danish and Japanese lifespan disparity appear to be unexpected and are therefore difficult to capture in a forecast generated by any model that has not been designed for this specific task. If we look instead at the more regular mortality trends in Italy, for example, we can see that the Lee-Carter models tend to generate rather conservative forecasts of progress in the average lifespan; that is, they tend to systematically underestimate the observed trajectories and yield overall MAPEs for e_0 of 1.1 %. By contrast, the forecasts of the Bohk and Rau model yield a smaller overall MAPE for e_0 (0.4 %) than the forecasts of the other two models, and they sometimes systematically overestimate the additional years of life. Examining lifespan disparity reveals even more differences between the approaches, particularly between the two Lee-Carter models in the forecasts of Japanese female mortality. The rotating variant appears to capture the flattening decline of lifespan disparity in the forecast years much better than the original model, and thus substantially improves forecasting performance: the overall MAPE for $e_0^\dagger$ in Japan is substantially lower for the rotated Lee-Carter variant (8.6 %) than for the original model (10.7 %). Also clearly visible for lifespan dispersion in Japan is that the further in time the reference period is, the more forecasts of the rotating variant diverge from those of the original model and converge with those of the Bohk and Rau model. Given that the rotation starts when life expectancy exceeds 75 years and that Japanese women reached this point in the early 1970s, this finding is not really surprising. As a consequence, the forecast that relies on data from 1950 to 1975 is also the forecast in which the rotation has the largest effect on the results. This finding demonstrates the need for time-variant survival improvements in order to capture dynamic trends in the variability of the age at death. The remaining deviations from the real values indicate that refining (or developing) forecasting approaches may help to account for patterns in lifespan disparity, such as the compression, shifting, and expansion of mortality. However, we do not expect forecasting errors to be equal to 0 because they show more signs of overfitting than of high forecasting performance.

Also of note is that the predictive ability of forecasts that rely on data from 1950 to 1975 appears to be lower than that of forecasts based on more recent mortality trends. Because this effect can be seen for the average lifespan and also partly for lifespan disparity, we speculate that it may be attributable to the delayed onset of the old-age

mortality decline in the 1970s, which was crucial for future mortality developments in all three populations. Hence, if a major driving trend of mortality in the forecast years is missing in the reference period, the forecasting performance may be substantially reduced.

If we restrict our analysis to ages above 65, the relation of errors (MAPEs) for remaining years of life and lifespan disparity reverses. An exception is Japan, which shows larger errors for lifespan disparity than for remaining years of life at age 65, but only in the validation settings 1 and 2. Most likely, the onset of old-age mortality decline (Kannisto 1994) causes the reversal in the error pattern. Analyzing the magnitude of errors across all four validation settings provides evidence that the more years that are included in the reference period since the onset of the old-age mortality decline, the more accurate are the forecasts of remaining life expectancy at age 65. Given that the survival improvements at older ages primarily induced a parallel downward shift of the force of mortality on the log scale, the effects were large for e_{65} but only marginal for $e^{\dagger}_{65}$. This development is widely known as *shifting mortality* and has been described in detail by, for example, Bongaarts (2005) and Canudas-Romo (2008). Japan is the world record leader in terms of life expectancy thanks to exceptionally large old-age mortality improvements; we assume that these deviant/special trends in mortality may have caused larger changes in the variability at death that have not been captured in the forecasts and thus lead to larger errors for $e^{\dagger}_{65}$ in the last two validation settings (Cheung and Robine 2007; Wilmoth and Robine 2003).

Summary and Concluding Remarks

Our analysis has shown that some methods—among them, the original Lee-Carter model, which is considered a golden standard in mortality forecasting—struggle to account for trends in lifespan disparity. This shortcoming, often caused by rather time-invariant survival improvements, has not been shown so clearly yet because the toolkit for evaluating the forecast performance focused on, for example, life expectancy at birth and age-specific death rates. These measures of *central tendency* are typically used to analyze *ex post* to what extent forecasts deviate from their realized values. Although these parameters of central tendency are useful for assessing how precisely average mortality has been forecasted, they cannot be used to determine whether the forecasted underlying mortality developments are plausible. This may be a serious drawback because similar average lifespans can result from different underlying mortality developments, which describe a dynamic age shift of survival improvement from younger to older ages in many highly developed countries in the last decades. As a consequence, small forecast errors of average lifespan do not necessarily indicate plausible trends in the forecasted underlying mortality dynamics. To assess whether the forecasts of the underlying developments are also plausible, we propose to use measures of lifespan disparity in the evaluation procedure. Despite many suitable measures of lifespan dispersion, we employed $e^{\dagger}_{0}$ as a measure of *spread* to tackle this problem.

In illustrative mortality forecasts for women in Italy, Japan, and Denmark—three populations who differ substantially in terms of lifespan disparity (see Fig. 1)—$e^{\dagger}_{0}$ was a useful addition to the common toolkit for evaluating the predictive ability of

forecasting approaches. We used the original Lee-Carter model (Lee and Carter 1992), its rotating variant proposed by Li et al. (2013), and the model of Bohk-Ewald and Rau (2017) to predict mortality up to 2009. Because the three approaches differ primarily in their ability to capture dynamic age shifts in the distribution of deaths, they are particularly suitable for evaluating how well they are able to forecast actual developments in average lifespan and lifespan disparity. To examine the sensitivity of our results, we chose four reference periods instead of just one: 1965–1990, 1960–1985, 1955–1980, and 1950–1970. We then compared the forecasted values of the average lifespan and lifespan disparity with the actual values.

The comparative analysis revealed that irrespective of the reference period, forecasting performance basically depends on the regularity (or continuation) of mortality trends and the ability of the approaches to capture them appropriately. Although the forecasts of life expectancy at birth generated by the Lee-Carter models are rather conservative, the forecasts generated by the Bohk and Rau model often have small forecast errors but also a few upward outliers. Moreover, the Japanese forecasts were found to be precise when we looked at average lifespan only, but they turned out to be rather inaccurate when we took lifespan disparity into account as well. Hence, the models were not able to capture the flattening decline of Japanese lifespan disparity in the forecast years, although the rotating model and the Bohk and Rau model fared better than the original Lee-Carter model because of time-variant survival improvements.

However, the remaining deviations from the observed values indicate that the refinement or the development of forecasting approaches should focus not only on average mortality but also on lifespan disparity. This indication may be particularly important given the concentration of mortality improvement potentials at the highest ages. Improving mortality at those ages could imply that people will probably live beyond current maximum ages. Hence, it will be crucial for forecasting approaches to be able to capture multiple trends in the (right) tail of the lifespan distribution (stagnation or expansion). As a consequence, the approaches should be able to forecast further reductions in mortality not only up to the current maximum ages but also to higher ages beyond. Doing so requires a high degree of modeling flexibility, which has been missing in existing approaches.

To summarize, our analysis illustrates that the joint evaluation of the average lifespan (e_0) and the life years lost ($e_0^\dagger$) provide new insights that we believe are needed for a comprehensive evaluation of the predictive performance of mortality forecasts. We also suggest that these new insights should be used when improving or developing new methods for forecasting mortality. Until now, these approaches were exclusively designed to capture the almost linear increase in life expectancy at birth. Hence, it is not surprising that forecasts of the average lifespan turn out to be more accurate and yield smaller forecast errors. The incorporation of lifespan disparity as a quality criterion or even central outcome may substantially improve the methodology.

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Online Resource 1

Lifespan Disparity as an Additional Indicator for Evaluating Mortality Forecasts

Christina Bohk-Ewald, Marcus Ebeling, and Roland Rau

A Extending the age range beyond 110+

The data used are death counts and exposures by single age, 0 to 110+, from the Human Mortality Database (2015). To enable forecasting approaches to shift deaths to ages beyond 110+, we extend the age range of mortality data, like Ševčíková et al. (2016), with the model of Kannisto:

$$\mu_x = \frac{\alpha e^{\beta x}}{1 + \alpha e^{\beta x}} \tag{2}$$

as it is described in Thatcher et al. (1998, p. 16). We fit the model with the function *optim* in *R* (2015) to mortality at ages 80 to 110 using a Poisson log-likelihood for women in Italy, Denmark, and Japan for each year between 1950 and 2009. We then use the fitted Kannisto models to smooth mortality for ages 80 to 110, and to predict mortality for the ages above 110 in each year. Figure S1 depicts such mortality data for ages 80 to 130 for Italian, Danish, and Japanese women in the year 1950. The model of Kannisto is particularly suitable for populations with low mortality. It is also applied by, e.g., the Human Mortality Database and the United Nations (2014) to fit old-age mortality.

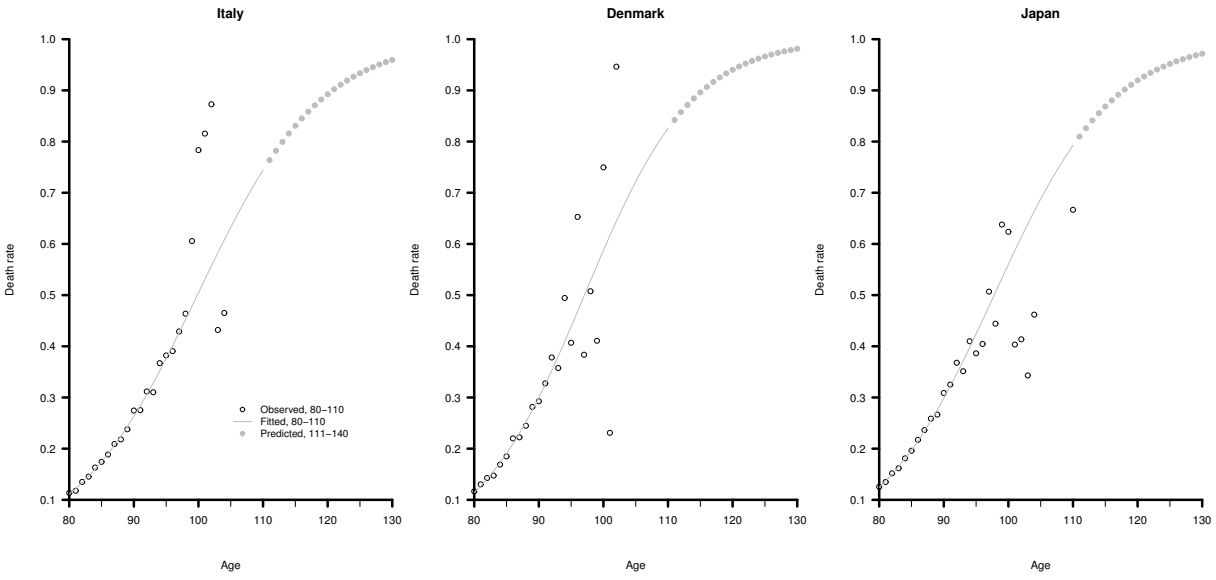

Fig. S1 Extended age range of mortality data of Italian (left), Danish (center), and Japanese (right) women in 1950. We used the model of Kannisto to fit (gray line) observed mortality (black circles) at ages 80 to 110, and to predict it at ages 111 to 130 (gray dots).

B Forecasts until 2009 with reference periods 1960-1985, 1955-1980, and 1950-1975

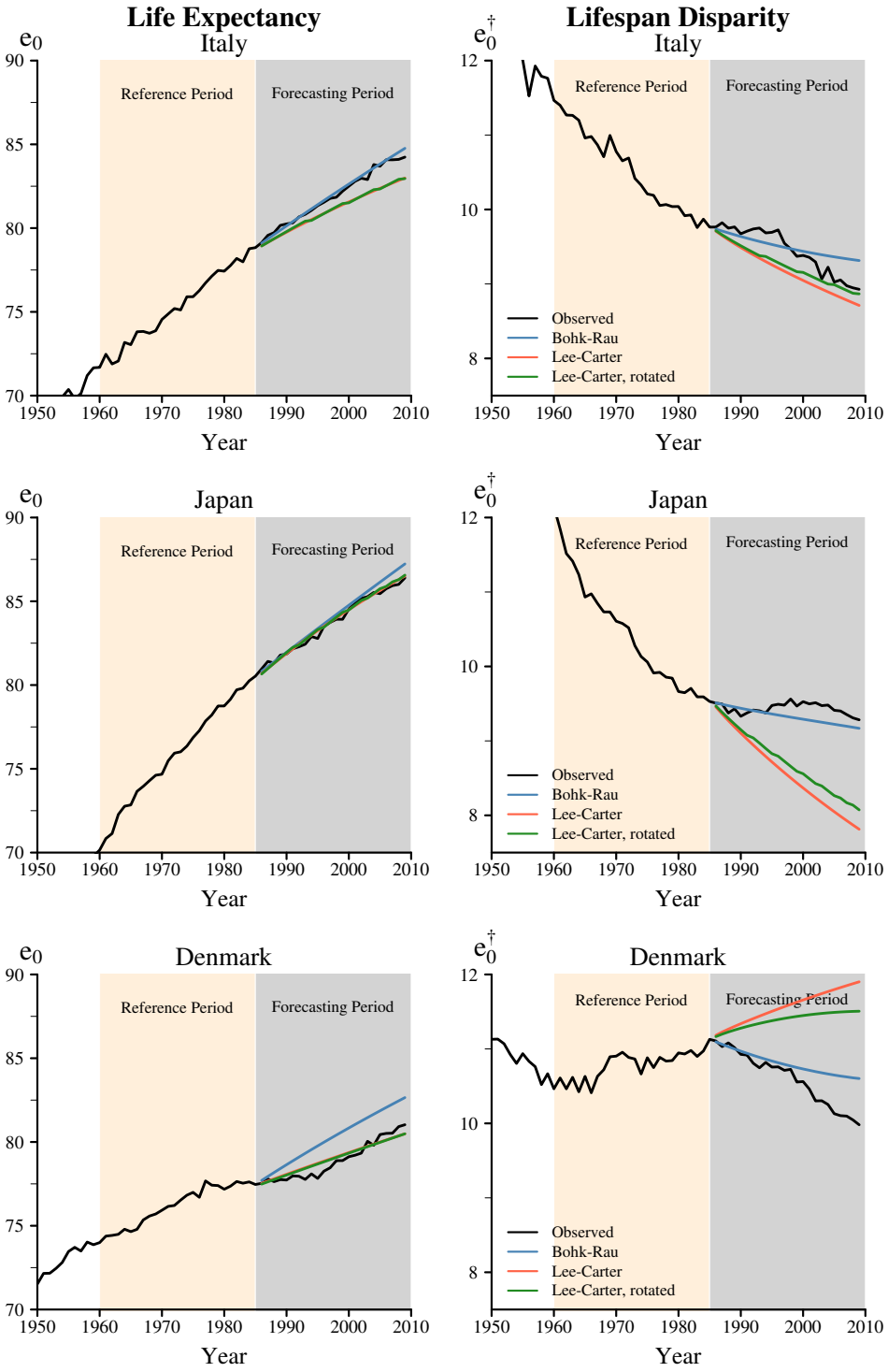

Fig. S2 Life expectancy at birth (left panels) and life years lost at birth (right panels) for women in Italy (top), Japan (center), and Denmark (bottom); observed data are in black, forecasted data are in red (Lee-Carter model), green (rotating variant), and blue (Bohk-Rau model); reference period: 1960-1985.

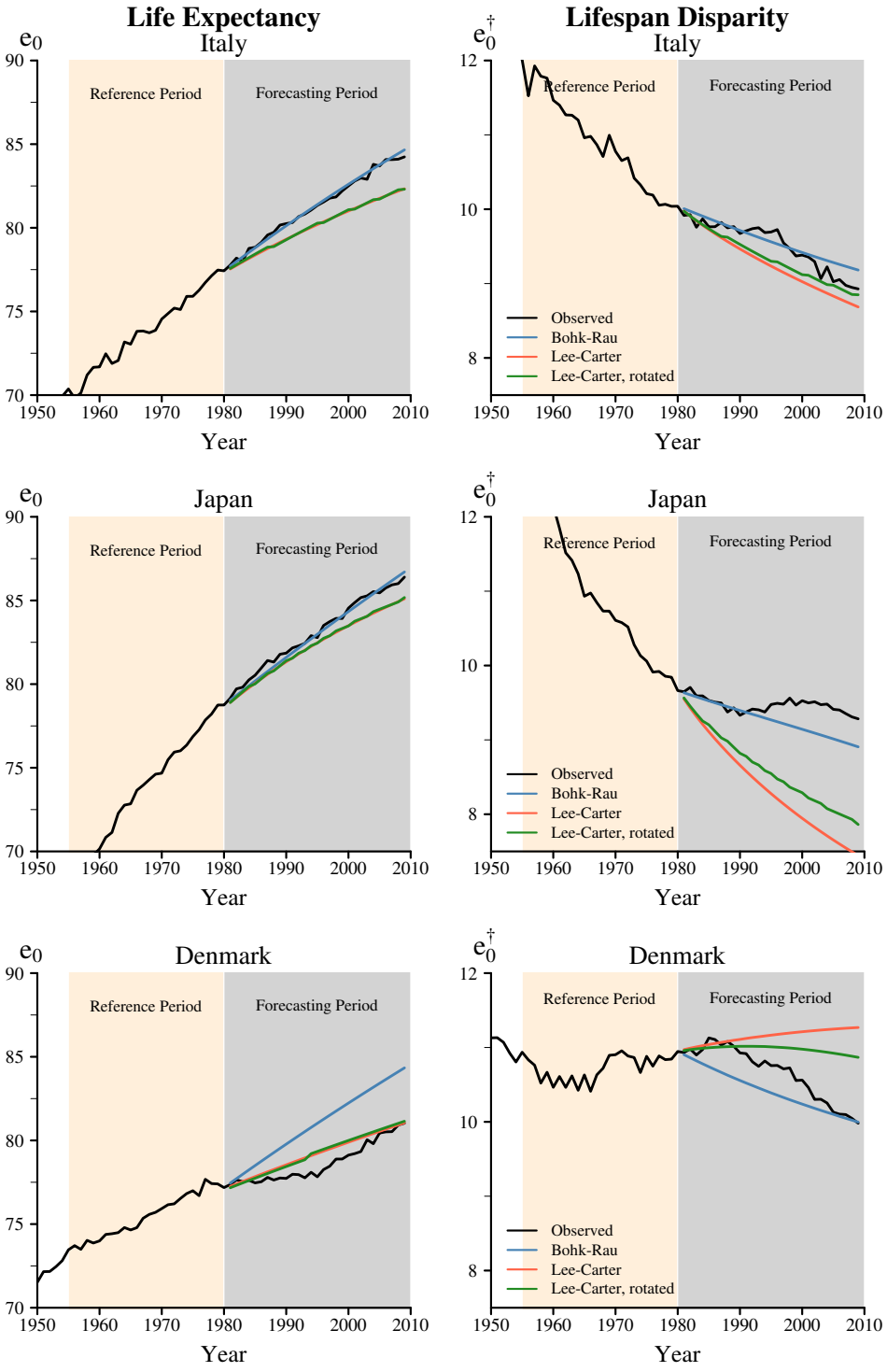

Fig. S3 Life expectancy at birth (left panels) and life years lost at birth (right panels) for women in Italy (top), Japan (center), and Denmark (bottom); observed data are in black, forecasted data are in red (Lee-Carter model), green (rotating variant), and blue (Bohk-Rau model); reference period: 1955-1980.

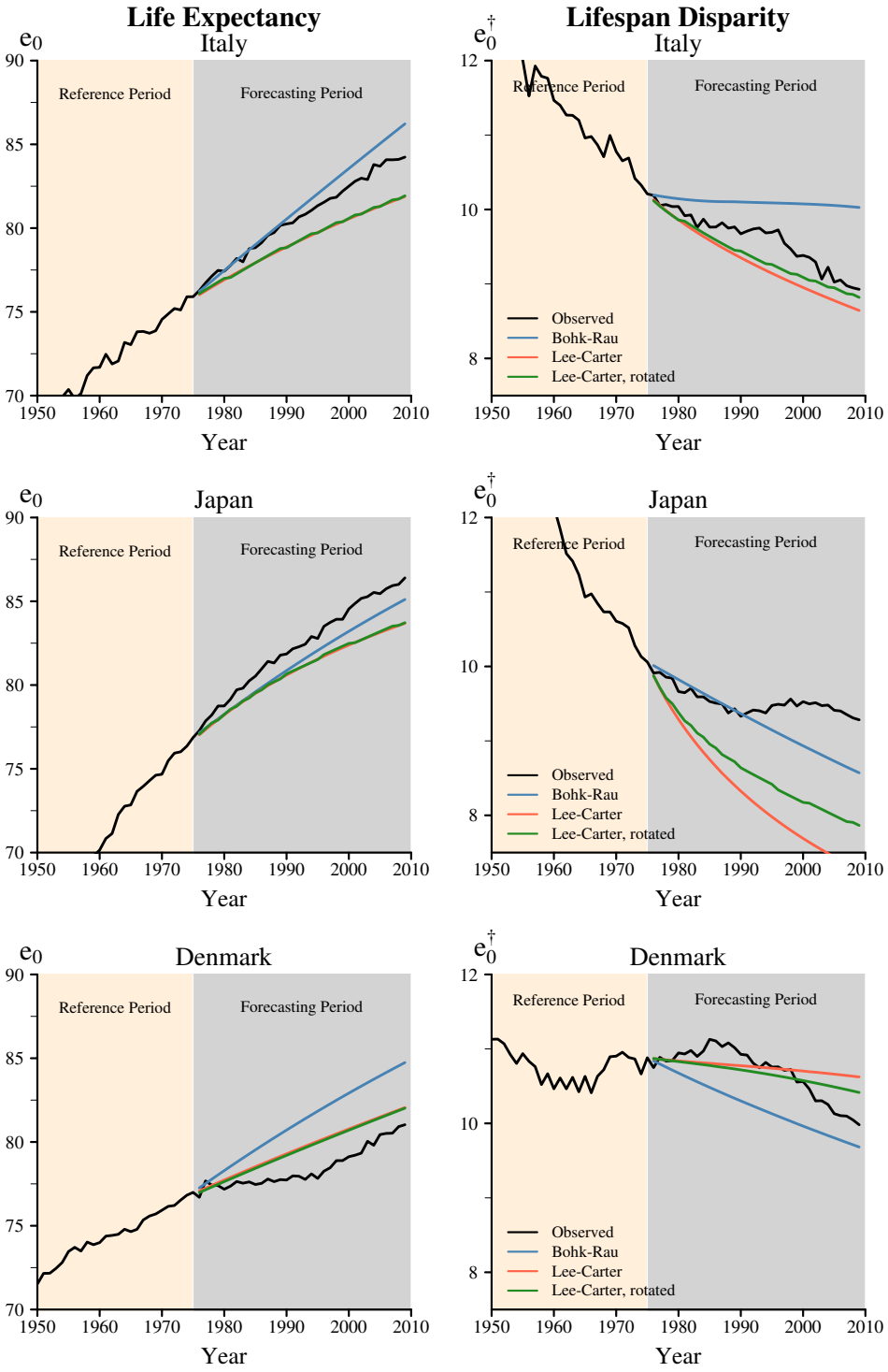

Fig. S4 Life expectancy at birth (left panels) and life years lost at birth (right panels) for women in Italy (top), Japan (center), and Denmark (bottom); observed data are in black, forecasted data are in red (Lee-Carter model), green (rotating variant), and blue (Bohk-Rau model); reference period: 1950-1975.

C MAPE estimates for e_{65} and $e_{65}^{\dagger}$

Country	Measure	LC	LC, rotated	Bohk–Rau
Validation 1 (Ref.years: 1965–1990; Forecast years: 1991–2009)				
Italy	e_{65}	0.016	0.013	0.014
	$e^{\dagger}_{65}$	0.009	0.008	0.021
Japan	e_{65}	0.015	0.012	0.008
	$e^{\dagger}_{65}$	0.056	0.050	0.012
Denmark	e_{65}	0.020	0.018	0.026
	$e^{\dagger}_{65}$	0.016	0.013	0.010
Validation 2 (Ref.years: 1960–1985; Forecast years: 1986–2009)				
Italy	e_{65}	0.046	0.040	0.008
	$e^{\dagger}_{65}$	0.016	0.012	0.034
Japan	e_{65}	0.025	0.018	0.007
	$e^{\dagger}_{65}$	0.050	0.041	0.020
Denmark	e_{65}	0.054	0.046	0.062
	$e^{\dagger}_{65}$	0.051	0.045	0.044
Validation 3 (Ref.years: 1955–1980; Forecast years: 1981–2009)				
Italy	e_{65}	0.055	0.049	0.006
	$e^{\dagger}_{65}$	0.027	0.022	0.020
Japan	e_{65}	0.068	0.056	0.018
	$e^{\dagger}_{65}$	0.067	0.055	0.008
Denmark	e_{65}	0.063	0.057	0.099
	$e^{\dagger}_{65}$	0.009	0.010	0.012
Validation 4 (Ref.years: 1950–1975; Forecast years: 1976–2009)				
Italy	e_{65}	0.065	0.057	0.041
	$e^{\dagger}_{65}$	0.026	0.020	0.076
Japan	e_{65}	0.105	0.085	0.050
	$e^{\dagger}_{65}$	0.071	0.054	0.029
Denmark	e_{65}	0.066	0.060	0.102
	$e^{\dagger}_{65}$	0.011	0.011	0.009

Table S1 Mean of the Absolute Percentage Errors (MAPE) for e_{65} and $e^{\dagger}_{65}$ over the forecast years by country and method. MAPEs are shown for four validating settings that all forecast mortality until 2009, but use different historical periods.

Country	Measure	LC	LC, rotated	Bohk-Rau
Average across all validation settings				
Italy	e_{65}	0.046	0.040	0.017
	$e^{\dagger}_{65}$	0.020	0.016	0.038
Japan	e_{65}	0.053	0.043	0.021
	$e^{\dagger}_{65}$	0.061	0.050	0.017
Denmark	e_{65}	0.051	0.045	0.072
	$e^{\dagger}_{65}$	0.021	0.019	0.019

Table S2 Mean of the Absolute Percentage Errors (MAPE) for e_{65} and $e^{\dagger}_{65}$ over all validation settings by country and method.

D Empirical frequencies for e_0 and $e_0^\dagger$

Country	Measure	LC	LC, rotated	Bohk–Rau
Validaton 1 (Ref.years: 1965–1990; Forecast years: 1991–2009)				
Italy	e_0	100.00	100.00	94.74
	$e_0^\dagger$	10.53	31.58	36.84
Japan	e_0	100.00	100.00	94.74
	$e_0^\dagger$	0.00	0.00	0.00
Denmark	e_0	100.00	100.00	89.47
	$e_0^\dagger$	0.00	0.00	42.11
Validation 2 (Ref.years: 1960–1985; Forecast years: 1986–2009)				
Italy	e_0	79.17	91.67	91.67
	$e_0^\dagger$	0.00	20.83	41.67
Japan	e_0	100.00	100.00	87.50
	$e_0^\dagger$	0.00	0.00	50.00
Denmark	e_0	100.00	100.00	54.17
	$e_0^\dagger$	0.00	0.00	37.50
Validation 3 (Ref.years: 1955–1980; Forecast years: 1981–2009)				
Italy	e_0	55.17	72.41	89.66
	$e_0^\dagger$	13.79	34.48	48.28
Japan	e_0	100.00	100.00	48.28
	$e_0^\dagger$	0.00	0.00	96.55
Denmark	e_0	100.00	100.00	55.17
	$e_0^\dagger$	13.79	13.79	37.93
Validation 4 (Ref.years: 1950–1975; Forecast years: 1976–2009)				
Italy	e_0	64.71	79.41	88.24
	$e_0^\dagger$	5.88	26.47	2.94
Japan	e_0	64.71	100.00	26.47
	$e_0^\dagger$	2.94	2.94	82.35
Denmark	e_0	100.00	100.00	41.18
	$e_0^\dagger$	20.59	17.65	32.35

Table S3 Empirical frequencies, in %, for the 95% prediction intervals of e_0 and $e_0^\dagger$ over the forecast years by country and method. The empirical frequencies are shown for four validating settings that all forecast mortality until 2009, but use different historical periods.

E Empirical frequencies for e_{65} and $e_{65}^{\dagger}$

Country	Measure	LC	LC, rotated	Bohk–Rau
Validaton 1 (Ref.years: 1965–1990; Forecast years: 1991–2009)				
Italy	e_{65}	94.74	100.00	100.00
	$e^{\dagger}_{65}$	21.05	10.53	57.89
Japan	e_{65}	100.00	100.00	47.37
	$e^{\dagger}_{65}$	0.00	0.00	63.16
Denmark	e_{65}	100.00	100.00	57.89
	$e^{\dagger}_{65}$	31.58	36.84	73.68
Validation 2 (Ref.years: 1960–1985; Forecast years: 1986–2009)				
Italy	e_{65}	4.17	29.17	83.33
	$e^{\dagger}_{65}$	4.17	12.50	66.67
Japan	e_{65}	70.83	95.83	50.00
	$e^{\dagger}_{65}$	4.17	4.17	91.67
Denmark	e_{65}	100.00	100.00	45.83
	$e^{\dagger}_{65}$	0.00	0.00	8.33
Validation 3 (Ref.years: 1955–1980; Forecast years: 1981–2009)				
Italy	e_{65}	13.79	20.69	93.10
	$e^{\dagger}_{65}$	0.00	0.00	75.86
Japan	e_{65}	6.90	41.38	24.14
	$e^{\dagger}_{65}$	0.00	0.00	93.10
Denmark	e_{65}	100.00	100.00	55.17
	$e^{\dagger}_{65}$	48.28	41.38	89.66
Validation 4 (Ref.years: 1950–1975; Forecast years: 1976–2009)				
Italy	e_{65}	20.59	35.29	94.12
	$e^{\dagger}_{65}$	2.94	2.94	61.76
Japan	e_{65}	5.88	38.24	23.53
	$e^{\dagger}_{65}$	2.94	0.00	94.12
Denmark	e_{65}	100.00	100.00	61.76
	$e^{\dagger}_{65}$	35.29	41.18	88.24

Table S4 Empirical frequencies, in %, for the 95% prediction intervals of e_{65} and $e^{\dagger}_{65}$ over the forecast years by country and method. The empirical frequencies are shown for four validating settings that all forecast mortality until 2009, but use different historical periods.

A. Author contributions

Authors	Title	Contributions
Marcus Ebeling, Frederik Peters & Roland Rau	The concept of equivalent time as a simple indicator for the assessment of survival progress	ME and FP conceived the study. ME conducted the analysis. ME, FP, and RR prepared the manuscript. All authors contributed to drafting and critical revision.
Marcus Ebeling	How has the lower boundary of human mortality evolved and has it already stopped to decrease?	ME conceived the study, conducted the analysis and prepared the manuscript.
Marcus Ebeling, Roland Rau & Annette Baudisch	Rectangularization of the survival curve reconsidered: The maximum inner rectangle approach	ME and RR conceived the study. ME conducted the analysis. ME, RR, and AB prepared the manuscript. All authors contributed to drafting and critical revision.
Marcus Ebeling, Karin Modig, Anders Ahlbom & Roland Rau	The effects of increasing longevity and changing incidence on lifetime risk differentials. A decomposition approach	ME, RR, and AA conceived the study. KM conducted the data acquisition. ME conducted the analysis. ME, KM, AA, and RR prepared the manuscript. All authors contributed to drafting and critical revision.
Christina Bohk-Ewald, Marcus Ebeling & Roland Rau	Lifespan disparity as an additional indicator for evaluating mortality forecasts	ME and CBE conceived the study. ME and CBE conducted the analysis. CBE, ME, and RR prepared the manuscript. All authors contributed to drafting and critical revision.
	Papers in the appendix	
Roland Rau, Marcus Ebeling, Frederik Peters, Christina Bohk-Ewald & Trifon I. Missov	Where is the level of the mortality plateau	RR conceived the study and conducted the analysis. All authors prepared the manuscript. All authors contributed to drafting and critical revision.
Marie-Pier Bergeron-Boucher, Marcus Ebeling & Vladimir Canudas-Romo	Decomposing changes in life expectancy: Compression versus shifting mortality	ME and MBB conceived the study. MBB conducted the analysis. MBB, ME, and VCR prepared the manuscript. All authors contributed to drafting and critical revision.

B. Annexed research papers

B.1. Appendix paper I: Where is the level of the mortality plateau?

LIVING to 100
SOCIETY OF ACTUARIES
INTERNATIONAL SYMPOSIUM

Where Is the Level of the Mortality Plateau?

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Presented at the Living to 100 Symposium
Orlando, Fla.
January 4–6, 2017

Where Is the Level of the Mortality Plateau?

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Using data from the "International Database on Longevity", Gampe (2010) found a plateau of mortality — which is implied in a logistic model — at ages 110 and above. In her study, Gampe pursued a nonparametric approach. Hence this plateau was not the consequence of forcing a certain parametric shape of the mortality trajectory. Gampe estimated a level of the force of mortality of about $\mu_{110+} = 0.7$, corresponding to a probability of dying of approximately 0.5. Gampe's results strengthened the initial findings that were reported at the "Living to 100" conference in 2005 (Robine et al., 2005).

Our research question can be expressed simply as: Can we find support for this upper limit of the force of mortality of 0.7, i.e. the chance to survive another year is approximately the chance of tossing a fair coin?

1 Introduction

Few empirical findings are as regular as the exponential increase in mortality at adult ages. From one age to the next, mortality rises with a constant rate of slightly more than

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10 percent. This log-linear increase in the force of mortality, $\mu(x)$, is well approximated by Benajmin Gompertz's "law" for the force of mortality, $\mu(x) = ae^{bx}$ with age x, intercept a and slope parameter b. Most researchers agree that the rate of change decreases after about age 80 or 85, a phenomenon commonly called "mortality deceleration" (e.g., Horiuchi and Wilmoth, 1998). As usual when analyzing differences in aggregate measures over time, age, region or any other dimension, there are three possible strains of explanation (Vaupel and Canudas-Romo, 2002). The first among them is problematic data. It is well known that registration of death counts is more precise than the estimates for populations at very high ages, the latter often being inflated. With the same number of events in the numerator but with a larger denominator, age-specific mortality tends to be artificially low. Alternatively, there could be an *actual* decrease in the rate of aging, potentially caused by a slowing down of metabolic process on the individual level (e.g. Ukraintseva and Yashin, 2001).

However, it is selective mortality that is most often referred to when mortality deceleration is discussed. The idea is simple: Populations are heterogeneous. Even if the risk of dying increases at the same pace for all individuals, some are probably frailer than others. Those individuals would die, on average, earlier than their more "robust" peers. With less and less frail individuals alive, the observed force of mortality for the total population tends to level off. The simplest so-called frailty model that can explain the leveling off is probably the one introduced by Vaupel et al. (1979), where the baseline mortality is multiplied for individual i by a factor z_i that follows a Γ distribution with a mean of 1. Vaupel and Yashin (1985) and Vaupel (2010) contain more intuitive illustrations than the more technical initial paper from 1979. A thorough discussion of other mixing distributions than the Γ for univariate frailty models and of multivariate frailty models can be found in Wienke (2010).

But what will be the shape of the trajectory at even higher ages when data become very sparse? Will mortality continue to rise, albeit at a slower pace than at younger ages?

Will it reach a maximum? Using various parametric models, Thatcher et al. (1998) concluded that a model with a logistic shape[1] fitted the data best. Also Thatcher's analysis in 1999 concluded that such a model with a mortality plateau should be employed for the estimation of mortality at advanced ages. In other applications of the logistic model, such an asymptotic maximum is called "carrying capacity."

Intensive research during the past decade supported the notion of such a logistic pattern. Theoretical, mathematical and empirical studies point toward a "Gompertzian" age-specific hazard on the individual level with a gamma distribution for frailty across individuals (e.g., Finkelstein and Esaulova, 2006; Missov and Finkelstein, 2011; Steinsaltz and Wachter, 2006). The findings of the past 10 years are succinctly summarized in Missov and Vaupel (2015, p. 69):

> The observed leveling-off of human mortality rates at ages 110+ has four major implications for the generating mechanism. First, plateaus can be modeled in the framework of multiplicative (proportional) or additive hazards, but not by accelerated failure time models. Second, the distribution of unobserved heterogeneity has a regularly-varying-at-zero density at the starting age and converges subsequently to the gamma distribution. Third, in a proportional hazards setting the baseline cumulative hazard is the inverse of the negative logarithm of any completely positive function, and the well-known gamma-Gompertz pair can be derived as a special meaningful case. Fourth, in an additive hazards setting plateaus are generated by taking the latter proportional hazards pattern and adding a frailty-independent term that levels off with age. Many conjugate pairs, i.e., pairs of baseline mortality and frailty distributions, can produce the same mortality pattern. The only demographically meaningful multiplicative model that holds *at* the plateau is the gamma-Gompertz.

[1] Either a general logistic model or a simplified version, the Kannisto model. The quadratic model was excluded, since it would lead, ultimately, to a force of mortality of 0.

Using data from the International Database on Longevity,[2] Gampe (2010) found, indeed, a plateau of mortality—which is implied in a logistic model—at ages 110 and above. In her study, Gampe pursued a nonparametric approach. Hence this plateau was *not* the consequence of forcing a certain parametric shape of the mortality trajectory. Gampe estimated a level of the force of mortality of $\mu(x)_{110+} = 0.7$, corresponding to a probability of dying of $q(x)_{110+} = 1 - e^{\mu(x)_{110+}} \approx 0.50$. Gampe's results strengthened the initial findings that were reported at the "Living to 100" conference in 2005 (Robine et al., 2005).

Our research question can be expressed simply as: Can we find support for this upper limit of the force of mortality of 0.7, corresponding to a probability to survive another year of approximately tossing a fair coin?

2 Data and Methods

All our estimates are based on data from the Human Mortality Database (2016). Following the standard literature (e.g. Brillinger, 1986; Carstensen, 2007; Keiding, 1990), we estimated our parametric models in a maximum-likelihood framework assuming that age-specific deaths $D(x) \sim \text{Poisson}(\bar{\mu}(x)N(x))$, where $N(x)$ denotes the number of person-years lived (= exposure time) at age x. The log-likelihood for parameter vector θ that needs to be optimized given the data is:

$$\log -\mathcal{L}(\theta|D(x), N(x)) = \sum_{x=\alpha}^{\beta} \left[D(x)\log(\bar{\mu}(x)) - \bar{\mu}(x)N(x)\right]. \tag{1}$$

We typically looked at ages $\alpha = 80$ until $\beta = 109$. In our application, the population level hazard $\bar{\mu}(x)$ in Equation 1 was substituted by the respective hazard function.
Our main model was the gamma-Gompertz model that is commonly expressed as (see, for instance Missov and Vaupel, 2015, p. 674):

[2]This database is available from the Max Planck Institute for Demographic Research at `http://www.supercentenarians.org/`.

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

with intercept a and slope b from the Gompertz distributions and the variance of the gamma distribution γ. Thus, the parameter vector θ that needs to be optimized consists of three elements (a, b, γ).

For reasons of better numerical stability when fitting the data, we used the alternative parametrization via the Gompertz modal age at death M (see, for instance, Missov and Vaupel, 2015):

$$\bar{\mu}(x) = \frac{be^{b(x-M)}}{1 + \gamma e^{-bM}\left(e^{bx} - 1\right)} \tag{3}$$

The mortality plateau for this model can be expressed as $\lim_{x\to\infty} \bar{\mu}(x) = \bar{\mu}^* = b/\gamma$.

3 Results

3.1 Selection of Models

Before we settled on the model of Equation 2 (or Eq. 3), we also estimated alternative models to make sure that the gamma-Gompertz approach is the best model. The simplest model is the pure Gompertz model. It can be expressed in the traditional way and via the mode, which we used in the actual estimation (see Horiuchi et al., 2013, also for the reparametrization of the other models via the mode).

$$\text{traditional} : \bar{\mu}(x) = ae^{bx} \qquad \text{via mode} : \bar{\mu}(x) = be^{b(x-M)}. \tag{4}$$

William Makeham (1867) added an age-independent constant to account for a nonsenescent component ("background" mortality):

$$\text{traditional} : \bar{\mu}(x) = ae^{bx} + c \qquad \text{via mode} : \bar{\mu}(x) = be^{b(x-M)} + c. \tag{5}$$

Likewise, the gamma-Gompertz model of Equations 2 and 3 can also be extended via a Makeham term:

$$\text{traditional} : \bar{\mu}(x) = \frac{ae^{bx}}{1 + \frac{a\gamma}{b}\left(e^{bx} - 1\right)} + c \qquad \text{via mode} : \bar{\mu}(x) = \frac{be^{b(x-M)}}{1 + \gamma e^{-bM}\left(e^{bx} - 1\right)} + c. \tag{6}$$

Please note that models 4 and 5 do not imply a mortality plateau. Mortality in the Gompertz and Gompertz-Makeham scenarios continues to rise exponentially. Although the overwhelming majority of researchers provide evidence for mortality deceleration, Gavrilov and Gavrilova (2011) argue that there are no signs of mortality deceleration (in their data set for the United States). That is why we have included models 4 and 5.

The best model was selected based on the minimum AIC value for each of the four models. The criterion was estimated using the standard approach as outlined by Akaike (1974, p. 716):

$$\begin{aligned} \text{AIC} \quad = \quad & (-2)\log(\text{maximum likelihood}) + 2(\text{number of} \\ & \text{independently adjusted parameters within the model}) \end{aligned}$$

The results for the years 1980–2010 for a selection of seven large countries, separately for women and men, are shown in Figure 1.

The predominant color is green, indicating that in 340 out of the 434 separately estimated models ($\approx 78\%$), the gamma-Gompertz is better suited than the other models. The gamma-Gompertz-Makeham model is the best model in 21% of all cases. But if we had dropped the United States from our sample, a country with weak data quality at those ages, according to Jdanov et al. (2008), this number would drop to 15%. With the Gom-

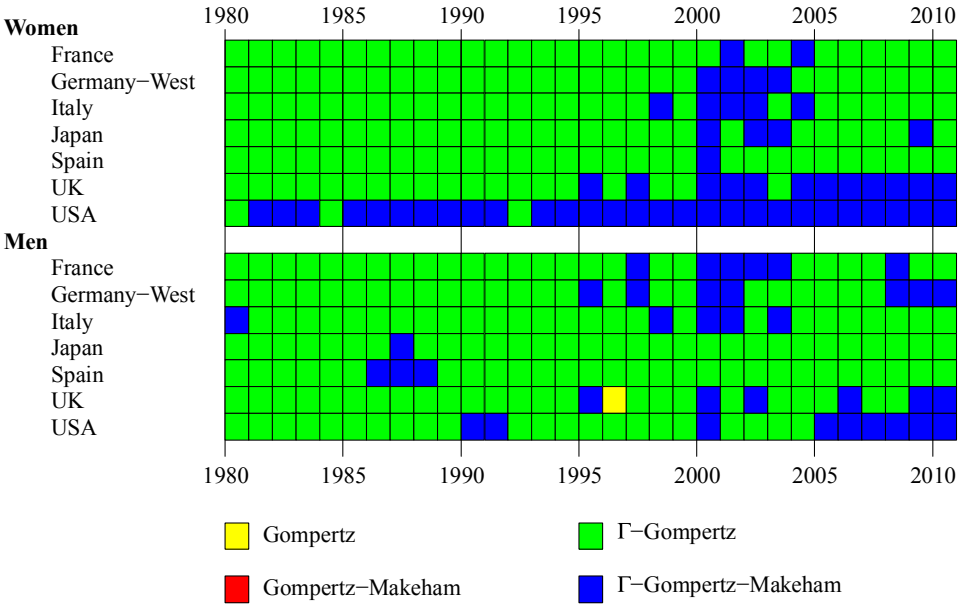

Figure 1: Best Models When Comparing Gompertz, Gompertz-Makeham, Gamma-Gompertz and Gamma-Gompertz-Makeham Models Note: For seven large countries, 1980–2010, separately for women and men. Source: Own estimations based on data from the Human Mortality Database (2016).

pertz model being best in the United Kingdom in 1996, there is only one case where the data supported a model without deceleration and, therefore, without a plateau.

3.2 Graphical Fit of the Gamma-Gompertz Model

Selecting the best out of a variety of models does not guarantee in any way that the model fits the data well, regardless of the selection criterion (AIC, BIC, r^2, ...). As a simple example: A quadratic model is (necessarily) at least as close to the data as a straight line in linear regression. But it might not capture the essential features of the data to be fitted (e.g., a third-order polynomial). Likewise, we need to check that the gamma-Gompertz model is not only the best of the four models but also a close fit to the data. We did this on a visual basis: The blue lines in the six panels of Figure 2 show, for 2010 data for females

from Japan, Spain, Sweden, France, Germany and Italy, how extremely well the selected gamma-Gompertz model actually fits the observed death rates (plotted with plus signs). Hence, we cannot expect that our estimates for $\bar{\mu}^*$ are the spurious outcome of a poor model choice. In each graph, the green horizontal line denotes the value of 0.7, which we would have anticipated from Gampe (2010). The red horizontal line depicts the level of the mortality that derived from the parameter estimates ($\hat{b}/\hat{\gamma}$, see Missov and Vaupel, 2015). With the exception of Spain (upper right panel), our estimates suggest a mortality plateau that is slightly higher than the expected 0.7 but lower than 0.8.

3.3 Estimates of the Mortality Plateau

Having identified the gamma-Gompertz model as providing a good fit to the data and being statistically superior to the other models according to the AIC, we next estimated the mortality plateau for Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany (West), Italy, Japan, Norway, Spain, Sweden, Switzerland, the United Kingdom and the United States. The empirical discrete density function for all years and countries for women and men is depicted in Figure 3 with gray vertical bars. According to our estimates, the actual level of the mortality plateau would be higher than 0.7. It would be slightly below a population hazard level of 1, which can be translated to a probability of dying of $1 - \exp(-1) = 0.6321206 \approx 63\%$. The red and blue lines depict estimated densities for females and males, respectively. These results suggest that the median of the estimated mortality plateaus is lower for women than for men. The median estimate for the plateau of the population hazard of males is about 1.2 or 70%. We would not claim that our results contradict Gampe's estimates: 573 out of the 637 individuals that formed the basis for Gampe's estimate of 0.7 were females (Gampe, 2010, p. 224). We would have also expected that the estimates are less dispersed. The variation in mortality plateaus for males is particularly striking.

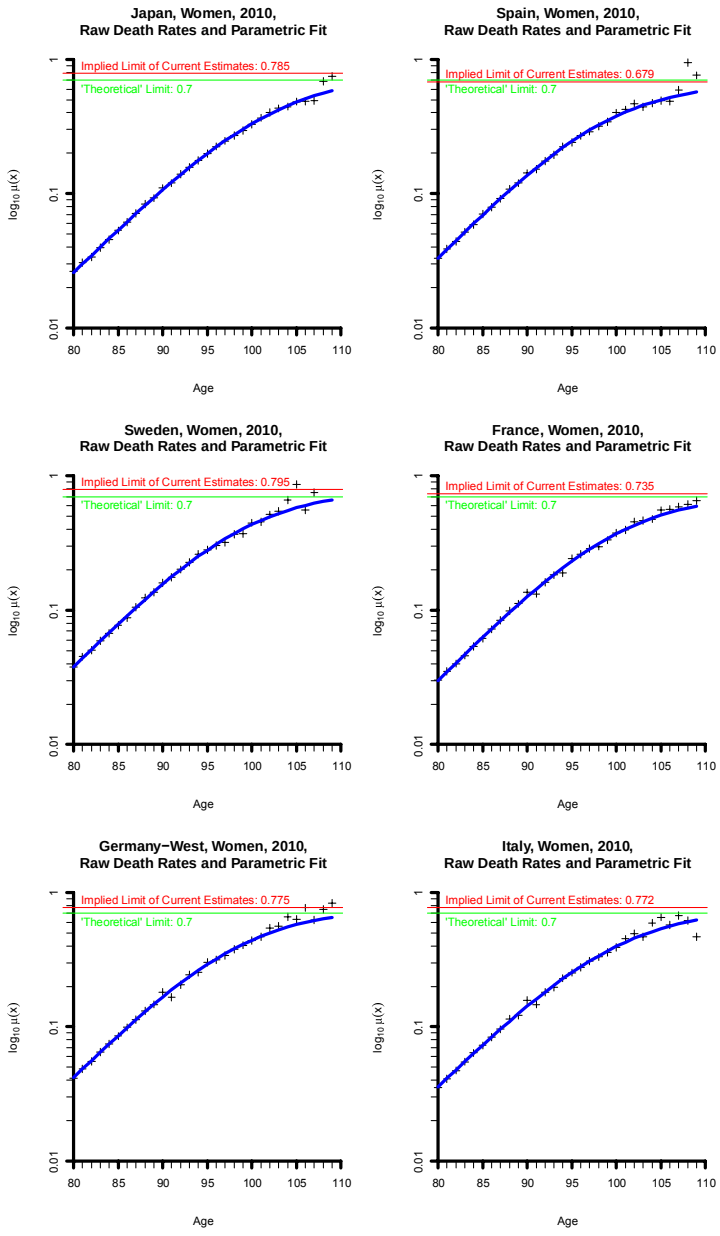

Figure 2: Raw Death Rates (+) and Fitted Force of Mortality (Blue Lines) at Ages 80–109 Note: For Japan, Spain, Sweden, France, Germany-West and Italy in 2010. Additional horizontal reference lines were included in green for Gampe's (2010) "theoretical" limits of 0.7 and in red for the ones obtained from our estimation. Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

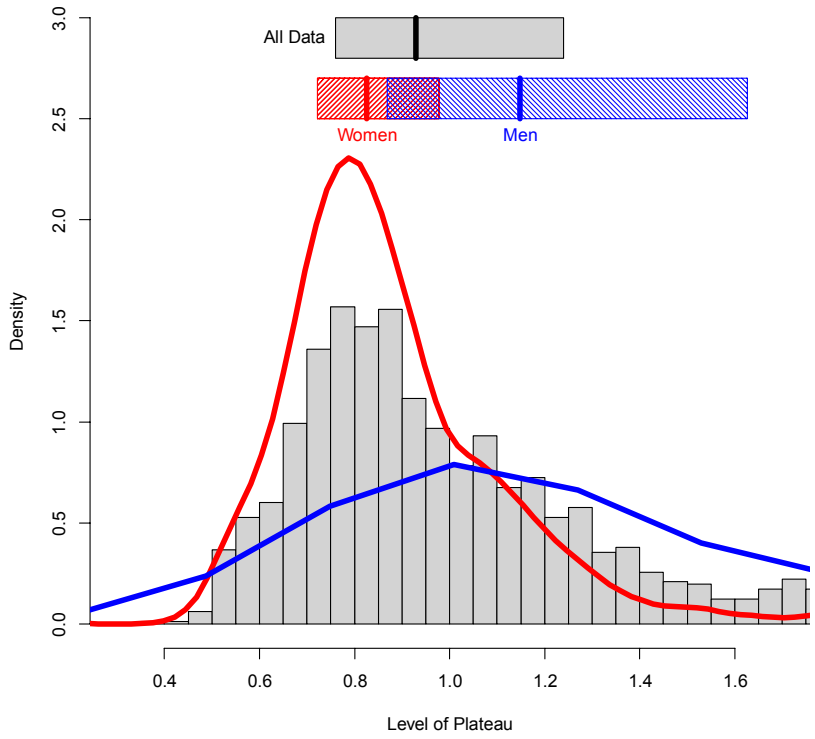

Figure 3: Empirical Density Function of Mortality Plateaus for Women and Men, 1960–2010 Note: Data for Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany (West), Italy, Japan, Norway, Spain, Sweden, Switzerland, United Kingdom and United States. Red curve is estimated density for women, and blue curve is estimated density for men for the same countries and years. Horizontal rectangles are interquartile ranges of mortality plateau for both sexes combined (gray), women (red) and men (blue). Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

Therefore, we decided to simulate data with a "true" signal of $b = 0.14$ and $\gamma = 0.2$, which results in a mortality plateau of $\bar{\mu}^* = b/\gamma = 0.14/0.2 = 0.7$ but different "population sizes." The simulations were repeated 1,000 times for each of the three population sizes of 1,000, 10,000, and 100,000 individuals. The results of the simulation are shown in

Figure 4. The median values in each of the three simulations are close to what we would have expected. We can also see that the variation around the median strongly depends on the population size. If only 1,000 individuals were alive at age 80, which is the beginning of the age interval for our estimations, "typical" values would range from about 0.58 to 0.92. The variation we have observed in our empirical data cannot be explained by small population size alone, though. Even in 1960, more than 10,000 women as well as men age 80 were alive in Sweden, one of the smallest countries we have analyzed.

That is why we analyzed the data by decade (see Figure 5) and by country (see Figure 6) separately for women and men. The results are inconclusive, however. If the mortality plateau had changed systematically from one decade to the next, we might have observed a decrease in the variation. With the exception of the last decade, we have seen a fairly stable median for women during the 40 years of analysis with little change in the variation. The lack of change in the variation is even more the case for males, as both panels of Figure 5 illustrate. Also, the analysis by country did not improve our understanding of the mortality plateau substantially. If each country had its own specific level of mortality, a large part of the variation observed in Figure 3 could have been easily explained. Unfortunately, this is not the case.

The level of the mortality plateau is the ratio of the slope parameter b and the variance of the gamma distribution γ. We investigated, therefore, whether the variation in the mortality plateau is mainly attributable to one of the two factors. Figure 7 shows a range of theoretical b values on the x-axis and of theoretical gamma values. Their ratios, i.e., the level of the respective mortality plateaus, are included as contour lines. The color images in the panels depict a two-dimensional density plot of 1,632 gamma-Gompertz estimates, which also formed the basis for Figure 3.[3] Shades of red denote relatively low densities, whereas blue, purple and violet colors show the areas of many estimates being close to each other. It seems that the variation in the level of the mortality plateau is primarily

[3] 51 calendar years for 16 countries, separately for women and men.

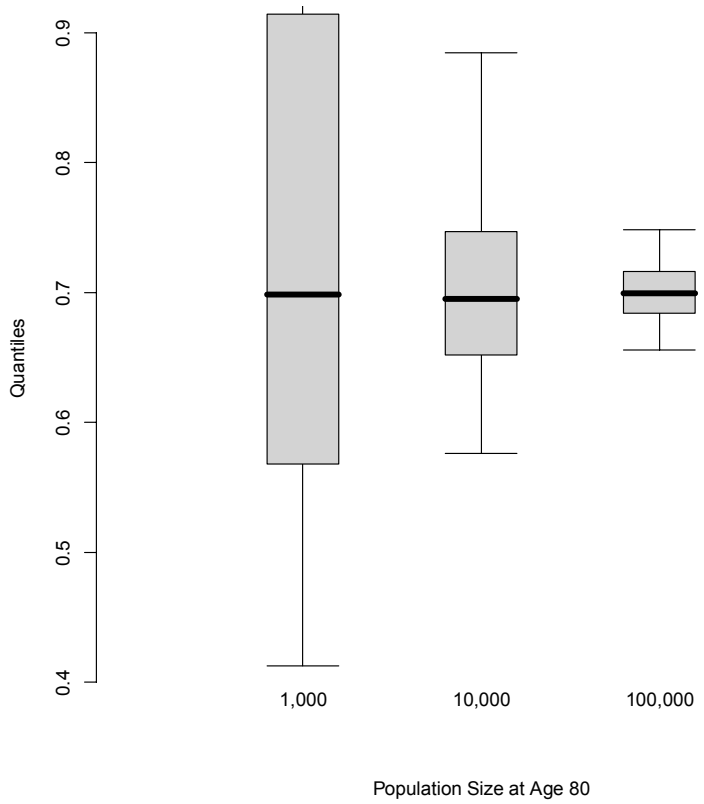

Figure 4: Boxplots of Simulation with Sample Sizes 1,000, 10,000 and 100,000 Where the "True Signal" Equals $\bar{\mu}^* = b/\gamma = 0.14/0.2 = 0.7$ Note: Simulations were repeated 1,000 times for each population size. The gray vertical bars depict the interquartile range, containing a solid black line for the median value. The "whiskers" of the boxplot represent the empirical 95% interval. Source: Own simulations.

due to variation in the estimates for γ, while the estimates for the slopes cover a much smaller range (0.10 to 0.15 for women; 0.07 to 0.12 for men).

We also analyzed another potential impact on our estimates: the numerical method employed. All our estimates were based on standard numerical methods, i.e., the Nelder-Mead algorithm, which is the default setting for R's general purpose optimizer `optim` (R

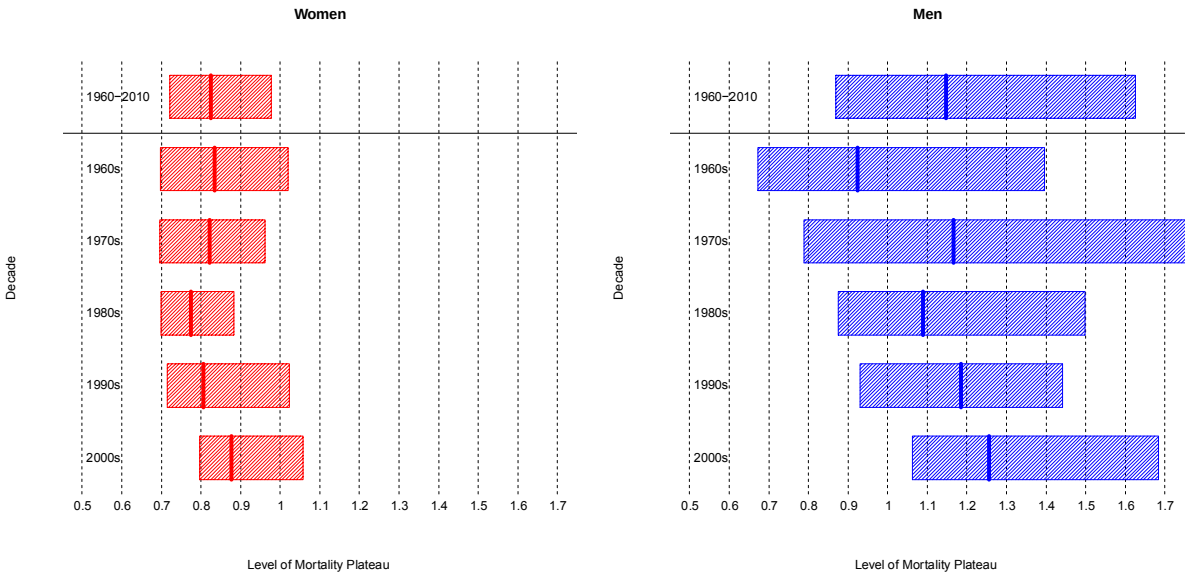

Figure 5: Interquartile Range and Median Values for Mortality Plateaus by Decade of Analysis, 1960–2010 Note: Data for women (left in red) and men (right in blue) in Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany (West), Italy, Japan, Norway, Spain, Sweden, Switzerland, United Kingdom and United States. Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

Core Team, 2015). Using the same set of countries with high data quality as in Figure 2, we compared the estimates of `optim` with a genetic algorithm as implemented in the R package `rgenoud` (Mebane, Jr. and Sekhon, 2011; Sekhon and Mebane, Jr., 1998), using population sizes of 1,000 and 100 generations. We used the same starting values of $b_0 = 0.1$, $M_0 = 85$, and $\gamma_0 = 0.3$, i.e., values that are in the same order of magnitude but not too close, for `optim` as well as `rgenoud`. The results are presented in Table 1 (page 16).

It is easy to see that maximizing the log-likelihood with the standard approach or with a genetic algorithm yields estimates that are nondistinguishable. The largest difference was 0.0046 for the modal age at death M. The parameters b and γ differed by 0.00042 or

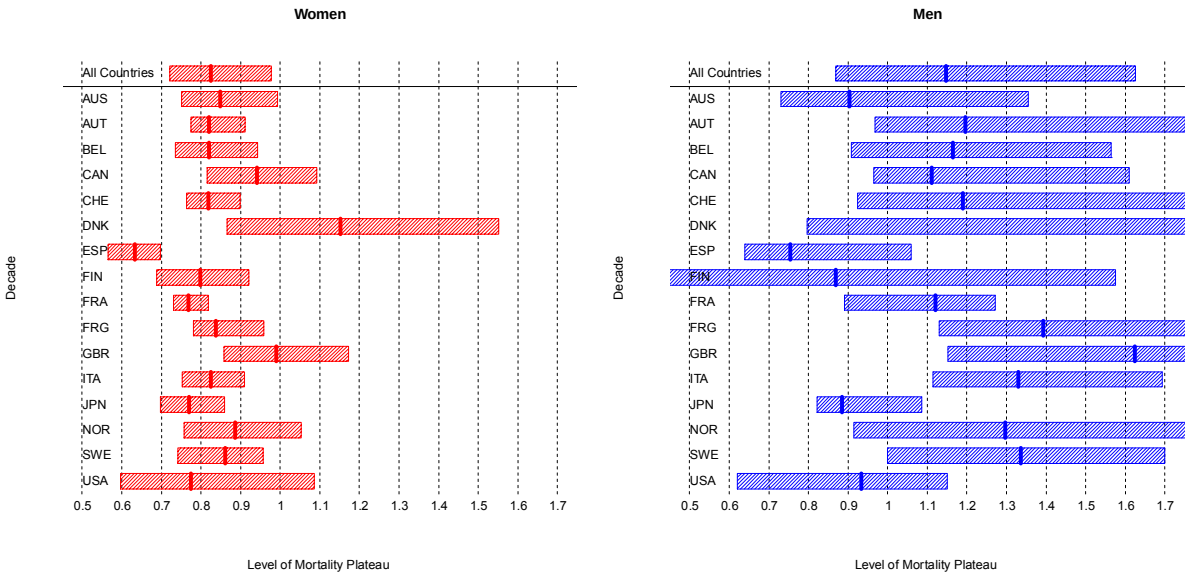

Figure 6: Interquartile Range and Median Values for Mortality Plateaus by Country, 1960–2010 Note: Data for women (left in red) and men (right in blue). Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

less. Hence, the large variation in mortality plateaus cannot be attributed to non- or ill-converging numerical methods. But we discovered that with the exception of Spain, the remaining five countries estimated the variance to be close to 0.20 and also little variation for the slope.

We therefore decided to estimate the gamma-Gompertz model for countries with the best grade for data quality (Jdanov et al., 2008), with high life expectancy and a population size of at least 10 million for the years 2005–2010, to be safe to have sufficient numbers at old ages to not run into numerical problems. The final set of countries consisted of Belgium, France, Germany, West Germany, Italy and Japan. We added the estimates for b and γ for these countries in these years as white dots to the density plot. The results are given in Figure 8 (page 17). Those recent estimates for countries with low mortality, excellent data quality and large numbers suggest that there seems to be in fact a mortality plateau—

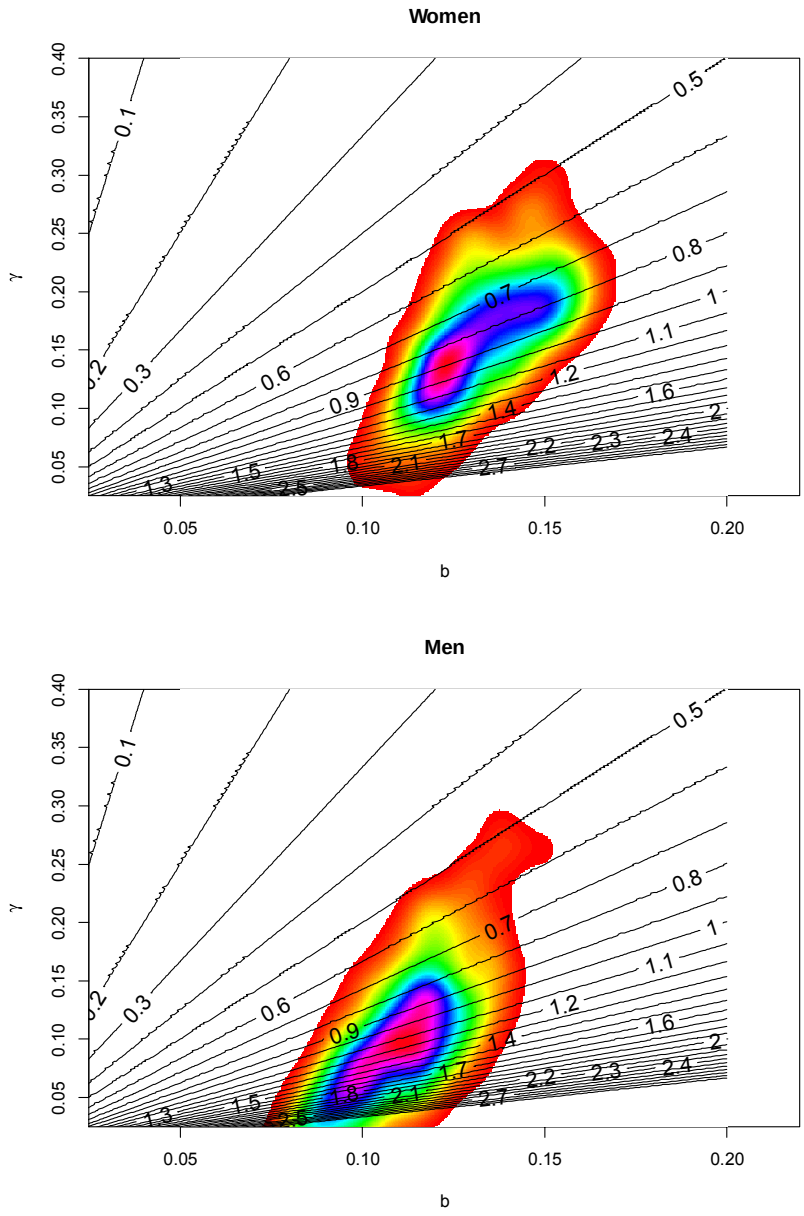

Figure 7: Two-Dimensional Density Plot of Estimates for b and γ with Corresponding Levels of the Mortality Plateau for Women and Men Note: Plateau levels shown as contour lines for women (upper panel) and men (lower panel). Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

Table 1: Parameter Estimates for Gamma-Gompertz Model for Women, Using Two Different Estimation Strategies Note: Data for women in Japan, Spain, Sweden, France, Germany (West) and Italy in 2010 (see Figure 2 for actual fit). For estimation strategies, `optim` refers to standard numerical optimization in R, while `genetic` employs the genetic optimization approach of package `rgenoud` (Mebane, Jr. and Sekhon, 2011; Sekhon and Mebane, Jr., 1998) with a population size of 1,000 and 100 generations. Both approaches used the same starting values of $b = 0.1$, $M = 85$, and $\gamma = 0.3$.

Country	**Estimated Parameter and Estimation Approach**					
	b		M		γ	
	`optim`	`genetic`	`optim`	`genetic`	`optim`	`genetic`
Japan	0.15350	0.15365	91.40880	91.40417	0.19548	0.19680
Spain	0.16006	0.15998	89.55558	89.55333	0.23565	0.23524
Sweden	0.15942	0.15948	88.75105	88.75217	0.20049	0.20069
France	0.15914	0.15925	90.27741	90.27510	0.21657	0.21707
Germany (West)	0.15724	0.15723	88.09153	88.09380	0.20280	0.20282
Italy	0.15433	0.15428	89.20267	89.20105	0.19981	0.19957

at least for females—that is located at about 0.8 (median: 0.799; IQR: 0.775–0.829). This translates into a probability of dying of 0.55%. The picture remains blurred for males. Even the reduction to the set of countries for which we would expect highly reliable data did not result in an estimate for a mortality plateau with little variation around it. The median value for the population hazard was 1.246 (equivalent to a probability of dying of about 71%) with an interquartile range of 1.1324–1.5487, mainly due to considerable differences in the estimates for γ.

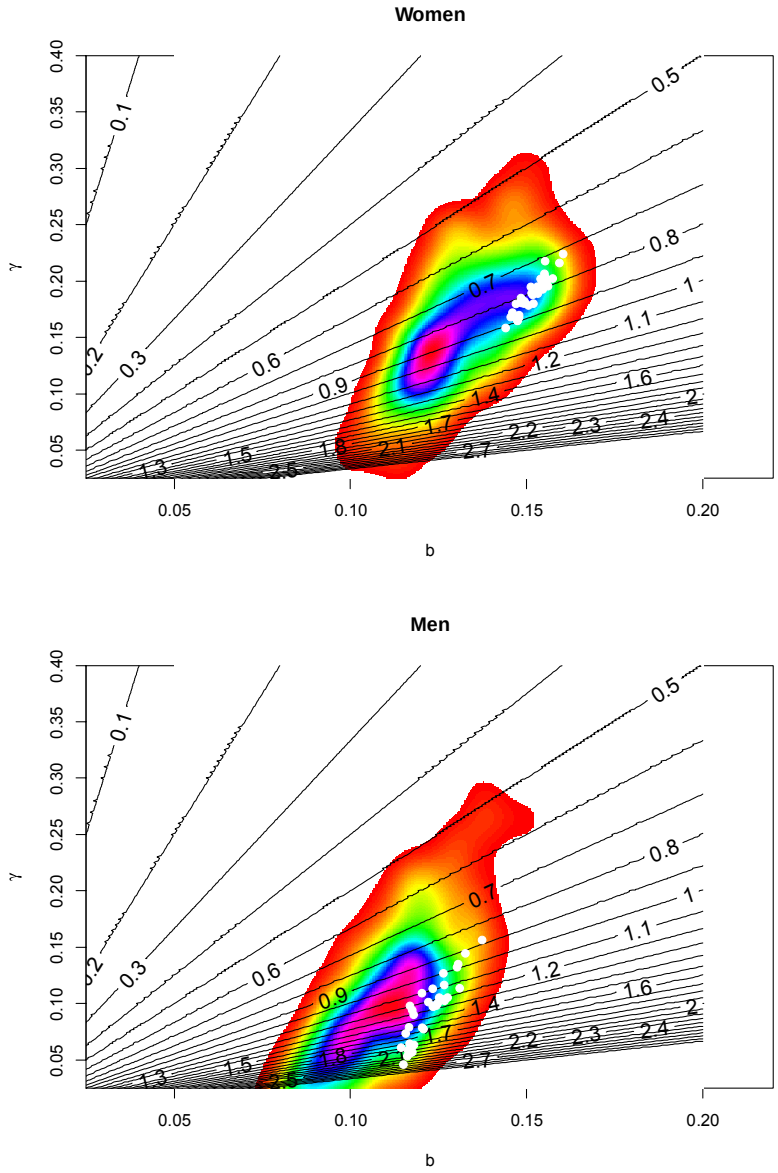

Figure 8: Two-Dimensional Density Plot of Estimates for b and γ with Corresponding Levels of the Mortality Plateau and Estimates by Country Note: Plateau levels shown as contour lines for women (upper panel) and men (lower panel). White dots represent estimates for Belgium, France, Germany, Germany-West, Italy and Japan. Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

4 Summary

The goal of our analysis was to test whether we can find support for a plateau of the population hazard at a level of 0.7 as estimated by Gampe (2010) with a nonparametric approach. Recent theoretical, mathematical and statistical findings suggest that "the only demographically meaningful multiplicative model that holds *at* the plateau is the gamma-Gompertz" (Missov and Vaupel, 2015, p. 69).

We estimated these gamma-Gompertz models with data from the Human Mortality Database. With about a level for the plateau of 0.8, our results for women were slightly higher than the expected 0.7. The mortality plateau for males was even higher, though. We estimated a level of approximately 1.2. Translated into probabilities of dying, we could not replicate the chance of the toss of a fair coin but about 55% for women and 70% for men. Since Gampe's original estimates were based on primarily female data, we were not surprised by the difference between women and men. More worrisome was the large variation in our estimates for 16 countries for the years 1960–2010. Differentiating by country or by decade did not provide any insights why there was such a large variation in mortality plateaus. Also, small population sizes were not able to explain the large variation as simulation studies have shown.

Only when we restricted ourselves to large countries (more than 10 million inhabitants), high data quality according to Jdanov et al. (2008), low mortality and recent years to have enough survivors at advanced ages were we able to narrow down the corridor for females. Our mortality plateau of 0.8 for women is slightly higher than the one predicted by Gampe (2010) with a slope parameter b of about 0.14–0.15 and a γ parameter slightly below 0.2. Our refined approach for males was less successful. Even after selecting highly reliable data only, we estimated still large variation for the mortality plateau, mainly caused by γ estimates ranging from 0.05 to 0.15.

To conclude: We would claim that there is, indeed, support for a mortality plateau for

women. At 0.8, it is slightly higher than suggested by Gampe (2010). If a mortality plateau exists for males, it is higher than for females, but our estimates were not convincing. This could be the outcome of there being still very few old men alive at very advanced ages. Hence, a mortality plateau might not exist at all, or we have to wait until enough centenarians and semi-supercentenarians are male to have more robust estimates.

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B.2. Appendix paper II: Decomposing changes in life expectancy: Compression versus shifting mortality

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Decomposing changes in life expectancy: Compression versus shifting mortality

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Demographic Research: Volume 33, Article 14
Research Article

Decomposing changes in life expectancy: Compression versus shifting mortality

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Abstract

BACKGROUND

In most developed countries, mortality reductions in the first half of the 20th century were highly associated with changes in lifespan disparities. In the second half of the 20th century, changes in mortality are best described by a shift in the mortality schedule, with lifespan variability remaining nearly constant. These successive mortality dynamics are known as compression and shifting mortality, respectively.

OBJECTIVE

To understand the effect of compression and shifting dynamics on mortality changes, we quantify the gains in life expectancy due to changes in lifespan variability and changes in the mortality schedule, respectively.

METHODS

We introduce a decomposition method using newly developed parametric expressions of the force of mortality that include the modal age at death as one of their parameters. Our approach allows us to differentiate between the two underlying processes in mortality and their dynamics.

RESULTS

An application of our methodology to the mortality of Swedish females shows that, since the mid-1960s, shifts in the mortality schedule were responsible for more than 70% of the increase in life expectancy.

CONCLUSIONS

The decomposition method allows differentiation between both underlying mortality processes and their respective impact on life expectancy, and also determines when and how one process has replaced the other.

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1. Background

Human mortality has undergone remarkable declines over the years. The increase in life expectancy is probably the best expression for the dramatic mortality decline in the last 170 years (Oeppen and Vaupel 2002). Improvements in living conditions, nutrition and medicine are among the main reasons for this development (Riley 2001; Oeppen and Vaupel 2002). These changes in economic, social, and sanitary conditions first triggered an important decline in infant, child, and early adult mortality, which contributed to the reduction in lifespan disparities (Wilmoth and Horiuchi 1999; Edwards and Tuljapurkar 2005; Vaupel, Zhang, and van Raalte 2011). As individuals became more homogeneous in their ages at death, a compression of the distribution of deaths in a more narrow age-interval was observed in many low-mortality countries in the first half of the twentieth century (Fries 1980; Wilmoth and Horiuchi 1999; Kannisto 2000, 2001; Cheung et al. 2009). Fries (1980) hypothesized that this dynamic can be interpreted as a compression of deaths against the upper limit of the human lifespan. Assuming a nearly negligible role for premature mortality, he stated the limit of the average age at death as approximately 85 years, with 95% of all deaths occurring in an age range of 4 years deviation (Fries 1980). The "compression of mortality hypothesis" motivated a rich discussion on the occurrence and interpretation of this development. Several studies provided evidence for a compression, but emphasized that the achieved mortality levels differ substantially from Fries' predictions (Nusselder and Mackenbach 1996; Wilmoth and Horiuchi 1999; Cheung et al. 2005).

After the period of strong compression, low-mortality countries entered a new era of change. Since the second half of the twentieth century, the main contributions to the increase in average age at death shifted from infant and early adult ages to old and very old-ages (Christensen et al. 2009). This generated changes in the mechanisms behind the increase in life expectancy (Wilmoth and Horiuchi 1999; Edwards and Tuljapurkar 2005; Smits and Monden 2009). The new mechanism behind improvement in life expectancy is best illustrated by a shift in the distribution of death toward older ages with a shape remaining nearly constant (Yashin et al. 2001; Bongaarts 2005; Cheung et al. 2005; Cheung and Robine 2007; Canudas-Romo 2008). Vaupel (1986), Vaupel and Gowan (1986) and Bongaarts (2005) were among the first to articulate the idea of shifting mortality. Canudas-Romo (2008) deepens this idea by studying the variability around and the change of the modal age at death. He finds that over time mortality shifts to higher ages, with approximately constant variability in age at death. He concludes that the shifting mortality pattern might be the new dynamic behind mortality improvements, subsequent to the compression process.

The ages at which mortality reductions occur tend to determine the dominating mortality dynamic: compression or shift. Compression is more pronounced when mortality reductions occur at very young and adult ages (Nusselder and Mackenbach 1996;

Wilmoth and Horiuchi 1999; Kannisto 2000; Cheung et al. 2005). On the other hand, shifting mortality requires changes at old and very old-ages (Canudas-Romo 2008). Vaupel, Zhang, and van Raalte (2011) report relatively stable variability patterns for survivors beyond age 50 in the last 100 years. Engelman, Caswell, and Agree (2014) and Engelman, Canudas-Romo, and Agree (2010), however, provide evidence for a modest expansion of lifespan variability for survivors at older ages, resulting from mortality improvement at these same ages.

The measurement of compression and shifting mortality is an important issue, as both dynamics translate differently into survival, mortality density and hazard distributions (Wilmoth and Horiuchi 1999). Alterations are, however, visible in all three functions due to their interrelation. For instance, in a mortality compression context, the survival curve becomes more rectangular with increasing concentration of deaths at old-age, which is a well-known phenomenon called rectangularization (Nusselder and Mackenbach 1996; Wilmoth and Horiuchi 1999; Cheung et al. 2005). Simultaneously, the old-age bulk of deaths in the distribution of death becomes more pronounced, thereby reducing variability of the age at death. In the hazard distribution, the slope becomes steeper, with mortality reductions being more pronounced at younger ages (Wilmoth and Horiuchi 1999; Robine 2001).

In a shifting mortality context, these three functions also undergo transformations. The downward slope of the survival curve will shift to higher ages with an equal shape. Similarly, the density distribution will shift towards older ages with a shape also remaining constant. In the hazard distribution, the same pattern requires a constant slope depicted by a parallel shift of the logarithmic force of mortality toward higher ages (Bongaarts 2005; Canudas-Romo 2008). In this context, Bongaarts (2005) suggested fixing the shape parameter of mortality models and assumed that only scale and background parameters can vary over time. Vaupel (2010) also describes a postponement of senescence rather than a fundamental change of the age-pattern of mortality for the period starting around 1950.

In the assessment of the shifting mortality period, the modal age at death has been an extensively used indicator. By shifting the modal age at death towards older ages, the deaths around this age move along with it (Canudas-Romo 2008). This indicator also has several advantages in the investigation of survival at old-ages. First, it is nonsensitive to mortality changes at younger ages. Second, it reflects the most common lifespan. Third, a change of the modal age can only be realized if there are pulling forces, meaning mortality improvement at ages older than the mode (Kannisto 2000; Canudas-Romo 2010). In fact, the modal age at death has shown an accelerated pace of increase since the onset of the old-age mortality decline (Kannisto 2000; Wilmoth and Robine 2003; Canudas-Romo 2008). Since the beginning of the 21st century, this indicator has received increasing attention and has become a key indicator of lifespan, especially since longevity extension became determined by adult and old-age mortality (Kannisto 2000,

2001; Bongaarts 2005; Cheung and Robine 2007; Canudas-Romo 2008, 2010; Ouellette and Bourbeau 2011; Horiuchi et al. 2013).

Therefore, compression and shifting mortality are observed respectively by changes in the variability of the age at death and in the modal age at death. Both dynamics also have different implications regarding changes in mortality: the former reflects changes in lifespan disparities, while the latter provides information about changes in the timing of mortality.

Considering the two periods of change in mortality development, two questions arise. First, what is the impact of compression and shifting mortality dynamics on the increase of life expectancy over time? Second, how and to what extent did one process replace the other? Additionally, considering the impact of child and young adult mortality reductions on the appearance of compression, one might further ask, if only adult and old-ages mortality is analyzed, how does the impact of both dimensions change?

To approach these questions, a new methodology to study changes in compression and shifting mortality over time and their effect on life expectancy is presented. We quantify the gains in life expectancy due to changes in the timing of mortality and changes in lifespan disparities, respectively. Using newly developed parametric expressions of the force of mortality (Horiuchi et al. 2013; Missov et al. 2015), we decompose the change in life expectancy between two distributions by the contribution of a shift in the modal age at death and a change in variability of the age at death.

This paper is divided into four sections, with this background as the first section. In the following section, we introduce the decomposition methodology, at first in general terms and then for the Gompertz, Gompertz-Makeham and Siler models. The third section presents an illustration of the methodology applied to discrete data, followed by the fourth section, in which we present our conclusions.

2. Methods and data

2.1 Decomposing life expectancy

In order to explain the dynamics behind changes in mortality, demographers have developed several techniques to decompose changes in life expectancy by different components of mortality, such as ages and causes of death. Some methods focus on discrete differences between two life expectancies (Pollard 1982; Arriaga 1984; Pressat 1985; Andreev, Shkolnikov, and Begun 2002; Firebaugh et al. 2014) while others consider continuous changes (Vaupel 1986; Keyfitz 1977; Vaupel and Canudas-Romo 2003; Beltrán-Sánchez, Preston, and Canudas-Romo 2008; Horiuchi, Wilmoth, and Pletcher 2008). We follow the latter approach of a continuous decomposition of changes in life expectancy by vari-

ability and shifting effects using a recent expression of the Gompertz mortality model.

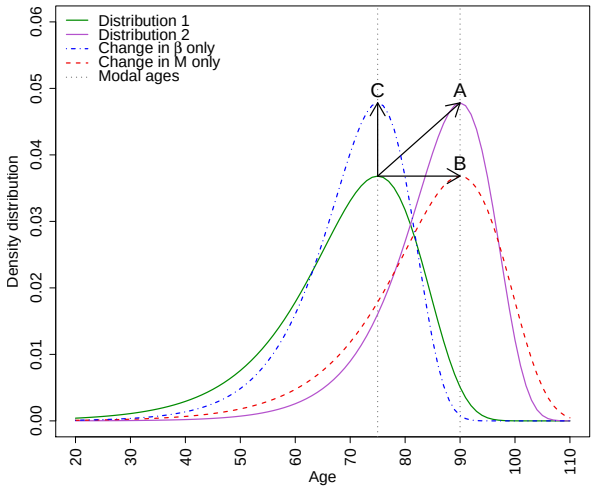
Figure 1 shows the distribution of deaths for Gompertz parameters under two scenarios. It illustrates how changes in mortality can be decomposed into effects due to changes in variability and the shifting of mortality. Assuming a general change of mortality between the two distributions (in Figure 1 as the arrow denoted as A), the shifting effect is the hypothetical change resulting only if the modal age at death (M) would have changed between those two distributions (in Figure 1 as arrow B). The variability effect is the hypothetical change produced only if the slope of the hazard function (β) changes from one distribution to another (in Figure 1 as arrow C). The latter transformation C, of changing the slope of the hazard distribution, also changes the shape of the density distribution, and thus their variability (Wilmoth 1997).

Changes in life expectancy at birth over time (denoted as $\dot{e}_{0,t}$) can thus be decomposed into two components

$$\dot{e}_{0,t} = \Delta\beta + \Delta M, \tag{1}$$

where $\Delta\beta$ and ΔM are the gains in life expectancy resulting from changes in the shape parameter and modal age at death, respectively. In the following section we present the methodology of the decomposition for the Gompertz force of mortality and then generalize it to other parametric functions of mortality.

Figure 1: **Illustration of the shifting and variability effects in the density function of the distribution of deaths for simulated data from a Gompertz model with a combination of shape parameters $\beta_1 = 0.10$ and $\beta_2 = 0.13$ and modal ages at death $M_1 = 75$ and $M_2 = 90$**



2.2 Decomposing senescent mortality: Gompertz

Gavrilov and Gavrilova (1991) defined the Gompertz (1825) law of mortality as one of the most successful models expressing mathematically the senescent age-pattern of mortality. In this article, we refer to senescent mortality as the increase over age in the force of mortality occuring after a certain age, representing aging and physiological deterioration (Bongaarts and Feeney 2002; Bongaarts 2005; Horiuchi et al. 2013). The Gompertz approach allows a good approximation of adult mortality patterns over age and time for many countries. However, the Gompertz model does not fit infant, child and oldest-old mortality well. Other parametric models, such as the Makeham (1860) and Siler (1979), have addressed some of these problems by including additional parameters capturing background and infant mortality. The Gompertz model is, however, broadly used to describe the distribution of adult death from age 30 to 90, having the advantage of being simple and offering a good fit to senescent mortality. The decomposition methodology introduced here will be presented through the Gompertz model, but it will be demonstrated that the method can be applied to other parametric models.

It has been shown by Horiuchi et al. (2013) and Missov et al. (2015) that the hazard rate as expressed by the Gompertz model can be rewritten using the modal age at death instead of the timing parameter α_t as

$$\mu_{x,t} = \alpha_t e^{\beta_t x} = \beta_t e^{\beta_t (x - M_t)}, \tag{2}$$

where β_t is the shape parameter at time t of the Gompertz hazard function $\mu_{x,t}$, and M_t is the modal age at death. This parametrization has some advantages: 1) the parameter M_t has a clearer interpretation than α_t (Horiuchi et al. 2013), and 2) there is a lower correlation between the parameters when the Gompertz is expressed using the modal age at death (Missov et al. 2015).

The parametrization presented in equation (2) also gives a starting point for decomposing changes in life expectancy due to changes in variability and shifting mortality. Shifting mortality is observed through changes in the modal age at death, which is captured by the parameter M_t. Additionally, as presented in Appendix A, it can analytically be shown that the shape parameter β_t is the main carrier of variability changes.

Let a dot on top of a variable denote its derivative with respect to time (Vaupel and Canudas-Romo 2003). The change over time in the force of mortality ($\dot{\mu}_{x,t}$) can be decomposed into respective components of change for the shape ($\dot{\beta}_t$) and the mode ($\dot{M}_t$):

$$\dot{\mu}_{x,t} = \dot{\beta}_t \left[\mu_{x,t}(\frac{1}{\beta_t} + x - M_t)\right] - \dot{M}_t \ [\beta_t \mu_{x,t}]. \tag{3}$$

Equation (3) can be expressed in more general terms to be used in other models of mortality. The components of change for the shape ($\dot{\beta}_t$) and modal age at death ($\dot{M}_t$) are each

multiplied by a weighting function of the corresponding hazard rate, denoted as $f_i(\mu_{x,t})$, with i corresponding to the parameters β and M,

$$\dot{\mu}_{x,t} = \dot{\beta}_t \; f_\beta(\mu_{x,t}) \; - \dot{M}_t \; f_M(\mu_{x,t}). \tag{4}$$

As with the hazard distribution, we can derive the time change of life expectancy. In general terms, life expectancy at birth is expressed as

$$e_{0,t} = \int_0^\omega l_{a,t} \, da,$$

where $l_{a,t}$ is the survival function and the radix of the population is one. Therefore, changes in life expectancy at birth through time ($\dot{e}_{0,t}$) can be expressed by:

$$\dot{e}_{0,t} = \int_0^\omega \dot{l}_{a,t} \, da = - \int_0^\omega l_{a,t} \int_0^a \dot{\mu}_{x,t} \, d_x \, d_a, \tag{5}$$

where $\dot{l}_{a,t}$ is the time derivative of the survival function $l_{a,t}$. By substituting equation (4) in equation (5), we can estimate the change in life expectancy at birth due to changes in the modal age at death and changes in the shape parameter as:

$$\dot{e}_{0,t} = \underbrace{-\dot{\beta}_t \int_0^\omega l_{a,t} \int_0^a f_\beta(\mu_{x,t}) \, d_x \, d_a}_{\Delta\beta} + \underbrace{\dot{M}_t \int_0^\omega l_{a,t} \int_0^a f_M(\mu_{x,t}) \, d_x \, d_a}_{\Delta M}. \tag{6}$$

The first term in equation (6) represents the gain in life expectancy resulting from a change in variability ($\Delta\beta$), corresponding to a compression pattern, while the second term is the gain in life expectancy produced by a shift in the modal age at death (ΔM), indicating a shifting pattern. These are the equivalent terms of equation (1) in the Gompertz model.

Equations (4) and (6) allow further generalizations to other parametric models expressing senescent mortality using the modal age at death (M) and a shape parameter (β). Horiuchi et al. (2013) present this parametrization for the Logistic and Weibull models. Table 1 includes the elements of the decomposition equations for the Gompertz, Logistic and Weibull models.

As mentioned previously, the Gompertz model does not fit young age mortality well, and tends to fit mortality over age 30 better. Hence, for the application of the Gompertz decomposition, we will start our life table at age 30, and $e_{0,t}$ will represent the life expectancy at age 30, indicated as $e_{30,t}$ in the tables.

Table 1: Hazard (μ_x), modal age at death (M), life expectancy at birth (e_0) and decomposition weights ($f_\beta(\mu_{x,t})$ and $f_M(\mu_{x,t})$) for three mortality models

	Gompertz	**Logistic**	**Weibull**
$\boldsymbol{\mu_x(\alpha,\beta)}$	$\alpha e^{\beta x}$	$\frac{e^{\alpha+\beta x}}{1+e^{\alpha+\beta x}}$	$\alpha\beta x^{\beta-1}$
$\boldsymbol{\mu_x(M,\beta)}$	$\beta e^{\beta(x-M)}$	$\frac{\beta e^{\beta(x-M)}}{1+\beta e^{\beta(x-M)}}$	$\frac{(\beta-1)x^{\beta-1}}{(M)^\beta}$
$\boldsymbol{M}$	$\frac{ln(\beta)-ln(\alpha)}{\beta}$	$\frac{ln(\beta)-\alpha}{\beta}$	$\left(\frac{(\beta-1)}{\alpha\beta}\right)^{\frac{1}{\beta}}$
$\boldsymbol{e_0}$	$\int_0^\omega e^{-e^{-\beta M}(e^{\beta a}-1)}da$	$\int_0^\omega \left[\frac{\beta e^{-\beta M}+1}{\beta e^{\beta(a-M)}+1}\right]^{\frac{1}{\beta}} da$	$\int_0^\omega e^{-\frac{(\beta-1)a^\beta}{\beta M^\beta}} da$
$\boldsymbol{f_\beta(\mu_{x,t})}$	$\mu_{x,t}(\frac{1}{\beta_t}+x-M_t)$	$\frac{\mu_{x,t}(\frac{1}{\beta_t}+x-M_t)}{1+\beta_t e^{\beta_t(x-M_t)}}$	$\mu_{x,t}\left[\frac{1}{(\beta_t-1)}+ln(\frac{x}{M_t})\right]$
$\boldsymbol{f_M(\mu_{x,t})}$	$\beta_t\mu_{x,t}$	$\frac{\beta_t\mu_{x,t}}{1+\beta_t e^{\beta_t(x-M_t)}}$	$\mu_{x,t}\frac{\beta_t}{M_t}$

Note: To simplify the equations, the time component (t) was not added as subscript to the parameters α_t, β_t and M_t in the first four lines of the table. However, the parameters can also vary over time (t) in these equations.

2.3 Extending the model beyond senescent mortality

With the previous methodology, only senescent mortality can be decomposed. The decomposition is thus limited to adult and old-age mortality, and might bring only limited understanding of mortality changes over time. As mentioned previously, compression of mortality has been strongly linked to reductions in infant, child and early adult mortality, which is not considered when decomposing the Gompertz model. Modeling mortality at all ages needs more complex models, and additional parameters often need to be added.

Equation (1) can be generalized to allow the inclusion of parameters other than β and M, as

$$\dot{e}_{0,t} = \sum_{i=1} \Delta_i, \tag{7}$$

where Δ_i is the change in life expectancy at birth due to a change in the parameter i.

2.3.1 Gompertz-Makeham

A Makeham (1860) variant can be added to each of the models presented in Table 1 (Horiuchi et al. 2013). Assuming that the modal age at death (M_t) estimated by the Gompertz model in equation (2) applies to the Gompertz-Makeham model, the hazard function can be expressed as

$$\mu_{x,t} = c_t + \beta_t e^{\beta_t (x - M_t)}, \tag{8}$$

where c_t is the Makeham term. Adding the parameter c_t improves the fit of the Gompertz function at younger ages, but still without capturing the decrease in infant mortality. The Makeham term is an age-independent component which captures the extrinsic or "background" mortality risk. The Makeham term has a more influential effect at younger ages and is often associated with adult or early adult mortality, which is especially important for the variability effect.

Equivalent to the decomposition presented in equation (6), we can estimate the change in adult life expectancy due to changes in the different parameters of the Gompertz-Makeham model using equation (5). As expressed by equation (7), change in life expectancy is then estimated as

$$\begin{aligned} \dot{e}_{0,t} = & \underbrace{- \dot{c}_t \int_0^\omega l_{a,t}\, a\, da}_{\Delta c} \underbrace{- \dot{\beta}_t \int_0^\omega l_{a,t} \int_0^a [e^{\beta_t (x - M_t)} (1 + \beta_t (x - M_t))]\, dx\, da}_{\Delta \beta} \\ & + \underbrace{\dot{M}_t \int_0^\omega l_{a,t} \int_0^a [\beta_t^2 e^{\beta_t (x - M_t)}]\, dx\, da}_{\Delta M}, \end{aligned} \tag{9}$$

where $\dot{c}_t$ is the change in the background mortality level, $\dot{\beta}_t$ is the change in the rate of mortality increase over age and $\dot{M}_t$ is the change in the modal age at death.

2.3.2 Siler

The Siler (1979) model extends the Gompertz model by including two additional terms, capturing both the decrease over ages of infant mortality and the "background" mortality risk. By using the Gompertz model with the parametrization presented in equation (2), we can express the Siler model as

$$\mu_{x,t} = \alpha_t \, e^{-b_t x} + c_t + \beta_t e^{\beta_t (x - M_t)}, \tag{10}$$

where α_t and c_t are timing parameters for infant and background mortality, the parameters b_t and β_t are the constant rates of mortality change over age for infant and senescent mortality, respectively, and M_t is the modal age at death. By including the infant and background parameters, the Siler model provides a more detailed estimation of the variability and shifting effect by modeling mortality at all ages.

Decomposition of changes in the Siler model is expressed by changes in 5 different parameters: $\dot{\alpha}_t$ is the change with respect to t in the initial level of mortality (age 0), $\dot{b}_t$ is the change in the rate of infant mortality decrease over age, $\dot{c}_t$ is the change in the background mortality level, $\dot{\beta}_t$ is the change in the rate of mortality increase over age for senescent mortality, and $\dot{M}_t$ is the change in the modal age at death.

As generally presented in equation (7), the gain in life expectancy at birth for the Siler model is estimated by

$$\begin{aligned}
\dot{e}_{0,t} = & - \underbrace{\dot{\alpha}_t \int_0^\omega l_{a,t} \int_0^a [e^{-b_t x}] \, dx \, da}_{\Delta\alpha} + \underbrace{\dot{b}_t \int_0^\omega l_{a,t} \int_0^a [\alpha_t e^{-b_t x} x] \, dx \, da}_{\Delta b} \\
& - \underbrace{\dot{c}_t \int_0^\omega l_{a,t} \, a \, da}_{\Delta c} - \underbrace{\dot{\beta}_t \int_0^\omega l_{a,t} \int_0^a [e^{\beta_t (x - M_t)} (1 + \beta_t (x - M_t))] \, dx \, da}_{\Delta\beta} \\
& + \underbrace{\dot{M}_t \int_0^\omega l_{a,t} \int_0^a [\beta_t^2 e^{\beta_t (x - M_t)}] \, dx \, da}_{\Delta M} .
\end{aligned} \tag{11}$$

There are, however, some implications of adding the Makeham term and the Siler infant mortality term to the Gompertz parametrization presented in equation (2). The parameter M, reflecting the modal age at death evaluated from senescent mortality only, could differ from the modal age at death evaluated from the total mortality. Horiuchi et al. (2013), however, found that the modal age at death for senescent mortality, when adding a Makeham term to equation (2), is nearly equal to the modal age at death for total mortality. We found similar results with the Siler model. For example, the total modal age at death for Swedish female mortality in 2010 fitted with a Siler model was 88.46, and the senescent modal age at death was 88.49. As the mortality level at old-ages tends to be determined by senescent mortality, with only limited influence from infant and background mortality, the senescent and total modal ages at death will generally be similar (Horiuchi et al. 2013). It is important to recall that the modal age at death is determined by old-age mortality only. To help understand the role of young age mortality on shifting and compression, we decided to overlook these small differences. In the following sections, we

will refer to the senescent modal age at death as the modal age at death.

When using the Gompertz model, the variability effect is captured by the parameter β_t (Appendix A) and the shifting effect by the parameter M_t. However, with a Gompertz-Makeham model or a Siler model, more parameters will influence variability changes. Canudas-Romo (2010) analytically demonstrated that, in a mortality declining scenario, as the one experienced in developed countries, the mode will be maintained when reduction of mortality occurs at younger ages than the modal age at death. Using a Siler model, Engelman, Caswell, and Agree (2014) showed that improvement in childhood components of mortality (α_t and b_t) and in background mortality parameter (c_t) influenced lifespan variability reduction. The first four terms of the above equation would then have an impact on variability reduction. The variability effect could then be divided into four distinct effects: $\dot{\alpha}_t$, $\dot{b}_t$, $\dot{c}_t$ and $\dot{\beta}_t$. The shifting effect is still captured by $\dot{M}_t$. This partition between the five Siler parameters emphasizes the impact of changing mortality at young ages on lifespan disparities, in contrast with the effect of mortality reductions at older ages on shifting mortality.

2.4 Data

The data source used in this study is the Human Mortality Database (HMD: http://www.mortality.org). The HMD (2015) compiles census and vital statistics information for the populations of entire countries. The HMD has high quality historical mortality data for industrialized countries; the data series are constructed according to a common protocol, making the HMD a unique comparison tool. For our illustrations, data for all the HMD countries, excluding Eastern European countries, have been used for years 1900 to 2010 (Table 2). We justify the data exclusion because there are different age-patterns of mortality in the excluded countries than to those included in the illustrations in recent decades. Nevertheless, our methodology can easily be extended to those countries although with different mortality parameters.

The decomposition is applied to the mortality of Swedish females and to the average female mortality in the selected HMD countries[4]. The Gompertz, Gompertz-Makeham and Siler models are fitted to observed mortality trends using a Poisson log-likelihood procedure. The estimation procedures of derivatives such as those in equations (6) to discrete data are presented in Appendix B.

[4]The parameters of the mortality models are estimated for each country independently and then averaged over all countries (with equal weight) to obtain the HMD average.

Table 2: Selected HMD countries and years with available data used for the illustration

Country	Years	Country	Years
Australia	1925-2010	Japan	1947-2010
Austria	1947-2010	Luxembourg	1960-2009
Belgium	1900-2010	Netherlands	1900-2009
Canada	1921-2010	New Zealand	1948-2008
Chile	1992-2005	Norway	1900-2009
Denmark	1900-2010	Portugal	1940-2010
Finland	1900-2010	Spain	1908-2010
France	1900-2010	Sweden	1900-2010
Germany	1990-2010	Switzerland	1900-2010
Iceland	1900-2010	Taiwan	1970-2010
Ireland	1950-2009	United Kingdom	1922-2010
Israel	1983-2009	United States	1933-2010
Italy	1900-2009		

Source: HMD (2015)

3. Illustration

3.1 Gompertz decomposition

Table 3 presents the decomposition of life expectancy at age 30 by M and β for Swedish females at the beginning, middle, and end of the 20th century and for the HMD females average, between 2000 and 2005. For the three periods selected and for both populations, changes in the modal age at death (ΔM) are the main components driving the change in life expectancy.

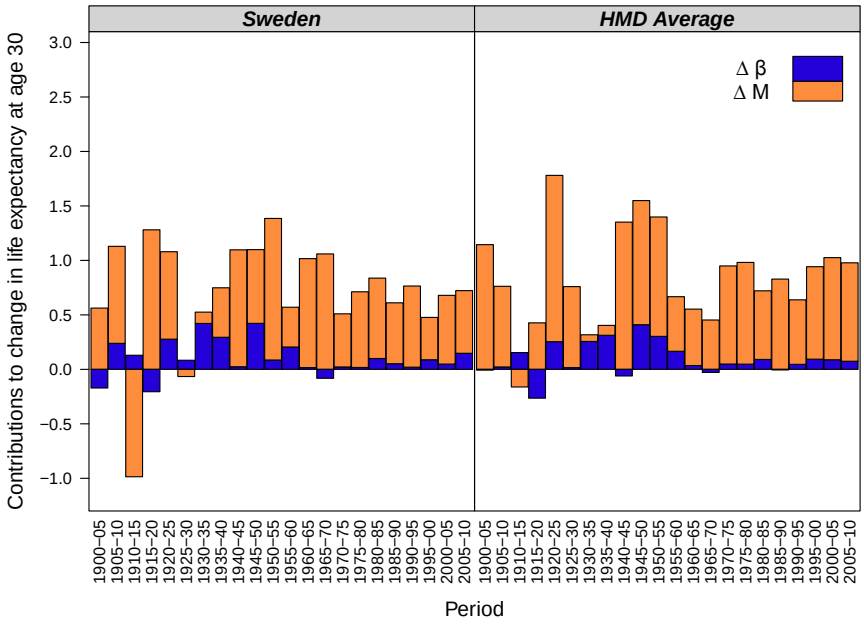
To further study the year-to-year changes, Figure 2 presents the decomposition from 1900 until 2010 in 5-year intervals for Swedish and HMD average females. Over most periods, the gains in life expectancy at age 30 were mainly the result of a shift in the modal age at death (ΔM). Until the end of the 1950s, variability reduction contributions to changes in life expectancy ($\Delta \beta$) have been more important than in the following periods. However, even during those years, changes in life expectancy were mainly driven by changes in the mode. Figure 7 in Appendix C presents similar results for 25 of the HMD countries.

Table 3: Female life expectancy at age 30 ($e_{30,t}$) and its decomposition due to changes in the Gompertz parameters, Sweden and HMD average, 1900, 1950, and 2000

	Sweden			HMD Average	
	1900	1950	2000	2000	(min, max)
$e_{30,t}$	37.82	44.18	52.04	51.43	(49.23, 54.79)
$e_{30,t+5}$	38.21	45.56	52.72	52.45	(50.40, 55.67)
$\dot{e}_{30,t}$	0.39	1.39	0.68	1.03	
$\Delta\beta$	-0.17	0.09	0.05	0.09	(-0.11, 0.22)
ΔM	0.56	1.30	0.63	0.94	(0.48, 2.08)
$\Delta\beta + \Delta M$	0.39	1.39	0.68	1.03	

Source: HMD (2015) and authors' own calculation.
Note: By rounding the numbers to the second decimal point in the table, the sum of the contributions ($\sum \Delta_i$) might differ slightly from the difference in life expectancy ($\dot{e}_{30,t}$).

Figure 2: Trends over time of the Gompertz parameters' contribution to changes in female life expectancy at age 30 ($\dot{e}_{30,t}$), Sweden and HMD average, 1900-2010



Source: HMD (2015) and authors' own calculation.

Changes in variability of senescent mortality alone would not have been sufficient to generate the important gains in life expectancy at age 30 observed since 1900. An explanation for this small variability effect compared with the important shifting effect for senescent mortality still needs to be provided. A possible explanation is that as only senescent mortality is analyzed by the Gompertz model, it does not consider the ages essentially responsible for mortality compression, i.e., infant, child and early adult (Cheung and Robine 2007). To address the latter aspect of how mortality at young ages has influenced changes in life expectancy, we present results for the Gompertz-Makeham and Siler models in the next sections.

3.2 Gompertz-Makeham decomposition

The Gompertz-Makeham model can help us understand the impact of early adult mortality changes on compression and shifting mortality. Table 4 presents an application of the decomposition of life expectancy at age 30 using the Gompertz-Makeham model for Swedish and HMD average females at three points in time. Among the parameters influencing variability changes (β and c), the Makeham term (c) has a similar influence on life expectancy changes than the shape parameter β, for most of the times studied in Table 4.

Table 4: Female life expectancy at age 30 ($e_{30,t}$) and its decomposition due to changes in the Gompertz-Makeham parameters, Sweden and HMD average, 1900, 1950, and 2000

	Sweden			HMD Average	
	1900	1950	2000	2000	(min, max)
$e_{30,t}$	32.50	43.30	51.41	50.75	(49.02, 53.66)
$e_{30,t+5}$	32.95	45.04	52.09	51.70	(49.52, 54.49)
$\dot{e}_{30,t}$	0.45	1.74	0.68	0.95	
Δc	0.11	0.77	0.02	-0.09	(-0.73, 0.63)
$\Delta \beta$	-0.12	-0.03	0.05	0.11	(0.00, 0.36)
ΔM	0.46	1.00	0.61	0.93	(0.48, 2.01)
$\Delta c + \Delta \beta + \Delta M$	0.45	1.74	0.68	0.95	

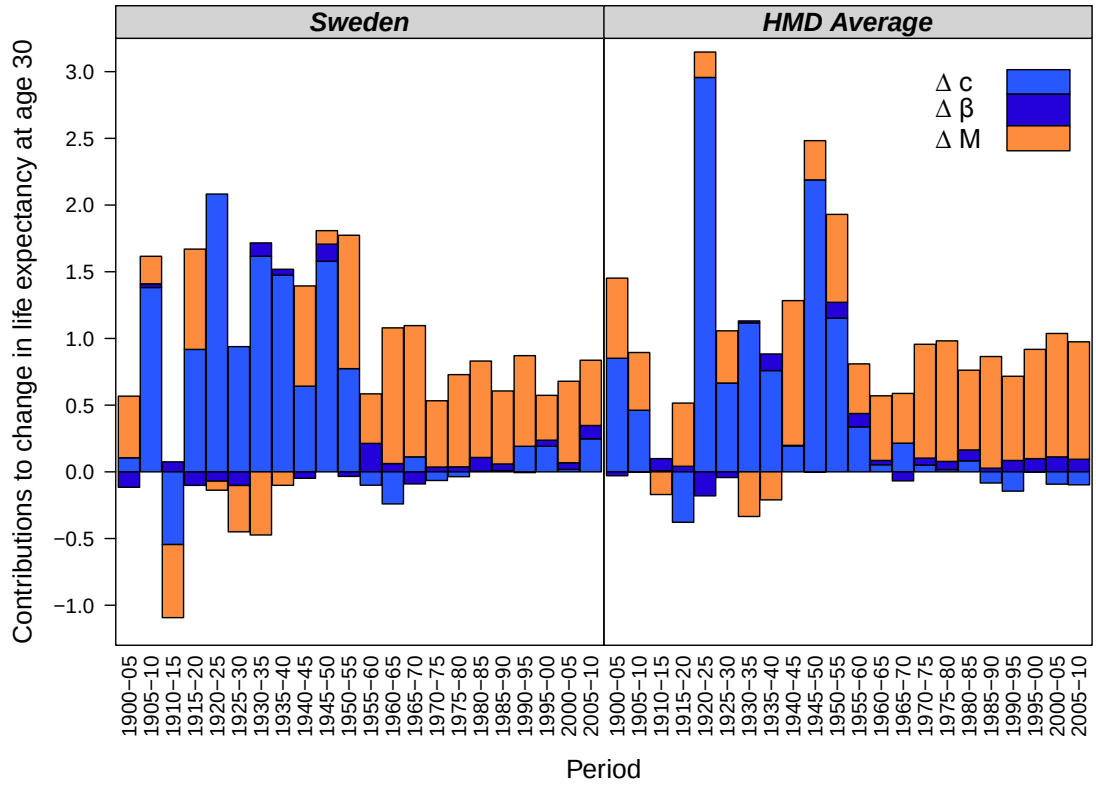
Source: HMD (2015) and authors' own calculation.
Note: By rounding the numbers to the second decimal point in the table, the sum of the contributions ($\sum \Delta_i$) might differ slightly from the difference in life expectancy ($\dot{e}_{30,t}$).

Figure 3 presents the decomposition for 5-year periods between 1900 and 2010 for Swedish and HMD average females. The gains in life expectancy at age 30 before the 1950s were mainly driven by changes in variability of the age at death, which is essentially captured

by changes in the parameter c. After this initial period of variability decline, changes in life expectancy at age 30 are mainly the result of shifting mortality (ΔM).

The inclusion of a parameter capturing early adult background mortality appears essential, then, to demonstrate the effect of variability reduction on life expectancy at age 30. Figure 8 in Appendix C shows similar results for the selected HMD countries. The next section presents an application of the Siler decomposition, in order to understand the role of infant mortality on the changes in life expectancy.

Figure 3: Trends over time of the Gompertz-Makeham parameters' contribution to changes in female life expectancy at age 30 ($\dot{e}_{30,t}$), Sweden and HMD average, 1900-2010



Source: HMD (2015) and authors' own calculation.

3.3 Siler decomposition

Table 5 and Figure 4 present the results of life expectancy decomposition, using a Siler model, for female mortality in Sweden and HMD average at ages 0 and older. Similar

to the Gompertz-Makeham decomposition, the results suggest that changes in life expectancy at birth before the 1950s were mainly the result of variability reductions. Gains in life expectancy due to changes in the parameter β are still small, and the main gains are due to variability reductions coming from changes in infant and background parameters.

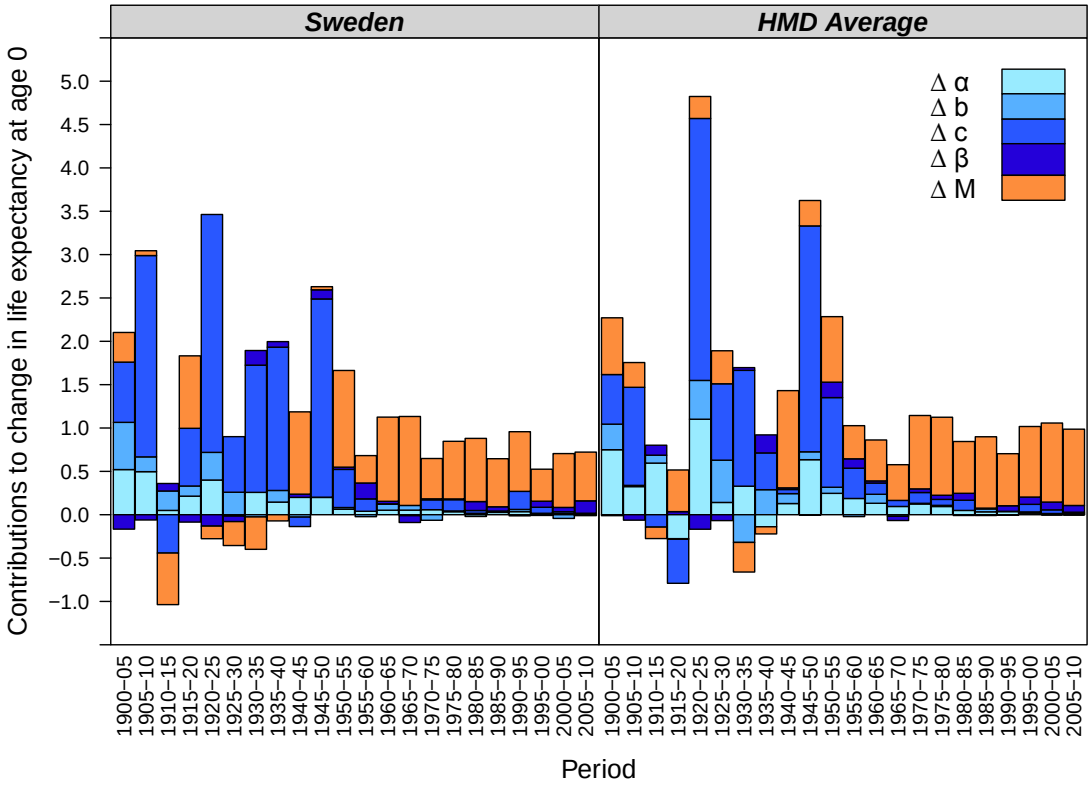
Since the mid-1960s, the modal age at death has been the key parameter leading the changes in life expectancy. Changes in the modal age at death were responsible for more than 70% of the increase in $e_{0,t}$ since 1965 for females from both Swedish and HMD average. Figure 9 in Appendix C presents similar results for 25 of the HMD countries.

Table 5: Female life expectancy at age 0 ($e_{0,t}$) and its decomposition due to changes in the Siler parameters, Sweden and HMD average, 1900, 1950 and 2000

	Sweden			HMD Average	
	1900	1950	2000	2000	(min, max)
$e_{0,t}$	56.06	72.78	81.68	80.93	(78.92, 84.16)
$e_{0,t+5}$	58.00	74.44	82.34	81.99	(79.92, 85.01)
$\dot{e}_{0,t}$	1.94	1.66	0.66	1.06	
$\Delta\alpha$	0.52	0.06	0.01	0.01	(-0.04, 0.04)
Δb	0.54	0.02	-0.04	0.00	(-0.07, 0.05)
Δc	0.69	0.44	0.02	0.04	(-0.16, 0.23)
$\Delta\beta$	-0.17	0.03	0.05	0.09	(-0.07, 0.26)
ΔM	0.34	1.11	0.62	0.91	(0.48, 2.01)
$\Delta\alpha$+Δb+ Δc+$\Delta\beta$+ΔM	1.94	1.66	0.66	1.06	

Source: HMD (2015) and authors' own calculation.
Note: By rounding the numbers to the second decimal point in the table, the sum of the contributions ($\sum \Delta_i$) might differ slightly from the difference in life expectancy ($\dot{e}_{0,t}$).

Figure 4: Trends over time of the Siler parameters' contribution to changes in female life expectancy at age 0 ($\dot{e}_{0,t}$), Sweden and HMD average, 1900-2010

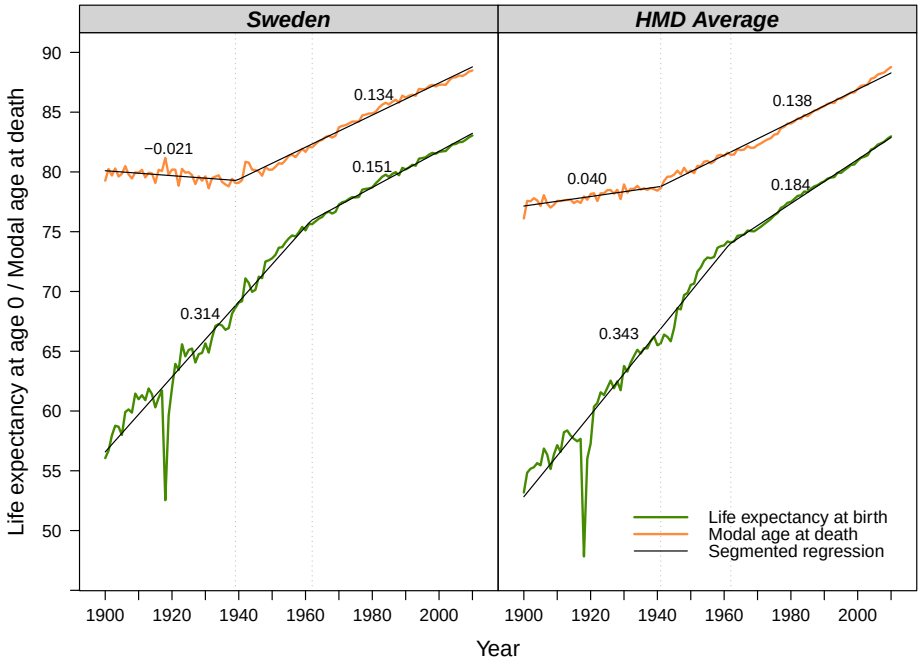


Source: HMD (2015) and authors' own calculation.
Note: Appendix D presents these results in terms of relative differences.

3.4 Life expectancy and modal age at death

Figure 5 shows the life expectancy at birth and the modal age at death between 1900 and 2010 for females from Sweden and HMD average. Until the beginning of the 1940s, life expectancy increased around 4 months per year on average, while the modal age at death stayed nearly constant. The gains in life expectancy over this first period were in great part the result of improvements in infant and background mortality, and thus, variability reductions (Figure 4).

Figure 5: Modal age at death and life expectancy at birth and their respective segmented regression for females, Sweden and HMD average, 1900-2010



Source: HMD (2015) and authors' own calculation.
Note: The slopes and breakpoints of the modal age at death and life expectancy trends are calculated with a segmented regression methodology (Camarda, Vallin, and Meslé 2012).

A second period followed in which the mode begins to increase at a faster pace while life expectancy increase keeps its previous pace. Between 1940 and 1965, the mode contribution to changes in life expectancy increased, while variability contributions gradually lost importance. During these years, variability contributions to changes in life expectancy decreased from more than 60% to less than 30% of the total gains (Appendix D). This period is one of acceleration in the increase of the modal age at death and marks the transition from compression to shifting mortality.

Since the mid-1960s, the mode and life expectancy have been increasing at a comparable pace of around 2 months per year. This change in the pace of life expectancy coincides with the change of pace observed by Vallin and Meslé (2009) for the best-practice life expectancy. Life expectancy is still increasing at a marginally faster pace than the mode due to small extra contributions from variability reduction. Nevertheless, gains in life expectancy are mainly driven by changes in the modal age at death and thus shifting mortality (Figure 4). This parallels the 3-phases transition described by Cheung et al. (2009).

4. Discussion and conclusion

Using recent parametrization of the Gompertz model, we separated changes in life expectancy by the variability reduction effect, captured by β, from the shifting mortality effect, captured by M. The methodology is then extended to other parametric representations of mortality, and particularly to the Gompertz-Makeham and Siler models, to consider the effect of young adult, child and infant mortality changes. This new decomposition method, using parametric models, allows us to understand and quantify the respective impact of shifting mortality and variability changes on life expectancy.

Our results suggest that mortality compression was the main driver of change in life expectancy at birth before the 1950s, due to a decrease in infant and background mortality. After this period, changes in life expectancy became gradually dependent on the shift in the senescent modal age at death. These results are consistent with the findings of other studies looking at changes in the modal age at death and at different variability measures (Wilmoth and Horiuchi 1999; Robine 2001; Yashin et al. 2001; Canudas-Romo 2008). The results also confirm the increasing importance of the modal age at death as a key indicator of lifespan. The modal age at death has increased since the beginning of the 1940s and has become the main driver of longevity extension since the 1960s. An important feature of this indicator is that, in populations that experience declining mortality, its change is only determined by old-age mortality.

In the above illustrations, the results of the decomposition are presented for female life expectancy only. However, similar results are found when decomposing male life expectancy, but with a shifting pattern appearing later in time. Shifting mortality became the main driver of life expectancy increase in the late 1970s for males (results available from the authors).

We asked previously how and to what extent, one process replaced the other. Our methodology allows us to observe and quantify the gradual replacement of a compression pattern by a shifting pattern in a relatively short period of time. We can also observe that, even if shifting the modal age at death is explaining a great deal of the life expectancy increase nowadays, lifespan variability reductions still play a role in mortality changes.

The results are, however, sensitive to the selected parametric model, and especially if the model is able to include infant and background mortality parameters. The dominating effect of variability reductions on life expectancy increase in the first half of the 20th century can only be seen when using the Gompertz-Makeham and Siler models in the above illustrations. When only senescent mortality is analyzed, the compression of mortality only has a minor impact on life expectancy improvement, even before the 1950s. It could thus be theorized that in a context where infant and young adult mortality is low, as it is the case in most industrialized countries, variability reductions will have limited impact on life expectancy, and shifting the modal age at death would be the main dynamic that would allow life expectancy to increase.

The choice of the parametric model for senescent mortality might also have influenced the results. The illustration section presented the application of the methodology to discrete data using a Gompertz model. Other models could, however, have been more appropriate, such as the Logistic, to consider the deceleration in the hazard at very old-ages. However, an application using the Logistic model shows that the results are very similar to the findings obtained with the Gompertz model (Appendix E).

A previous attempt to quantify the effect of shifting the mortality schedule on life expectancy has been done by De Beer and Janssen (2014). Their procedure consists in evaluating the effect on life expectancy of changing the value of their model parameters on life expectancy. However, to the authors' knowledge, our current study is the first attempt to quantify the gain in life expectancy produced by a change in variability and shifting mortality. Our procedure allows us to differentiate between both underlying mortality processes and their respective impact on life expectancy, and also to determine when and how one process has replaced the other.

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Appendices

Appendix A: Changes in variability: effects of β and M

We stated that changing the parameter β of the Gompertz hazard equation will have an effect on variability of the age at death. In this section, we evaluate this effect by looking at a measure of variability, namely e-dagger ($e^\dagger$), and attest the contribution of each of the parameters in the Gompertz hazard to the change in variability. Among the different indicators used to measure variability of the age at death (Robine 2001; Wilmoth and Horiuchi 1999; Vaupel, Zhang, and van Raalte 2011), we focus on $e^\dagger$, a measure of lifespan disparity often interpreted as the average years of life expectancy lost due to death:

$$e_t^\dagger = \int_0^\omega H_{x,t} l_{x,t}\, d_x, \tag{A1}$$

where $l_{x,t}$ is the survival distribution, and $H_{x,t}$ is the cumulative hazard, equal to:

$$H_{x,t} = e^{\beta_t (x - M_t)} - e^{-\beta_t M_t} = \frac{1}{\beta_t}\mu_{x,t} - e^{-\beta_t M_t}.$$

Therefore, $e_t^\dagger$ can be written as

$$e_t^\dagger = \frac{1}{\beta_t}\int_0^\omega \mu_{x,t} l_{x,t}\, d_x - e^{-\beta_t M_t}\int_0^\omega l_{x,t}\, d_x,$$

leading to

$$e_t^\dagger = \frac{1}{\beta_t} - e^{-\beta_t M_t} e_{0,t}. \tag{A2}$$

Wrycza (2014) also showed this relation for Gompertz-Makeham entropy using the standard parametrization. It is possible to quantify the respective effects of β_t and M_t on $e_t^\dagger$ by looking at its time derivative, denoted by a dot on top of the variable. From equation (A2) changes in $e_t^\dagger$ over time ($\dot{e}_t^\dagger$) can be expressed by components of changes for both Gompertz parameters:

$$\dot{e}_t^\dagger = -\dot{\beta}_t \left[\frac{1}{\beta_t^2 e_{0,t}} - M_t e^{-\beta_t M_t}\right] e_{0,t} - \dot{e}_{0,t}[e^{-\beta_t M_t}] + \dot{M}_t[\beta_t e^{-\beta_t M_t} e_0]. \tag{A3}$$

As shown by equation (6), $\dot{e}_{0,t}$ can be decomposed by a factor of change of M_t and β_t. Therefore, $e_{0,t}$ contributions to changes in $e_t^\dagger$ can be distributed into M_t and β_t contributions, obtaining

$$\dot{e}_t^\dagger = \underbrace{-\dot{\beta}_t \left[\frac{1}{\beta_t^2} - (M_t e_{0,t} + F_\beta) e^{-\beta_t M_t} \right]}_{\delta\beta} + \underbrace{\dot{M}_t [(\beta_t e_{0,t} - F_M) e^{-\beta_t M_t}]}_{\delta M}, \tag{A4}$$

where $\delta\beta$ and δM are the gains in $e_t^\dagger$ produced by a change in parameters β_t and M_t, respectively, and F_β and F_M are the terms multiplying $\dot{\beta}_t$ and $\dot{M}_t$ respectively in equation (6):

$$F_\beta = \int_0^\omega l_{a,t} \int_0^a \mu_{x,t} (\frac{1}{\beta_t} + x - M_t) \, d_x \, d_a \tag{A5a}$$

$$F_M = \int_0^\omega l_{a,t} \int_0^a \beta_t \mu_{x,t} \, d_x \, d_a. \tag{A5b}$$

Table 6 shows an application of the $e_t^\dagger$ decomposition to Swedish and HMD average female data. It is shown that the main factor of variability changes comes from changes in the parameter β_t. However, increasing the modal age at death produced a small increase in lifespan disparities.

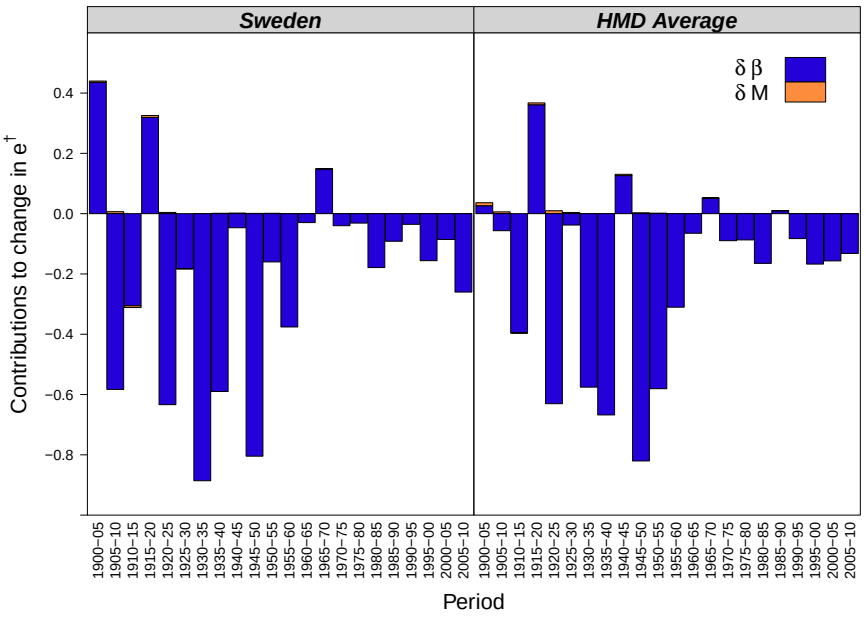
Changes in $e_t^\dagger$ are thus driven by both Gompertz parameters. In general, increasing β_t will lead to a smaller variability of the age at death, while increasing M_t would have the opposite effect. These results are consistent with the results of Engelman, Caswell, and Agree (2014). Using a Siler model, the authors show that a decrease in the timing parameter for senescent mortality (α_2) will increase the variability (Engelman, Caswell, and Agree 2014). However, Table 6 and Figure 6 show that this variability expansion resulting from a shift in M_t will generally be too small to drive substantial changes in lifespan disparities. The assumption that mortality compression is produced by an increase in β_t is then confirmed.

Table 6: **e-dagger ($e_t^\dagger$) and its decomposition due to changes in the Gompertz parameters, Swedish and HMD countries average, females, 1900, 1950 and 2000**

	Sweden			HMD Average
	1900	1950	2000	2000
$e_t^\dagger$	13.0425	9.7848	8.8384	9.2218
$e_{t+5}^\dagger$	13.4824	9.6259	8.7529	9.0654
$\dot{e}_t^\dagger$	0.4399	-0.1589	-0.0855	-0.1564
$\delta\beta$	0.4356	-0.1601	-0.0856	-0.1567
δM	0.0043	0.0012	0.0001	0.0003
$\delta\beta + \delta M$	0.4398	-0.1589	-0.0855	-0.1564

Source: HMD (2015) and authors' own calculation.
Note: By rounding the numbers to the fourth decimal point in the table, the sum of the contributions ($\sum \delta_i$) might differ slightly from the difference in e-dagger ($\dot{e}_t^\dagger$).

Figure 6: **Trends over time of the Gompertz parameters' contribution to changes in e-dagger ($e_t^\dagger$) for females, Sweden and HMD average, 1900-2010**



Source: HMD (2015) and authors' own calculation.

Appendix B: Applying the decomposition to discrete data

The estimation procedure of our methodology can be done to discrete data by estimating the functions at their midpoint over a certain time interval (Preston, Heuveline, and Guillot 2001; Vaupel and Canudas-Romo 2003). As suggested by Vaupel and Canudas-Romo (2003), if data are available between time t and $t + h$, the midpoint value of the function $v_{x,t}$ was estimated by

$$v_{x,t+h/2} = v_{x,t} \left(\frac{v_{x,t+h}}{v_{x,t}} \right)^{1/2}. \tag{B1}$$

The derivative of the function $v_{x,t+h/2}$ was estimated by

$$\dot{v}_{x,t+h/2} = v_{x,t+h/2} \, \frac{ln[\frac{v_{x,t+h}}{v_{x,t}}]}{h}. \tag{B2}$$

In some cases, it could make more sense to assume a linear change in the interval (Vaupel and Canudas-Romo 2003). In these cases, we used

$$v_{x,t+h/2} = \frac{v_{x,t+h} + v_{x,t}}{2} \tag{B3}$$

and

$$\dot{v}_{x,t+h/2} = \frac{v_{x,t+h} - v_{x,t}}{h}. \tag{B4}$$

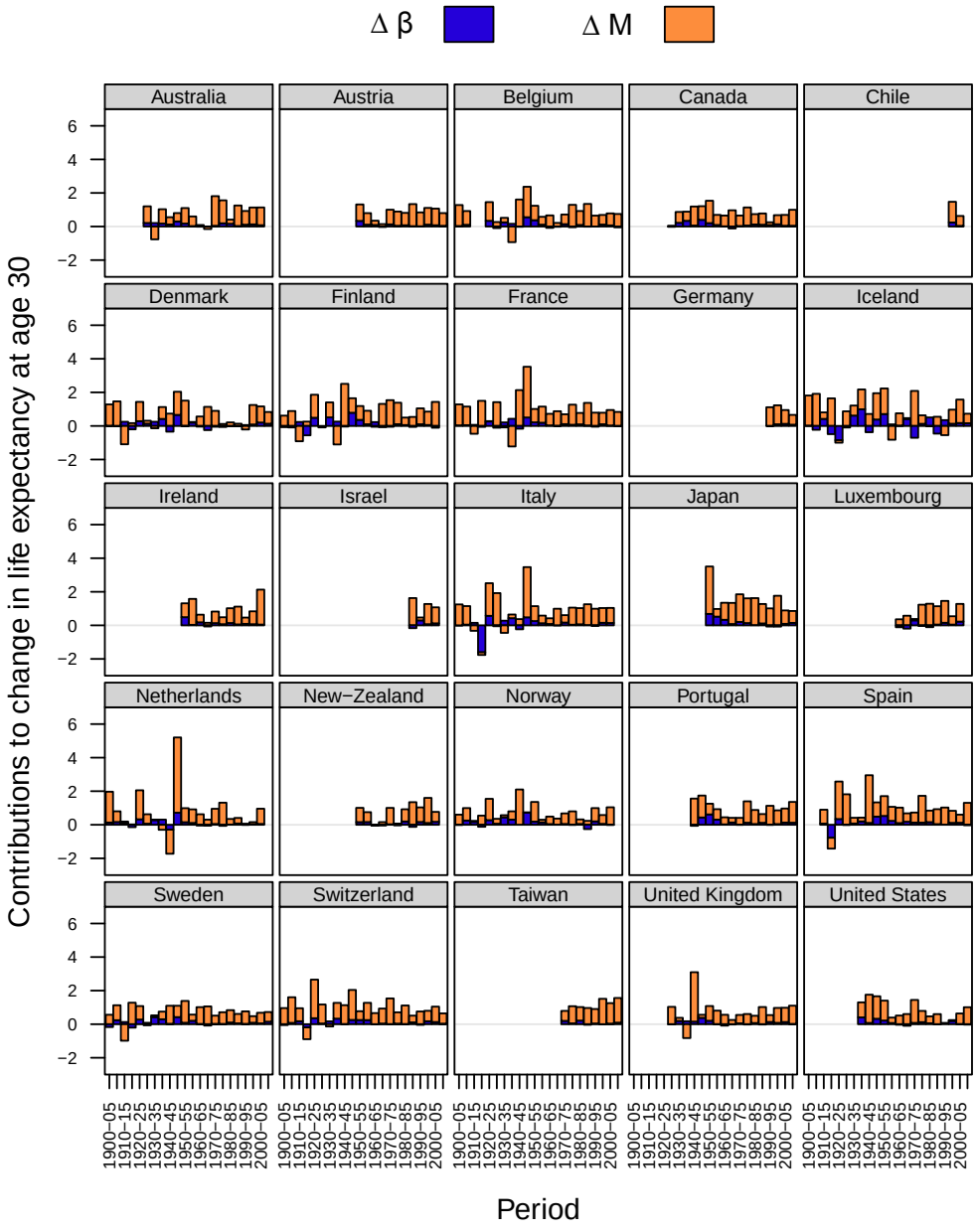
We used these latter estimates for the change over time of the life expectancy ($\dot{e}_{0,t}$). The other functions were estimated by assuming an exponential change, as presented in equations (B1) and (B2). It is important to note that these procedures generate annual estimates, and also that the midpoint of each term multiplying $\dot{\beta}_t$ and $\dot{M}_t$ in equation (6) should be estimated. For example, the annualized ΔM for the period t to $t + h$ ($\Delta M_{t+h/2}$) using the Gompertz model is calculated as

$$\Delta M_{t+h/2} = \dot{M}_{t+h/2} \int_0^{\omega} l_{x,t+h/2} \, \beta_{t+h/2} \, H_{x,t+h/2} \, dx, \tag{B5}$$

where $H_{x,t}$ is the cumulative hazard at time t and age x. In the illustration section, the results are presented for five-year periods. As the above equations are valid for annual changes, the decomposition is applied to yearly differences, which are then summed up to equal for longer periods. Yearly estimates are generally more accurate than the estimates for longer periods. Similar methodology is also applied for the Gompertz-Makeham and Siler models.

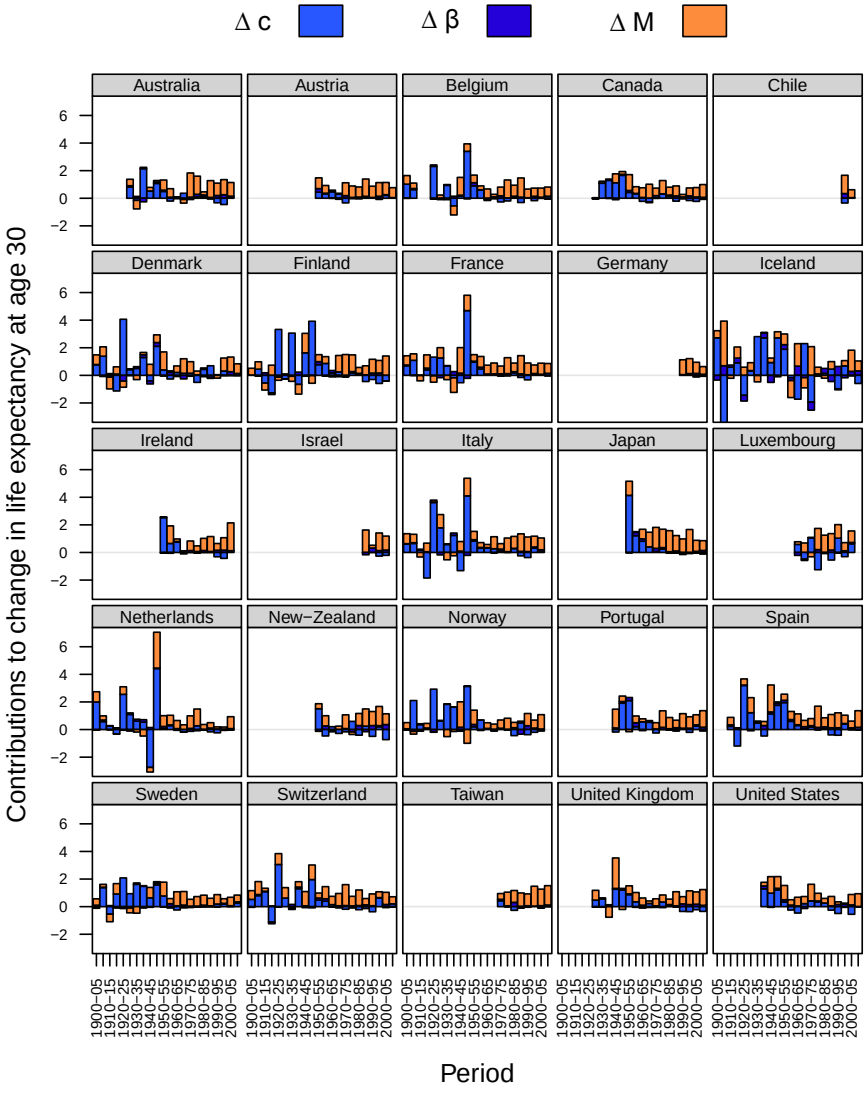
Appendix C: International comparison

Figure 7: **Trends over time of the Gompertz parameters' contribution to changes in female life expectancy at age 30 ($\dot{e}_{30,t}$), HMD countries, 1900-2010**



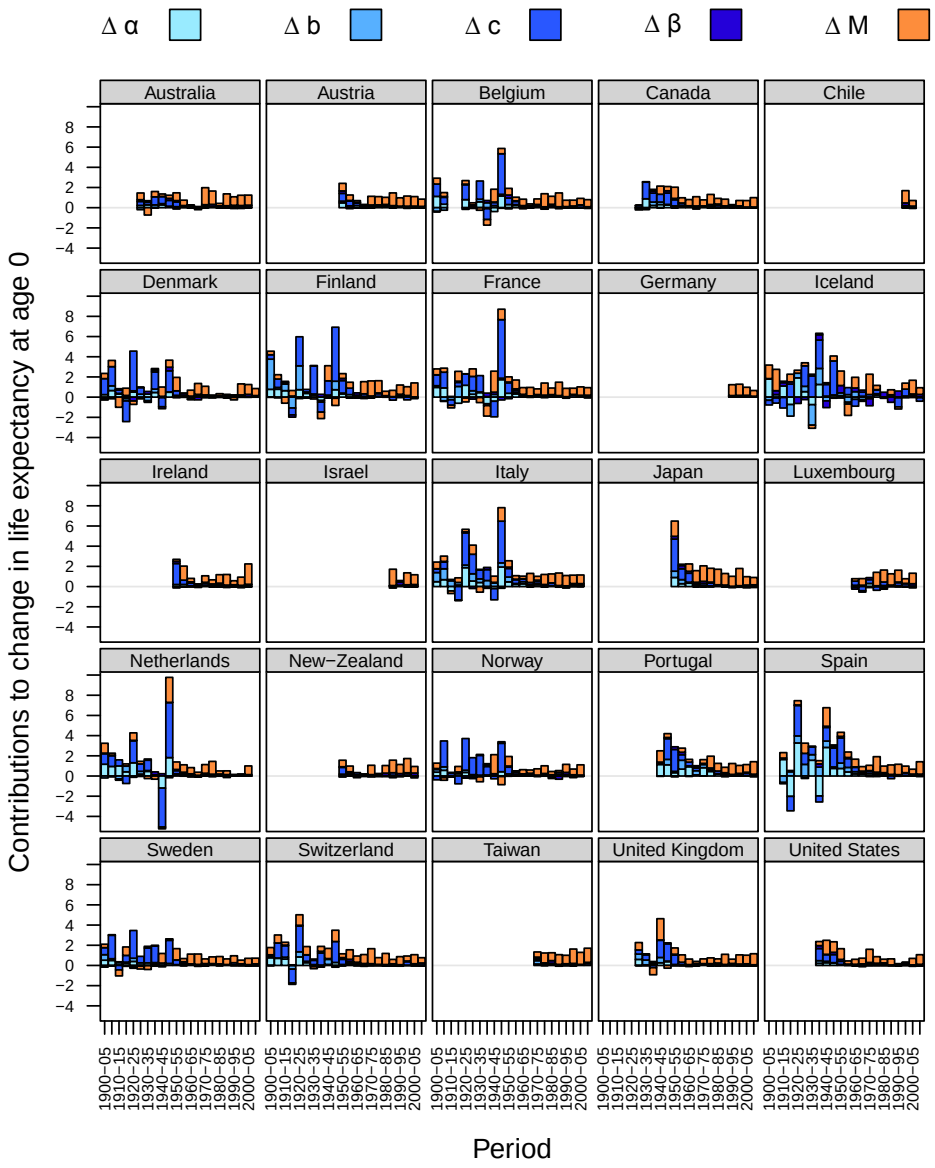
Source: HMD (2015) and author's own calculation.

Figure 8: **Trends over time of the Gompertz-Makeham parameters' contribution to changes in female life expectancy at age 30 ($\dot{e}_{30,t}$), HMD countries, 1900-2010.**



Source: HMD (2015) and authors' own calculation.

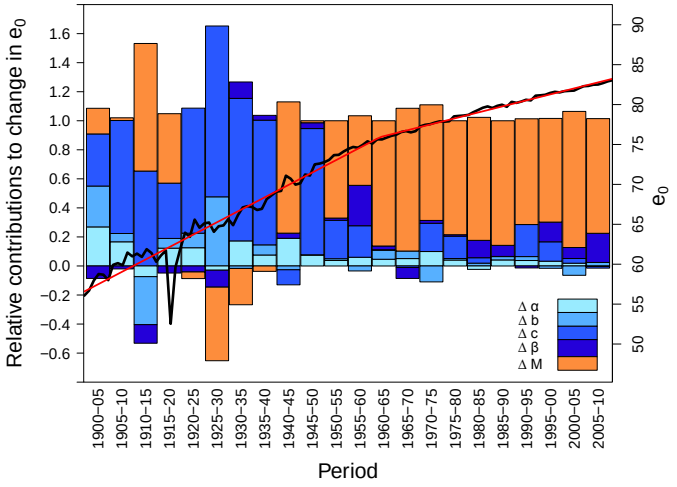
Figure 9: Trends over time of the Siler parameters' contribution to changes in female life expectancy at age 0 ($\dot{e}_{0,t}$), HMD countries, 1900-2010



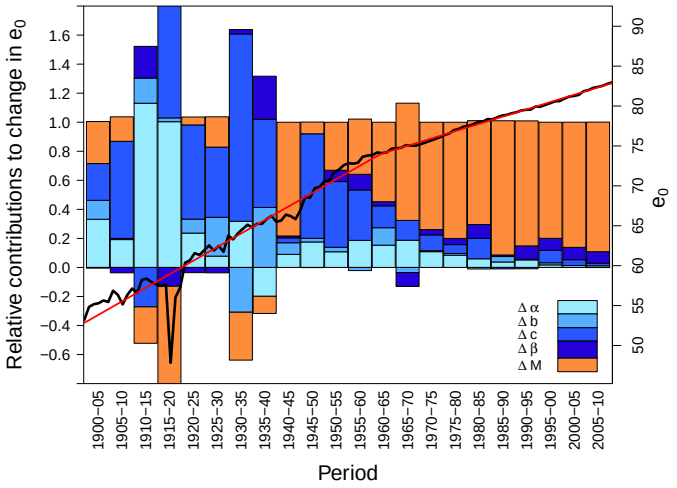
Source: HMD (2015) and authors' own calculation.

Appendix D: Relative differences

Figure 10: **Female life expectancy at birth ($e_{0,t}$) and relative gain in life expectancy due to changes in the Siler parameters, Sweden and HMD average, 1900-2010. The black and red lines are the life expectancy at birth observed (in black) and modeled (in red) presented in Figure 5.**



a) Sweden



b) HMD average

Source: HMD (2015) and authors' own calculation.
Note: The sum of the contributions ($\sum \Delta_i$) for each bar equal to 1 in this figure.

Appendix E: Life expectancy decomposition: Gompertz, Logistic and Weibull models

Table 7: Female life expectancy at age 30 ($e_{30,t}$) and its decomposition due to changes in the modal age at death (ΔM) and shape ($\Delta\beta$), using Gompertz, Logistic and Weibull models, Sweden and HMD average, 1900, 1950 and 2000

	Sweden			HMD Average	
	1900	1950	2000	2000	(min, max)
			Gompertz		
$e_{30,t}$	37.82	44.18	52.04	51.43	(49.23, 54.79)
$e_{30,t+5}$	38.21	45.56	52.72	52.45	(50.40, 55.67)
$\dot{e}_{30,t}$	0.39	1.39	0.68	1.03	
$\Delta\beta$	-0.17	0.09	0.05	0.09	(-0.11, 0.22)
ΔM	0.56	1.30	0.63	0.94	(0.48, 2.08)
$\Delta\beta + \Delta M$	0.39	1.39	0.68	1.03	
			Logistic		
$e_{30,t}$	37.93	44.25	52.13	51.50	(49.30, 54.90)
$e_{30,t+5}$	38.33	45.64	52.80	52.53	(50.46, 55.78)
$\dot{e}_{30,t}$	0.40	1.39	0.67	1.02	
$\Delta\beta$	-0.17	0.07	0.04	0.08	(-0.15, 0.22)
ΔM	0.57	1.32	0.62	0.94	(0.48, 2.12)
$\Delta\beta + \Delta M$	0.40	1.39	0.67	1.02	
			Weibull		
$e_{30,t}$	38.50	44.33	52.09	51.46	(49.36, 55.05)
$e_{30,t+5}$	38.98	45.77	52.74	52.47	(50.47, 55.88)
$\dot{e}_{30,t}$	0.48	1.44	0.65	1.01	
$\Delta\beta$	0.05	0.02	0.02	0.04	(-0.03, 0.08)
ΔM	0.42	1.41	0.62	0.97	(0.50, 2.10)
$\Delta\beta + \Delta M$	0.48	1.44	0.65	1.01	

Source: HMD (2015) and authors' own calculation.
Note: By rounding the numbers to the second decimal point in the table, the sum of the contributions ($\sum \Delta_i$) might differ slightly from the difference in life expectancy ($\dot{e}_{30,t}$).

C. Eidesstattliche Versicherung

Ich erkläre hiermit, dass ich die vorliegende Arbeit ohne unzulässige Hilfe Dritter und ohne Benutzung anderer als der angegebenen Hilfsmittel angefertigt habe; die aus fremden Quellen direkt oder indirekt übernommenen Gedanken sind als solche kenntlich gemacht.

Die Arbeit wurde bisher weder im Inland noch im Ausland in gleicher oder ähnlicher Form einer Prüfungsbehörde zur Erlangung eines akademischen Grades vorgelegt.

Rostock, 9. Mai 2018

Marcus Ebeling